Emergency Room Crash Course Online 'Mini Series'

Session 3: Small Animal Emergencies You Will See and How to Deal with Them- GDV and Blocked Cats

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CANINE GASTRIC DILATION/VOLVULUS SYNDROME

Theory refresher

Risk factors

A large number of risk factors have been suggested for acute gastric dilatation/volvulus syndrome (GDV). Some are supported by sound clinical evidence; others are more controversial or anecdotal. The cause of this condition however remains unknown and is likely to be multifactorial. Suggested risk factors include:

- Large/giant breed
- Purebred
- Narrow and deep chest
- Feeding of single daily meal
- Large amount of food consumed per meal
- Exercise after eating
- Eating quickly, especially if accompanied by aerophagia
- Increasing age
- Fearful/agitated demeanour
- Hereditary factors

- Increased length/laxity of hepatogastric ligaments
- Reduced gastric motility
- Delayed gastric emptying
- Increased gastrin levels
- Abnormal oesophageal motility

Small breed dogs and cats can be affected but this is rare.

Pathophysiology

The pathophysiology of GDV is multifactorial and involves the cardiovascular and gastrointestinal systems in particular. Respiratory compromise may also be severe.

Significant pathophysiological mechanisms are as follows:

- An increase in intra-abdominal pressure that compresses the caudal vena cava, portal vein and splanchnic vasculature. This causes a decrease in venous return to the heart, reducing cardiac output and potentially arterial blood pressure. As dilatation progresses, an

- increasing amount of blood is sequestered in the abdominal vasculature. There is therefore likely to be a component of *obstructive shock* in GDV patients.
- Myocardial function is further compromised by dysrhythmias and reduced contractility that occur due to the effects of reactive oxygen species produced in the abdomen as well as ischaemia and acidosis. There may therefore be a component of *cardiogenic shock* in GDV patients although this is usually difficult to objectify and may be the least significant form of shock here.
- Perfusion of the gastric wall is compromised as a result of both compression of capillaries due to raised intraluminal pressure and a reduced blood supply due to systemic hypoperfusion. This results in ischaemia, necrosis and potentially perforation. Intraluminal haemorrhage occurs due to mucosal necrosis.
- The short gastric arteries branch off the splenic artery and supply the greater curvature of the stomach. They may be avulsed when the stomach rotates during acute GDV resulting in potentially severe haemorrhage and further reducing perfusion to the stomach wall.
- Gastric and intestinal motility is severely diminished and significant volumes of fluid may be sequestered in the gastrointestinal tract. Fluid third-spacing and blood loss lead to a component of *hypovolaemic shock* in GDV patients.
- The distended stomach presses on the diaphragm reducing intrathoracic volume and impairing movement of the diaphragm during inspiration. This results in hypoventilation due to reduced tidal volume and a compensatory increase in respiratory rate occurs.

Cardiovascular SHOCK in GDV (* main types):

HYPOVOLAEMIC*	• Haemorrhage: into stomach, intestines; from short gastric artery avulsion • Gastrointestinal fluid third-spacing
OBSTRUCTIVE*	• Increased abdominal pressure causes reduced venous return
Distributive	• Systemic vasodilation from inflammatory mediators, reactive oxygen species
Cardiogenic	• Myocardial dysfunction from reduced contractility +/- dysrhythmias

Clinical signs

The most classical clinical signs of GDV are non-productive retching and drooling, often but not always within a few hours of the evening meal. Some dogs appear restless, pacing around, possibly panting, and being unable to settle. They may become progressively lethargic and owners may

notice abdominal distension. Some owners are very aware of the possibility of 'bloat' and will mention this at the time of the initial phone call. Progression with GDV can be very rapid and sadly sometimes the first time the owner notices there is a problem the dog is already collapsed and moribund, occasionally already dead.

Physical examination

There is a spectrum of presentation in terms of severity with GDV. Some dogs appear relatively normal walking around and looking reasonably bright; these cases may have variable degrees of abdominal distension when examined.

However a significant proportion of dogs with GDV present with hypoperfusion/shock that can vary from mild to very severe (tachycardia, weak-absent femoral pulses, pale mucous membranes, slow capillary refill time, recumbency, depression/obtundation). Pulse deficits may be identified if a dysrhythmia is present.

Respiratory status may be unremarkable but panting is common and some dogs are cyanotic.

The abdomen may be mildly-to-severely distended – this can often be appreciated just by observation in the worse cases – and palpation reveals tympany that is localised cranially or perhaps more diffusely as well as discomfort. Drooling may be identified. Rectal temperature may be normal, increased or decreased.

Clinical evaluation

The priority in dogs with GDV is to commence stabilisation with fluid therapy and subsequently perform gastric decompression. Any tests that are performed should be done concurrently without compromising patient stabilisation.

An emergency database including manual packed cell volume (PCV), plasma total solids, blood urea nitrogen and glucose is recommended as a minimum. If facilities allow then measuring blood lactate concentration is also highly recommended (see below). Plasma electrolytes, especially potassium, should also be measured, especially if ventricular dysrhythmia requiring specific therapy is present.

Hyperlactataemia in GDV patients:

Measuring blood lactate concentration is most useful clinically to improve our ability to detect systemic hypoperfusion/shock in any patient, i.e. type A lactic acidosis due to absolute tissue oxygen deficiency. Many dogs with GDV are hyperlactataemic at presentation and serial monitoring of this parameter during treatment can help provide information regarding the patient's progress. There are a couple of points that it is essential to bear in mind…

1. At the current time, with the evidence available, a single lactate measurement should *not* be used prognostically with respect to making treatment recommendations to the owner. A few papers have

been published over the last 10-15 years that have tried to establish a cut-off value associated with a poorer prognosis but these papers have also been subjected to critique and do not constitute a reliable powered evidence base. This author and many others has successfully managed dogs to discharge that presented with blood lactate concentrations ranging from normal (< 2.5 mmol/l) to > 15 mmol/l (i.e. very severely elevated). It is true to say that animals with more severe hypoperfusion or that have suffered more severe organ compromise, i.e. gastric +/- splenic necrosis, usually have higher lactate levels at presentation but this does not reflect reversibility or necessarily correlate to outcome – this remains to be sufficiently evidence-based and recommending euthanasia on the basis of severe hyperlactataemia is not recommended by this author.

2. Serial measurements are much more useful than a single measurement and *may* have some prognostic implications. In general, animals in which hyperlactataemia persists through initial stabilisation and especially post-operatively may be expected to have greater morbidity and more prolonged hospitalisation, and perhaps greater mortality although the latter in particular is not robustly evidence-based.

Treatment

1. Initial stabilisation:

The priority in GDV patients is to commence appropriately aggressive **intravenous fluid therapy**. This is ideally done using two large bore (e.g. 14-16 gauge) short catheters, one in each cephalic vein. Administration of resuscitative fluids via saphenous catheters will be much less effective due to obstruction of the caudal vena cava. In the majority of cases fluid resuscitation can be achieved through the use of an isotonic crystalloid alone (e.g. Hartmann's solution). In some cases, the additional use of small volume resuscitation fluids (colloids, hypertonic saline) may be applicable if available. Examples are:

- Cases that fail to respond adequately to crystalloid therapy alone
- Extremely large dogs where administration of large volumes of crystalloid may take too long – canine GDV is one of the main uses of hypertonic saline in companion animal practice
- Animals in which only limited intravenous access has been established

[Why give fluids before decompression?

GDV patients are often in cardiovascular shock that may be largely hypovolaemic in aetiology. Appropriately aggressive fluid therapy usually results in significant improvement in the patient's condition. Admittedly in some cases this can only be transient, in others the response relatively poor, because obstructive shock is also a major cause of poor cardiac output in these patients. In some cases relieving the obstruction is key to improving patient stability.

Nevertheless it is always recommended to administer some fluid resuscitation before gastric decompression. Gastric decompression will allow reactive oxygen species, hydrogen ions and inflammatory mediators sequestered in the abdomen to reach the heart which can worsen
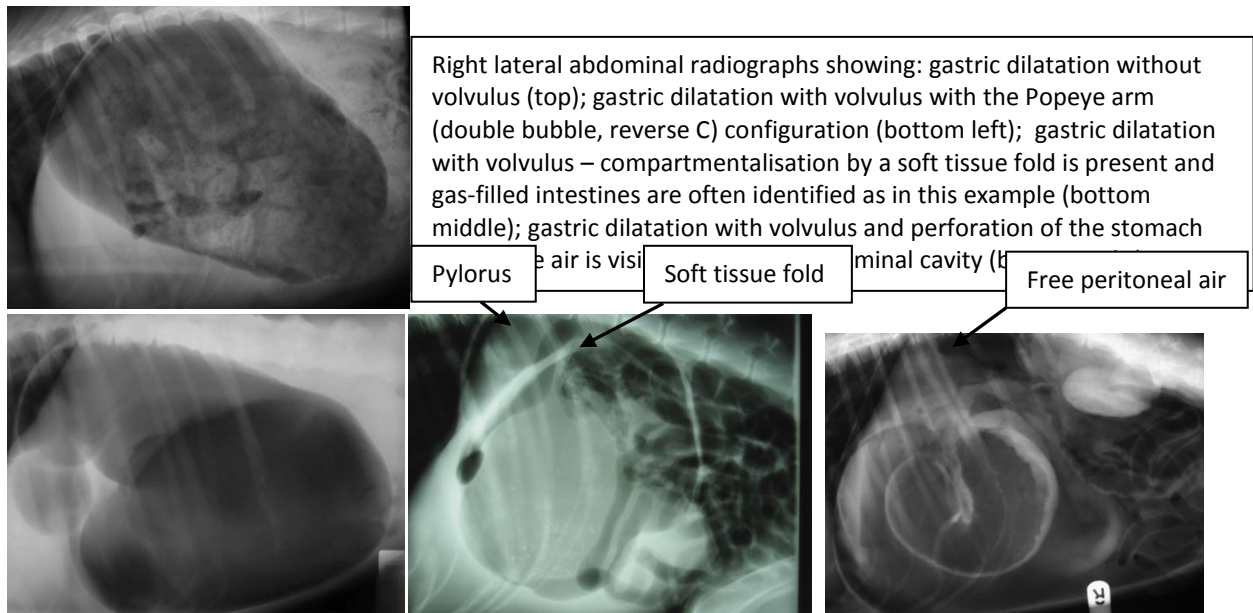
myocardial function. Pre-treatment with fluids may go some way to countering any detrimental effect of this myocardial suppression on systemic perfusion.]

