

Input–output relationships of a somatosympathetic reflex in human spinal injury

Rachael Brown · Alexander Burton ·
Vaughan G. Macefield

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Abstract

Objectives Spinal cord injury results in loss of supraspinal control of sympathetic outflow, yet preservation of spinal reflexes. Given the importance of reflex activation of sympathetic vasoconstrictor neurones to the generation of autonomic dysreflexia, we assessed the input–output relationship of the spinal somatosympathetic reflex induced by electrical activation of cutaneous afferents over the lower abdominal wall.

Methods In 13 spinal cord-injured subjects (C4–T10) we tested the hypothesis that the magnitude and duration of the vasoconstriction is directly related to the magnitude and duration of the stimulus train. Cutaneous vasoconstriction was measured with photoelectric plethysmography over a finger and toe; continuous blood pressure was recorded by radial artery tonometry, heart rate by ECG chest electrodes and sweat release by skin conductance. Four sets of trains of cutaneous electrical stimuli (20 Hz 1 s, 20 Hz 20 s, 20 Hz 1 s alternating on-and-off for 20 s and 1 Hz 20 s) were applied to the abdominal wall (10 mA) at 2-min intervals.

Results Nine subjects showed vasoconstrictor responses to the stimulus trains. On average, both the magnitude and duration of the responses were similar irrespective of the type of stimulus train.

Interpretation We conclude that there is a non-linear relationship between somatic inputs and sympathetic vasoconstrictor outputs, and argue that a sustained vasoconstriction need not imply continuous sensory input to the spinal cord.

Keywords Spinal reflex · Spinal cord injury · Electrical stimulation · Blood pressure · Cutaneous blood flow

Introduction

It has been known for over 50 years that sympathetic reflexes can be evoked by stimulation of somatic afferents. Electrical stimulation of peripheral nerves or dorsal roots induces a biphasic response comprising a short-latency and a long-latency component. The latter depends on supraspinal circuitry, as demonstrated by its abolition following high spinal transection or decerebration, whereas the short-latency component is a spinal reflex that is arranged segmentally [6, 7, 14, 23, 24]. It has been shown that electrical stimulation of thoracic dorsal roots induces reflex activation of sympathetic preganglionic neurones with stimulation frequencies of up to 20 Hz, the amplitude of the responses decreasing at higher frequencies [1]. For some preganglionic neurones single afferent stimuli could induce reflex responses after an interval of only 12 ms, but tetanic stimulation of the afferents did not lead to augmented responses [2]. The afferent axons are believed to be myelinated, given that long-latency reflex responses can be evoked at stimulation intensities of $1.5 \times$ sensory (dorsal root) threshold (T), and short-latency components at $5T$ [23], yet most studies have used maximal stimuli (e.g. $50T$), namely, intensities that also activate unmyelinated

R. Brown (✉) · A. Burton · V. G. Macefield
Spinal Injuries Research Centre,
Prince of Wales Medical Research Institute,
Sydney, NSW 2031, Australia
e-mail: rach.brown@unsw.edu.au

V. G. Macefield
School of Medicine, University of Western Sydney,
Sydney, NSW, Australia

fibres. Indeed, it can be shown from studies in the spinal cat that pronounced reflex responses could be elicited from electrical stimulation; however, the intensities needed to elicit such a response were noxious, thereby activating both myelinated and unmyelinated fibres [11, 12]. Apart from these early studies on the relationships between the intensity of the somatic inputs and the sympathetic responses in the cat, little is known of the input–output relationships for somatosympathetic reflexes. Moreover, even less is known about these reflexes in humans.

