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Practical Techniques for Emergency Patients Mini Series

Session Three: Emergency Therapeutic Procedures

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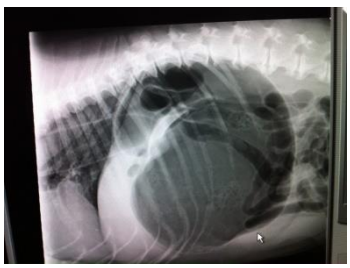

Practical Techniques in the Emergency Room: Emergency Therapeutic Procedures

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This webinar will build on the material covered previously, including several procedures that may be performed in the emergency room when stabilising patients and managing common emergency presentations.

Treatment of pleural space disease

In any patient with suspected pleural space disease, thoracocentesis may be both diagnostic and life saving. There is a low complication risk, although cardiac/vena cava puncture may occur or a pneumothorax may develop (usually subclinical). Thoracocentesis may usually be performed successfully with minimal or no sedation in animals with enough pleural space disease to cause overt respiratory distress. Where sedation is required, low doses of either butorphanol (0.1-0.4 mg/kg) or methadone (0.1-0.2 mg/kg) may be considered, alone or in combination with a benzodiazepine (midazolam 0.1-0.3 mg/kg) if considered necessary.

To perform a thoracocentesis:

- Oxygen should be provided during the procedure
- The thorax should be clipped ventrally for fluid and dorsally for air
- The area should be cleaned with surgical scrub and spirit although surgical asepsis is not necessary
- The choice of needle is dictated by patient size and space occupier; in cats a butterfly needle is usually sufficient whereas in larger dogs it may be easier, and quicker, to use a longer larger gauge catheter or needle
- The patient is typically positioned in sternal recumbency. In patients with severe orthopnea, a standing position may be preferred to facilitate continued elbow abduction and free movement of the sternum for full chest expansion. In either case, the needle or catheter should be inserted at rib space 7-8, approximately 1/3 of the way from the bottom for fluid and 1/3 of the way down from the top for air (or at the point of dullness or as guided by ultrasound). A line moving directly upwards at the level of the xiphoid process can serve as a useful marker for intercostal space 8 in an emergency situation to avoid counting rib spaces. If the patient is positioned in lateral recumbency and is suspected to have a pneumothorax, the needle may be inserted at the widest part of the chest to locate the air pocket that is likely to have formed. Alternatively, the site of needle entry may be determined on the basis of TFAST imaging if a suitable pocket of fluid has been identified in cases of pleural effusion.
- The needle should be angled perpendicularly to the chest wall and placed cranial to the rib, to avoid blood vessels and nerves running along the caudal aspect of each rib

- A sudden loss of resistance is often detected as the needle enters the pleural space. Alternatively, as the needle is advanced to a seemingly appropriate depth, a small amount of pressure can be applied using a 5 ml syringe. Once fluid or air has been obtained, the bevel of the needle should be reorientated ventrally or laterally so that it sits parallel to the thoracic wall, thereby reducing the risk of laceration to the underlying lung tissue.
- When the needle is in place within the pleural space, it should be stabilised by resting part of the hand or several fingers against the lateral thoracic wall. In the event of sudden patient movement, this may allow concurrent movement of the needle, reducing the likelihood of accidental needle removal.
- A 3-way tap should be used to allow repeated drainage into a syringe while not allowing air to leak into the chest.

As the pleural space is evacuated it is common to see a rapid improvement in the patient's respiratory rate and breathing pattern, with the chest excursions improving as the lung is able to expand more fully. Fluid samples should be stored in a sterile container for culture (aerobic and anaerobic) and in EDTA for cytology. It is good practice to collect any samples at the start of the drainage procedure, to ensure their collection and to minimise any contamination of the sample.

Trouble-shooting thoracocentesis: If negative pressure is obtained during the procedure, the needle may be gently redirected to see if any further fluid or air may be obtained. If a catheter is being used, it should be examined to rule out the possibility of kinking, especially at the site of insertion into the chest, which can prevent pleural contents from being removed. It is also possible that a catheter or needle may become blocked by either fibrin or blood within the pleural cavity. If this occurs, apply a small amount of positive pressure on the plunger of the syringe to clear the needle (this can be facilitated by leaving a small volume of fluid or air in the collection syringe during emptying). Alternatively, use of a smaller syringe may help to resolve the problem by decreasing the amount of suction at the needle tip. Ideally no more than 5 cm of negative pressure should be applied with the syringe to make this occurrence less likely. In severe cases where there is repeated occlusion of the needle, it may be necessary to place a chest drain to allow complete drainage of the pleural space.

