

# Activity and Exercise Intolerance After Concussion: Identification and Management of Postural Orthostatic Tachycardia Syndrome

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**Background and Purpose:** Postural orthostatic tachycardia syndrome (POTS) is increasingly recognized as a complication affecting recovery from concussion. Individuals with POTS demonstrate refractory dizziness, lightheadedness, cognitive dysfunction, fatigue, headache, chronic pain, nausea and gastrointestinal dysmotility, activity and exercise intolerance, syncope, and tachycardia. Subtypes of POTS may include hypovolemia, hyperadrenergic states, autonomic neuropathy, and underlying autoimmunity, which may variably impact response to rehabilitation in varying ways. The subtle presentation of POTS postconcussion is often mistaken for underlying anxiety, conversion disorder, or lack of motivation for recovery. This article will present clinical features of POTS that may arise after concussion, and propose a role for physical therapists in the diagnosis and management of POTS during concussion recovery.

**Summary of Key Points:** Data recorded and entered into a database during clinic visits from a large pediatric institution indicate that 11.4% of individuals diagnosed with POTS report onset of symptoms within 3 months of sustaining a concussion. Activation of the sympathetic nervous system can result in lightheadedness, shortness of breath, chest pain, tachycardia, palpitations on standing or with exertion, and activity and exercise intolerance. Identified comorbidities in people with POTS such as joint hypermobility and autoimmune disorders can further influence recovery.

**Recommendations for Clinical Practice:** Physical therapists may identify signs and symptoms of POTS in a subset of individuals who remain refractory to typical interventions and who exhibit symptom exacerbation with orthostatic activity. Incorporation of an individualized POTS exercise program into current established concussion interventions may be useful, with emphasis on initial recumbent exercises and ongoing physical therapy assessment of exercise tolerance for dosing of activity intensity and duration.

**Video Abstract available** for more insights from the authors (see Supplemental Digital Content 1, available at: <http://links.lww.com/JNPT/A211>).

**Key words:** *lightheadedness, human movement system, mental foginess, refractory dizziness, syncope*

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## INTRODUCTION

The World Health Organization diagnostic criteria for post-concussion syndrome require that 3 or more symptoms of headache, dizziness, fatigue, irritability, difficulty with concentration and mental tasks, memory impairment, sleep disturbances, heightened emotional responses, and poor stress tolerance must be present and interfering with the ability to participate in life activities.<sup>1</sup> Early reports of dizziness and mental foginess have been associated with prolonged recovery following concussion in high school athletes.<sup>2,3</sup> Physical therapists address symptoms related to pain, headache, dizziness, and imbalance, and should incorporate interventions targeted to restore the interpretation of vestibular, oculomotor, cervicothoracic, and somatosensory afferent input through activities that progressively challenge central sensory processing and motor control responses.<sup>4-6</sup> Among people experiencing prolonged recovery from injury, symptoms over time become multifactorial and require astute clinical reasoning skills to differentiate underlying causes for symptom triggers and to develop customized intervention plans until biomarkers or imaging techniques offer stronger evidence to localize brain trauma and neurophysiological involvement.

The pathophysiology following concussion has been well described to include a series of metabolic events resulting in altered cerebral blood flow, inflammatory processes, and disrupted axonal communication.<sup>7,8</sup> Diffuse axonal injury causes damage to cortical and subcortical regions of the brain, potentially impacting regulation of body temperature,

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sleep cycles, digestion, and other autonomic functions.<sup>9</sup> A growing body of evidence demonstrates that there is a transient cardiovascular autonomic dysfunction during the acute and subacute period following sports-related concussions and blast exposures in service members.<sup>10-12</sup> Heart rate variability studies have attempted to measure sympathetic nervous system reactivity following concussion, although the evidence is conflicting.<sup>13-15</sup> Two recent studies combined heart rate variability with autonomic function testing during a 14-day subacute recovery period and at a protracted time frame as a mechanism to further study effects of altered autonomic function after concussive injuries.<sup>10,16</sup> Ongoing efforts to develop imaging techniques and biomarkers to confirm and localize concussion injuries have contributed to the understanding that dysregulation of the autonomic nervous system can occur as a consequence of injury.<sup>17</sup>

