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Quantification of Equine Hoof Lameness Using an Electronic Hoof Tester

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ABSTRACT

Lameness and the treatment of lameness in equine athletes is a major concern of horse owners, trainers, and veterinarians. Although it is generally agreed that inflammatory pain is the cause of most equine lamenesses, there have been few systematic attempts to objectively quantify the pain or its relief by analgesics. Furthermore, the use of analgesic drugs in competing horses is highly controversial, since lameness is a major performance impairing condition.

In this study, an electronic hoof tester was used to objectively measure and record the degree of pain in footsore horses. A hoof tester was modified by placing a force transducer at the tip of one of the jaws. Compressive force was applied to 22 loci along the white line, sole, and frog of the foot. The force (kg) required to cause a withdrawal reaction, termed the hoof compression threshold (HCT), was recorded on a portable chart recorder and digital voltmeter.

Preliminary studies indicate that both the electronic hoof tester and procedure are sufficiently sensitive to detect (1) differences in HCT between lame and sound hooves, (2) painful and non-painful areas of the same foot, and (3) differences in the degrees of lameness in a foot before and after analgesic medication.

These data suggest that the calibrated hoof tester may be a valuable diagnostic tool for the objective assessment of equine foot lameness and its treatment.

INTRODUCTION

Hoof testers of various types have been used routinely by equine veterinarians to diagnose foot lameness.[1] However, the utility of this device has been limited by obligatory reliance on subjective assessment of the response to hoof compression. There have been no systematic attempts to standardize the hoof test procedure or calibrate the hoof tester itself. Therefore, determination of the location and severity of equine lameness varies among diagnosticians. This has made evaluation of the time course and treatment of lameness difficult and imprecise.

There have been several attempts to induce and to quantitate lameness in equines. However, most studies have employed invasive methods and/or have relied on subjective assessment of results. Some objective data have been obtained. Using a forceplate, Pratt[2] electronically measured the "unsteadiness" of a sore limb during weight bearing. Phenylbutazone (2 gm/1000 lg.) reportedly reduced this unsteadiness. Jefcott[3] reported a reduction in locomotor performance by measuring gait parameters following lactate-induced myositis. Using this model, Jones[4] observed pain and swelling at the injection site and a reduction in length of stride. Phenylbutazone diminished the pain, swelling and stride shortening. However, changes in pain and swelling were measured subjectively. Equine pain models developed by Pippi et al [5,6] were unable to detect significant changes in superficial, visceral, or deep somatic pain threshold following single anti-inflammatory doses of non-steroidal anti-inflammatory drugs.

To objectively quantify hoof lameness in the equine, a direct measure of hoof sensitivity is preferred. Since the inflammatory pain

underlying acute lameness, podotrochlitis, and laminitis is often manifest as "sensitivity to the hoof tester", an electronic calibrated hoof tester was developed. This permitted a direct measurement of the compressive force (kg) required to elicit a "pain reaction" in lame horses. Evidence is presented demonstrating the severity and localization of hoof pain, and its alleviation by local analgesia, using an electronic hoof tester.

METHODS

Hoof testing was performed on 5 horses suffering from a variety of pathologic conditions. One individual was afflicted with chronic laminitis. Two horses possessed clinical and radiographic signs of navicular disease. The remaining animals presented with signs of acute forelimb lameness.

igure 1) All hooves were examined using the modified hoof tester shown in Figure 1. An electronic force transducer (Model U2A, Hottinger Baldwin Measurements Inc., Framingham, MA) was fitted to the tip of one of the jaws of the tester. The transducer measured the isometric force (kg) applied to the exposed tip. The aperture of the tester jaws were enlarged when necessary by changing the position of the variable pivot.

igure 2) The technique of hoof compression was similar to that described by Szabuniewicz.[1] The lame hoof was cleaned thoroughly and held either between the legs of the examiner or with the hand most proximal to the animal. Contact between the brachial musculature and examiner was maintained throughout the test. In all cases a "two-handed" test was performed allowing a greater range of compressive force application to

the foot. Gradually increasing force (over 1-2s) was applied at 22 individual loci on the palmar and posterior surfaces of the hoof. Loci 1-20, located on the palmar surface of the left foot, are shown in Figure 2. Loci 1 and 11 are located at the angle of the hoof wall. Loci 12-18 are positioned around the sole. Loci 12 and 18 contact the bars, and the mid-collateral sulcus of the frog contains loci 19 and 20. Locus 21 (not shown) is positioned over the central sulcus of the frog. Locus 22 (not shown) consists of two compression points along the posterior surface of the heel near the bulbs. All loci are arranged so that pressure is always applied in a medial to lateral direction on either the left or right foot.

