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HYPOMAGNESAEMIA AND HYPOMAGNESAE-MIC TETANY INDUCED IN LACTATING COWS BY CHANGING THE DIET

By

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Ever since *Sjollema* in 1930 showed that cows suffering from grass tetany were in a hypomagnesaemic state, the question of magnesium deficiency and its causation has been the main problem in tetany research work. Although analytical investigations have shown that no magnesium deficiency is to be observed in tetany prone pasture grass, nearly all research workers agree with the theory that the magnesium absorption from this pasture is insufficient. The motivation is a lower Mg availability in pasture grass than in usual stall fodder. Feeding experiments performed by *Kemp et al.* (1961) have given support to this theory. They fed two groups of cows, one with hay, concentrates and beets in the composition generally used for stall feeding and the other with early grown pasture grass, and found a higher utilization of Mg in the first group. They estimated the amount of available Mg required for normal Mg balance to be at least 2.5 g per day for maintenance plus 0.12 g for each litre of milk. However, an exact amount of Mg needed in the diet to cover this requirement is difficult to estimate. *Blaxter & McGill* (1956) suggested that 5.4 g Mg is needed for maintenance and 1.84 g being sufficient to cover each gallon of milk. Bearing in mind, however, the interdependence between fodder composition and Mg availability, no exact figures can be given.

Bartlett et al. (1954) fed cows a diet containing 4.7 g Mg per day without getting significant hypomagnesaemia, and *Rook* (1961, 1963) demonstrated normal serum Mg levels in cows as

long as the daily Mg supply in the experimental diets amounted to 2 to 3 g. Reduced serum Mg levels were not observed until the supply was reduced to 1.5 g/day. Serum Mg values as low as 0.8 mg per cent were obtained with diets containing 0.8 g Mg daily. Tetanic convulsions were observed only once. In tetany-prone pasture grass the serum Mg-depressing effect is obvious, despite the fact that the content of Mg is very seldom found to be less than 0.11 per cent of dried matter, equivalent to an intake of about 16 g Mg a day. Consequently the Mg availability must be extremely low in such grass. How this comes about has been discussed for more than 30 years, but no exact conclusion has hitherto been drawn. At present, one simply has to accept the hypothesis that the first step in the development of tetany is the hypomagnesaemic condition produced by the exceptionally low availability of Mg in early grown, highly dressed pasture grass. The next step, however, — the outbreak of tetanic convulsions — presupposes a decrease in the serum Ca level as well, and the problem is whether this happens as an inevitable consequence of sudden reductions in the serum Mg level, or whether some other active compound capable of releasing the reaction is present at the same time.

In the present work the idea has been to expose high-lactating cows to sudden reductions in the dietary Mg from a normal level of about 25 g per day to a very low intake, in agreement with what possibly happens in early spring when cows are let out directly from the stall to a new-grown, highly dressed pasture. As long as reactions were observed by analyses of the serum, or clinically, the feeding was continued unchanged until possible occurrence of tetany. In those cases, however, where the falling tendency of serum Mg stopped, and the cows remained apparently healthy in spite of a low serum Mg level, oral addition of supplementary substances, possibly able to increase the tetany-prone effect of the diet, had to be given. The compounds selected for this purpose were Na_2HPO_4 and Na_2SO_4 . They were tried, first and foremost because of the reduced serum Ca level observed by administering these compounds to healthy ruminants (*Ender et al.* 1957; *Dishington* 1965), but also because of the relatively high contents of alkalis, phosphorus, and sulphur found in pasture grass in which the tetany-proneness has been markedly increased by overdressings with $(\text{NH}_4)_2\text{SO}_4$ (*Havre & Dishington* 1962).

