Wide QRS complex tachycardia and alternating bundle branch block aberration: What is the mechanism?

Received: 7 January 2022 | Revised: 21 February 2022 | Accepted: 14 March 2022

DOI: 10.1002/joa3.12704

Journal of Arrhythmia Open Access WILEY

SPOTLIGHT

Wide QRS complex tachycardia and alternating bundle branch block aberration: What is the mechanism?

Hidehiro Iwakawa MD, PhD[1] | Ken Terata MD, PhD[1] | Haruwo Tashiro MD[1] | Yoshihisa Abe MD, PhD[2] | Hiroyuki Watanabe MD, PhD[1]

[1]Department of Cardiovascular Medicine, Akita University Graduate School of Medicine, Akita, Japan

[2]Department of Cardiovascular Medicine, Akita City Hospital, Akita, Japan

Correspondence
Hiroyuki Watanabe, MD, PhD, Department of Cardiovascular Medicine, Akita University Graduate School of Medicine, 1-1-1, Hondoh, Akita 010-8543, Japan.
Email: hirow@doc.med.akita-u.ac.jp

Keywords: aberration, atrial tachycardia, left bundle branch block, refractory period, right bundle branch block

A 70-year-old man with no structural heart disease was referred to our hospital for evaluation and treatment of wide QRS complex tachycardia with a left bundle branch block (LBBB) morphology (Figure 1A). An electrocardiogram (ECG) showed atrial bigeminy with an alternating bundle

