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FLUID THERAPY PHYSIOLOGY

Fluid Therapy Physiology: Why It Works (PDF)

Why it works — the physiology behind every fluid plan: body-water compartments, osmosis and Starling's forces, and how crystalloids, hypertonic saline and colloids distribute. The full companion to the online article at vet-ebooks.com.

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IN SHORT

Intravenous **fluid therapy** is applied physiology. Once you understand where the body keeps its water — and the two forces (**osmosis** and **Starling's hydrostatic/oncotic pressures**) that move it — the whole subject falls into place: why an isotonic crystalloid spreads through the extracellular space, why hypertonic saline pulls water out of cells, why a colloid stays in the vessel, and why the **wrong** fluid can do harm. This guide walks that physiology figure by figure — then turns it into the numbers you use at the cage side: the **shock bolus, the dehydration-deficit formula and the maintenance rate** for dogs and cats.

KEY POINTS

1. About **60% of body weight is water**: two-thirds sits **inside cells (ICF)**, one-third **outside (ECF)**; of the ECF, roughly three-quarters is interstitial and one-quarter is plasma.
2. Water moves by **osmosis** — it follows solute. In the ECF the dominant solute is **sodium**, so "where sodium goes, water follows."
3. At the capillary, **Starling's forces** (hydrostatic pressure pushing out, colloid oncotic pressure pulling in) decide the net leak between plasma and interstitium.
4. An **isotonic crystalloid** distributes through the whole ECF, so only about a quarter stays in the vessel; **hypertonic saline** draws water out of cells; a **colloid** holds water in the vessel.
5. **Dehydration** (an interstitial/intracellular water deficit) is not the same as **hypovolaemia** (an intravascular volume deficit) — and the two are corrected differently.
6. Because fluids redistribute, the **wrong choice or rate** causes real harm: volume overload, dilution, or cerebral injury from correcting sodium too fast.

About the images

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01 Why fluid therapy is applied physiology

Intravenous fluids are the most-used "drug" in veterinary practice, yet they are the one we most often give by habit. The difference between a fluid plan that saves a patient and one that harms it is almost always **physiology**: knowing where the body stores water, what holds it there, and what happens to each type of fluid *after* it drips into a vein. Master a handful of principles — compartments, osmosis, Starling's forces

and the behaviour of sodium — and every clinical decision about **fluid therapy in dogs and cats** becomes something you can reason out rather than memorise — from choosing the fluid to calculating the rate.

02 The body is mostly water — and it lives in compartments

Roughly **60% of an adult animal's body weight is water** (higher in neonates, lower in obese or geriatric patients, because fat holds little water). That water is not one pool but several, separated by cell membranes and the capillary wall. About **two-thirds is intracellular (ICF)** and one-third is **extracellular (ECF)**; the ECF is split again into the **interstitial fluid** (around the cells, ~3/4 of the ECF) and the much smaller **intravascular** or plasma volume (~1/4 of the ECF, only about 8–10% of total body water). This matters enormously, because an intravenous fluid is delivered into that small intravascular slice — and where it travels from there depends entirely on its composition.

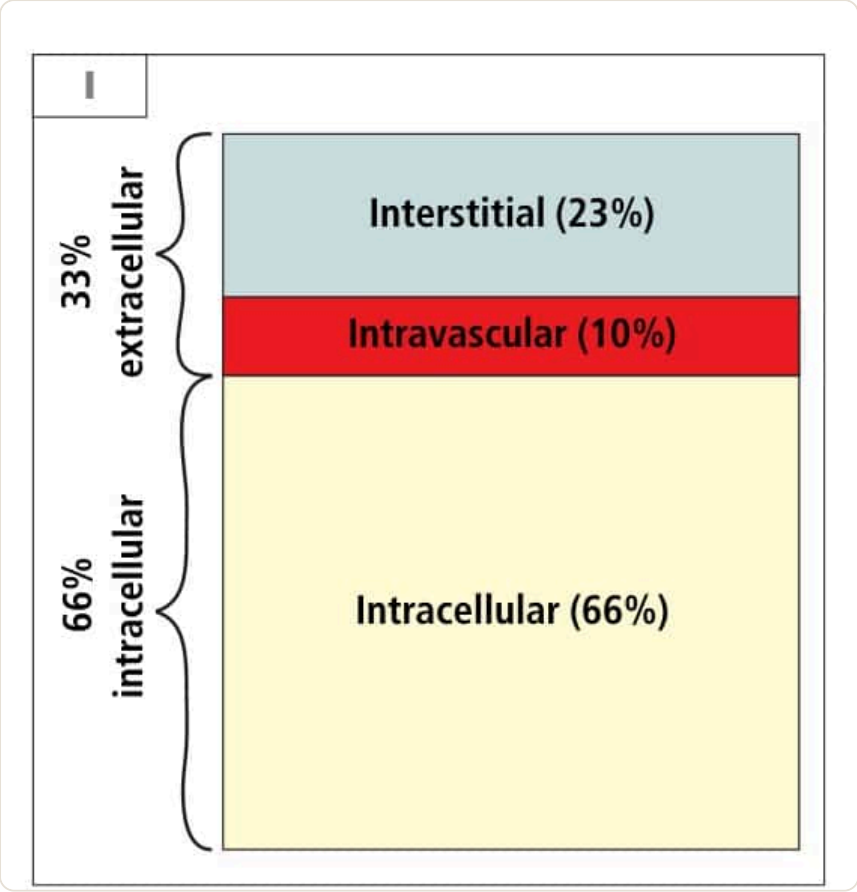

Fig 1 — The body-water compartments. About 60% of body weight is water: two-thirds intracellular, one-third extracellular — and the extracellular fluid is mostly interstitial, with only ~10% in the vessels.

