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Evaluation of pulmonary wedge pressure in horses with and without left heart abnormalities detected by echocardiography

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Summary:

30 horses with echocardiographically detected left heart abnormalities and 10 horses without cardiac alterations were examined clinically, electrocardiographically and echocardiographically. A right heart catheterization was performed in all horses with a fluid-filled Swan-Ganz-catheter to obtain the pulmonary wedge pressure (PWP) as an index of left ventricular diastolic function.
The mean values for pulmonary wedge pressure showed significant differences ($p = 0.0003$) between the horses with left heart alterations (21.6 ± 5.4 mmHg) and the healthy control group (16.3 ± 2.8 mmHg).
PWP seems to be influenced by various left heart diseases. Seventeen out of 30 horses with left heart alterations showed PWP values within the normal range. Therefore, the PWP cannot be used as an index for left heart disease in general. But elevations of the pulmonary wedge pressure seem to indicate hemodynamic changes and therefore the grade of decompensation of several left heart diseases.
The assessment of the PWP might be useful especially in cases where precise assessment of hemodynamic relevance and thus prognostic evaluation is not possible by echocardiographical and clinical examination only.

Keywords: Pulmonary wedge pressure, poor performance, diastolic heart function, echocardiography

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Bestimmung des pulmonalen Kapillardruckes bei Pferden mit und ohne echokardiographisch festgestellter Anomalie des linken Herzens

30 Pferde mit echokardiographisch festgestellter Anomalie des linken Herzens und 10 herzgesunde Pferde wurden klinisch, elektrokardiographisch und echokardiographisch untersucht. Bei allen Pferden wurde eine Katheterisierung des rechten Herzens mit einem flüssigkeitsgefüllten Swan-Ganz-Katheter durchgeführt, um den pulmonalen Kapillardruck (pulmonary wedge pressure, PWP) als Index für die diastolische Funktion des linken Ventrikels zu messen.
Die Mittelwerte des pulmonalen Kapillardruckes der Pferde mit Veränderungen am linken Herzen (21,6 ± 5,4 mmHg) waren signifikant verschieden ($p = 0,0003$) von denen der gesunden Kontrollgruppe (16,3 ± 2,8 mmHg).
Der pulmonale Kapillardruck scheint von verschiedenen Erkrankungen des linken Herzens beeinflußt zu werden. 17 der 30 Pferde mit Veränderungen am linken Herzen hatten pulmonale Kapillardrucke im normalen Bereich. Deshalb kann der pulmonale Kapillardruck nicht als Index für Erkrankungen des linken Herzens allgemein benutzt werden, jedoch scheint ein erhöhter PWP hämodynamische Veränderungen anzuzeigen und somit den Grad der Dekompensation bei vielen Linksherzerkrankungen.
Die Bewertung des pulmonalen Kapillardruckes kann speziell in den Fällen von Nutzen sein, wo eine Beurteilung der Bedeutung der Hämodynamik und somit eine Beurteilung der Prognose mittels Echokardiographie und klinischer Untersuchung allein nicht möglich ist.

Schlüsselwörter: Pulmonaler Kapillardruck (PWP), mangelnde Leistung, diastolische Herzfunktion, Echokardiographie

Introduction

Before the development of echocardiography, cardiac disease in the horse was diagnosed by auscultation, electrocardiography and clinical signs of congestive heart failure (*Holmes* 1977, *Reef* 1991).
The evaluation of heart diseases in the horse has been revolutionized by echocardiography. Cardiac size and function – especially chamber enlargement, valve motion abnormalities and changes in cardiac wall motion – can be assessed by using various modes of echocardiography (*Reef* 1991, *Long* et al. 1992). Doppler echocardiography furthermore provides a noninvasive means of evaluating intracardial blood flow. It allows to semiquantitate the severity of valvular regurgitations more accurately (*Long* 1990, *Reef* 1991). By this the detection of heart diseases in early stages and the formulation of a more accurate prognosis for life and performance seems to be possible. Nevertheless, the exact quantification of the regurgitated blood volume – and by this the evaluation of the hemodynamic significance of a valvular insufficiency – remains difficult. In contrast to humans the horse seems to be a poor subject for quantitative blood flow analysis (*Reef* et al. 1989). A detected regurgitant jet of high velocity and intensity does not always represent a large amount of regurgitated blood. The quantitative evaluation of doppler flow signals in the horse is particulary limited because of the large angles obtained between the ultrasonic beam and the blood flow, especially in the mitral valve. An angle correction often results in overestimation of peak velocity, without angle correction peak velocity usually is underestimated (*Reef* et al. 1989, *Weinberger* 1991).
A lot of horses are referred for cardiac examination to rule out, whether heart disorders are contributing to poor performance. In these cases the differentiation between physiological and pathological regurgitations is sometimes difficult. Therefore, objective measures to evaluate the early stages of cardiac failure in horses

