Evaluation of hoof wall surface temperature as an index of digital vascular perfusion during the prodromal and acute phases of carbohydrate-induced laminitis in horses

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Objective—To evaluate the use of hoof wall surface temperature (HWST) as an indirect indicator of digital perfusion and to describe HWST patterns during the prodromal and acute phases of carbohydrate-induced laminitis in horses.

Animals—30 adult horses without foot abnormalities.

Procedures—Three experiments were performed. In the first, HWST was measured in 2 groups of horses acclimatized to hot (n = 6), or cold (6) environments and exposed to cold (15 C) ambient temperature. In the second experiment, HWST were measured in both forefeet of 6 horses before and after application of a tourniquet to 1 forefoot to induce vascular occlusion. In the third experiment, HWST were recorded in 12 horses before and during the prodromal and acute phases of carbohydrate-induced laminitis.

Results—Mean HWST of hot-acclimatized cold-challenged horses was significantly less than that of cold-acclimatized cold-challenged horses at all times. Transient episodes of high HWST were observed during prolonged cold-induced vasoconstriction. Hoof wall surface temperature significantly decreased during arterial occlusion and increased during reperfusion. Digital hypothermia was observed during the prodromal phase of carbohydrate-induced laminitis.

Conclusions and Clinical Relevance—Determination of HWST is a valid technique to evaluate digital perfusion under appropriate controlled conditions in horses. Digital hypothermia detected during the prodromal phase of laminitis is consistent with decreased digital vascular perfusion or metabolic activity. If administered to horses during the prodromal phase, agents that enhance digital perfusion may prevent development of laminitis. (*Am J Vet Res* 2001; 62:1167–1172)

Studies evaluating digital circulation in horses during the prodromal and acute phases of laminitis have yielded conflicting data. Following onset of lameness, digital blood flow has been reported to increase,[1,2] decrease,[3,4] or increase with a concomitant decrease in submural perfusion as a result of arteriovenous shunting.[3,5] Correlation and interpretation of these data are difficult because of the different experimental designs used in each study. It has also been hypothesized that digital hemodynamics vary markedly depending on the presence and severity of the pathologic cascade operative during the acute post-lameness phase of laminitis.[6] If this hypothesis is valid, it is possible that data from previous studies accurately reflect digital perfusion at different times during progression of laminitis.

Fewer reports describe digital hemodynamics during the prodromal or developmental phase of laminitis. Similar to data collected after development of lameness, results of these studies fail to allow a unified conclusion. Authors of 1 study, in which hoof wall temperature was measured as an indirect indicator of digital hemodynamic status, concluded that digital perfusion is not decreased in the prodromal phase of laminitis.[7] Based in part on these data, a hypothesis has been proposed that induction of vasoconstriction in the prodromal phase is protective, whereas vasodilatation predisposes to increased severity of laminitis.[8,9] On the other hand, data collected in our laboratory provide preliminary evidence that **hoof wall surface temperature (HWST)** decreases significantly 8 to 12 hours immediately prior to the first appearance of lameness in horses with experimentally induced laminitis.[6] Digital hyperthermia developed only after onset of lameness. Development of a low-flow or ischemic period during the prodromal phase of laminitis is also supported by reports of increased digital peripheral vascular resistance prior to development of lameness[10] and results of pharmacologic and histologic studies that suggest administration of agents that promote or enhance digital perfusion is warranted as a preventive treatment against laminitis.[11,12]

Given the diametrically opposed clinical implications of conclusions drawn from results of previous studies, it is important to determine which conclusion is valid. If the mechanisms involved during prodromal laminitis involve digital hypoperfusion, the use of vasoconstrictive agents would further reduce perfusion and increase severity of any ischemic effect. However, if vasodilation increases exposure of laminar epithelium to toxic agents, it is logical to assume that administration of a vasodilator during the prodromal phase of laminitis would be contraindicated.

