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Paroxysmal atrial fibrillation in seven dogs with presumed neurally-mediated syncope

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Abstract *Objectives:* To document the electrocardiographic findings of vagally-induced paroxysmal atrial fibrillation following a presumed reflex syncopal episode in the dog.
Animals: Seven dogs with a syncopal episode followed by a paroxysm of atrial fibrillation recorded on a 24-hour Holter.
Methods: Twenty-four hour Holter monitors were retrospectively reviewed, analysing the cardiac rhythm associated with syncopal events. Each recording was analysed from 10 min before the syncopal episode to until 10 min after a normal sinus rhythm had returned.
Results: Nine episodes were recorded in seven dogs, with one patient experiencing three events during one Holter recording. Five of the seven dogs presented with underlying structural heart disease. In two the syncopal episodes occurred following exercise, two associated with coughing and three were during a period of rest. All dogs had documented on the Holter recording a rhythm abnormality during syncope. The most common finding leading up to the syncopal event was development of a progressive sinus bradycardia, followed by sinus arrest interrupted by a ventricular escape rhythm and then ventricular arrest. This was then followed by an atrial fibrillation. The atrial fibrillation was paroxysmal in seven recordings and persistent in two. In two dogs, the atrial fibrillation reorganised into self-limiting runs of atypical atrial flutter.

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Conclusions: This combination of electrocardiographic arrhythmias are probably caused by an inappropriate parasympathetic stimulation initiating a reflex or neurally-mediated syncope, with abnormal automaticity of the sinus node and of the subsidiary pacemaker cells and changes in the electrophysiological properties of the atrial muscle, which promoted the paroxysmal atrial fibrillation.

Introduction

Syncope is defined as a transient loss of consciousness due to global cerebral hypoperfusion and it is characterized by rapid onset, short duration, with a spontaneous and complete recovery.[1] In humans syncope is divided into reflex (vasovagal, situational or carotid sinus syndrome), cardiac (arrhythmias and structural heart disease) and orthostatic (autonomic failure, volume depletion or hypotensive drugs).[1] Reflex syncope is an inappropriate and intermittent neural response to a trigger and is classified based on the efferent autonomic nervous pathway involved. The response is the combination of a cardioinhibitory mechanism (predominance of parasympathetic tone) and/or vasodepressor mechanism (loss of sympathetic tone) that results in a fall in systemic blood pressure and cerebral hypoperfusion.[1] The vagal cardiac activity during the cardioinhibitory mechanism determines the genesis of different arrhythmogenic patterns.[2] The vagal activity inhibits impulse generation in the sinoatrial node generating bradycardia or sinus arrest,[3] slows conduction through the atrioventricular node, generating atrioventricular block[4] and alters the electrophysiological properties of atrial and pulmonary veins cells.[5] Vagal stimulation shortens the effective refractory period and action potential in the atrial tissue, favouring the formation of re-entry circuits, dispersion of repolarisation and the development of atrial fibrillation.[6] In a similar way, acetylcholine shortens the action potential duration of the sleeves of the pulmonary veins enhancing calcium transient and Na—Ca exchange current, and when acting in conjunction with high sympathetic tone this facilitates the development of early afterdepolarisation in the pulmonary veins that enable ectopic foci.[7] These electrophysiological alterations create the substrate for the genesis of the vagal atrial paroxysmal atrial fibrillation. Furthermore, the autonomic nervous system can modulate the initiation, maintenance and termination of a functional atrial flutter (Well's type II), characterised by very rapid rhythm originating under vagal stimulation with no excitable gap in the circuit and cannot be entrained.[8,9] Atrial fibrillation and atrial flutter are re-entrant arrhythmias and there is an interrelationship between them.[10] The conversion from atrial flutter to atrial fibrillation is due to an acceleration of atrial rate due to the disappearance of areas of slow conduction, decrease in the length of functional block and occurrence of unstable re-entrant circuits of very short cycle lengths with various locations and shape, which disappear and reformed.[11] The peculiar combination of neurally-mediated syncope and development of atrial fibrillation has been previously described in human.[12]

The aim of this study was to determine the arrhythmia and mechanism that led to the development of atrial fibrillation, associated with an episode of syncope and to ascertain if such a rhythm was consistent with a neurally-mediated mechanism for the loss of consciousness.

Materials and methods

Patients

Case records were retrospectively searched for syncope that had been documented on Holter and associated with a bradyarrhythmia followed by atrial fibrillation. Seven cases were found; these included: three Boxers, one Beagle, one Pug and two mixed breed dogs. Five dogs were male and two female. Age ranged from 5 to 12 years (median: 9.3 years). Body weight ranged from 10 to 40 kg (median: 20.8 kg). Five dogs had underlying structural heart disease including degenerative mitral valve disease (n = 3), dilated cardiomyopathy (n = 1) and aortic stenosis with 'afterload mismatch' (n = 1). Four dogs were in well-controlled heart failure, being medicated with furosemide, pimobendan and an angiotensin converting enzyme inhibitor. The Holter recordings were collected from two referral centres, Clinica Veterinaria Malpensa (Cube Holter Cardioline;