Asian Journal of Research in Cardiovascular Diseases

Volume 5, Issue 1, Page 51-58, 2023; Article no.AJRCD.96177

Serie of Vagal-Mediated AV Block About 2 Cases

Haless Kamal [a], Tamir El Mehdi [a*] and Habbal Rachida [a]

[a] *Service of Cardiology, Ibn Rochd University Hospital, Morocco.*

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://www.sdiarticle5.com/review-history/96177

Case Study

Received: 04/03/2023
Accepted: 08/05/2023
Published: 22/05/2023

ABSTRACT

Vagally mediated atrioventricular (AV) block is defined as a paroxysmal AV block, localized within the AV node, associated with slowing of the sinus rate. All types of second-degree AV block, including pseudo-Mobitz II block, and complete AV block, may be present. In this article, we present a serie of 2 patients with recurrent syncope accompanied by transient second AV block at Ibn Rochd University Hospital in Casablanca – Morocco.

Our patient were a 30-year-old man and a 23 years old woman. They were referred for recurrent syncope which was not associated with any other symptoms nor special medical history. At their admission, the clinical examinations were generally normal, their paraclinical exams too, including ECG; echocardiography and biological data. In both of cases the Holter-ECG revealed transient AV block, and their explorations of autonomic nervous system showed major vagal hyperactivity. Effort test was performed too for searching AV block at effort, but they with no particularity. For treatment, we suggested a therapeutic plan composed of lifestyle modification and other measures. Pacing was not indicated considering the good improvement after two years of treatment and serious follow-up.

Physiological vagally hypertonia is frequent at young and athlets people. Vagally mediated atrioventricular (AV) block is defined as a paroxysmal AV block associated with slowing of the sinus rate secondary to a surge in parasympathetic activity. Syncope is a common symptom and it may

**Corresponding author: E-mail: mehdi.tamir@gmail.com;*

be associated with identifiable trigger. It may be asymptomatic as noticed on Holter recordings. Extrinsic vagal AVB also known as vasovagal syncope, which is typically treated with lifestyle modification, isotonic maneuvers, and rarely medication or pacemaker insertion.
Informed consent was obtained from all patients prior to their inclusion in this case report. All patients were informed of the case report's purpose. Measures were taken to protect the confidentiality and privacy of patients. We disclose no conflicts of interest that may have influenced the publication.

Keywords: Vagally AVB; syncope; rythmology; Holter ECG; AVB.

1. INTRODUCTION

A paroxysmal atrioventricular (AV) block that is localized to the AV node and slows the sinus rate is known as a vagally mediated AV block. There could be any kind of second-degree AV block, including complete AV block and pseudo-Mobitz II block. The vast majority of patients have typical AV conduction. Vagally mediated AV block is benign, it can be recorded as an asymptomatic or symptomatic event (syncope/pre- syncope). This type of AV block-related syncope has to be identified and treated as neurally mediated syncope.

Vagally mediated AV block has received little published research, and in clinical settings, it frequently goes undetected. The two examples in this study feature recurrent syncope and a transitory second AV block.

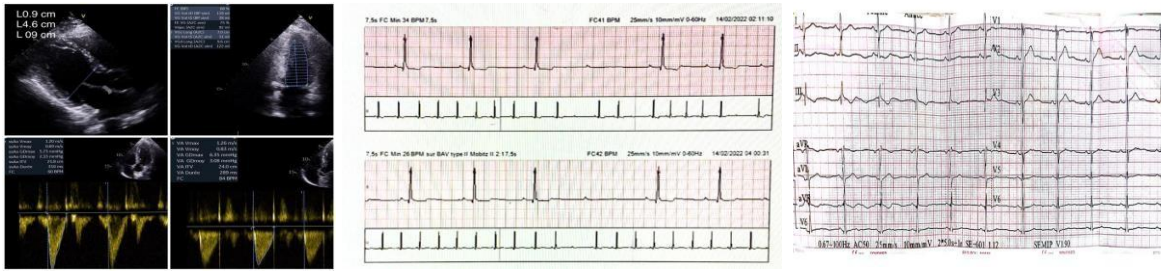
2. OBSERVATION

2.1 First Observation

A 30-year-old male with recurrent syncope was referred to our cardiology center. The patient had no known medical conditions, and neither his syncope nor any other symptoms or prodromal signs were observed. He was aware and hemodynamically stable upon admission. Overall, the physical examination was unremarkable. His biological data, echocardiogram (Figs. 1–3), and ECG were all in the normal range. His syncope's precise cause wasn't discovered until a Holter-ECG showed transient AV block (Fig. 2). Additionally, a routine exercise ECG was done to check for exercise AV block. After all, we investigated his autonomic nervous system, which revealed significant vagal hyperactivity. In fact, he is receiving attentive monitoring and ongoing Holter-ECG recordings that have demonstrated significant improvement as a result of our guidance on lifestyle modification and other measures.

