RUMEN ACIDOSIS WITH SPECIAL EMPHASIS ON DIAGNOSTIC ASPECTS OF SUBCLINICAL RUMEN ACIDOSIS: A REVIEW

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Summary. Increased genetic potential for high milk yield and economic increased benefits in feeding rations high in starch, may increase the occurrence of rumen acidosis in Danish dairy herds. Together with enhanced focus on nutritional production diseases in recent years, the demand for professional knowledge and advisory work is substantial. The present paper reviews rumen acidosis, its etiology, pathogenesis, occurrence, significance, diagnostics and prophylaxis with special attention to Subclinical Rumen Acidosis (SRA). Details of the rumenenocentesis procedure for obtaining rumen fluid are presented together with original observations on rumen pH determinations performed on such samples compared to samples obtained by stomach tube. Differences and similarities between the subclinical and the acute form are illustrated. Our understanding of fundamental parts of pathogenesis, significance and diagnostic of set is still insufficient. It appears that the resulting metabolic acidosis is best reflected in urine. Most other suggested diagnostic parameters lack evidence of usefulness under practical conditions. Adjustments of suboptimal feeding and management routines are believed to be essential prophylactic steps.

Keywords: cattle, rumen, subclinical acidosis.

DIDŽIOJO PRIESKRANDŽIO ACIDOZĖ, ATSIŽVELGIANT Į DIAGNOSTINIUS DIDŽIOJO PRIESKRANDŽIO SUBKLINIKINĖS ACIDOZĖS ASPEKTUS: APŽVALGA

Santrauka. Padidėjęs aukšto pieno produktyvumo karvių genetinis potencialas ir ekonomiškai išaugęs pelnas, šeriant karves racionu su dideliu krakmolo kiekiu gali padidinti didžiojo prieskrandžio acidozių skaičių Danijos galvijų bandose. Kartu su išaugusiu dėmesiu mitybos ligoms paskutiniaisiais metais, labai svarbus yra profesionalių žinių poreikis ir konsultacinis darbas. Šiame straipsnyje apžvelgiama didžiojo prieskrandžio acidozė, jos etiologija, patogenezė, paplitimas, reikšmė, diagnostika ir profilaktika, ypatingą dėmesį skiriant didžiojo prieskrandžio subklinikinei acidozei (SRA). Taip pat pateikiamos detalės apie rumen enocentezės procedūrą didžiojo prieskrandžio turiniui paimti ir originalūs tokiu būdu paimto prieskrandžio turinio pH nustatymo įvertinimai, lyginant su pavyzdžiais, paimtais zondu. Pateikiami subklinikinės ir ūmios acidozės skirtumai ir panašumai. Mūsų supratimas apie pagrindinius patogenezės etapus, diagnostikos reikšmę iki šiol yra nepakankamas. Akivaizdu, kad medžiagų apykaitos acidozė geriausiai atsispindi šlapime. Daugumai kitų siūlomų diagnostinių parametrų nepakanka jų naudingumo įrodymo praktikoje. Manoma, kad esminiai profilaktikos etapai yra optimalus šėrimo sureguliavimas ir priežiūra.

Raktažodžiai: galvijas, didysis prieskrandis, subklinikinė acidozė.

Introduction. Concern in animal welfare has been increasing in recent years (Swanson, 1995; Bain et al., 1998; Dawkins, 1998). This is reflected in an intensive debate about the living conditions of production animals. As a consequence production diseases have come into the glare of publicity as these sufferings to a wide extent arise as a consequence of the production conditions in which the animals are confined and fed. Within the primary milk production enterprise, particular interest has been given to the nutritional production diseases, including SRA (Enemark, Jørgensen, 2001).

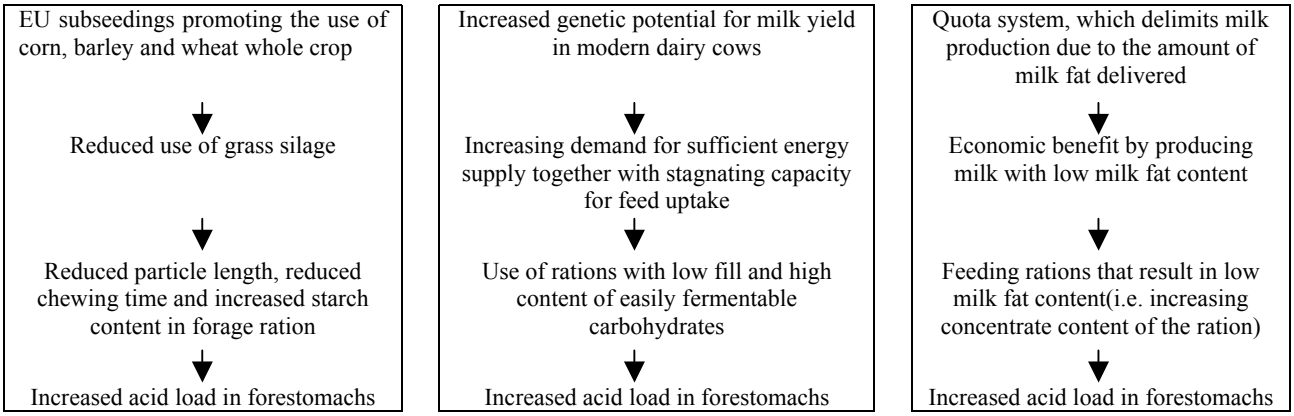

Figure 1. **Schematic view of factors, which may indirectly affect the occurrence of subclinical rumen acidosis**

The subclinical type of rumen acidosis was first mentioned by Dirksen (1965), who characterised the condition as *chronic latent rumen acidosis* and claimed the condition to occur far more frequently than its acute, clinical manifestation.

In connection with intensive milk production, as seen in Denmark, several conditions may indirectly influence an increased occurrence of SRA (Figure 1). In particular, economical considerations may be expected to be of great influence. Recent analyses of 2918 winter feeding schedules and 2024 summer feeding schedules respectively show a growing tendency towards an increased content of starch per SFU (Scandinavian feeding unit for cattle) to the detriment of sugar content and digestible cell walls (National Department of Danish Cattle Husbandry, 1995, 1996). Especially barley and wheat contents in feeding schedules rises while turnip contents are falling. This points at a growing importance of SRA in relation to the health of Danish dairy cows.

Literature on this disease is scarce and not much is known about its incidence. In Denmark this may be reflected in inadequate registration (Blom, 1993), underlining the need for further research into the disease (Jørgensen et al., 1993a). Limited knowledge of the symptomatology as well as the quality of the diagnostic methods normally applied in practice (Enemark and Jørgensen, 2001) are likely to be among the most important reasons for this.

Thus, the purpose of this paper is to update knowledge on aetiology, pathogenesis, symptoms, prevalence, importance, diagnostics and prophylaxis of SRA in dairy cattle.

