ECG of the Month

This feature is being sponsored by the Academy of Veterinary Cardiology. Readers of the JAVMA are invited to submit contributions. Contributions should include: a brief description of the case (150 words); good contrast glossy photographs (5 X 7 in) of tracings, with ECG lead, voltage calibration scale, and paper speed indicated; and a discussion of the abnormality.

Send comments and tracings to Dr. Phillip Ogburn, Department of Small Animal Clinical Sciences, College of Veterinary Medicine, University of Minnesota, 1352 Boyd Ave, St Paul, MN 55108, or Dr. John-Karl Goodwin, Department of Veterinary Clinical Science, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA 70803-8410.

A 2-month-old 80-kg Holstein heifer was anesthetized for umbilical hernia repair. Results of preoperative physical examination, CBC, and serum biochemical analysis were within reference limits. Anesthesia was induced with 6 mg of xylazine, 120 mg of ketamine, and 3 g of guaifenesin, combined and administered IV. Following orotracheal intubation, a mixture of 2.0% halothane and oxygen (flow, 3 L/min) was administered through an out-of-circle vaporizer with a large-animal anesthetic machine to maintain anesthesia; the calf breathed spontaneously. A base/apex ECG was recorded and appeared normal. Heart rate was 90 beats/min. Indirect mean arterial blood pressure was 90 mm of Hg.

Twenty minutes after initiating halothane administration and prior to surgical incision, the heart rate had decreased to 70 beats/min and an arrhythmia was observed (Fig 1). Mean arterial blood pressure was 83 mm of Hg.

ECG Interpretation and Discussion

The first complex of the ECG appeared to be conducted normally through the heart following initiation at the sinoatrial (SA) node; however, the PR interval of 140 ms was shorter than the reference PR interval for cattle (170 to 250 ms; Fig 1). The first complex, therefore, more likely represented separate impulses from the SA node and from a focus in or near the atrioventricular (AV) node. The ectopic focus was presumed to have been junctional because QRS and T complexes were normal in appearance and duration, whereas impulses originating in the ventricles cause bizarre and prolonged QRS complexes and large inverted T waves. The SA node discharged only slightly before the AV node in the second complex, resulting in partial superimposition of the P wave on the QRS complex. In the third complex, the AV node discharged before the SA node, and the atrial depolarization wave (P wave) occurred immediately before the ventricular repolarization wave (T wave). The impulse initiated by the SA node was transmitted to the ventricles, producing the fourth QRS complex. This complex was termed a “capture beat” because the SA node “captured” control of the ventricles. The PR interval of the capture beat was slightly prolonged (280 ms), probably because of partial refractoriness of the AV node. The P wave before the capture beat might have been confused with sinus depolarization caused by retrograde conduction. However, as evidenced by regularity of the PP interval, a sinus beat was expected here and retrograde conduction did not seem to have occurred.

The arrhythmia was repeated until the seventh QRS complex. Here again, the P wave immediately preceded the T wave, but in this case, the impulse initiated by the SA node occurred 20 ms sooner than the SA node impulse that followed the third QRS complex, contacted a refractory AV node, and did not appear to be transmitted. However, prolongation of the associated RR interval (similar to that observed following the fourth QRS complex) indicated concealed conduction. This occurs when an impulse is conducted into the AV node, making the node refractory, but not initiating ventricular depolarization and thereby concealing the impulse.

The arrhythmia may be better understood by simultaneously viewing the ECG and a Lewis diagram (Fig 1). Lewis diagrams pictorially represent impulse conduction through the heart. The 3 large spaces divided by horizontal lines represent the SA node and atria, the AV node, and the ventricles, respectively, from top to bottom. Impulse initiation at sites other than the SA node is indicated by a dot and transmission is indicated by vertical lines, the angle of which describe the speed of transmission. Small horizontal lines indicate transmission blockade. In the Lewis diagram from this calf, SA and AV node depolarization was regular. Slowed AV node conduction during the capture beat was evident in the decreased slope of the line through the AV node.

This arrhythmia is AV dissociation, because the competing pacemaker sites in the SA and AV nodes discharge independently. However, this calf had incomplete AV dissociation because capture beats were evident. Complete AV dissociation is characterized by atrial complexes that are never transmitted to the ventricles.1 The arrhythmia in this calf also could have been termed AV dissociation with interference, because impulses from the SA node were intermittently conducted and interfered with impulses initiated by the AV node.2 Atrioventricular dissociation can result from decreased SA node automaticity (suspected in this heifer), dis-

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ruption of AV node conduction, or enhanced AV node or ventricular automaticity. Atroventricular dissociation is differentiated from third-degree AV block in that AV block refers strictly to impaired conduction through the AV node. Third-degree AV block is usually characterized by an atrial rate much greater than the ventricular rate.

