

A long-distance relationship: a case report of extreme Mobitz type I

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Introduction

The PR interval is a consistently measured metric on electrocardiograms (ECG), used to assess the timing of conduction from sinus node activation to earliest ventricular activation. The PR interval is composed of transmyocardial conduction across the right atrium, followed by conduction through the atrioventricular (AV) node, followed by conduction down the His-Purkinje system. Prolongation of the PR interval is defined as longer than 200 ms, and the delay in conduction can occur at the level of the atrium, the AV node, the His-Purkinje system, or any combination of the above. As conduction system disease progresses, atrioventricular block may occur intermittently. The pattern of atrioventricular delay or block is classified as first-degree, second-degree Mobitz type I, second-degree Mobitz type II, and third-degree, with the characteristics of each pattern yielding important clues as to the location of the conduction delay or block.¹⁻⁴ AV node conduction impairment can vary dramatically from patient to patient, and even to varying degrees within the same patient, at different times, with some patients demonstrating markedly prolonged PR intervals. Advanced infranodal conduction disease usually presents with a Mobitz type II pattern, where conducted beats typically demonstrate normal or mildly prolonged PR intervals, which tend to remain relatively constant. On the other hand, advanced AV node disease usually presents with a Mobitz type I pattern, where conducted beats typically demonstrate prolonged PR intervals that tend to vary in a predictable pattern of incremental prolongation until a nonconducted P wave resets the cycle.^{2,3} Third-degree heart block represents the completion of conduction block, where atrial and ventricular activity are electrically independent without electrical communication between them.⁴ Of interest, even first-degree AV "block" with preserved AV conduc-

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KEY TEACHING POINTS

- During rhythm analysis, it is important to correctly identify P waves and QRS associations, especially in the context of AV node disease.
- Clinical ECG interpretation should be done in the context of previous ECGs and telemetry data.
- Identification of AV node disease severity influences treatment plan.

tion is associated with adverse outcomes such as atrial fibrillation, pacemaker implantation, pseudo-pacemaker syndrome, heart failure, and mortality.^{5,6} We present an interesting case of extreme Mobitz type I with an unusual atrioventricular relationship.

Case report

A 60-year-old man with tetralogy of Fallot had serial repair surgeries over the years, including a Blalock-Taussig shunt and a subsequent tetralogy of Fallot repair with pulmonic valve replacement, tricuspid valvuloplasty with chordal reattachment, and closure of a cleft in the septal tricuspid valve leaflet. He also had a history of dual-chamber pacemaker implantation for second-degree AV block and right bundle branch block. Because of the recurrent tricuspid valve dysfunction, he underwent repeated tricuspid valve surgery with intraoperative explantation of his pacemaker leads. Postoperatively, the surgical temporary epicardial pacing wires were not initially used because of adequate native conduction and reasonable ventricular rates. Postoperative ECGs showed the characteristic native QRS morphology seen in repaired tetralogy of Fallot patients, including a wide right bundle branch block (QRS duration 206 ms), notching in the terminal portion of the QRS, and small epsilon waves seen in some leads. More importantly, various forms of asymptomatic Mobitz type I AV block were seen on serial ECGs (Figure 1). When 2:1 AV conduction was seen (Figure 2), temporary pacing was implemented. He

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Figure 1 Immediate postoperative electrocardiogram showing Mobitz type 1 atrioventricular block.

subsequently underwent biventricular pacemaker implantation to achieve AV and VV synchrony, in the context of concurrent LV dysfunction and an anticipated high burden of ventricular pacing.

On subsequent review of the postoperative ECGs, the AV relationships were more complex than initially appreciated. When long-cycle Mobitz type I pattern conduction was seen, P waves were noted to become "buried" within QRS complexes at times, ruling out a direct relationship between them. When P waves were tracked forward and backward from the longer R-R interval gaps that indicated a blocked P wave and recognizing that shorter R-R intervals did not

represent junctional beats, it became clear that PR relationships were markedly prolonged. In the example shown in Figure 1, the true PR intervals range from 640 to 1170 ms, with PR intervals overlapping each other owing to PR exceeding PP intervals (Figure 3A). When all ECGs were reviewed, the maximum PR interval observed was over 1300 ms, the shortest PR interval was 640 ms, and no junctional escape beats were seen on any ECG. Only in this context of recognizing the extremely impaired nature of AV conduction could the true PR relationship in the 2:1 AV conduction example be deciphered (Figure 2). It is the "isolated" P wave that conducts to the subsequent QRS complex with a PR



Figure 2 Postoperative day 2 electrocardiogram showing 2:1 atrioventricular block.



Figure 3 A: Actual PR relationships during Mobitz type 1 atrioventricular (AV) conduction, including "overlapping" PR intervals, with PR interval varying between 640 ms and 1170 ms. B: Actual PR relationships during 2:1 AV conduction, with PR interval of approximately 1070 ms and the P waves immediately preceding the QRS complexes being the ones that block.

interval of 1070 ms, while the P wave that immediately precedes each QRS complex is the one that actually blocks (Figure 3B).

Discussion

In the presented case, cursory ECG assessment led the intensive care unit clinical team to conclude that AV conduction in the postoperative setting followed a simple Mobitz type I pattern with typical PR intervals. Therefore, surgical temporary pacing wires were not used until the patient developed 2:1 AV conduction with more marked bradycardia. With careful ECG analysis, assigning each QRS to the correct P wave, the severity of AV node dysfunction could be properly assessed and managed. The extreme prolongation of the PR interval in this case suggests that the location of greatest AV conduction slowing is in the AV node, despite evidence of His-Purkinje disease in the form of right bundle branch block that which is related to the patient's congenital heart disease and history of surgical repair. An electrophysiology study would have been of academic interest, as it might have demonstrated a prolonged HV interval contributing to PR prolongation, but given the extreme nature of this patient's AV conduction slowing and the much greater range of clinically observed AH intervals versus HV intervals, it remains likely that AV node disease explains at least the majority of the observed PR relationships. Although the risk of sudden and severe bradycardia was likely low in the context of AV node disease, permanent pacemaker reimplantation was appropriate to restore proper AV synchrony, prevent excessive bradycardia when 2:1 AV block occurred, and preempt the likely further progression of AV conduction slowing in the future. Careful pacemaker programming is needed to preserve appropriate atrial-sensed, ventricular-paced behavior with physiologic AV intervals, especially during times of faster sinus rates when native AV conduction will occur with P waves falling in and around the preceding QRS complexes.

Conclusion

AV conduction patterns can become complex in the setting of advanced AV node disease, and more dramatically so in the context of congenital heart disease and valve surgery. Correct associations between specific P waves and QRS complexes should be ascertained in the context of synthesizing all available rhythm data, to properly appreciate AV conduction disease severity. **Funding Sources:** Publication of this article was funded in part by the Temple University Libraries Open Access Publishing Fund.

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