CHAPTER I : ORAL AFFECTIONS IN COWS

Handling cattle is a daily practice in farming. It is used in all interventions on animals. Approach techniques are essential for isolating an animal and guiding it into the restraint setup. Halters and tie knots can help improve the efficiency of restraint equipment. Finally, approach and contact techniques can be used to calm the animal during the intervention. Cattle handling practices are based on a good knowledge of animals. The equipment is simple, but it must be used taking into account the animals' behavior. Contact with the animal must be done calmly, respecting the safety of the handler and the well-being of the animal.

1. CONGENITAL AFFECTIONS

1.1 Agnathia

Lack of development of the lower jaw. Severe mandibular hypogenesis and agnathia are, however, very rare in large ruminants; these extreme anomalies are mainly encountered in the Jersey breed (Leipold, 1996).

1.2 Prognathia

Lack of development of the upper jaw. As in many species, vitamin D deficiency in the fetus causes congenital rickets which manifests clinically by swelling of the limb joints, skull deformation, maxillary prognathia, and dental abnormalities. In adults, hypovitaminosis D causes osteomalacia with bone softening, muscle spasms, and hypotonia (Amstutz et al, 2002, Mouthon, 2000, Radostits et al, 2000, cited by Duval-Desnoes, 2005).

1.3 Palatoschisis

The palatine cavity may have a single or double fissure. It is an absence of tissue in the buccal vault leading to communication between the nasal cavities and the mouth. It is a cleft of the hard palate that is more or less wide and extends backward.

2. STOMATITIS

2.1 Catarrhal Stomatitis

This is a diffuse inflammation of the buccal mucosa. It results from direct aggression by a physical or chemical agent. The animal salivates excessively (ptyalism) without any apparent lesion. Inflammation can be seen through a more pinkish color. This is a primary stomatitis that is infrequent.

2.2 Papular Stomatitis

Bovine papular stomatitis is a benign viral disease characterized by the appearance of proliferative or erosive oral lesions, located mainly on the muzzle, lips, gingival margin, tongue, and palate. It primarily affects young bulls and heifers and is transmissible to humans. Caused by a DNA virus of the genus *Parapoxvirus* related to the bovine pseudo-cowpox virus. It appears following immunosuppression. Papular lesions are found that resemble vesicles but are firmer with epidermal elevation. They are found on the muzzle, lips, gingival margin, and palate. It needs to be recognized

because it is a zoonosis (it causes lesions on the hands called "milker's nodules"), but also to avoid confusion with foot-and-mouth disease.

It is a viral disease (parapoxvirus) that is benign. However, the calf may die of starvation because it stops feeding. It is a disease affecting young cattle (aged 2 weeks to 2 years) but especially calves under 6 months.

Transmission is direct or indirect (via drinking water or feed). The lethality of the disease is low but morbidity can reach 100% as the disease is highly contagious. The incubation period (between infection and the appearance of clinical signs) is 2 to 5 days. The first signs are:

- Appearance of erythematous foci (=redness) on the muzzle, nasal wing, and lip. The lesion may have a rosette appearance (red outline and white center).

Lesions very similar to those seen in pseudo-cowpox are found in calves that have suckled infected cows, the two diseases being otherwise very similar (Jegou-Penouil et al, 1997). For pseudo-cowpox, there is no specific treatment, only symptomatic treatment with the use of local antiseptics.

There is no effective vaccine; measures are solely sanitary, with quarantine of purchased animals (Sylvain, 2008). Streaks of redness on the buccal mucosa and absence of hyperthermia are noted; then there is mild hypersalivation, and the erythematous foci transform into flat, yellowish papules. Then after 16 days, the old lesions have disappeared.

Gloves must be worn during clinical examination because it is a zoonosis. To confirm the diagnosis, complementary examinations can be performed:

- Histology, which is very rarely done in practice,
- Virus identification by electron microscopy.

This disease can be confused with:

- Foot-and-mouth disease. For differential diagnosis, we look at the teats and feet. If no lesions are seen, it is not foot-and-mouth disease. Age is also taken into account. The two diseases do not affect the same age group. Here, young cattle are affected.
- Other stomatitis.

Healing is spontaneous, but it can be treated with iodinated glycerin. Transmission occurs through direct contact, so animals must be separated.

2.3 Vesicular Stomatitis

2.4 Ulcerative Stomatitis

This is oral inflammation characterized by the presence of ulcers leading to tissue loss in the mouth. The ulcers are superficial and very red and lead to the formation of a false membrane. It is very often associated with a systemic infectious phenomenon, more specifically malignant catarrhal fever (coryza gangrenosum) in cattle.

At autopsy, lesions are found mainly in the digestive tract. Ulcers of the oral cavity, visible during the animal's life, continue into the esophagus: shallow longitudinal erosions are found there, as well as in the mucosa of the forestomachs. The abomasum shows the same lesions, although often

more marked. In the intestine, signs of catarrhal enteritis are observed, as well as erosions at the Peyer's patches (Boss, 2009).

2.5 Croupous Stomatitis: Necrobacillosis or Canker

This is an ulcerative condition (localized to the skin and mucous membranes) caused by *Fusobacterium necrophorum*, a Gram-negative anaerobic bacterium. The stomatitis is necrotizing, affecting calves aged 2 weeks to 3 months, and calves may also have stridulous laryngitis if they are under one year old. The presence of a putrid odor, due to the smell of necrosis. Deep ulcers covered with a false membrane and necrotic deposit develop on the cheeks, gums, or tongue. The croup (false membrane) develops significantly. If laryngitis is present: there will be a moist, painful cough, severe dyspnea, fever, difficulty swallowing, and laryngeal sounds. This is called stridulous laryngitis.

Treatment involves:

- Debriding the lesion, i.e., removing all necrotic tissue and leaving healthy tissue.
- Administering antibiotics (penicillins, tetracycline, sulfonamides).
- Applying Iodinated glycerin to the lesion.

3. TONGUE AFFECTIONS

Possible conditions:

- Glossitis (inflammation of the tongue),
- Glossoplegia (paralysis of the tongue),
- Macroglossia (abnormal increase in tongue volume).

3.1 Macroglossia

This is a congenital disease characterized by hypertrophy of the tongue. Sometimes, it occurs during muscular hypertrophy ("double-muscled" calf) in Charolais and Belgian Blue breeds, for instance, preventing suckling without assistance and, due to the strong growth potential of the animals, leads to malnutrition or growth defects. The tongue protrudes constantly but remains satisfactorily mobile (Duval-Desnoes, 2005). It might perhaps not be so surprising to note that macroglossia depends less on genes enhancing the effect of the mh gene for muscular hypertrophy on the tongue muscles than on this environment severely deficient in selenium (Frederic and Guyot, 2003).

Jaw deformation may be possible. Calves have difficulty suckling and are more susceptible to many diseases because they do not properly ingest colostrum. They are therefore at risk of dying from starvation or diarrhea

3.2 Actinobacillosis or Wooden Tongue

It's not only the tongue that is affected; other organs are too. The name of the bacterium must be known: *Actinobacillus lignieresii*. (Memory aid: this disease is also called wooden tongue, and wood contains lignin, which sounds like lignieresii). All ruminants are affected: cattle, sheep, and goats. Young cattle (growing animals) are more affected. Calves, on the other hand, are not affected because they drink milk and therefore have no lesions in the mouth. The bacteria enters through trauma to the mucosa and then multiplies. Granulomatous lesions develop (with necrosis and

suppuration), followed by pus formation. Since it is a bacterial infection, it will be managed by antigen-presenting cells. The draining lymph nodes enlarge. The infection remains localized to the initial area.

- The tongue, if severely affected, will protrude from the oral cavity. It is inflamed, hard, and not very mobile.
- As there are lesions on the tongue (in the deep part), the animal will have difficulty swallowing, will drool, and will have difficulty eating (hence anorexia).
- Upon inspection of the oral cavity, pain on manipulation, swelling of the infected area, budding and inflammatory lesions in the soft tissues, and granular pus are observed.
- The nodes are enlarged, with the presence of granulomas, which aids in differential diagnosis.

Clinical Diagnosis:

- Ptyalism, anorexia, hanging tongue – these are the signs
- Hard tongue, presence of granulomas.
- Lymph nodes draining the region are enlarged (Important for differentiation from actinomycosis).

Therapeutic Diagnosis:

Treat for the suspected disease; if the cow recovers, the diagnosis was correct. Here, a good response to iodide treatment is indicative of actinobacillosis.

Differential Diagnosis:

- Salivation: could suggest rabies, presence of a foreign body in the mouth or esophagus, or stomatitis.
- Enlarged lymph nodes: tuberculosis testing (but in that case, there is no tongue issue)
- Deformation of the intermandibular space: actinomycosis, edema of the intermandibular space (to check for edema, press with thumb; if the thumbprint remains, it is edema).

Treatment:

- Iodides are used to dry out the granulomas, causing them to rupture, allowing antibiotics to enter.
- Potassium iodide can also be administered orally, but efficacy is lower.
- Then antibiotics are used, possibly penicillin or streptomycin.
- Surgery can also be performed, but it is rarely done. Response to treatment is fairly good.

This is a sporadic disease, so prevention is complicated.

4. AFFECTIONS OF THE TEETH AND JAW

4.1 Actinomycosis

Actinomycosis, also called "lumpy jaw," is caused by a Gram-positive bacterium: *Actinomyces bovis*. The bacterium affects bone and thus hard tissues. This is the basis for differential diagnosis. The disease is sporadic. It has a very poor response to treatment because it is very difficult to deliver antibiotics to the bone.

Initial trauma is necessary for inoculation of the germ; the bacteria enters through wounds in the buccal mucosa or through dental alveoli (during tooth eruption). Granulomatous lesions develop (at the inoculation site and jaws), potentially leading to problems with mastication and feed prehension.

Symptoms:

- Bony swelling, hard, painful for the animal.
- Difficulty prehending food.
- Very moderate lymph node response because it involves bone. Therefore, no enlarged lymph nodes.
- Sticky, viscous pus may discharge.

5. AFFECTIONS OF THE ESOPHAGUS

- Esophagitis (inflammation of the esophagus)
- Esophageal stenosis (narrowing of the lumen of the organ)
- Esophageal diverticulum (dilation of a portion of the esophagus)
- Functional disorders
- Megaesophagus
- Esophageal obstructions (foreign body)

5.1 Esophagitis

Possible causes:

- BVD (mucosal disease form)
- IBR
- Malignant catarrhal fever (coryza gangrenosum)
- Necrobacillosis
- Trauma (esophageal intubation)

Symptoms:

- Sometimes dysphagia (swallowing problem) with feed rejection
- Nail-slit ulcers.

Esophagitis is often asymptomatic; it is discovered at autopsy.

The course of the disease depends on the cause: healing is possible, or gangrene may occur.

5.2 Esophageal Obstruction

Intraluminal obstructions (the most common) can occur due to a foreign body, or extraluminal obstructions can arise from an abscess or a lymph node pressing on the esophagus.

If a ruminant is given a large, uncut piece of feed, obstruction can occur. Blockage preferentially occurs in 3 narrowed zones: the exit of the pharynx, the thoracic inlet, and the cardia.

Symptoms: Depend on the degree of obstruction.

- If complete obstruction:
  - Onset: agitation, anxiety, head extension, anorexia.
  - Then rapidly: ptyalism, efforts to swallow and regurgitate.

- Then: rumen bloat (observable swelling on the left).
- Foreign body sometimes palpable in the esophageal groove.
- If the animal cannot expel gas, the rumen will swell. If the rumen swells, it compresses the vena cava and lungs, and the cow asphyxiates.
  - If incomplete obstruction:
- Similar onset
- Salivation
- Intermittent bloat

Treatment:

Remove the foreign body (FB) and prevent bloat. In practice, this order is reversed. Bloat must be prevented immediately, then address the FB. Trocarization of the cow is necessary. The priority is that the cow survives. Only afterward can removal of the FB be considered. If the FB is in the anterior cervical region, it can be brought up by external taxis (external pressure on the esophagus), gradually moving it up. An esophageal probe can also be used to push the foreign body down, but there is a risk of damaging the esophagus. The esophagus heals poorly (esophagotomy is strongly discouraged).

7. INDIGESTION – INRUMINATION – BLOAT

7.1 Indigestion

Indigestion is defined as the cessation of reticulo-ruminal motility, resulting in a failure of digestive transit.

They are most often diagnosed in cattle after weaning, especially in adults. Causes of indigestion can be classified into 3 groups:

Group 1: Conditions not located in the reticulo-rumen

- In this case, the cessation of motility is secondary.
- Certain general infectious diseases.
- Esophageal conditions, especially obstructions.
- Abomasal conditions.
- Peritonitis.
- Certain neuromuscular diseases (tetanus, milk fever).
- Severe febrile or inflammatory syndrome.
- Traumatic reticulo-peritonitis.

Group 2: Conditions located in the reticulo-rumen

- T.R.P. (Traumatic reticulo-peritonitis)
- Vagal indigestion or Hoflund syndrome.

Group 3: Disorders of ruminal fermentation affecting rumen content

- Simple indigestion,
- Acute lactic acidosis,

- Acute frothy bloat

Conditions of the wall and fermentation disorders are often reciprocally linked.

7.2 Inrumination

- Inrumination is defined by the absence of the phase of chewing food returned from the rumen to the mouth to undergo reduction of fiber particle size and salivation.
- Causes: Overlap with causes of indigestion and can be classified as extradigestive (febrile syndromes, painful) and digestive, including all conditions of the reticulo-rumen. The combination of rigorous clinical examination and judicious complementary tests (ultrasound, blood biochemistry) allows good diagnostic reliability in bovine gastrointestinal conditions justifying surgical intervention (Schelcher et al, 2013).

7.3 Bloat

- Bloat is a symptom characterized by gas accumulation in the reticulo-rumen: the bovine "swells," or "balloons."
- Gas is:
  - in free form (gaseous bloat).
  - trapped in many small bubbles, forming foam (frothy bloat). Rumen distension in the dorsal region causes, in less marked cases, filling and then bulging of the left flank hollow (apple-shaped left abdominal profile). When bloat is severe, rumen distension is such that the abdomen is dilated on the left upper and lower regions, but also on the right. Bloat is related to a failure to eructate gases produced in the rumen.

A. Acute Bloat

It can be gaseous or frothy.

- Acute gaseous bloat is associated with many causes of indigestion, including:
  - ruminal origin (wall lesions or fermentation disorders)
  - extraruminal origin, such as:
    - Hypocalcemia (milk fever),
    - Peritonitis,
    - Pharyngeal lesions,
    - Esophageal obstruction,
    - "Cardia drowning".
- Acute frothy bloat is almost always due to dietary origin, with the consumption of alfalfa or grasses at a very early growth stage.

B. Chronic Bloat It is always gaseous and can evolve continuously or recurrently, with alternation of phases of ruminal distension and normal phases. In recurrent gaseous bloat, the bovine "balloons" after a meal, then the distension reduces until the next ingestion. In adults, conditions causing chronic bloat are often located in the wall of the reticulum or rumen: (traumatic reticulo-peritonitis, abscess, tumor, actinobacillosis, paramphistome infestation, listeriosis, tetanus).