Thoracocentesis in patients with chylothorax may have additional challenges. Chyle is a bacteriostatic fluid that irritates the pleural and pericardial surfaces. Chronic chylous effusion may lead to severe fibrosing pleuritic and pericarditis. This may be suspected when the lungs appear rounded on a thoracic radiograph in the presence of minimal pleural fluid. It is not uncommon for a pneumothorax to develop in this patient population following thoracocentesis, which may be severe. Patients should be monitored closely during and following the procedure and consideration given to removing only sufficient fluid to relieve respiratory distress and hypoxia in cases with a confirmed diagnosis.

If air or fluid is rapidly re-accumulating in the chest after thoracocentesis has been performed, then unilateral or bilateral chest drains may be placed to allow for repeated or continuous drainage.

Common examples include traumatic pneumothorax, pyothorax and chylothorax. Trocar chest drains were previously used for this purpose. Their routine use has fallen out of favour owing to concerns for their large size, need for general anaesthesia for their placement and concerns for damage to intra-thoracic structures when placed using a force technique in which the drain in held perpendicular to the chest wall and placed with a sharp blow of the hand. If used, the drain should be placed using haemostats or similar to create a subcutaneous tunnel and their tips subsequently used to penetrate the intercostal muscles and gain entry to the pleural space. A full picture guide to placement is currently available on the Vygon website (www.vygonvet.co.uk).

MILA chest drains are being used more commonly for management of pleural space disease, working well in the majority of patients. They are of a smaller size and can be placed under sedation allowing a more rapid recovery in comparison to trocar type drains requiring general anaesthesia for placement. The MILA chest drains are placed using a Seldinger technique (over the wire approach), can be maintained for several days and as one size can be used in many patients limit the need to have several sizes of chest drain in stock at any time. They can be placed in one or both sides of the chest as required. A full picture guide and video of the placement is currently available on the MILA website (www.milainternational.com). Whilst intermittent pleural drainage is likely to be adequate in the vast majority of patients, continuous pleural drainage may be required in a small proportion of cases. A commercially available continuous drainage system may be connected to the chest drain, or a Heimleich (flutter) valve considered in patients with pneumothorax.

A recent case series described the use of an autologous blood-patch for the treatment of persistent pneumothorax in 8 dogs. Non-coagulated blood, at a volume of 5-10 ml/kg was collected from the jugular vein and then immediately placed into the pleural space via chest tube or thoracocentesis needle. A short course of antimicrobial treatment was provided to each of the dogs following the procedure. The procedure was reported to be successful in 7/8 of the cases, although was repeated in several cases. It appears to have been well tolerated although infections were reported in 2/8 cases as a complication. Its use may be considered in patients with persistent pneumothorax as an alternative to euthanasia.

Essentials of Cardiopulmonary resuscitation (CPR)

Cardiopulmonary resuscitation (CPR) is used to treat cardiopulmonary arrest (CPA), an otherwise fatal condition. Despite ongoing interest in this area, survival to hospital discharge rates have historically been reported to be around 6-7 %. In 2012, the RECOVER initiative (Reassessment Campaign on Veterinary Resuscitation), via an exhaustive literature review of all human and animal CPR research, generated the first set of veterinary-specific evidence based guidelines as to best practice in veterinary CPR in an attempt to improve outcomes. These guidelines may be accessed free of charge at http://www.acvecc-recover.org/. Drug dose sheets and CPR algorithms to facilitate CPR in practice may also be obtained from this website free of charge.

Preparedness and prevention

Early recognition of and response to CPA are both essential for a successful outcome. If left untreated, cerebral hypoxia results in complete biologic brain death within 4-6 minutes of CPA. In the majority of cases undergoing CPA in small animal practice, it is as a result of sedation and anaesthesia, and these cases are the most likely to have a successful outcome. Any condition causing an abnormality within one of the major body systems may however progress to CPA, leading to a more challenging case in which to obtain return of spontaneous circulation (ROSC) and survival to hospital discharge. Common causes of CPA in dogs and cats include vagal stimulation, hypovolaemia, traumatic injuries such as pneumothorax, severe electrolyte disturbances such as hyperkalaemia, acid base disturbances, cardiorespiratory disorders such as congestive heart failure, hypoxia or pericardial effusion, and severe systemic conditions such as neoplasia and sepsis. Potential signs of impending CPA include:

- Dramatic changes in respiratory rate, effort or rhythm (such as agonal breathing, a decreased rate or sudden increased rate)
- Significant hypotension (systolic blood pressure < 50 mmHg, normal > 90-100 mmHg)
- Irregular or inaudible heart sounds
- Changes in the heart rate or rhythm
- Change in mucous membrane colour (white or cyanotic)
- Fixed, dilated pupils
- Patient distress and/or vocalisation
- Collapse