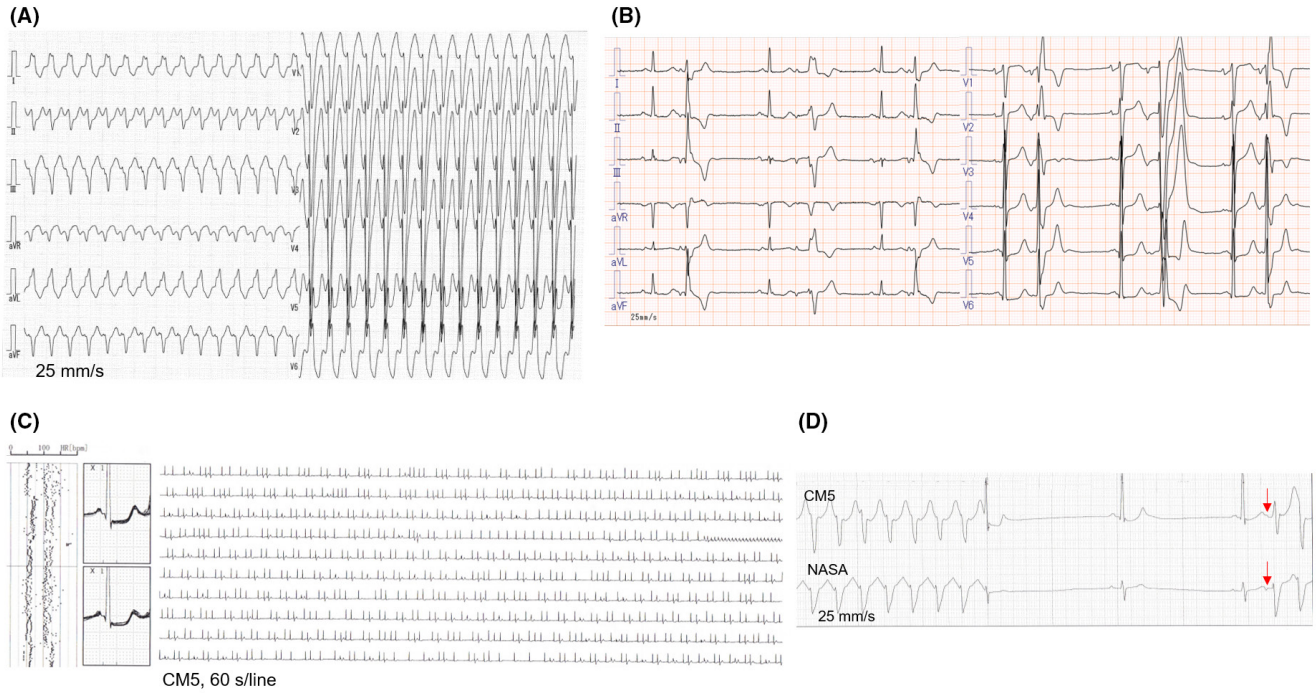

FIGURE 1 (A) Electrocardiography (ECG) shows wide QRS complex tachycardia with a left bundle branch morphology, at a rate of 180 beats/min. (B) The ECG shows atrial bigeminy with alternating bundle branch block. Although the atrial premature complex (APC) coupling interval is constant, the PQ interval during the left bundle branch block aberration is longer than during the right bundle branch block beats. (C) Holter ECG recording. *Left panel*: heart rate trend and superimposed ECG waveforms. *Right panel*: ECG strips in CM5 lead (60 s/line) demonstrating incessant APCs and nonsustained wide QRS complex tachycardia. (D) Holter ECG strips in CM5 and NASA leads show termination and initiation of wide QRS complex tachycardia. This tachycardia is initiated by P-wave (arrows)

