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OEDEMA & STARLING FORCES

How Oedema Forms in Animals: The Starling Forces That Move Fluid Across Capillaries (PDF)

Oedema in animals (edema) is clinically noticeable excess interstitial fluid — and it always comes from the same place: the capillary. Inside: Seven labelled figures, one reference table and a 16-question self-test.

Contents

The 12 sections of this guide, in order.

IN SHORT

Oedema in animals (edema) is **clinically noticeable excess interstitial fluid** — and it always comes from the same place: the **capillary**. Fluid movement across the capillary wall is set by four **Starling forces** — the hydrostatic pressures **Pc** (blood, pushing out) and **Pi** (interstitium), and the colloid osmotic pressures **πc** (plasma, pulling in) and **πi** (interstitium). In a healthy capillary a small **net filtrate** leaves and is returned to the blood by the **lymphatics**. **Oedema forms** when any of four things breaks that balance: **↑ Pc** (heart failure), **↓πc** (hypoalbuminaemia), **↑ permeability** (inflammation, sepsis, burns) or **lymphatic block**. Master the four forces and every oedema you meet clinically — pulmonary oedema, ascites, bottle jaw, lymphoedema — falls into one of those four buckets.

KEY POINTS

1. **Oedema in animals** is *clinically noticeable* excess interstitial fluid — a symptom, not a disease. It always starts at the **capillary**: the balance of filtration and reabsorption has tipped, or the **lymphatics** can no longer drain the small net filtrate that normally leaves.
2. Fluid movement across a capillary is governed by the **four Starling forces**: hydrostatic pressures **Pc** (blood, pushing out) and **Pi** (interstitium, ~−7 mmHg in most tissues), plus colloid osmotic pressures **πc** (plasma, pulling water in at ~25 mmHg) and **πi** (interstitium, ~1 mmHg in most systemic tissues, much higher in the lung).
3. The **Starling equation** nets them: **NFP = (Pc − Pi) − σ(πc − πi)**. When this is positive fluid filters out; when negative, it is reabsorbed. Along one capillary **Pc falls** from ~35 (arteriolar) to ~15 mmHg (venular), so *filtration dominates early, reabsorption late*, with a **small net filtrate** returned by lymph — the classical picture, refined below by the revised Starling principle, under which most capillaries filter along their whole length and the lymphatics return all of it.
4. **Albumin is king**: it makes up most of the ~25 mmHg plasma colloid osmotic pressure. Drop plasma albumin (protein-losing enteropathy, protein-losing nephropathy, liver failure) and πc falls — fluid stops being pulled back in and **oedema forms in animals**.
5. The **lymphatic safety net** returns the day's **net filtrate plus escaped protein** via lymph capillaries → nodes → thoracic duct → subclavian veins. Lymph is driven forward by the **skeletal-muscle pump** and **respiratory pump**, with one-way valves. When it fails you get **lymphoedema**.
6. Four mechanisms produce every oedema in veterinary practice: **(1) ↑ Pc** (heart failure, venous obstruction), **(2) ↓πc** (hypoalbuminaemia), **(3) ↑ permeability** (inflammation, sepsis, burns, allergy — snake bite), and **(4) lymphatic block**.
7. Where oedema pools tells you which mechanism is at work. **Pulmonary oedema** = left heart failure (dog mitral disease); **ascites** = right heart failure, hypoalbuminaemia or liver disease; **bottle jaw** = protein loss to *Haemonchus* in sheep/goats/cattle; **peripheral/limb oedema** = lymphatic block or systemic congestion.

8. Every tissue — the lung especially — has three **safety factors** that activate the moment interstitial fluid begins to accumulate: rising Pi opposes further filtration, rising Pi promotes lymph flow (up to **10× baseline** in the lung), and the extra lymph flow flushes protein out of the interstitium (washdown of πi). The lung adds two advantages of its own — a rich pulmonary lymphatic supply and a **tight alveolar epithelium**. Overrun them (LHF pushing pulmonary Pc past ~25 mmHg) and you get cardiogenic **pulmonary oedema**.

01 Why capillary fluid balance matters in animals

Every clinical case of swelling — the panting Cavalier with wet lungs, the Yorkie with the pot-belly, the sheep with the puffy jaw — is really a story of one microscopic vessel gone wrong. **Oedema in animals** (edema, the American spelling) is defined precisely and usefully as *clinically noticeable excess interstitial fluid*. It is a sign, not a disease. It always starts at the same place: the **capillary**, the vessel where blood actually meets tissue and where every gram of water, every ion and every nutrient that leaves the circulation has to squeeze through. Understand what normally moves fluid across that capillary wall and, more importantly, what tips the balance, and every oedema you meet in practice belongs to one of only four buckets.

The framework for that understanding is a set of four pressures — the **Starling forces** — named after Ernest Starling, who worked them out at the turn of the twentieth century. They describe how much fluid should leave the capillary and how much should return, and they are as central to clinical medicine as the cardiac cycle itself — because failure of the heart raises capillary pressure and drives oedema, just as failure of the liver drops plasma protein and does the same. If you have read our companion article on cellular homeostasis, this is the vessel-scale story of how homeostatic fluid balance is achieved — and how it fails. This article walks through it from the ground up: the capillary bed, the ways water and solutes cross the wall, the four Starling forces, the Starling equation, why albumin is king, the lymphatic safety net, the four mechanisms of oedema, where the fluid pools clinically, and why the lung is (usually) hard to flood.

02 The capillary bed: where exchange happens

Every capillary bed sits at the same anatomical junction. Blood enters at an **arteriole**, passes through a smooth-muscle guard called the **precapillary sphincter**, spreads into a fine **network of capillaries**, then drains into a **venule**. The sphincter is the traffic policeman: it opens when the tissue needs blood (working muscle in an exercising horse, a fed intestine after breakfast) and closes when it does not, matching capillary flow to demand. That regulation matters clinically because sepsis paralyses these sphincters and floods the tissues with blood at low pressure — the maldistributive shock of septic patients is partly a capillary problem, not just a cardiac one.

The **capillary wall** is basically nothing: a single layer of endothelial cells sitting on a thin basement membrane. But there are three flavours of that wall, and the flavour is chosen by the tissue's job. **Continuous capillaries** have tight junctions between the endothelial cells and only very narrow *clefts* (about 4 nm wide) — these are the tightest, and they line skeletal muscle, skin, lung and, most tightly of all, the brain (where they form the blood-brain barrier). **Fenestrated capillaries** have circular pores of 40–80 nm punched through the endothelial cells — you find them where filtering and rapid solute movement matter, at the *kidney glomerulus*, along the gut mucosa and in the endocrine glands. **Discontinuous** (sinusoidal) capillaries have wide inter-endothelial gaps large enough for whole cells to cross, in *liver sinusoids*, *spleen* and *bone marrow*. Cunningham's Ch23 lays out the three types on p242 with a lovely comparison figure; PDA Ch11 covers the same ground around p484. This anatomical spectrum sets a rule of practice: **capillary permeability is a property of tissue**, not a single number. A vet's job is to know which capillary the case is playing out in.

