A clinical review of pathophysiology, diagnosis, and treatment of uroabdomen in the dog and cat

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Abstract

Objective – To review current literature regarding uroabdomen in dogs and cats with respect to etiology, diagnostic approach, medical and surgical treatment, and prognosis.

Etiology – Uroabdomen in dogs and cats is most often associated with vehicular or blunt trauma. This condition may also result from urinary tract obstruction, traumatic bladder expression or catheterization, neoplasia, and postoperative leakage following abdominal or urogenital surgery.

Diagnosis – Disruption to the urinary tract should be considered when a patient is diagnosed with azotemia, hyperkalemia, and abdominal effusion. By comparing the creatinine concentration of the abdominal fluid to the serum or plasma creatinine concentration, a diagnosis of uroabdomen can be made if the creatinine ratio is ≥2:1. In most patients imaging studies with contrast are necessary to identify the exact source of urine leakage and to determine therapeutic options.

Therapy – Uroabdomen is a medical emergency, not a surgical emergency. Acute management involves stabilization of the patient with IV fluid therapy and treatment of hyperkalemia. Urinary diversion and, in some cases, peritoneal dialysis are necessary to stabilize the patient until life-threatening conditions such as hyperkalemia or concomitant injuries such as pulmonary contusions resolve. Once the patient is stable for anesthesia, surgical repair, if indicated, may be performed.

Prognosis – The prognosis of patients with uroabdomen depends on the extent of urinary and nonurinary injuries as well as the development of complications. Potential complications include dehiscence or urine leakage following surgical repair of the urinary tract, urosepsis, unresolving azotemia secondary to renal damage or underlying renal insufficiency, or stricture formation in the urinary tract.

Keywords: hyperkalemia, ureteral rupture, urethral rupture, urinary bladder, uroperitoneum

Introduction

Uroabdomen results from rupture of the urinary tract with subsequent accumulation of urine in the peritoneal cavity, retroperitoneal cavity, or both. Uroperitoneum generally develops from disruption of the distal ureters, bladder, or proximal urethra whereas uroretroperitoneum results from injury to a kidney or proximal ureter with the peritoneum remaining intact. With either condition, the presence of urine in the abdomen causes severe electrolyte and metabolic changes. Such derangements exert deleterious effects on cardiac and renal function and become life-threatening if not promptly corrected. This review focuses on uroabdomen in dogs and cats with emphasis on common causes, methods of diagnosis, medical stabilization, surgical repair options, and prognosis.

Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>CRI</td>
<td>continuous rate infusion</td>
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<tr>
<td>CT</td>
<td>computed tomography</td>
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<td>FAST</td>
<td>focused abdominal sonography for trauma</td>
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<tr>
<td>GFR</td>
<td>glomerular filtration rate</td>
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<tr>
<td>MRI</td>
<td>magnetic resonance imaging</td>
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<tr>
<td>PD</td>
<td>peritoneal dialysis</td>
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<td>SUB</td>
<td>subcutaneous ureteral bypass</td>
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Etiology and Pathophysiology

Uroabdomen in dogs and cats is most often associated with vehicular or blunt trauma to the abdomen or pelvis. The urinary bladder is the most common site of rupture.\textsuperscript{2,4,7,11–15} A retrospective study of uroperitoneum in 26 cats documented uroabdomen secondary to blunt abdominal trauma in 13 cats, of which 11 had a ruptured bladder. Dogs and cats develop uroperitoneum because their urinary bladder is located within the peritoneal cavity.\textsuperscript{4} Conversely, two-thirds of people with a ruptured bladder develop uroretroperitoneum because the human bladder is located in the retroperitoneal space and is lined by peritoneum on the superior and lateral surfaces.\textsuperscript{16}

The probability of bladder injury correlates directly with the degree of bladder distention at the time of trauma.\textsuperscript{17,18} The rapid rise of intraperitoneal pressure caused by the traumatic event may cause rupture of a distended bladder with a thin wall and stretched muscle fibers.\textsuperscript{1,16,17,19} Male dogs are at an increased risk of bladder rupture because their long, narrow urethra cannot easily adapt to a rapid rise in intravesical pressure.\textsuperscript{1,7,20,21} Patients that incur pelvic fractures also have an increased likelihood of uroabdomen due to deformation of the pelvic canal in addition to laceration of the bladder or urethra from sharp bone fragments.\textsuperscript{4,17,22,23} Rupture of the prostatic or membranous portion of the urethra is the most common site of injury in dogs with pelvic fractures.\textsuperscript{24}

Ureteral rupture secondary to external trauma is much less common than bladder rupture in dogs, cats, and people. The narrow diameter and mobility of the ureters as well as their protection by the retroperitoneal space, dorsal body wall musculature, and peritoneal organs help reduce the likelihood of damage.\textsuperscript{18,25} If a ureteral rupture does occur, it most often occurs in the proximal portion.\textsuperscript{26} A case report published in 2006 documented 2 dogs that developed bilateral ureteral ruptures from motor vehicular trauma.\textsuperscript{12} Another retrospective study highlighted 10 animals with traumatic rupture of the ureter, of which 8 had sustained motor vehicular trauma.\textsuperscript{27}

Injuries to the renal parenchyma following trauma are uncommon. The kidneys are less tightly attached to the body wall in the cat relative to the dog; but, in both species, they are protected by the spine, dorsal body musculature, perinephric fat, and rib cage.\textsuperscript{1,3} Types of renal injuries include contusions, fissures, or lacerations of the renal parenchyma, or vascular pedicle injuries.\textsuperscript{3,14,22} Fractures of the spine or last 3 ribs in a dog or cat with uroabdomen should raise concern for a ruptured kidney.\textsuperscript{14,15}

Nontraumatic causes of uroabdomen in dogs and cats include spontaneous bladder rupture secondary to urethral obstruction, bladder neoplasia,\textsuperscript{4,28} manual bladder expression, cystocentesis, urethral catheterization, genitourinary tract surgery, or accidental injury to the urinary tract during abdominal surgery.\textsuperscript{2,4,13,27,29,30} Injury during parturition is the most common cause of uroperitoneum in foals but this has not been reported in canine or feline neonates.\textsuperscript{29} Table 1 lists the most common causes of nontraumatic bladder rupture in people.

