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Emergency and Critical Care Nursing Mini Series

Session 2: Stabilising and Nursing the Critical Patient

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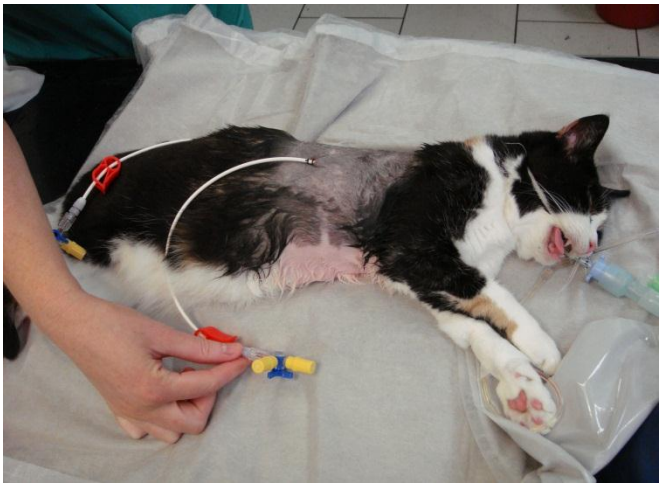

The Dyspnoeic Patient

Initial Presentation
Initial stabilisation of dyspnoeic animals requires that the inspired oxygen concentration is increased. Extremely dyspnoeic animals that cannot adequately ventilate and oxygenate even with oxygen supplementation may require anaesthesia to intubate and gain control of the airway, and to instigate positive pressure ventilation.

OBSERVATION

Initially observing the patient prior to a physical examination is invaluable in recognizing dyspnoeic patients.

Changes in ***POSTURE***- The patient will adopt positions to minimize any restriction to air flow, restraining the patient prevents these adaptations and can lead to decompensation. Postural changes include:

- Flared nostrils, open mouth breathing
- Extension of the neck, and lifting the head
- Abduction of elbows to minimize chest wall compression
- Sitting or lying in sternal recumbency, and shifting positions

Changes in ***BREATHING PATTERN***: Normal respiration is at 15-30 breaths per minute; little chest movement is seen as the diaphragm does most of the work. Inspiration is usually equal in length to expiration. Abnormalities include:

- Abdominal effort- contraction of abdominal muscles to help with expiration.
- Paradoxical breathing- severe dyspnoea, intercostals contraction draws the diaphragm forward and the abdomen is sucked in.
- Inspiratory phase longer than expiratory phase, or vice versa

PHYSICAL EXAMINATION

Cyanosis will only be present in severe hypoxaemia (<80% arterial blood saturation), so while it is definitely a sign supplementary oxygenation is required, the absence of cyanosis does not mean all is well, moderate hypoxic animal will still have pink mucous membranes. Pulse oximetry can be useful in confirming the presence of hypoxaemia.

Auscultation of the respiratory system as part of a rapid physical examination provides vital information to help localise the cause of respiratory distress. The cervical trachea, lung fields and heart should be auscultated. Abnormal sounds, or asymmetry in sounds from one lung compared to the other are relevant. Abnormalities may include:

- Wheezes?- narrowing on the airways likely (inflammation, masses etc). If on inspiration, upper airway pathology suspected. If occur on expiration, suspect lower airway disease, eg feline asthma.

- Crackles?- air bubbling through fluid, or opening and closing of small airways. Often indicate pulmonary oedema, haemorrhage or exudates in the alveoli.

- Muffled sounds?- suspect pleural space disease, liquid, air, or diaphragm rupture.

- Heart sounds- In dogs the absence of a murmur or dysrythmia means heart failure as a cause of dyspnoea is unlikely. This is more difficult in cats, as they usually suffer myocardial disease rather than valvular disease.

History, observation and physical examination should help to localise the cause of dyspnoea to one of four areas; upper airway, lower airway, lung parenchyma, or the pleural space. Having localised the source of the problem, specific stabilisation techniques can be applied where suitable.

