# Medical and Interventional Management of Upper Urinary Tract Uroliths

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**KEYWORDS**

- Endoscopic nephrolithotomy • Endourology
- Extracorporeal shockwave lithotripsy (ESWL) • Nephrolithiasis • Stone dissolution
- Subcutaneous ureteral bypass device (SUB) • Ureteral obstruction
- Ureteral stenting

**KEY POINTS**

1. Nephroliths are often clinically silent. When non-obstructive and of an amenable stone type, dissolution should be attempted.
2. When problematic, nephrolithotomy can be considered. Depending on stone type, size, and species, extracorporeal shockwave lithotripsy or endoscopic nephrolithotomy are preferred techniques.
3. Obstructive ureterolithiasis should be addressed immediately to preserve kidney function. Because of decreased morbidity and mortality and versatility for all causes, interventional techniques for kidney decompression are preferred by the authors.
4. Proper training and expertise in these interventional techniques should be acquired before performing them on clinical patients for the best possible outcomes.

## INTRODUCTION

Upper urinary tract uroliths are a common problem in our small animal patients and are being more commonly recognized with the increased use of diagnostic imaging as part of a minimum database. Appropriate medical, surgical, and/or interventional management is necessary for the best outcomes and preservation of kidney function. In many instances, canine and feline nephroliths are clinically silent for many years, whereas ureteroliths are typically found as a more urgent clinical dilemma. However, when nephroliths are problematic, or become obstructive ureteroliths, intervention is

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indicated. The content of this article is based on best evidence, when available, although much is the anecdotal experience of the authors.

## NEPHROLITHIASIS

Most nephroliths found in canine and feline patients are clinically silent for many years and require diligent monitoring to ensure they do not mobilize to become a ureteral outflow tract obstruction. Dissolution of upper urinary tract stones should always be attempted when non-obstructive and stone type is amenable (eg, struvite, +/− urate and cysteine). The best practice to accomplish stone dissolution is described in the sections below.

## MEDICAL MANAGEMENT

Dissolution of upper urinary tract stones should always be attempted when non-obstructive and stone type is amenable, and given the likelihood of stone recurrence in many cases, preventative measures should be instituted whenever possible. Stone type can often be carefully predicted when considering signalment, radiographic appearance, urine microbiologic culture, fasted and fresh urine pH, fresh urine crystals, or, when available qualitative and quantitative crystallographic analysis. We use these predictions to help make decisions on the best treatment options for the patient (**Table 1**).

### *Struvite Dissolution and Prevention*

Canine upper tract uroliths have been reported to be of struvite content in 50% to 60% of cases.[1–3] Feline upper tract stones, on the other hand, are rarely composed of struvite, with 92% being calcium oxalate and 8% being dried solidified blood stones.[3,4] Struvite uroliths are composed of magnesium ammonium phosphate hexahydrate, and are more likely to form when urine is oversaturated with these minerals. In canine patients, struvite uroliths typically occur secondary to a urinary tract infection that is associated with urease-producing bacteria, such as *Staphylococcus* spp, *Proteus* spp, or *Klebsiella* spp. Bacterial urease converts urea to ammonia thus making the urine alkaline. This increase in urine pH results in precipitation of calcium and magnesium phosphates, and subsequent aggregation and stone formation. Female dogs are more likely than male dogs to be affected with struvite stones owing to their increased risk for ascending urinary tract infections. [1,3] This is likely secondary to their shorter and wider urethra, and the close association of the vulva to the anus. Struvite uroliths classically have a radiopaque appearance, are a rounded shape with some flat surfaces, and can be excessively large at times (**Fig. 1**). In the kidney, these stones are often a staghorn appearance, forming the shape of the renal pelvis and protruding into the calices and down the proximal ureter (see **Fig. 1**).

Sterile struvite stones have been reported, but are rare in dogs.[5] In cats, however, sterile struvite stones are more typical, so dissolution does not typically require antibiotics.[6] However, struvite stones in cats are typically only found in the lower urinary tract. If stones are visualized in the kidneys or ureters on radiograph in a cat than they are nearly always calcium oxalate in composition.[7]

Struvite stones can typically be dissolved in essentially all cases, regardless of their size, number, or location, as long as they are non-obstructive, surrounded by urine, and composed of purely magnesium ammonium and phosphate, rather than mixed with calcium apatite/phosphate. In the event a struvite stone is causing a ureteral obstruction than a stent would be ideal to decompress the collection system and allow urine to drain around the stone while dissolution occurs. Dissolution of ureteroliths

**Table 1**
**Upper tract stone type predictions**

| | Signalment | Sex and Species | pH (Fasting and Fresh) | Crystals | Radiographic Appearance | Presence of Infection |
|---|---|---|---|---|---|---|
| Struvite | Any breed | Female canines | >7.0 | Variable/ unpredictable | Staghorn/soft/rounded | Present: urease-producing bacteria |
| Urate | Dalmatians, Bulldogs, PSS dogs, or cat | Canine > feline, male or female | <7.0 | Usually present | Minimally opaque, staghorn | Absent |
| Cystine | English Bulldogs, Newfoundlands, Mastiffs, Labrador Retrievers, male intact dogs | Canine, male and female | Variable | Usually present | Large, minimally opaque | Absent |
| Calcium oxalate | Terriers, Schnauzers, Shih Tzu, Poodles, other metabolic derangement for hypercalciuria | Canines and feline, male and female | <7.0 | Variable/ unpredictable | Very opaque, sharp and irregular, small or large, mix mineralization | Absent |

*Abbreviation:* PSS, **portosystemic shunt**.