The most important scientific words translated into English (Chapter 1)

Terme Technique (Français)	Traduction Scientifique (English)
Agnathie	Agnathia
Prognathie	Prognathism
Palatoschisis	Cleft palate (Palatoschisis)
Stomatite catarrhale	Catarrhal stomatitis
Ptyalisme	Ptyalism (excessive salivation)
Stomatite papuleuse bovine	Bovine papular stomatitis
Zoonose	Zoonosis
Stomatite vésiculeuse	Vesicular stomatitis
Stomatite ulcéreuse	Ulcerative stomatitis
Stomatite croupale / Nécrobacillose	Croupous stomatitis / Necrobacillosis
Fusobacterium necrophorum	*Fusobacterium necrophorum*
Dyspnée	Dyspnea
Glossite	Glossitis
Glossoplégie	Glossoplegia
Macroglossie	Macroglossia
Actinobacillose (Langue de bois)	Actinobacillosis (Wooden tongue)
Actinobacillus lignieresii	*Actinobacillus lignieresii*
Granulome	Granuloma
Actinomycose (Gros museau)	Actinomycosis (Lumpy jaw)
Actinomyces bovis	*Actinomyces bovis*

Terme Technique (Français)	Traduction Scientifique (English)
Œsophagite	Esophagitis
Sténose œsophagienne	Esophageal stenosis
Diverticule œsophagien	Esophageal diverticulum
Mégasophage	Megaesophagus
Obstruction œsophagienne	Esophageal obstruction
Indigestion	Indigestion
Inrumination	Inrumination (failure to ruminate)
Météorisation (ballonnement)	Bloat
Acidose lactique aiguë	Acute lactic acidosis
Méthémoglobine	Methemoglobin

CHAPTER II: GASTRIC SECTOR DISORDERS

1. FEEDING BEHAVIOR DISORDERS

1.1 Appetite Disturbances

1.1.1 Increased Intake Level

Normal appetite in ruminants is 2 to 3 kg of DM/100 kg body weight. Increased appetite (bulimia or hyperorexia) manifests as polyphagia (the animal eats excessively). The increase can be primary or secondary.

Primary: physiological

Polyphagia can be a consequence of starvation (due to stomach hypermotility from emptiness). It also appears within 2 months following calving. Intake level also increases when the animal is presented with palatable feeds (e.g., cabbage).

Secondary: mainly pathological

It is observed in paratuberculosis (diarrhea), in cases of internal parasitism, and during convalescence from infectious diseases.

1.1.2 Decreased Intake Level

Inappetence (partial absence of appetite) and anorexia (complete absence of appetite) result in anophagia.

Primary: physiological

It is observed in the last 2 months of gestation.

Feed also plays a major role in anophagia (very unbalanced E/N ratio). A single deficiency can decrease appetite and thus cause multiple deficiencies (e.g., cobalt deficiency).

Treatment to increase appetite:

First, correct the primary cause. Then administer digestive stimulants that may bring improvement.

- Strychnine orally: 10 to 20 g
- Parasympathomimetics parenterally:
  - Pilocarpine: 100 to 200 mg SC
  - Vitamin B1: 10 mg/kg IM
- Rumen seeding (feed bolus or 1 to 2 liters of rumen fluid)

1.1.3 Pica or Allotriophagy

Pica is the tendency to ingest non-food substances, caused by deficiency disorders, organic diseases, or psychological issues. These disorders can be sporadic or enzootic (due to deficiency). Sometimes they are due to monotonous feeding lasting too long. Clinically, the animal licks walls, bark, trees, the skin of conspecifics, and sometimes ingests manure, bedding, and slurry. Sheep and lambs lick and nibble wool soaked in urine or feces of their neighbors.

Pica can be due to:

1.1.3.1 Deficiency Disorders: The most common. These disorders can be primary or secondary.

- *Primary disorders:* Represented by intake deficiencies (energy, nitrogen, minerals) quantitatively or qualitatively (especially for minerals: copper and cobalt). The predominant cause is sodium deficiency due to excess potassium in the feed (potassium fertilizer, molasses-based feed). In energy deficiency, pica is especially observed in acetosis of dairy cows.
- *Secondary disorders:* Represented by absorption disorders observed in gastrointestinal parasitism and paratuberculosis.

1.1.3.2 Organic Disorders: Due to chronic abdominal pain like chronic abomasitis, traumatic reticulo-peritonitis-pericarditis.

1.1.3.3 Psychological Disorders: Imitation of deficient animals.

In case of salt, protein, and vitamin deficiencies, animals try to replace the missing element with substances they find around them. This is why animals sometimes pick up objects that are of no use to them, which frequently leads to digestive disorders due to foreign bodies in cattle.

Diagnosis: Based mainly on clinical signs.

Prevention: Check diet balance and rationing.

Treatment:

- Allow the animal free access to a salt lick block.
- For copper, the recommended intake is 10 mg/kg DM.
- Cobalt supplementation of around 0.04 mg/kg DM can also be recommended, especially if cobalt utilization is blocked by excess molybdenum.
- Add vitamins: A D3 E
- Trenbolone acetate (anabolic agent) (bovine: 100 mg, ovine: 10 mg)
- Phosphorus injection: 14 mg/10 kg.

1.1.4 Licking

Lickomanias are diseases manifested by compulsive licking of conspecifics or self-licking. It is poorly understood. The frequency of the condition is much higher when suckling time is reduced during early life, as in single feedings. Too small a living area seems to increase boredom and the onset of the disease. Copper deficiency (indirect effect on iron deficiency) also causes this behavior.

Animals that suckle each other can cause chapping or skin infections of the udder.

There is no real treatment once the bad habit is established; halters or "anti-suckling" rings can be used. Tranquilizers are effective.

To prevent the disease, it is necessary:

- Increase daily amounts of milk or milk replacer;
- Provide 2.5 m² of space per heifer at weaning; pass through the group often;
- Supplement milk replacer with 15 mg of copper sulfate per kilogram.

2. TRAUMATIC RETICULO-PERITONITIS (T.R.P.)

Definition

It is a serious condition caused by the embedding of a sharp, penetrating, septic foreign body in the reticulum wall. It results in a painful syndrome, signs of indigestion with a usually slow progression towards healing, rarely towards death, and often towards chronicity.

It is also referred to as traumatic gastritis or indigestion, gastritis due to foreign body, T.R.P. Its economic importance: about 12% die from T.R.P., and about 50% of slaughtered cattle carry lesions. Its medical importance is mainly due to the resulting indigestion and its chronicity. Species affected are mainly cattle, exceptionally sheep. It is a disease of both extensive and intensive farming. Its resurgence occurred after the use of wire for fences or hay bales. It appears especially during the three months following calving when finely divided feeds are used. It appears mainly due to the farmer's inattention or negligence, leaving foreign objects within reach of the animals.

Etiology

The reticulum is located just below the cardia, and the reticular surface makes metal particles fall into it easily; being dense, they remain there. The body in the reticulum (e.g., a nail) will embed itself in the anterior part after reticular contraction, as the posterior wall moves forward.

The foreign body is generally metallic. T.R.P. can occasionally occur with increased intra-abdominal pressure, at the end of gestation, during expulsive efforts of parturition; sometimes during pica due to Ca, P deficiency, and in dairy cows with ketosis.

There is ingestion, then perforation, and then migration of the foreign body, causing various lesions, but especially traumatic pericarditis. These are painful and digestive manifestations, with cessation of motor phenomena in the forestomachs. Atony spreads to the entire digestive tract via sensory nerve impulses inhibiting the bulbar center due to the painful manifestations.

a/ Peracute form: Rare. It starts very abruptly with hyperesthesia, anxiety. The animal rolls on the ground due to colic. Respiration is accelerated, with hyperthermia (40 to 41°C). The course rapidly leads to healing or the acute form.

b/ Acute form:

- **2-1 / Initial phase:** The animal separates from the herd, is anxious, shows stiffness, with a sudden drop in milk production. Then comes the established phase.
- **2-2 / Established phase:** Manifests with several different symptoms:
  - **Painful manifestations:** The animal exhibits particular postures in gait, recumbency, and emits a groan interrupting expiration.
- **Particular postures:** It presents a hunched back, with the apex at the withers (6th and 7th thoracic vertebrae), to reduce pain.
- The limbs are gathered under the body, the head extended, held low, elbows abducted. Respiration is short and shallow.

- **Gait:]** It moves without mobilizing the spine; the animal moves as a whole. It refuses to go down a slope or turn on its forelimbs.
- **Recumbency:** Is rare. The animal makes efforts to lie down, hesitates. Sometimes lies down, first bending the hindquarters. Then recumbency is very prolonged.
- **Urination and defecation** are painful, performed with moans.
- **Respiration** is costal and shallow.
- **Digestive symptoms:** Anorexia, inrumination, slight bloat, and constipation.
- **Palpation and auscultation:** Atony of the anterior gastric sector, then the entire digestive tract.
- **General symptoms:** Moderate hyperthermia (39.5°C), slight tachycardia (< or = 80 to 90 beats/min).
- **Withers sign:** Pinching the withers makes the cow arch its back. Bracing accentuates the pain. By pinching the withers between gathered fingers and the palm, this compresses the abdominal area where the foreign body is embedded (Schelcher et al, 2013). The visceral serosa of the reticulum is red, with localized fibrin patches. Sometimes the foreign body is free in the reticulum (moved backward). Blood analysis shows hyperleukocytosis (10,000 to 15,000 per mm^3) with neutrophilia.

The foreign body may return to the reticulum and gradually dissolve. There may be complications of traumatic pericarditis or progression to chronicity. Death can occur after generalized acute peritonitis; there is alteration of the His bundle and coronary arteries.

c/ Chronic form

It manifests as poor general condition, decreased milk production, capricious appetite, dull and dry hair, and indigestion with chronic overload. In this form, diagnosis is difficult using specific tests. The course leads to emaciation or Hoflund syndrome. There is presence of fibrous adhesions between the reticulum and the diaphragm, spleen, liver,... Often, there is materialization of the path taken by the foreign body (fibrous cord), sometimes interspersed with abscesses. On section, we note a fibrous envelope with brownish magma (digestion by oxidation of the foreign body). Hematology shows slight hyperleukocytosis without neutrophilia, but monocytosis (7 to 10%) and eosinophilia (5 to 10%).

Diagnosis

- **Positive:**

** Acute form: The following investigations should be undertaken:

- Withers sign: kyphosis gives lordosis with a groan.
- Stick test: a solid stick of large diameter is placed transversely under the animal, in the xiphoid region, just behind the point of the elbow. It must be lifted slowly but vigorously and then suddenly released; repeat the maneuver over the entire area between the xiphoid appendix and the umbilicus; groan if painful.
- Palpation-pressure, percussion (gong) with a heavy hammer.
- Electro-magnetoscopic detection.

- Hematological examinations.

1 -2/ Chronic form

The animal presents poor general condition. In this form, attempts will be made to perform:

- Diet-meal test: put the animal on a diet for 48 hours, then give it forage. Reactivation of motor phenomena occurs, resulting in pain and groans.
- Leukocyte formula.

2- Differential: The disease should be differentiated from:

- Abomasal ulcers.
- Generalized peritonitis (of digestive or genital origin).
- Immunological bronchopneumonia, in this case, withers sign is often positive.
- Abdominal hypersensitivity sometimes at the end of gestation (large fetus).
- Ketosis (in the month following calving for high-producing dairy cows).
- Abomasal displacements.

Prognosis: Guarded. Recovery is infrequent. Often complications of pericarditis.

Treatment

There are two therapeutic approaches to T.R.P.: medical (conservative) or surgical (rumenotomy) (Radostits et al, 2000). Each has its benefits, and each T.R.P. case must be evaluated individually by the practitioner to determine the most appropriate treatment.

A. Conservative Treatment

Economic constraints and the satisfactory effectiveness of medical treatment have now extremely limited surgical indications. Administration of a magnet and antibiotics, most often accompanied by anti-inflammatories, form the basis of T.R.P. treatment. In practice, the cow is isolated and confined, for example, in a box, to limit its movements and facilitate adhesion formation.

To prevent the weight of the abdominal mass from pressing on the reticulum, it is preferable to place the forelimbs on an incline 20 cm high (Radostits et al, 2000). Broad-spectrum antibiotic therapy administered parenterally for three to five days is recommended to control peritonitis. The most commonly used antibiotics are penicillins, oxytetracycline, and ceftiofur (Streeter, 1999; Radostits et al, 2000). Routes of administration are usually intramuscular and intravenous. The intraperitoneal route offers no advantage over the previous ones, as the inflammatory process makes the peritoneum permeable to antibiotics, which then distribute rapidly via the bloodstream. A magnet should be administered to attempt to trap and neutralize the foreign body in a fasted animal. To be swallowed, the magnet must be placed behind the base of the tongue to avoid any regurgitation reflex. A five to six-hour delay is then recommended before feeding the animal again.

B. Surgical Treatment

Before any rumenotomy, the diagnosis of T.R.P. must be confirmed by exploratory laparotomy via the left flank. The wall of the dorsal sac of the rumen is then opened, and part of the rumen contents is emptied, then exploration of the reticulum is performed to determine areas of

adhesion and search for the foreign body (which may sometimes not be found, e.g., during migration out of the reticulum or if it is enclosed in adhesions, or due to corrosion) (Remy and Ravary, 2004). This treatment has the advantage of providing both satisfactory treatment and a definitive diagnosis. Although it is the best treatment, rumenotomy is not always essential.

Treatment

- **Hygienic:** Put the animal on a diet for 24 to 48 hours while raising its forequarters.
- **Conservative:** This treatment consists of raising the forequarters; the sick animal must be restrained from leaving the platform by its chain and side barriers; transverse bars prevent the animal from slipping. Elevation of the forequarters changes the direction of penetration of the foreign body, causing it to fall back into the reticulum.
- **Medical:**

  In treatment, the following can be used:

  - **Anti-infectives:** sulfonamides and antibiotics.
    - Sulfonamides: sulfadimerazine: 12 to 20 g/100 kg BW (33%: 3 ml/10 kg) sulfamethopyridazine (Sultirene) bovine: 4 to 5 g/100 kg IM or orally. ovine: 500 mg/10 kg
    - Antibiotics: peni-strepto: 5 M-5 g to 10 M-10 g for a bovine. 1 to 2 M and 1 to 2 g for a sheep; terramycin: 5 g intraperitoneally. Repeat 3 times at 24 to 48-hour intervals.
  - **Enzymatic anti-inflammatories:** these are substrates that lyse fibrin. Alpha-chymotrypsin can be injected IM at a dose of 10 to 20 ml/100 kg for 3 days.
  - **A magnet:** to be administered orally.
- **Surgical:** Surgical treatment by left laparotomy in T.R.P. Exceptional. Only if the animal is of great value, with temperature less than or equal to 39.5°C, pulse less than or equal to 80-90 beats/min, the accident is less than 10 days old, and there is no pericarditis. In this case, perform a left laparotomy after a 24-48 hour fast.

b/ Chronic form

Treatment gives no results; send the animal to slaughter.

Prophylaxis

Do not leave wires lying around; check for foreign bodies in silage. For highly exposed animals, administer permanent magnets to all (6 to 7 cm^2 long and 1 cm^2 wide). In practice, the best therapeutic approach would be to treat the bovine medically for at least three days and then, if no signs of improvement are seen during this period, perform a rumenotomy (Radostits, 2000; Rémy and Ravary, 2004). The best way to reduce the number of T.R.P. cases in herds is, evidently, the preventive administration of magnets. This is a true prophylactic measure in animals older than eight months. Thus, according to one study, the incidence of T.R.P. decreased by 90% in cattle over eighteen months old that had ingested magnets (Poulssen, 1976). Generally, the administered magnet is 7.5 cm long and its diameter varies from 1 to 2.5 cm. It is preferably encased in a plastic structure to prevent adhering foreign bodies from remaining sharp and thus capable of causing wall trauma.

