ABSTRACT

Plasma Ca, P, Mg, parathyroid hormone, and 1,25-dihydroxyvitamin D were measured in blood samples taken daily from d 5 before until d 15 to 30 after calving in 28 aged dairy cows (5 yr or older) and 9 first lactation cows. Subnormal plasma Ca concentrations were defined as being below the lower limit (2.18 mmol/L) of the 99% confidence interval for lactating cows outside the month of calving. A parturient minimum in plasma Ca and P concentrations occurred usually within 24 h after calving in all animals.

Plasma mineral changes very similar to those at parturition were observed in 50% of the aged cows at one, two, or even three later occasions during the 1st mo postpartum. Interval between subsequent subnormal Ca minima in these Ca-cycling cows was 7 to 10 d. The function of the Ca regulating endocrine systems appeared adequate.

Calcium cycling with increased amplitude could be induced by feeding 200 µg/d of 1,25-dihydroxyvitamin D₃ for about 5 d around parturition (8 animals). The hypocalcemic episode was more pronounced than in untreated cows, and Ca cycling was traced throughout the 1st mo of lactation. Similar treatment also induced Ca cycling in 4 heifers, whereas 8 untreated heifers showed no tendency to Ca cycling in the postparturient period. The hypothesis is put forward that variations in intestinal Ca absorption induced by 1,25-dihydroxyvitamin D₃ are the primary cause of the cyclic changes in plasma Ca postpartum of the aged dairy cow.

INTRODUCTION

Extracellular Ca concentrations are maintained within a narrow range by the integrated operation of several endocrine feedback systems. In dairy cows a transient period of hypocalcemia regularly develops at the onset of lactation. This hypocalcemia is caused by an imbalance between Ca output in colostrum and influx of Ca to the extracellular pool from gut and bone. The hypocalcemia develops in spite of apparently adequate function of the parathyroid and vitamin D endocrine systems (7, 18) and leads to paresis in 5 to 20% of aged cows in many highly selected dairy breeds. The endocrine adjustments triggered by this hypocalcemia are thought to secure a sufficient inflow of Ca to the extracellular pool from gut and bone to meet the mammary Ca requirements and allow for the maintenance of plasma Ca within narrow ranges throughout the early part of lactation.

Plasma Ca returns to normal within 24 to 72 h after the minimum during normal calving and in uncomplicated cases of milk fever (7, 14, 18, 25). The signal leading to the homeostatic changes in Ca fluxes in connection with the onset of lactation is thus switched off. The decline from the parturient peak plasma 1,25-dihydroxycholecalciferol [1,25-(OH)₂D₃] and parathyroid hormone (PTH) some 2 to 4 d after calving (6, 7, 18) is consistent with an apparent stabilization of Ca metabolism early postpartum. Constant plasma Ca concentrations after the parturient hypocalcemia usually indicates adequate and rapid adaptations of Ca metabolism to the demands of lactation.

The magnitude of change in Ca fluxes from the nonlactating to the lactating state is dramatic with a three- to sixfold increase in Ca losses from extracellular fluid (16, 24). In studies
on the use of 1α-hydroxylated metabolites of vitamin D for milk fever prevention, we have regularly sampled cows for 2 to 4 wk after parturition. The finding of pronounced, transient hypocalcemia in many untreated control cows prompted questions as to the completeness of the adaptation in Ca homeostasis following parturient hypocalcemia. The present study examines the homeostatic control of plasma Ca more closely in high yielding dairy cows after 1 wk postcalving with special emphasis on the effects of age and of preparturient treatment with 1,25-(OH)₂D₃.