Analgesia should also be administered during this early period. This is ideally with a pure (full) opioid and appropriate options include methadone or fentanyl; morphine is ideally avoided due to its potential emetic effect. Non-steroidal anti-inflammatory agents should not be used in any animal with hypoperfusion. Furthermore, the inevitable compromise to the gastrointestinal mucosa during acute GDV warrants judicious and delayed use of these agents.

Acute GDV can usually be diagnosed on the basis of history, signalment and physical examination, and radiography is generally not required. However, radiography may allow differentiation of gastric dilation alone from gastric dilation with volvulus.

Radiography should not be performed until after fluid therapy and analgesia have been commenced and only if the patient is compliant as is usually the case. Radiography may be performed during the initial fluid resuscitation but should not delay gastric decompression. A single radiograph with the patient in right lateral recumbency is all that is typically required.

Thoracic radiography may be indicated in dogs suspected of having cardiac disease or inappropriate respiratory compromise (e.g. due to aspiration), or to rule out coexisting disease (e.g. neoplasm) in older animals.

Right lateral abdominal radiographs showing: gastric dilatation without volvulus (top); gastric dilatation with volvulus with the Popeye arm (double bubble, reverse C) configuration (bottom left); gastric dilatation with volvulus – compartmentalisation by a soft tissue fold is present and gas-filled intestines are often identified as in this example (bottom middle); gastric dilatation with volvulus and perforation of the stomach — free air is visible in the abdominal cavity (bottom right)

Pylorus Soft tissue fold Free peritoneal air

2. Gastric decompression:

Once fluid resuscitation is well under way, gastric decompression is performed. This is usually done under sedation which involves potentially topping-up the pure opioid already administered and adding in a benzodiazepine (e.g. diazepam 0.5 mg/kg IV).

Gastric decompression is ideally done via **orogastric intubation** (see description at end of notes). It must be remembered that the ability to pass a stomach tube does not exclude the presence of some degree of gastric volvulus in gastric dilatation/volvulus syndrome (mild to moderate volvulus may still allow the stomach tube to pass through the lower oesophageal sphincter). **Needle decompression** is a quick and easy procedure that is effective in relieving gaseous distension. It does involve blindly placing a needle through a gastric wall that may already be severely compromised; also potential risk of gastric contents leaking into abdomen (although there is currently no published evidence that this results in clinically significant complications). It should perhaps therefore be reserved for the following types of cases:

- Animals that are intolerant of orogastric intubation despite appropriate sedation
- Animals that are intolerant of orogastric intubation and that are deemed too unstable to sedate
- Animals in which a stomach tube cannot be passed

3. Surgery:

Depending on the timeframe, the pure opioid and benzodiazepine already administered can act as premedication for general anaesthesia as well. If needs be additional doses can be given, as for example can fentanyl at induction. If facilities allow, additional on-going strategies include continuing fentanyl or morphine infusions intraoperatively and potentially also a midazolam infusion

for example. Essentially the aim is to try and reduce the doses of cardiovascular-depressant anaesthetic drugs required to maximise safety in these critical patients. Close monitoring of vital signs, ideally also aided by a multiparameter monitor, is essential during general anaesthesia. Intravenous fluid therapy is continued during surgery as appropriate to support perfusion.

Intravenous broad-spectrum antibiosis (e.g. amoxicillin/clavulanic acid 20 mg/kg IV) is typically given preoperatively and repeated during surgery; however it does not need to be continued for longer than the perioperative period in all patients and this decision should be guided by findings at surgery.
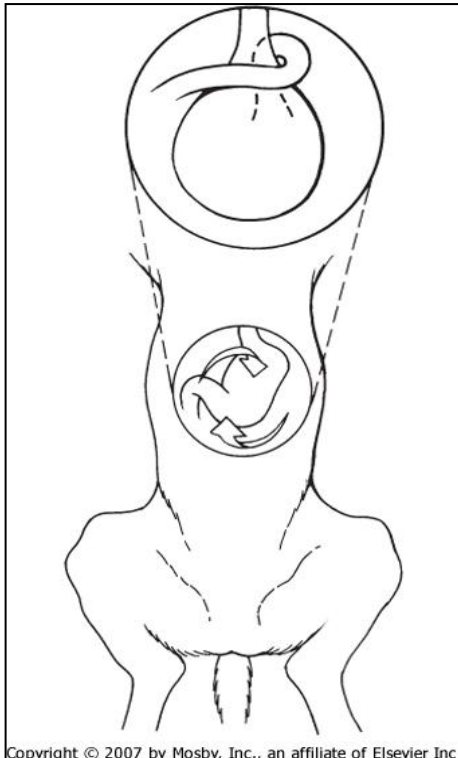
Although anti-clockwise rotation has been reported, the stomach usually twists clockwise. A 180° rotation – such that the pylorus comes to lie craniodorsally to the body of the stomach on the left of the abdomen – is most common, but 90°, 270° and 360° rotations may also occur. The spleen usually rotates with the stomach (attached by gastrosplenic ligaments) to a position right of midline.

Direction of gastric rotation in most dogs with GDV

(http://from-the-doghouse.com/Scoop%2BHowl/bloat-torsion.jpg)
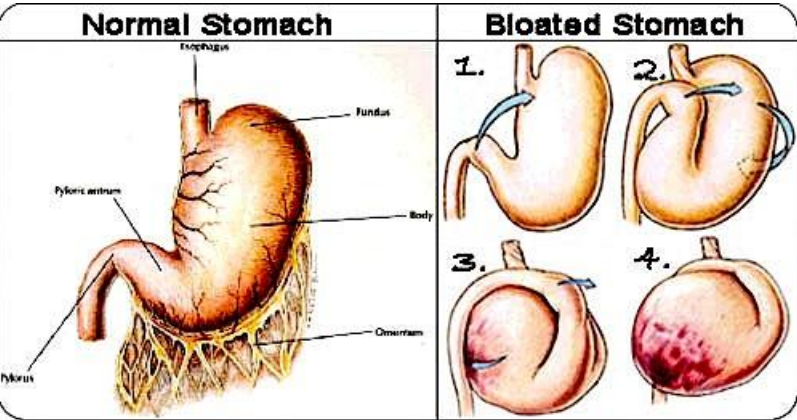
Is surgery always necessary?

The aims of surgical intervention in dogs with acute GDV are to:

- Decompress, empty and lavage the stomach if this has not already been achieved
- Derotate the stomach if necessary
    - A gastrotomy may be necessary if the stomach cannot be sufficiently decompressed to derotate it without opening it up and manually removing some of the contents; ideally to be avoided if at all possible though.
- Evaluate the viability of the stomach and perform invagination/gastrectomy if indicated
- Perform gastropexy between the pyloric antrum and the right body wall to prevent volvulus in the future
- Evaluate the viability of the spleen and perform a splenectomy if indicated
- Lavage the abdomen to remove haemorrhage and possibly gastrointestinal contents

In animals in which gastric volvulus persists despite decompression or in which the presence of volvulus cannot be excluded, surgical intervention is mandatory but should only be performed when the patient is haemodynamically stable.

As described above, there are compelling reasons for surgical intervention in patients presenting with gastric dilatation alone. There are cases however in which owners may not be willing to provide consent, for example due to financial constraints or because of the age of the pet. Such cases can be successfully managed conservatively but the owner must be advised of the risks associated with this approach, particularly a lack of information regarding the viability of the stomach and spleen, and the possibility of recurrence both overnight and subsequently.

There are a variety of gastropexy techniques, that have their relative pros and cons. Incisional (or muscular flap) gastropexy is widely used nowadays:

Advantages:
Easy and quick to perform
Gastric lumen not opened

Disadvantages:
Less strong the circumcostal or belt-loop pexy
Does not provide direct post-operative access to gastric lumen like tube gastropexy does

1. Make an incision in the seromuscular layer of the gastric antrum
2. Make an incision in the right ventrolateral abdominal wall by incising only the peritoneum and internal fascia of the rectus abdominis or transverse abdominis muscles.

3. Suture the edges of the incisions in a simple continuous pattern using 2-0 absorbable or non-absorbable suture:
   - Suture the cranial margin first, then the caudal margin
   - Make sure the muscularis layer of the stomach is in contact with the abdominal wall muscle

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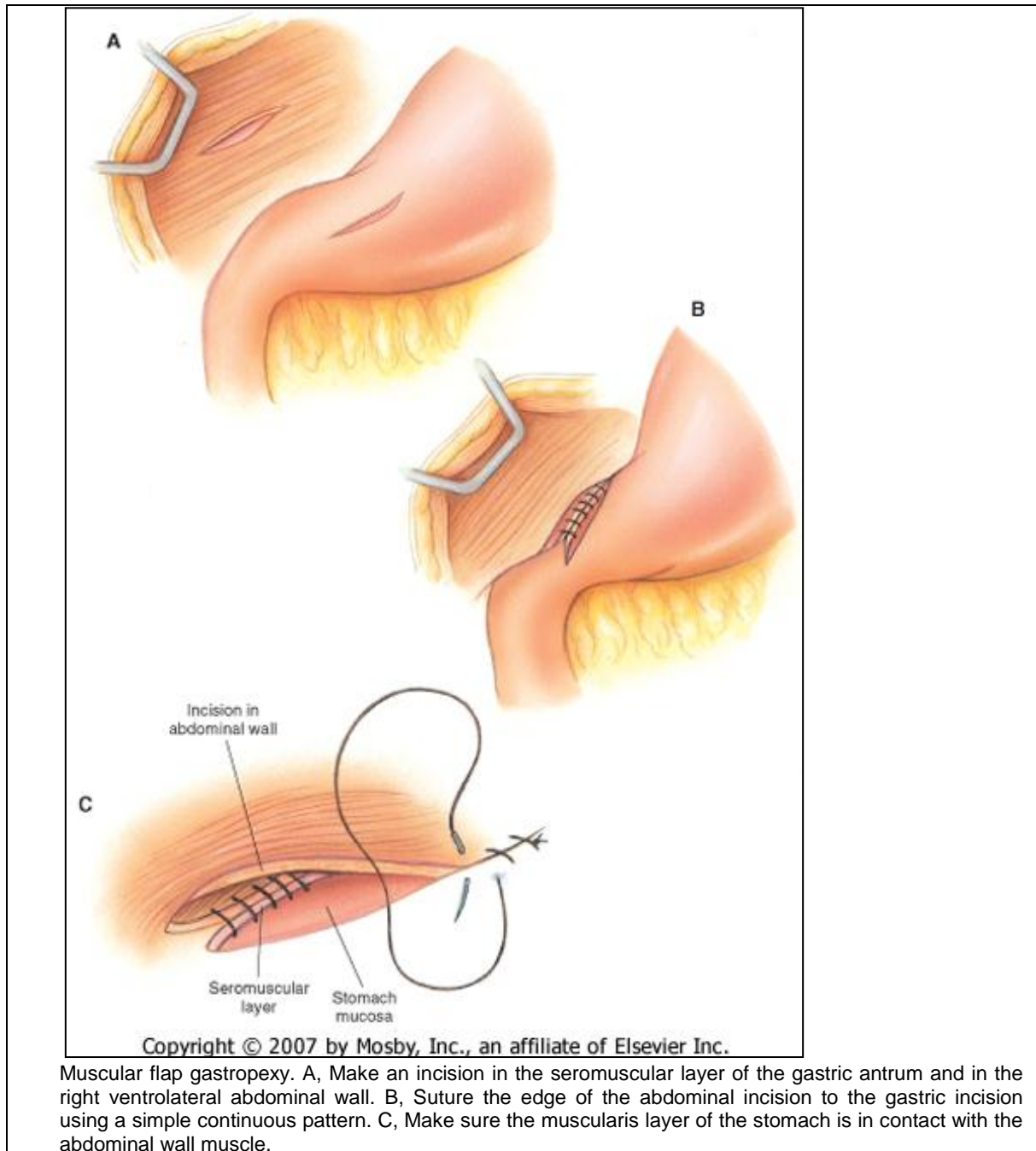
Muscular flap gastropexy. A, Make an incision in the seromuscular layer of the gastric antrum and in the right ventrolateral abdominal wall. B, Suture the edge of the abdominal incision to the gastric incision using a simple continuous pattern. C, Make sure the muscularis layer of the stomach is in contact with the abdominal wall muscle.