Definition. The applied nomenclature is confusing and uncoordinated as it varies between countries as well as within countries. It appears from Table 1 that there is some doubt as to whether SRA should be characterised by its presumed duration or by its lack of clinical manifestations. This terminological confusion is largely due to the complex nature of the disease which makes it difficult to exactly delimit the normal physiological rumen conditions to subclinical rumen acidosis. Based on biochemical and microbial conditions in the rumen fluid, it is possible to distinguish between the above-mentioned conditions (Figure 2). Thus, as concerns the rumen environment, SRA can be defined as a condition characterised by a rumen pH between 5.0 and 5.5 where the total concentration of short chain fatty acids (SCFA) has been increased, where the ratio between acetic acid, propionic acid and butyric acid has been shifted towards propionic acid and butyric acid, and where accumulated concentrations of lactic acid in the rumen fluid does not exceed 5-10 mmol per litre (Hibbard et al., 1995). The rumen microbial flora is characterised by a dominance of Gram-negative bacteria though the number of Gram-positive bacteria is increasing (Hibbard et al., 1995; Owens et al., 1998). In contrast, the acute clinical rumen acidosis is characterised by pH values below 5.0, accumulation of lactic acid up to 300 mmol per litre (Hyldgaard-Jensen and Simensen, 1966) and defaunation (absence of protozoa) of the rumen fluid. Here the rumen microbial flora is dominated by Gram-positive bacteria of which the majority belong to the genus *Lactobacillus* (Dunlop, 1972).

Table 1. **Nomenclature concerning subclinical rumen acidosis in different countries**

USA	Subacute rumen acidosis (SARA) Rumen acidosis Subclinical rumen acidosis (SRA) Acidosis Mild acidosis
UK	Metabolic acidosis Off feed Simple indigestion Milk acidosis
Germany	Chronisch-latente Pansenazidose (Chronic latent rumen acidosis) Subklinische Pansenazidose (SRA) Metabolische Pansenazidose (Metabolic rumen acidosis)
Denmark	Subklinisk vomacidose (SRA) Vomacidose (Rumen acidosis) Sur vom (acidic indigestion)

Etiology and Pathogenesis. Subclinical and acute, clinical rumen acidosis respectively, represent two different disease entities. This is explained by differences in their pathogenesis and aetiology (Figure 3).

Regulation of rumen pH

When unregulated, the final product of the catabolism of carbohydrates, the SCFA, tend to lower rumen pH, whereas a low rumen pH is primarily counteracted by neutralisation and absorption.

Neutralisation

Saliva has a dominant intraruminal buffering effect at pH values above 6.0. It has a pH value of 8.4 (Kay, 1966) and is rich in bicarbonate and phosphate (Kaufmann and Hagemeister, 1969). The saliva secretion, and thus the quantity of saliva running into the rumen, is influenced by the chewing - and rumination time. Fiber with a certain particle length and elasticity in the feed ration is essential to the length of the chewing- and rumination time (Van Soest, 1994).

Absorption

Ionised as well as non-ionised SCFA is absorbed through the ruminal mucosa. Consequently, an optimal absorption capacity of the rumen mucosa is critical to the pH- regulating mechanisms. In practice, this relates to an often expedient change from dry cow ration to lactation ration. Dirksen et al. (1984) showed that an adaptation period of minimum 4 weeks is required in order to achieve optimum proliferation of the rumen mucosa. Transport of the ionised SFCA fraction is an energy consuming process involving the secretion of bicarbonate into the rumen fluid (Ash and Dobson, 1963, Gäbel, 1990), whereas a passive diffusion transports the non-ionised part. At pH > 6.0 the majority of the SCFAs are in the ionised form (pKa = 4.8) making the active transport dominant. Increased SCFA production resulting in decreasing pH therefore favour the passive diffusion which is faster than the active transport. Thus a rising concentration of SCFA in the rumen fluid is counterbalanced by an increased absorption (Carter and Grovum, 1990; Dijkstra et al., 1993).

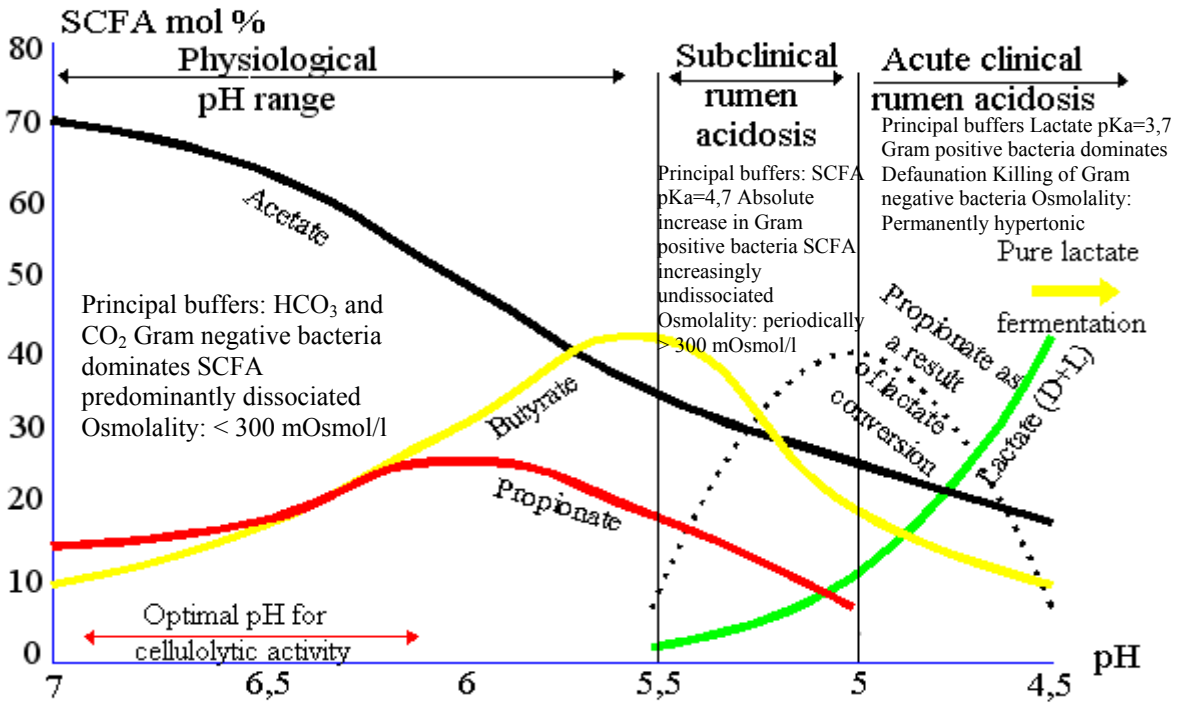

Figure 2. **Fermentation pattern and rumen environment characteristics in relation to rumen pH. The feed ration contained a considerable amount of fodder beets causing a relatively high concentration of butyric acid (modified after Kaufmann & Rohr)**

