

# 2:1 Pulsus and electrical alternans during atrioventricular reciprocating tachycardia in a healthy young man: A case report



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## Introduction

Pulsus alternans (PA) is a hemodynamic phenomenon characterized by a beat-to-beat alternation in pulse amplitude that results in a clinically appreciable pulse rate half of the electrical rate. It was first described in 1872 by Traube<sup>1</sup> and many theories have been postulated regarding its pathophysiology. PA is usually associated with severe heart disease and can eventually result in pulseless electrical activity.<sup>2-4</sup> It has rarely been described in healthy individuals. In contrast, electrical alternans (EA) is defined by alternating QRS amplitudes on an electrocardiogram. It is a benign process that often occurs in the setting of fast supraventricular tachycardias<sup>5</sup> and is usually frequency-dependent. Another possible etiology for EA is pericardial effusion. The interaction between mechanical alternans and EA is unclear, mainly owing to the limited number of studies that have included simultaneous recordings of both types of alternans. Different mechanisms may provoke both PA and EA, the former mainly because of a hemodynamic and inotropic impairment, and the latter owing to local disorders of the electrical impulse conduction.<sup>5-7</sup> Herein we report a rare case of a 22-year-old male patient with a structurally normal heart who presented to an emergency department with recurrent palpitations and dyspnea. Electrocardiogram, echocardiogram, and electrophysiological study (EPS) demonstrated a supraventricular tachycardia (SVT) due to a left accessory pathway (AP) and a 2:1 mechanical and electrical alternans.

**KEYWORDS** Atrioventricular reciprocating tachycardia; Accessory pathway; Electrical alternans; Pulsus alternans; Supraventricular tachycardia; Tachycardiomyopathy  
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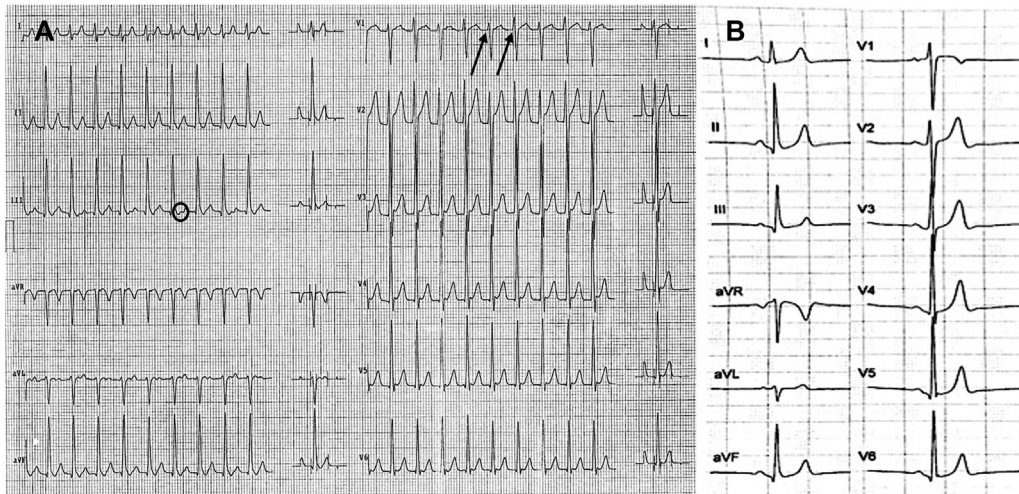
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## KEY TEACHING POINTS

- Pulsus alternans is often indicative of severe left ventricular systolic impairment and poor prognosis.
- Electrical alternans is a frequency-dependent benign process that usually occurs during supraventricular tachycardias.
- Occurrence of both pulsus and electrical alternans in healthy people with a normal heart is rare.
- Ablation of the arrhythmic substrate could be a definite therapy, probably preventing the occurrence of a tachycardiomyopathy.

