Managing acute renal failure (ARF) begins with preventing it with fluid support and appropriate monitoring of critically ill patients, animals undergoing general anesthesia, and other at-risk patients. For patients that present with acute onset of severe azotemia, rapidly implementing an appropriate diagnostic scheme and effective therapeutic plan is a crucial determinant of patient prognosis.

Azotemia can be defined as increased serum blood urea nitrogen (BUN), creatinine, or other nitrogenous waste products. Azotemia may be categorized according to prerenal, renal, and postrenal causes. Prerenal azotemia occurs secondary to a decreased glomerular filtration rate (GFR) and is accompanied by concentrated urine. It is most commonly associated with dehydration, although it may be associated with profound hypovolemia or hypotension. It should resolve with adequate fluid support and correction of the primary disease process.¹

Postrenal azotemia results from blockage of the urinary system downstream from the kidneys. The most common causes of postrenal azotemia are urinary obstruction (i.e., ureteroliths, cystic calculi, urethral calculi) or urinary tract rupture.¹ Postrenal azotemia should resolve once the obstruction has been cleared unless it has been long-standing enough to cause secondary tubular damage. Accurately monitoring urinary output (UOP) after correcting the obstruction is critical in detecting postobstructive diuresis, in which UOP may easily exceed 7 ml/kg/hr. If UOP is not adequately monitored, fluid supplementation may not meet demand and animals may become dehydrated and develop prerenal azotemia.

True parenchymal renal failure is characterized by the presence of azotemia with isosthenuria. Common causes of parenchymal renal failure include toxins, infection, and ischemic insults.² Toxic agents include ethylene glycol, lilies, grapes and raisins, aminoglycosides, NSAIDs, contrast agents, anesthetics, and other therapeutic medications. When prescribing potential nephrotoxicants, practitioners should ensure that animals are adequately hydrated before administering medications, review conditions that may increase risk (e.g., hypokalemia, concomitant diuretic use), and

ABSTRACT:
Acute renal failure (ARF) is a serious disease, and affected patients have a guarded prognosis. The best success in managing the disease is in preventing it whenever possible. Treatment of spontaneous ARF should be aimed at eliminating ongoing damage and supporting patients until the renal parenchyma can regenerate. Specific therapeutic aims include correction of fluid imbalances and electrolyte derangements, provision of adequate nutrition, and symptomatic supportive care to control ulceration and vomiting. Successful outcome depends on early, aggressive treatment and supportive care.
implement appropriate monitoring for early signs of renal toxicosis (e.g., regular urine sediment analyses for casts, urine γ-glutamyl transferase:creatinine and N-acetyl-β-D-glucosaminidase:creatinine ratios, biochemical profiles). Infectious causes include leptospirosis, babesiosis (especially Babesia microti–like infections), and other sources of pyelonephritis. Parenchymal ischemia may occur secondary to hypotension, hypovolemia, sepsis, or multiple-organ dysfunction, as may be seen with acute pancreatitis or severe trauma. Electrolyte derangements and acid–base abnormalities may occur with all three forms of azotemia and cannot be used to distinguish between the forms. In addition, it is often difficult or impossible initially to differentiate ARF from acute exacerbation of chronic renal disease in patients for which previous blood work and urinalyses are not available. This is especially true in animals that do not present with small kidneys or anemia. Differentiation may require careful evaluation of the signalment, history, physical examination findings, abdominal radiographs, abdominal ultrasonogram, and therapeutic response. In some cases, renal biopsy may be helpful to distinguish between these conditions, underlying causes, and associated prognoses.

