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CALCIUM EFFECTS ON RENAL CONSERVATION OF MAGNESIUM IN COWS

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HALSE, K.: *Calcium effects on renal conservation of magnesium in cows.* Acta vet. scand. 1984, *25*, 213—228. — Urinary excretion rates for Ca, Mg and P were compared to simultaneous measurements of the same elements in blood plasma of cows recovering from moderate hypocalcaemia post partum and in 1 animal during recovery from hypocalcaemia induced by fasting.

The renal conservation of Mg was found to be increased during the hypocalcaemia, theoretical tubular reabsorption rates being correlated to plasma Ca with $r = -0.7$, $P < 0.001$. The relationship was observed in the plasma Ca range from about 2.0 to 2.6 mmol/l.

The findings may explain the occurrence of hypermagnesaemia in hypocalcaemic cows and the delay in the development of hypomagnesaemia seen in cows with fasting-induced hypocalcaemia.

parturition; fasting; hypocalcaemia; improved renal conservation; hypermagnesaemia; Ca-Mg interrelation; phosphaturia.

Measurements both by *Sjollema* (1933) and by *Jonsgård* (1972) show that about 40 % of cows with milk fever have plasma Mg concentrations well above 1 mmol/l. Occasional values as high as around 1.5 mmol/l at the time of treatment for the disease were recovered by both authors. The indication would be that the regulation of blood Mg is influenced by variations in calcium metabolism. This belief is strengthened by the demonstration by *Marr et al.* (1955) of an abrupt decrease in serum Mg from hypermagnesaemic levels during successful Ca treatment of cows with milk fever.

Plasma Mg can be high also in the absence of milk fever symptoms in cows which are moderately hypocalcaemic post partum (*Dishington* 1975). Paradoxically, elevated Mg levels are

seen occasionally even in cows made hypocalcaemic by fasting (*Halse* 1961).

The apparent inverse relationship between plasma Ca and Mg indicated by the references above could be due to Ca-dependent variations in renal handling of Mg. The importance of kidney function in determining the plasma Mg level becomes self-evident when account is taken of the fact that normally the total extracellular Mg is filtered through the glomeruli several times a day. Even moderate variations in glomerular filtration rate or tubular reabsorption rate must influence the blood Mg level at which a steady state condition is attained.

Actually, a renal mechanism which could account for inversely related variations in plasma Ca and Mg was proposed by *Barker et al.* (1959) and by *Samiy et al.* (1960), i. e. competition between the 2 elements for tubular reabsorption. The hypothesis of absorptive pathways common to the 2 elements both in the intestinal wall and in renal tubules was advanced by *Alcock & MacIntyre* (1962). In sheep *Care & van't Klooster* (1965) found that Ca and Mg interferred with the absorption of one another from the ileum, indicating facilitated diffusion by means of a limiting common carrier system.

The hypothetical common carrier system has not yet been identified. Reference is made to reviews of Mg metabolism by *Nordin* (1976) and by *Ebel & Günther* (1980) and to the review of renal handling of Mg by *Dirks & Quamme* (1978). The renal antagonism between Ca and Mg has been confirmed. In recently parathyroidectomized rats (*Quamme* 1982) infusion of Ca resulted in an increase in the urinary excretion of Mg. In the same animals it was shown by microperfusion of segments of single renal tubuli that the reabsorption of Mg was reduced, mainly in the loop of Henle, by increasing the concentration of Ca in the perfusion fluid.

MATERIALS AND METHODS

The cows used were of the Norwegian Red breed, of varying age, considered to be in good health at the time of observation. Measurements were made on 6 indoor-fed animals during 7 normal, uncomplicated parturitions. Feed rations consisted of hay, grass silage, Beckman-treated straw, roots and concentrates. Cow No. 1 which was observed during 2 successive parturitions 13

months apart, was also used for a 48 h fasting experiment during the intervening lactation period, 3 months post partum. Before being fasted she was fed some concentrates and 40 kg of fresh grass per day.

Signs of paresis were not observed during or after the fast. When re-feeding was initiated the cow was normally alert. The rectal temperature was normal, 39°C, but the body surface felt cool. Rumen motility was reduced and the appetite was low. However, after 5 days she was back at a daily consumption of 40 kg of grass and 7 kg of concentrates. No change in food consumption was noted in connection with a transient decrease in plasma Ca after 10 days of re-feeding.

