Other Common Emergencies



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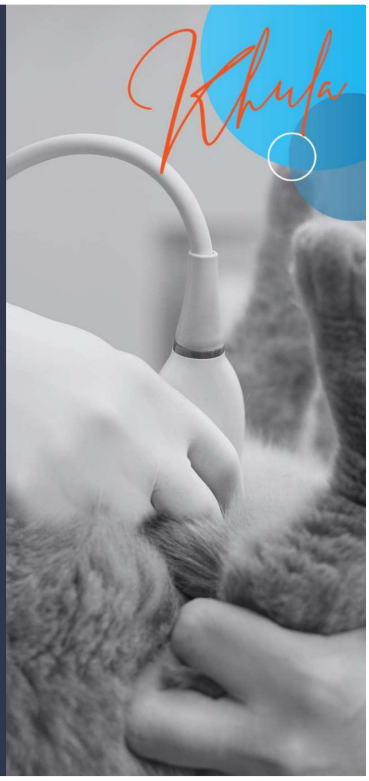
Other Common Emergencies

1. *Gastric Dilatation and Volvulus*
2. *Intoxications*
3. *Snake Bites*
4. *Urethral Obstruction*



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INTRODUCTION



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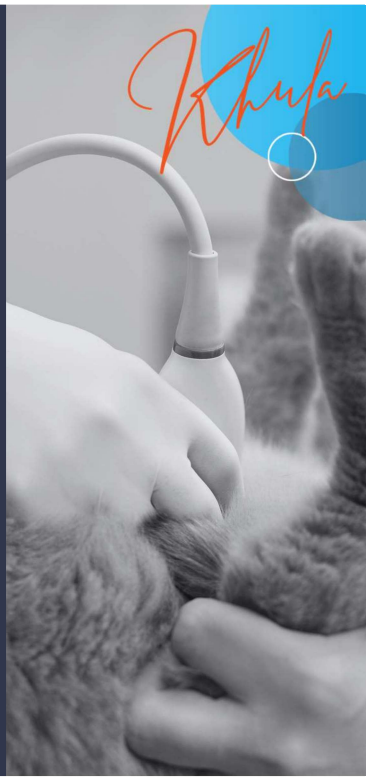


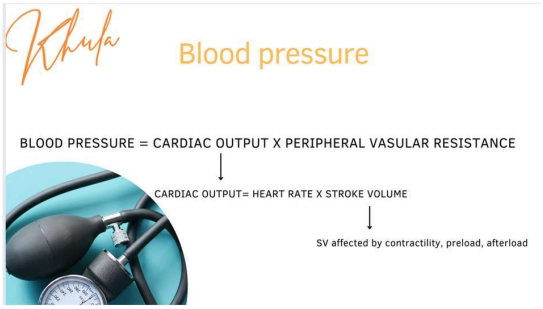
GDV

- Enlargement of the stomach associated with rotation on its mesenteric axis aka gastric torsion or bloat
- Simple dilatation refers to a stomach that is engorged with air or froth but not malpositioned



- **Dilatation** = condition in which an organ or structure is stretched beyond its normal dimensions
- **Dilation** = the act of stretching a cavity or orifice

Clinical signs and exam findings



- The distended stomach compresses the caudal vena cava and portal vein which reduces venous return (pre-load) and subsequently stroke volume and cardiac output + MAP >> myocardial ischaemia
- Obstructive shock and inadequate tissue perfusion affects multiple organs, including the kidneys, heart, pancreas, stomach, and small intestine

Clinical signs and exam findings

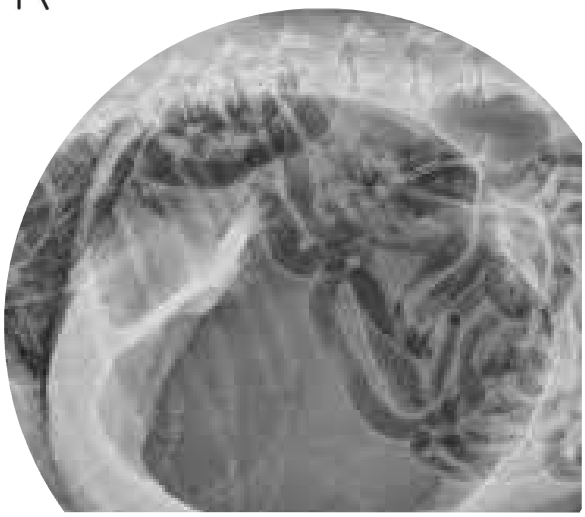
- Progressively distending and tympanic abdomen
- Pain
- Arched back
- Nonproductive retching
- Hyper-salivation
- Restlessness
- Splenomegaly
- Weak peripheral pulses
- Prolonged capillary refill time
- Pale mucous membranes
- Tachycardia
- Arrhythmias (more common with gastric necrosis)



X-rays



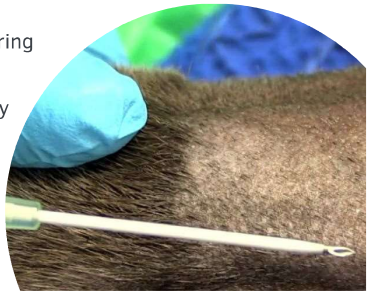
- Radiographs:
  - Radiographs are necessary to differentiate simple dilatation from dilatation plus volvulus
  - Right lateral and dorsoventral views facilitate filling the abnormally displaced pylorus with air so that it can be easily identified
    - Positioning these animals for a ventrodorsal view may lead to aspiration/decompensation
  - On a right lateral view of a dog with GDV
    - The pylorus lies cranial to the body of the stomach
    - Separated from the rest of the stomach by soft tissue (“reverse C sign” or “double bubble”)
  - Free abdominal air suggests gastric rupture
  - Air within the wall of the stomach indicates necrosis



Medical Management

Stabilising the patient's condition is the initial objective

- **Oxygen** therapy
- One or more large-bore intravenous catheters should be placed in either a jugular or both cephalic veins
  - Shock rate fluid (See module 4 part 1)
- **Pain control**
- Broad-spectrum **antibiotics**
- Collecting **bloods** for baseline testing
  - PCV/TS (CBC), biochem, LYTES, blood gas, lactate (see next slide), PT/PTT
- **Decompress stomach** as much as possible (see next slide)
  - Enlarged stomach may hinder respiration and make it difficult for the animal to ventilate during induction of anesthesia. It also impairs venous return.
- **Electrolyte** and acid-base abnormalities should be corrected as much as possible before surgery
  - After shock dose fluid therapy
- ECG should be performed to detect **cardiac arrhythmias**
  - Treat with lidocaine before surgery if they are significant > long runs of ventricular tachycardia which are affecting cardiac output



Plasma lactate

Prognostic value:

Initial and serial plasma lactate concentrations can be used to guide decision making in individual dogs

Pre-operative measurement of plasma lactate

- <4 mmol/L - Survival is more likely and the chance of complications less
- >6 mmol/L - gastric necrosis and greater expense more likely
  - Dogs with gastric necrosis are approximately 10 times more likely to die than those that do not have gastric necrosis.

Serial monitoring

- Falls in plasma lactate of greater than ~40% after fluid resuscitation are likely to indicate better survival
- Many dogs with persistent hyperlactatemia over 24-48 hours do not survive

Exploratory laparotomy should always be recommended irrespective of the plasma lactate concentration


Topics in Companion Animal Medicine
Volume 29, Issue 3, September 2014, Pages 71-76

Topical Review

Plasma Lactate Concentration as a Prognostic Biomarker in Dogs With Gastric Dilation and Volvulus

Erin Mooney BVSc (Hons), Dip. ACVECC [a], Cameron Raw BVSc (Hons) [b], Dez Hughes BVSc (Hons), MRCVS, Dip. ACVECC [a]

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Gastric decompression

- Should be performed while shock therapy is initiated

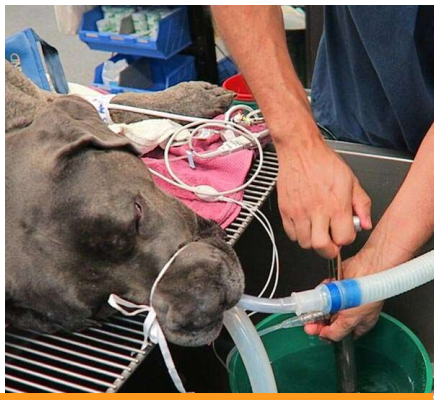
- The stomach may be decompressed with
  - Nasogastric tube
  - Percutaneously with large-bore intravenous catheters
  - Small trocar
  - Stomach tube



Stomach tube

- Place ET tube with cuff (prevent aspiration)
- The stomach tube should be measured from the point of the nose to the xiphoid process and a piece of tape applied to the tube to mark the correct length
- Lubricate the tube
- Pass the tube to the measured point by placing the animal in different positions
  - Never force the tube, can cause oephageal perforation
  - If struggling to pass the tube, perform percutaneous decompression of the stomach first as this may relieve pressure on the cardia and allow the tube to enter the stomach
- Remove air and then flush the stomach with warm water
  - Failure to lavage the stomach usually results in rapid re-dilatation after the tube is withdrawn
  - If blood is seen in the fluid from the stomach, prompt surgical intervention is warranted because this may indicate gastric necrosis

If the stomach tube still cannot be passed then surgery to manually decompress should be performed



Note: You cannot differentiate GDV from gastric dilatation without volvulus simply because you are able to pass a stomach tube. Stomach tubes frequently can be passed in dogs with twisted stomachs

Surgical Treatment

- Surgery should be performed as soon as the animal's condition has been stabilised, even if the stomach has been decompressed
- Rotation of an undistended stomach interferes with gastric blood flow and may potentiate gastric necrosis

The goals of surgical treatment

- Decompress the stomach and correct any malpositioning
- Inspect the stomach and spleen- identify and remove damaged or necrotic tissue
- Perform a gastropexy to prevent/reduce the chance of subsequent GDV



Anaesthesia

- Numerous anaesthetic protocols, personal choice
- Suggestions
  - Methadone and diazepam/midazolam as pre-medications
  - Propofol/alfaxan to induce
  - Maintenance Isoflurane or sevoflurane

Surgical Treatment

Surgical prep

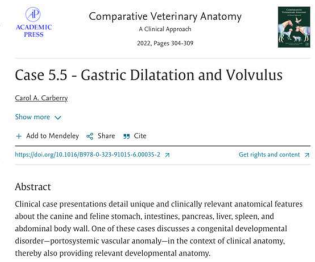
- Try and do as much prep before putting dog under GA
- Putting the dog on its back can cause respiratory issues and decrease venous return further- work quickly
- When ready induce and place in dorsal recumbency
- Abdomen is prepared for a midline abdominal incision
  - Mid-thorax to the pubis

Top tips:

- Prepare **emergency drugs** before and work out CRI for dopamine/norepi incase of intra-op hypotension
- Caution with deep chested dogs and diaphragm attachment
- **Lidocaine CRI** can be very useful in these patients: 2 mg/kg (IV bolus) followed by constant rate infusion (CRI) of 50µg/kg/minute. Not only will it help with intra-op pain control but some studies suggest that it can decrease the occurrence of cardiac arrhythmias, AKI and hospitalisation in GDV patients. It also acts as a scavenger of reactive oxygen species and as an inflammatory modulator and counteracts several pathways in reperfusion injury.



