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REVIEW

Autonomic function as a missing piece of the classification of Paralympic athletes with spinal cord injury

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Objective: When someone suffers a spinal cord injury (SCI) many organs, including those of the cardiovascular (CV) system, cease to be controlled by the autonomic nervous system (ANS). Response to physical activity fails to meet the needs of the body and typically results in low blood pressure (BP), and in turn, reduced endurance and performance. This study examines the effect of SCI on the ANS of elite athletes and possible effect on their CV functions and ultimately their performance. The study also provides input on evidence of boosting and the current classification system. Finally, authors are exploring a possibility for future research in assessing whether consideration of ANS function would strengthen current Paralympic classification systems.

Study design: MEDLINE, SportDiscus, Embase databases and the official Paralympic website were reviewed. In total, 60 manuscripts and five website documents were reviewed.

Result: Athletes with high-level SCI affecting the ANS have limited ability to regulate their heart rate and BP in response to exercise. According to current Paralympic classification systems, these athletes are grouped with competitors who have similar motor control but intact ANS, thereby potentially putting them at a disadvantage within their own classification category. High-level SCI athletes with ANS dysfunction are also the only athletes who experience episodes of autonomic dysreflexia (AD). Whereas AD is a state of uninhibited sympathetic discharge, it is called 'boosting' when intentionally induced during competition. Boosting has been shown to improve sporting performance but can also cause serious complications due to extreme rises in BP. Therefore, boosting has been banned by the International Paralympic Committee (IPC). Despite this ban some elite high-level SCI athletes continue to boost. The IPC recognizes that the current classification systems are not the gold standard and further work is needed to create a more evidence-based classification.

Conclusions: Further research is needed to determine if the inclusion of ANS parameters contributes to strengthen classifications systems in Paralympic sports. This includes the development of a simple, valid and reliable bedside assessment of autonomic function that can be used to reliably compare athletes with or without ANS dysfunction thereby enabling further research into the isolated effect of ANS dysfunction on sporting performance. Researchers who are studying individuals with SCI, and who have CV parameters as their outcomes, should ensure a homogenous study group by the presence or absence of ANS function in addition to level of lesion so as to eliminate the potential for confounding variables that lead to inaccurate interpretation of results.

Introduction

An even playing field for athletes is a major intention of the classification system developed for Paralympic sports.[1]

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The International Paralympic Committee (IPC) recognizes that the current classification system is not perfect, and that ongoing effort must be made to ensure fair, evidence-based classification.[2] Classification is very complex and a sport-specific process. However, the primary focus, with the exclusion of the categories for visual or intellectual impairment, is to use remaining or impaired motor control as the defining criterion to group athletes into categories.[1]

Exclusive use of motor function for classification may be perceived as a limitation of current Paralympic classification systems as this approach does not take into account conditions that affect the autonomic nervous system (ANS), which is known to control heart rate (HR), blood pressure (BP), and other factors, which could negatively impact sporting performance.[3–6] This creates a unique challenge for a specific population of Paralympians: athletes who have a spinal cord injury (SCI) and consequently impairment of autonomic control. In all sports, these athletes are grouped together with individuals who have similar motor control but intact ANS, thereby potentially putting them at a disadvantage.[1] Furthermore, among the athletes with SCI there is significant variability of autonomic dysfunctions that are presently not taken into consideration during IPC classification.

To further complicate matters, athletes with SCI who are potentially at a disadvantage due to ANS dysfunction are also the only athletes who are able to 'boost.'[7] Boosting is a state of intentionally induced autonomic dysreflexia (AD) by an athlete during training or competition.[8] Boosting has been shown to increase sporting performance compared with unboosted states.[7,9,10] However, there are significant health risks associated with boosting, as an increase in BP during AD can cause serious adverse health events such as intracerebral bleeds, seizures, myocardial ischemia and even death.[11–18] Therefore the practice of boosting has been banned in the Paralympics.[19,20]

The purpose of this study is to explore the complex issue of the impact of ANS dysfunction in sports for persons with SCI by, (1) describing the effect of SCI on the ANS, (2) reviewing how the current classification system can create disadvantages within the categories that include athletes with SCI affecting the ANS, (3) considering how ANS dysfunction can impact sporting performance during boosted and unboosted states, (4) exploring the evidence on frequency of use of boosting in sport and (5) discussing the IPC's position on the practice of boosting and the current classification system. A review of the existing literature involving athletes with SCI and documentation from the IPC was performed in order to help guide future work in this area.

