Treatment of Supraventricular Tachycardia in a Horse

Dorothy D. Whelchel, DVM, MS, DACVIM; Brett S. Tennent-Brown, BVSc, MS, DACVIM; Amanda E. Coleman, DVM, DACVIM; Gregg S. Rapoport, DVM, DACVIM; Uriel Blas-Machado, DVM, PhD, DACVP; Herbert W. Maisenbacher, VMD, DACVIM; Brenton C. Credille, DVM, PhD, DACVIM and Steeve Giguère, DVM, PhD, DACVIM^a.

From the University of Georgia, College of Veterinary Medicine, Athens, GA 30602, USA (Whelchel, Coleman, Rapoport, Blas-Machado, Credille, Giguère), University of Melbourne, Faculty of Veterinary and Agricultural Sciences, Werribee, VIC 3030, Australia (Tennent-Brown) and Veterinary Heart Care, Virginia Beach, VA 23462, USA (Maisenbacher)

Please address correspondence to Dr Dorothy Whelchel, East Campus Drive, College of Veterinary Medicine, University of Missouri, Columbia, MO 65211.

Email: whelcheld@missouri.edu

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Running title: Supraventricular tachycardia in a horse

Abbreviations

AV: atrioventricular

AVNRT: atrioventricular nodal reciprocating tachycardia

ECG: electrocardiogram

FAT: focal atrial tachycardia

LVID_d: left ventricular internal diameter during diastole

LVID_s: left ventricular internal diameter during systole

LAD: left atrial diameter

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OAVRT: orthodromic atrioventricular reciprocating tachycardia

PCL: pacing cycle length

SVT: supraventricular tachycardia

TVEC: transvenous electrical cardioversion

VRR: ventricular response rate

VTH: veterinary teaching hospital

Abstract

Objective - To describe the treatment of persistent supraventricular tachycardia (SVT) in a young horse in endurance training.

Case Summary - A 6-year-old Arab gelding in endurance training presented for a dysrhythmia and decreased performance. Supraventricular tachycardia was diagnosed and conversion to a normal sinus rhythm was achieved following administration of a constant rate infusion of amiodarone. However, reversion to SVT occurred shortly after initiation of ridden exercise. A second attempt to convert the dysrhythmia with amiodarone failed, but normal sinus rhythm was achieved with transvenous electrical cardioversion (TVEC). Post-mortem examination of the heart revealed extensive fibrous replacement of most of the left atrial myocardium; these changes likely provided the structural substrate for the dysrhythmia. The underlying cause of the fibrosis was not identified.

New or Unique Information Provided - Supraventricular tachycardia is a form of supraventricular tachyarrhythmia rarely diagnosed in the horse. A recent report has described sudden death of a horse following attempted conversion of SVT with oral flecainide acetate. In the present report, we describe short-term conversion of SVT in a horse using intravenous amiodarone with no significant adverse effects. When the dysrhythmia recurred, the animal was donated for teaching purposes and conversion was achieved with TVEC. Normal sinus rhythm persisted for 2 weeks until the horse was euthanized for post mortem evaluation of the heart. Intravenous amiodarone or transvenous electrical cardioversion could be considered as treatments for supraventricular tachyarrhythmias other than atrial fibrillation in the horse.

Key words: Supraventricular tachycardia; amiodarone; transvenous electrical cardioversion

Introduction

Although atrial fibrillation (AF) is commonly reported in horses, other forms of supraventricular tachyarrhythmia are considered rare in this species.¹ There are no well-documented recommendations for the management of horses with supraventricular tachyarrhythmias other than AF because of the limited number of reports in the literature. Quinidine, a class Ia antiarrhythmic, is most commonly used to treat AF. However, some authors have suggested that caution is required when using quinidine as the sole therapeutic agent because its vagolytic effects might increase atrioventricular (AV) conduction and, subsequently, ventricular response rate (VRR). Further, quinidine is associated with a rare risk of serious ventricular tacharrhythmias.² A recent report has described the sudden death of a horse following administration of flecainide acetate, a class Ic antiarrhythmic, for treatment of SVT characterized as fast atrial tachycardia (AT) with a high-grade second-degree AV block.³ Flecainide was administered after a combination of digoxin and quinidine failed to convert the horse to a sinus rhythm.³

