Pathophysiology of the Bovine Stomach

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Bovine stomach diseases are quite common and complicated. A knowledge of their origin and mechanisms is essential to recognize and to control them. A distinction between transit disorders and disorders of ruminal fermentation is convenient for the reticulo-rumen. What we know today concerning the abomasum is that the analytic approach is the only possible way.

1-Reticulo-Rumen: transit disorders
J. Espinasse

A well-blended mixture of liquids, solids and gases is transported through the reticulo-rumen in the rumen biomass. The fermented liquid-solid component leaves the rumen via the reticulo-omasal orifice and the fermentation gases accumulated in the dorsal sac are expelled by the cardia. Any malfunction in this gastric complex leads to the condition known as indigestion.

1. Cessation of slowing down of gas and liquid-solid transit (Ruminal indigestion) (Fig. 1)

1.1 *Of central origin*

Accidental traumatic reticulo-peritonitis may lead to a total blockade in transport both upwards and downwards from the rumen. This condition results from enbedding in the reticular wall of a usually metallic foreign body that is ingested with the food and, on perforation, produces peritonitis. In most cases the offending object is a piece of wire (58%), although nails (36%) and miscellaneous objects (6%) are also found. In industrialized, countries, metallic foreign bodies may be present in the reticulum in up to 90% of normal cattle, dairy cows in particular, and residual traumatic lesions of reticulo-peritonitis may be observed on slaughter in as many as 70% of dairy cows; 25% of the animals which exhibit clinical signs are likely to develop serious complications whereas the remaining 75% recover after conservative treatment or routine surgical intervention (3).

The ingestion of foreign bodies is facilitated in cattle by the lack of dietary selectivity while the structure and vigorous contractions of the reticulum ensure adhesion to the visceral wall and subsequent embedding. Penetration may be further facilitated by pregnancy and/or parturition. The reticuloruminal atony observed during traumatic reticulo-peritonitis may be due both to peritoneal pain which leads to inhibition of the gastric motor centres located in the brain stem (20) and to fever, which is in itself associated with peritonitis (22).

Most of the acute infectious diseases encountered in ruminants (bronchopneumonia, mastitis) are also accompanied by reticuloruminal atony during the febrile period. Not all febrile conditions inhibit rumen motility, however. The fever caused by a rickettsia transmitted by ticks (tick born fever) in goats (35) is an example. The effects of gram negative bacteria endotoxin on rumen motility have received particular attention and are independant of hyperthermia. They seem to act on the gastric motor centres by producing E2 prostaglandins and above all by another mechanism of inhibition that may involve opioid-type substances such as β endorphin which is antagonized by naxalon (central effect). The initial phase would also involve relaxation of smooth muscles in the reticulo-rumen wall, leading to disapearance of the vagal afferent impulse, along with an increase in tonus of these same muscle fibres (peripheral effect) (25).

