Introduction

Feline urethral obstruction (UO) is a common complication of feline lower urinary tract disease (FLUTD) in male cats. Much of what is considered standard of care remains unchanged. Goals of therapy include patient stabilization, a diagnostic work-up to identify the underlying cause, restoration of urethral patency, supportive care, and long term management of the underlying disease. This session will address clinically applicable updates and controversies regarding care of feline UO. A complete review of UO is beyond the scope of this session.

Emergency Stabilization

Feline UO has the potential to cause severe morbidity and even mortality due to the resultant hyperkalemia, metabolic acidosis, cardiovascular compromise, arrhythmias, uremia, and acute kidney injury. Once obstruction occurs, these metabolic derangements occur within 24 hours and, if untreated, death ensues within 3-6 days. Despite this, evidence (Lee 2003) and clinical experience demonstrates that only a small percentage (~12%) of patients present severely metabolically affected. Still, most patients do benefit from some form of stabilization.

Fluid choice: Fluid therapy in the form of isotonic crystalloids is the cornerstone of stabilization. Traditionally, normal saline (0.9% NaCl) was considered the fluid of choice over concerns of exacerbating hyperkalemia through the administration of potassium containing fluids. However, evidence now supports that balanced isotonic crystalloids such as Lactated Ringer’s and Plasmalyte are not only safe, but probably more ideal. Two separate studies (Cunha 2010 and Drobatz 2008) demonstrated that balanced crystalloids corrected metabolic acidosis faster in cats with UO and did not delay time to normalization of potassium as compared to normal saline. These findings make sense within the context of fluid physiology. First, the amount of potassium in these solutions (4-5mEq/L) is proportionally negligible compared to total body potassium stores and plasma concentrations. Considering boluses in cats rarely exceed a blood volume (~250mL), clinicians are effectively giving a cat a single mEq of potassium during stabilization. Second, balanced isotonic solutions contain a buffer that combats metabolic acidosis whereas saline is acidifying due to its lack of a buffer, low strong ion difference, and supraphysiologic chloride content. Finally, inorganic acidosis potentiates hyperkalemia so correcting it helps lower the potassium concentration. Key point: reach for your balanced crystalloid of choice when treating blocked cats!

Fluid resuscitation and rate:

Anecdotally, the author has witnessed that clinicians tend to under-resuscitate blocked cats due to their fear of “fluid overload.” Concern over fluid overload stems from the fact that the cat’s shock organ is the lung and severe UO can cause acute kidney injury/failure. Although possible, fluid overload appears to be uncommon among blocked cats and is typically not fatal. In one published abstract (Ostroski 2013), a teaching hospital with a high annual case load of blocked cats (~80-100) only reported fluid overload in 17 cats over a 10 year period. Sixteen cats
survived to discharge and one was euthanized due to reobstruction. Curiously, 7 cats were diagnosed with underlying heart disease. Thus, it is important to consider occult hypertrophic cardiomyopathy if fluid overload does occur. This aforementioned study matches the author’s clinical experience in that fluid overload in blocked cats is uncommon and most cats with urethral obstruction need and can handle aggressive fluid therapy.

Consider titrated boluses (10-20ml/kg) of balanced crystalloids for patients with cardiovascular compromise and/or a higher initial rate (40-60ml/hour per cat) for the first 4-6 hours. Be cautious of diagnosing acute renal failure in an under-resuscitated cat, especially in the context of a concentrated urine specific gravity. Once the urine output increases (>0.5ml/kg/hr), fluid therapy may be tailored to the individual patient taking into account diuresis, replacement of dehydration, and ongoing losses from post-obstructive diuresis. It is important to remember that fluid therapy is dynamic and the patient’s needs can rapidly change. For example, although rates of 20-30ml/hour are appropriate for most cats following stabilization, patients that develop post-obstructive diuresis can require rates of 50-100ml/hour for a short period of time to maintain euvoolemia. For this reason, urine output monitoring every 2-4 hours via a closed collection system is recommended, especially in the face of severe metabolic compromise. Studies (Francis 2010 and Frohlich 2015) suggest that the presence of a metabolic acidosis is significantly correlated with the development of a post-obstructive diuresis. Unfortunately, the duration of significant diuresis is highly variable but usually relatively short lived (6-12 hours). The author recommends slowly tapering the fluid rate every 2-4 hours while closely monitoring perfusion parameters to prevent iatrogenically perpetuating significant polyuria. **Key point:** Don’t under-resuscitate blocked cats in fear of fluid overload.

