Animals requiring fluids come in various shapes and sizes. All patients need to be thoroughly assessed to ensure they receive appropriate fluid therapy

Fluid therapy in small animals

1. Principles and patient assessment

ROBERT GOGGS, KAREN HUMM AND DEZ HUGHES

Robert Goggs graduated from Liverpool in 2004. He is currently a third-year resident in small animal emergency and critical care at the Royal Veterinary College (RVC).

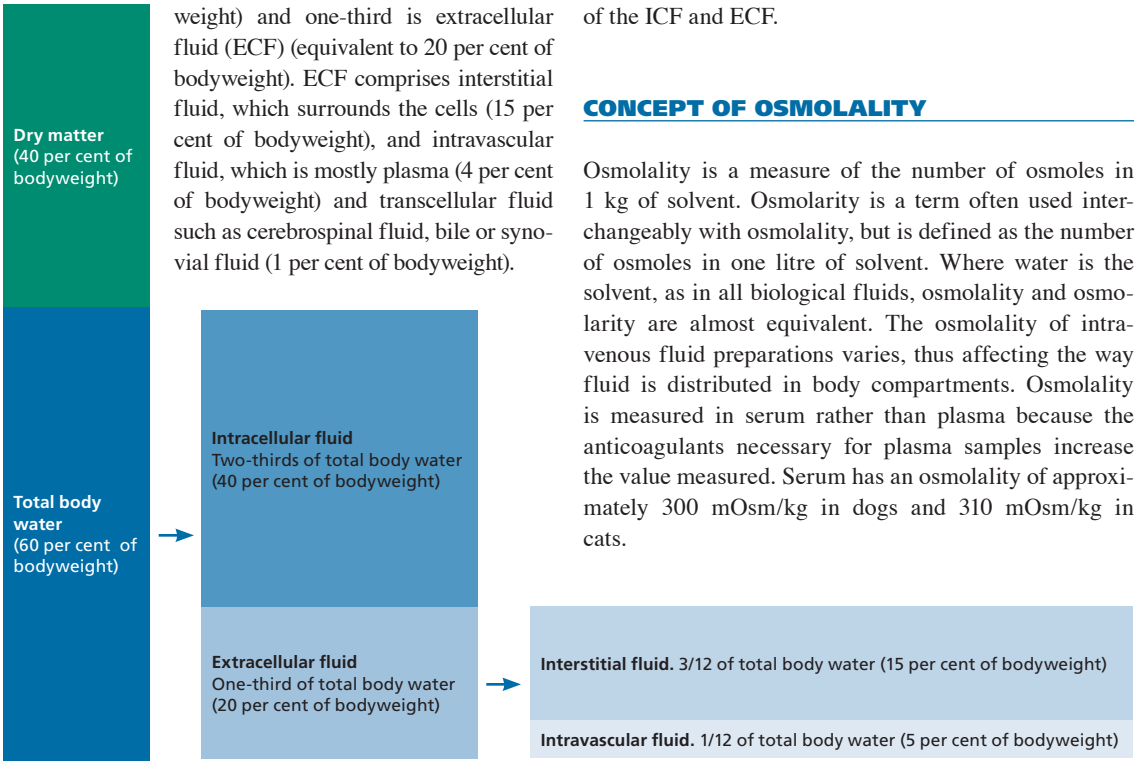
Karen Humm graduated from Cambridge in 2001. She is currently a third-year resident in small animal emergency and critical care at the RVC.

Dez Hughes graduated from Liverpool in 1990. He was a senior lecturer and director of the Emergency and Critical Care service at the RVC from 2001 to 2007.

THE administration of fluid therapy is commonly used in veterinary medicine to combat dehydration, hypovolaemia and hypoperfusion, to maintain intravascular volume and osmotic pressure, and to correct electrolyte imbalances. An understanding of the physiology of body fluids is important to ensure that the most appropriate fluid is chosen for a given situation. This article, the first in a series of three, describes the principles of fluid therapy and highlights the key aspects of patient assessment. Articles in the February and March issues of *In Practice* will discuss the individual properties of crystalloid and colloid solutions, respectively.

