**Urine a Mess: Disorders of Micturition**

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Micturition refers to the process of storing and periodically voiding urine. Disorders of urine storage usually lead to urinary incontinence, whereas disruption of urine voiding leads to incomplete emptying, dysuria, or urine retention. Micturition is a complex integration of central, sympathetic, parasympathetic, and somatic nervous systems, with resultant muscular activity. The two functional units of the lower urinary tract include the reservoir/pump (urinary bladder) and the continence/conduit (urethra). The urinary bladder and proximal urethra are composed of smooth muscle and are thus under autonomic nervous system control while the distal urethra is composed of skeletal muscle and thus under somatic nervous system control.

**DISORDERS OF MICTURITION**

There are several different ways of classifying including problems with storage vs. voiding, if the urination problem occurs with a full bladder vs. empty bladder, and whether the urination problem is neurogenic vs. myogenic in origin. It is important to establish status of urinary bladder contractile force and patency of urethral outlet, determine whether disorder is primarily neurogenic or myogenic, and determine underlying etiology or contributing factors. In addition to routine collection of historical information and performing a complete physical examination, a complete neurological examination and rectal palpation should be performed. Have the owners describe what the pet is doing and whether there is a good urine stream or not. If possible observe urination or have the owners’ video record the pet urinating. Depending on the underlying disorder of micturition additional diagnostic testing should always include a urinalysis and urine culture. Laboratory evaluation may include a CBC, biochemical analysis, and infectious disease testing. Abdominal imaging by survey radiography and possibly ultrasonography or contrast radiography should be considered. Cystoscopy or exploratory laparotomy may also be considered.

**Problems with Storage**

**Bladder Overactivity**

Bladder overactivity occurs due to hyperexcitability of the storage phase. This results in inability to permit adequate bladder filling because of urgency. Patients have increased frequency of urination, pollakiuria, inappropriate urination. Often urethral irritation or spasm is present. Examples of bladder hyperactivity include cystitis, urocystolithiasis, chemical stimulation (cyclophosphamide). The treatment is to relax bladder using antimuscarinic agents (propantheline, oxybutynin, tolterodine) and antispasmodic agent (oxybutynin, flavoxate, tolterodine). These drugs decrease detrusor activity and have urethral anti-spasmodic effects. Other drugs may help with refractory incontinence by increasing urine storage including tricyclic antidepressants such as imipramine, amitriptyline. These may improve bladder storage by several mechanisms including anticholinergic, alpha-adrenergic, and beta-adrenergic effects.

**Bladder Atony**

Bladder atony may be due to neurogenic or myogenic causes. Bladder atony is associated with bladder overdistention but the patient does not posture to urinate. The treatment is to stimulate bladder contraction. This should only be done if the urethra is relaxed pharmacologically as well. Manage large over-distended bladder with urinary catheterization. Pharmacologically, bethanechol is a parasympathomimetic with direct cholinergic activity that stimulates or augments smooth muscle contraction. Metoclopramide has been shown to stimulate canine ureteral smooth muscle in vitro and anecdotally to stimulate bladder contraction in human beings with bladder atony associated with diabetes mellitus. It appears to stimulate bladder contraction in some dogs and cats.
Problems with Voiding

Increased Outlet Resistance
Inability to void due to increased outlet resistance may occur because of mechanical problems (e.g., urethral obstruction from a stone or mass) or functional problems (e.g., urethral spasm or neurogenic). The treatment is to relieve the urethral obstruction or relax the urethra if neurogenic.

Relieve the obstruction - The urethral obstruction should be relieved by inserting a urethral catheter that may be left in place or performed intermittently or by repeated cystocentesis.

Relax the urethra - Urethral relaxation is accomplished by administering sympatholytic agents that antagonize alpha adrenergic receptors (e.g., phenoxybenzamine, prazosin, tamsulosin). Tamsulosin is an effective drug that is administered once a day and builds up in prostatic and urethral smooth muscle tissue. Skeletal muscle relaxants (e.g., diazepam, dantrolene, baclofen) may relax the urethral skeletal muscle (external urethral sphincter); however, they have less effect than alpha adrenergic blockers.