Amiodarone, a class III antiarrhythmic, is used to treat focal atrial tachycardia (FAT) in children.⁴ Amiodarone has also been used to treat AF in horses and as a pre-treatment suggested to prevent the immediate recurrence of AF following transvenous electrical cardioversion (TVEC).⁵ TVEC appears to be a safe and effective means of treating AF in horses and is regarded by some as the treatment of choice for this dysrhythmia.⁶ TVEC has also been used to treat atrial flutter⁷ in a horse and this treatment modality might, therefore, have application in the management of other supraventricular dysrhythmias in horses. This report describes the conversion of SVT in a horse using intravenous amiodarone followed by the use of TVEC when the dysrhythmia recurred.

Case Report

A 6-year-old Arab gelding was referred to the Veterinary Teaching Hospital (VTH) for evaluation of a cardiac arrhythmia first detected approximately 4 weeks before presentation. The horse was

purchased 5 months earlier and had recently begun to train for endurance racing. Using a heart monitor and auscultation, the owners routinely monitored heart rate before, during, and after exercise. The owners reported that the horse's heart rate averaged 36/min at rest with a possible second degree AV block noted every 3 to 4 beats. After a 5-mile trot, a heart rate of 180/min was recorded with an occasional irregularity noted. In addition, the owners reported that the cardiac recovery index, the time to return to a normal resting heart rate after exercise, was prolonged after a 10-mile ride.⁸ No other changes in performance, exercise tolerance or respiratory effort were noted. There was no known history of previous illness or exposure to monensin or other toxins; however, the gelding had reportedly been bitten by an unidentified snake as a yearling.

On physical examination, the horse was bright, alert and responsive, weighing 435 kg with a body condition score of 6/9. Cardiac auscultation revealed a rate of 36/min and an irregularly irregular rhythm. No murmurs were appreciated. Respiratory rate was 20/min; respiratory effort and pulmonary auscultation were normal. Rectal temperature was 99.3°F (37.4°C). Mucous membranes were pink and moist with a capillary refill time of less than 2 seconds. Peripheral pulses were of normal intensity with a palpable pulse for every audible heartbeat. Jugular venous distention, abnormal jugular venous pulses or peripheral edema were not present and no other abnormalities were appreciated on the initial physical examination. A complete blood count and plasma biochemistry analysis were normal, as was plasma fibrinogen concentration (8.82 µmol/L [300 mg/dL]; reference range, 2.94-11.76 µmol/L [100-400 mg/dL]).

Base-apex electrocardiography (ECG) revealed supraventricular tachycardia (SVT) with intermittent second-degree atrioventricular (AV) block (Fig. 1). Atrial (p' wave) rate was 188/min and average ventricular response rate (VRR) was 40/min. The second-degree AV block was thought possibly to be physiologic; however, the presence of an occasional R-R interval approaching 3.4 seconds raised the suspicion of concurrent AV nodal pathology. p'R intervals were variable, but remained within the normal published reference range (<0.5 seconds).¹

No clinically significant abnormalities in chamber size or myocardial function were appreciated on transthoracic 2-dimensional echocardiography at rest.^a Left ventricular chamber sizes