Once a diagnosis of ARF is suspected, it is important to provide appropriate monitoring and support to achieve a successful outcome. During hospital admission, samples should be collected and submitted for a complete blood count, a biochemical profile, a complete urinalysis, and an aerobic urine culture. If available, a venous blood gas analysis should be conducted to help manage acid–base disturbances. If leptospirosis is suspected, pretreatment serum and urine samples should be collected for titers and polymerase chain reaction testing, respectively; in addition, patients and urine should be handled with gloves to limit the risk of zoonosis (see Leptospirosis section). Collecting blood to measure ionized calcium, osmolality, and ethylene glycol may greatly aid diagnosis of ethylene glycol intoxication. If toxicosis is suspected, it is important to discontinue the putative toxin. In cases of acute intoxication, inducing emesis (if the animal is alert enough to protect its airway and is not already vomiting), gastric lavage, and/or administering activated charcoal are indicated. Cathartics should
be used with caution because many commonly used cathartics stimulate osmotic diarrhea, which may worsen dehydration.

### MONITORING AND INSTRUMENTATION

Although fluid therapy is key in treating ARF, the risk of oliguria or anuria makes adequate monitoring of hemodynamic parameters and UOP critical in preventing fluid overload. Placing a jugular catheter is essential in monitoring central venous pressure (CVP; Figure 1). CVP can be easily measured using a fluid extension line and ruler if a clinic does not have commercial manometers. A soft urinary catheter, preferably a Foley, with a closed urinary collection system should be placed (with sterile technique) and used to limit trauma and infection from prolonged catheter placement. Once a catheter has been placed, the bladder should be drained so that UOP can be accurately monitored. Baseline determinations of CVP, arterial blood pressure (BP), body weight ($BW_{kg}$), and temperature as well as an estimate of dehydration should be recorded. In addition, a fundic examination should be conducted to detect retinal lesions suggestive of historical or current severe hypertension. Measurements should be repeated frequently because patient condition may change radically in short periods (Table 1). Once these data have been recorded, fluid deficits should be replaced over the first few hours of treatment using a fluid that best mimics the deficit.

### INITIAL FLUID REPLACEMENT

Adequate rehydration is critical in managing ARF for a variety of reasons. Most animals with ARF have a component of prerenal azotemia in addition to renal azotemia. Because of the loss of renal-concentrating ability, the degree of prerenal azotemia can be determined only by the amount of improvement in azotemia with rehydration. Because persistent dehydration prolongs renal hypoxia and increases tubular cell death, restoring a normal circulating volume is essential.

Animals that are severely hypovolemic may benefit from an initial fluid bolus (i.e., 45 to 90 ml/kg divided into quarters and administered until resolution of signs of hypovolemia and shock). Isotonic polyionic fluids, such as 0.9% saline solution and Normosol-R (Abbott Laboratories), are usually recommended for initial replacement. Saline is often a first-line therapy in patients with hyperkalemia, but special attention to pH is indicated because saline may worsen acidosis. Normokalemic or hypokalemic animals may benefit more from Normosol-R because of its alkalinizing effects. Lactated Ringer’s solution may also provide adequate acid–base buffering in cases without concurrent hepatic dysfunction. Initial replacement goals should be calculated as follows:

$$\text{Replacement volume (ml)} = BW_{kg} \times \frac{\text{Estimated} \% \text{ dehydration}}{1,000}$$

It is important, even during initial fluid rehydration, that CVPs be monitored for circulatory overload. This is especially true in patients with circulatory compromise or myocardial disease. Once a patient has been adequately rehydrated—based on skin turgor, heart and respiratory rates, BP, hematocrit, total protein level, and CVP—the next step is to reevaluate UOP. It is important to remember that adequate rehydration may take 2 hours to 2 days depending on a patient’s condition at presentation. Therefore, it is essential to vigilantly recheck patients and relevant laboratory parameters to avoid both over- and undertreatment.

