# ECG of the Month

A 7-year-old 4.7-kg (10.3-lb) neutered male Russian Blue cat was examined by the referring veterinarian because of lethargy and inappetence of 2 days' duration. Cardiac auscultation revealed the only abnormality identified during physical examination, which was an irregular cardiac rhythm with a frequency of approximately 300 beats/min and interrupted with periods of distinctly lower (albeit undocumented) frequency. The cat had no history of cardiac abnormalities. The cat was immediately referred to the emergency service of a veterinary teaching hospital.

At the referral evaluation, the cat was bright, alert, and responsive and had a body condition score of 5/9. No signs of respiratory distress were evident; respiration was costoabdominal in nature at a frequency of 42 breaths/min. The femoral pulses were fair, regular, symmetric, and synchronous with the heartbeat; pulse rate was 210 beats/min. Cardiac auscultation revealed a regular rhythm with a frequency of 210 beats/min; no murmur or gallop was detected. The cat's mucous membranes were pink with a capillary refill time < 1 second. The rectal temperature was 38.6°C (101.5°F).

This report was submitted by Viktor Szatmári, DVM, PhD; from the Department of Clinical Sciences, Faculty of Veterinary Medicine, Utrecht University, 3584 CM, Utrecht, Netherlands.

Address correspondence to Dr. Szatmári (v.szatmari@uu.nl).