Graded exercise testing has been proposed as a method to classify ongoing symptoms 3 weeks postinjury into 3 subtypes of neurological postconcussion disorders.<sup>18</sup> Individuals unable to exercise at maximal exertion following sports-related concussion because of increased symptoms have been categorized as having physiological postconcussion disorders, likely related to ongoing reduction in cerebral blood flow and prolonged effects of the metabolic energy crisis as well as autonomic dysfunction.<sup>19</sup> The recommendations for the management of identified physiologic postconcussion disorders include earlier initiation of subsymptom aerobic exercise alongside treatment for potential visual, vestibular, and somatosensory impairments.<sup>20</sup> In a retrospective study, 20 consecutive patients who sustained a concussion injury were evaluated to determine whether dizziness described as lightheadedness during quiet standing and exercise could be attributed to dysautonomia. Results showed significant tachycardia on tilt-table testing, altered adrenergic function during baroreflex testing, and variable standing norepinephrine (NE) levels, providing evidence linking dizziness and autonomic nervous system dysfunction.<sup>21</sup>

Dizziness is a poorly defined term depicting multiple sensory experiences often resulting from altered peripheral sensory afferent input and complex central cortical and subcortical sensory-motor processing. The complexity of symptom etiology related to dizziness has been described to involve an intricate bilateral relay of neurological connections extending from the vestibular nuclei through the parietoinsular vestibular cortex and thalamus and across the corpus callosum.<sup>22</sup> In this context, lightheadedness in upright postures may be an indicator of dysautonomia compared with other descriptors of dizziness, such as vertigo or a persistent sense of motion, that are likely more indicative of peripheral or central vestibular dysfunction.

Researchers and clinicians recognize exercise intolerance in some individuals postconcussion despite graded exercise, Return to Play and Return to Duty exertion training programs that provide parameters for progressive cardiovascular training zones.<sup>23-25</sup> Autonomic nervous system dysregulation could account for failure to complete exertion training successfully in some cases due to orthostatic and activity intolerance.<sup>10,26</sup> Adjustments to heart rate (HR) training parameters should be made to disrupt the perpetuated

sympathetic nervous system activity, with supplementary strength and resistance training in semirecumbent positions. In such cases, the presence of a type of dysautonomia referred to as postural orthostatic tachycardia syndrome (POTS) could preclude concussion exertion program participation due to new or easily provoked symptoms. Identification of POTS requires additional patient education for personal health management to avoid severe exacerbation of a myriad of symptoms and, in some cases, syncopal events that could create risk of further head trauma. The purpose of this article is to present clinical features of POTS that may arise in some individuals postconcussion, to propose physical therapy screening for POTS as routine care after concussion injuries, and to introduce a modification to currently established concussion exercise training programs in the presence of POTS.

## OVERVIEW OF POTS

Postural orthostatic tachycardia syndrome is a form of dysautonomia characterized by sustained tachycardia and variable complex symptoms that occur upon standing.<sup>27</sup> It is estimated that 1 to 3 million Americans have POTS with a similar prevalence internationally. Onset of POTS typically occurs in adolescent girls and women of childbearing age, and women are affected more often than men by 4.5:1.<sup>28,29</sup> Associated symptoms include dizziness, headache, nausea, visceral pain, heaviness of the extremities, reduced mental clarity, and generalized fatigue.<sup>28,30,31</sup> Postural orthostatic tachycardia syndrome is also often accompanied by disturbed sleep, poor body temperature regulation, and gastrointestinal complaints of bloating, constipation, abdominal pain, and early satiety. Peripheral edema and skin discoloration patterns including livedo reticularis or acrocyanosis are common; in some cases, bladder dysfunction can occur.

In a retrospective study of 722 patients with POTS enrolled in a pediatric patient database, 11.4% of patients identified a temporal link to a concussive event precipitating their POTS symptoms.<sup>32</sup> In another study, a vestibular questionnaire issued to 50 consecutive patients diagnosed with POTS revealed that 100% of subjects experienced lightheadedness upon standing, 46% reported vertigo, 98% experienced unstable gait, and 40% identified with a “swimming” sensation, while 48% reported a “rocking” sensation.<sup>33</sup> In this cohort, a mean composite autonomic symptom score (COMPASS) score of 52 in combination with vestibular symptoms indicated the potential for peripheral and central neurophysiology as well as cerebral hypoperfusion contributing to symptoms of dizziness. While there are many potential triggers of POTS, there is growing awareness that head trauma and rapid deceleration injuries can be a stimulus for deregulation of the autonomic nervous system.