### URINE OUTPUT AND DIURESIS

Once a patient is fluid replete, the goals of fluid therapy are to promote mild volume expansion and reestablish adequate GFR. Once GFR has been restored to an appropriate level, UOP should be at least 1 to 2 ml/kg/hr. If UOP remains below this level, the patient is either inadequately rehydrated or suffering from oliguric or anuric renal disease. Fluid administration may be continued to

### Table 1. Recommended Schedule for Patient Evaluation

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Minimum Frequency</th>
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<tbody>
<tr>
<td>Body weight</td>
<td>Two or three times per day</td>
</tr>
<tr>
<td>Temperature</td>
<td></td>
</tr>
<tr>
<td>BP</td>
<td></td>
</tr>
<tr>
<td>Heart and respiratory rates</td>
<td>Three times per day</td>
</tr>
<tr>
<td>UOP</td>
<td>Four times per day</td>
</tr>
<tr>
<td>Urine specific gravity</td>
<td></td>
</tr>
<tr>
<td>CVP</td>
<td></td>
</tr>
<tr>
<td>Venous blood gas</td>
<td>One or two times per day</td>
</tr>
<tr>
<td>Electrolyte levels</td>
<td></td>
</tr>
<tr>
<td>Packed cell volume</td>
<td></td>
</tr>
<tr>
<td>Total solids</td>
<td></td>
</tr>
<tr>
<td>Biochemical profile</td>
<td>Every other day</td>
</tr>
<tr>
<td>Urinalysis with sediment evaluation</td>
<td></td>
</tr>
</tbody>
</table>
correct residual dehydration until CVP has increased to 8 to 10 cm H₂O (or 5 to 8 cm H₂O in animals with cardiovascular compromise). At this point, fluid administration should be adjusted to match ongoing sensible and insensible losses (see box on this page), and other approaches (discussed later) must be tried to reestablish appropriate UOP (i.e., >1 to 2 ml/kg/hr). If CVP increases to greater than 10 cm H₂O without a significant increase in UOP, the patient is in danger of fluid overload.

Ongoing fluid administration should be based on the type of fluid required or lost. Insensible losses should be replaced with half-strength saline (0.45% sodium chloride with or without 2.5% dextrose) or 5% dextrose to avoid development of iatrogenic hypernatremia and hyperchloremia. Although these complications occur uncommonly in patients that are drinking on their own, it is not uncommon in those given nothing by mouth to help control vomiting. Animals with hypoproteinemia may require fluid support with synthetic colloids or plasma. Monitoring colloid osmotic pressure is the most accurate way to assess ongoing colloid needs because synthetic colloids do not contribute to total protein measurements. Animals with clinical signs of anemia may require support with oxygen-carrying products (e.g., Oxyglobin, Biopure) or blood transfusions. When considering the use of colloids or blood products, it is essential to check the patient’s BP and CVP because these products may significantly increase those parameters. In patients with hypertension or elevated CVP, use of such products is often contraindicated.

**DIURETICS**

If fluid therapy has not restored UOP, diuretics may be administered. Interestingly, converting oliguric or anuric ARF to a polyuric state is not associated with improved outcome but does facilitate patient management.6,7 Diuretics most commonly administered to reestablish UOP are mannitol, furosemide, and dopamine. These drugs may be administered serially or in combination, and this decision should be made on a case-by-case basis using clinical judgment and experience.

Mannitol is believed to be the most effective choice for initiating diuresis, but it is contraindicated in fluid-overloaded or hyperosmolar patients (e.g., those with ethylene glycol intoxication, uncontrolled diabetes, or hypernatremia). Mannitol is an osmotic diuretic and is administered as a slow intravenous bolus (i.e., 0.5 to 1 g/kg). If a positive response is not seen within 60 minutes, further use is contraindicated because it may worsen fluid overload and pulmonary edema. If significant diuresis is achieved, mannitol may be administered via constant-rate infusion (CRI; 1 to 2 mg/kg/min) for the next 24 to 48 hours.

Dopamine, a catecholamine and epinephrine precursor, can be administered at a low dose (i.e., 1 to 3 µg/kg/min IV) to stimulate dopaminergic receptors, thus improving renal perfusion. This dose is lower than the conventional renal dose because of recent recognition that sick animals may have greater sensitivity to nondopaminergic effects than did the healthy animals used to establish the original renal dose.8 At the dose just cited, dopamine has little effect on β and α receptors, systemic vascular resistance, or blood pressure. It is important to note that at higher doses, β and then α effects dominate, leading to increased inotropic effects, systemic vascular resistance, and decreased renal perfusion.

