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Influence of valvular insufficiency and recurrent airway obstruction on haemodynamics and therapy in warmblood horses with atrial fibrillation

Heidrun Gehlen^{a,*}, Kirstin Bubeck^a, Karl Rohn^b, Peter Stadler^a

^a Equine Clinic, University of Veterinary Medicine Hanover, Foundation, D-30173 Hanover, Germany

^b Department of Biometry, Epidemiology and Information Processing, University of Veterinary Medicine Hanover, Foundation, D-30173 Hanover, Germany

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Abstract

The aim of this study was to investigate the potential haemodynamic effects of valvular insufficiency and recurrent airway obstruction (RAO) in horses with atrial fibrillation (AF).

Therefore in ten healthy horses (group 1) and 40 horses with AF a clinical examination, a lung examination, echocardiography and right heart catheterization for measurement of intracardic and pulmonary pressures were performed.

According to the clinical findings the horses with AF were subdivided into 4 groups (group 2: AF; group 3: AF/valvular insufficiency; group 4: AF/RAO; group 5: AF/valvular insufficiency/RAO).

Most of the horses of group 3 and 5 suffered from two valvular insufficiencies (mitral and tricuspid valve insufficiency: n = 11, mitral and aortic valve insufficiency: n = 2). The remaining horses showed a single mitral (n = 6), tricuspid (n = 2) or aortic valve insufficiency (n = 1) or more than two valvular insufficiencies (n = 4).

In group 2 right ventricular mean pressure (RVPm) was higher than in group 1 and 4 (P < 0.025); diastolic right ventricular pressure was higher than in group 1; PWP was higher than in group 1 and group 4; PDP was lower compared to group1. Compared to group1 in group 3 left atrial diameter (LA) was greater; the PAPs was higher and the PDP lower (P < 0.05). In group 4 RVPm and PWP was lower compared to group 2. In group 5 LA, fractional shortening and diastolic left ventricular diameter were greater, PWP and PAPs were higher and PDP lower compared to group1.

Twenty six of the 40 horses with AF (65%) were treated. Successful cardioversion to sinus rhythm occurred in 15 horses (58%). Therapy was successful in 50% of the treated horses of group 2 and 3, in 67% of the treated horses of group 4 and in 63% of the treated horses in group 5.

In conclusion the presence of valvular insufficiency or RAO influences the haemodynamics of horses with AF. © 2007 Published by Elsevier Ltd.

Keywords: Atrial fibrillation; Warmblood horse; Right heart catheterization; Pulmonary artery wedge pressure; Echocardiography; Heart valve insufficiency; RAO; Equine

1. Introduction

Horses with AF alone, which is also known as idiopathic AF, mostly show neither any clinical symptoms of cardiac disease, except the presence of the arrhythmia on auscultation, nor any echocardiographic or hemodynamic anomalies (Deem and Fregin, 1982; Reef et al., 1988). Until now there has been no consensus about the effects of AF on cardiac haemodynamics, especially at rest (Bertone and Wingfield, 1987; Reef et al., 1995). It has been reported that there is not necessarily deterioration of haemodynamics in these horses at rest, as atrial contraction has only little influence on ventricular filling at rest, and that there is a good chance for successful therapy in these horses (Reef et al., 1988, 1995; Muir and Mc Guirk, 1984). On the other hand, several studies on horses have reported changes in

^{*} Corresponding author. Tel.: +49 511 856 8185; fax: +49 511 856 7671. *E-mail address:* heidrun.gehlen@tiho-hannover.de (H. Gehlen).

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cardiac haemodynamics at rest before and after successful treatment of AF, so that AF alone seems in some cases to influence haemodynamics at rest (Muir and Mc Guirk, 1984; Killip and Boer, 1964).