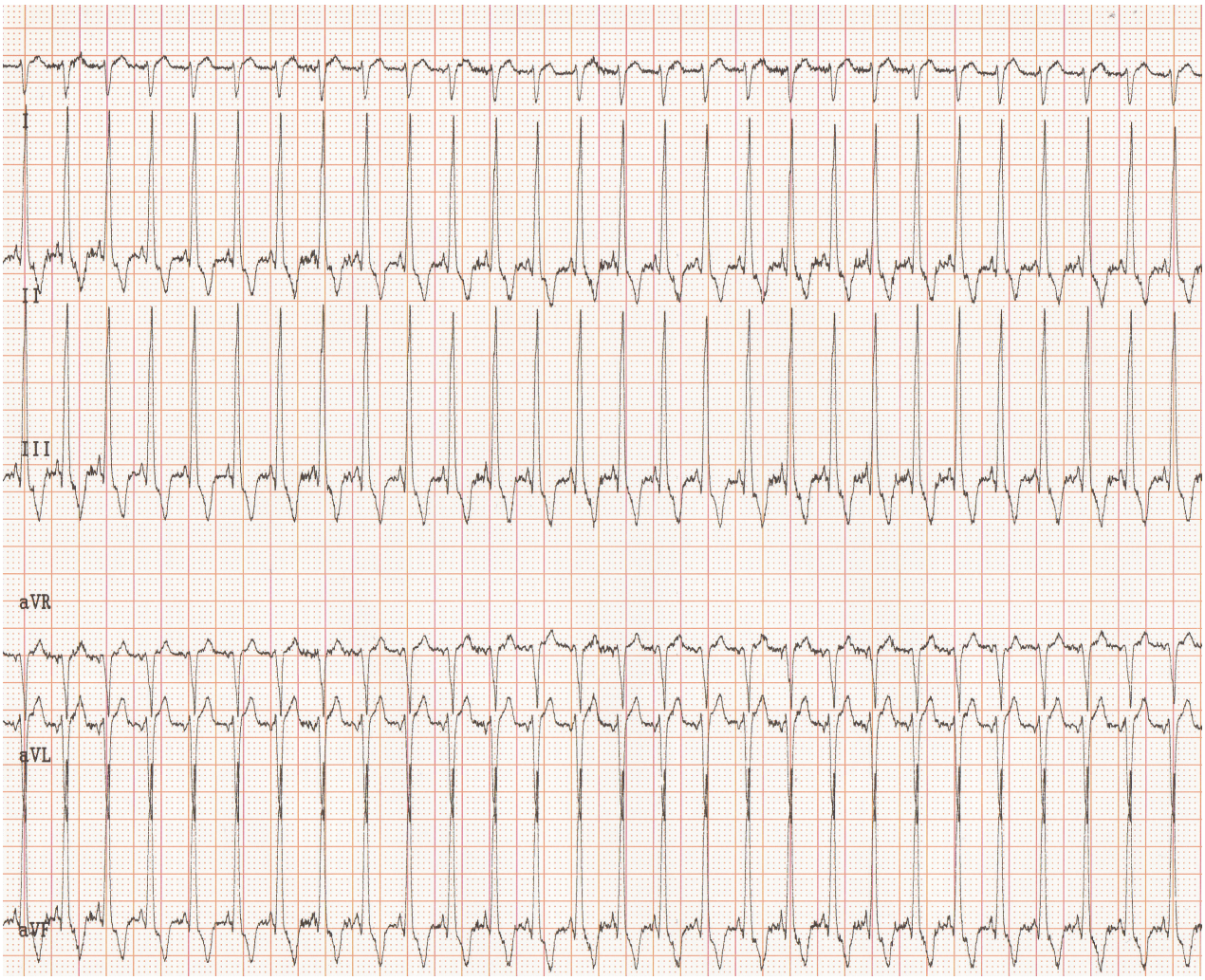


**Figure 1**—Portion of a 6-lead surface ECG obtained from a Russian Blue cat for which an irregular cardiac rhythm had been identified by the referring veterinarian during an evaluation because of lethargy and inappetence of 2 days' duration. This portion of the ECG recording shows a sinus rhythm. Notice the short PQ intervals with abnormally wide and tall QRS complexes and discordant T waves, compatible with a left bundle branch block configuration. In the upstroke segment of the R waves, a subtle notch (splintering) is apparent. These findings are indicative of ventricular preexcitation as a result of an accessory atrioventricular pathway. Paper speed = 25 mm/s; 1 cm = 1 mV.

The initial diagnostic evaluation of the cat included clinicopathologic analyses. The results of a CBC were unremarkable. Plasma biochemical abnormalities included mild hypokalemia (3.1 mmol/L; reference range, 3.4 to 5.2 mmol/L), mild hypocalcemia (2.25 mmol/L; reference range, 2.36 to 2.86 mmol/L), mild hypophosphatemia (0.77 mmol/L; reference range 0.89 to 2.05 mmol/L), and mild hyperproteinemia (79 g/L; reference range, 54 to 70 g/L). Standard 6-lead ECG was performed for evaluation of the previously identified tachyarrhythmia.

## ECG Interpretation

A 6-lead ECG recording was obtained with the cat in right lateral recumbency. In the approximately 5-minute ECG recording, 3 types of pattern were identified, each of which showed a sinus rhythm with an identical frequency of 200 beats/min. The first portion of the recording **(Figure 1)** had short PQ intervals (0.04 seconds; reference range, 0.05 to 0.09 seconds) and abnormally wide and tall QRS complexes (QRS-complex duration, 0.07 seconds [reference range, 0.02 to 0.04 seconds]; R-wave amplitude, 2.7 mV [reference range, 0.18 to 0.90 mV]) with discordant T waves, compatible with a left bundle branch block morphology.[1,2] The R waves had a subtle notch (splintering) in their upstroke segment.[2] This pattern spontaneously converted to a sinus rhythm with physiologic PQ intervals and narrow QRS complexes of normal appearance with concordant T waves **(Figure 2)**. Finally, a ventricular pseudobigeminy pattern appeared, which was characterized by a combination of the 2 previously observed types of PQRST complexes **(Figure 3)**. This third ECG pattern had alternating short and physiologic PQ intervals (0.04 and 0.07 seconds, respectively). The physiologic PQ intervals were followed by QRST complexes of normal appearance (QRS-complex duration, 0.025 seconds; R-wave amplitude, 0.9 mV); however, the short PQ intervals were followed by abnormally