Human spinal cord injury (SCI) provides a clinical model to examine spinal somatosympathetic reflexes. Interruption of the descending vasomotor pathways by a spinal lesion leaves the spinal sympathetic preganglionic neurones below the lesion deprived of supraspinal control, yet open to sensory inputs from below the lesion. Indeed, visceral or somatic stimuli originating below the lesion can reflexly activate sympathetic vasoconstrictor neurones and cause sustained and dangerous increases in blood pressure (autonomic dysreflexia), classed as medical emergencies because of the life-threatening complications that may arise [10, 13, 17, 18, 26, 27]. Autonomic dysreflexia is more common with lesions above T6, as this is above the level of sympathetic outflow to the splanchnic vascular bed, which, because of its large volume, can cause significant displacement of blood volume and increases in blood pressure if its vessels constrict [26]. While visceral inputs, such as those from a distended bladder, are known to be potent triggers of dysreflexia, it is generally assumed that any somatic stimuli must be noxious in order to induce significant increases in blood pressure. Indeed, the term “somatosympathetic reflexes” has been used specifically to refer to the autonomic responses to noxious stimuli originating below lesion [22]. However, using brief electrical stimuli (1 s, 20 Hz) applied to the abdominal wall (below lesion), we recently showed that cutaneous vasoconstriction, along with increases in blood pressure and compensatory decreases in heart rate, could be induced by stimulus intensities that were considered innocuous when applied to able-bodied subjects [3]. Moreover, we have shown that selective activation of muscle or cutaneous nociceptors, by intramuscular or subcutaneous injections of hypertonic saline below lesion, does not cause cardiovascular signs of dysreflexia, bringing into question the existing dogma on the sensory inputs required to evoke somatosympathetic reflexes [4].

The purpose of the current study was to extend our recent work on spinal somatosympathetic reflexes by examining the input–output relationships of the vasoconstrictor responses induced by electrical stimulation of myelinated cutaneous axons. Specifically, we wanted to test the hypothesis that the magnitude and duration of the vasoconstriction was directly related to the duration of the

stimulus. Accordingly, we would expect that a 20 s train of electrical stimuli would cause a longer period of vasoconstriction than that caused by a 1 s train of identical frequency. Equally, we would expect that a train of 20 pulses delivered over 20 s (i.e. a stimulation frequency of 1 Hz) would cause a *smaller* vasoconstrictor response than that caused by delivering the 20 pulses over 1 s (stimulation frequency 20 Hz). Because of the spinal cord injury, we were able to examine the operation of these below-lesion somatosympathetic reflexes in the absence of supraspinal influences. No subject could feel the stimulus. By extension, we can also rule out any supraspinal component to these reflexes, which in the current study are spinal by definition.

Methods

Studies were performed in 13 subjects with spinal cord injury; 6 quadriplegic subjects (C4–C6; ASIA A–C) and 7 paraplegic subjects (T2–T10; ASIA A–B). Each of these subjects had stable blood pressure at rest, was not taking antihypertensive medication and did not present with ongoing infection or other co-morbidity. All participating subjects gave written (or oral and witnessed) informed consent, and the study received approval from the Human Research Ethics Committees of The University of New South Wales and Prince of Wales Hospital. Subjects were studied resting comfortably in a semi-reclined position with their eyes closed, at a comfortable ambient temperature. Care was taken to ensure no external noise interfered with the experimental procedure, so as to minimise spontaneous arousal responses. Continuous blood pressure was monitored non-invasively using radial artery tonometry (CBM-7000, Colin Corp., Japan). ECG was recorded via standard Ag–AgCl ECG chest leads and respiration was recorded via a strain gauge transducer attached to a strap around the chest (Pneumotrace, Morro Bay, CA, USA). Two markers of cutaneous sympathetic nerve activity were recorded; pulsatile changes in skin blood volume, which reflects changes in skin blood flow, were measured from photoelectric plethysmographs on the pads of the index finger and big toe (IR Plethysmograph, ADInstruments, Sydney, Australia), and sweat release was measured from changes in electrical conductance of the glabrous skin on the second and third fingers and toes (GSR amplifier, ADInstruments, Sydney, Australia).

