HOW I TREAT...

The cat with a blocked bladder

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Pathogenesis of obstruction

Urethral obstruction (UO) is a potentially life-threatening manifestation of feline lower urinary tract disease. The male cat has a long and narrow urethra (compared to the female), and is much more likely to develop an obstruction. It has long been held that the presence of a physical obstruction, such as a calculus or urethral plug (or much less commonly stricture or neoplasia), is responsible for occluding the lumen of the urethra in these cases, but there is evidence to suggest that mechanical obstruction secondary to urethral spasm and edema may play an equally important role (1,2). These conditions are thought to be brought about by underlying feline idiopathic cystitis (FIC). The pathogenesis of FIC is still unclear but it appears to be a sterile inflammatory process, as attempts to consistently isolate a bacterial or viral cause have been unsuccessful.

Instead, extensive investigation into neurohumoral alterations in cats with FIC has demonstrated that the disease may be related to an imbalance between the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis brought about by stressful situations (3). This imbalance is thought to result in impaired blood flow and release of inflammatory mediators which cause edema, smooth muscle spasm and pain within the lower urinary tract. Pain, in turn, can contribute to the escalation of urethral smooth muscle dysfunction and urethral inflammation, thereby creating a vicious cycle. These conditions, either independently or in conjunction with a physical obstruction such as a plug or a stone, ultimately lead to urethral obstruction in cats.

KEY POINTS

- **Feline urethral obstruction may occur secondary to physical obstruction (stones, mucous plug) as well as functional obstruction (inflammation, urethral spasm, edema).**
- **Heavy sedation, lubricated flush, and appropriate technique are essential to ensure the risk of urethral trauma is minimized.**
- **Severe hyperkalemia is considered to be the most life-threatening aspect of obstruction.**
- **Awareness and monitoring for post-obstructive diuresis is important to maintain appropriate fluid balance.**
- **Analgesia and urethral relaxants are important components of therapy both during hospitalization and after discharge.**

Pathophysiology of obstruction

Complete obstruction of the urethra leads to the accumulation of urine and pressure within the urethra and urinary bladder which, once the tissue can no longer distend, will result in pressure necrosis and mucosal injury. Pressure within the urinary bladder is then transmitted up the ureters to the kidneys with subsequent reduction of glomerular filtration. Within 24-48 hours of obstruction the kidney's excretory ability ceases, resulting in an accumulation of blood urea nitrogen, creatinine, phosphorus,

potassium, and hydrogen ions in the blood, which largely contributes to the clinical signs associated with UO.

Uremia can cause depression, nausea, vomiting and anorexia. The combination of decreased food and/or water intake and ongoing gastrointestinal losses from vomiting and diarrhea can result in dehydration and potential for hypovolemia. Severe hyperkalemia is considered to be the most life-threatening aspect of UO because of its effects on the cardiovascular system. Elevations in serum potassium affect electrical conduction through the heart by diminishing the rate of depolarization, resulting in bradycardia. If the serum potassium level gets high enough electrical activity in the heart can cease altogether, resulting in asystole. Severe metabolic acidosis can lead to denaturing of proteins, enzymatic dysfunction and catecholamine hyposensitivity.

Given these changes, it seems likely that hypotension and cardiovascular collapse could develop in the latter stages of urethral obstruction. However, a study assessing blood pressure at the time of presentation in 28 blocked cats, which included some animals with significant illness, found no incidence of hypotension (4). Instead it was found that more severely affected patients (having higher blood urea nitrogen, creatinine, and potassium concentrations) tended to be normotensive, whereas less severely affect patients tended to be hypertensive, suggesting that several factors (*e.g.*, pain, stress) may have served to offset any tendency towards hypotension.

History and clinical signs

The classic history associated with UO involves a male cat which has been vocalizing and straining unproductively in the litterbox. However, these signs may be difficult to distinguish from a cat with FIC. Ideally, determination of whether urine is produced can help decide if UO has occurred. Unfortunately, cats with cystitis will often urinate very small amounts frequently, and sometimes outside the litter box, making it difficult to know for sure if the cat is producing urine. In addition, multi-cat households can present a challenge for owners to keep track of whether or not the cat has been urinating.

