

# ECG of the Month

A 4-year-old 1.4-kg (3.1-lb) male Chihuahua was referred for sudden onset of ataxia and stupor. Physical examination revealed a rectal temperature of 37°C (98.6°F) and a respiratory rate of 32 breaths/min. The dog's heart rate was 60 beats/min, and auscultation revealed a pronounced regularly irregular rhythm, no murmurs, and crackles bilaterally in the dorsal aspects of the lung fields. Tetraparesis was evident, and reflexes in all limbs were diminished; ataxia was detectable during ambulation. Cranial nerve function was intact but a delayed menace response was detected in each eye. Abnormal findings of a CBC included leukocytosis, neutrophilia, a left shift, a moderate number of neutrophils with toxic changes, and thrombocytosis. Serum biochemical analyses revealed severe hypoglycemia; high activities of alanine aminotransferase, alkaline phosphatase, and aspartate aminotransferase; mild electrolyte derangements, including hypochloremia, hypocalcemia, hypokalemia, and hypomagnesemia; and mildly low creatinine concentration. Results of urinalysis were within reference limits. Serologic assessments of the dog yielded negative results for *Dirofilaria immitis* antigen and antibodies against *Borrelia burgdorferi*, *Ehrlichia canis*, *Toxoplasma gondii* (IgG and IgA), *Neospora caninum* (IgG), and *Rickettsia risticii* (IgG). Thoracic radiography revealed moderate to marked alveolar infiltrate in the caudal portions of the lung lobes. Treatment received prior to echocardiography included dexamethasone (0.2 mg/kg [0.09 mg/lb], IV, q 8 h), amoxicillin-clavulanic acid (22 mg/kg [10 mg/lb], PO, q 12 h), and lactated Ringer's solution (7 mL/kg/h [3.18 mL/lb/h], IV). Echocardiography-

ically, no abnormalities were detected in cardiac chamber sizes or systolic function; blood flow across all valves and outflow tracts was apparently normal, without evidence of valve insufficiency or obstruction. Electrocardiography was also performed.

Following the cardiac evaluation, the dog was anesthetized and magnetic resonance imaging (with administration of gadolinium [0.2 mg/kg, IV, once]) and collection of a sample of CSF were performed. Magnetic resonance imaging revealed uptake of contrast agent in the left hippocampal gyrus, and CSF analysis revealed mildly high protein content. The dog was discharged from the hospital 24 hours after the initial evaluation; the presumptive diagnosis was encephalitis. The prescribed treatment included clindamycin hydrochloride (9 mg/kg [4.09 mg/lb], PO, q 12 h), doxycycline calcium (10 mg/kg [4.5 mg/lb], PO, q 24 h), and prednisone (0.7 mg/kg [0.32 mg/lb], PO, q 12 h).

## ECG Interpretation

The initial ECG findings were indicative of an exaggerated sinus arrhythmia and wandering pacemaker (Figure 1). The R-wave amplitude varied; smaller complexes (amplitude, 1.0 to 1.3 mV) occurred during periods of faster heart rate, and taller complexes (amplitude, 1.4 to 1.6 mV) occurred during periods of slower heart rate. Although R-wave amplitude varied, the height of each wave was within the reference range (upper limit, 2.5 mV<sup>1</sup>); the QRS complexes were of normal duration (upper reference limit, 0.06 seconds<sup>1</sup>) and morphology. The slower intrinsic rates were followed by a taller R wave, and the faster intrinsic rates were followed by a smaller R wave. A wandering pacemaker was detected, and P-wave amplitude varied from 0.15 to 0.4 mV (upper reference limit, 0.4 mV<sup>1</sup>). Taller P

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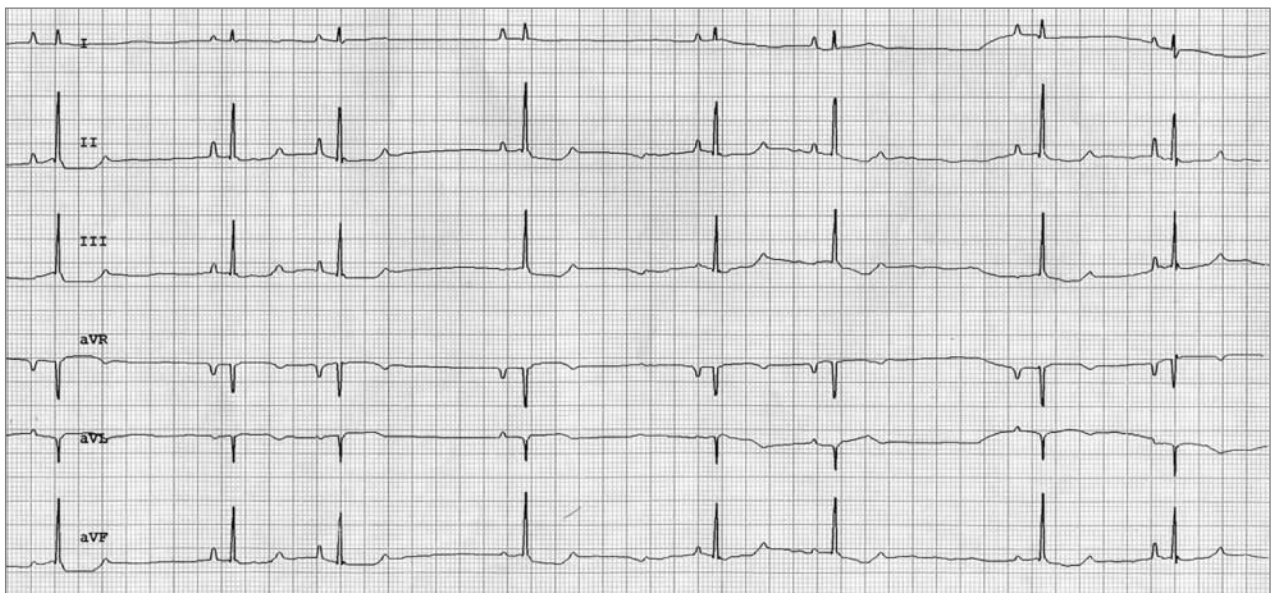