MATERIAL AND METHODS

The experiments were performed with 15 cows of the NRF breed, stalled at The Veterinary Research Station, Wöyen. They had a live weight of 405 to 600 kg, and the milk yields varied from 12 to 21 kg per day. None of the cows had previously shown tetanic symptoms. The preliminary stall feeding consisted of hay, alkali-treated straw, fodder beets, and concentrates in amounts covering all requirements. The ration supplied the cows with 20 to 25 g Mg per day. To make the experimental diet poor in Mg, most of the Mg-rich compounds were replaced by wheat flour, cellulose, and whale-meat meal. Shortages on minerals and vitamins were replaced by supplements. As far as could be calculated, the nutritive value was not changed except for the reduced Mg content. Cows weighing 500 kg, with a milk yield of 15 kg per day, received daily 15 kg alkali-treated straw, 5 kg fodder beets, 2.5 kg wheat flour, 2.5 kg fodder cellulose, 1.5 kg whale-meat meal, and diluted mineral acids in amounts sufficient to neutralize the excess of alkali. The vitamin requirement was covered by A+D vitamin supplement, and a mineral mixture of CaCO_3 and Na_2HPO_4 was given to obtain a normal dietary Ca/P ratio. The daily ration was found to contain 9.5 f.u., 1526 g of digestible crude protein, 55 g Ca, 44 g P, and 4.8 g Mg (2.55 g Mg for maintenance + 0.15 g for each kg of milk). To minimize the risk of reduced appetite, cellulose — a rather uncommon foodstuff today — was given in advance in gradually increasing amounts; the decisive change to the Mg-deficient fodder was, however, performed abruptly. All the cows were under daily veterinary control, and blood samples for analysis were drawn at least once every day.

The experiments extended over 2 years. Seven of the 15 cows were used as test animals only once, the other 8 were used several times, interrupted by shorter or longer periods of rest. The fodder supply was calculated in proportion to the milk yield, and the appetite was under strict control. During the first days after the fodder had been changed, the cows usually refused some food, especially the cellulose. Nevertheless, there was no marked deficit in f.u. and protein intake during these days. Two tests, however, had to be terminated on the 6th day of feeding because of sudden and complete loss of appetite throughout a whole day.

Serum Mg levels showed falling tendencies in all animals. In cases where the cows tended to accommodate themselves to

the reduced magnesium supply with a low serum Mg value, $\leq 10 \mu\text{g/ml}$, the presumed trigger effect of Na_2HPO_4 or Na_2SO_4 was tried. Relatively high oral doses of these compounds — viz. 125 g — were given twice with 1 hour's interval without changing the fodder.

RESULTS

A survey of all experiments is presented in Tables 1 and 2. Section 1 deals with 15 experiments carried out with cows that had not been test animals before, and are demonstrated in Table 1. A review of the 20 repeated experiments, section 2, is presented in Table 2.

Comments to Table 1

All 15 cows in the first experiment reacted acutely to the Mg-deficient feeding with decreases in the serum Mg level. The decreasing rate varied; but, except for 1 cow, whose serum Mg stopped dropping at a level of $13 \mu\text{g/ml}$, they all reached $10 \mu\text{g/ml}$ or lower values within 2 to 18 days. By continued feeding only the two cows *Songfryd* and *Anne* got tetany. *Songfryd* was fed the experimental diet for 4 days when she collapsed in tetany. The serum Mg value fell exceptionally rapidly and a decreasing tendency was observed in the serum Ca level already within 2 days. Her appetite was good until she collapsed. *Anne*, a cow in her first lactation with high yields, was used as a test animal as early as one month after parturition. She had been exposed to the experimental diet for 7 days when she contracted tetany. To what extent the condition could be attributed to the Mg-deficient fodder *per se* is, however, difficult to judge, because the cow developed a total loss of appetite and was taken out of the experiment the day before she collapsed.

Holdis was in the same experimental group as *Anne*. For some unknown reason she also suddenly refused to eat and was removed from the experiment the following day. She, however, began to eat again as soon as the fodder was changed to normal, and remained healthy.

The other 12 cows had a good appetite and were clinically healthy despite the pronounced hypomagnesaemia as long as the experimental fodder was given.