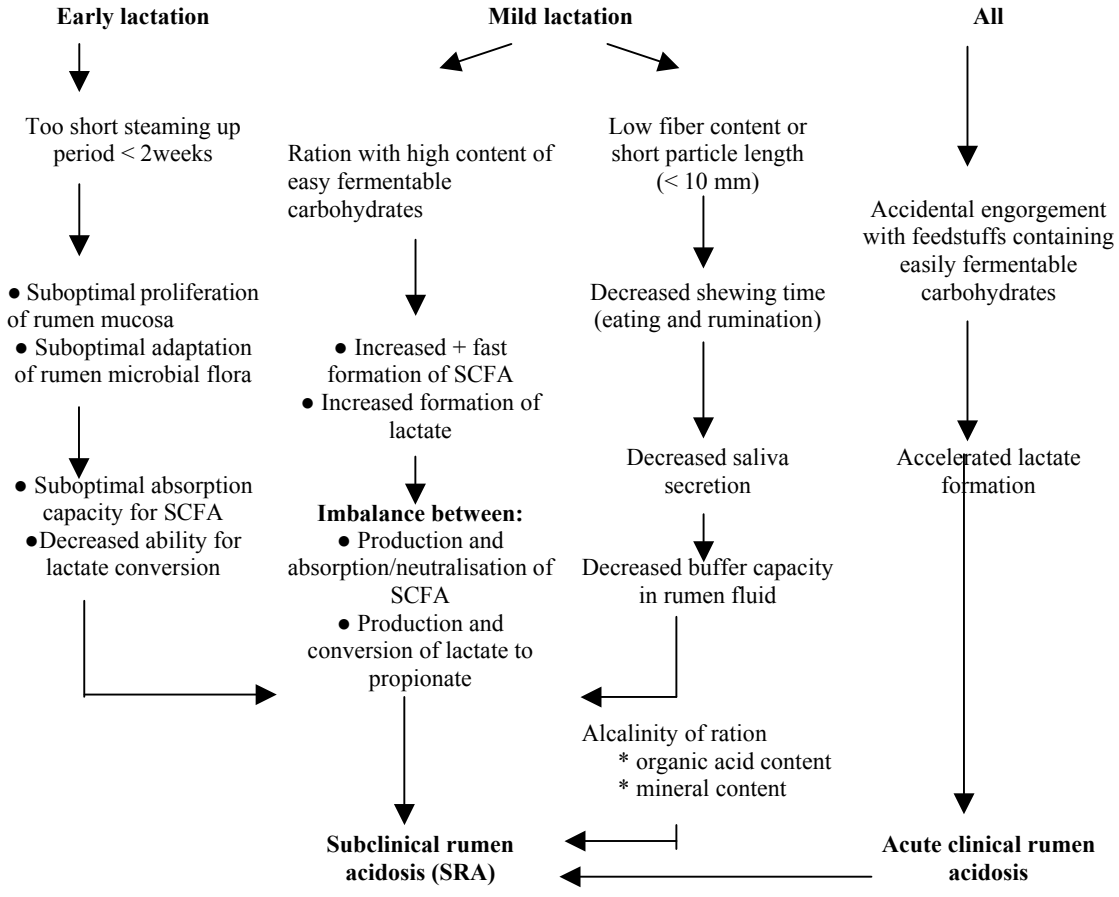

Figure 3. **Schematic view of causal relationships in subclinical and acute clinical rumen acidosis in cows. See text for details**

Acute lactic acidosis (barley poisoning; grain engorgement)

The condition is well described (Hyldgaard-Jensen and Simesen, 1966; Dunlop, 1972). It usually results from accidental consumption of large amounts of easily fermentable carbohydrates as found in grain, grits, fodder beet, turnips, waste bread, apples or other feeds containing high amounts of accessible starch, sucrose, lactose, saccharose, fructose or glucose (Krogh, 1959; Simesen and Konggaard, 1970; Dunlop, 1972; Giesecke et al., 1976; Shukken et al., 1985). The carbohydrates are intraruminally converted to glucose which via pyruvate results in the formation of SCFA, as illustrated in Figure 3. If this process takes place too fast and to a large extent, natural regulators of forestomach pH are overloaded. Consequently, the rumen pH drops drastically below 5.5. This results in the one-sided favouring of acid-resistant, lactogenic bacterial species such as *Lactobacillus spp.* and *Streptococcus spp.* (Mackie et al., 1978). Under normal circumstances, lactic acid is only present in small quantities in the rumen fluid (<5 mmol/l) and is usually controlled by a relatively acid-resistant lactolytic bakterial flora dominated by *Megasphera elsdenii* and *Selenomonas ruminantium* and possibly by protozoa (Mackie et al., 1978; Colemann, 1980; Counotte et al., 1983; Mackie et al., 1984; Nagaraja et al., 1992; Williams et al., 1991; Mendoza et al., 1991). Rumen pH is further reduced by the relatively fast and large production of lactic acid (pKa = 3.8) leading to the deterioration of lactolytic bacteria.

At rumen pH below 5.0 lactogenic bacteria dominate resulting in excessive lactic acid production whereby D- and L lactic acid isomers accumulate in the rumen fluid. The rumen contents becomes hypertonic and fluid is drawn into the rumen from the extra cellular compartment, resulting in a systemic dehydration. Presumably intoxication and systemic acidosis causes the cow to resent drinking, which would otherwise counteract the hyperosmolality. In addition to lactic acid, the contents of histamine, tryptamine and tyramine in the rumen fluid are increased thereby contributing to the clinical picture after absorption into the blood (Koers et al., 1976; Dirksen, 1986). Presumably the dominant toxin is D-lactic acid, which is only formed microbially and which is only slowly transformed by the mamal organism (Dunlop and Hammond, 1965). Also its excretion is relatively slow as compared to the L-lactic acid which is a normal part of the anaerobic glycolysis in muscle tissue (Dunlop, 1972).

The condition is clinically characterised by intoxication, the symptoms of which are loss of appetite, rumen atony, diarrhoea, grinding (pain) and paresis. The consequences of failure to treat, or the late treatment of the disease, is coma or death (Dirksen, 1986). Among clinical findings are lactic acidosis, haemoconcentration, dehydration, varying degrees of rumenitis and hyperosmolality. As a consequence of the reduction in blood bicarbonate content, the buffer capacity of the blood is severely reduced. In fatal cases blood pH may drop below 7.0 (Aslan et al., 1995).

Subclinical rumen acidosis

As mentioned above, under normal physiological conditions a balance exists between production of SCFA and their absorption and neutralisation, respectively. The most important SCFA are acetic, propionic and butyric acid. Their total and relative concentrations depend, among other things, on the source of carbohydrates, pre-treatment of feed and feeding portions. The decomposition of starch generally favours production of propionic acid while decomposition of cellulose primarily results in the formation of acetic acid, and fermentation of soluble carbohydrates results in a relatively large formation of butyric acid (Figure 4).

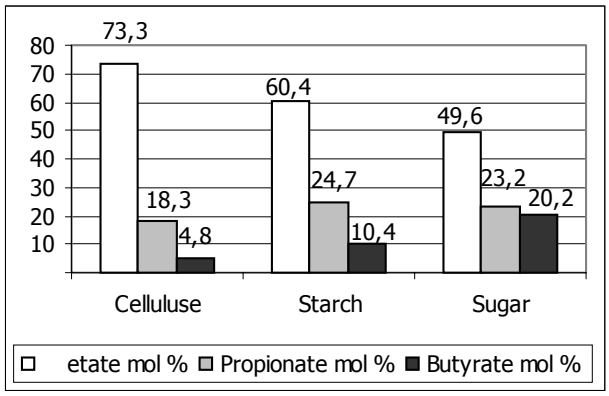

Figure 4. **The influense of various types of carbohydrates on the fermentation pattern in the rumen (after Ørskov & Ryle, 1990)**

If the content of easily metabolised carbohydrates in the feed ration is increased considerably at the expense of the structure content or, if the size of particles in the roughage is limited, then chewing and rumination time is shortened and consequently the saliva secretion is reduced (Kaufmann and Hagemeister, 1969). At the same time the SCFA production is increased to such an extent that it exceeds the absorption capacity of the rumen mucosa. The result is a lowering of the pH as well as a shifting of the rumen fluid's buffer capacity to an area around pH 5. In this situation the volatile fatty acids and their corresponding bases (pKa = 4.7) will constitute the most essential buffer system (Gäbel, 1990).

