



Gait analysis in the horse: The use of distal limb mounted Inertial Measurement Unit sensors for the detection of induced ataxia in horses

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Abstract

Reasons for performing study: Inertial Measurement Unit (IMU) sensor based techniques are becoming more popular for use in horses as a tool for objective locomotor assessment. Using currently proposed algorithmic methods only limited information about stride variables can be obtained for incoordination.

Objective: To compare several limb kinematic parameters in neurologically sound horses before and after induction of ataxia by intravenous alpha-2 agonist administration.

Study design: Prospective validation study comparing IMU sensors and motion capture with force plate data as the gold standard.

Materials & Methods: This was a pilot study. Kinematic stride parameters were obtained with IMU sensors from 2 horses at walk and at trot. The horses were measured with and without sedation with detomidine. Horses were instrumented with 8 IMU sensors, 4 placed on the limbs; one on each limb, and 4 placed on the upper body; head, withers, sternum and tuber sacrale.

Results: Comparing the stance duration (StD, $P = 0.012$), swing duration (SwD, $P = 0.017$) and stride duration (StrD, $P = 0.012$) at walk of neurological sound horses with, and without sedation, the results were significantly different. At trot the results were significantly different; StD ($P = 0.018$), SwD ($P = 0.019$) and StrD ($P = 0.011$). Comparing the coefficient of variation (CV) of the StD, SwD and StrD of neurological sound horses, the StD ($P = 0.017$) and StrD ($P = 0.012$) were significantly different, conforming a higher inter stride variation. The SwD ($P = 0.263$) was not significantly different. At trot, the CV of StD ($P = 0.123$), SwD ($P = 0.400$) and StrD ($P = 0.208$) were not significantly different.

Conclusion: The kinematic stride parameters of sedated horses is best observed and diagnosed at walk. Apparently, the pendulum at trot makes coordination for sedated horses more easy.

Ethical Animal Research: The study was approved by the local ethics committee in compliance with the Dutch Act on Animal Experimentation. **Sources of funding:** This study was funded by STW Valorisation Grant 13448.

Competing Interests: Not applicable.

Keywords: Gait, kinematic stride parameters, induced ataxia, horse, Inertial Measurement Units.

Introduction

Injuries of the locomotor system cause substantial economic and welfare implications in the sport horses. Lameness can be defined as an alteration of the normal gait pattern as a result of a functional or structural disorder of the locomotor and/or neurologic system (Morris EA, et al. 1991), most

commonly caused by pain. In equine practice, ataxia, associated with spinal cord diseases also causes a gait disorder and this is frequently observed. Neurological gait abnormalities can be recognized by clinicians, compared with lameness, when faults and anomalies in hind limb movements can be observed. Subtle signs of spinal ataxia can

easily be confused with mild musculoskeletal lameness (Reed SM, et al. 2004). Hind limb lameness and ataxia frequently must be distinguished by the practitioner, because ataxia is most commonly caused by spinal cord disorders that result in gait abnormalities most prominent in the hind limbs (Ishihara A, et al. 2009).

In equine practice obvious and suspected ataxia is a relatively common problem. The biomechanical mechanisms of ataxic gaits are yet not completely understood (Stroback et al. 2006). Horses with ataxia may show gait abnormalities due to lack of coordination of muscle movements. Foals are usually able to stand within 2 hours postpartum and are able to move in a coordinated manner shortly thereafter (Acworth. 2003). The postural control system is functional at birth in precious species such as the horse, the more complex motor patterns needs to undergo maturation to respond to perturbations (Nauwelaerts et al. 2013). Study of Nauwelaerts, 2013, indicates that newborn foals were initially quite unstable, which is indicative of poor postural control. In the first week of life the magnitudes of these variables decreased rapidly, and then more slowly over the following weeks and months as muscular strength and neuromotor control improved. They are suggesting that neonatal foals, with their incompletely developed neuromuscular reflexes, weal musculature and inexperience in postural control, rely on open-loop control, but over the first 2 weeks of life foals learn to integrate sensory input and change to a closed-loop mechanism.

