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CASE REPORT

First-Degree Atrioventricular Block and Unexpected Symptoms: A Case Study and Diagnostic Journey

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ABSTRACT

Marked first-degree atrioventricular (AV) block is traditionally considered a benign finding, particularly in young athletes. However, an excessively prolonged PR interval can precipitate a significant hemodynamic compromise known as "pseudo-pacemaker syndrome". This study presents the case of a 23-year-old male avid runner with a two-year history of unexplained exertional dyspnea. Although his condition was initially attributed to vagal tone, a resting electrocardiogram revealed a PR interval of 400 ms. Treadmill exercise testing demonstrated a pathological failure of the PR interval to shorten during tachycardia. Crucially, while resting echocardiography was unremarkable, exercise echocardiography revealed the emergence of significant diastolic mitral regurgitation and fusion of mitral inflow E and A waves, confirming severe AV dyssynchrony. An electrophysiology study confirmed that the block was located within the AV node. Consequently, the patient was scheduled for permanent pacemaker implantation. This case highlights that marked first-degree AV block is not invariably benign. Comprehensive evaluation with exercise echocardiography is therefore essential to identify functional hemodynamic obstruction in symptomatic patients, and pacing therapy to restore physiologic AV timing is justified.

Keywords: atrioventricular dyssynchrony, exercise echocardiography, first-degree atrioventricular block, pseudo-pacemaker syndrome

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INTRODUCTION

First-degree atrioventricular (AV) block is traditionally viewed as a benign conduction delay, defined by a PR interval exceeding 0.20 seconds. However, marked prolongation of the PR interval can lead to significant AV dyssynchrony, a rare phenomenon commonly known as "pseudo-pacemaker syndrome" (PPMS). In this condition, atrial contraction occurs against closed AV valves, causing elevated atrial pressures and reduced cardiac output. Consequently, patients may experience unexpected symptoms typically associated with heart failure, including palpitations, dyspnea, and syncope, even in the absence of overt structural heart disease. Because PPMS is rare, diagnosis relies heavily on correlating symptoms with electrocardiographic findings after ruling out other etiologies. This case report highlights the diagnostic challenges of marked first-degree AV block and the necessity of recognizing PPMS in guiding appropriate therapeutic intervention.

CASE REPORT

Two years prior to this case, a 23-year-old male avid runner presented with a history of exertional dyspnea and an inability to achieve his usual running distance. At that time, an electrocardiogram (ECG) performed during an annual check-up revealed sinus rhythm at 62 bpm with 4:3 and 5:4 Wenckebach AV conduction and a prolonged PR interval of 360 ms in the first conducted beat **(Figure 1A)**. No further management was initiated at that time.

Two years later, a follow-up annual check-up ECG demonstrated sinus bradycardia at 57 bpm with 1:1 AV conduction, but with a further prolonged PR interval of 400 ms **(Figure 1B)**. Given the patient's persistent exercise intolerance, a cardiac investigation was initiated.

Due to his age and history of athletic conditioning, heightened vagal tone was initially suspected; therefore, treadmill exercise stress testing was performed. Although the patient was able to exercise through Stage 3 of the Bruce protocol, physiologic shortening of the PR interval was not observed **(Figure 1C)**, and the test was eventually terminated due to dyspnea.

The failure of the PR interval to shorten during exertion suggested that his exercise intolerance resulted from AV dyssynchrony or PPMS. To further evaluate hemodynamics, resting and exercise stress echocardiography were performed. While resting echocardiography showed no diastolic mitral regurgitation (MR) **(Figure 2A)**, significant diastolic MR became evident during exercise **(Figure 2B)**. Furthermore, the normal mitral valve inflow pattern **(Figure 2C)** evolved into a fusion of the E and A waves during exercise **(Figure 2D)**. These echocardiographic findings were highly suggestive of hemodynamic compromise consistent with PPMS.

