Autonomic Nervous System in Paralympic Athletes with Spinal Cord Injury

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KEYWORDS

• Autonomic nervous system • Autonomic dysfunction • Boosting
• Paralympic athletes • Spinal cord injury

KEY POINTS

- Intactness of the ANS is crucial for the performance of athletes. Following SCI, damage to critical autonomic innervation can result in changes of the response to physical activity, compromising performance.
- Athletes with SCI exhibit a lesion-dependent impairment of cardiovascular, respiratory, and thermoregulatory response to exercise that could limit endurance performance and place some athletes at a significant disadvantage during exercise training and competition.
- Individuals with SCI suffer from BP liability, either an extremely low resting BP and orthostatic hypotension or a sudden increase of BP, known as AD; both alterations in BP have a negative impact on the athletes' performance.
- If not taken care of properly, AD is a serious threat to the athletes' health and could even lead to fatal consequences.
- A combined assessment of sensorimotor and autonomic functions following SCI could help to understand the complexity of this devastating injury and to classify athletes more appropriately.

INTRODUCTION

Spinal cord injury (SCI), one of the most debilitating conditions, immediately alters an individual's life. Every year, up to half a million people worldwide sustain this

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devastating injury, that is, traumatic and atraumatic SCI.[1] This results in an enormous burden of disability and decreased quality of life (QoL).[2] The prevalence of traumatic SCI is high, yet differs among countries, such as Canada (1298 per million),[3] the United States (906 per million),[4] or Australia (681 per million).[1,4] Most individuals sustaining SCI are younger than 30 years of age and predominantly male.[4] Because these individuals are either planning to continue to exercise or start to compete in recreational or elite Para sport, it is important to know what potential challenges they might face following SCI.[5]

Damage to the spinal cord results in loss or impairment of sensorimotor function that presents as paralysis (ie, lost or diminished motor control) and impaired/abolished sensation. Individuals with SCI are also prone to developing secondary complications arising from damage to the autonomic nervous system (ANS). Because ANS-related impairments heavily impede QoL, recovery of ANS function is ranked among the top priorities for individuals with SCI.[2] The ANS is a control system that acts largely unconsciously and regulates multiple body functions including blood pressure (BP), heart rate (HR), breathing rate, voiding, bowel motility, and sexual arousal. Human athletic performance depends on a coordinated and fully functioning ANS.[6] According to a consensus statement from the International Olympic Committee, impairment of athletic performance "is manifested by an inability to maintain strength, power, speed, endurance, and consequently sport-specific neuromotor skill performance, prompted by premature fatigue as a result of a complex process involving multiple physiologic systems and mechanisms."[7] Therefore, loss or diminished autonomic control following SCI can have substantial negative impact on athletic performance, ranging from poor performance caused by fatigue, to life-threatening conditions.[8] For that reason, it is crucial to recognize these factors when recommending physical/exercise activities for individuals with SCI. These issues become even more obvious when individuals with SCI are involved in competitive sport. This article presents evidence that the athletic performance of individuals with SCI is affected by ANS dysfunction associated with SCI. Also discussed are factors compromising the athletic performance.

ORGANIZATION OF THE AUTONOMIC NERVOUS SYSTEM

Classically, the ANS is subdivided in two components: the sympathetic and parasympathetic nervous systems. Both sympathetic and parasympathetic ANS possess central and peripheral nervous system components. Lately, the autonomic neuronal circuits within the gastrointestinal tract have become recognized as a separate component of ANS, known as the enteric ANS.[9] The sympathetic nervous system is known to be responsible for the fast-physiologic changes in the body (eg, fight-or-flight response). Sympathoexcitatory neurons located within medulla oblongata provide tonic input via descending spinal pathways to the spinal sympathetic preganglionic neurons (SPNs).[10] The SPNs are predominately located within the lateral horn of the spinal cord from T1 to L2. Axons of these neurons leave the spinal cord through its ventral roots. Thereafter, with the exception of SPNs directly synapsing onto the adrenal glands, most SPNs conjunct with postganglionic neurons of the sympathetic chain ganglia, which are located ventrolateral to the vertebral column. The sympathetic postganglionic fibers extend to end organs (as known from animal studies), such as the heart (T1-5), gastrointestinal tract (T6-11), kidney (T10-12), lower urinary tract, and reproductive organs (T10-L2) and associated blood vessels of the upper (T1-5) and lower body (T6-L2) (**Fig. 1**).

The parasympathetic nervous system is predominantly a slower system and mostly acts while the body is at rest (eg, after eating; known as "rest and digest" responses).