The Role of Sympathetic Activity in the Pause-Dependant Onset of Atrial Arrhythmia

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Background: Recent studies showed that late phase 3 early afterdepolarization (EAD) is a novel mechanism of atrial arrhythmogenesis. To induce late phase 3 EAD in vitro, long pauses and additional sympathetic stimulation are required. We sought to determine if the same sequence of events precede paroxysmal atrial tachycardias (PATs) in ambulatory dogs.

Methods: We created myocardial infarction, complete atrioventricular block and infused nerve growth factor into the left stellate ganglion (LSG) in 8 dogs. In addition, we performed simultaneous continuous (24/7) long-term recording of left stellate ganglion (LSG) nerve activity and electrocardiogram using a radiotransmitter manufactured by Data Sciences International. One pair of electrodes was used to record stellate ganglion nerve activity from the lower portion of LSG. Another pair of widely spaced bipolar electrodes was implanted in subcutaneous tissue for surface ECG recording. PAT was defined as an abrupt increase of atrial rate to more than 3 times of baseline, or greater than 200 bpm.

Results: The simultaneous recording lasted 55±40 days. PATs were recorded in all 8 dogs (mean of 7.8±5.7 episodes/dog/day). A characteristic pattern of onset of PAT was observed in 64% of the episodes. This pattern includes profound sinus bradycardia or sinus arrest for > 3 s followed by an abrupt increase of sympathetic discharge (figure1). The average rate of atrial arrhythmia was 196.2±21.7 bpm. The average of minimal atrial rate before the atrial arrhythmia initiation was 36.8±17.5 bpm.

Conclusions: Spontaneous onset of PAT in this canine model is characterized by a long pause followed by bursts of sympathetic discharges. These patterns are consistent with the induction of arrhythmia by late phase 3 EAD.

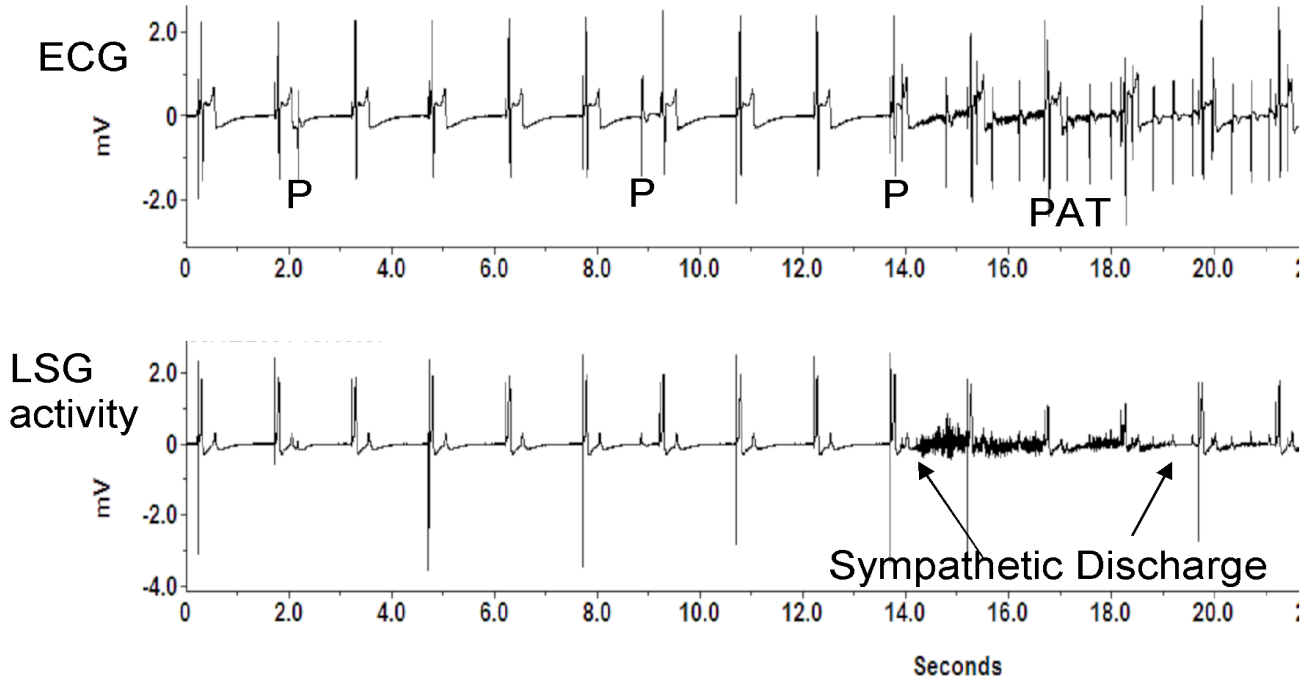

Figure 1: Profound sinus bradycardia followed by bursts of sympathetic discharge leading to PAT of 190 bpm.