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Anaesthetic Case Challenges Mini Series

Session Two: Shock – How to deal with different types of shock in anaesthetised patients

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How to deal with shock in the anaesthetised patient

INTRODUCTION

Shock is a condition in which oxygen delivery to the tissues is insufficient to cover for oxygen consumption. Shock can be caused by inadequate delivery (more often) or excessive consumption (less frequently).

oxygen delivery ≠ oxygen consumption

Oxygen delivery (DO_2) to the tissues depends on cardiac output (CO) and arterial oxygen content (CaO_2)

$$DO_2 = CO \times CaO_2$$

Where cardiac output is the product of stroke volume (SV) and heart rate (HR)

$$CO = SV \times HR$$

And **arterial oxygen content** is the sum of the oxygen bound to haemoglobin and the one dissolved in blood

$$CaO_2 = ([Hb \times 1.34] \times SaO_2) + (0.003\ PaO_2)$$

Any factor affecting CaO_2, CO or DO_2 will cause shock. When oxygen delivery to the tissues is insufficient, cellular metabolism must switch from aerobic to anaerobic and if this condition is not promptly corrected cellular dysfunction and shock may occur due to the release of substances such as lactate, nitric oxide and cytokines.

Initially **compensatory mechanisms** kick in and the sympathetic nervous system is activated causing the release of adrenaline and noradrenaline with consequent tachycardia and vasoconstriction. Hormonal changes start as well; the renin-angiotensin-aldosterone system (RAAS) and the anti-diuretic hormone (ADH) cause vasoconstriction and renal retention of sodium and water. These compensatory mechanisms are able initially to restore DO_2 to organs but over time, when these fail, and when the shock is decompensated the clinical situation worsens and life-threatening DIC, SIRS or MODS may be seen. The clinical signs that can be seen in the different compensatory phases of shock are listed in the table below

	Compensatory shock	Early decompensated shock	Decompensated shock
Temperature	Normal to low normal	Slight to moderate hypothermia	Moderate to marked hypothermia
HR	Tachycardia (>180 bpm) Cats could be bradycardic	Tachycardia (>150 bpm) Cats could be bradycardic	Bradycardia (<140 bpm) Cats could be bradycardic
Mucous membrane	Normal to pale (hyperaemic in distributive shock)	Pale	Pale to gray/muddy
CRT	Normal to slightly prolonged. Difficult to assess in cats (<1 sec; rapid in distributive shock)	Prolonged (<2 sec) Difficult to assess in cats	Prolonged (>2 sec) Difficult to assess in cats
Respiratory rate	Tachypnoea (>50 breaths/min) More severe in cats	Tachypnoea (>50 breaths/min) More severe in cats	Tachypnoea, Bradypnoea or agonal More severe in cats
Blood pressure	Slight hypotension to normal	Mild/moderate hypotension	Marked hypotension refractory to fluid therapy
Mentation	Responsive	Obtunded	Very obtunded, stuporous, comatose

Shock is classified based on its aetiology:

- **Hypovolemic shock** is probably the type of shock most commonly seen in our small animal patients undergoing anaesthesia. This is due to low blood volume, often caused by haemorrhage but also from severe dehydration

- **Cardiogenic shock** occurs when the pumping effect of the heart is insufficient to provide adequate perfusion. This can be seen in patients with severe cardiac disease (DCM, HCM, severe valvular regurgitation)
- **Obstructive shock** occurs following an obstruction to blood flow. A typical example of a patient with obstructive shock who will need to undergo general anaesthesia is a patient with GDV. Patients with pericardial effusion or tension pneumothorax could also be presented for anaesthesia.
- **Distributive shock** generally occurs as a result of sepsis. Sepsis will cause peripheral vasodilation, increased vascular permeability and vascular pooling and the patient will present with red mucous membranes (rather than pale). Sepsis will also cause hypovolaemia and myocardial dysfunction and if DIC occurs, the formation of small clots can cause obstruction to blood flow; hence distributive shock is a mixed type of shock. Anaphylactic reactions can also cause distributive shock.
  A subtype of distributive shock is the **neurogenic shock**, where strong sympathetic stimulation (from CNS injury, airway obstruction) causes imponent vasoconstriction and decreased forward flow.
- **Hypoxaemic and anaemic shock** occur when, in the face of adequate perfusion, there is insufficient oxygen content to meet tissue needs. This can be cause by severe anaemia (anaemic) or severe pulmonary pathology (hypoxaemic)
- **Metabolic shock** is caused when, for some reason, the cells are unable to use that is adequately delivered to them. Causes for this type of shock are sepsis induced mitochondrial dysfunction, hypoglycaemia or cyanide toxicity – causing disruption of the electron transport chain and of the Krebs cycle. Causes include hypoglycaemia, cyanide toxicity or mitochondrial dysfunction (as occurs with sepsis).

