

# Urinary Tract Emergencies

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Emergencies of the upper and lower urinary tract are commonly seen in small animal practice. The goal of this article is to discuss the most common of these emergencies, with a focus on diagnosis and treatment. This article discusses urethral obstruction in the dog and cat, acute renal failure, and uroperitoneum.

## **Urethral obstruction**

Urethral obstruction is one of the most common emergencies seen involving the urinary system. Untreated, it can rapidly progress to a life-threatening crisis that is characterized by severe electrolyte and acid-base disturbances.

Urethral obstruction is more common in male cats and dogs than in female animals, but both sexes can be affected. The most frequent cause of obstruction is mucous plugs (in cats) or uroliths (in cats and dogs); however, obstruction can occur secondary to neoplasia as well as from strictures associated with trauma.

Most mucous plugs in cats are associated with magnesium ammonium phosphate (struvite) crystals [1]. Uroliths in cats are most commonly struvite or calcium oxalate [1,2]. In dogs, struvite and calcium oxalate uroliths are also the most prevalent [3]. Neoplasms that can result in urethral obstruction include squamous cell carcinoma, transitional cell carcinoma, and other less frequently seen tumors [4].

Regardless of the underlying cause, prolonged urethral obstruction results in fluid deficits that lead to hypovolemia and decreased tissue perfusion. The adverse effects on cardiac output, and thus tissue perfusion, are exacerbated by the profound electrolyte and acid-base disturbances seen with obstruction. In addition to azotemia, urethral obstruction is

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associated with metabolic acidosis, hyperkalemia, hyperphosphatemia, and hypocalcemia [5].

Metabolic acidosis occurs in urethral obstruction as a result of an inability to excrete hydrogen ions via the urinary system. Lactic acidosis secondary to low cardiac output may contribute to a worsening acid-base status. Profound metabolic acidosis ( $\text{pH} < 7.2$ ) has far-reaching effects in the body, affecting the respiratory, cardiovascular, and central nervous systems. Initially, the response of the body to acidosis is to increase the minute ventilation by increasing the respiratory rate and/or the tidal volume. Severe acidosis can predispose the patient to cardiac arrhythmias as well as to decreases in cardiac contractility and an inotropic response to catecholamines [6]. In the central nervous system (CNS), acidosis can result in neurologic signs that can range from depression to coma [6]. This may be further compounded by poor tissue perfusion and uremia.

Hyperkalemia is the electrolyte disturbance most commonly associated with urethral obstruction. Hyperkalemia occurs because of a decrease in renal excretion of potassium as well as from the shifting of potassium from the intracellular space in response to acidosis. Potassium plays a major role in the determination of resting cell membrane potential. Initially, extracellular hyperkalemia can make the cell more excitable, but as potassium elevations become severe, the resting membrane potential may become less than the threshold potential, thus making the cell unable to repolarize after depolarization [7]. Clinically, this effect is seen in muscle tissue and the conduction system of the heart [7].

The changes that are seen on the electrocardiogram (ECG) in response to hyperkalemia include bradycardia, decreased amplitude or absent P waves, a widened QRS complex, tall T waves, a shortened QT interval, and ST segment depression. Elevation in extracellular potassium also results in generalized muscle weakness. It is extremely important to remember that the severity of clinical signs does not necessarily correlate with the magnitude of change in the serum potassium concentration. Thus, a cat with a serum potassium level of 8.0 may be severely compromised, whereas another cat with a serum potassium level of 9.0 may be hemodynamically stable. Decisions about the treatment of hyperkalemia should be based on the overall clinical assessment of the patient and not just on clinical laboratory parameters.

In addition to hyperkalemia, urethral obstruction can result in ionized hypocalcemia [5,8]. This exacerbates the effects of any concomitant hyperkalemia [9]. The metabolic acidosis may ameliorate the severity of the ionized hypocalcemia because it favors a shift of calcium from the protein-bound fraction of total calcium to the ionized calcium fraction. Clinically, hypocalcemia may result in neuromuscular hyperexcitability, decreased cardiac contractility, and peripheral vasodilation [10].