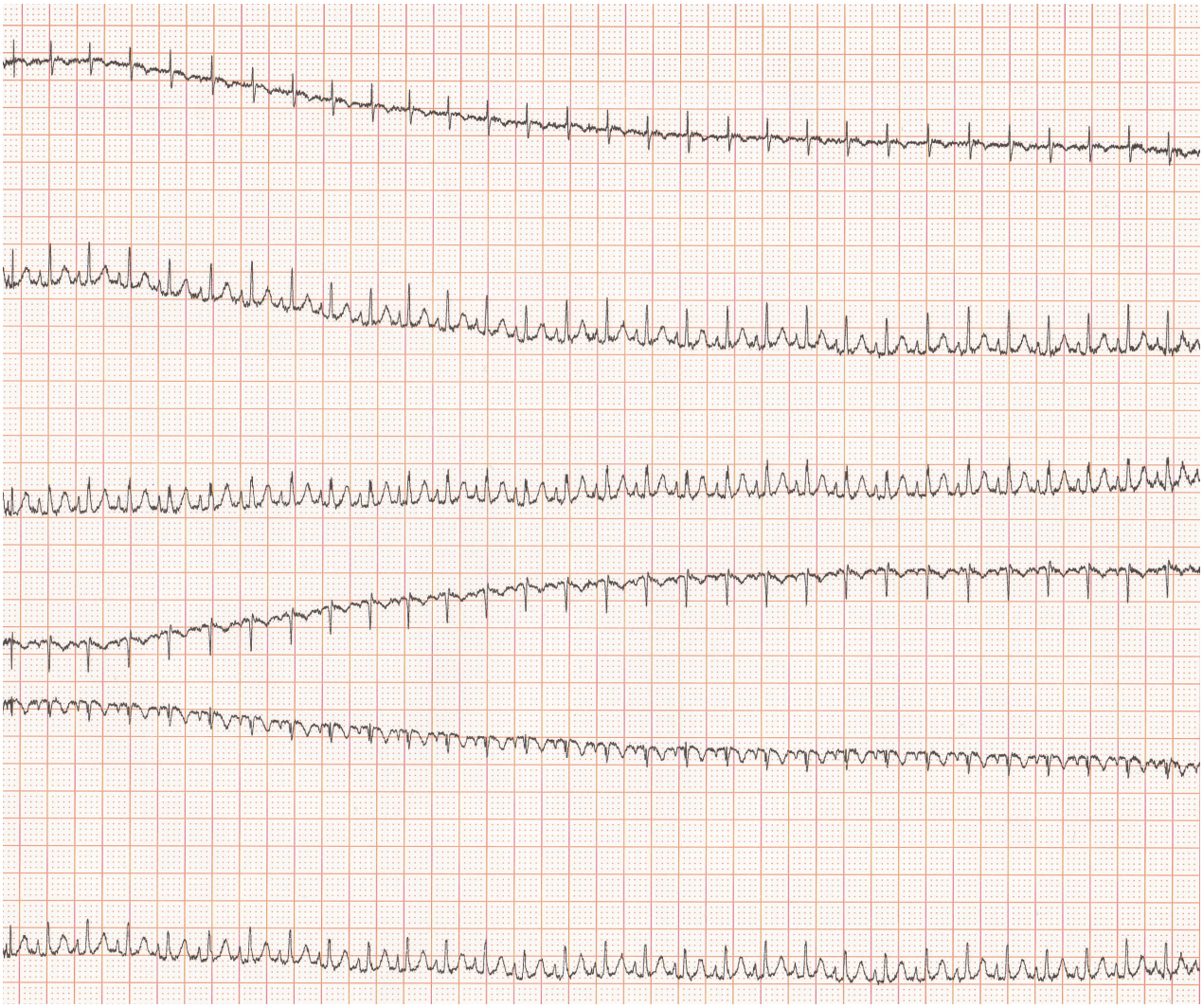

**Figure 2**—Another portion of the 6-lead surface ECG obtained from the same cat a few minutes after that in Figure 1. The leads displayed from the top to the bottom are I, II, III, aVR, aVL, and aVF. This portion of the ECG recording has evidence of a sinus rhythm with physiologic PQ intervals, QRS complexes of normal appearance, and concordant T waves. Paper speed = 25 mm/s; 1 cm = 1 mV.