4. Post-operative management:

Post-operative management involves close on-going monitoring of cardiovascular status including for clinically significant dysrhythmia (see below). Fluid therapy is continued to support perfusion and hydration requirements; this is typically using an isotonic crystalloid alone but occasionally GDV dogs will develop hypoalbuminaemia or suspected vasculitis and a synthetic colloid is added in to try and provide oncotic support. Analgesia is continued and adjusted based on regular reassessment. Typically a pure opioid with/without other agents such as lidocaine or ketamine is used initially

switching to buprenorphine at the appropriate time. NSAIDs should ideally be withheld until it is apparent that the patient is stable and recovering well.

As well as routine database monitoring (e.g. PCV/TS, urea, lactate) serum potassium levels in particular should be monitored and supplemented as necessary, and the usual postoperative considerations such as wound management, appropriate bedding and bladder management attended to. Some dogs will benefit from anti-nausea/anti-emetic medication as well and metoclopramide and ranitidine may offer some benefit if gastrointestinal hypomotility is present.

Water is usually offered as soon as the patient has fully awoken from surgery and food offered for example 6-12 hours later.

Electrocardiogram abnormality has been reported to occur in approximately 40% of dogs with acute gastric dilatation/volvulus. Ventricular premature complexes (VPCs) and ventricular tachycardia (VT) are the most common, both pre- and postoperatively.

In general, ventricular dysrhythmias arising secondary to another condition such as acute GDV only warrant anti-dysrhythmic therapy if they are considered to be compromising the patient's perfusion based on physical examination and blood pressure measurement if available. In addition, VT should be treated if it occurs at rates greater than 180 beats per min or if R-on-T phenomenon is identified. Lidocaine is administered typically by giving a 2 mg/kg bolus IV and repeating this up to three more times if sufficient response is not achieved initially; an infusion is then started at 50-80 µg/kg/min.
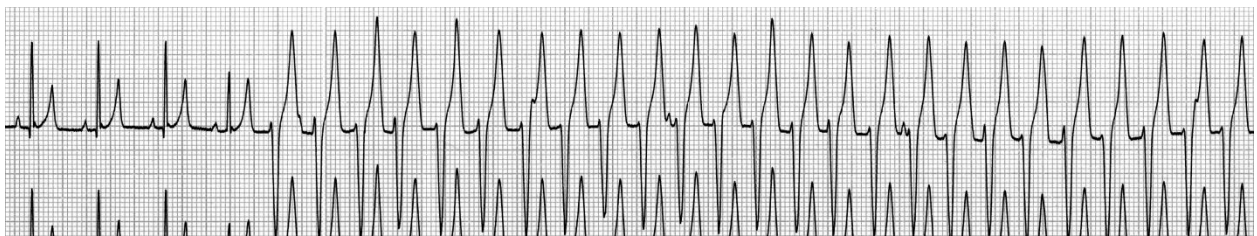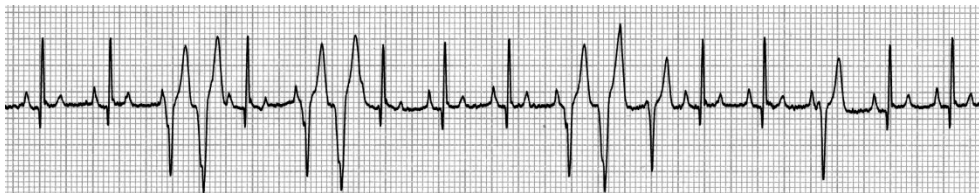
Ventricular premature complexes (top) and ventricular tachycardia (bottom)

R-on-T

The prognosis associated with acute canine GDV is generally good if appropriate timely treatment can be provided. In a small proportion of cases, intra-abdominal pathology is very severe at the time of surgery, in particular with respect to gastric necrosis, and a decision to euthanase the patient may be made; clearly to an extent this decision is also influenced by the expertise and experience of the surgeon and the ability to provide intensive post-operative monitoring and care. However surgical options are available for most patients and the reported survival rates (albeit often from referral centres) are extremely high nowadays.

Gastric decompression – Orogastric intubation

Equipment

Equipment list:
- Roll of tape with a hollow core or mouth gag
- Appropriately-sized smooth stomach tube, preferably with multiple fenestrations
- Lubricant for stomach tube
- Means of marking the stomach tube (e.g. piece of tape, suitable marker pen)
- Bucket (or similar) for collecting gastric effluent

Procedure

1. Ideally position the patient in sternal recumbency but allow him/her to adopt their preferred position
2. Pre-measure the stomach tube from the nostrils to the last rib and mark it
3. Place the roll of tape (or gag) longitudinally in the mouth between the upper and lower incisors/canines to prevent chewing of the tube
4. Lubricate the tube well and pass it through the roll of tape down the oesophagus; the opening of the oesophagus lies dorsal and to the left of the larynx (i.e. dorsal and to the right of the larynx as you are facing the patient's head).
   - There is usually some resistance as the tube passes through the distal oesophageal sphincter; advance the tube gently with a rotating movement and avoid excessive force to minimise the risk of perforation.
5. Collect any gastric effluent and examine it grossly
6. Kink the tube prior to removal to reduce the risk of aspiration.

Complications/Notes

Complications:

Clinically significant complications associated with orogastric intubation are rare. They include:

Perforation of the oesophagus or stomach if excessive force is used during placement of the tube or if the patient is inadequately restrained
Aspiration of gastric contents:
- Any patient with an absent gag reflex should have endotracheal intubation performed with a well-fitting cuffed tube prior to orogastric intubation
- Gastric *lavage* is not recommended in any patient without an endotracheal tube in situ

Notes:

Depending on the patient in question and his/her clinical condition, variable degrees of manual restraint are required for orogastric intubation. However, as always, excessive manual restraint is to be avoided in any potentially unstable or critically ill animal. Conservative sedation, for example

using a pure opioid with a benzodiazepine, can go quite a long way to improving patient compliance and comfort during the procedure.

Gastric decompression – Percutaneous needle decompression

Equipment

Equipment list:
- Clippers
- Surgical scrub materials
- Sterile gloves
- 16, 18 or 20 gauge over-the-needle intravenous catheter (or similar sized hypodermic needle)

Procedure

1. Restrain the patient in sternal recumbency or standing if preferred; left lateral recumbency may also be used
2. Identify where the abdomen is maximally distended on the right side
3. Percuss the site for tympany to indicate that the stomach and not the spleen for example underlies the chosen site
4. Clip and aseptically prepare the chosen area
5. Insert the intravenous catheter percutaneously into the stomach at the chosen site and remove the stylet. Gas with a characteristic smell is released immediately through the catheter.
   - Gastric emptying can be encouraged by gentle manual compression of the cranial abdomen but be gentle.

Complications

Quick and easy procedure that is usually effective in relieving gastric distension in patients in which this is largely due to gas accumulation. However, it does involve placing a needle through a gastric wall whose viability is unknown at the time of puncture and which may already be severely compromised (e.g. in canine gastric dilatation/volvulus). Although there is currently no published evidence that this procedure results in clinically significant complications, orogastric intubation may be more rational as a first-line decompression procedure with needle decompression being reserved for patients:
- Intolerant of orogastric intubation despite appropriate sedation
- Intolerant of orogastric intubation that are too unstable to sedate
- In which a stomach tube cannot be passed

Another possible complication is placement of the catheter into another abdominal organ.

