URINARY INCONTINENCE: REFRACTORY INCONTINENCE CAN BE FIXED

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Micturition is the physiologic process of voiding and storage of urine. Disorders of micturition interfere with these normal processes and can often lead to urinary incontinence, or the inability to voluntarily control the flow of urine through the urethra. We will discuss the anatomy and physiology of the urinary bladder/urethra, the micturition reflex, the various causes of urinary incontinence, the diagnostic process of isolating a cause for incontinence and finally various medical, surgical and minimally invasive interventional treatments for urinary incontinence.

CONTROL OF MICTURITION

The control of micturition is at the level of the lumbosacral spinal cord and modified by the brain and upper motor neuron spinal cord. The lumbar spinal cord segments innervate the sympathetic input to the beta-adrenergic receptors in the urinary bladder and the alpha-adrenergic receptors at the bladder neck and internal urethral sphincter. Parasympathetic neurons from the sacral spinal cord segments travel through the pelvic nerve to the cholinergic receptors in the urinary bladder. The pudendal nerve originates from the sacral spinal cord segments as well, providing somatic input to the external urethral sphincter. Sympathetic activity maintains urine storage by relaxing the bladder via beta-adrenergic receptors, and contracting the internal urethral sphincter by alpha-adrenergic receptors. Voluntary contraction of the external urethral sphincter by the pudendal nerve also aids in urine storage. Filling of the urinary bladder stimulates the stretch receptors, which send impulses through the pelvic nerve and spinal pathways to the brain stem and cerebrum. Parasympathetic impulses are transmitted from the micturition center through the spinal tracts and the pelvic nerve to simulate the cholinergic receptors and initiate voiding: detrusor contraction. During contraction of the detrusor muscle, concurrent inhibition of the sympathetic and somatic input to the urethra and bladder neck allows urethral opening and voiding can occur.

CAUSES OF URINARY INCONTINENCE

Urinary incontinence can be divided into neurogenic and non-neurogenic disorders.

**Neurogenic incontinence** can result from any brain or spinal lesions that interrupt the micturition reflex as discussed above. Incontinence is most likely to occur with a lower motor neuron disorder affecting either the sacral nerves or the pudendal nerves. This can occur as a congenital malformation of the sacrum (Manx cats), sacral trauma/fractures, or lumbosacral disease (IVDD, neoplasia, stenosis, etc). Upper motor neuron lesions more often cause urinary retention resulting in an over distended urinary bladder that is difficult to express due to the intact sphincter mechanism. If the urinary bladder becomes over distended urine may leak out the urethra as the intravesicular pressure surpasses the urethral closure pressure, causing overflow incontinence. Most animals with neurogenic incontinence present with other signs of neurologic deficits outside of the urinary tract like fecal incontinence and other lower motor neuron signs of the hindlimbs.

**Non-neurogenic incontinence** can be due to functional or anatomic disorders of the urethra or bladder. Urethral incompetence is a functional disorder associated with incontinence occurring most often in neutered female dogs, otherwise referred to as “estrogen responsive incontinence” or “sphincter mechanism incompetence (USMI).” This is the most common acquired cause of incontinence is dogs, though has been seen in young dogs that are incontinent since birth and has been reportedly associated with ureteral ectopia in a majority of cases. The other common non-neurogenic functional cause includes detrusor instability, which is the failure of the bladder to relax during the storage phase. Anatomic
disorders like ureteral ectopia, vaginal strictures, persistent paramesonephric remnant with vaginal pooling, or a patent urachus have been suggested to cause incontinence. Lower urinary tract disorders (like infections, tumors or urolithiasis) can cause incontinence secondary to an increased urge to urinate. Animals with incomplete voiding, due to partial obstructions may have periods of incontinence as well.

1) Urethral Sphincter Mechanism Incompetence (USMI)

This is mainly a condition of spayed female dogs, though can occur prior to neutering. It has been shown that decreases in maximal urethral closure pressures and functional urethral length occurs during the first 12-18 months after spaying, resulting in a deterioration of urethral closure function. This process can continue with age. Approximately 20% of female dogs will develop USMI an average of 3 years after their spay. Dogs >20kg (31%) are more likely to develop USMI than those <20kg (9.3%). For dogs spayed before their first heat the risk of incontinence drops by 50%, but it is suggested that ovariohysterectomy (OHE) or ovariectomy (OE) be performed between 3 months of age and the first heat. OHE before 3 months of age has been associated with an increased incidence of USMI.

