How to Diagnose and Treat Ventricular Tachycardia

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1. Introduction
Although ventricular arrhythmias are less common than atrial arrhythmias, they are more likely to be life threatening and associated with underlying cardiac disease or a multisystemic disorder. Common etiologies include electrolyte abnormalities (hypomagnesemia and hypokalemia), drug administration, or primary myocardial disease. Ventricular tachycardia is characterized by a rapid cardiac rhythm that originates in the ventricle below the bundle of His in the conduction system, in the surrounding ventricular myocardium, or both. It can be characterized by an irregular or a regular rhythm. Diagnosis of ventricular tachycardia is straightforward with an electrocardiogram, but identification of the etiology often remains a challenge for veterinary practitioners. Several treatment options exist, and a discussion of the anti-arrhythmic therapies is included below.

A refractory case of ventricular tachycardia that was ultimately successfully treated with amiodarone will be presented to illustrate the diagnosis and principles of therapy.

2. Materials and Methods
Diagnosis of ventricular tachycardia cannot typically be made based on auscultation alone. Loud booming heart sounds can occasionally be heard and are associated with the simultaneous production of two heart sounds during periods of atrioventricular disassociation. Ventricular tachycardia can be uniform (regular rhythm) or multiform (irregular rhythm). Multiform ventricular tachycardia is associated with more rapidly progressive signs of congestive heart failure and increased electrical instability, which can result in ventricular fibrillation. If the QRS complex occurs within the preceding T wave, this is called the “R on T” phenomenon, and it is associated with an increased chance of ventricular fibrillation. QRS complexes are wide and bizarre and not associated with P waves (Figs. 1–3). A series of four or more ventricular premature depolarizations is diagnostic of ventricular tachycardia.4

A single lead (I) is sufficient to diagnose most rhythm disturbances in the horse. ECG tracings in ventricular tachycardia reveal abnormal QRS complexes that are often wide and bizarre in appearance and not associated with P waves. The T waves may be large and follow the QRS complex immediately. If a differentiation between ventricular and supraventricular rhythms cannot be made, additional leads may be used. ECG evaluates the rhythm and
rate only. Because of the pathway of depolarization in the horse (the deeply penetrating Purkinje system and depolarization from ventricular endocardium to epicardium occurring explosively and in many directions at one time), chamber sizes cannot be determined. Usually a base-apex lead is used:

LA (+): Attach to apex beat behind left elbow.
RA (−): Attach dorsal to right scapular spine.
RL (ground): Attach anywhere (usually right side of the neck).

After the diagnosis of ventricular tachycardia has been established, additional diagnostic testing for an underlying etiology should be considered. Myocarditis, myocardial necrosis, fibrosis, neoplasia, bacterial endocarditis, hypoxia, ischemia, electrolyte or metabolic disturbances, anesthesia, drug administration, sepsis, endotoxemia, toxic myocardial injury, aortic root rupture, and autonomic nervous system imbalance have all been associated with the development of ventricular tachycardia. Underlying gastrointestinal disorders or sources of sepsis should be ruled out. A serum or plasma biochemical profile will determine if electrolyte abnormalities are contributing to the conduction disturbance. Because ventricular tachycardia may be a consequence of myocarditis, measurement of myocardial muscle enzymes such as creatinine kinase-MB (CK-MB), α-hydroxybutyrate dehydrogenase (HBDH) lactate dehydrogenase (LDH 1 and 2), or preferably, cardiac troponin I (<0.15 ng/ml) is recommended. However, normal values do not rule out myocardial damage. Suspected viral etiologies of myocarditis in horses include equine herpes virus and equine influenza. Testing should be considered in horses with a history of fever. Finally, an echocardiogram is necessary to

Fig. 1. Base apex ECG recorded in lead I. Sinus tachycardia is detected (HR = 60 bpm). The test is recorded at a paper speed of 50 mm/s and a sensitivity of 20 mm = 1 mV.

Fig. 2. Base apex ECG recorded in lead I. The normal QRS complex (solid arrow) is followed by a run of ventricular tachycardia. The first abnormal complex is indicated (open arrow). The test is recorded at a paper speed of 50 mm/s and a sensitivity of 5 mm = 1 mV.

Fig. 3. Uniform ventricular tachycardia is detected. The R-R interval is regular. The P-P interval is regular, but the P waves (arrows) are often buried in QRS complexes or T waves. The test was recorded at a paper speed of 50 mm/s and a sensitivity of 5 mm = 1 mV.
determine if underlying cardiac disease such as valvular insufficiency or endocarditis is present.

