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How Oedema Forms in Animals

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.

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How Oedema Forms in Animals

STUDY NOTES · BASIC SCIENCES · PHYSIOLOGY · UPDATED 2026-07-05

CONTENTS

LEARNING OBJECTIVES

After working through these notes you will be able to:

- ✓ Define oedema in animals and state that it is clinically noticeable excess interstitial fluid arising from failure of capillary fluid balance.
- ✓ Name the four Starling forces and give typical resting mmHg values for a systemic capillary in a dog.
- ✓ Write the Starling equation and explain what net filtration pressure means at the arteriolar vs venular end of a capillary.
- ✓ Explain why albumin is the dominant contributor to plasma colloid osmotic pressure and how hypoalbuminaemia causes oedema.
- ✓ Describe how the lymphatic system returns the net filtrate to the blood and what happens when it fails.
- ✓ List the four mechanisms of oedema and give one common veterinary example of each.
- ✓ State the safety factors that protect the lungs from oedema and how heart failure overwhelms them.

TL;DR

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.

AT A GLANCE

WHAT OEDEMA IS	Clinically noticeable **excess interstitial fluid** — the failure of capillary fluid balance
WHERE FLUID MOVES	Across the **capillary wall**: continuous, fenestrated or discontinuous, depending on tissue
FOUR STARLING FORCES	**Pc** (out ~18 mmHg mean; 35 arteriolar → 15 venular) · **Pi** (~−7 mmHg) · **πc** (in ~25) · **πi** (out ~1)
ALONG ONE CAPILLARY	**Pc falls** arteriolar (~35) → venular (~15); πc steady → filtration early, reabsorption late
NET RESULT	Small **net filtrate** per day, returned by lymphatics; balance is dynamic not zero
COLLOID PRESSURE	Plasma proteins (mostly **albumin**) generate ~25 mmHg pulling water back in
FOUR MECHANISMS OF OEDEMA	↑ **Pc**, ↓ **πc**, ↑ **permeability**, **lymphatic block**
WHERE IT POOLS	**Pulmonary** (LHF), **ascites** (RHF/hypoalbuminaemia), **bottle jaw** (parasitism), **lymphoedema**
LUNG SAFETY	3 safety factors + **~10× lymph reserve** & tight alveolar epithelium keep the lung dry

01 Why oedema matters

- **Oedema in animals** = clinically noticeable excess interstitial fluid; a *sign*, not a disease. Every case belongs to one of four buckets (see §7).
- **Where it starts:** the **capillary** — the vessel where blood meets tissue and where every gram of water leaves the circulation.
- **Framework:** four pressures, the **Starling forces**, decide how much fluid leaves and how much returns. Failure of any = oedema.

02 The capillary bed & wall types

- **Layout:** arteriole (high P, high O_2) → **precapillary sphincter** → capillary network (exchange) → venule (low P, deoxygenated). Pc falls ~35 → ~15 mmHg from arteriolar to venular end.
- **Continuous** — muscle, skin, lung, brain; tight junctions, narrow clefts ~4 nm (brain = tightest, blood-brain barrier).
- **Fenestrated** — kidney glomerulus, gut, endocrine glands; pores 40–80 nm, leaks small proteins.
- **Discontinuous** (sinusoids) — liver, spleen, marrow; wide gaps let cells & proteins cross.

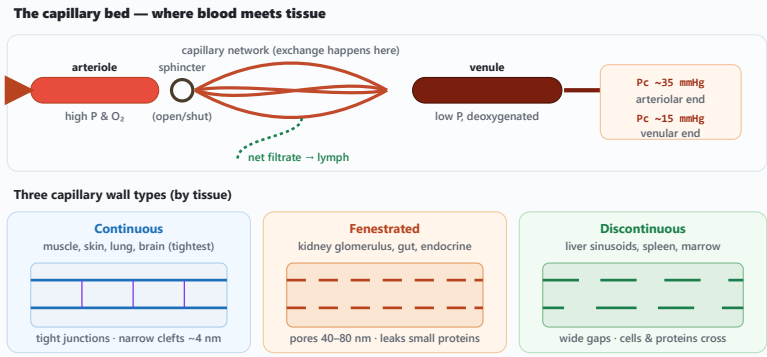

Fig 1 — The capillary bed: arteriole → sphincter → capillary → venule. Inset: 3 wall types (continuous / fenestrated / discontinuous).

