Urinary incontinence occurs commonly in dogs and rarely in cats. Cases of refractory urinary incontinence can be extremely frustrating to treat, and may result in euthanasia or surrender. In this talk we will discuss the pathophysiology and most common etiologies of incontinence in dogs, and contrast this with cats. We will also explore treatment options for refractory cases.

**Pathophysiology of the lower urinary tract:**

Urinary incontinence is defined as an involuntary escape or leakage of urine during the storage phase of the urinary cycle. This most commonly results in a complaint of urine dribbling or dripping along with episodes of normal voiding.

Proper maintenance of continence depends on an intricate interplay of neural signaling and muscle activity. The bladder itself is made up of smooth muscle known as the detrusor muscle. The urethra is made up of both smooth muscle which forms the internal urethral sphincter (IUS) and skeletal muscle distally which makes up the external urethral sphincter (EUS). The lower urinary tract has two distinct phases: storage and voiding.

During the storage phase, the bladder functions as a low resistance reservoir for urine. At low volume, afferent stretch receptors within the bladder fire at low frequencies. Low frequency afferent signals inhibit parasympathetic neurons and excite sympathetic neurons, primarily from the hypogastric nerve which leads to bladder relaxation by activating β-adrenergic receptors. Also importantly during the storage phase, both the internal and external urethral sphincters are contracted. For the IUS, this is again due to sympathetic innervation from the hypogastric nerve which activates α-adrenergic receptors. (Hypogastric = hold). For the EUS, contraction is due to autonomic or voluntary innervation from the pudendal nerve.

At high bladder volume, the stretch receptors start to fire at higher frequencies and this stimulates the cerebral cortex to initiate the voiding phase. The signals from the brain leads to inhibition of the sympathetic tone to the IUS and increased parasympathetic tone via the pelvic nerve which causes detrusor contraction. (Sympathetic/Hypogastric = hold, Parasympathetic/Pelvic = pee). During voiding, the brain also shuts off the pudendal nerve and relaxes the EUS. The detrusor muscle is contracted by activation of muscarinic receptors until
the bladder pressure is greater than the outflow resistance of the IUS and EUS leading to outflow of urine.

Why do we care so much about all these nerves and receptors? Well, actually a lot of the medications we use to treat urinary incontinence act on these specific receptors, such as Proin or phenylpropanolamine, which acts to increase sympathetic activity, primarily of the $\alpha$-adrenergic receptors of the IUS. Additionally, we occasionally see cases in which we may have sub-optimal response to treatment due to a lack of knowledge of how the lower urinary tract physiology works, such as when bethanechol (which stimulates the muscarinic receptors leading to detrusor contraction) and Proin (which causes increased urethral sphincter tone) are given together. It’s also extremely important to understand the anatomy and physiology of the lower urinary tract system to properly localize the underlying cause of the incontinence.

**Diagnostic approach to urinary incontinence:**

When working up a urinary incontinence case, it is crucial that we differentiate between a voiding phase disorder such as overflow incontinence versus a storage phase disorder such as urethral sphincter mechanism incompetence or USMI. The first step is differentiating “big bladders” or voiding disorders vs. “Small bladders” or storage disorders. Patients with voiding disorders have difficulty completely emptying the bladder and may have overflow incontinence. Patients with storage disorders are often incontinent or may have pollakiuria- increased small volume urination.

**A complete history and physical exam are the most important parts of the diagnostic workup!** A physical exam should include a thorough neurologic exam as abnormalities in any of the aforementioned nerves can lead to storage or voiding disorders. The bladder should be palpated carefully before and after voiding and an estimate or ultrasonographic measurement of bladder size performed. Observation of the patient while it is urinating is a critical part of the exam. Lower motor neuron lesions are associated with easy manual expression and decreased sphincter tone due to loss of sympathetic activity. Upper motor neuron bladders are difficult to express and have increased sphincter tone due to damage in the brain or brainstem or thoracolumbar spinal cord leading to loss of inhibition of the sympathetic innervation from the pudendal or hypogastric nerves, causing an inability to relax the EUS/IUS and initiate voiding. A classic example of this is the “down dachshund” with a T-L lesion who is difficult to manually express. These patients are more likely to develop overflow incontinence.

A minimum database consists of basic bloodwork and urinalysis along with a sterile urine culture, ideally obtained via cystocentesis. Diagnostic imaging includes survey radiographs, contrast cystourethrogram, intravenous pyelogram, or ultrasound of the lower urinary tract. Advanced procedures such as endoscopic evaluation of the lower urinary tract or CT scans can be performed especially in search of an ectopic ureter or other congenital abnormality.
Functional studies such as cystometrogram and urethral pressure profiles can also be performed. Many cases are simply diagnosed via empiric response to therapy.

