



Breathing pattern and thoracoabdominal asynchrony in horses with chronic obstructive and inflammatory lung disease

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ABSTRACT

The aim of the study was to show that changes in thoracoabdominal asynchrony (TAA) between quiet breathing and CO₂-induced hyperpnoea can be used to differentiate between horses with healthy airways and those suffering from inflammatory airway disease (IAD) or recurrent airway obstruction (RAO).

The level of TAA was displayed by the Pearson's correlation coefficient (PCC) of thoracic and abdominal signals, generated by respiratory ultrasonic plethysmography (RUP) during quiet breathing and hyperpnoea. Changes in TAA were expressed as the quotient of the PCCs (PCCQ) during normal breathing and hyperpnoea. Horses with RAO and IAD showed significant higher median PCCQ than healthy horses. Median PCCQ of horses with RAO and IAD was not significantly different. Horses affected by a pulmonary disorder showed lower TAA compared to the control group.

This study suggests that TAA provides a useful parameter to differentiate horses with RAO and IAD from healthy horses.

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1. Introduction

Lung function testing in horses is of importance in early diagnosis and severity assessment of pulmonary diseases, such as inflammatory airway disease (IAD) and recurrent airway obstruction (RAO). Unfortunately, the application of pulmonary function tests in horses is still limited to veterinary schools, research institutes or referral clinics, since the use of the equipment is either too invasive or not adaptable to field conditions (Marlin and Deaton, 2007). Under stable or race track conditions a suitable test method would provide an early diagnosis and enable adequate treatment. Non-invasive techniques and equipment that measure changes in breathing pattern by assessing thoracoabdominal asynchrony would be such a suitable technique under field conditions.

Hoffman (2002b) stated that among all common pulmonary function tests oscillometry and flowmetric measurements tend to fulfill these requirements. However, the results of most pulmonary function tests depend on a wide range of normal values and might not be sufficiently discriminative to diagnose pulmonary dysfunction, unless the disease is severe (Marlin and Deaton, 2007). As stated by Leguillette (2003) the diameter of the upper airway is smaller than the added cross sectional area of all terminal bronchi and thus a relatively large number of bronchi have to be constricted to impede lung function and cause clinical signs of respiratory disease. Therefore, in research, a combination of diagnostic

tools is used to increase diagnostic sensitivity (Hoffman, 2002a). Respiratory inductance plethysmography (RIP) has been used to quantify thoracoabdominal asynchrony (TAA) (Hoffman et al., 2007). Simultaneously, the circumferential changes of thorax and abdomen, and the airflow at the nostrils were measured and both output signals were compared. Unfortunately, phase differences between circumference changes of the thorax and abdomen did not correlate with the severity of airway obstruction (Hoffman, 2002a). Thus it is recognized that a reliable diagnosis can only be made by a combination of physical examination, airway endoscopy, bronchoalveolar lavage fluid (BALF) cytology and lung function testing (Hoffman, 2002a). Moreover, the sensitivity of lung function tests can be increased by inducing hyperpnoea (Pirrone et al., 2007) or by bronchoprovocation tests such as histamine challenge (Hoffman, 2002a). Pirrone et al. (2007) found that changes to lung mechanics in horses affected with IAD become more obvious during hyperpnoea. The sensitivity of the measurement of dynamic compliance, lung resistance and respiratory power was increased using a rebreathing method. The respiratory ultrasonic plethysmography (RUP) system, as described by Schramel et al. (2012) uses ultrasonic waves to measure the circumferential changes of the thorax and abdomen during breathing. Two ethanol-filled rubber tubes with an ultrasound sender on one end and a receiver on the other are placed around the horse's abdomen and thorax. Data are sent to a computer via bluetooth and recorded.

The aim of the present study was to evaluate if the respiratory ultrasonic plethysmography (RUP) provided a reliable and easy

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method for the differentiation between healthy and diseased horses. The hypothesis was that the changes in the thoracoabdominal asynchrony between normal tidal breathing and CO₂-induced hyperpnoea are associated with the respiratory mechanical impairment present in IAD or RAO.

2. Materials and methods

2.1. Animals

From 26 February 2009 to 10 September 2009 all horses ($n = 34$) presented for a routine pulmonary consultation for a non-infectious respiratory pathology, to the Department of Equine Internal Medicine, Vetmeduni Vienna, were included in the study. An additional 12 horses from the Equine Hospital of the Vetmeduni Vienna were also included. All experimental procedures were approved by the Ethical Commission of the Vetmeduni Vienna (protocol number: 68205/162-II/106/2009). For the patients examined at the equine clinic informed consent of the owner was obtained. None of the patients had been treated for RAO or received any medications used to treat chronic airway diseases for a minimum of three months prior to the study.

Patients were allocated to one of three groups: Healthy-group, IAD-group and RAO-group based on clinical signs, history, video endoscopic findings and percentage of polymorphonuclear cells in the BALF cytology sample. BALF parameters were used as described in the consensus statement on RAO (Robinson, 2003) and IAD (Couetil et al., 2007). Diagnosis of IAD was obtained based on results of the clinical examination, airway endoscopy and BALF cytology according to the consensus statement on IAD (Couetil et al., 2007). Horses were judged to be healthy based on the BALF cytology, physical examination, endoscopic examination and absence of clinical signs attributable to chronic respiratory diseases.

