



# Valvular regurgitations, heart murmurs and cardiac dimensions in elite eventing horses

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# Valvular regurgitation, heart murmurs and cardiac dimensions in elite eventing horses

## Abstract

**OBJECTIVE:** To assess the characteristics of heart adaptation and the prevalence of valvular regurgitation and heart murmurs in elite eventing horses.

**ANIMALS:** 14 fit elite (competing at CIC2\* level and higher) eventing horses and 15 untrained warmblood horses.

**METHODS:** All horses underwent a physical examination, cardiac auscultation and B-Mode, M-Mode and Colour Flow Doppler echocardiography. Eventing horses were further classified by training intensity and competition level to determine possible intensity effects.

**RESULTS:** Healthy and fit eventing horses had a higher prevalence of valvular murmurs of the aorta, mitral and tricuspid valve than horses that were not trained. Also the prevalence of valvular regurgitations of all the valves detected with Doppler echocardiography was higher in the eventing group than in the control group. Right ventricle diameter (RVD), interventricular septum in diastole (IVSd), calculated left ventricle mass (LVmass) and mean wall thickness (MWT) were significantly higher in eventing than control horses. Competition level groups (control, CIC2\* and  $\geq$ CC12\*) differed in IVSd, LVmass and MWT while training level groups differed in RVD, IVSd and MWT.

**CONCLUSION:** This group of eventing horses did not show left sided heart adaptations characteristic for volume load. However, the increase in IVSd could be due to strength training or volume load of the right ventricle.

## Introduction

Stamina is defined as the ability to remain a certain speed for as long as possible. Performance on stamina is mainly influenced by the oxygen utilisation for the aerobic energy production. This can be measured as the maximal oxygen consumption expressed in  $VO_{2max}$  that is calculated by the Fick equation. The Fick equation [ $VO_{2max}=Q(CaO_2-CvO_2)$ ] is defined as the cardiac output times the difference in oxygen concentration between arterial and venous blood. The horse has a couple of mechanisms to influence these parameters to maximise the oxygen consumption during exercise. By spleen contraction, the horse has the ability to increase its Packed Cell Volume by 65% and so increases the arterial oxygen concentration. Also cardiac output can be increased 6-fold during maximal intensity exercise as consequence of the increased heart rate and stroke volume.<sup>[29]</sup>

Endurance training has the goal to increase the  $VO_{2max}$  over time. The Packed Cell Volume and the maximal heart rate can not be influenced by training<sup>[3]</sup>. So the remaining factor, the stroke volume, is the key to increase the aerobic capacity<sup>[19]</sup>. Because stroke volume is determined by the LVID, the LVID is correlated with the  $VO_{2max}$ <sup>[22,23]</sup>. Through this, a positive correlation exists between LVID and race performance in both Thoroughbreds<sup>[31]</sup> and Standardbred trotters<sup>[5]</sup>.

In humans, it is assumed that training can cause heart adaptations in two different ways<sup>[18]</sup>. Prolonged training on speed and stamina results in an increase in LVID, MWT and LVmass as seen in Standardbred trotters<sup>[5,6]</sup>, Thoroughbreds<sup>[33,31]</sup> and humans<sup>[18]</sup>. Strength trained athletes would have an increase in MWT and LVmass as a consequence of the increase resistance as seen in dressage horses<sup>[25]</sup> and humans<sup>[18]</sup>. De-conditioning results in a decrease in these parameters<sup>[11]</sup>. This training induced adapted heart is called a sport heart<sup>[6, 18]</sup>.

At the same time as the development of a sport heart as a result of training, there is an increase in prevalence of valvular regurgitation in both Standardbred trotters<sup>[6]</sup>, Thoroughbreds<sup>[32]</sup> and humans<sup>[15]</sup>. Although valvular regurgitations are regarded as undesirable during sales activities<sup>[28]</sup>, no association exists between the prevalence of a valvular regurgitation and race performance in Thoroughbreds and Standardbred trotters<sup>[6,30]</sup>. RVD is positive correlated with the prevalence of tricuspid regurgitation. So it is believed that valvular regurgitation can be a result of the training induced heart adaptations and is a feature belonging to a sport heart<sup>[9]</sup>.

Training is not the only cause for changes in heart morphology. Cardiac or vascular diseases, also valvular regurgitations, can be very demanding for the heart. The heart has to compensate for this flaw in circulation to remain the necessary cardiac output and blood pressure.<sup>[21]</sup> These diseases can cause an impaired performance or even the end of a sport career<sup>[17]</sup>. Systolic mitral murmur is the most common sign reported in cardiac disease<sup>[21]</sup>.

For the clinician it can be challenging to differentiate between heart adaptations as a consequence of training and cardiovascular disease. More often, horses in which a heart murmur is discovered during a pre-purchase examination are referred to a specialist

who is asked to give a prediction if this flaw in circulation may impair future performance.<sup>[28]</sup>

For eventing horses, stamina is very important. An eventing competition is composed of three parts. The achievements during the dressage test, the cross-country phase and the show-jumping test are transformed in penalty points. The combination that has the least penalty points is the winner. The cross-country phase is the most demanding part of the competition. Over a 3.000-6.500 meter track with 30-40 jumps the horses need to achieve a mean speed of 550 – 570 meter per minute. Because the cross-country takes more than 5 minutes and is mainly at sub-maximal intensity the main part of the energy is delivered by aerobic energy production<sup>[10,27]</sup>.

An international eventing competition under the auspices of the international equestrian federation (FEI) has four levels that are characterized by one to four stars. Each level has an inclining level of all three phases in difficulty, length, height of the fences and speed in cross-country. The levels are divided in two types of formats. In CIC competitions the cross-country phase is not longer than 3.500 meters, while the CCI competition is longer than 3.500 meter. World and Olympic championships are CCI4\* level competitions with allowed times in cross-country of more than eleven minutes.