(LVID_d 11.5 cm, reference range 11.9 ± 0.7 cm; LVID_s 7.7 cm, reference range 7.4 ± 0.7 cm) and fractional shortening (33%, reference range 32-45%) were within normal limits.⁹ Left atrial diameter as measured in the longitudinal axis from the left parasternal window (LAD 12.4 cm, reference range 12.82 ± 0.78 cm) was also within normal limits.⁹ Color flow Doppler showed mild mitral and pulmonic valve regurgitation. Serum cardiac troponin I (cTnI)^b concentration was normal (0.02 µg/L [0.02 ng/ml] [reference range 0.01-0.03 µg /L [0.01-0.03 ng/ml)]).¹⁰ Serum antibody titers for infectious agents potentially associated with myocarditis including *Streptococcus equi* subsp. *equi*, Equine Influenza virus, Equine Herpes virus 1 and Equine Viral Arteritis virus were inconsistent with recent infection and a Coggins' test for Equine Infectious Anemia was negative. Based on the ECG and echocardiographic findings, the gelding was diagnosed with SVT of unknown cause. Empiric treatment for myocardial inflammation with a tapering course of dexamethasone^c and stall rest was subsequently initiated. Dexamethasone was initially administered at a dose of 0.05mg/kg IM once daily and the dose decreased by 5 mg every 5 days. No change in the gelding's condition was noted by the owners during or following steroid therapy.

On re-evaluation one month after initial presentation, ECG revealed persistence of the SVT. Plasma electrolyte concentrations were again normal and echocardiographic findings were similar to those described for the initial evaluation. As the persistent dysrhythmia impeded the horse's athletic ability for endurance riding and no structural abnormalities were identified on resting 2-dimensional echocardiogram, pharmacologic cardioversion to normal sinus rhythm was attempted with amiodarone^d using a previously described protocol.¹¹ Radiotelemetry was used to continuously monitor the horse's ECG.^e A loading dose of 5 mg/kg/h was administered for one hour followed by a constant rate infusion of 0.83 mg/kg/h for 23 hours.¹¹ When the SVT failed to convert to normal sinus rhythm, the amiodarone infusion was continued at 1.7 mg/kg/h for an additional 24 hours. After 48 hours of therapy, conversion to normal sinus rhythm was achieved. No clinical side effects were observed during amiodarone treatment. Following discontinuation of the amiodarone infusion, the gelding remained in normal sinus rhythm and was discharged the following day on a two-week course of oral amiodarone (5mg/kg q 24h) and strict stall rest.

Since amiodarone therapy has been associated with hepatopathy in people, dogs and horses, plasma biochemistry analyses were serially performed over the weeks following discharge.¹¹ Mild hyperbilirubinemia (total bilirubin 64.98 $\mu\text{mol/L}$ [3.8 mg/dL]; reference range, 1.71-61.56 $\mu\text{mol/L}$ [0.1-3.6 mg/dL]) was detected but considered clinically insignificant as serum bile acid concentration and plasma γ -glutamyltransferase and sorbitol dehydrogenase activities remained within normal limits.

Three weeks after cessation of oral amiodarone, physical examination findings were normal and an ECG revealed a normal sinus rhythm. Serum cardiac troponin I concentration and plasma biochemistry analysis (including measurement of bilirubin concentration) were normal. Urinary fractional excretions of sodium, potassium and chloride were normal. Light ridden exercise was subsequently initiated, during which the horse was ridden at a trot 2 or 3 times weekly for 8 to 13 km.

After one month of light exercise, the owners reported that the horse was tachycardic at a walk and trot. When evaluated, resting heart rate was 44/min and an irregularly irregular rhythm was present. All other physical examination findings, a complete blood count, plasma biochemistry analysis and plasma fibrinogen concentration were normal. Electrocardiography revealed reversion to SVT; echocardiographic findings were unchanged. Pharmacologic conversion with amiodarone was again attempted, but conversion to sinus rhythm was not achieved following 30 hours of intravenous amiodarone infusion at 1.7 mg/kg/hr. Since the drug has a long half-life, conversion can occur after termination of amiodarone infusion.¹¹ The gelding was therefore discharged to the owners' care for continued monitoring; however, the dysrhythmia persisted 2 weeks after discharge. At this time, the horse was donated for teaching purposes.

Transvenous electrical cardioversion (TVEC) under general anesthesia was subsequently attempted as previously described.⁶ Briefly, cardioversion electrodes (9.5cm titanium wire coils^f) were placed via an indwelling catheter into the right atrium and left pulmonary artery under ultrasonographic guidance. After induction of anesthesia, proper catheter placement was confirmed using radiography. The cardioversion catheters were connected to a biphasic defibrillator (Lifepak12^g), with the right atrial electrode as the cathode and the pulmonary arterial electrode as the

anode. An ECG was monitored and shocks were synchronized with the R wave. Conversion to normal sinus rhythm was achieved with the first shock using an energy of 50 J. The cardioversion catheters were removed and the gelding recovered uneventfully from anesthesia.