A centrally located and easily available crash cart or container, containing all the necessary drugs and CPR equipment, should be maintained in the practice and regularly audited. Easily visible aids including CPR drug dosing charts and CPR algorithms can also be a useful prompt and ensure CPR is delivered promptly and well. Regular staff training including CPD to improve knowledge base and practical scenario role play using the practice equipment will also be beneficial. A debrief should also be performed following any incidence of CPA to evaluate team performance and critically assess areas that can be targeted for future improvement, and also serve as a refresher for those present. Effective CPR requires several people to be involved. Ideally one person should ‘run’ the CPR attempt, giving clear commands to the other members of the team, monitoring the effectiveness of the attempt and giving real time feedback. If there are sufficient personnel, one person should be given the role of recording the CPR attempt, listing the timing and doses of any drugs administered and any response to therapy noted. Closed loop communication should also be used during CPR to avoid miscommunication. The loop is considered closed when the team leader gives a message, the receiver repeats this back, and the team leader then confirms the message typically by saying ‘yes’. Commands should also be directed towards specific people to ensure that tasks are actioned and that other people can continue their roles otherwise undisturbed.

Having a standardised assessment to detect CPA as soon as possible is essential. It should be applied immediately to any patient that becomes acutely non-responsive. CPA can be assumed to be present in any non-anaesthetised patient who is unconscious and not breathing. This initial assessment should take at most 10 seconds and consists of a brief evaluation of airway (A), breathing (B) and circulation (C). If it is unclear, then it should be assumed that CPA has occurred and CPR commenced immediately rather than pursuing additional testing. This approach confers minimal risk to the patient and avoids any delay in starting resuscitation which is known to markedly decrease survival rates in people.

Basic life support

Basic life support (BLS) includes chest compressions to restore blood flow to the tissues, and ventilation for oxygenation of arterial blood and removal of carbon dioxide from the venous blood. *Performing good quality BLS is likely to make the most significant difference to outcome during CPR.*

Chest compressions

It is essential that chest compressions are initiated as soon as possible after CPA is recognised and if sufficient personnel are present, should not be compromised in order to obtain airway access or ventilation. Effective chest compressions form the cornerstone of successful CPR and the goal is to maximise myocardial and cerebral perfusion. The patient should be placed in either left or right lateral recumbency and external chest compressions performed at a depth of one third to one half the width of the chest, and at a rate of 100-120 compressions per minute regardless of animal size or species. A two handed technique should be used in the vast majority of cases with the hands laid on top of one another, the elbows locked and the shoulders positioned directly over the hands. This technique engages the core muscles and allows sustained compression force and reduces responder fatigue.

There are 2 major theories as to how blood flow occurs during external chest compressions:

1. Cardiac pump theory – in smaller dogs, those with a keel-chest conformation (such as Greyhounds), and cats, chest compressions are performed directly over the heart. Arterial blood flow is a result of the direct compression of the ventricles and blood is allowed to flow back into the heart during the phase of decompression, when the pressure is released. This mechanism therefore works well in small patients and those with compliant chest walls.
2. Thoracic pump theory – in larger patients, chest compressions should be performed more caudally, over the deepest part of the chest rather than directly over the heart. Closed chest compressions directly over the heart would not be able to generate sufficient force to empty the ventricles sufficiently. Forward arterial blood flow in larger patients therefore occurs as a result of a generalised increase in intra-thoracic pressure which drives blood out of the chest. The resultant negative pressure during the phase of decompression allows blood to return back to the thorax and blood flow to continue.

Regardless of the theory utilised for compressions during CPR, it is imperative that compressions are continued with minimal interruptions once commenced to maximise continued blood flow to the major organs. Cycles of 2 minutes of uninterrupted compressions should be used since it can take upwards of one minute to achieve maximal blood flow to the heart. Assessment of the ECG arrest rhythm and pulse palpation requires a brief pause in compressions and should be accomplished during a brief cessation (2-5 seconds only) of compressions at the end of each 2 minute BLS cycle. A new team member should take over chest compressions for each new cycle to prevent fatigue and inadequate compressions being performed. It is also essential that the chest be allowed to recoil fully between compressions to allow to refilling of the cardiac chambers prior to the next compression. Recoil is often one of the first things to be compromised by compressor fatigue, in which case 'leaning' on the chest may be noted.

Interposed abdominal compressions may be used to facilitate venous return from the abdomen and improve cardiac output. Abdominal trauma as a result is not a common occurrence and so it may be considered when sufficient personnel are present. BLS provision otherwise should not be negatively impacted to allow for abdominal compressions to be performed.

