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Emergency Room Crash Course Online 'Mini Series'

Session 1: Boluses or Low Rates- How to Use Fluid Therapy in Shock Versus Dehydration

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SHOCK AND SYSTEMIC PERFUSION ASSESSMENT

Shock

Shock essentially occurs when **systemic tissue oxygen delivery reaches a critically low level**. This may be due to:

- Systemic hypoperfusion
- Severe hypoxaemia
- Severely reduced oxygen-carrying capacity, typically due to severe anaemia

Inadequate oxygen delivery to cells means that they are unable to produce energy - there is a discrepancy between oxygen delivery and cellular oxygen demand. If the degree of shock is sufficiently severe or prolonged, irreversible cell damage can occur and treatment is invariably unsuccessful.

Systemic hypoperfusion is the most common cause.

Systemic perfusion

Perfusion:

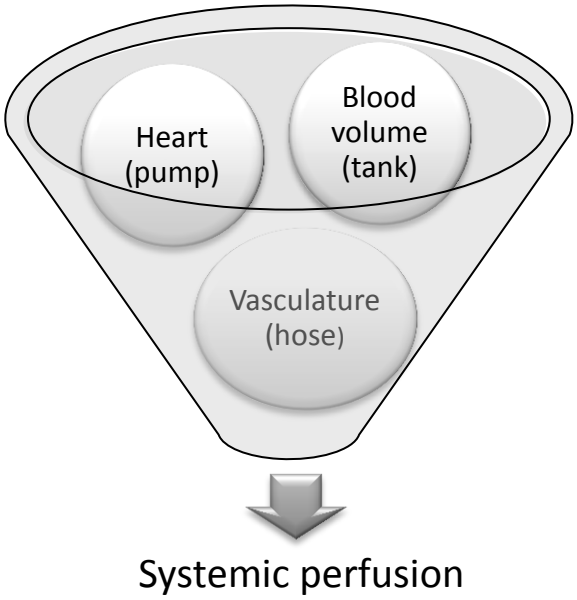
"the passage of a fluid through a specific organ or an area of the body." (Mosby's Medical Dictionary, 8th edition. © 2009, Elsevier)

"Bathing an organ or tissue with a fluid" (McGraw-Hill Concise Dictionary of Modern Medicine. © 2002 by The McGraw-Hill Companies, Inc)

What we are talking about here is assessing *systemic or generalised tissue/organ perfusion, i.e. the 'macrocirculation'.* In veterinary medicine, cardiovascular examination forms the mainstay of this assessment. There is much debate about the sensitivity and accuracy of physical examination parameters for assessing systemic perfusion and on-going research and investigation in human medicine into additional or indeed alternative and more sensitive parameters. One such parameter is blood lactate concentration which will also be discussed here. However physical examination is easy to perform and repeat and free of charge (after the initial fee!); it remains crucial in veterinary medicine. These notes will also mention systemic arterial blood pressure measurement and measuring urine output.

Systemic perfusion is essentially determined by three things and that one or more of these may be abnormal in any individual patient:

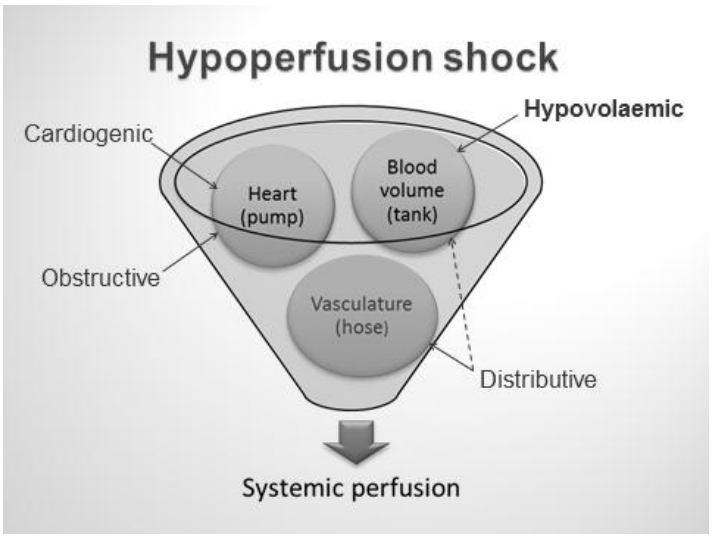
- The tank, i.e. intravascular fluid/blood volume
- The pump, i.e. how well is the heart able to pump blood.
- The hose: what is the size of the vascular space; are the perfusing vessels dilated or constricted? And is there a problem with flow through the container? Is flow obstructed in any way?

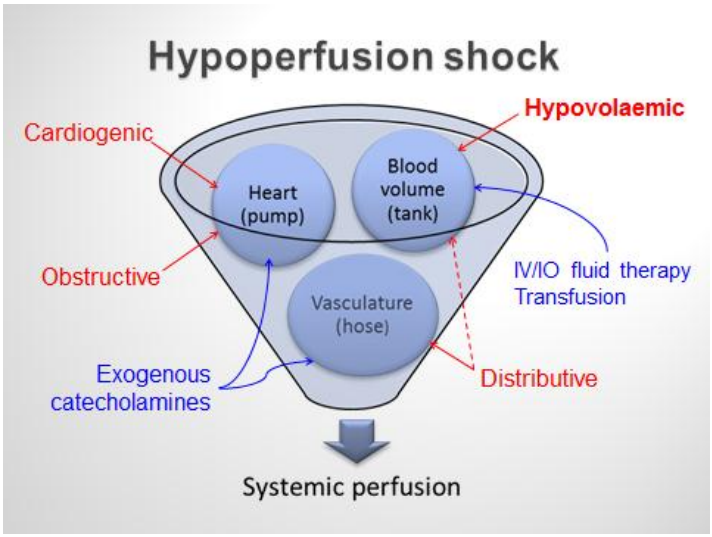


There are **four types of hypoperfusion-related shock** but clearly more than one type may be present in the same patient:

Hypovolaemic shock
Distributive shock
Cardiogenic shock
Obstructive shock

Remember that in any patient in shock from systemic hypoperfusion, there may be one or more of the four forms occurring concurrently. Various disorders can cause systemic hypoperfusion via different mechanisms that may affect the intravascular fluid volume, the size of the vascular container and/or the cardiac pump.





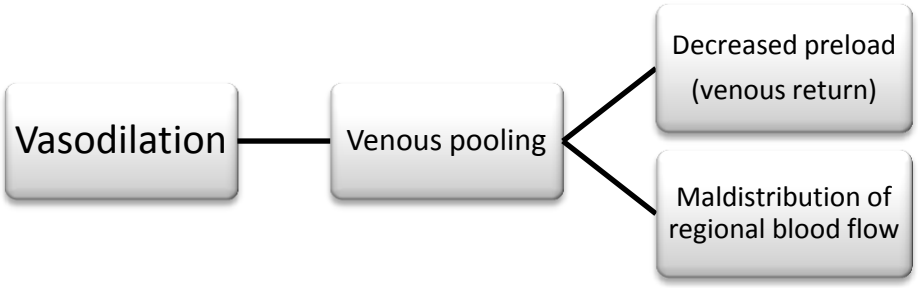
Hypovolaemic shock

Hypovolaemia = reduction in *effective circulating intravascular volume*
Effective circulating intravascular volume = that part of the extracellular fluid (ECF) within the vascular space that is 'perceived' by the body (i.e. by the relevant receptor mechanisms) as effectively perfusing tissues.
Most common cause of systemic hypoperfusion in dogs and cats; fluid loss is hypertonic or isotonic (tonicity is related to sodium concentration).
Fluid loss may affect just the intravascular space (e.g. early on in haemorrhage) or the extravascular space as well (e.g. salt and water loss, for example in vomiting and diarrhoea).

See more below

Distributive shock

Generalised inappropriate vasodilation (predominantly due to the vasomotor effects of inflammatory mediators) occurs so that the size of the vascular container is increased, sometimes quite spectacularly, i.e. there is abnormal vessel tone with decreased afterload. This means that even if no loss of intravascular fluid has occurred (i.e. there isn't an absolute hypovolaemia), systemic perfusion may be inadequate with a concurrent state of relative hypovolaemia and fluid being distributed in an abnormal way.



Assessment of perfusion parameters in distributive shock can be more challenging with sometimes less predictable changes. As with hypovolaemic shock, the treatment of distributive shock involves the use of aggressive intravenous crystalloid fluid therapy to 'fill the container'. Many of these patients have systemic vasculitis and there may therefore be a rationale to use a synthetic colloid early in these patients.

More severe cases can have spectacular vasodilation with minimal improvements in perfusion despite significant intravenous fluid resuscitation. The early use of vasopressors in patients with moderate-to-severe distributive shock may be rational to 'shrink the container'. Myocardial depression is thought to be a component of septic shock and an agent with a positive inotropic effect may be rational.

Causes:

- Systemic inflammatory response syndrome (SIRS)
    - Sepsis
    - Severe acute pancreatitis
    - Major tissue trauma
    - Neoplasia
    - Burn injury
- Anaphylaxis/anaphylactoid reaction
- 'Neurogenic' (spinal) shock

Dogs:

- Sepsis and severe acute pancreatitis are the two most common causes of distributive shock in dogs.

- Early on when intravascular volume remains adequate, dogs in distributive shock typically have a *hyperdynamic vasodilatory* cardiovascular picture, i.e. tachycardia, hyperdynamic pulses, markedly hyperaemic mucous membranes and a fast capillary refill time.
- With the onset of concurrent severe hypovolaemia, tachycardia progresses but pulses become weaker and CRT more prolonged. However, unlike dogs with uncomplicated severe hypovolaemia, dogs in *severe hypodynamic* distributive shock are likely to retain colour in their mucous membranes (due to lack of peripheral vasoconstriction) that may be normal in appearance or hyperaemic through to red.

Cats:

- Major tissue trauma is a relatively common cause of SIRS in cats and when this occurs as a result of bite wounds, sepsis may also be present.
- As with hypovolaemia, and unlike dogs, cats in distributive shock are more likely to present with a hypodynamic cardiovascular picture that is typical of hypovolaemic shock in this species

Cardiogenic shock

When presented with a patient in shock it is tempting to start aggressive intravenous fluid therapy in all cases. However such therapy may clearly be disastrous in patients with cardiogenic shock and so it is important to always be alert to findings – signalment, historical and clinical – that may implicate cardiac dysfunction as the cause of the shock and to respond appropriately in terms of fluid use.

Primary systolic or diastolic cardiac dysfunction results in an inability of the heart to pump blood adequately with reduced cardiac output (forward failure) and consequent haemodynamic abnormalities that may include systemic hypoperfusion (cardiogenic shock). Cardiogenic shock is essentially an extreme manifestation of forward heart failure with pump malfunction.