are needed before disease is sufficiently severe to cause overt cardiac enlargement detectable by echocardiography.
Ventricular function depends on preload (ventricular filling), afterload (resistance of the peripheral blood vessels) and myocardial contractility. In order to maintain cardiac output during strenous exercise a sufficient filling of the left ventricle is necessary. Cardiac output therefore is influenced by diastolic function as well as systolic function of the left ventricle. Left ventricular filling or the preload of the left ventricle is influenced by the circulating blood volume, the strength of atrial contraction and left ventricular diastolic function (*Buchwalsky* 1992).
Hemodynamic disturbances due to regurgitation of the mitral or aortic valves, insufficient ventricular diastolic relaxation or an elevated endsystolic bloodvolume, lead to an increase in left ventricular enddiastolic pressure. This backward-failure is usually seen before a decrease in cardiac output appears (forward-failure) (*Curtius* 1990, *Buchwalsky* 1992). An increase in left ventricular enddiastolic pressure leads to an elevated left atrial pressure which is transmitted retrograde to the pulmonary veins and to the pulmonary capillaries, where it can be measured as an increase in the pulmonary wedge pressure. An elevated PWP can be obtained a long time before massive clinical signs of cardiac failure occur (*Buchwalsky* 1992). PWP therefore reflects the hydrostatic tendency for pulmonary edema and provides an index of left ventricular preload and diastolic left heart failure (*O'Quin* and *Marini* 1983). PWP seems to be the most important hemodynamic determinant of exercise capacity in chronic heart failure of humans (*Packer* 1990). It can be obtained by right heart catheterization with a flow-guided balloon-tipped catheter (*Buchwalsky* 1992).
Evaluation of PWP is well established in human medicine for determination of left heart failure. The assessment of PWP in the horse was first described by *Milne* et al. (1975). Only a few reports exist about PWP values in horses with poor performance due to heart failure (*Nuytten* et al. 1988, *Sustronck* et al. 1991). No inve-

Tab. 1: Reference values for standardised imaging technique for B-Mode- and M-Mode- echocardiography (*Stadler* et al. 1988, *Robine* 1990, *Rewel* 1991, *Weinberger* 1991). Fractional shortening is given in %, all other values in mm.

standard view	#	parameter	reference values [mm]	
			endsystolic	enddiastolic
RCDLA (B-mode) right caudal long axis	1	internal diameter of left atrium		105 ± 12
	2	internal diameter of mitral valve area		100 ± 9
	3	internal diameter of left ventricle at level of chordae tendineae		128 ± 7
	4	internal diameter of left ventricle at level of papillary muscles		93 ± 11
	5	diameter of left ventricular free wall		32 ± 6
	6	diameter of interventricular septum		34 ± 5
	7	internal diameter of right atrium		73 ± 15
	8	internal diameter of left ventricle		67 ± 8
	9	diameter of right ventricular free wall		15 ± 2
RCDLA-Ao (B-mode) right caudal long axis aorta	10	internal diameter of aortic root		80 ± 4
RCDSA (B-mode) right caudal short axis	11	diameter of interventricular septum	42 ± 5	29 ± 5
	12	diameter of papillary muscle	53 ± 5	37 ± 6
	13	diameter of left ventricular free wall	33 ± 5	25 ± 4
	14	internal diameter of left ventricle	66 ± 13	110 ± 12
	15	internal diameter of right ventricle	36 ± 8	44 ± 7
	16	diameter of right ventricular free wall	20 ± 2	15 ± 2
RCRLA (B-mode) right cranial long axis	17	internal diameter of pulmonic artery root		56 ± 7
	18	internal diameter of right atrium		71 ± 11
LCDLA (B-mode) left caudal long axis	19	internal diameter of left atrium		118 ± 11
	20	internal diameter of mitral valves		107 ± 8
RCDLA (M-mode) parameters obtained at level of the chordae tendineae	21	internal diameter of left ventricle	84 ± 9	119 ± 11
	22	fractional shortening of left ventricle		28 ± 5 %
	23	diameter of interventricular septum	42 ± 8	33 ± 6
	24	amplitude of interventricular septum movement		12 ± 6
	25	diameter of left ventricular free wall	33 ± 7	27 ± 6
	26	amplitude of left ventricular free wall movement		20 ± 5

stigations are published about the relation of echocardiographic findings and PWP in horses.
The aim of the study was to evaluate, if horses with left heart diseases show different values for PWP compared to horses without heart disorders. Furthermore, our intention was to find out, if pulmonary wedge pressure is able to provide useful additional information concerning hemodynamic compensation of an echocardiographically diagnosed left heart alteration.

