## Plasticity of neurovascular transmission following spinal cord injury

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Bladder distension or minor injuries below the lesion in spinally injured people often lead to episodes of high blood pressure. This condition, termed autonomic hyperreflexia, is generally thought to result from the loss of descending inhibitory pathways and subsequent changes within the spinal cord. However, measurements of sympathetic nerve activity in spinal patients indicate that basal sympathetic nerve traffic is greatly depressed and that evoked reflex discharges are weak but are associated with long lasting vasoconstriction (Stjernberg *et al.*, 1986). These findings are hard to reconcile with the hyperreflexic syndrome unless the vascular response to sympathetic nerve activity is increased. Therefore we investigated whether spinal cord injury produces an enhancement of sympathetic neurovascular transmission. The studies were performed on tail and mesenteric arteries isolated from rats with spinal cord transections. This lesion was performed at T8 (tail artery) or T4 (mesenteric artery) under anaesthesia with ketamine (60 mg/kg) and xylazine (10 mg/kg) administered i.p. Seven weeks post lesion, neurally evoked contractions of both arteries were markedly increased in the amplitude. In both arteries neurally evoked contractions were markedly increased in amplitude. In tail artery this change can, in part, be explained by a generalized increase in the sensitivity of the vascular smooth muscle to vasoconstrictor agents. In accord with this finding, it has been reported that the vascular sensitivity to infused noradrenaline is increased in tetraplegics (Arnold *et al.*, 1995). However, in mesenteric arteries there was no change in vascular reactivity and a decreased clearance of noradrenaline by neuronal uptake contributes to the augmented contractions. The findings indicate that an augmentation of neurovascular transmission contributes to autonomic hyperreflexia and that the mechanisms underlying this change differ between tail and mesenteric arteries.

Arnold JM, Feng QP, Delaney GA & Teasell RW (1995) *Clinical Autonomic Research*, **5**: 267-270.
Stjernberg L, Blumberg H & Wallin BG (1986) *Brain*, **109**: 695-715.