Urethral obstruction as well as manual bladder expression and urethral catheterization predispose cats to developing uroabdomen from bladder or urethral rupture.\textsuperscript{4,24,30} In the aforementioned study of 26 cats with uroperitoneum, 9 of the cats which had not sustained blunt abdominal trauma were males with urethral obstruction. Two of the 9 cats had spontaneous rupture of the urinary bladder, and the other 7 developed uroabdomen following urethral catheterization. One cat in this study developed a uroabdomen secondary to bladder neoplasia which has also been reported in dogs.\textsuperscript{9} In male dogs, urethral rupture from a calculus or traumatic catheterization is more likely to occur at the penile portion of the urethra and cause extravasation of urine into surrounding tissues rather than uroabdomen.\textsuperscript{21,24}

Complications associated with urogenital and abdominal surgery include urine leakage and uroabdomen.\textsuperscript{31,32} Complications following ureterotomy, urethrotomy, cystotomy, or temporary cystostomy include leakage of urine from the surgical site, dehiscence of devitalized tissue, or subsequent ureteral or

### Table 1: Etiologies of uroperitoneum or uroretroperitoneum in people

- **Iatrogenic**:\textsuperscript{1,18,22,25}
  - Renal, ureteral, cystic, or urethral injuries during surgical, endourologic or percutaneous procedures
  - Ureteral obstruction due to surgical ligation of a ureter
- **Urine leakage**:\textsuperscript{22}
  - Dehiscence following renal transplantation or ureteral diversion procedures
  - Following cystotomy for bladder stones or cystoscopy if the bladder wall is affected by chronic inflammation, infection, or neoplasia
- **Increased back pressure/obstruction**:\textsuperscript{18,22}
  - Pelvic masses
  - Pregnancy
  - Retroperitoneal fibrosis
  - Posterior urethral valves
  - Uroliths in the ureter or urethra
- **Other predisposing factors**:\textsuperscript{1,18,22,60,84}
  - History of trauma
  - Neuropathic bladder dysfunction following a stroke
  - Postpelvic radiation
  - Previous pelvic surgery
  - Alcohol binge drinking
  - Chronic bladder infections
  - Pressure necrosis or chemical irritation from indwelling urinary catheters
urethral obstruction from swelling.\(^{13,31}\) During ovariohysterectomy, ureteral ligation or trauma can occur.\(^{33}\) Nephrectomy or percutaneous renal biopsy rarely result in urine leakage.\(^{34}\)

**Complications of Uroabdomen**

In patients with uroabdomen, potassium-containing urine accumulates in the abdominal cavity and is reabsorbed into the systemic circulation down a concentration gradient, resulting in hyperkalemia.\(^{4,9,11,35–39}\) In an attempt to maintain homeostasis, renal excretion of potassium is increased through the effects of the principal cells and aldosterone on the Na\(^+/K^+\)-ATPase pumps in the distal tubule and collecting ducts.\(^{40}\) Because potassium cannot be absorbed across the peritoneal membrane as quickly as it is excreted into the peritoneal cavity, the concentration of potassium in the abdominal fluid remains higher than that of peripheral blood.\(^{8,9,35,37}\)

Hyperkalemia increases the resting membrane potential of cells in the body, which reduces the gradient between the resting membrane and threshold potential, and subsequently causes an increase in cell membrane excitability. In cardiac myocytes, this increased excitability can result in life-threatening cardiac arrhythmias progressing from bradycardia to ventricular fibrillation or asystole.\(^{6,8,38,41,42}\) Cardiac conduction disturbances may be seen and usually depend on the degree of hyperkalemia in addition to concurrent abnormalities such as metabolic acidosis, hypocalcemia, and hypotension, which can exacerbate the detrimental effects of hyperkalemia on cardiac electrical conduction.\(^{6,8,10,41}\)

**Initial Stabilization**

Initial stabilization of patients that have sustained severe trauma or have evidence of hypovolemic shock should take place immediately upon presentation to the hospital.\(^{38,43,44}\) Reestablishing tissue perfusion and oxygen delivery may be accomplished by IV administration of isotonic or hypertonic crystalloids or colloids. The fluid rate is dependent on the severity of shock and whether hemorrhage, if present, is ongoing or has stopped, and is tailored to meet specific endpoints of resuscitation.\(^{44,45}\) Concomitant conditions such as pulmonary contusions or traumatic brain injury also need to be considered when administering fluid therapy.\(^{44}\) Oxygen supplementation may be indicated during resuscitation from shock. Physical examination abnormalities such as an abnormal cardiac rhythm or palpable abdominal fluid wave warrant an ECG and abdominocentesis, respectively.\(^{8,39,46}\)

Two studies evaluating the initial resuscitation of hyperkalemic cats with urethral obstruction determined that the IV use of isotonic crystalloid solutions are acceptable, and that choosing a balanced electrolyte solution such as Normosol-R\(^{\text{®}}\) instead of 0.9% sodium chloride may more rapidly improve the acid-base status and not exacerbate preexisting acidosis.\(^{2,38,47}\) Historical recommendations for shock fluid boluses range from 60 to 90 mL/kg for dogs and 40–60 mL/kg for cats.\(^{45,48}\) Aggressive fluid administration, however, may cause detrimental effects such as interstitial edema, dilution of coagulation factors, and dislodgement of blood clots in areas of prior hemorrhage.\(^{38,44}\) Therefore, administering crystalloid fluids in increments of one-quarter of the total shock volume with reevaluation of endpoints such as the patient’s mentation, capillary refill time, heart rate, blood pressure and, ideally, urine output, following each bolus is recommended to determine ongoing fluid requirements.\(^{38,43,44}\)

Although fluid resuscitation is underway, baseline diagnostic testing including PCV, total plasma protein, blood glucose, and concentrations of serum electrolytes should be obtained. If the patient is mild to moderately hyperkalemic with a serum potassium concentration >5.5 mmol/L but <7.5 mmol/L, fluid therapy alone may promote potassium excretion by improving glomerular filtration rate (GFR).\(^{10}\) In patients with serum potassium exceeding 7.5 mmol/L, additional therapies may be necessary to reduce serum potassium or protect the heart from hyperkalemia-induced electrical disturbances.\(^{10,41,42,49}\) Table 2 lists the expected ECG abnormalities associated with hyperkalemia in dogs and cats. In people with hyperkalemia, and likely veterinary patients, however, the serum potassium concentration is not always associated with the expected ECG findings because the sensitivity of ECG is low.\(^{41}\)

