A HANDBOOK ON RUMINANT INTERNAL MEDICINE

DEPARTMENT OF CLINICAL MEDICINE AND THERAPEUTICS
MADRAS VETERINARY COLLEGE, MADRAS-600 007
MIL NADU VETERINARY AND ANIMAL SCIENCES UNIVERSITY
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The book entitled, "A hand book on Ruminant Internal Medicine" is being published by the Department of Clinical Medicine and Therapeutics, Madras Veterinary College, Madras 600 007. It has been a long cherished need for the publication of a hand book for bovine practitioners covering the different aspects of ruminant's internal medicine. Since, the undergraduate curriculum does not sufficiently cover the variety of problems faced by field veterinarians, the availability a hand book to understand the recent concept of ruminant's internal diseases is the need of the hour.

Catching up with the recent developments in dairy industry, the Government is importing exotic breeds of cattle to boost the milk production. Many private agencies have taken up dairy industry all over the country. In order to achieve and maintain higher milk yield, the animals need timely and correct treatment, which is the responsibility of the veterinarians. In order to equip with the modern advancements made in the practice of biatrics, this book will go a long way to the field veterinarians.

The contributions from the authors of the various articles have been arranged in such a way that the fundamental knowledge on anatomy, physiology and pharmacology is obtained before going through the ruminant's internal diseases, their diagnosis and treatment. In fact, most of the articles were forming the part of the curriculum and syllabi of the short term study course on 'Ruminant Medicine' conducted in the Department of Clinical Medicine and Therapeutics since 5 years for the benefit of the practising veterinarians in the field.

My thanks are due to the associate editors for their zeal and enthusiasm with which they worked to make the publication of this book. I am indebted to acknowledge the cooperation from the authors of the various articles for their earnest cooperation for the publication of this book.
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ANATOMY OF THE RUMINANT-STOMACH
AND
ITS APPLICATION IN CLINICS

Dr. C. VIJAYARAGAVAN

Introduction:

The Ruminants derive their name from the Latin word "Ruminare" which means that one chews over again and again. The Rumen is popularly known as "Paunch" the term referring to the "fermentation vat" it possesses, as a main digestive organ.

The ruminant stomach consists of 4 compartments, the Rumen, Reticulum, Omasum and Abomasum, the first 3 compartments being well known as fore-stomach as they are non-glandular.

In a cattle of medium size, the capacity of the stomach varies from 115 to 150 litres, which is shared in the proportions of 80%, 5%, 7% and 8% by the 4 compartments, respectively. In sheep and goat the capacity of the stomach is only 15 to 18 litres and the rumen and omasum are relatively smaller. The relative capacities of the 4 compartments being 71%, 8%, 2% and 19%.

Development:

Because of the structural identity, initially the fore-stomach was believed to be mere dilatations of the oesophagus, which was disproved by subsequent research where a simple dilatation, just posterior to the stomodeal portion was identified as the primordium of the ruminant stomach.

Rumen is the first compartment that gets differentiated from the primitive stomach as early as 40 days. By 60 days all the four compartments get fully established and all of them lie in a cranio-caudal straight line. From 90 days by differential growth apparent straight cranio-caudal continuity is lost, and the subsequent morphological changes in the individual organs in addition to sequential positional rotations bring them to the final topographical orientations.

The relative sizes of the four compartments change with age. In the new born calf, rumen and reticulum are about half of the capacity of the abomasum and are non-functional as the calf is only on milk diet. At 8 weeks the combined capacity of the rumen and reticulum equals that of abomasum and at 12 weeks the rumen and reticulum are twice the capacity of abomasum. The omasum grows slowly during these periods and reaches its capacity equal to abomasum at about one and a half years.

Function:

The fore-stomach of the ruminant provides for extensive pre-gastric microbial fermentation as against the caecum and colon of non-ruminants. Out of the four compartments it is the rumen and reticulum that have an environment conducive for microbial growth and fermentation.

Retention of fibrous food for a longer time and making of coarse food into smaller particles by remastication, facilitates increased microbial fermentation. In addition, rumen bacteria synthesise aminoacids, proteins and B-complex vitamins. The lining stratified squamous epithelium of cornified variety of the rumen does not bar the absorption of volatile fatty acids, and transport of sodium ions, a unique speciality of the epithelium of this kind peculiar to ruminants alone.
Topographical Anatomy and its Application in clinics:

The ruminant stomach occupies nearly 3/4 of the abdominal cavity. The rumen fills the entire left half of the abdomen, except small spaces occupied by the spleen and part of the small intestine, and extends well into the right half. The rumen is in contact with the left abdominal wall from the 8th intercostal space to the transverse plane of the tuber coxae. It is divided into dorsal and ventral sacs by the two longitudinal grooves running on either side of its length connected at the extremities by the transverse grooves to result in the formation of blind sacs at both the ends.

The contractions of its dorsal sac may be readily palpated or auscultated in the left paralumbar fossa, bounded in front by the last rib and its cartilage, above by the lumbar transverse processes and behind by the ilium. In the same manner auscultation of the sound of the rumen on the left cage in addition to the usual left paralumbar fossa, would enable differential diagnosis in conditions like abomasal displacement, caecal dilatation or dislocation. The ventral sac of the rumen is enclosed in the greater omentum and lies on the ventral abdominal wall caudal to the transverse plane of the ninth costo-chondral junction. Motility of the ventral sac is an event of sequence of dorsal sac motility and hence special efforts are not taken to observe its rhythm separately.

The reticulum or honey-comb is in contact with the left-abdominal wall, at the ventral ends of the 6th and 7th intercostal spaces, with greater part of it lying on the left of the median plane. The diaphragmatic surface is concave and lies against the diaphragm and liver, and inturn also contacts the pericardium and lungs.

Auscultation at the ventral ends of the 6th or 7th intercostal space, eight fingers above the sternum (William's method) with simultaneous pressure on the rumen at the left paralumbar fossa, is used to diagnose traumatic reticulo pericarditis, fibrosis of the reticular wall, peritonitis and diaphragmatic hernia. The same site is preferred by many to observe reticular movements, which are heard as a rumbling gurgle accompanied by a liquid pouring sound.

It is well known that the oesophagus opens into a shallow vault, in the rumino reticular wall, called as Atrium Ventriculi. Hence the cardia, the termination of the oesophagus can be auscultated in the area of the atrium ventriculi, which is approximately at the junction of the upper and middle third of the 8th intercostal space. This site is located by first drawing a curved line from the point of elbow to the upper aspect of the last rib indicating the position of the diaphragm, which is intersected by another horizontal line from the submandibular region, at the position of cardia. The animal evinces a grunt, when the probang - passed, reaches the cardia in early cases of diaphragmatic hernia.

The omasum or many folds or many plies or psalterium is the last division of the non glandular stomach of a ruminant and it is in contact with the right abdominal wall in the ventral parts of the 7th to 9th intercostal spaces and with the abdominal floor in a small area between the xiphoid cartilage and the right costal cartilages. But in sheep and goat the omasum has no contact with the abdominal wall.

The auscultation of the omasum is done on the right side at the 9th intercostal space on a level with shoulder joint. This site is very useful for the diagnosis of omasal impaction, acute abomasitis, and pyloric stenosis. Normal omasal sounds are heard as gurgling sounds as ingesta passes through omasum to abomasum.

Likewise, percussion field can be easily located in a semicircle from a hand's breadth above the costal arch, limited hindwards by the 10th rib or 10th intercostal space. The percussion sound will give clear gas tone a little more dull and heavier than the lung tone. The omasal field will become smaller in chronic diarrhoea and functional disorders like stenosis between the reticulum and omasum. But in pyloric stenosis the omasal field will be larger.
The fundus of abomasum or rennet or true-stomach lies on the abdominal floor in the xiphoid region in the mid ventral line, while the pyloric part curves around to the right and hence it is near the ventral end of the right 9th or 10th intercostal space. In sheep and goat the abomasum lies on the abdominal floor along the right costal cartilages.

Abomasal samples can be collected by a puncture with a needle (4-8 cms) midway between the xiphoid cartilage and the umbilicus. Further the abdominal paracenthesis poses a great problem in ruminants, because of the massive rumen lying on the entire area of the floor of the abdomen. The recommended site for paracenthesis being 3-4 cms medial and 5-7 cms cranial to the foamen for the left subcutaneous abdominal vein, 16 gauge 5 cms long hypodermic needle may be suitably used.

Internal features:

The functional significance of the fore-stomach are important for any therapeutician, as these compartments play a major role in the digestion in ruminants and in any disorder they warrant his attention for necessary correction. Hence a brief description of the structures in the interior of these compartments will be of some use for the better understanding of their function.

The external grooves on the rumen are represented by pillars on its internal aspect, which are mere folds of the wall strengthened by additional muscle fibres. Dorsal to the right longitudinal pillar is an accessory pillar. The left longitudinal groove/pillar does not reach the caudal transverse groove/pillars just as the right side one which gives off the dorsal and ventral coronary grooves/pillars which divide the caudal blind sacs from the main dorsal and ventral sacs, respectively.

The mucous membrane of the rumen is brown or black except on the margins of the pillars, where it is pale. Except the chief pillars and the mid-dorsal wall of the dorsal sac the rumen is studded with large papillae with high vascularity. The papillae are of 4 types based on their shape, namely 1. short conical 2. Tongue shaped 3. Filiform and 4. Spatulate. The papillae are largest and numerous in the atrium. Whereas in the blind sacs the papillae per unit area are less. Among the blind sacs the anterior dorsal blind sac has the least number of papillae. In general the stimulus for the development of papillae in the early life is the presence of organic acids particularly the volatile fatty acids, though the order of preference of substance for the greater development of papillae are 1. Butyrates, 2. Propionates 3. Sodium citrates, glucose and sodium chloride.

Sloughing of older papillae is more in the early age groups than in the later age groups. Normally the density of the papillae decreased with increase of age and this is attributed to the growth and increase in the area of rumen wall with the number of papillae remaining more or less the same. Lack of papillae on the mid-dorsal wall, usually after one year of age is associated with the regular presence of a large bubble of gas on the top of the ingesta preventing the roof of the rumen coming in contact with the stomach contents.

In the reticulum the mucous membrane is raised into folds about 1cm, high, which encloses four, five or six sided cells. The cells are sub-divided by smaller folds and are studded with pointed horny papillae. The cells grow smaller and gradually disappear near the reticular groove. At the reticulo-omasal orifice there are peculiar horny papillae which resemble the claws of a small bird.

The presence of the reticular or esophageal groove connecting the cardia to the reticulo-omasal orifice does not warrant any special mention, because of the functional familiarity.

The cavity of the omasum is occupied by about hundred longitudinal muscular folds-the laminae omasi which spring from the greater curvature. The largest of these about half a dozen in number have a superior convex attached edge and a thick concave free edge. A groove, sulcus omasi, extends from the reticulo-omasal opening to the omasal abomasal opening. It is free from laminae.
The cavity of the abomasum is divided into two parts by a constriction. The abomasum is the true stomach and is lined by a glandular epithelium. In the first part there are about a dozen or more spiral folds. The second part is narrow and pear-shaped and presents a yellowish brown mucous membrane with transient transverse folds. A transient semicircular Torus pyloricus is formed at the abomasal duodenal junction in the lesser curvature.

Normal rumino-reticular cycle.

The wave of contraction starts at the reticular groove and the reticulum undergoes two rapid contractions the first being peristaltic and the second antiperistaltic. A spiral form of wave of contraction then proceeds over the anterior rumen and the cranial pillar contracts to form a septum dividing the rumen into anterior and posterior sacs. The contraction of the cranial pillar then involves the longitudinal pillars forming a prominent boundary between the dorsal and ventral sacs of the rumen and shortens the side walls of the rumen. At this time the caudal and dorsal coronary pillars contract compressing the caudo-dorsal blind sac of the rumen. The caudo ventral blind sac and ventral sac then contracts with dilation of the caudo-dorsal blind sac and relaxation of the cranial pillar.

A second wave of contraction passes back over the caudal and dorsal coronary pillars, the longitudinal pillars, the cranial pillar and finally the cranial sac of the rumen. When this antiperistaltic wave reaches the rumen antrum, the cardia opens which is accompanied by their eructation or regurgitation phase of rumination.

These contractions of the Rumino-reticulum continues day and night throughout the life, repeating themselves at 30-60 seconds intervals. The rate of contraction, increases during feeding and recumbency. It is apparent that the distension of the rumen provides the major stimulus for reflex control of the rumino-reticular cycle but the number of secondary rumen contractions are affected by other stimuli perhaps tactile. During reticular contraction the lower end of the reticular groove is drawn into a funnel around the reticulo-omasal orifice which gets dilated with a sudden rush of reticular contents into omasal canal.

Blood Supply:

The three primary branches of the coeliac artery the first collateral of abdominal aorta, namely, hepatic, splenic and left gastric, with their collaterals supply the various compartments of the ruminant's stomach. The venous drainage from the ruminant stomach being by the splenic and gastro-duodenal veins, which are tributaries of the portal vein.

Nerve Supply:

The ruminant stomach is mainly under the innervation of vagus, which also carries parasympathetic fibres to all the viscera under its control.

The vagus enters the abdomen as dorsal and ventral vagal trunks. The dorsal vagal trunk gives branches to the coeliac plexus for distribution along with the post ganglionic sympathetic fibres to different regions of the ruminant stomach and intestines. It innervates the dorsal surface of ruminant stomach and intestines. It innervates the dorsal surface of rumen, the right surface of the reticulum, the junctional area between reticulum and omasum. One of the two main branches of this trunk, the right ruminal, innervates the entire right side of the rumen, posterior blind sacs and the posterior 1/3 of the left side of the rumen. Another major branch the left gastric, innervate both the surfaces of the omasum, visceral surface of the abomasum and pylorus.

The ventral vagal trunk gives several branches to the right face of the reticulum, area ventral to the oesophagus, left side of the atrium and fundus of the reticulum. The long pyloric branch of it innervates the pylorus and anastomoses with dorsal and ventral vagal trunks. The continuing trunk innervates the omasum and pylorus.
The liver is the largest gland of the body. The most important function of it is the secretion of bile. In the foetus, it is a blood forming organ during prenatal life. An important function of liver is the storage of glycogen, which it synthesizes from the carbohydrates received through the portal blood. It converts the products of protein catabolism to urea and uric acid which are discharged into the blood stream and then removed by the kidneys. It also removes the waste products resulting from the breakdown of red blood cells in the spleen, from the blood. These are the end products of haemoglobin catabolism and are discharged in the bile as bile pigments. The liver is also capable of extracting harmful substances from the blood and detoxifying them.

Gross or macro anatomy

The liver is reddish brown in colour. Suckling and pregnant animals and those on a fattening diet have a yellowish brown liver because of the presence of fat, whereas emaciated or starving animals have a dark reddish brown liver.

The size and weight of the liver vary greatly. Because it stores fat and glycogen, it weighs more in well fed animals than in emaciated ones. The weight of the liver always decreases with age. In ox the liver weighs 3 to 10kgs which forms 1.03 to 1.54% of the total body weight.

The serosal covering of the liver gives a smooth and glossy appearance. The liver consists of small lobules which are visible to the naked eye only when surrounded by a noticeable amount of inter lobular tissue. The liver has a granular appearance when freshly ruptured.

The liver is firm, yet somewhat elastic to touch and quite friable. When in situ, it molds itself readily to the neighbouring structures, but it flattens out when removed from the carcass in the fresh state.

The diaphragmatic surface of the liver is convex and lies against the concavity of the diaphragm. Its visceral surface faces mostly caudally and is related to the stomach, duodenum, colon, jejunum and to the right kidney. These structures indent the liver and accordingly produce impressions as oesophageal notch, reticular impression omasal impression, umbilical fissure, portal fissure, pancreatic impression, cystic impression and the renal impression. These impressions disappear when the liver is removed in the fresh state, but remain if the liver is embalmed in situ.

The liver lies in the intrathoracic portion of the abdominal cavity. It is displaced entirely to the right of it is oriented in such a way that the rounded dorsal border of the liver lies in the median plane and that the sharp peripheral border is directed mainly to the right, but ventral to the oesophageal notch also to the left and in the region of right triangular ligament also dorsally.

When in situ, the liver is encased between diaphragm cranially and the viscera caudally and it is prevented from sliding on the surface of the diaphragm by hepatic ligament. The lesser omentum consists of hepatogastric and hepatoduodenal ligaments and extends from the area of the proximal portion of the duodenum. In addition the visceral surface of the liver is attached to the root of the mesentery by the portal vein. Cranially the liver is attached to the diaphragm by the caudal vena cava, the coronary ligament which is ventral and lateral to the caudal vena cava and by the crescent shaped falciform ligament to the sternal part of the diaphragm. In the free edge the falciform ligament contains the round ligament of liver which is the vestige of the umbilical
vein. In the suckling animal the umbilical vein is a nonfunctional, thick walled vessel, which poses a threat as a route for infection as long as the stump of the umbilical cord has not dried up.

The right and left triangular ligaments, attach the respective lobes of the liver to the diaphragm.

Owing to its intrathoracic position, the examination of the liver in the live animal is difficult. The liver can be percussed in a narrow zone along the basal border of the lung high in the right eleventh or twelfth intercostal space. Biopsy of the liver can be taken at the eleventh intercostal space. The percussion area is small corresponding to the area of contact with body wall. A detectable increase signifies a disproportionate enlargement of the organ. The preferred site for Biopsy of liver is through XI inter costal space in the plane of lower part of coxal tuber. The trocar is directed to meet the diaphragm and thus the liver is at right angles in order to ensure clean puncture. This route avoids the larger vessels.

Blood and Nerve Supply:

The portal vein enters the liver carrying venous blood rich in freshly absorbed nutrients, from the intestines and blood from the stomach, pancreas and spleen. This is the functional blood supply to the liver and constitutes the raw material for its metabolic functions. The nutritional blood brought into the liver by the hepatic branches of the hepatic artery is intended solely for the nourishment. The deep lymphatics leave the porta of the liver to the lymphnodes near the porta. The superficial lymphatics from the serous coat of the liver pass also to the caudal mediastinal, phrenic and sternal lymphnodes. The innervation of the liver is by the branches of the vagus and sympathetic nerves, which reach it from the coeliac ganglion. The hepatic vein directly open into the caudal venacava, which is embedded in the substance of the liver. Branches of the right and left hepatic ducts carry from all parts of the liver, to the porta. Here the right and left ducts unite and form the common hepatic duct, which after receiving the cystic duct from the gall bladder becomes the bile duct.

Gall Bladder:

The gall bladder is a pear shaped sac lying in a fossa on the visceral surface of the liver with which it is firmly united. The gall bladder stores bile temporarily and discharges it into the duodenum when food enters it from the stomach. It protrudes from the border of the liver.

Micro anatomy or histology:

The liver is almost completely surrounded by visceral peritoneum. The serous membrane covers the organ and is reflected over other structures-biliary duct system, arteries, veins which exist or enter the liver. The capsular connective tissue is continues with the interstitial connective tissue that serves as a supportive stroma for the parenchyma.

Hepatic lobules, comprise the morphologic units of the liver. These prismatic polygonal masses of tissues are comprised of plates or lamina of hepatocytes inter digitated between anastomatic hepatic sinusoids. The plates of cells and sinusoids appear to radiate from a centrally positioned vessel, the central vein.

The hepatocytes, comprising the parenchyma of the organ are polyhedral cells, the boundaries of which are usually distinct. The histologic appearance of the hepatocytes depend upon the physiologic state of the organism at the time of sampling. Fasted animals have small turbid and indistinct outlined hepatocytes. After a feeding the hepatocytes enlarge become distinct outlined and are filled with numerous glycogen and lipid inclusions causing a foamy appearance.

The hepatic sinusoids are the intralobular vascular supply. Blood from the interlobular vessels is transported through the sinusoids to the central veins. The sinusoids comprise a vastly anastomatic network that separates the hepatic plates from each other.
The biliary system of liver consists of bile canaliculi, intra hepatic ducts and extrahepatic ducts for the conduction of bile from the hepatocytes to the duodenum. The bile canaliculi are the smallest components of this system and are formed between the adjacent hepatocytes. Bile canaliculi become confluent with small interlobular ducts located at the periphery of the lobules and lined by cuboidal epithelium.

The portal triad, consisting of an interlobular bile duct and branches of hepatic artery and hepatic portal vein are specially obvious in regions between three or more lobules in which there is an accumulation of interlobular connective tissue; a portal canal or portal area. This area is an important landmark in the study of normal and abnormal liver.

The histologic organisation of the liver may be considered from three perspectives - morphologic, secretory and vascular. The morphologic unit is the hepatic lobule, the secretory or functional unit is the portal lobule and the vascular unit the hepatic acinus.

The hepatic lobule as described previously, is that histologic entity which has a centrally positioned vein and is bounded peripherally by varying amounts of interlobular connective tissue. The hepatic lobule is especially prominent in the pig because of the extensive amounts of interlobular connective tissue. The lobulated pattern of the liver may become more prominent in all species in certain types of disease processes characterised by an increase in interlobular connective tissue.

The portal lobule, as the secretory or functional unit of the liver, is a consideration of the organisation of the liver from the basis of its exocrine function. All of the bile canaliculi drain into interlobular bile ducts. A single, large interlobular bile duct, located within a portal area, eventually drains the exocrine secretions from adjacent hepatic lobules. The interlobular bile duct within the portal area then becomes the central focus of the portal lobule.

The hepatic acinus as the vascular unit of the liver, represents the organisation of the liver from the perspective of the vascular supply to the hepatic lobules. Branches of the hepatic portal vein and hepatic artery radiate from the portal area and extend between hepatic lobules. The end branches of these vessels open into the hepatic sinusoids. Because the axis of the hepatic acinus is perpendicular to the axis of the portal area, the sinusoids and parenchyma of two adjacent hepatic lobules as well as inter lobular blood vessels comprise the acinus. (The interlobular bile ducts follow the same pattern, but the vascular supply is the primary focus of this perspective). Three zones (peripheral, intermediate and centrolobular) are identifiable on the basis of their proximity to the interlobular vessels. The perspective afforded by consideration of the organization in terms of the vascular supply is significant in pathology. Certain disease processes of the liver parenchyma are manifested in a zonular pattern related to the end branches of interlobular vessels.

Gall bladder:

The primary functions of the gall bladder are to store, concentrate, acidify and deliver the bile to the duodenum upon demand. Bile is prevented from entering the duodenum by the sphincter of oddi, a smooth muscular modification surrounding the common bile duct as it passes through the duodenal wall. The sphincter remains closed during fasting and forces bile from the hepatic ducts and common bile duct into the cystic duct and gall bladder. When food enters the mouth, the tone of the sphincter decreases. Cholecystokinin pancreaticmin is released from intestinal cells by stimulation from fatty acids, protein digestive products and calcium in the duodenum. Relaxation of the sphincter of oddi and contraction of the smooth muscle of the gallbladder and associate ducts results from CCKPZ secretion. Substances that cause the gall bladder to contract are called cholagogues. The smooth muscle of the gall bladder and ducts is innervated by both divisions of the autonomic nervous system.

The food of herbivorous mammals contains more amount of carbohydrates than proteins whereas the food of carnivorous mammals is richer in proteins. Being an important metabolic organ, the structure of liver
should be such as to meet the different metabolic requirements. The end product of carbohydrates digestion in the intestine, glucose, on being transported to the liver is converted into glycogen and stored in hepatic cells. So, mammals which have a carbohydrate rich food should have livers which can store relatively large amounts of glycogen, when compared to those which have a protein rich food. In the latter type of mammals the structure of liver should be such that it deals with the metabolism of protein more efficiently. Protein is not stored in the liver and for an efficient protein catabolism, large quantities of protein in the form of amino acids and polypeptides must be brought to the liver. The type of liver that deals with a protein rich food should have a capacity to store more amounts of blood than the type of liver which deals more with carbohydrates.

The hepatic tubular in which the ratio: \( \frac{\text{Blood storage capacity}}{\text{Total mass of hepatic cells}} \) is greater, has a relatively greater capacity to store more glycogen than the hepatic saccular and is hence fit to deal more efficiently with the carbohydrate rich foods of herbivorous mammals. The hepatic saccular on the other hand in which the ratio: \( \frac{\text{Blood storage capacity}}{\text{Total mass of hepatic cells}} \) is greater, has relatively more blood storage capacity than the hepatic saccular and is hence fit to deal efficiently with the protein rich foods of carnivorous mammals.

Herbivorous mammals possess a tubular liver, while carnivorous and omnivorous mammals have either a saccular liver, or a liver which is transitional between the two types.
DIGESTION IN RUMEN

Dr. R. GOVINDA RAO

In the rumen, the chemical constituents of plant origin which are ingested by the ruminant undergo microbial fermentation to produce both microbial cells which are subsequently utilized as source of protein and other nutrients by the host animal. The waste products of microbial metabolism, many of which are utilized by the animal for either energy or biosynthesis.

Since extensive microbial degradation takes place in the rumen, the metabolism of ruminant animal is different from that of simple-stomached animal. Hence, the principle feature of the digestive physiology in the ruminant is the microbial digestion and fermentation which occurs on a large scale in the first two parts of the stomach, namely the rumen and reticulum. The factors of digestion in the ruminants are 1) Mechanical 2) Secretory activity of salivary glands and 3) Microbial. The mechanical factors are responsible for the mixing of rumen contents through the regular contraction of rumen and reticulum, rumination cycles and elimination of rumen gases by eructation. Any factor which interferes with the mechanical events in the rumen and reticulum will result in conditions like Tympanitis (Bloat), indigestion. The mechanical factors can be best studied by means of rumen fistula.

The food swallowed in a normal manner in a mature ruminant animal goes to the anterior dorsal sac. Some of the heavier food finds its way into the reticulum. The lighter ingesta and the heavier one collect in the rumen and gradually fill it up. There is a collection of gas above the food mass. In well-fed cattle there is not much of liquid. In fasting animals free liquid is present in ventral sac. The moist ingesta of the dorsal sac float on this liquid.

At regular intervals throughout the day the portions of the ruminal and reticular contents are returned to the mouth for remastication and reinsalivation. In the intervals in the rumen the ingesta are subjected to energetic mixing, kneading and churning action by rumen and reticular movements.

There are two waves of contraction:
1. Primary waves
2. Secondary waves

In contraction, the movement of rumen and reticulum are closely associated. First, the reticulum undergoes a sharp contraction followed by another immediately. This helps in passing the liquid material from reticulum into rumen. Before the second contraction of the reticulum, the rumen contraction proceeds as follows. The anterior pillars of the rumen begins to contract. It proceeds backward involving practically the longitudinal pillars, posterior pillars and dorsal coronary pillars. When these structures relax ventral coronary pillar muscles of ventral sac go into contraction. This causes the ingesta, to move forward and upward into the anterior dorsal region. Following the primary waves of the contraction, there is a secondary wave of contraction which proceeds in the same way as the former one.

The rate of rumen contraction are

- Eating: 2 to 3/mt 168/hr
- Ruminating: 2/mt 138/hr
- Resting: 1 to 2/mt 108/hr
Rate of reticular contraction

- Eating: 85/hr
- Rumination: 66/hr
- Resting: 69/hr

During contractions, the rumen produces sounds which are heard readily by auscultation. Reticulum also produces sounds which can be heard ventrally over the seventh rib on the left side. The major contractions of the rumen and reticulum are dependent upon vagus though they may possess a certain degree of automaticity. These contractions disappear after vagotomy and it is abolished by atropine, adrenaline and surgical anesthesia. The propulsive movements are also abolished and may lead to indigestion. The stagnation of food within this organ leads to loss of rumination and oesophageal groove reflex.

Eructation

Normally as per Hungate et al. (1955), a cow weighing 500 kg with 70 kg of rumen contents produce slightly more than 2 litres of gas per minute. The quantity and rate of gas evolved from rumen of cattle vary greatly with feed. Feeding causes production of 20 litres/30 min and at rest 5 to 10 litres/30 min. Roughly the rumen gas has the following composition:

- 30 to 65% CO₂
- 20 to 25% CH₄
- 7.000% N
- 0.56% O₂
- 0.18% H₂ and 0.01% H₂S.

These relative amounts are variable under different feeding regimes. The rumen gases are produced by bacterial metabolism. Methane is formed by the reduction of CO₂ by methanogenic bacteria. Hydrogen, formate and succinate are hydrogen donors, for this reaction. CO₂ is evolved during the fermentation of carbohydrates and deamination of amino acids. It also arises from salivary bicarbonates as a result of neutralisation of fatty acids formed during fermentation and by exchange of acids in the rumen epithelium during the absorption of fatty acids. The rumen gas is eliminated by eructation by absorption from digestive tract and elimination with faeces.

Eructation is the mechanism whereby the ruminant animal belches or gets rid of the large quantities of gas produced in the forestomach as a result of microbial fermentation. The events are described in detail by Dougherty (1961), (1968) and Dougherty et al. (1965).

The events of the eructation mechanism are associated with secondary rumen contractions for the most part. The biphasic contractions of the reticulum and the beginning phases of normal primary cycles clear the cardiac area of the ingesta. The reticulo ruminal fold and the cranial pillars remain in the contracted state acting as a dam restraining the ingesta from filling the reticulum. Dilatation of the reticulum, coinciding with contraction of the dorsal and caudodorsal blind sacs and relaxation of the caudoventral blind sac allows rumen gas to be forced into the cardiac area. During periods of high gas production two or more secondary contractions may occur.
Then cardia relax and admit the gas into the thoracic oesophagus. When the oesophagus is filled with gas the cardiac sphincter closes and pharyngeo-oesophageal sphincter relaxes. This causes the gas to be forced out of the oesophagus into the nasopharynx. Then part of the gas escapes through mouth and part is expired through the respiratory passages. The tension receptors in the area around cardia have a stimulatory action on eructation. This area is stimulated by gas pressure in the rumen and it is inhibited when the area is covered with ingesta, water, foam or mineral oil, overfilling of rumen with liquids.

Rumination

Popularly known as chewing the cud involves regurgitation of ingesta, reinsalivation, remastication and reswallowing of the cud or bolus. These four phases with light pause is called "a cycle of rumination".

Regurgitation:

The return of food to the mouth is called regurgitation. This food comes partly from the rumen and partly from reticulum. The food mass is highly mixed with liquid. The material in the region of the cardia is of such a nature which can easily enter into the oesophagus. First, there is an inspiratory effort with a closed glottis. This causes a fall in intrapulmonic, intrathoracic and intraoesophageal pressures. The pressure inside the rumen is high. The cardia relaxes and the food enters into the dilated part of the oesophagus. The pressure difference causes the flow and it has been recorded in a cow during regurgitation, a negative pressure of 40 mm Hg is created inside the oesophagus. Then reverse peristalsis of oesophagus helps in the upward passage of the bolus to the mouth. The rumen does not contract during regurgitation. It is mainly carried out by the skeletal muscles.

Remastication and reinsalivation:

When the regurgitated bolus reaches the mouth, with the first few jaw movements the animal swallows much of the liquid present in the bolus. Then the remastication of the bolus is completed during reinsalivation, the submaxillary gland is practically inactive and parotid gland only function (alkaligenic glands). Redeglutition: The process proceeds in the same way as that of deglutition. Due to higher specific gravity and additional reticular contraction which precedes the next regurgitation, the ruminated bolus enters the reticulum.

Functions of salivary gland secretions in ruminants:

Broadly, depending upon the type of secretion, the salivary glands of ruminants are divided into 1) Alkaligenic glands 2) Mucogenic glands.

Alkaligenic glands:

The paired parotid, inferior molar, buccal and unpaired palatine glands form, the alkaligenic glands. These glands secrete saliva containing high concentrations of bicarbonates with very little mucin. The secretion of alkaligenic glands is isotonic with the blood and has the following composition.

Cations: Na+ 180 mEq/l

K+ 20 mEq/l 190 mEq/l

Anions:

Cl– 13 mEq/l

HCO3– 100 to 140 mEq/l

HPO42– 10 to 50 mEq/l
The bicarbonate content of parotid saliva is 4 times as great as in bloodstream but Cl⁻ is only one fourth concentration.

Nitrogenous Constituents:

<table>
<thead>
<tr>
<th>Constituent</th>
<th>Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea Nitrogen</td>
<td>10 mEq/l</td>
</tr>
<tr>
<td>Protein Nitrogen</td>
<td>1.3 mEq/l</td>
</tr>
<tr>
<td>Total Nitrogen</td>
<td>12.4 mEq</td>
</tr>
</tbody>
</table>

Mucogenic glands:

The paired submaxillary, sublingual lateral glands and the unpaired pharyngeal glands form the main mucogenic glands. These glands secrete saliva which is rich in mucoprotein (Mucin) and very low in bicarbonate. The alkalogenic glands secrete almost continuously whereas mucogenic glands secrete only during feeding. The total amount of saliva secreted varies in ruminants, when compared to other animals and it ranges from 90-200 litres per day in cattle, sheep 6 to 15 litres per day and goats 5 to 8 litres per day.

In addition to its functions like moistening the food, facilitating mastication and swallowing, providing stimulus for taste receptors and assisting in thermoregulation, it has got four more important functions in ruminant.

1) Potassium and phosphates in saliva provides proper medium for bacterial growth.
2) The large quantities of bicarbonates present in saliva help in the neutralisation of the volatile fatty acids produced by the rumen bacteria and convert them into respective sodium salts of fatty acids. This helps in the maintenance of pH within normal limits in rumen.
3) The high content of mucin helps in the prevention of frothing of rumen contents and act as a antifrothing agent and prevents bloat.
4) Urea present in the saliva is utilised by the rumen bacteria for the synthesis of microbial protein which is subsequently utilised by the ruminant animal.

Functions of rumen microorganism

The principle feature of the digestive physiology in the ruminant animal is the microbial digestion and fermentation which occurs on a large scale in the rumen and reticulum. In fact, most of the ruminant disorders are more due to the end products of deranged bacterial and protozoal metabolism than the disorders in the host-system itself.

One of the important factor for the health of the ruminant animal is the regulation of rumen eco-system. The composition of the rumen microbial population varies due to many factors. The important ones are:

1) The kind of feed available  2) The amount of feed
3) the frequency of feeding and  4) host factors such as
   a) Composition and amount of saliva  b) the size of the rumen
   c) the rate of passage  d) and appetite,
These factors affect the kinds of rumen microbes and their activities. If these changes are rapid, they are accompanied by a change, which may prove disastrous to host animal. The microbial degradation is broadly classified into microbial degradation of a) carbohydrates b) proteins and c) fats.

**Microbial degradation of carbohydrates:**

The diet of ruminants consists of cellulose, hemicellulose, fructosans, xylosans, starch and fermentable sugars. The carbohydrates are degraded by extracellular as well as intracellular digestion. The microbes first attach themselves to the food particles and disintegrate or disaggregate them. Then by means of exoenzymes secreted by the microbes the polysaccharides are hydrolysed and converted into oligosaccharides (3 to 10 sugar molecules). The oligosaccharides are absorbed and first they are converted into monosaccharides. These sugars are utilised for energy purposes by Embden-Meyerhoff pathway and produce volatile fatty acids and excrete them into the rumen as metabolic waste products.

In aerobic bacteria and in other tissues where oxygen is available pyruvic acid enter the tricarboxylic acid cycle and get completely oxidised to CO₂ and H₂O whereas the rumen microbes being anaerobic, the pyruvic acid is converted into the following volatile fatty acids.

i) **ACETIC ACID:** (CH₃ COOH) Pyruvic acid is converted into acetic acid by two ways 1) Oxidative decarboxylation yielding acetate, carbodioxide and Hydrogen.

\[ 2 \text{CH}_3 \text{CO COOH} \rightarrow 2 \text{CH}_3 \text{CO COA} + 2 \text{CO}_2 + 2 \text{H}_2 \]

\[ 2 \text{CH}_3 \text{CO COA} \rightarrow 2 \text{CH}_3 \text{COOH} + 2 \text{ATP} \]

2) Phosphoroclastic split yielding acetate and formate. The formate is further metabolized into CO₂ and H₂. The hydrogen produced in the above two reactions are utilized to reduce Co2 to CH4 or for the production of succinate or propionate or lactate or butyrate or eliminated as hydrogen.

Pyruvic acid conversion to acetic acid gives the micro organism two more ATP.

ii) **Propionic acid:** (CH₃ CH₂ COOH). It is formed by two mechanisms.

1) The Co2 fixation to pyruvic acid forms oxaloacetic acid and through reduction it is converted into malate then to fumarate and succinate. Decarboxylation of succinic acid gives rise to propionic acid.

2) The second pathway is the reduction of pyruvate to lactate, acrylate and then to propionic acid.

iii) **Butyric acid:** (CH₃ CH₂ CH₂ COOH) Two moles of acetyl CoA condenses to form acetoacetyl CoA which through B-hydroxy butryl CoA, Crotonyl CoA, Butyryl CoA is converted to butyric acid.

iv) **C3 and C4,C5 branched chain volatile acids:** The valeric, isovaleric, isobutyric and D2 methyl butyric acids are formed from the deamination of the following acids:

Valine.......isobutyrate

Leucine.......isovalerate

Iso-leucine---D2 Methyl butyrate-n-valerate, arginine, lysine and proline are all degraded to delta amino valeric acid which on catabolism yields ammonia and valeric acid (These amino acids are essential for the normal growth of the rumen bacteria).
Nitrogen metabolism in ruminants:

The protein requirements of ruminants is met by the microbial protein synthesised by the microbes and undegraded protein which escapes ruminal degradation (dietary proteins which escapes microbial breakdown in the rumen). The diet of ruminants contain such as protein, aminoacids, ammonia, nitrate and urea etc. The following are the important events in the microbial protein metabolism.

i) The proteinases and peptidases present in the microorganism hydrolyse the protein to peptides and free aminoacids.

ii) The amino acids are either utilized by bacteria for synthesis of protein and other microbial cell constituents such as cell wall and nucleic acids.

iii) The amino acids are metabolized to volatile fatty acids, CO₂ and ammonia.

iv) The urea is hydrolysed to ammonia by urease activity of bacteria.

v) Compounds such as nitrates are reduced to ammonia.

vi) Ammonia is utilized for the synthesis of microbial cell components such as protein.

The digestion of proteins in the rumen proceeds by a steady rate of hydrolysis of peptides of decreasing chain length to free aminoacids by the proteinases and peptidases present in the microbial cells. Then the amino acids are degraded by fermentative deamination. The end products are CO₂, NH₃ and short chain fatty acids.

The amino acids are broken down by processes called 1) oxidative decarboxylation 2) stickland reaction.

The breakdown of amino acids are an important source of branched chain V.F.A. and C₅ and C₆ acids which are nutritionally essential for microbial growth and function.

Ammonia is the principle soluble nitrogenous constituents of the rumen fluid. Its concentration is influenced by the quantity and solubility of the dietary proteins, the quantity of urea that enters into rumen through saliva, diffusion through rumen wall and rate of absorption. The ammonia thus produced is utilized by the bacteria for protein synthesis except few species which directly incorporate aminoacids in their protein molecule. If the ammonia production is too rapid, the bacteria is not able to utilize them and becomes a waste. The efficient utilization of vegetable protein depends upon its solubility. Greater the solubility, greater is the rate of hydrolysis and ammonia formation and less efficiently utilized by the bacteria. Microbial protein synthesis depends upon the available energy and carbon skeleton derived from carbohydrate fermentation and (N) from the protein and NPN substances. If the amount of ammonia production is disproportionate to the available energy and carbon, the NH₃ will accumulate in the rumen in excess and cause disorders. Increased quantities of sugars and starches decrease the concentration of NH₃ and increase the speed of utilization.