To confirm the level of the block, the patient underwent an electrophysiology study. The baseline rhythm showed first-degree AV block with a PR interval of 335 ms, an Atrio-Hisian interval of 285 ms, and an His-Ventricular interval of 50 ms **(Figure 3A)**. Atrial pacing with a basic cycle length of 800 ms and extrastimulus demonstrated an AV nodal conduction block at 460 ms **(Figure 3B)**. These results confirmed that the level of the block was within the AV node.

The combination of clinical manifestations and diagnostic findings confirmed the diagnosis of PPMS. Consequently, the patient underwent permanent pacemaker implantation utilizing left bundle branch area pacing programmed to DDD mode. Following the procedure, the patient experienced a significant clinical improvement in exertional dyspnea; however, follow-up hemodynamic and electrophysiological investigations have not yet been performed to objectively quantify these post-implantation changes.

DISCUSSION

First-degree AV block is frequently dismissed as a benign electrocardiographic finding, particularly in young, athletic individuals where it is often attributed to high vagal tone. However, when the PR interval is markedly prolonged (typically > 0.30 s), it can precipitate a specific form of hemodynamic compromise known as PPMS.[1,2] This case study highlights the critical importance of correlating exertional symptoms with dynamic changes—or the absence of changes—in AV conduction, rather than relying solely on resting electrocardiography.

The pathophysiology of PPMS mirrors that of pacemaker syndrome, yet it occurs during sinus rhythm. The core mechanism is AV dyssynchrony caused by an extreme delay in ventricular activation. When atrial contraction occurs too early relative to the subsequent ventricular systole, or effectively "too late" relative to the preceding one, atrial systole occurs against closed AV valves.[3] This phenomenon results in the loss of the atrial kick, increased pulmonary capillary wedge pressure, and retrograde blood flow manifested as jugular "cannon A waves." In our patient, this mechanism was confirmed via exercise echocardiography, which demonstrated the emergence of significant diastolic MR and the fusion

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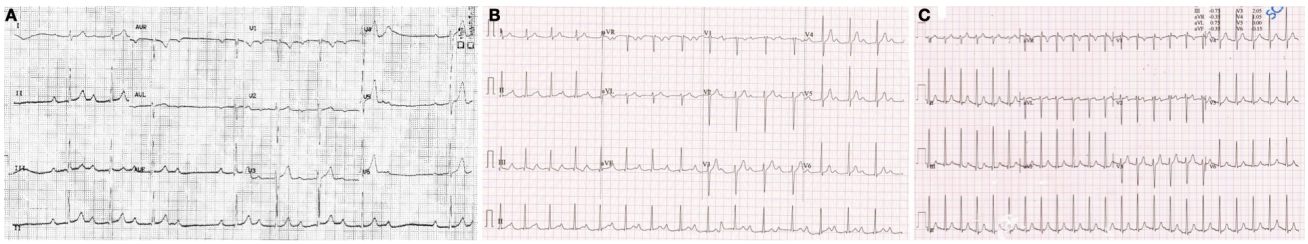


Figure 1 Serial electrocardiograms and response to exercise stress testing. (A) 12-lead electrocardiogram obtained 2 years prior to presentation showing sinus rhythm with Wenckebach AV conduction and a prolonged PR interval of 360 ms. (B) Follow-up electrocardiogram showing sinus bradycardia with 1:1 AV conduction and a PR interval of 400 ms. (C) Electrocardiogram obtained at peak exercise during the treadmill stress test showing no shortening of the PR interval.

