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Topical application of clonidine relieves hyperalgesia in patients with sympathetically maintained pain.

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Author information

Abstract

Patients with reflex sympathetic dystrophy or causalgia characteristically have ongoing pain and pain to light touch (hyperalgesia). Some of these patients obtain relief of their pain following interruption of sympathetic function to the affected area and, therefore, have sympathetically maintained pain (SMP). Evidence suggests that the pain and hyperalgesia in SMP are related to activation of peripheral adrenergic receptors. We wished to determine the contribution of alpha 1- and alpha 2-adrenergic receptors in SMP and thus examined the effects of local application of adrenergic agents in patients with SMP. The alpha 2-adrenergic agonist clonidine, available as a transdermal patch, was delivered topically to the patients' hyperalgesic skin. In four patients with SMP, clonidine eliminated or substantially reduced hyperalgesia to mechanical and cold stimuli. In three of these patients the effects were confined to the skin region beneath the patch, suggesting a peripheral and not central effect. The relief of hyperalgesia was not due to a local anesthetic effect since touch thresholds were unaffected. Topical clonidine did not relieve hyperalgesia of similar severity for two other patients whose hyperalgesia and pain were unaffected by sympathetic ganglion blocks (i.e., diagnosed as having sympathetically independent pain). In two SMP patients, intradermal injection of norepinephrine or phenylephrine (a specific alpha 1-adrenergic agonist) at a site treated with clonidine evoked intense pain and rekindled the pre-clonidine hyperalgesia at that site. It is likely that clonidine locally blocks the release of norepinephrine via activation of alpha 2 receptors on the sympathetic terminals. This study suggests, therefore, that SMP is mediated via alpha 1-adrenergic receptors located in the affected tissue.

Comment in

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