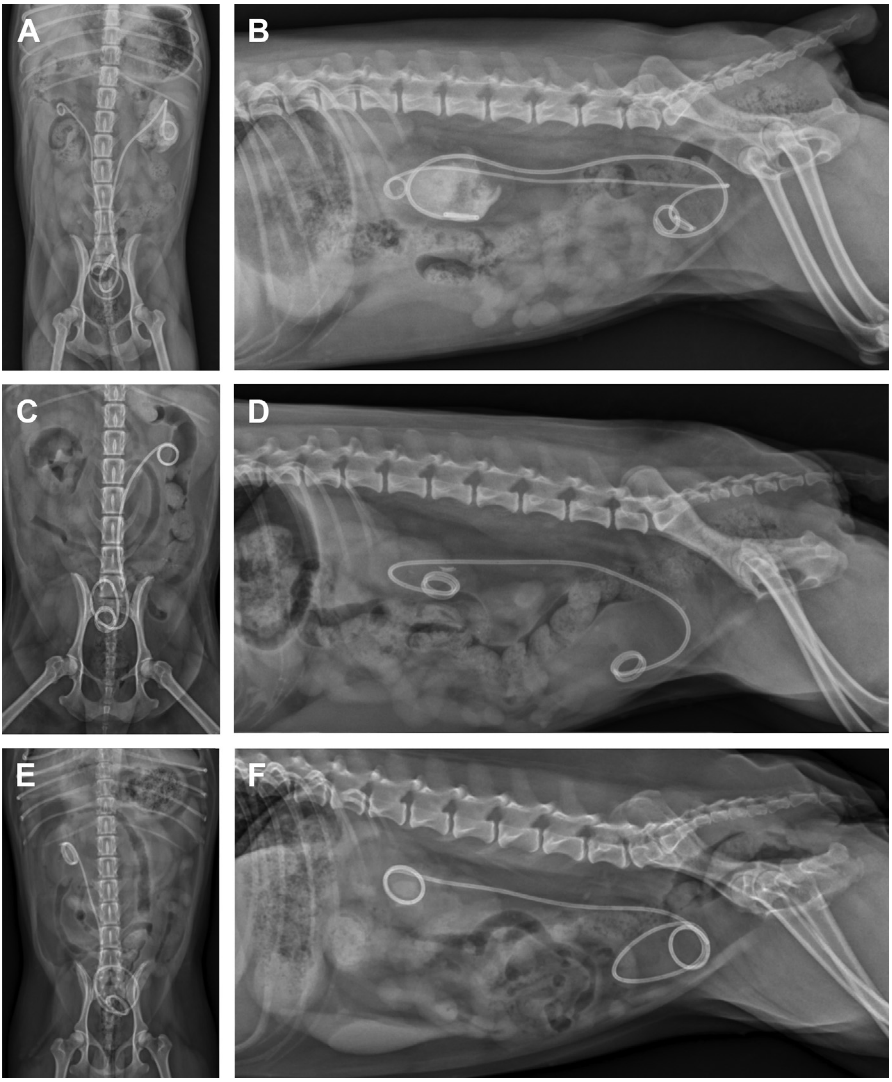


**Fig. 1.** Radiographs of dogs with renoliths of varying stone types. (*A*) Mixed calcium stone. Note the staghorn appearance in the ventrodorsal view. (*B*) Struvite renoliths. (*C*, *D*) Cystine renoliths. Note the oval shape. (*E*, *F*) Calcium oxalate renoliths. Note the sharp and irregular outline, and relative radiopacity.

often takes 2 to 6 weeks. Appropriate dissolution management involves instituting appropriate antibiotic therapy beyond the entire course of stone resolution, and promoting an acidic urine pH either with medication (eg, DL-methionine: 75–100 mg/kg po q12) or a neutral urine pH that is controlled through a diet where the key minerals of the stone are limited (eg, Hill's C/D or Royal Canine Urinary SO).[1,3,8] Care should be taken to avoid acidification if stone type is unknown and the clinician is performing a dissolution trial without clear evidence of struvite composition. In that case neutral urine pH with a dissolution diet, while controlling the urinary tract infection, is the safest approach. Inappropriate acidification could result in progression of calcium oxalate stone size and number. Increased water intake should be encouraged to achieve a urine-specific gravity of less than 1.020. Owners should add 1 to 2 additional walks per day to encourage bladder and crystal elimination.

Although stone dissolution is being implemented, repeat urinalysis (with a fasted urine pH), urine culture, and radiographs are serially recommended every 4 weeks. This is to monitor dissolution progress. In the authors' experience, within 2 to 6 weeks ureteroliths should be nearly dissolved, and within 3 months nephroliths should have decreased in size by greater than 25%. Time to full dissolution can take 6 to 9 months for large nephroliths, and the authors recommend that antibiotic therapy should be continued at the full dose for 4 weeks beyond when the stones are radiographically absent. Bacteria can be gradually released from the stone matrix as dissolution occurs, and maintaining antibiotic treatment is mandatory to ensure that additional struvite deposition does not occur.[9] Following complete dissolution, routine serial urine testing is recommended to monitor and treat infections as early as possible. They are often recurrent infections, but prompt treatment will prevent future stone formation. If, despite appropriate treatment and owner compliance, the stone is not dissolving, than stone removal should be considered. Consideration should be given to addressing any anatomic or functional risk factors for recurrent urinary tract infections (eg, recessed vulva, persistent paramesonephric remnant, urinary incontinence, urachal diverticulum).

