

# Ventricular premature pacing to reveal slow pathway conduction: A case of dual ventricular response with ventriculoatrial block



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## Introduction

Dual atrioventricular (AV) nodal physiology is a common finding during electrophysiologic study forming the substrate for AV nodal reentrant tachycardia. In some circumstances, a sinus beat may result in dual ventricular response (DVR) owing to conduction via fast pathway (FP) and slow pathway (SP). This phenomenon, known as “double fire” or “dual AV nodal nonreentrant tachycardia,”<sup>1–3</sup> can sometimes mimic atrial fibrillation owing to irregular ventricular activation.<sup>4</sup>

Several conditions are required for DVR to occur: (1) there must be a significantly long SP conduction; (2) the effective refractory periods (ERP) of the AV node distal common pathway and the His-Purkinje system (HPS) must be shorter than the difference between the conduction times of the FP and SP; (3) conduction through the SP to the HPS necessitates that the subsequent sinus beat is sufficiently delayed; and (4) retrograde ventriculoatrial (VA) conduction is poor or absent.<sup>1</sup>

## Case report

A 39-year-old man was referred for electrophysiologic evaluation for recurrent palpitation. The patient was treated for atrial fibrillation twice by pulmonary vein isolation without improvement of his symptoms. A 12-lead surface electrocardiogram (ECG) of his clinical arrhythmia is shown in Figure 1A. There are irregular QRS complexes having a group-beating pattern. An enlargement of V<sub>1</sub> showing this pattern is illustrated in Figure 1B. The 3-beat repetitive pattern consists of a sinus P waves with normal PR followed by a “junctional beat,” likely due to conduction via the SP, followed by a second sinus P wave with a lengthened PR. This lengthening is likely due to retrograde concealment into the FP from the “junctional beat.” The possibility of conduction via an interme-

diate pathway cannot be excluded. This second sinus P wave is not accompanied by junctional beat.

The patient underwent an electrophysiologic study and a catheter ablation under conscious sedation. With the mild sedation, the irregular rhythm converted to regular sinus rhythm. Baseline intracardiac conduction intervals were not remarkable: sinus cycle length was 1115 ms, atrial-His (AH) interval was 126 ms, His-ventricular interval was 44 ms. Ventricular pacing revealed no VA conduction even on isoproterenol infusion. Since double ventricular response was highly suspected from the clinical ECGs, we tried to elicit long-conducting SP conduction with atrial extrastimuli and with incremental atrial pacing with and without isoproterenol infusion. However, the antegrade SP could not be demonstrated by any atrial drive train. No AH jump was demonstrable. ERP of the antegrade FP was 450 ms. The longest achievable AH interval during incremental atrial pacing just prior to AV nodal block was 300 ms. While one can debate whether this AH interval is a SP conducted beat or the “last gasp” of the FP prior to block, this AH interval was clearly not long enough to explain the patient’s clinical arrhythmia. In the absence of being able to demonstrate a long-conducting SP, other etiologies of the junctional beat noted on clinical ECGs would need to be considered more seriously. Those include triggered junctional extrasystoles and ventricular nodal or ventricular Hisian reentry.

We thus devised an alternative pacing method to elicit SP conduction, as illustrated in Figure 2. A ventricular premature beat (V2) was delivered from the right ventricular apex with coupling interval of 700 ms after baseline pacing from the high right atrium at 900 ms (A1). This was followed by an atrial paced beat (A2) from the high right atrium. This pattern of pacing consistently revealed a long SP conduction of A2. Details are noted in the figure legend. Retrograde conduction of V2 blocked in both the SP and FP, as there was no VA conduction and block in the HPS is unlikely in the absence of HPS disease. The retrograde concealment in the FP prevented A2 from conducting down the antegrade FP. However, owing to the long SP conduction, the distal portion of the SP had