## PATHOPHYSIOLOGY OF POTS

Understanding the heterogeneous pathophysiology of POTS subtypes may improve medical and rehabilitative management. It is important to note that people with POTS may fall under multiple phenotypes and have underlying conditions affecting central and peripheral pathophysiology. Because of the numerous comorbidities associated with POTS noted in Table 1, a detailed interdisciplinary medical workup is

**Table 1. Comorbidities of Postural Orthostatic Tachycardia Syndrome**

Antiphospholipid syndrome
Anxiety disorders
Autoimmune thyroiditis
Celiac disease
Chiari malformation
Depression disorders
Diabetes
Eosinophilic esophagitis
Fibromyalgia
Hypermobile Ehlers-Danlos syndrome
Hypermobility spectrum disorders
Lyme disease
Mast cell activation syndrome
Median arcuate ligament syndrome
Migraine
Mitochondrial diseases
Multiple sclerosis
Myalgic encephalomyelitis/Chronic fatigue syndrome
Neurocardiogenic syncope
Neuromyelitis optica
Paraneoplastic syndrome
Porphyria
Sarcoidosis
Sjögren's syndrome
Small fiber neuropathy
Syringomyelia
Systemic lupus erythematosus

Adapted with permission from Pavlik D et al.<sup>34</sup>

indicated.<sup>34-37</sup> There are currently 3 clinical subtypes described in the literature: hypovolemic, neuropathic, and hyperadrenergic POTS.

Hypovolemia is often a contributing factor in POTS, as plasma volume, red cell volume, and total blood volume are reduced compared with control subjects in studies.<sup>28,38</sup> In POTS, the renin-angiotensin-aldosterone system responsible for the regulation of plasma volume does not function properly, impairing the typical water and sodium retention needed to build plasma volume.<sup>39</sup>

Approximately 50% of people with POTS experience a partial sympathetic denervation of blood vessels in the lower extremities, resulting in a reduced vasoconstriction response during upright posture, sometimes referred to as neuropathic POTS.<sup>30,40</sup> Impaired venous return upon standing causes blood pooling in the lower limbs and within the abdominal cavity, which lowers stroke volume and results in compensatory tachycardia and reduced exercise capacity. This leads to a hyperdynamic blood flow, also termed “high flow” POTS.<sup>41</sup>

A smaller subset of individuals with POTS develop excessive plasma NE greater than 600 pg/mL upon standing and profound sympathetic activity that often results in anxiety, palpitations, tachycardia, tremulousness, and sometimes postural hypertension with greater than 10-mm Hg systolic BP increase upon standing.<sup>42</sup> The excessive sympathetic tone is considered to be of central origin in hyperadrenergic POTS, but smaller increases in plasma NE upon standing, which are not uncommon in POTS, may also result as a compensatory response to hypovolemia and peripheral autonomic neuropathy. In some patients with POTS, a deficiency in the NE transporter protein may also lead to an excessive NE response.<sup>43,44</sup>

## MANAGEMENT OF POTS

The cardiovascular manifestations of POTS include reduced stroke volume, reduced blood and plasma volume, and decreased cardiac chamber size mimicking prolonged bed rest despite many individuals with POTS participating actively in sports and recreation prior to the onset of POTS symptoms.<sup>45,46</sup> Initial medical management of POTS typically includes specific guidance to increase daily fluid intake and sodium consumption and to wear compression stockings to encourage peripheral vasoconstriction. Pharmacological support often includes agents such as fludrocortisone to expand blood volume,  $\beta$ -blockers to reduce tachycardia, midodrine to reduce orthostatic hypotension, pyridostigmine to increase venous return and decrease tachycardia, and stimulants to reduce mental clouding, often referred to as brain fog.<sup>28,28</sup>

Some individuals with POTS respond favorably to progressive exercise training, resulting in gradual increase in blood volume, exercise capacity, and stroke volume along with improved left ventricular diastolic function.<sup>47-50</sup> Recommendation for exercise to manage POTS stems from a study that engaged primary care physicians in the distribution of a 3-month unsupervised exercise program, referred to as the Levine protocol, for patients with POTS.<sup>51</sup> Of the 41% who completed the program, 71% reported significant reduction in POTS symptoms. The majority of subjects (59%) failed to complete the program, with 24% reporting other medical problems limiting their ability to complete the program, 16% reporting difficulty gaining access to needed exercise equipment, and 40% reporting that the exercise program was too physically challenging.