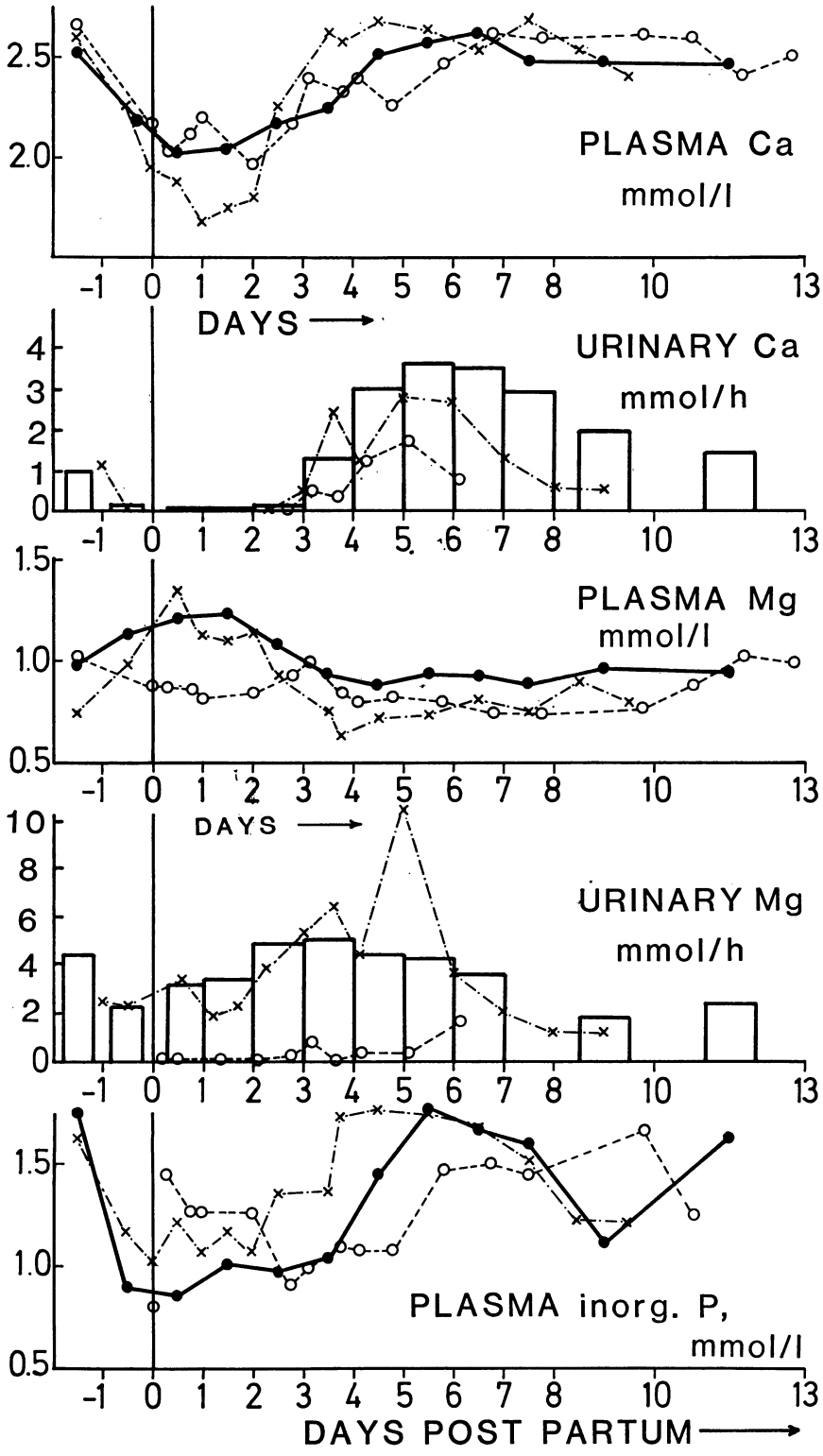
Urine was collected quantitatively in plastic bags by means of Folley catheters kept permanently inserted into the bladder for up to a fortnight. The total period of urine collection from the date of calving varied in length between 8 days and a fortnight according to the tolerance of the animals to the catheter.

The length of the urine collection intervals varied between 6 and 24 h, short intervals being preferred on the first days post partum while mineral levels were changing rapidly. Varying numbers of urines were obtained before calving, and 1 animal was not sampled at this stage.

