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Practical Emergency Techniques for Advanced Practitioners Mini Series

Session One: Advanced cardiovascular stabilisation

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Classification of shock states

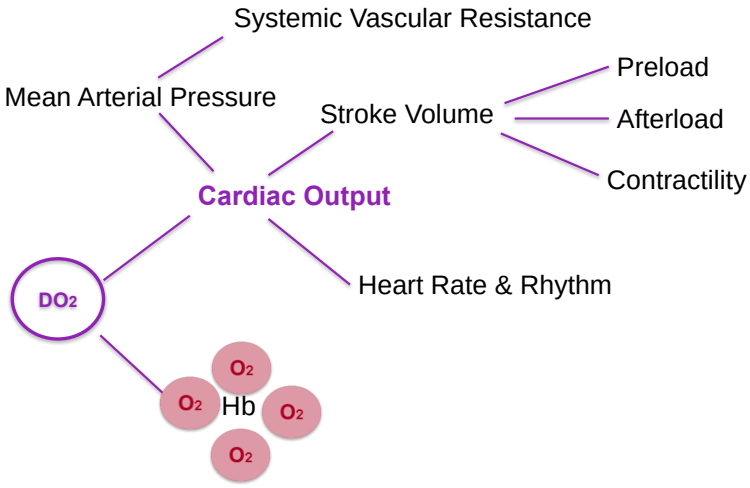
With any emergency patient presentation, initial triage and assessment of the 3 major body systems (cardiovascular, respiratory and neurologic) should be performed to determine whether the patient is unstable and therefore requires immediate treatment. The primary goal when assessing the cardiovascular system is to determine whether the patient is showing any sign of shock. Shock is defined as inadequate oxygen delivery (DO_2) to cells, resulting in inadequate cellular energy production. Left untreated, this leads to cellular dysfunction, progressing to organ dysfunction and ultimately death. Provided shock is detected and treated quickly, even severe shock may be successfully treated and fully reversible. Unfortunately, if left untreated for a critical period of time, it may become irreversible and lead to the death of the patient.

There are several classification systems for shock to categorise the causes. The simplest way of thinking about this is to divide them into: failure of the circulatory system to deliver blood to tissues (hypoperfusion), failure of the blood to carry enough oxygen (severe anaemia, hypoxaemia, methaemoglobinaemia) and failure of the tissues to utilise oxygen (sepsis or cyanide toxicity). Of these conditions, hypoperfusion is the most common in small animal veterinary patients. In cases of hypoperfusion, shock is a direct result of poor tissue perfusion from low or unevenly distributed blood flow causing a critical decrease in oxygen delivery in comparison to oxygen consumption. This can be divided further into hypovolaemic, cardiogenic, vasodilatory and obstructive shock states.

A more advanced classification of shock which is more comprehensive and considers the underlying cause in more detail, includes the following:

1. Hypovolaemic shock: a decrease in circulating blood volume
    - Haemorrhage, excess fluid losses (polyuria, GI losses, third spacing, burns), severe dehydration
2. Cardiogenic shock: a decrease in forward flow from the heart.
    - Congestive heart failure, cardiac arrhythmia, cardiac tamponade, drug overdose (B-blockers, anaesthetic agents, calcium channel blockers)
3. Distributive shock: a marked increase or decrease in systemic vascular resistance or maldistribution of blood
    - Sepsis, obstruction (arterial thrombus), anaphylaxis, catecholamine excess (pheochromocytoma, severe stress), gastric-dilatation volvulus
4. Metabolic shock: deranged cellular metabolic machinery
    - Hypoglycaemia, cyanide toxicity, mitochondrial dysfunction, cytopathic hypoxia of sepsis
5. Hypoxaemic shock: a decrease in oxygen content in arterial blood
    - Anaemia, severe pulmonary disease, carbon monoxide toxicity, methaemoglobinaemia

As shown by the following figure, there are numerous factors contributing to cardiac output and subsequent oxygen delivery (DO_2) to tissues, which if compromised, would result in a shock state.



Hypovolaemic shock

Hypovolaemic shock, caused by a decrease in circulating blood volume or preload, remains the most common cause of shock. Assessment of a patient's perfusion parameters (heart rate, pulse quality, mucous membrane colour, capillary refill time, extremity temperature and mentation) are vital in the clinical diagnosis of shock. They are also useful when determining the presence of compensatory mechanisms, as shown in the figure below providing details of hypovolaemic shock in dogs, and subsequent response to therapy as changes are typically reversed.

	Mild (compensatory)	**Moderate (early decompensatory)**	**Severe (late decompensatory)**
Heart rate	130-150	150-170	170-220
Mucous membrane colour	Normal to pinker than normal	Pale pink	Grey, white or muddy
Capillary refill time	Rapid (<1 sec)	Approximately normal (1-2 sec)	Slow (>2 sec) to absent
Pulse amplitude	Increased	Mild to moderately decreased	Severely decreased
Pulse duration	Mildly reduced	Moderately reduced	Severely reduced
Metatarsal pulse	Easily palpable	Just palpable	Absent
Mentation	Usually normal	Depressed	Severely depressed
Extremities	Usually normal	Normal or cool	Cold

Patients presenting with a history of trauma are thought to have haemorrhage as a cause of hypovolaemic shock until proven otherwise. Common sources of bleeding in the body include the pleural, pericardial, peritoneal and retroperitoneal spaces, within the parenchyma or beneath the capsule of internal organs, external wounds, the gastrointestinal tract, and into fracture sites.

