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1 **The approach to urethral obstruction in the cat Part 1: Presentation and stabilisation**

2

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16

17 **Background:**

18 Urethral obstruction is one of the most commonly encountered emergencies in feline
19 medicine, with a wide range of clinical signs from dysuria to peri-arrest. Such patients are
20 some of the most challenging cases in feline medicine with several factors to consider;
21 however, they can be equally rewarding.

22

23 **Aim:**

24 This is the first of three articles in this series. The aim of this first article is to summarise a
25 step wise approach from presentation through to diagnosis of urethral obstruction. The
26 second article will focus on urethral catheterisation, and immediate post-catheterisation
27 management. The third is to discuss the management behavioural and long-term
28 management needed once the cat has gone home.

29

30 **Introduction**

31 Despite being a common emergency in general practice, feline urethral obstruction remains
32 a complex condition to manage, with several controversies to consider. With their longer

1 and narrower urethra, this is predominantly a condition of male cats; however, it can
2 occasionally be seen in female cats too.

3

4 Feline urethral obstruction usually occurs as part of Feline Lower Urinary Tract Disease
5 (FLUTD); this is the umbrella term for all causes of cystitis and its associated presentations in
6 cats (Gerber *et al.* 2005). The most common causes of obstruction are urethral plugs
7 (composed of proteinaceous debris in which crystals can become trapped) and urethral
8 spasm, both of which are common consequences of feline idiopathic cystitis (FIC aka 'Stress
9 cystitis') and cause 65-90% of cases of urethral obstruction. Other causes include urolithiasis
10 (10-35% of cases), of which calcium oxalate and struvite are the most common types of
11 stones. Strictures and urethral trauma were reported in 15% of cases in a separate study
12 (Slater *et al.* 2020). Blood clots occur occasionally, as can neoplasia of the urethra, prostate
13 or bladder. Very rare cases of feline urethral obstruction are associated with significant
14 perineal or intra-pelvic pathology or trauma.

15

16 **Triage**

17 When the cat arrives with a history of unproductive straining, one of the first questions to
18 be answered is: **how stable is the patient?** This is because there is a spectrum of
19 presentations, ranging from mild cystitis-like signs to peri-arrest. A **focussed evaluation** is
20 needed to answer this question.

21

22 The key components of the triage examination in dysuric cats are:

- 23 - Mentation and whether this is appropriate for the environment
- 24 - Mucous membrane colour
- 25 - Capillary refill time
- 26 - Heart rate and rhythm
- 27 - Pulse quality and whether it is synchronous with heart rate
- 28 - Respiratory rate and lung sounds
- 29 - Abdominal palpation for bladder size
- 30 - Rectal temperature could be considered at this point if it won't cause excessive
31 stress; axillary temperatures are less likely to be reliable in a hypovolaemic cat

32

1 These parameters provide rapid information on the stability of the major body systems that
2 are likely to result in acute decompensation: the cardiovascular, respiratory and central
3 nervous systems. If these vitals do not provide cause for concern, then the full clinical
4 examination can be completed.

5

6 It should be noted that whilst bradycardia can be associated with hyperkalaemia, it is also a
7 common presentation in cats with any form of shock.

8

9 Additional information to be ascertained from a **secondary survey** once the patient has
10 been identified as stable includes:

- 11 - Hydration status
- 12 - Abdominal discomfort
- 13 - Weight, taking into account the bladder volume
- 14 - Rectal examination, if collapsed, if not then perform this once the cat is
15 sedated/anaesthetised

16

17 **Emergency diagnostics**

18 As a matter of urgency an **intravenous catheter should be placed** and secured, from which
19 blood can be withdrawn. As these patients typically have circulatory collapse once they have
20 been obstructed for a length of time, it may be necessary to use several one millilitre
21 syringes with only slight negative pressure applied. Depending on the in-house analysers
22 available, having a store of pre-coated lithium heparin syringes (such as those used for
23 arterial samples) can be helpful when there is sluggish blood flow from the catheter.

24

25 **Emergency bloodwork** is the diagnostic equivalent of physical triage, where the key
26 parameters likely to change your management in the acute period are rapidly assessed.

