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RUMEN PHYSIOLOGY

# How the Rumen Works: Fermentation, VFAs and Rumination in Cattle, Sheep and Goats (PDF)

Rumen physiology in cattle, sheep and goats is the story of a giant fermentation vat that turns grass — the cellulose no mammal can digest alone — into energy, milk and meat. Inside: Nine labelled figures, one reference table and a 17-question self-test.

# Contents

The 11 sections of this guide, in order.



IN SHORT

**Rumen physiology in cattle**, sheep and goats is the story of a giant **fermentation vat** that turns grass — the cellulose no mammal can digest alone — into energy, milk and meat. The **four-compartment stomach** (reticulum, rumen, omasum, abomasum) hands feed to a dense **rumen microbiome** of bacteria, protozoa and fungi that own the enzymes the animal lacks. They ferment carbohydrate into **volatile fatty acids (VFAs)** — **acetate**, **propionate** and **butyrate** — plus $CO_2$, methane and heat. **Rumination** (chewing the cud) and 100–190 L/day of buffered saliva keep the vat mixed and its pH near 6.0–6.8. Understand this and you understand **ruminant digestion**, why **propionate** makes a dairy cow's glucose, and how the system fails in **subacute ruminal acidosis (SARA)**, **rumen bloat** and milk-fat depression.

KEY POINTS

1. **Rumen physiology in cattle**, sheep and goats explains the single biggest trick in veterinary nutrition: how a herbivore lives on grass. Mammals make no enzyme that can break the β-1,4 bonds of **cellulose**. The ruminant solves this by outsourcing digestion to a vast microbial culture housed in a fermentation vat — the reticulorumen — and then digesting the microbes. This is **ruminant digestion**: fibre in, volatile fatty acids, microbial protein, milk and meat out.
2. The ruminant has a **four-compartment stomach**: **reticulum, rumen, omasum and abomasum**. The first three form the *forestomach* and make no acid or enzymes of their own; only the **abomasum** is the 'true' stomach, secreting hydrochloric acid and pepsin like a dog's or a cat's. The rumen alone fills roughly 75% of the abdominal cavity in an adult cow.
3. Feed is handled by **rumination** — chewing the cud. The animal swallows coarse feed, the reticulorumen mixes it with rhythmic contractions, a bolus is regurgitated, re-chewed to grind the fibre finer and soak it in saliva, then re-swallowed. A dairy cow secretes **100–190 litres of saliva a day**, rich in bicarbonate and phosphate, which buffers the acids the microbes make. Fermentation gas is expelled by **eructation** (belching).
4. The **rumen microbiome** is the workforce. Densities reach **~$10^{10}$–$10^{11}$ organisms per millilitre** of rumen fluid: anaerobic **bacteria** (the bulk of the fermentation), **protozoa** (ciliates that graze bacteria and store starch) and anaerobic **fungi** (which attack tough plant fibre). Between them they carry the **cellulase** and other enzymes the host genome does not encode. The relationship is a true symbiosis.
5. Fermentation converts plant carbohydrate into **volatile fatty acids (VFAs)** plus gases and microbial cells. For every mouthful of fermentable carbohydrate the microbes release **VFAs** (the animal's main energy currency), **carbon dioxide** and **methane ($CH_4$)**, **heat**, and new microbial biomass. A cow produces **30–50 litres of fermentation gas an hour**, most of it belched away.
6. The three major VFAs are **acetate, propionate and butyrate**, produced on a fibre diet in a ratio of roughly **70 : 20 : 10**. **Acetate** is used for energy and is the main precursor of **milk fat**. **Propionate** is the *only glucogenic VFA*: it feeds **gluconeogenesis** in the liver to make glucose, which supplies 65–80% of a dairy cow's glucose and thus milk lactose. **Butyrate** is converted to ketones and fuels the rumen epithelium itself.

7. VFAs are **absorbed directly across the rumen wall**, not passed downstream first. The wall is carpeted with finger-like **papillae** that hugely increase surface area; VFAs cross mostly in their protonated (uncharged) acid form. Together with the bicarbonate delivered in saliva, this absorption keeps the healthy **rumen pH near 6.0–6.8**. When acid production outruns buffering and absorption, pH falls and disease follows.
8. Ruminants make high-quality protein from cheap nitrogen. Microbes build **microbial protein** from ammonia, and that ammonia can come from dietary protein *or* from **non-protein nitrogen (NPN)** such as **urea**. The cow then digests the microbes downstream in the abomasum. This is why urea can be fed to cattle — but overfeeding NPN releases ammonia faster than the microbes can use it, and ammonia toxicity can kill.
9. The rumen is **made, not inherited**. A newborn calf, lamb or kid is a functional *monogastric*: milk must bypass the undeveloped rumen. The **oesophageal (reticular) groove** closes reflexly during suckling and channels milk straight to the abomasum. As the young animal starts eating solids, **butyrate** drives growth of the rumen papillae and the microbial population establishes, so that by weaning the animal is a fully functional ruminant.
10. When the system fails, the failures are classic and examinable. **Subacute ruminal acidosis (SARA)** follows grain overload (rumen pH < 5.5, lactate accumulation). **Rumen bloat** is failure of eructation — frothy (legume) or free-gas — distending the left flank and compressing the lungs. **Left displaced abomasum (LDA)** follows gas and atony in the fresh cow. **Milk-fat depression** follows a low-fibre ration that shifts the VFA ratio away from acetate. Every one of these is read straight off the physiology above.

## 01 Why the rumen matters in veterinary medicine

Every dairy cow, feedlot steer, ewe and dairy goat you will ever treat runs on a trick no dog, cat or human can perform: it lives on grass. Grass is mostly **cellulose**, and no mammal makes an enzyme that can break the β-1,4 bonds that hold cellulose together. This is the central puzzle that **rumen physiology in cattle**, sheep and goats solves. Instead of digesting fibre itself, the ruminant keeps a vast, permanent culture of microbes in a warm, churning fermentation vat — the rumen — lets them do the digesting, and then lives off what they produce. Grass goes in; energy, protein, milk and meat come out. The animal is, in effect, a mobile fermenter with legs.

That is why the rumen is the single most important organ in farm-animal medicine. Get its physiology and you can explain almost everything that goes wrong in ruminant practice: the feedlot steer that founders after a night in the grain store, the bullock that bloats on lush clover, the fresh dairy cow that goes off her feed with a displaced abomasum, the herd whose milk fat suddenly crashes. Each of these is a failure of one specific step in a chain we can follow from first principles. The chain is called **ruminant digestion**, and it starts with fermentation: the microbes ferment the feed into **volatile fatty acids (VFAs)**, which the animal absorbs and burns for energy, turns into milk and lays down as meat. The VFAs are the whole point — they are why a cow can thrive on a diet that would starve a dog.

This article walks the whole system from the ground up, exactly as your textbook does: the four-compartment stomach, how feed moves and rumination works, the microbiome, fermentation, the three VFAs and why propionate is king, VFA absorption and pH, protein from urea, how the rumen develops in the calf, and how it fails in disease. Along the way it links to two companion pieces. The energy the VFAs carry is ultimately spent at the level of the cell, which is covered in our article on cellular homeostasis. And when heavy parasitism or protein loss drops a ruminant's plasma proteins, the fluid shifts that follow — the 'bottle jaw' of the wormy sheep — are explained in our article on oedema and the Starling forces. Both are worth reading alongside this one. The primary source throughout is Cunningham's Ch31 ('Digestion: The Fermentative Process', pp339–360), supported by *Physiology of Domestic Animals* Ch15.

## 02 The four-compartment stomach: reticulum, rumen, omasum, abomasum

The ruminant does not have four stomachs; it has one stomach with **four compartments**, arranged in series. In the order that feed passes through them they are the **reticulum**, the **rumen**, the **omasum** and the **abomasum**. The first three are called the *forestomach*, and the single most important fact to fix in your mind is this: **the forestomach makes no acid and secretes no digestive enzymes of its own**. All of the digestion that happens there is done by microbes. Only the fourth compartment, the **abomasum**, is a 'true' stomach in the sense a dog or cat owner would recognise — it secretes hydrochloric acid and pepsin and digests food (and microbes) chemically.

The **reticulum** is the most cranial compartment, sitting just behind the diaphragm. Its lining is folded into a distinctive honeycomb pattern, and functionally it works as one unit with the rumen — together they are often called the **reticulorumen**. Because it is the lowest point of the forestomach, the reticulum tends to catch heavy or sharp swallowed objects; a swallowed nail or piece of wire settles here, which is why 'hardware disease' (traumatic reticuloperitonitis) begins in the reticulum. The reticulum also generates the contraction waves that drive mixing and rumination.

The **rumen** is the star of the show and by far the largest compartment. In an adult cow it fills roughly **75% of the abdominal cavity** and can hold well over 100 litres of fermenting digesta. It is the fermentation vat: a warm (~39°C), anaerobic, continuously mixed chamber that houses the entire microbial population. Its wall is carpeted with finger-like **papillae** that vastly increase its surface area for absorbing the VFAs the microbes produce. When a clinician talks about 'the rumen' doing the digesting, this is the compartment they mean.

