Adaptations following partial denervation of the rat tail artery maintain normal neurovascular control

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Nerve injuries that cause loss of vascular innervation produce disordered regulation of skin blood flow and altered skin function. This study investigated the effects of partially denervating the rat tail artery. Sympathetic axons travel to this artery via two large ventral collector nerves (VCN) and two smaller dorsal collector nerves. One or both VCN were transected ~1 cm proximal to the base of the tail under anaesthesia with ketamine/xylazine (60 and 10 mg/kg i.p.). Pieces of artery from 5-7 cm along the tail were removed from 1 or 7 week lesioned and unoperated control rats anaesthetized with pentobarbitone (100 mg/kg i.p.) and killed by exsanguination. Artery segments were mounted in wire myographs. Adjacent pieces of artery were prepared for immunohistochemistry using antibodies to tyrosine hydroxylase (TH). At one week, arteries from rats with both VCN cut were almost completely denervated and had much smaller neurally evoked contractions than did control arteries. In arteries from rats with one VCN cut, neurally evoked contractions to 100 pulses at 1 Hz were not changed in amplitude after 1 or 7 weeks; neither was the blockade produced by the α-adrenoceptor antagonists prazosin (10 nM) or idazoxan (0.1 μM). However, the facilitatory effect of idazoxan on contractions to 10 pulses at 10 Hz was reduced for 1-week lesioned arteries, suggesting that α_2-adrenoceptor-mediated autoinhibition of noradrenaline release was diminished in parallel with the reduction in the perivascular plexus of varicose TH+ nerve terminals. Thus transmission from the residual axons is rapidly potentiated and neurovascular control is maintained despite the reduction in number of innervating axons.