Management of severe hyperkalemia in people and animals is focused on antagonizing the effects of potassium on myocardial cells, followed by reduction of hyperkalemia through redistribution and elimination.
Table 3: Treatment of severe hyperkalemia

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism of action</th>
<th>Dose</th>
<th>Comments</th>
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<tbody>
<tr>
<td>Insulin and 50% Dextrose</td>
<td>Insulin-mediated simultaneous movement of glucose and potassium into cells via stimulation of Na⁺-K⁺-ATPase activity in skeletal muscle ¹⁻⁵</td>
<td>Dogs and cats: Give 0.5 units/kg regular insulin IV and, for every unit of insulin administered, give 2 grams (4 mL) of 50% dextrose diluted IV to prevent hypoglycemia ²</td>
<td>In people, dextrose alone is not recommended because the rise of endogenous levels of insulin is less likely to have a therapeutic benefit ⁸</td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>Improves extracellular acidosis and indirectly increases activity of Na⁺-K⁺-ATPase causing a shift of potassium into the cell in exchange for hydrogen ³⁻¹²</td>
<td>Give 0.7–1 g/kg (1.4–2 mL/kg) diluted and given slowly IV over 3–5 minutes ²</td>
<td>Controversial as a first line therapy. May be more efficacious if given with other proven therapies for treatment of hyperkalemia ¹⁰, ¹³⁻¹⁶</td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>Stimulation of Na⁺-K⁺-ATPase activity ²⁻⁴, ⁶</td>
<td>Dogs and cats: Give 0.01 mg/kg IV slowly ²</td>
<td>Use of β₂-adrenergic agonists as a single agent treatment for humans with hyperkalemia is not recommended ⁶, ⁷</td>
</tr>
<tr>
<td>Terbutaline</td>
<td>Same as above ¹, ³, ⁴</td>
<td>Give 1–2 mEq/kg IV slowly over 15 minutes ²</td>
<td>Calcium antagonizes the effect of hyperkalemia on the myocardium but does not lower potassium concentration ², ⁶, ¹⁷</td>
</tr>
<tr>
<td>10% Calcium Gluconate</td>
<td>Reduces cardiac excitability by reestablishing the normal gradient between the resting membrane and threshold potentials ¹, ⁶</td>
<td>Give 0.5–1.5 mL/kg IV slowly over 5–10 minutes. An ECG should be monitored during the infusion for evidence of bradycardia or exacerbation of the arrhythmia ²</td>
<td>Calcium antagonizes the effect of hyperkalemia on the myocardium but does not lower potassium concentration ², ⁶, ¹⁷</td>
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Calcium, either as calcium gluconate or calcium chloride, may be administered IV to negate the effects of hyperkalemia on the myocardium, but this therapy does not reduce serum potassium concentrations ¹⁰, ⁴¹, ⁴² Serum potassium concentrations may be decreased by the administration of IV fluids, dextrose, insulin with dextrose, β₂-agonists (may also be inhaled), or sodium bicarbonate ⁵⁰⁻⁵⁹. The ultimate solution for hyperkalemia is establishing urinary diversion. Treatment of severe hyperkalemia with the aforementioned drugs is indicated in patients with cardiac arrhythmias consistent with hyperkalemia ⁸, ⁸¹. Because people manifest cardiac disturbances at potassium concentrations >6.7 mmol/L, including ventricular fibrillation, treatment is instituted when the potassium is >6.5 mmol/L despite the lack of an abnormal ECG in some patients ⁶, ⁴¹. Ventricular pacing can also be used in people with severe bradycardia or asystole secondary to hyperkalemia ⁶. Other reasons to pursue more aggressive treatment of hyperkalemia include the presence of neuromuscular weakness, decreased myotatic reflexes, or severe metabolic disturbances that may exacerbate the consequences of hyperkalemia ¹⁰, ⁴¹.

Though management of life-threatening conditions such as hypovolemia and hyperkalemia is vital, other aspects of patient care including pain management should not be overlooked. Chemical peritonitis secondary to uroperitoneum can be painful ²⁸, ⁶⁰. Reversible pure mu agonists such as fentanyl, morphine, or hydromorphone may be used for analgesia in patients with uroabdomen. These drugs may be administered by constant rate infusion (CRI) or by intermittent injections. Caution should be used in patients that are unstable as opioids can exacerbate hypotension and hypoventilation. If complications occur following administration of pure mu opioids, naloxone can be used to reverse the effects ⁶¹. Abstaining from medications that are not reversible or that may have detrimental effects on renal or gastrointestinal blood flow is recommended during initial stabilization ⁶¹, ⁶². In patients with uroabdomen and concurrent hypovolemia, or postrenal azotemia, the use of nonsteroidal anti-inflammatory drugs is not recommended ⁶¹, ⁶³. Dexmedetomidine ⁵ may exacerbate cardiac toxicity in patients with electrolyte derangements, azotemia, or cardiac arrhythmias because of the severe bradycardia, peripheral vasoconstriction, and decreased cardiac output caused by the drug ⁶¹. Dexmedetomidine also increases urine output, which may be contraindicated in patients with urinary obstruction. Multimodal analgesic options, in addition to opioids, include ketamine and lidocaine which are used as CRIs, gabapentin, and local anesthesia ⁶¹. As ketamine is excreted unchanged in the urine, its use in animals with uroperitoneum may lead to prolonged sedation and anesthesia because it will be continuously reabsorbed until the uroabdomen is drained. Due to the potential for toxicity, lidocaine should be used with caution in cats ⁶⁴, ⁶⁵.
**Diagnosis of Uroabdomen**

Most veterinary patients presenting with uroabdomen have a history of trauma. In people, assessment of the genitourinary system is frequently neglected because victims of blunt trauma have an average of 3 other major injuries. In the initial evaluation of an animal that has sustained motor vehicular trauma, concomitant urinary tract trauma may also be overlooked as these patients often present with other life threatening conditions such as hypovolemic shock, pulmonary contusions, traumatic brain injury, and fractures. Some dogs and cats presenting after motor vehicular trauma show no initial clinical signs of urinary tract disruption, whereas others develop nonspecific signs of vomiting, anorexia, weakness, and lethargy secondary to progressive azotemia, hyperkalemia, metabolic acidosis, and hypovolemic shock. Patients may also manifest abdominal pain with or without signs consistent with urinary tract trauma.

