ECG of the Month

The Academy of Veterinary Cardiology sponsors this feature. Readers of the *JAVMA* are invited to submit contributions. Contributions should include a brief description of the case (150 words); good quality contrast glossy photographs (5 X 7 in) of tracings, with the components of a QRS complex labeled; figure legends with information on ECG lead, paper speed, and voltage calibration; an ECG interpretation; and a discussion of the abnormality. Two hard copies of the manuscript and each figure must be submitted, along with an electronic copy on a 3.5-in PC-formatted disk. Submissions that are complete will be sent to the feature coordinator, Dr. Robert Hamlin, at The Ohio State University for review.

A 13-year-old neutered male European Shorthair cat was evaluated at the University of Berne Small Animal Clinic because of suspected pleural effusion. The cat's general physical condition was poor; its respiration was labored with open-mouthed breathing occasionally. The respiratory rate was 64 breaths/min, femoral pulse rate was 240 beats/min, and rectal temperature was 38.6°C (101.5°F). Palpation of the ventrocervical region of the neck revealed a mass (2 X 1 cm). On auscultation of the thorax, the heart sounds appeared to be dull and arrhythmic, but no distinct heart murmur could be detected. The respiratory sounds were muffled in the lower third portion of the thorax, and the percussion sounds were also dull over the lower half of the thoracic wall. No treatment had been administered to the cat. Thoracocentesis was performed, and approximately 300 mL of milky-white fluid was withdrawn. Subsequently, an ECG examination was performed with the cat in right lateral recumbency. Tracings from leads I, II, III, aVR, aVL, and aVF were recorded for approximately 2 minutes. Cardiac ultrasonography revealed a massive dilatation of all 4 chambers of the heart and moderate hypokinesis of the left ventricle. Serum **thyroxine (T_4)** concentration was 110 nmol/L (reference range, 17 to 48 nmol/L). The cardiac abnormalities were interpreted as an intermediate form of cardiomyopathy with signs of systolic pump failure and likely chronic myocardial damage associated with hyperthyroidism. The prognosis was considered to be poor, and the cat was euthanized. A necropsy could not be performed.

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ECG Interpretation

Examination of the ECG tracings (**Fig 1**) obtained after thoracocentesis and before the ultrasound examination revealed tachycardia of 260 beats/min with periodic short pauses (ie, missing QRS complexes), which was interpreted as being of supraventricular origin. On the slow-speed ECG recordings (5 mm/s), the periodicity of the missing QRS complexes was evident (**Fig 2**). The QRS complexes after the short pauses were preceded by P waves of normal appearance. The QRS complexes of leads II and aVF (not shown) were positive with normal amplitudes, but there was some notching of the R waves. Leads I (R wave of 1.1 mV) and aVL (not shown) were also positive. The calculated mean electrical axis was 18° in the frontal plane (reference range, 0° to 160°).

On careful examination of the QRS-T complexes, P waves were detected in the preceding ST segments. They appeared regularly at a rate of approximately 300 P waves/min (PP intervals of 0.2 seconds). With the aid of a ladder diagram (**Fig 3**), a progressive prolongation of the PQ interval from 0.06 to 0.16 seconds followed