The **omasum** is a curious, tightly packed compartment whose lining is folded into dozens of leaves or plies (hence its country names, the 'book' or 'many-plies'). Digesta is squeezed between these leaves, and the omasum's main job is to **absorb water and residual VFAs**, concentrating the digesta before it passes on. The **abomasum**, finally, is the true glandular stomach. It secretes hydrochloric acid and pepsin exactly as a monogastric stomach does, dropping the pH sharply and beginning enzymatic protein digestion — digesting both the feed residue and, crucially, the enormous crop of microbes flowing out of the forestomach. Those microbes are the animal's main protein supply, a point we return to below.

**Callout — the trap that catches every student.** The rumen has no digestive enzymes of its own — the microbes own them all. When you read that a cow 'digests cellulose', remember she does not: her microbes do, and she digests the microbes. The only compartment that behaves like a dog's or cat's stomach is the abomasum.

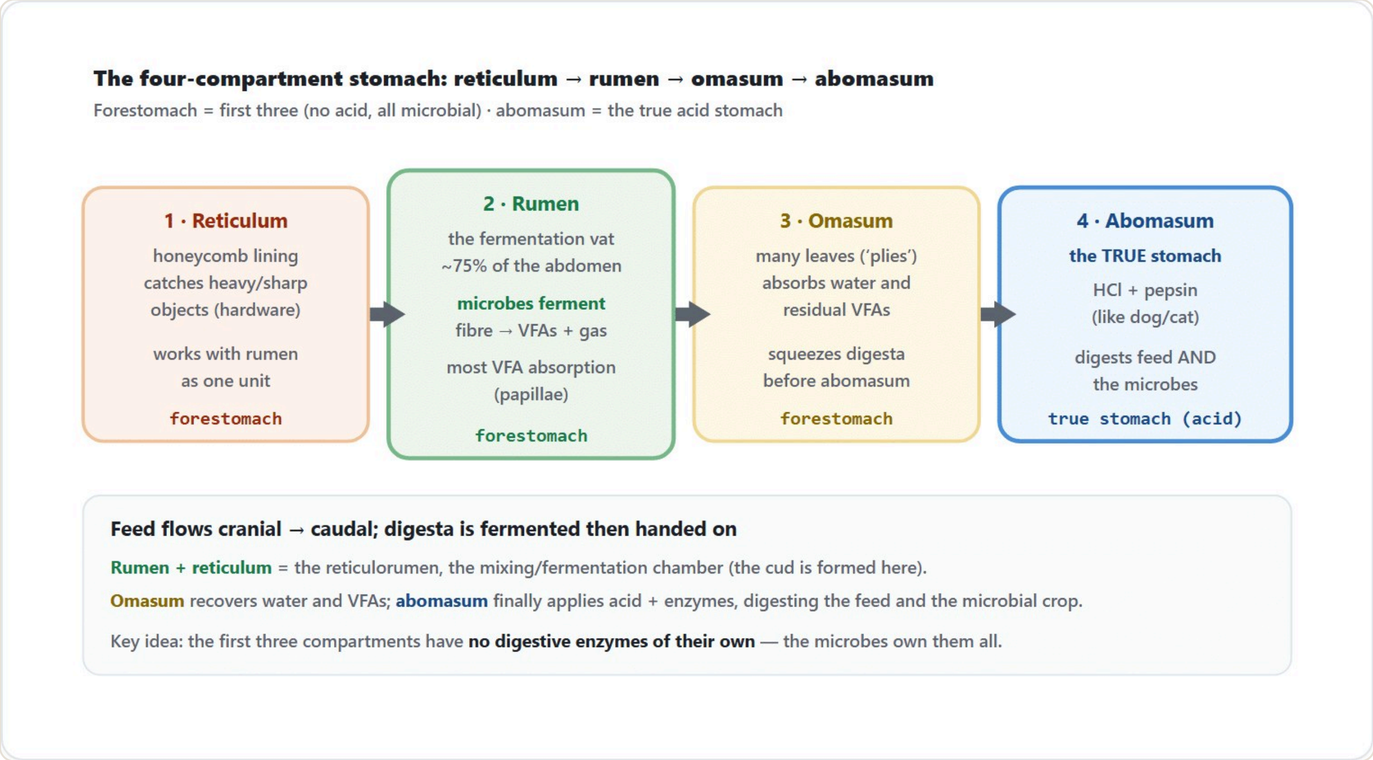
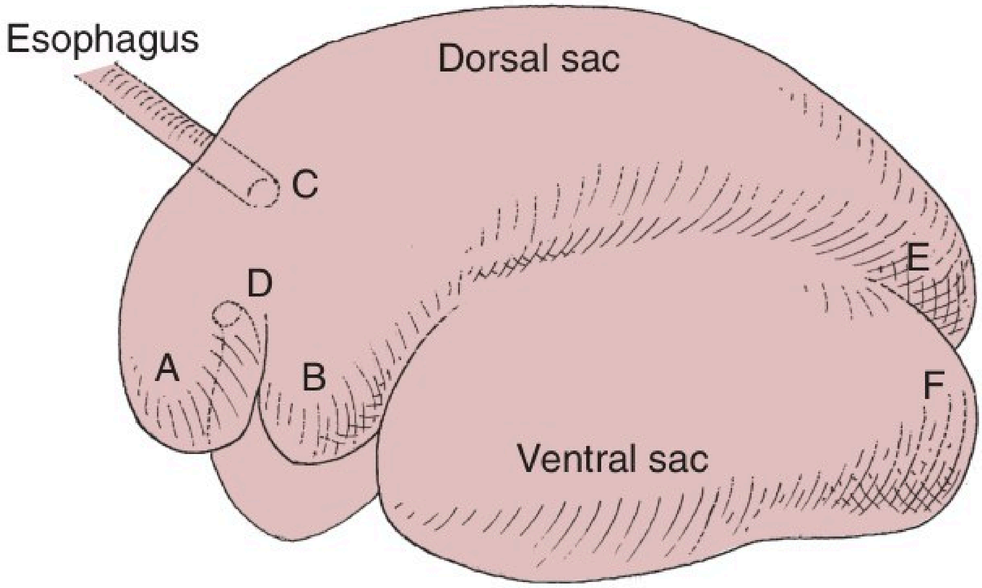



Fig 1 — The four-compartment stomach. Feed flows reticulum → rumen → omasum → abomasum. The first three (forestomach) make no acid and no host enzymes; the rumen (~75% of the abdomen) is the fermentation vat; the abomasum is the true acid stomach.




• **Fig. 31.7** Rumen anatomy. (A) Reticulum; (B) cranial sac; (C) cardia; (D) reticulo-omasal orifice; (E) caudal-dorsal blind sac; (F) caudal-ventral blind sac.

Fig 1a — The real anatomy of the reticulorumen. The huge rumen is divided into **dorsal** and **ventral sacs** by muscular pillars; the small **reticulum** (A) sits cranially at the **cardia** (C) where the oesophagus enters, and digesta exits to the omasum through the **reticulo-omasal orifice** (D).

Source: Cunningham 6e Ch31 Fig 31.7, p 347.

## 03 How feed moves and rumination works (chewing the cud)

A grazing cow does not chew her food properly the first time. She tears off grass, gives it a few cursory chews and swallows it, coarse and barely broken, into the reticulorumen. The real chewing happens later, during **rumination** — chewing the cud — and this is one of the defining behaviours of a healthy ruminant. A cow spends roughly **eight hours a day** ruminating, and a cow that stops chewing her cud is a cow that is unwell.

The cycle runs like this. Coarse feed is **swallowed** into the reticulorumen, where rhythmic **contractions** mix and stratify the contents: gas rises to the top, fibrous mat floats in the middle, and fluid with fine particles settles below. Every so often the reticulum contracts in a way that carries a bolus of coarse material back up the oesophagus — the animal **regurgitates** it into the mouth. She then **re-chews** this cud thoroughly, grinding the fibre far finer and soaking it in saliva, before **re-swallowing** it. The finer, wetter material now ferments faster and eventually passes on; the coarse material that is not yet fine enough stays behind for another cycle. It is a beautifully simple sorting-and-grinding loop.

The contractions themselves come in two overlapping patterns. **Primary (mixing) contractions** sweep through the reticulorumen roughly once a minute, mixing digesta and pushing it toward the omasum. **Secondary contractions** are specifically concerned with moving the gas cap toward the cardia so it can be belched out — the process called **eructation**. Eructation matters enormously: the microbes produce 30–50 litres of gas an hour, and if the animal cannot belch it away, the rumen distends and the animal bloats (a failure mode we cover below).