In 10 cases oral Na_2HPO_4 supplementation was tried: *Sonni*, *Seiergod*, *Krone*, *Åse*, and *Ragnlin* reacted to the dosing with

Table 1. Section 1. Fifteen experiments with cows never before exposed to the magnesium-deficient diet.

Cow	Immediately before the Mg-deficient diet was given				The day the serum Mg-declining tendency seemed to weaken				Immediately before the "trigger" supply				"Trigger" supply		Highest serum P value after the supply		Observations corresponding to the lowest serum Mg value recorded after the supply				
	serum mineral values mg/100 ml		date	serum mineral values mg/100 ml		date	serum mineral values mg/100 ml		date	serum mineral values mg/100 ml		Ca	Mg	P	Ca	Mg	P	serum mineral values mg/100 ml	Ca	Mg	P
	Ca	Mg		P	Ca		Mg	P		Ca	Mg										
Sonni	10.1	1.9	4.9	10/2	10.0	0.9	5.3	20/2	10.3	0.9	5.4						7.7	6.9	0.3	6.4	Tetany
Seiergod	10.4	1.9	5.8	17/2	10.0	0.8	5.8	20/2	9.4	0.8	6.6						9.6	7.2	0.4	8.7	Tetany
Snuppa	9.7	2.3	4.6	9/2	9.5	0.7	4.0	14/2	8.7	0.7	4.9						5.4	7.1	0.6	2.8	Subtetany
Krone	9.9	2.3	4.8	9/2	10.5	0.9	5.6	13/2	10.6	0.8	7.9						10.2	7.4	0.4	9.0	Tetany
Åse	10.0	2.6	3.5	16/4	9.6	0.7	5.5	19/4	7.8	0.8	4.5						7.8	7.2	0.9	7.8	Tetany
Stjerne	10.1	1.9	3.6	25/4	9.9	0.9	4.6	4/5	9.6	0.9	6.4						7.0	9.2	0.6	5.2	Healthy
Holdis*)	6/12	10.9	2.6	5.3	11/12	8.4	1.0	7.4	12/12	9.9	0.8	6.3									Healthy
Anne*)	6/12	11.2	2.8	4.9	12/12	10.0	0.9	6.8	13/12	6.7	0.6	2.0									Tetany
Rødhette	6/12	10.0	2.3	3.3	12/12	12.5	1.0	3.8	22/12	10.6	1.0	5.5									Healthy
Linda	6/12	10.1	2.8	5.2	18/12	11.2	1.3	6.0	22/12	10.6	1.3	5.5									Healthy
Ragnin	8/1	10.4	2.0	4.6	12/1	9.0	0.8	4.1	18/1	8.8	0.8	4.1					7.4	6.8	0.6	5.6	Tetany
Songfryd	30/4	10.1	1.6	2.9	2/5	7.8	0.7	3.3	4/5	6.4	0.3	3.1									Tetany
Rengås	30/4	11.0	2.7	4.4	8/5	9.5	1.0	5.8	22/5	10.8	1.3	6.7					5.9	9.8	1.4	5.3	Healthy
Sibylla	30/4	10.5	2.3	4.1	5/5	9.6	1.0	6.7	29/5	11.0	0.9	6.6					6.6	9.6	0.8	6.5	Healthy
Brita	30/4	10.4	2.0	3.1	5/5	8.8	0.9	6.9	15/5	10.2	1.3	4.1					10.7	9.2	1.3	7.8	Healthy

*) Experiment terminated because of lack of appetite.

