# ECG of the Month

A 10-year-old 3.9-kg (8.6-lb) spayed female domestic shorthair cat underwent a recheck evaluation because of hypertrophic obstructive cardiomyopathy with severe left atrial enlargement. At the time of the recheck evaluation, the cat had had 2 previous episodes of congestive heart failure (1 year and 1 month earlier). Previous ECG examinations revealed sustained supraventricular tachycardia with intermittent ventricular premature complexes (VPCs), all of which improved with sotalol treatment. Other than cardiovascular disease, the cat had a history of chronic interstitial kidney disease and episodes of acute kidney injury related to pyelonephritis. The cat was being treated orally with furosemide (0.77 mg/kg [0.35 mg/lb], q 12 h), sotalol hydrochloride (2.1 mg/kg [0.95 mg/lb], q 12 h), rivaroxaban (0.64 mg/kg [0.29 mg/lb], q 24 h), clopidogrel bisulfate (4.8 mg/kg [2.18 mg/lb], every other day), benazepril hydrochloride (0.32 mg/kg [0.15 mg/lb], q 24 h), and maropitant citrate (1.0 mg/kg [0.45 mg/lb], q 24 h, as needed). The recheck evaluation was considered a routine follow-up; the owners had been giving the cat its treatments as prescribed, and the cat had been doing well at home with no clinical signs.

On physical examination, the cat was bright, alert, and responsive; heart rate, respiratory rate, and rectal temperature were within reference intervals. Cardiac auscultation revealed no murmur or gallop sound. The cat's heart rate was 150 beats/min, and the heart rhythm was noted to be irregular. Systolic blood pressure measured by means of Doppler ultrasonography was 120 mm Hg. Two-view thoracic radiography was performed and revealed cardiomegaly with no evidence of congestive heart failure. A CBC was performed, and all variables were within reference intervals. Serum biochemical analyses revealed mild hyperglycemia (137 mg/dL; reference interval, 7 to 126 mg/dL), mild azotemia (BUN concentration, 36 mg/dL [reference interval, 18 to 33 mg/dL]; creatinine concentration, 1.5 mg/dL [reference interval, 0.7 to 1.8 mg/dL]), and mild hypermagnesemia (2.7 mg/dL; reference interval, 1.8 to 2.6 mg/dL). Serum thyroxine ($T_4$) concentration was considered normal (2.8 µg/dL; reference interval, 1.0 to 3.9 µg/dL). Six-lead ECG was performed to evaluate the cat's irregular heart rhythm.

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## ECG Interpretation

A 6-lead ECG recording **(Figure 1)** revealed an underlying sinus arrhythmia with a mean heart rate

