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Exercise-Induced Complete Heart Block and Sinoatrial Exit Block in Baseline Bifascicular Block

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Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Male, 72-year-old
Final Diagnosis: Heart block
Symptoms: Chest discomfort • lightheadedness • near-syncope
Clinical Procedure: Pacemaker implantation
Specialty: Cardiology • Endocrinology and Metabolic

Objective: Rare disease

Background: Bifascicular block is typically asymptomatic and incidentally diagnosed, yet it may progress to advanced conduction disturbances. In patients with unexplained syncope and conduction abnormalities, exercise stress testing can diagnose transient conduction disease. While not routinely indicated for known atrioventricular block, stress testing may clarify etiology in patients without resting high-grade block. We present a case of syncope in a patient with bifascicular block, in whom exercise stress testing revealed complete atrioventricular block and sinoatrial exit block.

Case Report: A 72-year-old man with chronic right bundle branch block and left anterior fascicular block presented with recurrent exertional syncope. Baseline electrocardiogram revealed bifascicular block, prompting exercise stress testing. During transition from Stage 1 to Stage 2 of the Bruce protocol, he developed transient complete atrioventricular block and near-syncope, followed by Mobitz II atrioventricular block and repetitive sinoatrial Wenckebach block during recovery. Computed tomography coronary angiography showed no obstructive lesions. A dual-chamber permanent pacemaker was subsequently implanted. Follow-up showed minimal pacing, and resolution of symptoms.

Conclusions: This case highlights the diagnostic utility of exercise stress testing in patients with conduction abnormalities and unexplained syncope. The concurrent occurrence of atrioventricular block and sinoatrial exit block during stress testing is rare. This directly guided our management. Stress testing reproduced transient high-grade atrioventricular block and sinoatrial exit block, and preceded progressive conduction deterioration over 4 years, to near-continuous ventricular pacing. Stress testing provides symptom-electrocardiogram correlation; the provoked atrioventricular block may indicate advanced conduction-system disease progression, and documentation of syncope with high-grade block supports timely pacing.

Keywords: Atrioventricular Block • Sinoatrial Block • Exercise Test • Cardiac Electrophysiology • Bundle-Branch Block • Pacemaker, Artificial • Electrocardiography

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/949566

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Introduction

Bifascicular block is defined as a combination of either right bundle branch block (RBBB) and left anterior fascicular block (LAFB), or RBBB and left posterior fascicular block (LPFB), or LAFB and LPFB [1,2]. In the majority of cases, clinical presentation of bifascicular block is asymptomatic and is identified incidentally. For patients with asymptomatic bifascicular block, no further evaluation or therapy is typically undertaken, but newly published data challenge this notion and suggest the possibility of inevitable progression of the conduction disease [3].

While exercise stress testing is not indicated for those with known second- to third-degree heart block, it does provide utility in further differentiating and evaluating symptoms in patients with conduction disorders who lack evidence of atrioventricular (AV) block on a resting electrocardiogram (EKG). Worsening of AV conduction during exercise stress testing helps determine the risk of progression to a higher-degree block and can establish whether there is an indication for pacemaker implantation. Furthermore, exercise stress testing can help in differentiating causes of syncope when there is uncertainty between conduction, arrhythmic, or ischemic etiologies, and it is recommended by current guidelines [2].

Here, we present a case of a patient with chronic bifascicular block including RBBB and LAFB, who had multiple episodes of syncope. As a part of workup, he underwent exercise stress testing. During exercise, the patient syncopized, with EKG changes showing third-degree AV block and sinoatrial exit block. The simultaneous AV block and sinoatrial exit block accompanied symptom reproduction. Longitudinal progression of conduction disease was then seen on device follow-up. The direct diagnostic and management implications of the case are discussed in light of the relevant literature.