Gastric lavage under general anaesthesia

Equipment

Equipment list:
- Roll of tape with hollow core or mouth gag
- Appropriately-sized smooth stomach tube, preferably with multiple fenestrations:
    - The procedure can be made more efficient by using two stomach tubes (one to pour lavage fluid down, the other to collect the effluent) or a commercially available double lumen stomach tube.
    - The procedure below describes lavage using one single lumen tube only
- Lubricant for stomach tube(s)
- Means of marking the stomach tube (e.g. piece of tape, suitable marker pen)
- Bucket (or similar) for collecting gastric effluent and lavage fluid
- Funnel and jug for performing lavage
- Lavage fluid (approximate total volume of 25-100 ml/kg): tepid tap water is fine although warmed normal saline or buffered lactated Ringer's solution can also be used

Procedure

1. Ensure the patient is under general anaesthesia and intubated with a well-fitting cuffed endotracheal tube
2. Position the patient in sternal recumbency
    - Clearly the patient will be in dorsal recumbency for gastric lavage being performed during GDV surgery
3. Pre-measure the stomach tube from the nostrils to the last rib and mark it
4. Place the roll of tape (or gag) longitudinally in the mouth between the upper and lower incisors/canines to prevent chewing of the tube if the patient was to awaken suddenly.
5. Lubricate the tube well and pass it through the roll of tape down the oesophagus; the opening to the oesophagus lies dorsal and to the left of the larynx (i.e. dorsal and to the right of the larynx as you are facing the patient's head).
    - There is usually some resistance as the tube passes through the distal oesophageal sphincter; advance the tube gently with a rotating movement and avoid excessive force to minimise the risk of perforation.
6. Collect any gastric effluent and examine it grossly
    - Correct tube placement can obviously be confirmed by manual palpation of the tube through the gastric wall during GDV surgery
7. Pour 5-10 ml/kg of the lavage fluid down the tube from a height using a funnel and jug if available; then lower the free end of the stomach tube below the level of the patient to allow stomach contents to flow under gravity out of the tube into the effluent bucket.
8. Repeat this lavage as necessary depending on what the procedure is being performed for; the lavage is often performed 5-10 times but it can be many more if the aim is to breakdown and dissolve large quantities of food for example. The typical end-point is lavage fluid that is clear of gastric contents.

- Gentle manipulation of the stomach from the outside can help to recover the lavage fluid, as can careful repositioning of the patient periodically.
9. Kink the tube prior to removal to reduce the risk of gastric lavage fluid running into the oesophagus or into the oropharynx and being aspirated.
10. Finally rinse out the mouth with tap water to remove any gastric contents; aspiration can be used to make this cleaning out more effective.

Complications

Clinically significant complications associated with gastric lavage are rare if the described procedure is adhered to. They include perforation of the oesophagus or stomach if excessive force is used during placement of the tube. Aspiration of gastric contents or lavage fluid is another possible complication.
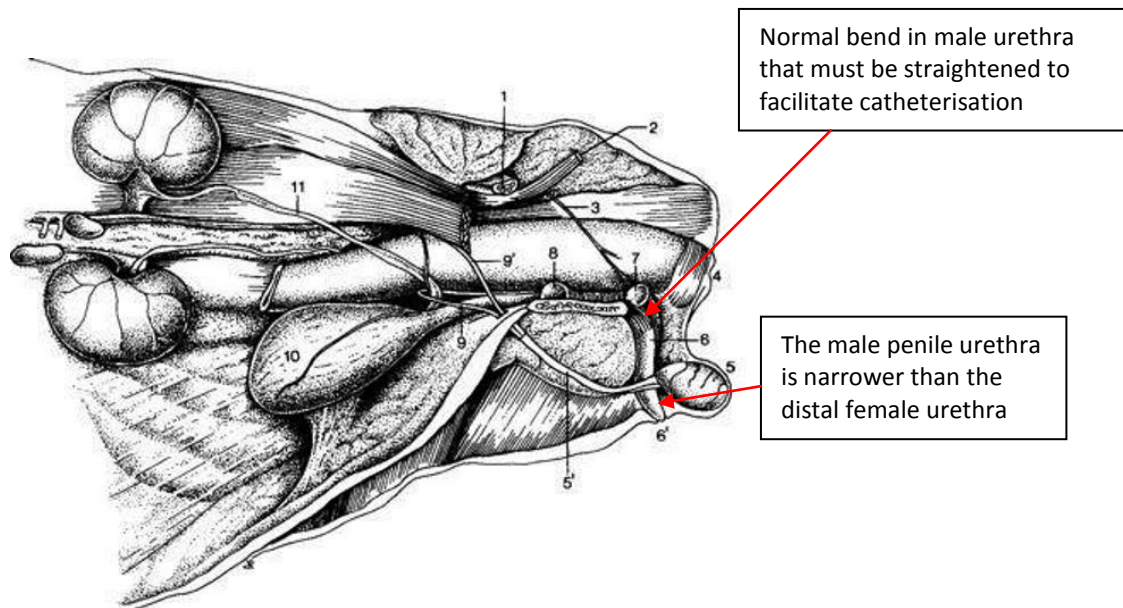
TOMCAT URETHRAL OBSTRUCTION

Theory refresher

Urethral obstruction

Mechanical urethral obstruction is much more common in male cats due to the narrow diameter of the penile urethra and it occurs very infrequently in females.

> Normal bend in male urethra that must be straightened to facilitate catheterisation

> The male penile urethra is narrower than the distal female urethra

"Figure 15-18: The reproductive organs of the tomcat in situ, left lateral view. 1, Shaft of ilium; 2, sciatic nerve; 3, pudendal nerve; 4, anus; 5, left testis in scrotum; 5′, spermatic cord; 6, penis; 6′, prepuce; 7, bulbourethral gland; 8, prostate; 9, deferent duct; 9′, testicular vessels; 10, bladder; 11, left ureter"

[This figure is adapted from the eBook of "Textbook of Veterinary Anatomy, 3rd edition" by K. M. Dyce, DVM S, BSc, MRCVS; W.O. Sack, DVM, PhD, Dr. med. vet.; and C. J. G. Wensing, DVM, PhD.]

Causes of urethral obstruction:

- Urethral plugs – the most common cause in male cats
- Uroliths may also cause obstruction, potentially in combination with a plug
- Urethral spasm
- Urethral strictures
- Tumours (rare) e.g. prostatic carcinoma

Urethral plugs are often made of struvite material in a proteinaceous matrix. The most common **uroliths** in cats are made of struvite or calcium oxalate. Unlike in dogs, struvite urolithiasis in cats is not typically associated with urease-producing bacterial infection.

Feline lower urinary tract disease (FLUTD)

Lower urinary tract disorders in cats include:

- Urolithiasis
- Feline idiopathic cystitis
- Urinary tract infection
- Anatomical defects
- Behavioural disorders
- Neoplasia

These disorders may all be associated with a constellation of clinical signs that are considered under the umbrella term of feline lower urinary tract disease (FLUTD):

- *Dysuria* = a general term used to describe difficult and painful urination
- *Stranguria* = straining to urinate, typically painful
- *Pollakiuria* = voiding small amounts of urine frequently; 'spotting'
- *Gross haematuria* = blood visible in urine macroscopically

FLUTD occurs most commonly in 2- to 6-year old cats. An increased risk has been reported for example in cats confined indoors and those restricted to dry food diets with inadequate fluid intake. Depending on the underlying lower urinary tract disorder, partial or complete urethral obstruction may occur and this clearly has significant implications from an emergency perspective.

Cats presenting with lower urinary tract signs but without obstruction usually have a small or empty bladder and require symptomatic medical therapy, analgesia in particular. Many of these cats will have idiopathic cystitis and will improve within 3-5 days.

Clinical signs/Initial communication

Cats with urethral obstruction may show a variety of clinical signs of variable severity. In some cases the signs are non-specific (e.g. anorexia, lethargy, vomiting). An abnormal pelvic limb gait that is often presumed to be the result of trauma may be all that is reported; alternatively the cat may be showing non-specific vocalisation or apparent discomfort, especially when picked up. Not all cats with urethral obstruction show consistent urinary signs early on.

Emergency consultation should be recommended for all cats showing lower urinary tract signs. Male cats in particular may have complete urethral obstruction which can be rapidly fatal. Both unobstructed male and female cats require analgesia as a minimum as these clinical signs are typically associated with varying degrees of pain.

In some tomcats with urethral obstruction owners report that their cat is straining non-productively but are unable to distinguish stranguria from constipation.

All male cats with signs of potential obstruction that either have a history of lower urinary tract disease or that are confined indoors (at increased risk of urethral obstruction) should be examined.

Physical examination

Physical examination findings can vary in cats with urethral obstruction. Some cases appear relatively normal except for the presence of a medium-to-large typically hard/rigid bladder, while others can be very severely compromised and form the focus of these notes…

Major body system examination (primary survey):

Cardiovascular system: may be consistent with variable degrees of hypoperfusion/hypovolaemia and an inappropriately severe bradycardia may be present.

> The causes of hypoperfusion/hypovolaemia in blocked cats are relatively poorly defined but may include:
> - The effects of hyperkalaemia on cardiac output
> - The effects of acidaemia on vasomotor control
> - A component of dehydration where this has developed

The 'classic' presentation for a cat in shock is hypodynamic with bradycardia (often 130-150 bpm), pallor, slow CRT, weak-absent femoral pulses, hypothermia and depression/obtundation. Cats with clinically significant hyperkalaemia may however have even lower heart rates, i.e. inappropriately severe bradycardia.

Respiratory system: often unremarkable; sometimes tachypnoea is present which may reflect pain and metabolic status

Central nervous system: the sickest cats are typically depressed-obtunded and recumbent.
Pain assessment: (presumed) moderate-to-severe pain

Secondary survey:

Abdominal palpation: Moderate-to-large rigid bladder is a common finding
Body temperature: Hypothermia is a common finding

> Although rare, urinary bladder rupture may occur due to urethral obstruction and therefore the absence of a palpable bladder does not exclude this condition. Cats with urethral obstruction and bladder rupture are very likely to be moribund and severely azotaemic.
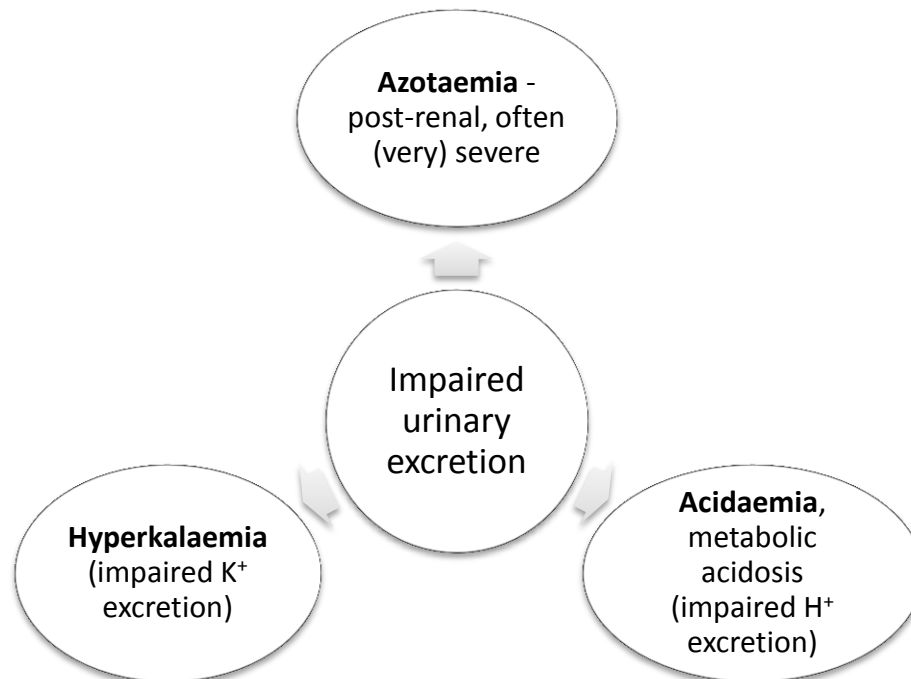
Clinical evaluation

Clinical pathology:

Starting to stabilise the patient is the priority (see Treatment below) over performing blood tests and treatment is always directed at the patient not at a specific blood parameter.