Vascular access is essential in the management of cases with hypovolaemic shock but may be challenging in patients with collapsed blood vessels with severe hypoperfusion. If possible, short, large bore catheters are preferable to allow a rapid rate of fluid administration. If intravenous access is difficult to delayed due to the patient's condition, a cut-down approach to the blood vessel or intra-osseous access may provide rapid patient access for the administration of fluids. Setting a short and defined time limit (< 5 minutes) on any attempt at percutaneous catheterisation, such an alternative approach should be considered.

Vascular cut down
In dogs the lateral saphenous vein is preferred owing to its size and location under relatively thin skin. The hair should be clipped from the site and the skin prepared as much as time allow. A scalpel blade is used to incise the skin immediately adjacent to the blood vessel, running in a line immediately adjacent to the vessel. Once the incision is made, the skin should be retracted to expose the vein. Haemostats are then used to bluntly dissect around the vessel, applying pressure to the tips directly onto the vein to strip away perivascular fascia. The vein should be freed from its fascia in all directions so the haemostats can be passed underneath the vein and the instrument positioned such that the vein is stretched out over the two handle shafts. The vein is now exteriorised, free from fascia, and

occluded at the top and bottom by the pressure of the handles. It is also immobilised to facilitate catheter placement using a smaller than normal gauge over-the-needle catheter. The stylet is removed, a connector attached and the haemostats removed. The wound margins should be apposed over the catheter, the skin wound temporarily closed, and the catheter secured. In cats, the medial saphenous vein is often used in preference, using the same technique as described above.

Catheters inserted using this technique should always be considered to be contaminated and should be removed as soon as the patient has been stabilised and more appropriate longer-term vascular access secured. Until this time, the would should be cleaned as for any laceration and any remaining hair removed around the site. The wound should be flushed liberally while the catheter remains in place. Next, the site should be compressed using sterile swabs, the catheter removed and direct pressure applied to provide haemostasis. Once any bleeding has stopped, the wound may be dressed and left to heal by secondary intention. If the size of the incision is considered too large, skin sutures may be placed but it is essential that the distal third of the wound be left open to drain.

Intra-osseous catheterisation
Intra-osseous catheterisation involves placing a needle into a bone. The technique is simple to perform, does not require any specialised equipment, and can be very useful. Intra-osseous catheters may be placed in the trochanteric fossa of the femur, the wing of the ileum, the proximal humerus, and the tibial tuberosity. Of these sites, the trochanteric fossa of the femur is the preferred site in dogs and cats. In small or neonatal animals, a standard 22-gauge hypodermic needle is used. Bone marrow needles can be used in larger patients whose bones have already ossified. Intraosseous drills can also be used to facilitate insertion of an intraossous catheter in an ossified bone (see below). In smaller patients whose bones have not yet ossified, the shaft of the hypodermic needle may become clogged with cortical bone debris during placement. This may be avoided by using a spinal needle containing an inner stylet than can be removed following correct placement, or using a piece of surgical wire in the shaft of the needle to prevent clogging. In the event the needle does become clogged, it may be removed and replaced with an identical one through the hole that was created.

The site of insertion should be clipped and cleaned. Local anaesthetic may be injected down to the level of the periosteum to decrease any discomfort associated with catheter placement. The stifle should be adducted so it is pushed toward the central midline and the trochanteric fossa is rotated laterally. This positioning helps to decrease the risk of trauma to the sciatic nerve. The needle tip is pushed through the skin, and using a simultaneous pushing and twisting motion, it is pushed into the groove in the intertrochanteric fossa, through the periosteum, and into the shaft of the femur. There will be a loss of resistance felt as the needle enters the shaft of the femur. Once the needle has been placed correctly, you should be able to push the hub of the needle back and forth and move the leg. Aspiration of bone marrow also confirms correct placement. Ideally radiographs should be taken to confirm correct placement. A T-connector should be attached to the hub of the needle and the catheter flushed with saline. Non-heparinised saline should be used on very small patients to prevent the development of a coagulopathy. There should be very little resistance felt when flushing the catheter. If the fluid does not flow freely, the needle should be rotated 90 to 180 degrees to ensure the bevel has not become lodged against the wall of the bony cortex, causing an occlusion. The catheter should be secured using tape or suture. The catheter may then be used to deliver fluid therapy (crystalloid, colloids and blood products), drugs and parenteral nutrition. Also, whilst very fast rates may be reached, rapid infusion rates can cause discomfort in some patients.

Relative contraindications include fracture of the proposed catheter site, bacterial infection or sepsis, or the presence of a skin wound or infection over the proposed site of catheterisation.

EZ-IO catheter placement
The EZ-IO system (www.vidacare.com) provides a new technique for performing intraosseous catheterisation in patient with ossified bones. A hand-held, battery-powered drill is used to rapidly place (< 10 seconds) a purpose-made intraosseous catheter. Any of the anatomic sites listed above may be used for intraosseous catheterisation using the technique, although the greater tubercle of the humerus, or tibial tuberosity are often preferred.

