

Diseases of the Ruminant Stomachs and Intestines of Cattle

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General Comments on Ruminant Gastrointestinal Dysfunction

In the study of diseases of the ruminant stomachs the first consideration is the clinical and laboratory findings which attract special attention to the stomachs. The first step is the recognition that a basic syndrome of ruminant gastrointestinal dysfunction occurs without evidence of disease in another system which might suggest that the dysfunction is secondary.

The clinical findings which suggest primary ruminant gastrointestinal dysfunction include the following:

- * Inappetence to anorexia, failure to regurgitate and chew the cud.
- * Ruminal atony or hypermotility observed visually, and detectable on auscultation.
- * Abnormal rumen contents palpable through the abdominal wall. The contents may feel dry or fluid splashing sounds may be detectable.
- * Visible abnormality of the abdomen, either distension or gauntness.
- * Abdominal pain, usually subacute, and characterized by humping of the back, or acute colicky signs of kicking at the belly and stretching. Pain may also be detectable on percussion or deep palpation of the abdomen if there is peritonitis either local or diffuse.
- * Abnormal faeces. The faeces are usually increased in amount and sweet-sour smelling in carbohydrate engorgement. In most other diseases of the ruminant stomach the amount of faeces is reduced (scant) and the faeces are pasty, foul-smelling and appear overdigested because of the long sojourn in the alimentary tract. A complete absence of faeces for 24-48 hours is not uncommon with diseases of the ruminant stomach and may be confused for an intestinal obstruction.
- * The temperature, heart rate and respirations are variable and often normal. If there is an associated acute peritonitis the temperature is usually elevated in acute diffuse peritonitis with toxæmia, it is commonly normal or subnormal and in subacute and chronic peritonitis the temperature is usually normal. In most other diseases of the ruminant stomachs except carbohydrate engorgement and abomasal torsion where dehydration, acidosis and gastric infarction occur, vital signs may be within the normal range.

The stomachs of ruminants are closely associated anatomically and functionally, and disease of one usually affects the others. The rumen is easily examined clinically and experimentally and it is usually used as an indicator of the state of the other stomachs. Bacterial digestion and fermentation, and physical maceration by contraction of the stomach walls are the two main functions of the forestomachs and the two are interdependent. Thus abnormality of one leads to the abnormality of the other and of the two the motility is most readily examinable. Ruminal motility is therefore used as an index of digestive function in the ruminant.

The plain muscle of the forestomachs has no intrinsic contractile power and the movement of the walls of these organs depends upon the integrity of the afferent and efferent nerves and the reticuloruminal motor center of the medulla. Both afferent and efferent fibres are carried in the vagus nerves and damage to one or other branches of nerve causes interference with normal movements and produces the syndrome of vagus indigestion.

When food enters the forestomachs it normally divides into layers, an upper layer of free gas and a lower layer of fluid containing gas bubbles and suspended food particles. A layer of undigested fibre floats on top and heavy material such as grain sinks to the bottom, often in the reticulum. Much mixing of the contents of the rumen and reticulum takes place during ruminal movements which occur at a rate of 1-3/minute, the more rapid rate occurring soon after feeding.

The movements occur in cycles commencing with a double reticular contraction, the second of which is accompanied by a strong contraction of the anterior dorsal sac of the rumen. These contractions pour the fluid reticular contents over the bulky food mass in the rumen. A contraction of the ventral sac follows and fluid is returned to the reticulum. During each reticular contraction fluid and food particles, particularly heavy grain, pass into the reticulo-omasal orifice and into the omasum and abomasum. It is this passage of heavy grain directly into the abomasum, without in many instances proper digestion in the rumen, which may lead to overloading of the abomasum and resultant displacement or torsion of this organ. It may also be important in the pathogenesis of enterotoxaemia caused by *Clostridium perfringens* Type D. If the floor of the reticulum is fixed to the ventral abdominal wall by adhesions

it may be impossible for fluid to pass into the reticulo-omasal orifice. This may be a factor in the development of some forms of vagus indigestion.

Eructation contractions occur in the dorsal sacs, pass forward to the cardia of the oesophagus and in conjunction with a reticular relaxation depress the level of the reticular fluid. The cardia relaxes and gas is expelled. If the ruminal contents are frothy it may be impossible for the cardia to be cleared and eructation to occur; ruminal tympany follows. Eructation contractions are independent of mixing contractions, their rate depending upon the pressure of the gas in the rumen. They occur for the most part, immediately after the mixing contractions.

Rumination also depends upon additional ruminal contractions which are interposed before mixing movements of the rumen. These special contractions keep the area of the oesophageal cardia flooded with reticular fluid. A voluntary movement by the animal follows: an inspiratory effort is made with the glottis closed; the negative pressure in the thorax is greatly increased and the reticular fluid, carrying some floating ingesta, is carried up to the pharynx. Defects of regurgitation are usually due to inability to create the necessary negative pressure in the thorax; this may occur in chronic pulmonary emphysema. In these circumstances there are usually visible efforts at regurgitation, often accompanied by grunting. Regurgitation ceases as soon as ruminal atony occurs because of absence of the ruminal contractions necessary to keep the cardiac region filled with fluid. However, regurgitation contractions play no part in the movement of the bolus up the oesophagus. Regurgitation also diminishes when regurgitation contractions are not stimulated by coarse fibre in the rumen. Cattle on pelleted or finely ground diets ruminate little or not at all. Rumination is also depressed by excitement and fear.

Rectal examination of the abdominal cavity is a useful aid in the assessment of the size of the rumen and consistency of its contents. A grossly enlarged abomasum due to impaction, dilatation or torsion may be palpable rectally. Occasionally an enlarged omasum is palpable too. **However, rectal findings are often negative because the abnormalities are in the anterior half of the abdomen.**

The examination of ruminal fluid for abnormalities is a valuable aid in the diagnosis of diseases of the ruminant stomachs. Changes in the pH of ruminal fluid and protozoan numbers and their activity occur in carbohydrate engorgement and other forms of indigestion. Paracentesis may be a valuable aid in the diagnosis of peritonitis associated with diseases of the ruminant stomachs and is discussed under special examination.

The differential diagnosis of causes of gastrointestinal dysfunction in ruminants is summarized in the Table.

In contrast with most other parts of the ruminant alimentary tract, and the stomach of non-ruminants, specific lesions of the mucosa of the forestomachs are uncommon. Penetration of the wall by metallic foreign bodies is a

common disease and dealt with below under the heading of traumatic reticuloperitonitis, but it is the peritonitis which causes interference with ruminal motility. Rarely there are actinomycotic or neoplastic lesions at the fundus of the reticulum which interfere with proper functioning of the oesophageal groove and lead to a syndrome of vagus indigestion described later. Rumenitis does occur commonly but only as a secondary change in acute carbohydrate engorgement and it is this which has such damaging effects on gut motility and fluid and electrolyte status and eventually kills most cows. The rumenitis may have a long-term effect on gut motility but its main significance is as a portal for infection leading to the development of hepatic abscesses. Ingested animal hairs, and plant spicules and fibres are also credited with causing rumenitis but no clinical signs have been associated with the lesions. Because of the high prevalence of rumenitis lesions in cattle on heavy concentrated feed, especially when the feed is awned barley, the awns have been incriminated as traumatic agents. In acute arsenic poisoning there is an early post-mortem dehiscence of the ruminal mucosa but no apparent lesions during life.

Some of the physiological factors which affect reticulorumen function and the clinical factors which cause reticulorumen dysfunction are summarized in the Table. As mentioned earlier, when reticulorumen dysfunction is present, particularly hypomotility, the problem is to decide if the cause is directly associated with the stomachs and/or the other parts of the alimentary tract, or if the cause is due to an abnormality of another system. Differentiation requires a careful clinical examination including simple laboratory evaluation of the rumen contents.

Reticuloruminal function and dysfunction

The ruminant and its rumen.

Motility. There are two independent contraction sequences.

Primary cycle - about every minute consists of a biphasic contraction of the reticulum followed by a monophasic contraction of the dorsal ruminal sac and then by a monophasic contraction of the ventral ruminal sac. These movements are concerned primarily with mixing the rumen contents and with assisting the passage of rumen contents into the omasum.

Secondary cycle - about every two minutes, are confined to the rumen and consist of a contraction of the dorsal sac followed by a contraction of the ventral sac. Usually associated with eructation.

The primary cycle movements of the reticulo-rumen and wholly dependent upon an extrinsic nerve supply. The forestomach is innervated by the vagi and splanchnic nerves, although only the vagi are essential for cyclical movements. In the vagi at the level of the diaphragm, the ratio of sensory nerve fibres to motor nerve fibres is 9:1 and, on this purely numerical basis, the vagi should be regarded as having a significant sensory role. Normal cyclical movements are

present after sectioning either the left vagus, the right vagus, the ventral trunk or the dorsal trunk. Further sectioning causes permanently impaired motility and total vagotomy results in complete stasis. By inference, therefore, a clinical lesion must destroy more than 50 per cent of the vagal innervation, if it is to produce permanent reticulo-ruminal hypomotility. In some clinico-pathological studies of "vagus indigestion" only 20% had nerve degeneration at necropsy.

The primary cycle movements result from coordinated nervous activity which emanates from *gastric centers* in the hind-brain and passes down vagal motor fibres to the various compartments of the forestomach. The effects of these vagal motor discharges may be enhanced by anticholinesterases (e.g., neostigmine) and abolished by drugs which block transmission either at preganglionic or postganglionic cholinergic nerve endings (e.g., atropine). During the quiescent period between primary cycle contractions, there are no nerve impulses passing down vagal motor nerve fibers to the reticulo-rumen which in part explains the inefficiency of parasympathomimetic drugs (e.g., Carbachol) in stimulating reticulo-ruminal movements in contrast to their effects on other regions of the gut.

The gastric centers must receive sensory inputs which arise principally from low-threshold tension receptors in the reticulum, from acid receptors in the abomasum and from mechanoreceptors and possibly chemoreceptors in the mouth. If the reticulum is distended, the tension receptors are stimulated and the excitatory sensory input to the gastric centers is increased. This reflexly increases the frequency, the amplitude and the duration of the primary cycle contractions. Such a mechanism probably accounts for the hypermotility observed after feeding or during a mild degree of bloat and, conversely, the reduced motility seen after a period of starvation or as a secondary feature of any disease which includes anorexia. In 75 per cent of the cases of vagus indigestion studied by Neal and Edwards there was histological evidence of induration and fibrosis affecting the medial wall of the reticulum and it is tempting to hypothesize that these lesions could abolish normal tension receptor activity and eliminate the excitatory drive to the gastric centers. This could account for those cases of hypomotility not accompanied by significant vagal damage. However, it is

difficult to explain the hypermotility of the rumen which is more common, at least in the earlier stages of the disease, than hypomotility.

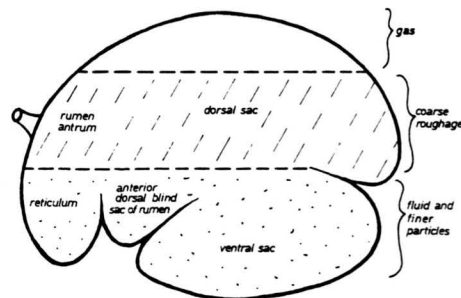
Acid receptors in the abomasum stimulate reticulo-ruminal movements and results in an increased flow of rumen contents into the abomasum.

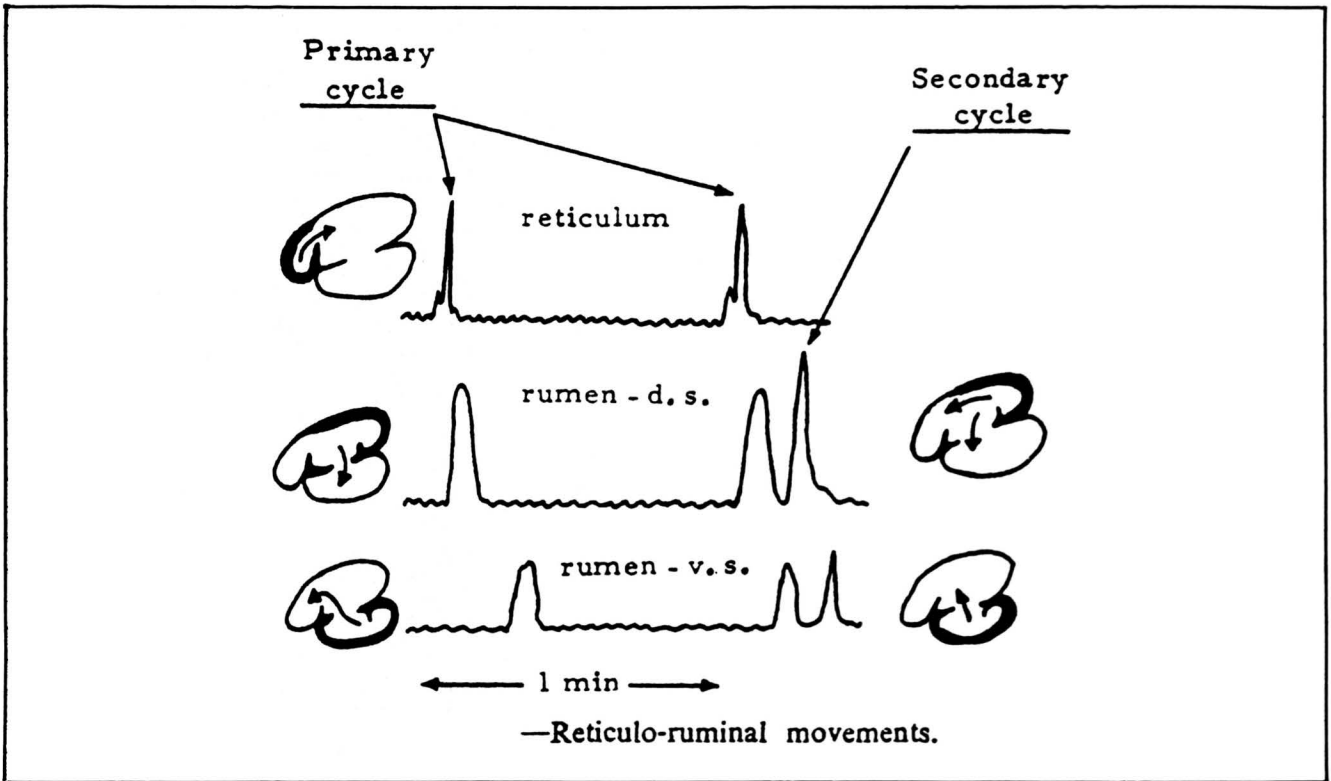
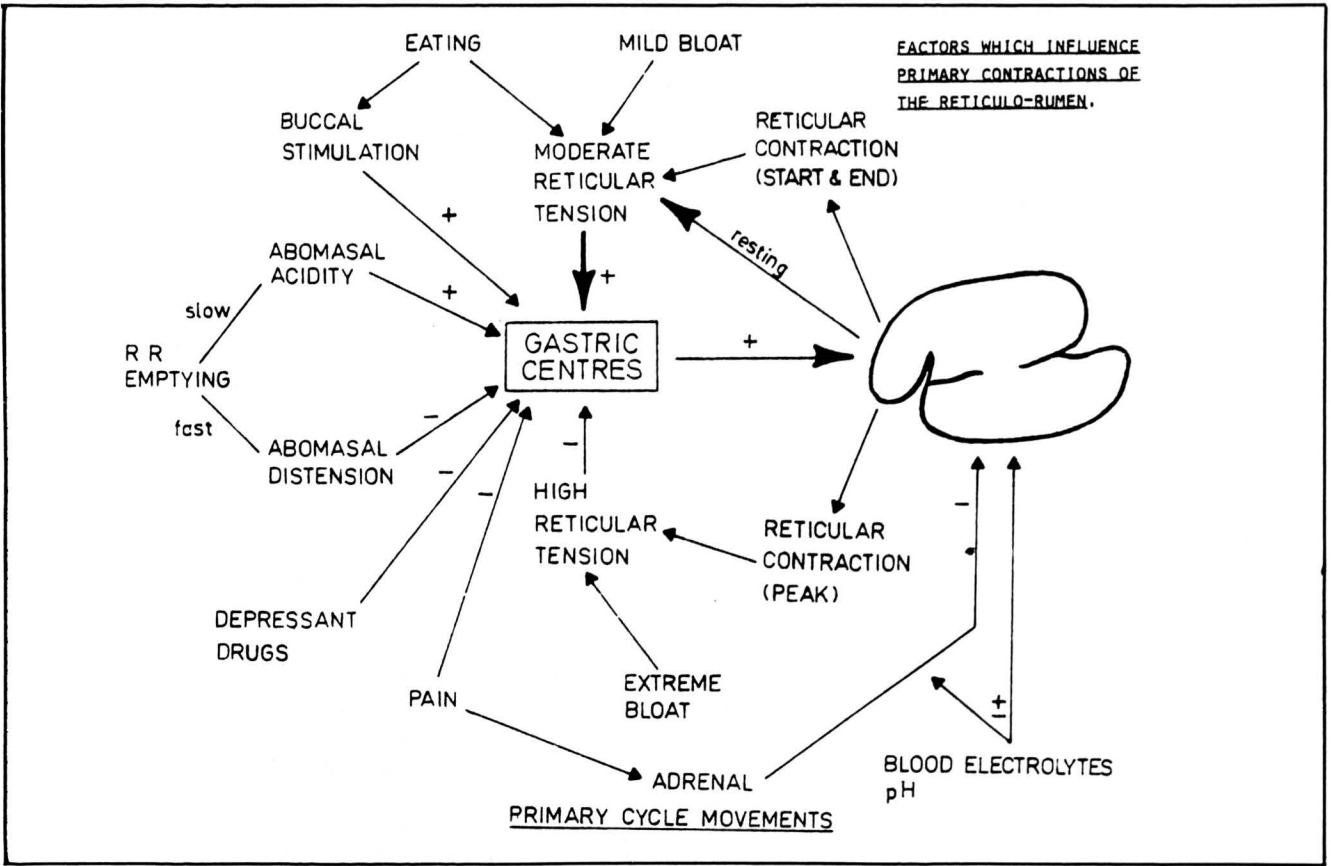
Counteracting the excitatory sensory inputs to the gastric centers are inhibitory inputs. One of these arises from high-threshold tension receptors in the reticulum innervated by sensory vagal fibres. They are activated normally at the peak of the reticular contraction thereby modifying reflexly (a) the final part of the reticular contraction and (b) the ruminal contraction. The combination of low-threshold (excitatory) reticular tension receptors and high-threshold (inhibitory) reticular tension receptors provides the reticulorumen with a mechanism for exerting some degree of control over the biochemical activity of the rumen micro-organisms. Increasing the frequency and the force of primary cycle (mixing) movements tends to increase the fermentation of rumen contents, whereas hypomotility depresses fermentation. Tension receptors in the abomasum also provide inhibitory sensory input to the gastric centers. In the event of abomasal over-filling, such as in impaction or displacement, this reflex inhibition may contribute to the reticulo-ruminal hypomotility observed.

