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Emergency Surgery Mini Series

Session 3: Surgical Emergencies of the Urinary System

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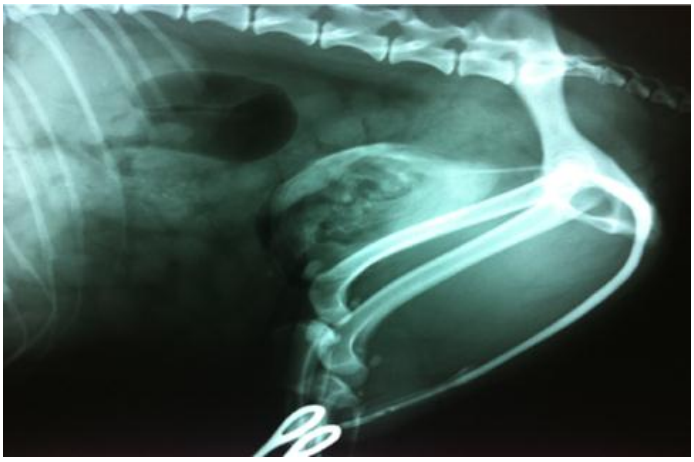

Emergency management of the patient with urinary tract obstruction

Introduction

The most common form of urinary tract obstruction faced in general practice is the patient (canine or feline) with urethral obstruction. There are many potential causes such as obstructive urolithiasis, FLUTD, FIC, trauma, spasm, stricture, neoplasia, bladder herniation and inflammation e.g. granulomatous urethritis. Regardless of the cause, a predictable sequence of metabolic derangements may ensue. A clear understanding of the pathogenesis is imperative so that appropriate diagnostic tests can be performed to identify them, and so that tailored treatment can be instituted to stabilise the patient. Furthermore, ongoing monitoring of these parameters is required to assess the response to therapy.

The severity of clinical signs and indeed the severity of the physiological derangements will depend on the completeness and duration of the obstruction. This will encompass the acute partial obstruction that is systemically well with no metabolic derangements to the acidotic, shocked patient. The aggressiveness of the stabilisation and treatment will reflect this. In all cases urethral obstruction should be treated as an EMERGENCY as azotemia develops after 2-3 days and death after 3-6 days if not resolved. Furthermore, prolonged bladder distension may result is permanent detrusor damage and a dysfunctional atonic bladder.

The physiological effects of post-renal obstruction

1. Volume deficits

Most patients will have some degree of dehydration with the severity depending on the presence or absence of concurrent vomiting, diarrhoea or reduced water intake. This reduces peripheral tissue perfusion which by definition is shock. The reduced tissue perfusion causes a decrease in renal perfusion and will incite a degree of pre-renal azotemia and acidosis. Long-standing cases with complete obstruction can be volume depleted and in hypovolemic shock.

2. Electrolyte abnormalities

The most important is hyperkalemia. The kidney is responsible for excretion of excess potassium from the blood. Obstruction results in a failure of Potassium excretion through the renal tubules and a resultant rise in serum levels. Patients with post-renal obstruction usually become acidotic. Potassium (K+) is an intracellular ion. During periods of acidosis, K+ ions are translocated from the cells in exchange for Hydrogen ions (H+) thus increasing the

severity of hyperkalemia. Other electrolyte abnormalities can also occur for example hyponatremia or hypochloremia if the patient has been vomiting. These also need to be identified and corrected. Hyperkalemia has profound effects on cardiac electrical activity because it lowers the resting potential of the cardiac myocyte. These manifest as bradycardia, spiked T waves, widened QRS, prolonged PR interval and potentially ventricular arrhythmias and arrest. These patients represent a significant anaesthetic risk and these need to be corrected first with continued monitoring thereafter.

3. Azotemia

Most patients will have some degree of azotemia. A degree of this may be pre-renal in origin because of the volume depletion and reduced renal perfusion. Of course many animals presenting with renal obstruction may also have underlying renal disease that could be a contributing factor. However, the most significant is the post-renal obstruction. This prevents the excretion of urea and creatinine from the renal tubules thus elevating serum levels significantly. Unfortunately it is not possible to determine how much each component contributes to the azotemia initially and therefore one cannot guarantee that this will normalise after treatment. The signalment and history can often help determine the likelihood of pre-existing renal disease. Azotemia affects the pharmokinetics and metabolism of many anaesthetic drugs and complicates anaesthesia. It can also interfere with platelet function and increase the risks of urinary tract haemorrhage. Attempts should be made to improve this beforehand but it is often less correctable that the volume and electrolyte abnormalities without relieving the obstruction or performing urinary diversion.

4. Pain

Urinary tract obstruction is an extremely painful condition and the significance and effects of this are frequently underestimated. It may be more difficult to recognise signs of pain in these patients compared to other disease or injuries and therefore a proactive approach to analgesia should be employed.

Initial investigations

Initial tests focus on the identification of the above metabolic derangements and their severity. A blood sample should be collected and a minimal data-base would include:

- PCV/TP
- Electrolytes (especially Potassium)
- Urea / Creatinine
- pH

Ideally a full biochemistry, electrolyte and acid-base analysis should be performed but the above parameters will allow a reasonable assessment of the metabolic status of the patient and direct initial fluid and medical therapy. These tests will need to be repeated frequently throughout the treatment period to ensure successful resolution of the abnormalities.

Specific diagnostic imaging to determine the underlying cause (e.g. radiography, ultrasound or contrast studies) come later once the patient is stabilised.

Initial stabilisation

1. Fluid therapy and volume support

Good peripheral intravenous access should be secured and intravenous fluid therapy instituted. The choice of fluid is dependent on the exact electrolyte and acid-base status. Typically 0.9% NaCL is the most appropriate choice. However, the kidneys are a clever organ and so long as peripheral tissue perfusion is re-established then they can often improve the situation. Although Hartman's solution (lactated Ringer's) contains a small amount of potassium this is not usually of clinical relevance because it alkalinising effect will drive potassium ions back into cells. The initial rate of fluid administration will depend on the degree of volume depletion or shock. Guidelines are 90 mls/kg for dogs and 55 mls/kg for cats. However I find these somewhat empirical and prefer to administer repeat boluses of 10-20 mls/kg until I improve the patients hemodynamic (HR, pulse quality, CRT etc.) and metabolic parameters (as assessed by repeat blood work). This way fluid therapy is tailored more specifically toward the individual patient.

2. Bladder decompression or temporary urinary diversion

It is important to initially decompress the bladder especially if it is over-distended. This will help prevent permanent damage to the detrusor muscle and relieve pressure on the renal tubules. Accompanied with appropriate fluid therapy this will help establish diuresis and improve metabolic parameters. There are several ways to do this and the most appropriate will depend on the individual case. Attempts to forcefully manually express the bladder should NOT be undertaken as this could result in bladder rupture.

In the relatively stable patient where there is a diagnosis of reversible urethral obstruction (e.g. cat with FLUTD or dog with urolithiasis) then they may be sedated or anaesthetised for an attempt at urethral catheterisation or retrograde urohydropulsion (see below for description of the techniques). Great care must be taken during attempts at catheterisation as iatrogenic urethral rupture could result. After successfully relieving the obstruction and catheterising the bladder, it is completely emptied and a sample is kept for urinalysis. The catheter is generally replaced with a soft silicon Foley type catheter pending definitive treatment or continued medical therapy.

Some patients will have more severe metabolic derangements that prevent sedation or anaesthesia for urethral catheterisation or may have more challenging urethral obstructions that will not be straight-forward to relieve (e.g. neoplasia). In these patients, gentle cystocentesis should be performed initially. This will achieve a temporary decompression of the urinary tract and help establish diuresis. This will facilitate ongoing correction of the metabolic abnormalities so that more involved diversion or definitive treatment can be performed later.

Ideally one does not want to repeatedly perform cystocentesis as this is painful for the patient and risks urine leakage into the peritoneal cavity. In this instance, if the patient is not ready for definitive resolution of the urethral obstruction then urinary diversion by way of a cystostomy tube is a good alternative. The traditional description for cystostomy tube placement involves an exploratory coeliotomy under GA and is obviously quite involved and probably does not lend itself well in this situation. Two good alternatives exist. The first is the use of a pigtail catheter. These can be placed over a guide wire similar to the Seldinger technique (ideally under ultrasound guidance). These are usually straightforward to place especially if the bladder is distended and easily palpable through the abdominal wall. Because they have a small diameter they can often be placed under sedation and local analgesia in systemically ill patients. They provide a good way to drain the bladder repeatedly over a short period (~few days) whilst the patient is stabilised adequately for definitive treatment.

The second option is place a standard cystosomy tube using the minimally invasive inguinal approach recently described (Bray et al 2006). The bladder is accessed via a minor grid laparotomy just cranial to the inguinal skin fold. Following the placement of several cystopexy sutures to secure the bladder to the body wall a purse string in placed on the lateral bladder wall (as for a standard cystostomy). The tube is then brought into the peritoneal cavity through a separate small incision. A stab incision is made into the bladder lumen in the middle of the purse string and the tube is inserted and balloon inflated with saline. The purse string is tied and the body wall and skin are sutured routinely. Finally, a finger trap secures the tube to the skin at the stoma in a usual manner. This technique is much less invasive and much more rapid than the traditional one. In some moribund animals it can be performed with sedation and local anaesthesia alone, or a very short anaesthetic (15-25 mins) in those that are less affected. This can be used for temporary urinary diversion for as long as necessary before definitive treatment of the urethral obstruction.

3. Correction of hyperkalemia

In the vast majority of patients, establishment of diuresis with intravenous fluids and bladder drainage will normalise the potassium levels. Some more severely affected patients may require more aggressive medical treatment if they fail to respond fully to the above therapy or demonstrate significant cardiac abnormalities.