We did not attribute this arrhythmia to xylazine treatment because the rhythm disturbance was not detected immediately after xylazine administration. However, the half-life of xylazine in calves is 36 minutes, and this arrhythmia could have been xylazine-mediated. Xylazine, an $\alpha_2$-adrenergic agonist, has been shown to cause SA block, first- and second-degree AV block, AV dissociation, and sinus arrhythmias in various species. Although most xylazine-induced arrhythmias are vagally mediated, diminished sympathetic tone also is found following $\alpha_2$-adrenergic agonist administration, secondary to presynaptic stimulation. Because they are not always vagally mediated, arrhythmias induced by $\alpha_2$-adrenergic agents are not always responsive to anticholinergic administration.

Because the arrhythmia was temporally most closely associated with halothane administration, a halothane-induced arrhythmia was suspected. Dysrhythmias most frequently associated with halothane anesthesia include bigeminy, sinus bradycardia, wandering pacemaker, junctional rhythms, and nodal and ventricular premature contractions. Halothane can directly slow the rate of SA node discharge, by slowing the rate of SA node depolarization (phase 4) and by increasing the threshold for the generation of action potentials, thereby decreasing automaticity. Indirect halothane-mediated SA node depression seems to be mediated primarily through a decrease in sympathetic nervous system tone, allowing unopposed vagal tone to predominate. If the SA node discharge rate is slowed to less than that of other cardiac pacemakers, the AV node may take control of cardiac rhythm and probably was the initiating foci for the arrhythmia in this calf. Although vagally mediated arrhythmias are common, halothane does not consistently affect parasympathetic nervous activity.

Halothane also induces arrhythmias by sensitizing the myocardium to increased plasma catecholamine concentrations, leading to propagated impulses from ectopic sites within the atria or ventricles. Although exogenous catecholamines (isoproterenol, dopamine) were not administered to this calf, endogenous catecholamines can be released in various circumstances. Circulating catecholamine concentrations often increase following induction of anesthesia. Surgical stimulation has been shown to cause endogenous release of catecholamines; however, in this calf, arrhythmias appeared prior to incision. Concentrations of epinephrine and norepinephrine increase following ketamine administration. Hypercapnia also can cause increased circulating catecholamine concentrations. In halothane-anesthetized horses, hypercapnia has been shown to increase the likelihood of arrhythmias secondary to catecholamine administration. Blood gas concentrations, however, were not monitored in this calf. In addition to circulating catecholamines, abnormal serum electrolyte concentrations also can precipitate arrhythmias. In this calf, however, intraoperative serum biochemical evaluation did not reveal electrolyte abnormalities.

Whereas excessive halothane concentrations can increase sensitivity to circulating catecholamines, inadequate halothane concentrations also can cause arrhythmias by allowing sympathetic nervous system tone to increase. Appropriate concentrations of halothane decrease sympathetic nervous system activity and reduce catecholamine release from adrenergic nerve endings and from the adrenal medulla in some species. Therefore, although the general recommendation is to decrease the inspired concentration of halothane in response to halothane-induced arrhythmias, increasing
the concentration might be more prudent in some instances.15

Although AV dissociation during anesthesia is usually benign, to ensure that adequate cardiac output was maintained, atropine was administered (0.04 mg/kg of body weight, IV) over 1 to 2 seconds, without response. Lack of response to atropine probably indicated that the arrhythmia was not vagal mediated, as discussed previously. Because the calf had been breathing 2.0% halothane for 30 minutes, adequate anesthetic depth was presumed and inspired halothane concentration was decreased. Within 20 minutes of decreasing the vaporizer setting to 1.5%, the arrhythmia was abolished and a sinus rhythm of 90 beats/min was observed. However, 10 minutes after the arrhythmia disappeared, the calf began to move in response to surgical stimulation, and a vaporizer setting of 2.0% was again established. The sinus rate slowed and the arrhythmia returned within 20 minutes of increasing the vaporizer setting. At that point, the procedure was nearing completion, and further treatment was not initiated. Mean arterial blood pressure did not change substantially at any time during anesthesia.

After decreasing halothane concentrations, the rate of sinus node impulse initiation increased, thereby reestablishing the sinus node as the predominant cardiac pacemaker. If arrhythmias had coincided with the onset of surgery, increasing the halothane concentration might have been more prudent. If the arrhythmia had been deemed life-threatening, isoflurane anesthesia could have been substituted, because isoflurane is less arrhythmogenic than halothane and does not sensitize the myocardium to catecholamines.16 Although cardiac output was not measured directly, maintenance of adequate perfusion pressure (as measured by mean arterial pressure) led us to assume that output was not compromised. If cardiac output had been considered to be inadequate, other chronotropic drugs (eg, isoproterenol) could have been used if changing anesthetic depth had been ineffective.

The calf recovered without complications. Review of an ECG obtained 1 hour after recovery from anesthesi revealed normal sinus rhythm, with a rate of 100 beats/min.

References