Cardiovascular Function in Individuals with Incomplete Spinal Cord Injury: A Systematic Review

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Background: There is a clear relationship between the neurological level of spinal cord injury (SCI) and cardiovascular function; however, the relationship between completeness of injury and cardiovascular function is less straightforward. Traditionally completeness of injury has referred to neurological (motor/sensory) completeness. Recently, a number of studies have started to investigate autonomic completeness of injury. **Objective:** To investigate the relationships between cardiovascular function and neurological and autonomic completeness of injury. **Methods:** A literature search was conducted in November 2012 through MEDLINE, Embase, and CINAHL. Twenty-one studies were included in this review. **Results:** In acute SCI, there is no clear consensus about whether resting heart rate (HR), blood pressure (BP), or prevalence of BP abnormalities differs between neurologically complete and incomplete SCI. In chronic SCI, there is limited evidence that there is less prevalence of autonomic dysreflexia and improved heart rate variability in response to provocation in neurologically incomplete SCI; however, resting HR and BP appear similar between neurologically complete and incomplete SCI. There is growing evidence that BP and HR at rest and during orthostasis is enhanced in autonomically incomplete SCI. Numerous studies report that neurological completeness does not agree with autonomic completeness of injury. **Conclusions:** For acute SCI, there is no clear consensus whether cardiovascular function differs between complete and incomplete. For chronic SCI, the studies to date suggest that autonomic completeness of SCI is more strongly related to cardiovascular function than neurological completeness of injury. Thus, clinicians and scientists should account for autonomic completeness of injury when assessing cardiovascular function in SCI. **Key words:** autonomic nervous system, blood pressure, completeness of injury, heart rate

Spinal cord injury (SCI) affects 250,000 individuals in North America, with approximately 11,000 new cases per annum.¹ The lifetime health costs of an individual with cervical SCI can be as high as \$3.3 million.² Much of this cost is related to the treatment of secondary complications arising from the SCI. One of the most debilitating secondary consequences of SCI is alterations in autonomic cardiovascular control that, combined with paralysis, predispose individuals with SCI to a higher incidence of cardiovascular disease (CVD).³ This predisposition to CVD not only results in early mortality but also reduces quality of life.

It is now well recognized that the extent of impairment in cardiovascular function is related to the neurological level of injury,⁴ whereby persons with cervical/high-thoracic injuries exhibit a greater impairment in cardiovascular function and an earlier onset of CVD than those with low-thoracic/lumbar injuries. Injury to the cervical/upper thoracic cord disrupts descending input

to the sympathetic preganglionic neurons that control the critical splanchnic bed resulting in neurogenic shock during the early stages of SCI and long-lasting resting hypotension post injury.⁵ Loss of supraspinal tonic inhibitory input to the sympathetic preganglionic neurons provides an environment in which sensory-evoked reflexes can cause unopposed below-lesion sympathetic activation, leading to profound vasoconstriction and consequent episodes of hypertension. This condition, termed autonomic dysreflexia (AD), affects up to 90% of individuals with motor complete cervical SCI⁶ and can lead to a sequelae of cardiovascular effects, such as myocardial infarction,⁷ intracranial hemorrhage,⁸ and even death.⁹

Whilst the relationship between lesion level and cardiovascular dysfunction is well established, the

relationship between the severity (ie, completeness) of SCI and cardiovascular control is less clear. Historically, completeness of injury has referred to motor/sensory (neurological) completeness of injury, as assessed during the well-established neurological examination that is conducted in accordance with the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI).¹⁰ More recently, however, a number of researchers have started to investigate the concept of autonomic completeness of SCI and its effect on various bodily functions following injury. There is still no consensus on the best method to assess autonomic completeness of injury. Recently, International Standards to Document remaining Autonomic Functions after SCI (ISAFSCI) have been developed and revised.¹¹ It is now recommended that this assessment form is used as an adjunct with the ISNCSCI. These ISAFSCI standards, however, provide a simple documentation of autonomic dysfunction after SCI, rather than assessment of autonomic completeness of injury. Hence, there is still no assessment of autonomic completeness of injury in the clinical SCI setting, nor is there a clear understanding of the relationship between autonomic and neurological completeness of injury. A better understanding of such relationships will assist researchers in the development of appropriate levels of stratification within their studies and will assist clinicians in understanding the unique cardiovascular profiles of individuals with varying severities of SCI.