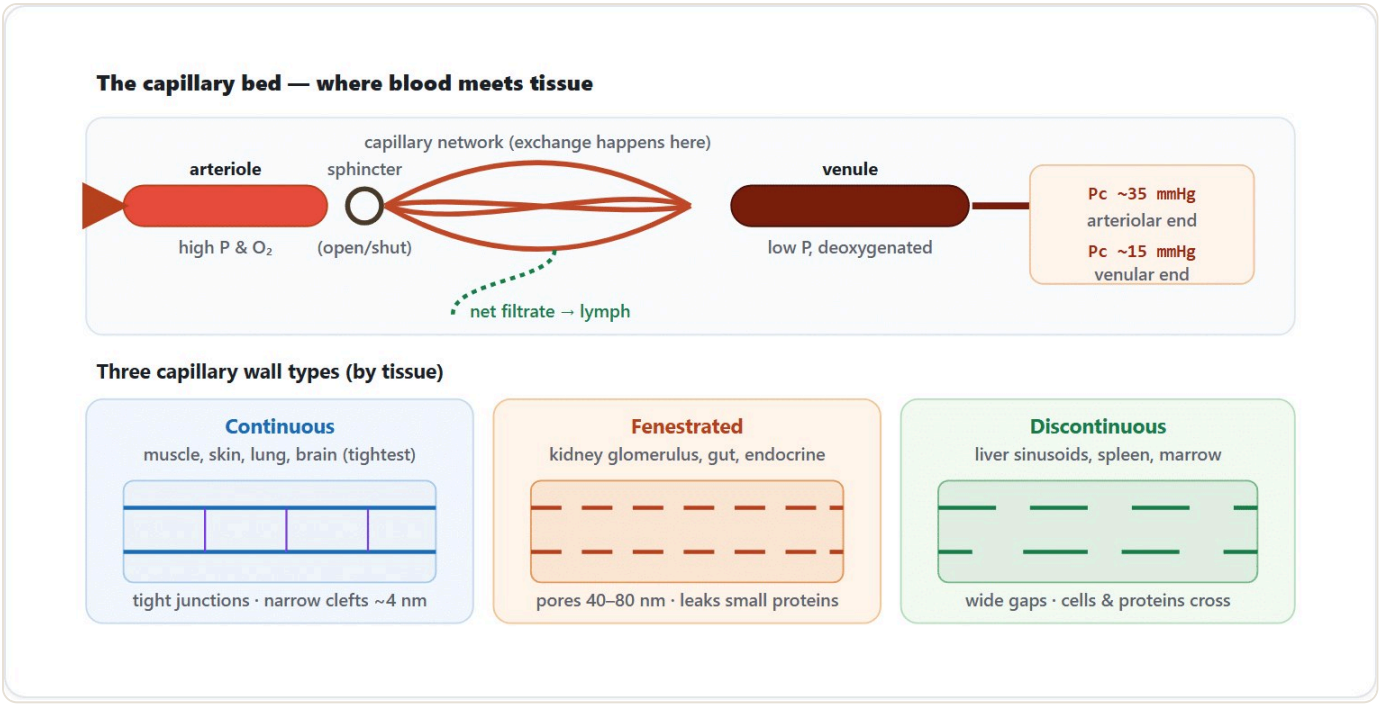

Fig 1 — The capillary bed and its three wall types. Blood enters at the arteriole under high pressure, passes the precapillary sphincter into the exchange network, and drains at the venule. Below: continuous (tight, e.g. muscle/skin/lung/brain), fenestrated (kidney/gut/endocrine) and discontinuous (liver/spleen/marrow) capillary walls.

03 How water and solutes cross the capillary wall

Once you know where the capillary is you can ask a second question: *how* does anything cross that impossibly thin wall? Cunningham (Ch23 p243) lays out four routes. First, **diffusion of lipid-soluble molecules** straight through the endothelial cell membrane — carbon dioxide, oxygen, steroid hormones, most anaesthetic (anesthetic) gases all take this shortcut. Second, **diffusion of small water-soluble molecules** through the endothelial *clefts* and (where they exist) fenestrations — water itself, sodium, chloride, glucose, urea. Third, **pinocytosis** (endocytotic vesicles that shuttle large molecules across the cell) — slow, low-volume, but vital for a few large solutes and for immunoglobulin transport. Fourth, and the one that matters for oedema, **bulk flow**: water plus its dissolved small solutes squeezed through the pores together, en masse, driven by a net hydrostatic pressure. Bulk flow is what the Starling forces control — and its net direction is what oedema is all about.

For diffusion the rate follows **Fick's law**: flux is proportional to the concentration gradient across the wall, the surface area available, and the permeability of the wall to the molecule, and it is inversely proportional to the wall thickness. Fick's law explains, for example, why gas exchange fails in **interstitial pulmonary oedema** even before the alveoli fill: fluid in the interstitium thickens the diffusion path, and Fick's law then predicts a fall in oxygen transfer. It is a small piece of physiology with an enormous clinical footprint.

04 The four Starling forces on one capillary

Consider one systemic capillary in a resting dog and slice it in cross-section. There are exactly four pressures acting on the fluid inside it, and only four. Learn them as opposing pairs.

1. **Pc — capillary hydrostatic pressure**. This is the blood pressure inside the capillary lumen, pushing fluid *out* through the wall. It *falls along the length* of the capillary: about **35 mmHg** at the arteriolar end, dropping to about **15 mmHg** at the venular end. Cunningham quotes a nominal mean of ~**18 mmHg** for the systemic capillary; teaching values sometimes round to 25–30. Pc is the force most under clinical influence: it rises when the heart fails or a vein is obstructed.
2. **Pi — interstitial hydrostatic pressure**. This is the pressure of the fluid *outside* the capillary in the interstitial space, and it opposes Pc. Its value is famously counter-intuitive: in most loose tissues it is slightly *negative*, around **−7 mmHg** in a resting dog (Guyton and Cunningham both cite this figure). A negative Pi actually *helps* fluid leave the capillary, because it pulls it out. In tight, encapsulated tissues (kidney parenchyma, bone marrow) Pi is closer to zero or slightly positive, which is one reason those tissues resist oedema.
3. **πc — plasma colloid osmotic pressure**. This is the osmotic pull generated by the *proteins* dissolved in plasma (mainly albumin), acting *into* the capillary because those proteins cannot cross the wall freely and so hold water in. In a normal dog πc is about **25 mmHg**. Since

these proteins are large and mostly reflected back at the wall, πc is a very stable inward force — unless the plasma protein pool collapses (hypoalbuminaemia).

4. **πi — interstitial colloid osmotic pressure**. This is the small oncotic pull of whatever proteins have leaked out into the interstitium, acting *out* of the capillary (in the direction of the leaked proteins). In most systemic tissues it is small — Cunningham quotes **~1 mmHg** as the nominal value — because normal lymphatic washdown keeps interstitial protein concentration very low. The *lung* is a striking exception, sitting at **~18 mmHg** because the pulmonary capillary is a little more permeable to protein than the systemic wall. πi also rises sharply in *inflammation*, when protein escapes en masse — and that rise then *promotes* further fluid loss.

Callout — the trap. Students often want to memorise "outward = Pc + πi; inward = πc + Pi". That is *almost* right but hides two things. First, Pi is subtracted rather than added (see the equation next). Second, a negative Pi (loose tissue) means subtracting a negative — i.e. adding — so it acts outward. The safe way is not the mnemonic but the equation itself.