Heparinized blood for the determination of plasma minerals was drawn from the jugular vein at the beginning and end of each urine collection interval, and the mean of analyses of the 2 samples were used in the comparison of blood and urine composition.

Urine aliquots were preserved with HCl and stored together with blood plasma at + 5°C until analyses could be performed, within a week's time.

For the determination of Ca and Mg the urines were evaporated and dry-ashed in platinum vessels. Urinary phosphorus was measured after wet-ashing with H_2SO_4 and HNO_3 in micro-Kjeldahl flasks. Ca was measured complexometrically with EGTA by means of a microadaptation of the principle described by *Ringbom* (1963) using Zn^{++} and Zincon for the end-point detection. Mg was found by difference after titration of Ca + Mg with EDTA in ethanolamin buffer with Eriochrome black T as indicator. Both titrations were performed with an EEL titrator (Evans Electroselenium) Phosphate was measured photometrically by conventional molybdate methods.


PLASMA Ca
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RESULTS

Calcium and magnesium

The following points are illustrated in Fig. 1: 1. Plasma Mg was increased during postparturient hypocalcaemia in 6 out of 7 parturitions. 2. One cow which marked herself out by developing phosphaturia (Fig. 2) still showed the inverse relationship between Ca and Mg in blood plasma. 3. The single animal failing to show a Ca-related increase in plasma Mg excreted exceptionally low amounts of Mg with the urine. 4. Ca effects on renal conservation of Mg are indicated by the fact that plasma Mg and renal Mg excretion rate tended to vary inversely during recovery from hypocalcaemia.

The last-mentioned point is further illustrated by the observations from individual cows in Fig. 3. Within all animals the ratio between Mg excretion and plasma Mg was lower at the time of Ca-minimum than after recovery, when plasma Ca culminated.

Additional evidence of Ca-effects on Mg metabolism was obtained in the fasting experiment (Tables 1 and 2). When the animal became hypocalcaemic, Mg disappeared from the urine simultaneously with Ca while plasma Mg was still at or slightly above the initial level.

The second drop in plasma Ca, 10 days after termination of the fast (Table 2), could be due to a regulatory backlash after a period of overstimulation of mineral mobilizing mechanisms. The important point in the present context is the fact that the spontaneous hypocalcaemia, occurring while dietary Mg was available, was accompanied by a 50 % increment in plasma Mg. To the retention of extracellular Mg which must have taken place corresponds a decrease in urinary Mg during the preceding collection interval.

A renal calcium treshold at 2.3 mmol/l is indicated in Table 3. In the averages are included all observations after calving from the 7 parturient cows in Fig. 1.

Figure 1. Concentrations in plasma and urinary excretion rates of Ca and Mg. Inorganic P in plasma. Variations with time post partum. Fully drawn curves and columns give means from 5 or 4 (inorg. P) parturitions. Separate curves: (O) calving no. 1, low urinary Mg; (×) calving no. 4, phosphaturia (See Fig. 2).

Table 1. Effects of 48 h of fasting on minerals in blood plasma and urine, concentrations against excretion rates. Urinary Mg decreasing before plasma Mg when the animal became hypocalcaemic. Transient hypophosphataemia. Phosphaturia during recovery. Cow No. 1, 3 months post partum, not pregnant. Milk yield from 28 to 7 kg/day during the fast, 22 kg/day after 5 days of re-feeding.

	Blood plasma, mmol/l Mean ± s			Urine, mmol/h Mean ± s			Number of samples	
	Ca	Mg	P	Ca	Mg	P	Plasma	Urine
Before fast	2.65±0.08	1.04±0.02	1.65±0.25	0.42±0.26	4.46±0.47	0.13±0.01	7	6
Fasting 12—24 h	2.03—1.93	*1.12—1.06*	*1.02*—1.69	0.02	*0.11*	0.16	2	1
After fast 0.5—1.5 days	2.03±0.26	*0.45±0.04*	2.75±0.30	0.01	0.03	0.28	3	2
After fast 2—3 days	2.46±0.14	*0.84*±0.05	2.19±0.28	0.07	*0.81*	*6.76*	3	2