Lipid metabolism in ruminants:

Metabolism of lipids by rumen micro-organisms are as follows:

1. Hydrolysis of esterified fatty acids.
2. Biohydrogenation of unsaturated fatty acids.
3. Lipid Biosynthesis in the rumen.
4. Metabolism of Phytal to phytanic acid in the rumen.
The triglycerides undergo rapid hydrolysis in the rumen. The rumen microbes produce two enzymes a cell bound esterase and a lipase. They are of extracellular in nature and they prefer diglycerides than triglycerides. The degradation of Galactolipids proceeds through successive deacylation and results in transformation of monogalactosyl-glycerases. The phospholipids are hydrolysed into respective fragments.

Biodehydrogenation of unsaturated fatty acids:

The unsaturated fatty acids such as linolenic acid and linoleic acids which are present in pasture grass are rapidly hydrogenated and yield cis-trans dienoic acid, cis-trans monoenoic acid and finally stearic acid. The products of biodehydrogenation in the rumen are absorbed in the ileum and any unsaturated fatty acids that escapes biodehydrogenation in the rumen are biodehydrogenated, in the caecum and colon. The bacteria involved in biodehydrogenation are Butyribrio fibrisolvens, Ruminococcus albus, Eubacterium etc.

Lipid biosynthesis:

Allison proved that Ruminococcus flavifaciens cellulolytic bacteria is able to synthesize C15 fatty acids from C14 acids. Acetate is able to donate C14 labelled material into 16:0 16:1 16:2 fatty acids. This showed acetate served as the prime molecule for the synthesis of fatty acids.

Phytal to phytanic acid:

The leaf tissue contains phytal. This occurs in the alcohol moiety or chlorophyll. This alcohol is oxidised to phytanic acid and incorporated into the rumen organism.

Glucose homeostasis in ruminants

The normal blood glucose concentration in ruminants range from 30 to 60 mg/100 ml. Very little glucose is absorbed from the alimentary tract of the ruminants and they mainly depend on liver for glucose. The glucose is required mainly. 1) for the oxidative requirements of the nervous system for energy purpose 2) It forms the base for the formation of NADPH and glycerol for the synthesis of fat, 3) formation of muscle glycogen. 4)development of the foetus and finally 5)for the synthesis of milk sugar lactose. A cow in milk requires about 1500g/day out of which more than 60% is utilized for milk sugar synthesis.

In ruminants the glucose requirement is mainly met by the following ways:

1) GLUCONEOGENESIS: Nearly 2/3 of glucose requirement of the ruminant animal is met by this process. The main source of substrate for this is proteins. Protein on hydrolysis yield several amino acids. These amino acids on further oxidation yields NH3 and keto acids. The keto acids in turn enters into the tricarboxylic acid cycle and yield glucose through the formation of oxaloacetic acid, (Alpha) - Ketoglutaric acid and pyruvic acids.

2) PROPIONIC ACID: Nearly 1/3 of glucose requirement of ruminant animal is met by the above volatile fatty acid. The other two volatile fatty acids namely acetic and butyric acids are ketogenic. The propionic acid formed in the rumen enters the liver where it is converted into glucose by the following way:

Propionic acid condenses with CoA and forms Propionyl CoA, which is converted into methyl melonyl CoA in the presence of vit B12. Succinyl CoA is formed from methyl melonyl CoA in the presence of Biotin. Succinyl CoA by way of TCA cycle forms oxaloacetate via fumurate and malate. Glucose is formed from oxaloacetate via phosphoenol Pyruvate and reversible glycolysis.
3) Glyceraldehyde: Only a small quantity of glucose is formed by glyceraldehyde. It is converted into phosphoglyceraldehyde→Glucose.

4) Lactic acid: small quantities of glucose is formed from lactic acid

Physiological basis of ketosis:

Derangement of glucose metabolism in high yielding cows causes a disease called Ketosis. At the time of peak yield of milk (6 to 8 weeks after parturition) there is a great demand for blood glucose for the formation of lactose. If the demand of glucose is not met, then animal will develop hypoglycemia and associated changes. When there is hypoglycemia, the animal body mechanism, tries to overcome it by increasing the speed of fat metabolism in liver with the formation of acetyl CoA. Normally the acetyl CoA enters the TCA cycle by combining with oxaloacetic acid and gets itself completely oxidised. Whereas in a ketotic cow due to depression of glucose metabolism enough Oxaloacetate is not formed. Hence, the acetyl CoA cannot enter TCA cycle and it is converted into ketone bodies. The liver cannot utilise ketone bodies and finds its way in the blood stream for utilisation in peripheral tissues. The tissues also utilise small quantities and excess accumulates in blood. When the threshold value exceed, it is excreted in urine and milk and causes the disease. So the primary cause of ketosis is Oxaloacetate deficiency. Sometimes it is said CoA deficiency also cause ketosis because CoA is necessary for the formation of oxaloacetate from pyruvic acid.

At the time of parturition and during peak lactation if the animal is put to excessive stress it may precipitate in ketosis. The stress causes the animal to secrete excess ACTH from anterior pituitary which in turn stimulates the adrenal cortex to produce excess glucocorticoids. The glucocorticoids by gluconeogenesis increases the blood glucose. If the stress is continuous, the adrenal cortex exhausts its capacity to synthesise glucocorticoids and the animal may become permanently ketotic.

If there is cobalt deficiency in the feed Vit. B12 synthesis interfered and this in turn interferes in the conversion of propionyl CoA to methylmalonyl CoA and formation of glucose from propionic acid.

Since the proteins are important for the ruminant blood glucose requirements, the feed must contain enough TDN to meet these energy needs at the time of peak lactation.

In ketosis the animal fail to maintain blood glucose level by the above mechanism and it overshoots in the direction of excessive fat mobilization and catabolism.

In a healthy ruminant animal which possess a normal rumen ecosystem the following end products are produced on diet consisting of carbohydrates, proteins, fat, minerals, electrolytes and water.

Rumen fluid parameters useful to clinician

- Total volatile fatty acids: The concentration of VFA in the rumen vary over very wide range, but the usual range is from 60 to 120 mEq/litre of rumen liquor.

- The proportion of acetic acid usually ranges from 60 to 70 percent, propionic 10 to 20 percent and butyric acid from 10 to 15 percent, branched chain isomers of C4 acids and straight and branched chain C5 acids are present in small quantities (isobutyric, isovaleric, 2 methyl butyric and n-valeric acids).

- Ammonia: The concentration of ammonia in the rumen vary greatly with the diet. The usual range of concentration for animals on mixed ration is 5 to 25 mg/100ml. The ammonia concentration will be lowest in animals fed with high starch ration and highest when animals are fed with young summer grass.
The pH of the rumen liquor is usually within the range of 5.2 to 7. After feeding, the pH decreases and the speed and extent to which it decreases depends upon the composition of the feed. The lowest pH is usually observed to 2 to 4 hours after feeding and during fasting it ranges towards neutrality. The other physiological parameters which help the clinicians to know whether the rumen functions normally are:

- **Osmotic Pressure**: Osmotic Pressure of rumen liquor is similar to blood. The freezing point depression of rumen liquor is between 0.46 and 0.695°C (sheep) 0.54 and 0.59°C (cattle).
- **Surface tension**: The normal surface tension of rumen fluid is 45 - 59 dynes per centimeter for bovines and 31.00 ± 3.00 dynes per centimeter.
- **Chloride content**: Normally ranges from 15-25 mEq/lit. It may increase when there is reflux of abomasal content (30 to 100 meq/l)
- **Total acidity**: Normal rumen total acidity is between 8 - 25 units. It may increase to 70 units in hyperacidity (lactic acid). The volume of 0.1N NaOH required to neutralise 10ml of rumen liquor multiplied by ten gives the clinical units.
- **Viscosity**: In normal animal the rumen liquor viscosity varies from 1.53 to 1.77 when fed diet containing concentrates. Whereas when fed with grass or hay alone it ranged from 1.12to1.300.

The bacteria and protozoa: The normal bacteria count ranges from 10^8 to 10^10/ml of rumen liquor. The protozoa count ranges from 10^5 to 10^6/ml of rumen liquor.

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MICROBIAL ECOSYSTEM IN THE RUMEN

Dr. A.T. Venugopalan

The rumen contains a mass of moist, often replenished plant material held at constant temperature in a medium devoid of oxygen. These features favour the growth of very large numbers of anaerobic protozoa, bacteria, fungi and viruses. Each microbe ferments some component of the digesta and together producing the microbial bodies and volatile fatty acids used as food by the hosts. The organic matrix of the cell wall of plants comprised of cellulose, hemicellulose, pectins, lignins and small amounts of bound protein.

In studying microbes associated with animals we are concerned with the nature of the microbe-host interaction and its magnitude. The primary task of the ecologist in approaching the microbial habitat in an animal is

i) to discover by direct observation the major microbes present

ii) to determine the major physicochemical factors in the environment.

At the first International Microbial Ecology symposium, Savage (1978) reported "for more animal types including human, the gastro-intestinal microbiology is not understood." The rumen of the domestic ruminants was an exception to the above statement. Fundamental aspects of rumen microbiology have recently been shown to have been overlooked (Bauchop, 1979).

Study of the rumen ecosystem possessed many advantages compared with the human hindgut. Uses of surgically modified animals permitted ready access to the rumen contents at different stages of digestion. The direct microscopical examination of materials of the rumen have been neglected for many years. The direct observation remain the cornerstone of any ecological study even in the ecology of micro-organisms. All possible tools of microscopic observation can be exploited, including the full range of techniques in light microscopy transmission and scanning electron microscopy. The scanning electron microscopic studies of rumen microbiota revealed extensive colonization of plant fragments by protozoa. The involvement of protozoa in physical degradation of plant tissues was also seen. In addition, adhesion of large population of anaerobic fungi to plant fragments in the rumen, stomach and hind gut of a wide range of herbivorous animals was seen. Scanning electron microscopy (SEM) has also been used to study adhesion of rumen bacteria to plant fragments in anaerobic cultures. All major colonization of plant fragments by gut microbes appears to occur at sites of physical damage or on internal tissues such as the inside surfaces of the hollow stems of grasses. Intact cutinized plant surfaces had relatively few micro-organisms present.

Importance of chewing in plant digestion

The extent of digestion of plant materials in the gut results from the combined effects of chewing, microbial fermentation, digestive enzymes of the animal and detrition of the gut. The importance of physical damage of plant material due to chewing in relation to microbial fermentation has been little studied even in ruminants fermentation. Examination of faecal plant fragments from animals with major hindgut fermentations (rabbit, horse and elephant) indicated that very little degradation of plant material occurred compared with that in ruminants. The food ingested by both groups is exposed to chewing. However, the extent of physical damage due to chewing could be one important difference between the two groups as only during rumination chewing is repeated and extended over long periods.
Anaerobic fungi of rumen

Large numbers of anaerobic phycomycetous fungi colonize plant fragments in the rumen of cattle and sheep feeding on fibrous diets. The magnitude of this rumen flora has so far been overlooked. The routine practice of microbiological studies of rumen is to strain rumen contents and discard the solid fraction. It is within the solids that the rumen anaerobic fungi are to be found. Highest populations were found with stalky fibrous diets. The quantity of fungi rhizoid tissues and their enzymatic activities holds the key to assessing the importance of these fungi in the rumen fermentation. The broad distribution of anaerobic fungi in widely differing herbivores, indicates that they are well adapted to the gut environment and suggest that they are important.

Treatment to improve cell wall degradation

Complex interactions occur between rumen microbial flora and plant cells during the process of degradation. Variations in the adherent microbial populations exist with different forages. Treatments to improve quality will result in marked changes in the structures of cell wall which is often attached more readily by unattached nearby bacteria.

Conclusion:

The symbolic fermentation by the myriads of rumen microorganisms permit the ruminants to utilize forages not digested by other mammals. The best postulate to explain the evolution of the rumen microbial symbiosis is that it has conferred a survival advantage on the host, able to utilize the otherwise indigestable food components. The highly organized structure of plant materials imposes spatial relationships on microbial colonization. In the rumen ecosystem understanding of these relationship is required for the integration of the accumulated knowledge of the microbia involved in plant digestion. Microbial ecologists can look forward to existing discoveries in the gut ecosystem.

REFERENCES:


Existence of Rumen protozoa was known since mid 19th century. Two types of protozoa are seen in the rumen (i) majority of them are ciliates and (ii) few are flagellates.

Rumen ciliates are comparatively larger in size to flagellates, have cilia on the entire body or restricted to anterior part as tufts. Two nuclei- a macronucleus and a small micronucleus are present. The latter will be difficult to see in a living ciliate. Contractile vacuoles are seen. Skeletal plates and concretion vacuoles may or may not be present. Ciliates multiply asexually by transverse binary fission and sexually by conjugation.

Important rumen ciliates are

1. *Isotricha sp*  
   About 120 μm; cilia all over the body surface, with rows of cilia parallel to the body axis.

2. *Dasytricha sp*  
   About 100 μm; cilia all over, but rows of cilia run obliquely to body axis.

3. *Entodinium sp.*  
   Cilia restricted only to the adoral area as tuft.

4. *Ophryoscolex sp.*  
   Cilia restricted to adoral and left side of the body and without operculum. Spines at the posterior part of the body.

5. *Epidinium sp.*  
   Similar to ophryoscolex but has two contractile vacuoles.

   Body ovoid with operculum at the centre of the anterior end.

7. *Eodinium sp.*  
   With operculum at the anterior end and a single wide skeletal plate occupying nearly half of the body surface.

8. *Ostracodinium sp.*  
   With single slender skeletal plate. Macronucleus hooked.

   With single slender skeletal plate and rod shaped macronucleus.

10. *Eremoplastron sp.*  
    With two slender skeletal plates and rod shaped macronucleus.

11. *Diploplastron sp.*  
    With two large skeletal plate and E or F shaped macronucleus.

    With four skeletal plates.

    Large size with five skeletal plates.

Flagellates encountered in the rumen are

   They are seen in young ruminants before the establishment of ciliates and also in ruminants in which ciliates were lost due to some reasons.

Young animals develop ciliates in their rumen through contact, grooming, licking, and saliva of adult ruminants.

All ciliates produce large amount of starch like reserve polysaccharides, some rapidly hydrolyse a variety of different proteins and produce ammonia and liberate amino acids and peptides. Rumen protozoa are capable of hydrogenating unsaturated fatty acids.
Rumen protozoa are digested by the host and give as much as 1/5 of its total animal protein. Nearly 00g of protozoa has been used per day by a ruminant.

Protozoa utilise chiefly bacteria to obtain their protein supply. Hence the bacteria - protozoa, - mammal food chain. Though protein of ciliate is less, it is of superior quality and give better growth.

Rumen ciliates can be seperated from ruminal content, stained and counted.

Whenever digestion in the forestomach become upset, larger species of infusoria disappear first followed by medium and finally smaller species.

Disturbance of microbes upset ruminal function leading to disorders which affect animal productivity. Noculation of ruminal cud is the best treatment for ruminal disorders than the conventional medication.

Faunated lambs gain weight rapidly than protozoan free lambs. Digestibility of dry matter protein, fibres and organic matter were higher in faunated lambs.

Animal performance improves in faunated animals and greater digestibility accompanied by higher levels of ruminal volatile fatty acids occur, indicating more complete ruminal digestion.

REFERENCES


There is a continued need for agricultural research to seek improved efficiency of production and within the livestock sector much of the research effort is directed towards nutrition, since feed is a major cost of production. In case of the ruminant, such feeds include forage, whether fresh or conserved forages, as well as compound feeds. Because of the high costs of feeds any increase in the efficiency with which an animal converts these into milk or meat can have substantial effect on the overall economic efficiency of the production system.

The ruminant poses a particularly interesting challenge to anyone involved in livestock nutrition. The microbial population of its rumen allows to utilize large quantities of forages, which are unsuitable for inclusion in rations for non-ruminant species. In addition, these microbes can synthesize substantial quantities of protein from non-protein nitrogen. As such the ruminant does not compete, directly with man and non-ruminant species of farm livestock for much of its feed requirements.

The feed requirements of dairy cattle can be divided into that needed for:

1. Maintenance
2. Gain in body weight
3. Reproduction
4. Lactation or milk synthesis.

As we are interested in milk production, it directly involves the changes that may take place in the rumen environment and fermentation process, physiological and biochemical changes that may take place inside the rumen. Before going to the subject proper, we must know broadly the composition of milk and its major constituents and synthesis etc.

Composition of milk from different species

<table>
<thead>
<tr>
<th>Species</th>
<th>Fat %</th>
<th>Protein %</th>
<th>Lactose</th>
<th>Ash % (Total solids %)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ayshire</td>
<td>4.1</td>
<td>3.6</td>
<td>4.7</td>
<td>0.713.1</td>
</tr>
<tr>
<td>Brown Swiss</td>
<td>4.0</td>
<td>3.6</td>
<td>5.0</td>
<td>0.713.3</td>
</tr>
<tr>
<td>Guernsey</td>
<td>5.0</td>
<td>3.1</td>
<td>4.9</td>
<td>0.714.4</td>
</tr>
<tr>
<td>Holstein</td>
<td>3.5</td>
<td>3.1</td>
<td>4.9</td>
<td>0.712.2</td>
</tr>
<tr>
<td>Jersey</td>
<td>5.5</td>
<td>3.9</td>
<td>4.9</td>
<td>0.715.0</td>
</tr>
<tr>
<td>Zebu</td>
<td>4.9</td>
<td>3.9</td>
<td>5.1</td>
<td>0.814.7</td>
</tr>
</tbody>
</table>

Milk contains three characteristic components, viz., fat, protein and lactose. The quantities of these and other components of milk vary among species, although the composition is affected by many genetic and environmental factors.
Among the major constituents certain constituents are either synthesized in the acinar cells or filtered based on the permeability which explains the interior structure of the bovine mammary gland.

**Milk secretion process:**

The constituents of milk are produced by the epithelial cells in two ways. One group of compounds, which includes milk fat, most of the protein components, and lactose is synthesized in the epithelial cells from blood precursors and then released into the lumen of the alveolus. The remaining milk constituents pass from the blood across the epithelial cells or between them into the alveolar lumina without alteration by the cells. In some cases, they are bound to other compounds, but synthesis does not take place within the cells.

Milk is in osmotic equilibrium with the blood flowing through the udder throughout the period and milk remains in the udder and not just during its formation. Changes in milk composition occur as water moves in and out of the udder in response to changes in the osmotic pressure of blood. The volume of water secreted in the milk is thus changed to maintain osmotic equilibrium between the blood and milk. Since lactose accounts for a large part of osmotic pressure in milk, an increase in its secretion rate causes an increase in water movement into milk and consequently lactose plays a major role in governing the rate of milk secretion.

**Blood precursors of milk constituents:**

The rate of milk secretion is partially dependent upon the availability of blood precursor that flow through the mammary gland. This in turn depends upon the rate of blood flow through the udder and the composition and uptake of the blood constituents by the mammary gland.

The blood flow: milk yield ratio also increases with advancing lactation and with a drop in yield due to illness.

**Blood precursors of the milk constituents in the ruminant:**

<table>
<thead>
<tr>
<th>Milk constituent</th>
<th>Blood precursor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>Water</td>
</tr>
<tr>
<td>Lactose</td>
<td>Glucose</td>
</tr>
<tr>
<td>Protein</td>
<td>Amino acids</td>
</tr>
<tr>
<td>Casein</td>
<td>Blood serum albumin</td>
</tr>
<tr>
<td>Lactoglobulin</td>
<td>Immuno globulins</td>
</tr>
<tr>
<td>Lactalbumin</td>
<td></td>
</tr>
<tr>
<td>Milk serum albumin</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat</td>
<td>Acetate, B. hydroxybutyrate, blood lipids</td>
</tr>
<tr>
<td>Fatty acids</td>
<td>Glucose, glycerol from triglycerides</td>
</tr>
<tr>
<td>Glycerol</td>
<td>Minerals</td>
</tr>
<tr>
<td></td>
<td>Vitamins</td>
</tr>
<tr>
<td>Minerals</td>
<td></td>
</tr>
<tr>
<td>Vitamins</td>
<td></td>
</tr>
<tr>
<td>Water, vitamins &amp; minerals</td>
<td>Transferred from blood plasma to milk</td>
</tr>
<tr>
<td>Lactose</td>
<td>Synthesized from glucose</td>
</tr>
<tr>
<td>Casein, B. lactoglobulin, lactalbumin</td>
<td>Amino acids</td>
</tr>
<tr>
<td>Milk serum albumin, immuno globulins</td>
<td>Transferred from blood</td>
</tr>
</tbody>
</table>
Milk Protein Composition

Milk protein is made up of a number of specific proteins. Among them the major components are casein, -lactalbumin, and β-lactoglobulin. Each major component is broken down into subunits based upon their molecular weight, isoelectric point, and other chemical properties. The chemical composition of some of the components of the protein is determined by the genetic make up of the animals. It is now possible to relate the variants of the milk protein to the blood type of the animal.

Milk Fat Composition:

Milk fat is the most variable component of milk. This is true for the percentage composition of the fat and fatty acid composition of the triglycerides within and among species. Most of the milk fat is made up of triglycerides. The major precursors for milk lipids are glucose, acetate, B Hydroxybutyrate and triglycerides of the chylomicron and the low density lipoproteins from the blood. The short chain fatty acids from C4 to C14 palmitic acid are synthesized within the mammary gland from acetate derived as absorbed acetate in the ruminant or from glucose in the non-ruminant animal.

About 30% of the palmitic acid is from acetate and the remainder from the triglycerides in the blood. Stearic & oleic acid is from plasma triglycerides and stearic can also be converted to oleic. In bovine, glucose can supply 70% of the milk lipid glycerol.

Direct effects of Rumen VFA on milk composition

<table>
<thead>
<tr>
<th>VFA</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetic acid</td>
<td>Increase in milk yield with major milk constituents, specific increases in milk fat content, tendency to decrease in milk protein content.</td>
</tr>
<tr>
<td>Propionic acid</td>
<td>Fat content is depressed, protein content increased</td>
</tr>
<tr>
<td>Butyric acid</td>
<td>Fat content is increased.</td>
</tr>
</tbody>
</table>

Nutritional influences on milk quality:

1. Decreasing the amount of particle size of dietary roughage and increasing the amount of soluble carbohydrate may depress milk fat content and increase that of milk protein. These changes are dependent on an alteration in fermentation pattern characterised by an increase in the proportion of propionic acid and a decrease in that of acetic and butyric acids and possibly other associated changes in digestion.

2. Carbohydrate accounts for some 60-80% of the dry matter of the diet of cattle, and is present as structural materials (cellulose, hemi-cellulose), as storage material (hexosans, pentosans) and as simple sugars. The amount of dietary carbohydrates of the physical and chemical characteristics affect both the fat and protein contents of milk.

3. High concentrated diet and high proportion of readily soluble carbohydrates reduce particle size and depress milk fat.

1. Extreme depression 3-4 other symptoms are reported - It depends on individual cow and within breed and it is referred as low fat syndrome which can be seen even in a single diet.

2. Decreased fat content - Change in fat composition, unsaturated acids increased, saturate

3. Decreased (C18:1) (this is again due to change in pattern of fermentation).
4. Typical composition of short chain fatty acids in a high fibre-low concentrate is as follows.

<table>
<thead>
<tr>
<th>Acetic acid</th>
<th>60-70%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Propionic acid</td>
<td>17-20%</td>
</tr>
<tr>
<td>Butyric acid</td>
<td>7-12%</td>
</tr>
</tbody>
</table>

A gradual reduction of fibre content of the diet by substitution of starch concentrate leads to propionic acid (40-45). On other occasion, the reduction in acetic acid is associated initially with an increase in the proportion of propionic acid and a decrease in the proportion of butyric acid. This reduction in milk fat is related to the increase in the ratio of C3/C2 acids in rumen liquor. Sharp (1974) showed 69 variation in milk fat which is accounted for by variation in the major proportion of the 3 major short chain acids, as determined by multiple regression analysis. Linear regression analysis showed that 60 of the variation is due to propionic acid 52 due to butyric acid and 44 due to acetic acid.

4. Changes in carbohydrate constituents in the diet that depress milk fat content increases milk protein content and there is proportionate increase in all the major milk protein (case in B. lactoglobulin & Lactalbumin).

5. Underfeeding causes a slight depression in lactose content of milk (Rowland, 1948) and more marked changes are observed during starvation (Smith Howat & Ran, 1931, 1960) especially intermittent stages. This mainly depends on associated changes in milk yield, but mainly from a decrease in the relative contribution of lactose to the osmotic pressure of milk as the volume of secretion is depressed. The normal lactational trend in lactose content demonstrates this relationship and the effects of underfeeding are especially provisioned with cows in advanced lactation (Dawson & Rock 1972).

6. Depression in milk protein content following the incorporation of a wide variety of fats in the diet have been reported but no mechanism has been proposed.

7. In addition to the nature of diet and physiological factors, the milk yield and its constituent composition may change due to change in the pattern of rumen fermentation because of certain metabolic diseases.
The climatic environment influences nearly every economic aspect of animal agriculture, its yield and composition, animal growth, reproduction, milk production and the efficiency of conversion of foodstuffs to economic units. Climate is a combination of elements that includes temperature, humidity, rainfall, air movement, radiation, barometric pressure and ionisation. The universe is divided into different climate regions according to latitude, temperature and wind. As per latitude the world is classified into five zones, the torrid, two temperate and frigid zones.

**Torrid (Tropical)**

Bounded by North and South by the tropics of Cancer and capricorn.

**Two temperate:**

The two zones between the tropics and the Arctic and Antarctic circles.

**Two Frigid Zone:**

These are the two polar Zones the arctic and antarctic.

Koppen has classified the world on the basis of temperature as follows:

1) Tropical belt: All months hot (over 18°F) 20. N to 16.S latitude.
2) Sub - tropical belt: 4-11 months hot over 68°F and 1-8 months temperate
3) Temperate belt: 4-12 months hot over 50°-68°F)
4) Cold belts: 1-4 month temperate and then below 50°F
5) Polar belts: All months cold

**Climate of India:**

The various factors controlling the climate (viz. latitude, altitude (ography), land and water distribution and air currents) play important role in producing a wide variety of climate in the subcontinent of India.

Latitude of the country extends from 8.N to about 38° N i.e. well before the tropic of cancer. The seasons are classified as (January - February), winter; (March-May) Hot weather period; (June- September) South-West monsoon; (October - December) post-monsoon period.

**Climate of Tamil Nadu**

There are two types of climate. They are the "macro climate" and micro climate. Macro climate is the outdoor climate and it is controlled by climatic factors. The micro climate is the indoor climate or the climate immediately surrounding the animal. This climate is the one which deeply affects the maintenance of health, productivity and reproducitvity of animals.
To achieve near optimum ratio in cattle or buffaloes, we would like a climatic environment having an air temperature of 13-11°C, a relative humidity of 60-70%, a wind velocity of 6-8 km/h, and a medium level of solar radiation similar to that of occurring in the tropical latitude in spring and fall seasons. There should also be a fertile soil with adequate and evenly distributed rainfall to produce crops of high quality in plentiful supply and finally, the environment should be free from diseases and parasites (Mc Dowell, 1973).

**Literature review:**

No matter which type of production is under consideration, whether it is growth, fattening, reproduction or milk production, there obviously must be some overall effects due to extremes in temperature and humidity. Nutrition and ambient temperature-humidity are major factors in animal environment. Some of the ruminal parameters influenced by the climatic environment in cattle are pH, oxidation reduction potential volatile fatty acids, lactic acids and ammonia. Hence, informations concerning the interaction of diet and temperature humidity should be useful in the feeding management of cattle in different climate.

Climate exerts extremely complex influences on humoral and cellular immunity but this remains a field for much research (Hyslop, 1974). Heidrich et al. (1967) reported that the occurrence of mastitis associated with C. pyogenes is commonly appearing in summer months.

Mc Dowell (1972) indicated that high environment temperature promote liquidation and depression of water soluble carbohydrate, minerals and vitamin in forage crops which are responsible for milk fever, Downer cow-syndrome and ketosis in ruminants.

Rumen oxidation reduction potential (ORP) is a measure of microbial activity in the rumen. The normal ORP in vitro fermentation for healthy animal was 400 mV ± 15 to 13 mV (Broberg 1957). Hogen (1954) reported in sheep that the highest ruminal NH3 concentrations were seen under those conditions of climate and pasture growth conducive to rapid feed intake. Voluntary food intake of cattle falls with rising environment temperature. Misra et al. (1983) found that water intake in the buffaloes were reduced above an ambient temperature of 32°C.

Alleve (1961) demonstrated that in buffaloes there was a decline of abomasal secretion together with a decrease of acidity as the environmental temperature rose, until finally free hydrochloric acid disappeared and the secretion lost its digestive activity. Secretion was restored after, by moving the animals into a cooler environment: the most effective way of restoring the secretion was to cool the buffaloes under a shower.

Alleve (1961) further reported that in buffaloes in a hot dry environment, 28-44°C, the contraction of smooth muscle in all parts of digestive tract become weak and infrequent the reticulum and omasum being more affected than the rumen. Passage of digesta from the rumen was depressed and cooling the animals under a shower soon led to restoration of function.

Scott and Moody (1960) reported that there is evidence that in hot environment a ration with a low fibre content has a beneficial effect on the animals thermal balance and on its production.

Teighton and Rupal (1960) observed that in warm weather a ration high in protein and low in fibre tend to keep body temperature, respiratory and heart rates low and milk yield high.

The experiment conducted at U.S.A. at the University of Missouri to investigate the effect of climatic environment on the feed intake capacity and other ruminal Parameters indicated the following results.

1) Generally the cows in hot environment manifested the signs of heat stress in the way of an increase in body temperature, pulse and respiration rates. Their feed intake was depressed by about 40%.

2) The various ruminal parameters like pH, ORP, VFA, lactic acid and ammonia etc., which in turn are influenced by the ambient temperature-humidity, diet and their interaction are highly significant.
Thus from the above literature review it appears that more research needs to be conducted to find out the environmental physiology of ruminants.

**Future:**

Further research is needed to document the diseases in cattle and buffaloes due to climatic stress which is not well documented at present in India.

With increasing intensification of animal husbandry, the housing, microclimate in relation to production diseases need more intensified research.

In tropical countries, the optimum climatic condition needed for various animal production has not been worked out thoroughly, hence elaborate research is needed to fix up the optimum environmental condition.

"Environmental profiling work" have to be started for whole of India on relation with livestock production.

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ACIDOSIS IN RUMINANTS

Dr. P. DHANAPALAN

Introduction

In ruminants, excessive ingestion of feeds which are rich in readily available carbohydrates leads to the condition called 'ACIDOSIS'. Though the 'ACIDOSIS' in ruminants often mean the condition caused by excessive production of lactic acid (grain engorgement of ruminants, founder, over-eating, impaction of rumen, rumen overload), it should also equally be remembered that the present concept of classification of acidosis leads to the term "RUMEN ACIDOSIS COMPLEX" (Dirksen, 1983) which also comprises other pathologic changes in the acid-base status of rumen contents, such as the low milk fat syndrome, atypical ketosis, cerebrocortical necrosis (molasses toxicity), ruminitis - liver abscess-complex, acute laminitis and latent hydrochloric acidosis of the reticulo- ruminal contents. However, this article focusses attention mainly on acute carbohydrate engorgement of ruminants.

Etiology

In Tamilnadu and other parts of India, nowadays it has become a routine practice to rapidly increase the concentrate portion of the feed in order to have them on a high-milk -yield: (or) even an all concentrate diet as soon as possible. System of feeding like this results in the frequent occurrence of a problem commonly referred as "ACIDOSIS", thus meaning the syndrome in ruminants brought about by the excessive ingestion of feeds which are rich in readily available carbohydrates, such as starch and sugar (Elam, 1976).

Ruminants not accustomed to grain diet, suffer from acute digestive disturbances (Hungate, 1952) and often death may occur in many cases within 24 hours, after the consumption of large quantities of grain. This, condition is frequently encountered in sheep, goat and cattle. Often, the accidental excess consumption of grains, inexperienced feeding schedule with sweets like payasam etc. during festival seasons form the causal factors for acidosis in ruminants. Depending on the topographical areas, engorgement of apples, grapes, flour, brewer's grain, sugar beet, baker's dough, bread etc. form the causal factors for acidosis (Blood et al. - 1983).

Schedule of feeding also leads to the change in acid-base status of the body system. When increasing the concentration of grain in the ration from one level to another, if the increment is too high, the total amount of grain consumed by some cattle will be excessive. A hungry cattle that had sudden access to grains adlibitum, get affected by acidosis. Without a proper period of adjustment, if a high-level grain ration is fed to ruminant, it leads to acidosis.

Pathogenesis of acidosis:

In acute ruminal lactic acidosis ie. the most severe form, of fermentative indigestion, within 2-6 hours, there is a marked change in the microbial population in the rumen. High availability of bacteria that are rapid fermenters produce lactic acid as an end product (Gerry, 1990) ie. there is a marked increase in the number of streptococcus bovis which utilizes the carbohydrates to produce large quantities of lactic acid. The rumen fluid pH is 6.2 to 7.2 normally. Now, because of the production of excess lactic acid, the rumen pH gets lowered to 5 or less, at which point the cellulolytic bacteria and protozoa are destroyed. Both D and L forms of the acids are produced and the 'L' isomer is utilised much more rapidly than D - isomer which accumulates and causes severe lactic acidosis. Dunlop and Hemmond (1965) suggested the term "D-lactic acidosis" rather than as simple 'acidosis'.

D - LAC TIC ACIDOSIS
Inhibition of ruminal activity may be due to lactic acid entering the duodenum and exerting a reflex inhibitory action on the rumen and the diarrhoea is often due to the reduction in the net absorption of water from the colon. In ruminant acidosis, the compensatory mechanisms are over-stretched, and the hydrogen ion concentration in blood increases. i.e. plasma pH falls. Among the many compensatory mechanisms, chemical buffering is particularly important, esp. the bicarbonate system in plasma, because its constituents are subjected to both renal and respiratory perfusion and hence to the glomerular filtration rate, ultimately leading to anuria and intermittently animal may experience shock and death.

In uncomplicated chemical ruminitis, there is sloughing of rumen mucosa and healing with scar tissue and some mucosal regeneration. Hence there is a chance for the spread of bacteria like *Sphaerophorus necrophorus* and *Corynebacterium pyogenes* into the damaged mucosa of the rumen and thereby into liver esp. in conditions involving the hepatic abscesses.

Histamine gets absorbed well from the intestinal loops and laminitis may occur in acidosis affected cattle. Further fungal species like Mucor and Rhizopus, may multiply, thus invading the ruminal vessels and causing the thrombosis and infarction. Further, the detectable changes in the cellular and biochemical composition of the CSF suggests that blood - brain barrier may also be affected.

The changes in the rumen fluid and blood are summarized as follows:-

1. Change of rumen fluid pH from a normal of 6.2 - 7.2 to 5 or below 5.
2. Normally Gram negative bacteria prevails more in rumen but in acidosis, Gram positive lactobacilli prevail.
3. Rumen fluid is watery than normal and has a milky grey colour (Dirksen, 1980) and has a pungent sour smell and shows fast sedimentation of the solid particles and there is almost absence of flotation of solid particles. The methylene blue reduction time is greatly prolonged in the range around pH 5.0
   Absence of protozoa in the sample should be taken as an important criteria than pH of the sample especially during collection of sample, 2 - 3 days after occurrence (Gerry 1990)
4. There is an increase in the osmolality of ruminal contents, thus leading to high osmotic pressure and subcutaneous oedema.
5. There is a decline in standard bicarbonate and blood pH and increase in organic phosphate, glucose and pyruvate in the serum. The clear haemoconcentration is evidenced with augmented values in total serum protein, hemoglobin, hematocrit, urea and residual nitrogen.
6. Toxins other than lactic acid such as histamine, tyramine and tryptamine may be absorbed which contribute to the clinical picture.

Clinical signs:-

Depending on the time of presentation of the patient after the consumption of excess grains, the exhibition of clinical signs vary. Clinician should identify the animal whether it needs emergency approach or a routine approach. Severe acidosis usually exhibits signs like Kussmaul breathing (rapid and deep) and cardiovascular disturbances. It should always be remembered that in ruminal acidosis, the clinical signs may be obvious before the systemic acidosis reaches full severity (Mullen, 1976)

Mildly affected animals show transient inappetance, depression, suspended rumen activity, greyish green rumen fluid with reduction in milk yield.

In severely affected animals, complete inappetence, sudden drop in milk yield, muscular tremors, grinding of teeth, unrest, colic symptoms, watery - foamy diarrhoea, reluctance to move, unsteady and
staggering gait and at times profound lameness due to laminitis occur. Often recumbency follows after about 48 hours. Some animals appear to make a temporary improvement but become severely ill again on the 3rd and 4th day.

Clinical pathology and diagnosis:

Dehydration together with rise in hematocrit from a normal of 30 - 32% to 50 - 60% in terminal stages occur. Anuria with fall in blood pH, plasma bicarbonate, rumen fluid pH occurs with predominance of Gram positive bacteria in the rumen.

Diagnosis is made from history, clinical signs, examination of rumen fluid, estimation of sodium bicarbonate in plasma, and this condition is to be differentiated from milk fever mainly, in which dehydration is not a marked feature and history of calving further helps to diagnose this.

Treatment:

The primary purpose of effective clinical approach is 1) to correct the ruminal and systemic acidosis. 2) to prevent the further production of lactic acid 3) to restore the forestomach motility.

The treatment procedures are summarized as follows:

1) Administration of fluids and alkalinizers  
2) Transfusation with rumen cud  
3) Antibiotics, Antihistamine and vitamins.  
4) Antacids administration  
5) Managemental aspects  
6) Surgical means

Fluids: (including NaHCO3) and alkalinizers:

Depending on the degree of dehydration, several litres of physiological saline solution in the form of continuous drip are administered partly by I/V and partly by S/C routes. The saline solution is supplemented by adding 13 gm sodium bicarbonate per liter, thus giving sodium bicarbonate in sufficient amount during the 1st 12 hours, depending on severity.

If over dosage of buffers is suspected, go in for S/C or oral dosing.

Blood et al (1983) stated that systemic acidosis is treated by 5% I/V NaHCO3 at the rate of 5 litres for a 450 kg animal, given initially over a period of 30 mts will usually correct the systemic acidosis, this is to be followed by isotonic NaHCO3 (1.3% at rate of 150ml/kg. B.W., given over the next 6 - 12 hrs.) Michell (1990) stated the formula of calculation of deficit of sodium bicarbonate as follows:

Deficit of plasma bicarbonate (in mmol/litr) x BW (in kg) x 0.2 (factor) mmol / kg is the deficit throughout the extra-cellular fluid.
Depending on the severity and hepatic damage, Ringer's lactate or Ringer's acetate solutions are selected for fluid therapy.

Davis (1981) stated that rapid administration (or overdosage with sodium bicarbonate) may lead to extra cellular fluids hyperosmolality, CSF acidosis and intra-cranial haemorrhage. Delirium, depression and coma represents clinically the CSF acidosis. If Ringer's lactate is to be used, it must be preceded by another buffer (NaHCO₃) that will correct the acid-induced depression (hepatic lactate metabolism).

In the acute stages, liver protective agents such as amino acid, vit B12 are recommended.

**Administration of antacids:**

100 - 500 gm Na HCO₃, Magnesium Hydroxide and/or 1 to 1.2 kg Baker's (or) brewer's yeast in 10 litre of warm water are pumped into rumen and follow it by kneading of the rumen to promote mixing.

**Managemental aspects:**

Restrict water for 12 - 24 hours but provide straw. (Blood et al., 1983) Limit the water supply because already they might have taken more water. All animals should be exercised every hour for 12 - 24 hours to encourage movement of the ingesta through the digestive tract.

**Surgical means:**

With severe acidosis, the therapy depends on what stage the animal is brought to the veterinarian.

In the early stages of indigestion with acute overloading of the rumen, both rumenotomy and removal of the rumen contents with subsequent rinsing of the rumen is the best and the safest methods.