Antatomy


Comparative Veterinary Anatomy
A Clinical Approach
2022, Pages 304-309

Case 5.5 - Gastric Dilatation and Volvulus

Carol A. Carberry

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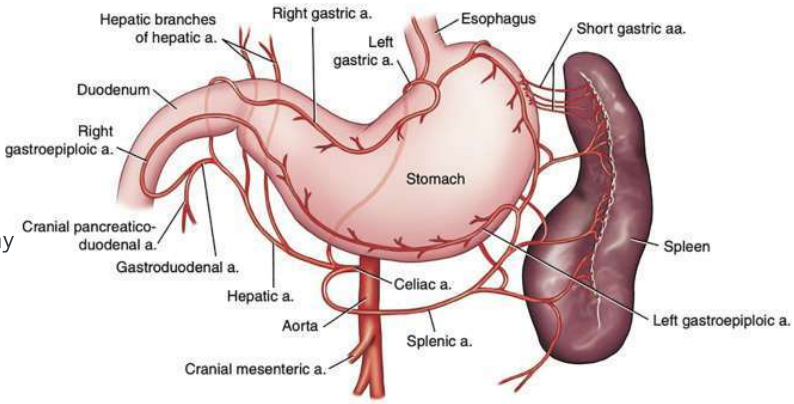
Abstract

Clinical case presentations detail unique and clinically relevant anatomical features about the canine and feline stomach, intestines, pancreas, liver, spleen, and abdominal body wall. One of these cases discusses a congenital developmental disorder—portosystemic vascular anomaly—in the context of clinical anatomy, thereby also providing relevant developmental anatomy.

Blood supply to the stomach

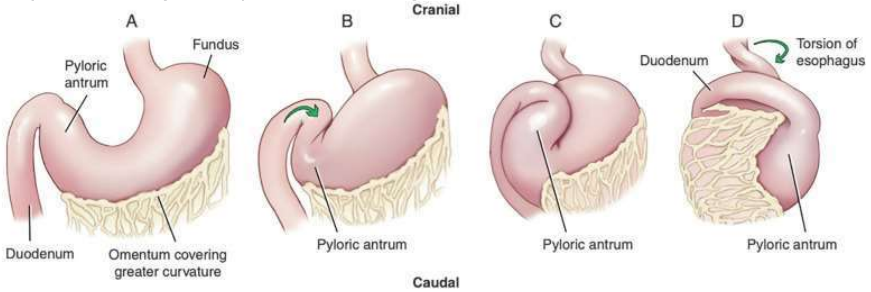
- The celiac artery give rise to:
  - **Gastric artery:** the lesser curvature
  - **Gastroepiploic artery:** the greater curvature

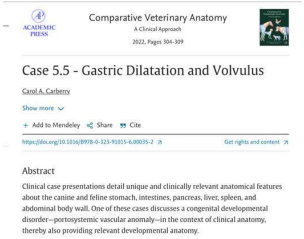
- The splenic artery give rise to:
  - **Short gastric arteries:** the greater curvature
    - Rupture of the short gastric arteries in dogs with GDV is common and may contribute to blood loss and gastric infarction or necrosis



Antatomy

- Greater omentum arises from the greater curvature of the stomach and covers the intestines
- Stomach rotates in a clockwise direction when viewed from the surgeon's perspective
  - With the dog on its back and the clinician standing at the dog's side, facing cranially
  - The rotation may be 90 to 360 degrees (usually is 220 to 270 degrees)
  - The duodenum and pylorus move ventrally and to the left of the midline and become displaced between the esophagus and stomach.
- The spleen usually is displaced to the right





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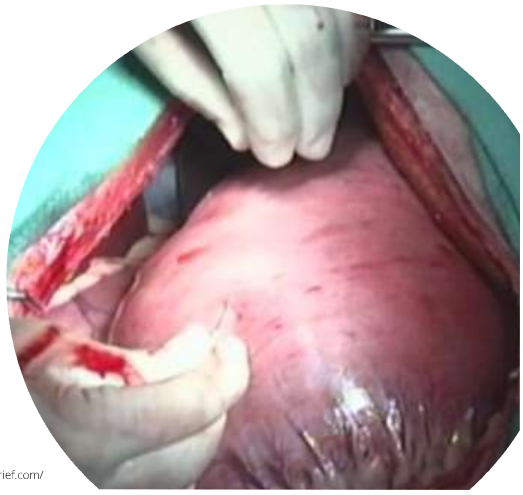
Surgical Treatment

- Upon entering the abdominal cavity the first structure noted is the greater omentum, which usually covers the dilated stomach

- Decompress the stomach before repositioning
  - By using a large-bore needle/catheter (14/16G) attached to suction/large syringe
- Pass an orogastric stomach tube
  - Intra-operative manipulation of the cardia usually allows the tube to be passed into the stomach without difficulty
- If adequate decompression is still not achieved a small gastrotomy incision can be performed to remove the gastric contents (avoid spillage)



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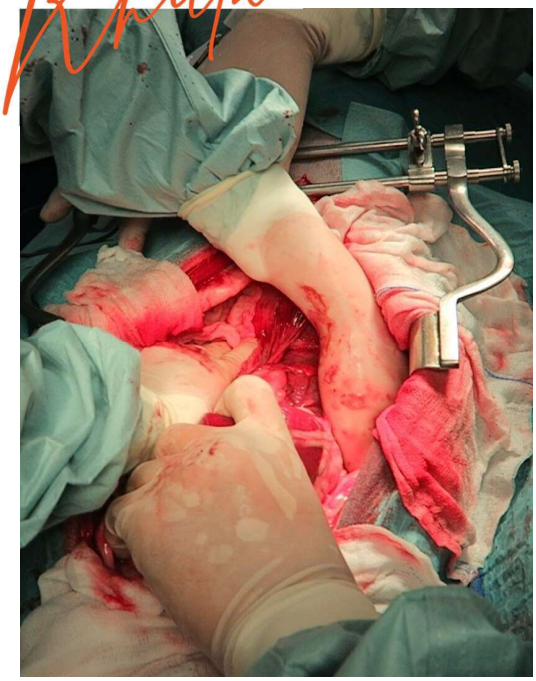


Derotation

For a clockwise rotation
- Decompress stomach first
- Rotate it counterclockwise:
  - Grasp the pylorus (usually found below the oesophagus, left body wall) with the right hand and the greater curvature with the left
  - Push the greater curvature of the stomach toward the table with left hand while simultaneously elevating the pylorus towards the incision with right hand
- Gastric lavage (funnel, warm water, bucket)

Spleen/Stomach

Assess the spleen:

- Check to make sure the spleen is normally positioned in the left abdominal quadrant
- Evaluate the spleen after derotation and allow time for any congestion to resolve before deciding on splenectomy
- If there is splenic necrosis (often at the head) or significant infarction, perform splenectomy
- Confirm the gastrosplenic ligament (short gastric arteries) is not torsed

Assess the stomach for necrosis

- Remove or invaginate necrotic gastric tissue
  - If you are uncertain whether gastric tissue will remain viable, invaginate the abnormal tissue
  - Avoid entering the gastric lumen if possible
- Palpate the intraabdominal esophagus to ensure that the stomach is derotated

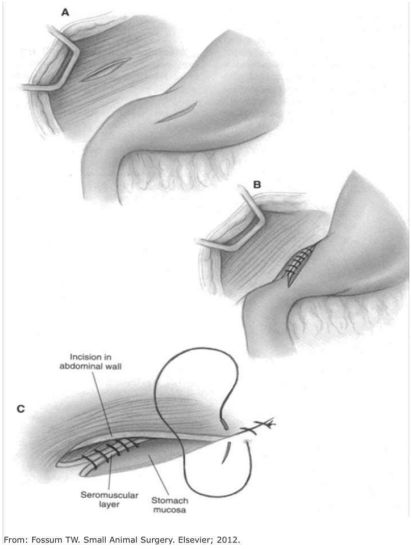
Gastropexy

Perform a permanent gastropexy

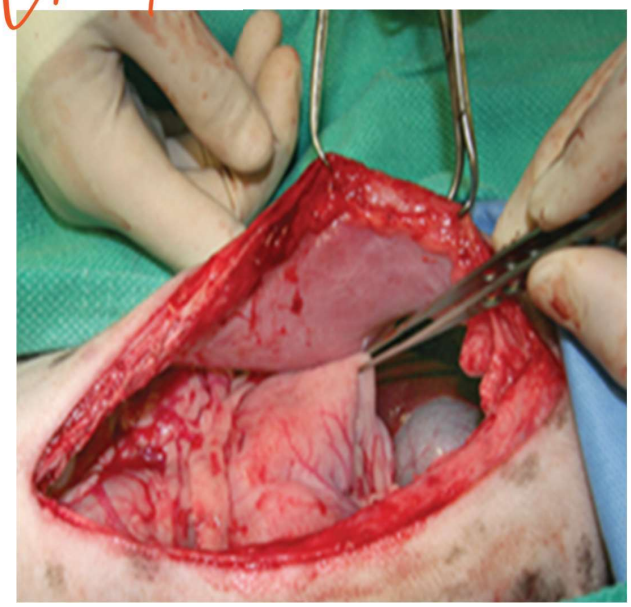
- Does not guarantee that dilatation or volvulus will not recur but makes it less likely
- Gastropexy should always be performed in conjunction with abdominal exploration and derotation of the stomach
- Absorbable (polydioxanone or polyglyconate) or non-absorbable (polypropylene) suture material may be used for the gastropexy (0 or 2–0)

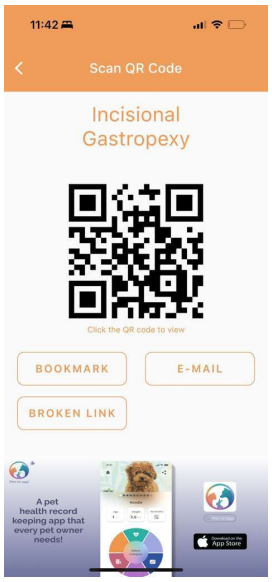
Procedure incisional gastropexy

- Incise stomach (NOT full thickness)
- Incise R body wall
- 2 lines stitches
- Simple continuous pattern
- Knot quality



From: Fossum TW. Small Animal Surgery. Elsevier; 2012.