2) Ureteral Ectopia

Ureteral ectopia (UE) is a congenital abnormality of one or both ureters in which the ureteral orifice is located distal to the bladder neck. This can be in the urethra, vagina, uterus or vestibule in a female dog, or anywhere along the urethra in a male dog. Ureteral ectopia is a result of dysembryogenesis of the urinary system. Intramural ureters enter the bladder neck in the normal position, fail to open into the bladder lumen, tunnel beyond the normal ureteral orifice position and exit distally at variable locations within the urogenital tract. Ureteral ectopia has been commonly associated with multiple anomalies of the urinary tract > 90% of cases, including absent, small or irregular kidneys, renal dysplasia, hydronephrosis, dilated ureters, ureteroceles, tortuous ureters, a pelvic bladder, abnormal uroterovesicular junction shape, and USMI. The history of urinary incontinence usually can be traced to the time of weaning and is often mistaken as a behavioral problem. Male dogs may present later in life than female dogs, likely due to their long urethra, allowing for urine storage until a concurrent issue results in polyuria (renal insufficiency), pollakiuria, or urgency (urinary tract infection).

Although UE has been reported in both pure and mixed breed dogs, it seems to occur with greater frequency in the Labrador and Golden retriever, Siberian Husky, Terrier, Newfoundland and Poodle (miniature and toy). The most common clinical finding is persistent or intermittent urinary incontinence since birth or weaning, with or without normal voiding patterns. Urinary incontinence may occur secondary to urine drainage being distal to the trigone and internal urethral sphincter mechanism, physical disruption of the sphincter musculature by a displaced intramural ureter, or associated primary sphincter mechanism incompetence. Sphincter mechanism incompetence and other voiding dysfunction have been reported in 89% of female dogs evaluated by use of urodynamic studies. Other associated urinary conditions such as urinary tract infections (64%), renal agenesis (4.8%), hydroureret (50%) or hydronephrosis (27.1%), short urethras and persistent paramesonephric remnants (89.0%), have been reported concurrently in female dogs with UE. Many of these animals are relinquished because of urinary incontinence issues or kept as outdoor pets.

DIAGNOSTIC EVALUATION

It is important to distinguish between failure of urine storage and failure to fully void. A good history, physical examination and neurologic examination is necessary to rule out neurogenic causes. If the neurologic examination is normal it is important to determine if the urinary bladder remains full after urination. If the bladder is full a mechanical (tumor, stone, stricture) or functional (reflex dyssynergia) obstruction should be considered with incontinence due to “overflow”. If the bladder is empty and dysuria is not present than USMI and UE should be considered. If there is a history of dysuria, than urge incontinence or detrusor instability should be considered.

A urinalysis should be done and collected for urine culture in all incontinent animals. Bacteriuria via cystocentesis is highly suggestive of a urinary tract infection (UTI) and this too may cause urge incontinence. A majority of dogs with UE have UTI’s, so a positive culture does not rule out UE and is often associated with other causes of incontinence and dysuria (neoplasmia, strictures, stone disease, UE, etc).

Imaging like radiography and ultrasonography are helpful tools to look for anatomic or structural defects. If UE’s are not identified on ultrasound it does not rule out this condition and further evaluation is recommended, as ultrasound is approximately 50% sensitive for UE detection. Cystourethrography and
Vaginography can be helpful in looking for vaginal stenosis or stricture, pelvic bladders, urachal diverticuli, stone disease or neoplasia. Intravenous pyelography may be helpful for detecting ureteral ectopia, in conjunction with a double contrast cystourethrogram, but this is accurate less than 50% of the time making cystoscopy or CT a more valuable diagnostic tool. If neurologic disorders are evident than spinal radiographs, CT or MRI should be considered.

**Endoscopic imaging** is our diagnostic of choice, due to the precision and sensitivity of urinary tract evaluation and the potential for diagnostic and therapeutic intervention during the same procedure (cystoscopic-guided laser ablation of the ectopic ureter (CLA), collagen injections for USMI, balloon dilation for urethral or vaginal strictures, lasering of persistent paramesonephric remnants or vaginal septums, minimally invasive stone retrieval, neoplasia diagnosis and biopsy, etc).

**Urodynamic Testing** measures urethral and bladder pressure, volume, and flow. Due to the need for specialized equipment, the availability is variable. Urodynamic testing can be helpful when the diagnosis is uncertain or the response to treatment is poor.

**TREATMENT**

The treatment for neurogenic incontinence requires treating the underlying disease process. Medical management for USMI, overflow incontinence (from bladder atony), or urge incontinence should be considered.

