EP/Device Rounds

Jumping Across the Gap - A Series of Atrial Extrastimuli

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Abstract

The "gap phenomenon" is an interesting phenomenon in electrophysiology arising from the differences in refractory periods at two or more levels of the atrioventricular (AV) conduction system. We present a patient with dual AV nodal physiology in whom the AH jump mediates the gap phenomenon. We also briefly discuss the other mechanisms of gap phenomenon that have been described in this setting.

Key words: gap phenomenon; Atrial Extrastimuli

Case

A 43-year-old female presented with recurrent palpitations, documented narrow QRS tachycardia and normal electrocardiogram during sinus rhythm. During the electrophysiology study, single atrial extrastimuli were introduced from the proximal coronary sinus at a drive cycle length of 600 ms, starting at a coupling interval of 360 ms and decrementing by 10 ms. At longer coupling intervals, atrioventricular (AV) conduction was seen with a narrow QRS. At a coupling interval of 330 ms (figure 1, panel A), conduction was seen with right bundle branch block. At a coupling interval of 320 ms, infra-Hisian AV block occurred (panel B). As the extrastimulus coupling interval was reduced to 260 ms, AV conduction resumed (panel C) and at 250 ms, narrow complex tachycardia was induced (panel D). What is the explanation for the responses seen?

Discussion

The phenomenon where AV block is seen during a window of coupling intervals with intact conduction at both shorter and longer coupling intervals has been designated as a conduction gap and was initially described by Moe et al [1]. This constitutes a discontinuity in the AV conduction curve and, in the classical explanation, is due to the functional refractory period of the proximal conducting tissue (AV node in this case) being shorter than the effective refractory period (ERP) of the distal conducting tissue (the His-purkinje system in this case). Thus, in panel B, since the H1H2 interval is 342 ms and the ERP of the His-purkinje system is longer than this, the impulse blocks in the His-purkinje system. In panel C, because of prolongation of A2H2 exceeding the decrease in A1A2, the H1H2 is longer (375 ms) and AV conduction occurs.
Figure 1: Response to atrial extrastimuli. Response to atrial extrastimuli at four different coupling intervals are shown in the four panels. In each tracing, three ECG leads, a high right atrial electrogram (HRA), His bundle recording (His d), proximal (CS 9,10) and distal coronary sinus (CS 3,4) recordings and recording from right ventricular apex (RV d) are shown. Extrastimulus coupling interval (A1A2 ) and the A2H2 intervals are marked for each panel.

Interesting in this case is the co-existence of dual AV nodal physiology with infranodal gap phenomenon. In the setting of dual AV nodal physiology, a different mechanism for gap phenomenon due to collision of the two wavefronts has also been described [2]. However the block would be AV nodal in that situation. Gap phenomenon in the fast pathway conduction due to delay in the proximal AV nodal region has also been described as another interaction of dual AV nodal physiology and conduction gap [3]. From the H1H2 curve, it is seen that extrastimuli that result in AV block correspond to the nadir of the H1H2 curve, specifically intervals shorter than 350 ms. This is consistent with the classical explanation for the gap phenomenon in our patient, with the AH jump providing the increase in proximal conduction time to allow resumption of distal conduction.

References


**Figure 2:** AV conduction curves. AH interval of the extrastimulus (A2H2, denoted as dark circles) and H1H2 intervals (squares) are plotted against the coupling intervals (A1A2). H1H2 intervals associated with conduction block after the His are denoted by an empty square while the others H1H2 intervals are filled.