Similar conditions may arise when newly calved cows are adapted too fast to a lactation ration thereby exceeding the absorption capacity of the rumen mucosa. Finally, a coarse feed containing large amounts of lactic or butyric acids may markedly affect the acidity of the rumen fluid. Presumably, lactic acid is not accumulated to any appreciable extent in cases of subclinical rumen acidosis as amylolytical and lactolytical bacteria are generally balanced (Mackie et al., 1978). This can possibly explain lengthy, apparent non-pathogenic conditions of rumen pH between 5.0 and 5.5. In contrast, experience shows that even minor irregularities in feed uptake, as seen in connection with other diseases, or deceased feed intake as seen around calving and during transport, may cause rumen acidosis when the feeding activity is resumed. (Enemark, Jørgensen, 2002).

The symptomatic picture of SRA is diffuse and inconsistent (Nordlund et al., 1995), often manifested by varying or reduced appetite and sub-optimal production and perhaps loose faeces (Britton and Stock, 1986). Paraclinical findings are not nearly as distinct as in cases of acute clinical rumen acidosis although a slight dehydration,

varying degrees of compensated metabolic acidosis, aciduria, hypercalcuria, hyperphosphataemia, hypokalaemia and hypercalcaemia may occur (Fürll, 1994).

Occurrence. Although knowledge on the incidence of SRA is limited, the disease is believed to be commonly occurring as evidenced by the fact, that addition of buffers to TMR rations is almost standard in North American dairy herds (Erdman, 1993). At the herd level, usually two distinct risk groups are defined. One risk group is cows in early lactation exposed to too quick adaptation to energy rich feed rations resulting in low rumen pH, whereas the other risk group is cows in mid-lactation who, due to their large feed intake, are particularly sensitive to sudden changes of feed or faults in feed composition and -delivery (Nordlund et al., 1995). Danish reports show a 0.2% occurrence of rumen acidosis (Blom, 1993). This figure gives no indication of the actual occurrence as only few veterinarians include analysis of rumen fluids in their examination of patients (Enemark and Jørgensen, 2001). It is more likely that the figure expresses the occurrence of acute clinical rumen acidosis which is easier to diagnose due to its specific history and its clear symptomatic picture. Nor are there in Germany (Dirksen, personal information) or in USA (Nordund, personal information) any statistics of occurrences of SRA. Most likely, regional differences can be expected based on varying use of feeds (Andersen, 1991; National Department of Danish Cattle Husbandry, 1996).

Importance

SRA has been mentioned as a possible etiological factor for a number of diseases (Dirksen, 1985*;* Nordlund et al, 1995; Nocek, 1997) . Unfortunately, documentation is in many cases extremely inadequate. Possible implications and probable causes are briefly described below and illustrated in Figure 5.

Rumenitis

Rumenitis is a frequent sequela to rumen acidosis. At present, the pathogenesis is not fully understood but an increased production of SCFA, particularly butyrate and propionate, as well as a temporary rise in ruminal lactate and fluctuations in the osmolality of the rumen fluid may be involved in the development of rumenitis (Dirksen, 1985; Krehbiel et al., 1995). The stage between parakeratosis (thickening of the stratum cornea of the rumen mucosa) and rumenitis appears undefined (Dirksen, 1985). Parakeratosis*,* when occurring as a consequence of acute increased lactate production caused by induced clinical acute rumen acidosis, may affect SCFA absorption in the long run (Krehbiel et al., 1995). Mucosa lesions as part of rumenitis may serve as an entrance for *Fusobacterium necrophorum,* and more rarely *Acanobacterium pyogenes*, with subsequent colonisation. Embolic spreading to the liver result in abscess formation (the rumenitis liver abscess complex*)*, occasionally with metastasis to the pulmonary circulation via the posterior vena cava causing rupture of minor pulmonary arteries into the bronchi (the caudal vena cava syndrome). Clinically the aforementioned episodes may lead to epistaxis and/or haemoptysis, characterised by bloody, foaming expectorate around the muzzle and nostrils. Generally, the outcome of these cases is fatal (Nordlund et al., 1995).

Metabolic acidosis

Lactate, and in particular D-lactate, is responsible for the profound, uncompensated metabolic acidosis seen in cases of acute clinical rumen acidosis (Dunlop, 1972), whereas in cases of subclinical rumen acidosis the role of lactic acid is less clear.

It is uncertain to which extent the low rumen pH is reflected in systemic metabolic acidosis and hence whether a metabolic acidosis is always to be expected. Probable decisive factors are the depth of the pH fall as well as the duration of episodes where pH is below a physiological acceptable value (e.g. 5.5) (Nocek, 1997). It has not yet been determined whether lactate has any influence (Counotte et al., 1983; Höltershinken et al., 1997), as it apparently does not accumulate in the rumen fluid (Hibbard et al., 1995).

Among the short-chained volatile fatty acids, only the acetic acid reaches the peripheral circulation. Butyric acid is transformed largely in the rumen wall into hydroxy- butyric acid whereas all the propionic acid is converted into glucose in the liver (Owens et al., 1998). However, German research has shown that serious cases of intracellular acidosis may occur even under low-graded, chronic acidosis conditions (Lachmann and Siebert, 1980; Lachmann et al., 1985). On a speculative basis this may compromise the cell function in the rumen wall and the liver, resulting in high SCFA concentrations in the peripheral circulation causing metabolic acidosis (Owens et al., 1998). Recent research indicates that long-lasting metabolic acidosis may also cause damage on the organism in the form of reduced glucose dependend insulin secretion (Bigner et al., 1996), increased cortisol secretion (Ras et al., 1996), reduced fagocytosis activity (Rossow and Horvath, 1988) and reduced migration speed of neutrophiles (Hofirek et al., 1995). It has been shown in man that metabolic acidosis results in increased protein catabolism and consequently growth-impairment (Bailey, 1998). Furthermore, bovine chronic metabolic acidosis *ante partum* affects the steroid hormone concentration around the time of calving, weakens the contractility of uterine smooth muscles (Ras et al., 1996), strains the liver function (Lechowski, 1997) and causes dystocia. Also, weak born acidotic calves and increased disease incidence during the neonatal period has been documented (Ras et al., 1996). Immunosuppression and suboptimal metabolism may be primary complications in long-lasting cases of metabolic acidosis. This may explain reduced resistance to diseases such as respiratory diseases (Mwansa et al., 1992) as well as low production results in herds suffering from SRA (Nordlund et al., 1995). Impaired liver function may partly explain the connection identified between aciduria and increased concentrations of liver enzyme, as described by Markusfeld (1987). His conclusion was however, that the acidosis is probably a reflection of an increased mobilisation of fat.

The apparent importance of systemic acidosis is interesting in relation to the use of anionic salts applied for the prophylaxis of milk fever in late pregnancy (Jardon, 1995). At present, no investigations seem to focus on whether the metabolic acidosis hereby induced may have any hazardous effects either on the dry cows of the herd or on their foetuses.