There are several options for patients that require more aggressive treatment to lower potassium levels.

(i) Dextrose saline infusion: This aims to stimulate insulin release which in turn will drive potassium intra-cellularly hence lowering serum levels. Pure dextrose or dexrose in half strength saline should be avoided if the patient has concurrent hyponatremia.

(ii) Dextrose saline infusion plus insulin: This can be used if dextrose alone is unsuccessful. 0.5 iu/Kg of regular insulin is given IV followed by 2g of dextrose (4mls of the 50% solution) per unit of insulin and administered as a 2.5% constant rate infusion. The effect should be seen within one hour of administration. Blood glucose levels should be monitored to ensure the patient does not become hypoglycaemic.

(iii) Bicarbonate therapy: Intravenous bicarbonate can shift potassium into cells in exchange for H+ ions. It is usually employed for the treatment for severe acidosis (see below) rather than hyperkalemia but it may be useful particularly if there is concurrent acidosis that

requires its use. It needs to be administered extremely slowly (over 30 mins) and an ECG should be monitored throughout.

(iv) Loop diuretics: These drugs are uncommonly used but result in active renal secretion of potassium and can help lower serum levels. Because water is also lost it is important that the patient is well hydrated before use of these is contemplated and that a urine flow is established either naturally or by some form of urinary diversion.

(v) 10% Calcium gluconate: Although this does not lower potassium levels it may provide transient myocardial protection from the arrhythmogenic effects of hyperkalemia whilst awaiting other therapy to take effect. 0.5-1.0 ml/Kg should be given slowly over 10-20 minutes whist an ECG trace is monitored. If severe bradycardia develops then the infusion is stopped.

4. Correction of metabolic acidosis

Similar to the situation with hyperkalemia, it will be possible to resolve acidosis in most patients after institution of fluid therapy and establishment of diuresis. In patients that fail to respond to volume restoration or have profound metabolic acidosis (pH < 7.0) then one should consider bicarbonate therapy.

This decision should not be undertaken lightly because administration of bicarbonate has the potential to cause severe complications. Additional electrolyte disturbances (hypokalemia, hypernatremia and hypocalcemia) and metabolic alkalosis may occur. This increases haemoglobin affinity for oxygen that can reduce oxygen delivery to tissues (= shock). Bicarbonate has a very high osmolarity hence it can cause significant shifts of fluid into the vascular space or interstitial spaces resulting in cellular dehydration. Finally it is metabolised into carbon dioxide - if a patient has impaired ventilation and cannot expel this, then a paradoxical respiratory acidosis develops and the CO2 can diffuse into the CSF producing a CNS acidosis. Therefore one must be able to monitor a patient throughout administration and have the ability to repeatedly measure and interpret blood-gases.

The dose for bicarbonate is:

NaHCO3 (mEq) = 0.3 x bodyweight (Kg) x Base deficit (mEq/L)

It is best to give half the calculated dose slowly over a few hours and then re-assess the acid-base balance, only giving the remainder if required. The aim of therapy would be to achieve a pH of 7.2 and normalised potassium levels.

There are empirical dose ranges available for bicarbonate therapy (e.g. 1-2 mEq/Kg given over 20-30 mins) for when the base excess is not known, but caution is advised when one cannot monitor the response and referral to a specialist centre should be considered.

Tips for urethral catheterisation

As mentioned above, urethral catheterisation is the simplest way to decompress a full bladder and in cases of obstructive urolithiasis or cats with FLUTD it may also be therapeutic. In most cases it requires at least sedation and in fact in cats I prefer general anaesthesia as it tends to provide greater urethral relaxation. It is therefore only appropriate to do this in either; a patient that presents early without significant metabolic derangements or after stabilisation in those patients that are systemically unwell. If it is planned for after stabilisation then other methods of bladder decompression (cystocentesis or temporary cystostomy tube) need to be considered in the interim.

Dog

In dogs catheterisation is usually required because of obstructive uroliths. These may be diagnosed by plain or contrast radiography. Following diagnosis catheterisation is usually performed with a 6F-10F standard urinary catheter with the aim of displacing the urolith(s) into the bladder. The catheter should be well lubricated with KY jelly prior to attempting to pass it. The penis should be prepared with dilute iodine antiseptic and the tip of the catheter needs to be kept sterile. The catheter needs to be kept within its sterile wrapper and 'fed out of it' as it is advanced to prevent contamination. It is useful if an assistant can keep the penis extruded from the prepuce throughout the procedure. If not then it can be temporarily extruded to allow insertion of the catheter tip into the urethra. It should be well inserted before releasing to prevent it slipping out and getting directed into a dead-end of the prepucial fornix.

Uroliths typically obstruct the urethra at the base of the os penis. Advance the catheter until the obstruction is met. A water soluble lubricant and saline mix is infused under pressure with a gentle backward and forward movement of the catheter. If the urolith(s) can be palpated, then gentle massage can help displace them. (In the more unusual situation that they have obstructed the urethra at the ischial arch then they may be palpated/massaged per rectum). If

the urolith(s) are successfully displaced in a retrograde direction, then the procedure is repeated as many times as necessary to re-locate them into the bladder. Great care needs to be taken to prevent urethral trauma/rupture and the catheter should NEVER be forced. If attempts at catheterisation fail, then retrograde urohydropulsion should be considered (see below).

Cat

Just like the situation in dogs the urethral obstruction can often be palpated in the penile urethra or the pelvic urethra (per rectum). Gentle massage can be helpful at dislodging the obstruction. In cats with FLUTD, the obstruction may consist or urethral plugs, mucous, crystals or uroliths often compounded by urethral spasm. The penis should be extruded from the prepuce and gentle traction applied in a caudal and dorsal direction. This will straighten the urethral and help passage of the catheter.

A 3.5F, end-hole, atraumatic catheter (without a stylet) should be used as this will allow gentle flushing as it is advanced. Once again, it should be lubricated with KY jelly and kept sterile to reduce the chance of introducing bacteria. As the catheter is slowly advanced, gently flush with sterile saline to help relieve the obstruction. Progress is often slow and gradual but patience required so that it is NEVER forced as this risks swelling that will hinder progress or worse still, cause urethral rupture. If the obstruction is within the first few mm of the penile urethra and proving difficult, I have found it useful to use an intravenous catheter instead. The shortened length and small diameter seems to be helpful at relieving these obstructions. As soon as the obstruction is alleviated then one can return to using a longer urinary catheter to reach and decompress the bladder.

The bladder should be completely emptied of urine and then repeatedly flushed with sterile saline until clear. I normally leave a catheter in place for 1-4 days in cats with obstructive FLUTD. It is important that the catheter is replaced with one that is suitable for long-term use (e.g. atraumatic silicon catheter). Most cats to not appreciate repeated bladder emptying therefore I tend to attach these to a closed collection system. Commercial ones are available but an empty intravenous fluid bag and extension set can be adapted to suit this function. The catheter is sutured to the prepuce with fine suture material. The collection system should be taped to the tail to prevent it pulling on the prepuce and an Elizabethan collar should be placed. If attempts at catheterisation fail then retrograde urohydropulsion may be performed by adapting the technique used in the dog (see below) although it is more challenging in cats due to their small size.

Retrograde urohydropulsion

Defined as the use of a catheter and sterile fluid / lubricant, to relieve urethral obstruction, by gently propelling uroliths from the urethra back into the bladder under pressure. Correctly performed this can lead to successful resolution of urethral obstruction in the vast majority of cases. Sedation or general anaesthesia coupled with good analgesia (usually opiates) is usually required to perform this procedure.

Technique

- Restraint the patient in lateral recumbency with the upper limb drawn caudally.
- Draw back the prepuce to expose penis and the urethral opening.
- Lubricate the tip of catheter and pass it into penile urethra whilst maintaining sterility.
- Advance it up to the level of the obstruction - typically between the base of the os penis and the ischial arch.
- Attach the syringe of saline / lubricant mix (45 ml of sterile saline with 15 ml of water-soluble lubricant in 60 ml syringe) to the catheter. This lubricates the urolith(s) and the urethral wall at the level of the obstruction assisting in their displacement.
- Occlude penis around catheter with finger and thumb or better still an atraumatic clamp.
- Place finger per rectum to palpate and occlude intra-pelvic urethra proximal to obstruction (It can be helpful to have an assistant do this).
- Inject the saline / lubricant mix from the syringe until pressure builds in the intra-pelvic urethra - this was palpably dilate urethra.
- Quickly release finger pressure from the intra-pelvic urethra.
- This should cause a rapid pressure change and propel the urolith(s) back into the bladder. Urolith(s) will often be felt passing the tip of your finger.
- Repeat if necessary, advancing the catheter as calculi become dislodged until it reaches bladder.
- This procedure has an extremely high success rate at relieving obstructing urethral uroliths in dogs and it is the exceptional case that requires surgical management (e.g. urethrotomy).
- A cystotomy can then be performed to remove retropulsed uroliths.
- If metabolically stable, patients should proceed directly to cystotomy if possible to prevent repeat obstruction.
- If cystotomy is not going to be performed immediately (e.g. further stabilization required), then leave a large diameter (ideally silicon based) urinary catheter in place to prevent repeat obstruction.

Post-obstructive diuresis

Relief of a post-renal obstruction causes massive diuresis. There is usually a temporary loss of renal concentrating ability which increases the potential for significant fluid and electrolyte (mainly potassium and sodium) losses. It is therefore essential that these are quantified and replaced as necessary. Urine output loss and intra-venous fluids are titrated to ensure that the fluids are replaced and blood should be collected twice daily (for 1-2 days) to measure electrolyte and acid-base parameters so that they can appropriately supplemented. Obviously leaving a urinary catheter connected to a closed system in place facilitates this.