The movement patterns of these ataxic horses can be compared with the movement pattern of neurological sound horses. Neurological sound horses show changes in the movement pattern after sedation with an alpha-2 agonist drugs, such as detomidine (Buchner et al. 1999). The dose-dependent clinical ataxia of alpha-2 agonist drugs is readily apparent (Hamm et al. 1995). Effects of sedation with detomidine are low height of head, low heart rate and long duration of motion cycle and transient ataxia.

It is recognized that the movement pattern of clinically ataxic horses is significantly different from the movement pattern of neurologically sound horses (Stroback et al. 2006). The movement pattern of sedated horses is significantly different from the movement pattern of ataxic horses (Stroback et al. 2006). The motion cycles of a single limb as well as between the 4 limbs of an

ataxic horse walking were significantly different.

The stance phase duration is the most reliable motion pattern parameter for differentiating ataxic and neurologically sound horses, resulting in shorter stance phases with ataxia (Stroback et al. 2006).

Horses with ataxia may be spending more time trying to place the limbs (effectively lengthening the swing phase) or constantly trying to replace an unstable stance with a potentially stable one after the next stride (Stroback et al. 2006). A complete stride is defined as touch-down of one limb to the subsequent touch-down of the same limb (Starke et al. 2012).

We hypothesized that neurological sound horses show significantly higher inter stride variation after sedation with detomidine, which will reflect as a higher CV of the measured variables; StD, SwD en StrD. In this research detomidine is used to induce transient ataxia in neurologically sound horses. The purpose of the study reported here was to identify such stride parameters in clinical cases and the use for reliable detection, quantification and differentiation of ataxia in horses.

Materials and methods

Horses

In this study, two clinic-owned Warmblood horses were used. In this study two mares were examined. The neurological sound mares were sedated with detomidine, to induce ataxia.

The horses were led in hand by an experienced handler, and lunged on the left and right rein, on hard surface (Figure 1).



Figure 1. Horse with a hind limb lameness, led in hand by an experienced handler.

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Data acquisition

The horses included in this study were fitted with one IMU sensor on each metatarsal/metacarpal bone in order to measure specific limb parameters namely; StD, SwD and StrD. There were four IMU sensors attached to the upper body of the horse; head, withers, sternum and tuber sacrale.

The eight IMU sensors were attached to the limbs and the upper body using tape and specially designed, costume made holsters. During the data collection the horses were wearing brushing boots, the four costume made holsters with IMU sensor were placed over the brushing boots. The IMU sensors were placed on the lateral aspect of the limb, and was placed with z-axis aligned with middle of the cannon bone (figure 2).



Figure 2. IMU sensors attached to the limbs. A. Fore limbs. B. Hind limb.

For the data collection, horses were wearing a surcingle which the withers sensor and sternum casebox was secured to. The IMU sensor for the head was secured to the bridle, tape was used to secure the IMU sensor to the tuber sacrale (figure 3).

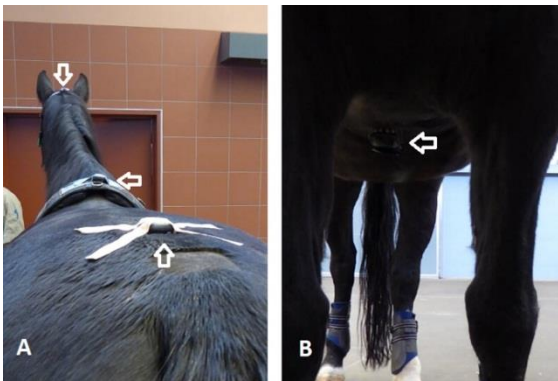


Figure 3. IMU sensors placed on the upper body of the horse. A. The IMU sensors for the data collection of the head, withers and tuber sacrale are marked with an arrow. B. The IMU sensors for the data collection of the sternum is marked with an arrow.