After rumenotomy-with subsequent removal of ruminal acidotic contents, appropriate degree of ruminal filling should also be established so as to stimulate the forestomach motility. Firm, inspissated material in the ventral ruminal sac which develops during prolonged ruminal stasis can often be softened by administering 1 - 2 gallons of mineral oil and then kneading the left flank vigorously. Cathartic agents like Magnesium sulphate (0.5 to 1 g/kg) may also promote the passage of abnormal ruminal contents.

An alternative to rumenotomy is rumen lavage. Glucose infusion and glucocorticoid injections are not indicated in acidosis cases.

**Rumen cud transplantation**

In the advanced stages of the disease that is in cases where liquefaction of the rumen content has already occurred, it is recommended to withdraw through a tube as much rumen fluid as possible. Subsequently antibiotics or yeast upto 2 Kg (or) the two combined with ample water may be administered through the naso gastric tube. Antacids also have proved successful at this stage.

Repeated inoculation of rumen contents from healthy animals is indicated to replenish the normal rumen flora. Gerry (1990) suggested that inoculation with 1 liter of rumen fluid is appropriate for calves, while a minimum of 3 litres is required for adult cattle and 8 to 16 litres is desirable. It is always best to transfer the rumen fluid from the donor to recipient immediately, but if the rumen fluid contains active and healthy microflora, it will remain viable for upto 9 hours in room temperature (or) upto 24 hours under refrigeration.
Antibiotic

If no rumenotomy is opted, orally administer 10 to 20 gm chlortetracycline in conjunction with upto 50 litre water, the latter given in several doses distributed over 24 hours. Administration of antibiotics should perhaps be repeated after 6 to 8 hours.

Antihistamines:

Should be given to acidosis cases.

Others:

2 - 4 gm thiamine dichloride (Vitamin B1).

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OMASAL DISORDERS

Dr. R.V. SURESH and Dr. V. GNANAPRAKASAM

Omasum is located within the rib cage and is practically inaccessible to routine clinical examination such as palpation, percussion and auscultation. Evidence of disorders of this organ can be obtained only by performing exploratory laparotomy and rumenotomy (Rosenberger, 1979) and at autopsy (Blood et al., 1983).

The projection field of the omasum is on the right side between the 7th and 9th ribs on level with caudal edge of the lung field at anterior limit. The area is in contact with diaphragm and the chest wall varies from 8-24 cm.

Firm pressure palpation under the lower part of the right costal arch or in the area of the 7th to 9th right intercostal spaces with knuckles may evoke pain response. Swinging palpation might detect the rebound of an abnormally hard omasum as during impaction. Pole test as that of reticulum reveals enlarged or indurated omasum in the xiphoid region with grunting. Pain percussion give clearer results than deep palpation.

Acoustic percussion of the omasum on the right side between the 7th and 9th ribs gives damped sound, but difficult to distinguish from the response, with organ like lung, liver, abomasum and intestine. When the omasum is abnormally enlarged there is an increase in the area of dullness and the intensity of damping; these characteristics are diminished when the omasum is smaller than normal or displaced.

Auscultation of omasum is done with the aid of phonendoscope, on the right side at the 9th intercostal space on a level with shoulder joint. It will give a rustling sound. Normally, auscultation at the centre of the omasal field may not succeed in picking up the rustling sound made by omasal contraction, which is synchronous with reticular contraction, because of louder sounds made by other stomach compartments.

Omasum puncture is performed with a needle 15-18 cm long, inserted into the 9th intercostal space on a level with the shoulder joint, to a depth of 10-15 cm. The free end of the needle usually describes an irregular rotating movement, which is diminished or absent during disorders of omasal motility.

Omasal contents can be extracted during rumenotomy by inserting two or three fingers through the rumino-reticular orifice and carefully detaching the material lying between the laminae of the omasum. Normally, the contents are dark brown or green, almost dry and crumble. Coarse undigested food, pasty or fluid masses are abnormal. Determination of the dry matter content is of diagnostic importance. Dry matter content is normally 15.33%. Abnormal dryness of omasum is 37.7%.

Primary disease of omasum is rare. Blood et al. (1983) considered omasal impaction to be a doubtful entity, diagnosed at autopsy. Davies (1965) successfully diagnosed a case of omasal impaction and corrected by surgery.

Omasal impaction has been reported by several authors throughout the world (Swarbrick, and willins 1967; Blampied, et al., 1964; Albert and Ramey, 1965 and Blood et al., 1983). Omasal paresis, omasal blood, omasal dilatation, omasitis with fenestration of the omasal leaves, torsion of omasum, perforation of omasum, omasal distension, obstruction of reticulo-omasal orifice, spasm of omasum are the other diseases of omasum.

Primary disorders of omasum occurs secondary to other conditions of digestive disorders like intestinal invagination, botulism, bovine viral diarrhoea and haemoglobinuria.
Omasal impaction

The most commonly reported disease of omasum, chronic omasal impaction is difficult to diagnose. It usually confirmed at autopsy. When omasum is enlarged and excessively hard, and it is likely to cause death a primary cause. It has been reported to occur in animals along with other disease. Omasal impaction also occurs when feed is rough fibrous particularly alfalfa stalk, loppings from fodder trees, in drought condition and sheep fed on the ground, the impaction is due to accumulation of soil in omasum.

Chronic recurrent bouts of indigestion occur and are manifested by decreased rumen motility, infrequent scanty faeces, refusal to eat grain and a negative ketone test (McDonald and Witzel, 1968). Pain may be elicited and the hard distended viscus palpated or deep pressure under the right costal arch or in the 7th to 9th tercostal spaces on the right side.

More acute form of omasal atony in cattle occur in which atony may be primary or secondary other diseases like post parturient haemoglobinuria, low nutritional level and parturient paresis Lamplie et al., 1964; Albert and Ramey, 1965 (Swarbrick and Willis 1967 and Blood et al., 1983).

The omasum is grossly distended, the leaves or laminae show patches of necrosis and are associated with peritonitis. Necrosis of the ruminal lining may also be present (Blood et al., 1983).

Clinical manifestation

Complete anorexia, cessation of defecation, an empty rectum, subacute abdominal pain and disinclination to move or lie down. Sometimes recurrent stomach cough suggest that it is a carrier of pleuro-pneumonia (Blood et al., 1983).

Physical Examination: Pressure palpation by using the knuckles on the right side between 7th and 9th ribs may reveal abnormal sensitivity; swinging palpation might detect the rebound of an abnormally hard masum during omasal impaction. Pain percussion of the right side between 7th and 9th ribs with pain seeking hammer gives clear results than by deep palpation (Rosenberger, 1979).

Acoustic percussion, over the enlarged omasum, will reveal increased area of dullness.

Therapy

1. Treat the primary cause.
2. Saline purgative, stimulants as for rumen impactions
3. Large dose of liquid paraffin
4. Symptomatic

Prevention

1. Correct the deficiency status
2. Phosphorus supplementation 1 gm/gallon of water, urea, molasses, salt, universal lick containing 8.6% phosphoric acid.
3. Avoid dry, coarse fibrous feeds without water for a long time
4. Treat the primary disease in time.
Autopsy: Swarbrick and Wilkins (1967) observed that the omasum was very hard, heavy and enlarged. The sulcus was filled with tightly packed fibrous ingesta. The material between the leaves of the omasum was dry and hard. The laminae appeared to be abnormally thin, black in colour, very friable, necrotic but no histopathological abnormality was detected. Omasal laminae were dry and pale. Omasitis and fenestration of omasal leaves. Leaves and walls of omasum show patches of necrosis and are associated with peritonitis. Slight increase in keratinised cells. Scars were found in the folds of omasum. Break in continuity of epithelium. Infiltration of phagocytic cells into the surrounding tissues.

2. Omasal paresis

Dirksen (1981) stated that the ventral vagal trunk after it leaves the ventral aspect of the oesophagus it ramifies in the area of the reticulum, omasum, abomasum, liver and diaphragm. This region is referred as the "storm center of the abdominal cavity" and is susceptible to inflammation and is usually affected in reticulo-peritonitis due to various causes.

The involvement of this nerve may produce functional disturbances depending on which parts of ventral vagus are disrupted. It may result in anterior functional stenosis (at reticulo omasal opening) or posterior functional stenosis (at pylorus).

Functional stenosis between reticulum and omasum is called as anterior functional stenosis.

(a) With atony of the reticulum and rumen due to both vagi disrupted.
(b) With maintained or enhanced motor activity of the rumen and reticulum or possibly atony of the reticulum.

Functional stenosis at the level to the pylorus is called as posterior functional stenosis.

(a) Permanent: with or without atony of the reticulum.
(b) Periodically relapsing.

Anterior functional stenosis simply expressed results from paralysis of the omasum. In this case, the withdrawal of digesta from the reticulum ceases. Both form of functional stenosis, display a relatively uniform clinical picture. The history of such patients usually reveals, a duration of the disease of several weeks with progressive inappetance, emaciation and dehydration. The history will further reveal recurrent bloat, progressive distention of the abdomen, changes in the consistency and amount of the faeces.

On general examination, the appearance of the abdominal circumference observed from behind displays a characteristic outline. The whole left side including the paralumbar fossa is distended while the abdominal wall on the right side protrudes only in the lower part. The animal exhibits ventral curvature of the spinal cord (Lordosis). The animal displays dullness, its body temperature is normal or subnormal. The skin will be dry and inelastic. The haircoat will be dull and roughy. The respiration rate may be increased, normal or markedly decreased. The visible mucous membranes will be pale:

Rectal examination of affected animals reveals distension and overload of the rumen. Because an impediment of cructation is present at the same time, chronic recurrent tympany of rumen develops. The rumen activity is increased, rumen contraction are superficial and on auscultation, splashing and gurgling sounds will be heard instead of normal typical crackling sounds.

The rumen contents is foamy and dark green in colour. The faeces are scanty formed and covered with mucous.
Auscultation of omasum will reveal fluid sounds instead of normal rustling in anterior functional stenosis.

In case of posterior functional stenosis, that is pyloric stenosis, both impaction and distension of the abomasum occur first. Posterior functional stenosis usually possible to diagnose only two or three days later, at a time, when secondary dilatation of the omasum and rumen or additionally reticulo-omasal stenosis and back flow of abomasal contents have developed. Functional stenosis can be detected by following method.

**By colour solution method:** By administering the patient with coloured solution in form of medicinal charcoal with 1 to 2 litres of 10% sodium sulphate or sodium carbonate solution in order to induce the oesophageal groove reflex. After 2 to 3 minutes, the abomasum is punctured to test, whether the coloured fluid has passed into the abomasum. If the coloured fluid has reached and able to detect in abomasum then the possibility of anterior functional stenosis can be ruled out.

**Atropine test:** Injection of atropine to cattle will paralyse the vagus. In cases with vagus bradycardia the heart rate is less and after atropine sulphate injection will increase the heart rate. Bradycardia is noticed in functional stenosis due to lesion in the vagus. Bradycardia is also noticed in conditions like abomasal displacement, torsion of caecum and other painful condition of the abdominal cavity. Differentiation between these conditions can be made by injection of atropine sulphate 30-40 mg. subcutaneously. There will be 16% or more increase in heart rate in functional stenosis. The atropine test in cases of doubt may give valuable indication of whether or not functional stenosis is present.

**Laparotomy:** This will not only reveal the type of vagus lesion but also the underlying cause. With anterior functional stenosis, the rumen and reticulum will be distended and filled with digesta mixed with foam. Both the omasum and abomasum are small in size and empty. The reticulo-omasal orifice is widely open and there will be no tension on palpation. In posterior functional stenosis (Pyloric Stenosis) the omasum and abomasum will be distended and filled with firm digesta. The rumen may also be dilated but not to an appreciable size.

3. Omusal bloat:

Omasal bloat is extremely rare. Gas filled omasum is dilated and tense.

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DISEASES OF THE ABOMASUM ASSOCIATED WITH CURRENT FEEDING PRACTICES

Dr. R. Venkataraman

Abomasum, the fourth stomach, being the true digestive organ of ruminants, any inflammatory condition affecting it is usually proceeded by derangement of the other three forestomachs. Since the forestomachs were also affected to greater or lesser degree, abomasitis cannot be classified as primary or secondary.

Abomasum occupies most of the hypochondric region and lies on the floor of the abdomen near the median plane. The fundus or the anterior fluid sac of the abomasum is located in the xiphoid region in close proximity to the reticulum. The pylorus joins the duodenum at the ventral aspect of the right ninth or tenth rib. Because of the anatomical position the direct examination of the abomasum is limited.

In bualtric practice, disease conditions of abomasum are encountered like abomasal displacement, abomasal torsion, abomasal impaction and abomasitis (Abomasal ulceration).

The abomasal portion on displacement are not very commonly encountered because of the feeding practices in our country where the grain feeding is not common. However, abomasitis and abomasal impaction are commonly encountered. Diagnosis of abomasitis is usually difficult and so knowledge about the diseases of abomasum is incomplete.

The various factors involved in the causation of abomasitis are exposure to chip coarse and irritant pastures, mouldly and fermenting feed, sudden change of feed, over feeding with bran, household garbage leaves and accidental ingestion of sand. The current feeding practice of increasing the concentrate in the ration and also eating more silage may result in abomasitis. Season also play a role in the incidence of the disease. More humid climates, rainy season and winter months will predispose the disease conditions. Stress factors due to faulty management practices like overcrowding, lack of exercise and contaminated feed and water also predispose the disease condition. Diagnosis of the condition may be attempted based on the latest technology in clinical diagnostic methods and laboratory analysis.

The abnormal functions of the abomasum are suspected only by the history in relation to pattern of feed intake ie rejecting concentrate and feeding roughage, and taking water. The clinical symptoms observed are grinding of teeth, semisolid dung, presence of occult blood, reduction in the milk yield and inappetance. Though the clinical observations can be taken as cardinal signs in the diagnosis, the clinical materials like abomasal fluid and blood must be examined to get more information. As per the statement of Wells and Halsted-"A physician who depends on the laboratory to make his diagnosis is probably in experienced, one who says that he does not need a laboratory is uninformed. In either instance, the patient is in danger".

ABOMASOCENTESIS will be helpful for collecting the abomasal fluid directly for diagnostic purposes. It will be also useful for infusing drugs directly into the abomasum to avoid the possible delayed action, dilution of the drug, possible breakdown and absorption of the drugs in between. It may be also useful to provide quicker onset of action of drugs possibly with a lower dosage which will be economical to the patient owner.
The site for the abomasocentesis is three centimeters to the right of the intersection of the longitudinal line connecting the xiphoid cartilage and the umbilicus and the vertical line connecting the middle of the tenth rib and the midventral line. A needle of approx 16G is used for the same to collect the abomasal fluid. The normal pH becomes more acidic and the pH may range between 2 and 3. The pepsinogen, chloride and potassium content of the abomasum is found increased. Likewise the blood chemistry reveals increased pepsinogen and decreased chloride and potassium levels which will give an indication of abomatous. In addition there will be evidence of dehydration. The normal levels of the various parameters are:

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Treatment of the condition may be tried by making use of Kaolin and activated charcoal. A combination of 100gms of kaolin and 50 gms of activated charcoal in 200ml of water may be infused directly into the abomasum for 2 to 3 days.
The cardiologists often find heart disease such as valvular insufficiencies long before there is heart failure, and the urologist may find kidney disease such as glomerular nephritis long before there is renal failure. Unfortunately there are usually no clinical signs of liver disease until there is failure of one or more of the liver's functions, since, the liver has so many functions, there are many possible signs of liver disease depending some what on which functions are failing to meet the animal's need.

Diagnosis of liver disease is similar to that of any other disease, three elements are paramount, a history, a thorough physical examination and appropriate laboratory tests. Clinical diagnosis of Liver disease in cattle is difficult because:

1. There may not be specific symptoms (like jaundice and tenderness within liver percussion field).
2. Liver function tests will fail to reveal the disorder as long as 1/3rd of liver parenchyma is functioning and there is no impairment of bile flow.
3. Metabolism of bile pigments is quite different in cattle from man and other domestic animals.
4. Liver function test results are not of greater value when taken individually, their overall evaluation is of value.
5. Results of these tests vary according to the nature, degree and duration of the liver disorder.

History:

1. Important when there is evidence of exposure to some liver toxins.
   - Pyrrolizidine alkaloid containing plants
   - The iatrogenic exposure
   - Mouldy feed
   - Clay pigeons
   - Tar paper
2. Grazing on wet ground infested with snails
3. Previous illness
4. Recent inoculation
5. Weight loss
6. Location of the animal
7. Excess energy intake (late lactation/nonlactation period)
8. Clinical signs
Physical examination:

Inspection

Palpation

Percussion

Observation:

Mucous membrane

- Evidence of Jaundice

Skin

- Photosensitization

- Exceptional tendency to bleed.

Palpation:

- Normally not palpable

- Healthy liver palpable when severe pulmonary emphysema/
  Some other swelling in thoracic/ abdominal cavity

- Enlarged liver is palpable dorsally behind the right costal arch.

- Enormously enlarged excessively displaced liver palpable rectally.

Percussion:

- Complete dullness 3 - 4 finger wide and length of the palm of the hand

- Displaced forward by 1-3 finger in advanced pregnancy and abdominal swelling

- Displaced backwards by 1-3 finger in pulmonary emphysema

- Enlargement of liver percussion field to 5 fingers width is pathological if sensitive to pain.

- Sub-tympanic on percussion
  Liver enlarged greatly
  Gall bladder enlarged
  Right side displacement of abomasum
  Healthy liver displaced by pneumoperitoneum

Very sensitive to acoustic percussion - Acute liver damage

If gall bladder inflamed acutely, centre of pain is cranioventral to the liver percussion field between central and ventral thirds of costal arch.

Reaction to pain percussion is less evident or absent in chronic liver disease.

Clinical signs of Hepatic insufficiency depends on the functions of the liver and the pathological conditions due to loss of function.
Functions of liver

1. Secretion of bile
2. Protein metabolism
3. Carbohydrate metabolism
4. Fat metabolism
5. Erythropoiesis
6. Iron metabolism
7. Detoxification
8. Vitamin metabolism and storage

pathological condition due to loss of function

Jaundice, Photosensitisation
Hypoproteinaemia, Edema
Hypoglycaemia, irritability
Enteritis
Anemia
Toxaemias
Pyrexia
Renal failure
Bleeding

The clinical signs of icterus (or) jaundice develops when the yellow pigment bilirubin accumulates in plasma and other tissues. Bilirubin is derived primarily from hemoglobin during the normal removal of aged erythrocytes by reticulo-endothelial cells. In the plasma, bilirubin is in unconjugated form, bound to albumin. Hepatocytes remove the unconjugated pigment and form bilirubin diglucuronide and other water soluble conjugates which are excreted in the bile. Over production of bilirubin is responsible for the icterus observed in acute haemolytic anaemia. Defective excretion of bilirubin occurs either in hepatocellular dysfunction (or) in obstruction of the biliary tract.

In sheep and cattle dying of terminal hepatic insufficiency, there usually is a significant clinical elevation in plasma bilirubin but the value may not be high enough to produce clinical icterus. This is owing either to the apparent reserve for bilirubin excretion (or) degradation, clinical icterus when present in ruminant, is most frequently associated with haemolytic anaemia eg. Anaplasmosis (cattle) copper poisoning (sheep).

Encephalopathy

In cattle, the syndrome of hepatic encephalopathy typically has an abrupt onset in actually a terminal manifestation of chronic cirrhosis. Clinical signs were similar to primary diseases of brain. Animals would be dull, stand apart from other animals, charging other animals, unrestrained bellowing. Progressive dysmetria and ataxia, tenesmus with prolapse of rectal mucosa etc. The causes of the liver encephalopathy were :

1. Increased Ammonia level in blood and CSF
2. Urea cycle enzyme deficiency
3. Mercaptans production
4. Increased short chain fatty acids level in circulation
5. High concentration of aromatic amino acid
6. Increased Neurotransmitters
7. Impaired hepatic metabolism.
Ascites:

When associated with liver disease, ascites is indicative of a chronic process and one which almost always is characterised by cirrhosis. Ascites has been reported in association with liver Abscess in cattle where thrombosis of the hepatic vein adjacent to the abscess causes marked hepato megaly. It is important to differentiate between action caused by liver from other primary diseases. High protein in ascitic fluid is indicative of liver pathology (2 to 2.5 g.dl). However liver can be implicated for the cause of ascites only after further investigations.

PATHOGENESIS OF ASCITES:

Whenever there is hepatic injury:

1. There will be reduction in plasma protein leading to reduced colloidal osmotic pressure leading to Ascites.
2. Hormones are not inactivated leading to increase in reabsorption of sodium chloride and water leading to ascites.
3. There will be reduction in hydrostatic pressure in portal, hepatic veins and sinusoids by regenerating nodules, causing disturbances in the portal circulation leading onto hepatic dysfunction, hepatic insufficiency leading on to Ascites.
4. Similarly carcinoma of Liver and heavy liver fluke infestation can also cause disturbances in portal circulation leading on to ascites.

Photosensitisation:

Photosensitisation in ruminants results from intake of a variety of plants, algae, Fungi and drugs and may be primary (or) hepatogenous in nature. In primary photosensitisation the photodynamic agent or its precursor is present in feed (or) water; following ingestion and absorption, sensitisation of the skin occurs with subsequent development of dermatitis on exposure to sunlight. In hepatogenous photosensitisation, liver damage results in reduced biliary excretion of the photodynamic pigment phylloerythrin a normal metabolite of chlorophyll.

Liver function tests:

A wide range of tests have been described to help the clinician to decide whether the liver is involved in the disease of a particular patient and to differentiate between different types of hepatic dysfunction. Laboratory tests by themselves will very rarely give a diagnosis, and frequently will not give a useful initial guide, but can be used very effectively in the majority of cases to distinguish between the two (or) three possibilities which remain after a thorough clinical examination. The tests used in liver disease can be broadly divided into those assessing impaired functional capacity and those detecting damage.

Liver function tests routinely used:

1. Arginase
2. Alanine aminotransferase (ALT/GPT)
3. Aspartate aminotransferase (AST), (GOT)
4. Glutamate dehydrogenase (GLDH)
5. Gamma glutamyl transferase (GGT)
6. Isocitrate dehydrogenase (ICDG)
7. Lactate dehydrogenase (LDH)
8. Malate dehydrogenase (MDH)
9. Ornithine carbamyl transferase (OCT)
10. Sorbitol dehydrogenase (SDH)
11. Blood ammonia concentration

Currently used liver function tests which are having recent advances:
12. Serum bilirubin
13. Serum alkaline phosphates (SAP) activity
14. Serum bile acids (SBA)
15. Sulfobromophthalein and Indocyanine green (BSP) & (ICG)
16. Galacto elimination capacity (GEC)
17. Tests to assess coagulation disorders
18. Lipoproteins

New procedures in liver function tests
19. Procollagen type III peptide (PIIIP)
20. Ligandin
21. Propionate loading test in ruminants
22. Aminopyrine breath test
23. Serum gunase test
24. Serum antimitochondrial antibodies
25. Serum antinuclear antibodies.
27. Serum hyaluronate levels
28. Plasma and salivary caffeine levels
29. 5-Nucleotidase
30. Plasma kallikrein levels
31. Plasma prolidase index
General guidelines for liver function tests:

These tests detect liver disease based on assessment of function potentials of the organ.

They have been designed either to quantify the liver derived substrate in blood (or) they measure or exogenous compound.

Appropriate group of tests should be selected according to the situation under investigation.

Enzyme specificity and duration of illness are important.

Liver damage may occur in association with or as a result of damage in the other organs which themselves alter the enzyme levels.

The half-life of SDG, GLDH and arginase is short.

In horses, starvation and illness unassociated with liver damage will increase bilirubin level and ratio of free to conjugated.

Bilirubin is not a guide to jaundice.

Alkaline phosphatase will increase when corticosteroids and anticonvulsants are given.
CALFHOOD DISEASES AND THEIR MANAGEMENT

BY DR. R. BALASUBRAMANIAN.

One of the most important aspects in livestock production is minimising the economic loss by early identification of calfhood diseases and their management since it is the healthy calves that are going to be the nuclei for adult cattle and their production. Therefore the importance of the same need not be over emphasised.

In urban areas and metropolitan cities, the calves particularly the male calves are simply allowed to die of starvation and neglect which in tum could be attributed to various factors like economy, lack of space for housing of calves and other managemental defects. If this trend is allowed to be continued, then there might be a stage where in, finding healthy calves will be a rarity which will adversely affect the livestock improvement and production programmes. With these few points in mind, the salient points are given pertaining to calfhood diseases and their management. However, before proceeding, it is very important that new born calves should have access to good quality colostrum soon after they are born since it is absolutely necessary in protecting the calves against early calfhood diseases.

Postnatal diseases are classified into three group viz:

<table>
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<th>Delayed postnatal diseases</th>
<th>Late postnatal diseases</th>
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<td>(within 48 hrs of birth)</td>
<td>(2 days to 7 days)</td>
<td>(1 wk to 4 hrs)</td>
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<tr>
<td>eg. 1. Malnutrition due to poor mothering</td>
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<td>e.g. white muscle disease</td>
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<tr>
<td>2. Exposure to cold (hypothermia)</td>
<td>2. Mammary incompetence and consequent starvation,</td>
<td>Enterotoxaemia</td>
</tr>
<tr>
<td>3. Low vigour due to malnutrition</td>
<td>3. Hypogammaglobulinaemia (susceptible to infection like colibacillosis)</td>
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</tr>
<tr>
<td>4. Specific diseases (navel ill, Colibacillosis)</td>
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Calf mortality rate of 20% can reduce the net profit by 38%. In well managed herds calf mortality usually does not exceed 5% from birth to 30 days of age. In certain cases expansion programme in a herd is not commensurate with calf rearing facilities and labour. Haphazard planning, over crowding, the failure to feed colostrum early enough, labour shortages etc contribute to calfhood diseases.

Causes of malnutrition among calves: During pregnancy malnutrition of dams, inclement weather, old animals with unsatisfactory milk flow, poor maternal behaviour prolonged parturition.

Causes for inadequate mothering: Malnutrition - lack of warmth and protection.
Causes for reduced vigour of calves:

- Calves with poor birth weight
- Malnutrition.
- Intrauterine and postnatal infections.
- Extreme weather conditions (cold/hot).
- Multiple births (being one).
- Predators like: Crows, rats and bandicoots.

The above neonatal malnutrition and predation could be averted by taking appropriate measures.

Neonatal infection in cattle due to:

- Escherichia coli
- Listeria monocytogenes
- Pasteurella sp
- Streptococci/Salmonella sp
- Enteritis caused by enterotoxigenic E. Coli, salmonella sp
- Rotavirus & Coronavirus
- Cryptosporidia sp
- Clostridium perfringens type A, B & C
- Resp. tract disease caused by Infectious broncho rhinotracheitis.
- Persistent viremia with Bovine virus diarrhea virus and leukosis caused by virus of Bovine viral leukosis.

Epidemiology

If the infection is intrauterine, then infection through placenta (existing endometritis). In such cases, control measures should be taken accordingly i.e. isolation of affected aborted animals, proper disposal of placenta and fetus and strict sanitation and hygiene and early identification of the causative agent for the same.

If the disease is postnatal then infection is through navel or ingestion.

Infection through contamination of environment - soiling of udder, bedding by infected uterine discharge or from the discharge of other infected newborn animals.

In the case of Intrauterine infection:

1. Causative agent can be detected from uterine exudate of dam and in foetus at birth.
2. Clinical signs are observed at birth.
3. Intrauterine infection are usually associated with abortion (or) death of foetus (or) death of dam.

Exception: It is stated that infection of bovine foetus between 45 days and 125 days of gestation with bovine virus diarrhea virus can result in persistent viremia, immunotolerance and birth of clinically normal calf which may develop foetal mucosal diseases at 6-8 months of age or older.

Resistance to infection in newborn calves:

i) Calves are born agammaglobulinaemic and virtually not resistance to infection till they have ingested colostrum early enough and absorbed sufficient quantities of lactoglobulin from colostrum.
ii) Immune system is less mature in calves than in adults and therefore does not respond effectively to antigen.

iii) If the antibody level is high in precolostral serum then it denotes intrauterine infection. (For diagnostic purpose it is useful), and so also presence of immunoglobulins and specific antibodies in aborted fetuses (useful in diagnosis of abortion in cattle).

iv) In infectious diseases of intestines and respiratory tract in calves - morbidity and mortality rate higher with low levels of serum immunoglobulins.

v) Other factors for lowered resistance: High levels of corticosteroids result in lymphopenia. Therefore decreased phagocytic defence (Note: Cortisol and cortisone concentration is high in new born calves and decline by 15 days of life).

Transfer of Passive immunity in calves: In calves, absorption of colostrum though continues up to 24 hrs., maximum absorption occurs with in 6 - 8 hrs after birth. Absorption of colostral immunoglobulin is not specific since other protein like serum globulin and albumin are also absorbed. However, it is important to note that absorption of immunoglobulin in calves is directly proportional to the concentration of colostral immunoglobulins. Successive parturition increases the level of immunoglobulins in cattle. In certain cases, like udder oedema, premilking is practised 3 to 9 days before parturition. It is to be noted, premilking reduces the level of immunoglobulin and consequently this can result in increase in morbidity and mortality rate.

Colostral immunoglobulin present in the intestine can prevent enteric disease. Circulating immunoglobulins are necessary for protection against septicemia but they do not prevent diarrhoea. Colostrum fed to calves soon after birth provides protection against colisepticaemia but does not prevent diarrhoea of enteric colibacillosis. Colostrum provides serum immunoglobulins which prevent or minimize invasion of bacteria and provide mechanical protection against invading organisms by saturating the macromolecular transport system of the intestine.

Vitamin A: Carotene breakdown - Vitamin A in alcohol form and placenta is not permeable for this whereas Vitamin A in Cod - liver - oil - it is permeable.

In colostrum deprived calves, there is low leukocytic count. Therefore less phagocytic activity and consequently more prone to infection.

Pathogenesis: Readers are impressed upon, that each and every specific infection due to causative agent mentioned earlier is not dealt separately in as much as they produce bacteremia, septicemia or viremia as the case may be and therefore only the general principles applicable are mentioned, which otherwise will become voluminous and another text.

Infection in neonates either causes septicemia with severe systemic reaction (or) bacteraemia with few or no systemic signs followed by localisation in various organs.

If portal of entry is navel - causes ‘Navel ill’

Extension to liver (or) via Urachus to bladder resulting in chronic ill health or systemic septicemia.

In new born infection - localisation in joints which may either be non-suppurative or suppurative.

Less common - Pan ophthalmitis

Valvular endocarditis.

meningitis.
Dehydration and electrolyte imbalance can be expected irrespective of diarrhoea or not probably due to deprivation of fluid as much as to loss of fluid.

**Clinical Signs**

a) When slow spreading

   Umbilical cord inflammation (omphalophlebitis) fever, depression, anorexia and signs referable to localisation like
   i) Endocarditis (with cardiac murmur)
   ii) Panophthalmitis - (pus in the anterior chamber of eyes)
   iii) meningitis - (rigidity, pain and convulsion)
   iv) Polyarthritis - (with lameness-swollen joints - may rupture with discharge of pus)

b) When spread of infection is rapid

   Clinical signs of septicaemia like:
   - fever
   - prostration
   - coma
   - petechiated
   - dehydration
   - acidosis with rapid death.

**Clinical Pathology:**

**Serum:**

i) Precolostral Blood

   From still births to neonates before ingestion of colostrum (when and if in utero infection suspected)

ii) Post colostral blood

   To assess the level of serum immunoglobulin obtained from colostrum after ingestion. Should be done 24 to 48 Hrs after birth and most reliable. This will determine whether the calf has ingested sufficient colostrum or not early enough i.e. with in 6 - 8 Hrs of birth. This could be used as a monitoring technique where

   a) Large number of calves purchased for rearing
   b) to investigate the cause of high rate of morbidity and mortality in newborn population in a herd.
   c) in individual calves - useful for assessing prognosis and whether gammaglobulin therapy is needed or not.

**Note:** Immunoglobulins are more prophylactic than curative.

**Suitable methods used for evaluation of Serum immunoglobulins:**

1. **Refractometer:**

   Used under field conditions

   - 24 Dairy calves (Home reared)
   - 32 Market calves (purchased)
   - 24 (single suckled calves)

   **Refractometer values (g/D1):**

   - 4.80 ± 0.16
   - 5.08 ± 0.09
   - 6.06 ± 0.23

**Note:** The above values are only guidelines. However, it is advised to have "Norms" for our local calves including Buffalo calves under the prevailing conditions in respective areas.
2. Zinc Sulphate Turbidity Test (ZST)

Interpretation of ZST:

i) 0-5 - Little or no absorption of immunoglobulins and no protection.
ii) 6-15 - Inadequate absorption and only minimal protection.
iii) 16-20 - Improved absorption and protection.
iv) above 20 - Adequate absorption and good protection.

Limitation of ZST:

1. Requires more time and facilities
2. Haemolysed blood samples - will give artificial high readings
3. In dehydrated animals - elevated readings due to haemoconcentration.

Emphasis:

1. These tests are most valuable to assess immunoglobulin status of animals before they become ill.
2. An excellent aid in epidemiological investigation to study whether neonates are receiving sufficient good quality colostrum early enough or not.

3. Single Radial diffusion test is also used.

For accurate measurement: Paper electrophoresis, single radial diffusion test.

Note: Modus operandi of these tests could be had from relevant books.

Diagnosis: Early enough investigation and PM and identification of the etiological agent is essential.

Treatment and Management:

1. At the outset, find out the etiological agent if possible
2. Drug sensitivity test and start treatment - ideal but not practicable.
3. Tentatively choose antibiotic/therapeutic agent according to previous experience.
4. The drug metabolizing enzyme activity is lower in new born calves (upto 6 wks)
5. Decreased metabolism can affect the duration of action of lipid soluble drugs.
6. Functional immaturity of kidneys decreases the renal excretion of certain drugs and metabolites.
   (eg) Half life of chloramphenicol in new born calves is virtually double that of adult cattle.
7. Individual treatment necessary for maximum survival rate (since no satisfactory mass method of medication or water supply for suckling ailing animals).
8. Supportive fluid therapy.
9. Provision of antibodies to sick & weak new born calves through blood transfusion or hyperimmune serum, especially where immunoglobulin status is unknown. Whole Blood @ 10 to 20 ml/kg body wt I/V in neonatal diarrhoea with shock. Serum, or plasma - Half the above dose.

Note: Blood to be taken from

a) oldest adult in the herd.
b) Not from animal nearing parturition (since there is drain of gammaglobulin to mammary gland).

Principles of control and prevention of infectious diseases of Newborn:

1. Removal of the etiological agent from environment of the newborn.
2. Isolation of newborn from infectious environment.
3. Non specific resistance of newborn calves should be increased and maintained.
4. Increasing the specific resistance of newborn through vaccines wherever indicated and practicable.

Note:

Buffalo Calves:

1. Early calf mortality in buffalo calves due to Ascariasis is not uncommon (because of transplacental infection). Deworming with suitable safe anthelmintic on day one could be advised to minimise economic loss.
2. While following the methods suggested it is advisable to evaluate and have separate norms for buffalo calves.
GASTRO-INTESTINAL EMERGENCIES IN RUMINANTS

Dr. S. AJITH KUMAR

Disease conditions causing acute abdominal pain in ruminants need emergency attention and treatment. Acute abdominal pain will be expressed by clinical symptoms like paddling of legs, kicking of belly, grunting, grinding of teeth (Bruxism) and rolling on the ground.

Causes of abdominal pain

1. Inflammatory conditions/lesions of gastro-intestinal tract e.g. Acute abomasitis, Abomasal Ulceration.

2. Mechanical interference involving both the lumen and the blood supply of a portion of the gut. e.g.: Torsion of abomasum, intestinal Obstruction.

3. Referred abdominal pain due to affections of organs other than Gastro Intestinal tract. Knowledge of this will help in the differential diagnosis. e.g.: Torsion of Uterus/rupture of uterus, cystitis, Acute retention of urine.

Differential diagnosis of different disease conditions causing acute abdominal pain

1. Ruminal impaction

   Doughy consistency or ruminal mass on external palpation and on rectal examination.

2. Acute bloat (Tympany)

   Pass a probang through the mouth. Froth may comes in frothy bloat without relieving the abdominal distension. Gas and abdominal distension can be easily relieved in free gas bloat due to failure of the eructation mechanism (e.g. Choke)

3. Acute Lactic acidosis

   History of overfeeding with easily digestible carbohydrates or fruits.

4. Ascariasis in Calves

   Diagnosis can be made by finding the characteristic parasitic egg during the faecal sample examination. After treatment with piperazine derivatives in calves with ascariasisis, drenching of water should not be done for 8 hours, since the water will wash away the medicine from the already detached worms, causing clumping of worms, intestinal obstruction and death of the animal.

5. Rumenitis

   History of ingestion of any foreign bodies/garbage/excess Carbohydrates/ mouldly feed. Enlarged lymphnodes can be palpated over left paralumbar fossa.

6. Acute abomasitis

   Pasty dung, grinding of teeth, reluctant to eat concentrates.

7. Abomasal Ulcerations

   Sudden onset of anorexia, grinding of teeth, reduction in milk production.
Sip water continuously, reluctant to eat straw. Pyrexia may be noticed in abomasal ulcerations associated with systemic infections like theileriasis, Rinderpest etc. Soft to fluid dung, occasional black tarry coloured dung (malena) associated with the shedding of blood from the ulcers. Anaemia during complete blood examination. Pain on right side behind the Xiphoid cartilage in ulcers cause perforation and peritonitis. Sand particles causing abomasitis/ulcers can be felt by deep palpation. Occult blood examination of the dung can be carried out if melena is not prominent.

8. Left side abomasal displacement

Palpation in the left para lumbar fossa may reveal a 'NOTHINGNESS' between the body wall and the rumen.

Abomasum may be palpated in the upper left para lumbar fossa immediately behind the rib as a semicircular dome in severe cases.

Auscultation over the upper left ribcase may reveal ping sounds (like dropping of coin to a glass full of water)

Paracentesis of abdomen at left para lumbar fossa with a sterilized needle - Large quantities of blood tinged fluid with pH 2-4 (Lip Tak Test) faeces scanty, soft and dark.

9. Dilatation and right side displacement and torsion of abomasum

Abomasal torsion along with right sided displacement can cause acute obstruction of the alimentary tract.

Distented abomasum may be detectable on palpation immediately behind and below the right costal arch. Percussion reveals fluid splashing sound. Auscultation ping sound or high pitched tympanitic sound on the right side. Rectal examination reveals distended abomasum in the right lower quadrant of the abdomen. In torsion, distended tense resilient viscus is palpable. Faeces scant, soft and dark in colour. Rupture of abomasum may occur and cause sudden death. Paracentesis of the distended abomasum will reveal large quantities of blood-tinged fluid with pH 2-4.

10. Omasal impaction

Due to ingestion of dirt and sand

Inco-ordination, blindness, mania and death. Abdominal palpation on right side using knuckles or the ball of the hand or swinging palpation may help to detect the rebound of an abnormally hard omasum.

11. Traumatic reticulo peritonitis (hard-ware disease)

Arched back, reluctant to move, when walking down hill there will be grunting, sudden drop of milk, capacious appetite - eating greedily, suddenly ceases eating followed by grunting. Reduced appetite to grain, recurrent bloat.

Rare cases attack of acute abdominal pain with kicking of the belly and rolling.

Some cases become recumbent and unable to get up. Body temperature and pulse rate increased. Blood examination reveal leukocytosis, neutrophilia with shift to left especially in early stages. Various methods to elicit pain reaction such as back grip, pole-test can be done. Pain on left side behind Xiphoid Cartilage.

