An analysis of a 4:2 atrioventricular block, a rare occurrence

Nicholas Mouser, MD, Alexander Ivanov, MD, Prashant Bhave, MD, FHRS

From the Division of Cardiovascular Medicine, Wake Forest University School of Medicine, Winston-Salem, North Carolina.

Introduction

The 2018 ESC guidelines define syncope as "[total loss of consciousness] due to cerebral hypoperfusion, characterized by rapid onset, short duration, and spontaneous complete recovery."[1] Because these episodes disrupt everyday life and can cause significant morbidity and mortality, syncope is a common reason for presenting to the emergency department (ED).[2] Not only are older patients more likely to present with this symptom, but they are also more often hospitalized and are at increased risk for unfavorable outcomes.[2] Cardiac conduction disease is an important etiology of syncope that occurs more frequently in the elderly.[3] Patients who suffer from conduction disturbances may be asymptomatic or experience a broad spectrum of symptoms from syncope to hemodynamic instability.[3]

Atrioventricular (AV) heart block is one type of conduction disease that can lead to syncope. Patients with chronic bifascicular block (BFB) are at risk for degeneration to AV block, particularly if syncope is present.[4] Here, we discuss an 81-year-old female patient with recurrent syncope who presents with a rare form of second-degree AV block with a 4:2 pattern of block. To date, there have been rare occurrences of 4:2 AV block published in the literature, but the mechanism has yet to be fully elucidated. In this case report, we further explore plausible mechanisms behind such findings and offer guidance for managing patients who present with cardiac causes of syncope.

Case report

An 81-year-old woman with a history of hypertension and hypothyroidism presented to the ED for the third time in a 4-month period for syncope. Work-up during prior admissions was unrevealing except for a new BFB and prolonged PR interval of 264 ms. The patient was encouraged to follow-up with neurology after her second ED visit.

KEY TEACHING POINTS

- Syncope is a common complaint in the emergency department and is more likely to afflict the elderly. Because syncope is associated with increased morbidity and mortality, it is important to consider conduction disease as the etiology of syncope.
- A 4:2 pattern AV block is a rare yet high-grade block that merits consideration for pacemaker placement. Possible mechanisms for this finding include a 4:3 block superimposed on a 3:2 block.
- About 25% of patients with syncope have a bifascicular block (BFB). Syncope of undetermined etiology with the presence of BFB should be referred for pacemaker placement as a IIA recommendation according to ACC/AHA/NASPE, as studies show that pacemaker placement reduces symptomatic events.

Because she endorsed prodromal lightheadedness and identifiable triggers, her neurologist placed an ambulatory cardiac patch monitor to assess for malignant bradyarrhythmias.

During this third ED admission, the patient denied chest pain and was not on any AV nodal blocking agents. Complete blood count, electrolytes, and thyroid-stimulating hormone were unremarkable. Initial electrocardiogram (ECG) showed sinus tachycardia with 1:1 AV conduction, a prolonged PR interval of 240 ms, right bundle branch block (RBBB), and left anterior fascicular block (LAFB) (Figure 1). Shortly after the first ECG was obtained, she had an episode of asystole while on the bedpan owing to AV block requiring brief cardiopulmonary resuscitation before return of spontaneous circulation. Repeat ECG demonstrated a 4:2 AV block pattern with RBBB and LAFB, which was originally interpreted as normal sinus rhythm, 2:1 AV block with RBBB premature ventricular complexes in a bigeminy pattern (Figure 2). Electrophysiology was consulted for further management.

KEYWORDS Syncope; 4:2 atrioventricular block; Bifascicular block; Pacemaker; High-grade AV block
(Heart Rhythm Case Reports 2024;10:525–528)

Address reprint requests and correspondence: Dr Nicholas Mouser, Atrium Health Wake Forest Baptist Medical Center, 1 Medical Center Blvd, Winston-Salem, NC 27157. E-mail address: nicholasamouser@gmail.com.