Experimental protocol

Following a 10-min resting period, a series of electrical stimuli were delivered from a software-controlled optically isolated source (Stimulus Isolator, PowerLab, ADInstruments,

Sydney, Australia). Stimulus pulses (1.8–10 mA, 1 ms) were delivered via 1 cm Ag–AgCl surface electrodes applied bilaterally to the abdominal wall, 4–5 cm apart, midway between the umbilicus and pubis. Four different trains were used: (1) 20 Hz for 1 s (20 pulses), (2) 1 Hz for 20 s (20 pulses), (3) 20 Hz for 20 s (400 pulses) and (4) 20 Hz, alternating on-and-off for 1 s, over 20 s (200 pulses). Accordingly, we could examine (a) the effects of stimulus intensity (20 vs. 1 Hz) on the vasoconstrictor responses while keeping the number of pulses constant ($n = 20$), (b) the effects of stimulus duration by increasing the number of pulses while keeping the frequency constant (20 Hz for 1 s and 20 Hz for 20 s), and (c) the effects of repetitive stimuli of constant duration and frequency (20 Hz for 1 s delivered as a single train or as a set of ten trains delivered 1 s apart). Each stimulus was delivered 2 min following the end of the previous stimulus train, and the full set of stimuli was delivered three times. The stimulus intensity was adjusted until clear sympathetic reflex responses were seen or up to a maximum of 10 mA.

Analysis

Changes in skin blood flow were measured from the photoelectric pulse plethysmography signal, which was high-pass filtered (1 Hz) and the pulse amplitude was calculated from this derived channel. The heart rate was also calculated from this derived channel in order to avoid electrical artefacts caused by the stimulation. Vasomotor and sudomotor responses were accepted as stimulus-related only if they could be reproduced repeatedly. The amplitude and duration of the cutaneous vasoconstrictor response was calculated using Peak Parameters software (Chart 5, AD Instruments, Sydney, Australia). The maximum blood pressure and heart rate responses (>5% change) within 10 s of the stimulus were also calculated. For each subject a 10 s baseline period preceding the stimulus was averaged. For vasomotor, blood pressure and heart rate responses, there was large intra-individual variability between the same stimulus parameter, so the group average was based on mean values calculated for each subject within a particular stimulus parameter. For sudomotor responses, only the presence or absence of reproducible responses was noted. The analysis of variance was used for statistical evaluation of the data (Prism 5, GraphPad Software, USA).

Results

Our primary aim was to determine whether the magnitude or duration of an electrically evoked spinal somatosympathetic reflex, in this case cutaneous vasoconstriction, was dependent on the intensity and/or duration of the somatic

(cutaneous) stimulus. As illustrated for the C4 (ASIA A) subject in Fig. 1, brief trains of 20 Hz for 1 s, delivered in an on–off pattern for a total duration of 20 s (i.e. 200 pulses), caused a vasoconstrictor response in the foot that was no longer than that evoked by a single 20 Hz train of 1 s duration (i.e. 20 pulses). The same was true for the magnitude of the vasoconstriction. Vasoconstriction in the hand was also similar in magnitude and duration for both stimulus trains. In this subject there were large increases in blood pressure with compensatory bradycardia, which for both stimuli were similar in magnitude and duration; however, the duration of the bradycardia was longer for the 20 Hz 1 s on–off pattern in this subject. Given the pronounced blood pressure responses to mild electrical stimuli delivered below lesion in this subject, we decided not to deliver the longer stimulus train (20 Hz, 20 s).

Of the 13 subjects, 9 showed vasomotor responses to the cutaneous electrical stimulation. However, not all subjects responded to each stimulus parameter, and did not always have the full range of responses (i.e. vasomotor, sudomotor, blood pressure and heart rate). Eight subjects responded with both cutaneous vasoconstriction in the foot and increases in blood pressure. The average data from those subjects that did respond are presented in Table 1; it can be seen that there were no significant differences in the magnitude or duration of the vasoconstriction for each of the stimulus trains. The vasoconstriction was clearly not limited to the cutaneous vascular beds, as evidenced by the significant increases in arterial pressure. There were no differences in the magnitude of the systolic pressure with the different stimulus trains, except the 1 Hz, 20 s stimulus that caused only a minor increase in one subject. There was, however, a varying degree in the duration among the four different stimulus trains, with the brief 20 Hz, 1 s stimulus train causing the longest response (Table 1). The increases in blood pressure were sufficient to induce bradycardia in four subjects across three of the stimulus trains. The 1 Hz, 20 s train failed to produce any decreases in heart rate, presumably because the increase in blood pressure was inadequate to engage this vagally mediated compensation.