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## KEY TEACHING POINTS

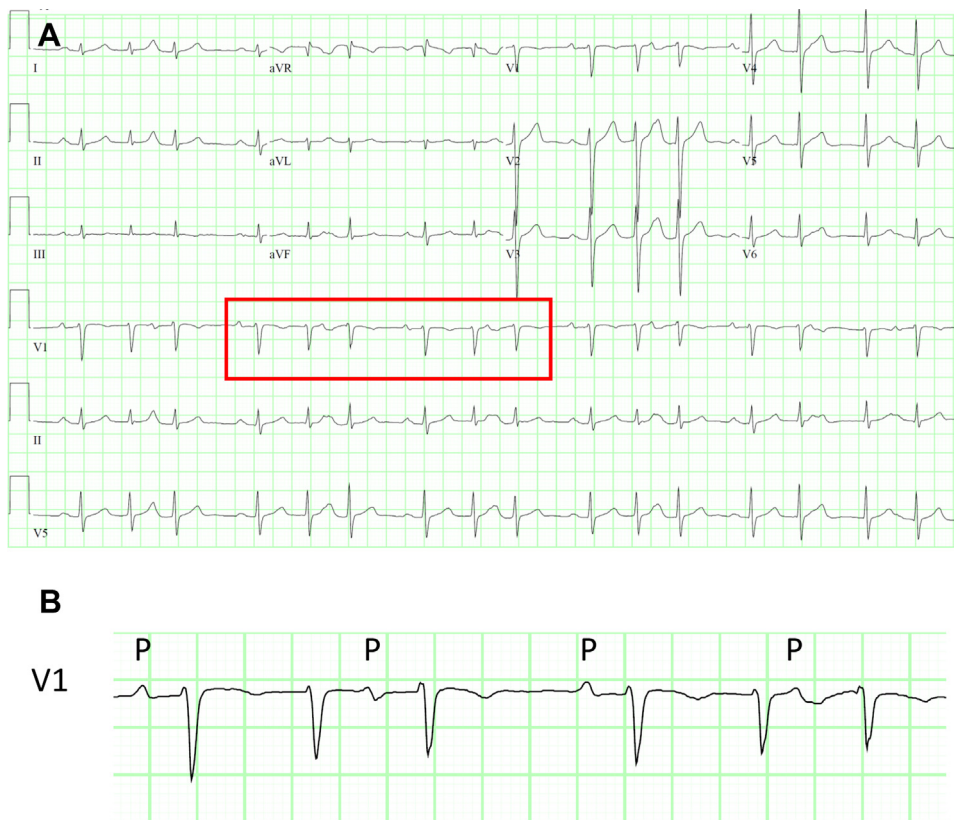
- Dual ventricular response mimics atrial fibrillation, and careful electrocardiogram reading is needed.
- Ventricular premature pacing maneuvers can be useful in revealing antegrade slow pathway consistently.
- Some retrograde fast pathway and slow pathway penetration is likely to exist even when ventriculoatrial conduction is not seen.

recovered from the retrograde concealment of V2, allowing A2 to conduct via the SP to generate V3. Alternative etiologies of V3 mentioned above are excluded, as withholding A2 invariably prevented V3.