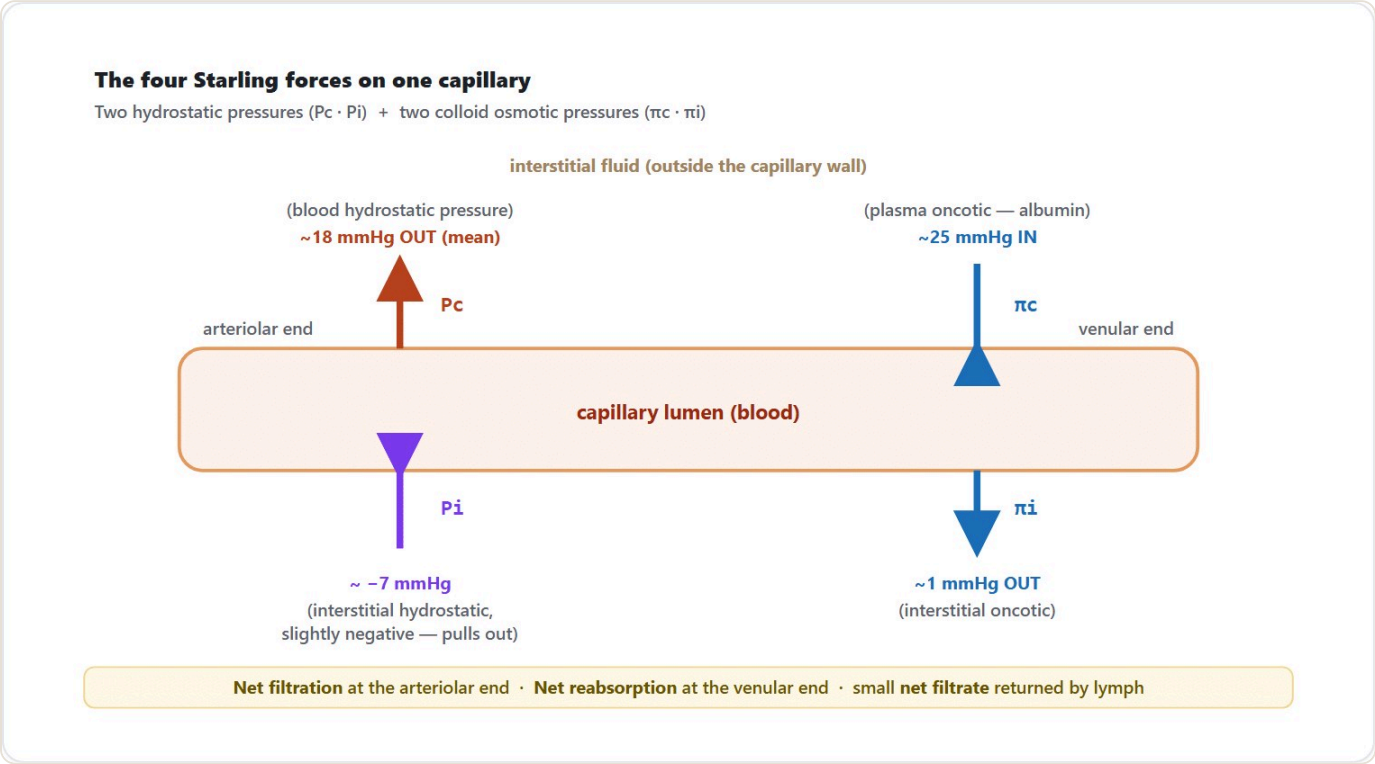

Fig 2 — The four Starling forces on one capillary. Pc (~18 mmHg mean, ~35 arteriolar → ~15 venular) pushes fluid out; πc (~25 mmHg) pulls it in; πi (~1 mmHg systemic) pulls out; Pi (~−7 mmHg) is a small extra outward pull in loose tissue. Net filtration at the arteriolar end, net reabsorption at the venular end; the small excess is returned by lymph.

05 The Starling equation: filtration vs reabsorption along one capillary

The four forces are pulled together in **Ernest Starling's equation**, which is the physiological heart of everything that follows. Written in its teaching form:

> **Net filtration pressure (NFP) = (Pc − Pi) − σ(πc − πi)**
>
> . When NFP is *positive*, fluid filters **out** of the capillary; when it is *negative*, fluid is **reabsorbed** back in. σ is the **reflection coefficient**, a number from 0 (freely permeable to protein) to 1 (fully impermeable). In a healthy systemic capillary σ is close to 1.

Plug Cunningham's nominal resting-dog numbers into the equation at the arteriolar end. Pc is ~35, Pi is ~−7, πc is ~25, πi is ~1. So NFP = (35 − [−7]) − 1×(25 − 1) = 42 − 24 = **+18 mmHg** outward at the arteriolar end: *fluid filters out*. Now do the same at the venular end, where Pc has fallen to ~15: NFP = (15 − [−7]) − 1×(25 − 1) = 22 − 24 = **−2 mmHg** — a small net inward pressure. Fluid *reabsorbs*. Take the average of the two ends (using Cunningham's mean Pc of ~18 mmHg): NFP = [18 − (−7)] − (25 − 1) = 25 − 24 = **+1 mmHg** outward across the whole capillary. That tiny positive residual is the whole point: *filtration dominates at the arteriolar end, reabsorption dominates at the venular end, and a small net filtrate leaves the capillary per unit time* — which the lymphatics carry away.

Callout — two common textbook errors. (1) "*Filtration = reabsorption*" is wrong. Every good textbook, and every one honest with the data (Cunningham Ch23 p246), says filtration slightly exceeds reabsorption. The excess is what the *lymphatics* carry away. (2) "*Starling forces*

stop at the venular end" is also wrong. They do not stop; they simply flip sign as Pc falls below πc on the way through.

The physiological consequence is quiet but continuous: a small, protein-rich **net filtrate** leaves the capillaries of every tissue in the body, every second, and has to be returned to the blood or that tissue will swell. Cunningham (Ch23 p247) puts the whole-body figure at roughly **2–4 L per day** across a resting adult mammal — small compared with plasma volume (~3 L in a 20 kg dog) but enough to double the interstitial space within a day if not returned. The vessel that returns it is the lymphatic capillary, and the mechanism is the topic below.

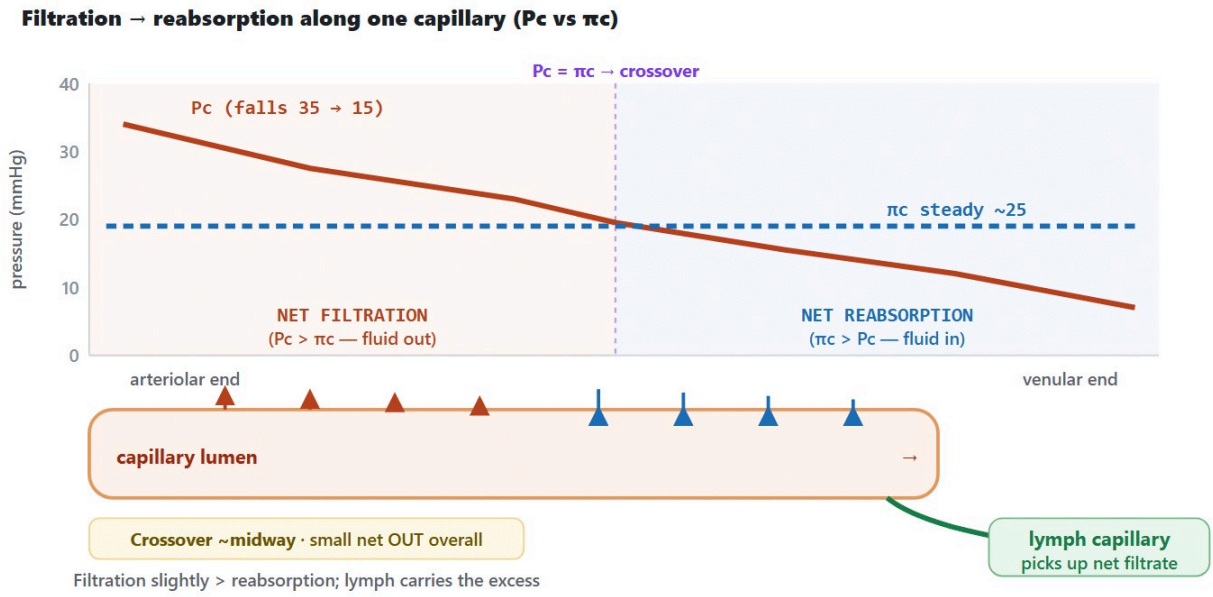

Fig 3 — Filtration vs reabsorption along one capillary. Pc falls from ~35 mmHg (arteriolar) to ~15 mmHg (venular); πc stays steady at ~25. Where Pc > πc fluid filters out; where πc > Pc fluid is reabsorbed. Small net filtrate leaves the bed and is collected by the lymphatic capillary. (Cunningham Ch23 pp245–247.)

06 The revised Starling principle: the glycocalyx, and why reabsorption mostly does not happen

The four-force model above is how the subject is taught, and it remains the right way to reason about oedema at the cage side. It is also, in one specific respect, an oversimplification worth knowing about — because the correction changes how you think about fluid therapy.

The classical model predicts filtration at the arteriolar end and **reabsorption at the venular end**, with lymph mopping up a small excess. Direct measurement does not support that in most tissues. The refinement, set out by **Levick and Michel in 2010** and now standard, rests on the **endothelial glycocalyx**: a gel-like layer of proteoglycans and glycoproteins coating the luminal surface of every capillary. It is the glycocalyx, not the endothelial wall as a whole, that acts as the molecular sieve.

Two consequences follow. First, the oncotic pressure that actually opposes filtration is the one in the **tiny sub-glycocalyx space** beneath that layer — which the filtration itself keeps almost protein-free — not the oncotic pressure of the interstitium at large. So raising interstitial protein does *not* reduce the opposing gradient the way the classical equation implies. Second, and more practically, in most tissues at steady state there is **filtration along the whole length of the capillary and essentially no venular reabsorption**. Fluid goes out; the **lymphatics bring all of it back**. This is often called the *no-reabsorption rule*, and it makes the lymphatic system far more central than the classical diagram suggests.

There are real exceptions — the renal peritubular capillaries, the intestinal mucosa and lymph nodes do reabsorb — and reabsorption also happens *transiently* after acute blood loss, when a falling capillary pressure pulls interstitial fluid back into the circulation. That internal autotransfusion is a genuine and useful compensation; it is simply not the steady state.

Why this matters clinically The glycocalyx is fragile. It is stripped by sepsis, inflammation, ischaemia (ischemia)–reperfusion, major surgery and, importantly, by **aggressive volume loading** — atrial natriuretic peptide released by a distended atrium degrades it directly. Once it is damaged, the reflection coefficient falls, protein leaks, and the patient becomes oedematous at a capillary pressure that would previously

have been safe. It is a large part of why over-resuscitation makes oedema worse rather than better, and why colloids retain far less of their theoretical advantage in a leaky patient than in a healthy one.