The final electrolyte disturbance commonly seen with urethral obstruction is hyperphosphatemia. This occurs because of a decrease in renal clearance

of phosphorus. The presence of hyperphosphatemia may contribute to the development of hypocalcemia as well as contributing to the metabolic acidosis.

### *Clinical signs and the initial database*

The clinical signs associated with urethral obstruction include stranguria or dysuria, vocalizing, lethargy, anorexia, vomiting, excessive licking of the perineum, and diarrhea [5]. On physical examination, the most striking finding is an extremely firm bladder on abdominal palpation. In addition, the body temperature of these animals may range from hypothermic to hyperthermic. They may be bradycardic or tachycardic. They may have an elevated respiratory rate and poor peripheral pulse quality. Lethargy and depression may also be appreciated [5].

An initial database for the urethral obstruction patient should include, if possible, measurement of packed cell volume (PCV); total solids; blood urea nitrogen (BUN); blood glucose; venous blood gas; and electrolytes, including ionized calcium, potassium, sodium, and chloride. Blood pressure measurement and an ECG provide information on the cardiovascular effects of the metabolic changes, whereas a complete blood cell count (CBC) and serum chemistry profile give more extensive information regarding the patient's overall status. Survey radiographs, contrast cystography, and ultrasonography more clearly characterize the cause of the obstruction but should be withheld until the animal has been stabilized. On relief of the obstruction, a urine sample should be taken for urinalysis as well as urine culture. Urine samples should be analyzed within 60 minutes of collection to minimize the effects of time and temperature on crystal formation [11].

### *Treatment*

The ultimate goal of treatment is to relieve the obstruction and re-establish urethral patency. The hemodynamic stability of the patient must first be addressed, however.

In addition to relieving the obstruction, therapy should be instituted for correction of acid-base abnormalities, azotemia, and electrolyte disturbances (Table 1). The mainstay in achieving these goals is fluid therapy. In the past, 0.9% sodium chloride (NaCl) has been the fluid of choice because it contains no potassium, but it may contribute to the metabolic acidosis seen in these patients. Recent data show that a balanced electrolyte solution, even with potassium, results in no clinically significant difference in acid-base or electrolyte parameters [12]. Animals that have had a urethral obstruction frequently exhibit a postobstructional diuresis as the uremia is corrected; this is especially true of animals that have been obstructed long enough to exhibit electrolyte or acid-base alterations. This is of vital importance in determining fluid rates, because the required rate is often far greater than what would be chosen for a normal animal. It is not remarkable for an

Table 1  
Useful drugs for electrolyte and acid-base disturbances

Drug	Dose range	Frequency	Route	Indications
Calcium gluconate	50–100 mg/kg	PRN	IV	Hyperkalemia, hypocalcemia
Regular insulin	0.1–0.25 U/kg	Every 2–4 hours	IV	Hyperkalemia
Dextrose	0.5 g/kg	PRN	IV	Hyperkalemia
Sodium bicarbonate	0.3 × base deficit × BW (kg), give 1/3–1/2 of this amount	PRN <sup>a</sup>	Slow IV (15–30 minutes)	Metabolic acidosis, hyperkalemia

*Abbreviations:* BW, body weight; IV, intravenous; PRN, as needed.

<sup>a</sup> Sodium bicarbonate can result in cerebrospinal fluid acidosis and cerebral edema if given overzealously. It also lowers ionized calcium.

average-sized cat with a urethral obstruction to require a fluid rate of 40 to 50 mL/h (in a 5-kg cat, that is administration of fluids at roughly 8–10 mL/kg/h). Monitoring of urine output is invaluable in guiding changes in fluid administration rate. If the animal also has evidence of cardiac disease, fluid therapy should be administered with caution and a central venous pressure (CVP) line can also be used to guide therapy.

