A 1.5-year-old sexually intact male Labrador Retriever was referred for investigation of diarrhea of 3 months’ duration. Previous treatments had included deworming and administration of antimicrobials (metronidazole and spiramycin) with a concurrent dietary change. Physical examination revealed poor body condition (body condition score, 2/9; weight, 21.6 kg [47.5 lb]), pale mucous membranes, slow capillary refill time (>2 seconds), strong precordial beat, strong pulse, no heart murmur, and slow heart rate (40 beats/min). The dog was weak and had signs of depression. Rectal temperature was 37.8°C (100°F).

Diagnostic evaluations included a CBC, serum biochemical analysis, assessment of serum electrolyte concentrations, coagulation profile, trypsin-like immunoreactivity assay, urinalysis, abdominal ultrasonography, thoracic radiography, and cardiac evaluation. Hematologic and serum biochemical values were within reference ranges, as were serum concentrations of potassium (4.7 mmol; reference range, 3.7 to 5.8 mmol), sodium (141 mmol; reference range, 141 to 152 mmol), and calcium (10.1 mg/dL; reference range, 8 to 12 mg/dL). Only serum glucose concentration was mildly low (78 mg/dL; reference range, 80 to 110 mg/dL). The result of the trypsin-like immunoreactivity assay was considered normal (5.1 ng/mL; reference range, 5 to 35 ng/mL). Activated partial thromboplastin time was 17.8 seconds (reference range, 9 to 20 seconds), and partial thromboplastin time was 9.3 seconds (reference range, 5 to 8 seconds); plasma fibrinogen concentration was 253 mg/dL (reference range, 100 to 400 mg/dL). Abdominal ultrasonography revealed that the mucosal layer throughout the entire small intestine was inhomogeneous and mildly hyperechogenic; there was mild thickening (0.42 mm) of the wall of the large intestine.

The dog underwent evaluation of its cardiac rhythm and contractility. No episode of syncope had been detected during the dog’s entire life. Electrocardiography (Figure 1) and complete echocardiography were initially performed. All echocardiographic values were within reference limits. Fractional shortening was 43% (reference range, >27%), and ejection fraction was 74% (reference range, 40% to 100%). Left ventricular end-diastolic diameter determined with allometric scaling was 1.73 (reference range, 1.27 to 1.85); left ventricular end-systolic diameter determined with allometric scaling was 0.99 (reference range, 0.71 to 1.26). The left atrial-to-aortic root ratio was 1.37 (reference range, <1.5). Aortic and pulmonary flows were laminar, and the transmitral valve pattern was considered normal (ratio between early [E] and late [atrial] ventricular filling velocities [ie, E wave-to-A wave ratio], 1.7; reference range, 1 to 2). Systolic blood pressure

Figure 1—Surface 12-lead ECG tracings obtained from a 1.5-year-old Labrador Retriever that was evaluated because of diarrhea of 3 months’ duration. Notice the marked sinus bradycardia; heart rate is 40 beats/min. The P-QRS coupling is always present, and the P-QRS-T intervals are all within reference limits. Paper speed = 50 mm/s; 1 cm = 2 mV.
(determined via a Doppler technique) was within reference limits (120 mm Hg). Thoracic radiographic findings were unremarkable.

During exploratory laparotomy to obtain biopsy specimens of the small and large intestines, the pancreas appeared small and hypoplastic. Full-thickness biopsy specimens of the pancreas and of portions of the entire intestinal tract were obtained. Tissue samples underwent histologic examination, which revealed moderate to severe lymphocytic-plasmacytic inflammatory bowel disease with concurrent severe pancreatic lymphocytic-plasmacytic infiltrates with parenchymal atrophy.

**ECG Interpretation**

A 12-lead ECG recording (Figure 1) obtained from the dog during the initial evaluation revealed sinus bradycardia (SB) with marked respiratory sinus arrhythmia (40 beats/min). No other arrhythmias were detected during the 5-minute ECG recording. P waves were consistently present in tracings from both the standard and precordial leads, with no alteration of the PR interval (0.10 seconds; reference range, 0.06 to 0.13 seconds) and no isolated P waves. The QRS complex morphology was within normal limits (duration, 0.04 seconds [reference range, < 0.06 seconds]; amplitude, 1.2 mV [reference range, < 3.0 mV]), and there was no alteration of the ST segments (isoelectric) or T waves. The P-P intervals were variable with a regular pattern associated with an increased heart rate during inspiration and a decreased heart rate during expiration.

On the basis of the clinical and ECG findings for the dog of this report, an increased vagal tone was suspected. Atropine sulfate (0.04 mg/kg [0.018 mg/lb], IV) was administered, and an increase in heart rate (to 100 beats/min) was identified via ECG.

To evaluate cardiac rhythm over a longer period of time and to exclude any pathological cause of SB, Holter monitoring was suggested. During 24-hour Holter monitoring, no arrhythmia was detected except for SB. Mean heart rate was 50 beats/min, minimum heart rate was 27 beats/min, and maximum heart rate was 100 beats/min, which occurred during exercise (Figure 2). Heart rate variability did not differ markedly when the dog was sleeping or awake.

