A Rational Basis for the Prevention of Parturient Paresis

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Although several regimes have been reported which successfully reduce the incidence of parturient paresis, a program which is both universally acceptable and successful has not yet emerged. In this paper fundamental aspects of the syndrome will be discussed aiming to improve management practices through a better understanding of the basic processes involved in the disease. Since control by restriction of the prepartal dietary calcium intake does not appear to have attracted widespread attention as yet, the rationale for its use will be emphasized. It is hoped that this will encourage practitioners to trials of this conservative method which is fundamentally sound and free of hazards. It is recognized that restriction of the prepartal calcium intake will not be practical under certain systems of management; e.g., where cows are pastured during the prepartal period and where only leguminous roughages are available. Therefore, alternate programs will be discussed which may prove useful in those instances.

Fundamental Aspects of Parturient Hypocalcemia

Since the clinical signs of parturient paresis are related to development of hypocalcemia (20,28,30), attempts to prevent the disease have naturally been directed toward mitigation of the hypocalcemia which develops in association with parturition. Nearly all cows exhibit a decline in plasma calcium concentration during the parturient period. The extent of this decline varies among cows (Fig. 1) and usually only those with plasma calcium concentrations below 5mg/100 ml develop paresis (20,28,30). In most instances the initiation of lactation appears to be the precipitating factor in the development of the hypocalcemia. Milk contains about 1.3 gm of calcium per liter and the concentration in colostrum is even higher, being approximately twofold greater. If the sudden loss of blood calcium into the milk is not offset by a rapid and sustained increased inflow (either from gut or bone) of calcium into the blood, then hypocalcemia will ensue. Although cessation of the fetal calcium drain and a temporary reduction in outflows to bone (accretion) and feces (endo-

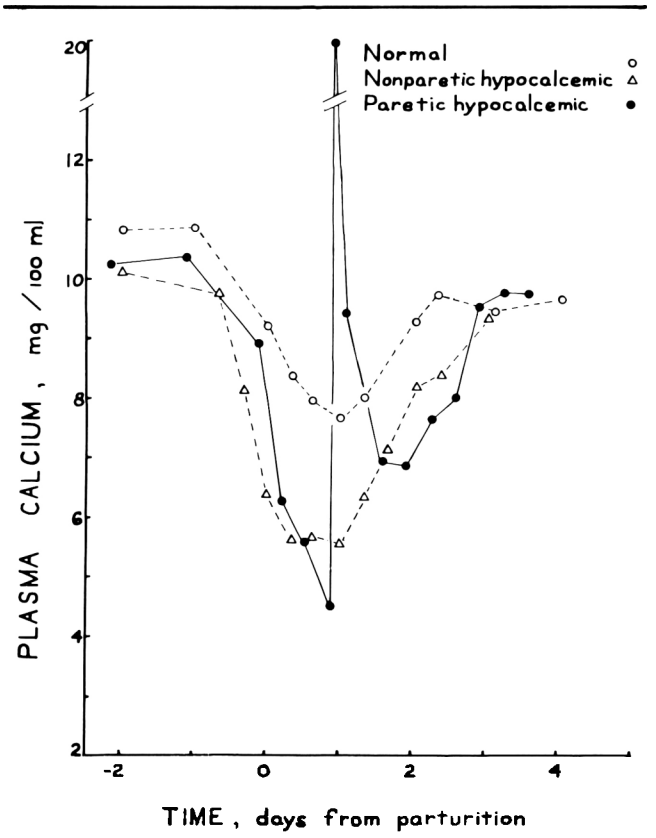

Fig. 1 – Varying Degrees of Hypocalcemia in Parturient Cows. These observations suggest that some of the processes responsible for the development of hypocalcemia in the paretic cows are at work in apparently normal cows. Note that the time of onset and recession of the hypocalcemia is similar in all 3 groups. Development of hypocalcemia is apparently due to an imbalance between the calcium flows into and out of the blood; i.e., outflow exceeds inflow. The initiation of lactation is the factor responsible for the increase of calcium outflow. The severity of the hypocalcemia is dependent upon the magnitude of the difference between outflow and inflow. Any temporary interruption of an inflow (e.g., calcium absorption from the gut) would contribute to this imbalance and augment the development of hypocalcemia. Note that paresis developed only after the plasma calcium concentration fell below 5 mg/100 ml. Therapeutic intervention (intravenous calcium administration) was required to restore blood calcium and alleviate the clinical signs. (Reproduced from G. P. Mayer et al, Clin. Orthop. Related Res., No. 62, 79-94, 1969, through the courtesy of J. B. Lippincott Company.)

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genous) may aid homeostatic adjustment, the magnitude of these changes is insufficient to totally compensate for the lactational flow of calcium (39). The initial adjustment to the lactational calcium drain appeared to be an increase in calcium absorption in mature cows on a high calcium diet (39). Calcium absorption was the dominant inflow of calcium into the blood during the last four weeks of gestation and for two weeks postpartum after which time release of calcium from bone increased (39). The emphasis which these findings place on the role of calcium absorption in the homeostatic adjustment to the lactational calcium loss is consistent with the importance of this flow in calcium homeostasis of lactating cows as demonstrated previously by other workers. Hypocalcemia can be induced in lactating cows by either fasting (40) or by the injection of drugs which reduce gastrointestinal motility (33) and presumably thereby decrease calcium absorption.