Case Report

A 72-year-old man with a past medical history of treated prostate cancer, hyperthyroidism, and inguinal hernia was referred for evaluation after experiencing 2 syncopal episodes. The patient was first seen 3 years before presentation for a routine checkup, the EKG showed RBBB and LAFB with a QRS duration of 159 ms (**Figure 1**). The first episode of syncope was 2 years prior to presentation and the most recent was 1 month prior to presentation (see the timeline in **Figure 2**). Both episodes occurred during exertion, although the most recent one was preceded only by static exercise (first episode: running on subway platform to catch a train; second episode: ceiling hole drilling). There was no prodrome associated with either event; both events were sudden in nature. The patient denied anginal symptoms, dyspnea, or confusion. No incontinence was noted.

During evaluation, an EKG was performed, which again showed bifascicular block. He was then referred for a stress test. Given recurrent exertional syncope without prodrome in the setting of baseline bifascicular block, the working differential included intermittent high-grade AV block, ventricular arrhythmia, ischemia-related conduction disturbance, autonomic/reflex

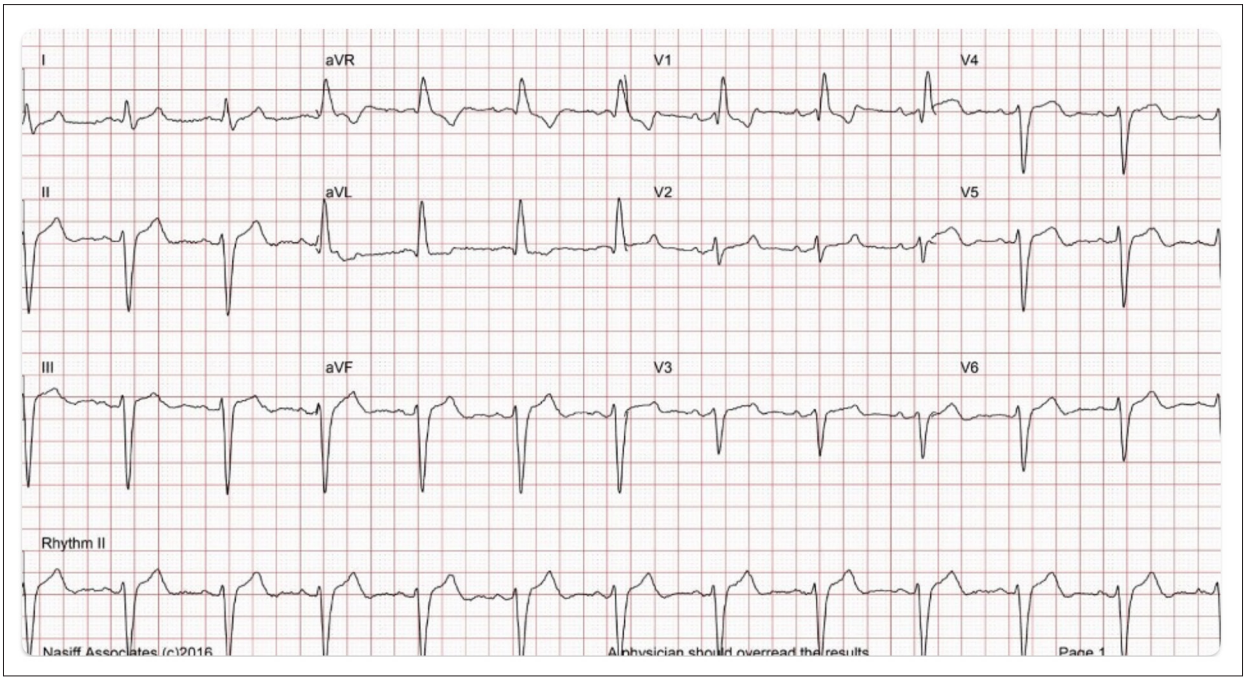

Figure 1. Patient's electrocardiogram from 2018 showing bifascicular block.

