

Tachycardia-induced cardiomyopathy long after a pacemaker implantation for the treatment of unusual 2:1 atrioventricular block: What is the mechanism?



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Introduction

Device interrogation sometime provides useful information about previous arrhythmic events. We describe a patient with atrioventricular (AV) block in whom a pacemaker interrogation found the unpredictable cause of pleural effusion.

Case summary

A 49-year-old man experienced syncope while he crossed a street, and he was transferred to our hospital. An electrocardiogram (ECG) showed a second-degree 2:1 atrioventricular block with narrow QRS complexes (Figure 1A). Curiously, the RR intervals were not constant while the PP intervals remained steady during the 2:1 AV block (Figure 1B). The echocardiogram revealed a normal left ventricular systolic function and no morphologic abnormalities. No significant coronary stenosis was observed on the coronary angiogram. No electrolyte abnormalities were noted. He did not have any relevant medical history and did not take any medications. Because the 2:1 AV block did not improve, a dual-chamber pacemaker (Advisa DR MRI A3DR01, Medtronic, Minneapolis, MN) was implanted.

The alternating RR intervals during the 2:1 AV block seen in the present case were uncommon. A quite similar ECG finding was however previously reported.¹ The authors offered the concealed conduction concept as the causal mechanism.² The term “concealed conduction” is used when a proximal atrial impulse penetrates the AV node but fails to

traverse it completely. The concealed penetration into the AV node is inferred by its aftereffects on the propagation of succeeding impulses. The authors specifically described the phenomenon as follows. When blocked atrial impulses penetrate deeper into the AV junction, the subsequent PR intervals are long, while the impulses blocked at its shallow level are followed by shorter PR intervals. Importantly, they stated that when 2 levels of concealment are postulated in an ECG showing normal QRS complexes, the conduction disturbance is most probably confined within the AV node. Thus, the interesting phenomenon we observed suggested that our patient may have had intranodal block rather than infranodal block. The former is generally considered more benign than the latter.³ The current guidelines,³ however, clearly state that permanent pacemaker implantation is indicated for second-degree AV block with associated symptomatic bradycardia, regardless of the site of the block. His pacemaker implantation was based on this guideline.

One year later, the patient saw his family physician for dyspnea on effort. A chest radiograph demonstrated a pleural effusion. He was prescribed 20 mg of furosemide. He had a quick improvement in his symptom, and the pleural effusion was no longer seen 2 weeks after his initial visit. The furosemide was then withdrawn. He never experienced the recurrence of the symptom. Another 8 months later, he was seen in our outpatient clinic for a regular pacemaker check.

There are a wide variety of diseases that can result in pleural effusion.⁴ However, given that only a brief administration of the oral diuretic cured his pleural effusion without recurrence, any chronic disease or disease resulting in irreversible damage was unlikely. Anyway, he had never presented with the symptom until he was implanted with the pacemaker. Therefore, it was essential to check for any pacemaker-related defects, such as pacemaker failure, lead perforations, or heart failure due to chronic right ventricular pacing.

The echocardiogram findings and lead positions remained the same, and no pacemaker failure was noted. Instead, a number of atrial fibrillation (AF) or atrial tachycardia (AT) events were recorded by the pacemaker (Figure 2A). During

KEYWORDS Atrioventricular block; Autonomic tone; Tachycardia-induced cardiomyopathy

ABBREVIATIONS AF = atrial fibrillation; AT = atrial tachycardia; AV = atrioventricular; ECG = electrocardiogram (Heart Rhythm Case Reports 2016;2:107–110)

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KEY TEACHING POINTS

- Alternations of the ventricular cycle length could be observed even during 2:1 second-degree atrioventricular (AV) block, and this observation may be explained by different depths at which blocked impulses penetrate into the AV junction.
- Tachycardia-induced cardiomyopathy could be one of the causes of decompensated heart failure, even in patients with AV block needing a pacemaker.
- Interrogation of pacemakers often provides some clues about the cause of previous cardiac events.

the AT/AF, the pacemaker mode was automatically switched from DDD to DDIR. An extremely rapid ventricular response was noted during some AT/AF events, in particular soon after the beginning of the AT/AF events (Figure 2B). A cardiac compass report (Figure 2C) showed that the AT/AF persisted for about 6½ months and terminated spontaneously. A medical interview with the patient proved that his dyspnea coincided with the first occurrences of the AT/AF events. According to the records of the thoracic impedance in his pacemaker, it markedly decreased also soon after the beginning of the AT/AF events (Figure 2D).