The patient should be placed in lateral recumbency for catheter placement. If the greater tubercle of the humerus is being used for placement, the landmarks include the scapular spine and the acromium. The site should be clipped and skin prepared prior to infiltration with local anaesthesia as

described above. A stab incision may be made to facilitate placement of the catheter. A catheter is selected based on the size of the patient and is loaded onto the drill. The tip of the needle is pushed through the skin and placed directly into the periosteum of the greater tubercle. Forward pressure is then applied, taking care that the needle does not slip off the cortical bone. The power button is depressed on the drill and the catheter placed directly into the bone. The stylet is removed and connector attached. Correct placement of the catheter should be confirmed, as described above. If there is any pain associated with infusion of high fluid rates, an infusion of lidocaine may be used (1-2mg/kg, 2% solution) to improve patient comfort. Drug doses for intraosseous administration are the same as for intravenous doses. The catheter may be used until the patient is stable and alternate vascular access has been secured.

Treatment of hypovolaemic shock

Treatment of hypovolaemic shock is based on early recognition of the condition and rapid restoration of the cardiovascular system so that DO_2 to the tissues is normalised as soon as possible. The mainstay of therapy for all forms of shock except cardiogenic shock is based on rapid administration of large volumes of intravenous fluids to restore an effective circulating volume and tissue perfusion. Regardless of the type of fluid deficit, an isotonic crystalloid solution is the fluid of choice in most cases. It is important to note the fact that all isotonic crystalloid fluids will redistribute from the vascular space into the extracellular fluid compartment over time owing to the small size of the water molecules and electrolytes. It is known that at 30 minutes post infusion, only 25% of the isotonic crystalloid administered will remain in the intravascular space. This can be a reason for a possible worsening in the clinical condition of a patient a period of time following initial stabilisation and require repeated fluid resuscitation in the face of ongoing losses.

Hypertonic saline solutions may also be considered, especially in patients where large volumes of isotonic crystalloids would be challenging to deliver, or in patients where concurrent intracranial hypertension is suspected. Hypertonic saline solutions (HTS) are given at a dose of 5 ml/kg (dog) or 3 ml/kg (cat) over 15-20 minutes and volume resuscitate the intravascular space by redistribution of fluid from the other body fluid compartments. Contraindications to the use of hypertonic solutions include the presence of dehydration, sodium disturbances and concurrent cardiac disease.

The use of HTS for resuscitation has several theoretical benefits aside from the small dosing volume. Its use is less likely than large-volume isotonic crystalloids to contribute to volume overload, interstitial oedema of the tissues, or disruption of the normal endothelial glycocalyx. It also is known to have favourable rheological effects which can improve blood flow in shock, thereby increasing DO_2 to tissues. In addition, it is known to enhance cardiac performance in excess of the effect anticipated by the increase in preload, likely by increasing cardiac contractility. There are also immunomodulatory effects of hypertonic saline that may prevent reperfusion injury during fluid resuscitation and resultant secondary inflammatory injury and which can also be helpful in treating the inflammatory state in the brain that is known to occur following traumatic brain injury.

In patients with peritoneal bleeding and ongoing evidence of hypoperfusion, placement of an abdominal wrap may help to slow or even arrest bleeding and may even improve blood pressure. This technique may be considered in cases with abdominal bleeding of any cause, whether that be to stop traumatic bleeding or help to further stabilise a patient with bleeding of a neoplastic origin prior to surgery. Abdominal wraps placed encorporating the pelvic limbs and pelvis in addition to the abdomen may help in patient immobilisation and slowing the haemorrhage associated with pelvic or femoral fractures. An initial layer of cast padding or rolled cotton is used to distribute pressure evenly, before a second adhesive bandage layer is placed. Although the incidence of adverse effects is relatively low, abdominal wraps have been associated with complications. For this reason, abdominal counterpressure should only be used in veterinary patients suffering from abdominal, pelvic or femoral haemorrhage that do not initially stabilise with adequate fluid therapy or are in decompensatory shock at the time of presentation. During placement of the wrap, the heart rate, blood pressure, respiratory rate and effort, mucous membrane colour, and other indices of oxygenation and ventilation (such as pulse oximetry or arterial blood gas analysis) should be monitored. While exerting the desired tamponade effect to control bleeding, the secondary increase in blood pressure provided by the wrap may actually exacerbate haemorrhage which needs immediate recognition and treatment. Other possible complications include a decrease in perfusion to essential organs as a result of pressure increases applied to the whole abdomen, which can result in compromise of the renal, cardiovascular,

visceral, hormonal, pulmonary, and neurologic systems. Decreased glomerular filtration rate, urine production, venous return, pulmonary compliance, and splanchnic perfusion, as well as increased risk of bacterial translocation have been documented in both human patients and experimental scenarios of intraabdominal hypertension. Abdominal wraps have also been shown to decrease tidal volume, cause tachypnoea, and compromise ventilation. For this reason, the placement of an abdominal wrap is contraindicated in cases with respiratory compromise, such as pulmonary contusions, pleural space disease, or diaphragmatic hernia. The placement of a wrap may unmask the presence of unknown respiratory complications in a trauma patient.

