Voluntary limb-load distribution in horses with acute and chronic laminitis

David M. Hood, DVM, PhD; Ilka P. Wagner, DVM, MAg; Danny D. Taylor, MS;
Gordon W. Brumbaugh, DVM, PhD; M. Keith Chaffin DVM, MS

Objectives—To compare limb-load distribution between horses with and without acute or chronic laminitis.

Animals—10 horses with carbohydrate-induced acute laminitis, 20 horses with naturally occurring chronic laminitis, and 20 horses without foot abnormalities (controls).

Procedures—Limb-load distribution was determined, using a custom-designed system that allowed simultaneous quantification of the mean percentage of body weight voluntarily placed on each limb (ie, mean limb load) and the SD of the mean load over a 5-minute period (ie, load distribution profile [LDP]). Load distribution profile was used as an index of frequency of load redistribution.

Results—Mean loads on fore- and hind limbs in control horses were 58 and 42%, respectively, and loads were equally and normally distributed between left and right limbs. In addition, forelimb LDP was greater, compared with hind limbs, and was affected by head and neck movement. In comparison, limb-load distribution in horses with chronic laminitis was characterized by an increase in the preferential loading of a forelimb, a decrease in total forelimb load, and an increase in LDP that was correlated with severity of lameness. In horses with carbohydrate-induced acute laminitis, mean limb loads after onset of lameness were not different from those prior to lameness; however, LDP was significantly decreased after onset of lameness.

Conclusion and Clinical Relevance—Quantification of limb-load distribution may be an applicable screening method for detecting acute laminitis, grading severity of lameness, and monitoring rehabilitation of horses with chronic laminitis. (*Am J Vet Res* 2001; 62:1393–1398)

The most frequently used criterion to judge disease severity and therapeutic response in horses with laminitis is discomfort expressed by the horse.[1,2] Expressed discomfort, in turn, has been interpreted as pain and subjectively graded on the basis of changes in gait or stance.[3,4] Various scales, such as the Obel[5,a] and clinical[6] grading systems, attempt to make this assessment less subjective. However, the reliability of these scoring systems depends on the training, perception, objectivity, skill, and number of individuals doing the evaluation. Further complicating the use of expressed discomfort as a clinical index of severity of laminitis are the differences in pain tolerance and learned behavioral responses to discomfort by individual horses.[2,5,6] Moreover, not all changes in gait or stance are pain-related.

The stance assumed by horses with severe forefoot laminitis is considered pathognomonic; both the hind and forefeet are placed forward of their normal positions.[6] It is believed that this stance shifts some weight from the fore- to the hind feet and, thus, may serve as a mechanism to decrease pain.[3,4,7,8] It follows that measurement of limb load in horses with laminitis might be a sensitive and objective means of grading disease severity and therapeutic response. The purpose of the study reported here was to characterize and compare limb-load distribution between horses with and without acute or chronic laminitis. The hypothesis tested was that limb-load distribution can be quantified and used as an index of expressed discomfort in affected horse.

Materials and Methods

Animals—Fifty horses were evaluated in 2 experiments. In the first, limb-load distribution was evaluated before and after induction of acute laminitis by feeding a carbohydrate diet[9] to 10 horses without foot abnormalities. Horses for this experiment had been used as controls in a pharmacologic intervention study.[10] In the second experiment, 20 horses with chronic laminitis and 20 unaffected horses were evaluated. Horses with chronic laminitis were obtained from several sources, making it impossible to define the exact duration of disease in all horses. During the brief period of the study, the clinical status of all horses with chronic laminitis was judged to be static. Affected horses were between 4 and 20 years old and represented a number of breeds (7 Quarter Horses, 7 Arabians, 2 Appaloosas, 1 Thoroughbred, 1 Paint, 1 Welsh/Thoroughbred cross, and 1 Tennessee Walking Horse) and both sexes (12 sexually intact females, 7 castrated males, and 1 sexually intact male). Median Obel lameness grade was 2, and median clinical score was 3; most (11/20) horses were moderately affected (Obel grade of 2 or 3; clinical score of 3 or 4). Unaffected horses served as the control group; these horses were obtained from the herd of clinically normal horses maintained by The Hoof Project and were matched with the chronically affected horses for age and breed.