There were no statistically significant differences in the magnitude or duration of the cutaneous vasoconstrictor responses in the foot as a function of the stimulus train when the cohort was separated into quadriplegics and paraplegics, other than that the magnitude and duration of the vasoconstrictor response for the 1 Hz 20 s stimulus was smaller than those for the other three stimulus trains for the paraplegics. Cutaneous vasoconstriction in the hand for the quadriplegics also showed little difference in magnitude or duration as a function of stimulus train, with the percentage of vasoconstriction and (duration) for each stimulus train being: 20 Hz, 1 s train 45 ± 6 (12 ± 1); 20 Hz 20 s train

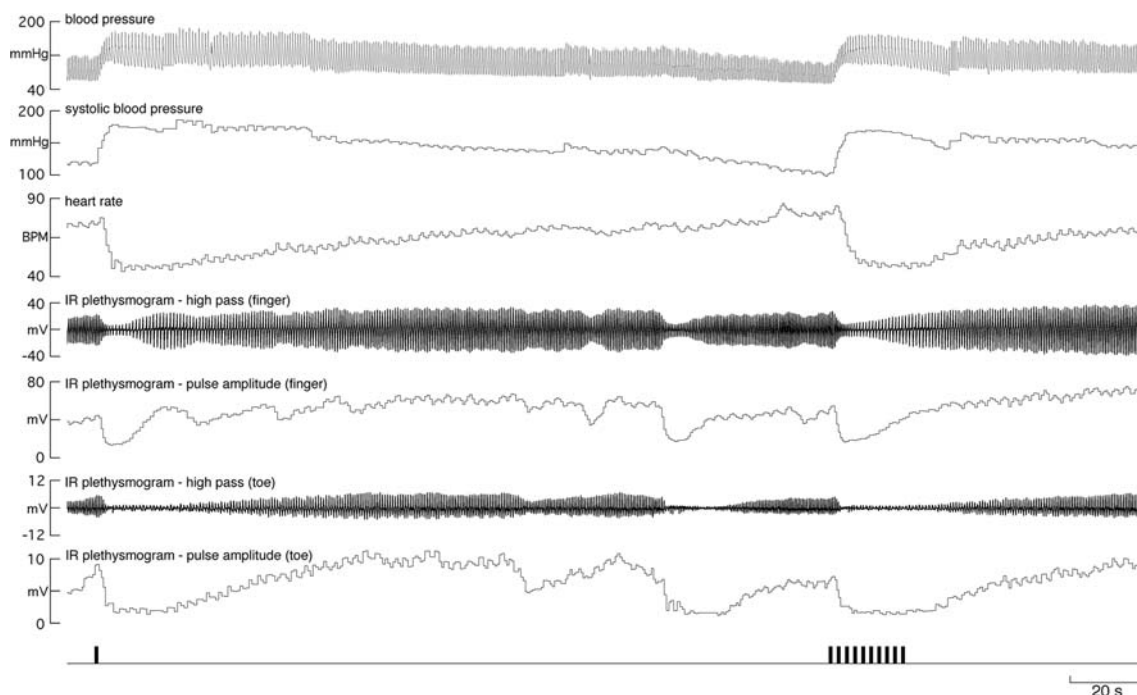


Fig. 1 Sympathetic reflex responses to electrical stimulation in a C4 ASIA A spinal subject. Blood pressure (radial artery tonometry), heart rate and cutaneous blood flow (IR plethysmography, finger and toe) responses to electrical stimulation of the abdominal wall in a C4 ASIA A spinal subject. Two different trains of electrical stimuli are

represented at the *bottom* of the figure. A single 20 Hz train of 1 s duration evoked responses that were similar in both magnitude and duration to those evoked by the brief stimulation trains of 20 Hz 1 s duration which were delivered in an on–off pattern for a total duration of 20 s