03 How things cross the wall

- **Diffusion** — lipid-soluble (O_2, CO_2, ethanol) crosses freely through endothelial cells. **Fick's law**: rate ∝ area × concentration gradient.
- **Pores / clefts** — small water-soluble (water, ions, glucose) pass through 4 nm intercellular pores (only ~1% of wall area, so diffusion here is slower).
- **Pinocytosis / transcytosis** — endocytotic vesicles ferry large molecules (albumin, globulins) across; slow.
- **Bulk flow** — net movement of fluid + solutes through pores under a pressure gradient; the substrate of the Starling equation.

04 The 4 Starling forces

- **Pc** — capillary hydrostatic pressure; pushes fluid **OUT**; ~18 mmHg mean (35 arteriolar → 15 venular).
- **Pi** — interstitial hydrostatic pressure; slightly **NEGATIVE** (~ −7 mmHg); pulls fluid OUT (adds ~7 to net filtration).
- **πc** — plasma colloid osmotic (oncotic); pulls fluid **IN**; ~25 mmHg (mostly albumin).
- **πi** — interstitial oncotic; pushes fluid OUT; ~1 mmHg in most systemic tissues (per Cunningham) — low because lymph "washes down" escaped protein. Lung is the exception at ~18 mmHg.
- **Starling equation**: NFP = (Pc − Pi) − σ(πc − πi). σ (reflection coefficient) = 1 when wall fully reflects protein, <1 when leaky.

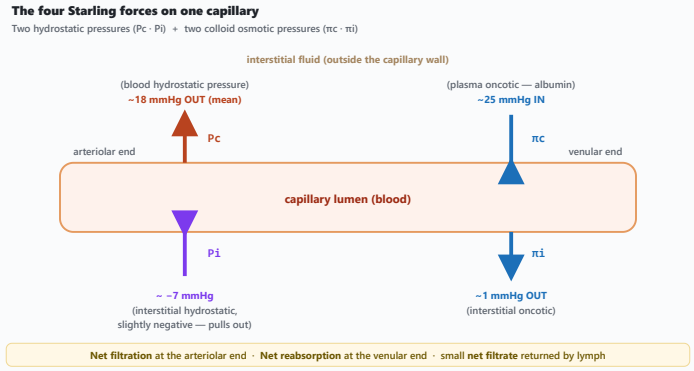

Fig 2 — The four Starling forces: Pc & Pi (hydrostatic) + πc & πi (colloid osmotic).

05 Filtration → reabsorption along a capillary

- **Arteriolar end:** Pc (~35) > πc (~25) → net **filtration** (fluid out).
- **Venular end:** Pc (~15) < πc (~25) → net **reabsorption** (fluid back in).
- **Crossover ~midway;** filtration slightly > reabsorption overall → small **net filtrate** leaves the vasculature — picked up by the lymph.
- **Trap:** textbooks that draw "reabsorption = filtration" are wrong. If they matched, the lymphatics would have no job. The imbalance is on purpose.

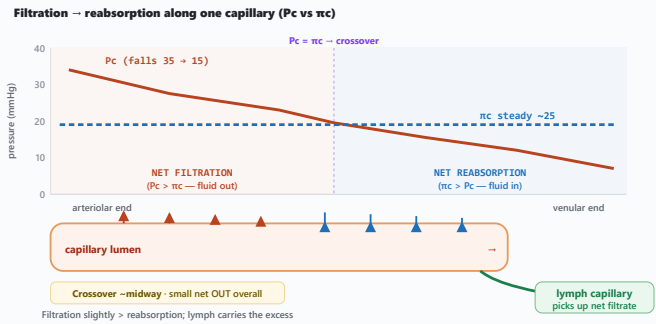

Fig 3 — Along one capillary: Pc falls 35 → 15, πc steady ~25; net filtration at the arteriolar end, net reabsorption at the venular end.