03 Osmosis: water follows solute

Water crosses cell membranes freely; most solutes do not. When a membrane separates two solutions of different solute concentration, water moves — by **osmosis** — toward the side with *more* solute, until the concentrations equalise. The pull a solution exerts is its **osmolality**; the part of it due to solutes that *cannot* cross the membrane ("effective osmoles") is its **tonicity**, and tonicity is what actually shifts water between compartments. In the ECF the overwhelmingly dominant effective osmole is **sodium**: raise or lower plasma sodium and you drive water out of, or into, the cells. This single idea — water follows solute, and in the ECF that solute is sodium — explains most of what fluids do.

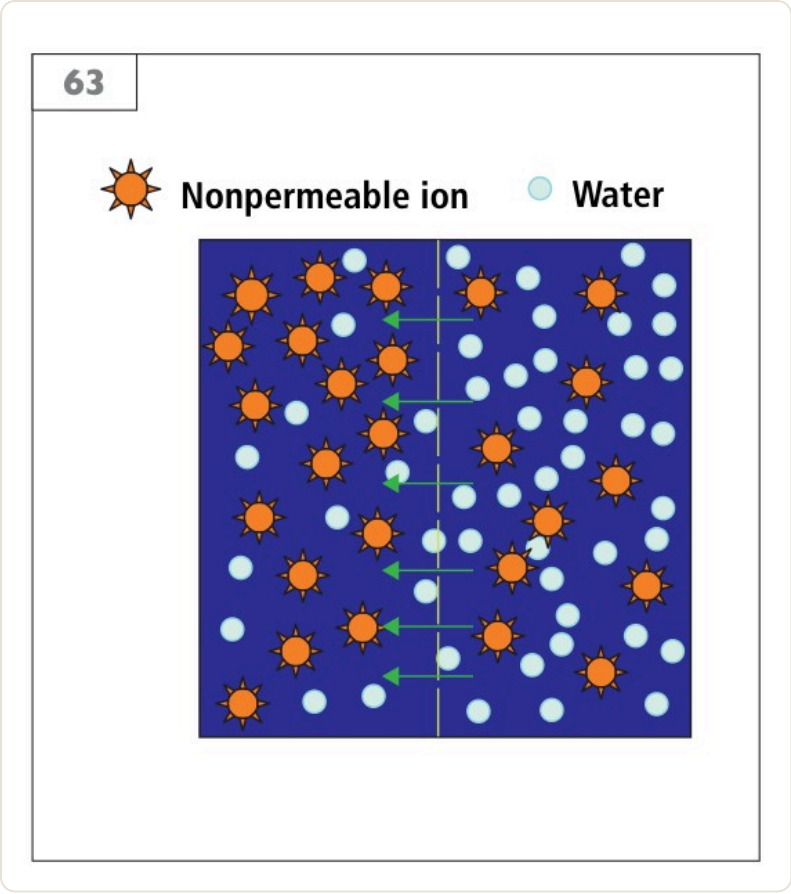

Fig 2 — Osmosis across a semipermeable membrane. Non-permeable particles (orange) cannot cross, so water (pale) moves toward the more concentrated side until it is balanced — the engine behind every fluid shift.

04 Starling's forces at the capillary

Between plasma and the interstitium the barrier is the capillary wall, and here water is governed not by osmosis alone but by **Starling's forces**: the **hydrostatic pressure** inside the capillary (P_C) pushes fluid *out*, while the **colloid oncotic pressure** of plasma proteins (π_C, mostly albumin) pulls fluid *back in*. The interstitium has its own, smaller, opposing pressures. The net balance normally favours a slight outward leak, which the lymphatics return. Fluid therapy tips this balance: infusing crystalloid raises P_C and dilutes π_C, pushing water into the interstitium (oedema), whereas a colloid raises π_C and holds water in the vessel. Understanding this equation is what lets you predict oedema before it happens.