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Post-operative care

- Supportive care: anti-emetics, pain control (NSAID?), fluid therapy, GIT protectants
- NGT placement and feeding started when patient is fully awake
- Monitor electrolytes and acid-base/blood gas
- Monitor PCV/TS, PT/PTT, Albumin
- Lactate
- ECG
  - Ventricular arrhythmias are common in dogs with GDV and usually begin 12 to 36 hours after surgery
  - Treatment of cardiac arrhythmias includes maintenance of normal hydration and correction of electrolyte imbalances.
  - Some anti-arrhythmic drugs (i.e., lidocaine) are ineffective when the animal is hypokalaemic
  - Arrhythmias require treatment if they interfere with cardiac output – poor peripheral pulses, prolonged CRT, heart rate consistently > 160-180bpm
  - Dose:
    - A test bolus of lidocaine given intravenously (2 mg/kg bolus) can be used to determine responsiveness to this drug
    - Can repeat 1-3 boluses but no more than 6-8 mg/kg of lidocaine over 15 min to prevent toxicity
    - If the arrhythmias diminish or stop with bolus, lidocaine should be given by a continuous intravenous infusion (start at 50µg/kg/minute and titrate up)
  - Lidocaine toxicity include muscle tremors, vomiting, seizures, hypotension- STOP if seen
    - Half life is 15-30 min

Complications

- Sepsis and peritonitis may be caused by gastric necrosis or perforation if devitalised tissue is not adequately removed.
  - Placing and abdominal drain (Jackson Pratt) allows for diagnostic peritoneal lavage
- Disseminated intravascular coagulation- DIC (16%of cases)
  - Assessment of clotting parameters + PLT counts (D-dimers/FDP?/TEG)
  - Treat with plasma
- Cardiac arrhythmias are common in dogs with GDV (45.5%)

- Prognosis
  - With timely surgery the prognosis is fair
    - Mortality rates 45% and higher have been reported BUT mortality rates as low as 10% are becoming more common
    - The prognosis is poor if gastric necrosis or perforation occurs or if surgery is delayed
    - Reperfusion injury causes tissue damage that ultimately results in death after correction of GDV
  - Lactate measurements



Decontamination

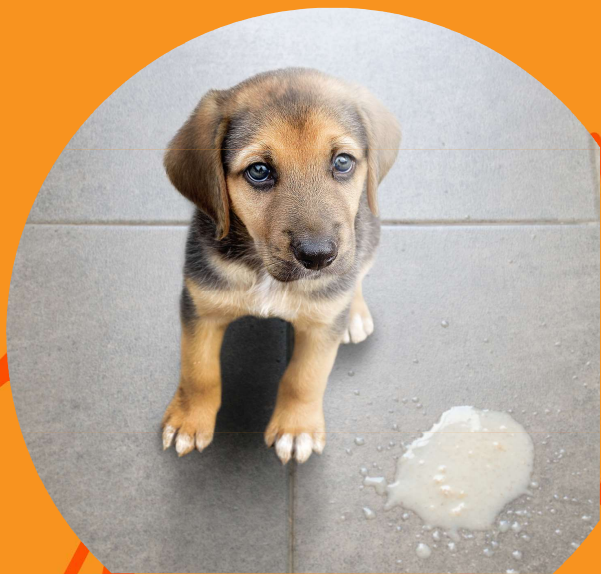

Image: https://www.cliniciansbrief.com/article/art-triage

Induction of emesis

Induction of emesis

- Emesis should still be considered even several hours after ingestion of toxins (e.g. chocolate- slow absorption)
- Induction of emesis is contraindicated if the patient is showing respiratory distress, seizuring or is extremely weak

- Apomorphine:
  - Dose: 0.03 mg/kg IV; 0.04mg/kg IM ; 0.08mg/kg SC
  - Topical conjunctival dose 0.3mg/kg or a tablet can be placed in the conjunctival sac
    - Once emesis is achieved and the vomitus is clear the conjunctival sac must be throughly rinsed with saline to terminate its effects
    - Reversal agent is metoclopramide
  - **NOT in cats**

Induction of emesis

- Xylazine
  - Mainly cats
  - Dose: 0.44 mg/kg IM
  - Not always reliable (60% of cats)
  - Reversal agent yohimbine or atipamezole

- Dexmedetomidine (my go to in cats)
  - Dose: 7-10 mcg/kg IM
  - Not always reliable
  - Reversal agent atipamezole

- Hydrogen peroxide 3%
  - Less commonly used
  - Risk of oesophagitis, hemorrhagic gastritis, and air embolism
  - **NOT in cats**

- Liquid dishwashing detergent (NOT cats)
- Syrup of ipecac (can be cardiotoxic if not vomited) (NOT cats)

Gastric lavage

Indicated when emesis is contraindicated or when induction of emesis has not achieved adequate results

- Light sedation that allows ET tube placement and cuffing
- Pre-measure a large bore hole stomach tube from the tip of the nose to the last rib
- Lubricate the end with KY gel and gently pass into the oesophagus and into the stomach
- Instil 10- 20ml/kg of tepid tap water into the stomach via the tube (use a funnel)
  - The free end of tube is then lowered to allow drainage via gravity
  - This is repeated until the fluid runs clear
  - Apply gentle pressure to the stomach to facilitate drainage
  - Move the patient into different positions to ensure complete lavage is achieved
  - Activated charcoal can be mixed with the fluid to enhance the effectiveness of the lavage
- Kink the tube and pull it out when the stomach as been lavaged adequately

Adsorbents

Activated charcoal:

- Decreases absorption and enhances elimination of organic compounds from GI tract
- Dose 1-4 mg/kg orally in dogs, mixed with 50-200ml of water (6-12ml /kg)
  - This can be given after gastric lavage prior to removal of the tube
  - This dose should be repeat every 4-6 hrs and combined with a cathartic to prevent relapse in intoxication where enterohepatic recirculation occurs (e.g chocolate toxicity)
  - Feeding activated charcoal mixed in food may alter efficacy
  - One should aim to have at least 2hrs between oral medication as it can delay or alter absorption
- Activated charcoal will result in black stool and owners should be warned
- Risk for aspiration and subsequent pneumonia in patients with altered mentation, so airway protection is necessary during administration
- Ensure adequate hydration and allow free access to water (if not contraindicated) to prevent iatrogenic hypernatremia
- Monitor electrolytes following treatment
- NOT indicated for toxicants such as ethanol, ethylene glycol, xylitol, heavy metals (e.g. ferrous sulfate, iron, zinc, lithium), nitrates, sodium chloride, and chlorates



Cathartics

Cathartics assist in elimination of toxins and toxin-bound activated charcoal

- They should be used with caution in very old or young patients and those with pre-existing real disease, hypertension and congestive heart failure
- Use cathartics no more frequently than every 3rd dose when administering multiple charcoal doses to avoid inducing diarrhoea and/or hypernatremia

  - Lactulose
  - Sorbitol 70%
  - Sodium sulfate
  - Magnesium sulfate
  - Magnesium citrate



Bathing

- If exposure was dermal, bathe the patient with mild dish soap in lukewarm water, and avoid hypothermia



Chocolate toxicity



Image: https://www.cliniciansbrief.com/article/art-triage

Chocolate toxicity

Contains two toxins that could be potentially lethal to dogs

- Theobromine
- Caffeine
- Both are methylxanthine alkaloids

Chocolate toxicity

- Common due to it's pleasant odour and palatability
- More in dogs due to their indiscriminate eating habits and affinity for sweet things
- Can be 'seasonal' with it more likely to occur at Easter and Christmas time
- Occurs more frequently in homes with children
- Diagnosis is usually made on history and clinics signs as most owners have either witnessed the exposure or have found evidence of the exposure
- Death is due to cardiac arrhythmia, respiratory failure, hyperthermia, congestion, oedema or a combination of these
- Pancreatitis may be seen 24-72hrs after ingestion



Mechanism of action

Metabolism

- Methylxanthines are rapidly absorbed from the gastrointestinal tract however the fat content of the chocolate and the formation of chocolate 'bezoars' may slow this absorption
- Metabolism of theobromine occurs in the liver with enterohepatic recirculation occurring prior to excretion in urine
  - This prolongs the half life of theobromine which is 17.5 hrs, where as caffeine only has a half life of 4.5 hrs
- Reabsorption of methylxanthines and their metabolites can occur from the urinary bladder
- They can also cross the placenta and pass into milk affecting unborn foetuses

Mechanism of action

- Theobromine inhibits phosphodiesterase causes increased levels of intracellular cyclic adenosine monophosphate (c-AMP)
  - This increases circulating catecholamines (ephinephrine and norephinine)
    - Leads to vasoconstriction, tachycardia and CNS stimulation
- Theobromine antagonises adenosine receptors interfering with uptake and storage of intracellular calcium within striated muscles causing contractions
- It also causes relaxation of the smooth muscles of the respiratory and urinary tracts and acts locally as a gastric irritant increasing secretion

