Intra-Atrial Conduction Block Mimicking Atrioventricular Nodal Block After Multiple Catheter Ablation Procedures for Atrial Tachycardia in a Patient with Cardiomyopathy

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Intra-Atrial Conduction Block. A 42-year-old woman with a history of cardiomyopathy and multiple ablation procedures for atrial tachycardia developed intra-atrial conduction block that mimicked atrioventricular (AV) nodal block during radiofrequency ablation at the cavotricuspid isthmus. She was treated with atrial pacing (from the coronary sinus), which overcame intra-atrial conduction block and resulted in AV nodal conduction. *(J Cardiovasc Electrophysiol, Vol. 23, pp. 1258-1261, November 2012)*

atrial flutter, atrioventricular node, AV block, cardiomyopathy, catheter ablation, pacemaker

Atrioventricular (AV) nodal block during catheter ablation of typical atrial flutter is unusual but has been previously reported. The mechanism is thought to be due to either direct injury[1] to the AV node or the distal conduction system or to the artery supplying the node.[2] However, intra-atrial conduction block mimicking AV nodal block in a patient undergoing catheter ablation at the cavotricuspid isthmus has not been previously reported.

Case Report

A 42-year-old woman with a history of postpartum cardiomyopathy presented for a repeat ablation procedure for recurrent atrial tachycardia (AT). She had previously undergone 2 ablation procedures for AT. During her first procedure, macroreentrant ATs involving the right atrial (RA) septum and the cavotricuspid isthmus (CTI) were targeted. The former was targeted in a linear fashion, between the superior and inferior venae cavae. During the second procedure, macroreentrant ATs from the left atrial (LA) roof, mitral isthmus, and anterior wall were ablated. Shortly after the procedure, the patient developed recurrent AT despite antiarrhythmic medications. This resulted in deterioration of the left ventricular ejection fraction (EF), from 0.40 at baseline to 0.20 during tachycardia, with worsening of her functional status. The LA diameter was 51 mm.

The patient presented to the laboratory in AT with a cycle length of 420 ms. The diagnosis was macroreentry involving the LA appendage. Linear ablation (Thermocool, Biosense-Webster, Diamond Bar, CA, USA) from the anterolateral mitral annulus, along the posterior base of the appendage, and to the anterosuperior aspect of the left superior pulmonary vein (PV), terminated the tachycardia to sinus tachycardia with a first degree AV block, which was her intrinsic rhythm. Antral PV isolation and complete linear block at the mitral isthmus and roof were achieved to prevent recurrence. Since the patient had previously undergone ablation of typical flutter, conduction across the CTI was checked. While pacing from the mid coronary sinus (CS), there was evidence of conduction, prompting radiofrequency (RF) energy delivery at the CTI (6 o'clock in the left anterior oblique view) using 35 watts of irrigated RF energy. Shortly after commencing with RF energy delivery, a double potential appeared on the distal bipole of the ablation catheter, suggestive of conduction delay/block (Fig. 1). However, the double-potential interval was not constant, and appeared to keep increasing despite discontinuation of RF energy. When atrial pacing from the CS was discontinued, the resultant rhythm was sinus tachycardia with apparent complete AV nodal block (Fig. 2). CS pacing was immediately reinstituted, resulting in 1:1 AV nodal conduction. Biatrial mapping revealed that majority of atrial tissue was activated by the sinus rhythm wavefront. However, the sinus rhythm wavefront blocked at the LA roof, and the inferoposterior LA, which was depolarized only by the paced wavefront from the CS. The sinus rhythm wavefront also blocked at the low RA septum, and the mid and proximal aspects of the CS; and these regions could only be activated during CS pacing.