Although these previous studies[6,7] were similar in that hoof wall temperature was assessed in both as a measure of digital blood perfusion, there were also important differences between the two. In one,[7] horses were maintained at an ambient temperature of 10 C, whereas in the second, ambient temperature was 19 C.[6] Additionally, some of the horses in the first study

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received analgesics for signs of distress associated with the induction of laminitis.[7] Data from that study were normalized to a common time (48 hours) following administration of the carbohydrate diet, whereas data from the second study[6] were normalized to the onset of lameness. Thus, it is possible that design differences were responsible for the opposing results observed and conclusions drawn from these studies.

Skin surface temperature is commonly used as a noninvasive indirect method to assess local perfusion.[12-14] However, the ability of this technique to detect changes in perfusion of the equine digit under varying environmental conditions has only been partially described.[15] Hoof wall surface temperature is the thermal equilibrium between the external temperature to which the hoof is exposed and the exothermic metabolic and mechanical activity and vascular supply of the underlying viable tissues. The specific heat of the stratum medium is not known at this time. Because of the relative thickness of the stratum medium, compared with the stratum corneum of skin, determination of HWST may be a less sensitive method to reflect changes in perfusion of underlying tissue, compared with determination of skin surface temperature. The effect of changing a single factor, such as blood flow, on HWST cannot be easily predicted because of the multiple physiologic interactions between ambient temperature and exothermic activities of the foot.

Because of differences between HWST and skin surface temperature measurements, and because of the clinical importance of conclusions drawn from data describing HWST during the prodromal and acute phases of laminitis, we chose to investigate the use of HWST as an indicator of digital perfusion in horses. Three experiments were performed. The first was designed to define the effect of ambient temperature on HWST of horses acclimatized to hot and cold environments. The second was designed to validate HWST as an indirect assessment of digital perfusion under moderate ambient temperatures. The third experiment was designed to determine whether digital hypothermia develops during the prodromal phase of carbohydrate-induced laminitis. The hypothesis tested in this third experiment was that digital hypothermia, consistent with decreased submural perfusion, is present during the prodromal phase of laminitis in horses.

Materials and Methods

Animals—Thirty adult horses with healthy feet were used; different horses were used in each experiment. Horses were considered to have healthy feet if the following criteria were met: no history of recent lameness, no abnormalities detected during a lameness examination, normal limb-load distribution as determined by use of force plate analysis, and no evidence of pathologic changes on lateromedial and dorsopalmar radiographic views of the digits. Limb-load distribution was quantified by use of a custom-designed computerized system consisting of 4 independent force plates.[16] This system allows quantification of the mean load, expressed as percentage of body weight, placed on each foot over a 5-minute period, using a data sampling rate of 0.1 seconds. Limb-load distribution was considered normal when the mean load placed on the forefeet was not different from established reference values for forefeet (mean ± SD, 28.69 ± 0.70%) and hind feet (21.32 ± 0.70%).[16]

For all experiments, horses were maintained outside in paddocks for at least 3 months prior to determination of HWST. For each experiment, horses were moved to a controlled laboratory environment, bedded on wood shavings, and allowed a minimum of 3 hours to acclimatize to the laboratory environment. Laboratory acclimatization was aimed at limiting psychogenic or stress effects on digital perfusion. During this period, each horse was instrumented for detection and recording of HWST. All experiments were completed following review and approval of the Laboratory Animal Care Committee at Texas A&M University.

Experiment 1—The first experiment was designed to define relationships between ambient temperature and HWST when horses were challenged by mild cold stress. Two groups of 6 horses each were acclimatized to different ambient temperatures. The first, identified as the hot-acclimatized cold-challenged group, was evaluated in August 1999 after horses were acclimatized to high ambient temperatures (mean ± SD, 28.7 ± 1.54 C) and relative humidities (66.86 ± 10.58%). The second group (cold-acclimatized cold-challenged) was evaluated in February 2000 after horses were acclimatized to low ambient temperatures (12.08 ± 3.36 C) and high relative humidities (75.00 ± 12.93%). After acclimatization, horses were moved to a controlled laboratory environment in which ambient temperature and humidity were maintained at 15.0 ± 1.8 C and 78%, respectively. Following acclimatization and instrumentation, HWST of both forefeet were assessed at 15-minute intervals for 1 hour. If HWST was near the ambient temperature or if digital hypothermia (HWST < 23 C) was detected, the data collection period was extended to determine the duration of digital hypothermia. The maximum data collection period was 6 hours.