## Case report

The patient was well until around 18 years old, when he developed recurrent palpitations. He was initially managed with metoprolol at a dosage of 50 mg twice a day and was doing well until the age of 22 years, when he developed an hour of persistent palpitations and dyspnea. He presented to the emergency department, where an electrocardiogram revealed a narrow QRS tachycardia (Figure 1) with 165 beats per minute (bpm). The QRS amplitude varied beat-to-beat. On exam, he had a blood pressure of 100/60 mm Hg and a pulse rate of 75 bpm measured by contemporary cardiac auscultation and peripheral pulse inspection. An S3 sound was also appreciated. Routine laboratory tests including blood cell counts; cardiac, renal, and hepatic function tests; troponin; and D-dimer tests were all normal. Chest radiography was unremarkable. A 2-dimensional echocardiogram documented a 2:1 mechanical impairment of the left ventricle with a 2:1 loss of systolic function (mechanical activity rate at 75 bpm, electrical activity rate at 150 bpm). A parasternal long-axis with M-mode view showed a 2:1 opening of the aortic valve, a 2:1 incomplete opening of the mitral valve

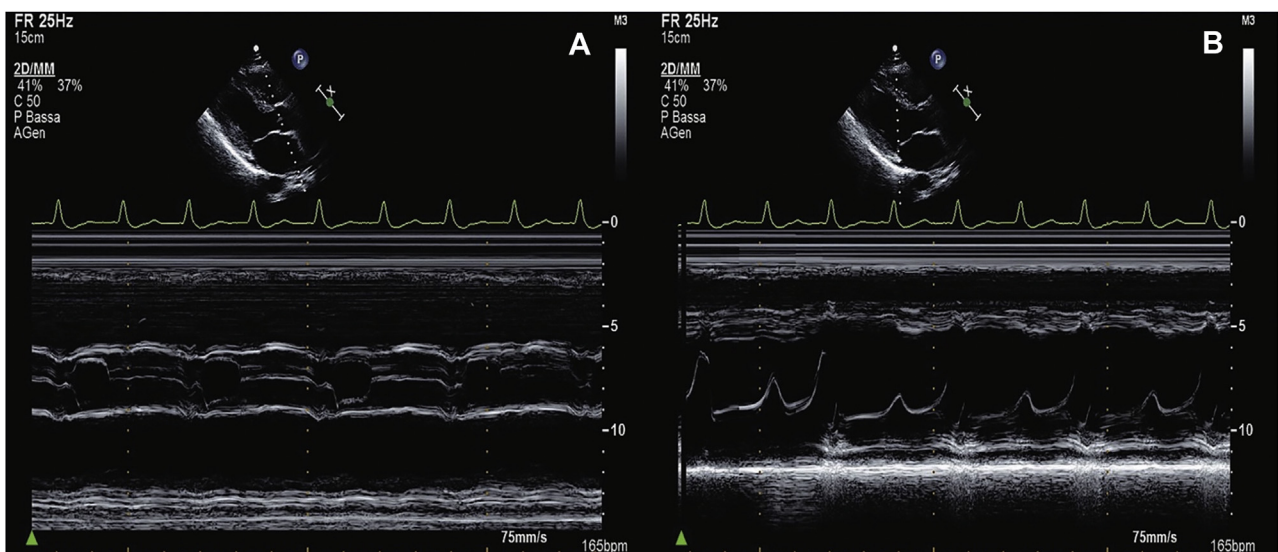


**Figure 1** A: A 12-lead electrocardiogram of the patient showing a narrow QRS tachycardia at a heart rate of 165 beats/min and retrograde p waves (circle), best seen in inferior limb leads. Electrical alternans, namely the beat-to-beat alternation of the QRS amplitude, can best be appreciated in lead V<sub>1</sub> (arrows). B: A 12-lead electrocardiogram of the patient at sinus rhythm. Retrograde p waves are no longer seen.

