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CORRESPONDENCE

Attenuation of autonomic dysreflexia during functional electrical stimulation cycling by neuromuscular electrical stimulation training: case reports

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To the Editor:

It is with great interest that we read the case report by Gill et al. [1]. Gill et al. reported the positive effects on blood pressure regulation during exercise (functional electrical stimulation) in two patients with spinal cord injuries (SCI) in a recent issue of Spinal Cord Series and Cases (2020; 6: 12). Individuals with high thoracic or cervical SCI often suffer from autonomic dysfunction, which can manifest as autonomic dysreflexia (AD), a surge in blood pressure (BP) initiated by a sustained noxious or non-noxious stimulus below the level of the lesion, or orthostatic hypotension (OH), the inability to raise BP in response to gravitational stress [2]. While AD is a sign of sympathetic over-activation, OH is a sign of sympathetic failure [3]. Autonomic dysfunction is often not recognized because many individuals remain asymptomatic [4, 5]; however, unrecognized AD can be fatal [6, 7]. The primary prophylactic for AD is antihypertensive medications, but these drugs can result in subsequent OH [8]. Further, treatment for AD has remained unchanged since 1997 [9]. Therefore, investigations into the effects of exercise training on cardiovascular control in individuals with SCI, and AD, is pivotal.

Exercise limitations following SCI are well known. Impaired neural control of skeletal muscles results in deconditioning and various negative outcomes that are generally focused on the reduction in mitochondrial mass and oxidative capacity [10, 11], in addition to interrupted sympathetic vascular innervation [12]. During exercise, individuals with higher and more severe injuries may have a loss of sympathetic engagement resulting in "exertional hypotension" [12], which is a cascade of impaired vaso- and venoconstriction, reduced venous return, and impaired cardiac output [13]. These outcomes ultimately reduce oxygen delivery to working muscles thereby impacting exercise performance. However, if some sympathetic vasomotor function remains intact, one would expect an individual with SCI to have a more typical exercise pressor reflex (EPR). The EPR broadly describes the cardiovascular responses to exercise; typically the exercise-induced increase in BP. That said, the EPR originates from active skeletal muscle tissue and leads to sympathetically mediated increases in cardiac output and BP during exercise [14–17]. Disease states shift the EPR away from cardiac output to systemic vasoconstriction mediated increases in BP. In other words, when there is dysfunction in the cardiovascular system, the BP response to exercise is exaggerated and potentially mediated at the microvascular level.

Gill et al. [1] published a case study on two individuals with motor complete C-6 injuries undergoing neuromuscular electrical stimulation (NMES) resistance training and lower extremity cycling. Participant A underwent 12 weeks of NMES assisted resistance training twice weekly and then crossed over to 12 weeks of lower body cycling with functional electrical stimulation (FES), and Participant B underwent 12 weeks of passive limb movement followed by FES-assisted lower body cycling. It should be noted that at rest Participant A was hypertensive and Participant B was hypotensive. Brachial BP was taking every 2–5 min during each training session. Training sessions were halted if BP exceeded 130/80 mmHg, and resumed once BP fell below 130/80 mmHg. The acute BP response to exercise was calculated as a difference from baseline (exercise – resting)

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and plotted by session number for each condition and participant. The data show a trend in lowering BP over the duration of both the resistance training with NMES (Participant A) and FES lower body cycling (both participants).

The results from these case reports are of great interest to those of us interested in understanding cardiovascular control following SCI. However, we contend that the findings, as presented, do not reflect improvement in AD with NMES as stated in the report. Although the findings do not rule out a potential beneficial effects of NMES on the magnitude of BP rise during AD, an equally plausible explanation is that NMES elicited an improvement in cardiovascular or skeletal muscle function (e.g., mitochondrial and microvascular adaptations) thereby reducing the magnitude of the BP response to exercise (e.g., reduced EPR). For example, in other pathologies (e.g., hypertension, heart failure, peripheral artery disease, and others) the EPR is exaggerated [14–17]; however, if the stimulus altering the EPR is alleviated the exaggerated response can be partially corrected [18, 19]. The exaggerated BP response to exercise is potentially impacted by vascular function independent of overt cardiovascular disease (e.g., sub-clinical vascular dysfunction) [17]. In individuals with SCI, adverse changes in cardiac and vascular function are well known [20]. In able-bodied individuals, vascular improvements following exercise is also well documented. Thus, we contend that the improvements in BP control during NMES reported by Gill et al. [1] is likely due to typical exercise-induced adaptations to exercise as opposed to an attenuated rise in BP in response to AD.

The authors hypothesized that these changes in BP during exercise could be the result of desensitization of peripheral nociceptors following NMES. However, the authors neglected to address any other potential exercise adaptations found in humans. For example, improvements in mitochondrial function and aerobic capacity [21] and how these effects may impact microvascular function and BP. In addition, repeated, rather than sustained, stimuli may not elicit AD, because of the naturally occurring stimuli relief that is inherent in the NMES protocol.

The improvements in BP control during exercise reported by Gill et al. [1] do lend to our understanding of cardiovascular control following SCI and its adaptation following training. While these beneficial adaptations to NMES may indeed reflect an attenuated BP response to sympathetic stimuli below the level of lesion, we contend that there needs to be extreme caution when interpreting these findings as an attenuation of AD. At minimum, we suggest that the other various well-known mechanisms that contribute to the acute increase in BP during exercise, and its adaptation to training in humans, should be prefaced in context with the complex mechanisms that elicit AD.

Compliance with ethical standards

Conflict of interest The authors declare no competing interest.

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