McPherson, E. A., Lawson, G. H., Murphy, J. R., Nicholson, J. M., Fraser, J. A., Breeze, R. G. and Pirie, H. M. (1978) Chronic obstructive pulmonary disease (COPD): identification of affected horses. *Equine Vet. J.* **10**, 47–53.

Morris, M. J. and Lane, D. J. (1981). Tidal expiratory flow patterns in airflow obstruction. *Thorax* **36**, 135–142

Robinson, N. E. and Sorenson, P. R. (1978). Pathophysiology of airway obstruction in horses: a review. *J. Am. Vet. Med. Ass.* **172**, 299–303.

Sasse, H. H. L. (1971). *Some pulmonary function tests in horses*. Proefschrift, Utrecht.

Slauson, D. O. and Hahn, F. F. (1980). Criteria for development of animal models of diseases of the respiratory system. *Am. J. Pathol.* **101**, S103–S122.

Spörri, H. (1971). Zur Typendifferenzierun des Lungenemphysems. *First meeting of the association of veterinary medicine specialists of large domestic animals,* pp. 42–48.

Spörri, H. and Leemann, W. (1964). Zur Untersuchung der Lungenmechanik bei Großtieren. *Schweiz. Arch. Tierheilk.* **106**, 699–714

Willoughby, R. A. and McDonell, W. N. (1979). Pulmonary function testing in horses. *Vet. Clin. North America*: **1**, 171–196.

Exercise Tolerance in Standardbred Trotters with T-wave Abnormalities in the Electrocardiogram

S. G. B. PERSSON and P. FORSSBERG
Department of Medicine, Faculty of Veterinary Medicine, Swedish University of Agricultural Sciences, Box 7018, S-750 07 Uppsala, Sweden.

Summary

The T-wave configuration is very labile in the horse and varies with fluctuations in the vagal tone but the clinical significance of T-waves of abnormal polarity and amplitude is still obscure. In this study an attempt was made to differentiate pathological and physiological T-wave changes by inducing an increased vagal tone with a nose twitch and to elucidate the diagnostic significance of the prevailing abnormal T-waves by exercise tolerance testing on a high-speed treadmill.

In most horses, application of a nose twitch causes a decrease in the heart rate and an increased frequency of sinoatrial and 1st and 2nd degree atrioventricular (AV) blocks indicating a rise in the vagal tone. If this reflex vagotonia occurs, the majority of "abnormal" T-waves return to normal in parallel with the decrease in the heart rate. In 18 racehorses with a history of decreasing performance presented to the university clinic, abnormal T-waves were recorded even after applying a nose twitch. Heart rate, respiratory rate and blood lactate responses to a standard exercise test on a high-speed treadmill did not differ significantly from a reference population of apparently sound Standardbreds. The mean red cell volume (CV), however, was significantly larger in the horses with abnormal T-waves, and both the exercise and recovery heart rates were significantly higher than predicted from CV. The blood lactate accumulation during exercise was less in this group than predicted.

It was concluded that abnormal T-waves per se are not associated with decreased exercise tolerance, but may indicate a vago-regulatory imbalance related to overtraining and red cell hypervolemia.

Index terms: Blood volume; heart rate; blood lactate; treadmill exercise; horse.

Introduction

The clinical significance of T-wave abnormalities in the equine electrocardiogram (ECG) is still not known. Anomalies of the repolarization phase of the ECG have been suggested to indicate myocardial disease (Steel, 1963; Bergsten and Persson, 1966; Persson, 1969; Fregin, 1975; Holmes and Rezakhani, 1975). The T-wave is very labile in the horse, however, making the evaluation of its diagnostic significance extremely difficult. A number of both physiological and pathological factors influence the polarity and amplitude of the equine T-wave and exercise and excitation with increased heart rate (HR) rapidly give rise to T-wave changes resembling those commonly recorded in connection with infectious diseases and myocarditis (Steel, 1963; Kroneman, 1965; Bergsten and Persson, 1966; Fregin, 1975). Previous attempts at elucidating the genesis of T-wave abnormalities have generally been based on studies of a possible relationship with poor performance (Steel 1963; Persson, 1969; Fregin, 1975; Rose and Davis, 1978; Rose *et al.*, 1979, 1980; Stewart *et al.*, 1983). The purpose of this investigation was to study the exercise tolerance on a high speed treadmill of Standardbred racehorses with T-wave abnormalities and a history of fading racing performance. The exercise tolerance was assessed on the basis of heart rate (HR) and blood lactate (LA) responses to a standard work test relative to the total red cell volume (CV) as previously described (Persson, 1983).