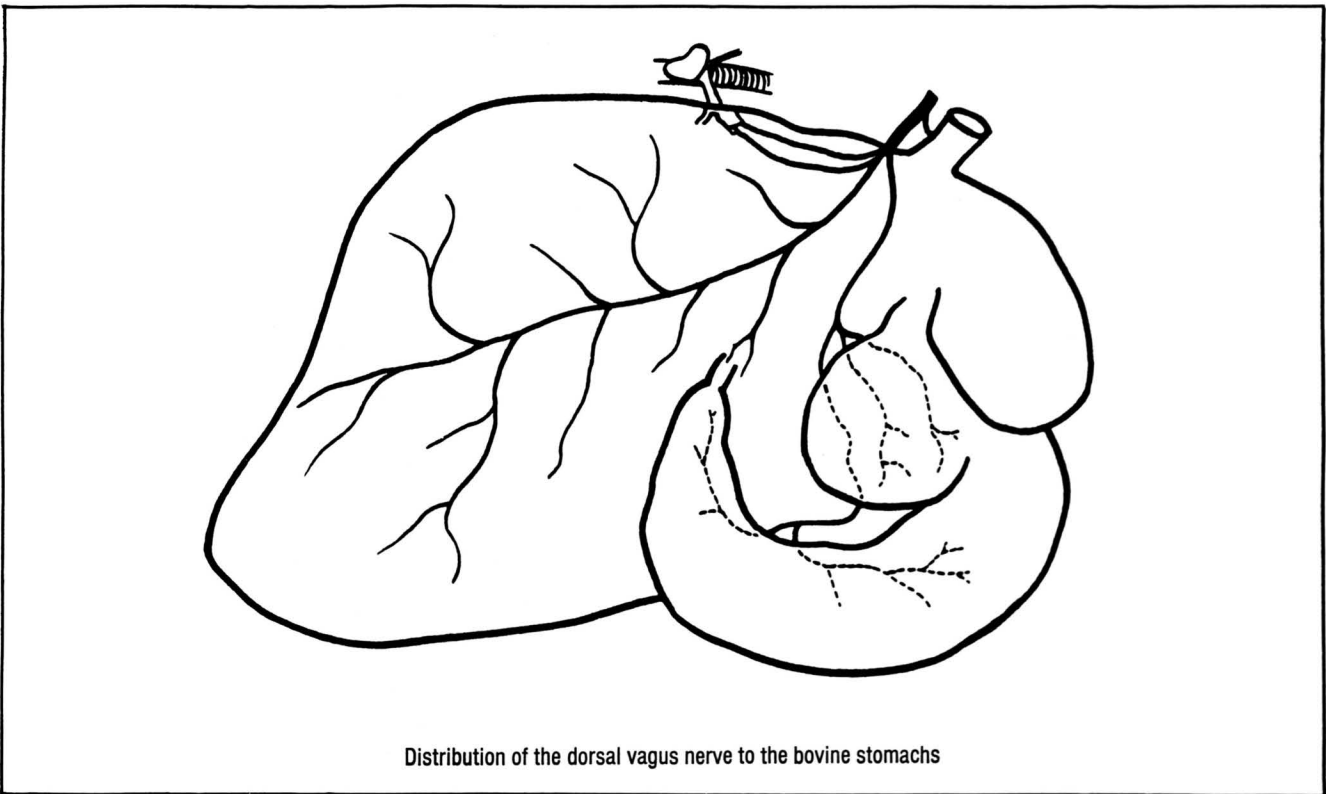
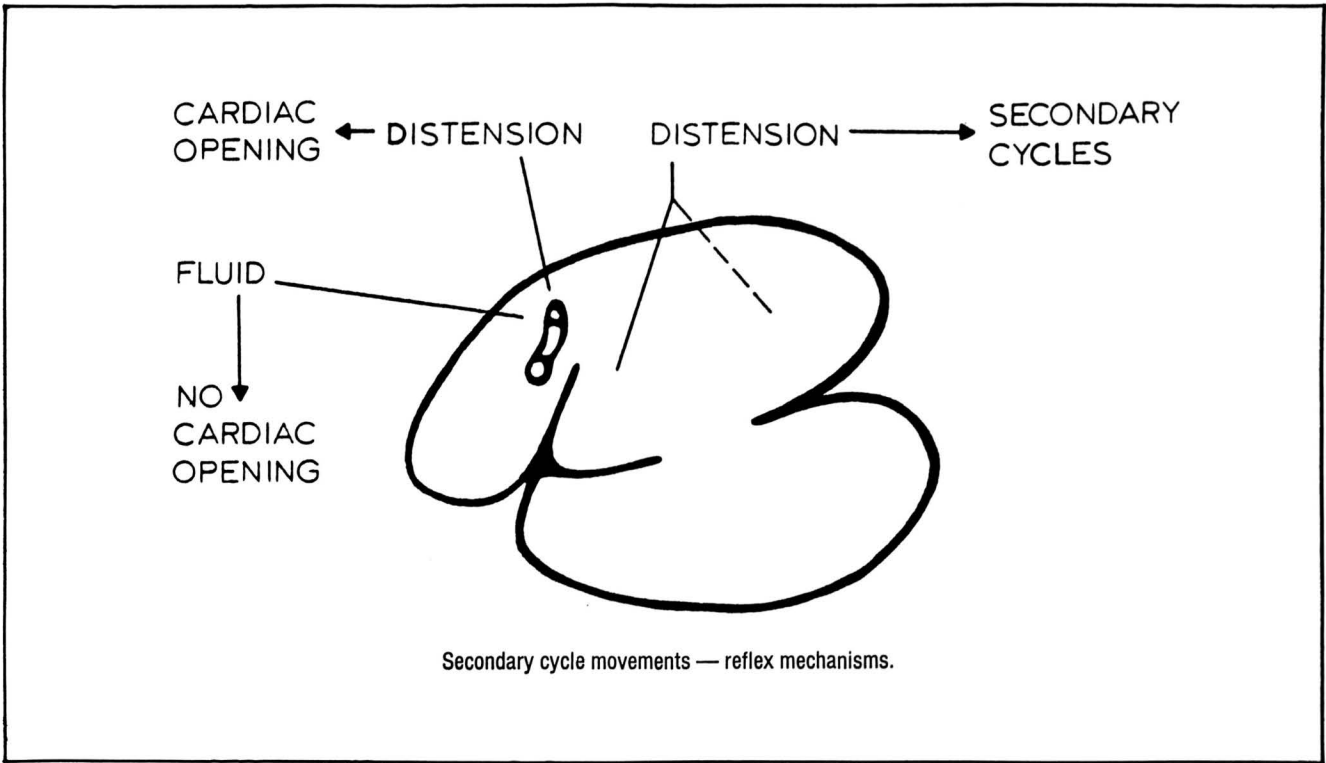
The principal factors which affect primary cycle movements are summarized in the figure and the table.

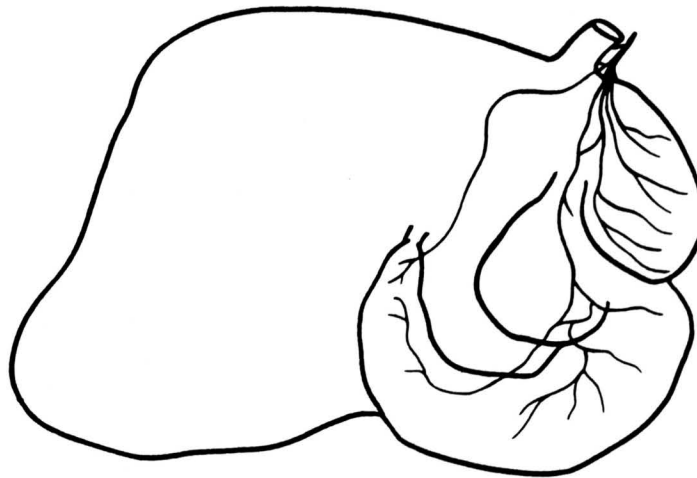
The secondary cycle movements involve only the rumen and are concerned principally with moving in a cranial direction the gas layer in the dorsal sac. The reflex excitation of secondary cycles is independent of that for primary cycles. The primary stimulus for secondary contractions is an increase in ruminal tension. The secondary contractions are associated with eructation. Secondary contractions move gas in a cranial direction to the cardia leading to a rise in gas tension in the region of the cardia which triggers the reflex opening of the cardia and the other events involved in eructation. Reflex cardiac opening does not occur if the cardia is covered with fluid (including foam). How can this information be used to determine the cause of *chronic bloat* in calves six to eight months of age, usually after weaning and within a few weeks after arrival in the feedlot?

Stratification of fluid layer, roughage layer and gas cap of normal rumen.









The Use of Pharmaceuticals, Fluid Therapy and Antimicrobials for Diseases of the Ruminant Stomachs, Abomasum, Intestines and Peritoneum of Cattle

1. Fluid Therapy:

Balanced fluid and electrolytes given by continuous intravenous infusion are indicated for dehydration and acid-base imbalances. The intravenous route is preferred because fluids given orally are poorly absorbed in the presence of rumen stasis.

The composition of the fluids is probably not critical for most situations. Balanced electrolyte mixtures (Ionalyte, Proionate) are satisfactory. The addition of glucose at 5% is indicated for recently calved cows to minimize mobilization of body fat stores and the fatty liver syndrome.

Large amounts of fluid are usually necessary to correct obvious clinical dehydration, and acid-base imbalances in mature cattle.

700 Kg cow

Hydration therapy (first 4 to 6 hrs)

50 to 8 litres

Maintenance therapy (next 20 to 24 hrs)

35 to 70 litres

(For further details see *Blood, Henderson & Radostits 5th Edition, pp. 35-50*)

2. Antimicrobial Therapy:

Other than the specific infections of the intestines the most common indication for the use of antimicrobials is for the treatment of peritonitis. Assuming that most cases of peritonitis in cattle are due to penetration of the reticulum by a foreign body it

3. Alkalinizing Agents:

Magnesium hydroxide at the rate of 500 g per 500

Kg B.W. given orally along with 2 to 4 gallons of water, usually only once for the treatment of carbohydrate engorgement (ruminal lactic acidosis). Sodium bicarbonate may be used as an alternative.

seems logical that a broad-spectrum antimicrobial is indicated. A mixed bacterial flora containing Gram-positive and Gram-negative aerobic and anaerobic bacteria are usually present. Sulfamethazine intravenously at a rate of 150 mg per Kg B.W. daily for 3 to 5 days is the drug of choice. Sulfa-methazine enters serous cavities effectively and therapeutic blood levels are maintained for 24 hours following an intravenous dose.

4. Rumen Transplant:

Rumen juice contains ruminal bacteria and protozoa and volatile fatty acids. Two to four gallons of fresh rumen juice strained from the rumen contents of a normal cow are indicated for cattle which have been anorexic for several days or in which the rumen microflora have been destroyed as in grain overload. Rumen contents placed directly into the rumen of cattle during a rumenotomy will often initiate normal rumination within a few hours provided that the original lesion is not impairing reticulo-rumen function or there is no toxemia present. Rumen transplantation is considered to be the best available ruminatoric.

5. *Diocetyl sodium sulfosuccinate (DOSS)*:

May be useful for emulsification and removal of crude oil and petroleum products from the rumen. May also be useful for the treatment of impaction of the abomasum. Mix 120-180 ml of 25% solution of DOSS with 10 liters of mineral oil for 450 Kg B.W. animal daily for 3 days. Fluid therapy with balanced electrolytes is also necessary.