The most important scientific words translated into English (Chapter 2)

Terme Technique (Français)	Traduction Scientifique (English)
Boulimie / Hyperorexie	Bulimia / Hyperorexia
Polyphagie	Polyphagia
Anorexie	Anorexia
Pica / Allotriophagie	Pica / Allotriophagy
Carence (cuivre, cobalt)	Deficiency (copper, cobalt)
Lichomanie	Lickomania (compulsive licking)
Rétículo-péritonite traumatique (R.P.T.)	Traumatic reticulo-peritonitis (T.R.P.)
Corps étranger	Foreign body
Péricardite traumatique	Traumatic pericarditis
Atonie (du rumen)	Atony (of the rumen)
Signe du garrot	Withers sign
Hyperleucocytose	Hyperleukocytosis
Neutrophilie	Neutrophilia
Rumenotomie	Rumenotomy
Laparotomie exploratrice	Exploratory laparotomy

CHAPTER III HYDROCHLORIC ACIDOSIS (OMASAL REFLUX)

1 Definition

Hydrochloric acidosis of the rumen is linked to a phenomenon of high obstruction (at the level of the abomasum or duodenum) or generalized gastrointestinal stasis (Hoflund syndrome). The obstruction prevents the passage of chyme from the abomasum to the intestine and leads to reflux of omasal content rich in hydrochloric acid into the rumen. This results in acidification of the rumen content (chloride content of rumen fluid increases from 15 to 25 mEq/l, exceeding 30 mEq/l and up to 100 mEq/l). pH varies from 6.8 to 4.4. Injection of apomorphine (emetic for carnivores) causes a "pseudo-vomiting" of abomasal contents into the rumen.

2 Etiopathogenesis

The hydrochloric acid from omasal reflux corresponds to acid lost, non-recoverable in the intestine, for the plasma, resulting in metabolic blood alkalosis with hypochloremia (50 instead of 95 mEq/l). This is accompanied by hypokalemia (2 instead of 4-5 mEq/l) due to anorexia (decreased intake of forage rich in K; an adult herbivore ingests 3000 to 6000 mEq/l/K+/day) and leakage of K+ into the extracellular space. During metabolic alkalosis, the kidneys react by conserving H+ and Cl- and producing alkaline urine.

Paradoxically, in cattle with hypokalemia associated with metabolic alkalosis, aciduria is observed (renal reabsorption of Na+ in exchange for H+).

Prognosis: Unfavorable.

Treatment: Consists of administering laxatives (Mg hydroxide: 0.5 to 1 kg/day). Then inject Ca gluconate subcutaneously to increase abomasal motility.

To combat metabolic alkalosis and hypokalemia, intravenous solutions rich in NaCl and KCl can be administered (8 to 10 liters of 0.85% NaCl solution, 4 liters of 1.1% KCl solution, not exceeding 3 mEq/kg body weight).

After this emergency treatment, rumenotomy may be performed in cases of partial abomasal obstruction to inject Mg sulfate directly into the abomasal cavity. This rumenotomy also allows massage of the abomasum.

In severe cases of total obstruction, right laparotomy should be considered, possibly followed by abomasotomy or pyloromyotomy.

3 VAGAL INDIGESTION "HOFLUND SYNDROME"

3.1 Definition

This is a disorder of gastric motility due to vagus nerve injury, which can be a consequence of traumatic reticulo-peritonitis, and results in chronic indigestion with or without persistence of rumen motility. This is called Hoflund syndrome.

3.2 Causes

Vagus nerve lesions are caused either by fibrosis interrupting nerve conduction due to traumatic reticulitis, or by compression by a leukotic or actinobacillary lymph node, or by a diaphragmatic hernia leading to vagus nerve stretching.

3.3 Characteristics

Four syndromes are described due to vagus nerve (X) involvement:

1. Functional stenosis of the reticulo-omasal orifice with reticulo-ruminal atony (when both vagus nerves are severed in the thoracic and/or abdominal region);
2. Functional stenosis of the reticulo-omasal orifice with preserved reticulo-ruminal motility (when the ventral vagus is severed at the cardia and the dorsal vagus just after the emergence of the rumen nerves);
3. Functional pyloric stenosis with or without preserved reticular motility (when the ventral vagus is severed at the cardia and the dorsal vagus at the level of the omasum);
4. Incomplete functional pyloric stenosis (with similar, more distal sections).

Hoflund Syndrome Type I:

Stenosis of the reticulo-omasal orifice with rumen hypermotility mainly due to excess secondary contractions. The animal presents poor general condition, distension of the left flank with slight bloat. Progressive distension of the left flank, then the right flank, gives the rumen an L-shape (confirmed by rectal exploration) and the abdomen an apple shape on the left, pear shape on the right. There is softening of the contents and often bradycardia.

The course is often favorable. Treatment is based on alkaloids (e.g., morphine).

Hoflund Syndrome Type II:

Interruption of the main vagal trunks innervating the reticulum and rumen, with stenosis of the reticulo-omasal sphincter and atony. Symptoms are characterized by distension of the left flank, increased consistency of the rumen contents with a tendency to dehydration. The course leads to organic decline. There is no treatment; send the animal to slaughter.

Hoflund Syndrome Type III:

Manifests as atony of the omasum and abomasum with intermittent pyloric stenosis, leading to poor general condition. There is an impact on other sectors with gastrointestinal atony, anorexia, abomasal dilation and overload. The course tends toward recovery if hygienic treatment is used, with evacuants ($MgSO_4$, Na_2SO_4), mild laxatives, and evacuating alkaloids.

Hoflund Syndrome Type IV:

Characterized by atony of the omasum and abomasum with permanent pyloric stenosis, resulting in digestive and absorption disorders corresponding to the following symptoms: poor general condition, anemia, dehydration, emaciation. The abomasum increases in volume and presents overload (palpation at the level of the right flank slope or sometimes transrectal palpation).

Prognosis is grave, as there is no treatment. Send the animal to slaughter.

The most important scientific words translated into English (Chapter 3)

Terme Technique (Français)	Traduction Scientifique (English)
Acidose chlorhydrique	Hydrochloric acidosis
Reflux omasal	Omasal reflux
Alcalose métabolique	Metabolic alkalosis
Hypochlorémie	Hypochloremia
Hypokaliémie	Hypokalemia
Acidurie paradoxale	Paradoxical aciduria
Laparotomie droite	Right laparotomy
Abomasotomie	Abomasotomy
Pyloromyotomie	Pyloromyotomy

CHAPTET IV RUMEN ACIDOSIS INDIGESTION OR ACUTE RUMEN OVERLOAD INDIGESTION

1 Definition

This is a general intoxication of the body resulting from the distribution of a diet too rich in energy, occurring abruptly or slowly, producing an abnormal amount of lactic acid in the rumen. It is characterized, in the acute form, by digestive symptoms, general symptoms, and rapid death; and a latent form (much more frequent), discreet, with extradigestive symptoms.

Ruminal lactic acidosis (as opposed to hydrochloric acidosis, secondary to abomaso-ruminal reflux of HCl) is a consequence of the consumption of a diet rich in rapidly and highly fermentable carbohydrates (RHFC) by cows (which are adapted to digest and metabolize forage-based feed) that stimulates a certain bacterial population, shifting microbial digestion towards the production of lactic acids, particularly lactate (Brugere-Picoux, 1983).

Figure 27: Diagram of carbohydrate fermentation in the rumen [Acetic acid = --- = butyric acid = Propionic acid • • • • = Lactic acid] (Rosenberger, 1977)

2 Importance

- **Medical:** leads to death in acute intoxication.
- **Economic:** considerable, latent acidosis results in decreased ADG (average daily gain), decreased milk production, decreased milk fat percentage.

3 Epidemiology

- **/Acute form:**

The disease appears especially in dairy farming, sporadically and accidentally (e.g., a cow accessing grain reserves) and also after switching from a forage diet to a cereal diet without sufficient transition (young bull).

- **/Chronic form:**

These are enzootic diseases that appear:

- in dairy cows fed corn silage (0.6 to 0.7 UF/kg DM) or in cases of insufficient long fiber.
- in young bulls fed the same way as dairy cows and additionally corn grain, beets, pulp, brewers' grains (1 UF/kg DM).

4 Etiology

These are rapidly available and easily fermentable carbohydrates (R.A.E.F.C.) derived from:

- Cereals: wheat > barley > corn,
  - Immature cereals are more energetic than ripe cereals;
  - Ground cereals > whole grain cereals.
- Potatoes, apples, beets, pulp, brewers' grains.
- Corn silage, which provides starch and a large amount of lactic acid.

Predisposing factors: They act very quickly. These factors include:

- Excessive intake due to miscalculation or measurement error.
- Feed change (replacing one energy-rich feed with another energy-rich feed). For example, freshly harvested cereals are more dangerous than those harvested long ago.
- Dominance of animals: the most vigorous animals are fed first; they are the dominant ones. After the dominants are removed, the others rush to the feed and develop acute acidosis.
- Social facilitation: animals in groups ingest much more than the same animals isolated.
- A rich diet following a restrictive diet.
- Abrupt removal of roughage leads to the latent form.
- Sudden drop in temperature increases intake and consequently leads to acute acidosis.

Many feeds cause lactic acidosis, with, however, very different inductive capacities, demonstrated experimentally (Slyter, 1976). The differences are related partly to the amount of degradable carbohydrates and partly to the fermentation rate. The intensity and speed of microbial fermentation depend on the chemical composition and presentation of the feed. Risky feeds are chemically characterized by high richness in cytoplasmic storage carbohydrates. The most frequently implicated feeds are those rich in starch (cereals) as well as those rich in soluble carbohydrates (fruits, molasses, beet). Water-soluble carbohydrates generate lactate in vitro more quickly than insoluble fractions. The fermentability of starches depends on their origin (higher fermentability for wheat and barley compared to corn and sorghum), preservation method, and vegetative stage (fermentability of immature and ensiled cereal grains compared to dry grains). In vitro lactate production decreases according to the following scale: barley - wheat > lemon pulp > beet pulp - corn > very wet corn > sorghum (Cullen et al, 1986).

Furthermore, in some silages, the presence of preformed lactic acid contributes to rumen acidification. Thus, in corn silage, lactic acid content (normally between 8 and 12% DM) is higher when moisture is high (Sauvant et al, 1999). The organic acid content of silage (mainly lactic and acetic) varies greatly depending on storage conditions - from 300 to over 1000 mM/kg of ingested dry matter (IDM). These values correspond approximately to 10% of the VFAs produced by the same silage during its ruminal digestion. Various feed treatments increase the risk of acidosis. Thus, reducing feed particle size increases the possibilities for microbial attack, and therefore the degradation rate. Chewing time (during ingestion and rumination) decreases, resulting in less salivation, and therefore a reduction in buffering substances arriving in the rumen. The average particle size of a ration must be greater than 4 mm, though uncertainty about this criterion is high. For cereal grains, the risk of acidosis increases in the order: crimped grain, coarse meal, fine flour. Cooking

5 BIOCHEMICAL MODIFICATIONS OF RUMEN CONTENT

5.1 Lactic Acid Accumulation

The decrease in pH during the first 8 hours of acidosis would not be due to an increase in lactic acid quantity, but to an increase in the production of other fatty acids (Satge, 1993). If acidosis

continues, lactic acid concentration peaks 7 to 24 hours after overconsumption of RHFC and then declines. L and D isomers are formed in the same proportions for lactate concentrations of 100 to 500 mosm/L and pH below 5 (Roque, 1991). Lactate concentration in the rumen depends on 4 factors: production, microbial utilization, lymphatic or blood absorption, and evacuation to the lower digestive tract. Absorption only significantly affects concentration at the lowest pH values (below 5-5.5). Although the latter two factors should not be neglected, it seems that the initial accumulation of lactate is mainly linked, following the consumption of large quantities of RHFC, to an imbalance between the production and consumption of this acid, lactate being an intermediate in the production of VFAs.

At physiological pH values, and down to values of 5 to 5.5, germs capable of producing lactate are varied. As seen previously, the majority of these bacteria (R. amylophilus, S. amylolytica, ...) decrease in number as pH decreases, while S. bovis becomes dominant. In response to the large amounts of starch present in the rumen (feed intake and disappearance of storage capacities of rumen protozoa), the lactate dehydrogenase of S. bovis will be activated, allowing the production of lactate at the expense of acetate. Lactic acid production then becomes an outlet for intracellular protons, this production being one of the only mechanisms for controlling intracellular pH (Counotte and Prins, 1981). When ruminal pH reaches values below 5, a drop in intracellular pH is observed in parallel. This drop in pH inhibits pyruvate production by pyruvate formate lyase and stimulates lactate dehydrogenase activity. Lactate formation then increases inexorably. The decrease in pH contributes to creating an ecological niche favorable to lactobacilli. On the other hand, while lactic acid production by rumen bacteria is favored, its utilization is decreased when ruminal pH decreases. The optimum pH for lactate fermentation is 6 to 6.5 for most bacteria, with only M. elsdenii being able to continue its fermentative activity at pH below 5.5. This regulatory mechanism is quickly overwhelmed during acute acidosis, promoting ruminal lactate accumulation. Both lactate isomers, L and D, are produced. When the lacticolytic flora is eliminated (at pH below 5), the evolution leads to the formation of a pool of D-L lactate. This is not metabolized by bacteria and must be metabolized by the ruminant (the D isomer is practically not metabolized) or eliminated in feces. This accumulation also causes an increase in ruminal osmolarity, which rises from a value of 280 mOsm/L to nearly 400 mOsm/L (Radostits et al, 2000). This increase in osmolarity causes water to be drawn from the systemic circulation, leading to hemoconcentration, dehydration, hypovolemic shock, and diarrhea.

5.2 Volatile Fatty Acid Accumulation in Subacute Acidosis

In cattle, following induction of subacute acidosis in animals adapted to a high-forage or high-concentrate diet, parallel to a decrease in pH to values of 5 to 5.5, volatile fatty acid concentrations increased, while lactate concentrations remained normal (below 5.5 mM). The decrease in ruminal pH in dairy cows with subacute acidosis would be more related to an accumulation of volatile fatty acids alone, rather than an accumulation of acid (Goad et al, 1998; Oetzel et al, 1999).

5.3 Other Biochemical Modifications of the Rumen

Other ruminal biochemical modifications, besides the appearance of acidity, occur as a consequence of disturbances in the bacterial balance. Release of bacterial endotoxins has been observed. Thus, cattle fed diets with high proportions of concentrates had higher lipopolysaccharide (LPS) concentrations in ruminal fluid than cattle fed only hay. Other results showed that LPS concentration increased significantly during periods of grain consumption compared to hay consumption. It is likely that subacute acidosis leads to increased lysis of Gram-negative bacteria, which would increase ruminal LPS concentration and initiate the inflammatory response (Nagaraja et al, 1978; Gozho et al, 2005). The observation of thrombocytopenia and increased plasma prostaglandins E2 and F in a cow with acute acidosis is consistent with an inflammatory syndrome initiated by endotoxins (Andersen and Jarlov, 1990). Their toxic potency would, however, be lower than that of E. coli or Salmonella LPS (Roque, 1991). The presence of ruminal wall lesions would facilitate their absorption.

In conclusion, for a dairy cow ingesting 15 kg of barley meal. This quantity passes into the rumen where the microflora (especially Streptococcus bovis) produce VFAs, C2, C3 especially, C4, and also lactic acid. The pH drops from 6 to 5. Rumen acidity causes the disappearance of protozoa and a decrease in cellulolytic germs, especially Gram-negative. Another flora is favored: lactic flora (streptococci and lactobacilli), which, with the remaining starch, produce large amounts of lactic acid. This lactic acid has two forms: an L form easily metabolized and a D form difficult to metabolize in the liver. This exaggerated production of lactic acid and its absorption leads to hyperlactacidemia. This imbalance in the rumen microbial population also causes

a deficiency in vitamin synthesis (B1) and a decrease in phosphorus absorption. Consequently, the accumulation of lactic acid will increase osmotic pressure (twofold), drawing water into the rumen.

This leads to extracellular, then cellular, dehydration. As long as the pH is between 7.5 and 6.0, the buffering system plays its role; balance is ensured by inorganic basic phosphates and carbonates in saliva. But if the amount of acid produced is too great, the neutralization capacity of this buffering system can be overwhelmed. As soon as the pH drops below 6, VFA salts release free fatty acids, which are then rapidly absorbed.