27

28 At our institution, emergency bloodwork would encompass the following:

- 29 - Packed cell volume
- 30 - Total solids
- 31 - Glucose
- 32 - Lactate

- 1 - Electrolytes
- 2 - Blood smear evaluation

3

4 If residual blood is available, priority should be given to evaluating renal parameters and,
5 ideally, ionised calcium concentration.

6

7 **Blood pressure** should be obtained, with Doppler measurement being the most reflective of
8 true systolic blood pressure in cats (Waddell *et al.*2004). Whilst the measured pressure
9 might not always be an accurate reflection of body wide perfusion, it can be useful to
10 provide an objective baseline to help with decision making. Blood pressure should, ideally,
11 be determined during the initial diagnostic evaluation, prior to blood sampling as this may
12 worsen situational hypertension ('white coat effect'). However, this phenomenon is unlikely
13 to cause interference in collapsed cats as sympathetic drive should already be maximal. A
14 systolic blood pressure below 90 mmHg is consistent with a mean arterial pressure below 65
15 mmHg, at which point renal perfusion is inconsistent and fluid resuscitation is urgently
16 required. Hypotensive cats carry a more guarded prognosis.

17

18 **A point of care ultrasound (POCUS) examination** is very useful in these patients. It can be
19 performed without the need for extensive clipping of the coat and does not require
20 advanced equipment or skill. Cover the probe with a glove or cling film containing
21 ultrasound gel, then spray spirit into the parted hair to achieve a decent acoustic window
22 whilst also protecting the probe.

23

24 The aim of the POCUS is not to evaluate the urogenital system in great detail, but instead to
25 evaluate whether there is free fluid within the abdomen, and whether or not the urinary
26 bladder appears intact. In feline urethral obstruction, there is often a small amount of free
27 abdominal fluid, although not typically detected, larger volumes are expected with rupture
28 along the urinary tract and subsequent uroabdomen (Figure 1)

29

30 To aid identification of small volumes of free abdominal fluid, POCUS involves focusing on
31 four main areas in the abdomen:

- 32 - Between the liver lobes and diaphragm

- 1 - Between the spleen and left kidney
- 2 - Between the liver and right kidney
- 3 - Around the bladder

4

5 Whilst most patients presenting with urethral obstruction are typically young male cats that
6 are otherwise healthy and capable of tolerating fluid administration, in older patients or
7 those with pre-existing heart disease, it is sensible to evaluate the heart via ultrasound
8 before giving intravenous (IV) fluids. Scanning the cat from the right side of the chest, over
9 the apex beat, allows the **left atrial to aorta (LA:Ao) ratio** to be measured (Figure 2). Cats
10 with urethral obstruction are usually hypovolaemic, so the ratio will be low, whereas cats
11 with ongoing heart disease are likely to have left atrial enlargement, so the ratio may be
12 increased. A ratio greater than 1.6 is consistent with enlargement of the left atrium, and
13 caution should be exercised with any fluid therapy.

14

15 With the triage examination performed and these diagnostic results obtained, a wealth of
16 information is rapidly available to aid in clinical decision making.

17

18 **ECG** can be used in these patients if available. This has the benefit of allowing a continued
19 recording of the heart rate. It can also be useful in evaluating the effect of hyperkalaemia
20 (see below). Whilst consistent ECG findings and bradycardia can be a specific finding for
21 hyperkalaemia, their absence does not exclude it and this should not be solely relied upon.

22

23 **Stabilisation**

24 **Analgesia**

25 One of the first aspects to be addressed is the provision of analgesia. Cats with urethral
26 obstruction are incredibly painful secondary to severe bladder distension, and some may
27 also have pain from preceding inflammation of the bladder wall. Interpreting clinical
28 parameters and making fluid therapy decisions is difficult when pain is stimulating the
29 sympathetic drive, manifesting as raised blood pressure, heart and respiratory rates.

30

31 Cats with urethral obstruction are often unstable when first presented, making it difficult to
32 know whether or not surgical intervention needs to be considered. A full opioid, which

1 should maximally stimulate and bind to the receptors, is therefore advised, if possible.
2 Methadone (0.2 mg/kg IV) is a reasonable option, which leaves potential for increasing the
3 dose, if necessary. Whilst buprenorphine is very effective in cats, the concern is that if the
4 analgesia proves inadequate and a more potent opioid becomes necessary, the
5 agonist/antagonist effect may impede the full opioid effect for six to eight hours.