### *Urate Dissolution and Prevention*

Urate stones include stones composed of uric acid, ammonium urate, ammonium biurate, and other salts of uric acid. Factors that promote urate urolith formation include hyperuricosuria, concentrated urine, and an acidic pH. In normal cats and dogs, uric acid is formed as an intermediate product of purine metabolism in the liver. Purines are converted to hypoxanthine, then to xanthine, and then to uric acid by xanthine oxidase. Uric acid is then converted to allantoin by uricase. Allantoin is highly water soluble and is excreted in urine. Thus, dogs with portovascular anomalies, or defects in uric acid transport, are predisposed to urate stone formation. Mutations in the LC2A9 gene that encodes for urate transport in the liver and kidney has been identified Dalmatians, English Bulldogs, and Black Russian Terriers.[10–12] This genetic disorder follows an autosomal recessive pattern of inheritance. The mutation results in decreased conversion of uric acid to allantoin and hyperuricasemia. There is no re-absorption of uric acid following glomerular filtration, and thus hyperuricuria, which results in stone formation.

In cats with urate stones, the pathogenesis is less clear. One theory is that, being strict carnivores, cats have limited ability to upregulate and downregulate the production of catabolic enzymes of amino acid metabolism. Thus, increased dietary protein results in increased purine formation and oversaturation of urea in the urine. Portovascular anomalies in cats, as well as dogs, can also result in stone formation.[13] Urate stones are typically radiolucent but can be visible, minimally so, on radiographs. Dogs with urate stones most commonly have urate crystalluria on a fresh urine sample.

Urate stones are amenable to medical dissolution. However, medical dissolution is only considered effective approximately 30% of the time.[14] In the face of uncorrected liver disease, dissolution is not considered medically appropriate, as the medications target xanthine oxidase in the liver, and cannot be appropriately metabolized. Care should be taken to appropriately screen patients for portovascular anomalies with serum bile acid testing, protein C levels and/or diagnostic imaging. For dissolution, a purine-restricted diet that promotes an alkaline urine pH should be fed exclusively.[14–16] Allopurinol, a xanthine oxidase inhibitor, can be given at a dose of 15 mg/kg po q12 to inhibit conversion of xanthine to uric acid.[16] However, care should be taken as this medication can promote xanthine stone formation if purine intake is not controlled.[17] If, despite appropriate medical management, dissolution is not achieved, removal of problematic stones should be considered.

### *Cystine Dissolution and Prevention*

Cystine uroliths are composed of cystine molecules linked by disulfide bonds, and occur with hyperexcretion of cystine in urine.[18] Cystine stones can be suspected when there are multiple small ovoid stones with minimal radiopacity (see **Table1**). Because of the small size and minimal radiopacity, ultrasound can be a helpful diagnostic tool for detection. Cystine crystals are commonly seen in the fresh urine sample of dogs with cystine stones and should be carefully assessed. Cystine stones are more commonly found in dogs than cats, and in male dogs over females.[19] Over-represented breeds include Newfoundlands, Mastiffs, English Bulldogs, Labrador Retrievers, Irish Terriers, Scottish Deerhounds, Corgis, Australian Cattle Dogs, and Chihuahuas.[19]

In normal urine filtration, the cystine amino acid passes through the glomerular filtration barrier and is reabsorbed in the proximal convoluted tubule. In patients with cystinuria, there is a defect in the carrier proteins responsible for re-absorption of cystine. This defect can be the result of a mutation in a gene that affects appropriate function of these receptors in the proximal convoluted tubule, such as the SLC3A1 or the SLC7A9 gene.[20] The mutation in the SLC3A1 is at exon 2 and encodes for a renal basic amino acid transporter. This is the most common mutation in Labrador Retrievers.[20] The mutation in the SLC7A9 gene encodes intramembrane transporter protein, and this is most commonly found in Miniature pinchers.[20] Genetic screening is available for the recognized mutations and for testosterone-associated cystinuria. Testosterone-associated cystinuria has been reported in Mastiffs, Scottish Deerhounds, and Irish Terriers, and neutering is the recommended treatment.[21]

For the dissolution of cystine stones, a restricted-amino acid diet that is low in cystine, while also promoting alkaline urine pH (>7.5), should be fed exclusively. In addition chelators such as 2-mercaptopropionylglycine (20 mg/kg po q12 h) could be considered, and are reported to be effective in dissolving stones in approximately 53% of cases.[22] The addition of potassium citrate can also be considered. Neutering has been associated with decreased cystinuria in specific breeds of dogs.[20] If dissolution is not successful, or the cost of the medication is prohibitive, than removal of the stones can be considered, when necessary.

### *Calcium Oxalate Prevention*

Calcium oxalate stones are the most common stone type in both dogs and cats, and are most likely to form with hypercalciuria.[23–25] Hypercalciuria can occur secondary to various pathologic processes including: increased jejunal absorption of calcium, impaired renal tubular re-absorption of calcium, increased bone demineralization, metabolic acidosis, primary hyperparathyroidism, and feline idiopathic hypercalcemia. Hypercalciuria can occur with certain medications and dietary factors including loop diuretics, corticosteroids, urinary acidifiers, increased dietary calcium, increased intake of vitamins C and D, and low B6.[26–28] Hyperoxaluria has been reported to occur in dogs in which there is decreased degradation of dietary oxalate secondary to decreased enteric *Oxalobacter formigenes*.[29]

Male dogs are over-represented for developing calcium oxalate stones, as are several breeds including Bichon Frise, Cairn Terriers, Chihuahuas, Lhasa Apso, Pomeranians, Maltese, Miniature Schnauzers, Shih Tzu, Keeshond, Yorkshire Terriers, Mini Poodles, and Chihuahuas. Calcium oxalate stones can be suspected when radiographs show uroliths that are moderately to markedly radiopaque with sharp projections, and in feline patients in which there are radiopaque upper tract stones (nephroliths and/or ureteroliths) (**Fig. 2**; see **Table 1**), With calcium oxalate stones the urine pH is usually less than 7.0. A urine culture is most commonly negative, but in some cases secondary urinary tract infections are seen, most typically associated with either *E coli* or *Enterococcus* spp. In addition to imaging and urine testing, total body and ionized calcium levels should be evaluated with parathyroid testing if indicated. Calcium oxalate stones are not amenable to dissolution and therefore require removal or bypass if they are problematic (see treatment options below). However, if they are non-problematic nephroliths or ureteroliths, routine monitoring is recommended.