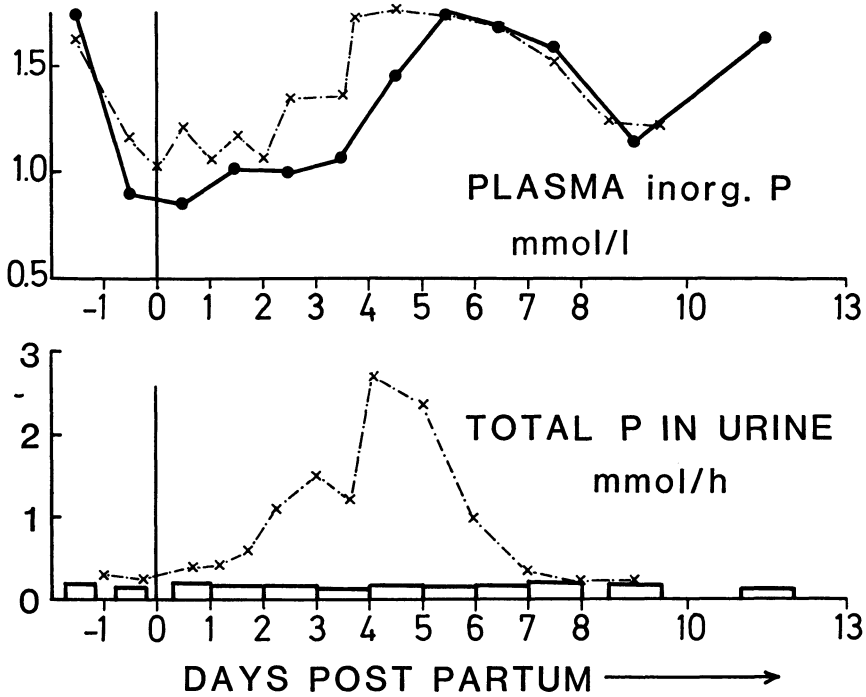

Figure 2. Total P in urine, mmol/h against time post partum, compared to plasma inorganic P. Fully drawn curve and columns: 4 parturitions. Separate curves (×): calving no. 4 with phosphaturia.

Table 2. Spontaneous hypocalcaemia in cow No 1 on the 10th day of feeding after a two-days' fast (Table 1). Mineral concentrations in plasma and urinary excretion rates. Decreased Mg excretion and hypermagnesaemia. Hypophosphataemia followed by phosphaturia. Plasma values at the beginning and end of four 24 h urine collection intervals.

Days of feeding	Blood plasma, mmol/l			Urine, mmol/h			Milk kg/d
	Ca	Mg	P	Ca	Mg	P	
8	2.99	0.94	1.21				21.1
				0.71	8.36	0.26	
9	2.75	0.96	1.32				20.6
				0.67	*5.73*	0.24	
10	*1.83*	*1.56*	*0.61*				19.2
				0.06	9.6	0.29	
11	2.51	1.13	1.95				22.5
				0.92	4.8	*2.9*	
12	2.70	0.92	2.27				23.3