DIFFERENTIAL DIAGNOSIS OF CAUSES OF GASTROINTESTINAL DYSFUNCTION

Disease	Epidemiology and History	Clinical Findings	Clinical Pathology	Response to Treatment
Simple indigestion	Dietary indiscretion, too much of a palatable, or indigestible or change of or damaged or frozen food. Can be outbreak	Simple gastrointestinal atony. Voluminous faeces during recovery	All values normal. Slight changes in ruminal acidity, should be self-buffered	Excellent just with time. Usually a mild purgative
Carbohydrate engorgement	Access to large amount readily fermentable carbohydrate when not accustomed. Enzootic in high grain rations in feedlots	Severe gastrointestinal atony with complete cessation ruminal activity. Fluid splashing sounds in rumen. Severe dehydration, circulatory failure. Apparent blindness then recumbency and too weak to rise. Soft odoriferous faeces	Haemoconcentration with severe acidosis, pH of 4.5 in rumen, serum phosphorus levels up to 10 mg/dl. No living protozoa in rumen	Intensive fluid and electrolyte therapy necessary for survival. Rumenotomy or rumen lavage may be necessary. Alkalinizing agents
Ruminal tympany	Frothy bloat on lush legume pasture, or low roughage feedlot ration, especially lucerne hay. Free gas bloat secondary, occasionally primary on preserved feed	Gross distension of abdomen, especially high up on left. Sudden onset. Severe pain and respiratory distress. Rumen moves vigorously until end. Sloppy faeces. Resonance on percussion over rumen.	Nil	Excellent if in time, stomach tube for free gas. Froth-dispersing agent in frothy bloat. Severe cases may require trocarization or emergency rumenotomy
Acute traumatic reticuloperitonitis	Exposure to pieces of metal. Sporadic. Usually adult cattle	Sudden onset gastrointestinal atony with mild fever. Pain on movement and percussion on xiphoid, humped back. Constipation common. Lasts 3 days, then improvement begins	Neutrophilia and shift to left	Good to conservative medical treatment, or surgical treatment
Chronic traumatic reticuloperitonitis	Previous history of acute local peritonitis	Inappetence to anorexia; loss of weight; temperature, pulse and respirations normal; rumen small and atonic, chronic moderate bloat common, faeces scant, grunt may be detectable on deep palpation over xiphoid, reticular adhesions on laparotomy	Haemogram depends on stage and extent of inflammation	Unfavourable
Vagus indigestion	May or may not have history of acute local peritonitis. Inappetence and progressive distension of abdomen during late pregnancy and no response to treatment with laxatives	Progressive distension of abdomen, scant soft faeces, anorexia, rumen distended with well macerated and frothy contents, persistent moderate bloat, hypermotile initially and atonic later, temperature normal, heart rate variable, large L-shaped rumen rectally, abomasal impaction in some, large loss of weight, eventual recumbency dehydration & weakness	Varying degree of dehydration, alkalosis, hypochloraemia and hypokalaemia	Inadequate response to treatment medically or surgically. Mild cases near term may respond spontaneously following parturition
Abomasal impaction (dietary)	Excessive intake of poor quality roughage during cold weather. Outbreaks. Cattle eating crops contaminated with sand	Anorexia, moderate abdominal distension, weight loss, scant faeces, weak, recumbent. Abomasum palpable through abdominal wall or rectally	Alkalosis, hypochloraemia, hypokalaemia and dehydration	High case fatality rate. Fluids, laxatives. Slaughter for salvage may be indicated

Disease	Epidemiology and History	Clinical Findings	Clinical Pathology	Response to Treatment
Left-sided displacement of abomasum (LDA)	High-level grain diets, immediately post-partum dairy cows, inactivity	Acetonaemia in cow within days after parturition, inappetence, faeces soft and amount variable (usually reduced). Ketonuria. Rumen sounds present but faint. Pinging sound on percussion and auscultation of left side	Ketonuria. Paracentesis into displaced abomasum yields pH of 2 and no protozoa	Good response following surgical correction
Right-sided displacement of abomasum (RDA)	Usually 2-4 weeks post-partum	Anorexia, scant faeces, poor milk production, moderate dehydration, rumen sluggish, fluid-filled viscus under right costal arch, may be palpable per rectum, progressive and commonly results in torsion	Alkalosis, hypochlor-aemia, hypokalaemia	High case fatality rate. Fluid therapy. Slaughter for salvage
Torsion of abomasum	Sequel to right side displacement of abomasum (RDA)	History of RDA followed by sudden onset of acute abdominal pain, distension of right abdomen, loud "pinging" sound on percussion. Distended tense abomasum palpable per rectum, marked circulatory failure, weakness, blood-stained faeces, death in 48-60 hours	Dehydrating alkalosis, hypochlor-aemia	Laparotomy, abomasotomy and drainage. High case fatality rate
Primary acetonaemia (wasting form)	Overfat cows in late pregnancy or insufficient intake of energy in early lactation. May be high silage diet in heavy producing indoor cattle	Cow dull, off feed, small amounts firm balls of faeces, not pasty, lose condition milk yield down. Rumen activity depressed	Ketonuria and hypoglycaemia	Dextrose i.v. and propylene glycol orally, or corticosteroids i.m. Usually excellent response
Acute intestinal obstruction	May be heightened activity, e.g. during sexual activity. Often no particular history	Sudden onset short period acute pain. Kicking at belly, rolling. Brief period of diarrhoea with normal gut sounds initially, then all intestinal sounds cease including ruminal sounds. Dehydration commences. Distended loops of gut and grey to red foul-smelling rectal contents	Gradual development of dehydration and haemo-concentration over 3-4 days	Surgery is necessary
Abomasal ulcer	Immediately after (2 weeks) parturition. High producers on heavy grain feed. In intensive feeding systems disease is becoming enzootic some areas	Gastrointestinal atony with melaena. May be sufficient blood loss to kill. More likely prompt recovery after 4 days. Perforation of ulcer leads to death in a few hours	Blood in faeces. On perforation may be leucocytosis and left shift. Anaemia due to haemorrhage	Alkalinizing agents by mouth. Surgery if medical treatment unsuccessful
Pregnancy toxaemia of beef cattle	Fat beef cattle, deprived of feed in last month of pregnancy. Commonly have twin pregnancy	Complete anorexia, rumen stasis, scant faeces, ketonuria, weak and commonly recumbent	Ketonaemia, increase in non-esterified fatty acids, ketonuria, increase in liver enzymes	Poor response to therapy. Fluids, anabolic steroids, insulin
Fatty liver syndrome	Fat dairy cow, few days following parturition or may have had LDA for several days	Complete anorexia, rumen stasis, almost no milk yield, ketonuria initially but may have more later	Ketonaemia, increase in liver enzymes	Poor response to therapy. Glucose, insulin, anabolic steroids

Disease	Epidemiology and History	Clinical Findings	Clinical Pathology	Response to Treatment
Caecal dilatation and/or torsion	Single case. Dairy cow, early lactation, inappetence, faeces may be scant. Severe cases have history of mild abdominal pain.	Systemically normal. Rumen only slightly sluggish, tympanic sounds over right upper flank which may be distended. Rectally can feel enlarged cylindrical movable caecum with blind end	Nothing diagnostic, but has haemoconcentration, compensated hypochloraemia, hypokalaemia and alkalosis	Good response to surgical correction. Unfavourable prognosis with severe torsion and gangrene
Acute diffuse peritonitis	Following acute traumatic reticuloperitonitis, uterine rupture at parturition, rupture of rectum, post-surgical	Acute toxemia, fever followed by hypothermia, weakness, tachycardia, recumbency, groaning, moderate distension, scant faeces, palpate fibrinous adhesions rectally	Leucopenia, neutropenia, degenerative left shift. Haemoconcentration. Paracentesis positive	Usually die
Chronic ruminal tympany	Beef calves 6-8 mos. of age following weaning; feeder cattle after arrival in feedlot	Chronic free-gas bloat, relapses after treatment, no other clinical findings	Nil	Good response to surgical ruminal fistula or insertion of corkscrew-type trocar and cannula and leave in place for few weeks
Omasal impaction	Uncommon. Single cases in pregnant cows with vagus indigestion. Feedlot cattle with abomasal impaction dietary in origin	Inappetence to anorexia. Scant faeces, abdominal distension. Rectally feel large distended round hard viscus below kidney	Nil	Slaughter for salvage. Treat as for abomasal impaction

THE EFFECTS OF SOME COMMON CLINICAL EXCITATORY AND INHIBITORY INFLUENCES ON PRIMARY CYCLE MOVEMENT OF THE RETICULO-RUMEN

Clinical Afferent Input	Clinical Findings and Responses to treatment
EXCITATORY INPUTS	
Low threshold reticular tension receptors Increased reticular tension After feeding Mild ruminal tympany	Increases frequency, duration and amplitude of primary cycle contractions and mixing promotes fermentation.
Decreased reticular tension Starvation Anorexia	Decreases frequency, duration and amplitude of primary cycle contractions and decreases fermentation
Lesions of medial wall of reticulum Chronic induration and fibrosis due to traumatic reticuloperitonitis	Causes hypomotility of rumen contractions and may be explanation for atony in some cases of vagus indigestion. Some cases are characterized by erratic hypermotility.
Acid receptors in abomasum Increases in abomasal acidity following emptying of organ	Increases primary cycle movements which increases flow of ruminal contents into abomasum to maintain optimum volume and to decrease acidity
Buccal cavity receptors Following eating	Increased reticulo-rumen activity

Clinical Afferent Input	Clinical Findings and Responses to Treatment
INHIBITORY INPUTS	
High Threshold reticular tension receptors Peak of reticular contraction Severe ruminal tympany Ruminal impaction with forage, hay, straw (not necessarily grain overload)	Depression of primary cycle movements, ruminal hypomotility, depression of fermentation because of failure of mixing
Abomasal tension receptors Impaction, distension or displacement of abomasum	Abomasal impaction, dilatation and torsion may result in complete ruminal stasis. Left-sided displacement of abomasum usually does not cause clinically significant hypomotility
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Pain Visceral pain due to distension of abomasum or intestines. Severe pain from anywhere in body	Moderate to total inhibition of reticulo-rumen move- ments possible with visceral pain. The degree of inhibition from pain elsewhere will vary
Depressant drugs Anaesthetics, central nervous system depressants Prostaglandin E	Inhibition of primary and secondary cycle movements, and eructation resulting in ruminal tympany
Changes in rumen contents Marked decrease (below 5) or increase (above 8) in pH of ruminal fluid. Engorge- ment with carbohydrates or protein-rich feeds. Absence of protozoa in ruminal acidosis and in lead and other chemical poisoning	Inhibition of primary and secondary cycle movements and lack of fermentation. Rumen transplant promotes return to normal activity
Changes in body water, electrolytes and acid- base balance Hypocalcaemia	Inhibition of primary and secondary cycle movements and of eructation resulting in ruminal tympany which responds to treatment with calcium
Dehydration and electrolyte losses, acidosis, alkalosis	Inhibition of reticulo-rumen movements which gradually return to normal with fluid and electrolyte therapy
Peritonitis Traumatic reticuloperitonitis	Inhibition of primary and secondary cycle movements and of eructation resulting in ruminal tympany. Return of primary movements is good prognostic sign. Lesions must heal without involvement of nerve receptors or adhesions which will interfere with normal motility
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Toxaemia/fever Peracute coliform mastitis	Inhibition of primary and secondary cycle movements which return to normal with treatment of toxaemia
Ruminal distension Early ruminal tympany	Increased frequency of secondary cycle movements and of eructation
Covering of cardia (fluid or foam) Ruminal tympany Recumbent animal	Cardia does not open, failure of eructation resulting in ruminal tympany. Clearance of cardia results in eructation

Most of the sensory inputs are transmitted to gastric centres in the dorsal vagal nerve nuclei from which the efferent outputs originate and pass down the vagal motor nerve fibres. Modified according to Leek (1).

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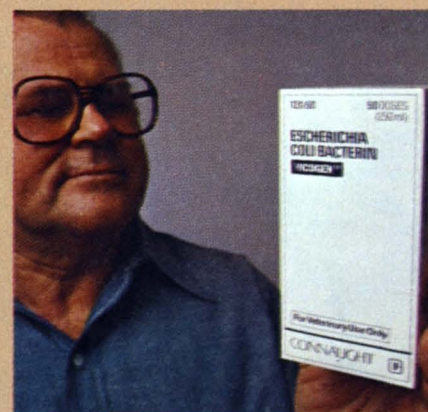
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Clinical Examination of the Digestive Tract and Abdomen of Cattle

1. *History*

Obtain a complete detailed history.

a) *Time of Occurrence*

- Pregnant and dry.
- Recent parturition.
- Season of year.

b) *Nature of Diet*

- Composition.
- Chopped or long hay or straw.

c) *Speed of Onset*

d) *Appetite*

e) *Feces*

- Consistency
- Amount-Large volume diarrhea
- Small volume diarrhea
- Color
- Odor

f) *Previous Treatment*

- What was used?
- Any response.

2. *Systemic State and Habitus*

- Standing, ability to walk normally.
- Recumbent and unable to rise.
- State of collapse.
- Grunting.
- State of hydration (degree of dehydration).
- Temperature, pulse and respirations.

3. *Visual Adspection of Abdomen*

Examine the contour of the abdomen as viewed from the rear of the animal. See Figures a to j for abdominal contours associated with various abnormal states of the abdomen.

4. *Close Examination of the Oral Cavity*

Use a Drinkwater gag to properly explore the oral cavity. Examine the mucous membranes, dorsum of the tongue, teeth and pharynx.

5. *Close Examination of the Left Abdomen and Rumen*

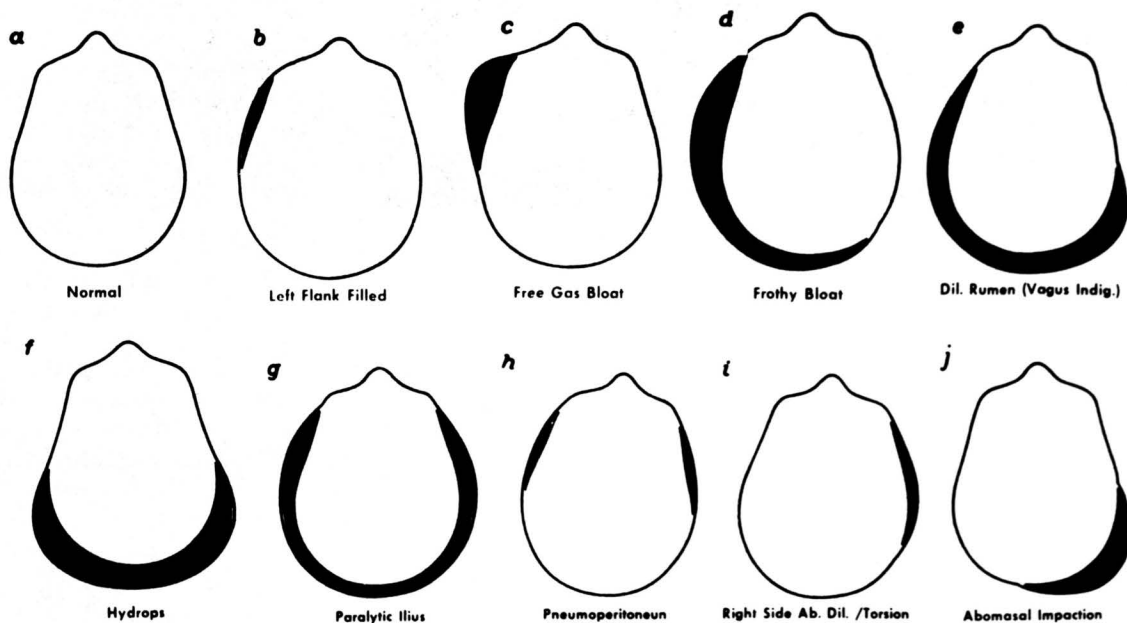
a) *Visual adspection and palpation of the left flank and paralumbar fossa.* Can you see the rumen in the left paralumbar fossa? Is the rumen moving?

Observe the movement of the rumen in the left flank first, and follow this by holding the palm of the hand against the rumen to determine the "lift" which occurs during rumen contraction. In vagus indigestion, there may be three to seven vigorous incomplete contractions of the rumen per minute. These contractions may not be audible because the rumen contents are porridge-like and do not cause the normal rumen-roughage sounds. However, the contractions are visible and palpable as waves of undulations of the left flank.

Can you palpate the rumen through abdominal wall? Does the ruminal ingesta feel normal (doughy), firm as if excessively dry or is the rumen not palpable because it may be displaced medially, or is it smaller than normal?

b) *Auscultation of the Rumen and Left Flank.*

In the normal animal on a roughage diet there are two independent contraction sequences of the reticulo-rumen. The *primary cycle*, which recurs approximately every minute, consists of a diphasic contraction of the reticulum followed by a monophasic contraction of the dorsal ruminal sac and then by a



monophasic contraction of the ventral ruminal sac. These movements appear to be concerned primarily with "mixing" the rumen and contents and with assisting the passage of rumen contents into the omasum. The *secondary cycle* movements, which occur at intervals of about two minutes, are confined to the rumen and consist of a contraction of the dorsal sac followed by a contraction of the ventral sac. The former causes the fluid contents of the dorsal sac to be forced ventrally and the gas layer to be forced cranially to the region of the cardia where eructation takes place. Contractions of the dorsal and ventral sacs cause undulations of the left paralumbar fossa and lower flanks which are readily visible and palpable.