07 Colloid osmotic pressure: why albumin is king

Of the four Starling forces, three (Pc, Pi, πi) are set locally by the vessel and the tissue. Only one is set by the composition of the blood itself: **πc**, the plasma colloid osmotic pressure. Because the capillary wall is impermeable enough to protein that it holds a σ near 1 in most tissues, the plasma proteins act like a giant osmometer, pulling water back into the vessel. That pull is worth about **25 mmHg** in a healthy resting dog and roughly the same in the horse, cat, cow and sheep. Take away those proteins and the whole balance collapses.

Not all plasma proteins pull equally. **Albumin**, at ~35–45 g/L in a normal dog and cat, contributes roughly **65–80%** of πc despite being only about half of total plasma protein by mass. It punches above its weight because albumin molecules are small (~66 kDa) and numerous, and osmotic pressure depends on the *number* of solute particles, not their mass. Globulins, though roughly equal to albumin by mass, contribute only a small proportion of πc because they are much larger. So the practical rule is *albumin is king*: track it and you have tracked πc.

The clinical consequence is decisive. Any disease that drops plasma albumin drops πc and, once the drop is large enough to overwhelm the lymphatic reserve, oedema in animals appears. There are only a handful of relevant categories. **Protein-losing enteropathy (PLE)** is the classic in dogs — lymphangiectasia in a Yorkshire terrier or a soft-coated Wheaten losing whole-protein-strength lymph into the gut. **Protein-losing nephropathy (PLN)**, especially glomerular disease in a middle-aged dog, spills albumin into the urine. **Liver failure** reduces synthesis — the liver makes all the body's albumin — so end-stage cirrhosis in a dog or chronic hepatitis in a cat give hypoalbuminaemia over time. **Severe starvation or malabsorption** can achieve the same result, though it is rarer in Western practice. The clinical threshold at which oedema appears is roughly **albumin < 15–20 g/L** in a dog (or a total protein under ~35 g/L). Below that, watch for ascites, pleural effusion and subcutaneous pitting oedema.

Two more points on πc matter. First, the **reflection coefficient σ** is not a constant. In healthy systemic capillaries σ is close to 1; in inflamed or septic tissue σ falls sharply (down to 0.3–0.6), which is why sepsis oedema is protein-rich (exudate) rather than protein-poor (transudate). Second, giving crystalloid fluids intravenously **dilutes** πc — every litre of saline drops plasma albumin concentration — which is a physiological reason not to over-fluid a hypoalbuminaemic patient.

08 The lymphatic safety net

If the Starling forces normally leave a small net filtrate every day, where does it go? Not back through the capillary wall — the small residual outward NFP even at the venular end sees to that. The lost fluid, and the small amount of protein that inevitably escapes with it, is picked up by the **lymphatic capillaries**: blind-ended, very leaky endothelial tubes that sit alongside every tissue capillary bed. Lymphatic capillaries drain into progressively larger lymph **vessels**, which pass through **lymph nodes** (for immune surveillance) and finally into the **thoracic duct**, which empties into the **subclavian vein** at the base of the neck — returning the collected fluid and protein back to the bloodstream, and closing the loop.

Lymph does not move under its own steam. Lymph vessels have no muscular pump of their own equivalent to the heart, and they run against gravity in the standing animal. Two *external* pumps drive them. The **skeletal-muscle pump**: every time a limb muscle contracts it squeezes the lymphatics running between the muscle fibres (fibers), and one-way valves inside the lymph vessels keep the fluid moving centrally rather than sloshing back. The **respiratory pump**: each inspiration lowers intrathoracic pressure, which sucks lymph from the abdomen and limbs into the thoracic duct and onwards. That is why *immobility* promotes oedema (a recumbent hospitalised dog gets more distal-limb swelling than a walking one) and why post-operative encouragement of movement matters even after surgical procedures unrelated to the lymphatic system itself.

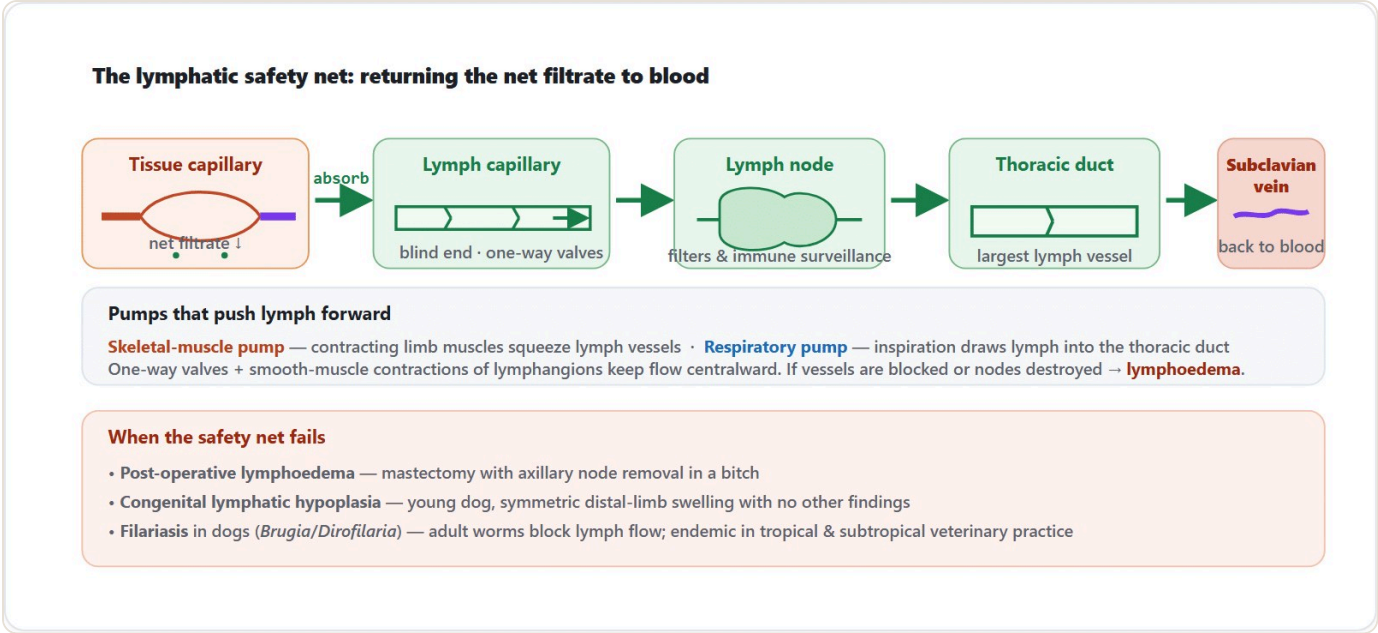

Fig 4 — The lymphatic safety net. Net filtrate leaves the tissue capillary bed, enters a blind-ended lymph capillary through overlapping endothelial flaps that act as one-way valves, and passes via afferent vessels through lymph nodes to the thoracic duct, which empties into the subclavian vein — returning fluid and escaped protein to the blood.

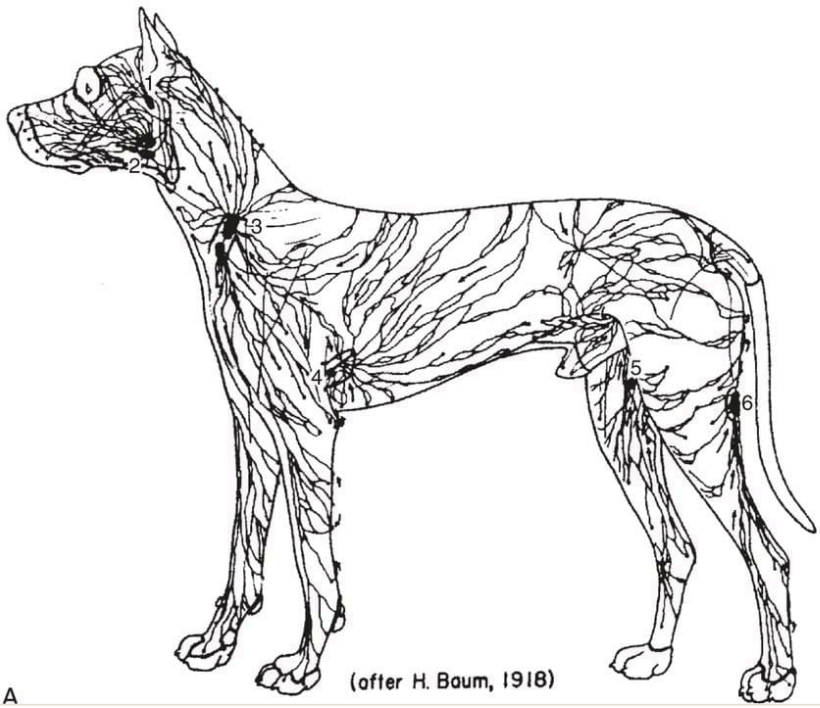

Fig 4a — The lymphatic anatomy of a dog. The vessels drain net filtrate + escaped protein from every tissue back to the subclavian veins; when this system fails (lymphoedema), Starling balance can no longer be corrected downstream.