Surgical management of urethral obstruction

In canine patients urethral obstruction is typically caused by obstructive urolithiasis. In all but the most challenging cases urethral obstruction secondary to uroliths can be relieved by retrograde urohydropulsion. These cases then require a cystotomy (standard or video assisted) to retrieve them from the bladder. In those exceptional cases in which the obstruction cannot be relieved or when there is repeated obstruction or stricture at the level of the os penis (despite appropriate medical management) then a prescrotal urethrotomy or a scrotal urethrostomy may be appropriate respectively. THESE PROCEDURES ARE NOT EMERGENCIES AND ARE THUS ONLY PERFORMED AFTER MEDICAL STABILISATION.

Cystotomy

Relevant anatomy

The bladder is located in the caudal abdomen and/or pelvic canal in the dog depending on size. It is composed of a mucosa (transitional uroepithelium), submucosa (strength holding layer), detrusor muscle and serosa. Its wall is thin unless it is diseased. It is held by a ventral median ligament and two lateral reflections of the peritoneum - the lateral ligaments of the bladder. The ventral median ligament attaches to the linea alba and may be cut to improve exposure and mobility. The distal ureters travel in the lateral ligaments before entering the dorso-caudal aspect of the bladder at the trigone and need to be avoided during surgery. The blood supply is via the cranial and caudal vesical arteries originating from the umbilical and urogenital arteries respectively. The cranial blood supply is only present in ~50% of dogs. The hypogastric and pelvic nerves supply sympathetic and parasympathetic innervation to the bladder respectively and the pudendal nerve supplies somatic innervation to the external sphincter (and striated muscle of the urethra). These are located dorsal to the bladder, close to the bladder neck and this area should be avoided.

Bladder healing and basic principles

The bladder heals rapidly in most cases and reaches ~100% of its normal strength within 2-3 weeks. An absorbable suture material is therefore suitable. Monofilaments are preferred over multifilaments as there is less bacterial adherence and less tissue drag. Polydioxanone or polyglyconate are suitable choices. The former may have slightly better performance in infected urine. Poliglecaprone may not have sufficient tensile strength especially in infected urine. One should use a swaged on needle with a taper point to minimise trauma to the bladder wall. The bladder should be handled with stay sutures and atraumatic forceps

(Debakey) and use of electrocautery should be minimised to limit swelling. Various suture patterns (one vs. two layer and interrupted vs. continuous) have been assessed and there is no good evidence to support one over the other. A simple continuous appositional pattern is suitable in most cases as it is rapidly placed and creates a robust closure with good resistance to urine leakage. The suture should engage the submucosa as this is the strength holding layer but avoid penetrating the mucosa as suture could act as a nidus for urolith formation or indeed get exposed to infected urine thus speeding hydrolysis. This can be challenging to perform in very thin bladders. If there is concern regarding urine leakage then a second inverting layer (Cushing or Lembert) may be placed.

Approach

Peri-operative antibiotics are usually warranted as the most common urolith (struvite) in dogs is associated with infection. One may wait until samples are collected intra-operatively before administration. A caudal, ventral midline coeliotomy approach is used. In male dogs the skin incision starts at the umbilicus but needs to curve into a parapreputial position from just cranial to the prepuce. The subcutaneous tissue and preputial muscle (and associated vessels) need to be divided along this line. The prepuce is retracted to allow the linea alba to be divided on the midline from the umbilicus to the pubis. Care should be taken not to inadvertently stab the bladder during the approach, although the bladder is not usually over-distended as some method of decompression (catheterisation, cystocentesis) will have been performed before surgery whilst the patient was stabilised. The ventral median ligament of the bladder attaches to the linea and can be divided. The edges of the body wall can be protected with moistened laparotomy sponges and an abdominal retractor (Gosset or Balfour) used o maintain retraction. The bladder can be isolated and packed off with moistened swabs. NB: It is ESSENTIAL TO COUNT IN ALL SWABS AT THE BEGINNING OF THE PROCEDURE AND COUNT THEM ALL OUT AT THE END TO DOCUMENT THAT NONE HAVE BEEN INADVERTENTLY LEFT BEHIND.

Cystotomy

It is useful to place a stay suture into the bladder apex to facilitate atraumatic manipulation. Gentle palpation of the bladder often detects the cystic uroliths. A ventral cystotomy should be performed in all cases. Dorsal cystotomies have no advantages in terms of risk of urine leakage or adhesion formation and they provide less access and a greater risk of iatrogenic damage to the trigone or the bladder innervation. A stab incision in made into the bladder lumen with a No. 11 scalpel blade usually on midline avoiding any major vessels. If a sterile urinary catheter is not already in place then urine may be suctioned by placement of the suction tip through the stab incision. Avoid direct contact with the mucosa to prevent swelling. The incision is lengthened with sharp cutting scissors. The bladder is very vascular

(especially if there is cystitis present) and bleeds significantly. Try and limit use of electrocautery to significant point bleeders. Mild bleeding can be controlled with pressure from a moistened swab and but it will ultimately cease during closure. Stay sutures can be placed on each side of the incision to facilitate atraumatic retraction and access into the bladder lumen. Forceps or a spoon curette (in the case of heavy 'slit' urolithiasis) may be used to remove uroliths. It is vitally important to flush the urethra in both normograde and retrograde directions to ensure that the urethra is patent and ALL uroliths have been retrieved. It is common for some uroliths to migrate down into the bladder neck and pelvic urethra out of direct visualisation. The bladder lumen should be flushed thoroughly. Samples of the uroliths are submitted for stone analysis and culture. A small bladder wall biopsy can also be collected in cases with suspect infection but a negative urine culture as this has greater sensitivity. The abdominal cavity should be lavaged with warm saline and gloves instruments changed if there was a bacterial cystitis present.

Closure

A one-layer, simple continuous, appositional suture pattern is suitable in most cases. One should ensure that the submucosa is incorporated and attempt to avoid penetrating the mucosa. Polydioxanone with a swaged taper point needle is my preferred suture and 3/0-4/0 is usually suitable depending on patient size and bladder wall thickness. If there is concern about urine leakage a second inverting layer (Cushing or Lembert) may be placed. It is a good idea to wrap the bladder in omentum.

It can be beneficial to leave a urinary catheter in place post-operatively. This has several advantages:

- It prevents bladder distension and excessive tension on the cystotomy closure
- It may help reduce stricture formation at the site of urethral obstruction
- It allows measurement of urine output (should be >1mg/kg/hr)

These potential advantages need to be weighed up against the inherent risk of an ascending urinary tract infection and a decision made based on individual patient risk factors.

Video-assisted cystoscopy

This is a relatively new and minimally invasive way to remove cystic uroliths and avoids a coeliotomy. Having flushed or retropulsed the uroliths from the urethra back into the bladder the patient is positioned in dorsal recumbency. A small (~1-2 cm) preputial incision is made through the skin and linea alba. Baby gelpis can be used to maintain retraction. The bladder is located and temporarily pexied to the linea. A purse string in inserted into the wall of the bladder and a stab incision is made into the centre of it. A lapraroscope cannula and scope are placed into the bladder and the lumen is thoroughly explored. A urethral catheter is placed and saline is flushed into the bladder under pressure. The bladder distends as the pressure increases. When the scope is removed the uroliths are effectively flushed out of the laparoscopic cannula and collected. Larger uroliths that do not fit through the cannula can be manually lifted out using forceps. The bladder is thoroughly flushed and inspected with the scope to ensure all uroliths have been retrieved. The cannula is removed and the mini-cystotomy is closed with one or two interrupted sutures. The pexy sutures are released and the midline wound closed routinely. Patient's recover quickly from this procedure due to its minimally invasive and rapid nature.

Prescrotal urethrotomy (dog)

For those exceptional cases in which uroliths have caused an obstruction at the base of the os penis but attempts at retrograde urohydropulsion have been unsuccessful, then a urethrotomy may be considered as a salvage procedure. It is important to remember that there are likely additional uroliths in the urinary bladder and a concurrent cystotomy will be required. Occasionally urethrotomy may be required to allow biopsy of obstructive or neoplastic lesions in the urethra.

Relevant anatomy

A prescrotal urethrotomy between the base of the os penis and scrotum is usually performed because uroliths commonly obstruct at the entrance to the os penis, and the urethra is surrounded by less cavernous tissue at this level. The urethra is found on midline deep to the subcutaneous tissue. The paired retractor penile muscles lie on its ventral surface and the paired white corpora cavernosa flank either side of it. The urethra is ~ 4 mm wide in male dogs and has a purple colour as it is encircled by a thin rim of corpous spongiosum.

Approach

The patient is positioned in dorsal recumbency with the pelvic limbs extended caudally. Care should be taken to avoid trauma to the delicate scrotal skin when clipping and preparing the pre-scrotal area. The caudal abdomen should also be prepared to allow for a cystotomy if necessary. The prepuce is flushed with a dilute antiseptic and draped into the surgical site to allow access for catheterisation.

Technique

A catheter is passed into urethra to help identify the level of the obstruction. A 2-3 cm skin incision is made on the ventral midline just caudal to the os penis. The subcutaneous tissue is dissected and the penis is identified. The retractor penis muscle is retracted to one side to expose the purple urethra. The urethra is incised on its midline just caudal to the obstruction using a number 15 scalpel blade. Profuse haemorrhage normally occurs as the vascular corpus spongiosum is incised. This can normally be controlled with gentle pressure. The incision can be extended with the scalpel or iris scissors. Mosquito forceps are used to gentle grasp and remove obstructing uroliths. Care should be taken to avoid urethral trauma. The catheter should be advanced all the way into the bladder to ensure the complete urethra is patent. A catheter should then be passed from the urethrotomy in a normograde and retrograde direction and the urethra vigorously flushed with sterile saline. A cystotomy may follow to remove additional uroliths from the bladder as required.