To our knowledge it is not known if the training for the big endurance component of the cross-country phase can cause training induced heart adaptations in eventing horses, like racehorses. And also, if highly trained eventing horses have a higher prevalence of heart murmurs as in racehorses. This can be important information when eventing horses are examined and clinicians need to decide if findings are physiologic or pathologic.

The goal of this research is to reveal which training induced heart adaptation are typical for our group elite eventing horses compared to adult, breed matched, untrained horses. It also examines if this trained group has a higher prevalence and grade of audible heart murmurs and regurgitations on echocardiography. Our hypothesis is that eventing horses have the same training induced heart adaptations seen in racehorses that is also accompanied with a higher prevalence of heart murmurs, as also seen in racehorses.

## Materials and methods

### *Horses*

In this research thirty healthy warmblood horses underwent a physical and echocardiographic examination. Horses in the control group had a minimum age of three years with a mean $\pm$ SD age of 3,8 $\pm$ 0,7 years. It consisted of two stallions (13%), six mares (40%) and seven geldings (47%). The mean weight and height were 555 $\pm$ 14 kilogram and 1,65 $\pm$ 0,04 meter, respectively. They were housed in stables and/or in a field. None of control group horses had been exercised or trained in their life. Only two horses were worked minimally. Those two horses were lunged in trot at slow speed for less than two weeks with a maximum of 1 hour per week.

The ten geldings (71%) and four mares (29%) that formed the eventing group had a mean age of 11,4 $\pm$ 0,9 within a range of seven to eighteen years. The mean weight and height were 551 $\pm$ 11 kilogram and 1,65 $\pm$ 0,04 meter, respectively. All horses competed in the season they were examined, of which five (36%) competed at CIC2\*, three (21%) at CCI2\*, one (7%) at CIC3\*, three (21%) at CCI3\* and two (14%) at CCI4\* level. Because of the low number of horses in each group the horses competing at CCI2\* level and higher were pooled. For statistical analysis of competition level three groups were formed: Control (level 0), CIC2\* (level 1) and CCI2\* and higher (level 2).

### *Data Registration*

The names of the horse, the names of the owners, the dates of birth and dates of examination and highest competition levels were registered in an electronic document. After this, each horse was assigned an individual code. This code was used to ensure anonymity of both horse and owner. The data obtained from the examinations were filled in standardised electronic forms. The University Animal Ethics Board provided a positive advice for performing this study.

### *Clinical examination*

The clinical examinations were performed with the horses at rest in their home environment, in the clinic or at the competition stable area. The height of the withers was measured using a height measuring stick. Bodyweight of the horses was indirectly determined via measurement of the thoracic girth circumference using a weight tape [VIRBAC]. ECEIM Specialist Equine Internal Medicine dr. Inge Wijnberg and the author performed both a physical circulation examination. The findings of dr. Wijnberg were used for the analysis. The examination of the arterial system consisted of the frequency, amplitude, rhythm and equality of the peripheral pulse. The colour of oral and conjunctiva mucosa, the capillary refill time, turgor and peripheral temperature were examined for the capillary system. The venous system was examined by the Jugular vein and the presence of oedema and enlarged superficial skin veins. Heart auscultation was performed in a systematic manner using a stethoscope [LITHMANN]. The rhythm, intensity of the heart sounds, and the presence of heart murmurs were determined. When a heart murmur was observed the point of maximal intensity and timing was determined. Further more, it was graded according to table 1. Systolic murmurs with a punctum maximum on the atrioventricular valves or diastolic murmurs with a punctum

maximum on one of the semilunar valves were defined as valvular murmurs of the corresponding valve. Other murmurs were defined as flow murmurs, e.g. systolic with punctum maximum over the aorta.

Table 1: Classification of heart murmurs.

Grade	Characteristics
0	None audible heart murmur
1	Soft murmur, difficult to hear
2	Easy audible murmur, restricted to projection place of one valve
3	Easy audible over more than one projection place
4	Easy audible, with fremitus
5	Audible without stethoscope, with fremitus.

### *Echocardiographic examination*

Stefanie Veraa, ECVDI Specialist Veterinary Diagnostic Imaging, performed the echocardiographic examination at the same place and not more than one day apart from the clinical examination. None of the horses had to be clipped or sedated. All measurements were made while the horse had a rest heart frequency. B-Mode, M-Mode and Color flow Doppler were performed using a 1,5MHz phased array sector transducer [Esaote MyLabFive]. The measured parameters are presented in table 2. All measurements were performed in triplicate. Table 3 presents parameters that are calculated with the mean measurements of the M Mode. A base-apex ECG lead was used for timing.

Table 2: Echocardiographic measurements.

Mode	Parameter	Abbr.
B- mode	Left Atrium Diameter (systole)	LADs
	Pulmonary Artery Diameter	PAD
	Aorta Diameter	AOD
M Mode	Right Ventricle Diameter	RVDd/s
	Interventricular Septum	IVSd/s
	Left Ventricle Diameter	LVDd/s
	Left Ventricular Posterior Wall	LVPWd/s
	E-Point Septal Separation	EPSS

*d/s = Diastole / Systole*

Table 3: Calculated Parameters

Meaning	Abbr.	Formula
Mean Wall Thickness	MWT	$=(IVSd+LVPWd)/2$
Relative Wall Thickness	RWT	$=(IVSd+LVPWd)/LVDd$
Mass of left ventricle <sup>[16]</sup>	LVmass	$LVmass = 1.04 X$ $([LVDd + LVPWd +$ $IVSd]^3 - LVDd^3) -$

Colour Flow Doppler was used to determine the severity of valvular regurgitations. Categories were defined on basis of the distance the regurgitate jet was protruding away from the valve, as presented in table 4.