Materials and methods

40 horses (35 warmblooded horses, 2 thoroughbreds, 3 standardbreds) were examined clinically, electrocardiographically and echocardiographically for evidence of cardiac or pulmonary disease. 50% of the patients were dressage and show jumping horses in category M and S or race horses. Auscultation of the puncta maxima of the cardiac valves was performed. Detected heart murmurs were evaluated for intensity (grade I to V), timing, quality and location. An electrocardiogram with the Einthoven-derivations was obtained from each horse.
Echocardiography was performed with a sector scanner (Microimager 1000®, Ausonics, and Vingmed® CFM 700 E, Sonotron) using a 2.5 MHz probe. Measurements were done in B-Mode, M-Mode and pulsed wave Doppler-Mode and in some cases colour flow Doppler-Mode, referring to standardised images and normal values for warmblooded sport horses of *Stadler* et al. (1988), *Robine* (1990), *Rewel* (1991) and *Weinberger* (1991) as shown in tab. 1. For each horse 26 values in B-mode, 9 values in M-mode and semiquantitative graduations of detected valvular regurgitations in Doppler-mode were obtained from six different standard views (right caudal long axis, right caudal long axis aorta, right caudal short axis, right cranial long axis, left caudal long axis and left caudal long axis aorta (*Stadler* et al. 1988). Qualitative abnormalities, e.g. thickening of valves or mitral valve fluttering were also obtained. Semiquantitative graduation (grade + to +++) of valvular regurgitation was performed by evaluation of intensity, timing, peak velocity and area of the regurgitant jet in the Doppler echocardiogram.
After the examination the horses were classified into two groups. Group I (10 horses) included only horses whithout clinical, electrocardiographical or echocardiographical evidence of cardiac abnormalities. No horses were accepted for this group with a murmur, with abnormalities in the ECG other than 1st or 2nd degree atrioventricular block or with deviations exceeding the reference values more than 2 SD (tab. 1) in echocardiographical examination. No horses with detected valvular regurgitations were accepted for this group.
Group II (30 horses) included horses with abnormalities of the left heart detected by echocardiography (e.g. chamber enlargement, hypertrophy, valvular insufficiency or stenosis, alterations in left ventricular fractional shortening) (see tab. 2). Measurements exceeding reference values more than 2 SD were graded as pathologic. Additional right heart alterations did not lead to an exclusion from the study.
Subsequently a right heart catheterization was performed with a flow-directed balloon-tipped Swan-Ganz-catheter (Biosensors International®) of 160 cm length and 5 french diameter. The catheter has a central major lumen which is connected with a fluid-filled pressure transducer device (Gould®). A smaller second lumen exists in the wall and is used for inflation of a latex balloon at the tip of the catheter. A 1.0 ml syringe is attached via a stopcock to the minor lumen to inflate the balloon with 0.5 to 0.8 ml of air.

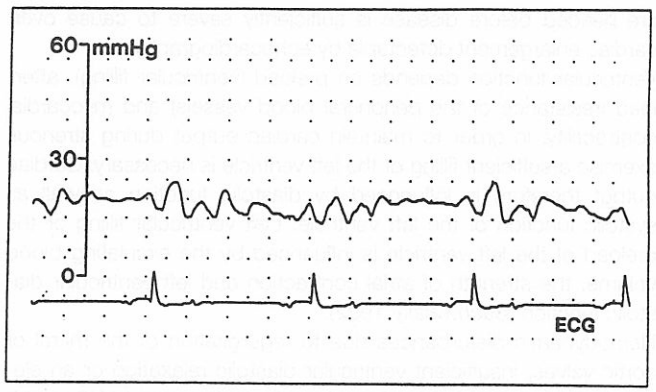

Fig. 1: Tracing of the pulmonary wedge pressure.

