A 15-year-old 5.5-kg (12.1-lb) spayed female domestic medium hair cat was referred to the University of Georgia Veterinary Medical Teaching Hospital for further evaluation of hyperthyroidism. A high circulating serum thyroxine (total T$_4$) concentration was detected via routine screening as part of a yearly geriatric examination. The owner reported the cat had no changes in urination, defecation, thirst, appetite, or activity. The only abnormalities noted at home were an unkempt coat and vomiting of hair balls 1 to 2 times weekly. A diet change to a senior formula had been recommended 1 year prior, but there was no other pertinent medical history. The cat was not receiving any medications at the time of evaluation.

A CBC and serum biochemical panel revealed no notable abnormalities. The serum total T$_4$ concentration was elevated at 7.6 µg/dL (laboratory reference range, 0.8 to 4 µg/dL). A urinalysis revealed low urine specific gravity (1.019), and results of bacterial culture of a urine sample yielded no growth. Physical examination identified a palpable bilateral thyroid gland nodule that was more pronounced on the right side of the neck. Cardiothoracic auscultation revealed bradycardia (160 beats/min) and a grade 2/6 left parasternal systolic heart murmur. No abnormal lung sounds were noted. Given the abnormal cardiovascular findings, thoracic radiography and echocardiography were performed. The results of thoracic radiography were unremarkable. A complete echocardiogram indicated normal cardiac structure and function with mild, hemodynamically insignificant mitral valve regurgitation. This was thought to be secondary to either mild myxomatous valve degeneration or mild mitral valve dysplasia. A 6-lead ECG recording was obtained to further characterize the persistent bradycardia (Figure 1).

**ECG Interpretation**

The ECG tracings indicated the presence of a rare dysrhythmia termed escape-capture bigeminy. Escape-capture bigeminy is evidenced by the repetitive sequence of an escape beat followed by a normally conducted sinus beat. Second-degree atrioventricular block (AVB) was also present. A nonconducted P wave followed each sinus beat, causing a pause that terminated with an escape complex. The atrial rhythm was regular and sinus in origin with a rate of 222 depolarizations/min and a cycle duration of 270 milliseconds. The ventricular rhythm noted was irregular with a rate of 140 depolarizations/min and was characterized by paired complexes that had 2 separate morphologies.
The sinus complexes were composed of an Rs morphology in the lead II tracing and were conducted with a left ventricular enlargement pattern (QRS complex duration, 50 milliseconds). These complexes use the normal atrioventricular conduction system. Occurring immediately before each sinus complex, the escape complexes had an Rs morphology in lead II with a more prolonged QRS complex duration (60 milliseconds). Escape beats originate spontaneously from either the ventricle or atrioventricular node region in response to bradycardia. Escape beats occur when a more rapid rhythm to override the ventricular tissue impulse is not present. This phenomenon is called overdrive suppression and is a protective mechanism against asystole. The morphology of the escape complexes was similar to that of the sinus conducted beats. This suggested they originated from within the tissues of the atrioventricular node (nodal or junctional complex) or from a high-ventricular focus. The escape interval, or the interval between normally conducted sinus complexes, was 420 milliseconds, which correlated to an instantaneous heart rate of 142 depolarizations/min.

Throughout the ECG tracings, there were P waves that were not followed by QRS complexes, which was consistent with second-degree AVB. On cursory evaluation, it appeared as though there was only 1 blocked P wave. However, the escape complexes were distorted because of summed blocked P waves that created small deflections. The P waves had a perfectly regular rhythm sometimes described as P waves that march. There were 3 P waves for every 2 QRS complexes, meaning that 2 of every 3 sinus impulses were blocked within the atrioventricular node. This is described in the human medical literature as second-degree AVB with 3:2 conduction. In the dog of the present report, 1 of the 2 QRS complexes was an escape complex; thus, even though the atrial-to-ventricular depolarization ratio was 3:2, the more accurate atrial-to-ventricular depolarization ratio was 3:1 because only 1 depolarization occurred through the normal conduction system. The blocked P wave that followed the sinus complex was considered pathological and secondary to changes within the conduction system. The blocked P wave that followed the escape complex was considered physiologic owing to the interference following retrograde conduction of the escape beat into the atrioventricular node, as has been noted in a previous case report.

The cat was administered methimazole (0.5 mg/kg, PO) every 12 hours. On reevaluation 1 month later, the cat was euthyroid with a serum total T4 concentration of 1.1 µg/dL (laboratory reference range, 1 to 4 µg/dL). At this time, an ECG examination revealed persistence of bradycardia and a heart rhythm consistent with escape-capture bigeminy. Three months after the initial evaluation, the cat was brought to the university emergency service because of clinical signs of congestive heart failure with pleural effusion, pulmonary edema, and ascites. Results of an ECG examination indicated progression of the conduction system disease to third-degree AVB. The congestive heart failure was considered secondary to third-degree AVB and severe bradycardia. The cat was again found to be euthyroid with a serum total T4 concentration of 1.0 µg/dL. An epicardial pacemaker was placed via surgical laparotomy, and the congestive heart failure resolved shortly after implantation.