3. Treatment of Ventricular Tachycardia
Treatment is indicated if there are signs of cardiovascular collapse, the heart rate is excessively high (>120 beats per min [bpm]), the complexes are multif orm, or there is detection of the “R on T” phenomenon. Pulmonary edema, if present, should be treated with furosemide (1–3 mg/kg, IV or IM). Table 1 describes anti-arrhythmic therapies for the horse. The following list describes drugs that can be used in the treatment of ventricular tachycardia in the order that they are usually attempted:

1. Lidocaine is often used as a first-choice therapy (0.5 mg/kg, IV bolus every 3–5 min for a total of 1.5 mg/kg; therapy can then be continued as a constant rate infusion (CRI) at 0.05 mg/kg/min). Lidocaine is a class IB drug, which is a sodium-channel blocker and acts to shorten the refractory period. The most common adverse effect is excitation. Neurologic side effects can typically be controlled with diazepam or a reduction in the rate of administration. Boluses and infusions are typically well tolerated and cause minimal hemodynamic or electrophysiologic changes at non-toxic doses.6

2. Magnesium sulfate is the second choice for therapy (IV infusion of 1 g/min to a maximum of 25 g). Magnesium sulfate can be effective when added to fluids of normomagnesemic or hypomagnesemic horses, and it is particularly useful in quinidine-induced torsades de pointes. Magnesium may cause hypotension but has no other adverse cardiovascular effects. In hypomagnesemic patients, magnesium sulfate acts as a cofactor for the Na/K ATPase pump. In horses with normal serum magnesium concentrations, a calcium channel-blocking mechanism is believed to account for its action.7 The authors often use magnesium in combination with lidocaine and other therapies as a 25-g IV slow bolus twice daily (in 1 l of isotonic fluids).

Treatment with lidocaine and magnesium are considered to be ideal therapeutic options in circumstances where continuous monitoring is not available. The additional therapies listed below should be considered in hospitals where continuous monitoring and ECG are available. Referral of these cases for additional diagnostics and therapy should be offered if they are evaluated in the field. Each of the following drugs has the potential to cause life-threatening adverse effects and should be administered under strict supervision.

3. Procainamide (2 mg/kg, IV bolus every 5–10 min up to a total of 20 mg/kg) is a class IA anti-arrhythmic that also acts by sodium-channel blockade. This drug has been reported to be the drug of choice for ventricular tachycardia in conscious horses.7 Unlike quinidine, this drug does not interact with digoxin. Although procainamide may have similar side effects as quinidine, the authors

Table 1. Anti-Arrhythmic Therapy in Horses

<table>
<thead>
<tr>
<th>Drug</th>
<th>Indications</th>
<th>Dose</th>
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<tbody>
<tr>
<td>Amiodarone</td>
<td>AF, VT</td>
<td>5 mg/kg/h for 1 h followed by 0.83 mg/kg/h for up to 48 h</td>
</tr>
<tr>
<td>Atropine</td>
<td>Sinus bradycardia,</td>
<td>0.005–0.01 mg/kg IV</td>
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<td></td>
<td>Vagally induced arrhythmias</td>
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<tr>
<td>Bretylium tosylate</td>
<td>Life-threatening VT</td>
<td>3–5 mg/kg, IV (up to 10 mg/kg total dose)</td>
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<tr>
<td></td>
<td>Ventricular fibrillation</td>
<td></td>
</tr>
<tr>
<td>Digoxin</td>
<td>SVT, AF (rate control)</td>
<td>0.0022 mg/kg, IV, q 12 h</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.011 mg/kg, PO, q 12 h</td>
</tr>
<tr>
<td>Diltiazem</td>
<td>SVT</td>
<td>0.125 mg/kg IV over 2 min (can repeat every 10 min up to a total dose of 1.125 mg/kg)</td>
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<tr>
<td>Lidocaine</td>
<td>VT</td>
<td>0.5 mg/kg, IV bolus every 3–5 min for a total of 1.5 mg/kg. Therapy can then be continued as a CRI at 0.05 mg/kg/min</td>
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<tr>
<td>Magnesium sulfate</td>
<td>VT</td>
<td>1 g/min, IV to a maximum of 25 g</td>
</tr>
<tr>
<td>Procainamide</td>
<td>VT, AF, atrial and ventricular arrhythmias</td>
<td>2 mg/kg, IV bolus every 5–10 min up to a total of 20 mg/kg; 25–35 mg/kg, PO, q 8 h</td>
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<tr>
<td>Propafenone*</td>
<td>Refractory VT, AF, atrial arrhythmias</td>
<td>0.5–1 mg/kg in 5% dextrose slowly IV over 5–10 min; 2 mg/kg, PO, q 8 h</td>
</tr>
<tr>
<td>Propanolol</td>
<td>SVT</td>
<td>0.03–0.2 mg/kg, IV</td>
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<tr>
<td></td>
<td>Unresponsive VT</td>
<td>0.38–0.78 mg/kg, PO, q 8 h</td>
</tr>
<tr>
<td>Quinidine gluconate</td>
<td>VT, AF</td>
<td>1–2 mg/kg, IV bolus repeated every 10 min up to a total dose of 12 mg/kg</td>
</tr>
<tr>
<td>Quinidine sulfate</td>
<td>AF</td>
<td>22 mg/kg, NGT, q 2 h for four doses followed by 22 mg/kg, q 6 h</td>
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*a Intravenous formulation not available in the United States. VT = ventricular tachycardia; SVT = supraventricular tachycardia; AF = atrial fibrillation.*
have noted few toxic effects in conjunction with its administration.

