

# A Comparison of Electrocardiographic Findings in Racehorses Presented either for Routine Examination or Poor Racing Performance

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## Summary

*Electrocardiograms (ECGs) were recorded and full blood counts and a range of plasma biochemical measurements were performed in 131 horses presented with a history of poor racing performance. A comparison group of 129 horses (routine group) presented for musculoskeletal problems or heart score determination also had ECGs recorded. The incidence of various ECG abnormalities in the two groups of horses was compared by  $\chi^2$  analysis, while the age of the horses, P wave duration, P–R interval, mean QRS interval (leads I, II and III), Q–T interval and electrical axis were compared in the two groups using an unpaired Student's *t* test.*

*The haematological and plasma biochemical values for horses in the poor performance group were all within the normal ranges for our laboratory. The poor performance group of horses had significantly higher incidences of T wave abnormalities in three or more leads and second-degree atrioventricular block than horses in the routine group. Other abnormalities in the poor performance group included intra-atrial block, intra-ventricular block and atrial fibrillation. These ECG abnormalities were not found in the routine group. There were no significant differences in the age of the horses nor in any of the various ECG intervals, when the two groups of horses were compared.*

## Introduction

Retrospective diagnosis of the cause of a racehorse's poor performance is always difficult. In addition to the usual problems involving physiological, biochemical or musculoskeletal derangements, factors such as the state of the track, ability of the jockey and placement in the field can affect the outcome of a race. In Australasia, much emphasis has been placed on haematology and plasma biochemistry (Steel and Whitlock, 1960; Sykes 1966; Stewart *et al.*, 1970; Stewart and Steel, 1975; Williamson 1975; Stewart *et al.*, 1977) in assessing fitness, and clinical significance has been attached to small variations in an individual horse's haematology (Marbach 1978). While haematological and plasma biochemical disturbances can affect a horse's performance, it is true that a single blood sample collected from a racehorse will usually reveal values lying within the normal ranges.

Racing performance has also been reported to be affected by abnormalities of the electrocardiogram (ECG) (Steel 1963; Fregin 1975; Rose and Davis, 1978). This study was undertaken to examine whether poorly performing racehorses had higher incidences of ECG abnormalities than a group of racehorses presented for routine examination. Where possible, horses presenting with ECG abnormalities were re-examined after various periods of rest.

### *Materials and Methods*

A total of 260 racehorses (Thoroughbreds and Standardbreds) that had electrocardiograms recorded between February 1977 and May 1982 were selected for inclusion in this study. The total comprised 163 Thoroughbreds and 97 Standardbreds of which there were 107 geldings, 69 stallions and 84 mares. The ECGs were recorded with a Tritex electrocardiograph (DLC Trading, Melbourne) using the leads I, II, III, aVR, aVL, aVF, CV.LA, CR.LA, CF.LA and CL.LA. The left apex (LA) position of Steel (1963) was used, with the exploring electrode located approximately 5 cm caudal to the point of the elbow. The ECGs were assessed using the criteria of Steel (1963). Abnormal T waves were classified as those which were positive or diphasic in lead I, negative in aVR and positive in leads CV.LA, CR.LA, CF.LA and CL.LA. Horses with T wave abnormalities in three or more leads were compared to those with abnormalities in less than three leads. Depending on the reasons for presentation, the horses were placed into two groups:

1. *Routine Group* (129 horses): These were horses presented for musculoskeletal problems or for routine 'heart score' determination (Steel 1963).
2. *Poor Performance Group* (131 horses): Because of the difficulty in defining poor racing performance, horses selected for inclusion were those with a history of decreased racing performance compared with previous performance, as judged by the trainer. The most common history was of a horse that worked well in trackwork, raced well in the initial stages of a race but faded out of the race over the final 200 to 400 metres. Most horses showed no other signs of decreased exercise tolerance.

In this group of horses, a full blood count and plasma biochemistry (aspartate aminotransferase, creatine kinase, alkaline phosphatase, gammaglutamyl transferase, sodium, potassium and chloride) were also performed.