06 Why albumin is king

- **Plasma proteins ~7 g/dL**; albumin dominates πc (~25 mmHg). Even a modest drop in albumin drops πc disproportionately → oedema.
- **Threshold for oedema:** albumin < ~15–20 g/L in dogs — think PLE (protein-losing enteropathy), PLN (protein-losing nephropathy), liver failure, severe starvation, burns.
- **Reflection coefficient σ** falls in inflammation/sepsis → protein leaks with fluid → protein-rich oedema (exudate); σ = 1 in normal wall = protein stays in.

07 The lymphatic safety net

- **Blind-ended lymph capillaries** in every tissue absorb the net filtrate + escaped protein; one-way valves keep it centralward.
- **Pumps that push lymph:** skeletal-muscle pump (contracting limb muscles squeeze vessels), respiratory pump (inspiration draws lymph into the thoracic duct), and rhythmic contraction of lymphangions.
- **Route:** tissue → lymph capillary → lymph node (filter + immune surveillance) → thoracic duct → subclavian vein → blood.
- **Failure = lymphoedema** (post-op axillary node removal, congenital hypoplasia, filariasis).

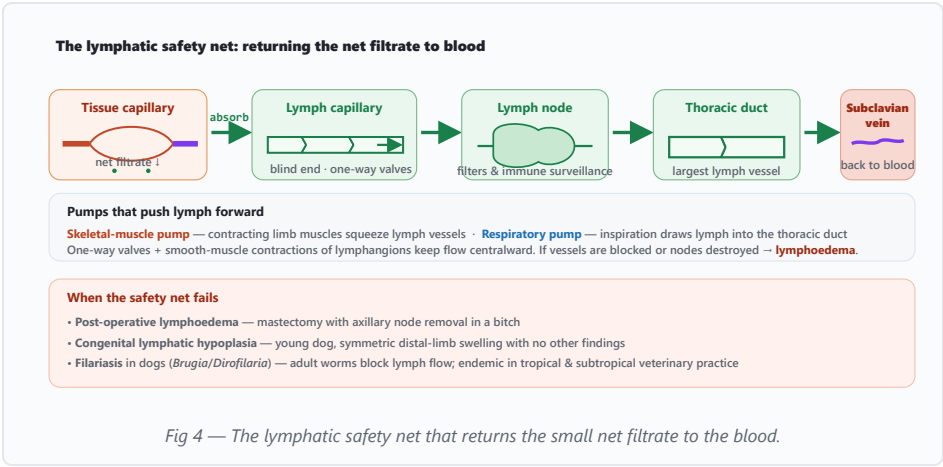

Fig 4 — The lymphatic safety net that returns the small net filtrate to the blood.

08 The 4 mechanisms of oedema

- **1 ↑ Pc** (raised capillary hydrostatic) — heart failure, venous obstruction, over-transfusion. **Vet:** Cavalier KCS with mitral regurgitation → LHF → pulmonary oedema. Fluid = transudate (wall intact).
- **2 ↓ πc** (low plasma oncotic) — hypoalbuminaemia (PLE, PLN, liver, starvation). **Vet:** Yorkie with PLE → ascites + peripheral pitting oedema. Fluid = transudate.

- **3 ↑ permeability** (leaky wall — σ falls) — inflammation, sepsis, burns, allergy, envenomation. **Vet:** septic dog → generalised protein-rich oedema; snake bite → local swelling. Fluid = exudate.
- **4 Lymphatic block** — surgery (node removal), tumour compression, filariasis. **Vet:** post-mastectomy axillary lymphoedema in a bitch. Fluid = often protein-rich (lymph carries protein).