Methods for literature review

We conducted a keyword literature search of articles, practice guidelines, and review articles published between 1980 and December 2009 in English and included the internet databases MEDLINE, SportDiscus and EMBASE. The keywords 'spinal cord injury', 'paraplegia' and 'tetraplegia' were combined with 'blood pressure', 'heart rate', 'exercise', 'boosting', 'autonomic dysreflexia' and 'sport performance.' Abstracts were reviewed for identification of information on trained athletes with SCI as subjects. The IPC documents were accessed from the website http://www.paralympic.org.

Effect of SCI on the ANS

In order to appreciate the complexity of the current Paralympic classification of athletes, a basic understanding of the effects of SCI on the human body is essential. Injury to the spinal cord is typically associated with loss of motor and sensory functions. However, SCI can also lead to an interruption of pathways of the ANS from its central origins in the brain to the peripheral organ innervation, thus affecting autonomic control of various organs, including those of the cardiovascular (CV) system (Figure 1).[5,6,13,21]

The ANS is comprised of both the parasympathetic and sympathetic nervous system. With respect to CV control, the parasympathetic is prominent during restful states and is mediated through the vagus nerve, acting to decrease HR. Vagal control of the heart originates from the medulla and is usually spared following SCI.[5,6,21] The vagus nerve innervates only the heart with no effect to blood vessels. The sympathetic nervous system innervates both the heart muscle and smooth muscles within peripheral blood vessels, and is predominant in times of physiological stress and exercise, acting to increase HR and BP.[5,21] Sympathetic innervation to the heart and the majority of blood vessels for the upper extremity originates from the upper thoracic segments (T1–T5); the vasculature beds in the gut and lower extremities are under the control of the more caudal T6–L2 spinal sympathetic neurons.[5,22,31,32] Injury below T6 tends to spare cardiac and most blood vessel control, whereas SCI above T6 can interrupt supraspinal sympathetic control to the heart, after which spinal circuits become solely responsible for the generation of sympathetic activity below the level of injury.[5,6,22] For the purpose of this paper, the term 'high-level SCI' will denote an injury above T6. A high-level SCI affecting the ANS results in low resting sympathetic tone below the level of injury, with mostly unopposed parasympathetic (vagal) tone to the heart. This leads to lower levels of BP and HR at rest and abnormal CV responses to physiological stressors including exercise, which will be discussed later in this review.

Until recently, completeness of SCI was assessed by the residual motor and sensory functions with use of the American Spinal Injury Association (ASIA) Impairment Scale (AIS).[23] However, this scale does not necessarily reflect the degree of damage to the ANS as there can be preservation of autonomic function in those with motor and sensory-complete SCI. Conversely, there can be loss of autonomic function in those with motor and sensory incomplete SCI.[24–26] The need for an evaluation and documentation of residual autonomic functions following SCI has been recognized by clinicians and scientist worldwide. As a result, a new system to document the impact of SCI on autonomic function was recently proposed.[27]

The current Paralympic classification and its implications for SCI athletes with ANS dysfunction

