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Evaluation of Clinical and Clinical Chemical Parameters in Periparturient Cows

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ABSTRACT

Certain blood parameters and clinical symptoms have been connected with milk fever and a hypocalcemic condition in the cow. The present study intended to establish a mutual connection between relevant blood parameters and potentially valuable background information about the cow and its observed clinical symptoms at calving. Two veterinarians were summoned within 12 h of parturition of 201 cows, distributed among 41 Danish commercial herds. Cows were at different parity levels (2 to 10) and breeds and management differed broadly among herds. A blood sample was taken from the vena jugularis or the tail vein and was subsequently analyzed in the laboratory. Furthermore, 13 different clinical symptoms were recorded as categorical data. We investigated associations among the data obtained. We assessed an interpretative model for actual blood calcium level with blood parameters and background knowledge of the animals. We established a path analysis using background knowledge, blood parameters, and results of clinical examinations to uncover causal connections among the variables.

Twenty-six percent of the animals were diagnosed as having milk fever and subsequent blood analyses revealed a high frequency of hypocalcemia within the general range from 0.69 to 2.73 mmol of Ca per liter. Rectal temperature, inorganic blood phosphate, and potassium were all directly correlated with blood calcium, while glucose, lactate, and magnesium were inversely associated with calcium. Blood osteocalcin was significantly lower in hypocalcemic animals, indicating that de novo synthesis of bone was arrested during hypocalcemia. A mixed effect linear interpretative model explained 75% of the variation in blood calcium. Clinical symptoms like mood, appetite, muscle shivering, rumen motility, and paresis were individually correlated with blood calcium and were thereby predictive of hypocalcemia. The path analysis showed the central role of calcium in affecting the clinical symptoms. However, several other factors contributed to hypocalcemia.
(**Key words:** milk fever, hypocalcemia, calcium status, osteocalcin)

Abbreviation key: AP = alkaline phosphatase.

INTRODUCTION

The transition between late pregnancy and early lactation, from calving until 3 to 4 wk postpartum, is a high-risk period for disease incidence in the dairy cow. The risk is especially high around parturition. Blood calcium drops to subnormal levels immediately before and during parturition, and the decrease in available body calcium may eventually continue below the levels of normal, optimal function, resulting in pathologic milk fever or parturient paresis.

Research into this condition has focused on blood calcium, ionized calcium, or total calcium measurements during periparturient hypocalcemia because of the immediate connection between blood calcium and milk fever pathology. Blood phosphorus status has also been studied because of the connection between phosphorus and calcium deposition and because of the prominent challenge to both calcium and phosphorus balances at the onset of lactation. Several other parameters, such as the connection with the mobilization of calcium from body stores, have been considered. One possible mechanism by which the cow may overcome hypocalcemia is by massive mobilization of calcium from the bone to the blood. Parameters connected with bone resorption in the periparturient period have consequently been considered (Black and Capen, 1971; Damir et al., 1994; Liesegang et al., 1998) to understand and describe the mechanisms behind the complex and delicate system known as calcium homeostasis.

However, in practice, most diagnoses of parturient paresis are founded on common veterinarian clinical observations. Rectal temperature, surface tempera-

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ture, muscle shivering and ataxia, rumen motility, paresis etc. are, considered collectively, those checks most often monitored. The obvious advantage of clinical observations is that diagnoses can be generated immediately (i.e., on the spot), an almost necessary prerequisite to successfully combat acute milk fever.

Subclinical conditions of milk fever known as hypocalcemia may be difficult to unveil by standard clinical procedures. However, hypocalcemia may be of importance in practical farming. Numerous reports associate parturient hypocalcemia and periparturient disorders in cattle (Curtis et al., 1983; Massey et al., 1993). Because hypocalcemia affects animal health and dairy farm economics, diagnosing it is important.

The aim of the present investigation was 1) to describe the distribution of blood calcium in a group of parturient cows and to elucidate the connection between calcium and other potential (relevant) blood parameters, including those connected with the bone accretion; 2) to implicate and estimate the general knowledge about the cows (background knowledge) with blood parameters to describe factors connected with, and important to, the low calcium syndrome (hypocalcemia); and 3) to strengthen the potential hypocalcemia diagnosis that might be given under cow-side conditions, i.e., not using invasive methods. We evaluated the connection between easily available information (background knowledge and clinical observations) and actual blood parameters including calcium, the latter being the defining parameter of hypocalcemia and milk fever.

MATERIALS AND METHODS

Cows and Housing Systems

Cows from 41 individual, commercial herds in Hanherred in Jutland, Denmark, were studied from February 2 to September 16, 1998. Housing systems, feeding, and management differed among herds. Both tie-stall and loose-housed systems were represented. Six breeds of cattle were represented [i.e., Danish Red and White (n = 19), Danish Jersey (n = 2), Danish Red Cattle (n = 17), Simmental (n = 1), Danish Friesian (n = 160), and crossbreds (n = 1)], in total 201 animals. The distribution of cows across parities was 5 of parity 2, 68 of parity 3, 67 of parity 4, 35 of parity 5, 7 of parity 6, 5 of parity 7, 7 of parity 8, 4 of parity 9, and 3 of parity 10. None of the animals was treated with prophylactic Ca gels against milk fever.

Blood Samples

All blood samples were taken by a veterinarian within 12 h of calving. Blood was drawn from the vena jugularis or vena coccygea with Vacutainers and Li-heparinized tubes. Samples were stored on ice during transport to and centrifuged immediately after arrival at the laboratory (2000 × g, 20 min). Plasma was pipetted into polyethylene tubes and stored immediately at –18°C until further analysis.