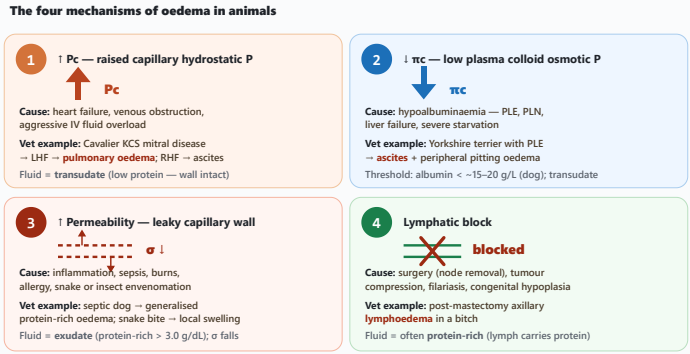

Fig 5 — The four mechanisms of oedema in animals.

09 Where oedema pools clinically

- **Pulmonary oedema (chest)** — left heart failure raises pulmonary Pc; classic Cavalier KCS with mitral disease, or DCM boxer.
- **Ascites (abdomen)** — right heart failure, hypoalbuminaemia (PLE Yorkie, liver failure), portal hypertension.
- **Bottle jaw** (submandibular oedema) — ruminant parasitism (*Haemonchus* in sheep/goats) causing hypoproteinaemia.
- **Peripheral / lymphoedema** — lymphatic block (post-op, filariasis, congenital) or systemic venous congestion.

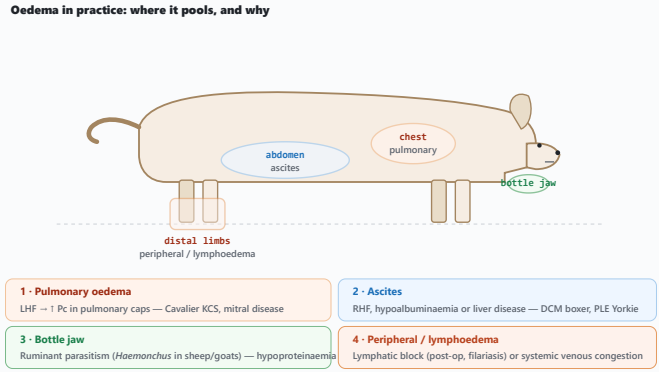

Fig 6 — Where oedema pools in animals: pulmonary, ascites, bottle jaw, peripheral / lymphoedema.

10 Why the lungs are (usually) protected

- **Cunningham defines 3 safety factors** that limit oedema in ANY tissue — all activated by increased interstitial fluid volume.
- **Safety factor 1 — rising Pi opposes filtration.** As fluid accumulates, Pi rises from ~−7 mmHg toward zero, reducing the (Pc − Pi) driving force.
- **Safety factor 2 — rising Pi promotes lymph flow.** Pulmonary lymph flow can rise ~**10×** baseline before it is overwhelmed.
- **Safety factor 3 — lymph flow washes down πi.** Extra lymph flow flushes protein out of the interstitium, keeping the (πc − πi) gradient large.
- **Lung advantage 1** — a well-developed pulmonary lymphatic supply (breathing helps propel lymph).
- **Lung advantage 2 — tight alveolar epithelium.** Fluid crossing into the interstitium does not readily reach the air spaces — alveolar flooding is a *late* event.
- **Caveat (Cunningham p248):** the pulmonary Pc is ~12 mmHg vs ~18 systemic (a little lower), but pulmonary πi is ~18 mmHg vs ~1 systemic (much higher, because the lung wall is a little more permeable to protein). Net: lung NFP = +9 mmHg (higher filtration force than systemic +1). The lung is not spared by low filtration — it is spared by the safety factors + tight epithelium.
- **When these fail:** LHF pushing pulmonary Pc past ~25 mmHg → cardiogenic pulmonary oedema. Sepsis/ARDS → permeability oedema.