Table 1 Average changes to electrical stimulation for all stimulus parameters

Stimulation Trains	Blood flow		Blood flow		Blood pressure ↑					Heart rate ↓			
	Finger		Toe		Systolic		Mean						
	% ↓	Duration	% ↓	Duration	mmHg	%	Duration	mmHg	%	Duration	bpm	%	Duration
20 Hz, 1 s	45 ± 5	14 ± 2	52 ± 8	26 ± 7	23 ± 7	20 ± 5	58 ± 19	19 ± 5	23 ± 6	48 ± 17	21 ± 4	26 ± 6	45 ± 21
<i>n</i>	5	5	7	7	6	6	6	6	6	6	3	3	3
20 Hz, 20 s	57 ± 7	20 ± 4	54 ± 9	26 ± 4	19 ± 2	18 ± 3	44 ± 10	13 ± 2	18 ± 3	40 ± 10	15 ± 7	19 ± 4	23 ± 7
<i>n</i>	6	6	5	5	5	5	5	5	5	5	2	2	2
20 Hz, 1 s—on/off	42 ± 7	20 ± 3	45 ± 9	27 ± 7	21 ± 9	20 ± 8	26 ± 3	17 ± 7	23 ± 10	27 ± 3	26 ± 8	31 ± 12	102 ± 72
<i>n</i>	7	7	7	7	6	6	5	6	6	5	2	2	2
1 Hz, 20 s	31 ± 7	14 ± 1	48 ± 10	26 ± 6	7	7	19	7	9	8			
<i>n</i>	4	4	4	4	1	1	1	1	1	1			

Average changes in systolic and mean arterial pressures, heart rate and cutaneous blood flow in the finger and toe for all four trains of electrical stimuli. Values are presented as the mean of the peak response ±SE

n the number of subjects who responded to a given stimulus

58 ± 10 (17 ± 6); 20 Hz 1 s on–off train 36 ± 8 (20 ± 4); 1 Hz 20 s train 31 ± 7 (14 ± 1). For the paraplegics, systolic blood pressure was similar in both magnitude and duration across all stimulus trains. However, the quadriplegics showed differences in the duration of the

response among the stimulus trains, with the 20 Hz 1 s stimulus causing the longest response.

The subjects were also separated into complete and incomplete spinal cord injuries, the results of which are shown in Table 2.

Table 2 Average changes to electrical stimulation for all stimulus parameters in both complete and incomplete spinal cord injury

Stimulation Trains	Blood flow		Blood flow		Blood pressure ↑						Heart rate ↓		
	Finger		Toe		Systolic			Mean					
	% ↓	Duration	% ↓	Duration	mmHg	%	Duration	mmHg	%	Duration	bpm	%	Duration
Complete SCI													
20 Hz, 1 s	56 ± 8	19 ± 5	54 ± 14	29 ± 12	23 ± 10	19 ± 8	48 ± 27	19 ± 8	23 ± 9	43 ± 25	28	37	86
<i>n</i>	2	2	4	4	4	4	4	4	4	4	1	1	1
20 Hz, 20 s	54 ± 5	28 ± 1	42 ± 23	22 ± 4	15 ± 6	14 ± 6	21 ± 1	9 ± 2	13 ± 4	22 ± 2	8	15	16
<i>n</i>	2	2	2	2	2	2	2	2	2	2	1	1	1
20 Hz, 1 s—on/ off	51 ± 8	24 ± 3	52 ± 13	36 ± 10	28 ± 18	27 ± 17	22 ± 6	23 ± 15	32 ± 20	20 ± 5	34	43	173
<i>n</i>	4	4	4	4	3	3	3	3	3	3	1	1	1
1 Hz, 20 s	15	13	41 ± 21	27 ± 15	7	7	19	7	9	8			
<i>n</i>	1	1	2	2	1	1	1	1	1	1			
Incomplete SCI													
20 Hz, 1 s	39 ± 1	12 ± 1	49 ± 7	22 ± 6	23 ± 4	20 ± 1	77 ± 21	18 ± 3	23 ± 2	59 ± 24	18 ± 2	20 ± 1	25 ± 7
<i>n</i>	3	3	3	3	2	2	2	2	2	2	2	2	2
20 Hz, 20 s	58 ± 10	17 ± 6	63 ± 5	29 ± 7	21 ± 1	20 ± 2	60 ± 8	16 ± 3	22 ± 4	52 ± 13	22	22	30
<i>n</i>	4	4	3	3	3	3	3	3	3	3	1	1	1
20 Hz, 1 s—on/ off	30 ± 8	16 ± 5	37 ± 13	15 ± 4	14 ± 1	13 ± 1	29 ± 3	11 ± 1	14 ± 2	31 ± 11	18	19	30
<i>n</i>	3	3	3	3	3	3	3	3	3	3	1	1	1
1 Hz, 20 s	36 ± 6	14 ± 1	54 ± 7	25 ± 1									
<i>n</i>	3	3	2	2									

Average changes in cutaneous blood flow in the finger and toe, systolic and mean arterial pressures and heart rate for all four trains of electrical stimuli in both complete and incomplete spinal cord injury. Values are presented as the mean of the peak response ±SE

n the number of subjects who responded to a given stimulus

Sudomotor responses were scarce, with only three subjects displaying a response in the hand. However, these sudomotor responses were not reliable, occurring very infrequently. No sudomotor responses were seen in the foot for any stimulus train.

Ten of the thirteen subjects were assessed for completeness of the spinal cord injury in terms of autonomic function below the lesion. Above-lesion cutaneous electrical stimulation (1.8–8 mA, 1.0 ms pulses at 20 Hz, 1 s trains) was delivered via 1 cm Ag–AgCl surface electrodes applied bilaterally (4 cm apart) to the forehead. No subject displayed any cutaneous vasoconstrictor or sudomotor responses in the toes (below lesion) to the stimulation, indicating complete interruption of descending sympathetic pathways in these subjects. While this assessment was not undertaken for the other three subjects (and one of these had no vasoconstrictor responses in the toes), we do believe that our results are still valid: for the two subjects with vasoconstrictor responses there were no differences in response duration between a short stimulus train (20 Hz 1 s) and a long train (20 Hz 20 s).

Experimental records from a T2 ASIA A subject are shown in Fig. 2. It can be seen that the magnitude and duration of the responses evoked by the brief 20 Hz 1 s train (20 pulses) are similar to those evoked by both the 20 Hz stimulus that lasted for 20 s (i.e. 400 pulses) and the 20 Hz 1 s stimulus delivered in an on–off pattern for 20 s (i.e. 200 pulses). However, the magnitude of the vasoconstriction in the toe was greater (33%) for the 20 Hz 20 s stimulus than for the 20 Hz 1 s stimulus (16%) or for the 20 Hz 1 s on–off stimulus (23%), and the magnitude and duration of the systolic blood pressure increase was greater for the brief 20 Hz (1 s) stimulus. The 1 Hz 20 s duration evoked only a small increase in systolic blood pressure.

Discussion

Recording effector-organ responses to electrical stimulation of somatic nerves both above and below lesion is a useful means of indirectly assessing sympathetic function