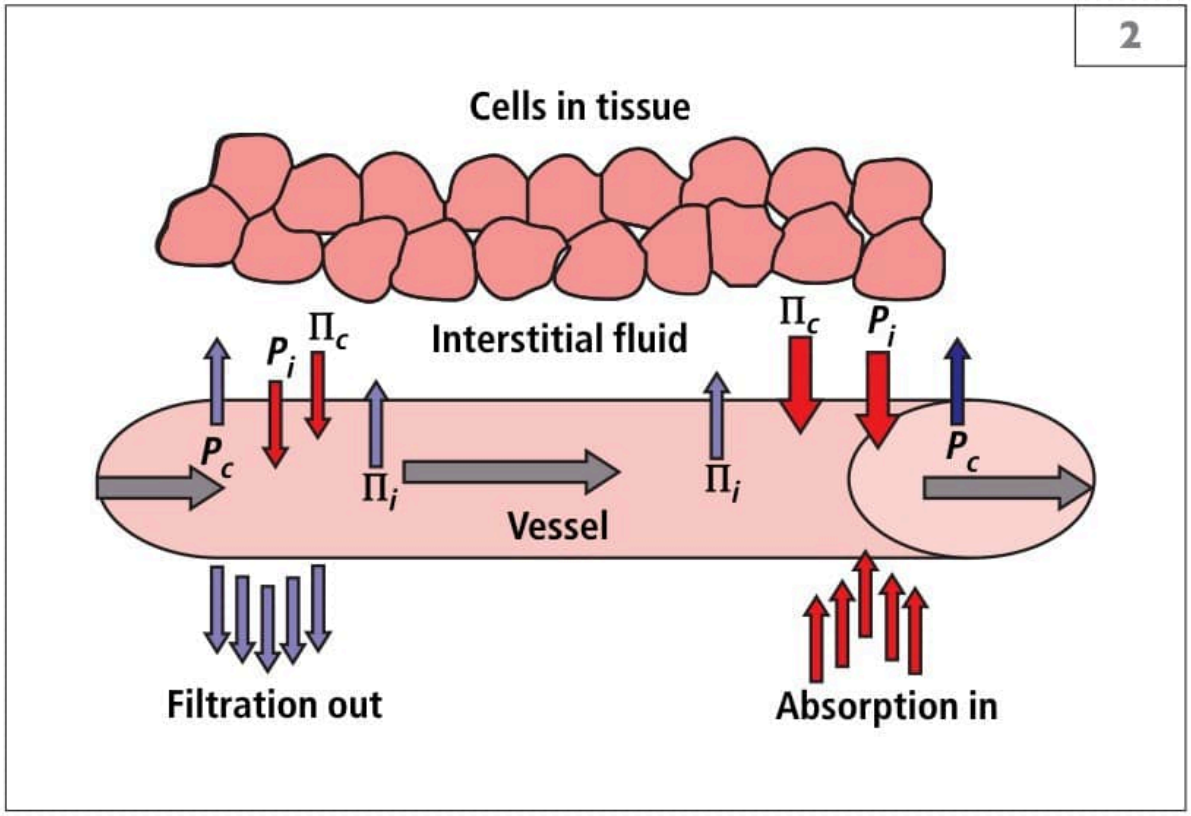

Fig 3 — Starling's forces at the capillary. Hydrostatic pressure (P) pushes fluid out of the vessel; the oncotic pull of plasma protein (π) draws it back — their balance sets filtration out versus absorption in.

05 Dehydration is not hypovolaemia

Two words are used loosely at the cage side but mean different things. **Dehydration** is a deficit of *total body water* — mostly interstitial and intracellular — and it builds over hours to days (vomiting, diarrhoea, not drinking). You detect it by **parameters of hydration**: skin-tent, sunken eyes, tacky membranes, weight loss. **Hypovolaemia** is a deficit of the *intravascular* volume — the blood the heart can pump — and it threatens perfusion *now* (haemorrhage, severe fluid loss, shock). You detect it by **parameters of perfusion**: heart rate, pulse quality, capillary refill time, mucous-membrane colour, blood pressure. The distinction is clinical dynamite: hypovolaemia needs rapid intravascular refill (boluses), whereas dehydration is replaced steadily over many hours.

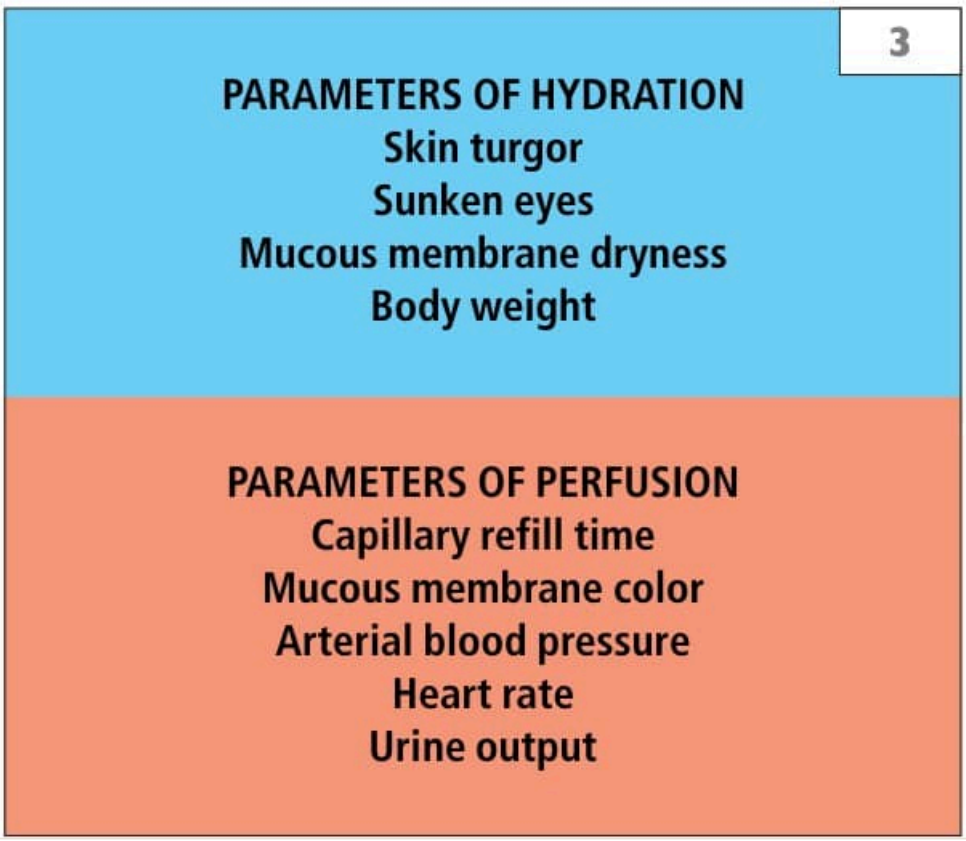

Fig 4 — Hydration vs perfusion. Hydration parameters (skin turgor, eyes, membranes, weight) reveal an interstitial water deficit; perfusion parameters (refill time, pulse, blood pressure) reveal an intravascular one.

06 How the body defends its blood volume

The body guards its circulating volume fiercely, because perfusion cannot wait. When intravascular volume falls, **baroreceptors** in the aortic arch and carotid sinus sense the drop and trigger a compensatory cascade: sympathetic tone rises, releasing adrenaline and noradrenaline, which raise heart rate and contractility and cause **vasoconstriction** — all to hold up cardiac output and blood pressure. In parallel, the kidney's renin–angiotensin–aldosterone system and ADH retain sodium and water. These reflexes are why a patient can look deceptively stable in early (compensated) shock — and why, once they fail, decompensation is abrupt. Reading them correctly tells you how hard, and how fast, to resuscitate.

