Learning Objectives:
1. Understand the basic neurophysiology as to how urine is voided or stored.
2. Develop a list of differential diagnoses for urinary incontinence
3. Understand how to develop a diagnostic plan likely to identify the definitive cause for the urinary incontinence
4. To determine when PSMI is likely and when to best prescribe estrogens or pheynylpropanolamine.
5. To recognize that placement of urethral bulking agents or installation of an artificial urethral sphincter can restore urinary continence in patients that have failed medical management.

Micturition is the process of urinary storage (filling) and voiding (emptying). Approximately 99% of the time, the bladder and urethra are occupied in the storage of urine whereas only 1% of the time is occupied with voiding. Incontinence is the involuntary passage of urine. The affected animal typically appears to be unaware urine leakage, at least initially. Increased grooming of the perineum suggests awareness of the problem at some point in its clinical course. Urinary incontinence is much more common in dogs than in cats. When encountered in cats, urge incontinence from cystitis must be excluded as the most likely consideration. Complex neurological input to the bladder and urethra are involved in coordinating the proper times for filling and storage of urine (not reviewed here). The normal physiology of urination involves both voluntary and involuntary (i.e. autonomic) components of the nervous system. Most forms of incontinence occur only when bladder pressure exceeds urethral resistance pressure. Incontinence may occur when bladder pressure is abnormally high (transiently or persistently), urethral pressure is low, or a combination of these factors. Exceptions to this rule occur when the incontinence is due to anatomical defects such (e.g., ectopic ureter) that bypass the normal urethral sphincter mechanism.

The causes of urinary incontinence classically are divided into neurogenic or non-neurogenic categories. Non-neurogenic causes are most common, and a thorough neurological examination is important in making this distinction. Another approach is to divide causes of incontinence into those associated with small to normal-sized bladder and those associated with an enlarged bladder. Most cases of urinary incontinence (especially non-neurogenic causes) are associated with a small to normal-sized bladder at presentation.

It is important to differentiate loss of voluntary control from behavioral changes or recent onset of polyuria and polydipsia. Development of polyuria as a result of an underlying disease such as chronic renal failure may result in the animal urinating in the house because it is not being allowed outdoors frequently enough. Some owners may misinterpret this situation as the animal losing control of urinations. It is important to question the owner about the presence of dysuria or hematuria suggestive of urinary tract infection (UTI), inflammation or partial urinary tract obstruction. It is important to identify any previous episodes of trauma or surgery adjacent to or involving the urinary tract that could have affected micturition. Previous response to (or lack of response to) antibiotics or anti-inflammatory drugs may suggest the nature of an underlying condition.

It is helpful to personally observe the animal urinating. Characterize the animal’s ability to initiate a urine stream, the diameter of the stream, any interruptions of the stream, and any apparent pain. Observe the perineal region of females for wetness or foul odor. Palpate the bladder, urethra, and the prostate gland in males. Determine the size (i.e., small, normal,
enlarged) and position of the bladder before voiding. Determine the presence or absence of pain associated with palpation of the bladder (and prostate gland in males). Determine bladder size after voiding. An enlarged bladder after attempts to void suggests urine retention. Identify any masses in the bladder or prostate gland in males that could be associated with outflow obstruction, inflammation, or altered function. Evaluate the urethra on rectal examination in both males and females. Depending on the clinical circumstances, digital vaginal examination in female dogs can be performed to evaluate for presence of a mass in the region of the vestibule or external urethra. Perform a complete neurologic examination to assess the cerebrum, brainstem, and spinal cord. Rectal examination and determination of anal tone is an important first step in ruling out neurogenic causes of urinary incontinence. Pay special attention to the local sacral reflex arc including anal tone on rectal examination. Poor anal tone suggests the possibility of a LMN bladder. Evaluate the bulbocavernosus reflex (manual compression of the bulbus glandis or clitoris causes contraction of the anal sphincter via the pudendal nerve). Evaluate the perineal reflex (pricking or stroking the skin of the perineum) causes ventroflexion of the tail and contraction of the anal sphincter). In animals with incontinence and an enlarged bladder, passage of a urethral catheter to rule out intraluminal obstruction can be helpful. Extramural masses may not be identified by this procedure. In select cases it is useful to collect and measure residual urine in the bladder after the animal has voided. Residual urine volume > 0.4 ml/kg body weight suggests partial obstruction to urine outflow or functional inability to empty the bladder.