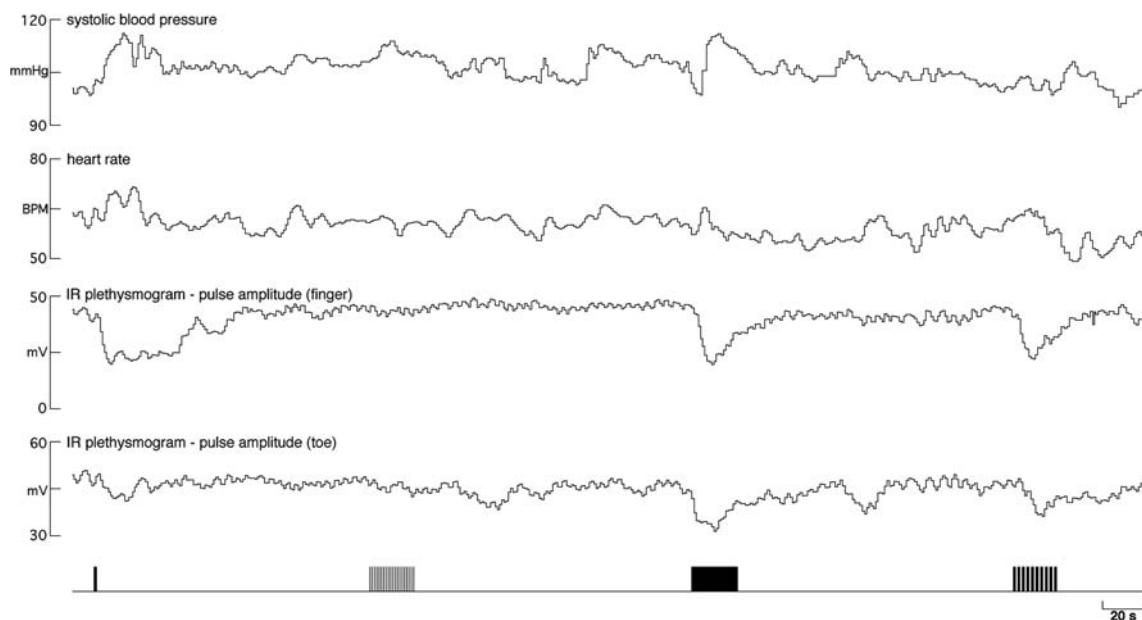


Fig. 2 Sympathetic reflex responses to electrical stimulation in a T2 ASIA A spinal subject. Systolic blood pressure, heart rate and cutaneous blood flow (finger and toe) responses to electrical stimulation of the abdominal wall in a T2 ASIA A spinal subject. All four trains of electrical stimuli are represented at the *bottom* of the figure. There was very little difference in the magnitude and duration

of the responses evoked by the brief 20 Hz 1 s stimulus, 20 Hz 20 s stimulus and the 20 Hz 1 s stimulus delivered in an on-off pattern. Note that the stimulation train of 1 Hz 20 s duration caused only a small increase in systolic blood pressure. No other responses were noted

[3, 5, 9, 20, 21]. An increase in blood pressure can infer an increase in muscle and/or splanchnic vasoconstrictor drive, while cutaneous vasoconstriction and sweat release can infer an increase in cutaneous sympathetic nerve activity. However, these previous studies had not examined the input–output relationships of somatosympathetic reflex responses, how the magnitude or duration of the effector-organ response is influenced by the magnitude or duration of the sensory input. The current study extended from our previous work, in which we used brief trains of electrical stimulation (1 s, 20 Hz) above (forehead) or below (abdominal wall) the lesion to assess the integrity of sympathetic pathways through and below a spinal lesion, as measured by changes in cutaneous blood flow, sweat release, blood pressure and heart rate [3]. By using electrical stimuli we had control over the number, frequency and duration of the stimulus pulses. We have shown that the magnitude and duration of the somatosympathetic reflex response to electrical stimulation applied below lesion is not linearly related to the duration and intensity of the sensory input; there was no difference in magnitude or duration of cutaneous vasoconstriction below the lesion for each of the stimulus parameters.

In able-bodied subjects, cutaneous electrical stimulation causes reflex cutaneous vasoconstriction [27]. However, these responses are related to the associated sensory arousal, rather than to a spinally mediated sympathetic

response. Following spinal cord injury similar reflex responses are also present below the level of injury [8], and these are due to spinal reflexes [27]. It is these spinal sympathetic reflexes, triggered below the lesion, which can cause autonomic dysreflexia; in able-bodied subjects, baroreflex-mediated withdrawal of sympathetic drive counteracts the rise in blood pressure caused by vasoconstriction [27]. Given the important role of the sympathetic nervous system in the generation of autonomic dysreflexia, it is important to know the extent of autonomic disturbance following SCI. However, changes to the sympathetic nervous system following a SCI receive less attention than changes to the somatic nervous system. The International Standard for Neurological and Functional Classification of SCI, as defined by the American Spinal Injury Association (ASIA) score, currently does not include assessment of autonomic nervous system function [19].