The purpose of this systematic review is to investigate the relationship between cardiovascular function and neurological and autonomic completeness of injury.

Methods

Search strategy

A literature search was conducted in November 2012 through MEDLINE, Embase, and CINAHL. The search strategy included the terms for completeness of injury and cardiovascular function in SCI, including spinal cord injury, paraplegia, tetraplegia, cardiovascular, cardiac, vascular, heart rate, blood pressure, orthostatic hypotension, autonomic dysreflexia, blood flow,

deep vein thrombosis, sympathetic nervous system, autonomic nervous system, rehabilitation, American Spinal Injury Association, American Spinal Injury Association Impairment Scale, neurological, complete, incomplete, sympathetic skin response, catecholamines, epinephrine, norepinephrine, heart rate variability, and blood pressure variability.

Study eligibility criteria

Studies were included in the review if they were original articles that reported cardiovascular function by either neurological completeness of injury or autonomic completeness of injury in humans with SCI. We only included studies that defined neurological completeness of injury according to Frankel (prior to 1992) or ISNCSCI or that defined autonomic completeness of injury using one or more of the following methods: microneurography, sympathetic skin response, catecholamines, blood pressure response to orthostasis, heart rate variability, or blood pressure variability. Studies were excluded if they were case reports, case series, or reviews. Any studies that included fewer than 3 individuals with incomplete SCI or failed to stratify their cohort by neurological/autonomic completeness of injury were omitted. Because so few studies have reported cardiovascular function by neurological/autonomic completeness of injury, we were liberal with inclusion of studies that had low methodological quality, such as no between-group statistical comparisons. We therefore included any study that provided either the baseline values for cardiovascular function or commented on such differences within the results section. Study eligibility was checked by 2 of the authors. A total of 93 studies were initially reviewed for eligibility and 21 studies were included (**Figure 1**).

Results

Acute SCI

A total of 9 studies have compared cardiovascular function between individuals with neurologically complete and incomplete acute SCI (**Table 1**).

Figure 1. Search strategy.

Three of the 9 studies provided no between-group statistical comparisons.

Heart rate

Incidence of bradycardia was investigated in 5 studies.¹²⁻¹⁶ Piepmeier et al¹² reported that incidence of bradycardia was reduced in cervical incomplete versus cervical complete injuries, although no statistical comparisons were performed. Lehmann et al¹³ reported a statistically significant reduction in incidence of bradycardia in cervical incomplete versus complete injuries. Conversely, Gonzalez et al¹⁴ and Levi et al¹⁵ reported no difference in resting heart rate (HR) between cervical incomplete and complete injuries. Huang et al¹⁶ also reported no differences in resting HR between incomplete and complete injuries, although these analyses were limited by the lack of stratification for lesion level. In the only study to investigate electrocardiographic

differences between incomplete and complete injuries, it was reported that individuals with cervical incomplete SCI exhibited a lower prevalence of ECG abnormalities (bradycardia, repolarization changes, AV block, supraventricular tachycardia, and ventricular tachycardia) than cervical complete SCI, however no statistical analyses were performed.¹²

Resting blood pressure

Six studies compared resting blood pressure (BP) between neurologically incomplete and complete acute SCI.¹⁴⁻¹⁹ Gonzalez et al,¹⁴ Huang et al,¹⁶ and Levi et al¹⁵ reported no differences in BP between incomplete and complete SCI. Conversely, Tuli et al¹⁷ and Vale et al¹⁸ reported that systolic BP (SBP)/mean arterial pressure was higher in cervical incomplete versus cervical complete SCI. It should be noted that the study by Huang et al¹⁶

Table 1. Summary of studies reporting cardiovascular function in acute neurologically complete versus incomplete SCI