Diagnosis of uroabdomen is based on history and physical exam findings along with laboratory evaluation and imaging studies. Physical exam may reveal lethargy, tachycardia, bradycardia or other arrhythmias, severe abdominal pain, a palpable fluid wave in the abdomen, and bruising of the inguinal region or perineum. Trauma patients, in general, often have similar clinical signs without uroabdomen, making diagnosis more difficult or delayed. Patients can have hematuria and display signs of stranguria, dysuria, or anuria. The urinary bladder may or may not be palpable. A palpable urinary bladder does not rule out a leak or rupture of the urinary tract (including the bladder itself), and voiding of urine without gross hematuria does not exclude a diagnosis of a ruptured bladder.

In a human report that reviewed bladder trauma literature, ruptured bladders were almost always accompanied by gross hematuria, pelvic fractures, or both. Microhematuria alone may be present but is more often associated with bladder contusions and less often with bladder rupture. In human hospitals, diagnostic imaging of the urinary tract to look for evidence of urinary tract trauma or rupture is recommended for trauma patients that present with gross hematuria with or without pelvic fractures, microhematuria with pelvic fractures, and pelvic fractures alone.

Initial diagnostic testing performed for all animals suspected to have urinary tract trauma should include a CBC and measurement of serum electrolyte concentrations, a serum biochemistry profile, and acid-base status, if available. The most common abnormalities associated with uroabdomen are azotemia, metabolic acidosis, and electrolyte derangements including mild hyponatremia, hyperphosphatemia, and hyperkalemia. The severity of serum biochemical changes will depend on the time elapsed from the onset of uroabdomen to diagnosis as well as the rate of diffusion for various molecules across the peritoneal membrane. Unlike azotemia and hyponatremia, which often develop in 24 hours following the onset of uroabdomen, hyperkalemia may not develop for 48 hours or more. Patients that continue to void urine and are maintained with IV fluid therapy may take longer to show changes in their laboratory values relative to the 24–48 hours in which changes are seen in most patients with uroabdomen. Conditions that have similar clinical signs and laboratory abnormalities to uroabdomen such as hypoadrenocorticism and acute renal failure should be ruled out.

**Evaluation of Abdominal Effusion**

If abdominal effusion is noted on abdominal palpation, radiographs, or focused assessment with sonography for trauma (FAST) examination, a sample of the effusion should be collected by abdominocentesis with or without ultrasound guidance, and analyzed for biochemical and cytologic characteristics. Urine within the peritoneal cavity can appear as a transudate, modified transudate, or exudate, with variation caused by hemorrhage or inflammatory cells. Because urine is a chemical irritant, it can result in nonseptic neutrophilic inflammation with a cell count >5,000 nucleated cells per microliter, a specific gravity of >1.025, and total solids of >3.0 gram/dL. A septic effusion may develop if bacteria is present, and is diagnosed cytologically by the presence of intracellular bacteria.

To confirm that the effusion is originating from the urinary tract, the ratios of creatinine and potassium in the abdominal effusion to peripheral blood can be compared as demonstrated in Table 4. Creatinine diffuses slowly across the peritoneal membrane and yielding a less reliable marker for diagnosis of uroabdomen. Because both potassium and creatinine remain in high concentrations in the abdominal fluid, they serve as useful indicators to diagnose uroabdomen.

In a retrospective study evaluating creatinine and potassium concentration ratios of abdominal fluid to peripheral blood in dogs with uroperitoneum, a creatinine ratio greater than 2:1 was predictive of uroabdomen with a specificity of 100% and sensitivity of 86%. When comparing potassium concentration ratios, a ratio of greater than 1.4:1 was also consistent with uroabdomen with both the specificity and sensitivity being 100%. In the same study all dogs had a creatinine concentration of their abdominal effusion that was at least 4 times that of...
**Table 4:** Guidelines for diagnosis of uroabdomen in dogs and cats

<table>
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<th>Laboratory evaluation</th>
<th>Clinical significance</th>
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<td>Creatinine of abdominal effusion is ≥ 2 times that of peripheral blood</td>
<td>Considered diagnostic to uroabdomen</td>
</tr>
<tr>
<td>Creatinine of the abdominal effusion &gt; 1 but &lt; 2 times that of peripheral blood</td>
<td>Suggestive for uroabdomen but additional criteria necessary to obtain diagnosis</td>
</tr>
<tr>
<td>Potassium of the abdominal effusion &gt; peripheral blood</td>
<td>Suggestive for uroabdomen. The higher the ratio, the more strongly suggestive</td>
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normal peripheral blood. In the previously mentioned study of 26 cats with uroperitoneum, ratios of creatinine and potassium concentrations of abdominal effusion compared to peripheral blood were 2:1 and 1.9:1, respectively.

**Imaging Studies**

Following initial stabilization and laboratory diagnostics, abdominal imaging is necessary to document the location of the disruption in the urinary tract. Imaging modalities include abdominal radiographs, abdominal ultrasound, retrograde positive contrast cystography, excretory urography, and computed tomography (CT). In human medicine, CT is the diagnostic test of choice. In veterinary medicine, radiography is the modality of choice as it is accessible to most veterinarians and can be utilized in the majority of cases to assess disruption in the urinary tract through contrast radiography (Figures 1A–2C).

Survey radiography is limited as a sole diagnostic tool in animals with abdominal effusion due to limitations in serosal detail. Radiographic findings that increase the index of suspicion of urinary tract leakage include the presence of retroperitoneal or peritoneal effusion, inability to visualize the margins of the bladder, and pelvic fractures. Visibility of a urinary bladder does not exclude a small leak from the bladder or disruption of another portion of the urinary tract.