During the examination the horses were restrained in a stock. The skin over the distal part of the left or right jugular vein was shaved and surgically scrubbed. Because of the small diameter of the Swan-Ganz-catheter no Seldinger-technique for catheter insertion was necessary. In contrast to that a Teflon-needle (2.7 × 80 mm, G 12, Vygonüle®, Vygon® Aachen) was inserted into the vein and a sterile cover was wrapped around the horses neck. With surgically gloved hands the Swan-Ganz-catheter was connected to the pressure monitoring device. The pressure transducer was pla-

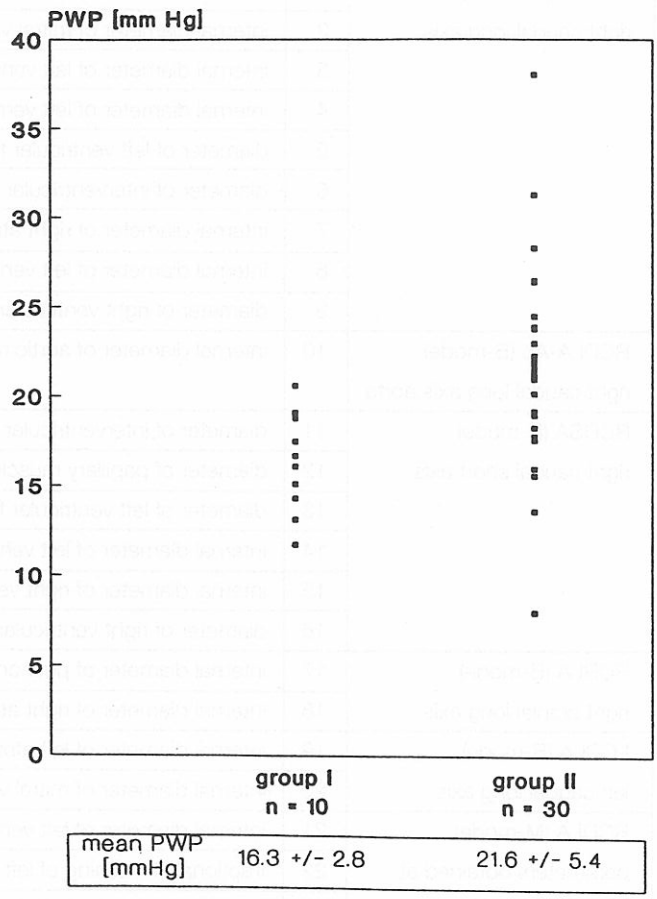

Fig. 2: Pulmonary wedge pressure values
group I = horses without evidence of heart disease
group II = horses with left heart alterations.
The statistical difference of the mean pulmonary wedge pressure between group I and group II was significant ($p = 0.0003$).

Tab. 2a

horse #	breed + use	history	clinical examination			ECG	echocardiographic examination										PWP
			heart rate	murmur	others		LA [mm]	LV [mm]	IVS [mm]	Ao [mm]	PPM [mm]	FS [%]	valves	MVI	AVI	others	[mmHg]
1	wbl pleasure	coughing	42	no	–	normal	123	117	27	75	44	36	–	–	–	–	11.7
2	wbl pleasure	wound	32	no	–	AVB	125	111	31	85	31	38	–	–	–	–	14.3
3	wbl pleasure	lameness	32	no	–	normal	115	113	27	73	34	34	–	–	–	–	20.6
4	wbl dress. M	coughing	36	no	–	normal	116	117	28	73	42	38	–	–	–	–	16.7
5	wbl pleasure	lameness	42	no	–	normal	112	120	28	72	30	32	–	–	–	–	15.1
6	wbl dress. S	lameness	32	no	–	normal	125	121	33	81	37	36	–	–	–	–	17.5
7	wbl show jump. M	wound	28	no	–	normal	138	142	33	95	28	26	–	–	–	–	13.1
8	wbl pleasure	lameness	36	no	–	normal	125	117	40	72	38	28	–	–	–	–	19.1
9	wbl breeding	lameness	36	no	–	normal	120	101	34	88	36	36	–	–	–	–	16.1
10	wbl pleasure	lameness	48	no	–	normal	108	104	26	73	32	33	–	–	–	–	18.8