Experiment 2—Six horses were used in the second experiment, which was designed to determine the ability of HWST to accurately reflect changes in digital perfusion. This experiment was completed in February 2000. Following acclimatization to the laboratory environment (ambient temperature, 19.0 ± 1 C; humidity, 75%), HWST were recorded at 15-minute intervals for 1 hour. The medial and lateral palmar digital arteries and veins in 1 randomly selected forefoot of each horse were then occluded for 30 minutes by placing a tourniquet around the metacarpophalangeal joint. The opposite forefoot served as the nontreated control. Recordings of HWST were continued at 10-minute intervals during the period of vascular occlusion. Digital perfusion was reestablished by removal of the tourniquet. During the 60-minute reperfusion period, HWST were recorded at 10-minute intervals. In addition, horses were examined for lameness or signs of discomfort secondary to initiation or discontinuation of vascular occlusion.

Experiment 3—Twelve horses that had been used as controls in a study of experimentally induced laminitis were used in the third experiment. Following instrumentation and acclimatization to the laboratory environment (ambient temperature, 19 ± 1 C; humidity, 75%), each horse was evaluated for 24 hours before (control period) and 72 hours after (experimental period) induction of laminitis by administration of a carbohydrate diet.[17] During both control and experimental periods, HWST were recorded and horses evaluated for signs of lameness at 4-hour intervals. Lameness was defined as a change in gait as determined by subjectively evaluating each horse's willingness to walk out of its stall onto a hard rubber-surfaced mat and then onto a hard flat concrete surface. The focus of the lameness evaluation was

the detection of the point that lameness first appeared and not severity of lameness; accordingly, lameness was not scored.

Measurement of hoof wall surface temperature—Skin surface thermistors[a] were placed on the dorsal surface of the hoof wall approximately one third of the distance from the coronet to the ground surface. This location approximates the middle of the dorsal parietal surface of the distal phalanx. Thermistors were isolated from the environment, using styrofoam pads (2 × 2-inch squares) secured with elastic tape. All thermistors were pre-calibrated to ensure a common baseline. The HWST recording system was capable of differentiating temperatures within ± 0.05 C. Thermistor leads were secured to the forelegs with light bandages.

Statistical analyses—In the first experiment, HWST were compared over time and between groups by use of repeated measures ANOVA. In the second experiment, data were plotted and tested by use of a repeated measures ANOVA followed by a Tukey honestly significant difference comparison of means. If results of ANOVA revealed significant differences, a Student paired *t*-test was used to determine whether HWST 10, 20, and 30 minutes after vascular occlusion and 10 and 20 minutes after reperfusion were different from those measured at the same times in the control foot. In addition, HWST of control and occluded feet prior to application of the tourniquet were compared by use of a repeated measures ANOVA.

In the third experiment, HWST and the time to onset of lameness were initially plotted against time and analyzed, using the time of administration of the carbohydrate diet as time 0. Subsequently, data for all horses were normalized to the onset of lameness, replotted, and reanalyzed. For normalization, onset of lameness was determined subjectively. For each data set, plots were visually examined for trends, and data were compared over time by use of a repeated measures ANOVA, with time and horse as factors and HWST as the dependent variable. For all tests, $P \leq 0.05$ was considered significant.

Results

Experiment 1—Hoof wall surface temperature of hot-acclimatized cold-challenged horses was significantly ($P < 0.001$) less than that of cold-acclimatized cold-challenged horses at all times during the 1-hour data collection period (**Fig 1**). Mean (± SD) HWST during this period in hot-acclimatized cold-challenged horses was 18.98 ± 0.22 C, whereas mean HWST in cold-acclimatized cold-challenged horses was 29.39 ±1.81 C. Significant changes in HWST were not detected during the first hour of data collection in either group. In addition, HWST was not significantly different among horses of the same group. Because of the relative digital hypothermia detected in hot-acclimatized cold-challenged horses, the data collection period was extended for this group. Five of these 6 horses subsequently developed a transient digital hyperthermia (**Fig 2**). Mean onset of hyperthermia in these 5 horses was 192 ± 91 minutes after initiation of data collection (372 minutes after being moved to the cold laboratory environment). Hyperthermia was transient and peaked some 90 minutes after its onset. In 4 of these 5 horses, digital hyperthermia involved a single digit. There was no apparent predilection for which forefoot became hyperthermic.