Study	Level	N	Method	Outcome measures	Main findings	Statistics
Piepmeyer (1985) ¹²	C	45 (22)	Frankel	Prevalence of bradycardia and ECG abnormalities	1. Greater prevalence of bradycardia in motor complete vs incomplete. 2. Greater prevalence of ECG abnormalities in motor complete vs incomplete	No
Lehmann (1987) ¹³	C T/L	48 (17) 23 (ns)	AIS	Prevalence and time course of bradycardia and hypotension	1. Greater prevalence of persistent bradycardia in motor complete cervical SCI vs all other groups	Yes
Gonzalez (1991) ¹⁴	C	31 (13)	Frankel	Supine BP and HR	1. No differences in supine BP and HR between complete and incomplete	Yes
Levi (1993) ¹⁵	C	39 (11)	Frankel & AIS	Supine BP, HR, and other hemodynamics	1. No differences in any hemodynamic measurements between groups	Yes
Vale (1997) ¹⁸	C T	45 (25) 32 (8)	AIS	MAP, prevalence of vasopressors	1. Cervical motor and sensory complete exhibited lower MAP on admission and greater need for vasopressors	No
Ilman (2000) ²⁰	C T/L	10 (7) 4 (2)	AIS	BP in response to HUT	1. Similar drop in BP in response to HUT between cervical complete vs incomplete	No
Tuli (2007) ¹⁷	C	577 (134)	AIS	SBP, HR, and incidence of neurogenic shock	1. SBP higher in AIS C&D vs AIS B 2. HR higher in AIS C&D vs AIS A 3. Greater incidence of neurogenic shock in AIS A&B vs AIS C&D	Yes
Sidorov (2008) ¹⁹	C HT LT	55 (21) 12 (2) 22 (7)	AIS	SBP, incidence and time course of OH	1. No differences in SBP between incomplete cervical, high-thoracic, and low-thoracic SCI 2. Greater prevalence of OH during first month in cervical complete vs cervical incomplete	Yes
Huang (2011) ¹⁶	N. comp N. incomp	48 72	AIS	SBP, DBP, HR, incidence of AD	1. No differences in any variable between motor complete and incomplete	Yes

Note: AD = autonomic dysreflexia; AIS = American Spinal Injury Association Impairment Scale; BP = blood pressure; C = cervical; Comp = motor complete; DBP = diastolic blood pressure; ECG = electrocardiogram; HR = heart rate; HT = high thoracic; HUT = head-up tilt; Incomp = motor incomplete; L = lumbar; LT = low thoracic; MAP = mean arterial pressure; n = total number of participants (values in parentheses represent number of incomplete participants); N. comp = neurological complete; N. incomp = neurological incomplete; ns = not stated; OH = orthostatic hypotension. SBP = systolic blood pressure; T = thoracic.

was limited by the grouping of lesion levels when the sample was stratified by completeness of injury, and the study by Vale et al¹⁸ was limited by no statistical comparisons. Sidorov et al¹⁹ conducted a comprehensive assessment of BP responses by lesion level and completeness of injury and reported no differences in SBP between individuals with cervical, high-thoracic, and low-thoracic incomplete SCI, whereas cervical complete SCI exhibited a lower SBP than thoracic complete SCI; unfortunately, no direct statistical comparisons were made between complete and incomplete SCI with the same injury level.

Prevalence of blood pressure abnormalities

Sidorov et al¹⁹ reported that prevalence of orthostatic hypotension (OH) was reduced and persisted for less time in acute cervical incomplete versus complete SCI. Conversely, Illman et al²⁰ reported that there was a similar decline in BP in response to head-up tilt (HUT) in persons with incomplete versus complete injuries. Both studies were limited by a lack of statistical comparisons. The only study to investigate differences in the incidence of neurogenic shock reported that neurogenic shock occurred less frequently in cervical incomplete versus complete SCI.¹⁷ The only study to investigate incidence of AD reported that no differences exist between incomplete versus complete injuries; however, no distinction was made between different lesion levels.¹⁶

Conclusions for acute SCI

There is no consensus about whether resting HR, BP, or incidence of OH and AD are different between individuals with complete and incomplete acute SCI. Approximately half of the studies are limited by a lack of between-group statistical comparisons or did not separate their cohort by lesion level when stratifying by neurological completeness of injury. The 2 studies that statistically compared resting HR and BP between incomplete and complete individuals with the same level of injury reported contrasting findings. There is limited evidence from one study to suggest that individuals with cervical incomplete SCI exhibit a reduced prevalence of neurogenic shock compared

to individuals with cervical complete SCI. Based on the present literature, there is insufficient evidence to conclude whether individuals with a neurologically incomplete SCI exhibit enhanced cardiovascular function compared to their neurologically complete counterparts. Thus, the management of cardiovascular function in acute SCI should depend on clinical observation at the time of assessment and not on neurological completeness of injury.

