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ECG of the Month

An 8-year-old 28-kg (62-lb) spayed female Golden Retriever was evaluated at an emergency referral hospital because of suspected ethylene glycol toxicosis. The dog had become markedly polyuric and polydypsic 2 days prior to the evaluation. On the day of the evaluation, the dog had developed ataxia and vomiting. Initial serum biochemical analysis revealed the following abnormalities: BUN concentration, 58 mg/dL (reference range, 7 to 27 mg/dL); creatinine concentration, 7.6 mg/dL (reference range, 0.5 to 1.8 mg/dL); and phosphorus concentration, 13.2 mg/dL (reference range, 2.5 to 6.8 mg/dL). After 2 seizure events, the dog was admitted to the hospital and treatment with fluids, antiemetics, broad-spectrum antimicrobials, gastric protectants, calcium gluconate, furosemide, and mannitol was initiated. A plasma sample was tested with an ethylene glycol veterinary test strip (manufacturer information not available) at this time, and the result was negative. After 2 days of hospitalization, the dog became anuric and was referred to the University of California-Davis William R. Pritchard Veterinary Medical Teaching Hospital Emergency Service for further diagnostic testing and treatment.

At the referral hospital, the dog was laterally recumbent, obtunded, tachypneic, and clinically overhydrated. Rectal temperature was 39°C (102.0°F), heart rate was 150 beats/min, and respiratory rate was 80 breaths/min. Initial clinicopathologic analyses revealed the following abnormalities: BUN concentration, 94 mg/dL (reference range, 11 to 33 mg/dL); serum creatinine concentration, 10 mg/dL (reference range, 0.8 to 1.5 mg/dL); serum potassium concentration, 3.3 mEq/L (reference range, 3.6 to 4.8 mEq/L); and a high anion gap metabolic acidosis with high serum osmolality. Sodium bicarbonate (1 mEq/kg [0.45 mEq/lb]) was administered IV over a 4-hour period. At this time, a plasma sample was tested with an ethylene glycol veterinary test strip[a] and the result was positive. Serum antibodies against *Leptospira* spp were not detected. Hemodialysis was performed, and supportive care was initiated. One dose of methadone (0.1 mg/kg [0.045 mg/lb], IV) was administered in the evening prior to the dog's second dialysis treatment. An arrhythmia and intermittent head tremors were noted during this dialysis treatment. Electrocardiography was performed the following day.

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ECG Interpretation

The initial 6-lead ECG recording revealed a mean heart rate of 90 beats/min but an irregular rhythm; heart rate varied from 45 and 115 beats/min (**Figure 1**). The most apparent abnormalities were the predominance of negative P waves in the lead II and III tracings and evidence of second-degree atrioventricular (AV) block. The sinus node, left atrium, and AV node were considered as the possible origin of the negative P waves. The 2 latter locations constitute ectopic sites and would result in atrial tachycardia and nodal tachycardia. When the negative P waves first became evident, the most common rate of the negative P waves was 125 beats/min (P-P interval, 0.48 seconds [reference range,[1] 0.38 to 0.86 seconds]); subsequently, the rate of the negative P waves was unchanged or increased (shortening of the P-P interval). There was second-degree AV block, second-degree AV block with termination of the rhythm generated by the negative P waves, or cessation of the rhythm produced by the negative