A single case report describes the physical therapy evaluation and intervention of an adult who previously overcame her POTS diagnosis through medical management and exercise but had a relapse following brief fever and malaise.<sup>52</sup> The physical therapist was able to confirm the exacerbation with a systems review, test of upright tolerance monitoring HR and blood pressure (BP), and a Rockport Fitness Test (1-Mile Track Walk Test) to estimate maximal aerobic capacity ( $\dot{V}O_{2\max}$ ).<sup>53</sup> Based on the subject's previous success overcoming POTS using submaximal exercise and presenting health status, cardiovascular reconditioning with target HR ranges between 60% and 85%  $HR_{\max}$  and a semirecumbent strengthening program were conducted for 4 weeks, with weekly PT sessions to provide assessment and modulation of activity recommendations. At the time of discharge, the subject's exercise tolerance improved and she no longer met the diagnostic criteria for POTS. In a telephone follow-up 4 weeks postdischarge, she reportedly maintained improvements, though the reported estimated  $\dot{V}O_{2\max}$  increased only to 60% to 70% that of her gender and age-matched peers, perhaps reflective of a persistent mild autonomic dysfunction in some individuals that can result in ongoing lesser symptoms and the need for persistent pharmacological management.<sup>54</sup>

## POTS AND CONCUSSION: DIFFERENTIAL DIAGNOSIS

Orthostatic intolerance and activity or exercise intolerance after concussion may be key indicators of POTS. Table 2 contains a detailed review of systems that could be performed

**Table 2. Review of Systems for Postural Orthostatic Tachycardia Syndrome and Concussion**

Cardiovascular/Pulmonary system	Assess heart rate, respiratory rate, blood pressure, and presence of edema.
Integumentary system	Note altered skin coloration such as acrocyanosis or livedo reticularis and general skin integrity.
Musculoskeletal system	Observe gross abnormalities in range of motion including any apparent joint hypermobility; inquire regarding general areas of musculoskeletal pain or inflammation.
Neuromuscular system	Inquire regarding any changes in sensation or signs of neuropathy, especially in the distal extremities. Assess presence of muscle spasm/fasciculation.
Mental status	Inquire regarding any cognitive changes or loss of consciousness from fainting.

alongside the COMPASS 31, a symptom questionnaire that identifies nonspecific autonomic dysfunction in the domains of orthostatic intolerance, vasomotor, secretomotor, gastrointestinal, bladder, and pupillomotor functions.<sup>55,56</sup> Scores on the COMPASS 31 range from 0 to 100, with higher scores indicating greater severity. This tool has been shown to have preliminary clinical utility in quantifying symptom burden in individuals who have been diagnosed with POTS via autonomic function testing as compared with healthy controls, particularly related to the orthostatic intolerance and pupillomotor domains.<sup>57</sup> Future research investigating the validity of the COMPASS 31 in people with POTS and concussion injuries would allow recommendations for clinical application, improved communication to primary care providers, and identification of the need for specialty care referrals involved in concussion management.

Orthostatic testing offers objective data for clinical decision making. Baseline HR and BP should be obtained after 10 minutes of quiet supine rest. Immediate transition to standing is provocative and requires monitoring for symptoms of dizziness, unsteadiness, headache, nausea, tremulousness, sweating, and syncope while recording symptom provocation, HR, and BP after 3, 5, 7, and 10 minutes of standing. According to the 2015 Heart Rhythm Society International Consensus statement,<sup>58</sup> POTS is defined as variable constellation of symptoms that occur with standing, and the diagnostic criteria for POTS presented in Table 3 are based on Tilt Table Testing.<sup>58-60</sup> The active 10-minute standing test has not been validated as a diagnostic test; thus, individuals with sustained significant HR elevation and/or orthostatic symptoms during a standing orthostatic test should be referred for medical evaluation. Ideally, a diagnosis of POTS should be confirmed with passive Head Up Tilt testing, though patient access to autonomic testing can be challenging.<sup>61</sup>

