A 20-hour-old Hanoverian colt weighing 50 kg (110 lb) was evaluated because of inability to nurse. The foal was born unobserved to a primiparous mare at 325 days of gestation. Abnormalities identified on physical examination of the foal included congested, dry mucous membranes; moderate dehydration; tachycardia (heart rate, 122 beats/min; reference range, 70 to 100 beats/min); tachypnea (respiratory rate, 56 breaths/min; reference range, 20 to 40 breaths/min); hypothermia (rectal temperature, 36.8°C [98.2°F]; reference range, 37.2°C to 38.9°C [99°F to 102°F]); and mild edema of all 4 limbs. Hematologic and serum biochemical analyses revealed a modest left shift (bands, 600/µL; reference range, 0 to 150 bands/µL), hypoglycemia (37 g/dL; reference range, 121 to 233 g/dL), high serum creatine kinase activity (6,488 U/L; reference range, 40 to 909 U/L), partial failure of passive transfer (serum IgG concentration measured by radi- cal immunodiffusion assay, 632 g/dL; reference limit, >800 g/dL), and selenium deficiency (serum selenium concentration, 0.087 µg/mL; reference range, 0.17 to 0.25 µg/mL). The calculated sepsis score was 14. A blood sample was submitted for bacteriologic culture and antimicrobial susceptibility testing. A consumptive coagulopathy was diagnosed on the basis of prolonged values of prothrombin time (12.9 seconds; reference range, 10.3 to 11.5 seconds), partial thromboplastin time (72.6 seconds; reference range, 50.5 to 53.1 seconds), high serum concentration of fibrin degradation products (>80 but <160 µg/mL; reference range, >10 but <40 µg/mL), and decreased plasma antithrombin III activity at 69% of pooled equine plasma (reference range, 89% to 124%).

Indirect mean arterial blood pressure and arterial blood gas values were within reference limits. Initial ECG analysis revealed a sinus tachycardia. Serum concentrations of cardiac troponin I (0.15 µg/L; reference range, 0.03 to 0.51 µg/L), cardiac troponin T (0.02 µg/L; reference range, 0.01 to 0.032 µg/L), and creatine kinase-myocardial band isoenzyme (CK-MB; 5.1 µg/L; reference range, <1.0 to 9.3 µg/L) were also determined (reference values based on the authors’ experience).

Initial treatment on day 1 of hospitalization (day of admission) included a plasma transfusion (40 mL/kg [18.2 mL/lb], IV) and administration of dextrose (0.1 g/kg [0.045 g/lb], IV), potassium penicillin (22,000 U/kg [10,000 U/lb], IV, q 6 h), amikacin (20 mg/kg [9.1 mg/lb], IV, q 24 h), flunixin meglumine (0.25 mg/kg [0.12 mg/lb], IV, q 8 h), thiamine (10 mg/kg [4.55 mg/lb], IV, q 12 h), and a preparation of selenium and vitamin E (0.03 mg of selenium/kg [0.023 mg/lb] and 1.3 U of vitamin E/kg [0.59 U/lb], IM, once).