Clinical Investigations

Two veterinarians participated in the present study, inspecting all cases personally. Thirteen different clinical symptoms were recorded as categorical data on a scale of 1 to 3, where 1 was normal, and 3 was seriously affected. The symptoms were depression (mood), excitement, appetite and rumination, muscle shivering, ataxia (failing coordination of muscle contractions), stepping of hind legs, abnormal jaw and tongue movements, tail lifting, relaxation of rectum, defecation, rumen contractions, paresis, and surface temperature between hip crests. Furthermore, rectal temperature was recorded (1 decimal readings).

Basic observations, i.e., date of calving, breed of the animal, identity of the farm, identity of the veterinarian, number of preceding calvings (parity), and whether the cow had been milked before the blood sample was drawn, were also recorded.

Analyses

The blood plasma was analyzed for pH and inorganic ions, Na^+, Cl^-, K^+, and Ca^{++}, with selective electrodes of the ABL model 605, blood gas and electrolyte analyzer (Radiometer Medical A/S, Copenhagen, Denmark). Glucose and lactate concentrations were determined by means of bioelectrodes with the same equipment. Due to possible escape of carbon dioxide from blood samples (and subsequent increase in pH) during transport from harvest to analysis, only corrected values of ionized Ca were used (cCa^{++} [pH = 7.40]) as given directly by the analyzer and described by Radiometer Reference Manual and Siggaard-Andersen et al. (1981). Total plasma calcium, magnesium, inorganic phosphorus concentrations, and plasma alkaline phosphatase (**AP**) activity were analyzed according to standard procedures using an OpeRA Chemistry System autoanalyzer (Bayer Corporation, Terrytown, NY). The plasma concentration of osteocalcin was measured with an immunoradiometric assay manufactured by Medgenix Diagnostics, Belgium.

Statistical Methods

All statistical analyses were performed in the S-plus system (see Venables and Ripley, 1997). Preliminary investigations into the distribution of total calcium ac-

cording to the parity of the cows, calving seasons, and clinical observations are shown as box plots, where the limits of the middle half, the median, and extreme points are highlighted. The final estimates were computed by linear mixed regression models, considering total blood calcium as the response variable. A random factor for the farm where the animals were kept was introduced (farm effect) to account for the different general conditions between farms. More specifically, we fitted models of the form:

$$y_{ij} = x^{T}_{ij}\,\beta + u_i + e_{ij} \qquad i = 1, \ldots, 41, j = 1, \ldots, n_i,$$

where y represents total calcium in blood samples, i identifies the farm, j identifies the animal, and n_i the number of observations available for the ith farm. The vector x_{ij} represents the fixed effects, while u_i (the farm effect) and e_{ij} are independent normal variables with mean 0 and variance σ^2_u and σ^2_e, respectively, which are the between-farms and within-farms variances.

Parameters were estimated by maximum likelihood, using the S-plus function Lme. Osteocalcin levels between groups were tested using Welch's test (t-test with unequal variances).

A path analysis was used to model the association between background knowledge, blood analyses, and clinical observations. Variables were subdivided into four groups, which included hypothesized direct and indirect causal paths that formed the null model. Only those paths having biological plausibility according to the findings or which have been documented in the references were illustrated in the diagram.

Path coefficients were estimated by linear regression or logistic regression, depending on whether the variable considered as the response was continuous or discrete. Notice that all the discrete variables were transformed into dichotomous variables, by aggregating the two upper categories into a single one. Herd effects were included in all the models considered in the analysis as baseline fixed effects, along the lines of Erb et al. (1985).

RESULTS

Data concerning ataxia and stepping of hind legs were incomplete and were not included in the statistical analyses. Basic information such as identity of veterinarian was not further used in modeling because of obvious confounding with season of the year. Measurement of adjusted ionized calcium in the blood samples is only defined within the pH interval between 7.2 and 7.6 (Siggaard-Andersen et al., 1981; Thode et al., 1990). Due to transport of blood samples, only 61 samples had retained a pH within this interval, the remaining observations of adjusted ionized calcium therefore had to be discarded. Figure 1, however, shows the correlation between total calcium measurements and adjusted ionized calcium in the 60 samples, where both were measured (r = 0.963, $P < 0.001$). On average, a fraction of 0.515 ± 0.032 (SD) of total calcium was ionized.

Figure 2 shows the histogram of frequencies as well as the estimated probability density of total blood calcium. Total calcium ranged from 0.69 to 2.73 mmol/L, with an average value of 1.75 ± 0.49 mmol/L (SD). Furthermore, Table 1 summarizes the noncategorized data.

Several simple correlations are obvious among the continuous variables. Rectal temperature, blood potassium, and inorganic phosphate were each associated with total blood calcium (r = 0.458, 0.548, and 0.679, $P < 0.001$, respectively). Figure 3 shows the scatter plots. Blood osteocalcin was also directly associated with blood calcium (r = 0.190, $P < 0.01$). Inverse relationships existed between blood calcium and blood glucose, lactate, and magnesium (r = –0.456, –0.250, and –0.257, $P < 0.001$, respectively). Box plots of total blood calcium, blood glucose and magnesium are shown in Figure 4.

Figure 4 also indicates the close single associations between both parity of the calving cow, season of the year of calving, and blood calcium. Rank correlations showed r = –0.312 and –0.254, respectively, both significant, $P < 0.001$.

