

Changes in kinematic variables observed during pressure-induced forelimb lameness in adult horses trotting on a treadmill

Kevin G. Keegan, DVM, MS; David A. Wilson, DVM, MS; Bryan K. Smith, MS; Daniel J. Wilson, PhD

Objective—To determine whether kinematic changes induced by heel pressure in horses differ from those induced by toe pressure.

Animals—10 adult Quarter Horses.

Procedure—A shoe that applied pressure on the cuneus unguulae (frog) or on the toe was used. Kinematic analyses were performed before and after 2 levels of frog pressure and after 1 level of toe pressure. Values for stride displacement and time and joint angles were determined from horses trotting on a treadmill.

Results—The first level of frog pressure caused decreases in metacarpophalangeal (fetlock) joint extension during stance and increases in head vertical movement and asymmetry. The second level of frog pressure caused these changes but also caused decreases in stride duration and carpal joint extension during stance as well as increases in relative stance duration. Toe pressure caused changes in these same variables but also caused maximum extension of the fetlock joint to occur before midstance, maximum hoof height to be closer to midswing, and forelimb protraction to increase.

Conclusion and Clinical Relevance—Decreased fetlock joint extension during stance and increased head vertical movement and asymmetry are sensitive indicators of forelimb lameness. Decreased stride duration, increased relative stance duration, and decreased carpal joint extension during stance are general but insensitive indicators of forelimb lameness. Increased forelimb protraction, hoof flight pattern with maximum hoof height near midswing, and maximum fetlock joint extension in cranial stance may be specific indicators of lameness in the toe region. Observation of forelimb movement may enable clinicians to differentiate lameness of the heel from lameness of the toe. (*Am J Vet Res* 2000;61:612-619)

of treatment strategies and to more consistently teach recognition of lameness in horses to veterinarians in training.

Lameness of the foot of a forelimb affects the kinematic pattern of forelimb movement in horses.^{9,11-13} Stance phase duration increases in horses with lameness of the foot induced by application of pressure to the sole.¹³ This finding has been supported by other studies of experimentally induced lameness in horses.^{9,14} Other equine kinematic studies, particularly those involving lameness of the more proximal portion of the limb, have failed to confirm this correlation or have revealed the opposite findings (ie, a decreased stance phase duration with lameness).¹⁵⁻¹⁷ It is also a commonly accepted maxim that horses with foot lameness, and especially horses with navicular disease, have a shorter cranial phase to the stride (ie, shorter forelimb protraction) when compared with clinically normal horses that are not lame.^{18,19} However, the association between length of the cranial phase of the stride and foot lameness of a forelimb has not been supported by results of studies. A model of sole-pressure-induced foot lameness caused slightly less caudal retraction of the forelimb, but a change in protraction was not reported.¹¹ In a study conducted by our laboratory group,⁹ horses with navicular disease had decreased stance phase duration after a palmar digital nerve block; however, there was not a significant change in forelimb protraction, retraction, stride duration, or stride length. Inconsistencies between the results of these and other studies may be attributable to differences in the lameness models being evaluated. Lameness induced by application of pressure to the bottom of the foot may not be kinematically equivalent to that in horses with navicular disease. Horses with navicular disease may have a substantial secondary toe component of lameness, most notably attributable to pedal osteitis and sole bruising, and may move in a manner that differs from horses with isolated heel pain.

The objective of the study reported here was to determine whether kinematic changes induced by excessive pressure on the cuneus unguulae (frog) differ from those induced by pressure to the toe region of the hoof. Another objective was to determine whether kinematic changes caused by heel pain induced by excessive pressure on the frog are similar to kinematic

Within the past few years, computerized kinematic gait analysis has been used to study movement of clinically normal and lame horses.¹ Several investigators have described various kinematic variables that are characteristic of a normal gait in horses,²⁻⁵ gait of horses with superior movement qualities,^{6,7} and gait of horses with specific lameness abnormalities.^{3,6-11} These investigative efforts are worthwhile, because they improve attempts to objectively measure lameness. Objective evaluation of lameness, in turn, will assist veterinarians to more reasonably validate effectiveness

Received May 12, 1999.

Accepted Aug 30, 1999.

From the Department of Veterinary Medicine and Surgery, College of Veterinary Medicine (Keegan, DA Wilson); the Department of Food Science and Human Nutrition, College of Human Environmental Sciences (Smith); and the Department of Physical Medicine and Rehabilitation, Rusk Rehabilitation Center (DJ Wilson), University of Missouri, Columbia, MO 65211.

Supported by the University of Missouri, College of Veterinary Medicine Committee on Research; the USDA, Section 1433, Animal Health Formula Fund; and private donations.

The authors thank Dr. Donald Schmidt, Don Conner, and Brian Reasoner for technical assistance.

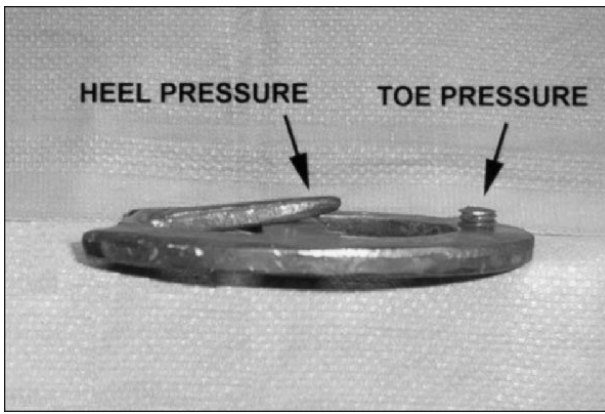


Figure 1—Custom-made bar shoe designed for applying pressure to the cuneus unguulae (frog) through adjustment of a screw (4.8 mm in diameter) that pressed an adjustable heart-shaped bar into the frog (heel pressure) or to the toe by adjusting a screw (11.1 mm in diameter) that pressed directly into the sole at the toe of the foot (toe pressure).