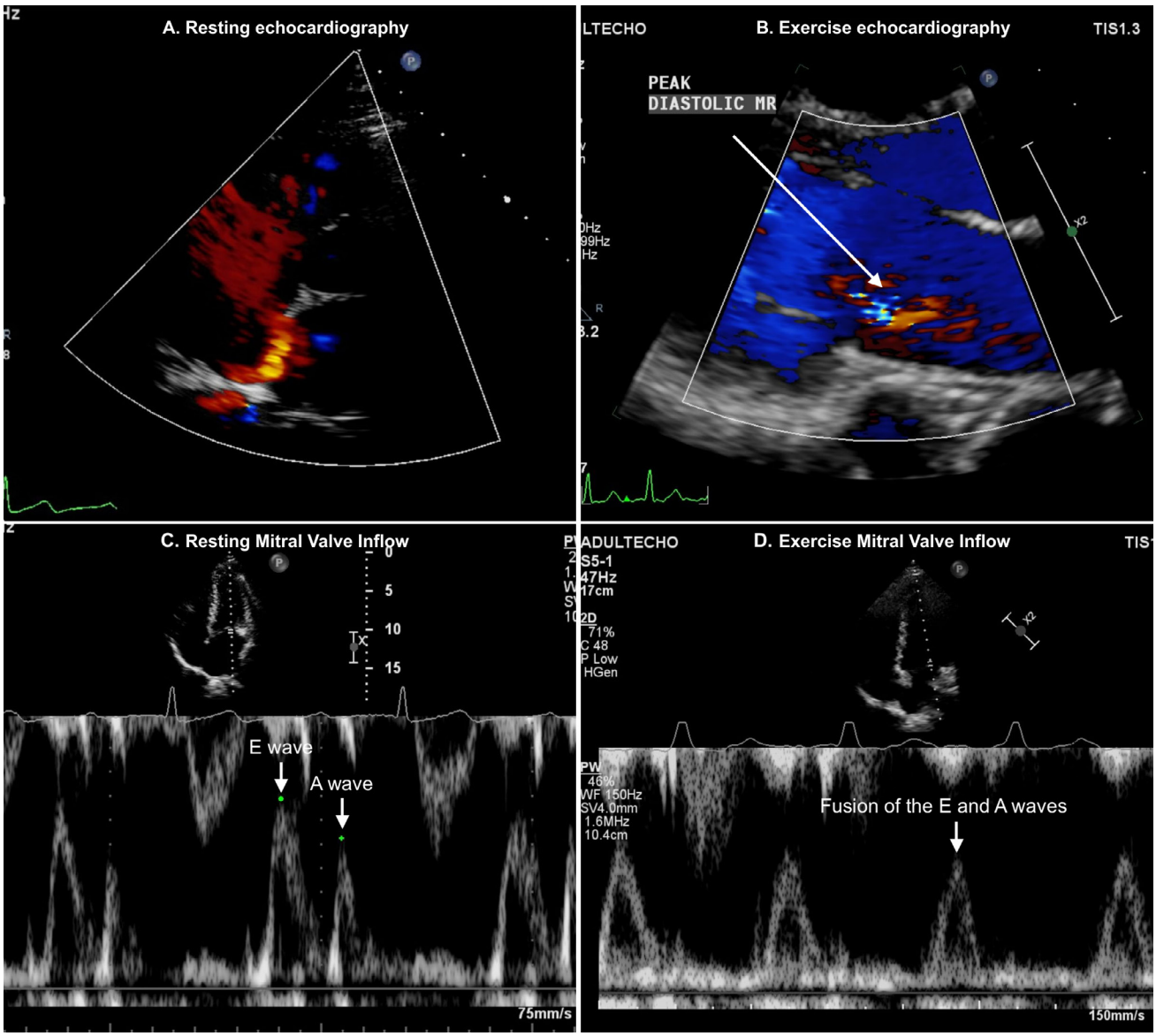


Figure 2 Comparison of resting and exercise echocardiography. (A) Resting echocardiogram showing the absence of diastolic MR. (B) Echocardiogram performed during exercise demonstrating the emergence of significant diastolic MR. (C) Normal mitral inflow pattern at rest with distinct E and A waves. (D) Fusion of the E and A waves during exercise, indicating atrioventricular dyssynchrony.