6

7 Maropitant may have some visceral analgesic properties and could be considered in
8 conjunction with the proven analgesics above, particularly as these patients are likely to
9 experience nausea associated with the shock state.

10

11 Non-steroidal anti-inflammatory medications (NSAIDs), whilst often a mainstay in these
12 patients when stable, should be avoided at presentation. Renal and gastrointestinal
13 perfusion is likely to be inconsistent due to hypovolaemia and if anaesthesia were to
14 become necessary then vasodilation and worsening hypotension is a possibility, which could
15 add further insult to already injured kidneys.

16

17 **Fluid therapy**

18 This is an area in which several controversies exist. The first is ***Whether to give fluids or***
19 ***not?*** Whilst it would seem counter-intuitive to provide more fluid when there is obstruction
20 of urine output, this is a necessity in order to stabilise the patient. The fluid will help to
21 restore renal perfusion and dilute the plasma potassium.

22

23 The next question is ***Which fluid type should be used?*** An isotonic crystalloid is most often
24 selected, and the decision is then whether to use 0.9% sodium chloride or Hartmann's
25 (lactated Ringer's). The theoretical advantage of saline is that it includes no additional
26 potassium, so it should address the hyperkalaemia more effectively. The disadvantage is
27 that it has an acidifying effect, whereas Hartmann's is alkalising, which is relevant in these
28 patients because they are likely to be acidaemic. However, the evidence indicates that the
29 type of isotonic crystalloid utilised has no significant difference in prognosis or the ability to
30 correct the hyperkalaemia. The only difference was that those patients receiving
31 Hartmann's normalised their acid-base disturbances more quickly (Cunha *et al.* 2010). This is
32 because improved renal perfusion allows for greater excretion of potassium into the urine,

1 in excess of the potassium accumulated from the fluid therapy. As such, Hartmann's is
2 typically the first line fluid choice in our hospital.

3

4 The next question to be addressed is ***What volume of fluid should be administered?*** This
5 can be answered by deciding what we are trying to address. These patients are likely to be
6 dehydrated, at least to some degree, with a loss of fluid in the interstitial space; however,
7 this is not a major concern in the acute setting. Dehydration is not life threatening in itself,
8 however, it does become so when it leads to loss of fluid from the intravascular space
9 causing hypovolaemia with resultant kidney hypoperfusion. This gives rise to azotaemia and
10 an inability to excrete potassium. This is the problem that must be targeted first.

11

12 Administering rapid intravenous shock doses of fluids, which is the cat's blood volume (50
13 ml/kg), was historically recommended in patients with hypovolaemia and shock. However,
14 this can result in fluid overload with associated complications, especially in cats. Fluid
15 overload can manifest as chemosis, serous nasal discharge and pulmonary oedema or
16 pleural effusions secondary to cardiac overload. The approach has now shifted to dividing
17 the shock dose of fluids and giving aliquots of it over a short space of time to restore
18 intravascular volume. Boluses for hypovolaemic cats usually consist of 5-10 ml/kg being
19 administered over 10-15 minutes, depending on how haemodynamically stable the patient
20 is. In those with severe hypovolaemia, larger volumes can be administered.

21

22 After each bolus, the patient is then re-evaluated for improvement in perfusion parameters:

- 23 - Mentation
- 24 - Heart rate
- 25 - Pulse quality
- 26 - Mucous membrane colour
- 27 - Capillary refill time
- 28 - Blood pressure

29

30 Lactate can also be measured periodically as an objective marker of increased perfusion, if
31 available. Approximately a quarter of the bolus will stay in the intravascular space, with the
32 remainder redistributing to the interstitial space. The amount which is drawn to the

1 extravascular space may increase with dehydration. This can manifest as an initial response
2 to the fluid bolus, then recurrence of clinical signs of hypoperfusion after 30 - 60 minutes as
3 the fluid leaves the intravascular space. Once the perfusion parameters have normalised,
4 the fluid rate can be set to replace the degree of dehydration, to address ongoing losses,
5 and provide maintenance fluids. If there are no signs of dehydration, such as dry mucous
6 membranes, prolonged skin tent or sunken eyes, then subclinical dehydration of 5% can be
7 assumed.