Since the patient's intrinsic conduction did not return, a decision was made to implant a dual chamber pacemaker, with the atrial lead (St. Jude 1158T, St. Paul, MN, USA) placed in the CS, and a ventricular lead (Medtronic 5076, Minneapolis, MN, USA) in the right ventricular apex (Fig. 3A,B). The pacing threshold of the atrial (CS) lead was 0.75 V at a pulse-width of 0.5 ms, and remained stable at the last device interrogation. The pacing mode was AAIR/DDDR (Medtronic Adapta), with a lower and upper rate of 60 and 150 beats per minute, respectively. Three months after the ablation procedure, the EF improved to 0.45. At the last follow-up, 15 months after the ablation procedure, the patient remains free of mode-switch episodes in the absence

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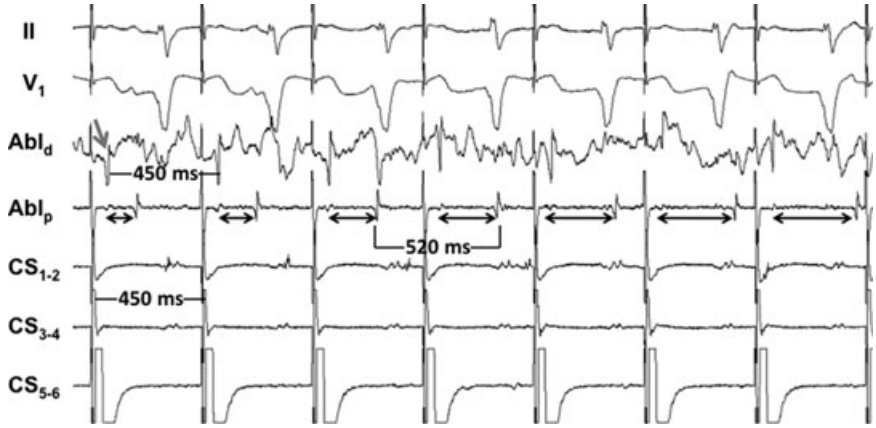

Figure 1. *Effect of RF energy delivery at the cavotricuspid isthmus during pacing from the coronary sinus (CS). Note the emergence of double potentials on the proximal bipole of the ablation catheter (double-headed arrow), the interval of which seems to be prolonging. The stimulus-to-QRS interval is 250 ms, and was shorter than the PR interval during sinus rhythm (330 ms, not shown), indicating that the paced wavefront engages the AV node more quickly as compared to the sinus rhythm wavefront.*

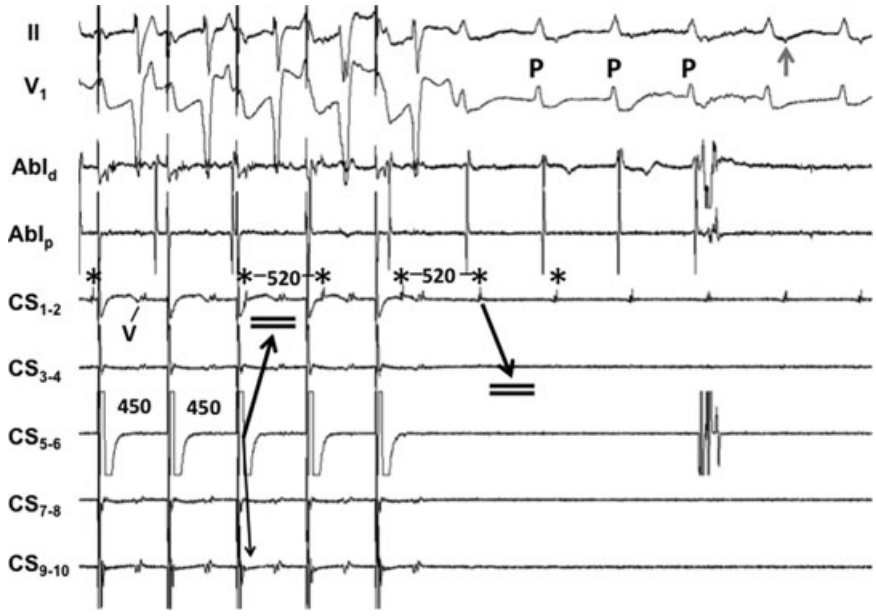

Figure 2. *(Continuation of Fig. 2 at a slower paper speed). Upon cessation of pacing from the mid CS, the resultant rhythm is sinus tachycardia ("P") with lack of AV nodal conduction due to intra-atrial block. During pacing from the mid CS, the distal CS electrogram (*) is not advanced to the pacing rate, consistent with conduction block from the mid to the distal CS. There is also entrance block from the distal to the rest of the CS during sinus rhythm. The gray arrow refers to terminal negativity of the p-wave, consistent with impaired conduction over Bachmann's bundle. The artifact on the recording is due to the abrupt withdrawal of the ablation catheter. V = far-field ventricular electrogram.*

of antiarrhythmic medications. Device interrogation also revealed that the percentage of RV pacing was 44%. However, this was likely an overestimate as a 12-lead electrocardiogram at the same time showed evidence of pseudofusion, i.e., the QRS complex was narrow (92 ms).

Discussion

Although prior reports have documented the possibility of AV nodal block during RF ablation at the CTI, the mechanism is likely direct injury to the AV node, or to the right coronary artery. In this patient, RF energy delivery at the CTI resulted in intra-atrial block, which mimicked AV nodal block. It is likely that in this patient who had undergone multiple prior ablation procedures, input into the AV node during sinus rhythm was dependent upon conduction through the low RA/cavotricuspid isthmus. This input into the septum/AV node was inadvertently eliminated during ablation, resulting in apparent AV nodal block. However, the AV node could still be engaged during CS pacing, confirming that the compact AV node was indeed intact.

Anatomic Considerations

A prior anatomic study showed that the AV node contains posterior extensions[3] that may have important electrophysiologic implications. The majority of specimens had both right and left posterior extensions. Interestingly, the rightward extensions extended beyond the anterior margin of the CS and continued on to the inferoposterior free wall of the right atrium, i.e., the CTI region, in one-third of the anatomic specimens. Prior to RF ablation at the CTI in our patient, the sinus rhythm wavefront likely engaged the AV node via the right posterior extension (Fig. 4). However, conduction over these fibers was probably impaired secondary to atrial uncoupling, both related to prior ablation and the presence of cardiomyopathy (PR interval prior to the first ablation procedure was 240 ms). The right posterior extension was inadvertently eliminated resulting in apparent AV nodal block. It is possible that the posterior input extended to the CTI region in this patient, where it was injured by RF ablation. However, the compact AV node could still be engaged during CS pacing, likely via the *left* posterior AV nodal extension. If the left input were absent in our patient, as it is in about one-third of the human specimens, CS pacing may not have been able to overcome the intra-atrial conduction block, and the patient would have required ventricular pacing, with its attendant implications.

Apart from conduction block at the low RA as a result of RF energy delivery at the CTI, there is also evidence of other conduction derangements. Conduction over Bachmann's bundle was probably impaired as evidenced by the p-wave morphology during sinus rhythm. Terminal negativity of