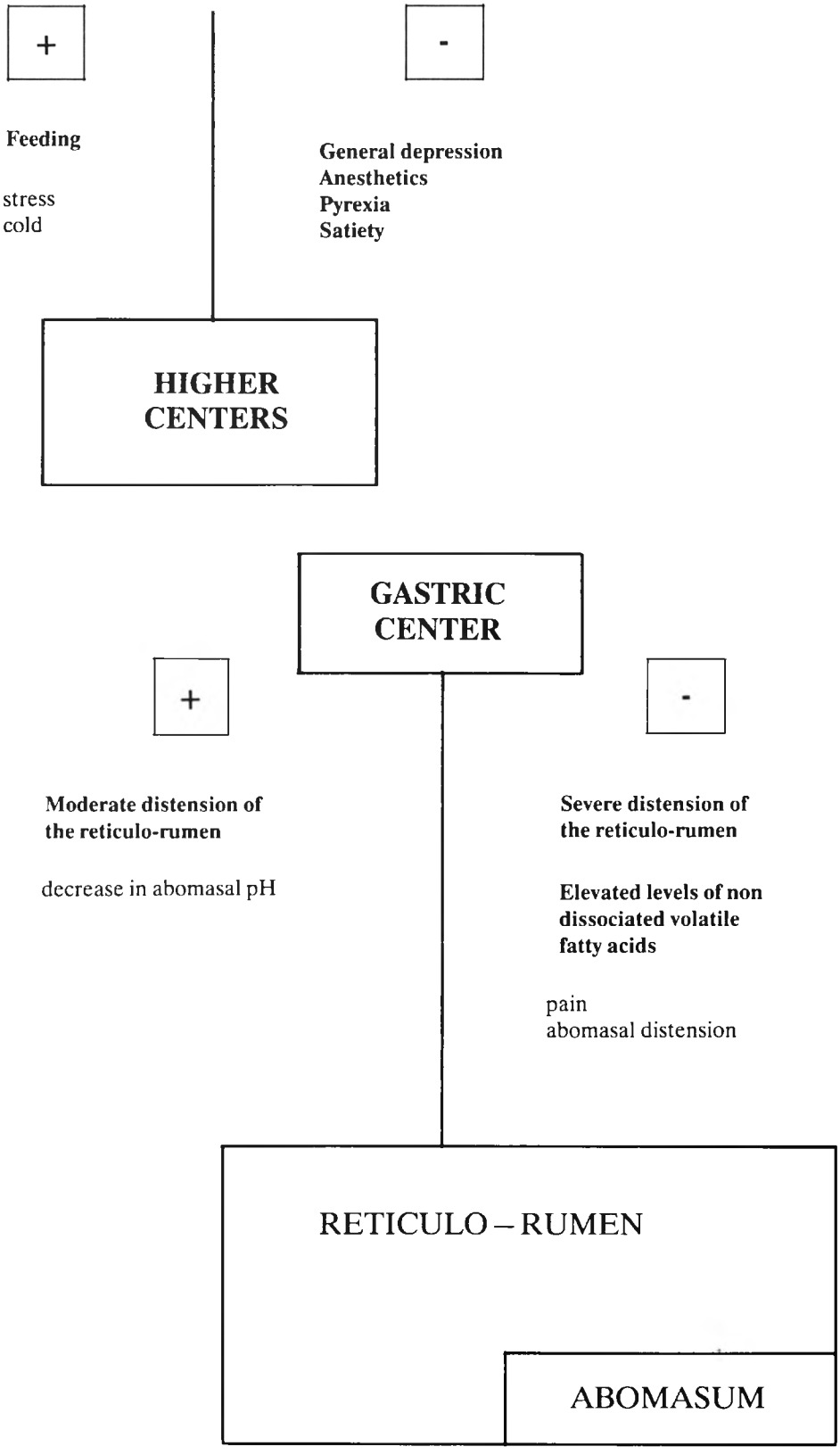
Stress (due to cold or the presence of a dog trained to exhibit agressivity) might be expected to induce ruminal hypomotility (cathecolamine secretion). The opposite effect has been observed under experimental conditions. This might be explained by exacerbation of the vago-vagal reflex with relay to the gastric motor centres of the medulla oblongata (24).

Xylazine which, because of its sedative, analgesic and myorelaxant properties, is frequently used in buiatrics, may sometimes produce undesirable effects such as tachycardia, tachypnea and tympany in ruminants. Its inhibiting effects on the gastric centres and resulting adverse effects on rumen motility have been demonstrated. These are immediately reversible on injection of an antagonist such as tolazolin (32). It is relevant here to mention that in sheep, xylazine mainly alters the frequency and quality of the primary cycle contractions so that these animals are less susceptible to ruminal tympany than cattle (6).

1.2 *Of peripheral origin*

The moderate distension of the rumen that occurs after meal ingestion helps to maintain rumen motility by stimulating the tension receptors present in very high densities in the reticular wall and in the cranial sac of the

Figure 1: Reticulo-rumen motility: major excitatory (+) and inhibitory factors (-) (12)



rumen (low-threshold tension receptors). During anorexia induced by a pathological digestive or extra-digestive condition, these mecano-receptors are no longer solicited which results in a modification of rumen motility (22). Observations that rumen contractions continue in the total absence of fibrous content after four weeks of artificial liquid feeding in sheep (27) emphasize the importance of absence of food intake in this form of indigestion. The lack of food ingestion leads to a suppression of mastication which is, through the sensorial receptors, one of the most effective stimulants of the reticuloruminal motility. A return of normal appetite (with or without the use of orexigens such as a benzodiazepine as brotizolan (or the now abandoned "masticadour") is known to accelerate convalescence after all forms of indigestion.

