General Session IV

Dr. Neil Anderson, *presiding*

The Effects of Cold Exposure on Neonatal Calves

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Beef cow calf management schemes often result in birth of calves during winter. Environmental stress of newborn beef calves during winter is highly likely because weather-related conditions such as cold, fluctuating air temperatures, and excessive wind and precipitation often prevail during this period. Moreover, the problem is made worse by little opportunity to provide either natural or man-made shelter for these animals, especially under range calving conditions. Poor management and inadequate housing may result in environmental stress of dairy calves as well. Other conditions which contribute to environmental stress of young calves include overcrowding of animals in the calving yards, poor drainage of surface water, and failure to provide clean and dry bedding. Field and laboratory studies have shown a direct relationship between exposure of young calves to adverse winter weather and an increased incidence of enteric and respiratory diseases in these animals.[1,3] Weak calf syndrome is a specific example of a disease believed to be caused in part by exposure to adverse weather.[4]

Thermoregulation in Animals

Review of some basic principles of thermoregulation is required first of all in order to develop a better understanding of the changes seen in cold stressed calves. Thermoregulation in animals involves the important principle of homeothermy.[5] Homeothermic animals are capable of regulating their normal core body temperature within narrow limits (±3.5°F) regardless of ambient air temperature within normal variable limits. A variety of avian and mammalian species have homeothermic capabilities. Homeothermy is described by the following equation where HP = heat production, HS = heat storage, HL = heat loss, HN = sensible heat loss, and HE = evaporative heat loss.

$$HP \pm HS = HL = HN + HE$$

Heat production must equal heat loss in order for homeotherms to maintain their core body temperature within narrow limits.

Sensible heat loss occurs by conduction, convection, and radiation and is determined in part by presence of peripheral insulation barriers such as thickness of the nornal skin, subcutaneous fat, and hair coat and also by an outer boundary of surface air. Sensible heat loss may be significantly increased by an increase in wind velocity and by evaporative heat loss due to wetting the body surface. Young calves are especially susceptible to heat loss due to a high ratio of body surface area to body mass; thin skin, subcutaneous fat layer, and haircoat; and evaporation of surface water and fluids from birth. In addition, heavier weight calves and calves up to 1 week of age seem to be as susceptible to severe cold as lighter weight and younger animals.

Various terms are used to describe the environment depending on ambient air temperature and the response of animals. The thermoneutral zone generally includes temperatures between 68 and 86 F and is described as the temperature zone where heat production by thermoneutral metabolism is independent of air temperature. Critical temperature is the lower limit of the thermoneutral zone and varies with species and with age. For example, the critical temperature for piglets, lambs, and calves are approximately 95°F, 84°F, and 55°F, respectively. These data illustrate the well-known intolerance of young piglets to cold and the apparent tolerance of young calves to moderate cold. Within the thermoneutral zone, there is described the zone of thermal comfort where homeothermy is maintained by small variations in evaporative and sensible heat losses. Temperatures within the cool zone induce reactions designed to conserve body heat but there is no increase in heat production. Heat conservation reactions in animals include seeking warmer environments, crowding, piloerection, and cold-induced vasoconstriction. The cold zone includes temperatures below the critical temperature which induce increased heat production by cold thermogenic mechanisms. Extremely cold temperatures induce summit metabolisms where maximal heat production is attained. Intolerable cold is the temperature zone where sensible heat loss exceeds heat production by cold thermogenesis and results in hypothermia.

Cold thermogenesis is achieved by shivering and nonshivering thermogenesis. Shivering is described as a tremor or rhythmic involuntary contraction of skeletal muscle and is controlled by the somatic nervous system. Shivering muscles require an increased supply of oxygen and oxidizable substrate. The utilizable byproduct from shivering muscles is dispersed as heat which warms the blood and surrounding tissues. Nonshivering thermogenesis is controlled by the autonomic nervous system and mediated by the catecholamines, noradrenaline and adrenaline. The principle benefit derived from nonshivering thermogenesis by young calves is the direct thermogenic effect of catecholamines on brown fat. In addition, nonshivering thermogenesis also serves to mobilize oxidizable substrates from white fat, liver, and skeletal muscle and to potentiate shivering.

Cold stress in animals causes other metabolic responses as well. Thus, changes in plasma concentrations of glucose, free fatty acids, lactate, glycerol, catecholamines, and glucocorticoids have been useful indicators of stress reactions in animals.

Cold Stress in Young Calves

A number of controlled laboratory studies have recently been conducted to determine the effects of moderate and severe cold exposure on young calves. These studies were designed in part to simulate naturally-occurring adverse weather conditions. Following is a categorical description of the changes that the veterinary practitioner could expect to find in cold stressed calves based on the results obtained from these studies.

