



Autonomic Nervous System: Chapter 24. Sensory-autonomic interactions in health and disease (Handbook of Clinical Neurology)

Peter D. Drummond

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Although sensory and autonomic nerve fibres generally do not interact directly, both may exert influences on blood flow during inflammation. For example, the sympathetic neurotransmitter noradrenaline/norepinephrine evokes axon reflexes, a response that involves release of vasoactive neuropeptides from the peripheral terminals of primary nociceptive afferent fibres. As well as boosting inflammation, this mechanism could play a role in normal renal function and heat dispersal from the skin. In certain disease states, aberrant communication between sensory and autonomic nerves might not only aggravate symptoms but also contribute to clinical deterioration by altering local circulatory dynamics. For example, in certain forms of neuropathic pain, an aberrant expression of α 1-adrenoceptors on primary nociceptive afferents may provide a framework for cross-talk between sensory and autonomic nerve fibres. In addition to evoking pain and other unpleasant symptoms, this cross-talk could aggravate inflammation and disrupt nutritive perfusion of affected tissues. Finally, in disorders such as cluster headache, intense bursts of trigeminal nociceptive activity may trigger trigeminal-parasympathetic vasodilator reflexes which, in turn, provoke secondary vascular disturbances that amplify pain. A clearer understanding of sensory-autonomic interactions both in health and disease may provide a basis for new treatment approaches for conditions that respond poorly to conventional treatments.

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