Normal micturition consists of a urine storage phase (bladder relaxed and filling with closed urethra), and urine voiding phase (bladder contracting and emptying with relaxed urethra). Appropriate storage and voiding depend on intricate and coordinated interaction of the nervous system, urinary bladder and urethra. Storage disorders manifest as urinary incontinence, voiding disorders manifest as urine retention (usually). Successful treatment depends foremost on accurate problem localization/description, and neurophysiologic understanding.

Lower urinary tract anatomy and neurophysiology of micturition
Key anatomic components of the lower urinary tract (LUT): 1) detrusor muscle: smooth muscle of bladder body and neck; 2) internal urethral sphincter (IUS): formed from the smooth muscle of the urethrovescicular junction; 3) external urethral sphincter (EUS): includes striated muscle encircling portions of the urethra distal to the IUS; and 4) ureterovesicular junction: proximal to IUS, at junction of bladder body and neck. The urethral closure mechanism = bladder neck + smooth and striated urethral musculature = the “outflow tract” or “outlet.”

Sympathetic (adrenergic) input controls the storage phase via hypogastric stimulation of detrusor beta receptors (smooth muscular relaxation and bladder filling) and alpha-1 bladder neck/urethral (IUS) receptors (outlet closure maintaining continence). During storage, pudendal (voluntary) nerve input stimulates nicotinic cholinergic EUS receptors, causing contraction of striated (EUS) muscle, and additional outlet closure when needed (e.g., when coughing/sneezing; to temporarily override urge to void when inappropriate).

Parasympatheitcs (cholinergics) rule voiding. Detrusor stretch receptors transmit filling sensation/voiding urge via pelvic nerve and pain/distension via hypogastric nerve to higher centers. The pelvic nerve stimulates contraction via detrusor muscarinics, raising bladder pressure. Simultaneously, sympathetic input to the outlet is inhibited at the level of the brainstem, allowing IUS and EUS relaxation. When bladder pressure exceeds closure pressure voiding occurs, then the system is “reset” for filling.

Urinary incontinence is involuntary urethral urine leakage, and occurs most in spayed female dogs, less in male dogs, and rarely in cats. The culprit is usually urethral sphincter(s) failure.

Causes of incontinence
Neurogenic
In neurogenic incontinence, leakage is rarely the sole apparent abnormality; usually pelvic limb deficits or a sacrococcygeal (“tail-pull”) presentation is present. UMN lesions generally produce sphincter hypertonus and retention; LMN lesions cause sphincter hypotonus and incontinence, as can brain diseases and some generalized neurologic disease (e.g., dysautonomia). Treatment and prognosis for control of leakage depend primarily on resolution of underlying cause (e.g., surgical management of intervertebral disk herniation; fixation of sacral fracture).

Non-neurogenic
Urethral sphincter mechanism incompetence (USMI)
USMI is the most common cause of canine incontinence. It’s seen most often in spayed, medium to large-breed adult bitches, but can occur congenitally, and the development and degree of USMI in a given patient may be multifactorial. Castrated and intact male dogs can (uncommonly) develop USMI, as can cats (very rarely). A recent report suggests OHE prior to 6-7 months as a predisposing factor in bitches weighing >20 kg at maturity. In otherwise healthy, neurologically normal dogs with concentrated urine and without LUT inflammation, diagnosis of USMI is confirmed by positive response to drugs that increase outlet resistance. USMI causes intermittent incontinence, seen most often during recumbency, and particularly during sleep.

Alpha-1 agonists (e.g., phenylpropanolamine[PBA]) and estrogens (e.g., diethylstilbestrol, estriol) effectively treat USMI. Most (80-95%) USMI bitches respond very well to one or a combination of these. If inadequate response to a single agent, combination therapy may be more effective. PPA is first-line therapy for USMI cats and male dogs. Male dogs can also be treated with injectable testosterone, alone or with PPA. Unfortunately, only ~45% of male dogs with USMI are medically well-controlled.