Determinants for the Development of Hypocalcemia

Only approximately 5 to 10% of dairy cows fail the homeostatic test at the onset of lactation and develop a severe enough hypocalcemia to induce clinical signs. How do these cows differ from the normally calving cows? A number of factors which may be involved will be discussed.

Quantity of Milk Calcium

The decline of plasma calcium concentration of mastectomized parturient cows is minimal and of short duration (36). This indicates the essentiality of milk calcium loss to the development of hypocalcemia. However, as a group, parturient paresis cows do not appear to suffer a greater loss of calcium into the milk than nonaffected cows (10,23). Thus, it appears that while the lactational calcium drain most certainly contributes to the development of hypocalcemia, its magnitude is not the dominant factor in determining the degree of hypocalcemia at parturition.

Age

The incidence of parturient paresis is age related, rarely occurring prior to the third lactation (15,24). The resistance of young cows is probably due to a more rapid bone calcium turnover (21), a lower rate of milk production, and less gastrointestinal dysfunction at parturition (32).

Parathyroid Hormone

Since parathyroid hormone acts to increase blood calcium concentration, it was initially thought that the hypocalcemia of this syndrome might be due to a parathyroid insufficiency (14). This has not been substantiated by either evaluation of parathyroid gland ultrastructure (5) or measurements of plasma parathyroid hormone concentration (31) in affected and control cows. The parathyroid response to hypocalcemia in cows which developed parturient paresis was equal to that of nonaffected parturient cows (31). However, a hyperactive parathyroid response in some cows appeared to exert a mitigating influence on the development of hypocalcemia (31).

Calcitonin

Although the discovery of calcitonin, a hypocalcemic hormone secreted by thyroid parafollicular cells, has stimulated several investigations of its possible relation to the development of parturient hypocalcemia, an etiologic role for it in this syndrome remains to be established. Capen and Young (6) were the first to present evidence suggesting a possible etiological role for calcitonin in this syndrome. They found that thyroid glands from cows with parturient paresis contained less extractable calcitonin than control parturient cows. They interpreted the depleted thyroids as an indication of an abrupt release of calcitonin near the time of parturition and suggested that this release might contribute to the development of hypocalcemia. The extremely low rates of bone resorption observed in cows during the prepartal and immediate postpartal periods (39) raised doubts with regard to a significant hypocalcemic effect of calcitonin at parturition since the hormone lowers blood calcium by inhibiting bone resorption (17,44). Moreover, intravenous infusion of exogenous calcitonin to mature cows either immediately before or after parturition failed to induce hypocalcemia (29). Recent development of a radioimmunoassay for bovine calcitonin has permitted measurement of plasma calcitonin concentration in parturient cows. Plasma calcitonin concentration was not increased above the prepartal level during the development of hypocalcemia in cows with parturient paresis (Mayer, Blum, Deftos and Potts, unpublished data). These observations do not exclude calcitonin from an etiologic role in this syndrome. Diminished prepartal bone resorption may be the consequence of a prolonged increased calcitonin secretory rate. The interrelationship between diminished bone resorption and gastrointestinal dysfunction in the development of hypocalcemia will be discussed more explicitly below.

Gastrointestinal Function

Diminished food intake and decreased

alimentary activity which occur in mature cows at parturition are indications of altered gastrointestinal function which may contribute to the development of hypocalcemia (32,33). Moodie and Robertson (32) found that diminished food intake in old cows (four or more lactations) during the last four days prepartum preceded the development of hypocalcemia. Food intake was not reduced in young cows (less than four lactations) and a lesser decline in plasma calcium concentration was observed (32). Reduced fecal output, reduction in primary rumen movements, and loss of rumen sounds, which have been observed in both young and old cows at parturition (33), may be taken as an indication of alimentary stasis which is thought to contribute to the development of parturient hypocalcemia (33). Support for this view is found in the hypocalcemia of lactating cows which follows drug induced alimentary stasis (33). Stasis most likely contributes to a reduction in calcium absorption through a failure to move ingesta from the rumen to sites of absorption in the small intestine. Prompt evacuation of the bowel following intravenous calcium administration to cows with parturient paresis suggests that hypocalcemia itself may contribute to intestinal hypomotility and stasis. Thus it is possible that hypocalcemic induced gut stasis may exacerbate the development of hypocalcemia. Lest the reader conclude that alimentary stasis in parturient cows is due solely to hypocalcemia, it should be re-emphasized that signs of stasis (i.e., diminished food intake, decreased rumen sounds, and reduced fecal output) often precede the development of a significant degree of hypocalcemia (32,33). With usual practices of dairy cattle husbandry (i.e., high calcium prepartal diet), gastrointestinal calcium absorption is likely to be the major inflow of calcium into the blood of mature (four years of age or older) parturient cows. In view of this dependence upon calcium absorption, any interruption of normal gastrointestinal function which leads to a temporary impairment of calcium absorption could seriously jeopardize calcium homeostasis (Fig. 2).

