Title

ECG of the Month.

Journal

Journal of the American Veterinary Medical Association, 249(10)

ISSN

0003-1488

Authors

Bélanger, Catherine
Visser, Lance C
Stern, Joshua A

Publication Date

2016-11-15

DOI

10.2460/javma.249.10.1138

Peer reviewed

ECG of the Month

A 6-year-old 4.2-kg (9.2-lb) castrated male Yorkshire Terrier mix was evaluated because of a suspected anaphylactic reaction secondary to a bee sting on the right pelvic limb. Shortly after being stung, the dog vomited and became tachypneic, which prompted the dog's owner to seek veterinary medical attention. The dog was not receiving any medication at the time of the evaluation, and no previous medical problems were reported. At the evaluation, the dog appeared anxious and aggressive. Physical examination findings were unremarkable except for an irregular heart rhythm and a heart rate of 66 beats/min. A single lead II ECG was acquired **(Figure 1)** to characterize the dog's irregular rhythm and relative bradycardia.

ECG Interpretation

The ECG recording (Figure 1) was most consistent with a distributional pattern called escape-capture bigeminy.[1] We proposed 2 possible explanations for this dog's underlying bradyarrhythmia that established the conditions for escape-capture bigeminy. Both explanations involved dysfunction of the sinus node. One possibility was that the dog had sinoatrial block with a fixed conduction ratio of, for example, 2:1 (illustrated by a ladder [Lewis] diagram **[Figure 2]**). In this scenario, the dog's sinus rate was 66 depolarizations/min, but for every 2 sinus node depolarizations, only 1 was conducted to the atrial myocardium. An alternate explanation was that the dog had profound sinus bradycardia and the sinus rate was 33 depolarizations/min. The junctional escape complexes present throughout the tracing did not alter the sinus node's cycle duration. Sinoatrial node entry block, lack of retrograde atrioventricular (AV) conduction, or a combination of both could explain this finding. On close inspection, the junctional escape complexes did not appear to depolarize the atria in a retrograde manner because there was no overt evidence of P waves preceding or superimposed on the QRS-T complexes. However, retrograde AV conduction to the atria could not be completely excluded because only a single lead (II) tracing was acquired.

The atrial rhythm in this dog was deemed of sinus origin at a fixed rate of 33 depolarizations/min. The ventricular rhythm was irregular (mean rate, approx 70 depolarizations/min) and distinguished by a recurrent alternating pattern of paired QRS complexes of 2 slightly different morphologies. The upright, narrow QRS complexes with consistent P waves and a fixed PQ interval represented impulses originating in the sinus node that followed normal AV conduction. The upright and narrow QRS complexes without P waves and with slightly increased voltages (relative to voltages of the normal QRS complexes) represented junctional escape complexes. These were deemed junctional escape complexes because of their upright and narrow morphology and their occurrence following a pause, albeit brief. The junctional escape interval from the normally conducted QRS complexes was consistently 1,000 milliseconds, equivalent to an instantaneous rate of 60 depolarizations/min (a typical AV node-His bundle depolarization rate of dogs[2]). The timing of the sinus P waves that were conducted normally to the atria and ventricles provided the so-called sinus capture complexes, which consistently fulfilled the escape-capture sequence. Depression of the J-point (ST segment depression) was present throughout the tracing, suggesting injury or ischemia to the left ventricular subendocardium or right ventricular subepicardium.[3]

Following the ECG examination, the dog was administered diphenhydramine hydrochloride (10 mg, IM) for a suspected anaphylactic reaction secondary to the bee sting and discharged to the client. The dog was reevaluated 10 days later, and an ECG examination was repeated **(Figure 3)**. A normal sinus rhythm was present throughout the duration of the ECG recording, and the J-point had normalized. The dog's owner reported complete resolution of the dog's clinical signs within 24 hours after the initial evaluation.

This report was submitted by Catherine Bélanger, DMV; Lance C. Visser, DVM, MS; and Joshua A. Stern, DVM, PhD; from the Department of Medicine & Epidemiology, School of Veterinary Medicine, University of California-Davis, Davis, CA 95616.

Address correspondence to Dr. Visser (lcvisser@ucdavis.edu).