Table 4: definition of the regurgitation classification

Grade	Definition	Length of jet
0	None	0
1	Mild	0- 15 mm
2	Moderate	15-30 mm
3	Severe	>30 mm

#### *Registration of training status of competition horses*

After the examinations, the riders of the horses were interviewed about the training of their horses. Information about the competition history of the horse, the stable management, and eventually the training schedule were registered in a standardized form (Appendix A). All horses were ridden minimally five days a week. The majority of days they were trained on dressage and completed minimally one show jumping training per week.

Besides this, the majority of horses completed one or two training sessions on speed and/or stamina per week. Based on this information, the horses were categorized in three groups. Group 1 contains horses that were not specifically trained on stamina. These horses were only trained for dressage and jumping. Group 2 is composed of horses that, beside the dressage and jumping training, were trained on stamina. This was either done in water of a minimum height of one meter for a minimum of one time a week and a minimum duration of 25 minutes or gallop sessions (>400meter/min) minimally one time a week for a minimum duration of five minutes. Horses in the control group were classified as group 0. The interview revealed that two (14%) horses were not trained specifically for stamina (group 1). Four horses (28%) were trained in water and eight (57%) horses were galloped (Group 2).

#### *Statistical analysis*

Statistical analysis methods were chosen based on consultancy of the University statistician dr. J. Van de Broek. Descriptive analysis of height, weight and echocardiography findings was performed using the calculated mean. Difference in height and weight between groups was tested using a T-test. Descriptive analysis of valvular murmurs was performed using the frequency of severity in both groups. For the difference in the prevalence of valvular regurgitation detected with Doppler echocardiography descriptive analysis was performed.

Univariate General linear model of SPSS v.20 with body weight as covariate was used for analysing the difference in echocardiographic measurements between group, competition level and training intensity. A p-value of less than 0.05 was considered to be significant. Normality of the variables was check by the Q-Q plot of the residues. Post-hoc Bonferroni correction was used to determine which group caused the difference.



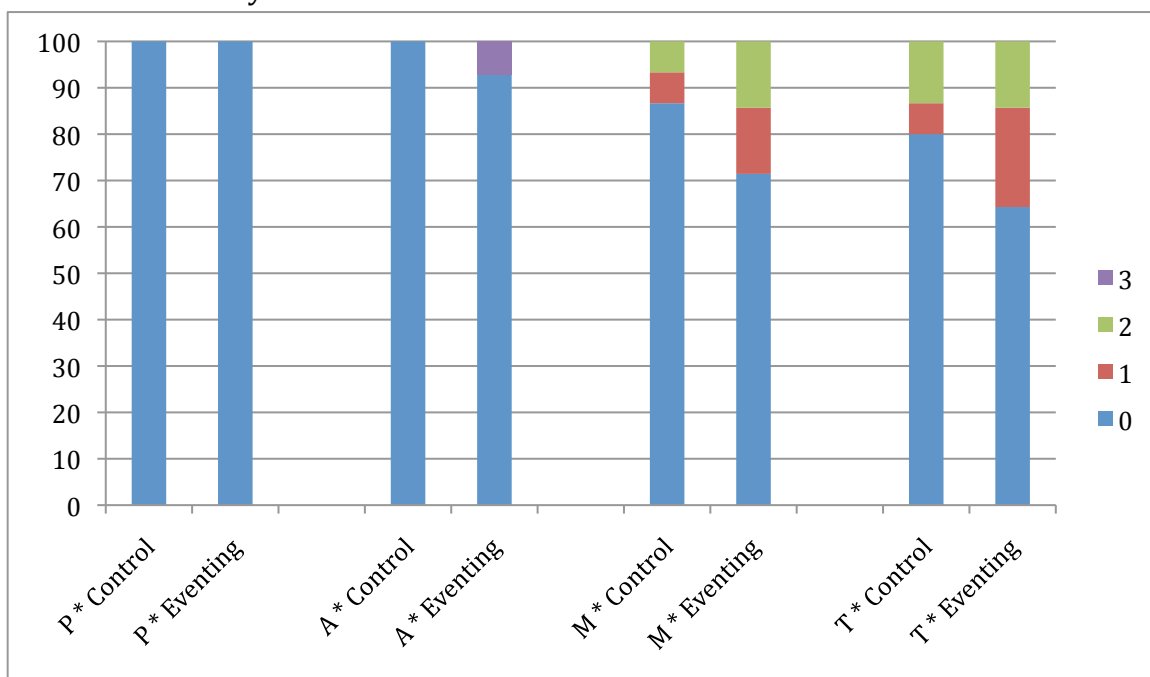
## Results

No difference in both weight and height existed between eventing and control horses. None of the horses showed abnormalities during the clinical examination. Both author and dr. Wijnberg detected the same clinical findings and timing and localisation of murmurs although the author classified some murmurs differently. Though, the one 3/5 murmur detected by dr. Wijnberg was classified the same by the author.

### *Murmurs*

In none of the horses a diastolic murmur of the pulmonary valve was detected. One eventing horse (7%) had a 3/5 diastolic murmur of the aorta valve while none of the other horses had a diastolic aorta murmur. In the control group, one horse (7%) had a 1/5 and one horse (7%) had a 2/5 systolic murmur of the mitral valve. In the eventing group two horses (14%) had a 1/5 and two horses (14%) had a 2/5 systolic murmur of the mitral valve. This makes a prevalence of systolic mitral murmurs of 13 % in the control group and 29% in the eventing group. The prevalence of systolic tricuspid murmurs was 20% in the control group since one horse (7%) had a 1/5 murmur and two horses (13%) had a 2/5 murmur of the tricuspid valve. In the eventing group, three horses (21%) had a 1/5 and two horses (14%) had a 2/5 systolic murmur of the tricuspid valve accounting for a prevalence of 36%. Two eventing horses (14%) had a systolic flow murmur over the aorta valve.

GRAPH 1: Intensity of the valvular murmurs.