### Fluid Therapy Calculation for Fluid-Replete Patients

The goal of fluid therapy for fluid-replete patients is to match the animal’s needs. Insensible losses include moisture lost through the respiratory and cutaneous systems. In noncritically ill animals, water loss in feces is often considered part of insensible losses. Because animals with renal failure often develop GI derangements, we recommend that GI losses be included when calculating sensible losses.

<table>
<thead>
<tr>
<th>Insensible losses (ml/hr)</th>
<th>= (20 × BW&lt;sub&gt;kg&lt;/sub&gt;) ÷ 24</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensible losses (ml/hr)</td>
<td>= (UOP + GI losses) ÷ Hr since last quantitation</td>
</tr>
<tr>
<td>Fluid rate (ml/hr)</td>
<td>= Insensible + Sensible rates</td>
</tr>
</tbody>
</table>

**Fluid therapy for adequate rehydration, mild volume expansion, and reestablishment of GFR is critical in treating ARF.**
There is significant controversy regarding continued use of dopamine in humans with ARF. Human studies have shown limited to no improvement in morbidity, mortality, or need for dialysis in established ARF cases. Based on these data, dopamine is not currently recommended in treating ARF in humans. A few studies have shown that the combination of dopamine and furosemide may confer benefits in treating certain causes of ARF in humans. Because the applicability of these results to veterinary medicine has been debated, it is worthwhile to expand on potential pitfalls of such an application.

ARF in humans most commonly occurs secondary to severe hypovolemic events or use of radiocontrast materials (probably secondary to severe medullary ischemia). In dogs, a higher proportion of cases occurs secondary to primary disease processes or exogenous toxicants unrelated to medullary ischemia. In addition, dopaminergic receptors are primarily located in the renal vasculature in dogs, whereas receptors are more heavily concentrated on renal tubular cells in humans. The increased presence of dopaminergic receptors on tubular cells versus the concentration on vascular cells in humans may account for the decrease in positive dopamine-associated renal blood flow changes compared with those in dogs. In fact, because humans do not benefit as strongly from renal dilation effects, they may be more likely to develop increased renal hypoxia and damage. In humans, natriuresis and chloride release stimulated by dopamine may lead to increased medullary oxygen consumption and tubular workload in addition to increased tubular flow-through and washout of casts and necrotic cells. In short, there are several significant limitations in using humans as a model for canine ARF; randomized controlled trials in dogs are necessary to resolve the ongoing debate.

In our experience, dopamine administration has been associated with significant increases in UOP. Differences in receptor activity in cats lead to poor renal effects at safe doses; thus dopamine administration is not recommended.

Furosemide, a loop diuretic, should be administered alone or with a CRI of dopamine. Because furosemide acts on Henle’s loop, it is potassium-wasting and may facilitate correction of hyperkalemia. Furosemide is usually administered as an intravenous bolus (2 to 4 mg/kg) and is repeated every 6 to 8 hours if it successfully stimulates diuresis. Alternatively, after initial administration, furosemide may be administered via CRI (0.1 to 1 mg/kg/hr) because some publications have suggested that greater diuresis relative to the total furosemide dose may be achieved via CRI. These studies were in healthy dogs, but research in humans and horses has also supported improved diuresis and kaliuresis via CRI. Syringe pumps, additional fluid bags, or buretrol dosing may be useful for dopamine and furosemide CRI. Recent work has documented good short-term stability of furosemide in crystalloid solutions.

Recent work at Ontario Veterinary College suggests that diltiazem, a calcium-channel blocker, may be useful in stimulating diuresis in dogs. Diltiazem has anti-endothelin effects that lead to decreased renal vasoconstriction. In addition, diltiazem may decrease renal vascular tone and tubular cell death, thereby decreasing cast formation and tubular blockage. The recommended protocol is 0.3 to 0.5 mg/kg slow IV bolus followed by 1 to 5 µg/kg/min CRI. Because diltiazem may have antipressor effects, it is important to carefully monitor BP when initiating diltiazem treatment. Possible mitigating effects on hypertension may be an added benefit of diltiazem administration in some patients. It is also important to monitor heart rate to avoid cardiovascular compromise.