2.2 Second Observation

A 23-year-old lady who had experienced syncope was brought to the emergency room. Similar episodes that occurred frequently but at erratic intervals were displayed by her. There were no transfusions, food or drug allergies, past traumas, or poisonings. She was born, grew, and developed normally. When she regained consciousness after the syncope event, she reported feeling weak, diplopia, and nauseous. She was awake and hemodynamically stable at admission. At the auscultation location of each valve, there was no cardiac murmur, tremor, or pericardial friction. Valsalva maneuvers and carotid sinus pressure responses were evaluated. Sinoatrial rhythm on the ECG was normal (Fig. 4). The results of the laboratory tests, echocardiogram, and brain MRI scan were all normal (Fig. 6). The 24-hour ECG revealed equal distances between the P-P interval and RR interval, P wave and QRS wave had a fixed connection for all of them, however for that one, Mobitz-II paroxysmal second-degree atrio-ventricular block was diagnosed (Fig. 5). Her autonomic profile displayed significant vagal hyperactivity, per the results of the autonomic nervous system test (Fig. 7). "We conducted an effort-test for searching an AV block at random effort." During the Valsalva maneuver, blood pressure and heart rate responses revealed a typical rise in blood pressure that became visible a few seconds after the maneuver started. We proposed a therapeutic program for the condition that included lifestyle changes, isotonic exercises, a salty diet, magnesium-rich vitamin therapy, compression stockings, and cardio-tonic (chlorhydrate etilefrine). Our rythmopole did not indicated pacing considering the good improvement after two years of treatment and well surveillance.



Figs. 1-3. Physical examination Echocardiography

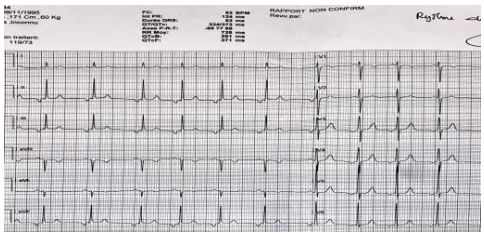


Fig. 4. Shows her basic ECG, it was normal with coronary sinus

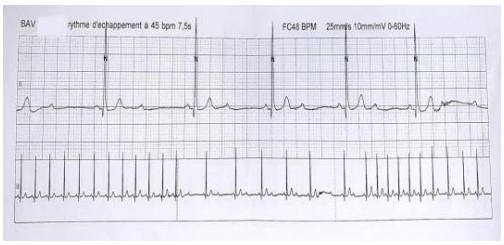


Fig. 5. Shows her normal echocardiography with EF estimated at 62%

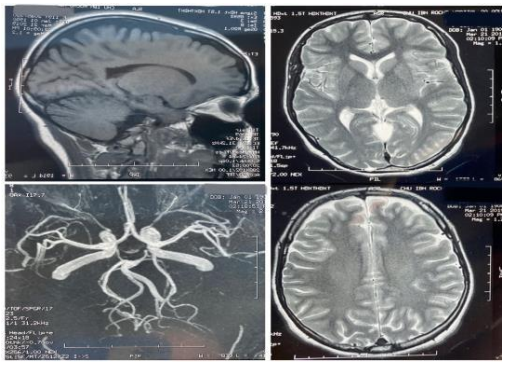


Fig. 6. Shows her normal Cerebral MRI

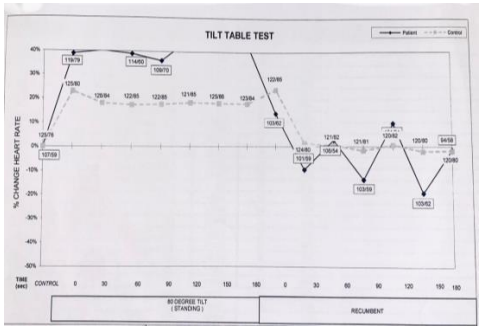


Fig. 7. Our goal of tilt table testing was Reproduction Symptoms and to correlate them with BP and HR values. Later we discussed the quite different parameters for identifying and quantifying orthostatic hypotension (OH, including immediate and delayed OH), diagnosing postural tachycardia syndrome (POTS), and provoking neurally mediated hypotension (NMH) or neurally mediated syncope (which includes vasovagal syncope). None of these were notable