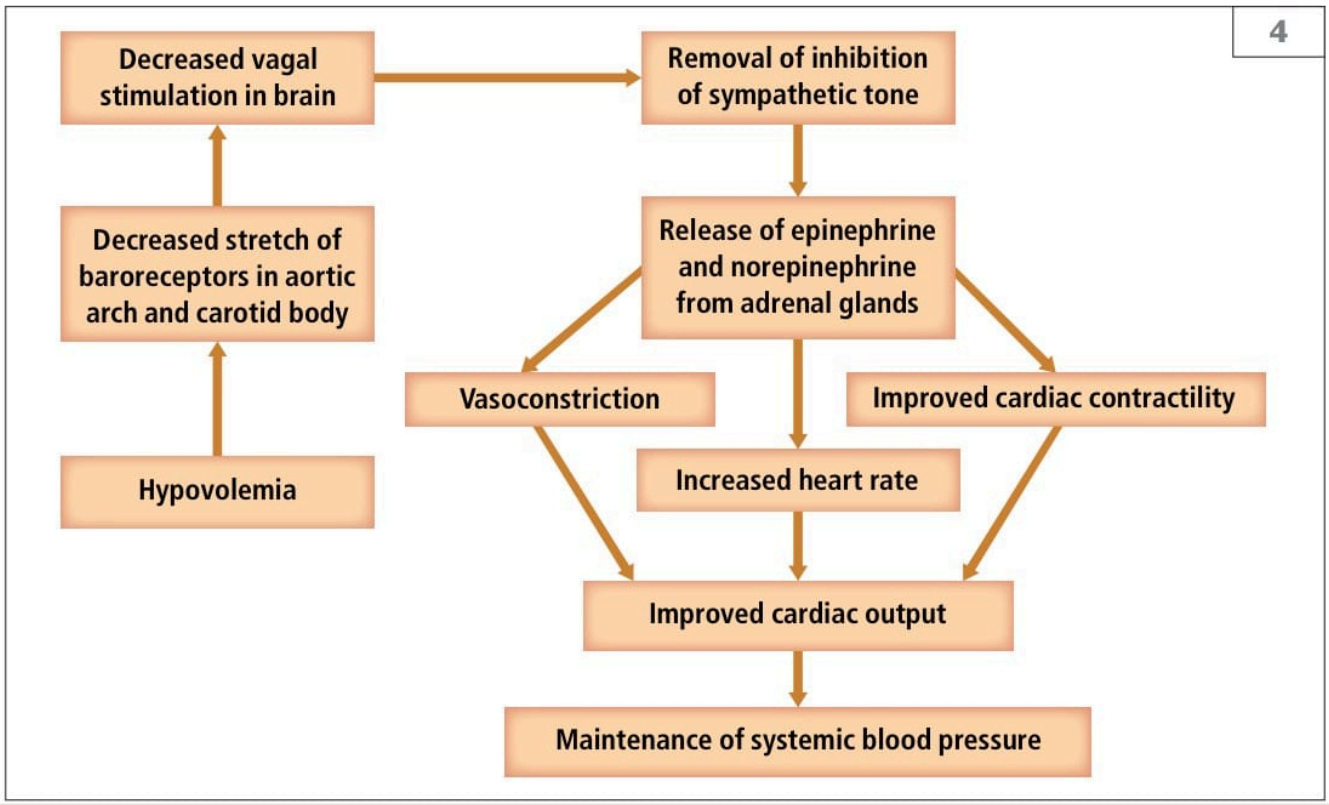

Fig 5 — The baroreceptor response to hypovolaemia. Falling volume unloads the baroreceptors, driving sympathetic output — vasoconstriction, higher heart rate and contractility — to defend cardiac output and blood pressure.

07 Crystalloids: what's in the bag, and where it goes

A **crystalloid** is water with small, membrane-crossing solutes — sodium, chloride, a buffer (lactate, acetate or gluconate) and sometimes potassium, calcium or dextrose. Because these solutes distribute across the whole ECF, an **isotonic** crystalloid (0.9% saline, lactated Ringer's — **Hartmann's** in much of the world — or Plasma-Lyte) follows suit: within an hour or so, only about **a quarter of the volume remains in the vessel** and the rest has moved into the interstitium. That is exactly why crystalloids are excellent for replacing interstitial dehydration but comparatively inefficient — and short-lived — for filling the vascular space in shock, where several times the "lost" volume may be needed. The choice between fluids comes down to matching their sodium and buffer content to the patient's problem.