None of this would work without **saliva**, and ruminants produce staggering amounts of it. A dairy cow secretes **100–190 litres of saliva a day** — far more than any monogastric of comparable size. Ruminant saliva is not mainly for lubrication; it is a **buffer**, rich in bicarbonate and phosphate. Every time the cow chews her cud she pours buffered saliva into the rumen, neutralising the acids the microbes are constantly making and helping hold the pH in the healthy range. This is why *effective fibre* in the ration matters so much: fibre stimulates chewing, chewing stimulates saliva, and saliva buffers the rumen. Cut the fibre and you cut the saliva, and the rumen turns acid. Cunningham (Ch31 Figs 31.8–31.9) sets out this contraction-and-flow sequence in detail; *Physiology of Domestic Animals* Ch15 reinforces the buffering role of saliva across species.

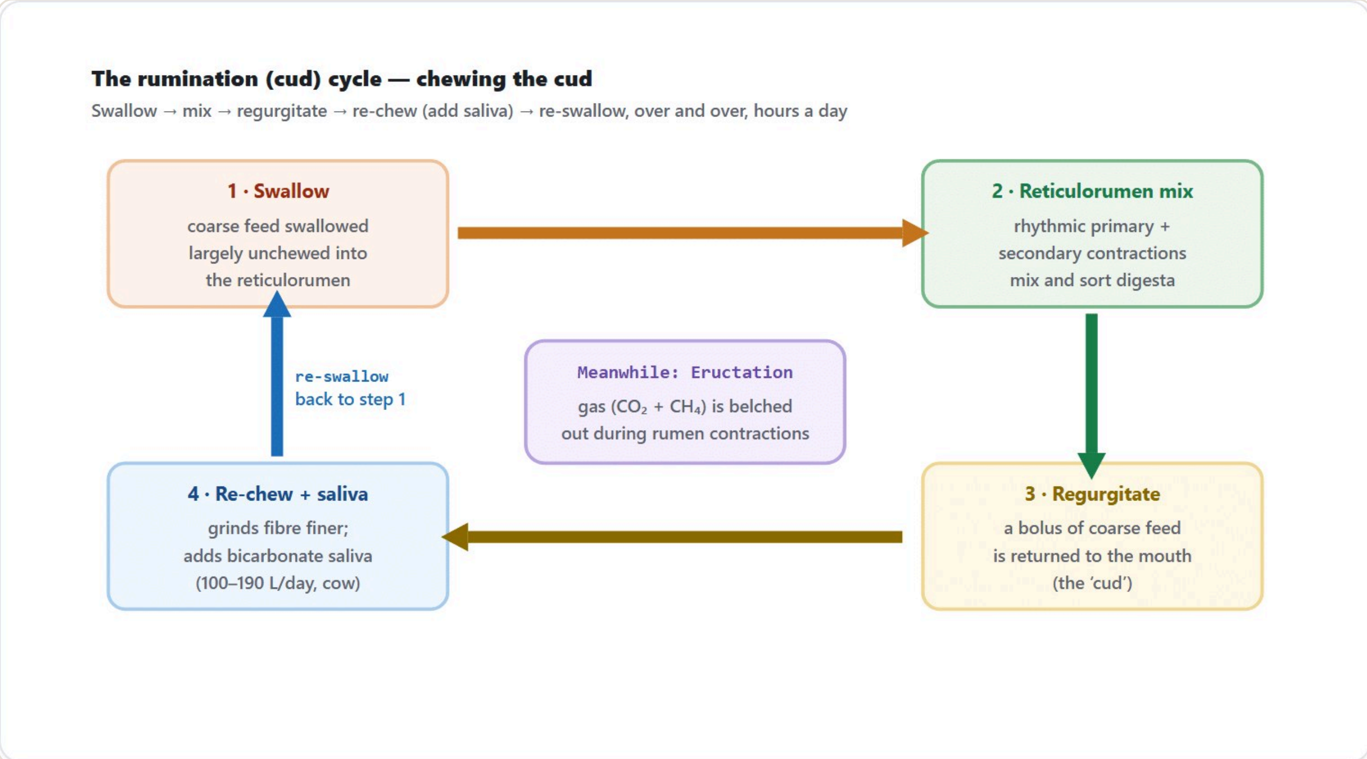
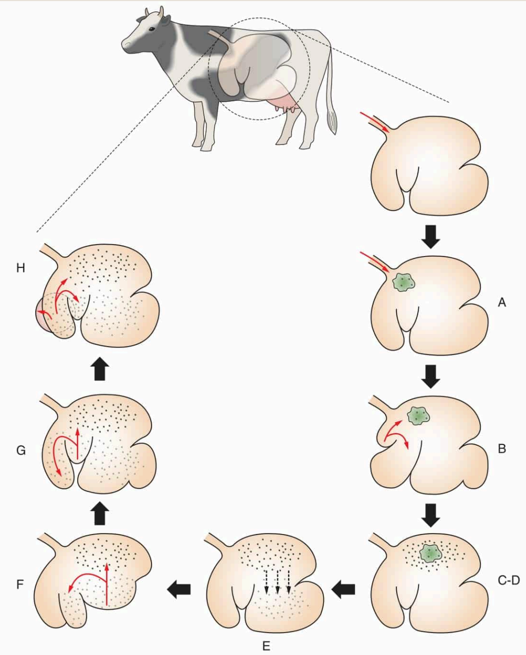



Fig 2 — The rumination (cud) cycle. Swallow coarse feed → reticulorumen mixing → regurgitate a bolus → re-chew with buffering saliva → re-swallow. Eructation runs alongside, belching $CO_2$ and methane. Saliva 100–190 L/day (cow) delivers the bicarbonate buffer.




• **Fig. 31.9** The sequential figures illustrate the pattern of flow of feed material through the reticulorumen from its arrival at the cardia (*A*) to its exit via the reticulo-omasal orifice (*H*). Details of the sequential flow pattern are given in the text.

Fig 2a — How feed actually moves through the reticulorumen. The eight panels (A–H) trace one full mixing sequence — coordinated muscular contractions circulate the digesta, float the fibre mat for regurgitation, and pass fine, fermented material on to the omasum. This choreography is what rumination and eructation depend on.

Source: Cunningham 6e Ch31 Fig 31.9, p 350.

## 04 The rumen microbiome: bacteria, protozoa and fungi

The **rumen microbiome** is the workforce that makes the whole system possible, and its scale is hard to overstate. Every millilitre of rumen fluid contains on the order of **$10^{10}$ to $10^{11}$ microorganisms** — tens of billions per millilitre — living in a strictly **anaerobic** environment (there is essentially no oxygen in the rumen). Three broad groups share the work.

**Bacteria** are the most numerous and do the bulk of the fermentation. Different species specialise: some are fibrolytic (they attack cellulose and hemicellulose), some are amylolytic (they ferment starch), some ferment sugars, some use lactic acid, and some — the methanogens, which are actually archaea — make methane. The community is dynamic: change the diet and the balance of species shifts within days. Feed a lot of grain and the starch-fermenting and lactate-producing species bloom, which (as we will see) is how grain overload turns the rumen acid.

**Protozoa** are large ciliated single-celled organisms, visible under a low-power microscope, that graze on bacteria and engulf and store starch granules. By eating starch and holding it, protozoa actually *buffer* the rate of fermentation, smoothing out the acid production after a grain meal; their motility on a slide is also a handy clinical index of rumen health. **Anaerobic fungi** are the third group; their filaments physically penetrate and weaken tough, lignified plant fibre, opening it up for the bacteria to finish. Together these three groups carry the **cellulase** and the whole armoury of fibre- and starch-digesting enzymes that the ruminant's own genome does not encode.

The relationship is a genuine **symbiosis**. The animal gives the microbes a perfect home: a warm, anaerobic, continuously mixed chamber, a steady supply of feed, and buffered saliva to keep the pH tolerable. In return the microbes digest the fibre the animal cannot, produce VFAs the animal absorbs for energy, synthesise microbial protein and even manufacture B-vitamins (thiamine, niacin and others) and vitamin K for the host. A healthy adult ruminant does not need dietary B-vitamins because its microbes make them — another fact with clinical teeth, because a rumen upset (as in acidosis) can disrupt thiamine supply and precipitate polioencephalomalacia. The lesson throughout is that in ruminant medicine you are never treating one animal; you are treating an animal and its microbial ecosystem together.

## 05 Fermentation: from cellulose to VFAs and gases

**Fermentation** is the chemical heart of **rumen physiology in cattle**, sheep and goats, and it is worth being precise about what it produces. When the microbes ferment plant carbohydrate — cellulose, starch and sugars — they do not make it into glucose for the animal to absorb. Instead they break it down anaerobically into a defined set of end-products: **volatile fatty acids (VFAs)**, the gases **carbon dioxide and methane**, **heat**, and new **microbial cells**. Follow each of these and you have the whole energy economy of the ruminant.