A minimal database for animals with incontinence should include a urinalysis and urine culture. If indicated by other findings, a hemogram and serum biochemical profile may be performed to identify systemic disease processes. Urinary tract imaging is performed if the diagnosis is not apparent after initial evaluation or if empirical treatment has not been effective. Survey abdominal radiographs allow the clinician to determine the position of the bladder (i.e., intrabdominal or intrapelvic), determine the size of the bladder (i.e., small, normal-sized, enlarged), determine the shape of the bladder and if Is there a distinct vesicourethral junction or not. The presence of radiopaque calculi can be determined. Contrast radiography is helpful to rule out anatomic abnormalities or radiolucent calculi. Excretory urography can be used to evaluate for the presence of ectopic ureters (see later), ureteral dilatation, or dilatation of the renal pelvis. Cystography and urethrography can be used to evaluate for the presence of anatomic abnormalities (e.g., urachal remnant, urethral diverticulum) and masses or radiolucent calculi in the bladder or urethra. Ultrasonography can help exclude anatomical abnormalities, masses, and calculi.

Urethrocystoscopy is helpful to evaluate the vestibule, vagina, urethra, and bladder and to identify abnormal ureteral openings, masses, and other anatomic abnormalities (e.g., urachal diverticulum). Contrast-enhanced excretory urography combined with computed tomography can be considered when a definitive diagnosis has not been obtained by other imaging procedures. Urodynamic studies are not necessary for the diagnosis and management of most dogs with urinary incontinence seen in primary care practice.

**Primary sphincter mechanism incompetence (PSMI)**

PSMI is the most common cause of urinary incontinence in adult female dogs seen in primary care practice. Incontinence in spayed female dogs previously was called hormone-responsive, or estrogen-responsive, incontinence. This occurs approximately 3 years after ovariohysterectomy in approximately 20% of female dogs neutered between their first and second heat cycles; in dogs spayed before their first estrus, the incidence is reported to be 9.7% (Stocklin-Gautschi 2001;57:233-6). PSMI may occur in any breed of dog or in mixed breed dogs but some breeds are over-represented including the Doberman pinscher, Giant Schnauzer, Old English Sheepdog, Rottweiler, and Boxer. The German Shepherd and Dachshund are underrepresented in some reports of dogs with PSMI. PSMI is more common in large dogs (> 20 kg) in which the incidence of incontinence may be as high as 30%( Arnold S.
5.1% of bitches in a recent reported were found to have spay-related urinary incontinence (Veronesi MC. Acta Vet Hung 2009). Bitches weighing more than 10 kg were nearly 4 times as likely to develop post-spay PSMI than those less than 10 kg (de Bleser B. Vet J 2011). Urinary incontinence can occur before spaying in some breeds such as Greater Swiss Mountain dogs, Soft Coated Wheaten terriers, Dobermans and Giant Schnauzers. The main mechanism for the development of urinary incontinence with PSMI has traditionally been attributed to low urethral closure pressure, though some dogs have a bladder component to this form of urinary incontinence (Nickel RF Vet Rec 1999). Urethral closure pressure is decreased 12 to 18 months following spaying in normal dogs (Arnold S. Schweizer Archiv fur Tierheilkunde 1997; Salomon JF; Vet Record 2006) and it is speculated that this pressure continues to decline with age.

Urine leakage while the dog is sleeping or lying down is the most common historical finding in dogs with uncomplicated PSMI. Intra-abdominal pressure increases when dogs lie on their sides and this factor may explain why many affected dogs have incontinence while sleeping (i.e. increased intra-abdominal pressure cannot be transmitted to the bladder neck and proximal urethra if these organs lie outside the abdominal cavity in pelvic canal as is the case in many affected dogs). Another contributing factor may be increased parasympathetic (relative to sympathetic) nervous activity while sleeping. Signs of dysuria (e.g. increased frequency, straining, hematuria) typically are not present in dogs with PSMI unless complicated by UTI. UTI may develop in dogs with PSMI due to the wicking effect of a urine-soaked perineum and decreased host defenses against bacterial ascent associated with decreased urethral pressure. Abnormal perivulvar anatomy (e.g., recessed vulva) also may predispose to UTI. Incontinence may develop in dogs predisposed to PSMI or may worsen in animals with mild PSMI if polyuria and bladder distension develop as a consequence of another underlying disease process (e.g. chronic renal failure, hyperadrenocorticism, steroid administration, high salt diet). Physical examination findings usually are unremarkable in dogs with uncomplicated PSMI and routine laboratory tests such as urinalysis and urine culture are normal or negative.