Horses without foot abnormalities were included in the study if they met the following criteria: no history of recent lameness, no abnormalities detected during a clinical lameness examination, and no evidence of abnormalities on lateromedial and dorsopalmar radiographic views of any digit. Inclusion criteria for horses with chronic laminitis included a clinical diagnosis of bilateral laminitis of at least 3 months' duration and radiographic evidence of displacement of the distal phalanx relative to the hoof wall in both forefeet.

Received Sep 11, 2000.
Accepted Dec 21, 2000.
From the Departments of Veterinary Physiology (Hood, Wagner, Taylor, Brumbaugh) and Large Animal Medicine and Surgery (Chaffin), College of Veterinary Medicine, Texas A&M University, College Station, TX 77843-4466; and the Hoof Diagnostic and Rehabilitation Clinic, PO Box 10381, College Station, TX 77842 (Hood).
Supported by The Hoof Project Foundation, College Station, Tex, and private funds.

Capsular rotation, phalangeal rotation, and vertical displacement of the distal phalanx were considered equal criteria of phalangeal displacement.

To limit potential effects of shipping, all horses were allowed a minimum of 1 week to acclimatize to the laboratory environment. During this period all horses were trained to stand calmly in the measurement stanchion for 15 minutes while loosely cross-tied to restrict movement of their heads and necks. In experiment 1, horses were not cross-tied, whereas in experiment 2, all horses were loosely cross-tied for data collection. Tranquilization or other forms of chemical restraint were not used. During the acclimatization period, chronically affected horses were weaned off nonsteroidal anti-inflammatory drugs if possible. Horses that we were unable to wean were treated with phenylbutazone (2 mg/kg of body weight, PO, q 12 h) for the week prior to assessment. Experimental protocols were reviewed and approved by the Laboratory Animal Care Committee at Texas A&M University.

Experiment 1—The limb-load distribution of horses with acute laminitis was defined by comparing voluntary limb loading of 10 horses without foot abnormalities prior to and at the onset of lameness resulting from development of carbohydrate-induced laminitis. Prior to administration of the carbohydrate diet, horses were evaluated subjectively and by use of the force-plate system at least 5 times at 4-hour intervals. Following administration of the diet, horses were evaluated subjectively at 4-hour intervals for signs of lameness. At the first indication of lameness, force-plate analysis was performed. All horses were subjectively evaluated for lameness for the 4 to 8 hours following onset of lameness.

Lameness was determined by subjectively evaluating each horse's willingness to walk out of its deeply bedded stall (shavings) onto a firm rubber-surfaced mat and then onto a hard flat concrete floor. Each horse was then positioned in a stanchion on a firm rubber floor for 5 to 6 minutes and reexamined as it was walked back to its stall. Willingness to turn sharply to the left and right was also assessed. The goal of the subjective lameness analysis was to identify the onset of lameness. Accordingly, any reluctance to walk from the stall, any change in gait, or any stiffness in turning was defined as lameness. All subjective evaluations were performed by the same person (DMH), and if evidence of lameness at any given time was noted as questionable, that horse was reassessed 4 hours later. An increase in number or severity of signs of lameness during the latter evaluation confirmed that the horse was lame at the former evaluation.

Experiment 2—Limb-load distribution of horses with chronic laminitis was described by comparing voluntary limb loading of 20 control horses with that of 20 horses with naturally occurring chronic laminitis. On the day of limb-load evaluation, lameness was clinically assessed[6] and assigned an Obel grade[5,a] and a clinical score[5] by 1 of the authors (DMH). The order in which horses were evaluated was dependent on the date they were entered in the study.