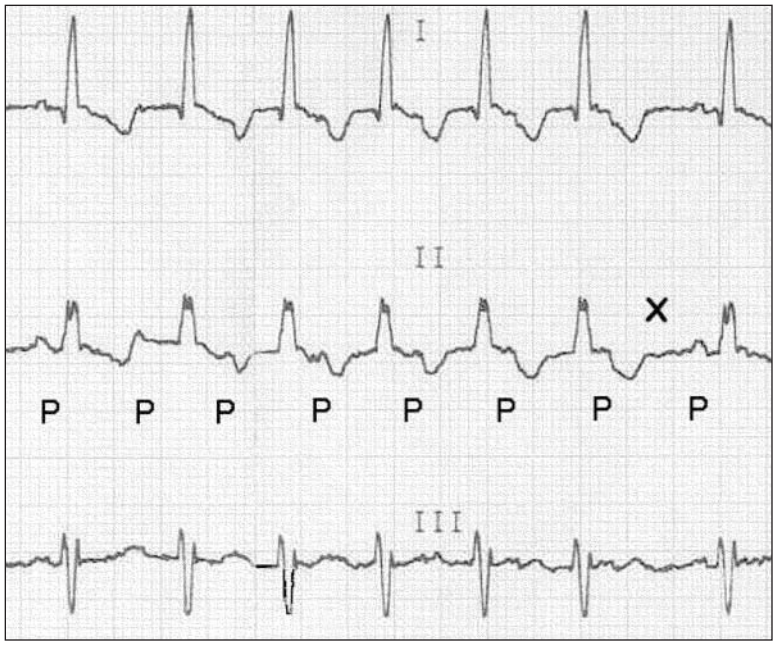

Figure 1—Electrocardiographic tracings (leads I, II, and III) recorded from a 13-year-old cat with pleural effusion and hyperthyroidism. Notice that the heart rate is 260 beats/min and the dominant rhythm is a supraventricular tachycardia. In the lead-II tracing, a distinct P wave associated with a QRS complex of normal appearance is detected at only every seventh heartbeat (to right of X). The QRS complexes in the lead-I and -II tracings are positive and notched, but the lead-III tracing has deep S-waves. Paper speed = 50 mm/s; 10 mm = 1 mV.

by an isolated P wave without a QRS complex (ie, second-degree **atrioventricular [AV]** block) was detected at every seventh heartbeat. This irregularity was detected throughout the ECG tracing. Therefore, the final ECG diagnosis was supraventricular (likely sinus) tachycardia with second-degree AV block Mobitz type I; the ratio of P waves to QRS complexes was fixed at 7:6 (Wenckebach's periodicity).

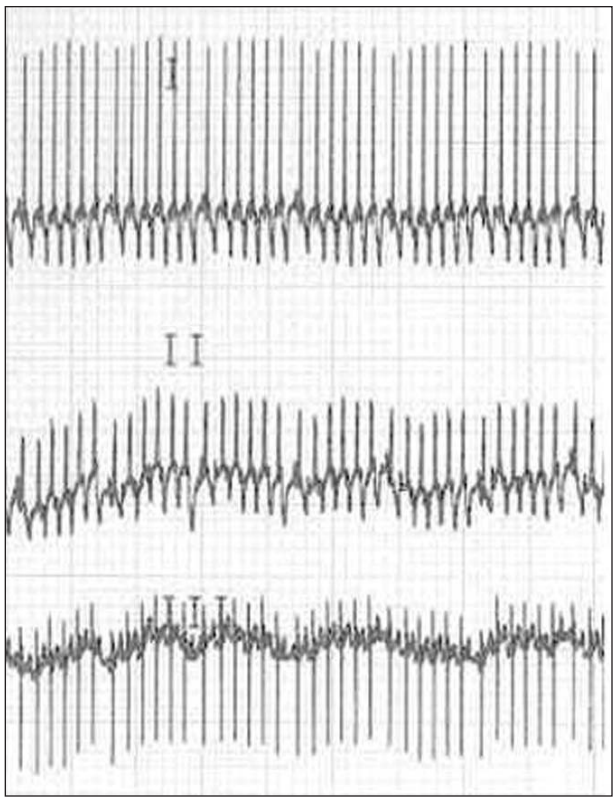

Figure 2—Electrocardiographic tracings (leads I, II, and III) recorded from a 13-year-old cat with pleural effusion and hyperthyroidism. Notice the regular irregularity of the cardiac rhythm; after 6 ventricular complexes, a slightly longer RR interval is detected. Paper speed = 5 mm/s; 10 mm = 1 mV.

Discussion

Second-degree AV block is rarely observed and reported in cats, and possible causes that have been suggested include inflammatory or degenerative myocardial lesions of the conduction tissues associated with cardiomyopathy,[1] toxoplasmosis, and feline infectious peritonitis.[2] Rishniw et al[3] reported a case of a cat with second-degree AV block associated with nonclinical hyperthyroidism. Fife and Cote[4] described a cat with second-degree AV block and no clinical signs of disease. In a report by Johnson and Sisson,[5] 2 cats with clinical signs associated with second-degree AV block were described; 1 of the cats had hyperthyroidism, whereas the other cat had dilated cardiomyopathy of unknown origin. All of these cats had second-degree AV block Mobitz type II with slow sinus rates and an occurrence of ventricular escape beats. To our knowledge, only 1 case of second-degree AV block Mobitz type I with Wenckebach's periodicity in a cat has been reported[6]; however, that was an artificially induced arrhythmia. The Wenckebach's periodicity (or phenomenon) is characterized by a supraventricular rhythm, commonly of sinus origin, with normal or prolonged PR intervals (underlying first-degree block is most common); with each successive beat, the PR interval gradually lengthens until a beat is dropped.