Site	Respiratory pattern	Audible noise	Auscultation findings	Disease Example
Upper airway	Inspiratory effort	Yes	Referred upper airway noise	Laryngeal paralysis BOAS
Lower airways	Expiratory effort	No	Expiratory wheezes	Feline asthma
Pulmonary parenchyma	Mainly inspiratory but mixed patterns possible	No	Harsh sounds and/or crackles	Pulmonary oedema Pneumonia Pulmonary contusions
Pleural space	Short shallow inspiration	No	Dull lung sounds	Pleural effusion Pneumothorax

Oxygen Supplementation.

Oxygen is frequently administered to emergency patients as part of their stabilisation, as ongoing therapy, or pre-oxygenating prior to anaesthesia.

Remember the delivery of oxygen from the atmosphere to tissues relies on:

i) Oxygen being efficiently inhaled via the respiratory tract, to the alveoli.

ii) Oxygen being exchanged across the alevoli membrane.

iii) Oxygen being carried via haemoglobin in red blood cells.

iv) Red blood cells reaching the required tissues via the circulation.

So, any animal, with any problem at each stage of this process, will benefit from their inspired oxygen levels being increased:

Stage i) and ii)- dyspnoea, obstruction,pneumonia, pulmonary oedema.

Stage iii)- anaemia, or formation methaemaglobin due to toxicity.

Stage iv)- redcued cardiac output, hypovolaemia, distributive shock.

IF IN DOUBT, GIVE OXYGEN, DON'T JUST RESERVE IT FOR ANIMALS WITH RESPIRATORY PROBLEMS.

Initial stabilisation of dyspnoeic animals requires that the inspired oxygen concentration is increased. This can be done before and during a rapid thorough physical exam, then allow the animal to rest with further oxygen therapy prior to further work-up or treatment.

Dyspnea is a sensation of difficulty in breathing experienced by patients with compromised respiratory function. This sensation is caused by low oxygen, or high carbon dioxide partial pressures in arterial blood. These blood levels effect the ventilator motor centres in the brain- and this produces the clinical signs. So by increasing the oxygen levels in the blood, we can reduce the sensation, and get a less 'panicky' animal.

PRACTICAL OXYGEN SUPPLEMENTATION-

Inspired room air has an inspired oxygen concentration of approx 21%. By supplementing this level we can help our dyspnoeic patients. Several techniques exist, which one is selected depends on patient size, conformation, temperament, likely duration, and available equipment.

MASK;

A variety of sizes are available. Use on patients that are lying still and will tolerate the mask; distressed animals will struggle, panic and potentially decompensate.

Flow rates of 5-6l/min may increase inspired concentration to 70-80%

The mask must not be too tight fitting or rebreathing of carbon dioxide will occur.

Short term use, or periodic use. Useful for initial supplementation, or for pre-oxygenating prior to GA.

'FLOW-BY';

By holding the end of an anaesthetic circuit near to the nostrils or mouth of an animal we can supply oxygen with less stress than a mask. Tolerated better if a stream of oxygen is NOT pointed directly into the nares. Useful during physical exam etc.

BUSTER COLLAR & CLINGFILM;

Some animals will tolerate a Buster collar being placed and then sealed with clingfilm. The end of the oxygen supply can then be fed into the collar.

OXYGEN CAGES;

Oxygen cages are convenient, and an easy means of increasing inspired oxygen. However they are usually only suitable for small dogs and cats. But they do isolate the patient making it difficult to monitor.

Some oxygen cages will not increase the inspired concentration of oxygen above 50% which is insufficient. Once the door is opened this concentration drops to that of room air. Most commercial oxygen cages now have a fan and thermostat to prevent overheating.

Improvised cages can be made, or cat carriers placed in large clear polythene bags.

INCUBATORS;

Paediatric incubators can be purchased second-hand at NHS auctions etc. They are useful as oxygen concentrations of 80-90% are achievable, and they usually have a thermostat for temperature control.

NASAL 'PRONGS';

Human nasal prongs can be placed in the patient and secured behind the ears. These work best in large breed dogs that are immobile. The prongs usually need taping in place.