The term ''classification'' for participants in Paralympic events refers to the ''process by which athletes are assessed by reference to the impact of impairment on their ability to compete in… (a specific sporting event)''.[1] Classification is undertaken to allow for fair competition and to ensure that the strategies, skills and talent of the athlete determine
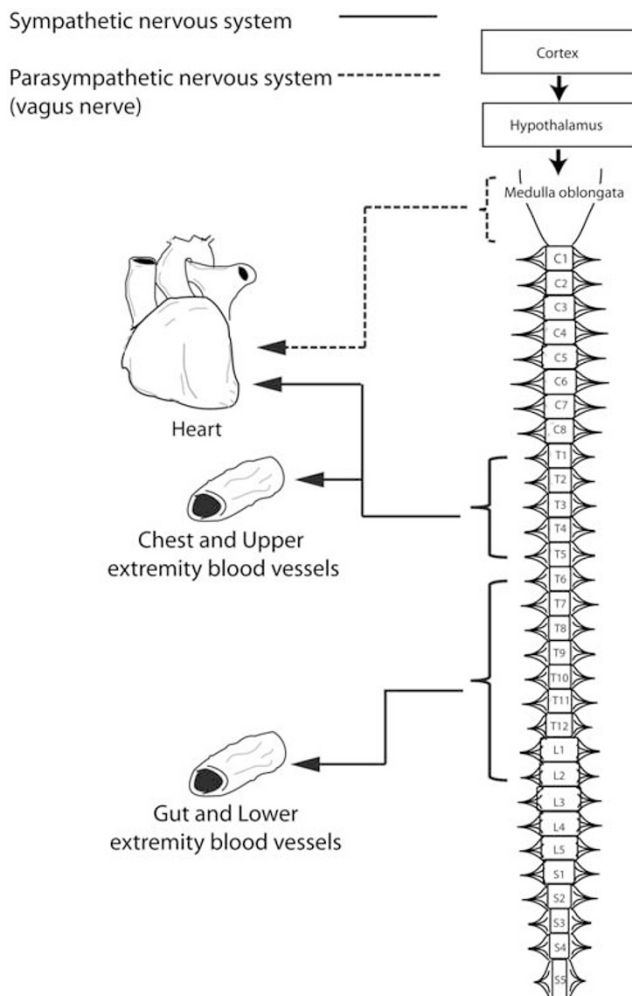
Figure 1 Schematic diagram of autonomic control of the CV system. The cerebral cortex and hypothalamus provide tonic and inhibitory inputs to the various nuclei within the medulla oblongata, where CV control is coordinated. Parasympathetic control of the heart occurs via the vagus nerve (vagus N) that exits at the level of the brainstem (interrupted line). The preganglionic fibers of the vagus N then synapse with postganglionic parasympathetic neurons in ganglia on or near the target organ. Descending sympathetic pathways provide tonic control to spinal sympathetic preganglionic neurons (SPNs) involved in CV control. SPNs are found within the lateral horn of the spinal cord in segments T1–L2 and exit the spinal cord via the ventral root. They then synapse with postganglionic neurons located in the sympathetic chain (paravertebral ganglia). Finally, the sympathetic postganglionic neurons synapse with the target organs, heart and blood vessels (solid lines). The heart receives dual innervation, parasympathetic from the vagus N and sympathetic from the upper thoracic segments of the spinal cord (T1–T5). Blood vessels in the upper portion of the body receive sympathetic innervation from the T1 to T5 spinal sympathetic preganglionic neurons, whereas the vasculature beds in the gut and lower extremities are under the control of the more caudal T6–L2 spinal sympathetic preganglionic neurons. Afferent feedback for CV system from the central and peripheral baroreceptors and chemoreceptors is not shown.

competitive success, as opposed to their underlying impairment.[1] In addition to the presence of a relevant impairment, it must be demonstrated that the impairment impacts sport performance.

Based on the most recent IPC Classification Code (2007),[1] classification for Paralympians is performed by a classification panel. This panel allocates the athlete to a sport class, thereby determining eligibility to compete in Paralympic sports. The assessment is primarily comprised of four parts: (1) review of medical documentation of the impairment; (2) physical examination to establish the presence of an impairment, which allows the athlete to participate in the Paralympic sport; (3) technical assessment in which the classifiers observe the athlete perform sport-specific tasks in a simulated environment to determine to what extent performance is limited by the impairment; and (4) observation assessment in which the classifiers observe the athlete performing sport-specific skills during a training session or competition.

For the non-vision or non-intellectual impaired, the athlete population of interest for this review, every sporting event has a different classification. However, all the classifications have an exclusive focus on motor function of the arms, trunk and lower extremities. Some sports also consider deficits in skin sensation.[1]

Present classification does not take into account the ability to modulate the functions of heart, blood vessels and other sympathetically controlled organs due to underlying ANS impairment.[1] For example, in the wheelchair racing classification there are four sport class profiles, which are based on loss of strength in the arms, trunk and legs as assessed during the physical examination session.[2] The technical and observation assessments examine how these impairments affect leg positioning, arm function and trunk stability in the sport. The sport class is used to adjust for the level of impairment of the athlete to allow comparison across competitors.

Using the classification for wheelchair basketball as an example of a team sport, each athlete is assigned a score from 1 to 4.5 based on trunk, limb, and hand function.[33] The higher the motor impairment, the lower the number. At any given time in a game, the five players on court must not exceed a total of 14 points. This ensures a mix of individuals with motor impairments that are comparable across teams during the competition.

