A 14-year-old 2.7-kg (5.94-lb) spayed female domestic shorthair cat was examined at The Ohio State University Veterinary Medical Center because of weight loss of 6 months’ duration and decreased appetite of 3 days’ duration. The cat was not receiving any medication at the time of the evaluation. The cat’s medical history included a cystotomy for removal of uroliths performed 4 years prior to the evaluation. Pertinent physical examination abnormalities were a palpable thyroid gland nodule and abnormal cardiac auscultation characterized by an irregular rhythm and a bradycardia of 130 beats/min. The remainder of the physical examination findings were unremarkable.

A CBC, serum biochemical analysis, and urinalysis were performed and revealed no clinically notable abnormalities. Serum thyroxine (total T4) concentration was markedly high (10.5 µg/dL; reference interval, 1.0 to 3.0 µg/dL), consistent with hyperthyroidism. Indirect systolic arterial blood pressure (measured by means of a Doppler ultrasonographic method) was 130 mm Hg. Echocardiography was performed, and the heart appeared unremarkable with the exception of mild, 4-chamber dilatation. A standard 6-lead ECG was acquired to characterize the cat’s irregular rhythm and bradycardia (Figure 1).

**ECG Interpretation**

The ECG recording (Figure 1) was consistent with a distributional pattern termed escape-capture bigeminy, which in this cat was characterized by 3 factors: ventricular escape complexes, second-degree atrioventricular (AV) block, and the critical timing of the ventricular escape interval relative to the dominant sinus node discharge rate and generation of P waves. The atrial rhythm was sinus in origin and regular at a rate of 195 depolarizations/min, with a cycle duration of 310 milliseconds. The ventricular rhythm was irregular at a rate of 135 depolarizations/min and distinguished by a recurrent pattern of paired QRS complexes of 2 morphologies that were consistently followed by a pause. The upright, narrow QRS complexes (in lead II) represented sinus impulses following normal AV conduction. The wider, negative QRS complexes were escape complexes that follow a blocked P wave (second-degree AV block). The escape interval from the normally conducted QRS complexes was consistently 520 milliseconds, which was equivalent to an instantaneous rate of 115 depolarizations/min. The origin of the escape complexes was most likely ventricular, but a junctional origin with a conduction abnormality was also considered.

On close examination of the ECG recording, it was determined that 2 nonconducted P waves followed every apparently normal P-QRS-T complex. The first blocked P wave was identified immediately after the T wave of the conducted QRS complexes. However, the second blocked P wave could not be identified in the surface ECG traces, and it was deduced from the dominant and regular sinus rhythm (Figure 2). The escape-capture sequence was fulfilled by the timing of the next sinus P wave, which was conducted normally into the ventricle to produce a so-called sinus capture with a normal QRS complex.

In classifying the conduction sequence for the second-degree AV block, the presumption is a 3:2 ratio of P waves to QRS complexes, as described in the human medical literature1,2 because for each set of 3 P waves, 2 QRS complexes are evident. However, for the cat of this report, a typical 3:2 conduction sequence was not evident because one of the QRS complexes was a ventricular escape. The atrial-to-ventricular depolarization ratio was 3:2, but the AV conduction ratio was 3:1 because...
only 1 P wave was conducted to the ventricles and 2 P waves were blocked. The authors’ interpretation was that 2 mechanisms were likely operational for the AV block. The first P wave was blocked as a result of pathological changes in the AV conduction system, whereas the second P wave was blocked physiologically because of interference dissociation following retrograde conduction of the ventricular escape complex into the AV conduction system. The proposed mechanisms were illustrated in a ladder (Lewis) diagram (Figure 2).

The cat was administered methimazole (0.9 mg/kg [0.4 mg/lb], PO q 24 h for 14 days, then q 12 h thereafter), and a repeated ECG examination was performed after 2 months of treatment (Figure 3). At this time, the cat was euthyroid (serum thyroxine concentration, 2.4 µg/dL), and its AV conduction disease had progressed to third-degree AV block. The cat’s appetite was much improved and weight gain was noted (weight, 3.1 kg [6.8 lb]).

Discussion

The cat of the present report had a rarely reported, bigeminal rhythm called escape-capture bigeminy. First described by Bradley and Marriott3 in 1958, escape-capture bigeminy can only occur when there is marked discrepancy between the escape interval and the effective intersinus interval; the effective intersinus interval is defined as the time between conducted sinus impulses.2 Although several forms of escape-capture bigeminy are known, including ventricular or atrial escape-capture bigeminy, the most commonly reported form in people occurs with sinus node dysfunction (with sinus bradycardia or sinoatrial block) or second-degree AV block.1 Requirements for escape-capture bigeminy to endure are that the effective intersinus interval must exceed the sum of the escape interval and the refractory period following the escape complex and that the escape complex cannot alter the sinus node’s cycle duration (eg, through retrograde AV nodal conduction).2,4 For the cat of the present report, the effective intersinus interval (920 milliseconds) was longer than the escape interval (520 milliseconds) and the refractory period as a result of the second-degree AV block, and the ventricular escape complexes did not alter the sinus cycle duration because of the lack of ventricular-to-atrial conduction secondary to interference dissociation. As a result of this physiologic collision, there was mutual extinction of the opposing electrical wavefronts, which occurred within the AV conduction system in this case (Figure 2).3 Because of the failure of the escape complexes to traverse the AV node, the sinus node pacemaker could