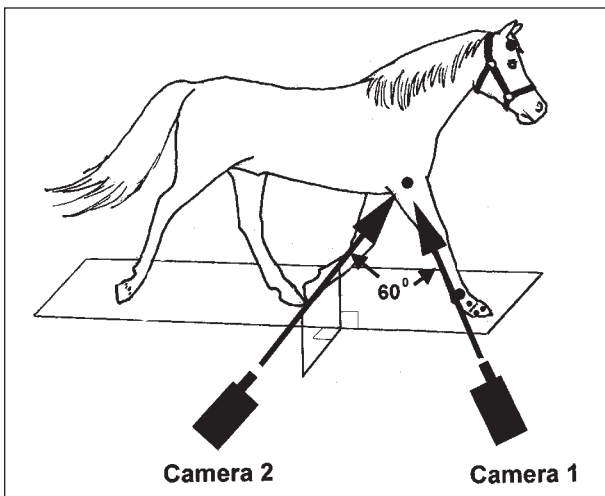


Figure 2—Diagram depicting location of cameras for filming horses on a treadmill. The 2-dimensional position of reflective markers from 2 camera views (frontolateral and lateral) were used to determine 3-dimensional position of markers in each video frame.

changes that have been documented for horses with navicular disease.

Materials and Methods

Horses—Ten adult Quarter Horses (3 geldings, 7 mares) ranging from 5 to 16 years old were used in the study. All horses were believed to be clinically normal on the basis of results of complete lameness examinations performed before and after hoof trimming performed by an experienced farrier. All horses were trained to trot on a treadmill^a at a speed (range, 2.6 to 3.6 m/s) at which they could maintain exercise for 5 minutes while maintaining stable position on the treadmill without excessive coaxing by the handlers. Several horses were accustomed to exercise on the treadmill from use in previous studies. Horses unfamiliar with treadmill exercise were trained during ≥ 3 sessions conducted during 3 separate 24-hour periods.

Induction of lameness—Each horse was fitted with a custom-made, wide-webbed, adjustable heart-bar shoe applied to the foot of the right forelimb. The shoe could apply

reversible pressure to the frog or to the sole at the toe (Fig 1). Pressure on the frog could be induced by tightening a screw (4.8 mm in diameter) through a threaded hole in the bar, which in turn pressed an adjustable steel heart-bar into the frog. Pressure on the toe could be induced by adjusting a screw (11.1 mm in diameter) that pressed directly into the sole at the toe of the foot. A similar bar shoe, but without screws, was applied to the foot of the left forelimb to maintain weight balance between the forelimbs. All procedures were approved by a university animal care and use committee.

Each horse was initially filmed before inducing lameness (condition = sound) while trotting on the treadmill at the speed determined for that horse. The screw in the bar then was tightened to induce pressure on the frog until mild lameness was evident in the right forelimb when the horse trotted (condition = heel 1). Another level of heel lameness was evaluated by further tightening the screw 4 or 5 additional rotations, causing the heart-bar to press deeper into the frog. If the increased lameness made it difficult for the horse to maintain position on the treadmill, the screw was loosened 2 or 3 rotations, and the horse was reevaluated. Each horse was filmed again when lameness was greater than for the first condition but still sufficiently mild to permit the horse to maintain position on the treadmill at the previous speed (condition = heel 2).

After evaluation of each of the levels of pressure-induced lameness of the frog, the screw was loosened so that pressure was not applied to the frog, and the horse was allowed to trot on the treadmill until lameness was not evident. Then, a third lameness evaluation was conducted after the toe screw was tightened to induce lameness. The toe screw was tightened until it contacted the sole at the toe. This evaluation was performed when the resulting lameness of the right forelimb was clearly visible, but the horse could still maintain position on the treadmill when trotting at the previous treadmill speed (condition = toe). Pressure-induced lameness of the frog always was evaluated before pressure-induced lameness of the toe, because pressure-induced lameness of the frog always was rapidly reversible, whereas pressure-induced lameness of the toe frequently resulted in a degree of residual lameness that persisted for minutes to hours after cessation of pressure.

Computer-assisted kinematic gait analysis—Computer-assisted kinematic gait analysis was performed for each condition, using a procedure similar to that described in other studies.⁹ Briefly, 2 cameras^b were used to record the horses on the treadmill (60 frames/s, shutter speed of 1/1,000 s). Cameras were situated on the right side of the treadmill with one directed at a 30° and the other at a 90° angle to the longitudinal axis of the treadmill (Fig 2). These positions allowed frontolateral and lateral views (Fig 3) of the horses trotting on the treadmill. Cameras were positioned approximately 4 m from the treadmill. Three-dimensional position of reflective markers (0.75-cm diameter markers for the foot and proximal interphalangeal [pastern]; 1.25-cm diameter markers for the metacarpophalangeal [fetlock] joint, carpus, decubital [elbow] joint, and poll) attached to the right forelimb and head of each horse were tracked by use of a computer and performance analysis software^c connected to a videotape recorder.^d A minimum of 900 frames (15 seconds) of continuous videotape were analyzed for each condition, which translated to 15 to 20 full strides of continuous motion for each horse for each condition. Values for 19 variables used for assessment of stride displacement, stride time, and joint angle (carpus and fetlock) were determined from the 3-dimensional positional data (Appendix). Data were smoothed by use of a digital signal filter (cutoff frequency, 10 Hz). Joint angle measurements were obtained from the palmar aspect of the forelimb and calculated as the angles formed between adjacent markers in the limb's sagittal plane. Mean values for the kinematic variables were calculated for



Figure 3—Field of view of camera 1 (frontolateral view; left) and camera 2 (lateral view; right) from Figure 2. The laboratory was darkened to increase contrast between markers, horse, and environment. Reflective markers were illuminated by halogen lamps positioned behind each camera.