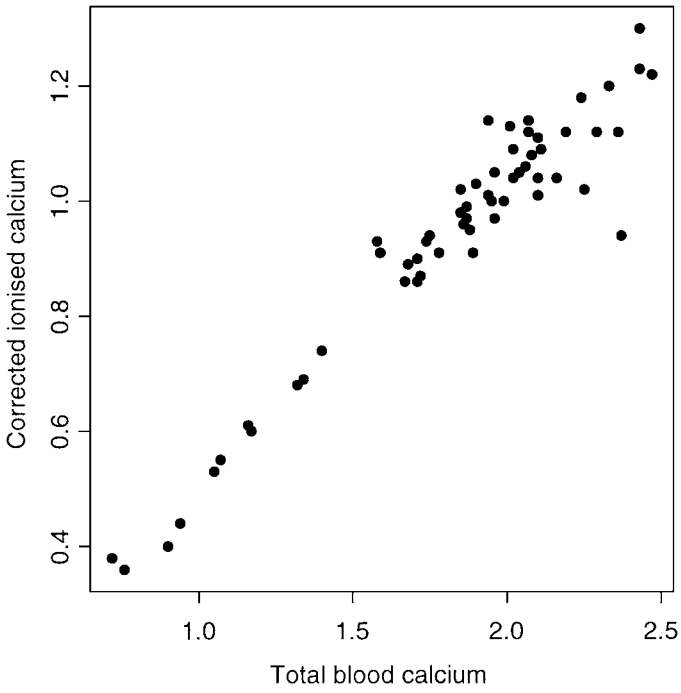

Figure 1. The association between total blood calcium measurements (abscissa) and corected ionized calcium (ordinate) in calving cows, n = 60.

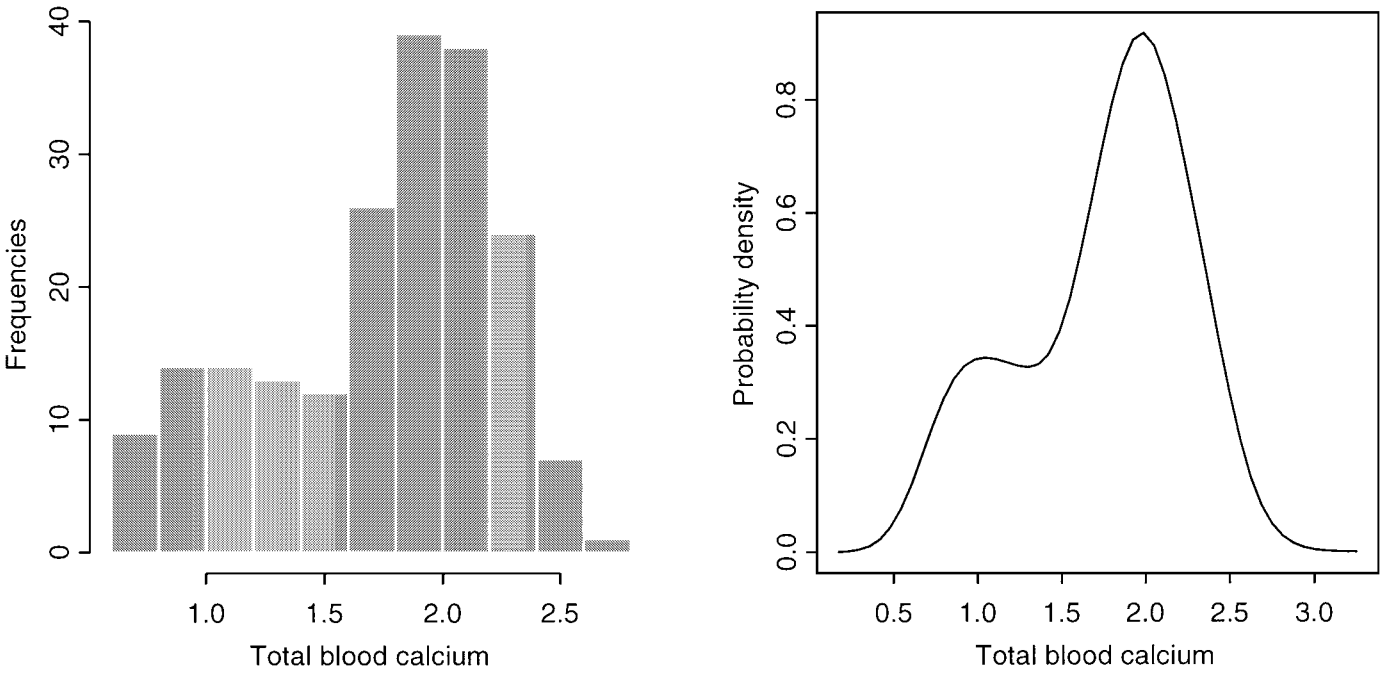

Figure 2. Total blood calcium in calving cows. Left: histogram of frequencies; right: estimated probability density (the pattern highlighted in the histogram is drawn).

A model for interpretation of total blood calcium from a concert of available background information and the clinical chemical observations in the blood is given in Table 2. The model retains only significant factors. The corresponding fitted values plotted versus total blood calcium are shown in Figure 5.

Selected exploratory analyses concerning the distribution of blood calcium on graduated discrete indicators of milk fever are shown in Figure 6. A subdivision of the variables for further causal paths is shown in Figure 7. A path analysis connecting background information, blood parameters, and clinical observations is shown in Figure 8.