One distinguishing feature of UO *versus* cystitis is that affected cats start to show signs of systemic illness as the obstruction progresses; this could include vomiting, lethargy, anorexia, and abdominal pain which progress to changes in mentation and lateral recumbency. These signs are fairly non-specific if UO is not suspected, so obstruction should be considered as a differential for any sick male cat!

Clinical signs can vary considerably depending on the stage at which the patient is presented. Cats that present early may not have any striking physical exam findings aside from a firm, distended urinary bladder. In the "healthy" blocked cat this is the most definitive method to distinguish between obstruction and cystitis (as cats with cystitis should have a small, barely palpable bladder). If the obstruction has been present for more than 24 hours the patient may show signs of systemic illness such as dehydration, bradycardia, and hypothermia.

The presence of bradycardia in male cats should always raise concern for hyperkalemia, as the normal stress response to hospital presentation should result in tachycardia (although cats in septic or cardiogenic shock can also demonstrate bradycardia). In fact, the combination of bradycardia (HR < 140) and hypothermia (< 96.6°F/36°C) has been found to be 98% predictive of serum potassium level greater than 8 mEq/L in cats with urethral obstruction (5).

Initial diagnostics and stabilization

Presentation of the "sick" blocked cat warrants immediate medical attention. An IV catheter should be placed and initial blood samples for packed cell volume (PCV), total protein (TP) and blood gas or chemistry panel (which should include glucose, urea and creatinine) obtained if possible. Fluid therapy should be started immediately to support vascular volume and help dilute the serum potassium concentration, even if bladder decompression cannot be performed immediately. There is some debate as to the optimal type of fluid to use. Traditionally, 0.9% NaCl has been considered the fluid of choice because it has a greater dilutional effect on potassium; however it is also an acidifying solution which may serve to exacerbate metabolic acidosis.Conversely, balanced electrolyte solutions are alkalinizing but contain small amounts of potassium (typically 4-5 mEq/L) which may have less of a dilutional effect. A recent study comparing 0.9% NaCl and a balanced electrolyte solution showed no difference in outcome parameters (survival, length of stay) or in reduction of serum potassium levels, although acid-base anomalies corrected more rapidly in the latter group (6).

Overall it seems that the fluid chosen does not matter as long as an adequate volume is administered. If cardiovascular

collapse is present it may be necessary to administer crystalloid "shock doses" (40-60 mL/kg) in bolus fractions (1/4-1/3 of the calculated shock dose over 15-20 minutes, repeated as necessary) to rapidly restore vascular volume and reverse signs of cardiovascular instability. If resuscitation is not necessary, fluid rate should be based on replacement of dehydration added to the maintenance fluid requirement. If time does not allow more accurate determination of fluid rate, it is reasonable to start at a rate of 10 mL/kg/h in the initial stages, provided there is no indication of underlying heart disease.

An ECG should be obtained (even if the patient is not demonstrating bradycardia) to determine any effects hyperkalemia might be having on electrical conduction in the heart. Classic ECG changes associated with hyperkalemia include prolonged P-R interval, diminished or absent P waves (1), widened QRS complexes (2) and tall, tented T waves (3) ***(Figure 1)***. As hyperkalemia worsens, ECG changes can progress to atrial standstill, ventricular fibrillation or asystole. While de-obstruction and IV fluids will ultimately be the primary means of eliminating potassium and reversing the adverse affects of hyperkalemia, this process takes time. If the patient has significantly bradycardia (HR < 140) then immediate intervention to protect the heart (using calcium gluconate) and to promote intracellular shift of potassium (by regular insulin, dextrose, and/or sodium bicarbonate) should be employed ***(Table 1)***.

Given that calcium gluconate does not serve to decrease potassium levels, if it is administered the patient should also receive dextrose, or insulin and dextrose; note that dextrose should be given when insulin is administered to prevent the development of hypoglycemia.