On the basis of the results of the histologic examination of the biopsy specimens, the dog was fed a homemade diet (boiled turkey and potato) and pancreatic enzymes were added. One month after the first visit, the dog underwent a cardiac recheck examination and 24-hour Holter monitoring (Figure 2) to assess heart rate variability. At this time, the dog's weight had increased (25 kg [55 lb]) and it was bright and alert. Mucous membrane color was normal, pulses were strong, and mean heart rate during auscultation ranged from 60 to 70 beats/min. During Holter monitoring, mean heart rate was 67 beats/min, minimum heart rate was 40 beats/min, and maximum heart rate was 148 beats/min. No arrhythmia was identified during 24-hour monitoring. The patterns of the Holter tachograms obtained at the initial evaluation and the 1-month recheck examination were different, in that the exaggerated heart rate variability due to the increased vagal tone detected initially was notably diminished. Ten months after the referral examination, the dog was doing well and had gained weight (29 kg [63.8 lb]); its mean resting heart rate had increased to 120 beats/min.

**Discussion**

Sinus bradycardia is defined as a regular rhythm < 60 beats/min in awake dogs; healthy dogs may have lower heart rates during sleep. In clinical practice, SB is an uncommon rhythm disturbance and generally is secondary to drug overdose in anesthetized dogs. In conscious dogs, SB can be associated with physiologic and pathological conditions, such as hypothermia, hypoxia, hyperkalemia, or any increase in vagal tone (attributable to vagal reflexes, intestinal obstruction, urethral obstruction, or intracranial mass). Other causes known to decrease heart rate are related to administra-

![Figure 2](image-url)
anorexia nervosa, SB is the most common ECG alteration. 

Insulin has a direct influence on heart rate as well. The circulating concentrations of insulin results in increasing sympathetic tone, thereby increasing heart rate. 

The diagnostic algorithm for dogs that are suspected of having SB is to exclude any pathological, life-threatening cause such as hypoxia, hyperthermia, or electrolyte disturbances. When serum potassium concentration exceeds 5.5 mEq/L, ECG recordings may reveal SB, tall and peaked T waves, low-amplitude or absent P waves, low-amplitude R waves, and prolonged PR and QRS intervals. 

The ECG changes generally worsen with increasing serum potassium concentration, but there may be coexistent acid-base and electrolyte imbalances, which could alter or mask ECG signs of hyperkalemia. The dog of this report did not have any detectable electrolyte imbalance, and SB as a result of hyperkalemia was ruled out.

Another important differential diagnosis is sick sinus syndrome, an arrhythmia that is caused by an imbalanced electrical impulse arising from an altered sinoatrial conduction system. 

The most frequent clinical sign of sick sinus syndrome is sinus arrest or sinoatrial block (48.5% of dogs with sick sinus syndrome). 

Affected dogs also commonly have periods of sustained tachyarrhythmias co-existing with bradyarrhythmia. 

For the dog of this report, sick sinus syndrome was ruled out on the basis of the Holter monitoring findings.

Given the dog's history and clinical findings, SB secondary to vagotonia was suspected. During both 24-hour periods of Holter monitoring, an increase in heart rate secondary to exercise was identified. This finding is indicative of a sympathetic response to exercise and confirmed vagotonia. For the dog of this report, a sympathovagal imbalance as a result of increased parasympathetic activity was probably the cause of the SB. In malnourished humans, SB is likely to develop because of increased vagal tone, which is generated as what can be thought of as an economizing mechanism (ie, the body's attempt to cope with caloric deprivation). 

In young people affected by anorexia nervosa, SB is the most common ECG alteration. 

It is also well-known that body mass index has a positive correlation with heart rate in people with irritable bowel syndrome. 

Great fluctuations in body weight over time are rare, and the parasympathetic system is thought to have a central role in the regulatory mechanisms involved in balancing energy intake and energy consumption. In healthy human volunteers who have lost 10% of their body weight, increased parasympathetic activity has been identified (characterized by a decrease in heart rate and an increase in the R-R interval) with concomitant reduction in sympathetic activity. 

The dog of this report was severely underweight; thus, it can be argued that parasympathetic activity was enhanced and sympathetic action was decreased as a mechanism to enable the dog to cope with a change in energy homeostasis. Furthermore, it has been shown that increasing circulating concentrations of insulin results in increasing sympathetic tone, thereby increasing heart rate. 

Insulin has a direct influence on heart rate as well. The dog of this report had histologic signs of exocrine pancreatic dysfunction, with apparent atrophy of the acinar portion of the pancreas. Whether this finding was a concausal effect in the pathogenesis of SB is less clear. Insulin resistance in obese humans stimulates the sympathetic nervous system, thereby increasing the heart rate, compared with findings in normal individuals. Also, obese normotensive normoglycemic patients have a significant decrease in heart rate and circulating insulin concentration when they lose weight. 

To the author's knowledge, an association of pancreatic dysfunction with low heart rate in dogs has not been reported. Whether circulating insulin concentration has an effect on heart rate in dogs has yet to be determined, but the physiopathologic interplay of insulin metabolism and heart rate in dogs might warrant investigation.

In a previous report of dogs with malnutrition, only low-voltage QRS complexes were identified as the major alteration in ECG findings. Sinus bradycardia has not been identified as a finding in malnourished dogs, although SB is very common in malnourished humans. 

For humans with malnutrition, SB; low-amplitude P waves, QRS complexes, and T waves; and prolonged QT intervals have been reported. Veterinary practitioners should be aware that marked cachexia can be associated with SB because of an energy-sparing mechanism and pathological vagotonia. Sinus bradycardia is reversible if the primary cause of weight loss is determined and treated. 

References