In auscultation of the rumen, the stethoscope head is placed in the middle of the left paralumbar fossa area. After sufficient contractions have occurred, the stethoscope should then be moved anteriorly to determine if rumen contractions can be heard in the region that becomes occupied with a left displacement of the abomasum. In the normal animal, rumen movement will be heard in this area which is in the second last intercostal space. The type, strength and frequency of rumen movements should be noted. The rumen sounds of the normal animal consuming roughage are booming-crackling sounds. When the rumen contains less coarse roughage or primarily grain, the sounds may be much less distinct but still possess a crackling characteristic.

The presence of fluid-tinkling or fluid-splashing sounds, usually along with a static rumen, suggests that the rumen contains an excessive quantity of fluid, and that the coarse ingesta is not floating on the fluid layer of the rumen contents as in the normal animal. Fluid-splashing sounds suggest diseases such as grain overload, and the atonic rumen associated with prolonged anorexia (chronic diffuse peritonitis, abomasal or omasal impaction.) The sounds are similar to those present in left sided displacement of the abomasum. To assist in the differential, the outline of the rumen can be percussed to observe a much wider area of metallic sound than one would normally expect in displaced abomasum. Also, by tapping the transverse processes on the vertebrae on both the right and left sides, the high-pitched metallic sounds are audible on auscultation over the left paralumbar fossa.

In left sided displacement of the abomasum, the metallic "ping" is usually not producible over a large area as in the atonic rumen.

Rumen sounds are most frequent and strongest immediately after roughage feeding and rumination.

In *vagus indigestion* of cattle, the contractions of the rumen will usually be very obvious and frequent (3 to 6/minute). The contractions are visible as prominent undulations of the wall of the left flank which are visible and palpable but may not be audible because

the rumen contents are homogeneous and porridge-like due to prolonged maceration in the rumen.

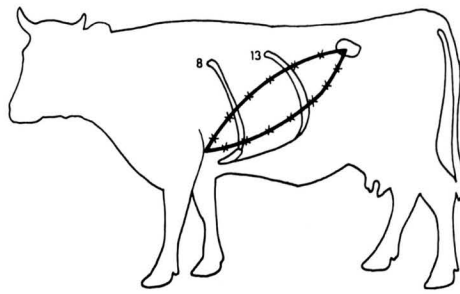
Causes of "Pings" in the Abdomen in Cattle

Left Side	Right Side
Left side displacement of the abomasum.	Right sided dilatation and torsion of abomasum.
Atonic Rumen containing fluid and varying amounts of gas (grain overload, chronic peritonitis)	Cecal dilatation and torsion, Torsion of mesentery Torsion of spiral colon.
	Intestinal tympany Colonic and rectal tympany Post-parturient cow Intussusception

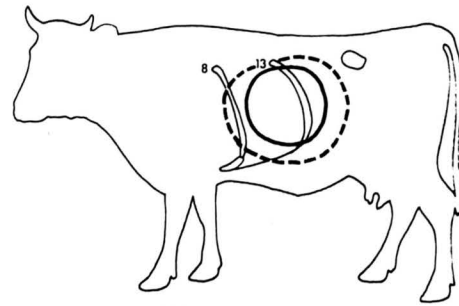
Auscultation and percussion of the left paralumbar fossa are over an area extending from mid-way of the 9th rib to the 13th rib is used to detect the presence of a "ping" associated with left sided displacement of the abomasum. In *some* cases, the distended abomasum can be palpated as a soft swelling in the anterior aspect of the left paralumbar fossa. Percussion can be accomplished with a flick of the finger or ideally with a percussion hammer.

c) Examination of Rumen Contents.

Examination of the rumen contents is often essential to establish an accurate diagnosis of diseases of the rumen. The color, depending on the feed to a limited extent, will be a green, olive green, or brown green. At pasture, the color of course is very green, with root crops the color tends to be grey, and with silage or straw the color is mostly of a yellow brown nature. The color of rumen content will be a milky grey in grain overload, and greenish black in cases where rumen stasis is of long duration and where putrefaction is occurring within the rumen. The consistency of the rumen content is normally slightly viscid, and a watery rumen content is indicative of inactive bacteria and protozoa. Excess froth is associated with frothy bloat. The odor of the rumen content is normally aromatic and although somewhat pungent, not objectionable to the nose. A mouldy, rotting odor usually indicates protein putrefaction, and an intensely sour odor indicates an excess of lactic acid formation, due to grain or carbohydrate engorgement. The pH of the rumen varies according to the type of feed and the time interval between the last feeding and taking a sample for pH examination. The normal range, however, is between 6.2 and 7.2. Consequently the pH of rumen content should be examined immediately after the sample is obtained, using a wide range pH paper. High pH values will be observed in cases where putrefaction of protein is occurring in the rumen and when the saliva content is high. Low values of pH are found after

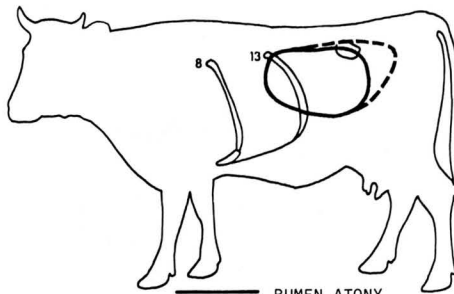


AUSCULTATION SITES

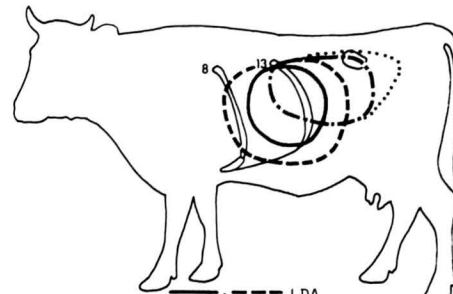


LDA

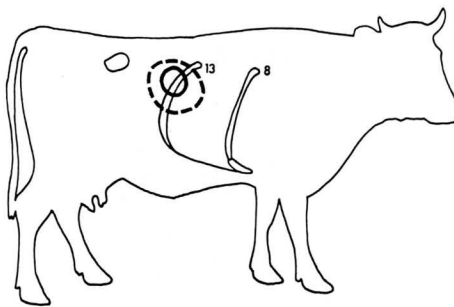
Left-sided Gas-filled Viscus (Pings)



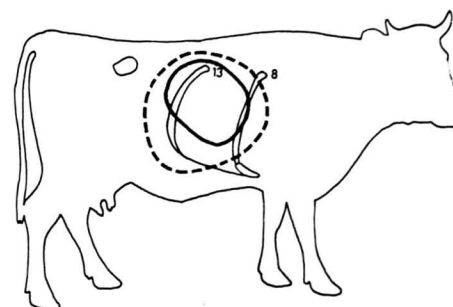
— RUMEN ATONY
- - - PNEUMOPERITONEUM



— LDA
- - - RUMEN ATONY
..... PNEUMOPERITONEUM

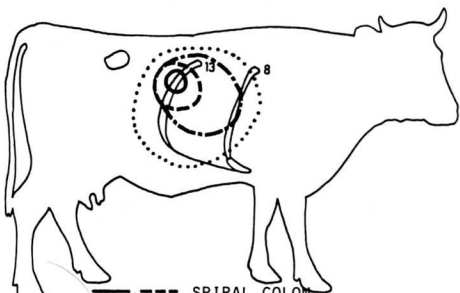


SPIRAL COLON

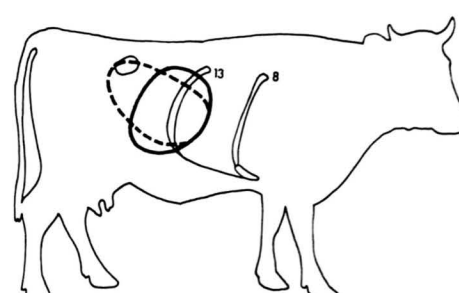


— RDA
- - - RDA VOLVULUS

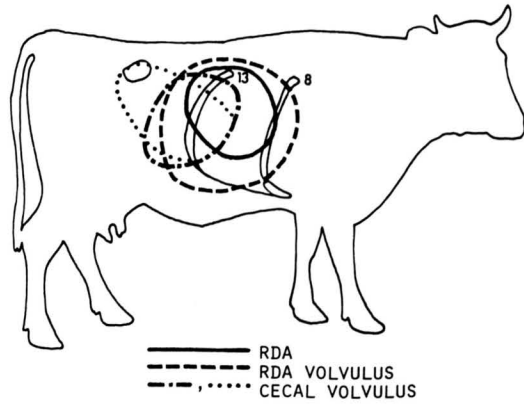
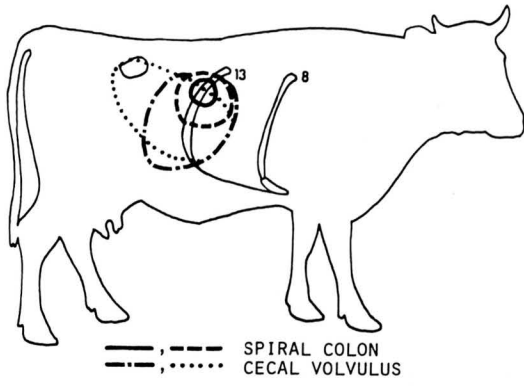
Right-sided Gas-filled Viscus (Pings)



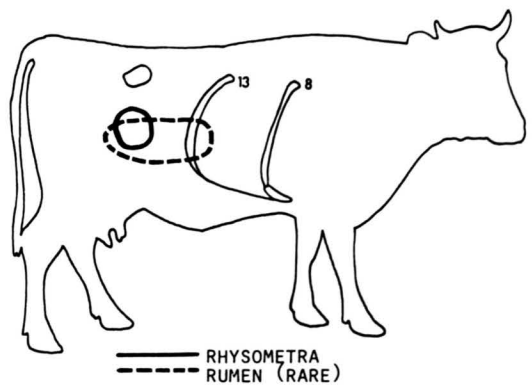
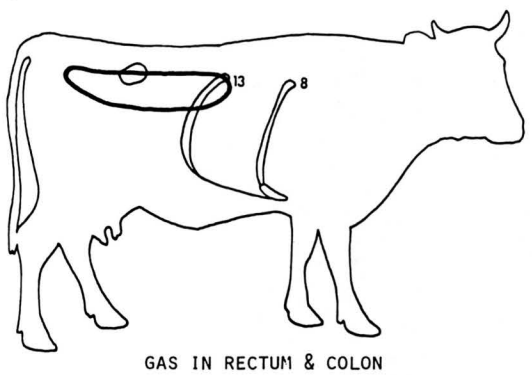
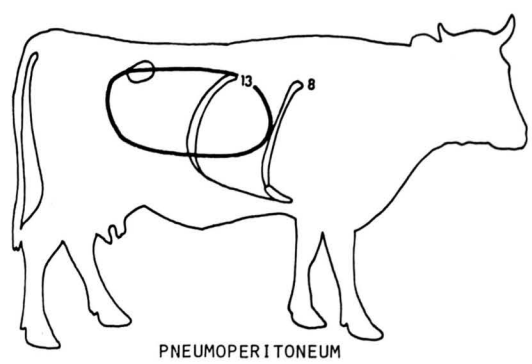
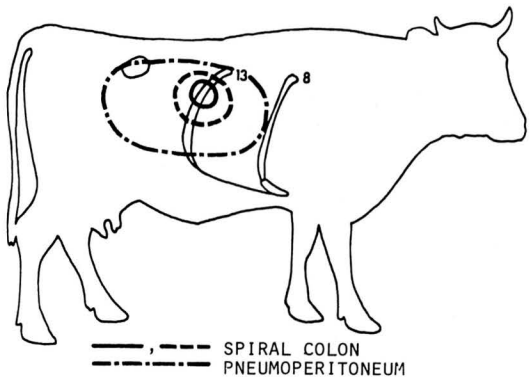
— SPIRAL COLON
- - - RDA
..... RDA VOLVULUS