NASAL CATHETERS;

A nasal catheter can be placed in one or both nostrils and then connected to an oxygen source. Silicone feeding tubes are usually best tolerated. The catheter needs to be sutured or glued

TRANS-TRACHEAL OXYGEN;

In some cases, eg upper airway obstruction, it may be necessary to deliver oxygen below the level of the obstruction, this can be done with a trans-tracheal catheter, or a needle tracheostomy

Long term oxygen therapy requires that the oxygen is humidified to prevent irritation and dessication of mucous membranes.

UPPER AIRWAY

The upper airway consists of the nose, mouth, pharynx, larynx and trachea. Causes of respiratory distress at this level are related to partial or complete obstruction, this may be due to a number of factors:

- Brachycephalic obstructive airway syndrome (BOAS)
- Foreign body
- Space occupying lesions (abscess, neoplasia)
- Laryngeal paralysis
- Laryngeal or pharyngeal Oedema
- Trauma.

Patients with upper respiratory tract obstruction tend to present with audible inspiratory noise (stridor) with a long drawn out inspiratory phase. In cases with an acute onset, such as a foreign body, the patient is often very stressed and panicked.

Stabilisation relies on oxygen supplementation, which helps to calm the patient; gentle technique is required as they often resent handling of the face or neck. Cases of laryngeal paralysis will often respond to cooling, anti-inflammatories to combat oedema, and low doses of sedative as an anxiolytic.

In cases where there is no response to medical management, or where a foreign body is suspected then urgent control over the airway needs to be gained. Following pre-oxygenation, general anaesthesia is induced and the patient intubated with an endo tracheal tube. If intubation is not possible, the clinical team should be ready to immediately place a tracheostomy tube.

The procedure is generally carried out under general anaesthetic. Preparing the patient as far as possible beforehand reduces anaesthetic time; if possible clip the hair from the ventral neck and carry out an initial preparation of the skin before inducing GA.

- ***Clip the ventral region of the neck, position the animal in dorsal recumbancy.***
- ***A ventral midline incision is made over the trachea from the caudal larynx to the 7th tracheal ring.***
- ***Subcutaneous tissue is incised, and the sternohyoid muscles bluntly separated in the midline.***
- ***Place Gelpi retractors to expose the trachea***
- ***An incision is made circumferentially between the 4th and 6th tracheal rings (no more than 40% of the circumference)***
- ***Stay sutures are placed around the tracheal rings proximal and distal to the tracheotomy.***
- ***Introduce the tracheostomy tube***
- ***Partially close the proximal and distal parts of the skin wound, but leaving the tracheostomy fully accessible***
- ***Secure the tube by tying tape in a bow around the neck.***
- ***Apply antibiotic cream to the area surrounding the wound and apply a sterile dressing.***

The correct post-operative care of the patient and their tracheostomy tube is vitally important in order to ensure patient survival and prevent post-operative complications.

The following procedure should be followed for the suctioning of tracheostomy tubes

1. ***The patient should be pre-oxygenated for several minutes by applying a source of oxygen close to the opening of the tube***
2. ***Aseptic technique should be followed throughout the following procedure, which includes the use of sterile surgical gloves***
3. ***A suitable suction catheter is selected, which should be soft, pliable and sterile***
4. ***Sterile saline should be instilled, dependent upon the patient's size, usually between one and five millilitres, not more frequently than hourly***
5. ***The inner cannula of the tracheostomy tube, if used, is removed and cleaned***
6. ***The suction catheter is inserted to the level of the carina without a vacuum***
7. ***Intermittent light suction is applied while the catheter is withdrawn in a circular motion. Suction can be applied either via a suction unit or by using a sterile 20-50ml syringe***
8. ***The entire suction time should not exceed 15 seconds – if repeated suction is required then the patient should be allowed to relax and pre-oxygenate again before repeating the process***

9. ***Re-insert a sterile inner cannula while the removed cannula is cleaned and sterilised. If no inner cannula is being used, then it may be necessary to carefully clean the opening of the tube using cotton buds and saline, to remove any build up of secretions. Scrub solutions should be kept away from the tube and the incision as irritation may occur.***

10. ***The skin around the tube should be cleaned using a chlorhexidine solution***