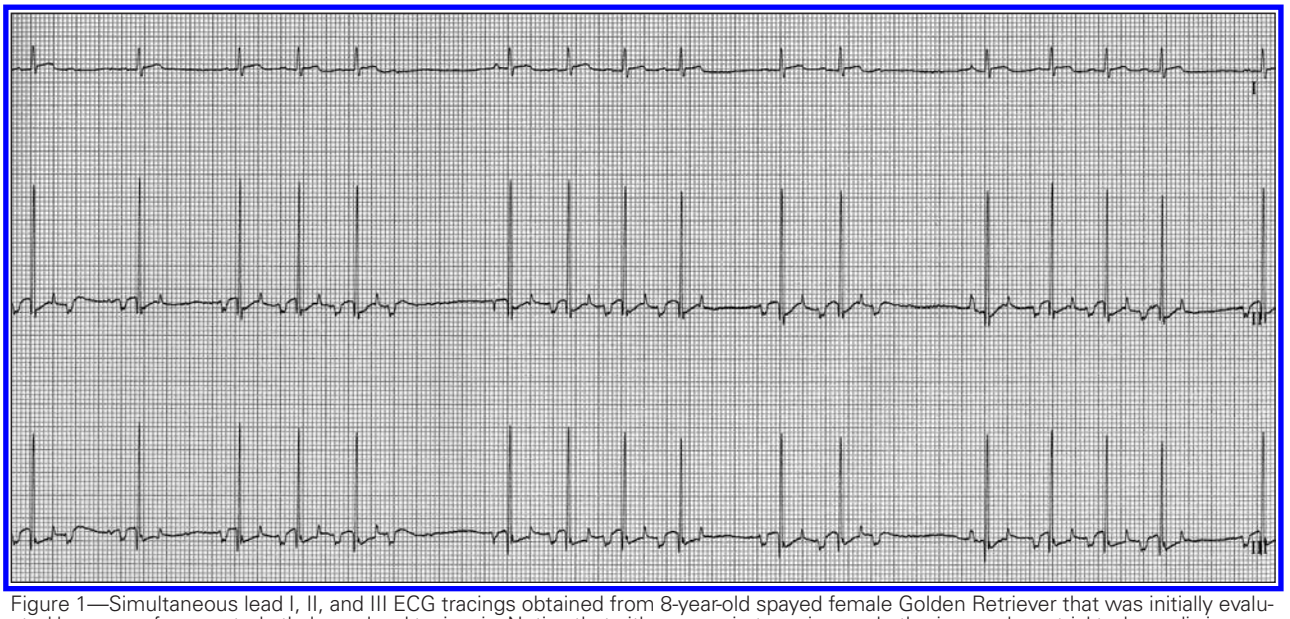

Figure 1—Simultaneous lead I, II, and III ECG tracings obtained from 8-year-old spayed female Golden Retriever that was initially evaluated because of suspected ethylene glycol toxicosis. Notice that either a respiratory sinus arrhythmia or a slow atrial tachycardia is present. First-degree and Mobitz type I second-degree atrioventricular (AV) block are also present. Paper speed = 25 mm/s; 1 cm = 1 mV.