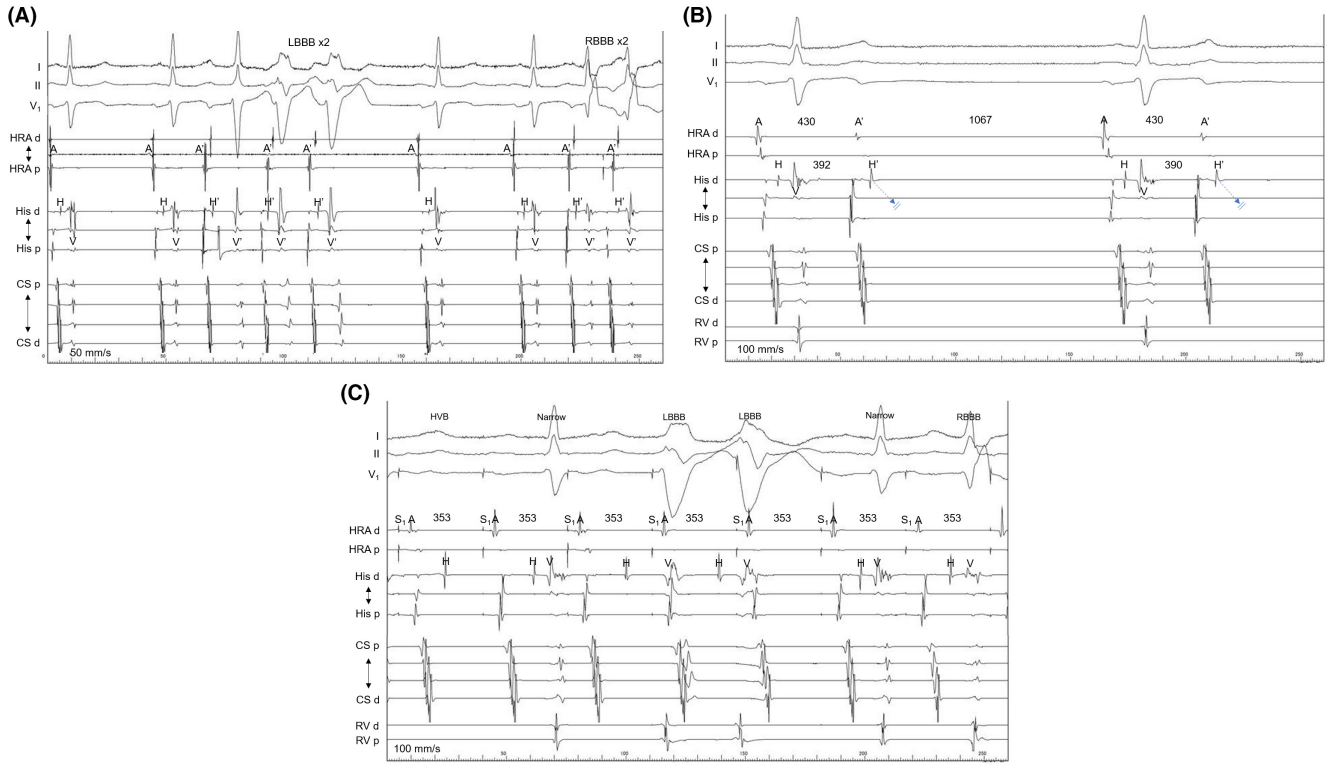

FIGURE 2 (A) Nonsustained atrial tachycardia (AT) with left and right bundle branch block aberrations. The earliest atrial electrogram is recorded in the His-bundle region during AT. Note that the QRS morphology on the third beat (first premature beat) shows an intraventricular conduction disturbance with prolongation of the His-ventricular interval. (B) Premature atrial ectopic beats with the earliest atrial electrogram recorded at the His-bundle region occur in a bigeminal fashion. Premature ectopic impulses are blocked below the His-bundle region. (C) Programmed atrial stimuli with a pacing of 170 beats/min reveal a His-ventricular block and subsequent alternating bundle branch block aberration. Abbreviations: I, II, and V1, surface electrocardiographic leads; HRA, His, CS, and RV, intracardiac electrogram recorded from the high right atrium, His-bundle region, coronary sinus, and right ventricular apex, respectively; p and d, proximal and distal electrodes, respectively; A', H', and V', premature ectopic beats; LBBB, left bundle branch block; RBBB, right bundle branch block; HVB, His-ventricular block. Numbers express each interval in ms

branch block (ABBB) aberration (Figure 1B). Holter ECG showed incessant atrial premature complexes (APCs) and nonsustained tachycardia with multiple QRS morphologies, and one of the tachycardia episodes began with P-wave (Figure 1C,D). He underwent an electrophysiology study (EPS) under minimum sedation. All antiarrhythmic drugs were stopped for at least 5 half-lives before EPS. The baseline atrial-His (AH) and His-ventricular (HV) intervals were 85 and 38 ms, respectively. Nonsustained atrial tachycardia (AT) occurred frequently in the earliest atrial electrogram (EGM) recorded in the His-bundle region, with a narrow QRS complex and left or right bundle branch block (RBBB) configuration (Figure 2A). Occasional APCs with HV block (HVB) were documented (Figure 2B). Retrograde conduction was concentric, with the earliest atrial EGM recorded at the coronary sinus ostium. Atrial extrastimuli pacing was difficult because pacing trains were interrupted by frequent APCs. Programmed atrial stimuli with the pacing of 160, 170, and 180 beats/min reproducibly demonstrated the alternating LBBB and RBBB aberration (Figure 2C and Supplementary Figure). What is the mechanism?

Although clinically documented wide QRS tachycardia was not inducible, wide QRS tachycardia was considered to be AT with LBBB aberration, based on the Holter ECG findings and the nonsustained AT with LBBB aberration seen during the EPS. Figure 3A depicts a laddergram for the ABBB aberration during atrial stimuli pacing of 170 beats/min. This phenomenon can be explained by three mechanisms: the refractory periods of the bundle branches, which depend on the preceding cycle length; the functional difference in the refractory period between the left and the right bundle branches; and the concealed retrograde transseptal conduction.

The first captured atrial beat is blocked below the His-bundle region, followed by a narrow QRS and LBBB morphology. The third captured beat conducts down the right bundle branch (RBB), resulting in LBBB morphology, so the left bundle branch (LBB) is in a refractory period during the third beat, indicating a longer preceding cycle length of the LBB than the RBB. Therefore, LBB activation must be completed before RBB activation at the first beat. The LBBB morphology persists during the fourth beat, likely due to concealed retrograde transseptal conduction (the so-called "linking" phenomenon).[1] The third beat conducts down the RBB, and the impulse then crosses the septum and retrogradely activates the LBB. The fourth beat also conducts down the RBB because the LBB is once more in a refractory state for the atrial impulse. The fifth beat shows an AH block, and the sixth beat conducts down both bundle branches (resulting in a narrow QRS). Consequently, the seventh captured atrial beat conducts down the LBB (resulting in

RBBB morphology) because of the long-short sequence in the RBB, in turn, due to concealed retrograde transseptal conduction from the RBB to LBB during the fourth beat.

