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# Thermal sweating following spinal cord injury

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# Thermal sweating following spinal cord injury

## **Abstract**

A complete spinal cord injury prevents neural connections between distal sites and higher neural structures. While it has previously been demonstrated that an isolated spinal cord can elicit non-thermal sweating independently of the hypothalamus [1-3], the ability of the spinal cord to control sweating in response to thermal stimuli, without hypothalamic influence, is less clear. The majority of early literature indicates that thermal sweating is absent below a complete spinal cord injury (SCI) [4-7], yet several studies suggest otherwise [8-11]. However, invasive measures have failed to observe altered sympathetic activity when thermally stimulating insensate regions [12], which is inconsistent with the observations of sweating below a SCI.

## **Keywords**

spinal, injury, following, thermal, sweating, cord

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## **Authors**

Bradley Wilsmore, J D. Cotter, Andrea MacDonald, A. Zeyl, Guy M. Bashford, and Nigel Taylor

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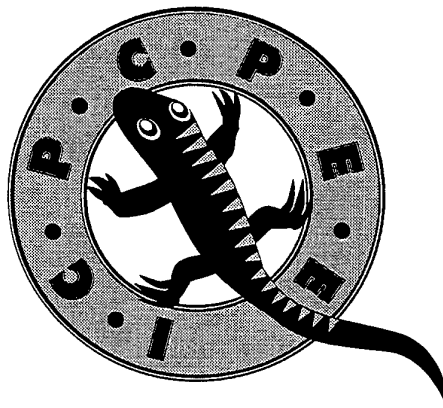
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## PAPER 7: THERMAL SWEATING FOLLOWING SPINAL CORD INJURY.

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### INTRODUCTION

A complete spinal cord injury prevents neural connections between distal sites and higher neural structures. While it has previously been demonstrated that an isolated spinal cord can elicit non-thermal sweating independently of the hypothalamus [1-3], the ability of the spinal cord to control sweating in response to thermal stimuli, without hypothalamic influence, is less clear. The majority of early literature indicates that thermal sweating is absent below a complete spinal cord injury (SCI) [4-7], yet several studies suggest otherwise [8-11]. However, invasive measures have failed to observe altered sympathetic activity when thermally stimulating insensate regions [12], which is inconsistent with the observations of sweating below a SCI.

There are two main limitations within many of the above studies. Firstly, for the spinal cord to be deemed to have initiated sweating independently of the hypothalamus, it must be confirmed that neural connections are absent. Since most clinical verifications of SCI completeness do not evaluate autonomic completeness, and since sweat glands are sympathetically innervated, it is imperative to evaluate the integrity of autonomic function. In several cases, where sweating was observed below a SCI [9-11], autonomic completeness was not reported, and presumably not tested. Indeed, it has been demonstrated that complete somatosensory separation can exist while residual autonomic function remains [1, 13]. Hence, we are unable to determine whether sweating below a SCI is the result of surviving or regenerated neural connections, or whether it is spinally mediated. In those studies reporting sweating below a SCI, sweat rate is substantially reduced [8-10]. Such a pattern may occur in the presence of a massive, but incomplete disruption of the neural pathways. Thus, such sweating may have been initiated by the hypothalamus.

Secondly, if sweating is present below a confirmed complete SCI, it must be confirmed whether or not its origin is thermal, or resulting from other afferent feedback (*e.g.* pain or localised pressure). Autonomic dysreflexia and muscle spasms are the major cause of non-thermal sweating in spinal patients. Such sweating, both above and below the SCI, is a common clinical observation [1-3]. However, since sweat rate is directly affected by local temperature [14], non-thermal sweating may be similarly altered, despite being initiated by non-thermal factors. Therefore, the aim of the current project was to investigate the possible existence of thermally-induced sudomotor control in subjects with a complete SCI, using measures of sweat rate ( $\dot{m}_{sw}$ ), and sweat expulsion frequency ( $f_{sw}$ ).

### MATERIALS AND METHODS

Eight subjects with clinically complete SCI (C5-L1) and 10 non-injured controls were studied. Clinical examination, performed by an experienced medical practitioner, verified somatosensory completeness. Since several studies have shown residual sensory and motor innervation despite clinically-verified complete SCI [13, 15-17], additional verification was achieved by clamping mean body temperature above the sweat threshold, while the leg blood flow was occluded and the leg was cooled with 7.9°C water. A concomitant decrease in forehead  $\dot{m}_{sw}$  in SCI subjects was interpreted to have resulted from intact sensory

connections. Such subjects were classified as physiologically incomplete SCI, and are not reported herein.