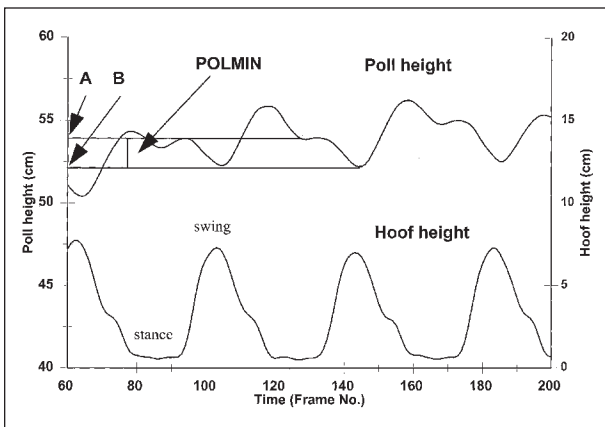


Figure 4—Method for determining severity of forelimb lameness. Height of poll at first minimum during stance phase of the lame forelimb (A) and height of poll at second minimum during swing phase of the lame forelimb (stance phase of the unaffected forelimb; B) were determined. Difference in minimum poll height during right and left forelimb stance phases (POLMIN) was used as a measure of lameness degree for each horse at each condition. A value of POLMIN = 0 indicates a horse that is not lame. A positive value for POLMIN indicates lameness of the right forelimb, and a negative value for POLMIN indicates lameness of the left forelimb.

all horses and all conditions. The **difference in minimum poll height (POLMIN)** during right and left forelimb stance phases was used as a measure of lameness degree for each horse for each condition (Fig 4). A POLMIN value significantly different from 0 indicated lameness: a positive value indicated lameness of the right forelimb, and a negative value indicated lameness of the left forelimb.

To estimate accuracy of the method, a separate experiment was conducted, using identical conditions of camera positioning and lighting. A free-standing inverted T-shaped pendulum (94 × 57 cm) was constructed. Reflective markers were attached to the pendulum at specific positions. From the distance between markers and the law of cosines, several angles (ranging from 30 to 360°) could be defined. The pendulum was filmed, using identical conditions, after it was set in motion to freely traverse 360° arcs of motion. Angles and distances were calculated for 300 frames during which time the pendulum traversed 2 complete 360° arcs. Experimentally determined angles and distances were compared

with values for known angles and distances, and system accuracy was reported as the **root mean-squared (RMS) error**.²⁰ Experimentally determined angles and distances were compared with mean angles and distances, and system precision was reported as RMS error.²⁰

Data analyses—Variables were evaluated for all 4 conditions by use of a general linear model, repeated-measures ANOVA. Means of variables for each horse or condition were weighted according to the number of strides analyzed. Data from 1 horse obtained during condition heel 1 could not be used because of technical difficulties associated with the filming procedure. Post hoc comparisons among conditions were evaluated by use of the Fisher Least Squared Difference test. Values of $P \leq 0.05$ were considered significant.

Results

Accuracy and precision of the method—Accuracy of the experimental method (RMS systematic + RMS random error) was 0.32 cm for the determination of distance measurements and 0.36° for the determination of angle measurements. Precision of the experimental method (RMS random error) was 0.14 cm for distance measurements and 0.35° for angle measurements. Therefore, there was a bias of 0.18 cm in distance measurements and no bias for angle measurements in the experimental system.

Severity of lameness—Mean difference in POLMIN before induction of lameness (condition = sound) was 0.5 cm (range, -6.0 to 3.6 cm), which was not significantly different from 0. Mean POLMIN for heel 1, toe, and heel 2 were 5.6 cm (range, 3.1 to 12.6 cm), 7.7 cm (range, -1.4 to 13.7 cm), and 15.1 cm (range, 6.7 to 26.9 cm), respectively. These values correspond to severity of lameness of the right forelimb (most severe to least severe) of heel 2 > toe > heel 1 > sound. For a particular horse, order of severity of lameness (by condition) may have differed, but for each horse, POLMIN was lowest for sound and highest for heel 2, compared with heel 1 and toe. Mean POLMIN was not significantly different between heel 1 and toe.

Effect of pressure-induced lameness of the frog—The first level of pressure-induced lameness resulted in

Table 1—Mean values of variables that changed significantly when lameness was induced in the right forelimb of 10 horses

Variable	Condition*			
	Sound	Heel 1	Toe	Heel 2
FETMAX (degrees)	245.0 ^{b,c,d}	239.5 ^{a,d}	240.6 ^{a,d}	232.8 ^{a,b,c}
FETIME (ms)	2.7 ^c	-0.1 ^c	8.5 ^{a,b,d}	1.7 ^c
CARMAX (degrees)	188.1 ^d	187.6 ^d	187.8 ^d	186.7 ^{a,b,c}
HOOFTIME (ms)	57.6 ^c	56.7 ^c	45.9 ^{a,b,d}	58.5 ^c
STRDUR (ms)	732.3 ^{c,d}	721.7 ^d	716.9 ^b	704.6 ^{a,b}
PCTSTA (%)	37.5 ^d	37.9 ^d	38.1	39.6 ^{a,b}
PROT (cm)	48.8 ^c	49.1 ^c	51.5 ^{a,b,d}	48.1 ^c
POLMIN (cm)	0.5 ^{b,c,d}	5.6 ^{a,d}	7.7 ^{a,d}	15.1 ^{a,b,c}
POLEXCUR (cm)	5.7 ^{b,c,d}	9.4 ^{a,d}	10.5 ^{b,d}	16.5 ^{a,b,c}

*Sound = Clinically normal horse before lameness was induced. Heel 1 = First level of lameness induced by pressure on the cuneus unguulae (frog). Toe = Lameness induced by pressure on the toe. Heel 2 = Second level of lameness induced by pressure on the frog. FETMAX = Maximum metacarpophalangeal (fetlock) joint extension during stance. FETIME = Time of maximum fetlock joint extension. CARMAX = Maximum carpal joint extension during stance. HOOFTIME = Time of maximum hoof height. STRDUR = Stride duration. PCTSTA = Percentage of stance duration. PROT = Maximum limb protraction. POLMIN = Difference in minimum poll height during right and left forelimb stance phases. POLEXCUR = Vertical poll excursion.