Chronic SCI

Eight studies have compared cardiovascular function between individuals with neurological complete and incomplete chronic SCI, and 2 of these studies also stratified their sample by autonomic completeness of injury (**Table 2**).

Resting cardiovascular parameters

Five studies have compared BP in incomplete and complete SCI.^{6,21-24} Three studies reported no difference in resting SBP or diastolic BP (DBP) between complete and incomplete injuries,²¹⁻²³ whereas one study reported that the diurnal rhythmicity in BP was intact in individuals with a cervical incomplete SCI and absent in 91% of individuals with a cervical complete SCI.⁶ That same study reported that only 27% of cervical incomplete SCI exhibited AD in response to urodynamic assessment, whereas 91% of individuals with cervical complete SCI exhibited a dysreflexic response.⁶ Similarly, Helkowski et al²⁴ conducted a retrospective chart review of 332 patients with cervical SCI and found a reduced prevalence of AD in persons with incomplete cervical SCI when compared to cervical complete SCI. Only one study has compared cardiorespiratory and metabolic indices between incomplete and complete SCI and found no differences in resting oxygen uptake, carbon dioxide production, or minute ventilation between groups.²⁵ The only study to directly investigate resting cardiovascular function in individuals with neurologically incomplete SCI reported that individuals with the highest (cervical) injuries exhibited the lowest HR in the supine and seated position, along with a lower supine and seated

Table 2. Summary of studies reporting cardiovascular function in chronic neurological and/or autonomically complete versus incomplete SCI

Study	Level	N	Method	Outcome measures	Main findings	Statistics
Grimm (1995) ²⁷	C	14 (7)	AIS	HRV at rest and in response to provocation	1. Cervical incomplete exhibited a higher LF component of HRV at rest. 2. Cervical incomplete exhibited a higher LF & HF component of HRV in response to HUT. 3. Cervical incomplete exhibited a higher LF component of HRV in response to provocation.	Yes
Grimm (1997) ²²	C LT	20 (10) 10 (ns)	AIS	SBP, DBP, HR, HRV at rest and in response to provocation	1. No between-group differences in SBP, DBP, or HR at rest or in response to HUT 2. Cervical incomplete exhibited a higher LF and HF spectral component of HRV at rest and in response to provocation.	Yes
Curt (1997) ⁶	C LT	22 (11) 9	AIS	SSR, 24-h BP monitoring, urodynamic examination	1. Cervical incomplete individuals exhibited normal BP rhythmicity, whereas 91% of cervical complete individuals did not. 2. AD was evident in 27% of cervical incomplete and 91% of cervical complete.	Yes
Helkowski (2003) ²⁴	C	332 (ns)	AIS	Incidence of AD	1. Incidence of AD was higher in AIS A&B vs AIS C&D.	Yes
De Carvalho (2005) ²⁵	C	31 (12)	AIS	VO ₂ , VCO ₂ , HR, VE, RER	1. No differences in any variable between complete and incomplete at rest	Yes
Rosada-Rivera (2011) ²¹	C HT LT	20 (6) 10 (2) 9 (0)	AIS	SBP, HR, HRV	1. No differences in any variable between complete and incomplete	No
Sahota (2012) ²³	N. comp N. incomp A. comp A. incomp	17 9 12 14	AIS; BPV and NA	SBP, SV, TPR, CBFV at rest and in response to HUT	1. No differences in any variable between neurological complete and incomplete 2. Autonomically incomplete individuals exhibited an increased SBP and HR at rest and during HUT.	Yes
Ravenbergen (2012) ²⁸	N. comp N. incomp A. comp A. incomp	18 9 13 15	AIS; SSR, BPV, and NA	ECG intervals and variability	1. No differences in ECG intervals between neurological or autonomic complete and incomplete 2. T _{peak} -T _{end} and QT/VI variability was increased in autonomically complete	Yes