Depot gonadotropin releasing hormone (GnRH) analogs may help USMI refractory to PPA and estrogens. GnRH analogs down-regulate production/secretion of FSH and LH, and multifactorially improve continence; bladder capacity is improved but urethral pressures don’t increase. In a recent report GnRH analogs +/- PPA controlled incontinence within 5-10 days in 12/13 dogs with either USMI refractory to PPA and estrogens, or an inability to take PPA.
Endoscopic submucosal bulking injections (crosslinked collagen or extracellular matrix) into the proximal urethra can help refractory USMI. These injections narrow the urethral lumen and improve outflow resistance. Effects may last up to several years, but then may need to be repeated, and the mucosa may not tolerate more than one or two repeated injections.

Surgical procedures including colposuspension, cystourethropexy, and seromuscular urethral slinging have also been used for refractory USMI. Singly, durability of effect has been limited, but a recent report on colposuspension/urethropexy combination surgery shows significantly increased length of efficacy. Artificial hydraulic sphincters have also been used, and transobturator vaginal tape procedures may (with practice) prove a less invasive, economical, effective procedure for USMI bitches.

**Detrusor instability/overactive bladder/urge incontinence**

Detrusor instability is failure of bladder relaxation during storage, and causes leakage due to involuntary, uninhibited detrusor contraction. Less common than USMI, it’s characterized by intermittent incontinence, often with activity or excitement, and may resemble pollakiuria or inappropriate urination since dogs will often posture when leaking. Detrusor instability may occur alone or in combination with USMI in male or female dogs, and is treated with antimuscarinic (anticholinergic) agents (e.g., imipramine, oxybutynin, dicyclomine) that increase bladder capacity and decrease spasticity.

**Ectopic ureter**

Ureters implanting anywhere but the trigone are termed ectopic, and ureteral termination distal to the trigone conducts urine to the proximal urethra or vagina and usually causes continuous dribbling from birth. Cystoscopic laser ablation (ideal), surgical reimplantation into bladder, or nephrectomy if kidney is severely hydronephrotic is required for correction. Ectopia may be unilateral or bilateral, and other urinary abnormalities are often also present (e.g., short, wide urethra; USMI). The presence of multiple anomalies may necessitate medications to achieve continence even after correction of ectopia; thorough pre-op urinary tract assessment refines prognosis and helps predict need for ongoing medical management.

**Prostatic disease**

Incontinent males should be screened for bacterial prostatitis, prostatic neoplasia, and other prostatic disease (rectal palpation, urine/prostatic fluid analysis, ultrasonography) since these diseases can engender urine leakage.

**Cats**

Cats are rarely incontinent, but common causes for feline incontinence include congenital anomalies, neurologic injury/malformation, viral disease, and USMI. Juveniles should be screened for ectopic ureters and vaginoureteral anomalies, and tested for FeLV, once LUT inflammation and polyuria are excluded. PPA may be effective in non-neurogenic adult USMI. Urinary/fecal incontinence occur with tail-pull injury and in Manx with sacral malformation.

**Diagnostic approach to incontinence**

History and physical (including observed voiding) inform lesion localization; signalment alone will narrow differential list. Minimum data base in incontinence includes CBC/chemistry (rule out polyuric diseases) urinalysis and culture. Polyuria may precipitate or exacerbate incontinence by overwhelming bladder capacity, so poorly concentrated urine should be confirmed and investigated, if persistent. Most incontinent dogs don’t need imaging at initial workup, but is recommended for incontinent pets: <1 year old; with onset following a surgical procedure; with continuous leakage; who are male; with leakage from anatomically abnormal sites; with recurrent UTI, recurrent vaginitis, hematuria, crystalluria, or azotemia; and when considering surgical correction.

Survey radiography detect radio-opaque uroliths and gross bladder, vertebral, or pelvic malformations. Cystoscopy is the gold standard for detecting abnormalities of the ureters and urethra; excretory urography (with radiographs or CT) is helpful when cystoscopy is not available. Ultrasound helps assess UUT/LUT anatomy, and detects prostatic or trigonal masses interfering with normal outlet closure. Digital vaginal exam, vaginoscopy and/or contrast vaginogram help evaluate presence, position and severity of vestibulovaginal stenosis, or the possible presence of vaginal urine pooling.

**Relapsing or refractory incontinence**

When relapse or failure of presumed USMI response occurs, re-assessing dose, selection and compliance with medications, re-screening for infection or polyuric disease, ruling out an underlying anatomic anomaly/lesion or mixed disorder, behavioral component, or senility may be helpful. If these possibilities are ruled out, surgical interventions may be appropriate.
Postrenal azotemia occurs when any process distal to the renal tubules interferes with urine collection, containment, or excretion, and the resulting retention of wastes can rapidly cause life-threatening fluid, electrolyte, and acid–base derangement. Acute postrenal azotemia (usually urethral obstruction, ureteral obstruction, or traumatic urinary tract rupture) is common, particularly in cats, and may be fatal without rapid correction. Because postrenal azotemia results from urinary tract rupture or obstruction, rather than intrinsic damage to the kidneys, it has the inherent potential for reversibility; thus, the possibility of a primary postrenal disease or component should be initially investigated in every azotemic patient.

**Lower urinary tract obstruction (LUTO)**
LUTO is usually diagnosed by history and by palpation of a turgid, painful bladder. Owners may report unproductive attempts to urinate, which are sometimes mistaken for constipation. Urolithiasis and neoplasia are the most common causes of canine LUTO. Urethral stones obstruct more male than female dogs due to the smaller relative urethral diameter and longer length in males; the urethral curving around the ischium; and, most restrictive, the presence of the os penis. Calcium oxalate and ammonium urate stones are the stone types that most frequently cause urethral obstruction due to their small size, tendency to occur multiply, and increased incidence of both types in males.

Urolithiasis or mucocysticuleal urethral plugs cause most feline LUTO. Mucocysticuleal plugs are generally associated with feline idiopathic cystitis (FIC) and are seen in both males and females, but they cause obstructive disease almost exclusively in males because of comparative urethral anatomy. Survival rate is 90-95% with treatment, and recurrence rate of FIC-associated obstruction is 15-40%. Predisposed cats tend to be one or more of the following: young adults, overweight, dry food eaters, and indoor-only. The following factors seem to decrease risk of re-obstruction: use of a 3.5 Fr (rather than 5 Fr) indwelling urethral catheter following relief of obstruction, longer duration of catheterization, increasing water intake, environmental modification to reduce stressors, dietary management, indoor-outdoor (versus all-indoor) lifestyle, and urethral relaxation (e.g. with prazosin). The following do not change the risk of re-obstruction: empirical antibiotics, glucocorticoids, anxiolytics, and NSAIDs. Older cats are more likely to re-obstruct than are younger cats.

**Upper urinary tract obstruction (UUTO)**
Acute UUTO may occur due to intraluminal, extraluminal, and intramural causes, such as uroliths, nonmineralized material, trauma, neoplasia, proliferative disease, ureteroceles, inflammation, fibrosis, stricture, and inadvertent surgical trauma or ligation. For UUTO to cause azotemia, bilateral disease—either bilateral ureteral obstruction, or unilateral ureteral obstruction with dysfunction or absence of the contralateral kidney—must be present.

The most common cause of UUTO is calcium oxalate urolithiasis (CaOx), usually in cats and small dogs. Incidence of CaOx has increased dramatically in the past 20 years (now >90% of analyzed nephroliths and ureteroliths). CaOx stones form in renal parenchyma, and may stay there or pass into the ureters and bladder. Formation risk is multifactorial, including degree of urine saturation with calculogenic minerals, urinary crystallization/ aggregation/growth inhibitors, and urinary crystal aggregation/ growth promoters. UUTO can be simultaneous, bilateral obstruction; however, acute unilateral obstruction with prior contralateral obstruction (causing dysfunction) is much more common. These pets frequently have one firm, atrophied, nonpainful smaller kidney, and one larger, painful, turgid-feeling kidney. Some astute owners may detect subtle clinical signs during an initial unilateral obstruction (e.g., antisocial behavior, flank licking, or back or abdominal pain), leading to earlier detection of the presence of kidney/ureteral stones.

Trauma, renal biopsy, or renal hematuria can cause UUTO with clotted blood. Accumulated debris (e.g., from inflammation or ureteral trauma, fungal granuloma, sloughed renal papilla) may also result in a nonmineralized UUT obstruction. In our experience, such obstruction may occur due to acute renal tubular necrosis from toxin, acute pyelonephritis, or luminal trauma from presence or passage of stones. Experimentally, these obstructions often occur proximally in the UUT, may be acutely bilateral, and can be milked to a dilated region the ureter or the ureteropelvic junction for removal. Ureteral damage (inflammation, fibrosis, mineralization, stricture) can cause complete or partial UUTO. Iatrogenic ureteral ligation, transection, crushing, or devascularization occurs most often during uterine body ligation during OVH, particularly when the bladder is distended and ureters are slackened. Postoperative progressive ascites, azotemia, and/or ureteral/renal pelvic dilatation mandate prompt, aggressive assessment (ascites cytology/chemistry, excretory urography/antegrade pyelography, retrograde cystography, exploratory laparotomy).

Treatment for acute UUTO depends on etiology. If metabolic derangement is not life-threatening, medical management with fluids, analgescics, and ureteral relaxant(s) may permit passage of an intraluminal obstruction in 1-3 days. Severe derangements require
medical stabilization and/or hemodialysis, and/or surgical relief of obstruction (ureteral stent, ureterotomy/re-implantation, subcutaneous ureteral bypass).

**Urinary tract rupture**
Rupture of UUT or LUT results in urine collection in the retroperitoneum, peritoneum, or subcutaneous tissues. Presenting signs may be associated with trauma, uremia, chemical irritation (cellulitis, urine peritonitis), or, if urine is infected, septic issues. Causes of ureteral rupture include surgical damage, calculi, neoplasia, infection, and blunt or penetrating trauma (uncommon). Bladder or urethral rupture occurs most often secondary to caudal abdominal trauma (dogs), urethral catheterization (cats), or excessive manual pressure during bladder expression. Preexisting LUT pathology (e.g., urethral or bladder tumors, bladder wall damage from prolonged obstruction) can cause or predispose to rupture from instrumentation or compression.

**Common laboratory abnormalities**

**Azotemia**
LUTO pets may or may not be azotemic, but UUTO cats are some of the most azotemic pets we see. Some will have uremic oral ulcers. Very high BUN often correlates with marked post-obstructive diuresis.

**Metabolic acidosis**
IV crystalloids and relieving obstruction usually corrects acidosis (which can be severe); if not, adding sodium bicarbonate is appropriate.

**Hyperphosphatemia**
Hyperphosphatemia can drive hypocalcemia, but is corrected by relief of obstruction and fluid therapy. Phosphate binders are not usually used/needed in acute settings.

**Hyperkalemia**
Hyperkalemia can be marked (>10 mmol/L), can stop normal cardiac function, and is usually the direct cause of death when obstructed animals are left untreated. IV crystalloids and relieving obstruction usually suffice to correct potassium, but if cardiotoxicity is present, IV calcium, IV sodium bicarbonate, and/or insulin/dextrose may be needed to buy time to relieve obstruction.

**Hypocalcemia**
May be present and may be moderate to severe, but not treated unless the pet is clinical.
Anatomy and physiology
The prostate, a glandular organ surrounding the proximal urethra, is the only canine accessory sex gland, and results in disease in ~75% of intact male dogs over their lifetimes. Secretory epithelial and stromal tissue are admixed within a capsule containing alpha-1 adrenergically-innervated smooth muscle. Growth and function are androgen-mediated (chiefly dihydrotestosterone); castration induces quick prostatic atrophy (~50% at 3 weeks, and ~70% within 9 weeks).

Diagnostic testing
Physical examination
The prostate is usually palpable per rectum at the pubic brim. Manually tipping the bladder caudally and/or elevating the dog’s front end can move the prostate caudally into reach. The normal prostate is bilobed, soft-firm, smooth, symmetrical, mobile, and nonpainful. Prostates of intact Scotties may be up to 4 times larger than prostates of equally-sized other breeds’.

