

# A unique case of pulmonary embolism presenting as a paroxysmal atrial tachycardia instigated only by recumbency and stooping



Nagesh Chopra, MD

From the OhioHealth Heart & Vascular Physicians, Section of Cardiac Electrophysiology, Department of Cardiology, Riverside Methodist Hospital, Columbus, Ohio.

## Introduction

Atrial tachyarrhythmias occur in ~4%–14% of patients presenting with pulmonary embolism (PE),<sup>1,2</sup> sinus tachycardia being the most common, followed by atrial tachycardia (AT), atrial fibrillation, and atrial flutter, in that order.<sup>1–3</sup> Although AT has been described with PE, its exclusive occurrence with changes in body posture after PE has not been previously reported. In fact, atrial arrhythmia occurring *de novo* with changes of posture in itself is a rare phenomenon, except perhaps in the setting of positional irritation from a venous catheter in the right atrium<sup>4</sup> or possibly sympathetic surge from baroreceptor reflex.<sup>5</sup>

Here we describe an unusual case of paroxysmal AT occurring only when lying down or bending, in an otherwise healthy female subject presenting with an acute PE.

## Case report

A 36-year-old white woman with no significant comorbidities presented with a 4-day history of intermittent palpitations and mild dyspnea. Palpitations were positional and occurred *only* when recumbent or bending forward; none occurred when sitting, standing, or walking. The palpitations were initially sustained but when clinically evaluated had become paroxysmal, albeit still positional. There was no history of chest pain, syncope/presyncope, cough, or hemoptysis. There was no recent history of long-distance travel, trauma, childbirth, or abortion.

The patient had been taking Loryna (drospirenone/ethinyl estradiol), a daily oral contraceptive pill, for the last 7 months and only recently switched to Microgestin (ethinyl estradiol/norethindrone) 2 days prior to admission. She did not smoke

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**Address reprint requests and correspondence:** Dr Nagesh Chopra, OhioHealth Heart & Vascular Physicians, Department of Cardiology, Section of Cardiac Electrophysiology, Riverside Methodist Hospital, 3535 Olentangy River Road, Columbus, OH 43214. E-mail address: nagesh.chopra@ohiohealth.com.

or use alcohol or illicit drugs. Her father developed unprovoked deep vein thrombosis at 60 years of age, for which he was on warfarin anticoagulation.

Her vital signs including oxygen saturation at room air were normal except for mild sinus tachycardia (90–110 beats/min). Her systemic examination was unremarkable.

A complete blood count and complete metabolic profile were within normal range. An elevated D-dimer at 0.56 micrograms/milliliter (normal range 0.22–0.49 micrograms/milliliter) prompted a computed tomography pulmonary angiogram, which revealed a left lower lobe subsegmental pulmonary embolus with no other acute cardiopulmonary findings (Figure 1). A transthoracic echocardiogram showed a structurally normal heart and a normal left ventricular ejection fraction without evidence of right heart strain.

The salient and consistently reproducible telemetry finding was of short paroxysms of AT when recumbent or bending forward (Figure 2). To further investigate this phenomenon, a continuous 12-lead electrocardiogram monitoring was performed during these maneuvers (Figure 3). These maneuvers consistently and reliably induced paroxysmal AT at 140 beats per minute associated with palpitations.

In light of a recent PE and continued clinical improvement, the patient was treated conservatively. Subcutaneous enoxaparin was initiated as a bridge to therapeutic warfarin anticoagulation with an international normalized ratio aim of 2–3. Metoprolol 25 mg orally twice daily was initiated to suppress the AT. The oral contraceptive was stopped. She was uneventfully discharged on warfarin and metoprolol and within 2 weeks of discharge, her symptoms of postural palpitations resolved completely. A limited hypercoagulable workup has been negative to date.

## Discussion

Despite its protean presentation, acute PE has never been associated with a “postural” AT. Here we describe an unusual case of a 36-year-old woman who presented with a PE and episodic palpitations due to paroxysms of AT

### KEY TEACHING POINTS

- Palpitations due to atrial tachycardia initiated by a recumbent or a stooping posture is a rare symptom that poses a diagnostic challenge to the clinician.
- Atrial tachycardias are commonly encountered in patients presenting with acute pulmonary embolism.
- Acute pulmonary embolism should be ruled out if atrial tachycardia instigated by positional body changes is clinically encountered.

instigated *only* by recumbency or a stooping posture. This association has not been previously described.