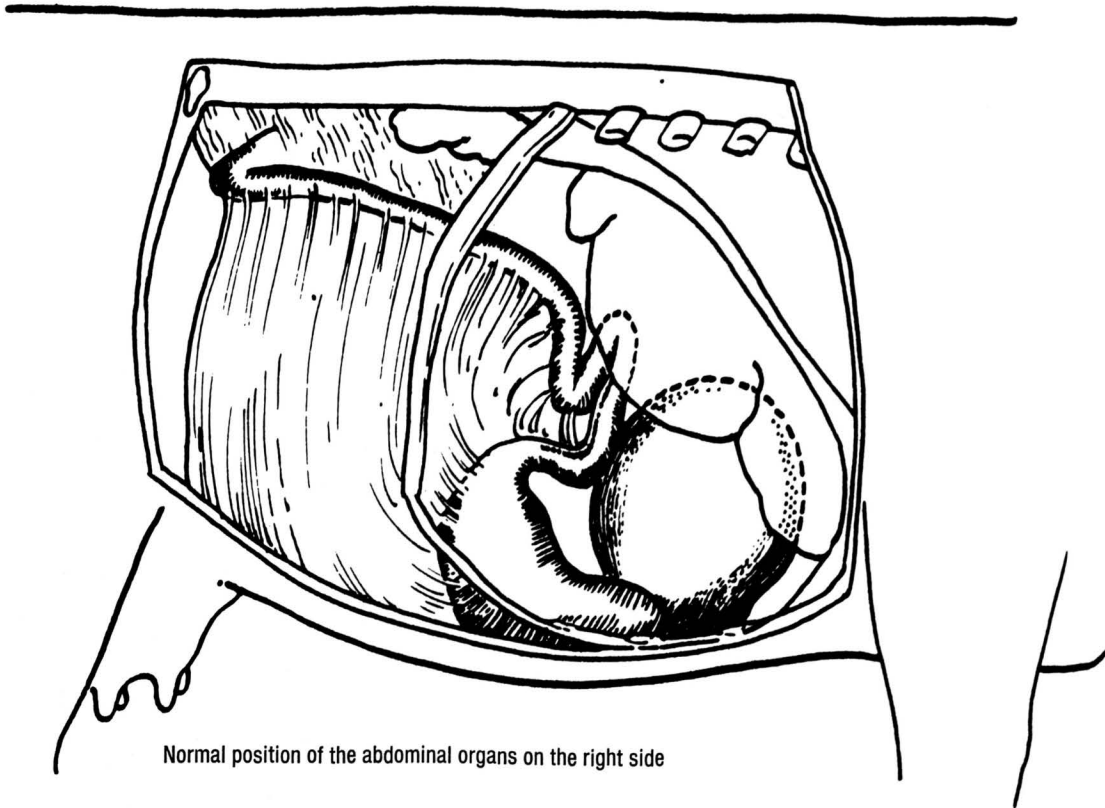
CECAL VOLVULUS



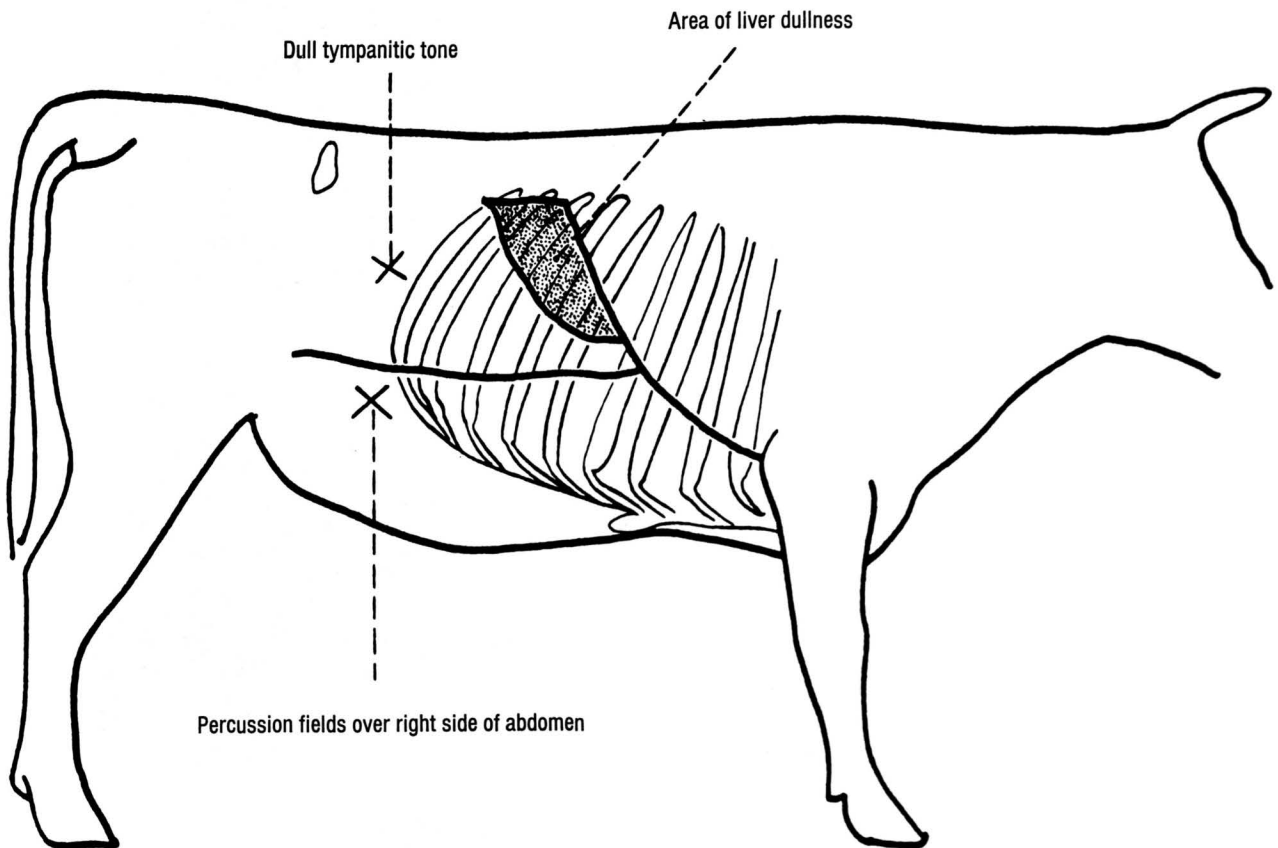
Right-sided Gas-filled Viscus (Pings)



Right-sided Gas-filled Viscus (Pings)



Normal position of the abdominal organs on the right side



Percussion fields over right side of abdomen

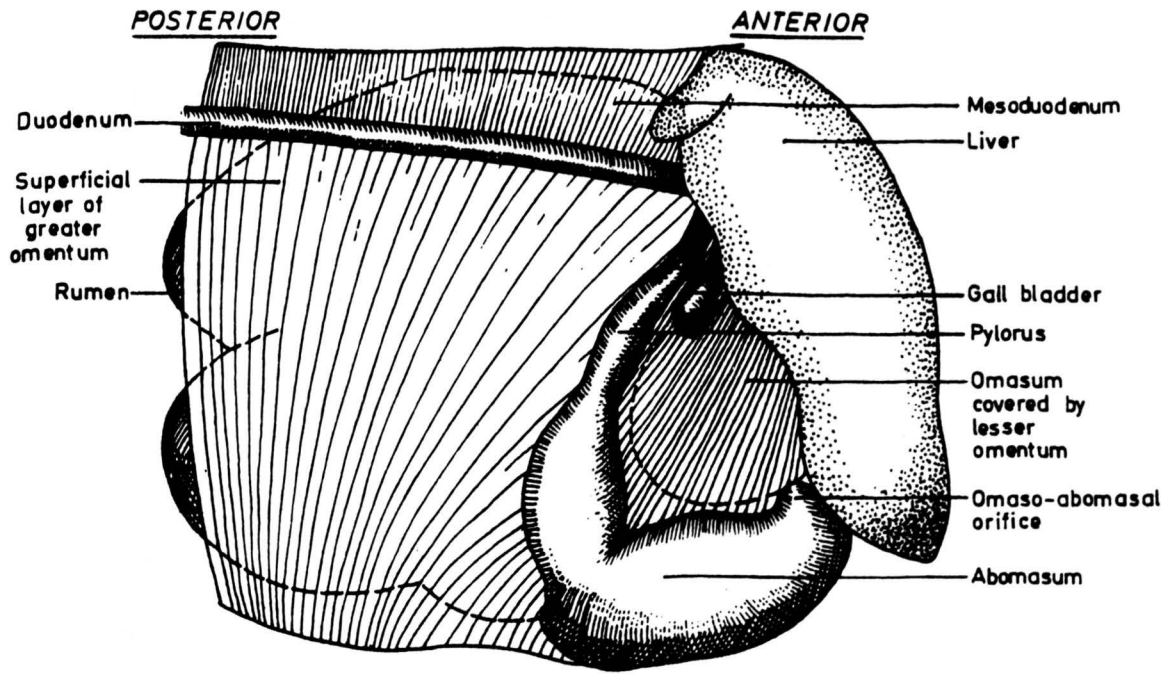


FIG. 1 - The normal disposition of the abomasum and omasum viewed from the right.

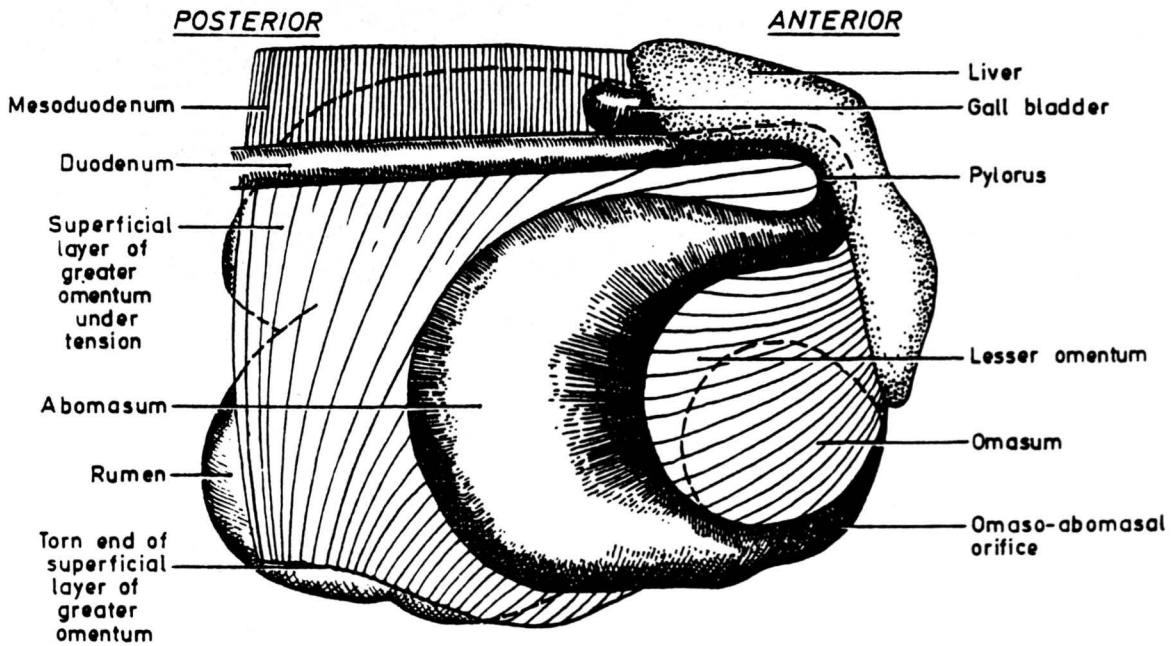


FIG. 2 - Clockwise torsion of the abomasum viewed from the right.



Now, 6 antigens in one vaccine

help protect size of calf crop... 6 ways.

Boosts breeding efficiency. Now just one dose, one vaccine, one time through the chute! You shield your client's herd against the 6 major causes of infertility, delayed breeding, abortion and strung-out calf crops. New Tri-Vib® GHP can protect against all 3 cattle serotypes of *Campylobacter fetus*, which cause vibriosis—plus 3 serotypes of *Leptospira: grippotyphosa, hardjo, and pomona*. One dose, one vaccine—all with time and labor-saving ease.

Special proven adjuvant. Antigenic response of Tri-Vib GHP is greatly enhanced by its special adjuvant, which has been proven on millions of cattle. High SN titers and challenge studies have demonstrated the high immunogenicity and efficacy of all 6 antigens—both singly and combined.

Wide margin of safety. All 6 antigens are *killed*. And tested for purity and safety. The entire herd may be

vaccinated at one time, with no increased risk to pregnant or lactating cows. And you can vaccinate at any time to suit you or the owner.

Broad-spectrum protection. It's difficult to predict which of 3 serotypes of *Vibrio* or 3 serotypes of *Lepto* a herd may encounter. All are widespread. Protect your client and yourself with broad-coverage, *killed*, specially-adjuvanted Tri-Vib GHP.

Tri-Vib® GHP

Campylobacter fetus—Leptospira grippotyphosa-hardjo-pomona bacterin



Fort Dodge Laboratories, Fort Dodge, Iowa 50501

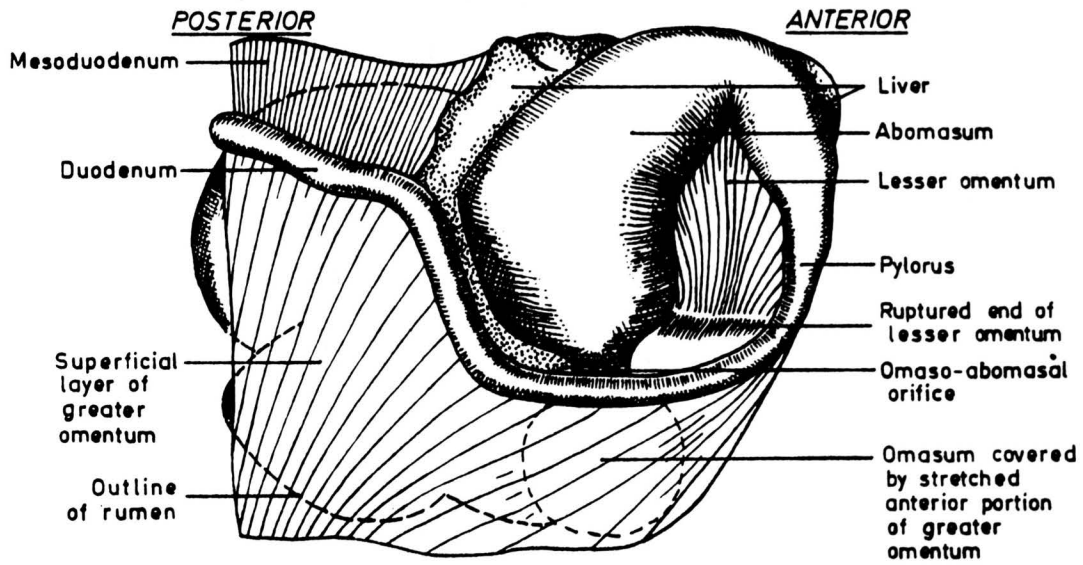
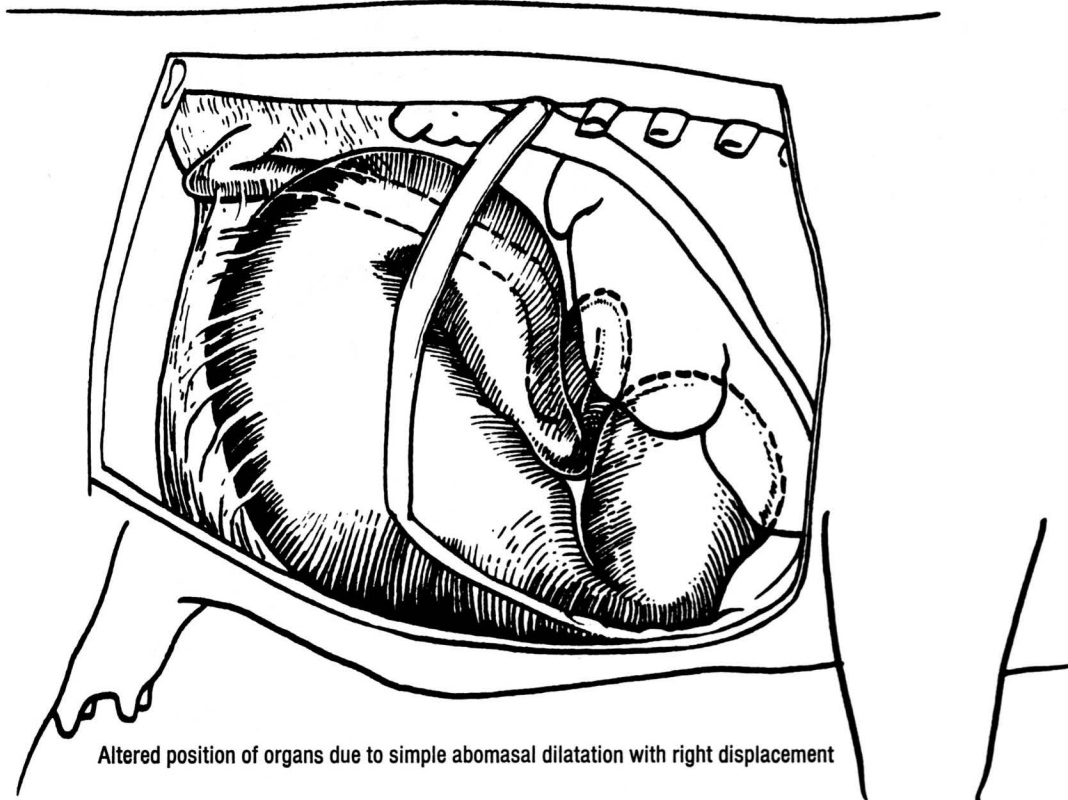
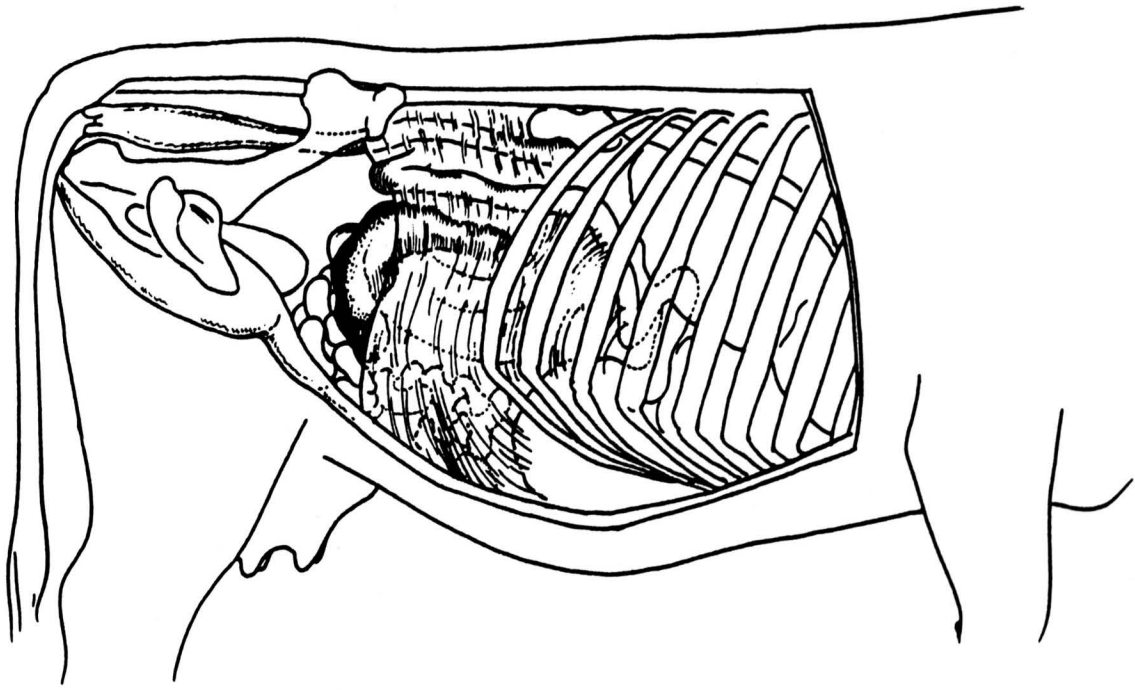
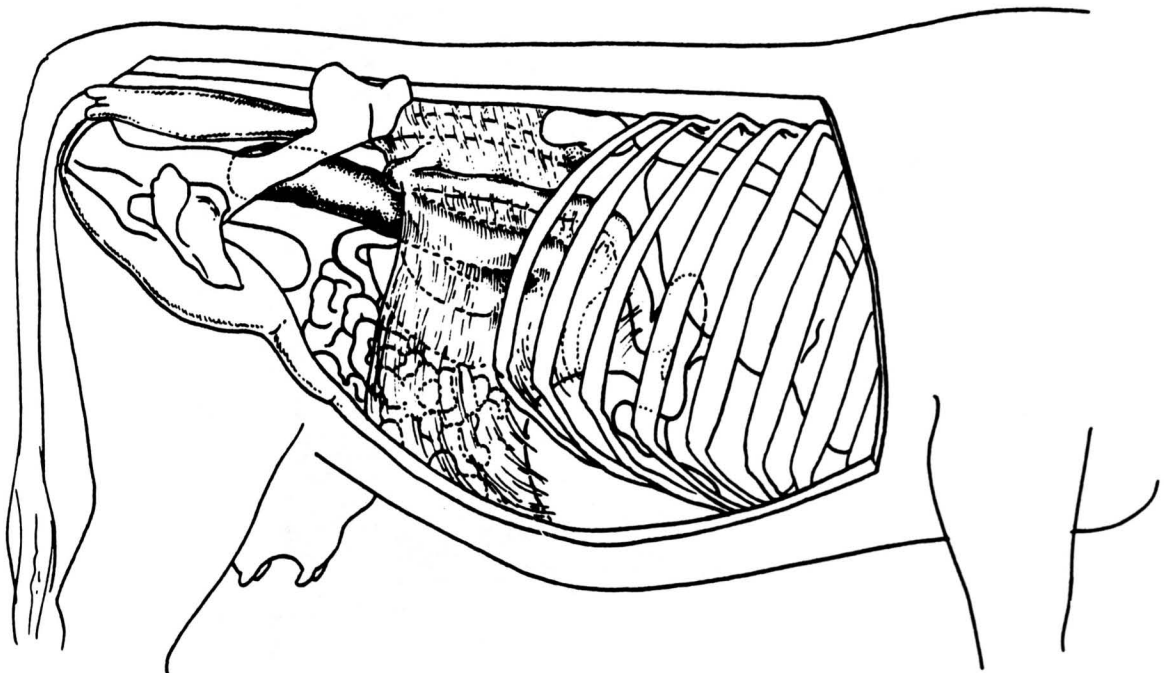