Determination of limb-load distribution—Limb-load distribution was quantified by use of a custom-designed computerized system consisting of 4 independent force plates.[11] This system allows quantification of the mean load, expressed as percentage of body weight, placed on each limb over a 5-minute period, using a data sampling rate of 0.1 seconds. Each rectangular force plate had a load cell mounted in each corner that was capable of measuring forces from 0 to 500 kg. Voltages from the load cells on each plate were summated, and the summated signal, which represented the force on each plate and thus the load on each limb, was digitized by a computer-based measurement system. This arrangement allowed accurate detection of the load on each limb independent of hoof placement on the force plate. Task-specific software facilitated calibration (from 0 to 200 kg) and data collection. Data were collected while horses were restrained in a stanchion built around the force plate system.

During data collection, the software sampled the loads on each plate every 0.1 seconds, summated the loads from each plate to determine the total load for each horse, and calculated and plotted the mean percentage of the total load placed on each limb (ie, limb load). In addition, the software also calculated the SD of the mean of the load data for each limb collected during the data collection period. The data collection process was automated so that the operator was only able to intervene by pausing data collection to allow repositioning of the digits should a horse step off a plate.

During data collection, the mean percentage of the total load that was placed on each limb (ie, limb load) and the frequency distribution and SD of the limb load were displayed and recorded. Examination of the raw data indicated that the temporal distribution of the load placed on a limb, as depicted by the frequency distribution of the limb load, was symmetrical around the mean of the load data collected during the data collection period and appeared to vary proportionally with severity of lameness. Accordingly, the SD of the limb load was used as an index of limb-load variation (ie, frequency of load redistribution) and was referred to as the **load distribution profile (LDP)**.

Statistical analyses—In experiment 1, mean left and right limb loads for fore- and hind limbs determined immediately prior to administration of the carbohydrate diet (control period) were compared by use of paired *t*-tests with values determined at the onset of lameness. Left and right LDP of fore- and hindlimbs determined during the control period were also compared by use of paired *t*-tests with data obtained at the onset of lameness. In experiment 2, mean limb load and LDP were compared between left and right forelimbs and between fore- and hind limbs in each group (ie, control horses and horses with chronic laminitis) by use of an unpaired *t*-test. Mean limb load and LDP of the control horses in experiment 2 and the control data in experiment 1 were compared, using an unpaired *t*-test. Comparisons (unpaired *t*-test) were also made of the LDP of the chronically affected horses in experiment 2 and the LDP data recorded at the time of first lameness in experiment 1. Because limb-load data for horses with chronic laminitis were nonparametric, mean limb load was compared between groups by use of a Mann-Whitney rank-sum analysis, and a Kruskal-Wallis test was used to determine the significance of the association between clinical score or Obel grade and LDP or limb load. The mean difference in limb load between left and right fore- or hind limbs was calculated by subtraction for each horse. Correlations between Obel lameness grade and mean forelimb load or LDP and between clinical lameness score and mean forelimb load or LDP were determined by use of Spearman rank correlation analyses. For all tests, $P \leq 0.05$ was considered significant.

Results

Acute laminitis—The initial appearance of lameness in horses with acute laminitis was subtle, consisting of stiffness of gait and reluctance to step onto hard surfaces. Mean (± SD) time between administration of the carbohydrate diet and onset of lameness was 33 ± 7.46 hours (range, 20 to 44 hours). Following its appearance, severity of lameness was progressive over the next 4 to 8 hours.

During the control period, mean forelimb load

(57.50 ± 1.6%) was significantly ($P < 0.001$) greater than hind limb load (42.42 ± 1.5%). However, loads were equally distributed between left and right limbs, because left limb loads (forelimb, 28.63 ± 2.6%; hind limb, 20.66 ± 2.6%) were not significantly (forelimb, $P = 0.869$; hind limb, $P = 0.453$) different from right limb loads (forelimb, 28.88 ± 2.3%; hind limb, 21.76 ± 2.1%). At the onset of lameness, mean fore- and hindlimb loads (57.40 ± 1.8% and 42.59 ± 1.8%, respectively) were not significantly ($P = 0.768$ and $P = 0.6015$, respectively) different from loads determined during the control period.