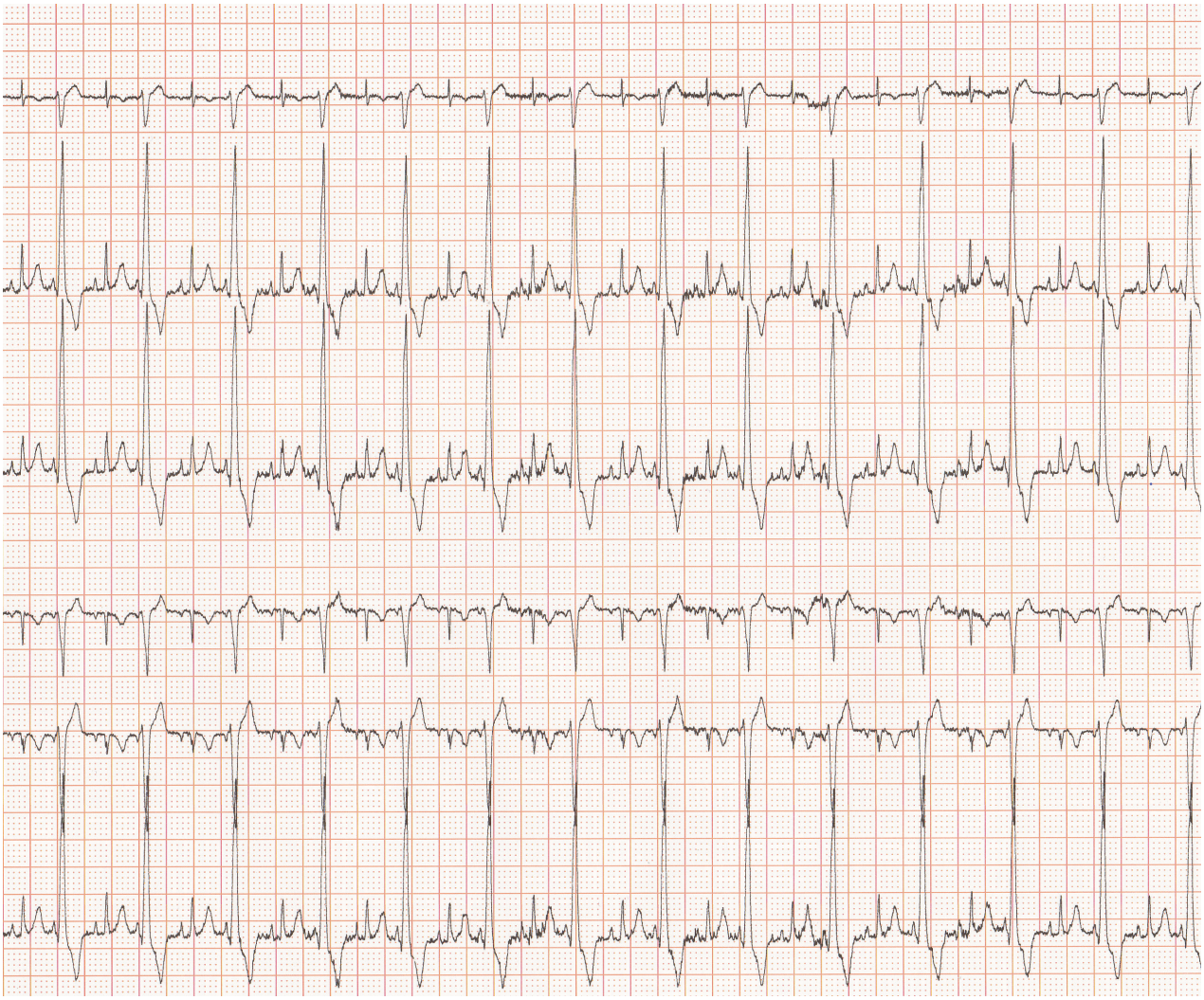

**Figure 3**—Another portion of the 6-lead limb surface ECG obtained from the same cat a few minutes after that in Figure 2. The leads displayed from the top to the bottom are I, II, III, aVR, aVL, and aVF. This portion of the ECG recording has evidence of a sinus rhythm with a ventricular pseudobigeminy pattern. Notice the 2 types of PQ intervals (short or physiologic) and the corresponding QRS complexes (abnormally wide and tall or narrow [ie, normal], respectively) that appear in an intermittent, alternating pattern. These findings are indicative of intermittent ventricular preexcitation. Paper speed = 25 mm/s; 1 cm = 1 mV.

wide and tall QRS complexes (QRS-complex duration, 0.07 seconds; R-wave amplitude, 2.7 mV) with discordant T waves. The mean electrical axis of both types of QRS complexes was within the reference range (0° to +160°); however, the abnormally wide and tall QRS complexes had a right shift, compared to the narrow complexes. In all recordings, the narrow QRS complexes had concordant T waves, whereas the abnormally wide and tall QRS complexes had discordant T waves. The intermittent fixed, short PQ intervals with the aberrant (wide, tall, and splintered) QRS complexes and discordant T waves were compatible with ventricular preexcitation as a result of the presence of an accessory atrioventricular (AV) pathway.[2-5]