STABILISATION

Whatever the cause of shock is, stabilisation of the patient is FUNDAMENTAL before anaesthesia. According to the CEPSAF study (Brodbelt, 2008), the mortality rate increased massively between ASA 1-2 to ASA 3-5 patient. Hence stabilisation is fundamental to decrease mortality risk. Unfortunately, often quick surgery is necessary for the stabilisation process itself, hence often the anaesthetist will need to anaesthetise patient in shock and sometimes unstable.

	Healthy ASA 1-2	Sick ASA 3-5	Overall
Dogs	1 in 1849	1 in 72	1 in 601
Cats	1 in 895	1 in 71	1 in 419

Treatment of shock must be carried on as an **emergency** and should be aimed to the underlining physiological cause. Also, treatment is dynamic, the patient needs to be constantly re-assessed. Heart rate, blood pressure, pulse rate, quality, colour of the mucous membranes, temperature of extremities, mentation, blood glucose, lactate, PCV and TS are all parameters which will need to be reassessed several times during stabilisation and beyond. In fact, sometimes it is possible to stabilise a patient but until the cause of shock is addressed this situation may not be permanent.

Hypovolemic shock/obstructive:

Hypovolemic or obstructive shock are primarily treated with fluids administered in large volumes.

Crystalloids are the fluids of choice for resuscitation of hypovolaemic patients. A bolus of 20-30 ml/kg should be administered and repeated if necessary, until the patient's cardiovascular parameters are normal.

Hartmann's is a balanced isotonic solution containing potassium in similar amounts that are found in the plasma. This solution also contains lactate which is metabolised to bicarbonate in the liver and counteracts acidosis (often present in septic patients)

After 1 hr only 1/3 of the volume administered remains into circulation. In septic patients, because of the decreased albumins and increased capillary leakage present, the use of crystalloids may lead to increased interstitial accumulation of fluids.

Saline 0.9% is another isotonic crystalloid (hence will cause the same problems mentioned for Hartmann's) but it is also hyperchloraemic compared to plasma and may cause/worsen acidosis which often is already present in patients with shock

Hypertonic saline has a very high osmolality, shifting water very rapidly and efficiently from the intravascular space with consequent intravascular expansion. This effect is transient and lasts approximately 30 minutes. Hypertonic saline also increases cardiac output and tissue perfusion hence is recommended in case of hypovolaemic shock. The administration of hypertonic saline must be followed by isotonic crystalloids's administration.

Hydroxyethyl starches (HES) are fluids containing macromolecules and will increase colloid osmotic pressure and intravascular volume for a more prolonged period of time compared to crystalloids. The most used nowadays is Voluven® (Fresenius Kabi). This particular fluid has shown to have major benefits in hypo-proteinaemic patients, patients with capillary leakage or who wouldn't benefit from an "interstitial" crystalloid overload. In the latest studies looking at goal directed fluid-therapy, voluven was better than crystalloids to increase intravascular volume and optimise CO.

Human serum albumins are quite expensive and can cause anaphylactic reactions in veterinary patients. These reactions can be immediate or delayed (up to three weeks). **Canine albumins** can be imported from the USA. Albumins would be administered mostly in distributive shock when capillary leakage is an issue.

Independently from the fluid chosen for initial resuscitation, what counts is that the status of the patient and the effects of the fluids are re-assessed constantly, and the patient does not become fluid overloaded.

Although a distinction in between blood pressure and blood flow to organs should be made, with the latter being more important to life than the first, it is still important to maintain blood pressure within limits which will allow the major organs to 'autoregulate' the blood flow they receive.

Blood pressure can be measure in several ways, but the invasive measurement remains the gold standard, being more reliable and continuous. If possible, an arterial catheter should be placed in every septic patient and the aim is to maintain a MAP of above 65-70 mmHg (or a Doppler blood pressure of about 90 mmHg in cats and 100 mmHg in dogs)

An observation for patients in obstructive shock is the location of the IV cannula. Fluids must be administered where they will be able to return to the heart without being 'obstructed'. Hence, for example, in a patient with GDV, fluids and drugs must be administered in a cephalic vein and not a saphenous vein.

