

# Cardiac Arrhythmias on the Racecourse

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## Summary

*Nineteen horses, presented with arrhythmia after racing, were examined by electrocardiography at the racecourse. Thirteen horses had atrial fibrillation, of which 11 spontaneously reverted to sinus rhythm, the majority within 24 hours, and one responded to treatment with quinidine sulphate. Ten of these horses suddenly slowed during the race. Subsequent observations have shown no evidence of recurrence of this arrhythmia and subsequent racing performance has been satisfactory.*

*Premature beats of atrial origin were recorded in four horses, three of which were examined because of disappointing racing performance. In two horses the arrhythmia was ventricular in origin. These arrhythmias were generally transient. The occurrence of premature beats appeared to be exaggerated during heart rate slowing after work. During a race, when heart rate is high, rhythm may be regular.*

*Index terms: Atrial fibrillation; premature beats.*

## Introduction

Although there are many causes of poor racing performance, human analogy causes the heart and circulation to be primary suspect systems when a horse unexpectedly fades during a race. In terms of the total number of racing horses, however, the incidence of circulatory problems is quite low. Nevertheless, when fading occurs during a race it is usual to pay particular attention to the circulation in attempting to explain the cause.

On British racecourses the stewards select horses for drug testing, including any in which performance may give rise to suspicion of the improper administration of medicants. Veterinary officers examine the heart by auscultation noting heart rate and particularly rhythm. If arrhythmias are found, owners are advised to consult their local veterinarians on return to their home stables. Examination at home often reveals a horse with regular rhythm both at rest and after work. The diagnosis of this transient cardiac arrhythmia (T.C.A.) therefore remains a matter of speculation.

In Japan, Amada and Kurita (1975) reported five cases of paroxysmal atrial fibrillation in racehorses. Because the heart spontaneously reverted to sinus rhythm, atrial fibrillation was suspected as the likely cause of T.C.A. Atrial fibrillation is seen most commonly in large horses. The chaotic rhythm, which is the usual clinical feature, is present at all heart rates. These features make a diagnosis by auscultation highly accurate, but for certainty, particularly when the arrhythmia is transient, it is desirable to obtain an electrocardiogram (ECG) on the racecourse soon after the end of the race.

### Materials and Methods

For examination of horses immediately after racing, an Instromedix Cardiodyary (Instromedix Inc., Beaverton, Oregon, USA) comprising a microprocessor which records up to three 32-second periods, sampling the ECG waveform every 4 msec proved most convenient. The lead used was a bipole Lead Y (Holmes, 1984) (xiphoid + to front of chest -). The ECG records were then transmitted by telephone to a central CARDIOTEL receiver and transcribed at a paper speed of 25 mm/sec and a sensitivity of 1 mV = 1 cm.

### Results

Nineteen horses with arrhythmia after racing were examined and diagnosed during the period from July 1, 1984 to March 31, 1986, as shown in Table 1. As no clinical observations were made on individual horses before the race, there is no proof that they were in sinus rhythm but certainly none of them gave cause for concern before the race began.

*Atrial fibrillation.* Thirteen horses had atrial fibrillation. They ranged in age from 2 to 11 years (average 5.6 years). Table 2 shows the type of race following which atrial fibrillation was diagnosed and the signs shown. Nine of the horses suddenly and unexpectedly faded during the race. Sinus rhythm returned within 24 hours in eight of the thirteen horses. Two horses were not examined until two days and nine days, respectively, after racing and at that time both were in sinus rhythm. One horse was still fibrillating after 45 hours and was successfully treated orally with quinidine sulfate. One horse, still fibrillating 48 hours after the race, was not treated but was in sinus rhythm on day 13. Another horse was still arrhythmic seven days after racing, was treated with quinidine sulphate and did not respond. Further inquiries into the history of this animal indicated previous poor performances and it may have been fibrillating for considerable time representing the established form of this arrhythmia. The horse which did not respond to quinidine therapy was withdrawn from further racing. No information was available for two other horses but the remaining ten have all returned to racing. One was given 23 weeks rest but the others were all back on the racecourse in from three to 93 days (average, 38 days). There was no interruption in their racing schedule and some have performed extremely well. Up to the end of December 1985 one horse had run 18 times with no recurrence of arrhythmia. So far as is known, none of them have subsequently redeveloped atrial fibrillation.