**Medical management**

The treatment of choice for bladder atony, in order to increase bladder contractility is Bethanecol (D: 5-25 mg PO TID; C: 1.25-7.5 mg PO TID). The treatment for urge incontinence after the underlying disease is treated is oxybutynin (D: 1.25-5 mg PO BID-TID; C: 0.5-1.25 mg PO BID-TID) or propantheline (D: 7.5-30 mg PO TID; C: 5-7.5 mg PO TID or 7.5 mg PO q 72 hr). For USMI the need to increase urethral resistance is necessary and this is best accomplished with phenylpropanolamine (PPA) for urethral smooth muscle contraction (D: 12.5-50 mg PO TID, 1-2 mg/kg PO TID), DES (D: 0.02mg/kg – max of 1mg/dog PO SID for 5 days then 0.1-1 mg PO q 7 d), or Methyltestosterone (D-males: 0.5 mg/kg/day), Testosterone cypionate (2.2 mg/kg IM q 30 days). Bone marrow suppression is the biggest concern with the use of DES in female dogs though this is rare.

For USMI alpha agonists (PPA) have improved incontinence in 75-90% of cases, and 50% of cases by sensitizing the alpha-receptors for endogenous and exogenous catecholamines. This may potentiate the effects of PPA. Dogs should have their blood pressures carefully evaluated before and after use of an alpha-adrenergic agonist and hypertension is a contraindication to its use. The use of a GnRH analogue has been reported to result in continence in some dogs that failed routine medical therapy. The GnRH analogue (Leuprolide) reduces the concentrations of FSH and LH that develop after OHE, and may play a role in USMI.

Appropriate antibiotic treatment of any concurrent urinary tract infection is necessary. In dogs with concurrent ureteral ectopia, treatment for 6 weeks is recommended with an appropriate antibiotic based on bacteriologic culture and sensitivity testing. Since the ureterovesicocuticular junction is typically abnormal in dogs with UE concurrent pyelonephritis is suspected and dogs should be treated for such.

**USMI**

**Endoscopic bulking agents for USMI**

Urethral bulking agents are used in dogs that failed medical management of USMI. Injections are performed via cystoscopic guidance in the periurethral tissue at junction of the mucosa and submucosa. Success has recently been reported to be as high as 90% with collagen alone and up to 90% with the addition of PPA. A success of 68% in 40 dogs was recently reported to last a mean of 17 months (ranging from only a few months to a few years). The authors have tried different types of bulking agents, like hydroxyapatite, and have not found a benefit when compared to bovine collagen (Contigen- Bard Urologic).

**Surgical intervention**

The problem with surgical techniques to increase urethral resistance is that dysuria may occur, and most fixations are not permanent, lasting only a few weeks to a few months. For most surgeons the preferred technique is colposuspension. This is intended to move the intrapelvic bladder neck to an intra-abdominal position so that the intra-abdominal pressure can act on the bladder and urethra and increase urethral resistance. A cure rate of 50% has been previously reported.

**The Hydraulic Occluder**
A group at the University of Florida preliminarily reported on a small number of dogs with the use of a surgically placed **hydraulic occluder** for USMI. This is currently under investigation in our practice with over 20 dogs having this performed and the details will be discussed in the presentation. We feel this is a great alternative for those that fail traditional therapies. In a recent abstract at ACVIM 2009, we reported a 77% continence rate in dogs that failed everything else.

ECTOPIC URETERS

**Cystoscopic-guided laser ablation**

Over 95% of dogs with ectopic ureters transverse intramurally and are candidates for this minimally invasive procedure. This is done with the use of fluoroscopy, cystoscopy and a diode or holmium:YAG laser. This procedure is performed on an out-patient basis at the time of cystoscopic diagnosis, allowing one anesthesia. Thus far, in our experience, continence has been maintained in over 70% of patients with long term follow-up. This technique is fast, effective and minimally invasive, avoiding laparotomy, cystotomy, urethrotomy and ureterotomy. To date this procedure has been performed in both male and female dogs with success.

**Surgical intervention**

Three surgical techniques are discussed for treatment of UE, and these include: neoureterocystostomy, ureteral reimplantation and ureternephrectomy (for unilateral disease). The outcome for post-surgical continence is variable ranging between 40-71%.

**CONCLUSION**

Urinary incontinence presents a major frustration to owners and veterinarians. With the appropriate diagnosis, and treatment regimen cure treating this dilemma is possible in a majority of cases. As more alternatives become available (bulking agents, laser ablation, hydraulic occluders, etc) we hope to have greater success in future treatment outcomes.

**REFERENCES AVAILABLE UPON REQUEST**