Materials and Methods

T-wave group. Over a 5 year period (1981–1985) abnormal T-waves were diagnosed according to criteria stated below concomitantly with exercise tolerance testing on a high-speed treadmill in 18 Standardbred racehorses (2 mares, 11 stallions and 5 geldings) presented to the clinic with a history of failing racing performance for evaluation of exercise tolerance. Horses varied in age between 3 and 10 years (mean age = 5.8 years). Reexaminations were done 1–3 times in 9 of the horses at varying intervals and all were normalized electrocardiographically within the next 4 months (mean = 2 months).

Reference group. A total of 314 examinations were made for determinations of reference values for total red cell volume (CV) and exercise tolerance data. In one part (n = 192) of the reference group neither ECG examination nor exercise tolerance testing was done and these horses served as reference subjects only with respect to the red cell volume. The sex distribution of this material was 95 mares (30%), 57 geldings (18%), and 162 stallions (52%). The total number of horses was 288. Twenty-one horses were examined 2–4 times (mean = 2.3) with time intervals ranging between 4 and 40 (mean = 14) months. Three of the stallions were gelded between examinations.

The horses in this group were all Standardbred trotters considered to be normal as they showed no signs of disease likely to affect the work capacity or were performing satisfactorily on the track as judged by their trainers. The state of training varied. The majority consisted of horses which were training and racing regularly. Others were sedentary experimental animals which had with a few exceptions raced previously but had not been in training for several years. A minority of this group was clinical patients treated for minor ailments thought not to affect the exercise tolerance, e.g. dermatological diseases or horses presented for routine examinations for soundness, including exercise tolerance testing.

Electrocardiography. The ECG was recorded (Mingograph 804, Siemens-Elema, Stockholm) using the leads suggested by Brooijmanns (1957). The unipolar chest leads "facing" the left ventricular wall and the bipolar leads II and III were considered most significant for detection of T-wave abnormalities. Thus, special attention was paid to leads CV_1 (exploring electrode situated midline on the sternum 10 cm caudal to the points of the elbows), CV_2, and CV_3 (exploring electrodes in the left 6th intercostal space 1/3 and 2/3 of the distance upwards from the midline of the sternum to the level of the shoulder joint, respectively) and to leads II and III. Abnormal repolarization was considered to prevail when T-waves were entirely positive and peaked in at least three of these leads at a heart rate below 40 bpm. In an attempt to exclude horses with transiently positive T-waves in these leads due to excitement a recording was also made after application of a nose twitch. This will often bring about a decreased HR by inducing an increased vagal tone, as indicated by a concomitantly increased frequency of sinoatrial and 1st and 2nd degree AV blocks. If this reflex vagotonia occurs most of the allegedly abnormal T-waves return to normal in parallel with the decrease of the HR and spuriously pathological T-wave changes can be recognized to a considerable extent (Persson and Ullberg, unpublished observations).

Exercise-tolerance testing. The work test was performed on a high-speed treadmill (SIKOB, Stockholm) using a standardized test protocol of four sequential increments in treadmill velocity with a constant (6.25%) slope as previously described (Persson, 1983). Heart rate was monitored continuously by a bipolar ECG lead and recorded during the last 15 sec at each speed. The heart rate response to exercise was expressed as the HR at 9 m/sec (HR-9, bpm) and as the treadmill velocity corresponding to HR 200 (V_{200}, m/sec) extrapolated from the linear pulse/velocity relationship. The HR five minutes after cessation of the work test (HR5′, bpm) was used as an indicator of the recovery HR. At the same time the post-exercise respiratory rate (RR5′) was recorded.

Blood-lactate levels (LA) were determined enzymatically (Boehringer, Test combination No. 124842) in venous blood obtained before, during and after exercise with a catheter inserted and fixed in position in a jugular vein before the work test. The blood lactate response to the standardized exercise was expressed as the blood lactate at 9 m/sec (LA-9, mmol/l), immediately after exercise (LAO′) and as the treadmill velocity performed at a blood lactate of 4 mmol/l (V_{LA4}, m/sec) interpolated from the exponential LA/V relationship (Persson, 1983).

