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Background: Syncope is a recognized problem in Boxers and often is the result of rapid ventricular tachycardia (VT).

Affected dogs may have echocardiographic evidence of dilated cardiomyopathy, but frequently have normal echocardiograms.

Although VT is probably the most common cause of syncope in Boxers, neurocardiogenic bradycardia can also occur.

Objective: We describe 7 Boxers with comorbid VT and neurocardiogenic bradycardia, wherein the syncope was secondary to bradycardia rather than VT.

Animals: Seven Boxers were selected from a larger population of Boxers with Holter-documented VT because these dogs had documented bradycardia at the time of syncope.

Methods: Retrospective study.

Results: Although all dogs had Holter-documented VT, the etiology of the syncopal episodes was consistent with neurocardiogenic bradycardia.

Clinical Importance: Neurocardiogenic bradycardia or VT can occur as isolated problems in Boxers. In some Boxers, VT and potential or manifest neurocardiogenic bradycardia coexist. The administration of a β-blocker or sotalol to such dogs can aggravate or precipitate neurocardiogenic bradycardia-related syncope.

Key words: Cardiomyopathy; Event recording; Holter recording; Neurocardiogenic bradycardia.

Syncope is a common problem in Boxers. Two common causes of syncope in this breed are ventricular tachycardia (VT) associated with cardiomyopathy and neurocardiogenic bradycardia. The former is more common and is usually associated with many ventricular premature complexes (VPC) when a Holter recording is performed within a day or two of the syncopal event. It is potentially lethal and is usually treated with mexiletine or sotalol. Neurocardiogenic bradycardia is less common and is not necessarily associated with cardiomyopathy. Syncope typically occurs infrequently and may not require treatment. When a Holter recording performed soon after a syncopal event in a Boxer contains no or few VPC, syncope secondary to bradycardia should be strongly considered. Neurocardiogenic bradycardia can coexist with cardiomyopathy in Boxers because both conditions are common. β-blocker or sotalol treatment, unlike mexiletine, administered for VPC may uncover or aggravate neurocardiogenic bradycardia.

Materials and Methods

Seven Boxers were retrospectively selected from a larger population of Boxers evaluated between 2002 and 2005 with Holter-documented VT, because these Boxers had documented bradycardia at the time of syncope. There were 4 males and 3 females ranging in age from 3 to 7 years.

Case Examples

Case 1

A 6-year-old male, 39-kg Boxer was presented with the chief complaint of syncope. The dog had experienced several syncopal episodes between 1 and 5 years of age. Each episode was associated with exertion coupled with excitement. Recently, the dog had experienced 3 episodes over a span of approximately 2 weeks. One episode was associated with exertion and 2 episodes occurred when the dog arose after awakening.

On physical examination, an arrhythmia was auscultated, which was thought to be consistent with the occurrence of VT. Cardiac ultrasound examination identified a left ventricular end-diastolic dimension of 52 mm (normal, ≤48 mm), left ventricular end-systolic dimension of 40 mm (normal, ≤38 mm), left ventricular fractional shortening of 23% (normal, ≥30%), and an E-point to septal separation of 10 mm (normal, ≤8 mm) consistent with mild dilated cardiomyopathy (DCM).

The heart rhythm was further evaluated. A static ECG was performed and the only abnormalities identified were isolated VPC. The morphology of these VPC was consistent with a right ventricular origin. An ambulatory ECG (Holter) recorder was fitted to the dog. During the ensuing 24 hours, the dog was normally active in the home environment. On 1 occasion, the dog collapsed immediately on arising from sleep and standing up.

Clinically relevant Holter analysis results included 9,583 VPC, VT, and an episode of bradycardia. The VPC comprised approximately 183 couplets, 68 triplets, and 27 episodes of nonsustained (6–26 beats) VT at rates of 250–300 beats/min (bpm) (Fig 1A). According to the patient diary, the episodes of VT were not associated with overt clinical signs.
The syncopal episode was associated with sudden sinoatrial arrest, which followed an increase in the sinus rate associated with awakening and rising (Fig 1B). The sinoatrial arrest was punctuated by occasional sinus beats, ventricular escape beats, and sinus bradycardia with a gradual return to normal sinus rhythm. The period of bradycardia lasted approximately 45 seconds.