^aValue is significantly ($P \leq 0.05$) different from value for sound. ^bValue is significantly ($P \leq 0.05$) different from value for heel 1. ^cValue is significantly ($P \leq 0.05$) different from value for toe. ^dValue is significantly ($P \leq 0.05$) different from value for heel 2.

significant changes in 3 of 19 variables (Table 1). **Maximum fetlock extension during stance (FETMAX)** decreased significantly ($P = 0.007$) from a mean of 245.0° to 239.5°. Values for FETMAX during stance decreased in 8 of 9 horses for which data were obtained. In 1 horse, FETMAX increased by 1.3°. Mean POLMIN and mean total vertical poll excursion during 1 complete stride (POLEXCUR) were significantly increased after pressure-induced lameness of the frog. Mean POLMIN increased significantly ($P = 0.04$) from 0.5 cm to 5.6 cm. The POLMIN increased for all 9 horses for which data were obtained. Mean POLEXCUR increased significantly ($P = 0.03$) from 5.7 cm to 9.4 cm, and POLEXCUR increased in all 9 horses for which data were obtained. Significant differences were not detected in carpal angles, in stride-temporal (stride or stance duration) or stride-displacement (limb protraction or retraction) variables, or in variables describing hoof flight during the swing phase of the stride (maximum hoof height or timing of maximum hoof height).

The second, more severe, level of pressure-induced lameness of the frog resulted in significant changes in the same 3 variables (FETMAX, POLMIN, POLEXCUR) in the same directions as evident for the first level of pressure-induced lameness. However, the second level of pressure-induced lameness of the frog also resulted in significant changes in 3 additional variables (Table 1). Mean maximum carpal extension during stance (CARMAX) decreased significantly ($P < 0.001$) from 188.1° to 186.7°, mean stride duration (STRDUR) decreased significantly ($P < 0.001$) from 732.3 to 704.6 milliseconds, and mean percentage stance duration (PCTSTA) increased significantly ($P = 0.01$) from 37.5 to 39.6%. Values for CARMAX and STRDUR decreased for all 10 horses in the study, whereas PCTSTA increased in 7 horses, did not change in 2 horses, and slightly decreased in 1 horse (from 39.1 to 37.7%).

Significant differences were not detected in stride-displacement variables or variables describing hoof flight during the swing phase of the stride.

When the first and second levels of pressure-induced lameness of the frog were compared, significant differences were detected for 2 joint-angle variables (FETMAX and CARMAX), 2 stride-temporal variables (STRDUR and PCTSTA), and both poll-movement variables (POLMIN and POLEXCUR). Means for FETMAX and CARMAX were significantly less (6.7°; $P = 0.001$ and 0.9°; $P = 0.007$, respectively) for the second level of pressure-induced lameness. Mean STRDUR was significantly ($P = 0.03$) less (difference of 17.1 milliseconds), but PCTSTA was significantly ($P = 0.04$) greater (1.7%), for the second level of pressure-induced lameness. Means for POLMIN and POLEXCUR were significantly ($P < 0.001$) greater (9.5 and 7.1 cm, respectively) after the second level of pressure-induced lameness. Significant differences were not detected in stride-displacement variables or variables describing hoof flight during the swing phase of the stride.

Effect of pressure-induced lameness of the toe—Pressure-induced lameness of the toe resulted in significant changes in 7 of 19 variables. Mean FETMAX decreased significantly ($P = 0.02$) from 245.0° to 240.6°. Values for FETMAX decreased in 9 of 10 horses. In 1 horse, FETMAX increased by 2.3°. Both variables describing poll vertical movement (POLMIN and POLEXCUR) were significantly increased after pressure-induced lameness of the toe. Mean POLMIN increased significantly ($P = 0.002$) from 0.5 cm to 7.7 cm. Mean POLEXCUR increased significantly ($P = 0.006$) from 5.7 cm to 10.5 cm. Values for POLMIN and POLEXCUR increased in all 10 horses. Mean STRDUR decreased significantly ($P = 0.04$) from 732.3 to 716.9 milliseconds; however, mean PCTSTA did not change significantly after pressure-induced lameness. Values for STRDUR decreased in 9 of 10 horses. In 1 horse, STRDUR did not change after pressure-induced lameness of the toe.

Three other variables were changed significantly after pressure-induced lameness of the toe, none of which were changed significantly after either level of pressure-induced lameness of the frog. Before induction of lameness, FETMAX was 2.7 seconds before midstance, but after pressure-induced lameness of the toe, FETMAX was significantly ($P = 0.01$) earlier during stance (8.5 seconds before midstance). After pressure-induced lameness of the toe, FETMAX was earlier during stance in 7 of 10 horses, at the same time during stance in 2 horses, and later during stance in 1 horse. **Maximum hoof height during the swing phase of the stride (HOOFTIME)** was significantly ($P = 0.004$) closer to midswing after pressure-induced lameness of the toe (mean time of maximum hoof height [HOOFTIME], 45.9 milliseconds) than before induction of lameness (HOOFTIME, 57.6 milliseconds). After pressure-induced lameness of the toe, HOOFTIME was closer to midswing in 9 of 10 horses. In 1 horse, HOOFTIME was farther from midswing (during the caudal phase of swing) after pressure-induced lame-

ness of the toe. Mean length of **maximum forelimb protraction (PROT)** after pressure-induced lameness of the toe (51.5 cm) was significantly ($P = 0.001$) greater than that before induction of lameness (48.8 cm). Value for PROT increased in 9 of 10 horses. In 1 horse, PROT decreased slightly (by 1.0 cm) after pressure-induced lameness of the toe.