For the data collection the protocol “The application of limb mounted IMU sensors for the detection of lameness and ataxia in horses”, is used for individual horses.

Procedure of the horses

The IMU sensors were attached to horse 1 (630kg) and horse 2 (580kg), as described in the protocol. The horses were measured at walk and trot before the horses were sedated with 0,6ml detomidine (horse 1), and 0,3ml detomidine (horse 2). Between the moment of sedation and the second measurement there was a period of five minutes. The second measurement of horse 1 included walk at the straight line and the left and right rein, horse 2 was also measured at trot. Horse 1 was measured a third time 66 minutes after sedation. This third measurement of the horse included walk and trot on the straight line, and walk on the left and right rein.

Data processing

The collected data was exported into Matlab (r2005a) and processed used custom made scripts. The kinematic stride parameters (StD (seconds), SwD (seconds) and StrD (seconds) were calculated for each stride for each limb according to previously described and validated methods (Braganca FM et al. 2015). The mean value and standard deviation is calculated of each limb of all correct strides (seconds). The coefficient of variation (CV) was calculated as followed: $[\text{standard deviation}/\text{mean}] * 100$.

The data collected from the IMU sensors placed on the upper body, were not used in this paper.

Results

The detection of changes in kinematic stride parameters in sedated horses by the use of limb mounted IMU sensors was performed safely without any complications for all horses. The data of the neurological sound horses before sedation were compared with the data for horses after sedation. The measured mares were graded mildly to moderately ataxic after sedation.

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The collected data was exported and processed using custom made scripts. Of all the corrects strides, the mean and standard deviation were analyzed for each limb; Left Front (LF), Right Front (RF), Left Hind (LH) and Right Hind (RH). This was calculated of both horses, under different conditions, with and without sedation with detomidine. Horse 1 was measured three times, the

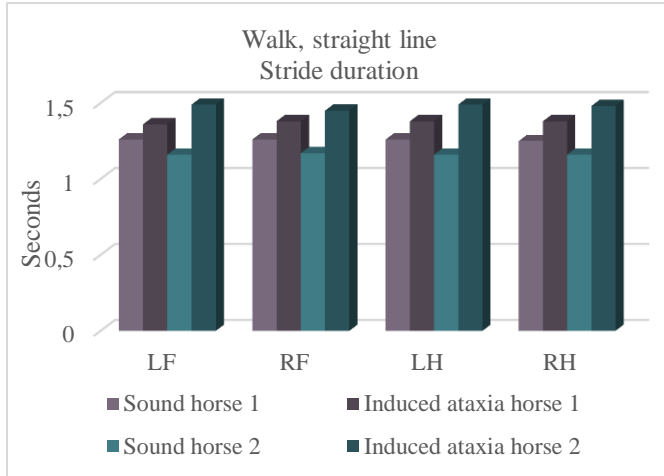


Figure 4. Walk, straight line. The StrD of the two horses, before and after sedation with detomidine.

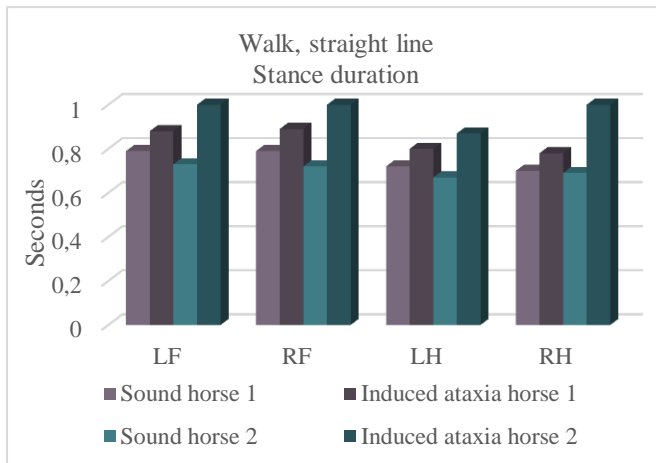


Figure 5. Walk, straight line. The StD of the two horses, before and after sedation with detomidine.