Mexiletine\textsuperscript{4} (5.1 mg/kg PO q8h) was prescribed for the VT. The client was advised to curtail the patient’s exertion and excitement as much as practical and that the bradycardia episodes would not likely lead to sudden death. A follow-up Holter recording was performed after 5 days of mexiletine treatment. The salient features of the results were 789 VPC comprising 785 singles and

![Fig 1. (A) Holter recording with paroxysms of ventricular tachycardia interspersed with sinus rhythm that is punctuated by ventricular premature complexes. (B) Later in the recording, an abrupt onset of bradycardia occurred that was triggered by a sudden change of activity level. There is a pronounced sinoatrial arrest, followed by escape beats. Normal sinus rhythm was restored after approximately 45 seconds.](image)
2 couples. Subsequently, occasional startle, exertion, or excitement-triggered episodes of presyncope continued, serial Holter recordings contained progressively more severe VPC, and serial echocardiographic analysis identified progressive DCM until congestive heart failure developed after 13 months.

**Case 2**

A middle-aged, male Boxer with a history of syncope was evaluated. Syncope had occurred at 3- to 6-month intervals for a period of approximately 2 years. Each syncope event occurred after the onset of sudden, vigorous activity, although syncope did not occur during most periods of activity. Echocardiographic results were consistent with mild DCM. A Holter recording contained 1,850 VPC with right ventricular origin morphology, 73 couplets, 26 triplets, and 4 paroxysms (5–14 beats) of rapid (250 bpm) VT. Mexiletine (5 mg/kg PO q8h) was prescribed. A follow-up Holter recording 5 days later contained 1,021 VPC with 7 couplets, 2 triplets, and no VT. During the ensuing month, 2 additional episodes of syncope occurred. An event recorder was fitted to the patient for 7 days, but no episodes of weakness or syncope occurred. The client was then issued a chest-press ECG recorder to carry with him at all times when the dog was present. After approximately 2 months, exertion-associated syncope was documented to be the result of sinoatrial arrest. The ECG printout contained sinoatrial arrest with irregularly spaced junctional and ventricular escape complexes. The sinus rhythm gradually returned to normal approximately 1 minute after the onset of syncope. The dog’s echocardiographic abnormalities progressed after the initial echocardiogram, and serial Holter recordings contained more severe VPC, including frequent nonsustained VT. Echocardiographic data indicated imminent congestive heart failure after approximately 7 months. A syncope event occurred, and the dog was immediately but briefly attacked by 2 family Boxers. The patient recovered quickly but died suddenly approximately 15 minutes later.

**Case 3**

A 7-year-old male Boxer owned by a veterinarian had been examined by echocardiography and Holter recording 8 times over a period of 3 years. Echocardiographic results were normal, whereas Holter recordings contained 650–1,475 VPC, without VT. Given the absence of syncope and VT, the dog was not treated for the arrhythmia. Subsequently, the owner auscultated a more severe arrhythmia in the absence of syncope or presyncope. Several days later, an echocardiogram was normal but a Holter recording contained 16,189 VPC with numerous couplets, triplets, and 9 episodes of paroxysmal (4–11 beats) VT at rates of 200–250 bpm. Sotalol (1.25 mg/kg PO q12h) was prescribed. After 2 doses of sotalol, a severe syncopal event occurred as the patient was climbing stairs, and the veterinarian auscultated severe bradycardia. There were long periods of apparent asystole punctuated by irregular beats that gradually increased in frequency until the dog recovered after 2 minutes. Sotalol was replaced by mexiletine, and no additional syncopeal episodes have occurred after 26 months. Serial echocardiogram and Holter recording analyses indicated an absence of DCM, VPC without VT, and abnormal bradycardia has not been documented.

**Case 4**

A middle-aged, male Boxer was evaluated because of several episodes of exertion-associated syncope. The owner believed that she had detected a very slow heart rate (apex beat) during 1 episode of syncope. Echocardiography results were normal. A Holter recording contained 13,465 VPC (right ventricular origin morphology) with couplets, triplets, and 6 paroxysms (4–6 beats) of rapid (250–300 bpm) VT. Sotalol (1 mg/kg PO q12h) was prescribed. The number of syncopeal episodes increased during the ensuing week, and 1 episode occurred during a Holter recording. Syncope was associated with exertion and was the result of sudden sinoatrial arrest (Fig 2). Escape beats began after approximately 7 seconds, and normal sinus rhythm was restored after approximately 30 seconds. Sotalol treatment was replaced by mexiletine. Serial echocardiography results remained normal. Four serial Holter recordings performed at approximately 3–4-month intervals have contained neither VT nor abnormal bradycardia. Exertion- or excitement-induced syncope continues to occur at 3–6-month intervals.