Common findings in sick cats with urethral obstruction include:
- Severe azotaemia
- Variable hyperkalaemia
- Acidaemia with metabolic acidosis

Many of the clinical pathological abnormalities identified in cats with urethral obstruction are the result of impaired urinary excretion; in particular azotaemia, hyperkalaemia and metabolic acidosis are due to impaired excretion of blood urea nitrogen and creatinine, potassium ions and hydrogen ions.

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                    ┌─────────────────┐
                    │  Azotaemia -    │
                    │ post-renal,often│
                    │  (very) severe  │
                    └─────────────────┘
                            ▲
                    ┌─────────────────┐
                    │    Impaired     │
                    │    urinary      │
                    │    excretion    │
                    └─────────────────┘
              ◄                            ►
   ┌─────────────────┐            ┌─────────────────┐
   │  Hyperkalaemia  │            │   Acidaemia,    │
   │  (impaired K⁺   │            │    metabolic    │
   │   excretion)    │            │    acidosis     │
   └─────────────────┘            │  (impaired H⁺   │
                                  │   excretion)    │
                                  └─────────────────┘
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Azotaemia - post-renal, often (very) severe

Impaired urinary excretion

Hyperkalaemia (impaired K^+ excretion)

Acidaemia, metabolic acidosis (impaired H^+ excretion)

Urethral obstruction is a major cause of post-renal azotaemia that frequently is (very) severe. However in most cases the azotaemia fully resolves, often within 24-48 hours, with appropriate treatment. Very occasionally, prolonged urethral obstruction and back pressure on the kidneys may result in sufficient renal injury as to cause a degree of renal azotaemia that may persist long-term; however this is a rare finding. Cats that are severely hypoperfused at presentation will also have a pre-renal component to their presenting azotaemia due to reduced renal blood flow.

It is important to realise that the **degree of azotaemia at presentation should not be used as a prognostic indicator** and renal insufficiency must not be assumed until full treatment for urethral obstruction has been provided.

Electrocardiography:

Electrocardiography will be informative, both in demonstrating the presence and nature of a bradydysrhythmia and for subsequent monitoring. Ideally it should be started while awaiting blood test results or simultaneously with treatment for hyperkalaemia; however if this is not possible due to lack of staffing, hyperkalaemia should be addressed as the priority.

The most severe and most recognisable signs of hyperkalaemia are those affecting the **heart**. The clinical manifestations of hyperkalaemia reflect alterations in cell membrane excitability and of greatest concern are the potentially life-threatening effects on cardiac conduction. An electrocardiogram (ECG) displaying cardiac dysrhythmias can be a good indicator of hyperkalaemia if serum potassium levels are not immediately available in an animal with suggestive history and physical examination findings. However the ECG is not a good substitute for serum potassium levels to determine the degree of abnormality present.

Electrocardiogram changes associated with hyperkalaemia include:
- Sinus bradycardia
- Prolongation of the PR interval
- Widening and bizarre appearance of the QRS complex, including sinoventricular rhythm
- Tall T waves
- Decreased amplitude, widening or complete absence (atrial standstill) of the P wave
- Asystole or ventricular fibrillation may be seen in association with cardiopulmonary arrest

Although ECG changes do tend to progress as hyperkalaemia worsens, there is no direct correlation between blood potassium concentration and ECG changes in clinical sick patients.

Prior to calcium gluconate administration:

Peaked T wave   VPC   Absence of P wave
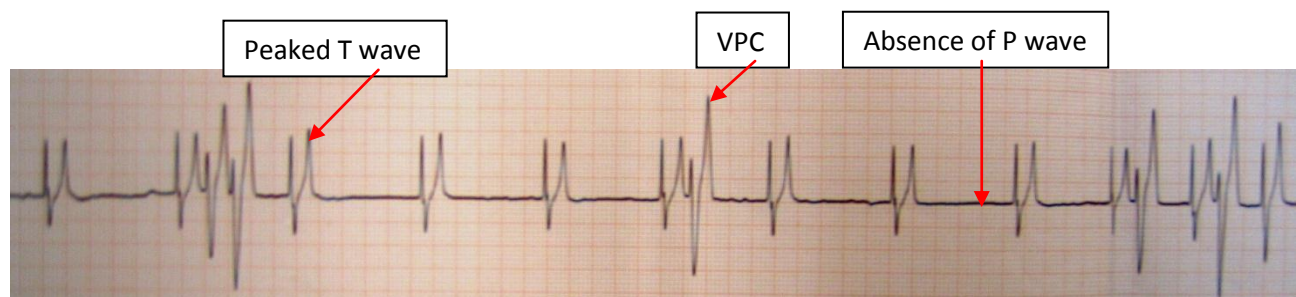
The rhythm strip shows atrial standstill with absence of P waves, as well as peaked T waves and ventricular premature complexes (VPCs).

After calcium gluconate administration

P waves now visible

Electrocardiogram following administration of calcium gluconate. P waves are visible and there are no VPCs in the post-treatment strip.

Treatment

1. Intravenous fluid therapy to address perfusion abnormalities:

A replacement isotonic crystalloid is the fluid of choice in urethral obstruction to restore perfusion and enhance urinary potassium excretion. Either Hartmann's (buffered lactated Ringer's solution) or 0.9% sodium chloride (physiological, normal saline) can be used. Normal saline (0.9% sodium chloride) has traditionally been recommended due to its lack of potassium; however this fluid may contribute to existing metabolic acidosis (it promotes a hyperchloraemic metabolic acidosis). There is *no clinically significant* difference between the use of 0.9% sodium chloride solution and Hartmann's (buffered lactated Ringer's solution) which contains a small amount of potassium. The priority is very much to start the cat on one or other of these solutions at a rate that is appropriate for the degree of hypovolaemia.

All cats with urethral obstruction will benefit from intravenous fluid therapy prior to catheterisation. One of the concerns often raised with regard to fluid therapy prior to urethral catheterisation is that of further filling a bladder that may already be quite distended. However urine production will be non-existent or minimal in hypoperfused cats with urethral obstruction; bladder will be filling extremely slowly if at all. This is because of a decrease in net filtration pressure resulting from:

- A decrease in the pressure promoting glomerular filtration and urine production. In a cat without pre-existing renal disease, once systemic hypoperfusion reaches a certain level, urine output becomes highly dependent on kidney perfusion. In an obstructed cat with hypotension and consequently reduced renal blood flow, urine production will be absent or minimal.
- An increase in the pressure opposing glomerular filtration and urine production. Due to back pressure transmitted from the urethral obstruction to the kidneys via the bladder and ureters.

By the time urine production is increasing following successful resuscitation, interventions will be underway to relieve the obstruction.

2. Hyperkalaemia:

The severity of clinical signs resulting from hyperkalaemia does not necessarily correlate with the absolute increase in serum potassium concentration. The treatment required is therefore dependent not just on the cat's serum potassium level but also very importantly on whether the hyperkalaemia is assessed to be clinically significant.

If treatment is deemed necessary, it should be performed before urethral catheterisation is attempted; treatment is directed at improving the cat's clinical status and not at the serum potassium concentration per se.

In some cases, intravenous fluid therapy and restoring urine production and drainage is all that is necessary for hyperkalaemia to resolve. However fluid therapy alone is not sufficient if hyperkalaemia is assessed as causing ECG changes and other cardiovascular compromise. Further treatment options in such cases are summarised in the table below.

If serum electrolytes cannot be measured but clinically significant hyperkalaemia is suspected on the basis of electrocardiography or perhaps physical examination alone, empirical use of calcium gluconate, and potentially insulin with glucose, is appropriate. This treatment can be life-saving and is unlikely to cause any significant harm if not required.

Agent	Dose / route	Comments
10% calcium gluconate	0.5-1.0 ml/kg IV bolus over 30-60 seconds	Rapid onset of action and first line choice in a crisis; indicated in all patients with clinically significant hyperkalaemia Short duration of action (often 10-15 minutes) Monitor ECG during administration Does not lower serum potassium concentration but restores normal cell membrane excitability stabilising cardiac conduction Will also address possible ionised hypocalcaemia Bolus can be repeated while other measures are used to directly reduce hyperkalaemia
Neutral (Regular, Soluble) insulin	0.25(-0.5) IU/kg IV	Slower onset of action (can be more than 15 minutes) Lowers serum potassium concentration by moving potassium (as well as glucose) into cells
Glucose solution	0.25-0.5 g/kg IV	Intravenous glucose supplementation usually required for several hours (monitor and adjust accordingly) In non-diabetic patients, some clinicians administer glucose alone without exogenous insulin relying on the resulting endogenous insulin spike to reduce hyperkalaemia
Sodium bicarbonate	1-2 mmol/kg slow IV (repeat if necessary)	Also lowers serum potassium concentration by moving potassium into cells

		Effect can persist for several hours
		Access to on-site acid-base analyser preferred
		Typically only used if patient has concurrent severe acidaemia

(* Note: when calcium gluconate is given intravenously for the treatment of hypocalcaemia, it should be given slowly (e.g. over 20-30 minutes) as a constant rate infusion. However this is clearly not a rational approach in a cat with life-threatening hyperkalaemia and calcium gluconate is administered much more quickly in these cases.)

3. Analgesia:

Urethral obstruction is clearly very painful. Pure (full) opioids are the gold standard analgesics with a rapid onset of action. A bolus of methadone or morphine is usually given at a dose range of 0.1-0.3 mg/kg IV depending on the individual cat; however in general, lower doses should be used until the cat is more stable.