Radiographically, the bladder neck is more caudally positioned (often within the pelvic canal) in some dogs with sphincter mechanism incompetence as compared to continent female dogs. This radiographic feature has been referred to as a “pelvic bladder”. Maximum urethral closing pressure (MUCP) is significantly lower and functional profile length (FPL) significantly shorter on urethral pressure profiles (UPP) in affected female dogs as compared to continent female dogs.

Phenylpropanolamine (PPA) is the initial treatment of choice to restore urinary continence in dogs with PSMI. Incontinence is controlled in 75-90% of female dogs with PSMI treated with the α-adrenergic agonist PPA at a dosage of 1.0-1.5 mg/kg PO q12h or q8h (standard preparation). PPA is also available as a sustained release product (Cystolamine®; 75 mg capsule). More than half of the dogs that failed to respond with the standard formulation of PPA became continent when treated with a sustained release formulation of this drug (Bacon NJ 2002). Once daily administration may be desirable for many owners. The recommended dosage of Cystolamine® is ½ capsule PO q24h for dogs < 18 kg, 1 capsule PO q24h for dogs 19-45 kg, and 1 ½ capsules PO q24h for dogs > 45 kg. MUCP is increased on the UPP after treatment with PPA. Virtually all affected dogs have some improvement in continence after treatment with PPA. The largest dose should be given at night to control incontinence while the dog is sleeping. In dogs with incontinence only at night, dosing only at night can be effective. PPA may become less effective with prolonged use (so-called tachyphylaxis). Occasionally, simply increasing the dosage of PPA is sufficient to regain control of continence. Potential adverse effects include restlessness and hypertension. Relative contraindications to use include known underlying cardiac disease, chronic kidney disease, or systemic hypertension. Although systemic hypertension did not develop after months of PPA exposure to young dogs in an experimental setting, we have observed client-owned dogs with PSMI on PPA that have
developed systemic hypertension. We recommend systemic blood pressure be measured before beginning PPA treatment and periodically thereafter to identify the development of systemic hypertension. Pseudoephedrine was found to be an unsatisfactory alternative treatment to PPA for PSMI in dogs due to a combination of less urodynamic effect on urethral closure, lower continence scores, and more adverse effects (Byron J. JVIM 2007).

Estrogens are an effective treatment for PSMI in many dogs and can be given much less frequently than PPA. Incontinence is controlled in 60-80% of affected dogs treated with estrogens alone for PSM. Estrogen increases the sensitivity of urethral α-adrenergic receptors to catecholamines; they also may increase the number of receptors. The MUCP increased on the UPP after treatment with estrogens for a week in one study, but has not been measured in nearly as many studies as with PPA treatment. Diethylstilbestrol (DES) is dosed at 0.1-1.0 mg (0.02 mg/kg) per dog PO for 3-5 days followed by 0.1-1.0 mg PO every 3 to 7 days. DES has become more difficult to obtain because it is no longer used in human patients but it is available from veterinary compounding pharmacies. Premarin® (conjugated estrogens - obtained from pregnant mare’s urine) is dosed at 20 μg/kg PO q3d or q4d. This drug contains sodium estrone sulfate (50-65%), and sodium equilin sulfate (20-35%); estrone is converted to estradiol. Although published information on the use of Premarin® in dogs with PSMI is lacking, we have had success with this product in our hospital. Oestriol (Incurin®, a naturally-occurring, short-acting estrogen; licensed for use in incontinent neutered female dogs in the USA by the FDA as of July 2011 and in Europe since 2000) is dosed at 2 mg per dog per day for 1 week followed by reduction to minimally effective daily dose (0.25 to 2.0 mg per dog per day) and finally alternate day dosing (dose not related to body weight). In one study, 61% of dogs achieved continence and 22% improved for an overall response rate of 83% with oestriol treatment; no hematologic abnormalities were identified. Similar beneficial outcomes were shown in the as of yet unpublished series of dogs used to gain FDA approval in the USA for estriol treatment of PSMI to control estrogen-responsive urinary incontinence in ovariohysterectomized female dogs(Freedom of Information Summary Original New Drug Application; NADA 141-435 Incurin (Estriol) Tablets Dogs Approved July 25, 2011). Potential complications of treatment with estrogens include induction of the clinical signs of estrus, perineal alopecia, and bone marrow suppression. We have not encountered bone marrow suppression in dogs receiving low dose intermittent estrogens; this is most often seen after use of long-acting injectable estrogens such as estradiol cypionate or with overdose.

