The Effects of Cold Stress on Neonatal Calves
I. Clinical Condition and Pathological Lesions

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ABSTRACT

Newborn Holstein-Friesian calves were cold stressed by immersion in water at 15 to 17°C until the core body temperature was lowered by 10°C. Non-cold stressed calves were immersed in water at thermoneutral temperature (35 to 37°C).

The time required to lower the core body temperature of the cold stressed calves by 10°C was 172 ± 87 minutes (mean ± SD). The time required for the core body temperature of the cold stressed calves to return to normal after immersion was 400 ± 140 minutes (mean ± SD).

Differences were observed between cold and noncold stressed calves in the shivering response during immersion and the clinical condition after immersion. Cold-induced pathological lesions were confined to tissues located peripherally, particularly in the hind legs. Significant differences were observed between cold and noncold stressed calves in the incidence of subcutaneous edema in the ventral sternum ($P \leq 0.025$), subcutaneous hemorrhage in the hind legs ($P \leq 0.025$), synovitis ($P \leq 0.025$) and hemorrhage ($P \leq 0.05$) of the synovial membranes of the hock joints and hemorrhage ($P \leq 0.05$) into the hock joint cavities.

The similarities between the clinical observations and pathological lesions observed in the present study and those reported for cold stress in newborn infants and the weak calf syndrome are discussed.

RÉSUMÉ

Cette expérience visait à provoquer le stress du froid chez deux groupes de veaux Holstein-Friesian nouveau-nés, en les immergeant dans un bassin d'eau dont la température variait de 15 à 17°C, jusqu'à ce que leur température descende de 10°C. On immergea par ailleurs un groupe de veaux témoins dans un bassin d'eau dont la température ne variait que de 35 à 37°C.

Il fallut 172 ± 87 minutes (moyenne ± DS) pour faire descendre la température des veaux expérimentaux de 10°C. Le temps requis pour ramener à la normale la température de ces veaux fut de 400 ± 140 minutes (moyenne ± DS).

On nota des différences relatives au frissonnement des veaux expérimentaux et des témoins, durant leur immersion, et à leur condition clinique ultérieure. Les lésions attribuables au froid se limitaient surtout aux tissus périphériques des membres postérieurs. On nota des différences appréciables entre les veaux expérimentaux et les témoins, relativement à l'incidence d'oedème sous-cutané sternal ($P < 0.025$), d'hémorragie sous-cutanée des membres postérieurs ($P < 0.025$), d'inflammation ($P < 0.025$) et d'hémorragie ($P < 0.05$) des gaines synoviales tarsiennes, ainsi qu'à celle d'hémorragies ($P < 0.05$) articulaires tarsiennes.

Les auteurs commentent les similitudes entre les observations cliniques et les lésions qu'ils notèrent, au cours de cette expérience, et celles que rapportent des

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articles antérieurs, en ce qui concerne le stress du froid, chez les enfants nouveaunés, et le syndrome des veaux faibles.

INTRODUCTION

Many calves in the western states are born during winter and early spring. Calving during this time often occurs under harsh weather conditions. Field studies have shown that calf mortality rates are higher during winter and early spring than at other times of the year (11, 14, 17, 21). Cold and fluctuating air temperatures and excessive wind and moisture are common weather-related thermal (cold) stressors during this period and may contribute to reduced survival of newborn calves (11, 21).

Newborn calves are more susceptible to the effects of cold exposure than are mature cattle (6, 20) because their cold defense and heat conservation mechanisms are not fully developed. Factors which may enhance excessive loss of body heat by calves include a relatively high ratio of body surface to body mass, thin skin, small quantity of subcutaneous fat, poor cutaneous vascular control and evaporative heat loss from the wet skin at birth (6, 11, 20).

Previous studies have described the physiological (1, 2) and pathological (7) changes observed in cold stressed lambs. The authors are not aware of similar studies in calves.

The work reported here was undertaken to determine the clinical and pathological effects of cold stress on newborn calves. We now report that calves exposed to cold stress sufficient to cause hypothermia exhibit clinical signs and pathological lesions similar to those seen in calves born under stressful range conditions.