https://doi.org/10.1016/j.hrcr.2024.05.004

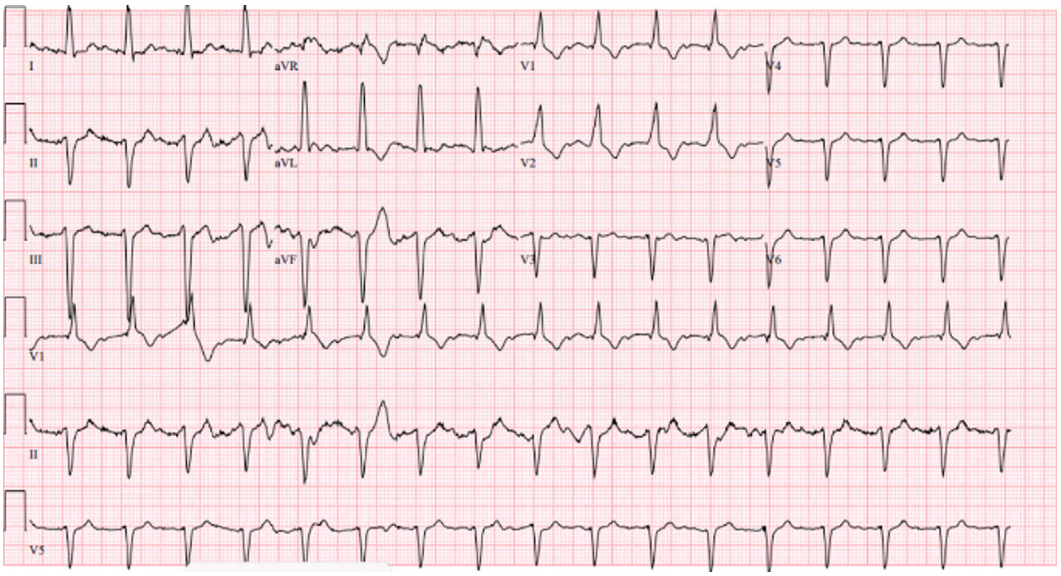

Figure 1 Initial electrocardiogram obtained on admission showing sinus tachycardia, prolonged PR interval, right bundle branch block, and left anterior fascicular block.

The following day, an echocardiogram was obtained and showed normal biventricular size and systolic function with a left ventricular ejection fraction of 60%–65%. Biatrial sizes were normal. Mild aortic and mitral valve thickening were present, but there was no significant valvular stenosis, regurgitation, or calcification. Her ambulatory cardiac patch monitor was removed, and processing of the monitor data was expedited. Results demonstrated pauses owing to paroxysmal high-grade AV block. Her presenting syncopal event was correlated to an 8.5-second pause. Her episode of asystole while on the bedpan corresponded with a 44-second pause. Ultimately the patient underwent implantation of a dual-chamber pacemaker with left bundle branch area pacing for high-grade heart block, and her syncopal episodes resolved.

Discussion

Mechanism of 4:2 AV block

In the case described above, the patient was an 81-year-old woman who presented with syncope and ECG findings showing BFB and first-degree AV block that ultimately degenerated into a high-grade AV block with a unique 4:2 pattern, a finding that is exceedingly rare. The repeat ECG was interpreted as a 2:1 AV block with RBBB premature ventricular complexes in a bigeminal pattern (Figure 2).

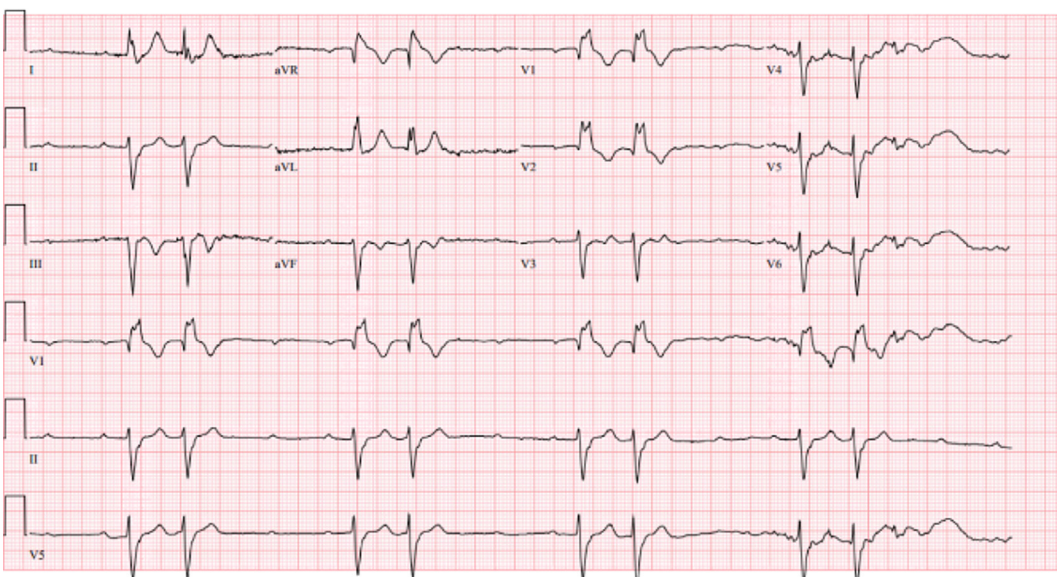

Figure 2 Repeat electrocardiogram showing sinus tachycardia with 4:2 AV block and a right bundle branch block and left anterior fascicular block.