Mean forelimb LDP during the control period (6.78 ± 2.6) was significantly ($P = 0.007$) greater than mean hindlimb LDP (4.21 ± 2.0), indicating that frequency of load redistribution between left and right forelimbs was greater, compared with hindlimbs. At the onset of lameness, fore- and hind limb LDP had significantly decreased to 3.54 ± 1.3 ($P = 0.005$) and 1.98 ± 1.4 ($P = 0.001$), respectively. However, this decrease in LDP appeared transient, because LDP rapidly increased in individual horses as the lameness became more severe over the subsequent 4 to 8 hours.

Chronic laminitis—Mean forelimb load of control horses (58.09 ± 1.3%) was significantly ($P < 0.001$) greater than mean hind limb load (41.91 ± 1.3%). These values were not significantly different from data collected during the control period in experiment 1. Again, similar to values determined during the control period in experiment 1, left limb loads (forelimb, 28.93 ± 1.3%; hind limb, 20.77 ± 2.3%) were not significantly (forelimb, $P = 0.996$; hind limb, $P = 0.569$) different from right limb loads (forelimb, 28.92 ± 1.7%; hind limb, 21.34 ± 2.6%). Mean forelimb LDP (4.24 ± 1.6) in control horses was significantly ($P = 0.002$) greater than hind limb LDP (2.87 ± 2.2). However, LDP between left (forelimb, 4.26 ± 1.6; hind limb, 2.77 ± 2.1) and right (forelimb, 4.20 ± 1.5; hind limb, 2.98 ± 2.2%) limbs did not differ.

Despite having bilateral chronic forelimb laminitis, mean forelimb load (54.53 ± 2.8%) of affected horses was significantly ($P < 0.001$) greater than mean hindlimb load (45.05 ± 2.9%). Moreover, mean loads did not differ significantly between left and right limbs (left forelimb, 26.93 ± 4.7%; right forelimb, 27.73 ± 4.1%; left hind limb, 22.24 ± 2.8%; right hind limb, 23.09 ± 2.6%). However, individual horses did prefer bearing weight on 1 forelimb; the mean difference in left-to-right forelimb load in affected horses (6.62 ± 5.1%; range, 0.4 to 20.89%) was significantly ($P < 0.001$) greater than that in control horses (2.34 ± 1.9%; range, 0.09 to 5.41%). Calculations made on the basis of body weight indicated that the mean weight that affected horses shifted between forefeet was 28.66 ± 22.4 kg (range, 22.99 to 99.65 kg). However, there was no preference for right or left forelimb loading; 7 horses preferentially loaded the left forelimb, 8 preferentially loaded the right forelimb, and 5 did not preferentially load either forelimb. When preferential loading of 1 forelimb was detected, it was accompanied by simultaneous unloading of the opposite forelimb. Mean forelimb LDP of affected horses (8.93 ± 3.1) was significantly ($P < 0.001$) greater than mean hind limb LDP (5.31 ± 2.7); no significant differences in LDP were found between left and right limbs (left forelimb, 9.02 ± 2.9; right forelimb, 8.99 ± 3.4; left hind limb, 5.56 ± 2.8; right hind limb, 5.24 ± 2.6).

Mean forelimb load was significantly ($P < 0.01$) less and hind limb load significantly ($P < 0.01$) greater in horses with chronic laminitis, compared with control horses (forelimb load, 54.53 ± 2.8 vs 58.09 ± 1.3%; hind limb load, 45.05 ± 2.9 vs 41.91 ± 1.3%). Moreover, the mean difference in fore-to-hind limb load of affected horses (9.38 ± 5.31%; range, –1.68 to 17.10%) was significantly less than that of control horses (16.18 ± 2.6%; range, 11.62 to 22.22%). Calculations made on the basis of body weight indicated that the mean weight that affected horses shifted from the fore- to the hind limbs was 13.67 ± 12.2 kg (range, -2.57 to 39.83 kg). Mean fore- and hind limb LDP of horses with chronic laminitis (forelimb, 8.93 ± 3.1; hind limb, 5.31 ± 2.7) were significantly (fore limb, $P < 0.001$; hindlimb, $P = 0.029$) greater, compared with control horses.