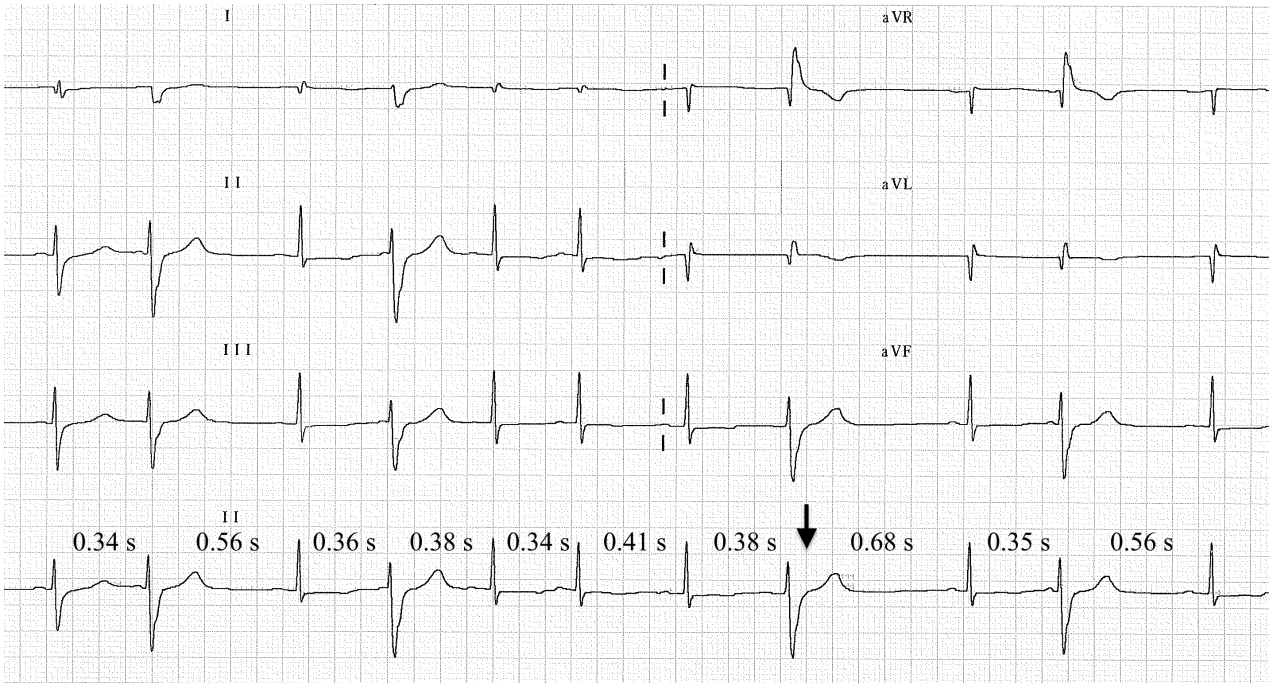


**Figure 1**—Six-lead ECG tracing obtained from a cat with hypertrophic obstructive cardiomyopathy and severe left atrial enlargement and a history of ventricular premature complexes and supraventricular tachycardia. The cat also had a history of chronic interstitial kidney disease and episodes of acute kidney injury related to pyelonephritis. The cat underwent ECG examination after an arrhythmia was auscultated during a recheck evaluation. Notice that the underlying sinus arrhythmia is interrupted by premature complexes with right bundle branch (RBB) morphology following so-called long-short R-R interval cycles. The durations (in seconds) of the R-R intervals are indicated between QRS complexes. The eighth QRS complex (arrow) does not have a distinct P wave associated with it. These Ashman phenomenon–like findings are indicative of a phase 3 conduction aberrancy caused by long-short R-R cycles. Paper speed = 50 mm/s; 1 cm = 1 mV.

of 140 beats/min. Normal sinus P-QRS-T complexes were identified on the basis of upright P waves (amplitude, 0.1 mV; duration, 0.04 seconds), apparently normal PR intervals (0.10 seconds), and tall, narrow QRS complexes (amplitude, 1.0 mV; duration, 0.03 seconds) in lead II. Premature complexes interrupted the sinus arrhythmia, resulting in a so-called long-short R-R cycle in which 2 sinus beats with a long R-R interval were followed by a premature complex with a short R-R interval. The premature complexes had P waves with a short PR interval (0.06 seconds) and wide, bizarre QRS complexes (duration, 0.08 seconds). The premature complexes were consistent with atrial premature complexes (APCs) of right bundle branch (RBB) block morphology and were characterized by wide QRS complexes (duration, 0.08 seconds) and deep S waves in leads I, II, III, and aVF. The mean electrical axis was deviated to the right. Identical wide and bizarre complexes were evident during similar long-short cycles, but without association to distinct P waves.

## Discussion

In 1947, Gouaux and Ashman[1] described the Ashman phenomenon as ventricular conduction aberrancy associated with alterations in the refractory period attributable to variations in R-R cycle lengths in patients with atrial fibrillation. The R-R interval preceding a QRS complex will directly affect the refractory period of the conductive tissues, in that a long R-R interval results in a longer refractory period of the bundle branch fibers.[2] When an early impulse with a shorter R-R interval reaches the Purkinje fibers, the fibers are more likely to be in phase 3 of the preceding action potential, namely when potassium ions are flowing out of the cells. During this period, the membrane potential is repolarizing and the fibers are refractory to incoming impulses. The refractory period of the RBB is typically longer than that of the left bundle branch, and the RBB more commonly develops phase 3 conduction aberrancy.[2] When an RBB phase 3 conduction aberrancy occurs, the impulse is blocked at the level of the RBB but is conducted normally down the left bundle branch, through the left ventricle, then to the right ventricle, and in a retrograde manner up the RBB where it is terminated, resulting in a QRS complex of RBB block morphology **(Figure 2)**. When the following impulse arrives at the His bundle, the RBB is in its refractory period again and the conduction aberrancy recurs. The RBB block is terminated when a sufficiently long R-R interval allows the RBB time to repolarize and become susceptible to the subsequent impulse.