Within the actual classification systems, individuals with high-level SCI whom we know are at risk for ANS dysregulation are grouped into the same category as individuals with similar motor function but intact ANS regulation. Therefore, the effect of SCI on exercise responses in SCI athletes with impaired ANS compared with those with intact ANS must be explored.
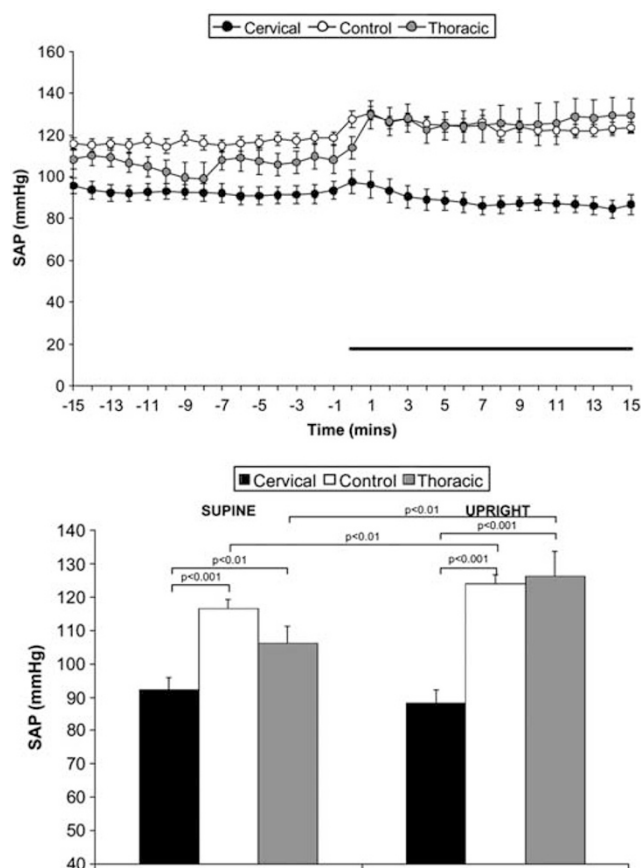
Effect of SCI on elite athletes' exercise performance

The important components of CV control needed for exercise performance include appropriate BP and HR regulation and reflex response to exercise.[29,34] This requires appropriate sympathovagal balance in regulation of the CV system, resulting in sufficient blood redistribution to the muscles during exercise. This is possible only with proper sympathetic control of the cardiac and regional blood vessels

in addition to skeletal muscle pump activity.[4,13,34] Furthermore, appropriate catecholamine release by peripheral sympathetic nerves is also paramount for maintaining good muscle activity during exercise.[35] Other systems also influence exercise performance, including respiration, muscular function, temperature and sweat regulation.[4,34] Unfortunately, all of these systems, including autonomic control of the CV system, are commonly affected by SCI and the extent of dysfunction of these systems depends on the level and severity of the injury.[24,28,36,37]

The current Paralympic athlete classification emphasizes motor function and does not take into account CV dysfunction or other systems under autonomic control. In order to reconsider the current classification system, we need to take a careful look at what CV dysfunction is experienced by SCI athletes and how it could affect exercise performance.
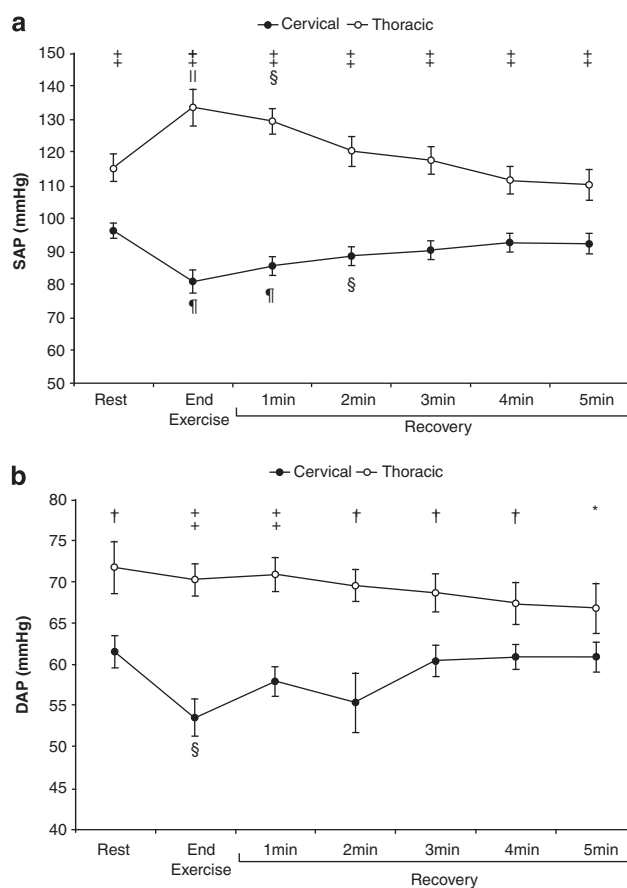
It is well known that following SCI, baseline resting BP is inversely correlated with the level of injury; individuals with high level SCI have resting systolic BPs on average 15–20 mm Hg lower than able-bodied individuals (Figure 2).[13,26] A similar pattern can also be seen with HR,

resulting in a stroke volume that is lower in individuals with higher levels of SCI.[26,38] There is also peripheral pooling of blood in the lower extremities due to decreased sympathetic tone, in addition to altered muscle pump activity of the skeletal muscles below the level of injury.[39] In general, the most commonly described phenomenon in response to exercise in high-level SCI individuals is poor HR and BP response. Furthermore, individuals with SCI also are prone to develop a post-exercise-induced hypotension that could further affect their performance (Figure 3).[40]