Obel lameness score **(Fig 1)** and clinical grade **(Fig 2)** were inversely related to mean forelimb load and directly related to LDP. Spearman rank correlation analysis indicated that a decreasing forelimb load was associated with an increasing Obel grade ($r = -0.5147$) and clinical score ($r = -0.6424$), whereas an increasing

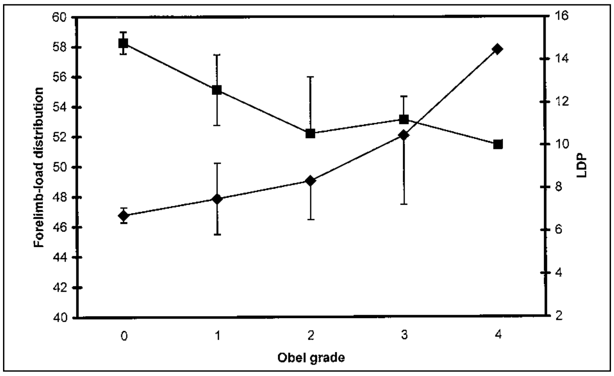

Figure 1—Forelimb-load distribution (% of body weight; squares) and load distribution profile (LDP; SD of the mean load during the data collection period; diamonds) versus Obel lameness grade in 20 horses with chronic bilateral forelimb laminitis.

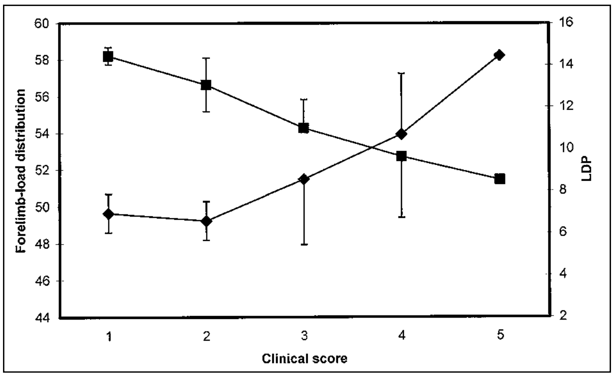

Figure 2—Forelimb-load distribution and LDP versus clinical score in 20 horses with chronic bilateral forelimb laminitis. *See* Figure 1 for key.

LDP was associated with an increasing Obel grade (r = 0.5941) and clinical score (r = 0.6236). However, results of ANOVA indicated that mean forelimb load and LDP did not vary significantly when Obel grade (P = 0.0659 and P = 0.0900, respectively) or clinical score (P = 0.0921 and P = 0.0574, respectively) was used as the categorical variable.

Discussion

Results from the present study indicated that horses without foot abnormalities voluntarily placed 58 and 42% of their body weight on the fore- and hind limbs, respectively, with the load equally distributed between the left and right limbs. These data are consistent with those of previous reports.[12-14] Our results were the mean of approximately 682 data points/limb collected over 5 minutes, whereas previous results reflect single or averaged multiple measurements over briefer periods. Mean limb loads were not different between control and healthy horses prior to administration of a carbohydrate diet. For horses resting in a diagonal stance, mean fore- and hind limb loads are still 58 and 43%, respectively, but the load is not distributed equally between left and right limbs.[11]

As defined in the present study, LDP is a simple index of the frequency of limb-load redistribution over the 5-minute data collection period. By definition, the SD of load on any given limb is a calculated index of dispersion of load data collected during the data collection period relative to the mean load. Given that the dispersion of load data was symmetrical around the mean, SD also serves as an indicator of how still a foot is on the plate. The term LDP was chosen to distinguish the SD of the load on each limb during the 5-minute data collection period from the SD of the mean limb loads for each group.