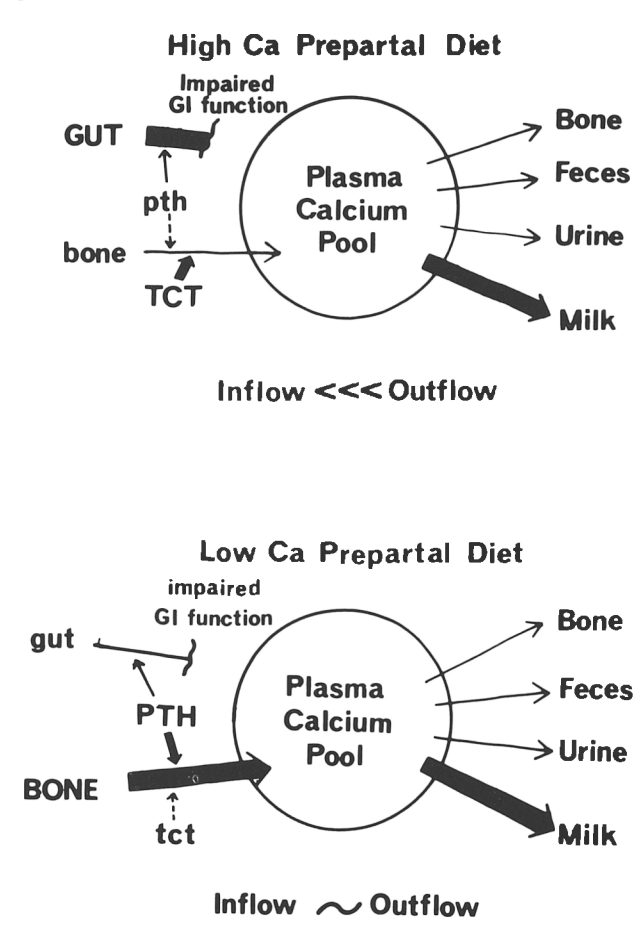

Fig 2– Possible Mechanisms Involved in the Development of Parturient Paresis. The role of gastronintestinal dysfunction in the pathogenesis of the syndrome as determined by the influence of prepartal dietary calcium intake on humoral mechanisms. Prepartal dietary calcium intake is known to influence the incidence of parturient paresis. This figure is a schematic presentation of the mechanisms which may be involved. When animals are fed a high-calcium diet, calcium absorption from the gut becomes the major inflow of calcium into plasma. Under these conditions the rate of calcium mobilization from bone (bone resorption) is minimal. This low rate of bone resorption may be related, in part, to two factors. High-calcium diets (1) suppress parathyroid activity and (2) may provoke an increased secretion of calcitonin, an inhibitor of bone resorption. Thus, it appears that the naturally high-calcium diet of the prepartum cow may render her calcium homeostatic mechanisms more dependent upon calcium absorption from the gut. Due to prolonged inhibition by calcitonin (TCT), the bone may be slow to respond to the increased concentration of parathyroid hormone (PTH) in the plasma at parturition. Under these circumstances a temporary interruption of calcium absorption through an impairment of gastrointestinal function, which may be assiciated with parturition, could seriously hamper the cow's ability to compensate for the calcium drain imposed by the onset of lactation. Consequently, if inflow of calcium into the plasma is less than the outflow, plasma calcium concentration will decline. If the hypocalcemia becomes severe (<5 mg/100 ml), paresis may develop. On the other hand, if the dietary calcium intake is restricted in the prepartum period, bone resorption may be increased due to stimulation of the parathyroid glands and a reduction in the thyrocalcitonin secretion rate. Under these circumstances, an interruption of calcium absorption at parturition is not likely to have such a pronounced adverse effect on calcium homeostasis. (Reproduced from G. P. Mayer in **Parturient**

Preventive Programs

Restriction of Prepartal Dietary Calcium

Rationale

From the foregoing, it appears that in many instances gastrointestinal function may play a determining role in the development of hypocalcemia. These instances arise when the parturient cow is dapendent almost wholly on calcium absorption for the replenishment of blood calcium

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(Fig. 2). In cows, calcium absorption increases as the dietary calcium intake is increased, and bone resorption varies in a reciprocal manner with calcium absorption (Ramberg, Mayer and Kronfeld, unpublished data). Hence, one would predict that cows on a high calcium prepartal diet would have a low rate of bone resorption with the major calcium inflow being calcium absorption. This expectation approximates the results of calcium balance and tracer kinetic studies on prepartal cows consuming an excess of dietary calcium (39). In these studies calcium absorption constituted the major inflow of calcium into the blood with very little coming from bone. Under these conditions, a temporary interruption of calcium absorption as a result of a gastrointestinal dysfunction could seriously impair the cow's ability to offset the lactational calcium loss, particularly if bone were slow to respond to the action of parathyroid hormone. Some experimental evidence suggests that the latter is a possibility. The prepartal increase in plasma calcium concentration in response to parathyroid extract administration was less than the response obtained approximately one week postpartum (Martig and Mayer, unpublished data). Furthermore, increased plasma parathyroid hormone concentration has been observed in parturient cows (31), but mobilization of calcium from bone, as determined by calcium balance and tracer kinetic studies, appears to be delayed until about 10 days postpartum (39). Since the action of calcitonin on bone is antagonistic to that of parathyroid hormone (parathyroid hormone stimulates, whereas calcitonin inhibits bone resorption) the apparent sluggish response to parathyroid hormone near parturition might arise through a prolonged exposure of the bone to the action of calcitonin during the prepartal period. Since oral calcium loading leads to an increased secretion of calcitonin (7), the high calcium prepartal diet may stimulate increased secretion of calcitonin while simultaneously depressing parathyroid hormone secretion (high calcium diets induce parathyroid atrophy [16, 42]). Both these effects may tend to diminish the response of bone in the parturient cow and render her reliant upon gut absorption of calcium.