Due to the accumulation of lactic acid, a pH drop from 4.5 to 3.9 is common, representing a 100 to 1000-fold increase in the concentration of H+ ions in the rumen.

6 Metabolic Acidosis

Absorption of lactic acid occurs in the rumen and also in the intestines. This causes hyperlactacidemia; blood pH drops from 7.4 to 7.2, hence a decrease in alkaline reserve (from 25 to 15 mEq/l) and absorption of endotoxins characterized by shock and laminitis.

- **Symptoms**

a/ Acute form

High concentration of undissociated VFAs in the rumen and blood is characterized by gastrointestinal atony (by reflex and hormonal regulation).

Resorption of a large amount of D-lactic acid results in depression and nervous disorders due to central nervous system involvement. Water drawn into the rumen results in decreased diuresis, decreased blood pressure, characterized by tachycardia and initial respiratory acceleration.

Decreased blood pH leads to decreased alkaline reserve and increased partial pressure of CO_2 in the blood. This stimulates the bulbar cardiac and respiratory centers (acceleration of heart and respiratory rates).

Water movement into the rumen leads to extracellular then cellular dehydration with decreased diuresis, decreased blood pressure with tachycardia and respiratory acceleration (tachypnea). Dehydration increases urine density and results in hemoconcentration. Later, when blood pH drops below 7.30, CO_2 transport is decreased and it accumulates in tissues. There is cellular anoxia.

Nerve cells are the most sensitive. These facts explain the adynamia and even coma in animals.

b. Chronic form

There is a metabolic shift towards the production of C3 (propionic and lactic) with increased papillae, agglutination, and then detachment. We move from hyperkeratosis to parakeratosis and ulcers. Liver abscesses are explained by the passage of germs through the damaged rumen wall. There is a decrease in milk butterfat due to decreased production of VFAs C2 and C4.

Atony can lead to abomasal displacement or cecal torsion.

Hyperlactacidemia results in chronic laminitis at the podophyllum.

The frequency of diseases increases, such as enterotoxemia and fat cow syndrome (obesity).

C. Symptoms and Lesions:

- **/ Acute form:** Severe and accidental. It manifests with various symptoms:
  - **General:** represented by ataxia or tetaniform symptoms; with hypothermia at the end of the course, ataraxia (mental calmness), tachypnea, signs of dehydration, and tachycardia.
  - **Digestive:** the hollow and slope of the left flank are distended with a doughy, elastic sensation on external or transrectal palpation; on auscultation, contractions disappear with crackling sounds. pH is between 4 and 5.
  - **Nervous:** generally depressive; characterized by indifference, drunken gait, amaurosis. Sometimes tetaniform symptoms manifested by hyperesthesia, teeth grinding, chewing movements, and locomotor disorders.
  - **Biochemical:** there is an increase in urine density with pH equal to 5 (instead of 8) and lactaturia; hematocrit rises from 30 to 35%. There is a decrease in blood pH from 7.4 to 7.2 and bicarbonate

from 25 to 15 mEq/l. Finally, hyperglycemia due to propionic and lactic acids metabolized into glucose is noted.

The course of this acute form most often leads to death, after a few hours to 2-3 days. Sometimes, healing is heralded by clear, yellowish diarrhea with a sour odor. Temperature returns to normal, locomotor activity reappears. Complications of laminitis and dermatitis may occur.

At autopsy, congestion and sometimes petechiae on serous membranes (non-specific) are noted. In the rumen, the content has a sour odor and is soupy. pH is around 5. Cereal grains are found. The subepithelium is reddish, congested, the mucosa detaches very easily.

Hyper- and then parakeratosis of the rumen: This is increased keratinization leading to hyperkeratosis (papillae are more elongated, brownish, forming a brush-like mat) and parakeratosis (papillae agglomerate, shorten, and form cauliflower-like buttons).

Rumen ulceration: Lactic acid is corrosive, causing degeneration and necrosis of the rumen wall. This manifests as loss of epithelial substance with reddish bases and raised, indurated edges, a few centimeters in diameter. Ulcers require the presence of traumatic elements (e.g., hair) on the weakened epithelium. Ulcers will spread, and germ passage is possible.

Liver abscesses: A slaughterhouse finding; abscesses range from a few millimeters to a few centimeters. No symptoms are visible; these lesions mainly result in decreased performance.

D. Complications

- **Dermatitis:**

  Less frequent; it is eczema of the pulp, brewers' grains. It manifests as raised, congestive plaques with agglutination of hair, ending in crusts.

- **Decreased butterfat**

The increase in propionic acid relative to acetic acid, which is involved in milk fatty acid synthesis, will decrease milk fat. There is a metabolic shift towards fattening; propionic acid, which is glucogenic, causes hyperglycemia, which leads to hyperinsulinemia and inhibition of hormone-sensitive lipase (in the liver), leading to hepatic steatosis.

- **Abomasal displacement and cecal torsion**

These displacements are enabled by atony of these organs. Latent acidosis provides many undissociated VFAs, which cause atony (by reflex and humoral regulation).

- **Risk of cerebrocortical necrosis**

Given the decreased synthesis of vit B1 by rumen microorganisms and the high demand for vit B1 in lactic acid metabolism, NCC may occur.

- **Laminitis, a major complication of lactic acidosis**

In acute acidosis, the shift in microbial balance in the rumen leads to the formation of numerous metabolites that are normally scarce or absent. These include: histamine, thiamine, ethyl alcohol, and endotoxins. Under normal conditions, blood histamine levels remain low due to the

presence of histaminase. But histamine production can be so great that it overwhelms the available histaminase.

Also, the blood will carry histamine, especially since the liver itself is overwhelmed. Histamine reaching the podophyllum increases capillary permeability and allows toxic factors (histamine, lactic acid, and products of Gram-negative lysis) to pass into foot tissues.

At this level, mast cell degranulation occurs, and released histamine amplifies the process, resulting in pain and lameness. In the chronic form, the phenomena repeat regularly with peaks of lactacidemia.

In the foot, the metabolism of the keratogenous membrane is disturbed, resulting in alterations in horn quality, with flattening of the foot and outward deviation, with transverse striations and elongation of the hoof.

Diffuse aseptic pododermatitis (this term is preferred to the one borrowed from equine medicine) is defined as inflammation of the pododerm following systemic disorders in cattle. The etiology and pathogenesis of this condition are not yet precisely known or remain debated, but a multifactorial origin of laminitis is accepted. Many factors have already been identified, including:

- Digestive and metabolic disorders (rationing errors, energy/nitrogen/fiber balance);
- Management of animals between calving and the start of lactation;
- Age, growth, and conformation of the animal;
- Acute inflammatory processes (metritis, mastitis);
- Localized hoof trauma;
- Overweight, lack or excess of exercise;
- Housing;
- Season and climatic conditions.
- **Different types of laminitis**

The classification of laminitis episodes as acute, subclinical, subacute, or chronic has been used for several decades to describe them according to severity and duration.

/ Acute Laminitis

Symptoms of acute laminitis correspond to acute inflammation of the pododerm, i.e., vasodilation, congestion, and erythema of the latter, associated with significant pain encountered during the first stage of pathogenesis, during vascular disorders (Forsberg et al, 1997). It is a sudden, severe attack of the pododerm that occurs exceptionally in herds, most often following (accidental) concentrate overload or another condition.

It can affect the front or hind feet and can affect all claws simultaneously. It is characterized by severe, frank lameness, and animals have difficulty walking or even standing. They often have a hunched back, stiff forelimbs positioned behind their normal position, and hind limbs just under the abdomen, giving them a posture also observed in cases of traumatic reticulo-peritonitis (Chen and Wolin, 1979). Finally, heat can be felt in the feet, a digital pulse easily perceptible at the common digital artery, and upon percussion, the claws are generally very painful (Hungate, 1966).

/ Chronic Laminitis

This is a condition that evolves more persistently with much less obvious systemic signs, except for a decline in general condition (Chen and Wolin, 1979).

Chronic laminitis lesions develop some time after an episode of acute or subacute laminitis. They occur mostly on the outer claws of the hind limbs and appear preferentially during the calving period. Characteristic claw deformations are often encountered during such episodes, which then take on a more or less pronounced shape depending on the severity of the condition; the anterior border is curved, growth rings are rough, diverging, and they descend towards the posterior surface. The bulb is high. The coronet is very horizontal, and finally, the sole becomes full.

/ Subacute Laminitis

It follows episodes of vascular damage, hemorrhage, and thrombosis of the pododerm occurring during the second phase of laminitis pathogenesis, during the rotation of the distal phalanx within the horn capsule.

This form of laminitis preferentially occurs two to three months after calving, clinically characterized by a hesitant gait, without true lameness, and preferentially affecting once again the outer hind claw (other claws may, however, also be affected). The claws are not yet deformed at the subacute stage, but may become so if this episode extends into chronic laminitis, which is not inevitable. However, the appearance of yellow and red discolorations in the horn of the sole and white line may be noted, following pododerm irrigation disorders common during calving. In subacute laminitis, ulcers may also appear on the outer hind claw most often, and the inner claw may also be affected but only by hemorrhages. In this specific case, ulcers cannot be considered as the direct consequence of persistent localized compression (chronic laminitis) but as due to the combination of three elements: the typical location, an irrigation disorder, and hemorrhages. The few scars left by such a laminitis episode are considered normal for cows and heifers around calving (Hungate, 1966; Chen and Wolin, 1979).

/ Subclinical Laminitis

The term subclinical laminitis has long been used to describe the early stage of a laminitis episode, before clinical manifestations on the hoof surface appear. Lesions characterizing such episodes are thus observed during preventive trimming, as they do not cause lameness and are not visible on the hoof surface. It would therefore be more appropriate to call these lesions "sequelae of subclinical laminitis". In such episodes, no change in position is observed, and the gait is normal, without lameness. Indeed, subclinical laminitis, by reducing the quality of the horn produced (yellow, soft horn), predisposes the foot to other conditions such as sole ulcers (circumscribed pododermatitis) common in dairy farming, interdigital dermatitis lesions, leading to lameness (Chen and Wolin, 1979).

- **Epidemiology**

At 18 months of age, about 85% of heifers have lesions on the outer hind claws, of which only 10% carry lesions described as severe. Furthermore, there is a significant difference between the severity of lesions occurring on the outer claw and those on the inner claw.

At 24 months and 30 days after calving, about 90% of heifers have lesions on the outer claws, but these are exclusively severe. Again, there is a significant difference between the outer and inner claw, which for its part is affected by severe lesions in only 20% of heifers.

Finally, at 24 months and 70 days after calving, the situation is critical since 100% of heifers have severe lesions on the outer claw compared to 35% on the inner claw. This study thus highlighted several elements. First, subclinical laminitis is a condition frequently occurring in conventionally managed dairy herds (calves raised on straw bedding then turned out to grass, housed on concrete floors during winter), without major errors in rationing (heifers even receiving less concentrates than recommended). Next, this condition occurs very early in heifers, from 5 months of age, and intensifies with animal age, peaking 30 to 70 days after calving. Deep sole hemorrhages occur as early as one month after calving and develop to appear at 70 days after calving, in the form of diffuse hemorrhages of the sole and white line. They are then always associated with deep hemorrhages at the typical site of ulcer occurrence. It is also noteworthy that all heifers examined then had a concave sole.

- **/ Pathogenesis of laminitis**

From the first publications on bovine laminitis to the present day, many scientists have studied the pathogenesis of laminitis, and several theories (regarding its establishment) have emerged.
It is now certain that episodes of bovine laminitis follow blood circulation disorders occurring in the pododerm that cause its ischemia.

//First stage: vascular disorders in the pododerm.

- **Different hypotheses**

Several hypotheses regarding the exact nature of these vascular disorders occurring in the pododerm of cattle have been formulated and are still debated, including:

- Opening of arteriovenous shunts upstream of capillaries leading to pododerm ischemia;
- Formation of microthrombi inside the capillaries of the pododerm, also causing its ischemia;
- An increase in blood pressure within the capillaries, following vasoconstriction, causing edema, increased interstitial pressure, subsequently leading to pododerm ischemia; (Cheng et al, 1976).

and leading to the opening of arteriovenous shunts. Ischemia of downstream tissues occurs, causing lesions of the pododerm and epidermis (germinal cells) due to insufficient nutrient and oxygen distribution (Nocek, 1997). Thus, the involvement of shunts is suspected by many scientists as playing a very important role in the pathogenesis of laminitis.

- **/Origin of vascular disorders**

Several hypotheses explaining the origin of vascular disorders occurring in the pododerm have been formulated. The most plausible factors regarding the etiology of bovine laminitis are vasoactive substances that would cause a pathological vascular response in the pododerm, leading to claw lesions (Forsberg, 1997). When bovine laminitis was first described in the 1960s, the release of histamine following degradation of dietary protein was presented as the cause of laminitis.

// Histamine release

This release generally occurs following decarboxylation of histidine, contained in protein-rich cereals, into histamine by lactobacilli or by an enzyme of Escherichia coli present in large quantities in the digestive tract (Allison and al, 1975). However, it has not been possible to induce laminitis symptoms by injecting histamine alone into the blood of horses and cattle. Only manifestations related to histamine administration have been observed, such as increased digital pulse, respiratory rate, and dilation of peripheral veins (Chen and Wolin, 1979). Conversely, it was possible to see laminitis symptoms appear by drenching cattle with cereals and then administering a histamine injection (13).
This histamine release may also follow feed contamination by aflatoxins B1, hepatotropic, produced by Aspergillus flavus. Signs of vitamin A deficiency, metritis, laminitis, and calf diarrhea have been described following the hepatotoxicity of these toxins. Indeed, these prevent vitamin A storage, cause immunosuppression (by suppression of specific antibodies) and cause liver lesions, promoting histamine production.

Furthermore, histamine, by its action on smooth muscle fibers, can cause myometrial relaxation, hence the appearance of metritis by penetration of germs from the external environment, leading in severe cases to release of endotoxins into the bloodstream (Agarwal et al, 2002). Finally, histamine can be released during tissue destruction, notably during liver abscesses occurring after penetration of Fusobacterium necrophorum into the bloodstream during an episode of ruminal acidosis. Another explanation regarding the etiology of bovine laminitis could be the release of endotoxins into the bloodstream.

// Endotoxin release

Endotoxins are lipopolysaccharides constituting the wall of Gram-negative bacteria contained in the digestive tract, released during bacterial lysis. They then pass into the bloodstream and initially cause a stress response (hyperglycemia, hyperlactemia, increased cortisol concentration) followed by a dose-dependent response including leukopenia, thrombocytopenia, hypocalcemia, and hypozincemia following activation of the inflammatory cascade (An et al, 2005).

Early stages of laminitis (no clinical symptoms but only primary pododerm lesions on histopathology) by inducing ruminal acidosis in two cows and then injecting them 48 hours later with endotoxins intravenously. During this experiment, it was concluded that endotoxin release caused an increase in norepinephrine accompanied by that of circulating cortisol, which could be the cause of peripheral vasoconstriction. During intravenous administration of endotoxins, the appearance of norepinephrine release within 3 to 15 minutes following intravenous injection was observed. This was followed by an increase in blood cortisol 4 hours after injection, and the animals' digits were felt cold 1 to 5 hours after injection, a sensation that lasted for 3 to 5 hours. The same conclusions were drawn from this experiment, namely that the increase in norepinephrine and cortisol in the blood could be the cause of peripheral vasoconstriction, explaining the cold sensation felt at the extremities.

If endotoxins played a role in the pathogenesis of laminitis, it would likely be an indirect effect on the pododerm. Indeed, hemodynamic disorders can be induced by endotoxin release; however, these also have the capacity to release other vasoactive agents, notably prostaglandins and vasoactive amines, which could then participate in the appearance of the observed vascular disorders (Flint, 1997). Thus, during endotoxination, cellular mediators such as prostaglandins or cytokines are released to protect the animal's body and eliminate these endotoxins.