11 Clinical reasoning & the fluid-therapy caution

- **Pattern-match:** location + protein content of the fluid. Transudate (low protein) = ↑ Pc or ↓ πc; exudate (protein-rich > 3 g/dL) = ↑ permeability or lymphatic.

- **Treat the cause, not the sign:** heart failure → diuretic + preload reduction; hypoalbuminaemia → treat underlying + fluids sparingly (colloids if severe); inflammation → treat the root; lymphatic → drainage/support.
- **Fluid-therapy trap:** don't over-transfuse a hypoalbuminaemic patient with crystalloids — you push more Pc against a lower πc and worsen the oedema.

RED FLAG

Rapidly progressive pulmonary oedema in a small-breed dog with a left-apical systolic murmur — mitral disease → LHF. IV furosemide, oxygen, referral for echocardiography.

> *Every case of oedema is a story of one microscopic vessel gone wrong — four Starling forces, one balance, one of four ways to break it.*
>
> — after Cunningham 6e Ch23 & PDA 3e Ch11.

KEY TERMS — QUICK GLOSSARY

Oedema (edema)	Clinically noticeable excess interstitial fluid — the sign, not the disease.
Capillary	The smallest blood vessel; where exchange between blood and tissue occurs. Three wall types by tissue.
Endothelium	The single-cell layer lining every capillary; its junctions and vesicles set permeability.
Continuous capillary	Tight endothelium, small clefts — muscle, skin, lung, brain (tightest of all).
Fenestrated capillary	Endothelium with pores 40–80 nm — kidney glomerulus, gut, endocrine glands.
Discontinuous capillary	Wide gaps large enough for cells to cross — liver sinusoids, spleen, bone marrow.
Bulk flow	Movement of water plus dissolved small solutes through pores driven by hydrostatic pressure.
Pc (capillary hydrostatic pressure)	Blood pressure inside the capillary pushing fluid out — ~18 mmHg mean (Cunningham); ~35 arteriolar, ~15 venular.
Pi (interstitial hydrostatic pressure)	Pressure in the interstitial space — slightly negative (~−7 mmHg) in most loose tissues.
πc (plasma colloid osmotic pressure)	Osmotic pull of plasma proteins on water — ~25 mmHg; ~65% from albumin.
πi (interstitial colloid osmotic pressure)	Osmotic pull of the small amount of protein in the interstitium — ~1 mmHg in most systemic tissues (per Cunningham); ~18 mmHg in the lung.
Reflection coefficient (σ)	How impermeable the wall is to protein (1 = fully reflecting, 0 = freely leaking). Falls in inflammation and sepsis.

Starling equation	NFP = (Pc − Pi) − σ(πc − πi). Positive = filtration; negative = reabsorption.
NFP (net filtration pressure)	The Starling-equation resultant — a few mmHg outward on the arteriolar end, inward on the venular end.
Filtration	Fluid leaving the capillary lumen for the interstitium under net outward Starling force.
Reabsorption	Fluid returning from the interstitium into the capillary lumen under net inward Starling force.
Lymphatic capillary	Blind-ended, valved endothelial tube that drains the small net filtrate — returns it via the thoracic duct to blood.
Lymphoedema	Oedema from lymphatic failure — post-operative, congenital, filariasis. Protein-rich.
Transudate	Low-protein oedema fluid (~<1.5 g/dL total protein, SG <1.017) — typical of ↑Pc and ↓πc.
Exudate	Protein-rich oedema fluid (>3.0 g/dL) — typical of ↑permeability (inflammation, sepsis).
Ascites	Free fluid in the peritoneal cavity — right heart failure, hypoalbuminaemia, liver disease.
Bottle jaw	Submandibular oedema from severe hypoproteinaemia in ruminants — classic *Haemonchus* infestation.
Pulmonary oedema	Fluid in the pulmonary interstitium and alveoli — cardiogenic (LHF) or non-cardiogenic (ARDS).
Hypoalbuminaemia	Low plasma albumin (<20 g/L in dogs) — from PLE, PLN, liver failure or severe starvation.

QUICK REVISION — REMEMBER THESE

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.