The **VFAs** — chiefly acetate, propionate and butyrate — are the prize. They are short-chain fatty acids, they are absorbed straight across the rumen wall, and they supply the great majority of the animal's usable energy. We devote the next two sections to them because they matter so much. The point to grasp here is that fermentation converts fibre the animal could never use into a fatty-acid fuel it can absorb and burn.

The **gases** are the unavoidable by-product. Fermentation releases large volumes of carbon dioxide, and the methanogenic archaea combine hydrogen (a fermentation intermediate) with $CO_2$ to make **methane ($CH_4$)**. This is more than a curiosity: methane production is the rumen's **hydrogen sink**. Fermentation generates hydrogen, and if hydrogen were allowed to build up it would poison the fermentation by inhibiting the enzymes that regenerate the microbes' cofactors. By mopping hydrogen up into methane, the methanogens keep fermentation running — at the cost of exhaling a potent greenhouse gas and losing 6–12% of the feed's gross energy. A cow makes **30–50 litres of gas an hour**, and virtually all of it is belched away by eructation.

The **microbial cells** are the fourth product, and they are not waste at all. As the microbes ferment carbohydrate they grow and multiply, and this new microbial biomass flows steadily out of the rumen with the digesta, to be digested downstream in the abomasum. This is the animal's main source of high-quality protein — a point important enough to have its own section below. Finally, fermentation is exothermic: it produces **heat** (the 'heat of fermentation'), which helps keep the ruminant warm and is one reason a well-fed cow tolerates cold weather. Cunningham (Ch31 Fig 31.1) lays out the fermentation pathways in full; the take-home is that one input, fermentable carbohydrate, splits into four outputs: fuel (VFAs), gas, protein and heat.

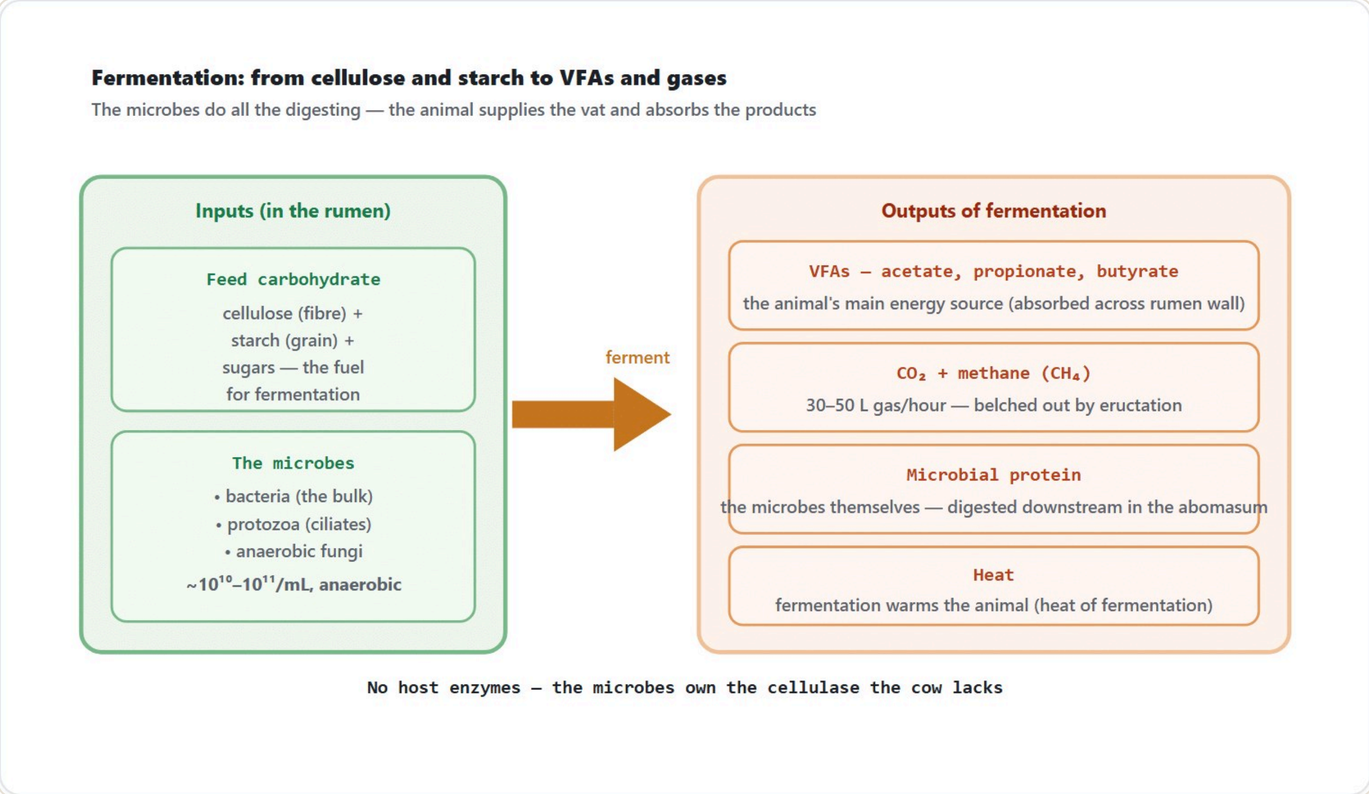



Fig 3 — Fermentation inputs and outputs. Feed carbohydrate (cellulose, starch) plus the microbes (bacteria, protozoa, fungi) ferment to VFAs (energy), $CO_2$ and methane (belched), microbial protein (digested downstream) and heat. No host enzymes are involved — the microbes own the cellulase. (Cunningham 6e Ch31 Fig 31.1, p340.)

## 06 The three VFAs: acetate, propionate and butyrate — and why propionate is king

If you learn only one thing from this article about **rumen fermentation**, learn the three **volatile fatty acids** and their fates. They are **acetate** (two carbons), **propionate** (three carbons) and **butyrate** (four carbons). On a normal fibre (forage) diet they are produced in a ratio of roughly **70 : 20 : 10** — acetate dominant. That ratio is not fixed: feed more grain and the pattern swings toward **propionate** (and total acid production rises, dropping rumen pH). The shifting ratio is the hinge on which several production diseases turn, so it is worth understanding each VFA's destiny.

**Acetate** is the most abundant VFA on a forage diet. It is absorbed and either oxidised for energy in tissues throughout the body or used as the building block for fat synthesis. Critically, acetate is the **main precursor of milk fat**: the mammary gland uses acetate (and butyrate-derived building blocks) to synthesise the fatty acids of milk. This single fact explains milk-fat depression: anything that lowers the proportion of acetate — chiefly a low-fibre, high-grain ration — starves the udder of its milk-fat precursor, and butterfat falls.

**Propionate** is the VFA that runs the dairy cow, and the reason is a piece of biochemistry worth stating plainly: **propionate is the only glucogenic VFA**. Acetate and butyrate cannot be turned into glucose, but propionate can. It is carried to the liver and fed into **gluconeogenesis**, the pathway that manufactures glucose. This matters because a ruminant absorbs almost *no* glucose from its gut — the microbes ferment any dietary sugar or starch before the animal can absorb it as glucose — so the ruminant has to *make* its glucose from scratch. Propionate supplies **65–80% of a dairy cow's glucose**, and that glucose becomes, among other things, the **lactose of milk**. A high-yielding cow is, in a real sense, glucose-limited and therefore propionate-limited: her milk output depends on how much propionate her rumen can deliver.

**Butyrate** has the smallest share of the three. As it crosses the rumen wall much of it is converted into **ketone bodies** (chiefly β-hydroxybutyrate), which the animal uses for energy. Butyrate also directly **fuels the rumen epithelium** — the wall cells preferentially burn it — and, importantly, butyrate is the main stimulus for **growth of the rumen papillae** in the developing young animal, a fact we return to when we discuss rumen development. A separate clinical note: in the fresh dairy cow in negative energy balance, excessive butyrate and body-fat mobilisation can push ketone production too far into **ketosis**.

**Callout — the exam-winning fact.** Propionate is the ONLY glucogenic VFA — a dairy cow's glucose comes from it, not from the sugar in grass. Acetate and butyrate feed energy and fat; propionate feeds glucose (and hence milk lactose). If you remember one VFA, remember why propionate is king.