The test protocol also included determination of the total red cell volume (CV). The total blood volume was calculated from the Evans blue dye space and the hematocrit after mobilization of the splenic red cell reservoir induced by the work test (Persson, 1967). CV was derived from the difference between the total blood and plasma volumes.

Statistical analysis. Calculations were carried out by standard methods (Hald, 1952) using a computer and the SAS program. Tests of differences between groups and between observed and predicted normal values in the test group were done with Student's t-test and tests of paired data, respectively. The results are presented as mean values and standard deviations (SD) unless otherwise indicated.

Results

An example of an ECG with abnormal T-waves is shown in Fig. 1. Red cell volume and exercise test data for the reference and T-wave groups are exhibited in Table 1, which also shows "normal" exercise test values predicted from regressions of these variables on CV/BW (Table 2).

There was no difference between the groups with respect to heart rates during and after the standard exercise test as is evident from the almost identical mean values for HR-9, V_{200}, and HR5′ in the two groups. The post-exercise respiratory rate did not differ significantly between the groups, but, the lactate response to exercise was lower in the T-wave group, the mean LA-9 and LAO′-values being approximately 20% below those in the reference group. The lactate threshold (V_{LA4}) did not differ significantly

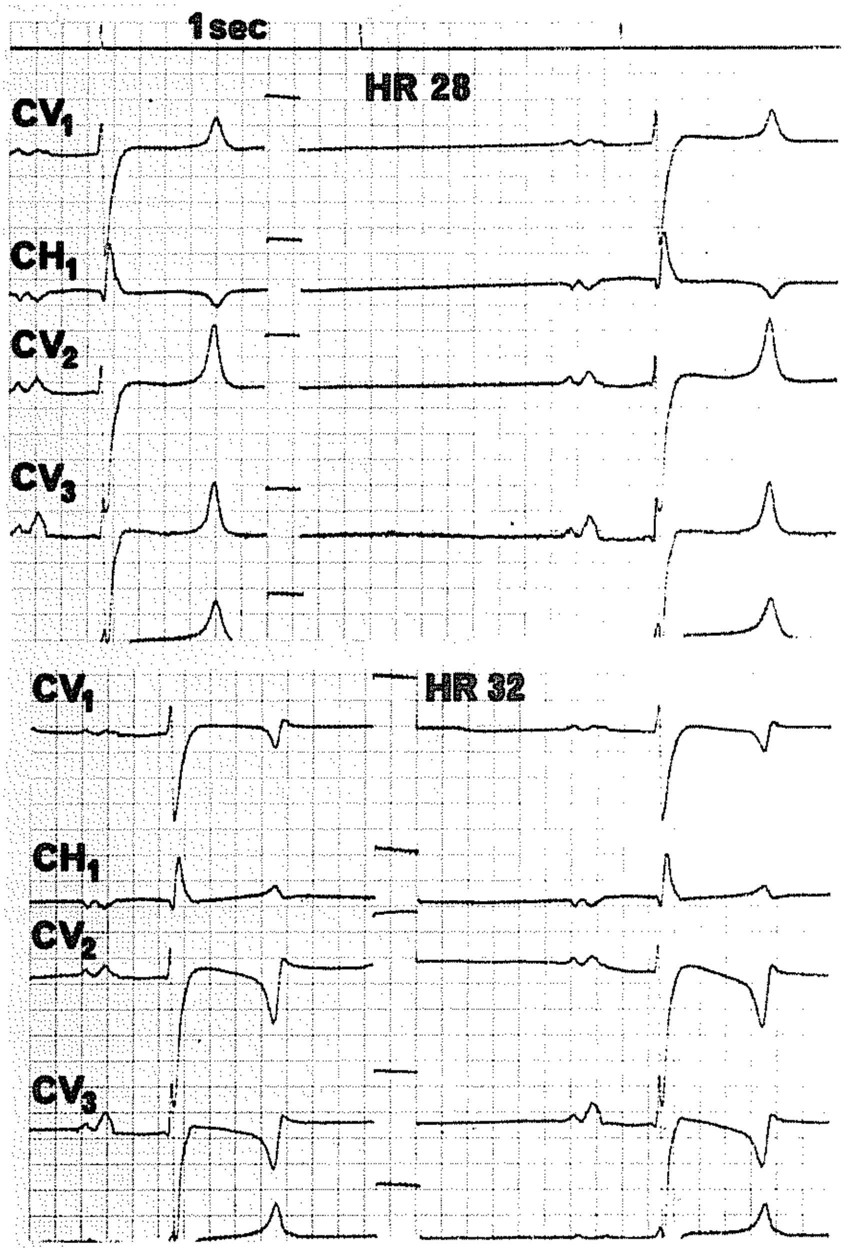

FIGURE 1 ECG with abnormal T-waves in a Standardbred trotter (above) and the same horse with normal T-waves three months later (below). Unipolar chest leads with exploring electrodes facing the apex of the heart (CV_1) and in the left 6th intercostal space facing the left ventricular wall (CV_2 and CV_3). For further details see the text. (CH_1 is a lead with the exploring electrode in the thoracic aperture, but is not discussed in this study).
HR = heart rate