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**.

MEMORY AIDS

PILL — the four causes of oedema — **P**ressure up (Pc ↑ — heart failure), **I**nsufficient albumin (πc ↓ — PLE/PLN/liver), **L**eaky wall (↑ permeability — inflammation/sepsis), **L**ymph blocked (lymphatics fail — lymphoedema).

Starling forces: OUT vs IN — **Out of the capillary**: Pc (blood pressure) + πi (tissue oncotic). **Into the capillary**: πc (plasma oncotic) − Pi (interstitial pressure). Net a few mmHg out at the arteriolar end, in at the venular end.

Albumin is king — **Al**bumin makes **most** of the ~25 mmHg πc. Drop it → fluid drops *out*. Watch albumin whenever you see oedema.

Where oedema pools = which mechanism — **Lungs** = left heart. **Belly** (ascites) = right heart, hypoalbuminaemia or liver. **Jaw** (bottle jaw) = parasite protein-loss in ruminants. **Limb** = lymphatic block.

TEST YOURSELF — ACTIVE RECALL

Cover the answers and try to retrieve each one from memory first — self-testing beats re-reading.

1. **What is oedema in animals, and what causes it in general terms?**
2. **Name the four Starling forces, give typical resting values, and state the direction of each.**
3. **Write the Starling equation and explain each term.**
4. **Name the four mechanisms of oedema in animals and give a vet example of each.**

5. **Why does hypoalbuminaemia cause oedema, and at what plasma albumin does it usually appear?**
6. **What are the safety factors against pulmonary oedema, and how does left heart failure overwhelm them?**
7. **How is a transudate different from an exudate, and why does the difference matter clinically?**
8. **Why is a hypoalbuminaemic dog at risk from aggressive IV fluid therapy?**
9. **What is bottle jaw and what causes it?**

ANSWERS

1. Oedema is **clinically noticeable excess interstitial fluid**. In general terms it means the balance between filtration out of the capillary and reabsorption back in (plus lymphatic drainage) has tipped so that fluid accumulates faster than it can be cleared. Any of four things can cause it: raised Pc, lowered πc, raised permeability, or lymphatic block.
2. In a resting systemic capillary of a dog: **Pc** ~18 mmHg on average (~35 at the arteriolar end, ~15 at the venular end) pushing fluid *out*; **Pi** ~−7 mmHg (slightly negative in loose tissue) — a negative Pi actually pulls fluid *out* of the capillary; **πc** ~25 mmHg pulling water *in*; **πi** ~1 mmHg pulling water *out* (a small effect; the lung is the exception, at ~18 mmHg).
3. **NFP = (Pc − Pi) − σ(πc − πi)**. Pc drives fluid out, Pi (subtracted) is the interstitial pressure, πc pulls fluid back in, πi is the interstitial osmotic pull, and **σ** is the reflection coefficient of the wall to protein (1 = fully reflecting, 0 = freely permeable). A positive NFP means net filtration; negative means net reabsorption.
4. **(1)** ↑ **Pc** — left heart failure in a Cavalier with mitral regurgitation causing pulmonary oedema. **(2)** ↓ **πc** — a Yorkshire terrier with protein-losing enteropathy developing ascites. **(3)** ↑ **permeability** — a dog with sepsis or a snake bite developing local or generalised protein-rich oedema. **(4) Lymphatic block** — a dog with post-operative axillary lymphoedema after mastectomy.
5. Albumin generates most of the ~25 mmHg plasma colloid osmotic pressure. When albumin falls, πc falls with it, so at the venular end the inward pull is no longer strong enough to reabsorb the fluid that filtered out at the arteriolar end. The lymphatics compensate up to a point, but once plasma albumin drops below about **15–20 g/L** in dogs oedema (ascites, pleural effusion, peripheral) appears.
6. Cunningham describes **three** safety factors that limit oedema in ANY tissue — all activated by increased interstitial fluid volume: (1) **rising Pi** directly opposes further filtration; (2) rising Pi **promotes lymph flow** (up to ~10× baseline in the lung); (3) the extra lymph **washes down πi**, keeping the πc−πi gradient large. The lung adds two of its own: a well-developed pulmonary lymphatic supply and a **tight alveolar epithelium** that keeps interstitial fluid out of the air spaces. Left heart failure raises left-atrial pressure, which back-pressures the pulmonary capillaries; once pulmonary Pc exceeds ~25 mmHg the safety factors are overrun and cardiogenic pulmonary oedema appears.
7. A **transudate** is low-protein (total protein <1.5 g/dL, SG <1.017) — typical of raised Pc (heart failure) or lowered πc (hypoalbuminaemia), where the wall stays intact and the fluid squeezed out is essentially protein-free. An **exudate** is protein-rich (>3.0 g/dL) — typical of raised permeability (inflammation, sepsis, snake bite), where σ falls and protein leaks with the fluid. Cytology on abdominocentesis or thoracocentesis narrows the differential immediately.
8. Aggressive fluids raise Pc without raising πc (colloid osmotic pressure). In a hypoalbuminaemic animal πc is already low, so any further widening of the Pc−πc gap drives more fluid straight into the interstitium and can precipitate pulmonary or peripheral oedema. Fluid these patients cautiously and consider colloid support if truly required.
9. **Bottle jaw** is soft, pitting **submandibular oedema** under the jaw of ruminants. It is the classic sign of severe hypoproteinaemia from chronic *Haemonchus contortus* infestation in sheep and goats (and calves) — the abomasal blood-sucking parasite drops plasma protein until πc cannot hold fluid in the capillary.

