Received: 18 September 2021 | Revised: 30 October 2021 | Accepted: 16 November 2021

DOI: 10.1002/joa3.12659

Journal of Arrhythmia Open Access WILEY

An atypical case of atrioventricular block in an elderly man: A fortuitous escape

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1 | ECG FOR STUDENTS AND ASSOCIATED PROFESSIONALS

A 64-year-old man presented with exertional dyspnea for 6 months. Evaluation revealed coronary artery disease with significant stenosis of proximal left circumflex artery and mild left ventricular dysfunction. On examination, he was observed to have persistent bradycardia. He denied any history of syncope or presyncope. Baseline electrocardiogram (ECG) showed sinus bradycardia and 1:1 atrioventricular (AV) conduction. PR interval was prolonged with a duration of 440 ms. The QRS complex was normal with a duration of 110 ms.

A 24-h Holter recording was performed. The maximum and minimum heart rates were 88 and 32 beats per minute. A strip from that recording is shown in Figure 1.

What is arrhythmia? What is the mechanism of this rhythm abnormality?

2 | COMMENTARY

The first three beats are normally conducted sinus beats at cycle length (CL) of 1360 ms with prolonged PR interval, similar to the baseline ECG (not shown). After the third beat, there is an atrial premature complex (APC) occurring after a duration of 1080 ms from the last P wave (orange arrowhead, Figure 2). This APC is not conducted to the ventricles, and there is a noncompensatory pause after the third QRS complex. There is an escape beat following the APC, with a morphology similar to that of the conducted beats. A nonconducted sinus P wave can be seen at the beginning of this complex (arrow, Figure 2). This sinus beat is followed by an ectopic atrial beat at a CL of 1400 ms which is again not conducted. Subsequently, there is a resumption of sinus rhythm at the original CL of 1360 ms. However, none of these sinus P waves are conducted to the ventricles. There is an escape rhythm at CL of 2000 ms which is likely of His bundle origin. There is complete atrioventricular (AV) dissociation following the APC, consistent with paroxysmal AV block (PAVB). After five escape beats, there is an appropriately timed sinus beat which is conducted to the ventricles. There is subsequent resumption of normal sinus rhythm at the original CL of 1360 ms.

PAVB is a poorly defined clinical entity characterized by sudden and unexpected change from 1:1 AV conduction to complete AV block. It is an important but under-recognized etiology of syncope, as it is usually associated with delayed emergence of an adequate escape rhythm.[1] PAVB has been classified into three types: (i) intrinsic paroxysmal AV block because of intrinsic disease of the AV conduction system, (ii) extrinsic vagal paroxysmal AV block which is linked to enhanced parasympathetic influence on cardiac conduction, and (iii) extrinsic idiopathic paroxysmal AV block which is seen in subjects with normal AV conduction and without heart disease and is proposed to be secondary to an increased susceptibility to adenosine.

Figure 1 is an example of intrinsic PAVB which is commonly seen in elderly individuals with structural heart disease and baseline conduction system abnormalities. Premature atrial or ventricular beats or sinus slowing itself have been associated with the onset of intrinsic PAVB. This is in contrast to extrinsic idiopathic PAVB in which initiation occurs without any premature complexes or decrease in sinus rate. In Figure 2, the sinus rate remains unchanged, and the PR interval is constant prior to the onset of AV block. This differentiates it from extrinsic vagal PAVB as the latter is associated with progressive slowing of sinus rate and PR prolongation prior to the onset of block, with a persistent slow sinus rate during the block.

The mechanism by which a premature beat like the APC in this figure leads to intrinsic PAVB involves the phenomenon of Phase