Urethral stent - In patients with urethral obstruction due to neoplasia, a urethral stent may be placed. Usually self-expanding metallic stents composed of nitinol are used. These are placed with fluoroscopic guidance. Most dogs are incontinent after placement as many transitional cell carcinomas involve the entire length of the urethra.

Low profile cystostomy catheter - A low profile cystostomy catheter is a mushroom-tipped catheter that is surgically implanted into the urinary bladder through the ventral abdominal wall lateral to midline. A cystopexy is also performed. The catheter sits just above the skin surface and contains 1 or 2 valves to prevent leakage. It provides urinary diversion; however, owners must empty the urinary bladder 2 to 3 times per day.

Paradoxical Incontinence
Paradoxical incontinence occurs when there is outflow obstruction resulting in bladder overdistention. The increased bladder pressure results in “leaking” of urine through or around obstruction. Usually the patient dribbles urine with a full bladder and is unable to void. It may be due to functional or mechanical outflow obstruction and is often associated with bladder atony.

Decreased Outlet Resistance
Decreased urethral tone and outlet resistance results in incontinence, which may be neurogenic, myogenic, or anatomic in origin. The most common cause is urethral sphincter mechanism incompetency in female dogs.

Ectopic ureters - Normally, the ureters enter at the trigone; however, occasionally they may terminate distally. They may either be extramural (where the ureter bypasses its normal insertion and inserts into the urethra or vagina at a distal point) or, more commonly, intramural (where the ureter enters the bladder at the trigone but tunnels in the wall before opening). Extramural ectopic ureters are surgically corrected. Intramural ectopic ureters may also be surgically corrected; however, laser ablation of the medial wall results in better continence (85% vs. 65%).

Urinary incontinence - Urinary incontinence refers to the unconscious release of urine and is most often due to urethral sphincter mechanism incompetency (USMI). It is uncommon in male dogs and male and female cats, but may occur in up to 20% of spayed female dogs. Usually urination while awake is normal.

Treatment: Pharmacologically - The treatment of urinary incontinence is to stimulate the urethral smooth muscle resulting in increased tone of the internal urethral sphincter. Administration of sympathomimetics (e.g., alpha agonists: phenylpropanolamine) results in continence in 85–90% of patients. Once a day treatment may be as effective as three times a day administration and is associated with fewer side effects. Estrogen replacement therapy (estriol, diethylstilbestrol, Premarin) may increase alpha adrenergic receptor responsiveness and improve urethral vascularity and other mucosal characteristics. They are safe and reasonably effective (40–65%); however, estriol (Incurin) is reported to have a 93% excellent response rate. Gonadotropin releasing hormone (GnRH) analogs have also been used. In ovariectomized dogs, chronically unsuppressed FSH and LH release (due to lack of negative feedback) may contribute to urinary incontinence. Administration of GnRH analogs paradoxically
reduces FSH and LH over time. It was found effective in 12/13 dogs in one study and in another study 9/23 dogs were continent from 70–575 days with another 10/23 having partial response; however, the 23 dogs also responded to PPA.

Treatment: Non-pharmacologically - In patients with USMI that are unresponsive to pharmacological therapy, there are several potential treatments. Urethral bulking involves injection of an agent submucosally in the proximal urethra via cystoscopy. It is thought to create artificial urethral cushions improving urethral closure (coaptation). It may also function as central filler volume increasing length of smooth muscle fibers and closure power of internal urethral sphincter. There are no bulking agents available for use in veterinary medicine. Historically, glutaraldehyde cross-linked collagen was used, but has been withdrawn from market. A study with polydimethylsiloxane has promising results. Artificial sphincter/urethral occluding device is similar to a blood pressure or vascular cuff that is placed surgically around proximal urethra with a loose fit. A tube connects the device with a subcutaneously implanted injection port providing a means to increase pressure within the device and therefore urethral pressure in area of internal urethral sphincter. Continence rates are high; however, they may require adjustment with time. Although surgical techniques (e.g., slings, plication, culposuspension) are available, long term continence rates are low.