Correct fermentation in the rumen requires a large amount of water (85 to 90% of the total contents). Prolonged insufficient fluid intake leads to a modification in the consistency of the ruminal contents with subsequent adverse effects on rumination and reticulo-rumen motility (7).

The prolonged reduction in solid nutrient and water supplies concomitant with starvation, brings about a rapid reduction in the rumen population of bacteria and protozoa. Exaggerated alterations in the principal dietary components may also be responsible for changes in fermentation, at least two weeks being required for the micropopulation to adjust their fermentation product yields. If, for example, a bovine animal goes without transition from a diet containing a high level of concentrate to a ration based on coarse food stuffs it will tend to increase the amount ingested. The appetite is in fact checked not so much by the progressive distention of the rumen as by the quantity and nature of the metabolites resulting from ruminal digestion which in the present case is globally insufficient. The excessive amount of food ingested, associated with the fermentary incapacity of the micropopulation rapidly leads to indigestion due to rumen overload (22).

This type of indigestion occurs commonly in calves during weaning. If the ration does not contain enough easily digestible nutritive elements or if the calf prefers straw or hay (pica), the vicious circle represented in figure 2 may be set up (10). The undigested coarse fodder accumulates in the rumen which dilates progressively due to an influx of afferent inhibitors produced by epithelial receptors sensitive to mechanical stimulation (high-threshold tension receptors). The sensitivity of these same epithelial receptors to chemical stimulation (acid solutions acting more by titrable acidity and in non-dissociated form than by pH, except in the case of butyric acid; alkaline hypo or hyperosmotic solutions) suggests that in ruminal lactic acidosis and alkalosis resulting from putrefaction of the rumen contents or excess non-protein nitrogen, the adverse effects on rumen motility would result from an impulse of afferent inhibitors of mechanical and/or biochemical origin. In other circumstances, reticuloruminal atony may result from the action of other metabolites (histamine) derived from the rumen micropopulation (11) or from substances present in the feed (slaframine produced by a fungal parasite of the leguminosae: Rhizoctonia leguminocola) (14).

Continuous elimination of the products of metabolism of the rumen micropopulation, either by absorption through the ruminal wall (volatile fatty acids, ammonia in particular) or passage into the distal portion of the digestive tract (feed residues, bacteria, protozoa), or by eructation (carbonic acid, methane) is necessary to correct rumen function. LIEBICH *et al.* (23), showed that in dairy cows fed on a drying-off diet during the dry period, the surface area of absorption of the rumen papillae, together with their absorptive capacity, was reduced as a result of structural involution. After parturition, and with a production ration, a progressive return to normal was observed, spread over a minimum period of four weeks. These facts should be compared with previous observation (7) concerning the frequency of rumen contractions, intraruminal pressure and the appetite of dairy cows during parturition. A reduction in the frequency of rumen contractions at the time of parturition, a reduction in intra-ruminal pressure two weeks before and up to one week after parturition, a decrease in comsumption of concentrates and hay four to five days before parturition, was observed with more rapid renewal of consumption of concentrates than of hay in the week following parturition (Fig. 3). These authors suggest that biochemical, absorption and reticuloruminal motility interrelationships would explain the poor appetite and indigestion frequently observed in dairy cows during the peri-partum period.

Ruminal hypomotility during the peri-partum period may also result from hypocalcaemia. In sheep, intravenous infusion of disodium EDTA leads to a reduction in rumen contractions correlated with hypocalcaemia even before the clinical signs are expressed (18). The Ca++ ion is, in fact, indispensable to the liberation of acetylcholine from the neuromuscular synapsis and could also target the gastric centres of the brain stem (22). It should be noted that early ruminal hypotonia and problems associated with subsequent digestive transport facilitate the development of clinical hypocalcaemia by reducing the calcium supply at a particularly critical period in the dairy cow's production cycle.