Though controversial, cystocentesis can also be a part of initial stabilization by allowing immediate relief of pressure within the urinary tract and more rapid resumption of glomerular filtration. This could be especially important when a busy emergency department does not afford the time necessary to relieve the obstruction by passing a urinary catheter. In addition, only minimal sedation (or even no sedation) is usually necessary to perform cystocentesis (as opposed to heavy sedation/anesthesia for catheterization) and a "pure" urine sample can be obtained for urinalysis or urine culture. Finally, relieving the back-pressure against the obstruction (whether stone, plug or spasm) may make for easier passage of a urinary catheter. The major concern raised against performing cystocentesis in cats with urethral obstruction (UO) is the potential for tearing or rupture of a distended and friable bladder with resultant uroabdomen. A recent prospective study of 45 blocked cats demonstrated that development of clinically significant abdominal effusion, as evidenced by abdominal ultrasound, occurs rarely after cystocentesis and that this procedure can be safely performed (7).

Urethral catheterization

Passage of a urinary catheter to relieve a physical obstruction is generally considered to be essential in the management of UO. In order to optimize the likelihood of successful catheterization and minimize damage to the

Figure 1. Potential ECG changes associated with **(a)** moderate (approx. 6.0-8.0 mEq/L) and **(b)** severe (> 8.0 mEq/L) hyperkalemia.

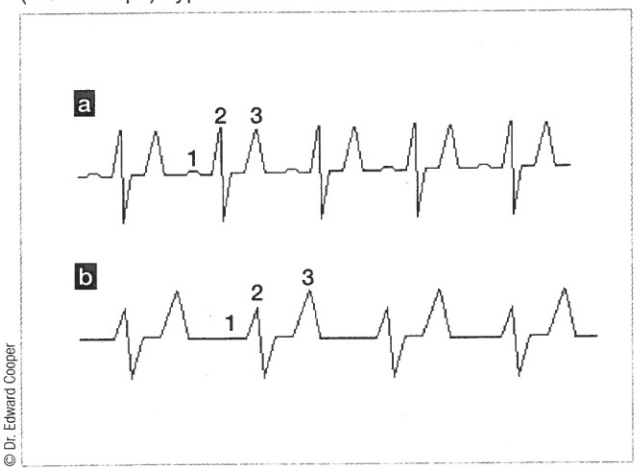

Figure 2. Catheter flushing apparatus, including two syringes, 3-way stopcock, extension tubing, sterile saline and sterile lubricant.

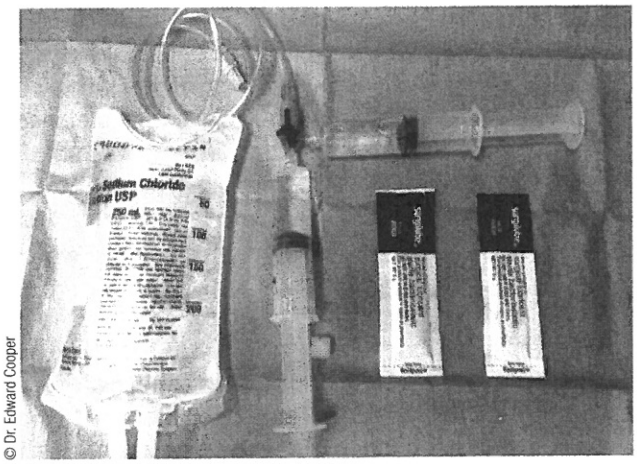

Table 1. Emergency dose rates for severe hyperkalemia.