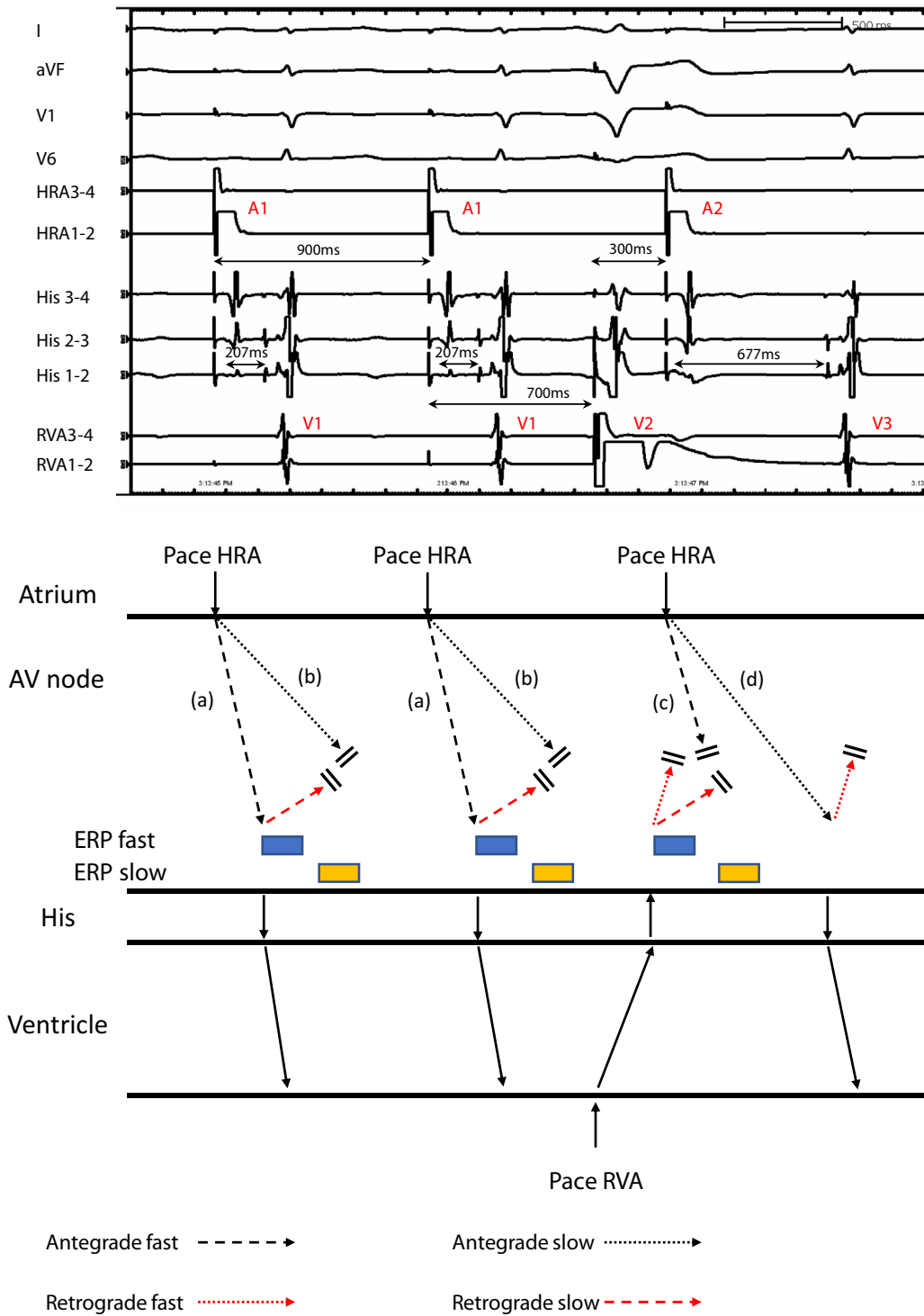
The above demonstration of a long SP conduction was then used as an endpoint for SP ablation. A standard SP ablation was performed during atrial overdrive pacing. Following the ablation, repeating this pacing maneuver consistently showed that A2 blocked in the AV node, verifying absence of SP conduction. Clinical follow-up demonstrated no recurrence of the frequent runs of his arrhythmia.