Closure

The urethrotomy may be left open to heal by second intention or sutured without any difference in healing expected. When left to heal by second intention there can be significant ongoing haemorrhage (especially after urination) and there is potential for some urine scald (petroleum jelly should be applied to the skin around the wound to minimise this). It is my preference to perform a primary closure. Fine (4/0-5/0) absorbable material should be used. An interrupted or continuous pattern are suitable. The latter may control haemorrhage better. The urethra may be closed over a urethral catheter to avoid taking bites of the dorsal wall and compromising its lumen but it is NOT left in place after the procedure as it would promote stricture formation. The subcutaneous tissue and skin may be closed routinely. Performed correctly, stricture is a rare complication and more likely to be secondary to trauma caused by the obstructing urolith than the procedure itself.

Perineal urethrotomy (dog)

This is an uncommonly performed procedure in dogs. In rare instances it may be required to; remove uroliths obstructing the membranous urethra at the ischial arch, biopsy neoplastic or inflammatory lesions at the same level, or to perform rigid cystoscopy in male dogs. The technique is the same as that described fro prescrotal urethrotomy with the exceptions that there is often greater haemorrhage due to the urethra being encircled by a greater amount of the corpous spongiosum and that the wound needs to be closed to control haemorrhage and prevent urine leakage into the subcutaneous tissues.

Urethrostomy

Urethrostomy is a salvage procedure to create a new urethral opening and permanently bypass lesions in the distal urethra. The indications include; recurrent obstruction due to urolithiasis / FLUTD that cannot be managed medically, severe urethral trauma, stricture or neoplasia. In male dogs it may be performed in a pre-scrotal, scrotal, perineal or pre-pubic location. The scrotal location is preferred because the urethra is widest and lies superficially in this location; however, it does require concurrent castration in entire animals. The other locations should be avoided due to this high incidence of complications.

In the male cat urethrostomy may be performed at the perineal, trans-pelvic (sub-pubic or trans-ischial) or pre-pubic locations. A perineal urethrostomy is preferred but a trans-ischial is a useful technique for those cats with more proximal obstructions or in those requiring revision of a failed perineal urethrostomy. Similar to the situation in dogs, pre-pubic urethrostomy in the cat carries a high risk of complications (stricture, incontinence, ascending UTI's and urine scald) and should be avoided in all but the exceptional cases.

Fortunately due to improved understanding of the pathogenesis of urolithiasis and FLUTD, significant improvements have been made in the medical management of these conditions making urethrostomy an uncommon procedure.

Scrotal urethrostomy (dog)

Relevant anatomy

The testes and scrotum (in intact male dogs) or the scrotal remnant (in castrated dogs) overlie the urethra at this point. Otherwise the anatomy is similar to that described in the pre-scrotal location with the retractor penis muscle over lying the urethra with is flanked on each

side by the white corpora cavernosum and encircled by a thin layer of corpus spongiosum. The urethra is widest at this level and surrounded by the least amount of vascular spongiosum making it the optimal site for urethrostomy in male dogs. This helps minimise the risk of the two main complications; stricture and haemorrhage.

Approach

The patient is positioned in dorsal recumbency with the pelvic limbs extended caudally. Care should be taken to avoid trauma to the delicate scrotal skin when clipping and preparing the pre-scrotal area. The prepuce is flushed with a dilute antiseptic to allow it to be included into the surgical field. Ideally a sterile catheter is placed to facilitate identification of the urethral midline during surgery.

Technique

An elliptical incision is made around the base of the scrotum (or scrotal remnant). In entire male dogs a castration and scrotal ablation should be performed in a routine manner. Care should be taken to ensure that the incision is not excessively wide to prevent tension during closure. The subcutaneous tissue is dissected to expose the urethra. The retractor penile muscle is dissected from the urethra and retracted laterally. A Gelpi retractor is useful to maintain exposure. Several interrupted absorbable sutures may be placed between the penile tunic and subcutaneous tissues to keep that urethra in a superficial position. An incision is made into the urethra on midline using a No. 15 or 11 blade. It is useful to incise over a urethral catheter to maintain a midline position. The incision can be extended with a scalpel or iris scissors. The most common error is to make the urethral opening excessively small which will promote STRICTURE formation. The incision needs to 5-8 times the urethral width. For most canine patients this will equate to 3-4 cm in length. The incision often looks excessively long for the surgeon inexperienced with this technique but it is surprising just how much contraction occurs in the post-operative period. Haemorrhage is usually brisk but can be controlled with gentle pressure. Use of electrocautery on the urethral mucosa should be avoided.

Closure

The mucosa is sutured to the skin using an interrupted or continuous pattern. Some surgeons recommend a use of non absorbable material citing variable absorption of absorbable materials but I routinely use PDS without issue and this avoids the requirement for suture removal (which often requires sedation). There is some evidence that a continuous pattern may reduce the amount of post-operative haemorrhage compared to interrupted patterns but both are sufficient and correct placement and apposition of the mucosa to the skin is more

important than pattern. It is best to pre-place four sutures at each corner of the stoma, and then fill the spaces in between. It is essential to take a full thickness bite of the mucosa but only include the dermis and epidermis of the skin for perfect apposition. This will limit haemorrhage from the underlying spongiosum. It is a good idea to cut the ear closest to the mucosa shorter than the one on the skin edge to minimise irritation. The subcutaneous tissue and skin at either end of the new stoma can be sutured routinely.

Post-operative care

It is essential that the patient wears an Elizabethan collar over the post-operative period to prevent self-trauma of the wound which would promote stricture formation. Good analgesia is important. Patients typically have periods of intermittent brisk haemorrhage after this procedure which is typically associated with urination or excitement. This can last 4-7 days and needs to be discussed with owners beforehand. This can be difficult for owners to manage at home and it is often best to hospitalise these patients until that resolves. Urine scald is a possible complication. Vaseline can be applied to the skin around the stoma (but not on the wound) if there is evidence of skin irritation developing.

Perineal urethrostomy - PU (cat)

Relevant anatomy

Important anatomical landmarks include the ventral ligament of the penis and ischiocavernous muscles which attach the penis ventrally to the pubis and laterally to the ischium respectively. It will be important that these are identified and sectioned to release the penis sufficiently so that a tension free closure is performed. The bulbourethral glands are a very important landmark. The penis should be dissected sufficiently so that these lie at the skin surface without retracting into the pelvic canal. This suggests that there will not be excessive tension at the new stoma which would predispose to dehiscence or stricture. The retractor penile muscle lies on the dorsal surface of the penis and needs to be removed. In castrated male cats this will be significantly atrophied. Dorsal dissection should be minimised to preserve innervation.

Approach

PU is typically performed with the patient positioned in sternal recumbency with the pelvic limbs hanging over the end of the operating table and the tail elevated. It is important to pad the end of the table. A purse string suture should be placed in the anus to prevent faecal contamination of the surgical site. An approach with the patient in dorsal recumbency has

been described for those cases that require a concurrent cystotomy (rare) to avoid having to re-position the patient. The procedure is a little more challenging in this position.

Technique

An elliptical incision is made around the scrotum and prepuce. Entire male cats are castrated through this incision. A mixture of sharp and blunt dissection is continued around the penis to free it from its attachments. Dorsal dissection is limited to prevent iatrogenic damage to the innervation. Dissection is continued ventrally. The ventral ligament of the penis is transected to free the penis from the pubis. The ischiocavernosus muscles are either transected or elevated from the ischium on each side of the penis. Haemorrhage can be profuse from these muscles and the use of electrocautery is helpful. The bulbourethral glands need to be identified and dissection is continued to free the penis sufficiently so that these rest at the level of the skin without retracting into the pelvis. The retractor penile muscle (or remnants of it in castrated males) is removed. The urethra is opened on its dorsal midline distally and opened in a proximal direction with iris scissors to the level of the bulbourethral glands. At this level the urethra should be 4-5 mm in diameter. To ensure that it is wide enough the tips of mosquito forceps are inserted and should easily pass to the level of the hinge. Interrupted dorsal sutures (10, 12 and 2 O'clock positions) are pre-placed to adequately spatulate the opening. Each side is then sutured for a distance of ~1.5 cm. I prefer to place interrupted non-absorbable sutures alternating from side to site. The ear closest to the mucosal surface can be cut short to prevent irritation and the one closest to the skin left long to facilitate removal. A mattress suture is placed through the cavernous tissue ventrally and the penis is amputated before closing the ventral corners of the stoma.

Post-operative care

Patients need an Elizabethan collar and good analgesia to prevent self-trauma to the wound. It is best to put shredded paper or foam into the tray instead of gravel to prevent it sticking to the wound. The stoma does not need to be cleaned and minor blood clots do not require removal if there is a normal stream of urine. There is a high incidence of bacterial cystitis (17-57%) in cats following PU. However this was not reported in normal cats that had a PU performed therefore suggesting that this was a result of the underlying uropathy, emphasising the need for ongoing medical management of this condition.

Complications

Reported complications include; stricture, ascending UTI, recurrence of clinical signs, urine extravasation and dehiscence although most cases have satisfactory long-term outcome. The most significant complication is stricture formation. This is usually caused by poor adherence to the technical aspects of the procedure. Insufficient dissection to adequately free the urethra results on retraction cranially and tension on the stoma. This causes wound contraction and subsequent stricture. Poor apposition of the mucosa and skin can result in urine leakage into the subcutaneous tissues resulting in inflammation and stricture formation.