As noted in the introduction, noxious somatic stimuli such as those generated by pressure sores, ingrown toenails and the like, are often cited as a cause of autonomic dysreflexia. However, this dogma needs to be readdressed, as studies in both spinalised rats [16] and spinal human subjects [3, 25] have found that non-noxious stimuli can also trigger episodes of autonomic dysreflexia. Furthermore, our recent study found that specific activation of pain receptors in muscle and skin below the lesion in spinal cord injured

subjects failed to trigger autonomic dysreflexia [4]. The present study, like our earlier study [3], used electrical stimulus intensities that were considered to be non-noxious in able-bodied subjects. Despite this, we observed increases in blood pressure (and compensatory bradycardia) that were comparable to those encountered during recordings of autonomic dysreflexia. Accordingly, these results, and the results of the studies mentioned above, argue strongly against *noxious* somatic stimuli being essential for evoking autonomic dysreflexia. It must be noted, however, that noxious cutaneous stimuli can and *does* trigger episodes of autonomic dysreflexia. In the spinal cat, Jänig et al. [11, 12] have demonstrated that noxious electrical, thermal and mechanical stimulation of skin can elicit pronounced and prolonged reflex activity in cutaneous postganglionic neurones [11, 12]. Why there is such a difference in somato-sympathetic reflexes to noxious stimulation in the spinal cat and human is not known, though it does need to be recognised that evolution has seen an increase in encephalisation and a decrease in the role of spinal circuitry, at least with respect to sensorimotor control.

Limitations

Given the difficulty in performing peroneal-nerve micro-neurography following spinal cord injury [15], we did not attempt to record muscle or cutaneous sympathetic nerve activity directly. Rather, we recorded the responses of the effector organs: changes in blood pressure brought about by increases in sympathetically mediated vasoconstriction in the muscle and splanchnic vascular beds, vagally mediated falls in heart rate brought about by the baroreflex, decreases in skin blood flow brought about by sympathetically mediated vasoconstriction in cutaneous vessels and increases in sweat release brought about by increases in sudomotor drive. While sudomotor responses to somatic stimuli below lesion were rare, as observed previously [3], cutaneous vasoconstriction did prove to be a more robust measure of the state of sympathetic activation below lesion. Given that muscle and cutaneous vasoconstrictor neurones are co-activated by stimuli below lesion [27], it is reasonable to infer an increase in muscle vasoconstrictor (as well as splanchnic) sympathetic neural outflow whenever we see evidence of cutaneous vasoconstriction, particularly when we see an increase in blood pressure. What we do not know, however, is whether the duration of the vasoconstriction is a true reflection of the sympathetic neural outflow per se, or of the reactivity of the vascular smooth muscle. However, it is known that the vascular responsiveness of the rat tail artery (which supplies the skin of the tail) is augmented by spinal cord injury, and that the duration of the vasoconstriction outlasts the duration of the stimulation [28].

Conclusions

Our observations suggest that the sympathetically mediated vasoconstrictor responses to somatic afferent stimulation may not be directly related to the degree of sensory input (i.e. duration, number of pulses or stimulation frequency). If there was a direct relationship between the responses and the degree of input, we would have expected the duration of the vasoconstriction to be twenty times as long for a 20 Hz 20 s stimulus than a 20 Hz 1 s stimulus. As the reflex response was not directly dependent on the input intensity, the recruitment of cutaneous (and muscle and splanchnic) vasoconstrictor neurones may be limited to the initial afferent barrage evoked by a stimulus train. Whether this is due to habituation of the spinal somatosympathetic reflex or neuromuscular fatigue of the blood vessels remains to be determined.

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