Figure 3. Anticlockwise torsion of the abomasum viewed from the right.

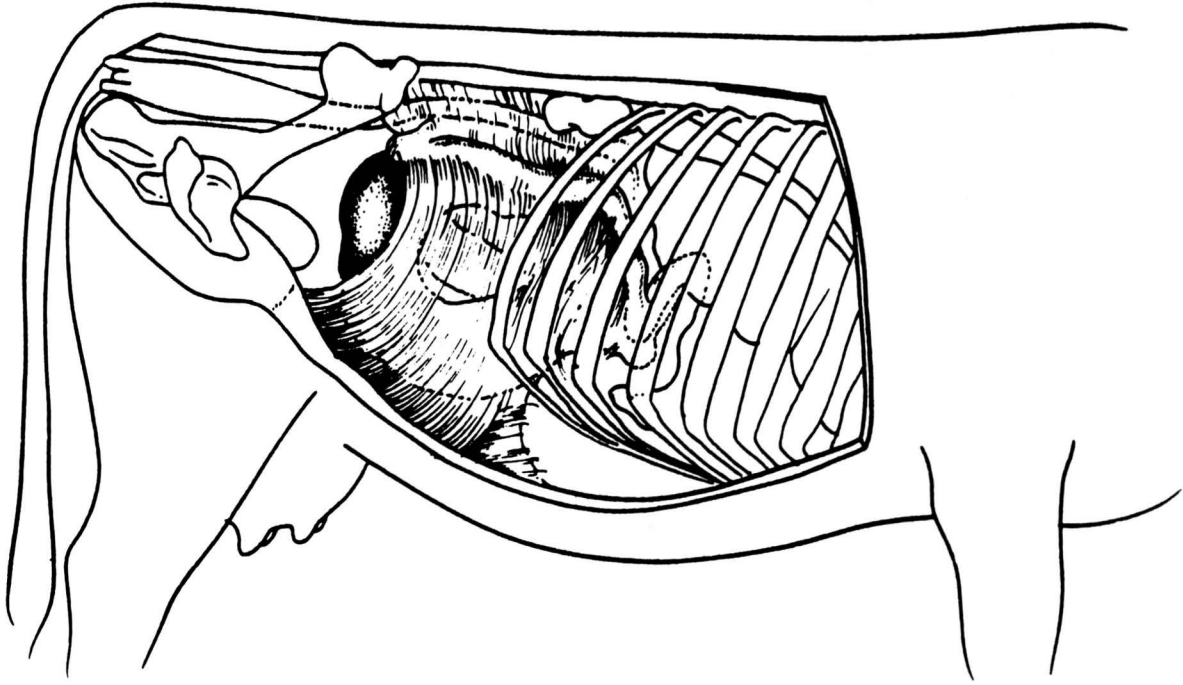




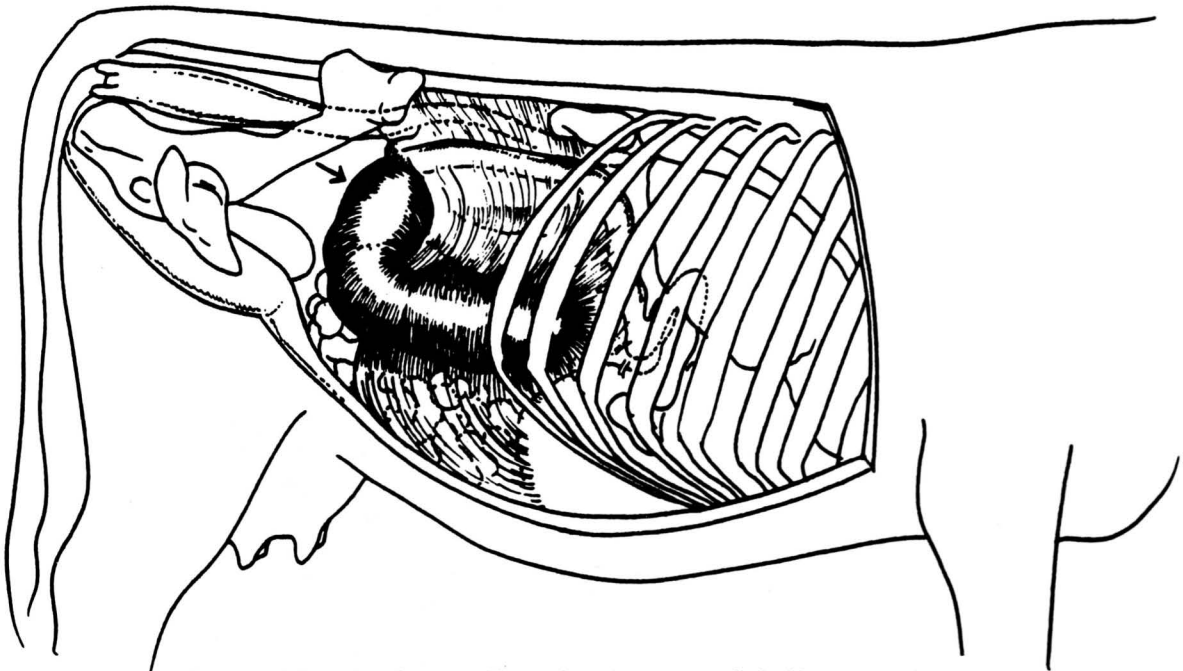
Normal position of cecum with apex buried in small intestines



Normal position of cecum projecting into pelvic cavity



Torsion and distention of cecum with apex buried in small intestine (palpable per rectum)



Torsion and distention of cecum with apex lateral to omentum (palpable per rectum)

the feeding of carbohydrates. In general, a value below 5 is considered very suggestive of carbohydrate engorgement and this pH level will be maintained for a matter of between 6 and 24 hours after the animal has actually consumed the carbohydrate ration. Examining the rumen content for floatation and sedimentation is perhaps one of the simplest and most revealing examinations that one can perform. Normally the finer particles will sink to the bottom of a test tube containing rumen content in a few minutes. The large fibrous particles are held in the top layer of the rumen content amidst fermentation gas bubbles. In normal rumen contents, the time lapse between filling a test tube and sedimentation time is about 5-10 minutes. In an active rumen sample, the small particles that first sink will later rise to the surface. In abnormal rumen samples, on the other hand, the sedimentation rate will be extremely fast with a delayed or absent floatation observed, due to complete absence of a secondary floatation period. Protozoa content of the rumen liquor contains about 30 types of protozoa. These organisms are obligatory anaerobic and apparently not important for the life of the animal or for the fore stomach development in the calf. The protozoa do, however, serve as a simple indicator of sudden disturbances in rumen fermentation, since the rapid death of many protozoans indicates that the substrate is unsuitable for their development or survival. The bacterial content of rumen liquor ranges to approximately ten million per cc of rumen content. In grain overload or carbohydrate engorgement, there will be a shift from the usual balance between gram positive and gram negative bacteria to a sample where gram positive cocci predominate.

6. Close Examination of the Right Abdomen

The contour of the right flank should be examined by *visual adspersion* for evidence of distention which may be due to a gas-filled viscus, impacted abomasum, ascites or a gravid uterus. In severe distention of the rumen, the ventral sac may also distend the lower half of the flank.

A combination of deep palpation, ballottement (deep bouncing palpation), percussion and auscultation, and succession (shaking the animal) are used to detect the presence of viscuses which are distended with gas and/or fluid, or ingesta.

Gas filled viscuses which result in a "ping" in the inner right flank include:

- Right sided dilatation and torsion of the abomasum
- Cecal dilatation and torsion
- Torsion of the coiled colon
- Descending colon and rectum filled with gas in a cow with tenesmus.

- Intestinal tympany of uncertain etiology
- Torsion of the root of the mesentery in young calves
- Intussusception

Fluid-splashing sounds may be audible on deep ballottement or succussion for the following reasons:

- Fluid-filled intestines in enteritis
- Fluid-filled abomasum in abomasal ulceration.

Auscultation of the right flank may reveal fluid-rushing sounds for the following reasons:

- Early enteritis
- Early acute intestinal obstruction

Palpation of a heavy viscus in the right flank caudal or ventral to right costal arch may be due to:

- Omasal impaction
- Abomasal impaction
- L-shaped rumen with the ventral sac moved to the right
- Enlargement of the liver

In summary, clinical examination of the right flank is done to detect the presence or absence of viscuses which are distended with gas, fluid or ingesta. Gas-filled viscuses are usually situated in the upper half of the right flank and impacted viscuses are usually in the lower half of the flank. A rectal examination will often assist in the identification of the distended organ. A laparotomy may be necessary to accurately identify the organ, the extent of the distention or displacement are the viability of its tissue and circulation. It is not possible on rectal palpation to determine the severity of the ischemic necrosis which may be present, which is important for the prognosis.

7. Rectal Exploration of the Abdomen

Rectal examination of the abdomen of cattle (of a sufficient size to allow a rectal examination) is a very useful aid in the diagnosis of diseases of the digestive tract. The abnormalities detectable include:

- The nature and amount of feces in the rectum and colon.
- Distention of viscuses with gas, fluid and ingesta.
- Displacement of viscuses.
- Tension of mesenteric bands.
- Peritoneal adhesion.

The specific abnormalities of the digestive tract, which are commonly palpable on rectal examinations, include the following: (See Figures a to l which illustrate the abnormalities through a transverse section of the abdomen.)

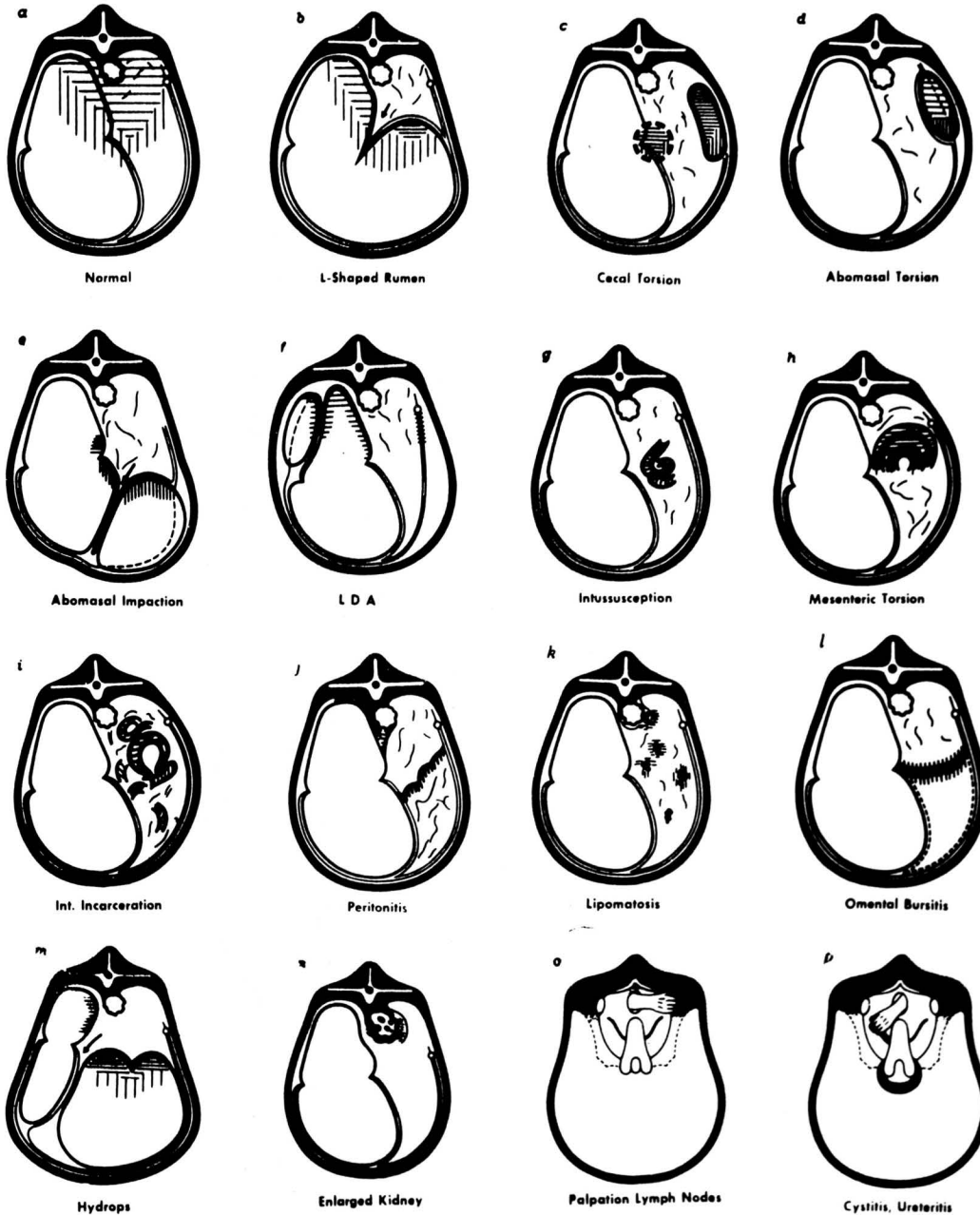
- a) Normal
- b) L-shaped rumen. (occurs commonly in vagus

indigestion and other diseases of the rumen characterized by gradual distention of the rumen.)

