

# RUMINAL HYPERKERATOSIS AND RUMINITIS IN YOUNG CALVES DUE TO RUMINAL DRINKING

T. Haenichen, L. Bettinelli, W. Hermanns, G. Dirksen

Veterinary Faculty  
University of Munich  
Germany

## Introduction

As early as 1982, Dutch authors (2) pointed out that loss of contraction of the esophageal groove of initially healthy veal calves causes chronic indigestion (latent chronic ruminal acidosis), so-called "ruminal drinking syndrome." In subsequent years, the etiology of the disease has been studied intensively (6,7). In advanced stages, the ruminal mucosa of such calves is substantially proliferated and hyperkeratotic.

The forestomach disease described by the Munich team (4,5,3), on the other hand, is due to acute complications of primary ailments or is the result of feeding errors. Inborn and acquired "drinking weakness"/anorexia (both have a multitude of causes), as well as force-feeding by stomach tube may cause the esophageal groove to close incompletely or not at all. As a result of this, larger amounts of fluid flow into the rumen and reticulum often followed by an acute ruminal acidosis. The forestomachs of calves diseased in this manner were investigated pathomorphologically in a systematic study (details see 1) that also made note of clinical findings. The results of this investigation will be conveyed below.

## Materials and Methods

The material consisted of 37 calves: Deutsches Fleckvieh (30), Schwarzbunte (3) and Braunvieh (4). The animals were divided into four groups based on their age in weeks. Usable histories and clinical findings were only available for 29 of the calves referred from the II Medical Animal Clinic of the Veterinary Faculty. The stomachs of recently expired animals (including those euthanized on the basis of unfavorable prognoses) were initially filled with 7 % buffered formaldehyde and left in the fixative for two days.

The subsequent macroscopic evaluation of stomach contents and mucosa was followed by sampling of ten representative locations for histological examination. Histological examination was preceded by stereomicroscopic evaluation of ruminal villi form and number of villi per unit mucosa. In the case of histological samples, epithelial layers were counted and their average thicknesses measured.

## Results

The forestomachs from the four age groups basically show the same types of pathological alterations; they vary only in degree. In this regard it is important to take into account age-dependent differences in the maturity level of the mucosa. Chronic changes are not evident until the fourth week of life.

### Macroscopic Findings

When examined from outside, the (normally smooth) serosal surface of highly altered forestomachs exhibits a structure similar to the convolutions of the cerebral cortex, a whitish-yellow color deviation and hyperemic subserosal vessels. Palpation reveals both thickening and increased density of the ruminal wall.

The following deviations from the norm, which often appear in combination, can be seen macroscopically or with the stereoscope:

1. Slate grey discoloration of the villi tips: This finding is present in both normally-configured mucosa and in conjunction with more severe degrees of mucosal alteration; the caudal ruminal sacs and the lateral ruminal wall are affected frequently.

2. Broad areas of mucosal discoloration, as well as sloughing of superficial epithelial layers: Corresponding membranous structures are encountered

in the ruminal contents. If they originate from the reticulum, their mesh-like structure often remains evident. The reticulum, esophageal groove, pillars and ventral ruminal sac are particularly affected:

3. Clumping of villi: Over a vast area the ruminal villi are enlarged, rounded and more closely spaced. They may adhere to one another on account of the milky ruminal contents and the proliferated epithelial layers. In the case of severe clumping of villi the entire mucosa is thickened and folded.

4. Mucosal Erosions and Epithelial Necroses: Whereas less extensive erosive and necrotic changes in the epithelium are first detectable under the stereo- or microscope, severe alterations are macroscopically evident on the basis of corresponding epithelial defects, some of which possess a crusty coating. Typical locations are the pillars, the caudal sacs and lateral walls.

5. Mucosal Ulcers: Complications of the above-mentioned changes (especially the erosions and epithelial necroses) take the form of ulcers; aside from this, ulcerous scars devoid of villi can already be seen by the fourth week of life. The pillars and lateral ruminal walls are affected. Individual animals display ulcers with no alterations of the neighboring mucosa in the ventral ruminal sac and in the caudal sacs.

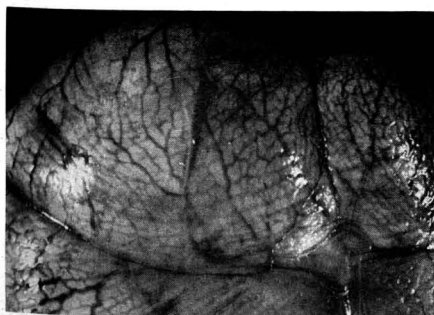


FIGURE 1 Ruminitis: serosal surface, calf, 3 weeks.