Tab. 2b

horse #	breed + use	history	clinical examination			ECG	echocardiographic examination										PWP
			heart rate	murmur (punctum maximum)	others		LA [mm]	LV [mm]	IVS [mm]	Ao [mm]	PPM [mm]	FS [%]	valves	MVI	AVI	others	[mmHg]
11	wbl pleasure	coughing	28	s I-II (MV)	–	normal	119	108	23	84	33	28	MV-thickening B-notch	+	–	–	19.0
12	wbl pleasure	coughing	36	s II (MV) s I (T)	–	normal	135	104	29	77	48	***40***	B-notch	++	–	–	21.9
13	wbl pleasure	cachexia	52	ls II (MV)	–	normal	121	131	28	83	***19***	***44***	–	+(+)	–	–	21.5
14	wbl show jump. S	murmur	40	s III-IV (MV) s I-II (TV)	–	normal	130	126	34	83	34	***39***	–	++	–	–	17.8
15	thb breeding	murmur	36	d II (AV)	–	normal	135	110	30	85	24	37	MV-fluttering	–	+++	–	21.7
16	wbl show jump. M	colic	36	ls IV-V (MV)	–	normal	123	104	29	86	36	***41***	–	+++	–	–	15.9
17	wbl dress. M	murmur	28	d I-II (AV)	–	normal	115	120	28	79	30	34	–	–	+++	–	20.2
18	wbl show jump. S	murmur	28	s II-III (TV) d I (AV)	–	AVB	***148***	138	36	86	40	***39***	B-notch	+	++	–	***26.5***
19	wbl dress. M	roarer	36	ls III (MV)	–	normal	135	112	33	73	39	35	B-notch	++(+)	–	–	***22.0***
20	thb breeding	poor performance	36	s IV-V (MV/TV)	–	normal	***147***	136	33	71	35	37	B-notch	–	–	VSD	***26.4***
21	wbl dress. M	poor performance, murmur since birth	40	s IV (MV)	–	normal	***151***	142	30	79	34	***39***	MV-prolapse	+++	–	–	***23.9***
22	wbl dress. S	collapse	36	s I-II (MV)	–	AVB	135	114	34	81	33	***44***	–	(+)	+	–	21.3
23	wbl pleasure	cachexia	40	s I (MV) s I-II (TV)	–	normal	135	118	36	89	42	21	–	(+)	–	–	19.2
24	wbl show jump. M	collapse	48	s I (MV)	–	aES vES	120	130	35	81	46	38	–	–	+(+)	–	19.0
25	wbl military	poor performance	32	–	–	normal	122	121	30	77	28	36	B-notch	–	–	–	***24.5***

Tab. 2c

horse #	breed + use	history	clinical examination			ECG	echocardiographic examination										PWP
			heart rate	murmur (punctum maximum)	others		LA [mm]	LV [mm]	IVS [mm]	Ao [mm]	PPM [mm]	FS [%]	valves	MVI	AVI	others	[mmHg]
26	wbl pleasure	poor performance	32	s I (MV)	–	normal-	112	118	29	***64***	37	32	aortic stenosis	(+)	–	–	***23.8***
27	wbl pleasure	murmur	40	d I-II (AV)	–	AVB	133	113	37	85	31	***39***	B-notch	–	++	–	***23.8***
28	wbl pleasure	poor performance, cachexia	44	s I-II (MV)	–	normal-	***143***	115	34	***98***	27	***45***	–	+(+)	+(+)	–	18.3
29	wbl dress. M	poor performance, lung bleeding	32	s II (MV)	–	normal-	***181***	135	25	79	***23***	***42***	MV-prolapse B-notch	+++	–	–	***28.3***
30	wbl show jump. M	poor performance	40	–	–	aF	130	124	33	83	***46***	32	–	+(+)	–	–	21.0
31	wbl on pasture	collapse	46	s III (MV) s III-IV (TV)	–	normal-	106	116	30	74	36	35	–	–	–	VSD	***8.0***
32	show jump. S	poor performance	36	s II (MV)	–	normal-	***142***	112	46	87	37	***45***	–	+	(+)	–	13.6
33	wbl pleasure	dyspnoe	40	s III (MV)	–	normal-	***146***	127	35	86	30	***49***	–	+++	++	–	17.6
34	wbl dress. S	poor performance, coughing	28	d III (AV)	–	AVB	134	138	27	72	35	***39***	MV-fluttering	(+)	++(+)	–	***23.0***
35	wbl dress. S	poor performance	36	d IV (AV) s III (TV)	–	normal-	***143***	***155***	30	87	33	32	AV-thickening	–	++	–	***22.0***
36	wbl pleasure	poor performance	48	s IV (MV) s II (TV)	–	aF	***151***	***151***	28	83	33	36	–	++	+	–	***31.3***
37	stb racing	poor performance	32	–	–	normal-	112	120	35	72	44	27	B-notch	(+)	–	–	***22.3***
38	thb racing	weakness after racing	36	s I (MV) s III (TV)	–	normal-	117	124	40	77	35	38	–	+	(+)	–	18.6
39	wbl pleasure	dyspnoe	36	–	–	normal-	123	116	36	***70***	32	36	B-notch	+	–	–	21.0
40	stb racing	collapse	36	s III (MV) d II (AV) s II (TV)	sternal edema, lung edema	normal-	***162***	***147***	30	***67***	29	27	B-notch	+++	–	–	***38.0***