For prevention of additional stone formation, any known cause of increased calciuria should be addressed. Aiming for a non-acidic urine pH (6.8–7.2) and a specific gravity of less than 1.020 may help in slowing or preventing recurrence. Increased urine concentration also predisposes stone formation, so adding water to food and encouraging drinking is important. Urine-specific gravity should be <1.020 to encourage polyuria and crystal elimination rather than stagnancy. Potassium citrate (50–100 mg/kg po q12) can be given to help prevent aciduria and chelate calcium in the urine.[30] Hydrochlorothiazide (1–2 mg/kg po q12) can also be added.[31,32]

## REMOVAL OF PROBLEMATIC NEPHROLITHS

When nephroliths are problematic, removal is warranted. Considerations for nephrolithotomy include: (1) progressive loss of renal parenchyma secondary to increasing stone size, (2) pyelonephritis that is persistent despite appropriate duration of medical management, and (3) obstruction at the ureteropelvic junction with associated of hydronephrosis. Pain associated with nephroliths is usually only present with concurrent obstruction, pyelonephritis, or associated pyonephrosis.

Traditional open surgical options for nephrolithotomy include nephrotomy, pyelotomy, or a salvage ureteronephrectomy. Complications associated with these procedures can result in significant morbidity and mortality.[33–35] One study reports a 10% to

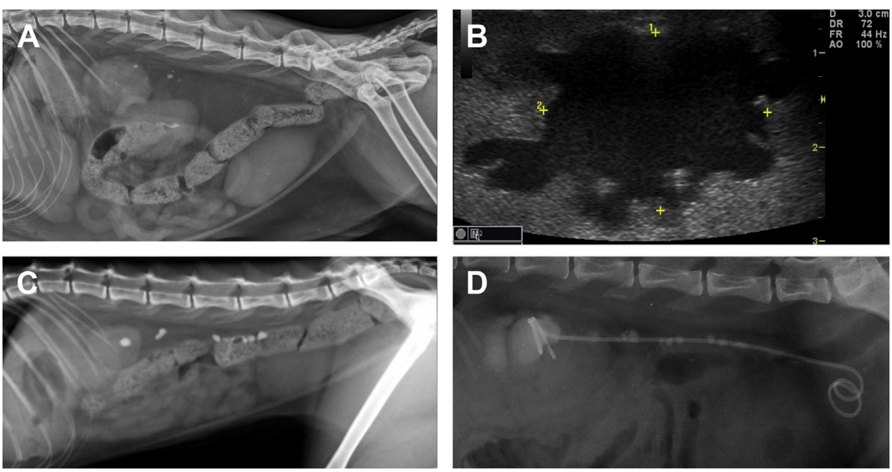


**Fig. 2.** *A*, *C*) Cats with multiple calcium oxalate ureteroliths. (*B*) Marked renal pelvic dilation in a cat secondary to obstructive ureterolithiasis. Note the calyx dilation. (*D*) Ureteral stent in a dog placed to relieve an obstruction secondary to multiple calcium oxalate ureteroliths.

20% decrease in glomerular filtration rate when healthy cats underwent a nephrotomy.[34] Nephrotomy would be expected to have a more detrimental effect on kidney function for clinical patients with previous kidney injury and exhaustion of compensatory mechanisms by the time intervention is being pursued. In a report of dogs undergoing traditional surgery for nephrolithiasis, there was a 23% complication rate associated with the procedure and approximately 43% of dogs had some stone fragments remaining after surgery.[35] For the dogs undergoing a nephrectomy, 67% of developed renal azotemia.[35]

Therefore, if stone removal is indicated, decisions on procedures to minimize kidney trauma and preserve kidney function should be considered. The 2016 ACVIM consensus statement on the management of urinary tract stone disease recommends that interventional techniques, such as extracorporeal shockwave lithotripsy (ESWL), percutaneous endoscopic nephrolithotomy, and surgically assisted endoscopic nephrolithotomy, should be considered over traditional surgery to decrease kidney functional loss for stone removal.[36,37]

### *Extracorporeal Shockwave Lithotripsy*

ESWL uses high-energy acoustic pulses to fragment stones into small pieces by creating microcracks in the stone, which expand until fragmentation occurs. The external shockwaves pass through a water medium (water bag), then a coupling gel, through soft tissue structures (eg, skin, muscle, fat, and the kidney/ureter), and then onto the stone(s). Fluoroscopy is typically used to direct the shockwaves onto the stone to ensure the energy is delivered in the appropriate plane. Shock waves of different energy levels are targeted at the stone anywhere from 1000 to 3500 times until fragments are small enough to pass through the ureter and into the urinary bladder for natural voiding.[38,39] However, fragments are typically at least 1 mm in diameter or larger making this technique difficult in very small ureteral lumens, such as the cat (~0.3 mm internal diameter). In addition to the high risk of developing ureteral obstruction, ESWL is not recommended for cats as feline calcium oxalate stones are resistant to fragmentation[21,40] In dogs, treatment is successful, with an 85% success rate and less than 1% mortality rate. However, 30% of dogs require more than one treatment to achieve the necessary degree of fragmentation, and 10% of dogs developed a transient ureteral obstruction as it can take weeks for all of the stone fragments to move into the bladder.[41] This technique is not recommended when the stone diameter is 1.0 to 1.5 cm or greater, as the amount of fragments and debris generated increase the risk for ureteral obstruction. Prophylactic ureteral stent placement before ESWL could be considered to help minimize the risk of ureteral obstruction and allow for passive ureteral dilation.[42,43] Transient hematuria is commonly seen. Pancreatitis is another reported complication and was documented in 2% to 3% of dogs.[44]

