**ECG CHALLENGE**

**Multifaceted Left Bundle Branch Block**

*What Are the Mechanisms?*

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**ABSTRACT**

Understanding different mechanisms of aberrant conduction is critical to better evaluate the need for cardiac pacing. Aberrant conduction is caused by 4 distinct electrophysiologic mechanisms: phase 3 block, acceleration-dependent block, phase 4 block, and concealed transseptal conduction. This case offers a unique opportunity to review all aberrant conduction mechanisms in the same patient. *(Level of Difficulty: Intermediate.)* *(J Am Coll Cardiol Case Rep 2022;4:306–309) © 2022 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).*

**CASE REPORT**

A 59-year-old woman with cardiomyopathy (left ventricular ejection fraction: 20%) presented with left bundle branch (LBB) block (LBBB) morphology wide complex tachycardia *(Figure 1A)*. Electrophysiologic study confirmed the presence of nonsustained focal atrial tachycardia with LBBB aberrancy. Subsequent electrocardiograms (ECGs) revealed intermittent LBBB *(Figures 1B and 1C)*, while at other times, she had narrow QRS complexes *(Figure 1D)*.

Based on the surface ECGs from Figures 1A to 1C, what electrophysiologic mechanism can explain the development of LBBB?

A. Phase 3 block  
B. Acceleration-dependent block  
C. Phase 4 block  
D. Concealed transseptal conduction  
E. All of the above

**DISCUSSION**

The correct answer is E.

Aberration is defined as transient bundle branch block caused by a supraventricular impulse transmission during periods of physiologic refractoriness or pathologic depressed conductivity. Aberrant conduction can be explained by following mechanisms:

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Phase 3 block (tachycardia-dependent aberration) occurs when an impulse falls during phase 3 of the preceding action potential when a significant proportion of sodium channels are in an inactivated state, thus rendering the bundle branches refractory. Figure 1A illustrates physiologic phase 3 LBBB during supraventricular tachycardia. The right bundle branch (RBB) refractory period shortens to a greater degree compared to the LBB at increasing heart rates. Therefore, RBB block phase 3 aberrancy is more common at slower heart rates and LBBB aberrancy at faster heart rates.\(^3\)

Acceleration-dependent aberration occurs despite the impulse falling after completion of phase 3 or during early phase 4. This is typically seen with diseased His-Purkinje system with reduced or less negative resting membrane potential (RMP) during phase 4.\(^1\) Additionally, the refractory period in pathologic states can extend beyond full repolarization, further reducing excitability (postrepolarization refractoriness).\(^2\) As a result, slight changes in heart rate, at rates well beyond the duration of action potential, are associated with development of aberration. Figure 1B shows pathologic acceleration-dependent LBBB aberration at a heart rate of 80 beats/min. The clues to this mechanism are based on the facts that aberration occurred repetitively with atrial pacing at an unusually low heart rate (>80 beats/min), with slow decremental pacing and in the presence of severe underlying cardiomyopathy. Coexistence of both tachycardia-dependent and acceleration-dependent aberration mechanism in the same patient is a unique phenomenon that would need to be further studied.

Phase 4 block (pause-dependent aberration) occurs when an impulse is blocked despite falling long after the completion of repolarization (late phase 4). Typically, His-Purkinje cells exhibit flat RMP during phase 4. However, diseased His-Purkinje myocytes can develop progressive diastolic depolarization, resulting in less negative RMP and sodium channel inactivation. Figure 1C shows sinus bradycardia at 59 beats/min with pathologic LBBB aberration. As sinus heart rate accelerates to 66 beats/min (Figure 1D), the RMP remains negative enough to have sufficient sodium channels activated and sustained conduction through both bundle branches.

Concealed transseptal conduction (retrograde concealment) accounts for the perpetuation of physiologic LBBB aberration in Figure 1A. During LBBB, the impulse exits the RBB; activates the right ventricle; and then, via transseptal conduction, activates the left ventricle and LBB retrogradely. Retrograde penetration into the LBB may result in antegrade block for subsequent impulses. This results in persistence of the aberration indefinitely or may resolve with distal migration of the site of retrograde block in the LBB.

In this case, narrow QRS complex was observed only within a heart rate window of 60 to 80 beats/min (Supplemental Figure 1). The succession of various aberrant conduction mechanisms made LBBB appear almost permanent. However, a careful analysis of 12-lead ECGs and telemetry strips revealed LBBB aberrancy. The HV (50 ms) and RBB-V (35 ms) intervals remained similar with and without aberration (Supplemental Figure 2). Importantly, phase 3 block and concealed transseptal conduction are considered physiologic and do not warrant any further investigation. Contrarily, acceleration-dependent block and phase 4 block indicate a diseased His-Purkinje system.\(^1,2\)

The patient underwent biventricular cardiac resynchronization therapy for cardiomyopathy and LBBB.

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FIGURE 1 12-Lead Electrocardiograms

(A) A 12-lead electrocardiogram showing supraventricular tachycardia at 168 beats/min with left bundle branch block.

(B) A 12-lead electrocardiogram showing normal sinus rhythm at 80 beats/min with left bundle branch block.

(C) A 12-lead electrocardiogram showing sinus bradycardia at 59 beats/min with left bundle branch block.

(D) A 12-lead electrocardiogram showing normal sinus rhythm at 66 beats/min with left ventricular hypertrophy and repolarization abnormalities.
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KEYWORDS aberrant conduction, cardiomyopathy, intraventricular conduction delay, left bundle branch block

APPENDIX For supplemental figures, please see the online version of this paper.