Ultrasonography is a noninvasive technique that can be used to assess the architecture of the kidneys and other structures and to confirm the presence of fluid. However, ultrasonography is of limited utility for determination of the site of disruption in the urinary tract. The ureters are difficult to image sonographically due to their small size although they may be imaged as they enter the urinary bladder. The urinary bladder is best imaged when it is moderately full so that the bladder wall and lumen may be evaluated. Patients with urinary tract leakage may have a collapsed, empty urinary bladder, exhibiting a thicker wall and reduced definition.

The entire urethra can be evaluated via ultrasonography; however, due to interference by the pubic bones, a transrectal approach is necessary for assessment of the caudal portion of the urethra, which may or may not be feasible.

A more recent use of ultrasound to evaluate bladder rupture incorporates the use of a microbubbled sterile saline solution. One study reported the prospective use of contrast cystosonography in 2 dogs with ascites from suspected bladder rupture. For each dog, the investigators infused microbubbled saline slowly through indwelling urinary catheters over 10–15 seconds; and, at the same time, the abdomen was evaluated sonographically using harmonics. In both cases, the bladder remained small or collapsed but microbubbles were seen to pass into the surrounding ascites supporting bladder rupture. Both dogs also underwent radiographic positive-contrast cystography before or after

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Figure 2: (A) Lateral radiograph of a 10-month old, intact male, mixed breed dog with retroperitoneal effusion that was hit by a car and sustained pelvic fractures in addition to a right femoral fracture and a Salter-Harris fracture of L5. Radiograph courtesy of The University of Tennessee, College of Veterinary Medicine. (B) Excretory urographic study of the dog depicted in part (A) of this figure. There is a large amount of contrast in the retroperitoneal space secondary to a left proximal ureteral rupture. Radiograph courtesy of The University of Tennessee, College of Veterinary Medicine. (C) Ventrodorsal radiograph of the young dog in part (A) of this figure following excretory urography which confirms a left proximal ureteral rupture resulting in leakage of contrast (arrows). There is no evidence of contrast in the distal ureter indicating a complete rupture. The right kidney and ureter are still intact allowing contrast to accumulate in the urinary bladder as seen in the ventrodorsal and lateral views. Radiograph courtesy of The University of Tennessee, College of Veterinary Medicine.

The clinical value of contrast cystosonography relative to retrograde contrast cystography for the diagnosis of bladder rupture is unknown. Cystosonography requires special equipment, expertise, and expense while retrograde positive contrast procedures may be performed in many veterinary hospitals with standard radiographic equipment.

Because retrograde positive contrast cystography can be easily performed to evaluate the bladder or urethra for rupture, it remains the diagnostic of choice in most clinical settings. A 10–20% solution of a water soluble, organic, iodinated contrast medium such as iohexol is infused into the bladder following aseptic bladder catheterization and removal of residual urine. Retrieval of urine does not rule out a ruptured bladder. The amount of contrast medium necessary to distend the urinary bladder is approximately 10 mL/kg, or until there is resistance of the syringe during infusion of contrast. Postcontrast radiographs are performed immediately following infusion to identify leakage of contrast medium, which will appear as an irregular pattern within the peritoneal cavity or outlining the intraperitoneal viscera. If no contrast is noted, repeating the radiographs 5–10 minutes later may allow for smaller leaks to become visible. Important recommendations to ensure that the positive contrast cystogram is of diagnostic quality include making sure the colon is devoid of feces, the acquisition of orthogonal or oblique views if necessary, and achieving adequate distention of the urinary bladder with contrast medium.

A tear in the proximal urethra near the trigone of the bladder can result in uroabdomen. Leakage of urine from the caudal aspect of the urethra results in severe bruising.
and swelling of the inguinal and perineal regions due to extravasation of urine causing local tissue inflammation and necrosis. If a urethral tear is suspected, a retrograde positive contrast urethrogram can be performed using a 10–20% solution of a water soluble, organic, iodinated contrast medium. Ideally this is performed before urethral catheterization for a cystogram. The urinary catheter is prefilled with contrast to prevent introduction of air bubbles. In male dogs, a urethrogram is best accomplished by catheterizing the distal urethra with a balloon-tipped catheter and inflating the balloon enough to prevent reflux of contrast medium. For smaller dogs and cats in which a balloon-tipped catheter will not fit, passing the widest catheter possible and gently clamping the tip of the urethral orifice with an atraumatic clamp will help reduce backflow of contrast material. Injection of 10–15 mL of contrast medium in dogs and 5–10 mL in cats is adequate for urethral filling. A lateral radiograph is performed toward the end of the infusion to assess for leakage. A lateral view is usually adequate but oblique and ventrodorsal views may be necessary to further assess for leakage of contrast.

Urethral catheterization may be more difficult in female dogs. Another approach for a urethrogram in female dogs without a urethral catheter is a vaginourethrogram. With heavy sedation or anesthesia, a large balloon-tipped catheter is inserted into the vagina rather than the urethra and the balloon is inflated to prevent loss of contrast medium. The contrast is injected through the catheter and will fill the vagina initially until a noticeable amount of resistance is met during the injection as the contrast reflexes into the urethra. Radiographs are then taken to assess the integrity of the urethra. Diagnosis is obtained if there is visualization of contrast leakage into the peritoneal cavity or into the periurethral tissues. Leakage of contrast into the periurethral tissues with the presence of contrast medium reaching the urinary bladder is consistent with a partial urethral tear. If periurethral leakage of contrast occurs without visualization of contrast in the bladder despite adequate instillation of contrast, a complete urethral tear should be suspected. Figure 1A and B shows an example of a dog with a rupture of both the urinary bladder and proximal urethra (partial rupture).