Ventilation – airway and breathing

Patients should be intubated and ventilated as soon as possible after chest compressions are commenced. Any foreign material, vomitus or other debris seen during initial airway assessment should be rapidly cleared with suction as part of initial airway management. Intubation should be performed with the patient in lateral recumbency to minimise interruptions to chest compressions; this is a skill that may be practiced at other times to ensure proficiency during CPR. The endotracheal tube should be placed using a laryngoscope to ensure rapid correct placement with minimal increase in vagal tone caused by laryngeal stimulation. The cuff should be inflated and the tube secured in place to prevent dislodgement. A stylet may be used to facilitate tube placement in more challenging cases, and a tracheostomy may need to be performed as an alternative during instances of complete upper airway obstruction where intubation is not possible with a smaller sized endotracheal tube or urinary catheter.

Ventilation should be performed with 100% oxygen at a rate of 10 breaths per minute, with an inspiratory time of 1 second and a tidal volume of around 10 ml/kg (equivalent to a normal respiratory chest excursion if tidal volume monitoring is not available). This low ventilation rate is adequate during CPR due to the low flow blood state and should not be exceeded so as to prevent decreases in cerebral and myocardial blood flow via hyperventilation causing hypocapnia and subsequent vasoconstriction. In addition, the resultant increase in intra-thoracic pressure caused by hyperventilation will also diminish venous return and the effectiveness of chest compressions. Ventilation and chest compressions should be performed simultaneously during CPR with no need to halt one to perform the other.

An Ambu bag or anaesthetic circuit (flushed of any anaesthetic gases) may be used to provide breaths although the former has the benefit of not having to ensure correct use of a pop-off valve to avoid excessive airway pressures.

Any lack of normal chest wall movement during ventilation, poor ventilation or an absence of lung sounds is suggestive of a poorly positioned endotracheal tube or severe pleural space disease such as pneumothorax, large volume pleural effusion or diaphragmatic rupture. Consideration should also be given to the length of the endotracheal tube in relation to the patient size to rule out single bronchial intubation via an overlong tube.

Monitoring during CPR

Largely as a result of lack of adequate pulse quality and patient motion artefact, many patient monitoring devices are of limited use. Pulse oximetry, doppler and oscillometric blood pressure measurement are all affected and do not provide useful data during CPR.

Palpation of the femoral pulse

Pulse palpation provides valuable real time feedback on the effectiveness of chest compressions in generating forward blood flow and cardiac output. A pulse should be felt for each chest compression and a worsening in pulse quality during CPR may indicate caregiver fatigue or poor technique that needs to be immediately addressed. Femoral pulses should also be checked during brief pauses in compressions to assess the patients ECG rhythm and determine whether there is ROSC, where pulses will be palpated in the absence of chest compressions being performed.

Electrocardiography

Many importance decisions in advanced life support are based upon the ECG arrest rhythm diagnosis. The ECG rhythm is highly susceptible to motion artefact and can only be interpreted during brief pauses in chest compressions between cycles of BLS. The team leader should clearly announce the rhythm diagnosis and check other team members agree, and chest compressions be resumed as soon as possible. The most common arrest rhythms in veterinary patients are asystole and pulseless electrical activity (PEA). Ventricular fibrillation (VF), although common in people, is far less common in veterinary patients with CPA.

Capnography

End tidal CO_2 monitoring can be very useful during CPR and is resistant to motion artefact so can be assessed continually. The $ETCO_2$ value can be used as a marker of the efficacy of chest compressions because when minute ventilation is constant (as in the case of CPR where ventilation occurs at a constant rate), $ETCO_2$ is proportional to cardiac output. A very low $ETCO_2$ value during CPR (< 10-15 mmHg) has been associated with a reduced likelihood of ROSC (Return of Spontaneous Circulation) in both dogs and people. A low value obtained during CPR should therefore prompt immediate assessment of chest compression technique and change made as indicated.

Consideration should also be given to an underlying disease process that may be impairing chest compression efficacy, such as pericardial or pleural space disease. An $ETCO_2$ of at least 15 mmHg should be targeted during CPR. The $ETCO_2$ reading will also substantially increase upon ROSC and so can be a useful early indicator of ROSC during CPR.

Advanced life support

Advanced life support (ALS) consists of drug therapy and electrical defibrillation, if indicated, once BLS procedures have been started. It is important to note that the addition of any ALS procedures should not negatively impact the delivery of good quality BLS as in the absence of good BLS, ALS procedures are unlikely to be successful.