In most cases these patients can have a revision surgery which allows the urethra to be more thoroughly dissected in a cranial direction and freed from its pelvic attachments and a new stoma created. If not then a trans-ischial or sub-pubic urethrostomy may be performed as a salvage procedure.

Trans-pelvic / Trans-ischial urethrostomy (cat)

Relevant anatomy

This technique creates a new stoma between the pelvic urethra just cranial to the bubourethral glands and the underlying skin by way of an ischial ostectomy. The adductor muscles (gracilis and external obturator) have to be elevated of the caudoventral pubic symphysis to allow the ostectomy to be completed.

Approach

The patient is positioned in dorsal recumbency with the limbs extended cranially (to provide access to the perineum). The ventral abdomen, pelvic and perineal regions are clipped and aseptically prepared. A purse string suture is placed in the anus.

Technique

In the original description (Bernard and Viguier, 2004), a mini coeliotomy and cystotomy were performed to allow normograde placement of a urinary catheter from the bladder into the proximal urethra to the obstruction site. This helped identify the urethra during surgery but may not be strictly necessary and certainly not in those cases that are able to be catheterised in a retrograde manner. The scrotum and prepuce are excised with an elliptical incision which extends cranially to the cranial margin of the pubis. The penis is extended caudally and its ventral surface is denuded of soft tissue. The bulbourethral glands are identified. The

adductor muscles are elevated off their midline pubic attachments to expose the caudal pubis and ischium. Rongeurs are then used to remove a section of the ischium (~ 10mm x 15 mm) from a caudal to cranial direction in a piecemeal fashion. The pelvic urethra (cranial to the bulbourethral glands) is exposed through this ischial ‘window‘. The urethra is opened with a longitudinal incision on its ventral midline from the bulbourethral glands to the cranial edge of the ostectomy. The urethral mucosa is then sutured to the overlying skin without tension using non absorbable material in an interrupted pattern. The penis distal to the bulbourethral glands is amputated and the wound cranial and caudal to the new stoma is closed routinely. As per the original description the catheter was removed and the cystotomy closed routinely.

Post-operative care

This is similar to that described for PU.

Complications

Complications would be expected to be similar to those reported for PU but this technique has only been reported in a small case series with 11 cats to date. It produced an acceptable stoma in all cases. One case developed a stricture but did not have associated clinical signs. Two other cats had an episode of idiopathic LUT disease which responded to medical therapy.

This technique has been described in one dog for the management of a non- neoplastic urethral lesion just distal to the ischial arch with a positive outcome (Liehman et al. 2010).

Surgical management of ureteral obstruction

Ureteral obstruction

Partial or complete ureteral obstruction in dogs and cats may occur secondary to urolithiasis, ureteral ectopia, ureteral neoplasia, ureterocoeles and iatrogenic ligation during OVH. All of these would be considered rare in small animals except uretrolithiasis in cats. The ureteroliths may form unilaterally or bilaterally, cause complete or partial obstruction and are commonly composed of Calcium oxalate. Given this variation in severity one can expect a range of presenting clinical signs. Some cats will present with mild clinical signs of inappetence, retroperitoneal discomfort and normal urination (especially if unilateral) whereas others may present with profound azotemia, dehydration, depression and severe metabolic derangements when obstructed bilaterally or when unilaterally obstructed with concurrent renal insufficiency. Many of these patients have underlying chronic renal insufficiency and the obstruction pushes them into failure.

Just as with urethral obstruction the initial priority is stabilisation of the patient from a metabolic and cardiovascular perspective. Establishing diuresis is important. Repeated biochemical and electrolyte monitoring is essential so that response to medical management can be assessed and adjustments to treatment made as necessary. One of the greatest challenges with these cases is to determine what proportion of the azotemia is secondary to the obstruction and how much is due to underlying renal failure. It is often impossible to determine this pre-operatively especially in bilateral cases. However, if a patient has unilateral obstruction and is profoundly azotemic, it suggests poor compensation from the contra-lateral kidney and concurrent renal failure is likely. An improvement in the degree of azotemia following diuresis is promising but it is important to inform clients that we cannot predict the degree of recovery following treatment.

Diagnosis is made on the basis of signalment, clinical signs and imaging. Because radio-opaque Calcium oxalate uroliths are most common in cats, plain radiography is valuable and can be definitive. Abdominal ultrasound also has a role and may be performed conscious whilst the patient is stabilised. The ureterolith may be visualised (with accompanying acoustic shadow) but more importantly the degree and level of obstruction can be estimated. It is possible to assess for secondary pylectasia or hydronephrosis. If there is no ureterolith detected in the face of obstruction the ureter may be blocked due to a stricture at the site of a previous obstruction or blood clots. The contra-lateral kidney may be assessed in unilateral obstructions and if shrunken may suggest chronic renal failure.

Medical therapy can be appropriate in some patients particularly those with acute obstructions. Diuresis is used to stimulate ureteral flow and dilation with the hope that the ureterolith(s) is propelled into the bladder. It could then be removed more simply by cystotomy. Other drugs have been used to stimulate ureteral dilatation; the most promising results have been with the tricyclic antidepressant amitriptyline. It is important imaging is repeated frequently to monitor progress. Failure to progress worsens renal damage secondary to the back-pressure from obstruction and surgery may be indicated. Reported survival times are better with surgery compared to medical management but surgery in not without risk.

The normal feline ureter has been estimated to have a diameter of 0.4mm which makes ureteral surgery extremely challenging. At surgery both ureters need to be inspected. There is usually evidence of megaureter proximal to the obstruction. Occasionally the ureterolith can be palpated. It is most common for the obstruction to occur in the left ureter and in the proximal one third. Following dissection and isolation of the involved segment a small ureterotomy incision is made over the ureterolith(s), which are removed. Avoid trauma to the ureteral artery. A 4/0 piece of suture material is used to test the patency of the ureter in both directions. The ureter needs to then be sutured in an interrupted or continuous pattern with 9/0 nylon. A short piece of 4/0 suture material may be used as a temporary stent to avoid catching the far wall during closure and removed just before the last suture is tied. Ophthalmic instruments and magnification are essential. Possible complications include urinary leakage, dehiscence and stricture formation. Some clinicians advocate the use of a temporary nephrostomy tube to divert urine during the healing process.

Because of the inherent challenges associated with conventional surgery on such a small structure, the use of double pigtail catheters have gained favour but are only available in specialist centres. These are placed through a cystotomy or via the renal pelvis using fluoroscopic guidance. No attempt is made to remove the ureterolith - instead these act as a stent allowing urine to flow around them. These can be placed bilaterally. These are well tolerated and avoid the potential complications associated with surgery and stricture is of less concern because the stent will remain in situ long-term. Reported complications include suboptimal stent placement, infection and stranguria which is usually transient.

Disease Conditions

Feline lower urinary tract disease (FLUTD)

FLUTD is an umbrella term used to describe a group of symptoms commonly displayed by cats with lower urinary tract disease. These symptoms include:

- Pollakiuria
- Dysuria
- Stranguria (possibly non productive)
- Hematuria
- Periuria
- Vocalisation
- Penile or vulval licking

NB: Some owners will misinterpret these signs as constipation or tenesmus.

There are several potential causes of cystitis / urethritis that can lead to this syndrome. These include:

- Urolithiasis
- Bacterial urinary tract infections
- Neoplasia
- Anatomical abnormalities (e.g. persistent urachus)
- Idiopathic i.e. Feline Idiopathic cystitis (FIC)

It was originally considered that urolithiasis was a common cause of this syndrome and it fact it was known as feline urological syndrome. It is now known that most cats that are fed a dry food will have crystalluria and not all cats with crystals will go on to form uroliths and obstruct. Therefore if one finds crystals in the urine of a cat with FLUTD it is important not to assume that this is the primary cause. Of course in some instances uroliths may form and cause inflammation of the bladder and urethra and even lead to obstruction. Struvite (Magnesium ammonium phosphate) and Calcium oxalate stones are most common. Struvite are commonly associated with infection and acidifying diets designed to prevent Struvite have seen a rise in Oxalate stones in recent years.

Bacterial urinary tract infections are relatively uncommon in cats - particularly young to middle aged cats. The flushing effect of bladder emptying and the high concentration of cat urine are natural inhibitors. Therefore antibiotics should NOT be used as part of the first line treatment of cats present with FLUTD. In older cats or those cats with systemic diseases (e.g. renal insufficiency or diabetes mellitus) that produce more dilute urine, the rate of bacterial infection is higher and may more likely be involved. However, urine should be collected by cystocentesis for microscopic examination and culture prior to antibiotic therapy being instituted.

Cats may develop neoplastic lesions in the bladder or urethra and be responsible for these signs. The most common is the transitional cell carcinoma (TCC) which has a predilection site for the trigonal area and bladder neck. Physical obstruction and peri-tumour inflammation may lead to the clinical signs. It typically affects older patients.

Undoubtedly the most common cause of FLUTD is FIC. The cause of FIC is unknown but it likely involves aberrant interactions between the neurological supply to the bladder and GAG layer on the mucosal surface of the bladder. It mainly affects young to middle aged, overweight cats living in multi-cat households. Patients typically cope poorly with environmental stress. It is generally a self-limiting disease that resolves within 1-3 days regardless whether treatment is administered or not. The proteinaceous matrix from the bladder mucosa may mix with crystals and form mucosal plugs that can lead to obstruction. This typically occurs in the narrow penile urethra of male cats and they may subsequently present with complete obstruction requiring emergency management (as described previously). Urethral spasm may be a concurrent complicating problem or occur alone. Unfortunately this condition is recurrent in nature and as many as two thirds of patients will have a recurrent episode within 12 months.