Causes of cardiogenic shock included cardiomyopathy, valvular disease, and severe tachy- or bradydysrhythmias. Depending on the underlying cause, treatment of cardiogenic shock may include diuresis for congestive heart failure, anti-dysrhythmic therapy and potentially use of a positive inotrope. All patients will likely benefit from supplemental oxygen and minimal stress is essential.

Obstructive shock

Obstructive shock is the result of obstruction of arterial blood flow from the heart or of venous return to the heart. The most common example of this in small animals is pericardial tamponade. Other causes include severe pneumothorax, constrictive pericarditis and pulmonary thromboembolism (PTE). Depending on the cause some of these patients may benefit from judicious fluid therapy but care is needed and cardiovascular status may not improve unless the obstruction can be relieved.

Systemic perfusion assessment

Cardiovascular examination – always the priority!

- Plus subjective cardiac contractility on emergency ultrasound

Arterial blood pressure
Blood lactate concentration
Urine output

Cardiovascular examination

Evaluation involves examination of the following:

- Heart rate and rhythm
- Heart sounds
- Pulse quality and rate
- Mucous membrane colour
- Capillary refill time
- (Mentation; temperature of the extremities)

The most important questions to answer are:

- Is systemic perfusion normal? Perfusion is a key concept in emergency medicine.
- Is there a murmur or gallop sound suggesting possible primary cardiac disease?

Heart rate and rhythm

Normal adult range in **dogs**: 70-120 bpm

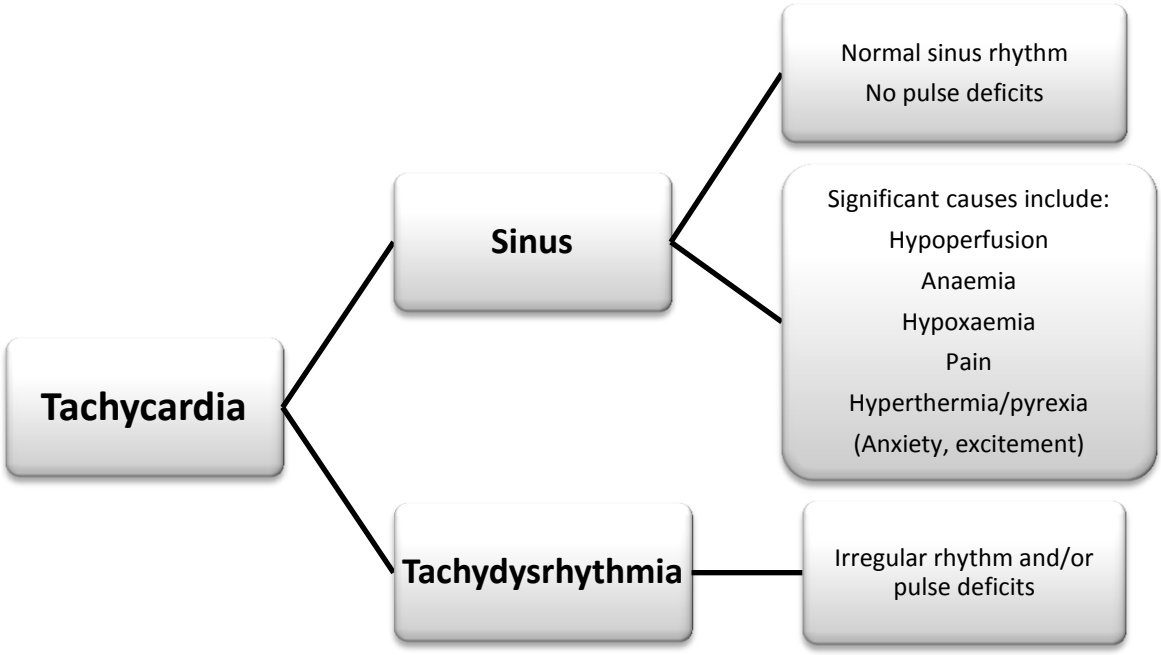
- Larger dogs usually have slower rates than smaller dogs
- Potentially affected by various other factors
- Interpret the measured HR in the context of each individual patient

In general when interpreting physical examination findings it is essential to ask not just if the measured value falls within quoted 'normal ranges', but whether it is *appropriate for the individual patient in question.*

Normal adult range in **cats**: 160-200 bpm (at initial consultation)

- More susceptible to 'stress' and much higher rates (e.g. 220-240 bpm) may be found in healthy cats

Tachycardia



Bradycardia

Both sinus bradycardia and bradydysrhythmias considered significant. Causes include:

- High vagal tone: many causes, e.g. respiratory disease, intra-abdominal disorders
- Drug side effect: especially anaesthetic agents and (pure) opioids
- Cardiac conduction disturbances (e.g. atrioventricular block)
- Brain disease (secondary to raised intracranial pressure)
- Hyperkalaemia (e.g. urethral obstruction, Addison's disease)
- Hypoglycaemia (probably only if moderate-to-severe)
- Hypothermia

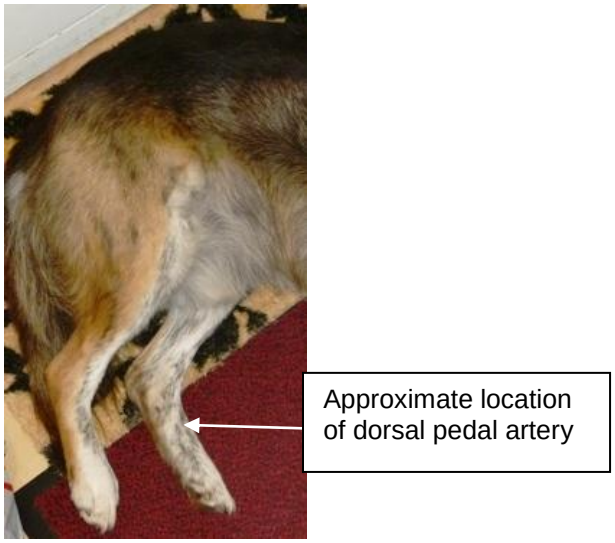
Heart sounds

Most dogs with clinically significant heart disease have a readily audible murmur.
Many cats with clinically significant heart disease have a readily audible gallop sound (third heart sound) or murmur.
Murmurs are more likely to be audible at higher rates or in the presence of greater sympathetic stimulation of the heart.
Quiet heart sounds may be due to muffling (especially from pericardial effusion; also e.g. pleural effusion, mass/organs between stethoscope and heart) but possibly also hypovolaemia.

Normal cardiac auscultation does not exclude the presence of clinically significant heart disease.

Pulse quality and rate

Arterial sites: especially *femoral* (medial aspect of proximal hindlimb) and *dorsal pedal* (metatarsal; distal to hock on craniomedial aspect).



Normal arterial pulse: readily palpable and strong
Stronger or easier than normal to feel: 'bounding', 'snappy', 'hyperdynamic'

- Typically tachycardic
- Causes e.g. early shock, anaemia

Weak, very weak/'thready' or absent: e.g. later stages of shock; low output (forward) heart failure

Pulse deficit: pulse rate lower than concurrent heart rate

- Due to dysrhythmia with some heart beats unable to generate palpable peripheral arterial pulse
- Perform ECG to evaluate further

Mucous membrane colour

Normal salmon pink appearance requires there to be enough oxyhaemoglobin in enough red blood cells in the capillaries of the mucous membrane site being examined. Abnormalities are:

Pallor
Injection/congestion
Cyanosis
Other abnormalities

Pallor

Causes of:

- Poor blood flow due to vasoconstriction (e.g. common in shock; also hypothermia)
- Overall lack of circulating red cells in anaemia

Injection/congestion

Increased blood flow (hyperaemia) gives membranes that range from being pinker than normal right through to bright red
Mild cases, e.g. due to tachycardia and increased cardiac contractility in early shock
More severe cases are usually due to vasodilation and tachycardia, e.g. systemic inflammation, vasodilatory anaphylaxis, hyperthermia.

Cyanosis

Bluish or purplish discoloration of the mucous membranes or skin due to a severe decrease in the amount of oxygen dissolved in the perfusing blood.
Central cyanosis typically indicates life-threatening systemic hypoxaemia due to circulatory and/or ventilatory failure.
It may be difficult to detect if perfusion is very poor or the patient has a packed cell volume (PCV) < 15% (as > 5 g/dL of deoxyhaemoglobin is required for cyanosis to be visible).

All cyanotic patients require urgent intervention with oxygen supplementation and addressing the underlying cause if/as possible.

Other abnormalities

Icterus (jaundice) is a yellow appearance of the mucous membranes or skin due to hyperbilirubinaemia.

Petechiae (pinpoint) and **ecchymoses** (larger) are the result of superficial bleeding. They are typically due to severe thrombocytopenia, platelet function disorders (thrombopathias), or a localised or systemic vasculopathy.

Increases in the concentration of **other types of haemoglobin** besides oxy- or deoxyhaemoglobin, e.g.

- Methaemoglobinaemia in paracetamol (acetaminophen) poisoning causes brownish discolouration
- Carboxyhaemoglobinaemia in carbon monoxide poisoning causes a cherry-red appearance

Capillary refill time

Affected by blood flow to the site and therefore varies predominantly according to whether the underlying vessels are constricted or dilated:

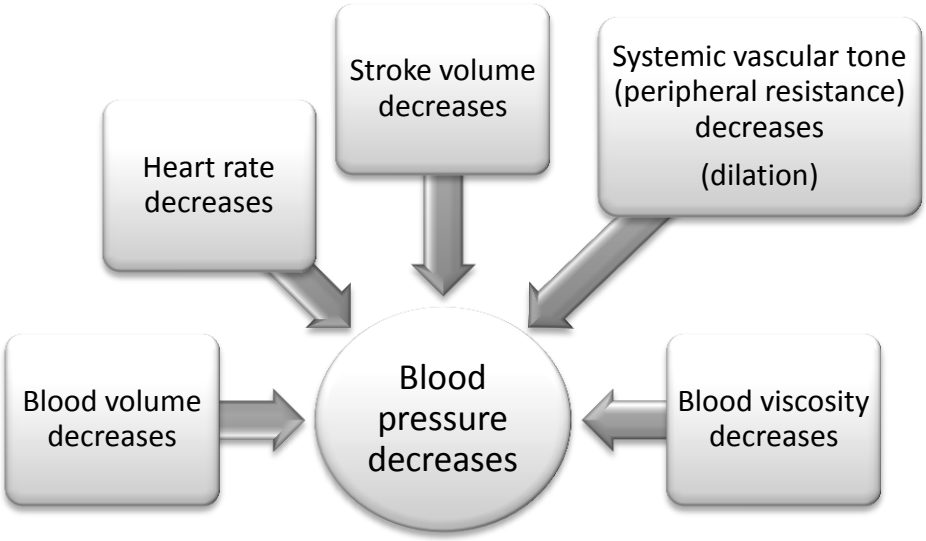
- Normal: 1-2 seconds
- Slow (> 2 seconds): especially in peripheral vasoconstriction (e.g. shock, hypothermia)
- Fast (< 1 second): fastest times especially seen in vasodilatory states (e.g. systemic inflammation, hyperthermia) – often accompanied by tachycardia and congestion.
    - CRT may also be fast in mild (early compensatory) hypovolaemic shock.