In animals that show hemodynamic compromise because of their acid-base and electrolyte disturbances, specific remedies are indicated in addition to fluid therapy. A number of treatment options can be used. For the hyperkalemic patient, calcium gluconate, regular insulin, and bicarbonate may all be considered.

Intravenous administration of 10% calcium gluconate at a dose of 50 to 100 mg/kg treats the cardiovascular effects of hyperkalemia and corrects the ionized hypocalcemia that is often present. Calcium does not have a direct effect on the serum potassium concentration; instead, it re-establishes a more normal difference between the resting membrane potential and the depolarization threshold in the cell, thus ameliorating the effects of the high potassium. The benefit of calcium gluconate administration is immediate [13].

Intravenous administration of regular insulin (0.1–0.25 U/kg) has a direct effect on potassium. The insulin promotes the movement of glucose intracellularly, carrying potassium with it. As the serum potassium level decreases, the resting membrane potential of the cell tends to normalize. When this drug is used, it is important to provide dextrose to the patient. In addition to administering a bolus of dextrose at the time that the insulin is given, it is advisable to supplement the intravenous fluids with dextrose, because the effects of intravenously administered regular insulin can last from 2 to 4 hours. Another approach that uses the same underlying physiology is to give just a dextrose bolus (0.5 g/kg administered intravenously, dilute 1:3); this method depends on the endogenous secretion of insulin from the patient's pancreas [13].

A final remedy for hyperkalemia is the administration of sodium bicarbonate ( $0.3 \times \text{base deficit} \times \text{body weight (kg)} = \text{total amount of bicarbonate (mEq)}$  needed to correct pH to 7.4; administer one third to one half of this amount as an intravenous bolus over 10–15 minutes). Sodium bicarbonate lowers the extracellular potassium concentration by promoting the cellular uptake of potassium in exchange for the movement of hydrogen ions out of the cell. Sodium bicarbonate also treats the metabolic acidosis but may further lower the ionized calcium. The effects of sodium bicarbonate are seen within 30 to 60 minutes and may persist for hours after administration [13].

Once the hemodynamic stability of the patient has been addressed, specific therapy to relieve the obstruction should be undertaken. This is most commonly accomplished by placing a urinary catheter. The techniques for relief of obstruction differ in the dog and cat; thus, both are discussed. The passing of a urinary catheter in an obstructed animal is often an uncomfortable procedure. If the animal is hemodynamically stable, sedation should be used (see article in this issue on anesthetic protocols for common emergencies).

Before attempting to pass the catheter, it is worthwhile to extrude the penis and visually inspect it for any mucous plugs or grit; it is sometimes possible to relieve an obstruction by gently teasing a plug out of the tip of the penis. If a plug is not seen, the catheter should be passed. In the cat, the obstruction is initially relieved using an open-end tom-cat catheter (Kendall Co., Mansfield, MA) or a Minnesota olive-tipped catheter (EJAY International, Glendora, CA). At this point, the urethra is flushed copiously with sterile saline in an attempt to break up the plug or push it back into the urinary bladder. Under no circumstances should the catheter be forced up the urethra; this can result in trauma to the urethra and may lead to iatrogenic rupture of the urethra [14]. In addition to flushing the urethra, the penis should be retracted caudad to straighten the normal flexure of the feline urethra.

Once patency has been re-established, the bladder should be copiously flushed with sterile saline. The tom-cat catheter should not be left in the bladder. Instead, it should be replaced with a soft 3.5- or 5-French red rubber catheter (Kendall Co.) that is then connected to a closed urine collection system. To minimize injury to the wall of the bladder, avoid inserting an excessive length of catheter into the bladder. An excessively long catheter placed in the bladder can also become tied into a knot and then requires surgical removal.