Note: A. comp = autonomic complete; AD = autonomic dysreflexia; A. incomp = autonomic incomplete; BP = blood pressure; AIS = American Spinal Injury Association Impairment Scale; BPV = blood pressure variability; C = cervical; CBFV = cerebral blood flow velocity; DBP = diastolic blood pressure; ECG = electrocardiogram; HF = high frequency; HT = high-thoracic; HRV = heart rate variability; HUT = head-up tilt; LF = low-frequency; LT = low-thoracic; n = total number of participants (values in parentheses represent number of incomplete participants); NA = noradrenaline; N. comp = neurological complete; N. incomp = neurological incomplete; ns = not stated; QT/VI = QT variability index; RER = respiratory exchange ratio; SBP = systolic blood pressure; SSR = sympathetic skin response; SV = stroke volume; T_{peak}-T_{end} = interval between the peak and end of the T wave; TPR = total peripheral resistance; VCO₂ = carbon dioxide production; VE = minute ventilation; VO₂ = oxygen uptake.

BP than individuals with low-thoracic SCI.²⁶ The study also reported that 23% of individuals with cervical incomplete SCI exhibited OH, whereas only 9% of individuals with thoracic incomplete SCI exhibit OH; this difference approached, but failed to reach, statistical significance.

Heart rate variability

Heart rate variability (HRV), which is used as a proxy to infer autonomic function, was investigated in 3 studies.^{21,22,27} Rosado-Rivera et al²¹ reported no difference in time-domain parameters of HRV between complete and incomplete injury in individuals with cervical or high-thoracic SCI. Grimm et al^{22,27} conducted 2 studies that investigated the frequency-domain of HRV at rest and in response to various provocation maneuvers. The initial study was limited to a relatively small sample of individuals with cervical complete and incomplete SCI,²⁷ whereas the second study included a greater sample size and included a control group of individuals with thoracic SCI.²² In both studies, the low-frequency component of HRV, which is thought to broadly reflect sympathetic activity, was increased at rest and in response to provocation in cervical incomplete compared to cervical complete SCI. Conversely, the high-frequency component of HRV, which is thought to broadly reflect parasympathetic activity, was initially reported to be no different at rest between incomplete and complete cervical SCI²⁷ but was later found to be increased in cervical incomplete.²² In response to provocation, both studies reported an increased high-frequency component of HRV in cervical incomplete versus cervical complete injuries.

Differences in cardiovascular function between autonomic complete and incomplete chronic SCI

Two studies have compared cardiovascular function between individuals with autonomically complete and incomplete SCI.^{23,28} Sahota et al²³ reported that individuals with an autonomically incomplete SCI (established via catecholamines and BP variability) exhibited a higher SBP and HR at rest and in response to HUT. Ravensbergen et al²⁸ reported that variability in the electrocardiogram

(defined using the QT variability index and the transmural dispersion of repolarization), which may provide an indication of the risk of developing lethal ventricular arrhythmias, was improved in individuals with autonomically incomplete SCI (established via sympathetic skin response [SSR], catecholamines, and low-frequency power of SBP) compared to persons with autonomically complete SCI. It is important to note that neither of the 2 studies that investigated autonomic completeness of injury further stratified their sample by lesion level; thus, the reported differences between autonomically complete and incomplete SCI may also be due to a between-group difference in lesion level. A further point of note is that when individuals in both of these studies were stratified by neurological completeness of injury rather than autonomic completeness of injury, there were no differences in any variable between neurologically complete and incomplete individuals.

Chronic SCI conclusions

For neurological completeness of injury, resting metabolic parameters and BP appear similar between individuals with a chronic cervical or high-thoracic complete and incomplete SCI. There is limited evidence to suggest that persons with incomplete cervical SCI exhibit preserved diurnal BP variations, a lower incidence of AD, and increased low- and high-frequency HRV compared to persons with complete cervical SCI. When autonomic completeness of injury is considered, there is stronger evidence that persons with autonomically incomplete SCI exhibit enhanced BP and HR control at rest and in response to HUT along with reduced variability in the electrocardiogram compared to persons with autonomically complete SCI. However, no studies have stratified their sample by autonomic completeness of injury and lesion level. Based on the present literature, autonomic completeness of injury appears to be more strongly related to cardiovascular function than neurological completeness in chronic SCI. Thus, clinicians and scientists should account for both autonomic and neurologic completeness of injury when assessing cardiovascular function in chronic SCI.

