ECG of the Month

The Academy of Veterinary Cardiology sponsors this feature. Readers of the *JAVMA* are invited to submit contributions. Contributions should include a brief description of the case (150 words); good quality contrast glossy photographs (5 X 7 in) of tracings, with the components of a QRS complex labeled; figure legends with information on ECG lead, paper speed, and voltage calibration; an ECG interpretation; and, a discussion of the abnormality. Two hard copies of the manuscript and each figure must be submitted, along with an electronic copy on a 3.5-in PC-formatted disk. Submissions that are complete will be sent to the feature coordinator, Dr. Robert Hamlin, at the Ohio State University for review.

A 10-year-old 430-kg (946-lb) Arabian gelding was evaluated for a sudden onset of lethargy and recumbency. There were 2 puncture wounds on the ventral aspect of the lower lip and extreme edema of the face, oral cavity, neck, and chest. Oral mucous membranes were pink with a capillary refill time of 1 to 2 seconds. The horse was tachycardiac (80 beats/min) with an arrhythmia that could be auscultated and had a jugular pulse that extended to the proximal region of the neck. The horse stood only after vigorous stimulation. Dexamethasone (40 mg, IV) was administered.

The results of a CBC indicated dehydration (PCV 53%) and mild band neutrophilia (180 cells/µl). Abnormalities detected by serum biochemical analysis included a high BUN concentration (29 mg/dl), high aspartate transaminase (2911 U/L) and creatine kinase (34,000 U/L) activities, and a low total protein concentration (4.7 g/dl). Electrolyte values were within reference limits. A base-apex lead ECG was performed,[a] using a 2-clip transmitter (**Fig 1**).

ECG Interpretation

Electrocardiography revealed paroxysmal ventricular tachycardia with frequent **ventricular premature complexes (VPC)**. The ectopic QRS complexes (Fig 1) are multiform in nature, with the first and third ectopic QRS complexes in the tracing having a polyphasic deflection, the second ectopic QRS complex a predominantly negative deflection, and the last 3 ectopic QRS complexes having a predominantly positive deflection.

Quinidine sulfate (10 g/via nasogastric tube, q 8 h), procaine penicillin G (22,000 U/kg [10,000 U/lb] of body weight, IM, q 12 h), and dexamethasone (100 mg, IV, q 24 h) were administered for 5 days. The arrhythmia was less severe on day 5 and not detected on day 6 (**Fig 2**). The horse had a bright and alert attitude, and the edema of the neck, chest, and head resolved. The dosages of quinidine sulfate and dexamethasone were reduced on day 7, and administration was discontinued on day 8.

The gelding's heart was auscultated often during the 2.5 years following the illness. No arrhythmia or other cardiac problems have been detected. The horse has regained approximately 100 kg of body weight that was lost during its illness and has returned to full working performance.

Discussion

Base-apex leads are often used in the performance of ECG in horses, because the electrical leads are

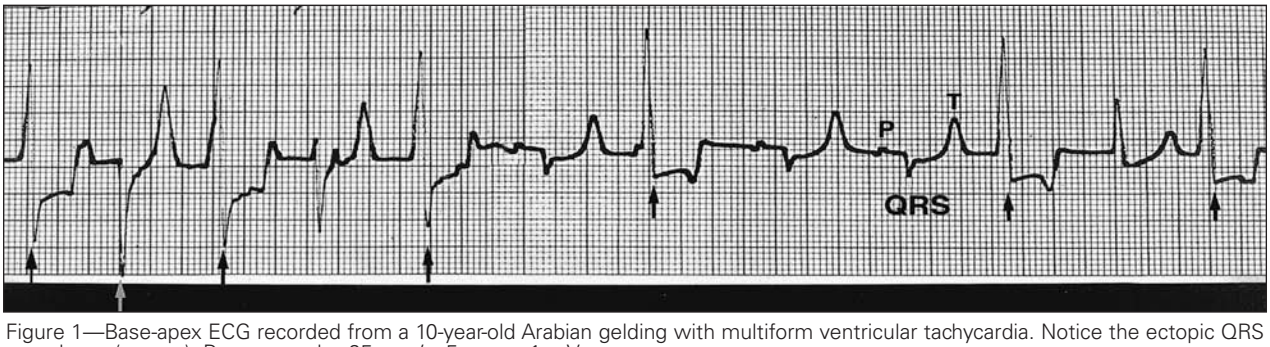

Figure 1—Base-apex ECG recorded from a 10-year-old Arabian gelding with multiform ventricular tachycardia. Notice the ectopic QRS complexes (arrows). Paper speed = 25 mm/s; 5 mm = 1 mV.