In the dog, retropulsion of stones is accomplished using a different technique. A red rubber or Foley catheter is introduced into the penis up to the level of the obstruction, and the tip of the penis is occluded. The pelvic urethra is digitally compressed via the rectum, and saline flush is infused into the urethra. This flush distends the urethra and “floats” the stones. When the pressure at the pelvic urethra is removed, the stones move

retrograde into the bladder. Some stones may be adhered to the bladder wall and do not float, but distention of the urethra may allow passage of the urinary catheter around the stone. At that time, a red rubber catheter can be advanced into the bladder and connected to a closed collection system.

In animals in which the obstruction cannot be relieved by catheterization, a number of alternatives may be considered. One of the first techniques that may be used is cystocentesis. Decompression of the bladder may facilitate catheterization by decreasing the pressure exerted against the urethral obstruction from the bladder side of the urethra. It is important to remember that the integrity of the bladder wall may not be normal and that urinary bladder rupture may occur, however [15]. Other options for relief of a urethral obstruction include urethrostomy, cystostomy, and voiding urohydropropulsion [16–18]. The reader is referred elsewhere for a detailed discussion of these techniques [18–20].

Once a urethral obstruction has been relieved and immediate acid-base and electrolyte issues have been addressed, the animal should be continued on intravenous fluids and the urine output should be monitored. Additional monitoring, including serial electrolytes (this may vary from every 4 hours to once daily depending on patient stability), a continuous ECG, and serial blood pressure measurements may also be appropriate. Even though an animal may have initially presented with hyperkalemia, it is not uncommon for that same animal to be hypokalemic in the days after initial stabilization. In addition, medications like phenoxybenzamine or prazosin may be started to relax the urethra and thus ameliorate urethral spasm. In the absence of bacteriuria on urinalysis, antibiotic therapy is relatively contraindicated while a urinary catheter is in place. Finally, it may be of benefit to consider pain management (see article in this issue on pain management in the emergency patient). In light of the recent insult to the renal system, it may be prudent to avoid nonsteroidal anti-inflammatory drugs (NSAIDs) and to consider other analgesic options.

### **Uroperitoneum**

Uroperitoneum occurs when the integrity of the lower urinary tract has been breached, allowing leakage of urine into the abdomen. It is important to remember that although this may be caused by rupture of the urinary bladder, damage to other areas of the urinary tract may also result in uroperitoneum. Alternatively, damage to the upper urinary tract (kidneys and ureters) may result in uroretroperitoneum but no peritoneal cavity fluid accumulation. The most common cause of uroperitoneum is trauma; however, it can also be seen with neoplasia, prolonged urinary tract obstruction, and overzealous urinary catheter placement [21].

Clinically, animals with uroperitoneum may be presented in hemodynamic collapse. They can have metabolic acidosis, chemical peritonitis, profound azotemia, hyperkalemia, hypernatremia, and hyperphosphatemia.

When urine moves into the peritoneal cavity (or the retroperitoneal cavity), a number of important effects are seen. First, the presence of a large osmotically active particle, such as creatinine, results in the movement of water into the peritoneal cavity at the expense of the intracellular and intravascular fluid. This third spacing can result in significant volume depletion and hemodynamic compromise. Although creatinine diffuses across membranes, its movement is slower than that of smaller solutes, such as potassium and urea. The rapid movement of the smaller solutes down their concentration gradient from the peritoneum to the intravascular space results in elevated concentrations of these solutes in the blood. Sodium and chloride, which normally are found in higher concentrations in the blood than in the urine, diffuse into the peritoneum, thus furthering the contraction to the intravascular fluid compartment. The dehydration that occurs in these patients further exacerbates abnormalities by decreasing the glomerular filtration rate, and thus the excretion of urea and creatinine.

The presence of sterile urine within the peritoneal space is irritating, resulting in peritonitis, whereas the presence of infected urine can cause septic peritonitis. Metabolic acidosis results not only from hypovolemia but from failure to excrete hydrogen ions from the body. Instead, hydrogen ions accumulate in the abdominal cavity, are reabsorbed into the circulation, and result in depletion of the buffering capability of the body.