Body posture has significant effects on atrioventricular (AV) nodal conduction, likely mediated via modulation of the autonomic nervous system.<sup>6</sup> Accordingly, upright posture enhances AV nodal conduction and facilitates AV nodal reentry<sup>7,8</sup> or enhances AV conduction during AT.<sup>9</sup> Furthermore, a dependent body posture can abort an AV nodal-dependent supraventricular tachycardia (SVT),<sup>10</sup> akin to a Valsalva maneuver. Although sitting or standing can instigate or worsen a paroxysmal SVT,<sup>5,11</sup> assumption of a supine position instigating a paroxysmal SVT is exceedingly rare.<sup>12</sup>

In our patient, clinical findings during SVT most consistent with AT include (1) abrupt onset without preceding PR prolongation, (2) “warming up” with shortening of the R-R interval as the tachycardia progresses, (3) termination of

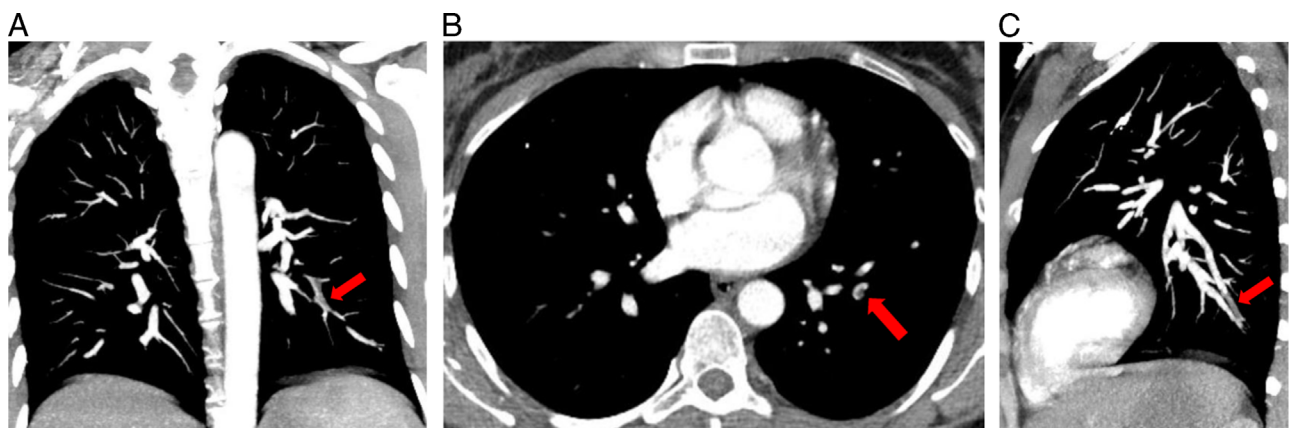
the SVT without AV block (with a QRS), and (4) isolated premature atrial contractions with the same P-wave morphology as during SVT (Figures 2 and 3).

P-wave morphology during AT reveals superior frontal plane axis in leads II, III, and aVF (negative P waves); the initial portion of the P wave is isoelectric in V<sub>1</sub>, which coincides with a positive P wave in aVL followed by the positive terminal P wave in V<sub>1</sub>. This is most consistent with an inferior left atrial focus, likely toward the atrial septum (Figure 3).

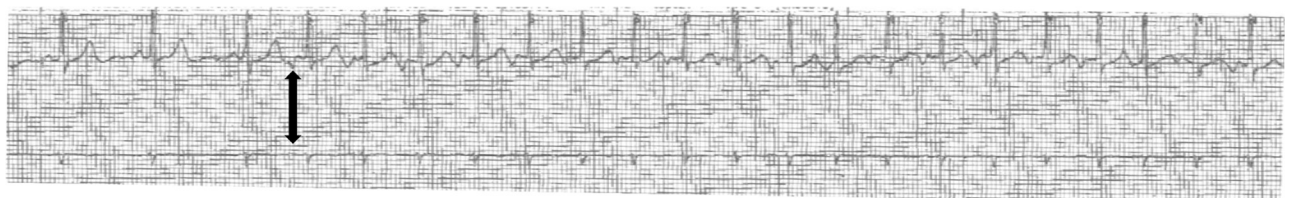
It is possible, albeit exceedingly unlikely, that the simultaneous occurrence of paroxysmal AT and PE is coincidental in our patient. This is supported by no prior history of palpitations until the patient’s presentation to the hospital and by resolution of palpitations due to paroxysmal AT with appropriate treatment of PE, without recurrence to date. Under these circumstances, one is obligated to believe that the PE is causally linked to the “positional” paroxysmal AT.

Occurrence of atrial tachyarrhythmias in patients presenting with PE is a predictor of poor prognosis with increased morbidity and mortality.<sup>2,13</sup> Despite this strong association, it remains speculative whether myocardial ischemia or right ventricular wall distension is the cause of this relation.