TABLE 1. Mean values ± standard deviations (SD) for age, body weight (BW, kg), total red cell volume divided by body weight (CV/BW, ml/kg), heart rate at 9 m/sec (HR-9, bpm), interpolated treadmill velocity producing HR = 200 (V_{200}, m/sec), heart rate 5 minutes after the standard treadmill exercise test (HR5′, bpm), respiratory rate 5 min after the exercise test (RR5′), blood lactate concentration at 9 m/sec (LA-9, mmol/l), blood-lactate concentration immediately after cessation of the exercise test (LAO′, mmol/l), and interpolated treadmill velocity producing a blood lactate level of 4 mmol/l (V_{LA4}, m/sec) in a reference group of apparently healthy horses and in horses with abnormal T-waves, measured values and values predicted from the relationships to CV/BW (see Table 2).

	Reference Group				T-Wave Group Measured Values				T-Wave Group Predicted Values		
	Mean	SD	N	t-Value	Mean	SD	N	t-Value	Mean	SD	N
AGE	4.6	2.6	315	2.45*	5.8	1.9	18				
BW	436	36	315	n.s.	447	47	18				
CV/BW	67.3	11.8	314	5.24†	84.6	13.8	18				
HR-9	211	17	118	n.s.	211	10	18	5.80†	191	16	18
V_{200}	8.31	0.93	122	n.s.	8.35	0.66	18	6.26†	9.54	0.98	18
HR5′	84	9	95	n.s.	86	11	18	4.29†	75	8	18
RR5′	130	21	96	n.s.	136	15	18				
LA-9	5.9	2.7	96	2.75**	4.6	1.5	17	2.33*	3.5	2.0	17
LAO′	7.4	3.5	99	2.00*	6.1	2.1	18	2.89**	4.2	2.8	18
V_{LA4}	8.5	1.2	100	n.s.	9.2	1.3	17	n.s.	9.8	1.1	17

N = number of observations. t-values for differences between reference and T-wave groups and between measured and predicted values in the T-wave group with significance levels denoted as: $P < 0.05$ = *, $P < 0.01$ = ** and $P < 0.001$ = †

TABLE 2. Regressions of tilted (6.25%) treadmill exercise test parameters on total red cell volume divided by body weight (CV/BW) in apparently healthy Standardbred trotters (see the text).

	N	R	B_0	B_1	SD	SD%
HR-9	118	0.80	292	−1.19	10.0	4.7
V_{200}	122	0.87	3.49	0.0715	0.46	5.6
HR5′	95	0.64	124	−0.578	7	8.6
LA-9	96	0.56	15.8	−0.145	2.2	37.6
LAO′	99	0.64	23.0	−0.223	2.7	37.3
V_{LA4}	100	0.70	3.44	0.0756	0.87	10.2

N = number of examinations; R = correlation coefficient; B_0 = intercept; B_1 = regression coefficient; SD = standard deviation and SD% = variation coefficient. Other abbreviations as in Table 1.

between the groups. The red cell volume (CV/BW) was significantly higher (25%) in the T-wave group (Fig. 3) which also had a higher mean age.