Although buprenorphine is thought to be a very effective analgesic in cats, it has a slower onset of action (can be up to 45 min) and is therefore not appropriate as the first choice. Buprenorphine (0.01-0.02 mg/kg IV q 6-8 hours) is typically used following urethral catheterisation.

NSAIDs are contraindicated in all hypovolaemic or dehydrated patients. In addition, glucocorticoid administration may be considered during or following urethral catheterisation if significant urethral mucosal inflammation is suspected, and the concurrent use of both types of agent is contraindicated. Ideally NSAIDs should not be administered to cats with urethral obstruction until azotaemia has fully resolved.

Local anaesthesia should be incorporated into urethral catheterisation (see below).

Analgesia is very important in the management of cats with urethral obstruction. These cats are often very painful as a result of the urethral obstruction and severe lower urinary tract inflammation. Additional noxious stimulation is associated with urethral catheterisation and an in-dwelling urethral catheter.

4. Hypothermia:

Should hypothermic hypovolaemic cats be warmed aggressively?

Hypothermia significantly decreases the cardiovascular response to fluid resuscitation. However it is possible that re-warming the patient too quickly before administering sufficient fluid therapy may worsen their perfusion status as the increase in body temperature may cause peripheral vasodilatation thereby increasing the intravascular space. Not everyone agrees with this – some people believe that non-sweating animals like dogs and cats do not vasodilate (significantly) with warming especially when core temperature is low and there is even some suggestion that cats in shock may already be relatively vasodilated peripherally compared to dogs. These individuals therefore argue for more aggressive warming early on. However the most widely accepted recommendation is to limit on-going heat loss

with passive warming (e.g. wrap in blankets, place in incubator) during initial fluid resuscitation. In many cases as the patient's perfusion improves their rectal temperature will often increase notably; however more aggressive warming can be performed following initial fluid resuscitation if still thought necessary. Active re-warming measures include for example using a forced-air warming device (e.g. Bair-Hugger®, Arizant).

5. Sedation and urethral catheterisation:

The author's approach to cats with urethral obstruction is to stabilise them as thoroughly as possible with fluid resuscitation and treatment for hyperkalaemia such that chemical restraint beyond pure opioid analgesia is then required for urethral catheterisation. This approach allows sedation to be titrated upwards as needed including conversion to general anaesthesia in the knowledge that the patient has been stabilised thoroughly to maximise safety.

An alternative approach is to attempt catheterisation earlier at a time when the patient remains more moribund and therefore may not require additional chemical restraint. If this approach is adopted it is recommended to:
- Administer pure opioid analgesia first and allow enough time for analgesia to take effect
- Abort the procedure if the patient is non-compliant as additional stress may prompt potentially fatal decompensation; stabilise more thoroughly, administer sedation and try again.

The procedure for tomcat urethral catheterisation is described below.

Urinalysis should be routine in all cats with urethral obstruction including specific gravity, dipstick analysis, and especially sediment examination for bacteria. Haematuria and proteinuria are common findings.
- Intracellular bacteria are suggestive of infection
- Extracellular bacteria may also be significant, especially in a cystocentesis sample

Urine should be submitted for microbiology if there is suspicion of infection, although this is relatively rare in male cats with urethral obstruction.

THE DEBATE ABOUT CYSTOCENTESIS

Some clinicians prefer to perform cystocentesis very early on in the sickest cats with urethral obstruction in order to provide immediate urine drainage; catheterisation may then not be attempted for several hours to allow stabilisation. Cystocentesis is a simple technique to perform, especially in a full bladder, requires minimal if any chemical restraint, and allows rapid bladder drainage; this may potentially also facilitate urethral catheterisation by relieving the pressure on the urethral obstruction in some cases. The procedure also provides uncontaminated urine samples for bacteriology. In a very small proportion of blocked cats, urethral catheterisation is unsuccessful and cystocentesis is a convenient option pending further management.

However, whenever cystocentesis is performed, there is a risk of subsequent urine leakage into the abdomen and uroperitoneum. When cystocentesis is performed on a hard tense bladder, the sudden release of pressure could potentially significantly increase the risk of urine leakage – the effect may be rather like popping a balloon. In addition, the integrity of the bladder wall may be compromised in

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obstructed cats, increasing the likelihood of bladder wall rupture following cystocentesis. Delayed urine leakage is more likely in animals in which dysuria and bladder distension recurs following cystocentesis.

It is possible to very successfully manage cats with severe hyperkalaemia and acidaemia due to urethral obstruction without performing cystocentesis early on and it is strongly recommended that cystocentesis is reserved for those cases in which urethral catheterisation cannot be performed. If bacteriology is required, the first urine sample collected following urethral catheterisation may be used.

If cystocentesis is performed, either as a first line or preferably only because urethral catheterisation is unsuccessful, the bladder should be emptied as much as possible. This is for two reasons. Firstly achieving maximum decompression may facilitate urethral catheterisation as mentioned above. Secondly if urethral catheterisation fails subsequently, the bladder will already be almost empty and repeat aspiration will not be needed in the short-term. Cystocentesis is therefore best performed here using a three-way tap attached to a butterfly needle or to extension tubing connected to a needle/intravenous cannula.

DOES A URETHRAL CATHETER ALWAYS HAVE TO BE LEFT IN SITU?

The short answer to this question is no. However it is crucial to bear in mind that the answer to this question is very much contextual, i.e. it depends on the individual patient. In the sickest of cats with severe azotaemia, hyperkalaemia, acidaemia and subsequent post-obstruction diuresis, it is essential that on-going diuresis continues once the blockage has been relieved. As such the author would recommend leaving a catheter in situ for this group of patients to try and ensure that metabolic abnormalities improve in a timely fashion. Although urethral catheters do sometimes fail, the risk is likely to be lower than that of the cat re-obstructing if a catheter is not left in situ. In other words, in the author's opinion, the benefits of leaving a catheter in place in the sickest cats outweigh the risks/adverse effects.

However in other patient populations it is reasonable and gaining popularity, not to leave a catheter in situ. For example patients with mild or no clinicopathological abnormalities or ones with a recurrent history especially where urethral spasm is considered a significant contributing factor.

WHAT IF URETHRAL CATHETERISATION IS NOT POSSIBLE?

It is possible to catheterise the vast majority of blocked cats with patience, perseverance, and plenty of urethral flushing. However this is not always the case and repeated attempts are not benign – at the very least there is likely to be progressive inflammation and potentially haemorrhage secondary to the trauma; in the worst case scenario, iatrogenic urethral rupture may occur.

If catheterisation cannot be achieved then cystocentesis is rational. In some cases this may actually facilitate catheterisation – but not always! A tube cystostomy is then indicated to provide temporary urinary diversion; depending on available facilities and expertise, this may necessitate referral. A mushroom-tipped tube is used and percutaneous techniques are available.

Repeated cystocentesis as a short-term management strategy is very strongly discouraged – either tube cystostomy should be performed or the cat should be referred as an emergency. Each episode of cystocentesis is associated with an increased risk of subsequent uroperitoneum as well as additional

tissue injury and possible compromise of blood supply.

What about diagnostic imaging?

Diagnostic imaging is indicated in cats with urethral obstruction. Although most cats will be blocked by urethral plugs or spasm, a proportion will have discrete stones. These are likely to cause recurrent urethral obstruction unless removed via cystotomy at the appropriate time. However much consideration has to be given to the timing of radiography.

Diagnostic imaging is not urgent following successful catheterisation and therefore *a risk-benefit assessment is extremely valid early on.*

Ultrasonography can be used to detect urinary calculi in the bladder (cystoliths) including those that may be radiolucent on plain radiography. It is non-invasive, quick, and relatively simple to perform without interfering with patient stabilisation as long as a mobile scanner is available.


Urinary bladder calculi will usually produce a curvilinear echo with distal acoustic shadowing.

It is worth remembering that ultrasonography cannot adequately assess the whole urethra.

Plain *radiographs* will show radioopaque (mostly struvite, oxalate) cystoliths and maybe urethroliths:

- A useful mnemonic for remembering the radioopaque uroliths is 'P*** Off Cambridge University'; P = (ammonium magnesium **p**hosphate) struvite; O = oxalate; C = cystine; U = urate.
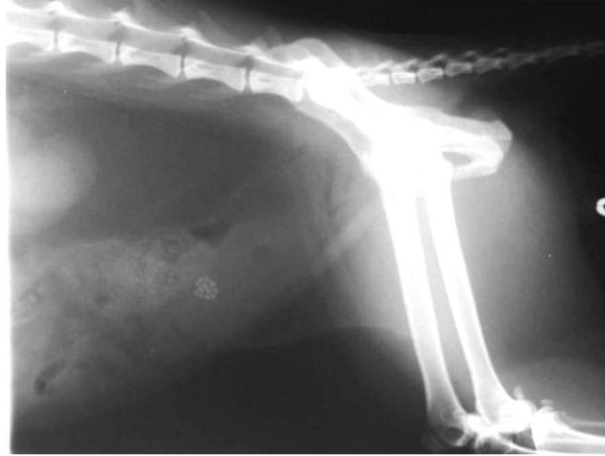
Radioopaque uroliths in the bladder of a cat with urethral obstruction; the radiograph was taken the day after the cat presented.

Contrast studies are likely to be needed for urethroliths and radiolucent cystoliths – both of these are less common in blocked cats and contrast studies would not be considered routine in a cat presenting for its first episode of urethral obstruction.

Chemical restraint for catheterisation provides a potentially useful opportunity for radiography to be performed. However the most appropriate time to perform radiography depends on a number of factors, for example:

- The individual patient: every attempt should be made to minimise the duration of chemical restraint and interventions early on in patients that are more severely affected at presentation, i.e. delay imaging until the next day even if additional judicious sedation is then required.
- The practice circumstances: what are the practical implications of performing radiography that may prolong sedation e.g. distance to radiography room, reliability of equipment etc? If a significant delay is anticipated during the early period that necessitates extra sedation/anaesthesia despite having already unblocked the cat, radiography should be postponed until the next day.