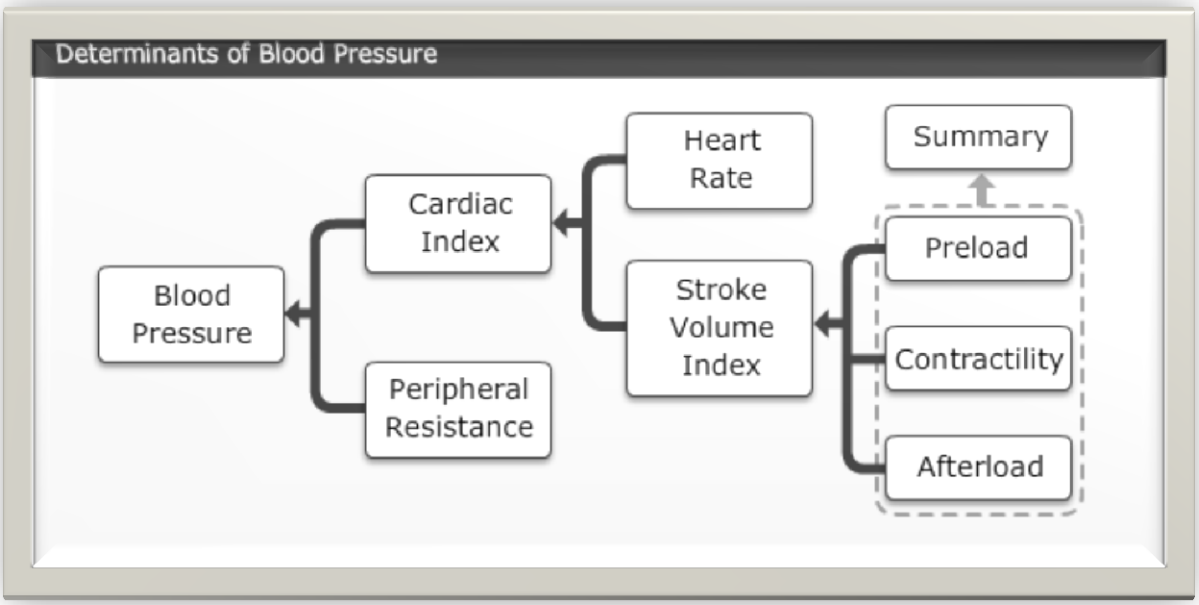
Common causes of cardiovascular abnormalities				
Hypovolaemia	Heart disease/ failure Cardiogenic shock	Systemic inflammation including sepsis	Anaemia	Several others, e.g. pain, hyperthermia or pyrexia, central neurological cause

Systemic arterial blood pressure

Measurable hypotension can be a late event in hypovolaemia so a patient may be in shock with reduced organ perfusion despite normal arterial blood pressure.

Systemic arterial blood pressure depends on a number of factors.





(Preload = volume of blood in ventricle; Contractility = force of contraction applied; Afterload = resistance to contraction)

Normal arterial blood pressure (mmHg)	Adult dog	Adult cat
Mean	60-100	60-100
Systolic	90-140	80-140
Diastolic	50-80	55-75

Mean arterial pressure (MAP) is most important.
There is no direct correlation between systolic or diastolic pressure and MAP.
Peripheral indirect blood pressure measurement may be less accurate in:

- Very small animals
- Hypotension
- Dysrhythmias
- Peripheral oedema
- Vasoconstriction

Doppler ultrasonography measures systolic blood pressure; this technique may be the most reliable indirect method in animals under 10 kg or those with hypotension or dysrhythmias.
There is some suggestion that the Doppler technique may underestimate systolic blood pressure by 10-15 mmHg in cats and more closely approximate MAP; however this was in one single study of anaesthetised healthy cats so caution is needed in interpretation.
One shortcoming of using Doppler ultrasonography is the inability to measure diastolic blood pressure; the significance and importance of diastolic hypotension is something that so far has received little attention but is likely to in the future.

Oscillometric sphygmomanometry devices usually measure MAP and then use this to calculate systolic and diastolic pressures via algorithms; MAP may therefore be most reliable. Newer devices may be more reliable in general in cats and smaller dogs than older devices were.

The recommended guideline for indirect BP measurement is that the width of the cuff should be 30-40% of the circumference of the leg at the site of the artery being used. Too small a cuff will likely yield a falsely high pressure; too large a cuff, a falsely low pressure.

Basic approach to hypotension

Address the underlying cause if possible. Causes include:

- Decreased cardiac output due to reduced circulating volume (hypovolaemic shock) or blood flow obstruction (obstructive shock)
- Myocardial failure (cardiogenic shock)
- Severe tachydysrhythmia or bradydysrhythmia (cardiogenic shock)
- Decreased systemic vascular resistance due to peripheral vasodilation, e.g. sepsis (distributive shock)

Administer aggressive intravenous fluid therapy tailored to resuscitation end-points (typically physical examination parameters, blood pressure, lactate) – contraindicated for cardiogenic causes.
Consider early use of vasopressor/inotrope, especially in distributive shock. Reliable blood pressure monitoring is preferred when using vasopressors or inotropes.

Blood lactate measurement

Basic lactate physiology

Lactate production is a protective response by the body to allow cellular energy production to continue when tissue oxygen supply is inadequate for aerobic metabolism.
When lactate production exceeds lactate clearance (mostly by the liver and to a lesser extent the kidneys), plasma lactate rises (hyperlactataemia).
Hyperlactataemia is a marker of the severity of tissue hypoxia. It is not always associated with acidaemia or indeed abnormal base deficit depending on buffer reserves and other existing acid/base disturbances.
In normal dogs, arterial and venous, both jugular and cephalic, lactate concentrations are very similar and differences are not clinically significant.

Causes of hyperlactataemia

Two important points about lactate to bear in mind are:

1) Not every patient will show the same degree of hyperlactataemia for the same severity of systemic hypoperfusion and in fact in a small proportion of patients despite reduced tissue oxygen delivery lactate does not seem to rise.

2) There are some patients that have so-called ‘cryptic’ shock where hyperlactataemia is present due to reduced tissue oxygen delivery and yet haemodynamic parameters including blood pressure are normal (normotensive shock), i.e. based on our usual assessment the patient does not appear to be in shock.

Type A hyperlactataemia (Due to tissue hypoxia)	Absolute oxygen deficiency	Generalised systemic hypoperfusion (Shock) MOST COMMON CAUSE	Hypovolaemic Distributive Cardiogenic Obstructive
		Local hypoperfusion	Aortic (and other) thromboembolism Splanchnic ischaemia, especially with gastrointestinal necrosis
		Severe hypoxaemia	E.g. P_aO_2 less than 30-40 mmHg
		Severe anaemia without hypoperfusion	Typically PCV less than 10-15 %
	Relative oxygen deficiency (energy requirements greater than aerobic metabolism can provide)	Increased glycolysis: Hyperlactataemia typically resolves quickly in these cases	Extreme muscle activity (e.g. seizures, trembling/tremors) Strenuous exercise
Type B hyperlactatemia (No clinical evidence of tissue hypoxia) For some/many of these causes the mechanism(s) for plasma lactate increase remains to be clarified. Furthermore it may be that in some cases occult hypoperfusion exists but cannot be detected. *Hyperlactataemia in sepsis is likely multifactorial, especially from hypoperfusion, hypermetabolism and possibly abnormal lactate metabolism. *(Based on Hughes, D. Lactate: What Does It Really Tell Us? IVECCS Proceedings 2010)*		Underlying diseases	Sepsis* Severe liver disease with insufficiency Neoplasia (especially lymphoma) Diabetes mellitus Phaeochromocytoma Thiamine deficiency
		Many different drugs and toxins, e.g.	Paracetamol (acetaminophen) Cyanide Adrenaline (Epinephrine) Ethanol Ethylene glycol Glucose Insulin Morphine Nitroprusside Propylene glycol (e.g., in activated charcoal) Salicylates Terbutaline
		Congenital metabolic defects	Mitochondrial myopathy Defects in gluconeogenesis
		Miscellaneous	Alkalosis/hyperventilation Hypoglycaemia

Clinical aspects of hyperlactataemia

Single lactate measurements should not be used prognostically but serial measurements have a role. It is more valid to monitor whether lactate normalises or not with therapy which will be affected by the severity of the underlying problem and how well it can be controlled (e.g. blood loss versus sepsis).

Moderate-to-severe hyperlactataemia is typically due to systemic hypoperfusion or muscle activity (seizures, exercise).

Systemic hypoperfusion is the most common cause of hyperlactataemia (Type A) and lactate is most often used as an objective marker of the severity of shock:

- Plasma lactate tends to increase in proportion to the severity of the hypoperfusion; but it is essential to realise that this does not reflect reversibility.
- In hypoperfused patients without an additional on-going cause of hyperlactataemia (e.g. sepsis, neoplasia), hyperlactataemia should resolve relatively quickly (minutes to a few hours) with successful fluid therapy offering a comparatively real-time marker of improving perfusion status.
- In some patients plasma lactate appears to increase initially when aggressive fluid therapy is started before then trending downwards. This is thought to be because improved perfusion to anaerobic tissues allows accumulated lactate to be collected by the circulation; this is then cleared by the liver and kidneys.

In the early stages of hypoperfusion when compensatory mechanisms are adequate, hyperlactataemia may well not be present. The onset of hyperlactataemia typically suggests decompensation (with tissue oxygen deficiency) and should always therefore be considered a serious finding.

Plasma lactate may rise during blood sampling if significant or prolonged restraint is required or prolonged venous occlusion occurs; such hyperlactataemia is typically only mild. However more marked hyperlactataemia may occur if there is trembling or other muscle activity during restraint, especially in cats.

Urine output

'Normal' urine output must be interpreted in the context of each individual patient and a range of **0.5-2 ml/kg/hour** is typical. Normal urine production requires adequate renal perfusion, fluid balance and kidney function. An intact urinary tract is then necessary for normal external urine voiding.

The kidneys receive a significant percentage of the cardiac output and renal vasoconstriction and decreased urine output is an appropriate response to hypovolaemia. Reduced urine production (oliguria) and increased urine specific gravity (hypersthenuria) are suggestive of fluid deficit and the need for more rapid infusion. Therefore normalisation of urine output may be a useful end-point of fluid resuscitation.