11. ***The stay sutures should be examined along with the cleanliness of the ties. If the ties are contaminated, if they are contaminated then new ties should be placed before the removal of the contaminated ties***

12. ***Ensure that the patient's airway is patent and the tube is secure. Make sure the patient is comfortable. Sterile swabs may be placed beneath the phalanges of the tube to improve comfort; a sterile dressing should also be applied to cover the skin surrounding the tracheostomy site, e.g. primapore, Smith & Nephew.***

Clinical signs that the tube requires suction/cleaning:

- Dyspnoea
- Distress
- coughing
- harsh sounds from the tracheostomy tube
- discharge from the tube
- patient discomfort

LOWER AIRWAY DISEASE

Disease of the lower airway usually refers to problems with the small bronchi, and coughing is common (non-productive). Dyspnoea, with expiratory effort, and wheezes audible on auscultation are common findings. Common disorders include;

- Feline asthma
- Bronchial irritation
- Bronchitis
- Foreign body
- Smoke inhalation

These animals usually present due to crises or flare ups of existing problems, or when the disease becomes end stage.

LUNG PARENCHYMAL DISEASE

The alveoli are concerned with gas exchange. Interference with the ability to expel carbon dioxide and absorb oxygen in the alveoli will give rise to dyspnoea. Interference in the process may be caused by filling or collapse of alveoli, or an increase in the thickness of the diffusion barrier due to infiltration. Common disorders include:

- Pulmonary oedema
- Neurogenic pulmonary oedema (choking, strangulation, seizures)
- Pneumonia
- Pulmonary contusions
- Pulmonary haemorrhage
- Pulmonary inflammatory diseases

PLEURAL SPACE DISEASE

The pleural space is the potential space that exists between the pleura of the lungs, and the pleura of the chest wall. Accumulation of air, fluid, or soft tissue within the pleural space leads to reduced ventilation and so poor respiratory function. The most important physical finding with these patients is the presence of dull or diminished lung and heart sounds.
Common presenting signs in pleural space disease;

- Increased respiratory rate and effort
- Dyspnoea
- Cough
- Dull or muffled lung and heart sounds

Common causes of pleural space disease include;

- Pneumothorax (open, closed)
- Pleural effusion
  - Transudate
  - Modified transudate (eg CHF, lung lobe torsion)
  - Exudate (FIP)
  - Septic exudates (Pyothorax)
  - Haemorrhage (Trauma, coagulopathies)
- Diaphragmatic hernia.

Stabilisation relies of on oxygen supplementation, minimal stress, and vascular access followed by thoracocentesis. It is important that in all cases of suspected pleural space disease that thoracocentesis is performed ***before*** any attempts at radiography. Thoracocentesis is both diagnostic and therapeutic, and will rapidly improve the condition of patients with pleural effusion or pneumothorax.(see practical technique at end of chapter) Thoracic radiographs can then follow thoracocentesis, removal of effusion will also now make underlying pathology easier to visualise. The results of thoracocentesis couple with radiography should allow a diagnosis to be made.

- ***A wide area around the intended site is clipped and surgically prepared.***
- ***A butterfly needle with an incorporated extension set, or an intravenous catheter with a separate extension set is used, connected to a syringe via a 3-way tap. By using the extension set you allow the animal some movement without it dislodging the needle.***
- ***Point of insertion is the 7th or 8th intercostals space, cranial to rib.***
- ***If there is just air present, the needle is inserted in the dorsal third, if an effusion is present drainage is more efficient in the ventral third, and if there is a mixture of air and fluid, drain from the mid-point.***
- ***The needle is inserted at 45 degrees to the chest wall with the bevel facing the lung to try and minimise trauma.***
- ***Large volumes may be present.***
- ***If more than 1 pathological process going on, volume removed may be smaller than clinical signs suggested: eg pulmonary contusions and pneumothorax.***
- ***Drain both sides of the chest, and take radiographs afterwards to assess how efficient the drainage has been and whether a cause of the effusion is now obvious.***

If there is uncertainty as to whether the needle has entered the thoracic cabity, then a 'hanging' drop technique can be used- a drop of saline is placed in the hub of a catheter as it is advanced through the body wall, as the tip enters the chest the drop will be sucked in by negative pressure (or blown out if a tension pneumothorax is present!)