The kg of force required to produce a significant "painful" reaction was designated the hoof compression threshold (HCT). A reaction to hoof compression was considered painful when a hoof withdrawal occurred, or when a fasciculation, tremor or spasm of the brachial or antebrachial musculature was elicited. Force application was terminated immediately when a response was elicited. Baseline HCTs were determined prior to drug administration. An equal or greater amount of force was applied to each locus after drug treatment to insure uniformity in HCT determination over time.

The output of the force transducer was monitored on a portable polygraph. Both the rate of applied force (kg/s) and the threshold response could be evaluated from these recordings. A typical recording is shown in Figure 5. Force application rate was determined by calculating the slope of the rising phase of the analog signal. The signal peak or threshold was displayed in millivolts on a digital

voltmeter equipped with a "peak-hold" function. The force transducer was calibrated before each test by applying a standard 10 kg load to the tip. HCT was calculated by converting millivolts to kg.

Analgesia was produced by the administration of 6 ml of mepivacaine hydrochloride 2% (Carbocaine-V®, Winthrop Laboratories, NY) to the medial and lateral branches of the palmar nerves of each foot tested. Hoof compression thresholds and the number of "pain sensitive" loci were determined just prior to, and at 30, 60, 90 and 120 min post-injection.

RESULTS

The site and severity of hoof pain was assessed using the hoof test procedure described above. The localization of painful or hyperalgesic sites was determined by partitioning the palmar and posterior surfaces of the hoof into discrete regions or loci. The application of compressive force at each of these loci elicited either a positive (aversive) response or no reaction. By plotting the number of positive responses as a function of the locus of hoof compression, the anatomical distribution of painful hoof regions was determined. A distribution of the cumulative number of positive responses obtained before and after mepivacaine (Carbocaine) is shown in Figure 3. These data were collected during 6 hoof tests on 11 hooves each. Hoof tests were performed at 30-60 min intervals. The results indicate a tri-modal distribution in positive responses to hoof compression. A large number of responses occurred at loci 4-6 and 13-16, which anatomically correspond to the medial surfaces of the hoof wall and sole, and toe. A large number of responses were also observed around loci 19-21. Sensitivity to hoof compression in this

region is usually pathognomonic for navicular disease. Only a few responses were observed at loci 11 and 18, which represent more postero-lateral regions of the hoof.

ure 3) Pain relief by local analgesia was quantified by determining the reduction in the number of responsive loci and changes in hoof compression threshold, following drug administration. Figure 4 shows that mepivacaine (Carbocaine®) reduced the number of responsive loci in a time-dependent fashion. At 30 minutes virtually all positive responses were eliminated. Analgesia persisted for 60-90 min. The number of positive responses returned to baseline levels between 90 and 120 min post-injection. A slight increase in the number of responsive loci was observed at 120 and 180 min post-drug. Table 1 shows the time-dependent changes in HCT after mepivacaine (Carbocaine®) administration. A significant ($P<0.05$, Student's t-test) elevation in threshold was observed at the 30 and 60 minute tests. This agrees with the times during which a maximal reduction in responsive loci was observed. These data indicate that lame hooves, rendered analgesic by mepivacaine, could withstand greater compressive force at all previously responsive loci.

gure 4)

Table 1

Time Course of the Effects of Carbocaine on Hoof Compression Threshold (HCT)

Variable	Control	30	60	90	120
HCT	40.9 ± 1.7	56.8 ± 1.4*	56.9 ± 1.4*	42.3 ± 2.7	45.3 ± 2.9
Loci Tested	(82)	(82)	(82)	(16)	(48)

Values represent the mean (± SEM) applied force (kg) which elicited a response
*Values are significantly different ($P<0.05$) from pre-drug control.