Williams method of reticular ausculation - grunt or absence of normal reticular sounds.
12. Diaphragmatic hernia

Recurrent tympany

Cardia auscultation: *grunt can be auscultated while passing the stomach tube through Cardia region.*

Intestinal sounds can be auscultated in thorax confirmed by radiography.

13. Acute intestinal obstruction

Tenesmus can be noticed. Rectal examination reveals mere traces or total absence of faeces in the rectum. Presence of yellowish or greyish white, occasionally reddish to brown-black mucus plug. Foul smelling rectal contents.

14. Gut tie or pelvic hernia

Usually occur in castrated young animals. Legs held further back than normal - Stretched out stance. Ballooned gut with restricting cord on rectal palpation.

15. Diseases of the liver

Dissection of the liver with increased tension of the capsule and lesions of the capsule will cause abdominal pain. Acute swelling of liver occur in congestive heart failure and acute inflammation. Neoplastic lesions or abscesses of liver can also cause abdominal pain.

Displacement of the liver into the thoracic cavity during diaphragmatic hernia produce severe abdominal pain. Rupture of the liver as a result of trauma may cause death from haemorrhage.

**Treatment of gastrointestinal emergencies**

1. Analgesics e.g. Baralgan inj (Hoechest)

2. Spasmolytics e.g. : Buscopan inj (German Remedies) contain Hyoscine - n. butyl bromide 20mg/ml.

3. In severe cases, use sedatives

   *Best Choice inj Xylazine (Xylaxin, Indian immunologicals)*

   Dose: - 0.1 - 0.2 mg/kg body weight intramuscularly.

   Xylazine should not be given in intestinal torsion or hernia.

   Animal should be undisturbed while giving Xylazine.

4. Antibiotics to check the infection.

   Preferably broad Spectrum antibiotics like Oxytetracycline can be used.

5. Treatment of specific conditions like lactic acidosis, and frothy bloat should be instituted after proper diagnosis.

6. Emergency laparotomy should be done in conditions like intestinal obstruction, torsion of abomasum, diaphragmatic hernia and traumatic reticuloperitonitis.
RUMEN COLLAPSE IN CATTLE

DR. B. NAGARAJAN AND DR. V. GNANAPRAKASAM

Digestive disorder in cattle is the most common problem faced by every field veterinarian in India. Diagnosis of a particular problem and differentiating it from other diseases are important to give correct treatment and prognosis. In the big list of Digestive disorders of Cattle, a new condition called 'Rumen collapse' has been added, in the recent years.

WHAT IS RUMEN COLLAPSE?

In physical methods of examination, simultaneous auscultation and percussion of the bovine abdomen is carried out in diseased condition in which gas and fluid distension occur in a particular organ, e.g. Displaced abomasum, dilated caecum. This distension of organ will make the visceral peritoneum of the distended organ to be against the parietal peritoneum. This will make the visceral peritoneum of the distended organ to be against the parietal peritoneum. This will create tympanic resonance or 'Ping' sound while doing percussion and auscultation externally. Apart from diseases like Ruminal tympany, left displacement of abomasum and pneumoperitoneum, a condition called 'Rumen Collapse' also creates 'Ping's sound in left side abdomen of cattle. This condition occurs when the cattle is affected with a primary severe inflammatory diseases like Pneumonia, metritis or Mastitis.

CLINICAL SIGNS

Since this condition is occurring along with a primary inflammatory condition, there will be fever, toxemia and the animal will be anorectic for several days prior to the detection of abdominal ping sound. Percussion of the left paralumbar fossa and surrounding area will reveal a rectangular shaped ping in the left upper quadrant of the abdomen of cattle. 'Ping' can be heard in the left paralumbar fossa and extended in the dorsal direction above the level of the transverse process of the lumbar vertebra. There is no correlation between the rumen distension and ping sound. Rumen will not be palpable at left side paralumbar fossa due to shrinkage of the rumen.

On rectal examination, the left upper quadrant of the abdomen will be devoid of viscera and dorsal sac of the collapsed rumen can be appreciated at the left ventral quadrant of the abdomen. Left kidney will be displaced at mid abdomen ventrally.

DIAGNOSIS

'Ping' sound occur in left abdominal area of cattle in left displacement of the abomasum, ruminal tympany and occasionally in pneumoperitoneum.

In rumen collapse dorsal extent of the ping extents above the level of the transverse processes of the lumbar vertebrae (Fig 1). On rectal examination dorsal sac of the rumen will be collapsed with ventral displacement of left kidney into mid abdomen.

In left displacement of Abomasum, 'ping' occurs under the left rib cage and it is located along a line from the left elbow to the left tubercoxae (Fig 2). By succussion and auscultation fluid can be detected in left displacement of abomasum which is not present in case of rumen collapse. 'Liptak' test can be performed to diagnose left side displacement of abdomen by aspirating the fluid on the left side in the middle of the last inter costol space and finding the PH.
Pneumoperitoneum can be differentiated from rumen collapse by observing a similar 'ping' sound in the right dorsal abdominal quadrant, causing left side 'ping' extending to dorsal midline (Fig.3). In pneumoperitoneum, rectal examination will be very difficult because the air pressure will compress the rectal wall. In ruminal tympany distended gas can be easily detected at left paralumbar fossa by palpation and percussion.

Fig. 1. Typical area of tympanic resonance observed in rumen collapse. The paralumbar fossa is depicted as a triangle within the area.

Fig. 2. Typical area of tympanic resonance for an average-sized LDA.

Fig. 3. Area of tympanic resonance observed in pneumoperitoneum or rumen tympany.

TREATMENT

Primary inflammatory diseases like septic metritis, mastitis or pneumonia should be treated with specific Antibiotics. Non-steroidal anti-inflammatory agents should be used to treat endotoxaemia. If there is Ketosis, it should be treated with Dextrose. Acid base electrolyte abnormalities and dehydration should be corrected. Rumen fluid transfusion should be done when there is stasis.
Reproductive behaviour signs from estrual behaviour to parturition directly or indirectly affects the digestive function, more effect on ruminant digestion and rumen. For example various patterns of courtship display, motor activities and postures are to ensure fertilization, pregnancy and propagation of the species. Begeting the young one is a luxury and pregnancy lactation and kids are essential for meat, egg and other by products.

Psychological aspects of reproduction & digestion:

The encounter with sexual partner is the first step of reproductive behaviour. In free living animals it is influenced by pre existing social structure. In species there is difference. In other species like rabbit, the territory is occupied by a permanent couple or harem and the male animals avoids any encounter outside his territory. Even in case of male rabbits there is a period after sometime it considers it his territory. During this acclimatization, feeding is lessened.

Sequence of sexual behaviour and digestion:-

In male animals, sniffing and licking are habits acclimatized. In female animals, during estrous, there is increased motor activity, restlessness and moving at slightest disturbance. Interest to feeding is also less, since endocrine mechanisms is involved in the sexual behaviour. Steroids of male and female animals have close biochemical simulations. In the male due to hormonal effect the search for the female in estrum. In the female, pheromones are the substance which attract the male, and during this period the normal feeding habit is reduced. Sexual steriods initiate sexual motivation-steroid hormones in blood stream and maximum in CNS (estrogens) since the nervous structures are impaired it has bearing on the feeding habits, inappetence. In cystic ovary, nymphomaniac cows may be as sexually aggressive as a bull in seeing out and attempting to mount a cow approaching or in estrum. The homosexual characteristics of cattle are aggrevated during this disease. The affected cows because of their actions are often spoken of as "bullers". These cows especially if on pasture liable to lose weight during the disease because of constantly moving about attempting to mount and stirring up the other cows and its appetite is reduced.

Digestive involvement in the reproductive process:

In normal pregnancy and gestation no changes are exhibited. but in case of improper artificial insemination, it results in anorexia, acute peritonitis. In such cases treatment is usually with corticosteroids and antibiotics. Levamisole, an immunostimulant can also be attempted. But when there is short gestation period with abortion or premature birth, adverse disease factors contributing to it from endometrium and placenta. Stress conditions also cause abortion and at the sametime the animals go in for inappetence.

When the cause is due to consuming of verratrum californium has been reported in cows, there is digestive disturbance. One of the importance digestive change which happens and looks pathological is loose dung but when a buffalo is near parturition, it is a physiological one. When Bacterial cause like Brucella abortus, leptospirosis varied digestive changes are observed. This may be due to the stress and change in the homeothermic condition, rise of temp and the feeding changes into inappetence. Therapy for acute stages of
leptospirosis includes the parenteral administration of large doses of antibiotics including penicillin. 3 millions units and streptomycin 5 gms twice daily or the tetracylines 2.5-5 gms daily per 1000 lbs animal for 5 days.

In case of viral abortion in cattle like, Infectious bovine rhinotracheitis (red nose) and infectious pustular vulvo-vaginitis (IBR-IPV) virus the clinical forms of the disease are varied. In the upper respiratory form (or) red nose there is temp 104-107°F, anorexia, depression and reddened nasal mucous membranes, nasal discharge and pustules and in conjunctival form there is marked lacrimation changing to a mucopurulent profuse discharge from the eyes. In the neonatal digestive form in young calves from birth to 2.3 weeks of age is associated with a high mortality. It resembles E.Coli exhibiting necrotic lesions in the mouth, pharynx, oesophagus, fore stomach with diarrhoea and death within 3 days. The meningoencephalitic form is characterised by dullness, incoordination, tremors, amaurosis, opisthotonus, coma and death. The vulvo vaginal form is characterised by generalized septicemia, in preputial form-pustules and ulcerations of the penis and prepuce. The prenatal (or) abortive form is characterised by intrauterine death of the fetus and abortion 2-5 or more days later. IBR-IPV can be transmitted through semen.

The all above forms, are characterised by a viremic and septicemic phase with an elevated temp depression, and anorexia lasting from 2-10 days.

Diagnosis is by serological examination. Treatment is done by localized antibiotics. Vaccination is done as prevention. But pregnant animals should not be vaccinated.

Mycotic abortion:-

Aspergillus spp are most common. The fungi mentioned probably exercise their pathogenic effect by invasion of the fetal and placental tissues but is also possible for abortion to result from a mycotoxicosis. It is considered that infection takes place by the respiratory or the alimentary route and that haematogenous spread accounts for placental involvement. There is no evidence to indicate that there is an ascending infection of the genital tract in cattle. The incubation period depends upon the infection dose and may be less than 2 months. The foetus may appear normal. But often there are characteristic cutaneous lesions in the form of irregular raised plaques resembling ring worm, most frequently seen on the back and sides, sometimes hair follicles and dermis are invaded by the fungus. Placentomes are swollen and necrotic and the allanto-chorion thickened, brown and leathery. Abortions occur late in gestation and the placenta in the cow is frequently retained. Fungal hyphae may be demonstrated in smears of fetal stomach contents. Sections taken from the placenta stained by Gridley’s method or PAS show the distribution of hyphae in the tissues. Varied digestive abnormalities are observed.

Hydrellantois (dropsy of the allantois):

Depending on the degree of the involvement and the stage of pregnancy, the signs it normally develops rapidly within 5-20 days and is characterised by distended uterus and enlarged abdomen. Spontaneous abortions at 6-9 months may frequently be observed. In more severe cases the amounts of fluid may reach 20-50 gallons. Later, digestive symptoms with anorexia, lack of ruminations and constipation are noted. The condition is frequently misdiagnosed as indigestion, bloat or traumatic gastritis. Pulse elevated 90-140/ min and is weak wiry. The cow may exhibit anxiety, restlessness and an expiratory grunt. The gait is stiff, slow and cautious. The animal loses body condition and eventually is unable to rise. Dislocation of hips or backward deviation of the rear limbs may occur and the cow lies on her sternum looking like a "bloated bull frog". Rarely rupture of the prepubic tendon (or) ventral hernia may occur due to excessive weight of the uterus.
Displacement of genital organs and digestion

Torsion of uterus: May be due to deep capacious abdomen, relative instability of the uterus, excessive foetal movements and above all the attachment of the broad ligament unlike in other species is dorsolateral in cattle. In 45 to 90 or even 180 torsion, symptoms may be completely lacking prior to parturition. When it is 180 degrees or more, definite signs of abdominal pain usually may be noted, such as anorexia, constipation, lack of rumination, weak and slow rumen contractions, rapid pulse rate, restlessness or colicy symptoms, treading and tailswitching. Symptoms may be confused with traumatic gastritis, indigestion, pyelonephritis or intussusception. Uterine torsion in ewe is characterised by a stiff, stilited gait and a stretched, "saw horse" attitude resembling signs of peritonitis and intussusception or volvulus. Diagnosis is made by rectal and vaginal examinations. Treatment is carried out adopting simple rolling of the animal or by schaffer's method. In cases which fail to respond to this lapar hysterotomy should be done. In delayed cases, it may result in rupture of the uterus with peritonitis, internal bleeding due to rupture of large uterine vessels, retained placenta, septic metritis and perimetritis are common sequelae to torsion of the uterus.

Retention of foetal membranes (retained placenta) and digestion

If the foetal membranes is not expelled within 8-12 hours after parturition, the condition is known as Retained Placenta. If the condition persist for a long time i.e. more that 12 hours, the secondary bacterial infection may occur. This leads to pyrexia and anorexia. The treatment for this condition is manual removal of the placenta with inserting suitable antibiotics into the uterus and administering antibiotics parenterally. Duration of treatment may be 2-3 days.

Impairment of digestion due to metritis

It is the inflammation of the uterus. The causes may include retained placenta & dystocia. It may be acute, chronic or septic metritis. In septic metritis there will be copious sero-sanguineous discharge. The commonest symptom is anorexia & pyrexia. This is due to the bacterial toxins. The treatment include treating the uterus with oxytetracycline and administering antibiotics parenterally.

Cervico-vaginal prolapse

It is the prolapse of vagina, or (cervix) occur during oestrus or prior to parturition or after parturition. This condition is due to excess straining, which causes loss of energy from the animal and the attitude of the animal will be towards, straining which leads to anorexia. If the prolapsed mass is lacerated or inflamed there may be secondary bacterial invasion leading to the systemic disturbances which causes reduced rumen motility & anorexia. The treatment involves, conventional type of treatment, i.e., prolapsed mass is washed with antiseptic solution and reduced and vulvar retention suture will have be applied with antibiotic cover.

Dystocia

In dystocia the normal P1, P2, P3 of the calf will be altered due to many causes. The dystocia may be due to foetal anamoly or enlarged size of foetus or due to narrow pelvis. in dystocia, the animal will be restless and strains continuously. This cause loss of energy & reduction in ruminal motility which intern leads to anorexia.
Post parturient indigestion

It is rather not surprising to note that in towns and cities people who, rear cows and buffaloes to “pump in” these feed following parturition. The sudden stress, animal is not able to adjust and goes into varied states of indigestion with pellety dung etc. A study in this department of Obstetrics and gynaecology (Rajasekaran and Venkataswamy, 1981) revealed marked variation in normal, post partum animals to indigestion following parturition. In the normal post partum, in she buffaloes, the rumen liquor colour was greenish, consistency thick, odour normal aromatic, pH 7.3, acetic acid 47.3 Molar %, propionic acid 22.5 Molar % and Butyric acid 12.51 Molar % T.V.F.A. was 85 m.moles / litre, total protozoa 3.86x10^5 /ml. total bacteria 11.16 * 10^7 /ml. the total nitrogen 860 mg/litre and Ammonia nitrogen 61.60 mg/litre and in post partum indigestion in she buffaloes, the appearance was dark brown the consistency and putrid odour and the values 7.9, 59.64, 26.75, 13.61 %, 69.64, 2.00, 6.43, 600.00 and 91.28 respectively of the parameter’s given in normal post partum animal.
REPRODUCTIVE DISORDERS AFFECTING RUMINANTS.

Dr. S.A. ASOKAN

Reproductive disorders in ruminants is a serious problem causing severe economic loss to the farmer especially, when the objective is to achieve increased milk production through planned breeding programme.

The life time production of the dairy cow commences with the heifer calf born at the right time, calving down at two years of age and subsequently calving regularly every twelve months and for as many years as possible, provided such an animal has the right genetic background and is fed for optimum economic yield then she will make the maximum contribution to the profitability of the herd.

Fertility is a sensitive indicator of general health for it can be affected by any disease localised elsewhere in the body such cases of secondary "symptomless". Infertility may be elucidated only through checking of all body systems and the environmental conditions.

Of the total reproductive disorders 80.15 percent were recorded during the first 90 days of the calving (Jadhav et al., 1992) The post partum disease are grouped together and termed as 'Parturition syndrome'. There is a steady rise in the incidence of this syndrome during the last three decades. Contributing factors for this syndrome may not only be genetically determined, but may also be due to unfavourable managerial system and less time devoted to care and attention of individual animal.

Parturition is a major recurring inevitable stress on the dairy cow which involves parturition and feeding of the calf with high milk supplies which finally overload the control capacity of the cow and leads to illness depending on the genetic and different performance criteria. The illness manifests in terms of retained placenta, parturient paresis, acetonemia, mastitis, endometritis, ovarian dysfunction etc., (Tommer, 1975). When stress continues and the potential for compensation has been exhausted break down occurs resulting in number of pathological conditions. Therefore, in order to reduce expenditure on reproductive disorders maximum attention should be given towards sexual health of animals during early lactation. A high standard of sanitation should be maintained during calving time in order to avoid contracting infections. Occurrence of reproductive disorders was highest in rainy season (30.08%) as compared to other seasons which may be due to difficulty in maintaining the standard of sanitation (Jadhav et al., 1992).

It is indicated that the cow with disturbed liver function prior to calving are particularly prone to contract the symptoms of parturition syndrome after calving because their physiological control capacity is restricted.

There are certain specific gynaecological and obstetrical conditions having influence over the G.I. tract of ruminants. Few hours before calving the cow may exhibit anorexia and restlessness. Heifers may show signs of abdominal pain as indicated by kicking at the abdomen, treading, tail switching, frequent lying down and getting up and frequent bowel evacuation.

Toxic indigestion of advanced pregnancy occurs in stabled cows late in the winter confinement period, wherein normal activity of gastrointestinal tract appears impaired possibly due to large gravid uterus.

In uterine torsion there is sluggish ruminal motility and a constipated pelleted dung is voided. This is due to twist in the uterine horns which might have interfered with normal bowel movements. In severe cases of uterine torsion due to a toxic degenerative process, foetid, olive green dung is commonly observed.

There is always close inter-relationship between digestion and the infective process in the reproductive tract. For example in septic metritis and puerperal metritis anorexia is the important symptom exhibited by the animal. This is because of histamine production by the degenerative cells which interferes with the motility of the intestines.
Mechanism of histamine production

*Injury (inflammation)*
- Stimulates histamine decarboxylase enzyme
- Converts histidine amino acid in the cell
- Histamine
- Increased body temperature
- Reduced Rumen motility

SPECIFIC GYNAECOLOGICAL/ OBSTETRICAL CONDITIONS INFLUENCING G.I. TRACT OF RUMINANTS

<table>
<thead>
<tr>
<th>Condition</th>
<th>Symptom</th>
<th>Mechanism</th>
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<tbody>
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<td>Oestrum</td>
<td>Inappetence</td>
<td>Neurohormonal</td>
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<tr>
<td>Allburn</td>
<td>Inappetence</td>
<td>Inflammatory process</td>
</tr>
<tr>
<td>Genititis</td>
<td>Inappetence</td>
<td>Mechanical stress and Hormonal</td>
</tr>
<tr>
<td>Advanced Pregnancy</td>
<td>Inappetence</td>
<td>Mechanical stress and Hormonal</td>
</tr>
<tr>
<td>Retention</td>
<td>Anorexia and diarrhoea</td>
<td>Mechanical stress and Hormonal</td>
</tr>
<tr>
<td>Ovarial</td>
<td>Inappetence</td>
<td>Stress and Mechanical</td>
</tr>
<tr>
<td>Torsion</td>
<td>Diarrhoea and constipation</td>
<td>Stress and Mechanical</td>
</tr>
<tr>
<td>Ovarial Torsion</td>
<td>Anorexia</td>
<td>Degenerative process</td>
</tr>
<tr>
<td>Inappetence</td>
<td>Stress and Mechanical</td>
<td></td>
</tr>
</tbody>
</table>

In principle, any medicament which stimulates ruminal and liver function and activates the metabolism suitable. Treatment should in all cases have positive influence on ruminal and liver function and help avoid energy deficit and intoxication.

To obtain higher success, the veterinarian should select the preparation of treatment which he considers post suitable for metaphylaxis on a particular farm and for individual animals which are known to him to be susceptible to certain diseases.
The four important aspects of rumen function are

1) Rumen motility
2) Microbial metabolism
3) Associated secretory process and
4) Neuro-hormonal control.

Adaptation to the coarse and bulky character of forage is achieved by ruminants with the help of regular mixing movements of the reticulorumen. The reticulo-rumen is innervated by the Vagus nerve and their mixing movements are accelerated by the process of feeding and rumination. Microbes present in the rumen help in the conversion of food into volatile fatty acids (VFA). The other important functions of rumen include the following.

1) Conversion of nonprotein nitrogenous (NPN) substances like Urea, ammonia into microbial Protein.
2) Vitamin B1 synthesis.
3) Lipid hydrogenation.

Hypomotility states

When animal husbandry systems become more intensive where excessive amounts of easily fermentable carbohydrates are fed to ruminants, the chances for the occurrence of ruminal atony also increased. Hyperthermia (fever) may precipitate ruminal stasis. Conditions like shock wherein there is release of substances like Catecholamines, Serotonin, Prostaglandins, Plasma Kinins etc., resulted in reflex inhibition of extrinsic reticulorumenal contractions. Hypocalcemia is another factor which may produce hypomotility of rumen.

Drugs altering ruminal motility

The search for drugs which act directly on the ruminal motility has not proved successful. Pilocarpine when administered induces copious flow of saliva and thereby producing increased chewing movements which reflexly increases the rumen motility. Chewing movements are one of the Physiological factors which reflexly will increase rumen motility. In case of ruminal stasis due to hypocalcemia, administration of calcium will correct this condition and will improve rumen motility. Carbachol and similar Parasympathomimetic agents usually increase the amplitude of the reticular contraction but not the frequency and this increase is followed by inhibition. Histamine and acetylcholine inhibit both ruminal and reticular activity. Atropine also inhibits rumen motility. Adrenergic drugs will inhibit reticulorumen motility.

Pharmacological basis of treatment of rumen dysfunction

I. Antimicrobial Therapy

Although the growth stimulating effects of oral administration of certain antibiotics have been widely demonstrated in Poultry and Swine, less such information is available on this type of response in ruminants.

When crystalline Aureomycin hydrochloride was added to the diet of calves, a growth stimulation
effect and lowering of the incidence of diarrhoea was noticed. But when the same drug was administered in lambs they depressed the growth and reduced the diameter and length of wool fiber.

When Sulfadiazine, Sulfathiazole and Sulfamerazine were administered to ruminants they were excreted through the ruminal wall and the salivary glands. These Sulfonamides had a slightly inhibiting effect on cellulose digestion and on the activity of rumen infusoria. Sulfathiazole was the most rapidly excreted member in this group.

Oral administration of Sulfonamides to ruminants may produce changes in the rumen flora. Prolonged oral administration of Sulfonamide may produce Vitamin K deficiency which may result in haemorrhage. Decreased cellulose digestion, glucose fermentation and inappetance may also occur. The beneficial effects provided by the judicious use of orally administered Sulfonamides far outweighs any detrimental effects that may accrue as a result of their action on rumen microorganism.

On the other hand rumen protozoal concentration increased following antibiotic supplementation. Culture studies have shown that Sulfadimidine was the only Sulfonamide of those tested which was toxic for Isotricha even in subtherapeutic doses. Chlortetracycline also yielded similar results. On the other hand Penicillin and Streptomycin even in large doses had a favourable effect on viability of cultures.

A) In cases of ruminal acidosis

Streptococcus bovis has been implicated as the cause for rumen acidosis. Bacitracin completely inhibited the growth of this organism at all concentration. Oxytetracycline also produced such an effect. Oral administration of 2-4 mg/kg body weight of Neomycin at least three times a day or administration of 10-20 mg/kg body weight of Chlortetracycline or 50 mg/kg body weight of Chloramphenicol can also be recommended. Erythromycin (10 mg/kg) which is specific for gram positive bacteria may be more beneficial than broad spectrum antibiotics. Penicillin and Chlortetracycline administered with grain will prevent the onset of lactic acid fermentation.

B) In cases of bloat

Administration of Procaine Penicillin at a level of 100,000 units orally has been reported to decrease bloat without Penicillin residues appearing in milk. However continued use of antibiotics to prevent bloat has resulted in the development of Penicillin resistant organisms.

II. Anthelmintics in ruminants

In ruminant animals oral dosing with the Benzimidazoles will remove all the major adult gastrointestinal parasites and many of the larval stages. The delivery of Benzimidazoles directly into the rumen by specialized intra-ruminal injection has now been introduced in many countries with a view of improving both the spectrum and effectiveness of the anthelmintics. Ruminal deposition acts to slow its Pharmacokinetics and appreciably increase the potency of the compound. Nitroxynil is metabolised by rumen bacteria which destroys its activity. Hence it is unsuitable for oral administration.

III. Other agents

The following two effective approaches can be made for the prevention of bloat.

1. Decreasing ruminal protozoa

   Copper Sulphate given orally at the dose of 4.4 g/100 kg body weight will kill protozoa. An organic antiprotozoal agent Dioctyl Sodium Sulpho-Succinate decreased the incidence of bloat.

2. Breaking down and preventing the formation of a stable foam

   Surfactants like poloxalene acted in this manner.
Ruminant Toxicology

Dr. Jeyasekaran Samuel and Dr. V. Gnanaprapakasam

Toxicology is a study of poisoning. Toxicology is concerned with the effects of therapeutic agents administered in excess and of substances having only a toxic action. In addition, toxicology is concerned with environmental health problems that may require investigation of the toxic properties of water containing industrial or sewage wastes and of toxic vapour from Industrial plants and numerous other things hazardous to life.

Problems of poisonous plants and control measures

The economic impact of livestock poisoning by plants is enormous. It is estimated in our survey that poisonous plants affect 34% of the cases. Measures to prevent poisoning from plants include:

1. Learn identification of toxic principles of poisonous plants on large.
2. Use good grazing management to maintain range in condition least conducive to high poisonous plant populations.
3. Adjust stocking rates so that animals have ample forage plants in relation to poisonous plants.
4. Supplement salt, minerals and nutrients whenever necessary.
5. Avoid grazing areas where poisonous plants are abundant.
6. Use a class of livestock not generally poisoned by plants present.
7. Avoid turning hungry animals into ranges containing poisonous plants. Hungry animals are less selective and select poisonous plants.
8. Provide water adequately at all times to prevent non-selective grazing following water deprivation and subsequent watering.
9. Reduce poisonous plant population with mechanical or chemical methods.

Problems of poisoning from feed

Symptomatology, epidemiology and pathology are all essential and must be included in establishing a correct diagnosis and intelligent analysis of any outbreak of poisoning due to feed. Where these data are not considered and evaluated specific agents may be falsely incriminated. Various agents causing food poisoning have been discussed. Toxicants of industrial origin, heavy metals, air pollutants, solvents and vapour effects on animals...
### CHARACTERISTICS OF G.I. UPSET FROM CHEMICAL POISONS

<table>
<thead>
<tr>
<th>Specific</th>
<th>Food concerned</th>
<th>Symptoms</th>
<th>Onset of symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antimony</td>
<td>Foods Cooked in Cheap gray Enamelled Cooking utensils</td>
<td>diarrhoea, painful tenesmus,</td>
<td>With in a few minutes to an hour</td>
</tr>
<tr>
<td>Arsenic</td>
<td>Sodium fluoride mistaken for baking soda Powder or flour.</td>
<td>often haemorrhagic, diffuse abdominal pains and diarrhoea occur with great consistency convulsions, toxic or clonic spasms of certain groups of muscles, (eye muscle, facial muscles and those of lower extremities) contraction of pupils and paresthesia in extremities occasionally present; may be confused with botulism</td>
<td>10 minutes and longer</td>
</tr>
<tr>
<td>Fluoride</td>
<td>Sodium fluoride mistaken for baking soda Powder or flour.</td>
<td>Abdominal Pain, and diarrhoea in acute cases</td>
<td>50 minutes and longer</td>
</tr>
<tr>
<td>Mercury</td>
<td>By licking</td>
<td>Salivation, great thirst, and abdominal pain; watery diarrhoea within 2 hours.</td>
<td>2.30 minutes after ingesting bichloride of mercury.</td>
</tr>
<tr>
<td>Iodates</td>
<td>Water</td>
<td>Cyanosis, and diarrhoea</td>
<td>Several days</td>
</tr>
</tbody>
</table>

There is a great potential for occupational exposure to these chemicals both in production and in use. Residues of insecticides remain in the produce of man and animals are exposed even to low levels of chemicals. Numerous incidents of acute poisoning with insecticides from eating food which was grossly contaminated either during storage or shipping occur. Insecticides also cause accidental poisoning.
Emergency treatment and management of animal poisoning

Principles of Treatment of Poisoning of drugs are:

I. Terminating the toxic exposure or preventing more poison being taken.
II. Providing supportive care or symptomatic care.
III. Giving antidotal therapy (specific)
IV. Hastening the elimination of absorbable poison or the excretion of poison.

I. Terminating the toxic exposure

The chemical nature of the substance to which the animal is exposed is very important. For example: strong acids from insoluble acid proteinates and hence prevention is limited.

Alkali is more dangerous than acids. They form alkali proteinates and also forms soaps due to fat. The scope will penetrate rapidly into the tissues.

A. Inhalation

For the termination of toxic exposure due to inhalation:
1. Remove from the exposure and
2. Provide assisted ventilation.

B. Eye contact

Clean the eye thoroughly by ophthalmic irrigating solution or tap water for a period of minimum 5 minutes to maximum 15 minutes.

Then the eye should be instilled with a local anaesthetic. Then the eye should be irrigated with water or saline for about 30 minutes; Then the eye should be examined and antibiotic ointment is applied.

C. Skin contact

1. Wash with plenty of water and soap
2. Fat soluble poisons should be removed by organic solvents (Eg. Benzene, Alcohol).
3. For Alkali, wash with weak acid solution or vinegar and water.
4. For acids, wash with weak alkaline solution. However, for Alkali and acid exposures do not use a chemical antidote. The neutralisation may cause a thermal reaction and the heat generated may increase the irritancy. The time involved to mix these solution makes them less desirable. So pouring of plain water for 10 to 15 minutes may be effective.
5. If systemic absorption occurs after contact, cleaning with detergent should be undertaken. Eg. True pesticides.
D. Ingestion

The termination of toxic exposure due to ingestion may be undertaken:

1) By Lavages
2) By (Absorbent) activated charcoal
3) By Catharsis or laxatives
4) By local antidotes
5) By Rumenotomy

1. Lavages: Rumen can be lavaged by use of Rumen Fluid Extraction pump. The rumen contents should be pumped out immediately. Then fill the rumen with adequate normal saline and then pump it out. Repeat the technique until the toxic materials are completely removed.

2. Absorbent: Activated charcoal absorbs the toxin (Middle sized molecule) and it will not detoxify it. So it only minimise the absorption of toxin. Then laxative or osmotic cathartics or colonic lavage should be given.

   The proper technique for utilizing activated charcoal is as follows: (1) Makes slurry of the charcoal using water. The proper dose is 2 to 9g/kg of body weight in a concentration 10 charcoal/3-5 ml of water. (2) Administer the charcoal by a stomach tube. (3) Thirty minutes following administration of the charcoal, a cathartic of sodium sulfate should be administered. Some charcoal should remain in the rumen and should be followed by a cathartic to prevent resorption of the toxicant. Advantages are: (1) It is better than the universal antidote. The tannin and Magnesium oxide will interfere with the absorptive capacity of activated charcoal. (2) Effective for Mercuric chloride, Strychnine, Morphine, Atropine, Barbiturates, Ethyleneglycol, HCN.

3. Cathartic or laxatives: In case of chemical poisoning, osmotic cathartics like sodium sulphate is superior to Magnesium sulphate since Magnesium will be absorbed and act as CNS depressent.

   Saline purgative should be given after 1 1/2 hour of administration of activated charcoal

   Dose: Magnesium Sulphate 1 lb/cattle, 4 gm/sheet.

   It should not given in the case of corrosive poisons since there is severe gastro enteritis.

Mineral oil or Vegetable oil: They have a good value in the case of lipid soluble toxicants.

4. Local antidote:

   Chemical antidote

   Acid: Large quantity of water
   Magnesium Oxide - Neutralize
   Soluble Calcium hydroxide -

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Do not use substances which produce CO2, Mild solution of sodium Bicarbonate can be used for eye irrigation

Alkalis 5% Acetic acid
5 to 10% Citric Acid
5 to 10% Tartaric acid
Boric acid for Eye rinsing

Tannin: effective in the case of copper, mercury, zinc, some alkaloids like Aconite, Brucine, Cocaine, Nicotine and Pilocarpine poisonings.

Lugol's iodine: Partially precipitate lead, Mercury, Silver and Strychnine.

Milk and Eggs for phenol and heavy metals.
Calcium compound: Effective in fluorides, oxalic acid and poisons which damage the walls of blood vessels and cause pulmonary oedema (Phosogens and sulphur oxide)

Sulphur compound: (Sodium thiosulphate, cystine and cysteine) Effective in metalloids (Arsenic, Antimony, Bismuth and in heavy metals) (Mercury, copper, zinc, cadmium). It acts by forming insoluble sulphides. It is also effective in HCN poisoning.

Universal antidote: Poisoning by unknown substances in animals, the following mixture may help to neutralize it.

Activated or powdered charcoal - 12 parts
Light Magnesium oxide - 2 parts
Tannic acid - 1 part
Kaolin - 1 part

Dose:
Cattle: 240 Gm two or three times daily
Calves: 2 tablespoon two or three times daily
Sheep: 1 tablespoon two or three times daily

S. Rumenotomy
In ruminants, it is advisable to remove the rumen contents by rumenotomy.

II. Supportive therapy
Supporative therapy must be given as follows to preserve the functions:

(1) Control of the CNS activity (2) Maintenance of Respiration (3) Control of body temperature (4) Maintenance of cardiovascular system.

After the crisis is over
5) Fluid therapy (6) Nutritional therapy
1. Control of the CNS activity

Depression: CNS depression can be considered as respiratory depression since both are related. Management is similar in both.

a) Effect of intravenous analeptic will remain only for short period.

b) The comatose condition will return if the case is not properly monitored.

c) Analactics may produce convulsions

Cattle: Doxapram (Dorpram) 5 to 10 mg/kg
Bemegride 10 to 20 mg/kg
Pentylene tetrazol 6 to 10 mg/kg

Artificial respiration or respiratory support is very important. Better do not use Analactics.

CNS Hyperactivity: Convulsion to mild excitation. If there is no specific antidote, use Diazepam. If it is too not available, use short acting barbiturates.

a) Pentobarbital sodium is the choice for convulsion. But, a respiratory depressing dose is required. (Respiratory support is mandatory).

b) for long term CNS hyperactivity. Inhalent anaesthesia is preferred.

c) Convulsion due to intoxication: Centrally acting skeletal muscle relaxants and mild tranquilizers are effective.

Cattle: Metho carbamol 110 mg/kg/i/v
Glyceryl Guaicelate 110 mg/kg/i/v
Diazepam 0.5 mg kg i/v or i/m.

d) If there is stimulation due to stimulant drugs phenothazine tranquilizers are preferred,

e) If hyperactivity is due to pain, use analgesics. Keep the animal in a quiet dark room to prevent additional auditory and visual stimulation.

2. Maintenance of respiration

a) Provide patent airway by introducing an endotracheal tube if the patient is in coma or do tracheotomy,

b) Restore respiration by mechanical respiration.

c) Avoid over ventilation which will lead to alkalosis.

3. Control of body temperature

a) Hypothermia: To control the body temperature use Blankets, Infraed lamps, heating pads or circulating warm water pads. Animal must be placed in a warm place.

b) Hyperthermia: Ice bags or cold water baths and enema can be used. Cold peritoneal dialysis can be given. To avoid dehydration fluids should be administered.
4. Cardio-vascular support

For the maintenance of cardio-vascular function there should be:

a) Adequate circulating volume of blood
b) Adequate cardiac function
c) Adequate tissue perfusion
d) Adequate acid base balance

If there is loss of cells and volume then blood transfusion is essential.

Hypovolemia due to lose of fluid alone can be effectively supported with plasma volume expander or lactated Ringer’s solution.

To have an adequate tissue perfusion Dexamethasone 2 to 10 mg/kg can be given. To aid cardiac activity, following therapy is effective.

a) Cardiac massage
b) Intravenous calcium borogluconate slowly
c) Glucagon 25 to 50 Microgram/kg i/v
d) Digoxin 0.2 to 0.6 mg/kg i/v
   To combat metabolic acidosis.

a) Sodium bicarbonate 2 to 4 M.Eq/kg i/v for every 15 minutes.
b) 1/6 molar lactate 16 to 32 ml/kg
c) Ringer’s solution 120 ml/kg
d) Tham buffer 300 mg/kg

To combat metabolic alkalosis:

0.9% NaCl 10 ml/kg

It should be followed orally with ammonium chloride 240 mg/kg day in divided doses.

Blood pressure and pulse are measured: If blood pressure is low, find out whether it is due to simple Hypotension or vascular shock- continuous drip of norepinephrine in saline may be indicated in simple hypotension. But, it is contra-indicated in the case of vascular shock.

III. Systemic and specific antidotes:

Immediate treatment with systemic antidote should be carried out to promote excretion of the absorbed toxin.

Systemic antidotes are classified according to their mechanism of action.

a) Render the poison inert (for Eg.) BAL in Arsenic.
b) Accelerating the metabolic conversion of the toxic product to non-toxic one.
c) For eg. Thiosulfate in HCN poisoning.
Pesticide poisoning:

Organophosphorous compound:

a) Symptomatic: Atropine sulphate: 0.25 mg to 0.5 mg/kg body weight 1/3 must be given in I/v slowly and rest by i/m or s/c repeat at interval of 36 hours over a period of 24 to 40 hours.

b) Release of CHE block by: Organophosphorous compound.

2. PAM chloride or iodide: Iodide is preferred. 25-50 mg/kg body weight as solution to be given within 6 minutes in i/v (Maximum 100 mg/kg by i/v drip).

Combined treatment: Atropine with PAM is effective in large animals. First 12.5 to 20 mg of Atropine in 10 ml of water I/V followed by 50 mg of PAM dissolved in 100-50 ml of normal saline s/c but, not more than 50 ml at a site.

Death in OP poisoning is due to respiratory failure as there is copious tracheal secretions. Hence, atropine will be of immense use if administered through a nebulizer.

Contraindication: Morphine, Theophylline, and Coramine.

Carbamate Pesticides: Atropine only indicated-PAM is also effective.

Chlorinated hydrocarbon:

1. Sedation by chloral hydrates or barbiturates 2) Calcium Borogluconate i/v 3) Glucose saline i/v 4) Atropine can be tried 5) In last stage artificial respiration.

Hastening the elimination of absorbed poison

1. By Diuretics

2. By increasing the saliva
PATHWAYS IN DIAGNOSIS OF GASTRO INTESTINAL DISORDERS IN RUMINANTS

Dr. V. GNANAPRAKASAM

In buiatrics practice it is advisable to follow a routine scheme for the investigation and thereby can avoid many mistakes in diagnosis. Though it may not be possible to discuss everything in this paper, the salient points only will be discussed in short.

The clinical diagnosis procedures can be divided into three parts.

1. Routine general clinical examination.
2. Specific examination of abdomen and abdominal cavity.
3. Special examinations.

