EVOLVING KNOWLEDGE OF DIGESTIVE DYSFUNCTION

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INTRODUCTION

Digestive dysfunction or indigestion has been a subject of great interest for every bovine practitioner since dairy husbandry has gained momentum. In the dairy cow most attention has been drawn towards the rumen since the outcome of the digestive processes in the rumen for a great part determine the productivity of the cow. Recently nutritionists have investigated feeding methods aimed at avoiding ruminal degradation, in order to cope with the enormous demand for glucose and proteins in a dairy cow producing more than 40 liters of milk per day. Under those nutritional regimes the outcome of the microbial fermentation however remains of great importance. Even small disturbances in the microbial digestion can have an immense impact on the production, and may, when occurring in the second half of the lactation period, ruin the total performance in that lactation period. It is therefore important to recognise and classify any indigestion as early as possible. The same is true for digestive disturbances in replacement stock. These animals have to experience an undisturbed growth during their first two years of life. Only in that case they are able to respond to the present tremendous demands for production. After a short introduction into the classification of the digestive disturbances in the ruminating bovine, this lecture will concentrate on disorders of throughput namely dysfunction of the reticular groove reflex and functional stenosis of the reticulo-omasal orifice and of the pyloric sphincter.

RUMINANT INDIGESTION

In ruminants as in other herbivores microbial fermentation takes place in those parts of the digestive tract that provide the necessary conditions.These conditions are:

- sufficient storage capacity,
- slow passage, resulting in a long retention time
- maintainance of a fluid state at neutral pH,
- a low redox potential (0.2 volts or less),
- a nitrogen source to build bacterial protein
- a possibility to remove byproducts such as VFA (by buffering and absorption) and CO_2 and CH_4 (by eructation and absorption).

The rumen of a cow is a voluminous blind sac. Powerful cyclic contractions result in a thorough mixing 24 hours a day. Saliva provides a low surface tension. A large amount of food as a microbial substrate can be handled, the cow spends 1/3 of the day eating. The pH is relatively neutral and maintained by addition of large amounts of saliva, highly buffered at pH of about 8. The saliva provides bicarbonate necessary for bacterial growth and important for maintaining the buffering

capacity of the rumen. The rumen epithelium also secretes bicarbonate when other anions are absorbed. The rumen absorbs about 70% of the VFA's produced. VFA's are directly transported to the blood. Undigested material remains within the rumen. The long roughage particles form a course layer, floating in the upper part of the ruminal fluid. A relatively small amount of ruminal gas is present in the upper part of the course layer. During the rumen contractions the layer of fibres is rinsed through by the rumen fluid, removing digested particles and inoculating fresh roughage with rumen microbes. Hay particles may stay in this layer up to 5 days, with a half live of 40 - 80 hours. The roughage material is ruminated and when the particles become small enough they become afloat in the rumen fluid and leave through the outlet wich is located fairly low, in order to prevent long fibres to escape fermentation. The omasal orifice and the rumino-reticular fold play a role in filtering the course material. Secondary rumen contractions bring the accumulated gas (CO_2 and CH_4) forward to the cardia, whereupon eructation follows.

Rumen fermentation can either be affected by a disturbance of throughput or by an unbalanced diet. Imbalance of the diet can result from a sudden change in the composition of the diet or from a sudden increase of the amount provided. Unsuitable, toxic or spoiled feeding-stuffs also may affect ruminal fermentation. It is well established that the microbial digestion has an enormous adaptation capacity, but when, as a consequence of the energy demands of the high producing cow, the composition of the diet is already changed unidirectionally that capacity is greatly reduced.

By disturbance of throughput any kind of disturbance that results in a diminished flow of food particles and gas in an oral or aboral direction is understood. An impossibility of transport in the oral direction resulting in a disturbance of the eructation process and free gas bloat, can be the consequence of diseases of the esophagus, the diaphragm, the reticulum or the cardia and of space occupying processes in the thorax. In most of these cases rumination has also ceased. In some cases only rumination is inhibited while eructation is not disturbed. In these cows a large amount of undigested long fibrous particles is present in the faeces. The latter can also be found in cows where the contractions of the reticuloruminal fold are inhibited, so that its filtration function is disturbed.

The first example of a disturbance of throughput that will be discussed in detail is the failure of the reticular groove reflex in young calves. The second will be the functional stenosis also called vagal indigestion or Hoflund syndrome.