Approach to a case of FLUTD

The first thing to establish is whether the patient is obstructed or not. For obvious reasons male cats are more likely to obstruct. Physical examination would usually detect unproductive stranguria, a tense, over-distended and painful bladder and possibly systemic signs (bradycardia, dullness, depression, dehydration etc.) depending on the extent and duration of obstruction. In this instance it is vital to immediately obtain a minimal biochemical database (PCV, TP, electrolytes, urea, creatinine), institute intravenous fluid therapy and decompress the bladder. Once stabilised the patient can be sedated or anaesthetised for catheterisation. Anaesthesia usually provides better analgesia and urethral relaxation thus improving success

rate. (See earlier section for more detail on practical technique). The bladder should be flushed with sterile saline until clear fluid is returned. If catheterisation is easily accomplished and there is a good stream of urine with bladder expression following the procedure, I normally avoid leaving a catheter in place as they can incite inflammation and urethral spasm that is difficult to differentiate from the disease process. If a patient is difficult to catheterise, then I leave a soft silicon catheter in place for a few days. It is important to keep the patient hospitalised and monitor urination for at least 24 hours after catheter removal.

For those patients that are not obstructed at presentation (and following successful unblocking of an obstructed case) institution of medical therapy may be considered. This generally involves;

- Good analgesia e.g. buprenorphine
- Anti-inflammatories e.g. meloxicam if no contra-indications such as renal insufficiency exist
- Antispasmodics e.g Prazosin or phenoxybenzamine for smooth muscle relaxation and dantrolene for skeletal muscle. Note these are not licensed for cats in the U.K.
- Remember antibiotics are avoided unless there is a specific UTI diagnosed. This is rarely the case. When using them base the choice on culture and sensitivity results and avoid using fluoroquinolones or third generation cephalosporins as first line drugs to help prevent selection for resistance. Do NOT use antibiotics whilst a urinary catheter is in place is possible. Culture the tip on removal and treat then.

As mentioned above the vast majority of cases are self-limiting in nature and therefore improvement could be mistakenly attributed to medical therapy. Treatment decisions may need to be made on an individual patient basis. It may be reasonable to avoid specific treatment in a patient that presents with a first episode and is unobstructed or easily unblocked. A more aggressive approach with therapy may be taken in those cases which are more severely affected, or have several recurrent episodes over a short period of time.

Prevention of future episodes is important in all cases and involves:

- Encourage increased water intake e.g. drinking fountains, ice cubes made of stock, wet food. Aim to get the urine S.G. < 1.035
- Stress reduction e.g. hiding places, adequate litter trays, scratching posts, minimise conflict over food etc., clean litter trays regularly, feline facial pheromones, xylkene
- Supplementation with GAG's. Solid evidence to support use lacking.

Where do specific diagnostic investigations fit in?

It is reasonable to institute symptomatic therapy the first time a case presents with FLUTD especially if the clinical signs are mild, the patient is young to middle aged and not obstructed (of course in the obstructed case baseline bloods will be needed for stabilisation).

Specific tests may be performed in patients presenting with recurrent disease, a prolonged bout that is not responding to medical therapy, severe signs or when contemplating surgical treatment. Tests may include some or all of the following:

- Urinalysis: Urine is best collected by cystocentesis for microscopic sediment exam and bacterial culture. This can determine if there are signs of inflammation/pyuria and rule in or out an infection. This is particularly important in older patients or those with systemic disease that reduces urine concentration as these predispose to UTI.
- Radiography: Plain radiography is useful for detecting radio-opaque uroliths and contrast (negative or positive) may be useful to delineate radiolucent uroliths and bladder/urethral masses. Anatomical abnormalities or bladder wall thickening secondary to cystitis may also be seen.
- Biopsy: Collected by suction through a catheter, cystoscopy or open biopsy. Can be useful to diagnose neoplasia in the presence of a mass.

NB: FIC is a diagnosis made by EXCLUSION and can therefore only be made when other causes of FLUTD have been ruled out with the above investigations.

Where does surgery fit in?

Due to improved understanding of the causes and pathogenesis of FLUTD and improved medical treatments available perineal urethrostomies (PU's) are less frequently required. However this surgery still has a place. The main indication for a PU is the male cat with an obstruction in the penile urethra that cannot be relieved, the patient with frequent episodes of obstruction (~ every few weeks) in the face of good medical therapy or the prolonged bout (several days) that is failing to respond to medical therapy and catheterisation. It is important to do a full diagnostic work-up as outlined above before surgery to rule out any primary cause or obstruction of the pelvic urethra or bladder as a PU will not help these.

It is important that the surgery is carried out with attention paid to the technical aspects of the procedure. Failure to do so could result in serious complications and the need for a challenging revision surgery (see previous for description of the technique). Client education is important as patients may still shown signs of FLUTD and will require ongoing preventative measures, although complete obstruction will be prevented.

If a specific aetiological diagnosis is made during investigations (e.g. cystic uroliths, bladder mass) then there may be a clear indication for surgery (e.g. cystotomy to remove uroliths or biospy/remove mass as appropriate).

Urolithiasis

Urolithiasis is a common problem in both dogs and cats. It is outside the scope of these notes to cover all the information pertaining to this subject. The reader is referred to specific medical texts for further information. This section will cover the principles underlying urolith formation, pathogenesis and treatment of the most common types.

Urolith formation

Various theories exist regarding the mechanisms of urolith formation. The most commonly accepted one relates to precipitation. Dogs normally have urine that is supersaturated with various salts. Increasing concentration of these salts (relatively reduced water content in urine) may allow them to cross this threshold and come out of solution forming crystals. Crystals can amalgamate and form uroliths. This is the most important mechanism hence the reason why increasing water content is an essential part of the long-term management in most patients. Other factors may also play a role in this including urinary retention, a suitable pH for crystallisation or the presence of a nidus. Another theory is that the urine of dogs (or cats) predisposed to urolithiasis may contain crystallisation promoters. These are proteins (potentially arising from inflammation) that act as a scaffold to initial crystals to bind to. Likewise normal urine normally contains crystallisation inhibitors and it has been suggested that patients with urolithiasis may have reduced quantities.

Specific urolith types

The most common urolith encountered is Struvite (Magnesium ammonium phosphate) which is typically associated with urinary tract infection in dogs followed closely by Calcium oxalate. Prevalence of the latter has been increasing over the last decade as many diets were acidified to reduce the risk of Struvite formation. Urate uroliths are encountered commonly in Dalmatians (and English Bull Terriers) due to defective uric acid metabolism. They are also seen in patients with liver disease most commonly congenital portosystemic shunts. Cystine, Silicate and mixed uroliths are encountered occasionally but will not be discussed in detail here.

Calcium oxalate

These uroliths occur in a monohydrate (most common) or dihydrate form. They may contain small amounts of phosphate. Middle aged to older male dogs are typically affected and breeds such as the miniature schnauzer, miniature poodle, Yorkshire terrier, Lhasa apso and Shih Tzs' are over-represented. They typically occur in patients with transient post-prandial hyercalcaemia. Increased intestinal absorption of calcuim increases serum levels. This in turn suppresses parathyroid hormone which increases renal excretion of calcium promoting hypercalcuric.state. In rare cases, impaired renal resorption may cause calcium to leak into the urine. Any cause of hypercalcaemia may raise urine excretion levels such as primary hyperparathyroidism, lymphoma, hypercalcaemia of malignancy or Vitamin D toxicosis as well as hyperadrenocorticism (due to the effects of glucocorticoids). High dietary oxalate levels may also predispose, as may reduced intestinal flora (specifically Oxalobacter formigenes) that is responsible for degradation of oxalate at gut level. Approximately 1/3-1/2 are associated with urinary tract infection in dogs. An acidic urine pH favours calcium oxalate formation.

Calcium oxalate stones are radio-opaque which facilitates diagnosis. Unfortunately they are not amenable to dissolution thus when diagnosed need to be removed by urohydropulsion or surgery. Following removal they should be submitted for analyisis to confirm the diagnosis and allow long-term preventative measures to be put in place. This is particularly important with this urolith type because they have a tendency to recur (recurrence rate of 50% within three years has been reported). All patients should be investigated to rule out hypercalcaemia, hyeradrenocorticism and metabolic acidosis. If a specific disease is diagnosed then treatment should ensue and if underlying cause is ruled out then a preventative diet should be instituted.

There are various suitable diets available commercially. Additionally as with all uroliths, water intake should be increased. Simply changing to a wet/canned diet will help significantly. One should aim to achieve a pH of < 1.020 and assess response by monitoring this regularly. Avoid strong acidifying diets and renal diets. The later are low in phosphate which is useful for binding calcium in the intestine. Target urine pH is controversial but generally within a range of 5.5-7.5. Specific drug therapy can be considered in cases continuing to produce a crystalluria on follow up sediment exam despite increased water intake and diet alteration. Thiazide diuretics reduce urinary excretion of calcium (an effect that is enhanced when combined with Hill's u/d diet). Potassium citrate administration may be useful as citrate forms a soluble complex with calcium and mildly alkalises the urine increasing calcium oxalate solubility.

Struvite

This is the most common urolith encountered in dogs. In ~95% of patients it is associated with a urinary tract infection and therefore young-middle aged female dogs are more commonly affected compared to males. If uroliths are seen in patient < 1 year of age then be suspicious of struvite because of the association with infection. Miniature schnauzers, Bichon Frises and Cocker spaniels are over-represented.

Urease producing bacteria (Staphylococcus, Klebsiella, proteus and pseudomonas sp.) are an important part of the pathogenesis. These enzymes split urea into ammonia and CO2. Hydrolysis of ammonia forms ammonium ions which alkalinise the urine (decreasing the solubility of struvite) and become available to bind with the magnesium and phosphate ions. Bacterial cystitis and the toxic effect of ammonia on the mucosal lining of the bladder produce inflammatory, organic debris that can act as a nidus for crystal formation. Bacteria become trapped within layers of the urolith. Struvite urolths can form in the cat in the absence of infection.