Unlike the previous types of shock, because cardiogenic shock is a failure of the heart to effectively pump blood to tissues and not a lack of volume, giving copious amounts of fluids to these patients could aggravate the clinical situation. Positive inotropes (e.g., dobutamine – see later) should be administered instead.

Distributive shock

Distributive shock is the most difficult to address and a patient in septic shock will probably require more steps to be stabilised other than fluid resuscitation.

Sepsis is a life-threatening condition caused by an infection leading to dysregulated host response and, too often, death. The mortality rate in human affected by sepsis is of about 1 in 4. In small animals, the mortality rate is believed to be similar.

Treating a patient in sepsis is very challenging and often unrewarding. Because of the lack of consensus on what treatment approach is effective, international guidelines were published by a panel of experts. The *Surviving Sepsis Campaign* was first published in 2004 and after this reviewed every 4 years, with the latest review being published in 2016. It is important to understand that guidelines do not replace the clinician's decision-making capabilities.

- **SIRS:** Systemic Inflammatory Response Syndrome: the patient presents clinical signs of systemic inflammation in response to infectious or non-infectious insults (including trauma, pancreatitis, burns, neoplasia…).

Systemic Inflammatory Response Syndrome Criteria for Dogs and Cats		
Species	Dogs	Cats
Temperature °C	< 37.2, > 39.2	< 37.8, > 40
Heart rate (bpm)	> 140	< 140, > 225
Respiratory rate (bpm)	> 30	> 40
WBC X 10^3/µl	<6, >19	< 5, > 19

- **Sepsis:** life threatening organ dysfunction caused by a dysregulated host response **to infection**
- **Septic shock**: a subset of sepsis with circulatory and cellular/metabolic dysfunction associated with a higher risk of mortality

Common causes of SIRS are trauma, burns, pancreatitis, major surgery, immune mediated disease and neoplasia. Common causes of sepsis are septic abdomen, bite wounds, pyothorax, pyometra and parvoviral enteritis.
The pathophysiology of sepsis is complex. Normally an insult would cause an inflammatory response which is localized to the area. This inflammatory response will cause release of inflammatory mediators and acute phase proteins. Sometimes this localized response can become systemic. Physiologically the organism will produce a secondary anti-inflammatory response which, if excessive will lead to immunosuppression and immunoparalysis. Sepsis is a pathological disrupted equilibrium in between the physiological pro-inflammatory and anti-inflammatory responses of the organism.

Treatment
Fluid-therapy:
Initial resuscitation with fluids is a medical emergency and should be started immediately. The guidelines state that at least 30 ml/kg of IV crystalloids should be administered within the first 3 hr and additional fluids guided by frequent reassessment of the haemodynamic status.
The use of hydroxyethyl starches (HES) in septic patients is generally contraindicated because in 3 large clinical trials in human patients a clinical benefit of voluven could not be demonstrated and actually there was ample evidence of harm where the patients in the voluven group presented a higher incidence of need of renal replacement therapy and 28 days mortality rate. A drug alert was issued in June 2013 and in October 2014 the following recommendations were issued:

- Because of risk of kidney injury and mortality, HES solutions must no longer be used in patients with sepsis or burn injuries or critically ill patients
- HES solutions may continue to be used to treat hypovolaemia caused by acute blood loss. However, the doctor should monitor the patient's kidney function after HES administration

This said, in veterinary medicine some (including the author) would use Voluven in septic patients when TP are low and volume resuscitation is still necessary
An initial target MAP of 65 mmHg must be reached which may require the use of vasopressors.
Lactate levels should be normalised as soon as possible.
What fluid to use for this purpose will depend on the underlining cause and fluids have been addressed previously in these notes

Antimicrobial therapy:
The Surviving Sepsis Campaign suggests that:

- Administration of IV antimicrobials must be started as soon as possible after recognition and within 1 h of sepsis or septic shock
- If the cause of sepsis is unknown, empiric broad spectrum therapy - with one or more antimicrobials – should be instituted to cover all likely pathogens. This empiric therapy should then be narrowed once pathogen identification and sensitivities are established and/or adequate clinical improvement noted

- The guidelines recommend against sustained systemic antimicrobial prophylaxis in patients with SIRS of non-infectious origin (pancreatitis, burns.)
- The dosing of the antibiotics is to be optimised based on accepted pharmacokinetics and pharmacodynamics of those drugs in critically ill patients
- Empiric combination therapy (using at least two antibiotics of different antimicrobial classes) should be aimed at the most likely bacterial pathogen
- Treatment duration of 7-10 days should be adequate for most serious infections.