Table 3. Summary of studies investigating the agreement between neurological and autonomic completeness of injury

Author	Level	N	Outcome	Main findings
Curt (1997) ⁶	C LT	22(11) 9	AIS vs SSR	1. 3/11 patients with neurologically incomplete injury exhibited no SSR 2. No patient with neurologically complete SCI exhibited preservation of SSR.
Curt (1996) ²⁹	C T	29 (13) 41 (31)	AIS vs SSR	1. No patient with a neurologically complete SCI between C3 and T3 could elicit an SSR. 2. In patients with complete SCI between T4 and T8, palmar SSRs could be elicited. 3. In patients with complete SCI between caudal to T8, palmar and plantar SSRs could be elicited. 4. Approximately 50% of individuals with incomplete SCI exhibited no SSR. 5. No patient with AD exhibited preserved SSR. 6. In patients with incomplete SCI, only 47% exhibited normal SSRs.
Claydon (2006) ³²	C HT LT	14 (7) 7 (2) 4 (1)	AIS vs SSR	1. SSR were not reliably predicted by AIS. 2. 40% of individuals with AIS A exhibited some preservation of SSR. 3. 30% of subjects with AIS B, C or D exhibited no preservation of SSR.
Cariga (2002) ³⁰	AIS A (C3-L1) AIS B (C3-C4) AIS C (C4-T1) AIS D (C4-T1)	17 4 4 4	AIS vs SSR	1. 1/11 patients with a complete injury rostral to T6 exhibited preserved palmar SSR. 2. 1/2 patients with a complete injury at T6 exhibited preserved palmar SSR. 3. All patients with a complete injury caudal to T6 exhibited preserved palmar SSR. 4. 1/4 patients with an incomplete injury (AIS C) rostral to T1 exhibited no palmar SSR. 5. 4/4 patients with an incomplete injury (AIS C) rostral to T1 exhibited a palmar SSR.
Claydon (2006) ³¹	C T	19 (7) 8 (1)	AIS vs SSR	1. Only one individual with cervical SCI (AIS B) exhibited preserved SSR. 2. 8/8 individuals with thoracic SCI exhibited preserved palmar SSR. 3. 2/8 individuals with thoracic SCI exhibited preserved plantar SSR.

Note: AD = autonomic dysreflexia; AIS = American Spinal Injury Association Impairment Scale; C = cervical; HT = high thoracic; LT = low thoracic; SCI = spinal cord injury; SSR = sympathetic skin response; T = thoracic.

Agreement between neurological completeness of injury and autonomic completeness of injury

The majority of studies that have assessed autonomic completeness of injury have used SSR; hence, we will use SSR as our measure of autonomic completeness when comparing the agreement between neurological and autonomic completeness of injury (**Table 3**). In a series of 2 experiments by Curt et al,^{6,29} it was reported that no patient with a neurologically complete cervical SCI could elicit a palmar or plantar SSR, individuals with an injury caudal to T4 could elicit palmar SSRs, and individuals with an injury caudal to T8 could elicit palmar and plantar SSRs. It is of note that in the 1996 study Curt et al²⁹ reported that approximately half of the individuals with neurologically incomplete SCI exhibited no preservation of SSRs and no patient with AD exhibited preserved SSRs. More recently, Cariga et al³⁰ and Claydon et al^{31,32} reported a similar lesion-level dependency of SSR, although in all 3 studies it was reported that at least one individual with a neurologically complete cervical/high-thoracic SCI was able to elicit palmar SSRs. When Claydon et al³² examined the relationship between neurological and autonomic completeness of SCI, they reported that 40% of individuals with an ASIA Impairment Scale (AIS) A exhibited some preservation of SSR and 30% of individuals with an AIS B, C, or D exhibited no preservation of SSR.

Agreement between neurological and autonomic completeness of injury conclusions

Neurological and autonomic completeness of injury represent distinct neurological entities resulting in potentially unique cardiovascular profiles; hence, agreement between autonomic and neurologic completeness of injury is weak. When lesion level is considered, the agreement improves; however, 3 out of 5 studies still report that at least one individual with a neurologically complete high-thoracic/cervical SCI exhibited preserved SSRs. Similarly, all studies report that some individuals with neurologically incomplete injuries exhibit no preservation of SSRs.