3. DISCUSSION

A paroxysmal first, second, or third-degree AV block mediated by the vagally is characterized by a slowing of the sinus rhythm. Vagal input decreases sinus node function and AV nodal conduction but has no effect on the His-Purkinje system's conduction velocity. The results of certain electrophysiological investigations in which this type of AV block has been duplicated suggest that the site of vagally induced AV block is within the AV node [1,2,3]. The frequency of vagally induced second- or third- degree AV block is unknown. It is probably under-reported in the literature because of its low recognition and unpredictable nature.

Mobitz type II AV block is described as the occurrence of a single non- conducted P wave coupled with constant PR intervals before and after the blocked impulse, provided that there are at least two consecutive conducted P waves and the sinus rate (or the PP interval) is constant, according to definitions codified in 1978 by WHO [4] and the American College of Cardiology [5]. The delay that is surrounded by the blocked P wave must be two PP cycles long.

Purely functional and often benign, vagally mediated paroxysmal AV block invariably results from an increase in parasympathetic activity at the atrioventricular node [6]. Some cases of vagal overactivity have a clear explanation since it happens in conjunction with symptoms of increased vagal tone, such as vomiting, coughing, trouble swallowing, and hiccups, but this is not the case for the great majority of patients [1-3].

One of the physiopathological elements of vasovagal syncope is acute vagal hypertonia; in cardioinhibitory forms, it can cause an auriculoventricular block that causes a more or less prolonged asystole [7,8].

In susceptible persons, this cardiodepressive response might be seen during an activity test's recovery phase or a tilt test.

In 9% of cases on a systematic ECG, type I blocks of the first or second degree can be attributed to persistent vagal hypertonia, especially in athletes who are training hard. These asymptomatic blocks, which go away with exercise, should be regarded as physiological, similar to those seen in young people during the night [9,10].

The high activation of the parasympathetic nervous system is assumed to be the cause of transient atrioventricular blocks of varying degrees documented during left temporal epileptic convulsions, which may help to explain some abrupt epileptic fatalities caused by protracted asystole [11].

Secondary vagal hypertonia may be the source of nocturnal atrioventricular blocks in sleep apnea syndrome; many cases have been recorded [12], though much less frequently than sinus pauses.

A differential diagnosis must be made between an AV block that is vagally mediated and an intrinsic AV block, which is caused by anatomical involvement of AV conduction. The parasympathetic nervous system is likely engaged because sinus node function and AV nodal conduction are both simultaneously decreased. This shows that the block's mechanism is above rather than within the two nodes. Therefore, an intrinsic AV block is effectively ruled out when a second- or third-degree AV block and sinus slowing both exist at the same time. When there is only a modest increase in PP intervals, which is consistent with sinus arrhythmia, it may be challenging to differentiate between vagally mediated and intrinsic AV block. Some writers [13,14] believe that an increase in this interval of as small as 40 ms in the presence of paroxysmal AV block indicates that the block is vagal in origin.

One percent of all admissions to emergency rooms are due to syncope [15,16,17], which is a common symptom. The common underlying cause of syncope is a worldwide transitory cerebral hypoperfusion brought on by severe cardiac rhythm abnormalities (bradycardia or tachycardia) and/or severe hypotension [18]. While the electrocardiograms of our patients between episodes were normal, they experienced recurring syncopal episodes. An Holter-ECG revealed transitory AV block after which the precise cause of syncope was discovered. Before the assaults, the patients experienced episodes of weakness and nausea. It appears that syncope was caused by paroxysmal reflex AV block as a result.

About 40% of patients with recently-onset chronic AVB experience syncope as their primary concomitant symptom [19,20,21]. However, it's likely underreported [22] how common syncope brought on by paroxysmal AVB is. The diagnostic yield has grown recently as a result of more accessible long-term ECG monitoring technologies [23,24]. Vagally induced atrioventricular (AV) block may be asymptomatic, as seen on Holter recordings, especially at night, and may be linked to specific triggers like vomiting, micturition, vigorous coughing, or phlebotomy [6].