I. Routine general clinical examination

A. History:

Exact history of the case, feeding and managerial conditions particularly about appetite, thirst, manner of taking feed, details about parturition and defecation are to be ascertained.

B. Observation:

Environmental conditions, posture and gait, general condition of the body, condition of the hair coat, rumination, eructation to assess the rumen fermentation (normally once every 2 minutes), vomition, determination of reticulo ruminal movement by sub lumbar movement and abdominal shape are to be observed.

The visual survey of abdominal shape yields findings that not only guide the subsequent examination but are valuable as direct diagnostic clues as well. Abdominal distension may be visually assessed by comparing the abdomen with other parts of the body.

a) Slight distension of Lt. flank: Mild recurrent bloat due to penetrating foreign bodies.
b) Distention of Lt. paralumber fossa: Sub acute bloat due to obstruction of oesophagus by foreign bodies
c) A bowel shaped swelling in the left flank just behind the last rib: left side abomasal displacement.
d) Gross bilateral distension: In acute bloat due to dietetic errors.
e) Round appearance in left side of abdomen and distension in the lower right abdominal quadrant (Papple shape): in vagus indigestion.
f) Overall tense and barrel shaped distension: in advanced paralytic ileus with secondary ruminal tympany
g) Right sided abdominal distension in the upper caudal quadrant: Distended caecum and colon.
h) Distension of lower right quadrant: Abomasal impaction.
i) Asymmetric enlargement of right side: Abomasal volvulus, cecal volvulus
j) A swelling of the abdomen in the hypochondric region: Overload of abomasam or functional pyloric stenosis.
C. Temperature and pulse:

The observation of pulse is more important than temperature. High fever (41-41.5°C) with 80-85/min. pulse rate is not a serious case when compared to slight rise of body temperature with high pulse rate (128/minute).

D. Physical examination:

Techniques to be used for physical examination are:

a) Palpation is to gain information through the sense of touch as to the consistency, extent, temperature and sensitiveness of a part.

The methods: 1. Direct palpation by fingers or hand

2. Indirect palpation (Rectal examination)

b) Percussion can be practised by a plexor (Hammer) and pleximeter. Percussion carried out for

1) assessing the sensitivity to pain by a heavier hammer (Pain seeking percussion hammer) and

2) to set in vibration and emit audible sounds by small cushioned acoustic percussion hammer.

c. Auscultation: Best equipment to be used is phonendoscope.

Areas of various examinations:

RUMEN is located on the left side in the abdominal cavity, extending from the thorax and diaphragm anteriorly to the pelvis posteriorly. Ventrally in the hind part, it goes over to right side, somewhat above right flank fold. Auscultation and percussion can be conducted in left side over a wide area extending from the rib caudally to the tubercoxae. The percussion and auscultation area of rumen can be divided into (a) hollow of left flank and (b) abdominal wall within the costal arch. During auscultation, the attention is to be paid to nature, strength and frequency of sound. The time interval between 2 rumen motility is very important in the diagnosis than the number of motility per minute.

RETICULUM lies in the cranial region of the abodminal cavity slightly to the left of the midline, opposite the 6th to 8th ribs. In the animal standing normally, this area can be indicated by placing the hand on the abdomen immediately caudal to the point of the left elbow. The area behind the lower lung field at the level of the 6th to 8th rib in left side is to be percussed. The reticular movements can be heard as a rumbling gurgle, accompanied or followed by a liquid pouring sound by placing phonendoscope on the ventral aspect of left 7th rib.

OMASUM is located to the right of the rumen and reticulum, dorsally and forwards by liver and diaphragm. It is close to the right abdominal wall behind long line. Percussion field which is mostly easy to find is located in a semi-circle from a hand’s breadth above the costal arch, limits hindwards by 10th rib or 10th intercostal space. The percussion sound gives clear gas tone, a little more dull, heavier than the lung tone. The omasum field becomes smaller in chronic diarrhoea and functional disorders like stenosis between reticulum and omasum. In pyloric stenosis, the omasal field becomes larger. Auscultation at the centre of the omasal field may not succeed in picking up the rustling sound made by omasal contraction which is synchronous with reticular contraction because of louder sounds made by other stomach compartments. Fluid sound may be heard instead of rustling sound during anterior functional gastric stenosis.
**ABOMASUM** is occupying most of the hypochondric region and lies on the floor of the abdomen near the median plane. The fundus or anterior blind sac of the abomasum is located in the xiphoid region in close proximity to the reticulum. The pylorus joins the duodenum at the ventral aspect of the abomasum in the adult bovine limited by thick tense ventral abdominal muscles.

**Examination:**

**EXAMINATION OF BUCCAL CAVITY:** During the examination of mouth, observe the temperature, secretion of saliva, odour of mouth, gum, condition of teeth and tongue.

**EXAMINATION OF THROAT AND OESOPHAGUS** is restricted to external inspection and palpation. For probing the oesophagus, probang or stomach tube can be intubated. During the intubation, if the animal fails to extend the tongue properly to swallow, check for obstructions, injuries and paralysis. Oesophageal stricture can be recognised sometimes by failure of thicker tube to pass a point that a thin tube can pass. If there is difficulty in passing the probang in the region of diaphragm and cardia, when the animal is bloated, it will be surmised that undigested fibres have formed as plug in cardia and it will be too bulky to regurgitate during the attempt for rumination. In contrast if there is no difficulty during intubation in repeatedly bloated animal, the rumen sample must be examined. If the rumen sample is quite normal i.e. microorganisms and appearance except some lack of moisture, it indicates the enlargement of retro-pharyngeal lymph nodes.

During intubation in the animals which are having inappetance the probang can pass through a firm mass down to rumen floor and moves up to the flank region where it can be felt. But on the other hand, during the passage of probang, if the rumen fluid gushes out forcefully than before the probang enters into the rumen, it indicates that the cause for inappetance is not in the rumen. It may be due to ketosis, liver abscess or paralysis of prehension organs.

**Examination of abdomen and abdominal cavity:**

**Rumen:** Left side.

The consistency of rumen contents mostly of reticulorumen and condition of lymphglands can be ascertained by palpation in the hollow of left flank.

Palpation in hollow of left flank: Normal rumen motility rate is 7 to 12/5 minutes or 1 to 2/min. But the time interval between two motility is more important than numbers. The interval between 2 motility should not be more than 2 minutes.

- Tense and bulging - Bloat
- Light doughy or firm - Impaction
- Fluctuation and Splashing during punching - Functional stenosis
- Lack of resistance and lifeless impression. There is gap between the wall of rumen and the abdominal wall - Ruminal atony

Enlarged lymph glands - Rucnitis

By auscultation of the rumen in hollow left flank of healthy animal, the periodic rustling sound increasing to a crescendo then falling will be heard. In anorectic animals, the sound will be louder due to emptiness.

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Right side: The tension of abdominal wall can be tested by palpating its area between right side fold and hollow of flank.

Pain - intussusception and early peritonitis

Slackness without tension - severe generalised peritonitis.

Reticulum: External palpation is not possible. Reticulum can be auscultated and percussed to diagnose various disorders which are discussed later.

Omasum: can be percussed and auscultated to diagnose certain diseases of abomasum and rumen.

Abomasum: While palpating the projection field of abomasum if the animal shows the signs of pain, indicates extensive abomasitis or ulceration. Palpation with both fists in the xiphoid region may provoke a bound of abomasum which is hardened by accumulation of sand and impaction. Percussion and auscultation can be used only during displacement.

Rectal examination: The rectal examination in cattle provides valuable information. The information regarding state of rumen will be added along with palpation through left flank. In cattle, caudal sac of rumen are readily palpable when the rumen is distended as in bloat or vagus indigestion which may push well into elvis. In rumen acidosis forestomach contents are fluid and entire dorsal sac is palpable. By raising the abomasal floor, posterior part and ventral blind sac are also palpable. In functional stenosis, the posterior entral, blind sac or rumen extends well beyond the midline to the right ventral quadrant of the abdominal cavity just in front of pelvis. In right abomasal displacement, a large tense abomasum occupies the right dorsal quadrant of the peritoneal cavity or may fill the entire right half as far as pelvis. In left abomasal displacement, the abomasum can be felt only when it is exceptionally dilated or excessively displaced. It was felt like a tense oval balloon to the left of the dorsal sac of rumen.

In evaluation of caecum, a rectal examination will confirm the diagnosis. In caecal volvulus or cecal islocation, tip of caecum or flexed portion of caecal volvulus will be detected as 10 to 15 cm diameter gas and fluid filled viscus in right hemiabdomen resembling a flexed human knee. It will be found in pelvic inlet just anterior to the brim of the pelvis. Transabdominal palpation can be done my making a fist and gently ushering the abdominal wall to ascertain the presence of masses or localised pain in the colon or caecum. The ver is normally not palpable. It can be felt only when greatly enlarged. The lymph nodes of the abomasal cavity are felt normally or when enlarged as hard, nodular, variously sized, more or less round bodies, lying in various positions. A large group of lymph gland nodes is always found in the vicinity of the anterior mesentric artery. This group is just within reach of fingers; in certain diseases it may be enlarged and painful.

The peritoneum is palpable only in its posterior position. Normally it is smooth and painful on palpation. In peritonitis, it becomes rough or nodular and palpation causes pain.

Examination of dung: The dung provides a readily available source of information on the state of gastrointestinal tract. So dung should be inspected visually and examined digitally for abnormal components at the time of rectal examination. Form, degree of firmness, colour, presence or absence of blood particles, necus foreign materials, blood and tissue should be noted.

I. Specific examination

a. Pain sensitivity tests for foreign bodies in reticulum:

During these tests, someone must stand at the animal's head or to keep phonendoscope over trachea to ear the painful grunt of animal.
a) Back grip
b) Pole test
c) Pain percussion

By using pain seeking percussion hammer, percuss on the side of chest and abdomen after drawing 5 horizontal and 5 vertical lines. Localize the area of increased sensitiveness and diagnose.

d) Leading the animal up and down a steep slope.
e) Head’s Zone test.

B. William’s reticular grunt test:

Williams by combining auscultation of the reticulum with palpation of rumen, identified the reticular grunt which is a useful piece of clinical evidence. The bell of a phonendoscope held in the left hand and placed at the interspace between the 7th and 8th rib at the level of the costochondral junction will detect a “tinkling” sound, when the reticulum contracts. Simultaneously the rumen will be palpated by right hand in the hollow of left flank to establish co-relation between movements of the reticulum and the rumen. By hearing, the reticular grunt 2 or 3 seconds before primary ruminal contractions, the TRP will be diagnosed.

C. Cardia auscultation:

This technique is used to diagnose the diaphragmatic hernia in early stages. Locate the area of cardia as follows: Draw a slightly curved line drawn from the point of the elbow to the upper aspect of last rib.
which indicates separation of the thoracic and abdominal cavity by diaphragm. Draw another horizontal line from the submandibular region caudally which will bisect the previous line approximately at the junction of the upper and middle third of 8th intercostal space indicating location of cardia. Keep the phonedoscope at the location of cardia, to observe the grunt. Then ask the other man to intubate. In case of diaphragmatic hernia, the animal will evince the grunt when the probang passes the cardia.

D. Pressure palpation:

Applying the digital pressure over the lower part of the 6th to 8th left intercostal space or by deep palpation with clenched fist under xiphoid area, the reticular grunt can be induced in case of TRP.

E. Acoustic percussion of rumen:

By using acoustic percussion hammer to percuss rumen the following diagnosis can be made.

Ruminal atony: Resonant sound in upper 1/3rd and dull sound in lower 2/3rd of left abdomen.

Left abomasal displacement: Metallic sound especially in the middle third of left abdomen in obliquely oval zone, if you percuss on imaginary line drawn from left paralumbar fossa to the left elbow.

Bloat: Drum like sound in the hollow of left paralumbar fossa.

T.R.P. An excessively loud sound may be detected underneath the rumen, close to the xiphoid cartilage (Box sound)

F. Acoustic percussion of reticulum:

Dull sound: Due to thick adhesion large abscess, tumor impaction with sand

Box sound: In foreign body in reticulum.

G. Pain percussion of rumen:

Animal evinces pain in case of rumenitis and large abscess of rumen.

H. Diagnostic procedure of left side displacement of abomasum:

a) Auscultation alone:

Spontaneous sounds typical of displacement of the abomasum are heard by auscultation. In auscultation of the junction of the middle and upper thirds of the left flank on the anterior border of the last rib, a single or a short series of tinkling sounds or high-pitched tympanitic sounds, caused by contraction of gas-filled abomasum are heard. In some cases the sound will be heard readily, but in cases where there is only a slight lower displacement, it may take from 5 to 15 minutes to detect the sound. Since the sound may be heard at intervals of upto 15 minutes, auscultation should be continued for at least that period. The abomasal tinkling sound is like that of silver striking crystal. Similar sounds may arise from a static rumen, but they are audible over a much longer section of the left flank.
b) Auscultation and percussion:

Simultaneous auscultation and percussion has proved to be the most rapid and reliable method of diagnosis. By use of this method each suspected case of displaced abomasum could be checked.

A high-pitched sound is heard which varies in percussion with the acoustic hammer and auscultation of the upper third of the rib cage while the phonendoscope placed on one of the ribs 11,12,13 or on the corresponding intercostal space at the level of the costochondral junction.

This sound is heard only when the phonendoscope lies directly over the displaced organ. Thus by moving the phonendoscope from point to point, noting the presence or absence or resonance, the approximate size and position of the abomasum can be mapped out. The sound may be best described as that of hitting on an empty tin or can be the sound of silver against crystal. Confusion may arise in certain forms of rumen dysfunction in which the rumen contents are of fluid consistency surrounded by a layer of gas as occurs in cases of so-called "vagus indigestion". In these cases percussion and / or ballottment of the left abdominal wall results in resonant sound not unlike those due to a displaced abomasum being heard, but spontaneous sounds resembling those due to a displaced abomasum are not heard in such cases.

e) Auscultation and ballottment:

Simultaneous ballottment and auscultation may stimulate typical sounds of abomasal displacement. Auscultation is performed as before and at the same time the lower left flank is vigorously ballotted in an upwards direction. In case of abomasal displacement a loud ringing and splashing sound is heard.
I. To detect impacted omasum:

   a) PRESSURE PALPATION OF OMASUM: By using the knuckles or the ball of the hand, pressure palpation can be done on the right side between 7th. and 9th ribs being limited anteriorly by caudal edge of lung field to assess the sensitivity. The abnormal sensitivity reveals the impaction.

   b) PAIN PERCUSSION using pain seeking hammer gives clear results than by palpation.

   c) BY APPLYING SWINGING PALPATION the rebound of abnormally heard omasum can be detected.

   d) BY USING POLE TEST the grunt will confirm the impacted abomasum.

J. Acoustic percussion of omasum:

   When omasum enlarged area of dullness will be more

   When omasum is shrunken or forced away from the chest wall - Area of dullness will be diminished

K. Diagnostic procedures on right side displacement of abomasum:

   Percussion and auscultation:

   PERCUSSION FIELDS IN RIGHT SIDE.

   Percussion of the right flank discloses reduction in the area of liver dullness in mild cases, while in advanced cases the area of liver dullness is practically absent.

   In varying degrees of dilation and torsion three different forms may be recognised by percussion and auscultation at the right area which is normally occupied by the liver.

   Simultaneous percussion and auscultation over the above mentioned area is carried out by means of the fingers and a phonendoscope. The procedure involves percussion of the right posterior area. Over the normal liver region and auscultation of the same area will the phonendoscope.
At the onset of dilatation, tympany occurs along the line extending back from the liver, and dullness occurs below the line.

In an advanced and uncomplicated dilatation, the liver is pushed forwards by the dilated abomasum so the area of liver dullness become less than normal, and it is anterior to the usual location. Behind the dullness there is a horizontal limit between an enlarged tympanitic area dorsally and fluid sound ventrally.

At the onset of a torsion the liver is pushed completely away from the rib cage, and the extending abomasum lies between the abdominal wall and later. At a torsion no liver dullness can be detected, and a horizontal limit divides the right flank between tympanitic sound dorsally and dullness ventrally.

Ballotment and auscultation:

Ballotment and auscultation of the right flank at or just below the paralumbar fossa produces gurgling and loud resonant splashing sounds. A loud sound with a resonant tone can be reproduced by a short slap on the right flank. The sound sometimes can be heard at a distance when the animal walks or gets up.

L. Anorectal auscultation and percussion:

Auscultation of the colon and rectum is of utmost value in a routine physical examination. The presence of borborygmus indicates the contraction (Colic) but not motility or transit rate (Diarrhoea). Auscultation percussion will yield a 'ring' under right side last rib. This should not be confused with a right abomasal displacement or a caecal volvulus, both of which can occur and give rise to pain in this area. Rectal examination will confirm the caecal disorders.

III. Special examinations.

X-Ray
Haematological examination
Trocarization
Tapping abomasal fluid in the left side of abdomen
Tapping omasal fluid and abomasal fluid
Testing intraperitonial cavity
Ferroscopy
Endoscopy
Testing the function of the oesophageal groove
Omasal pressure test,
Exploratory rumenotomy
Analysis or rumen, omasal and abomasal contents
Phonoruminography.
Indigestion in cattle constitutes one of the most important problems which causes great economic losses to Indian farmers. Although indigestion is a very common complaint in buiatric practice it represents a challenging diagnostic problem, because of the non-specific nature of manifestations of clinical signs. In day to day practice many diagnosis of various diseases would be difficult without the assistance of various examinations such as those involving milk, blood, urine, dung etc, of the sick animals. It is the rumen with its varied activities that makes a ruminant different from other animals. It is logical therefore to check the physical and chemical properties of the ruminal content, when one is confronted with an anomaly of unknown cause in these animals.

Examination of rumen fluid was introduced for clinical diagnosis during 1950. Some of the tests described below are easy to carry out in day to day practice and they enable biochemical disorders of fore-stomach digestion to be confirmed or excluded. For this purpose sufficient rumen fluid can be collected easily from the sick animals by using Rumen Fluid extraction pump.

**Rumen fluid extraction Pump**

![Diagram of Rumen Fluid Extraction Pump]

Rumen fluid extraction pump (for Cattle and Sheep and Goat) consists of:-

1. A specially designed suction strainer.
2. Three meter nylon tube with perforation at the tip which is covered by a stainless steel spiral tube.
3. A suction pump.
4. An air tight sampling bottle with two way ‘T’ joint
Use of Rumen Fluid Extraction Pump

1. To diagnose various digestive and toxic problems in ruminants.
2. To evacuate the rumen contents in various diseases (Rumen acidosis etc.) as treatment to save the animals.
3. To collect rumen contents from healthy animals for cud transplantation to sick animals to revive the normal flora and fauna.
4. To young calves for quick and healthy growth.

Limitation

If the animal has been starving for considerable time and the rumen content is semisolid in nature, rumen fluid may not be obtained by using the rumen fluid extraction pump. In such cases physiological saline is to be pumped in and massaged well before the collection.

Preparation of the instrument for collection of rumen fluid

1. Unscrew the suction strainer and see that the perforations at the end of the nylon tube are clean. If there is any blockage it should be cleaned. Also make sure that the full length of the nylon perforated end is projecting out.
2. Check the suction pump for its working condition. The washer may be damaged due to shrinkage or brittleness if it is not used regularly. So, to overcome this problem, remove the piston from the suction pump and widen the leather washer by hand and apply either grease or oil.
3. Fix the two way 'T' joint tightly to the sampling bottle to prevent the air leakage.
4. Fix the nylon tube from the suction strainer into the vertical end of the two way 'T' joint. Horizontal end of joint should be connected to the suction pump. If they are not properly fixed, rumen fluid cannot be collected.
5. Check the entire set by dipping the suction strainer into a bucket of water and operating the suction pump.
6. Apply liquid paraffin over the stainless steel spiral sound of the instrument which is ready for the collection.

Important points to be observed before and after the collection of rumen fluid

1. About 200ml of Rumen fluid sample is to be collected for various tests.
2. The rumen fluid can be kept in room temperature for 5 hours and in refrigerator for 24 hrs.
3. After collection, rumen fluid container should be immediately closed air tight
4. During collection discard the first collection of rumen fluid and then collect the sample so that error in pH can be minimised.
**Collection procedure:**

![Collection of Rumen Fluid](image)

**Cattle**

Restrain the animal with nose grips. Open the mouth by pulling out the tongue to one side. Hold the head of the animal high and intubate (introduce) the stainless steel spiral sound with suction strainer until it reaches the rumen. Collect the rumen fluid by operating the suction pump.

**Sheep and Goat**

Restrain the animal in between the knees of an attendant at the level of the forelimbs. Then instruct the attendant to raise the head above, insert the mouth gag across the mouth and intubate the rumen fluid extraction pump by introducing suction strainer through the hole in the mouth gag. Pass over the tongue and past the epiglottis into the cranial part of the oesophagus. At this level, care should be taken not to enter into the trachea and cause any injury. Once you make sure that the suction strainer is in oesophagus, pass further down the oesophagus until it reaches the rumen. You can also visualise the tube passing through the oesophagus on the left side of the neck of the animal. When the suction strainer is dipped into the rumen contents, operate the suction pump and collect the required quantity of rumen fluid.
After use

For longevity and efficient usage of this instrument, clean the suction strainer with suction line and collection bottle in running water without any rumen contents and dry it before packing.

Examination of rumen fluid

The common tests that can be conducted in the Rumen fluid to pinpoint the correct diagnosis

**Colour**

**Odour**

**Consistency**

Sedimentation activity time (SAT) or Stratification

**pH**

**Cellulose digestion test**

**Glucose fermentation test**

**Redox potential or Methylene blue reduction test (MBR Test)**

**Volatile fatty acid, lactic acid, Total acidity (Titratable acidity)**

**Chloride**

**Protozoa**

**Bacteria**

**Nitrate toxicity**

**Hydrocyanic acid toxicity**

Take 100 ml of rumen fluid in a beaker and conduct the following bio-physical examination (A to D) in well lighted area

**A) Colour**

**Normal colours**

**Pure green**

- in grazing

**Yellowish brown**

- in straw feeding

**Grey/Brownish green**

- concentrate and straw feeding

**Abnormal colours**

**Dark brown/Dark green**

- Simple inactivity of flora and fauna, Rumen acidosis.

**Slightly milky**

- Chronic rumen acidosis.

**Milky green**

- Acute rumen acidosis.
Dark Green
- Hydrochloric acidosis
Greenish black
- Vagus indigestion, decomposition of food

B) Odour:

Normal
- Aromatic

Abnormal:

Stale/indifferent
- Inactive rumen juice
Acrid (Acid)
- Lactacidosi/hydrochloric acidosis/Pyloric obstruction
Foul/Putrid
- Protein overfeeding
Slightly ammoniacal
- Rumin alkalosis [urea poisoning]
Musty or faecal
- Vagus indigestion

C) Consistency

Slightly visous
- Normal
Extremely viscous
- Saliva mixed
Watery
- Inactive rumen fluid
Foaming
- Abomasal dilatation or frothy bloat
Mixture of watery and foamy
- Rumen decomposition [E. Coli, Proteus]
Slimy pulp
- Overfeeding
Semiliquid
- Vagus indigestion

D) Stratification or Sedimentation Activity Time (SAT)

100 ml freshly collected rumen contents, filtered through gauze if necessary is observed as it settles a glass cylinder.
Normal

Fine food particles and infusoria - begin to settle at once.
Larger and more fibrous particles - carried upward forming broad upper layer

The above process of complete sedimentation and floatation - takes 4 to 8 minutes.

Abnormal

Rapid sedimentation and absent or retarded floatation - inappetence, starvation and feed without nutritive value
Rapid floatation with abundant foam and Solid components remain in suspension for long time - Decomposition in rumen
Absence of solid particles and gas bubbles. Hence no sedimentation and floatation - Acute rumen acidosis
Pulpy to firm above and fluid below - Traumatic reticulo peritonitis
Absence of stratification - Vagus indigestion

E) Hydrogen ion concentration (pH)

Should be measured immediately after collection without much exposure to atmospheric air. It can be measured by indicator paper in field conditions.

Significance

Physiological variation - 5.5-7.0.
Ration rich in crude fibre and/or protein - Higher range 6.0-6.8
Ration rich in starch or sugar - Lower range (5.5-6.0)
After starvation for 24 hrs. or more - upto 8.5
Urea poisoning
Rumen decomposition
Hydrochloric acidosis - 4.3-7.0 [average 6.3]
Rumen acidosis - 4.0-5.5 Even may reach less than 4.00 in acute cases.

F. Cellulose digestion test (CDT)

Take 10ml of strained rumen fluid and add 0.3 ml of 16% glucose solution in a capped test tube. Suspend a thread of pure cellulose (free from any synthetic fibres) or single strand of unmercerized cotton thread in the rumen fluid. The lower end of the cotton thread is tied with a glass bead or other weight, which must immerse in rumen fluid. Then tightly close the test tube and incubate at body temperature (39°C either in incubator or near a light bulb).
Significance

Normally, digestion of cellulose takes place within 48-54 hrs. So, in fully active rumen fluid the weight in the lower end of cotton thread will fall to the bottom of the tube within that time due to digestion of cotton thread. If the thread has not broken within the normal time it should be interpreted as cellulose digestion time is being delayed due to inactive rumen fluid.

G. Glucose fermentation

It is performed in a fermentation saccharometer. Take 10 ml of rumen fluid and add 0.5 ml of 16% glucose solution in the saccharometer and keep at 39°C. The result is read after 30-60 minutes.

Significance

Normal rumen fluid containing active microflora will ferment the glucose and result in formation of gas.

1-2 ml gas/hr  -  Rumen fluid containing active microflora
No gas  -  Rumen fluid containing inactive microflora and acute rumen acidosis.
Decreased gas  -  Rumen decomposition, rumen alkalosis, acute rumen acidosis.
Normal/increased gas  -  Latent rumen acidosis
Increased gas  -  Foamy bloat
h) Redox potential or methylene blue reduction time (MBR Test)

This is measured by using a redox dye, methylene blue. Take 20 ml of freshly collected rumen fluid and add 1 ml of 0.03% methylene blue solution and mix. Measure the time required for decolouration of the sample using a sample of fluid as a basis for comparison.

**Significance**

It is one of the most reliable tests to assess the microbial status of rumen fluid

In normally active microflora - Methylene blue reduced within 3 minutes

<table>
<thead>
<tr>
<th>Condition</th>
<th>Time Required</th>
</tr>
</thead>
<tbody>
<tr>
<td>Only straw ration</td>
<td>3-6 minutes</td>
</tr>
<tr>
<td>Inactive flora due to ration poor in structure</td>
<td>More than 15 minutes</td>
</tr>
<tr>
<td>Inappetance</td>
<td></td>
</tr>
<tr>
<td>Rumen acidosis</td>
<td>Less than 5 min above pH 5.2 More than 5 min below pH 5.2</td>
</tr>
<tr>
<td>Hydrochloric acidosis</td>
<td>More than 5 min</td>
</tr>
</tbody>
</table>

i) Nitrite reduction

10 ml of strained rumen fluid is placed into each of 3 test tubes and 0.2 (tube 1), 0.5 (tube 2) or 0.7 (tube 3) ml of 0.025% Potassium nitrite solution is added and kept in water bath at 39°C. Every 5 minutes 1 drop from each tube is placed in the small well of a ceramic plate and 2 drops each of reagent 1 (2 ml of sulfanilic acid in 30% acetic acid to make 200 ml), and reagent II (0.6 ml alphanaphthylamine, 16 ml concentrated acetic acid, 140 ml distilled water), until the disappearance of red colour which will provide information on the activity of microbes that degrade and synthesize nitrogenous compounds. The presence of red colour indicates that still nitrite is present.

**Significance**

Rumen fluid from cattle fed with mixed ration - Nitrite should disappear in

<table>
<thead>
<tr>
<th>Condition</th>
<th>Time Required</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rumen fluid from cattle fed with mixed ration</td>
<td>5-10 minutes- tube 1</td>
</tr>
<tr>
<td></td>
<td>120 minutes-tube 2</td>
</tr>
<tr>
<td></td>
<td>30 minutes-tube 3</td>
</tr>
<tr>
<td>Rumen alkalosis, Green fodder</td>
<td>Rapid reduction in all tubes</td>
</tr>
<tr>
<td>Ruminal decomposition, Bloat, lack of appetite, deficient ration, Hydrochloric acidosis, Acute lactic acidosis</td>
<td>Slow reduction in all tubes</td>
</tr>
<tr>
<td></td>
<td>No notable reduction even after 45 minutes</td>
</tr>
</tbody>
</table>

J. VOLATILE FATTY ACIDS (V F A)

For every 20 ml of rumen fluid, 1 ml of saturated mercuric chloride solution is added and sent to the laboratory for determining total and individual fatty acids.
Normal:

<table>
<thead>
<tr>
<th>Acetic acid</th>
<th>-</th>
<th>50-65 mol%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Butyric acid</td>
<td>-</td>
<td>10-20 mol%</td>
</tr>
<tr>
<td>Formic, valeric, caproic and high fatty acids</td>
<td>-</td>
<td>15 mol%</td>
</tr>
</tbody>
</table>

VFA concentration increased on concentrate ration and also 3-5 hours after feeding.

VFA concentration decreased on completion of fermentation (pH increased).

Abnormal:

- Loss of appetite, Ration poor in structure, Digestive disorders.
- Total VFA decreased
- Increasing amounts of easily digestible carbohydrate - Proportion of individual acids change.

### CHLORIDE

To 0.1 ml of the chloride standard solution, add deionized water 1 ml and put 0.2 ml indicator. Then titrate the standard with mercuric nitrate solution. The end point is slight, but permanent violet colour. This will be the standard. Repeat the same with rumen liquor sample.

Calculation

\[
\text{Test reading} \times 100 = \text{-m Val/litre.}
\]

This is useful in distinguishing hydrochloric acidosis from lactic acidosis

**Normal level of chloride content in rumen fluid**: 15-25 mVal/litre.

**Abnormal**:

- Lactacidosis - Less than 40 mVal/litre.
- Hydrochloric acidosis - More than 25-30 mVal/litre.

May increase to 30-100 mVal/litre in

a) reflux of abomasal content as a result of obstruction
b) supplementation of ration with sodium chloride
c) functional or anatomical pyloric stenosis
d) abomasitis
e) abomasal ulcer
f) sand in stomach or intestine
g) abomasal displacement
h) cellulitis of mesentery at its attachment to the abomasum
i) Paralytic ileus

L. Total Acidity (Titratable acidity)

One or 2 drops of phenolphthalein are added to 10 ml of rumen fluid and the mixture is titrated with N/10 NaOH until it becomes flesh coloured. The volume of NaOH required (in ml) multiplied by 10 gives clinical units of total acidity:

Normal rumen fluid - 8.2 clinical units

Significance

In hyperacidity (lactic acidosis or hydrochloric acidosis) Upto 70 units
Lactacidosis - significantly increased.
Hydrochloric acidosis - moderately increased.

M. PROTOZOA

Both ciliates and flagellates are present in rumen. But only ciliates are of physiological importance in virtue of their numbers and mass. The majority of ruminal protozoa belong to the family Ophryoscolecidae. Their numbers and size distribution provide information on protozoal activity within the forestomach.

Place a drop of rumen fluid on a slide and cover with a cover glass and examine under lower power (80 or 100 magnification)

Assess the following Characters:

1. Density

vigoroum - ++++
Abundant - +++ (More than 30 Protozoa/low power field 100 x)
Moderate - ++ (10-30 Protozoa/L P F)
Few - + (1-10 Protozoa/L P F)
None -

More the density of protozoa, more active is the rumen fluid.

Simple inactivity of flora and fauna
Rumen decomposition/alkalosis
Acute rumen acidosis

2. Proportion of large, medium and small infusoria:

Large and medium ciliates in large numbers
Only small ciliates

Active rumen fluid
Forestomach indigestion
Proportion of dead to live protozoa:

- Severe digestive disorders like Rumen acidosis (pH less than 5) - Entire microfauna dead.
- Moderate digestive disorder - Proportion of dead to live increase.

Iodophilic activity:

Add a drop of Lugol’s iodine to 2-3 drops of rumen fluid on a glass slide and place a cover slip on it and examine under the microscope. Iodophilic activity of protozoa can be recognised by black colouration of starch contents in the protozoa. It can be graded as 0, +, ++, +++ depending on quantity of starch contained. The quantum of starch content of protozoa reflect the degree of digestive disease.

5. Total protozoal count

5 ml of strained rumen liquor (S R L) is diluted to 20 ml with 10% formal saline. From this, 10 ml of the mixed rumen liquor is taken and 10 drops of 2% Eosin is added to colour the protozoa. The diluted rumen liquor is charged in a haemocytometer with Neubar ruling. 8 sq. m.m is counted and its average is multiplied by 50,000. The result is expressed as total counts per ml ($n \times 10^5$).

Normal counts:

- Mixed ration - $1 \times 10^5$/ml
- Concentrate ration - $1 \times 10^6$/ml
- In disease - Decreased or Nil
- Bloat - Decreased total count, disappearance of Holotrichs
- Acidosis - Nil; if present only few Entodinia sp.

N. BACTERIA

An air dried smear of rumen fluid is strained by Gram’s method and observed under the microscope.

Basic criteria for interpretation are:

1) Presence or absence of morphologically distinguishable bacterial species characteristic of normal rumen flora, the so called leading bacteria.

2) Multiplicity or uniformity of forms.

3) The ratio of gram positive to gram negative bacteria.

4) Comparison of the smear from the sick animal with that of the healthy animal receiving the same ration.

In the normal pH of rumen fluid - Gram negative bacteria is dominant.

- Rumen lactacidosis - Proliferation of gram positive cocci and rods at the expense of gram negative bacteria.
- Hydrochloric acidosis - Gram positive less than Gram negative bacteria.
Total bacterial counts

Add 5 ml of strained rumen liquor (SRL) and dilute to 25 ml with 10% formal saline. Take 1 ml of this fluid and again diluted 100 times with 0.85% saline. 0.01 ml of this sample is spread over an area of one square centimeter making use of the guide plates. After the smear had dried, it is stained with Newman's stain. At least 30 fields should be counted in each smear and the average count is to be multiplied by the dilution factor and microscopic factor. The result is expressed as total counts per ml (X x 10^7).

Significance: Normal 10^7 to 10^12/ml.

NITRITE/NITRATE TOXICITY

Modified diphenylamine test: A drop of the test solution (0.5g of diphenylamine, 20 ml distilled water and concentrated sulphuric acid sufficient to make 100 ml) is mixed with a drop of rumen fluid. An intense blue colour within 10 seconds indicates a concentration of greater than 6% nitrate which is more than the permissible level.

HYDROCYANIC ACID TOXICITY

Reagent papers are prepared by mixing 0.5g of picric acid and 5g sodium carbonate in 100ml water. Filter papers are dipped in the reagent and allowed to dry in a dark place. A drop of rumen fluid is placed on the test paper. A red discolouration is a positive reaction. The test is designed only to detect free hydrocyanic acid. It may not be positive when cyanides are present in different forms.
MACROSCOPIC ANALYSIS OF DUNG

Dr. S. PRATHABAN and Dr. P. DHANAPALAN

Bovine faeces is an "information carrier" as it travels through the entire digestive tract of our patient and deserves proper attention. This is particularly true because the macroscopic findings obtainable from faeces are not only indicators of diseases of the digestive tract itself, but also can provide valuable directional clues for the differential diagnosis of diseases elsewhere.

Quantity of the faeces

Healthy pre weaning calves on milk of 8 litres pass an average of 50-60gm of faeces, Periods of 2 to 5 days without bowel movements are by no means unusual in this age groups. In adult cattle the passage of ingesta through the digestive tract takes 1.5 to 4 days, due to the food reserves stored in their system of fore stomach, "Fasting faeces" without fibre particles are not observed in cattle even after a period of starvation. Mature cattle generally pass some faeces every 1.5 to 2 hrs amounting to a total of 30 to 50 kg per day in 10 to 24 portions.

Reduction in the bulk of faeces

I) Interference with the feed and water intake
II) Interruption (or) retardation of the passage through the alimentary tract
III) Increased resorption of fluids in the large intestine.

Diagnosis

a) Check the prehension, mastication and swallowing of the feed,
b) Examine the forestomachs (for signs of functional or anatomical stenosis or omasal paresis)
c) Percussion and auscultation of the right and left abdominal walls (To establish any displacement of abomasum, small intestine or caecum)
d) If the condition is accompanied by colic, rectal examination.
e) If there are any indications of intestinal obstruction (traces or absence of faeces in the rectum, instead presence of tenacious pasty yellowish or grey white (or) reddish to brown - black mucus), the patients abdominal walls should be lifted (preferable with a plank) to allow palpation of the affected segment of intestine by rectal examination.

f) Explorative laparotomy

Apparent increase in faeces

Collection of stagnating masses of excreta in the rectum.

Paralysis of bowel segments concerned, as well as tail and bladder (spinal cord lesions, dystocia, after mating, due to mutual mounting of cows during heat, other traumatic injuries, tumours etc.)

Clinical findings:- Exploring hand may find that anal contractions are weak or have ceased altogether, rectal tube is flabby, distended and bulging with faeces. Similar findings are also seen in mild fever after surgical opening of the abdomen.
Colour of the faeces

The colour of the faecal material is influenced by the nature of the feed (particularly Chlorophyll content), the bile (stercobilin) secreted into the ingesta by the passage rate through the digestive tract and occasionally also by admixtures of other substances.

Calves reared on milk alone - golden yellow faeces
when hay (or) straw is ingested - pale - brown
Disease (scour) - grey white to grey yellow
Adult cattle on green forage - olive - green
Adult cattle (hay straw ration) - olive - brown
Adult cattle (concentrate ration) - olive - grey
Haemolytic Jaundice - dark olive - green to blackish green (other symptoms halmoglobinuria, water added to the dung is quickly discoloured to yellowish - green colour)
Obstructive jaundice - pale olive (Absence of bile other symptom enlarged liver percussion field)
Abomasal cler (common sequel to Theileriasis). - tarry coloured
Intestinal haemorrhage - chocolate brown to blackish tarry appearance
Rectal haemorrhage - Red brown colour with clots and redstreaks
Ruminal acidosis - light grey - green colour
Drugs - charcoal - Black colour

Odour of the faeces

primary diarrhoea :- evil smelling with gas bubbles.
secondary diarrhoea :- do not possess grossly abnormal odour, and usually well digested.
lactoacidosis :- sour pungent smell.
poisons - can be identified on the basis of faecal odour.

Consistency of the faeces

Depends on

1). Fluid in the ration
2). Length of sojourn of ingesta in the large intestine.

Calves normally pass faeces with firm porridge like consistency, which turn slimy or greasy to pasty when milk substitutes are fed.

Normal bovine faeces are medium porridge like consistutency, on hitting the ground they form 1 or 2 roundish dung of the size of dinner plates which are evenly distributed without any appreciable amounts of squirting sideways.
moderate thickening (Faecal discs) - Inadequate water supply.
faecal balls - severe dehydration stasis (along the gut) functional peritonitis (urine output is also less or pain or grunt evinced on urination) ketosis (urine output more, clear colourless urine)

These faecal balls appear shiny as they are coated with mucus.
mucus plug paralytic ileus (scanty faecal material covered with mucus)
sticky, tenacious excreta made up of chronic regional enteritis coccidiosis mucus and fibrin
Foamy, whipped creamlike consistency lactacidosis
Reduction in consistency diarrhoea

Degree of communition of faeces

This is judged by the length of the undigested fibre in the dung.

1 to 2 cm - poorly digested
0.5 cm - moderately digested
5 cm - well digested.

poorly digested:
- a) failure of rumination (faulty dentition)
- b) accelerated passage of feed (common in recent TRP)

moderately digested: - Normal function
well digested: - Abomasal displacement, spiral colon, caecal dilation and volvulus.