#### *Physical examination and laboratory parameters*

On physical examination, these animals may show signs of hemodynamic compromise. In addition, they may have abdominal distention, abdominal pain, and a palpable fluid wave. They may also show signs of external trauma. The presence of a palpable urinary bladder does not rule out the possibility of uroperitoneum. If urine has extravasated into subcutaneous tissues, this may result in inflammation with pain and swelling in the affected area; this can be seen with a ruptured urethra, where urine leaks into the perineum and can then dissect down the hind limbs. Clinically, this area appears bruised, edematous, and painful.

The diagnosis of uroperitoneum is made by comparison of abdominal fluid creatinine and potassium with that of serum creatinine and potassium. In dogs, a ratio of abdominal fluid creatinine to serum creatinine of 2:1 and a ratio of abdominal fluid potassium to serum potassium of 1.4:1 are considered diagnostic for uroperitoneum [22]. In cats, the diagnostic ratios are 2:1 for creatinine and 1.9:1 for potassium [14,22]. The measurement of urea is of less use in making this diagnosis because it rapidly diffuses across membranes to reach equilibrium; this means that disparity between urea measurements in abdominal fluid and serum may not show clear-cut differences.

A number of other findings may provide supporting evidence. The initial database may include a PCV, total solids, venous blood gas, electrolytes,

serum chemistry, CBC, and abdominal radiographs. These data may show metabolic acidosis, azotemia, hyperkalemia, and evidence of volume contraction. On survey radiographs, there is a loss of abdominal detail. Positive-contrast radiography is the study of choice for determining the site of urine leakage. Excretory urography is helpful in identifying leaks in the kidneys and ureters, whereas a retrourethrocytogram is helpful in identifying leaks in the urethra and urinary bladder.

### *Treatment*

Treatment of uroperitoneum involves the correction of acid-base and electrolyte disturbances as well as definitive treatment of the rupture. As with urethral obstruction, the mainstay of therapy is fluids, with definitive therapy of hyperkalemia warranted based on clinical signs. In addition to these interventions, drainage of the abdominal cavity is important. This can be achieved by placement of a peritoneal dialysis catheter into the abdomen. Peritoneal dialysis is an excellent technique not only for the drainage of urine but for the resolution of acid-base abnormalities, azotemia, and electrolyte disturbances [23]. Placement of a urinary catheter into the bladder is of benefit in keeping the bladder decompressed.

After the metabolic disturbances have been stabilized, definitive repair of the rupture must be considered. This commonly is achieved by surgical repair, although in certain instances, such as iatrogenic bladder rupture, medical management involving decompression of the urinary bladder with an indwelling urinary catheter has been advocated [24]. The reader is referred elsewhere for a detailed discussion of surgical techniques [19,20,25].

### **Acute renal failure**

Receiving 20% to 25% of the total blood flow in the body, the kidney is a major route for the excretion of waste products as well as the maintenance of water and electrolyte balance. Although this abundant blood flow is needed to maintain adequate renal function, it also makes the kidney especially sensitive to changes in blood flow as well as blood-borne toxins. The distribution of blood flow within the kidney is not uniform; instead, most blood flow goes to the renal cortex, whereas less than 10% of all the blood flow that the kidney receives goes to the renal medulla. This is approximately the same amount of blood as is received by resting skeletal muscle [26]. The abundance of blood flow that the kidney receives makes it especially vulnerable to the effects of toxins, and the disparity of blood flow within the kidney makes the renal medulla susceptible to ischemic insult and the cortex susceptible to toxins.