Clinical experience suggests that some dogs require both PPA and estrogens for optimal control of incontinence suggesting synergism of effect, though one study indicated that adding PPA to estradiol did not result in additional increases in urethral resistance. One report suggested that abnormal bladder storage function may be part of the pathophysiological mechanism in many female dogs with refractory urinary incontinence (Nickel AJVR 1997). Some dogs that fail to respond to PPA alone respond with the addition of flavoxate or oxybutynin. The injection of botulinum toxin into the submucosa of the urinary bladder of 11 dogs with PSMI resulted in urinary continence for most dogs averaging a 5 month effect (Lew S; Acta Veterinaria Hungarica 2010). These findings suggest that bladder detrusor instability may complicate PSMI in some dogs.

The use of gonadotropin-releasing hormone (GnRH) analogues is sometimes effective as treatment of PSMI. Decreased estrogen concentrations after spaying lead to extremely increased concentrations of gonadotropins: follicle-stimulating hormone (FSH) and luteinizing hormone (LH). The provision of GnRH analogues decrease FSH and LH concentrations by down regulating gonadotropin receptors in the pituitary gland. Treatment with GnRH analogues controlled incontinence in 60% of a small group of dogs with PSMI that had failed PPA treatment or were not able to take this medication due to side-effects. The original hypothesis that increased FSH and LH concentrations in spayed dogs affects urethral resistance has not been supported by further investigations as there was no correlation between MUCP on UPP
and plasma concentrations of FSH and LH. Spayed incontinent female dogs actually had lower FSH and LH concentrations than did spayed continent female dogs. GnRH analogues (i.e., leuprolide) had no effect on MUCP or other UPP parameters regardless of clinical response observed. GnRH analogues may have a direct effect on the bladder to cause detrusor muscle relaxation. Leuprolide had no effect on UPP of spayed continent beagles before or 8 weeks after treatment. Cystometrograms of treated dogs showed increased bladder threshold volume and increased compliance (i.e. increased volume without an increase in pressure). The dosage is 5-10 mg leuprolide (Lupron®) intramuscularly. There have been no recognized adverse effects and a single injection may last 6 to 8 months. GnRH analogues may be useful when dogs do not respond to PPA or when PPA is contraindicated (systemic hypertension, underlying cardiac disease, nervousness on PPA).

Urethral bulking agents can be effective in the treatment of PSMI in humans and dogs. Successful implantation of urethral bulking agents avoids the need for daily medication. The bulking agent and implantation process are expensive and may not have long duration of effect in some dogs. Successful implantation requires special equipment and technical expertise. Medical grade Teflon® was used as the first urethral bulking agent in dogs but was soon replaced by gluteraldehyde treated bovine cross-linked collagen (Contigen®; Bard). Submucosal urethral collagen injections improve continence in most dogs that have failed PPA treatment for PSMI. The goal is to create cystoscopically-visible 360° apposition of the urethral mucosa by submucosal implantation of collagen at 3 sites: the 12 o’clock position (0°), 4 o’clock position (120°), and 8 o’clock position (240°) approximately 1 to 1.5 cm caudal to the vesicourethral junction. A 50-80% response rate with collagen alone as treatment is reported. Collagen injections often render PPA more effective than prior to collagen injections in dogs not completely continent after collagen injections. In one study, collagen injections controlled incontinence in 27/40 dogs treated for an average of 17 months (range, 1-64 months). A recent study using collagen for urethral bulking involved 21 female dogs with PSMI and 10 with ectopic ureter (Byron JK JVIM 2011). Dogs of this study had a significant increase in continence score after the procedure. Mean (SD) duration of continence in dogs without addition of medication was 16.4 (15.2) months, and 5.2 (4.3) months in dogs needing additional medical therapy. The degree of coaptation of the urethra following the bulking procedure was not related to continence scores. Since the long-term success of collagen implantation is not related to the degree of urethral coaptation, it may be related to an increased force of sphincter muscle contraction secondary to stretching of the sarcomere following the bulking agent implantation; vascular smooth muscle and striated muscle are known to exert increased contractile force when stretched (Byron JVIM 2011). Client satisfaction was excellent despite a variable duration and degree of improvement of the urinary incontinence. The duration of improvement following collagen implantation is variable and there are no identified factors that will predict the degree or duration of improvement in any individual dog. Five dogs in this study had a second series of collagen injections. Often these injections are placed into the previous injection sites to augment the degree of urethral coaptation. Loss of the beneficial effect following collagen implantation occurs in some dogs possibly due to loss of volume as absorption of the phosphate buffer occurs; occasionally there is complete loss of the implanted collagen into the urethral lumen.