- c) Cecal torsion. (commonly palpable as long distended organ usually movable may feel blind end.)
- d) Abomasal torsion. (commonly by palpable as tense viscus in lower right half of abdomen.)
- e) Abomasal impaction. (not palpable in late pregnancy.)
- f) Left-sided displacement of the abomasum. (usually cannot palpate the displaced abomasum but can

often feel rumen which is usually smaller than normal.)

- g) Intussusception (not always palpable, dependent on location of intussusception and the size of the animal.)
- h) Mesenteric torsion-usually palpable.
- i) Intestinal incarceration-commonly palpable.
- j) Peritonitis-only palpable if peritoneum of posterior aspect of abdomen affected.
- k) Lipomatosis-commonly palpable as "lumps" in the abdomen and pelvic cavity.
- l) Omental bursitis - not common.



As part of the differential diagnosis of digestive tract disease in the post-parturient cow, the uterus should be examined carefully for evidence of retained placenta and metritis. The toxemia caused by retained fetal membranes and post-partum metritis may cause anorexia, rumen stasis, paralytic ileus, scant feces and sometimes a “ping” in the right flank all of which may be interpreted as primary digestive tract diseases.

8. *Gross Examination of Bovine Feces*

The gross appearance of bovine feces are not only indicators of diseases of the digestive tract itself, but can provide valuable clues for the differential diagnosis of diseases elsewhere.

a) *Amount of Feces*

In adult cattle, the passage of ingesta through the digestive tract takes 1½ to 4 days. Mature cattle generally pass some feces every 1.5-2 hours, amounting to a total of 30 to 50 kg per day in 10 to 24 portions. A reduction in the bulk of feces can be due to:

- i) interference with the feed or water intake.
- ii) a retardation of the passage through the alimentary tract.
- iii) increased resorption of fluids in the large intestine.

To determine which factor is responsible, it is necessary to examine the feed and water intake, prehension, mastication, swallowing, the forestomach, and abomasum, and the intestines. This often necessitates a rectal examination of the palpable abdominal viscera and in some cases, an exploratory laparotomy.

An apparent increase in the amount of normal feces in the rectum is usually associated with paralysis of the rectum.

In diarrhea, the feces are passed more frequently than normal and contain a higher water content (over 90%) than normal.

Failure to defecate for 24 hours or more is abnormal and the continued absence of feces leads to the well recognized and usually fatal syndrome of intestinal obstruction. Paradoxically, in many cases the intestine is not obstructed and clinical signs are due to paralytic ileus and associated intestinal tympany. The term paralytic ileus is used to describe intestinal distension from loss of the muscular action of the intestine in the absence of any direct mechanical occlusion. Diseases which can result in paralytic ileus include:

Diffuse peritonitis

Intestinal tympany (idiopathic ileus, thought to be dietary in origin - increase in concentration of volatile fatty acids in concentrate-fed cows).

b) *Color of the Feces*

The color of the feces is influenced by:

- i) the nature of the feed.

- ii) the concentration of bile in the feces.
- iii) the passage rate through the digestive tract.

Calves reared on cow's milk normally produce gold-yellow feces which become pale brown when hay or straw is eaten. The feeding of milk substitutes adds a grey component to a varying degree.

The feces of adult cattle on green forage are dark olive green, on a hay ration, more brown-olive, while the ingestion of large amounts of grain produces grey-olive feces. A retardation of the ingesta causes the color to darken. The feces become ball-shaped and dark brown with a shiny surface due to the coating with mucus. Diarrheic feces tend to be paler than normal because of their higher water content and lower concentration of bile.

The presence of large amounts of bile produces a dark olive-green to black-green color such as in cattle with hemolytic anemia. In cattle with obstruction of the common bile duct, the feces are pale-olive because of the absence of bile pigments.

Hemorrhages into the digestive tract depending on the amount, location and rate of passage will result in feces which vary from chocolate-brown to blackish-tarry (melena). Hemorrhage in the abomasum and small intestine usually causes black tarry feces. Hemorrhage in the rectum usually causes a red-brown discoloration distributed throughout the feces or red streaks of frank blood adhering to the fecal particles.

c) *Odor of the Feces*

Fresh bovine feces are not normally very malodorous. Objectionable odors are usually due to putrefaction or fermentation of ingesta which are usually associated with inflammation. The feces in cattle with salmonellosis may be fetid while in advanced pericarditis with visceral edema due to passive congestion the feces are profuse but do not possess a grossly abnormal odor.

d) *Consistency of Feces*

The consistency of the feces is dependent on the water content. The type of feed and the length of time the ingesta has remained in the digestive tract normally excrete feces of a medium to firm porridge-like consistency. After transition to a plant diet, the first solid particles begin to appear. Normal bovine feces are of a medium porridge-like consistency. A moderate thickening leads to the passage of fecal discs of a more solid consistency and severe dehydration causes the formation of firm balls of feces arranged in facets inside the rectum, the surfaces of which are dark and coated with mucus. The feces of cows with left sided displacement of the abomasum commonly are pasty in appearance. Sticky and tenacious feces are commonly seen in obstruction of the forestomachs (vagus indigestion, chronic peritonitis).

e) *Degree of Comminution of Feces*

The proportion of insufficiently comminuted (poorly digested) plant particles in the feces is dependent on the duration and thoroughness of rumination and the rate of passage of ingesta through the forestomachs. The length of time the ingesta is in the post-ruminal digestive tract seems to have no appreciable influence on its trituration. Poor comminution of feces indicates failure in rumination and/or accelerated passage of ingesta through the forestomachs. Thus in some cattle with acute traumatic reticuloperitonitis, the feces may contain small walnut sized chunks of undigested plant fibers which presumably have escaped the cellulose digestive processes of the forestomachs. The presence of large numbers of kernels of grain in the feces is associated with the ingestion of large quantities of unprocessed grain like whole wheat or barley.

f) *Other Substances in the Feces*

Mucus. The presence of mucus on the surface of fecal balls suggests a prolonged sojourn of the ingesta in the large intestine. The presence of a plug of mucus in the rectum is suggestive of a functional obstruction (paralyticileus).

Fibrin. In enteritis, large quantities of clear watery mucus may be passed which sometimes clot to form gelatinous masses.

In fibrinous enteritis, fibrin may be excreted in the form of long strands which may mold into print of the intestinal lumen (*intestinal fibrinous casts*).

Blood. Blood in the feces may originate from the following locations:

- i) The swallowing of blood coughed up from pulmonary hemorrhage (not common). Usually detected as *occult blood* with *Hemetest tablets*.
- ii) Hemorrhage into the abomasum. Acute hemorrhage usually appears as black-tarry beces (melena). Chronic hemorrhage as occult blood.
- iii) Hemorrhagic enteritis of small intestines. The feces are uniformly dark red.
- iv) Hemorrhagic enteritis of the large intestines. Blood originating in the cecum or colon appears as frank blood evenly distributed throughout the feces. Blood originating in the rectum appears as streaks or chunks of frank blood unevenly distributed throughout the feces.

9. *Detection of Abdominal Pain*

Cattle with acute local or diffuse peritonitis may *grunt* with almost every expiration. The grunt is exaggerated in the recumbent position. However, grunting may also be caused by severe pneumonia, pleurisy and severe pulmonary emphysema. Careful auscultation and percussion of the lungs is therefore necessary to exclude the presence of pulmonary disease.

Not all *grunts* occur spontaneously. Deep palpation of the abdomen using the closed hand or knee is often necessary to elicit a grunt. Auscultation over the trachea is often necessary to hear the grunt. The grunt is best produced if pressure is applied to the abdomen at the end of inspiration and the beginning of expiration. The inspiratory and expiratory tracheal sounds are auscultated for 6 to 8 respirations and then without any particular warning the pressure is applied to the abdomen. A grunt cannot be elicited in a normal animal. The presence of a grunt means the presence of a peritoneal lesion (stretching, inflammation, edema). The absence of a grunt does not exclude the presence of a peritoneal lesion. In acute traumatic reticuloperitonitis the grunt may be absent in 3 to 5 days after the initial penetration of the reticulum.

A rigid bar or wooden pole may be necessary to apply pressure in large cattle (large cows and bulls). The bar is held by two people in a horizontal position just behind the xiphoid sternum while a third person auscultates over the trachea when the bar is lifted quickly up into the abdomen.

Several attempts should be made to elicit a grunt before concluding the absence of a grunt.

Pinching of the withers is also used to elicit a grunt. In the average sized cow, pinching of the withers causes the animal to depress it's back. In an animal with an inflammatory lesion of the peritoneum, depression of its back will commonly result in a grunt which may be audible without auscultation over the trachea but auscultation will usually be necessary.

10. *Clinical Examination of the Digestive Tract and Abdomen of the Calf.*

The clinical examination of the digestive tract and abdomen of the calf may be more difficult than in the adult animal. The rumen in the pre-ruminant calf is not yet functional and thus, cannot be used as an indicator of the state of the alimentary tract as is possible in the adult animal. Also, a rectal examination of the abdomen is not usually possible until the animal is about ten to 12 months of age, depending on the breed. A digital examination of the rectum of young calves is useful, however, to determine the presence or absence of feces, which may provide an indication of the presence of diarrhea which may not yet have begun. A complete absence of feces may suggest the presence of an acute intestinal obstruction, acute diffuse peritonitis or atresia coli.

The oral cavity of the calf is easily examined and should be part of the clinical examination of every sick calf.

A common problem in calves under two months of age is acute abdominal distention. The distention may appear to be symmetrical and it is difficult to determine if the distention is in the rumen, abomasum,

intestines or peritoneal cavity. A rectal examination of abdomen is not possible.

The common causes of acute abdominal distention in calves include:

- (See table for differential diagnosis)
- Abomasal torsion
- Abomasal dilatation (fluid, milk, abomasal ulcers and hair balls)
- Acute enteritis (the diarrhea may have not yet begun)
- Acute diffuse peritonitis.

Examination of the abdomen of the young calf includes visual inspection of the contour of the abdomen to determine which side is most distended, deep palpation and ballottement of each flank to determine the presence of fluid-splashing sounds which indicates a fluid filled viscus and percussion and auscultation to determine the presence of a gas-filled viscus. Sitting the calf on its hindquarters and allowing

the viscera to move to the caudal part of the abdomen may allow visual adspction and palpation of a distended abomasum below the xiphoid sternum. With the calf in lateral recumbency, careful palpation and simultaneous auscultation may reveal the location of the distended viscus. However, it is often necessary to do an exploratory laparotomy to determine the cause. A stomach tube should always be passed into the rumen to relieve any pressure caused by the accumulation of gas or fluid. In the case of severe distention of the abdomen accompanied by severe abdominal pain (kicking, bellowing, rolling, getting up and lying down) it may be necessary to relieve pressure with a large guage needle (12 to 14 gauge, 3 to 4 inch). The most common cause of severe abdominal distention which can be particularly relieved in a young calf is abomasal torsion.

Paracentesis abdominis is easily done in the calf, at least three taps should be done before concluding a negative tap. To avoid puncture of the abomasum choose sites which are caudal to the umbilicus.

DIFFERENTIAL DIAGNOSIS OF DISEASES OF THE DIGESTIVE TRACT AND ABDOMEN
OF YOUNG CALVES PRESENTED WITH DISTENTION OF THE ABDOMEN

Disease	History, Clinical and Laboratory Findings, Treatment
<u>Abomasal Torsion</u>	Always acute to peracute, one week to six months of age, acute abdominal pain, bellowing, up and down, severe tight distention of abdomen, loud ping and fluid-splashing sounds right side, emergency surgery necessary.
<u>Abomasal Dilatation</u> (fluid, milk, hair balls and often abomasal ulcers)	Chronic or acute onset, calves one to six months of age, history of abnormal feces, may be unthrifty, mild to moderate abdominal distention and pain, fluid-splashing sounds over right flank, dehydration, negative peritoneal fluid, surgery.
<u>Perforated Abomasal Ulcers</u>	Acute onset, sudden collapse, calves two weeks to three months, hand-fed or nursing calves, weakness, recumbency, tachycardia, mild to moderate abdominal distention, mild or no abdominal pain, abdominal splinting occasionally, <u>positive paracentesis</u> , feces variable.
<u>Torsion of Root of Mesentery</u>	Sudden onset, found in state of collapse, abdominal pain common, moderate abdominal distention, distended loops of intestine visible and palpable over right flank, blood-stained peritoneal tap, fluid-splashing sounds on palpation and auscultation, scant feces, emergency surgery.
<u>Acute Diffuse Peritonitis</u> (not due to perforated abomasal ulcer)	Usually in calves under three weeks of age. Toxemia, temperature variable, weak, may be grunting, splinting of abdominal wall, mild abdominal distention, scant feces, fluid-splashing sounds over right flank (due to paralytic ileus), <u>positive paracentesis</u> , commonly associated with enteric colibacillosis, polyarthritis and umbilical and urachal abscess.
<u>Atresi coli</u>	Calf usually under ten days of age, progressive distention of abdomen, bright and alert for first few days then becomes depressed, no feces only thick mucus from rectum, insertion of tube into rectum may lead to blind end but often blind end is near spiral colon. Surgery indicated but often unrewarding.

Disease	History, Clinical and Laboratory Findings, Treatment
<u>Intussusception</u>	May have history of diarrhea, now scant blood-stained feces, depressed, will not suck or drink, dehydrated, contour of abdomen may appear normal or slightly distended, fluid-splashing sounds and small ping may be audible, blood-stained peritoneal fluid, pre-surgical diagnosis often difficult, surgery necessary.
<u>Peracute to Acute Enteritis</u>	Usually in calves under three weeks of age, acute onset of abdominal pain (kicking, stretching), won't suck or drink, may not yet appear dehydrated, temperature variable (if elevated is reliable), mild to moderate abdominal distention, fluid-splashing sounds, continuous loud peristaltic sounds on auscultation, digital examination of rectum may stimulate defecation of foul-smelling soft-watery feces, peritoneal tap negative.

11. *Use of the Stomach Tube (Usual Size and Colorado Kingman Tube - inside diameter 3 cm).*

In adult cattle presented with severe abdominal distention due to gross distention of the rumen it is difficult, if not impossible, to assess the status of the abdomen. To determine if the rumen is distended and/or to relieve the pressure a large bore stomach tube should be passed. In vagus indigestion, the rumen may be grossly distended with fluid contents which will gush out through a large bore tube. In some cases 20 to 30 gallons of rumen contents may be released. If no contents are released the contents may be frothy or mushy and the rumen end of the tube will plug almost instantly. Rumen lavage may then be attempted using water hose to deliver from five to ten gallons of water at a time followed by back drainage by gravity flow. After the rumen is partially emptied it is usually possible to more accurately assess the rumen and the abdomen.

