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Original Research

Effect of Tranquilization or Sedation on the Gait of Lame Horses

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ABSTRACT

Many clinicians are reluctant to tranquilize or sedate a horse with a subtle lameness during the course of a lameness examination, because they believe this might make lameness less evident. Previous studies have found sedation or tranquilization did change the locomotion pattern, however they did not appear subjectively to decrease the degree of lameness. This study investigated the effects of sedation or tranquilization on gait of lame horses at frequent intervals over a 45 minute period of time to determine if a commonly administered sedative, detomidine HCL, or tranquilizer, acepromazine, had a significant effect on gait over a period of time that might correlate with time spent during a lameness examination that involved several diagnostic analgesic procedures. A wireless, inertial, sensor-based, motion analysis system was used to determine the degree of lameness with and without administration of detomidine or acepromazine. Based on the results of this study, intravenous administration of 10 mg acepromazine or 10 mg detomidine does not appear to affect the degree of lameness in horses.

1. Introduction

When lameness is subtle, subjective identification and evaluation for change in gait after diagnostic analgesia may be difficult. For unruly horses or for procedures difficult to perform such as synoviocentesis of the navicular bursa, sedation with an α_2-angonist such as xylazine or detomidine may greatly facilitate and increase the safety of a procedure. Many clinicians, however, are reluctant to tranquilize or sedate a horse with a subtle lameness during the course of a lameness examination because they believe this might make lameness less evident [1–3]. The clinical experience of some lameness experts is that even low doses of xylazine, which like other α2-angonist sedatives, has analgesic properties that can significantly ameliorate or even abolish lameness [3]. A kinematic study of lame horses showed that sedation using detomidine caused no change in the degree of lameness, but did change the locomotion pattern with the authors concluding that sedation could interfere with subjective interpretation of diagnostic analgesia if lameness was subtle [4,5]. But for horses that required sedation with xylazine to perform navicular bursal injections, sedation did not appear subjectively to decrease the degree of lameness [6], and authors of a study that used inertial sensors to objectively evaluate the effect of a low dose of xylazine (0.25 mg/kg) or acepromazine (0.025 mg/kg) concluded that these drugs did not change lameness intensity [7]. Another study using inertial sensors to objectively evaluate the effect of a low dose of xylazine (0.3 mg/kg), however, found that although xylazine did not affect the evaluation of a hind limb lameness at 20 and 60 minutes, some horses with forelimb lameness had significantly decreased head movement asymmetry at 60 minutes that could have been interpreted as a positive response to diagnostic analgesia had it been performed near that time [8].

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Acepromazine, however, has no analgesic properties so its administration during a lameness might facilitate recognition of a subtle lameness [3,9]. In fact, it has been claimed that tranquilization with acepromazine will invariably increase the degree of lameness if the lameness is caused by pain [10].

Because of conflicting results of clinical studies concerning the use of a sedative or a tranquilizer during a lameness examination, we compared the gait of unsedated lame horses to their gait when sedated with detomidine or tranquilized with acepromazine to determine if treatment with these drugs might increase or decrease the degree of lameness sufficient to interfere subjectively or objectively with interpretation of results of a lameness examination. A wireless, inertial, sensor-based, motion analysis system (Lameness Locator; Equinosis LLC, Columbia, MO) was used to determine the degree of lameness. Recent studies indicate that this inertial sensor system provides appropriate accuracy (sensitivity and specificity) and repeatability in evaluation of gait when the horses trot [1–4,6–8,11,12].

2. Materials and Methods

2.1. Study Design

Ten horses (range, 409–510 kg; average, 468.9 kg body weight) that were determined by subjective evaluation two of the investigators (J.T. and J.S.) to be lame were selected from our teaching herd. These horses were, at different times, each randomly placed into group A (horses administered a 10 mg of acepromazine; Boehringer Ingleheim Vetmedica, Inc, St. Joseph, MO), group B (horses administered 10 mg of detomidine HCL; Pfizer Animal Heath, New York, NY), and group C (no treatment, control group). The study was crossover in design; horses moved to a different group at 5-day intervals until each horse had been in each group. Horses were trotted in a straight line for at least 25 strides while wearing sensors for objective evaluation using a wireless, inertial, sensor-based, motion analysis system (Lameness Locator). The same handler trotted each horse for each trial. At the end of the trials, lameness scores obtained with the Lameness Locator were subjected to statistical analysis to determine and compare the effect of sedation, tranquilization, or no treatment on gait. All procedures were approved for use by the Animal Care and Use Committee.

2.2. Data Analyses

Front leg lameness was calculated as vector sums (VSs) for all lameness trials (Fig. 1). Hind limb lameness was calculated as the sum of hip drop and hip hike (HS) for all lameness trials (Fig. 2). Vector sum and HS data were modeled using repeated measures analysis after evaluating residual plots for normality of data (PROC MIXED, SAS 9.1; SAS Institute Inc, Cary, NC). Vector sum and HS data were subjected to a $\log_{10}$ transformation to approximate a normal distribution. Correlated data were accounted for using the following linear model [13–15]:

$Y = X\beta + Z\mu + e$, where Y was the vector of observations, X was the treatment design matrix (treatment with acepromazine, detomidine, or control; left or right leg; lameness evaluations at 5, 10, 15, 20, 25, 30, 35, 40, and 45 minutes), β was the vector of fixed treatment effects, Z was the random effects design matrix (horse), μ was the vector of random block effects, and e was the vector of experimental error. To account for the nonindependence of observations within horses, five correlation structures were tested (compound symmetric, first-order autoregressive, Toeplitz, unstructured, and variance components) [13–15]. Models were compared using Akaike's information criterion [13–15]. Horse was included in models as a random effect [13–15]. The Kenward-Roger correction was used for all models [13–15]. The *P* values of multiple comparisons were adjusted using Tukey–Kramer method [16]. *P* values $\leq .05$ were considered significantly different.

3. Results

Vector sums and HS data were subjected to $\log_{10}$ transformation to approximate a normal distribution. Vector sum and HS data modeled using first-order

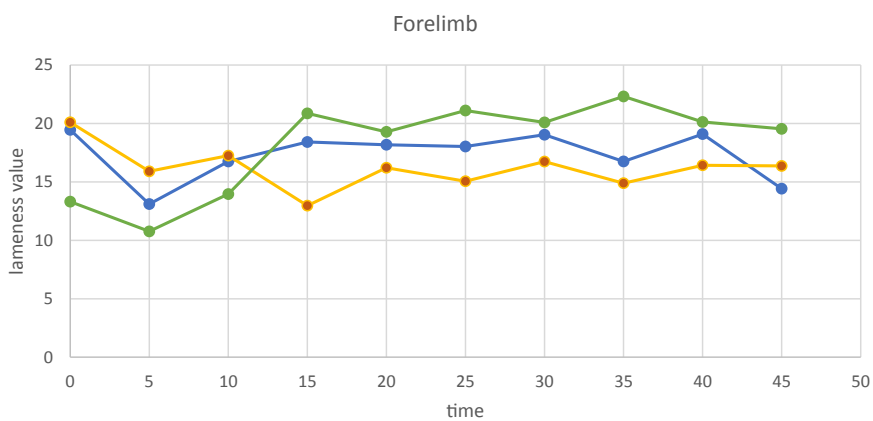

Fig. 1. Median vector sum values of horses with forelimb asymmetry after treatment with acepromazine (blue line), detomidine (orange line), or no treatment (green line).