Drug therapy

Drug therapy should ideally be delivered intravenously or via the intra-osseous route. The most central blood vessel closest to the heart should be used when possible to maximise drug delivery and action. Should the patient not have established venous access at the time of CPA, a vascular cut down is often required. If this is not practical or possible, certain drugs may also be delivered via the endotracheal tube, as represented by the following mnemonic 'NAVEL'. Drug doses usually need to be increased when delivered via this route.

N – Naloxone
A – Atropine
V – Vasopressin
E – Epinephrine (adrenaline)
L - Lidocaine

Intra-cardiac drug delivery is no longer recommended due to concerns for serious myocardial injury and the need for a cessation of chest compressions during administration. As a valuable part of CPR preparedness, a drug dosage chart should be in plain view in the CPR ready area of the hospital and/or specific drug calculations carried out for patients considered to be at high risk of CPA and keep with the patient's notes.

Specific drugs that may be used during CPR include vasopressors, parasympatholytics, reversal agents, anti-arrhythmic drugs, intravenous fluids, and alkalinising therapies.

Vasopressors

Vasopressor therapy is very important during CPR in order to redirect blood flow to the vital organs via peripheral vasoconstriction, regardless of the inciting cause of the arrest. The catecholamine epinephrine (adrenaline) is the most commonly used vasopressor drug in CPR and its vasoconstriction is mediated via alpha-1 receptor agonism. The vasoconstrictive effects of epinephrine predominate in the periphery while sparing both myocardial and cerebral vasculature and preserving blood flow to these organs.

Early in every CPR attempt epinephrine should be administered at a low dose of 0.01 mg/kg IV/IO and can be given every other cycle of CPR. After prolonged CPR (> 10 minutes), a higher dose of 0.1 mg/kg IV/IO may be considered as it may be associated with a higher rate of return of spontaneous circulation. The higher dose should be reserved for this scenario only as its routine use is associated with worsened neurologic outcome in people.

Epinephrine may also be administered via the endotracheal route at a dose of 0.02 mg/kg (low dose) or 0.2 mg/kg (high dose). The drug should be given after feeding a long catheter down the ET tube and diluting the drug 1:1 with isotonic saline or sterile water.

Vasopressin may be considered as an alternative to epinephrine. It is a vasopressor drug that acts via peripheral V1 receptors and is given at a dose of 0.8 u/kg IV/IO. It has the potential benefit of being effective in an acidic environment, unlike epinephrine where alpha receptors may become unresponsive to the drug when blood pH decreases. It may also be given endotracheally and can be used interchangeably with epinephrine or in combination with it during CPR.

Parasympatholytics

Atropine in the most commonly used parasympatholytic drug used during CPR. Its use may be considered in all cases of CPR at a dose of 0.04 mg/kg IV/IO every other cycle of CPR. It should definitely be given in cases with asystole or PEA associated with an increase in vagal tone, such as as seen with gastrointestinal, respiratory or ocular disease. Brachycephalic breeds should also be given parasympatholytic therapy as standard during CPR. Atropine may also be administered via the endotracheal tube at a dose of 0.08 mg/kg (double the standard dose).

Reversal agents

Reversal agents should be administered as soon as possible during CPR if any reversible sedative or anaesthetic drugs were given to the patient prior to CPA. These drugs should also be immediately discontinued if they are still being administered at the time of CPA. Commonly used reversal agents include:

- Naloxone (0.04 mg/kg IV/IO) for opioids
- Flumazenil (0.01 mg/kg IV/IO) for benzodiazepines
- Atipamezole (0.05 mg/kg IV/IO) for alpha-2 agonists

Intravenous fluid therapy

Intravenous fluids should only be administered to patients with hypovolaemia, in which case they are beneficial in helping to restore preload, may increase the efficiency of chest compressions and can improve blood flow to the myocardium and brain. Intravenous fluids may be harmful if given to hypervolaemic or euvolaemic patients as they tend to increase central venous pressure (and hence right atrial pressure) rather than arterial blood pressure in patients with CPA.

Elevations in right atrial pressure may subsequently decrease blood flow to both the myocardium and brain, worsening outcome.

Alkalinising therapy

Sodium bicarbonate therapy is indicated in patients with pre-existing acidaemia thought to have contributed to CPA, and in cases of prolonged CPR (> 10-15 minutes) where severe acidaemia often occurs due to concurrent lactic acidosis and hypercapnia from carbon dioxide build up. Acidaemia can result in severe vasodilation, cardiac dysfunction and inhibition of normal enzymatic and metabolic activity. Sodium bicarbonate is given intravenously at a dose of 1 mEq/kg, once, diluted. Its use should ideally be reserved for cases with severe acidaemia (pH < 7) of a metabolic origin as determined by blood gas and acid base analysis. Alternatively, it may be used on the basis of CPR duration without blood gas measurement where unavailable in refractory cases.