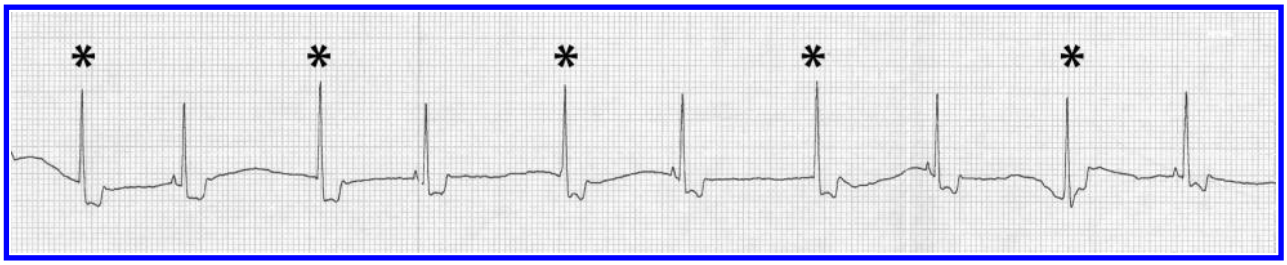

Figure 1—Lead-II ECG tracing obtained from a 6-year-old dog that was examined because of a suspected anaphylactic reaction secondary to a bee sting. The rhythm diagnosis is sinus bradycardia and escape-capture bigeminy. Notice the junctional escape complexes (asterisks) are each followed by a sinus capture complex. Depression of the J-point (ST segment) is also apparent. Paper speed = 25 mm/s; 1 cm = 1 mV.