Placement of an in-dwelling urinary catheter attached to a closed collection system is reasonably easy to achieve in most cases. However placement of an in-dwelling urethral catheter is not considered a routine part of fluid resuscitation and the associated discomfort and risks of hospital-acquired infection must be borne in mind. Urine output monitoring definitely has a role in a proportion of cases (e.g. very sick animals, post-operative cases).

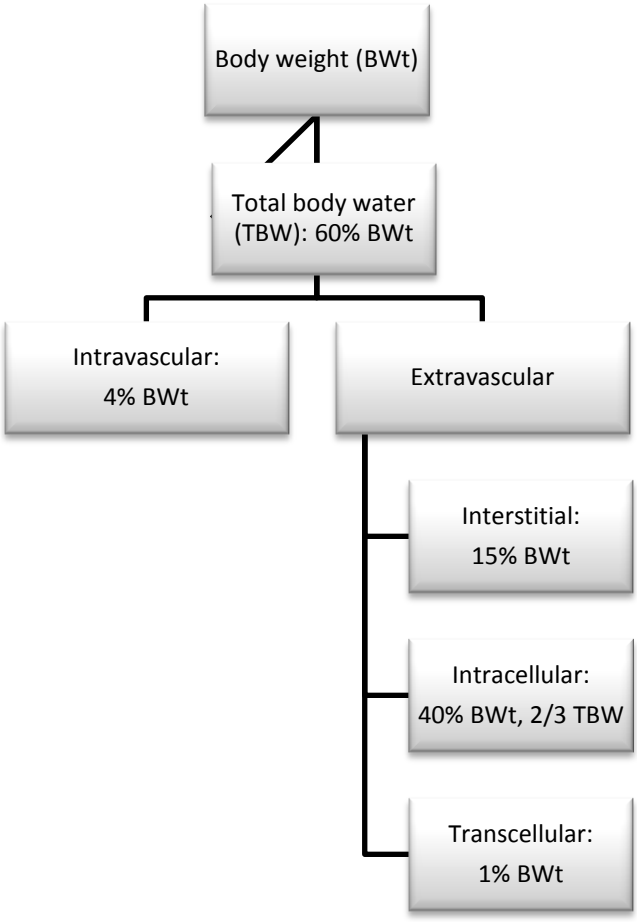
Although not ideal, if an in-dwelling urinary catheter cannot be used, it is possible to estimate urine output using pre-weighed incontinence pads. The patient is only allowed to urinate on incontinence pads which are then weighed; each 1 g increase in weight is approximately equal to 1 ml of urine production

HYPOVOLAEMIA and DEHYDRATION

It is essential to prioritise perfusion assessment and intravascular volume status over hydration concerns.

Hypovolaemia and dehydration are not the same although both are treated with fluid therapy. Hypovolaemia is the most common cause of abnormal perfusion and shock in dogs and cats and can be fatal. A basic understanding of body fluid distribution is essential to appreciate the concepts of perfusion, intravascular volume status, shock and dehydration.

Body fluid compartments



The intravascular fluid compartment is considerably smaller than the extravascular compartment and yet normal intravascular volume status is essential as it is responsible for tissue perfusion. Hypovolaemia is essentially an intravascular volume deficit whereas dehydration predominantly affects the extravascular compartment.

Fluid (water and solutes) continually shifts between compartments under the influence of osmotic concentration gradients and oncotic and hydrostatic pressure gradients (Starling's forces). In normal animals physiological water losses (through the urinary, respiratory and cutaneous routes) are well compensated by fluid movement between compartments. Abnormal fluid losses may be iso-, hypo- or hypertonic and their effects depend on both their magnitude and what changes occur in extracellular fluid osmolarity.

Hypovolaemia

Hypovolaemia = reduction in *effective circulating intravascular volume*
Effective circulating intravascular volume = that part of the extracellular fluid (ECF) within the vascular space that is 'perceived' by the body (i.e. by the relevant receptor mechanisms) as effectively perfusing tissues.
Most common cause of systemic hypoperfusion in dogs and cats; fluid loss is hypertonic or isotonic (tonicity is related to sodium concentration).
Fluid loss may affect just the intravascular space (e.g. early on in haemorrhage) or the extravascular space as well (e.g. salt and water loss, for example in vomiting and diarrhoea).

Causes of hypovolaemia

Most common:

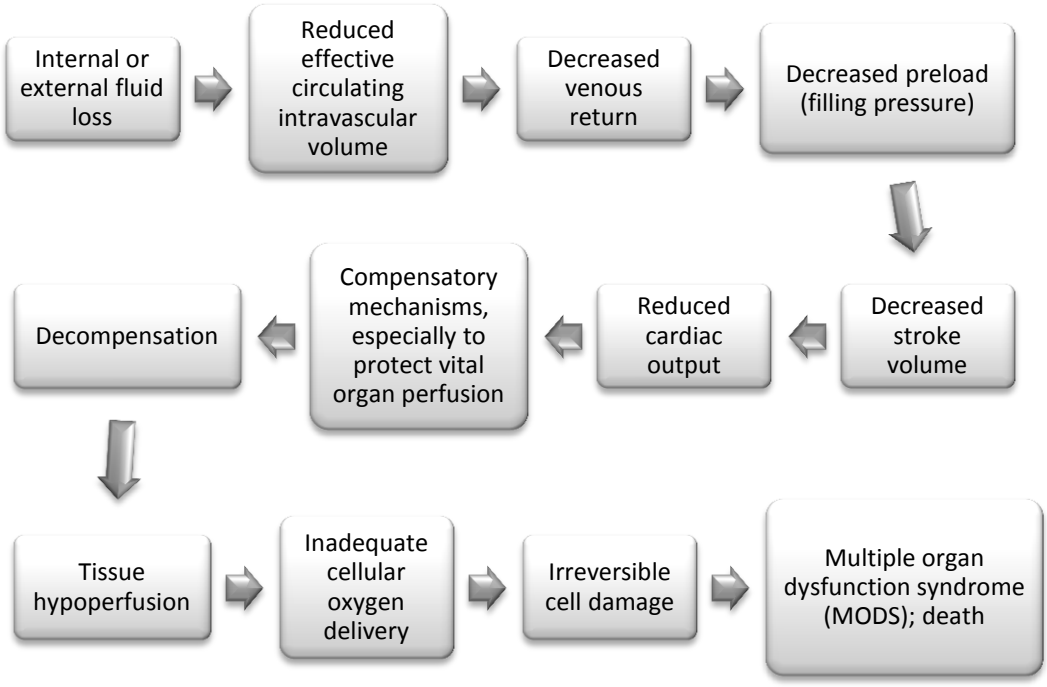
- Haemorrhage:
    - Trauma, coagulopathy, ruptured tumour
- Vomiting and diarrhoea
- Severe dehydration

Less common:

- Third-space fluid loss:
    - Gastrointestinal tract*
    - Peritoneal, pleural or interstitial space
- Burn injury (hypovolaemia is common but burn injury is relatively uncommon in dogs and cats)
- Severe polyuria
- Neoplasia

*The gastrointestinal tract is a large space into which considerable fluid loss may occur, for example in patients with severe gastroenteritis.

Pathophysiology of hypovolaemia



"Shock breeds shock": in shock the tissues of the cardiovascular system also become under perfused which compromises their function reducing their ability to compensate for the shock state, i.e. a vicious circle ensues.

Compensatory mechanisms:

The three main compensatory responses to hypovolaemia in a timeframe of minutes are:

- Increase in heart rate (positive chronotropy)
- Increase in cardiac contractility (positive inotropy)
- Vasoconstriction

These are sympathetic nervous system driven.

Ideally patients will be identified when in the compensatory stage of shock and aggressive intervention started. The prognosis is certainly not hopeless once the patient has started to decompensate (i.e. the physiological reserve is overwhelmed) but decompensation may herald a rapid deterioration into the irreversible stage. Unfortunately some patients do not present until they are already in late decompensation or in an irreversible state of shock – although this may be highly suspected at the time of presentation, there isn't a definitive way of knowing this at the outset.

The hypovolaemic dog

In dogs with 'uncomplicated' hypovolaemia physical examination perfusion parameters tend to change in a predictable way both as hypovolaemia progresses and as normovolaemia is restored.

Perfusion parameter	Mild hypovolaemia Compensatory	Moderate hypovolaemia Early decompensatory	Severe hypovolaemia Late decompensatory
Heart rate	120-150 bpm	150-170 bpm	170-220 bpm
Femoral pulse	Bounding, snappy	Weak	Very weak or thready
Dorsal pedal pulse	Readily palpable	Just palpable	Not palpable
Mucous membrane colour	Normal, pinker	Pale pink	Very pale/white
Capillary refill time	≤ 1 second	1-2 seconds	> 2 seconds or not detectable
Mentation	Usually normal	Depressed	Severely depressed
Extremities	Usually normal	Cool (or normal)	Cold

Compensatory phase/mild hypovolaemia:

- Sympathetic nervous system activation
- Increase in heart rate and cardiac contractility
- Peripheral vasoconstriction
- Hyperdynamic clinical picture

Early decompensatory phase/moderate hypovolaemia:

- Progressive failure of compensatory mechanisms results in reduced tissue perfusion

Late decompensatory phase/severe hypovolaemia:

- Systemic perfusion is severely compromised; hypodynamic clinical picture
- Usually tachycardic but some dogs may have an inappropriately 'normal' or slow heart rate – this is due to failure to sustain the compensatory chronotropic response

Note that physiological sinus tachycardia is unlikely to exceed 220-240 bpm in a dog (300 bpm in a cat). Faster heart rates are likely to be due to tachydysrhythmia.

The hypovolaemic cat

Given that the typical hypovolaemic cat has a hypodynamic clinical picture, a cat presenting like this should be seen as having a life-threatening problem but the prognosis is not necessarily grave if appropriate treatment is provided.

'Cats are not small dogs' and the classic presentation for the hypoperfused cat is a hypodynamic picture with a hyperdynamic picture (tachycardia, hyperaemic membranes, fast CRT) being rarely identified.

Typical hypovolaemic cat:

- 'Inappropriate' bradycardia – heart rate 120-160 bpm
- Weak or absent pulses
- Pale mucous membranes
- Prolonged (> 2.5 seconds) or undetectable CRT
- Depression
- Hypothermia

The reason why cats tend to present with this clinical picture most often remains unclear. In particular the tendency towards bradycardia is unexplained but may well represent a difference in sympathetic-parasympathetic balance between cats and dogs or a greater degree of autonomic dysfunction in cats. Or potentially a relative dominance in cats of a cardiovascular reflex known as the Bezold-Jarisch reflex may be involved but this is just conjecture.

[There remains much to be unravelled about the BJR but its physiological roles may include blood pressure regulation, homeostatic response to hypovolaemia, and effector of haemodynamic changes during myocardial ischaemia. Stimulation of cardiac inhibitory receptors (by currently unknown stimuli) may trigger the reflex causing bradycardia and peripheral vasodilation with consequent hypotension. This reflex has been studied experimentally in cats with artificial stimulation of the reflex but its role in non-experimental cats remains entirely unclear.]