Excretory urography should be considered in patients in which no leakage is noted from the bladder or urethra during retrograde cystography, if urinary catheterization is difficult, or if a urinary tract disruption proximal to the urinary bladder is suspected. Excretory urography is the most common radiographic modality used to assess ureteral patency and integrity. Survey radiographs are performed as a baseline and to confirm that the colon and gastrointestinal tract are clear of contents that may result in artifact. Contrast media such as io-hexol or iopamidol are infused rapidly as an IV bolus through a peripheral catheter at a dose of 425–880 mg iodine per kg of body weight. The dose of contrast medium may need to be increased or readministered in patients with renal failure due to their inability to concentrate contrast in the urine, unless contrast nephropathy is a concern. The dose should not exceed 35 g of iodine. A ventrodorsal abdominal radiograph is performed between 5 and 20 seconds after contrast is injected, and at 5 minutes, 20 min, and 40 min following injection. Oblique views should be obtained at 3–5 min after injection to image the ureteral insertion into the urinary bladder. A lateral radiograph is performed at 5 minutes for general assessment. Abnormalities that would indicate disruption in the urinary tract include extravasation of contrast surrounding a kidney or discontinuity of a ureter with extravasation of contrast medium into the retroperitoneal space consistent with kidney or ureteral rupture. Contrast may also be visible around the urinary bladder if there was avulsion of a ureter from its insertion on the bladder. Figure 2A–C illustrates an example of a dog with a proximal ureteral rupture.

Documented side effects following IV administration of contrast media such as io-hexol or iopamidol include exacerbation of azotemia in patients with concurrent renal disease, hypotension, and bradycardia. In people, the pathophysiology of contrast-induced nephropathy is thought to result from direct toxicity of contrast medium to nephrons, microshowers of cholesterol emboli to the kidneys, and contrast-induced intrarenal vasoconstriction. These factors result in hypoxic and ischemic insult to the kidneys with subsequent development of acute renal failure. In animals there is controversy regarding whether contrast agents induce vasoconstriction or vasodilation but direct toxicity related to osmolarity of the contrast agents is similar to that seen in people. In a well-hydrated, normotensive patient producing adequate amounts of urine, excretory urography should not be a contraindication even in the presence of azotemia. However, in a patient with a known history of chronic kidney disease, there is an increased risk due to reduced GFR and fewer nephrons to clear the contrast. Use of nonionic, water-soluble, iodinated contrast media is recommended in these high-risk patients. In all cases, advising owners of the risks of contrast-induced nephropathy is recommended.

CT and magnetic resonance imaging (MRI) are other diagnostic modalities that can be used to evaluate urinary tract disruption. In human medicine, CT is the imaging modality of choice especially if additional abdominal imaging is necessary for trauma victims over retrograde positive contrast cystography or intravenous urography. CT excretory urography has been.
investigated in healthy animals due to the difficulty in interpreting radiographic excretory urograms for conditions such as ureteral ectopia. Benefits of CT excretory urography include lack of superimposition of surrounding structures which improves visualization of the ureter, ability to reconstruct 2-dimensional and 3-dimensional images of the ureter, and the allowance for administration of lower concentrations or volume of contrast medium. One pitfall of CT excretory urography noted in veterinary patients is peristaltic contractions of the ureter causing loss of visualization due to the disappearance of contrast medium. Multiple scans of a region of interest may then be necessary and patients will receive higher doses of radiation.

MRI is also utilized in the human medical field to assess the urinary tract. MRI urography does not expose patients to ionizing radiation and it may offer more functional information; however, MRI is a less established imaging modality and the image quality is variable relative to CT.

**Urinary Diversion**

Once uroabdomen is diagnosed, the patient may not be stable for more advanced diagnostics or surgery to correct the disruption in the urinary tract. In situations in which the patient is not responsive to the stabilization treatments listed above, temporary measures can be performed to reduce urine accumulation within the abdomen. A urinary catheter should be placed in the bladder to empty the urine that is continually being produced as long as at least 1 kidney and ureter are intact. If a urethral catheter cannot be passed due to obstruction or damage to the urethra; repetitive cystocentesis or placement of a cystostomy tube should be considered.

Repetitive cystocentesis carries the risks of bladder rupture, infection, or leakage and placement of a cystostomy tube requires general anesthesia for which the patient may not be a good candidate. Peritoneal drainage using a needle or peritoneal catheter can be used to drain fluid from the abdomen. The catheter may be aseptically placed in a conscious patient using local anesthetic block, and will promote removal of urine and expedite stabilization. Peritoneal catheters are preferred as they can be maintained for longer intervals and used for peritoneal dialysis (PD) if needed.

PD may be used as a presurgical treatment for patients with uroabdomen and severe azotemia and hyperkalemia >7.5 mmol/L (>7.5 mEq/L). This technique is discussed in greater detail in the review by Labator and Ross found elsewhere in this issue. Briefly, PD utilizes the peritoneum as a semi-permeable membrane through which solutes equilibrate between plasma and dialysate via osmosis. A commercially made PD catheter is placed into the abdomen using aseptic technique and attached to a closed-collection system. Simple intra-abdominal catheters with a stylet can be used in emergency situations and placed percutaneously using local anesthesia. Patients with surgically placed PD catheters and omentectomy tend to have fewer complications from the standpoint of catheter occlusion or poor drainage; however, surgical placement is more important in patients that are expected to require PD for more than 3 days. Premixed dialysate can be purchased or it can be made up of isotonic crystalloid fluids such as lactated Ringer’s solution. Dextrose may be added to increase the osmolarity of the dialysate, which will pull additional fluid into the abdomen. If only solute removal with no fluid removal is desired, a 1.5% dextrose solution is adequate. If fluid removal is desired such as in overhydrated patients, a 2.5% and 4.25% dextrose solution is recommended. Heparin can be added to the dialysate to reduce fibrin formation. Once the solute has had time to equilibrate, usually 30–40 minutes, the dialysate, into which excess solutes and water has equilibrated, is removed from the abdomen. The amount of time necessary for equilibration will depend on the volume of dialysate infused and the serum solute concentrations. The recommended volume for infusion in small animals is approximately 30–40 mL/kg.

Maintenance of PD catheters is labor-intensive and ideally requires 24-hour monitoring, but does not require special equipment. Daily management requires aseptic technique when working with the catheter, maintenance of sterile, dry bandaging over the catheter site, and frequent monitoring of vital signs and laboratory parameters. Warming the dialysate 2–3 degrees Fahrenheit above body temperature will improve patient comfort and enhance vasodilation. The frequency of treatments and duration of therapy are determined by the metabolic improvement of the patient.