If possible, cultures should be made before starting antimicrobial therapy, but in the veterinary world it is unrealistic to expect these to be back within the hour. Hence, if the pathogen is unknown a four quadrants therapy should be started as soon as possible, taking into consideration

- The location of the infection
- The ability of the antibiotic to penetrate the site
- Suspected local bacterial flora
- Previous exposure to antimicrobials

Bactericidal antibiotics should be preferred to bacteriostatic ones.
A very good guide to veterinary antibiotic treatment based on the different body systems can be found at:
www.bsava.com/Portals/0/resources/documents/PROTECT_Poster_Nov_2014_2916.pdf

Analgesia
Should be titrated to effect, administered IV if possible and pure mu agonist should be preferred to other drugs in case the patient may require surgery. It is advisable to start with low doses and increase if required.
Drugs used for a septic patient could include one or more of the following:

- Methadone 0.1 mg/kg IV increments - up to 0.4 mg/kg IV q 4-6 h
- Fentanyl CRI 0.1-0.5 mcg/kg/min
- Lidocaine CRI (dogs) 25-50 mcg/kg/min
- Ketamine CRI 1-3 mcg/kg/min
- Paracetamol 10 mg/kg IV

NSAIDS should not be administered until the patient is no longer at risk of hypovolaemia or hypotension and until the kidney function of the patient has not been investigated.

Vasopressors/inotropes
If the patient is not responsive to fluids but remains haemodynamically unstable then pressors and/or inotropes should be considered
In veterinary medicine a consensus has not been reached regarding which drug is better.
In human patients, the guidelines recommend:

- Norepinephrine as a first drug of choice
- Addition of vasopressin or epinephrine to increase the MAP to target or to decrease the amount of norepinephrine
- Dopamine as an alternative in highly selected patients (low risk tachyarrhythmia or bradycardia)
- Against low dose dopamine for renal protection
- Reserve dobutamine for patients with persistent hypoperfusion despite adequate fluid loading and use of vasopressors
- Reduce or discontinue any drug in the face of worsening hypotension or arrhythmia
- All patients requiring a vasopressor should have an arterial catheter as soon as practical if resources are available

As mentioned earlier, flow is the most important factor. Hence a drug as phenylephrine causing vasoconstriction and decreased cardiac output will increase blood pressure but may decrease blood flow to organs such as the gut.
Norepinephrine:

- Increases MAP due to vasoconstriction
- Has little effect on HR
- Cause a slight increase in contractility
- Dose CRI 0.1 – 2 mcg/kg/min

Epinephrine:

- Increases HR and lactate
- Decreases splanchnic perfusion (up to 48 hr in humans)

- Can cause tachyarrhythmias
- Dose: CRI 0.05-1 mcg/kg/min

Dopamine:

- Failed to normalise BP in up to 40% people with hypotension
- Causes a higher incidence of tachyarrhythmia in people
- Had a worse outcome in shock human patients when compared to norepinephrine
- Cats and dogs are different from people and dopamine is widely used and effective
- Dose 5-20 mcg/kg/min
- Dose dependent effects
    - Low dose – dopamine receptors
    - Medium dose –β receptors
    - Higher dose – α receptor

Dobutamine:

- Mostly increases cardiac contractility and HR
- The slight decrease in SVR caused, results in increased CO
- Can be used in conjunction to drugs which are mostly effective on vessel tone
- Dose 5-20 mcg/kg/min. Stick to lower doses in cats as in this species dobutamine could cause seizures

Vasopressin:

- Acts on V1 receptors causing vasoconstriction
- Potentiates the effects of α-agonists aallowing to decrease doses used
- Is synthetized in hypothalamus, stored in the hypophysis and secreted in response to a decrease in BP, intravascular volume or increased osmolality
- Prolonged hypotension may lead to depletion of vasopressin
- Dose: 0.5-5 mU/kg/min

Phenylephrine

- Pure α-agonist
- Causes vasoconstriction and reflex bradycardia
- Increases BP but decreases CO and likely flow
- Dose: 0.5-5 mcg/kg/min