spontaneous decreases in serum Ca followed by tetanic convulsions. They had been hypomagnesaemic for 10, 3, 4, 3, and 6 days, respectively, when the supplement was given. *Krone* was the most seriously affected and died despite treatments with calcium gluconate and magnesium chloride. The others, however, recovered after treatment and transfer to normal feeding supplemented with MgO. *Snuppa*, who got Na_2HPO_4 on the 5th day of hypomagnesaemia, reacted with clinical symptoms of subtetany. *Stjerne* and *Sibylla* received the "trigger" supplies on the 9th and 24th days, respectively, of hypomagnesaemia, but remained clinically unaffected even though some further decreases in the serum Mg levels occurred. *Rengås* and *Brita* did not react to the supplies at all. These 2 cows had, to some extent, been able to raise their serum Mg levels again during the feeding period, and this increasing tendency before dosing might perhaps have been the reason that the "trigger" supply had no influence. Individual susceptibility might, however, also play an important part in these experiments.

Comments to Table 2

In all 20 repeated experiments the serum Mg level dropped to hypomagnesaemic values, about 10 $\mu\text{g}/\text{ml}$, within 3 to 18 days of Mg deficient feeding. Prolonged feeding resulted in 3 cases of tetany: *Sonni* collapsed in her third experiment and *Ase* in her first and third experiment. In 17 cases the cows remained healthy on a very low serum Mg level. By abrupt oral administration of Na_2HPO_4 , Na_2SO_4 , or both to the hypomagnesaemic cows in 15 cases, 11 distinct reactions were observed. Convulsions appeared in 4 cases: *Sonni* collapsed after supplementation in her 5th experiment, *Ase* in her third, and *Holdis* in her second and third experiment. Subtetanic convulsions were observed in 3 cases: the second experiment with *Sonni* and both experiments carried out with *Seiergod*. Slighter reaction with borderline symptoms were observed in 4 cases: the first experiment with *Sonni*, the first with *Snuppa*, the first with *Holdis*, and the only repeated experiment with *Linda*. In 4 cases the supply had no influence.

Conclusion

Thirty-five feeding experiments were carried out to demonstrate the reaction of relatively high lactating cows to sudden reductions in the daily dietary Mg supply. The reduction was

Table 2. Section 2. Twenty experiments repeated with 8 cows in section 1.

