A 10-year-old 61.5-kg (135.3-lb) spayed female Akita was referred to the emergency service at the Queen Mother Hospital for Animals for investigation of lethargy, anorexia, and vomiting of 24 hours’ duration and a single episode of collapse. On initial evaluation, the dog was quiet but alert and responsive. Mucous membranes appeared congested, with normal capillary refill time. Pulses were synchronous but hypodynamic. Heart rate was 180 beats/min, and rhythm was regular. Cardiac auscultation revealed no evidence of murmur or gallop sounds. Respiratory rate was mildly high (32 breaths/min), but respiratory effort appeared normal. Mild abdominal distension with an obvious fluid wave and hepatomegaly were evident via abdominal palpation. Rectal temperature was 38.2°C (100.8°F). Noninvasive systolic blood pressure measurement (determined via Doppler sphygmomanometry) was slightly low (110 mm Hg; reference range, 120 to 180 mm Hg).

The results of a CBC and venous blood gas and electrolyte analyses were unremarkable. Serum biochemical analysis revealed markedly high alanine aminotransferase activity (2,437 U/L; reference range, 13 to 88 U/L) and mildly high ammonia concentration (81 µmol/L; reference range, 0 to 70 µmol/L). The presence of ascites was confirmed ultrasonographically, and a sample of the fluid was collected via abdominocentesis. Results of cytologic examination of the fluid sample were consistent with a protein-rich transudate (protein concentration, 38.6 g/L; WBC count, 0.60 × 10⁹ WBCs/L).

Initial fluid resuscitation resulted in a reduction in heart rate to 84 beats/min and improvement in peripheral pulse quality. At this time, an arrhythmia was evident. Electrocardiography was subsequently performed (Figures 1 and 2).

Twenty-four hours after the initial evaluation, the dog was anesthetized and underwent abdominal ultrasonography. This ultrasonic examination revealed resolution of ascites and hepatomegaly with increased echogenicity of the hepatic parenchyma and mild congestion of the portal and hepatic vasculature. Needle-core biopsy specimens of the liver were obtained for histologic examination, which revealed mild...
degenerative, hyperplastic, and inflammatory changes that were considered nonspecific. Echocardiography revealed normal chamber dimensions and no evidence of ventricular systolic dysfunction that could suggest a cardiac cause of transient ascites and hepatic congestion.

The dog was discharged from hospital 7 days after the initial evaluation. The presumptive diagnosis was hepatopathy of undefined cause. Treatment with s-adenosylmethiononone (18 mg/kg [8.2 mg/lb], PO, q 24 h) and potentiated amoxicillin (18.5 mg/kg [8.4 mg/lb], PO, q 12 h) was initiated. On follow-up examination at the hospital 1 month later, clinical signs were reported to have completely resolved and serum alanine aminotransferase activity was found to be within reference range.

During the 2-month period after the follow-up examination, the dog was reevaluated on 2 subsequent occasions because of recurrence of collapse, sudden-onset vomiting, and transient ascites. Repeated abdominal ultrasonography revealed the presence of thrombi and absence of normal venous flow in the portal vein and hepatic veins, raising suspicion of liver lobe torsion.1,2 This diagnosis was supported by evidence of malpositioning of the left liver lobes, as determined via CT. Because of torsion effects, the left lateral lobe was subsequently excised during exploratory laparotomy.

**ECG Interpretation**

A single-ECG lead II trace recorded after the dog received initial fluid resuscitation revealed an instantaneous heart rate that ranged from 50 to 85 beats/min. Most QRS complexes were narrow (duration, 0.04 seconds; reference upper limit, 0.06 seconds) and had R waves of normal height (1.4 mV; reference upper limit, < 3 mV). Among the complexes, the PR interval and P-wave morphology varied in association with alternation in the R-R interval from 720 to 1,120 milliseconds. Three P-wave morphologies were observed (Figure 1). Frequent QRS complexes of prolonged duration (0.08 seconds; reference upper limit, 0.06 seconds) were evident throughout the recording, with a variable presence of P waves of negative orientation before or buried within them.

Another single-ECG lead II trace (obtained shortly after the first recording) revealed a predominately regular rhythm with a ventricular rate of 120 beats/min (Figure 2). A series of consecutive, monomorphic complexes of prolonged QRS duration (of similar morphology to those in Figure 1) was noted. Occasional capture and fusion beats were evident in the recording. The ECG diagnosis was marked sinus arrhythmia with subsidiary atrial escape complexes and intermittent accelerated idioventricular rhythm (AIVR).