Tab. 2 a,b,c: Anamnesis and results from clinical, electrocardiographical and echocardiographical examination and heart catheterization. Only selected values are shown for the echocardiographical examination. Deviations from echocardiographic reference values (see tab. 1) and deviations from PWP ± 2 SD from reference values of PWP are shaded.
group I (horse # 1–10)
group II (horse # 11–40)

aES = atrial extrasystoles
aF = atrial fibrillation
Ao = internal diameter of aortic root
AV = aortic valve
AVB = atrioventricular block grade II
AVI = aortic valve insufficiency
B-notch = bump in the M-mode-echocardiogramm of mitral valve closing
d = holodiastolic murmur
FS = fractional shortening of the left ventricle
IVS = diameter of the interventricular Septum obtained from the right caudal long axis view
LA = internal diameter of the left atrium obtained from the left caudal long axis view
LV = internal diameter of the left ventricle obtained from the right caudal long axis view
ls = late systolic murmur
MV = mitral valve
MVI = mitral valve insufficiency
PPM = diameter of the papillary muscle obtained from the right caudal short axis view
PWP = pulmonary wedge pressure
s = holosystolic murmur
stb = standardbred
thb = thoroughbred
TV = tricuspid valve
vES = ventricular extrasystoles
VSD = ventricular septal defect
wbl = warmblooded horse

ced at a level with the shoulder joint which is reported to coincide with the level of the right atrium (*Milne* et al. 1975). The whole system was flushed with heparinized saline solution (20 000 IU/ 1 000 ml 0.9% NaCl-solution) and calibrated. Subsequently the heart catheter was inserted through the Teflon-needle into the jugular vein and the balloon was inflated with 0.5 ml of air. The location of the tip of the catheter was verified by constant blood pressure monitoring while passing the right atrium, right ventricle and pulmonary artery. From here the catheter was advanced until the balloon was wedged in a small pulmonary artery vessel. This could be recognized by a sudden pressure drop and the typical shape of the PWP (fig. 1). In this position the balloon blocked the small vessel against the right heart pressure. By this the pressure in the pulmonary capillaries could be obtained.
The mean PWP-pressure-values were obtained as the area under the curve by electronic integration of the phasic pressure signals. Measurements were made on five consecutive heart cycles.
After the recording of the PWP, the balloon was deflated and the catheter was protruded to the right ventricle. The catheter then was advanced again into the wedge-position. By this the recording of the PWP was repeated three times. From these three PWP-values the mean PWP for each horse was calculated.
Statistical analysis of the data was completed using a students-t-test for differences between body weight, age, heart rate and PWP. A probability level of $p < 0.05$ was considered statistically significant.

Results

Eight mares, 27 geldings and 5 stallions were examined. Mean age was 10.6 ± 5.2 years in group I, and 7.5 ± 3.9 years in group II. Mean body weight was 562.5 ± 65.8 kg in group I, and 534.0 ± 74.7 in group II. No statistically significant differences were found in age and body weight between group I and II. History, results from clinical and electrocardiographical examination, selected values from echocardiographical examination and values for PWP are shown in tab. 2. The 30 horses with left heart abnormalities (group II) showed various problems. Mitral valve insufficiencies were detected 22 times, aortic valve insufficiencies 13 times. Two cases of ventricular septal defect were diagnosed.
Some horses showed right heart alterations additionaly to the left heart abnormalities (tricuspid valve insufficiency in 18 horses and pulmonary valve regurgitation in 7 horses).
21 patients had combined regurgitations in two or three valves. Alterations in left heart dimensions (dilatation or hypertrophy) were seen in 16 horses.
The mean values for pulmonary wedge pressure showed significant differences ($p = 0.0003$) between the horses with left heart alterations (group II) (21.6 ± 5.4 mmHg) and the horses without cardiac abnormalities (group I) (16.3 ± 2.8 mmHg) (fig. 2).