If pharmacologic intervention does not stimulate diuresis or overhydration occurs, peritoneal dialysis or hemodialysis should be pursued without delay.

**pH ALTERATIONS**

Acidosis occurs commonly with ARF because of production of metabolic acids, decreased filtration and excretion of acids, and decreased resorption of bicarbonate ($\text{HCO}_3^-$). Mild acidosis can usually be corrected with fluid therapy alone. More severe acidosis (pH < 7.2 or $\text{HCO}_3^- < 16$ mEq/L) may need to be directly addressed if fluid rehydration does not normalize pH and
HCO$_3^-$ abnormalities. Using pH to calculate the base deficit is preferable to using HCO$_3^-$, which may underestimate the degree of acidosis:

\[
\text{Bicarbonate dose (mEq)} = \frac{\text{BW}_{\text{kg}} \times \text{HCO}_3^- \text{ deficit}}{(\text{HCO}_3^- \text{ ideal} - \times \text{V}_d)}
\]

In this equation, \(V_d\) is the volume of distribution. This is a dynamic volume that changes rapidly in response to the HCO$_3^-$ concentration and with HCO$_3^-$ administration. We recommend using a conservative factor for \(V_d\) (i.e., 0.4 to 0.5) because overcorrection is clinically more problematic than undercorrection. The HCO$_3^-$ replacement dose should be administered in serial slow boluses over several hours, and HCO$_3^-$ should be measured frequently until it normalizes. Some animals require ongoing HCO$_3^-$ supplementation. Because of the risks associated with excessive HCO$_3^-$ administration and alkalosis, HCO$_3^-$ should be administered judiciously and with appropriate attention to monitoring.

**ELECTROLYTE ABNORMALITIES**

Animals with ARF often initially have hyperkalemia from decreased glomerular elimination of potassium and increased extracellular movement of potassium. Extracellular potassium movement occurs during acidosis as compensation for intracellular movement of hydrogen ions to decrease circulating acidemia. As acidosis is corrected, hydrogen ions shift extracellularly, facilitating return of potassium to the intracellular space. This may lead to normokalemia or hypokalemia, depending on renal potassium clearance. Oliguric or anuric animals may have persistent hyperkalemia because of inadequate renal potassium elimination. Because potassium may change dramatically after rehydration and normalization of acid–base disturbances, serial monitoring of potassium is important to guide potassium supplementation and fluid choice. In addition, unless pathologic changes from hyperkalemia (e.g., abnormalities on an electrocardiogram, bradycardia) are evident, attempts to correct potassium levels are not warranted until rehydration has been achieved.

Hyperphosphatemia secondary to poor renal clearance is common and can lead to pathologic mineralization of soft tissues when the calcium $\times$ phosphorus product exceeds approximately 70. Intervention is directed at controlling the calcium $\times$ phosphorus product by decreasing intestinal absorption of phosphorus by using phosphate binders administered with food and feeding phosphorus-restricted foods. Given the importance of providing adequate nutritional support and the difficulty in getting animals with ARF to eat, it is usually best to wait to attempt phosphorus reduction until the animal is out of crisis and eating well on its own.

Calcium abnormalities are very common and difficult to interpret or treat. Total and ionized calcium are variably increased or decreased and may change rapidly with treatment of acidosis and dehydration. Calcium alterations are rarely clinically important, and treatment is not recommended unless clinical signs of hypocalcemia develop.$^{20}$ Hypermagnesemia also commonly occurs with ARF as a result of decreased renal excretion. Magnesium abnormalities, like calcium abnormalities, do not generally require direct treatment.$^{21,22}$

**GASTROINTESTINAL DISTURBANCES**

Animals with ARF may develop a number of gastrointestinal (GI) disturbances. Vomiting may occur secondary to the effect of profound uremia on the chemoreceptor trigger zone. Because vomiting may stimulate excessive vagal tone, causing profound bradycardia and, potentially, “vomit and die” syndrome, administering appropriate antiemetics is essential.

