Catheter ablation as a treatment of atrioventricular block

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BACKGROUND Symptomatic second-degree atrioventricular (AV) block is typically treated by implantation of a pacemaker. An otherwise healthy AV conduction system can nevertheless develop AV block due to interference from junctional extrasystoles. When present with a high burden, these can produce debilitating symptoms from AV block despite an underlying normal AV node and His–Purkinje system properties.

OBJECTIVE The purpose of this study was to describe a catheter ablation approach for alleviating symptomatic AV block due to a ventricular nodal pathway interfering with AV conduction.

METHODS Common clinical monitoring techniques such as Holter and event recorders were used. Standard electrophysiological study techniques using multipolar recording and ablation catheters were utilized during procedures.

RESULTS A 55-year-old woman presented with highly symptomatic, high-burden second-degree AV block due to concealed and manifest junctional premature beats. Electrophysiological characteristics indicated interference of AV conduction due to a concealed ventricular nodal pathway as the cause of the AV block. The patient’s AV nodal and His–Purkinje system conduction characteristics were otherwise normal. Radiofrequency catheter ablation of the pathway was successful in restoring normal AV conduction and eliminating her clinical symptoms.

CONCLUSION Pathways inserting into the AV junction can interfere with AV conduction. When present at a high burden, this type of AV block can be highly symptomatic. Catheter ablation techniques can be used to alleviate this type of AV block and restore normal AV conduction.

KEYWORDS Accessory pathway; Atrioventricular block; Catheter ablation; His extrasystole; Junctional premature beat

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Introduction

The occurrence of second-degree atrioventricular (AV) block is relatively common, especially in situations of higher vagal tone. When it is transient and not associated with significant symptoms, no treatment is needed. However, treatment is generally recommended when episodes of second-degree AV block are more prolonged and are associated with troublesome symptoms that interfere with daily activities, even if those are related to AV nodal block. With the rare exception of excessive vagotonia for which parasympathetic ganglion ablation has been reported to be useful,1 treatment of symptomatic second-degree AV block generally requires insertion of a pacemaker to maintain AV synchrony.2 We report a case of symptomatic and frequent second-degree AV block unrelated to vagotonia that was successfully treated by radiofrequency ablation. To our knowledge, this is the first case of AV block successfully treated with conventional cardiac catheter ablation.

Methods

Standard electrocardiographic (ECG) monitoring techniques including 12-lead ECG and Holter and event monitoring were used to assess patient symptoms and to correlate them with concurrent cardiac rhythms. Electrophysiological studies were performed using multipolar pacing and recording catheters. Surface ECGs and intracardiac electrograms were recorded using a GE Healthcare Mac-Lab CardioLab recording system (GE Healthcare, Wauwatosa, WI). Programmed stimulation was performed using a Bloom Electrophysiology (Fisher Medical, Wheat Ridge, CO) multichannel programmable stimulator. Catheter mapping was performed using a Biosense CARTO 3 mapping system (Biosense Webster), Radiofrequency ablation was performed using a contact-force sensing saline-cooled catheter (ThermoCool SmartTouch, Biosense Webster, Irvine, CA), and radiofrequency energy was delivered via a Stockert GmbH SmartAblate system (Biosense Webster, Inc, Irvine, CA).

Results

A 55-year-old woman with a long history of palpitations attributed to premature atrial contractions and premature ventricular contractions presented with new onset of spells of lightheadedness lasting 5–10 seconds. Twelve-lead ECG
showed normal sinus rhythm and normal conduction intervals without evidence of ventricular preexcitation (see Supplemental Figure 1). Ambulatory Holter monitors showed brief episodes of 2:1 AV block associated with symptoms of lightheadedness. These occurred briefly in the daytime, once or twice a day, unrelated to sinus slowing or preceding PR prolongation. These Holter recordings also showed episodes of various premature beats associated with palpitations (see Figure 1 legend for detailed description). A referral was made for consideration of pacemaker implantation because of symptomatic second-degree AV block.

At the consultation, a review of the Holter findings indicated that clinical vagal reflexes were unlikely to be the cause of the 2:1 AV block events. No PR prolongation or PP interval prolongation preceded these events. Other than during the episodes of AV block, conduction properties of the AV junction appeared to be relatively normal: the AV junction was clearly able to conduct at faster rates at various times. Even at times adjacent to the occurrence of 2:1 AV block, premature atrial beats seemed to conduct through the AV junction. An exercise test showed normal AV conduction throughout the test, and the patient achieved a peak heart rate of 166. There was no ECG evidence of His–Purkinje system (HPS) conduction abnormalities. Thus, based on the findings shown in Figure 1, the agent causing AV block was suspected to be either 1:2 AV nodal conduction or junctional premature beats (JPBs).