8

9 **Hyperkalaemia**

10 **Pathophysiology**

11 Hyperkalaemia, and its resultant cardiotoxic effect, is the immediate concern in these
12 patients. Understanding the mechanism by which elevated potassium causes these
13 deleterious effects can be helpful in appreciating the treatment options.

14

15 Potassium is essential for myocardial cells to generate and transmit an action potential. The
16 action potential is generated, and the myocardial cell is depolarized by the influx of positive
17 Na^+ ions from the extracellular space to the negative inside of the cell membrane. Once the
18 cell is depolarized, it is refractory to the generation of further action potentials. To restore
19 negativity to the inside of the cell, and allow future action potentials to occur, the sodium-
20 potassium-ATPase pump pushes potassium ions (K^+) out of the cell and the positive charge
21 with it.

22

23 In states of hyperkalaemia, the potassium concentration across the cell membrane is no
24 longer present to the same degree as normal. This reduces the amount of potassium that is
25 ejected from the cell, and consequently the inner cell membrane stays too positive and
26 remains refractory. In effect, the resting potential (or baseline cell polarity) is elevated and
27 more positive. With the cells unable to repolarize, the clinical manifestation of this is
28 bradycardia or atrial standstill if severe.

29

30 This process is summarised in Figure 3.

31

1 **Diagnosis**

2 Definitively diagnosing hyperkalaemia is reliant upon measuring serum electrolyte
3 concentrations. The degree of increase can be summarised as:

- 4 - Mild > 5.5 mmol/l
- 5 - Moderate > 6.5 mmol/l
- 6 - Severe > 7.5 mmol/l

7

8 Myocardial toxicity is possible once the serum potassium concentration is above 6.0 mmol/l;
9 however, the likelihood will vary depending on other factors, particularly the serum ionised
10 calcium concentration.

11

12 ***Hypocalcaemia exacerbates the effect of the hyperkalaemia*** by decreasing the threshold
13 potential, meaning the initial action potential is generated easily, but repolarising the cell in
14 the face of hyperkalaemia becomes more difficult if not impossible. It is therefore important
15 to assess blood calcium concentration in these cases. Ionised hypocalcaemia is present in
16 75% of cats with urethral obstruction; its severity correlates with more serious
17 cardiovascular compromise and a poorer prognosis (Drobatz *et al.* 1997). This is summarised
18 in Figure 3l.

19

20 Surrogate markers of hyperkalaemia include bradycardia identified on clinical examination
21 and electrocardiogram (ECG) alterations. ECG findings suggestive of hyperkalaemia include
22 sinus bradycardia, which can progress to an atrioventricular block or atrial standstill if
23 severe. Other features of the ECG include an absence of P waves, widening of the QRS
24 complex and the classically reported tenting of the T waves. As the hyperkalaemia becomes
25 severe, it can present as a sine wave. An example of this is shown in Figure 4, in which P
26 waves are absent and T waves spiked.

27

28

29 **Treatment**

30 Treatments, dosages and important notes are summarised in Table 1.

31

Calcium gluconate 10%	0.5 – 1.5 ml/kg	<ul style="list-style-type: none"> • Ideally give over 20 – 30 minutes, but can be given over 5 minutes if necessary • Monitor with ECG or auscultation • Lasts for 20 minutes
Glucose	<p>0.5 g/kg if used alone for mild hyperkalaemia</p> <p>2 g per unit of insulin given as a bolus followed by a glucose infusion</p>	<ul style="list-style-type: none"> • Dilute 1:4 with 0.9% sodium chloride to minimise risk of phlebitis
Neutral insulin	0.25 – 0.5 IU/kg	<ul style="list-style-type: none"> • Blood glucose should be monitored for up to 24 hours after • Glucose CRI will likely necessary
Terbutaline	0.01 mg/kg	<ul style="list-style-type: none"> • Give IV slowly • Tachycardia may occur • Variable efficacy • This is of theoretical benefit, with no published studies on its clinical use; its use would be off licence
Sodium bicarbonate	1 – 2 mEq/kg	<ul style="list-style-type: none"> • Rarely necessary, and other measures attempted first to address pH.