Any abdominal wrap should only be left in place as long as necessary to achieve cardiovascular stability (< 12 hours in all cases). If cardiovascular stability has not been achieved within this time period with concurrent fluid therapy and blood products as needed, surgical exploration to control the bleeding should be considered. Assuming patient stability has been achieved, the wrap should be removed gradually by a few cm very 15-20 minutes (sooner if there is any respiratory compromise) and the patient monitored closely. If there is a drop of blood pressure of more than 5 mm Hg, an increase in heart rate by more than 5%, or development of pale mucous membranes, removal of the wrap should be discontinued until haemodynamic stability is again achieved. Rapid removal of the wrap can result in a severe hypotensive episode and should be avoided.

Tranexamic acid is an additional consideration in patients with ongoing bleeding despite standard therapy. It is an antifibrinolytic drug that slows the rate of clot breakdown and may help to stop bleeding of any cause (not just in patients with confirmed or suspected hyperfibrinolysis such as Sighthounds). It is given as a dose of 10-15 mg/kg IV q8. Higher doses have not been shown to be more effective but are likely to induce emesis.

Cardiogenic shock

Cardiogenic shock occurs for the reasons stated above, and causes a decrease in oxygen delivery by affecting several important components of the tree of life such as afterload, arrhythmias, systemic vascular resistance depending on the exact cause. Clinical features of cardiogenic shock include hypoperfusion, extremes of heart rate, an irregular heart rhythm, a new or increased intensity of heart murmur, the presence of gallop rhythm in cats, and concurrent respiratory signs or evidence of cardiac failure. Pericardial effusion may be suspected based on signs of right sided pressure elevation, fluid visualisation on TFAST, pulsus paradoxus and electrical alternans on ECG.

Distributive shock

Of the causes of distributive shock listed above, sepsis is one of the most common. Clinical features that should raise immediate concern for the presence of a severe inflammatory or septic condition include injected mucous memrbanes with a rapid capillary refill time (< 1 second), and hyperdynamic pulses during the early stages of shock before evidence of decompensation becomes apparent. Many of the causes may also present with concurrent hypovolaemic shock owing to concurrent fluid losses from the intravascular space. Treatment with intravenous fluids may well unmask the clinical features of distributive shock over time as the patient fails to improve as suspected.

Metabolic shock

Metabolic shock differs from the other forms of shock being discussed as it results in decreased cellular energy production because of deranged cellular metabolic machinery rather than a lack of oxygen directly. Treatment is based on elimination of the underlying cause where possible.

Hypoxaemic shock

Hypoxaemic shock occurs due to a marked decrease in the oxygen content in arterial blood, whether that be due to a lack of carrying capacity (anaemia) or lack of oxygen dissolved in the blood (severe respiratory disease). Treatment with oxygen therapy and ensuring there is no issue with circulating volume etc. is usually effective.

Advanced patient assessment techniques

At the present time, there are many techniques available in veterinary practice, including patient side ultrasound, shock index, blood pressure measurement, central venous pressure measurement and blood gas sampling and interpretation.

Patient side ultrasound techniques
AFAST (abdominal focused assessment with sonography for trauma or triage or tracking) involves rapid patient-side ultrasound of the abdomen to specifically look for the presence or absence of free peritoneal fluid. It is not a substitute for a thorough physical examination or a full ultrasound exam, but can instead provide the emergency clinician with valuable information on their patient. Four standardised sites are visualised as part of the AFAST scan and each assessed for the presence or absence of fluid.

1. Diaphragmatic hepatic view
2. Cysto-colic view
3. Hepato-renal
4. Spleno-renal

The exam is best performed in right lateral recumbency but can be performed instead on a patient in sternal or standing depending on what is possible and tolerated in the emergency patient. Once all 4 sites have been assessed for free fluid, an abdominal fluid score (AFS) is recorded, ranging from 0 (no sites positive for fluid) to 4 (free fluid seen in all sites). This can be useful for patient monitoring and case discussion at shift handover. It can also be useful for the detection of a haemoabdomen (in cases where an initial AFS is zero but increases over time), ongoing haemorrhage (increasing fluid score), or resolution of a haemoabdomen (fluid score decreases over time). If fluid is visualised, it is common practice to take a sample via abdominocentesis for further investigation.

Patient side ultrasound can also be used to provide valuable information on the heart structure and function in the emergency patient. More specifically, it can look for systolic dysfunction, cardiac chamber enlargement etc. It is not a replacement for a full echo but can help build up a picture of the condition of the cardiovascular system rapidly and guide emergency therapy.

A new measurement is the CVC-CI (caudal vena cava compressibility index). In this, the caudal vena caval diameter and its respiratory variations can be used to estimate patient volume status. A subcostal view is used to generate these measurements. The transducer is positioned just caudal to the xiphoid, typically with the patient in right lateral recumbency similar to the diaphragmatico-hepatic (DH) view. The marker is pointed toward the elbow and the probe angulated toward the head almost parallel with the body wall. Respiratory fluctuations in the caudal vena cava are only seen when right atrial pressure (RAP) is not elevated. If RAP is greater than 15 mmHg, consistent with hypervolemia or right heart failure, no fluctuations are noted. A variation greater than 50% in caudal vena caval diameter with respiration can predict a positive hemodynamic response to volume expansion with intravenous fluids.