In control and healthy horses prior to administration of the carbohydrate diet, LDP of the forelimbs was greater than that of the hind limbs. This reflects that in horses without foot abnormalities, the forelimbs were subjected to a greater frequency of loading and unloading than the hind limbs. The increased forelimb LDP can be attributed to changes in the position of the anterior portion of the trunk that accompany and counterbalance movements of the head and neck. Characteristically, as a horse moves its head to look in any given direction, load would increase on the opposing limb.[12] The LDP of healthy horses prior to administration of the carbohydrate diet was greater than that for the control group in experiment 2; however, this difference was not significant (P = 0.091) and was thought to reflect a difference in training between groups. In addition, horses in experiment 1 were not cross-tied during data collection, which allowed these horses greater freedom to move their heads and shift their weight from side to side.

At the onset of lameness, mean fore- and hind limb loads in horses with carbohydrate-induced laminitis were not different from loads recorded during the control period or for control horses in experiment 2. Instead, onset of lameness was characterized by a transient decrease in the frequency of loading and unloading of the limbs, which was reflected in a decrease in LDP. Intuitively, these data are compatible with the detection of an unwillingness of affected horses to move because of the initial discomfort associated with pathologic changes to the digit or systemic changes associated with carbohydrate overload. Because the onset of lameness in these horses typically occurred when they were recovering from the gastrointestinal insult imposed by carbohydrate overload, we believed that the transient decrease in LDP was related to digital discomfort. As such, a decrease in LDP may be useful for identifying the initial onset of lameness in horses with acute laminitis. If a decease in LDP reflects a pain-induced unwillingness to move, it implies that pain is present in all 4 feet during the acute phase of laminitis. This implication is distinct from the typical hypothesis that there is a preferential involvement of the forefeet as laminitis progresses past the peracute phase. An obvious explanation for this latter hypothesis is that the higher loads placed on the forelimbs of healthy horses predisposes the forefeet to collapse, compared with the hind feet.

Limb-load distribution in horses with chronic laminitis was significantly different from limb-load distribution in control horses or horses with acute laminitis. Limb-load distribution in horses with chronic laminitis was characterized by a reduction in forelimb load, a nonuniformity of left-to-right load distribution, and an increase in LDP. The shift in weight from the fore- to the hind limbs was relatively small, representing only 3 to 4% of body weight (mean ± SD, 13.67 ± 12.2 kg). The physiologic basis and clinical relevance of fore-to-hind limb-load distribution in horses with chronic laminitis is unknown at this time. It is possible that the shift in loading from the fore- to hind limbs was associated with a pain-induced alteration in the stance whereby the forelimbs were partially unloaded. Alternatively, this shift in load may reflect the loss of mass of anterior body structures, such as the pectoral muscles, as a result of disuse atrophy and hypertrophy of the hindquarters, which is commonly observed in horses with a history of prolonged laminitis.

Even though selection of horses for inclusion in the chronic laminitis group was dependent on a diagnosis of bilateral forefoot laminitis, most horses in this group preferentially loaded 1 forelimb. If magnitude of voluntary forelimb load is related to pain aversion, it follows that the forelimb with the smallest limb load is more painful than the limb that is preferentially loaded. Our data infer that even when bilateral, chronic laminitis is often more severe in 1 foot. That the magnitude of the load (weight) shifted between opposing forelimbs was more than twice the magnitude of the load shifted from the fore- to the hind limbs indicated that horses can more readily shift weight between forelimbs than between fore- and hind limbs. This interpretation is consistent with results of a previous study[11] indicating that in horses without laminitis or other foot abnormalities, unloading a forelimb results in either an increased loading of the opposite forelimb or an increased loading of the opposite fore- and ipsilateral hind limbs. Except for brief periods during motion, horses may be physically incapable of shifting substantial load (weight) to the hind limbs.

Determination of mean load for both right and left forelimbs may thus be a useful method for detecting and defining the relative severity of disease in horses with bilateral chronic forelimb laminitis.

