Anesthesia Case of the Month

In cooperation with the American College of Veterinary Anesthesiologists

History

A 6-year-old 477-kg (1,049-lb) Andalusian-bred mare was referred to the Equine Teaching Hospital of Barcelona for surgical repair of a complicated bilateral mandibular fracture. The mare was 8 months pregnant and had no history of heart disease.

A fast (80 beats/min), irregularly irregular cardiac rhythm was detected during the preanesthetic examination, and results of subsequent electrocardiography were consistent with atrial fibrillation (**Figure 1**). Despite the absence of clinical signs of heart failure, echocardiography was performed, although a complete ultrasonographic examination could not be performed because the mare was fat. The fetal heart rate, determined by means of transabdominal ultrasonography, was 100 to 110 beats/min.

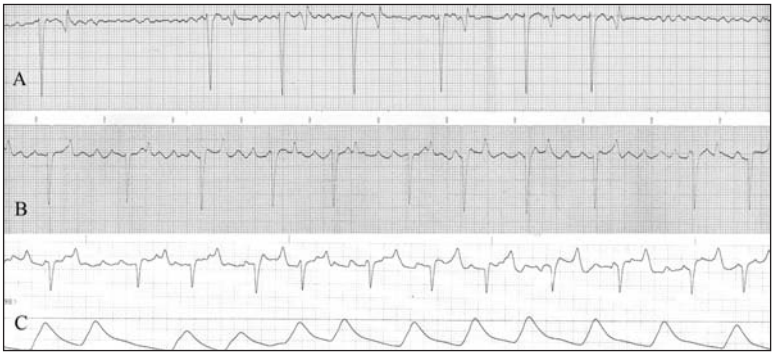

Figure 1—Electrocardiographic recordings obtained from a pregnant 6-year-old mare at the time of admission for treatment of a mandibular fracture (A), following administration of 4 doses of quinidine sulphate (B), and during general anesthesia (C; arterial blood pressure is also shown). Notice the regularly irregular cardiac rhythm with a high rate of P waves (200 P waves/min) and atrioventricular block following administration of quinidine. Base-apex lead; paper speed = 25 mm/s; 1 cm = 1 mV.

Mild neutrophilia and subclinical potassium deficiency (potassium excretion fraction, 15.8%; reference range, 23% to 48%) were detected on analysis of blood and urine samples. The PCV was 33%, and the total plasma protein concentration was 7.2 g/dL. The potassium deficiency was corrected in 2 days by nasogastric administration of potassium chloride (30 g, q 12 h). However, the appearance of the ECG remained unchanged. Therefore, treatment with quinidine sulphate (22 mg/kg [10 mg/lb], q 2 h, administered through a nasogastric tube) was initiated. After the third dose, the mare began to show signs of mild abdominal discomfort and soft feces, and the fourth dose was delayed for 4 hours. Six hours later, electrocardiography revealed a regularly irregular, supraventricular escape rhythm (cardiac rate, 60 beats/min) with blocked P waves (200 P waves/min). Surgery was performed the same day.

Question

What would be a suitable choice of anesthesia for this pregnant mare?

Answer

Atrial fibrillation is a common arrhythmia in horses that causes ventricular contraction disturbances and decreases ventricular filling and cardiac output during anesthesia.[1] Positioning mares that are in an advanced stage of pregnancy in dorsal recumbency can also decrease cardiac output, as the weight of the gravid uterus can cause compression of the caudal vena cava and aorta, decreasing venous return.[2]

For these reasons, phenothiazine and α_2-adreonoceptor agonists, tranquilizers, and sedatives that are commonly used in horses were avoided because of their cardiovascular depressant effects. Instead, the mare was premedicated with butorphanol (0.04 mg/kg [0.018 mg/lb], IV), and 5% guaifenesin was administered IV until a relaxant effect was observed. An IV bolus of propofol (1 mg/kg [0.45 mg/lb]), ketamine (1 mg/kg), and midazolam (0.025 mg/kg [0.011 mg/lb]) was then used for anesthetic induction. Anesthetic induction was smooth without any complications.

An 18-mm nasotracheal cuffed tube was placed to allow a suitable approach for surgery, and the mare was positioned in dorsal recumbency on the surgery table. Anesthesia was maintained for 3 hours with isoflurane, which was delivered through a semiclosed, circle, large animal anesthetic circuit that incorporated an out-of-circle precision vaporizer. Mechanical ventilation was performed with a large animal ventilator.[a] The vaporizer was set to deliver isoflurane at a concentration of 2.5%; the oxygen flow was 6 L/min. Bilateral perineural mandibular blocks were performed to provide analgesia. A 22-gauge spinal needle was inserted 3 inches from the caudomedial edge of the mandible and directed cranially to the mandibular foramen, and 10 mL of 2% mepivacaine was administered. Lactated Ringer's solution was infused IV at a rate of 8 mL/kg/h (3.6 mL/lb/h).

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Arterial blood pressure, end-tidal partial pressure of carbon dioxide, arterial saturation of hemoglobin with oxygen, and an ECG were continuously monitored[b] during anesthesia. An arterial blood sample was collected 60 minutes after the beginning of the anesthetic procedure and submitted for blood gas analysis and determination of electrolyte concentrations; results were within reference limits. Mean arterial blood pressure ranged from 68 to 90 mm Hg; inotropic treatment was not required.