If repeated thoracocentesis is required due to fluid or air building up again in the pleural space, a chest drain is indicated.

CHEST DRAINS (THORACOSTOMY TUBE)

Thoracic drains are placed for the evacuation of air or an effusion (pyothorax, haemothorax etc) from the chest cavity to re-establish the negative pressure which is normally present and essential to ventilation.

If the patient is cooperative it should be possible to clip a large area of the required side of the chest and surgically prepare it with the patient in sternal recumbency and being re-oxygenated. You can then anaesthetise and intubate the patient, then rolling them into lateral recumbency prior to placing the drain.

- ***Sedate and administer local or general anaesthesia (the procedure may be carried out in the conscious patient in an emergency situation although adequate analgesia should always be provided)***
- ***Clip the side of the chest where the drain is to be placed and aseptically prepare.***
- ***Measure and mark the tube (from the point of introduction to the level of the second rib)***
- ***When placing a drain with a trochar, a skin incision is made in the dorsal third of the chest over the tenth intercostal space.***
- ***The trochar is introduced into the incision and tunnelled subcutaneously in a cranioventral direction, until the tip of the trochar lies over the 7th intercostal space.***

- ***At this stage the trochar handle is elevated so that it is perpendicular to the chest wall, and the heel of the hand used to firmly push the trochar through the intercostal muscles, whilst the other hand grips the trochar tightly 2 cm from the tip to prevent it penetrating too far.***

- ***Once into the chest, the trochar is withdrawn slightly so the tip doesn't cause damage, the handle is lowered again, and the trochar is advanced a few centimeters in a cranioventral direction.***

- ***The drain is then pushed off the trochar and advanced to a pre-measured level.***

- ***The tube must be clamped before the stylet is fully removed.***

- ***The drain is connected to a 3 way tap with bungs or a collection system.***

- ***The drain is then attached to the body wall using a Chinese fingertrap suture or a commercial fixation device.***

Drains can be placed without a trochar, using haemostats, or over a guide wire via a Seldinger technique.

It is important the positioning of the tube is confirmed. Radiographs should always be taken. Both lateral and dorso-ventral views are required. It is vital that the positioning of the tube is checked to make sure it will drain efficiently, it will be as comfortable as possible for the patient, and that obvious complications can be avoided.

'Shock'& Emergency IVFT

Definitions:

Dehydration:
Refers to a reduction in the total water content of the body. This may be a loss of water from any of the body compartments (intracellular, interstitial or intravascular), but usually infers intracellular or interstitial fluid losses. Dehydration in the absence of shock is usually not life threatening.

Hypovolemia:
Refers to a decrease in the intravascular fluid, and if significant, will result in hypoperfusion, shock, multiple organ failure, and death. It therefore requires prompt recognition and treatment. Hypovolemia may result from blood loss, severe vomiting, severe diarrhoea, excessive panting, and marked polyuria.

Hypoperfusion:
Refers to a state of decreased blood flow through the tissues, the consequence of which is decreased oxygen and nutrient delivery to the tissues, and decreased metabolic waste removal from the tissues. Although there are several causes of hypoperfusion, the most commonly encountered causes of hypoperfusion are the result of hypovolemia.

Shock:
'Shock' is usually taken to mean a significant compromise in oxygen delivery to tissues due to tissue hypoperfusion. This leads to cell damage, and if not corrected to organ dysfunction, organ failure, and death. Cell damage and cell death will lead to the release of inflammatory mediators which can lead to SIRS (Systemic Inflammatory Response Syndrome). Shock needs to recognised and treated as early as possible to optimise outcome.

Tissues may be hypoperfused due to:

Decreased circulating blood volume *(Hypovolaemic Shock)*

Decreased ability of the heart to pump blood *(Cardiogenic Shock)*

Decreased ability of vascular system to maintain vasomotor tone *(Distributive Shock)*

Obstruction of venous return (Obstructive Shock)

Although it is interesting to divide diseases into different forms of shock, if shock can be identified, and cardiogenic shock can be ruled out, then fluid therapy will be the cornerstone of treatment in the remaining forms of shock. It is possible to have several variables contributing to shock and several forms of shock may be present in the same animal at the same time.