## Discussion

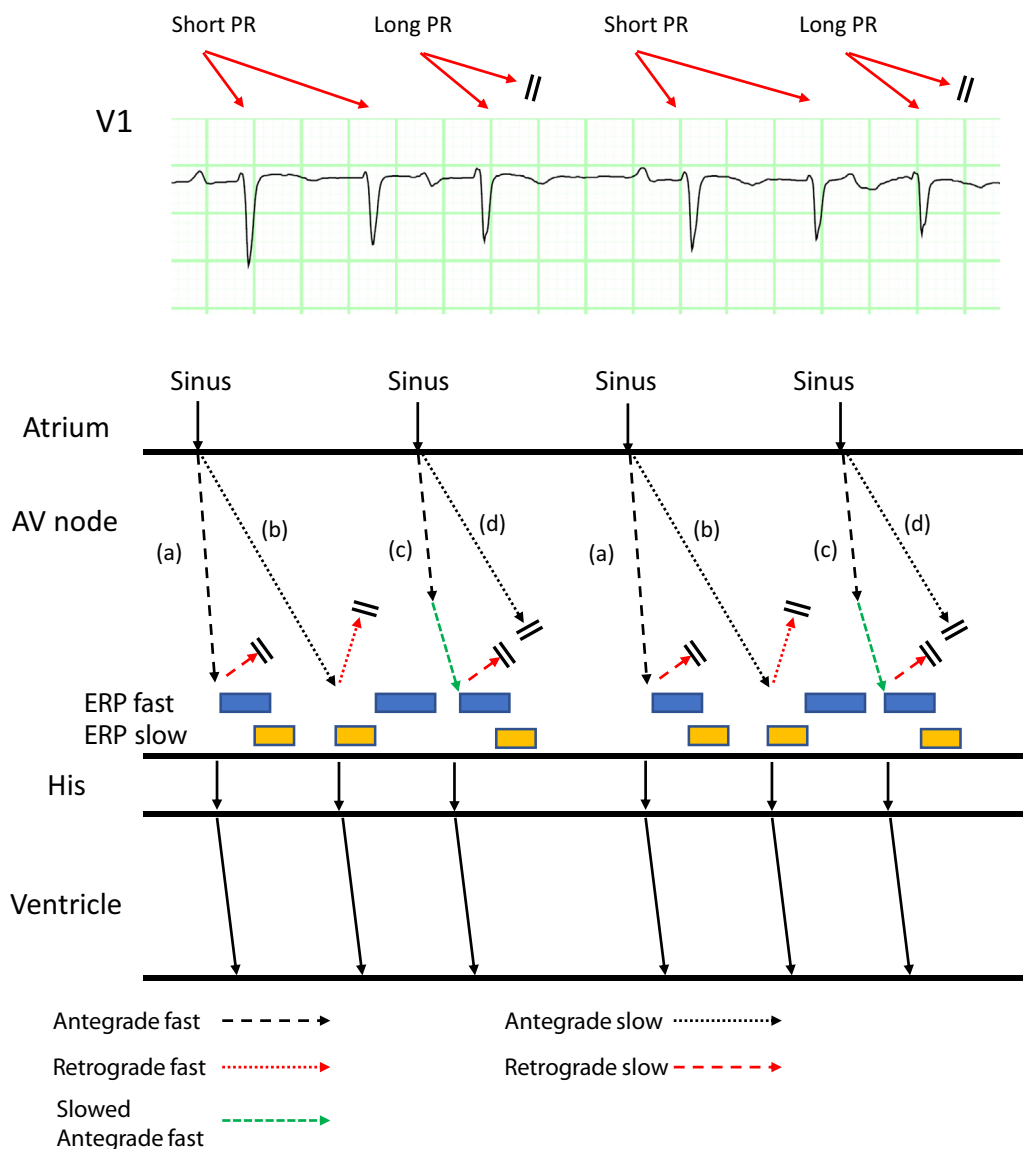
This case report represents an unusual manifestation of DVR where the patient was symptomatic from a repetitive pattern of SP conduction and block during sinus rhythm generating an irregular AV conduction pattern. [Figure 3](#) is a ladder diagram that illustrates the conduction pattern seen on ECG shown in [Figure 1](#). This repetitive pattern starts with a sinus P wave that conducts normally via the FP. As the FP impulse reaches the compact node, it also penetrates and blocks in the SP retrogradely ([Figure 3 \[a\]](#)). Both SP and FP are known to block in the retrograde direction, as the patient manifested no VA conduction with ventricular pacing at any cycle length below sinus rate. It is likely that retrograde block in the SP occurs rather distally to allow anterograde conduction of the SP beat. Thus, the same P wave that conducted normally via the FP also conducts down a rather slow SP. By the time this anterograde SP wavefront arrives at the distal SP, the path has recovered from the preceding retrograde penetration by the FP wavefront. Thus, the SP impulse was able to conduct to the His bundle, generating the double response ([Figure 3 \[b\]](#)). However, the SP impulse also retrogradely penetrates the FP, impeding the next sinus beat's conduction via the FP and causing PR prolongation by slowing antegrade FP conduction ([Figure 3 \[c\]](#)).



**Figure 1** **A:** Twelve-lead surface electrocardiogram of the apparent clinical arrhythmia. Every other sinus P wave was associated with 2 subsequent QRS complexes. **B:** Magnified view of lead V<sub>1</sub>. The 3-beat repetitive pattern consists of sinus P waves with normal PR followed by a junctional beat. Second sinus P waves were conducted with longer PR, which were not followed by junctional beats.