Post-catheterisation management

Intravenous fluid therapy must be continued following urethral catheterisation using an isotonic replacement crystalloid at a rate that is adequate to:

- Keep up with urine output
- Provide for maintenance requirements
- Correct any existing dehydration

A post-obstructive diuresis often occurs early on following urethral obstruction. The diuresis is reportedly most commonly osmotic due to excretion of accumulated urea and its severity may correlate to the severity of post-renal azotaemia. The diuresis is also thought to be due to excretion of accumulated sodium and administered fluids. The post-obstruction diuresis can result in large volumes

of urine being excreted, and dehydration will develop or worsen if adequate fluid therapy is not provided during this period. However, excessive fluid therapy is also to be avoided as it may promote renal medullary washout with the cat temporarily losing the ability to concentrate its urine.

An empirical rate of 6-8 ml/kg/hour is usually appropriate initially. The rate is then adjusted based on urine output (this can be readily quantified using a closed urinary collection system) and measures of hydration, especially packed cell volume and serum total solids.

If insulin was administered prior to catheterisation it is important to monitor blood **glucose** in the short-term and provide intravenous supplementation (2.5-5%) until no longer necessary.

Analgesia is continued in the form of buprenorphine (0.02 mg/kg IV q 6 hours).

Despite possible hyperkalaemia at presentation, cats with urethral obstruction may develop **hypokalaemia** following catheterisation as a result of post-obstructive diuresis and the likelihood of anorexia. Serum potassium should be monitored and supplementation provided as necessary.

Following urethral catheterisation, cats require close attentive monitoring and nursing:

- These cats are not only in a strange environment but also have been, and continue to be, subjected to multiple interventions.
- Furthermore they have at least two (intravenous, urethral) in-dwelling catheters, all of which constitutes a significant stress to the cat.
- Usual standards of care for example with respect to TLC, clean dry bedding, gentle handling, and grouping of interventions are essential.

In-dwelling urethral catheters and closed collection systems need to be checked regularly to ensure that they remain in place, are not leaking, and have not become blocked or disconnected.

Removing the urethral catheter:

In the absence of premature removal by the patient!, the urethral catheter is usually left in situ for 24-48 hours by which time azotaemia has typically fully resolved. As long as the gross appearance of the urine has improved considerably, it is not appropriate to wait for the urine to appear grossly normal before removing the catheter. It is likely that even the softest of catheters may contribute to some (albeit potentially minimal) degree of on-going mucosal trauma and haemorrhage.

Following catheter removal, it is highly recommended to keep the cat in the hospital until a normal volume of urine has been voided without obvious difficulty or discomfort. If lack of urination is thought to be behavioural, the cat may be sent home for the night with strict instructions to monitor urination and provide an update the following morning/represent the cat as necessary.

An NSAID is often administered prior to discharge.

The routine use of antimicrobial therapy is contraindicated in the presence of an in-dwelling urethral catheter and this practice is likely to select positively for more resistant bacteria. Furthermore the practice of starting antibiotics once the catheter has been removed is also to be discouraged. Urinary tract infection is relatively rare in tomcats with urethral obstruction and antibiosis should only be

commenced if a urinary tract infection has been confirmed.

Diet:

Short-term priority is to tempt to eat offering highly palatable foods rather than attempting to enforce a prescription urinary support diet
Many short-term reasons to be inappetent (hospitalisation, in-dwelling catheters, Elizabethan collar); force-feeding is both unnecessary and likely to induce food aversion.

Prognosis

With rational intensive management, even the most moribund of cats with urethral obstruction has an excellent prognosis for full short-term recovery; variable, but often good, prognosis long-term
- Euthanasia is not therefore rational early on on clinical grounds – clearly other owner- or patient-related factors may come into play

Long-term prognosis clearly depends on underlying urinary tract disorder and response to on-going management

WHAT ABOUT ATRACURIUM?

Worth trialling but how many practices stock it?

Galluzzi F, De Rensis F, Menozzi A, Spattini G. Effect of intraurethral administration of atracurium besylate in male cats with urethral plugs. J Sm Anim Pract 2012. 53(7):411-415.

Objective: To evaluate the effect of intraurethral administration of atracurium besylate for urinary obstruction resulting from urethral plugs in male cats.

Methods: Forty-five male cats were divided into the treatment group (n=25), in which 4 mL atracurium besylate solution (0·5 mg/mL) was injected into the urethral lumen, and the control group (n=20), treated with saline. All cats were then submitted to retrograde flushing until the removal of the occlusion was obtained.

Results: The percentage of cats in which the plug was removed at the first attempt was significantly (P<0·05) higher in the treatment group (64%) than in the control group (15%). Moreover, the mean (±SD) time required for the removal of the urethral obstruction was significantly shorter in the treatment group than in the control group (21·1 ±16·2 seconds versus 235·2 ±132·4 seconds; P<0·001).

Clinical Significance: The results of this study indicate that in adult male cats with urethral plugs, urethral administration of atracurium besylate increases the proportion of animals in which the obstruction is removed at the first attempt and reduces the time required to remove the urethral plugs.

Additional points from paper:

Many intravenous agents, both smooth and striated muscle relaxants, have been tried to try and relax the urethral musculature and aid relief of an obstruction. But they have limited efficacy and carry risks of side-effects due to systemic administration.

Atracurium besylate:
- A curare derivative
- Neuromuscular blocking agent via acetylcholine antagonism at the nicotinic receptors of the neuromuscular junction; it therefore causes striated muscle paralysis
- Metabolism does not depend on hepatic or renal function; it can therefore safely be given to patients with compromised renal function without an increased risk of adverse effects. (Normal metabolism: rapid inactivation by plasma esterases or by spontaneous degradation.)

This study excluded penile tip obstructions – because urethral irrigation cannot be done in these cases.

This study only included cats where the obstruction was in the post-prostatic/penile urethra – reachable by a 32 mm long catheter:
- This is because the portion of the urethra closer to the bladder is mostly under smooth muscle control on which atracurium has no effect; the distal portion on the other hand is mostly under striated muscle control.

- The distal portion is where urethral plugs are found most often anyway as the urethral lumen narrows here in the cat.

Recurrent cases were excluded

All cats received acepromazine and butorphanol as premedication and then propofol/isoflurane anaesthesia.

4 ml of a 0.5 mg/ml atracurium solution was used – this concentration and volume was empirically derived.

The process:
- Urethral obstruction established
- Cases allocated in a random non-blinded alternating fashion to the atracurium or the saline group
- The solution was then injected steadily via an intravenous catheter attached to a 5 ml syringe – injected over a 5 minute period while holding the urethral opening closed with fingers to avoid spillage
- The urethra was then flushed in a retrograde fashion for 20 seconds
- If this did not relieve the obstruction, the procedure was repeated again

Results suggest that atracurium use led to more cats being successfully catheterised at the first attempt and therefore less time for overall plug removal.

No muscle weakness or other side-effects were reported in the atracurium group

There is no mention in the paper regarding cardiovascular status, electrolytes/acid-base etc.

[Mean urethral pressure is lower in neutered versus entire male cats therefore theoretically neutered block cats in general should need less flushing and be easier to unblock; the beneficial effect of atracurium, assuming this is true, may therefore be less in neutered versus entire cats.]

Future directions:
- Other concentrations of atracurium
- Different solution volume
- Atracurium must be absorbed across the urethral mucosal surface to have this effect; understand absorption kinetics etc better.
- Try same using other agents

There were limitations in terms of how well matched the groups could be with respect to the duration of obstruction, bladder size and so on.

Summary

The most important points to remember about how to manage a tomcat with urethral obstruction are:

- The most severely affected cats are profoundly hypovolaemic and hypothermic with bradycardia and severely reduced mentation; clinically significant hyperkalaemia may be suspected on the basis of a heart rate that is inappropriately low.
- All cats with urethral obstruction will benefit from intravenous fluid therapy prior to catheterisation. An isotonic replacement crystalloid is the fluid of choice to restore perfusion and enhance urinary potassium excretion; 0.9% sodium chloride or Hartmann's (buffered lactated Ringer's solution) may be used.
- There is little rationale in performing bladder drainage before adequate fluid resuscitation.
- NSAIDs should not be administered to cats with urethral obstruction until azotaemia has fully resolved.
- Urinalysis should be routine in all cats with urethral obstruction, especially sediment examination for bacteria.
- Diagnostic imaging is indicated to exclude uroliths that require surgical removal; however it is not urgent following successful catheterisation.
- Following removal of the urethral catheter, the cat should remain hospitalised until a normal volume of urine has been voided without obvious difficulty or discomfort.
- Urinary tract infection is relatively rare in tomcats with urethral obstruction and antibiosis should only be commenced if a urinary tract infection has been confirmed.
- Cystocentesis should be reserved for those cases in which urethral catheterisation cannot be performed. However repeated cystocentesis as a short-term management strategy is very strongly discouraged – either tube cystostomy should be performed or the cat should be referred as an emergency.

Management Key Steps – Tomcat Urethral Obstruction
Shailen Jasani

Major system examination	• Especially cardiovascular for hypoperfusion • Inappropriately severe bradycardia suggests significant hyperkalaemia
IV fluid therapy	• 0.9% sodium chloride or buffered lactated Ringer's solution • 5-20 ml/kg initial bolus, repeat until perfusion normalised
Hyperkalaemia therapy	• Specific therapy if cardiovascularly significant - physical exam ± ECG • 10% calcium gluconate (0.5-1.0 ml/kg IV) stabilises myocardium - recommended in most cases • Neutral (Regular, Soluble) insulin (0.25(-0.5) IU/kg IV) + Glucose (0.25-0.5 g/kg IV) lowers potassium
Analgesia	• Pure (full) opioid preferred e.g. methadone/morphine 0.1-0.3 mg/kg slow IV - start low until cat more stable • Buprenorphine: slow onset of action • NO NSAID until hypovolaemia/dehydration and azotaemia resolved
Urethral catheterisation	• Sedate once more stable (e.g. 2 mg/kg ketamine IV + 0.2 mg/kg midazolam IV; top-up 1 mg/kg IV ketamine doses • Propofol, alphaxolone or inhalant anaesthesia may be needed - MUST be stable enough • Avoid medetomidine/dexmedetomidine
Cystocentesis - last resort	• ONLY if catheterisation unsuccuessful • Repeated cystocentesis strongly discouraged
Urinalysis	• Using first sample collected on catheterisation • Especially sediment examination for crystals and bacteria
After catheterisation	• Continue fluid therapy - usually 6 ml/kg/h initially ± 2.5-5% glucose infusion if insulin given • Buprenorphine (e.g. 0.01-0.02 mg/kg IV every 6-8 hours) • Potassium supplementation may become necessary
Catheter removal	• Usually after 24-48 hours • Ensure can urinate adequately before discharge home • NSAID (e.g. meloxicam 0.3 mg/kg SC) at discharge
Antimicrobial therapy	• NOT routinely indicated • Very low incidence of bacterial infection in blocked tomcats

Urethral catheterisation – Tomcat

Most common indication is urethral obstruction due to plugs, spasm or discrete calculi. Other indications include urethral rupture (traumatic or iatrogenic) and to monitor urine output.