Should hypothermic hypovolaemic cats be warmed aggressively?

Hypothermia significantly decreases the cardiovascular response to fluid resuscitation. However it is possible that re-warming the patient too quickly before administering sufficient fluid therapy may worsen their perfusion status as the increase in body temperature may cause peripheral vasodilatation thereby increasing the intravascular space. Not everyone agrees with this – some people believe that non-sweating animals like dogs and cats do not vasodilate (significantly) with warming especially when core temperature is low and there is even some suggestion that cats in shock may already be relatively vasodilated peripherally compared to dogs. These individuals therefore argue for more aggressive warming early on.

However the most widely accepted recommendation is to limit on-going heat loss with passive warming (e.g. wrap in blankets, place in incubator) during initial fluid resuscitation. In many cases as the patient's perfusion improves their rectal temperature will often increase notably; however more aggressive warming can be performed following initial fluid resuscitation if still thought necessary.

'Complicated' hypovolaemia

Hypovolaemia is not always 'uncomplicated'. A proportion of hypovolaemic patients have other abnormalities that may affect one or more of the physical perfusion parameters described. Common examples include:

- Mucous membranes may remain pale despite appropriate fluid therapy in an animal with severe anaemia
- Mentation may remain depressed in an animal with head trauma despite appropriate fluid therapy as a result of raised intracranial pressure
- Dogs suffering an Addisonian crisis may be inappropriately bradycardic despite poor perfusion for a variety of reasons of which one is hyperkalaemia
- Animals with urethral obstruction or urinary tract rupture may be inappropriately bradycardic due to hyperkalaemia

Dehydration

For clinical purposes, dehydration is water **and solute loss in excess of intake.**
Excess loss in disease states may occur via the:

- Gastrointestinal tract: most common route in small animals, especially with diseases that cause vomiting and diarrhoea
- Urinary tract with polyuria (e.g. chronic renal failure)
- Less common routes include the respiratory system (excessive panting), the skin (i.e. burns), and third-spacing (ascites, peritonitis, pleural effusion).

Excess fluid loss in disease states is usually associated with a decrease in water intake (anorexia).
Fluid loss is hypotonic or isotonic (tonicity is related to sodium concentration).

Fluid loss in dehydration is mainly from the extravascular compartment. The effect on intravascular volume depends on both the degree of fluid loss and the tonicity (isotonic or hypotonic) of the fluid.

Hydration assessment

History

The history can provide an indication of potential fluid deficits. The most important information includes:

- Food and water consumption
- Gastrointestinal losses, i.e. vomiting and diarrhoea: number of episodes and approximate quantities
- Urinary losses: frequency and volume of urination

If the history is suggestive of fluid and electrolyte loss in excess of intake, then the patient should already be considered to be 5% dehydrated.

Physical examination

Physical examination hydration parameters are related to interstitial volume. They are:

- Moistness of mucous membranes
- Skin turgor (elasticity)
- Presence and degree of globe retraction (sunken eyes)

Perfusion parameters become relevant if dehydration is severe enough to cause hypovolaemia.

There a number of different versions of the table below that are used to approximate dehydration on the basis of physical examination.

Severity of dehydration (estimated % of body weight)	Progression of physical examination findings
<5% (NONE)	Normal Assume dehydration based on history

SOME: • Mild (5-6%)	Skin turgor mildly reduced
• Moderate (6-10%)	Skin turgor progressively reduced Mucous membranes dry Eyes sunken
SEVERE (10-15%)	Complete loss of skin turgor Mucous membranes very dry Eyes severely sunken and dull Progressive signs of hypovolaemia, ultimately leading to shock and death

These guidelines only provide a crude estimation of fluid loss in the dehydrated patient. There are a number of potential inaccuracies that must be borne in mind. For example, skin turgor can be affected by the degree of subcutaneous fat present (with obese animals having increased turgor), and mucous membrane moistness can be affected by salivation (e.g. due to nausea). Moreover these sorts of findings are likely to have quite high variability between individual clinicians – i.e. two people assessing the same patient may well come up with a different severity of dehydration if specific percentages are required; hence broader categories of dehydration severity are most rational. In the context of dehydration, it is probably better to under- rather than overestimate severity.

Short-term changes in body weight reflect changes in fluid balance rather than nutritional status

Clinical pathology

All of the following may increase with dehydration:

- Packed cell volume (PCV) and serum total solids (TS)
- Blood albumin (and globulin) concentration
- Blood urea and creatinine concentrations (pre-renal azotaemia is typically mild) and urine specific gravity

Urine specific gravity in dehydration:

Ideally azotaemia should be interpreted alongside a concurrent urine specific gravity (USG). Ideally USG should be determined prior to fluid therapy; however it is essential not to delay fluid therapy unnecessarily for the sole purpose of obtaining a urine sample.

In a dehydrated patient USG prior to fluid therapy should be increased as the kidneys will try to compensate for the fluid loss; this is achieved by enhancing water and sodium reabsorption in the distal part of the renal tubules under the influence of antidiuretic hormone (ADH) and the mineralocorticoid aldosterone.

In some diseases the kidneys are unable to concentrate the urine despite the presence of dehydration due to abnormalities in the renal medullary concentrating gradients (renal medullary washout).

PARENTERAL FLUID THERAPY

While parenteral fluid therapy can be life-saving, especially in non-cardiogenic shock, as with most

drugs it also has the potential to cause harm. Inadequate fluid administration is clearly a concern but remember that over-zealous administration can also be harmful for example causing tissue interstitial oedema, pulmonary oedema being one main concern, and possible dilutional coagulopathy.

Types of parenteral fluids

Crystalloids

Crystalloids are electrolyte solutions that can pass freely out of the bloodstream through the capillary membrane.

Crystalloid solution	Na^+	K^+	Cl^-	Ca^{2+}	Osmolality (mOsm/L)
REPLACEMENT:					
Hartmann's (buffered LRS)	131	5	111	2	272
Lactated Ringers solution	130	4	109	1.5	273
0.9% NaCl ('normal' saline)	154	0	154	0	308
Normosol-R	140	5	98	0	295
Plasmalyte 148	140	5	98	0	295
MAINTENANCE:					
0.45% NaCl + 2.5% dextrose (glucose) [requires additional potassium]	77	0	77	0	203
Normosol-M + 5% dextrose	40	13	40	0	363
Plasmalyte M + 5% dextrose	40	16	40	2.5	377
Plasmalyte 56 + 5% dextrose	40	13	40	0	363
OTHERS:					
0.45% NaCl ('half strength' saline)	77	0	77	0	154
0.9% NaCl + 5% glucose	154	0	154	0	560
0.18% NaCl + 4% glucose	30	0	30	0	
7.2% NaCl (hypertonic saline)	1232	0	1232	0	~2450
5% dextrose in water (D5W) [equivalent to 100% free water)	0	0	0	0	252

Electrolyte concentrations in *mmol/L*

They are classified as *isotonic, hypertonic* or *hypotonic* based on how their tonicity compares to that of extracellular fluid; the tonicity is related to the sodium concentration and it is the tonicity (plus effects on hydrostatic and oncotic pressure, i.e. Starling's forces) that determines how the crystalloid solution is distributed between fluid compartments following administration into the bloodstream.

Isotonic crystalloids

Replacement isotonic crystalloids

Their tonicity and electrolyte composition are similar to that of extracellular fluid. They are an appropriate first choice in the vast majority of cases and are used in two main ways:

- At high rates to treat hypovolaemia either alone or in combination with other fluid types (colloid, hypertonic saline)
- At low rates to replenish fluid deficits in dehydrated patients

The two most commonly used replacement crystalloid solutions are buffered lactated Ringers solution (LRS, Hartmann's solution, compound sodium lactate) and 0.9% sodium chloride (normal strength or physiological saline). They are both typically classified as isotonic although in reality LRS is slightly hypotonic compared to mammalian extracellular fluid.

Following intravascular administration, these fluids equilibrate relatively quickly with the interstitial space and 75-85% of the administered volume is likely to have left the bloodstream 30-60 minutes after infusion. This is why large volumes are required to effectively expand the intravascular compartment in hypovolaemia and is also the reason that these solutions are used to replenish extravascular fluid losses in dehydration.

0.9% sodium chloride vs. buffered lactated Ringers solution – does it matter which one I use?

The composition of these fluids is not the same and the following is noteworthy:

- LRS contains a small amount of calcium and potassium so 0.9% NaCl is usually used in hypercalcaemia and hyperkalaemia
- LRS contains a source of bicarbonate (lactate) and is therefore considered to be alkalinising; 0.9% NaCl does not contain a source of bicarbonate and may contribute to acidaemia via a hyperchloraemic metabolic acidosis – it may therefore be less desirable in acidaemic patients.

While it is worth bearing the above considerations in mind when choosing which fluid to use, in reality in most cases, it makes no clinically significant difference which one is used – the emphasis being on 'no clinically significant difference'. It is definitely more important to make sure that a patient in need of fluid resuscitation receives one or other of these solutions rather than worrying about more theoretical concerns or reported contraindications.

LRS contains a small amount of calcium which can bind to the citrated anticoagulant in blood products. This can inactivate the anticoagulant and promote the formation of clots in the donor product. LRS should not therefore be mixed with blood products.

Maintenance isotonic crystalloids

Commercial maintenance crystalloid solutions are more-or-less isotonic and have an electrolyte composition that is more similar to insensible electrolyte losses in healthy animals. There is therefore an argument for using these solutions in animals requiring maintenance fluid therapy only, i.e. once hypovolaemia and dehydration have been corrected.

However the potassium content of maintenance fluids is relatively high and they are therefore limited to slow infusion rates. Furthermore the majority of hospitalised patients are not 'healthy' and have on-going electrolyte losses and reduced enteral intake. Maintenance crystalloid solutions are therefore very infrequently indicated for sole use in veterinary medicine; some hospitals routinely keep hospitalised patients on maintenance fluids at a maintenance rate, and then use additional replacement fluids to meet additional requirements.

Replacement isotonic crystalloid solutions can be used, and frequently are, for continued fluid therapy in hospitalised patients. However it is important to be aware that:

- These solutions typically contain little or no potassium – therefore hypokalaemia may develop in animals with reduced potassium intake and supplementation may be required.

- These solutions have a relatively high sodium content that can lead to mild hypernatraemia – this is rarely clinically significant but noteworthy nonetheless.

Hypertonic saline

E.g. 3% or 7.2%-7.5% sodium chloride solution
Causes intravascular volume expansion by drawing water in from the extravascular space down an osmotic gradient. Although transient, a rapid intravascular volume expansion is seen (typically within 5 minutes) – in dogs the haemodynamic effect is comparable to a 60-90 mL/kg bolus of replacement crystalloid.