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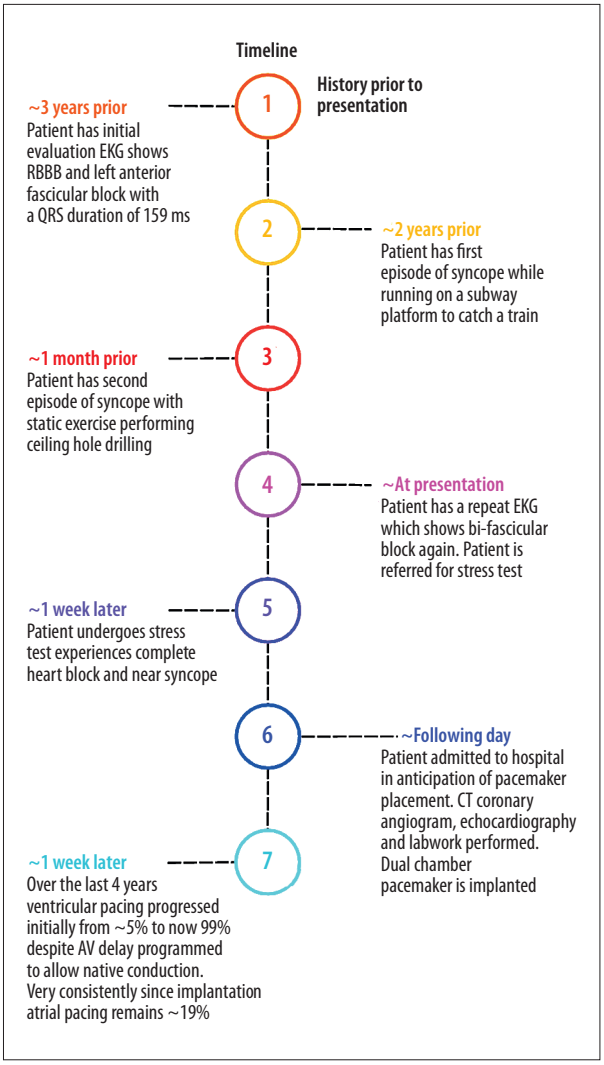

Figure 2. Timeline of events prior to pacemaker implantation and including follow-up. AV, atrioventricular; CT, computed tomography; EKG, electrocardiogram; RBBB, right bundle branch block.

syncope, and infiltrative disease. Because symptoms were reproducible with exertion yet infrequent, treadmill testing was selected to attempt real-time symptom-EKG correlation and to assess for ischemia. Ambulatory monitoring was considered less likely to capture an event, and an invasive electrophysiology (EP) study was deferred pending noninvasive correlation. While the patient was undergoing the test, during transition from stage 1 to stage 2 of the standard Bruce protocol, sudden onset of complete heart block was noted immediately following a long coupled premature ventricular contraction (PVC) (although we cannot completely rule out conduction with alternating bundle branch block) which was accompanied by near-syncope (**Figure 3A**). Notably, the block appeared abruptly rather than being preceded by progressive PR interval prolongation, supporting a distal conduction system mechanism. The patient was assisted to prevent a fall and assisted to a chair. The test was immediately terminated. After a few seconds, AV conduction improved; however, the patient remained in type II second-degree AV block, with a significantly longer PR interval (159 ms to 192 ms) and a varying pattern of conduction. The conduction pattern now showed left bundle branch block (LBBB) (152 ms) morphology, suggesting RBBB combined with left posterior branch block, or a more severe block in the main trunk of the left bundle branch (**Figure 3B**). Later in recovery (~3 min later), AV conduction returned, with bifascicular block, but the patient now developed repetitive episodes of sinoatrial Wenckebach type block (**Figure 3C**). As soon as the patient was seated, he felt better and confirmed that his symptoms were similar to his prior syncopal episodes.