The gallbladder halo sign, taken at the DH view of the AFAST scan, is reported to be helpful as a marker for anaphylaxis in dogs since their shock organ (where most mast cells reside) is their liver and gastro-intestinal tract (unlike humans and cats that have the lungs as their shock organ). In the acutely collapsed dog however, the other major rule out for ultrasound finding of a gallbladder halo sign, is acute cardiac tamponade seen with pericardial effusion since any cause of acute hepatic venous congestion may result in swelling of the gallbladder wall and thus the sonographic creation of the gallbladder halo sign.

Patient side ultrasound may also be used to estimate bladder volume and hence urine output without the need for urinary catheterisation. This can provide valuable information in the emergency patient to guide fluid therapy and facilitate assessment of renal function. This could inform the clinician on perfusion following trauma, or the severity of any kidney injury and options for management. An estimate for bladder volume is obtained from measurements taken from the AFAST cysto-colic view using the formula: L x W x 0.3 x Pi. The width measurement is taken by turning the ultrasound probe counter-clockwise into the transverse orientation maximising the oval or circle for measurement. This is reported to obtain values within 10% of that collected from bladder emptying in initial studies, although it is possible the formula may underestimate very large volumes of urine. Extra care must be

taken when performing the cysto-colic view in this instance as probe pressure may change urinary bladder shape and hence the measurements taken.

Pericardial effusion is a major cause of cardiovascular instability and cardiogenic shock, and can be readily diagnosed with confidence using patient side ultrasound tools. It is very important that multiple views of the pericardial site and DH site are used to ensure the fluid present is contained within the pericardial sac, and not within the pleural space or an enlarged cardiac chamber. Altering the depth of the ultrasound image so the pericardium is seen in the far field to help make this clear. In addition, moving the probe away from the heart chambers and towards the apex of the heart can help confirm the presence of pericardial effusion, generating the so called 'bull's eye' view and the 'racetrack sign' at the DH view, created by the rounding of pericardial effusion between the pericardial sac and the apex of the heart. Once pericardial effusion has been diagnosed, the patient should be evaluated for the presence of cardiac tamponade, as evidenced by collapse of the cardiac chambers during diastole. The presence of fluid in the pericardial sac surrounding the cardiac chambers makes this easy to visualise.

Shock index

The shock index is a calculation that looks at the relationship between heart rate and blood pressure to identify early shock or occult hypoperfusion, even in the presence of outwardly normal cardiovascular parameters. It is calculated by heart rate/systolic blood pressure. In people, a shock index of > 0.9 requires immediate attention. A recent study looking at shock index evaluation in dogs presenting to the emergency room showed the shock index to be significantly higher in dogs with clinical signs of shock than those without. A cut off of 1 for the shock index have a sensitivity and specificity of around 90%. The shock index is not a replacement for assessment of the perfusion parameters but may help with patient monitoring and detection of occult hypoperfusion.

Direct arterial blood pressure (DABP) measurement

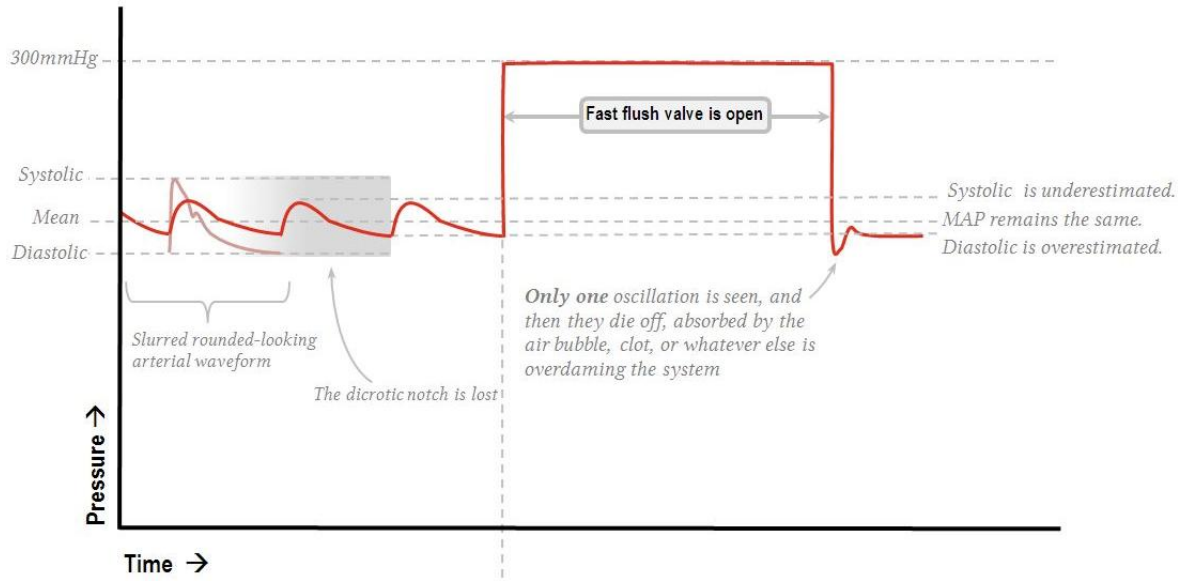
Hypotension is commonly seen in shock states and can be defined as a systolic blood pressure < 90 mmHg or a mean blood pressure < 65 mmHg. Measurement of blood pressure can be a useful monitoring tool in patients with cardiovascular instability. Direct arterial blood pressure measurement is considered the gold standard in both human and veterinary medicine. It involves placement of an arterial catheter (usually into a peripheral artery such as the dorsal pedal artery) which allows continuous monitoring and arterial blood sample collection as required. The arterial catheter is secured in place to prevent accidental dislodgement and connected to a flushed T-connector. This is then attached, via saline filled non-compressible tubing, to a disposable pressure transducer which converts the blood pressure to a measurement. An inflated pressure bag allows for the system to be continuously flushed, the pressure bag being required to maintain the system under constant pressure that must be higher than the systolic arterial pressure. The weight of any air or atmospheric pressure must be eliminated from the measurements taken. This is achieved by opening the transducer to air and adjusting the display system to read zero. By doing so, all pressure contributions from the atmosphere are negated, and only pressures that exist in the vessel will be measured. The measurements are typically displayed on a multi-parameter monitor, where a series of waveforms and the patient's real time systolic and diastolic pressures are displayed, in addition to the mean arterial pressure.