Cow	First day of normal feeding	Immediately before the Mg-deficient diet was given			The day the serum Mg-declining tendency seemed to weaken			Immediately before the "trigger" supply			"Trigger" supply	Highest serum P value after the supply	Observations corresponding to the lowest serum Mg value recorded after the supply						
		date	Ca	Mg	P	date	Ca	Mg	P	date			Ca	Mg	P	serum mineral values mg/100 ml	Ca	Mg	P
Sonni	8/3	10/4	10.0	2.5	4.3	16/4	10.1	1.05	4.3	19/4	10.1	1.1	4.5	Na ₂ HPO ₄	6.7	7.5	0.75	6.4	Borderline
Sonni	1/5	16/5	10.8	1.8	3.9	21/5	10.1	1.0	3.0	30/5	6.1	0.6	6.1	Na ₂ HPO ₄ + Na ₂ SO ₄	9.5	6.6	0.45	9.5	Subtetany
Sonni	5/6	8/1	10.5	2.2	—	11/1	9.2	0.9	4.7	13/1	6.8	0.45	4.7	none	5.7	10.2	1.1	4.9	Tetany
Sonni	15/1	20/2	9.5	2.7	4.7	8/3	10.8	1.0	6.0	21/3	10.8	1.2	4.1	Na ₂ HPO ₄	6.4	7.1	0.5	5.8	Healthy
Sonni	25/3	30/4	10.4	2.0	3.6	3/5	10.1	0.9	4.5	8/5	9.3	0.7	5.7	Na ₂ HPO ₄	8.5	8.7	0.7	5.5	Tetany
Seiergod	21/2	10/4	10.6	2.5	5.6	18/4	9.2	1.05	6.8	19/4	10.1	1.2	6.2	Na ₂ HPO ₄	8.8	6.7	0.6	8.4	Subtetany
Seiergod	28/4	12/5	10.5	2.2	5.3	27/5	5.8	1.1	4.6	30/5	9.2	1.2	7.3	Na ₂ HPO ₄	8.8	8.8	0.75	5.6	Subtetany
Snuppa	25/2	10/4	9.6	2.6	4.1	17/4	9.1	1.05	4.9	19/4	10.6	1.2	6.0	Na ₂ HPO ₄	6.9	8.8	0.75	5.6	Borderline
Snuppa	2/5	12/5	9.6	3.0	4.2	27/5	6.2	0.9	4.0	30/5	7.3	0.75	4.6	Na ₂ SO ₄	6.2	7.2	0.9	6.2	Healthy
Åse	20/4	12/5	9.8	2.2	4.1	21/5	10.1	1.1	4.3	27/5	6.6	0.4	4.7	none	7.3	6.3	0.3	4.6	Tetany
Åse	28/5	20/2	9.4	2.7	5.6	10/3	9.3	0.8	4.3	21/3	8.9	0.5	4.3	Na ₂ HPO ₄	7.3	6.3	0.3	4.6	Tetany
Åse	22/3	30/4	9.8	2.4	3.7	5/5	9.5	1.05	6.1	9/5	7.5	0.6	6.4	none	—	—	—	—	Tetany
Sjerne	10/5	6/12	10.8	2.6	3.6	15/12	10.6	1.0	5.5	22/12	10.7	1.4	5.4	none	—	—	—	—	Healthy
Sjerne	23/12	5/1	9.5	1.6	10.6	12/1	9.8	1.2	5.1	25/1	10.6	1.7	6.1	Na ₂ HPO ₄	8.2	9.6	1.55	7.4	Healthy
Holdis	13/12	5/1	9.5	2.1	5.8	13/1	10.2	1.0	5.1	25/1	9.0	0.5	5.3	Na ₂ HPO ₄	9.0	8.1	0.5	7.5	Borderline
Holdis	28/1	20/2	9.0	2.6	6.5	2/3	9.9	1.05	5.0	21/3	10.1	0.5	6.5	Na ₂ HPO ₄	9.1	6.6	0.4	5.7	Tetany
Holdis	22/3	30/4	10.3	2.2	3.1	5/5	10.6	0.9	5.4	1/6	10.4	0.7	—	Na ₂ HPO ₄	—	—	—	—	Tetany
Linda	23/12	8/1	9.7	1.8	5.6	13/1	9.3	0.8	5.7	23/1	8.4	0.6	7.0	Na ₂ HPO ₄	9.9	7.5	0.7	9.4	Borderline
Ragnlin	19/1	20/2	9.1	2.6	4.8	1/3	10.5	1.1	3.9	21/3	10.7	1.3	3.9	Na ₂ HPO ₄	4.8	9.5	1.5	3.5	Healthy
Ragnlin	26/3	30/4	10.9	2.4	3.2	7/5	10.4	1.1	4.9	1/6	10.3	1.1	4.7	none	—	—	—	—	Healthy

made from a normal level of 25 g Mg per day to 4.3 to 5.6 g Mg per day proportional to the milk yield. This reduction produced pronounced hypomagnesaemic serum Mg values within 2 to 18 days in all experiments. Continued Mg-deficient feeding resulted in tetany in 5 cases, whereas 30 remained clinically healthy in spite of a marked hypomagnesaemia. Oral doses of Na_2HPO_4 , Na_2SO_4 or both given to 25 hypomagnesaemic cows resulted in 9 cases of tetany, 4 cases of subtetany, and 4 cases of slighter borderline symptoms. In 8 cases the cows remained unaffected.