Obviously all cows on a high calcium prepartal diet do not become severely hypocalcemic and paretic at parturition. If adequate gastrointestinal function is maintained, problems of calcium homeostasis should be minimal. Only those cows which experience impairment of gastrointestinal function are likely to encounter serious difficulty in the homeostatic adjustment to lactation. The low incidence of parturient paresis in young cows, under three years of age (15,24), may now be logically explained by their lack of complete reliance on gut calcium absorption due to a more rapid bone calcium turnover (21) and by their having less likelihood of gastrointestinal dysfunction at parturition (32).

Low calcium prepartal diets shift the dominant role in calcium homeostasis to the mobilization of bone calcium and minimize the role of calcium absorption (Fig. 2). Consonant changes in endocrine status would act to augment the dominance of bone resorption with this diet. Since low calcium diets induce parathyroid hypertrophy (42), an increased secretion of parathyroid hormone would be expected. Likewise diminished absorption of calcium should lead to a decrease in calcitonin secretion. While low calcium diets would not be expected to reduce the frequency of gastrointestinal dysfunction in parturient cows, the diminished reliance upon calcium absorption would tend to minimize the importance of gut function in calcium homeostasis of these animals.

Practical Aspects

Boda and Cole (1) were the first to demonstrate that the incidence of parturient paresis could be reduced by feeding a low calcium diet during the prepartal period (last month of gestation). Although their description of the diet as "low calcium-high phosphorus" accurately describes the relationship of the quantities of these two elements on a relative basis, it is misleading from the standpoint of the actual quantity of phosphorus present (Table 1) which is near the amount recommended by the National Research Council (37). As will be noticed upon examining Table 1, the incidence of parturient paresis relates better to the quantity of calcium in the diet than to either the Ca:P ratio or the quantity of phosphorus. In order to reduce the dietary calcium to an

TABLE 1

Relation of Prepartal Dietary Calcium to the Incidence of Parturient Paresis (1, 2)

No. of Cows	Diet gm/d Ca	Diet gm/d P	Ratio Ca/P	Parturient Paresis % Incidence
5	6	24	0.25	0
16	6	20	0.30	0
49	8.5-10	88-102	0.10	6
26	11	44	0.25	8
19	11	74	0.15	10
20	34	34	1.0	15
19	120	20	6.0	26

extremely low level, it was necessary to use a source of roughage which is not commonly or abundantly available (i.e., oat hay). This among other things has probably distracted from the desirability of the program and prevented its widespread acceptance. However, undesirable aspects of the experimental ration should not deter one from applying the principle established by these experiments (i.e., the incidence of parturient paresis can be influenced by the prepartal dietary calcium intake) particularly in view of the accumulation of previously discussed experimental data which support the fundamental soundness of this approach.

Although it is unlikely that parturient paresis can be completely abolished in a herd without resorting to extremes of dietary manipulation, restriction of the prepartal dietary calcium intake to a level approximating that recommended by the National Research Council (Table 2) can be achieved through conservative changes in management and is likely to prove beneficial. In most instances the prepartal calcium intake can be effectively reduced by avoiding the use of leguminous roughages and removal of calcium supplements from the concentrate and the free-choice minerals. Table 3 lists approximate calcium content of common cattle feedstuffs. From this it is readily apparent that roughages are of prime consideration in this program. As little as 10 lbs. of alfalfa hay will supply twice the recommended quantity of calcium for a large (700 kg) Holstein cow in the last trimester of gestation. When corn silage is available there will be no problem in reducing the calcium intake. Next in order of preference would be nonleguminous hays such as prairie or timothy hay. With these, the intake is not likely to drastically exceed the recommended allowance since 20 lbs. of either would provide only 30 gm of calcium. Although results of extensive field trials using this approach are not available, our own experiences with several problem, high incidence herds have demonstrated the merits of this approach.

TABLE 2

Approximate Calcium and Phosphorus Requirements for 1500 lb. Dairy Cow (37)

Physiological State	Dietary Requirement gm/day	
	Ca	P
Maintenance	18	18
Pregnant	34	32
Lactating, 44 lb/day*	66	54
Lactating, 88 lb/day†	130	98

*Cow producing less than 77 lb. of milk/day requires approximately 1.1 gm of Ca and 0.8 gm P in the diet for each pound of milk produced. These requirements are in addition to those of maintenance.

†For cows producing over 77 lb. of milk daily, the dietary requirement is 1.3 gm Ca and 0.9 gm P per pound of milk produced. These requirements are in addition to maintenance requirements.