With the exception of V_{LA4}, the recorded mean exercise-test values in the horses with abnormal T-waves differed significantly from those predicted from their respective red

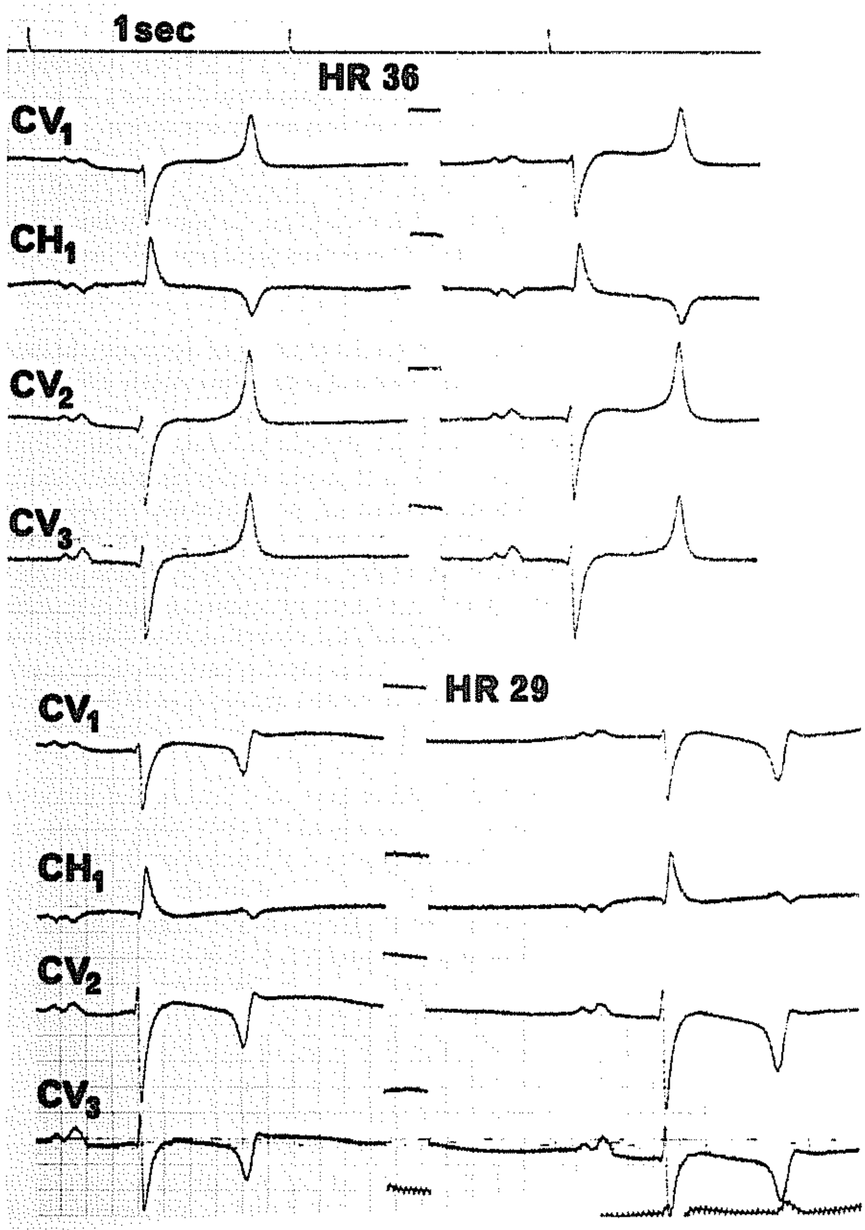

FIGURE 2 Abnormal T-waves (above) which were normalized in 8 days (below). Abbreviations as in Fig. 1.

cell volumes. Thus, the heart-rate response to exercise was increased as reflected in the higher HR-9 and HR5′ and the lower V_{200} values in this group. Although lower than in the reference group, the blood lactate accumulation, was higher than predicted from the CV/BW.

Discussion

Changes of the T-wave polarity and amplitude in the horse have been suggested to be associated with a number of both physiological and pathological factors. The T-wave configuration is particularly labile in the horse and affected by excitation, exercise and changes in limb position causing transient reversal of polarity and amplitude (Steel, 1963; Kroneman, 1965; Holmes and Rezakhani, 1975). Therefore, Steel (1963) warned against interpretation of T-wave changes at HR exceeding 42 bpm. In this study HR ranged between 26 and 42 (mean = 33.5) before and between 26 and 40 (mean = 32.4) bpm after application of a nose twitch which reduced HR in half the group by 1–7 bpm.