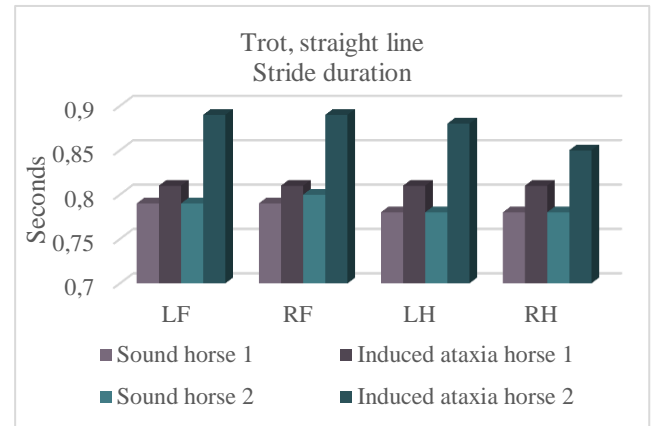
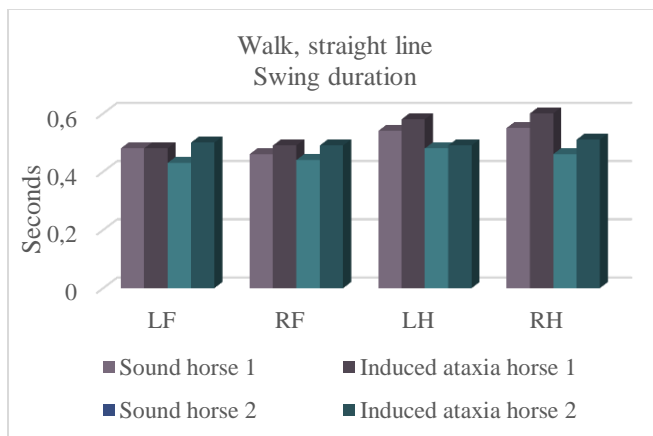


Figure 7. Trot, straight line. The StrD of the two horses, before and after sedation with detomidine.

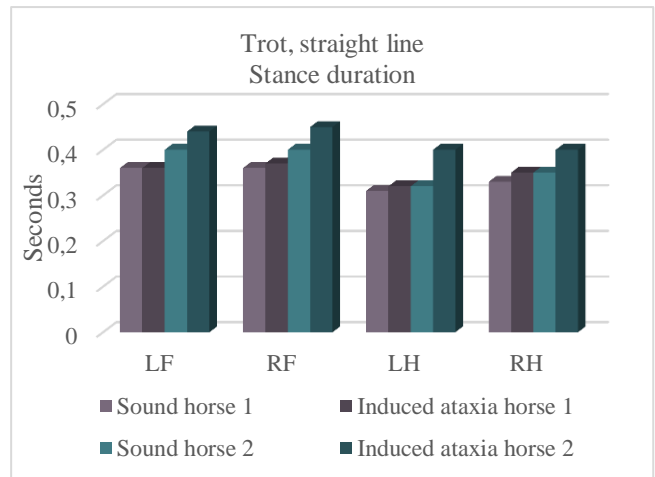


Figure 8. Trot, straight line. The StD of the two horses, before and after sedation with detomidine.

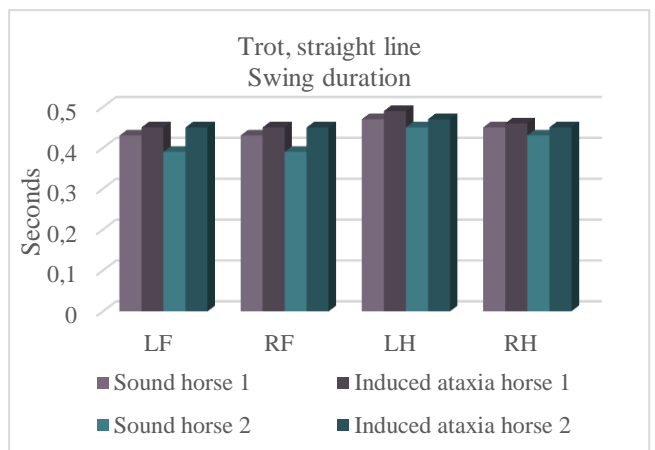


Figure 9. Trot, straight line. The SwD of the two horses, before and after sedation with detomidine.

third measurement was used for the calculations. The specific details of calculations are given in figure 4 – 9.