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**Figure 2**—Lead II ECG tracing from the ECG recordings in Figure 1 and a Lewis (ladder) diagram of the cat’s rhythm disturbance. In the ladder diagram, the upper zone represents atrial activation (A), the middle zone represents AV conduction (AV), and the lower zone represents ventricular activation (V). The P waves can be inferred to consistently appear in the trace at a regular interval of 310 milliseconds. Each sinus impulse is always followed by a blocked P wave (arrow), and the blocked P wave is then followed by a ventricular escape complex that occurs simultaneously with a sinus-initiated P wave (asterisk) within the AV nodal region. The pattern in which an escape complex is always followed by a sinus capture has been termed escape-capture bigeminy. Paper speed = 50 mm/s; 1 cm = 1 mV.

**Figure 3**—Repeated 6-lead ECG tracings obtained from the cat in Figures 1 and 2 after treatment with methimazole for 2 months. At this time, a euthyroid state had been restored. Notice that the cat’s AV conduction disease has progressed from second-degree AV block to third-degree, or complete, AV block. Paper speed = 50 mm/s; 2 cm = 1 mV.
operate uninterrupted, which permitted regular P waves to march through the ECG traces at a regular interval of 310 milliseconds. The P wave following each conducted QRS complex was clearly blocked, and it can be presumed that the next P wave was blocked physiologically as a result of superimposition of the atrial and ventricular activities. Although this is a plausible explanation (Figure 2), it cannot be proven without an intracardiac electrophysiologic assessment. The alternative consideration is that the cat had 3:1 block in which 2 P waves are anatomically blocked within the AV conduction system and 1 P wave was conducted.

In the veterinary medical literature, there are 2 previously published ECG reports—both of which involved cats—that represent other examples of escape-capture bigeminy. However, the concept of escape-capture bigeminy was not discussed in either of those publications. The relatively fast ventricular escape rhythm identified in the cat of the present report and the other published examples is a common feature of cats with bradyarrhythmias and is likely instrumental in maintaining this rhythm.

It is uncertain what role hyperthyroidism played in the conduction disease of the cat in the present report. The association between AV block and cats with hyperthyroidism has been reported, but a cause-and-effect relationship has not been established. Treatment of this cat's hyperthyroidism would be expected to improve the rhythm disturbance if the hyperthyroidism was an important contributing factor because arrhythmias in cats, in general, are reported to resolve following management of hyperthyroidism. There are several case reports of humans with thyrotoxicosis who had concurrent second- or third-degree AV block, yet the mechanism by which the 2 conditions are related remains unclear and speculative. In most human cases, the AV block resolved following treatment of the thyrotoxicosis.

It is possible that the thyrotoxicosis in the cat of the present report actually improved AV conduction. Once euthyroidism (serum thyroxine concentration, 2.4 µg/dL) was restored after 2 months of treatment with methimazole, the cat's AV conduction disease progressed to third-degree AV block (Figure 3). Thyrotoxicosis results in an exaggerated response to catecholamines, including an increase in the number of or affinity of catecholamines for cardiac β-adrenergic receptors. This, in turn, might have led to somewhat enhanced AV conduction during the hyperthyroid state and, conversely, decreased AV conduction once euthyroidism was achieved. Alternatively, the conduction disease might have simply progressed over time.

In cats, second- and third-degree AV block can be associated with drug toxicoses (eg, β-adrenergic receptor antagonists, digoxin, and nondihydropyridine calcium channel blockers), treatment with sedatives or anesthetic agents (eg, α1-adrenergic receptor agonists), or structural heart disease, including cardiomyopathies, infiltrative disease (inflammatory or neoplastic), endo- or myocarditis, infarction, or fibrosis (often with a concurrent cardiomyopathy). In a case series of cats with third-degree AV block, most cats had cardiomyopathy. Studies of the conduction system in cats with hypertrophic cardiomyopathy and third-degree AV block revealed considerable degeneration and fibrous replacement of the AV conduction system, which was thought to develop secondary to the effects of hypertrophic cardiomyopathy and aging. However, given the lack of an identifiable cardiomyopathy in the cat of the present report, age-related degeneration or fibrosis of the AV conduction system was suspected as the primary mechanism. Because of the lack of clinical signs, treatment for the rhythm disturbance was not initiated. At the last recheck examination (8 months after initial evaluation), the cat had no clinical signs with complete AV block and a stable escape rate of 120 beats/min.

References