Clinical condition and behavioral reaction. Severely cold stressed calves show signs of profound physical weakness, depression, and loss of vigor.[6] Animals often segregate themselves and spend long periods of time in sternal or lateral recumbency. They are reluctant to stand or walk and have great difficulty in achieving either of these locomotor postures. When standing, they assume either a closed or uneven stance and their head is lowerd and back is arched. Severly cold stressed calves have difficulty in nursing and frequently show loss of appetite. In addition, their eyes appear glazed and they have poor response to stimuli. Intensive and constant shivering is seen during the early stages of cold stress but then becomes intermittent or nonexistent after the core body temperatures has been lowered by 8 to 10 F. Death occurs in severely cold stressed calves due to a combination of many factors; however, it is ultimately due to respiratory failure. Exposure of calves to moderate cold temperature (32 F) causes intermittent shivering and slight depression but no other signs similar to those described in severely stressed animals.[7]

Temperature response. Core body temperature is one of the best indicators of the thermoregulatory capabilities of animals and can be determined, for example, by measuring temperatures deep within the colon or mixed venous blood within the right ventricle. Core body temperatures decrease linearly as animals become progressively hypothermic.[6,8] Severe cold may lower core body temperature by as much as 18 F in 12 hours or less. Death may occur in calves after more prolonged cold exposure even though their core body temperature has not been lowered by 18 F. Rectal and oral temperature also decrease linearly with progressive hypothermia and are usually only 1 to 1.5 F colder than core body temperatures. Temperatures within large muscles in the pectoral and thigh regions also decrease in severely cold stressed calves despite intensive shivering by these large muscle groups. Finally, subcutaneous temperatures decrease most rapidly, especially in the extremities, where they may approach those of the ambient air temperature. Peripheral tissues often feel cold and clammy by digital palpation. Exposure of young calves to moderate cold is not likely to lower core body, rectal, oral, or muscle temperatures whereas subcutaneous temperatures are likely to decrease.

Clinical pathologic research—hematology. Hematologic findings seen in cold stressed calves often represent trends rather than significant and absolute changes. Nevertheless, severe cold stress tends to lower the total number of leukocytes in peripheral blood of calves.[9] This lowering of total leukocytes may be as much as 45% and is due primarily to a decrease in segmented neutrophils although numbers of lymphocytes and monocytes decrease as well. Other hematologic changes include slight increases in total erythrocytes, hemoglobin concentration, and packed cell volume.

Clinical pathologic response—serum chemistry. Severe cold stress causes significant and rapid increases in plasma concentrations of glucose, cortisol, and catecholamines.[10,11] Glucose concentrations are most likely increased because of the hyperglycemic effects of the elevated cortisol and catecholamines and to the tendency for insulin levels to decrease. Later, glucose concentrations are likely to be low due to excess catabolism by shivering muscles. In contrast, concentrations of cortisol and catecholamines tend to remain elevated throughout cold stress and for several hours during recovery. Plasma concentrations of alkaline phosphatase, aspartate aminotransferase, and lactate dehydrogenase tend to increase during cold stress and remain elevated for several hours during recovery. There is little change in all other plasma constituents except for moderate increases in plasma concentrations of phosphorous and iron.

Cardiovascular response. Severe cold stress causes an initial increase in aortic blood pressure followed by a significant decrease as hypothermia progresses.[12] Similarly, there is an initial and significant increase in heart rate (180 beats/minute) during early stress which is followed by a significant decrease in heart rate to 95 beats/minute later on. Heart sounds become progressively muffled and are barely discernible in severely hypothermic animals. Electrocardiograms (lead III) show an increase in the number of peaks and

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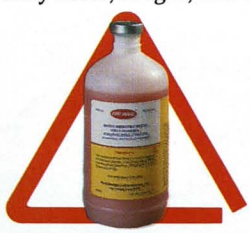

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erratic wave forms as hypothermia progresses. Cardiac output and stroke volume are also decreased in hypothermic calves.

Absorption of colostral immunoglobulins. Severe cold stress and subsequent hypothermia causes a 1.5- to 3.5-hour delay in onset of absorption of colostral immunoglobulins M, G_1, and G_2.[13] In addition, there is a significant decrease in the rate of absorption of these immunoglobulins for the first 15 to 18 hours after feeding of colostrum. Thus, cold stressed and hypothermic calves remain hypogammaglobulinemic and more susceptible to diseases during the first critical hours of life. In contrast, normothermic calves subjected to moderate cold temperatures are able to absorb colostral immunoglobulins as well as noncold stressed calves.[14]

Other host defense mechanisms. Lymphocytes from young calves exposed to moderate cold tend to show an increase in stimulation index to mitogens.[15] In contrast, exposure of the same animals to moderate cold has no affect on activity of complement in plasma[16] or the *in vitro* bactericidal activity of their neutrophils.[17]

Lesions. Exposure of young calves to moderate or severe cold will consistently induce lesions. These lesions include extensive subcutaneous ecchymotic and suffusion hemorrhages and edema in the distal portions of the extremities and ventral to the sternum.[6,7] Cold exposure also causes extensive unilateral or bilateral hemorrhage into the hock joint cavity.