Some vagotonic responses, such as coughing [25] and swallowing [26]; Tomlinson and Fox, 1975 [27] Wik and Hillestad, [28] have been linked to sporadic occurrences of full heart block. According to Strasberg et al. [29], who observed two patients with recurring vagally induced full AV block without any precipitating events, reflex

vagally mediated paroxysmal AV block may constitute a distinct clinical condition.

In our second example, the carotid sinus massage caused a normal slowing of sinus node discharge without causing any AV block. Similar to how the sinus rate naturally slowed during the Valsalva maneuver, our patient's retching reflex failed to cause an AV block. It is well known that the effect of vagal stimulation depends on a number of variables, including the stimulation's intensity, mode, and resting sympathetic activity [30,31]. The correlation of nausea and vomiting with these episodes is noteworthy, despite the fact that it may be challenging to regulate all these variables in a laboratory setting to generate a spontaneous event.

Extrinsic vagal AVB (EV-AVB), also known as vasovagal syncope (or neuro- cardiogenic syncope), is typically treated with lifestyle changes, isotonic maneuvers, and rarely medication or pacemaker insertion [6], in contrast to intrinsic AVB (I-AVB) and extrinsic idiopathic paroxysmal atrioventricular block (EI-AVB), which are treated with pacemaker implantation [13,14].

We defined paroxysmal vagally mediated AV block (EV-AVB) as a syncope- causing entity. It is well known that some patients who experience unexplained syncope may benefit from pacemaker implantation [32], supporting the notion that these individuals have paroxysmal brady-arrhythmia. Even when a spontaneous asystolic reflex has been observed, cardiac pacing has a much decreased effectiveness in avoiding syncopal recurrences in individuals with paroxysmal vagally mediated AV block (EV-AVB). Studies that particularly looked into the impact of cardiac pacing in people with EV-AVB are lacking. In fact, individuals with vasovagal syncope, of which EV-AVB patients are a minority, have been included in all research on cardiac pacing. Therefore, the only conclusion that can be drawn is that the outcome of pacing in the EV-AVB subgroup is the same as that of the whole population. Dual-chamber cardiac pacing in patients with syncope and documentation of asystolic pause (either sinus arrest or AVB) by means of ECG monitoring was associated with a not infrequent recurrence rate of syncopal events (12-25% at 2-year follow-up) in the ISSUE 2, SUP 2, and ISSUE 3 trials [33,34,35]. When bradycardia is the cause of syncope, cardiac pacing is the most successful treatment; nevertheless, syncope may reoccur due to the coexistence of a vasodepressor reflex, which is present to some extent in almost all patients.

Researchers have looked into how theophylline treatment affects the prevention of syncopal recurrences. In a few small observational studies, patients with reflex syncope who received the drug saw a recurrence rate of between 12% and 22% [36,37,38].

According to the European recommendations on syncope [39], it is generally agreed that syncope caused by vagally mediated AV block should be identified and treated as neurally mediated syncope. We think that since vagally induced AV blocks are benign, pacemaker implantation is not necessary in patients who have them. Only the potential emergence of symptoms should be monitored for these patients [40]. After two years of follow-up and repeated Holter-ECG recordings, our patients who did not have pacemaker installation are doing well.

There is widespread agreement that vagally mediated AV block is benign even in the lack of strong evidence since it is localized within the AV node rather than the His-Purkinje system and, more importantly, because it is not a manifestation of anatomical involvement of AV conduction. Additionally, a vagally mediated death seems to be a rare occurrence and has never been proven in people with certainty [41]. However, vagally mediated AV block can result in syncope, which may come and go [42-44].

4. CONCUSION

Physically athletic young people frequently experience vagal hypertonia, especially at night. Vagally induced atrioventricular (AV) block is characterized as a paroxysmal AV block accompanied by sinus rate slowing as a result of an increase in parasympathetic activity. Syncope is a common symptom that can have obvious causes (such as vomiting or coughing uncontrollably), but it can also occur without any symptoms as seen on Holter recordings. Extrinsic vagal AVB, often referred to as vasovagal syncope, is frequently treated with dietary changes, isotonic exercises, and only rarely with medication or pacemaker implantation.

