Confounders of Vasovagal Syncope: Orthostatic Hypotension

Victor C. Nwazue, MD[a,b], Satish R. Raj, MD, MSCI[a,b,*]

KEYWORDS

• Syncope • Vasovagal syncope • Orthostatic hypotension • Autonomic dysfunction
• Blood pressure

KEY POINTS

- Most patients who present with syncope have vasovagal (reflex) syncope, but neurogenic orthostatic hypotension can also cause syncope, especially in older patients.
- Patients with neurogenic orthostatic hypotension have a fall in blood pressure greater than or equal to 20/10 mm Hg within 3 minutes of assumption of an upright posture.
- Neurogenic orthostatic hypotension can often be differentiated from vasovagal syncope by its differing hemodynamic patterns during tilt table test and differing clinical characteristics.
- Treatment of orthostatic hypotension focuses on improving symptoms through careful attention to hydration; bolus ingestion of water (osmopressor response); and the judicious use of short-acting pressor agents.
- A significant proportion of patients with orthostatic hypotension can also have supine hypertension, which may necessitate the use of short-acting pressor agents overnight.

NOT ALL SYNCOPE IS VASOVAGAL SYNCOPE, EVEN WITH A NORMAL HEART

Cardiologists are trained to assess syncope patients for life-threatening causes. This usually involves a detailed evaluation to exclude valvular heart diseases, myocardial diseases, and cardiac arrhythmia. After these potentially lethal causes of syncope have been excluded, most cases are ascribed to vasovagal syncope (VVS). Cardiologists working in a syncope clinic or in a tilt table laboratory quickly realize that there are other causes for syncope and presyncope, including neurogenic orthostatic hypotension (nOH) and postural tachycardia syndrome. This article highlights some contrasting clinical characteristics between nOH and VVS (**Table 1**), and then reviews the clinical evaluation and management of nOH. The clinical characteristics of postural tachycardia syndrome are reviewed elsewhere in this issue.

A striking difference between nOH and VVS is the different hemodynamic patterns during tilt table tests. These disorders each can have a distinct hemodynamic pattern during tilt table testing.

Research Funding: Supported in part by NIH grants R01 HL102387, P01 HL56693, and UL1 TR000445 (Clinical and Translational Science Award).
Conflicts of Interest: None.
[a] Division of Clinical Pharmacology, Department of Medicine, Autonomic Dysfunction Center, Vanderbilt University School of Medicine, AA3228 Medical Center North, 1161 21st Avenue South, Nashville, TN 37232–2195, USA; [b] Division of Clinical Pharmacology, Department of Pharmacology, Autonomic Dysfunction Center, Vanderbilt University School of Medicine, AA3228 Medical Center North, 1161 21st Avenue South, Nashville, TN 37232–2195, USA
* Corresponding author. Division of Clinical Pharmacology, Departments of Medicine and Pharmacology, Autonomic Dysfunction Center, Vanderbilt University School of Medicine, AA3228 Medical Center North, 1161 21st Avenue South, Nashville, TN 37232–2195.
E-mail address: satish.raj@vanderbilt.edu

Cardiol Clin 31 (2013) 89–100
http://dx.doi.org/10.1016/j.ccl.2012.09.003

Table 1
Clinical comparison of vasovagal syncope and neurogenic orthostatic hypotension

Features	Vasovagal Syncope	Neurogenic Orthostatic Hypotension
Typical age	Any age; first episode usually in second or third decade	>50 y
Gender (% female)	60%	40%
Symptoms with body position change	After prolonged sitting or standing	Immediately with sitting or standing
Syncope	+++	++
Presyncope	+	+++
Orthostatic Hypotension	+/- (usually only at time of faint)	+++++
Hemodynamic pattern with head-up tilt	Sudden drop in BP and HR	Early and progressive decline in BP

Abbreviations: BP, blood pressure; HR, heart rate.

During head-up tilt patients with VVS often hold a steady blood pressure (BP) for several minutes (often >10 minutes) after head-up tilt, before they develop symptoms and drop their BP rapidly (**Fig. 1**A). Patients with nOH are not able to maintain BP with head-up tilt. Their BP starts to fall immediately and orthostatic hypotension usually develops within 2 to 3 minutes of tilt (see **Fig. 1**B).

HEMODYNAMIC PHYSIOLOGY OF STANDING: HEALTHY AND ORTHOSTATIC HYPOTENSION

With the assumption of an upright posture, there is a downward shift of approximately 500 mL of blood to the dependent areas (mainly abdomen and legs). This gravitational shift in blood results in decreased venous return, decreased cardiac output, and eventually decreased BP (**Fig. 2**A).[1] This "unloads" the baroreceptors, and triggers a reflex sympathetic activation with a resultant increase in heart rate (HR) and systemic vasoconstriction (countering the initial decline in BP). In a healthy individual, the net effect of assumption of upright posture is an increase in HR of 10 to 20 bpm, a minimal change in systolic BP, and an approximately 5 mm Hg increase in diastolic BP.

In patients with nOH (see **Fig. 2**B), the efferent limb of the baroreflex cannot be adequately engaged. This can result in a lack of sympathetically

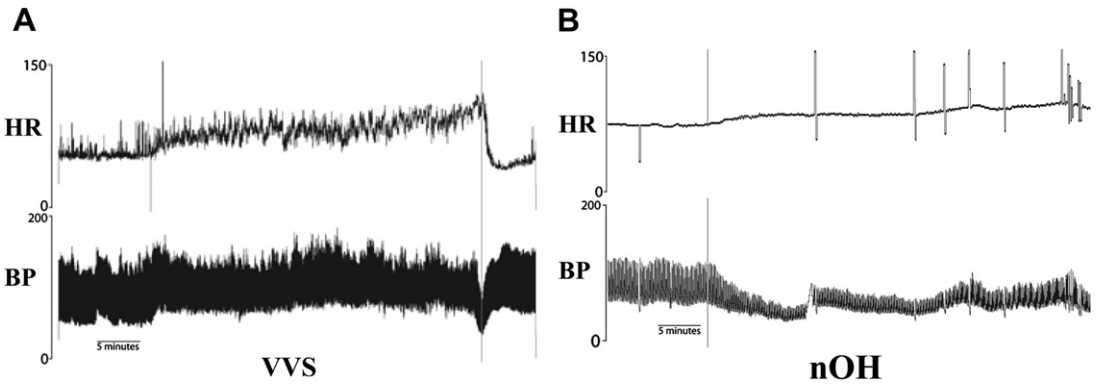

Fig. 1. Head-up tilt test traces from a patient with VVS and nOH. (*A*) With VVS, the heart rate (HR) and BP increase a little bit at the onset of tilt, and they are maintained for more than 25 minutes before a sudden precipitous drop in BP before the table is returned to the supine position. (*B*) With nOH, the BP falls almost immediately when the table is tilted up with only minimal changes in HR. (*From* Raj SR, Sheldon RS. Head-up tilt-table test. In: Saksena S, Camm AJ, editors. Electrophysiological disorders of the heart. 2nd edition. New York: Saunders; 2012. p. 73–6–73–8; with permission.)