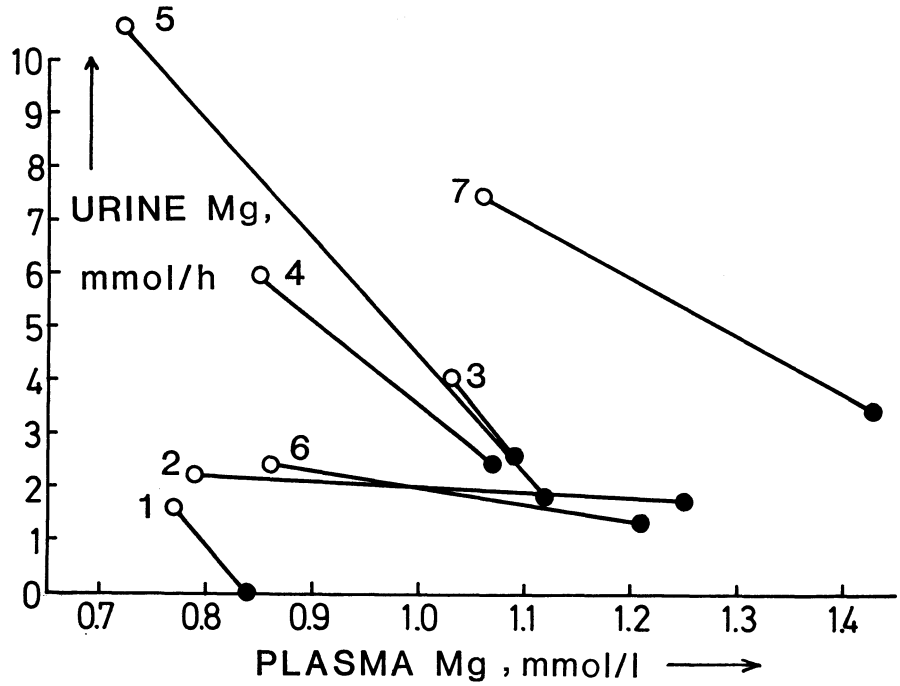

Figure 3. Plasma concentration and urinary excretion rate for Mg at time points of minimum (●) and subsequent maximum (O) in plasma Ca after calving. Same material as in Fig. 1. Calving number denoted in the graph. No. 1 and 7, the same animal in different years. This cow was also used in the fasting experiment (Tables 1 and 2).

Table 3. Apparent renal threshold for calcium at 2.3 mmol/l in blood plasma during recovery from hypocalcaemia; 7 parturitions. Same material as in Fig. 1.

Number of observations	Plasma Ca mmol/l range	Urine Ca mmol/h mean±s	Urine Ca mmol/h range
26	1.6—2.19	0.033±0.004	0.0025—0.12
7	2.2—2.29	0.051±0.016	
9	2.3—2.39	0.59 ±0.24	0.11—8.95
40	2.4—2.68	2.6 ±0.42	

Phosphorus

Plasma inorganic P was invariably low (Fig. 1) while the parturient cows were hypocalcaemic (correlation coefficient, plasma Ca and P, first 7 days post partum: $r = +0.7$). In the

fasting experiment plasma P behaved differently. Low levels were observed after a recent decrease in plasma Ca (Tables 1 and 2). Thereafter it began to increase while the hypocalcaemia persisted (Table 1). Phosphaturia was associated with Ca recovery both after fasting and in one animal post partum (Fig. 2). Mineral regulating mechanisms may have been specially strongly activated in the phosphaturic parturient cow since she showed a faster than average return to normal Ca levels (Fig. 1). The main point for the following discussion would be the fact that Mg and Ca metabolism appeared similarly interrelated in cows with and without phosphaturia.

Statistical evaluation

In Tables 4 and 5 practically equal effects of variations in plasma Ca and Mg on urinary Mg excretion rate are obtained by the different correlation and variance tests performed (r_{part} giving correlation between two parameters when the third parameter is maintained constant). Included in the estimates were all observations before and after parturition used in Fig. 1 (n = 100) and 27 sets of observations from the fasting experiment (8 days before to 12 days after the fast) partly reproduced in Tables 1 and 2.

Attention is drawn to the similarity between coefficients of regression obtained for the two materials (Table 4).

Table 4. Correlation coefficients, partial (r_{part}) and multiple (as R^2), and regression equations. Same materials as in Fig. 1 and Tables 1 and 2.

	Calving n = 100)		Fasting (n = 27)	
Variables	r	r_{part}	r	r_{part}
Mg_p—Ca_p	—0.5***	—0.6***	+0.3	—0.11
Mg_u—Mg_p	+0.22*	+0.43***	+0.57**	+0.51**
Mg_u—Ca_p	+0.27**	+0.45***	+0.65***	+0.61**

Levels of significance
*) $P < 0.05$ **) $P < 0.01$ ***) $P < 0.001$
Calving: $Mg_u = 7.25 \times Mg_p + 4.75 \times Ca_p - 14.8$ $R^2 = 0.24^{***}$
Fasting: $Mg_u = 6.5 \times Mg_p + 5.5 \times Ca_p - 15.7$ $R^2 = 0.57^{***}$
Mg_u: mmol Mg/h in urine
Mg_p and Ca_p: mmol/l of Mg and Ca in plasma.

A higher R^2 value was arrived at for fasting than for parturition. However, since the parturient material was larger, equal levels of significance ($P < 0.001$) were attained. Otherwise, R^2 values well below unity would be predictable, since renal excretory rates undoubtedly are influenced by other variables in addition to plasma Ca and Mg.

Table 5. Variance of urinary Mg, percentages explained by variations in plasma Mg and Ca. Same materials and same symbols as in Table 4.