Apart from experimental studies, endotoxins are released, for the most part, during bacterial lysis of Gram-negative germs (including Escherichia coli) when digestive metabolism is disturbed, especially when the digestive content is too acidic. The second, less significant source of endotoxin release is an episode of mastitis or metritis (Chen and Wolin, 1979).

Under physiological conditions, the mucosa of the digestive tract prevents toxins released into the ruminal fluid from being absorbed into the bloodstream; however, when it is weakened or injured, these penetrate the bloodstream and trigger the prostaglandin cascade. An imbalance between thromboxane and prostacyclin then occurs, leading to the production of thrombi, which obstruct blood capillaries in the pododerm. Blood circulation is then interrupted, and the resulting outcome can be compared to a heart attack occurring in the foot.

Liver integrity is very important in preventing the dissemination of endotoxins into the vascular system, as it functions to purify the blood arriving from the digestive tract via the hepatic portal system. It thus acts as a filter but also as a producer of proteins involved in the acute phase of inflammation (fibrinogen, haptoglobin, ...), when stimulated by cytokines (An et al, 2005). However, liver performance may be diminished in the period around calving, following accumulation of triglycerides in hepatocytes (hepatic lipidosis), resulting in decreased capacity of the liver to eliminate circulating endotoxins. Many conditions can then occur, including laminitis, which has a higher incidence at this precise time, especially in heifers.

Diagnosis:

- **Acute form:**
  - **Epidemiological:** Ask questions about access to grain reserves, a sudden change in diet (e.g., to ground corn).
  - **Animal examination:** Look for depression, anorexia, oliguria, tachycardia, rumen atony. Measure pH (4 to 5), lactacidemia, and possibly alkaline reserve.
  - **Differential:** Should differentiate from:
    - Overload indigestion: rare; occurs after ingestion of very indigestible feed or in very tired animals.
    - Parturient paresis: appears 24 hours to 3 days after parturition; animal responds to IV calcium salt infusion.
    - Acetosis: appears in the month following parturition and manifests with appetite disorders, pica, and constipation.

- Cerebrocortical necrosis: appearance of nervous signs in hypo in cattle (in hyper in sheep) with hyperlactacidemia and increased pyruvic acid levels. It mainly affects young animals after a diet change. Animal responds to IV vit B1 treatment.
- Grass tetany: appears in high-producing dairy cows shortly after calving and results in convulsions and death within a few hours. There is a tendency towards rumen alkalosis. It manifests as hypomagnesemia and responds to IV magnesium salt infusion.
- Listeriosis: characterized by behavioral and sensitivity disorders (hyperesthesia), falls, tonic-clonic seizures, hyperthermia (> 40°C), hyperleukocytosis, neutrophilia without monocytosis.

5.6 Correction of Metabolic Acidosis

In severe cases, with a clinical picture consisting of recumbency, hypothermia, severe depression, significant ruminal distension, tachycardia (110 – 130 beats per minute), and a ruminal pH of 5 or lower, a rumenotomy is indicated. The rumen is emptied, cleaned using a siphon, and rumen fluid (10 to 20 liters) is provided, along with a few handfuls of hay. Rumenotomy corrects ruminal acidosis, and the use of an alkalinizing agent is then unnecessary (Radostits et al, 2000). Most of the lactic acid and substrates allowing its formation are removed. Oral or intraruminal administration of molecules such as magnesium oxide or magnesium hydroxide after evacuation of all rumen contents can lead to metabolic alkalosis (Belbis, 2007).

In less severe cases, where affected animals are still standing but still show symptoms of depression, tachycardia (90 – 100 bpm), moderate ruminal distension, and for which pH is between 5 and 6, an alternative to rumenotomy is ruminal lavage, if the necessary equipment is available. A rubber tube 25 to 28 mm in diameter is introduced into the rumen, and hot water is pumped inside until distension of the left paralumbar fossa is observed, at which point the rumen can then drain by gravity. The rumen can be almost completely emptied after 10 – 15 necessary irrigations (Radostits et al, 2000). If gastric lavage is successful, alkalinizing agents are not necessary, but correction of systemic acidosis must be performed. Nevertheless, depending on circumstances (intoxication of a large number of animals, equipment unavailable, ...), rumenotomy or ruminal lavage may not be feasible. Oral administration of alkalinizing agents, such as magnesium carbonate or magnesium hydroxide, is then indicated [82]. They should be mixed with 8 to 12 liters of hot water and introduced via a tube directly into the rumen. Initial doses of 1 g/kg body weight may be followed by lower doses repeated at 6 and 12-hour intervals (Belbis, 2007).

A. Correction of Hydro-electrolyte Balance and Hypovolemic Shock

Systemic acidosis is treated by intravenous administration of 5% sodium bicarbonate at a quantity of 5 L for a 450 kg animal, over about 30 minutes. This will correct systemic acidosis. This infusion is followed by that of an isotonic sodium bicarbonate solution (1.3%), at a dose of 150 ml/kg body weight over the next 6-12 hours. Furthermore, in an adult bovine in hypovolemic shock, 1 liter per 25 kg body weight per hour can be administered, i.e., 20 liters in one hour for a 500 kg bovine. Due to the severity of dehydration, the administration rate of solutions can be 10 to 12 liters per hour.

B. Restoration of Ruminal Flora

Acidosis is accompanied by a significant modification of the ruminal microflora, notably a loss of the cellulolytic population. Restoration of the ruminal flora using rumen fluid from healthy animals is then indicated (Radostits et al, 2000). A slaughterhouse is the best source of rumen contents, but it is also possible to obtain it from live animals by collecting directly from the mouth during rumination the regurgitated bolus.

Rumen fluid can also be collected by siphoning the rumen using a tube inserted into the rumen via the mouth or a vacuum pumping system. Better results are obtained if 20 to 30 liters of water are pumped into the rumen, allowing the rumen fluid to drain by gravity as needed (Radostits et al, 2000). The rumen content thus collected is administered directly into the rumen after rumen emptying if a rumenotomy is performed. Repeated doses of rumen fluid can be given as needed. Commercial preparations containing dried ruminal fluid extracts are available and provide certain bacteria as well as the substrates necessary for their activity. This restoration of the ruminal flora, also called transfaunation, is a therapy applicable to other ruminal conditions, such as simple indigestion (Belbis, 2007).

- **Adjuvant Treatments**

Acidosis, especially subacute acidosis, often causes complications (laminitis, liver abscess, etc.), which should be prevented during treatment of ruminal acidosis. Adjuvant treatments include the use of antihistamines (to prevent laminitis), parasympathomimetics to stimulate ruminal motility, or thiamine or yeast to stimulate lactic acid metabolism (Lynch and Martin, 2002).

The effectiveness of these additive treatments is difficult to assess, and it seems unlikely that any of them are of major importance.

- **Treatment of Chronic Cases**

Treatment essentially consists of promoting restoration of a normal microbial population in the rumen by providing ad libitum good quality roughage. This provision of roughage will restore normal salivary secretion, which, with its alkaline pH and buffering capacity, represents the primary physiological factor of acid-base balance in the rumen. To accelerate the animal's recovery, administration of 2 to 4 liters of rumen fluid is particularly recommended. Lyophilized rumen fluid can also be used, a treatment of choice for chronic acidosis, at a dose of 3 g per day for 5 consecutive days (Belbis, 2007).

- **Prevention of Ruminal Acidosis**
- **Sanitary Prophylaxis**

If acute lactic acidosis, mainly linked to accidents, does not require major preventive measures (protection of concentrate stores, vigilance in concentrate distribution, ...), a number of sanitary measures are recommended to combat subacute acidosis. Providing rations sufficiently rich in fiber, allowing adequate chewing and salivation of feed, is recommended. Furthermore, the addition of concentrates to the ration must be done gradually to ensure a period of gradual adaptation to this diet.

(Radostits et al, 2000) recommend, in fattening cattle, starting with small amounts of grain (on the order of 8 to 10 g/kg body weight), and increasing this amount by 10 to 12% every two to four days.

Treatment

It is quite expensive but justifiable.

1-Theoretical: It would be necessary to try to stop fermentations, acidosis, dehydration, shock,...

- Ruminal acidosis: use bases, anti-infectives orally:
  - peni-strepto 5M, 5g (bovine) and IM, 1g (ovine).
  - terramycin: 3 to 5 g (bovine) and 500 mg (ovine).
- Metabolic acidosis: undertake infusion of bicarbonate solution (12.5 g/l), bovine: 1 to 2 liters; ovine: 500 ml.
- Dehydration: give non-glucose physiological saline.
- Promote lactic acid utilization: in tissues and in the liver. Administer vit B1 at a dose of 10 mg/kg.
- Combat shock: give dexamethasone: 20 mg for a bovine and 0.5 mg for a sheep.
- Cardiorespiratory analeptics: heptaminol: bovine = 2 g; sheep = 0.5 g.
- Prevention of laminitis and dermatitis: administer phenergan at a dose of 0.5 to 1 mg/kg IM.

2-Practical:

✓ Orally via tube, rehydrate rumen contents with 10 liters of cold water to stop fermentations. Add Na or Ca bicarbonates (500 g to 1 kg per bovine) and anti-infectives (e.g., sulfonamides).

✓ Parenterally, administer vit B1, corticosteroids, antibiotics, analeptics, hepatoprotectants (acetylmethionine), and possibly phenergan.

Prophylaxis:

✓ Acute form: Diet must be balanced. Practice transition when changing feed (from corn to another corn; or from barley to corn). Transition should take place over 3 weeks. Try to prevent accidents.

✓ Latent form: Balance the diet. Add buffering substances to silage (urea, Ca carbonate: 5 kg/tonne of wet matter to increase pH).

The most important scientific words translated into English (Chapter 4)

Terme Technique (Français)	Traduction Scientifique (English)
Acidose ruminale lactique	Ruminal lactic acidosis
Surcharge en glucides rapidement fermentescibles	Overload of rapidly fermentable carbohydrates
Streptococcus bovis	*Streptococcus bovis*
Lactobacilles	Lactobacilli

Terme Technique (Français)	Traduction Scientifique (English)
Hyperlactacidémie	Hyperlactacidemia
Déshydratation extracellulaire	Extracellular dehydration
Choc hypovolémique	Hypovolemic shock
Parakératose ruminale	Ruminal parakeratosis
Abcès du foie	Liver abscesses
Entérotoxémie	Enterotoxemia
Syndrome de la vache grasse	Fat cow syndrome
Cécité (Amaurose)	Amaurosis (blindness)
Laminite (Pododermatite aseptique diffuse)	Laminitis (Diffuse aseptic pododermatitis)
Histamine	Histamine
Endotoxines (LPS)	Endotoxins (Lipopolysaccharides)
Transfaunation	Transfaunation

CHAPTER V RUMEN ALKALOSIS INDIGESTION

1 Definition

It is acute ammonia intoxication due to hydrolysis of dietary urea by ureases of the ruminal flora. Clinically, it is characterized by a sudden onset, a rapidly fatal course, and vascular lesions (congestion, hemorrhage). Its importance is currently increasing, as attempts are made to incorporate non-protein nitrogen (NPN) into feed. It is an important disease especially in intensive farming (controlled feeding).

2 Epidemiology The disease appears in the context of intensive feeding.

- In dairy farming: NPN is distributed as a supplement in the milking parlor discontinuously and abruptly.
- In beef cattle farming: the disease has an enzootic aspect. It rarely appears because in this case, feed is distributed ad libitum, and thus consumption is more spread out.

After liver damage, there is a decrease in the conversion of ammonia to urea. Ammonia is the toxic form.

Extrinsic factors: The diet. Errors can occur in composition, distribution, or presentation.

These errors are:

- Energy imbalance (e.g., 150 g urea/day + straw; this ration is energy-deficient).
- Sudden change in energy source (e.g., switching from barley to corn) can result in temporary disturbances in energy utilization, leading to a peak of NH_3 genesis.
- Temporal dissociation between energy supply and NPN supply.
- Absolute excess of NPN from ingestion of beet leaves or crowns, cabbage, supplemented with urea; hence increased NH_3 doses.
- Increased total nitrogenous matter intake via the diet.
- Intensively cultivated spring pastures.

3 Review of Ammonia Utilization

In the rumen, there are bacteria that can synthesize their proteins from NH_4^+; others require amino acids; and others are indifferent: amino acids or NH_4^+. 50 to 80% of dietary proteins in the rumen yield hydrocarbon skeletons and NH_3. If there is an imbalance between NH_3 formation and utilization, there is an ammoniogenesis peak and intoxication. The role of carbohydrates is important in a diet containing NPN. It is twofold:

- Bacteria use them as an energy source for metabolic transformations.
- Furthermore, their fermentation releases large quantities of fatty acids which aim to maintain rumen pH at a normal value. This pH, which the deamination of nitrogenous products tends to raise to abnormal values. Ammonia (NH_3) is particularly toxic. The higher the rumen pH rises, the more non-ionized ammonia is formed. Initially, the buffering system can play its role by forming NH_4^+ ($NH_3 + H^+$), which is not absorbed by the rumen and is used for microbial protein synthesis.

When pH rises above normal values, H^+ ions decrease or disappear, leading to formation of ammonia, which will be absorbed by the rumen in an attempt to decrease the rise in rumen pH.

In other tissues

After its passage into the blood, ammonia will be combined with glutamic acid to mask its toxicity.

Glutamic acid + ATP + NH_3 → Glutamine + ADP + Pi (synthetase)

This glutamine serves as a blood transporter of ammonia; it leads this metabolite to the liver and kidney, where glutaminase releases ammonia.

L-glutamine + H_2O → L-glutamate + NH_3

Ammonia is then either integrated into urea or eliminated in urine.

In tissues, especially in the central nervous system, ammonia utilization occurs via three main pathways:

/Biosynthesis of glutamic acid:

α-ketoglutarate + NH_3 + NADPH → glutamate + $NADP^+$ + H_2O (glutamate dehydrogenase)

/ Biosynthesis of glutamine:

Glutamate + NH_3 + ATP → Glutamine + ADP + Pi (glutamine synthetase)

/ Biosynthesis of carbamoyl phosphate

4 ETIOPATHOGENESIS OF RUMEN ALKALOSIS

If 100 g of urea is distributed to an animal, it results in 50 g of ammonia in 100 liters of rumen fluid; which is equivalent to 500 mg of NH_3 per liter. Nitrogenous matter intake from feed is around 250 mg of NH_3 per liter. We would have a total of 750 mg of NH_3 per liter. Symptoms appear from 1 g/l. In the rumen, the introduced urea is quickly degraded into ammonia with release of carbon dioxide. This rapid hydrolysis results in a significant increase in pH in the forestomachs, promoting alkalosis (pH increases from 6 to 7.9). For absorption, the rumen wall is practically impermeable to NH_4^+ ions; only NH_3 diffuses and is responsible for toxicity when pH is high. Therefore, there is a shift from NH_4^+ to undissociated NH_3. Absorption thus increases, and ammonia passes into the blood and goes to the portal vein to reach the liver. The portal ammonemia threshold is 50 mg/l, and arterial concentration is 10 mg/l. Therefore, if concentration is higher, there is blood alkalosis and hyperammonemia.

Symptoms:

/ Digestive Atony: Several elements may be responsible for this motor inhibition.

- Ammonia or urea released into the blood can sensitize higher nerve centers, which in turn via nervous pathways could induce inhibition of motility.
- Ruminal alkalosis and toxic products (e.g., histamine) from the environment and blood alkalosis could be responsible for hypomotility.
- Finally, it could simply be a consequence of local action corresponding to chemosensitivity of the rumen wall to ammonia, modifying in turn the activity of the bulbar motor center.