### Hyperkalemia – ECG findings
Traditionally, studies on ECG changes associated with experimentally induced hyperkalemia in otherwise healthy animals produce predictable changes at escalating potassium concentrations that we all memorized early in our veterinary careers. Changes (in order of progression) include tall, spiked T waves, prolonged QRS complexes and P-R intervals, depressed P waves, atrial standstill, and eventual asystole or other terminal arrhythmias. One would logically assume that a tachycardia on physical exam could be used to rule out life threatening hyperkalemia among block cats. Unfortunately, this is not the case. In one study of cats with naturally occurring hyperkalemia, ECG abnormalities correlated with experimentally reported reference ranges less than half the time (Tag 2008). Furthermore, wide-complex tachyarrhythmia has been reported in cats with naturally occurring UO (Norman 2006). **Key point:** Tachycardia and ECG findings do not rule out metabolic compromise – obtaining a potassium concentration is imperative.

### Hyperkalemia – emergency management
The emergency management of hyperkalemia involves numerous strategies, all of which contribute small changes that add up significantly. First, calcium gluconate (1ml/kg of 10% solution) is immediately administered to restore the resting membrane potential of cardiomyocytes. Due to the short lived (10-15 minutes) mechanism of action, therapies that lower the potassium must quickly follow. It is imperative to recognize that fluids and restoration of urethral patency are the single most important part of managing hyperkalemia. Together, they dilute the potassium concentration, resolve metabolic acidosis (which shifts potassium intracellularly), and promote renal excretion of hydrogen ions. Fluids should be started
immediately – not after unblocking occurs. Since organizing supplies and technical help to perform the unblocking can take time, drugs are often given to temporarily shift potassium back into the cell, effectively lowering the intravenous potassium concentration. The co-administration of dextrose (1mL/kg of 50% solution) and insulin (0.25U/kg) intravenously is well established in the literature and together are capable of decreasing the potassium concentration by 0.5-1 mEq/L. In the author’s experience, rapid intervention with fluids, calcium gluconate, dextrose, and insulin have been sufficient to stabilize even the most severe hyperkalemias in a short amount of time. Some sources advocate the use of sodium bicarbonate intravenously at 1-2mEq/kg IV over 10-15 minutes. However, clinical studies have found it to be weak and inconsistent in reducing hyperkalemia (0-0.4mEq/L) relative to other therapies. Additionally, it takes 10-15 minutes to administer during which time other therapies and fluids cannot be administered. Finally, sodium bicarbonate has the potential for complications such as worsened ionized hypocalcemia (already a problem among blocked cats), hypotension, hypernatremia, and/or paradoxical acidosis in the central nervous system. For these reasons, the author rarely administers this drug. Curiously, evidence exists for the potential role of beta-2 agonists such as albuterol or terbutaline in lower potassium concentrations by up to 0.5-1.0mEq/L. For hyperkalemia refractory to standard therapies (fluids, insulin, and glucose), clinicians may want to consider these drugs prior to the administration of sodium bicarbonate due to their ease of administration and safety. Albuterol may be administered via an inhaler with spacer and mask (e.g. AeroKat) at standard dosages while terbutaline can be given subcutaneously or intramuscularly at 0.01mg/kg. **Key points:** Fluid therapy, calcium gluconate, insulin, and dextrose will appropriately manage most cases of hyperkalemia. For refractory hyperkalemia, perhaps beta-2 agonists are a better choice than sodium bicarbonate.

**Ultrasound findings**
With the increasing availability of ultrasound, more and more clinicians are using this diagnostic technique on patients with UO, either as a focal/emergency scan or a complete diagnostic exam. Given the medical adage, “look and you will find,” it is prudent to consider commonly observed pathologies and their clinical significance (or lack thereof). A very recent retrospective study (Nevins 2015) described the following common ultrasonographic findings among 87 cats with naturally occurring urethral obstruction (and without cystocentesis): echogenic urine sediment, bladder wall thickening, pericystic effusion, hyperechoic pericystic fat, and increased urinary echoes. Specific to the kidneys, mild pylectasia, renomegaly, perirenal effusion, hyperechoic perirenal fat, and ureteral dilation were observed. The authors found no association among any findings and the risk for recurrent UO. Most of the findings make sense within the current paradigm of UO pathophysiology; mainly an underlying inflammatory bladder disease that forms gross urinary constituents that causes mechanical obstruction and resultant ascending genitourinary pressure. One finding that is surprising and previously unreported is the presence of pericystic fluid. Although the exact nature of fluid and its cause (e.g. transmural leakage of urine vs pericystic fluid) has not been studied, bladder rupture appears to be uncommon and similar findings have been reported elsewhere (Cooper 2013 and Hall 2015). **Key points:** Don’t let ultrasound findings change your prognosis. Don’t assume bladder rupture if free fluid is found during an ultrasound exam.

