Scientific Report

**Atrioventricular dissociation in a horse with clinical signs of colic**

Rezakhani, A¹; Goodarzi, M¹ and Chandler, K. J.²

¹Department of Clinical Sciences, School of Veterinary Medicine, University of Shiraz, Shiraz, Iran; ²Moray Coast Veterinary Group, Moray Coast, Morayshire, Scotland

*Correspondence: A. Rezakhani, Department of Clinical Sciences, School of Veterinary Medicine, University of Shiraz, Shiraz, Iran. E-mail: rezakhania@yahoo.com*

**Summary**

A horse presented to the large animal clinic with clinical signs of colic. Clinical examination was carried out and because of cardiac irregularity a base apex lead ECG was recorded. On the basis of criteria of normal rhythm, incomplete atrioventricular dissociation was diagnosed and its clinical significance was discussed.

**Key words:** ECG, Base apex lead, A-V dissociation, Horse

**Case history and clinical examination**

A thirteen-year-old gelding with history of anorexia, depression, hyperpnoea, yawing and clinical signs of colic was examined. On clinical examination, the animal had a heart rate of 76 beats per minute (bpm), respiratory rate of 21 breaths per minute and body temperature 37.9°C. There was no gastrointestinal movement audible on auscultation. Because of tachycardia and cardiac rhythm irregularities, a base apex electrocardiogram (ECG) was recorded before (A, B and C) and after treatment (D) (Fig. 1) for confirmation of cardiac dysrhythmias. Blood samples were taken for hematological and biochemical analysis. The animal had a neutrophilia (83%) with packed cell volume of 28%. Total protein, calcium, phosphorus and chloride concentrations were within normal limit.

Four liters Liquid paraffin were administrated orally and penicillin and streptomycin (4+4) given by intramuscular injection and phenylbutazone was injected intravenously (4.4 mg/kg) for five consecutive days. The horse died suddenly 5 days after being discharged from the hospital. No post mortem examination was carried out.

**ECG interpretation**

The first two impulses in trace A seem to be sinus origin with normal P-waves, QRS complexes and T-waves, the third, fifth, seventh, ninth and eleventh in the same trace occurring earlier than expected with no relationship between P-waves and their QRS complexes which are slightly different in comparing with the sinus impulses. In trace B, the first four impulses are normal and then there are 5 impulses with no visible P-waves (in two impulses the P-wave can be seen on the S-T segment) and there is a fusion beat (F). The 4 impulses at the end of this trace are again sinus origin. In trace C, there is no association between P-waves and QRS complexes (sinus rate is about75bpm and ventricular rate around79 up to 83bpm). There seems to be two types of QRS complexes in this trace, that is, junctional (J) and ventricular (V) ones. P-wave is sometimes seen on the S-T segment of QRS. Finally in trace D (recorded after medical therapy) all the impulses look like to be from the sinus node and having all the criteria of a regular rhythm but the heart rate is around 75 bpm that is, sinus tachycardia.

**Discussion**

In the normal animal with regular
Fig. 1: Four traces of ECG recorded on a base apex lead from a horse with clinical signs of colic. Paper speed is 25 mm/sec and sensitivity 5 mm equal to 1 cm. The different waves of a normal impulse are marked. There are junctional (J), ventricular origin (V) and fusion (F) impulses on the traces.

cardiac rhythm, an impulse originates from the sinus node and depolarizes both atria and produces the P-wave. Each impulse passes the atrioventricular node with a delay and then depolarizes the entire ventricular myocardium inducing a QRS complex. In this situation there is close association between P-waves and QRS complexes. A regular rhythm is present when in a given lead: all P-waves and QRS complexes are the same shape, each P-wave has a QRS and the P-R interval is in a normal limit, which is about 0.20 to 0.40 second for a normal adult horse. In normal and regular rhythm the number of sinus rate and ventricular rate, that is, the number of P-waves and QRS complexes are the same otherwise the relationship of the atria and the ventricles either partially or completely disrupted. Two main examples are third degree-atrioventricular block and A-V dissociation.

A-V dissociation implies that the atria and the ventricles are stimulated by two separate foci of impulse formation. In this case, the sinus node controls the atria and the A-V node or a focus in the ventricles takes the control of the ventricles. This can happen either when the discharge of the sinus node is slower than a lower pacemaker or a focus in the A-V node or in the ventricle has higher automaticity than the sinus node. If the discharges of both the atria and the ventricles are the same rate, then although there is dissociation but there is relationship between the p-waves and the QRS complexes. In most cases the A-V node or the ventricles have higher automaticity than the S-A node so association between P-waves and QRS complexes will be disrupted. In the case presented here the relationship between P-waves and QRS complexes are variable, sometimes the rhythm seems to be regular and then after a few beats this relationship is disrupted. This can be due to an incomplete A-V dissociation with atrial rate very close to the ventricular rate (75 bpm versus 79bpm with junctional or 83 bpm with ventricular origin). In dissociation there can be variable
ventricular conduction possibly as a consequence of local conduction blocks. By definition, the atria and ventricles are beating independently and ventricular activity is arising in that chamber so theoretically each ventricular complex can be described as ectopic beats. However, in this case, close similarity between the ventricular complexes during the arrhythmia and those observed when rhythm has returned to sinus suggest that most ventricular complexes are probably conducted normally through the conduction system (Physick-Sheard, 2003, personal communication), that is, most of them are junctional type. In this case QRS of impulses originating from the AV node are almost in the same shape of the normal sinus impulses. Six impulses in trace A and five impulses in the middle of trace C have a slightly different QRS complexes, which indicate that these are originating from the ventricles and producing ventricular tachycardia.

Premature ventricular contractions and ventricular tachycardia have been reported in horses (Holmes, 1980; Reef, 1989). Clinical significance of PVC depends on clinical signs and the frequency of ectopic beats. If the ectopic beats occur infrequently and disappear after exercise they will be taken as non-significant but if they occur frequently and are multifocal then they should be taken as pathological dysrhythmia (Physick-Sheard, 1999). Although ventricular tachycardia has been reported in a horse with no heart problem (Nielsen, 1990) it is a serious cardiac dysrhythmia as it may end in ventricular fibrillation (Scarratt and Lombard, 1981; Patteson1996; Kiryu et al., 1999). Ventricular tachycardia has been reported in cases with sudden death and horses with severe cardiovascular dysfunction (Vibe-Peterson and Nielson, 1980; Leblond et al., 2000). A-V dissociation is not a sign of heart disease per se in all cases. In animals it is often associated with systemic diseases, specifically abdominal crisis and also during anesthesia, in cases with or without cardiac abnormality, electrolyte imbalance (Miller et al., 1987; Grubb et al., 1996). In human, it has been reported during anesthesia, due to drugs such as digitalis, atropine, quinidine; infectious diseases or sometimes in ischemic heart diseases (Hurst et al., 1990).

In case reported here the cause(s) could be either due to homeostatic disturbance as a result of abdominal problem or myocardial disturbance. Whether the sudden death of animal was related to the dysrhythmia, abdominal disturbance or both is not clear, as no post mortem examination was carried out after death.

References

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