While the peripheral afferent inhibitor influx starts mainly in the reticulo-rumen, the presence in the abomasum of tension receptors and epithelial receptors that behave like the tension receptors and epithelial receptors in the rumen would imply other possible sources of rumen motility modification. The abomasal tension receptors are slow-adaptation mecano-receptors, with a low threshold in relation to the slower conduction fibres. Their stimulation may lead to the inhibition of ruminal contraction and vagal

gastric efferent nerve activity observed during abomasal distention.

The negative retroaction brought about by filling the abomasum would reduce forestomach motility and limit transport to distal compartments, thus avoiding subsequent overload (31). These relationships would explain atony of the reticulo-rumen concomitant with abomasal dilatation, with or without displacement of the latter. Apart from the tension receptors, the presence of mucosal receptors in the abomasum would also provide an explanation for rumen dysfunction. In fact, whereas the administration of fatty acid into the abomasum tends to stimulate rumen motility, injectin of lactic acid or hydrochloric acid (equivalent acidity to that of lactic acid) into the duodenal bulb momentarily inhibits the extrinsic contractions of the reticulo-rumen through a long vagal reflex (27). These data help in an understanding of the motility problems associated with ruminal lactic acidosis.

2. Cessation or slowing down of gas transit (Ruminal Tympany)

Effective evacuation of the gases of fermentation presupposes the existence of an antiperstatic ruminal contraction (secondary, cycle B), free functioning of the lower oesphageal sphincter and of the oesophagus.

Partial or total blockade of the oesophagus by a foreign body whether dietary (apple) or otherwise (plastic), obstruction of the oesophagus by food during pharangeal paralysis (rabies, listeriosis, botulism) or oesophageal spasm (tetanus), traumatic or post-traumatic lesions of the oesophageal wall (laceration, abscess, stricture, partial ectasia), oesphageal compression (thymic lymphosarcoma, cervico-mediastinal adenopathy) are various pathological events that can hinder or prevent eructation. It should be stressed in connection with ruminal tympany observed as a consequence of respiratory disease in young cattle that in addition to the possible role of the mediastinal adenopathies, alterations in intra-pleural pressure which modify lower oesophageal sphincter function(19) may intervene.

It is the foam characteristic of frothy bloat that may both obstruct and inhibit correct function of the lower gastro-oesophageal sphincter. A meal containing highly fementable and thus acidogenic substances is also considered to be unable to correctly stimulate the mecano-receptors situated near the sphincter. This non-activation reduces the frequency of the inhibiting afferent nerve impulses that regulate sphincterial tonus and thus increases the lower gastro-oesophageal tonus. The cardia may also fail to open as a result of various local or perilocal organic lesions mainly of the oesophageal groove (papilloma or other tumours, actinomycosis, actinobacillosis, injury due to embedding of a foreign body).

Reticular groove malfunction observed in the "ruminal drinking" syndrome of calves fed on milk replacer diets is another possible cause of bloat. The gases produced by bacterial fermentation of the milk (lactic and butyric) accumulate in the rumen and are not evacuated (5). The absence of the reticular groove closure reflex has also been described as a possible complication of neonatal diarrhoea (8) and in both cases, lesions of hyperkeratosis may be found in the rumen wall.

In ruminants in lateral or latero-dorsal recumbency (due to paralysis or sliding during transport in a too narrow vehicle), the cardial orifice may be submerged by the level of liquid with resulting difficulty or impossibility in eructation. Chronic bloat has also been reported in cases of ruminal torsion about the longitudinal axis (28).

In the acute, occasionally fatal cases of tympany due to the absorption of gas (mainly hydrogen sulphide), toxic amines (principally histamine), or cardio-respiratory effects of thoracic compression, the abnormal motility varies as a function of ruminant species. In bloated sheep, the A and B cycles disappear simultaneously at an early stage. In cattle, cycle A disappears first and the B cycles increase before disappearing. These observations would indicate the essential role of stenosis of the lower oesophageal sphincter at least in free-gas, tympany and/or acute frothy bloat (4).