Clinical signs

Clinical Signs of toxicity

- 3 main body systems affected
  - cardiovascular
  - central nervous systems
  - gastrointestinal
- The respiratory system and urine system are affected but to a lesser extent
- Clinical signs are usually seen within 2-4 hrs after consumption but theobromine concentrations can take up to 10 hrs to peak and it can take up to 24 hrs for the true extent of the intoxication to be appreciated
- Symptoms can last up to 72 hrs
- Animals with pre-existing heart conditions as well as seizure disorders may be at higher risk

CVS	CNS	GIT	RESPIRATORY	URINARY
Tachycardia	Restlessness	Vomiting	Tachypnoea	Polydipsia
Hypertension	Hyper excitability/activity	Salivation	Cyanosis	Polyuria
Arrhythmias (PVC)	Ataxia	Diarrhoea		
	Tremors	Bloating		
	Convulsions/Seizures			
	Hyperthermia			
	Coma			

Chocolate content

Toxic dose

- Toxic doses may vary between individuals due to a decreased ability to metabolise and eliminate theobromine (Polymorphism in cytochrome P450 enzymes)
- Not all chocolate contains the same amount of theobromine because the methxanthines content will vary among cocoa beans well as between brands of products

Compound	Theobromine (mg/g)	Caffeine (mg/g)
White chocolate^	0.0088	0.0299
Milk chocolate 10%	2.04	0.21
Dark, sweet chocolate 35%	4.58	0.71
Semi-Sweet chocolate chips	4.87	0.78
Bakers, unsweetened chocolate 50-60%	13.86	1.66
Dry cocoa powder	25.99	2.47
Instant cocoa powder	4.80	0.53
Cocoa beans	21.16	not available
Coffee beans	0	21.16
Cocoa bean hulls*	8.99	not available

Table 1: Relative concentrations of Methylxanthines in various form of chocolate

^ The amount of theobromine in white chocolate is insignificant and there is more risk of pancreatitis due to higher fat content

*Cocoa bean hulls are often used in the garden for mulch and contain high concentrations of theobromine. These may be attractive to dogs due to the characteristic smell

Calculating methylxanthine consumed

The total amount of methyxanthine consumed per kilogram body weight must be calculated to determine if the patient is at risk for toxicity....

- Calculate total amount of methylxanthine found in 1 gram of a certain product of chocolate:
  - Add the amount of theobromine to the amount of caffeine (Refer to table 1)
  - For mixed products such as chocolate covered jelly or nuts assume 100% is chocolate
  - One should always work on a 'worst-case-senario' situation

2. Calculate how much was actually consumed by multiplying this number by the approximate number of grams of chocolate consumed

3. Divide the total amount of methylxanthines consumed by the body weight to see how many mg/kg was ingested

- **Mild symptoms**
  - Dose 20-40mg/kg of theobromine
  - Vomiting, agitation, hyper excitability and restlessness
- **Moderate symptoms**
  - Dose 40-50mg/kg of theobromine
  - Heart related signs
- **Severe symptoms**
  - Dose > 60mg/kg
  - seizures, arrhythmias, coma and death

Treatment

There is no specific antidote for methylxanthines and thus treatment is supportive and symptomatic.

- Elimination of further absorption > see previous slides (emesis, gastric lavage, activated charcoal, cathartics)
- Elimination of already absorbed toxin (IVFT, u - Cath)
- Symptomatic treatment



Image:
https://commons.wikimedia.org/wiki/File:Dog_vomiting_with_apomorphine.jpg

Elimination of absorbed toxin

Intravenous fluid therapy

- Fluid diuresis is used to improve elimination of theobromine, after enteroheptic circulation, via the kidneys
- 2-3 times maintenance
    - Monitor respiratory rate and cardiac condition

Urinary catheterisation

- Prevents resorption of methylxanthines and their metabolites from the urinary bladder



Image:
https://dogstodaymagazine.co.uk/

Symptomatic treatment

- Arrhythmia - continuous ECG monitoring is advised
    - Ventricular tachycardia/ premature contractions:
        - Arrhythmias require treatment if they interfere with cardiac output
            - poor peripheral pulses, prolonged CRT, heart rate consistently > 160-180bpm
        - Dose:
            - A test bolus of lidocaine given intravenously (2 mg/kg bolus) can be used to determine responsiveness
            - Can repeat 1-3 boluses but no more than 6-8 mg/kg of lidocaine over 15-30 min to prevent toxicity
            - If the arrhythmias diminish or stop with bolus, lidocaine should be given by a continuous intravenous infusion (start at 50µg/kg/minute and titrate up)

- Arrhythmia - continuous ECG monitoring is advised
    - Supraventricular arrhythmia:
        - Diltiazem/ Metoprolol/ Propanol
        - These drugs may delay renal excretion of methylxanthines and should be used with caution

Symptomatic treatment

- Hypertension
  - Blood pressure should be evaluated frequently and if noted this should be treated.
  - **NOTE**: Arrhythmia should be addressed first as control of these could subsequently control hypertension

- Tremors and Seizures
  - Diazepam: Cats/dogs: 0.5mg/kg IV or rectally
  - Midazolam: Cats/dogs: 0.5 mg/kg IV/rectally or 0.2 mg/kg intranasally
  - Keppra: Dogs: 30–60 mg/kg IV slow bolus over 5–15 min, Cats: 20 mg/kg IV
  - If seizures are refractory to the above, general anaesthesia can be induced using
    - Propofol: 3- 6mg/kg IV (to effect) followed by CRI 0.1- 0.4mg/kg/minute
    - It is safer to intubate these patients
    - Monitor closely for respiration depression (ETCO2, RR, SPO2)

  - If tremors are present without seizures
    - Methocarbamol (Robaxin)
    - Dose: 50-220 mg/kg IV slowly, to effect
    - Do not exceed 330 mg/kg/day

Symptomatic treatment

- Gastric irritation
  - H2 receptor blockers/ Proton Pump inhibitors/Sucralfate

- Vomiting
  - Anti-emetic therapy is indicated if vomiting continues
  - Metoclopramide/ Maropitant citrate

- Hyperthermia
  - Hyperthermia will usually resolve when CNS signs are controlled
  - Cool with fans and ice packs in the cage (not against the patient)



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Ongoing Monitoring

- One should continue to monitor vitals including temperature
- An ECG should be attached to the patient to monitor for arrhythmias
- BP and serum electrolytes should be closely monitored
- Monitor for signs of pancreatitis, rhabdomyolysis



Organophosphate toxicity



Image: https://mikebolhuis.co.za/

Organophosphate (OP) and carbamate (CM) toxicosis

- Exposure to OP/CM (Aldicarb)
  - Household, garden, and agricultural insecticides
  - Dogs are most commonly intoxicated
  - Malicious poisoning of dogs

- Absorbed via most routes, including transdermally and inhalation

- OP/CM are cholinesterase inhibitors
  - By inhibiting acetylcholinesterase (AChE) they prolong the activity of acetylcholine at nerve endings
  - Overstimulation of cholinergic nerves occurs, particularly at muscarinic and nicotinic synapses



Image: https://mikebolhuis.co.za/

Organophosphate (OP) and carbamate (CM) toxicosis

- Oganophosphates
  - Require activation after absorption
  - **Irreversibly** bind to AChE
  - Persistence of OPs on the AChE molecule can result in prolonged duration of clinical signs, particularly nicotinic effects

- Carbamates
  - They are immediately active upon absorption
  - **Reversibly** bind to AChE
  - Effects of CMs are usually of short duration (≤4 hours)
    - Patients that survive an acute episode of CM toxicosis may appear normal within a few hours

Clinical Signs

Classic OP/CM Toxicosis:

- Clinical disease can occur within 30 minutes of ingestion/inhalation but can delayed for up to 12-24 hours after dermal exposure
- Severity depends on the relative toxicity of the OP/CM and the amount to which the patient was exposed
- Not all patients show the same clinical signs at the same level of exposure (Individual variation)

- Nicotinic effects:
  - Skeletal muscle fasciculations, tremors, convulsions, profound muscle weakness
  - Hyperthermia can develop secondary to this

- Muscarinic effects:
  - "SLUDGE" syndrome
    - i.e. salivation, lacrimation, urination, defecation, gastrointestinal distress, emesis

- CNS effects:
  - Apprehension, seizures, and depression

- Death can occur > asphyxia from excessive bronchial and pulmonary secretions, seizures, or respiratory compromise/arrest

Clinical Signs

Note: Two other syndromes have been described but are uncommon

Intermediate Syndrome (IMS)

- Occur 7-96 hours following acute exposure in dogs and cats
- Nicotinic dysfunction
  - Profound muscle weakness, especially neck flexors, facial muscles, ocular muscles, proximal limbs, and respiratory muscles

Delayed-Onset Toxicosis:

- OPs are highly lipid soluble, so are distributed and stored in body fat
- Conditions like anorexia can result in rapid mobilisation of fat and release stored OPs
- Signs occur days to weeks after exposure and include paresis of limbs, ventroflexion of neck, proprioceptive deficits, and generalized weakness

Diagnosis

Atropine Response Test:

- In cases where OP/CM toxicosis is suspected,
  - Listen to the heart rate and take note
  - Administer a preanesthetic dosage of atropine (0.02 mg/kg IM)

- If heart rate increases (>5 bpm), pupils dilate, and/or hypersalivation diminishes
  - Clinical signs are **not** from OP/CM exposure because it takes approximately 10 times more atropine to counteract the effects of OP/CM

Cholinesterase Activity Assessment:

- Measurement of AChE in whole blood, brain, or retinal samples can be performed
- Activity that is ≤25% of normal, in conjunction with characteristic SLUDGE signs, is considered diagnostic for AChE toxicosis
- Values that are ≤50% of normal are indicative of significant exposure
- Note: that reactivation of blood cholinesterase activity can occur during collection and transport of samples after exposure to CM, resulting in false-negative results

Decontamination

- Emesis
- Gastric lavage
- Activated charcoal
- Bathing



Image: https://mikebolhuis.co.za/

Treatment

- Manage any life-threatening clinical signs first
  - Provide oxygen
  - Mechanical ventilation may be required in patients with hypoventilation and respiratory arrest
  - Treat hyperthermia (Usually resolve when tremors/seizures are controlled)