It seems to be obvious that the extent of hemodynamic changes in horses with AF depends on several accompanying factors, particularly on additional cardiac and/or respiratory diseases. Right heart cardiac pressure measurements provide information on cardiac and pulmonary haemodynamics for the diagnosis and differentiation between valvular insufficiency and RAO in the horse (Muylle et al., 1986). Precapillary hypertension may be caused by interstitial or obstructive pulmonary diseases, thrombemboli, alveolar hypoxia, or left sided heart disease (Nuvtten et al., 1988; Nyman et al., 1991). In these cases one could find an increased pulmonary artery pressure (PAP) combined with a normal pulmonary artery wedge pressure (PWP, Buchwalsky, 1994). Postcapillary hypertension may be a result of acute or chronic mitral (MVI) and/or aortic valve insufficiency (AVI) with a progressive pressure increase from the left side of the heart to the pulmonary veins, the lung capillaries, and the lung arterioles and arteries. In these cases there is an increase in both PAP and PWP (Muylle et al., 1986). Pulmonary driving pressure (PDP) is the difference between the mean PAP and PWP. PDP is also used to differentiate between pulmonary hypertension induced primary by valvular insufficiency or RAO, as PDP is increased in the latter (Muylle et al., 1986). As there are no published data about the hemodynamic influences of additional valvular insufficiency and/or RAO on AF and on successful therapy in Warmblood horses, we investigated echocardiographic and cardiac pressure parameters in horses with AF.

2. Materials and methods

This study included 10 healthy Warmblood horses (group 1) with neither cardiovascular nor respiratory disease and 40 Warmblood horses with AF (group 2; Table 1). Although the exact duration of AF was unknown, AF had been detected by the referring veterinarian in all cases at least five to six months prior to presentation.(date of the last clinical examination).

The animals were subjected to a general physical examination, electrocardiography, echocardiography, and a detailed examination of the respiratory tract.

Table 1 Basic data of the horses

| Group | Age (yr) | Weight (kg) | Height (cm) | HR (bpm) |
|--------------------|------------|-------------|-------------|-----------|
| 1 (n = 10) | 4 ± 1 | 554 ± 40 | 167 ± 5 | 38 ± 5 |
| 2(n=7) | 9 ± 5 | 557 ± 48 | 172 ± 3 | 38 ± 7 |
| 3 (<i>n</i> = 13) | 8 ± 4 | 584 ± 51 | 171 ± 5 | 38 ± 10 |
| 4(n = 7) | 10 ± 5 | 560 ± 52 | 168 ± 3 | 36 ± 4 |
| 5 (<i>n</i> = 13) | 10 ± 4 | 582 ± 51 | 170 ± 4 | 41 ± 8 |

Mean age, weight, height and heart rate at rest.

In addition radiography of the thorax and right heart catheterization were performed in all horses at rest consisting of measurement of intracardiac pressure, pulmonary artery pressure (PAP), and pulmonary artery wedge pressure (PWP). Pulmonary driving pressure (PDP) was then calculated using the following formula: PDP (mmHg) = mean PAP (mmHg) – PWP (mmHg).

2.1. Examination of the lungs

The diagnosis of RAO was based on clinical signs during auscultation and thoracic percussion, arterial blood gas analysis (ABG), endoscopic examination, cytological evaluation of respiratory tract secretions (TBS) and characteristic radiographic findings.

2.2. Echocardiographic examination

Standard long and short axis two-dimensional real-time echocardiograms were performed in all horses. B-mode was used to measure the left atrial diameter (LA) using the left parasternal long axis view and the aortic (AO) and pulmonary diameters (PA). M-mode was used for the echocardiographic measurement of the left ventricular internal diameter during systole (LVDs) and diastole (LVDd), fractional shortening (FS%), interventricular septum during systole (IVSs) and diastole (IVSd), and the left ventricular free wall during systole (LVWs) and diastole (LVWd) using the right parasternal long axis view. Continuous wave and color-flow doppler echocardiography were used for detecting valvular insufficiencies.¹

Differentiation between pathological valvular insufficiency and physiological valvular back flows were made by flow velocity and flow duration (Patteson, 1996).

2.3. Right heart catheterization

After echocardiography, right atrial mean pressure (RAPm), right ventricular mean pressure (RVPm), right ventricular systolic (RVPs) and diastolic (RVPd) pressure, pulmonary arterial mean (PAPm), systolic (PAPs) and diastolic (PAPd) pressure, pulmonary arterial wedge pressure (PWP) and pulmonary driving pressure (PDP) were measured with a flow-directed balloon catheter (Swan-Ganz catheter², 7 French, 160 cm length). The catheter was inserted into the right jugular vein through an introducer system.³ The level of the shoulder joint was used as a reference marker for all pressure measurements. The animals were continuously monitored during echocardiography and heart catheterization, with a base-apex lead ECG.⁴

¹ Vingmed 750, Vingmed CFM 800.

² Arrow, Erding GmbH, Germany.

³ Cook, Peel away[®], Brisbane, QLD, Australia.

⁴ Hellige, Servomed, (SMR 211 and SMK 231, Vicom-sn) Germany.

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