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LETTER TO THE EDITOR

Hemodynamic responses to the cold pressor test in spinal cord-injured individuals; control of the splanchnic vascular bed is the key factor

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Recently, Catz *et al.*[1] reported on the hemodynamic effects of a cold pressor test (CPT) of the hand in spinal cord-injured (SCI) individuals with T4-6 paraplegia, C4-7 tetraplegia and control individuals. They demonstrated an increase in heart rate in all three groups. Blood pressure increased in controls and tetraplegia but not in paraplegia. The authors suggest an independent thoracic spinal mechanism to be responsible for the hemodynamic response to a CPT of the hand in SCI individuals. We have read this article with great interest, because hemodynamic responses in SCI individuals are still not fully understood. However, we would like to challenge the authors and readers to view the results and conclusions from a different angle.

A CPT will elevate sympathetic activity, which will increase heart rate, cardiac contractility and peripheral resistance.[2] Consequently, blood pressure will increase, as it is the result of cardiac output (heart rate and cardiac contractility) and total peripheral resistance.[2] Two major vascular beds are, principally, responsible for the increase in peripheral resistance: the splanchnic and lower limb vascular beds, both innervated below T6.

A CPT of the hand in T4-6 paraplegia will elevate sympathetic activity above lesion level and, therefore, will increase heart rate and cardiac contractility. However, the splanchnic and lower limb vascular beds are innervated below the spinal cord lesion and do not contribute to the hemodynamic response to a CPT of the hand. Consequently, blood pressure can only marginally increase in T4-6 paraplegia in response to a CPT of the hand, as was shown by Catz *et al.*[1]

In C4-7 tetraplegia a CPT of the hand, a nociceptive stimulus below the spinal cord lesion, will elevate sympathetic activity below lesion level, better known as autonomic dysreflexia (AD).[3] This exaggerated overall sympathetic activity, without supraspinal control, will increase peripheral resistance, heart rate and cardiac contractility, because the heart is innervated from T1-4. Vasoconstriction of the splanchnic and lower limb vascular beds combined with an increase in cardiac output will increase blood pressure in C4-7 tetraplegia, as presented in the study by Catz *et al.*[1] In C4-7 tetraplegia, some individuals have a spinal cord lesion that can interfere with afferent fibers involved in the CPT of the hand (C6-T1), therefore, blood pressure did not reach values usually seen during AD.[1,3]

We believe, that neural control of the splanchnic and lower limb vascular beds is the key factor in hemodynamic responses to a CPT, as well as the location of the CPT (that is above or below the spinal cord lesion). The splanchnic vascular bed is of great interest, because AD only occurs in SCI individuals with a spinal cord lesion above T6.[3] It seems that supraspinal control over the splanchnic vascular bed is essential in the hemodynamic response upon a sympathetic stimulus.

It would be of interest to study the hemodynamic response of the splanchnic and lower limb vascular beds to a sympathetic stimulus in SCI individuals, and maybe use a cold application to the forehead in tetraplegia (above lesion level), to avoid AD.

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References

1 Catz A, Bluvshtein V, Pinhas I, Akselrod S, Gelernter I, Nissel T *et al.* Cold pressor test in tetraplegia and paraplegia suggests an independent role of the thoracic spinal cord in the hemodynamic responses to cold. *Spinal Cord* 2008; **46**: 33–38.
2 Freeman R. Assessment of cardiovascular autonomic function. *Clin Neurophysiol* 2006; **117**: 716–730.
3 Krassioukov A, Claydon VE. The clinical problems in cardiovascular control following spinal cord injury: an overview. *Progr Brain Res* 2005; **152**: 223–229.