Figure 5 shows a representative analog signal recorded from the output of the hoof tester transducer. Note the increase in peak or threshold, and the absence of a pain response 30 min after mepivacaine. The threshold and pain response returned to pre-drug control levels 90 min after mepivacaine. No significant differences between slopes of the analog signals were obtained, indicating uniformity in the rate of force application in the hoof test procedure.

igure 5)

DISCUSSION

Lameness is defined as an abnormal gait or stance due to disease or injury.[7] It is most often manifest as a compensatory response to pain. The pain underlying lameness is usually due to the release of inflammatory substances at the site of injury or disease. Navicular disease or podotrochlitis is a common cause of equine lameness,

representing around one-third of all cases of chronic forelimb lameness in the horse.[8,9]

There have been several attempts to induce and quantitate lameness in the horse.[2-4,10] While pain can contribute significantly to the clinical signs of lameness, there have been no systemic attempts to directly quantify this pain. This is probably due to the difficulty in obtaining reproducible experimental models, and the lack of quantitative instruments or techniques in the equine. To address the latter inadequacy, we constructed a calibrated electronic hoof tester which measured the amount of force (kg) applied to various regions of the foot. Horses with chronic and acute lameness were chosen which precluded the need for an experimentally-induced lameness model. By partitioning the palmar (and posterior) surface of the foot into discrete regions or loci, we established standardized sites of hoof compression. This permitted comparisons of pain sensitivity within a given hoof and between hooves of different subjects. It also provided uniformity of comparisons among investigators.

Our results indicated that painful regions of the foot could be objectively differentiated from non-painful regions (1) by the presence or absence of an aversive reaction at a given locus and (2) by the appearance of a lower HCT at sensitive versus non-sensitive loci. Differences in the degrees of hoof pain and lameness among horses could be similarly distinguished. Local analgesia clearly reduced the number of pain-sensitive loci and concommittantly raised HCT. This occurred in a time-dependent fashion and was correlated with subjective improvement in ambulation. The more severe the lameness, the greater the elevation

in HCT and reduction in numbers of pain-sensitive loci following analgesic treatment.

The time course of the analgesic effects of mepivacaine (Carbocaine®) was similar to that reported in humans following a bilateral ulnar nerve block and pinch test.[11] The duration of analgesia was shorter than that reported following a palmar metacarpal nerve block in the horse.[12] However, noxious thermal stimuli were used to evaluate non-inflammatory pain threshold in the latter study.

Sound hooves do not typically respond to the hoof tester. Therefore, "normal" values were dependent upon the degree of force application chosen by the examiner. However, in the presence of overt lameness, HCTs were consistantly lower. These values more appropriately represent "hyperalgesic" thresholds which reflect underlying inflammatory processes. Calibrated compression of the rat forepaw following Brewer's yeast injection has been used to assay the analgesic effects of non-steriodal anti-inflammatory drugs (NSAIDs).[13] Since the inflammatory processes underlying pain in both the rat paw and equine hoof are probably similar, the calibrated hoof tester should be a valid tool for assaying the effects of NSAIDs in the horse as well.

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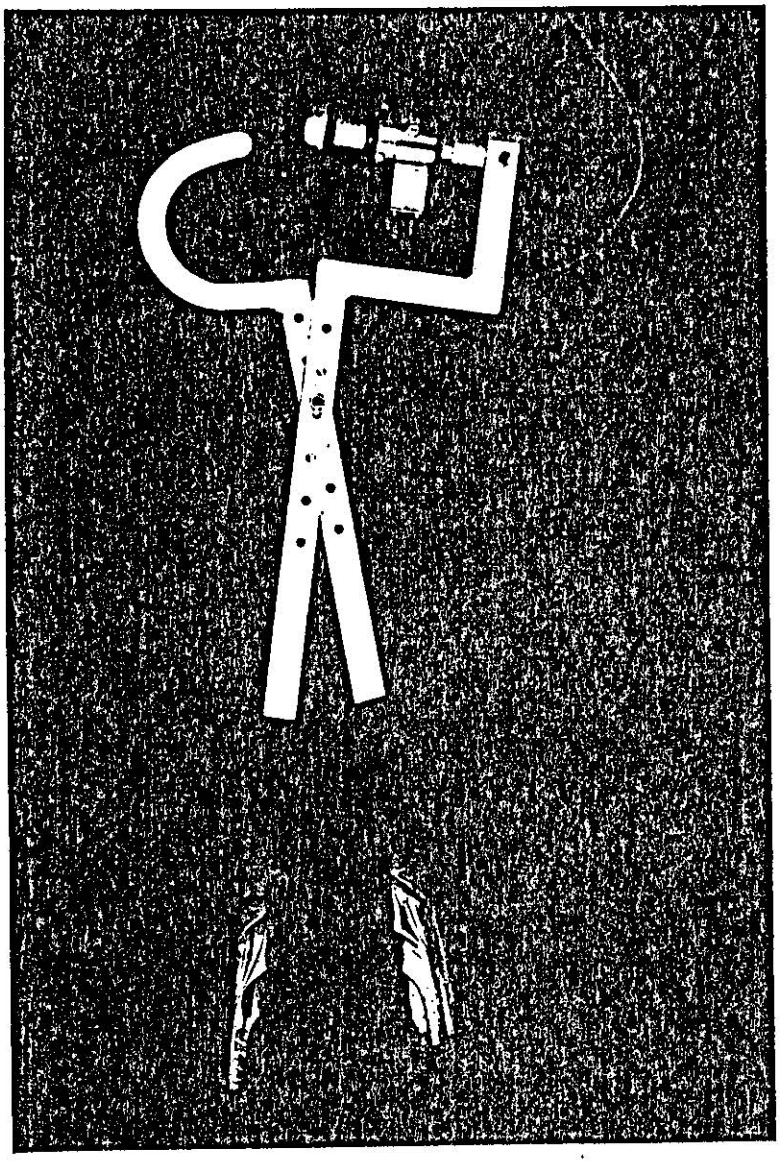