Once an arterial catheter has been placed, the utmost care must be used to maintain it on a long-term basis. It should be treated like any other catheter, including the use of aseptic technique and daily bandage changes. The heparinised saline bag should be changed every 24 hours, and the high-pressure tubing associated with the transducer should be replaced every 72 hours. Any disconnection will result in rapid patient haemorrhage and so the system requires constant vigilance and is reserved for recumbent unstable patients.

The waveform produced can also provide valuable information when dealing with the patient. Peak ejection of blood from the heart occurs during the highest point on the waveform and is associated with systole. The downstroke of the waveform is associated with a drop in the blood pressure. Midway through the downstroke, a notch, called the dicrotic notch, may be visible, indicating closure of the aortic valve. The dicrotic notch also represents the beginning of diastole. The remainder of the waveform's downstroke represents blood flow into the arterial tree, with the lowest point representing

diastole. Other useful information can also be provided, especially regarding the effects of arterial perfusion on the major organ systems. For example, a state of poor perfusion exists when cardiac arrhythmias (e.g., intermittent ventricular premature contractions) are associated with a dampened waveform appearance in conjunction with an abnormal MAP.

A thorough knowledge of the potential problems associated with arterial catheters and their waveform indications can simplify the troubleshooting process and ensure continuous DABP monitoring. One common problem involves waveform dampening or loss. A good arterial line trace has a distinct dicrotic notch, and after the fast flush test there are two oscillations only.



The figure above shows an overdampened waveform, likely as a result of air bubbles in the system although other causes may include blood clots, or kinks in the catheter or tubing (less likely if non-compressible tubing is used). In all cases of waveform dampening or loss it is important to ensure that the line is not clamped off, and then the line and catheter should be flushed. If the catheter has migrated against the vessel wall, changing the patient's position may resolve the problem. Inaccurate readings (i.e., lower systolic and higher diastolic values) may occur when the waveform is dampened. Furthermore, inaccurately low readings may occur in patients with severe peripheral vasoconstriction (e.g., due to severe hypovolemia or high-dose pressor agents).

In the event of a sudden change in pressure, it is essential to ensure that the transducer has not moved and is still at the level of the heart and that no one is leaning on the patient's line or a major blood vessel. More important, a sudden change in pressure can indicate that cardiac arrest has occurred or is imminent, so the patient's pulse and end-tidal CO_2 production should be assessed immediately to make sure this is not the case. When the accuracy of DABP measurements is questioned, it is not necessary to spend a great deal of time troubleshooting the system. Instead, assess the patient's cardiovascular status (e.g., respiration, pulse quality, mucous membrane colour, etc.) and combine those findings with the indirect blood pressure readings to help ensure timely intervention during a potential cardiovascular crisis.

Approach to the patient with persistent cardiovascular instability

Despite rapid assessment and appropriate initial stabilisation measures, there are a subset of veterinary patients that may have persistent cardiovascular instability, recognised as persistent hypoperfusion or hypotension. In order to identify therapeutic targets, it can be useful to review 'branches' of the tree of life (the factors that contribute to cardiac output and DO_2). These are discussed in more detail below.

1. Is there effective circulating volume

It is likely that the patient will have received some initial fluid therapy but the big question is whether this is effective. Considerations include whether any fluid deficit has been accounted for and whether the ongoing losses are greater than initially thought. Fluid losses into the GI tract and urinary tract may be much higher than initially thought in some cases, and ongoing internal bleeding may be another cause of increased fluid requirements. Repeated AFAST may show in increased fluid volume in the latter instance. Making attempts to track 'ins and outs' to measure fluid output from urinary catheters, faecal Foley catheters and surgical drains can be helpful. It is important to periodically check they are draining correctly where possible, using patient ultrasound, to make sure true outputs are adequately represented. An increasing packed cell volume, total solids and/or sodium can all suggest increased fluid losses from the body. In some cases where the blood protein is low, the extravasation of isotonic crystalloids may be more exaggerated than normal, meaning cardiovascular stability cannot be achieved or a very short lived. It may be necessary to consider the use of a colloid in such cases.

2. Heart rate and rhythm

Is there are extreme of heart rate (in either direction) or an abnormal rhythm that is affecting perfusion, treatment should be targeted at this. ECG monitoring of unstable patients can be useful to monitor progress but also detect such problems.