Medication	Dose (IV)	Rate of administration	When to administer
Isotonic crystalloid	10-15 mL/kg 10 mL/kg/hr	15-20 minutes Constant rate infusion	Shock Initial replacement fluid rate
Calcium gluconate	50-150 mg/kg	Over 5 minute period	Bradycardia, major ECG changes
Regular (soluble) insulin	1 unit	IV bolus	If Ca gluconate given Potassium > 8 mEq/L
50% dextrose	0.5 g/kg	Over 3-5 minute period	If Ca gluconate given Potassium > 8 mEq/L
Sodium bicarbonate	1 mEq/kg	Over 5 minute period	Potassium > 10 mEq/L

urethra, heavy sedation/analgesia or anesthesia is recommended ***(Table 2)***. Vocalization or movement during catheterization attempts, reflecting insufficient sedation, is likely to be associated with significant urethral spasm and an increased risk of urethral trauma. Under these circumstances higher doses or additional medications should be given. Once the patient is sedated/anesthetized, the perineal area should be clipped, prepared and draped in order to minimize risk of contamination. An open-ended semi-rigid catheter (polypropylene or polytetrafluoroethylene) can be used to initially relieve the obstruction. Creating a mixture of saline and sterile lubricant (in a ratio of 5:1) as the flush solution will serve to deposit lubricant along the entire length of the urethra and potentially decrease urethral damage ***(Figure 2)***. Another helpful technique is to pull the prepuce caudally once the catheter is seeded in the penile urethra ***(Figure 3)***. This will straighten out the urethra, thus making passage of the catheter easier and less traumatic. Once the initial catheter is in place the urinary bladder can be emptied and flushed. Given that a polypropylene catheter is rigid and can cause significant urethral irritation, it should be withdrawn and replaced by a softer indwelling catheter (typically 3.5 or 5 Fr) which is then sutured in place. It has recently been demonstrated that use of a 3.5 Fr urinary catheter may be associated with less risk of immediate re-obstruction when compared to a 5 Fr catheter (8).

Once the patient has been stabilized and the obstruction relieved, it is important to obtain abdominal radiographs (perhaps even a single lateral view) encompassing the entire lower urinary tract to assess catheter placement and to identify the presence of calculi, as management will differ if stones are present.

Post-obstructive care

Fluid therapy and monitoring urine output are important aspects of post-obstructive care. Patients which have had prolonged obstruction are at risk for a post-obstructive diuresis resulting in massive urine production. This diuresis is thought to occur secondary to the accumulation of osmotically active substances in the blood, pressure necrosis, medullary washout and/or anti-diuretic hormone resistance brought about during the obstructive process.

One study has demonstrated that this can occur in up to 46% of cats in the post-obstructive period (9). While severity of azotemia was not found to be associated with likelihood, blood pH did have a significant negative correlation. It is very important to keep up with urinary losses in these patients, as the cat can quickly become severely dehydrated and hypovolemic; do not fear the high fluid rates required to keep up with losses in these patients!

Another potential concern is for inadequate urine production (< 1mL/kg/hr) after the obstruction is relieved, which may occur as a result of obstruction in the collection system or dehydration. True oliguria can occur as a result of progression to acute renal failure, but this appears to be very uncommon in urethral obstruction.

Another important aspect of post-obstructive care is analgesia and sedation. Cystitis and obstruction, in addition to urethral catheterization, are painful and

could be associated with risk of re-obstruction. Buprenorphine (0.01-0.02 mg/kg q8H) generally provides sufficient pain control and has the benefit of transmucosal administration if desired. If buprenorphine is not sufficient, fentanyl (2-4 µg/kg/hr) as a continuous rate infusion (CRI) is recommended. Because of the potential to cause excitability and hyperthermia, hydromorphine is typically avoided. Acepromazine (0.05 mg/kg IV/IM or 0.5 mg/kg PO) can provide adequate sedation to decrease stress and agitation, so long as the patient is stable (*i.e.,* resolution of dehydration/hypovolemia). The α-antagonist effects of acepromazine might promote urethral relaxation and decrease the risk of re-obstruction once the urinary catheter is removed.