Figure 1. Electronic hoof tester. Transducer is located at top right attached to angular jaw. Holes along shank are adjustable pivot positions.

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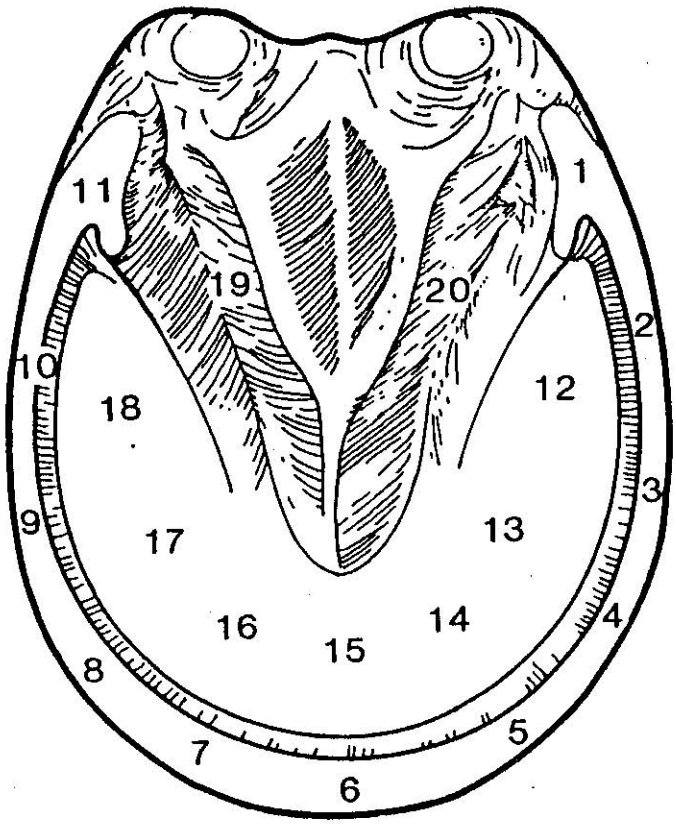

Figure 2. Palmar surface of the left forefoot with superimposed hoof test loci (1-20). Right side of hoof is medial and left side is lateral.