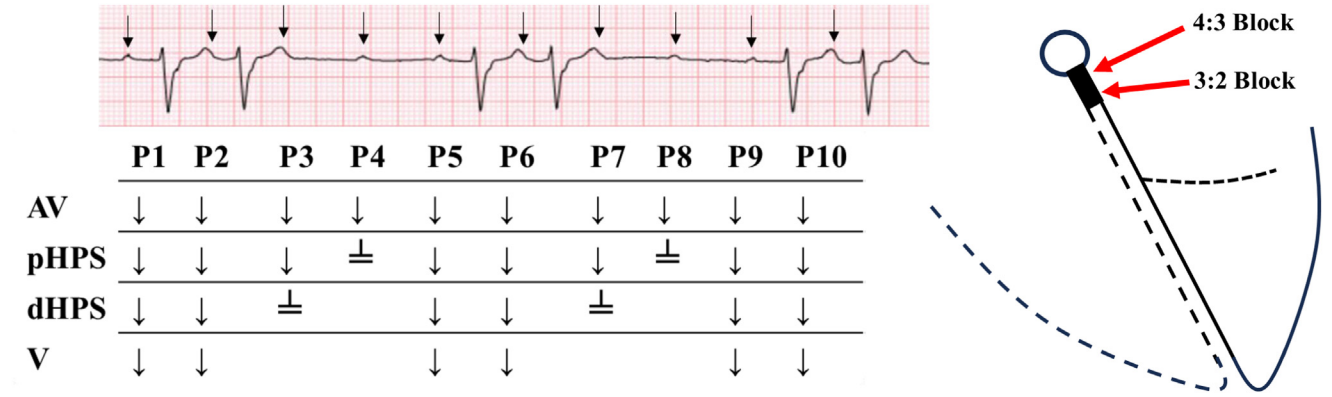

Figure 3 On the left, electrocardiogram shows lead II with vertical arrows indicating P-wave impulses. In the ladder diagram, all impulses are conducted through the AV node (AV). There is 4:3 block in the proximal His-Purkinje system (pHPS) and 3:2 block in the distal His-Purkinje system (dHPS). This results in a 4:2 conduction to the ventricles (V). The right image provides a visual display of the multilevel blocks within the HPS. The dashed lines of the right bundle branch and left anterior fascicle indicate chronic block while the solid line of the left posterior fascicle indicates possible conduction.

However, the ECG demonstrated clear P waves for P1 and P4 with P2 and P3 buried in the preceding T waves (Figure 3); this, in conjunction with the observation that the QRS complexes are all alike, suggests that the QRS complexes are all conducted.

The unusual pattern of conduction (2 conducted p waves followed by 2 nonconducted p waves) suggests the presence of a multilevel block in which there is a proximal 4:3 block and a more distal 3:2 block. Discerning the levels of each block in this case is challenging. The second p wave of each couplet of conducted impulses is buried in the preceding t wave, yet there does not seem to be any prolongation of the PR interval. As such, the apparent fixed PR interval suggests that both blocks are infranodal (Figure 3). The superimposition of these blocks would explain why the third and fourth impulses fail to conduct, giving rise to the observed ECG findings.

Upon literature review, there were only a few cases of ECG-captured 4:2 conduction block. One case report by Jastrzebski and Kukla[5] described a patient with inferior wall myocardial infarction who developed advanced second-degree AV block in a 4:2 pattern with RBBB and LAFB. They offer 3 plausible explanations for such findings: (1) 4:1 AV block with supernormal conduction: in this case, supernormal conduction is thought to occur if the P2 stimulus falls in the later stage of phase 3 repolarization of the Purkinje fibers during which time the ability to respond to stimuli has been restored[6]; (2) the presence of a 2-level block with 4:3 conduction in the upper level and 3:2 conduction in the lower level; and (3) development of 2 populations of Purkinje cells in the remaining, injured fascicle. One population of cells suffers from prolonged recovery giving rise to a phase 3 block of the P3 impulse. After the pause that ensues from the blocked P3, the second population of cells exhibits a spontaneous diastolic depolarization resulting in a phase 4 block, so P4 is not conducted.

Although the exact mechanism remains uncertain, it is possible that several of the pathophysiologic phenomena explained above could give rise to similar ECG findings manifesting as a 4:2 pattern block. If true, this would indicate that the 4:2 pattern block is not specific for any one disease process. Whatever the case, this particular pattern indicates high-grade block, and appropriate management should include consideration of a permanent pacemaker.