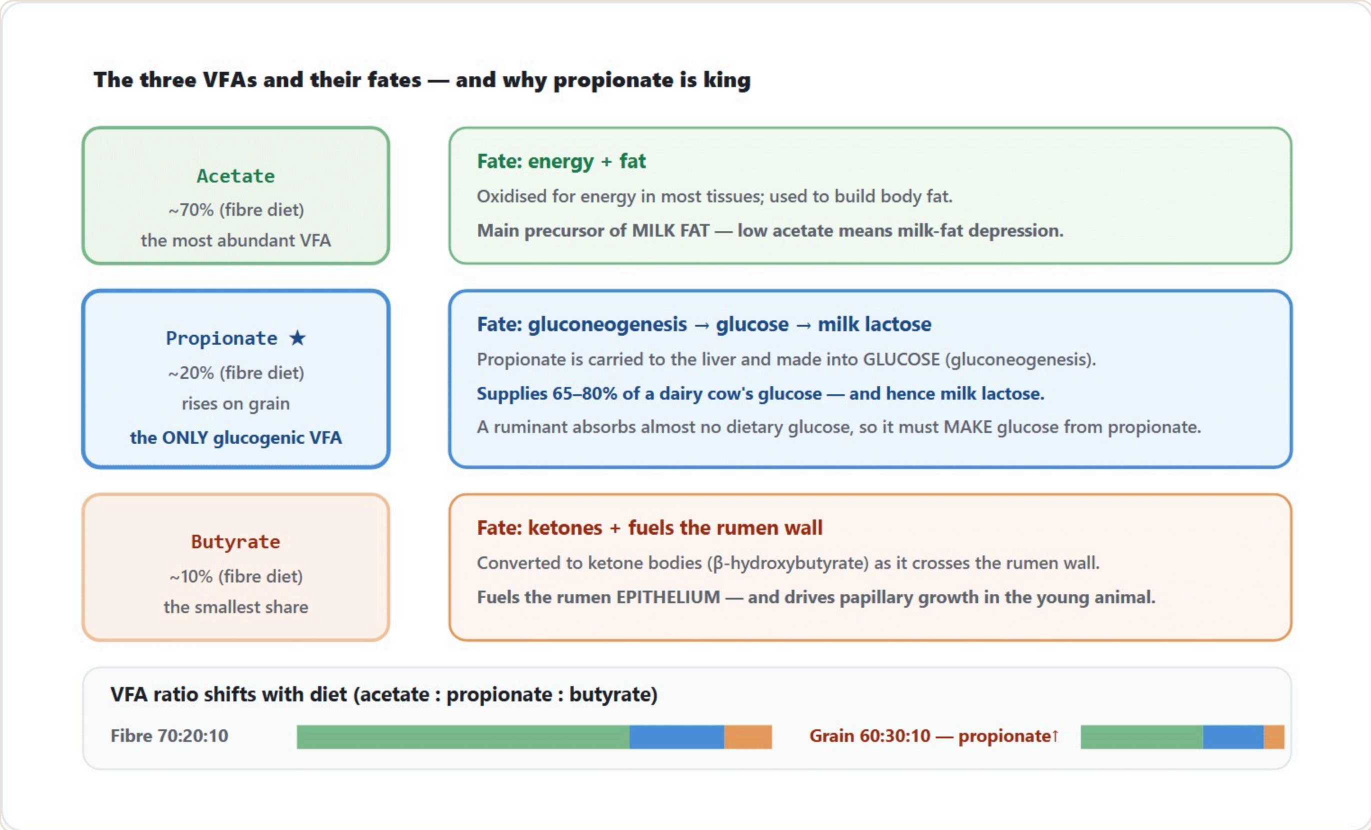



Fig 4 — The three VFAs and their fates. Acetate (~70%) → energy + milk fat; propionate (~20%) → gluconeogenesis → glucose → milk lactose (the only glucogenic VFA, 65–80% of a dairy cow's glucose); butyrate (~10%) → ketones + rumen epithelium. The ratio shifts from 70:20:10 on fibre toward propionate on grain.

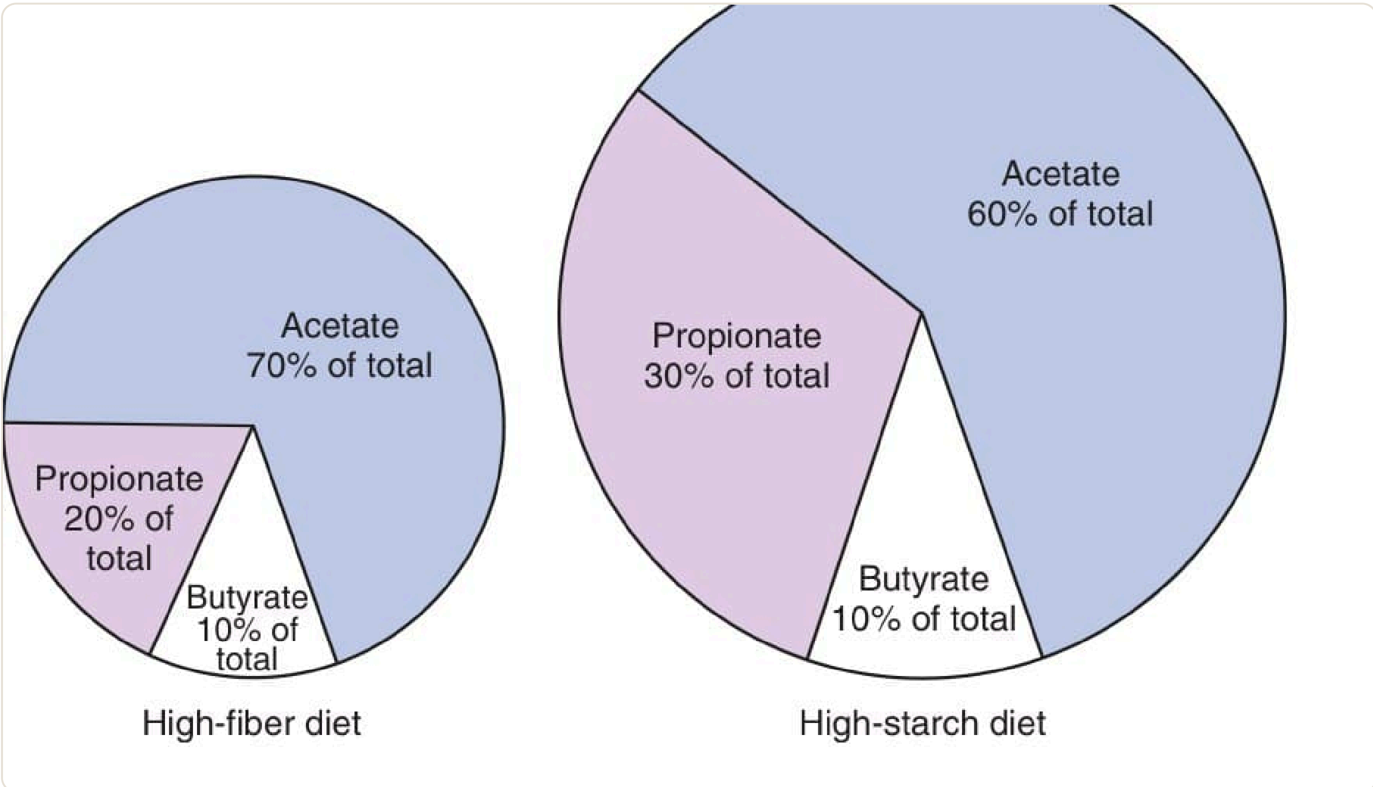



Fig 4a — Diet sets the VFA ratio. Moving from a **high-fibre** to a **high-starch (grain)** ration lifts **propionate** from ~20% to ~30% of total VFAs at the expense of acetate — more glucose (and milk yield), but a lower rumen pH and the risk of acidosis.

Source: Cunningham 6e Ch31 Fig 31.3, p 344.

The three volatile fatty acids, and why the ratio between them is the single most useful number in ruminant nutrition.

| VFA | SHARE ON A FORAGE DIET | WHERE IT GOES | WHY IT MATTERS |
|---|---|---|---|
| **Acetate** | ~70% | Oxidised for energy; the substrate for milk fat | Dominant on fibre. Drop the fibre and you drop the butterfat |
| **Propionate** | ~20% | To the liver, and into gluconeogenesis | Supplies **65–80% of a dairy cow's glucose**, which becomes the lactose of milk. Rises on grain |
| **Butyrate** | ~10% | Largely metabolised by the rumen wall itself | Feeds the papillae, and drives their development in the young ruminant |

## 07 VFA absorption and rumen pH

Producing VFAs is only half the story; the animal has to absorb them, and it does so in an elegant way. The VFAs are **absorbed directly across the rumen wall** — they do not have to travel all the way to the intestine first. The rumen wall is carpeted with countless finger-like **papillae** whose sole job is to multiply the absorptive surface area, and the more VFA a ration produces, the longer and denser those papillae grow. Most VFA crosses the wall in its **protonated (uncharged) acid form**, which diffuses across the epithelial membrane readily; the epithelium then metabolises some of it (especially butyrate) and passes the rest into the portal blood. Removing acid by absorbing it is itself one of the two main ways the rumen defends its pH.

The other way is **buffering**, and this is where the saliva comes back in. The bicarbonate and phosphate poured into the rumen with 100–190 litres of daily saliva continuously neutralise the acids the microbes make. Between them — bicarbonate buffering and papillary absorption — a healthy rumen holds its pH in a remarkably stable band of about **6.0 to 6.8**. That band is not arbitrary: the fibre-digesting bacteria work best near neutrality, so keeping the pH up keeps fibre digestion efficient. It is a self-reinforcing loop when it works well: fibre stimulates chewing, chewing makes saliva, saliva buffers acid, and the fibre-digesting microbes stay happy.