Treatment of Cold Stressed Calves

A major objective in treatment of cold stressed calves is to provide supplemental heat in order to decrease sensible heat loss. Cold stressed calves may be treated by rewarming the exterior or the core of the body or by a combination of both procedures. Treatment methods designed to rewarm primarily the body core include intravenous administration of prewarmed saline or electrolyte solutions or allowing the animal to respire prewarmed water-saturated air. An electric steamer fitted with a face cone facilitates the latter technique. Core rewarming results in initial and preferential warming of visceral organs and skeletal muscles and passive rewarming of peripheral tissues. Following is a discussion of 3 practical methods designed to rewarm the exterior body surface[8]. In principle, external rewarming induces dilation of superficial vessels and rewarming of peripheral blood which is subsequently carried to the body core.

Rewarming calves with a water tank. Cold stressed calves may be rewarmed by total body immersion in a tank filled with water at 112°F. The tank can be constructed with ¾" exterior plywood and should be 4' (L) x 2'6" (W) x 3' (H) in order to accomodate larger size calves. The sides of the tank should be reinforced with steel angle iron and rods to withstand excessive water pressure. All corners should be thoroughly sealed with a waterproof material and a spigot can be placed near the bottom to facilitate draining the water. The tank should also be lined either with fiberglass or galvanized metal. Muslin slings, each with leg holes, can be used to stabilize the front and rear of the calf in a floating position during rewarming. In addition, a rope halter should be used to keep the animal's head above water. The warm water should be circulated at all times with a submersible utility pump. Respiration, heart rate, and rectal temperature can be easily monitored during rewarming. The rewarmed calf should then be removed from the tank, thoroughly dried, and placed in a warm dry environment for additional treatment that might be required. The time required to rewarm cold stressed calves with severe hypothermia by this method ranges from 45 to 60 minutes.

Rewarming calves with electric lamps. Two or three infrared heat lamps, 250 watts each, may be used to rewarm cold stressed calves. The animal should be thoroughly dried and then placed within a partial enclosure to concentrate the heat generated from the lamps. Position the lamps approximately 10" from the body surface and locate them over the pectoral, dorsal thoracic, and thigh regions. Respiration, heart rate, and rectal temperature can be easily monitored during recovery. Surface tissues outside the direct beam of heat will not rewarm rapidly and will remain cool. The time required to rewarm cold stressed calves with severe hypothermia by this method will be approximately 130 minutes.

Rewarming calves with electric heat pads. Electric heat pads, 50 watts each, may be used as a third method for external rewarming of cold stressed calves. The animal should be thoroughly dried and then restrained in lateral recumbency. The heat pads should be secured over the posterior cervical/pectoral, thoracic, and lumbar regions with tie strings. The animal should also be wrapped in a heat-reflecting aluminized blanket to conserve the external heat generated. Surface tissue temperatures not covered by the heat pads will not rewarm rapidly and will remain cool. The time required to rewarm cold stressed calves with severe hypothermia by this method is approximately the same as that for heat lamps.

A number of observations and tests can be made to monitor progress during assisted recovery of cold stressed calves. In general, many of the changes seen in hypothermic calves during severe cold stress are reversed during assisted recovery. For example, calves will begin intensive shivering, regain their former alertness and awareness of surroundings, and respond normally to stimuli. Core body, rectal, and muscle temperatures will increase linearly. Subcutaneous temperatures of the extremities tend to remain colder than deeper body temperatures with the exception of those calves that recover in warm water. Many hypothermic calves placed at room temperature (77°F) will recover spontaneously with no assistance. However, these animals often require greater than 3 hours to recover and do so primarily by shivering thermogenesis. Further, these unassisted calves tend to remain depressed due in part to exhaustion from prolonged shivering. Total number of leukocytes and individual leukocytic cell types return to normal values in assisted and unassisted rewarmed calves.

Similarly, values for total number of erythrocytes, hemoglobin concentration, and packed cell volume also return to normal. Plasma concentrations of glucose may decrease or remain low during recovery depending upon the initial duration of cold exposure. As indicated earlier, plasma concentrations of catecholamines, cortisol, and certain enzymes tend to remain elevated during and after recovery. Aortic blood pressure and heart rate increase rapidly and soon after the start of rewarming. Heart sounds become more discernible by auscultation as rewarming progresses. Electrocardiographic wave forms also return to normal by the time the animal has fully recovered.

Conclusions

Cold stress is an environmental hazard that potentially can produce life-threatening changes in susceptible young calves. Calves may die either directly or indirectly as a result of cold exposure. A combination of adequate case history, clinical signs, and laboratory data often provide enough information necessary to make a correct diagnosis. Several methods of treatment are available to effectively and rapidly assist hypothermia calves during recovery. The fluid and electrolyte balances in hypothermic calves should also be taken into account when considering the most effective treatment regimen. It is important to realize the resilience and fortitude of young calves even though they have been severely cold stressed. These natural qualities, coupled with prompt and vigorous treatment, greatly enhance their chances for uneventful and complete recovery.

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