WHEAT PASTURE POISONING. II. TISSUE COMPOSITION OF CATTLE GRAZING CEREAL FORAGES AND RELATED TO TETANY

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Summary

Thirty-two aged Hereford and Angus cows (avg 10.2 yr old) grazed mixed cereal (wheat and rye) pastures from December 6, 1979 to April 23, 1980 (138 d). On d 104 (March 19), five cows developed tetany. On the day of tetany, the mean plasma Ca of the cows with tetany was 4.1 mg/dl. Cows with tetany had plasma Mg concentrations of 1.7 mg/dl on the day of tetany, but 15 d before tetany and 6 d post-tetany, levels were lower (1.2 mg/dl). Cows with tetany had large increases in plasma parathyroid hormone (PTH) as well as vitamin 1, 25 dihydroxy D3 [1,25(OH)2D], plasma glucose, lactic acid and β-hydroxybutyric acid. Plasma K of these cows followed a similar trend as plant K, except it was not as pronounced. Plasma inorganic P of these same cows was exceedingly low, <3 mg/dl several times, although the forage P did not indicate a P deficiency. Levels of plasma hydroxyproline in these cows indicated that bone resorption may have been low during most of the grazing season, even though levels of plasma hydroxyproline were initially high and high subsequent to tetany. High levels of plasma glucose and lactic acid were associated with the time of tetany. Apparently, cows developed inappetence around the time of tetany, as suggested by the high levels of plasma β-hydroxybutyric acid. Cows with wheat pasture poisoning appeared to respond to the severe hypocalcemia with large increases in plasma 1,25(OH)2D. Plasma 1,25(OH)2D changes were similar to plasma hydroxyproline changes (and possible bone resorption rates) and seemed inversely proportional to plasma Ca and directly proportional to plasma PTH concentrations. Generally, cerebrospinal fluid (CSF) composition was not affected as much as plasma composition, except for the low levels of CSF Ca. These data indicate that a metabolic deficiency of Ca (hypocalcemia) was the cause of tetany.

(Key Words: Wheat Pasture Poisoning, Calcium Tetany, Plasma Composition, Beef Cattle.)

Introduction

When mature beef cows graze on winter wheat and other cereal grains, they often develop a condition known as wheat pasture poisoning. "Wheat pasture poisoning symptoms begin with undue excitement, incoordination and loss of appetite. As the condition progresses, viciousness, staggering and falling develops. Nervousness becomes more apparent with muscular twitching, particularly of the extremities. The animal has an anxious expression and may grind its teeth and salivate profusely. The third eyelid protrudes or flickers as is seen in tetanus. General tetanic contractions of the muscles follow until the animal nears a state of prostration; however, a sudden noise or merely touching the animal will cause a reflex response. Next to develop are labored breathing..."
and a pounding heart, followed by a comatose condition. If left untreated, convulsions with periods of relaxation will be seen, which terminate in death. Six to 10 h usually are required from the time the first symptoms develop until the animal passes into the comatose condition. If treatment is not initiated before coma, there is little chance of recovery (Crookshank and Sims, 1955).

Death losses from animals grazing wheat pastures may be as high as 20% on some pastures and 2 to 3% are common (Stewart et al., 1981). Because wheat pasture poisoning occurs sporadically and is influenced by multiple factors, it is difficult to evaluate. The purpose of this study was to periodically sample the tissues of mature beef cattle grazing cereal pastures and to relate these data to the occurrence of tetany.

Experimental Procedure

Details concerning the procedures for forage sampling and analysis have been described earlier (Bohman et al., 1983). Thirty-two aged Angus and Hereford cows (avg age 10.2 yr) were obtained from the Meat Animal Research Center at Clay Center, Nebraska, and transported to El Reno, Oklahoma, on December 4, 1979. Two days later, they were weighed and allocated to four approximately equal groups according to breed, weight and age. These groups were each allocated at random to one of the following treatments: (1) control, 1.1 kg cottonseed hulls (IFN 1-01-599; CSH); (2) 1.1 kg CSH plus 220 g potassium chloride (KCl); (3) 1.1 kg CSH plus 50 g magnesium oxide (MgO) and (4) 1.1 kg CSH plus 125 g calcium carbonate (CaCO3). During the first week, they were maintained on grass hay (IFN 1-00-703) and CSH; thereafter, they grazed cereal forage in three contiguous pastures that were composed of a mixture of equal parts of wheat (Triticum aestivum, IFN 2-05-176) and rye (Secali cereali, IFN 2-04-013) forage until April 22, 1980. Each treatment was represented in each pasture as nearly equal as possible. Five additional cows (total) were added to the groups April 1 to utilize the extra forage available at this time. Cows were gathered daily, placed in individual stalls and fed the supplements (treatments).