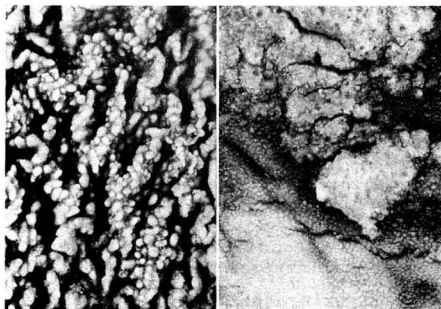


FIGURE 2. Ruminitis: clumping of villi and folding of mucosa, calf 4 weeks (left) ; necrosis of mucosa, calf, 1 week (right)

#### Histological Findings

The investigations confirmed that cellular nuclei or fragments thereof are found in the upper layers of the Stratum corneum of the ruminal mucosa of calves over one week in age, and in desquamated cells. In other words, there is a physiological parakeratosis.

In the case of pathological proliferation of the cornified layers, which is often incorrectly referred to as dyskeratosis, one is actually dealing with a parakeratotic hyperkeratosis. The following pathological processes can generally be distinguished in all three of the ruminant forestomachs:

1) Disturbances in epithelial cornification (parakeratotic hyperkeratosis). This process may be accompanied by a sloughing of the epithelial layers in the sense of erosion.

2) Inflammatory changes in the mucosa (ruminitis). Such changes may be isolated or appear in conjunction with the disturbances in cornification.

Inflammatory processes are almost always involved in the young calves which make up our patient group, and in some cases inflammation actually predominates. An isolated disturbance in keratinization was the exception and, when present, could only be seen in a few of the locations examined. The dark discoloration of the villi tips or of larger mucosal areas corresponds histologically to an infiltration of the epithelium with neutrophilic granulocytes. A frequent

finding in ruminitis is a "hood-like" hyperkeratosis of the villi tips with granulocytic infiltration. In many cases changes in the epithelium and Lamina propria are evidenced by inflammatory cellular infiltrates alone, whereas much deeper levels of the mucosa become inflamed in erosive-ulcerative processes. Severe ruminitis was already demonstrable in 2 of 6 animals in the first week of life. Otherwise, the degree of mucosal lesions generally tends to increase with age.

The hyperkeratosis was objectivized morphometrically; on the other hand, determination of the number of villi per unit mucosa did not yield usable results. The number of villi was reduced in four-week-old calves with a high degree of villi clumping. Aside from the changes in the ruminal mucosa, inflammation and/or disturbances in cornification of the reticular mucosa, and in many cases of the omasal mucosa, could be demonstrated as well.

Table 1 represents an evaluation of

**Table 1**

**Primary disease of twenty five-calves with ruminitis due to "ruminal drinking" or force-feeding**

Inborn "drinking weakness"	Neonatal diarrhea	Diseases of other organs
Force-feeding	7	pneumonia by aspiration 2
Ruminal drinking	2	polyarthritits 1
thereof 3 malformations	thereof 2 meningitis	

Drinking weakness may be presumed inborn in 9 of 25 evaluatable cases. Birth defects (cardiac defects, renal dysplasia) were present in three of these nine cases. In addition to this, the absolute birth weight of many animals was very low. Diseases such as meningitis, pneumonia and, especially, neonatal diarrhea were diagnosed in the group of animals with acquired drinking weakness (13).

**Discussion**

First of all it should be emphasized, that even very young calves (by the end of the first week of life) may display severe changes in the mucosa of the forestomachs. In the picture of the disease described here, inflamma-



**FIGURE 3.** Hyperkeratosis omasum, calf 1 week, H & E 200 x, (left); infiltration of hyperkeratotic epithelium with granulocytes, rumen calf, 2 weeks, H & E 200 x (right).

the anamneses with respect to whether "drinking weakness" was an inborn or acquired ailment.

tory processes predominate over keratinization disturbances. This differs from the mucosal changes described by Dutch authors in conjunction with the ruminal drinking syndrome in veal calves.

From both a physiological as well as a pathological standpoint it would appear that the milk calf should no longer be considered as a monogastric animal. The forestomachs of the young calf are not merely functionless sidechambers of the abomasum. Along with the esophageal groove, they fulfill physiological digestive tasks from the time of birth. They have a transport function and their physiological development is only insured when certain conditions with respect to fermentation and bacterial composi-

tion of the contents are met. One may assume that the height of the epithelium in the forestomachs and the villous profile in the rumen of milk calves are not constant. The present investigations indicate a large capability for adaptation to the existing fermentation conditions, and the boundary to pathological hyperkeratosis is a gradual one.