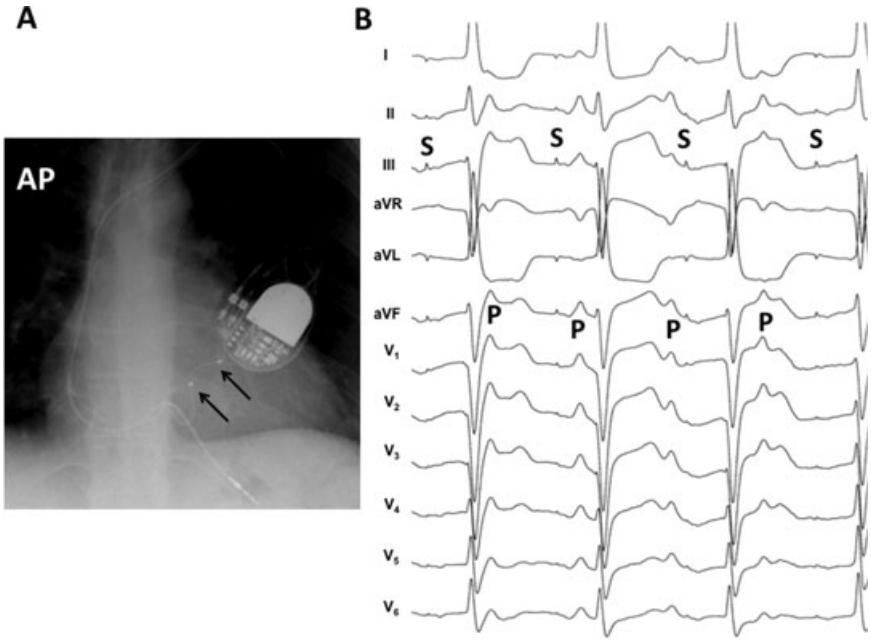

Figure 3. *A: Postoperative chest X-ray in the anteroposterior projection. The atrial lead was placed in the mid coronary sinus (arrows). The ventricular lead was placed in the right ventricular apex. B: A 12-lead electrocardiogram obtained after pacemaker implantation (50 mm/s). The intrinsic rhythm is sinus tachycardia ("P") at 520 ms, which is dissociated from the pacing stimuli ("S") delivered at 860 ms. Atrial pacing via the CS electrode results in intrinsic AV nodal conduction and a narrow QRS complex (92 ms).*

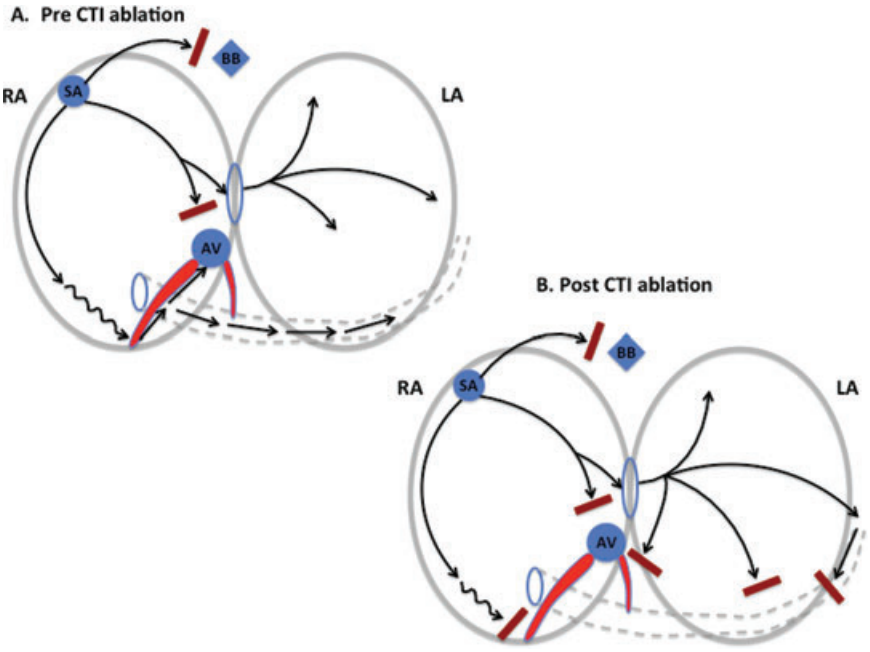

Figure 4. *Proposed conduction patterns during sinus rhythm prior to (A) and post (B) ablation at the cavotricuspid isthmus (CTI). The right and left posterior extensions of the atrioventricular (AV) node are shown in red. The narrow oval between the right and left atria denotes the fossa ovalis. The dashed tubular structure represents the coronary sinus (CS). See text for details. BB = Bachmann's bundle; LA = left atrium; RA = right atrium; SA = sinus node.*