Jugular blood samples were taken three times in December, bimonthly in January and February and weekly during March and April before feeding the supplements. Initial and terminal body weights were taken after a 16-h period in which no feed or water was consumed. Feed and water were available before determination of body weight at other times. Cerebrospinal fluid (CSF) samples were taken initially and at 6, 11, 15, 18 and 20 wk of the study from the lumbosacral space via needle puncture. Blood and CSF samples were taken and immediately placed on ice and transported to the laboratory. At the laboratory, the packed cell volume (PCV) was determined, plasma separated and both the plasma and CSF stored at −10°C until analysis. The following analyses were conducted on the plasma: alkaline phosphatase (Marsh et al., 1959), glucose (Hoffman, 1937), blood urea nitrogen (BUN; Marsh et al., 1965), lactic acid (Apstein et al., 1970), inorganic P (Fiske and Subbarow, 1925), hydroxyproline (Bannister and Burns, 1970) and β-hydroxybutyric acid (Zivin and Snarr, 1973). These methods were adapted for AutoAnalyzer techniques (Technicon®). In addition, parathyroid hormone (PTH) was determined by radioimmunoassay (Berson et al., 1963; Mayer and Hurst, 1978) and Ca, Mg and K were determined by atomic absorption spectrophotometry. Plasma 1,25(OH)2D concentrations were also determined (Horst et al., 1981). Calcium, Mg, K, glucose, pH, urea nitrogen (UN) and lactic acid were determined on the CSF. The data was analyzed according to analysis of variance procedures described by Nie et al. (1975) using a Cyber 730 computer system.

Results and Discussion

On March 19, 104 d after the initial sampling, five cows developed tetany. These cows were treated IV with 500 ml Ca-Mg gluconate solution7. One of these cows calved 10 d before tetany and the remaining cows calved within 15 d after tetany (avg 4.6 d). Three of the five cows that developed tetany relapsed within 2 d and required additional treatment with Ca-Mg solution. One cow attempted to calve during her third relapse and died. The other cows

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8Cal-Dextro #2, Fort Dodge Laboratory.
Figure 1. Plasma Ca, Mg and PTH concentrations of five cows that developed tetany.
recovered soon after treatment with the Ca-Mg solution. Some cows in all experimental groups (one-MgO, one-KCl, three controls), except for those receiving CaCO₃, developed tetany.

The plasma Ca, Mg and PTH concentrations of the five cows that developed tetany are shown in figure 1. Low plasma Ca initially was present in all five cows (4.4 to 7.6 mg/dl). All five cows had their greatest plasma Ca (8.6 to 9.6 mg/dl) concentration on d 89 or 96. Plasma Ca then decreased precipitously to exceedingly low levels culminating in tetany (3.0 to 5.3 mg/dl) on d 105. After one or more treatments with the Ca-Mg solution, plasma Ca concentration increased to previous levels and stabilized at near normal concentrations. Normal plasma Ca levels are 9 to 12 mg/dl (Maynard et al., 1979). Individual plasma Ca concentrations rarely exceeded 9 mg/dl while the cows were grazing cereal pasture, and the mean plasma concentrations of each treatment group were always below 9 mg/dl.