On day 2 of hospitalization, an arrhythmia was detected and an ECG strip was recorded (Figure 1). Results of arterial blood gas analysis and indirect mean arterial blood pressure measurement remained within reference limits, as did serum electrolyte and glucose concentrations. No gross morphologic abnormalities were detected via echocardiography. However, a repeated hemostasis profile revealed increased prothrombin time and partial thromboplastin time (14.9 and 97.9 seconds, respectively) and decreased antithrombin III activity at 52.2% of pooled equine plasma, which may have contributed to the generation of microparticulate thrombi that embolized within the myocardium. Because of the frequency of the premature contractions, the foal was administered hypertonic saline (7.2% NaCl) solution (3 mL/kg [1.36 mL/lb], IV, once) followed by quini-
is still refractory, its conduction may be blocked.3 In sinus origin. If the P wave is so early that the AV node at a different point than that targeted by a beat of aberrant conduction may occur if depolarization is selectively refractory to depolarization. Alternatively, ventricular conduction sometimes occurs because the ventricular receptive to being depolarized by an SPC, but aberrantly conducted. The ventricular conduction system is usually nonconducted, and occasional beats had aberrant conduction; its use in horses as an antiarrhythmic agent has been extensively studied, and it was considered the drug of choice. Because the foal’s heart rate was only mildly high, treatment with digitalis to decrease the heart rate was not considered appropriate. The foal was monitored via cardiac auscultation, indirect arterial blood pressure measurements, and ECG. Approximately 18 hours after instituting quinidine treatment, the foal had signs of abdominal discomfort. Ultrasonographic examination of the abdomen was within normal limits, and no reflux was obtained via nasogastric intubation. The ECG findings indicated a decrease in the frequency of premature contractions. The signs of abdominal pain were alleviated following administration of butorphanol (0.05 mg/kg, IV), and quinidine treatment was discontinued.

The foal remained hospitalized and was treated for presumed sepsis for 8 days. During this period, the frequency of premature contractions decreased to < 1/min at times of rest but increased with excitement and exercise. The clotting times, plasma antithrombin III activity, and fibrin degradation products concentrations were all within reference values for age-matched foals, and the frequency of supraventricular premature contractions (SPCs) was < 1/min at discharge on day 8. The arrhythmia had resolved at a recheck ECG examination performed 6 months after discharge.

**ECG Interpretation**

On the initial ECG tracing, a sinus rhythm with supraventricular premature complexes occurring in 33% of the beats at a ventricular rate of 120 beats/min and an atrial rate of 140 beats/min was identified. Atrial trigeminy with SPCs following 2 normal cardiac cycles was also evident. Most of the atrial premature beats were nonconducted, and occasional beats had aberrant conduction (Figure 1). The differently shaped but upright P waves indicated depolarization originating from a supraventricular focus outside the sinus node. Supraventricular premature contractions may be conducted normally or with aberrancy or may be nonconducted. The supraventricular conduction system is usually receptive to being depolarized by an SPC, but aberrant conduction sometimes occurs because the ventricular muscle fibers are not completely repolarized and therefore are selectively refractory to depolarization. Alternatively, aberrant conduction may occur if depolarization is triggered by a supraventricular beat that enters the AV node at a different point than that targeted by a beat of sinus origin. If the P wave is so early that the AV node is still refractory, its conduction may be blocked.4 In the foal of this report, no compensatory pause was observed following the SPCs; the P-P interval was prolonged as a result of resetting of the sinoatrial node (in which the SPC enters the sinoatrial node and interrupts its timing).

**Discussion**

A variety of cardiac arrhythmias can occur in neonates during the transitional phase from intrauterine to extrauterine life. Newborn foals may be predisposed to supraventricular arrhythmias caused by increased vagal tone immediately after birth.5 Increased vagal tone shortens the action potential duration in atrial myocardial cells, thereby permitting development of supraventricular arrhythmias. The onset of breathing at birth results in decreased pulmonary vascular resistance, and ligation of the umbilicus results in increased systemic vascular resistance. These changes in vascular resistance lead to increased pulmonary blood flow and left atrial pressure. Alternatively, diastolic tension of the atrial myocardium may lead to changes in conduction and, consequently, development of supraventricular arrhythmias. In overtly healthy newborn foals, transient myocardial hypoxia during adaptation to extrauterine life may be detected as elevated ST segments on ECG tracings.4,6 Most arrhythmias resolve within the first few minutes to hours after birth without requiring treatment; the normal growth and development of most foals provide evidence that major myocardial lesions are unlikely.6

At the time of initial examination, the foal of this report was 20 hours old; an arrhythmia was not detected before day 2 of hospitalization, and that arrhythmia was more pronounced with exercise and excitement. These findings suggested that the arrhythmia was unlikely to be a result of excessive vagal tone or normal neonatal adjustment to extrauterine life. Serum electrolyte abnormalities that could cause changes in myocardial membrane potential and refractory period were ruled out on the basis of serum electrolyte concentrations.