Metoclopramide is commonly used as a first-line antiemetic. Metoclopramide acts directly on dopaminergic receptors in the brain as well as increases lower esophageal sphincter tone and enhances intestinal motility. Efficacy appears to increase when metoclo-
Treating Acute Renal Failure

Figure 2. Fluid administration.

Concurrent fluid administration using two bags to maintain a steady infusion of metoclopramide while allowing adjustment of the total fluid rate.

Labels like this can be easily made using a computer spreadsheet program. The animal's weight can be entered and doses automatically calculated and placed on the label for printing.

Metoclopramide is administered via CRI; the CRI half-life is very short, and animals may begin vomiting within minutes of stopping therapy. It is recommended that metoclopramide be administered via syringe pump so that the dose can be adjusted separately from that of fluids. If metoclopramide must be added to fluids, two fluid bags should be used so that the bag containing metoclopramide can be administered at a CRI and the fluid volume adjusted using the second bag (Figure 2). Because metoclopramide is light sensitive, fluid lines and bags should be shielded from light.

If metoclopramide does not provide adequate control, alternatives include the newer antiemetics ondansetron (0.1 mg/kg slow IV bid or tid) and dolasetron (0.6 to 3 mg/kg IV q24h); these dosages are for dogs. Cost may be problematic for administering these drugs in larger animals. The most efficacious antiemetic is chlorpromazine, which is also light sensitive and may be associated with sedative effects.

Oral ulceration may occur secondary to uremic vasculitis and requires symptomatic control with saline or dilute chlorhexidine solution washes. A mouthwash containing equal parts of a magnesium and aluminum hydroxide–containing solution (e.g., antacids), diphenhydramine, and lidocaine viscous may be administered to alleviate discomfort.

GI ulceration occurs as a result of uremic vasculitis and circulating hypergastrinemia due to decreased GFR. Administering H₂ blockers limits acid secretion from hypergastrinemia. Famotidine and ranitidine are the most commonly used H₂ blockers; the dose should be decreased by one-third to one-half to compensate for decreased renal clearance. The most effective medications for limiting intestinal ulceration and esophagitis are proton pump inhibitors such as omeprazole, but their use is limited by the need for oral administration. Parenteral proton pump inhibitors such as pantoprazole may be obtained from human hospitals. There are currently no published refereed papers on veterinary pantoprazole use, but extrapolated dosages include 10 to 40 mg/dog IV q24h or 0.7 to 1 mg/kg IV q24h.

Animals that tolerate oral medications may also benefit from sucralfate administration. Sucralfate should be crushed and administered as a slurry to facilitate binding to ulcers in the esophagus and stomach. When using sucralfate, it is important to remember that it will bind to a number of commonly administered medications and decrease their absorption. Administration should be temporally spaced from that of other oral drugs.

HYPERTENSION

Systemic hypertension is a common complication in cases of acute and acute-on-chronic renal failure. Persistent hypertension can lead to worsening of nephron loss and glomerular hypertension. Treatment can be com-
complicated by the limited availability of safe parenteral antihypertensives. In cases of severe hypertension, particularly if vomiting is persistent, nitroprusside therapy should be considered. Once vomiting has been controlled, amlodipine and angiotensin-converting enzyme (ACE) inhibitors may be administered to control systemic and glomerular hypertension. When ACE-inhibitor therapy is initiated, renal values should be closely monitored because the drugs may exacerbate renal disease. In humans, a 10% increase in creatinine is anticipated when initiating ACE-inhibitor therapy; increases above that are associated with a negative renal effect. It is also important to lower doses and/or dosing intervals because ACE inhibitors are primarily cleared by the kidneys; renal dysfunction is associated with increased ACE inhibitor half-lives. These effects may be decreased with benazepril, which is partially cleared by biliary excretion; biliary excretion has been shown to increase in animals with decreased renal clearance. Aciero and Labato recently wrote a detailed review on treating hypertension.