  - Tremors and Seizures
    - Diazepam: Cats/dogs: 0.5mg/kg IV or rectally
    - Midazolam: Cats/dogs: 0.5 mg/kg IV/rectally or 0.2 mg/kg intranasally
    - Keppra: Dogs: 30–60 mg/kg IV slow bolus over 5–15 min, Cats: 20 mg/kg IV
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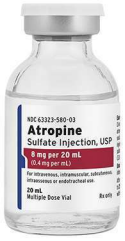
      - It is safer to intubate these patients
      - Monitor closely for respiration depression (ETCO2, RR, SPO2)

  - If tremors are present without seizures
    - Methocarbamol (Robaxin)
    - Dose: 50-220 mg/kg IV slowly, to effect
    - Do not exceed 330 mg/kg/day

Treatment

- Atropine:
  - Anticholinergic drug treats **MUSCARINIC EFFECTS-** bronchial secretions and bradycardia
  - Give 1/4 dose of 0.2-0.4 mg/kg IV and the remainder IM or SC
  - It can be repeated q 4-8 hrs as needed
  - Monitoring for signs of **atropine toxicosis:** behaviour changes, hyperthermia, erythema, dry mouth
  - Atropine does not control nicotinic (e.g. tremors, fasciculations) or CNS (e.g. seizures) effect



- Pralidoxime Chloride (2-PAM):
  - Cholinesterase reactivator treats **NICOTINIC EFFECTS -** weakness and muscle fasciculations
  - Ideally administer 2-PAM within the first 24 hours of exposure for greatest efficacy
  - Because CMs bind reversibly and are short acting 2-PAM is generally not used for CM intoxications

Note: Phenothiazines (e.g. acepromazine) and their derivatives are contraindicated because they can potentiate the clinical effects of OP/CMs

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Supportive Care

- Intravenous fluid therapy
- Balance electrolytes
- Analgesics
- Antiemetics
- Ensure adequate nutrition
- Nursing care
- Urinary catheters

Monitoring

- Monitor for pancreatitis for 72 hours
- Monitor patients with severe diarrhea for evidence of intussusception
- Monitor for atropine toxicosis in patients receiving atropine

Other Common Emergencies

1. *Gastric Dilatation and Volvulus*
2. *Intoxications*
3. *Snake Bites*
4. *Urethral Obstruction*

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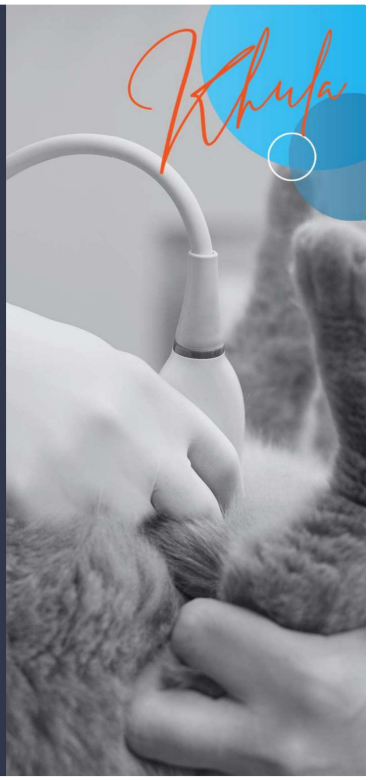
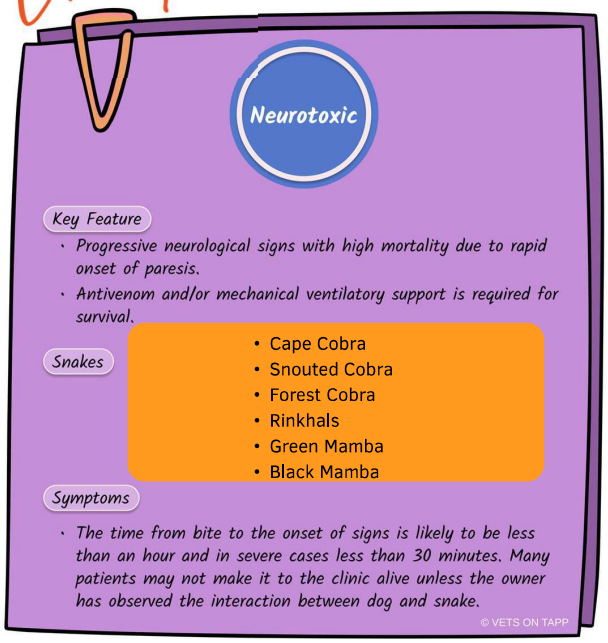


Snake bites

- Clinicians should know which venomous snake species are most likely to occur in their area and be able to identify them
- The most common bite site in dogs is cranial to the shoulders. The loose skin around the neck and face are commonly involved
  - This can be important when bitten by a cytotoxic snake as it can lead to respiratory compromise
- Venom can be broadly classified as **neurotoxic, haemotoxic and cytotoxic**
- Some snakes have a combination of venoms and the clincian should have an understanding of the consequences of all types of venom
- The amount of venom injected can be highly variable and contributes to the severity of clinical signs observed in snakebite patients
- In cases where a photo of the snake, the snake itself, or a good description of the snake is not presented symptomatic treatment and continual monitoring should be started



www.hoedspruitreptilecentre.com



CAPE COBRA (Naja nivea)
- **Distribution:** Western, Northern and Eastern Cape, Northwest, Free State
- **Colour:** Varied between yellow, brown, black, cream and a speckled phase
- **Length:** 1.4-1.6m
- **Venom:** Neurotoxic
- **Venom Effects:** Progressive Weakness and Paralysis



SNOUTED COBRA (Naja annulifera)
- **Distribution:** KZN, Mpumalanga, Northwest, Limpopo, Gauteng
- **Colour:** Yellowish brown with a yellow belly, or black and cream bands
- **Length:** 1.8-2.5m
- **Venom:** Neurotoxic & Cytotoxic
- **Venom Effects:** Progressive Weakness and Paralysis along with Painful Progressive Swelling



FOREST COBRA (Naja subfulva)
- **Distribution:** Northern Coastal Bush KZN, Limpopo along the Pafuri River in KNP to Eastern Soutpansberg Mountains
- **Colour:** Black back half with a yellowish-brown front half
- **Length:** 2-2.7m
- **Venom:** Neurotoxic & Cytotoxic
- **Venom Effects:** Progressive Weakness and Paralysis along with Painful Progressive Swelling

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Neurotoxic

Key Feature

- Progressive neurological signs with high mortality due to rapid onset of paresis.
- Antivenom and/or mechanical ventilatory support is required for survival.

Snakes

- Cape Cobra
- Snouted Cobra
- Forest Cobra
- Rinkhals
- Green Mamba
- Black Mamba

Symptoms

- The time from bite to the onset of signs is likely to be less than an hour and in severe cases less than 30 minutes. Many patients may not make it to the clinic alive unless the owner has observed the interaction between dog and snake.

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RINKHALS (Hemachatus haemachatus)

- **Distribution:** KZN, Mpumalanga, Western Cape, Eastern Cape, Northwest, Limpopo, Free State, Gauteng
- **Colour:** Black, brown or olive with white throat bands or black and yellow/orange body bands with yellow throat bands
- **Length:** 1.0-1.5m
- **Venom:** Cytotoxic & Neurotoxic
- **Venom Effects:** Progressive Weakness and Paralysis along with Painful Progressive Swelling



GREEN MAMBA (Dendroaspis angusticeps)

- **Distribution:** Coastal Bush KZN, Northern Coastal Bush Eastern Cape
- **Colour:** Uniform green with irregular yellow scales
- **Length:** 1.8-2.5m
- **Venom:** Neurotoxic
- **Venom Effects:** Progressive Weakness and Paralysis with or without minor swelling



BLACK MAMBA (Dendroaspis polylepis)

- **Distribution:** KZN, Mpumalanga, Northern Cape, Northern Coastal parts Eastern Cape, Northwest, Limpopo, Gauteng
- **Colour:** Dark Olive, greyish brown or gunmetal grey
- **Length:** 2.8-3.2m but up to 4.5m
- **Venom:** Neurotoxic
- **Venom Effects:** Progressive Weakness and Paralysis with or without minor swelling

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Neurotoxic

- Neurotoxic venom works at the neuromuscular junction and causes progressive neurological signs often starting with the loss of the swallowing reflex and paralysis of the tongue and jaw. This may be noticed as saliva dripping from the mouth. This is followed by limb weakness which finally progresses to paralysis.
- As the respiratory muscles become paralysed the breathing shallows and eventually stops, leading to cyanosis and death.
- Placement of an endotracheal tube and mechanical ventilation using an AMBU bag, closed circuit anaesthetic machine or a ventilator is needed to keep these patients alive until enough antivenom has been given to reverse the paralysis or until the venom has worked its way out of the patient's body if no antivenom is available.

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Neurotoxic

Considerations

- Good nursing care is vital with these patients while they are being ventilated.
- A urinary catheter should be placed and the patient should be turned on a regular basis.

Monitor

- Heart rate, pulse quality, capillary refill time, blood pressure
- Body temperature
- End-tidal CO_2 and SpO_2
- Urine output and colour
- The bite site should be closely monitored for any cytotoxic changes

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Haemotoxic

Key Features

- Venom-induced consumption coagulopathy (VICC) leading to active bleeding

Snakes

- Vine snake
- Boomslang

Symptoms

- Haemotoxic venom is known to induce a consumption coagulopathy similar to disseminated intravascular coagulation (DIC)
- Haemotoxic venom contains haemorrhagins, procoagulants enzymes (that cause activation of clotting factors II and X), thrombin-like substances and/or venom toxins known cause depletion of platelets and marked thrombocytopaenia.
- Haemotoxic venom causes active bleeding, seen from the gums, the bite site, gastro-intestinal, respiratory and urinary tracts.