Autoregulation allows the kidney to maintain a relatively constant renal blood flow and glomerular filtration rate (GFR) over mean arterial blood



pressures ranging from 70 to 170 mm Hg. Without autoregulation, relatively small increases in blood pressure would result in a marked increase in GFR and urine production. Conversely, at less than a mean arterial pressure of 70 mm Hg, renal blood flow and GFR decline in direct relation to decreases in mean arterial pressure. Thus, decreases in urine production in the hypotensive patient may reflect the effect of decreased renal blood flow and are not always evidence of oliguric or anuric renal failure.

### *Pathophysiology of acute renal failure*

Acute renal failure is most commonly caused by an ischemic event or a bloodborne toxicant. Other conditions that can result in acute renal failure include immune-mediated disease, pyelonephritis, leptospirosis, hypercalcemia, and urinary tract obstruction.

The kidney is especially vulnerable to toxicant injury because of its abundant blood supply, whereas the processes of tubular secretion, countercurrent multiplication, and reabsorption can concentrate toxicants even further. Because the cortex of the kidney receives most renal blood flow, it is the prime target of intoxicant-induced injury. A variety of possible compounds can result in acute renal failure, including aminoglycosides, metals, snakebite and bee venoms, myoglobin, hemoglobin, certain chemotherapeutic agents, radiographic contrast agents, and ethylene glycol [27–31]. Recently, acute renal failure has been associated with lily ingestion in cats and with ingestion of raisins and grapes in dogs [32,33]. A number of different types of lilies have been implicated, including the Tiger lily, day lily, Easter lily, Stargazer lily, and Asiatic hybrid lily, but the underlying toxic insult remains unknown [32,34]. The toxic insult is also unclear in raisin and grape ingestion; however, the amount needed to cause acute renal failure is alarmingly small (0.32–0.65 oz/kg in one report [35] and 0.41–1.1 oz/kg in another report [33]).

When the systemic blood pressure drops to less than 70 mm Hg, renal blood flow decreases in a linear fashion with blood pressure. As the oxygen and energy supply to the kidney are compromised, cellular ATP and energy become depleted and cell membrane pumps fail, resulting in cell swelling because of the intracellular accumulation of sodium and water. As calcium accumulates within the cell, a number of cellular processes, including oxidative phosphorylation within the mitochondria, are deranged [36,37]. A loss of renal autoregulation occurs, which is thought to be caused by increased responsiveness to renal nerve stimulation, which, in turn, has been related to increases in intracellular calcium in the afferent arteriole cells of the glomerulus [38,39]. Cell swelling and vasoconstriction contribute to vascular stasis and predispose to the formation of microthrombi, which occlude renal vessels and further contribute to renal ischemia. The decrease of energy substrates also predisposes to the formation of oxygen-free radical species, which results in further cellular damage [36,37].

A number of events and conditions can result in renal ischemia, including shock, anesthetic-induced vasodilation and hypotension, hyperthermia, hypothermia, extensive cutaneous burns, NSAID administration, hyperviscosity/polycythemia, and disseminated intravascular coagulation (DIC) [36].

### *Clinical signs*

The clinical signs of acute renal failure are nonspecific and include lethargy, depression, anorexia, vomiting, and diarrhea. Because the disease course of acute renal failure is brief, these animals frequently do not have weight loss. Animals with acute renal failure may be polyuric and polydipsic, but they can also be anuric or oliguric. Physical examination may reveal large painful kidneys. Laboratory data show elevations in BUN and creatinine and may show elevations in potassium and phosphorus. Calcium can be increased or decreased. Animals in acute renal failure frequently have a marked metabolic acidosis, and the CBC may show a stress leukogram. Urinalysis reveals isosthenuric urine and may show proteinuria, hematuria, glucosuria, and the presence of granular casts, which may indicate acute tubular injury [37]. The fractional excretion of sodium

$$Fe_{Na} = \frac{Urine_{Na}}{Plasma_{Na}} \times \frac{Plasma_{Creatinine}}{Urine_{Creatinine}} \times 100\%$$

