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Permanent junctional reciprocating tachycardia in a dog

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Abstract A 5-year-old male English Bulldog was presented with a 1-year history of paroxysmal supraventricular tachycardia (SVT) partially responsive to amiodarone. At admission the surface ECG showed sustained runs of a narrow QRS complex tachycardia, with a ventricular cycle length (R—R interval) of 260 ms, alternating with periods of sinus rhythm. Endocardial mapping identified the electrogenic mechanism of the SVT as a circus movement tachycardia with retrograde and decremental conduction along a concealed postero-septal atrioventricular pathway (AP) and anterograde conduction along the atrioventricular node. These characteristics were indicative of a permanent junctional reciprocating tachycardia (PJRT). Radiofrequency catheter ablation of the AP successfully terminated the PJRT, with no recurrence of tachycardia on Holter monitoring at 12 months follow-up.

A 5-year-old male English Bulldog was presented to our institution with a history of episodic weakness caused by a supraventricular tachycardia (SVT) treated with amiodarone (7.5 mg/kg once a day orally) for 1 year. Despite therapy, the dog experienced several episodes of the same tachyarrhythmia lasting up to 13 h that were documented by Holter monitor recordings. The dose of amiodarone could not be increased due to occurrence of severe gastrointestinal side effects. Upon admission the dog was bright and alert with normal body condition score and no signs of congestive heart failure. Thoracic radiographs and echocardiography were within normal limits. A 12-lead ECG showed runs of a narrow QRS complex (60 ms) tachycardia with a ventricular cycle length (R—R interval) of 260 ms, negative P′ waves in the inferior leads (II, III and aVF) with a mean

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Abbreviations

AP	accessory pathway
AV	atrio-ventricular
AVRNT	atrioventricular reentrant nodal tachycardia
CS	coronary sinus
FAT	focal atrial tachycardia
HV	his-ventricular
PJRT	permanent junctional reciprocating tachycardia
RV	right ventricle
SVT	supraventricular tachycardia
VA	ventriculo-atrial

electrical axis in the frontal plane of −45°, a long RP' (140 ms) and an RP'/P'R of 1.16 (Fig. 1A). The arrhythmia alternated with periods of normal sinus rhythm with PQ duration in the lower limit of the reference range (75 ms) and a normal QRS configuration (Fig. 1B). According to the electrocardiographic characteristics of the SVT, four possible diagnoses were considered: focal atrial tachycardia (FAT) originating from the coronary sinus (CS), atypical (fast-slow or slow—slow variant) atrio-ventricular reentrant nodal tachycardia (AVRNT), permanent junctional reciprocating tachycardia (PJRT) and orthodromic atrioventricular reciprocating tachycardia (OAVRT) with prolonged ventriculo-atrial (VA) conduction time secondary to the effects of amiodarone on the accessory pathway (AP) conduction and refractoriness. Given the potential for ablation of the arrhythmic substrate in all these different types of SVT, an electrophysiologic study (EPS) was scheduled 4 days after discontinuation of amiodarone.

The EPS was performed under general anesthesia, and the dog prepared as previously described.[1] Using a modified Seldinger technique, three venous accesses were obtained: one in the left external jugular vein and two in the right femoral vein. From the jugular vein a decapolar electrode catheter[c] was positioned in the coronary sinus; a quadripolar electrode catheter[d] was inserted through the right femoral vein and positioned at the atrioventricular (AV) node region to record His bundle potentials; finally, a 7-Fr catheter[e] was placed alternatively in the right atrium, right ventricle and tricuspid valve annulus for mapping and programmed stimulation. Surface and intracardiac ECG signals were displayed on a recorder[f] at a paper speed of 150 or 300 mm/s. Intracardiac ECGs were recorded at filter settings of 50—500 Hz. Pacing was performed with stimuli that were twice the diastolic threshold and 2 ms in duration.