In recurrent, gaseous bloat, lesions of the rumen wall (parakeratosis, hyperkeratosis, ruminitis, ulcers) may lead to dysfunction of the secondary cycles associated with eructation. These lesions may be of mechanical origin (polypropylene), infectious (digestive form of infectious bovine rhinotracheitis, localization of papular stomatitis in the stomach and bovine mucosal disease) and/or metabolic (ruminal lactic acidosis, ruminal hydrochloric acidosis by abomasal reflux, possibly complicated by bacteria (*Fusobacterium necrophorum)* or fungi (Mucor, Aspergilius) often in this later case after all antibiotic treatment by oral route (1, 3, 33).

3. Cessation or slowing down of liquid-solid transit (vagus indigestion or Hoflund's Syndrome)

Movement of the liquid-solid mixture may be impeded as a result of reticuo-omasal sphincter failure and/or dysfunction of the propulsive contractions of the rumen. The rumen in fact consists of a fine layer of undigested fibre floating on a liquid that contains a suspension of fine food particles. The presence of liquid in the ventral sac is explained by the sieving effect of the fibrous layer on the coarse particles. A two-way circulation of the ingested material also exists in which the reticulo-rumen plays the role of regulator. The contractions of the dorsal sac of the rumen separate the fine particles from the coarse particles by a pressing effect. These fine particles are found near the reticulo-omasal orifice whose efficacy in emptying the reticulo-rumen is linked to the pressure difference existing between the omasal canal and the reticulo-rumen. The reticulo-omasal orifice is relaxed during food intake, and during rumination opens at the end of each primary con-

traction.

Different types of lesions may modify the profile of the reticulo-omasal orifice and/or omasal canal. The obstructive lesions may be due to tumours (papilloma, carcinoma, lymphosarcoma), localized infections (actinomycosis, actinobacillosis) or foreign bodies (phytobezoar, trichobezoar (16), placenta, string, plastic). The compressive lesions may consist of abscesses (of the liver, reticulo-rumen wall, diaphragm, peritoneum) or tumours (21). The above anomalies may sometimes deform the reticulo-omasal orifice and omasal canal by stretching. Under other circumstances such deformation may result from adhesions between the reticulo-rumen and right side region of the diaphragm, from reticulo-peritonitis sequellae in which the ventral vagal trunk or its ramifications may be compressed and/or from a state of ischemia (2, 13, 30).

So far as abnormalities in ruminoreticular motility are concerned, any lesions in the vicinity of the vagus nerve between the neck and thorax constitute potential causes of vagus indigestion as the nerve may be involved in an inflammatory or tumoral process, compressed, or ischemic with neuropraxis as in traumatic injury and abscess of the pharynx, peri-oesophageal traumatism and phlegmons, cervical and/or thoracic adenopathy, mediastinitis, diaphragmatic hernia of the reticulum. The possible role of empyema of the omental bursa (34), necrotic lesions of the abdominal fat (15), possible stretching of the vagus nerve or ischemia due to thrombosis of the vascular network of the lesser abomasal curvature following torsion of the abomasum (30) may also be added to this list. In a study of 43 cases of vagus indigestion, NEAL and EDWARDS (29), detected pneumogastric lesions in only 9 animals whereas most of the others i.e. about 30, exhibited reticular wall lesions (abscesses, adhesions). LEEK (22), therefore considers it unlikely that the majority of cases of reticulo-rumen motility dysfunction in Hoflund syndromes are directly related to vagal lesions even if brachycardia responding to atropine is observed. They would more often result from lesions of the ruminoreticular wall produced by chronic inflammatory phenomena, probably painful, that affect the tension receptors. (26).

