Received: 6 September 2018 | Revised: 30 September 2019 | Accepted: 8 October 2019

DOI: 10.1002/ccr3.2539

CASE REPORT

Clinical Case Reports Open Access WILEY

Applying sinus node function testing to evaluate perioperative management for patients suffering from tachycardia-bradycardia syndrome before atrial fibrillation ablation

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Funding information
This study was supported by the Clinical Research Award of the First Affiliated Hospital of Xi'an Jiaotong University, China (No. XJTU1AF-CRF-2015-007).

Abstract

Sinus node (SN) function is an important prognostic factor of atrial fibrillation (AF). However, AF ablation guideline has not recommended SN function test before ablation. SN function testing before AF ablation in patients with tachycardia-bradycardia syndrome should be beneficial to determine further therapy strategies and prognosis.

KEYWORDS

ablation, atrial fibrillation, permanent pacemaker, prolonged RR, sinus node dysfunction, vagal stimulation

1 | INTRODUCTION

Sinus node (SN) function is important for evaluating the prognosis of atrial fibrillation (AF). Electrical and structural remodeling promotes both AF and sinus node dysfunction (SND). Two patients manifested tachycardia-bradycardia syndrome (TBS), yet developed different prognoses. Therefore, SN function testing in TBS is necessary to determine further therapy approaches and prognosis.

Sinus node function is an important prognostic factor of atrial fibrillation (AF), especially in AF ablation. Prolonged RR interval after conversion of AF, which is the basis of tachycardia-bradycardia syndrome (TBS), often requires implantation of a permanent pacemaker for rhythm control.[1] Previous studies have reported that both AF and prolonged RR can be eliminated after ablation so that a pacemaker might be averted in most patients.[2] However, some patients have complained about disease relapses, contradicting the point that treatment of one may cure the other. Therefore, it is imperative for clinicians to formulate better therapies according to the mechanisms of this syndrome.

The mechanisms of TBS remain ill-defined. There are different pathologies, including intrinsic factors, such as sinus node dysfunction (SND), progressive fibrosis, electrical remodeling, fibrotic atrial cardiomyopathy, parasympathetic stimulation, and vagal stimulation, as well as extrinsic factors, such as inflammation, ischemia, and drug use. SND is the most common cause of TBS among these factors. Consequently, SN function testing before ablation is a superior choice to determine prognosis. Here, we report two patients who both suffered from prolonged RR after AF conversion yet developed different prognoses, one of whom had recurrent prolonged RR and was implanted with a permanent pacemaker due to SND.

2 | CASE REPORT

Two female patients with a diagnosis of TBS, who manifested with prolonged RR on termination of AF, were admitted to our hospital. The electrocardiogram (ECG) of patient A showed bradycardia after episodes of AF before ablation (ECG not obtained). She was treated with ablation

but suffered significant prolonged RR up to 6.48 seconds (Figure 1) during the ablation after AF conversion. As a result, she was implanted with a temporary pacemaker immediately after the ablation for heart rate control. On the third postoperative day, the temporary pacemaker was removed, and she no longer had any discomfort or documented bradycardia after ablation. Patient B had been afflicted by syncope due to episodes of prolonged RR up to 4.92 seconds (Figure 2) during the past 3 months before hospitalization. She was treated with the same instruments, and the procedure went smoothly without complications. However, on the first day after ablation, she underwent several prolonged RRs again, with the longest RR interval of up to 7.05 seconds (ECG not obtained); therefore, she was immediately implanted with a permanent pacemaker to maintain the normal sinus rhythm and to prevent malignant cardiac events. Transesophageal atrial pacing (TEAP) was performed six months postablation for both patients. The result of patient A indicated normal SN and atrial ventricular node function, suggesting her prolonged RR might be due to transient vagal stimulation-induced electric remodeling. ECG monitoring was conducted for patient B 3 months postpermanent pacemaker implantation, which showed her whole course pacing rate. We also carried out an atropine test for patient B, which indicated that her heart rate was only 60 bpm and her PR interval was not obviously affected after administration of 2 mg atropine, further indicating that the prolonged RR was due to SND.

3 | DISCUSSION

Our case report consists of two patients afflicted by TBS with different causes and prognoses after ablation. The main learning point is that SN function testing is valuable for guiding clinical management and informing prognosis. The relevant mechanisms and management strategies according to our cases will be discussed.

AF and SND interact with each other by electric remodeling due to fibrosis. Fibrous accretion in SN tissue associates with a decrease in the heart rate in itself and extended sinoatrial (SA) conduction time in mammalian hearts. A decline in velocity of phase 0 depolarization has been observed around SN and possibly represents a reduction in the sodium current. Moreover, fibroblasts can separate pacing cells, thus reducing the SA rhythm via decreases in adjacent cellular junctions. Accordingly, all of these alterations account for the prolonged SA conduction course and the SA block of senility.[3]

The bidirectional relationship between AF and SND has been studied in many in vivo trials. Electrophysiological mapping of patients with AF and SND has shown defective atrial myocardium, slow atrial conduction, extensive atrial scarring, abnormal patterns of SN activation, and aberrant SA conduction pathways. These clinical findings suggest that an intervention process would be indispensable to redress the abnormal function of the SN complex; moreover, an expanded range of ablation is usually needed to terminate this abnormality.[4]

Controversy remains regarding whether a prolonged RR interval after AF termination results from autonomic regulation or SND. Marked sinus bradycardia induced by vagal stimulation is a common phenomenon in the clinical setting. This alteration results in significant dispersal of refractoriness in the atrial myocytes via cholinergic effects on the I_{kAch} and I_{Ado} channels, thus establishing the basis for temporary functional reentry and fibrillation.[5] However, some studies have reported a significant increase in the heart rate (HR) following AF ablation. The expanded pulmonary vein isolation (PVI) area may create deeper transmural lesions in the atrial myocardium, with more extensive destruction of the vagal fibers, leading to attenuated parasympathetic activities. Reduced parasympathetic modulation was found in both the TBS and control group patients without SND and is considered as an essential effect of PVI. Although the mean HR increased markedly in the control group, it did not significantly alter in the TBS group, possibly due to the pre-existing SND. Therefore, the prolonged RR interval with sinus

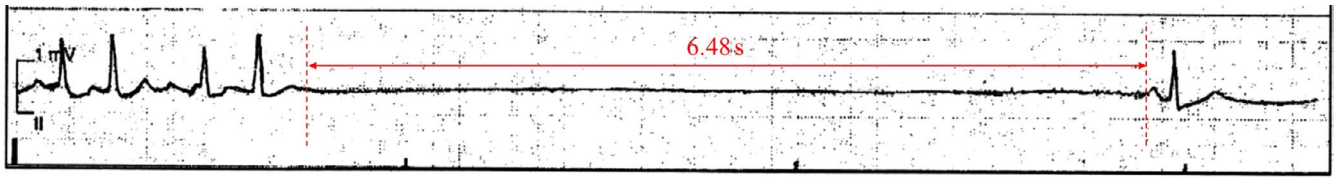

FIGURE 1 ECG of patient A before ablation: prolonged RR interval by 6.48 s

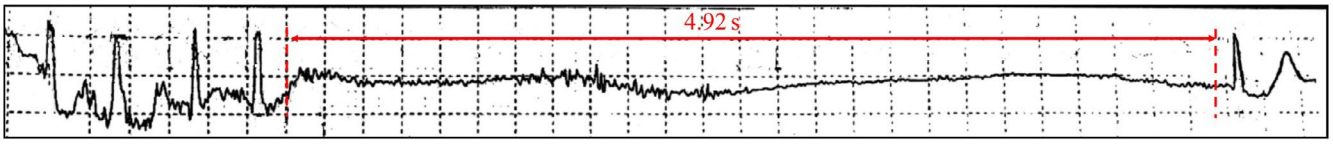

FIGURE 2 ECG of patient B before ablation: prolonged RR interval by 4.92 s

bradycardia after ablation was more likely due to SND but not autonomic regulation.[6-8]

It is clear that as AF and SND are caused by electric remodeling, therapy of one may cure the other. However, structural remodeling, such as fibrosis, further promotes the tendency to develop both arrhythmias. Accordingly, imaging tests and SN function examinations that can detect the extent of fibrosis and SN function are vital to improve the understanding of the atrium and the SN.[9,10]

Studies have suggested that the most useful method in the evaluation of SN function is to combine the atropine and exercise tests and to evaluate the sinus node recovery time (SNRT).[11] While intracardiac electrophysiologic study (IEPS) remains the gold standard in the evaluation of SNRT, TEAP is emerging as a minimally invasive method that is safe, effective, convenient and inexpensive and can be performed in outpatient service.[12] We did not perform invasive IEPS on our patients during AF ablation, as this invasive examination is difficult to be accepted by patients postprocedure. Thus, TEAP was chosen as an alternate method to evaluate SN function. The accuracy in diagnosing and characterizing various types of supraventricular arrhythmias between TEAP and IEPS was shown to be highly consistent; therefore, TEAP should be considered as a substitution method for IEPS to avoid possible complications due to the invasive nature of IEPS.[12-14]

In conclusion, SN function testing is often neglected for patients with TBS in the clinic, and AF guidelines for ablation have not yet recommended SN function testing before atrial fibrillation.[15] The present two cases indicate that SN function testing before AF ablation in patients with TBS might be beneficial to determine further therapy strategies and prognosis.

ACKNOWLEDGMENTS

We appreciate the patients' cooperation in this study.

CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

CFS and HBL: provided the data. DH: wrote the paper, and JQS: revised it. DH and HBL: performed the follow-up work.

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How to cite this article: Han D, She J, Sun C, Li H. Applying sinus node function testing to evaluate perioperative management for patients suffering from tachycardia-bradycardia syndrome before atrial fibrillation ablation. *Clin Case Rep*. 2019;7:2505–2507. https://doi.org/10.1002/ccr3.2539