CONSENT

As per international standard or university standard, patient(s) written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

REFERENCES

1. Baron SC, Huang SK. Cough syncope presenting as Mobitz type II atrioventricular block. An electrophysiologic correlation. Pacing Clin Electrophysiol. 1987;10:65–9.
2. Nakagawa S, Koiwaya Y, Tanaka K. Vagally mediated paroxysmal atrio-ventricular block presenting as 'Mobitz type II' block (Letter). Pacing Clin Electrophysiol. 1988;11:471–2.
3. Kakuchi H, Sato N, Kawamura J. Swallow syncope associated with complete atrioventricular block and vasovagal syncope. Heart. 2000;83:702–4.
4. WHO/ISC Task Force. Definition of terms related to cardiac rhythm. Am Heart J. 1978;95:796–806.
5. Suravicz B, Uhley H, Borun R, et al. The quest for optimal standardization of terminology and interpretation. Am J Cardiol. 1978;41:130–45.
6. Bansal R, Mahajan A, Rathi C, Mehta A, Lokhandwala Y. What is the mechanism of paroxysmal atrioventricular block in a patient with recurrent syncope? J Arrhythmia. 2019;35:870–872.
7. Talwar KK, Edvarsson N, Varnaukas E. Paroxysmal vagally mediated AV block with recurrence syncope. Clin Cardiol. 1985;8:337-40.
8. Meyles I, Kapfansky E, Yahini JH, Hanne-Paparo N, Neufeld HN. Wenckebach A-V block: a frequent feature following heavy physical training. Am Heart J. 1974;90:426-30.
9. Brodsky M, Wu D, Denes P, Kanakis C, Rosen KM. Arrhythmias documented by 24 hour continuous electrocardiographic monitoring in 50 male medical students without apparent heart disease. Am J Cardiol. 1977;39:390-5.
10. Young D, Eisenberg R, Fish B, Fisher JD. Wenckebach A-V block (Mobitz I) in children and young adults. Am J Cardiol. 1977;40:393-9.
11. Altenmüller DM, Zehender M, Schulze-Bonhange A. High-grade atrioventricular block triggered by spontaneous and stimulationinduced epileptic activity in the left temporal lobe. Epilepsia. 2004;45:1640-4.
12. Lazarus A, Py A, Guerin F, Valty J, Le Heuzey JY. Arythmies et syndrome d'apnées obstructives du sommeil de l'adulte. Arch Mal Cœur. 1993;86:1753-9.
13. Lange HW, Ameisen O, Mack R, et al. Prevalence and clinical correlates of non-Wenckebach, narrow-complex second-degree atrioventricular block detected by ambulatory ECG. Am Heart J. 1988;115:114–20.
14. Barold SS, Hayes DL. Second degree atrioventricular block: a reappraisal. Mayo Clin Proc. 2001;76:44–57.
15. Blanc JJ, L'Her C, Touiza A, Garo B, L'Her E, Mansourati J. Prospective evaluation and outcome of patients admitted for syncope over a 1 year period. Eur Heart J. 2002;23: 767–8.
16. Farwell D, SulkeN. Howdowediagnose syncope?J Cardiovasc Electrophysiol 2002;13: 9–13.
17. Disertori M, Brignole M, Menozzi C, Raviele A, Rizzon P, Santini M, et al. Management of patients with syncope referred urgently to general hospitals. Europace. 2003;5: 283– 91.
18. Moya A, Sutton R, Ammirati F, Blanc JJ, Brignole M, Dahm JB et al. The Task Force for the Diagnosis and Management of Syncope of the European Society of Cardiology(ESC) Guidelines for the diagnosis and management of syncope (version 2009). Eur Heart J. 2009;30:2631–71.
19. Aste M., Oddone D., Donateo P. Syncope in patients paced for atrioventricular block. Europace. 2016;18:1735–1739.
20. Langenfeld H., Grimm W., Maisch B. Course of symptoms and spontaneous ECG in pacemaker patients: a 5-year follow-up study. Pacing Clin Electrophysiol. 1988;11:2198–2206.
21. Proclemer A., Ghidina M., Gregori D. Trend of the main clinical characteristics and pacing modality in patients treated by pacemaker: data from the Italian Pacemaker Registry for the quinquennium 2003–07. Europace. 2010;12:202– 209.