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VINE SNAKE (Thelotornis capensis)

- **Distribution:** KZN, Mpumalanga, Northwest, Limpopo, Gauteng
- **Colour:** Cryptically coloured resembling a stick
- **Length:** 1.2-1.5m
- **Venom:** Haemotoxic
- **Venom Effects:** Bleeding



BOOMSLANG (Dispholidus typus)

- **Distribution:** Widespread throughout South Africa where tree habitat Is found
- **Colour:** Grey, Brown, Green, Red, Blue, Green with Black "bands", black backs with yellow bellies
- **Length:** 1.5-2.0m
- **Venom:** Haemotoxic
- **Venom Effects:** Bleeding

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Haemotoxic

Considerations

- Be aware that haematomas can form at venipuncture sites so jugular sticks should be avoided.
- Fresh whole blood or fresh frozen plasma transfusions may be necessary and life-saving.
- In humans, antivenom is effective even when active haemorrhage is seen.

Monitor

- Serial monitoring of prothrombin time (PT) and activated partial thromboplastin time (aPTT)
- Monitoring of Buccal Mucosal Bleeding Time (BMBT) may be useful
- Heart rate, pulse quality, capillary refill time, blood pressure
- Respiratory rate, respiratory effort
- Packed cell volume, total solids
- Urine output and colour

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Cytotoxic Venom

Key Feature

- *Progressive swelling with high morbidity and low mortality. Antivenom is infrequently required for survival*

Snakes

- Mozambique spitting cobra
- Forest cobra
- Snouted cobra
- Rinkhals
- Gabon adder
- Night adder
- Puff adder
- Stiletto snake

Symptoms

- *Swelling is usually present within 2 hours of the bite, peaking between 12 and 24 hours and is significantly reduced by 72 hours (without the use of antivenom).*
- *Swelling is localised to the bite site and is typically non-painful but can vary depending on which snake has bitten the patient.*
- *If the swelling is in the region of an airway it can lead to airway obstruction and death by asphyxiation.*

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MOZAMBIQUE SPITTING COBRA (Naja mossambica)

- **Distribution:** KZN, Mpumalanga, Northern Coastal Bush Eastern Cape, Northwest, Limpopo, Gauteng
- **Colour:** Brown with an orange/salmon belly and black bands on the neck
- **Length:** 1.2-1.6m
- **Venom:** Cytotoxic
- **Venom Effects:** Painful Progressive Swelling



FOREST COBRA (Naja subfulva)

- **Distribution:** Northern Coastal Bush KZN, Limpopo along the Pafuri River in KNP to Eastern Soutpansberg Mountains
- **Colour:** Black back half with a yellowish-brown front half
- **Length:** 2-2.7m
- **Venom:** Neurotoxic & Cytotoxic
- **Venom Effects:** Progressive Weakness and Paralysis along with Painful Progressive Swelling



SNOUTED COBRA (Naja annulifera)

- **Distribution:** KZN, Mpumalanga, Northwest, Limpopo, Gauteng
- **Colour:** Yellowish brown with a yellow belly, or black and cream bands
- **Length:** 1.8-2.5m
- **Venom:** Neurotoxic & Cytotoxic
- **Venom Effects:** Progressive Weakness and Paralysis along with Painful Progressive Swelling



RINKHALS (Hemachatus haemachatus)

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- **Colour:** Black, brown or olive with white throat bands or black and yellow/orange body bands with yellow throat bands
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Snakes

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Symptoms

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GABOON ADDER (Bitis gabonica)

- **Distribution:** Coastal Northern KZN
- **Colour:** Various shades of pastel colours with blocks along the back and triangles down the sides
- **Length:** 1.2m can get bigger
- **Venom:** Cytotoxic
- **Venom Effects:** Mixed Painful Progressive Swelling & Bleeding



PUFF ADDER (Bitis arietans)

- **Distribution:** Throughout South Africa
- **Colour:** Colour varies but has V-shaped markings down the back pointing towards the tail
- **Length:** 0.9-1.2m but up to 1.4m
- **Venom:** Cytotoxic
- **Venom Effects:** Mixed Painful Progressive Swelling & Bleeding



COMMON OR RHOMBIC NIGHT ADDER (Causus rhombeatus)

- **Distribution:** KZN, Mpumalanga, Western Cape, Eastern Cape, Northwest, Limpopo, Free State, Gauteng
- **Colour:** Dark brown Rhombic markings on the back. Body colour varies from light grey to brown. Characteristic "V"shape marking on the head.
- **Length:** 40-60cm. Max 1m
- **Venom:** Cytotoxic
- **Venom effects:** Moderate local swelling and pain.

Photo Credit: Neville's Snake and Reptile Rescue, Eastern Cape.



BIBRON'S STILETTO (Atractaspis bibronii)

- **Distribution:** KZN, Mpumalanga, Northern Cape, Northern Coastal Bush Eastern Cape, Northwest, Limpopo, Free State, Gauteng
- **Colour:** Body brown to blackish, belly may be white
- **Length:** 40-60cm, max 98cm.
- **Venom:** Cytotoxic
- **Venom effects:** Moderate swelling with potential of causing local tissue necrosis.

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Cytotoxic Venom

- Systemic signs are generally associated with blood loss into the tissues leading to hypovolemic shock. Symptoms such as pale gums, tachycardia, hypotension, hypothermia, weakness may be experienced, which can progress to hypoxia, collapse and multi organ dysfunction.
- It is important that patients that are showing signs are carefully observed for progressive swelling.

Considerations

- Upper airway obstruction may require tracheostomy tube placement.

Monitor

- Heart rate, pulse quality, capillary refill time, blood pressure
- Respiratory rate, respiratory effort
- Packed cell volume, total solids, in-saline agglutination (ISA)
- Urine output and colour

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Antivenom

- Antivenom consists of polyclonal antibodies that are generated by immunising animals (usually horses) with small amounts of snake venom.
- Antivenom neutralises a fixed quantity of venom
- It is challenging to know how much venom has been injected into a patient as dry bites are possible.
- Due to this, the dose of antivenom needed is not set in mg/kg or ml/kg as it is for other drugs.
- Small dogs often have worse outcomes compared to large dogs as the volume of venom injected to mass ratio is larger.
- In general, it is not advised to use antivenom in patients who are not showing clinical signs.
- All patients bitten by neurotoxic snakes that are showing symptoms should receive antivenom.
- Antivenom should be used in cytotoxic snakebites if the patient is deteriorating and systemic signs are seen.
- Adverse effects of antivenom include a rash, urticaria, angio-oedema, bronchospasm, hypotension, vomiting, diarrhoea, and on rare occasions anaphylaxis.

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Identification

https://www.africansnakebiteinstitute.com



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Identification

POLYVALENT ANTIVENOM SPECIES



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BLACK MAMBA (Dendroaspis polylepis)
- **Distribution:** KZN, Mpumalanga, Northern Cape, Northern Coastal parts Eastern Cape, Northwest, Limpopo, Gauteng
- **Colour:** Dark Olive, greyish brown or gunmetal grey
- **Length:** 2.8-3.2m but up to 4.5m
- **Venom:** Neurotoxic
- **Venom Effects:** Progressive Weakness and Paralysis with or without minor swelling



GREEN MAMBA (Dendroaspis angusticeps)
- **Distribution:** Coastal Bush KZN, Northern Coastal Bush Eastern Cape
- **Colour:** Uniform green with irregular yellow scales
- **Length:** 1.8-2.5m
- **Venom:** Neurotoxic
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- **Length:** 2-2.7m
- **Venom:** Neurotoxic & Cytotoxic
- **Venom Effects:** Progressive Weakness and Paralysis along with Painful Progressive Swelling

MONOVALENT ANTIVENOM SPECIES



BOOMSLANG (Dispholidus typus)
- **Distribution:** Widespread throughout South Africa where tree habitat is found
- **Colour:** Grey, Brown, Green, Red, Blue, Green with Black "bands", black backs with yellow bellies
- **Length:** 1.5-2.0m
- **Venom:** Haemotoxic
- **Venom Effects:** Bleeding

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OTHER SPECIES



VINE SNAKE (Thelotornis capensis)
- **Distribution:** KZN, Mpumalanga, Northwest, Limpopo, Gauteng
- **Colour:** Cryptically coloured resembling a stick
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- **Length:** 40-60cm. Max 1m
- **Venom:** Cytotoxic
- **Venom effects:** Moderate local swelling and pain.

Photo Credit: Neville's Snake and Reptile Rescue, Eastern Cape.

Even though localized symptoms could seem extreme, there is no antivenom for the treatment of stiletto and night adder bites.

DISCLAIMER
The authors and editor have exerted every effort to ensure that the clinical procedures and recommendations described herein are based on current knowledge and state-of-the-art information obtained from acknowledged authorities, texts and journals. However, they cannot be considered absolute and universal recommendations. Each patient's situation must be considered

Reporting Snake Bites



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Ocular Envenomation

- *Ocular envenomation is caused by snakes that spit their venom.*
- *This leads to large, painful corneal ulcers and chemosis followed in some cases by a sterile uveitis and/or hypopyon.*
- *Treatment involves flushing of the eye with large volumes of an isotonic irrigation agent such as Lactated Ringers.*
- *1 liter per eye has been suggested in some texts.*
- *The pain can be severe and the patient may require sedation to allow proper irrigation.*
- *Fluorescein can be used to assess the extent of the corneal ulceration.*
- *Topical antibiotics are indicated and topical atropine can assist in pain management. Oral pain control should be considered.*
- *Topical corticosteroids should **not** be used.*

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MOZAMBIQUE SPITTING COBRA (Naja mossambica)

- **Distribution:** KZN, Mpumalanga, Northern Coastal Bush Eastern Cape, Northwest, Limpopo, Gauteng
- **Colour:** Brown with an orange/salmon belly and black bands on the neck
- **Length:** 1.2-1.6m
- **Venom:** Cytotoxic
- **Venom Effects:** Painful Progressive Swelling

Other Common Emergencies

1. *Gastric Dilatation and Volvulus*
2. *Intoxications*
3. *Snake Bites*
4. *Urethral Obstruction*