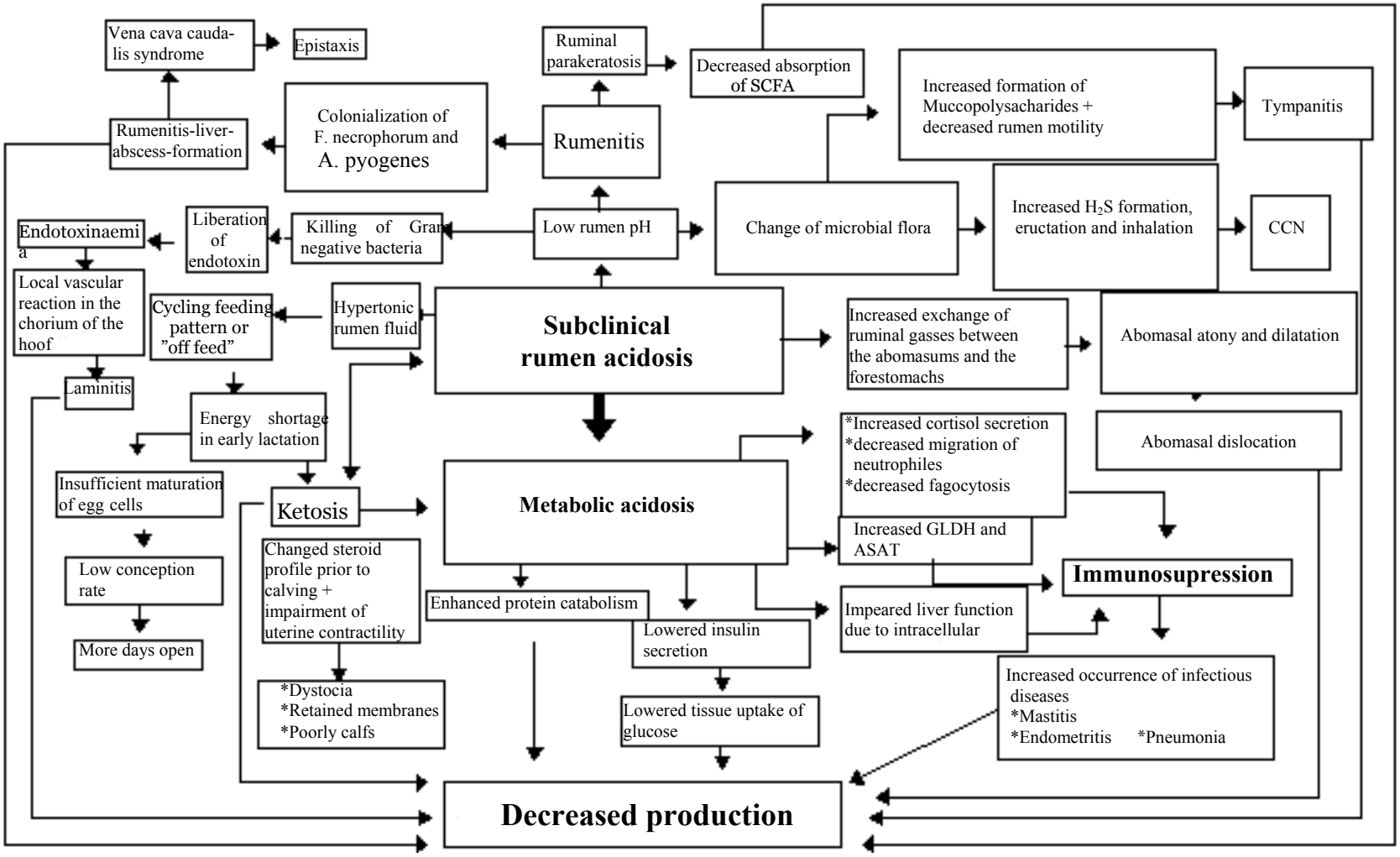

Figure 5. **Etiological importance of subclinical rumen acidosis and metabolic acidosis, respectively, in the pathogenesis of various production diseases. See text for details**

Feed intake and ketosis

The relationship between SRA and ketosis is not yet fully understood. Both conditions have cows in early lactation as the risk group. Whether ketosis or rumen acidosis is the primary condition is unclear. It may work both ways since it can be argued that the resulting reduced intake provoked by rumen acidosis may cause a secondary ketosis based in the immense need for energy in early lactation, or the other way round, that a primary ketosis caused by severe fat mobilisation also result in fluctuating feed intake, inducing rumen acidosis, when the cow regains its appetite. The causal relationship is presumably very complex and may vary from case to case. In the case of SRA the described changes in the feeding pattern may well be linked to changes in the osmolality of the rumen fluid as values considerably higher than 300 mOsm restrict feed intake and reduce the bacterial fermentation of fiber and starch (Carter and Grovum, 1990).

Abomasal displacement and abomasal ulcers

SRA has frequently been mentioned as an etiological factor to abomasal displacement (Svendsen, 1969; Markusfeld, 1987; Olson, 1991). Although a causal relationship has not been proven, increased backwards and forward flow of ruminal derived gasses (SCFA, CO_2 and CH_4) between the abomasum and the forestomachs is believed to result in abomasal atony and dilatation and subsequent dislocation (Svendsen, 1969; Sarashina et al., 1990). The theory is supported by the finding that a low fiber content in the feed ration is the most important single factor in the occurrence of abomasal displacement (Hultgren and Pehrson, 1996; Shaver, 1997; Cameron et al., 1998), and that the establishment of a functional fiber mat in the floating layer is believed to be of importance to a more gradual production and absorption of SCFA in the forestomachs (Olson, 1991).

Occurrence of abomasal ulcers have been linked to intensive management and feeding of highly acidic diets consisting of concentrates and silage (Rebhun, 1995). The pathogenesis is not yet fully understood, but feed induced acidosis has been shown to result in abomasal ulceration in goats (Aslan et al., 1995).

Laminitis

North American researchers assert laminitis as the most significant sequela to SRA, and a prevalence of more than 10% is maintained as being indicative of a subclinical rumen acidosis problem in the herd (Nordlund and Garret, 1994; Garret, 1996b). Numerous investigations prove a connection between starch contents in feed rations and the occurrence of laminitis (Manson and Leaver, 1988; Mortensen, 1993; Wells et al., 1995; Nocek, 1997; Svensson and Bergsten, 1997). The pathogenesis is still uncertain, but it is presumed that vasoactive endotoxin released intraruminally is absorbed into the blood circulation and locally induces a vascular reaction causing vasoconstriction and hypoxaemia resulting in pododermatitis (Andersen and Jarløv, 1990; Boosman, 1990; Andersen, 1994).

Osteoporosis and loosening of the gastrocnemius tendon

In cases of chronic acidosis bone matrix acts as a buffer system as Ca^{++} and P^{+} are released in exchange of H^{+} (Schülke et al., 1975; Cakala, 1981; Fürll et al., 1993). The conditions seem to have particular significance in beef calves on low-structure and concentrate rich rations and may be of consequence in cases of loosening of the gastrocnemic tendon and osteoporosis where the causes of these conditions cannot be related to insufficient supply of D-vitamin and minerals (Lachmann and Seffner, 1979).

Tympanitis

This disease is of particular significance in fattening beef calves, but, as already mentioned tympanitis may also constitute a problem in dairy herds on high concentrate rations. Experience with a high-yielding Canadian dairy herd where less servere cases of tympanitis occur daily, indicates this (Inger Rask, personal communication). The causal relations have not yet been established. The combination of reduced rumen motility caused by a low fiber ration and hence a low rumen pH, excessive production of mucopolysaccharides and release of still unknown macro molecules, from rumen bacteria due to bacterial disintegration, is thought to result in the formation of a stable foam hindering eructation of produced gas (Cheng et al., 1998). Also rumen stasis, as a result of low rumen pH, may allow for the accumulation of free gas (Rebhun, 1995).