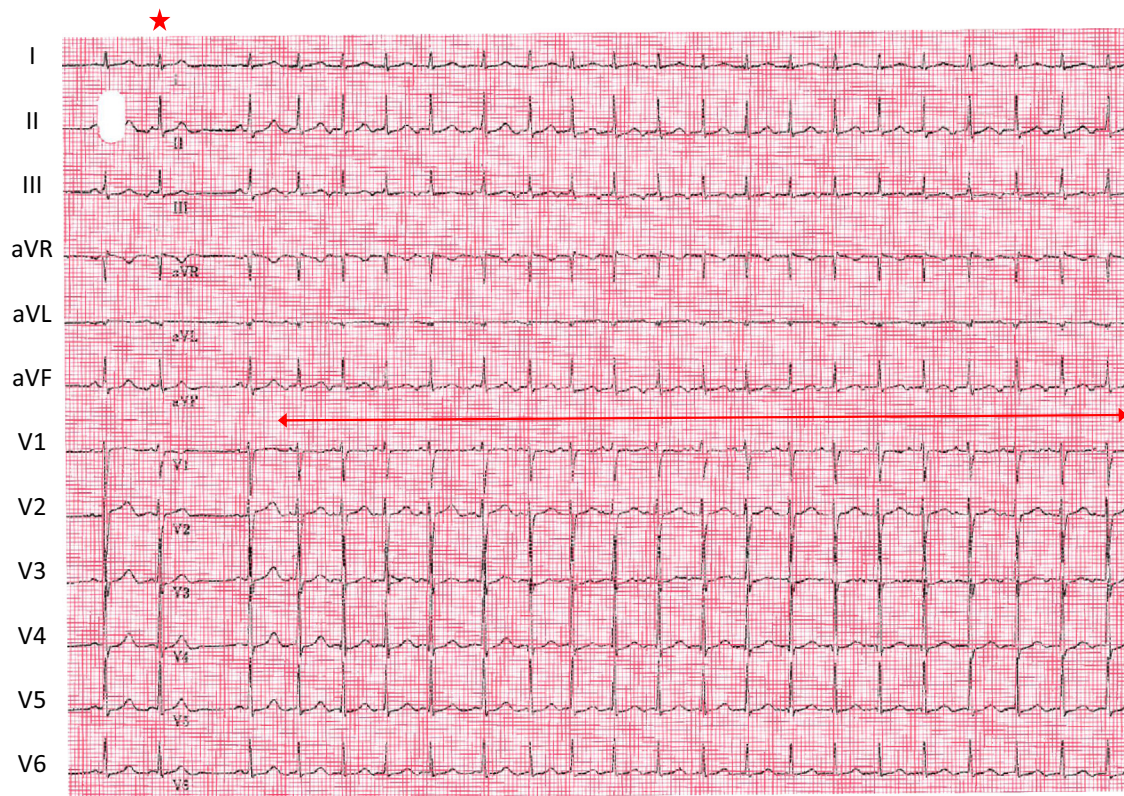
Although atrial tachyarrhythmias are common in PE, the underlying cause remains elusive. The classic notion of atrial stretch from elevated right heart pressure from a PE instigating atrial arrhythmias remains largely unchallenged.<sup>3</sup> It is likely that a small PE, such as in our patient, will not by itself cause the mechanical stretch needed to instigate AT. Accordingly, under these circumstances, it is intriguing to



**Figure 1** Computed tomography angiogram of the chest. Views of the chest computed tomography angiogram revealing evidence of filling defects in the segmental branches of left pulmonary artery in the left lower lobe of the lung, consistent with pulmonary embolism: **A:** coronal plane; **B:** axial plane; **C:** sagittal plane.



**Figure 2** Telemetry strip: Initiation of atrial tachycardia (marked by *double arrow*) instigated by lying down or bending.



**Figure 3** Twelve-lead electrocardiogram morphology of atrial tachycardia. The 12-lead electrocardiogram displays initiation of atrial tachycardia. The P-wave morphology is most consistent with an inferior left atrial focus of origin (see text for details).

speculate that further increase in right heart pressure owing to increase in venous return with maneuvers like recumbency or stooping are required to cause the mechanical stretch needed after a small PE to instigate AT. Furthermore, this mechanical stretch would be most profound in the coronary sinus, since it is a pliable, extracardiac structure, and has an opposite blood flow. It is much less likely for the thrombus to have embolized directly to the coronary sinus because of its reverse blood flow and no evidence of the same on the computed tomography angiogram.

## Conclusion

In summary, we describe an intriguing case of positional, paroxysmal AT occurring on recumbency and stooping, in a healthy woman presenting with an acute PE. Although positional AT is exceedingly rare, the possibility of a PE should be entertained when one is encountered.

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