/ Nervous Signs:

NH_3 is toxic to the central nervous system; convulsions develop rapidly when brain ammonia levels reach 9 mg%. This toxic action causes an increase in aerobic glycolysis, leading to increased production of α-ketoglutaric acid, an element needed to reduce NH_3 levels in the CNS. This results in uncoupling of the Krebs cycle, leading to disruption by NH_3 of the energy metabolism of nerve cells, which also manifests as humoral alterations such as: hyperammonemia, hyperglycemia, hyperpyruvicemia, and also an increase in glutamine and lactate.

- **/ Hydro-electrolyte Disorders**

Chelates of ammoniaco-magnesium phosphates are formed, masking magnesium from its normal sites in the enzyme system. This results in signs identical to grass tetany due to hypomagnesemia.

- **/ Symptoms:**
  - **General:** The animal presents poor appetite, intense thirst, rumination is irregular or absent, feces are diarrheic with an ammoniacal odor, milk production is decreased, and general condition is deteriorated (rough and dull hair, skin stuck to thoracic wall, asthenia, rapid and weak pulse).
  - **Nervous:** They are tetaniform; manifested by behavioral disorders (restlessness, ears flapping, bellows, kicks, and gallops at the slightest excitement) and motor disorders (lateral recumbency, paddling, and tonic-clonic contractions).
  - **Digestive:** In general, there is no overload; disorders evolve slowly. Palpation and percussion are non-painful. On auscultation, there is cessation of motor and biochemical phenomena. Rumen fluid is very fluid, greenish-black, with a putrid odor, and pH

5. The microbial population consists of coliforms, Proteus, or molds. Cellulose digestion time is greatly increased.

Lesions:

Generalized congestion of the carcass is noted, with congestion of the tracheal mucosa and lungs (with edema); hemorrhagic phenomena of serous membranes (endocardium); and degenerative phenomena in the liver (especially microscopic lesions).

-Biochemical: Biochemical results show:

- Increased activity of certain enzymes in the blood (Arginase, Ornithine Carbamyl Transferase (OCT), Gamma-Glutamyl transpeptidase, Glutamate Oxaloacetate Transaminase (GOT), Glutamate Pyruvate Transaminase (GPT)).
- Increased ammonemia, uremia (from 0.3 to 1-1.5 g/l), urinary urea (10 to 15 g/l instead of 1 g/l).
- Also increased concentration of glutamine and lactate in cerebrospinal fluid.

Creatinine should be assayed to rule out renal lesions.

Diagnosis:

- **Epidemiological:** Check the amount of urea in the ration, a lack of energy supply or lack of homogeneity, absence of transition, liver parasitism, ...

- **Clinical:** Look for digestive atony and nervous signs.
- **Lesional:** Look for congestion, hemorrhage, and degeneration.
- **Differential:** Should differentiate from:
  - Grass tetany: which has the same treatment.
  - Vitular neuropathy: rarely presents as hyper.
  - Ketosis.
  - Certain enterotoxemias: which also appear with increased nitrogen; their course is less rapid.
  - Certain encephalomyelitis: e.g., rabies, but its course is longer.
  - Poisoning by lead, phosphorus, organophosphates, or organochlorines: in these cases, measure rumen pH, blood and urinary urea.

Prognosis: severe.

Treatment:

Must be early and sustained, before the first tetanic contractions. If the urea dose is less than or equal to 50 g/100 kg BW, treatment may sometimes succeed.

- **Stop NH_3 production:** Give a bovine 25 to 30 liters of cold water to dilute and lower the temperature of the rumen contents. Antibiotics (peni-strepto, tetracycline) can also be administered via tube and trocar.
- **Limit NH_3 absorption:** Acidify; give acetic acid 120 g for 60 g of urea; or 5 to 10 liters of vinegar (50 g/l acetic acid in vinegar). Repeat within the hour at half dose.
- **Promote NH_3 elimination:** Give glutamic acid and magnesium solutions (e.g., Mg glutamate will yield glutamine and chelates of ammoniaco-magnesium phosphates).
- **Convalescence treatment:** Distribute lipotropic factors (for the liver) and vit A to convalescent animals.

Prophylaxis: To prevent the disease:

- Use compounds that release urea more slowly (biuret, triuret, cyanuric acid).
- Treat for fascioliasis. Do not give urea during treatment and to animals with high uremia.
- Urea distribution must be moderate, gradual, and supplemented with energy.
  - Transition should take place over 3 weeks for the gradual modification of bacterial profile.
  - Add energy to rations supplemented with urea. Barley, wheat, oats are more effective than corn, unless the latter is gelatinized.
  - Example of a supplemented ration: 5 kg of urea/tonne of silage, equivalent to 15 g of urea/kg DM, which doubles the nitrogen supply of the corn silage itself. (Corn silage: 0.8 UF/kg DM and 40 g MAD/kg DM [30% DM]).

How to improve urea utilization

- Use non-protein nitrogen.
- Neutralize urease in the rumen (Payne, 1983).

The most important scientific words translated into English (Chapter 5)

Terme Technique (Français)	Traduction Scientifique (English)
Alcalose ruménique	Rumen alkalosis
Intoxication ammoniacale aiguë	Acute ammonia intoxication
Azote non protéique (ANP)	Non-protein nitrogen (NPN)
Uréase	Urease
Hyperammoniémie	Hyperammonemia
Tétanie (signes tétaniformes)	Tetany (tetaniform signs)
Hyperuricémie	Hyperuricemia

CHAPTER VI LEFT DISPLACEMENT OF THE ABOMASUM (LDA)

Left displacement of the abomasum is currently one of the main reasons for surgery in modern dairy herds; its incidence varies from 0.35% to 4.4% across bovine populations, sometimes reaching up to 15% in some herds, even 20% (Intensive farming).

- Left displacement of the abomasum is the most common positional abnormality of the abomasum, accounting for 85% to 95.8% of all displacements.
- The body of the abomasum progresses towards the left costal wall, then posteriorly and dorsally towards the left flank.
- It progresses between the rumen and the abdominal wall, in the left flank in the subcostal region, and can extend under the flank hollow when significantly dilated.

1 ETIOLOGY

1.1 Predisposing Factors for Left Displacement of the Abomasum

- **Breed:** Breed predispositions to LDA have been proposed (e.g., Holstein), but some authors disagree on this subject.
- **Age:** LDA can be diagnosed in animals of any age; however, the risk of developing LDA increases with the age of the animal, with the number of gestations. In dairy cows, it is highest between 4 and 7 years. Although a significant frequency of LDA is reported following the first calving, most authors agree that it increases between the fourth and sixth calving.
- **Calving date:** All authors agree that LDA is most often diagnosed in the period immediately following calving. 57% of cases

are encountered in the first two weeks following calving, 80.2% in the month following, and between 85 and 91% in the first six weeks postpartum.

- **Intercurrent Diseases:** At the time LDA is diagnosed, the animal very frequently presents with one or more other diseases. Some authors find this in 74% of LDA cases, such as metritis, mastitis, ketosis, hypocalcemia. According to numerous studies, ketosis and metritis are then the most frequently found diseases, which is often explained by the proximity to calving in many LDA cases.

NB: a cow with ketosis is over 39 times more likely to develop LDA than a cow without ketosis.

- **Season:** Metritis and twinning are risk factors for LDA, while mastitis does not appear to be a factor. As abomasal motility depends on calcemia, many authors state that subclinical hypocalcemia is a risk factor for abomasal displacement. The majority of LDA cases occur from October to April, when dairy cows are not on pasture.
- **Nutrition:**

  Around calving, significant dietary changes occur both in composition and volume of intake, as

the dairy cow switches from a diet consisting almost exclusively of forage to a diet very rich in concentrates.

- Important precautions must then be taken both before calving and after, regarding feeding management (dietary transition periods) and the quality of the feed distributed.
- Abrupt dietary changes before calving in preparation for the upcoming lactation predispose to abomasal displacements. It is recommended to feed dry cows a diet that meets their needs and is appropriate for their body condition.
- Cows should receive a low-energy ration during the first half of the dry period; then concentrates can be added in the three to four weeks before calving.

1.2 Triggering Factors for Left Displacement of the Abomasum

a) Mechanical, Anatomical Theory

- The gravid uterus lifts the rumen and pushes the abomasum cranially and to the left;
- following calving, the rumen moves back down to the lower abdomen and thus traps the abomasum, preventing it from returning to its normal anatomical position.
- Gas and ingesta then accumulate in the abomasum, which dilates.
- However, this theory does not explain the occurrence of abomasal displacements in calves and male cattle.

b) Metabolic Theory

- Abomasal atony is then a prerequisite for abomasal displacement; all authors now share this view.
- Abomasal atony or hypotonia leads to dilation of the organ due to gas accumulation, dilation that precedes its displacement.
- Experimental studies have confirmed the validity of this theory; abomasal dilation is essential for its displacement, but this dilation is only possible when the motility of the organ is impaired.
- Causes of abomasal atony include:
  - hypocalcemia,
  - histamine release,
  - endotoxemia,
  - alkalosis,
  - hyperinsulinemia,
  - duodenal acidification,
  - prostaglandins.

1.3 CONSEQUENCES OF ABOMASAL DISPLACEMENTS

At the level of the abomasum, the parietal cells of the abomasum secrete hydrochloric acid and simultaneously release bicarbonate ions into the blood. However, due to the significant slowing of transit during LDA, chloride ions are trapped in the abomasum, and blood bicarbonate concentration therefore increases. Metabolic alkalosis thus develops following this hypochloremia.

2 CLINICAL DIAGNOSIS

A) History

The animal is generally presented in the peripartum period for milk production significantly lower than expected or for a drop in milk production associated with decreased appetite, variable appetite, or intermittent anorexia.

- Some authors report selective appetite: the amount of concentrates ingested is considerably reduced, while the bovine continues to eat forage. Generally, the farmer also reports a concomitant disease (metritis, retained placenta, mastitis, ...).

Furthermore, weight loss is often observed. Defecations are rare. Laval also describes rather dry feces. They may be darker than usual.

B) Clinical Examination

- **General Examination**

Body temperature, heart rate, and respiratory rate are within normal ranges. Observation from a distance may reveal weight loss.

- **Specific Examination of the Abdominal Cavity**

/ Inspection

On inspection, depending on the degree of abomasal dilation, the abdominal wall may appear tense along the left hypochondrium, sometimes immediately behind the last rib in the flank hollow.

/ Palpation

During palpation of the left sublumbar fossa, tension may be detected in its cranial part.

/ Percussion

When performed alone, percussion provides little information if the veterinarian is not accustomed to it. Percussion alone may reveal a resonant zone limited caudally by the rumen and extending cranially to the thoracic cavity.

/ Auscultation

During auscultation of the left flank, ruminal contraction sounds are rare and heard mainly in the caudal part because during its displacement, the abomasum slips between the left abdominal wall and the rumen.

- Hydro-aerial sounds, corresponding to the movement of liquids in the gaseous atmosphere of the abomasum, may be heard during prolonged auscultation (greater than five minutes) in the left thoraco-abdominal zone.
- Flicking is performed on the abdominal wall or on the rib cage, around the stethoscope head placed on the wall or in the intercostal spaces.
- Clear metallic sounds "ping" are then heard in the projection zone of the displaced abomasum. This characteristic sound is also audible during auscultation.

3 TREATMENT

Medical Treatment

- Medical treatment primarily aims to restore proper abomasal motility so that it expels the gas it contains. It includes correction of:
  - electrolyte and metabolic disorders (treatment of hypocalcemia, potassium disorders, dehydration, acid-base disorders, acetonemia),
  - administration of parasympathomimetic agents and molecules used to treat concomitant diseases.
- However, with medical treatment alone, the therapeutic success rate is very low, estimated at less than 5%, corresponding to cases of spontaneous recovery. The use of medical treatment alone is therefore inconceivable; however, it is useful as a supplement to a surgical correction method to accelerate the animal's recovery.

"Rolling" Technique

- After a 36-48 hour fast, the cow is placed on its right side, then placed in dorsal recumbency using ropes.
- Its abdomen is then vigorously massaged to facilitate repositioning of the abomasum to its physiological position on the midline while the cow is rocked in a 90° arc for a few minutes.
- It is then rolled into left lateral recumbency, before it returns to sternal recumbency.

To facilitate its repositioning, the abomasum may undergo paracentesis (surgical fixation of the abomasum) while the animal is in dorsal recumbency. When transitioning to left lateral recumbency, the rumen slides along the left abdominal wall and blocks the abomasum in the right part of the abdominal cavity.

- Advantages of this method: simplicity and speed, low cost, no specific equipment needed, and it is not very traumatic for the cow.
- However, this technique also has many disadvantages, the main one being its low long-term cure rate. Therefore, this technique is now hardly used.
- 75% recurrence is described.

4 EXPLORATORY LAPAROTOMY

- To identify the cause of abdominal distension, an exploratory laparotomy via the left flank should be performed.
- It aims to specifically search for possible adhesions in the reticulum region that may be related to Hoflund syndrome and to determine the nature of the rumen content.
- Upon opening the abdominal cavity, the extremely dilated abomasum is visible in the left flank hollow. A diagnosis of left abomasal displacement is thus established. No adhesions on the reticulum, nor any abscesses that could cause Hoflund syndrome, are detected.
- Once the abdominal cavity is opened on the left flank, it is decided to proceed with surgical abomasopexy via this side.

- Postoperative antibiotic therapy is implemented, based on amoxicillin at a dose of 15 mg/kg intramuscularly, in a single injection,
- combined with anti-inflammatory treatment (carprofen, Rimadyl®, at a dose of 1.4 mg/kg subcutaneously).
- After opening the left flank, the abomasum is fixed to its anatomical position using a pexy in the ventral paramedian region (slightly to the right if possible).
- The goal is to induce adhesion formation between the abomasal wall and the peritoneum in the ventral region so that the abomasum no longer moves during dilation.
- Two skin sites are surgically prepared (clipping, scrubbing, disinfection) and locally anesthetized in the right paramedian area; two landmarks are determined for the exit of the pexy sutures.
  - **the first, 5 to 10 cm (depending on the animal's size, here small) behind the xiphoid appendix and 5 cm to the right of the paramedian line,
  - ** the second, 6 to 8 cm behind the first point;
- In the left flank hollow, more or less close to the ribs and more or less low depending on the animal's size and the length of the surgeon's arms.
- After opening the flank, the greater curvature of the (displaced) abomasum is visible through the laparotomy wound after incising the peritoneum.
- The abomasum is fixed 3 cm from the attachment of the greater omentum, in the middle of the greater curvature.

5 OMENTOPEXY VIA THE RIGHT FLANK

- The procedure is performed on the standing animal. After surgical preparation of the right flank, the abdominal wall is incised. The abdominal cavity is explored; the diagnosis of left abomasal displacement is confirmed by palpating the abomasum along the left abdominal wall, passing the arm over the rumen.
- The abomasum is then punctured using a needle connected to tubing exiting outside the abdominal cavity.
- When gas has escaped from the abomasum, it is repositioned on the right side of the animal by grasping the pyloric part with a full hand.
- Success rates vary from 86 to 93.8%.

6 ABOMASOPEXY VIA THE RIGHT FLANK

- Abomasopexy via the right flank is a technique very similar to omentopexy via the right flank; these techniques simply differ in the method of abomasal fixation. Whereas in right flank omentopexy, the fixation concerns the greater omentum which, through its attachments to the abomasum, prevents it from re-displacing.
- From a technical standpoint, abomasopexy via the right flank is more difficult to perform, as the abomasum, located under the rumen, is harder to reach than the greater omentum.

- After surgical preparation of the left flank, a laparotomy is performed on the standing animal (the incision is made 2 to 4 centimeters behind the last rib).
- The surgeon then immediately finds the dilated abomasum to the left of the laparotomy wound.
- The abomasum is then punctured with a needle connected to tubing (which exits outside the abdominal cavity) to deflate it.
- A continuous suture is then placed in the abomasal wall along the greater curvature, three centimeters from the greater omentum.