Mean plasma Mg exceeded 2 mg/dl the first week that the cows were on cereal forage. For most of the tetany cows, the levels gradually decreased until tetany. About the time of tetany, the plasma Mg remained the same or increased (1.72 to 2.45 mg/dl). The plasma Mg in two cows dropped to .8 mg/dl 6 d after tetany, while in two levels remained above 1 mg/dl. Only one cow (No. 8) had both low concentrations of plasma Ca (3.5 mg/dl) and Mg (.5 mg/dl) at tetany. Although plasma Ca was at concentrations often associated with tetany in all cows, only in cow No. 8 was the plasma Mg at concentrations usually associated with tetany. This cow (No. 8) was more hyperirritable as assessed visually than the other four cows with tetany. Plasma PTH concentrations increased 8 d before tetany in cows that developed tetany. The increases in plasma PTH began to increase before plasma Ca decreased. Apparently, the target tissues (bone and kidney) were either not responsive to the elevated PTH levels or the increases in PTH were not great enough or prolonged enough to prevent hypocalcemia and tetany in these cows. The changes in mineral and hormonal constituents of cows with tetany more closely resembled those changes seen in cows with milk fever than those seen in cows with conventional grass tetany. However, cows that develop milk fever usually do so within 24 h of parturition (Littledike et al., 1981), but in the present study, the initial signs of tetany were dissociated from the time of parturition by at least 3 d and up to 15 d. Wheat pasture poisoning has been used as a synonym for grass tetany, grass staggers, hypomagnesemia, winter tetany and lactation tetany (Fontenot, 1980); however, the present data strongly suggest that wheat pasture poisoning is different metabolically and, to a certain extent, symptomatically from these diseases. Earlier, Horn (1983) indicated that cattle that had died or were afflicted with wheat pasture poisoning were more prone to be hypocalcemic rather than hypomagnesemic. The current data confirms and extends these earlier observations.

Dietary supplements had no significant effect on animal tissue composition when measured. Cattle were fed the supplements early each morning, then allowed to graze the cereal pastures. The cereal forage was very succulent and palatable, and the cows were reluctant to consume the cottonseed hulls that were the carrier for the mineral supplements. Thus, only 47% of the supplement offered was consumed. The daily supplement consumption was similar for all treatments. The average mineral intake was 95 g KCl, 63 g CaCO₃ and 23 g MgO·cow⁻¹·d⁻¹ for the various groups. The supplemental intake of Ca and Mg was approximately equal to the National Research Council recommended levels for pregnant beef cows. However, the supplemental K only increased the K intake by 12 to 16% because the forage K ranged between 2.5 to 5% of the feed intake (Bohman et al., 1983). This low level of supplemental K was not reflected in plasma K concentrations. Supplemental feeding could not be shown to influence plasma constituents in any of the groups of cows.

A comparison of plasma Mg and Ca concentrations of cows that developed tetany (tetany cows) and cows that did not (nontetany cows) is shown in figure 2. Plasma Ca concentrations did not differ (P<.05) between the two groups of cows until the weekly sample obtained the day before tetany. At this time, the nontetany cows had plasma Ca concentrations of 6.7 mg/dl, while the tetany cows had plasma Ca concentrations of 5.7 mg/dl. The tetany cows had plasma Ca concentrations of 4.1 ± 1.1 mg/dl on the day of tetany. No sample was taken from nontetany cows on this day. Six days after tetany, the plasma Ca was 6.8 mg/dl in nontetany cows and 7.9 mg/dl in the tetany animals (P<.025). Some of the tetany cows relapsed and were treated for several days with
a Ca-Mg gluconate solution during the 6-d period following tetany.

Except near the beginning and the end of the experiment, mean plasma Mg concentrations of the tetany cows were consistently lower than those of the nontetany cows. The lowest plasma Mg concentrations occurred 15 d (February 19) before and 6 d (March 25) after tetany (P<.05). Although the tetany cows were hypomagnesemic (1.18 and 1.20 mg/dl, respectively) at these times, the degree of hypomagnesemia would not be considered severe enough to make the cows tetany-prone (Grunes et al., 1970). These data suggest that the hypocalcemia was primarily responsible for the tetany and that the hypomagnesemia, which probably contributes to the clinical signs, was not in the tetany-prone range during development of the clinical signs in four of the five cows developing tetany.

The plasma PTH of all the cows (figure 3) was relatively high at the beginning of the study, a time when the plasma Ca was low, but it then decreased until d 89 (March 4). The plasma PTH concentrations of both groups of cows then increased for a 2-wk period, peaking at tetany. Although the tetany cows appeared to have lower levels of plasma PTH during the entire study, the differences for any sampling period were not significant statistically. Contrary to expectations, the level of PTH increased (at least 8 d) before a detectable decrease in plasma Ca. Subsequent to tetany, a secondary peak of plasma PTH occurred and high plasma PTH concentrations were maintained in the nontetany cows. In general, plasma PTH increased when plasma Ca was low and decreased when plasma Ca was high.