CONCEPT OF BODY COMPARTMENTS

In a healthy dog or cat, approximately 60 per cent of bodyweight is water. This total body water is distributed between different compartments (see diagram below). Approximately two-thirds of it is intracellular fluid (ICF) (equivalent to 40 per cent of bodyweight) and one-third is extracellular fluid (ECF) (equivalent to 20 per cent of bodyweight). ECF comprises interstitial fluid, which surrounds the cells (15 per cent of bodyweight), and intravascular fluid, which is mostly plasma (4 per cent of bodyweight) and transcellular fluid such as cerebrospinal fluid, bile or synovial fluid (1 per cent of bodyweight).



Distribution of total body water

Although the water in these areas is mobile, it remains distributed at these approximate levels due to the constancy of electrolyte and protein concentrations within the compartments. Thus, if the electrolyte and protein concentrations alter, a change in fluid distribution will result. In other words, the number of osmotically active particles in each space determines the volumes of the ICF and ECF.

CONCEPT OF OSMOLALITY

Osmolality is a measure of the number of osmoles in 1 kg of solvent. Osmolarity is a term often used interchangeably with osmolality, but is defined as the number of osmoles in one litre of solvent. Where water is the solvent, as in all biological fluids, osmolality and osmolarity are almost equivalent. The osmolality of intravenous fluid preparations varies, thus affecting the way fluid is distributed in body compartments. Osmolality is measured in serum rather than plasma because the anticoagulants necessary for plasma samples increase the value measured. Serum has an osmolality of approximately 300 mOsm/kg in dogs and 310 mOsm/kg in cats.

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An osmole is a mole ($6{\cdot}022 \times 10^{23}$ particles) of a non-dissociable substance. The osmolality of a fluid is not affected by the size, weight, chemical formula or valence of the molecule dissolved. Thus, one mole of glucose, albumin, Mg^{2+} or Cl^- dissolved in 1 kg of solvent would all generate one osmole, and each individual component would have an equal effect on plasma osmolality. However, one mole of NaCl in 1 kg of solvent will dissociate into one mole of Na^+ ions and one mole of Cl^- ions, generating a two osmolar solution. In vivo, the majority of serum osmolality is due to sodium, potassium, chloride, bicarbonate, urea and glucose. Larger molecules like albumin are present in much lower numbers and so have a lesser effect. Osmolality is measured by freezing point depression, whereby each osmole of solute dissolved depresses the freezing point by a known amount. An approximate osmolality can be calculated using the formula (where all values are in SI units):

$$\text{Osmolality (mOsm/kg)} \approx 2([Na^+] + [K^+]) + [\text{Glucose}] + [\text{Urea}]$$

The difference between the calculated and the measured osmolality is known as the osmolal gap and is due to osmotically active solutes such as albumin that are not accounted for by the formulae.

The situation in vivo is complicated by the concept of tonicity, which is the effective osmolality. Although some molecules such as urea are abundant in body fluids, they move across membranes freely and therefore do not generate an osmotic effect. Only molecules such as sodium and glucose, which are restricted in their movement by the presence of semipermeable membranes, can affect tonicity due to their osmolality.

Some fluids used in fluid therapy have a high osmolality. For example, 7·2 per cent NaCl (hypertonic saline) has an effective osmolality of 2462 mOsm/kg, which is far higher than that of serum. When infused into the intravascular space, the increased osmolality in this compartment causes an influx of water from other areas in order to equilibrate the osmolality across all compartments. Infusion of 7·2 per cent NaCl therefore results in a large but temporary increase in intravascular volume. Fluids such as Hartmann's solution and 0·9 per cent NaCl are often thought of as being equivalent to serum in osmolality, although in dogs they are slightly hypotonic (272 mOsm/kg) and hypertonic (308 mOsm/kg), respectively; in cats they are hypotonic and isotonic, respectively.