An ABBB aberration concomitant with atrial bigeminy is occasionally observed, and a few case reports have mentioned its mechanisms.[2,3] However, in all cases, the proposed mechanisms were

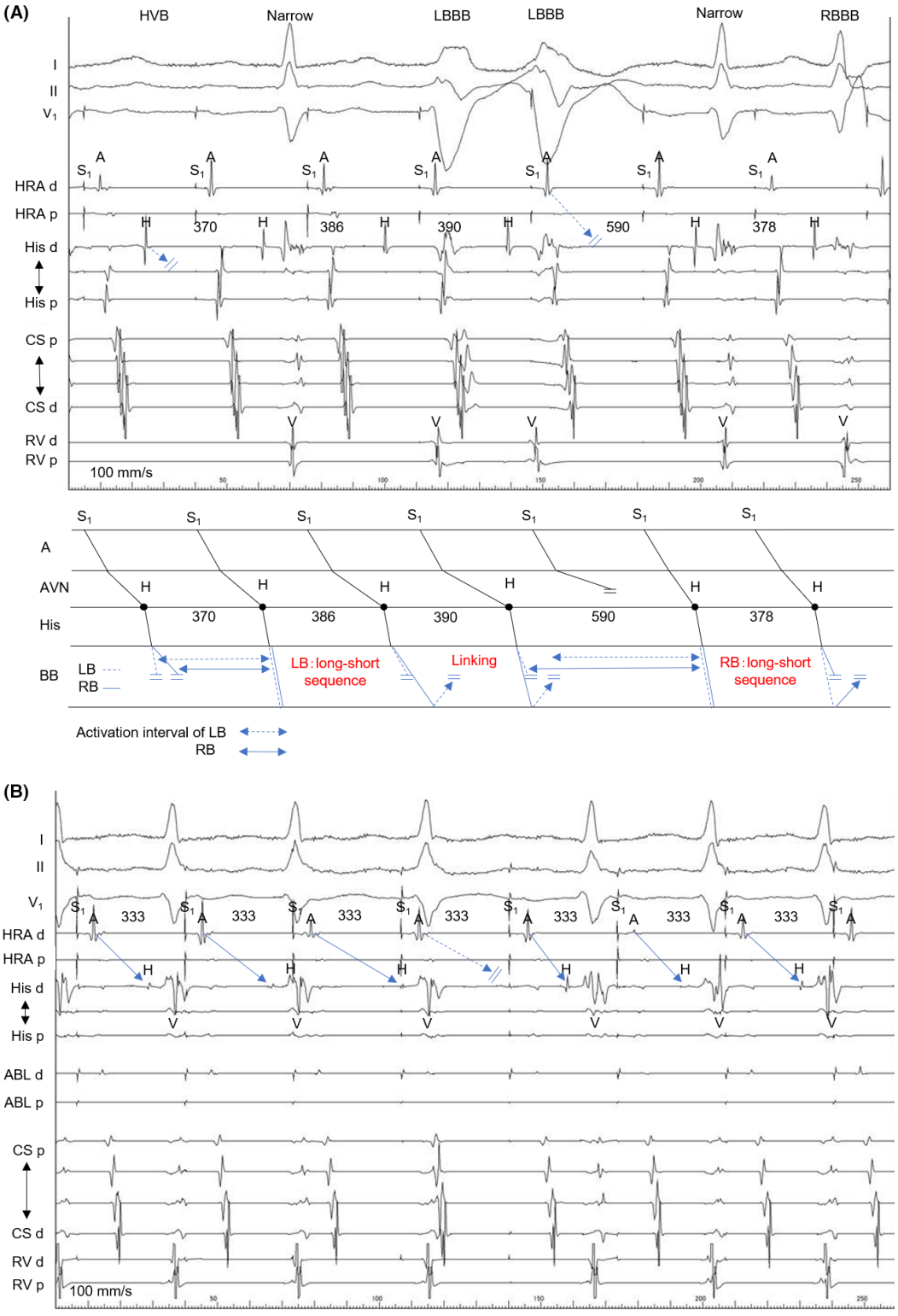

FIGURE 3 (A) *Upper panel*: Alternating bundle branch block aberration with atrial stimuli pacing of 170 beats/min. *Lower panel*: The laddergram; see the text for details. Note that the third beat shows prolongation of the His-ventricular interval due to simultaneous conduction delays over both branches. (B) After eliminating the atrial premature contractions, an atrial-His block, but not a His-ventricular block, is seen. LB, left bundle; RB, right bundle; other abbreviations as in Figure 2

based on surface ECGs because atrial bigeminy is usually treated conservatively. To the best of our knowledge, this is the first case of ABBB assessed via intracardiac EGM. Some reports considered a marked prolongation of the PQ interval of an aberrant beat as AH interval prolongation.[3] However, this case demonstrates that it mainly consists of HV interval prolongation due to simultaneous conduction delays over both branches (Figure 3A, the third beat).

During detailed mapping within the right atrium, the earliest atrial activation site of the APC/AT was recorded in the anterior part of the tricuspid annulus, at a distance of 17 mm from the His-bundle. Administering 4 mg adenosine triphosphate (ATP) transiently suppressed the APC/AT, suggesting that the APC/AT was ATP sensitive. Radiofrequency ablation at this site successfully eliminated the APC/AT. The postoperative AH and HV intervals were 77 and 39 ms, respectively. After elimination of the APC/AT, HVB and ABBB were not reproduced by programmed atrial stimulation with or without an infusion of isoproterenol (Figure 3B), suggesting the conduction disturbance in the His-Purkinje system (HPS) before ablating APC/AT was a functional mechanism. One study reported that impulses arriving at the HPS at varying intervals showed unpredictable degrees of conduction delay in the HPS.