Clinical features for the differential diagnosis of POTS in individuals who demonstrate activity and exercise intolerance postconcussion are illustrated in Supplemental Digital Content 2 Appendix 1, International Classification of Functioning, Disability and Health Model for Identification POTS, avail-

able at: <http://links.lww.com/JNPT/A212>. The presented International Classification of Functioning, Disability and Health model elucidates the similarities between symptoms of POTS and concussion and the shared medical history contributions that are often associated with prolonged recovery from concussion. Standard performance of a 10-minute standing test on initial physical therapy evaluation postconcussion would allow gathering of data regarding the incidence of orthostatic intolerance, early identification of the potential presence of autonomic dysfunction, and the possibility that POTS may be a poor prognostic factor in poor concussion recovery. Subsequent performance of orthostatic testing at monthly reassessment intervals for those with symptoms of unclear etiology, who exhibit poor response to conventional treatment or develop new-onset syncope, might reveal individuals who initially fall underneath the sustained elevation in HR diagnostic criteria but develop POTS over time. A schematic of the proposed integration of orthostatic testing in physical therapy management in people who sustain concussion or whiplash injuries is illustrated in Supplemental Digital Content 3 Appendix 2, Recommendations for the Management of POTS, available at: <http://links.lww.com/JNPT/A213>. Identification of POTS could more effectively identify individuals who benefit from a modified physical therapy intervention plan and allow a more accurate prognosis of recovery time frames.

## EXERCISE TOLERANCE IN INDIVIDUALS WITH POTS AND CONCUSSION

Exercise training for individuals diagnosed with POTS and concussion must be monitored and individualized. A clinician guide to the development of a POTS exercise program for pediatric and adolescent patients was produced at the Children's Hospital of Philadelphia (CHOP) and is based on the Levine protocol.<sup>51</sup> The underlying premise of the adapted exercise program is that individuals with POTS benefit from initial exercise training in a recumbent or seated position due to orthostatic intolerance that elicits tachycardia and excessive symptom provocation. The exercise guidelines, available through the Dysautonomia International Web site, include calculations for HR parameters and recommendations for rate of perceived exertion (RPE) during activity.<sup>62</sup> The suggested preliminary Base Pace training zone for use in people with POTS is derived from age-related HR calculations and provides an adjustment to the HR training zones of current return to sport and activity concussion exertion training programs. It is anticipated that those individuals requiring preliminary recumbent exercises in a Base Pace training zone will require a protracted length of time to successfully tolerate 20 minutes of activity at this low intensity as it will be challenging to remain within the

**Table 3. Age-related Diagnostic Criteria for POTS<sup>58-60</sup>**

Age, y	Criteria
Adolescents: 12-19	HR increase > 40 bpm or sustained orthostatic HR > 120 bpm; <20/10 mm Hg change in BP
Adults: >19	HR increase > 30 bpm or sustained orthostatic HR > 120 bpm; <20/10 mm Hg change in BP

Abbreviations: BP, blood pressure; bpm, beats per minute; HR, heart rate.

prescribed HR parameters due to sympathetic nervous system activation.

Heart rate training zone calculations should be modified for individuals with POTS due to persistent tachycardia causing exercise intolerance. Maximal HR is determined using the standard calculation (220 – age), but in the presence of POTS, the resting heart rate (RHR) must be determined by the quiet *supine* rest of orthostatic testing, as orthostatic postures would produce an elevated RHR. Subtracting RHR from the maximal HR determines heart rate reserve (HRR). In typical exercise training, a percentage of HRR is used to identify a targeted training zone. In the presence of POTS, the maximal steady state (MSS) is first calculated as 75% of the HRR plus the RHR (MSS = 0.75 [HRR] + RHR) to account for heightened sympathetic activity, ±5 beats per minute (bpm) for the MSS training zone.