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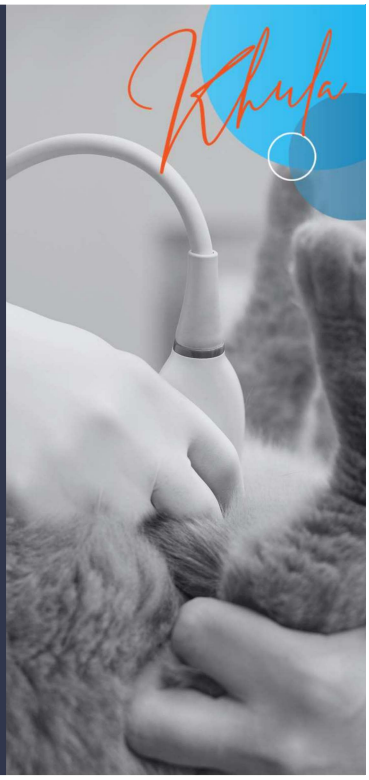


Khula



- Castrated vs. uncastrated males?
- Indoor cats & litter hygiene
- Diet
- Seasonal > Spring - territorial roaming / rainy season
- Breed predisposition
- STRESS, multicat households, new cat/family member

Khula

History



- Increased frequency of urination
- In and out of the tray
- Straining to urinate
- Crying or vocalising when urinating
- Failure to pass urine/only small drops
- Haematuria
- Urinating in inappropriate places
- Anorexia/lethargy/depression/hiding

Khula

- Shock
- Weak / Collapsed
- Pale mm/ slow CRT
- Bradycardia (100 – 140 bpm)
- Large, hard bladder
- Bruising of tip of penis



PRESENTATION

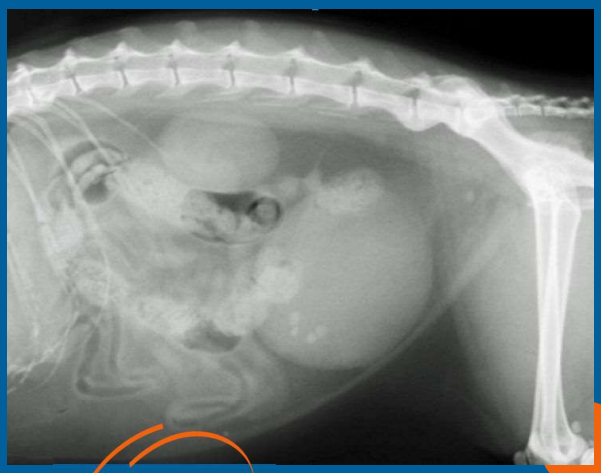
Depends on severity and duration

Khula

METABOLIC DERRANGEMENTS

- Azotaemic
- Hyperkalaemic
- Acidotic

+++ RISK OF CARDIAC ARREST



STABILISATION

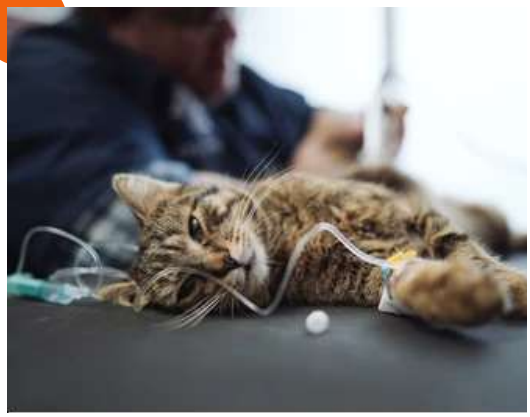


Image: shutterstock.com - 1678675432

- Baseline chem + electrolytes + CBC (PCV/TS)
- IV catheter placement
- Fluid therapy: crystalloid - LRS (4mmol K) vs. 0.9% Saline (acidifying)
- Shock rates 5-10ml/kg boluses over 15-30 min, reassess HR/CRT/Pulse/RR/mentation/ BP
- Treat hyperkalemia

Khula

Khula

HYPERKALEMIA

K+

- ECG helps us determine how significant the hyperkalemia is.
- ECG -inappropriate bradycardia, spiked T wave, Loss of P waves, +/- ST segment depression > progress to wide/bizarre complexes.
- We should base treatment on the status of the animal, perfusion status and ECG, not necessarily the potassium concentration. Some patients will have K at 6.8 but are experiencing severe changes on ECG and other will be above 7 with no changes.

+++ RISK OF CARDIAC ARREST

STABILISATION

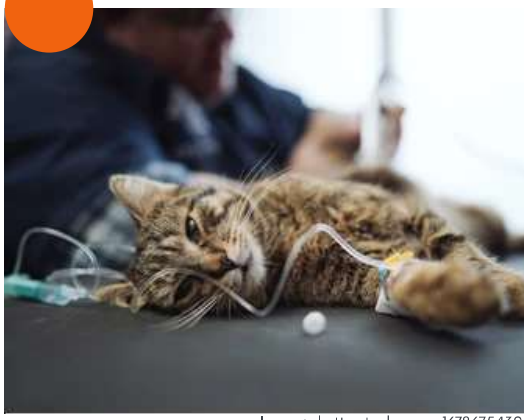


Image: shutterstock.com - 1678675432

10% Calcium gluconate

- This does NOT lower potassium but antagonises its effect at the muscle and provides cardiovascular protection
- Its effects last about 20-30 min which should allow a safer GA and time to unblock
- Ideally given while watching the ECG
- Dose: 0.5 - 1.5 ml/kg IV
- Rate: 1 ml/min
- This can be repeated

Khula

STABILISATION



Image: shutterstock.com – 1678675432

Dextrose

- Only after initial fluid resuscitation has been completed. Dextrose can be used alone or in combination with short acting Insulin
- On its own Dextrose stimulates endogenous insulin release. This results in K being taken up with glucose.
- Bolus: 1 ml 50% dextrose/kg body weight + equal volume saline.

  e.g
  3 kg cat gets 3ml 50% dextrose + 3ml saline mixed in the same syringe and given slow IV
- CRI: 2.5% Dextrose Infusion

  To make a 2.5% solution:
  add 50mL of 50% dextrose (or 25g dextrose) to a 1 L bag of fluids

STABILISATION



Image: shutterstock.com – 1678675432

Dextrose and short acting insulin

- Only after initial fluid resuscitation has been completed, if given before can cause arrhythmia and death.
- Insulin (Actrapid) 0.25 - 0.5 IU/kg IV

PLUS

- Bolus: Dextrose 2g per unit of insulin given
  *dilute with equal volume of saline and give slow IV

  (Each mL of 50% Dextrose contains 0.5 g dextrose)

  e.g
  5 kg cat at 0.25 IU/Kg = 1.25 Units
  2g dextrose x 1.25 units = 2.5 g dextrose
  2.5mg / 0.5 = 5ml dextrose
  5ml dextrose + 5ml saline
- Insulin is supposed to last 4 hrs but can last longer in some patients so follow up with 2.5% Dextrose CRI (Avoid a hypoglycaemic seizure)

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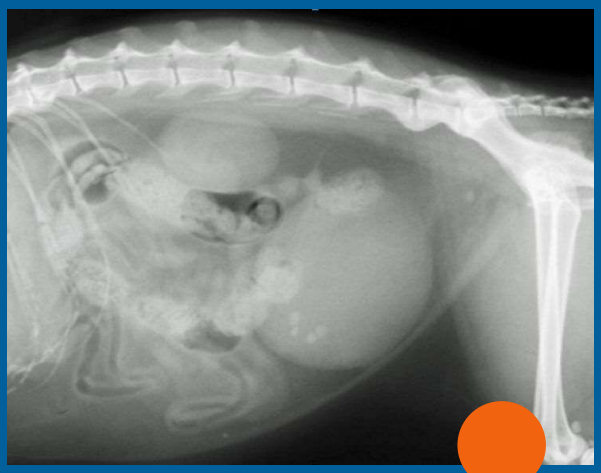
DIAGNOSTICS

- Bloods
- Xrays - always include prepuce (1 view vs 2?)
- U/S
- UA



CYSTOCENTESIS



Image: https://www.atdove.org/video/ultrasound-guided-cystocentesis

To perform a cystocentesis?
YAY / NAY

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SEDATION/ GA



Image: shutterstock.com - 1678675432

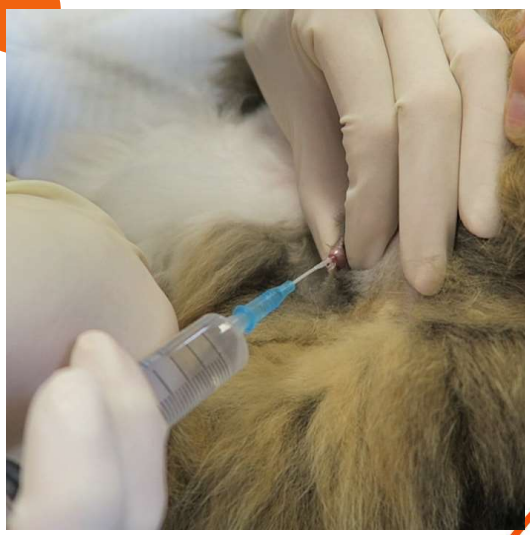
Sedation vs GA

- In many cases, a cat may be unblocked without a full GA.
- So go low and slow with the sedation/GA
- Avoid the following if possible: ACP/thio/domitor/propofol/ gas

Combination of opiod + benzodiazepines is usually sufficient

- Temgesic 0.02mg/kg IV or Methadone 0.2 mg/kg IV
- Midazolam/Diazepam 0.25mg/kg IV
- ± Alfaxan as needed (Ketamine can be used in cardiovascular stable patients)