The cat of the present report had a history of VPCs and supraventricular tachycardia secondary to hypertrophic obstructive cardiomyopathy and se-

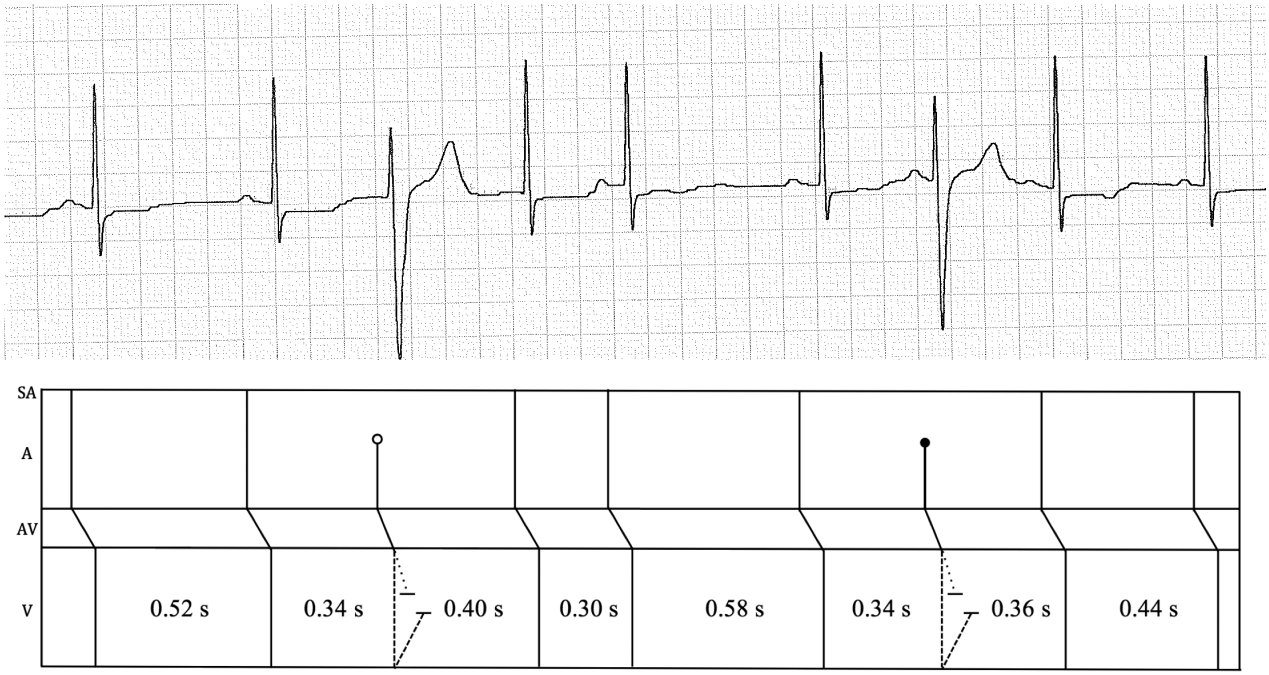


**Figure 2**—Lead II ECG tracing, obtained from the cat in Figure 1, with a corresponding Lewis (ladder) diagram of the proposed mechanism of aberrant conduction. In the ladder diagram, normal sinus beats begin at the sinoatrial node (SA) and are conducted through the atria (A) to the atrioventricular node (AV), down the His bundle, and through the ventricles (V). An atrial premature impulse originates in the atria (black circle) and conducts through the atrioventricular node but is blocked at the level of the RBB (dotted line) because the RBB is still in its effective refractory period. The impulse is conducted through the left bundle branch and left ventricle and then depolarizes the right ventricle, where it terminates (dashed line). The following sinus beat is conducted normally because there has been sufficient time for the RBB to repolarize. The third complex lacks a distinct P wave and originates at a supraventricular focus (white circle) away from the atrial premature complex focus but is conducted in similar fashion down the atrioventricular node. The durations (in seconds) of the R-R intervals are indicated. Paper speed = 50 mm/s; 1 cm = 1 mV.