Following a week of stall rest, the gelding was trotted for 30 minutes on a lunge line daily. Normal sinus rhythm was maintained for 2 weeks until the horse was euthanized for post-mortem evaluation of the heart. Neither enlargement of the cardiac chambers nor changes in ventricular wall thickness were appreciated grossly. However, greater than 75% of the left atrial epicardial and endocardial surfaces were pale yellow or tan and firm (Fig. 2A). Similar, although less extensive, changes were present in the right atrium. Widely scattered, irregular white or gray plaques measuring less than 10 mm in diameter were present on the endocardial surfaces of the left ventricle (Fig. 2B). The intimal surfaces of the ascending aorta were opaque with longitudinal irregular grooves (Fig. 2B). The AV and semilunar valves were intact and appeared grossly normal. Microscopically, there was severe fibrosis and steatosis of the atrium; interlacing collagen bundles and adipose tissue replaced most of the atrial myocardium (Fig. 2C). Individual myofibers varied in cross-sectional diameter and degenerate myofibers often had moderate sarcoplasmic swelling and vacuolization. The endocardium of the left ventricle was thickened with increased connective tissue. The endocardial plaques observed grossly consisted of subendocardial collections of mineralized myofibers (Fig. 2D). No evidence of active myocarditis was identified and a specific cause for the tissue changes was not determined.

Discussion

With the exception of atrial fibrillation, supraventricular tachyarrhythmias - an umbrella term used to describe any dysrhythmia originating from “above” the ventricles - are rarely reported in horses.^{12,13} To the authors’ knowledge, this is the first reported case of conversion of SVT to sinus rhythm in a horse using amiodarone and TVEC. Recently, the sudden death of a horse with an SVT characterized as fast AT with a high-grade second-degree AV block was reported after oral administration of flecainide acetate.³ SVT is most often encountered as a complication of quinidine sulfate administration.¹ However, this dysrhythmia can also occur with hypokalemia² and in association with

chronic monensin ingestion.¹² Causes of SVT in people include digitalis and other toxicities, metabolic disturbances, focal atrial disease, atrial scarring following surgery and acute myocardial infarction.¹⁴

SVT is a unifying term used to describe a number of cardiac dysrhythmias that can originate from either abnormal atrial circuits (focal atrial tachycardia [FAT]), the AV junction (atrioventricular nodal reciprocating tachycardia [AVNRT]), or an extra-nodal accessory pathway (orthodromic atrioventricular reciprocating tachycardia [OAVRT]).¹⁵ Each of these dysrhythmias typically results in a narrow-QRS tachycardia unless aberrant ventricular conduction is also present.¹⁶ The presence of AV block in the case described here rules out the possibility of OAVRT, which requires both a non-nodal aberrant muscular connection between the atria and ventricles and a functional AV node for maintenance of the macro-reentrant circuit (ie, 1:1 AV conduction is obligatory).¹⁷ Since the same is not true for FAT or AVNRT,¹⁷ one of these mechanisms was suspected to be the cause of the SVT in the case reported here. Surface ECG can provide some clues as to the pathway maintaining an SVT (ie, FAT vs. AVNRT) in people, but extrapolation to horses is difficult. Furthermore, some overlap in ECG changes exist between the 2 mechanisms and definitive diagnosis can be difficult.¹⁸

The degree of AV block noted in the horse of this report warrants further comment. In a study evaluating AV nodal electrophysiology, the ventricular response rate (VRR) in normal, conscious horses subjected to rapid atrial pacing was independent of pacing cycle length (PCL) when the PCL was between 1200 and 300 msec (50-200 impulses/minute).¹⁹ Normal horses paced at a PCL of 300 msec (80 msec faster than the p'-p' interval of the horse of this report) exhibited VRR of 41 ± 8 /min, corresponding to an AV conduction ratio of 0.2 ± 0.04 . Therefore, as in other species, some degree of AV block is expected in the normal equine heart subjected to rapid atrial rates. In the horse of this report, instantaneous VRR as low as 18/min were recorded; based on the results of previous studies, this could indicate AV nodal pathology but might also reflect physiologic alterations in vagal input to the AV node.^{19,20}