		<ul style="list-style-type: none">• Has numerous side effects that should be taken into account.
--	--	--

1 CRI – constant rate infusion

2

3 **Calcium gluconate**

4 This is the first line medication to treat life-threatening bradycardia associated with
5 hyperkalaemia. Calcium gluconate will alter the electrical charges of the cell membrane,
6 which increases the threshold potential, and thus redresses the balance of the resting and
7 threshold potentials needed to restore normal electrical activity. Calcium gluconate will not
8 alter the serum potassium concentration, but will act as a cardioprotectant providing time
9 to institute other methods to address the hyperkalaemia.

10

11 Calcium gluconate 10% is dosed at 0.5 – 1.5 ml/kg (equating to 50 – 150 mg/kg of calcium
12 gluconate). It should be given by slow IV, over 20 minutes. Ideally an ECG should be in place
13 whilst the calcium is being infused, or if unavailable then monitoring the heart rate with
14 auscultation is an alternative. Potential side effects include bradycardia and ECG changes
15 including sinus bradycardia, shortening of the QT interval and S-T elevation.

16

17 The beneficial effects of calcium are relatively short lived, lasting from 20 to 30 minutes,
18 providing time to address the hyperkalaemia directly by removing the urethral obstruction.

19

20 **Fluid bolus**

21 As discussed above, a fluid bolus will have several beneficial effects. By restoring renal
22 perfusion and glomerular filtration, the surplus potassium can be filtered and excreted into
23 the urine. Additionally, acidosis promotes the movement of potassium from the intracellular
24 to extracellular space. Fluid therapy, particularly with Hartmann’s solution, will address the
25 acidaemia and cause a shift of potassium back into the intracellular space.

26

27 **Glucose and insulin**

28 If the hyperkalaemia is severe, if it is causing a significant bradycardia, or the potassium
29 levels have not improved with fluid therapy, then glucose and insulin is indicated. The

1 insulin causes the glucose to be taken up into the cell, and this takes potassium ions with it,
2 driving an intracellular shift. Glucose alone may help stimulate endogenous insulin release;
3 however, exogenous insulin will facilitate this more rapidly. Whilst this will not reduce the
4 body wide potassium levels, it will decrease serum levels, sparing the myocardium of the
5 deleterious effects.

6

7 Regular (soluble) insulin is dosed at 0.25 – 0.5 units/kg IV as a slow bolus.

8

9 Dextrose is then administered at 4ml of 50% dextrose per unit of insulin given. However, it
10 should be noted that 50% dextrose has high osmolarity and will cause phlebitis if given into
11 a peripheral vein at this concentration. It should therefore be diluted with 0.9% saline to a
12 concentration of 1:4.

13

14 There is the risk of hypoglycaemia following the insulin administration, even when a glucose
15 bolus is also given. Frequent monitoring of the blood glucose concentration is essential, and
16 a dextrose infusion is likely to be needed. If necessary, a 2.5% dextrose infusion can be
17 made by adding 50ml of 50% dextrose (25 g dextrose) to 1 litre of fluid; this is then given as
18 part of the fluid plan in the hours that follow.

19

20 **Terbutaline**

21 This is a β_2 agonist, which is often used as a bronchodilator in emergency situations. It also
22 has the additional benefit of stimulating the Na-K-ATPase on cell membranes, with the
23 resultant effect that potassium is driven intracellularly, thus decreasing the circulating
24 hyperkalaemia. Side effects can include tachycardia by stimulating the adrenergic receptors
25 on the myocardial cells.

26

27 **Sodium bicarbonate**

28 If the cat is severely acidotic and hyperkalaemic, then bicarbonate can be used to correct
29 the acidosis and drive the potassium intracellularly. However, this is rarely necessary, as
30 fluid therapy alone is usually sufficient to correct this. In addition, there are side effects
31 associated with sodium bicarbonate administration which could pose serious risks, such as
32 ionised hypocalcaemia, hypernatraemia and central acidosis.