Three kinematic variables differed significantly between lameness induced by the first level of frog pressure and lameness induced by toe pressure. After pressure-induced lameness of the toe, FETMAX was significantly ($P = 0.002$) sooner (8.5 milliseconds before midstance), whereas it was at midstance after the first level of pressure-induced lameness of the frog. Meanwhile, HOOFY was significantly ($P = 0.01$) closer to midswing after pressure-induced lameness of the toe (mean HOOFTIME, 45.9 milliseconds) than after the first level of pressure-induced lameness of the frog (mean HOOFTIME, 56.7 milliseconds). Mean PROT for pressure-induced lameness of the toe (51.5 cm) was significantly ($P = 0.005$) greater than that for the first level of pressure-induced lameness of the frog (49.1 cm). Similar differences in these 3 variables also were evident between pressure-induced lameness of the toe and the second level of pressure-induced lameness of the frog.

Discussion

Horses selected for use in the study reported here were found to be without lameness during an initial lameness examination. Although mean POLMIN for the horses before induction of lameness was 0.5 cm and indicative of typical symmetric vertical head movement in clinically normal horses, values ranged from -6.0 to 3.6 cm. These initial poll-movement asymmetries detected at the beginning of the study in a few horses may have indicated a deficiency of the original examination to detect lameness or an increased sensitivity of computerized kinematic analysis. Another explanation is normal variability (noise) or asymmetry (offset) of poll vertical movement in clinically normal horses. In a previous study in our laboratory,²¹ it was suspected that treadmill locomotion had a sparing effect on the severity of perceived forelimb lameness in horses with navicular disease. Therefore, we considered that an increased sensitivity for computerized kinematic analysis in this study was unlikely. Asymmetric vertical poll movement has been reported in other studies^{9,10,15,22} to be a good indicator of forelimb lameness in horses. Because we measured changes in kinematic variables using each horse as its own control, the initial poll vertical movement asymmetries deserved mention but were not considered detrimental to the purposes of the study.

The amount of screw tightening required to produce lameness was determined subjectively by visual evaluation of each horse trotting on the treadmill after initial screw adjustment. The goal was to create a mild but easily determinable amount of lameness. The resulting lameness varied among the experimental subjects. In some cases, during the first level of pressure-induced lameness of the frog, the lameness decreased while the horses were trotting on the treadmill. Care

was taken to film each horse before the induced lameness disappeared completely, but in a few instances, the lameness became extremely mild and was difficult to observe before filming could be completed. Nevertheless, the amount of lameness, as measured by POLMIN, was always greater after adjusting the screws than for the sound condition. This indicated that we were successful in producing temporary lameness in the right forelimb in each horse after applying pressure to the frog or toe.

After the first level of pressure-induced lameness of the frog, FETMAX decreased, POLEXCUR increased, and vertical poll movement became less symmetric (POLMIN increased). These same changes have been reported in other kinematic studies of forelimb lameness in horses and, in our opinion, can be considered good general indicators of forelimb lameness when comparing the same subject before and after treatment.^{9,11-13} During forelimb lameness, horses actively resist the downward momentum of the head that occurs before stance.²² Reduced downward momentum of the head decreases the force of weight that bears on a painful limb. Asymmetric vertical poll movement is recognized empirically by equine practitioners as a head nod and is almost universally equated with forelimb lameness. In a previous study,²¹ 6 of 6 clinicians agreed that kinematically measured asymmetry of vertical poll movement correlated best with subjective evaluation of lameness, compared with all other kinematic variables studied.

Mean decrease in FETMAX after induction of the first level of pressure-induced lameness of the frog was 5.5°. That small change in fetlock angle at full extension was significantly different because of the high repeatability between strides for each horse. Whether such a small change would be detectable by an observer is unknown. Using kinematic gait analysis, Holmström et al⁷ reported that FETMAX was highly correlated with subjective score of gait quality in young trotting horses during exercise, and they concluded that an expert at equine gait evaluation would be able to detect this. However, in a previous study,²¹ our laboratory group found that kinematically measured FETMAX during stance was among the top 3 kinematic variables correlated with subjective evaluation of lameness for only 2 of 6 equine clinicians.

The significant decrease in FETMAX and increases in POLMIN and POLEXCUR indicated that the degree of lameness after the first level of pressure-induced lameness of the frog was sufficiently different than before induction of lameness, even though the induced lameness was mild and difficult to determine by subjective visual evaluation in some horses. Changes in FETMAX during stance and POLMIN and POLEXCUR for the second level of pressure-induced lameness of the frog were more dramatic than after the first level of pressure-induced lameness. Larger decreases in FETMAX and larger increases in POLMIN and POLEXCUR for the second level of pressure-induced lameness of the frog indicated a substantial increase in severity of lameness, which was easy to appreciate subjectively.

The second level of pressure-induced lameness of the frog resulted in significant changes in 3 other vari-

ables that were not changed significantly after the first level of pressure-induced lameness. Mean CARMAX decreased by 1.4°. This decrease was small and unlikely to be detected by observation, but it was significantly different because of the small variability within each horse. In a previous study²³ that used a transient radio-carpal joint arthritis model, the carpus was less extended during stance after lameness but only when the induced lameness was characterized as severe and visible at a walk.²³ After the second level of pressure-induced lameness of the frog, mean STRDUR decreased by 27.7 milliseconds; however, at the same time, mean PCTSTA increased from 37.5 to 39.6%. Therefore, swing phase duration, which was not directly measured, decreased. Because the horses were constrained to trot on the treadmill at the same speed for all conditions, decreased stride duration after induction of lameness also represented decreased stride length. Decreased stride length classically has been equated with forelimb lameness in horses, especially lameness secondary to navicular disease.^{18,19} However, in a previous study⁹ of horses with navicular disease and lameness approximately equivalent to our first level of pressure-induced lameness of the frog (as determined on the basis of POLMIN values), STRDUR did not increase significantly after palmar digital nerve block. In the study reported here, STRDUR did not decrease significantly until after the second level of pressure-induced lameness of the frog and not until poll vertical movement asymmetries were much higher (15.1 cm for heel 2 vs 5.6 cm for heel 1). Therefore, although carpal extension during stance and STRDUR changed with varying conditions of heel lameness, changes in these 2 variables are not as sensitive as changes in fetlock extension during stance or changes in the symmetry of vertical head movement.