Anecdotally, three other points are worth mentioning regarding ultrasound. First, the author has seen a limited number of cases where urinary sediment was so severe or a blood clot so large that
medical management failed and surgery was needed to clean out the bladder. Ultrasound may have a role in identifying these cats sooner. Second, the author has found ultrasound to be a highly valuable tool to screen for uroliths in cases where cost precludes abdominal radiographs. Finally, ultrasound can be used in place of radiographs to confirm proper indwelling urinary catheter placement.

**Cystocentesis**
Cystocentesis carries numerous theoretical advantages and disadvantages. Arguments for its use include obtaining a sterile sample for urinalysis and culture as well as decreasing pressure, which restores glomerular filtration and may make unblocking easier. Critics cite numerous risks such as the extravasation of urine through the needle track, bladder laceration, and aortic puncture or tear. Despite these concerns, clinical experience and studies (Cooper 2013 and Hall 2015) suggest that a single cystocentesis (by experienced personal) appears to be safe [key point]. Anecdotally, bladders that rupture tend to appear very diseased (purple to black) on gross inspection.

**SUPPORTIVE THERAPIES (POST-STABILIZATION PERIOD)**
**Does standard of care matter?**
Oftentimes, clients (and sometimes veterinarians) wonder if hospitalization for standard of care for feline UO really matters. “Isn’t there a cheaper option?” they ask. Although evidence is limited, standard of care does appear to improve outcomes and lower the risk of recurrent UO. One published study (Cooper 2010) of cats with naturally occurring UO described a protocol without urethral catheterization consisting of sedation, reduced environmental stress, and intermittent cystocentesis. Curiously 73.3% (11/15) cats cleared their obstruction and had a similar 30 day reobstruction rate as reported in the veterinary literature. Limitations of the study include a small study population (n=15) and the exclusion of cats that were severely sick on admission. Notably, those that did not clear their obstruction had severe consequences (uroabdomen or hemoabdomen) that led to euthanasia in three cats. Data by the author (Seitz 2016) demonstrates that standard of care (indwelling catheterization and hospitalization for supportive therapy) significantly reduces the incidence of recurrent UO within 30 days as compared to single catheterization and outpatient supportive care (10.9% vs 31.1%, respectively). Cats treated with outpatient care were 3.7 times \( (p = 0.0175, 95\% \text{ CI } 1.2 – 11.4) \) more likely to experience recurrent UO than cats treated with standard of care. Survival in this study was comparable to the current veterinary literature. **Key Points:** Standard of care is superior to alternative therapies regarding risk for recurrence and survival to discharge. However, alternative therapies appear reasonable if cost prohibits standard of care.

**Catheter size:**
In humans, consensus guidelines by the CDC (Gould 2009, pp12) recommend “the smallest bore catheter possible, consistent with good drainage, to minimize bladder neck and urethral trauma.” In male cats, a size 3.5 Fr catheter fulfills these criteria most of the time. A recent veterinary study (Hetrick 2013) supported this recommendation by documenting a two-fold increased risk for re-obstruction within the first 30 days of discharge from hospitalization whenever a size 5 Fr indwelling catheter was used compared to a size 3.5 Fr. However, a different study (Eisenberg 2013) did not find catheter size influenced risk for recurrence. A larger catheter size (e.g., 5Fr) may be beneficial in patients with large amounts of urinary constituents that result in obstruction
of urine flow of smaller catheters. Further studies are needed to better determine the advantages and risks of indwelling catheter size in patients with FUO. **Key Point:** It is reasonable to start with the smallest catheter size possible; however, if it becomes obstructed with urinary constituents, a larger size may be considered.

Although not considered new information, it should be noted that catheter material choice is established in the literature (Lees 1980 and Gould 2009). Although rigid catheters made of polypropylene (e.g. “Tomcat” catheters) are appropriate for the initial unblocking, they contribute to worsening hematuria and severe histologic lesions relative to softer materials such as polyvinyl, silicone, or latex. **Key point:** More appropriate indwelling catheters include *red rubber* and *Slippery Sam* catheters.