Another frequent consideration in the post-obstructive period is whether the patient should be given antibiotics. It has traditionally been accepted that the incidence of bacterial infection in feline lower urinary tract disease is very low (< 2%), but more recent studies have suggested a higher incidence, ranging from 25-40% (10,11). A recent prospective study, specifically in patients with feline urethral obstruction, found zero positive cultures at presentation, but 6/18 cats (33%) developed urinary tract infections while catheterized (12). However, another recently completed study of 31 cats also showed no positive cultures at presentation, and only 13% (4/31) went on to develop a positive culture (13). Given the low incidence, performing a urine culture and sensitivity at the time of catheter removal is recommended to determine if a urinary tract infection (UTI) has been introduced. As there is significant potential for contamination during catheter removal, the practice of submitting the catheter tip for culture should be avoided.

Electrolytes and renal values should be monitored every 12-24 hours and should rapidly correct to normal. Typically if there is not significant reduction in renal values within 24 hours, complications (*e.g.*, renal failure, uroabdomen) may have occurred. Hypokalemia can develop (especially in patients with post-obstructive diuresis) and potassium should be supplemented accordingly. The urinary catheter should remain in place until the cat is clinically improved, blood work has normalized, post-obstructive diuresis has resolved, and urine is free of major debris, clots or plugs in order to help minimize the risk of immediate re-obstruction. Note that a closed catheter system is preferred, although in some situations an open catheter may be used if there is concern that the patient will become entangled. Once the catheter has been removed

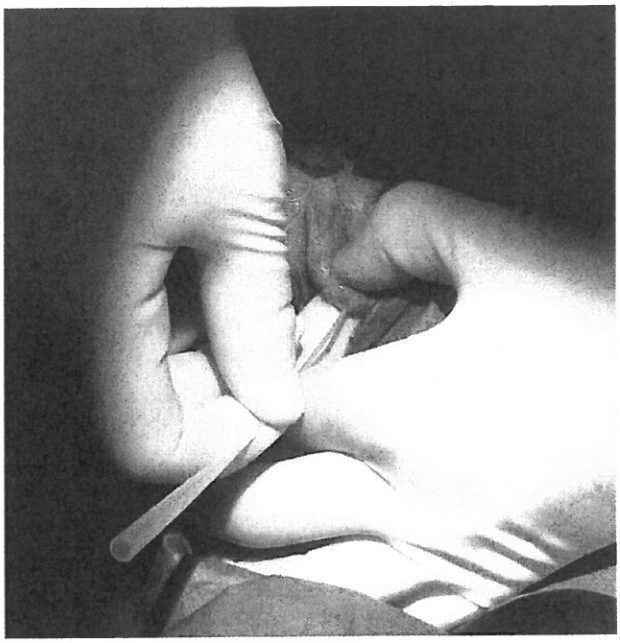

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Figure 3. Initial urinary catheter placement. The perineal region is firstly clipped, prepared and draped. Once the catheter is introduced into the distal urethra, the prepuce is pulled caudally to straighten the urethra and facilitate passage.

Table 2. Suggested anesthesia protocols when treating urethral obstruction.

Stable patient
• **Premedication/sedation** - Ketamine (5-10 mg/kg) + diazepam/midazolam (0.25-0.5 mg/kg) IV/IM or - Buprenorphine (0.01-0.02 mg/kg) and acepromazine (0.03-0.05 mg/kg) IV/IM • **Induction** - Propofol (1-4 mg/kg IV, to effect) • **Maintenance** - Inhalant anesthesia (isoflurane/sevoflurane)
Unstable patient*
• **Sedation** - Buprenorphine (0.01-0.02 mg/kg) + diazepam/midazolam (0.25-0.5 mg/kg) IV/IM or - Methadone (0.2-0.25 mg/kg) + diazepam/midazolam (0.25-0.5 mg/kg) IV/IM

*Typically general anesthesia is not only not required for very unstable cats (metabolically deranged, minimally responsive), it may have too much cardiovascular impact, and it is usually possible to unblock these patients under sedation alone.

the cat should be observed for 12-24 hours to ensure effective spontaneous urination prior to discharge.