Table 1. Arithmetic mean, standard deviation, minimum, and maximum values of measurements on numeric variables in 201 cows immediately after calving.

Variable	Mean value	SD	Min. value	Max. value
Rectal temperature, °C	38.5	0.7	35.3	40.4
Parity	4.3	1.6	2	10
K^+, mmol/L	4.2	0.6	2.2	5.7
Na^+, mmol/L	142.9	3.2	123.0	159.0
Cl^-, mmol/L	105.2	4.4	86.0	119.0
Glucose, mmol/L	4.8	1.9	0.3	12.2
Lactate, mmol/L	2.2	1.9	0.5	10.4
Osteocalcin, ng/ml	9.2	5.0	0.6	38.1
AP, IU/L[1]	50.2	25.3	17.0	207.0
Inorganic P, mmol/L	1.3	0.6	0.3	2.8
Mg, mmol/L	1.0	0.2	0.3	1.6
cCa (pH = 7.4) mmol/L[2]	0.93	0.22	0.36	1.30
Total Ca, mmol/L	1.75	0.49	0.69	2.73

[1]Activity of alkaline phosphatase, International Units per liter.
[2]Adjusted value for ionized calcium, 61 samples only.

DISCUSSION

Based solely on the clinical examination, 58 animals out of 201 were diagnosed as having milk fever. This high frequency (26%) is well above the average for the entire country (approximately 5% milk fever incidence, Blom 1993). When, we subsequently consulted the national central cow registration, we confirmed that we were not called out to all calvings, as agreed upon before the study began. The biphasic distribution of blood calcium in the group provided evidence that farmers called the veterinarian mainly in apparent milk fever cases. In other words, the animal material is not a representative group of Danish cows, but a group with a high incidence of milk fever or hypocalcemia.

Plasma calcium mainly exists in three forms: bound to proteins, complex bound to organic/inorganic acids such as citrate and phosphate, and ionized, i.e., free from covalent linkage. The free, ionized calcium will undoubtedly be the physiologically active calcium. However, an equilibrium between blood ionized and nonionized calcium exists: infusion of calcium borogluconate, calcium chloride, calcium propionate, or calcium

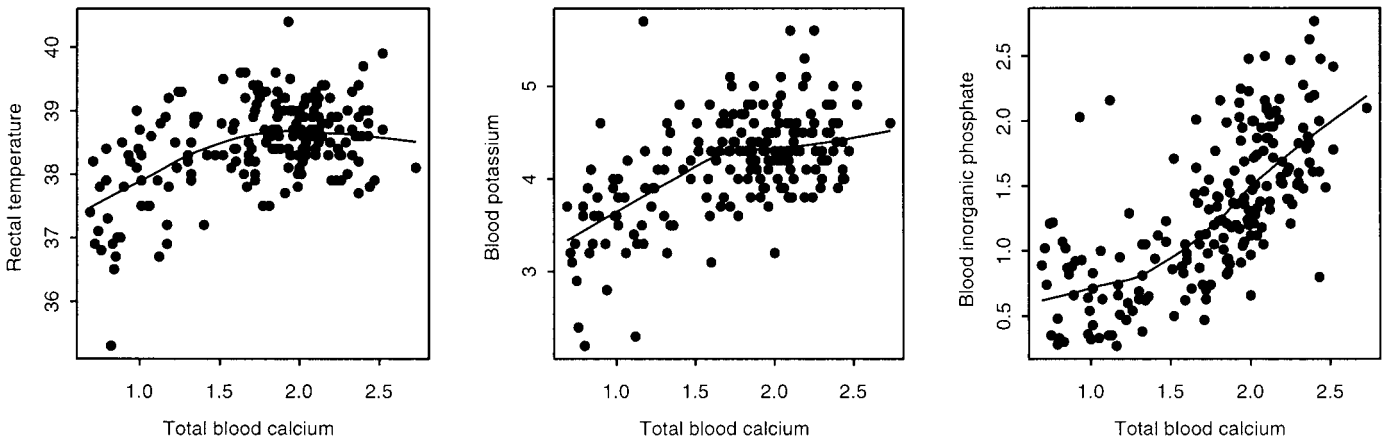

Figure 3. The association between blood calcium and rectal temperature (left panel), blood potassium (middle), blood inorganic phosphate (right). A "smoother" is superimposed to visualize the main pattern in data.

