Editorial Commentary

Exercise Training in Postural Orthostatic Tachycardia Syndrome

Blocking the Urge to Block \(\beta\)-Receptors?

Michael J. Joyner

When heart rate is “too high,” perhaps the reflex therapeutic response is to give \(\beta\)-blockers. These drugs are certainly used to treat the excessive heart rate responses to standing seen in the Postural Orthostatic Tachycardia Syndrome (POTS). \(^1\) In this context, since POTS was defined as an identifiable syndrome, ideas about inadequate peripheral vasoconstriction, altered blood volume regulation, and baro-reflex dysfunction emerged as pathophysiological explanations for it. \(^2\) However, the final common symptom pathway that seems to prompt many patients to seek medical attention is typically dominated by concerns about a high heart rate response to standing.

Over the last 5 to 10 years, a number of investigators interested in exercise and familiar with bedrest deconditioning began to ask simple questions about the potential role of deconditioning in POTS and other forms of orthostatic intolerance. \(^3\)-\(^5\) The driving forces behind these questions were observations in POTS patients that included low exercise tolerance, high heart rate responses during submaximal exercise, and reduced stroke volume during exercise. These responses to exercise are all considered hallmarks of the untrained or deconditioned state. Another key physiological response to deconditioning is a loss of circulating blood volume, which has also been noted in POTS. Importantly, when otherwise fit individuals undergo periods of experimental bedrest or space flight–induced deconditioning, the heart rate responses to orthostatic stress can also increase dramatically. \(^3\),\(^4\),\(^6\) These observations raised the possibility that endurance exercise training might be an effective treatment for POTS.

Along these lines, Fu et al.\(^7\) demonstrated recently that carefully monitored and graded endurance exercise training can relieve and even cure POTS in most patients. The responses that these investigators noted with exercise training in POTS were essentially the physiological reverse of the bedrest deconditioning studies.

No the question is how does exercise training compare with what might be called standard therapy with \(\beta\)-blockers?

In this edition of Hypertension, Fu et al.\(^7\) compare 12 weeks of adult fitness style endurance exercise training with medical therapy using \(\beta\)-blockers on orthostatic and other physiological responses in POTS patients. They demonstrate that, although both therapies reduced the heart rate response to standing, only exercise training increased the aldosterone: renin ratio. \(^7\),\(^8\) In addition, exercise training but not drug therapy increased blood volume and total hemoglobin mass. Finally, exercise but not propranolol improved quality of life.

POTS is a complex syndrome, and the patient narratives associated with it frequently begin with a flu-like or acute illness followed by a prolonged period of inactivity. Frequently, there is diagnostic confusion until patients reach a tertiary referral center where people familiar with the syndrome and other forms of orthostatic intolerance practice. This suggests that any symptoms associated with the acute disease may have been amplified by periods of inactivity. In addition, POTS is also associated with somatic hypervigilance and catastrophizing, which makes it likely that patients who experience it will overinterpret any symptoms they have, and any worry about symptoms might tend to reinforce the inactive state. \(^7\) Was the improved quality of life with exercise training strictly physiological? Or, did the training somehow also blunt the somatic hypervigilance and/or empower the patients, perhaps by demedicalizing POTS?

Along these lines, early ambulation and aggressive rehabilitation strategies have replaced or are replacing conservative approaches in many conditions. For many years, prolonged bedrest was mandated after myocardial infarction only to be replaced over time by much more aggressive and effective approaches. Likewise, postoperative strategies in surgery have also started to focus on less bedrest and more activity. From a public health perspective, physical activity is clearly seen as a broadly effective measure for the prevention or treatment of many diseases, especially cardiovascular disease and diabetes mellitus.

In summary, the observations that exercise training can improve exercise tolerance, increase stroke volume, increase blood volume, and normalize the heart rate responses to standing in patients with POTS provide convincing evidence that calibrated endurance exercise training should play a primary role in the treatment of this syndrome. These findings also raise important questions for other chronic conditions like fibromyalgia and chronic fatigue syndrome. To what extent is secondary deconditioning exacerbating the symptoms? In this context, evidence is starting to accumulate that safe and effective exercise training and behavioral programs

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can be devised for these conditions to alleviate symptoms and, more importantly, suffering. Patients with POTS and other syndromes likely to be amenable to treatment by exercise training have real symptoms, real suffering, and in many cases their conditions may have been amplified by somatic hypervigilance and excessive medicalization. Perhaps the final questions are philosophical. How does a skilled clinician use the information from Fu et al to help these complex patients? How can we block the urge to block β-receptors and encourage a wide range of patients to exercise, including those with POTS?

Disclosures

None.

References


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