3. Low systemic vascular resistance (SVR)

Some causes of low SVR have already been mentioned when discussing shock earlier in the webinar. This is commonly present in patients with sepsis and typically the main reason behind ongoing instability despite initial fluid therapy. Physical exam findings including injected mucous membranes, rapid capillary refill time and in some cases hyperdynamic pulses can be suggestive. It should also be suspected in any patient with sepsis or severe inflammatory disease without seeing these signs. Treatment with vasopressors (see below) to achieve patient stability, and removal of the source of inflammation where possible (source control) are vital measures.

4. Impaired cardiac contractility

Failure of systolic function and subsequent pumping of blood from the heart may be an additional contributing factor to cardiovascular instability. On patient side thoracic ultrasound, the cardiac ventricles appear to be doing little and this is supported by cardiac measurements if taken. Treatment with positive inotropes (dobutamine infusion or pimobendan) can be considered. It is also important to review the patient for any underlying cause. Sepsis may be associated with myocardial dysfunction and decreased systolic function, but other causes include hypocalcaemia and acidosis.

5. Acidosis

In addition, acidosis can be a cause of cardiovascular instability in its own right. Acidosis can cause impaired contractility, cardiac arrhythmias and vasodilation, resulting in a decreased systemic vascular resistance. Patients with severe acidosis may also have mental depression and respiratory changes as the body tries to compensate. Acidosis can also result in an attenuation of the normal response to catecholamines (most of the commonly used vasopressors and inotropes with the exception of vasopressin).

Any acidosis associated with the presence of shock (metabolic acidosis due to lactate or azotaemia, or respiratory acidosis secondary to recumbency and respiratory depression) usually improves with fluid therapy and initial stabilisation. Severe, life-threatening, acidosis (usually where the pH is around 7.0 or less), separate life-saving treatment may be necessary. Sodium bicarbonate is the treatment of choice (see webinar 3 for further details of administration and considerations).

6. Is there hypoxaemic shock present?

Assessment of oxygenation and/or trial treatment with oxygen therapy can help unstable patients. Treatment of any underlying cause of hypoxaemia will also be necessary.

Additional factors for consideration include:

7. Hypoglycaemia
8. Anaemia (especially in cats)
9. Concurrent drug therapy (examples include acepromazine, anaesthetic drugs and vasodilating drugs such as prazosin)

Rationale use of vasopressor and inotropic agents

Vasopressors and knowing which patients to use them in and how to initiate therapy can be simple and practical. Indications for vasopressor therapy include

- Sepsis – decreased systemic vascular resistance. Vasopressors are often required and should be initiated in any septic patient with an incomplete response to fluid therapy after consideration of the factors listed above. This should go along with early antibiotic therapy and rapid source control in order to achieve the best outcome for the patient.
- During CPR to achieve peripheral vasoconstriction and increase DO_2 to the vital central organs
- Anaphylaxis – vasopressor therapy can stop degranulation of mast cells and histamine release as well as acting to augment vascular tone and treat hypotension.
- Anaesthesia in the presence of hypotension as many commonly used drugs have some vasodilating effect. If hypotension cannot be removed by a dose reduction, concurrent vasopressor therapy may be needed.

Historically, vasopressor therapy has been initiated later in the clinical course of a patient's management, only after a sustained period of hypotension as an option of last resort. There is data from human medicine however that may need us to challenge this approach and initiative therapy earlier in cases that need it. In a study looking at early versus delayed norepinephrine in patients with septic shock, each hour delay in vasopressor therapy increased the mortality by 5.3%.

The table below contains a list of commonly used vasoactive agents, showing the receptors the drugs target and their relative effects on inotropy (cardiac contractility), heart rate, vascular tone (vasoconstriction) and blood pressure.

Catecholamine pressors

Drug	β_1	β_2	α	V_1	V_2	Inotropy	HR	Vascular tone	BP
Dopamine	++	+	++	0	0	↑↑	↑↑	↑↑	↑↑
Epinephrine	+++	+++	+++	0	0	↑↑↑	↑↑↑	↑↑↑	↑↑↑
Norepinephrine	+	0	+++	0	0	↑	Varies	↑↑↑	↑↑↑
Phenylephrine	0	0	+++	0	0	0	↓	↑↑↑	↑↑↑
Vasopressin	0	0	0	+	+	0	↓	↑↑	↑↑
Terlipressin	0	0	0	++	+	0	↓	↑↑↑	↑↑

Non-catecholamine pressors

Dobutamine	++	+	+	0	0	↑↑	↑	↓	Varies

Inotrope

The most commonly used drugs in emergency veterinary practice at the present time include norepinephrine as the first-choice vasopressor, and pimobendan or dobutamine for improving cardiac contractility. Phenylephrine may be used more under anaesthesia to treat anaesthesia mediated hypotension owing to its preferential effect on vascular tone.

Dobutamine is an inotrope and, as mentioned, used to treat impaired contractility. It is important to note that it is an inodilator, meaning is can cause some degree of vasodilation and possible hypotension. This should be considered in the clinical patient that is thought may have a concurrent decreased systemic vascular resistance from decreased vascular tone as its use may result in

hypotension. Should this occur then the use of dobutamine be reconsidered, a lower dose used, concurrent contributing factors to hypotension also addressed and more vasopressor therapy given. Dobutamine may also cause some ventricular arrhythmias and so it should also be used with caution in patients with pre-existing arrhythmias or those that develop them during therapy.