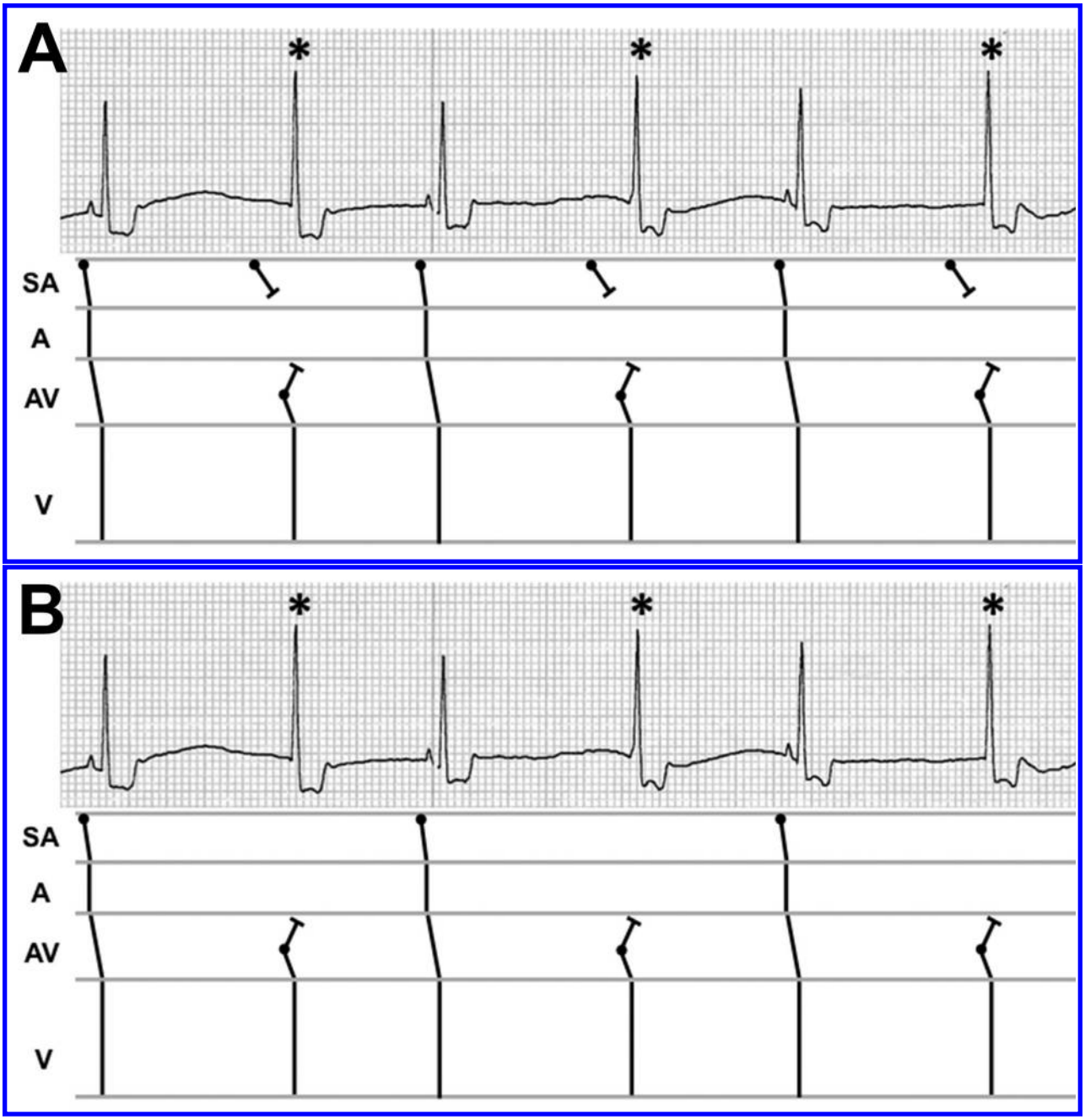

Figure 2—Lead-II ECG tracing from the dog's ECG recording in Figure 1 and ladder diagrams of the 2 proposed mechanisms for the dog's underlying bradyarrhythmia. In each ladder diagram, SA represents sinoatrial node conduction, A represents atrial activation, AV represents atrioventricular node or junction conduction, and V represents ventricular activation. One possible explanation (A) is that there is 2:1 SA block with a sinus rate of 66 depolarizations/min. An alternate explanation (B) is that there is marked sinus bradycardia with a sinus rate of 33 depolarizations/min. For escape-capture bigeminy to persist, the junctional escape complexes (asterisks) cannot reset the sinus node via retrograde atrioventricular conduction. Paper speed = 25 mm/s; 1 cm = 1 mV.

Discussion

The dog of the present report had a bigeminal rhythm termed escape-capture bigeminy. This bigeminal rhythm disturbance is considered exceedingly rare because unique conditions are necessary for this rhythm to persist. Both of our proposed explanations (Figure 2) for this dog's underlying bradyarrhythmia had the conditions required for escape-capture bigeminy to persist. For persistence of escape-capture bigeminy, the effective intersinus interval (ie, the time between conducted sinus impulses [approx 1,800 milliseconds in this dog]) has to exceed the sum of the escape interval (approx 1,000 milliseconds in this dog) and its refractory period, and the escape complex cannot alter the sinus node's cycle duration (eg, because of retrograde AV conduc-

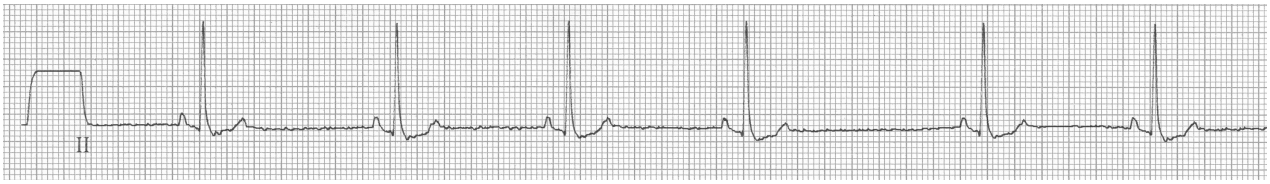

Figure 3—Repeated lead-II ECG tracing obtained from the dog in Figures 1 and 2 ten days after treatment with diphenhydramine hydrochloride because of the suspected anaphylactic reaction secondary to a bee sting. Notice the dog's sinus bradycardia and J-point depression have resolved, and the ECG tracing reveals a normal sinus rhythm with a mean heart rate of 90 beats/min. Paper speed = 50 mm/s; 1 cm = 1 mV.

tion).[1,4] Although these proposed explanations can be considered plausible, neither can be proven without results of an intracardiac electrophysiological assessment.