Although myocarditis and myocardosis are considered to occur frequently in the horse (Fregin, 1975) and apparently are reflected in T-wave abnormalities (Steel, 1963; Bergsten and Persson, 1966) it still is doubtful whether T-wave changes are always associated with significant myocardial disease. They are often thought to be due to neuro-hormonal regulatory influences even if great care is taken to avoid emotional stress and tachycardia at the ECG recording. In favor of the existence of "neurodystonic" T-wave changes is the fact that abnormal T-waves frequently are normalized very rapidly (Persson, unpublished observations). In the present study normal T-waves were found at reexamination after 8 days in one horse (Fig. 2) and restoration of repolarization anomalies has been known to occur overnight (Persson, unpublished observations). Further, various ventricular repolarization abnormalities have been reported in hard-training human athletes showing no signs of organic heart disease (Chignon and Distel, 1976; Venerando, 1979) and "silent" T-wave inversion without clinical significance appears to occur frequently in man (Ostrander, 1970).

The T-wave group did differ, however, with respect to CV/BW and blood-lactate response to exercise. The most marked difference was in CV/BW (Fig. 3) and, in fact, 1/3 of the horses in the T-wave group had values for CV exceeding 2 SD of the predicted normal value when age and sex variations were taken into account (Persson, 1967; 1969; 1983). The difference was not due to the relatively larger number of stallions in the T-wave group as the difference prevailed ($P < 0.001$) when the stallions of the two groups were compared in this respect (mean CV/BW = 71.5 and 86.8 ml/kg, respectively) and neither was it due to the higher mean age in the T-wave group as age alone does not influence CV/BW after five years of age (Persson, 1967).

A decrease of the blood lactate accumulation in relation to work performed is generally regarded as an effect of training (von Engelhardt, 1977; Gollnick and Saltin, 1982) and an inverse relationship exists with CV (Table 2). Therefore, the lower mean values for LA-9 and LAO′ in the T-wave group was to be expected considering the larger CV in this group. The reductions were not proportional to the increment of CV, however, as the values for both LA-9 and LAO′ were higher than those predicted from the CV/LA regressions (Table 2), suggestive of an incongruence in the training induced adaptation of the energy metabolism towards aerobic predominance (Åstrand and Ro-

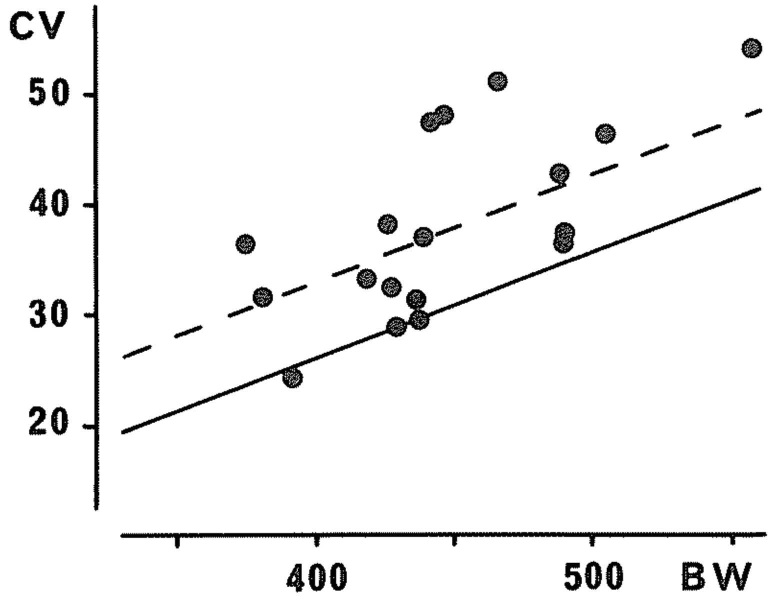

FIGURE 3 Total red cell volume (CV, liters) in relation to body weight (BW, kg) in Standardbred trotters with abnormal T-waves Normal relationship is denoted by the unbroken line +2 SD (dashed line).

dahl, 1977). This resembles the state of overtraining, which is characterized by red cell hypervolaemia and excessive HR- and LA-responses to exercise relative to CV and, probably, a dystonia of the autonomic nervous system (Persson, 1967; 1969; 1983).