Contrary to popular belief, an increase in PCTSTA with lameness is consistent with results reported in other studies of induced lameness in horses.^{11,13} It has been proposed that for horses with mild to moderate lameness of the foot, compensating head and neck movements and intralimb biomechanical adjustments redistribute the total force of weight bearing over a longer duration by increasing stance time.¹³ When the speed of movement is constrained by trotting on a treadmill, an increase in stance phase duration has been documented in horses with navicular disease and in horses with lameness induced by creating pressure on the solar surface between the quarters and bars.^{9,11} An increased stance phase duration also has been documented in a single horse with chronic sesamoiditis.²⁴ Increased stance duration was not documented in horses with lameness of the carpus of similar severity,^{15,23} but a decrease in stance phase duration has been documented in a single horse with a fracture of the third carpal bone.¹⁶ Stance phase duration is highly dependent on speed of the horse, and, if not controlled, speed of a horse may voluntarily decrease with lameness.²⁵ We believe that results of the study reported here, in conjunction with results from other studies, indicates that, using conditions of constrained constant speed, an increased stance phase duration in horses may be an indicator of foot lameness of moderate degree. However, at some point along a scale of lameness severity, a horse must bear weight for a shorter

period on the affected limb (ie, stance phase duration will decrease), because horses with severe lameness may be completely unable to bear weight on an affected limb. In light of this common observation, results of the study reported here also suggest that with increasing severity of lameness, stance duration initially increases but then decreases as a threshold of lameness severity is reached, and this threshold probably is reached at a moderate to severe degree of lameness.

After pressure-induced lameness of the toe, the amount of decrease in FETMAX and increases in POLMIN and POLEXCUR indicated that the lameness induced was slightly greater than that induced by the first level, but less than that induced by the second level, of pressure-induced lameness of the frog. The amount of pressure-induced lameness of the toe was sufficient to detect a shorter stride duration (and therefore a shorter stride length), but an increase in any measure of stance phase duration was not detected. Whether this was a result of a decreased sensitivity of a change in stance phase duration to represent foot lameness or to a biomechanical idiosyncrasy of toe lameness in the horses was unknown. However, there were significant changes in 3 kinematic variables that were detected only after pressure-induced lameness of the toe. Pressure-induced lameness of the toe caused FETMAX to be earlier (before midstance), HOOFY to be later (closer to midswing), and PROT to increase, compared with values for before lameness induction and after both levels of pressure-induced lameness of the frog.

In a clinically normal horse at a trot, FETMAX should be at midstance.^{2,4,5} Except for impact at the beginning of stance, vertical ground reaction forces are greatest on a limb at midstance.^{26,27} In the study reported here, FETMAX was before midstance after pressure-induced lameness of the toe but not after either level of pressure-induced lameness of the frog, despite the significantly greater severity of lameness in the second level of pressure-induced lameness of the frog. At hoof impact, the caudal part of the hoof is loaded to a greater extent than the toe; however, soon afterward, the center of ground reaction force shifts forward toward the toe.²⁸⁻³⁰ It is possible that the biomechanical alterations used by horses to reduce this dorsal shifting of vertical ground reaction force develop only after the beginning of stance, causing FETMAX during cranial stance. In contrast, for lameness associated with the heel, a substantial reduction in force on the heel during impact would require biomechanical adjustments before the beginning of stance that would likely affect the entire stance phase. Mean FETMAX would be diminished, but peak FETMAX would still be at midstance. Previously, we found that FETMAX was before midstance in horses with navicular disease, but after palmar digital nerve block, FETMAX was at midstance.⁹ Horses in that study may have had a corresponding secondary toe lameness that was eliminated by the palmar digital nerve block. Pedal osteitis and toe bruising are expected secondary conditions of navicular disease, and painful loci of these conditions may be partially eliminated with a palmar digital nerve block.³¹ Analysis of results of the study reported here suggests that inducing heel lameness with pressure on the frog does not exactly mimic lameness caused by

navicular disease and, therefore, is not a good model to use in the study of kinematics of navicular disease.

After pressure-induced lameness of the toe, but not after pressure-induced lameness of the frog, increases in PROT were detected. Greater PROT was detected after pressure-induced lameness of the toe in 9 of 10 horses in the study, compared with values for the sound condition, and in all 10 horses, compared with values for the first or second level of pressure-induced lameness of the frog. Buchner et al¹¹ reported that the forelimb retraction angle was decreased in the lame and contralateral unaffected forelimbs after an induced foot lameness without a change in forelimb protraction angle; however, in that study, the induced lameness was caused by pressure to the solar surface at the quarter instead of the toe.¹¹ In a similar study of induced foot pain in the forelimb,¹³ investigators reported that protraction and retraction angles of the forelimb were both slightly increased but not to a significant degree. Authors of that study also suggested that this could have been attributable to the slightly prolonged stance phase duration they detected after induction of lameness. Increased forelimb protraction with toe lameness may be a compensatory mechanism by which a horse causes hyperextension of the distal interphalangeal joint immediately before hoof impact so that hoof impact is heel-first instead of flat-footed with concurrent toe and heel concussion. Our sample rate of 60 frames/s does not allow an unequivocal determination of heel-first or flat-footed landing, even in trotting horses, so we could not reliably test this explanation. In a previous study by our laboratory group,⁹ horses with navicular disease did not have changes in forelimb retraction or protraction after palmar digital nerve block. That association is curious when viewed in light of the common perception that horses with navicular disease have a shortened cranial phase to the stride,^{18,19} which ostensibly increases and returns to normal after palmar digital nerve block. Whether this commonly reported phenomenon is an illusion attributable to altered hoof flight during the swing phase of the stride or to a voluntary increase in speed of the horse because it simply perceives less pain after the nerve block is unknown.