Source: Cunningham 6e, Ch23 Fig 23.4A (after H. Baum 1918 / Sisson & Grossman's Anatomy of the Domestic Animal, vol 2, Saunders 1975), p 245.

The lymphatic system has a huge **reserve capacity**. Whole-body lymph flow can rise **tenfold or more** above baseline before overflow occurs — which is what protects tissues from mild rises in Pc or falls in πc. It is only when the lymphatic reserve is exceeded (heart failure, marked hypoalbuminaemia, severe inflammation, or lymphatic obstruction) that fluid actually pools clinically. The lung, as we will see below, has one of the largest reserves of all — roughly ten times its resting lymph flow — which is why cardiogenic pulmonary oedema does not appear until left-atrial pressure is really quite raised.

When the lymph system itself fails, the picture is called **lymphoedema**. Because lymph carries the escaped protein *as well as* the fluid, lymphoedema is *protein-rich* — you can measure this in the pooled fluid at surgery or on tap. Common causes include: **post-operative**

obstruction after removal of regional lymph nodes (an axillary lymphadenectomy after mastectomy is a classic cause of forelimb lymphoedema in a bitch); **congenital lymphatic hypoplasia** (young dogs, symmetric limb swelling, no other findings, no primary infection); **filariasis** in tropical settings, where adult worms block lymphatic vessels and drive massive limb enlargement (the human "elephantiasis" is the extreme).

09 How oedema forms in animals: the four mechanisms

Everything so far collapses into a very short list. There are exactly **four ways** the balance can tip toward oedema, and every disease you will meet in practice is one (or a combination) of these four. Cunningham (Ch23 pp247–250) sets them out in this order, and PDA covers the same list around pp490–492:

1. **Raised capillary hydrostatic pressure (↑ Pc).** Anything that raises Pc drives more fluid out at the arteriolar end and reduces reabsorption at the venular end. The commonest cause in practice is **heart failure** — left ventricular failure raises left atrial pressure, which backs up into the pulmonary capillaries and drives **pulmonary oedema**; right ventricular failure raises central venous pressure, which backs up into the systemic capillaries and drives **ascites** and peripheral oedema. Other causes: *venous obstruction* (a tumour compressing the caudal vena cava, thrombosis), *volume overload* from aggressive IV fluid therapy, and portal hypertension in liver disease (which drives ascites through ↑Pc in mesenteric capillaries). Fluid = usually **transudate** (low protein), because the wall itself is intact.
2. **Reduced plasma colloid osmotic pressure (↓πc).** Anything that drops plasma albumin drops πc and lets fluid stay in the interstitium. The causes are hypoalbuminaemia: **protein-losing enteropathy** (lymphangiectasia, IBD), **protein-losing nephropathy** (glomerular disease), **liver failure** and **severe starvation or malnutrition**. The clinical animal is usually a middle-aged dog with ascites (and often pleural effusion and peripheral pitting oedema too). Fluid = **transudate**.
3. **Increased capillary permeability (↑ permeability, ↓σ).** When the capillary wall itself becomes leaky — the reflection coefficient σ falls — both fluid and *protein* escape into the interstitium. Because protein escapes, πi rises sharply, and that in turn accelerates further fluid loss (a positive-feedback loop). Causes: **inflammation** (any local injury, from a skin abscess to peritonitis), **sepsis** (endotoxin makes the endothelium leak everywhere), **burns** (severe thermal injury to the wall itself), **allergic reactions** (histamine widens endothelial junctions), and **envenomation** (snake and insect bites often act by direct or histamine-mediated wall damage). Fluid = **exudate** (protein-rich, > 3.0 g/dL).
4. **Lymphatic block.** When the lymphatic drainage fails, the small daily net filtrate cannot return, and both fluid and escaped protein pool in the tissue. Causes: **surgical or oncological removal of nodes** (post-mastectomy limb lymphoedema); **tumour (tumor) compression** or invasion of major lymphatics; **filariasis**; **congenital lymphatic hypoplasia**. Fluid = **protein-rich**, often chylous if the thoracic duct is involved.

The reason this list matters at the cage-side is that *each mechanism has a signature*, and the signature tells you where to look. Watch which force has shifted, and remember: Pc up drives transudate; πc down drives transudate; permeability up drives exudate; lymphatic block drives protein-rich fluid. A fluid analysis after a tap of any effusion is often the single most useful test in an oedema workup.

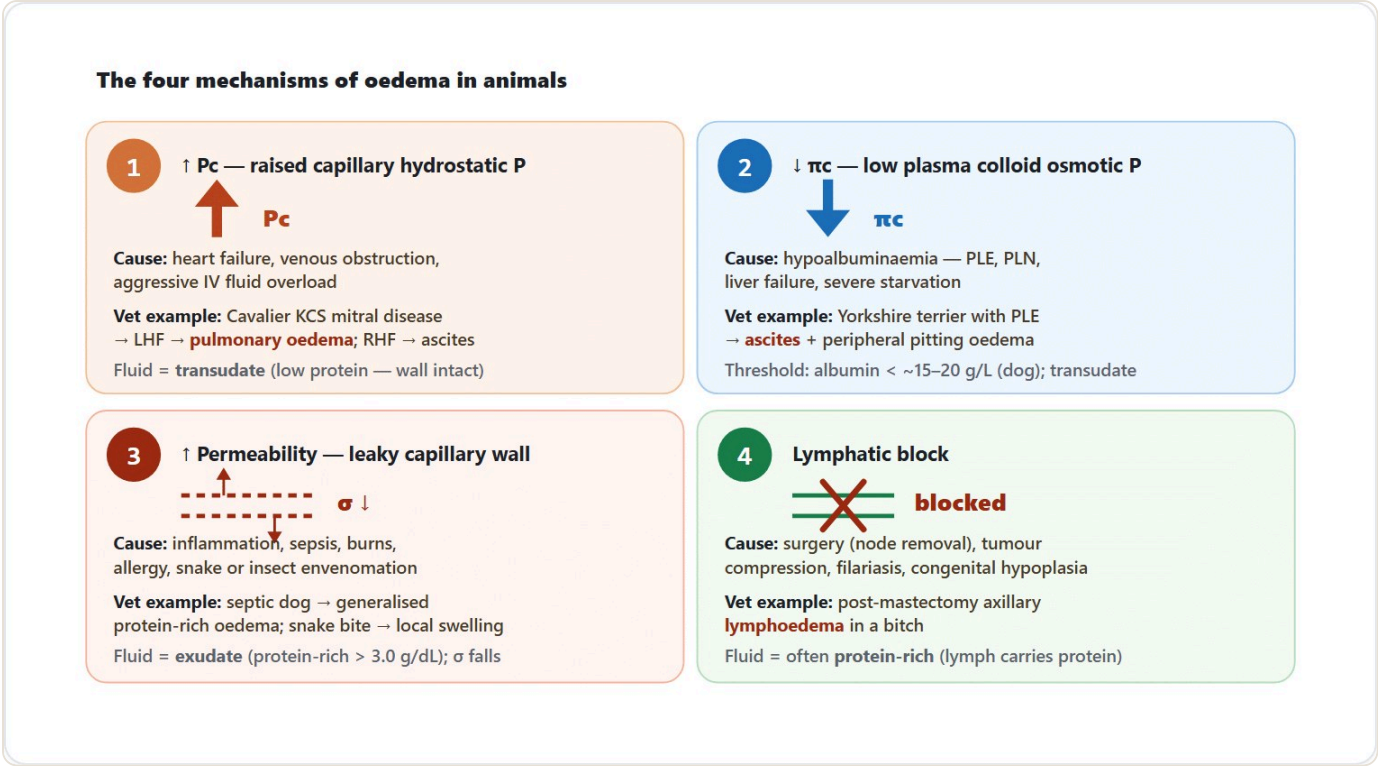

Fig 5 — The four mechanisms of oedema in animals: raised Pc (heart failure → pulmonary oedema/ascites); lowered πc (hypoalbuminaemia → ascites); raised permeability (inflammation/sepsis → exudate); and lymphatic block (post-op, filariasis → lymphoedema).