In the able-bodied population, higher fitness levels have been related to improved autonomic CV control, and conversely, physical inactivity has been associated with reduced autonomic CV tone.[41,42] Also, a low resting HR and high vagal tone have been reported in endurance-trained able-bodied athletes when compared with sedentary able-bodied individuals, suggesting physical activity is a

Figure 2 Systolic arterial pressure (SAP) responses to the orthostatic stress in individuals with cervical, thoracic SCI and able-bodied volunteers. The upper panel shows mean data for each group, every minute, for the duration of the test; the horizontal line denotes the upright seated portion of the test. The lower panel shows the average values of SAP for each group when supine and upright. Supine SAP was lower in cervical SCI than the other two groups ($P < 0.01$). When upright, SAP decreased in 50% of cervical SCI (-15 ± 3 mm Hg, $P < 0.05$) (with permission from Claydon and Krassioukov[26]).

Figure 3 Systolic (SAP, a) and diastolic (DAP, b) arterial pressure responses to exercise. Resting seated SAP and DAP were significantly higher in individuals with thoracic than those with cervical SCI. After exercise, SAP was increased in individuals with thoracic SCI, but was decreased in those with cervical SCI. After exercise, DAP was unchanged in individuals with thoracic level SCI, but was decreased in those with cervical SCI. During the recovery from exercise, BPs began to return to the resting levels in both groups, but were consistently elevated in individuals with thoracic compared with cervical SCI. *$P < 0.05$; †$P < 0.01$; ‡$P < 0.001$ cervical versus thoracic; §$P < 0.05$; ¶$P < 0.001$ within-group comparison against the resting condition. Note the change in scale in the lower panel (with permission from Claydon et al.[40]).

modulator of resting vagosympathetic cardiac control.[43,44] Athletes with SCI have also demonstrated some ability to improve CV modulation through exercise,[44,45] although not to the extent of athletes with intact ANS.[46,47]

To address the question as to how ANS dysfunction relates to Paralympic classification, it is important to look specifically at the response to exercise in SCI athletes. Hence, the studies chosen for this manuscript were selected for individuals with high levels of fitness, either determined by the study author based on number of hours per week of training, or due to participation in a national or international level sport.

These studies consistently show that SCI athletes with injuries above T6 have lower maximal HR (due to altered sympathetic tone and lower catecholamine release),[48–51] lower maximal oxygen uptake (VO_2)[48,50] and lower peak power (W)[48,49] in response to submaximal and maximal exercise in comparison with athletes with lower levels of SCI and athletes with intact ANS. It is also known that athletes with cervical SCI have smaller heart volumes in comparison with able-bodied athletes with similar levels of training and body size.[52] Atrophy of cardiac muscle following high-level SCI is thought to be due to a combination of factors including, (1) impaired sympathetic drive to the heart and (2) decreased venous return from lack of sympathetic vasoconstriction and muscle paralysis below the lesion resulting in disturbed redistribution of blood flow and venous pooling.[53,54] Consequently, stroke volume and cardiac output are decreased, compromising VO_2 and oxygen delivery to exercising muscles.[4] It has also been observed that athletes with cervical SCI have lower maximal HR and peak VO_2 in response to exercise compared with athletes with injury between T1 and T6.[50]

Conversely, SCI athletes with injury below T6 have intact autonomic control of the heart and are, therefore, able to modulate HR in response to exercise.[48,55,56] However, sympathetic innervation to blood vessels below the level of injury can still be impaired, resulting in a smaller stroke volume due to decreased venous return to the heart from blood pooling in the peripheries.[48,57] As a result, SCI athletes with injury below T6 compensate for a lower stroke volume with higher HR responses during exercise compared with their able-bodied counterparts, and are able to achieve a comparable cardiac output to athletes without SCI.[55,58,59] Impaired regulation of blood vessels below the mid-thoracic lesion can also lead to lower BPs in the exercise recovery phase.[58]

In addition to the level of injury there is limited evidence in the SCI athlete population that completeness of injury also affects CV responses during exercise. Data from our laboratory demonstrates that there are independent interactions between preservation of palmar sympathetic skin responses (suggestive of incomplete autonomic injury) and the upright mean BP ($r = 0.5887$, $P = 0.005$) during the orthostatic stress in individuals with cervical SCI.[26] Goosey-Tolfrey et al.[60] in a small group of athletes with cervical SCI demonstrated lower maximal HR, lower peak VO_2 and lower peak power in individuals with complete lesions compared with those with incomplete lesions. Given

our present understanding of the impact of ANS control on CV function we have to be cautious with interpretation of results when investigators group the results from AIS-complete and -incomplete injuries together for analysis.