CONSEQUENCES OF FAILURE OF THE RETICULAR GROOVE REFLEX.

A syndrome defined by unthriftiness, depression and retarded growth in young dairy calves and associated with the presence of milk in the rumen was recognised and reported by a number of clinicians in the early "fifties". In 1982 a first report was published concerning a similar syndrome in veal calves. The syndrome was further characterised by the clay-like faeces these calves produced, too solid and too white in color. Extensive studies revealed that the syndrome was caused by a

failure of the reticular groove reflex, the calves became "ruminal drinkers".
These ruminal drinkers in fact act as ruminants although they are on a liquid diet. The ingested milk stays in the rumen for a considerable time and the calves are often seen ruminating. The ingested milk is partly digested by the microflora that establishes in the rumen and volatile fatty acids and lactate are produced. Later on a foul smelling liquid containing casein clots can be removed from the rumen. Most of the calves show various degrees of ruminal tympany and the appetite is very low. The calves may die of suffocation because of severe tympany or may suffer from concurrent diseases such as pneumonia, abomasal ulcers or chronic peritonitis. At necropsy the rumens of the calves show severe hyperkeratosis, apparently induced by the propionic and butyric acids produced. There is a hyperplastic villus atrofy of the proximal jejunum, which is supposed to be caused by the large number of bacteria present in the fluid entering the small intestine.
Treatment is aimed at the removal of the bacterial flora present in the rumen and at training the calf by stimulating the reticular groove reflex. The rumen is emptied and washed and tetracyclin (200mg) is applied intraruminally. Training is started by allowing the calf to suck the herdsman's fingers before offering good quality milk of a temperature of about 40° C. The effect of the training can be examined by ballottement of the ventral rumen and the results are confirmed by a rapid change in color and consistency of the faeces. After a succesful training the calf should be offered milk through a floating teat. Calves that do not respond to training should be weaned abruptly and the development of a normal rumen flora can be stimulated by administering rumen fluid from a cow on a normal ration. However, growth performance of such weaned calves is often unsatifactory.
During the last 10 years a number of reports have been published about a ruminal drinking syndrome in dairy and beef calves during the pre-weaning period. In these calves ruminal drinking can occur when for some reason the reticular groove reflex does not function. Especially calves that suffer from other diseases, that are force fed or given additional amounts of milk may become ruminal drinkers.
The pathogenesis of ruminal drinking is not fully understood although the reticular groove reflex has been studied intensively. The closure of the reticular groove is based on a reflex arc, with receptors in the mouth and pharynx. A large number of salts are believed to initiate the reflex but since it has been proven that the reflex for a large part becomes conditioned, the results of the salt tests are to be mistrusted. Obviously one never can be sure whether the reflex was not evoked by signals such as noises from buckets, a regular feeding time, the presence of a certain person in the room and many other stimuli. That maintaining the reflex conditioned is very important in bringing the milk in the abomasum, means that a good drinking management of the calves during the preweaning period is important in preventing ruminal drinking. Irregular feeding times, low quality milk replacer, long distance transports and any other stressful situation can provoke ruminal drinking. The nature and persistency of the "ruminal drinking initiating" process determines whether it is

temporary or the complete clinical picture of a ruminal drinker develops. Ruminal drinking can be temporary and the reticular groove reflex can be restored by the time of the next feeding. It can also become permanent through alterations in the motility pattern of the developing rumen, the exact nature of which is still unknown at present. It is generally believed that thirst stimulates the closure of the reticular groove. Vasopressin administered intravenously stimulates closure in the adult cow. Since ruminal drinking is associated with stress it is postulated that beta-endorphin release may inhibit vasopressin release from the neurohypophysis. However we have tested different dosages of vasopressin in healthy veal calves and found no consistent closure after the injection. Intensive study of the role of neuropeptides in the etiology of ruminal drinking is necessary. Calves that are ruminal drinkers usually have a different drinking behaviour. Their drinking behaviour can be described adequately by "gulping", just as if they drink water in stead of milk. Recently it was found that in calves sucking effected lower incidence and shorter duration of groove openings during milk intake, resulting in less spilage into the reticulum.
In conclusion, in any preweaning calf that shows signs of digestive disturbances with off coloured faeces and that shows inappetence and unthriftiness ruminal drinking may be primarily or secondarily involved.

FUNCTIONAL STENOSIS OR VAGUS INDIGESTION.