in the urine may also be increased in acute renal failure; this is indicative of a significant tubular insult, because a normal animal with prerenal azotemia should be able to reabsorb more than 99% of the filtered load of urine sodium [37]. In animals with other underlying conditions that favor sodium reabsorption (eg, congestive heart failure, hepatic failure, nephritic syndrome), the fractional excretion of sodium may be normal despite tubular dysfunction [37]. Another test that may be helpful in the diagnosis of acute renal failure is carbamylated hemoglobin measurement. Carbamylated hemoglobin is the product of a reaction between hemoglobin within the red blood cells and cyanate, which equilibrates with urea in the blood. As the red blood ages, carbamylated hemoglobin accumulates and can be measured to help differentiate acute from chronic renal failure, because chronic renal failure can be expected to result in a higher carbamylated hemoglobin concentration [40,41].

### *Therapy*

One of the main components in the treatment of acute renal failure is fluid therapy. The goal of initial fluid therapy is to correct hemodynamic abnormalities, expand the vascular volume, and correct electrolyte abnormalities. A balanced electrolyte solution is appropriate for achieving

this goal. After volume expansion, fluid therapy should be continued at a rate that facilitates diuresis; usually, a rate around 5 to 6 mL/kg/h is a good starting point. Once volume replete, a crystalloid that provides free water, such as 0.45% NaCl, may be indicated. Many of the crystalloid solutions do not provide free water and therefore can contribute to an iatrogenic hyponatremia if the animal has no other source of water intake. It is extremely helpful to monitor urine output when trying to decide on the correct fluid rate. Once an animal is volume replete and normotensive, urine output should be at least 1 to 2 mL/kg/h. Daily monitoring of the patient can provide valuable information for guiding fluid therapy as well; this includes urine output, body weight, hemodynamic parameters, and serial electrolytes. If fluid therapy is not adequate, the patient may show weight loss as well as an increase in serum sodium. By contrast, an increase in body weight may be a result of the correction of volume deficits. Urine output in the polyuric renal failure patient can be voluminous, and aggressive fluid administration may be needed to stay abreast of fluid loss. By contrast, if a patient is anuric or oliguric, overzealous fluid administration can result in volume overload, pulmonary edema, and death. CVP monitoring is invaluable for the monitoring of volume status. A consistent increase in CVP is indicative of impending fluid overload. CVP can be easily done in practice but does require the placement of a central line. Central lines are also of value in the acquisition of serial blood samples as well as allowing for the administration of hypertonic solutions, including total parenteral nutrition.

Other therapies in acute renal failure are targeted at alleviating the clinical signs and underlying causes of the acute renal failure. The most common bacterial isolates from the urinary tract in dogs include *Escherichia coli* (44.1%), *Staphylococcus* spp (11.6%), *Proteus* spp (9.3%), *Klebsiella* spp (9.1%), *Enterococcus* spp (8.0%), and *Streptococcus* spp (5.4%) [42,43]; thus, reasonable antibiotic choices in pyelonephritis could include ampicillin (22 mg/kg administered intravenously three times daily) and enrofloxacin (5 mg/kg administered intravenously once daily). Because renal failure is frequently associated with increased gastric acid secretion and vomiting, drugs like ranitidine (2 mg/kg administered intravenously three times daily), famotidine (0.5 mg/kg administered intravenously twice daily), and sucralfate (0.5–1 g administered orally two to four times daily) can all be helpful. Antiemetics like metoclopramide (continuous rate infusion [CRI] at 1 mg/kg per 24 hours administered intravenously) and chlorpromazine (0.2–0.5 mg/kg administered intramuscularly) can also be used. Chlorpromazine can induce hypotension and therefore should not be used in a volume-depleted or already hypotensive patient. It is also important to address nutrition in these critical patients. If possible, enteral nutrition is desirable, but parenteral nutrition can be used if needed.