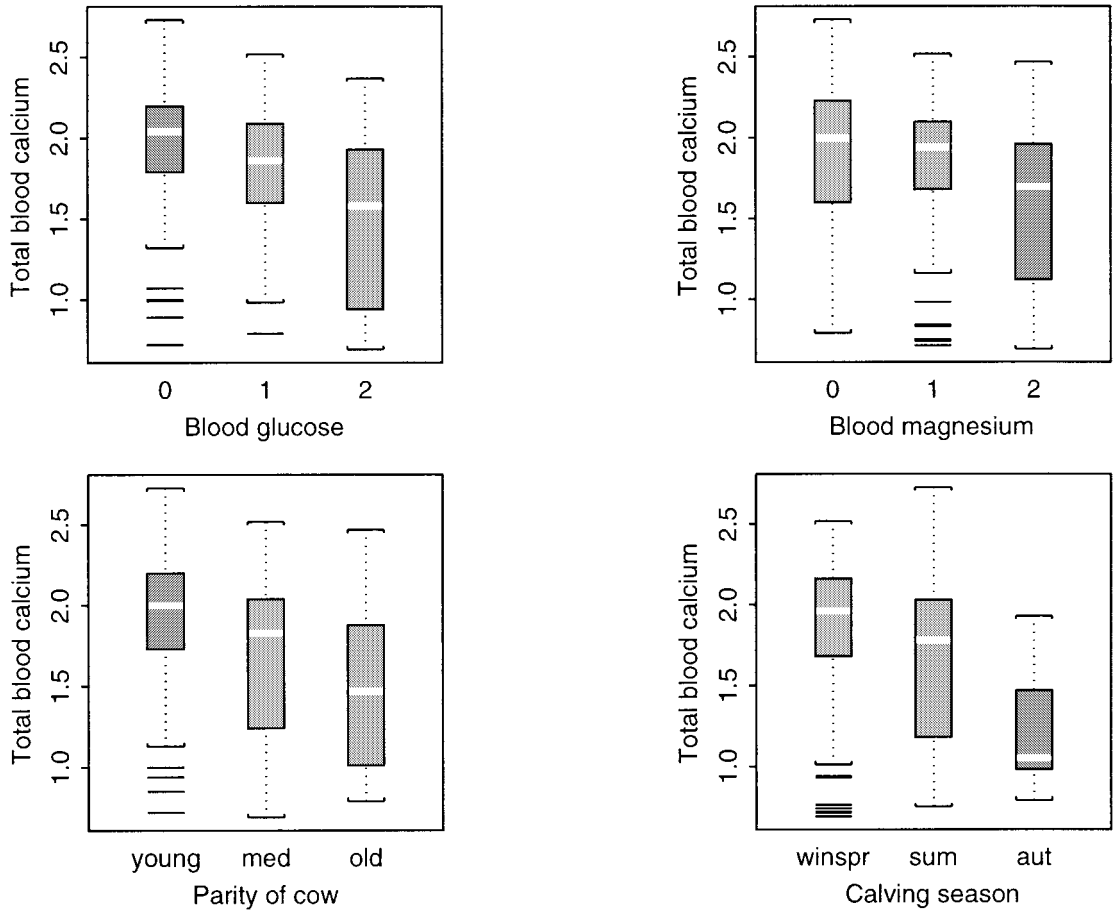

Figure 4. Upper row: The association between blood calcium (abscissa, 1/3-quartiles) and blood glucose (left) and blood magnesiuim (right). Below: The association between parity of cows and blood calcium around calving (young = parity 2+3), medium = parity 4+5, old = parity 6–10), the connection between calving time (season of the year) and blood calcium. win(ter)+spr(ing) = February, March, Arpil, n = 120; sum(mer) = June, July, August, n = 71; aut(umn) = September, n = 9. Explanation to box plots: white area in boxes represents median value, shaded box represents 50% of observations, i.e., lower limit of second quartile and upper limit of third quartile, brackets represent variation range of observations excluding extreme values (outliers) that are marked as 'loose bars' (outside brackets).

Table 2. Parameters in the final mixed effect linear model with an additive random effect of farm for interpretation of blood calcium levels, based on background knowledge of cows and conditions and clinical chemical analyses of blood. The model explains approximately 75% of the total variability. Fixed effects estimates in the mixed model.

Parameter	Estimate	Std. Error	*P*-value
Intercept	–4.837	0.918	<0.001
K	0.206	0.036	<0.001
Na	0.034	0.006	<0.001
Glucose	–0.044	0.009	<0.001
Lactate	–0.044	0.010	<0.001
Inorganic P	0.169	0.135	0.211
Mg	0.593	0.268	0.027
Season 1	0.503	0.290	0.082
Season 2	1.116	0.286	<0.001
Parity	–0.691	0.200	<0.001
iP:sea 1	0.345	0.142	0.015
iP:sea 2	0.159	0.144	0.267
Mg:sea 1	–0.714	0.303	0.018
Mg:sea 2	–0.997	0.298	<0.001
iP:parity	0.392	0.147	0.007
lak:parity	0.080	0.032	0.011
Variance components estimates(s):			
		Value	Std. error
Between farm variance		0.02109	0.00810
Within-farms variance		0.04688	0.00521

lactate leads to a rapid distribution between ionized and total calcium (Blum et al., 1972; Kvart et al., 1982), indicating that the calcium-binding proteins in the blood are not saturated. Consequently, several reports reveal a fairly good correlation between ionized and total calcium in the cow irrespective of physiological state or paretic or nonparetic conditions (Blum et al., 1972; Kvart and Larsson, 1978; Kvart et al., 1982). Particularly close correlations are obtained when ionized calcium is corrected for the changes in blood pH (Lincoln and Lane, 1990), as is the case in the present study. We concluded that, unless specifically biased, total calcium was a valuable indicator of physiologically active ionized calcium and thereby of hypocalcemia and milk fever.

Both hypocalcemia and milk fever, biochemically and clinically, may be ascribed to a deficiency in available calcium (Capen and Rosol, 1989; Radostis et al., 1997). Different elements in the Ca-homeostasis have been considered as being the precipitating factor. At the moment, however, refractivity of receptors to PTH or 1,25 $(OH)_2$ D seems to be a reasonable explanation (Goff et al., 1991; Horst et al., 1994). Although plasma calcium is the primary parameter in the pathogenesis, other parameters may play subsidiary roles in the pathophysiology.

