Effects of spinal cord injury on neurovascular function

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The consequences of spinal cord injury (SCI) for the function of sympathetic pathways in the periphery have generally been ignored. We have investigated the plasticity at the sympathetic neurovascular junction that follows disruption of sympathetic pathways in rats. These studies have demonstrated a marked enhancement in neurovascular transmission in arteries and veins to which ongoing sympathetic activity has been reduced following SCI (Yeoh, McLachlan & Brock, 2004; Brock, Yeoh & McLachlan, 2006). Data from humans with SCI indicates that this change is also present and that it contributes to generating episodes of high blood pressure (autonomic dysreflexia) triggered by bladder distension and other sensory stimuli (Teasell *et al.*, 2000). In arteries supplying skin (rat tail and saphenous arteries), the mechanisms underlying this arterial hyperreactivity differ from those in rat mesenteric arteries. In the former, the enhancement is primarily due to an increased reactivity of the vascular muscle to nerve-released noradrenaline, whereas the increased responsiveness in the latter vessels can be attributed to decreased removal of released noradrenaline by the neuronal noradrenaline transporter. In addition to these effects on neural activation of blood vessels, in both humans and animals SCI causes remodelling of arteries and veins, with marked reductions in their lumen diameter (*e.g.* de Groot, Bleeker & Hopman, 2005). This change is suggested to be an adaptation to reduced blood flow following SCI. Data from humans suggest that these changes in blood vessel function and structure following SCI are likely to contribute to autonomic dysreflexia.

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