Beginning exercise in the MSS training zone for those with POTS often results in elevated symptoms, tachycardia, and increased RPE. A base pace training zone offers a further reduction in training intensity and is determined as 75% to 85% of the MSS training zone; anything below the base pace becomes the recovery zone. Table 4 demonstrates calculated HR training zones for a 20-year-old with POTS and concussion who has a supine RHR of 76 bpm. As age increases and maximal HR reduces, the calculations for HR training zones will require further adjustment with appropriate clinical assessment of symptom provocation during supervised cardiovascular training.

Concussion exertion training protocols offer parameters for submaximal HR training zones and simultaneous balance and strengthening activities congruent with the recommendations in the CHOP POTS Exercise Program.<sup>23,62</sup> However, for the aforementioned 20-year-old without evidence of POTS, the target HR training zone of stage 1 (see Table 4) in the UPMC Exertion Protocol for Concussion is 30% to 40% of maximal exertion. Table 4 offers a comparison of the HR training zone

parameters, illustrating that the current recommendations for exertion training may not be tolerated if someone had undetected POTS. The initial base pace HR training zone recommended for those with POTS is slightly elevated at 125 to 145 bpm to accommodate for the anticipated sympathetic tone. Base pace exercise training appears to be aligned with stage 2 and 40% to 60% of maximal exertion in the UPMC protocol, indicating that the person with POTS would likely be working at a higher RPE or workload than would be desired in early recovery if an adjusted training zone was not utilized.

The challenge for individuals with POTS will be to find a recumbent exercise that permits sustained exercise for 3 to 5 minutes without exceeding the recommended HR range, symptom exacerbation, or elevation of RPE. As noted in the CHOP POTS Exercise Program, it may take 4 months of very gradual progression in the base pace training zone to tolerate 30 minutes of exercise before being able to attempt training in the MSS training zone, which correlates with stage 3 and 60% to 80% maximal exertion in the typical postconcussion exertion training programs. This protracted recovery time results in profound reduction in activity participation and can cause much personal stress and anxiety. Failure to recognize POTS as a contributing factor to exercise and activity intolerance can be misinterpreted by health care providers as a psychological overlay or noncompliance with recommended activity.<sup>63</sup>

Instruction in base pace training and development of individual warm-up, cooldown, and recovery techniques should help avoid excessive rapid rise in HR associated with POTS. A critical element involves initial training in mode 1 using a semirecumbent rowing machine, recumbent cycle or stepper, or in a swimming pool to prevent excessive HR rise outside of the base pace HR training zone. Activity progression and dosing should be addressed similarly to exertion training in standard postconcussion care, as demonstrated in Table 5, though it may take considerable time to progress to 15 to 20 minutes of exercise tolerance. A person is likely to remain in mode 1,

**Table 4. Sample Heart Rate Training Zone Calculations**

Training Zone	Heart Rate, bpm	Expected Rate of Perceived Exertion
CHOP POTS Exercise Program <sup>62</sup>		
Base pace training zone	127-144	2-4
0.75 (MSS) = 0.75(169) + 76		
0.85 (MSS) = 0.85(169) + 76		
Maximal steady state	164-174	5-8
MSS = 0.75 (HRR) + RHR = 0.75 (124) + 76		
MSS training zone = (MSS) ± 5 bpm		
Race pace = Above MSS	175-185	7-9
Recovery = Below base pace	<125	0-2
UPMC 5-Stage Exertion Protocol for Concussion <sup>23</sup>		
Stage 1: 30%-40% maximal exertion	113-126	Light aerobic conditioning
0.3 (HRR) + RHR = 0.3 (124) + 76		
Stage 2: 40%-60% maximal exertion	126-150	Light to moderate aerobic conditioning
0.4 (HRR) + RHR = 0.4 (124) + 76		
Stage 3: 60%-80% maximal exertion	150-175	Moderately aggressive aerobic exercise
0.6 (HRR) + RHR = 0.6 (124) + 76		
Stage 4: 80% maximal exertion	175	80% exertion avoiding contact
0.8 (HRR) + RHR = 0.8(124) + 76		
Stage 5: 100%	200	Full exertion for sports with contact
HRR + RHR = 124 + 76		

Abbreviations: HRR, heart rate reserve; MSS, maximal steady state; RHR, resting heart rate; UPMC, University of Pittsburgh Medical Center.

