Response to letter to editor regarding “visceral sensitization in postural tachycardia syndrome”

Ramesh K. Khurana

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I thank Dr. Farmer, Dr. Fikree, and Dr. Aziz [1] for their thoughtful input. The authors propose abnormal gastric myoelectric activity as another form of visceral hypersensitivity. They believe that joint hypermobility syndrome (JHS) is an important confounding variable that should have been controlled for in our study.

The authors cite data from various studies to support their contention. Seligman et al. [2] found enhanced variability of gastric myoelectric activity in PoTS patients and attributed gastrointestinal dysfunction to splanchnic hyperemia, anxiety, or abnormal connective tissue matrix in the gastrointestinal tract. Fikree et al. [3] used validated questionnaires in patients with JHS and gastrointestinal symptoms to conclude that this association may result from autonomic dysmotility and/or hypersensitivity factors. I concur with Dr. Farmer and co-authors that gastrointestinal symptoms in PoTS patients may be another possible manifestation of visceral hypersensitivity despite the fact that cited studies only allude to this issue.

Regarding JHS and PoTS, Gazit et al. [4] found PoTS in 4 of 27 (15 %) JHS patients but did not demonstrate cause-and-effect relationship specifically pertaining to PoTS. With the prevalence of JHS at about 20 % [3] and the heterogeneous nature of PoTS [5], we await definitive

proof that JHS is a confounding variable in studies of visceral hypersensitivity in PoTS.

The goal of our simple study was to use an objective correlate (heartbeat) of a disabling symptom (palpitation) to demonstrate visceral sensitization in PoTS and to provide a nidus for more sophisticated studies in this area.

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R. K. Khurana (✉)
Department of Medicine, MedStar Union Memorial Hospital,
Baltimore, MD, USA
e-mail: ramesh.khurana@medstar.net