To better describe the ECG findings, a Lewis (ladder) diagram was created (Figure 2). A ladder diagram is aligned with the ECG strip so that lines can be drawn

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**Figure 2**—Lead II ECG tracing from the ECG recording in Figure 1 with a synchronous Lewis (ladder) diagram. The atria (A), atrioventricular node (AV), and ventricles (V) are denoted as horizontal rows. The vertical lines depict the electrical activity of the heart over time. Impulse formation is depicted as a circle. Atrial activation is shown by a circle and line that traverses the atria and then the atrioventricular node. The time for atrioventricular conduction is shown on the x-axis. The P waves occur at regular intervals of 270 milliseconds. A nonconducted P wave consistent with second-degree AVB is depicted by a short, perpendicular line that ends within the zone of the atrioventricular node, representing the cessation of conduction in that region. Each blocked P wave is followed by an escape complex coupled to a sinus-conducted complex. This pattern is termed escape-capture bigeminy. Paper speed = 50 mm/s; 2 cm = 1 mV.
to illustrate the mechanisms of the arrhythmia. The ladder diagram differentiates between impulses that originate from the atria and those that originate from the ventricles and indicates their subsequent conduction and direction of travel. For the cat of the present report, the ladder diagram revealed that the sinus complexes originated in the atria and were then conducted through the normal conduction system to the ventricles. This was followed by atrial depolarization that was not conducted through the atroventricular node to the ventricle. Thereafter, the ventricular depolarization associated with the escape complex traveled in a retrograde fashion and collided with another nonconducted atrial impulse. This sequence repeated throughout the ECG recording.

Discussion

The cat of the present report had a rare rhythm termed escape-capture bigeminy. There have been only a few other instances of escape-capture bigeminy reported in the veterinary medical literature. This rhythm was first described in 1958 and consists of either a nodal or ventricular escape beat paired with a conducted sinus beat. This rhythm is most commonly identified with underlying AVB, atrioventricular dissociation, or sinoatrial block. For this rhythm to occur, there must be a marked difference between the escape interval and the conducted intersinus interval, with the intersinus interval exceeding the sum of the escape interval and its refractory period. In the cat of the present report, the effective intersinus interval (820 milliseconds) was longer than the escape interval (420 milliseconds). Moreover, for this rhythm to persist, the escape complexes cannot change or alter the impulse generation of the sinus node through retrograde conduction. For this cat, the escape complexes did not activate the sinus node in a retrograde fashion because the ventricular impulse collided with the electrical wavefront generated by the nonconducted P wave. These 2 colliding wavefronts produced interference dissociation, which prevents contradirectional electrical stimulation. Because the ventricular escape complex does not reset or change the sinus node discharge, the P waves occur with perfect regularity.

Critical to interpretation of ECG findings is the underlying conduction system disease, because escape-capture bigeminy commonly occurs in the setting of conduction system disturbances. Causes of second-degree AVB include increased vagal tone, degenerative change (fibrosis), infiltrative disease (inflammatory or neoplastic), toxic effects of β-adrenoceptor antagonists or nondihydropyridine calcium-channel blockers, and hypertrophic cardiomyopathy. Up to 89% of cats with hypertrophic cardiomyopathy have been found to have histologic lesions within the conduction system. For the cat of the present report, the echocardiographic findings eliminated infiltrative disease and hypertrophic cardiomyopathy as potential causes for the AVB, and the cat had received no drugs prior to the development of the arrhythmia.

Hyperthyroidism is commonly associated with tachyarrhythmias, more specifically sinus tachycardia and atrial fibrillation. In people and cats, sinus tachycardia is the most common clinical sign of hyperthyroidism. In people, the prevalence of atrial fibrillation increases dramatically from 2.3% for persons with normal thyroid gland function to 13.8% for those with overt hyperthyroidism. Hyperthyroidism has also been reported to cause sick sinus syndrome, sinoatrial block, and first-, second-, and third-degree AVB in people; therefore, the direct cardiac effects of elevated thyroxine concentration include a positive chronotropic effect and a negative dromotropic effect. In addition, an increased grade of AVB in people may be a marker of an impending thyroid storm.

The etiopathogenesis of AVB with hyperthyroidism is not currently known, but there are several hypotheses. Excessive amounts of circulating thyroxine have been shown to induce interstitial inflammation within the conduction system and to cause focal inflammation or myocarditis within the myocardial tissue. Histologic findings in people with Graves disease and AVB favor this theory of a nonspecific inflammatory response within the conduction system. In patients with Graves disease, AVB has also been linked to infection, digitalis use, and hypercalcemia. The last theory suggests that hyperthyroidism excites the autonomic nervous system and aggravates an underlying preexisting hypervagotonia. There are no definitive case reports of hyperthyroidism causing AVB in the veterinary medical literature, but that suspicion was raised for 1 cat. In people, there is generally a return of normal atrioventricular conduction once euthyroidism has been reestablished.

The cat of the present report developed progressive conduction system disease and was found to have third-degree AVB 3 months after the initial evaluation. There were several possible mechanisms for the development of third-degree AVB in this case. First, Miller et al have proposed that there is no association between treatment of hyperthyroidism and progression of AVB, but rather there is either a spontaneous exacerbation or reemission of AVB with or without treatment. Another suggested mechanism for third-degree AVB development is that high thyroxine concentration increases atrioventricular conduction, and once euthyroid, the patient actually becomes predisposed to an increased grade of AVB because of the slowed conduction that occurs with resolution of excessive thyroxine concentration. Moreover, inflammatory lesions of the conduction system and myocardium secondary to hyperthyroidism in people may not be reversible; however, in cats, further studies into histologic lesions associated with hyperthyroidism would be needed. Lastly, the cat of the present report may have had a preexisting conduction system disease that progressed naturally.

References