P= Pulmonary valve; A= Aorta valve; M= Mitral valve; T= Tricuspid valve

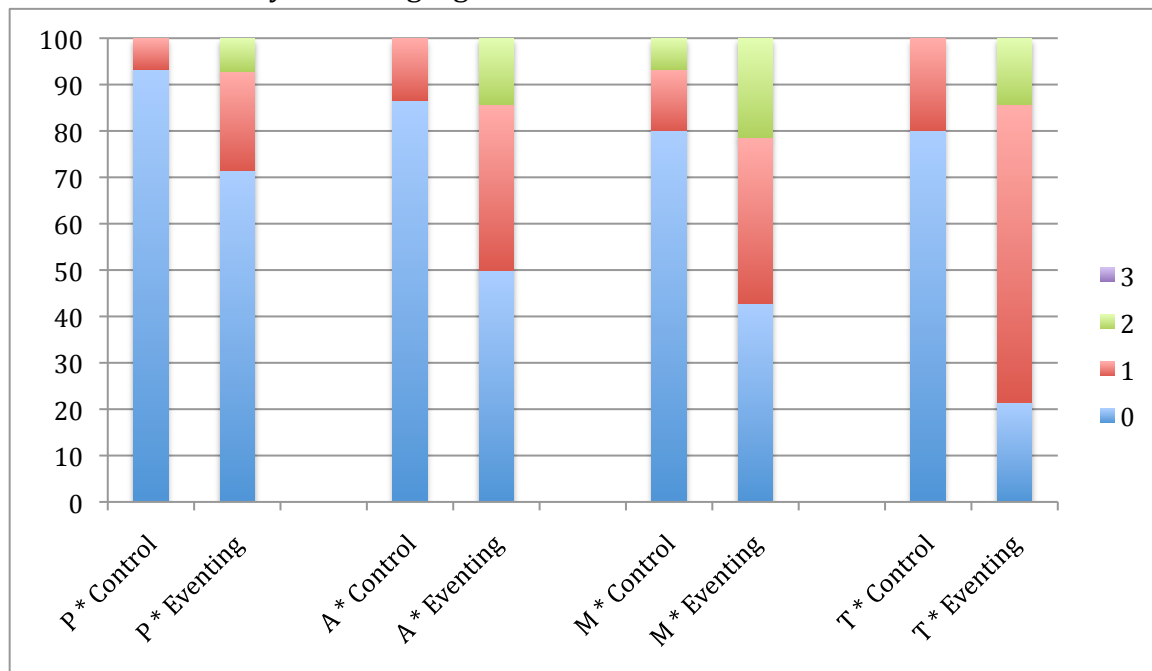
### *Regurgitations*

With Doppler echocardiography a mild valvular regurgitation of the pulmonary valve was detected in one horse (7%) out of the control group. In the eventing group three horses (21%) had a mild regurgitation and one horse (7%) had a moderate

regurgitation of the pulmonary valve. The prevalence of pulmonary valve regurgitation in the control group is 7% and in the eventing group 29%. Two (13%) of the control horses had a mild regurgitation of the aorta valve while five (36%) eventing horses had a mild and another two (14%) had a moderate regurgitation of the aorta valve. This accounts for a prevalence of aorta valve regurgitation of 13% in the control group and 50% in the eventing group.

Regurgitation of the mitral valve was shown in 20% of the control group of which two (13%) were mild and one was moderate (7%) in intensity. 57% of the horses in the eventing group had mitral valve regurgitation of which five horses (36%) had a mild and three horses (21%) had a moderate intensity regurgitation. Three control horses (20%) had a mild regurgitation of the tricuspid valve. A regurgitation of the tricuspid valve was detected in 79% of the eventing horses of which nine (64%) were mild and two (14%) were moderate intensity.

GRAPGH 2: Intensity of the regurgitation



P= Pulmonary valve; A= Aorta valve; M= Mitral valve; T= Tricuspid valve

### Echocardiography

Of the echocardiographic measurements, the RVDd (p=0,039), IVSd (p=0,003), LVmass (p=0,019) and MWT (0,013) were significantly different between the eventing and control group (table 5). There is a difference in mean of 13%, 14%, 2% and 1% of RVDd, IVSd, LVDD and LVPW respectively between control and eventing horses.

Table 5: Echocardiographic measurements and P- value obtained with GLM with bodyweight as covariate between control and eventing horses.

Parameter	Mean ±SD		P-value
	Control	Eventing	
RVDd (mm)	25 ±3	28±5	0.039*

IVSd (mm)	29 ±3	33 ±4	0.003*
LVDd (mm)	112 ±9	114 ±9	0.435
LVPWd (mm)	31 ±3	31 ±3	0.684
IVSs (mm)	48 ±4	48 ±5	0.948
LVDs (mm)	65 ±7	67 ±8	0.436
LVPWs (mm)	45 ±5	46 ±4	0.076
EPSS (mm)	8 ±3	8 ±3	0.725
LADs(mm)	120 ±9	119 ±8	0.937
PAD (mm)	52 ±5	55 ±5	0.053
AOD (mm)	70 ±6	72 ±5	0.167
LVODd (mm)	172 ±11	178 ±13	0.057
LVmass (g)	3862 ±767	4400 ±971	0.019*
MWT (mm)	30 ±2	32 ±3	0.013*

RVD: Right Ventricle Diameter; IVS: Interventricular Septum; LVD: Left Ventricular Diameter; LVPW: Left Ventricular Posterior Wall; EPSS: E-Point Septal Separation; LAD: Left Atrium Diameter (systole); PAD: Pulmonary Artery Diameter; AOD: Aorta Diameter; LVOD: calculated Left Ventricle Outer Diameter; LVmass: calculated Left Ventricle Mass; MWT: calculated Mean Wall Thickness; d: diastole; s: systole.