There are a variety of other urethral bulking agents that have been used in human medicine but none have been reported in the veterinary literature– collagen has been the most commonly used agent in dogs. Contigen® (Bard) has been the gold standard for urethral bulking in veterinary medicine for a long time, but Bard stopped manufacturing this product in 2011. RegGain® (Avalon Medical; Stillwater, MN) was formulated for veterinary use as a direct replacement for Contigen®. ReGain® contains a higher concentration of collagen in suspension compared to the Bard product (42 mg/ml vs 35 mg/ml ); this could mean more bulking per ml of suspension delivered to the urethral submucosa. ReGain® does not require refrigeration as did Contigen® and the company claims to have improved the collagen
crosslinking technology compared to Contigen. Use of this formulation of collagen has yet to be reported in veterinary medicine. ReGain® is substantially less expensive than the human product ($250 or 2.5 ml in a prefilled syringe). Researchers at the University of Tennessee are currently in the process of evaluating polydimethylsiloxane (PDMS; Macroplastique®-Uroplasty, Inc Minnetonka, MN) in a clinical study of female dogs with PSMI. This product has used to treat European women with stress induced urinary incontinence since 1991 and was FDA approved for use in the USA in 2006. PDMS outperforms collagen urethral bulking in studies of humans with urinary incontinence. In an interim report on 22 dogs with PSMI, 17 were fully continent without other medications and 3 were improved 1 month following urethral implantation with PDMS. Three dogs experienced blepharedema and urticarial as an allergic reaction attributed to the PVP gel component of the PDMS injection— all responded to antihistamine treatment; one dog experienced transient urethral obstruction that required an indwelling urethral catheter (Bartges JW; JVIM 2011). Longer-term outcome scores for urinary incontinence will be reported at the conclusion of this 2-year study. It has yet to be determined whether PDMS will be marketed to veterinarians.

There are a variety of surgical treatments for PSMI that will not be reviewed in these notes (all designed to increase urethral closure pressure). We recommend urethral bulking treatment over standard surgeries in most dogs. The recent development of a urethral hydraulic occluder (artificial urethral sphincter - AUS) is a much less invasive surgical technique that is being used at our institution and is currently offered as the first option in most dogs with PSMI. This technique was originally described in cadaveric dogs that showed highly increased MUCP following placement of the AUS (Adin AVJR 2004). The long-term (26-30 months) efficacy of this occluder was demonstrated in 4 clinical female dogs with PSMI by the same major investigator (Rose Vet Surg 2009). Data from over 20 dogs (Adin; ACVS Forum 2011) studied by the same group suggests about a 90% success rate for major improvement in urinary continence scores lasting long time periods following placement of the AUS; 2 dogs developed urethral obstruction months after the procedure that required removal of the AUS. In our institution placement of the AUS is now the preferred treatment over urethral bulking agents in most cases.

Sphincter mechanism incompetence occurs rarely in male dogs (castrated males, later in life) and is poorly responsive to medical therapy. Long-acting testosterone injections may be tried using testosterone propionate 2.2 mg/kg IM 3 times/wk or testosterone cypionate 5.5 mg/kg IM q30d. In a study of male dogs with PSMI a poor (20%) response to testosterone was observed. PPA also may be tried but the response (40-50%) is much lower than the response of females with PSMI. Vasopexy is a surgical alternative to increase urethral tone in intact male dogs with urinary incontinence. Collagen injection performed antegrade using a cystoscope inserted into the bladder via cystotomy has been used successfully in some male dogs with incontinence. Two male dogs were recently reported to have successful outcomes following placement of an AUS (Adin; ACVS Forum 2011).