Discussion

In human medicine PWP is used as an index for left heart failure due to coronary heart disease (*Buchwalsky* 1992) which is mainly unknown in horses. The results of this study show that the PWP seems to be influenced by various left heart diseases in the horse. The values of the PWP do not seem to be specific for a certain type of left heart disease but further investigations with larger amounts of patients would be necessary to prove this. Some horses in our study with diagnosed left heart alterations (group II) showed PWP values similar to those of group I. Only in 13 out of 30 horses of group II the obtained PWP values exceeded the reference values (mean PWP ± 2 SD) of the healthy horses. Therefore the PWP cannot be used as an index for cardiac alterations in general. Elevations of the pulmonary wedge pressure may indicate hemodynamic changes and therefore the grade of decompensation of several left heart diseases.
The values for mean PWP in this study are similar to those reported for healthy horses at rest by *Muylle* et al. (1985), *Nuytten* et al. (1988), *Sustronck* et al. (1991) and *Manohar* (1993). *Nuytten* et al. (1988) and *Sustronck* et al. (1991) reported on increased values of PWP in some horses with poor performance. In these studies chronic left heart failure was only assumed because of clinical examination and heart catheterization. No echocardiographical examination for characterization of the heart problem was performed. Nevertheless the values for PWP in their horses with assumed heart failure were closely related to our values of the horses with left heart alterations.
Most of the horses of our study were referred for cardiac examination with slight murmurs or with complaints of poor performance. The echocardiographical examination is very sensitive for detection of morphological alterations or valvular insufficiencies. The most common disorder in equine heart function are regurgitations of the mitral and aortic valve (*Else* and *Holmes* 1972). In this study these two types of valvular insufficiencies were frequently found, although some of the horses with marked valvular regurgitations did not show elevated PWP values at all.
In mitral valve insufficiency the regurgitant blood volume causes at first an increase in left atrial pressure or volume. Because of the Frank-Starling-mechanism atrial contractility increases in order to complete late diastolic ventricular filling. An increase in ventricular relaxation might as well be able to compensate hemodynamic disturbances for a long time before a severe increase in preload and PWP takes place. Marked dilatation finally is the result of the chronic volume overload (*Reindell* et al. 1988).
In the cases with aortic valve insufficiency the regurgitant blood volume leads to a volume overload in diastole and therefore to an increase in enddiastolic pressure (*Reindell* et al. 1988). The Frank-Starling-mechanism is able to compensate this by an increase in left ventricular contractility (*Buchwalsky* 1992).
Probably the low PWP values of the horses of our study with valvular insufficiencies but without chamber enlargement (e.g. horse # 11, 14, 17) can be explained by the compensatory mechanisms mentioned above. In these cases the regurgitations might be classified as clinically insignificant. Whereas the horses with elevated PWP values without chamber enlargement (e.g. horse # 19, 27, 37) might be in beginning functional decompensation, and chamber enlargement will occur subsequently.
There were some horses in our study without elevations of the PWP who showed severe left atrial or ventricular dilatation which should be indicative of functional decompensation (horse # 28, 32, 33). This shows that the PWP does not indicate the presence of heart diseases. In these cases the abnormal diastolic function might be masked because of increased myocardial compliance due to compensatory dilatation or excentric hypertrophy as seen in humans (*Gilbert* and *Glantz* 1989, *Buchwalsky* 1992). This emphasizes that for interpretation of the PWP a precise echocardiographical diagnosis is absolutely necessary.
In those cases of our study who showed marked chamber enlargement and elevated PWP, the elevation of myocardial compliance might have exceeded the morphological limits of compensation (e.g. horse # 21, 29, 36, 40). These horses did not show clinical signs of decompensation at rest. Therefore it might be of

interest for the prognostic assessment of an atrial or ventricular dilatation whether the PWP values show a marked increase or not.

All horses of our study with a severe increase of the PWP (>25.0 mmHg) had left heart dilatations. These horses obviously reached a final stage of their heart disease when even a chamber dilatation cannot mask hemodynamic failure anymore. Of course almost all of these horses had a history of poor performance. Only one patient of the 30 horses with cardiac abnormalities showed clinical symptoms of cardiac decompensation (pulmonary edema) at rest. A mild exercise provoked a hemodynamic crisis and collapse. In this horse the highest PWP value of this study (38.0 mmHg) was obtained. In those cases in which severe morphological alterations and clinical findings of cardiac decompensation are present at rest the prognostic assessment is easy. A further investigation of the PWP values of course is not necessary for an exact diagnosis.

No correlation was found in this study between increases in PWP and poor performance. The reason for this might be the inhomogenous group of patients – mainly consisting of show jumping horses and dressage horses. Especially the interindividual comparison of poor performers of those different training conditions seems to be impossible. Probably a compensation of cardiac alterations is more likely to occur when the abnormality develops gradually or the work load is not suddenly excessive (*Holmes* 1977). Further investigations and long term studies in larger amounts of patients with similar work loads are necessary to find out why some horses with cardiac abnormalities still work satisfactorily and some do not.

In conclusion, the pulmonary wedge pressure is not indicative of left heart disease. But the additional assessment of the PWP can be useful especially in cases where precise assessment of hemodynamic relevance and thus prognostic evaluation is not possible by echocardiographic and clinical examination.