During the basal study, a sinus rhythm with normal antegrade conduction times characterized by an AH interval of 64 ms (normal values 54—116 ms)[2] and a HV interval of 12 ms (normal values 30—44 ms)[2] was recorded. The HV interval was shorter than reported because the His catheter was placed just beyond the actual hissian area. During programmed ventricular pacing an eccentric VA activation along a concealed right postero-septal AP with decremental conduction properties was recorded. Ventriculo-atrial conduction times ranged from 140 to 185 ms (Fig. 2). Ventricular refractoriness was attained before the AP retrograde effective refractory period was reached, which was estimated to be less than 150 ms. Programmed atrial stimulation induced an AV reciprocating tachycardia with the earliest site of retrograde atrial activation at the proximal coronary sinus. To distinguish AVRT from AVNRT and FAT, during tachycardia, a timed ventricular extrastimulus was introduced during the His refractory period.[1] The QRS complex of the ventricular paced beat was anticipated with respect to the tachycardia cycle length, and interrupted the tachycardia without atrial retro-activation (Fig. 3).

According to endocardial mapping and response to electrophysiologic tests, a diagnosis of PJRT with antegrade conduction along the AV node and retrograde conduction along a concealed postero-septal AP was made. Mapping of the annular postero-septal region of the tricuspid valve was performed during tachycardia, and once the earliest site of atrial activation with simultaneous sharp and negative unipolar recording was established (Fig. 4), radiofrequency energy was delivered with a temperature control system.[g] Maximal catheter tip temperature and power were set respectively at 65 °C and 75 W. Radiofrequency energy was delivered for 60 s and successfully interrupted the tachycardia after 1.5 s, with median values of 18 W, 64 °C and 111 Ohms for power, temperature and impedance, respectively. The presence of VA dissociation during incremental ventricular pacing, 45 min post-ablation, verified the interruption of conduction along the postero-septal accessory pathway. After 12 months of serial follow-up Holter monitor recordings no recurrence of tachycardia could be documented.

[c] Polaris X, 7F, Boston Scientfic Corp., Genova, Italy.
[d] Explorer ST, 5F, Boston Scientific Corp., Genova, Italy.
[e] Std CrV Blazer II HTD, 4 mm, 7F; Boston Scientific Corp., Genova, Italy.
[f] EMS, 16 channels, MennenMedical, Manta, Genova, Italy.
[g] EPT 1000 XP, Boston Scientific Corp., Genova, Italy.

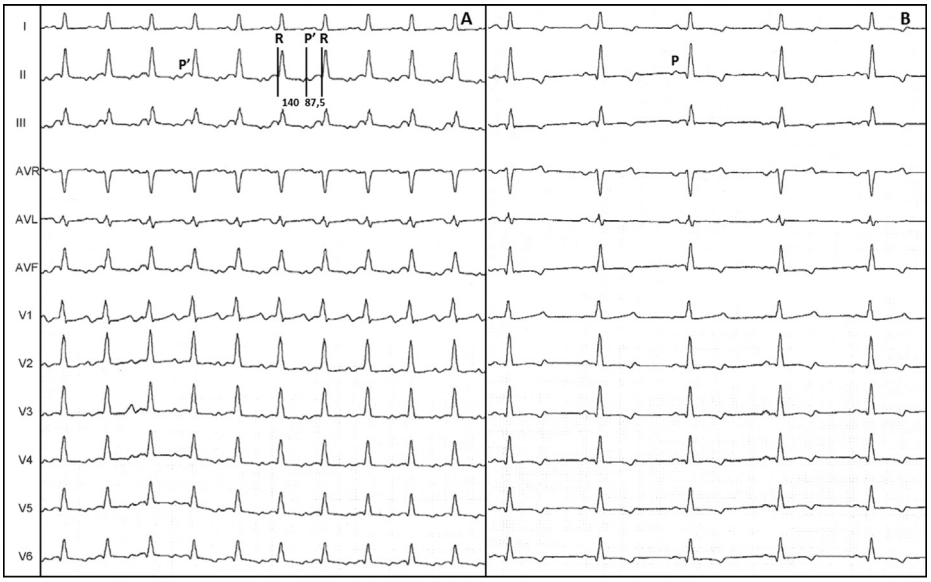

Figure 1 Twelve-lead surface ECG during a sustained run of permanent junctional reciprocating tachycardia (A), and during sinus rhythm (B). Rhythm strip A shows a narrow QRS complex tachycardia with a ventricular cycle length of 260 ms, a negative P′ wave in inferior leads (II, III and aVF) and a long RP' (140 ms). Rhythm strip B shows sinus rhythm with normal QRS complex and a PQ interval within the lower limit of the reference range (75 ms). (Paper speed 50 mm/s; 1 mV = 1 cm).