Other drugs

Calcium gluconate may be useful in cases with hyperkalaemia and/or hypocalcaemia that may have contributed towards CPA. Dextrose should also be considered in cases with hypoglycaemia. These drugs should be avoided unless being used to treat a specific disturbance as empirical use may be harmful to the myocardium and/or brain.

Defibrillation

Electrical defibrillation is the recommended treatment for ventricular fibrillation (VF) and pulseless ventricular tachycardia (VT). Both of these arrest rhythms are seen less commonly that asystole and pulseless electrical activity in small animal patients, the non-shockable rhythms. The recommended timing of defibrillation varies depending on how many minutes of CPA have elapsed, based on what is known about the progression of myocardial ischaemia. If the duration of VF is known or suspected to be 4 minutes or less, chest compressions should only be continued until the defibrillator is charged and then a shock delivered immediately. If the patient has been in VF for more than 4 minutes, one full cycle of chest compressions should be performed prior to defibrillation; this allows for blood flow and oxygen delivery to the myocardial cells and makes them more likely to respond favourably. The energy dose varies depending on the type of defibrillator used, monophasic or biphasic, the latter being preferred as they can defibrillate at a lower energy level resulting in less myocardial damage. Dosing for monophasic defibrillation begins at 4-6 J/kg, whilst for biphasic it begins at 2-4 J/kg. The dose may be increased by 50 % with each defibrillation attempt up to a maximum dose of 10 J/Kg. After each shock chest compressions should be resumed immediately and a full 2-minute cycle of BLS performed before a brief pause to check the ECG rhythm. If the patient is still in VF, debrillation should be repeated at the end of this cycle of BLS.

If a defibrillator is unavailable, a precordial thump may be considered as an alternative treatment option for a patient in ventricular fibrillation. A precordial thump provides mechanical defibrillation and is performed by striking the patient with the heel of the hand directly over the heart.

Open chest CPR

Open chest CPR is considerable more invasive and costly in comparison to closed chest CPR. It should be considered early in cases where standard closed chest CPR is unlikely to be effective. Examples of this include large or giant breed dogs especially those with a round or barrel-chest conformation, penetrating thoracic injuries, pericardial effusion and significant pleural space disease. If a patient is in abdominal surgery at the time of CPA, the heart can be most easily accessed via an incision through the muscular portion of the diaphragm, avoiding the need for thoracotomy.
In other cases, to perform open chest CPR, a left lateral thoracotomy is performed in the 4-5th intercostal space whilst the patient is in right lateral recumbency, and Finochietto retractors used to gain access to the thoracic cavity. The pericardium should be removed to facilitate compressions and address any pericardial effusion or disease, and the ventricles are compressed from apex to base to facilitate blood flow. If ROSC is achieved, then intensive care is required after the thoracotomy incision is closed and a chest tube placed. Open chest CPR should not be performed in situations where the required aftercare cannot be provided due to cost or practical considerations.

Practical techniques for toxicity cases

Intravenous lipid emulsion (ILE, typically 20% Intralipid™) is a cheap, readily available preparation that is being increasingly used to treat a variety of lipid-soluble toxicities in veterinary medicine. ILE has been used as part of both total and partial parenteral nutrition in veterinary and human medicine, as well as a vehicle for drug delivery (eg propofol). The therapy is typically administered through a peripheral vein as an initial intravenous bolus followed by a constant rate infusion at doses provided below.

There are several current theories as to how ILE works in the poisoned patient, including:

- Acting as a lipid sink by sequestration of lipid soluble toxins in the newly created intravascular lipid compartment following IV administration. Compartmentalisation of the toxin into the lipid phase results in a decreased free drug concentration available to the tissues.
- There is also evidence that the ILE may be able to draw lipid soluble toxins from the tissues where they are exerting their toxic effects back into the intravascular lipid compartment for excretion from the body.
- ILE may provide cardiac myocytes with energy substrates from the breakdown products comprised of free fatty acids

In veterinary medicine, the use of ILE has been suggested for a variety of toxicities including macrocyclic lactones (moxidectin, ivermectin), lidocaine, pyrethrins, calcium channel blockers and tremorogenic mycotoxin ingestion but its use may be considered for the treatment of any lipid soluble toxicity. At the present time, doses extrapolated from human medicine and parenteral nutrition have lead to a veterinary dosage recommendation of an initial bolus of 20% ILE solution of 1.5-4 ml/kg over 1 minute, followed by a constant rate infusion of 0.25 ml/kg/min over 30-60 minutes as a conservative dosing regime.