MATERIALS AND METHODS

Animals

Twenty-six colostrum deprived Holstein-Freisian bull calves were obtained at birth and used in the study. Each calf was weighed and then assigned to one of three experimental groups. Calves in groups 1 and 2 were cold stressed, whereas calves in group 3 were noncold stressed.

Treatment method

A water tank was constructed of plywood and used for cold treatment of the calves. The water tank (1.25 m long, 0.75 m wide and 1.12 m deep) was reinforced at the top and bottom on the outside by channel irons and steel rod braces and sealed with wood screws and fiberglass resin. The water level in the tank was sufficient to prevent the calves from touching the bottom of the tank. Each calf was suspended in a sternal floating position in the water, the body was supported and stabilized by two poplin slings with leg holes and the head was supported above water by a halter.

Calves in groups 1 and 2 were cold stressed by immersion in water at 15 to 17°C. The animals were removed from the water tank after the core body temperature (CBT) was lowered 10°C, transferred to a cold room at 4°C and bedded with wet wood shavings. Calves in group 1 were euthanatized at two hours, while those in group 2 were euthanatized at 72 to 96 hours after removal from the water tank. Calves in group 3 were immersed for two hours in water at thermoneutral temperature (33 to 35°C) and then transferred to a room which was maintained at 25°C and bedded with dry straw. Calves in group 3 were euthanatized at 72 to 96 hours after removal from the water tank.

The CBT of the calves was measured with a thermister[1] probe which was inserted through the anus and into the colon a distance of 20 cm. A telethermometer[1] was used to record the temperature. Core body temperatures were measured prior to immersion, every five minutes during immersion and at 15 minute intervals after the calves were removed from the water vat and until the temperatures had stabilized. Thereafter, CBT were measured approximately every 12 hours for the remainder of the experiment. The clinical condition (shivering and behavioral response) of calves was also noted during and after immersion.

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[1]Yellow Springs Instrument Company, Inc., Yellow Springs, Ohio.

Feeding schedule

All calves were fed one liter of pooled whole colostrum by stomach tube. Calves in groups 1 and 2 were fed when the CBT had been lowered 2°C or 5°C, respectively, whereas calves in group 3 were fed after 60 minutes of immersion. Calves in groups 2 and 3 were maintained on a diet of reconstituted milk replacer for the duration of the experiment.

Pathology and specimens

Gross pathological lesions were noted at necropsy. Tissues collected for histopathological examination included synovial membrane from the hock joints, skin from the distal portion of the legs and sections from most major internal body organs. The tissues were fixed in 10% (v/v) neutral buffered formalin and stained with hematoxylin and eosin according to established procedures (8).

Synovial fluid was collected from the open hock joint cavities of the calves at necropsy. The color was noted and the samples were stored (–20°C) until analyzed for hemoglobin. The amount of hemorrhage into the hock joint cavity was quantitatively estimated by measuring the concentration of hemoglobin in the samples of synovial fluid as determined by the cyanmethemoglobin technique.[2]

Statistical treatment

Gross and histopathological lesions observed in the calves were compared by the Chi-square method of analysis using 2 by 2 contingency tables (16). The amount of hemorrhage into the hock joint cavities of the calves was compared by Duncan's new multiple range test (19).

RESULTS

The mean CBT of calves in groups 1, 2 and 3 before immersion did not differ statistically. The mean preimmersion CBT for all calves was 38.4 ± 0.6°C (mean ± SD). The time required to lower the CBT of the cold stressed calves by 10°C was 172 ± 87 minutes (mean ± SD). The time required for the CBT of the cold stressed calves to return to normal after removal from the water tank was 400 ± 140 minutes (mean ± SD). The typical CBT response of a neonatal calf to cold stress is shown (Fig. 1). There was no obvious association between the body weight of the individual calves (mean body weight = 42.3 kg, range = 30.5 to 55.0 kg) and the rate at which CBT decreased during treatment or increased after removal from the water tank. The CBT of calves in group 3 were not affected by treatment.