[4] Therefore, incessant APC/AT could invoke heterogeneous changes in the activation intervals and refractory periods in the HPS. After 2 years of follow-up, the patient remains free from recurrence of the APC/AT and episodes of bradycardia.

In conclusion, we report a case of AT with an LBBB aberration and ABBB concomitant with a functional conduction delay in the HPS. Incessant APCs/AT could be associated with a functional conduction delay in the HPS.

ACKNOWLEDGMENTS

The authors would like to thank Yasutaka Osanai, Yuki Nibori, Kosuke Hamaura, and Hisashi Shimazaki for their technical assistance during the electrophysiological study.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest in this article.

ORCID

Hidehiro Iwakawa https://orcid.org/0000-0002-2441-3593
Hiroyuki Watanabe https://orcid.org/0000-0001-8690-2416

REFERENCES

1. Lehmann MH, Denker S, Mahmud R, Addas A, Akhtar M. Linking: a dynamic electrophysiologic phenomenon in macroreentry circuits. Circulation. 1985;71:254–65.
2. Ho RT, Wong KL, Kocovic DZ, Pavri BB. Alternating bigeminy. J Cardiovasc Electrophysiol. 2001;12:273–4.
3. Carbone V, Zito GB, Oreto L, Oreto G. Nonsustained atrial tachycardia with right or left bundle branch block aberration: what is the mechanism? Heart Rhythm. 2014;11:1845–7.
4. Denker ST, Gilbert CJ, Shenasa M, Akhtar M. An electrocardiographic—Electrophysiologic correlation of aberrant ventricular conduction in man. J Electrocardiol. 1983;16:269–77.

SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

How to cite this article: Iwakawa H, Terata K, Tashiro H, Abe Y, Watanabe H. Wide QRS complex tachycardia and alternating bundle branch block aberration: What is the mechanism?. J Arrhythmia. 2022;38:478–481. https://doi.org/10.1002/joa3.12704