Postural tachycardia syndrome is not caused by deconditioning

Editor:

We thank Oldham et al.[1] for their excellent study on low ventricular filling pressures as a cause of exercise intolerance and dyspnea. Although only 5 patients in this cohort had a tilt table test positive for postural tachycardia syndrome (POTS) and only 2 had autonomic neuropathy, we suspect that these findings are applicable to the majority of patients with POTS. POTS arises from multiple etiologies, but the hallmark features of exercise intolerance, orthostatic tachycardia, fatigue, dizziness, and, in many cases, dyspnea upon exertion or at rest are common to all patients, regardless of the underlying cause. Most patients undergo extensive noninvasive cardiac testing and are often told that their symptoms are secondary to deconditioning, given a common finding of poor exercise tolerance on an exercise stress test. Previously, a low stroke volume and decreased cardiac mass in patients with POTS have been attributed hypothetically to deconditioning.[2,3] This study provides the first objective evidence that low ventricular filling pressures in patients with POTS are contrary to what would be expected in deconditioned patients—high filling pressures.[1]

Although the benefits of exercise have been acknowledged in several studies, almost 60% of patients with POTS are unable to complete an exercise training program despite their efforts.[4-6] Importantly, Oldham et al.[1] demonstrate that exercise intolerance in POTS is not caused by a lack of maximum effort from the patient but that low ventricular pressures occur despite the maximum effort.

Patients often feel frustrated and blamed for their illness and a lack of improvement or recovery when they are labeled as deconditioned or told that they are not giving their maximum effort, regardless of whether they are exercising routinely as part of their therapeutic regimen or have not been sick long enough to become deconditioned. Deconditioning can occur secondary to prolonged bed rest and chronic inactivity in patients with POTS, but it does not appear to be a primary underlying mechanism. Larger studies comparing the invasive cardiopulmonary exercise tests in a cohort of patients with POTS versus sedentary healthy individuals are needed to further delineate the pathophysiology and possible therapy for POTS in terms of both tolerated exercise programs and pharmacotherapy.

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