12. *Paracentesis of the Abdomen.*

In cattle, the choice of sites for paracentesis is a problem because the rumen covers such a large portion of the ventral abdominal wall, and avoiding penetration of it is difficult. In cattle, one recommended site is left of the midline and 3 to 4 cm medial and 5 to 7 cm cranial to the foramen for the left subcutaneous abdominal vein (2). A teat cannula similar to the one described for use in the horse is recommended but with care and caution a 16 gauge 5 cm hypodermic needle may also be used. A syringe is attached to the needle which is pushed carefully and slowly through the abdominal wall. A twitch of the abdominal wall will occur when the peritoneum is punctured and at that point, vacuum is applied. It may be necessary to move the needle around a few different directions before fluid is obtained. Failure to obtain a sample does not preclude the presence of peritonitis. The exudate may be very thick, large masses of fibrin may be present or the peritonitis may be localized. In acute diffuse peritonitis, a sample is usually readily

obtainable. In local peritonitis or in long-standing cases, it may be necessary to attempt as many as four different sites. The most profitable sites are those in which, on an anatomical basis, there are recesses between the forestomachs, abomasum, diaphragm and liver. These are usually caudal to the xiphoid sternum and 4 to 10 cm lateral to the midline. Normal cattle may yield 1 to 5 ml of clear, serum-like fluid containing mesothelial cells, lymphocytes, neutrophils, a few red blood cells and occasional monocytes and eosinophils. Lymphocyte and segmented neutrophils are found in normal cattle in the ratio of approximately 1 : 1. The presences of large numbers of red blood cells is a grave prognostic sign (e.g. ruptured uterus). Inflammation or irritation of the peritoneum results in an increase in the total number and percentage of neutrophils. In chronic peritonitis, the number of neutrophils decreases while the number of monocytes increases.

The Use of Guidelines and Criteria to Decide When to do a Laparotomy in Cattle with Suspected Disease of the Digestive Tract and Abdomen.

Laparotomies are done in cattle to assist in the diagnosis of diseases of the digestive tract and/or to correct certain diseases for which a diagnosis has been made. When a significant lesion is found in laparotomy the surgery is usually justifiable whether or not the outcome is favourable. However, because a properly done laparotomy is time consuming and expensive the veterinarian would like to minimize the number of laparotomies in which no significant lesions are present. The challenge is therefore, to improve the accuracy of diagnosis before doing a laparotomy unnecessarily.

There are some well recognized diseases in which, if a clinical diagnosis can be made, a laparotomy is indicated. (In some cases slaughter for salvage may be more economic (see Table).

Other than the rumenotomy for the treatment of grain overload and the Caesarean section, the most common indication for a laparotomy in cattle is for the surgical correction of obstructions of the digestive tract (i.e., abomasal dilatation and torsion, intussusception and

Interpretation and Pathogenesis of Clinical Abnormalities Associated with Diseases of the Digestive Tract and Abdomen of Cattle

Clinical Abnormality	Interpretation, Pathogenesis
Anorexia, inappetence	Toxemia, distension of intestines and stomachs, enteritis, peritonitis
Scant feces, Includes small volume diarrhea	Reduced feed intake, functional obstruction of forestomachs and abomasum, paralytic ileus, strangulation obstruction
Large volume diarrhea	Profuse watery diarrhea usually associated with enteritis or carbohydrate engorgement
Dehydration	Failure to drink adequate amounts of water (due to toxemia or lesions of oral cavity), malabsorption due to enteritis, diseases of the forestomachs interfering with absorption of water, e.g. Vagus indigestion.
Tachycardia	Toxemia, acid-base imbalance, abdominal pain, distension of intestines.
Polypnea	Acid-base imbalance (torsion of the abomasum, severe enteritis, vagus indigestion), distension of the abdomen due to gas or fluid filled intestines.
Weakness and recumbency	Toxemia, severe dehydration, severe distension of abdomen, peritonitis
Colic (abdominal pain)	Sudden onset of distention of forestomachs, abomasum or intestines. Stretching of mesenteric bands. Strangulation of intestine in mesenteric tear or hernia (bulls).
Grunting with every respiration	Diffuse peritonitis (also pleuritis, pulmonary imphysema and advanced pneumonia), distension of stomachs or intestines.
Presence of grunt on deep palpation of ventral abdominal wall	Presence of a peritoneal lesion (stretching of the peritoneum, inflammation, edema, recent adhesions).
Abdominal distention	Most commonly due to gas or fluid-filled intestines and/or forestomachs and abomasum. Rarely due to pneumoperitoneum. Also due to ascites and hydroys-allantois-amnion.
Rumen distention	May be distended with gas, fluid or ingesta. Primary dietary ruminal tympany and grain overload. Secondary ruminal tympany due to peritonitis, vagus indigestion.
Rumen stasis	Toxemia, Metabolic (hypocalcemia), Fever, Ruminal acidosis, Distension of omasum or abomasum, peritonitis, vagal nerve injury.
Hyperactive Rumen	Early stages of primary dietary ruminal tympany. Vagal nerve injury.
Acidic Rumen pH	Ruminal acidosis associated with carbohydrate engorgement. Almost no other cause known.
Alkaline Rumen pH	Ruminal alkalosis associated with accidental consumption of high protein diet, urea poisoning.
Reduced or absent rumen protozoan activity	Ruminal acidosis (lactic acid inactivates protozoa). Primary starvation lasting more than 2 to 3 days. Ingestion of lead, arsenic and other poisonous substances.

Abnormal Foul-smelling Rumen contents	Putrefaction of rumen contents in static and defaunated rumen.
Presence of "ping" over left flank	Left sided displacement of abomasum. Atonic rumen with a gas cap.
Presence of loud or clear "ping" over right flank	Right side displacement and torsion of the abomasum, cecal dilatation and torsion, torsion of the spiral colon, gas in distended colon and rectum.
Presence of low pitched "pings" not clearly distinct over right flank.	Tympany of right paralumbar fossa in recently calved cows (2 to 3 days). Gas in distended colon and rectum. Fluid and gas-filled intestines with enteritis.
Distended upper right flank	Dilatation and torsion of abomasum. Cecal dilatation and torsion. Torsion of spiral colon.
Distended lower right flank Heavy viscous in right lower flank.	Impaction of the abomasum. Enlarged L-shaped rumen and distention of ventral sac to the right flank. Advanced pregnancy.
Fluid splashing sounds on ballottement of abdomen or succussion.	Fluid-filled intestines or forestomachs or abomasum. Usually associated with enteritis, paralytic ileus, or obstruction. Fluid-splashing sounds are <u>rarely</u> present when fluid is present in the peritoneal cavity.
Dropping Cuds	Cattle rarely vomit (arsenic poisoning). Dropping of aids usually associated with chronic inflammatory lesions of the reticulum and cardia resulting in lack of control of regurgitation and a larger than normal bolus of rumen contents is regurgitated which cannot be controlled by the animal.

volvulus, torsion of the root of the mesentery, torsion of spiral colon, cecal dilatation and torsion). If any of these diagnosis can be made, a laparotomy or slaughter is indicated.

In other cases, the diagnosis may be suspected but is not obvious and the indications for a *laparotomy, slaughter, euthanasia or conservative medical treatment* are not clear.

The major question is, "Under what conditions is a laparotomy indicated if the history and clinical and laparotomy findings *suggest* an obstruction (strangulation obstruction or functional) *but* the obstruction cannot be located on clinical examination?"

Some diseases which may elude diagnosis before laparotomy, and which are or may be amenable to surgical correction include:

1. *Intussusception and other strangulation obstructions of the small intestines* and located in the anterior aspect of the abdomen (not palpable per rectum). Depend on history of acute onset of colic, absence of feces and exudate in peritoneal tap. A progressively worsening clinical course warrants a laparotomy.
2. "*Atypical*" *left sided displacement of the abomasum* (a small percentage of cases are difficult to detect on

auscultation and percussion). When the typical LDA ping cannot be detected after several examinations over a period of a few days, a presumptive diagnosis may be made on the basis of ketosis in a recently calved cow (within the last week), the presence of rumen contractions but reduced intensity, normal vital signs (unless fatty liver is present), and fluid-gurgling sounds over the left flank.

3. *Traumatic reticuloperitonitis* with a persistently penetrating foreign body. Conservative medical treatment of immobilization in a stanchion, antibiotics and a magnet has not resulted in a beneficial response. Diagnosis depends on continued anorexia, mild fever,

grunt, rumen stasis, a hemogram indicating infection and peritoneal fluid containing exudate.

How long can you delay doing a laparotomy in a cow in which the history and clinical findings *suggest the possibility* of a surgically correctable obstruction of the digestive tract but the clinical evidence is not sufficiently definitive? For example, initial bouts of abdominal pain which have now subsided, complete anorexia, *absence* of feces, no palpable distended loops of intestine or viscuses, some degree of dehydration. Is this an intussusception located in the anterior aspect of the abdomen or a case of idiopathic paralytic ileus which will resolve spontaneously in three days? How long can the veterinarian wait before doing a laparotomy in a cow with an intussusception and still be early enough to correct the obstruction successfully?

The Use of Guidelines and Criteria to Decide When to do a Laparotomy
in Cattle with Disease of the Digestive Tract and Abdomen

Parameter/Criteria	Significance and Interpretation of Criteria
History	Does the history suggest a surgically correctable condition?
Systemic State, Habitus	Heart rate over 100 per minute suggests impending shock but rates below 100 do not preclude existence of an obstructive lesion. <u>Progressive</u> dehydration, progressive weakness, persistent recumbency indicates worsening situation and laparotomy unless can detect incurable lesion. Temperature unreliable. Complete anorexia suggests a severe lesion.
Abdominal Distention	Laparotomy indicated if distention of abdomen caused by distention of abomasum, cecum, intestines.
Nature and Amount of Feces	Scant or absence of feces for more than 36 to 48 hours indicates a <u>functional</u> or <u>physical</u> obstruction. In <u>functional obstruction</u> (i.e. peritonitis) some dark feces usually present. In <u>physical obstruction</u> (intussusception) feces are very scant and dark red due to leakage of blood into intussusceptus. Laparotomy indicated unless can determine that cause of absence of feces is not surgically correctable (diffuse peritonitis or impaction of abomasum or omasum).
Rectal Findings	Distended viscuses other than rumen (abomasum, cecum, small and large intestines) warrants laparotomy. Palpable "bread and butter" fibrinous inflammation in caudal part of abdomen suggests acute, diffuse peritonitis and laparotomy would not be rewarding.
Peritoneal Tap	Blood-stained exudate suggests leakage of the intestinal wall and warrants laparotomy if history and clinical findings suggest a strangulation obstruction.
Hemogram	A degenerative left shift in the leucocyte count suggests infection. Laparotomy indicated if the history and clinical findings suggest the possibility of strangulation obstruction.
State of the Rumen	The fullness of the rumen, the nature of the rumen contents and the regularity and intensity of the contractions are reliable indicators of the state of the digestive tract beyond the rumen. Complete rumen stasis suggests a significant lesion of the rumen itself or of the abomasum, intestines or peritoneum.

History

Significance and Interpretation of Criteria

Abdominal Pain (colic) and grunting.	Behavioural and postural signs of acute abdominal pain (colic) such as kicking at the belly, stretching the body suggest acute distention of the stomach or intestines with fluid and gas. A laparotomy is indicated if the clinical evidence suggests that obstruction is the cause of the distention. Spontaneous grunting with each respiration, which usually becomes pronounced in sternal recumbency, or the presence of a grunt on deep palpation of the abdomen suggests inflammation of the peritoneum.
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DISEASES OF THE DIGESTIVE TRACT AND ABDOMEN OF CATTLE IN WHICH A LAPAROTOMY IS INDICATED IF THE DIAGNOSIS CAN BE MADE

DISEASE	MAJOR CLINICAL FINDINGS
Left side displacement of the abomasum.	"Ping" over 9-12th ribs and other well recognized findings.
Right side displacement and torsion of the abomasum.	Distention of upper right flank, "ping", palpable per rectum.
Cecal dilatation and torsion.	Distention of upper right flank, "ping", long cylindrical mass palpable per rectum.
Torsion of spiral colon.	Distention upper right flank, "ping", distended loops of intestine easily palpable.
Intussusception.	Abdominal pain, absence of feces, distended loops of intestine, palpable intussusception.
Enteroliths.	Scant feces, abdominal pain, distended loops of intestine palpable pitting mass.
Severe life threatening ruminal tympany.	Severe distention of rumen, skin over rumen cannot be picked up, animal grunting, are lying down, mouth breathing cannot relieve with stomach tube or trocar.
Unidentifiable lumps palpable on rectal examination.	Chronic gastrointestinal atony, scant feces, large hard lumps palpable per rectum.
Peracute grain overload.	Weakness, recumbency, dehydration, tachycardia, rumen pH 5. (See Table for guidelines in the treatment of grain overload.)

DISEASES OF THE DIGESTIVE TRACT AND ABDOMEN OF CATTLE IN WHICH

THE INDICATION FOR A LAPAROTOMY MAY NOT BE OBVIOUS OR MAY NOT BE INDICATED

DISEASE	MAJOR CLINICAL FINDINGS AND INDICATIONS FOR LAPAROTOMY
Traumatic reticulo-peritonitis with persistently penetrating foreign body.	Rumen stasis, complete anorexia, no response to therapy for 3 days, positive hemogram, positive peritoneal tap, presence of grunt. Laparotomy indicated in valuable cow.
Vagus indigestion.	Gross distention of rumen, scant feces, anorexia, valuable cow or bull.
Diffuse peritonitis.	Toxemia, grunting, positive hemogram and peritoneal tap. Treat with antimicrobials for several days.
Abomasal impaction.	Distention of lower right flank with heavy viscus behind costal arch, doughy mass palpable per rectum in lower right quadrant. Slaughter for salvage or treat medically with mineral oil or D.O.S.S.

THE USE OF CERTAIN CLINICAL PARAMETERS IN ASSESSING THE SEVERITY OF GRAIN OVERLOAD IN CATTLE FOR THE SELECTION OF THE TREATMENT OF CHOICE

Clinical Parameters

Degree of Illness	Mental state & muscular strength	Degree of dehydration (% of BW)	Abdominal distension	Heart rate (min.)	Body temp. (°C)	State of rumen; fullness, consistency of contents, movements and pH	Treatment
Peracute	Severely depressed, weak, in lateral recumbency, unable to stand, apparent blindness, pupils dilated and slow response	8-12	Prominent	110-130	36.5-38	Distended with fluid complete stasis, sweet-sour smelling fluid contents. Rumen juice pH below 5 and usually about 4. No protozoa	Rumenotomy. Sodium bicarbonate 5 litres (5%) i.v. in 30 minutes followed by isotonic (1-3%) for 6-12 hours
Acute	Depressed, still able to walk but ataxic, complete anorexia, may want to drink water, pupils slightly dilated and slow response	8-10	Moderate	90-100	38.5-39.5	Distended with fluid, complete stasis, sweet-sour smelling fluid contents. Rumen pH between 5 and 6. No protozoa	Consider immediate slaughter. Rumen lavage or rumenotomy. Sodium bicarbonate intravenously as in peracute case. Feed hay

Subacute	Fairly bright and alert. Able to walk. No ataxia. May eat, usually wants to drink. Pupils normal	4-6 Just barely detectable clinically	Mild or none	72-84	38.5-39	Moderate distension with fluid, some doughy ruminal ingesta palpable, some weak ruminal contractions, rumen pH between 5.5 and 6.5. Some protozoa alive	Magnesium hydroxide 500 g/450 kg BW into rumen. Fluids if indicated. Feed hay. Should begin eating 24-36 hours
Degree of Illness	Mental state & muscular strength	Degree of dehydration (% of BW)	Abdominal distension	Heart rate (min.)	Body temp. (°C)	State of rumen; fullness, consistency of contents, movements and pH	Treatment
Mild	Bright and alert. Able to walk, no ataxia, eats and drinks normally	Not detectable clinically	Not significant	Normal	Normal 38.5-39	No detectable distension, ruminal contents palpable, ruminal contractions still present but not as strong as normal, rumen pH 6.5-7. Almost normal protozoa activity	Feed hay and observe for 48 hours. Watch for anorexia

THE MAKING OF A DIAGNOSIS AND THE DECISION ON WHETHER OR NOT TO DO A LAPAROTOMY IN A COW WITH GASTROINTESTINAL ATONY AND ANOREXIA.