Reflex Dyssynergia
Reflex dyssynergia refers to an incoordination between bladder contraction and urethral relaxation. The patient usually postures normally, initiates a good stream, but stream stops yet animal continues to posture and attempt to void. Treatment involves relaxing urethra as described. If bladder does not completely empty despite urethral relaxation, then add bladder stimulant.

Table. Drugs used to manage dogs and cats with micturition disorders.

<table>
<thead>
<tr>
<th>Agent</th>
<th>Mechanism of action</th>
<th>Recommended dosage</th>
<th>Adverse effects</th>
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<tr>
<td><strong>Agents used to increase urinary bladder contractility</strong></td>
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<tr>
<td>Bethanechol</td>
<td>Parasympathomimetic; direct cholinergic activity</td>
<td>D: 5–25 mg PO q8h&lt;br&gt;C: 1.25–7.5 mg PO q8h</td>
<td>Nausea, vomiting, salivation</td>
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<tr>
<td>Metoclopramide</td>
<td>Prokinetic; sensitizes to acetylcholine</td>
<td>D, C: 0.2–0.5 mg/kg PO q8h</td>
<td>Behavior changes</td>
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<tr>
<td><strong>Agents used to decrease urinary bladder contractility</strong></td>
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<tr>
<td>Propantheline</td>
<td>Parasympatholytic; acetylcholine blockade</td>
<td>D: 7.5–30 mg PO q8h&lt;br&gt;C: 5–7.5 mg PO q8h or 7.5 mg PO q72h</td>
<td>Nausea, vomiting, constipation, sedation, increased ocular pressure</td>
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<tr>
<td>Oxybutynin</td>
<td>Parasympatholytic; antispasmodic; detrusor relaxation</td>
<td>D: 1.25–5 mg PO q8–12h&lt;br&gt;C: 0.5–1.25 mg PO q8–12h</td>
<td>Nausea, vomiting, urine retention, diarrhea, sedation</td>
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<tr>
<td>Flavoxate</td>
<td>Direct smooth-muscle relaxant</td>
<td>D: 100–200 mg PO q6–8h</td>
<td>Weakness</td>
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<tr>
<td>Dicyclomine</td>
<td>Anti-muscarinic</td>
<td>D: 10 mg PO q6–8h</td>
<td>Nausea, vomiting, constipation, sedation, increased ocular pressure</td>
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<tr>
<td>Imipramine</td>
<td>Tricyclic antidepressant with anticholinergic, alpha- and beta-agonist effects, detrusor smooth muscle relaxation and urethral muscle contraction</td>
<td>D: 5–15 mg PO q12h&lt;br&gt;C: 2.5–5 mg PO q12h</td>
<td>Seizures, tremors, tachycardia, hyperexcitability</td>
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<tr>
<td>Amitriptyline</td>
<td>Tricyclic anti-depressant</td>
<td>D: 2.2–4.4 mg/kg PO</td>
<td>Sedation,</td>
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<tr>
<td>Agents used to increase urethral resistance</td>
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<td><strong>Estriol (Incurin)</strong></td>
<td>Reproductive hormone</td>
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<tr>
<td><em>D</em>: 0.5–2 mg PO q24h initially; followed by 0.5–2 mg PO q2–3d</td>
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<tr>
<td><strong>DES</strong></td>
<td>Reproductive hormone</td>
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<tr>
<td><em>D</em> (females): 0.1–1 mg PO q24h for 5 days (approximately 0.2 mg/kg) followed by 0.1–1 mg PO q7d</td>
<td>Signs of estrus, bone marrow suppression</td>
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<tr>
<td><strong>Premarin</strong></td>
<td>Reproductive hormone</td>
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<tr>
<td><em>D</em>: 20 mcg/kg q24hr x 7–10d; then q1–3d</td>
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<tr>
<td><strong>Testosterone propionate</strong></td>
<td>Reproductive hormone</td>
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<tr>
<td><em>D</em> (males): 2.2 mg/kg SQ or IM q2–3d</td>
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<td><em>C</em> (males): 5–10 mg IM as needed</td>
<td>Aggression, prostatic disease, perineal hernia</td>
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<td><strong>Testosterone cypionate</strong></td>
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<td><em>D</em> (males): 2.2 mg/kg IM q30d or 200 mg IM q30 d</td>
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<tr>