\* = P<0,05

There was a significant difference in IVSd (p=0,013), LVmass (p=0,039) and MWT (p=0,047) between different competition level groups (table 6). The post hoc Bonferroni revealed that IVSd (p=0,036) and LVmass (p=0,039) were significant larger in level 2 than level 0. Using Bonferroni, it was not possible to trace which specific level group caused the difference in MWT, probably because of the small sample size.

Table 6: Echocardiographic measurements and P- value obtained with GLM with bodyweight as covariate between competition level groups

Parameter	Mean			P-value
	Level 0	Level 1	Level 2	
RVDd (mm)	25	28	28	0.117
IVSd (mm)	29 <sup>§</sup>	33	34	0.013*
LVDd (mm)	112	107	118	0.186
LVPWd (mm)	31	30	32	0.900
IVSs (mm)	48	46	49	0.765
LVDs (mm)	65	61	71	0.086
LVPWs (mm)	45	46	47	0.169
EPSS (mm)	8	7	9	0.500
LADs (mm)	120	117	121	0.861
PAD (mm)	52	52	56	0.114
AOD (mm)	70	70	73	0.392
LVODd (mm)	172	170	183	0.063
LVmass (g)	3862	3853	4703 <sup>§</sup>	0.039*
MWT (mm)	30	31	33	0.047*

RVD: Right Ventricle Diameter; IVS: Interventricular Septum; LVD: Left Ventricular Diameter; LVPW: Left Ventricular Posterior Wall; EPSS: E-Point Septal Separation; LAD: Left Atrium Diameter (systole); PAD: Pulmonary Artery Diameter; AOD: Aorta Diameter; LVOD: calculated Left Ventricle Outer

Diameter; LVmass: calculated Left Ventricle Mass; MWT: calculated Mean Wall Thickness; d: diastole; s: systole.

\* =  $P < 0,05$

<sup>§</sup>=Significant different ( $P < 0,05$ ) from other groups with post hoc Bonferroni correction

Between the training intensity groups there was a significant difference in RVDd ( $p=0,047$ ), IVSd ( $p=0,010$ ) and MWT ( $p=0,040$ ) (table 7). Post hoc Bonferroni showed that IVSd of both group 0 and group 3 were different from group 2 while it was not possible to detect a significant difference between specific groups in RVDd and MWT, probably because of the small samples size.

Table 7: Echocardiographic measurements and P- value obtained with GLM with bodyweight as covariate between training intensity groups

Parameter	Mean			P-value
	Group 0	Group 1	Group 2	
RVDd	25	31	27	0.047*
IVSd	29	35 <sup>§</sup>	33	0.010*
LVDd	112	110	114	0.633
LVPWd	31	31	31	0.921
IVSs	48	46	48	0.854
LVDs	65	62	68	0.434
LVPWs	45	46	47	0.211
EPSS	8	4	9	0.070
LADs	120	121	119	0.953
PAD	52	53	55	0.147
AOD	70	74	72	0.278
LVODd	172	176	179	0.166
LVmass	3862	436	4406	0.069
MWT	30	33	32	0.040*

RVD: Right Ventricle Diameter; IVS: Interventricular Septum; LVD: Left Ventricular Diameter; LVPW: Left Ventricular Posterior Wall; EPSS: E-Point Septal Separation; LAD: Left Atrium Diameter (systole); PAD: Pulmonary Artery Diameter; AOD: Aorta Diameter; LVOD: calculated Left Ventricle Outer Diameter; LVmass: calculated Left Ventricle Mass; MWT: calculated Mean Wall Thickness; d: diastole; s: systole.

\* =  $P < 0,05$

<sup>§</sup>=Significant different ( $P < 0,05$ ) from other groups with post hoc Bonferroni correction

## Discussion

In this study 15 non-trained horses and 14 eventing horses that competed at CIC2\* level and higher underwent a physical and echocardiographic examination. It showed that healthy and fit eventing horses had a higher prevalence of valvular murmurs of the aorta, mitral and tricuspid valve than horses that are not trained. Also the prevalence of valvular regurgitations of all the valves detected with Doppler echocardiography was higher in the eventing group than in the control group. RVD, IVSd, LVmass and MWT were significantly higher in eventing than control horses. Competition level groups differed in IVSd, LVmass and MWT while training level groups differed in RVDd, IVSd and MWT.

The control group horses had a minimum age of three years to exclude an effect of normal physical growth<sup>[1]</sup>. We assumed that the limited activity of the control horses would not cause heart adaptations. This study used General Linear Model with bodyweight as covariate for the statistical analysis of the echocardiographic measurements. General Linear Model can correct for the influence of body weight. Many authors suggest that echocardiographic parameters were influenced by body weight<sup>[1,4,13,24,31]</sup>. Others disagree with this statement<sup>[14]</sup>. We wanted to include weight as covariate because of the wide range in weight in our horses. Gender is not considered in statistical analysis, since previous studies show that heart dimensions are not influenced by gender<sup>[1,34]</sup>. However, others contradict this by saying males have bigger hearts<sup>[31]</sup>.

Since there is no data available on prevalence's of valvular murmurs in untrained adult warmblood horses we could only compare our control group results with untrained but younger Thoroughbreds. Our control group had a prevalence's of valvular murmurs of the mitral and tricuspid valve (13% and 20%) that were comparable with the results obtained in 2-year-old Thoroughbreds before training (7% and 20%, respectively)<sup>[30]</sup>. Remarkably, a comparable study of the same author revealed higher prevalence's of 24% and 44% of mitral and tricuspid valve murmur in 2-year-old Thoroughbreds that were not trained yet<sup>[32]</sup>. The difference between the two Thoroughbred groups could be caused by different examination technique. The effect of the small difference in age remained uncertain.