Hypovolaemic shock is the most common form of shock seen in our patients, and occurs through haemorrhage (internal or external), or by severe fluid losses (GIT, kidneys, or 'third spacing' of fluid). Intravascular fluid compromises only a small proportion of total body water, losses from this compartment have a profound effect.

Recognising Shock

Signs of shock are associated with decreased blood flow to the tissues (mental depression), as well as the indicators that the animal has initiated a physiological response to maintain adequate oxygen delivery to the major organs.

To rapidly diagnose hypovolaemic shock we need to rely on the findings of our initial physical examination. Careful examination of perfusion parameters should allow recognition of shock, but also an accurate measure of the severity of the hypoperfusion present.

Detecting Changes in Perfusion Parameters:

Clinical Parameter	***Mild Hypovolaemia***	***Moderate Hypovolaemia***	***Severe hypovolaemia***
Heart Rate	***120-140***	***140-170***	***170-220***
Mucous Membrane	***Normal, or pinker***	***Pale pink***	***Pale/white/grey***
Capillary Refill	***Brisk (<1sec)***	***Normal (1-2 secs)***	***Slow or not detectable***
Pulse amplitude	***Increased***	***Decreased***	***Very decreased***
Pulse duration	***Mildly reduced***	***Reduced***	***Very reduced***
Peripheral pulse	***Easily Palpable***	***Faintly Palpable***	***Not Palpable***
Plasma lactate	***3-5mmol/l***	***5-8mmol/l***	***>8mmol/l***

Pulse quality, heart rate, capillary refill time and mucous membrane colour alter in a predictable way with worsening shock. By repeatedly assessing these perfusion parameters any improvement in the patient's condition during treatment can also be readily recognised as the perfusion improves.

Cats do not appear to follow the pattern of CV changes as outlined in the table above as reliably as dogs. Although in dogs close to death, the heart rate may slow just prior to cardiorespiratory arrest, in cats, bradycardia seems to occur at an earlier stage. A hypothermic bradycardic cat should raise particular concern. Cats with sepsis commonly present with heart rates in the 120-140bpm range.

Objective measures of assessing shock include blood pressure monitoring, blood lactate levels and urine output.

The presence of cardiogenic shock is important to recognize, as it is the only form of shock in which aggressive fluid therapy is contraindicated. Clinical signs are similar to hypovolemic shock but may also include coughing, pulmonary crackles and respiratory distress associated with left sided heart failure and jugular distension and ascites associated with right-sided heart failure.

Early septic shock often results in vasomotor dysfunction and cytokine-induced peripheral vasodilatation, high cardiac output (tachycardia), and hyperaemic mucous membranes (brick red) with a rapid capillary refill time (< 1 second). As septic shock progresses signs similar to hypovolemic shock predominate.

Hydration Deficits

Hydrations status is classically assessed by measuring physical parameters that will be affected by reduction of fluid in the interstitial and extracellular fluid.

Assessment includes:

- **Moistness of gums or cornea:**. other problems such as nausea can cause excessive salivation and make membranes appear moist even if dehydrated.
- **Skin turgor/skin tenting.** Dehydration causes the skin to remain tented for several seconds.
- **Retraction of the globe (sunken eyes).**

This assessment can only give a very rough approximation at best. Signs of dehydration can be very variable, skin turgor can vary as the animal gets older, and with body condition. The scheme may often underestimate the degree of dehydration, but this is not always a problem, as in contrast to hypoperfusion, these losses need to be replaced more gradually. Any animal that is showing any signs of hypoperfusion should be treated immediately and hydration status checked later once hypoperfusion is corrected.