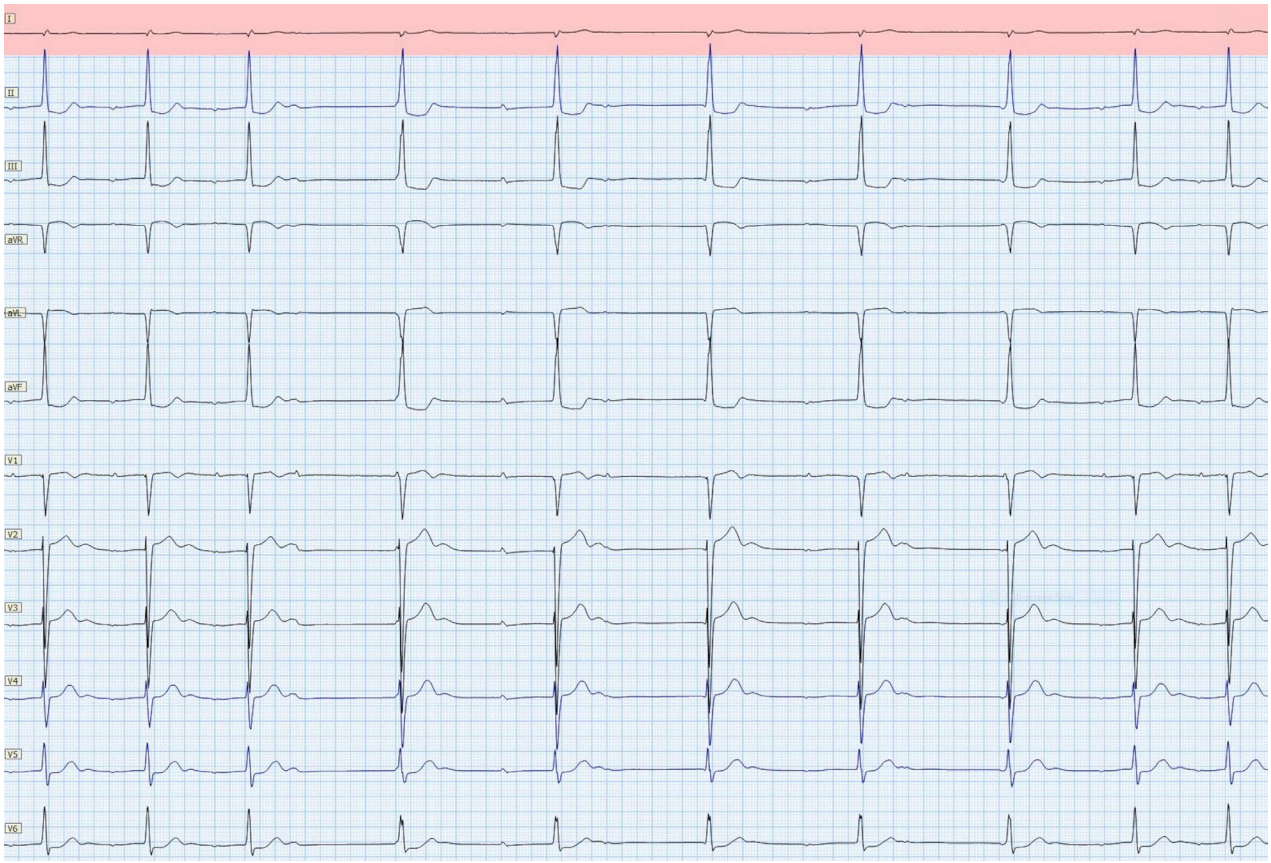

FIGURE 1 A strip from the 24-h Holter recording

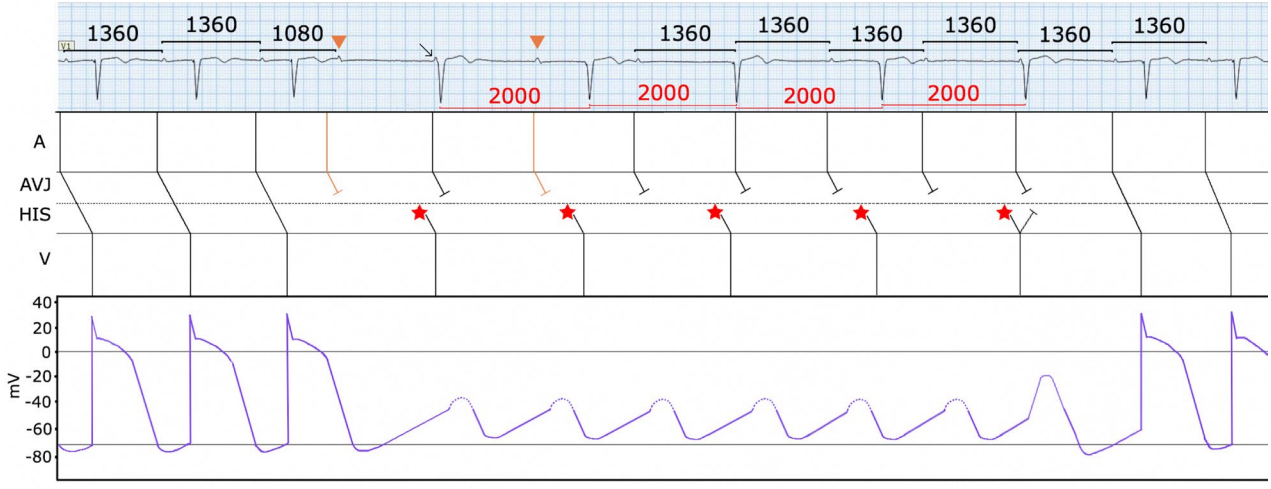

FIGURE 2 Annotated ECG recording, with ladder diagram, and membrane potentials. After the third beat, there is an atrial premature complex (APC) (orange arrowhead) occurring after a duration of 1080 ms from the last P wave. The pause after APC allows spontaneous diastolic deploarization in the diseased His bundle (bottom panel). The atrial beats reaching the His bundle lead to subthreshold action potentials because of inactive sodium channels, thus are not conducted further. There is an escape rhythm from the His bundle at a cycle length of 2000 ms, with a QRS morphology similar to the conducted beats. There is an appropriately timed fifth escape beat that resets the membrane potential to resting value, which allows subsequent conduction to resume normally

4 block also known as bradycardia-dependent block, deceleration block, or pause-dependent block. Spontaneous diastolic depolarization (SDD) during phase 4 of action potential is a normal electrophysiologic property of specialized automatic cells including His Purkinje system (HPS). With a decrease in the frequency of stimulating impulses, as in the post extrasystolic pause following the

initiating APC in this strip, spontaneous depolarization during phase 4 is enhanced (lowermost panel in Figure 2). However, it should be noted that SDD, by itself, cannot lead to conduction disturbances in a patient with normal HPS. Phase 4 block involves enhanced SDD in conjunction with generalized diastolic depolarization (hypopolarization) because of intrinsic HPS disease. Hypopolarization results in a less negative resting membrane potential. There is prolonged inactivation of sodium channels and diminished rate of rise of action potential which is the most important determinant of conduction velocity. Once a critical diastolic membrane potential is reached, conduction ceases and resumes only when an appropriately timed escape beat or premature beat resets the transmembrane potential to its maximal resting value.[2]