7. RIGHT DISPLACEMENT OF THE ABOMASUM (RDA) AND RIGHT ABOMASAL VOLVULUS (RAV)

7.1 Right Displacement of the Abomasum (RDA)

RDA generally affects older cows, between 3 and 6 weeks after calving.

- 12.5% / LDA.
- Same factors.
- Atony then gaseous distension.
- It dilates and undergoes slight rotation, clockwise when viewing the cow from the right.

7.2 Right Abomasal Volvulus (RAV)

Begins as a right abomasal displacement, by dilation. Then a torsion of the abomasum along an omaso-abomasal axis. Viewed from behind, it is a rotation of the abomasum; the torsion knot is located at the omaso-abomasal junction; the duodenum is then positioned forward of its normal position but remains behind the omasum in a counterclockwise direction. Development of an occlusive syndrome following duodenal occlusion. Cardiovascular consequences such as hypovolemic shock due to fluid sequestration in the abomasum, metabolic consequences such as alkalosis, hypochloremia followed by metabolic acidosis related to hypovolemia.

7.3 Clinical Presentation of RDA and RAV

Same symptoms as for LDA:

- Drop in milk production,
- Decreased appetite,
- Presence, on percussion-auscultation, of a "PING" on the right side in the corresponding auscultation area (under the last ribs, cranial to the flank hollow).

In case of abomasal torsion, the animal additionally shows signs of shock:

- Enophthalmos,
- Tachycardia,
- Complete cessation of transit and/or short-term diarrhea.
- Right abdominal distension and presence of melena (digested black blood in feces).

Without treatment, the animal may die in 1 to 3 days from dehydration and cardiovascular failure.

7.4 Differential Diagnosis

The presence of a PING on the right can also suggest:

- Peritonitis,
- Abscess,
- Cecal dilation, with or without torsion.

7.5 Treatment of RDA

Medical treatment is possible, with antispasmodics and fluid therapy. Surgery can also be used, performing an omentopexy, i.e., fixation of the omentum to the abdominal wall, via the right flank.

7.6 Treatment of Abomasal Volvulus

It is crucial to successfully detorse the torsion. Treatment necessarily involves surgery: most often, it involves right flank omentopexy.

7.7 Prophylaxis

Avoid energy deficit at the start of lactation by preserving the cow's intake capacity, which must have free access to hay, by preparing dry cows for calving through a dietary transition from a "dry" ration to a "lactation" ration, and by providing propylene glycol, a glucose precursor, to cows at the beginning of lactation. (Activates ruminal fermentation and the gastrointestinal tract).

Example: ACÉTAL®

- **Dosage**
  - **Dairy Cows**
    - For prevention: 50 ml of the solution twice daily for 4 days during the risk period (2 to 4 weeks after calving).
    - For treatment: 1. 250 ml of the solution twice daily for 4 days, then 125 ml morning and evening for the next 3 days.
- Postpartum infections such as mastitis and metritis must be prevented and treated quickly, with intrauterine treatment, and hypocalcemia must be prevented through management of the dry cow ration.
- Excessive body condition at drying off should be limited, i.e., avoid overly energetic rations for dry cows.

8. ABOMASAL ULCERS

Defined by a loss of wall substance.

- The mechanism is unknown.
- Clinical signs range from inapparent to severe.

Causes and Risk Factors: Causes are poorly understood. Factors mentioned include:

- **Dietary** (high energy content of the ration, copper deficiency, iron deficiency in calves),
- **Environmental** (cold and humidity, large temperature fluctuations),
- **Management** (stress of any kind),

- **Infectious** (clostridia, diplococci, fungi).

8.1 Symptoms

- Mainly in beef calves, dairy calves.
- There are 3 types of ulcers:

Type 1:

- Non-perforating, non-bleeding ulcers, causing in acute forms abdominal pain (colic, sunken eyes, arched back posture, slightly tense abdomen) and in chronic forms non-specific symptoms (irregular appetite, dull hair);
- Perforating ulcers progressing to death in a few hours or to chronic peritonitis, the latter occurring when turned out to pasture.

Type 2:

- Non-perforating but heavily bleeding ulcers, causing anemia (pale mucous membranes) and melena (digested dark-colored blood in feces).

Type 3:

- Perforating ulcers progressing to death in a few hours or to chronic peritonitis.

Clinical Presentation of Ulcers:

- Anorexia, partial to total,
- Fever, variable,
- Melena accompanied by anemia,
- Tachycardia, signs of shock.
- For perforating ulcers, there are signs of shock, abdominal pain, hyperthermia then hypothermia.
- In calves, may be confused with peritonitis or omphalophlebitis.
- In cows, may be confused with T.R.P. (traumatic reticulo-peritonitis), liver abscesses, peritonitis, Hoflund syndrome, or abomasal displacement with volvulus.

8.3 Treatment of Ulcers

- Treatment is based on oral administration of:
  - Antacids (calcium carbonate, sodium bicarbonate) and
  - Gastric protectants.
- Intravenous rehydration.
- Antibiotic therapy is indicated for infectious hypotheses (penicillin-streptomycin or sulfonamides).
- To combat the consequences of bleeding, vitamin K can be administered.

The most important scientific words translated into English (Chapter 6)

Terme Technique (Français)	Traduction Scientifique (English)
Déplacement à gauche de la caillette (DGC)	Left displaced abomasum (LDA)
Déplacement à droite de la caillette (DDC)	Right displaced abomasum (RDA)
Volvulus de la caillette (Torsion)	Abomasal volvulus (Right abomasal volvulus, RAV)
Atonie de la caillette	Abomasal atony
Alcalose métabolique hypochlorémique	Hypochloremic metabolic alkalosis
Ping (son métallique à la percussion-auscultation)	"Ping" (metallic sound on percussion-auscultation)
Abomasopexie	Abomasopexy
Omentopexie	Omentopexy
Hypocalcémie subclinique	Subclinical hypocalcemia
Ulcères de la caillette	Abomasal ulcers
Méléna	Melena

CHAPTER VII ENTEROPATHIES

A. DIARRHEAS

Definition:

Diarrhea is "the too-rapid evacuation of overly liquid stools." It results in the passage of soft feces, with the dry matter content of feces becoming less than 12% in calves. In cattle, it involves, following the intervention of numerous etiological factors, a sudden alteration of the physiological mechanisms controlling water and electrolyte secretion. Diarrheas therefore originate from an increase in osmolarity in the intestinal lumen, a disorder of water and electrolyte absorption, and motor dysfunction.

Mechanism:

/ Increase in intestinal osmotic activity: Malabsorption of sugars, proteins, and lipids leads to a prolonged stay in the intestinal lumen of products with high osmotic power. But it is essentially carbohydrate malabsorption, due to defective hydrolysis or mucosal absorption, that promotes water movement toward the intestinal lumen by osmotic effect. However, we must recall the physiological predisposition of cattle, in which we observe low digestive capacity and insufficient reabsorption. Similarly, we can mention that with increased pH, particularly in carbohydrate hydrolysis defects, there is decreased absorption by the colon.

/ Water and electrolyte absorption disorders: These disorders are observed especially during infectious and parasitic diarrheas, which act in two ways:

- Very high secretion of water and electrolytes by the jejunal mucosa induced by the action of microbial enterotoxin is possible; this, by increasing water secretion in the small intestine, creates an imbalance between the flow toward the lumen and the flow toward the mucosa that cannot be compensated by ileal or colonic reabsorption.
- Bacteria or parasites can induce mucosal lesions, particularly at the level of the colon, leading to a local vascular reaction with capillary dilatation and mucosal ulcerations that facilitate plasma exudation and decrease water and electrolyte absorption capacities.

/ Motor dysfunction: Motor dysfunction is not important in cattle. Acceleration of transit or its slowing can cause diarrhea because changes in transit time modify contact time. Acceleration can be caused by prostaglandins, serotonin, as well as by fatty acids and bile acids (not fully shown in text).

B. FUNCTIONAL ENTEROPATHIES

1. SPASMS

They are observed in cases of water colic. The onset is sudden, and the condition progresses rapidly toward recovery. These spasms are caused by the ingestion of large amounts of cold water and by sudden changes in external temperature.

2. DILATATION

It follows intestinal paresis in older animals. It is a paralysis of nervous origin, which may be peripheral (submucosal plexus) or central. Obstructions result in upstream hypertrophy followed by dilation. Clinical signs include slowed intestinal transit, constipation, and varying degrees of colic. Diagnosis is difficult. Treatment consists of administering linseed, sodium or magnesium sulfate, Genabiline, and, after softening, alkaloids.

C. MECHANICAL ENTEROPATHIES

1. OBSTRUCTIONS

They are rare in cattle, as the natural receptacle for foreign bodies is the reticulum. They occur mainly in young animals weaned for 5 to 6 months. The obstructive agents are phytobezoars or trichobezoars (composed of mucus, plant debris, and hair). The obstruction may be complete, resulting in intestinal occlusion, or partial, presenting as constipation.

2. OCCLUSIONS

Definition

L'occlusion est l'arrêt du cheminement normal des gaz et des matières de l'intestin qui se traduit par des troubles graves se manifestant par un syndrome humoral, de la déshydratation et d'acidose. L'évolution aiguë ou subaiguë se fait vers la mort.

Etiology:

- **Functional causes:** Intestinal occlusion (ileus) can be paralytic (cessation of peristaltic movements) in lesions of the central or peripheral nervous system, in thromboembolic vascular lesions, and in peritonitis, or spasmodic in certain helminthoses and lead poisoning.
- **Mechanical causes:** They are due either to strictures or to topographic abnormalities.

3. STRICTURES:

These are encountered in scars (from parasites or surgical intervention), in hematomas, or in steatonecrosis trapping an intestinal loop.

4. INCARCERATION:

This is strangulation due to a vicious anatomical arrangement. A loop of the small intestine is trapped in a fibrous band, either by adhesion (in chronic peritonitis, after enucleation of a corpus luteum, metroperitonitis, traumatic reticuloperitonitis from a foreign body), or by a hernial ring (in umbilical, inguinal, omental hernias, appearing especially at the jejunum after extraction of a large calf).

5. VOLVULUS:

This is rotation of the intestine on itself or around a pedicle.

6. KINKING:

It can be encountered on a remnant of the umbilical artery.

7. INTUSSUSCEPTION:

This is the most frequent cause; it appears on the ileum, either by spasm of the anterior segment and dilatation of the posterior segment, or most frequently in esophagostomosis and in cecal dilatation and torsion.

Pathogenesis:

The reduction in intestinal diameter and venous obstruction (pedicle) lead to edema and intraluminal and intraperitoneal exudation. Then, permeability disorders occur (especially to bacteria) with spasms, anoxia, and acidosis that will lead to necrosis. Pain confirms the spasm, colic, then occlusive shock. The humoral syndrome results in absorption disorders and increased losses in the injured part and in the abdominal cavity of water, electrolytes, and proteins, leading to decreased blood pressure and the appearance of tachycardia, anuria, and hyperuricemia.

Symptoms:

- **Initial phase:** The syndrome is more rapid if the lesion site is more anterior. Fleeting colic appears and ceases as soon as necrosis occurs. The animal shows a pain attitude.
- **Established phase:** Colic ceases; there is cessation of transit, which is early if the lesion is posterior. After an average of 48 hours, defecation is suspended or replaced by evacuation of a mixture of mucus and blood. Temperature is normal or slightly elevated; heart rate increases from 90 to 120 beats/min. The animal has a dry muzzle with crusts, the head is carried low, the eyes are sunken, and breathing is irregular with expiratory groans.

Outcome:

The disease either progresses toward healing after elimination of the necrotic cast, or toward removal of the obstacle (transport of the animal), or toward death.

Diagnosis:

Biochemical: Biochemical tests reveal hemoconcentration with increased hematocrit, serum proteins, and urea levels; and decreased chloridemia and kalemia (from 5 to 3 meq/l).

Physical signs:

- On inspection, distension of the right flank and sometimes both is noted.
- On palpation of the right flank, a relaxation of the wall is felt.
- On auscultation, there is digestive atony (silence). Rumen contractions are rare or absent.
- On rectal exploration with the animal first lying down, we note emptiness of the rectum; the glove shows mucus on its surface (positive "arm sign"), and we feel a snail shell (volvulus), the mesenteric cord, loops dilated with gas (upstream of the occlusion), and a balloon in the case of cecal torsion.

Treatment:

It is surgical. For medical preparation, one should:

- Infuse intravenously 1/3 isotonic glucose solution and 2/3 isotonic saline solution. The total volume is 5 to 20 liters.
- Administer potassium orally to combat hypokalemia responsible for muscle weakness.

- Prevent septic complications by antibiotic prophylaxis via the general or intraperitoneal route.
- Combat shock and support major functions using dexamethasone (3 or 4 times the anti-inflammatory dose), cardiorespiratory analeptics, and lipotropic factors for detoxification (which oppose fatty overload of the liver).

D. ORGANIC ENTEROPATHIES

Different causes can be at the origin of these organic enteropathies:

Parasitic: They are due to nematodes and protozoa (coccidiosis).

Toxic: The toxic agents that induce intestinal symptoms are:

- Nitrates and nitrites (green cereals)
- Molybdenum, which causes copper deficiency (diarrhea, hair discoloration, etc.), NaCl, beet collets and pulps (K+OH-), acorns (tannins), annual mercury (hemoglobinuria), apples (acidosis), organochlorines and organophosphates.

3.Infectious:

3.1 Non-specific

These conditions appear in newborns (colibacillosis, rotavirus, coronavirus, etc.) and in adults where the infection can be primary or secondary.

- **Primary:** Due to feeding errors (sudden diet change) resulting in indigestion, an imbalance of the flora favoring gram (+) or gram (-), leading to enterotoxemia.
- **Secondary:** To extensive hepatitis, resulting in portal hypertension. The latter leads to internal congestion, which manifests as chronic diarrhea and renal lesions that will give a uremic syndrome.

3.2 Specific

In mucosal disease (virus), paratuberculosis, salmonellosis, and winter dysentery.

DIARRHEIC COLIBACILLOSIS OF ADULT CATTLE

Colibacillosis in adults can cover two syndromes: one diarrheic that resolves spontaneously in a few days; the other is a true enterotoxic syndrome.

Etiology

Coliform bacteria in general are subdivided into "invasive" *E. coli* that possess one or more properties (virulence gene, resistance to the bactericidal power of serum, siderophore, surface antigens) that allow them to resist the body's defense mechanisms and multiply there (colibacillary septicemia); and enterotoxigenic *E. coli* that carry fine protein filaments (K99) allowing them to attach to enterocytes and produce a toxin responsible for water and electrolyte losses (colibacillary enterotoxicosis).

Symptoms: Three phases are distinguished:

- **A hyperthermia phase:** lasting 48 hours, temperature 41°C or higher, with depression, inappetence, and rumination cessation.

- **A diarrhea phase:** after 48 hours, the temperature drops to around 39.3-39.5°C, while profuse, somewhat grayish, foul-smelling diarrhea appears. Fecal emissions are frequent and often accompanied by mild straining, rarely colic. In 50% of cases, intestinal inflammation is such that the feces are clearly hemorrhagic. Indeed, reddish, undigested blood streaks are observed on the feces.
- **A precomatose or comatose phase:** after 1 to 2 days during which this diarrhea persists, the animal becomes exhausted, breathing accelerates, and it falls into a precomatose state with hypothermia (temperature around 37°C). Fatal outcome is then the rule. Death occurs in a coma as in colibacillary septicemia of the calf.

Pathogenesis:

The symptoms of the disease are attributed to a direct action of coliform bacteria on the intestine, causing exogenous and endogenous water loss, and to an action of colibacillary toxins causing acute hypotension, transudation into serous cavities, and a state of gastroplegia accompanying diarrhea.