MATERIALS AND METHODS

Animals. Norwegian Red cows from the University herd were fed according to, or slightly above, Norwegian standards (2). The experiments were conducted during the indoor feeding season and available feeds changed somewhat. Silage and concentrates were used throughout. In addition, freshly cut grass and root tops were fed during early autumn and roots during winter. The diet was supplemented with minerals (especially Mg), and daily intakes from the basal ration during the last weeks before calving were 45 to 60 g Ca, 25 to 30 g Mg, and 33 to 43 g inorganic phosphate (P). Cholecalciferol added to the concentrate supplied at least 9000 IU/d of vitamin D. Twenty-eight aged cows at third or later parturition and 9 first lactation cows were used for the study of plasma mineral composition. Blood sampling started 7 to 14 d before expected calving and ended 14 to 35 d after parturition. Reference data were also collected to establish limits for normal variations in plasma mineral concentrations. Analyses of samples from 129 cows in first to eighth lactation, grouped according to parity and stage of lactation, are given in Table 1.

Treatments. To test the potential effects of a prepartal stimulated absorption, synthetic 1,25-(OH)₂D₃ (200 μg/d, F. Hoffman La Roche, Basel, Switzerland) was given to a separate set of 8 cows and 4 first lactation cows. The 1,25-(OH)₂D₃ was incorporated in fatty acid pellets as described earlier (10). Pellets were given orally in the concentrate from about d 5 before expected parturition and daily until d 1 after parturition.

Blood Sampling and Analysis. Samples of venous blood (30 ml) were drawn daily about 3 h after start of morning feeding. Plasma was separated, usually within 2 h, and kept frozen (−20°C) until analysis. Plasma Ca, Mg, and P were determined as described earlier (14). Plasma samples were measured each week during the experiment. The between assay variation for Ca and Mg was below 2% as estimated by repetitive analyses of pooled cow plasma.

Plasma 1,25-(OH)₂D concentrations were measured by radioimmunoassay on extracts of plasma purified by high pressure liquid chromatography (11) and plasma immunoreactive parathyroid hormone by a homologous radioimmunoassay with guinea pig antibovine PTH antibody (9). The hormones were measured in

<table>
<thead>
<tr>
<th>Lactation no.</th>
<th>Days postpartum</th>
<th>n</th>
<th>Ca</th>
<th>Mg</th>
<th>P</th>
</tr>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>X SE</td>
<td>X SE</td>
<td>X SE</td>
</tr>
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<td>1</td>
<td>30−120</td>
<td>9</td>
<td>2.59 .12</td>
<td>.99 .08</td>
<td>1.52 .24</td>
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<tr>
<td>1</td>
<td>120 nl¹</td>
<td>29</td>
<td>2.61 .08</td>
<td>1.00 .09</td>
<td>1.55 .19</td>
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<tr>
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<td>30−120</td>
<td>26</td>
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<td>1.08 .19</td>
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<tr>
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<td>50</td>
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<td></td>
<td>15</td>
<td>2.57 .10</td>
<td>1.01 .11</td>
<td>1.09 .25</td>
</tr>
</tbody>
</table>

¹ nl = Nonlactating, pregnant cows.
selected samples taken near calving and close to the individual postparturient minima in plasma calcium concentrations.

**Treatment of Results.** In the presentation of data from individual animals days were numbered relative to the day of calving (d 0). Days after calving (dac) were positive and days before calving negative. Reference samples defined the lower range of normal Ca. The lower limit of the 99% confidence interval for plasma Ca as measured in lactating cows sampled from the second to the last month of lactation was 2.18 mmol/L. Plasma Ca concentrations below 2.18 mmol/L were interpreted as hypocalcemia, a disturbance of normal plasma Ca homeostasis. Aged, untreated cows were divided into two groups, “Ca cyclers” and “noncyclers” depending on whether plasma Ca <2.18 mmol/L was detected or not during the first 15 to 20 d of lactation. Each group contained 14 cows. Average parity (4.6 vs. 4.4 in Ca cyclers and noncyclers, respectively), whole lactation yield (mean ± SD) (6765 ± 1181 vs. 6691 ± 593 kg), and peak milk yield (30.8 ± 4.6 vs. 31.6 ± 4.1 kg) were the same in the two groups.