Complications associated with PD, aside from catheter occlusion, can include hypoalbuminemia, septic peritonitis, dialysate leakage, hypothermia, electrolyte derangements and, in rare cases, dialysis disequilibrium. In a study of 22 cats undergoing PD for acute renal failure, the risk of peritonitis increased with each additional day of treatment.

Renal replacement therapy such as intermittent hemodialysis or continuous renal replacement therapy is another option for patients with severe electrolyte derangements and azotemia. Bloom and Labato recently reviewed the indications and use of intermittent hemodialysis for veterinary patients including those with urinary tract disruption. Because this treatment modality is not readily accessible to most practitioners, it will not be discussed.
Surgical Repair

Though uroabdomen is a medical emergency, surgery is often necessary to repair the source of urine leakage. Ultrasound, contrast studies, or advanced imaging should be performed to localize the urinary tract disruption before surgery as an exploratory laparotomy is not a means to assess the function or patency of the entire urogenital tract. Before surgical repair, the patient should be deemed stable with respect to renal function, electrolyte derangements, and cardiac status to ensure the patient may be safely anesthetized.

Surgical repair of renal injuries will depend on the severity of damage. Renal contusions are treated conservatively while surgical exploration is indicated in patients that have sustained trauma to 1 or both kidneys resulting in uncontrollable hemorrhage, rupture, or avulsion of a ureter from the renal pelvis. In veterinary patients, renal lacerations can often be repaired with sutures, but rupture of the kidney or ureteral avulsion may warrant nephrectomy or partial nephrectomy to reduce morbidity. For lacerations, the renal parenchyma is apposed with digital pressure and the renal capsule is closed using a simple continuous pattern of absorbable suture material. A transperineal horizontal mattress can also be performed to reduce urine leakage and hemmorhage although care must be taken to prevent vascular strangulation or pressure necrosis from the sutures themselves. Peritoneum, grafts, or materials such as gelatin foam or tissue glue can be used to reduce hemorrhage.

In people with ureteral ruptures, immediate repair is recommended barring patient instability. Ureteral repair is difficult in cats and dogs due to the small size of the ureters. Ureteral avulsions from the renal pelvis can be reimplemented if sufficient ureteral length is present; however, care must be taken to avoid trauma to the ureter from manipulation and disruption of the blood supply by minimizing dissection. This is facilitated by using stay sutures and atraumatic forceps. Ureteral avulsions from the bladder are reattached by performing a ureteroneocystostomy or a bladder-flap ureteroplasty. If the ureteral length is not adequate, the kidney can be mobilized and repositioned caudally or the bladder can be moved cranially using a psoas hitch technique. Depending on the degree of trauma and location of ureteral injury, other options for repair of ureteral injuries include ureteroureterostomy in which each end of the ureter is implanted into the contralateral ureter. Stricture formation is a common complication following ureteral procedures especially in small patients. A ureteronephrectomy is an option if the ureter is severely damaged or if complications have occurred with a previous ureteral surgery.

Patients with ureteral injuries that are unstable can be managed with temporary urinary diversion using a percutaneously placed nephrostomy tube. Nephrostomy tubes can also be placed in dogs and cats in which there is concern for ongoing urine leakage following ureteral surgery or stenting. Ureteral stenting has the benefit of potentially reducing pressure at the site of ureteral damage, fostering repair by diverting urine flow and reducing urine leakage from the surgical site.

A small number of veterinary specialists have placed indwelling ureteral stents. Polyurethane double-pigtail stents can be placed in retrograde fashion using cystoscopic and fluoroscopic guidance or antegrade following a percutaneous or a surgically assisted pyelocentesis. Urereteral stenting is another alternative used in human medicine in cases in which ureteral stenting is not an option. SUB involves placing locking-loop nephrostomy and cystostomy catheters that are externalized by tunneling the tubes under the skin and connecting them via a special port. A cystectomy and nephroprostomy must be performed to prevent tension on the catheters. Although SUB may be a treatment option for ureteral trauma in future veterinary patients, it will likely be limited to very specific patients and, at this time, is only in the investigational stages.

Lacerations or ruptures of the bladder are repaired following debridement of necrotic or neoplastic tissues with a 1- or 2-layer continuous suture pattern. Up to 75% of the bladder can be removed and still maintain normal function. Notation of ureteral location relative to the injury should be made to prevent accidental transection or incorporation during repair.

Urethral lacerations or ruptures may need debridement, suturing, or urethral anastomosis with subsequent placement of a cystostomy tube and/or indwelling urinary catheter for days to weeks. Urine extravasation will cause delayed healing, and diversion of urine away from the site of injury may reduce the risk of stricture formation. Another option for urethral injury is a permanent urethrostomy proximal to the site of injury for which a prescrotal, scrotal, perineal, or antepubic urethrostomy may be indicated. A scrotal urethrostomy is the preferred technique in male dogs, prepubic urethrostomy in female dogs, and perineal urethrostomy in cats. A prepubic (antepubic) urethrostomy is considered a salvage procedure as it is difficult to perform and urinary continence will depend on adequate innervation of the lower urinary tract.
For less severe urethral injuries, an indwelling urinary catheter can be placed for 7–21 days to allow for healing of the urethra.\textsuperscript{107} Radiographs should be performed to document the location of the urethral catheter and to ensure it has not passed through the urethral defect.\textsuperscript{1} Complications of urethral trauma include strictures, ongoing urinary leakage, and urinary incontinence from damage to the vascular supply or innervation of the urethral sphincter.\textsuperscript{24}

Tube cystostomy is an alternative option for temporary or permanent urinary diversion in patients with severe bladder or urethral trauma or neoplasia.\textsuperscript{21, 24, 104, 107} Permanent cystostomy tube placement has been described in 7 dogs with urethral obstruction from suspected transitional cell carcinoma.\textsuperscript{110} The median survival for these dogs with the catheter in place was 106 days. No complications were documented during or following placement of the cystostomy catheters except for urinary tract infections, which resolved with antibiotics. The owners reported that the tubes were easily managed at home and minimal cleaning was required.\textsuperscript{110} A retrospective study evaluated the outcome and complications of 76 dogs and cats with cystostomy tubes. The 2 most common complications in this study were inadvertent tube removal and urinary tract infections. In most cases, the cystostomy tubes were easily replaced through the stoma following sedation or anesthesia.\textsuperscript{111}