Load distribution of chronically affected horses was also characterized by an increased frequency of loading and unloading of both the fore- and hind limbs. This was reflected by increases in fore- and hind limb LDP, compared with the control group. Observation of affected horses during the data collection period revealed that horses with high LDP sequentially loaded 1 forelimb while unloading the opposite limb. Examination of the frequency distribution of load on individual limbs during the data collection period revealed that load data were symmetrical around the mean limb load for that horse. Thus, the increase in LDP in chronically affected horses was likely a result of shifting the load (weight) off of 1 forelimb while simultaneously loading the opposite limb. Intuitively, an increase in LDP is the consequence of or represents a mechanism by which affected horses attempt to limit digital discomfort. Again, if limb unloading in horses with chronic laminitis is attributable to digital pain, and if unloading a forelimb is accompanied by loading the contralateral limb, it follows that the unloading of 1 forelimb will result in increased discomfort in the opposite limb. Thus, as the opposite limb is loaded, a point is reached at which the horse seeks to shift weight off (unload) that limb because of increasing discomfort. The end result is the pain-induced cycling of load between forelimbs. This pattern was partially disrupted in those horses that preferentially loaded a single forelimb. In that situation, LDP for both forelimbs were decreased, compared with control values. Regardless of the mechanism, the high correlation between severity of lameness (ie, Obel grade or clinical score) and LDP implies that LDP is a valid index of expressed lameness in horses with chronic laminitis, particularly when lameness is bilaterally symmetrical.

The physiologic importance of loading and unloading limbs is not necessarily limited to relief of discomfort. Three observations are germane to the clinical implications of these data. First, increasing load decreases digital submural perfusion.[15] Second, at the onset of initial lameness in horses with acute laminitis, foot perfusion is increased secondary to reperfusion hyperemia.[16-19] Finally, the submural laminar interface is weakened as a consequence of the pathogenic changes associated with laminitis.[20] Together these observations imply that there is little benefit and a potential risk to walking a horse with acute laminitis in an attempt to increase digital perfusion. Likewise, the high LDP that we observed in chronically affected horses implies that walking such horses in an attempt to increase blood flow through the foot is not necessary.

Two subjective scores were used in an effort to achieve a better description of disease severity. The Obel grade is the classic lameness index used to assess severity of laminitis. The clinical grading system that we used describes the relative athletic function of a horse with chronic laminitis. Mean forelimb load and LDP were associated with these subjective lameness indices. However, these associations were not significant when the subjective scales were used as categorical values in a Kruskal-Wallis analysis. The lack of a significant association may relate to several factors. The Obel grading system is a subjective ordinal system in which the scalar criteria allow a wide range of degrees of disease severity to be assigned a common discrete value. That is, a group of horses described as grade-2 lame may represent horses with clinically relevant differences in disease severity. The objective indices (eg, forelimb load and LDP) are, however, numerical data that reflect a continuous change relative to disease severity. Thus, when these numerical values are grouped under a single category, a wide SD of the numerical values is created.

A number of disease-related variables may also impact the ability of limb load and LDP to predict severity of disease. Duration of forelimb laminitis can affect forelimb load; horses affected for prolonged periods will have had more time to learn how to preferentially increase load to decrease discomfort than horses more recently affected. In addition, fore- and hind limb musculature will change over time so as to increase hind limb load. Likewise, in horses with bilateral laminitis, variability in disease severity between forefeet can affect forelimb load and LDP. In future studies that evaluate the use of limb-load distribution in horses, these factors must be considered in data analysis and interpretation. However, results of the present study do suggest that quantification of limb-load distribution may be of value for detecting the onset of lameness attributable to acute laminitis (detected as a decrease in LDP of the affected limbs), assessing relative disease severity in horses with bilateral laminitis (detected as a difference in mean limb load between forelimbs), describing severity of disease in horses with bilateral laminitis (detected as an increase in LDP), and monitoring rehabilitation of horses with chronic laminitis (detected as changes in mean limb load and LDP over time).

[a]Obel N. *Studies on the histopathology of acute laminitis*. PhD dissertation, Almqvist & Wiksells Boktryckeri AK, Uppsala, Sweden, 1948.