TABLE 3

Approximate Mineral Content of Common Dairy Cattle Feeds (34)

	gm/lb of Feed*	
	Ca	P
Alfalfa hay	7.0	1.1
Timothy hay	1.5	0.6
Prairie hay	1.5	0.4
Corn silage	0.5	0.3
Corn	0.1	1.3
Corn & Cob meal	0.2	1.0
Milo	0.1	1.3
Oats	0.4	1.5
Wheat bran	0.6	5.9

*air dry weight

Since lactation increases the requirement for calcium (Table 2), the calcium intake should be increased postpartum. Restriction of dietary calcium intake should be confined to the dry period of the cow's lactational cycle. Thus, legumes are excellent roughages for lactating cows, being rich in calcium and other nutrients as well. If legumes are not available for the lactating cows, it will probably be necessary to include a calcium supplement (e.g., ground limestone, steamed bone meal, or dicalcium phosphate) in either the concentrate mixture or free choice mineral supplements. At this point, it may be worthwhile to suggest that more careful attention should be given to the ration of lactating cows so as to avoid greatly exceeding the recommended allowance. Long term excess dietary calcium may be detrimental. This is suggested by a high incidence of ultimobranchial body tumors, ultimobranchial hyperplasia, and osteopetrosis, a condition of increased bone density (27), in bulls over six years of age, which normally are fed a gross excess of calcium. Since ultimobranchial cells secrete calcitonin (8) which acts to inhibit bone resorption (17,44) and oral calcium loading increases the secretion of calcitonin (7), a relationship between excess dietary calcium, ultimobranchial hyperactivity, and osteopetrosis has been postulated (27). Neither ultimobranchial hyperplasia, ultimobranchial neoplasia or osteopetrosis is found in cows, probably as a consequence of the lactational outlet for the normally high dietary calcium intake. However, it may be suspected that similiar mechanisms, but of lesser extent, may be at work in cows which are fed an excess of dietary calcium the year round. Such processes would have a deleterious effect on calcium homeostasis at parturition. A recent and apparently successful

program for prevention of parturient paresis (43) provides what may be an exemplification of this point. Calcium supplement was removed from the concentrate mixture and replaced with monosodium phosphate in an attempt to provide a year round balance of the calcium and phosphorus content of the ration. In so doing, the calcium intake of the cows was reduced from 189 down to 137 gm/day. The program required 189 days to attain maximum effectiveness which persisted until 232 days after the feeding practice was discontinued. Although the success of the program was attributed to the balancing of the ration by phosphorus supplementation, the reduction in the excess of dietary calcium should not be discounted. The latent onset and delayed recession of effectiveness of this program suggests that long term dietary management may be worthwhile. The challence may be to feed adequate but not excess calcium throughout the lifetime of the cow.

In formulating recommendations, one may use tables giving average analysis for feedstuffs which are readily available (34,37). However, when access is available to the services of a feed analyst, I would encourage the use of such service. Calcium content of feeds, particularly in the case of roughages, can vary depending upon climatic conditions, soil fertility, and stage of plant maturity.

Vitamin D

The calcemic effects of vitamid D which arise from its action on gastrointestinal calcium absorption (35) and its augmentation of parathyroid hormone stimulated release of calcium from bone (13) naturally aroused interest in its potential usefulness in the prevention of parturient paresis. Although short term feeding of massive quantities of vitamin D (20 million units per day) just prior to parturition offers protection from parturient paresis (22), imprecision in predicting the exact date of calving and the danger of toxicity arising from prolonged administration (more than seven days) of the vitamin has precluded the widespread use of this regimine. Prepartal parenteral administration of vitamin D_3 has proven to be a more popular method for the control of parturient paresis (41). The dose of 10 million units is injected intramuscularly two to seven days prior to the expected calving date. If the cow hasn't calved within 10 days, she must be retreated to maintain a level of protection. Repeated injections may increase the likelihood of calcification of vital organs (i.e., heart, kidney, and arteries) particularly if the dietary calcium intake is high (38). Other workers have had little success with a 10 million unit dose for large cows (9,25). Thus with this form of control, maximum effectiveness with minimum risk is dependent upon accurate prediction of the date of parturition.

Preliminary work in Wisconsin indicates that an active metabolite of vitamin D_3, 25-hydroxycholecalciferol (25-HCC), may be useful for the prevention of parturient paresis (3). Since recent work suggests that vitamin D_3 requires conversion to 25-HCC to become biologically active (12), one would expect the onset of protection to be more rapid following administration of 25-HCC than when giving the parent vitamin. Experiments appear to support this prediction. An oral dose of 1 mg of 25-HCC was effective when administered as late as 24 hours prepartum; whereas three days were required to attain maximum protection when oral vitamin D was used. It is recommended that this dose be repeated every 48 hours until parturition occurs. Since the 25-HCC acts and is turned over more rapidly than the parent vitamin, it is hoped, but not proven, that the danger of soft tissue calcification, associated with vitamin D administration, will be lessened.