For this study, the Wilcoxon signed rank test (non parametric test) was used, to compare the collected data before and after induction of ataxia. This test was chosen due to small number of samples (pilot study) and data was deviating from normality. In

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straight line at walk, the ataxic horses had significantly increased StD ($P = 0.012$), SwD ($P = 0.017$) and StrD ($P = 0.017$) in all limbs compared with the data of these horses before sedation. Ataxic horse also had significantly increased StD ($P = 0.018$), SwD ($P = 0.010$) and StrD ($P = 0.011$) when the horses were trotting in a straight line. The CV ($[\text{standard deviation}/\text{mean}] * 100$) was calculated for all limbs before and after sedation. These calculations are giving in figure 10 – 15. The CV of the StD ($P = 0.017$) and StrD ($P = 0.012$) were significantly increased in walk. The CV of the

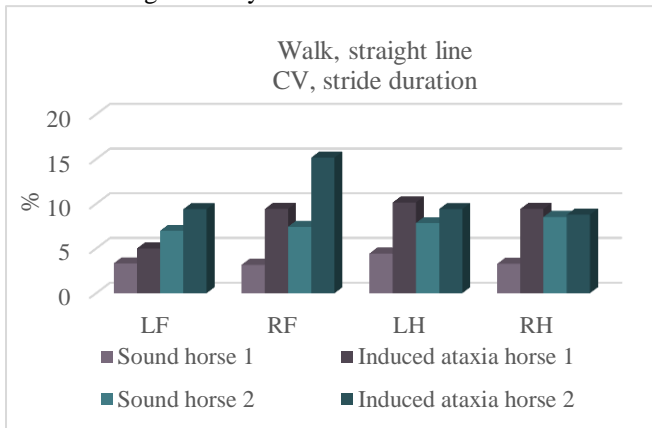


Figure 10. Walk, straight line. The CV of the StrD of the two horses, before and after sedation with detomidine.

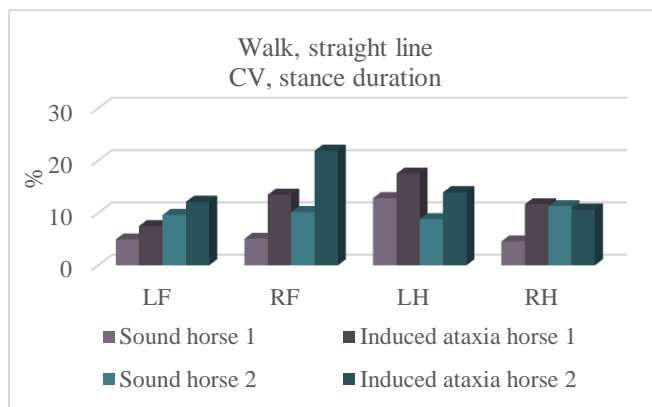


Figure 11. Walk, straight line. The CV of the StD of the two horses, before and after sedation with detomidine.