Hoflund published in 1940 a study in which he tried to elucidate the pathogenesis of a certain group of digestive disturbances he could recognize in the cow. He suggested that these types of indigestions were caused by injuries to certain branches of the vagal nerve and recognized four types of functional stenosis. He was able to reproduce these four types by cutting vagal branches at various points. He saw two types of stenosis between the reticulum and the omasum, one with atony of rumen and reticulum and one with normal or hyperactive ruminal and reticular motility. He also recognized two types of functional pyloric stenosis, one permanent and one incomplete.
Today the reticulo-omasal and the pyloric stenosis are still regularly found but the theory of the vagal nerve injuries is largely subsided. Mechanical obstructions can cause the same clinical symptoms and vagal nerve damage in other cases is seldom found.
The incidence of pyloric stenosis is much greater than that of reticulo-omasal stenosis because diseases that cause obstruction of the reticulo-omasal orifice are fairly seldom. Chronic reticuloperitonitis and liver abcesses are the main causes of this type of stenosis. Both reticulo-omasal stenosis and pyloric stenosis result in an accumulation of fluid in the rumen. There is anorexia and the course layer of fibres is broken down. This results in a so called splashy rumen. Functional pyloric stenosis is seen more often, partly because the abomasum is involved in most cases of reticuloperitonitis, since this peritonitis can easily spread caudally. Furthermore a number of gastrointestinal disorders cause impairment of passage of food from the abomasum to the small intestine and

subsequent abomasal distension. This results also in a distension of the rumen partly because of the obstruction and the breakdown of the layer of coarse fibres and partly because of reflux of abomasal contents into the rumen (the cow vomits in its rumen).
The clinical differences between a reticulo-omasal stenosis and a pyloric stenosis are only slight. In both cases the cows are anorectic and fecal passage is scant or has ceased completely. The abdomen shows a papple shaped distension and there is a splashy rumen. The coarse layer of fibres floating in the rumen has disappeared or is very thin. The prognosis is poor for both types of stenosis, although in those cases where obstructive diseases of the small or large intestine are the cause of the functional pyloric stenosis, treatment may be succesful. Also in cases of secondary indigestion were the severity of the functional pyloric stenosis may dominate the clinical picture, treatment of the primary cause together with a symptomatic treatment of the fluid and electrolyte imbalancemay be succesful.
On close examination there are distinct differences between a reticulo-omasal and a pyloric stenosis, as a consequence of the more severe changes in water and electrolyte homeostasis in a cow suffering from a pyloric stenosis. Abomasal reflux in cows with pyloric stenosis leads to sequestration of fluid and chloride in the rumen, apart from an already sequestrated amount of fluid and hydrogenchloride in the abomasum. In the rumen the buffering capacity prevents a severe drop in pH although in certain cases a pH of 5.5 can be found. In fact the sequestrated chloride- and hydrogenions are lost for the animal and this results in a hypochloremic hypokalemic metabolic alkalosis. As a consequence of the loss of water through the continuing production of saliva, the sequestration of abomasal secretions in the rumen and the temporary diuresis induced by the metabolic alkalosis severe dehydration develops. Dehydration and hypovolemia subsequently result in prerenal uremia. Therefore the clinical picture of cows suffering from pyloric stenosis reveals a more severe dehydration and uremia resulting in a typical ammoniacal-uremic odour in the expired air and from the mouth. During the coarse of the disease severe muscular weakness may develop as a result of the changes in the electrolyte concentrations accompanying the metabolic alcalosis.
Any gastrointestinal disorder that is accompanied by an impairment of food passage from the abomasum to the duodenum can cause the clinical entity known as pyloric stenosis. The impairment of food passage from the abomasum can be caused by atony or hypomotility of the abomasum through a direct effect on the smooth muscles of the abomasum or by interference with the movements of the abomasum through a subacute or chronic peritonitis in the region surrounding the pyloric part of the abomasum. It can also result from disorders in the small or even in the large intestine that affect the passage of digesta. Therefore functional pyloric stenosis can be found in a number of disorders such as left-sided and right-sided abomasal displacement, abomasal torsion, abomasal impaction, leucosis in the mesenteric lymphe nodes surrounding the pylorus, intestinal obstruction by intussusception , volvulus, mesenteric hernia or strangulation, intestinal obstruction by