Equipment

Equipment list:

- Minimum of 2 individuals usually required
- Clippers
- Surgical scrub equipment
- Sterile gloves
- Sterile lubricant
- Lidocaine 2% solution (without adrenaline)
- Feline urethral catheter – typically 3- to 5-French – see discussion below
- Lacrimal irrigation cannula or over-the-needle intravenous catheter with needle withdrawn
- Warmed 500 ml bag of 0.9% sodium chloride (normal, physiological saline)
- Fluid administration set
- Three-way tap
- 20 ml syringe
- Sterile additive-free containers
- Jug, bowl or other collection vessel
- Urine collection system

Procedure

1. Set up the fluid bag, administration set, three-way tap and 20 ml syringe
2. Generously clip around the preputial opening
3. Position the cat in lateral or dorsal recumbency
4. Perform a surgical scrub of the clipped area
5. Mix 2 mg/kg of lidocaine thoroughly with a small amount of sterile lubricant; use this to coat both the irrigation cannula and the urethral catheter
6. Extrude the penis with the non-dominant hand and evaluate its appearance; gently palpate the distal urethra for evidence of obstruction
   - If the obstruction is very distal, the tip of the penis may have a blue appearance and it may be possible to gently massage the obstructing material out
7. Gently insert the irrigation cannula or over-the-needle intravenous catheter (with the needle withdrawn to act as a stylet) into the urethra. If the cannula passes, it can be withdrawn and the urethral catheter inserted. Otherwise flushing should commence.
8. It is usually necessary to flush the urethra in order to facilitate catheter placement:
   - Inject the warmed saline in short sharp bursts with the catheter being simultaneously gently advanced in a twisting motion
   - In addition, once the catheter has passed a short distance from the external orifice, it is essential to release the penis back into the prepuce and straighten the normal bend in

the feline urethra in order for the catheter to pass further; this is achieved by pulling the prepuce caudally (and dorsally).

- NOTE: this step of flushing and advancing can take some time and progress can be slow. However with gentle handling and patience, most cats can be catheterised.

9. Once the catheter has been fully inserted, the bladder should be thoroughly lavaged with warmed saline using the fluid administration set, three-way tap and 20 ml syringe.

- The urine can be markedly haematuric and lavage is ideally continued until the fluid returning from the bladder is clear. However, this is dependent on the patient's compliance and a risk-benefit assessment of the level of chemical restraint required, e.g. if escalation to full general anaesthesia is going to be needed to allow lavage to be performed then it should probably be aborted.

10. The first urine sample obtained via the catheter prior to bladder lavage can be kept for analysis as required

11. Connect the urethral catheter to a urine drainage bag in a closed collection system. Tape the collection tubing both to the cat's tail and to the kennel subsequently in order to minimise pull on the urethral catheter.

12. Apply an Elizabethan collar to the cat to prevent interference with the urinary catheter

Great care must be taken throughout catheterisation to minimise trauma to the already inflamed and friable urethra and avoid rupture. Although it can take copious flushing and much patience to achieve catheterisation in some cases, it is almost always achievable.

Complications/Notes

Complications:

The most serious immediate complication of urethral catheterisation in obstructed tomcats is urethral rupture. Poor aseptic technique may also lead to bacterial contamination and potential infection. More long-term, trauma during catheterisation to the already inflamed urethral mucosa may potentially lead to stricture formation.

Chemical restraint for catheterisation:

In the most moribund of cats with urethral obstruction there is some debate about the 'best' approach in terms of when to perform catheterisation. Some clinicians advocate performing catheterisation as soon as fluid therapy has been commenced and emergency treatment for hyperkalaemia initiated. The rationale is that because the cat is moribund catheterisation can be performed without sedation; if this approach is used, it is still strongly recommended that analgesia be administered and allowed to take effect before catheterisation is attempted.

The alternative and preferred approach is to resuscitate the cat to the point that catheterisation cannot be performed without the need for varying degrees of chemical restraint in addition to opioid analgesia.

A combination of ketamine and midazolam can be used to good effect:
- Depending on how much methadone or morphine has already been administered, an additional dose may be given; e.g. if 0.1 mg/kg was given initially, a further 0.2 mg/kg may be given now.

- Ketamine predominantly undergoes renal excretion in cats and there is therefore the potential for this agent to accumulate in the occasional obstructed cat in which urinary drainage cannot be re-established in the short-term.
- Being a benzodiazepine, midazolam should promote urethral relaxation which is desirable.
- It is typical to start with doses of 2 mg/kg ketamine IV and 0.2 mg/kg midazolam IV; further doses of 1 mg/kg ketamine IV are given as 'top up' doses

Occasionally additional chemical restraint is required and in such cases propofol, alphaxolone or inhalant anaesthesia can be used. However it is essential to ensure the patient is as stable as possible before this.

Medetomidine should be avoided in all but the most stable of cats due to the potential for marked cardiorespiratory side effects.

Feline urethral catheters

A number of different types of feline urethral catheters are available. It is usually necessary to use a rigid catheter to unblock the urethra initially. Although softer catheters can be made stiffer by placing them in the freezer, a rigid catheter such as the **Portex® Jackson Cat Catheter** (Smiths Medical International Ltd, UK) which is widely available in the United Kingdom is typically used for unblocking. It also has the advantage of a stylet that assists with catheterisation further.

It is preferable not to leave a rigid catheter in situ as there is a greater likelihood of exacerbating urethral and bladder mucosal damage and discomfort. The Jackson catheter is also not very long and can be too short to drain the bladder reliably in bigger tomcats. The rigid catheter should ideally be removed (flushing through the catheter repeatedly as it is pulled out) and replaced with a less traumatic one.
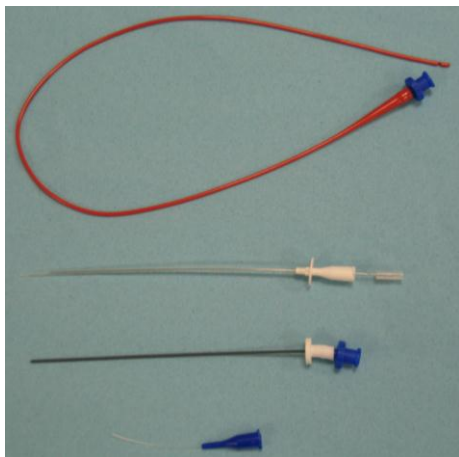
Three types of feline urethral catheter (from top to bottom): red rubber catheter; Portex® Jackson Cat Catheter; Slippery Sam®. A blue Little Herbert® adaptor is connected to the red rubber catheter and the Slippery Sam®. A lacrimal irrigating cannula is shown at the bottom.

MILA® Tomcat Urethral Catheters (from Direct Medical Supplies) have wings which can be separated from the catheter tube then repositioned at the appropriate length on the catheter once it has been measured against the cat. The two pieces are then attached together with a suture at each end of the

wings, and the wings are sutured to the prepuce using dedicated suture holes. As such it is essentially flexible in length. It is also soft and open-ended.
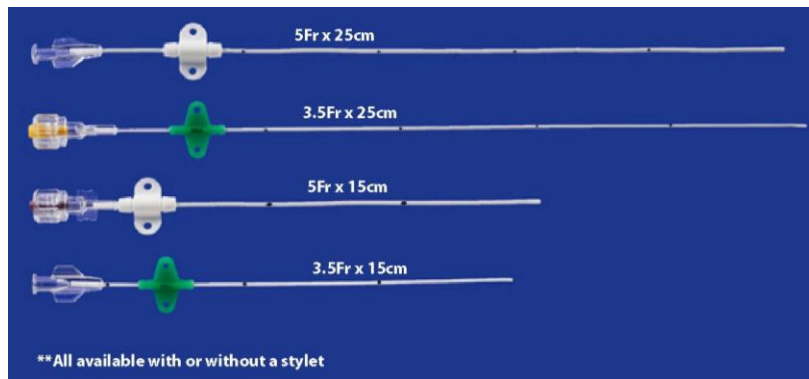
A selection of MILA® Tomcat Urethral Catheters (photograph courtesy of MILA International, Inc., Erlanger, KY USA)

MILA® Tomcat Urethral Catheter with suture wing (photograph courtesy of MILA International, Inc., Erlanger, KY USA)

3.5- or 5-French **red rubber urethral catheters** (e.g. Kendall Sovereign™, Tyco Healthcare Group LP, Massachusetts, USA) may also be used and are both soft and long. They are fixed to the preputial area using butterfly tapes.

The Slippery Sam® (SurgiVet, Smiths Medical Inc., Wisconsin, USA) urethral catheter can be readily sutured in place and connected to a collection system via a suitable luer lock adaptor (e.g. Little Herbert®, SurgiVet, Smiths Medical Inc., Wisconsin, USA). *However this catheter is not intended to be left in situ* – the warning from the manufacturer is that the catheter and silicone hub are not permanently affixed to each other and it is advised not to leave the catheter in place for more than 6 hours.
Feline urethral catheters:

Closed urinary collection system

The use of a closed collection system is highly recommended. Advantages include:
- Reduced risk of ascending infection
- Avoiding urine scalding of the perineal region
- Overall improved patient welfare

A Slippery Sam® connected to a closed system drainage set (Closed System Drainage Set (Drainset)®, Infusion Concepts, Halifax, UK) via a blue Little Herbert® adaptor.

If a proprietary urinary collection system is not available, one can be improvised using an intravenous fluid administration set attached to an empty fluid bag. The chances of success with such an improvised system can be maximised by pre-filling the administration set (as when setting up an intravenous drip) before it is connected to the urinary catheter and by minimising the amount of air that gets into the line subsequently.

If a closed collection system really cannot be made to work reliably, the urethral catheter can be bunged and the bladder can be manually drained intermittently using aseptic technique. *It is clearly essential to ensure that this is done regularly.*

If for some exceptional reason the urethral catheter must be left open, it is recommended to leave a short length of tubing connected to the catheter such that the voided urine will collect at a site away from the cat's perineum – this is better for the cat, the staff and the owners should they visit!