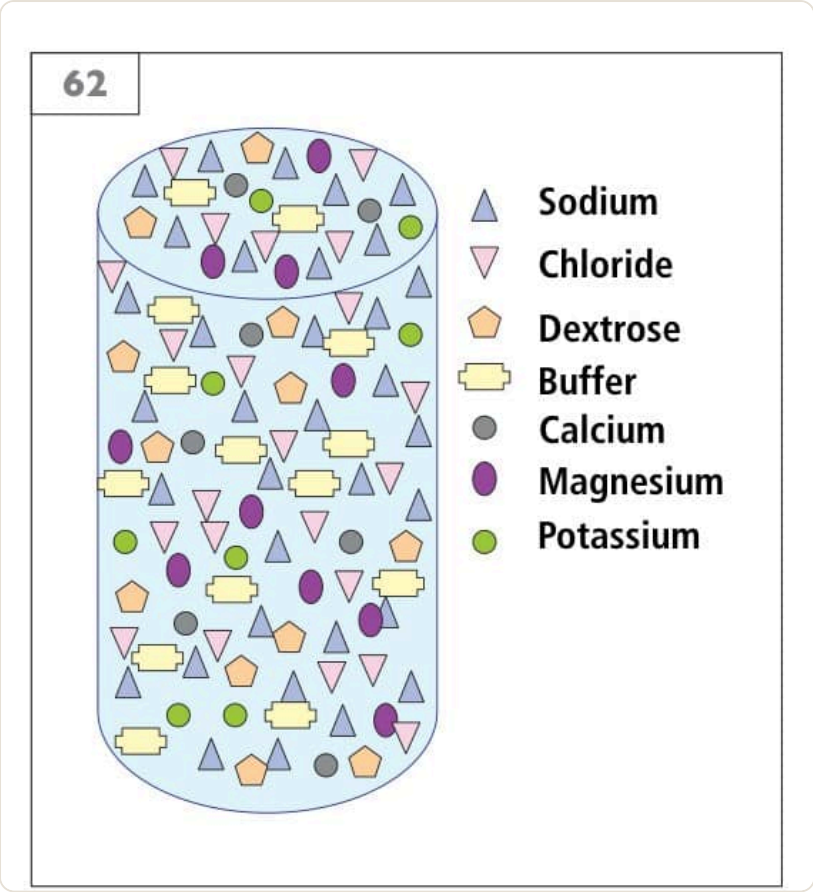

Fig 6 — What a crystalloid contains. A crystalloid is water carrying sodium, chloride and a buffer (± potassium, calcium, magnesium or dextrose); because these cross the capillary freely, the fluid spreads through the whole extracellular space.

08 Hypertonic saline: borrowing water from the cells

Push the sodium concentration far above plasma — **hypertonic saline** (e.g. 7.5%) — and the physics reverse. The infused bolus makes the intravascular space transiently *hyperosmolar*, so by osmosis water is drawn first out of the interstitium and then out of the cells into the vessel. A small volume therefore expands the circulation rapidly, which is its value in shock and in reducing brain swelling. But the effect is **borrowed and brief**: once osmolality re-equilibrates the water leaves again, so hypertonic saline is followed by an isotonic crystalloid (and often a colloid) to hold the gain. It is contraindicated when the patient is already dehydrated — there is no cellular water to spare.

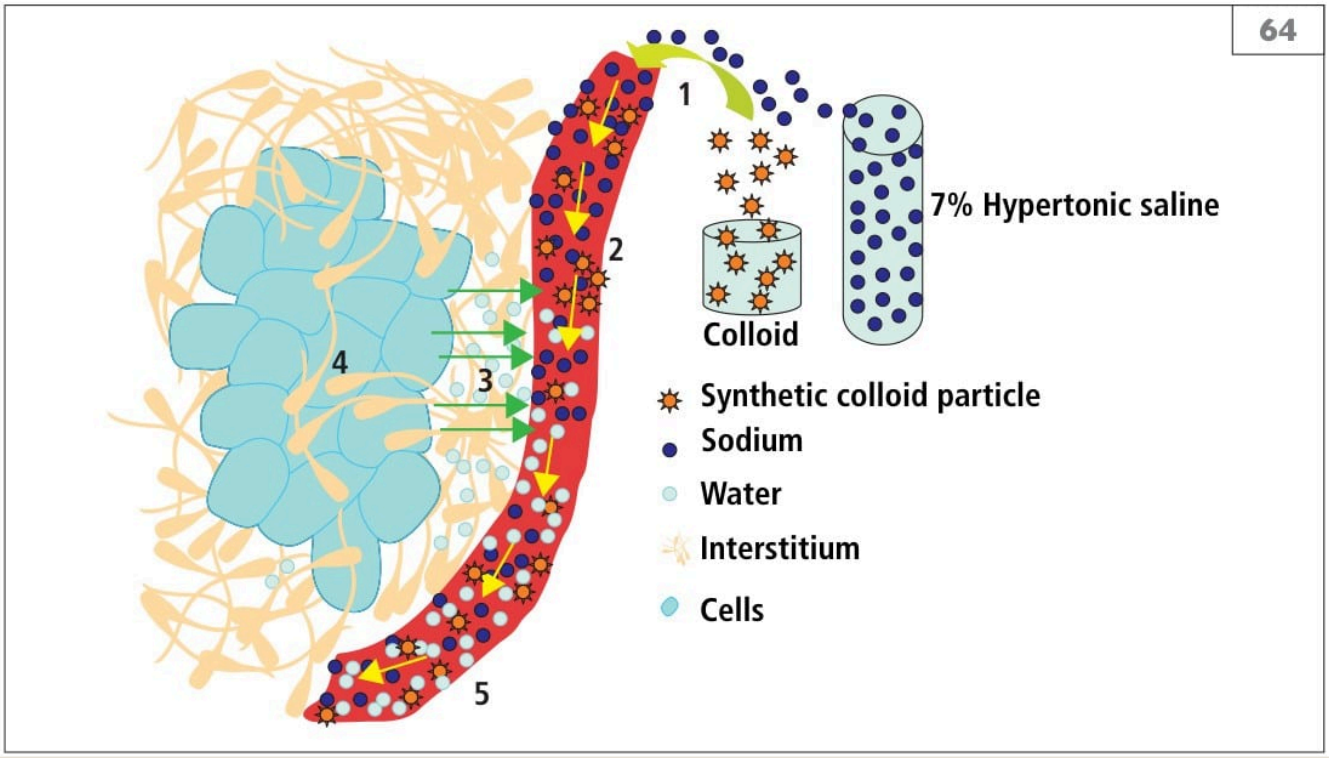

Fig 7 — How hypertonic saline works. A hypertonic bolus makes the vessel briefly hyperosmolar, so water is pulled from the interstitium (step 3) and the cells (step 4) into the circulation — a small volume, a fast but temporary expansion.