Pressure-induced lameness of the toe changed the shape of the hoof flight during the swing phase of the stride. The absolute value of HOOFY was not significantly changed, but it was detected closer to midswing. It has been suggested that the swing phase of the forelimb is entirely passive, resulting from elastic energy released from the flexor tendons of the forelimb at the end of stance phase.⁷ This view is supported by the finding that electromyographic signals in flexor muscles of the forelimbs are small during the swing phase of the stride.³² Maximum hoof height typically may be detected before midswing as the result of the passive recoil propelling the limb up instead of forward. Muscular contraction of the major flexors of the shoulder and elbow joint during breakover, when the ground reaction force exerted on the toe rapidly increases, may lift the limb and serve to reduce the force of breakover and diminish the elastic energy released from the flexor tendons. A hoof flight pattern with HOOFY closer to midswing could also be a mechanical prerequisite to affect the increased forelimb protraction detected in the

horses in this study. Interestingly, a hoof flight pattern with HOOFY near midswing correlated with subjective evaluation of improvement in mild to moderate lameness by most clinicians, despite the inability of computerized kinematic analysis to substantiate an association between hoof flight pattern and improvement in lameness.²¹ Further elucidation of the association between time of **maximum fetlock extension (FETIME)**, **HOOFTIME**, and PROT in horses with toe lameness needs to be accomplished. We are not aware of other studies that address these kinematic variables and the manner in which they change with lameness isolated to the toe region of the foot.

Evaluation of results of the study reported here should be considered in light of a few additional comments. Because of the involved process of data collection and evaluation, we used only 10 horses but evaluated 19 variables for each of 4 conditions. Methods to control experiment-wise error for multiple variables were not stringent. We were interested in identifying potential kinematic variables for further study. Also, because of the nature of the model used, we did not randomize treatment order. We cannot unequivocally claim that changes seen were not attributable to treatment order; however, we do not believe that changes in FETIME, HOOFTIME, and PROT after pressure-induced lameness of the toe were adequately explained by residual pressure-induced lameness of the frog, because we allowed the horses to recover and become clinically normal before inducing toe lameness. In addition, none of the variables in question were significantly changed with either level of pressure-induced lameness of the frog, which were not applied in random order. After pressure-induced lameness of the toe, FETIME got larger, and HOOFTIME got smaller. Experiment-wise error in calibration caused by nonrandom ordering of treatments would result in changes in these variables in the same direction (ie, they would both get smaller or both get larger). Lastly, increases in PROT were not accompanied by similar changes in any other limb- or hoof-displacement variables, providing evidence that there was not experiment-wise error in length measurements as a result of the order of treatments.

^aSato I, Equine Dynamics, Raymore, Mo.

^bPanasonic AG455P, Panasonic Corp, Secaucus, NJ.

^cAriel Performance Analysis System, Ariel Dynamics Inc, Trabuco Canyon, Calif.

^dPanasonic AG-7350, Panasonic Corp, Secaucus, NJ.

References

1. Barrey E. Methods, applications and limitations of gait analysis in horses. *Vet J* 1999;157:7–22.
2. Clayton HM, Lanovaz JL, Schamhardt HC, et al. Net joint moments and powers in the equine forelimb during the stance phase of the trot. *Equine Vet J* 1998;30:384–389.
3. Pourcelot P, Audigie F, Degueurce C, et al. Kinematic symmetry index: a method for quantifying the horse locomotion symmetry using kinematic data. *Vet Res* 1997;28:525–538.
4. Johnston C, Roepstorff L, Drevemo S, et al. Kinematics of the distal hindlimb during stance phase in the fast trotting standardbred. *Equine Vet J* 1996;28:263–268.
5. Back W, Schamhardt HC, Savelberg HH, et al. How the horse moves: 1. Significance of graphical representations of equine forelimb kinematics. *Equine Vet J* 1995;27:31–38.

6. Morales JL, Manchado M, Vivo J, et al. Angular kinematic patterns of limbs in elite and riding horses at trot. *Equine Vet J* 1998;30:528–533.

7. Holmström M, Fredricson I, Drevemo S. Biokinematic differences between riding horses judged as good and poor at the trot. *Equine Vet J* 1994;1(suppl):51–56.

8. Keegan KG, Wilson DJ, Wilson DA, et al. Effects of balancing and shoeing of the forelimb feet on kinematic gait analysis in five horses with navicular disease. *J Equine Vet Sci* 1998;18:522–527.

9. Keegan KG, Wilson DJ, Wilson DA, et al. Effects of anesthesia of the palmar digital nerves on kinematic gait analysis in horses with and without navicular disease. *Am J Vet Res* 1997;58:218–223.

10. Vorstenbosch MATM, Buchner HHF, Savelberg HHCM, et al. Modeling study of compensatory head movements in lame horses. *Am J Vet Res* 1997;58:713–718.

11. Buchner HHF, Savelberg HHCM, Schamhardt HC, et al. Limb movement adaptations in horses with experimentally induced fore- or hindlimb lameness. *Equine Vet J* 1996;28:63–70.

12. Buchner HHF, Savelberg HHCM, Schamhardt HC, et al. Bilateral lameness in horses—a kinematic study. *Vet Q* 1995;17:103–105.

13. Galisteo AM, Cano MR, Morales JL, et al. Kinematics in horses at the trot before and after an induced forelimb supporting lameness. *Equine Vet J* 1997;23(suppl):97–101.

14. Huskamp B, Tietje S, Nowak M, et al. Fubungs—und bewegungsmuster gesunder und strahlbeinkranker pferde-gemessen mit dem Equine-Gait-Analysis-System (EGA-System). *Pferdeheilkunde* 1990;6:231–236.

15. Peloso JG, Stick JA, Soutas-Little RW, et al. Computer-assisted three-dimensional gait analysis of amphotericin-induced carpal lameness in horses. *Am J Vet Res* 1993;54:1535–1543.

16. Clayton CM. Cinematographic analysis of the gait of lame horses III: fracture of the third carpal bone. *J Equine Vet Sci* 1987;7:130–135.

17. Clayton CM. Cinematographic analysis of the gait of lame horses IV: degenerative joint disease of the distal intertarsal joint. *J Equine Vet Sci* 1987;7:274–278.

18. Turner TA. Diagnosis and treatment of the navicular syndrome in horses. *Vet Clin North Am Equine Pract* 1989;5:131–144.