Recommended doses (over 5 minutes): Dogs: 4-7 mL/kg IV
Cats: 2-4 mL/kg IV

Indications:

Volume resuscitation: especially large/giant breed dogs where it may not be possible to administer isotonic crystalloids fast enough

- Commercial preparations including hypertonic saline and synthetic colloid (e.g. hypertonic saline with dextrans) are available; the inclusion of a colloid prolongs intravascular volume expansion.

Intracranial hypertension: hypertonic saline causes fluid to shift from the brain parenchyma into the circulation

- Hypertonic saline is especially useful in head trauma patients that are also hypovolaemic as it addresses both the hypovolaemia and the possible raised intracranial pressure.

The effect of hypertonic saline administration usually lasts for up to 30 minutes and is associated with osmotic diuresis and rapid sodium distribution. It must be followed by the use of a replacement isotonic crystalloid or colloid.

Contraindications:

Concurrent dehydration (these patients already have a deficit in their extravascular compartment which will be worsened by hypertonic saline use)
Heart failure or cardiogenic shock (due to the risk associated with a rapid and sizeable surge in intravascular volume and preload)
Active haemorrhage, especially pulmonary contusions
Oliguric renal failure

Transient hypernatraemia and hyperchloraemia are likely.

Hypotonic saline

Probably the only ready-made hypotonic saline solution that has a clinic use in small animals is 0.45% ‘half strength’ sodium chloride solution.

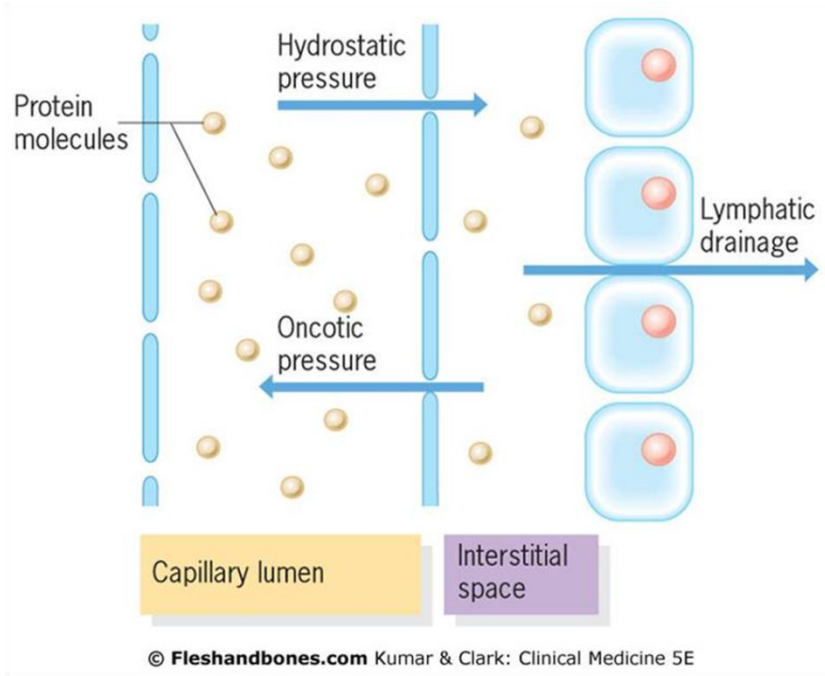
- This is used most commonly together with other solutions in the gradual correction of sodium abnormalities.
- It is also used occasionally at a low rate to replace fluid deficits while limiting the sodium challenge in dehydrated animals with heart disease.
- This solution is 50% free water

Hypotonic fluids should never be used in the resuscitation of hypovolaemia as the decreased sodium content of these fluids causes them to stay in the intravascular space for an even shorter period of time; they are therefore an inefficient resuscitation fluid. Furthermore when administered in large quantities hypotonic fluids can cause severe electrolytes abnormalities (especially severe hyponatraemia).

Synthetic colloids

Replacement crystalloid or synthetic colloid for reperfusion? Opinions vary here to an extent but replacement isotonic crystalloids are widely used as the first fluid of choice in virtually all patients. A synthetic colloid may be indicated early in certain populations ('capillary leak' syndrome; hypoproteinaemia) and in hypovolaemic patients showing poor response to crystalloids. But remember that commercial synthetic colloid preparations are typically colloid molecules in an isotonic crystalloid solution so you are still giving crystalloid when using a synthetic colloid. Remember also that synthetic colloids may have adverse effects and their safety in critical illness in particular remains to be clarified.

Colloid solutions consist of large molecular weight molecules that do not readily leave the bloodstream and therefore increase the colloid osmotic (oncotic) pressure of the plasma.



Overall, there is normally a small net movement of fluid out of the vessel.
With colloids we are manipulating oncotic pressure inside the vessel to "hold" the fluid in the vasculature.
This only works if the capillary endothelium is an effective barrier to movement of colloid – this may not be the case in patients with vasculitis or with small colloids.

Colloid molecules generally remain in the bloodstream for up to several hours. This draws fluid into and holds fluid in the vasculature causing plasma volume expansion. The volume and duration of plasma expansion that follows colloid administration depends in part on the specific colloid used – especially the size (molecular weight) – as well as the dose given and the species in question; other individual patient-related factors are also important.

Proprietary synthetic colloid solutions are often made up to a colloid concentration of 6% or 10% in 0.9% sodium chloride or buffered LRS (Hartmann's). Non-synthetic (natural) colloid solutions that are currently used therapeutically include plasma and albumin solutions.

The three types of synthetic colloid solutions in veterinary use currently are:

- **Hydroxyethyl starches** – variety of products with wide range of molecular weights; e.g.
  - Tetrastarch – e.g. Voluven 130/0.4®, Fresenius Kabi
  - Hetastarch – e.g. Hetastarch 600/0.7®, Baxter [currently unavailable?]
  - Pentastarch – e.g. HAES-Steril 200/0.5®, Fresenius Kabi [currently unavailable?]
- **Gelatins**, e.g.
  - Gelofusine®, Braun
  - Haemaccel®, Intervet
  - Relatively low molecular weight; short-lived effect
- **Dextrans**, e.g. Dextran-70®, Pharmacosmos
  - Available as 40Kd and 70Kd sizes
  - Used more in USA
  - Available as a mixture with hypertonic saline in UK (see above)

At the time of writing Gelofusine®and Haemaccel® are the only synthetic colloids licensed for veterinary use. Both are gelatins and they have very similar properties. The molecules in these solutions are smaller than in other synthetic colloids and in general they cause the shortest duration of plasma volume expansion and are excreted most quickly from the body. Despite being unlicensed, other synthetic colloids are therefore typically preferred due to greater perceived efficacy. Dextrans are solutions of glucose polymers which are commercially available in the United Kingdom although more commonly used in the United States of America. They contain larger molecules than the gelatins and are therefore generally associated with a slightly longer duration of action and slower excretion from the body.

Hydroxyethyl starch (HES) solutions are modified polymers of amylopectin (a plant starch) with the modification designed to increase their persistence in the circulation. Commercially available HES solutions include a variety of products with a wide range of molecular weights. Solutions are classified as:

- High molecular weight, e.g. hetastarch (at the time of writing this solution may be unavailable)
- Medium molecular weight, e.g. pentastarch (at the time of writing this solution may be unavailable)
- Low molecular weight, e.g. tetrastarch

In general HES solutions cause longer plasma volume expansion and are excreted more slowly than other synthetic colloids; they are therefore considered more clinically useful. High molecular weight solutions have the longest duration of effect but are also associated with the greatest side effects.

The most significant side effect associated with synthetic colloids is their potential to cause multifactorial dose-dependent clotting abnormalities. Colloids also have a greater potential to cause intravascular overload and allergic reactions have been reported rarely.

- Gelatins are the colloid reportedly most likely to induce an allergic reaction, but they are generally considered to have little effect on clotting in vivo.
- Dextrans are considered more likely to cause clotting abnormalities than gelatins and associated allergic reactions have also been reported. Acute renal failure has also been reported with one type of dextrans solution (Dextran 40).
- High molecular weight HES solutions are most likely to cause multifactorial dose-dependent coagulopathy; at the time of writing tetrastarch is considered the safest HES solution in this respect. Allergic reactions occur very infrequently with HES solution use but they have been reported.

In 2013 there was a worldwide recall of HES products due to concerns in human medicine over a possible increase in the need for renal replacement therapy and possible mortality associated with these fluids.

Indications

Short-term treatment for hypovolaemia:

Following attempts at fluid resuscitation using replacement crystalloids alone if the latter has proved ineffective. Some crystalloid therapy is usually continued in these cases and colloid therapy is typically discontinued first.

From the outset in animals in which there are specific indications: 'capillary leak syndrome'; hypoproteinaemia/hypoalbuminaemia.

More long-term use:

It is not common for patients to remain on long-term colloid therapy. However potential indications include:

- To minimise interstitial oedema in the presence of low plasma colloid osmotic pressure due to hypoproteinaemia; (in chronic hypoproteinaemia, the osmotic pressure of the interstitial compartment will also fall, increasing the likelihood of oedema).
- Systemic vasculitis or 'capillary-leak syndrome' (e.g. SIRS, sepsis)

A typical rate for on-going use is 1 ml/kg/h and it is generally recommended not to exceed 24 ml/kg/day.

Refractometer effects

Synthetic colloid administration will affect plasma total solids (TS) and urine specific gravity (USG) when measured using a refractometer:

The total solute concentration of most synthetic colloids is approximately 40 g/l so following administration, TS trends towards 40 irrespective of the animal's serum total protein concentration and refractometry is no longer a surrogate of plasma colloid osmotic pressure (COP). Refractometric TS may be lower than the true plasma total protein concentration in an animal with a plasma total protein concentration of more than 40 g/l.

USG increases dramatically following colloid administration and is no longer an indicator of renal concentrating ability.

The fluid plan

There is no set protocol for fluid therapy in any given situation and often the specific approach is determined both by the fluids and equipment available, and by the experience and preferences of the clinician involved.

A number of questions should be answered when determining the fluid plan:

Is fluid therapy indicated? If so, what is/are the indication(s)?
Which fluid(s) is/are appropriate?
How should the fluid therapy be administered?
How much should be given and for how long?
Are there specific considerations for the patient in question?

It is also very important that adequate monitoring is performed both to ensure that fluid administration is successful and safe, and to assess the patient's response.

The two most common indications for parenteral fluid therapy are:

1) **To correct perfusion deficits in hypovolaemia**: is the patient hypovolaemic, and if so, is this mild, moderate or severe?

2) **To replace fluid deficits in dehydration**: is the patient dehydrated and if so what is the estimated percentage dehydration?

Correction of electrolyte and acid-base abnormalities are other albeit less common indications for parenteral fluid therapy.

Hypovolaemia

Aim: restore the effective circulating intravascular volume and thereby restore adequate tissue perfusion.