## Discussion

Ventricular preexcitation is a rare conduction abnormality in cats.[2,4,5] Normally, the atria are electrically isolated from the ventricles by fibrous tissue, the so-called skeleton, and electrical activity travels from the atria to the ventricles exclusively through the AV node.[2] The AV node causes a physiologic conduction delay (which is represented by the PQ interval on a surface ECG tracing) so that the ventricles have sufficient time to fill with blood during diastole. Accessory pathways are congenital muscular bundles that provide an additional electrical atrial-ventricular conduction pathway near the AV node; these pathways are not associated with a conduction delay.[2-5] The lack of conduction delay appears on ECG tracings as shortened PQ intervals. Accessory pathways can provide antegrade (AV) or retrograde (ventriculoatrial) electrical conduction. In the cat of the present report, the AV conduction occurred intermittently, at times exclusively via the AV node (Figure 2) and at other times also via the accessory pathway (Figure 1). The accessory pathway in this cat most likely had an extranodal anatomic

localization, relatively far from the normal ventricular conduction system (the bundle of His). This supposition was based on the fact that QRS complexes during ventricular preexcitation had a wide and aberrant appearance, compatible with prolonged ventricular cell-to-cell depolarization (bundle branch block morphology), as opposed to physiologic rapid depolarization via the bundle of His and the left and right bundle branches, which would result in narrow QRS complexes.[2–5] The subtle notch of the wide and tall QRS complexes was most likely the result of a delta wave.[2–5] A delta wave can be seen in the upstroke segment of R waves (in lead II) in instances of ventricular preexcitation, caused by simultaneous ventricular depolarization via the AV node and accessory pathway, resulting in a sort of fusion beat.[2–5] The intermittent, alternating beat nature of ventricular preexcitation in the cat of the present report was a unique finding, although the phenomenon in a human has been described as a ventricular pseudobigeminy pattern.[3] It was difficult to find a plausible electrophysiologic explanation for the existence of this pattern. The presence of identical PQ intervals preceding all wide and tall QRS complexes made a true ventricular bigeminy (ie, alternating sinus and ventricular premature complexes) unlikely.[2,3] The presence of fixed PQ intervals with each wide and tall QRS complex was proof that these QRS complexes resulted from physiologic atrial depolarization; therefore, the possibility of an AV dissociation could be excluded.[2] In instances of an (isorhythmic) AV dissociation or a true ventricular bigeminy, the PQ intervals of the wide and tall QRS complexes would be variable because of the presence of 2 independent, competing foci—the sinus node and the ventricular ectopic focus—with frequencies that can never be exactly the same. Because the ventricular frequency (120 to 240 beats/min) was physiologic in the cat of the present report, a run of (ventricular) tachycardia was excluded in the first ECG pattern (Figure 1) simply on the basis of the frequency. In addition, in instances of ventricular ectopic beats (such as those associated with an idioventricular rhythm), occasional fusion beats would be expected with a different type of QRST-complex morphology. The different polarity and amplitude of the T waves of the narrow and the wide and tall QRS complexes reflect the difference in ventricular repolarization, which is a consequence of the difference in ventricular depolarization.[2] The morphological features of the T waves of the wide and tall QRS complexes are identical when ventricular depolarization does not take place through the physiologic conduction system, such as occurs with bundle branch blocks, ventricular premature beats, and ventricular escape complexes and in instances of ventricular preexcitation.[2]

Ventricular preexcitation can be clinically silent; however, at times, paroxysmal supraventricular tachycardia can occur as a result of an orthodromic AV reciprocating tachycardia.[2–5] These periods of tachycardia are usually the result of a macroscopic reentry circuit that uses the 2 anatomic electrical connections (the AV node and the accessory pathway) between the atria and ventricles, wherein anterograde conduction takes place via the AV node and retrograde conduction takes place via the accessory pathway.[2,4,5] No periods of paroxysmal tachycardia were identified for the cat of the present report during the ECG recording; therefore, no antiarrhythmic treatment was initiated. Nevertheless, the most likely explanation for the tachycardia detected by the referring veterinarian was suspected orthodromic AV reciprocating tachycardia.[2–5] The suspected paroxysmal tachycardia could have caused the cat's nonspecific clinical signs, or it might have been a coincidental finding. Because the cat recovered spontaneously within a day, the owner declined further cardiac investigations, such as Holter ECG monitoring and echocardiography.

## Acknowledgments

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