Osmotic pressure is defined as the potential pressure of a solution resulting from the osmoles dissolved in it – that is, the maximum pressure created by osmosis in a solution separated from another by a semipermeable membrane. Plasma tonicity (total effective osmotic pressure) should not be confused with colloid osmotic pressure (COP), which is also known as oncotic pressure and is the total osmotic pressure due to colloidal particles, particularly plasma proteins. COP represents only approximately 0·5 per cent of plasma tonicity, but is an important factor in transcapillary fluid dynamics (see later).

Hydrostatic pressure is another key component in fluid dynamics, and is defined as the pressure exerted by a fluid due to its weight. In biological systems, this refers to an intravascular, interstitial or ICF pressure against which osmosis must act in order for fluid shifts to occur.

EXTRACELLULAR FLUID HOMEOSTASIS

Capillaries are freely permeable to water and small solutes but relatively impermeable to macromolecules, particularly proteins. This results in a protein gradient between the intravascular and interstitial spaces, which acts to retain fluid within the vasculature. An opposing hydrostatic pressure gradient exists at the arteriolar end of the capillary bed.

The principles that govern fluid exchange were first described by Starling. The Starling-Landis equation derived from this work defines these relationships:

$$\text{Net filtration} = K_{fc}([P_{cap} - P_I] - \sigma_d[\Pi_{cap} - \Pi_I])$$

K_{fc} Filtration coefficient, P_{cap} Hydrostatic pressure of vasculature, P_I Hydrostatic pressure of interstitium, σ_d Capillary reflection coefficient, Π_{cap} Oncotic pressure of plasma, Π_I Oncotic pressure of interstitium

As such, Π_{cap}, which is equivalent to the COP, is the only component of this system that can be easily measured (using a colloid osmometer). It is also the one component that can be manipulated with fluid therapy.

At the arteriolar end, where hydrostatic pressure exceeds oncotic pressure, there is net fluid filtration – that is, fluid moves from the intravascular space to the interstitial space. At the venous end of the capillary bed, there is net but incomplete resorption of extravasated fluid from the interstitium back into the intravascular compartment. The remainder of the extravasated fluid is removed by the lymphatics.

The properties of the microvascular barrier vary with location, and hence the tendency for oedema formation varies accordingly. The interstitium has several defence mechanisms to limit fluid accumulation. The extravasation of fluid increases interstitial hydrostatic pressure and capillary oncotic pressure, while reducing the interstitial oncotic pressure. Fluid extravasation also increases the driving pressure for lymphatic drainage. Thus, the interstitium protects itself from oedema formation.

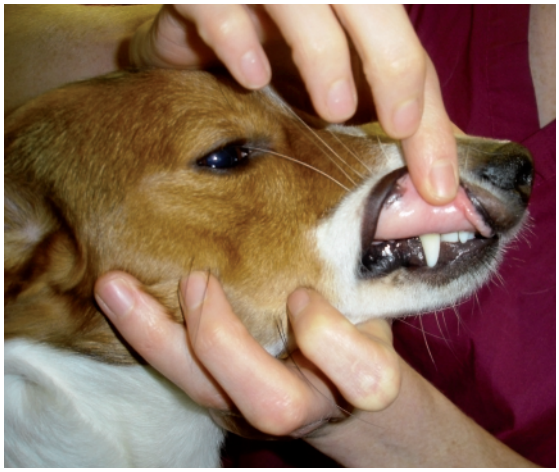
Oedema can result for a variety of reasons, and examining the situation for perturbations in the Starling-Landis relationship can elucidate most of these. Net filtration, and hence interstitial fluid formation, is favoured by:

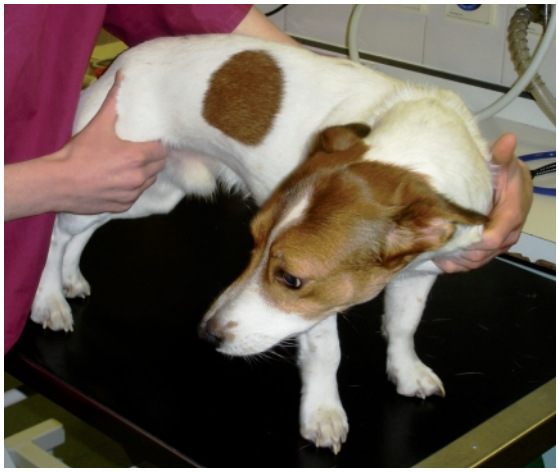
- Increased capillary permeability, capillary hydrostatic pressure or interstitial oncotic pressure;
- Decreased interstitial hydrostatic pressure or capillary oncotic pressure.

This relationship makes it possible to conceptualise the effects of hypoproteinaemia, venous congestion and capillary leakage on oedema formation. Recent research has highlighted the importance of the interstitium in oedema formation and suggests that in disease states such as burns, the disruption of certain interstitial cell-matrix binding mechanisms contributes to oedema formation (Wiig and others 2003). When evaluating fluid requirements of patients with oedema or body cavity effusions, consideration of the likely cause for the increased filtration or reduced resorption of interstitial fluid may assist in fluid therapy selection.

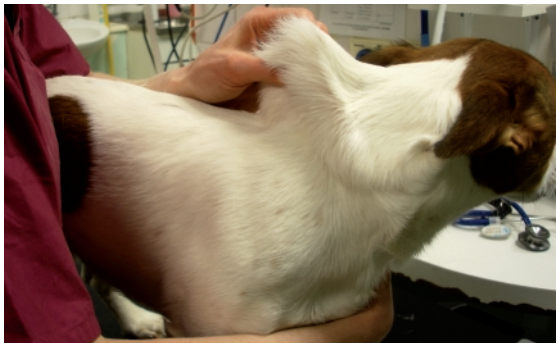
PATIENT ASSESSMENT

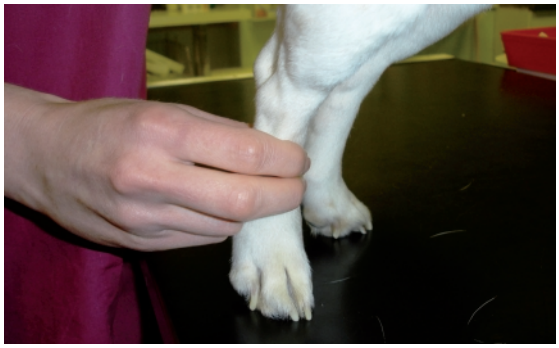
It is essential that a good physical examination is performed before fluid administration so that the correct fluid type is administered at an appropriate rate. In an emergency or critical care setting, the physical examination should





Assessment of (above left) mucous membrane colour and capillary refill time, (above right) femoral pulse quality, (below left) skin tent and (below right) metatarsal pulses