Individual cows differed in the timing of the hypocalcemic episodes relative to parturition. Calculation of daily changes in concentrations of plasma constituents therefore had to be synchronized to demonstrate changes in mineral and hormone concentrations. The average time interval between two adjacent Ca minima ranged between 7.8 and 9.2 d in Ca cyclers and cows treated with 1,25-(OH)2D3. Time of observation was somewhat arbitrarily divided into four periods of 10 days: -5 to 4 d after calving, which was the calving period; 5 to 14 dac; 15 to 24 dac; and in a few cows also the period 25 to 34 dac.

**Statistical Methods.** Correlation coefficients were calculated, and comparisons were of group means by Student’s t test and analyses of variance.

**RESULTS**

Plasma Minerals in Aged Cows

**Plasma Calcium Concentrations.** Daily average plasma Ca concentrations for all 28 cows are shown in Figure 1. Fairly constant concentrations were observed until d 1 before parturi-

![PLASMA CALCIUM (mmol/L)](image)

**Figure 1.** Plasma concentrations of calcium before and after calving in 28 aged cows. Averages and standard errors.
minimal Ca concentrations were higher than during the two previous minima, approaching the 2.18 mmol/L limit. The interval between consecutive Ca minima varied between 8.0 and 9.2 d (Table 2).

Average plasma Ca in the 14 "noncyclers," which were capable of maintaining Ca above the 2.18 mmol/L border 5 to 14 dac are in Figure 2A and plotted the same way as the cyclers. The minimum 2.03 mmol/L at parturition occurred .6 ± .4 dac, and 10 cows had Ca below the normal limit (Table 2). During 5 to 14 dac, minimum plasma Ca concentration was 2.33 mmol/L, i.e., well within the normal range. Individual minima occurred on nearly every day of this period. Only 5 cows were sampled 15 to 24 dac, and 1 cow showed a Ca concentration below 2.18 mmol/L (Table 2).

**Plasma Magnesium.** Concentrations increased at parturition with a peak coinciding with the

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**Table 2.** The average and standard error of the number of days after calving (dac) at which minimum plasma calcium was observed in experimental animals.

<table>
<thead>
<tr>
<th>Interval based on cycling (dac)</th>
<th>d</th>
<th>Q</th>
<th>Q + 1</th>
<th>Q + 2</th>
<th>Q + 3</th>
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</thead>
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<td>5 to 14</td>
<td>d</td>
<td>Q</td>
<td></td>
<td></td>
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<td>Q</td>
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<td>d</td>
<td>Q</td>
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<tr>
<td>25 to 34</td>
<td>d</td>
<td>Q</td>
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</tr>
</tbody>
</table>

1. **Q** = Quotient between the number of cows that showed a Ca minimum below the 2.18 mmol/L limit and the total number of cows sampled in the time interval.
2. **1,25-Dihydroxyvitamin D₃**.
Plasma PTH increased from about 1 ng/ml before parturition to an average of 5 ng/ml on the day of the Ca minimum at parturition in Ca-cycling and noncycling cows (Figure 4). Values varied markedly among animals, as demonstrated by the large standard errors. Height of the PTH peaks did not differ between the two groups. A second peak was also seen on d 10 in the Ca cyclers and on d 9 in the noncyclers, i.e., on the same day as the Ca minimum of each group. Magnitude of PTH responses in the two groups were similar, in spite of pronounced hypocalcemia on d 10 in the Ca cyclers. Samples from the Ca-cycling cows were analyzed also at 15 to 24 dac. The Ca minimum of this period (2.20 mmol/L) occurred, on average, on d 19. A small increase in plasma PTH was observed on the day of the Ca minimum, but another peak of similar magnitude followed 3 d later in spite of normal Ca concentrations (Figure 4). Nearly identical coefficients of correlation (total correlations) were observed between Ca and PTH in Ca minimum at d 1 (Figure 3; only cyclers shown). After parturition, plasma Mg increased sharply on the day of the first Ca minimum (Figure 3; P<.005; paired t test) but not on the day of the second minimum. No consistent changes in plasma Mg that could be related to Ca cycles were seen in the noncyclers (curves not shown).