A decreased level of plasma osteocalcin has previously been demonstrated around calving (Bernardini et al., 1994; Davicco et al., 1992; Naito et al., 1990). The present study confirms a direct correlation between blood calcium and osteocalcin (Naito et al., 1990). In 1989, Quintavalla et al. described the immediate connection between clinical paretic conditions and low blood osteocalcin versus nonparetic calving cows and high plasma osteocalcin. The present study further established an interesting connection between definite hypocalcemia and bone matrix synthesis, expressed as the osteocalcin level in plasma. A total calcium level of 1.50 mmol/L may be accepted to correspond to a borderline between slight hypocalcemic and severe hypocalcemic conditions (Kvart et al., 1982; Larsson et al., 1983). The present hypocalcemic group of animals, n = 53, is then significantly lower in plasma osteocalcin compared with the normocalcemic group, n = 146, i.e., 7.4 ng/ml versus 9.8 ng/ml ($P < 0.001$). Plasma osteocalcin is a well-established indicator of bone formation (Brown et al., 1984; Mosekilde, 1989). Parturient hypocalcemia consequently seems to arrest de novo synthesis of bone instantly because the average 'half-life' of osteocalcin in plasma is 60 to 70 min (Mosekilde, 1989), and the present blood samples were taken within 12 h of calving.

Moreover, the present study confirms numerous former reports, namely that changes in plasma phosphorus parallel those of plasma calcium as parturition approaches. Low blood phosphorus has been observed in

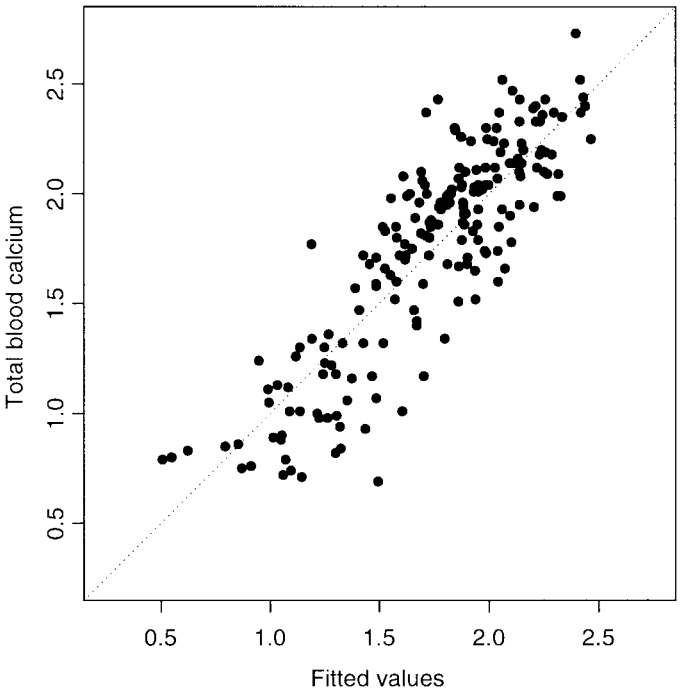

Figure 5. Fit of model based on background knowledge and clinical chemical analyses of blood for interpretation of blood calcium levels (n = 197). Fitted values obtained considering only fixed effects (abscissa) and blood calcium (ordinate). Fixed effects estimates are listed in detail in Table 2.