Although historically ESWL is thought to have minimal effect on the glomerular filtration rate (GFR) and the kidney itself, more recent reports in the human literature highlight short- and long-term effects that need to be considered before treatment. The repeated shockwaves necessary to cause stone fragmentation are also responsible for generating tissue damage, resulting in vascular hemorrhage. Dose-dependent renal fibrosis has been demonstrated in a canine experimental model.[45] Extracorporeal shockwave lithotripsy is contraindicated in patients that are coagulopathic. In addition, damage of surrounding tissues can occur, and in humans reported complications include: perforation of the colon, rupture of the hepatic artery, hepatic hematoma, pneumothorax, urinothorax, rupture of the spleen, acute necrotizing pancreatitis, dissecting abdominal wall abscess, rupture of the abdominal aorta, and iliac vein thrombosis.[46–58] These complications are very rare. Experimental models in pigs have shown that following certain protocols for energy settings and pulsing frequency, the injury can be reduced significantly.[59–66] Thus, ESWL is a viable option in our canine patients but should be performed by an experienced operator so that adverse side effects can be minimized.

In veterinary patients nephrolith size limitations are considered to avoid creating obstructive ureterolithiasis out of non-obstructive nephrolithiasis. The authors will always consider concurrent endoscopic ureteral stent placement before considering ESWL for this reason. The size limit for ESWL in the authors' practice is 1 to 1.5 cm in diameter. For stones larger than this, or composed of cystine, which are inherently ESWL resistant, an endoscopic nephrolithotomy is considered a better option.[40]

### *Endoscopic Nephrolithotomy*

Endoscopic nephrolithotomy can be considered with large nephroliths over 1 to 1.5 cm in diameter, or those that are ESWL resistant (eg, cystine). When compared with ESWL, laparoscopic-assisted nephrotomy, and traditional nephrotomy, endoscopic nephrolithotomy is reported to be the most kidney-sparing procedure, likely due to the spreading rather than transection of the renal parenchyma at the access point (**Fig. 3**).[67–69]

The approach can be percutaneous nephrolithotomy (PCNL) or surgically assisted endoscopic nephrolithotomy (SENL). These 2 procedures are performed similarly, using endoscopic and fluoroscopic guidance. The percutaneous approach uses ultrasonic guidance to gain access into the renal pelvis from the greater curvature of the kidney.[70] An 18-gauge renal access needle is directed onto the pelvis, onto the nephroliths. Once access is obtained, a pyeloureterogram is performed so the guide wire can be advanced into the renal pelvis, down the ureter, into the urinary bladder, and out of the urethra. This acts as the safety wire so that through-and-through access is maintained throughout the procedure. A dilation sheath is then advanced over the guide wire so that a balloon catheter (typically 8 mm) may be used to dilate a tract from the skin, through the renal parenchyma and into the renal pelvis. The balloon is inflated to 8 mm (24 F) and a 24 F access sheath is then advanced over the inflated balloon using fluoroscopic guidance, and onto the stone. The balloon is then deflated and the sheath remains in place providing access for a nephroscope directly onto the stone (see **Fig. 3**). Using intracorporeal lithotripsy, the stone is fragmented and pieces are either suctioned or extracted until the renal pelvis is free of stone material. After removal of all stone fragments, a double pigtail ureteral stent is placed to ensure patency in the event of ureteral spasm or passage of any remaining small fragments of mineral debris. When performed percutaneously, a locking loop nephrostomy tube is then placed to allow a nephropexy to occur at the access site and to prevent development of a uroabdomen. This tube remains in place for 2 to 4 weeks at home.

When performing this technique with surgical assistance (SENL), the same approach is taken but a routine laparotomy is performed, and, on completion of ureteral stent placement, the stoma in the kidney is closed with a single mattress suture. No nephrostomy tube is left in place.

The main risks of this procedure include hemorrhage, ureteral perforation, and urine leakage from the renal access tract. Use of the balloon dilator and sheath combination, as described above, helps to tamponade any potential bleeding that could occur during access. These procedures are thought to be the most kidney-sparing option for large nephrolithotomy as the renal parenchyma is stretched rather than transected for stone removal. PCNL/SENL was recently reported in 11 dogs and 1 cat.[70] The authors in this study preferred to modify the technique from a PCNL to SENL to allow for closure of the renal tract, which was preferred by the owners at home and technically