**Urinary incontinence in dogs:**

Urethral sphincter mechanism incompetence or USMI is the most common urinary storage disorder in the dog.³ It is thought to be a decrease in urethral pressure as a result of breakdown in urethral muscle tone and neuromuscular responsiveness.² It is most common in spayed female dogs, which is believed to be due to a reduction in sex hormones, primarily estrogen, although the pathogenesis still remains uncertain. Dogs with USMI have normal bladder capacity and can urinate normally and completely empty their bladder. It is important to determine involuntary incontinence from behavioral disorders such as submissive urination or inappropriate urination. Usually these dogs exhibit nocturia- urinating at rest or while sleeping, with a history of puddles or wet spots found on bedding or when getting up from a laying position. Treatment for USMI involves medical management as a first line treatment, with the most common drugs used being phenylpropanolamine (PPA, Proin®), an alpha-agonist and estrogens such as diethylstilbestrol (DES) or Incurin®, an estriol. Surgical options are also available for patients that fail medical management. The most promising procedures involve placing an artificial external urethral sphincter and urethral bulking agents such as collagen which are placed within the urethra via cystoscope.

USMI is rare in male dogs and extremely rare in cats. Response to treatment is variable in male dogs, with one study showing a response rate of only 43% in male dogs treated with phenylpropanolamine, versus the reported 75-90% in females.⁴ A recent retrospective study has shown success with testosterone cypionate in male dogs with USMI but had a very small sample size.⁵ Estrogens are contraindicated in male dogs due to risk of prostatic metaplasia and feminization.⁶

Congenital abnormalities such as ectopic ureters (EU) are another common cause of incontinence in dogs. EU can be intravesicular or extravesicular and can be intramural or extramural. Diagnosis is via cystoscopy, CT scan, or occasionally on ultrasound via Lasix study. These patients usually present at a younger age than those with USMI and often have constant dribbling. Males are often presented at a later age or may go undiagnosed due to longer urethral length making them more likely to be continent despite abnormal ureteral placement.⁷ Treatment for ectopic ureters is typically surgical for extra- or intra-mural EUs, or via transurethral laser ablation for intramural ectopics. Incontinence following correction of EUs is still very common, and is estimated to be anywhere from 25-67%.⁷

Detrusor hyperreflexia, also known as overactive bladder or urge incontinence, is the most common form of urinary incontinence in people, but is poorly defined in companion animals.² In people, overactive bladder causes a sudden urge to urinate due to involuntary bladder contractions that often leads to incontinence. Diagnosis of OAB in animals can be challenging
and is only definitively made using urodynamic studies such as cystometrography. Usually these patients have small, thickened or painful bladders and palpation can induce urination. Urinalysis and urine culture is essential. Treatment relies on identifying and treating any underlying causes and empiric treatment with antimuscarinics such as oxybutynin.

**Urinary incontinence in cats:**

Little data is available on the prevalence of urinary incontinence in cats and the underlying etiologies that cause it. Ectopic ureters were found to be the most common cause (10/19) of incontinence in one retrospective study of 19 cats presented for incontinence. In this study, urethral sphincter mechanism incompetence was the second most common disease (9/19), but was often accompanied by other congenital abnormalities and urinary tract infection. Feline incontinence is rare, regardless of the underlying cause. This may be due to differences in the feline lower urinary tract anatomy. Compared to dogs, cats seem to have more smooth muscle in the bladder neck and proximal urethra, which may confer better IUS activity. They also have a larger area of striated muscle and fibroelastic tissue within their EUS, leading to a larger “continence zone.”

Ectopic ureters in cats are thought to be equally distributed between male and female cats. The female predisposition in canine EU may reflect the lack of diagnosis in canine male ectopic ureters, however. Most ectopic ureters in cats are extramural, requiring surgical transplantation, and may be unilateral or bilateral. Cats with EU are also thought to be at risk for concurrent USMI, similarly to dogs.

Urethral sphincter mechanism incompetence or USMI is often associated with other congenital malformations in cats such as vaginal aplasia. Cats with USMI are often presented early in life, compared to dogs in which this is considered an acquired disease. Post-spay USMI is exceedingly rare in cats. Medical management with alpha-adrenergic receptor agonists such as PPA has been attempted, but efficacy is undocumented. Estrogens should be used with caution as they may contribute to mammary neoplasia. One report describes use of a hydraulic occlude in 3 female cats refractory to medical management of USMI. Another describes the use of deslorelin acetate in a cat diagnosed with post-spay USMI.

Acquired causes include damage to the spinal cord or innervation to the lower urinary tract. Spinal cord trauma such as vehicular trauma is the most common cause, but intervertebral disk disease can also occur. Iatrogenic damage to the lower urinary tract can occur secondary to perineal urethrostomy (PU) surgery. Urinary catheter placement following urethral obstruction can lead to detrusor atony. Other less common causes of acquired incontinence in cats are dysautonomia and feline leukemia virus-associated myelopathy.
References:


