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Proximal coronary sinus pacing induced prolonged asystole

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Case Report

Abstract

BACKGROUND: Atrioventricular node (AVN) is an interatrial septum structure located at the apex of triangle of Koch that modulates the transmission of impulses from atria to the ventricles. Vagally mediated AVN block with high frequency stimulation (HFS) was investigated in a few animal and human studies, but prolonged ventricular asystole that was induced by a low frequency proximal coronary sinus (CS) overdrive pacing has rarely been reported.

CASE REPORT: A 40-year-old man was admitted in our hospital for evaluation of syncope and palpitation. He presented with prolonged ventricular asystole by proximal CS pacing during electrophysiological study (EPS).

CONCLUSION: There is no comprehensive clinical study to investigate the association of vasovagal syncope with vagally mediated atrioventricular block (AVB) which is induced by posteroseptal area stimulation. Radiofrequency catheter ablation of ganglionated plexi (GP) located close to sinus node and AVN was reported to eliminate the vagal efferent output during vasovagal syncope as a new treatment strategy.

Keywords: Proximal coronary sinus; Pacing; Asystole

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Introduction

Atrioventricular node (AVN) is an interatrial septum structure which is richly innervated by sympathetic and parasympathetic fibers. Vagally mediated AVN block with high frequency stimulation (HFS) was investigated in a few animal and human studies. We report a vagally mediated atrioventricular block (AVB) with prolonged ventricular asystole that was induced by a low frequency (< 6 Hz) proximal coronary sinus (CS) overdrive pacing.

Case Report

A 40-year-old man with a history of palpitation and syncope for several years referred for ablation of atrial arrhythmia. He had no known past medical history. His physical exam and all lab work were within normal limits for his age. Electrocardiography (ECG) demonstrated normal sinus rhythm without delta waves or prolongation of P wave or QRS. Transthoracic echocardiography (TTE) showed preserved left ventricular (LV) systolic function [LV ejection fraction (LVEF) = 50%-55%] and mild mitral regurgitation. The recent episode of syncopeoccurred in standing position and was associated with a prodrome of diaphoresis and pallor that was typical for vasovagal syncope. Electrophysiological study (EPS) was performed to evaluate the cause of the patient's palpitation. Two quadripolar electrode catheters were inserted percutaneously via femoral vein and positioned in the right atrium (RA) and right ventricular (RV) apex.

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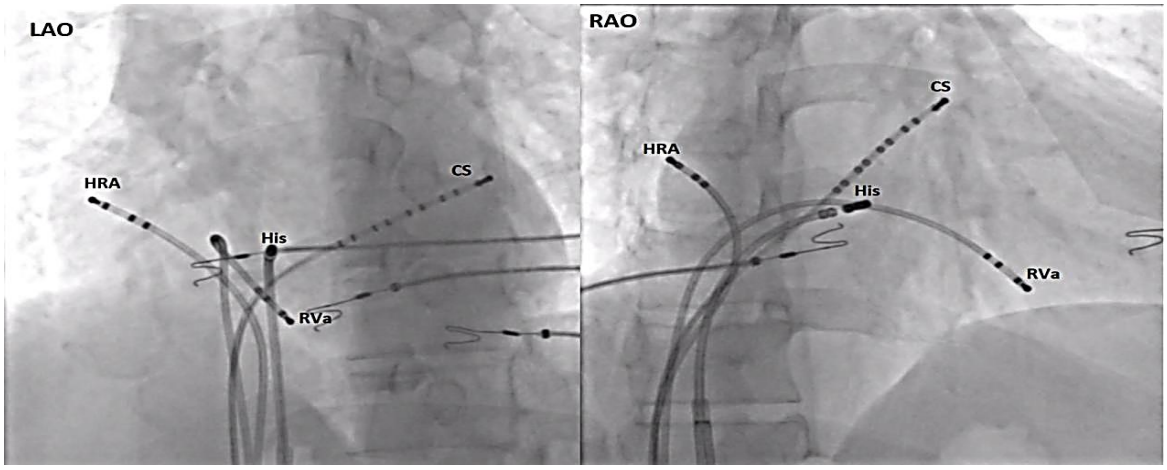

Figure 1. Left-anterior oblique (LAO) and right-anterior oblique (RAO) fluoroscopic views of catheter locations, including the decapolar catheter in the coronary sinus (CS), an ablation catheter at the His bundle (HB) region, and two quadripolar catheters in the right atrium (RA) and right ventricle (RV); as shown in the fluoroscopic views, the region of the HB recording is extended inferiorly toward the CS ostium.