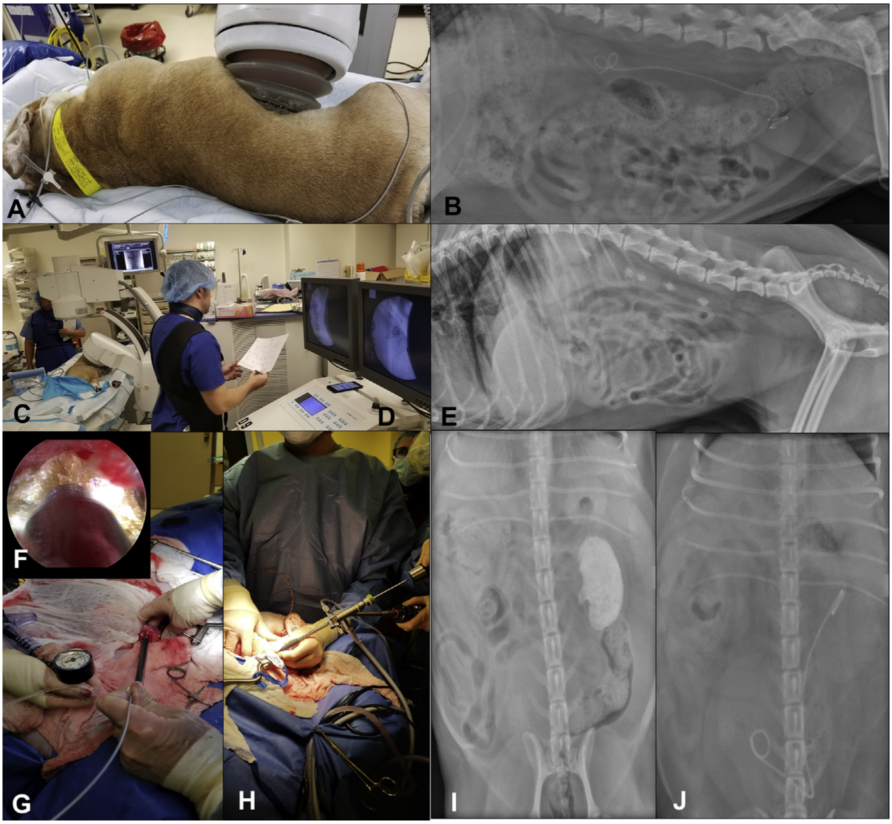


**Fig. 3.** (*A, C*) Positioning and monitoring for ESWL. Note the contact of the unit with the patient for appropriate conductivity. (*B, E*) Pre- and post-ESWL. Note stent placement to ensure patency while any fragments pass. (*D*) Note the continued monitoring via fluoroscopy during shockwave. (*F*) Endoscopic view of the renal pelvis during an SENL. (*G*) Balloon dilation to spread apart renal parenchyma and allow for placement of a nephroscope. (*H*) Positioning of the nephroscope and lithotripter. (*I, J*) Pre- and post-SENL.

provides a more caudolateral access point, expediting the procedure for stone removal and ensuring ureteral patency at completion. In this report, patient size was not seen to be a factor in success (range, 2.3–49 kg), but the appropriate equipment and proper training are necessary for a successful outcome.

### *Obstructive Ureterolithiasis*

Ureteral obstructions are a serious condition in veterinary patients, and with the advent of high-quality imaging and awareness they are becoming more frequently diagnosed in our canine and feline patients. Because of kidney functional loss associated with the increased hydrostatic pressure of a ureteral obstruction, this condition does require timely intervention.[71–73] In an experimental model using dogs with no pre-existing azotemia, a complete ureteral obstruction resulted in an immediate increase in renal pelvic pressure, decreased renal blood flow by 60% within the first 24 hours, and acute decrease in the glomerular filtration rate.[71] After 7 days, GFR was permanently decreased by 35%, and after 2 weeks by 54%. In clinical patients, where kidney damage has likely already been done, no hypertrophic mechanisms occur to aid in kidney functional recovery, and a worse outcome would be expected.

The composition of most ureteroliths in dogs (50%–60%) and cats (>92%) are calcium based, making dissolution completely contraindicated in cats, and should not be considered in dogs with struvite stones that have not yet accomplished ureteral decompression.[4,74–76] With medical management being effective in few feline cases (8%–13%), and traditional surgical interventions being associated with a relatively high post-operative complication rate (30%–40% in cats and ~7%-15% in dogs) and peri-operative mortality rate (18%–21% in cats and 6.25% in dogs), short-term medical therapy should be considered before any intervention (24–48 hours), if possible.[2,75–78]

The most common complications associated with surgery were due to ureterotomy site edema, re-obstruction from nephroliths that pass to the surgery site, stricture formation at the surgical site, missed ureteroliths not removed, and surgery-associated urine leakage.[2,75] Because of the high mortality rates and high complication rates, alternatives have been developed for animals over the past decade to avoid traditional open ureteral surgery.

Interventional alternatives have shown to result in immediate decompression, fewer peri-operative complications, lower peri-operative mortality rates, successful treatment for all causes of obstruction (stone, stricture, tumor), and a decreased recurrence rate of future obstructions, when compared with traditional options (40% with traditional surgery or medical management within 1 year).[4,74–76] The etiology of feline ureterolithiasis was recently shown to be associated with a ureteral stricture in approximately 33% of cases making surgical repair a far more complicated proposition.[4,76] Interventional therapies, like ureteral stents or the subcutaneous ureteral bypass (SUB) device are more amenable for all causes of ureteral obstructions.

Medical management for the treatment of ureterolith-induced obstructions typically uses a combination of intravenous fluid therapy and an alpha-adrenergic blockade with mannitol. In the authors' experience approximately 10% success is seen with medical management alone.