<td><strong>Phenylpropanolamine</strong></td>
<td>Alpha agonist; urethral smooth muscle contraction</td>
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<tr>
<td><em>D</em>: 12.5–50 mg PO q8h; 1–2 mg/kg PO q8h</td>
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<td><em>C</em>: 1.0–1.5 mg/kg PO q8h</td>
<td>Anxiety, cardiac arrhythmias, anorexia, hypertension</td>
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<tr>
<td><strong>Ephedrine</strong></td>
<td>Alpha agonist; urethral smooth muscle contraction</td>
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<tr>
<td><em>D</em>: 1.2 mg/kg PO q8h or 5–15 mg PO q8h</td>
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<tr>
<td><em>C</em>: 2–4 mg/kg PO q6–12h or 2–4 mg PO q8h</td>
<td>Anxiety, cardiac arrhythmias, hypertension</td>
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<thead>
<tr>
<th>Agents used to decrease urethral resistance</th>
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<tr>
<td><strong>Phenoxybenzamine</strong></td>
<td>Alpha antagonist; urethral smooth muscle relaxation</td>
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<tr>
<td><em>D</em>: 5–15 mg PO q12h</td>
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<tr>
<td><em>C</em>: 2.5–10 mg PO q24h</td>
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<tr>
<td><em>D, C</em>: 0.25 mg/kg PO q12h</td>
<td>Hypotension, tachycardia, vomiting, diarrhea, increased intraocular pressure</td>
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<tr>
<td><strong>Prazosin</strong></td>
<td>Alpha antagonist; urethral smooth muscle relaxation</td>
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<tr>
<td><em>D</em>: 1 mg/15kg PO q12–24hr</td>
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<tr>
<td><em>C</em>: 0.25–0.5 mg PO q12–24h</td>
<td>Hypotension</td>
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<tr>
<td><strong>Tamsulocin</strong></td>
<td>Alpha antagonist, urethral smooth muscle relaxation</td>
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<tr>
<td><em>D</em>: 0.03–0.2 mg/10kg q24h</td>
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<tr>
<td><em>C</em>: 0.02–0.04 mg/kg q12–24h</td>
<td>Hypotension</td>
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<tr>
<td><strong>Doxazosin</strong></td>
<td>Alpha antagonist, urethral smooth muscle relaxation</td>
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<tr>
<td><em>D</em>: 0.1–1.0 mg/kg PO q24h</td>
<td>Hypotension</td>
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<tr>
<td><strong>Terazosin</strong></td>
<td>Alpha antagonist; urethral smooth muscle relaxation</td>
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<tr>
<td><em>D</em>: 0.5–5 mg PO q12–24hr</td>
<td>Hypotension</td>
</tr>
<tr>
<td><strong>Terazosin</strong></td>
<td>Alpha antagonist, urethral smooth muscle relaxation</td>
</tr>
<tr>
<td><em>D</em>: 0.1–1.0 mg/kg PO q24h</td>
<td>Hypotension</td>
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<tr>
<td>Drug</td>
<td>Mode of action</td>
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<tr>
<td>Fiduxosin</td>
<td>Alpha antagonist, urethral smooth muscle relaxation</td>
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<tr>
<td>Diazepam</td>
<td>Striated muscle relaxation; central nervous system depressive effect</td>
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<tr>
<td>Dantrolene</td>
<td>Striated muscle relaxation; direct action</td>
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<tr>
<td>Acepromazine</td>
<td>Urethral muscle relaxation by neuroleptic effect; alpha antagonism</td>
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<tr>
<td>Aminopromazine</td>
<td>Smooth muscle relaxation</td>
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**REFERENCES**

Urine Agony: Feline Lower Urinary Tract Disease
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Prevalence of lower urinary tract disease is more common in cats between 1 and 10 years of age; whereas in dogs, the prevalence increases with advancing age. In cats greater than 10 years of age, bacterial urinary tract infection is most common. In young cats, idiopathic lower urinary tract disease occurs most commonly. Urinary tract infections and urolithiasis has been discussed previously in this conference; therefore, this discussion will focus on feline idiopathic cystitis (FIC).