Clinical guidance

Syncope is a major cause of morbidity and mortality, particularly as patients age.[2] As such, it is important for providers to be aware of the appropriate management for syncopal patients. While determining the underlying etiology of syncope can be challenging, using one of the available "syncope rules" can be useful for stratifying patients based on risk. The San Francisco Syncope Rule,[7,8] for instance, uses 5 parameters to evaluate patients: history of congestive heart failure, hematocrit less than 30%, ECG abnormalities (non–sinus rhythm or new ECG changes), shortness of breath, and a systolic blood pressure less than 90 mm Hg at triage. If any 1 of the 5 parameters is positive, the patient is considered high risk for a poor outcome. In the case described above, the patient met criteria for high-risk syncope, since ambulatory cardiac monitoring revealed episodes of non–sinus rhythm.

Another important consideration in the work-up of patients with syncope is the presence of BFB. Specifically, BFB may consist of a left bundle branch block or the combination of a RBBB with either a LAFB or left posterior fascicular block.[9] Of adults presenting with syncope, approximately 25% have evidence of BFB on ECG, and BFB alone is associated with increased mortality.[10] Therefore, ACC/AHA/NASPE guidelines[10,11] advise pacemaker placement as a class IIA recommendation for patients with syncope of undetermined origin and BFB, even in the absence of AV block. To further support this recommendation, the PRESS study[10] sought to quantify the benefits of pacemaker placement for this population and investigated

whether dual-chamber pacemaker would reduce symptom recurrence compared to a control with effectively negligible pacing. Results showed a significant reduction in combined symptomatic episodes, which included syncope, presyncope, and AV block of all degrees in the dual-chamber pacemaker treatment group compared to the control over a 2-year period. This study gives concrete evidence that pacemaker intervention is effective in this patient population.

It should be noted that the patient described in this report was twice admitted to the ED with syncope of unexplained etiology and BFB and was not recommended to have an ambulatory cardiac patch monitor until her visit with a neurologist. It is important on a primary care level that providers remain vigilant and make the appropriate referral for pacemaker placement in patients with syncope of undetermined origin and BFB.

Conclusion

Conduction system diseases are an important cause of syncope that can lead to increased morbidity and mortality, particularly in elderly patients. AV block is a type of conduction disturbance that can lead to syncope. Patients with underlying BFB are susceptible to progressing to high-grade AV block. While uncommon, a 4:2 AV block pattern is a unique finding that represents a high-grade AV block that requires permanent pacemaker placement. To prevent presyncopal and syncopal episodes and to prevent degeneration to AV block, patients with syncope and underlying BFB should be referred for pacemaker placement.

Funding Sources: None.

Disclosures: NM, AI, and PB have no disclosures nor conflicts of interest to report.

References

1. Brignole M, Moya A, de Lange FJ, et al. 2018 ESC Guidelines for the diagnosis and management of syncope. Eur Heart J 2018;39:1883–1948.
2. Sun BC, Emond JA, Camargo CA Jr. Characteristics and admission patterns of patients presenting with syncope to U.S. emergency departments, 1992-2000. Acad Emerg Med 2004;11:1029–1034.
3. Chow GV, Marine JE, Fleg JL. Epidemiology of arrhythmias and conduction disorders in older adults. Clin Geriatr Med 2012;28:539–553.
4. Martí-Almor J, Cladellas M, Bazán V, et al. Nuevos predictores de evolución a bloqueo auriculoventricular en pacientes con bloqueo bifascicular [Novel predictors of progression of atrioventricular block in patients with chronic bifascicular block]. Rev Esp Cardiol 2010;63:400–408.
5. Jastrzebski M, Kukla P. Atrioventricular block with 4:2 conduction pattern: what is the mechanism? J Electrocardiol 2009;42:684–686.
6. Elizari MV, Schmidberg J, Atienza A, Paredes DV, Chiale PA. Clinical and experimental evidence of supernormal excitability and conduction. Curr Cardiol Rev 2014;10:202–221.
7. Meisenheimer ES, Rogers TS, Saguil A. Canadian syncope risk score: a validated risk stratification tool. Am Fam Physician 2021;103:375–376.
8. Saccilotto RT, Nickel CH, Bucher HC, Steyerberg EW, Bingisser R, Koller MT. San Francisco Syncope Rule to predict short-term serious outcomes: a systematic review. CMAJ 2011;183:E1116–E1126.
9. Shabbir MA, Saad Shaukat MH, Ehtesham M, Murawski S, Singh S, Alimohammad R. Bifascicular block in unexplained syncope is underrecognized and under-evaluated: a single-center audit of ESC guidelines adherence. PLoS One 2022;17:e0263727.
10. Santini M, Castro A, Giada F, et al. Prevention of syncope through permanent cardiac pacing in patients with bifascicular block and syncope of unexplained origin. Circ Arrhythm Electrophysiol 2013;6:101–107.
11. Gregoratos G, Abrams J, Epstein AE, et al. ACC/AHA/NASPE 2002 guideline update for implantation of cardiac pacemakers and antiarrhythmia devices: summary article. Circulation 2002;106:2145–2161.