### *Ureteral Stenting*

The use of double pigtail ureteral stents has been reported in dogs and cats with excellent outcomes as an alternative to traditional surgery.[4,74,78–82] Ureteral stent placement allows for immediate kidney decompression, a decreased risk for ureteral stricture and urine leakage after surgery, and a decrease rate of obstruction recurrence. In feline patients ureteral stenting improved the mortality associated with traditional surgery (7.2%), but was associated with a 38% rate of long-term dysuria, most of which resolved with steroid therapy.[4] Nineteen percent of ureteral stents in cats needed to be exchanged, most commonly due to re-obstruction from a ureteral stricture or ureteral stent migration. Since the advent of the SUB device, both the short- and long-term complications have improved for ureteral obstructions in cats, and ureteral stents have been more reserved for dogs, in the authors' practice.

In dogs, ureteral stents can most commonly be placed endoscopically and fluoroscopically guided.[74] Passive ureteral dilation has been reported to occur within 2 weeks of placement, and in this report the dilation was significant enough to allow for post-stenting ureteroscopy with a 3.1-mm ureteroscope within 2 weeks.[43] Complications are uncommon but include stent occlusion, compression, migration, and proliferative tissue at the ureterovesicular junction. Ureteral stents needed to be exchanged in 14% of dogs, and this was most commonly due to obstructive ureteritis (**Fig. 4**). In dogs, ureteral stents are typically outpatient procedures performed during retrograde cystourethroscopy. This is the authors' preferred treatment for obstructive pyonephrosis allowing for renal pelvic lavage and decompression of the obstructive lesion simultaneously, avoiding invasive surgery.[83]

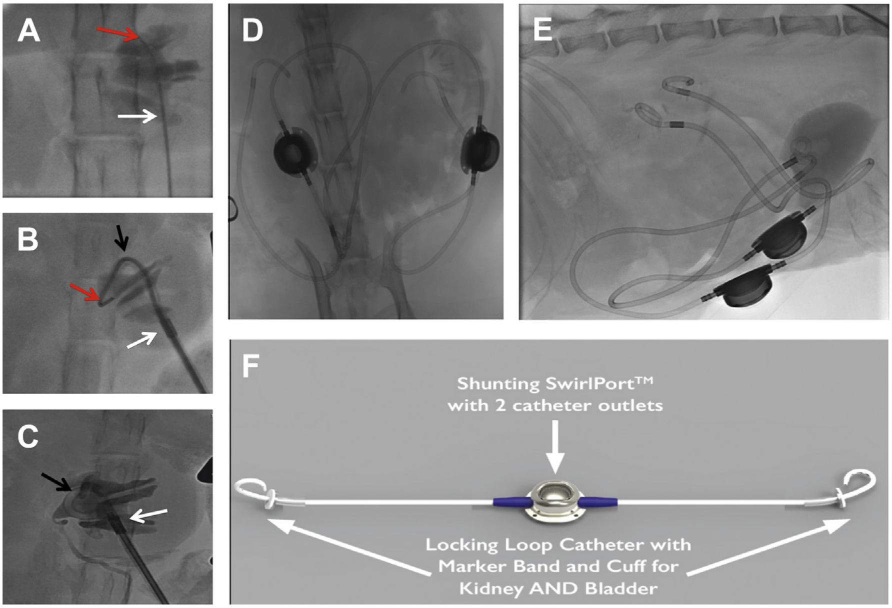


**Fig. 4.** Fluoroscopic images during placement of the SUB device. (*A*) A modified Seldinger technique is used to gain access to the renal pelvis. Note the guide wire (*red arrow*) being fed through an 18 g catheter (*white arrow*). (*B*) The 18 g catheter has been removed, and the nephrostomy tube (*black arrow*) is being fed over the guide wire (*red arrow*). Note the radiopaque marker (*white arrow*) that identifies the end of the fenestrations in the nephrostomy tube. (*C*) The guide wire has been removed and the pigtail (*black arrow*) has been locked in place. The radiopaque marker (*white arrow*) is positioned to ensure all fenestrations are within the renal pelvis. (*D*) Bilateral SUB with each nephrostomy catheter positioned so that is, going down the ureter rather than coiling the renal pelvis. This is preferred when there is a small degree of renal pelvic dilation. (*E*) Lateral view following bilateral SUB placement. Note the radiopaque markers on the cystostomy catheters are positioned inside the bladder. (*F*) A SUB device with 2 catheter outlets. This is preferred to a 3-way port in the case of any benign obstruction.

In most cases, retrograde stent placement via an endoscopic approach can be performed. In a study of 47 dogs with 57 ureters undergoing stent placement for various benign obstructions, long-term complications included urinary tract infection (26%), occlusion of the stent (9%), suspected ureteritis (5%), stent migration (5%), encrustation of the stent (2%), and hematuria (7%).[74] For the dogs experiencing infection post-procedure, all had infection present before stent placement and 72% were ultimately able to clear the infection.[74] If stones are suspected to be struvite, dissolution should be attempted following kidney decompression, and following dissolution the stent can be removed endoscopically.[74] If another stone type that is likely to be recurrent is suspected, the stent could be left in place long-term provided there are no complications. If stent exchange is indicated, the procedure can typically be performed on an outpatient basis.