In patients that are not-responding following this initial dosing strategy, intermittent bolus therapy at 1.5 ml/kg every 4-6 hours may be considered and can be followed by a constant rate infusion of 0.5 ml/kg/hr to be continued until there is resolution of clinical signs (not to exceed 24 hours), or until the serum is lipaemic.

At the present time there is little evidence to support the use of ILE in toxicity cases. There are several case reports and anecdotal experience available, but there have been no safety studies evaluating its use in veterinary patients. Complications of therapy in people are reported to include fat embolism and pancreatitis. Although these have not been reported to date in veterinary medicine, ILE should be used judiciously and a risk benefit assessment made of treating each individual case. It should not be used as a substitution for standard decontamination and stabilization of the poisoned patient. The most recent veterinary literature should be consulted for updates as to its use as more clinical experience is obtained.

Practical tips in for the management of feline urethral obstruction

The majority of emergency and critical care lectures discussing urethral obstruction focus on the metabolic instability that can be seen in such cases. It is vitally important that these cases are recognised and appropriately treated in order to reduce morbidity and mortality but equally it is useful to realise that only 12% of cats presenting to general practices with urethral obstruction are hyperkalaemic. Whilst is is ideal to measure serum electrolytes and renal parameters in every blocked cat, this can add to the cost of treating such patients.

A retrospective study of a large cohort of cats with urethral obstruction (Lee and Drobatz 2006) found there are several factors that can identify an increased likelihood of hyperkalaemia. These included the episode being the patient's first one, the cat going outdoors, being anorectic and/or vomiting. Additionally, physical examination findings of a heart rate < 120bpm and/or a rectal temperature < 35.9^{o}C were highly specific for the presence of hyperkalaemia. If none of these risk factors is identified and finances are a significant concern, then it may be reasonable to proceed on the balance of probability that the cat does not have significant metabolic derangements. This should be re-evaluated following relief of the obstruction as dark urine colour has been associated with the presence of azotaemia so if this is present, bloodwork and fluid therapy are likely indicated.

The standard of care for urethral obstruction involves relieving the obstruction through urethral catheterisation, typically under sedation or anaesthesia. General anaesthesia may be avoided and light sedation used instead if this is combined with local anaesthesia. A coccygeal nerve block using 0.1-0.2 ml/kg of 2% lidocaine (without preservative or adrenaline) injected into the epidural space has been demonstrated to effectively analgese and anesthetise the penis and can be highly effective in this setting. The nerve block is performed at the level of the sacrococcygeal joint (which can be identified by mobilizing the tail), or alternatively between the 1st and 2nd coccygeal vertebrae.

At the spinal cord of the cat typically ends around S1, subarachnoid injection and resultant complications are thought to be very unlikely. The block is typically effective within 5 minutes of administration and can provide analgesia for around an hour. Systemic absorption of lidocaine is considered to be highly unlikely.

When passing a urinary catheter to relieve the urethral obstruction, the catheter should be adequately lubricated and placed as far as possible into the urethra. Once the catheter is seated in the distal urethra, the entire prepuce should be pulled caudally in order to straighten the path of the urethra and facilitate catheter placement. The catheter should subsequently be gently advanced, using a twisting motion if necessary, in order to relieve any obstruction.

If this is not successful, the following steps should be considered:

- Change patient positioning as necessary
- Ensure adequate analgesia and sedation to limit muscle spasm at the level of the urethra that may hinder catheterization
- Vigorously flush using sterile saline whilst continuing attempts to advance the urinary catheter as described above
- Sterile lubricant may be added to the flush solution to facilitate catheter placement
- A low dose of intra-urethral lidocaine may be considered for topical urethral analgesia and to limit any urethral spasm.
- A small over the needle catheter may be used in challenging cases as an alternative to bypass or break down very distal urethral obstructions
- A guide wire may be placed as a means of bypassing a fixed obstruction, and then used as a line over which to advance a small gauge urinary catheter

If passage of a catheter is still challenging then atracurium besylate (4ml of 0.5 mg/ml solution) can be instilled into the distal urethra and this has been shown to be effective in relaxing the urethra, aiding successful passage of the catheter and reducing the time taken for catheterization to occur.

Studies indicate that re-obstruction rates following successful catheterization are lower if a 3.5 Fr catheter is used rather than a larger size. Leaving a catheter in place for a period of time rather than removing it immediately after relief of the obstruction is also associated with a lower rate of re-obstruction, as is the use of prazosin (0.2-1mg/cat BID), all of which may reduce morbidity and costs.