Percentage Dehydration	Clinical Signs
<5%	No detectable clinical signs. Increased urine concentration.
5-6%	Subtle loss of skin elasticity. (Tenting)
6-8%	Marked loss skin elasticity. Slightly sunken eyes Dry mucous membranes
10-12%	Tented skin stays in place Sunken eyes, protruded 3rd eyelid Dry mucous membranes

Treating Hypovolaemic Shock.
Opinions differ on the ideal fluid therapy plan for the hypovolaemic patient, but the overriding principle MUST be to rapidly increase the intravascular volume of the patient. The effect of resuscitation can be monitored by regular assessment (every 10-15 minutes) of perfusion parameters, and the fluid plan re-evaluated and revised based on these findings.

Before starting aggressive fluid therapy remember there are some contra-indications; cardiac disease, respiratory disease, and brain injuries. Anuric renal failure requires careful fluid therapy, but fluid is required to confirm the diagnosis. Cats have a smaller fluid volume and tolerate volume overload poorly, so rates and volumes are reduced by a third to a half. Occasionally, in suspected cases of ongoing internal haemorrhage that is not controlled (eg splenic rupture, hepatic laceration) a more limited resuscitation may be indicated.

Choice of Fluid:

Hypotonic Crystalloids (eg 0.18% saline + 4% dextrose, 5% dextrose)

These should never be used to correct hypovolaemia or used at rapid rates. They are extremely inefficient volume expanders, and can lead to dangerous decreases in sodium levels, and increases in the volume of interstitial fluid- reducing cell oxygen delivery.

Maintenance Crystalloid (eg Plasmalyte M)

Higher potassium levels preclude their rapid use.

Replacement Isotonic Crystalloids (eg Hartmanns, 0.9% saline)

Probably the most useful of the crystalloids, and used in >90% of cases. The sodium concentration is similar to extracellular fluid (130-154mml/l) so the fluid rapidly equilibriates within the extracellular space (intravascular and interstitial), this has two implications:

1) Only 25% of the fluid will remain in the intravascular space after an hour, so large volumes are required.

2) This extravasation of fluid may become a problem where an increase in interstitial fluid would cause problems- eg brain injuries, or pulmonary contusions.

Hartmanns is useful fluid for nearly all conditions, the sodium and chloride concentrations are similar to extracellular fluid of dogs, and it contains only a small amount of potassium.

The use of isotonic crystalloids is thought to be optimal in the majority of hypovolaemic patients, but in some cases the use of other fluid therapies (hypertonic saline, synthetic colloids) maybe more suitable

Hypertonic Saline (eg 7.2% Saline)

Hypertonic saline has approximately eight times the tonicity of plasma. It brings about rapid but short lived volume expansion by creating an osmotic gradient that ‘draws’ water in from the interstitial space. Patients that are also dehydrated will have a less pronounced intravascular expansion as interstitial fluid will be reduced. Hypertonic saline will redistribute and needs to be followed up with isotonic fluids.

Small volumes administered have a large haemodynamic effect, and so is useful in large patients with severe shock where an isotonic crystalloid bolus would be time consuming.

The fluid of choice for resuscitating hypovolaemic animals with concurrent traumatic brain injury. Avoid where uncontrolled bleeding is suspected, also in heart disease.

Other potentially beneficial effect: stimulatory effect on the myocardium? Anti-inflammatory effect?

Colloids

Colloidal fluids contain large molecules, these molecules act to hold water within the intravascular space, and limit the movement of water across the capillary wall. Colloids have a more profound volume expanding effect compared to the same dose of isotonic crystalloid, the effect should also be much longer lasting.

They will have the desired effect until they are broken down or degraded by the body, or until they leak from the vessels. The rate at which their effect diminishes depends on the molecules involved. Artificial colloids contain a mixture of molecules of differing weights.

Initial volume expansion will depend on the number of molecules, not the molecule size. The duration of the effect will be dependant on molecule size- the larger molecules will persist longer and take longer to degrade.

Broadly speaking the only effective colloids readily available in the UK are hydroxyethylated starch products: Voluven, or Pentastarch. Gelatin based products (eg Haemaccell) are small molecules, smaller than albumin, they are readily excreted by the kidney,and are small enough to cross the capillary endothelium, and as such have no advantage over isotonic crystalloids.

