The Role of Autonomic Function on Sport Performance in Athletes With Spinal Cord Injury

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Devastating paralysis, autonomic dysfunction, and abnormal cardiovascular control present significant hemodynamic challenges to individuals with spinal cord injury (SCI), especially during exercise. In general, resting arterial pressure after SCI is lower than with able-bodied individuals and is commonly associated with persistent orthostatic intolerance along with transient episodes of life-threatening hypertension, known as "autonomic dysreflexia." During exercise, the loss of central and reflexive cardiovascular control attenuates maximal heart rate and impairs blood pressure regulation and blood redistribution, which ultimately reduces venous return, stroke volume, and cardiac output. Thermoregulation also is severely compromised in high-lesion SCI, a problem that is compounded when competing in hot and humid conditions. There is some evidence that enhancing venous return via lower body positive pressure or abdominal binding improves exercise performance, as do cooling strategies. Athletes with SCI also have been documented to self-induce autonomic dysreflexia before competition with a view of increasing blood pressure and improving their performance, a technique known as "boosting." For health safety reasons, boosting is officially banned by the International Paralympics Committee. This article addresses the complex issue of how the autonomic nervous system affects sports performance in athletes with SCI, with a specific focus on the potential debilitating effects of deranged cardiovascular control.

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INTRODUCTION

Spinal cord injury (SCI) results in an array of dysfunctions that extend far beyond paralysis. In particular, autonomic dysfunction and the consequent cardiovascular, bladder, bowel, temperature, and/or sexual dysfunction that ensue impede quality of life and are among the highest priorities of recovery [1]. For individuals with SCI who partake in competitive sport, the cardiovascular and autonomic consequences of SCI, such as blood pressure, heart rate (HR), and temperature dysregulation, are of critical importance. The degree of altered cardioautonomic control is dependent on both the level and the completeness of SCI. For example, it is well known that cervical or high-thoracic SCI is associated with lifelong abnormalities in systemic arterial pressure control [2], whereby resting arterial pressure is lower than that of individuals with mid-to-low thoracic injuries or uninjured controls [3].

Further, high-level SCI is commonly accompanied by persistent orthostatic intolerance [4,5], along with transient episodes of hypertension, known as "autonomic dysreflexia," which are often accompanied by disturbances in HR and rhythm [6]. There is recent evidence that autonomic completeness of injury, that is, the degree of disruption to the descending spinal autonomic pathways, also plays a critical role in resting cardiovascular control, whereby those with an autonomic complete injury exhibit the most-severe disruption in resting cardiovascular function independent of injury level [7]. The effect of the injury level and completeness of injury on resting cardiovascular control in individuals with SCI has been reviewed extensively elsewhere [8,9].

In able-bodied athletes, the appropriate cardiovascular responses to exercise are controlled in a large part by the autonomic nervous system. After SCI, however, disruption

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of critical supraspinal control of the heart and blood vessels alters the cardioautonomic response to exercise and may curtail endurance performance. The purpose of this article is to review the present literature available on the complex relationship between autonomic function and aerobic exercise performance and/or exercise capacity in athletes with SCI, which specifically focuses on the cardiovascular response to exercise. We recognize that exercise performance in this population is an extremely complex issue and that multiple additional factors should also be considered, which, unfortunately, are out of the scope of this review. It is anticipated that, by identifying the major autonomic dysfunction that could impact athletic performance of athletes with SCI, this review will open new avenues for research and for the classification of wheelchair athletes.

MOTOR AND AUTONOMIC CONSEQUENCES OF SCI

When injury occurs to the fragile tissue of the spinal cord, various motor, sensory, and autonomic deficits are observed below the level of the injury. An SCI is classified in terms of the level of injury and completeness of injury according to guidelines set forth by the International Standards for Neurological Classification of Spinal Cord Injury. This assessment provides a standardized score, known as the American Spinal Injury Association Impartment Scale [10]. More recently, the international SCI community has developed and revised an autonomic component to the clinical classification of individuals with SCI [11]. Conversely, sports classification ranges from a robust system of bench tests, ball skills, and observation to no classification system. To date, classification is an ongoing area of research and development for the International Paralympic Committee. Although most sports do include some form of motor and/or sensory assessment, no sports currently use a test of the cardiovascular or autonomic nervous system. This potentially leads to some athletes competing at a distinct advantage or disadvantage within their respective classes, depending on the level and completeness of their injury [12].

The degree of motor, sensory, and autonomic dysfunction after SCI is largely level dependent, whereby those with cervical SCI (tetraplegia) exhibit greater impairments than those with thoracic SCI (paraplegia) (Figure 1). In the case of a neurologically complete SCI, there will be total interruption of the usual neuronal traffic from higher brain centers via the upper motor neuron. For neurologically incomplete SCI, there is partial preservation of motor function, sensory function, or both. Neurologic completeness of injury is particularly crucial for individuals with tetraplegia who compete in competitive sport because partial preservation of hand and arm function plays a pivotal role in manual wheelchair propulsion.

A less well documented consequence of SCI relates to impairments in autonomic function. Although we still do not fully understand the autonomic consequences of SCI, it is our present understanding that autonomic dysfunctions also vary, depending on the neurologic level of the SCI. For central autonomic control of cardiovascular function, the cerebral cortex and hypothalamus project to the various nuclei within the medulla oblongata, which houses the cardiovascular control center (Figure 1). The heart is under dual control of the parasympathetic and sympathetic nervous systems; whereas the arteries are controlled primarily by the sympathetic nervous system. Parasympathetic control of the heart is via the vagus nerve (CN X), which exits at the level of the brainstem. The preganglionic fibers of the vagus nerve then synapse with postganglionic parasympathetic neurons in ganglia on or near the heart. Supraspinal descending sympathetic pathways provide tonic control to spinal preganglionic neurons that regulate cardiovascular function. The majority of spinal preganglionic neurons are found within the lateral horn of the spinal cord in segments T1-L2 and exit the spinal cord via the ventral roots. Spinal preganglionic neurons synapse with postganglionic neurons located within paravertebral sympathetic chain ganglia. These sympathetic postganglionic neurons ultimately synapse with the target organs of the cardiovascular system, the heart (T1-T5) and blood vessels (T1-L2). Accordingly, after any neurologic level of SCI, the cranial parasympathetic nervous system will remain intact, whereas the sacral parasympathetic function will always be involved. The sympathetic nervous system, however, will be affected to a variable extent that depends both on the neurologic level of injury and the autonomic completeness of the injury (Figure 1).

AUTONOMIC CARDIOVASCULAR RESPONSES TO AEROBIC EXERCISE IN SCI

Cardiac Output, Stroke Volume, and HR

Cardiac output, the product of HR and stroke volume, is controlled by a complex array of central (autonomic), reflexive, humoral, and local mechanisms. During maximal exercise, cardiac output increases up to 7-fold in highly trained able-bodied athletes. Conversely, the only study that has investigated the cardiac output response to maximal arm exercise in SCI found that individuals with cervical SCI exhibit less than a 2-fold increase in cardiac output during maximal aerobic exercise [13]. A similarly attenuated cardiac output also has been noted during submaximal functional electrical stimulation cycling exercise in cervical but not thoracic SCI [14]. This limited rise in cardiac output in cervical SCI is due both to reduced stroke volume and HR, each of which is discussed below.

In healthy, able-bodied individuals, the stroke volume response to aerobic exercise is generally considered to increase up to approximately 50% of maximal oxygen uptake and then plateau, after which increases in cardiac output are mediated by HR [15]. Although there has been some