The prevalence of aorta, mitral and tricuspid valve murmurs (7%, 27% and 36%, respectively) in our eventing horses was most comparable with the prevalence's in National hunt horses (7%, 23% and 44%, respectively)<sup>[30]</sup> and a bit higher than in 5-year-old competing Thoroughbreds (0%, 20%, 20%)<sup>[32]</sup>. The reason that others reported a lower prevalence of mitral murmurs in national hunter (5,6%)<sup>[20]</sup> and mature flat racers (1,2%)<sup>[12]</sup> remains unexplained.

Our Doppler echocardiography of the control horses revealed a prevalence of the pulmonary, aorta, mitral and tricuspid valvular regurgitation (7%, 13%, 20% and 20%, respectively) that was higher than seen in untrained, 2-year-old Standardbred trotters (0%, 0%, 3%, 8%)<sup>[6]</sup> and untrained, 2-year-old Thoroughbreds (0%, 0%, 7,3%, 12,7%, respectively)<sup>[32]</sup>. The difference could have been caused by the difference in age. However, higher prevalence's of aorta, mitral and tricuspid valve regurgitation (38%,



29%, 75%, respectively) in other 2-year-old Thoroughbreds<sup>[30]</sup> suggested that the variation in results was caused by variations in echocardiographic technique.

Results of the eventing horses also suggest that there was a big variation in echocardiographic technique between different observers. The prevalence of valvular regurgitation of the pulmonary, aorta, mitral and tricuspid valve in our eventing horses (29%, 50%, 57% and 97%, respectively) was not really comparable with the prevalence's reported in trained, 5-year-old Standardbred trotters (42,5%, 60%, 37,5% and 87,5%, respectively)<sup>[6]</sup>. Our study showed also a higher incidence of mitral and tricuspid regurgitation than reported in trained 5,5-year-old Thoroughbreds (21,8% and 25,5%, respectively)<sup>[32]</sup> adult flat racers (1,2% and 4,5%, respectively) and National hunters (5,6% and 16,4%, respectively)<sup>[20]</sup>.

The increase in prevalence in trained horses is not only due to training. Age also causes an increase in prevalence, even after they reached maturity (>7 years old)<sup>[26]</sup>. Though, a study by Buhl showed that racing trotters had a higher change of having a valvular regurgitation than non-racing peers<sup>[6]</sup>. This all is in agreement with the statement that training induces a higher prevalence of valvular regurgitation, although age has to be taken into account.

It was plausible that there was a big variation in echocardiographic technique. However we can assume that studies comparing trained and untrained horses would use exactly the same technique in both groups and did have minimal inter-observer variation bias. The same applies to murmurs. These kind of studies in Standardbred trotter<sup>[6]</sup> and Thoroughbreds<sup>[32,20]</sup> show a higher incidence of valvular regurgitation and valvular murmurs in trained horses compared to untrained horses. This was in accordance to our results in which eventing horses had higher prevalence's of both valvular regurgitation and valvular murmurs. Further more, the prevalence of both murmur and regurgitation was higher in level 2 (horses competing in CCI2\* and higher) than level 1 (CIC2\*). This is suggestive that training induces an increased prevalence of both valvular murmurs and regurgitation.

The eventing horses had a significant bigger RVDd than the control horses. Because of the U-shape of the right ventricle that envelops the left ventricle, it is challenging to standardise the measurements of the RVDd. However, the day-to-day repeatability of the RVD measurement is acceptable<sup>[9]</sup>. Our results are in accordance with a study in National Hunters that showed an increase in RVD after a 3-year training period<sup>[9]</sup>. This was suggestive that the right ventricle mimics the left ventricle in its ability to undergo eccentric hypertrophy as a consequence of prolonged endurance training, as seen in humans<sup>[7]</sup>. Though, we could not demonstrate this in our results, since the group of horses not specifically trained for stamina (group 1) had the biggest RVDd while the horses trained for stamina (group 2) or competing at the highest level (Level 2) was not significant bigger.

The IVSd measured in our study was larger than measured in Thoroughbreds<sup>[31]</sup> and comparable to Standardbred trotters<sup>[34]</sup>. The IVSd measurements of a mixed population of warmblood horses in another study were smaller<sup>[1]</sup>. Our finding that eventing horses had bigger IVSd than the control horses was in accordance to studies in

Thoroughbreds<sup>[31,33]</sup> and high-level dressage horses<sup>[25]</sup>. Good performing endurance horses appear to have a thicker IVSd than poor performers<sup>[8]</sup>.

The LVPW of both groups was larger than seen in Thoroughbreds<sup>[31]</sup>, Standardbred trotters<sup>[11,34]</sup> and other warmblood horses<sup>[1]</sup>. The LVPWd increased as a consequence of training in both thoroughbred race horses<sup>[31]</sup> and warmblood dressage horses<sup>[25]</sup> which in contrast to our study. Detraining resulted in a decrease in LVPWd in Standardbred trotters<sup>[11]</sup>.

The larger MWT in eventing horses than control horses in our study was in accordance to the difference between trained and untrained Thoroughbreds<sup>[31,33]</sup>, standard bred trotters<sup>[6]</sup> and warmblood dressage horses<sup>[25]</sup>. The difference in MWT in our study was mainly due to the difference in IVSd. The reason why there was an increase in IVSd but not in LVPW remains unknown. A possibility is that the increase in IVSd is due to eccentric hypertrophy of the right ventricle and not caused by eccentric hypertrophy of the left ventricle which is seen in other studies<sup>[6,31,33]</sup>. As a consequence of the high amount of dressage training in our eventing horses it is also possible that the heart would responded as it is trained for resistance like dressage horses<sup>[25]</sup>.

The larger calculated LVmass in eventing horse compared to control horses was in line with the increase in LVmass as a consequence of training in Thoroughbreds<sup>[31,33]</sup> and Standardbred trotters<sup>[6]</sup>. That horses competing at higher level (level 2) had larger LVmass than the other groups was in accordance with the finding that there is a correlation between race performance and LVmass in Thoroughbreds<sup>[31]</sup>.