Experiment 2—Prior to initiation of vascular occlusion, mean HWST of the control digits was 29.36 ± 2.22 and that of the occluded digits was 29.45 ± 1.39 C. Mean difference in HWST between occluded and control digits in individual horses was 0.05 ± 1.31 C. During the control period, differences in HWST of occluded and control digits among horses were not significant ($P = 0.884$ and 0.55, respectively) over time.

Following application of the tourniquet, HWST rapidly decreased from control values (**Fig 3**). After 30 minutes of vascular occlusion, mean HWST in the occluded digit was 26.66 ± 1.01 C. Removal of the tourniquet resulted in a rapid increase of HWST in all horses. Within 30 minutes of reperfusion, HWST was at or greater than the baseline HWST recorded prior to occlusion. Mean HWST in the occluded digit during the ischemic and early reperfusion phases were significantly different from those in the control digit at the same times and from those in either digit measured during the late reperfusion phase. Mean HWST in the occluded digit 20 and 30 minutes after onset of ischemia and 10 minutes after onset of reperfusion were significantly ($P = 0.015$, 0.011, and $P < 0.001$, respectively) less, compared with control values measured at the same times. Following application of the tourniquet, signs of discomfort, including pawing and lifting and nuzzling the treated foot, were detected in

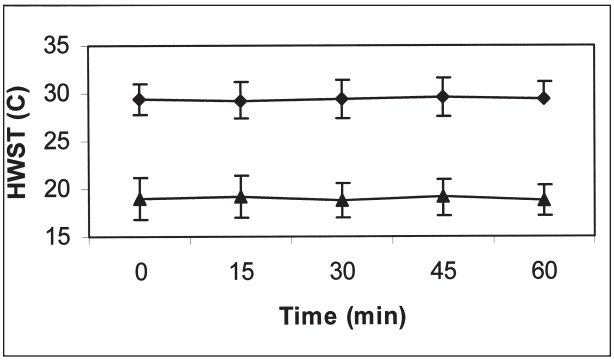

Figure 1—Hoof wall surface temperature (HWST) of hot- (triangles; n = 6) and cold-acclimatized (diamonds; 6) horses subjected to cold ambient temperature (15 C). Hoof wall surface temperatures were significantly ($P < 0.001$) different between groups at all times.

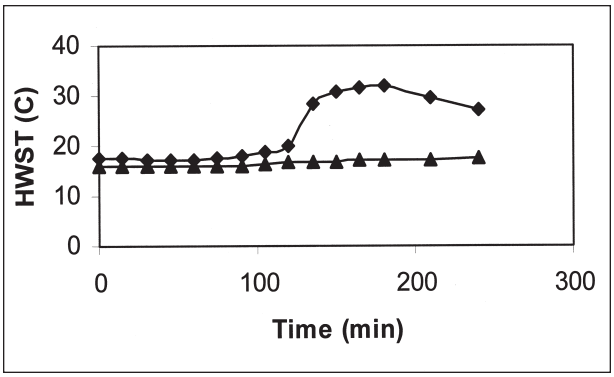

Figure 2—Hoof wall surface temperatures of the left (diamonds) and right (triangles) forefeet in a hot-acclimatized horse following exposure to cold ambient temperature (15 C). The increase in HWST in the left forefoot beginning at 120 minutes likely reflects an autoregulatory escape mechanism from cold-induced vasoconstriction.

all 5 horses. Signs of discomfort immediately ceased after removal of the tourniquet.