	Mg_u			Partial effects of	
	n	mean	Sum of squares	Mg_p %	Ca_p %
Calving	100	3.43	528.3	10.7	13.8
Fasting	27	3.59	297	23.3	34

Magnitude of a hypothetical tubular effect

The tubular reabsorption rates in Table 6 were arrived at by postulating constant glomerular filtration rates = 26 l/h (*Kolb* 1962) and constant ultrafiltrability of plasma Mg = 70 % (*Nordin* 1976). The material was the same as in Tables 4 and 5. To be noted are the high correlation coefficients between plasma Ca and Mg reabsorption ($r = -0.7$, Mg poor urines excluded), the close correspondence between parturient and fasting values and the fact that a variation as large as about 30 % in the reabsorption rate was obtained within a quite narrow Ca range, straddling the apparent renal Ca threshold (Table 3).

Deviating reabsorption rates when urinary Mg was < 1 mg/h (Table 6) may be explained by filtered loads below the level needed for saturation of tubular transport systems.

It should be made clear that the calculations in Table 6 provide no proof of a tubular Ca effect. A 30 % decrease in glomerular filtration rate when animals become hypocalcaemic could explain equally well the Ca-related changes in Mg excretion.

Table 6. Theoretical renal tubular reabsorption rate for Mg (Mg_r) inversely related to plasma Ca (Ca_p). Mg_r calculated as the difference between Mg filtered and excreted per h. Assumed constant: GFR = 26 l/h and ultrafiltrable Mg = 70 % of total plasma Mg.

Plasma Ca mmol/l range	Mg_r, mmol/h, mean and standard deviation		
	7 calvings urine Mg > 1 mg/h	Fasting urine Mg > 1 mg/h	Calving and fasting urine Mg < 1 mg/h
1.6—2.19	17.88±2.56 (22)	17.4±3.3 (2)	13.95±3.47 (9)
2.2—2.39	15.06±1.91 (16)	16.3±2.84 (4)	14.54±3.23 (3)
2.4—2.59	13.75±2.11 (42)	13.54±2.23 (7)	
2.6	11.09±3.74 (14)	12.30±3.32 (8)	

Number of observations in brackets.
Correlations, Mg_r to Ca_p:
Calving, r = —0.62***, within cows r = —0.73***
Fasting, r = —0.31 (n = 27), Mg-poor urines excluded:
r = —0.72*** (n = 21)
***: $P < 0.001$

DISCUSSION

Plasma magnesium as a dependent variable

Evidently plasma Mg can be regarded as dependent on: 1) the excess of absorbed Mg which must be excreted by the kidneys to obtain a steady state condition; 2) the glomerular filtration rate; 3) the rate of tubular reabsorption. The plasma Mg level at which a given amount of Mg (1.) is excreted per hour must appear Ca dependent if 2. or 3. are influenced by plasma Ca.

Since filtration rates were not measured, only the third alternative was tested quantitatively (Table 6). The findings were consistent with the hypothesis from the literature (see introduction) of competition between the 2 elements for renal tubular reabsorption.

Magnesium at sub-threshold levels of plasma calcium

It may first be noted that the threshold level obtained for Ca for the parturient cows in Table 3 (2.3 mmol/l) is in good agree-

ment with findings by *Hove et al.* (1983) of 2.2—2.3 mmol/l. Secondly, attention is drawn to the marked variations in estimated Mg reabsorption (Table 6) negatively correlated to moderate changes in plasma Ca in the vicinity of the Ca threshold (Table 3). Possibly, when urines are practically Ca-free, Mg reabsorption is facilitated by a sub-capacity load of Ca on a tubular carrier system common to the 2 bivalent ions. The point would be that Mg metabolism is influenced by Ca variations which are within a normal range.

Fasting reactions

The observations from the fasting experiment indicate that Mg and Ca metabolism are interrelated in the same manner at other stages of lactation as in newly calved cows (Tables 4—6). An important evidence of Ca effects on Mg is the delay in the development of fasting-induced hypomagnesaemia in Table 1. In this connection reference is made to previous measurements of fasting blood minerals in cows (*Halse* 1961) which show that delayed hypomagnesaemia is a regular phenomenon when blood Ca is decreased by fasting.

It is likely that the cow in Table 1 which was fasted had keto-acidosis when feeding was resumed (*Halse* 1958) and this may have influenced the renal handling of minerals.