Ca:P Ratio of Diet

Much attention has been focused on dietary calcium and phosphorus in the control of parturient paresis by the work of Boda and Cole (1) who reduced the incidence of parturient paresis by feeding a "low calcium-high phosphorus diet" (Ca:P ratio = 1:3) during the last 30 days of gestation. Kendell et al. (26) found that the incidence of the syndrome was lowest at a Ca:P ratio of 2.26 and increased when the ratio was narrowed to 0.95 or widened to 4.16. Gardner (18,19) has emphasized the importance of Ca:P ratio of 2.3 in the control of this disease reporting that cases are averted at a ratio of 2.3 and that the incidence rose to 18% when a ratio of 1:8 was fed (18). Bush and Stevens (4) report cases of the disease in cows consuming a ration with a Ca:P ratio of either 3 or 1.5. Stott (43) was able to drastically reduce the incidence of the syndrome in a problem herd by balancing the calcium and phosphorus content of the ration (Ca:P ratio for average cow = 1.6). This was accomplished mainly by replacing calcium supplements in the concentrate mixture with an appropriate quantity of monosodium phosphate which was then fed to all cows the year round. From the above results, it is reasonable to conclude that factors other than the Ca:P ratio, *per se*, are likely to be involved in the prevention of parturient paresis by dietary

management. It follows that feeding at a particular Ca:P ratio is unlikely to yield uniformly successful results. As has already been emphasized, the dietary calcium intake appears to exert a greater influence on the incidence of the syndrome than either the Ca:P ratio or the phosphorus content of the ration.

Conclusions

Avoidance of excess dietary calcium, especially during the prepartal period and possibly during lactation, appears to offer a fundamentally sound approach for reduction of the incidence of parturient paresis. Providing that phosphorus intake is adequate to meet recommended requirements, emphasis should be placed on management of the calcium intake rather than Ca:P ratio of the diet. Although low calcium prepartal diets successfully reduce the incidence of the disease, some beneficial results may be obtained by partial restriction of prepartal calcium intake where extreme reduction is not possible. When it is impractical to reduce calcium intake, intramuscular administration of vitamin D_3 may be considered. However, keep in mind that high calcium diets may potentiate the calcemic effect of the vitamin and lead to soft tissue calcifications even though clinical signs of toxicity may not be apparent. A solution to this dilemma may be forthcoming providing further evaluation of 25-hydroxycholecalciferol supports the expectation that it will prove less hazardous. Due to the low incidence in young cows, the cost of the program may be reduced by restricting use of vitamin D_3 or its metabolite to cows in their third or latter lacation. Finally, in order to reduce further complications (e.g., "downers") and undue economic loss, prompt and adequate treatment should be administered to cows as soon as clinical signs of the syndrome become apparent (11).

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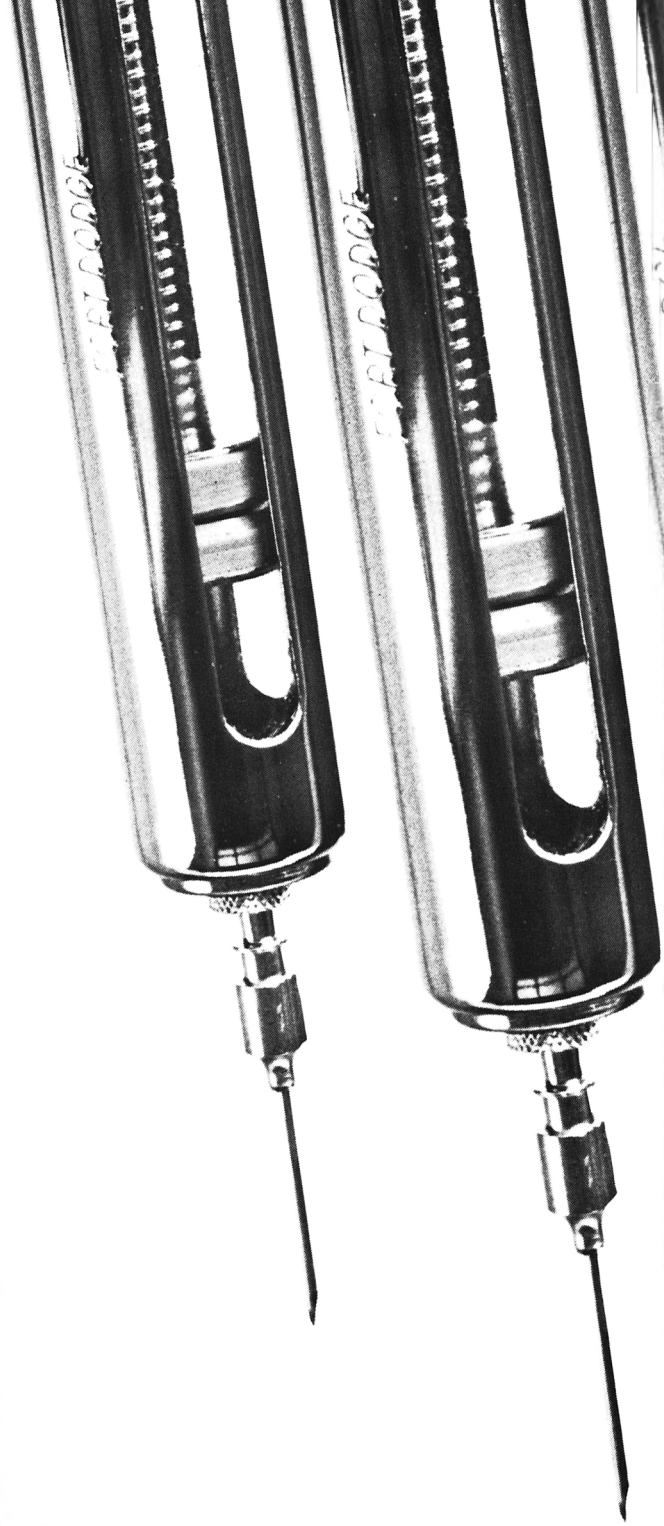

