SIMPLE INDIGESTION IN RUMINANTS

- Simple indigestion is a minor disturbance in ruminant GI function that occurs most commonly in cattle and rarely in sheep and goats.
- Simple indigestion is a diagnosis of exclusion and is typically related to an abrupt change in the quality or quantity of the diet.

ETIOLOGY:

- Almost any dietary factor that can alter the intraruminal environment can cause simple indigestion. The disease is common in hand-fed dairy and beef cattle because of variability in the quality and quantity of their feed.
- Dairy cattle may suddenly eat excessive quantities of highly palatable feeds such as corn or grass silage; beef cattle may eat excessive quantities of relatively indigestible, poor-quality roughage during winter.

- During drought, cattle and sheep may be forced to eat large quantities of poor-quality straw, bedding, or grain.
- Simple indigestion can result from suddenly changing the feed, using spoiled or frozen feeds, introducing urea to a ration, turning cattle onto a lush cereal grain pasture, or introducing feedlot cattle to a high-level grain ration

- Simple indigestion is usually associated with a sudden change in the pH of the ruminal contents, such as a decrease in ruminal pH due to rapid fermentation of ingested carbohydrates or an increase in ruminal pH due to forestomach hypomotility and putrefaction of ingested feed.
- It can also result from accumulation of excessive quantities of relatively indigestible feed that may physically impair rumen function. Multiple animals are usually simultaneously affected because simple indigestion has a nutritional basis, although the severity of the clinical signs can vary among animals.

CLINICAL FINDINGS:

- Clinical signs depend on the type of animal affected and cause of the disorder. Overfeeding of silage causes anorexia and a moderate drop in milk production in dairy cattle.
- The rumen is usually full, firm, and doughy; primary contractions are decreased in rate or absent, but secondary contractions may be present although usually decreased in strength.
- Temperature, pulse, and respiration are normal. The feces are normal to firm in consistency but reduced in amount. Recovery usually is spontaneous within 24–48 hr.

- Simple indigestion due to excessive feeding of grain results in anorexia and ruminal hypomotility to atony (stasis).
- The rumen is not necessarily full and may contain excessive fluid. The feces are usually soft to watery and foul smelling.
- The mechanism for diarrhea formation is uncertain but is most likely due to increased luminal osmolality as a result of the rapid degradation of ingested carbohydrates.
- The affected animal is bright and alert and usually begins to eat within 24 hr. A more severe digestive upset due to excessive feeding of grain is described as grain overload

DIAGNOSIS:

- A diagnosis of simple indigestion is based on a history of an abrupt change in the nature or amount of the diet, multiple animals being affected, and most importantly the exclusion of other causes of forestomach dysfunction.
- The diagnosis is confirmed by collection and examination of ruminal fluid, which may have an abnormal pH (<6 or >7), decrease in the numbers and size of protozoa, or prolonged methylene bluereduction time (a measure of bacterial metabolic activity).

- The systemic reaction and painful responses to deep palpation of the xiphoid in traumatic reticuloperitonitis are not seen.
- The history and the absence of ketonuria help eliminate clinical ketosis from consideration. The possibility of left displaced abomasum usually can be eliminated by simultaneous percussion and auscultation.
- Vagal indigestion, abomasal volvulus, and cecocolic volvulus become more readily detectable as they progress. Grain overload is differentiated from simple indigestion by its greater severity and the pronounced fall in the pH of the rumen contents to <5.5.

TREATMENT:

- Treatment is aimed at correcting the suspected dietary factors. Spontaneous recovery is usual when animals are fed a typical ruminant diet.
- Administration of ~20 L of warm water or saline via a stomach tube, followed by vigorous kneading of the rumen, may help restore rumen function in adult cattle. Magnesium hydroxide PO may be useful when excessive amounts of grain have been ingested, but magnesium hydroxide should only be administered to cattle documented to have low ruminal pH (<6); otherwise, excessive forestomach and systemic alkalinization can result.

- Purported rumenatorics (eg, nux vomica, ginger, tartar emetic, parasympathomimetics) are not recommended as ancillary treatments.
- If too much urea or protein has been ingested, vinegar (acetic acid) may be administered PO to return rumen pH to the normal range. If the number or activity of ruminal microbes is reduced, administration of 4–8 L of ruminal fluid from a healthy cow will help.
- Oral or intravenous electrolyte solutions may be needed to correct electrolyte and acid-base abnormalities, particularly in dehydrated cattle.