Numerous cases of escape-capture bigeminy in humans[1,4–6] and less frequently in cats[7–9] have been previously reported. To our knowledge, escape-capture bigeminy in dogs has never been reported. Several forms of escape-capture bigeminy are possible, but the most common forms in humans are associated with sinus node dysfunction or second-degree AV block.[6] Previous cases reported in the veterinary medical literature have all involved cats and were associated with second-degree AV block, most likely because of the relatively fast ventricular escape rhythm that is common in cats with bradyarrhythmias.[7–9] The case described in the present report is an example of escape-capture bigeminy secondary to transient sinus node dysfunction in a dog; the features of the case resemble those of junctional escape-capture bigeminy in a human in which sinus node dysfunction was diagnosed via electrophysiologic testing.[4]

It was our hypothesis that the anaphylactic response secondary to the bee venom contributed to the underlying bradycardia and J-point depression in the dog of the present report. In humans, most patients that have anaphylaxis secondary to an insect sting develop tachycardia.[10] However, in a subset of patients, bradycardia can develop following insect stings; this response is thought to be secondary to a Bezold-Jarisch (vasovagal) reflex triggered by a coronary vasospasm that results in heightened vagal tone.[10–12] In humans, J-point deviation secondary to coronary vasospasm can occur secondary to bee stings (and other allergenic insults) owing to release of vasoactive mediators from mast cells.[12] This is referred to as allergic angina or Kounis syndrome. This mechanism, coupled with the vomiting episode that can also be associated with increased vagal tone, could have contributed to the transient sinus node dysfunction in the dog of this report. This hypothesis was further supported by resolution of the bradyarrhythmia and normalization of the J-point following empirical treatment as detected at the 10-day recheck examination (Figure 3). An atropine response test could also have been performed to help implicate elevated vagal tone as a contributing factor of the bradyarrhythmia.

Acknowledgments

The authors thank Dr. Kate Farrell for assistance with case management.

References

1. Schamroth L, Dubb A. Escape-capture bigeminy. Mechanisms in S-A block, A-V block, and reversed reciprocal rhythm. *Br Heart J* 1965;27:667–669.
2. Tilley LP. Generation of the electrocardiogram: basic principles. In: Tilley LP, ed. *Essentials of canine and feline electrocardiography: interpretation and treatment.* 3rd ed. Philadelphia: Lea & Febiger, 1992;1–20.
3. Tilley LP. Analysis of canine P-QRS-T deflections. In: Tilley LP, ed. *Essentials of canine and feline electrocardiography: interpretation and treatment.* 3rd ed. Philadelphia: Lea & Febiger, 1992;59–99.
4. Henry SA, Makaryus AN, Loewinger L, et al. The great escape: junctional escape-capture bigeminy. *Am J Geriatr Cardiol* 2006;15:377–378.
5. Bradley SM, Marriott HJ. Escape-capture bigeminy; report of a case of A-V dissociation initiated by 2:1 S-A block with resulting bigeminal rhythm. *Am J Cardiol* 1958;1:640–643.
6. Schamroth L. Escape-capture bigeminy. In: Schamroth L, ed. *The disorders of cardiac rhythm.* 2nd ed. Oxford, England: Blackwell, 1980;329–331.
7. Fife WD, Cote E. ECG of the month. Second-degree atrioventricular (AV) block, type II advanced (3:1), with ventricular escape complexes. *J Am Vet Med Assoc* 2002;220:172–173.
8. Visser LC, Scansen BA, Bonagura JD. ECG of the month. Escape-capture bigeminy in a cat. *J Am Vet Med Assoc* 2014;245:52–54.
9. Tilley LP. Uncommon complex arrhythmias. In: Tilley LP, ed. *Essentials of canine and feline electrocardiography: interpretation and treatment.* 3rd ed. Philadelphia: Lea & Febiger, 1992;385–416.
10. Joint Task Force on Practice Practice Parameters; American Academy of Allergy, Asthma, and Immunology; American College of Allergy, Asthma, and Immunology, et al. The diagnosis and management of anaphylaxis: an updated practice parameter (Erratum published in *J Allergy Clin Immunol* 2008;122:68). *J Allergy Clin Immunol* 2005;115:S483–S523.
11. Brown SG, Blackman KE, Stenlake V, et al. Insect sting anaphylaxis; prospective evaluation of treatment with intravenous adrenaline and volume resuscitation. *Emerg Med J* 2004;21:149–154.
12. Scherbak D, Lazkani M, Sparacino N, et al. Kounis syndrome: a stinging case of ST-elevation myocardial infarction. *Heart Lung Circ* 2015;24:e48–e50.