The loop breaks when acid production outruns the animal's capacity to buffer and absorb it. Feed a large slug of rapidly fermentable grain and the starch-fermenting bacteria produce VFAs fast; in an overload, lactate-producing bacteria take over and pour out **lactic acid**, which is a far stronger acid than the VFAs. The rumen pH falls. When it drops below about **5.5** for several hours a day you have **subacute ruminal acidosis (SARA)**; drop it further and you have acute lactic acidosis, a medical emergency. As the pH falls, the fibre-digesting microbes die off (they cannot tolerate acid), fibre digestion collapses, and the whole ecosystem tilts further toward acid production — a vicious cycle. Understanding the pH band, and what pushes the rumen out of it, is the key to preventing the commonest production diseases of intensively fed cattle. Cunningham (Ch31 Figs 31.10–31.11) details VFA transport across the rumen epithelium.

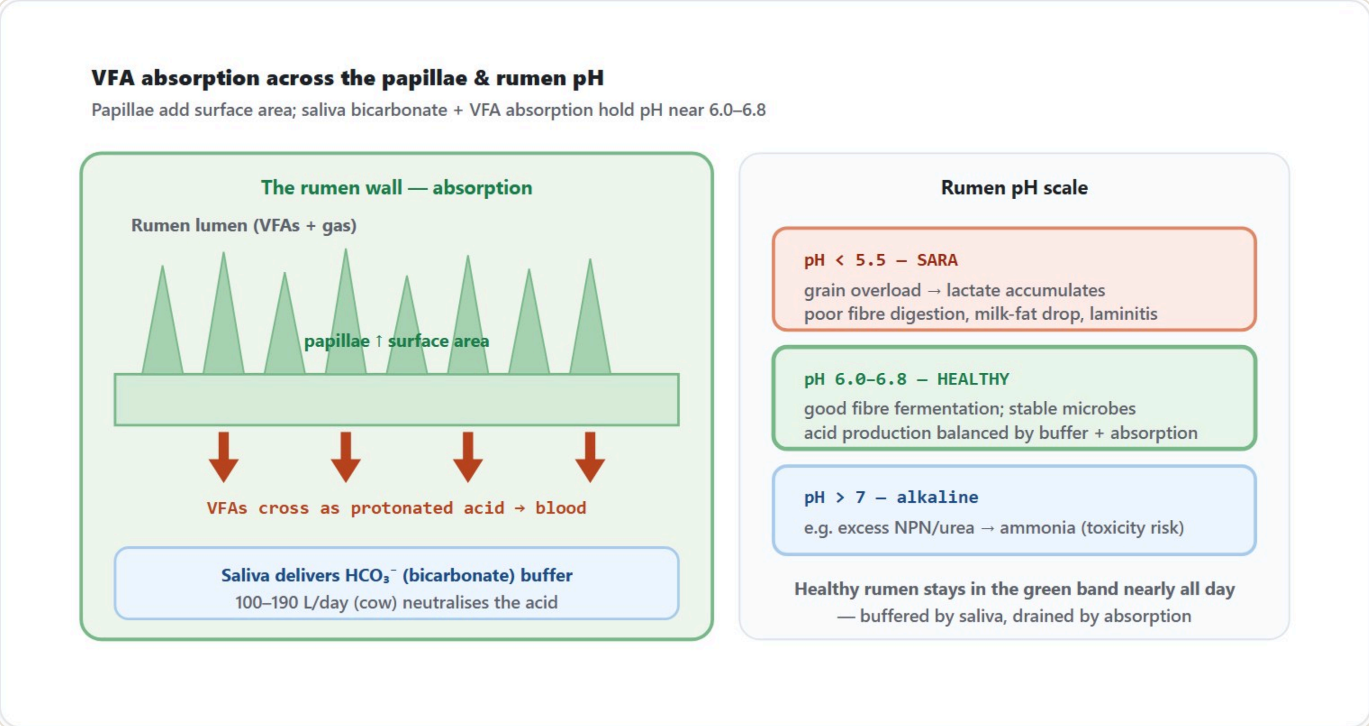



Fig 5 — VFA absorption and rumen pH. VFAs cross the papilla-covered rumen wall mostly as protonated acid; saliva delivers bicarbonate buffer. Together they hold the healthy rumen near pH 6.0–6.8; below ~5.5 (grain overload) is SARA. (Cunningham 6e Ch31 Figs 31.10–31.11, pp356–357.)

## 08 Making protein from non-protein nitrogen (the urea story)

One of the most remarkable features of **ruminant digestion** is that a ruminant can build high-quality protein out of remarkably cheap nitrogen — even out of nitrogen that is not protein at all. The mechanism runs through the microbes. Rumen microbes build their own cellular protein, the so-called **microbial protein**, from **ammonia**. They take ammonia, combine it with a carbon skeleton derived from fermenting carbohydrate, and assemble amino acids and then protein. The cow then digests those microbes downstream in the abomasum and absorbs their amino acids in the small intestine. In effect the cow eats her own microbial crop, and that crop is her main protein supply.

The clever part is where the ammonia comes from. It can come from ordinary dietary protein, which the microbes degrade to ammonia. But it can equally come from **non-protein nitrogen (NPN)** — simple nitrogen compounds such as **urea**. Microbial **urease** splits urea into ammonia and $CO_2$, and the microbes then capture that ammonia into microbial protein just as they would ammonia from any other source. This is why you can feed cattle urea as a cheap protein supplement: the urea itself is worthless to the animal, but the microbes convert it into valuable microbial protein. The ruminant also recycles nitrogen efficiently, returning urea to the rumen in the saliva and across the rumen wall, so that nitrogen is conserved rather than wasted — a real advantage on low-protein forage diets.

**Callout — a clinical caution on urea.** The safety of feeding urea depends entirely on the microbes capturing the ammonia as fast as urease releases it. Feed urea too fast, or to animals whose microbes are not adapted to it, and ammonia is liberated faster than it can be used. The excess ammonia is absorbed into the blood, overwhelms the liver's capacity to detoxify it, and causes **ammonia (urea) toxicity** — a rapidly fatal poisoning with tremors, bloat and convulsions. Urea must always be introduced gradually and mixed evenly in the ration.

Cunningham (Ch31 Figs 31.4–31.6) sets out microbial protein synthesis and the nitrogen-recycling cycle in detail. The concept to carry away is that the ruminant, uniquely, decouples the *quality* of the protein it absorbs from the *quality* of the protein it eats: no matter how poor the dietary nitrogen, the microbes upgrade it into consistent, high-quality microbial protein — provided the fermentation is healthy and there is enough fermentable carbohydrate to supply the carbon skeletons.

## 09 Rumen development: from milk-fed calf to functional ruminant

Here is a fact that surprises most students: a newborn calf, lamb or kid is **not** a functional ruminant. It is born a **functional monogastric**. Its rumen is small, undeveloped and essentially sterile — it has no established microbial population and cannot ferment anything. The young animal lives entirely on milk, and milk is a simple, high-quality food that needs the abomasum, not the rumen. So the neonate has a problem: how does it get milk to the abomasum without dumping it into a useless, undeveloped rumen where it would ferment abnormally and cause disease?

The solution is a beautiful reflex called the **oesophageal groove** (or reticular groove). When the young animal suckles, sensory receptors in the mouth and pharynx trigger a reflex that causes a muscular fold in the wall of the reticulum to **close into a tube**, forming a channel that carries the swallowed milk directly from the oesophagus, past the reticulorumen and omasum, straight into the abomasum. The milk bypasses the forestomach entirely. This groove-closure reflex is reliable in the suckling animal, which is exactly why we want young ruminants to *drink* milk from a teat rather than gulp it from an open bucket — teat-feeding triggers the reflex reliably; sloppy bucket-feeding may not, and milk that spills into the rumen ferments and causes trouble.

As the young animal begins to nibble solid feed, everything changes. Solid feed introduces the microbes (picked up from the environment and from other animals) and gives them a substrate to ferment. Fermentation begins, and it produces VFAs — and here **butyrate** plays a starring role. Butyrate (and to a lesser extent propionate) is the principal stimulus for the **growth of the rumen papillae**: as fermentation gets going, the rumen wall responds to the butyrate by developing the dense carpet of papillae it needs to absorb VFAs. The rumen enlarges, the microbial population establishes and matures, and over a few weeks the animal transforms from a milk-dependent monogastric into a fully functional ruminant able to live on forage. By the time of **weaning** the transition is essentially complete. This is why early access to good-quality solid feed (calf starter) accelerates rumen development and makes for an easier weaning.

**Callout — the principle to remember.** A calf is born a functional monogastric; the rumen is made, not inherited. It is built by the microbes and by the butyrate they produce — which is why solid feed, not milk, is what grows the rumen.