A double-chamber tachycardia was carefully ruled out via device interrogation. It was therefore assumed that the patient developed tachycardia-induced cardiomyopathy secondary to a rapid AT/AF, resulting in acute decompensated heart failure.⁵ He obviously had symptomatic second-degree AV block requiring a pacemaker 20 months before the last clinical visit. Thus, the rapid ventricular response during the AT/AF was very unlikely if his AV conductivity remained severely impaired. Prior to the pacemaker implantation, we carefully excluded any potential reversible causes of AV block, such as drug toxicity, electrolyte abnormalities, diseases with peritricioventricular node inflammation, transient injury or ischemia of the conduction system, or hyper-vagotonia.³ It is known that AV block could be reversed transiently in patients with cardiac sarcoidosis.³ However, the findings from his multiple echocardiographic examinations, ECGs, and device interrogations never suggested cardiac sarcoidosis.⁶ Did his AV block then heal spontaneously?

The patient presented with sinus rhythm at 68 beats per minute and a prolonged PQ interval of 270 milliseconds when the sensed AV delay of the pacemaker was changed from 150 to 300 milliseconds. The Wenckebach cycle length was determined to be 720 milliseconds (Figure 3), and the AV nodal effective refractory period was found to be 620 milliseconds. He was prescribed 5 mg of bisoprolol.

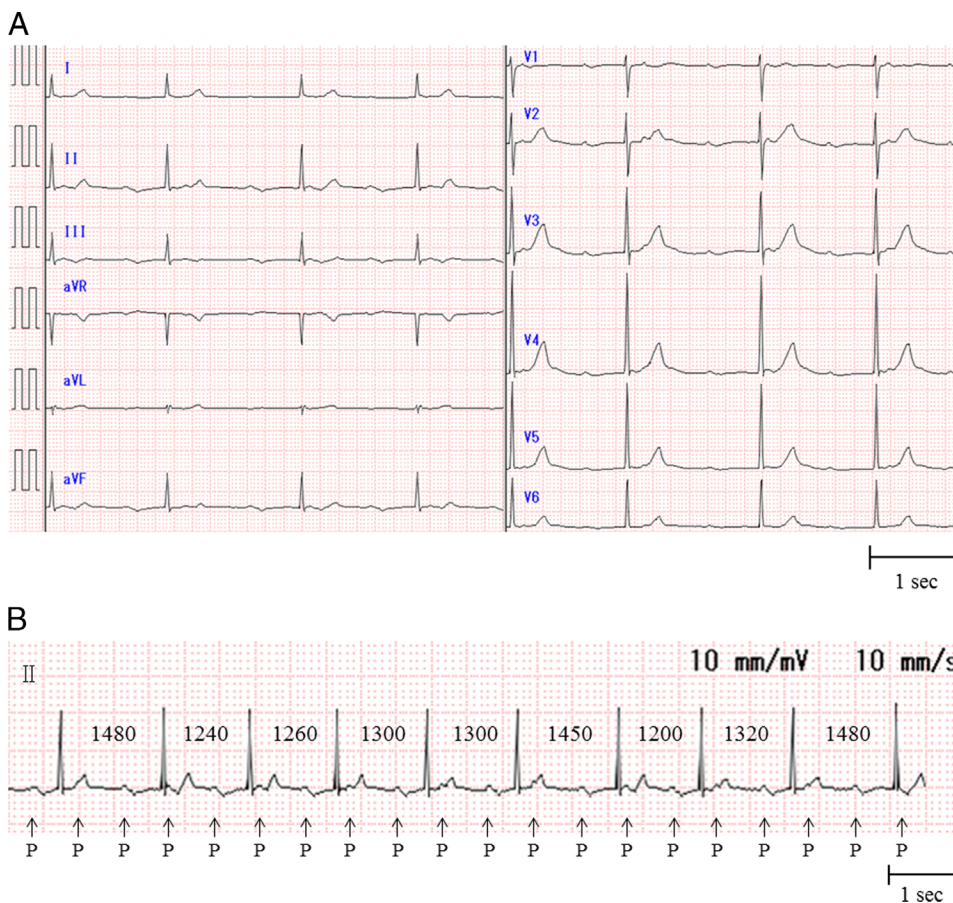


Figure 1 A: An electrocardiogram recorded at the patient’s initial clinical visit. A 2:1 atrioventricular block was noted. B: A long-duration recording of an electrocardiogram at the initial clinical visit. The RR intervals were irregular during 2:1 atrioventricular block.