Completely compatible—for 3-way prevention program

Working together, this team of three biologicals makes one big point: Powerful, all-around protection. Rea-Plex works against the two most common viral respiratory infections. Septobac against the most common bacterial pneumonia. Ultilep against the most common type of leptospirosis. As a specific, each is unsurpassed. In combination, your feedlot prevention program is well in hand.

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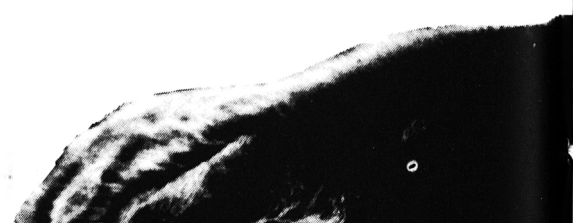

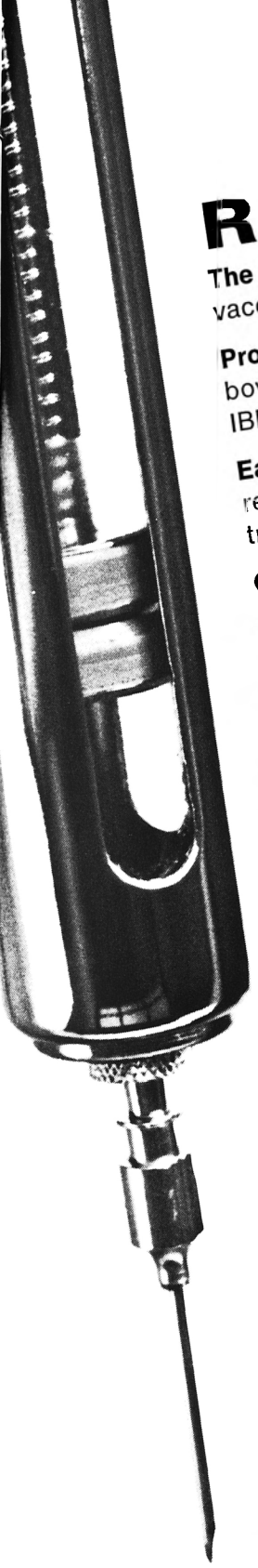

Rea-Plex®

IBR/PI-3 VACCINE

The "big one" in its field—Rea-Plex is today's most widely used, most trusted vaccine for bovine viral respiratory infections.

Proven on millions of calves—Efficacy and safety have been confirmed by bovine practitioners, in over 4 years of feedlot use—longer than any other IBR/PI-3 vaccine.

Easy to give—easy on the calf—Just one quick, easy injection—with minimum restraint, minimum stress. There's no problem of virus excretion, or transmission from vaccinates to non-vaccinates.

Only one Rea-Plex—Though widely imitated, Rea-Plex is produced by a patented process that can't be copied. No other vaccine is "just like Rea-Plex."

Septobac®

PASTEURELLA BACTERIN

Adds protection against pneumonia—Septobac adds a third dimension to the protection program—by helping prevent pasteurella-caused pneumonia. Compatible in every way with Rea-Plex and Ultilep, it may be used simultaneously.

Only trivalent Pasteurella bacterin—Only Septobac contains *P. multocida*, types A and D, and *P. hemolytica*, type 1. These three strains were isolated from calves with virulent infections.

"Biotized" for highest efficacy—In the exclusive Biotized process, bacteria are harvested at peak of vigor, during most active growth. This results in greater antigenic response.

Proven effective—Established potency. First potency tested pasteurella bacterin.

Ultilep®

LEPTOSPIRA POMONA BACTERIN

Absolutely 100% serum-free—No serum whatever is used in producing Ultilep. This reduces shock-producing potential—provides highest safety.

Adjuvant heightens antigenicity—The time-honored adjuvant, Gel 21, provides slow-release of killed organisms and antigenic fractions. This greatly enhances immunizing properties.

Field-proved potency and efficacy—Years of feedlot and herd use show Ultilep virtually trouble-free, and highly effective in preventing *L. pomona* infections.

200 Attend Breeding Soundness Symposium

An estimated 200 members, students and other veterinarians participated in the Reproduction Symposium October 7 and 8 at the New Bolton Center, Kennett Square, Pa., according to Dr. Barton Gledhill, the General Chairman. Included were representatives from France and the Netherlands. The event was the annual fall conference of the American Veterinarian Society for the Study of Breeding Soundness. The program was confined to equine and canine reproduction.

