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A Validated Test for Neurogenic Orthostatic Hypotension at the Bedside

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We read with interest the small study by Balagny and colleagues, performed in response to our publication on the orthostatic heart rate (HR) and blood pressure (BP) changes in the diagnosis of neurogenic orthostatic hypotension.¹ Our study, which was a multicenter collaboration within the Autonomic Disorders Consortium, showed that a rise in HR of <0.49 beats/min per mmHg of systolic BP fall provided optimal sensitivity and specificity to identify patients with structural lesions within the baroreflex arch and to distinguish them from those with hypotension due to dehydration or polypharmacy or both. The calculation provides a simple estimate of baroreflex gain that can be easily applied at the bedside. To systematically study the HR/ BP ratio with a multicenter approach, we standardized the extent of orthostatic stress by using a tilt table.

Balagny and colleagues tested the accuracy of the ratio within their hospital setting when patients were actively standing. There is a long-standing debate within the autonomic field as to whether tilt table testing and active standing provide the same degree of physiological stress.² A tilt table test involves the passive movement of a subject to the vertical position, whereas an active standing test involves central command and muscle activity. Moreover, when the subject is upright on the tilt table, body weight is supported, whereas in active standing the muscle pump is engaged through postural sway. Because of these differences, we are delighted that Balagny and colleagues reported that the <0.49 beats/min per mmHg threshold performed equally well during an active standing test to distinguish neurogenic from non-neurogenic orthostatic hypotension. Our hope is that the ratio becomes a widely used clinical tool to screen for neurogenic orthostatic hypotension. This is particularly important given the prognostic implications and the high number of patients with autonomic failure who go on to develop central nervous system deficits due to α -synuclein pathology.³

References

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Potential Conflicts of Interest
Nothing to report.

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