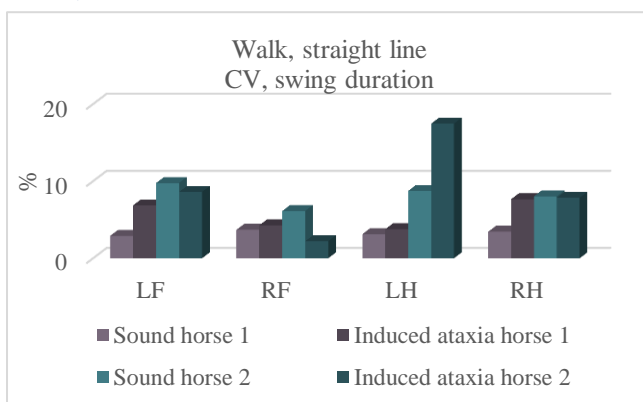


Figure 12. Walk, straight line. The CV of the SwD of the two horses, before and after sedation with detomidine.

SwD were not significantly increased ($P = 0.263$) in walk

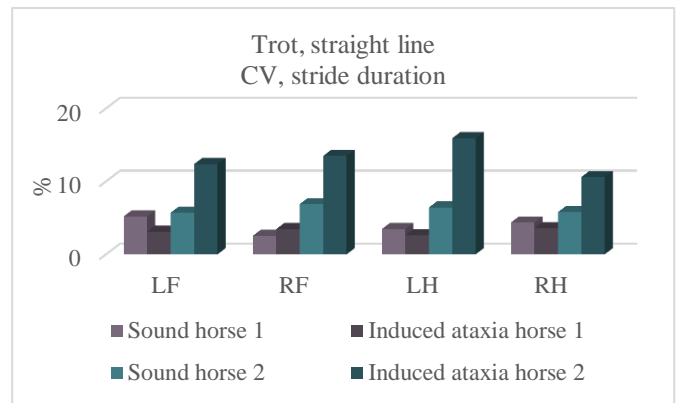


Figure 13. Trot, straight line. The CV of the StrD of the two horses, before and after sedation with detomidine.

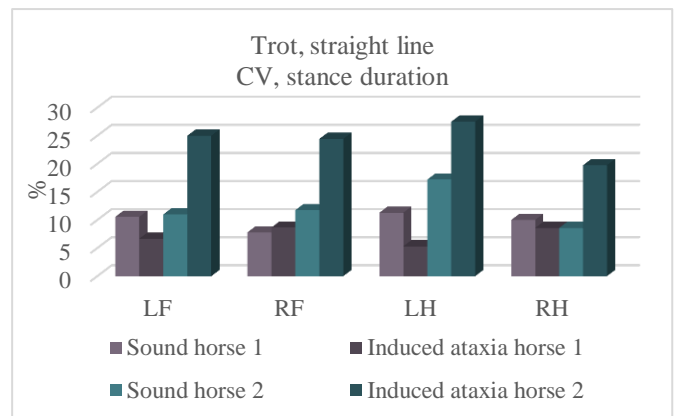


Figure 14. Walk, straight line. The CV of the StD of the two horses, before and after sedation with detomidine.

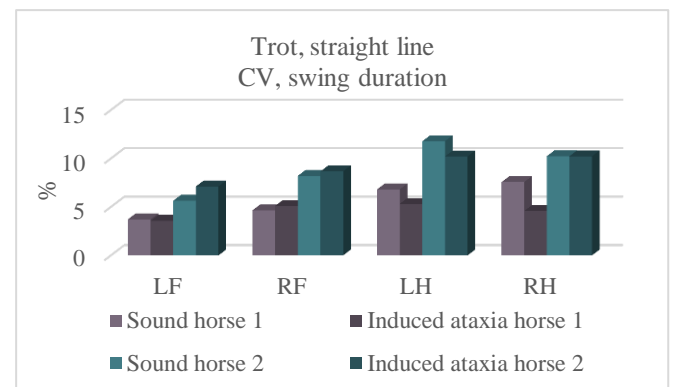


Figure 15. Walk, straight line. The CV of the SwD of the two horses, before and after sedation with detomidine.

In trot the CVs were not significantly increased, StD ($P = 0.123$), SwD ($P = 0.400$) and StD ($P = 0.208$).

Discussion

In this study we have quantified the higher inter stride variation in neurological sound horses after sedation with detomidine.