Ephedrine:

- Sympathomimetic amine which increases release norepinephrine
- Dose bolus 0.25-1 mg/kg or CRI 0.02-0.2 mg/kg/min
- Lasts 5-15 min
- Prolonged use can deplete norepinephrine stores. This phenomenon is known as tachyphylaxis

Blood glucose control:

The guidelines recommend

- A protocolised approach to blood glucose management in ICU patients with sepsis, commencing insulin dosing when two consecutive blood glucose levels are >180 mg/dL (10 mmol/L)
- To target to an upper blood glucose level ≤180 mg/dL (10 mmol/L)
- Blood glucose values should be monitored every 1-2 hrs until glucose values and insulin infusion rates are stable, then every 4 hrs thereafter in patients receiving insulin infusions
- Glucose levels obtained with point-of-care testing of capillary blood may not accurately estimate arterial blood or plasma glucose values – This difference is not reported in veterinary patients

Veterinary patients are often hypoglycaemic and if too low a CRI of Hartmann's or saline 0.9% containing glucose 2.5% or 5% should be started. A dextrose 5% solution is not recommendable as once the glucose is consumed by the patient it will leave pure water behind. A custom made 2.5 or 5% solution is recommended.

If hyperglycaemia were to occur in veterinary patients this should be tolerated up to a certain level (repeated measurements > 15-20 mmol/l in dogs and cats respectively) and if treatment is required, an CRI of regular insulin can be started (0.05 IU/kg/hr) but glucose and potassium should be monitored closely.

Source control
This is a fundamental step in treating patients with sepsis and should happen as soon as the patient is stable enough to undergo anaesthesia if this is necessary and ideally within 4 hours of admission

Nutrition
This is a step too often forgotten in the septic patient.
The guidelines recommend:

- Against the administration of early parenteral nutrition alone or parenteral nutrition in combination with enteral feedings in critically ill patients with sepsis or septic shock who can be fed enterally
- Early initiation of enteral feeding rather than a complete fast or only IV glucose in critically ill patients with sepsis or septic shock who can be fed enterally
- Suggest towards the use of prokinetic agents in critically ill patients with sepsis or septic shock and feeding intolerance
- Early trophic/hypocaloric or early full enteral feeding in critically ill patients with sepsis or septic shock; if trophic/hypocaloric feeding is the initial strategy, then feeds should be advanced according to patient tolerance

There are several ways of administering enteral food to veterinary patients:

	Need GA	Duration	Difficulty	Diet options
Naso-oesophageal	No	3-5 days	Easy	Few
Oesophagostomy	Yes	1-3 weeks	Easy	Many
Gastrostomy	Yes	1-3 months	Difficult	Many
Jejunal	Yes	1-2 weeks	Difficult	Few
Parenteral nutrition	No	1-3 weeks	Difficult	Many

The RER should be calculated as BW x $70^{0.75}$ and the diet should be formulated according to the patient and the situation.
Parenteral nutrition should be used only in patients in which enteral nutrition is not tolerated or comatose patients with gag reflex loss

Corticosteroids:
The recommendation of the Surviving Sepsis Guidelines is against intravenous hydrocortisone to treat septic shock patients if adequate fluid resuscitation and vasopressor therapy are able to restore hemodynamic stability. If this is not achievable, intravenous hydrocortisone is suggested.
Although this is difficult to prove, during prolonged periods of stress there may be an inadequate production of cortisol in relation to an increased demand. This condition is named CIRCI – critical illness-related insufficiency. Dogs and cats which remain unresponsive to fluid resuscitation and vasopressor/inotrope therapy may benefit of 2.3-3 mg/kg/day of hydrocortisone. If no improvement is seen in 24hr then the treatment should be stopped, otherwise it can be continued for 5 days and then tapered down over 2-3 days.