References

Buchwalsky, R. (1992): Einschwemmkatheter: Technik, Auswertung und praktische Konsequenzen. 3. Aufl., Perimed-spitta, Medizinische Verlagsgesellschaft, Erlangen.

Curtius, J. M. (1990): Echokardiographische und dopplerechokardiographische Untersuchungen zur diastolischen linksventrikulären Funktion. in: *J. M. Curtius* (editor): Diagnostische Sicherheit der Echokardiographie. Springer-Verlag, Berlin, Heidelberg, 30–41.

Else, R. W. and *Holmes, J. R.* (1972): Cardiac pathology in the horse. (1) Gross pathology. Equine Vet. J. 4, 1–8.

Gilbert, J. C. and *Glantz, S. A.* (1989): Determinants of left ventricular filling and of the diastolic pressure volume relation. Circ. Res. 64, 827–852.

Holmes, J. R. (1977): Prognosis of equine cardiac conditions. Equine Vet. J. 9, 181–182.

Long, K. J. (1990): Doppler echocardiography in the horse. Equine Vet. Education 2, 15–17.

Long, K. J., Bonagura, J. D. and *Darke, P. G. G.* (1992): Standardised imaging technique for guided M-mode and Doppler echocardiography in the horse. Equine Vet. J. m24, 226–235.

Manohar, M. (1993): Pulmonary artery wedge pressure increases with high-intensity exercise in horses. Am. J. Vet. Res. 54, 142–146.

Milne, D. W., Muir, W. W. and *Skarda, R. T.* (1975): Pulmonary Arterial Wedge Pressures: Blood Gas Tensions and pH in the Resting Horse. Am. J. Vet. Res. 36, 1431–1434.

Muylle, E., Nuytten, J., Deprez, P., van den Hende, C. and *Oyaert, W.* (1985): Comparison of Three Methods for the Evaluation of Soundness of Pulmonary Function in Horses. in: *E. Deegen* and *R. E. Beadle* (editors): Lung Function and Respiratory Diseases in the Horse. International Symposium in Hannover, Germany. Hippiatrika Verlagsgesellschaft, Calw, 65–66.

Nuytten, J., Deprez, P., Picavet, T., van den Hende, C. and *Muylle, E.* (1988): Heart failure in horses: Hemodynamic monitoring and determination of LDH_1 concentration. Equine Vet. Sci. 8, 214–216.

O'Quin, R. and *Marini, J. J.* (1983): Pulmonary artery occlusion pressure: Clinical physiology, measurement, and interpretation. Am. Rev. Respir. Dis. 128, 319–326.

Packer, M. (1990): Abnormalities of Diastolic Function as a Potential Cause of Exercise Intolerance in Chronic Heart Failure. Circulation 81, Suppl. III, 78–86.

Reef, V. B. (1991): Advances in echocardiography. Vet. Clin. North Am. (Equine Practice) 7, 435–449.

Reef, V. B., Lalazari, K., DeBoo, J., van der Belt, A. J., Spencer, P. A. and *Dik, K. J.* (1989): Pulsed-wave Doppler evaluation of intracardiac blood flow in 30 clinically normal Standardbred horses. Am. J. Vet. Res. 50, 75–83.

Reindell, H., Bubenheimer, P., Dickhuth, H.-H. and *Görnandt, L.* (1988): Das druck- und volumenüberlastete Herz. in: *H. Reindell, P. Bubenheimer, H.-H. Dickhuth, L. Görnandt* (editors): Funktionsdiagnostik des gesunden und kranken Herzens. Thieme Verlag, Stuttgart, New York, 113–148.

Rewel, A. (1991): Vergleichende Messungen von Herzdimensionen und Bewegungsmustern bei Warmblut-Sportpferden mit Hilfe der M-Mode-Echokardiographie. Hannover, Tierärztl. Hochsch., Diss.

Robine, F.-J. (1990): Morphologische und funktionelle Messungen am Herzen von Warmblutpferden mit Hilfe der Schnittbildechokardiographie. Hannover, Tierärztl. Hochsch., Diss.

Stadler, P., d'Agostino, U. and *Deegen, E.* (1988): Methodik der Schnittbildechokardiographie beim Pferd. Pferdeheilkunde 4, 161–174.

Sustronck, B., Deprez, P., Picavet, T. and *Muylle, E.* (1991): Hartcatheterisatie bij vier Paarden met een verminderd Prestatievermogen. Vlaams Diergeneeskd. Tijdschr. 60, 22–25.

Weinberger, T. (1991): Doppler-Echokardiographie beim Pferd. Hannover, Tierärztl. Hochsch., Diss.

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