Plasma K concentrations were highest at the beginning of the sampling period. Plasma K then decreased to lower levels during January and February, then increased toward the time of tetany and decreased thereafter until the end of the study. The tetany cows had lower levels of plasma K initially and at 75 d (P<.05). At other times, the levels were similar between the two groups of cows (figure 4). The forage levels of K were high (above 4% dry weight basis) initially and at the time of tetany (Bohman et al., 1983) and plasma K concentrations
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Figure 4. Plasma K concentration in tetany and nontetany cows. Plasma values differ between tetany and nontetany cattle (*P<.05). Each value is the mean ± SE.

Figure 5. Plasma glucose concentration in tetany and nontetany cows. Each value is the mean ± SE.

tended to reflect forage K concentrations throughout the experiment. The plasma K concentrations were in the normal range for cows (Crookshank and Sims, 1955; Scotto et al., 1972) despite the high K intake. Scotto et al. (1972) have shown that cattle can quickly eliminate excess plasma K.

Plasma glucose concentrations gradually increased in both groups of cows throughout the sampling period (figure 5). Although tetany cows consistently had lower mean concentrations of plasma glucose than the nontetany cows before tetany, plasma glucose was lower (P<.05) only once (d 8). At tetany, plasma glucose was approximately 50% higher in the tetany cows. A similar degree of hyperglycemia is seen in hypocalcemic cows with milk fever (Littledike et al., 1981); however, a paresis is present in cows with milk fever rather than the tetany seen in the cows with wheat pasture poisoning.

The pattern of changes in plasma lactic acid was similar to that of plasma glucose, although generally, the lactic acid differences were significant statistically (figure 6). During tetany, liver and muscle glycogenolysis and muscle anaerobic glycolysis would both be accelerated in response to the high metabolic rate of the muscle. The changes in plasma glucose and lactic acid are similar to the changes seen with epilepsy in man (Orringer et al., 1977). In both instances, the plasma glucose is high (145 to 160 mg/dl), potassium is normal and unmodified and plasma lactate is elevated.

The initial low plasma BUN reflects the low N intake of the cows fed grass hay and cottonseed hulls during the first 8 d of the experiment (figure 7). After the cattle were placed on wheat forage, the BUN rose within one sampling period to 20 to 25 mg/dl and was maintained at these levels until the first week in March (d 89). From that time, the BUN of both groups increased, but the increase in BUN of the tetany cows tended to be greater than that of the nontetany cows. Plasma BUN concentrations were higher (P<.05) in tetany cows than in the nontetany group on December 20 (d 14) and March 11, 18 and 25 (d 97, 104 and 111, respectively). After April 8 (d 124), as the wheat plant matured, the plasma BUN concen-
Mean plasma hydroxyproline concentrations were 1.4 μg/ml or more in both groups of cows at the beginning of the experiment while the cows were still consuming hay. However, plasma hydroxyproline decreased steadily in both groups of cows until d 89, and it then increased steadily until the end of the study (figure 8). At tetany, the plasma hydroxyproline in the tetany cows rose sharply and continued to rise until the end of the study, except for d 110. On d 8 (December 13), plasma hydroxyproline was significantly lower in the tetany cows; however, plasma hydroxyproline concentrations were higher (P<.05) in tetany cows than nontetany cows during April (d 119 to 138). If plasma hydroxyproline is a true indicator of the level of bone resorption that existed, then bone Ca was more important in maintaining Ca homeostasis at the beginning and the end of the experiment than during the several weeks before tetany. Thus, bone Ca may have played a role in the maintenance of plasma Ca when levels of N, lipids, K and aconitic acid were high in the forage and would tend to limit the uptake of both dietary Ca and Mg.

After tetany, the levels of plasma hydroxyproline increased in all cows. Because tetany cows calved an average of 2 wk before the nontetany cows, the higher hydroxyproline levels of this group during the post-tetany period may be a reflection of the increased demands of Ca due to lactation and mobilization of bone Ca to meet this demand.