In 1:2 AV nodal conduction, a sinus beat can generate a delayed activation of the ventricle via conduction through the slow pathway. However, should the conduction via the slow pathway block in the distal node due to its prematurity, it may then interfere with conduction of the next sinus beat. JPBs can act similarly. With this in mind, an electrophysiological study of the AV conduction system was performed. The baseline rhythm was normal sinus with normal AH and HV intervals. The His potential had normal morphology and duration (see Supplemental Figure 2). There was no
spontaneous occurrence of AV block or JPB during the study. 1:1 AV conduction occurred down to a cycle length (CL) of 430 ms, with Wenckebach block occurring at 420 ms. Block was above the level of the His bundle preceded by prolongation of the AH interval. There was absence of retrograde conduction at baseline when the patient was tested under a lightly sedated state. Following atropine infusion, 1:1 AV conduction occurred down to a CL of 320 ms, with block again occurring above the His. Retrograde 1:1 conduction now occurred down to a CL of 340 ms. Procaïnamide was infused up to 10 mg/kg. Mild prolongation of HV interval from 31 to 40 ms was noted, but conduction through the AV node and His bundle remained excellent. There was no notable jump in AH conduction time noted during atrial premature beat testing. An AV nodal slow pathway ablation was performed empirically based on presumed occurrence of double AV nodal conduction generating the JPBs. The PR interval remained at baseline postablation.

On follow-up, the symptoms associated with 2:1 AV block and premature narrow complex beats continued to occur but still with short durations. Ectopic JPBs, rather than 1:2 AV nodal conduction, were now considered to be a likely source of these arrhythmias. Thus, an inpatient trial of flecainide was initiated under ECG monitoring in an effort to suppress possible ectopy from the His or AV node. Baseline in-hospital monitoring showed occasional premature beats. After flecainide was started, the frequency of premature beats increased dramatically, and 2:1 AV block appeared with increasing burden at incremental drug doses from 50 to 150 mg twice daily (see Supplemental Figure 3). Flecainide was stopped, and the patient’s rhythm returned to baseline. She did not want a pacemaker because the frequency and duration of symptoms remained brief and tolerable after flecainide was stopped. The response to flecainide was puzzling at first because it was expected that junctional extrasystoles due to automaticity would respond well to flecainide. Because conduction slowing due to sodium channel blockade can readily facilitate reentry under the appropriate conditions, clinical suspicion now turned to a reentrant mechanism as the source of the JPBs.

A year and a half later, the patient had marked worsening of symptoms, now having 3:1 and 2:1 AV block that occurred frequently for prolonged durations virtually all day long as verified by ambulatory monitoring. Twelve-lead ECG during a clinic follow-up visit now showed 2:1 AV block but still showed normal PR intervals and QRS duration of the conducted beats (see Supplemental Figure 4, panel A). Although she still wished to avoid a pacemaker, the patient was now willing to accept it if it was the only treatment that would relieve her symptoms. A repeat electrophysiological study was proposed to search for a possible reentrant mechanism suitable for ablation. Barring the successful elimination of such a mechanism, a pacemaker implant would ensue.

Repeat electrophysiological study was performed using a decapolar catheter (2-5-2 mm spacing) on the right ventricular septum to assess His-bundle activity. Baseline rhythm on surface ECG was sinus with frequent nonconducted P waves mostly with 3:1 block (see Supplemental Figure 4, panel B),
but occasionally with 2:1 block. Frequent JPBs were noted with intact anterograde conduction and retrograde block (see Supplemental Figure 4, panel C). Baseline AH and HV intervals of conducted sinus beats were normal. His-bundle recordings of the JPBs showed anterograde activation of the His bundle that were similar to those seen during conducted sinus beats (Figure 2A). Despite careful adjustment of the His catheter to record as proximal as possible at high gain, no His extrasystoles were noted preceding the blocked sinus beats (Figure 2B). Occasional retrograde premature P waves were seen without anterograde conduction. No VA conduction was appreciated at baseline during ventricular pacing. Although the baseline rhythm in sinus was mostly 3:2 AV conduction, atrial pacing at a CL of 650 ms generated 2:1 AV conduction (see Supplemental Figure 5) that persisted during pacing down to a CL of 500 ms. The block was always above any recordable His-bundle potential. Of note, a JPB or blocked sinus beat only occurred after a normally conducted atrial beat suggesting HPS or ventricular participation in reentry into the AV node. Such a pathway, if present, could be targeted at areas where ventricular myocardium abuts AV nodal tissue, such as the base of the ventricles at the tricuspid or mitral annulus as well as subaortic valve regions.

At this point, ablation was pursued guided by 3-dimensional mapping and fluoroscopic guidance using an irrigated contact-force ablation catheter. Because of the concealed nature of the potential pathway, activation mapping was not possible. Using the 3-dimensional mapping system, location points were acquired to delineate the anatomy of the His bundle, and high-gain voltage mapping was performed to define the septal tricuspid annulus and to search for any pathway potential. None were found. Figure 3 shows the map and locations of ablation lesion delivery. With a lesion delivered 2–5 mm inferior to the His bundle, sinus rhythm suddenly transitioned to 1:1 AV conduction without blocked sinus beats or JPBs (Figure 4). Sinus rhythm then continued without occurrence of further JPBs or AV block (see Supplemental Figure 6, panel A). Postablation atrial pacing demonstrated 1:1 AV conduction occurring down to a CL of 520 ms. AV nodal block would occur at shorter CLs in a typical Wenckebach pattern. With isoproterenol infusion at 1 µg/min, 1:1 AV conduction persisted down to a pacing CL of 410 ms. Twelve-lead ECG recorded the next day continued to showed sinus rhythm with normal AV conduction (see Supplemental Figure 6, panel B). At 2-month follow up, the patient remained asymptomatic and continued to show normal sinus rhythm.

Discussion
Concealed junctional extrasystoles causing the subsequent sinus beats to block in the AV junction was first postulated by Langendorf and Mehlmam in 1947 based on surface ECG recordings in 1 patient. They observed the presence of JPBs and blocked atrial premature beats suggestive of a junctional origin. They also noted isolated sinus beats with PR prolongation or actual AV block. Because these phenomena all occurred in temporal proximity in their patient, the investigators conjectured that JPBs, blocking in both the anterograde and retrograde directions, were the cause of the sinus beats blocking in the AV junction. When His-bundle recordings in humans became practicable, Rosen and colleagues reported in 1970 a case of His extrasystoles that triggered AV block of the following sinus beats. Using the His-bundle recording, they showed that these His extrasystoles can block anterogradely in the more distal HPS as well as retrogradely in the AV junction and were thus unappreciated on the surface ECG. Nevertheless, these concealed His extrasystoles were the cause of intermittent AV block seen in sinus rhythm. Since their report in 1970, several other reports have shown similar findings in which concealed His extrasystoles can cause intermittent AV block of the subsequent sinus beat. Interestingly, all reports noted only single concealed His extrasystoles following sinus beats. Repetitive concealed His extrasystoles were not noted in any of those reports. We saw the same phenomena during
clinical monitoring and the electrophysiology procedure in our patient. These observations suggest that the occurrences of His extrasystoles in prior reports were also dependent on ventricular activation of the preceding sinus beat.

Several features of this case suggest that our patient’s symptomatic AV block was due to concealed JPBs. Baseline electrophysiological study showed normal AV conduction properties even when provoked with procainamide infusion. Thus, notable conduction abnormalities of the AV conduction system were excluded. 1:2 conduction via dual AV nodal pathways was also excluded after ablation of the slow pathway. Unlike prior reports of His extrasystoles, even with careful recording of the proximal His bundle, His extrasystoles were not apparent preceding the blocked beats. The patient did manifest anterogradely conducted JPBs, some with retrograde atrial activation, as well as nonconducted atrial premature beats that had characteristics consistent with retrogradely propagated JPBs. These observations indicated that the JPBs were likely the cause of her AV block and that they occurred above the His bundle, likely in the more distal AV nodal regions as the slow pathway regions had been ablated. Although a spontaneous junctional rhythm can occasionally compete with sinus beats, it usually manifests as a competing rhythm rather than extrasystoles and typically is suppressed with flecainide treatment. Flecainide has been shown to be an effective treatment in reducing junctional ectopy and has been used for the treatment of junctional ectopic tachycardia. Mechanisms of triggered activity are also not known to worsen with sodium channel blockade, whereas reentry can be facilitated by slowing of conduction. The observed worsening of AV block during treatment with flecainide in our patient suggests that slowing of conduction played a role in the increasing frequency of JPBs and AV block. Other observations also support a reentrant mechanism.

**Figure 4** Onset of first RF energy delivery at the successful site. The patient was experiencing bigeminal retrogradely conducted junctional premature beats at the time of RF onset (at beginning of panel). ECG lead aVF is gained up to show the retrograde P waves of the premature beats. RF energy was started at 15 W. At 10 seconds into delivery, the power was increased to 20 W. At 12 seconds, 1:1 AV conduction of sinus beats commenced. The power was then ramped up to 25 W at 17 seconds and continued for a total of 25 seconds of delivery. RF = radiofrequency.

**Figure 5** Location of the concealed ventricular nodal tract. Based on the site of successful ablation, the ventricular nodal tract likely connects from the base of the ventricular septum just below the His bundle across the tricuspid annulus into the distal AV node. The tricuspid valve leaflet in this figure is cut away to show the ventricular portion of the pathway. AVN = atrioventricular node; CS = coronary sinus; CT = crista terminalis; FO = fossa ovalis; RV = right ventricle; TV = tricuspid valve.
Additionally, the worsening of AV block during the last procedure going from 3:1 AV block to 2:1 AV block when the atria are paced at a CL of 650 and 500 ms is also consistent with facilitation of reentry at faster rates. It is unlikely that such a worsening was due to normal AV nodal Wenckebach phenomenon as 1:1 conduction through the AV node was much better after the ablation. The distal transitional regions of the AV node (NH regions) have refractory properties similar to those of the His bundle. Thus, refractoriness of these sites can shorten at faster rates and facilitate conduction of ventricular impulses into this region of the distal AV node. Slowing of conduction in the proposed accessory pathway during faster pacing is considered unlikely as ventricular activation is actually slower during 2:1 AV block as compared to 3:1 AV block. The observation that a blocked sinus beat or a JPB was always preceded by a conducted sinus beat also suggests participation of ventricular or at least hisian activation in generating the following AV block. Thus, our ablation attempts were aimed at interrupting a potential ventricular nodal pathway. A fascicular nodal pathway is also possible and cannot be excluded based on our observations. However, such a pathway would more likely manifest a pathway potential, which we did not note despite carefully mapping the perihisian region. The location of the successful ablation lesion would indicate that this pathway was located just inferior to the His bundle and thus likely connected across the annulus to the distal AV node. Figure 5 shows a graphical representation of the proposed pathway connecting from the ventricle to the distal AV nodal region and traversing the annulus just below the His bundle.

Our case report would fit with a model in which sinus beats (or atrial paced beats) block in the AV junction due to reentry (of the previous beat) from the ventricle into the distal node over a concealed ventricular nodal pathway. Despite our best attempts, we were unable to record a His-like potential associated with the blocked sinus beat even at high gains. Thus, there was never penetration of the concealed junctional extrasystole into the His bundle except when it conducted to the ventricle. Such a ventricular nodal reentrant beat would explain the dependence of the junctional extrasystole and the AV block on a preceding ventricular activation. As a variation on the theme, a concealed ventricular nodal pathway has been reported to cause narrow QRS tachycardia with VA block. Similar to the reports of concealed His extrasystoles noted earlier, our patient never manifested any evidence of anterograde conduction via the ventricular nodal pathway.

These observations raise an interesting paradigm. Perhaps many of the reported His extrasystoles causing AV block despite apparently normal-appearing AV conduction properties do not originate as ectopic foci in the His bundle but are reentrant beats using ventricular nodal pathways. Although His-bundle depolarization can be readily recorded, the source of such activation may come from the nodal regions or the transitional NH regions. Thus, those observed “His extrasystoles” may also be a result of ventricular nodal reentry through concealed ventricular nodal tracts. This model of reentry can also include pathways that connect into the His bundle or its branches. In that scenario, His activation would have a distinctly different activation than that of atrial conducted beats. These pathways may be present at birth. However, with changing conduction or refractory properties accompanying aging, scarring, or drug effects, reentry can be facilitated. Such a paradigm would raise the possibility of ablating these pathways without injuring the normal conduction system, as was achieved in our patient. To our knowledge, however, our patient is the first reported case of symptomatic second-degree AV block unrelated to vagotonia treated with conventional cardiac catheter ablation. This may be an under-recognized phenomenon and should be suspected in those with paroxysmal AV block and otherwise normal-appearing conduction, especially in the presence of junctional ectopy.

Conclusion
Our report demonstrated that JPBs when concealed on the surface ECG can appear to cause AV block of sinus beats. When these occur at high frequency, such as 2:1 or 3:1 block for any prolonged duration, a patient can become highly symptomatic and debilitated. These JPBs can be ablated by targeting regions of the ventricle near the distal AV node without causing undue harm to the normal conduction system, thus avoiding the need to implant a pacemaker. This outcome is likely due to ablation of a ventricular nodal conducting pathway that engenders reentry into the AV junction, interfering with otherwise normal conduction.

Acknowledgement
The authors would like to thank Tania Saliba who graciously provided the artwork seen in Figure 5.

Appendix
Supplementary data
Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.hrthm.2017.08.015.

References