Experiment 3—Mean HWST during the baseline period prior to administration of the carbohydrate diet was 32.31 ± 01.32 C for the 12 horses acclimatized and maintained at an ambient temperature of 19 ± 1 C. At this ambient temperature, significant changes in HWST were not detected during the 24-hour control period. Similarly, administration of the carbohydrate diet induced no immediate changes in HWST. However, relative digital hypothermia developed 8 to 12 hours preceding the onset of lameness (**Fig 4**). Hypothermia was first detected at a mean of 10 ± 3 hours prior to detection of lameness. When data were normalized to time of carbohydrate administration, HWST did not significantly ($P = 0.095$) change over time. However, when data were normalized to the onset of lameness, changes in HWST were significant ($P = 0.007$). Twelve and 8 hours prior to onset of lameness, mean HWST normalized to onset of lameness were 29.71 ± 3.85 C and 30.02 ± 4.3 C, respectively, and were significantly ($P = 0.025$ and 0.033, respectively) less than mean HWST measured during the control period. At the onset of lameness, HWST was again not significantly different from the control value.

Initial signs of lameness were subtle and consisted of gait stiffness and reluctance to step onto hard surfaces. Mean time from administration of carbohydrate to onset of lameness was 33 ± 7.46 hours (range, 20 to 44 hours). However, lameness progressed in severity over the next 8 to 12 hours. Lameness characteristic of the acutely affected horses was rarely achieved before 12 hours following onset of lameness.

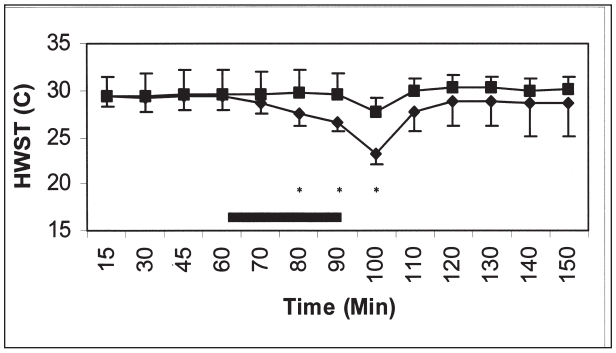

Figure 3—Mean ± SD HWST determined for 1 forefoot subjected to vascular occlusion (diamonds) and 1 control forefoot (squares) in 6 horses. The solid bar denotes the period of vascular occlusion. *Values significantly ($P < 0.05$) different between groups.

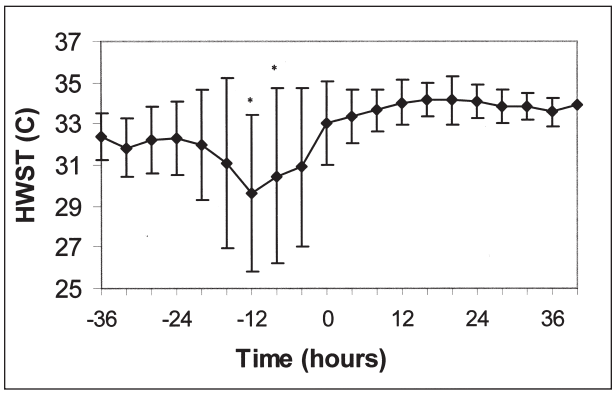

Figure 4—Mean ± SD HWST determined for 12 horses during the prodromal and acute phases of carbohydrate-induced laminitis. Data were normalized to the initial onset of lameness (time = 0). *Significantly ($P < 0.05$) less than the mean HWST recorded prior to carbohydrate administration.

Discussion

Results of our first experiment clearly indicated that the response of HWST to ambient temperature depends on the physiologic requirements of the digital submural tissues and the degree to which horses are acclimatized to the environment. The effects of thermal acclimatization were evidenced by the higher HWST in the cold-acclimatized cold-challenged group, compared with the hot-acclimatized group. The high mean HWST detected in cold-acclimatized horses exposed to a cold environment was interpreted to reflect a normal degree of digital perfusion that served to balance loss of heat from the foot. Although the mechanisms responsible for thermal acclimatization are not well delineated, such mechanisms designed to prevent digital heat loss need not be activated in cold-acclimatized horses because of accommodations already in place (eg, a heavy coat).