Treatment of incessant or symptomatic SVT of all types is indicated in human patients.¹⁶ Prevention of tachycardia-induced cardiomyopathy is another important consideration in people with

persistent SVT.¹⁵ In people, SVT often requires radiofrequency ablation of ectopic automatic foci or reentrant circuits.^{4,16,21,22} Amiodarone, an antiarrhythmic with predominantly class III activity, has been used successfully in children with FAT.⁴ Adverse reactions following amiodarone infusions to horses include transient hyperbilirubinemia without other evidence of hepatopathy, as was observed in the case described here.^{11,23} Hindlimb weakness that usually resolves within hours of discontinuing drug infusion and diarrhea have also been reported in horses.^{11,23} Plasma concentrations of amiodarone or its active metabolite, desethyleamiodarone, were not measured in this case. Although therapeutic monitoring would have provided additional information, the plasma concentrations of amiodarone and desethyleamiodarone associated with successful conversion or adverse side effects is variable between individual horses, likely due to the multi-compartmental pharmacokinetics of this drug in horses.^{11,23} Quinidine is considered the drug of choice for pharmacological conversion of atrial fibrillation in horses, a far more common arrhythmia in this species.² Based on human experience with quinidine²⁴ and results of equine electrophysiologic studies,¹⁹ there was concern that quinidine's anticholinergic effects might have enhanced AV nodal conduction, potentially increasing VRR in the horse of this report.²⁴ Horses undergoing rapid atrial pacing showed shortening of the AV nodal functional refractory period with an increase in VRR following quinidine administration.¹⁹

The horse of the current report reverted to SVT shortly after re-introduction to exercise and a second treatment with intravenous amiodarone did not result in conversion. Although the gelding did not show clinical signs of heart failure and repeated echocardiographic examinations were normal, marked myocardial changes were evident on post-mortem evaluation. Additional diagnostics including stress echocardiography and tissue Doppler imaging might have provided additional insight in this case and allowed a more accurate ante mortem prognosis. Although not detected on traditional 2-dimensional echocardiography, the degree of myocardial fibrosis observed histologically likely provided the structural substrate necessary to sustain the dysrhythmia, making it difficult or impossible to achieve long-term maintenance of sinus rhythm, although only short-term follow-up was possible following TVEC. The post-mortem finding of extensive myocardial fibrosis in this case is consistent with chronic myocardial injury. Although there was no histological evidence of active

myocarditis and serum cTnI concentrations were normal, chronic myocarditis can lead to end stage myocardial fibrosis affecting both the contractile and conduction systems.²⁵ In addition to myocarditis, myocardial fibrosis in the horse has been associated with vitamin E and/or selenium deficiency, monensin toxicity and snake bite envenomation.²⁵ Other cardiac dysrhythmias (including atrial fibrillation and paroxysmal SVT), congestive heart failure and extensive myocardial fibrosis have also been identified in horses following rattlesnake envenomation.^{26,27} The relevance of the historically reported snake bite in this case is unknown but myocardial fibrosis secondary to monensin, blister beetle or oleander toxicosis was considered unlikely based on the history and clinical findings.

In summary, the current case report describes the use of amiodarone and TVEC for treatment of SVT in a horse. Initial conversion to sinus rhythm was achieved with intravenous administration of amiodarone. Treatment was well tolerated with no adverse effects appreciated other than a mild and transient hyperbilirubinemia. However, shortly after re-introduction to ridden exercise, the horse reverted to SVT, at which point the horse was converted back to a normal sinus rhythm using TVEC. The degree of myocardial fibrosis indicated a prior insult to the myocardium and likely contributed to the development and persistence of SVT. In the future, intravenous amiodarone or TVEC could be considered as treatments for supraventricular tachyarrhythmias other than atrial fibrillation in the horse.