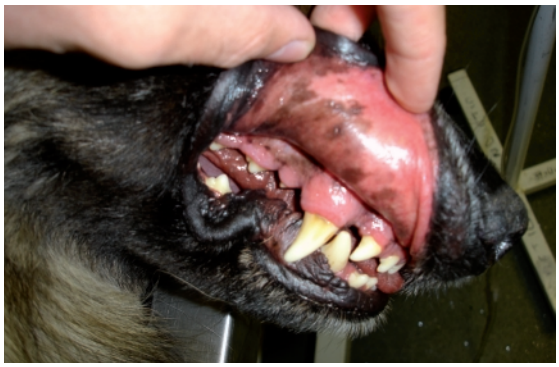


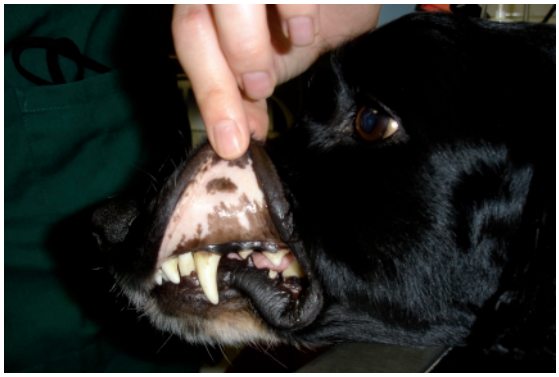
initially focus on the cardiovascular, respiratory and neurological systems. The patient's volume status, hydration status and any intercurrent disease processes should be assessed. Whether an animal develops hypovolaemia or dehydration in response to fluid loss depends on the rapidity of the loss in conjunction with the volume lost and the body compartment(s) from which the fluid originated.

Hypovolaemia

Hypovolaemia is defined as a reduction in intravascular volume. Findings of the physical examination of patients with hypovolaemia depend on the volume lost and the chronicity of that loss. In general, hypovolaemic patients present with tachycardia, abnormal pulse quality, and altered mucous membrane colour and capillary refill time (see table below). Alterations in mental status, cool extremities and tachypnoea may also be present. See Boag and Hughes (2005) for further information on the assessment of volume status.

Appropriate treatment for patients with hypovolaemia is immediate volume resuscitation with boluses of crystalloid or colloid fluids. It is important to differentiate hypovolaemia from cardiogenic shock, which may result in similar clinical signs but is due to pump failure rather than inadequate intravascular volume. Aggressive fluid therapy is contraindicated for cardiogenic shock and is likely to cause deterioration in the patient's condition. The patient's medical history, signalment and





(above) Congested mucous membranes in a dog with distributive shock secondary to septic peritonitis. (below) Dog with mucous membrane pallor, which may be due to haemorrhagic or hypovolaemic shock

GUIDELINES FOR THE ASSESSMENT OF UNCOMPLICATED HYPOVOLAEMIA IN DOGS

Clinical sign	*Mild (compensatory)*	*Moderate*	*Severe (decompensatory)*
Heart rate	130-150 bpm	150-170 bpm	170-220 bpm
Mucous membrane colour	Normal to pinker than normal	Pale pink	White, grey or muddy
Capillary refill	Vigorous, <1 second	Reduced vigour, 2 seconds	>2 seconds or absent
Pulse amplitude	Increased	Moderate decrease	Severe decrease
Pulse duration	Mild decrease	Moderate decrease	Severe decrease
Metatarsal pulse	Easily palpable	Just palpable	Absent
Plasma lactate concentration	3-5 mmol/litre	5-8 mmol/litre	>8 mmol/litre

Differentiating between hypovolaemia and dehydration

A common cause of hypovolaemia is blood loss due to, for example, a ruptured splenic mass. This loss is acute, large and solely from the intravascular compartment and, therefore, results in hypovolaemia. Distributive shock may also present with signs consistent with hypovolaemia, but the poor circulating volume in these patients is mainly due to a vasodilatory process that has increased the venous capacitance and reduced the proportion of vascular volume in the arterial vessels. These patients may additionally have losses from the intravascular space due to extravasation of fluid. Systemic inflammatory response syndrome (SIRS) occurring secondarily to sepsis, pancreatitis and tissue trauma are typical causes of distributive shock. Animals with SIRS also benefit from fluid therapy aimed at restoring intravascular volume. In contrast, a common cause of dehydration is chronic diarrhoea. Losses due to diarrhoea are hypotonic as a result of the excessive loss of water relative to solutes. When hypotonic losses occur over a longer period of time, the result is a sharing of the fluid loss between all body compartments.