NUTRITIONAL SUPPORT

Nutritional intervention may be required in animals with persistent anorexia or vomiting. Animals should be encouraged to eat as long as they are not vomiting; dietary modification is usually not appropriate until patients are eating well. Force-feeding is unlikely to be beneficial because it may accelerate development of food aversion and is rarely associated with significant caloric intake. Options for nutritional support in anorectic patients include using feeding tubes or parenteral nutrition. Enteral nutrition is preferred given the decreased relative cost and maintenance of increased enterocyte health. Nasoesophageal and esophagostomy feeding tubes can be easily placed and are well-tolerated in dogs and cats, respectively, but their use is limited in vomiting patients. Gastrostomy tubes are very effective, although placing them requires longer anesthesia than is needed for esophageal feeding tubes. Placing a gastrostomy or jejunostomy tube should be seriously considered in patients undergoing anesthesia for renal biopsy and/or peritoneal dialysis catheter placement. Jejunostomy tubes may be used in animals that are vomiting.

Parenteral nutrition is recommended in animals that are too unstable for anesthesia or tube placement or those with intractable vomiting. Parenteral nutrition should be seriously considered in patients that have remained anorectic or markedly inappetent for more than 2 to 3 days or those with refractory vomiting. Parenteral nutrition requires a devoted central catheter or catheter port to avoid risk of serious infection.

ANTIBIOTICS

Antibiotic therapy is indicated in patients with pyelonephritis or those in which leptospirosis is suspected. Four-quadrant antibiotic therapy (e.g., ampicillin or enrofloxacin) is rarely warranted in cases of ARF. When possible, antibiotic therapy should be selected based on culture and sensitivity of urine collected during admission to the hospital. Prolonged urinary catheter placement is associated with increased risk of nosocomial cystitis. Prophylactic antibiotic administration to prevent development of nosocomial cystitis is inappropriate because it is ineffective, promotes urinary colonization with a more resistant population of bacteria, and decreases GI colonization resistance.

LEPTOSPIROSIS

Although widespread vaccination has dramatically decreased the incidence of leptospirosis, recent work suggests that it is on the rise. In addition, there is an increasing prevalence of *Leptospira* serovars that are not commonly included in vaccinations. Leptospirosis is also being diagnosed more often in recently developed areas and locations on the perimeter of urban developments. Given the zoonotic potential of leptospirosis, accurate and early diagnosis is imperative. Unfortunately, diagnosis of leptospirosis has historically been very challenging. Culture is usually unrewarding because of the time it takes and difficulty isolating the organism. Identifying leptospires using darkfield microscopy is helpful, but the sensitivity of this test is poor. Therefore, antemortem diagnosis relies on history, clinical index of suspicion, and high initial or convalescent titers. In many cases, leptospirosis is never confirmed because animals die before convalescent titers can be obtained but after a long hospitalization that greatly complicates postmortem identification of the organism. A test using polymerase chain reaction technology has recently been validated to detect leptospirosis. Because the organism may rapidly disappear from urine when therapy is instituted, it is essential that pretreatment urine be collected for analysis.

Treatment of potential leptospirosis typically has two phases. Penicillin derivatives are highly effective against circulating leptospires and should be administered early to decrease total tissue damage if leptospirosis is a con-
Since then, there has been a change in the CE reviews. Fomepizole is known to be ineffective in preventing ARF in cats. However, in a recently reported pilot study, fomepizole was effective in preventing ARF in dogs at doses currently recommended for cats. Although hemodialysis centers continue to open throughout the United States, practitioners should remain familiar with this procedure through further reading before considering in-clinic use. The cost of peritoneal dialysis can be anticipated to be $6,000 to $15,000, depending on complications, length of treatment, and patient condition when initiating treatment. Hemodialysis costs are similar and vary by treatment center. This subject has also been recently reviewed.

Renal transplantation has historically been viewed as an extreme measure for special cases. That attitude continues to evolve, and transplantation is currently being offered for cats at multiple facilities throughout the United States. As a result, transplantation morbidity and mortality have significantly decreased, as has the associated cost. Criteria for patient selection, cost, risk factors, and complications have been well described in recent Compendium reviews.