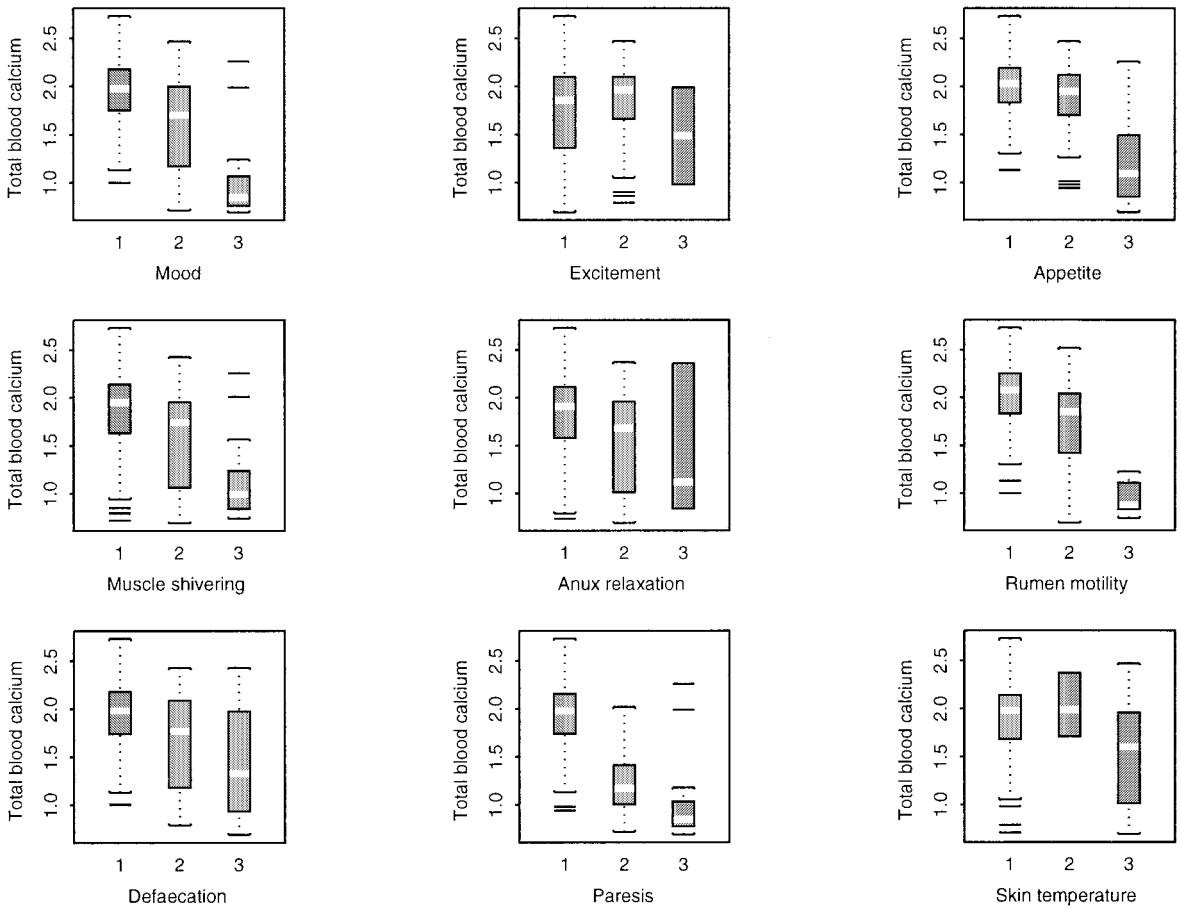

Figure 6. Distribution of blood calcium on grouped, discrete, clinical observations. For further details see text. Explanation to box plots: white area in boxes represents median value, shaded box represents 50% of observations, i.e., lower limit of second quartile and upper limit of third quartile, brackets represent variation range of observations excluding extreme values (outliers) that are marked as 'loose bars' (outside brackets).

clinical cases of parturient paresis and has even been credited with an influence on the clinical signs (Radostis et al., 1997). It has been debated whether hypophosphatamia that occurs in milk fever is the result of the hypocalcemia and recumbence rather than being a concurrent event (Daniel and Moodie, 1979; Radostis et al., 1997). The present data show close positive associations between plasma Ca and P both in paretic (categorical data paresis = 3; n = 21, $P < 0.01$) and "healthy" animals (categorical data paresis = 1; n = 151, $P < 0.001$), leaving no specific conclusions to this issue.

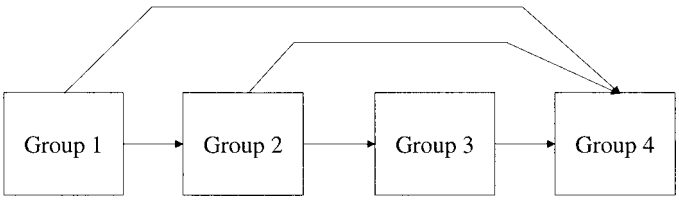

Figure 7. Path analysis null model. Group 1 = {breed, season, milking, parity}. Group 2 = {K, AP, Cl, glu, tCa, iP, blood osteocalcin, Mg, Na}. Group 3 = {rumen motility}. Group 4 = {muscle shivering, rectal temperature, appetite, paresis, defecation}.

Plasma magnesium levels normally rise at calving (Littledike et al., 1969; Phillippo et al., 1994; Radostis et al., 1997). Seen in connection with a physiological decrease in plasma Ca at calving, an inverse correlation between Ca and Mg exists, as the present study confirms. Radostis et al. (1997) reported that serum Mg is low in paretic animals (extremely low Ca levels). However, Blum et al. (1972) observed significant inverse correlations between Ca and Mg irrespective of the paretic or nonparetic condition of the cow. Our regression analysis on animal groups high and low in calcium, respectively, confirms this latter statement (details not shown). Blum et al. (1972) further observed that correlations between plasma Ca and Mg vanished immediately after curative infusion of Ca borogluco-

nate, indicating that the regulation of plasma Mg takes some time.

Potassium in blood plasma is, in the present study, closely associated with plasma calcium, i.e., hypocalcemia is accompanied by mild hypokalemia. We have no immediate explanation for this phenomenon. Most often plasma potassium concentrations are influenced by factors that alter the internal balance, i.e., the distribution of potassium across the erythrocyte membrane. The blood acid-base balance may trigger this exchange, and hypokalemia is in this respect connected with systemic alkalosis because K^+ and H^+ may exchange across the erythrocyte membrane (Carlson, 1989) or the renal tubular membranes to regulate plasma pH. Blood pH is maintained within a very narrow interval. We have observed no specific increase in blood pH around calving in other intensive studies (data not published). But it might be speculated that the observed stable pH around calving in some instances may be due to the sacrifice of plasma K^+.