Discussion

The anatomic distribution and electrophysiologic properties of atrioventricular AP and correlated arrhythmias were previously described in dogs.[1,3–8] In this species, AP were usually right-sided, single, and presented with an all-or-none pattern of conduction, and in 75% of cases were concealed.[1] It has also been reported that atrioventricular AP frequently cause AVRT and atrial fibrillation; atrial fibrillation in one case was pre-excited.[1,8] In this case report, we describe an accessory pathway with

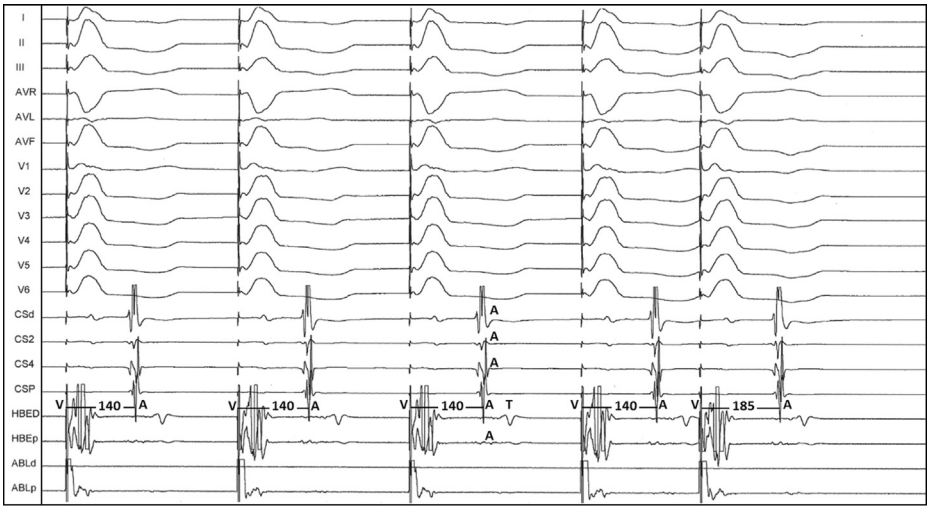

Figure 2 Surface 12-lead ECG and endocardial electrograms from the decapolar catheter in the coronary sinus (from CSd to CS4), the quadripolar catheter placed at the His bundle (HBED, HBEP) and the quadripolar catheter placed at the right ventricular apex (ABLd, ABLp) during programmed ventricular pacing (a train of 8 beats with a cycle length of 420 ms and an extrastimulus with a coupling interval of 285 ms). In the first part of the tracing notice after ventricular activation (V) the presence of eccentric atrial retro-activation (A) with the earliest site of atrial depolarization at the coronary sinus ostium (CS4) and a ventriculo-atrial conduction time of 140 ms, that lengthened up to 185 ms after the extrastimulus, to testify the presence of a postero-septal accessory pathway with decremental properties. CSd, coronary sinus distal; CSP, coronary sinus proximal; HBED, His bundle distal; HBEp, His bundle proximal; ABLd, right ventricular distal; ABLp, right ventricular proximal. (Paper speed: 150 mm/s; 22.8 mV = 1 cm).