In conclusion T-wave anomalies seem to have a diagnostic significance in the horse provided extreme care is taken to exclude T-wave changes of emotional origin possibly with the help of induction of increased vagotonia with nose twitching. In the majority of cases they do not seem to be caused by organic myocardial disease, however, as the HR response to exercise did not differ from that of the reference group. Abnormal T-waves may rather indicate a vago-regulatory imbalance associated with overtraining and red-cell hypervolaemia, particularly in poorly performing horses without a history of recent infectious disease.

Acknowledgment

This investigation was supported by grants from the Swedish Racing Board (ATG).

References

Åstrand, P.-O. and Rodahl, K. (1977). *Textbook of Work Physiology*. 2nd Ed. McGraw Hill, NY.

Bergsten, G. and Persson, S. G. B. (1966). Studies on the ECG in horses with acute strangles. *Proc. First Int. Conf. Equine Infect. Dis.*, Stresa, Italy.

Brooijmans, A. W. M. (1957). Electrocardiography in horses and cattle. Ph.D. Thesis, State University of Utrecht.

Chignon, J. C. and Distel, R. (1976). Vector cardiographic aspects of the abnormalities of the ventricular repolarisation in high competition athletes. *Adv Cardiol.* **16**, 55–56.

von Engelhardt, W (1977). Cardiovascular effects of exercise and training in horses. *Adv. Vet. Sci.* **21**, 173–205

Fregin, G. F. (1975). Acquired cardiovascular diseases affecting exercise performance—diagnosis, therapy and prognosis. *J. S. Afr. Vet. Ass.* **45**, 269–271.

Gollnick, P. D. and Saltin, B. (1982). Significance of skeletal muscle oxidative enzyme enhancement with endurance training. *Clin. Physiol.* **2**, 1–12.

Hald, A. (1952). *Statistical theory with engineering applications.* Wiley & Sons Inc., NY.

Holmes, J. R. and Rezakhani, A. (1975). Observations on the T wave of the equine electrocardiogram. *Equine Vet. J.* **7**, 55–62.

Kroneman, J. (1965). The electrocardiogram of the trained horse. Thesis, State University of Utrecht.

Ostrander, L. D. (1970). The relation of "silent" T wave inversion to cardiovascular disease in an epidemiologic study. *Am. J. Cardiol.* **25**, 325–328.

Persson, S. G. B. (1967). On blood volume and working capacity in horses. *Acta Vet. Scand.*, Suppl. **19**, 1–189.

Persson, S. G. B. (1969). Blood volume, state of training and working capacity of race horses. *Equine Vet. J.* **1**, 1–11.

Persson, S. G. B. (1983). Evaluation of exercise tolerance and fitness in the performance horse. *In: Equine Exercise Physiology.* Snow, D. H., Persson, S. G. B. and Rose, R. J. (eds.) Granta Editions, Cambridge pp. 441–457.

Rose, R. J. and Davis, P. E. (1978). The use of electrocardiography in the diagnosis of poor racing performance in the horse. *Aust. Vet. J.* **54**, 51–56.

Rose, R. J., Ilkiw, J. E. and Hodgson, D. R. (1979). Electrocardiography, heart score and haematology of horses competing in an endurance ride. *Aust. Vet. J.* **55**, 247–250.

Rose, R. J., Backhouse, J. W. and Ilkiw, J. E. (1980) Electrocardiography and haematology of horses competing in a three-day-event. *Aust. Vet. J.* **56**, 318–320.

Steel, J. D. (1963). *Studies on the electrocardiogram of the racehorse.* Australasian Med. Publishing Co. Ltd., Sydney.

Stewart, J. H., Rose, R. J., Davis, P. E. and Hoffman, K. (1983). A comparison of electrocardiographic findings in racehorses presented either for routine examination or poor racing performance. *In: Equine Exercise Physiology.* Snow, D. H., Persson, S. G. B. and Rose, R. J. (eds.) Granta Editions, Cambridge, pp. 135–143.

Venerando, A. (1979). Electrocardiography in sports medicine. *J. Sports Med. Phys. Fitness* **19**, 107–128.