Shivering was noted in cold stressed calves and began approximately ten minutes after immersion and reached maximum intensity by 40 minutes. Shivering became less intense or was no longer discernible after the CBT had been lowered by 6 to 8°C. Depression and increased relaxation of skeletal muscles were consistently observed in the cold stressed calves from the time of loss of the shivering response until removal from the water tank. Shivering resumed within several minutes after the calves were removed from the water tank and continued until the CBT had returned to near normal. Shivering and depression were not observed in calves in group 3 during treatment.

The clinical condition of calves in group 2 differed from that of calves in group 3 during the period between removal from the water tank and euthanasia. Calves in

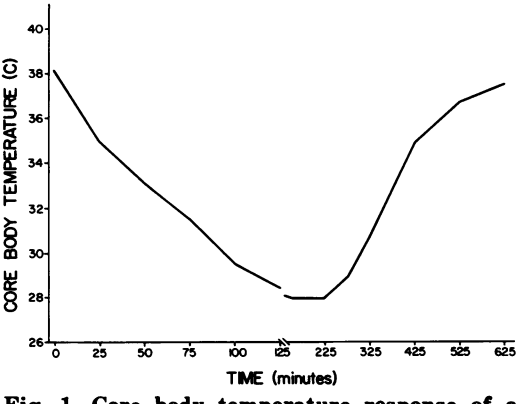

Fig. 1. Core body temperature response of a newborn calf subjected to cold stress. The animal was immersed in 16°C water at 0 time and removed from the water and transferred to a 4°C room at 125 minutes.

[2]Hycel Inc., Houston, Texas.

TABLE I. Incidence of Gross Pathological Lesions in Cold Stressed (groups 1 and 2) and Noncold Stressed (group 3) Neonatal Calves

Gross Pathological Lesions	Frequency of Occurence of Lesions[a]				Comparison Between Groups (P values)		
	Groups						
	1	2	3	1 & 2	1 vs 2	2 vs 3	1 & 2 vs 3
Subcutaneous hemorrhage in the forelegs	4/7[b]	9/9	4/9	—	NS	NS	—
Subcutaneous hemorrhage in the hindlegs	6/8	9/9	3/9	—	NS	<0.025	—
Subcutaneous edema in the legs[c]	5/15	8/18	1/18	13/33	—	—	<0.025
Subcutaneous edema-ventral sternum	0/8	7/9	1/9	—	NS	<0.025	—

[a]Numerator = the number of the calves with the lesion; denominator = the number of calves examined
[b]The forelegs of one calf from group 1 were excluded from analysis because of severe hemorrhage from dystocia
[c]Numerator = the number of calves with subcutaneous edema either in the forelegs or hindlegs; denominator = the number of pairs of forelegs and hindlegs examined
NS = Not significant

group 2 were usually in sternal recumbency, remained depressed and had greater difficulty in standing and maintaining their balance as well as feeding from a nipple bottle. In contrast, calves in group 3 were active and alert, appeared physically strong and readily nursed from a nipple bottle. The skin surface over the back and lateral thorax and abdomen of the calves in group 2 felt warm during recovery, whereas, the skin surface over the extremities, i.e. legs, ears and tail, felt cold.

The gross pathological lesions observed in the calves are summarized (Table I, Figs. 2-6). The lesions observed most frequently in the cold stressed calves (groups 1 and 2) included subcutaneous edema and ecchymotic and suffusion hemorrhages in the legs and subcutaneous edema in the ventral region of the sternum. No pathological lesions were observed in the internal body organs examined.

Subcutaneous hemorrhages were observed in the hind legs of calves from all experimental groups. The incidence of subcutaneous hemorrhage in the hind legs was significantly greater ($P \leq 0.025$) in the calves in group 2 than in the calves in group 3. Hemorrhages were most severe in the lateral and posterior surfaces of the hock joints and less severe in the metatarsal region. The hemorrhages in the hind legs were more severe in the calves in group 2 (Fig. 2) than in the calves in group 1 (Fig. 3). Subcutaneous hemorrhages in the hind legs of calves in group 3 were either comparatively mild or usually absent (Fig. 4).