DISCUSSION

The instantaneous effect of an abrupt reduced dietary Mg supply upon the serum Mg level obtained in these experiments indicates clearly that the cows had not been able to deposit available magnesium, even though the preliminary feeding was rich in this mineral. Very likely this inability is a general phenomenon in ruminants, and has to be looked upon as the main reason why they cannot obtain a normal Mg metabolism, even for shorter periods, without having access to an even flow of magnesium. To state the exact minimum of Mg required in the diet to avoid hypomagnesaemia seems to be impossible. The amount of dietary Mg available for absorption is not dependent merely upon the exact content of Mg in the diet, but just as much upon the dietary composition in other respects. The diet described by *Rook* (1961, 1963) did not influence the serum Mg level as long as the Mg content was above 1.5 g per day, and the experiments carried out by *Allcroft & Parr* (*Bartlett et al.* 1954) showed that a total support of 4 to 7 g Mg per day in the diet was sufficient to maintain normal serum Mg values. The diet used in the experiments described in this paper had, however, a marked serum Mg-reducing influence with a dietary Mg content calculated to 4.3 to 5.6 g per day. As to the severity of hypomagnesaemia and risk of tetany, however, the individual disposition seems to play an important part.

The experiments described were made as a part of the study of tetany in cattle, and the abrupt reduction in dietary Mg was performed to duplicate as closely as possible what seems to happen when the cows are exposed to tetany-prone pastures in the spring. The 5 cases of tetany provoked by no other influence than the Mg-deficient diet seem to indicate that the sudden reduction in Mg availability that occurs when cows are fed a

new-grown pasture could perhaps, under unfavourable conditions, be a releasing factor per se. The 30 other cases, in which the cows remained apparently healthy despite typical hypomagnesaemic serum values, prove, however, that cows are generally able to withstand relatively severe decreases in available dietary Mg without being attacked by tetany as long as other factors are not involved. Nevertheless, the disposition to illness is undoubtedly markedly increased when the serum Mg level is low, and it seems obvious that a major step towards tetany is made. In this situation avoidance of the disease merely depends upon the maintenance of an undisturbed serum Ca balance. According to analytical examinations of $(\text{NH}_4)_2\text{SO}_4$ dressed tetany-prone grass and dosing experiments carried out with healthy ruminants, we have in earlier papers pointed to alkalis, phosphates, and sulphates as the dietary compounds most likely to disturb this balance. In the present experiments the same compounds are given as supplements to already hypomagnesaemic cows. The clinical reactions observed in 17 out of 25 experiments made it evident that these compounds, when present in excess as in the $(\text{NH}_4)_2\text{SO}_4$ dressed pasture grass, undoubtedly have to be taken into account as factors contributing to tetany attacks.

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SUMMARY

Thirty-five experiments are demonstrated in which high-lactating cows were exposed to abrupt changes from a normal dietary Mg supply to an extremely Mg-poor diet. The preliminary diet was adequate, supplying the cows with about 25 g Mg per day. The experimental feeding was individually calculated, containing 2.55 Mg for maintenance and 0.15 g for each kg of milk. Daily serum mineral analyses showed a pronounced hypomagnesaemia in all 35 experiments within 2 to 18 days. In 5 experiments there was a simultaneous fall in serum Ca, and the cows collapsed in tetany. The other 30 cases remained clinically healthy in spite of the low serum Mg level as long as the diet was given unchanged. In 25 cases these already hypomagnesaemic cows were exposed to a relatively high oral supplementation of Na_2HPO_4 , Na_2SO_4 or both. In 9 cases the supply led to pronounced tetanic convulsions, in 4 cases a subtetanic state was observed, and in 4 cases the cows showed borderline symptoms, i.e., they reacted clinically as well as analytically immediately after the supply was given, but no attacks appeared, and the cows recovered as soon as normal fodder was given. In 8 cases the cows did not react to the "trigger" doses at all.