A chi-squared test was performed to compare the incidence of AV block, T wave changes in three or more leads in the routine and poorly performing groups of horses. An unpaired Student's t test was used to compare the ages and various ECG intervals between the two groups of horses.

### *Results*

The incidence of the various ECG abnormalities for the two groups of horses is presented in Table 1.

An example of an ECG with abnormal T waves is shown in Fig. 1. Some horses had more than one abnormality, so that the total figures do not represent total horse numbers. The results of the chi-squared tests comparing the incidence of abnormalities in the routine and poorly performing groups showed  $\chi^2$  values of 65.72 ( $p < 0.001$ ) for the comparison of T wave abnormalities in three or more leads, 2.18 (not significant) for the

TABLE 1. Incidence of ECG abnormalities in racehorses presented for routine examination or poor racing performance.

ECG findings	Routine group		Poor performance group	
	n	%	n	%
Normal	102	78.5	22***	16.0
T wave abnormalities in three or more leads	10	7.7	66***	48.2
T wave abnormalities in less than three leads	9	6.9	15	11.0
1st degree AV block	1	0.8	3	2.2
2nd degree AV block	7	5.4	16*	11.7
Intra-atrial block	—	—	6	4.4
Sinoatrial block	1	0.8	3	2.2
Intra-ventricular block	—	—	3	2.2
Atrial fibrillation	—	—	3	2.2
Total	130	100	137	100

\*  $p < 0.05$  } Significant differences from routine group.  
 \*\*\*  $p < 0.001$  }

comparison of T wave abnormalities in less than three leads, 4.64 ( $p < 0.05$ ) for comparison of the incidence of AV block, and 104.43 ( $p < 0.001$ ) for the comparison of normal ECGs between the routine and poor performance groups. All the results of haematology and plasma biochemistry for horses in the poor performance group fell within the normal ranges for our laboratory.

The data for the age of horses, P-wave duration, P-R interval, means QRS interval (leads I, II and III), Q-T interval and electrical axis for the two groups of horses are presented in Table 2. There were no significant differences between the groups for any of the measurements.

The percentage resolution of T wave abnormalities in the small group of horses where this information was available is presented in Table 3. After varying periods of rest ranging from less than three months to greater than 12 months, resolution occurred in only nine of the 26 horses. In four of these nine horses, T wave abnormalities recurred after 6 to 12 weeks of training.

### Discussion

A wide range of factors can result in the poor racing performance syndrome, with viral respiratory disease being the most obvious (Mumford and Rosedale, 1980). However, in cases where there are no apparent causes for poor racing performance, cardiorespiratory abnormalities may play a rôle (Persson 1969; Rose and Davis, 1977). In this study, the typical presenting history of the poor racing performance group of horses was a failure to run on over the last 200 to 400 metres of their races. Where ECG abnormalities were present, this history suggested a decrease in cardiac output at a time when additional cardiac reserve was required. An explanation for this decreased cardiac output may be

FIGURE 1. Abnormal T waves in L<sub>2</sub>, L<sub>3</sub> (positive and peaked), aVR (negative), aVF, CV, CR, CF and CL LA (positive and peaked) in an ECG from a horse in the poor performance group.