The results relating to hypothesis, that neurological

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sound horses show significantly higher inter stride variation after sedation with detomidine, which will reflect as a higher coefficient of variation of the measured variables; StD, SwD en StrD.

Horses with ataxia or sedated horses have gait pattern abnormalities, due to the lack of coordination of muscle movement. Because of the unacceptable high risk of injury, sedated horses may be measured only in walk, straight line and left and right rein. If possible, sedated horses were measured in trot, on the straight line.

At trot, horses use a “pendulum” mechanism during the symmetric trot gait (Stroback et al. 2006). Research of Stroback et al. 2006, showed that horses with ataxia found trot more easy than walk, due to this “pendulum” mechanism. Horses with ataxia may be able to use a “pendulum” mechanism during the symmetric trot gait to make balance coordination easier, but more difficult for an observer to recognize than at a walk (Ishihara A et al. 2009). Therefore ataxia is best documented and diagnosed at walk (Stroback et al. 2006).

In straight line at walk, the ataxic horses had significantly increased StD ($P = 0.012$), SwD ($P = 0.017$) and StrD ($P = 0.017$) compared with the data of these horses before sedation. Ataxic horse also had significantly increased StD ($P = 0.018$), SwD ($P = 0.010$) and StrD ($P = 0.011$) when the horses were trotting in a straight line. The outcome can be explained by the fact detomidine is slowing speed of horses after administration. Research of Lopez-Sanroman FJ et al. 2014, showed that horses had a significantly reduced speed starting 40 minutes after sublingual detomidine administration. It also resulted in a significantly reduced stride frequency (Lopez-Sanroman FJ et al. 2014).

The CV was calculated for all limbs before and after sedation. The CV (%) is calculated to prove incoordination results in a higher inter stride variation of the measured horses, which will reflect as a higher CVs, of the measured variables. The CV of the StD ($P = 0.017$) and StrD ($P = 0.012$) were significantly increased in walk. These results suggest incoordination of sedated horses, because they have significantly higher inter stride variation after sedation.

The CV of the SwD were not significantly increased ($P = 0.263$) in walk. In trot the CVs were not significantly increased, StD ($P = 0.208$), SwD

($P = 0.400$) and StrD ($P = 0.123$). This might suggest that the pendulum mechanism in trot makes coordination for sedated horses more easy, not resulting in a significantly higher inter stride variation after sedation.

The horses used in this study were sedated with detomidine. The stride parameters in seconds were significantly slower after sedation with detomidine. This is an effect of the application of detomidine, because detomidine is slowing the strides of the sedated horse. Study of the effect of oromucosal detomidine gel of L'Ami JJ et al. 2013, described the sedative and ataxic effect after administration. An evaluation of the sedative and ataxic effect was made, which gave maximal sedation effect of detomidine after 60 minutes. The head height after administration was lowest at 80 minutes. Research of L'Ami JJ et al. 2013 showed that the sedative effect of detomidine is bradycardia, after administration the maximal effect is at 60 minutes. 60, 120 and 180 minutes postadministration, the effect of sublingual administration of detomidine on the degree of ataxia was significant (L'Ami JJ, et al. 2013).

The horses were led in hand by an experienced handler, it creates a less uniform movement compared to movement on the treadmill, used in previous research. However, the horses were capable to achieve their own suitable speed for motion analysis at walk and at trot, when the horses were led in hand.

In another study by Stroback et al. 2006, the movement pattern of sedated horses was significantly different from movement pattern of ataxic horses, but the movement pattern of clinically ataxic horses was significantly different from the movement pattern of neurologically sound horses. The study showed that the movement pattern of sedation does not represent naturally occurring ataxia. A large number of ataxic horses were also lame during research of Stroback et al. 2006, due to the increased frequency of stumbling, falling or inadequate use of the locomotor system.

From the results of our study it seems likely that the incoordination of sedated horses can be measured with the IMU sensors, which is resulting in a higher inter stride variation.

This is a pilot study, only two horses were measured in this research. For more significant results, more horses should be measured.

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