When faced with an oliguric or anuric acute renal failure patient, a number of therapeutic interventions can be used (Table 2), but extrarenal causes, such as hypovolemia and hypotension, must first be ruled out. Until

Table 2  
Interventions in the oliguric/anuric patient

Drug	Dose range	Frequency	Route
Isotonic crystalloid	10–20 mL/kg over 15–20 minutes	PRN	IV
Furosemide	2–6 mg/kg	Q6–8h	IV
Mannitol	0.5–1.0 g/kg over 20 minutes	Q6h	IV
Dextrose	25–50 mL/kg of 10%–25% solution over 1–2 h	Q8–12h	IV

*Abbreviations:* h, hours; IV, intravenous; PRN, as needed; Q, every.

the animal has a blood pressure higher than 70 mm Hg and is volume expanded, aggressive therapy for anuric acute renal failure should not be pursued; instead, all efforts should be focused on normalizing blood pressure and volume status. The first therapy in the euvolemic, normotensive, anuric patient is usually fluid boluses. If an animal is volume overloaded already, a fluid bolus is contraindicated. A reasonable bolus would be isotonic crystalloid at a dose of 10 to 20 mL/kg over 15 to 20 minutes. There should be an increase in urine output within 30 to 60 minutes. Intravenous boluses of furosemide can also be given. Furosemide has been shown to exacerbate gentamicin toxicity; thus, it should not be used in cases of aminoglycoside-induced acute renal failure [37].

If urine output is still inadequate after fluid boluses and furosemide administration and the blood pressure is adequate for the production of urine, an osmotic diuretic can be tried. Osmotic diuretics should be used with extreme caution in animals with possible fluid overload. The main osmotic diuretic used is mannitol. Mannitol is an excellent osmotic diuretic and has some free radical scavenging capabilities. Mannitol is also a weak vasodilator. Mannitol is not metabolized; it is filtered at the glomerulus and excreted from the body. Therefore, if there is no GFR, mannitol remains in the vascular space and may contribute to volume overload [43]. Because of this concern, hypertonic glucose is sometimes used in place of mannitol. The advantage of hypertonic glucose is that it can be metabolized by the body; however, it does not possess the free radical scavenging abilities of mannitol and is less effective as an osmotic agent [43].

The final drug that is sometimes used in oliguric or anuric renal failure is dopamine. The use of dopamine in acute renal failure is a controversial topic. Classically, low-dose dopamine is thought to act on renal dopamine receptors to increase renal blood flow. Evidence has shown that the cat does not possess renal dopamine receptors of the same type as other species [44,45]; however, a recent study has identified a unique dopamine receptor in the feline kidney [46]. How much of a contribution this receptor makes in hemodynamic regulation of the feline kidney requires further research. In human medicine, dopamine has begun to fall out of favor as more studies have shown that dopamine does not prevent or reverse acute renal failure and does not improve patient outcome [47,48]. The author does not use dopamine at a “kidney” dose in acute renal failure.

Peritoneal dialysis and hemodialysis should also be considered in the acute renal failure patient [23,49,50]. This is especially true of the anuric acute renal failure patient, but the decision to pursue dialysis should not be made in the eleventh hour. Peritoneal dialysis is a viable technique for the private practice setting, but it is labor-intensive and can result in complications, including septic peritonitis and electrolyte imbalances [23]. Hemodialysis, although still fairly limited in veterinary medicine because of its lack of availability as well as expense, is being more widely used than ever before and is an excellent option for acute renal failure patients.

### *New directions*

Research is constantly being conducted to find better treatment options for acute renal failure. On the horizon are calcium channel blockers. In the human literature, the use of calcium channel blockers, such as diltiazem, for the prevention and treatment of acute renal failure is a topic of debate, but some evidence does support their use in acute renal failure [51,52]. In veterinary medicine, the use of diltiazem in the treatment of acute renal failure is also under investigation, but not enough evidence is present at this time to warrant its routine use in acute renal failure (Karol Mathews, DVM, DVSc, personal communication, 2004).

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