The patient was admitted to the hospital with anticipation of urgent pacemaker implantation. To exclude ischemia as a potential trigger for AV block, we elected to proceed with CT coronary angiography, as the least invasive option. The CT coronary angiogram showed a calcium score of 25, no obstructive coronary lesions, and no hilar adenopathy or infiltrative lung disease, hence making diagnosis of sarcoidosis much less likely. Echocardiography showed no significant structural heart disease. Blood chemistry analysis and complete blood count were normal. Thyroid stimulating hormone was 2.54 µIU/mL. Lyme titers were negative as was the antinuclear antibody panel. Given these findings, we elected not to proceed with invasive EP evaluation as it would be extremely unlikely to provide any additional information. Although a finding of AV nodal conduction abnormalities during invasive EP study may help to elucidate the nature of syncope in some cases, with the advanced AV conduction abnormality seen during the stress test, this additional data would not provide us with any evidence dissuading us from pacemaker implantation. Furthermore, in patients without structural heart disease, as in this case, the diagnostic yield with respect to inducibility of ventricular arrhythmias is very low.

A dual-chamber permanent pacemaker was implanted without complications. Over the following 4 years, ventricular pacing progressed from an initial rate of ~5% to 99% despite AV delay programmed to allow for native conduction. Very consistently since implantation, atrial pacing remained at ~19%. The increase in the proportion of ventricular pacing from 5% to 99% is an extremely important finding, corroborating the progressive deterioration of the conduction system that was exposed by exercise stress testing.

Discussion

Our case is quite unique, showing AV nodal and sinoatrial conduction disturbance during exercise. To our knowledge

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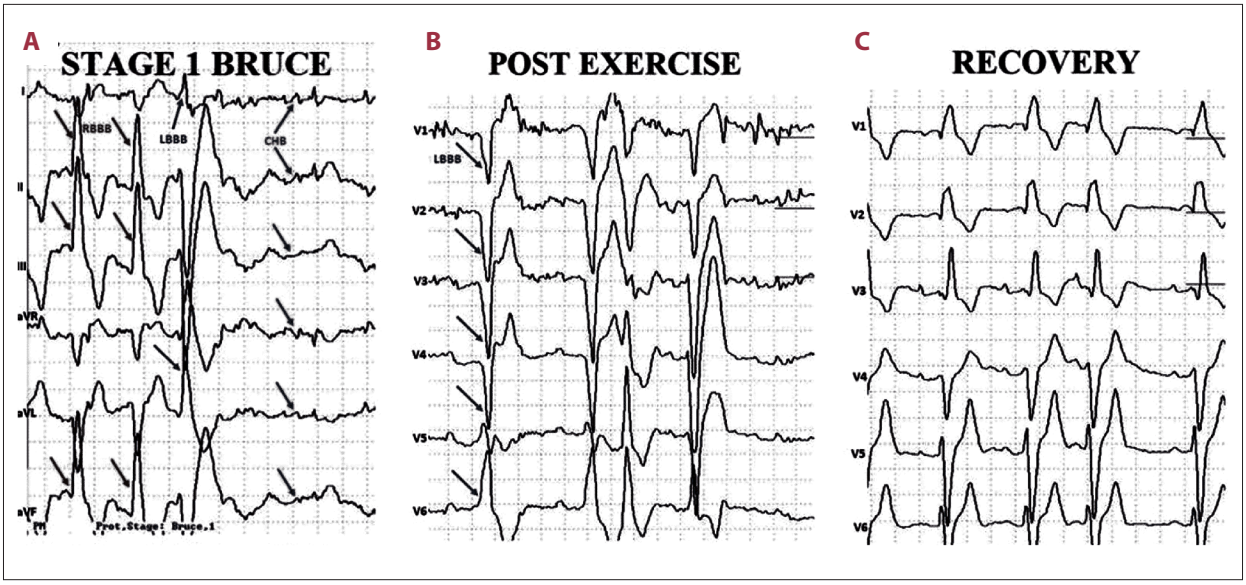

Figure 3. (**A**) Stress EKG showing Stage 1 Bruce protocol with PVC inducing CHB [green arrow]. (**B**) During the post-exercise period, second-degree Type II AV block was observed with LBBB. (**C**) At recovery, bifascicular block was observed with development of new second-degree Type I block. AV, atrioventricular; CHB, complete heart block; EKG, electrocardiogram; LBBB, left bundle branch block; PVC, premature ventricular contraction; RBBB, right bundle branch block.

no similar case has been reported in the literature. In a patient with bifascicular block and reproduced exertional syncope with documented complete AV block during stress testing, permanent pacing is supported by current guidelines [1,2]. Given the clear symptom-rhythm correlation and the subsequent progression of conduction disease, prolonged monitoring or EP testing was unlikely to change the need for pacing and therefore was not pursued as a prerequisite to therapy.