The composite picture afforded by the morphological alterations in the forestomachs examined, in conjunction with the histories and the clinical and clinical-chemical findings, allow the following conclusions to be drawn with respect to the pathogenesis of the ruminitis observed in these calves:

Relatively large volumes of fluid enter the reticulorumen on account of the aforementioned laxity or failure (bypass) of the esophageal groove reflex (latter due to force-feeding). Depending on their type, amount and residence time in the forestomachs, as well as the types of bacteria present therein, the carbohydrates in these fluid feeds are fermented to acetic, propionic, butyric and lactic acids. In some cases propionic acid fermentation predominates, in others butyric acid fermentation. The extent to which other nutrients contribute to the acidosis remains unclear. As long as the abovementioned acids are repeatedly formed in sufficient concentration and over a period of time, butyric and propionic acids will develop their stimulative effect on the growth of the mucosa in the forestomachs, thereby initiating its proliferation. The "physiological parakeratosis" then develops into a pathological parakeratotic hyperkeratosis, thereby leading to a higher degree of mucosal sensitivity to chemical and mechanical irritations. Lactic acid is considered to play the major role among the chemical stimuli, and is presumably responsible for the expansive inflammatory processes and accompanying epithelial losses. Additional mechanical insults may be introduced by ingested hairs or plant fibers, as has been demonstrated on numerous occasions.

If they have proceeded to an advanced stage, acidosis in the forestomachs and ruminitis determine the further course of the disease. If the ruminitis was initially viewed as the result of "ruminal drinking", it

now becomes the **cause** of dysfunction of the esophageal groove reflex - indeed even in calves with acquired anorexia. A vicious circle is thereby completed which is difficult to break by therapeutic means. The simultaneous negative effects of unphysiological acid production on metabolism, organ function and general patient status make themselves known. It is very questionable whether the juvenile rumen, with its more or less inflamed mucosa, is capable of absorbing notable amounts of acid. The deleterious systemic effects are likely to stem primarily from the outflow of gastric contents into the abomasum and intestines, and the resulting absorption of acids and toxins in these areas. Accordingly, these patients often display intermediate to severe metabolic acidosis in addition to signs of intoxication, exhaustion and circulatory insufficiency. In this manner it is possible for the systemic consequences of indigestion to lead to lethal illness, be it as the result of an inborn "drinking weakness" or as the complication of an acquired primary ailment.

#### Summary

The ruminal mucosa of 37 calves, up to four weeks of age and afflicted with ruminal drinking for various reasons, was examined both macroscopically and histologically for pathological changes. Parakeratotic hyperkeratosis and/or inflammatory alterations of the ruminal mucosa were seen in 25 cases. Aside from hyperkeratosis, the changes included a grey discoloration of the villi and/or more extensive areas of the mucosa, clumping of villi, mucosal necrosis, erosions or ulcerations, sometimes to the point of cicatrization. Reticulum and omasum were often affected as well.

The morphological changes to the ruminal mucosa seen in this study are the result of a prior ruminal acidosis characterized by production of volatile fatty acids (especially butyric acid) and lactic acid, and differ from those seen in veal calves suffering from the so-called "ruminal drinking syndrome".

### Hyperkeratose und Entzündung der Vormagenschleimhaut von jungen Milchkälbern nach "Pansen-trinken"

Von 37 bis vier Wochen alten Milchkälbern -darunter 29 intra vitam diagnostizierte "Pansen-trinker"- wurden die Vormägen pathologisch-anatomisch und histologisch untersucht. Parakeratotische Hyperkeratose und/oder Entzündung der Pansenschleimhaut wurden in 25 Fällen festgestellt. Außer Hyperkeratose umfaßten die Veränderungen graue Verfärbung einzelner Zotten oder ganzer Schleimhautareale, Zottenverklumpung, Schleimhautnekrosen, Erosionen und z.T. vernarbte Ulzera. Haube und Psalter waren häufig miteinbezogen.

Die morphologischen Veränderungen bei diesen Kälbern sind die Folge einer vorausgehenden durch Produktion von flüchtigen Fettsäuren (vor allem Buttersäure) und Milchsäure. Sie unterscheiden sich von denen beim sogenannten "Pansen-trinker-Syndrom" der Mastkälber.

### Hiperqueratosis de la panza y ruminitis en terneros de leche causadas por "beber en panza"

Se examinaron las membranas mucosas de la panza de 37 terneros hasta la edad de 4 semanas afectados por "beber en panza" buscandose macroscópicamente e histológicamente alteraciones patológicas. Hiperqueratosis paraqueratótica y/o alteraciones inflamatorias se hallaron en 25 casos. Además de la hiperqueratosis se podía observar una descoloramiento grisáceo de las vellocidades y/o áreas mas extensas, adherencias de las vellocidades o necrosis de la mucosa, erosiones o úlceras a veces cicatrizadas. El bonete y el libro también estaban afectados frecuentemente.

Las alteraciones morfológicas de la mucosa de la panza en éste estudio resultan de una acidosis ruminal procedida y estan caracterizadas por producción de ácidos grasos volátiles (sobre todo ácido butírico) y ácido láctico y así las alteraciones son distintas de las observadas en terneros cebados afectados del llamado "ruminal drinking syndrome".

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