Without doubt, to convincingly prove a periodicity according to Wenckebach's phenomenon, it is sometimes difficult to identify the progressive prolongation of the PQ interval associated with first-degree AV block until the blocked conduction and dropped beat occur. The high heart rate in the cat of this report may have helped to make the progressiveness of slowed AV conduction more easily detectable. Likely, the high rate of supraventricular impulses prevented the occurrence of ventricular escape beats; in cats with complete AV block, escape rhythms with 60 to 100 ventricular beats/min have been recorded and such escape beats would have escape intervals of 600 to 1,000 milliseconds. We speculated that hyperthyroidism in the cat of this

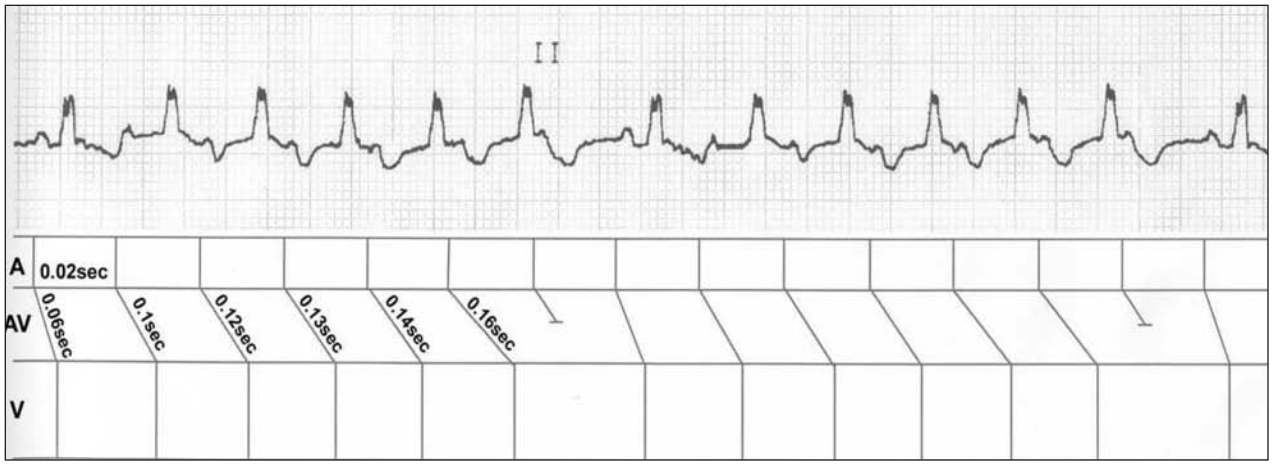

Figure 3—Lead-II ECG tracing recorded from the same cat as that in Figures 1 and 2, with a ladder diagram. Vertical lines in the ladder diagram mark the beginning of the P waves (A level) and QRS complexes (V level); connecting lines between the vertical lines indicate atrioventricular (AV) conduction (AV level). Notice that P waves are detected in the preceding ST-segments and occur regularly at a rate of approximately 300 P waves/min (PP intervals of 0.2 seconds). At every seventh heartbeat, a progressive prolongation of the PQ interval (from 0.06 to 0.16 seconds) is evident; it is followed by a pause and an isolated P wave without a QRS complex (ie second-degree AV block). Paper speed = 50 mm/s; 10 mm = 1 mV.

report may have contributed to our observations. First, the high supraventricular (presumably sinus) rate may have been a consequence of hyperthyroidism because the high serum T_4 concentration is known to directly stimulate pacemaker cells and the release of endogenous catecholamines[7]; the latter are additional stimuli for increased rates of pacemaker cells. However, this stimulation was insufficient to increase potential ventricular escape foci rates to a level at which they could interfere with the intrinsic rhythm. Feline ventricular myocardial cells probably have an upper limit of intrinsic (escape) pacemaker rates of approximately 100 beats/min. Second, a consequence of hyperthyroidism may have been an improvement of the underlying AV conduction that allowed it to conduct for up to 160 milliseconds during periods of second-degree AV block. The second-degree AV block detected in the cat of this report is somewhat similar to the physiologic episodes of second-degree AV block that occur at high heart rates in some horses. Interestingly, the notching of the QRS complex observed in this cat is an ECG abnormality that is frequently detected in felids with cardiomyopathy. It is usually explained as a conduction abnormality caused by scarring of the myocardium and is not of major clinical importance.

References

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