References

1. Hood DM. Prognosis of chronic laminitis, in *Proceedings*. 6th Congr Med Chirurgie Equine 1999;44–53.
2. Hood DM, Grosenbaugh DA, Chaffin MK, et al. Pathophysiology of chronic laminitis, in *Proceedings*. 39th Annu Meet Am Assoc Equine Pract 1993;199–200.
3. Linford RL. Laminitis (founder). In: Smith BP, ed. *Large animal internal medicine*. St Louis: CV Mosby Co, 1990;1158–1168.
4. Stashak TS. Lameness. In: Stashak TS, ed. *Adams' lameness in horses*. 4th ed. Philadelphia: Lea & Febiger, 1987;486–499.
5. Morgan SJ, Grosenbaugh DA, Hood DM. The pathophysiology of chronic laminitis: pain and anatomic pathology. *Vet Clin North Am Equine Pract* 1999;15:395–417.
6. Herthel D, Hood DM. Clinical presentation, diagnosis, and prognosis of chronic laminitis. *Vet Clin North Am Equine Pract* 1999; 15:375–394.
7. O'Grady SE. A practical approach to treating laminitis. *Equine Pract* 1993;9:867–875.
8. Pollitt CC. The pathophysiology of equine laminitis. In: Petersen GV, ed. *Foot lameness in horses*. Palmerston North, New Zealand: Veterinary Continuing Education, 1990;65–71.
9. Garner HE, Coffman JR, Hahn AW, et al. Equine laminitis of alimentary origin: an experimental model. *Am J Vet Res* 1975;36:441–444.

10. Brumbaugh GW Hood DM, Wagner IP. Effectiveness of BAY TG 1000 in prevention of equine laminitis, in *Proceedings.* 1st Int Equine Conf Laminitis Dis Foot 2001;71.

11. Hood DM, Hunter JF, Beltz WD, et al. Digital loading patterns in the normal standing horse. In: Hood DM, Wagner IP, Jacobson AC, eds. *Proceedings of the hoof project*. College Station, Tex: Hood DM, Wagner IP, Jacobson AC, 1997;36–43.

12. Snow D, Volin C. Anatomy for performance. In: Snow DH, Vogel CJ, eds. *Equine fitness: the care and training of the athletic horse*. London: David & Charles, 1987;23–45.

13. Harris SE. Balance and movement. In: Harris SE, ed. *Horse gaits, balance and movement*. New York: Maxwell Macmillan International, 1993;74–81.

14. Davies AS. Locomotion. In: Davies AS, ed. *Quadrupedal mechanics: anatomical principles of the musculoskeletal system*. Palmerston North, New Zealand: Massey University, 1981;40–47.

15. Baker SJ, Steinheimer D, Burt NW, et al. The effect of weight bearing on equine digital blood flow. In: Hood DM, Wagner IP, Jacobson AC, eds. *Proceedings of The Hoof Project*. College Station, Tex: Hood DM, Wagner IP, Jacobson AC, 1997;54–59.

16. Hood DM, Grosenbaugh DA, Mostafa MB, et al. The role of vascular mechanisms in the development of acute equine laminitis. *J Vet Intern Med* 1993;7:228–234.

17. Hood DM. The pathophysiology of developmental and acute laminitis. *Vet Clin North Am Equine Pract* 1999;15:321–343.

18. Hood DM, Wagner IP, Brumbaugh GW. 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. *Am J Vet Res* 2001;62:1167-1172.

19. Adair HS, Goble DO, Schmidhammer JL, et al. Laminar microvascular flow, measured by means of laser Doppler flowmetry, during the prodromal stages of black walnut-induced laminitis in horses. *Am J Vet Res* 2000;61:862–868.

20. Burt NW, Baker SJ, Wagner IP, et al. Digital instability as a potential prognostic indicator in horses with chronic laminitis. In: Hood DM, Wagner IP, Jacobson AC, eds. *Proceedings of The Hoof Project*. College Station, Tex: Hood DM, Wagner IP, Jacobson AC, 1997;105–115.