A decapolar catheter was also placed in the CS from the femoral vein. The ablation catheter was positioned at the His bundle (HB) region (Figure 1). Baseline intracardiac recording showed normal atrial-HB (AH) and His-ventricular (HV) intervals. Effective refractory period (ERP) of antegrade AVN was < 280 ms without isoproterenol infusion (Figure 2). Baseline recording intervals and pacing maneuvers considered normal supra and infra-Hisian conduction.

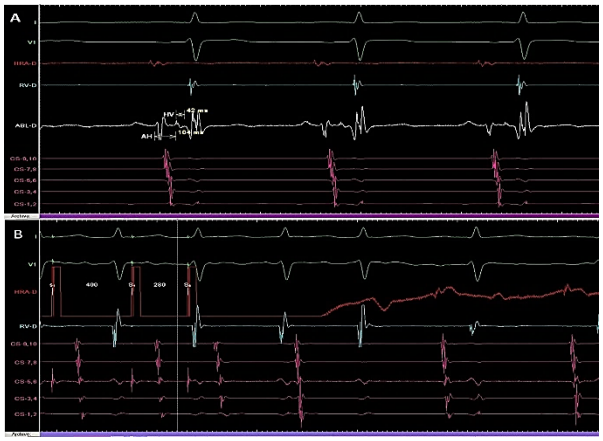

Figure 2. A) Normal baseline intracardiac intervals [atrial-His bundle (AH), His-ventricular (HV)]; B) Extrastimulation from the high right atrium (HRA) catheter leading to one to one antegrade conduction even with a premature cycle length (CL) of 280 ms

The AH interval was prolonged exaggeratedly during proximal CS pacing, which continued even after stopping the pace for a short time (Figure 3A). This response can be due to an antegrade concealed penetration of the atrial impulses into the AVN, but induction of atrial flutter (AFL) with atrial cycle length (CL) of 180 ms and 2:1 conduction ratio weakened the possibility of this explanation (Figure 3B).

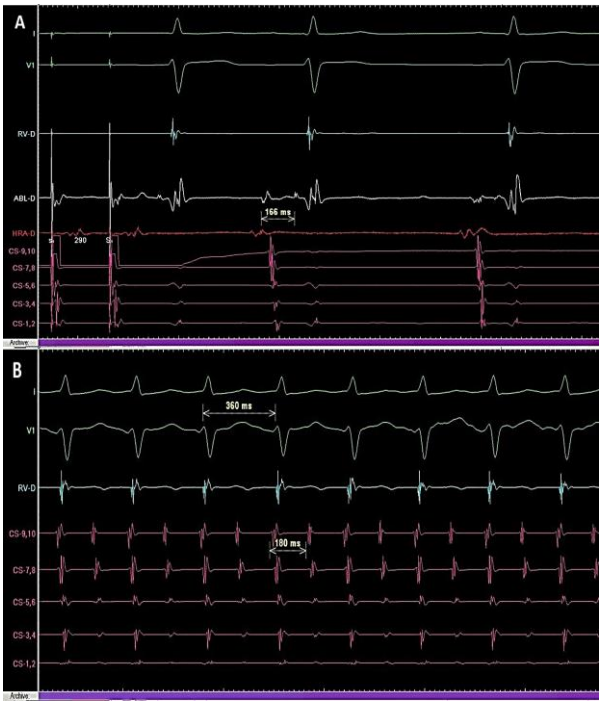

Figure 3. A) Atrial-His bundle (AH) prolongation after stopping the proximal coronary sinus (CS) stimulation with the cycle length (CL) of 290 ms; B) Atrial flutter (AFL) with the atrial CL of 180 ms and 2:1 atrioventricular (AV) ratio

Interestingly, overdrive pacing (6 V, 0.4 ms) from proximal CS at the CL of 180 ms resulted in prolonged ventricular asystole repeatedly (Figures 4A and 4B).