WHEN TO REFER OR ESCALATE

- 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. Chapter 23 'Capillaries and Fluid Exchange' (pp 241–252): capillaries as the anatomical site of exchange, and the three wall types (continuous / fenestrated / discontinuous) mapped to tissue function; the four routes of transcapillary movement (lipid-soluble diffusion, small-molecule diffusion through pores, pinocytosis, bulk flow) and Fick's law; the four Starling forces (Pc, Pi, πc, πi) with typical values (Pc ~18 mmHg mean, ~35 arteriolar, ~15 venular; Pi ~−7 in loose tissue; πc ~25; πi ~1 systemic / ~18 in the lung) and the Starling equation NFP = (Pc − Pi) − σ(πc − πi); net filtration at the arteriolar end and net reabsorption at the venular end, with a small net filtrate returned by the lymphatics; the lymphatic safety net with its ~10× reserve in the lung; the four causes of oedema (↑Pc, ↓πc, ↑permeability, lymphatic block); oedema as clinically noticeable excess interstitial fluid; the three general safety factors against oedema (rising Pi opposes filtration, rising Pi promotes lymph flow, lymph flow washes down πi) plus the lung-specific advantages (rich pulmonary lymphatics and a tight alveolar epithelium).
2. Sjaastad ØV, Sand O, Hove K. Physiology of Domestic Animals. 3rd ed. Oslo: Scandinavian Veterinary Press; 2016. Chapter 11 'The Cardiovascular System' (pp 482–494): capillary microcirculation and fluid exchange; the four Starling forces and their action along the capillary; the balance between filtration and reabsorption; the role of the lymphatic system in returning net filtrate and escaped protein; hypoalbuminaemia and inflammation as pathological drivers of oedema; species differences in lymphatic anatomy and capillary permeability; the practical bedside consequences for cardiovascular medicine.
3. Ettinger SJ, Feldman EC, Côté E. Textbook of Veterinary Internal Medicine. 8th/9th ed. St Louis: Elsevier; 2017–2024. Chapters on canine and feline degenerative valvular disease, dilated cardiomyopathy, congestive heart failure, protein-losing enteropathy, protein-losing nephropathy and hepatic disease — source material for the clinical reasoning H2 (pulmonary oedema thresholds, cardiac vs hypoalbuminaemic ascites, fluid-therapy caution) and for the vet-example animals used throughout.

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