What is the Pathogenesis of FIC?
By definition, FIC is an idiopathic disease and therefore the underlying cause is unknown. As noted earlier, it is entirely possible (and perhaps even probable) that FIC is not a single entity, but rather a syndrome that may have more than one underlying cause. This, for example, is evident in the ongoing search for a potential viral role in some cases of FIC. Nevertheless, in a number of different studies, both local bladder abnormalities and/or neurohormonal changes have been observed in at least a proportion of cats affected by FIC. While these changes are hard to interpret, and again it can be difficult to differentiate cause from effect (and sometimes even incidental observation), they do support the concept of complex underlying abnormalities and predispositions that may contribute to the development of FIC.

There are several proposed local bladder abnormalities in the pathogenesis of FIC. Studies in cats with idiopathic cystitis have shown that as in humans with interstitial cystitis, there is a decreased concentration of glycosaminoglycans (GAGs) in the urine of affected cats. Other local bladder factors have also been identified that may have a role to play in the pathogenesis of FIC. These include: an altered tissue or and/or urine concentration of inflammatory or other bioactive molecules such as complement c4a, thioredoxin, NF-κB p65, galeactin-7, I-FABP, fibronectin, and trefoil factor 2; mucosal muscarinic receptors have been reported to have increased sensitivity in cats with FIC, which could potentially enhance smooth muscle spontaneous contraction, although evidence of an overactive bladder has not been found in association with FIC; increased bladder tissue concentrations of norepinephrine and an increase in maximum urethral pressures and urethral closure pressures in affected cats; histological changes in the bladder wall including oedema, haemorrhage, vasodilation, occasionally ulceration, and a variable increase in the number of mast cells; there is evidence to support the presence of neurogenic inflammation and mediators of pain and inflammation in the bladder, with evidence of increased expression of transmitters such as ATP and nitric oxide, altered expression of purinergic receptors, increased numbers of substance P containing neurons, increased expression of high affinity substance P...
receptors, and increased excitability of afferent bladder neurons, with evidence that urothelial cells
themselves may be involved in the process.

As in humans with interstitial cystitis, a number of neuro-hormonal abnormalities have been
detected in cats with FIC that might have a role to play in the pathogenesis of the condition. These
observations include: an increase in plasma norepinephrine and dihydroxylphenylalanine in FIC cats
compared with normal cats, but without a concomitant increase in cortisol or adrenocorticotrophic
hormone (ACTH); an increase in tyrosine hydroxylase immunoreactivity in the locus coeruleus of the
brain of cats with FIC (during apparent quiescent periods), further supporting a role for increased
sympathetic activity in cats with FIC; potential adrenal insufficiency in cats with FIC evidenced by
significantly reduced responses to ACTH compared with healthy cats, and reduced volume of their
adrenal glands; differences in responses to the α2-adrenergic agent medetomidine in FIC cats compared
with normal cats. Collectively, these findings (although performed in a limited number of cats) lend
support to the fact that FIC appears to be associated with a stress response in many cats, but also suggests
an uncoupling of the normal stress responses with increased sympathetic stimulation but suppressed
adrenocortical responses.

**CLINICAL SIGNS OF LOWER URINARY TRACT DISEASE**
Causes of lower urinary tract disease in cats present with similar clinical signs including, but not limited
to pollakiuria, hematuria, stranguria, and inappropriate urination.

**WHAT ARE RISK FACTORS FOR FIC?**
Data from studies are conflicting for the most part. Some studies show a higher risk in males while others
show no gender predisposition. Most studies do not show a breed predilection; however, one study did
show a predisposition in long-haired cats. In another study, the following factors were found to be
associated with development of FIC: being more fearful than other cats in the same household, being
more nervous than other cats in the household, having a lower water intake, partaking in less hunting
activity, having lower activity levels, using a litter box, moving house, hiding when unknown visitors are
in the house, having a higher body condition score, and having less access to an outdoor environment.
Additionally, it is not uncommon for cats with FIC to have one or more other chronic diseases such as
inflammatory bowel disease, respiratory disease, and behavioral disorders.