09 Colloids: holding water inside the vessel

A **colloid** carries large molecules — natural albumin or a synthetic starch — that are too big to cross the healthy capillary wall. By staying in the plasma they raise its **colloid oncotic pressure**, and (because those charged molecules draw sodium, and water follows sodium) they hold water within the vascular space far longer than a crystalloid does. That makes colloids efficient volume expanders when the intravascular compartment must be filled and kept full. The caveat is the capillary: if it is leaky (sepsis, SIRS), the colloid — and its retained water — escapes into the interstitium, so the benefit is lost and interstitial oedema can worsen.

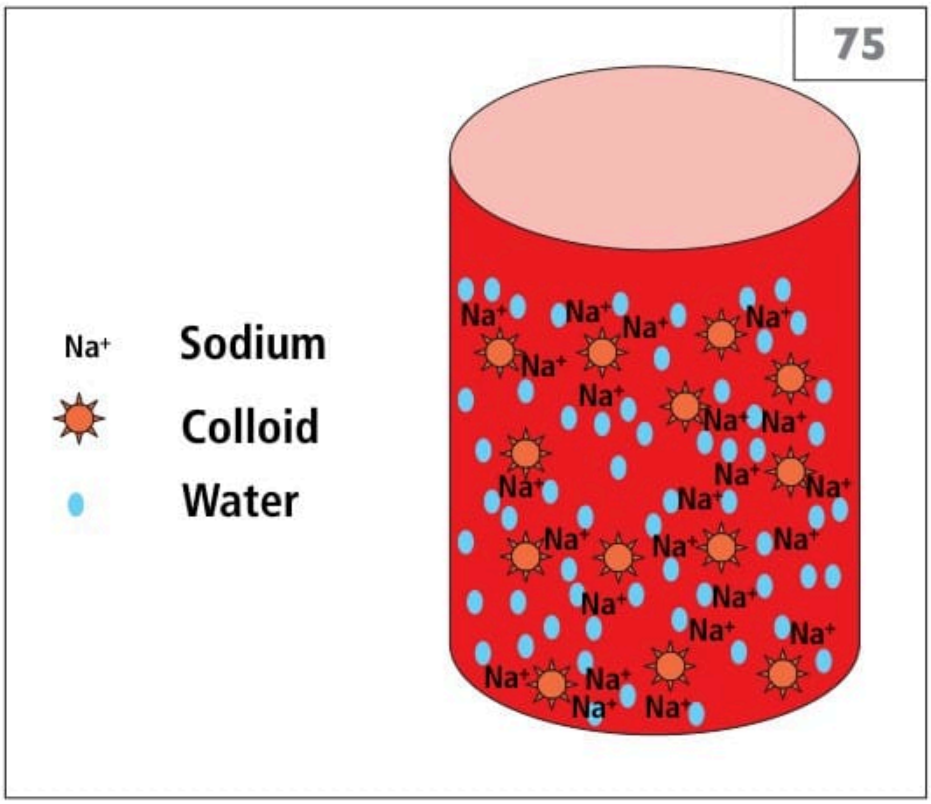

Fig 8 — How a colloid holds water in. Large colloid particles cannot leave the vessel; they attract sodium, and water follows — so the fluid stays intravascular and expands the circulation for longer than a crystalloid.

10 Fluid balance: ins, outs and ongoing losses

A fluid plan is a running balance sheet of what goes in against what is lost. Losses come in two kinds. **Sensible losses** can be measured — urine output above all, plus vomiting, diarrhoea (diarrhea) and wound or drain fluid. **Insensible losses** cannot be measured directly — the water lost through respiration and metabolism (and, in species that sweat, sweat) — and are estimated at roughly **20–30 mL/kg/day**. A complete plan replaces the existing *deficit*, meets the ongoing *maintenance* need, and keeps pace with any *continuing* losses; ignore the last and a patient that should be improving quietly falls behind.