Struvite uroliths are amenable to medical dissolution. This process is SLOW (3-4 months) and therefore NOT suitable for stones obstructing the urethra. One has to weigh up the risks and costs of medical dissolution versus surgical removal. There is a risk for urethral obstruction in male dogs as stones may become small enough to pass down the urethra and owners should be warned about this complication so that appropriate attention can be sought. In patients undergoing medical dissolution, antibiotics are required throughout as bacteria are constantly liberated from the layers of the stone. Repeat radiographs, urinalysis and culture from cystocentesis samples is required to assess response to therapy. Culture should become negative within one week and the urine pH should be < 7.0. Treatment should continue for one month following radiographic disappearance of the uroliths.

Caluloytic diets e.g. Hill's s/d may also be used to dissolve the uroliths. This diet has a restricted protein level and patients will likely develop a low urea and or albumin whilst on therapy. Due to the low protein this diet is not suitable for long-term use and a suitable maintenance diet (e.g. Hill's c/d) should be sought after dissolution. Long-term prevention focuses on detecting any conditions that predispose to recurrent UTI's and managing them appropriately.

Urate

These are composed of ammonium acid urate which is derived from the degradation and metabolism of dietary nucleic acids and endogenous purine ribonucleotides. Approximately 60% or urate stones occur in the Dalmatian with males over-represented. This is because of impaired hepatic metabolism of uric acid into the more soluble allantoin. Additionally Dalmatians may have recued ability to resorb uric acid in the proximal tubule further compounding the problem. In the remaining cases are usually secondary to hepatic disease, mainly PSS. Detection of this stone type in a non Dalmatian (or English bull terrier) breed should prompt further investigations into possible hepatic disease. These uroliths are radio-lucent therefore requiring ultrasound or positive/double contrast urethrocystography to diagnose. They can be associated with secondary UTI's.

Medical dissolution of urate stones is possible. Alkalinisation of the urine will increase the solubility of uric acid and decrease ammonium ion production. A diet low in protein and nucleic acids e.g. Hill's u/d is required to reduce urea formation. Drugs such as Potassium citrate (to alkalinise the urine) or allopurinol (reduces the formation of uric acid) may be beneficial in a patient failing to respond to dietary therapy alone, although long-term use of the latter may predispose to xanthine stone formation. Increasing the water intake and controlling secondary infections is imperative.

In patients with portosystemic shunts therapy should be targeted at managing this underlying cause (e.g. shunt ligation). I prefer to remove the uroliths via cystotomy at the time of shunt surgery and submit them for analysis and culture.

Silicate

These uroliths form because of a high intake of dietary silicates. There is a significant pre-disposition for male dogs particularly the GSD and OESD. They tend to form in acidic urine. They are typically jack shaped. They are reasonably radio-opaque making them visible on plain radiographs. They are not amenable to dissolution therefore require surgical removal.

Long-term prevention focuses on increasing water intake, limiting dietary silicate and alkalinizing the urine. U/d diet may assist with the latter two aims.

Cysteine

Cysteine is normally freely filtered by the glomeruli and then resorbed in the proximal tubule. These uroliths form secondary to an inherited disorder in renal tubular transport. Many breeds are affected including Bassett Hounds, dachshunds, Tibetan spaniels, English Bulldogs,

Yorkshire terriers Newfoundlands, Labradors and Rottweilers. Males are over-represented. Cysteine is relatively insoluble in acid and form mainly in acidic urine.

These uroliths are radiolucent. Dissolution is possible. Hill's u/d is suitable as it is low in protein and alkalinises the urine. Specific alkalinizers such as potassium cirate are rarely required. D-penicillamine binds to cysteine dramatically increasing its solubility and may be used if dietary therapy fails. Caution is required as this rug can interfere with wound healing, cause glomerulonephritis or lymphadenoptahy.

Mixed / Compound uroliths

Mixed uroliths are those which two minerals are mixed together or a single type constitutes less than 70% of the overall composition. Compound uroliths are denoted a central core surrounded by an outer layer of a different urolith type. These are complicated to deal with and specialist opinion should be sought.

Reflex dysnergia

This condition manifests as poor coordination between bladder contraction and urethral relaxation during urination. It is an uncommon condition that affects medium to large breed male dogs. It is primarily a neurological abnormality due to dysfunction within the autonomic ganglia (hypogastric and pelvic ganglia) supplying the bladder and urethra although other neurological lesions may also cause it. It is occasionally seen after urinary tract surgery or after stressful events (e.g. hospitalisation or anaesthesia). We are more frequently recognising this condition in some of our hospitalised patients.

Clinical signs

The symptoms of this condition can be confused with partial urinary tract obstruction. The patient usually displays acute onset of stranguria. The urine stream may be normal initially but may become thin or interrupted during urination with potential for the patient to obstruct completely. It is not uncommon to notice urine dribbling from the urethra after the straining has resolved (because of urethral relaxation). Even if urine is passed there is usually incomplete bladder emptying that can be palpated after.

What are the consequences of this?

Asides from the discomfort and distress of having an overfull bladder these patients are at risk of completely obstructing and developing the metabolic derangements discussed

previously. Additionally incomplete bladder emptying causes urine stasis and stagnation predisposing to the development of a UTI. Most importantly however, if undiagnosed or left untreated these dogs may develop bladder over-distension that disrupts the detrusor muscles leading to long-term bladder atony. This is basically a large, stretched, flaccid bladder that is incapable of functional contraction. The more severe and chronic this is, then the greater the risk of it becoming permanent. These dogs may actually present with paradoxical stranguria and over-flow incontinence.

Diagnosis?

Signalment, careful observation of urination and confirmation of incomplete bladder emptying after urination are strongly suggestive. In comparison to partial urethral obstruction these patients are easily catheterised. It is ultimately a diagnosis made by ruling out physical causes by way of positive contrast retrograde urethrography and or cystoscopy.

Treatment

Therapy will differ depending on whether there is secondary bladder atony or not. In the acute case medical therapy with a smooth muscle relaxant is required. Alpha receptor antagonists such as prazosin or phenoxybenzamine are usually employed. A more rapid response may be seen with the former. There is generally no need for a striated muscle relaxant as voluntary urinary control is intact but some clinicians may add in dantrolene.

NB: THESE DRUGS MAY CAUSE HYPOTENSION SO THERAPY SHOULD BE STARTED AT THE LOWER END OF THE RECOMMENDED DOSE RANGE AND GRADUALLY TITRATED UP IF NECESSARY. THE CLINET NEEDS TO BE WARNED OF THE SIGNS (LETHARGY, WEAKNESS, AND COLLAPSE).

The second aim of treatment is to keep the bladder decompressed and rested to prevent over-distension and atony whilst the drugs take effect. This is important because the alpha antagonists can take several days to take effect. Bladder decompression can be achieved with repeated manual expression (challenging), intermittent catheterisation or placement of a Foley urethral catheter connected to a urinary collection system. These are appropriate for the acute case but assessing the response to therapy can be difficult with a catheter in place and it may need to be removed for a trial and replaced as necessary.

For those patients in which bladder atony is suspected then the period of bladder decompression is often longer. A better option may be to place a cystostomy tube (see previous for description of the technique). The bladder should be kept small by continuous drainage or intermittent aspiration every four hours. This allows one to assess the response

more readily as the bladder can be allowed to fill at certain time points and urination observed. Residual volumes of urine in the bladder can be checked (generally < 0.3 mls/kg) afterwards and use of the cystotomy tube continued if the patient has not fully recovered. It may also be useful to add a parasympathomimetic drug such as bethanecol to stimulate detrusor contraction.

Prognosis

The prognosis is favourable for those patients with acute dysnergia that are diagnosed before bladder atony occurs. Unfortunately once secondary bladder atony occurs, the prognosis becomes guarded with the reversibility depending of the severity and chronicity of detrusor over-stretching. I have seen reflex dysnergia in patients with prostatic neoplasia. Clinical signs are initially attributed to the physical presence of tumour partially obstructing the urethra. However, in some cases the prostatic urethra is patent of retrograde urethrograms and clinical signs are attributed to peri-tumour inflammation causing urethral spasm or a neurological dysfunction of the closely associated autonomic ganglia. Urethral relaxants (in addition to COX 2 inhibitors) can significantly help control clinical signs in some of these patients.

Urinary tract neoplasia

Overall neoplasia of the lower urinary tract is uncommon but there are specific tumours that occur in dogs and cats that may lead to the patient presenting with urinary tract obstruction. These include in relative order of frequency:

- Bladder tumours: Usually transitional cell carcinoma with a predilection site for the trigonal area. Other malignant tumours have been described such as squamous cell carcinoma, leiomyosarcoma, rhabdomyosarcoma, hemangiosarcoma, lymphoma and benign masses such as leiomyoma and bladder polyps.

- Prostatic tumours: Usually carcinomas such as adenocarcinoma and transitional cell with some cases have elements of both on histopathology.

- Urethral tumours: These are rare and it is typically the proximal urethra that is affected with extension of a bladder or prostatic tumour into it. Benign granulomatous urethritis may produce an inflammatory mass lesion that could be mistaken for a tumour.