In summary, individuals with high-level SCI, including high-level performing athletes, have altered autonomic control of CV system that can impact exercise performance. With the current Paralympic classification, athletes with intact versus impaired ANS function, but similar motor function, are placed within the same sporting class. This creates the potential for an uneven playing field between Paralympic athletes and the application of inappropriate strategies to accommodate for a loss of (physiological) function. Boosting, or voluntarily induced AD, is a practice unique to athletes with high-level SCI, and is sometimes used by this population to improve exercise performance.[7]

Boosting, the intentional induction of AD

Experimental and clinical studies demonstrate that CV control in individuals with SCI are associated with unopposed parasympathetic regulation and sympathetic hypoactivity causing lower levels of BP and HR at rest and in response to physiological stressors including exercise. Conversely, physiological stimuli that are well tolerated in able-bodied individuals can become triggers for AD, characterized by uninhibited sympathetic activity generated by spinal cord circuits following high-level SCI (Figure 4).[30,61] The unstable BP control with episodes of dangerously high BP or cardiac arrhythmias resulting from AD can occur involuntarily with both noxious and non-noxious stimuli, and is defined as the elevation of systolic BP by at least 20 mm Hg from baseline.[11,62] Boosting, by comparison, is the intentional induction of AD by a SCI athlete during training or competition.[8] There are anecdotal reports of various methods of boosting during competition, including sitting on the scrotum, clamping of the foley catheter and breaking the big toe.

Only three studies specifically observe CV responses of SCI athletes in boosted versus unboosted states. Wheeler et al.[10] and Burnham et al.,[7] both used the same eight high-level SCI athletes who were elite tetraplegic road racers. In both studies, these athletes were observed when boosting and not boosting while performing a 7.5 km race and during a graded arm exercise test to maximal effort in a supervised environment. Schmid et al.[9] studied six elite high-level SCI athletes: three wheelchair racers and three wheelchair rugby players. These six athletes were observed performing a graded arm exercise test to maximal effort using a wheelchair ergometer. In each study, boosting was achieved by either increasing fluid intake before the event in order to over distend the bladder, or by prolonged sitting in the wheelchair before the exercise protocol.

During the graded arm exercise, Burnham and Wheeler found that, when boosted, athletes experienced a significant increase in peak HR, peak BP, circulating norepinephrine levels, maximal VO_2 and peak W (Table 1). However, despite the increase in peak HR, cardiac output was unchanged
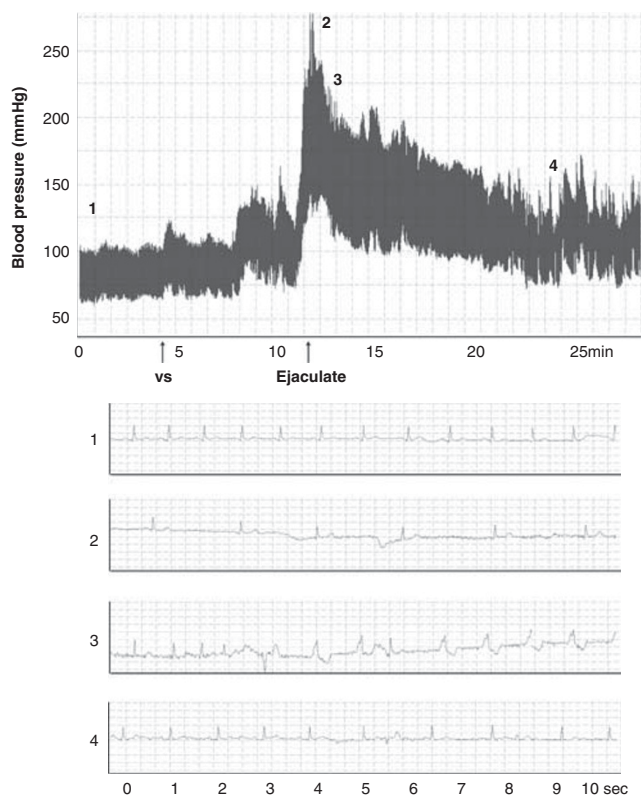
Figure 4 Changes in BP and HR in men with cervical spinal cord injury (C7 AIS (American Spinal Injury Association Impairment Scale) B) during the vibrostimulation (VS) procedure for sperm retrieval. BP (using a finger cuff) and three lead electrocardiogram (ECG) were recorded continuously (Finometer Pro Finapres Medical Systems, Amsterdam, Netherlands; Powerlab, ADInstruments, Bella Vista, Australia) during the procedure. BP (top diagram) during the procedure and four 10 s samples of ECG recorded during different periods of the procedure (bottom diagrams) are shown (1—at rest; 2—at ejaculation; 3–3 min following ejaculation; 4–20 min following ejaculation). Before VS (rest, recording 1) there was relative hypotension (100/65 mm Hg) with a regular HR of 78 bpm. With initiation of VS (first arrow) there was a gradual increase in arterial BP suggestive of a typical episode of AD. Finally, at the time of ejaculation arterial BP surged to 280/150 mm Hg accompanied by bradycardia at 38 bpm (recording 2) and a short run of premature ventricular contractions (PVCs) was observed 3 min following ejaculation (recording 3). At 15 min following ejaculation arterial BP was still slightly elevated (130 mm Hg, HR 66 bpm, recordings 4). During the next 20–25 min arterial BP and HR gradually returned to resting values. This episode of AD was accompanied by significant spasms in the upper and lower extremities, profuse sweating on the forehead and neck, and piloerection on the forearms. Interestingly, during this episode of AD the patient reported only a mild headache (personal observations) (with permission from Krassioukov[61]).