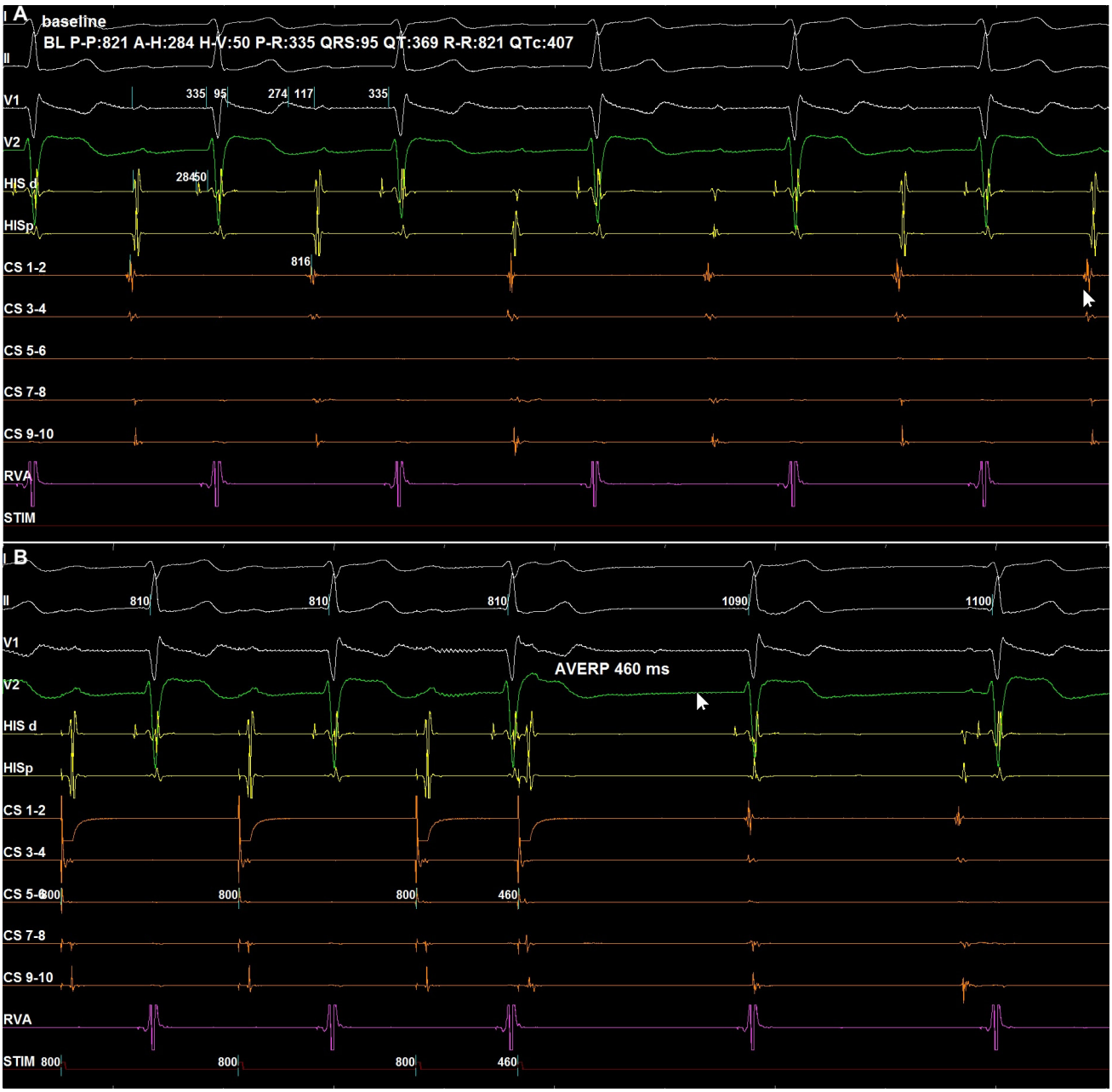


Figure 3 Electrophysiology study findings. (A) Baseline intracardiac electrograms showing first-degree AV block with a prolonged Atrio-Hisian interval of 285 ms and a normal His-Ventricular interval of 50 ms, localizing the conduction delay to the AV node. (B) Programmed atrial stimulation showing that pacing at a basic cycle length of 800 ms with an extrastimulus resulted in AV nodal conduction block at a coupling interval of 460 ms, which confirmed that the level of the block was located within the AV node.

of E and A waves during exertion, confirming that the prolonged PR interval was not merely electrical but hemodynamically obstructive.