The four mechanisms, and the diseases that reach oedema through each. Every oedematous patient you meet is one of these, or a combination.

MECHANISM	WHAT CHANGES	TYPICAL CAUSES	WHERE THE FLUID SHOWS
↑ Capillary hydrostatic pressure (P_C)	More fluid driven out at the arteriolar end, less reabsorbed at the venular end	Heart failure — left-sided backs up into the pulmonary capillaries, right-sided into the systemic; venous obstruction; volume overload from aggressive IV fluids; portal hypertension	Pulmonary oedema (left) · ascites and peripheral oedema (right)
↓ Plasma colloid osmotic pressure	The osmometer that pulls water back into the vessel weakens	Hypoalbuminaemia — protein-losing enteropathy, protein-losing nephropathy, liver failure	Ascites, and generalised oedema at low albumin
↑ Capillary permeability	The reflection coefficient σ falls, so protein leaks out and takes water with it	Inflammation, sepsis, burns, vasculitis, envenomation	Local at the site of injury, or generalised in sepsis
Lymphatic obstruction	The safety net that normally returns filtered fluid is blocked	Neoplasia, surgical node removal, lymphangitis, congenital lymphoedema	Firm, regional swelling distal to the block

10 Oedema in practice: where it pools, and why

The elegant part is that the anatomy of the animal narrows the differential further. **Where** oedema appears tells you which capillary bed has failed, and that alone often tells you which of the four mechanisms is at work. Walk through the common presentations.

Pulmonary oedema is fluid in the pulmonary interstitium (interstitial oedema) and then in the alveoli themselves (alveolar oedema, the more advanced form). It is almost always **cardiogenic** in a dog or cat — the raised left-atrial pressure of left ventricular failure, most classically in a small-breed older dog with degenerative mitral valve disease (the Cavalier King Charles Spaniel is the archetype), or a Doberman with dilated cardiomyopathy, or a cat with hypertrophic cardiomyopathy. The clinical picture is dyspnoea (dyspnea), orthopnoea (cat sitting sternal), a soft cough in the dog, and, radiographically, a caudo-dorsal alveolar pattern with cardiomegaly. Non-cardiogenic causes exist too (ARDS from sepsis or severe inhalation injury, electrocution, neurogenic pulmonary oedema after severe head trauma), and here the wall permeability, not Pc, is the culprit.

Ascites is free fluid in the peritoneal cavity. In veterinary practice it has three main patterns. First, *right heart failure* (dilated cardiomyopathy in a boxer, tricuspid valve disease in an older dog, pericardial effusion causing tamponade) — raised central venous pressure backs up into the systemic capillaries, especially the hepatic sinusoids, and drives transudate into the abdomen. Second, *hypoalbuminaemia* from PLE, PLN or liver failure — the transudate is often modified (some protein has escaped) and albumin is low on serum biochemistry. Third, *portal hypertension* from liver disease or a portosystemic shunt with acquired complications — raised portal Pc drives fluid across the visceral capillaries. A tap plus fluid analysis (protein, cell count, cytology) narrows the three quickly.

Peripheral (subcutaneous) oedema in the ventral abdomen, prepuce, mammary chain and distal limbs is common in advanced right heart failure and severe hypoalbuminaemia in dogs, and in generalised inflammatory or allergic reactions in any species. It is *pitting* when pressed — you leave a finger imprint that slowly refills. In horses, ventral oedema (the "stocking-up" below the abdomen and prepuce) can be seen with equine viral arteritis, purpura haemorrhagica, or heart failure; in cattle, ventral oedema is a classical sign of pericardial disease from hardware disease (traumatic reticulopericarditis).

Bottle jaw is a diagnostic sign in itself. It is soft, pitting **submandibular oedema** under the jaw of ruminants (sheep, goats, cattle) and, more rarely, camelids. The mechanism is severe *hypoproteinaemia*: chronic *Haemonchus contortus* parasitism drops plasma albumin (the parasite is a blood-sucker in the abomasum) until πc falls, and gravity concentrates the resulting oedema under the jaw of the grazing animal. Bottle jaw is almost pathognomonic of severe blood-suckling parasitism in a sheep or goat, though liver flukes, other chronic protein-losing enteric disease, or heart failure can occasionally do the same. Treat the parasite, not the swelling.

Lymphoedema is fluid confined to the drainage territory of a blocked lymphatic. It is often *asymmetric* (one limb, or one region of the body), *protein-rich* on tap, and *non-pitting* or only lightly pitting in chronic cases. The commonest causes in small-animal practice are post-operative axillary or inguinal lymphadenectomy (mastectomy in the bitch, lymphoma staging in the dog), congenital limb lymphoedema in young puppies, and post-traumatic scarring. In tropical veterinary practice, filariasis of the peripheral lymphatics is possible.

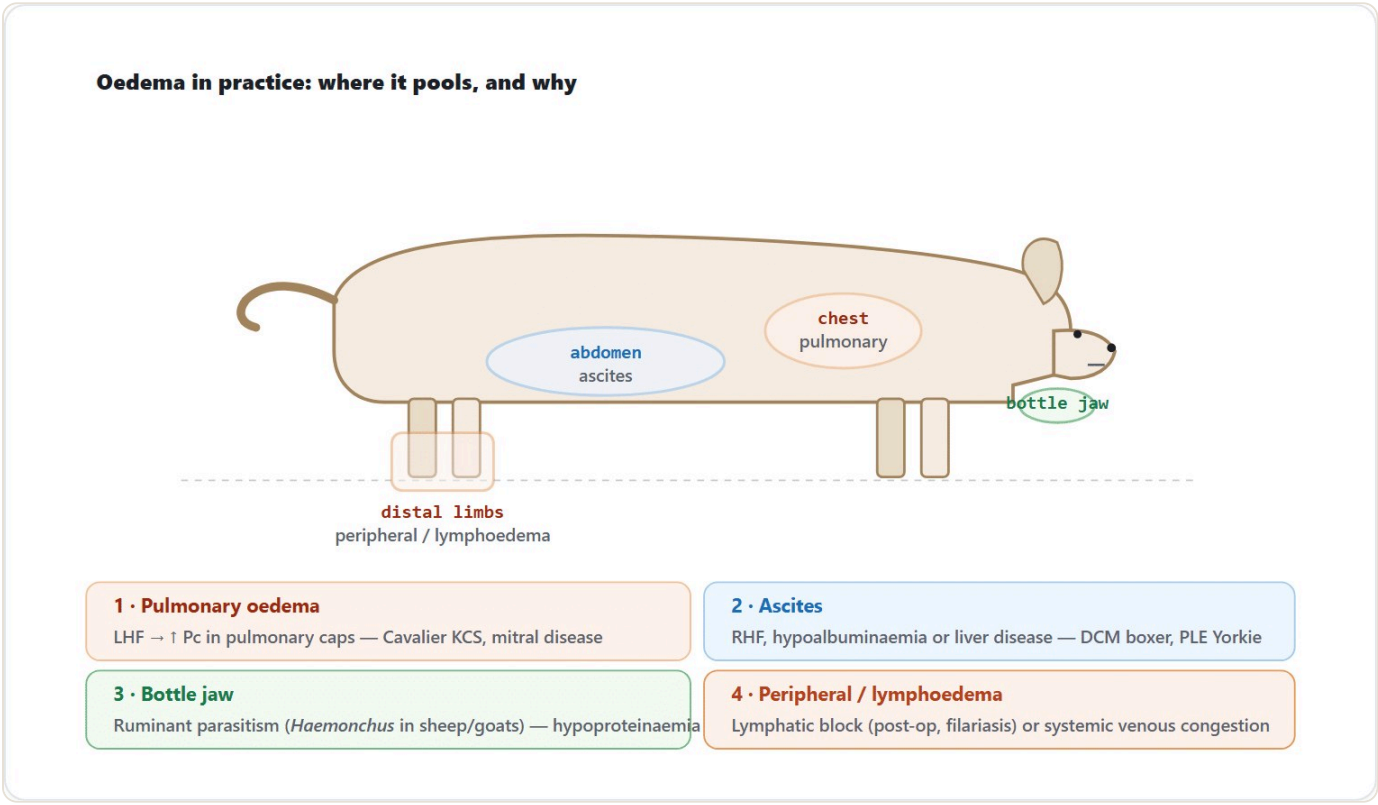

Fig 6 — Where oedema pools in a small-animal patient. Pulmonary oedema (LHF, mitral disease Cavalier); ascites (RHF, hypoalbuminaemia, liver); bottle jaw (parasitism in ruminants); peripheral/lymphoedema (lymphatic block or systemic congestion).