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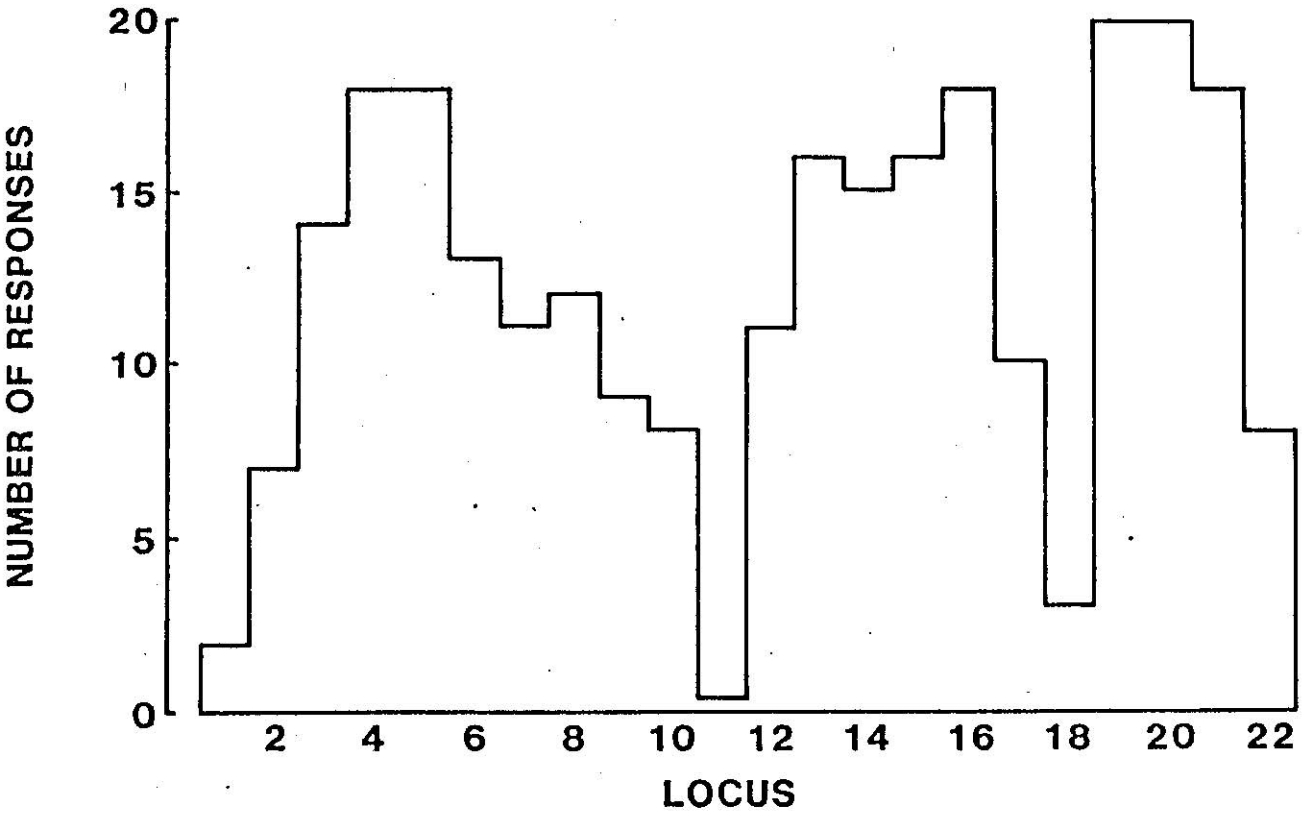

Figure 3. Plot of the cumulative number of positive responses to hoof compression at each locus.