**Table 5. Progressive Exercise Training Modes**<sup>51,62</sup>

Mode 1	Any recumbent activity (rowing, cycle, stepper, kicking in a pool)
Mode 2	Upright stationary cycle
Mode 3	Treadmill walking 0% grade; elliptical trainer (arms stationary)
Mode 4	Treadmill walking with incline; elliptical trainer (arms moving); jogging 0% grade
Interval training	Initiate early interval training on recumbent equipment and gradually progress to upright postures

tolerating less than 10 minutes of base pace activity for the first month or longer as the level of fatigue and deconditioning associated with POTS is comparable with that of chronic obstructive pulmonary disease or congestive heart failure.<sup>27</sup> It is not uncommon for people with POTS to have exacerbations and variable response to recumbent exercise, with the need to repeat a previous mode(s) over prolonged time frames due to variant phenotypes of POTS, medication changes, hormonal influences, viral illnesses, or simply balancing work or school responsibilities with exercise through profound fatigue.<sup>46,52,64</sup>

Strengthening exercises to target postural control, core strength, and joint stabilization are prescribed alongside exercise training to overcome deconditioning and musculoskeletal pain that occur with prolonged inactivity. Necessary balance and sensory processing activities that involve somatosensory, visual, and vestibular conflict are beyond the scope of this article but are integral to the overall wellness and return to activity in patients recovering from prolonged inactivity associated with concussion and POTS.<sup>65</sup> Monthly calendars, available as part of the CHOP exercise program available online through Dysautonomia International, offer structure and guidance to conduct cardiovascular exercise training in specific HR training zones 3 times per week, with twice-weekly strength training.<sup>62</sup> Regular interdisciplinary communication with medical providers regarding activity tolerance provides necessary feedback regarding efficacy of potential POTS pharmacological interventions and fosters ongoing collaborative patient medical management.

## COMORBIDITIES OF POTS AFFECTING EXERCISE TOLERANCE

Numerous comorbidities have been associated with POTS, some of which may interfere with successful exercise training. One-third of people with POTS have comorbid Ehlers-Danlos syndrome, a heterogeneous group of inherited connective tissue disorders associated with joint laxity, joint subluxations and dislocations, and arthralgia.<sup>66,67</sup> The diagnosis of hypermobile Ehlers-Danlos syndrome, the most common form of Ehlers-Danlos syndrome seen in POTS, remains clinical as there is no genetic marker yet identified.<sup>68-70</sup> The correlation between POTS and hereditary connective tissue disorders is particularly important in the postconcussion population as development of craniocervical instability, craniocervical dyskinesia, and Chiari brainstem compression requires critical medical attention and can be primarily related to concussion or whiplash injuries as well as hypermobile

Ehlers-Danlos syndrome.<sup>71,72</sup> Patients with POTS and comorbid hypermobility spectrum disorders should be instructed in exercise approaches that emphasize joint stabilization and core control. Excessive stretching and hyperextension of joints should be avoided.

In some individuals, including those with postconcussive onset of POTS symptoms, POTS may be the consequence of an autoimmune mediated autonomic neuropathy, which may require treatment targeted to the autoimmune condition in addition to exercise and other standard POTS treatments. A study of 100 consecutive patients with POTS found that 31% of patients had 1 or more antibody markers and 20% had comorbid autoimmune disorders such as Hashimoto's thyroiditis, rheumatoid arthritis, or systemic lupus erythematosus.<sup>35</sup> Postural orthostatic tachycardia syndrome has also been reported in association with Sjögren's syndrome and celiac disease, both of which are known to cause autonomic neuropathies.<sup>73,74</sup>

Postural orthostatic tachycardia syndrome can occur in association with mast cell activation disorders, in which mast cells degranulate inappropriately and release mediators or proliferate to produce inflammatory responses that can occur throughout the body, as mast cells are present in all types of tissues.<sup>75</sup> Exercise can be a trigger for mast cell degranulation in these patients and in severe cases may result in exercise-induced anaphylaxis.<sup>76</sup> Some individuals with POTS and concussion also experience chronic fatigue and disturbed sleep and can benefit from a variety of supportive therapies due to prolonged recovery time and reduced engagement in life activities.<sup>30</sup>

For individuals with known or suspected autoimmune diseases or mast cell activation disorders, further medical management and medication support may be necessary to suppress pain and inflammatory responses associated with exercise.<sup>37,75</sup> This can include a wide spectrum of interventions from regular antihistamine use to immunomodulation infusion therapies. Current research efforts are targeted at better understanding antibody receptor and neurotransmitter activity involved in immunological responses and autonomic nervous system function.<sup>17</sup> For some individuals, return to previous sport and intensity of activities may be inhibited by the development of autonomic small fiber neuropathies.