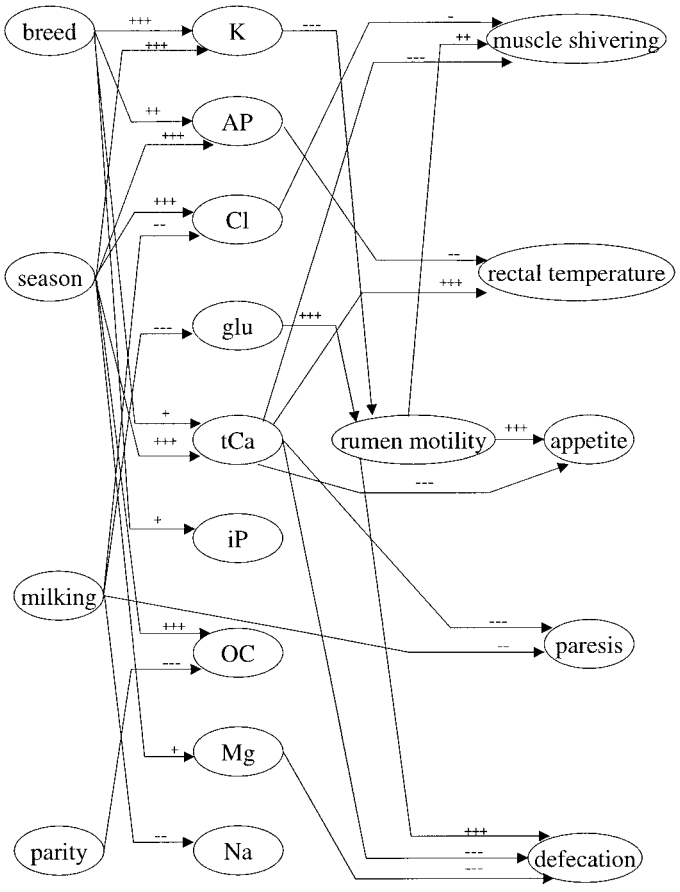

Figure 8. Path analysis diagram connecting background information of the cows, blood parameters and clinical observations at calving time, n = 197. + designates a straightforward relationship, while – designates an inverse relationship, where + = $0.10 < P < 0.05$; ++ = $0.05 < P < 0.01$; +++ = $P < 0.01$; analogue terminology for inverse relations. Herd effects have been included as baseline fixed effects. Note: Clinical (discrete) observations are characterized by 1 for normal conditions and 2 for abnormal conditions. Season has been coded with three levels, and symbols on the related arrows denote only the size of the effects. Milking: 2 milking before blood sample, 1 otherwise.

A negative association between blood calcium and glucose in parturient cows has been observed (Blum et al., 1972; Littledike et al., 1969). The rise in blood glucose during hypocalcemia has been interpreted as a result of a concomitant decrease in insulin (Blum et al., 1973; Littledike et al., 1968). Experimental infusion of calcium in hypocalcemic cows has further confirmed this by increasing the plasma level of insulin (Blum et al., 1973), suggesting that physiological concentrations of calcium are required for glucose stimulation of insulin secretion from β-cells in the pancreas (Capen and Rosol, 1989).

A grouping of the present data according to the discrete clinical observation paresis showed mean lactate values of 1.9, 2.7, and 3.7 mmol/L, respectively, for the three categories. The overall negative association between blood lactate and calcium was evidently connected with occlusion of blood vessels and subsequent anaerobic conditions in the tissues in situations during which there was only scarce muscle tonus and recumbence. Isolated correlations of sample results supported this: when total Ca > 1.50 mmol/L (n = 146), no tendencies were apparent, whereas when total Ca < 1.50 mmol/L (n = 54), a negative association between calcium and lactate was apparent ($P < 0.05$).

The present observation of decreasing blood calcium as a function of parity is in agreement with the fact that higher incidences of milk fever occur with increasing parity. Numerous reports support this association (e.g., Bendixen et al., 1987; Kusumanti et al., 1993). Moreover, Oetzel (1991), in a meta-analysis of nutritional risk factors in milk fever, found parity to be important in predicting the incidence rate, just as odds ratio analyses revealed an ever-increasing incidence rate by lactation number.

The present study confirms a seasonal influence on blood calcium content at calving time. Several other studies have found a similar connection. However, season is likely to be confounded with other impact factors such as housing, grazing, and exercise (the farm effect) (Houe et al., 1999).

Milk fever cases diagnosed by the veterinarians (n = 53) were significantly lower in blood calcium 1.17 ± 0.82 (2× SD) compared with "healthy" animals, 1.95 ± 0.62 (2× SD) mmol/L (n = 147), $P < 0.001$. The distribution is fairly identical with the biphasic distribution of blood calcium shown in Figure 2. Only in very few cases, could serious discrepancies between blood calcium and diagnosis be ascribed to misdiagnoses. However, the

consequence of a prudent milk fever diagnosis, i.e., an acute treatment against hypocalcemia (infusion of Caborogluconate), is generally very uncomplicated and in most cases preferable while the veterinarian is present compared with the consequences of negligence.

Several indicators used in the diagnosis of milk fever and hypocalcemia are closely associated with the calcium level in the blood, whereas other clinical observations used in this study seem to be of inferior value as single predictors. Rectal temperature, 'mood,' appetite, muscle shivering, rumen motility, and paresis are-as isolated observations-fairly predictive of blood calcium status. However, in a predictive model as the present, interrelations between factors are taken into account and eliminated, eventually resulting in surprising effects. Rumen motility especially has been confirmed experimentally to be very sensitive to the blood plasma level of ionized calcium (Jørgensen et al., 1998). Nevertheless, rumen motility will certainly—from "a cow side view"—be affected by and confounded with appetite (fractional rumen fill) and structure of ingested material (Nørgaard, 1989).

A path analysis model based on easily accessible background knowledge, blood analyses, and clinical observations will hardly satisfy practical veterinary diagnosis shaping. Our choice of clinical parameters may further have been insufficient to establish a descriptive connection. Additional parameters like heart rate and respiration rate could possibly have strengthened the prediction potential of the model. On the other hand, the veterinary diagnosis is not a computerized model of discrete indicators but rather an experienced synthesis of clinical observations, a judgement based on an accumulation of many detailed observations. The presented path analysis diagram may in this respect give hints to valuable connections between available information and observations.

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