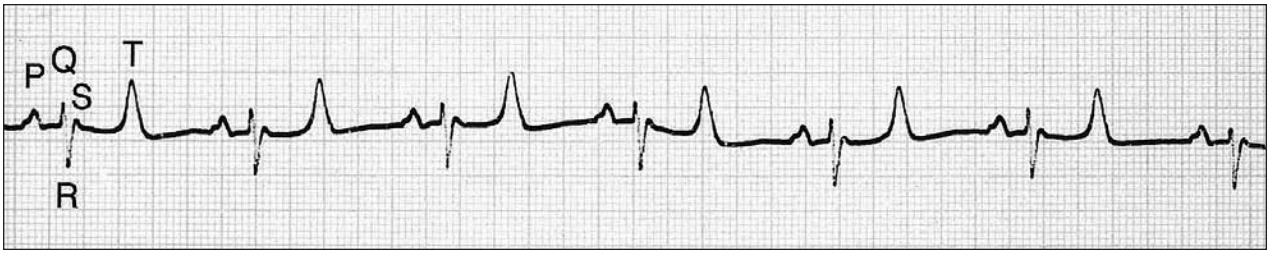

Figure 2—Base-apex ECG from the same horse as Figure 1 after conversion to sinus rhythm with quinidine sulfate. Notice that the deflections of the P wave, QRS complex, and T wave are normal in appearance. Paper speed = 25 mm/s; 5 mm = 1 mV.

aligned relatively parallel to the electrical axis of the heart, providing greater deflection of the waveforms generated, compared with standard lead configurations.[1] The base-apex lead is performed by placing the positive electrode over the left apex of the heart and the negative electrode over the base of the heart. The ground electrode may be attached anywhere on the horse. A common method of obtaining this lead configuration is to place the left forelimb electrode over the left apex by attaching the electrode to the thoracic wall at the sixth intercostal space just behind the point of the elbow joint. The right forelimb electrode is placed over the jugular furrow approximately two thirds of the way down the neck or at the top of the spine of the right scapula. Recording is on lead I.[2]

Spontaneous depolarization is a property of select cardiac tissues, most prominently in the sinoatrial node, but also found within the atrioventricular node and Purkinje fibers. Spontaneous activity is the result of a natural slow inward flux of sodium and a time-related decrease in the outward flux of potassium. Ectopic pacemakers develop when drugs, inflammation, ischemia, or other factors impair the ability of myocyte membranes to exclude sodium, thus, allowing an increase in ion flux and the generation of an action potential.[2]

Because of fairly complete penetration into the free walls of the ventricles by the conduction system, these chambers tend to be activated simultaneously with a burst of depolarization that cancels out much of the divergent electromotive forces, resulting in small QRS complex amplitudes.[2] The VPC are characterized by a change from the dominant form of QRST deflections and are not preceded by a true premature P wave.[3-5] An ectopic focus in the ventricles may originate anywhere within the ventricular myocardium. These foci generally do not possess inherent rhythmicity and develop irregularly and unpredictably. Activation generally occurs within muscle fibers and is later picked up by neighboring Purkinje pathways so that electrical activity is bizarre, compared with QRS complexes that are normal in appearance, with the degree of aberration depending on the site of the ectopic focus relative to the nearest unaffected conduction pathways. Because these QRS complexes tend to start in 1 ventricle and spread to the other rather than spreading to both simultaneously, as in the clinically normal situation, the aberrant complexes tend to express much larger electrical amplitudes and longer conduction times.[6] If the ectopic focus is in the left ventricle with electrical activity spreading from the left to the right, the pattern the QRS complex will have is a predominantly negative deflection. Those originating from the right ventricle will have a QRS complex with a predominantly positive deflection when evaluated with lead I.[7] Polyphasic QRS complexes probably arise from within the interventricular septum. Multifocal VPC are evident when the damage to the ventricles occurs in multiple areas and are expressed as a variety of configurations of the QRS complex, whereas focal VPC are evident when the source of irritation is from the same myocardial source and are expressed as ectopic QRS complexes of similar configuration.[8]

Ventricular tachycardia develops when 4 or more sustained ventricular extrasystoles occur at a rapid rate.[9,10] Ventricular tachycardia is grouped into paroxysmal (short term) or persistent (long term). Ventricular tachycardia may lead to poor ventricular filling, weak or variable arterial pulses, poor systemic perfusion, and cardiovascular collapse.[2] The persistent type of ventricular tachycardia is most serious because of its inherent ability to proceed into ventricular fibrillation, a terminal event.