Footnotes

^a GE Vivid 7 Dimension, GE Medical Systems, Milwaukee, Wisconsin, USA

^b ADVIA Centaur CTnI Ultra Assay, Immulite 1000, Siemens, Deerfield, Illinois, USA

^c Dexium, Bimeda Inc., Oakbrook Terrace, Illinois, USA

^d Amiodarone HCl, Bedford Laboratories, Bedford, Ohio, USA

^e Televet Telemetric RCG, Kruuse, Langeskov, Denmark

^f Cardioversion catheters, Rhythm Technologies, Irvine, California, USA

^g Lifepak 12 defibrillator, Medtronic, Minneapolis, Minnesota, USA

References

1. Bonagura JD. Cardiovascular diseases In: Reed SM, Bayley WM, Sellon DC, eds. *Equine Internal Medicine*. 3rd ed. Philadelphia: Saunders, 2009;456-459.
2. Reef VB, Marr CM. Dysrhythmias: assessment and medical management In: Marr CM, Bowen M, eds. *Cardiology of the horse*. 2nd ed. Edinburgh: Saunders, 2011;159-178.
3. Dembek KA, Hurcombe SD, Schober KE, et al. Sudden death of a horse with supraventricular tachycardia following oral administration of flecainide acetate. *J Vet Emerg Crit Care* 2014;24:759-763.
4. Mehta AV, Sanchez GR, Sacks EJ, et al. Ectopic automatic atrial tachycardia in children: clinical characteristics, management and follow-up. *J Am Coll Cardiol* 1988;11:379-385.
5. De Clercq D, van Loon G, Schauvliege S, et al. Transvenous electrical cardioversion of atrial fibrillation in six horses using custom made cardioversion catheters. *Vet J* 2008;177:198-204.
6. McGurrin MK, Physick-Sheard PW, Kenney DG, et al. Transvenous electrical cardioversion in equine atrial fibrillation: technique and successful treatment of 3 horses. *J Vet Intern Med* 2003;17:715-718.
7. Christmann U, van Loon G. ECG of the month. Atrial flutter. *J Am Vet Med Assoc* 2013;242:165-167.
8. Robert C, Benamou-Smith A, Leclerc JL. Use of the recovery check in long-distance endurance rides. *Equine Vet J Suppl* 2002:106-111.
9. Patteson MW, Gibbs C, Wotton PR, et al. Echocardiographic measurements of cardiac dimensions and indices of cardiac function in normal adult thoroughbred horses. *Equine Vet J Suppl* 1995:18-27.
10. Nath LC, Anderson GA, Hinchcliff KW, et al. Serum cardiac troponin I concentrations in horses with cardiac disease. *Aust Vet J* 2012;90:351-357.

11. De Clercq D, van Loon G, Baert K, et al. Intravenous amiodarone treatment in horses with chronic atrial fibrillation. *Vet J* 2006;172:129-134.
12. Muylle E, Vandenhende C, Oyaert W, et al. Delayed monensin sodium toxicity in horses. *Equine Vet J* 1981;13:107-108.
13. Guthrie AJ, Nichas E, Viljoen FV, et al. Sustained supraventricular tachycardia in a horse. *J S Afr Vet Assoc* 1989;60:46-47.
14. Prystowsky EN, Padanilam BJ, Waldo AL. Atrial fibrillation, atrial flutter, and atrial tachycardia In: Fuster V, Walsh RA, Harrington RA, eds. *Hurst's The Heart*. 13th ed. New York, NY: McGraw-Hill Professional, 2011.
15. Blomstrom-Lundqvist C, Scheinman MM, Aliot EM, et al. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias--executive summary. a report of the American college of cardiology/American heart association task force on practice guidelines and the European society of cardiology committee for practice guidelines (writing committee to develop guidelines for the management of patients with supraventricular arrhythmias) developed in collaboration with NASPE-Heart Rhythm Society. *J Am Coll Cardiol* 2003;42:1493-1531.
16. Olgin J, Zipes DP. Specific Arrhythmias: Diagnosis and Treatment In: Bonow RO, Mann DL, Zipes DP, et al., eds. *Braunwald's Heart Disease, A Textbook of Cardiovascular Medicine*. 9th ed. Philadelphia: Elsevier, 2012;771-824.
17. Mandel WJ. Cardiac arrhythmias: Their mechanisms, diagnosis and management. 3rd ed: Lippincott Williams & Wilkins, 1995.
18. Tilley LP. Uncommon complex arrhythmias In: Tilley LP, ed. *Essentials of canine and feline electrocardiography, interpretation and treatment*. 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 1992;389-390.
19. Schwarzwald CC, Hamlin RL, Bonagura JD, et al. Atrial, SA nodal, and AV nodal electrophysiology in standing horses: normal findings and electrophysiologic effects of quinidine and diltiazem. *J Vet Intern Med* 2007;21:166-175.