**Catheterization duration:**
Evidence is greatly conflicted regarding the ideal duration of catheterization. Two studies are in conflict as to whether duration of catheterization influences risk for urethral obstruction (Hetrick 2013 and Eisenberg 2013). CDC guidelines (Gould 2009) and veterinary evidence (Hugonnard 2015) support the use of shortened indwelling times in an effort to reduce the risk of bacteriuria and catheter-associated infections. **Key point:** At this time, catheterization duration should reflect individual patient needs with respect to monitoring urine output, clearing gross urinary debris, minimizing iatrogenic trauma, and minimizing infection.

**Antibiotics:**
The discussion of antibiotic use in cats with FLUTD and UO can get heated really quickly. Like many treatments in veterinary medicine, the author prefers an evidence-based approach. Rather than applying a rigid position to all patients, consider the risk factors unique to each patient and elucidate those factors with appropriate diagnostics. For example, it is well established that the incidence of bacterial infection is incredibly low (<2%) in otherwise healthy male cats with UO. In contrast, the incidence of infection dramatically increases if you are a cat that is over the age of 10 or living in Norway. Unfortunately, the primary treatment for urethral obstruction (i.e. urethral catheterization) increases the risk for infection as well. However, the risk for infection is still relatively low if appropriate sterile technique and catheter care is used and the catheter is pulled when medically appropriate. Finally, it is well established that antibiotics do NOT prevent catheter-associated infection, and, if it is to occur, the bug is more likely to be resistant. As a result, consider the following practical points when deciding on the use of antibiotics:

- The routine use of empirical antibiotics in young, otherwise healthy male cats living in the United States is not necessary – even after catheterization.
- Perform appropriate diagnostics. Perform a urinalysis at admission and ideally a urine culture (obtained via cystocentesis) after catheter removal to help detect the small percentage of cats that naturally have or iatrogenically develop bacterial cystitis.
- Identify risk factors that increase the potential for an infection, mainly being female, inappropriate catheter technique, or prolonged catheterization time. If a risk factor is present, urine culture becomes even more important.
- If empirical use of antibiotics are to be used due to the client declining standard of care (which includes appropriate diagnostics), do not start the antibiotic while the catheter is indwelling.
- If infection is suspected or confirmed while the catheter is indwelling, the antibiotic may be started.

**Urethral relaxant drugs**

Drugs that relax the urethra are commonly used in the management of UO. Drugs can be divided based on their ability to relax the smooth-muscle containing proximal urethra (e.g. prazosin, phenoxybenzamine, and acepromazine) or the skeletal-muscle containing distal portion (benzodiazepines). Although these drugs have a strong theoretical role, evidence that they affect outcomes by reducing the risk for obstruction is lacking. One double-blinded, placebo controlled study (Thomas 2012) presented in abstract form failed to show a reduction in the recurrence rate of UO in cats treated with prazosin one month after initial obstruction. However, the study appeared underpowered given its treatment group size (20 and 16) and its relatively low incidence for obstruction (5-8%) relative to other studies. No other peer reviewed clinical evidence exists. One retrospective study did demonstrate that prazosin may be superior to phenoxybenzamine in preventing recurrent UO (Hetrick 2013). However, the study design does not allow one to conclude that urethral relaxants in general preventing recurrent UO due to the retrospective nature and lack of a control. Although efficacy is lacking, these drugs due appear safe. At this time, the author commonly uses a benzodiazepine (with pain medications and other drugs) for sedation during the initial unblocking. Acepromazine can be considered in hemodynamically stable cats during hospitalization, as well. At this time, the author does discharge all patients with prazosin and a pain medication (e.g. buprenorphine) but recognizes evidence is lacking for the former.

**Intravesicular drugs**

Current standard of care involves instillation of isotonic fluids into the bladder to flush gross urinary constituents. However, intravesicular drugs have fallen out of favor over time due to their lack of efficacy or theoretical concern over increased permeability when the bladder wall is inflamed. Evidence still does not support the instillation of antibiotics, antiseptics, pain medications, or anti-inflammatories. However, one randomized, blinded, placebo-controlled pilot study (Bradley 2014) recently documented a reduction in incidence of recurrent UO that approached significance (P = 0.06) seven days after instillation of an intravesicular glycosaminoglycan (GAG). On the contrary, a larger randomized, blinded, controlled study using a different GAG product did not find a reduction of clinical signs or incidence of recurrence (Dellile 2015). Given the possible role of a defective GAG layer of the uroepithelium in cats with idiopathic cystitis, further research is indicated. However, at this time, research does not support the widespread application of this practice.

**References (alphabetical order).**