GRAIN OVERLOAD IN RUMINANTS

ETIOLOGY AND PATHOGENESIS:

- The disease is most common in cattle that accidentally gain access to large quantities of readily digestible carbohydrates, particularly grain.
- Grain overload also is common in feedlot cattle when they are introduced to heavy grain diets too quickly.
- Wheat, barley, and corn are the most readily digestible grains; oats are less digestible. Less common causes include engorgement with apples, grapes, bread, batter's dough, sugar beets, potatoes, mangels, or sour wet brewer's grain that was incompletely fermented in the brewery.

- The amount of feed required to produce acute illness depends on the kind of grain, previous experience of the animal with that grain, the nutritional status and condition of the animal, and the nature of the ruminal microflora.
- Adult cattle accustomed to heavy grain diets may consume 30–45 lb (15–20 kg) of grain and develop only moderate illness, whereas others may become acutely ill and die after eating 20 lb (10 kg) of grain

- Ingestion of toxic amounts of highly fermentable carbohydrates is followed within 2–6 hr by a change in the microbial population in the rumen. The number of gram-positive bacteria (such as *Streptococcus bovis*) increases markedly, which results in the production of large quantities of lactic acid.
- The rumen pH falls to ≤5, which destroys protozoa, cellulolytic organisms, and lactate-utilizing organisms, and impairs rumen motility. The low pH allows the lactobacilli to utilize the carbohydrate and to produce excessive quantities of lactic acid.
- The superimposition of lactic acid and its salts, l-lactate and d-lactate, on the existing solutes in the rumen liquid causes osmotic pressure to rise substantially, which results in the movement of excessive quantities of fluid into the rumen, causing fluid ruminal contents and dehydration.

- The low ruminal pH causes a chemical rumenitis, and the absorption of lactate, particularly d-lactate, results in lactic acidosis and acidemia. In addition to metabolic (strong ion) acidosis and dehydration, the pathophysiologic consequences are hemoconcentration, cardiovascular collapse, renal failure, muscular weakness, shock, and death.
- Animals that survive may develop mycotic rumenitis in several days and hepatic abscesses several weeks or months later. They may have evidence of ruminal epithelial damage at slaughter. The relationship between grain overload and chronic laminitis in cattle is unclear

CLINICAL FINDINGS:

- Carbohydrate engorgement results in conditions ranging from simple indigestion to a rapidly fatal acidemia and strong ion (metabolic) acidosis.
- The interval between overeating and onset of signs is shorter with ground feed than with whole grain, and severity increases with the amount eaten.
- A few hours after engorgement, the only detectable abnormality may be an enlarged rumen and possibly some abdominal pain (manifest by belly kicking or treading of the hindlimbs).

- In the mild form, the rumen movements are reduced but not entirely absent, the cattle are anorectic but bright and alert, and diarrhea is common. The animals usually begin eating again 3–4 days later without any specific treatment.
- Within 24–48 hr of the onset of severe overload, some animals will be recumbent, some will be staggering, and others will be standing quietly; all will be completely off feed. Immediately after consuming large quantities of dry grain, cattle may gorge themselves on water, but once ill they usually do not drink at all.

Body temperature is usually below normal, 98°–101°F (36.5°–38.5°C); however, in animals exposed to the sun in hot weather, it may be increased to 106°F (41°C). Respirations tend to be shallow and rapid, up to 60–90/min.

The heart rate usually is increased in accordance with severity of the acidemia; the prognosis is poor for cattle with heart rates >120 bpm. Diarrhea is common and usually profuse and malodorous.

The feces are soft to liquid, yellow or tan, and have an obvious sweet-sour odor. The feces frequently contain undigested kernels of the feed that has induced the overload. In mild cases, dehydration equals 4%–6% body wt, but losses may reach 10%–12% in severe cases.

- Acute laminitis may be present and is most common in those animals not severely affected; chronic laminitis may develop weeks or months later.
- Anuria is a common finding in acute cases, and diuresis after fluid therapy is a good prognostic sign.
- Death may occur in 24–72 hr, and rapid development of acute signs, particularly recumbency, indicates a need for aggressive treatment. A decrease in heart rate, increase in temperature, return of ruminal movement, and passage of large amounts of soft feces are more favorable signs.