Cerebro cortical necrosis (CCN)

CCN has been associated with rumen acidosis. The disease occurs primarily in beef calves aged 6-18 months. Under normal circumstances, thiaminase is not produced in the rumen, but under microbial conditions, as seen in rumen acidosis, a microbial flora is formed, presumably capable of producing thiaminase (Edwin et al., 1968; Morgan 1974). However, recent research show a connection between the occurrence of CCN and the quantity of intra-ruminally produced H_2S (Cummings et al., 1995a; Cummings et al., 1995b). The total sulphur intake as well as conditions which affect the sulphur reducing rumen bacteria are superior factors of significance to this production (Gould, 1998). The total sulphur intake can be affected by a high content of sulphur in drinking water, molasses and feed rations with added ammonium sulphate (Mella et al., 1974; Sager et al., 1990). It has also been established that the production of intra-ruminal H_2S is stimulated by low rumen pH. The further pathogenesis has been explained by the inhalation of eructed H_2S, which is then absorbed systemically via the alveoli before reaching the CNS (Dahme et al., 1983).

Reproduction

In addition to the above significance to calving and possibly to the calf, SRA may indirectly affect fertility. Thus, a cycling feeding pattern during early lactation may, via the subsequent energy shortage, result in insufficient maturation of the first wave of *post partum* ova (Britt, 1995).

Economy

Based on the above mentioned implications it is obvious that SRA is of great economical importance to the dairy industries. Consequences of SRA derive primarily from a reduced milk production (Figure 5). Present knowledge on occurrence, and its role in the aetiology and pathogenesis of other diseases does not enable us to estimate exact figures.

One American report showed that reduced feed intake alone, caused by SRA, led to reduced growth in beef calves, estimated to a loss of DKK 60-80 (10-13 US$) per animal, plus losses from liver abscess formation, which occurred at the15% level (Stock and Britton, 1996). Under Danish conditions the incidence of liver abscesses among fattening bulls may reach 50% in certain herds (Anonymous).

Diagnostics

Clinical signs

The clinical signs of SRA are subtle and often temporally separated in time from the inciting event, thus making diagnosis difficult. Because SRA is a herd problem the clinical signs are related to the herd, thus, the diagnostic exercise should not be based on the examination of a single animal but rather, it should include groups of cows or all cows, as this allows for individual variations. On an individual level, many, if not all of the signs described below, may have several causes besides SRA (Britton and Stock, 1986; Jørgensen et al., 1993a)

Cycling feeding pattern is described as the most consistent symptom of subclinical rumen acidosis (Britton and Stock, 1986). Typically, the picture is one of cyclic feed intake as the cow eat its meal and subsequently refuse further feed intake due to drastic fall in rumen pH and increased osmolality of the rumen fluid. Upon reestablishment of normal rumen conditions, appetite is often regained (Fulton et al., 1979). Such information is useful as regards herds with registered feed intake (automated feed dispensers), whereas in loose stalls, these changes in feeding behaviour will hardly be registered, thereby making them useless as indicators of SRA.

Clinical signs are, as already mentioned, inconsistent or, for natural reasons, non-existent. Nordlund et al. (1995) report on herds with a loose faeces which contain substantial amounts of undigested feed particles. Furthermore, examination of beef cattle showed that rumen motility, pulse and respiratory rate was significantly affected when energy rich feed rations were increased (Leedle et al., 1995). It is uncertain, though, how these parameters can be included in routine surveillance, not least since a considerable variation within and between herds is to be expected.

The intermittent diarrhoea and the presence of undigested particles indicate inadequate digestion and fast passage of feed. Cows in early lactation on a energy rich feed have a considerable drop in rumen pH after each feeding with subsequent light rumen tympany, loose manure, stupor, loss of appetite, decreased rumen motility, decreased rumination time and reduction in yield (Eddy, 1992). Due to the variation in feed intake, these herds will often have several cows in poor body condition. A yearly laminitis incidence of more than 10% and an increase in the laminitis incidence in the herd is often the first and typical sign which should lead to a suspicion of a SRA herd problem (Nordlund et al., 1995). Culling rate and number of inexplicable deaths within the herd may be exceptionally high (Nordlund and Garrett, 1994). In the United States *epistaxis* in cows from SRA herds is well known to the bovine practitioner (Nordlund et al., 1995; Garrett, 1996).

Paraclinical Investigations. Below is a brief description of a potential practice relevant number of diagnostic parameters related to rumen fluid, blood, urine and milk, and considered to be of possible relevance under field conditions.

Rumen fluid parameters

Monitoring of rumen pH, holds a central position in the diagnosis of rumen acidosis. In Denmark, various types of stomach tubes are used to sample rumen fluid. Sampling and evaluation of rumen fluid has, however never become part of routine examinations among veterinary practitioners as it is time consuming. Further, several investigations have shown that the diagnostic value of pH determination on rumen fluid sampled by stomach tube may be questionable as sample pH vary according to intra-ruminal localisation of the stomach tube, saliva contamination and time of sampling in relation to feeding (Hollberg, 1984; Höltershinken et al., 1992). An example elucidating the relationship between sampling location and pH is given in Table 2. These conditional variations add to the difficulties of comparing rumen pH in individual cows and herds. In an attempt to standardize rumen fluid sampling, with the aim of increasing safety in pH determination, Nordlund and Garret (1994) introduced the *rumenocentesis technique* (left-sided flank puncture Figure 6, 7) as an alternative to tube sampling (Table 3).

Figure 6. **Correct position for flanc puncture using the rumenocentesis technique**

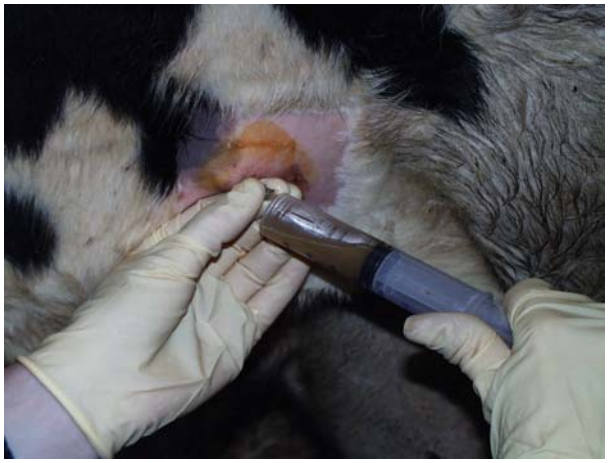

Figure 7. **Sampling of rumen fluid from the caudo-ventral rumen sack using the rumenocentesis technique. Notice the presence of air in the syringe which can be used to clear obstruating feed particles within the needle**

Table 2. **pH in rumen fluid, sampled by either rumenocentesis (rc) or by stomach tube (st) from 7 cow patients submitted to internal medicine**

(rc)	(st)	Difference (rc-st)
6.17	6.80	0.63
6.18	7.09	0.91
5.61	6.63	1.02
6.30	6.98	0.68
5.81	7.12	1.31
7.10	7.89	0.79
6.41	6.80	0.39
$\bar{x}$ =6.23	$\bar{x}$ = 7.04^{+++}	$\bar{x}$ = 0.82

+++ statistically significant difference between mean values (P = 0.0003)

The method was based on that described by Hollberg (1984). Garrett et al. (1999) applied an evaluation model in which a herd, or a certain group of cows, is defined as having SRA when a rumen pH below 5.5 is found in more than 4 (30%) of the cows from a sub population of 12 cows in the risk group (early or mid-lactation), whereas the finding of two to four cows with ruminal pH < 5.5 is considered borderline. If none or one of the 12 cows tested have ruminal pH < 5.5 the group is definitively classified as negative for SRA. This model has its limitations, since it applies to herds with either high (> 30 %) or low (< 15 %) prevalence of low ruminal pH.