Three additional Boxers with normal echocardiograms and Holter-documented VPC, which included paroxysmal VT, experienced one or more flight- or fright-related syncopal events. In each dog, syncope was documented to be the result of marked bradycardia (Fig 3). None of the episodes of bradycardia occurred immediately after VT.

**Discussion**

Syncope is a sudden and brief loss of consciousness from which recovery is spontaneous. The loss of consciousness is the result of brief cessation of cerebral blood flow. Syncope is a recognized problem in Boxers and often is the result of rapid VT. Affected dogs may have echocardiographic evidence of DCM, but frequently have normal echocardiograms. Although VT probably is the most common cause of syncope in Boxers, exertion- or excitement-associated syncope with no or relatively few VPC detected by Holter recording can also occur.

The etiology of the syncopal episodes in the Boxers described here is consistent with neurally mediated bradycardia. Neurally mediated bradycardia is referred to by numerous synonyms, including neurocardiogenic syncope, cardioneurogenic syncope, vasodepressor syncope, and vasovagal syncope, and is distinct from that of advanced heart blocks and sick sinus syndrome. The Holter recordings in the dogs described here did not contain the sustained or frequent abnormalities typical of sick sinus syndrome.
The pathophysiology of neurally mediated bradycardia is incompletely understood. Episodes can be triggered by activities or situations that result in either a sympathetic surge or a parasympathetic surge. The latter triggers result in situational syncopes. Surges of sympathetic activity can trigger intracardiac receptors that respond to loading or contractile conditions by evoking reflex vagal afferent traffic to the brainstem. The final common pathway associated with either sympathetic- or parasympathetic-initiated triggers is afferent vagal stimulation of the medullary vasomotor (vasodepressor) center. This center then responds with sympathetic withdrawal and mild or severely accentuated vagal efferent traffic.

Either neurocardiogenic bradycardia or cardiomyopathy-associated VT occurs as an isolated problem in Boxers. However, in some Boxers, as in the dogs in this report, VT and potential or manifest neurocardiogenic bradycardia coexist. The administration of β-blockers such as atenolol can aggravate or precipitate bradycardia-related syncope in cardiomyopathic Doberman Pinschers. This may also be a problem in some Boxers treated with sotalol and presumably β-blockers. Boxers with VPC that are treated with sotalol and then experience new or exacerbated syncopal episodes may be experiencing neurocardiogenic bradycardia.

Our experience is that when syncope in Boxers is the result of VT, 24-hour Holter recordings performed within 1–2 days of syncope contain many VPC and usually paroxysmal VT. However, in some of these patients, frequent VPC and VT may not occur for as long as 10–14 hours into the recording period. Therefore, isolated VPC and severe arrhythmia may not be detected by static ECG. Holter recordings performed in Boxers within a few days of syncope that contain no or few VPC may indicate that the syncope is caused by bradycardia.

Treatment of neurocardiogenic bradycardia and syncope in Boxers is problematic. Usually, the episodes are infrequent and treatment is often not required. Avoiding the instigating exertion or excitement that triggers the episodes sometimes is practical. However, for patients with frequent syncopal episodes, pacemaker implantation and anticholinergic drug therapy are therapeutic options. Anticholinergic drug treatment or pacemaker implantation can be prescribed for selected patients that experience frequent or predictable episodes. However, anticholinergic drugs are ill advised in patients with VPC. In humans, neurocardiogenic
Bradycardia and syncope are often preceded by increased sympathetic activity. There are 2 subsets of these patients. One subset experiences vagal-induced bradycardia and sympathetic withdrawal-induced vasodilatation and hypotension. The other subset experiences sympathetic withdrawal-induced vasodilatation and hypotension, whereas vagal outflow and bradycardia are absent or minimal. For either subset, no medical therapies have been proven to prevent syncope in large randomized clinical trials. We do not believe that neurocardiogenic bradycardia in Boxers in the home or natural setting is lethal.

Footnotes


References