Admixtures of other substances
mucus plug - Ileus
Long strands of fibrin - Croupous enteritis
Glassy particles free
mucus with water, thin consistency (sticking the floor) - Croupous enteritis (salmonellosis)
Fibrinous plugs - From inflamed bladder.
### Haemorrhage

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Swallowing large quantities of blood</td>
<td>- Tarry coloured</td>
</tr>
<tr>
<td>Haemorrhage into the rumen (rare, due to a FB or necrotising erosion)</td>
<td>- Tarry coloured, Check CBC, lymph nodes, physical examination.</td>
</tr>
<tr>
<td>Haemorrhagic enteritis</td>
<td>- highly offensive odour check feeding, faecal culture</td>
</tr>
<tr>
<td>Tumours in the intestinal tract</td>
<td>- recurrent melena, colic symptoms (rectal exam, laprotomy from rt.flank)</td>
</tr>
<tr>
<td>Micromelena</td>
<td>- Young animals - coccidiosis, Adults - regional enteritis.</td>
</tr>
<tr>
<td>Sand</td>
<td>- sand can be identified by dissolving the dung in water</td>
</tr>
<tr>
<td>Orally ingested poisons</td>
<td>- Not visible to the naked eye. A film of oil on the faeces will be suggestive of poisons</td>
</tr>
</tbody>
</table>
Among domestic farm animals the metabolic diseases achieve their greatest importance in dairy cows and pregnant ewes. The high producing dairy cow always verges on abnormality and the breeding and feeding of dairy cattle for higher milk yield is etiologically related to the diseases of metabolism. Because of their high prevalence and the high mortality, predictive systems are being set up. The interesting development has been the recognition of production diseases and the consequent development of metabolic profile tests.

The term 'metabolic profile' was introduced by Payne et al. (1970) to describe a system of serological monitoring of dairy herds as an aid in the assessment of their nutritional, metabolic and health status.

Basic concepts:

The traditional system of assessing the nutritional status of a dairy cow is to calculate the amount of nutrients being supplied and compare this with the requirements of the animals. In practical circumstances it may be difficult to estimate the quantity of food being eaten. In addition, nutritional interaction leading to variations in absorption and utilization of nutrients cannot always be allowed for, or indeed may not even be known. The metabolic profile test is intended to be a measure of balance between 'input' in terms of nutrients absorbed from gastrointestinal tract and 'output' in terms of requirements of those nutrients for maintenance, pregnancy and lactation. Therefore if 'input' is adequate to match the demands of output then levels of metabolites in the blood may fall. Initially, this fall may be slight and not manifested as a clinical abnormality. As the reduction in metabolic concentration progresses, there comes a stage when it may be seen as clinical disease. The metabolic profile test can therefore be used both as an early warning indicator of subclinical disease and as a diagnostic procedure. Properties of an ideal biochemical parameter for the metabolic profile test are:

1) The metabolite should be stable in the blood sample for a considerable period after collection.
2) It must be possible to analyse the metabolite accurately and the amount of laboratory error must be small in proportion to other possible sources of metabolic variations i.e. the herd, milk yield, etc.
3) The metabolite should be consistently related to nutritional status or some other well understood metabolic pathway and variations due to diurnal, prandial and other factors should be kept to minimum.
4) Factors such as age, sex, genotype and environmental stress should have no significant influence on the metabolite.

Objectives:

1. To monitor the health status of the herd.
2. To assess input, output and throughput relationship or to detect the qualitative and quantitative adequacy of the diet in a farm.
3. To diagnose nutritional imbalance and metabolic production diseases in early stage.
4. To select superior and healthy animals for purchase.

When to sample?

1. When there is change in the diet.
2. At the end of winter

3. At the end of summer

4. Preferably every month.

If a herd is being sampled on a regular basis, then blood samples should be taken at the same time on each occasion, to try to reduce the effect of prandial and diurnal variation.

One very important factor is that if dealing with a problem herd, the unaffected animals should be sampled. For example, if there is a rapid reduction in the yield from an early or disappointing peak, then affected cows that have dropped from peak have already reduced their yields to match the food intake being received and as such their blood levels will be returning to normal. It could be said that these animals have already compensated for the poor feeding. The correct animals to sample in this instance would be those cows that have reached and are maintaining a reasonable peak yield, since they are the animals that are most likely affected by an imbalance between input and output. It is a failure to appreciate this critical point that has resulted in many people finding little value in the metabolic profile test.

**Animals to be sampled:**

Cows in early lactation (4-8 weeks after calving) are to be sampled, because they would be more likely to produce economically beneficial results for the following reasons:

1. They are approaching the period of service, when nutrition needs to be at an optimum.

2. Peak yield is also reached at this stage and the importance of reaching a satisfactory peak to achieve an overall high lactation production is well known.

3. Cows are sampled before the period of "equilibration".

It is clearly important that normal cows for that particular herd are sampled. Any animal that is an exceptionally low or high yielder, has a long standing health problem, e.g. lameness, is in estrous, or has any other condition that might affect the blood chemistry should not be used.

Although a fairly specific stage of lactation has been suggested, there will be instances when other groups of animals need to be sampled. For example, in a herd with a high incidence of milk fever, late pregnant cows and if possible, a few clinically affected animals should be sampled. If fatty liver syndrome is suspected, blood samples taken at 7-14 days after calving and analysed for glucose and aspartate amino-transferase (AST) have been found to give the best prediction.

**Site of sampling:**

Blood may be taken from jugular or coccygeal vessels, although the site of sampling must be stated and the correct normal values used. Although there is statistically significant variation in blood concentration between the two sites for a wide range of metabolites, only the difference in blood phosphorous is great enough to affect the interpretation of the metabolic profile. Phosphorous levels in jugular blood are approximately 15-20% lower than in coccygeal blood, due to the drain of phosphate by salivary gland for its use as a ruminal buffer. Potassium levels are also approximately 5% lower than in coccygeal samples. The mammary vein should not be used as a sampling site since glucose levels especially tend to be lower and very variable.

1. Blood samples should be collected preferably at morning hours between 9.00 AM to 12.00 Noon, with appropriate anticoagulants.
2. Blood samples should be collected at the same time of day at every collection.
3. Samples should be collected with minimum excitement of animals.
4. If necessary urine samples should be collected from lactating cows to test for ketones and results should be correlated with blood glucose levels of each cow.
5. Rumen liquor, faeces and milk sample examinations are also suggested if necessary.

**Various forms of the herd metabolic profile test:**

I. Compton metabolic profile test

13 metabolites are used by Payne et al. (1970)

1. PCV
2. Haemoglobin
3. Albumin
4. Blood glucose
5. Blood urea nitrogen
6. Total protein
7. Calcium
8. Inorganic phosphorus
9. Magnesium
10. Potassium
11. Sodium
12. Copper
13. Iron

Kronfeld (1972) suggested 12 metabolites as alternative.

1. Haemoglobin
2. Blood glucose
3. Albumin
4. Total protein
5. Calcium
6. Phosphorus
7. Magnesium
8. Free fatty acids
9. Acetoacetate
10. B-hydroxybutyrate
11. Acetate dehydrogenase
12. Lactate dehydrogenase.

II. Mini metabolic profile test (Blowey et al., 1975).

3 metabolites

1. Glucose
2. Albumin
3. Urea nitrogen

III. Individual preventive examination


1. SGOT 1. SGOT 1. Aspartate amino-transferase (ASAT)
In the classical metabolic profile (compton) assessment, samples are obtained from cows which are representative of the 3 principal stages in the production cycle viz. (1) dry cows representing the maintenance component, (2) mid-yielding cows in mid-lactation and (3) high yielding cows at the peak of lactation.

In the so called 'Mini-profile test', cows are sampled at monthly intervals. The cows are between 4-8 weeks of post-calving at the time of bleeding, so as to (1) eliminate variations in blood levels due to the stage of lactation, (2) to permit correction of feeding imbalance during early lactation so as to improve overall lactation production and (3) to coincide with the period of conception.

In the individual preventive examination, the blood is sampled at 8 weeks prior to calving. It is considered that this procedure offered useful data concerning the susceptibility of each cow to the parturition syndrome i.e. metritis, retained placenta, hypocalcemia, the downer-cow syndrome and mastitis, because the parameters measures were considered to indicate the stage of energy balance and the liver efficiency of each animal. This concept of dairy herd monitoring emphasise on its diet and its production demands, and has found support in the Federal Republic of Germany as a prelude to the more classical profile approach.

Interpretation of results:

Blood results need to be considered in conjunction with a whole range of supplementary data including the following:

1) Details of individual animals sampled (age, stage of lactation, yield, etc)
2) A working knowledge of the ration, to enable a diet balance sheet to be calculated.
3) Cow condition, consistency of faeces and general cow contentment.
4) Total herd milk production and milk quality.
5) The reason for carrying out the metabolic profile.
6) Any clinical signs being shown by the animals sampled or any other animal should be noted.

Merits and demerits:

I. Compton metabolic profile test

Merits:

1. It is useful in the assessment of input-output relationship which is an attractive tool for veterinarian who is engaged in complete health cover of a herd.
2. It is able to detect qualitative and quantitative adequacy of the diet of cows expected to produce certain quantity of milk or return to oestrus within a desirable length of time following parturition.
3. It is a reliable test for early diagnosis of nutritional deficiency (nutritional imbalance) and metabolic disease (Production disease) which would be a major step forward in attempting to optimise livestock production and obtain maximum yield in minimum cost.
4. It aids in selection of superior individual.
Demerits:

1. Results are difficult to interpret by all veterinarians except by specified persons.
2. Test must be carefully planned. But it is expensive.
3. A regional diagnostic laboratory with automated analytical equipments should be available in the campus of the farm.
4. The test should not be undertaken unless the normal values for each laboratory measurements are available from the population within the area. The results from the groups are compared to the local population means.
5. It is difficult to identify the common causes of variation. The most consistent variation occurs between herds and next between lactational groups within the herd. It is important to note that the stability of the sample is in relation to time in transit.

Mini metabolic profile test:

Merits:
This test is conducted four weekly in order to assess the adequacy of protein, energy intake, and optimise nutrition for maximum production. Since samples are tested only for limited metabolites it is cheaper.

Demerits
Unable to assess the lipids, mineral and electrolyte metabolism.

II. Individual preventive examination

Merits

1. Less number of parameters
2. Easy to adopt in field conditions
3. It is assessing lipid, carbohydrate and protein metabolism, liver and rumen function.

Demerits

It is difficult to examine individual animals in a bigger farm.

Blood parameters measured

Energy status

Glucose:

It is subject to fairly precise homeostatic control. Hence, large changes in the rate of glucose utilization i.e. the throughput rate, are initially reflected in only relatively small changes in plasma glucose concentration. Glucose can only be considered as an indicator of energy status in lactating or late pregnant animals. There are a number of non-nutritional factors that elevate glucose level e.g. stress, excitement, severe cold, corticosteroid therapy etc.
Non-esterified fatty acids: (Free fatty acids, FFA)

In response to underfeeding, an animal mobilises body reserves by hydrolysing the neutral fat molecule. The long-chain fatty acids (NEFA) thus produced pass to the liver for degradation and subsequent release of energy. A high concentration indicates excessive mobilisation of body fat and hence an energy deficit. They are subject to considerable diurnal and prandial variations and also increase in rapidly with stress and excitement.

B-Hydroxy butyrate:

B-Hydroxy butyrate and other ketone bodies also increase in response to underfeeding and they are not affected by stress. Thus it is the most commonly used parameter.

Protein Status:

Urea:

Urea levels in plasma are primarily derived from rumen ammonia, although a certain amount will also arise from the hepatic deamination of amino acids. Part of the circulating urea is excreted via kidney, although a part is also recycled into the rumen, particularly at low plasma urea levels, via both saliva and directly across the rumen wall. The factors that lead to an increase in urea levels are:

1. An increase in protein intake.
2. An increase in the proportion of RDP in the ration.
3. A decrease in energy intake, leading to depressed rumen microbial ammonia assimilation and an increased leakage of ammonia from the rumen.
4. An increase in ruminal pH, again allowing a greater leakage of ammonia from the rumen, since free ammonia diffuses across the rumen wall more rapidly than ionised ammonium (NH₄⁺) radicle.
5. Increased body tissue catabolism and/or renal failure.

Albumin:

It is used as an indicator of long term protein status and of UDP intake. Factors that can lead to a depressed serum albumin include:

a) Reduced protein intake
b) Stage of lactation: Albumin values fall sharply after calving in some cows, but by no means all animals are affected. A more rapid rate of recovery as lactation proceeds has been correlated with improved fertility and higher milk yield.
c) A recent increase in protein intake, urea levels will rise within 2-3 days, but albumin may take several weeks to reflect the change.
d) Decreased energy status, depressing the rate of rumen microbial NPN assimilation.
e) Chronic infection, leading to elevated globulins and a compensatory decrease in plasma albumin levels.
f) Any chronic liver damage could depress albumin synthesis. eg. fatty liver syndrome.
g) Chronic intestinal parasitism.

Other blood parameters that have been found to be useful indicators of protein status include PCV, haemoglobin and total protein, all of which may take several weeks to respond to changes in dietary protein intake.
General status

Copper: is a poor indicator for the metabolic profile test, as the firm homeostasis is recognised.

Magnesium: Low levels indicate dietary deficiency, but at very high intake blood magnesium rises only to an upper limit and excess is excreted in the urine.

Phosphorus: Difficult to interpret. There are significant diurnal and daily fluctuations in plasma phosphorus levels that a series of blood samples over a limited period of time would be needed to assess dietary status.

Selenium: They are best assessed by sampling pregnant animals, receiving no supplementary concentrations, since such products normally contain quite high levels of trace elements.

Cobalt: Interpretation difficult. Although serum B12 is a reasonable indicator of cobalt status in sheep, no correlation in cattle is poor.

REFERENCES


RADIODIAGNOSIS OF G.I. TRACT DISORDERS IN ADULT RUMINANTS

Dr. N.N. BALASUBRAMANIAN

In bovine the extent of radiographic investigation employed to detect G.I tract problem is influenced by the following considerations.

1) Economic Consideration 2) Practicability 3) Size of the animal 4) Skill of the persons taking x-rays and availability of ancillary apparatus (Use of special intensifying screens, Grids etc) and availability of means to align the primary beam accurately with the grid. In the adult cattle to diagnose G.I. tract problems, the lateral view of the ventral thoracic field to cover the heart and diaphragam is the only practicable projection that is undertaken because of the above cited reasons.

Traumatic reticuloperitonitis and Diaphragmatic hernia are the common diseases of cattle where radiographic evaluation aids in establishing a definite diagnosis and in influencing the manner of treatment.

Conventional radiography of the reticulum is conducted with the animal in standing position. The horizontal beam is centered on the reticulodiaphragmatic caudal pericardial region and include the area confined ventrally by the sternum, and dorsally by the caudal vena cava. The purpose of this examination is to determine whether there are metallic (radiodense) or nonmetallic (radiolucent) foreign bodies. This projection can also be utilised to diagnose foreign bodies in the pericardium and diaphragmatic hernia (D.H). The information gained by the standing reticulography is often obscured by fluids in reticulum, pleural space or pericardial sac. Further, the forelimbs of cows or buffaloes cannot be easily pulled cranially when standing which result in superimposition of forelimb structures over the area of radiographic interest thereby decreasing the ability to see a foreign body. Lateral view with the animal in lateral recumbent position or dorsal recumbent position with the forelimbs stretched forward will show in a normal radio graph the part of the sternum, diaphragmatic dome, cardiac silhouette, reticulum and a portion of lung more clearly. When compared to lateral standing reticulography, it gives definite evidence of penetrating foreign bodies in reticulum, because they are surrounded by gas in reticulum. Radiographic diagnosis of D.H. in cows and buffaloes by taking lateral view of the reticulum area with the animal controlled in right lateral recumbency and the forelimbs stretched forward, is possible. The cassette (15" X 12") with or without grid (1:10) can be placed beneath the chest and behind the elbow. The cassette should be adjusted in such a way so as to cover the area from the 6th rib backwards and sternum upwards. Both plain and Barium contrast radiography can be taken with FFD. (100 cms), 80 to 120 kVp and mAs varying from 60 -160 depending on whether grid and contrast is used. For Barium investigation 1 kg of Barium sulphate mixed with a litre of water is to be drenched and radiographs taken after 20 minutes. A typical case of D.H. on plain radiograph shows interruptions of the diaphragmatic dome. Part of the reticulum is also seen herniating into the thoracic cavity. Pattern of reticular mucosa (honey combed structure) is noticeable. Frequently radio opaque objects can also be seen in the herniated portion of reticulum. A cluster of metallic objects accumulated on the thoracic side can give a fair indication of herniation.
ULTRASONOGRAPHY IN VETERINARY PRACTICE

Dr. R. RAMASAMY

SOUND: The word sound conveys a double meaning. It refers to

I) the mental sensation perceived by the ears and

II) the cause responsible for that perception, namely, the physical phenomenon external to the ear - the
Wave Motion which excites the auditory nerves.

The sound is due to the vibratory movement of some body, although the movement may be so slight and
rapid that is not visible.

ULTRASONICS: The sound waves in which the frequencies are above the limits of human audibility,
i.e. greater than 20,000 cycles per second, are referred to as Ultrasonics. Sound waves whose velocities are about
700 miles per hour are known as ‘Supersonics’. High frequency vibrations have generally been obtained
depending upon either Piezo-electric or the Magnetostriction effects.

ULTRATALK: A working knowledge of ultrasound terms provides not only the ability to understand
the literature of ultra sound but to communicate with others concerning specific sonographic observations.

- Echogenicity - Pertaining to echo strength.
- Anechoic - No echo (Black)
- Hypoechoic - Weak echo (Gray)
- Hyperechoic - Strong echo (White, Bright)

Reflection - Echo resulting from the ultrasound wave bouncing off a tissue interface and returning to
the scanner.

- Transducer - A device which emits high frequency sound waves and records returning echoes.
- Coupling Gel: A water soluble gel placed on the skin surface and / or the transducer head which
eliminates intervening air, thus ensuring unimpeded sound transmission.

The application of diagnostic ultrasonography to evaluate various conditions becomes increasingly
useful in veterinary practice. The ultrasonographer is an integral part of each ultrasonic examination on three
distinct levels.

i) The eye - to hand coordination necessary to locate the organs is the most basic level. It is relatively
easily learned. It requires only a few seconds in most cases. Difficulties, occasionally may arise when structures
such as a loop of bowel or fecal material disrupt the image,

ii) Ultrasonic anatomy must be evaluated. This requires a detailed working knowledge of the
concerned organ,

iii) Finally the basic principles of diagnostic ultrasound and the biological subject under study must
be constantly integrated.

Scanning technique, instrument adjustment, anatomic structure, and an understanding of the basic
concepts of acoustic physics combine to yield a technically correct and aesthetically pleasing ultrasound image.
The principles of ultrasonography are based on the ability of sound waves to be either reflected from or propagated through various tissue interfaces. The body is composed of many layers of tissues. A tissue interface occurs whenever tissues of different densities are in contact. When a pulsed electric current is applied to the piezo electric crystals in a transducer, vibrations characteristic of the crystals are produced. Tissues have the ability to either propagate or reflect the sound waves to varying degrees. Ultrasound waves travel through the body (approximately 1540m/Sec.) If the sound wave hits a barrier, echo may be produced. Echoes returning from the tissues are electronically captured by the receiver in the transducer. Echoes are converted into electrical impulses and displayed on the ultrasound screen.

Procedure: When scanning it is vital to achieve good contact between the skin and the transducer. The area must be clipped. If the skin is dirty or greasy it should be cleaned with spirit. Then a contact gel should be liberally applied. If the arc shaped or horizontal white lines mar the image the skin/transducer contact is not adequate. The preparation procedure should be repeated.

MEDICAL AND BIOLOGICAL EFFECTS OF ULTRASOUND:

1) The vibrations of the waves are used to produce a soothing massage action on affected joints. It is an useful tool in the treatment of muscular pain.
2) It is used to cure neuralgic and rheumatic complaints.
3) The most revolutionary use of this sound is in the treatment of mental patients.
4) Ultrasound imaging technology provides rapid, noninvasive access to the internal organs. The potential for assessing the internal organs in the same animals over time has afforded an unprecedented depth to investigations, dynamic changes in biological structures (heart, liver, spleen, kidney, reproductive organs etc.)

APPLICATIONS OF DIAGNOSTIC ULTRASOUND:

Liver: The liver normally appears relatively echoluent. Focal abnormalities are readily recognised as they cause disruption of the normal uniform liver parenchyma. Hepatic neoplasia, whether primary or secondary, may cause multiple focal areas of increased and decreased echogenicity. A similar picture may be seen in advanced cirrhosis.

Spleen: The normal spleen has a smooth contour and a uniform parenchyma which is slightly more echogenic than the liver. Focal lesions are readily seen as they disturb the normal homogeneity of the parenchyma. These lesions may be predominantly hypoechoic, hyperechoic or mixed.

Uterus: The normal non-pregnant uterus is not visible in the bitch or queen. However in cattle the cervix is easy to locate due to the increased echogenic response of the plicae circulares.

Cleaning ultrasound Transducers: Without proper care of the transducers, their life span can be shortened. So proper care of these instruments is essential. Proper cleaning, disinfecting and sterilization of an ultrasound transducer takes minimal time and effort. In the long run this can help and prolong the life of the probe.

To decrease the chances of having coupling gel dry into the transducer, prompt removal of the gel is necessary. This is accomplished by simply wiping the probe with a soft towel. In most instances only the face of the transducer can be disinfected. Disinfection involves wiping the face of the transducer with a cloth moistened in the disinfecting solution. When a sterile probe is needed, covering the transducer with a sterile sleeve works well.
LIVER BIOPSY IN RUMINANTS

Dr. B. NAGARAJAN

Liver is an organ of many diverse metabolic activities. So it gets involved in most of the diseases. Liver has vast functional reserve. Clinical liver disease occurs only when hepatic reserve become inadequate when atleast eighty percent of the liver is damaged and can no longer support normal functions. Usually no clinical signs of liver disease occur, until there is a failure of one or more of the liver's function.

The diagnosis of liver disease is difficult, but evaluation of its severity is even more difficult. Liver function tests will not reveal exact pathological (eg. Fatty change, necrosis, fibrosis or cirrhosis) conditions of the liver which is more important for treatment and for giving prognosis. Cytological examination of liver cells has a prognosis value since it is possible not only to demonstrate the presence of liver lesions but also evaluate the severity. The collection of small pieces of liver tissue from live cattle can be performed blindly without seeing the liver or under visual control by laparatomy. Liver biopsy can be done easily without any difficulty at any place. The technique requires some skill and anatomical knowledge. Liver biopsy has evolved as a valuable aid to clinical evaluation of patients with hepatic disease because of the need for diagnostic specificity as well as the fact the morphological alteration constitute the principle criteria for classifying most hepatic diseases. It confirms the liver disease and in many cases it determines the etiology. Diffuse and Zonal lesions occurring in most of the toxic, infectious and metabolic liver diseases can usually be diagnosed on liver biopsy. The only way that a definite diagnosis and thus biopsy may represent only one fifty-thousandth part of the organ. Focal lesions may be easily missed. However, modern diagnostic procedures such as radio- isotope and Ultrasonic scans and computerized tomography have greatly increased the chances of detecting and localizing focal disease.

The combination of fine needle aspiration and real time imaging with ultrasound may make biopsy a much more commonly practised procedure in next few years.

Liver biopsy technique is also useful for various biochemical and clinical investigations such as diagnosis of fatty liver, subclinical ragwart poisoning, estimations of Vitamin A, Vitamin B12, Copper, Zinc, Molybdenum, Manganese, Lead, Cadmium and drug residues, determinations of lipid, protein and glycogen content of the liver and to find out hepatic concentration of cytochrome p450. It is also useful in allograft dysfunction following liver transplantation.

Prebiopsy considerations

It is essential to evaluate each animal before doing needle biopsy in order to determine whether there will be unwarranted risk associated with biopsy procedure. In addition to a detailed clinical history and physical examination, the status of hepatic disease and the hemostatic mechanism should be carefully evaluated. Any abnormality in hemostatic mechanism must be corrected before biopsy is attempted because excessive haemorrhage is one of the most serious complication following liver biopsy. Biopsy should be performed only if the clotting time is within the normal limit, and no severe anaemic changes in the blood. However if the clotting time can not be performed, liver biopsy can be performed safely under the cover of Vitamin K injection at the rate of 80-250mg i/m.

Liver biopsy instruments

A number of instruments are available for percutaneous liver biopsy. A Vimsilverman needle can remove satisfactory liver specimen. The simplest instrument has been the "Tru-cut" biopsy needle. (Trovenaol
It is a disposable needle used periodically in human breast biopsies, but can be chemically sterilized and reused again.

Preparation of the site

The skin over the site should be shaved and cleaned aseptically. A local infiltration of 2% lignocain hydrochloride will help to reduce the animal's reaction during insertion of needle. A small stab wound is made through the skin at the site of insertion with a 11 Bard-Parker blade or with a 15G hypodermic needle.

Technique of liver biopsy

In Ruminants Liver biopsy is performed at the right side of the animal. Various authors described different sites for liver biopsy. Out of which the site described by Pearson and Craig (1939) looks easy and simple. The puncture site for cattle can be located by extending a horizontal line cranially from the middle of the right paralumber fossa. The needle is inserted where this line crosses the 11th intercostal space on its right side. The needle is directed slightly cranially and ventrally. This technique is also used in obtaining liver tissue from the dairy goat.

If Tru-cut needle is used, the liver sample is removed by sliding cutting needle over the specimen notch of the biopsy needle. If Vimsliverman needle is used, first the outer canula needle with a plain stylet is introduced into the liver. Then after removing the plain stylet, the forked stylet is introduced into the needle and bored into the liver tissue. Finally the outer canula needle is introduced further to cover the end of the forked stylet and both are removed together with a fragment of tissue.

The sheep is held in a standing position by an assistant, but in contrast to a cattle a transthoracic technique is used. The right 8th intercostal space is entered at the level of the lower part of the paralumbar fossa. The needle is directed caudally and slightly medially between the lungs and the chest wall until the diaphragm is felt. It is then thrust caudally on through the diaphragm into the liver.

Histopathological examination

Tissue fragments collected by biopsy will be fixed in a 25 ml collection tube which is having 10 percent formalin immediately after collection. Samples will be processed in paraffin wax and sections will be stained with Haematoxylin and Eosin. Special stains can be used where appropriate. Proper histopathologic interpretation of the biopsy specimen is important for specific diagnosis.

REFERENCES


DIAGRAM SHOWING THE SITE FOR LIVER BIOPSY
Laboratories). It is a disposable needle used periodically in human breast biopsies, but can be chemically sterilized and reused again.

**Preparation of the site**

The skin over the site should be shaved and cleaned aseptically. A local infiltration of 2% lignocain hydrochloride will help to reduce the animal's reaction during insertion of needle. A small stab wound is made through the skin at the site of insertion with a 11 Bard -Parker blade or with a 15G hypodermic needle.

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REFERENCES


NEWER CONCEPTS IN RUMINANT THERAPY

Dr. S. AJITH KUMAR and Dr. V. GNANAPRAKASAM

Introduction

Drug passage through the ruminant gastrointestinal tract is much different than that of monogastric animals. The drug residence time in rumeno-reticulum, that greatly alters drug blood-level profiles as compared to those observed in monogastrics. A drug reaches the intestine at a rate primarily determined by the rate of rumen emptying. The drug is then absorbed and enters the general circulation or passes out with the faeces.

Although the rumen is lined with stratified squamous epithelium, the nonionized form of weak organic acids and bases passively diffuses through the mucosa and intraruminal drug administration provides low blood levels and greatly extended duration when compared to monogastric animals. This is caused by slow rate of rumen emptying. In ruminants long times (3-5 days) are needed to achieve steady-state blood levels during multiple oral dosing. But in some cases, extended duration of activity caused by slow-rumen emptying is advantageous for prolonging blood levels. This occurs when doses have been increased sufficiently to provide efficacious drug levels. This principle contributes greatly to the long-lasting properties of many sulfa drugs in ruminants.

Chronic oral dosage with a broad spectrum antimicrobial agent (such as Tetracycline or sulfonamide) can suppress activity of ruminal microflora, thereby interfering with carbohydrate digestion. The ruminal microflora also can inactivate drugs by hydrolytic or reductive reactions. Sick animals may demonstrate decreased rumen activity. This in turn will result in a greatly increased rumen mean residence time, which will result in even longer duration and lower peak blood levels. For example, lower will be peak sulfamethazine blood levels in diseased cattle compared to normal animals. Decreased rumen motility of a sick or starved animal may result in subtherapeutic blood levels, even while therapeutic levels are obtained with the same dose in normal animals. On the other hand, increasing the dose sufficiently to sick animals can result in therapeutic blood levels for a much longer duration than that observed in normal animals. In this case depressed rumen motility enhances therapy.

Drugs acting on the alimentary system

Carbachol and similar parasympathomimetic agents usually increase the amplitude of the reticular contraction but not the frequency, and this increase is followed by inhibition. Acetylcholine and histamine inhibit the activity of both rumen and reticulum. So use of antihistamines in frothy bloat will increase the motility and frothiness inside these compartments leading to aggravation of the condition. Atropine inhibits movement of the rumen and etorphine has an even greater inhibitory effect which should be borne in mind when this drugs are used in surgical procedures. Administration of adrenergic drugs will inhibit of the reticulo rumen.

In ruminants, an appreciable absorption of both drugs and products of digestion taken place in the reticulo rumen where as in simple stomached animals only the non-ionised moiety of the drug passes through the gastric mucosa. Drugs such as pilocarpine, atropine, phenothiazine, cyanide and metabolites like ammonia and fatty acids, are absorbed through the rumen wall.
Sodium sulphate is the saline purgative of choice in cattle. Magnesium oxide and carbonate are primarily antacids with slight cathartic properties. Irritant cathartics secreted in the milk of lactating animals can cause purgation in the nursing offspring. The bitter stomachic such as nux vomica has not been shown to be effective for stimulation of appetite. Vitamin B complex have been advocated to enhance appetite. Antacids have been employed in the treatment of ruminal acidosis following excessive consumption of carbohydrate. This practice may cause complications as alkalinization will increase the absorption of histamine and other basic substances from the gastrointestinal tract. Morphine is ineffective for the management of severe visceral pain in cattle but xylazine is very effective at a dose rate of 0.1-0.2 mg/kg body weight.

Antimicrobial therapy

Stability towards chemical and metabolic inactivation within the reticulo-rumen and abomasum of ruminant species determines the amount of an oral dose which is potentially available for absorption.

Erythromycin is well recognized as a valuable antibiotic for use against a variety of gram positive bacterial pathogens. Recent reports indicates that high proportions of *Pasteurella haemolytica* isolates are sensitive to erythromycin in vitro. Intramuscular route of administration can be recognised as the only acceptable route in cattle at a does rate of 15 mg/kg body weight. Apart from the adverse effects like diarrhoea, there is little reason to use erythromycin orally in ruminants because of negligible drug absorption. Use of erythromycin in preruminant calves require higher dosage due to the limitations in the absorption. Serious adverse gastrointestinal effects associated with the oral administration may occur with parenteral administration since a high proportion of Erythromycin apparently is eliminated by biliary secretion.

Tetracyclines will interfere with bacterial flora in the rumen, so they should not be given orally to ruminants.

Ampicillin anhydrate should not be administered intraabdominally. The use of intramuscular and intra abdominal sodium ampicillin is suitable during abdominal surgery in cows. Ampicillin will appear in the milk and urine for at least 48 hour post-administration.

Trimethoprim will be metabolised by the microflora in the gastro intestinal tract of ruminants within 48 minutes. So oral dosing with Trimethoprim in ruminants is not effective.

Chloramphenicol in ruminants will be inactivated by a reductive reaction in the rumen. Since the rumen takes 8-12 weeks to develop and become functional, the bioavailable profile of drugs administered orally to neonatal calves is similar to that obtained in monogastric species. So the chloramphenicol administered as oral solution is well absorbed in neonatal calves and oral dosage (26 mg/kg at 12 hour intervals) will maintain therapeutic effective plasma concentrations. The half life of chloramphenicol which is 14 hours in one day old calf, decrease to 4.2 hours in 10-12 weeks old calves. This decrease is caused partly by increasing drug "trapping" in the developing rumen and partly by development of metabolic capacity. Similar changes noticed in the case of trimethoprim therapy of goats.

However Furaltadone at a dose rate of 10 mg/kg body weight twice daily orally is found tobe effective for simple indigestion ruminal simple impaction, acid indigestion and enteritis. Long acting parenteral preparations are most convenient dosage form in farm animals. A single dose of (25,000 units/kg body weight) of procaine penicillin - G (in buffered aqueous suspension) injected either intramuscularly or subcutaneously can maintain effective levels of penicillin-G for 24 hours although the half life of penicillin-G is less than an hour. Oxytetracycline with base 2- Pyrolidone is approved for intramuscular administration to cattle in which single dose (20 mg/kg body weight) will maintain effective serum concentrations of oxytetracycline for 48 hours.
Passage of antimicrobial agents into milk

Only the lipid-soluble non-ionized fraction of an organic electrolyte that is free, unbound in the plasma diffuses into milk. In milk weak acids (Benzy1 penicillin, Ampicillin, Cephalosporin, sulfamethazine, and cloxacillin) produce ratios of milk ultrafiltrate concentration to plasma ultrafiltrate concentration that are less than or equal to unity and organic bases (Erythromycin, tylosin, Lincomycin, Trimethoprim) excluding aminoglycoside antibiotics (which are) polar attain concentration ratios greater than one.

Anthelmintics for Ruminants

Parasitic gastro-enteritis in cattle has been treated with drugs including the benzimidazoles, imidothiazoles, tetrahydropyrimidines and avermectins.

A number of different benzimidazoles (BZS) are used currently like thibendazole, cambendazole, oxibendazole, mebendazole, flubendadazole, fenbendazole, albendazole and oxfendazole. The anthelmintic activity of febantel and thiophanate and recently introduced netobimin is due principally to the formation of benzimidazoles by in vivo ruminal and hepatic metabolism and so are commonly referred to as probenzimidazoles. Benzimidazoles are least toxic of all anthelmintics. Cambendazole, parbendazole, oxfendazole, and albendazole are found to be teratogenic, which limits their use in early pregnant animals. Parbendazole and cambendazole are not recommended for use in lactating animals. Fenbendazole, oxfendazole and albendazole have an extended spectrum of activity and are highly effective against adult, immature and arrested larval stages of important nematodes and also having activity against cestodes and trematodes.

All of the benzimidazoles and pro-benzimidazoles are ovicidal. Oxfendazole is the active metabolite of albendazole, fenbendazole and febantel following their absorption from the gut, biotransformation in the liver and resorption into the alimentary tract. Resorption of benzimidazoles such as albendazole via the liver and bile is important in its action against adult Fasciola.

Among imidothiazoles, levamisole is used at half the dose rate of tetramisole and is therefore safer but therapeutic index of these compound is relatively low. These are not ovicidal. Levamisole is non-teratogenic and is safe for use in pregnant animals.

Pyrantel and its methyl analogue morantel are two members of Tetrahydroprimidines. The drugs are administered by the oral route. Morantel is more potent than pyrantel requiring a lower dose rate. It can be used in pregnant animals. But it will be less active against larval stages and are not ovicidal. Morantel sustained release bolus has been used in cattle recently and found to be effective.

Ivermectin is the only Avermectin derivative used. It has a very broad spectrum of activity against gastro-intestinal lung nematodes and some cestoparasites, but without effect on cestodes and trematodes. In cattle it is used as liquid for subcutaneous injection except Newzealand where a drench is available.

After parenteral treatment Ivermectin will be active for 14 days against certain immature nematodes. Usual dose of 0.2 mg/kg of the drug is even active against Thelazia rhodesinesis which infects the eye and Parafilaria boviola. In pigs a dose of 0.3 mg/kg is required by subcutaneous injection against oesophagostomum spp. and stephanurus dentatus. In cattle longer half life after subcutaneous injection than oral administration of ivermectin.
Netobimin (Totabin) is a new anthelmintic and it can be prepared as an ionic salt (e.g., trisamine) which shows good water solubility (650 mg/ml) so offers great flexibility for administration. 7.5-20 mg/kg of netobimin by oral administration is very effective against many nematodes, *Moniezia benedeni* and developing larva, and adults of *Dictyocaulus viviparus* in cattle and sheep. Netobimin at a dose rate of 20 mg/kg is effective against the small liver fluke, *Dicrocoelium dendriticum*. Closantel is a salicylanilide effective at 7.5 mg/kg in sheep and cattle against *Fasciola hepatica*, *F. gigantica* and blood sucking nematodes such as *Haemonchus contortus*. But its efficacy against 4 weeks old fluke is only moderate. only the most interesting aspects of closantel is that it is highly effective against *H. contortus*, resistant to benzimidazoles or levamisole and that it can produce a sustained effect because of its long half life. If has been shown to be highly active at 15 mg/kg by oral route and at 7.5 mg/kg by intramuscular injection in sheep against flukes.

Triclabendazole at dose rate of 5-10 mg/kg orally will control *F. hepatica* at all stages in sheep. Nitroxynil a fasciolicide is metabolised by rumen bacteria which destroys its activity and so restricts administration to injection only.

Clorsulon, a sulphonamide is another new fasciolicide highly active against adult *F. hepatica*. At a dose rate of 2-4 mg/kg it is also effective against 8 weeks old immature fluke in cattle.

Oxyclzanide is the only Fasciolicidal agent permitted for use in milk ruminants with out mandatory discarding of milk.

Good control of tape worms can be obtained with several benzimidazoles and probenzimidazoles including mebendazole, fenbendazole, albendazole and febantel. Specific drugs such as Niclosamide can also be used. Praziquantel is effective against adult and larval stages but its relatively high cost may limit its use.

Now new drug delivery systems have facilitated single dose cattle like, pour-on application and intra ruminal application of anthelmantics.

Pour-on application is extremely convenient and it had gained wide spread acceptance with the use of levamisole. In sheep useful activity after pouron application is difficult to achieve.

A device of direct administration into the rumen of cattle using a repeating syringe is used for oxfendazole. The reticulo-rumen provides an ideal site for the location of devices which can release anthelmintic over an extended period either continuously or in pulsatile manner. Levamisole continuous release bolus contains 22g levamisole. Both levamisole and morantel bolus systems used have a major disadvantage that the release rates decline with time and this may result in considerable selection pressure for resistance.

In ruminants, administration of compounds like oxfendazole and albendazole directly into the abomasum by way of oesophageal groove bypass (by oral drenching) increase the absorption and excretion. So minimum duration of exposure of the parasites to the drug (for albendazole 72 hours) will not be achieved and the drug will be less effective. Capsules containing micronized Albendazole if administered by balling gun orally to the rumen, can reduce the usual oral dose to one fourth. Pulse release boluses are used as intra ruminal boluses which will release a full dose of anthelmintic at appropriate time intervals. Most devices are designed to release at intervals equal or slightly longer than the pre-patent period of the most problematic parasites usually at 21-40 days intervals.
TREATMENT OF DIGESTIVE DISORDERS IN RUMINANTS

Dr. S.R. SRINIVASAN AND Dr. P. DHANAPALAN

Removal of the primary cause of the disease is essential but the major part of the treatment of digestive disorders is supportive and symptomatic.

There is no sense in forcibly inducing the peristalsis of the reticulum and rumen with potent menatorics. Motor agents like Parasympathomimetic, Strychnine, Tartar emetic and Veratrine are unnecessary.

The basic objectives in the treatment are:

1. To restore physiological conditions of rumen contents in both physical and biochemical respects.
2. To eliminate the inhibitory factors.