1

2 **Cystocentesis**

3 Another controversy in the management of cats with urethral obstruction is **Whether to**
4 **perform cystocentesis or not?** Whilst the azotaemia that occurs in these cats is multi-
5 factorial, the major contribution is post-renal in origin. With the urethra obstructed the
6 bladder becomes excessively distended, causing back pressure via the ureters to the renal
7 pelves. At a microscopic level the pressure in the nephrons is markedly elevated, which
8 opposes glomerular filtration, resulting in the potassium and uraemic toxins remaining in
9 the circulation rather than getting into the filtrate.

10

11 The plan should be to stabilise and anaesthetise the patient as quickly as possible, so that a
12 urinary catheter can be placed. However, in some patients, significant pressure within the
13 bladder may confound efforts to pass a urinary catheter, and the ongoing bladder
14 distension and metabolic disturbances become the life-threatening issue. Cystocentesis may
15 then be needed to relieve bladder pressure and facilitate urinary catheter placement.

16

17 Unless the cat is so severely collapsed that wriggling is unlikely, it is best to perform
18 cystocentesis once the cat is sedated or anaesthetised. This reduces the risks of performing
19 this procedure on a severely distended bladder. The main risks are bladder rupture, or
20 laceration of a vessel resulting in a haemoabdomen. **Some degree of uroabdomen is always**
21 **likely;** however, it is unlikely to cause a problem unless the cat has a concurrent urinary
22 tract infection (UTI). Cats with FIC are at particular risk of uroabdomen as their bladder
23 urothelium and detrusor muscle are badly compromised by their underlying disease.
24 Vagovagal collapse has been reported to occur in cats and can be fatal (Odunayo *et al.*
25 2015). Whilst these risks are rare they must be communicated to the owner.

26

27 If cystocentesis is to be performed, some strategies can minimise the risk of iatrogenic
28 damage. Ideally, this is performed with two people, using the same equipment as would be
29 required for a thoracocentesis. One person inserts the needle or butterfly catheter into the
30 bladder, with ultrasound guidance if necessary. This person holds the needle stable and the
31 second person holds the butterfly catheter line, or additional tubing, connected to a three-
32 way tap and syringe. With the initial person focussing on keeping the needle steady, this

1 stops it moving unnecessarily within the bladder, preventing microtrauma or laceration of
2 the bladder wall. The second person then drains the bladder until it is empty. This removes
3 the pressure component and minimises the risk of urine leakage from the bladder. The urine
4 can be collected and stored for analysis and culture if indicated.

5
6 If uroabdomen does develop, while less than ideal, it is still preferable to ongoing
7 obstruction and secondary hyperkalaemia in a cat that is unable to be catheterised. Once a
8 urinary catheter is placed, this will keep the bladder decompressed, in the hope that any
9 bladder defect will close and heal, and even sizeable tears can heal when managed correctly
10 in this way. The abdominal effusion can be removed, if significant, by abdominocentesis to
11 decrease the potassium load. Severe cases of uroabdomen will need to be referred for more
12 extensive management.

13
14 One published study assessed intermittent cystocentesis instead of urethral catheter
15 placement where there were severe financial limitations (Cooper *et al.* 2010). Cats had
16 cystocentesis performed up to 10 times; four out of 15 cats developed abdominal effusion,
17 of which three had uroabdomen and one had haemoabdomen. Complications can therefore
18 be severe, but this risky and sub-optimal approach could be discussed with owners where
19 funds are severely constrained.

20

21 **CPD quiz**

22

23 **What are the most common causes of urethral obstruction?**

- 24 A. Uroliths and neoplasia
25 **B. Urethral spasm and proteinaceous plugs**
26 C. Uroliths and urethral spasm
27 D. Proteinaceous plugs and uroliths

28

29 **What is the left atrium to aorta ratio (LA:Ao), above which you would need to consider**
30 **how aggressively you would administer fluid therapy?**

- 31 A. < 1
32 B. < 1.3

1 C. < 1.6

2 D. < 2

3

4 **Which of the drugs commonly used in the emergency management of hyperkalaemia will**
5 **not alter the serum potassium concentration?**

6 A. Calcium Gluconate

7 B. Glucose

8 C. Insulin

9 D. Sodium bicarbonate

10

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