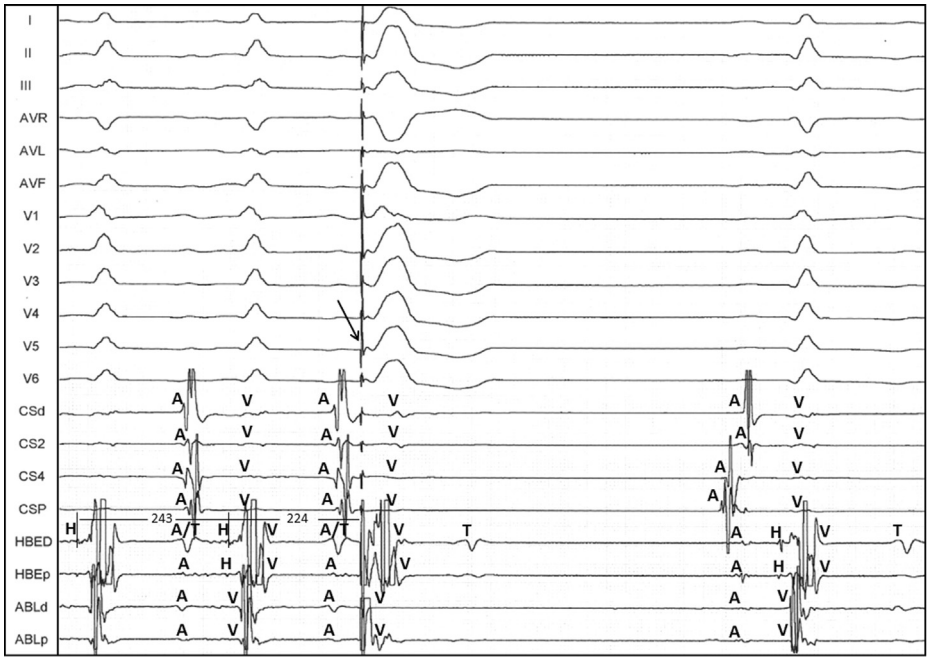

Figure 3 Surface 12-lead ECG and endocardial electrograms from the decapolar catheter of the coronary sinus (from CSd to CS4), the quadripolar catheter placed at the His bundle and the ablation catheter placed at the coronary sinus during tachycardia reset. The first beat represents the ventricular (V) and the atrial activation (A) during tachycardia. Notice eccentric ventriculo-atrial activation with the earliest site of atrial depolarization at the coronary sinus ostium (CS4). A ventricular paced beat was released during tachycardia when the His bundle was refractory. The ventricular extrastimulus (arrow) anticipated the tachycardia and terminated it without atrial retro-activation. This electrophysiologic test proved the presence of an alternative way to the atria represented by an atrioventricular accessory pathway. CSd, coronary sinus distal; CSP, coronary sinus proximal; HBED, His bundle distal; HBEp, His bundle proximal; ABLd, ablation distal, ABLp, ablation proximal; T, T wave. (Paper speed: 150 mm/s; 22.8 mV = 1 cm).

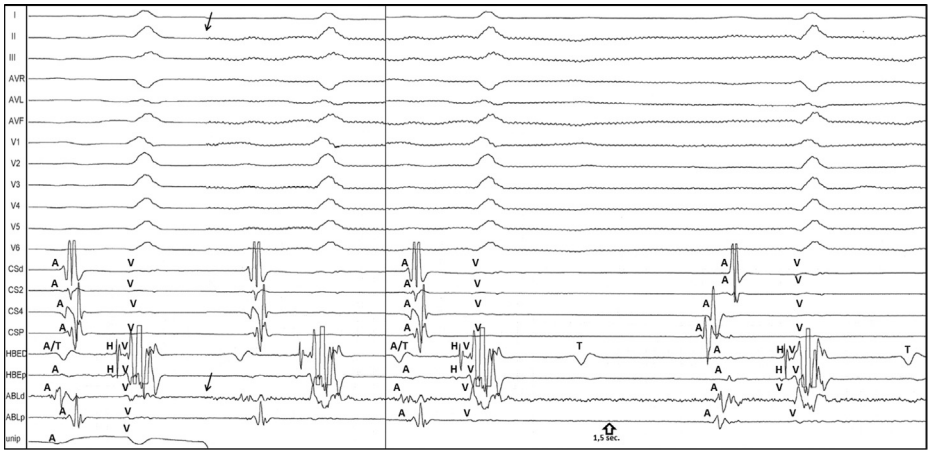

Figure 4 Surface 12-lead ECG and endocardial electrograms from the decapolar catheter of the coronary sinus (from CSd to CS4), the quadripolar catheter placed at the His bundle, the ablation catheter placed at the coronary body and the unipolar recording at the mapping site during tachycardia. Notice eccentric ventriculo-atrial (VA) activation with the earliest site of atrial depolarization at the coronary sinus ostium (ABLd) with a long ventriculo-atrial conduction due to the presence of an atrioventricular postero-septal accessory pathway. The epicenter of atrial activation was considered optimal at ABLd because of the concomitant presence of sharp and negative unipolar recording (black arrow). Only 1.5 s after starting radiofrequency current delivery (open arrow), tachycardia stopped along the accessory pathway (V not followed by A) and sinus rhythm resumed. CSd, coronary sinus distal; CSP, coronary sinus proximal; HBED, His bundle distal; HBEp, His bundle proximal; ABLd, ablation catheter distal; ABLp, ablation catheter proximal; unip; unipolar. (Paper speed: 300 mm/s; 22.8 mV = 1 cm).