### *Subcutaneous Ureteral Bypass Device*

The SUB device is an artificial ureter that consists of a combination of a locking loop nephrostomy tube, a cystostomy tube, and a subcutaneously placed metallic shunting port that connects the 2 catheters (Fig. 5). This device allows urine to flow from the

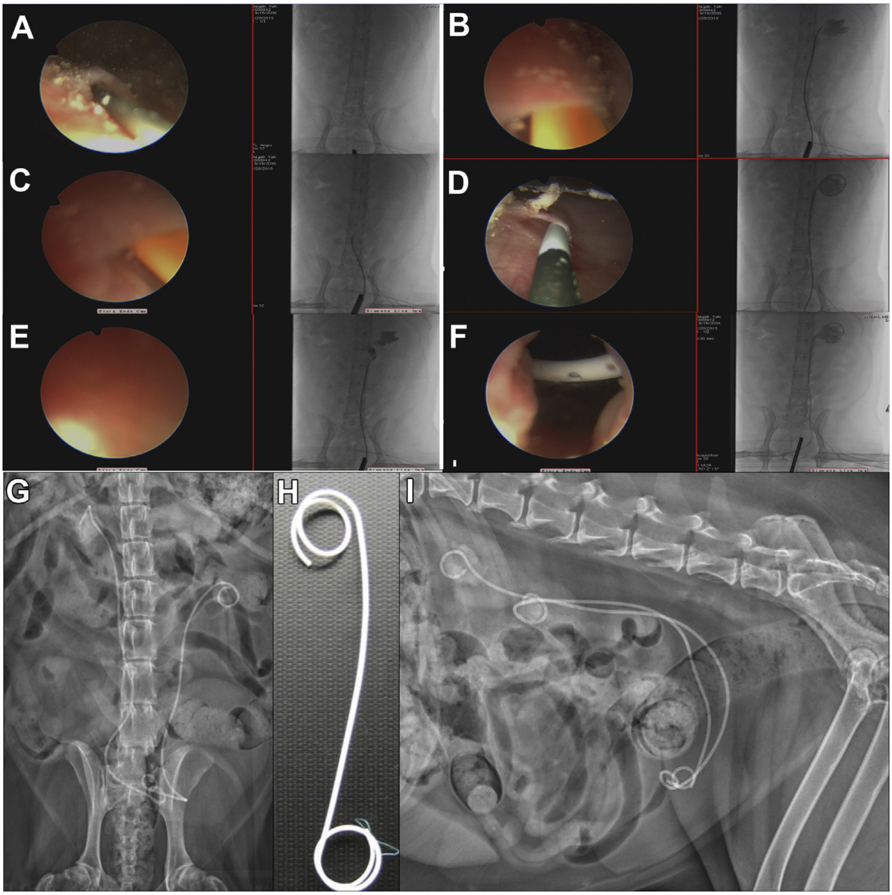


**Fig. 5.** *A–F*) Endoscopic and fluoroscopic retrograde placement of a ureteral stent in a dog. Note positioning of a guidewire into the ureteral orifice followed a ureteral marker catheter so that a ureteropyelogram can be performed in preparation for stent deployment. (*G, I*) Post-procedure radiographs confirming appropriate stent placement. (*H*) Example of a multi-fenestrated double -pigtail ureteral stent.

kidney to the urinary bladder without any manipulation of the ureter, avoiding the need to address the challenging underlying etiology (eg, stone(s), stricture, tumor, extraluminal compression) causing the ureteral obstruction.[83,84]

The use of the SUB device was recently reported for the treatment of benign ureteral obstructions 134 cats (174 ureters) with good to excellent outcomes.[76] In this population 33% of obstructions were due to strictures, 33% were bilaterally obstructed, and over 95% of cats were azotemic despite a unilateral ureteral obstruction. In this population the peri-operative mortality rate was 6.2%, the lowest for any procedure reported to date, and the median procedure time is 45 minutes. The median creatinine at presentation was 6.6 mg/dL and 2.4 mg/dL at the time of discharge. The overall mean survival time was 827 days, and the mean survival time for a non-renal cause of death was over 2251 days. There was no pre-operative predictor of long-term survival.

The most significant complication long-term was device mineralization (24%), with 13% developing a re-obstruction requiring a SUB device exchange. The other 11%

developed a patent ureter and were no longer obstructed despite a poorly flowing SUB device. Since the routine flushing protocol was performed using a tetra-EDTA solution, the mineralization rate declined to 4.5% (Berent, unpublished data collected between 2013 and 2017). Chronic urinary tract infections were seen in 8% of patients, and this was most common in cats that were infected before SUB device placement. Compared with feline ureteral stents, the SUB device was associated with dysuria in 8% of cats, most of which had a history of dysuria before their SUB placement.[76]

In the authors' practice, this device has essentially replaced the use of traditional surgery and double pigtail ureteral stents for the treatment of ureteral obstructions in feline patients. This is due to the combination of the ease of placement, the decreased rates of dysuria and re-obstruction, and the low peri-operative mortality.

The SUB device has also been used in a small group of dogs. A recent report showed that the SUB device in dogs was effective in relieving the ureteral obstruction in all cases, most of which were from a failed ureteral stent, but was associated with a 50% rate of device mineralization and ultimately SUB occlusion (Milligan M, Berent AC, Weisse C. Outcomes of SUB in dogs with benigng obstructions: 9 cases [2013-2017]. Unpublished data collected between 2013 and 2017). Because of this finding, and the low rate of ureteral stent occlusions, the authors prefer ureteral stents in dogs and the SUB device in cats.

## SUMMARY

Interventional options for the treatment of upper urinary tract obstructions in veterinary medicine has dramatically expanded over the past decade, and is continuing to do so. This is following the trend that has been occurring in human medicine over the past 30 years, and will likely continue to grow in our profession as more clinicians are getting trained, more literature is being published to show evidence of superior methods evolving, and clients are demanding higher standards for their pets.

It is highly recommended that operators get proper training before considering all of the procedures described as the learning curve is steep and complications should be avoided whenever possible. Training laboratories are available to further develop these skills. In addition, treatment of stone disease should always consider the stone type and the ideal method for bypass, removal, or monitoring.

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