Most patients that present with neoplasia of the lower genitourinary system are middle aged to older. The WHWT, Scottish terrier and Shetland sheepdog may all be predisposed to bladder neoplasia (TCC). It is important to remember that prostatic neoplasia is more likely in neutered male dogs compared to entire ones and should be suspected in any neutered male with an unexpectedly large prostate. Patients with neoplasia of the lower urinary tract may present with signs similar to other conditions including stranguria, dysuria, haematuria or partial obstruction i.e. poor/interrupted stream of urine. There may be other signs to raise the suspicion of this diagnosis such as lameness from bone metastases, tenesmus from local lymph node metastases, palpable masses on digital rectal examination, weight loss or general malaise. However, some patients may not have any of these signs and therefore further investigations are warranted in any older dog or cat that fails to respond to symptomatic therapy.

Diagnosis

Abdominal ultrasound examination is an excellent modality for the detection of bladder or prostatic neoplasia. The size and extent of the primary tumour can be defined and the local lymph nodes and other abdominal organs assessed for evidence of metastatic spread. Radiography, particularly contrast urethrograms and cystograms have a valuable role to play and can define the degree of urethral involvement/obstruction. Once a mass is visualised, sampling is required to make a definitive diagnosis. Microscopic examination of urine sediment may be useful for the detection of neoplastic cells in some cases although dysplastic cells due to inflammation may appear similar to neoplastic cells making diagnosis challenging. Suction or traumatic biopsy by aspirating the mass through a urethral catheter may be performed under ultrasound guidance and can yield useful samples. Prostatic massage can also be performed similarly with a catheter in place. However, if a prostatic tumour has not invaded the urethra a sample may not be obtained. Percutaneous fine needle or trucut biopsies may be performed as a last resort but this does carry a risk of tumour seeding. In female dogs cystoscopic biopsies may be obtained and in fact this may be done in male dogs via a temporary perineal urethrotomy but the risks of such a technique need to be weighed up against the potential benefits.

Treatment:

Unfortunately most neoplasia of the lower urinary tract is locally aggressive and metastatic in nature. Most bladder TCC's involve the trigone region of the bladder and are thus inoperable and surgery does not improve outcome for dogs with prostatic carcinoma. In the rare instance that a TCC involves the body or apex of the bladder or another tumour type is diagnosed

(with no evidence of metastases), then partial cystectomy my be appropriate combined with adjunctive therapy. SCC affecting the distal urethra may be managed with penile amputation and urethrostomy in select cases.

There are a number of chemotherapeutic options for bladder/urethral TCC. Most clinicians recommend use of a COX2 inhibitor such as piroxicam or meloxicam which has an anti-tumour effect; the mechanism of which is poorly understood. Often there is a role for other chemotherapeutic agents with the platinum drugs or mitoxantrone.

Palliation may be more important for these patients to either perform a urinary diversion technique or relive the physical obstruction. Cystostomy tubes may be placed so that the owner can empty the bladder several times per day. This will ultimately prevent the metabolic consequences and discomfort associated with obstruction and bladder distension. This will likely improve the patients' quality of life and can be combined with chemotherapy. If the patient and owner are coping well, then the tube could be exchanged for a more permanent, low profile cystostomy tube.

Palliative urethral stents are now available in some specialist centres and are placed in a minimally invasive way using fluoroscopic guidance. These are well tolerated and restore urinary flow. Unfortunately a proportion (10-20%) of patients may develop post-operative incontinence and this needs to be discussed with the client beforehand. Other complications include reflex dysnergia and ingrowth of the tumour and obstruction of the stent. Once again stenting may be combined with adjunctive chemotherapy to maximise outcome.

Emergency Management of Urinary Tract Injury

The presence of complete urinary obstruction or traumatic separation of its components (ureter, bladder, urethra) is an emergency, and immediate resolution is required. Signs of post-renal azotaemia will develop within a few hours of urethral obstruction. Urine leakage into retroperitoneal tissues from ureteral or urethral rupture will cause severe tissue injury within 24 hours, with bruising, pain and oedema being evident. Urine leakage into the abdomen is possibly tolerated for 24-48 hrs, but ultimately peritonitis and hypovolaemia will develop. The most life-threatening feature of the azotaemia is hyperkalaemia, which will predispose to bradycardia and cardiac arrhythmia. Potassium levels greater than 7.0 mEq/l are most likely to be associated with cardiac signs, with life-threatening complications increasing with more elevated levels. (Bjorling, 2003).

Correction of most fluid and electrolyte abnormalities can be accomplished by saline diuresis with 0.9% sodium chloride. The rate of infusion will depend on the degree of dehydration and cardiovascular status. In most instances, diuresis at twice maintenance infusion rates is adequate. Determination of effective urine output during the stabilisation period is essential to ensure renal function is adequate. Intermittent catheterisation, or placement of an indwelling catheter will enable quantification of urine production. In the normal animal, urine production of at least 2ml/kg/hr should be seen. If hyperkalaemia is severe, or cardiac complications are present, treatment with insulin and dextrose will enable more rapid correction of electrolyte balance.

Establishing an Excretory Route

For diuresis to be effective, the patient must be able to excrete urinary wastes which requires creation of an alternative route for urination if the “original” route is disrupted by obstruction or injury. With urethral stones, relief of the obstruction may be performed via intermittent cystocentesis, pre-pubic cystostomy, urethral catheterisation or hydropropulsion. Aspects of the individual case will dictate which option is most appropriate, and the clinician must weigh up the potential complications of each technique. If possible, attempts should be made to flush urethral calculi back into the bladder, thus avoiding the need for urethrotomy.

The simplest method is passage of a catheter. In some animals, it may be possible to pass a small diameter catheter about an obstruction. However, this is usually only possible if the obstruction is partial, or caused by multiple small stones. When catheter advancement is prevented, hydropropulsion is a very effective technique to flush calculi and debris back into

the bladder. In cats, vigorous flushing with saline during catheter advancement may facilitate passage of the catheter. Occlusion of the urethral opening during flushing may increase intraluminal pressure and force material back into the bladder. In large dogs, the technique of hydropropulsion can be dramatically enhanced if the pelvic urethra is held occluded by a finger placed in the rectum. With the finger placing firm pressure on the urethra proximal to the obstruction, a well-combined mixture of one part KY jelly and one part saline is infused until the urethra can be felt to distend. The urethral occlusion is quickly released whilst vigorous flushing continues. The combination of lubrication, urethral distension and increased intraluminal pressure is usually successful in relieving even the most stubborn material.

If urethral obstruction cannot be relieved by catheterisation or urohydropropulsion, direct bladder drainage via cystocentesis or prepubic catheterisation is required. Intermittent cystocentesis is usually well tolerated, but care must be taken with cats, particularly when the bladder is extremely distended. Prepubic catheterisation can be useful if a prolonged period of stabilisation is anticipated before corrective surgery is performed. The catheter is introduced into the bladder in a similar manner to cystocentesis. Catheter migration from the bladder is prevented by the clover-leaf shaped tip that is formed when the stylet is removed. Care must be taken to ensure all of the catheter tip is located within the bladder, and that sufficient ‘slack’ is provided before securing the catheter to the skin.

Temporary Urinary Diversion

Tube Cystostomy

Pre-pubic cystostomy is a very useful technique to allow urinary bypass of the urethra following severe trauma or pelvic injury, following prostatic surgery, or as long term management aid in cases of bladder atony or neoplasia. Tube cystostomy diverts urine away from the urethra while maintaining bladder drainage. This eliminates the potential for submucosal or subcutaneous leakage of urine from the edges of the urethral repair, and allows healing of the urethral epithelium to occur with minimal scarring and fibrosis. Healing is usually complete within 7-10 days, when normal urination can resume.

Technique

- Percutaneous methods or surgical methods are possible, although the surgical method is simpler and more predictable in outcome.

- Via a midline laparotomy, a cystostomy tube (e.g. Foley catheter) is placed through the abdominal wall via a paramedian stab incision and then placed into the bladder via another stab incision and the bulb of the catheter is inflated with saline. This is most useful if surgery has to be performed within the rest of the lower urinary tract/abdomen.

- An alternative is to make an initial dorsocranial-ventrocaudal directed incision in the caudolateral abdominal wall and use this to both access the bladder and bring the tube through the abdominal wall. The tube passes through a separate skin stab incision. The abdominal wall incision can be closed apart from where the tube passes through the wall.

- A purse string suture is placed in the bladder (ventrolateral body of bladder – avoid the bladder neck/trigone) around the tube using 3/0 absorbable suture material. The bladder is then tacked to the body wall. A finger trap suture secures the tube to the skin.

- The cystostomy tube is then secured to the body wall using a bandage or surgical netting dressing. This prevents inadvertent dislodgement by the animal standing on the drain.

Tube cystostomy has also been employed for prolonged periods as a palliative aid when the urethra is irreversibly obstructed due to neoplastic disease. Patient and owner tolerance was excellent, and some patients lived for many months longer than they would have if urinary diversion had not been employed. Complications of tube cystostomy include urinary tract infection, uroabdomen and peritonitis, premature removal, and leakage of urine from the stoma following removal.

Urinary infection should be expected to occur in all cases as a consequence of disruption to normal bladder defence mechanisms. Treatment with antibiotic is usually not necessary, and ideally, should be delayed until the tube is removed. A urine culture should be obtained immediately prior to tube removal to allow appropriate selection of antibiotic to be made. However, in a recent study, infections resolved in all cases without complication following tube removal and treatment with a broad spectrum antibiotic.

The potential for tube dislodgment and premature removal (which could lead to leakage of urine into the abdomen) is avoided by careful placement of the tube into the bladder at laparotomy. Laparotomy is preferred because the very mobile bladder of dogs and cats

makes placement by blind percutaneous means very difficult. In critical patients, a mini-flank laparotomy can be performed quickly (and oftentimes, under neuroeleptanalgesic sedation only) to accurately place the tube into the bladder.

Removal of the tube is straightforward. Mild leakage of urine from the stoma should be expected for up to 3 days after removal, but this is usually well tolerated.