Table 3. **Material and procedure of the rumenocentesis technique (flanc puncture) in cattle. The method is modified in accordance with Nordlund & Garrett (1994).**

Material	12-14 gauge needle (Ø 2 mm) . Length 14-16 cm.. Razor, ethanol, iodine, cotton, lidocaine 2%, 30 ml disposable syringe, 5 ml disposable syringe, sterile isotonic saline, pH-metre (e.g. Twin pH meter, Spectrum Technologies Inc. USA).
1	Select an appropiate number of individuals, preferrable 5-6 cows in early lactation (0 - 20 days in milk (DIM)) and mid lactation (45 - 120 DIM) respectively. Avoid cows with a bad temper.
2	Time of sampling is dependend on feeding strategy. In component fed herds sampling should be done within 2-4 hours after concentrate feeding. If a TMR-ration is fed sampling should be done within 4-6 hours *post prandial.*
3	Prepare, like for surgery, an area of 10x10 cm in the left flanc, 20 cm caudal to the last costae and on the level of the top of the knee joint (Figure 6).
4	Prepare the area with iodine and apply a local anastetic (5 ml 2% lidocaine cum Nordadrenaline). Due to withdrawel time for milk any use of local anastetic should be finished at least 7 hours before milking time.
5	Repeat desinfection of the area with ethanol and subsequent iodine.
6	Instruct the farmer to restraint the cow by means of a tail grip and introduce the needle into the rumen in a contineous movement. Spontaneous outflow of rumen fluid ensures correct placement.but must not always be expected.
7	Fix the 30 ml syringe, which should be filled with 15 ml air, to the needle and aspirate gentle (Figure 7). If no rumen fluid is obtained inject an appropiate amount of air to clear the needle of obstruating particles. Repeat gentle aspiration. If nessecary, repeat this proces until 5-10 ml of rumen fluid is sampled.
8	Dismantle the 30 ml syringe and fix a 5 ml syringe, filled with isotonic saline, to the needle. During slow withdrawel of the needle from its intraruminal position saline is contineously infused.
9	The area of puncturement is swept with iodine.
10	pH of rumen samples are measured imeadiately after sampling using a pH-metre. Duplicates are measured for each sample.

Table 4. **Most important changes in rumen fluid from cows and their significans (modified after Rosenberger)**

Colour	Gray-brown or green[3]	Dark brown/green	Milky/green	Slightly milky/brown
Odor	aromatic	slight ammonia odor	sticky/sour	sour
Viscosity	slightly viscous	variable	watery	slightly viscous
Flotation/sedimentation	4-8 min.	variable	no/fast	no/fast
PH	5.5-6.8	6.8-8.5	5.2-3.8	6.2-5.3
Methylene blue reduction	<3 min.	?	>5 min.	<3 min.
Glucose fermentation test	1-2 ml pr. hour	↓	↓	n/8
Number of infusoria[1]	+++	+/+++	n/↑-	+++
Microbial composition[2]	Gram neg. > Gram pos.	Gram neg. > Gram pos.	Gram pos. > Gram neg.	Gram neg. > Gram pos.[4]
Diagnosis	Active ruminal fermentation	Rumen alkalosis	Acute rumen acidosis[5]	Subclinical rumen acidosis

[1] Number of infusoria: - non, + few, ++ some, +++ plenty
[2] Mikrobial composition: Gram-negative or Gram-positive bacteria dominates.
[3] Depending on season (winter or pasture feeding).
[4] Absolute increase in Gram-positive bakteria.
[5] Intraruminal lactate concentration > 30mg/100 ml (3.3 mmol/l)

n = normal
↓ = decreased or prolonged
↑ = increased or shortened

Furthermore, under Danish conditions, the average herd size often collides with the number of cows needed for sampling (Table 3). Thus, the rumenocentesis technique may not be ideal for diagnosing SRA in low and middle sized herds. However, the present authors experience with the method has revealed that complications rarely occur, and if so, they generally occur as subcutaneous haematomas at the point of puncture. Yet, it must be expected that routine use of rumenocentesis, as compared to the above uncertainty in the interpretation of results, will provoke a discussion as to whether it is ethically justifiable to apply a method which is invasive, besides its limitations to diagnose a disease known to be brought upon the animal by certain production conditions. Even in the United States, the method is not yet being applied routinely.

Besides pH, Table 4 summarises the most commonly applied analyses for rumen fluid, and their interpretation, including the statement that the protozoon population is not affected at pH-values between 6.2-5.3. Some research indicates that partial defaunation may be observed in cases of SRA (Jørgensen et al., 1993b) and that great individual differences exist (Franzolin and Dehority, 1996).

Milk parameters.

Fat percentage. The fat percentage of the milk is influenced by several factors, including lactational state, breed and composition of feed rations (Spohr et al., 1992; Grummer, 1991). It has been known for long that a positively linear relationship between acetate:propionate in the rumen fluid and the fat percentage of the milk exists (Kaufmann, 1976). Sutton et al. (1987) expanded the above relation to include acetate + butyrate:propionate. Further, it has been documented that the fat percentage will drop drastically within 24 hours after induction of rumen acidosis (Nicpon and Hejlasz, 1985). This is believed to be caused by the proportional drop in the acetate:propionate ratio. In addition, a positive correlation exists between butyrate and the fat percentage of the milk (Sutton, 1988). Furthermore, it has been proved that the concentration of propionate alone can affect milk fat syntesis by means of three mechanisms: 1) an increase of blood glucose which will directly lower the fat percentage; 2) stimulation of the insulin secretion with subsequent decrease in tissue lipolysis leading to low production of Very Low Density Proteins (VLDP) in the liver which are of significance in milk fat synthesis; 3) increased propionate production leading to low intra-ruminal production of Vitamin B12 which causes a reduced conversion of propionate to succinyl-CoA with subsequent increase of the concentration of the intermediary metabolite methylmalonate (MMA) in the blood. MMA inhibits the fat synthesis in various tissues including the mammary tissue (van Soest, 1994). Thus, the fat percentage in the milk on an individual level seems to be a good indicator of the fermentation conditions in the rumen. To improve its diagnostic value, milk fat tests should be performed frequently (once a week) (Erdman, 1993). If carried out monthly, as in Denmark, brief periods of low fat percentages in the milk may remain unnoticed. On a group or herd level lactation curves may be useful as they can reveal a sudden drop of 1-2% in the average fat percentage of cows in mid lactation, which may occur during sudden changes in feed offered such as a insufficient fiber supply. For the purpose of interpretation of the fat percentage in cows in early lactation, lactation curves are hardly suitable. The initial low fat percentage generally registered in early lactation is influenced by several factors, including the general level of butterfat in the herd and the degree of fat mobilisation in the *post partum* cow. The depth of the initial drop in milk fat percentage between the first and second milk fat test *post partum* is thus hardly suitable for the evaluation of the fermentation pattern in the rumen.

Other biochemical markers in the milk have been linked to SRA. Potentially important markers are listed in Table 5. Inadequate experience in the use of these parameters under commercial conditions does however exclude them as recommendable monitoring tools for the time being.