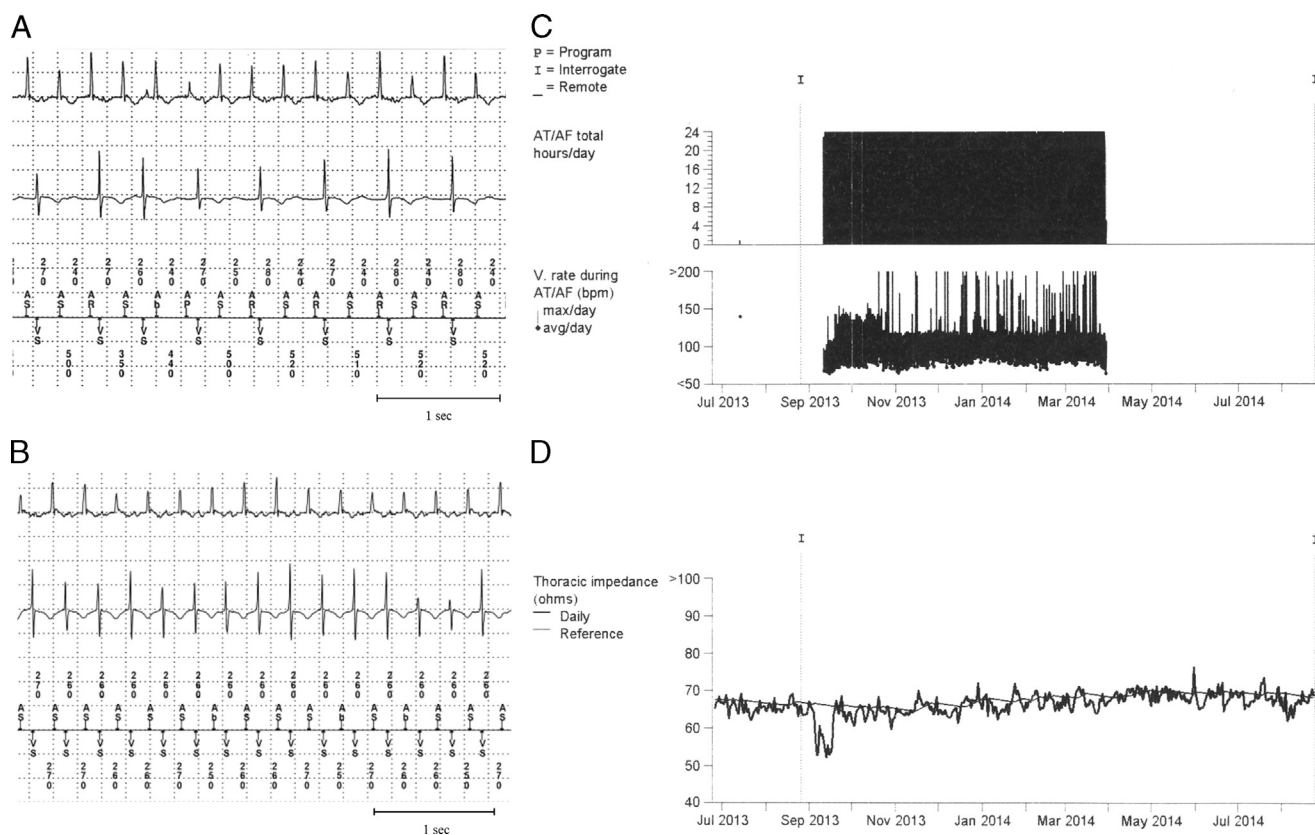


Figure 2 **A:** A device interrogation illustrating an event of an atrial arrhythmia with a cycle length of 240–280 milliseconds and irregular atrioventricular conductions. **B:** A device interrogation illustrating an event of an atrial arrhythmia with 1:1 atrioventricular conductions. **C:** A summary of the duration of the atrial tachycardia (AT)/atrial fibrillation (AF) burden and average or maximum heart rate during the AT/AF. **D:** The time course of the thoracic impedance.