19. Wright IM. A study of 118 cases of navicular disease: clinical features. *Equine Vet J* 1993;25:488–492.

20. Allard P, Blanche JP, Alssaoui. Bases of three-dimensional reconstruction. In: Allard P, Stokes IAF, Blanche JP, eds. *Three-dimensional*

analysis of human movement. Champaign, Ill: Human kinetics, 1995;19–40.

21. Keegan KG, Wilson DA, Wilson DJ, et al. Evaluation of mild lameness in horses trotting on a treadmill: agreement by clinicians and interns or residents and correlation of their assessments with kinematic gait analysis. *Am J Vet Res* 1998;59:1370–1377.

22. Buchner HHF, Savelberg HHCM, Schamhardt HC, et al. Head and trunk movement adaptations in horses with experimentally induced fore- or hindlimb lameness. *Equine Vet J* 1996;28:71–76.

23. Back W, Barneveld A, van Weeren PR, et al. Kinematic gait analysis in equine carpal lameness. *Acta Anat (Basel)* 1993;146:86–89.

24. Clayton CM. Cinematographic analysis of the gait of lame horses III: chronic sesamoiditis. *J Equine Vet Sci* 1986;6:310–320.

24. van Weeren PR, van den Bogert AJ, Back W, et al. Kinematics of Standardbred trotters measured at 6, 7, 8, and 9 m/s on a treadmill before and after 5 months of training. *Acta Anat (Basel)* 1993;146:154–161.

25. Riemersma DJ, Schamhardt HC, Hartman HC, et al. Kinetics and kinematics of the equine hind limb: in vivo tendon loads and force plate measurements in ponies. *Am J Vet Res* 1988;49:1344–1352.

26. Schryver HF, Bartel DL, Langrana N, et al. Locomotion in the horse: kinematics and external and internal forces in the normal equine digit in the walk and trot. *Am J Vet Res* 1978;39:1728–1733.

27. Savelberg HHLM, van Loon T, Schamhardt HC. Ground reaction forces in horses, assessed from hoof wall deformation using artificial neural networks. *Equine Vet J Suppl* 1997;23:6–8.

28. Ratzlaff MH, Hyde ML, Grant BD, et al. Measurement of vertical forces and temporal components of the strides of horses using instrumented shoes. *J Equine Vet Sci* 1990;10:23–35.

29. Barrey E. Investigation of the vertical hoof force distribution to the equine forelimb with an instrumented horseboot. *Equine Vet J* 1990;9(suppl):35–38.

30. Moyer W. Clinical examination of the equine foot. *Vet Clin North Am Equine Pract* 1989;5:29–46.

31. Jansen MO, van Raaij JAGM, van den Bogert AJ, et al. Quantitative analysis of computer-averaged electromyographic profiles of intrinsic limb muscles in ponies at the walk. *Am J Vet Res* 1992;53:2343–2349.

32. Schamhardt HC, Merckens HW. Objective determination of ground contact of equine limbs at the walk and trot: comparison between ground reaction forces, accelerometer data and kinematics. *Equine Vet J* 1994;17(suppl):75–79.

Appendix

Kinematic variables evaluated for 10 horses with induced lameness

Variable	Description*	Method of measurement
FETMAX	Maximum carpophalangeal (fetlock) joint extension during stance (degrees)	Maximum value of fetlock joint angle during stance
FETMIN	Maximum fetlock joint flexion during swing (degrees)	Minimum value of fetlock joint angle during swing
FETIME	Time of maximum fetlock joint extension (ms)	Interval between maximum fetlock joint extension and mid stance†
CARMAX	Maximum carpal joint extension during stance (degrees)	Maximum value of carpal joint angle during stance
CARMIN	Maximum carpal joint flexion during swing (degrees)	Minimum value of carpal joint angle during swing
PROT	Maximum limb protraction (cm)	Maximum hoof x – hoof x at midstance
RETR	Maximum limb retraction (cm)	Hoof x at midstance – minimum hoof x
HOOFY	Maximum hoof height during swing (cm)	Maximum hoof y during swing – minimum hoof y during stance
HOOF TIME	Time of maximum hoof height (ms)	Interval between maximum hoof y and midswing‡
STRDUR	Stride duration (ms)	Interval between hoof downII and next hoof down
STADUR	Stance duration (ms)	Interval between hoof down and toe up
CRSTADUR	Cranial stance duration (ms)	Interval between hoof down and midstance
CDSTADUR	Caudal stance duration (ms)	Interval between midstance and toe up
BRKOV	Breakover duration (ms)	Interval between heel up# and toe up
PCTSTA	Percentage of stance duration (%)	(STADUR/STRDUR) X 100
PCTCRSTA	Percentage of cranial stance duration (%)	(CRSTADUR/STADUR) X 100
PCTBRKOV	Percentage of breakover duration (%)	(BRKOV/CDSTADUR) X 100
POLMIN	Difference in minimum poll height during right and left forelimb stance phases (cm)	Difference in minimum poll height between right and left fore limb stance
POLEXCUR	Vertical poll excursion (cm)	Total vertical poll displacement during 1 complete stride

*Joint angle measurements made from palmar aspect of limb and recorded to the nearest 0.1°. Time measurements were recorded to the nearest 0.1 milliseconds and displacement measurements to the nearest 0.1 cm. †The position of the marker on the horizontal axis is indicated as x, and y is the height or position of the marker on the vertical axis. ‡Midstance is portion of stance when fetlock position in the x direction = carpal position in the x direction (ie, when metacarpus is perpendicular to the treadmill surface). §Midswing is portion during swing when hoof x = value of fetlock x at previous midstance. ||Hoof down is first time that velocity of hoof y = 0 and hoof height is within 0.25 cm of minimum value for that stride. Toe up is time when angle of the fetlock joint is equal to that observed at beginning of stance. #Heel up is first time that velocity of hoof y > 0 and hoof height is still within 0.25 cm of minimum value for that stride.