## SUMMARY AND IMPLICATIONS FOR PRACTICE

Postural orthostatic tachycardia syndrome is a syndrome encompassing a cluster of symptoms following a wide range of triggering events resulting in a persistent dysfunction of autonomic nervous system regulation.<sup>30,77</sup> Numerous symptoms of POTS and concussion can be indistinguishable, requiring astute history taking for subjective complaints correlated with provoking activities. While transient dysautonomia is recognized as a consequence of concussion, there is growing evidence that protracted elevation of sympathetic activity can result in exercise intolerance and poor response to physical therapy interventions.<sup>78</sup> Routine collection of data related to orthostatic testing upon initial evaluation and at regular intervals will confirm the incidence of POTS related to concussion and whiplash-related deceleration injuries. Early identification of POTS allows necessary communication with medical providers for refinement of the diagnosis, patient

education, medication support, and referral to specialists as needed. Monitoring of HR and BP during exertion training postconcussion is imperative to prevent misinterpretation of exercise intolerance with symptom exacerbation. Failure to recognize POTS as a cause for dizziness and protracted recovery disregards a major complication that could result in elevated symptoms, stress, anxiety, and depression due to poor response to vestibular and neuromuscular physical therapy interventions.

In the case in which transient dysautonomia is detected but does not resolve, or POTS is identified in protracted recovery with heavy symptom overlay, the COMPASS 31 may be a useful screening tool to identify the need to refer to medical providers for further diagnostic testing. Medical management in complex cases involving POTS often requires a series of medication trials to modulate a variety of gastrointestinal, hormonal, neurological, immunological, and allergy-related symptoms. Feedback to prescribing providers may help determine medication success or failure as related to exercise and activity tolerance in physical therapy, though development of an appropriate measurement tool is needed. At present, the Patient Specific Function Scale could aid in assessing progress toward reduction of impairments and designing patient-directed goals for return to graded participation in specific activities.<sup>79</sup>

Finally, research studies should be conducted in physical therapy to determine whether exercise tolerance improves and whether recovery from POTS can be achieved with physical therapy oversight of exercise training progressing from recumbent to upright activity in conjunction with core and joint stabilization as well as strength training. Studies including submaximal exercise training in subjects who have sustained known concussion and whiplash injuries with and without evidence of dysautonomia are necessary to determine the effect of addressing POTS in physical therapy on overall recovery from injury. Physical therapy involvement in management of POTS would assist in identification of comorbidities such as joint hypermobility, inflammatory responses, and neurological findings such as neuropathy and neuropathic pain that further limit exercise tolerance in a subset of individuals with a complicated and protracted recovery pattern.

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### Award for Early Career Professional in Neurology (Due September 1)

Supports members who are new professionals and show potential to make lasting contributions, by providing them financial assistance to attend CSM.

### Award for Service to the Academy (Due September 1)

Honors a member whose contributions to the Academy have been of exceptional value.

### Award for Excellence in Neurologic Research (Due September 1)

Honors a member who has demonstrated continuing excellence in research related to neurologic physical therapy science, theory, practice, or education.

### Award for Excellence in Neurologic Education (Due September 1)

Honors a member whose clinical or academic neurologic educational contributions have been of exceptional value.

### Award for Clinical Excellence (PT & PTA) in Neurology (Due September 1)

Honors a member whose major professional involvement is currently with the practice of neurologic physical therapy.

### Award for Outstanding Clinical Innovator in Neurology (Due September 1)

Honors a member who has translated recent evidence or emerging practice/business strategies into a program, initiative, or service to benefit patients/clients with neurologic impairment.

### Award for Outstanding Advocacy in Neurology (Due September 1)

Honors a member who is an advocate for the neurologic physical therapy profession and/or neurologic populations.

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Please take the time to recognize a deserving colleague.