Orthostatic changes in AV node conduction causing dizziness during paroxysmal atrial tachycardia

S.C. Agarwal*, C.B. Pepper

Department of Cardiology, Leeds General Infirmary, Leeds, UK

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Abstract
Dizziness in the upright posture is a common reason to seek medical advice and is often dismissed as benign orthostatic hypotension. We present an unusual case of dizziness in the upright posture due to possible changes in atrioventricular nodal conduction during atrial tachycardia.

Case report
A 60-year-old woman presented to the emergency department with a history of palpitation and dizziness when standing, relieved only by lying flat. She had previously seen a number of physicians with similar symptoms over a six year period. Her symptoms were intermittent and would last from a few minutes to a few hours. Full endocrinological assessment had been normal and a diagnosis of POTS (postural orthostatic tachycardia syndrome) had been made after it was noticed that she had a significant increase in heart rate during a tilt test.

There was no history of chest pain or shortness of breath. She was asymptomatic between the episodes and gave no history of any vasomotor symptoms during or between the episodes. There was no clinical evidence of structural heart disease. On investigation she had normal full blood count, electrolytes, thyroid function test and echocardiogram with a left ventricular end diastolic diameter of 4.8 cm, left atrial diameter of 3.5 cm, posterior wall thickness of 1.1 cm and estimated ejection fraction of 55–60%.

Her admission ECG showed a heart rate of 100 beats per minute (Fig. 1) when recumbent, rising to 200 beats per minute on attaining upright posture (Fig. 2). This was associated with dizziness and a fall in blood pressure to 60 mmHg. The ECG was initially interpreted as showing sinus tachycardia; however, close observation revealed a
second P wave hidden in the tail of the QRS suggesting a diagnosis of atrial tachycardia with 2:1 AV nodal block (Fig. 1). The 2:1 conduction of the P wave across the AV node when supine changed to 1:1 conduction when upright (Fig. 2) resulting in haemodynamic compromise and dizziness. The administration of adenosine in the upright posture did not restore sinus rhythm and revealed a P wave rate of 200 beats per minute (Fig. 3).

She was treated with beta blockers and flecaicnide to which she has responded very well with only very brief and infrequent episodes of postural dizziness. A further 24 h Holter monitor showed infrequent episodes of paroxysmal atrial tachycardia with 2:1 conduction of the P waves with no episodes of 1:1 conduction. The patient was offered an electrophysiological study with a view to ablation of the tachycardia focus: as she is free of symptoms she wished to continue with medical management.

**Discussion**

We describe a patient suffering from paroxysmal atrial tachycardia. During these episodes the P wave conduction ratio changed from 2:1 in the supine position to 1:1 in the upright posture, leading to increased ventricular rate and haemodynamic compromise which caused postural dizziness.

AV nodal conduction during high atrial rates is dependent upon the refractory period of the AV node. Autonomic changes on attaining the upright posture may result in a significant decrease in refractoriness compared with recumbency. The upright posture (compared with supine) significantly
augments sinoatrial, interatrial and atrioventricular conduction, shortens the sinus node recovery time and decreases the effective refractory periods of the right atrium, atrioventricular node and right ventricle [1,2]. Waxman et al. [3] reported termination of AV node re-entry tachycardia in the supine posture due to an increase in systemic arterial pressure and the baroreceptor reflex resulting in heightened vagal tone. Abboud et al. [4] suggested that upright posture decreases blood pressure, which reflexly increases sympathetic tone and reduces vagal tone. By a similar mechanism, patients with atrial flutter can have increased symptoms on exercise due to an increase in ventricular rate [5]. Exercise can not only increase AV node conduction in atrial flutter but also increase the flutter rate. We postulate that in our patient there was a change in the autonomic control of the AV node leading to decreased effective refractory period in the upright posture, compared with the supine posture, with 1:1 conduction of the P waves.

Since commencing beta blockers and flecainide, her symptoms have dramatically improved and she has only occasionally had an episode of lightheadedness despite ongoing episodes of paroxysmal atrial tachycardia. We attribute this to adequate AV nodal blockade preventing orthostatically mediated high ventricular rates.

References