The general principles involved in the treatment of digestive disorders are:

In case of lack of fibrous structure, offer adequately structured forage. Give 2 kg hay/day/adult bovine to ensure normal functioning of forestomach mechanisms.

Ruminotomy - if severe matting of rumen contents

Very thin linseed mucus in large amounts through stomach tube, when ruminal ingesta are dried out

If Alkaline rumen contents, acetic, lactic, propionic and their salts are given. To restore microbiological digestion give 3 to 5 L of rumen fluid from healthy cattle. Also give sodium propionate or calcium propionate 50-100 g.

Plant stomachic like pulvis Genetian 10 to 20 G.

Antipyretics if fever is there.

Palatable, readily digestible forage, with minimum crude fiber requirements.

Stimulate appetite with molasses 200-300 G, Grain 1-2 kg, Sugar beets or turnips.

Provide mineral supplementation

Drench of sodium sulphate or magnesium sulphate 300-500 G as laxatives if there is stasis of lower gut.

Simple indigestion

a) Starvation for few days.

b) In overfeeding, purgative drench of Epsom salt 400-600 G

c) In severe cases, stimulants to stimulate the rumen motility - Neostigmine 0.5 mg/100 lb B.W. I/M

d) In acidic pH - Mag. carb (or) Mag. hydroxide - 225 - 450 G.

e) In alkaline pH - 5% Acetic acid - 2 ml/kg orally.
f) Mineral oil 4 litres or more to move undigested material and slow down the absorption of toxic materials.

g) In ruminal atony, rumen cud transplantation indicated for 3 days.

h) 500 G Glycerine twice during the first day to prevent development of Acetonemia.

Chronic latent rumen acidosis:

1. Ration must contain minimum structured feed-roumage.

2. Stabilise fermentation process in the rumen by addition of buffer substances into the concentrate - sodium bicarbonate 1% is added to the concentrate.

3. Treatment of cereals with sodium hydroxide.

4. To prevent liver abscesses, addition of antibiotics is practised.

Acute lactic acidosis:

Treatment of milder cases consists of change of feed to hay and straw as well as the administration of antacids (100-300 g sodium bicarbonate or Calcium bicarbonate; 250 to 450 G of Magnesium carbonate or Magnesium hydroxide in 3-12 litres of warm water).

1. Early stages -

Rumenotomy and removal of the rumen contents is the best and safest method.

oral administration of 10 - 20 G Chlorotetacycline with 50 liters water is given in several doses distributed over 24 hrs.

2. Advanced Cases:

Withdraw as much fluid as possible through a tube. Antibiotics or yeast in amounts upto 2 kg or the two combined are applied with ample water through nasogastric tube.

3. Cud transplantation from healthy animals.

4. Intravenous administration of physiological saline or electrolyte solution to correct dehydration. To the saline, sodium bicarbonate is added @ 13 G/litre.

5. 2-4 G thiamine dichloride (Vit.B1) should be administered; partly by intramuscular and partly by intravenous route

6. Administration of Anti-histaminics

7. Administration of Calcium gluconate and Magnesium gluconate on the 3rd and 4th days.


9. Administration of Thiabendazole at normal anthelmintic dose to control secondary mycotic ruminitis.

10. Inj. Calcium borogluconate 90 ml in 300 ml of 20% Dextrose.

11. Glucose inj. and glucocorticoid inj. are not indicated.
atent hydrochloric acidosis:

1. Elimination of primary cause
2. Withdraw the hyperacidic liquified rumen contents and stimulate the microbial- biochemical digestive processes.
3. Correct impaired electrolyte and fluid metabolism
4. Intravenous administration of physiological saline solution.

Alkalosis of the reticulo ruminal contents:

1. Oral application of 1-2 lit. 2.5% kitchen vinegar, 50-70 ml lactic acid diluted with 8-10 lit. water or 100-200 G granulated glutamic acid in 1 lit. water orally (repeated after 2 hrs).
2. Change in the diet - Increase the proportion of readily digestible carbohydrates in the form of molasses, turnips, grain, good quality hay with simultaneous reduction in the protein level.
3. With conditions of paresis or spasms, calcium magnesium solutions, liver protective substances and antihistamines should be injected.

Putrefaction of the reticulo ruminal contents:

1. Initial treatment similar to that of rumen alkalosis.
2. With a long history of rumen putrefaction, oral administration of 5-10 G streptomycin daily for 2 - 3 days or other antibiotic such as chlortetracycline.
3. In critical cases, rumenotomy.

Simple inactivity of the ruminal flora and fauna:

1. Change to feed rich in sugar, protein, starch as well as addition of mineral and trace elements.
2. Cud transplantation 2- 5 lit. of rumen fluid from healthy donor through stomach tube. This should be combined with administration of a mixture of sodium propionate or calcium propionate (50 - 100 G), antipyretics (acetanilid 20 G) and minerals.
3. To prevent overdistension of the rumen resulting from accumulation of gas, temporary rumen fistulation may have to be performed.

Ruminal tympany (bloat):

a) Free gas bloat treated by passing stomach tube.
b) In frothy bloat - Reduce the stability of the foam and then remove the gas by administering oils (Liquid paraffin or any vegetable oil) mixed with detergents.
c) Chemicals such as Polaxalene and Dioctyl Sodium sulfo succinate to reduce the viscosity of fluid ingesta and thereby reducing the stability.
d) Synthetic antifoaming agents - Pluronics L 62 (Dose 30 ml/drench).
e) Injection of defoaming agents in left flank.


**Ruminitis or ruminal parakeratosis:**

a) Inclusion of good quality roughage at 10% of dry matter in the ration.

b) In secondary ruminitis due to acute rumen engorgement, thiabendazole 25 mg/kg body weight orally can be given.

c) Feeding of antibiotics to reduce the incidence of liver abscesses.

**Traumatic reticulitis:**

a) Keeping the animal to stand on an inclined plane with the front feet 25 cm higher than back for 10 to 14 days. Immobilization facilitates the formation of adhesions which limits the spread of infection by preventing the foreign body moving forward and to fall back into reticulum.

b) Antibiotic/Sulphonamide.

c) Mild laxatives like liquid paraffin.

d) Newer methods - by use of magnets introduced into reticulum.

e) Surgical removal of foreign body

**Omasal ulcers:**

a) The inciting cause, if determined should be removed.

b) Indigestible material should be removed from the diet.

c) Oral antacids like sodium bicarbonate and magnesium hydroxide.

d) Repeated dosing of kapectate solution.

e) If haemorrhage occurs, blood transfusion can be advocated.

f) Balanced electrolyte solution to compensate the loss of body fluids.

g) Adrenal corticosteroids should be avoided.

h) Surgical correction.

i) Local gastric sedatives and astringents together with anticholinergic drugs.

j) Haematinics and injection of parenteral coagulants.

**Vagus indigestion:**

As a rule there are no prospects of healing of vagal lesion. If partial vagus paralysis, prognosis is somewhat better.

1) Treatment is aimed at removal of cause as far as possible, i.e.

a) Remove foreign body

b) Open and drain reticular abscess.

2) Create temporary rumen fistula by inserting a rubber tube and repeatedly infuse through it rumen flora from healthy animals.
3) Vitamin B and stimulating preparations parenterally, such as calcium gluconate and ACTH or glucocorticoids.

4) Conservative treatment with one gallon (4 lit). of mineral oil for adult cow orally.

5) Systemic administration of parasympathomimetic drugs may provide temporary or partial improvement in incomplete obstruction.

6) Emptying the abomasal contents by abomasotomy.

7) A balanced electrolyte solution intravenously to restore chloride deficit and for rehydration - 40 lit. in one or two days.

8) Exploratory laparotomy and rumenotomy to assess the underlying cause and attempting to remove it.

9) If no marked improvement in 2 to 4 weeks slaughter is recommended.

Left displaced abomasum:

a) Attempt to return the abomasum to its normal position.

b) Conservative approaches (1) to increase gastrointestinal motility 2) to increase tonicity of abomasum, and 3) to expel its gas and return to normal position. For that, calcium, neostigmine and saline cathartics can be used.

c) Mechanical repositioning of abomasum with or without concurrent drug therapy.
   
i) The cow is forced to walk on a steep inclined surface or taken for a rough truck ride.
   
ii) Rolling the cow.

d) Surgical correction.

Right displacement of abomasum and abomasal torsion:

1) Surgical correction

2) Fluid therapy intravenously to replace plasma and extracellular fluid deficit, to replace chloride ion and correct metabolic acidosis - Ringer’s solution or Isotonic sodium chloride. First 8 litres rapidly followed by 5 litres per hour.

3) Antibiotics and intravenous glucose to combat ketosis.

Abomasal impaction:

1) Treatment in early stages is effective - 1 pound of magnesium hydroxide or magnesium sulphate to evacuate the abomasum.

2) Faecal softeners like mineral oil and dioctyl sodium sulphosuccinate are effective in softening the mass and ingesta and administered directly into the abdomen by passing a stomach tube through reticulo-omasal orifice.

3) Physical removal of abomasal contents by abomasotomy, but is unrewarding.

4) Supportive therapy by intravenous or oral fluids for rehydration.

5) Intravenous glucose and aminoacids to provide nutritional support.
Caecal torsion

1) Immediate surgery to remove the caecal content.
2) Supportive therapy with antibiotics and fluid therapy using balanced electrolyte solution like Ringer's acetate depending upon the state of hydration and degree of shock.

Diarrhoea:

a) Fluid and electrolyte replacement:
   i) Oral electrolyte containing glucose and sodium.
   ii) Intravenous fluids rich in bicarbonate to correct metabolic acidosis.

b) Non specific diarrhoeal therapy like opiate derivatives to increase segmental contraction and to retard the flow of ingesta.

c) Adsorbants like kaolin and pectin to adsorb toxins.

d) Broad spectrum antibiotics such as Chloramphenicol, Gentamicin or Nitrofurazone, if organism is susceptible.

Malabsorption syndrome

1. Therapy depends on cause
2. Removal of source from the diet
3. Neoplasms, right heart failure and Johne's disease respond poorly to therapy.
4. Until definite cause can be found, supportive therapy with improved nutritional intake with high quality protein diet like alfalfa hay with soyabean meal based concentrate that is rich in vitamins (Brewer's yeast) would be appropriate.

Fatty liver syndrome:

1. To provide energy balance, dexamethazone 10-20 mg i/m per 454 kg daily for 3-5 days. It produces mild hyperglycaemia and decrease milk production so as to reduce energy demands.
2. 200 G of glucose intravenously twice daily or 5% dextrose saline intravenously.
3. 2-4 p of rumen liquor twice daily to promote rumen protozoal activity.
4. In advanced cases, forced feeding of electrolytes and alfalfa meal with propylene glycol.
PRACTICAL ANTIMICROBIAL THERAPY IN RUMINANT PRACTICE

Dr. M. G. Jayathangaraj

The success of antimicrobial therapy in ruminant practice depends on the following factors:

(1) Whether the drug selected is economical and easily available.

(2) Whether the drug can act well when administered orally in cattle.

(3) Half-life of the drug, pH of the medium, pKa value of the drug.

(4) Whether suitable for the particular clinical condition encountered.

(5) Pharmacokinetics of the drug-in general.

The aim of antimicrobial therapy is to provide a sufficient concentration of a drug at the site of action and to maintain this concentration for a certain period of time.

Antibiotics are the complex organic chemicals synthesised by the microorganisms and the choice of a suitable antimicrobial agent is very difficult and the factors like availability and cost of antibiotic, effect of the agent on the organ and system infected, possibility of any potential toxic side effects, adverse reactions etc; are to be considered always.

Fever enhances the entry of penicillin into tissues and there is marked increase in penetrating capacity of drug into cerebrospinal fluid barriers which occurs during the acute phase of the bacterial meningitis. Fever inhibits gastric emptying in monogastric species and causes reticuloruminal stasis. In ruminant, pH of the medium in which the drug acts, pH of the drug, pKa value of the drug, lipid solubility of the drug, serum protein binding properties of the drug, dosage of the drug administered are the significant factors which tell upon the therapeutic response of the drug treatment. Clinicians should take more caution in treating pregnant animals because of plenty of factors involved in placental transfer and fetal uptake of drug. (Likewise entry of a drug into CSF largely depends on factors like lipid solubility and capacity to pass through the endothelial junctions of the brain.)

The antimicrobials can be classified as bactericidal and bacteriostatic

Bactericidal: penicillin, Macrolides, Nitrofurans of high concentration, Cephalosporins, polymixins, aminoglycosides.

Bacteriostatics: Nitrofurans of low dose, Sulphonamides, tetracyclines, trimethoprim, clindamycin, Lincomycin, Tiamulin, Chloramphenicol, certain macrolides like tylosin, oleandomycin, Spiramicin, and Novobiocin.

Tetracyclines:

Hansen et al. (1981a) concluded that intra-venous injection of tetracycline is hazardous, but collapse can be avoided by giving the injection very slowly over a period of not less than 5 minutes.

Though it is a broad spectrum antibiotic, it has comparatively more potent action against Gm + ve bacteria and are bacteriostatic than bactericidal. These diffuse more readily into CSF than chlor or oxytetracyline and in general, teta-cyclines diffuse with difficulty into CSF. These can pass through bovine placenta and thereby enter fetal circulation also. This group is able to pass into the microcosm of the prostatic...
gland. However, presence of calcium ions, or Manganese, Aluminium, Magnesium, Iron and bismuth ions limit the absorption of oral tetracyclines. Tetracyclines should not be given by oral route in ruminants. Newer tetracyclines have greater lipid solubility.

Sulphonamides:

Bushby (1980) stated that trimethoprim and sulphonamides are bactericidal but only when their inhibition of protein synthesis is circumvented by the presence of methionine, glycine and purine.

Because of their action on bacteria, though these are broadspectrum agents, they are classified into antibiotics group itself. They cross physiological barriers like CSF, fetus, milk, serous cavity etc; and are less active in the presence of pus, necrotic tissues etc; kidney is the main organ of excretion. These mainly act by affecting the folic acid synthesis which is required for the synthesis of bacterial cells. Among them, sulfaguanidine, succinyl sulfathiazole and phthalyl sulfathiazole are enteric sulfas. Sulfathiazole is not recommended for I/V use in domestic animals because of rapid elimination from plasma. Sulfadiazine penetrates well into CSF than sulfamethoxazole, sulfamethoxy pyridazine and acetyl sulfa methoxy pyridazine are currently available for use in dogs and cats. Sulpha ethoxy pyridazine has longer plasma half life. These are bacteriostatic in action but trimethopim potentiated sulphonamides exert bactericidal action. Dose rates have to be reduced in neonatal animals. Sulfa-methazine is the preferred drug in cattle because of their rapid absorption and prologed sojourn.

Penicillins and cephalosporins

Penicillins and cephalosporins are beta lactam antibiotics and other naturally occurring beta lactam compounds include aminopenicillanic acid, clavulanic acid, cephemycins and thenamycin (Powers et al, 1980)

Action of penicillins and cephalosporins is by means of inhibition of formation of bacterial cell wall. Susceptibility to bacterial beta lactamase enzymes and limited spectrum limit the usefulness of penicillin-G. Inactivation by gastric acids and not possessing of high antibiotic action like penicillin-G limits the utility of Methicillin. Lack of oral absorption lead to the development of hetacillin, pivampicillin, becampicillin and talampicillin.

Relative availability of amoxycillin and pivampicillin was 6.4 times greater than that of ampicillin trihydrate. Addition of clavulanic acid prevents the beta lactamase or penicillinase from destroying the bactericidal activity of amoxycillin.

Clinical use of cephalosporins has been more limited than usage of penicillins in veterinary medicine. Cefamandole, cefuroxine and cefataxime are the cephalosporins which are more stable in the presence of beta lactamases and thereby have a broader spectrum. They penetrate well into pleural, pericardial and synovial fluid and into most tissue spaces. A unique feature of several of more recent cephalosporins is that they are able to penetrate into CSF in amounts adequate to successful treatment of certain bacterial infections of CSF.

Aminoglycoside antibiotics, macrolides, lincomycin and spectinomycin

These are poorly absorbed from GI tract and do not diffuse well into CSF. They are rapidly excreted by normal kidney. All members have small therapeutic index, which make the clinician in dilemma. The dose of gentamicin is 2-3 mg/kg for 8 hrs in patients with normal renal function. Parenterally given kanamicin achieves good concentration in synovial fluid and streptomycin can well diffuse into the fetal blood and is the drug of choice in leptospirosis. Dihydrostreptomycin penetrates the acute abscesses well. Among all, streptomycin and dihydro-streptomycin have increased frequency of resistance. Neomycin persists for a longer time in edible tissues.
Base preparations of erythromycin for oral use are the coated tablets to prevent acid degradation in the stomach. Absorption of all forms of these basic drugs occur in the more favourable pH of small intestine. Oral absorption of spectinomycin is poor. Although macrolides and lincomycin are used as second line antibiotics against Gm+ve bacteria, they are of special value in pneumonia and mastitis because of their propensity to achieve high tissue concentration. Spectinomycin is of high value due to its low toxicity and usefulness against m-ve bacteria.

The dose and duration of aminoglycoside are the important risk factors in the development of nephrotoxicosis and gentamicin induced nephrotoxicosis has been recorded by Hinchaliff et al (1988).

Nitrofuran derivatives:

These are the synthetic compounds, acting by inhibition of the enzymatic oxidative process. Presence of pus, plasma and milk all reduces the antibacterial activity of nitrofurans.

Nitrofurazolidone is recommended in the treatment of the digestive tract infection in calves and is given at a rate of 10 mg per kg, orally BID, daily. Nitrofurans are commonly recognised as toxic agents to cattle and even in therapeutic doses, they induce muscular tremors, peripheral neuritis, seizures, and various gastrointestinal upsets. (Hausen et al, 1981a).

REFERENCES:


Mastitis is emerging as the most important disease affecting the bovine herd in the economic point of view. Treatments during clinical episodes and during lactation are less effective than treatments during subclinical stages or during the dry period. An early diagnosis and initiation of the proper treatment is absolutely essential in treating clinical mastitis.

Antibiotic therapy in mastitis.

Antibiotic therapy in clinical mastitis should be accomplished while minimizing the risk of contaminating the milk with antibiotic residues.

Before treatment of a cow with clinical mastitis is initiated, the mastitis should be classified as either mild or severe. Mild mastitis is characterized by abnormal appearing milk and slight swelling of the udder and severe mastitis is characterized by abnormal milk, severe swelling of the udder, agalactia in the affected quarter and systemic illness.

Next, aseptic milk samples should be collected from the clinically affected quarters to identify the causative pathogens.

Intramammary treatment of mastitis:

In clinical mastitis, the intramammary route for the administration of antimicrobial drugs is practical and convenient in animals without any occlusion of the teat canal or cistern.

It is also a suitable route for administration of long acting antimicrobial preparations at drying off as a part of mastitis control programmes.

Intramammary therapy can be divided into lactational therapy and dry cow therapy.

An antimicrobial intramammary drug should not be selected simply because it has broadest spectrum of activity. On a molecular basis penicillin G is probably the most effective antibiotic against penicillin-sensitive organisms. Penethemate is a basic ester of penicillin G which has enhanced physicochemical characteristics and therefore similar distribution properties and activity to penicillin G.

Combination of penicillin and aminoglycoside (e.g. dihydrostreptomycin, framycetin, neomycin and streptomycin) have been shown to be effective, where sensitivity tests are not available and the aetiological agent is not identified.

The aminopenicillins, cephalosporins (especially third generation) and tetracyclines are all highly effective against coliforms but are likely to have less activity against penicillin susceptible gram-positive organisms than penicillin G.

Many Staphylococcus aureus, responsible for clinical mastitis produce an enzyme, beta-lactamase which confers upon them resistance to the unprotected beta-lactum antibiotics (penicillin G, penethemate, ampicillin, amoxycillin). Cloxacillin and nafcillin are however highly effective against such bacteria.
Dry cow therapy designed to remove infections present in the udder and to present new infections establishing during the dry period. Syringes containing cloxacillin, nafcillin or cephalosporins may be indicated. These preparations usually provide antibiotic coverage for 3 weeks.

Inclusion of corticosteroid in the intramammary products is designed to reduce inflammation and restore the normal function of the udder. In combination with antibiotics, prednisolone (10mg) has been shown to speed up the resolution of the udder swelling.

When selecting an intramammary antibiotic, other considerations, such as the recommended period of treatment, number of administrations and milk withdrawal time should be considered.

In lactating cows it has been recommended that drugs which are well absorbed and distributed in the mammary gland should be given six times at 12 hours intervals and that poorly absorbed drugs should be given three times at 24 hours intervals. Some products, especially those containing cephalosporins require single administration only.

Systemic antibiotic therapy, preferably by intravenous route, frequent milking out, oxytocin injections and use of anti-inflammatory drugs can be adopted in severe cases of mastitis. In such cases intramammary antibiotics can be given after the last milking of the day.

Reasons for failure of antibiotic therapy in clinical mastitis:

1. The drug is unable to reach the effective concentration in all the sites of infection because of the following:
   a. Microabscess formation (S. aureus infections) in the mammary gland.
   b. Lactiferous duct blockage (milk clots, inflammation, exudate, oedema or intrinsic properties of the drug).
   c. Fibrosis of the mammary gland.
   d. Intracellular bacteria (S. aureus infections) cannot be reached.
   e. The drug is absorbed from the milk to the systemic circulation.
   f. The drug is milked out in subsequent milking.
   g. Treatments are not repeated in a timely fashion to maintain the minimum inhibitory concentration in tissue necessary to kill bacteria.

2. The bacteria were already killed by the Cow’s immune system before therapy was initiated.

3. The bacteria are refractory to the effect of the drug.
   a. The bacteria are not in the rapid growth phase required for the drug to be effective.
   b. The organism is resistant to appropriate antibiotics (e.g. Pseudomonas, Mycoplasma, Yeasts) or a drug with an incorrect bacterial spectrum of activity is selected.
   c. The organism has acquired antibiotic resistance.
   d. There is an emergence of L forms without cell walls, that resist Beta-lactum antibiotics

4. The affected quarter is reinfected as a result of the following:
   a. Inadequate duration of therapy
b. Poor hygiene

c. Poor udder shape

d. Contaminated Cannulas.

e. Milking machine malfunction

f. Damaged teat end.

g. Compromised immune system.

Treatment of mastitis caused by specific organisms

1. *Streptococcus agalactiae* mastitis:

   This is the only common pathogen which is highly sensitive to approved intramammary antibiotics.

2. *Staphylococcus aureus* mastitis:

   This type of mastitis having very poor response to antibiotic therapy. In vitro demonstration of sensitivity to staphylococcus to an antibiotic is no guarantee of therapeutic success. The cure rate is low because the organism forms microabscesses in the peripheral tissue, where intracisternal drugs cannot penetrate. The ability of the bacteria to survive inside epithelial cells, polymorphonucleocytes and macrophages may additionally contribute to their therapy resistance.

   Staphylococci causing mastitis are often resistant to penicillin. For this reason drugs not destroyed by penicillinase, such as cloxacillin, novobiocin, tetracyclines, cephalosporins and erythromycin are often used. Attempts at curing staphylococcal infection are usually best made during the dry period.

   In gangrenous mastitis associated with staphylococcal infection, the affected quarters should be milked as completely as possible and treated with antibiotics. Early parenteral treatment with large doses of a sulfonamide, penicillin, penicillin-streptomycin, tylosine or erythromycin may help to save the quarter or life of the cow. Antihistamines should be given to combat toxemia and parenteral administration of electrolytes is recommended. If the teat becomes gangrenous it should be amputated; multiple incisions of the mammary gland are not recommended. Affected cows should be isolated from the rest of the herd.

3. Mastitis caused by environmental *streptococci*:

   Environmental streptococci and coliform bacteria accounts for the majority of environmental clinical mastitis cases when an aetiological diagnosis is reached. Among systemic antibiotics only erythromycin distributes well to the mammary gland and demonstrates reproducible activity against *S. uberis* and *S. dysgalactiae*.

4. Coliform mastitis:

   Coliform organisms can cause mastitis ranging in severity from subclinical to peracute. Most of the clinical signs of coliform mastitis are thought to be due to the effects of endotoxin. Therapy for coliform mastitis should therefore focus primarily on removing endotoxin from the udder by using frequent and complete milkout, often after injection of 10 units of oxytocin intravenously. Toxic cows badly needs 10-20 litres of electrolyte intravenously, non steroidal antiinflammatory drugs (eg. flunixin meglumine), corticosteroids (10-20 mg of...
and recommended dosages of antihistamine. Some such cows may become hypocalcaemic and may need calcium replacement apart from the antibiotic therapy.

5 Summer mastitis:

Summer mastitis usually refers to severe clinical cases of suppurative mastitis occurring in heifers and dry cows in summer. *Corynebacterium pyogenes* predominates and the severity of infection is related to the presence of anaerobes. Foul odour of organic acids and indole in the secretion, produced by *Peptococcus indolius*, the common anaerobe, is the most reliable diagnostic feature.

Successful bacteriological cure of summer mastitis is rare in the dry period. Therapy is most important in keeping the animal clinically healthy.

The best therapy is to treat the animal with antibiotics intramuscularly, strip the quarter frequently and apply intramammary antibiotics in the evening. The pathogens are susceptible to a wide range of antibiotics including penicillin/Streptomycin preparations. A greater success has been claimed for erythromycin, which diffuses more readily when given as intramammary infusion.

Success has also been claimed for anti-anaerobic preparations containing metronidazole. Chlortetracycline can be useful in lactating animal but should not be used in the dry gland as all secretory function will be lost.

Mastitis caused by pathogens refractory to antibiotics:

A fairly large group of pathogens are refractory to all antibiotic treatment. Cows with these infections must recover on their own or be culled from the herd. This group includes Mycoplasma, Pseudomonas, Pasteurella, Serratia, Prototheca, Mycobacterium, Nocardia, Bacillus, Yeasts and fungi and *Actinomyces pyogenes*.

Mastitis with a negative bacterial culture:

Antibiotic therapy in these cows is difficult to justify, however it is equally difficult to identify these cows until after treatment is initiated. The treatment goal must be to reduce udder inflammation and return the milk to normal, and to control the infection. Anti inflammatory drugs may be indicated in place of antibiotic therapy.
FLUID THERAPY

Water constitutes approximately two thirds of body weight. Exact proportion of the water content vary with the fat content of the body. Two third of this water is in the intracellular fluid (ICF) and the rest in the extracellular fluid (ECF). Twenty five percent of the extracellular fluid (i.e.) 5% of the body weight is made up of plasma water and rest 75% (16% body weight) is interstitial fluid. The distribution of water within the body is governed by the hydrostatic and osmotic pressure.

Disorders of water and electrolyte metabolism:

1. Primary water depletion

This occurs when the intake is insufficient and inevitable losses continue. Intake may be curtailed by

   (1) Diseases affecting swallowing (Tetanus, esophageal obstruction)

   (2) Excess renal losses (Diabetes mellitus, failure of ADH). ECF is first affected and becomes hypertonic. Water is drawn into the ECF from ICF thus the loss is evenly distributed. Tissue catabolism liberates even more intra cellular water so that ICF contracts even further. Initially electrolytes are excreted to maintain ECF tonicity but later if the losses continue, to maintain ECF volume oliguria results, with high urine specific gravity and haemoconcentration. Thirst is marked and mucus membranes are dry. No significant hypotension or tachycardia unless the deficit is massive. Death occurs when approximately 40% of the water is lost due to medullary failure.

   Treatment: N/5 Saline (0.18%) and 4.3% Dextrose.

2. Mixed water and electrolyte depletion

   This occurs when the body secretions are lost in vomitus or diarrhoea as exudates. Water and electrolytes are lost but if the animal continues to drink, the plasma sodium level will tend to fall so that ECF becomes hypotonic. This provokes diuresis and also causes water move into the ICF in an attempt to maintain ECF osmolality. This results in an early decrease in ECF and sign of circulatory shock develops fairly early. After the initial diuresis hypovolemia affects renal function so that oliguria occurs. Thirst is often not marked, mucus membranes remain moist but the saliva is often viscous.

   Treatment : Isotonic (or) even hypertonic solutions are used.

3. Acid base disturbance:

   Hydrogen ions are generally produced by the oxidation of food stuff and tissues and immediately buffered by the buffering systems. Conventionally the hydrogen ions are measured as pH. Deviation of blood pH below 6.8 and above 7.8 are generally incompatible with life. The acid base disturbances may be due to metabolic (or) respiratory causes.
Diagnosis of disorders

An accurate history can give valuable information about the size and type of deficit present.

(eg) Mixed depletion - in vomition and diarrhoea

Comatose animals - primary waterloss.

It is very important to ask about the presence of clinical signs like diarrhoea, vomition, polyurea, noexia, panting and pyrexia. Clinical signs express the percent dehydration.

Loss of skin turgor (elasticity) - 5%
Loss of skin turgor + sunken eye balls - 5-7%
Loss of skin turgor + sunken eye balls + slow capillary refill time - 7-10%
Hypovolemia and shock - 12-15%

Dry mucus membrane can also indicate dehydration but panting will also cause dryness.

Changes in body weight can be a useful guide to the extent of loss if the normal weight is known.

Capillary refill time is another rough indication of the adequacy of the perfusion. A refill time over 2 seconds indicates hypotension, hypovolemia (or) peripheral vasoconstriction. This together with cool extremities and a rapid weak pulse indicates oligaeic shock, which is due to dehydration suggests a very large body fluid deficit.

PCV, Hb and Total plasma protein increases in dehydration. The PCV may be used to calculate fluid requirements. Approximately 10 ml/kg should be infused for each 1% raise in PCV over 45% (In anemia 5 to 10% should be added to the measured PCV)

How much fluid to use?

<table>
<thead>
<tr>
<th>Body wt. loss %</th>
<th>PCV</th>
<th>Fluid requirement (ml/kg bwt)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4-6</td>
<td>40-45</td>
<td>20-25</td>
</tr>
<tr>
<td>6-8</td>
<td>50</td>
<td>30-50</td>
</tr>
<tr>
<td>8-10</td>
<td>55</td>
<td>50-80</td>
</tr>
<tr>
<td>10-12</td>
<td>60</td>
<td>80-120</td>
</tr>
</tbody>
</table>

Plasma electrolytes: Measuring the levels of electrolyte will be very useful.

Urinalysis: pH of urine and specific gravity are useful.

Osmolality: This is useful to find out hypotonic (or) hypertonic dehydration. Serum osmolality is measured using the following formula:

Serum osmolality m osm/kg = 2 (serum Na + K) + blood glucose + BUN in milliosmols.

Normal serum osmolality is 280-310 m osm.

Central venous pressure: Normal CVP is 0 to 6 or 7 cm of H2O. Serial measurements should be made. Low CVP indicator is relative (or) absolute hypovolemia. Increase in CVP occurs in fluid overload (or) heart failure.
Urinary output: This is an indirect measurement of CVP. Normal urinary output is 1 to 1.5 ml/kg/hr. Less than 0.5 ml per kg body weight per hour is considered as oliguria.

Anion gap: Anion gap \( a = (Na + K) - (Cl + HCO3) \)

This is extensively used in man. Interpretation of anion gap is difficult if bicarbonate precursors are used. This is not useful during lactacidosis.

Acid-Base evaluation is done using acid base analysis. The results are derived by using nomograms. Most commonly used nomogram is siggaard-Anderson nomogram. Base deficit is calculated by using the nomogram. The derived figure is then used in the formula as follows:

Body weight x Base deficit x factor (0.4) = Total body base deficit

Methods of estimating the size of volume deficit:

1) Factorial approach based on net deficits resulting from

(a) abnormal losses (b) failure of intake to match normal losses

(eg) A 20 kg. female dog inappetent for 3 days, vomiting profusely once daily since the onset of the illness and urination decreased in frequency for last two days.

Calculation:

1.25 ml/kg/3 days

Inevitable water loss: \( (3 \times 20 \times 25) = 1500 \) ml

25 ml/kg/one day

Urinary water loss: \( (1 \times 20 \times 25) = 500 \) ml

4 ml/kg vomit losses: \( (4 \times 20 \times 1 \times 3) = 240 \) ml

Total deficit: 2240 ml

ECF deficit (33%) = 750 ml

plasma deficit (25% of ECF deficit) - 190 ml

The dog therefore needs approximately 200 ml rapidly to support its circulation and a further 2 litre given slowly to replenish the rest of the deficit.

2) Clinical approach:

Amount of fluid required is calculated by % of dehydration, Body weight x 10 = ml

e.g. 500 kg cow which is 10% dehydrated will require

\( 500 \times 10 \times 10 = 50000 \) ml or 50 litres

Treatment

Includes

1) Correction of the existing deficit
2) Meeting the daily requirements
3) Replacing the ongoing abnormal losses.
Correction of the existing deficit:

1) Restoration of the circulating blood volume

Plasma or plasma substitutes are preferable. The flow rate can be very fast with crystalloids in animals with no preexisting renal or cardiovascular disease. A rate of 90 ml/hr can be safely tolerated for the first 30 minutes to 1 hour. If rough estimates cannot be calculated, approximately 5-10 ml/kg colloid solution can be given quickly over 20-30 minutes in order to improve the condition and then the infusion may be changed to a crystalloid solution infused at a slower rate.

2) Replenishing the rest of the deficit:

This can be done very slowly thus making up the plasma deficit in 24-48 hrs. Fifty percent of the fluid is given in the first 6 hrs at the rate of 5-10 ml/kg/hr.

Meeting normal daily requirement

Normal daily needs vary between 40-50 ml/kg but in most species they tend to increase in illness and up to twice this need is required depending on the urine output. This should be supplied daily until the animal is able and willing to drink voluntarily again. Maintenance solutions are hypotonic.

e.g. N/5 saline with 4.3% dextrose.

Replacing ongoing abnormal losses

The volume of the abnormal losses can be estimated by the clinician and this amount must be added to the normal requirement. It may not be possible to measure losses in pleural and peritoneal effusions but often in vomiting and diarrhoeas, only a gross estimate can be made. Vomiting losses can be conservatively estimated at 1 ml/kg/vomit, while diarrhoea accounts for losses up to 200 ml/kg/day.

Rate of infusion

The fluid replacement may be as fast as possible and several veins can be cannulated simultaneously.

- Moderate deficit-Infusion rate may be slower
- Hypovolemic shock-3 ml/kg/minute for first 20 minutes
- Very severe cases- 10-15 ml/kg/hr
- Normal maintenance requirements-5 ml/kg/hr

The flow rate can be converted to drops per minute by using the following formula

\[
\text{Drops/minute} = \frac{\text{drops/ml} \times \text{ml/kg body wt} \times \text{Body wt in kg}}{60}
\]

When to stop?

1) On hearing moist rales, moist cough and serious nasal discharge indicating pulmonary edema,

2) Increase in CVP

3) Urine output.
Choice of fluids

1) ECF REPLACEMENT: This solution must restore the ECF volume. e.g. Ringer's lactate, Normal saline.

2) PLASMA SUPPORT: Plasma volume expanders.

3) ECF ALKALINSER; Bicarbonate or its precursor (lactate or acetate) as in Ringer's lactate or acetate.

4) ECF ACIDIFIER; Normal saline.

5) ECF DILUENT; 5% Dextrose solution.

6) MAINTENANCE SOLUTIONS; These are intended to substitute for normal drinking and for major electrolytes in food. These solutions should not be given subcutaneously.

7) NUTRIENT SOLUTIONS; glucose, alcohol or lipid emulsions and protein hydrolysates

8) CONCENTRATED DERIVATIVES; Usually solutions of a single salt. e.g. sodium bicarbonate, pot.chloride, Hypertonic saline.

Routes of administration

1) Alimentary, oral and rectal.

2) Subcutaneous.

3) Intraperitoneal.

4) Intravenous.

REFERENCES


CARE AND MANAGEMENT OF DOWNER COW/RECUMBENT CATTLE

Dr. B. NAGARAJAN

Downer Cow Syndrome is a common problem faced by every field veterinarian. It is a challenging, frustrating and agonizing for the veterinarian to diagnose the case and to give treatment and prognosis. A Downer cow is one, down for being at least 24 hours without apparent reason for being down (i.e.) no apparent reason prior to development of the sequellae of recumbency. It is often diagnosed when a definite diagnosis cannot be made. When a specific diagnosis is made, the term downer cow syndrome is not used. By itself it is not a disease, but it is a complication.

Downer cow will have the following features: The cow has been down longer than 24 hours.
- is not suffering from hypocalcemia. (i.e) The animal which is clinically parturient paresis but unable to rise after two calcium intravenous injections.
- There is no diagnosable cause
- Animal is in sternal recumbency
- It is usually related to the calving period.

When a cow become alert and gains control of forequarters following calcium therapy but remains recumbent due to inability to use hind quarters, it is called as "Creeper Cow". It may occur due to hypokalemia.

Risk factors for downer cow:
1. Peak lactation years of high producers.
2. Complications arise due to delayed or incomplete treatment of various diseases after parturition.
3. Poor housing conditions
4. Excess body fat
5. Septic conditions
6. Malnutrition

Reasons for downer cow:
There are many possible causes which result in a downer cow. However in most of the cases secondary damage is responsible for the downer cow problem, pressure damage resulting in obstruction of the blood supply causing ischemic necrosis with muscle and nerve damage. This secondary damage can result in permanent recumbency even when the primary cause has been successfully treated.

Primary causes of recumbency around parturition can involve metabolic disorders, toxaemia, injury during or following calving and improper management.
Metabolic disorders:

- Hypocalcaemia
- Hypomagnesaemia
- Hypokalaemia
- Acetonaemia

Toxaemia:

- Peracute / acute mastitis
- Acute Metritis
- Acute diffuse peritonitis
- Rupture of reticulum, abomasum, uterus
- Aspiration pneumonia
- Traumatic reticulitis / pericarditis.

Injuries during parturition:

- Ruptured uterus
- Internal haemorrhage
- Obturator Paralysis
- Fractured pelvis
- Searal displacement
- Sciatic nerve paralysis
- Exhaustion.

Injuries following parturition:

- Fractured Pelvis
- Fractured femur
- Rupture of the round ligament
- Dislocation of hip
- Rupture of adductor muscles
- Rupture of gastrocnemius muscle, tendon
- Damage to peripheral nerves (eg) tibial
- Pressure syndrome following parturient paresis.
Management:

Malnutrition
Overfat
Slippery floors
Delayed calcium therapy
Veterinary treatment (e.g.) Epidural anaesthesia.

Pathogenesis:

Primary factors
Sternal recumbency
Compression of soft tissues (Secondary factor)

- Contraction of functional muscles
- Muscle damage and haemorrhage
- Mechanical venous constriction in upper half of the hindlimbs
- Venous congestion and thrombosis
- Oedema of tissues
- Ischemic necrosis

Examination of recumbent cattle:

This includes:
- History
- Observation
- General examination
- Special examination
- Laboratory examination

1. History: In history the following questions should be asked to owner

- Was the calving difficult?
- Had it got up since calving?
- Did it have milk fever?
- Had treatments been given by the farmer?
- When the recumbency started?
- Before / during / soon after calving?
How long had it been recumbent?
Had position changed since recumbent?
How long had it been in current position?
Whether the floor is slippery?

Other particulars like proceeding symptom, early disease, frequency of paresis and mineral feeding 6-8 wks prior to calving also should be checked.

2. Observation of recumbent cattle:

Observe for:
General appearance
Position of the head, neck limbs, and tail in relation to body.
Angles of limb joints
Swelling / injuries
Unusual movement
State of feet
Surroundings.

3. General examination:

Examine for:
General state of health,
Mental status
respiration
Pulse
Temperature
Mucous membrane
Udder / reproductive and circulation system and CNS
Rectal examination, Vaginal examination.