US and urethrography
The prostate surrounds the urethra distal to the trigone and should be smoothly rounded. The lobes are easily distinguished, with diffuse fine mottling or whorls, but without cysts. Contrast urethrography may show reflux into the prostatic ducts, and urethral narrowing through the prostatic region.

Cytology
Prostatic cytology may be performed on prostatic fluid, massage samples, and aspirates. Most commonly, prostatic fluid is collected from intact dogs by manual ejaculation. Most dogs will ejaculate with manual stimulation, but ill or painful dogs may resist stimulation. Prostatic massage is preferred for fluid collection when cancer is suspected (usually more cellular), or when illness, pain, or disposition complicates ejaculation. Prostatic massage is done post-voiding, in lateral recumbency. The urethra is catheterized and the bladder is emptied, flushed with sterile saline, and again emptied. The catheter tip is withdrawn to just distal to prostate. Massage is performed per rectum, then 5-10 ml of saline flushed through the catheter. The catheter tip is advanced collect fluid and cells from bladder. Submit ejaculate and massage samples for cytology and culture.

FNA also yields culture/cytology samples. Take care if cystic disease, abscessation, or neoplasia is suspected. Inadvertent abscess rupture can be a surgical emergency, and slight risk of needle tract seeding accompanies certain neoplasia (e.g., TCC).

Histopathology
Traumatic catheterization (TC), endoscopy, US-guided core biopsy, or surgery can all provide biopsy samples. TC resembles prostatic massage sampling, except negative pressure is applied to the catheter, and the catheter is moved back and forth in the urethra to shear and collect tissue drawn in the side-holes. Endoscopic samples are often quite small due to size-limited urethroscopy in males (often TC samples are larger!), but US-guided cores provide excellent samples (don’t biopsy urethra!).

Prostatic diseases
Benign prostatic hyperplasia/hypertrophy
BPH comprises both hypertrophy and hyperplasia of stromal and secretory tissues, is a spontaneous, normal, aging change, occurs only in intact males (unless other androgen source is present), and is not pre-neoplastic. Many BPH dogs show no signs, but it may be found due to bloody discharge (from increased parenchymal vascularity and cystic change). In men BPH causes dysuria; this is rare in dogs due to minimal prostatic smooth muscle, and greater gland mobility in the dog. Dogs thus are more often presented for tenesmus or ribbon-stools, because prostatic enlargement impedes normal defecation before functionally or mechanically interfering with normal urination. Diagnosis is of exclusion: an intact dog with an enlarged but otherwise normal prostate, negative urine/prostatic-fluid/aspirate culture, and normal cytology. Castrates don’t get BPH without a non-gonadal androgen source. Secretory markers (e.g., PSA, prostate-specific esterase) aren’t useful in dogs.

Castration is preventative, curative, and the quickest, safest, most effective treatment. 5-alpha-reductase inhibitors, progestogens, and estrogens have also been used for management. Estrogens predispose to squamous metaplasia and prostatitis. Progestagens may resolve BPH without affecting semen quality, but increase appetite and predispose to diabetes mellitus and hypothyroidism. Finasteride (5-alpha-reductase inhibitor), decreases gland size without reducing spermatogenesis or libido (inhibits conversion of testosterone to DHT but doesn’t affect testosterone production). Dogs respond in 2-4 weeks with few side effects. Clinical signs recur off medication, so therapy is considered temporary treatment for owners who wish to breed, cryopreserve semen, or achieve show titles prior to castration. Saw palmetto doesn’t treat BPH.

Bacterial prostatic diseases
Prostatitis is diffuse bacterial infection/inflammation, and a prostatic abscess is a cystic collection of suppurative fluid in the parenchyma. Prostatitis can be acute or chronic. Acute prostatitis usually causes pain and/or fever, lethargy, and hyporexia, and may
cause dyschezia, abdominal pain, or pelvic gait abnormalities. Dysuria, pyuria, hematuria, and/or urethral discharge may be present. Chronic prostatitis may be silent (just pyuria and bacteriuria). Prostatic abscesses may present like acute prostatitis, or rupture, causing sepsis/acute abdomen. Prostatitis is assumed in any intact male dog with UTI.