The clinical relevance is direct. **Groove-closure failure** or clumsy feeding produces the '**ruminal drinker**' — a calf in which milk repeatedly spills into the undeveloped rumen, ferments abnormally, and causes ruminal acidosis, poor growth and ill-thrift. And any calf, lamb or kid weaned before its rumen has developed will do poorly, because it has lost the milk it depended on before gaining the fermentative capacity to replace it.

## 10 When the rumen fails: SARA, bloat, LDA and milk-fat depression

Because the rumen is a finely balanced fermentation system, its failure modes are predictable, and each one is read straight off the physiology we have built up. Four disorders dominate ruminant practice, and a graduate should be able to explain the mechanism of each from first principles.

**Subacute ruminal acidosis (SARA)** is the disease of intensive feeding. It follows **grain overload** or too abrupt a switch onto rapidly fermentable carbohydrate: the starch-fermenting and lactate-producing bacteria bloom, acid production outruns the saliva buffer and papillary absorption, and rumen pH falls below about **5.5** for hours at a time. The consequences ripple outward: fibre-digesting microbes die (so fibre digestion falls), the VFA pattern shifts (so milk fat drops), the rumen wall becomes inflamed (rumenitis), and acid and endotoxin damage can seed liver abscesses and trigger **laminitis**. The classic scenario is the feedlot steer pushed onto a high-grain ration too fast, or the dairy cow on a marginal-fibre TMR.

**Rumen bloat (ruminal tympany)** is a failure of **eructation** — the animal cannot belch away the 30–50 litres of gas the microbes make each hour, and the reticulorumen distends. In **frothy bloat**, typically on lush legume pasture (clover, alfalfa) or on finely ground grain, the gas is trapped in a stable foam that the animal cannot belch. In **free-gas bloat**, the gas is free but the escape route is blocked (an obstructed oesophagus, or an animal down in an abnormal posture). Either way the gas cap grows, the **left flank distends** (the paralumbar fossa balloons out), and the enlarging rumen presses forward on the diaphragm and lungs, causing **dyspnoea (dyspnea)**. Severe bloat kills by asphyxiation and is an emergency. The classic scenario is the bullock turned out onto lush clover.

**Left displaced abomasum (LDA)** is largely a disease of the fresh dairy cow. In the weeks after calving, the abdomen has just been vacated by the calf, feed intake is often poor, and abomasal motility (atony) and gas accumulation combine to let the gas-filled abomasum **float up and slip under the left rib cage**, trapped between the rumen and the body wall. The cow goes off her feed, milk yield drops, and on **percussion of the left flank** you hear the characteristic high-pitched metallic '**ping**' of a gas-filled viscus. LDA is common enough that in fresh-cow examinations the clinician routinely listens for the ping.

**Milk-fat depression** closes the loop back to the VFAs. A ration low in effective fibre and high in grain shifts the VFA ratio **away from acetate** toward propionate (and alters rumen biohydrogenation of fats in ways that further suppress milk-fat synthesis). Since acetate is the main precursor of milk fat, the butterfat percentage falls — often below 3%, sometimes low enough to invert the normal fat-to-protein ratio in the bulk tank. Milk-fat depression is not a sick cow so much as a herd-level ration problem, and its fix is a ration fix: restore effective fibre. Constable/Radostits and the Merck Veterinary Manual are the clinical references for the thresholds and management of all four disorders.

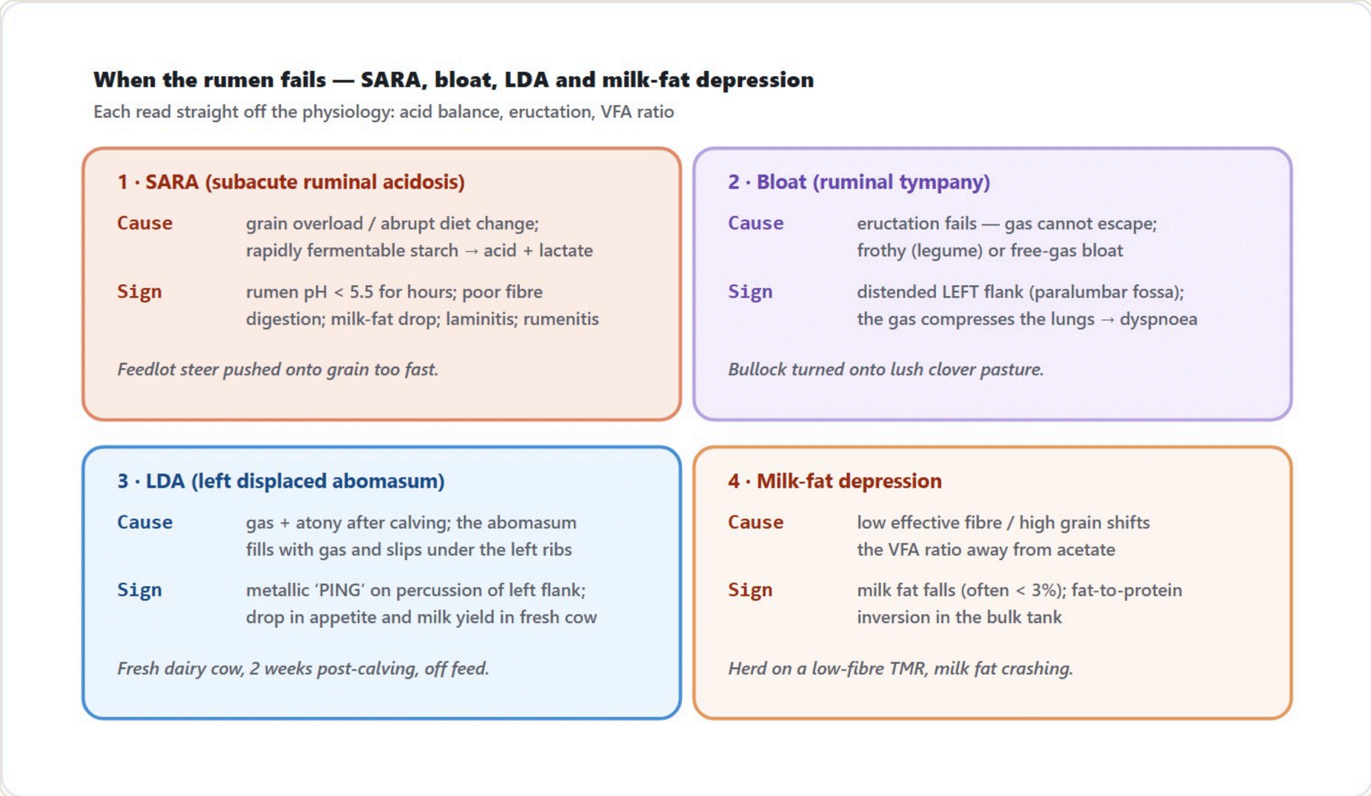



Fig 6 — When the rumen fails. SARA (grain overload → pH<5.5, lactate); bloat (eructation failure → left-flank distension, dyspnoea); LDA (gas + atony post-calving → left-flank 'ping'); milk-fat depression (low fibre → propionate shift → milk fat <3%). Each read off the physiology. (Constable/Merck + Cunningham 6e Ch31.)

## 11 Clinical reasoning and species notes

Everything above comes together at the level of the herd and the individual animal, and the clinician's most direct window onto the rumen is the **rumen fluid examination**. A sample drawn by stomach tube or rumenocentesis can be assessed on the spot: its **pH** (a healthy 6.0–6.8, low in SARA), the **motility of the protozoa** under the microscope (vigorous in a healthy rumen, sluggish or absent when the rumen is sick or acidotic), the **colour and smell** (a healthy olive-green with an aromatic, not sour or putrid, odour), and the **consistency**. A rumen whose protozoa have died and whose pH has crashed tells you at a glance that the microbial ecosystem has collapsed. When it has, one remedy is **transfaunation** — transferring rumen fluid from a healthy donor cow to reseed the microbial population in the patient. The very existence of transfaunation makes the central point of this article concrete: the rumen is an organ you can restart by restocking its microbes.

The **species** differences matter clinically too. Cattle are grazers, adapted to bulk forage; **sheep and goats** are more selective, with goats in particular behaving as **browsers** that seek out leaves, shrubs and a more varied diet, and tending to tolerate tannin-rich plants better than cattle. The basic four-compartment plan and the fermentation physiology are the same across all true ruminants, so the principles in this article apply to cattle, sheep and goats alike — but the practical feeding and the relative importance of different diseases differ. Note one important exception: **camelids** (llamas and alpacas) are *not* true ruminants. They ferment their feed and chew a cud, but they have a **three-compartment** forestomach rather than four, so although the fermentation logic is broadly similar, the anatomy and some of the clinical detail differ; do not simply extrapolate cattle rules to a llama.