At the Society business session, it was announced that the 1972 fall conference is scheduled for the Kellogg Center at Michigan State University. Dr. Fayne Oberst will serve as General Program Chairman.

In addition, the Professional Publications Committee will revise and update the present Volume VI manual on Bovine Semen Evaluation, will develop other manuals, and prepare and secure articles for publication to Society members. Dr. C. J. Bierschwal of Columbia, Missouri, will chair the group, assisted by Drs. Ed Carroll of Pueblo, Colorado, and Les Ball of Fort Collins, Colorado.

A catalogue of audio-visual materials on animal reproduction is to be prepared by Dr. Raimunds Zemjanis of St. Paul.

A state-by-state membership drive also will be conducted with a goal of 500 members with chairmen for each state to be designated by President Gledhill.

Officers elected for the coming year include: President Dr. Barton Gledhill of Kennett Square, Pa., Vice-president Dr. John Simons of Torrington, Wyoming, Secretary-Treasurer Dr. John Williams of Athens, Georgia. Dr. Wm. R. McGee of Lexington, Kentucky, was elected to the Board of Directors. Outgoing Board members were Dr. Marvin Beeman of Littleton, Colorado, and Dr. Raimunds Zemjanis of St. Paul.

A RATIONAL BASIS FOR THE PREVENTION OF PARTURIENT PARESIS

(Continued from page 8)

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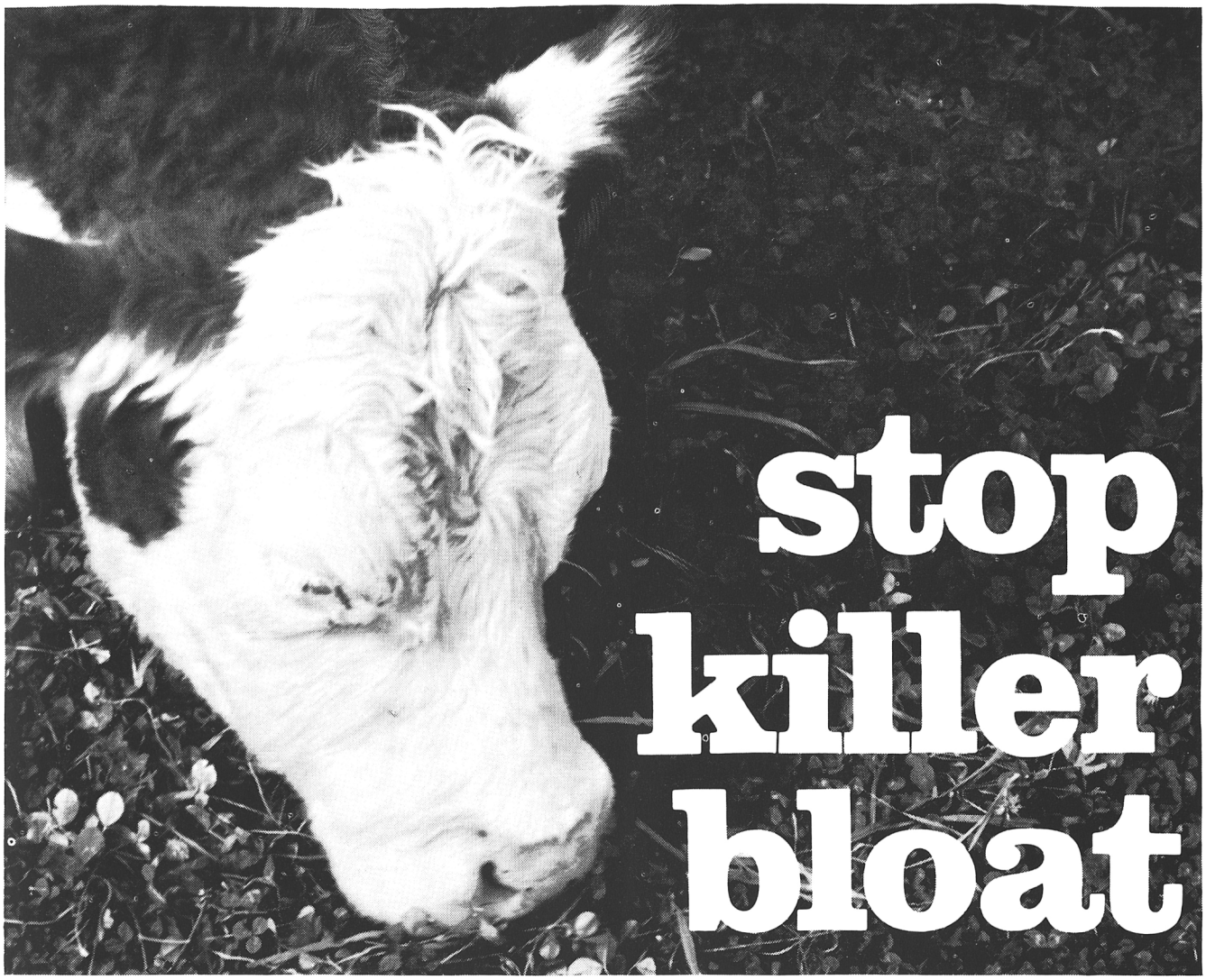

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