**Ectopic Ureter(s)**

Ectopic ureter is the most common anatomic abnormality causing urinary incontinence in dogs; it is very rare in cats. Patients are usually young at presentation (< 1 year of age; females tend to be presented at a younger age than males). Females are diagnosed more often than males, though ectopic ureters may be more common in males than appreciated because affected males frequently are not incontinent as a consequence of the length of urethra distal to the opening of ectopic ureter. Five of 22 female dogs with congenital ectopic ureter presented with delayed-onset urinary incontinence in one report (Thomas PC; JSAP 2010). It is likely that some female dogs presenting with delayed-onset urinary incontinence have ectopic ureters contributing to this process. Though ectopic ureter is often viewed as a simple plumbing bypass problem, it is at times more complex in that it can be associated with a short urethra,
low urethral closure pressure, and poorly described abnormalities in the formation of the urethra-vesicular junction. Abnormalities in the development of the kidney (single agenesis, renal hypoplasia) are encountered in some dogs as well as the finding of hydronephrosis and hydroureter.

Ectopic ureters are more common in certain breeds including Siberian huskies, Labrador retrievers, Golden retrievers, Soft-Coated Wheaten terriers, Newfoundlands, and Poodles. Related Entlebucher Mountain Dogs affected with ectopic ureter(s) have recently described both with and without urinary incontinence (North JVIM 2010). Bilateral involvement is detected more often than unilateral; early reports of primarily unilateral involvement likely were affected by limitations of imaging (i.e. lack of urethrocystoscopy). Most ectopic ureters in female dogs terminate in the urethra after tunneling from more proximal locations (Cannizzo K.J Am Vet Med Assoc 2003). Ectopic ureters may have their terminal opening still within the bladder, at the vesico-urethral junction, proximal to distal urethra, and the vestibule. Extramural ectopic ureters are reported rarely; extramural ectopic ureters fail to attach and open at the bladder trigone. Extramural ectopic ureters bypass the bladder and open directly into the urinary tract distal to the trigone or directly into the vagina or vestibule. Ectopic ureters uncommonly terminate in the vestibule. Ectopic ureters may terminate in the vagina or uterus, but we have not encountered this presentation in our hospital. In the Entlebucher Mountain Dog, ectopic ureter(s) within the bladder were not associated with urinary incontinence but were occasionally associated with hydronephrosis; termination points of the ectopic ureter in the urethra were associated with urinary incontinence and sometimes with hydronephrosis (North JVIM 2010). Ectopic ureters in male dogs usually are bilateral (15/16 dogs of which more than half were Labrador retrievers in a study recently completed at Ohio State University). Four of the ectopic ureters were associated with ureteroceles. Three did not have urinary incontinence. Surgical correction produced a satisfactory outcome in 11/12 dogs.

Excretory urography can establish a diagnosis of ectopic ureter but failure to identify an ectopic ureter on excretory urography does not eliminate the possibility of one being present. Both false positive and false negative results may occur. Oblique positioning of the patient, concurrent negative contrast cystography, and fluoroscopy all can aid in identification of an ectopic ureter. Observing the typical “J” configuration of the ureter as it enters the trigone does not insure that the ureter opens into the bladder at the normal location. Computed tomography in combination with excretory urography is the gold standard for diagnosis of ectopic ureter in male dogs because ectopic ureteral openings can be missed during urethroscopy due to limitations of the the procedure in males. Ectopic ureters occasionally can be identified on ultrasonography, especially if the ureter is dilated. Ultrasonography also is useful to identify hydroureter or hydronephrosis. The presence of these abnormalities in patients with urinary incontinence is compatible with a diagnosis of ectopic ureter. Hydroureter can result from obstruction of the submucosal segment of the ectopic ureter in the urethra, a developmental abnormality of the ureter, or the effects of UTI. Ultrasonography also can identify unilateral renal aplasia or hypoplasia that occasionally can be observed in association with ectopic ureter. Color flow Doppler ultrasonography can identify jets of urine entering the bladder at the trigone. The presence of such urine jets usually excludes ectopic ureter as a diagnosis, but it is not always possible to obtain the necessary images.