Subcutaneous hemorrhages were also observed in the region of carpal (Fig. 5) and fetlock (Fig. 6) joints of calves in all groups. The hemorrhages in the forelegs

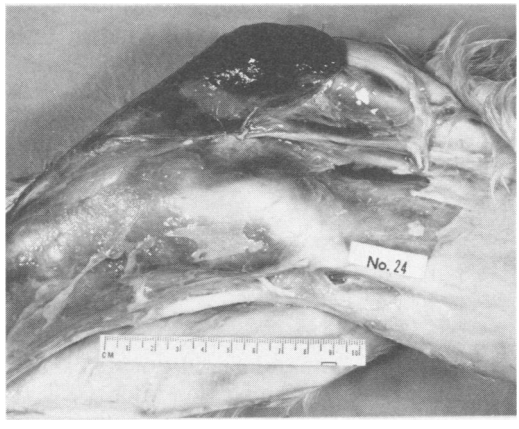

Fig. 2. Lateral view of the hock joint of a calf subjected to cold stress and then allowed to recover (group 2). Notice the extensive subcutaneous hemorrhage.

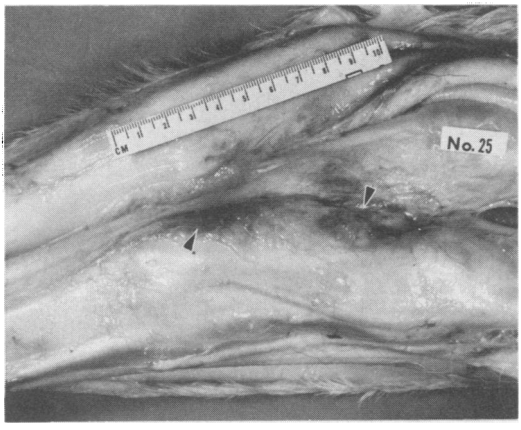

Fig. 3. Lateral view of the hock joint of a calf subjected to cold stress and then euthanatized (group 1). Notice the subcutaneous hemorrhage (arrows).

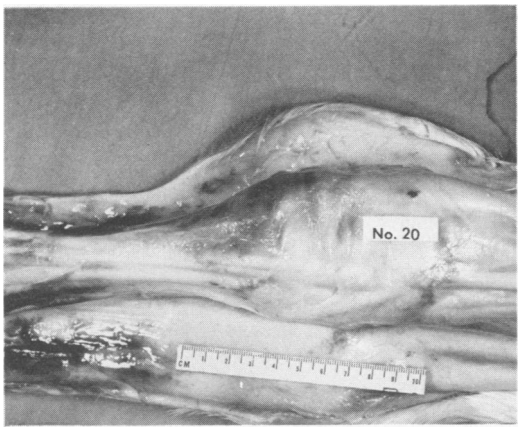

Fig. 5. Dorsal view of the carpal joint of a calf subjected to cold stress and then allowed to recover (group 2). Notice the subcutaneous hemorrhage.

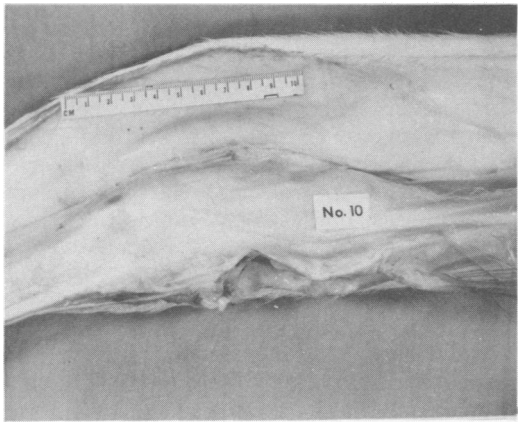

Fig. 4. Lateral view of the hock joint of a noncold stressed calf (group 3). Notice the absence of subcutaneous hemorrhage.