HYPOVOLAEMIA VERSUS DEHYDRATION

	Mild to moderate dehydration	*Acute hypovolaemia*
Intravascular volume	↓	↓↓↓
Interstitial volume	↓	↓/No change
Intracellular volume	↓	No change
Heart rate	No change	↑↑↑
Capillary refill time	No change	↑ Progressing to ↓
Skin turgor	↑↑	No change
Total solids/packed cell volume	↑	No change/↓total solids
Urine output	↓	↓
Pulse quality	No change	Hyperdynamic pulses (obvious but short) progressing to hypodynamic (weak and short)

clinical findings (eg, heart murmur or auscultation of crackles over the lung fields) may offer potential corroboratory findings that shock is cardiogenic in origin. It should be noted that, in some patients (eg, Dobermanns), cardiogenic shock may be present without an audible heart murmur or abnormal lung sounds.

Although initial clinical examination is the first step in detecting hypovolaemia, further diagnostic investigation involving, for example, blood lactate analysis, thoracic radiography, electro- or echocardiography or abdominal ultrasound may be necessary to identify the underlying cause.

Dehydration

Dehydration strictly refers to loss of pure water, although it is often used to describe the loss of iso- or hypotonic fluid from the body. Accurate evaluation of dehydration is difficult, but can be estimated by assessing the mucous membranes (tackiness, colour and capillary refill time) and skin turgor (see table, above right). Packed cell volume in conjunction with total solids and urine specific gravity may also help to estimate the level of dehydration.

History is useful to identify potential causes and to corroborate the findings of the physical examination. If a recent healthy weight is available for comparison, this may permit calculation of weight loss. In a euvolaemic but dehydrated patient, this lost weight is equivalent to the volume of water lost.

Regardless of the methodology employed to estimate the hydration status of an animal, it is easy to be misled. Emaciated and geriatric patients are particularly challenging as they have poor skin elasticity, which can mimic dehydration. It is commonly reported that 5 per cent dehydration is not detectable, while 12 to 15 per cent dehydration leads to a completely moribund animal and imminent death. It has been shown that the response to dehydration varies markedly between dogs and it is therefore difficult to assign an accurate value. To date, similar studies have not been performed in cats.

In emaciated or geriatric patients, when compatible history and physical examination findings are present, it is reasonable to assume 7 per cent dehydration and treat accordingly. Regular reassessment followed by adjustment of therapy is required to safely rehydrate the patient.

CLINICAL SIGNS OF DEHYDRATION

Level of dehydration	*Clinical signs*
<5 per cent	Not detectable
5-6 per cent	Subtle loss of skin elasticity
6-10 per cent	Definite delay in return of skin to normal position Eyes possibly sunken in orbits Possibly dry mucous membranes
10-12 per cent	Tented skin stands in place Eyes sunken in orbits Dry mucous membranes
12-15 per cent	As for 10-12 per cent, plus possible signs of shock (eg, tachycardia, cool extremities, rapid and weak pulses, prolonged capillary refill time)

Complications and contraindications

While fluid therapy is commonplace, it is not benign and can have adverse effects. Patients should be thoroughly examined for any intercurrent disease that may lead to difficulties in accommodating a fluid load. Particular attention should be paid to the cardiovascular and renal systems. Cardiac disease reduces a patient's ability to cope with acute fluid loads and may lead to decompensation and congestive failure. Fluid therapy should therefore be cautious and judicious in animals with pre-existing cardiac disease. Attempts should be made to identify patients with compensated heart failure before constructing a fluid therapy strategy, as fluid administration can lead to decompensation, resulting in increased morbidity and possibly death.

Animals with acute (oliguric) renal failure have a reduced capacity to excrete excess fluid, which potentially results in volume overload, a complication frequently encountered in patients on fluid rates designed to induce diuresis. Cases with chronic renal insufficiency are common. Extra care should be taken with such patients as they are less able to correct existing electrolyte abnormalities or those created by poor fluid therapy choices.

SUMMARY

Administration of intravenous fluids is an extremely versatile therapeutic modality. It is vital that the practitioner has a sound understanding of the concepts of body compartments, osmolality and ECF homeostasis in order to identify the cause of any particular fluid imbalance and choose the most appropriate remedy. Effective and safe fluid administration depends on comprehensive patient assessment, particularly the ability to distinguish between hypovolaemia and dehydration.

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Notes