Exercise-induced AV block is rare (~0.45%) and mostly seen in patients with preexisting infra-Hisian disease [4-8]. This seems to be the case with our patient. Interestingly, we were able to reproduce his symptoms with a treadmill stress test and correlate them with the presence of AV block.

It is assumed that the development of AV block during exercise is associated with prolongation of refractoriness of the infra-Hisian conduction system due to age-related fibrosis. Other mechanisms for worsening of AV conduction have been proposed; namely electrical remodeling of ion channels [9]. This certainly has the appearance of so called “phase 3” or tachycardia-dependent block. This is typically present in patients with underlying His-Purkinje abnormalities. Subtle impairment in electrophysiological properties of the conduction system, slight changes in coronary perfusion, changes in autonomic tone, and/or change in circulating catecholamines have been suggested as possible culprits [10]. In our patient, the AV block appears to start with a late coupled PVC. We theorize that the PVC retrogradely penetrated the left bundle and the AV node, rendering the left bundle refractory and slowing conduction in the proximal AV node. Slowing of conduction in the AV node created a proximal delay and facilitated conduction though the right bundle. This explanation seems plausible; and furthermore, the patient had also developed syncope, and presumably AV block, with static exercise. However, this remains a hypothesis. Alternative explanations include autonomic shifts during exercise/recovery, transient ischemia below CT resolution, and nonspecific age-related conduction-system fibrosis [10,11]. We do not believe that ischemia (well known to be responsible for transient AV block) could explain the AV block as CT coronary angiography did not reveal obstructive coronary disease [11]. Furthermore, ischemia would not explain symptoms during static exercise. Sarcoidosis can present in similar fashion; however, we did not see any biochemical or imaging evidence for sarcoidosis or other infiltrative process [12,13].

The sinoatrial Wenckebach pattern was repetitive and each of the P waves were of identical morphology, thus excluding the possibility of the premature atrial contractions simply resetting the sinus node. Sinoatrial exit block is not uncommonly seen (28%) in patients undergoing monitoring for a suspected arrhythmic cause of symptoms, and is associated with progression of sinus node dysfunction [14]. Available data show rare (0.65% per year) coincidence of sinus node dysfunction in patients with AV block who were implanted with a pacemaker [6]. We could not identify a potential common mechanism for the coexistence of sinoatrial and AV block in this patient. It is plausible that age-related fibrosis may be the mechanism responsible for both phenomena [15].

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The efficacy of exercise stress testing in this patient with syncopal symptoms provides an excellent example of the utility of its use in patients with exercise-induced symptoms as well as conduction disease. We performed the exercise stress test as an initial step in evaluation of this patient, given the limitations imposed by the health insurance carrier. However, it must be acknowledged that both EP studies and cardiac monitoring may provide more definitive data than stress testing can in cases where exercise stress testing cannot provide a definitive answer.

No further episodes of syncope were observed after pacemaker implantation. No adverse events related to the intervention were observed.

Conclusions

This case illustrates the utility of exercise stress testing in selected patients with bifascicular block and unexplained exertional syncope when resting EKG does not show high-grade AV block. Pertinent take-home messages include: exertional syncope in bifascicular block warrants provocative testing or rhythm correlation when feasible; exercise-induced high-grade AV block with symptom reproduction strongly suggests advanced conduction-system disease and may predict progression; and concomitant sinoatrial exit block may signal more diffuse conduction system involvement. In this patient, stress-provoked high-grade block predicted irreversible conduction deterioration, reflected by progression from ~5% to 99% ventricular pacing over 4 years of follow-up.

Patient Consent

Patient consent was obtained for data to be published.

Department and Institution Where Work Was Done

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Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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