Colloids are expensive, and their use can be associated with side effects such as a risk of dose-dependant coagulopathy. Routine use of colloids in resuscitation is therefore not advised. They be used in preference to crystalloids in hypovolaemic patients with severe pulmonary contusions.

Some risk of dose-dependant coagulopathy with some colloids.

ADMINISTRATION OF FLUID.

Route:

In emergency cases with hypovolaemic, fluid therapy needs to be administered intravenously. The site for intravenous access is usually a peripheral vein, critical patients may not be stable enough to withstand the sedation or general anaesthesia usually required to place a central line. In cases with severe cardiovascular collapse, or in small patients where IV access is difficult, the intraosseus route can also be used.

The aim of fluid resuscitation is to rapidly increase the intravascular volume. When using isotonic crystalloids, the large volumes required to achieve this expansion need to be administered over a short space of time. The choice of intravenous catheter can have a large effect on the maximum flow rate that can be achieved. Flow is maximised by the use of a wide bore, short catheter.

Volume of fluid:

Figures and guidelines exist giving set doses, of certain fluids; these must only be taken as guide lines, and each individual will need to have a plan tailored to suit them, dependant on the degree of hypovolaemia present. The emphasis must be on continuous monitoring to assess pefusion, and being prepared to alter the plan if there is no, or inadequate response.

The most important aspect to emphasise is that fluids should be delivered as a bolus over a short period of time and the effect assessed.

Isotonic crystalloids:

It has been recommended to administer 1/4 to 1/3 (20 to 30 ml/kg for isotonic crystalloids) of the calculated dose of "shock fluids" as a rapid intravenous bolus (over approx 5-10 minutes), while continually evaluating the animal. If the patient remains unstable, then 20-30 ml/kg boluses should be continued until hemodynamic stability is achieved. Ideally, resuscitation of the uncomplicated hypovolemic patients should be possible within 30 minutes of arrival at the hospital. If a patient fails to meet the desired parameters of stabilization following a full shock dose, then it must be re-evaluated to determine why end parameters have failed to be reached. It may be that the patient requires more than 90 ml/kg of fluids, and *the 1/3 to 1/4 boluses should be continued until the animal is cardiovascularly stable or the intravascular volume is expanded to the point at which further fluids are likely to result in fluid overload and signs of oedema.*

Synthetic colloids:
Such as Pentastarch, hetastarch and dextran 70 can be bolused at doses of 20ml/kg in dogs and 10 - 15 ml/kg in cats to treat hypovolemia (administer more slowly to cats to prevent nausea– i.e. 10-15 minutes). Although the shock dose is considered 20ml/kg in the dog, again, it is preferred to administer **5ml/kg boluses at a time**, with continual re-evaluation of the patient, until hemodynamic stability has been achieved. This will allow more precise administration of colloids to meet end points of resuscitation while reducing the risk of volume overload.

Hypertonic saline:
Unlike isotonic crystalloids or colloids, hypertonic saline, because of its mechanism of action and the shock dose is so small, is given as a single entire bolus. The dose is 4-6 ml/kg in both cats and dogs. It is possible to repeat the bolus, but this is not recommended if blood gasses cannot be followed (hypertonic saline can cause marked hyperchloremic metabolic acidosis and clinically relevant hypernatremia if given in excess).

Response To Treatment
There are many parameters that have been investigated to help answer this question, but a consensus has yet to be found. Some of these parameters are invasive and impractical in a general private practice setting (gastric pH monitoring, swan ganz catheterization, central venous pressure monitoring). However, there are several physical examination and diagnostic tests that can easily be followed that will help determine when a state of shock has been reversed. The objective is to treat the patient aggressively until the parameters in the table have been achieved.

Clinical parameter	*End goals to achieve*
Heart rate	< 120-140 beats per minute (dogs)
Peripheral pulses	Strong and regular
Mucous membrane colour	Pink
Capillary refill time	1-2 seconds
Blood pressure	> 90 mmHg systolic or 70 mmHg mean
Lactate level	< 2.5 mmol/L
Urine output	> 2ml/kg hour
Mental status	Improved, alert
pH	7.35 – 7.45
Hematocrit	Varies depending on inciting cause
Central venous pressure	0-5 cmH_2O