POSTURAL ORTHOSTATIC TACHYCARDIA SYNDROME (POTS): ITS RELEVANCE, AND IMPORTANCE, IN THE MANAGEMENT OF SPORT RELATED CONCUSSION.

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Keywords: (MESH): Brain Concussion; Post-Concussion Syndrome; Orthostatic intolerance; Postural Orthostatic Tachycardia Syndrome; Primary Dysautonomias; Exercise Test

EDITORIAL

Introduction

Recent evidence suggests that concussion affects the autonomic nervous system (ANS), and that dysfunction of the ANS may be a factor that can contribute to persistent post-concussion symptoms. [1-3] The 2017 Concussion in Sport Group (CISG) international consensus statement on concussion in sport [4] defined sport related concussion (SRC) as a "traumatic brain injury induced by biomechanical forces." The CISG expert consensus [4] further defined persistent symptoms following SRC as "failure of normal clinical recovery – that is, symptoms that persist beyond expected time frames (i.e. >10-14 days in adults and >4weeks in children)." Persistent symptoms following SRC does not represent a single pathophysiological entity, but a collection of post-traumatic symptoms that may be linked to coexisting and/or confounding factors, requiring a detailed multimodal clinical assessment to identify specific primary and secondary pathologies that may be contributing to persisting symptoms.[4] The American Medical Society for Sports Medicine (AMSSM) position statement on concussion in sport,[5] published in 2019, defined concussion as "a traumatically induced transient disturbance of brain function that involves a complex pathophysiological process." This pathophysiological process was described in the AMSSM position statement [5] as being "not completely understood but has been characterised as force delivered to the brain causing disruptive stretching of neuronal cell membranes and axons resulting in a complex cascade of ionic, metabolic and pathophysiological events."

Ongoing efforts to develop imaging techniques to identify concussion injury have contributed to the utilisation of diffusion tensor tractography (DTT) which have started to demonstrate traumatic or diffuse axonal injury of the neural tracts in patients following minor and indirect head trauma.[6,7] Diffuse axonal injury may also damage cortical and subcortical regions of the brain, which can impact autonomic functions.[8] The primary ANS control centre is located in the brainstem and is vulnerable to concussion injury, especially when rotational forces are transmitted through the cervical spine.[3] A recent systematic review exploring the impact of concussion on multiple aspects of ANS functioning, concluded that it is likely that concussion causes ANS anomalies.[3] This growing body of evidence in concussion has shown that autonomic dysfunction has the potential to impact cerebral perfusion and baroreflex efficiency,[3] as well as affect cardiac function during exercise.[1]

Aerobic exercise has been shown to improve ANS balance, CO_2 sensitivity, and cerebral blood flow regulation.[2,5] The CISG consensus statement, along with the AMSSM position statement, highlight data that suggests that an individualised symptom-limited, submaximal aerobic exercise program improves recovery of persistent post-concussive symptoms.[4,5] The Buffalo Concussion Treadmill Test (BCTT) – the most studied protocol – is a graded exertion test that methodically establishes the heart rate at which exercise-induced symptom exacerbation occurs in concussion patients.[1,2] Individuals unable to exercise at maximal exertion following SRC have been categorized as having 'physiological' persistent post-concussion symptoms, likely related to autonomic dysfunction.[4] The data collected during the BCTT is then used to prescribe an individualised, progressive symptom-limited aerobic exercise program.[2]

There has been recognition of ongoing exercise intolerance in some concussion patients, despite the use of an individualised, progressive symptom-limited aerobic

exercise program.[8] Autonomic nervous system dysfunction could account for the inability to successfully complete aerobic exercise training, in some cases, due to orthostatic intolerance.[8] A retrospective study of concussion patients who had dizziness symptoms, described as lightheadedness, during exercise and quiet standing, demonstrated significant tachycardia on tilt-table testing, providing evidence linking dizziness and ANS dysfunction.[9] It has been suggested that lightheadedness in upright postures may be an indicator of dysautonomia in comparison to other descriptors of dizziness, such as vertigo, that are more likely indicative of peripheral or central vestibular dysfunction.[8]

In cases of ongoing exercise intolerance, the presence of a type of dysautonomia known as postural orthostatic tachycardia syndrome (POTS) may need to be identified and managed before commencing a concussion aerobic exercise program.[8] While there are many potential triggers of POTS, there is a growing body of evidence that head trauma and rapid deceleration injuries can be a trigger for ANS dysfunction.[8]

POTS and Concussion

Postural orthostatic tachycardia syndrome is a type of dysautonomia characterised by sustained tachycardia and variable complex symptoms that occur upon upright postures.[10] Numerous symptoms of POTS and concussion can be indistinguishable. Dizziness, headache, nausea, vision changes, generalised fatigue, reduced mental clarity and disturbed sleep symptoms can be seen in both POTS and concussion.[8] Therefore, an astute clinical history of subjective complaints, as well as the routine assessment of orthostatic intolerance upon initial and follow-up evaluation, is required in all concussion patients.[8]

Orthostatic vital sign testing can provide objective data for clinical decision making. A baseline heart rate (HR) and blood pressure (BP) in quiet supine rest should be obtained, followed by the monitoring and recording of HR, BP and symptom provocation after the transition to standing.[8] Postural orthostatic tachycardia syndrome is defined by the worsening of symptoms, accompanied with an increased HR ≥30 beats per minute (bpm) or sustained HR >120 bpm when moving from a recumbent to a standing position, in the absence of orthostatic hypotension (>20/10 mm Hg drop in BP).[10] The identification of POTS can allow clinicians to more effectively recognise concussion patients who may benefit from a modified exercise therapy program.[8]

Why does the identification of POTS in concussion patients matter?

Postural Orthostatic Tachycardia Syndrome might be a reason that concussion patients may not be able to safely complete the BCTT (potentially due to orthostatic intolerance in the upright posture required for the test). Additionally, the BCTT is not an appropriate testing or exercise environment for all patients. For example, patients with significant balance problems may not be able to safely be assessed or exercise on a treadmill.

Second, individuals with POTS benefit from initial exercise training in a recumbent position and then gradually progressing to more upright exercise.[8] As such, the implementation of an individualised, progressive symptom-limited aerobic exercise program, beginning from a recumbent position and gradually progressing to more upright exercise training, may be considered appropriate in the management of concussion patients with POTS.

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