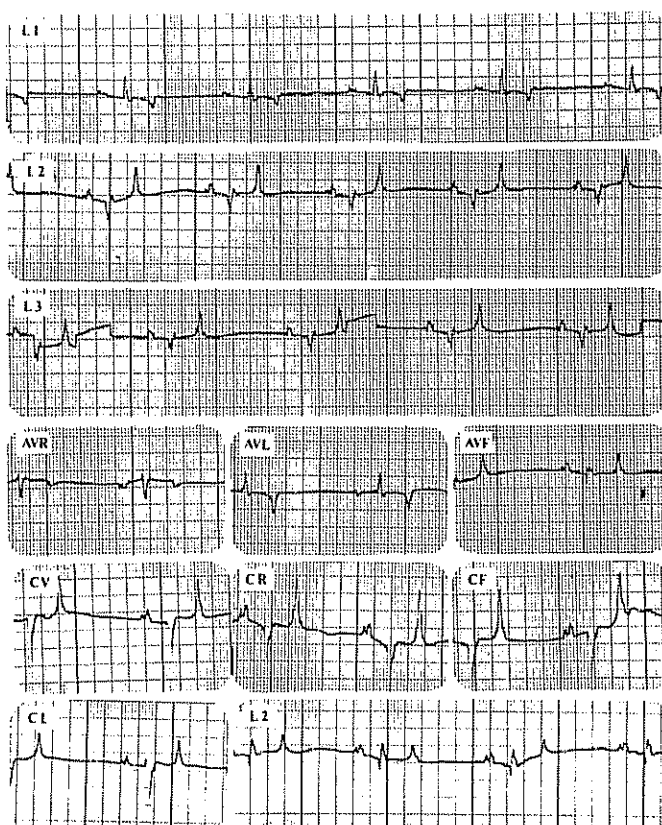


TABLE 2. ECG data (mean  $\pm$  SD) from racehorses presented for routine examination or poor racing performance.

Value	Routine group	Poor performance group
Age (years)	3.0 $\pm$ 1.5	3.6 $\pm$ 1.2
P wave duration (s)	0.14 $\pm$ 0.02	0.14 $\pm$ 0.02
P-R interval (s)	0.29 $\pm$ 0.05	0.30 $\pm$ 0.05
Mean QRS duration (s) (leads I, II and III)	0.113 $\pm$ 0.010	0.116 $\pm$ 0.011
Q-T interval	0.502 $\pm$ 0.038	0.508 $\pm$ 0.030
Electrical axis (degrees)	+20.6 $\pm$ 38.8	+22.8 $\pm$ 45.5
Number of horses	129	131

TABLE 3. Resolution of T wave abnormalities in 26 horses re-examined after various periods of rest.

Rest period	Number of cases resolved	Percentage resolution
< 3 months	3	11.5
3-6 months	4	15.4
6-9 months	1	3.8
9-12 months	—	
> 12 months	1	3.8
Total	9	34.5

found in the work of Hall *et al.* (1976), where horses with abnormal ECGs had higher heart rates during exercise at speeds of 400–800m/min than horses with normal ECGs. Preliminary data from telemetry ECGs performed on horses during a standardized treadmill exercise test confirm these findings in horses with T wave abnormalities and also suggest a slower heart rate recovery in these horses (Rose, Stewart, Hodgson and Allen, unpublished data). During exercise, horses with abnormal resting ECGs may thus have a shorter time for ventricular filling and hence a reduced stroke volume and cardiac output.

The significantly higher incidence of T wave abnormalities in three or more leads in the poor racing performance group, when compared to the routine group, indicates impaired cardiac function and may be one cause of the poor racing performance syndrome. Although some horses in the routine group also had T wave abnormalities, where these horses could be followed up, many raced poorly showing the typical signs of fading over the final 200–400 metres of a race.

Persson (1969) reported decreased aerobic capacity in horses with repolarization disturbances and suggested a decreased myocardial contractility as the probable cause in some cases. The occurrence of T waves of reversed polarity and increased amplitude has been described by many workers (Landgren and Rutqvist, 1953; Brooijmanns 1957; Steel 1963; Persson 1969; Fregin 1975; Rose and Davis, 1978; Rose *et al.*, 1979, 1980), while changes in T vector distribution have also been reported (Holmes and Alps, 1967; Holmes and Rezakhani, 1975). These T wave changes have been associated with a host of systemic physiological and pathological conditions including exertion, excitement, drugs, infectious diseases, vagal tone, serum electrolyte imbalance, hypoxia and myocarditis. As the T wave is highly labile and may be influenced by a great number of different factors, quantification of its diagnostic significance is extremely difficult. To eliminate some of the factors known to affect the T wave, all ECGs in this study were recorded in resting horses under standard quiet conditions. The horses then classified as having T wave abnormalities likely to affect racing performance were those in which reversal of T wave polarity and amplitude were apparent in a number of ECG leads, but which had no other clinically detectable abnormalities. A number of other workers have suggested that this type of T wave abnormality adversely affects a horse's performance (Steel 1963; Persson 1969; Fregin 1975; Rose and Davis, 1978; Rose *et al.*, 1979, 1980). This study confirms the significance of T wave abnormalities and demonstrates that a large percentage of