There has recently been an exciting development in the field of canine renal transplantation. Drs. Lithrop and Tillson at Auburn University have been developing a protocol for renal transplantation that would allow dogs to receive kidney grafts from unrelated donors. The procedure uses concurrent bone marrow and renal transplantation to decrease the incidence of graft rejection and facilitate eventual discontinuation of immunosuppressive medications. Successful transplantation into a pet was recently achieved. The dog had ethylene glycol intoxication, leading to irreversible renal failure. The dog had been maintained for several months with peritoneal dialysis and hemodialysis but remained anuric. Since transplantation, the dog has been discharged with normal urine production. At the time of discharge, the dog was maintaining normal renal values without treatment. As this protocol allows dogs to be weaned from immunosuppressives, it will be interesting to see whether adoption of this protocol will be associated with decreased development of opportunistic infections in cats.

EMERGING TREATMENTS

Although not yet available commercially, the use of cross-linked polyelectrolyte sorbents to correct overhydration in uremic animals has been recently described. Oral administration stimulates profound adsorption of water in the intestinal tract. Used at the recommended
dose, these sorbents may facilitate a 10% reduction in total body weight daily. The sorbents also bind non-selectively to uremic toxins, facilitating excretion via the GI tract and decreasing the need for dialysis.

CONCLUSION

ARF remains a challenging disease, and affected patients have a guarded prognosis. The key to successful treatment of ARF remains prevention. Fluid therapy and basic patient support continue to be the mainstays of treatment. Improvements in disease management focus on preventing iatrogenic complications through more aggressive clinical monitoring. Randomized controlled trials in animals are desperately needed to better elucidate appropriate veterinary treatment options.

REFERENCES


April 2005

COMPRENDUIM

Treating Acute Renal Failure


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1. Renal azotemia is characterized by
   a. hyposthenuria.
   b. isosthenuria.
   c. hyperphosphatemia.
   d. acidosis and hyperphosphatemia.

2. If adequate UOP (i.e., 0.5 to 2 ml/kg/hr) is not achieved after initial fluid resuscitation, the next step is to
   a. administer diuretics.
   b. institute dialysis or refer the patient for it.
   c. reassess the hydration status.
   d. increase the fluid rate.

3. Once adequate rehydration has been achieved, fluids should be adjusted to match fluid loss by type. Insensible losses should be replaced with
   a. isotonic crystalloids.
   b. colloids.
   c. hypertonic crystalloids.
   d. hypotonic crystalloids or 5% dextrose.

4. Which drug does not stimulate diuresis?
   a. dopamine
   c. diltiazem
   b. mannitol
   d. dobutamine

5. Mannitol is contraindicated in patients
   a. that are fluid overloaded.
   b. with ethylene glycol intoxication.
   c. that are oliguric.
   d. with hyperkalemia.

6. Dopamine should not be administered
   a. with furosemide.
   b. in animals that have failed to respond to mannitol.
   c. in cats.
   d. in animals with pyelonephritis.

7. Which statement is not a reason that human data on dopamine may not be applicable to dogs?
   a. Renal failure in dogs often involves very different pathophysiologic mechanisms than it does in humans.
   b. Dopaminergic receptors are distributed differently in the kidneys of dogs and humans.
   c. Humans may have more sensitivity to medullary ischemia secondary to dopamine.
   d. Humans can get severe hypertension with dopamine administration.

8. Which of the following is not believed to be a mechanism of action of diltiazem in treating oliguria?
   a. decreased renal vascular tone
   b. antiendothelin effects that lead to decreased renal vasoconstriction
   c. decreased systemic BP
   d. decreased cell death

9. Rehydration of patients with ARF and hyperkalemia leads to
   a. continued hyperkalemia.
   b. normokalemia.
   c. hypokalemia.
   d. It depends on whether the animal is polyuric, oliguric, or anuric.

10. GI ulceration with ARF is due to
    a. decreased blood flow.
    c. hypergastrinemia.
    b. uremic vasculitis.
    d. b and c