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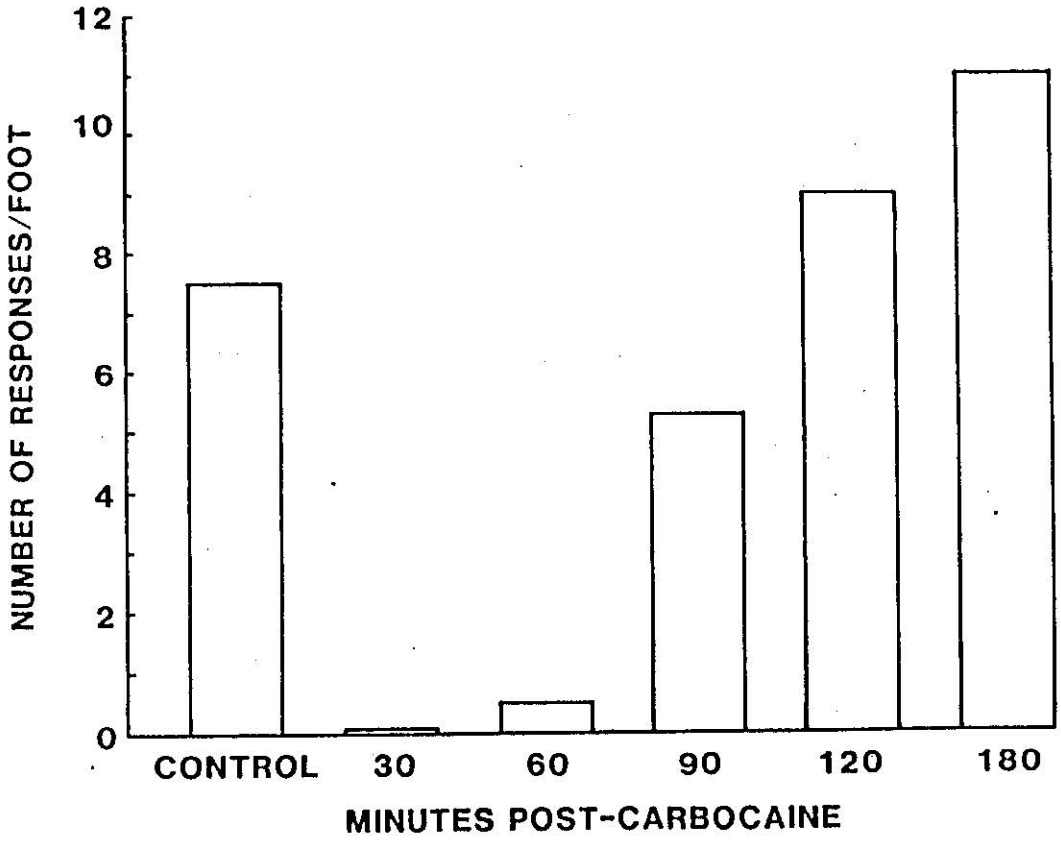

Figure 4. The effects of Carbocaine® on the number of pain responsive loci/foot.

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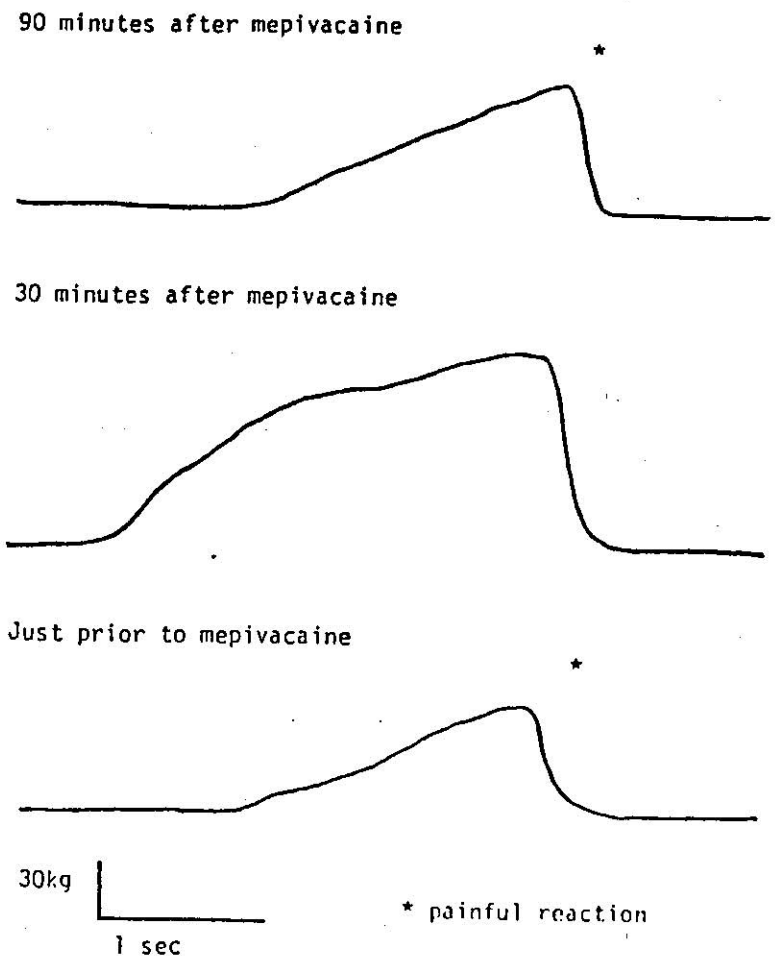

Figure 5. The effects of mepivacaine (Carbocaine®) on hoof compression threshold (kg).

From: "Quantification of Equine Hoof Lameness Using an Electronic Hoof Tester"