DIAGNOSIS:

- The diagnosis is usually obvious if the history is available and multiple animals are affected. The diagnosis can be confirmed by the clinical findings, a low ruminal pH (<5.5 in cattle unaccustomed to a high grain diet), and examination of the microflora of the rumen for presence of live protozoa.
- When only one animal is involved and there is no history of engorgement, the diagnosis is less obvious, but the clinical signs—a static rumen with gurgling fluid sounds, diarrhea, ataxia, and a normal temperature—are characteristic. Rumen fluid analysis in these animals is required to confirm the diagnosis of grain overload.

- To avoid an increase in pH on exposure to air, the pH of rumen fluid obtained by ororuminal stomach tube or ruminal paracentesis should be checked promptly.
- Normally, the pH in cattle on roughage is 6–7; in those on a high grain diet, 5.5–6. Values <5.5 are strongly suggestive of grain overload, and a rumen pH <5 indicates severe acidemia and metabolic acidosis

- Wide-range (2–11) pH indicator paper is suitable for field use. Ruminal fluid should also be examined microscopically if access to a laboratory is available; fluid from affected cattle will have decreased numbers of protozoa (particularly large and medium-sized protozoa).
- In grain overload, a Gram stain of ruminal fluid will reveal a change from predominantly gram-negative bacteria (normal) to predominantly gram-positive bacteria, with a concomitant loss in bacterial diversity.

- Increased blood d-lactate and l-lactate and inorganic phosphate concentrations, mild hypocalcemia, and reduced urinary pH are also seen, but it is seldom necessary to check such values to make a firm diagnosis.

- The diagnostic problem is to properly assess which animals require vigorous therapy (or slaughter), which require supportive therapy, which have only a mild indigestion that will correct itself if water and grain intake are restricted and hay and exercise are provided, and which need nothing beyond their routine care and ration. In an outbreak of overload involving several animals, it is necessary to identify those animals that need the most intensive therapy and those that will recover with minimal medical therapy.

TREATMENT:

- For all cattle suspected of having eaten large quantities of concentrate, it is believed that restricting water intake for the first 18–24 hr is helpful, although this has not been proved.
- If overload is serious, slaughter for salvage should be considered; in feeders nearing the end of their feeding period, it may well be the most economic choice.
- Mortality is high in severely affected animals unless aggressive therapeutic measures are started early

- In such animals, removal of rumen contents and replacement with ingesta taken from healthy animals is necessary.
- In animals still standing, rumenotomy is preferred to rumen lavage, because animals may aspirate during the lavage procedure and only rumenotomy ensures that all ingested grain has been removed.
- Rumen lavage may be accomplished with a large stomach tube if sufficient water is available.

- A large-bore tube (2.5 cm inside diameter, 3 m long) should be used, and enough water added to distend the left paralumbar fossa; gravity flow is then allowed to empty out what it will.
- Repeating this 15–20 times achieves the same results (and requires about as much time) as using rumenotomy to empty and wash out the rumen with a siphon.

- Emptying the rumen should be followed by rumen inoculation) and, if not accomplished before signs of severe illness are evident, by rigorous fluid therapy to correct the metabolic acidosis and dehydration and to restore renal function. Initially, over a period of ~30 min, 5% sodium bicarbonate solution should be given IV (5 L/450 kg).
- During the next 6–12 hr, a balanced electrolyte solution, or a 1.3% solution of sodium bicarbonate in saline, may be given IV, up to as much as 60 L/450 kg body wt..

- Urination should resume during this period.
- Usually, it is unnecessary and even undesirable to also administer antacids PO (or intraruminally), particularly if IV sodium bicarbonate has been administered. Procaine penicillin G (22,000 U/kg/day) should be administered IM to all affected animals for at least 5 days to minimize development of bacterial rumenitis and liver abscesses.
- Thiamine should also be administered IM to facilitate metabolism of l-lactate via pyruvate and oxidative phosphorylation; animals with grain overload also have low concentrations of thiamine in rumen fluid because of increased production of thiaminase by ruminal bacteria. There is no effective preventive treatment for mycotic rumenitis

PREVENTION:

- Accidental access to concentrates for which cattle have developed an appetite, in quantities to which they are unaccustomed, should be avoided.
- Feedlot cattle should be introduced gradually to concentrate rations over a period of 2–3 wk, beginning with a mixture of ≤50% concentrate in the milled feed containing roughage.