because of the significantly lower stroke volumes that occurred in the boosted state. In addition, there were no boosting-related changes seen in metabolism, as represented by levels of free fatty acids, glucose and lactate concentration. With respect to exercise performance, during the 7.5 km wheelchair race there was a significant decrease in racing time in the boosted (22.6 ± 6.6 min) compared with the unboosted state (25.6 ± 9 min), which translated to a mean performance increase of 9.2%.[7] There was also a lower rating of perceived exertion by the athletes during the boosted state.

These athletes also filled out a questionnaire on the practice of boosting.[7,10] All of the athletes reported having used boosting to improve performance during competition. Subjectively, they felt that boosting increased arm strength and endurance, decreased arm stiffness, improved breathing, and increased alertness and aggressiveness. Half of the individuals also reported that they could 'boost too much' and 5/8 felt they could not boost predictably. Side effects frequently experienced with boosting included headaches and excessive shivering and sweating. All of the athletes believed that the practice of boosting for the purpose of performance enhancement is widespread among competitive high-level SCI athletes.

In comparison with these eight tetraplegic wheelchair racers, all of the high-level SCI athletes in Schmid et al. ($n = 6$) denied using boosting to improve performance during a race.[9] However, many had experienced side effects of AD during training, and all had heard of boosting. In these athletes, responses to exercise during the boosted state in comparison with the unboosted state also showed that there was a significant increase in peak HR, peak BP, circulating norepinephrine levels, maximal VO_2 and peak W (Figure 5). These changes are consistent with those demonstrated in the studies by Wheeler et al. and Burnham et al. Unfortunately, none of the three studies had a control group with similar level of lesion and training but with intact sympathetic nervous system control to determine how much boosting compensated for the impact of autonomic dysregulation on exercise performance.

Blood pressure responses in Burnham et al. and Schmid et al. were significantly elevated in the boosted state, reaching as high as over 200 mm Hg systolic (Table 1).[9,10] Although limited, there is data showing improved performance with boosting in high-level SCI athletes, therefore it is very important to be aware of the potential dangers of uncontrolled AD. There have been no documented consequences directly linked to boosting in competition to date. However, there are numerous reports of significant consequences in clinical situations, including myocardial infarction and seizures.[15–18,63]

Due to potential health risks, the IPC and other governing bodies have struggled with how to address the issue of boosting.[64] Although boosting could be used to enhance performance, significant controversy arose as to whether boosting should be considered as a form of doping, mainly because doping at the time was considered to be 'the administration of or use of any substance foreign to the athlete's body, or of any physiological substance taken in abnormal quantity or taken by abnormal route of entry in to the body with the sole intention of artificially increasing performance in competition'.[64] Adding to the controversy is the difficulty in proving whether episodes of high BP identified during competition are intentionally induced, as spontaneous episodes of AD are a common occurrence in individuals with high level SCI.[65]

Concern for the potential health risks due to uncontrolled high levels of BP led the IPC to officially ban boosting in the Paralympic movement for health safety reasons.[19] Screening for AD in athletes before the competition was first instituted

Table 1 Boosting safety: BP and body temperature response during 7.5 km simulated race in elite tetraplegic athletes