11 Why the lungs are (usually) protected: the safety factors against oedema

Left heart failure feeds fluid straight into the pulmonary capillaries; every dog with mitral regurgitation is one bad day away from pulmonary oedema. Yet remarkably, mild elevations of pulmonary Pc produce no oedema at all, and even fairly marked elevations are tolerated for hours. Why? Because Cunningham (Ch23 p248) describes **three safety factors** that stop mild-to-moderate rises in filtration from becoming clinical oedema in ANY tissue — and the lung then adds two advantages of its own. All three general factors are activated by the very thing you might expect to cause oedema: an *increase in interstitial fluid volume*.

Safety factor 1 — rising Pi opposes further filtration. As soon as fluid begins to accumulate, the interstitial hydrostatic pressure rises from its resting ~−7 mmHg toward zero and eventually positive. Because Pi appears in the Starling equation as (Pc − Pi), a higher Pi directly reduces the driving force behind filtration — the tissue pushes back on the leaking capillary.

Safety factor 2 — rising Pi promotes lymph flow. Interstitial hypertension squeezes fluid into the lymphatic capillaries at their overlapping-endothelial-cell openings. In the lung, lymph flow can rise **tenfold or more** above baseline before it is overwhelmed — this is the biggest single reason mild pulmonary hypertension does not immediately flood the alveoli.

Safety factor 3 — lymph flow washes down πi. Because lymph carries protein out of the interstitium along with the fluid, sustained high lymph flow flushes protein *out*. That lowers πi (in systemic tissue) and keeps the (πc − πi) gradient large, so the venular end continues to reabsorb.

On top of these three, the lung has two further advantages that make it uniquely difficult to flood. **Lung advantage 1 — a well-developed pulmonary lymphatic supply** gives it a bigger safety-factor-2 reserve than most tissues; the constant pressure changes of breathing help propel lymph forward. **Lung advantage 2 — a tight alveolar epithelium.** The alveolar epithelial cells are joined by tighter junctions than the endothelium of the pulmonary capillary, so fluid that crosses from blood into the interstitium does not readily reach the alveolar air space. Alveolar flooding is a *late* event, not an early one: the interstitial reservoir has to fill first.

One caveat that Cunningham makes explicit (p248): the pulmonary interstitial oncotic pressure (πi) is nominally about **18 mmHg** in the lung — much higher than the ~1 mmHg of systemic tissue — because the pulmonary capillary wall is a little more permeable to protein than the systemic wall. Plug the pulmonary numbers into the Starling equation and the lung actually has a *higher* resting net filtration force (~+9 mmHg) than a systemic capillary (~+1 mmHg). The lung is *not* spared because filtration is inherently low; it is spared because those three safety factors + the lung-specific advantages catch the filtrate before it becomes oedema.

The clinical consequence: **interstitial pulmonary oedema** (fluid in the interstitium only) is common in early left heart failure and often shows as a soft cough and mild dyspnoea only, with radiographic peri-hilar interstitial infiltrates. Only when the interstitial reserve is exhausted do you get **alveolar pulmonary oedema** — the classic caudo-dorsal alveolar pattern, frank respiratory distress, and pink frothy sputum. The threshold is roughly a left atrial pressure of **25 mmHg** in an otherwise healthy animal — a threshold that is lowered by hypoalbuminaemia (less πc reserve), rapid rate of rise (no time for lymphatic response), or damaged alveolar epithelium (ARDS pattern, non-cardiogenic pulmonary oedema).

12 Clinical reasoning at the cage-side: reading oedema + the fluid-therapy caution

Bring it all together. A patient presents with oedema. You want two pieces of information: **where** the fluid is (pattern) and **what protein content** it has (tap and fluid analysis). Between them these narrow the differential to one of the four mechanisms.

- **Pulmonary infiltrates in a coughing/dyspnoeic older small-breed dog:** pulmonary oedema from left heart failure — most likely mitral valve disease. Confirm with echocardiography; treat with a loop diuretic (furosemide) as a preload-reducing measure, an ACE inhibitor and pimobendan for cardiac remodelling and contractile support.
- **Ascites + hypoalbuminaemia (<15–20 g/L) in a middle-aged dog:** workup for PLE (fecal cover +/− endoscopy), PLN (urine protein:creatinine ratio, renal biochem) and liver failure (serum bile acids, liver enzymes). Treat the underlying disease; support with an appropriate diet; consider colloid support in the sickest cases; be cautious with IV fluids.
- **Ascites in an older dog with an enlarged heart and jugular distension:** right heart failure — from tricuspid endocardiosis, dilated cardiomyopathy, or pericardial effusion with tamponade (a boxer or golden with a large heart and muffled sounds needs pericardiocentesis). Confirm echocardiographically; treat the underlying pathology.
- **Rapidly evolving generalised protein-rich oedema in a systemically ill dog:** think sepsis or SIRS, systemic inflammatory response after burns, or a bad allergic/anaphylactic response. Fluid on tap will be exudative. Source-control the infection, treat the underlying inflammation, support the circulation.
- **Submandibular pitting oedema in a sheep, goat or calf:** bottle jaw — treat the parasite (*Haemonchus*, other blood-suckers) with an appropriate anthelmintic and rebuild protein status; the oedema resolves in days to weeks.
- **Asymmetric limb swelling in a post-operative or young dog:** lymphoedema — treat with drainage, physiotherapy, compression bandaging where appropriate, and management of the underlying obstruction.

Fluid-therapy caution — a graduate-level rule. Do *not* aggressively fluid-load a hypoalbuminaemic animal. Every litre of crystalloid raises Pc and further dilutes πc, widening the Pc − πc gap and driving more fluid into the interstitium — you can precipitate frank pulmonary or peripheral oedema. Fluid these patients cautiously, prefer to correct the underlying protein loss first, and consider synthetic colloid or plasma in the sickest patients (bearing in mind the modern concerns about synthetic colloids and renal safety in dogs). This is one of the most common iatrogenic causes of oedema in a hospitalised patient and it is entirely preventable if you keep the Starling equation in mind. For the primary heart-failure animal that *is* in cardiogenic pulmonary oedema, remember the treatment is the *opposite*: diuretics to lower Pc, not IV fluids.

13 Quiz: test yourself

Sixteen questions across the capillary bed, the four Starling forces, the revised principle, the lymphatic safety net and the four mechanisms of oedema. Answers are beneath each question — cover them and work down.