Urethrocystoscopy is the gold standard for the diagnosis of ectopic ureters in female dogs. Visualization of both ureteral openings in their normal position (i.e., two C-shaped openings facing each other) in the trigone conclusively rules out ectopic ureter. A definitive diagnosis of ectopic ureter is made during urethrocystoscopy by visualization of additional openings in the urethra or vestibule. The ectopic ureter is classified as proximal, mid, or distal urethra. The ectopic ureteral openings in the urethra are always located dorsally or dorsolaterally as a consequence of the abnormal embryologic development. Other abnormalities that can accompany ectopic ureter include hydronephrosis, renal hypoplasia, pyelonephritis,
Hydroureter, bladder hypoplasia (rare, associated with bilateral ectopic ureters), and urethral sphincter mechanism incompetence.

Surgical transposition of the ureter is helpful in controlling incontinence but post surgical incontinence occurs in at least 50% of affected dogs (McLaughlin R. Vet Surg 1991; Ho LK. JAAHA 2011). The owner must be warned that many affected dogs have coexisting sphincter mechanism incompetence and remain incontinent after surgical correction. It is our opinion that surgical excision of the intramural portion of the ectopic ureter in the urethra (so-called “ureteral stripping”) in association with reconstruction of the vesico-urethral junction may improve the surgical success rate to 70-80% (McLoughlin MA. Compendium 2009). However, no difference in outcome was found in a study comparing neoureterostomy with ligation of the distal ureteral segment versus resection of the distal urethra. Urinary incontinence persisted in 50% to 70% of the dogs in this study regardless of how the intraurethral remnant was handled (Mayhew PD. JAVMA 2006). Submucosal urethral collagen injections can be used with success in some dogs with ectopic ureters that continue to have urinary incontinence after conventional surgery. The use of urethral bulking treatment with collagen was reported in 5 female dogs following ectopic ureter surgery; it was also used in 5 female dogs instead of surgery for dogs with proximally located ectopic ureters. The degree of urethral coaptation following collagen implantation was less complete in dogs with previous ectopic ureter surgery likely due to the effects of previous surgery and scarring making the submucosal injections more difficult (Byron JVIM 2011).

Endoscopic laser ablation of ectopic ureters has recently been reported. The laser can be used to ablate the submucosal tunnel and create a neo-ureterostomy in a more normal trigonal position. Continence was reported for a median of 18 months in 4 of 4 male dogs following laser ablation of ectopic ureter (Berent JAVMA 2008). In 13 female dogs that were able to be evaluated following laser ablation for ectopic ureter, 4 were completely continent without drugs, 5 were completely continent with drugs, and 4 improved on PPA but were still incontinent (Smith JAVMA 2010). Laser ablation is not expected to result in complete remission of incontinence in some dogs with ectopic ureters that have an associated PSMI. For those with persisting incontinence following laser ablation or other surgical procedures, urethral bulking agents (Byron JVIM 2011) and placement of an AUS (Berent JVIM 2009) remain options for further treatment. In 8 female dogs with ectopic ureter that had persisting urinary incontinence following ectopic ureter surgery or urethral collagen implantation, the degree of urinary incontinence improved following placement of an AUS (Berent JVIM 2009).

**SELECTED READING**


Nickel RF, Vink-Noteboom M, van den Brom WE. Clinical and radiographic findings compared with urodynamic findings in neutered female dogs with refractory urinary incontinence. The Veterinary record 1999;145:11-5


Table: Causes of urinary incontinence  (From Chew DJ, DiBartola SP, Schenck PA. Canine and Feline Nephrology and Urology, Elsevier 2010)

Neurogenic
Upper motor neuron
brains/brainstem
Neoplasia
Dysautonomia
Spinal cord
Dysautonomia
Intervertebral disk protrusion
Fibrocartilaginous infarct
Neoplasia
Infectious
Trauma
Lower motor neuron
Trauma
Congenital anomaly (e.g. Manx cat)
Reflex dyssynergy (detrusor-urethral dyssynergia)

Non-neurogenic
Primary sphincter mechanism incompetence
Anatomic abnormalities
Ectopic ureter
Patent urachus
Bladder extrophy
Urethrorectal and urethrovaginal fistulas
Ureterovaginal fistula after spaying
Female pseudohermaphroditism
Urethral diverticulum
Ureterocele
Paradoxical (obstructive)
Urge incontinence associated with UTI or inflammation
Post-prostatectomy incontinence (dogs)
Post-perineal urethrostomy incontinence (cats)
FeLV or FIV-associated incontinence (cats)
Idiopathic detrusor hyperactivity (“overactive bladder”)

Figure 1. Left panel: Injection needle through cystoscope – target site is 1-2 cm from bladder neck into urethra. Middle panel: Injection needle has been inserted submucosally and a test injection of bulking agent has been given. Right panel: the 3rd of 3 submucosal collagen injections have been given. Enough bulking agent is given in an attempt to provide visual occlusion of the 3 sites (the Merces-Benz sign).