**Discussion**

P-wave morphology and mean electrical axis of the P wave on a surface ECG reflect the origin and direction of atrial activation. The sinoatrial (SA) node of dogs is an elongated structure (up to 5 mm in length),3,4 and variation in the anatomic location of impulse formation within the SA node alone can cause alterations in P-wave morphology.3,6 Current understanding has moved away from the traditional explanation of impulse initiation from a single static site within the SA node in favor of a more dynamic process of atrial activation in dogs. Atrial electrophysiological mapping in dogs has identified a so-called pacemaker complex, allowing impulse initiation from multiple sites within and directly adjacent to the SA node.7 Additionally, further subsidiary atrial pacemaker sites have been identified; in combination, these sites extend over an area that is 10 to 15 times as large as that previously described for the SA node in dogs.8 Impulse formation occurs via the complex interaction between and coordination of primary and subsidiary pacemaker sites and is modulated by changes in sympathetic and parasympathetic tone.9 High parasympathetic (vagal) tone results in increasing the beat-to-beat cycle duration and typically shifting of the site of impulse initiation inferiorly.10 Site of impulse initiation is also associated with fixed PR and R-R intervals (representing time for impulse propagation via the ativoventricular node and rate of discharge, respectively). In the dog of the present report, the negative orientation of the P waves recorded initially suggested inferior-to-superior or left-to-right atrial activation and was associated with a fixed PR interval of 0.10 milliseconds. This could be consistent with an origin of impulse formation from an escape focus within the left atrium with propagation via the Bachmann's bundle or from a subsidiary pacemaker situated caudally within the right atrium and around the coronary sinus ostium.

The complexes with prolonged duration of QRS complexes in the ECG traces for the dog of this report were suggestive of ventricular escape beats (or fusion beats when associated with negative P waves), with a fixed discharge interval of 2,080 milliseconds. The deep and slurred S waves, leading to a net negative deflection in the lead II trace, were consistent with a right bundle branch block morphology. In anesthetized dogs, the finding of deep and slurred S waves has been shown to be a feature of ventricular complexes pace-induced from a subsidiary pacemaker in the left ventricle.4,10 Performance of a 12-lead ECG examination in the dog of the present report may have helped to more precisely localize different sites of impulse formation.

Accelerated idioventricular rhythm is considered a benign and self-limiting rhythm that has various causes, which are classified as cardiac (eg, ischemic heart disease11 or digitalis toxicosis12) or noncardiac (eg, splenic mass or torsion, gastric dilation–volvulus,13 gastrointestinal tract disease, and other abdominal organ disease).14 An AIVR must be differentiated from ventricular tachycardia or subsidiary pacemaker escape rhythms; such differentiation is principally achieved through analysis of the ventricular firing rate. In human medicine, a ventricular tachycardia is defined as any ventricular rate > 120 beats/min.14 No precise upper rate limits for AIVR have been established for dogs, but a range of 100 beats/min15 to 180 beats/min16 has been suggested. Normal ventricular rhythms are accepted to have a rate of discharge < 65 beats/min, even after sympathetic stimulation.16 The exact mechanism of AIVR is not known. It could be caused by abnormal automaticity as a result of delayed afterdepolarizations, reentry mechanisms, or triggered auto-
resultant tissue necrosis. In the dog of the present report, and abdominal disease in the dog of the present study, with arterial and venous thrombosis and spontaneous return to sinus rhythm was documented following surgical removal of the left lateral liver lobe. 

Liver lobe torsion is a rare finding in dogs, with the left lobes reported as being most commonly affected. Torsion results in major disruption to hepatic blood flow, with arterial and venous thrombosis and resultant tissue necrosis. In the dog of the present report, episodes of liver lobe torsion are hypothesized to have been transient because of the lack of supporting evidence of torsion detected during an early ultrasonographic examination. Liver lobe torsion may have resulted in a marked increase in vagal afferent stimulation, but reperfusion injury and ischemic disease following transient liver lobe torsion (leading to release of inflammatory cytokines and oxygen free radicals) and premature atrial or ventricular ectopy cannot be excluded. Echocardiographic examination excluded the presence of atrial distension or neoplastic lesions that could contribute to ectopic atrial firing. Overall, the presence of both atrial and ventricular escape beats and AIVR was considered to be secondary to concurrent abdominal disease in the dog of the present report, and spontaneous return to sinus rhythm was documented following surgical removal of the left lateral liver lobe.

References