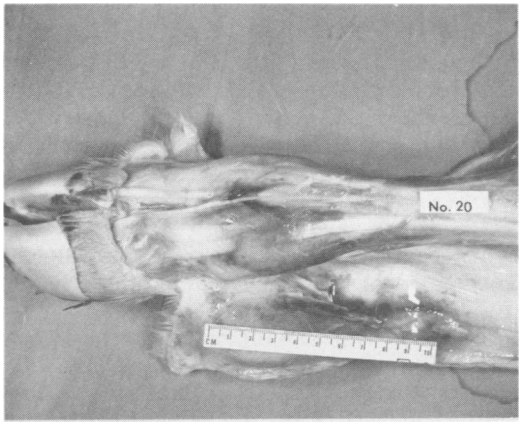

Fig. 6. Dorsolateral view of the fetlock joint of the calf in Figure 5. Notice the subcutaneous hemorrhage.

were generally less severe, however, than those observed in the hind legs. There was no significant difference ($P > 0.05$) between groups in the incidence of hemorrhage in the forelegs.

Subcutaneous edema in the legs was generally restricted to the tissues adjacent to the proximal ends of the large metacarpal and metatarsal bones. The combined incidence of subcutaneous edema in the legs of calves in groups 1 and 2 was significantly greater ($P \leq 0.025$) than in the calves in group 3.

The incidence of subcutaneous edema in the ventral region of the sternum was significantly greater ($P \leq 0.025$) in the calves in group 2 than in group 3.

The histopathological lesions observed in the synovial membranes of the hock joints of calves are summarized (Table II). The lesions included mild to severe hemorrhage and acute synovitis. The acute synovitis was characterized by accumulation of edema fluid, fibrin and infiltration of neutrophils into the tissue adjacent to the synovial membrane. Macrophages, lymphocytes and plasma cells were also present in smaller numbers. The incidence of synovitis and hemorrhage was significantly greater ($P \leq 0.025$ and $P \leq 0.05$, respectively) in the hock joints of the calves in group 2 than in group 3.

TABLE II. Histopathological Lesions in the Synovial Membranes and Adjacent Tissues of the Hock Joints of Cold Stressed (groups 1 and 2) and Noncold Stressed (group 3) Calves

Histopathological Lesions	Frequency of Occurence of Groups			Comparison Between Groups (P values)	
	1	2	3	1 vs 3	2 vs 3
Hemorrhage	5/8[a]	9/9	4/9	NS	<0.05
Synovitis	0/8	7/9	1/9	NS	<0.025

[a]Numerator = the number of calves with the lesion; denominator = number of calves examined
NS = Not significant

TABLE III. Color of Synovial Fluid from the Hock Joint Cavities of Cold Stressed (groups 1 and 2) and Noncold Stressed Calves (group 3)

Group No.	Color of Synovial Fluid[a]		
	Yellow	Red	
		Unilateral	Bilateral
1	1[b]	—	7
2	1	3	5
3	6	2	1

[a]Yellow coloration = bilateral; unilateral red = the contralateral side appeared yellow
[b]Number of calves observed

Red discoloration of the synovial fluid of the hock joint cavities was observed in calves from all groups and in most cases was bilateral (Table III). The incidence of discoloration of the synovial fluid was significantly greater in calves in group 1 ($P \leq 0.025$) and in group 2 ($P \leq 0.025$) when compared to calves in group 3. The concentration of hemoglobin in synovial fluid was determined as a quantitative estimate of the amount of hemorrhage into the hock joint cavities of calves (Table IV). The hemoglobin concentrations in the hock joint cavities was significantly greater ($P \leq 0.05$) in the calves in groups 1 and 2 than in group 3.