During anesthesia, electrocardiography revealed a progressive decrease in the rate of P waves to 130 P waves/min, although the supraventricular escape rhythm was unchanged. At the end of anesthesia, butorphanol (0.03 mg/kg [0.014 mg/lb], IV) was administered. Recovery was calm, and the mare stood up on the first attempt.

Atrial fibrillation was evident during electrocardiography performed 24 hours after surgery and during ECG monitoring performed during the subsequent 30 days of hospitalization. No new attempts to convert the cardiac rhythm to a normal sinus rhythm were undertaken. Well-being of the fetus was assessed several times during hospitalization, and no alterations were detected.

Discussion

Horses are predisposed to develop atrial fibrillation because of their high vagal tone and large atria. Atrial fibrillation is the most common arrhythmia and most common cardiovascular cause of poor performance in horses, with some affected horses showing signs of exercise intolerance, exercise-induced epistaxis, respiratory dysfunction, weakness, or syncope. However, atrial fibrillation is usually an incidental finding that does not cause major hemodynamic changes in resting horses without underlying cardiac abnormalities.[3] On the other hand, decreases in cardiac output and mean arterial blood pressure have been reported during general anesthesia of horses with atrial fibrillation. Thus, it is recommended to treat the condition before anesthesia.[1]

Because hypokalemia may predispose horses to develop atrial fibrillation, measurement of fractional excretion of electrolytes is recommended to detect electrolyte imbalances.[4] As was the case for the horse described in the present report, electrolyte abnormalities should be corrected prior to attempting to treat the atrial fibrillation. New and promising therapeutic protocols are being developed to treat atrial fibrillation; however, quinidine remains the drug of choice for conversion of atrial fibrillation to sinus rhythm, despite its potential adverse effects, and its use is indicated when there are no signs of severe underlying cardiac disease.[3,5] In a previous report,[6] oral administration of quinidine to a pregnant mare with atrial fibrillation did not result in any teratogenic or abortogenic effects. In the case described in the present report, treatment with quinidine resulted in supraventricular tachycardia. This is a common adverse effect of quinidine treatment caused by a sudden decrease in vagal tone at the atrioventricular node.[3]

Standard anesthetic procedures, which include administration of α_2-adrenoceptor agonists or phenothiazine, have been used in horses with atrial fibrillation. However, use of these sedatives and tranquilizers is not recommended in compromised patients because of their potential hemodynamic depressant effects. Hypotension is the most common adverse effect of phenothiazine, whereas bradyarrhythmias and decreases in cardiac output are potential adverse effects of α_2-adrenoceptor agonists.[4] Although administration of acepromazine can provide a beneficial antiarrhythmic effect in healthy horses, the potential for acepromazine to cause hypotension was considered too much of a risk to allow use of this drug in the mare described in the present report.

Butorphanol and guaifenesin were used to premedicate the mare described in the present report because they cause minimal cardiovascular depression and there are no contraindications to their use in pregnant mares.[2] When administered alone, butorphanol can have a mild excitatory effect in horses, but this was ameliorated by the immediate administration of guaifenesin.

Induction of anesthesia can have deleterious effects on cardiac function. Some anesthetics (eg, thiobarbiturates and halothane) can sensitize the myocardium to catecholamine-induced arrhythmias.[7] The effects of various anesthetics on atrial conduction has been investigated. Propofol has been shown to be effective in protecting the heart from excessive ventricular rates and is suggested to be an appropriate anesthetic in patients with supraventricular tachyarrhythmias.[8]

Doses of propofol currently used to induce anesthesia in adult horses may cause a decrease in systemic vascular resistance, arterial hypotension, respiratory depression, and unpredictable anesthetic induction, even when the horses have been premedicated with α_2-adrenoceptor agonists.[9] Addition of guaifenesin to the anesthetic protocol improves induction quality.[10]

In human beings, the combination of propofol and ketamine has been reported to preserve hemodynamic stability and respiratory function when used to induce and maintain anesthesia and to decrease the need for inotropic agents.[11] This has important implications for horses with atrial fibrillation because the use of catecholamines to treat hypotension may increase atrioventricular node conduction, leading to an irregular ventricular response with inadequate cardiac filling time.[12] Anesthetic induction protocols that involve various combinations of propofol and ketamine have been evaluated in horses with good results.[13] To our knowledge, however, their use for anesthetic induction in horses without prior administration of an α_2-adrenoceptor agonist has not been reported previously. In the horse described in the present report, midazolam was added to the propofol-ketamine combination in an attempt to decrease individual drug doses and achieve a good-quality induction.

Maintenance of anesthesia with volatile agents can have a variety of effects on the myocardium. Isoflurane has been shown to have antifibrillatory effects on atrial tissues in dogs and to cause temporary conversion of chronic atrial fibrillation to sinus rhythm during anes-

thesia.[14] Sinus rhythm was not apparent in the mare described in the present report, possibly because of the vagolytic effects of quinidine.

The propofol-ketamine-midazolam mixture used in the mare described in the present report allowed good induction and recovery quality with minimal sedation, caused minimal cardiovascular and respiratory depression, and did not affect fetal well-being. This combination could be beneficial in similar cases in which more depressant sedatives and anesthetics are contraindicated.

a. Model 2800, Mallard Medical, Irvine, Calif.
b. Model V24C, Agilent Technologies, Bablingen, Germany.

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