Two rules of thumb bring the physiology into daily practice. First, **change diets gradually**. The microbial population takes days to shift its balance to match a new ration, and every sudden change — onto grain, onto lush pasture, off feed and back on — risks acidosis, bloat or a displacement while the microbes catch up. Adaptation is everything. Second, **treat the herd, not just the animal**. A single case of SARA, bloat, LDA or milk-fat depression is usually the visible tip of a ration or management problem affecting the whole group; the sick individual is a sentinel. The clinician who fixes only the animal in front of them and ignores the feed bunk will simply see the next case tomorrow. Understanding **rumen physiology in cattle**, sheep and goats is what lets you read that sentinel correctly and fix the cause, not just the symptom.

## 12 Quiz: test yourself

Fifteen questions across the four compartments, rumination, the microbiome, the VFAs, rumen pH and the non-protein nitrogen story. Answers are beneath each question — cover them and work down.

1. Why can no mammal digest grass unaided?**Answer:** Grass is mostly **cellulose**, and **no mammal makes an enzyme that can break the β-1,4 bonds** holding it together. The animal rents the enzymes from its microbes.2. How much of a cow's abdomen does the rumen fill, and what does it hold?**Answer:** Roughly **75%** of the abdominal cavity, holding **well over 100 litres** of fermenting digesta.3. What are the physical conditions inside the rumen?**Answer:** A warm (**~39°C**), **anaerobic**, continuously mixed vat — with essentially no oxygen.4. How long does a cow spend ruminating, and why does it matter clinically?**Answer:** Roughly **eight hours a day**. **A cow that stops chewing her cud is a cow that is unwell** — it is one of the earliest signs you can see from the gate.5. How much gas do the microbes make, and what happens if it cannot escape?**Answer: 30–50 litres an hour**, virtually all belched away by **eructation**. If the animal cannot belch, the rumen distends and it **bloats**.6. How dense is the rumen microbial population?**Answer:** On the order of **$10^{10}$ to $10^{11}$ microorganisms per millilitre** of rumen fluid — tens of billions in every millilitre.7. How fast does the microbial community respond to a diet change?**Answer:** The balance of species **shifts within days**. That lag is exactly why ration changes must be made gradually.8. What do the methanogens do, and what does it cost?**Answer:** They **mop up hydrogen into methane**, which keeps fermentation running. The cost is a potent greenhouse gas and the loss of **6–12% of the feed's gross energy**.9. What is the VFA ratio on a normal forage diet?**Answer:** Roughly **70 : 20 : 10** — acetate : propionate : butyrate, with acetate dominant.10. Why is propionate described as king?**Answer:** It supplies **65–80% of a dairy cow's glucose**, and that glucose becomes, among other things, the **lactose of milk**.11. What keeps rumen pH stable, and in what band?**Answer: Bicarbonate and phosphate** poured in with **100–190 litres of daily saliva**, plus **absorption across the papillae**. Between them a healthy rumen holds pH at about **6.0 to 6.8**.12. How can a ruminant live on non-protein nitrogen such as urea?**Answer:** The microbes **build their own protein from ammonia**, and the cow then digests the microbes. She eats her own microbial crop.13. Is a newborn calf a functional ruminant?**Answer: No** — it is born a functional **monogastric**. Its rumen is small, undeveloped and essentially **sterile**, with no microbial population and no capacity to ferment anything. It lives entirely on milk, which needs the abomasum.14. What is the oesophageal groove, and what triggers it?**Answer:** A muscular fold in the wall of the reticulum that **closes into a tube** when the young animal suckles, triggered by sensory receptors in the mouth and pharynx. It carries milk **straight past the undeveloped rumen to the abomasum**, where it would otherwise ferment abnormally and cause disease.15. What is urea poisoning, and how does it kill?**Answer:** Excess ammonia is **absorbed into the blood and overwhelms the liver's capacity to detoxify it** — a rapidly fatal poisoning with **tremors, bloat and convulsions**.

## 13 Rumen physiology in cattle, sheep and goats — frequently asked questions

### How does the rumen work in simple terms?

The rumen is a large fermentation vat inside a cow, sheep or goat. The animal cannot digest grass on its own, because no mammal makes an enzyme that breaks down cellulose. So it houses billions of microbes (bacteria, protozoa and fungi) in the rumen that ferment the fibre into **volatile fatty acids (VFAs)** — the animal's main energy source — plus gas and new microbial cells. The animal absorbs the VFAs across the rumen wall, belches off the gas, and digests the microbes downstream for protein. In short: the microbes digest the grass, and the animal digests the microbes and their products.

### What are the four compartments of a cow's stomach?

In the order feed passes through them, they are the **reticulum**, the **rumen**, the **omasum** and the **abomasum**. The first three (the forestomach) make no acid and no enzymes of their own — all their digestion is done by microbes. The reticulum catches heavy or sharp objects; the rumen is the huge fermentation vat (about 75% of the abdomen); the omasum absorbs water and residual VFAs. Only the **abomasum** is the 'true' stomach, secreting hydrochloric acid and pepsin like the single stomach of a dog or cat, and it digests both feed and the microbes flowing out of the forestomach.

### What are VFAs and why do they matter?

**Volatile fatty acids (VFAs)** are the short-chain fatty acids — **acetate**, **propionate** and **butyrate** — produced when rumen microbes ferment carbohydrate. On a fibre diet they are made in a ratio of roughly 70:20:10. They matter because they are the ruminant's main source of energy: they are absorbed directly across the rumen wall and supply most of the animal's usable calories. Acetate feeds energy and milk fat; propionate feeds glucose production; butyrate feeds ketones and the rumen wall. Almost everything the animal does — grow, lactate, stay warm — runs on VFAs.

### Why is propionate the most important VFA?

Because **propionate is the only glucogenic VFA** — the only one the animal can turn into glucose. A ruminant absorbs almost no glucose from its gut (the microbes ferment dietary sugars first), so it must manufacture glucose itself by **gluconeogenesis** in the liver, and propionate is the main raw material. Propionate supplies 65–80% of a dairy cow's glucose, and that glucose becomes the lactose of milk. Acetate and butyrate cannot be made into glucose. So for a high-yielding dairy cow, milk output depends heavily on how much propionate her rumen delivers — which is why grain (which raises propionate) boosts milk yield but, in excess, risks acidosis.

### What is rumen acidosis (SARA)?

**Subacute ruminal acidosis (SARA)** is a drop in rumen pH below about **5.5** for several hours a day, caused by feeding too much rapidly fermentable grain relative to effective fibre. The microbes ferment the starch fast and (in overload) produce lactic acid, and acid is generated faster than the animal's saliva bicarbonate and VFA absorption can buffer it. The result is reduced fibre digestion, milk-fat depression, and, over time, laminitis, rumenitis and liver abscesses. Prevention is about ration balance — enough effective fibre to stimulate chewing and saliva — and gradual dietary change so the microbes can adapt.

### Can I download these rumen physiology study notes as a free PDF?

Yes — these are complete, illustrated veterinary study notes on rumen physiology in cattle, sheep and goats, free to download as a PDF with no signup. For more veterinary study notes, browse our veterinary physiology hub — including the companion articles on cellular homeostasis (where the energy from VFAs is finally spent) and oedema and the Starling forces (the 'bottle jaw' of the parasitised ruminant).

**When to refer**

- A cow or steer with acute grain overload (raided the feed store) — rumen pH crashing, staggering, profuse diarrhoea (diarrhea); a lactic-acidosis emergency needing urgent rumen decompression, fluids and alkalinising therapy — refer or treat aggressively.
- A bloated animal with a rapidly distending left flank and dyspnoea — frothy or free-gas bloat is an emergency; pass a stomach tube and, if severe and life-threatening, be prepared for emergency trocarisation of the left paralumbar fossa.
- A fresh dairy cow (within ~1 month of calving) off feed with a drop in yield and a metallic **ping** on left-flank percussion — suspect **left displaced abomasum (LDA)**; refer for surgical or toggle correction.
- A herd with falling bulk-tank milk fat (often < 3%) — investigate the ration for inadequate effective fibre / excess grain driving milk-fat depression and possible subclinical SARA; a whole-group nutrition review, not an individual-cow problem.
- Any animal after urea/NPN feeding showing muscle tremors, bloat, salivation and convulsions — suspect **ammonia (urea) toxicity**; a rapidly fatal poisoning — treat as an emergency (cold water and vinegar into the rumen buys time) and review how the urea was fed.

## Sources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3. Constable PD, Hinchcliff KW, Done SH, Grünberg W. *Veterinary Medicine (Radostits)*. 11th ed. Elsevier; 2017. Chapters on ruminal acidosis (SARA), bloat, left displaced abomasum and urea toxicity — clinical anchor for thresholds and management.
4. Merck Veterinary Manual (online). Sections on the digestive system of ruminants, ruminal acidosis, ruminal tympany (bloat), displaced abomasum and non-protein nitrogen (urea) poisoning — clinical reference for signs and treatment.


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