A 15-year-old 5.4-kg (11.9-lb) neutered male Maine Coon cat was evaluated because of a heart murmur and bradycardia. The cat had a history of weight loss and reduction in appetite of 4 to 6 weeks' duration. On physical examination, the cat's heart rate was approximately 110 beats/min and a grade 2/6 parasternal heart murmur was auscultated. Other physical examination findings were unremarkable. Because of the low heart rate, an ECG examination was performed (Figure 1).

**ECG Interpretation**

The 6-lead ECG examination performed at the initial evaluation revealed an underlying rhythm in a 3:2 (atrial-to-ventricular) ratio with an approximate atrial rate of 180 beats/min and an approximate ventricular rate of 120 beats/min (Figure 1). Evaluation of lead II (Figure 2) revealed that the first P wave was associated with a QRS complex of normal appearance, consistent with a normal sinus beat. The second P wave was associated with a wide upright QRS in leads II, III, and aVF suggestive of a left bundle branch block. The third P wave was buried in the T wave of the second QRS complex. The PR intervals before the first 2 QRS complexes were consistent prior to the blocked P wave, suggestive of a Mobitz type II second-degree atrioventricular (AV) block. The R-R interval preceding the wide QRS complex was short.

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and the R-R interval following the wide QRS complex was comparatively much longer. This pattern of short and long cycle lengths resulting in conduction block during periods of bradycardia was suggestive of an Ashman phenomenon.

**Discussion**

Atrioventricular block is defined as a delay or interruption in conduction of a supraventricular impulse through the AV node and bundle of His when the AV node is not physiologically refractory. For the cat of this report, a diagnosis of second-degree AV block was made, of which there are 2 types: type I and type II. Type I second-degree AV block (also called the Wenckebach phenomenon) is classically characterized by a progressive prolongation of the PR interval with successive beats until a P wave is blocked. Type II second-degree AV block is characterized by PR intervals of consistent duration prior to the blocked P wave. In human medicine, identification of a QRS complex with normal configuration can be suggestive of type I second-degree AV block, whereas an abnormal QRS complex configuration can indicate that type II second-degree AV block is present. However, QRS complex morphology is not a reliable means of differentiating Mobitz type I from type II second-degree AV block. In dogs, it is important to determine the type of second-degree AV block: type I can be a normal finding in some dogs and usually does not require treatment, whereas type II can progress to higher degrees of AV block and, consequently, affected dogs require pacemaker implantation. In contrast, third-degree AV block is typically tolerated by cats without pacemaker placement because of their relatively high ventricular escape rate as well as their sedentary lifestyles.

In addition to pathological lesions in the conduction system, intraventricular conduction delays can result from the effects of changes in the heart rate. Rate-dependent block or aberration can occur at either high or low heart rates. In bradycardia-dependent block or Ashman phenomenon, a wide QRS complex follows a short R-R interval and precedes a long R-R interval. The wide QRS complex can have right bundle or left bundle branch block morphology, although right bundle branch block morphology is more common. The Ashman phenomenon occurs because the duration of the refractory period of the myocardium determined by a long R-R interval of the preceding cycle will prolong the ensuing refractory period, and if a shorter R-R cycle follows, the beat terminating the cycle is likely to be conducted aberrantly. Because the refractory period of the right bundle branch is longer than that of the left bundle branch, the right bundle will still be in the refractory period when the supraventricular impulse reaches the His-Purkinje system, which usually results in a QRS complex with right bundle branch block morphology.

Furthermore, bradycardia secondary to complete AV block has been shown to prolong action potential duration in cardiac myocytes in experimental studies in dogs and rabbits. Changes in repolarization of cardiac myocytes induced by an altered pathway of activation have been identified following bradycardia or tachycardia in experimental studies in dogs and electrophysiologic studies in people and is termed cardiac memory. It is thought that cardiac memory is a relatively early change in electrophysiologic function of cardiac myocytes that is completely reversible but would cause more persistent functional and structural myocardial changes if the inciting cause for the development of cardiac memory continued. The pathophysiologic mechanism of cardiac memory is incompletely understood but postulated to involve an abnormal pattern of cardiac myocyte stretch resulting from the altered pathway of action potential conduction. This results in activation of angiotensin and other intracellular signaling systems as well as increased sympathetic neural activity, which precipitates an alteration of ion channel structure and density. The duration of these repolarization changes can be a few minutes (referred to as short-term cardiac memory) to several months (referred to as long-term cardiac memory). The chronic bradycardia in the cat of this report may have caused cardiac memory to prolong repolarization of the conduction system, which is a necessary component for the development of Ashman beats.

Ashman beats were first reported for humans in 1947, and such beats are a common ECG finding in people with atrial fibrillation. Identification of Ashman beats does not warrant initiation of treatment, but it is imperative to recognize the Ashman phenomenon because the aberrantly conducted beat can be misinterpreted as a ventricular premature complex and the prognosis and treatment for the 2 conditions are different.

For the cat of this report, echocardiography was subsequently performed and revealed mild dilation of the left ventricle, which was assessed as likely secondary to the low heart rate, but all other cardiac chambers were considered to be of normal size. An atropine response test was performed, but there was no improvement in ventricular response rate, which was assessed as consistent with Mobitz type II second-degree AV block. No treatment was instituted for the cat of this report, and eventually a diagnosis of pancreatitis was made.

**References**