**Figure 2** During atrial pacing at 900 ms cycle length (A1), a ventricular premature beat is delivered 700 ms after the last atrial stimulation (V2), followed by an atrial stimulation (A2). A2 then invariably conducted via a long slow pathway (SP) to the ventricle (V3). Upper panel: Surface electrocardiogram and intracardiac electrograms. Lower panel: Laddergram. Black and red arrows with large and small dotted lines depict each pathway (antegrade fast pathway [FP] and SP, retrograde FP and SP). Blue bar shows effective refractory periods (ERPs) of FP. Yellow bars show ERPs of SP. Conduction via the antegrade FP by a stimulus from high right atrium (HRA) (a). Conduction via the antegrade SP collided with (or interfered by) conduction via the retrograde SP distally (b). Conduction via the retrograde FP after V2 collided with conduction from the antegrade FP by a stimulus from HRA (A2) (c). Since the conduction via the retrograde SP by V2 terminated distally, ERP of the SP ended before the SP conduction of A2 arrived at the distal site of retrograde block, thus allowing A2 to propagate to the His via the antegrade SP (d).



**Figure 3** Upper panel: Magnification of lead V<sub>1</sub> as shown Figure 1. Sinus P waves with short PR seemed to be accompanied by 2 subsequent QRS complexes. On the other hand, sinus P waves with long PR seemed to be accompanied by only 1 subsequent QRS complex. Lower panel: Ladder diagram depicting the conduction pattern in the upper panel. As antegrade fast pathway (FP) impulse reaches the compact node, it also penetrates the slow pathway (SP) retrogradely but blocks distally in the SP (a). Impulse via the antegrade SP can conduct to the His bundle, generating double response, since the SP has recovered from preceding retrograde penetration by the FP (b). The SP impulse also retrogradely penetrates the FP impeding the next sinus beat’s conduction via the FP and causing PR prolongation (green dotted line) (c). Owing to delay of conduction through the antegrade FP, retrograde SP penetration is also delayed, retarding its recovery and resulting in antegrade block of the SP impulse (d). Black and red arrows with large and small dotted lines depict each pathway (antegrade FP and SP, retrograde FP and SP). Green dotted arrows depict slowed FP conduction. Blue bar shows effective refractory period (ERP) of FP. Yellow bar shows ERP of SP.

Because of the delay of antegrade FP conduction, its retrograde penetration into the distal SP is also delayed, thus extending the distal SP refractoriness, resulting in conduction block of the corresponding antegrade SP wavefront at the distal site (Figure 3 [d]). The presence of an intermediate pathway explaining the PR prolongation of beats (c) in Figure 3 cannot be excluded, although we did not see evidence of that before or after ablation.

Another unique feature of this case was the inability to demonstrate dual AV conduction using standard atrial programmed premature stimulation. Typically, SP conduction

can be demonstrated in cases of DVR. However, in this patient, despite having clinically very symptomatic repetitive ventricular responses, we were unable to definitively conclude that a long SP conduction was present using standard premature atrial pacing protocols. One can speculate as to why that occurred. It is possible that SP refractoriness increased with sedation, preventing the SP from delivering the late double response. Another possibility is that FP impulse invasion retrogradely into the SP was delayed, thus generating later recovery from concealment and interfering with SP conduction. Without demonstrating that such SP

was present, other explanations may also be postulated for his clinical arrhythmia. As the patient had VA block during ventricular pacing, we took advantage of this feature to unmask SP conduction using the pacing protocol shown in [Figure 2](#). A ventricular premature beat would penetrate the AV node prematurely, causing block in the FP and SP distally ([Figure 2 \[c\]](#)). A2 could not conduct down the FP owing to retrograde concealment into the FP by V2. The SP, having its distal refractoriness peeled back by V2, has time to recover from the distal retrograde block and allow the atrial paced beat to conduct via the SP ([Figure 2 \[d\]](#)). V3 in [Figure 2](#) is conclusively due to SP conduction of A2, as withholding A2 during testing also prevented V3. With a reliable demonstration of a long anterograde SP conduction, there was a good endpoint to judge the success of SP ablation.

Of note, because there was no retrograde FP conduction, acceleration of junctional rhythm during radiofrequency ablation of the SP would not generate retrograde atrial activation, eliminating 1 of the means for monitoring FP conduction during ablation. Thus, atrial pacing to overdrive junctional

rhythm was necessary during radiofrequency application to monitor the adequacy of anterograde FP conduction.

## Conclusion

We report a case of DVR with an irregular ventricular conduction pattern where SP conduction was unmasked with a special pacing protocol using premature ventricular pacing. This approach allowed divulgence of a long-conducting SP where standard programmed electrical stimulation during electrophysiologic study did not reveal such a pathway.

## References

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