**DIAGNOSTIC TESTING WITH LOWER URINARY TRACT SIGNS**
CBC and biochemical analysis are normal unless urethral obstruction is present. Urinalysis reveals
hematuria; however, pyuria and bacteriuria may be present with UTI. Urine culture is negative unless
UTI is present. Abdominal radiography and ultrasonography may be normal unless uroliths are present.
In cats with FIC, cystoscopy reveals small pin-point hemorrhages called glomerulations and bladder wall
biopsy often reveals submucosal edema, mucosal ulceration, possible submucosal inflammation, and
possible fibrosis. FIC is a diagnosis of exclusion.

**TREATMENT OF LOWER URINARY TRACT DISEASE**
**Urethral Obstruction**
Urethral obstruction may occur from uroliths or urethral plugs. Matrix-crystalline urethral plugs are
found only in male cats and approximately 84% of matrix-crystalline plugs contain a mineral component
with struvite being the most common mineral present. Uroliths have been discussed previously. Urethral
obstruction results in dehydration, azotemia, metabolic acidosis, hyperphosphatemia, hyperkalemia, and
eventually death. Treatment involves rehydration, relieving the urethral obstruction, and managing
hyperkalemia. After relieving urethral obstruction, an indwelling urinary catheter may be required. If
inserted, use a closed collection system, do not administer antimicrobial agents, and do administer
urethral relaxants (alpha adrenergic blockers).
Non-Obstructive Idiopathic Lower Urinary Tract Disease
There have been dozens of proposed treatments for cats with lower urinary tract disease; very few have undergone evaluation in a randomized controlled clinical trial.

Antimicrobial Agents
The role of microbial agents in feline lower urinary tract disease is controversial. In young adult cats evaluated at university referral hospitals, the incidence of bacterial urinary tract infection is 1% or less; however, approximately 1 in 3 cats seen at primary care facilities in Norway had bacterial urinary tract infection. Furthermore, older adult cats are more likely to have a bacterial urinary tract infection and as many as 50% of older cats with lower urinary tract signs have a bacterial urinary tract infection. Unless a UTI is present, administration of an antimicrobial agent is not warranted.

Urinary Tract Antiseptics and Analgesics
Methenamine and methylene blue are not indicated in cats as they may induce metabolic acidosis and Heinz body anemia. Phenazopyridine is an over the counter preparation available for use by women with recurrent vaginitis/cystitis that causes Heinz body anemia in cats.

Smooth Muscle and Skeletal Muscle Relaxants
Many cats with FIC have urge incontinence and inappropriate urination. Propantheline, an anticholinergic agent, minimizes force and frequency of uncontrolled detrusor contractions and may be beneficial in some cats; however, one study did not document a benefit. Phenoxybenzamine and prazosin are sympatholytic agents that decrease urethral tone and spasm and may help some cats. Cats with FIC have been found to have a dysregulation in their stress response with an increase in sympathetic autonomic nervous tone and a decreased hypothalamic-pituitary-adrenal response. Prazosin has more systemic effects than phenoxybenzamine and, therefore, may have benefit in cats with FIC to decrease the increased sympathetic nervous system activation. Diazepam and dantrolene are skeletal muscle relaxants that may decrease tone and spasm of the distal urethra.

Anti-Inflammatory and Analgesic Agents
Glucocorticoids have been used to decrease inflammation; however, studies have shown no benefit in cats with FIC. They are contraindicated in cats with urethral obstruction or those that have indwelling urinary catheters because they increase risk of UTI. Nonsteroidal anti-inflammatory drugs (NSAID) may decrease inflammation and pain; however, they are contraindicated with azotemia and dehydration. Buprenorphine and Torbugesic do not have anti-inflammatory properties, but do decrease pain and appear to make cats with FIC more comfortable.

