Time to reconsider the importance of autonomic function in Paralympic athletes with spinal cord injury

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TITLE PAGE

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Letter To The Editor:

We read the recent paper by Pelliccia and colleagues with great interest.[1] The authors are to be commended for conducting this study in a large sample (n=252) of Paralympic athletes. One of the main findings was that cardiac remodeling in Paralympic athletes differed by disability [i.e., spinal cord injury (SCI) and non-SCI)] and sport discipline (endurance and nonendurance). Herein we discuss pertinent consideration for individuals with SCI, who account for forty-four percent of the cohort. The authors ascribed the smaller left ventricular (LV) dimension in those with SCI compared to non-SCI to multiple factors, one of which is the alteration of descending autonomic outflow following SCI. Cardiovascular consequences following SCI are markedly determined by the neurological level of injury (NLI) and severity of damage to autonomic pathways.[2] Pelliccia *et al*, [1] excluded individuals with "quadriparesis", yet we assume that those with high-thoracic [first to the sixth thoracic segments (T1-T6)] are included in their analyses. Injuries at and above the T6 spinal segment can not only cause diminished sympathetic control to the peripheral vasculature but may also compromise sympathetic outflow to the heart (i.e., T1–T5 spinal segments).[3] Consequently, this can result in reduced circulating catecholamines and chronotropic incompetence, which impact cardiac mechanics and in turn exercise performance.[2] These lesion-dependent impairments in cardio-autonomic control compromise the physiological response to exercise and eventually may lead to reduced exercise-induced cardiac remodeling. Consequently, the authors may want to elaborate on the effect of NLI and severity of cardio-autonomic dysfunction on cardiac remodeling in their cohort, accounting for different NLI (high-, low- thoracic and lumbar). Furthermore, the mode of exercise training (e.g. endurance and power) is believed to determine the hemodynamic load imposed on the LV, and this load (i.e., pressure or volume) has been suggested to be the primary stimulus for LV eccentric or concentric remodeling.[4] In individuals with SCI ranging between T1 and the first lumbar

segment, Gates *et al*,[5] demonstrated no distinct LV remodeling in response to endurance or power training. The work of Pelliccia and colleagues is indeed worthy of praise. However, a deeper discussion around injury specific impairments and different patterns of LV remodeling via different exercise training stimuli (endurance vs. non-endurance), specifically in this population, would provide further valuable insight.

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