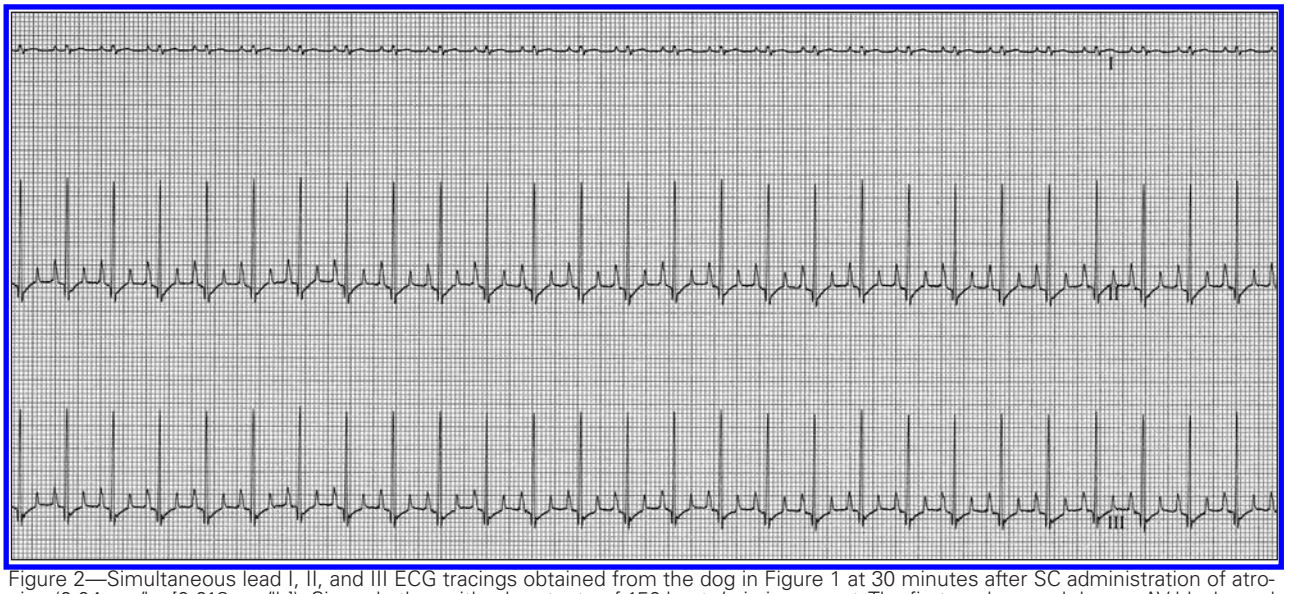

Figure 2—Simultaneous lead I, II, and III ECG tracings obtained from the dog in Figure 1 at 30 minutes after SC administration of atropine (0.04 mg/kg [0.018 mg/lb]). Sinus rhythm with a heart rate of 150 beats/min is present. The first- and second-degree AV blocks and wandering pacemaker are no longer present supporting the conclusion that they were secondary to high vagal tone. Paper speed = 25 mm/s; 1 cm = 1 mV.

P waves with a subsequent pause. The pause was then terminated by a sinus complex (positive P wave), the onset of the rhythm characterized by negative P waves again, or what appeared to be a fusion between the 2 P wave morphologies. The second-degree AV block was consistently Mobitz type I. First-degree AV block was also evident with the PR interval consistently prolonged (range, 0.14 to 0.19 seconds [reference range,[1] ≤ 0.13 seconds]); the longest PR interval occurred before development of the second-degree AV block. The duration of the P waves was shorter when the waves were positive (0.04 seconds) than when the waves were negative (0.06 seconds). The QRS complexes were narrow and upright; duration was considered normal (0.04 seconds [reference range, < 0.06 seconds]) with a high-normal amplitude (2.7 mV [reference range, < 3.0 mV]).

Another ECG examination was performed 30 minutes after SC administration of atropine (0.04 mg/kg [0.018 mg/lb]; **Figure 2**). The heart rate increased to 150 beats/min, and the PR interval decreased to 0.1 seconds; P wave morphology was considered normal, although the amplitude was slightly increased (0.5 mV [reference range,[1] < 0.4 mV). The 2 possible explanations for this atropine response were that the negative P waves represented an exaggerated wandering pacemaker resulting from high vagal tone and that this pacemaker disappeared once vagal tone was suppressed or the sinus rate increased to a rate that was faster than the rate of the supraventricular ectopic focus, thereby causing suppression of the ectopic focus depolarization.

A full or adequate response to the atropine response test is generally defined as achieving a heart rate > 160 beats/min.[1–3] The response of the dog of this report was therefore somewhat less than that anticipated from a clinically normal dog. Poor tissue perfusion and consequent delayed absorption of the drug were believed to be responsible for the slightly blunted response of the dog of this report.

Discusssion

For the dog of this report, the identified arrhythmia may have been a respiratory sinus arrhythmia and the administration of an opioid may have been responsible for its development. The apparent periodicity of the arrhythmia and the Mobitz type I second-degree AV block support this explanation of a vagally medicated arrhythmia. Respiratory sinus arrhythmia is an irregular cardiac rhythm characterized by a cyclic increase and decrease in heart rate that usually corresponds with respiration.[2,3] It is a common finding in clinically normal dogs because the predominant autonomic influence on dogs' hearts is parasympathetic.[2] Without vagal influence, the heart rate of a dog is consistently between 140 and 200 beats/min.[4,5] During respiratory sinus arrhythmia, the heart rate increases with inspiration. The control of this response is complex and involves respiratory center activation, reflex tachycardia in response to right atrial stretch, chemoreceptor activation, and the baroreceptor reflex.[4,5]

A wandering pacemaker is defined as variance in the morphology and amplitude of the P waves over time. This common ECG phenomenon results from a shift in the site of depolarization from the sinus node along the terminal crest of the right atrium toward or occasionally to the AV node.[1] This results in altered depolarization pathways through the atria and consequent changes in P wave morphology.[1] The P waves can appear upright, biphasic, isoelectric, or negative and can vary greatly in their morphology and amplitude.[4] Wandering pacemaker is often evident with respiratory sinus arrhythmia, and both phenomena are associated with an increase in vagal input.[6] As in the dog of this report, atropine administration generally eliminates both sinus arrhythmia and wandering pacemaker by decreasing parasympathetic tone. The mechanism behind the ECG finding of wandering pacemaker is complex and is an area of ongoing research.[7–9]