Septic inflammation in prostatic fluid, massage sample, prostatic aspirate, or biopsy sample is diagnostic. With ejaculate or massage sample, >10,000 cfu/ml significant; any aspirate or biopsy sample growth is significant. E. coli, Staph, Klebsiella, and Proteus are most common. US usually shows parenchymal mottling. Abscesses appear as cystic structures, with anechoic, echogenic, or mixed contents.

Culture/sensitivity on aspirate, massage, or urine directs antibiotic choice. Normal prostate/blood barrier prevents diffusion of many medications, but acute inflammation disrupts the barrier so most antibiotics initially penetrate. In general, though, best antibiotics for prostatitis are trimethoprim-sulfas, fluoroquinolones (especially enrofloxacin), and chloramphenicol. Initially 4-6 weeks of therapy is recommended, with culture 1 week and 1 month afterward. Removing predisposing factors (e.g., castration for BPH) greatly increases odds of cure. Re-culture should be performed at 3-6 month intervals. Abscesses require drainage and appropriate antibiotics. Traditionally, open surgery and marsupialization/omentialization were used, but recent reports of US-guided drainage/culture with reassessment q 1-6 weeks and re-drainage as needed is promising. In two small studies, median number of drainage treatments needed was 2, with abscess resolution in all dogs.

Castration speeds resolution of both prostatitis and prostatic abscess, and also helps prevent recurrence. Antibiotics for several weeks before castration facilitates drug delivery to deep prostatic tissues prior to inducing atrophy and involution.

**Prostatic cancers**
The only primary prostatic disease of castrates is cancer. Prostatic cancer occurs in < 0.6% of dogs, but 2-4x more in castrates. Beagles, Bouviers, Dobermans, English springers, German shorthair pointers, Scotties, Westies, and Shelties are predisposed (last three are also predisposed to TCC). Though castration has been shown to increase risk of prostatic ACA, the over-all risk in castrated males is still very low, and the benefits of castration likely outweigh this low risk in most cases. This assessment should be made for each pet individually.

Prostatic cancer is usually adenocarcinoma (ACA), with TCC next most common. Prostatic carcinomas are not androgen-dependent in dogs, nor mediated by hormonal changes once extant. In general, any castrated dog with prostatomegaly has cancer until proven otherwise. Clinical signs may be due to the primary tumor (dysuria/stranguria, urine retention, tenesmus, ribbon stools, altered gait) or to metastasis (liver, lung, pelvic/sublumbar nodes, spine/pelvis, colon, rectum, spleen). Fever, lethargy, weight loss, and hyporexia occur, especially with metastasis.

Neutrophilia, ALP elevation, hematuria and pyuria are common. Radiographic may include prostatomegaly and a periosteal reaction along pelvic bones, sometimes femurs. US may show hyperechoic parenchymal stippling (focal mineralizations), and blotchy, whorled parenchymal motting. TC, US-guided FNA, or core biopsy are reliable, non-invasive diagnostics.

Prostatectomy and chemotherapy for ACA can be helpful, but no therapy is curative, and most prostatic cancer has metastasized at diagnosis. Palliative therapy with piroxicam or carprofen can increase comfort and decrease inflammation, may have tumor anti-angiogenic effect, and can prolong survival. Chemotherapy is also used, but overall prognosis is poor. Urethral stenting, laser debulking, and percutaneous cystostomy tubes/buttons are options for dedicated owners.

**Paraprostatic cysts**
In intact males, remnants of the embryonic Wolffian ducts can fill with fluid, forming paraprostatic cysts, easily diagnosed with US. Clinical signs result from space-occupying intrapelvic cysts, with possible stranguria, bloody urethral discharge, and tenesmus, but many dogs show lethargy or decreased appetite, presumably from discomfort. Prostatic parenchyma is usually normal or shows BPH. Therapy is surgical resection; castration prevents further cyst formation.