Figure 1—Initial 6-lead ECG recording obtained from a Chihuahua that was evaluated because of sudden onset of ataxia and tetraparesis. An exaggerated sinus arrhythmia, wandering pacemaker, and variation in R-wave amplitude are evident. Notice that R-wave amplitude varies with smaller complexes (amplitude, 1.0 to 1.3 mV) that occur during periods of faster heart rate and taller complexes (amplitude, 1.4 to 1.6 mV) that occur during periods of slower heart rate. These findings are consistent with the Brody effect. Paper speed 50 mm/s; 1 cm = 1 mV.



Figure 2—Six-lead ECG recording obtained 30 minutes after SC administration of atropine (0.04 mg/kg [0.02 mg/lb]) to the dog in Figure 1. Notice that sinus tachycardia (heart rate, 190 beats/min) is present and the wandering pacemaker and Brody effect are abolished. Paper speed 50 mm/s; 1 cm = 1 mV.

waves (amplitude as much as 0.4 mV) preceded smaller QRS complexes at faster heart rates, and smaller P waves (amplitude, 0.15 to 0.2 mV) preceded all taller QRS complexes at slower heart rates.

A second ECG examination was performed 30 minutes after administration of atropine (0.04 mg/kg [0.018 mg/lb], SC) and revealed a sinus tachycardia with a rate of 190 beats/min (Figure 2). Constant R-wave and P-wave amplitudes were evident (2.0 and 0.4 mV, respectively).

## Discussion

In ECG traces, the QRS complex represents ventricular depolarization during the cardiac cycle. Under normal circumstances, R-wave amplitude in QRS complexes does not vary; the amplitude can be as much as 2.5 mV in dogs. Alterations in the R-wave amplitude of QRS complexes can be a result of pericardial effusion, and low-voltage complexes have been detected in cats, dogs, and humans with pleural effusion, pneumothorax, pulmonary thromboembolism, obesity, hypothyroidism, and variations in blood viscosity and intracardiac volume.<sup>2,3</sup> The initial ECG examination of the dog of this report revealed a pattern in which tall R-wave amplitudes of the QRS complexes were typically associated with slow intrinsic heart rates (ie, rates that varied from 60 to 81 beats/min). This variation in the amplitude of the R waves was likely attributable to the Brody effect.

The Brody effect is the variation in amplitude of the R wave that develops with changes in the intracardiac blood volume. When intracardiac blood volume increases, the amplitude of the following beat appears greater than the preceding beat on the surface ECG because of a higher level of conductivity. This phenomenon occurs because the conductivity of the myocardial electrical dipoles, as reflected in an ECG trace, is not homogeneous throughout the thorax. The resistivity of the cardiac muscle is approximately 5.6  $\Omega\text{m}$ , and intracardiac blood volume is approximately 1.6  $\Omega\text{m}$ . In contrast, the resistivity of the lungs is greater (approx 10 to 20  $\Omega\text{m}$ ). Because conductivity and resistivity are inversely proportional, blood makes the conductivity of the heart approxi-

mately 10 times greater than that of the lung parenchyma. The ECG lead field includes the intracardiac path more than it does the lungs. If one imagines the lead field lines that cross the chest, the lines converge as they pass radially through the intracardiac space and diverge tangentially as they pass outside the heart and back into the lung fields. When there is more blood in the chambers of the heart, the conductivity of the heart increases and the amplitude of the QRS complex measured on the surface ECG trace increases.<sup>4-7</sup> In the initial ECG examination of the dog of this report, an exaggerated sinus arrhythmia was detected. When the heart rate slowed, there was an extended diastolic period during which filling of the ventricles was enhanced. Such increased filling consequently increased the conductivity of the heart and was reflected on the surface ECG by increases in R-wave amplitudes during periods of slower heart rate. During periods of more rapid intrinsic heart rate, the reverse occurred and the R-wave amplitude decreased. The dog was not affected by pleural effusion, pneumothorax, pulmonary thromboembolism, obesity, or pericardial effusion, and these conditions are all less likely causes for the lower voltage complexes or variation in amplitudes detected via ECG.