1. How wide are the clefts in a continuous capillary, and where do you find them?**Answer:** About **4 nm**. They line **skeletal muscle, skin and lung** — and most tightly of all the **brain**, where they form the blood–brain barrier.2. What is a fenestrated capillary, and where does it sit? **Answer:** One with circular pores of **40–80 nm** punched through the endothelial cells, where filtering and rapid solute movement matter: the **kidney glomerulus**, the **gut mucosa** and the **endocrine glands**.3. What does Fick's law say?**Answer:** Flux is proportional to the **concentration gradient**, the **surface area** and the **permeability** of the wall, and inversely proportional to the **wall thickness**.4. Why does gas exchange fail in interstitial pulmonary oedema before the alveoli even fill?**Answer:** Fluid in the interstitium **thickens the diffusion path**, and Fick's law then predicts a fall in oxygen transfer. The alveoli do not have to flood for the patient to become hypoxaemic.5. What is capillary hydrostatic pressure at each end of a systemic capillary?**Answer:** About **35 mmHg** at the arteriolar end, falling to about **15 mmHg** at the venular end. Cunningham quotes a nominal mean of about **18 mmHg**.6. What is the reflection coefficient σ, and what is it in a healthy capillary?**Answer:** A number from **0** (freely permeable to protein) to **1** (fully impermeable). In a healthy systemic capillary it is **close to 1**.7. What is the revised Starling principle, and who set it out?**Answer: Levick and Michel, 2010**, now standard. It rests on the **endothelial glycocalyx** — a gel-like layer of proteoglycans and glycoproteins coating the luminal surface — and its consequence is that **steady-state reabsorption mostly does not happen**.8. How much is plasma colloid osmotic pressure worth, and across which species?**Answer:** About **25 mmHg** in a healthy resting dog, and roughly the same in the **horse, cat, cow and sheep**.9. What moves lymph, given that lymphatics have no pump of their own?**Answer:** Chiefly the **skeletal-muscle pump** — a contracting limb muscle squeezes the lymphatics between its fibres, and **one-way valves** keep the fluid moving centrally.10. What are the four ways the balance can tip toward oedema?**Answer: Raised capillary hydrostatic pressure**, **fallen plasma colloid osmotic pressure**, **raised capillary permeability**, and **lymphatic obstruction**. Every case is one of the four, or a combination.11. How do left- and right-sided heart failure differ in where the fluid goes?**Answer: Left** ventricular failure raises left atrial pressure, which backs up into the **pulmonary capillaries** — pulmonary oedema. **Right** raises central venous pressure, which backs up into the systemic capillaries — **ascites and peripheral oedema**.12. What is "stocking-up" in a horse, and what causes ventral oedema in cattle?**Answer:** Ventral oedema below the abdomen and prepuce, seen with **equine viral arteritis, purpura haemorrhagica or heart failure**. In cattle ventral oedema is a classical sign of **pericardial disease from hardware disease**.13. Why do the lungs tolerate a raised capillary pressure that would flood another bed?**Answer:** They carry **safety factors**: mild elevations of pulmonary P_c produce no oedema at all, and even fairly marked elevations are tolerated for hours.14. Two pieces of information narrow any oedema case. What are they?**Answer: Where the fluid is** (the pattern) and **what protein content it has** (tap and fluid analysis). Between them they point to one of the four mechanisms.15. A coughing, dyspnoeic older small-breed dog has pulmonary infiltrates. What is the likely diagnosis and treatment?**Answer: Pulmonary oedema from left heart failure**, most likely **mitral valve disease**. Confirm on echocardiography; treat with a loop diuretic (**furosemide**) to reduce preload, plus an ACE inhibitor and pimobendan.16. A middle-aged dog has ascites and an albumin under 15–20 g/L. What is the workup?**Answer: Protein-losing enteropathy** (faecal cover, endoscopy), **protein-losing nephropathy** (urine protein:creatinine, renal biochemistry) and **liver failure** (bile acids, liver enzymes). Be cautious with IV fluids.

14 Oedema in animals — frequently asked questions

What are Starling forces in simple terms?

The Starling forces are the four pressures that act on every capillary and decide whether fluid leaves the vessel or stays in it. Two hydrostatic pressures (Pc inside the vessel, Pi in the tissue) push fluid across the wall by ordinary pressure. Two colloid osmotic pressures (πc from plasma proteins, πi from a few leaked interstitial proteins) pull water by osmosis. In a healthy capillary the outward and inward forces almost balance, with a small net filtrate leaving — and the lymphatics collect and return it.

What causes oedema in animals?

Four mechanisms: (1) raised capillary hydrostatic pressure Pc — heart failure or venous obstruction; (2) reduced plasma colloid osmotic pressure πc — hypoalbuminaemia from protein-losing enteropathy, protein-losing nephropathy or liver failure; (3) increased capillary permeability — inflammation, sepsis, burns, allergy, snake bite; (4) lymphatic block — post-operative, congenital or filariasis. Every clinical oedema you meet falls into one (or a combination) of these four.

What is the difference between transudate and exudate?

Transudate is protein-poor (total protein < 1.5 g/dL, SG < 1.017) and typical of raised Pc (heart failure) or lowered πc (hypoalbuminaemia), where the capillary wall is intact and only water plus small solutes escape. Exudate is protein-rich (> 3.0 g/dL) and typical of raised permeability (inflammation, sepsis) where the wall itself leaks and both fluid and protein escape.

Why does hypoalbuminaemia cause oedema?

Albumin generates most of the ~25 mmHg plasma colloid osmotic pressure that pulls water back into the capillary at the venular end. Drop plasma albumin below ~15–20 g/L in a dog and πc falls; the venular end can no longer reabsorb, the lymphatics eventually saturate, and fluid

accumulates — typically as ascites, sometimes pleural effusion and peripheral oedema.

How does heart failure cause pulmonary oedema?

Left ventricular failure raises left atrial pressure, which backs up into the pulmonary veins and then the pulmonary capillaries, raising pulmonary Pc. Once the three safety factors and the two lung-specific advantages (rising Pi opposing filtration, ~10× lymph flow reserve, washdown of πi, rich pulmonary lymphatics, tight alveolar epithelium) are overwhelmed — typically at a left atrial pressure of about 25 mmHg — fluid pools first in the pulmonary interstitium and then floods the alveoli. This is the classical picture in a Cavalier King Charles Spaniel with degenerative mitral valve disease.

Is fluid reabsorbed at the venous end of the capillary?

Mostly not — this is the part of the classical teaching model that has been revised. Under the **revised Starling principle** (Levick & Michel, 2010) the endothelial **glycocalyx** is the true semipermeable layer, and the oncotic pressure that opposes filtration is the one in the protein-poor space just beneath it. In most tissues at steady state fluid filters out along the *whole* capillary and the lymphatics return all of it; there is no meaningful venular reabsorption. The exceptions are the renal peritubular capillaries, the intestinal mucosa and lymph nodes — and a transient reabsorption after acute blood loss, which is a real compensatory autotransfusion.

How is oedema treated?

By correcting the force that is out of balance, not by reaching automatically for a diuretic. Raised capillary pressure from **congestive heart failure** is the one that genuinely responds to furosemide plus treatment of the underlying heart disease. Low colloid osmotic pressure from **hypoalbuminaemia** needs the protein loss found and stopped — protein-losing enteropathy or nephropathy, liver failure, or a burn — because a diuretic here removes intravascular volume from a patient who has too little of it already. Increased permeability from sepsis, inflammation or **vasculitis** is treated by treating the cause. Lymphatic obstruction may need drainage or surgery. Diuretics relieve a symptom; only one of those four mechanisms is actually a diuretic problem.

Can I download these Starling forces study notes as a free PDF?

Yes — these are complete, illustrated veterinary study notes on the Starling forces and oedema in animals, free to download as a PDF with no signup. For more veterinary study notes on the cardiovascular system, browse our veterinary physiology hub.

When to refer

- A dyspnoeic, coughing older small-breed dog (especially a Cavalier King Charles Spaniel) with a systolic apical murmur — probable cardiogenic **pulmonary oedema** from mitral valve disease; stabilise with furosemide + oxygen, echocardiography, refer to cardiology.
- Ascites in a dog with plasma albumin <15–20 g/L — work up for **PLE/PLN/liver failure**; treat the underlying disease and fluid cautiously; consider referral for endoscopy or renal biopsy.
- Rapidly evolving generalised protein-rich oedema with fever and systemic illness — consider **sepsis or SIRS**; source-control, refer for intensive fluid, cardiovascular and inflammatory support.
- Persistent asymmetric limb swelling after mastectomy or node removal — probable post-operative **lymphoedema**; refer for physiotherapy, compression bandaging and long-term drainage management.
- Submandibular pitting oedema (**bottle jaw**) in a sheep, goat or calf — treat for *Haemonchus contortus* and other blood-sucking parasites; investigate the flock, not just the individual.

Sources

1. Klein BG (ed). *Cunningham's Textbook of Veterinary Physiology*. 6th ed. St Louis: Elsevier; 2020. **Ch 23 'Capillaries and Fluid Exchange' (pp 241–252)** — primary source for the Starling forces, the Starling equation, oedema mechanisms and the three safety factors.
2. Sjaastad ØV, Sand O, Hove K. *Physiology of Domestic Animals*. 3rd ed. Oslo: Scandinavian Veterinary Press; 2016. **Ch 11 'The Cardiovascular System' (pp 482–494)** — secondary source for capillary microcirculation and species differences in lymphatic anatomy.
3. Ettinger SJ, Feldman EC, Côté E. *Textbook of Veterinary Internal Medicine*. 8th/9th ed. Elsevier; 2017–2024. Chapters on valvular heart disease, CHF, PLE, PLN and hepatic disease — clinical anchor for pulmonary oedema, ascites and the fluid-therapy caution.

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