2.2. Experimental protocol

A physical examination was performed on each patient enrolled in the study. Parameters collected included rectal temperature, heart rate, respiratory rate and character of lung sound on auscultation. At the time of the examination the clinician was unaware of the patient's clinical history.

An arterial blood gas sample was withdrawn from the carotid artery, dorsal to the jugular groove, on the right side of the neck, using a 3.75 cm 18 g needle. The sample was collected in a 1 mL blood gas syringe, and immediately analyzed with a blood gas analyzer (Synthesis 25, Instrumentation Laboratory). Data obtained from each analysis were the partial arterial oxygen tension (PaO₂ mmHg), the partial arterial carbon dioxide tension (PaCO₂ mmHg) and the alveolar-arterial oxygen tension difference (P(A-a)O₂ mmHg).

All patients were sedated with intravenous butorphanol (Butomidor, Richter Pharma; 0.01 mg/kg) and detomidine (Domosedan, Pfizer; 0.01 mg/kg).

To determine the thoracoabdominal asynchrony the RUP system according to Schramel et al. (2012) was used on horses restrained in stocks. Two ethanol-filled rubber tubes, placed in the 11th intercostal space and behind the last rib respectively, were used to measure alterations of abdominal and thoracic circumference during breathing, with a 100 Hz sample rate. Data were recorded by the software Buxco XA Version 2.7.9 (Buxco Electronic Inc.). Data and waveforms were analyzed post hoc with the Microsoft Office Excel 2007 statistical add in.

Measurements were performed in a quiet environment, after five min of accommodation, during normal spontaneous breathing followed by CO₂-stimulated hyperpnoea. The latter was induced

with a flexible tube of 100 mm diameter and a length of 2.8 m, attached to an airtight mask fitted around each horse's nostrils. This setup created an increased dead space of about 22 L with low resistance and induced a level of hyperpnoea that was 1.5–2 times the basal tidal volume. The percentage of CO₂ in exhaled air was recorded during breathing at rest and during hyperpnoea in a randomly selected part of the study population ($n = 9$). The measurement was performed via side stream capnography (Capnomac Ultima Anesthesia Monitor, Datex Ohmeda). The sampling line was connected to a port of the airtight mask and every 10 s the CO₂ content in percent was recorded using an infrared measurement. During normal tidal breathing a mean maximum CO₂ level of 5.6% was measured in the system, while during breathing with increased dead space a mean maximum CO₂ level of 7.6% was reached.

A video endoscopic examination of the upper and lower airways was performed using a flexible video endoscope 170 cm in length, 11 mm in diameter (CF-Q145L, Olympus). The quality, location and amount of mucus were described.

BAL was performed after lung function measurements were finished. The BAL probe was placed under visual control in one of the main bronchi. Coughing was reduced by administration of 60 mL of a 2% lidocaine solution through the working channel of the video endoscope prior to sampling. After one minute 50 mL/100 kg of an isotonic saline solution was pumped through the BAL tube and subsequently removed by gentle suction.

Samples were analyzed by the Central Laboratory, Vetmeduni Vienna, shortly after collection. Cytological specimens were prepared by cytocentrifugation (Universal 32, Hettich) at 300g for five min. Nucleated cells were counted by use of an automatic cell counting system (ADVIA 120, Siemens Healthcare Diagnostics).

2.3. Scoring system

A scoring system (Table 1) was developed using data obtained from physical and endoscopic examination, and laboratory values. The scoring system described by Tilley et al. (2011) served as a model to develop the scoring system described in Table 1. A scale from 0 to 13 to determine the severity of the respiratory tract disease was used with a cutoff-value for RAO horses with ≥ 5 points.

2.4. Patient data analyses

Four to six consecutive regular breaths were analyzed during spontaneous breathing and during hyperpnoea. Mean amplitudes and standard deviation (SD) of the circumferential changes of abdomen and thorax were calculated, separately for spontaneous breathing and hyperpnoea.

The mean amplitudes (in cm) of the abdomen and the thorax were calculated during these breaths, separately for breathing at rest and at hyperpnoea. Then the difference in the mean amplitudes (in cm) between the thorax at rest and the abdomen at rest, abdomen at hyperpnoea and thorax at hyperpnoea, respectively was calculated using the following formula:

$$\begin{aligned} aR/tR &= \text{adR} \\ aH/tH &= \text{adH} \\ aR, &\text{ means amplitude abdomen at rest} \\ tR, &\text{ means amplitude thorax at rest} \\ \text{adR}, &\text{ means amplitude difference at rest} \\ aH, &\text{ means amplitude abdomen hyperpnoea} \\ tH, &\text{ means amplitude thorax hyperpnoea} \\ \text{adH}, &\text{ means amplitude difference hyperpnoea} \end{aligned}$$

In addition, results were compared between the control group and diseased horses (IAD and RAO).

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