Plasma β-hydroxybutyric acid was lowest near the beginning of the study and steadily increased until the time of tetany (figure 9). The increase in plasma β-hydroxybutyric acid tended to be greater in the tetany cows throughout the sampling period than in the nontetany cows. This ketone body reflects the catabolism of body fat and an increase in β-hydroxybutyric acid may reflect reduced consumption of forage. A large increase in plasma β-hydroxybutyric acid concentrations was associated with
the period of severe hypocalcemia tetany. This increase in plasma $\beta$-hydroxybutyric acid persisted for about 1 wk and then decreased to
the end of the study. These differences in plasma $\beta$-hydroxybutyric acid between the
tetany and nontetany cows were statistically
different ($P<.05$) on d 42, 61 and 110.

The PCV was relatively stable but high (44
97 to 51%) in all cows until tetany, when it
increased further (56%). Subsequent to the
tetany period, the PCV of the tetany cows
dropped and was significantly lower than for
the nontetany cows during the month of April
(43 vs 52%). The high level of PCV during most
of the study and at tetany suggests that the
cows were dehydrated, even though water was
available in each pasture (8 ha) at all times and
the water content of the forage was high.
Plasma alkaline phosphatase did not follow any
apparent pattern, nor was it modified signifi-
cantly at tetany. Plasma P concentration was in
the normal range initially, but decreased to low
levels (<3 mg/dl) when the cows were placed
on pasture (figure 10). The levels gradually
increased during January (d 28 and 42), then
decreased again during February. On d 75, the
plasma P concentrations were particularly low
in the tetany cows. However, at tetany, plasma
P levels were again normal, but soon decreased
again to lower concentrations. Plasma P reflected

Figure 8. Plasma hydroxyproline in tetany and
nontetany cows. Statistical significance between plas-
ma values of tetany and nontetany cattle is indicated
as follows: **$P<.01$; *$P<.05$; *$P<.1$. Each value is
the mean ± SE.

Figure 9. Plasma $\beta$-hydroxybutyric acid concentra-
tions in tetany and nontetany cows. Statistical signifi-
cance between plasma values of tetany and nontetany
cattle is indicated as follows: **$P<.01$; *$P<.05$; *$P<.1$.
Each value is the mean ± SE.

Figure 10. Plasma P concentration in tetany and
nontetany cows. Each value is the mean ± SE.
plant P content after the first month of the study. Even though plant P concentrations were not deficient (NRC, 1976), severe hypophosphatemia was present in the cows at several periods during the study (d 14, 75 and 117).

Plasma 1,25-(OH)\textsubscript{2}D concentrations in cows with tetany (figure 11) were initially quite high, but decreased to low concentrations when the cows were put on pasture. At the time of tetany and severe hypocalcemia, 1,25-(OH)\textsubscript{2}D concentrations markedly increased and then returned to the normal range. The plasma levels of 1,25-(OH)\textsubscript{2}D and PTH paralleled each other at this time, further substantiating the role of hypocalcemia in inducing tetany.

The concentrations of several constituents of the CSF of the cows are shown in table 1. The CSF Ca was initially low and appeared to increase with subsequent samples. The tetany cows had higher levels of CSF UN at tetany (wk 15; P<.05) than the other cows. The values of the other CSF constituents were not different between the two groups of cows at the six sampling periods. Most constituents of the CSF measured were lower initially than during subsequent periods.

With the high levels of plasma PTH and 1,25-(OH)\textsubscript{2}D and the extremely low level of

![Figure 11. Plasma vitamin 1,25(OH)\textsubscript{2}D concentration in tetany cows. Each value is the mean ± SE.](image)

### TABLE 1: COMPOSITION OF CEREBROSPINAL FLUID OF CALF GRAZING CEREAL FORAGE

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*Each value in the table is the mean ± SE.*

**P<.01.**
plasma Ca at tetany, the tetany was caused by hypocalcemia and not hypomagnesemia. Although hypomagnesemia did occur prior to and after tetany, the levels were higher than those usually associated with tetany. Undoubtedly, Ca and Mg metabolism are interrelated in this malady. Contreras et al. (1982) demonstrated that hypomagnesemia reduced the mobilization rate of Ca in experimental ruminants; hence, the hypomagnesemia condition before tetany may have made the cows more prone to hypocalcemia and, consequently, tetany.

**Literature Cited**