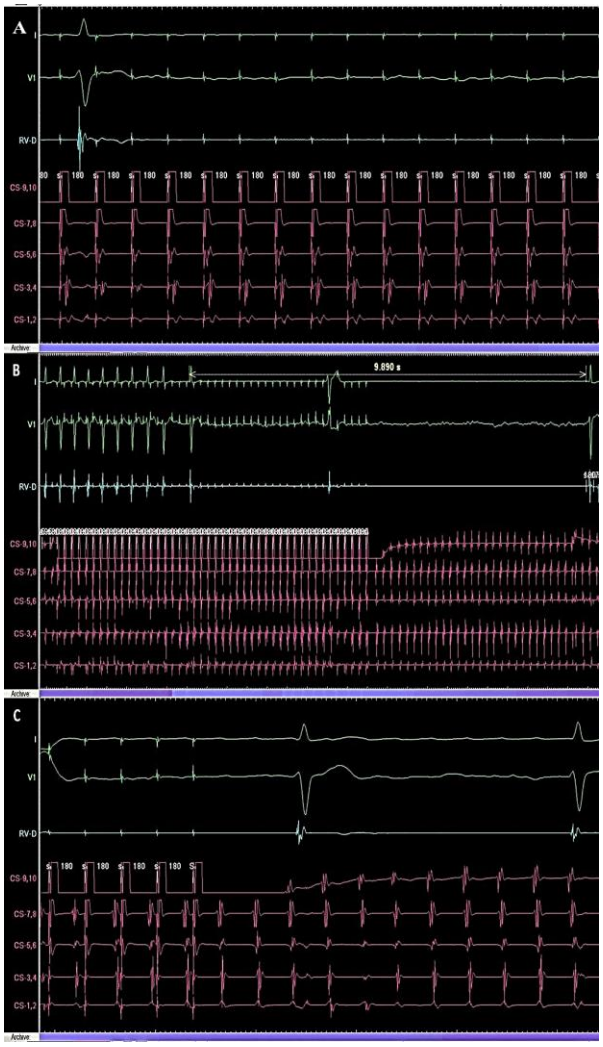

Figure 4. A and B) Prolonged ventricular asystole during proximal coronary sinus (CS) overdrive pacing with the cycle length (CL) of 180 ms; C) Atrioventricular (AV) nodal conduction suppressed again by proximal CS overdrive pacing without atrial capturing

This finding was not related to atrial capturing by proximal CS pacing and it even happened by pacing during atrial fibrillation (AF) rhythm (Figures 4C and 5A). The AVN conduction was not suppressed again by changing the site of stimulation from proximal to distal of CS or high RA (HRA) (Figure 5B). In this case, regarding to our evidence, the best explanation for ventricular asystole during proximal CS stimulation is a vagally mediated AVB.

Discussion

The AVN is the only normal electrical connection between the atria and the ventricles that modulates the transmission of impulses from atria to the ventricles. AVN is an interatrial septum structure which is located at the apex of triangle of Koch, beneath the RA endocardium. It is bordered anteriorely by the annulus of tricuspid valve, posteriorely by the tendon of Todaro, and inferiorely by the orifice of the CS ostium.[1-3]