horses have persistence of these abnormalities even with prolonged rest periods. In addition, where resolution of the T wave changes did occur with rest, the abnormalities returned upon resumption of training. Training and stress appear to play a rôle in the aetiology of T wave abnormalities, as certain trainers produce these changes in a high percentage of their horses.

In man, repolarization changes have also been reported and have been attributed to an even more diverse range of aetiologies (Ostrander 1970; Hartung 1972; Venerando 1979). Chignon and Distel (1976) reported a change in the T vector loop in elite athletes, reflected clinically as a feeling of tiredness and frequent poor performances.

Despite the clinical evidence indicating an association between T wave abnormalities and poor racing performance, acceptance of the physiological significance of T wave changes has been limited by the paucity of information on the genesis of the T wave. Since the T wave in the normal heart does not have an equal area and opposite polarity to the QRS complex, the sequences of activation and repolarization clearly differ (van Dam and Durrer, 1964). In examining the repolarization process, determining whether it is a propagated phenomenon with an exact time course is relevant (Schaefer 1957; van Dam and Durrer, 1964). It would seem probable that the anodal effect of repolarized tissue in recruiting recovery in adjacent muscle is important in explaining the spread of repolarization and hence the shape and direction of the T wave. In contrast, repolarization is also known to be a spontaneously occurring process in which each cell is potentially its own pacemaker (Crane field 1957). The resolution of arguments surrounding the significance of T wave abnormalities would appear to be extremely difficult, as information on T wave genesis can only be gained by direct electrode placement in the myocardium of living horses, a technique which itself produces repolarization disturbances (Kossmann 1957).

Of the other abnormalities found on ECG examination, only the incidence of T wave abnormalities in less than three leads and the incidence of second-degree atrioventricular (AV) block could be statistically compared in the two groups of horses. While there was no significant difference in the incidence of T wave abnormalities in less than three leads, the poor racing performance group had a significantly higher incidence of second-degree AV block than the routine group.

Whether second-degree AV block has a physiological or pathological basis is still unresolved. Holmes (1975) described it as the most common arrhythmia and doubted its clinical significance. In contrast, Steel (1963) regarded second-degree AV block as a condition likely to impair racing performance and reported an incidence of 8% in racehorses. The results of this study do not clarify the issue. Although a significant difference in incidence of second-degree AV block was found between the two groups of horses six of the poorly performing group had concurrent T wave abnormalities. When these horses were eliminated from the statistical analysis, there were no significant differences between the groups.

Most reports agree that vagal tone is involved in the production of second-degree AV block, as the condition can be abolished by atropine or exercise (Smetzer *et al.*, 1969a, 1969b). However, significant pathological changes have been found in the myocardium of horses with second-degree AV block (Marcus and Ross, 1967; Kiryu and Kaneko, 1977; Kiryo *et al.*, 1979). In two thorough histopathological examinations of 1274 sections from the hearts of ten horses with AV block, Kiryu and Kaneko (1977) and Kiryu *et al.* (1979) reported a uniformity of lesions, characterized by focal myocardial fibrosis with

neurovascular alterations in the right atrium, interatrial septum, the AV junction, perinodal region and cardiac nerves. They concluded that the pathology may have been due to the effects of severe exertion or systemic disease. It should, however, be noted that histological findings in the myocardium of horses with normal ECGs have not been reported, and the lesions reported by Kiryu *et al.* (1979) may be of limited significance.