20. Meijler FL, Kroneman J, van der Tweel I, et al. Nonrandom ventricular rhythm in horses with atrial fibrillation and its significance for patients. *J Am Coll Cardiol* 1984;4:316-323.
21. Chen SA, Tai CT, Chiang CE, et al. Focal atrial tachycardia: reanalysis of the clinical and electrophysiologic characteristics and prediction of successful radiofrequency ablation. *J Cardiovasc Electrophysiol* 1998;9:355-365.
22. Feld GK. Catheter ablation for the treatment of atrial tachycardia. *Prog Cardiovasc Dis* 1995;37:205-224.
23. De Clercq D, van Loon G, Baert K, et al. Effects of an adapted intravenous amiodarone treatment protocol in horses with atrial fibrillation. *Equine Vet J* 2007;39:344-349.
24. Josephson ME, Seides SF, Batsford WP, et al. The electrophysiological effects of intramuscular guanidine on the atrioventricular conducting system in man. *Am Heart J* 1974;87:55-64.
25. Buergelt CD. Equine cardiovascular pathology: an overview. *Anim Health Res Rev* 2003;4:109-129.
26. Dickinson CE, Traub-Dargatz JL, Dargatz DA, et al. Rattlesnake venom poisoning in horses: 32 cases (1973-1993). *J Am Vet Med Assoc* 1996;208:1866-1871.
27. Fielding CL, Pusterla N, Magdesian KG, et al. Rattlesnake envenomation in horses: 58 cases (1992-2009). *J Am Vet Med Assoc*;238:631-635.

Figure 1: Electrocardiogram tracing recorded from a 6-year-old Arab gelding evaluated for a cardiac dysrhythmia. The tracing shows atrial tachycardia with intermittent second degree AV block.

Electrocardiogram was performed with standard base apex configuration. Atrial (P wave) rate was approximately 188/min with a ventricular response rate of 40/min.

Figure 2: Gross post mortem and histopathologic images of the heart from a 6-year-old Arab gelding diagnosed with atrial tachycardia. **(A)** Left atrium showing yellow to tan discoloration (solid arrows) of the epicardial surface. **(B)** Left ventricular endocardial surface showing scattered, mineralized plaques (solid arrows). The semilunar valves (open arrows) are intact but the intimal surface of the ascending aorta is irregular in appearance with longitudinal grooves (asterisk). **(C)** Interlacing fibrocollagen bundles (blue) and adipose tissue (white) have replaced over 50% of the left atrial myocardium (red). The epicardium (solid arrow) is diffusely thickened with an increased accumulation of fibrous connective tissue. Masson's trichrome stain, bar = 2.83 mm. **(D)** Increased fibrous connective tissue and mineralization (solid arrow) expands the endocardium. The asterisk (*) indicates the lumen of the left ventricle. Hematoxylin and eosin stain, bar = 100 μ m.

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Author/s:

Whelchel, DD;Tennent-Brown, BS;Coleman, AE;Rapoport, GS;Blas-Machado, U;Maisenbacher, HW;Credille, BC;Giguere, S

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