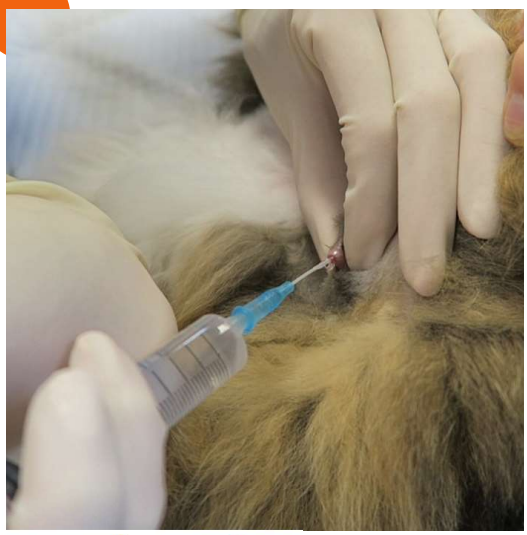
CATHETERISATION



Imagie: https://minivetguide.com/temporary-catheters-in-obstructed-flutds/

- Shave wide
- Sterile prep
- Use a drape
- Use sterile gloves

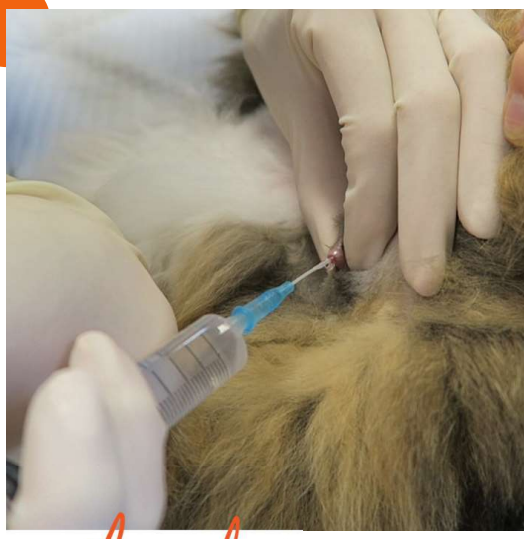
CATHETERISATION



Imagie: https://minivetguide.com/temporary-catheters-in-obstructed-flutds/

- I prefer lateral recumbency but many clinicians report dorsal recumbency to be much easier
- Extrude penis
- Massge penis tip with KY gel using my fingers - many mucous plugs sit just at the tip
- I use 22G IV jelco (stylet removed)
- Sterile saline, drawn up in 3ml syringes and hydropulse as I am advancing the catheter
- Short sharp flushes
- Once the IV catheter can pass up to the hub I switch to a stiff Tom Cat catheter
- Multiple size catheters may be needed and in some tough to dislodge stones it can take a while.
- Some times rotating the catheter between thumb and index finger while advancing helps

CATHETERISATION



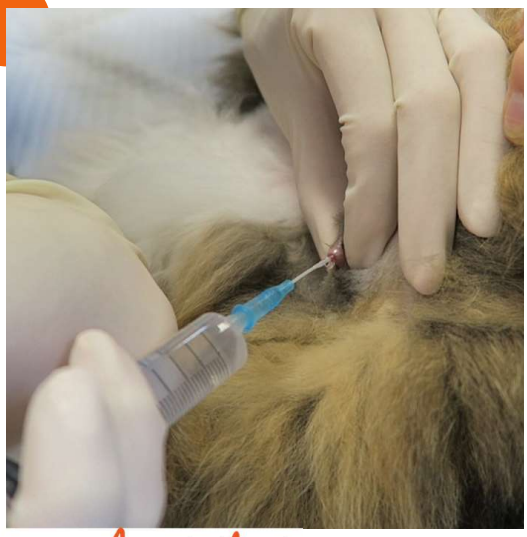
Imagie: https://minivetguide.com/temporary-catheters-in-obstructed-flutds/

- The biggest trick is to pull the prepuce over the penis caudally and toward to tail (dorsally) to straighten the urethra out over the pelvic brim to allow easy catheter advancement and prevent iatrogenic trauma

- NEVER EVER FORCE the catheter, majority of iatrogenic urethral rupture occur at the dorsal wall of the urethra at the point where the urethra passes over the pelvic brim.

- If some fluid is going in but not all is coming out keep checking the bladder. Iatgrogenic bladder rupture can occur with a partial blockage and you may need to intermittenly decompress cystocentesis

CATHETERISATION



Imagie: https://thevetguide.com/temporary-catheters-in-obstructed-flutds/

- Spasms can occur during unblocking and small volumes of lignocaine in the urethra can help
- 0.25ml - 0.5ml, wait 5 min then start again
- Total Dose: 2-4 mg/kg in the cat (NB! toxic in the cat at 6-10 mg/kg)

- If struggling remove catheter and ensure it has not come blocked.

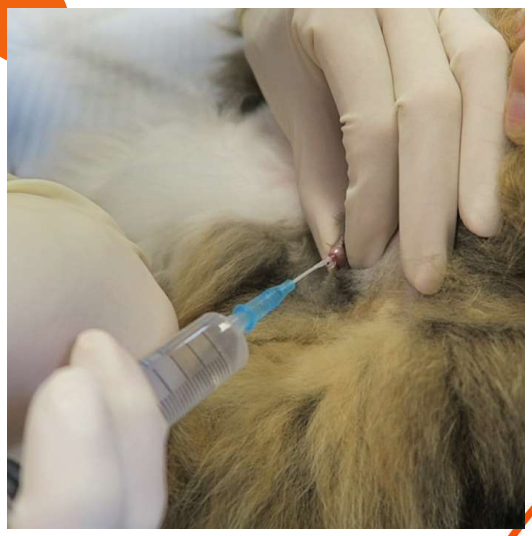
CATHETERISATION



Imagie: https://thevetguide.com/temporary-catheters-in-obstructed-flutds/

- Once in, collect a urine sample
- Flush the bladder with saline well, espesially if there is sandy substance
- The replace the catheter used to unblock with a soft indwelling one.
- 3.5 - 5 FG > Slippery Sam are my favourite but you can use a red rubber feeding tube (measure before - 2cm over pelvic brim)
- Secure to the prepuce with sutures
- Connect to a closed collection system (human urinary collection bag vs old IV drip bag)
- Secure to the tail, be sure to allow enough slack so when the tail is in upright position its not tugging on the penis/prepuce
- Buster collar

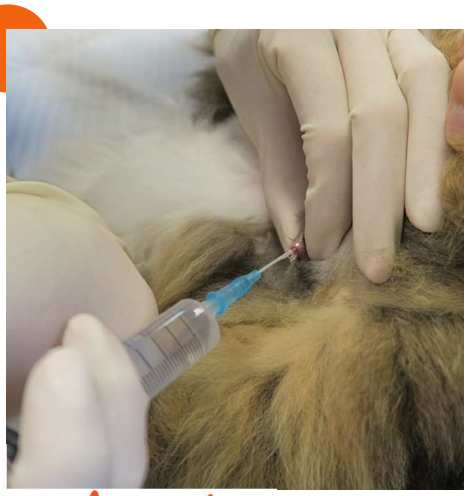
ALTERNATIVES



Imagie: https://minivetguide.com/temporary-catheters-in-obstructed-flutds/

- What to do if you cannot unblock
  - Repeated cystocentesis
  - Cystostomy
  - Cystotomy
  - Perineal urethrostomy

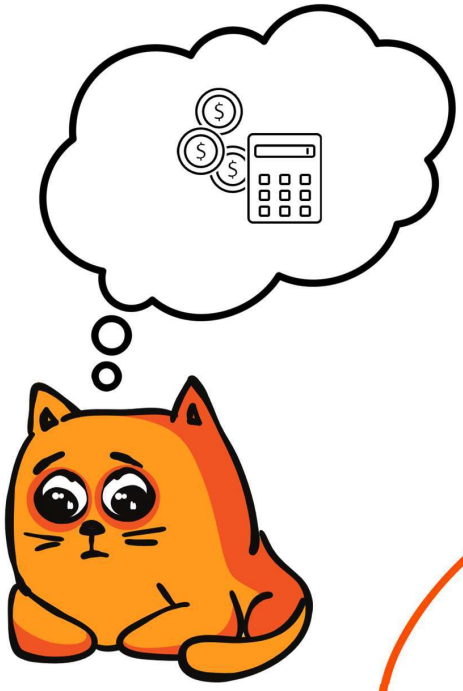
POST CATHETERISATION



Imagie: https://minivetguide.com/temporary-catheters-in-obstructed-flutds/

- Post obstructive diuresis
- Measure urine output q 4hrs. I try match ins and outs as much as possible.
- Recheck lytes every 4-6 hrs until K is normal and then every 12-24 hrs
- AB???? I do give if I have seen intracellular bacteria on in house UA (smear) and feel this has been a contributing factor to the blockage
- Culture and sens at U cath removal is useful
- Consider prazocin (smooth muscle) / dantrolene (skeletal muscle)
- Pain control - temgesic, gabbapentin
- Leave U Cath in for at least 24 hrs or until urine is clear ~ 3 days
- Warn owners of reblocking after U cath removal

COST SENSITIVE CASES



Action plan for the cost sensitive client

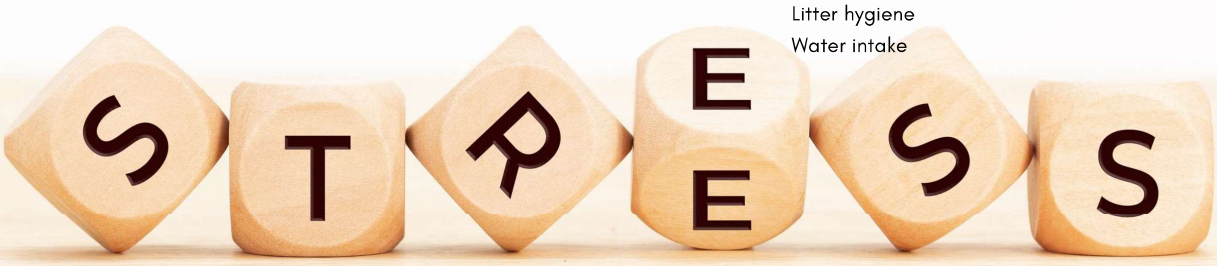
We can assume a lot based on basics, assume high K, assume azotemia, so we can skip a lot of the expensive diagnostics. Place U cath for at least 1 day, start on oral meds- prazocin/dantrolene/temgesic/gabbapentin

Unfortunately these are often repeat offenders and due to the multifactorial aetiology it can be very challenging to manage long term for the cost sensitive owner

CLIENT COMMUNICATION

Prognosis & Long Term Management

Education
Diet options - wet vs dry, urinary s/o, calm
Weight management
Stress - environmental vs medication
Litter hygiene
Water intake



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