HOFLUND (17) attempted, in his historical account, to group the different clinical expressions of vagus indigestion, based on experimental investigation, into four major syndromes.

i) functional stenosis between the reticulum and omasum with atony of the rumen and reticulum,
ii) functional stenosis between the reticulum and omasum with retained activity of the rumen and reticulum.
iii) permanent functional stenosis of the pylorus with or without activity of the reticulum,
iv) incomplete pylorus stenosis.

It would seem from the preceeding remarks that the modifications of this classification proposed by FERRANT and WHITLOCK (13) are nearer to reality:

1) failure of eructation (free gas bloat)
2) omasal transport failure,
3) abomasum blockade,
4) partial obstruction of the stomach.

We have already examined the causes of type 1 and 2 indigestions, and type 3 will be discussed further on. The 4th type of indigestion would tend to be confined to the pregnant female. The uterus rapidly increases in volume at the end of pregnancy, is pushed backwards and increasingly compresses the abomasum cranially which interferes with its own motility and with that of the reticulo-rumen. If an incomplete type 2 syndrome adds on to this situation, then digestive troubles may appear.

The difference between types 2-4 and 3 is not always obvious on clinical examination and is of physiopathological order (21). Pyloric stenosis is more brutal and hinders the passage and absorption of ingested and secreted liquids (saliva, gastric juice) and electrolytes in the intestine. This leads to dehydration, metabolic alkalosis (non-reabsorption of H+ ions) with hypokalaemia (non absorption of dietary K+ with continued renal elimination). It should be emphasized that dehydration may still be increased by temporary diuresis induced by alkalosis and that reflux of the contents of the abomasum towards the forestomachs may lead to hydrochloric acidosis of the rumen.

Figure 2: Pathogenesis of recurrent bloat in calves during weaning (10)

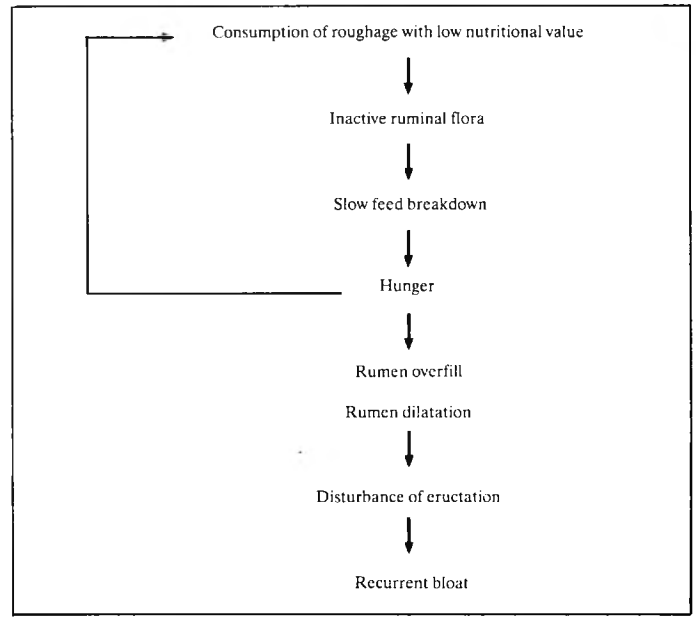
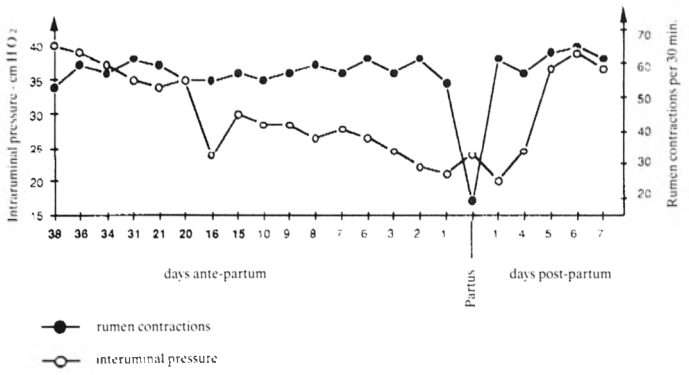


Figure 3: Frequency of rumen contractions and intrarumial pressure around partus (7)



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