Diagnosis:

Diagnosis of colibacillosis in adults is easier when there is concomitant mastitis or even arthritis. The rapidity of progression should also suggest colibacillosis. Failing all these, demonstration of coliform bacteria is easily done by culture from feces. No precautions are needed to perform these samples, and the results are 100% reliable.

Treatment:

It should be directed against the cause and against the consequences.

/ Antibiotics such as streptomycin, colistin, neomycin, ampicillin, and chloramphenicol are very effective against *E. coli*, but the sensitivity of each strain is very variable.

/ Good rehydration is important and often ensures success:

- Either intravenously with multiple prepared solutions or simply with isotonic NaCl and glucose solutions or with a 14% bicarbonate solution.
- Also orally using commercial preparations balanced in glycine, glycocol, and other ionic elements (potassium, sodium, magnesium).

E. ENTEROTOXEMIAS

Enterotoxemias are diseases characterized by a toxinfection with enzootic or even sporadic occurrence common to cattle, goats, and particularly sheep. They are due to the pathogenic action of strict anaerobic bacteria of the genus *Welchia* and clinically manifest as a gastro-entero-hepatic and renal syndrome.

Synonymy: They are also called "Strike," "Struk," "Pulpy kidney disease," "Coup de sang," "Fiel"; and in Algeria, they are called "Traf," "Djen," or "Bouferas."

Etiopathogenesis:

These conditions appear following the dissemination into the body via the bloodstream of bacterial toxins produced in the intestine by clostridia. Clostridia are found in the digestive tract of healthy animals; it is predisposing factors that allow significant multiplication of these bacteria.

1. Predisposing factors:

- **Species:** Ruminants, particularly sheep, are the most exposed. This predilection is due to the difficulty of eliminating toxins in sheep compared to other species (great length of the intestine, about 30 m, leading to a large absorption surface). The liver is smaller relative to its body weight and secretes less bile.
- **Breed:** They are observed in improved early-maturing, fast-growing, and more plethoric breeds.
- **Age:** Young animals are the most affected. In adults, they are often encountered in animals after culling.
- **Season:** These are mainly spring and summer diseases. They can sometimes occur in autumn, if the summer was dry, at the time of regrowth.
- **Climate:** Cooling produces sudden digestive atony that promotes bacterial proliferation. Climatic factors can also intervene by promoting intense growth of protein-rich grass.
- **Intestinal parasites (strongyles, esophagostomes):** create entry points for anaerobic bacteria through their traumatic action. Quite frequently, in emotional cattle not accustomed to being handled, fulminating enterotoxemia appears within hours following an antiparasitic treatment. This treatment causes physiological stress (adrenaline production), originating a fleeting hyperglycemia, a factor of digestive paresis that does not allow the elimination of toxic bacteria and thus causes enterotoxemia.
- **Feeding:** The nature of the feed and sudden changes in diet can cause enterotoxemia.
  - Excess protein (turning out to grass) and lack of cellulosic bulk produce proteolytic fermentations with upheaval of the ruminal flora leading to alkalosis and digestive atony, hence enterotoxemia.
  - To a lesser degree, excess carbohydrates (cereals) and lack of cellulosic bulk disrupt the ruminal flora and promote lactic fermentation, leading to acidosis and intestinal atony, which will result in enterotoxemia.
  - In young animals, rapid ingestion of large quantities of milk (the greediest) results in gastric distension, decreased gastric acidity, hence multiplication of anaerobes.

2. Determining factors:

2.1 The bacteria:

Anaerobic bacteria of the genus *Welchia* producing exotoxins of a protein nature are the determining agents of the disease. These bacteria express their pathogenic power in anaerobic environments and when the intestinal environment is modified (digestive atony) by the change from an acidic pH to a basic pH. Their main pathogenic action is linked to their toxin-producing power.

Septicemia is late and secondary; it occurs in the terminal phase of the disease when the bacteria invade the whole body after crossing the intestinal barrier.

2.2 Toxins: Eleven different antigens are distinguished, the main ones being represented by alpha, beta, epsilon, and iota toxins. These toxins are produced by clostridia as follows:

- *Welchia perfringens* or type A: alpha toxin
- *W. agni* or type B: beta and epsilon
- *W. agni* var. *paludis* or type C: beta
- *W. agni* var. *wilsdoni* or type D: epsilon
- *W. perfringens* or type E: iota
- **Alpha toxin:** strongly hemolytic, slightly lethal but quite necrotizing. It causes hyperthermia and increases permeability.
- **Beta toxin:** strongly lethal and necrotizing. It causes hemorrhagic inflammation of the intestinal mucosa, which, once destroyed, allows toxins to pass into the blood.
- **Epsilon toxin:** consists of a protoxin that, under the effect of trypsin, becomes strongly lethal and necrotizing. Its secretion requires a protein-rich environment.
- **Iota toxin:** responsible in calves for a rapidly fatal hemorrhagic and necrotizing diarrhea.

Symptoms and lesions:

Depending on the type of bacteria and the age of the animals, different clinical forms are observed:

- In sheep:

- **Type B: "Lamb dysentery":** This form is observed especially in lambs aged 1 to 15 days. It can be peracute, fulminating, or progress to death in a few days after anorexia, abdominal pain, and yellowish diarrhea quickly becoming blood-tinged. Lesions show hemorrhagic inflammation of the small intestine with necrotic areas or hemorrhagic halos. The colon is very inflamed.
- **Type C: "Necrotizing hemorrhagic enteritis of lambs":** This condition, found in lambs aged 1 to 3 days, resembles lamb dysentery. It is characterized by necrosis of the small intestinal mucosa (ileum and jejunum) with sometimes hemorrhagic intestinal contents. The abomasum may also be affected.
- **Type D: "Enterotoxemia. Pulpy kidney disease":** This clostridial disease can be encountered at any age but is more frequent in suckling lambs less than 2 weeks old or in fattening sheep. This condition is characterized by a fulminating course (sudden death) in the finest subjects of the flock. In less severe forms, nervous symptoms (drunken gait, convulsions, opisthotonos, circling, etc.), acute pulmonary edema (presence of white foam at the nostrils), and sometimes diarrhea may be observed, with progression most often fatal within 24 to 48 hours. Necrosis of liver cells results in hyperglycemia and consequently glucosuria. At autopsy, subjects dying of enterotoxemia show excellent body condition with stomachs full of food (undigested milk in the abomasum of lambs). In fulminating forms, softening of the kidneys, discolored and friable liver,

congestive or even hemorrhagic areas (intestine, heart, muscles), and lesions of edema and cerebral degeneration on histological examination are observed.

- **Type C: "Enterotoxemia of adult sheep":** It is encountered in sheep aged 6 to 24 months with a lesion picture identical to pulpy kidney disease.

- In cattle:

Occasionally in calves aged 15 days to 2 months. It results in sometimes hemorrhagic diarrhea with rapid weakening and death. Most often in growing young cattle, the severity of the lesions usually leads to death within a few hours.

At autopsy, a full rumen, empty hemorrhagic intestines, a congestive abomasum, and a yellowish, mottled liver are observed.

Diagnosis:

Gastro-enterotoxemias can be confused with chemical and plant intoxications, tetanus, listeriosis, and snake envenomation. Symptoms and lesions can guide diagnosis, but it can only be confirmed by identifying the microbial type involved by a specialized laboratory.

Treatment:

If the development of infection is detected early enough, massive injections of immediately active penicillin (G) every two hours can stop the disease. Unfortunately, the rapidity of progression generally prevents the implementation of this antibiotic therapy. However, when a first animal has died in a group, taking the temperature of the other animals makes it possible to detect and, if necessary, treat one or two sick animals. Their temperature is then elevated to 41-42°C. For animals at pasture, immediately treat animals that are "lagging behind." Tetracyclines and erythromycin can also be used. Some authors advise 3 injections of anti-gangrene serum at 2-day intervals at doses varying from 20 to 80 cm^3. Cardiotonics, analeptics, and lipotropics are effective adjuvants to specific therapy.

Prophylaxis:

/ Sanitary:

It is essentially based on balancing the dietary ration. Any dietary imbalance concerning energy excess, excess nitrogenous matter, and lack of fiber should be avoided.

/ Medical:

- **Cattle:** Battery calves should be vaccinated with 2 to 3 injections of vaccines at 15-day intervals, also administering phenergan and cortisone.
- **Sheep:**
    1. Vaccinate pregnant ewes twice at 15-day intervals with an annual booster.
    2. Vaccinate lambs at 2 months with a booster at 6 months for vaccinated mothers.
    3. When mothers are unvaccinated, administer a half-dose of vaccine at 2 weeks with a booster 7 days later.

The most important scientific words translated into English (Chapter 7)

Terme Technique (Français)	Traduction Scientifique (English)
Entéropathies	Enteropathies
Diarrhée osmotique	Osmotic diarrhea
Entérotoxine microbienne	Microbial enterotoxin
Phytobézoard / Trichobézoard	Phytobezoar / Trichobezoar
Occlusion intestinale	Intestinal occlusion
Invagination	Intussusception
Volvulus	Volvulus
Colibacillose diarrhéique	Diarrheic colibacillosis
Escherichia coli entérotoxigène	Enterotoxigenic *E. coli* (ETEC)
Entérotoxémies	Enterotoxemias
Clostridium (Welchia) perfringens	*Clostridium perfringens*
Toxine alpha, bêta, epsilon, iota	Alpha, beta, epsilon, iota toxin
Maladie du rein mou (Pulpy kidney disease)	Pulpy kidney disease

CHAPTER VIII LIVER DISEASES

A. HEPATITIS

A1. TOXIC HEPATITIS

Various causes result in the classic lesion of toxic hepatitis, which is centrilobular damage often accompanied by necrosis. These causes include:

- Minerals: arsenic, phosphorus, and sometimes selenium
- Organic poisons: carbon tetrachloride, hexachloroethane, etc.
- Plant poisons: ragwort (Senecio), heliotrope, lupine, clover, etc.

A2. INFECTIOUS HEPATITIS

Liver damage caused by infectious agents primarily results in hepatic necrosis. The most common conditions are salmonellosis, listeriosis, and leptospirosis.

A3. PARASITIC HEPATITIS

Severe hepatitis is encountered especially in liver fluke disease (fascioliasis), hydatid disease (echinococcosis), and during the migration of ascarid larvae.

A4. CONGESTIVE HEPATITIS]{.underline}

This is mainly seen in heart failure, causing centrilobular degeneration.

Symptoms:

Symptoms are highly variable; affected animals present with anorexia, psychical depression, episodes of excitement, muscle weakness, and jaundice (icterus). Often, the animal also shows alternating constipation and diarrhea, as well as photosensitization.

Lesions:

The liver is enlarged (hypertrophied). The lobular pattern is more visible due to engorgement of the centrilobular vessels or centrilobular necrosis.

In infectious hepatitis, lesions are disseminated. In parasitic hepatitis, lesions are traumatic in nature, with local hemorrhages beneath the capsule. In congestive hepatitis, vascular engorgement and fatty infiltration of the parenchyma are observed.

Diagnosis:

Diagnosis of hepatitis is difficult due to the diversity of symptoms encountered, unless jaundice and photosensitization have manifested.

Treatment:

Attempts should be made to support the animal during the acute phase of the disease and await hepatic tissue regeneration to restore normal functions.

- Provide intravenous feeding with glucose and electrolyte solutions.
- Administer amino acid mixtures containing methionine.
- However, protein should be avoided due to the risk of ammonia toxicity.
- Ensure a diet rich in carbohydrates and calcium, and low in fats and proteins.

B. LIVER DEGENERATION

Hepatic degeneration is often secondary to another condition, but it can be primary, resulting from fatty or amyloid overload.

Etiology

Primary degeneration occurs in high-producing animals receiving a rich diet, or in cows receiving insufficient food at the end of gestation.

Hepatic degeneration is also encountered in prolonged anorexia, prolonged high fever, toxemias accompanying infectious diseases, poisonings by phosphorus, lead, arsenic, carbon tetrachloride, and copper, toxemias due to molds, icterogenic photosensitization, as well as endocrine disorders.

Pathogenesis

Starvation or overfeeding can lead to adrenal degeneration, abnormal ketogenesis, and a fat-laden liver. Hepatic degeneration occurring after poisonings may result from the toxic agent interfering with hepatic protein synthesis.

Symptoms and Lesions

Symptoms vary with the clinical circumstances and their complications:

- *In ketosis*, the animal primarily shows nervous signs, manifested by tremors, head bobbing, stumbling (titubation), ruminal stasis, and intermittent anorexia. Ketone bodies are noted in the urine.
- *In liver degeneration*, the animal shows anorexia, signs of indigestion, moaning or grunting, rapid pulse with normal temperature, slightly congested mucous membranes, and moderate jaundice.
- *In malnutrition* during the terminal stages of gestation, animals present with anorexia, weakness, paresis, and anemic, sometimes icteric, mucous membranes.

Diagnosis

Diagnosis should be based on case history (commemorative findings) and the results of liver function tests.

Treatment

- A 5% dextrose solution intravenously.
- 60 g of choline chloride.
- 2 to 5 mg per 100 kg body weight of Dexamethasone intramuscularly.
- Provide a good quality diet.

C. HEPATIC CIRRHOSIS

Etiology

This is an enzootic disease that appears after ingestion of plants from the *Senecio* (ragwort) and *Crotalaria* families.

Clinical Findings

The disease manifests clinically by weight loss (emaciation), jaundice, disturbances of consciousness, involuntary movements and incoordination. From a lesional standpoint, it is characterized by an increase in connective tissue and degeneration of the parenchymal cells of the organ.

Diagnosis

Diagnosis is easy at necropsy. In contrast, diagnosis in living animals can be difficult due to the slow onset of signs of cirrhosis after plant ingestion. Among liver function tests, only biopsy can provide a definitive diagnosis of cirrhosis.

Treatment

Once cirrhotic lesions are established, therapy is hopeless.

Prophylaxis

In enzootic areas, the herd must be placed in an environment free of toxic plants. Add choline chloride to the ration at a rate of 30 g per cow per day.

C. LIVER ABSCESSES

Etiology

Hepatic abscesses are often encountered in cattle, accompanying infection of other organs. They are found in traumatic reticuloperitonitis, metritis, mastitis, omphalophlebitis, and also in diseases such as tuberculosis, actinobacillosis, and necrobacillosis. The most frequently encountered organisms are: *Spherophorus necrophorus* (now *Fusobacterium necrophorum*), *Escherichia coli*, *Corynebacterium pyogenes* (now *Trueperella pyogenes*), *Pseudomonas pyocyaneus* (*Pseudomonas aeruginosa*), and micrococci.

Symptoms

If abscesses are multiple, they cause progressive weight loss and weakening of the animal, inconsistent jaundice, and sometimes organ enlargement (hypertrophy) palpable behind the last right rib. Palpation and percussion reveal local pain.

Treatment

Even if the disease is diagnosed in the living animal, treatment yields no satisfaction (is ineffective).

The most important scientific words translated into English (Chapter 8)

Terme Technique (Français)	Traduction Scientifique (English)
Hépatite toxique	Toxic hepatitis
Lésion centrolobulaire	Centrilobular lesion
Hépatite infectieuse	Infectious hepatitis

Terme Technique (Français)	Traduction Scientifique (English)
Hépatite parasitaire	Parasitic hepatitis
Douve du foie (Fasciolose)	Liver fluke (Fascioliasis)
Hépatite congestive	Congestive hepatitis
Ictère	Jaundice (Icterus)
Photosensibilisation	Photosensitization
Dégénérescence du foie	Liver degeneration
Stéatose hépatique	Hepatic steatosis (fatty liver)
Cirrhose hépatique	Hepatic cirrhosis
Plantes du genre Senecio et Crotalaria	*Senecio* and *Crotalaria* plants
Abcès du foie	Liver abscesses
Fusobacterium necrophorum	*Fusobacterium necrophorum*
Trueperella pyogenes	*Trueperella pyogenes*