DISCUSSION

Many beef cattle management systems make provision for an early calving season to occur between January and March. The long range benefits derived from an early calving season include greater feed efficiency and utilization of forage feed supplies by more mature growing calves, heavier weaning weights and possibly greater resistance of older calves to stress associated with weaning. The disadvantages of early calving are that births are likely to occur during inclement weather and newborn calves often have no immediate protection from cold fluctuating air temperatures and a wet environment. The authors are not aware of recent cattle disease surveys or published estimates indicating annual losses of young calves specifically due to weather related stressors. Nonetheless, it seems reasonable that these stressors are often directly responsible for many calf losses each year.

Body heat is normally lost from the skin surface by radiation (5) and convection (3, 13). Additional heat is also lost by conduction (3, 13) and evaporation (12) when the skin and hair coat are wet. Many cattle producers have observed that young calves often seem remarkably resistant to cold temperatures provided their skin and hair coat remain dry. The present study was designed to cause a rapid loss of body heat and to overwhelm the thermoregulatory capabilities of the calves. It is possible that the conditions for producing cold stress in the present study were of greater

TABLE IV. Hemoglobin Concentration in the Synovial Fluid from Hock Joints of Cold Stressed (groups 1 and 2) and Noncold Stressed (group 3) Calves

Mean Log Hemoglobin Concentration (log mg/mL)				
Groups			Comparison Between Groups (P values)	
1	2	3	1 vs 3	2 vs 3
0.8024	0.7386	0.1734	<0.05	<0.05

magnitude than those which young range calves often endure under natural conditions. However, under cold and wet conditions the rate at which CBT is lowered in young range calves may well be as great as that shown by the calves in groups 1 and 2. If this is so then the net effects of cold stress on range calves may be just as damaging as those observed in our experimental calves.

Our data indicate that heavier weight calves had no apparent advantage over lighter weight calves in thermoregulation under conditions of cold stress. Perhaps the heavier weight calves would have been less affected by somewhat less severe cold stress than would the lighter weight calves.

Results showed that cold stress and hypothermia can consistently cause depression and temporary loss of shivering after the CBT has been lowered by at least 6 to 8°C. The thermoregulatory capabilities of the cold stressed calves, including shivering and presumably other mechanisms, were not permanently lost, however, since all the calves in group 2 showed a remarkable ability to regain normal body temperature after treatment in the water vat and during recovery in a cold (4°C) and wet environment. The common practice of placing cold stressed range calves under a heat lamp or in a warm room or a container of warm water is justifiable as supportive treatment intended to augment the thermoregulatory capabilities of these animals.

The probability of traumatic injury of calves in the present study was minimized because of the protective support system used to maintain the animals in a nearly fixed floating position during treatment in the water tank and the use of soft bedding during recovery. We, therefore, do not believe that the cold stressed calves were subjected to any more trauma than were the noncold stressed calves. Further, we conclude that the pathological lesions observed were related to cold rather than trauma.

The cold-induced pathological lesions observed in calves were confined to tissues located peripherally and which had greatest opportunity for contact with the cold and wet environment. The pathogenesis of the cold-induced subcutaneous hemorrhage and edema observed in peripheral tissues is unclear. Histopathological lesions that would suggest cold-induced injury to the microvasculature of the peripheral tissues were not found with consistency in cold stressed calves. Increased capillary permeability and fragility, damage to vascular endothelium and functional alterations of blood platelets and clotting mechanisms remain as possible explanations for the observed lesions.

The clinical signs and pathological lesions observed in calves in the present study are strikingly similar to those reported in cold stressed human infants (9, 10, 15). Cold injury is seen in infants usually less than ten days of age which have been born during the winter in homes with inadequate heating facilities. These environmental circumstances are similar to those experienced by cold stressed calves under range conditions.

The results of the present study support a previous report (4) indicating that environmental stress was closely related to heavy calf losses diagnosed as weak calf syndrome (WCS). The clinical signs and pathological lesions in calves in the present study are similar to those described for WCS (18) and suggest that environmental stress was a major etiological factor of this disease problem. Data from the present study support earlier observations (18) indicating that sampling synovial fluid from the hock joints and observing for evidence of hemorrhage is not a reliable indicator of specific disease.

