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LETTER TO THE EDITOR

Reply to JT Groothuis and MTE Hopman's letter

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The letter 'Hemodynamic responses to the cold pressor test in spinal cord-injured individuals; control of the splanchnic vascular bed is the key factor' by Jan T Groothuis and Maria TE Hopman[1] makes some important points. They wish 'to challenge the authors and readers' of the paper published by Catz *et al*. in 2007 on cold pressor test (CPT) in *Spinal Cord*,[2] 'to view the results and conclusions of that paper from a different angle.' In fact, they do suggest an important addition to the discussion in our paper. The fact that control of the splanchnic vascular bed is a key factor in the CPT mechanism is compatible with the findings of the paper and does not contradict its conclusions. The paper does not devalue the role of the splanchnic and lower limb vascular beds in the CPT mechanism. We noted the role of the splanchnic bed in autonomic cardiovascular control in another article,[3] and the paper suggests an explanation for the reduced response of the vascular bed in paraplegia, which involves a theoretical neural mechanism.

The suggestion of the authors in the letter that, in $C_{4\text{-}7}$ tetraplegia, CPT of the hand can produce a nociceptive stimulus below the spinal cord lesion that elevates sympathetic activity below level of the lesion is also compatible with the findings and conclusions of the paper. We are not aware of known pathways that convey sensory stimuli from cervical spinal cord segments down to the sympathetic columns in the thoracic spinal cord. Nevertheless, the paper also suggested a hypothetical sympathetic activation by CPT stimuli below the spinal cord lesion (SCL) level in tetraplegia, implying that the CPT reflex arch may not follow the customary CPT model, bypass the brainstem vasomotor center and include an alternative neural center located below cervical SCL in the thoracic spinal cord.

The fact that a below cervical SCL stimulus may be affecting the heart but not the vascular bed, however, is not sufficient to explain the significant blood pressure increase in tetraplegia but not in paraplegia in response to CPT. This is because cold application to the foot, which can affect the vascular bed and the heart in mid-thoracic paraplegia, also increases blood pressure in tetraplegia but not in paraplegia.[4] Although in $T_{4\text{-}6}$ paraplegia stimuli from the foot may not reach the heart through upper thoracic spinal cord segments to increase heart contractility and rate, they do reach the heart by an unknown route, affecting its rate. The effect of these stimuli is to reduce heart rate and not to increase it, a response not mediated through the baroreceptor mechanism because blood pressure is not significantly elevated, but possibly through the sympathetic chain.[4] The difference in the ability to affect both the heart and the blood vessels itself does not account for the difference in CPT response between paraplegia and tetraplegia. Rather, the difference is the result of the difference in the CPT effect on each of them, which probably depends on the circuits of a neural mechanism at the thoracic level. This implies that not only supraspinal but also spinal thoracic control over the splanchnic vascular bed is essential for hemodynamic responses to sympathetic stimuli. We think that, attributing the response to below cervical SCL hand CPT stimulus to autonomic dysreflexia is inappropriate, because the autonomic dysreflexia syndrome includes bradycardia and not tachycardia, and the autonomic dysreflexia trigger is usually in the lower part of the body.

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