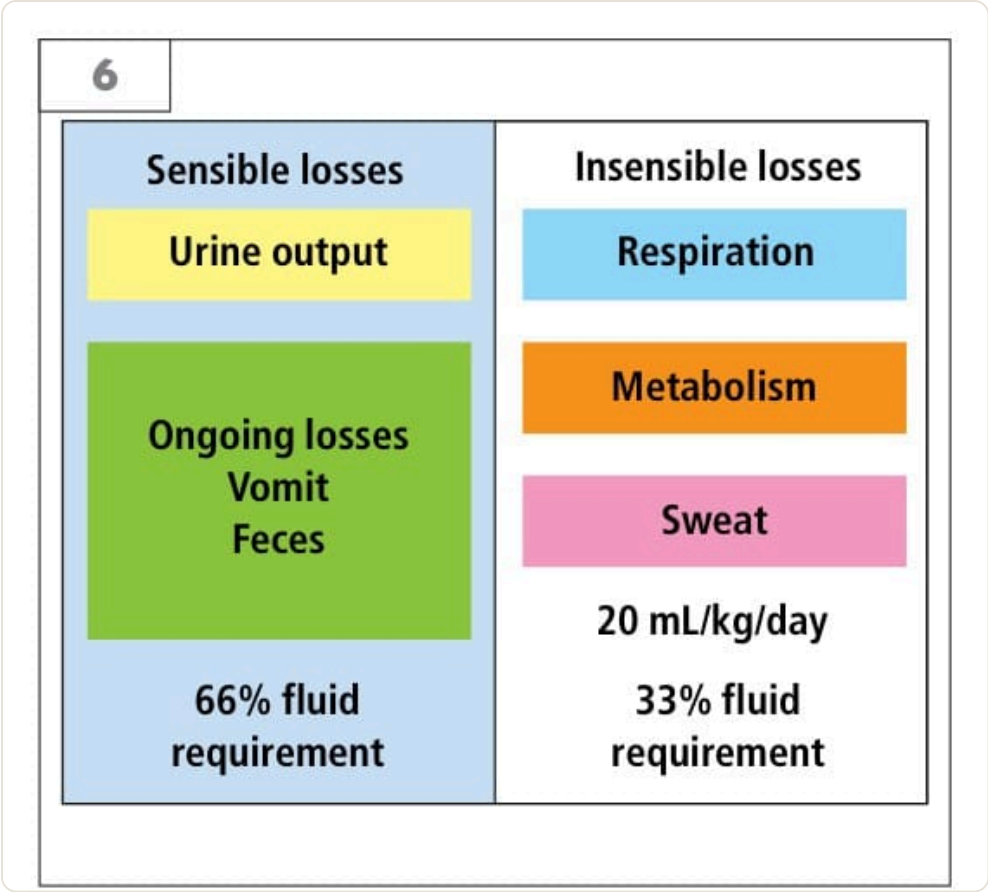

Fig 9 — Sensible and insensible losses. Sensible losses (urine, vomit, faeces) can be measured; insensible losses (respiration, metabolism, sweat) must be estimated — together they are the 'outs' a plan must match.

11 The three calculations: how much, and how fast

The physiology turns into a plan through three questions, each with a number attached. Work them in order — **perfusion first, then the deficit, then the day-to-day need** — and add any ongoing losses on top.

THE THREE FLUID CALCULATIONS **1 · Shock bolus** (perfusion threatened) — isotonic crystalloid **60–90 mL/kg (dog)** or **40–60 mL/kg (cat)**, about one blood volume — but given in **quarter aliquots** (~15–20 mL/kg dog, 5–10 mL/kg cat) over 10–15 minutes, reassessing perfusion between each and stopping when it normalises. **7.2% hypertonic saline 3–5 mL/kg** is a rapid small-volume alternative. **2 · Rehydration deficit** — **deficit (mL) = % dehydration × body weight (kg) × 1000**. E.g. 8% dehydration in a 20 kg dog = 0.08 × 20 × 1000 = **1,600 mL**, replaced over **12–24 hours** on top of maintenance. **3 · Maintenance** — about **2–3 mL/kg/hr (~40–60 mL/kg/day)**, but it scales with metabolic size (**≈ 70 × $BW_{kg}^{0.75}$ mL/day**), so small patients need more per kilogram — see the table below. Add ongoing losses on top.

Route matters too. Most fluids go **intravenously**; the **intraosseous** route is a lifesaver in collapsed patients and neonates when no vein can be raised, and **subcutaneous** fluids suit only mild, stable dehydration in a patient with normal perfusion. Whatever the route, the endpoints are the physiology you started with — perfusion parameters restored, hydration corrected, and no signs of volume overload.

Table 4
Daily maintenance fluid requirements based on body weight

kg Body weight	*mL Water per day*	*Approximate mL/hr*
1	100	4
2	130	5
3	160	7
4	190	8
5	220	9
10	370	15
15	520	21
20	670	28
25	820	34
30	970	40
35	1120	47
40	1270	53
45	1420	60
50	1570	65
55	1720	71
60	1870	78
65	2020	84
70	2170	90
75	2320	97
80	2470	103
85	2620	109
90	2770	115
95	2920	122
100	3070	128

Fig 10 — Daily maintenance fluid requirements. Maintenance scales with metabolic body size, so the per-kilogram requirement falls as the animal gets larger — a reference table beats a single 'mL/kg' rule.

12 Monitoring: how you know it is working, and when to stop

A fluid plan is a hypothesis. The numbers you calculated came from an estimated deficit and a guessed rate of ongoing loss, so the plan is only as good as the reassessment that follows it. Almost everything worth measuring is cheap.

Body weight is the most honest number on the chart. Weigh the patient at least twice daily on the same scales: one kilogram of acute weight change is very close to one litre of fluid, in either direction. **Packed cell volume and total protein** interpreted *as a pair* tell you more than either alone — both falling together suggests dilution, both rising suggests continuing dehydration, and a falling PCV with a steady total protein suggests you are looking at blood loss rather than water balance. **Urine output** should reach **1–2 ml/kg/hour** in a rehydrated patient; persistent **oliguria** below about 0.5 ml/kg/hour despite an apparently adequate volume is a warning that the kidney, not the plan, is the problem. Add capillary refill time, mucous membrane moisture, pulse quality and blood lactate, and you have a picture that no single value would have given you.

Fluid overload is easier to cause than to reverse, and cats, neonates and any patient with cardiac or renal disease reach it fastest. Watch for a rising respiratory rate and effort, new pulmonary crackles, **serous nasal discharge** — an early and often-missed sign — chemosis, jugular distension, peripheral or subcutaneous oedema, and unexplained weight gain. The correct response is to reduce or stop, not to add a diuretic reflexively; and remember that an over-filled patient has had its endothelial glycocalyx stressed, so it will leak more readily from then on.

The one hard number: potassium Isotonic crystalloids contain very little potassium — lactated Ringer's has about 4–5 mEq/L — so a patient that is hypokalaemic on presentation will usually be *more* hypokalaemic after a day of fluids, as the potassium is diluted and driven into cells. Supplementation is added to the bag on a sliding scale against the measured plasma potassium, and the cardinal safety rule is that the infusion rate must **never exceed 0.5 mEq/kg/hour**. Above that, potassium is a cardiac drug rather than a supplement.