Figure 3. Wide band of tissue from the cranial aspect off the urethral orifice (vestibule) to the dorsal aspect of the vaginal vault (cingulum). This is known as the parmesonephric remnant.

Figure 4. Top most opening is that to the vaginal vault (vestibulo-vaginal junction – sometimes referred to as the cingulum. Very distal urethral termination of ectopic ureter (middle opening). Bottom opening is that of the urethra.
Figure 5. IVP with oblique positioning, demonstrating that the ureters bypass the trigone.

Figure 6. IVP with negative contrast cystogram, demonstrating what appears to be the normal entry sites for both ureters. One ectopic ureter was proven by cystoscopy and surgery.

Figure 7. Bilateral ectopic ureters terminating in the proximal urethra. Large arrow notes the bladder lumen.

Figure 8. Opening at top is that of a midurethral ectopic ureter. The bottom opening is that of the urethra leading to the bladder.
Figure 9. Long arrow: pointing to a proximal urethral ectopic ureteral opening. Note also a small opening in a trough from the other ectopic ureter which terminates distally.

Figure 10. Note the fenestrations in the tunnel (trough) of one ectopic ureter. Multiple follicles appear along the trough.

Figure 11. Initial approach to urinary incontinence suspected to be from primary sphincter incompetence (PSMI) emphasizing accuracy of history and further characterization of the abnormal urinations. Physical examination to exclude neurogenic conditions and mass lesions is also important.
Figure 12. Diagnostic options for those dogs highly suspected to have PSMI.

PSMI Diagnosis = LIKELY:
- Age, Breed, Sex = Typical
- History = Typical
- No Physical Exam Abnormality

Mandatory Minimum Urinalysis
- USG > 1.030?
- Protein
- Sediment Activity?

Minimum Plus = ?
- Add Urine Culture & Susceptibility

Urine Culture = Positive
- Rx UTI & Reculture

Urine Culture = Negative

More Extensive:
- CBC
- Serum Biochemistry r/o Metabolic
- Abdominal X-Ray; ULS
- r/o Masses, Pelvic Bladder

Treat to
- Urethral Tone
- Estrogens
- Adrenergics
Figure 13. Considerations for the approach to the patient that fails traditional medical treatment of PSMI with estrogens and adrenergics.
“Pearls” for Urinary Incontinence
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1. A leaking female Doberman almost NEVER has ectopic ureter(s).

2. A leaking female Doberman almost always has primary sphincter mechanism incontinence (PSMI).

3. The incidence of PSMI in spayed female dogs is between 5 and 30% depending on report and country.

4. Most ectopic ureter(EU) are bilateral.

5. Most EU are intramural, terminating in the urethra.

6. Following traditional EU surgery, over 50% are still incontinent.

7. Administration of urethral bulking agents often restores urinary continence for those that have failed PPA or estrogen treatments.

8. Placement of an artificial urethral sphincter (hydraulic occlude) appears to be the best single treatment for PSMI for those that have failed PPA or estrogens, or for young dogs that owners do not wish to give medications. The AUS maintains continence for long times and the pressure can be adjusted via saline injections through the subcutaneous port.

9. PPA traditionally has been reported with the greatest success rate in regaining continence during treatment of PSMI.

10. Estriol treatments have recently show success rates for treatment of PSMI comparable to PPA.

11. PPA side-effects of systemic hypertension are worrisome.

12. Blood pressure should be measured before starting adrenergics and during treatment to detect the initial presence or emergence of systemic hypertension.

13. PPA should not be used in patients with underlying renal or cardiac disease.

14. CBC with platelet count should be measured before starting estrogens for PSMI and then at 1,3, and 5 months after to ensure no toxic effects on the bone marrow (extremely unlikely with low doses). Continue to monitor twice yearly thereafter.

15. PPA has become a restricted “controlled” substance in some regions of the USA recently.