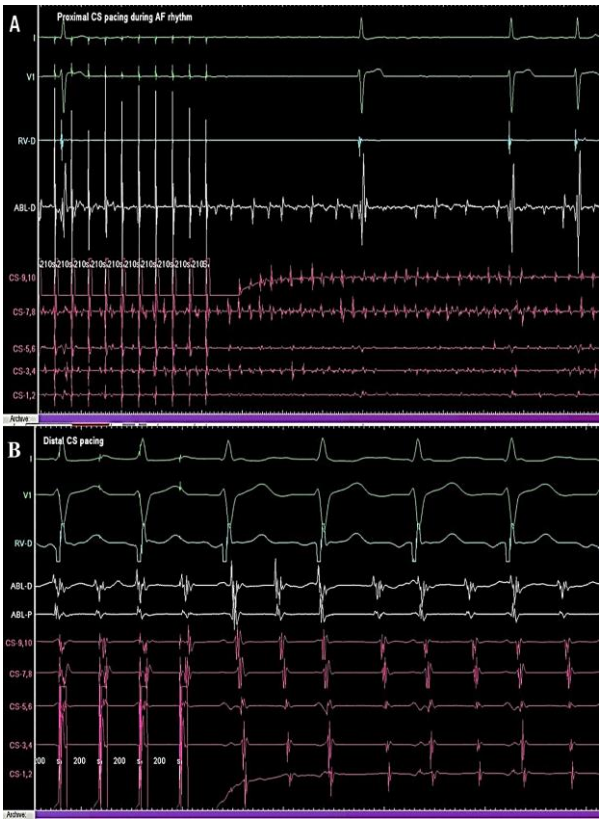

Figure 5. A) Proximal coronary sinus (CS) stimulation at the cycle length (CL) of 210 ms, while the patient being in atrial fibrillation (AF) rhythm associated with ventricular pauses; B) Distal CS pacing at the CL of 180 ms not resulting in atrioventricular (AV) nodal suppression, unlike proximal CS stimulation

The normal AV junction area can be divided into three distinct regions including: the transitional cell zone, the compact AVN, and the penetrating part of the HB. AVN limits the number of impulses conducted from atria to the ventricles. This function is important especially during very fast atrial rates such as AFL and AF.[2,3]

The AVN region is richly innervated by sympathetic and parasympathetic fibers. Sympathetic stimulation accelerates AVN conduction velocity and shortens refractoriness time, while vagal stimulation prolongs AVN conduction time and refractoriness. The negative dromotropic effect of vagal stimulation is due to activation of the inwardly rectifying potassium channel (IKACh), which results in hyperpolarization and action potential shortening of the AVN cells.[3,4]

Vagally mediated AVB can occur in various conditions including: cough or hiccups, swallowing or micturition, myocardial infarction (MI), carotid sinus massage, during radiofrequency ablation (RFA) in posteroseptal area or around superior pulmonary veins, and by HFS in posteroseptal area.[5]

Bianchi et al. investigated the effect of temporary HFS in 12 patients with AF rhythm. He implanted an atrial lead in the posteroseptal RA, where advanced AVB was achieved by HFS (4.3 V, 0.2 ms, 50 Hz).

In another study, he also investigated the effect of AVN stimulation (AVNS) to reduce ventricular rate during AF to prevent inappropriate implantable cardioverter-defibrillator (ICD) shocks. In 44 patients with paroxysmal or persistent AF who were candidates for cardiac resynchronization therapy (CRT), an RA lead was placed in the posterior RA for vagal stimulation. AVNS increased ventricular interval > 25% in 81% of patients without effect on safety profile of the CRT and defibrillation system.[6,7]

In our case, the overdrive suppression of AVN and antegrade concealed conduction of atrial impulses into the AVN is very unlikely because the ventricular asystole was induced by proximal CS pacing, while the patient's rhythm was AF. In addition, it occurred without atrial capturing. Vagally mediated AVN block by a low frequency, low amplitude proximal CS stimulation (6 V, 0.4 ms, 5 Hz) is an unusual finding during EPS. Our patient had a history of vasovagal syncope. There is no comprehensive clinical study to investigate the association of vasovagal syncope with vagally mediated AVB which is induced by posteroseptal area stimulation. As a new treatment strategy, radiofrequency catheter ablation of ganglionated plexi (GP) located close to sinus node and AVN was reported to eliminate the vagal efferent output during vasovagal syncope.[8]

Acknowledgments

None.

Conflict of Interests

Authors have no conflict of interests.

Authors' Contribution

SM: Final approval of the version to be published
JS and MM: Drafting of the article and revision for important intellectual content
FK: Conception and design

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