13 Which fluid for which patient

With the physiology in hand, choosing a fluid is a short decision. The everyday workhorses are the **balanced isotonic crystalloids** — lactated Ringer's, Plasma-Lyte and the like — which suit almost every patient that needs rehydrating or replacing. Reach for something else only when the problem is specific:

Balanced isotonic crystalloid (lactated Ringer's, Plasma-Lyte-148) — the default for dehydration, replacement and maintenance in most patients. **0.9% saline** (normal saline) — highest sodium and chloride, no calcium: hyponatraemia (hyponatremia), hypochloraemic (metabolic) alkalosis, hypercalcaemia (hypercalcemia), or running alongside blood products. **Hypertonic saline (7.2%)** — rapid, small-volume resuscitation and raised intracranial pressure; **never** in a dehydrated patient. **Colloid** (synthetic starch or albumin) — to expand and *hold* the intravascular space, when the capillary is intact. **Dextrose & hypotonic fluids** (5% dextrose, 0.45% saline) — to supply free water or dextrose (e.g. hypernatraemia, hypoglycaemia), **not** to resuscitate.

Match the fluid's sodium, tonicity and oncotic properties to the deficit you diagnosed, and the choice follows directly from the physiology above.

14 Fluid therapy — frequently asked questions

Why does only about a quarter of a bag of saline stay in the bloodstream?

Because an isotonic crystalloid's solutes (mainly sodium) distribute across the whole extracellular space. The intravascular compartment is only about a quarter of the ECF, so within an hour roughly three-quarters of the infused volume has moved into the interstitium.

What is the difference between dehydration and hypovolaemia?

Dehydration is a deficit of total body water (mostly interstitial/intracellular), judged by skin-tent, eyes and membranes; hypovolaemia is a deficit of circulating blood volume, judged by heart rate, pulse and capillary refill. Hypovolaemia is corrected rapidly with boluses; dehydration is replaced slowly over hours.

Why is hypertonic saline effective in such small volumes?

It transiently raises the osmolality of the blood, drawing water out of the interstitium and cells into the vessel. A small volume produces a fast intravascular expansion — but the effect is temporary, so it is followed by an isotonic fluid to hold the gain.

When would you choose a colloid over a crystalloid?

When you need to expand and *hold* the intravascular volume, because a colloid's large molecules keep water in the vessel longer. The exception is a leaky capillary (sepsis/SIRS), where the colloid escapes into the interstitium and the advantage is lost.

How do you calculate a fluid deficit in a dog or cat?

Deficit in millilitres = **% dehydration × body weight (kg) × 1000**. For example, an 8% dehydrated 20 kg dog has a 0.08 × 20 × 1000 = **1,600 mL** deficit, usually replaced over 12–24 hours *in addition to* maintenance fluids and any ongoing losses.

What is the maintenance fluid rate for dogs and cats?

Roughly **2–3 mL/kg/hour (about 40–60 mL/kg/day)**. Because the requirement scales with metabolic body size (≈ 70 × body weight$_{kg}^{0.75}$ mL/day), small patients need proportionally more per kilogram and large patients less — so a weight-based table is more accurate than a single rule of thumb.

What is the shock dose of IV fluids?

About **60–90 mL/kg in the dog and 40–60 mL/kg in the cat** of an isotonic crystalloid (roughly one blood volume) — but it is given in *quarter aliquots* and titrated to perfusion, not infused all at once. A 3–5 mL/kg bolus of 7.2% hypertonic saline is a small-volume alternative.

How do you monitor a patient on intravenous fluids?

Weigh them twice daily — a kilogram of acute change is about a litre of fluid. Track **packed cell volume and total protein together**, because the pair separates dilution from dehydration from blood loss. Aim for a urine output of **1–2 ml/kg/hour** and treat persistent **oliguria** as a signal that the kidney needs attention rather than more volume. Then watch for the early signs of overload: rising respiratory rate, serous nasal discharge, chemosis and unexplained weight gain.

How much potassium can you add to a fluid bag?

Enough to correct the measured deficit, at a rate that **never exceeds 0.5 mEq/kg/hour**. That ceiling is the one hard safety number in fluid therapy. It matters because standard isotonic crystalloids contain only about 4–5 mEq/L of potassium, so a hypokalaemic patient on

unsupplemented fluids typically gets worse, not better — the potassium is diluted and simultaneously driven into cells.

Can too much fluid be harmful?

Yes. Because fluids redistribute, over-resuscitation causes interstitial and pulmonary oedema, dilutes plasma proteins and clotting factors, and — if sodium is corrected too quickly — can injure the brain. The right fluid, rate and endpoint matter as much as giving fluid at all.

When to refer

- **The shocky, tachycardic patient** — hypovolaemia: refill the vessel fast, titrate to perfusion, and expect a crystalloid to need several times the lost volume.
- **The vomiting, skin-tenting patient** — dehydration: replace the deficit steadily over 12–24 hours with a balanced crystalloid.
- **The head-trauma or refractory-shock patient** — a small hypertonic-saline bolus buys rapid volume by borrowing cellular water.
- **The septic, leaky-capillary patient** — where colloids and their water escape into the interstitium, and fluid choice gets harder.

Sources

1. Mazzaferro E. *Small Animal Fluid, Electrolyte and Acid-Base Disorders: A Color Handbook*. Manson Publishing — source of the labelled diagrams (Chapters 1, 3 & 4) reproduced for educational reference.
2. DiBartola SP (ed). *Fluid, Electrolyte, and Acid-Base Disorders in Small Animal Practice*. 4th ed. St Louis: Elsevier Saunders — applied physiology of body fluids.
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