Although his third-degree AV block improved to first-degree AV block 20 months after the pacemaker implantation, the AV conductivity at rest still remained morbidly impaired. Why, then, did such a rapid ventricular response occur during AT/AF?

Commentary

The following is a putative mechanism for the observed phenomenon. An increased sympathetic activity with or without decreased parasympathetic tone elicited the AT/AF.⁷ Alternatively, the AT/AF itself activated the



Figure 3 An intracardiac recording during atrial pacing. Wenckebach periodicity was seen at an atrial pacing cycle of 720 milliseconds.

sympathetic system.⁸ This autonomic change also improved the patient's deteriorated AV conduction. The excessively increased ventricular rate and atrioventricular asynchrony in turn impaired the diastolic filling, resulting in a reduction in the cardiac output.⁹ This occurrence further increased the sympathetic activity. The patient fell into this vicious cycle. Because he presented with first-degree AV block at the last visit, his site of block was likely to be within the AV node.¹⁰ This hypothesis was actually consistent with our expectation before the pacemaker implantation. As is well known, the AV node is strongly influenced by the autonomic system.¹¹ Therefore, if the hypothesis is correct, the dramatic change in the autonomic tone was possibly a key mechanism whereby the deteriorated AV conductivity strikingly and even harmfully improved.

The important limitations were that we did not assess his AV conduction properties with the use of isoproterenol, atropine, or exercise testing, and we did not record a His bundle ECG. Finally, it was possible that antiarrhythmic drugs such as amiodarone were more appropriate than bisoprolol for the secondary prevention of his harmful tachycardia.

References

1. Watanabe Y, Dreifus LS. Levels of concealment in second degree and advanced second degree A-V block. *Am Heart J* 1972;84(3):330–347.
2. Langendorf R. Concealed A-V conduction; the effect of blocked impulses on the formation and conduction of subsequent impulses. *Am Heart J* 1948;35(4):542–552.
3. Epstein AE, DiMarco JP, Ellenbogen KA, et al. with the American College of Cardiology Foundation, American Heart Association Task Force on Practice Guidelines, Heart Rhythm Society. 2012 ACCF/AHA/HRS focused update incorporated into the ACCF/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society. *Circulation* 2013;127(3):e283–e352.
4. Porcel JM, Light RW. Diagnostic approach to pleural effusion in adults. *Am Fam Physician* 2006;73(7):1211–1220.
5. Sairaku A, Nakano Y, Oda N, Uchimura Y, Tokuyama T, Kawazoe H, Fujiwara M, Watanabe Y, Matsumura H, Kihara Y. Incomplete cure of tachycardia-induced cardiomyopathy secondary to rapid atrial fibrillation by heart rate control without sinus conversion. *J Cardiovasc Electrophysiol* 2014;25(10):1037–1043.
6. Soejima K, Yada H. The work-up and management of patients with apparent or subclinical cardiac sarcoidosis: with emphasis on the associated heart rhythm abnormalities. *J Cardiovasc Electrophysiol* 2009;20(5):578–583.
7. Chen PS, Chen LS, Fishbein MC, Lin SF, Nattel S. Role of the autonomic nervous system in atrial fibrillation: pathophysiology and therapy. *Circ Res* 2014;114(9):1500–1515.
8. Wasmund SL, Li JM, Page RL, Joglar JA, Kowal RC, Smith ML, Hamdan MH. Effect of atrial fibrillation and an irregular ventricular response on sympathetic nerve activity in human subjects. *Circulation* 2003;107(15):2011–2015.
9. Maisel WH, Stevenson LW. Atrial fibrillation in heart failure: epidemiology, pathophysiology, and rationale for therapy. *Am J Cardiol* 2003;91(6A):2D–8D.
10. Childers R. The AV node: normal and abnormal physiology. *Prog Cardiovasc Dis* 1977;19(5):361–384.
11. Damato AN, Lau SH, Helfant R, Stein E, Patton RD, Scherlag BJ, Berkowitz WD. A study of heart block in man using His bundle recordings. *Circulation* 1969;39(3):297–305.