The difference in mean LVD between eventing horses and control horses was 2% in this study. The LVD of control horses and horses competing at CCI2\* level and higher (level 2) differed 4,5%. This was far less than seen in racehorses. The LVD of Thoroughbreds increased 7% over a one-year training period<sup>[31]</sup>. In Standardbred trotters, the LVDD can increased 14% over a 4-year training period. Though, these results have to be nuanced. Because of the young age at which racehorses commenced training, there was also an effect of growth<sup>[5]</sup>. The increase in LVD that could be achieved in Standardbred trotters older than three year old is 6%<sup>[6]</sup>. The hypothesis, that eventing horses would have a bigger LVDD than untrained horses as a consequence of training, had to be rejected. This was in contrast with the training effect in Thoroughbreds<sup>[31,33]</sup> and Standardbred trotters<sup>[6]</sup>. Even high level warmblood show jumpers had bigger LVDD than untrained horses<sup>[25]</sup>. Probably the amount and intensity of the training commenced to eventing was not sufficient to cause an effect.

Both control and eventing group horses have a smaller LVDD than observed in Thoroughbreds at the time they commence training at 1,5 years of age while the mean weight of Thoroughbred was lower<sup>[31]</sup>. The LVDD of Standardbred trotters at 1,5 year of age was only little smaller than measured in our eventing group while these horses were lighter and did not commence training<sup>[6]</sup>. Another study showed that the LVD in Thoroughbreds was bigger than in warmblood horse<sup>[5]</sup>. That warmblood horses have smaller LVDD than race horses could possibly been caused by a genetic component that predisposes racehorses to have a higher aerobic capacity.

Our hypothesis was that eventing horses would have heart adaptations characteristic for endurance training. Though, our results suggest that eventing horses show more signs of resistance training because of the increase in IVSd. It is also possible that the increase in IVSd is due to eccentric hypertrophy of the right ventricle that is characteristic for endurance training. Than it remains unknown why the heart would show eccentric hypertrophy of the right ventricle without changes in the left ventricle. The higher prevalence of valvular murmurs and regurgitation in eventing horses compared control horses is in accordance results in racehorses and human athletes.

## References

- 1 Al-Haidar A, Farnir F, Deleuze S, Sandersen CF, Leroux AA, Borde L, Cerri S, Amory H. Effect of breed, sex, age and body weight on echocardiographic measurements in the equine species. *Res Vet Sci.* 2013 Aug;95(1):255-60.
- 2 Amory H, Votion DM, Fraipont A, Goachet AG, Robert C, Farnir F, Van Erck E. Altered systolic left ventricular function in horses completing a long distance endurance race. *Equine Vet J Suppl.* 2010 Nov;(38):216-9.
- 3 Betros CL, McKeever KH, Kearns CF, Malinowski K. Effects of ageing and training on maximal heart rate and VO<sub>2</sub>max. *Equine Vet J Suppl.* 2002 Sep;(34):100-5.
- 4 Brown DJ, Rush JE, MacGregor J, Ross JN Jr, Brewer B, Rand WM. M-mode echocardiographic ratio indices in normal dogs, cats, and horses: a novel quantitative method. *J Vet Intern Med.* 2003 Sep-Oct;17(5):653-62.
- 5 Buhl R, Ersbøll AK, Eriksen L, Koch J. Changes over time in echocardiographic measurements in young Standardbred racehorses undergoing training and racing and association with racing performance. *J Am Vet Med Assoc.* 2005 Jun 1;226(11):1881-7.
- 6 Buhl R, Ersbøll AK. Echocardiographic evaluation of changes in left ventricular size and valvular regurgitation associated with physical training during and after maturity in Standardbred trotters. *J Am Vet Med Assoc.* 2012 Jan 15;240(2):205-12.
- 7 D'Andrea A, Riegler L, Morra S, Scarafilo R, Salerno G, Cocchia R, Golia E, Martone F, Di Salvo G, Limongelli G, Pacileo G, Bossone E, Calabrò R, Russo MG. Right ventricular morphology and function in top-level athletes: a three-dimensional echocardiographic study. *J Am Soc Echocardiogr.* 2012 Dec;25(12):1268-76.
- 8 Fraipont A, Van Erck E, Ramery E, Richard E, Denoix JM, Lekeux P, Art T. Subclinical diseases underlying poor performance in endurance horses: diagnostic methods and predictive tests. *Vet Rec.* 2011 Aug 6;169(6):154.
- 9 Helweggen MM, Young LE, Rogers K, Wood JL. Measurements of right ventricular internal dimensions and their relationships to severity of tricuspid valve regurgitation in national hunt Thoroughbreds. *Equine Vet J Suppl.* 2006 Aug;(36):171-7.