In adults with PAVB, especially in the elderly, presyncope, syncope, or sudden cardiac death is the rule because PAVB has been classically described as having no adequate escape rhythm. This is believed to be because of shifting of the threshold potential toward zero. SDD may reach a particular level and remain stable at that point, and the escape beats that do occur are not conducted further because of the blocking effect of SDD on the neighboring fibers.[3]

3 | CONCLUSION

Our case was notable for asymptomatic PAVB because of the presence of a stable escape rate which has hitherto not been well described. Since SDD does not exist in the central AV node area, the locus of escape rhythm has to be at or distal to the His bundle. Intrinsic PAVB has been classically described to have a high likelihood of progression to persistent AVB unlike the extrinsic vagal or idiopathic forms. Accordingly, permanent pacemaker implantation is indicated in intrinsic PAVB.

CONFLICT OF INTEREST

The authors declare no conflict of interests for this article.

AUTHOR CONTRIBUTIONS

Anish Kapil and Sai D. Prakash were involved in drafting the article, Anish Kapil and Siddharthan Deepti were involved in image interpretation and revision of the article.

DATA AVAILABILITY STATEMENT

Data and images provided in the manuscript are archived in the Medical Records Department of the All India Institute of Medical Sciences, New Delhi.

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How to cite this article: Kapil A, Prakash SD, Deepti S. An atypical case of atrioventricular block in an elderly man: A fortuitous escape. J Arrhythmia. 2022;38:174–176. https://doi.org/10.1002/joa3.12659