*Premature atrial systoles.* Four horses were presented with supraventricular arrhythmias (excluding atrial fibrillation). A 2-year-old filly, which finished fourth in the race, showed occasional premature atrial beats followed by compensatory pauses when examined 25 min after racing. Heart rate then was 80/min. A 3-year-old gelding had a rate of 126/min and regular rhythm 5 min after a 2.5-mile hurdle race. One hour after

TABLE 1 Types of arrhythmia encountered after racing

Atrial fibrillation	13
Other supraventricular arrhythmias	4
Ventricular arrhythmias	2

TABLE 2 Atrial fibrillation, conditions of occurrence.

Signs	Type of race		
	Flat	Hurdles	Steeplechase
No signs, raced well and won	1	1	1
Unexpectedly weakened/ suddenly faded/pulled up	5	3	1
Very distressed/incoordinate after race	1	0	0

the race, rhythm was irregular and this arrhythmia, due to premature atrial systoles, lasted not more than 45 min. A 3-year-old filly had shown some post race arrhythmia after previous races but no records had been taken. Following a disappointing performance, premature atrial beats were recorded at a heart rate of 120/min 15 min after racing. There was a rhythmicity about their occurrence suggestive of atrial parasystole. The arrhythmia was transient and rhythm was regular 30 min after racing as heart rate slowed. A 2-year-old filly suddenly faded on the racecourse during a 1 mile flat race and came in last. One hour after the race, heart rate was 165/min and irregular. The ECG pattern suggested sinus arrhythmia and there was considerable variation in S wave amplitudes. The following day rhythm was regular, rate 37/min. After light exercise, one premature atrial beat was recorded at a rate of 48/min.

*Premature ventricular systoles.* Two horses had arrhythmia due to premature ventricular beats. In one horse which ran second in the race, premature systoles occurred occasionally at 30 and 45 min after racing and were followed by full compensatory pauses. In the other horse, which finished third in the race, there was ventricular tachycardia at a rate of 95/min 15 min after the end of the race. At 85 min, heart rate was 60/min and in sinus rhythm with occasional premature ventricular beats followed by full compensatory pauses. This horse raced again 134 days later. Examined before the race, one premature beat was recorded over 3 minutes. In sinus rhythm at a rate of 91/min at 25 min after the race, premature ventricular beats then appeared; singly, occasionally in pairs or threes but there were no long paroxysms of ectopic beats due to exit block from the ectopic focus. There was rhythmicity about the ectopic beats which represented transient ventricular parasystole. As heart rate slowed the "faster" sinus rhythm assumed control by getting ahead of the activation from the ectopic focus. This horse has run at least 12 further races up to the end of October, 1985 winning one and finishing second in two races and fourth in two races.

### Discussion

My clinical findings support the observations of Amada and Kurita (1975) that some horses may suddenly develop atrial fibrillation during a race resulting in unexpected, sudden loss of performance. This sudden onset of signs may also be observed in the hunting field or during other energetic activities such as team racing or eventing. It is probably due to the sudden onset of myaesthesia from circulatory impairment due to the effect of the arrhythmia on blood flow to the very active skeletal muscles (Holmes

*et al.*, 1986). No particular type of race seems to preclude the occurrence of atrial fibrillation. This arrhythmia may spontaneously revert to sinus rhythm within 24 hours. In one horse sinus rhythm had returned at 14 hours but the horses were not continuously monitored and the arrhythmia may have lasted for a much shorter time in some. This type of fibrillation may justifiably be called *paroxysmal*.

Not all horses fibrillating after a race show signs during the race and sometimes such horses may win (3/13 in this series, 23%). Horses which win, despite fibrillation when examined after a race, may have developed the arrhythmia at the end of the race or may have been fibrillating for some time before the race such that the circulation has adapted to the arrhythmia. The latter are probably unlikely to spontaneously revert and some may not respond to quinidine therapy.

As to how long a case may fibrillate, and still spontaneously revert to sinus rhythm, or after what length of time treatment should be commenced, remains to be determined. There may be considerable individual variation between horses. In one horse treatment was initiated after 45 hours of arrhythmia and the horse responded well. Another horse had been fibrillating for 48 hours. The owner refused to have the horse treated and when examined 11 days later the heart was under sinus control and this horse raced again 21 days after the previous race and rhythm remained regular. Therefore although the majority of horses with *paroxysmal* atrial fibrillation will revert to sinus rhythm within 24 hours, some horses may take longer. However, it would probably be unwise to assume that spontaneous recovery would occur in many horses after 48 hours and if arrhythmia was then present it would seem sensible to treat with quinidine.