Gradation in the severity of second-degree AV block could also be important. A single dropped beat may be of little consequence, whereas a regular block every two or three beats may be of greater clinical significance. Brown and Holmes (1978) found that peak aortic systolic pressures showed a progressive increase in horses with second-degree AV block until conduction is blocked, causing the pressure to fall again. However, in one case where this mechanism of blood pressure control was malfunctioning, the block occurred at lower aortic pressures. This could be one possible explanation for the difference in horses whose performance is seemingly affected or unaffected by the block.

To resolve some of the conflicting evidence regarding the functional significance of second-degree AV block, a careful pathological study comparing horses with normal ECGs and horses with AV block is required. In addition, the technique of cardiac pacing described by O'Callaghan (1977) may be useful in investigating AV block. In this technique, overdrive pacing is used to increase the heart rate well in excess of the basic natural rhythm. This induces autonomically mediated inhibitory responses which may reveal borderline conduction and rhythm irregularities.

Although there was no significant difference in the incidence of T wave abnormalities in less than three leads between the two groups of horses, it is our experience that this type of T wave change may herald a more serious ECG abnormality. A number of horses with one or two abnormal T waves (usually CR.LA and CL.LA) had developed T wave changes in more than three leads or second-degree AV block when subsequently examined (Rose and Davis, unpublished data).

The incidence of other abnormalities such as intra-atrial block, sinoatrial block, intra-ventricular block and atrial fibrillation was more difficult to assess objectively. Certainly, intra-atrial block, intra-ventricular block and atrial fibrillation only occurred in the poor performance group of horses. These abnormalities are usually associated with a severe reduction in performance, with follow-up examinations indicating no resolution with rest. Atrial fibrillation may, however, be responsive to quinidine sulphate, and successful racing performance has been reported following this therapy (Rose and Davis, 1977). The effect of sinoatrial block on performance is more equivocal, and in two horses that were re-examined the block disappeared spontaneously.

While the ECG is a useful diagnostic aid in assessing the racehorse with poor racing performance, it is no substitute for complete clinical evaluation. However, this study demonstrates that poorly performing racehorses do have a significantly higher incidence of ECG abnormalities than those presented for routine examination. In addition, we have confirmed that abnormalities of the T wave are the most common ECG finding in horses presented for poor racing performance.

### **Acknowledgement**

The authors gratefully acknowledge the financial assistance of the Australian Equine Research Foundation and Les Edwards and Son Pty Ltd.

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# The Effects of Vertical Force on Blood Flow in the Palmar Digital Arteries of the Horse

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## Summary

*In an investigation of haemodynamics in the equine digit, blood flow and anatomical structure of the palmar digital arteries were examined in 14 live and 23 dead horses. Gross anatomy was depicted by vinylite casting, microstructure by histology and blood flow by angiography. Angiograms of digits from both live and dead subjects were made without and with the application of vertical force to the limb.*

*The angiograms made during the application of vertical force showed termination of blood flow at two sites in the palmar digital arteries, 1. adjacent to the navicular bone and 2. in the solear canal. The site of closure adjacent to the navicular bone coincided with an intra-arterial sphincter and caused an increase in blood supply to the navicular bone via the proximal source as well as a redirection of blood flow to the coronary regions of the foot. Post-mortem studies showed that large vertical forces override the operation of the sphincter system and mechanically interrupt blood flow to the distal regions of the digit. Excessive forces prevent blood supply to the digit and navicular bone altogether. Within the solear canal, termination appeared to be directly dependent on retrograde blood flow, but coincided with either the sharp bend in the terminal arch or two arterial dilations which were separated by an actual narrowing of the arterial lumen.*

*It was concluded that closing mechanisms are present in the palmar digital arteries of most normal horses of Arabian ancestry and that these apparently prevent retrograde blood flow in the arteries of the digit should pressure therein rise during the support phase.*

## Introduction

Various authors (Adams 1972; Ackerman *et al.*, 1975) have suggested angiographic techniques to describe vascular alterations in the equine digit. These techniques necessitated general anaesthesia with the horse in lateral recumbency which could affect the *in vivo* situation. While anaesthesia may interfere with normal physiological vascular