Ventricular tachycardia and VPC often indicate myocardial damage and can result from septicemia, streptococcal infection, neoplasia, uremia, toxemia, or drugs such as inhalant anesthetics and digoxin.[9,11] The inciting factor for VPC in the horse of our report is believed to be a rattlesnake bite. This is supported by the symmetrical puncture wounds and inflammation on the horse's face, the fact that the insult occurred during a summer month in a rattlesnake endemic area, and the disappearance of the VPC coinciding with the resolution of the facial, cervical neck, and pectoral swelling. From the reported geographic distribution of venomous snakes, it is assumed that this horse was bitten by a *Crotalus viridis, a* prairie rattlesnake.[12]

Snake venom is an extremely complex mixture of enzymes, proteins, and peptides. To date, at least 26 enzymes have been characterized. Ten of these enzymes appear to be common to all venoms studied, but the venom from different families of snakes contain different concentrations of these enzymes.[13] Cardiac abnormalities are commonly associated with crotalid envenomation as acute and chronic sequelae. In a recent report of rattlesnake venom poisoning in horses, 56% of acutely affected horses had tachycardia, and 15% had cardiac arrhythmias. Sixteen percent had debilitating cardiac dysfunction lasting several weeks to several months, resulting in euthanasia in several instances. Reported abnormalities included tachycardia, severe dysrhythmias, myocardial necrosis and fibrosis, and cardiac dilation, although ventricular arrhythmias were not specifically cited.[14] Experimental crotalid envenomation in cats produces similar cardiac lesions as natural envenomation in horses. This includes extensive subendocardial and myocardial hemorrhage and ischemia, which result in chronic lesions resembling those of myocardial infarction.[13,15] Several interacting mechanisms appear responsible for the cardiac dysfunction and lesions seen. Cardiac collapse has been proposed to be secondary to decreased coronary perfusion caused by disruption of capillary basal lamina and endothelial damage produced by crotalid venom.[13] Depression of myocardial contractility caused by a myocardial depressant factor was the proposed mechanism for the profound decrease in cardiac output and hypotension induced with envenomation by the Brown Snake (*Pseudonaja* spp) of Australia after IV administration of venom in dogs. The hypotension could not be attributed to vasodilation, as peripheral vascular resistance increased after envenomation. In addition, there was no evidence that depression of cardiac output resulted from an anaphylactoid reaction.[16] Coronary vasospasm, resulting in myocardial ischemia, was documented in mice injected with *Atractaspis*

engaddensis venom, causing severe decreases in cardiac output and several conduction abnormalities including supraventricular and ventricular premature contractions. No effect was seen when the venom was administered directly to cardiac muscle preparations.[17]

Antibiotics are indicated in the treatment of poisonous snakebites, as they often lead to local infections and tissue necrosis.[15,18] Severe decreases in cardiac output resulting in shock are common with snake envenomation requiring IV fluid support.[15,18] The use of corticosteroids is somewhat controversial, but initial use to help stabilize capillary membranes to slow or prevent further fluid leakage from the vascular compartment may be beneficial. However, continued use may be contraindicated because of the risk of infection at the site of the wound and is recommended only if required to keep the patient systemically stable.[18] Follow-up treatment with nonsteroidal anti-inflammatory drugs is indicated. Tetanus prophylaxis should be addressed. The use of antivenin is also controversial, and timing may be critical. Delays of as little as 1 hour in administration may result in a substantial decrease in efficacy. No difference in survival or tissue necrosis occurred between treated and control rats if treatment with antivenin was delayed more than 4 hours.[15]

Specific antiarrhythmic treatment is indicated for those horses developing hemodynamically important arrhythmias. Quinidine and lidocaine hydrochloride are the most commonly used drugs for the treatment of ventricular arrhythmias in horses.[11] Quinidine blocks sodium current and multiple cardiac potassium currents by blocking open-state sodium channels and potassium channels, resulting in an increase in the threshold for excitability, a prolongation of the action potential, and a prolongation of the refractory period.[19] Quinidine tends to spare the sinoatrial node while suppressing ectopic pacemakers, thus, allowing the sinoatrial node to resume its normal function in controlling heart rhythm.[20] Because quinidine also produces α-receptor blockade, potentially resulting in hypotension, any dehydration deficits should also be addressed.[19]

[a]Cardiopet Inc, Little Falls, NJ.

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