Although urethral catheterisation is typically required to relieve obstruction, a recent small scale study described a protocol without catheterisation. It consisted of sedation and analgesia (acepromazine 0.25 mg IM or 2.5 mg PO every 8 hours, buprenorphine 0.075 mg PO every 8 hours, and medetomidine 0.1 mg IM every 24 hours), combined with decompressive cystocentesis and subcutaneous administration of fluids as needed. Cats were also placed in a quiet, dark environment to minimise stress. This protocol may be an option for cases with a functional blockage only (no evidence of urolithiasis or urethral plugs) and financial constraints which preclude catheterisation.

All of the cats in this study were confirmed to be metabolically stable with minimal bloodwork changes, normal rectal temperature and heart rate. ACP and medetomidine should only be used in stable patients (neither hypovolaemic nor dehydrated) and the complication rate in a larger cohort of patients is yet to be determined.

Emergency transfusion medicine considerations

Autotransfusion

If a blood transfusion is thought to be indicated in a patient with acute haemorrhage and no suitable blood products are available, then autotransfusion of the haemorrhaged blood can be considered as a life-saving alternative. This should only be considered if the blood remains in a sterile field such as a non-contaminated body cavity or a surgically prepped field where it is then suctioned into a sterile container. Blood should not be used if haemorrhage occurred more than 24 hours ago as leukocyte aggregates and red cell lysis will potentially result in a severe systemic inflammatory response if this is administered intravenously. Blood from traumatic haemobabdomen should also be used with caution as autotransfusion is contraindicated in the presence of uroabdomen or injury to the biliary tree.

The use of blood from bleeding neoplastic lesions is controversial as there is concern that this fluid will contain viable neoplastic cells that will metastasise if given intravenously. This has not been reported in the veterinary literature but it is something that must be considered in a risk/benefit analysis when deciding if autotransfusion is truly beneficial for a patient compared to not transfusing it. In the author's opinion, if the alternative to autotransfusion is likely death then the benefits of the procedure outweigh the risk of further neoplastic lesions at a later date.

If blood is to be autotransfused then it should be given through a filter as with any blood product. If haemorrhage has occurred within the past hour, then it is necessary to add an anticoagulant to the blood to prevent it from clotting. This should be at the rate of 6ml of citrate per 60ml syringe or 8ml of CPDA per 60ml syringe. Blood that is older that 1 hour (but less than 24 hours) which has collected in a body cavity does not necessarily need an anticoagulant as fibrin will have been broken down but the use of an anticoagulant is of little harm and is still a sensible precaution.

Xenotransfusion

The availability of banked blood has revolutionised transfusion medicine in dogs but in the UK, banked feline blood is not commonly available and we instead rely on collection of fresh whole blood from a live donor once a need is identified. This has many practical and biologic advantages but it can also be very challenging to deal with if blood is needed in a faster time than a donor can be identified, sedated and phlebotomised. Over the years, numerous case reports have described the administration of canine pRBCs to cats.

It is recognised that this has a substantially higher risk associated with it compared to administration of feline blood but xenotransfusion of this nature is being increasingly recognised as a viable treatment option in cases of desperation where feline blood is not available and patients may die without any transfusion at all.
The literature on this subject was recently reviewed (Bovens & Gruffydd-Jones, JFMS 2013) and interestingly there were very few problems noted in the sporadic case reports available for a first transfusion episode. More recently, other studies have found that canine and feline blood demonstrate in vitro incompatibility at least, highlighting that this approach should not be considered as a first line therapy.

The guidelines currently in place for the administration of a xenotransfusion at the RVC are as follows:

- Aseptically insert an injection spike into a port on the canine unit enabling the blood to be draw up into 10 or 20ml syringes for administration (draw up one syringe at a time and leave the remaining volume in the bag in the blood fridge until needed).
- Deliver transfusion through a syringe driver and haemonate filter as per usual practice for feline blood transfusion administration.
- Begin the transfusion at 0.5ml/kg/hr for the first ½ hour and then increase to 1ml/kg/hr for another ½ hour if the product is tolerated then this rate can be increased as per the patient's requirements.
- Monitor the transfusion **very** closely especially for the first hour – TPR assessments should be performed **every 15 minutes for the first hour** and then reduced to every hour if appropriate.
- All transfusion complications or reactions should be reported.
- Each syringe should be administered over no longer than 6 hours and the entire transfusion should be administered within 24 hours of insertion of the injection spike.
- If a transfusion reaction is suspected, then the standard transfusion reaction guidelines should be followed.
- A xenotransfusion should not be repeated. Attempts should be made to source feline type specific blood within 3 days of xenotransfusion as rapid haemolysis of the transfused cells is likely within this time frame in the majority of cases.