Basic approach:

Think in terms of bolus administration and repeated reassessment of perfusion.

The peripheral intravenous route is typically used; the intraosseous route is an invaluable alternative in some cases (e.g. small patient size, severely collapsed vasculature).

- Use one or more of the shortest but largest bore catheters possible to minimise resistance to fluid flow

Intravenous fluid boluses are administered until end-points suggestive of acceptable systemic perfusion are achieved

- Boluses are given via infusion pump, free flow or under pressure (pressure bag or manual squeezing) as appropriate

Performed over a short period of time – usually a few minutes to an hour but sometimes longer

May involve the use of both isotonic crystalloids and synthetic colloids (plus blood products or hypertonic saline if available)

Replacement isotonic crystalloids are the first choice in the majority of cases

Overall approach tailored to the requirements of each individual patient

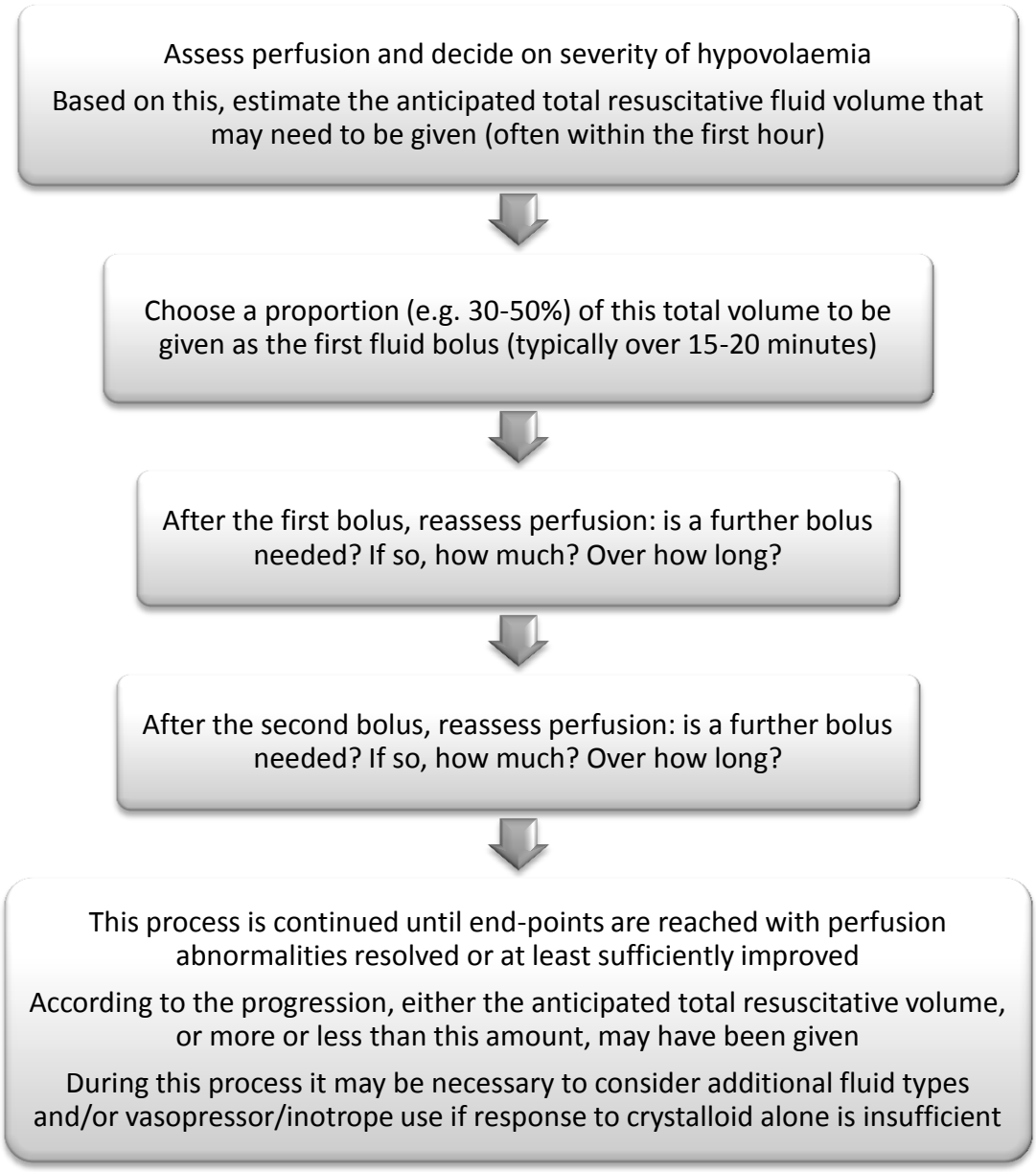
Dosing guidelines in hypovolaemia

Replacement isotonic crystalloids

	Size of first fluid bolus (mL/kg) (usually over 15-20 minutes)	Anticipated total resuscitative fluid volume (mL/kg) (Often within the first hour)		
Severity of hypovolaemia		Mild	Moderate	Severe
Dogs	**10-40**	**20-40**	**40-60**	**60-90**
Cats	**5-20**	**10-20**	**20-40**	**40-60**

Synthetic colloids

Dogs: 5-10 ml/kg boluses (traditionally up to a total of 20 ml/kg)
Cats: 2-5 ml/kg boluses (traditionally up to a total of 10 ml/kg)



If the patient's presenting history and subsequent assessment suggests concurrent dehydration, this must be taken into account with respect to on-going fluid therapy. Some clinicians subtract the volume of crystalloid administered during treatment of hypovolaemia from the calculated replacement deficit; others subject this to benign neglect on the basis that "the kidneys will work it all out in the end!".

Special considerations

The priority in treating hypovolaemic patients is to restore an effective circulating volume. Nevertheless there are some patient populations in which a less aggressive approach is warranted with smaller fluid boluses being used and/or fluid boluses being given over a longer period of time. Occasionally it is tolerated that a patient remains borderline hypovolaemic rather than risk fluid overload.

Scenarios warranting specific consideration include:

Recent or active haemorrhage
Lung pathology
Heart disease
Raised intracranial pressure
Chronic anaemia
Renal insufficiency

Recent or active haemorrhage

Much has been written about the 'best' approach to the resuscitation of haemorrhagic hypovolaemia. Most of this has been based on work done in human patients and in experimental animals. Essentially fluid therapy in haemorrhagic shock aims to:

- Restore intravascular volume
- Restore blood-oxygen carrying capacity
- Normalise coagulation status

Which fluid to use?

In terms of isotonic crystalloid versus synthetic colloid, there is no evidence-based consensus that one is 'better' than the other here and both have pros and cons. The majority of clinicians use a replacement isotonic crystalloid in the first instance and the potential for synthetic colloids to cause dose-dependent coagulopathy must not be overlooked in patients already bleeding.

Haemostatic resuscitation

As well as the potential for anaemia, bleeding patients have various reasons to be coagulopathic, both primarily, i.e. if a coagulopathy is the cause of bleeding in the first place and secondarily – for example due to inflammation from trauma; hypothermia ('lethal triad' of hypothermia, acidaemia and coagulopathy); clotting factor consumption; and dilutional coagulopathy from resuscitative fluid already administered. 'Haemostatic resuscitation' describes the aggressive early use of packed red blood cells and clotting products, a practice which is increasingly preferred in human patients as a way of restoring, or at least minimising impact on, oxygen-carrying capacity and coagulation status while improving intravascular volume. However lack of resources means that this approach is unlikely to be realistic in most veterinary environments in which avoiding both synthetic colloid use if possible and overzealous crystalloid administration may be sensible.

Hypotensive resuscitation

Haemorrhagic hypovolaemia is one example in which the concept of 'permissive hypotension' or 'hypotensive resuscitation' has been put forward as opposed to more standard 'normotensive resuscitation'. This involves titrating volume resuscitation to a sub-normal end-point that should nevertheless improve tissue oxygen delivery enough to maintain organ viability until such time as bleeding can be controlled. So for example in patients in which systemic arterial blood pressure can be reliably monitored, one may usually aim to resuscitate to a mean arterial pressure of 80 mmHg (or

a systolic blood pressure of 100 mmHg); in permissive hypotension the end-point might be MAP 65 mmHg (or systolic 80 mmHg).

By avoiding overzealous fluid resuscitation which targets normotension, the hope is to preserve the first and often best clot at any sites of bleeding. The need to control bleeding as soon as possible by either temporary (e.g. tourniquet, haemostatic agent) or permanent (i.e. surgical intervention) measures is important to stress although in some patients bleeding will be self-limiting. More aggressive fluid resuscitation is then used as required once haemorrhage is controlled.

In bleeding patients with head trauma, normotensive resuscitation would seem most rational to ensure adequate cerebral perfusion pressure and thereby try to minimise secondary brain injury; the importance of CPP in brain injury has a strong evidence base at least in human patients if not yet in clinical dogs and cats. This may also be true for spinal cord perfusion pressure in spinal cord injury but more evidence is needed.

Note that permissive hypotension is usually only considered in patients with on-going haemorrhage in which it is planned to intervene to control the bleeding and this technique is used to maintain organ viability in the meantime. This is a patient population that is encountered much less frequently in veterinary medicine than in human medicine so care is needed not to over-apply this resuscitative technique the evidence base for which is still scarce in humans, even more so in dogs and cats.

Lung pathology

The lungs have extensive capacity to cope with fluid but pathological lungs may be more prone to oedema than healthy lungs. This scenario is encountered most commonly in the form of pulmonary contusions following thoracic trauma. Animals suffering from severe trauma (thoracic or non-thoracic) or from other causes of shock may also develop acute lung injury (ALI) or acute respiratory distress syndrome (ARDS); this is especially true for cats.

In hypovolaemic animals with lung pathology that do not respond adequately to conservative crystalloid boluses, a synthetic colloid may prove effective. However in some types of lung pathology, the pulmonary vasculature can become leakier to colloid molecules despite their large size. Once the colloid molecules move into the pulmonary interstitium they may worsen pulmonary oedema and may persist there for some time. Nevertheless it is not possible to predict in advance whether this will occur and it is therefore recommended to assess the patient's response to a colloid bolus and discontinue this therapy if respiratory status worsens as a result.

Heart disease

Patients with heart disease requiring fluid therapy need to be treated extremely carefully. Administration of fluids will increase the circulating volume and the diseased heart may not be able to cope with the added load resulting in congestive heart failure. Hypovolaemia must still be treated but more conservatively than in an animal without heart disease, and fluid therapy should be discontinued as soon as possible.

The majority of dogs with clinically significant heart disease will have a murmur on auscultation. However failure to identify a murmur (or gallop sound) does not rule out clinically significant heart disease.