unique features proven by detailed endocardial mapping and not yet described in the dog: a posteroseptal bypass tract with unidirectional and decremental retrograde conduction mediating a PJRT. Permanent junctional reciprocating tachycardia or Coumel tachycardia is a form of circus movement tachycardia in which the anterograde limb of the circuit is the AV node and the retrograde limb is a concealed slowly conducting fiber with decremental properties usually located in the posteroseptal region.[9–12] Previous reports suggested that PJRT is an atypical form of AV nodal reentry,[13] leading to the misnomer PJRT. However, as soon as electrophysiologic, surgical, anatomic and ablation reports in patients with PJRT were accurately evaluated, the presence of a concealed posteroseptal accessory pathway with decremental conduction properties was confirmed.[14–16]

Permanent junctional reciprocating tachycardia is often an incessant tachycardia with long RP' interval, narrow QRS complex and most frequently inverted P waves in the inferior leads of the surface ECG.[17] In the dog reported here, an ECG at admission showed a narrow QRS complex tachycardia with a heart rate of 230 bpm, negative P′ waves in the inferior leads (II, III and aVF) with an axis on the frontal plane of −45°, indicating right-sided infero-to-superior atrial activation, a long RP' (140 ms) and RP'/P'R of 1.16. Despite these electrocardiographic findings being suggestive of a FAT arising from the low right atrial region,[18] other long RP' SVTs not yet described in dogs, such as atypical (fast–slow or slow–slow) AVNRT, PJRT, or OAVRT with prolonged VA conduction time induced by amiodarone could not be ruled out. Since all these tachycardias can present with the earliest site of atrial activation at the CS ostium area, only detailed electrophysiologic maneuvers can determine the exact underlying electrogenic mechanism.[19]

During the EP study, the ECG revealed a long RP' narrow QRS complex tachycardia with inverted P′ waves in the inferior leads, which matched previous recordings. Although adenosine can be used to help differentiate AV nodal-dependent narrow QRS complex SVTs from non-AV nodal-dependent SVTs among human patients with short RP' tachycardias, it is less useful in predicting the mechanism of a long RP' tachycardia, because atypical forms of AVNRT, adenosine-sensitive atrial tachycardia, and PJRT can all be terminated by adenosine[20]; therefore a pharmacologic test was not considered in this case. Furthermore, it has been reported that adenosine at doses up to 2 mg/kg did not slow AV nodal conduction in dogs.

First beat, fixed VA interval post-overdrive atrial pacing[1] ruled out FAT and showed that the tachycardia was AV nodal dependent. Chronic use of amiodarone in the dog herein reported could have had some electrophysiologic effects on the AV reentrant circuit such as a prolongation of the effective refractory periods of the atria, ventricles, His-Purkinje system and AP.[21] Amiodarone could also have lessened the rate-dependent shortening of retrograde effective refractory period of the AP during pacing, complicating our ability to make an accurate diagnosis.[22] The evidence of prolongation of VA conduction time during programmed ventricular stimulation allowed to rule out an OAVRT with prolonged VA conduction induced by amiodarone. Finally, differentiation between atypical AVNRT and PJRT was obtained with a tachycardia reset induced by a ventricular paced beat released during the refractory period of the His bundle, that anticipated the tachycardia and terminated it without atrial retro-activation. Other criteria that can be used to distinguish PJRT with this test are pre-excitation of the atria with the same sequence of activation and prolongation (>50 ms) of the local VA interval.[11] Unidirectional retrograde decremental properties of the AP were proven with programmed ventricular pacing that showed a variable VA conduction time ranging from 140 to 185 ms, that was no longer documented post-ablation, but instead resulted in VA dissociation.

In conclusion, this case report describes the electrocardiographic and electrophysiologic features of PJRT in a dog, an uncommon arrhythmia in humans, that can mimic, according to previous electrocardiographic criteria reported in dogs,[18] a FAT arising from the ostium of the coronary sinus. As the number of publications on canine clinical electrophysiology has increased in recent years, we believe the arrhythmia substrate described in this reports offer a valuable contribution to the diagnosis and management of challenging arrhythmias in dogs.

Conflicts of interest

None.

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