Alternative management protocols

Unfortunately, the ability to provide the optimal treatment course outlined above may be limited by an owner's financial constraints. In addition, there is evidence that UO is as much a mechanical obstruction (urethral edema and spasm) as a physical one (plug or stone). A recent study demonstrated that pharmacological manipulation (analgesia and sedation), a low stress environment, and intermittent cystocentesis can result in spontaneous urination without the need for catheterization (14), the less invasive approach being offered in lieu of euthanasia when traditional treatment for UO was prohibited by financial constraints. Cats in need of emergency stabilization based on significant physical exam/metabolic derangements were excluded. Treatment involved administration of standardized doses of acepromazine and buprenorphine, decompressive cystocentesis, and subcutaneous fluids as necessary for up to four days. The cats were also placed in isolated housing with minimal handling to reduce stress associated with the hospital environment. The average cost of treatment was much lower than the cost for traditional UO management, and of 15 cats treated using this protocol a successful outcome (spontaneous urination and survival to discharge) was recorded in 11 (73%) cases. Major complications resulting in euthanasia included the development of uro-abdomen or hemo-abdomen, although there was no evidence of overt bladder tear/rupture on *postmortem* examination. Follow-up was performed at 3 days, 3 weeks, and 1 year after discharge; there did not appear to be a greater risk of immediate or long-term re-obstruction with this protocol when compared historically to traditional management.

These results suggest that this protocol could serve as an alternative to euthanasia based on financial constraints, but it cannot be recommended as an alternative to traditional management (which carries a reported success rate of 91-94%) as no direct comparison between the two has been made.

In some cases, financial limitations might preclude the ability to hospitalize for treatment. Under those circumstances it may be necessary to offer euthanasia, especially for severely affected cats (hypothermia, bradycardia, lateral recumbency, etc). For those patients presenting in the earlier stages of obstruction that are not yet significantly ill, it may be possible to provide care on an outpatient basis, though this should be reserved as a last resort. One option would be to provide sedation and analgesia (acepromazine and buprenorphine) and bladder decompression through either catheterization or cystocentesis. Catheterization would offer the benefit of removing any physical obstruction but could also result in urethral damage or irritation and an increased risk of re-obstruction. Cystocentesis would likely be less expensive to perform and less injurious to the urethra, but might only provide temporary relief if a physical obstruction is present.

With either approach, the patient would be discharged with recommendations as outlined below in the hopes that continued analgesia and sedation will allow for spontaneous urination to occur. Aside from anecdotal reports and clinical experience, there is no evidence to support the merits of either of these approaches, nor is there information regarding the likelihood of success or recurrence. The client would have to be well-informed of the potential for treatment failure and follow-up phone calls to determine response would be strongly recommended.

At-home care

Given the potential for recurrence, at-home care may be extremely important to help decrease the likelihood of re-obstruction either immediately or in the future. Continued analgesia and sedation after discharge can be helpful, with administration of acepromazine and buprenorphine for 5-7 days. For patients demonstrating significant straining/urethral spasm after catheter removal, it may also be beneficial to administer prazosin (0.25-0.5 mg q12-24H) as an α-1 antagonist and urethral relaxant. Antibiotics should only be dispensed based on results of urine culture taken at the time of catheter removal. Other recommendations which have been made to help decrease the risk of re-obstruction include increasing water intake by switching to wet food, flavoring the water, or using a running-water bowl.

Given the questionable role that urinary crystals play in the pathogenesis of obstruction, it is unclear whether dietary manipulation of urinary pH to address crystalluria is beneficial, but since stress may play a potential role in the pathogenesis of this disease, environmental enrichment may also help (15, 16).

Prognosis

Depending on the underlying cause, there is an approximately 25-40% incidence of recurrence with UO (2,11).

With a second obstructive episode it becomes more likely that there will be subsequent occurrences, at which point it may be necessary to consider perineal urethrostomy. This surgical procedure (which is outside the scope of this paper) can significantly decrease the likelihood of UO, but does not serve to resolve signs of underlying FIC. In addition, these patients may be at an increased risk for UTI, although a recent study of 86 cats that underwent perineal urethrostomy showed good long-term quality of life with minimal risk of recurrence (17).

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