The experiments are discussed and the reactions compared with those observed when high-lactating cows are let out on tetany-prone, $(\text{NH}_4)_2\text{SO}_4$ -dressed pastures. The conclusion is drawn that a high Mg content in the diet prior to a Mg-deficient diet has little, if any, influence on the tendency to precipitate tetany. Considerable decreases in content, or availability, of dietary Mg always lead to decreases in serum Mg. Occasionally, under special unfavourable circumstances, when the drop in serum Mg is extraordinary rapid, the serum Ca level may drop as well and tetany occur without further stressing elements. In most cases, however, the results seem to support the suggestion that tetanic convulsions are not conditioned entirely by the reduced availability of Mg but are to a great extent dependent upon the serum Ca-reducing effect of co-operating, so-called trigger compounds, simultaneously present in the diet. In this connection the addition of Na_2HPO_4 and Na_2SO_4 to the Mg-poor diet has been studied and found to confirm the suggestion made in earlier papers (*Ender et al.* 1957;

Dishington 1965) that the high content of Na, K, P, and S found in $(\text{NH}_4)_2\text{SO}_4$ -dressed pasture grass seems to play a prominent part in making this grass tetany-prone.

ZUSAMMENFASSUNG

Hypomagnesaemie und hypomagnesaemische Tetanie bei melkenden Kühen hervorgerufen durch eine Futteränderung.

Fünfunddreissig Versuche, in welchen hochleistende Milchkühe einer plötzlichen Futteränderung von normal Mg-haltigem Futter zu ausgesprochen Mg-armen Futter ausgesetzt wurden, werden beschrieben. Allen Kühen wurde dasselbe Normalfutter verabreicht. Dieses enthielt 25 g Mg pro Tagesration, während das Versuchsfutter individuell berechnet wurde und nur 2,55 g Mg zum Erhalt erhielt + 0,15 g pro kg Milch. Tägliche Serummineral-Analysen zeigten einen deutlichen Fall im Serum-Mg-Spiegel und einen ausgesprochen hypomagnesaemischen Zustand in allen 35 Versuchen nach 2 bis 18 Tagen. In 5 Fällen zeigten ebenfalls die Serum-Ca-Werte schon im Laufe von wenigen Tagen eine fallende Tendenz mit einer ausgesprochenen Tetanie zufolge. In 30 Versuchen waren die Kühe weiterhin lebhaft trotz der sehr niedrigen Serum-Mg-Werte, so lange das Futter unverändert verabreicht wurde. In 25 Versuchen wurden den anscheinend gesunden aber hypomagnesaemischen Kühen plötzlich relativ grosse orale Zuschüsse von entweder Na_2HPO_4 oder Na_2SO_4 oder von beiden Stoffen verabreicht. Diese Zuschüsse hatten in 9 Versuchen typische Tetanieausbrüche zufolge und in 4 Versuchen wurde bei den Tieren ein subtetanischer Zustand festgestellt. In weiteren 4 Versuchen gab der Zuschuss eine deutliche Reaktion. Der Krampf blieb jedoch aus und die Kühe erholten sich ohne Behandlung. In 8 Fällen wurde keine Reaktion auf den Zuschuss registriert.

Das Forschungsprogramm sowie die Ergebnisse werden diskutiert und mit der Zusammensetzung des Futters und den Reaktionen der Kühe auf stark $(\text{NH}_4)_2\text{SO}_4$ -gedüngten Tetanieweiden verglichen. Die Versuchsergebnisse zeigen, dass das Speicherungsvermögen der Kühe was zugängliches Mg betrifft minimal ist. Ein grosser Überschuss von Mg im Futter vor der Futteränderung oder vor dem Auslassen auf die Weide hat geringen oder gar keinen Einfluss, wenn das neue Futter tetaniehervorrufend ist. Futter, welches Mg-Mangel oder herabgesetzte Möglichkeiten für eine Mg-Aufnahme bewirkt, wird immer einen Fall im Serum-Mg-Spiegel hervorrufen. In einzelnen Fällen unter besonders ungünstigen Umständen, wo der Serum-Mg-Gehalt besonders schnell herabsinkt, kann das Serum-Ca-Niveau ebenfalls gestört werden und eine Tetanie ohne weiteren äusseren Anlass hervorrufen. In den meisten Fällen lassen die Ergebnisse jedoch vermuten, dass der Tetanieausbruch nicht nur durch eine herabgesetzte Mg-Aufnahme sondern auch durch die Anwesenheit von „Trigger“-Stoffen bedingt ist. Auf den positiven Einfluss der Beigabe von Na_2HPO_4 oder $\text{Na}_2\text{HPO}_4 + \text{Na}_2\text{SO}_4$ wird hingewiesen als eine Unterstützung der früher hervorgesetzten Theorie (Ender et al. 1957; Dishington 1964). Infolge