vere left atrial enlargement. These arrhythmias were treated with sotalol, a potassium channel blocker with β-adrenoceptor blocking properties. Sotalol inhibits potassium ion channels, resulting in prolongation of phase 3 of the action potential and the effective refractory period.[3,4] In addition, the action of β-adrenoceptor blockers results in higher vagal tone, thereby promoting respiratory sinus arrhythmia.[4,5] For the cat of the present report, the sinus arrhythmia established by sotalol was interrupted by APCs, which were likely secondary to the cat's severe left atrial enlargement. The APCs had a short R-R interval following a longer R-R interval, which resulted in long-short cycles and wide QRS complexes of RBB block morphology. Although the traditional Ashman phenomenon was described as occurring during atrial fibrillation, the variable R-R interval and prolonged effective refractory period promoted by sotalol treatment in addition to the cat's underlying structural heart disease may have contributed to the Ashman phenomenon–like findings.

Throughout the ECG recording for the cat of the present report, there were wide, bizarre QRS complexes that were identical to the aberrantly conducted APCs but that lacked a distinct P wave. Two differential diagnoses for these complexes were supraventricular premature complexes or VPCs. If those complexes were supraventricular in origin, they originated from a focus separate from the APC focus. Like the APCs, the complexes had long-short cycles, resulting in aberrantly conducted complexes with RBB block morphology. However, these complexes could have been VPCs with RBB block morphology. If these complexes were VPCs, it was also possible that the APCs were instead a P wave preceding a VPC, giving the appearance of an association. This scenario may explain why the PR intervals of the premature complexes were shortened, because APCs more commonly have normal to prolonged PR intervals.[2] The presence of fully compensatory pauses would have supported the suggestion that the aberrant complexes were VPCs; however, fully compensatory pauses were not evident in the ECG recording for the cat of the present report. Although the origin of the complexes could not be definitively determined, we speculated that they were supraventricular complexes with a phase 3 conduction aberrancy.

In affected cats, conduction aberrancies do not require intervention, but their presence warrants further investigation, and they should be differentiated from ventricular arrhythmias. Ventricular arrhythmias often require specific treatment and can be associated with a poorer prognosis, particularly in cats with hypertrophic cardiomyopathy and concurrent arrhythmias.[6] There are limited reports regarding the Ashman phenomenon in the veterinary medicine literature, but identifying and understanding the development of conduction aberrancies are important to prevent misinterpretation of them as ventricular ectopy and subsequent mismanagement of patients. Phase 3 conduction aberrancy can be associated with other arrhythmias with long-short cycles and has also been observed with atrial tachycardia and atrial flutter.[7,8]

The exact mechanism by which the RBB has a longer refractory period than that of the left bundle branch is not completely understood, but it is theorized that structural and channel distribution differences between bundle branches have a role.[1,9,10] Left bundle branch block becomes more prevalent with more severe cardiac disease and slower heart rates but is more likely a result of phase 4 conduction aberrancy.[11] When disease affects cardiomyocytes, their automaticity can be enhanced, thereby causing a gradual increase in membrane potential during phase 4 of the action potential and leading to inactivation of the fast sodium channels required for successful impulse conduction. Reduced heart rates allow the membrane potential to increase sufficiently to cause the cardiomyocytes to be refractory to incoming impulses, resulting in a phase 4 conduction aberrancy and a block in conduction.

In the cat of the present report, the presence of wide, aberrant QRS complexes did not warrant specific intervention. The only alteration in the cat's treatment regimen was the addition of pimobendan (0.26 mg/kg [0.12 mg/lb], PO, q 12 h). Five months later, the cat developed bilateral hind limb aortic thromboembolism, pulmonary edema, and pleural effusion and was subsequently euthanized.

## References


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