A pivotal finding in this case was the failure of the PR interval to shorten during the exercise stress test. Physiologically, vagal withdrawal and sympathetic activation during exercise should accelerate AV nodal conduction. The paradoxical persistence of a prolonged PR interval at elevated heart rates leads to the encroachment of atrial systole upon the preceding ventricular systole, significantly compromising diastolic filling. As noted by Feliz et al., exercise testing is paramount in these evaluations, as it can unmask chronotropic incompetence of the AV node or reveal dual AV nodal physiology as the underlying etiology.[1,4] While the patient's electrophysiology study confirmed the block at the AV

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node level, the absence of rate-adaptive conduction suggested intrinsic nodal disease rather than vagal influence.

Therapeutic decision-making in PPMS involves balancing the risks of right ventricular (RV) pacing against the hemodynamic penalties of intrinsic AV block. Generally, preserving intrinsic conduction is preferred in order to avoid the ventricular dyssynchrony associated with RV pacing, which has been linked to increased risks of heart failure and atrial fibrillation in MOST and DAVID trials.[5,6] However, Aizawa et al. demonstrated that in patients with first-degree AV block, optimizing AV delay via pacing significantly improves stroke volume compared to preserving a pathologically long intrinsic PR interval.[7] Their study suggests that when the intrinsic PR interval is excessive, the benefit of restored AV synchrony outweighs the detriment of ventricular dyssynchrony, provided that left ventricular systolic function is preserved.

Current guidelines advise that pacemaker implantation for first-degree AV block should be restricted to cases where a clear correlation exists between symptoms and the AV delay.[8,9] In this case, the clear correlation between the patient's exertional dyspnea and the echocardiographic evidence of diastolic filling impairment justified the intervention. The goal of pacing here was not to prevent bradycardia, but to restore physiologic AV timing, thereby eliminating diastolic MR and optimizing cardiac output during exercise.

For this patient, the most appropriate definitive treatment is the implantation of a dual-chamber permanent pacemaker programmed to DDD mode. Given the patient's young age and history of athletic conditioning, optimizing the programmed sensed AV delay during exertion is a critical clinical concern. Therefore, the utilization of a dynamic AV delay algorithm (also known as rate-adaptive or rate-responsive AV delay) is highly appropriate. This algorithm is provided by all major pacemaker manufacturers under varying proprietary terminologies but shares common structural parameters and therapeutic goals. The primary benefit of dynamic AV delay is its ability to mimic the physiological response of the native AV node by automatically shortening the AV delay as the atrial rate increases.[10] This progressive shortening maintains optimal AV synchrony, prevents early closure of the mitral valve (A-wave truncation), and optimizes diastolic filling and stroke volume during sympathetic activation.[11] Crucially for an active individual, dynamically shortening the AV delay actively reduces the total atrial refractory period (TARP). By shrinking TARP, the pacemaker effectively raises the 2:1 block point, enabling 1:1 atrial tracking at significantly higher exertional heart rates before Wenckebach or 2:1 block behavior occurs, thereby preserving maximal exercise capacity and preventing sudden hemodynamic deterioration during intense physical activity.

This case underscores that "asymptomatic" first-degree AV block is a misnomer when the PR interval is markedly prolonged. Clinicians must therefore maintain a high index of suspicion for PPMS in patients with unexplained exercise intolerance, and a multimodal approach that utilizes exercise stress testing and echocardiography to demonstrate the functional consequences of the conduction delay is required in the diagnosis.

CONCLUSION

This case report illustrates that marked first-degree AV block is not invariably benign and thus requires a high index of suspicion for PPMS in patients with unexplained exercise intolerance. Subsequently, comprehensive evaluation with exercise echocardiography is crucial for identification of hemodynamic compromise, confirming that restoration of physiologic AV timing through permanent pacing is the definitive treatment for symptomatic relief.

Conflict of Interest

The author declares no conflicts of interest regarding this manuscript.

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Author Contributions

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Data curation: –
Formal analysis: –
Funding acquisition: –
Investigation: S.L.
Methodology: S.L.
Project administration: S.L.
Resources: S.L.
Software: –
Supervision: –
Validation: –

Visualization: –

Writing – original draft preparation: S.L.

Writing – review & editing: S.L.

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