Subjects rested supine in a climate-controlled chamber at 38.5°C (38.5% r.h.). Mean body temperature was clamped using a water-perfusion suit ( $T_w$  38.8°C, SD 0.5). Core temperature was measured from the oesophagus, rectum and auditory canal, and skin temperatures from 14 sites. The  $m_{sw}$  was measured at six sites simultaneously, using ventilated sweat capsules ( $3.16 \pm 0.05 \text{ cm}^2$ ; Multi-Site Sweat Monitor, Clinical Engineering Solutions, Australia). Two capsules, attached to the forehead and foot, were modified to yield  $f_{sw}$  data. These data were used to determine whether sweating at the forehead was of the same rhythm as that below the SCI (if present). This was essential for determining whether or not sweat below the SCI was under hypothalamic control, and thereby having a synchronous  $f_{sw}$  pattern.

Since major causes of non-thermal sweating below a SCI are bladder and bowel distension, and local tissue ischaemia, subjects emptied their bladder and bowel prior to a trial. To limit pressure-induced ischaemia, all subjects rested on a dry floatation cushion (ROHO<sup>®</sup> Inc, Belleville, IL., U.S.A.). This is a specialised mattress commonly used with spinal patients to minimise local pressure. Great care was also taken to avoid any tubing or thermistor cables lying between the subject and the mattress, and subjects were moved regularly to reduce localised pressure. These precautions minimised both the incidence and severity of spastic responses in the SCI subjects.

## RESULTS

All controls displayed sweat suppression with leg cooling. However, one SCI subject similarly experienced reduced forehead sweating, and was classified as an incomplete SCI. Seven clinically, and physiologically, complete SCI subjects remained. Sweating did not occur at the forehead in any quadriplegic subject ( $n=3$ ). Since preganglionic sympathetic fibres originate from neurons in the thoracic and lumbar segments only, a complete SCI above the thoracic segments results in the loss of all sympathetic and, subsequently, sudomotor control. Therefore, the ability to verify SCI completeness using limb cooling was not applicable within these subjects.

Whole body  $m_{sw}$  (averaged across six sweat capsules) for the control group was greater than observed in the SCI subjects ( $1.03$  and  $0.3 \text{ mg}\cdot\text{cm}^{-2}\cdot\text{min}^{-1}$  respectively;  $P<0.05$ ). This was primarily due to the absence of sweating at several sites in the majority of SCI subjects, regardless of large increases in mean body temperature ( $1.9^\circ\text{C}$  SD  $0.4$ ). However, of the seven complete SCI subjects, two had sweating below the SCI, *albeit* of a substantially reduced magnitude:  $< 0.1 \text{ mg}\cdot\text{cm}^{-2}\cdot\text{min}^{-1}$ .

In the control subjects, a delay of about 2 s occurred between a sweat expulsion at the forehead, and a corresponding expulsion at the foot. In the two SCI subjects with sweating below the SCI, the foot  $f_{sw}$  was only 53% of the forehead  $f_{sw}$ . However, when averaged over 50 min and across both subjects, 94% of the expulsions at the foot coincided with forehead sweat expulsions.

## DISCUSSION AND CONCLUSION

The major finding of the present study was that the spinal cord, when isolated from the hypothalamus, could not independently induce sweating in response to thermal stimuli. These results also demonstrate that, despite the complete absence of spinal cord function below the site of injury, the autonomic nervous system can still be partially intact, and responsive to hypothalamic signals.

Consistently sequential sweat expulsions from both the sensate and insensate skin of two subjects with clinically-verified complete SCI, are consistent with hypothalamic control of thermally-induced sweating, and cannot be deemed to be a spinally-mediated sudomotor

response. Such control may, however, be due to the survival, or regeneration, of sympathetic ganglia, independently of a complete SCI. This finding may help explain why several previous studies have found sweating below a clinically, and sometimes anatomically, complete SCI.

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*All experiments were approved by the University's Human Research Ethics Committee.*