- 10 Hill DW. Energy system contributions in middle-distance running events. *J Sports Sci.* 1999 Jun;17(6):477-83.
- 11 Kriz NG, Hodgson DR, Rose RJ Changes in cardiac dimensions and indices of cardiac function during deconditioning in horses. *Am J Vet Res.* 2000 Dec;61(12):1553-60.
- 12 Kriz NG, Hodgson DR, Rose RJ. Prevalence and clinical importance of heart murmurs in racehorses *J Am Vet Med Assoc.* 2000 May 1;216(9):1441-5.
- 13 Lombard CW, Evans M, Martin L, Tehrani J. Blood pressure, electrocardiogram and echocardiogram measurements in the growing pony foal. *Equine Vet J.* 1984 Jul;16(4):342-7.
- 14 Long KJ, Bonagura JD, Darke PG. Standardised imaging technique for guided M-mode and Doppler echocardiography in the horse. *Equine Vet J.* 1992 May;24(3):226-35.
- 15 Longhurst JC, Kelly AR, Gonyea WJ, Mitchell JH. Echocardiographic left ventricular masses in distance runners and weight lifters. *J Appl Physiol Respir Environ Exerc Physiol.* 1980 Jan;48(1):154-62.
- 16 Lord PF, Croft MA. Accuracy of formulae for calculating left ventricular volumes of the equine heart. *Equine Vet J Suppl.* 1990 Jun;(9):53-6.
- 17 Martin BB Jr, Reef VB, Parente EJ, Sage AD. Causes of poor performance of horses during training, racing, or showing: 348 cases (1992-1996). *J Am Vet Med Assoc.* 2000 Feb 15;216(4):554-8.
- 18 Naylor LH, George K, O'Driscoll G, Green DJ. The athlete's heart: a contemporary appraisal of the 'Morganroth hypothesis'. *Sports Med.* 2008;38(1):69-90
- 19 Ohmura H, Hiraga A, Matsui A, Aida H, Inoue Y, Asai Y, Jones JH. Physiological responses of young Thoroughbreds during their first year of race training. *Equine Vet J Suppl.* 2002 Sep;(34):140-6.
- 20 Patteson MW, Cripps PJ. A survey of cardiac auscultatory findings in horses. *Equine Vet J.* 1993 Sep;25(5):409-15.
- 21 Reef VB. Heart murmurs in horses: determining their significance with echocardiography. *Equine Vet J Suppl.* 1995 Sep;(19):71-80.
- 22 Saltin B, Strange S. Maximal oxygen uptake: "old" and "new" arguments for a cardiovascular limitation. *Med Sci Sports Exerc.* 1992 Jan;24(1):30-7.
- 23 Sampson SN, Tucker RL, Bayly WM. Relationship between VO<sub>2</sub>max, heart



- score and echocardiographic measurements obtained at rest and immediately following maximal exercise in thoroughbred horses. *Equine Vet J Suppl.* 1999 Jul;(30):190-4.
- 24 Slater JD, Herrtage ME. Echocardiographic measurements of cardiac dimensions in normal ponies and horses. *Equine Vet J Suppl.* 1995 Sep;(19):28-32.
  - 25 Stadler P, Rewel A, Deegen E. Die M-mode-Echokardiographie bei S-Dressur-,S-Springpferden und bei untrainierten Pferden. *J. Vet. Med. A* 40, 292-306 (1993)
  - 26 Stevens KB, Marr CM, Horn JN, Pfeiffer DU, Perkins JD, Bowen IM, Allan EJ, Campbell J, Elliott J. Effect of left-sided valvular regurgitation on mortality and causes of death among a population of middle-aged and older horses. *Vet Rec.* 2009 Jan 3;164(1):6-10.
  - 27 Tyler CM, Hodgson DR, Rose RJ. Effect of a warm-up on energy supply during high intensity exercise in horses. *Equine Vet J.* 1996 Mar;28(2):117-20.
  - 28 Verdegaal LJMM, Voorhout G, van Loon G, Sloet van Oldruitenborgh-Oosterbaan MM. Herzgeräusche als zufallsbefunde bei tierärztlichen kauf- oder verfassungsuntersuchungen – Befundung und verlauf bei 77 klinisch gesunden pferden ('Accidentally-found cardiac murmurs during pre-purchase examinations: inventory and follow-up of 77 horses'). *Pferdeheilkunde* 2002; 18: 263-272
  - 29 Young LE, Marlin DJ, Deaton C, Brown-Feltner H, Roberts CA, Wood JL. Heart size estimated by echocardiography correlates with maximal oxygen uptake. *Equine Vet J Suppl.* 2002 Sep;(34):467-71.
  - 30 Young LE, Rogers K, Wood JL. Heart murmurs and valvular regurgitation in thoroughbred racehorses: epidemiology and associations with athletic performance. *J Vet Intern Med.* 2008 Mar-Apr;22(2):418-26.
  - 31 Young LE, Rogers K, Wood JL. Left ventricular size and systolic function in Thoroughbred racehorses and their relationships to race performance. *J Appl Physiol* (1985). 2005 Oct;99(4):1278-85. Epub 2005 May 26.
  - 32 Young LE, Wood JL. Effect of age and training on murmurs of atrioventricular valvular regurgitation in young Thoroughbreds. *Equine Vet J.* 2000 May;32(3):195-9.
  - 33 Young LE. Cardiac responses to training in 2-year-old Thoroughbreds: an echocardiographic study. *Equine Vet J Suppl.* 1999 Jul;(30):195-8.
  - 34 Zucca E, Ferrucci F, Croci C, Di Fabio V, Zaninelli M, Ferro E. Echocardiographic measurements of cardiac dimensions in normal

Standardbred racehorses. J Vet Cardiol. 2008 Jun;10(1):45-51.

## Appendix A: Training evaluation form

### **Name of horse**

### **Code**

### **Owner/Rider**

Use in the past

Current competition level

Years in training

Years in training by current rider

Years competing

Years competing at 1\* level

Years competing at 2\* level

Years competing at 3\* level

Years competing at 4\* level

### **Training during a regular week (without competition before or after)**

M T W T F S S

### **Outside**

>Fiel

>Paddock

### **Walker**

Walk/trot/canter

### **Lunge**

Ground

>Harnessed

>Free

Walk/trot/canter

### **Dressage**

Ground

Walk/trot/canter

Walk uninterrupted

Trot uninterrupted

Canter uninterrupted

Half pass

Pirouette

### **Show jumping**

Ground

Duration

Warming up

N fences uninterrupted

Mean height fences

maximal height fences

### **Condition training**

Ground

Warming up

Description  
Heart rate monitor

**Hacking**

Ground  
Duration  
Walk/trot/canter/gallop

**Watertraining**

Description  
Water height

**Trainingsintensiteit according to rider** \_ /5

**Level of fitness according to rider** \_ /5