Amitriptyline
Amitriptyline is a tricyclic antidepressant that may have analgesic properties, stabilize mast cells, and decrease inflammation. In one uncontrolled study, 9 of 15 cats with idiopathic lower urinary tract disease improved with amitriptyline. One controlled study of cats with active lower urinary tract disease showed no benefit and cats receiving amitriptyline had a higher incidence of recurrence of lower urinary tract signs. The goal is to find a dose that will have a calming effect.

Glycosaminoglycans (GAGs)
Cats with FIC have decreased concentrations of GAGs in their urine. GAGs may have a protectant role at the mucosal-urine interface. Two controlled studies failed to show a difference in clinical signs between a GAG and placebo in cats with idiopathic lower urinary tract disease. There is one pilot study of intravesical instillation of a GAG (A-CYST) in cats presenting with urethral obstruction that showed reduction in repeated urethral obstruction (0% recurrence in GAG treated cats versus 43% in placebo treated cats); however, it was not statistically different.

Dietary Modification
In cats with matrix-crystalline plugs or with struvite crystalluria, feeding a struvite preventative diet may have some benefit. In one study of cats with idiopathic lower urinary tract disease, cats fed a canned diet had fewer recurrences than those fed a dry diet. In a more recent randomized controlled clinical trial, cats
with FIC had an 89% reduction in recurrences when fed a diet enriched with omega-3 fatty acids and anti-oxidants also containing L-tryptophan and alpha-casozepine.

**Maropitant**
Maropitant is used as an anti-emetic because it is a neurokinin inhibitor. It has been suggested that may aid in treating cats with FIC by reducing spasticity; however, no studies have been performed to validate this hypothesis.

**Stress Reduction and \Multi-Modal Environmental Modification (MEMO)**
The role of stress in eliciting clinical signs in cats predisposed to FIC is well documented. Decreasing stress by modifying environment may be beneficial. Cats do not respond to force, are territorial, and like to be in control of their environment. Minimizing stress and conflict may help some cats with FIC. Litter boxes and food should be away from noise and distractions. Cats like to climb, hide, scratch, and hunt; therefore, vertical and horizontal space should be provided. One food dish, water bowl, and litter pan should be available for each cat in the household with one additional of each. Additional information can be found at the Indoor Cat Initiative: https://indoorpet.osu.edu <VIN editor: link updated May 1, 2015>. Additionally, the ‘urinary stress’ diet that was shown to decrease recurrences by 89% is formulated to not only be anti-inflammatory but to also have a calming effect.

**Clomipramine and Fluoxetine**
These drugs are used for urine spraying / marking behavior. They appear to modify behavior may have some analgesic effects.

**Pheromones**
Feline facial pheromones may calm a cat; however, in one study of cats with FIC, no benefit was found.

**HOW DO I TREAT CATS WITH LOWER URINARY TRACT DISEASE?**

**Young Cat, First Episode**
- Urethral obstruction
  - Unobstruct
  - Radiographs, UA (other lab work?)
  - Indwelling catheter?
  - Torbugesic?
  - Diet change (likely)?
  - Antibiotics (peri-catheterizationMEMO?)
  - If persists or recurs, diagnostics
  - Urinalysis (minimum)
  - MEMO
  - Torbugesic?
  - Diet change? Likely - usually stones or plugs)
  - If persists or recurs
    - Do additional diagnostics
    - Diet?
    - Amitriptyline?
    - Glycosaminoglycans?

**Old Cat, First Episode**
- Urethral obstruction
  - Unobstruct
  - Radiographs, UA (other lab work?)
  - Indwelling catheter?
  - Torbugesic?
  - Diet change (likely) - stones or plug?
  - Others?
- Antibiotics (peri-catheterization)
- If persists or recurs - diagnostics
- No urethral obstruction
- Diagnostics
- MEMO
- Torbugesic?
- Diet change? Likely - urolithiasis - calcium oxalate)
- If persists or recurs
  - Do additional diagnostics
  - Torbugesic as needed
  - Diet?
  - Amitriptyline?
  - Glycosaminoglycans?

REFERENCES