Exposure of hot-acclimatized horses to cold ambient temperatures resulted in a significantly lower HWST. Two factors may have contributed to this low HWST. The first of these is the physiologic effort required to preserve core body temperature by decreasing cutaneous heat dissipation. Hot-acclimatized horses lack the accommodations to cold ambient temperatures that develop in cold-acclimatized horses. Lack of accommodation to cold ambient temperatures facilitates heat loss during cold challenge and mandates that other physiologic mechanisms be activated to maintain core body temperature. In the equine foot, this can be accomplished by peripheral vasoconstriction, intradigital arteriovenous shunting of digital blood away from the hoof wall, or countercurrent heat exchange in the extended laminar microcirculation. Activation of any of these mechanisms would result in low HWST. We believe that the increase in HWST detected in hot-acclimatized horses at later time points is evidence of an autoregulatory escape mechanism from cold-induced vasoconstriction. Physiologically, this vasodilatation may be triggered by an increase in concentrations of factors produced by metabolically active submural tissues in the absence of adequate vascular perfusion. Whether such vasodilatation is mediated locally or centrally is not known. A similar autoregulatory escape mechanism has been described in horses maintained in an extremely cold environment[7] and provides evidence that the submural tissues of the foot remain metabolically active even at low ambient temperatures.

Another factor contributing to low HWST in hot-acclimatized cold-challenged horses is the impact of ambient temperature on the thermal equilibrium of the foot. Hoof wall surface temperature represents the thermal equilibrium between the ambient temperature to which the foot is exposed and internal exothermic heat sources. As the magnitude of the thermal gradient

between external and internal temperatures increases, the ambient temperature begins to dominate HWST, thus reducing the ability of HWST to reflect alterations in digital perfusion. At high ambient temperatures, the ability of HWST to reflect changes in digital vascular perfusion decreases. Hoof wall surface temperature increases as ambient heat is absorbed, and at high ambient temperatures, this precludes detection of further increases secondary to vasodilatation. Likewise, the ability to detect a significant decrease in HWST secondary to vasoconstriction at high ambient temperatures would be lessened, because HWST cannot decrease to less than the ambient temperature.

At low ambient temperatures, the same concepts apply. The ability of HWST to reflect decreased vascular perfusion under such conditions would be compromised by 3 factors: an increased thermal gradient causing loss of heat to the environment, decreased digital perfusion, and decreased metabolic activity of viable tissue. Alternatively, increased digital perfusion is easy to detect at low ambient temperatures, because the thermal equilibrium of the foot changes in response to increased blood flow. This was evidenced by detection of an increase in HWST in 5 of the 6 hot-acclimatized cold-challenged horses in our study, presumably as a result of activation of an autoregulatory escape mechanism.

Results of our first experiment also clearly indicate that, if HWST is to be used to evaluate digital vascular perfusion, HWST must be measured at moderate ambient temperatures. Moreover, the optimal temperature for determination of HWST must be defined in relation to the degree of environmental acclimatization of each horse.

Results of our second experiment validate the hypothesis that, at moderate ambient temperatures, HWST is capable of reflecting acute sustained changes in digital perfusion. Thus, use of HWST as an indirect assessment of digital perfusion under appropriate conditions is valid. These results also allow insight into limitations of the use of HWST in horses. Determination of HWST is not a highly sensitive method for detecting acute changes in digital vascular perfusion. If the assumption is made that application of the tourniquet resulted in an immediate decrease in digital perfusion, the resultant decrease in HWST was somewhat delayed; HWST in the occluded foot became significantly less than that of the control foot only after 20 minutes of occlusion. This lag time was likely attributable to the time necessary for the stratum medium to dissipate heat into the environment. Although this lack of sensitivity somewhat limits the use of HWST as an index of acute changes in digital vascular perfusion, it does not adversely affect its potential usefulness in studies evaluating sustained alterations in digital perfusion.