Table 5. **Biological parameters in milk of potential importance to monitoring the occurrence of SRA in cattle**

Marker	Normal	"Pathogenesis"	Changes due to SRA
Fat-proteine- kvotient (FPK)	1 - 1,5	Decreased intraruminally acetate:propionate-ratio and subsequent decrease in milk fat content	< 1
Soxleth-Henkel-figure (SH)	6,4 - 6,8	Elimination of H^+ via the udder	> 8,0
Laktose	4,4 - 5,2 %	Increased intraruminal propionate formation → increased blood glucose	↑
Cl, Na, K	Cl: 25 - 31 mmol/l Na: 20 - 26 mmol/l K: 30 - 40 mmol/l	?	Cl ↑ Na ↑ K ↓
Milk-urea-nitrogen (MUN)	3,0 - 5,0 mmol/l	Energy (carbohy-drate) content of ration in favour of proteine → reduced ruminal NH_3 - formation → reduced hepatic urea formation	< 3,0 mmol/l

Blood parameters

Contrary to conditions in the United States, Great Britain and Germany (Rossow et al., 1990; Scholz, 1990; Nelson, 1996; Ruegg, 1996; Ward et al., 1996), the integration of metabolic profiles as part of an observation of diseases in dairy herds has not yet gained access in the Danish health advisory system. Until now, blood gas parameters have not been available for on-farm use. The recent launch of a transportable acid-base laboratory (IRMA7, Blood Analysis System, Diametrics Medical, Inc.7, St. Paul, MN, USA) makes it possible to include blood gas parameters as well as various electrolytes in the monitoring of dairy cows.

However Lachmann and Siebert (1980) found that the blood gas parameters were not notably affected in cases of chronic, metabolic acidosis, whereas Fürll (1994) emphasised the diagnostic value of acidosis induced hypercalcaemia and hyperphosphataemia. Aslan et al. (1995) demonstrated a positive blood glutaraldehyde coagulation test (Sandholm, 1974) presumably caused by rumenitis in clinical rumen acidosis induced in goats. Whether this test could be of any significance in cases of SRA has not yet been examined.

Urine parameters

The relatively small lung capacity of ruminants means that this organ only plays a minor part in the acid-base regulation in the organism. Acid elimination via the kidneys, on the other hand, is more important. The collection of urine samples from individual cows may cause problems to inexperienced personnel. In the author's experience efficient manual stimulation of the perineal area frequently, but not always, provoke spontaneous urination. A positive connection has been established between rumen pH and urine pH (Roby et al., 1985; Fürll, 1994) but it should be borne in mind that aciduria can be caused by several conditions (Markusfeld, 1987). Calculation of the renal Net-Acid-Base-Excretion (NABE) based on urine titration is claimed to be more accurate than a pH determination as acidotic conditions cause excretion of increased amounts of inorganic phosphate into the urine, acting as a buffer (Kutas, 1965; Lachmann and Seffner, 1979; Fürll, 1994).

Prophylaxis

Feeding and management

Subclinical rumen acidosis is so closely linked to feeding conditions that correction of feed rations and/or feed management is essential in solving the problem. A revision of the feeding schedule will in itself only rarely reveal any gravitating deficiencies. It is more important to focus on whether the quality of the feed corresponds to the one applied as basis for calculation of the feeding schedule. In this connection, a feeding control system applied in Denmark, the so-called one-day feeding control system, may be a useful tool in confirming or disconfirming whether feed intake and ration formulation are in accordance with the computer assisted feeding schedule. Table 6 lists frequent feeding and management problems as they occur in relationship to SRA and suggests how to solve these problems.

Table 6. **Commonly occurring feeding and management deficiencies resulting in subclinical rumen acidosis and suggested corrections**

Period	Problem	Correction	Effect
Transition period	Steaming up period shorter than 4 weeks	Start steaming up at least 2 - 3 week prior to calving	Optimal proliferation of rumen mucosa
	To intensive steaming up	Maximum increase of 0.5 kg concentrate/day	Moderate SCFA and lactate production
Lactation period	Irregularities in feed supply	Ensure a homogeneous ration and regular allocation	Stable balance between lactogenic and laktolytic bacteria.
	Sudden change in ration composition	Changes in ration composition should be carried out over a period of 14 days	Optimal adaptation of rumen bacteria
	High water content in ration	Optimal time of harvest Optimal ration composition	Counteracts low saliva production due to missing stimulation of ruminal mucosa
	Allocation of more than 3 kg of concentrate/ feeding	Increased feeding frequency TMR-feeding	Less fluctuation of rumen pH
	Silage with low pH	Optimize silage fermentation Temporary addition of buffers to the ration	Stabilization of rumen pH

Additives and SRA

In North American feed lots, chemical buffers are regularly added to feed rations (Erdman, 1991; Hutjens, 1988). In dairy herds, buffers are also added in cases where the fibre content in the feed rations is too low (Erdman, 1988). Documentation exists showing that adding 150 grams of $NaHCO_3$ to the lactation feed per day has a positive effect on the milk yield (Downer and Cummings, 1985). Similarly, a positive effect has been demonstrated on feed intake and milk fat percentage (Erdman, 1988). Table 7 lists recommended doses of various buffers. The ideal buffer should be water-soluble and have a pKa value close to the optimal physiological pH of the rumen fluid. $NaHCO_3$ (pKa = 6.25) meets these requirements and is the most frequently applied buffer, whereas the other buffers mentioned have only limited or no buffer effect but they do have an alkalinsing or neutralising effect. Normally, a single buffer is given but combinations of several buffers are possible with a documented positive influence on milk yield, fat percentage and dry matter intake (Hutjens, 1991).

Table 7. **Recommended doses of various buffers added to the feed rations of lactating cows (Hutjens, 1991)**

Product	Amount (g/day)
Sodium bicarbonate	110 - 225
Sodium sesquicarbonate	110 - 225
Magnesium oxide	50 - 90
Sodium bentonite	110 - 454
Calcium carbonate	115 - 180
Potassium carbonate	270 - 410

In our opinion, buffers should only be given temporarily

or diagnostically, since the rutine use may tempt farmers to additionally raise the carbohydrate content of the ration instead of feeding the rumen according to its natural demands. It has been suggested that yeast cultures be added to feed rations. Documented research shows varying effects of the addition of yeast (Williams et al., 1991; Aslan et al., 1995; Höltershinken et al., 1997). It is further suggested that various pro-biotics (Hutjens, 1991) and growth stimulants (Richardson et al., 1976; Shell et al., 1983; Burrin and Britton, 1986) are added but this is unlikely to be applied under Danish conditions. Genetic manipulation of lactolytical bacteria is a relatively new idea with the aim of increasing the lactate conversion capacity and acid resistance of the bacteria (Martin and Dean, 1989) which has not yet been documented.

Perspectives. The aetiology of SRA and its occurrence in early lactation places subclinical rumen acidosis in the borderland between traditional veterinary science and nutritional science. Accordingly, the success of a directed effort against SRA depends on cooperation between veterinary and nutritionist researchers. The complex aetiology and pathogenesis, together with the subclinical course of the disease, complicate demarcation, diagnosis and prophylaxis of the disease. Among the diagnostic parameters described in the present paper, none can stand alone and unambiguously be applied to determine subclinical rumen acidosis on a herd level. Routine monitoring of the acidity of the urine is, in the author's experience, the most efficient diagnostic tool. Combined with a thorough knowledge of the feeding situation of the herd, systematic routine health recording and occasional determination of rumen pH in rumenocentesis derived samples are all essential. Important aspects of its pathogenesis and aetiology, and their relationship to other significant production diseases, are unclear. Clarification of the importance of the fluctuation pattern of the rumen pH is desirable. At present, it is uncertain whether the lowest pH-value, or the time span of the period during which the rumen pH is below a threshold value, is decisive to the significance of subclinical rumen acidosis. Similarly, it would be useful to know the significance of acute, short or long, long lived but low-grade fluctuations in intra ruminal lactic acid concentrations to health and production. Such research would require the monitoring of the rumen environment continuously over considerable periods of time. Development of research methods suitable for this should be encouraged. Similarly, diagnostic tools for on-farm use, their application and interpretation should be further developed and evaluated.

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