There seems to be no predisposition to recurrence of this arrhythmia. Atrial fibrillation may have a number of causes. The occurrence of a self-correcting *paroxysmal* form provides an interesting facet on the nature of this arrhythmia. From the subsequent racing records of some of these horses, it seems unlikely that the irregularity is always accompanied by serious atrial pathology. In large horses with large atria and at rapid heart rates inhomogeneity of repolarization, with subsequent inhomogeneous excitability of the myocardium, may favor multiple re-entry. The breakup of the depolarization wave front creates circus movements which temporarily usurp the role of the pacemaker.

Although most cases of arrhythmia after racing may prove to be *paroxysmal* atrial fibrillation, it is necessary to consider other causes of arrhythmia in differential diagnosis. Many of the non-fibrillating post exercise arrhythmias appeared to be transient or to be exaggerated during the heart rate slowing following work. Atrial or ventricular parasystole may manifest in a transient form and be replaced during slow resting heart rates by occasional premature atrial or ventricular beats, respectively. Supraventricular arrhythmias, such as transient sinus arrhythmia and premature beats, may coexist and complicate the rhythm. Transient premature atrial beats with an apparent rhythmicity may perhaps have a relationship to respiration.

It is likely that most of the premature beats, of either atrial or ventricular origin, are a feature of the slowing heart after work rather than being present during work. The opportunity for premature beats to insert themselves in the normal rhythm is much less when the duration of the myocardial responsive period is shortened during severe tachycardia.

Although it is important to get an ECG record soon after the end of a race it is perhaps even more valuable to obtain an ECG when heart rate has slowed sufficiently for P or

f waves to be obvious. These waves frequently provide the key to diagnosis. This may require waiting for 30 to 45 minutes after racing. A further ECG examination 24–48 hours later is also valuable.

Although this survey disclosed 19 horses with post race arrhythmia, this may not represent the true incidence as only certain horses were selected for examination. The real incidence may be much higher

### **Acknowledgments**

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# Exertional Rhabdomyolysis Related to Malignant Hyperthermia Using The Halothane-Caffeine Contracture Test

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## Summary

*Five horses with exertional rhabdomyolysis were studied. Surgical biopsy was performed on the cutaneous omobranchialis muscle. The biopsy sample was then subjected to halothane-caffeine contracture testing and histopathological and histochemical analyses. Blood samples were obtained for serum biochemical analysis and selenium measurement. All horses had elevation of serum enzymes associated with myolysis. Three horses had contracture patterns that were abnormal, and two of those were similar to contracture patterns found in human and porcine malignant hyperthermia susceptible muscle. Histopathological and histochemical analyses indicated that the cutaneous omobranchialis muscle consists predominantly of type II fibers, but no abnormalities were noted.*

*Index terms: Horse; ATP-ase; fiber types; selenium.*

## Introduction

Malignant hyperthermia (MH) is an inherited myopathy that has been extensively studied in humans and swine as stress syndrome, anesthetic-induced MH, and pale soft exudative pork syndrome (Gronert, 1980; D'Allaire and Deroth, 1982; Seeler, *et al.*, 1983). MH has been described as a pharmacogenetic disease, since it is manifested on exposure to an initiating or "trigger" situation. Triggers of MH include stress, excitement or anxiety, exercise, injury, high environmental temperature, and exposure to volatile anesthetic agents and depolarizing muscle relaxants (Britt, 1976). The MH syndrome is caused by a generalized plasmalemmal defect, with sarcolemmal changes responsible for most of the signs noted. In response to a trigger situation, the sarcoplasmic reticulum of MH muscle releases large amounts of calcium into the sarcoplasm, causing pronounced and prolonged contracture (Denborough, 1979). This hypermetabolic state leads to hyperthermia, metabolic and respiratory acidosis, and cardiovascular instability. Myolysis leads to myoglobinuria which can result in renal failure (Ryan, 1979).

MH has been noted in other species. It has been described in racing greyhounds (Bagshaw *et al.*, 1978), and recently a colony of MH susceptible dogs has been established at the University of Saskatchewan in Canada (O'Brien *et al.*, 1983). For some time MH has been suspect as a factor in equine anesthetic and exertional myopathy (Rosenberg and Waldron-Mease, 1977; Klein, 1978). There are similarities in clinical, therapeutic, and laboratory findings. Clinically, MH individuals tend to be apprehensive and heavily muscled, and may have elevated serum creatine kinase (CK) and aspartate aminotransferase (SGOT) (Britt *et al.*, 1976; Britt, 1979). Muscle cramping occurs with