	Pre-race		Mid-race		Post-race	
	Boosted	Unboosted	Boosted	Unboosted	Boosted	Unboosted
Blood pressure (mm Hg)						
Systolic	155 (20)	106 (16)[a]	138 (23)	105 (18)[a]	132 (32)	99 (19)[a]
Diastolic	93 (22)	61 (11)[a]	78 (18)	59 (6)	62 (21)	55 (8)
Mean arterial pressure	114 (19)	76 (9)[a]	98 (19)	74 (7)[a]	85 (23)	69 (8)
Temperature (°C)	35.3 (0.7)	35.7 (0.6)			36.6 (0.7)	36.4 (0.7)

Abbreviation: BP, blood pressure.
There was significant increase in BP during boosted states, which were associated with improvement in performance during racing (measured as time to complete race, with permission from Burnham et al.[7]).
Numerals within parentheses indicate s.d.
[a]$P < 0.05$.
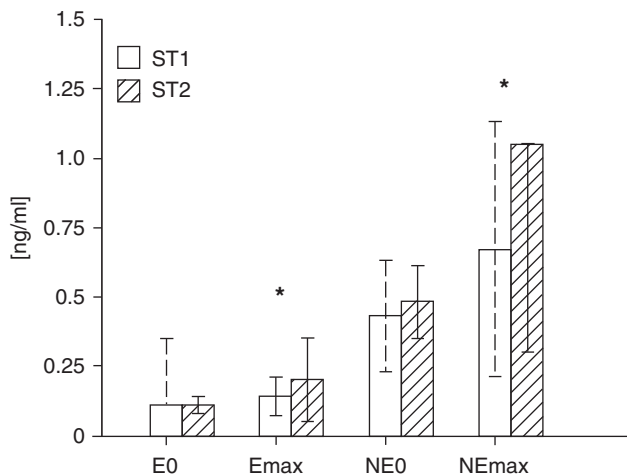
Figure 5 Epinephrine and norepinephrine levels at rest (E0, NE0) and maximum exercise (Emax, NEmax) in the incremental wheelchair exercise test without (ST1) and with (ST2) AD. Although no significant difference was found for epinephrine and norepinephrine at rest in both groups, there was a significantly higher value for epinephrine (0.20 ± 0.15 vs 0.14 ± 0.07 ng ml^{-1}) and norepinephrine (1.05 ± 0.75 vs 0.67 ± 0.46 ng ml^{-1}, *$P < 0.05$) after exercise with AD (ST2) (with permission from Schmid et al.[56]).

in the 1996 Atlanta Summer Paralympic Games and has continued since.[64] To screen for AD, IPC medical staff check athletes for signs of AD before a race or event, and take BP measurements. If the BP is significantly elevated (>180 mm Hg systolic), the athlete is given time to rest and attempt to lower their BP, which is subsequently re-measured.[19]

At the 2008 Beijing Summer Paralympic games, Bhambani et al. examined the perception of boosting using a self-reported questionnaire in Paralympians with SCI.[20] Of the 99 participants, a majority were involved in wheelchair rugby (54.2%) and familiar with the term boosting (54%). Only 10 participants admitted to boosting (wheelchair rugby (55.5%), wheelchair marathon (22.2%) and long distance racing (22.2%)). Individuals who responded positively on use of boosting during the competition had SCI at T6 or above (80%). The results from this study show that boosting

continues to be practiced by high-level SCI athletes despite the recognized health risks and IPC ban.

Conclusion

Athletes with high-level SCI and resulting ANS dysfunction present a unique challenge for a fair classification system for athletes who wish to participate in the Paralympic games. Complexity and variations in autonomic disorders make this process very challenging. Presently, classes are determined by a variety of processes that may include a physical and technical assessment, and observation in and out of competition. By focusing on assessment of motor function of Paralympians, we may be placing SCI athletes with significant ANS abnormalities at a disadvantage to competitors with similar motor function but preserved autonomic control. Additionally, the potential use of boosting by high-level SCI athletes in spite of the ban by the IPC further emphasizes the need for a review of the current approach. The IPC recognizes that the present classification system is not the gold standard and that further research is required to create a classification system that is more evidence-based.[1] Given that the principle of fair play is important in Paralympic sport, this issue needs to be addressed.

In order to determine whether Paralympic classification should take autonomic dysfunction into account, a simple, easily implementable standardized autonomic assessment of athletes with SCI needs to be established. Additionally, this assessment should be used to reliably compare athletes with or without ANS dysfunction, thereby enabling further research into the isolated effect of ANS dysfunction on sporting performance. Finally, researchers who are studying individuals with SCI, and who have CV parameters as their outcomes, should ensure a homogenous study group by the presence or absence of ANS function in addition to level of lesion so as to eliminate the potential for confounding variables.

Conflict of interest

The authors declare no conflict of interest.

Acknowledgements

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