Discussion

Implications of findings and suggests for future research

The relationship between completeness of injury and cardiovascular function is complex. This is partly due to the difference in location between the descending autonomic tracts and the spinothalamic and corticospinal tracts, which are assessed during neurological examination (**Figure 2**). Because the routine neurological assessment did not traditionally include any assessment of these descending autonomic tracts, it is perhaps not surprising that so many discrepancies exist as to whether cardiovascular function is different between individuals with neurological complete and incomplete SCI. Indeed, recent studies that have stratified their sample by autonomic completeness of injury all report enhanced cardiovascular function in persons with autonomically incomplete injuries. Unfortunately, no study has stratified their sample by both lesion level and autonomic completeness of injury. It is likely that the degree of remaining autonomic control will become a stronger predictor of remaining cardiovascular function as the level of injury moves cranially.

The importance of accurately documenting autonomic function has been recognized by the clinical community with the addition of an autonomic standards assessment to the ISNCSCI neurological examination.¹¹ These standards provide a simple bedside assessment of autonomic function that can be conducted at the time of neurological examination. Unfortunately, these standards do not classify individuals as autonomically complete or incomplete. Thus, it is advised that future studies that investigate cardiovascular function after SCI should include, at a minimum, the new ISNCSCI standards and ideally 1 of the 6 methods of assessing autonomic completeness of injury noted in this review. It is also of vital importance that studies that stratify their sample by autonomic completeness of injury further stratify their sample by lesion level, such that a clearer understanding of the relationship between autonomic completeness of injury,

Figure 2. A schematic diagram of a cross-section from the spinal cord with representation of the major spinal cord tracts. Three of these tracts (dorsal columns, corticospinal tract, and spinothalamic tract) are currently assessed during regular neurological examination in accordance with the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI). The descending vasomotor tracts, however, do not form part of the regular neurological examination. Since the descending vasomotor fibers are located dorsolaterally, it is common that sparing can occur even in the presence of complete neurological injury. Hence, we suggest that when the primary outcome of interest is related to cardiovascular function, an additional functional assessment of autonomic completeness of injury (eg, the sympathetic skin response [SSR]) should also be included.

neurological completeness of injury, and lesion level can be established.

Limitations

The major limitation of this systematic review is the small number of well-controlled studies that have accounted for lesion level and neurological and autonomic completeness of injury when assessing cardiovascular function. In an attempt to circumvent this limitation, we decided not to apply stringent criteria for study inclusion. Accordingly, we were unable to conduct robust statistical analyses across multiple studies. Instead, we present a descriptive analysis of differences in cardiovascular function by neurological and autonomic completeness of injury. Until

more well-controlled studies are available, we believe this descriptive method provides the best approach to synthesizing the available data on the relationship between completeness of injury and cardiovascular function.

Conclusions

In individuals with acute SCI, there is no clear consensus as to whether resting HR, BP, or prevalence of BP abnormalities differs between neurologically complete and incomplete SCI. In individuals with chronic SCI, there is limited evidence to suggest that persons with neurologically incomplete SCI exhibit less prevalence of AD and enhanced HRV in response to provocation; however, resting HR and BP appear similar between

neurologically complete and incomplete injuries. For individuals with autonomically incomplete SCI, the limited studies agree that resting cardiovascular function is enhanced. It is also clear that neurological and autonomic completeness of injury represent distinct neurological entities resulting in potentially unique cardiovascular profiles. Based on the evidence of this review, the inclusion of a test for autonomic completeness of injury is strongly encouraged when the outcome measure relates to cardiovascular function. When such a test is not available, clinicians and researchers should make use of the International Standards to Document Remaining Autonomic Function form, which is evidence-based, guideline-driven, and endorsed by the American Spinal Injury Association and International Spinal Cord Society.

Acknowledgments

Dr. C. West is supported by a Craig Neilsen Fellowship. Dr. Krassioukov is a recipient of a grant from the Canadian Heart and Stroke Foundation of Canada. Research in the laboratory of Dr. Krassioukov is supported by the Canadian Foundation for Innovation, BC Knowledge Translation Foundation, Canadian Institute for Health Research, and a grant from King Saud University.

Statement of ethics: This review article did not involve the use of human volunteers.

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