Norepinephrine is the most commonly used vasopressor medication. It is typically started in patients with vasodilation when it becomes clear that they are not going to be adequately perfused with fluid therapy alone. Absolute hypotension is an indication but in people it is started when there is a decrease in blood pressure of 40 mmHg from their baseline (for example if a patient's normal blood pressure is 160 mmHg and it has decreased to 120 mmHg which is not normal for them). All vasoactive drugs should be delivered via a dedicated catheter and should not be bloused to avoid marked adverse cardiovascular effects. A target blood pressure of a systolic pressure > 100 mmHg or a mean arterial pressure of > 65 mmHg should be used as a guide, along with regular reassessment of the perfusion parameters and overall clinical picture of the patient.

Doses of the commonly used vasoactive medications are as follows:

	Receptor Activity			Effect on*					
	β_1	β_2	α_1 & α_2	Contractility	Heart Rate	Cardiac Output	Vasomotor Tone	Blood Pressure	Dosage
Isoproterenol	+++	+++	0	↑↑↑	↑↑↑	↑↑↑	↓↓↓	↓↓↓	0.02-0.5 mcg/kg/min
Dopexamine	0	++	0	0	↑	↑↑	↓↓	↓↓	1-10 mcg/kg/min
Dobutamine	++	+	+	↑↑	↑	↑↑	↓	Variable	5-20 mcg/kg/min
Dopamine	++	+	++	↑↑	↑↑	Variable	↑↑	↑↑	5-20 mcg/kg/min
Ephedrine	+	+	+	↑	↑	↑	Variable	↑	0.25-1 mg/kg
Epinephrine	+++	+++	+++	↑↑↑	↑↑↑	↑↑	↑↑↑	↑↑↑	0.05-1 mcg/kg/min
Norepinephrine	+	0	+++	↑	Variable	Variable	↑↑↑	↑↑↑	0.1-2 mcg/kg/min
Phenylephrine	0	0	+++	0	↓	↓	↑↑↑	↑↑↑	0.5-5 mcg/kg/min
Vasopressin	0	0	0	0	↓	↓	↑↑	↑↑	0.5-5mU/kg/min
Angiotensin	0	0	0	0	0	↓	↑↑	↑↑	0.01-0.1 mcg/kg/min

*Effects are estimated for the higher dose ranges.
Activity ranges from no activity (0) to maximal activity (+++).
Possible cardiopressor effects include a decrease (↓), mild increase (↑), moderate increase (↑↑), or marked increase (↑↑↑).

Infusions may need to be calculated and prepared rapidly in an unstable patient; an easy guide to doing this is included below.

- Weight of patient 10kg
- Example starting dose of norepinephrine 0.05 mcg/kg/min
- Calculate the lowest pressor dilution rate to run at a rate of a simple number such as 1ml/hr
  - 0.05 mcg/kg/min x 10kg x 60 min = 30 mcg/hr
- You probably want enough for 24 hours
  - 30 mcg/hr x 24 hours = 720 mcg/24 hours
- My norepinephine is a 1mg/ml solution
  - 720 mcg/1000 x 1mg/ml = 0.72 ml norepinephrine
- This means you need 0.72 ml of norepi in 24 hours to run at 1 ml/hour
  - 24ml total – 0.72 ml norepinephrine = 23.3 ml saline diluent
  - At 1 ml/hr this delivers 0.05 mcg/kg/min
  - At 2 ml/hr this delivers 0.1 mcg/kg/min

Vasopressor therapy is typically initiated at the lower end of the dose range and its effect on the patient's blood pressure and perfusion closely monitored frequently. Typically, this means checking blood pressure every 10-15 minutes. Should a satisfactory effect be achieved the drug is continued at

this dose and the blood pressure monitored to ensure it remains stable. If not, the dose is typically increased in very small increments (for example by 0.1 mcg/kg/min in the case of norepinephrine or 1 mcg/kg/min in the case of dobutamine) and the process repeated until a desirable effect is achieved. If the drug has been sequentially titrated up towards the upper end of the dose range and cardiovascular stability has still not been achieved, a 2nd agent can be added if appropriate on top of the first infusion as long as there are no adverse effects.

In order to avoid sudden patient destabilisation, it is essential that infusions of vasoactive drugs are not stopped suddenly and should remain connected to the patient at all times. Even if the clinical effect of the drugs is not significant, sudden withdrawal could result in rapid patient deterioration and so is best avoided. New syringes of the infusions should be prepared well in advance of being needed to ensure there are no unnecessary breaks in therapy. When the time has (hopefully) some to wean the patient off the infusions, it is also essential that this is done gradually. This may be considered when the underlying disease process associated with the hypotension is resolving, and the blood pressure has remained stable for some time, comfortably within the targeted range as stated above. Dose reduction should be done very gradually and the blood pressure checked frequently. If the blood pressure holds despite dose reduction for a period of 1-2 hours, the dose may be further reduced. If the blood pressure drops and/or perfusion is decreased, the dose should be increased back to the last effective dose and the patient re-evaluated.