Thoracic radiography and especially echocardiography may help guide therapy by providing further information with respect to left atrial enlargement and therefore the likely ability of the patient to cope with fluid therapy.
Once hypovolaemia is corrected, fluid therapy is generally discontinued as soon as possible in animals with heart disease. In patients that require rehydration and maintenance fluid therapy,

strategies such as using a low rate (0.5-1 mL/kg/h) infusion of 0.45% (half strength) saline or subcutaneous fluid administration should be considered. These patients should be monitored very closely for signs of fluid overload.

Raised intracranial pressure

Causes of intracranial hypertension include:

- Head trauma
- Intracranial masses
- Severe seizures

Overzealous intravenous fluid therapy can lead to cerebral oedema and thereby exacerbate existing raised intracranial pressure. However the priority in protecting the brain from further secondary injury is to ensure that cerebral perfusion is adequate. Cerebral perfusion is highly dependent on systemic blood pressure so the approach here is to rapidly correct hypovolaemia but then discontinue aggressive fluid therapy as soon as possible. This is most often a concern in patients with both hypovolaemia and head trauma; hypertonic saline can prove very useful in such patients.

Chronic anaemia

Depending on the severity, animals with chronic anaemia may already be in a state of volume overload and are therefore at greater risk from aggressive fluid therapy. In addition fluids will cause haemodilution; although the same number of red blood cells is still circulating in the body, a lower PCV is likely.

Renal insufficiency

Fluid therapy is an extremely important consideration in patients with renal insufficiency. This is especially the case in animals with acute renal failure that frequently have either oliguria (reduced urine production) or polyuria (excess urine production). Patients with oliguric renal failure are vulnerable to fluid overload (as well as hyperkalaemia) while those with polyuria are susceptible to dehydration and hypokalaemia.

Monitoring of ‘ins and outs’ is very important in these cases, and intravenous fluid administration allows regular adjustment of ‘ins’ in response to ‘outs’. Animals with chronic renal insufficiency may be anaemic and in some cases this can be moderate-to-severe; these patients may therefore be in a state of volume overload. Although this is unlikely to be a significant consideration when subcutaneous or low rates of intravenous fluid therapy are administered, it may be of importance during aggressive volume resuscitation.

Dehydration

Aim: to replace deficits from all fluid compartments that are affected; this is mainly the interstitial and intracellular compartments in dehydration.

Basic approach:

- Corrected over a longer period of time than hypovolaemia – typically 12-48 hours depending on the severity and rate of onset
- Replacement isotonic crystalloids used alone

Rehydration is usually performed via the **intravenous** route. In some cases, **subcutaneous** fluid therapy may be a reasonable option.

Fluid requirement = Replacement volume + Maintenance requirement + On-going losses over chosen period of time

The initial fluid plan calculated for a dehydrated patient is an approximation and both the rate and the type of fluid used need to be reassessed. A range of different factors are taken into account that includes:

- Changes in physical examination parameters and especially in other measures of hydration – PCV/TS, body weight, urea/creatinine
- Whether the animal is eating/drinking (if allowed)
- A subjective assessment of the degree of on-going losses

Due attention must also be paid to the provision of supplementary potassium in particular in appropriate cases.

Replacement volume

Replacement volume (fluid deficit, mL) = % dehydration x body weight (kg) x 10

Percentage dehydration estimated on the basis of physical examination.

Maintenance requirement

Maintenance fluid requirements are related to metabolic rate which is a function of body surface area and varies with species and breed, and especially with age and size.

Formulae are available for calculating maintenance fluid requirements in dogs and cats. Depending on which formula is used, different figures may be obtained. Probably the most common recommendations are as follows:

- In animals weighing between 2-50 kg, use ([BW (Kg) x 30] + 70) mL/day
- In animals weighing < 2 Kg or > 50 Kg, use [$BW^{0.75}$ (Kg) x 70] mL/day

However, the fact that there are various different formulae perhaps illustrates that there is no definite consensus on which is the 'most correct'. An empirical approach with regular reassessment of the patient is therefore reasonable and practical.

Guidelines for maintenance requirements:

- Fully grown dogs: 1.5 mL/kg/h (larger dogs) up to 4 mL/kg/h (very small/toy breed dogs)
- Fully grown cats: 2-3 mL/kg/h
- Paediatric patients: 4-8 mL/kg/h; smaller and/or younger towards the upper end, larger and/or older towards the lower end.

Maintenance requirements for overweight animals should be calculated using a reduced body weight.

On-going losses

These are on-going losses such as vomitus and diarrhoea that occur over and above normal losses accounted for in maintenance requirements. It is practically very difficult, and often inherently inaccurate, to try and quantify these objectively and therefore one solution is to estimate the contribution from these additional on-going losses in terms of multiples of the patient's maintenance requirements. For example:

- A patient with no additional on-going fluid losses has no additional contribution to the initial fluid plan
- A patient with occasional vomiting daily may have half a maintenance requirement added to the initial fluid plan
- A patient with multiple episodes of vomiting daily may have an extra maintenance requirement added to the initial fluid plan
- A patient with multiple episodes of vomiting and profuse diarrhoea daily may have two extra maintenance requirements added to the initial fluid plan

These are very much guidelines only and on-going losses are re-estimated on a daily basis. Although this approach is somewhat subjective, as long as on-going losses and hydration parameters are reassessed daily and fluid rates are adjusted accordingly, this approach is both valid and clinically practical.

Example calculations

Example 1

A 3.5 kg adult female neutered domestic short-hair cat presents with a 2-day history of anorexia and lethargy of unknown cause. She is assessed as being 8% dehydrated, rehydration is to be performed over 24 hours, and there are no extra on-going losses evident initially.

Replacement volume (fluid deficit, ml) = % dehydration x body weight (kg) x 10
= 8% x 3.5 kg x 10
= 280 ml

Maintenance fluid rate = 2 ml/kg/h
Maintenance requirement over 24 hours = 2 ml x 3.5 kg x 24 h = 168 ml

No additional on-going losses beyond those accounted for in maintenance requirement.

Fluid requirement = Replacement volume + Maintenance requirement + On-going losses over chosen period of time

Fluid requirement = 280 ml + 168 ml + 0 ml = 448 ml over 24 hours
= approx 18.7 ml/h
= 5.3 ml/kg/h

Example 2

A 20.0 kg adult male neutered crossbreed dog presents with a 2-day history of anorexia, lethargy, multiple episodes of vomiting and passing watery bloody diarrhoea. He is assessed as being 10% dehydrated, rehydration is to be performed over 12 hours, and significant additional on-going losses are anticipated.

Replacement volume (fluid deficit, ml) = % dehydration x body weight (kg) x 10
= 10% x 20.0 kg x 10

= 2000 ml

Maintenance fluid rate = 2 ml/kg/h
Maintenance requirement over 12 hours = 2 ml x 20.0 kg x 12 h = 480 ml

Two extra maintenance requirements are added to account for the anticipated on-going losses.
On-going losses = 2 x 480 mL = 960 ml

Fluid requirement = Replacement volume + Maintenance requirement + On-going losses over chosen period of time

Fluid requirement = 2000 ml + 480 ml + 960 ml = 3440 ml over 12 hours
= approx 286.7 ml/h
= 14.3 ml/kg/h

Subcutaneous fluid therapy

Only isotonic crystalloid solutions should be used and the total volume is typically divided between several sites.
Depending on the species in question, a dose of 10-20 mL/kg is usually used and this route of administration is therefore most suitable for smaller dogs and cats and for exotics.

Examples in which this route may be appropriate:

- Mild dehydration
- Owners unable (or unwilling) to pay for intravenous fluid therapy
- Owners reluctant to leave their pets at the clinic
- Intermittent use to provide fluid support in chronic illnesses such as chronic renal failure.

The sole use of subcutaneous fluid therapy is inappropriate in patients with hypovolaemia as absorption is too slow due to poor peripheral perfusion.

Complications of skin sloughing, infection and pain are all potentially associated with repeated use of this route of fluid therapy. The fluid should be warmed to body temperature as cold fluids will cause pain and vasoconstriction of the skin vessels slowing down the rate of absorption.

Monitoring

Given the risk of volume overload with indiscriminate fluid administration, it is important to monitor animals receiving fluid therapy. The type and intensity of monitoring should be decided based on how susceptible each individual patient is thought to be to fluid overload.

It is increasingly being realised that more is not always better when it comes to fluid therapy, especially in certain populations where keeping the patient on the ‘dry’ side may be less harmful than excessive fluid administration. The relative pros and cons of keeping patients ‘wetter’ or ‘drier’ according to their disease states remains to be clarified in humans and even more so in dogs and cats.

There are two aspects to monitoring fluid therapy. The first is to monitor that the abnormalities for which the fluid is being administered are improving, i.e. **perfusion or hydration deficits are resolving**; rehydration is usually monitored using both clinical pathology parameters and body weight.

The second aspect is to monitor for **volume overload**, i.e. excessive fluid administration beyond that which the patient can excrete. Regular physical examination is the predominant method of monitoring for volume overload.

- Respiratory rate, effort and lung auscultation to detect pulmonary oedema
- Evidence of peripheral tissue oedema (e.g. chemosis, facial oedema, distal limb oedema)
- Serous nasal discharge

Urine output has a role in the monitoring of fluid therapy, with objective quantification indicated in specific patient populations.

Volume overload is treated by discontinuing or at least reducing the rate of fluid therapy and administering diuretic therapy if sufficiently concerned.

Central venous pressure

Central venous pressure (CVP) measurement requires an in-dwelling central venous catheter and is greatly facilitated by having a transducer and multi-parameter monitor; however it can be achieved using an improvised manometer. The pressure within the right atrium is measured and this is used as a proxy of the pressure in the central venous system from which is inferred the patient's intravascular volume status – it is essential to realise that CVP is a pressure not a volume and so for example a normovolaemic patient with cardiac dysfunction and low cardiac output (e.g. as may occur in some septic patients) can have a high CVP despite being volume deplete.

There is much debate about the usefulness of CVP and whether it is more helpful as a proxy of volume status or of volume responsiveness. The consensus in human medicine at this time appears to be that while there is some contested evidence that CVP is useful as a marker of fluid tolerance/safety (i.e. how likely you are to overdo it/overload the patient resulting in especially pulmonary oedema) its value is even more limited as a marker for fluid responsiveness (i.e. if and how well the patient may respond positively to a fluid challenge).

Central venous pressure monitoring is most robust using trends rather than single values, and may be especially helpful in cardiac patients. There are significant practical issues with central venous pressure monitoring and it not performed routinely outside of referral centres.

- Measurements from 0-5 cm H_20 are considered normal though it is the trend that is most important
- A rapid increase of 3-5 cm H_20 during fluid therapy suggests possible fluid overload and decreased fluid administration is indicated

Placement of a jugular catheter is not considered essential in the first phase of fluid resuscitation. Central venous catheter placement requires time and experience and fluid resuscitation should not be delayed in order to achieve this access.