sand, cinders or other foreign material, local adhesive peritonitis, diffuse peritonitis and compression of the abomasum by a pregnant uterus extending cranially.
Apart from these disorders causing a pyloric stenosis through a direct effect on the motility of the abomasum or on the passage from the abomasum another group of diseases can be recognized as causative. This group of so called secundary indigestions consists of serious diseases or inflammatory processes located outside the gastrointestinal tract. These diseases cause pyloric stenosis through an inhibition of the motility of the forestomachs and the abomasum. Among them are endometritis, septicaemia, coli mastitis, pyelonefritis, nephritis and other inflammatory processes, the location of which cannot always be found.
Treatment of patients suffering from pyloric stenosis is firstly directed towards the cause of the stenosis and in a number of cases the cow has to be operated. A fluid therapy with electrolyte solutions primarily aimed at restoration of the chloride concentration should be given when the primary cause of the functional stenosis cannot be detected, prior to the surgical intervention and in addition to the treatment of the primary cause. In our opinion it is not necessary to correct the metabolic alkalosis directly with acidifying solutions, for instance ammonium chloride as this is not without danger. If enough fluid is given intravenously (up to 30 to 40 liters of saline solution per day), electrolyte balans will recover spontaneously and uremia will disappear. Studies into the benefit or risk of the administration of potassium containing solutions to these cows have given conflicting results. It is important that the abnormal contents of the reticulorumen are removed using a stomach tube with a large bore. The treatment should be continued until appetite and faecal passage resumes. A number of cows will respond to this symptomatic treatment.
In a fairly large number of cows suffering from primary or secondary digestive disorders the syndrome of functional pyloric stenosis will develop and complicate the overall clinical picture. Anorexia, ruminal distension and disappearance of the coarse layer of fibres, which is replaced by fluid, muscular weakness and the absence of faeces are the consequences of this syndrome. Any treatment aimed at the primary cause of the disease has to be combined with treatment of the metabolic consequences of the functional pyloric stenosis. This is achieved mainly by the intravenous application of large quantities of a saline solution.
When presented with a patient with a history of indigestion we firstly examen the amount and the nature of the rumen contents through palpation. If the rumen is overfilled with fluid material, the layer of coarse fibers has become thin or has disappeared and the history does not indicate a disturbance of fermentation a tentative diagnosis of functional pyloric stenosis is made and irrespective of the outcome of further examination an intravenous administration of saline solution should be started.

CONCLUSIONS

Recently three examples of digestive dysfunction have been

studied intensively, namely ruminal drinking and functional stenosis of the reticulo-omasal orifice and the pyloric sphincter. In veal calves ruminal drinking causes a syndrome comparable to the "blind loop syndrome" in man. A microbial fermentation will be established in any part of the digestive tract where the necessary conditions are fulfilled. Ruminal drinking in dairy calves causes digestive dysturbances because the milk ingested in the rumen induces a dysturbance of fermentation. The functional stenosis of the reticulo-omasal orifice or the pyloric sphincter causes stasis of ruminal contents, overfilling with fluid and a subsequent breakdown of the layer of coarse fibres. The ruminal stasis and the abomasal reflux in cases of pyloric stenosis dysrupts the homeostasis of water and electrolytes in an animal, heavily depending on the recycling of water and electrolytes in the rumen, in order to maintain optimum condition for the microbial fermentation proces in the rumen, a vital proces for ruminants.

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Summary

Dysorders of the microbial fermentation are important causes of indigestion in cattle. This paper concentrates on disturbances of gastro-intestinal motility as a cause of such indigestions. Ruminal drinking is an important problem in veal calves but can also affect dairy calves in the pre-weaning period. In the veal calf an unwanted microbial fermentation is established in the undevellopped rumen, causing retention and fermentation of milk in the rumen. In dairy calves the milk, when entering the rumen in stead of the abomasum, also causes dysturbances of fermentation. The most important aspect of the treatment is training aimed at restoration of the reticular groove reflex.
Functional stenosis of the reticulo-omasal orifice or the pyloric sphincter is a primary or secundary consequence of a large number of digestive disorders of the gastrointestinal tract. Although the clinical picture of the reticulo- omasal stenosis and that of the pyloric stenosis does not differ greatly, upon close examination distinct differences can be detected. The physio-patholgic explanation of these differences is discussed. Treatment is given aimed at restoration of the fluid and electrolyte imbalance.