Nursing care
One should not under-estimate the benefits provided by good nursing care. A comfortable bed and turning the patient every 4 hours is essential in patients which are recumbent. In this case the person attending the patient should also consider a comfortable option for themselves (placing the patient in a cot or putting extra bedding for them if the patient is on the floor)
Mouth care and eye care, together with grooming are things one should not forget in recumbent critically ill patients

Placing a urinary catheter may help both in increasing the comfort of the patient (avoiding a full bladder) but also will allow urine output measurements. If a urinary catheter is not place the bladder should be expressed on a regular basis.
Water should be offered.
One or more IV cannulas should be placed in a clean fashion and the cannula site checked at least three times a day. These are common site of infection and bandaging may decrease external contamination.
Septic patients are often in pain and pain assessment performed with the aid of pain scoring sheets may be useful.
Even if non-ambulatory patients should be weighed at least twice a day and an increase in weight should be considered as possible fluid retention.
Critically ill patients often present with oedema, mostly if the limbs and of dependent areas. This may be due to low albumins often present in this category of patients or to fluid overload itself. If oedema is present, massage of the affected areas will help and one should check that bandaging or monitoring (mainly blood pressure cuffs) is not too tight

Other drugs:
Gastro protectants, antacids, anti-emetics and pro-kinetics can be added to the treatment of critically ill patients.
These include:

- Omeprazole 1 mg/kg IV daily
- Maropitant 1 mg/kg SQ/IV (dogs) SID
- Ondansetron 0.5-1 mg/kg SQ/IV
- Metoclopramide 0.5-1 mg/kg SQ, IV or as CRI in 24 hr

Because of the number of drugs these patients may be receiving in the 24hr, one must have organised kennel sheets and compatibility in between drugs should be checked

Last but not least, goals of care, prognosis and costs should be discussed upfront with the owners. The prognosis is often poor and the mortality rate high.

Cardiogenic and anaemic shock
Cardiogenic and anaemic shock will not require aggressive fluidtherapy.
Patient's with cardiac disease will require further investigations to identify the cause of disease and the state of the heart (dilation, contractility..). Adequate drugs should be administered as soon as possible
Patients with anaemic shock will probably require a blood transfusion before induction of anaesthesia. Increasing the amount of Hb will increase the arterial oxygen content and facilitate oxygen delivery to the tissues.
In less critical and more acute cases, a fluid bolus could be sufficient to restore volume (as this case would be a mixed anaemic/hypovolaemic shock) but care should be taken not to worsen the anaemia beyond a critical point.

Anaesthesia of the patient in shock (hypovolaemic, obstructive or distributive)

Even if the patient is stable before anaesthesia, most, if not all drugs will have effects of the cardiovascular homeostasis and will tip this precarious stability over the edge. All equipment must be prepared before induction of anaesthesia, drug doses calculated (including crash drugs, inotropes and pressors for CRIs) and any help you may need must be lined up. Decreasing anaesthetic time to a minimum is paramount in these patients.
Often a premedication with only an opioid (methadone 0.2-0.3 mg/kg IV or fentanyl 3-5 mcg/kg IV) is sufficient.
After pre-oxygenation (increasing the amount of oxygen dissolved in the blood) performed with a tight-fitting mask for about 5 min, induction of anaesthesia can be performed. Depending on how critical the patient is a combination of diazepam and Propofol, alfaxalone or etomidate in those patients with cardiogenic shock (only in these patients) can be used.

ASA classification	Induction drug	Comments
ASA II	Propofol or alfaxalone	Can be combined with diazepam
ASA III	Co-induction diazepam and Propofol or alfaxalone	
ASA IV	Co-induction diazepam/fentanyl	Could require a top up of alfaxalone
ASA V	Diazepam alone or fentanyl alone	

Patients with some type of GI obstruction may regurgitate at the time of induction. Suction should be available, tracheal intubation should be swift and the ETT cuffed whilst the head is still elevated.
Basic monitoring should consist of an ECG, capnography, blood pressure (ideally invasive), SpO_2, temperature. Additional useful monitoring could be CVP and PPV monitoring to assess the effects of fluid-therapy.
If maintenance occurs with isoflurane or sevoflurane, because both these drugs can cause hypotension, MAC sparing techniques should be used when possible

- Local blocks: TAP block, epidural, intrapleural/intraperitoneal
- Fentanyl CRI: 0.1-0.5 mcg/kg/min preceded by a bolus (1-5 mcg/kg) if this was not used for induction of anaesthesia. Higher doses of fentanyl will cause bradycardia (responsive to atropine) and hypoventilation. IPPV may be necessary.
- Lidocaine (not in cats) 25-80 mcg/kg/min preceded by a bolus (1-2 mg/kg). See more on lidocaine further down.
- Ketamine 2-10 mcg/kg/min preceded by a bolus (0.2-1 mg/kg)