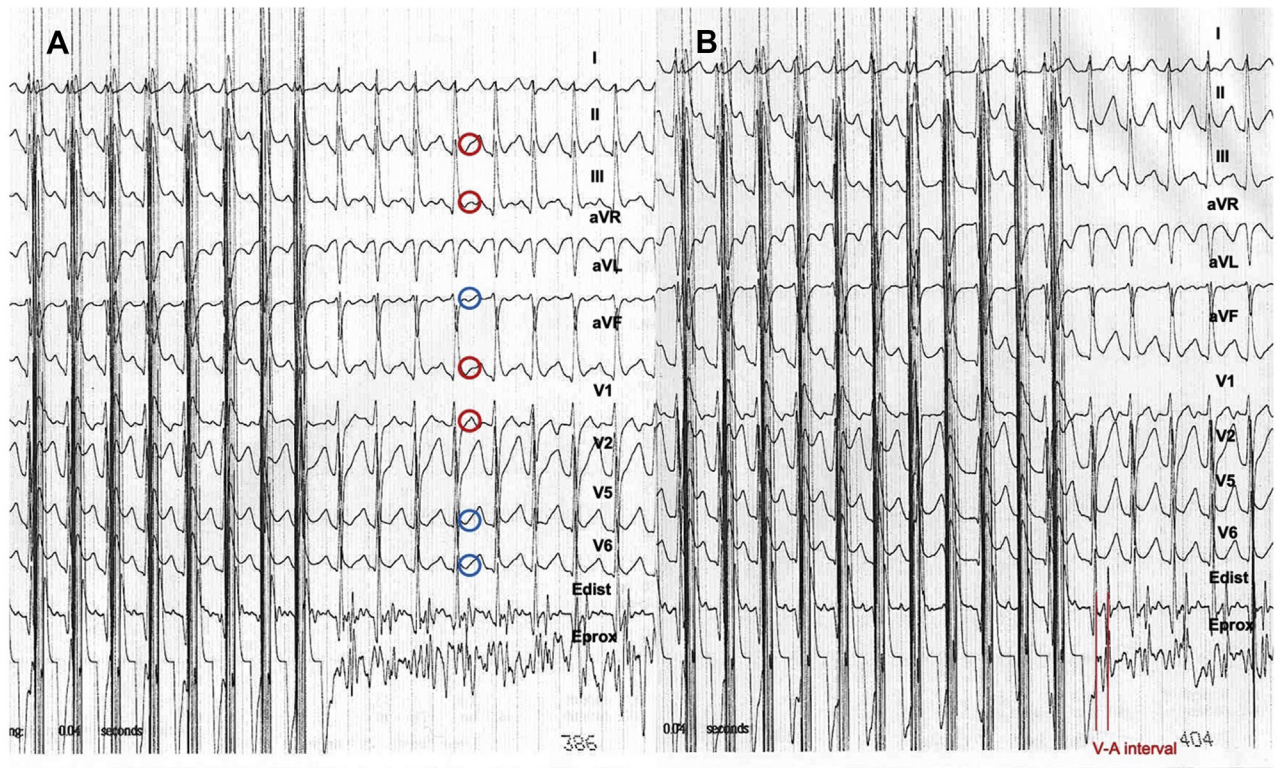
(Figure 2, Supplemental Video 1, Supplemental Video 2), and a 2:1 impairment of the ventricular thickening. The tachycardia was then interrupted by intravenous administration of 6 mg of adenosine. An echocardiogram performed after normal sinus rhythm was restored showed a structurally normal heart with a global normal ventricular and valvular function. The patient underwent a transesophageal EPS where the same arrhythmia was easily induced and soon after interrupted by overdrive pacing (Figure 3). Every time the arrhythmia was induced, the patient again experienced palpitations and dyspnea. The patient subsequently completed a transvenous EPS, where a left AP was found and ablated. The patient remained symptom free, with no recurrent palpitations and normal biventricular function on serial echocardiogram evaluations, after 8 years of follow-up.

## Discussion

A persistent but reversible form of PA can be found in tachycardiomyopathies in the setting of persistent and pathologically elevated heart rates.<sup>6,8</sup> A paroxysmal acute form of both PA and EA, as described in this case, appears to be rare. One similar case was reported in 1997 by Lu and colleagues,<sup>9</sup> but, differently from our case, the 27-year-old healthy patient had an incessant atrial tachycardia with EA and PA. He also recovered completely after ablation. Our patient's echocardiogram performed in sinus rhythm showed a normal global heart function. The question is why a healthy man with a normal heart could develop this abnormal event during a common SVT. Cardiac alternans can be both electrical and mechanical. EA manifests as a benign tachycardia-dependent beat-to-beat change in the QRS amplitude. PA, also referred to as mechanical alternans, is defined as a blood



**Figure 2** Parasternal long-axis with M-mode view at 2-dimensional echocardiogram. A: A 2:1 complete opening of the aortic valve can be seen. B: Mitral valve with a 2:1 incomplete opening and a subsequent inadequate ventricular filling.



**Figure 3** Surface and endocavitary tracings during transesophageal electrophysiological study (paper speed 25 mm/s). **A:** Supraventricular tachycardia (SVT) induction with retrograde p waves seen after the QRS complex (*circles*). Positive retrograde p waves in leads II, III, aVF, and V<sub>1</sub> (*red circles*) and negative retrograde p waves in leads V<sub>5</sub>, V<sub>6</sub>, and aVL (*blue circles*) are consistent with an orthodromic reciprocating tachycardia (ORT) using a left lateral accessory pathway. **B:** A ventriculoatrial interval of 100 ms (*red lines*) during SVT suggests ORT.

pressure change occurring on an every-other-beat basis, which can manifest in a succession of strong and weak pulses. The pathophysiology of PA in systolic dysfunction is attributed to 2 major mechanisms: Frank-Starling relationship and impaired ventricular contractility and calcium cycling. The first theory postulates that the alternation of strong and weak pulses relates to a variation of both end-diastolic and end-systolic volumes and to different diastolic filling periods.<sup>10</sup> In our patient the stronger beat was preceded by a greater end-diastolic volume after a longer filling period. This was followed by a lower end-systolic volume, leading to subsequent shorter filling period, lower end-diastolic volume, and weaker beat. The SVT itself could also affect the ventricular diastolic phase with both a defective left ventricular relaxation and a shortened diastolic filling time. Another model suggests that beat-to-beat changes in the inotropic state are due to impaired calcium metabolism within cardiac myocytes.<sup>11</sup> Mouse model experiments demonstrate that an overexpression of calsequestrin in the sarcoplasmic reticulum can impair contractility and result in PA.<sup>4</sup> The patient's intrinsic ventricular contractile state indeed could have some role as a contributing mechanism, as the stronger beat can be related to an augmentation of the contractile state regardless of the muscle length.<sup>11</sup> It is worth noting that there may be yet unexplained mechanisms for this patient's presentation. We cannot exclude a genetic or acquired pathology of the cardiac fibers that will manifest in the future. However, no sign of

cardiac dysfunction has emerged in the 8 years of follow-up and ablation appears to be curative.

## Conclusion

Here we report the first case of an acute 2:1 PA and EA during atrioventricular reciprocating tachycardia due to a left-sided AP and propose potential underlying mechanisms. The cause of this rare event remains uncertain.

## Limitation of the case report

The transvenous EPS that documented the atrioventricular reciprocating tachycardia using a left AP and excluded atrial tachycardia was performed in a highly specialized tertiary center; thus only the final report of the procedure is available, while endocavitary tracings are missing.

## Appendix Supplementary data

Supplementary data associated with this article can be found in the online version at <https://doi.org/10.1016/j.hrcr.2021.11.006>.

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