the p-wave in the inferior leads (Fig. 2) is consistent with Bachmann's bundle block.[4] However, interatrial conduction between the high right atrial septum and the anterior LA was intact, even after ablation at the CTI. Since the usual route of interatrial conduction (via Bachmann's bundle) was impaired, and the secondary route (via the CS) was unavailable, interatrial conduction was likely mediated by the fossa ovalis (Fig. 4).[5] The wavefront from the RA septum was unable to access the compact AV node, likely owing to conduction block between RA myocardium and the superior/anterior aspect of the compact AV node. There is also evidence of conduction block from the mid to the distal CS, since the distal CS electrogram could not be advanced to the pacing rate during mid CS pacing (Fig. 2). When CS pacing was discontinued, there was also entrance block to the mid and proximal CS during sinus rhythm (Fig. 2). Lastly, there was also entrance block to the low posterior LA (not shown). Although these areas were completely isolated from the remaining atrial mass during sinus rhythm, they could still be activated during CS pacing.

Choice of Device Therapy

In this patient with left ventricular dysfunction and heart failure, several pacing options were considered. These included a standard dual chamber pacemaker, dual chamber implantable cardioverter-defibrillator (ICD), and ICD with resynchronization capability. A standard dual chamber pacemaker, with the atrial lead in the RA appendage, and the ventricular lead in the right ventricular (RV) apex, would result in right ventricular pacing, with the possibility of electromechanical dyssynchrony. In this patient with structural heart disease, there was a concern of worsening left ventricular dysfunction related to RV pacing. Although biventricular

pacing has the potential to overcome the deleterious effect of apical RV pacing, it was felt that taking advantage of the intrinsic AV nodal conduction would be the best option in this young patient. Therefore, the atrial lead was placed in the mid CS, which resulted in intrinsic AV nodal conduction.

One could also consider adding another atrial lead in the RA appendage, which may in conjunction with the atrial lead in the CS, improve interatrial dyssynchrony. One could either program a very short RA-CS delay or program the device to a triggered mode where an atrial paced event (from the CS channel) would be delivered upon a sensed atrial event (in the RA channel). We chose to keep the procedure simple, however, and decided to implant the minimum number of leads. We were specifically concerned that if the CS lead dislodged, the patient would require RV apical pacing, with concomitant dyssynchrony and worsening EF and heart failure symptoms. This would have to be treated with implantation of 2 additional leads (RV ICD lead, and a left ventricular lead for resynchronization). If we had placed an additional atrial lead in an attempt to overcome atrial uncoupling, there would be potentially 5 leads in situ in this young patient, which obviously poses problems.

The disadvantage of the approach that was chosen is that AV synchrony was not restored, despite restoration of sinus rhythm. Although atrial pacing via the CS resulted in AV nodal conduction, the majority of atrial tissue was still activated by the sinus rhythm wavefront owing to various conduction derangements. Also, since there was complete entrance block to the pacing site in the CS, the CS electrode would not be expected to capture recurrent atrial arrhythmias unless the source of the arrhythmia resided in the low RA/LA and portions of the CS venous system.

Clinical Implications

It may be tempting to consider CS pacing in an effort to preserve intrinsic AV nodal function in patients who develop AV block as a result of catheter ablation of the slow pathway for supraventricular tachycardia or typical flutter. However, this is unlikely to be effective in the majority of such patients. First, one-third of the patients lack a left posterior extension, and hence the AV node cannot be accessed from the CS in these patients. Second, the unique set of conduction derangements described above probably facilitated our ability to take advantage of the left posterior AV nodal extension in this patient.

Conclusion

In patients with underlying structural heart disease or who require multiple procedures for atrial fibrillation/AT, catheter ablation may be complicated by further atrial uncoupling,[6-8] resulting in mechanical and electrical derangements. Therefore, one should tailor the ablation procedure to precisely target the culprit arrhythmia and avoid excessive ablation in these patients.

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