Reversible myocardial dysfunction is a well-recognized syndrome in adult humans and pediatric patients without cardiac disease.7 Recently, this dysfunction has been reported frequently in humans with sepsis or sepsis syndrome.5 Although the potential mechanisms underlying the etiopathogenesis of this phenomenon are numerous, direct ischemic injury to the heart associated with infarction or inflammatory mediators is considered highly likely in humans with systemic inflammatory response syndrome, sepsis, and septic shock who also have hypercoagulation and increased serum concentrations of the cardiac enzymes troponin I and T.8 In reversible myocardial dysfunction, ECG changes that accompany increases in cardiac enzymes include ST segment deviation. The notion that myocardial injury can result from altered hemostasis is further supported by data that link increased plasma concentration of fibrin degradation products with cerebral and pulmonary infarction and ECG changes.9 The fact that the period of ECG changes for the foal of this report is correlated with progressive worsening of the consumptive coagulopathy supports this notion and warrants further investigation.

Myocardial damage resulting from selenium deficiency and myodegeneration was a concern in the foal because of the high serum creatine kinase activity and low serum selenium concentration detected initially. Selenium deficiency can lead to myodegeneration of both skeletal and cardiac muscle.4 Selenium was administered IM to the foal on day 1 of hospitalization; within 3 days of hospitalization, serum creatine kinase activity decreased to within reference limits, which
decreased the likelihood of selenium deficiency as a cause of myocardial degeneration and arrhythmia.

Reference values for serum cardiac troponin I concentration in adult horses have been published; compared with those values (0 to 0.35 ng/mL), the foal of this report had greatly increased serum cardiac troponin I concentration. To the authors’ knowledge, serum concentrations of cardiac troponin I, cardiac troponin T, or CK-MB in healthy foals have not been published. However, we recently determined serum cardiac troponin I concentrations in healthy foals < 36 hours old (n = 19); the values ranged from 0.03 to 0.51 μg/L. Furthermore, in foals < 14 days old (n = 32), serum concentrations of cardiac troponin T and CK-MB ranged from 0.01 to 0.032 μg/L and < 1.0 to 9.3 μg/L, respectively. On the basis of these results, serum concentrations of cardiac troponin T and I and CK-MB determined initially for this foal were similar to those of age-matched healthy foals. It is possible that the values in the foal of this report would have been high if analyses had been performed at the onset of arrhythmia.

Birth trauma can cause thoracic injury. In dogs, ventricular tachycardia is a common post-traumatic complication that usually develops 24 to 48 hours after blunt trauma, and in humans, arrhythmia may result from direct cardiac trauma. Autonomic imbalances resulting in SPCs occur in humans for a variety of reasons, and supraventricular arrhythmias is the most common arrhythmia in infants. In the foal of this report, increased intensity of the arrhythmia in association with excitement and exercise implied involvement of the sympathetic nervous system or an autonomic nervous system imbalance that was possibly caused by thoracic trauma or sepsis and resulted in hypoxic myocardial foci. Pulmonary disease, known to provoke supraventricular arrhythmias in humans, was not evident in the foal. The foal had atrial premature contractions in 33% of the heartbeats. Supraventricular premature contractions are considered benign if they occur with a frequency of < 10% because the contraction of the atria is thought to have little impact on the cardiac output, except at maximal cardiac performance. Because of the frequency of the SPCs in the foal of this report, a decision to administer quinidine was made. Quinidine is a class I antiarrhythmic drug with negative inotropic and positive chronotropic effects that increases concealed conduction through the AV node and prolongs the effective refractory period for the atrial myocardium. At the time of discontinuing quinidine administration, the atrial premature contractions in the foal had decreased to < 1/min and were not considered to have a negative hemodynamic impact.

References