Plasma Phosphorus. Concentrations declined in the expected fashion at parturition, as shown for the cyclers in Figure 3. A significant drop in plasma P (P<.01, paired t test) was observed on the same day as the first Ca minimum after parturition occurred. A similar tendency to a decline on d 19 (the day of the second Ca minimum in these cows) was not statistically significant. Similar changes were not detected in the noncyclers.
cyclers and noncyclers ($r=-.46$ and $r=-.45$, respectively; $P<.001$).

After the parturient hypocalcemia, plasma $1,25-(OH)_2D_3$ increased two- to threefold from prepartal averages of about 50 pg/ml. Differences between cyclers and noncyclers were not detected (Figure 5). Plasma $1,25-(OH)_2D_3$ increased in the Ca cyclers from 40 pg/ml on d 5 to a peak of 100 pg/ml on the day of the Ca nadir in this period. No peak in plasma $1,25-(OH)_2D_3$ was detected in the noncyclers concurrently with the Ca nadir, but a 50 to 60% rise in average $1,25-(OH)_2D_3$ could be seen between d 8 and 12 (Figure 5).

**Effect of Prepartal 1,25-(OH)$_2$D$_3$ Treatment on Calcium Cycling**

Treatment with $1,25-(OH)_2D_3$ before and up to 1 d after parturition increased plasma Ca to hypercalcemic concentrations (Figure 6). From d 3 to 5 onward a steady decline in plasma Ca was seen in all treated cows. The average minimum of 1.55 mmol/L occurred on d 11.3 ± 0.6. All cows showed plasma Ca well below the 2.18 mmol/L limit, and 3 cows developed clinical milk fever. Plasma Ca became normal 2 to 3 d after the hypocalcemic nadir, but proper homeostatic control of plasma Ca was not regained for several weeks. Two additional minima in plasma Ca occurred on d 20.9 ± 0.7 and 30.0 ± 0.7 (Figure 6). Plasma Ca was below 2.18 mmol/L on d 21 in all 7 cows sampled. Similarly, the 4 cows sampled through the last minimum were at or below the 2.18 mmol/L limit. Changes in plasma P concentrations paralleled Ca closely (Figure 6). A high P concentration around parturition was followed by a steady decline to a minimum value on d 11, coincident with the first Ca minimum. A second minimum occurred on d 21. Changes were significant both from d 10 to 11 and from d 20 to 21 ($P<.05$, paired $t$ test). Changes in plasma Mg were reciprocal to the Ca concentrations (data not shown).

**Changes in Plasma Hormones in 1,25-Dihydroxyvitamin D$_3$ Treated Cows**

Plasma PTH was suppressed around parturition. Peaks in plasma PTH coinciding with the hypocalcemic minima on d 11 and 21 (Figure 7) clearly indicated that the parathyroid glands reacted adequately to low plasma Ca concen-
Figure 7. Plasma concentrations of parathyroid hormone (PTH) and 1,25-dihydroxyvitamin D [1,25-(OH)₂D₃] in nine aged cows fed 200 μg 1,25-(OH)₂D₃ daily from about d 5 before until d 1 after calving. Individual concentrations 5 to 14, 15 to 24, and 25 to 32 d after calving were synchronized using the Ca nadir of each cow for the four periods as the reference point before calculation of means and standard errors. Asterisks indicate day of the Ca minimum in each period. Broken lines indicate the border between adjacent periods.

Plasma 1,25-(OH)₂D concentration in the animals treated with 1,25-(OH)₂D₃ rose before calving to about 180 pg/ml and declined later to normal precalving concentrations. The response to the first hypocalcemic episode was low with a peak of 100 pg/ml 2 d after the hypocalcemic nadir in spite of the pronounced hypocalcemia. The reaction to the next hypocalcemic minimum appeared adequate with a sharp 1,25-(OH)₂D peak of 180 pg/ml on d 22 (Figure 7).

Plasma Minerals at Parturition in Normal and First Lactation Cows Treated with 1,25-Dihydroxyvitamin D₃

Plasma Ca declined at parturition in 9 untreated first lactation cows to an average nadir of 2.29 ± .03 mmol/L (Figure 8). Plasma Ca concentrations below the 2.18 mmol/L limit were not detected during the subsequent period of observation that ended on d 15 after calving (Figure 8). The hypercalcemic response to administered 1,25-(OH)₂D₃ resembled that observed in older cows. In contrast to the untreated animals, all first lactation cows treated with 1,25-(OH)₂D₃ showed hypocalcemia 5 to 15 dac (Table 2). Minima ranged between 1.79 and 2.15 mmol/L and occurred on d 9, 11, 13, and 14, respectively. Plasma P concentrations paralleled the described changes in Ca, whereas Mg declined during the hypercalcemic period in the 1,25-(OH)₂D₃-treated heifers (curves not shown).

DISCUSSION

The ruminant forestomachs smooth out variations in intestinal flow related to food intake and allow for a precise adjustment of intestinal Ca absorption to physiological needs. The low variability (4 to 5%) in plasma Ca concentrations of lactating cows sampled outside the calving month reflects the operation of efficient control mechanisms. Calving and the onset of lactation represent, however, major challenges to the Ca homeostatic system, and variability was much greater during the 1st mo of lactation. Fifty percent of the aged cows were incapable of maintaining plasma Ca above the
lower normal limit as defined by the 99% confidence interval of plasma Ca concentrations in cows outside the 1st mo of lactation. Hypocalcemic episodes lasting 1 to 2 d occurred two to three times with a periodicity of about 9 d (Table 2) both in the Ca cyclers and in the cows treated with 1,25-(OH)2D3. This Ca cycling has not received attention, probably due to the short duration of the hypocalcemia (Figure 2) and because most studies designed to test the effects of various measures to prevent milk fever discontinue blood sampling before the first hypocalcemic episode would be expected.

By plotting average Ca each day after calving, the hypocalcemic episodes were lost (Figure 1). The method of synchronizing the individual hypocalcemias in 10-d periods before calculating averages obviously carries a risk, however, because it will also synchronize physiologically insignificant plasma Ca changes. A fixed plasma Ca concentration (2.18 mmol/L) was therefore used to divide cows into Ca cyclers and noncyclers before calculation of average plasma Ca concentrations. Changes detected in the noncyclers could be regarded as Ca cycling of a lower intensity, or alternatively, as representing the "noise" in the plasma Ca regulating system of the cow. Sharp increases in PTH concentrations on the day of the first Ca minimum after calving (Figure 4) would indicate that the changes in the noncyclers did represent the same physiological changes as those discussed for the Ca cyclers, albeit with lower intensity.

The degree of instability of the Ca regulating system of the Ca cyclers was demonstrated by the fact that the average nadir on d 10 after calving was identical to the nadir at parturition. Chemical parameters measured in the present study did not give clues as to why some aged cows showed Ca cycling. Average age, parity, peak milk yield, or whole lactation yield did not differ between cyclers and noncyclers. A nonsignificantly lower Ca minimum in cyclers than noncyclers may, however, be related to the tendency to cycling. Changes in plasma PTH and 1,25-(OH)2D concentrations appeared appropriate in both groups.

The hypocalcemia, which occurs regularly at parturition in all aged dairy cows is accompanied by reduced plasma P and elevated Mg. Mammary demands for Mg are low compared with those for Ca and P, and the hypermagnesemia is probably of renal origin (3). The observed changes in plasma concentrations of P and Mg strengthen the concept of Ca cycling. When plasma P and Mg were averaged using days relative to the Ca minima as the grouping criterion (Figures 3 and 6), low P and high Mg concentrations coincided with the Ca minima both in the cyclers and in the cows treated with 1,25-(OH)2D3.

The effect of 1α-hydroxylated metabolites of vitamin D [1,25-(OH)2D3; 1-(OH)D3] on plasma mineral concentrations and intestinal Ca absorption has been well-documented in ruminants. No reports of Ca cycling appear to exist, although low plasma Ca were noted by some investigators toward the end of an observation period where the effects of 1α-hydroxylated vitamin D metabolites were tested in cows (13, 14, 19, 20). In the present study, 1,25-(OH)2D3 greatly modified plasma Ca regulation after parturition. Calcium cycling was induced in all cows studied, and amplitude of plasma Ca oscillations was augmented to the extent that clinical cases of hypocalcemia occurred.

The time interval between consecutive minima in plasma Ca from the parturient minimum onward was remarkably constant (8 to 9 d, Table 2) both in the Ca cyclers and during induced cycling in the cows treated with 1,25-(OH)2D3. This finding strongly suggests that a common mechanism with a time constant of about 8 d is responsible for the Ca cycling. In aged cows, this cycling is triggered by large changes in Ca fluxes at the onset of lactation, whereas 1,25-(OH)2D3 treatment, or more likely withdrawal of 1,25-(OH)2D3 treatment combined with a mammary Ca drain trigger the Ca cycling in cows treated with 1,25-(OH)2D3. Several lines of evidence point to fluctuations in intestinal Ca absorption as a cause of Ca cycling. Absorption of Ca increases rapidly after parturition (22, 23, 28). In two of five cows studied by van't Klooster (28), a transient lowering of 4SCa absorption was detected between d 10 and 12 after calving, i.e., in the same interval as the first Ca minimum occurred in the Ca cyclers. We have shown in the goat that a single dose of 1,25-(OH)2D3 will maintain an enhanced rate of Ca absorption for 6 to 10 d (10, 11). Similarly, duration of the hypercalcemic and hyperphosphatemic effects of 100 to 500 μg doses of 1,25-(OH)2D3 or 1-(OH)D3 in dairy cows were some 6 to 12 d (5, 13, 26).
Taylor (27) studied the kinetics of Ca-binding protein (CaBP) induced by 1,25-(OH)2D3 in the intestinal epithelial cells of chickens treated with 1,25-(OH)2D3. The CaBP staining cells were present within 24 h in the total population of enterocytes, and replacement of these cells occurred gradually by newly formed cells from the intestinal crypts, which lacked CaBP. About two-thirds of the activated cell population were lost in the course of 5 d. Although little is known about cell dynamics of the enterocytes in periparturient cows, the data just referred to fit well with the hypothesis that Ca cycling is secondary to fluctuations in intestinal absorption.

Cows in late pregnancy have plasma 1,25-(OH)2D concentrations of about 50 pg/ml. At parturition, a surge in plasma 1,25-(OH)2D concentration of about 1 to 3 d duration is observed; peaks are 100 to >200 pg/ml (1, 6, 7, 14) (Figure 5). Plasma Ca and PTH concentrations return to normal within 2 to 4 d and for about 1 wk the hypocalcemic stimulus for further PTH and 1,25-(OH)2D production is absent. By extrapolating from the experiments in goats (10, 11), one would assume that the effects of the 1,25-(OH)2D surge in stimulating intestinal Ca and P transport would last about 1 wk, thereafter to vanish. This fits well with the observation of the first Ca minimum on d 10 in the Ca cyclers and on d 11 in the cows treated with 1,25-(OH)2D3 where the last 1,25-(OH)2D3 dose was given on d 1. Further support for the hypothesis that cyclic changes in Ca absorption in early lactation are caused by rapid variation in the supply of Ca from the gastrointestinal tract are given by experiments showing how dependent plasma Ca homeostasis is on an hourly supply of absorbed Ca. Only a few hours with gastrointestinal stasis or with greatly reduced intestinal Ca content significantly reduced plasma Ca concentrations (12, 21).

High 1,25-(OH)2D may directly or through hypercalcemia suppress the activity of the renal 25-hydroxyvitamin D [25-(OH)D] 1α-hydroxylase (1-hydroxylase), which makes 1,25-(OH)2D from precursor 25-(OH)D. An apparent lack of hydroxylase activity has been connected to problems with Ca homeostasis in the postparturient period in cows treated with vitamin D and several of its metabolites (8, 17). In the present study such an inhibition could explain the unexpectedly low 1,25-(OH)2D peak observed in connection with the first hypocalcemic episode in the cows treated with 1,25-(OH)2D3 (Figure 7). The responsiveness of the parathyroid glands to hypocalcemia was not impaired as a result of the periparturient hypercalcemia in the animals treated with 1,25-(OH)2D3. It should be further emphasized that apparently adequate 1,25-(OH)2D and PTH responses were observed to both the parturient hypocalcemia and the subsequent plasma Ca nadir in the untreated Ca cyclers. It is therefore highly unlikely that inhibition of 1α-hydroxylase activity plays a physiological role under normal conditions in the periparturient cow.

Plasma Ca dropped much less in first lactation cows than in aged cows (Figures 1 and 8). An important factor in the buffering of hypocalcemia is the size of the readily exchangeable pool of Ca in bone. Calves and heifers have larger pool sizes than adult cattle (4). When 1,25-(OH)2D3 was given before calving, hypocalcemia (Ca <2.18 mmol/L) was observed even in first lactation cows, and extensive Ca cycling occurred in aged cows. The hypercalcemia that resulted from 1,25-(OH)2D3 treatment (Figures 6 and 7) may have inhibited Ca mobilization from bone, making the animals more susceptible to reduction in the Ca supply from the intestine.

Bone is capable of maintaining normal plasma Ca concentrations at parturition when stimulated by low Ca diets in late pregnancy. Too little is known, however, about the size of the bone pools of Ca and how they change near parturition. By averaging the results from 1 wk balance and Ca kinetic trials, Ramberg et al. (22, 23) reported that Ca absorption was increased for about 2 wk after parturition. After that time, a reduction in Ca absorption occurred that apparently was compensated for by increased transport of Ca from bone. Differences between individuals in the capacity to mobilize Ca may explain why some aged cows develop hypocalcemic cycling whereas others can buffer varying Ca inputs from the intestine more effectively and thus maintain normal plasma Ca concentration after parturient hypocalcemia.

The selection of dairy breeds for high milk production strains the homeostatic controls of the cow in the early part of lactation. Calcium
cycling, as described in the present paper, is an example of a temporary mismatch between mammary requirements and homeostatic capacity. The damped oscillations in plasma Ca do not apparently lead to clinical problems, because very few cases of milk fever are treated other than from 3 d before to 3 d after calving, at least in the Scandinavian countries. Calcium cycling may, however, be amplified by peripartal vitamin D treatment. Experiments designed to test the use of vitamin D compounds for milk fever prophylaxis should accordingly monitor plasma mineral concentrations and clinical cases of milk fever for at least 2 wk after parturition. High yielding dairy cows are, as a rule, in negative energy balance in early lactation, and their digestion and metabolism are very sensitive to changes in food intake and food quality. Low plasma Ca concentrations, even in the subclinical range, will inhibit rumen motility (15). Any occurrence of hypocalcemic episodes in the 1st mo after calving may therefore be of significance to the development of metabolic diseases such as ketosis in post-parturient cows.

REFERENCES