4. Quinidine gluconate (1–2 mg/kg, IV bolus repeated every 10 min up to a total dose of 12 mg/kg) is also a class IA drug, and it can be used for multiple ventricular and supraventricular arrhythmias. Quinidine gluconate is protein bound, and concurrent administration with digoxin can result in higher serum concentrations of both agents from competitive binding. It is more cost effective than therapy with procainamide; however, it is contraindicated in hypotensive patients, because it may cause peripheral vasodilation. Cardiovascular side effects of quinidine administration include hypotension, decreased cardiac contractility, prolongation of the QRS complex, rapid supraventricular tachycardia, ventricular arrhythmias, and sudden death. Supraventricular tachycardia is an idiosyncratic reaction rather than an indication of toxicity, and it occurs because of a sudden release of vagal tone at the atroventricular (AV) node. Supraventricular tachycardia (SVT) has traditionally been treated with digoxin (0.0022 mg/kg, IV, if heart rate [HR] is rapidly increasing and not responsive to stimulation or 0.011 mg/kg, PO, if HR is steady and returns to normal with stimulation).

5. Amiodarone (5 mg/kg/h for 1 h followed by 0.83 mg/kg/h for up to 48 h) is a class III anti-arrhythmic agent. It has been used in humans to treat atrial fibrillation (AF), atrial flutter, and ventricular tachyarrhythmias. In horses, amiodarone has been successfully used to treat chronic atrial fibrillation and refractoryventricular tachycardia. Amiodarone blocks sodium and calcium channels in addition to beta-adrenoceptors. It also prolongs repolarizations and increases the duration of the action potential. Oral bioavailability of amiodarone in horses is low and variable ranging between 6% and 34%. Amiodarone, it is a class III drug that lengthens the refractory period through potassium-channel blockade. The authors have not used this drug in clinical cases.

6. Esmolol is administered at 200 μg/kg over 1 min; if there is no response after 10 min, 500 μg/kg is administered over 1 min. If the horse converts, CRI can be maintained at 25 μg/kg/min until switched to oral propanolol. Esmolol is a class II beta-adrenoceptor antagonist. Like propranolol, it works by slowing the cardiac rate.

7. Propafenone (0.5–1 mg/kg in 5% dextrose, IV, slowly over 5–10 min) is a class IC drug that blocks sodium channels and is a beta-adrenoceptor antagonist. IV propafenone has been used successfully in horses for refractory ventricular tachycardia. Unfortunately, the IV formulation is currently not available in the United States.

8. Bretylium tosylate12 (3–5 mg/kg, IV; it can be repeated up to a total dose of 10 mg/kg) is reserved for life-threatening ventricular tachycardia or ventricular fibrillation. Like amiodarone, it is a class III drug that lengthens the refractory period through potassium-channel blockade. The authors have not used this drug in clinical cases.

A period of rest (4–8 wk) is recommended after successful treatment. Echographic and electrographic evaluation is necessary before return to athletic activity. A 24-h ECG recording or exercising ECG is more useful than just a brief resting ECG. Many horses can return to their prior level of performance if there is no functional residual damage to the heart.

4. Results and Discussion

The case discussed is a yearling Quarter Horse colt who presented to the hospital with a history of fever up to 105°F and tachycardia. The colt was diagnosed with ventricular tachycardia and myocarditis based on the results of ECG, echocardiogram, cardiac troponin I, routine laboratory evaluation, and additional diagnostics. Conversion to sinus rhythm was challenging and attempted with lidocaine, magnesium sulfate, procainamide, and quinidine; ultimately, it was successful with amiodarone. Additional supportive therapy included furosemide, flunixin meglumine, dexamethasone, broad-spectrum antibiotics, and judicious use of IV fluids.

Ventricular tachycardia is considered a life-threatening condition in advanced cases. A step-wise approach to diagnosis and antidysrhythmic treatment will result in the best outcome. Therapeutic monitoring during treatment must be done with periodic or continuous ECG readings; however, this limits feasibility of treatment to practitioners with monitoring systems. Amiodarone should be considered as a potential therapy in refractory cases, and reports of its use in horses have increased in the past several years.

References