A case series published in 2009 described a minimally invasive inguinal approach for cystostomy tube placement in 15 animals. The patients were placed in lateral recumbency with the dorsal pelvic limb retracted. The approach required minimal tissue dissection to locate the bladder and following tube placement a cystectomy was performed upon closure. This technique was reported to take generally less than 15 minutes and was utilized in 5 cats, which suffered from postrenal azotemia with no complications. The tubes were maintained for 10–49 days with no reported complications beyond what would be expected following the traditional tube cystostomy procedure.\textsuperscript{112}

The most common complications that occur following placement of cystostomy tubes include peristomal irritation and urinary tract infections. Damage to the cystostomy tube by the patient or loosening of the securing sutures can also occur and in some cases could result in uroabdomen if the tube is removed prematurely.\textsuperscript{111, 112}

Urinary bladder marsupialization is another reparative option, and often salvage procedure, for some patients in which the urethra cannot be repaired due to trauma or neoplasia; however, chronic cystitis and urinary tract infections are common sequela.\textsuperscript{113}

Complications, in general, following surgery to correct defects in the urinary tract include dehiscence of the incision with ongoing urine leakage, stricture formation of the ureter or urethra, or urinary incontinence.\textsuperscript{13, 24} Other conditions include unresolved azotemia, chemical peritonitis and urosepsis.\textsuperscript{4, 114, 115}

### Urosepsis and Antimicrobial Therapy

Urosepsis is uncommon in veterinary patients but can accompany uroabdomen if the patient has a concomitant urinary tract infection or received PD.\textsuperscript{4, 69} Cytology of the abdominal effusion may reveal intra- or extracellular bacteria and results of aerobic and anaerobic bacterial cultures of the abdominal effusion may show positive growth of organisms.\textsuperscript{114, 115} In the study describing 26 cats with uroperitoneum, the majority of the cats received empiric antimicrobial therapy. Three of the 5 cats for which aerobic bacterial cultures were performed had positive results on urine obtained from the peritoneum or bladder.\textsuperscript{4}

The use of antimicrobial in patients with uroabdomen without documented infection is clinician- and case-dependent in both the human and veterinary literature.\textsuperscript{4, 116–118} Antimicrobial therapy is indicated in high-risk patients with a predisposition to infection to reduce the development of urosepsis, but the use of antimicrobials in low-risk patients is controversial.\textsuperscript{117, 118} Patient risk factors that increase the likelihood of a pre-existing urinary tract infection include history of recurrent bacteriuria, recent urinary catheterization, chronic kidney failure, prostatitis, urolithiasis, upper or lower urinary tract neoplasia, or the presence of a neurogenic bladder.\textsuperscript{119–123} Additional predisposing circumstances include recent chemotherapy, treatment with glucocorticoids, or underlying immunosuppressive conditions such as hyperadrenocorticism, diabetes mellitus, feline leukemia, or feline immunodeficiency virus.\textsuperscript{106, 109, 112–114}

Hospitalized patients with increased metabolic demands following trauma or surgery are at risk for nosocomial infections.\textsuperscript{124}

Before administering antimicrobial therapy, samples for bacterial cultures should be obtained.\textsuperscript{125} Due to the progressive nature of sepsis, urinary diversion and prompt repair of the defect are recommended to remove the source of infection.\textsuperscript{116, 126} Antimicrobial choice in uroseptic patients is critical. Considerations must be given to the most common bacterial isolates from the urinary tract, patient factors such as renal and liver function, previous antimicrobial exposure, antimicrobial concentration at the site of infection, and pharmacodynamics of the antimicrobial.\textsuperscript{127–130} Mortality in people with urosepsis remains around 20–40%.\textsuperscript{112} To the authors’ knowledge, there are no veterinary studies that have published the prognosis for dogs or cats with urosepsis.
Uroabdomen in dogs and cats

Prognosis

The overall prognosis for small animals with uroabdomen will depend on aspects of each individual case. Factors impacting prognosis include the site and severity of the urinary tract insult, presence of other concomitant injuries, resolution of electrolyte and acid-base derangements during stabilization, restoration of adequate renal perfusion and function, and healing of the injured site of the urinary tract without complication.

Multiple injuries and lack of clinical signs can result in a delay in diagnosis of uroabdomen in patients that sustained trauma. In a human study evaluating 164 bladder ruptures in trauma patients, associated injuries were the most common cause for death; however, delayed diagnosis and treatment of ruptured bladders beyond 24 hours significantly increased the mortality rate. A delay in diagnosis of ureteral injuries is reported to have the largest impact on morbidity related to ureteral injury in human patients. Consideration and prompt recognition of urinary tract disruption is necessary to reduce morbidity and mortality; however, treatment of more emergent and critical conditions takes precedence during initial stabilization.

A study evaluating the prognosis of 20 dogs and 29 cats with urethral rupture found that the animals that had also sustained additional injuries were more likely to be euthanized or succumb to their concomitant injuries during the perioperative period. If the patients survived the perioperative period, their prognosis improved. In this study, the 2 most common reasons for euthanasia were due to the guarded prognosis or cost of treatment.

In the study of 26 cats with uroperitoneum, the prognosis for those cats treated for uroabdomen with no concomitant injuries was good with 61.5% discharged from the hospital. To the authors’ knowledge, there is no study to date that discusses the prognosis of dogs with uroabdomen. However, without treatment, death will usually occur within 3 days in dogs.

Conclusion

Uroabdomen is a life-threatening condition that requires rapid diagnosis and stabilization of electrolyte, acid-base disturbances, and azotemia before any consideration for advanced diagnostics, anesthesia, and surgery. Once the patient is deemed stable, diagnostics to determine the location of urinary tract disruption followed by surgical repair can be performed. Close monitoring in the postoperative period is important to document improvement or resolution of laboratory abnormalities and overall patient well-being. In addition, it is important to monitor for postoperative complications such as ongoing urine leakage from dehiscence of the surgical site, stricture formation, or progression to urosepsis.

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Footnotes