Hypercalcaemia

If plasma Mg tends to be high during hypocalcaemia, one would expect a Mg-depressant effect associated with high Ca levels. Actually, decreases in plasma Mg have been observed after treatment of rats with hypercalcaemia-producing doses of vitamin D (*Richardson & Welt* 1965) and in cows receiving Ca-elevating vitamin D-metabolites (*Hove et al.* 1983). The first mentioned authors found no change in faecal, urinary, muscle or total carcass Mg in the treated animals. They did not discuss renal mechanisms and suggested that the vitamin D treatment had resulted in a change in the balance between extra- and intracellular Mg. Urinary excretion rates for Mg of the cows studied by *Hove et al.* appeared unaffected by the treatment. Their material was not sufficient for the demonstration of significant effects on excretion in relation to filtered loads.

A calcium-dependent renal magnesium threshold

Estimates based on reabsorption rates from Table 6 indicate that Mg-poor urines can be produced at plasma Mg levels as high as 1 mmol/l when plasma Ca < 2.2 mmol/l (tubular reabsorption = filtered load). The corresponding value at Ca levels > 2.4 would be about 0.75 mmol/l of Mg. This last mentioned figure is in good agreement with threshold estimates from the literature (*Ender et al.* 1957). It also corresponds approximately to lower limits obtained for the Mg range of healthy cows in the field (*Halse* 1970). Direct measurements indicating enhancement of the Mg threshold during hypocalcaemia were referred to above (Results, Table 1).

Calcium or hormone effects

Seemingly convincing evidence of direct Ca effects on Mg transport was provided by *Quamme's* (1982) microperfusion experiments with single renal tubuli.

Additional Ca related effects on Mg metabolism mediated by Ca regulating hormones cannot be excluded. Parathormone can stimulate the tubular reabsorption of both Ca and Mg (*Burnatowska et al.* 1977). Tubular Mg transport may also be influenced by antidiuretic hormone, calcitonin and glucagon (*Rouffignac et al.* 1983). It is likely that several of the hormones mentioned become involved during the readjustment of mineral metabolism after hypocalcaemia: possibly overstimulation of mineral mobilizing mechanisms can lead to a calcitonin response. Attention is drawn to the findings during the recovery period in the fasting experiment (Tables 1 and 2). The cow became hypocalcaemic again between two periods of phosphaturia.

Biological and clinical implications

An improvement in the retention of extracellular Mg related to moderate decreases in plasma Ca could be of considerable homeostatic importance, delaying the development of hypomagnesaemia when the supply of minerals becomes marginal.

In the experiments referred to above (*Richardson & Welt, Hove et al.*) the moderate hypomagnesaemia of hypercalcaemic animals did not lead to detectable losses of body Mg. Whether or not an abundant supply of Ca can aggravate the hypomagnesaemia on Mg-poor diets is not known. Even when the kidneys

excrete large amounts of Ca, the Mg level at which the urine becomes practically Mg free may be well above the tetany range.

The plasma Mg level of cows with milk fever can vary as widely as from about 0.4 to 1.5 mmol/l (*Jonsgård* 1972). The variability may reflect variations in the availability of metabolic Mg. It is suggestive that in the present study postparturient hypermagnesaemia was not observed in one animal with exceptionally low Mg excretion rates (Fig. 1).

The inconsistent behaviour of Mg in milk fever could mean that the element is of limited clinical importance in the disease. It should be noted, however, that *Jonsgård* found a significantly better recovery rate for milk fever patients with plasma Mg > 1 mmol/l than for affected animals with lower Mg levels.

ACKNOWLEDGEMENT

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SAMMENDRAG

Innvirkning av kalsium på nyrekonserveringen av magnesium hos kyr.

Mengder av Ca, Mg og P utskilt per tidsenhet med urinen ble sammenholdt med samtidige konsentrasjoner av de 3 elementer i blodplasma fra kyr i løpet av normaliseringsperioden etter moderat hypokalsemi post partum og i et tilfelle etter fremkalling av hypokalsemi ved faste.

Nyrekonserveringen av Mg viste seg å være økt under hypokalsemi. Teoretiske tubulære reabsorpsjonshastigheter for elementet var korrelert til plasma Ca (interval ca. 2.0—2.6 mmol/l) med $r = -0.7$, $P < 0.001$.

Funnene kan forklare forekomsten av hypermagnesemi hos hypokalsemiske kyr og forsinkelsen i utviklingen av hypomagnesemi hos kyr med hypokalsemi etter faste.

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