Mean arterial blood pressure should be kept to about 60-70 mmHg. In these cases, this can be very challenging.
Fluid boluses (Hartmann's 10 ml/kg over 15 min) will be necessary, but care should be taken not to fluid overload the patient. If heart rate does not decrease, if MAP does not increase or if PPV<15 then the patient may just not be responsive to fluids. Pressors and inotropes may be necessary at the doses given above. Dopamine or a combination of dobutamine and noradrenaline are the preferred drugs in this situation.
For short term increase in blood pressure (while waiting for first incision), a bolus of ephedrine (0.05-0.1 mg/kg) can be used.
Patients in shock can develop cardiac arrhythmias at any time and for several reasons (hypovolaemia, hypoxaemia, surgical manipulation, toxin release, pain…). In patients with GDVs these can last several days post-op and a CRI of lidocaine started pre-operatively has been shown to decrease the incidence of arrhythmias, of AKI and of hospital stay in these patients.
Lidocaine has also been shown to decrease short term mortality in septic patients undergoing surgery.
Often septic patients will need abdominal lavages performed with warm fluids.These can cause sudden episodes of hypotension. The surgeon should remove the pressure cause by the fluids as quickly as possible and a bolus of fluids given. The temperature of the fluids should be checked.

Anaesthesia of the patient with cardiogenic shock

The choice of an anaesthesic protocol for patients with cardiac shock is dependent on what causes the cardiac insufficiency. Once again stabilisation is important, and wherever possible, any anaesthetic should be avoided in patients with decompensated cardiac disease. If for some reason this cannot be avoided then, any effort should be made to maintain CO.
Avoiding stress and consequent circulating catecholamines is fundamental in this type of patients as any increase in heart rate me push myocardial work beyond its limits.
Premedication should be restricted to drugs which will affect the cardiovascular system minimally but if the patient is in distress some form of sedation may be required as well. In some cases, for example when contractility is compromised, maintaining heart rate is paramount to maintain CO. In these cases, it would be advisable to avoid administering alpha2agonists. The use of these drugs is advantageous in patients with hypertrophic cardiomyopathy (HCM) with left ventricular outflow tract obstruction (LVOT), as, by slowing the heart rate and increasing preload, they allow both an increased diastolic filling and in some cases elimination of the LVOT
Acepromazine will decrease systemic vascular resistance and would be contraindicated in disease where an increased transvalvular pressure difference is not favourable. For the same reasons it would be indicated in patients with severe mitral valve disease it will promote forward flow through the aorta and decrease the regurgitation fraction.

Pre-oxygenation will increase the pulmonary oxygen reserves. Also, the level of oxygen dissolved in the blood and possibly facilitate its delivery

In severe cardiac shock, where maintaining cardiac homeostasis is paramount, induction of anaesthesia can be performed with etomidate. It is recommended to only use this drug in a restricted number of cases as it has shown to cause adrenal suppression with consequently diminished stress response and death in human ICU patients.

Other combinations with cardiovascular sparing effects are, in order from the most stable to least stable combination:

- Ketamine + diazepam – although ketamine should be avoided in patients with HCM
- Fentanyl + diazepam
- Alfaxalone + diazepam
- Propofol + diazepam
- Alfaxalone or Propofol alone

Drug	Dose (mg/kg)	HR	Contractility	SVR	CO	BP	Comments
Propofol	4-6 to effect	⇔	⇓	⇓	⇓	⇓	Vasodilation Better if combined with diazepam
Alfaxalone	2 to effect	⇑	⇔	⇔⇓	⇔⇑	⇔⇓	Better if combined with diazepam
Ketamine	5	⇑	⇑	⇑	⇑	⇑	Should be combined with diazepam
Etomidate	2	⇔	⇔	⇔	⇔	⇔	Very cardiovascular stable. Adrenal suppression Not licensed Should be combined with diazepam
Diazepam	0.2-0.4	⇔	⇔	⇔	⇔	⇔	Could use midazolam but not licensed for veterinary use. Can give dishinibition

Maintenance of anaesthesia in the patient with cardiac disease should involve as many MAC sparing techniques as possible – epidurals, local blocks, CRIs will allow to decrease the amount of inhalational anaesthetic and maintain blood pressure

Minimum monitoring required should include an ECG to identify arrhythmias, SpO2% to monitor saturation, blood pressure – ideally invasive and capnography.

Conclusions

Treating patients in shock and stabilising them as soon as possible is the first and probably the most important step in the anaesthetic management of this type of patients. Often, complete stabilisation is not possible and at this point time becomes the essence for survival.