The signs of discomfort that we detected in all horses following application of the tourniquet may have developed as a result of irritation attributable to pressure of the tourniquet on the forefoot. Alternatively, signs of discomfort may have reflected a decrease in digital perfusion or an increase in submural pressure associated with a compartment injury.

In our third experiment, digital hypothermia developed in horses during the prodromal phase of carbohydrate-induced laminitis; digital hyperthermia developed only after the onset of lameness. Severity and duration of the prodromal hypothermia in individual horses was variable, as was severity of lameness. At the time lameness first became evident, HWST had returned to control values. Given the nonspecific nature of HWST, it is possible that prodromal digital hypothermia and postlameness hyperthermia represent changes in digital perfusion or metabolic activity of the submural tissues. Regardless of the cause, detection of prodromal digital hypothermia indicates that pathogenic mechanisms leading to the development of laminitis are initiated prior to onset of lameness.

Results of pharmacologic studies provide circumstantial evidence that supports our conclusion that digital hypothermia reflects a decrease in digital vascular perfusion. Specifically, clinical trials of agents that enhance digital perfusion limit the development or severity of laminitis in horses when administered as preventives.[6,11] Although we assumed that prodromal digital hypothermia reflected a decrease in vascular perfusion, our results do not allow identification of the mechanism responsible for this decrease. Vasconstriction,[6,10] vascular occlusion,[18,19] or countercurrent heat exchange[6] in the extended submural microcirculation as a result of a low-flow state would all result in decreased HWST. Similarly, detection of digital hyperthermia after onset of lameness is compatible with an increase in perfusion associated with a reflex hyperemia and inflammatory vasodilation.

Alteration in the metabolic activity of the viable submural tissues cannot be totally dismissed as a potential cause of digital hypo- and hyperthermia. Biochemical reactions essential to cornification are, like most biochemical reactions, exothermic and contribute to overall HWST. Thus, it is possible that during the prodromal phase of laminitis, some agent or factor was acting to decrease the metabolic rate of submural tissue. Metabolic activity increases again during the acute phase of laminitis, resulting in digital hyperthermia. However, metabolic heat is likely to offer only a limited contribution to the thermal equilibrium of the hoof. The magnitude of heat produced during cornification is not known but is intuitively small given the low metabolic rate of cutaneous tissues.

Our conclusions in the present study are distinctly different from those of a previous study[7] in which a decrease in digital perfusion and, hence, HWST were not detected during the prodromal phase of laminitis. A major difference between these 2 studies is the ambient temperature at which experiments were completed. It is likely that the low ambient temperature used in the previous study precluded the ability to detect prodromal digital hypothermia.

When data obtained in the third experiment were normalized to time of administration of the carbohydrate diet, we did not detect significant changes in HWST over time. This was attributed to the biologic variation in the rate at which laminitis developed among individual horses. Mean time for development of lameness from the time of carbohydrate administration was 33 ± 7.46 hours (range, 20 to 44 hours).

Because digital hypothermia consistently developed 8 to 12 hours prior to lameness, normalization relative to diet administration had the effect of compiling data at times when individual horses were normothermic (prior to onset of hypothermia or at onset of lameness), hypothermic (immediately prior to onset of lameness), or hyperthermic (after onset of lameness). Alternatively, normalization of data to onset of lameness allowed a better correlation of individual horses in the same phase of laminitis. Normalization of data to onset of lameness may be a satisfactory method for reducing the number of horses required to detect significant differences in research studies of laminitis.

There was no apparent correlation between magnitude or duration of digital hypothermia and severity of lameness. This was reflected in the magnitude of the standard deviation associated with mean HWST measured during the prodromal phase. Magnitude of HWST decrease during this phase may be affected by multiple factors, including differences in severity of laminitis and degree of acclimatization to the ambient temperature as well as variations in the physiologic response of each horse to the inducing agent (ie, carbohydrate overload) and the relative accuracy in pinpointing the onset of lameness. Although the impact of some of these factors can be reduced via experimental design, it is questionable whether HWST can be used as an accurate predictor of impending lameness, especially under noncontrolled conditions.

[a]Thermistor 08442-15, Cole-Parmer Instrument Co, Vernon Hills, Ill.

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