dieser Theorie trägt der grosse Überschuss an K, Na, P und S, welcher in stark ammonsulphatgedüngten Weidegras vorhanden ist, bedeutend dazu bei, dass Tetanie auf diesen Weiden so häufig auftritt.

SAMMENDRAG

Hypomagnesemi og hypomagnesemisk tetani fremkalt hos melkekyr ved fôromlegning.

Det er beskrevet 35 forsøk hvor høytstående melkekyr har vært utsatt for en plutselig fôromlegning fra fôr med normalt Mg innhold til et utpreget Mg fattig fôr. Normalfôret var det samme for alle kuene og inneholdt 25 g Mg pr. dagsrasjon, mens det eksperimentelle fôret var individuelt beregnet og inneholdt bare 2,55 g Mg til vedlikehold + 0,15 g til hver kg melk. Daglige serum-mineral-analyser viste et tydelig fall i serum Mg-speilet, og en utpreget hypomagnesemisk tilstand i alle 35 forsøk etter 2—18 døgn. I 5 tilfeller viste også serum Ca-verdien fallende tendens allerede etter få dager, og kuene fikk tydelig tetani. I 30 forsøk var kuene fortsatt friske til tross for meget lave serum Mg verdier så lenge fôret ble gitt uforandret. I 25 forsøk ble disse friske, men hypomagnesemiske kuene utsatt for plutselige, relativt store, orale tilskudd av Na_2HPO_4 , Na_2SO_4 eller begge. I 9 forsøk resulterte tilskuddet i typiske tetaniutbrudd og i 4 forsøk ble det observert en subtetanisk tilstand hos dyrene. Fire forsøk ga tydelig reaksjon etter støtet, men krampen uteble og kuene kom seg uten behandling. I 8 tilfelle ble det ikke registrert noen reaksjon på støtet.

Opplegg og resultater er diskutert og sammenlignet med fôrets sammensetning og kuenes reaksjoner på sterkt $(\text{NH}_4)_2\text{SO}_4$ -gjødslete tetanibeiter. Forsøksresultatene viser at kuenes evne til å lagre tilgjengelig Mg er minimal. Et stort overskudd av Mg i fôret før omlegning eller før beiteslipp, har liten eller ingen innflytelse hvis det nye fôret er tetanifrembringende. Fôr som gir Mg-underskudd eller nedsatte muligheter for Mg-oppsugning, vil alltid gi fall i serum Mg-speilet. I enkelte tilfelle, under særlig uheldige omstendigheter når serum Mg-innholdet faller *særlig raskt*, kan også serum Ca-nivået bli forstyrret og tetani bryte ut uten annen ytre påvirkning, men i de fleste tilfelle synes resultatene å tyde på at ikke bare nedsatt Mg-oppsugning, men også tilstedeværelse av „trigger“-stoffer er en betingelse for tetaniutbrudd. Det positive utslag som er funnet ved tilskudd av Na_2HPO_4 eller $\text{Na}_2\text{HPO}_4 + \text{Na}_2\text{SO}_4$ er blitt pekt på som en støtte for en allerede tidligere fremsatt teori (Ender *et al.* 1957; Dishington 1964), om at det store overskudd av K, Na, P og S, som er tilstede i sterkt ammonsulphatgjødslet beitegras, må antas å være en betydelig medvirkende årsak til at tetani er så utbredt på disse beiten.

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