Several experimental studies<sup>8-10</sup> have been performed to investigate the Brody effect in animals during hemorrhage via venipuncture. In 1 study,<sup>8</sup> reduction in the blood volume of anesthetized cats and dogs was associated with an immediate reduction in the amplitude of the QRS complexes. This reduction was immediately reversed when the removed blood was returned to the circulation. In another study<sup>9</sup> in dogs, signals from a vector-ECG (Nelson-type) lead system were evaluated and the magnitude and angles of the electrical heart vector in spherical coordinates were calculated. The coordinates were represented as radial and tangential forces. During removal and subsequent replacement of considerable blood volumes from dogs, there was an increase in tangential forces as blood was removed and an increase in radial forces as normal blood volume was restored, which was supportive of the Brody effect.<sup>9</sup> In a study<sup>10</sup> of 10 Greyhounds, removal of approximately a third of each dog's blood volume resulted in a substantial reduction in the QRS complex voltage (mean

voltage was 74% of the baseline voltage that was measured before venipuncture). The results of these experiments illustrate the Brody effect via venipuncture-induced reduction in intracardiac blood volume.

Results of other studies<sup>11-13</sup> involving alterations in intracardiac blood volume by methods other than venipuncture have supported the Brody effect. In a study<sup>11</sup> of anesthetized young domestic pigs, heart rate was doubled by electrical pacing or a volume of blood (20 mL/kg [9.1 mL/lb]) was removed to decrease end-diastolic volume. Both venipuncture and doubling of the heart rate caused a decrease in QRS amplitude, compared with baseline amplitude values. Conversely, an increase in R-wave amplitude was evident when the pigs were administered phenylephrine IV to increase end-diastolic volume via  $\alpha_1$ -adrenoceptor activation with subsequent vasoconstriction.<sup>11</sup> In anesthetized mixed-breed dogs, intracardiac blood volume was decreased via occlusion of the superior vena cava or the inferior vena cava (or both simultaneously); P-wave amplitude increased and R-wave amplitude decreased as a result of predominantly tangential activation of the atrium and radial activation of the ventricle.<sup>12</sup> The R-wave amplitude decreased via the Brody effect because the radial forces correspond to the voltage of the QRS complexes. In another study<sup>13</sup> involving anesthetized dogs, decreases in R-wave amplitude were associated with decreases in end-diastolic volume during rapid atrial pacing. The results of the aforementioned experiments indicate that the Brody effect occurs as intracardiac blood volume changes through great vessel occlusion and pacing.

Although the Brody effect has been confirmed experimentally in several species, data that challenge the phenomenon have been reported.<sup>14-17</sup> A study<sup>14</sup> was performed in which decreases in end-diastolic volume associated with movement from sitting to standing positions were evaluated in humans, and no change in the R-wave amplitude was detected. In other human studies,<sup>15,16</sup> changes in blood viscosity (measured by changes in Hct) and effects of hemodialysis were not associated with alterations in the R-wave amplitude. Furthermore, ventricular chamber enlargement with subsequent increased end-diastolic volume did not affect R-wave amplitude.<sup>17</sup>

In addition to the Brody effect, a common result of high parasympathetic tone was the presence of a wandering pacemaker in the dog of this report. Taller P waves occurred only before smaller QRS complexes, and smaller P waves preceded every taller QRS complex and some smaller QRS complexes. A wandering pacemaker is an atrial arrhythmia that develops when the cardiac pacemaker site varies between the normal location of the sinoatrial node and other sites in the atrium or atrioventricular node. Changes in duration and amplitude of the P waves reflect the transiently changing location; these changes in P-wave morphology depend on the location in the atrium at which the depolarization begins. Vagal and sympathetic tones both induce shifts in the sites of impulse origination of sinus beats. This results in taller P waves when the heart rate is faster (sympathetic stimulation shifts the pacemaker site cranially) and smaller P waves when the heart rate is slower (vagal stimulation shifts the pacemaker site caudally).<sup>18</sup> In the initial ECG trace obtained from the dog of this report, the P-wave duration appeared normal but P-wave amplitude varied in the described manner. Taller P waves were evident when the heart rate increased (less

vagal tone), and shorter P waves were seen when the heart rate decreased (higher vagal tone).

Thirty minutes after administration of atropine, the Brody effect and wandering pacemaker patterns were abolished in the dog of this report. Sinus tachycardia with a heart rate of 190 beats/min was evident. Disappearance of the wandering pacemaker was achieved because the rhythm was then mediated via sympathetic tone rather than by vagal control and because the pacemaker site was exclusively within the sinoatrial node. The Brody effect was also eliminated because there was no longer a sinus arrhythmia to allow variations in ventricular filling or end-diastolic volume. The intracardiac volume was consistent; hence, R-wave amplitude did not vary. The dog's high vagal tone could have been a result of its primary neurologic disease; for example, an increase in intracranial pressure secondary to encephalitis could have resulted in direct activation of CNS centers or in stimulation of baroreceptor reflexes by the associated pressor response. Fortunately, the wandering pacemaker and the Brody effect in this dog represented benign conditions that can occur in association with high parasympathetic tone.

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