It would seem that cold stress is a common problem in areas of cold climate and deserves greater recognition as a major contributor to disease and death of young calves. Further research is needed to understand the pathogenesis of the changes resulting from cold stress and to develop effective methods for prevention of cold-induced disease.

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REFERENCES

1. ALEXANDER, G. Temperature regulation in the newborn lamb. IV. The effect of wind and evaporation of water from the coat on metabolic rate and body temperature. Aust. J. agric. Res. 13: 82. 1961a.

2. ALEXANDER, G. Temperature regulation in the newborn lamb. V. Summit metabolism. Aust. J. agric. Res. 13: 100. 1961b.
3. AMES, D. R. and L. W. INSLEY. Wind-chill effect for cattle and sheep. J. Anim. Sci. 40: 161. 1975.
4. CASSIDY, R. D. Final report of calf losses in Custer and Lemhi Counties of Idaho. National Animal Disease Center, Ames, Iowa. pp. 1-42. 1973.
5. CENA, K. Radiative heat loss from animals. In Heat Loss from Animals and Man. J. L. Monteith and L. E. Mount, Editors. pp. 35-38. London: Butterworths Scientific Publications. 1974.
6. GONZALES-JIMINEZ, E. and K. L. BLAXTER. The metabolism and thermal regulation of calves in the first month of life. Br. J. Nutr. 16: 199. 1962.
7. HAUGHEY, K. G. Cold injury in newborn lambs. Aust. vet. J. 49: 554-556. 1973.
8. LUNA, L. G. Manual of Histologic Staining Methods of the Armed Forces Institute of Pathology. 3rd Ed. pp. 32-46. New York: McGraw-Hill. 1968.
9. MANN, T. P. Hypothermia in the newborn: A new syndrome? Lancet 1: 613-614. 1955.
10. MANN, T. P. and R. I. K. ELLIOTT. Neonatal cold injury: Due to accidental exposure to cold. Lancet 1: 229-234. 1957.
11. MARTIN, S. W., C. W. SCHWABE and C. E. FRANTI. Dairy cattle mortality rate: Influence of meteorologic factors on calf mortality rate in Tulare County, California. Am. J. vet. Res. 36: 1105-1109. 1975.
12. McCLEAN, J. A. Loss of heat by evaporation. In Heat Loss from Animals and Man. J. L. Monteith and L. E. Mount, Editors. pp. 19-31. London: Butterworths Scientific Publications. 1974.
13. MITCHELL, D. Convective heat transfer from man and other animals. In Heat Loss from Animals and Man. J. L. Monteith and L. E. Mount, Editors. pp. 59-76. London: Butterworths Scientific Publications. 1974.
14. MOLL, T. The pathogenesis of diarrhea in the newborn calf, with special reference to physiologic functions and environmental conditions. J. Am. vet. med. Ass. 147: 1364-1366. 1965.
15. ROGERS, M. C., M. GREENBERG and J. J. ALPERT. Cold injury of the newborn. New Engl. J. Med. 285: 332-334. 1971.
16. SNEDECOR, G. W. and W. G. COCHRAN. Statistical Methods. 6th Ed. Ames: Iowa State University Press. 1967.
17. SPEICHER, J. A. and R. E. HEPP. Factors associated with calf mortality in Michigan dairy herds. J. Am. vet. med. Ass. 162: 463-466. 1973.
18. STAUBER, E. H. Weak calf syndrome: A continuing enigma. J. Am. vet. med. Ass. 168: 223-225. 1976.
19. STEEL, R. G. D. and J. H. TORRIE. Principles and Procedures of Statistics. New York: McGraw-Hill. 1964.
20. THOMPSON, G. E. Climatic physiology of cattle. Review of the progress in dairy science. J. Dairy Res. 40: 441-473. 1973.
21. WITHERS, E. W. Mortality rates and disease incidence in calves in relation to feeding management and other environmental factors. Br. vet. J. 108: 382-405. 1952.