22. Lee S, Wellens JJ, Josephson M. Paroxysmal atrioventricular block. Heart Rhythm. 2009;6:1229–1234.
23. Brignole M, Vardas P, Hoffmann E. Indications for the use of diagnostic implantable and external ECG loop recorders. Europace. 2009;11:671–687.
24. Locati ET, Moya A., Oliveira M. External prolonged electrocardiogram monitoring in unexplained syncope and palpitations: results of the SYNARR- Flash study. Europace. 2016;18:1265–1272.
25. Hatt G, Oldershaw PJ, Cull RE, Humphrey P, Ward D: Syncope caused by coughinduced complete atrioventricular block. PACE. 1982;5:564.
26. Sapm RP, Griffiths PH, Guz A, Eisele J: Syncope on swallowing. Br Heart J. 1971.33;617.
27. Tomlinson TW, Fox KM: Cminoma of the oesophagus with “swallow syncope.” Br Med J. 1975;2:315.
28. Wik B, Hillestad L. Deglutition syncope. Br Med J. 1975;3:747.
29. Strasberg B, Palileo E, Bauernfeind R, Swiryn S, Lam W, Rosen K: Clinically significant reflexly mediated paroxysmal A-V nodal block (abstr.) Clin Res. 1981;29:244A.
30. De la Fuente D, Jedlicka T, Moe CK: Time course of vagal effects on S-A and A-V nodes (abstr.). Fed Proc. 1969;28:269(61).
31. Moore EN, Spear JF: Effect of autonomic activity on pacemaker function and conduction. In me Conduction System of the Heart. Structure, Function and Clinical Implications. (Eds. Wellens HJJ, Lie KI, Janse MJ). Leiden, He Stenfert Kroese BV. 1976;100.
32. Gulamhusein S, Naccarelli GV, KO PT, Prystowsky EN, Zipes DP, Barnett HJM, Heger JJ, Klein GJ: Value and limitations of clinical electrophysiologic study in assessment of patients with unexplained syncope. Am J Med. 1982;73:700.
33. Brignole M, Sutton R, Menozzi C. Early application of an implantable loop recorder allows effective specific therapy in patients with recurrent suspected neurally mediated syncope. Eur Heart J. 2006;27:1085–1092.
34. Brignole M, Ammirati F, Arabia F. Assessment of a standardized algorithm for cardiac pacing in older patients affected by severe unpredictable reflex syncopes. Eur Heart J. 2015;36:1529–1535 .
35. Brignole M., Donateo P., Tomaino M. Benefit of pacemaker therapy in patients with presumed neurally mediated syncope and documented asystole is greater when tilt test is negative: an analysis from the third International Study on Syncope of Uncertain Etiology (ISSUE-3) Circ Arrhythm Electrophysiol. 2014;7:10–16.
36. Šinkovec M, Grad A, Rakovec P. Role of endogenous adenosine in vaso-vagal syncope. Clin Auton Res. 2001;11:155–161
37. Benditt DG, Benson W, Kreitt J. Electrophysiologic effects of theophylline in young patients with recurrent symptomatic bradyarrhythmias. Am J Cardiol. 1983;52:1223– 1229.
38. Brignole M, Gaggioli G, Menozzi C. Adenosine-induced atrioventricular block in patients with unexplained syncope: the diagnostic value of ATP testing. Circulation. 1997;96:3921–3927.
39. Moya A, Sutton R, Ammirati F, et al. Guidelines for the diagnosis and management of syncope (version 2009). The Task Force for the Diagnosis and Management of Syncope of the European Society of Cardiology (ESC). Eur Heart J. 2009;30:2631–71.
40. Vardas PE, Auricchio A, Blanc JJ, et al. Guidelines for cardiac pacing and cardiac resynchronization therapy. The Task Force for Cardiac Pacing and Cardiac Resynchronization Therapy of the European Society of Cardiology. Developed in collaboration with the Heart Rhythm Association. Eur Heart J. 2007;28: 2256–95.
41. Alboni P, Alboni M, Gianfranchi L. Simultaneous occurrence of two independent vagal reflexes: a possible cause of vagal sudden death. Heart 2011;97:623–5.
42. A case report of paroxysmal complete atrioventricular block in a patient with dextrocardia and repaired tetralogy of Fallot. Eur Heart J Case Rep. 2022;6(11):ytac428. Published 2022 Nov 14. doi:10.1093/ehjcr/ytac428.
43. Subcutaneous implantable cardioverter-defibrillator update allows for unexpected diagnosis of paroxysmal atrioventricular block causing recurrent syncope. HeartRhythm Case Rep. 2022;9 (1):59-60. Published 2022 Nov 9. DOI:10.1016/j.hrcr.2022.10.017.
44. Rupture of the right sinus of Valsalva aneurysm and formation of ventricular

septal dissection and third-degree atrioventricular block: A case report. SAGE Open Med Case Rep. 2022;10:2050313X221140658. Published 2022 Dec 19. DOI:10.1177/2050313X221140658.

Peer-review history:
The peer review history for this paper can be accessed here:
https://www.sdiarticle5.com/review-history/96177