Clinical signs:
Clinically downer cow and creeper cow can be differentiated in the following manner:

<table>
<thead>
<tr>
<th>Downer</th>
<th>Creeper</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mental status</td>
<td>Alert</td>
</tr>
<tr>
<td>When stranger comes</td>
<td>Tries to get up</td>
</tr>
<tr>
<td>Fetlock while raising</td>
<td>Pronounced fluxure of</td>
</tr>
</tbody>
</table>
4. Special examination:

In a recumbent cattle special attention should be given towards examination of:

a) Locomotor system
b) foot
c) Nervous system

a) Locomotor system:

— Observation of lying posture and any deviation from normal is noted.
— Stimulate to get up and observe the voluntary movement and muscle tone.
— Evidence of traumatic injuries like detached horn, haematoma, decubital sores, swelling of adductor, ischial, gastrocnemius muscles should be checked.
Palpation:

- Any muscle suspected of having been ruptured should be palpated especially gluteal and thigh muscles.
- Passive movement of hind limb is important. Roll on right and left side.
- Leg - flex, extend, abduct, adduct it and rotate it in circular movement to test hip joint mobility.
- During this period observe for pain and rectal examination is done.
- To check the movement of pelvic bones.
- To detect abnormal passive movement of parts of the limb or pelvis, palpable / audible cripitation.
- To check periproctal / perivaginal tissue swelling hard induration.
- Tail, anus, rectum and bladder are checked for normal muscle tone of emptying function.
- If sacral nerve is damaged:
  Muscle will be slack
  Rectum will have full of faeces
  Bladder will get distended with urine.
- Pressure is put on either end alternatively to test for fracture or detachment of sacroileal ligament.
  These conditions are characterised by abnormal mobility or with cripitation.

Reflexes:

Spinal reflex:

Do not attempt to test on the leg upon which the animal is lying.

Patellar reflex:

- Partly flex and support the limb. Then tap the middle of patellar ligament.
- Observe the reflex contraction of quadriceps muscle resulting the extension of stifle joint.

Flexor reflex:

- Stimulate the skin by artery forcep and observe the flexion of limb.

b) Examination of Foot:

Scrap and clean the foot before examination.

Dorsal aspect:

- Observe for swelling, fissures and grooves
- Flexion or extension, abduction or adduction may indicate the desire to relieve discomfort in particular areas of foot.
- Palpation and percussion should be done on the coronet to assess the pain response.

Plantar aspect:

The cleft of the foot is examined visually and by digital explanation. Sole, heel, bulb of heel and axial groove are examined by manual or mechanical pressure by hoof testers.

c) Nervous system:

a) Behaviour
b) Head posture
c) Examination of the eye
— Position of upper eye lid
— Observe the size symmetry of pupillary aperture -
— Pupillary light reflexes, Direct, and consensual
— Corneal reflex.

d) Body:
Head reaction to pinprick, cutaneous muscle reflex
Anal reflex
Micturition

5. Laboratory examination: it includes:
   Dung examination
   Urine analysis
   Haemogram
   Blood Chemistry
   Dung examination:
   See the colour, consistency, composition, frequency, volume and smell.

Urine examination:
Examine for proteinuria, Myoglobinuria, Bile pigments and ketone bodies.

Haematological examination:
PCV should be checked.

Blood chemistry:
In severe muscle damage and liver damage.
1. Serum aspartate transaminase will increase:
   if it is 200 IU/ ml - Guarded prognosis
   500 IU/ ml - Hopeless
2. SGOT may arise to 500 - 1000 sigma units in 6 hours. 2500 - 3000 units in 48 hours, return to normal after one week.

Normal Values

<table>
<thead>
<tr>
<th>Test</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPK</td>
<td>1-69 IU/L</td>
</tr>
<tr>
<td>SGOT</td>
<td>36-69 IU/L</td>
</tr>
<tr>
<td>Na</td>
<td>132 - 152 MEQ/L</td>
</tr>
<tr>
<td>K</td>
<td>3.9 - 5.8 m E/L</td>
</tr>
<tr>
<td>Ca</td>
<td>8.1 - 10.1 mg/dl</td>
</tr>
<tr>
<td>P</td>
<td>4.3 - 7.6 mg/dl</td>
</tr>
<tr>
<td>Mg</td>
<td>1.9 - 3.5 mg/dl</td>
</tr>
</tbody>
</table>
3. Creatinine phosphokinase:

   If it is more than 500 IU/L, poor prognosis.

4. Serum and skeletal muscle K will fall.

   The blood level of all the minerals are reduced but normal ratio of Ca, Mg and P remains undisturbed.

   The calcium level at least does not fall below 7 mg%.

Differential diagnosis:

   All the diseases which lead to recumbancy in cattle has to be differentiated. It is very helpful in giving the prognosis, identifying whether the case is a medical or surgical or obstetric and Gynaecological is important.

1. Medical problems which has to be differentiated are:

   Diseases of bone:

   Osteoporosis, Osteomalacia. In these conditions there will be weakness, staggering, fragile bone and animal will respond to phosphorus therapy.

   Diseases of muscle:

   Rupture of Adductor muscle - Frog like posture, Carpal extensor myositis - Forelimb stretched out, Crampiness - one or both hind leg extended back, contraction of the Ischial.

   Tearing of Fibularis Tertius muscle - Inability to flex hock.

   Cervical vertebra - muscle disease - Torticollis

   Diseases of joints:

   Acute arthritis

   Inflammation of hip joint

   Diseases of hooves:

   Fissured feet

   Laminitis - By using pedobaroscope it was found that high incidence of lameness of hind hooves cannot be associated with load distribution in pregnancy. Rapid change in load of pressure, behavioural, metabolic, nutritional metabolic and housing changes around the time of calving predispose cows to lameness.

   Disease of brain and CNS:

   * Sporadic bovine encephalitis:
     Encephalitis symptoms, yellow fibrin in peritoneum, fluid peritoneum.

   * Brachial / Radial paralysis:
     Inability to extend the foot and carpel joint. When limb is brought forward, the shoulder and elbow joint fail to flex and the claws are dragged on the ground.

   * Damage to peroneal nerve:
     Inability to extend the hock and fetlock.

   * Femoral nerve paralysis:
     Hindlimb collapse when placed on stifle while lifting.

   * Tibial nerve paralysis:
     Incomplete extension of hock, knuckling of fetlock.
Fibular nerve paralysis:
   Inability to flex hock and extend foot

Spastic paresis of hind quarters:
   Permanent contraction of gastrocnemius

Obturator paralysis: Frog like - flexed hind legs.

Painful condition of leg and body.

Staggers in calves, pseudolipidosis:
   Jerky movements, brain changes.

Metabolic disorders:
   Milk fever
   Hypomagnesaemia
   Transit fever
   Ketosis
   Fatty liver syndrome

Metabolic lameness - inadequate mineral phosphorous

Paralytic myoglobinuria - lameness, hard stiff swollen muscles of gluteal region
   Dark red urine,
   Sudden exercise following
   rest on high nutrition

Nutritional deficiencies:
   Rickets
   Mineral deficiencies
   Vit - A def - Oedema of leg feet brisket
   night blindness.

Poisoning
   Plant poisoning
   Ergot poisoning - grasstree macrozamia-
   Fescue lameness
   Paspalum staggers - Ergot poisoning

Toxaemic conditions:
   - Acute mastitis
   - Ingestion with toxaemia
   - Septic pneumonia
   - Peritonitis
Infectious diseases:

Black leg - hot painful swelling of the Leg.
Botulism - Flacid paralysis of the leg.
Tetanus - Rigidity of the muscles
Rabies - Rectal tenesmus accompanied by simultaneous flexion of hind limb. Progressive paralysis of hind quarters 2-4 days continued, terminal recumbency, terminal continued intermittent bellowing.

Parasitic diseases:

Tick Paralysis
Babesiosis
Setaria labiato - Papillosa - motor weakness ataxia
Sarco - Sporidiosis - Myositis microscopic cyst in heart, lung, tongue weakness, lameness, paralysis.

Other Medical causes:

- Weakness
- Ephemeral fever
- Pylonephritis
- Pneumonia
- Neurofibroma
- Starvation
- Heat stroke
- Mycotoxins
- Scours
- Ruminal impaction
- Foreign bodies

Surgical causes:

- Mechanical injury
- Dislocation of Patella
- Fracture of bones - Pelvis, femur 3rd phalanx
- Rupture of round ligament and adductor muscles.
O & G Causes:

- Prolapse of intervertebral disc
- Paralysis of nerves
- Septic metritis/vaginitis
- Ruptured tendons and muscle
- Ruptured uterus
- Calving injury/Paralysis
- Septicemia following calving
- Damage to spinal cord

Treatment of recumbent cow:

- Not specific
- Dosage of drugs may be calculated according to the need
- Ca,P,Mg,K and glucose
  Do not give calcium when temperature and digestion is abnormal
- Vit. E
- CNS Stimulant: Triplename hydrochloride (vetienzamine) 0.5 mg/Kg, slow I/V.
- Antibiotics for septic condition
- Analgesics
- Anti inflammatory
  If creeper, treat with
  - Insulin 150 to 200 unit every 36 hours. It raises the Ca level and favours the reentry of K from cell to ECF.
  - Hydrochlorthiazide: 0.25 to 5 mg i/v, i/m increases the urinary excretion of sodium and chloride without marked variations of body pH.
  - KCl replaces K and stimulates aldosterone secretion. Increased aldosterone acts through an integrated antagonism with insulin to normalise K level sodium chloride 50g, Kcl 20g, mix 10 lit of water give by stomach tube.

Management:

Care of recumbent cattle:

a) Good bedding to avoid complication like dislocation, fracture and non specific injury.

b) Epidermal necrosis and cellulitis (Bed sores):
   It is due to prolonged recumbency avoid moisture and excreta, apply protection bandages to carpus, hock and pastern and topical dressing.
c) Hypostatic congestion and ruminal tympany will occur in prolonged recumbency. So avoid lateral recumbency by placing balls of straw.

d) Ischemic muscular necrosis:
When an animal lies with one limb under her body for any length of time ischemia of that limb occurs due to restricted blood supply by pressure and then leads to necrosis. So, turn the animal every 2 hours and flex and extend for few minutes to restore normal blood circulation otherwise these will damage to branches of sciatic nerve.

e) Heat stroke:
If recumbent animal is kept on open area with hot sun, heat stroke will occur.

f) Neuromotor Psychosis:
- Animal may rise but for one reasons or another animal is unwilling to do so.
- It may fear that it will slip
- It may find it painful to try to rise or simply lost confidence that it can rise
- So be sympathetic in handling
- Place should not be slippery
- Animal should not be kept as it is facing that wall.
- More animals outside tend to rise than those indoor and cows in good condition also have better prognosis.
- Help the animal to rise by using hoists.
- Over recent years several inflatable bags have become available (Downer cow cushion). These have the advantage of being relatively comfortable and support the cow’s body and allow limb circulation to be restored.
- Tie a rope between 2 pasterns to avoid the hind feet slipping apart.

Prevention
- Occurrence of milk fever can be prevented by giving Vit D₃ 600 mg i/v 2 to 8 days before calving
- Retreat it again
- Calcium chloride 150 gm BID for 2 days prior to calving
- Calving in box stalls with straw bedding one foot depth.
- Treat milk fever in I stage itself.
- Exercise daily during dry period to maintain good muscle tone.

NUTRITION:
Adequate fibre 50% of straw in dry matter 25 - 40 gm / day
60 - 80 gm or more of calcium/day
Not more than 50% of dry matter as grains
Avoid abrupt dietary change from 2 weeks before parturition.

Prognosis in recumbent cattle:

It depends on the following factors:
- Period of recumbency
- Presenting signs
- Continual reassessment
- Biochemical assessment
- Nursing.

Period of recumbency:
Mostly cows get up within 4 days of becoming recumbent

Once the animal is down for 10 days the prognosis is poor

However there are cases of individual animals returning to their feet after 2 or 3 weeks or even a month.

Presenting signs:
Possible cause and prognosis can be made by observing the clinical signs and attitude changes in recumbent cattle.

<table>
<thead>
<tr>
<th>Position</th>
<th>Cause</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creeper or Crawler</td>
<td>Hypocalcaemia</td>
<td>Good</td>
</tr>
<tr>
<td>Attempts are made to</td>
<td>Hypomagnesaemia</td>
<td></td>
</tr>
<tr>
<td>rise with the hind</td>
<td>Hypophosphatemia</td>
<td></td>
</tr>
<tr>
<td>quarters being lifted</td>
<td>Peroneal paralysis</td>
<td></td>
</tr>
<tr>
<td>from the ground</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frog legged cow:</td>
<td>Hypocalcaemia</td>
<td>Mainly good</td>
</tr>
<tr>
<td>The hind limbs are</td>
<td>Hypomagnesaemia</td>
<td></td>
</tr>
<tr>
<td>partially flexed and</td>
<td>Hypophosphatemia</td>
<td></td>
</tr>
<tr>
<td>displaced distally</td>
<td>Peroneal paralysis</td>
<td></td>
</tr>
<tr>
<td>Hindlimbs rigidly</td>
<td>Obturator paralysis</td>
<td></td>
</tr>
<tr>
<td>extended rostrally</td>
<td>Tibial nerve damage</td>
<td></td>
</tr>
<tr>
<td>so they are in contact</td>
<td>adductor muscle damage</td>
<td></td>
</tr>
<tr>
<td>with the elbows of the</td>
<td>Often upper limb problems (eg)</td>
<td></td>
</tr>
<tr>
<td>front legs. If the legs</td>
<td>hip dislocation</td>
<td></td>
</tr>
<tr>
<td>placed in normal position</td>
<td>Hip joint Trauma</td>
<td></td>
</tr>
<tr>
<td>often they return to stancel</td>
<td>Rupture of ligament</td>
<td></td>
</tr>
<tr>
<td>Rest on one side</td>
<td>muscular degeneration</td>
<td></td>
</tr>
<tr>
<td></td>
<td>sciatic nerve damage</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Damage to upperside</td>
<td></td>
</tr>
</tbody>
</table>
If moved on to other side then returns to original position if due to muscle flaccidity then upper side is normal

Sciatic nerve damage peroneal paralysis pressure syndrome

Legs extended behind the animal Pubic damage Nerve damage Muscle damage

**ATTITUDE CHANGES AND PROGNOSIS**

<table>
<thead>
<tr>
<th>Attitude</th>
<th>Positive cases</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lateral recumbency with head back Chronic metabolic problems, Brain conditions or damage</td>
<td>Hopeless</td>
<td></td>
</tr>
<tr>
<td>Hyperaesthesia Mainly brain</td>
<td>Poor</td>
<td></td>
</tr>
<tr>
<td>Some show tetany or lateral recumbency Condition or damage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-alert Brain damage Hypermagnesaemia</td>
<td>Poor</td>
<td></td>
</tr>
</tbody>
</table>

Continual reassessment

- Recumbent cattle should be frequently reassessed
- Early stage it is best to visit daily and then every 2 to 4 days.
- At each visit full clinical examination should be performed and any changes noted.
- It will give an indication whether the animal is improving or deteriorating.
- In cases which appear to remain static, the serum enzyme levels may be helpful in assessing prognosis.

Biochemical assessment:

- It should be done where clinical examination fails to indicate the cow’s progress.
- It should be done to animals which appears to be the same at each visit.
- Single sample evaluation is unsuccessful
- Testing and interpretation are based on taking samples
- Starting after the animal has been down for longer than a day.
- Interval between successive tests should be greater than 24 hours.
- There will be fall in CPK after 2 days and in aspartate aminotransferase and urea after 3 days in recovered cattle.
- Combination of condition score, quality nursing and assessment of CPK, AST, Mg & Ca will help in assessment of prognosis.

NURSING:

Whatever be the capacity of veterinarian to diagnose, treat and say prognosis, all will be to no avail if there is inadequate nursing.
MODERN APPROACH IN THE PREVENTION AND THERAPY OF PRODUCTION DISEASES IN DAIRY COWS

Dr. K. VASU

The term 'production diseases' or metabolic diseases includes parturient paresis, hypomagnesemia, acetonemia and some other conditions all of which are attributed to an imbalance between the rates of 'input' of dietary nutrients and the 'output' of production. When the imbalance is maintained, it may lead to a change in the amount of the body reserves of certain metabolites or their 'throughput' and sufficiently large changes in 'throughput' will give rise to signs of production disease. Because of the emphasis being placed on preventive medicine, it may be possible to predict the occurrence of production disease in a herd of lactational group by monitoring certain components of blood on a regular basis. If the level falls below "normal" it is assumed that intake needs to be increased to compensate for the negative balance created by excessive output.

The metabolic profile test is based on the concept that the laboratory measurement of certain components of the blood will reflect the nutritional status of the animal with or without clinical abnormalities or in other words the ability of the laboratory to make an objective assessment of the input-output relationships and the test would be able to detect the qualitative and quantitative adequacy of the diet if cows expected to produce a certain quantity of milk or return to estrus within a desirable length of time following parturition.

Parturient paresis:-

Parturient paresis is a metabolic disease occurring most commonly about the time of parturition in adult female and is characterised by hypocalcemia, general muscular weakness, circulatory collapse and depression of consciousness.

Treatment:-

1) The standard treatment of parturient paresis is intravenous administration of calcium borogluconate 400-800ml of a 25% solution.

2) Administration of glucose 500ml of a 40% solution, sodium acid phosphate, 200ml of a 15% solution and magnesium sulfate at 200-400 ml of a 15% solution.

3) Udder inflation is a valuable alternative treatment in cows which do not respond completely to the initial treatment.

4) Oral administration of gels containing calcium chloride to increase the recovery rate and to prevent relapses.

General Nursing Procedure

1) If the cow is down for long period, she should be moved from side to side 3 to 4 times a day and the legs and bony prominences are massaged.

2) If recumbent for more than 48 hrs, she should be raised in a hip sling several times daily.
Control

1) Maintenance of appetite and avoidance of alimentary stasis in late pregnancy appear to be an important preventive measure.

2) Feeding of high calcium diet before calving is contra-indicated and may increase the incidence of milk fever especially if the diet is alkaline, so much so that the feeding of an acid type diet or a low calcium diet for the last 5 weeks of pregnancy as a control measure is desirable.

Management practices:

a) Avoid excessive calcium intake during dry period.

b) Feed adequate phosphorus to meet requirements or limit calcium intake to more than 100-125 g/day.

c) Avoid overeating by either reducing energy concentrates of the ration or restricting the intake during prepertum period.

d) Avoid stress at the time of parturition.

e) At calving the cow should receive oral dose of calcium gel followed by diet with a high calcium.

Calcium gel dosing

150g of calcium salt given by drench or in the feed 3 doses are given 24 hrs before, 1-2 hrs before and 10-14 hrs after calving is one of the best preventive measures.

Vit. D and its metabolites and analogs:

1) Single dose of vitamin D3 10 million units intramuscularly given 2-8 days before parturition is the most popular prophylaxis against milk fever.

2) Oral dosing with 20 million units of vitamin D2/day for 5 days to cows immediately prior to calving reduces the incidence of the disease.

3) 25 Hydroxycholecalciferol at a dose rate of 8mg 3-10 days before calving repeated at weekly intervals.

4) 1,25-Dihydroxy vitamin D3 200mg/day orally, reduce the incidence.

Miscellaneous Prophylactic Measures:

1) Ammonium chloride is fed with grain 25-100g/day during last few weeks of pregnancy, to produce acidosis and enhance calcium mobilization and ionization to prevent milk fever.

2) Cows should not be subjected to unnecessary exercise or excitement.

3) Good plane of nutrition during late pregnancy and gradual changes to lush pasture.

The downer - cow syndrome:

The downer cow syndrome is a condition which occurs in cattle following hypocalcemic parturient paresis and is characterized clinically by prolonged recumbency even after two successive treatment of calcium.
Treatments:

1. Injection of magnesium salts, phosphates, corticosteroids, stimulant tonics and vitamin E and selenium.

2. Fluid therapy by oral or parenteral route is indicated to cows which may not be drinking a normal amount of water.

3. Comfortable bedding and turn the cow from side to side several times to minimise the degree of ischaemic necrosis and para analgesia.

4. Physiotherapy in the form of muscle massage to restore the normal muscle activity in the affected limbs.

Control

Cows should be treated during the first stage of parturient paresis before they become recumbent. Once recumbent, they should be treated as soon as possible and cows should be well bedded with liberal quantities of straw. Frequent rolling of cows from one side to another on hourly basis.

Lactation tetany (hypomagnesemic tetany, grass tetany):

Lactation tetany is a highly fatal disease of all classes of ruminants and has the highest incidence in lactating cows. It is characterised by hypomagnesemia and usually hypocalcaemia and clinically by tonic-clonic muscular spasms and convulsions and death due to respiratory failure.

Treatment

1. Combination of calcium - magnesium preparation 500 ml of a solution containing 25% calcium borogluconate and 5% magnesium hypophosphite intravenously followed by a subcutaneous injection of a concentrated solution of magnesium salt, magnesium lactate, or magnesium gluconate 15% solution 200-400ml.

2. Feeding of magnesium rich supplements is recommended after parenteral treatment

3. Intramuscular injection of an ataratic drug before commencing specific treatment to control convulsions in acute cases.

Control:

1. Feeding of magnesium supplement: Daily feeding of 120 g of magnesium oxide, magnesium phosphate (53g/day)

2. Heavy magnesium bullets: Place a heavy bullet of magnesium in the reticulum from which is constantly liberates small amounts of magnesium about 1gm daily.

Top dressing of pasture: Calcined magnesite (1125kg/ha) or magnesic limestone (5500kg/ha). are used

4. Spraying pasture with a 2% solution of magnesium sulphate at fortnightly intervals.

5. Provision of shelter in an area where winter pasturing is practiced.

6. Time of Calving: Incidence is high during cold winter months.

7. Feeding on hay and unimproved pasture: Provision of some grain, or rough grazing reduce the incidence.
Ketosis of ruminants (acetonemia of cattle)

Ketosis in ruminants is a disease caused by impaired metabolism of carbohydrate and volatile fatty acids. Biochemically, it is characterised by ketonemia, ketonuria, hypoglycemia and low levels of hepatic glycogen.

Treatment

1. Intravenous injection of 500ml of a 50% solution of dextrose
2. Propylene glycol or glycerine (225gm twice daily for 2 days followed by 110g daily for 2 days) can be administered as a drench in the field.
3. Ammonium lactate 300g, daily for 5 days in repeated doses to be effective.

Hormonal therapy

1.) Dexamethasone - 25 mg
2.) Anabolic steroids 30mg followed by oral propylene glycol (100 ml twice daily).

Miscellaneous treatment:

1. Chloral hydrate : Initial dose of 30g orally followed by 7g twice daily for several days.
2. Vitamin B12 and cobalt 750mg 3 doses at 1-3 days intervals.
3. Addition of nicotinic acid in the feed (12g daily)

Control

1. Adequate caloric intake should be ensured.
2. Use silage or hay as maintenance ration supplemented with 1kg/day concentrate and gradually increased to 5kg daily at calving time.
3. After calving, the concentrate ration should be increased gradually (3kg/100kg. body weight for maintenance and 1kg/3kg milk)
4. Provide good quality and ground maize.
5. Adequate exercise.
6. Ration must contain adequate cobalt, phosphorous & iodine.
7. 110g of sodium phosphate daily for 6 weeks, commencing at calving or propylene glycol (350ml daily).
Surgical intervention for treatment of ruminal affections is required in a variety of conditions. Acute bloat, especially of the frothy type, necessitates rumenotomy, while dry bloat may require surgical manoeuvres of lesser magnitude such as rumenocentesis or rumenostomy (ruminal fistulation). Frothy bloat results sometimes due to intake of large quantities of leguminous feeds like alfalfa, which produce large amounts of froth that the animal cannot remove by eructation. Intake of large amounts of grains (acid indigestion, D-Lactic acidosis or carbohydrate engorgements) leads to excessive production of D-Lactic acid in the rumen, causing severe acidosis endangering life and requires emergency rumenotomy. Alkaline indigestion and ruminal impaction may also warrant rumenotomy. The commonest form of surgical pathology that requires surgical interference of the rumen is traumatic reticulitis or traumatic reticuloperitonitis. As cattle are nonselective feeders, they usually swallow sharp materials like glass, wire, nails etc. that cause traumatic reticulitis. The ingestion of these harmful metallic objects is generally accidental although phosphorus deficiency (causing allotriophagia or pica) may be the underlying cause. If such metallic items are wedged inside the cells of the reticular wall, the epithelium is gradually eroded and a painful ulcer forms. These foreign bodies can suddenly inflict serious injury, when there is a sudden rise in intra-abdominal pressure as occurs during calving. The foreign bodies then pierce the reticular wall and cause traumatic reticulitis or traumatic reticuloperitonitis. Depending on the location, direction and orientation of the foreign body, even the pericardium may be sometimes pierced by these objects causing traumatic reticuloperitonitis.

Diagnosis of ruminal disorders:

Ruminal impaction is diagnosed by palpation of the left paralumbar fossa. A resonant note on percussion of the left flank indicates bloat. Acid and alkaline indigestion are diagnosed by history and estimation of pH of the ruminal contents. Traumatic reticulo peritonitis is diagnosed by symptoms, radiography or sometimes by using metal detector.

Anatomical considerations:

The region of the left flank or the paralumbar fossa is characterised by the presence of loose fascia under the skin. The muscles that are encountered in this region are the obliques abdominis externus, the obliques abdominis internus and the transverse abdominis. The transverse abdominis muscle is in close contact with the parietal peritoneum, so much so, that when incised, both are cut simultaneously. The flank is innervated by the ventral branches of the thirteenth thoracic, the first, and second lumbar spinal nerves. The thoracic and lumbar nerves are accompanied by arteries and veins of similar names.

Local or regional anaesthetic procedures:

Linear infiltration anaesthesia:

Linear infiltration of a solution of 2% lignocaine hydrochloride along the proposed line of incision is the simplest form of local anaesthesia for ruminal surgery. However, since the presence of the local anaesthetic drugs directly at the site of incision interferes with wound healing, other forms of anaesthesia are often preferred.
Inverted ‘L’ block:

This kind of anaesthesia, referred to as field block is accomplished by two linear infiltration in the form of an inverted ‘L’. The whole thickness of abdominal wall should be infiltrated with lignocaine solution. One line of the inverted ‘L’ lies parallel to the transverse processes of the lumbar vertebrae and the other lines caudal and parallel to the last rib on the left side. Upto 20 ml of local anaesthetic solution is required for this block.

Paravertebral block:

In this technique, the thirteenth thoracic, and the first, second and sometimes the third lumbar spinal nerves on the left side are blocked to produce regional anaesthesia of the entire left flank region. To achieve this, a long (about 7 cm long; 18 G) needle is required. By following the last rib with index finger, the head of the rib can be felt about 5 cm lateral to the last rib. This is the site of injection for blocking the thirteenth thoracic nerve. The sites of injection for the lumbar spinal nerve lie about 5 cm away from the dorsal midline on the transverse line drawn immediately behind the spinous process of the particular vertebra. The needle is inserted to a depth of 5 cm and at each site about 15 ml is injected as the needle is withdrawn. This method has the advantage of desensitising the abdominal wall including the peritoneum completely and uniformly. It also produces good muscular relaxation.

Surgical procedures:

(a) Rumenocentesis:

Anesthesia and position: Rumenocentesis can be performed using local infiltration anaesthesia. The animal is controlled in standing position.

Procedure: Using a trocar and cannula of about 3/8” diameter, the skin in the centre of the hollow of the left flank is punctured. If the skin is too tough, a small incision can be made with B-P blade. After puncturing the skin, the trocar and cannula is moved a few cm away and the muscles and ruminal wall are punctured in a sharp thrust. This is done to ensure that the points of puncture of skin and the abdominal wall rumen do not coincide with each other. The trocar is then removed and the gas is allowed to escape. Some medicaments if required, may be administered intraruminally through the cannula. Removal of the cannula should be done only after replacing the trocar and while keeping the flank pressed with fingers.

(b) Rumenostomy or ruminal fistulation:

Anesthesia and position: Rumenostomy is either by local infiltration or by paravertebral block. The site is at the most prominent part of the rumen at the left paralumbar fossa. Standing position is preferred.

Procedure: A circular piece of skin 4-5 cm in diameter is removed and the muscles are exposed. The muscles are split and the ruminal wall is withdrawn and anchored to muscles and peritoneum by continuous chromic catgut suture. The protruding piece of rumen is incised to correspond to the skin edges. The edges of the skin and rumen are sutured together with silk.

(c) Rumenotomy:

Anesthesia and position: Inverted ‘L’ block or paravertebral anaesthesia are suitable. The operation is performed with the animal in standing position and the site is the left paralumbar fossa.
Pre-operative Preparation: The left flank area is shaved, washed with soap and water and the site is painted with an antiseptic like tincture of iodine. Whenever possible, fasting the animal for a period of 12 hours is advisable.

Procedure: A sterile drape is applied to the left flank. A 6-8" long vertical skin incision is made on the left flank. The incision is deepened by cutting through the three abdominal muscles (obliques abdominis externus, obliques abdominis internus and transverse abdominis) and the peritoneum, to expose the rumen. At this stage, hand may be passed into the abdominal cavity and the reticular area is palpated for the presence or absence of adhesions. The next step is the fixation of Weingart's rumenotomy frame or Mc lintocks rumenotomy sleeve.

The Weingart's frame is fixed to the dorsal commissure of the wound by a thumb screw. The first tissue forceps is inserted at the upper angle of the wound, pointing dorsally the rumen wall is then grasped firmly, gently withdrawn through the wound and the forceps hooked into the dorsal eye of the frame. The same manoeuvre is repeated with another tissue forceps at the ventral wound angle, but hooked into the ventral eye of the frame. The exposed rumen is incised vertically and the rumen hooks are hooked into the cut edges of the rumen wall, pulled away and hooked round the frame.

Alternatively, a Mc Lintocks rumenotomy sleeve is inserted through the incision in the rumen wall.

After removing some contents from the rumen the surgeons hand is inserted and a careful search for foreign bodies is made and they are removed from the rumen as well as the reticulum. If a magnet is carried in the hand it facilitates picking up pieces of metal. The cells of the reticulum are explored and any free or penetrating foreign bodies are retrieved.

The surgeon should then rescrub his hands and the rumen wall should be cleaned. The rumenotomy frame/sleeve is disengaged and the ruminal incision closed with a single or double layer of Lemberts suture using No.2 or No.3 chromic catgut. The muscle layers sutured with No.2 or No.3 chromic catgut. The skin incision is closed with a row of horizontal mattress sutures using silk or nylon.

Post-operative care:
- The wound is cleaned and dressed daily with mild antiseptics. Antiseptic ointments are locally applied. A course of antibiotics for 5 days is given to combat infection, if any. the skin sutures are removed on the 8th or 10th post-operative day.

Complications of ruminal surgery:
There are a few post operative complications like infection and peritonitis, which occurs, if proper asepsis is not followed during surgery. Other complications like emphysema of the operated site are not serious and regress spontaneously. Stitch abscess, if noticed, is treated by removal of a stitch to provide drainage and daily dressing.
ADVANCES IN SURGICAL MANAGEMENT OF RUMEN DISORDERS

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Affections of the forestomach of bovine especially those which are due to ingested foreign bodies have become the subject of attention almost all over the world. Among the four compartments of the ruminant stomach, the first two compartments namely the rumen and reticulum act as a unit, since a group of diseases present with dysfunction of rumen and reticulum. In recent years, surgical management of rumen disorders has been increasingly popular among veterinarians.

1. Acute bloat

Acute bloat can be either due to acute obstruction of oesophagus which interferes with eructation or due to the ingestion of appreciable quantities of feed which ferments rapidly causing bubbles with high surface tension resulting in frothy bloat.

The increasing distension of the rumen causes pressure on the diaphragm and ribs resulting in underventilation of the lungs. Further, the great abdominal pressure discourage return of venous blood from the venacava. The mounting intraruminal pressure forces absorption of toxic gases, of which methane is poisonous.

Surgical management of the condition in the first instance includes provision of intraruminal cannula and subsequent removal of obstruction in the oesophagus either by physical manipulation if possible or by oesophagotomy.

In the second instance of frothy bloat intraruminal injection or dosing of silicones by using a probang is essential in order to coalesce the bubbles.

2. Chronic bloat

This usually means that the animal can eructate but only after intraruminal pressure beyond a certain level. Various causes like enlarged mediastinallymphnodes pressing on the oesophagus, large abscess of the liver pressing on the reticular groove, neoplasms, diaphragmatic hernia of the reticulum and Hofflunds' syndrome due to intragastric adhesions are attributed towards this condition.

Surgical management

Serious consideration is to be made in order to justify the surgical correction of these basic causes by intrathoracic surgery, lancing up of liver abscess throug; the reticular wall, diaphragmatic repair etc. If the animal is sufficiently valuable, as a simpler alternative, the palliative operation of rumenostomy can be considered.

Rumenostomy (Ruminal fistulation)

Although various techniques have been practiced, creating an opening with a valve like action is recommended for the particular purpose described here.

Anaesthesia is either by paravertebral block or by local infiltration. The site is at the most prominent part of the rumen at the paralumbar fossa. A circular piece of skin 4-5cm diameter is removed and the muscles are exposed. They are split and the ruminal fold withdrawn and anchored to muscles and peritoneum by continuous chromic catgut suture. The protruding fold of rumen is incised to correspond to the skin edges. The edges of skin and rumen are sutured together with silk or braided nylon. This method has the result of allowing escape of gas, yet it has a valve like action which limits the amount of leakage.

3) Ruminal indigestion

This is a group of conditions where the primary seat of pathology is in the rumen. Potential surgical
indigestion can be divided into 3 groups; (i) acid indigestion, (ii) alkaline indigestion and (iii) primary ruminal impaction.

Acid indigestion occurs when the ox has over eaten on carbohydrates. Rapid acid fermentation causes a rapid drop in pH. Alkaline indigestion results from over ingestion of proteins or urea. In both instances ammonia is liberated and absorbed. The high ruminal pH has a very adverse effect on calcium and magnesium absorption. Primary ruminal impaction occurs after eating placenta or strips of plastic. The blood vessels of the placenta resist disintegration and like the plastic, may during rumen movements, encircle a large mass of coarse ingesta to form a hard impaction.

**Surgical management**

Besides the indicated intravenous medication for acidosis or alkalosis, severely affected cases require emptying of the rumen after rumenotomy. The emptied contents are replaced with food hay, wheat bran and 5-10 litres of rumen fluid. In addition, 100-200 gm of CaCO₃ plus 1-2 kg of cream are helpful. In alkalosis 10ml of concentrated HCl or 50-70ml lactic acid in a few litres of water can be added.

Impaction is almost always eventually fatal unless the impacted mass is removed after rumenotomy. Should dehydration or exhaustion be present, the bovine animal will require intravenous infusion of 5% dextrose saline, plus B complex and B12 vitamins, the later being important as they are powerful metabolic boosters and act as co-enzymes.

4. Foreignbody ruminoreticulitis:

Foreign body ruminoreticulitis is a disease condition, wherein the ingested foreign bodies cause inflammation of the rumen and reticulum. Bovines ingest the food by an instinctively rapid process with limited preliminary mastication. There appears to be no discrimination in the mouth over the palatability of the various ingested substances, resulting in swallow of undesirable matter including metallic objects.

When food materials are deficient of bulk or fibre, or individual nutrients, particularly salt, cobalt, or phosphorus, cattle may develop pica or allotriophagia. Cattle with this condition ingest materials other than normal food to satisfy their needs of bulk, fibre and nutrients. The ingested foreign bodies like nails, pins, pieces of metallic wires, bones, glass pieces, stones etc, may be lying free in the reticulum or may be penetrating. Foreign body in the perihiomul structures which may result in the affections of other visceral organs. The disease is of great economic importance because of the severe loss of production it causes and the high mortality rate.

The success rate in the recovery of animals with this condition by surgical management depends on early diagnosis. Usual diagnostic methods include total leukocyte count, differential count and radiography. Total leukocyte count and differential count alone cannot be relied upon as a diagnostic aid for this condition. Radiography reveals only the radio-opaque foreign bodies. So, physical findings pertaining to foreign body rumino reticulitis should be correlated with haematological, biochemical and radiological findings to assess the stage of the disease and for confirmation.

**Surgical management**

Surgical management of this condition involves the surgical removal of the aetiological foreign bodies form the rumen and reticulum by rumenotomy.

**Rumenotomy**

Rumenotomy is indicated for the removal of injurious foreign bodies, whose presence may cause traumatic ruminoreticulitis or traumatic reticuloperitonitis, or traumatic reticulopericarditis, materials such as plastic bags that are obstructing the reticulo-omasal orifice, and foreign bodies lodged in the distal oesophagus or over the base of the heart.
Rumenotomy is also indicated for evacuation of rumen contents in selected cases of rumen overload. Generally, rumenotomy is limited to those cases in which the causative agent is primarily in the rumen and reticulum. Finely ground feed stuffs, readily pass into the omasal-abomasal region, but coarser, more fibrous materials remain in the rumen for longer periods. Other indications for rumenotomy include rumen impaction and atony of the omasum or abomasum.

**Anaesthesia and surgical preparation**

The left flank area is prepared for aseptic surgery in a routine manner and local anaesthesia is instituted by line block, inverted L block, or paravertebral block.

**Additional instrumentation**

If one does not suture the rumen to the skin as described here, placement of a rumenotomy cuff (Mclintock cuff) or fixation ring (Weingart's frame) will be necessary.

**Surgical technique**

Rumenotomy is performed through a paralumbar incision (a 20 cm incision is generally sufficient) with the animal standing. In large cattle, the flank incisions for rumenotomies sometimes are made just caudal and parallel to the last rib, to place the incision closer to the reticulum. It is essential, however, to leave sufficient tissue caudal to the last rib for suturing (the incision should be approximately 5 cm (2 inch) caudal to the last rib).

Following opening and systematic exploration of the peritoneal cavity, it is necessary to anchor the rumen to the incision to avoid contamination of the abdominal musculature and peritoneum during the rumenotomy procedure. Use a pair of special wide-jawed forceps to grasp and exteriorise a fold of dorsal sac of the rumen to the incision to avoid contamination of the abdominal musculature and peritoneum during the rumenotomy procedure and affix it by continuous suture to the skin. Alternate techniques for isolating the rumen and preventing contamination include the use of stay sutures, a rubber Mclintock ruminotomy cuff, or a fixation ring (Weingart's). These alternatives are quicker than suturing the rumen to the skin, but they are also more easily displaced; the consequent contamination may be disastrous.

The rumen is incised with a scalpel, and the operator, wearing long rubber gloves, evacuates and explores the rumen. The inside of the rumen and reticulum are explored and if a foreign body is present, it is removed. To help locate foreign bodies, the reticulum can be gently picked up with the hand. The area where the foreign body is usually has extended adhesions and cannot be picked up. This is an ideal area to look for foreign bodies. Following this exploration, the reticulum may be swept with a magnet to pick up additional metallic debris. Fresh rumen contents can be placed in the rumen. Alkalizing products may be inserted at this stage in cases of rumen overload, and mineral oil may also be instilled when indicated. The surgeons contaminated gloves are then discarded.

The rumen incision is closed with a row of continuous inverting sutures of no.2 or 3 chromic catgut. A single layer is usually adequate but a double row may be necessary in a large, distended rumen. The surgical site is then irrigated with sterile normal saline prior to removal of the rumen-fixation suture or apparatus. Closure of the laparotomy incision has to be performed in the usual manner.

**Post operative management**

Postoperative medication varies with the indication for the rumenotomy. Although rumen overload often requires intensive fluid therapy, traumatic rumino-reticulitis requires little intensive care. Antibiotics are indicated following the removal of foreign bodies from the reticulum. Oral fluids can be administered following rumenotomy and mild osmotic laxatives, such as magnesium hydroxide, often promote gut mobility.