The sinoatrial node was first described as a distinct anatomic structure more than a century ago.[10] Exploration of the complex interactions between the structure and function of the sinus node continues. In histologic terms, the sinus node in dogs is not a single point as it is often depicted; on the basis of findings in 20- to 30-kg (44- to 66-lb) dogs, it extends approximately 11 mm and has a somewhat crescent shape.[11]

Sinus rhythms have long been recognized to originate from an area much larger than that of the anatomic

sinus node. In 1980, multiple origin points (distributed over a 40-mm area) for sinus rhythms in dogs were reported.[11] It was theorized that the different points of origin were sensitive to differing levels of autonomic input because they corresponded to distinct heart rates. Direct cardiac nerve stimulation has been used to confirm that shifts in the location of canine pacemaker activity are induced by changes in autonomic input to the heart.[12] Results of a more recent study[7] confirmed the multicentric nature of the human sinus node and the presence of preferential pathways of conduction to multiple electrical exit points.

In 1995, it was reported that total elimination of sinus pacemaker function in dogs required ablation of the entire crista terminalis from the right atrial junction with the cranial vena cava to the junction with the caudal vena cava (approx area, 4 × 1.5 cm).[13] This area far exceeds the anatomic limits of the sinus node. Furthermore, a study[5] of gene expression in mice has revealed that cardiac conduction tissue extends from the cranial vena cava along the crista terminalis to the AV node. Wandering pacemaker is thought to be the result of shifts in pacemaker activity within this fairly broad region.

The recent discovery of a discrete paranodal area in close proximity to the sinus node of human hearts may provide further explanation of this phenomenon. The tissue of this paranodal area is more extensive than that of the sinus node, and its histologic and immunohistochemical character is intermediate between that of tissues of the sinus node and the atrium.[8] The paranodal area may be responsible for the multicentric nature of the pacemaker activity of the sinus node, perhaps accounting for the observed ECG phenomenon of wandering pacemaker.[9] However, further investigation of the paranodal area will be required to elucidate its true role.

The other most likely explanation for the rhythm in the dog of this report was an atrial tachycardia focused in the left atrium. Supraventricular tachycardia (SVT) can be broadly classified into atrial or junctional tachyarrhythmias. Atrial tachyarrhythmias require atrial tissue for the initiation and maintenance of the arrhythmia. Focal atrial tachycardias that originate from the left atrium most commonly form in the region of a pulmonary vein.[14] They can be caused by abnormal automaticity, triggered activity, or micro-reentry. Junctional tachyarrhythmias require the AV node for initiation and maintenance of the arrhythmia. Atrial and junctional tachyarrhythmias can be differentiated by evaluation of P′ waves (ie, nonsinus atrial depolarizations). When the P′ waves occur closer to the preceding QRS complex than to the subsequent QRS complex, the SVT is classified as short RP′ SVT; when the P′ waves are further from the preceding QRS complex and closer to the subsequent QRS complex, it is classified as long RP′ SVT. Atrial tachyarrhythmias typically have long RP′ intervals, and junctional tachyarrhythmias typically have short RP′ intervals. Therefore, the long RP′ interval in the dog of this report would be suggestive of an atrial tachycardia originating in the left atrium.[15] In addition, the presence of Mobitz type I second-degree AV block typically rules out the possibility of a junctional tachycardia.[16]

Focal atrial tachycardia can be associated with rate increase (warm up) and rate decrease (cool down), resulting in a variation of the atrial rate, as was evident in the dog of this report. In addition, the ventricular rate can be irregular when second-degree AV block (Mobitz type I or type II) is present. Focal atrial tachycardia in dogs has been reported, and generally the ventricular rate (164 to 270 beats/min) is higher than that reported for humans (130 to 240 beats/min).[14] However, for the dog of this report, it is reasonable to expect that illness and sedation secondary to methadone administration may have contributed to a slower than typical rate. The arrhythmia in this case was of no clinical importance because it resolved with appropriate supportive care or as the effect of the methadone injection dissipated.

a. Kacey ethylene glycol diagnostic strips, Kacey Diagnostics, Asheville, NC.

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