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Resistance blood vessels are innervated by sympathetic and parasympathetic nerves which modulate the vascular tone. Other non-sympathetic sensory nerves originating from the spinal cord (lumbar and sacral) and, upon stimulation, cause a non-adrenergic and non-cholinergic (NANC) vasodilatation via the release of vasoactive neuropeptides, particularly calcitonin gene-related peptide (CGRP) (Kawasaki et al., 1988). CGRP is a polypeptide of 37 amino acids predominantly located in sensory neurons and peripheral nerve terminals of blood vessels (Suzuki et al., 1989; Gotoh et al., 1990). These neuropeptides, such as substance P, CGRP, and calcitonin gene-related peptide (CGRP), have been shown to induce vasodilation in the systemic circulation (Huang et al., 1996). On the other hand, several lines of evidence suggest that CGRP may also induce vasoconstriction (Aune et al., 1997; Jannmose et al., 1997; Korslund et al., 1997) and dihydroergotamine (Fritz et al., 1991). In the present study, the norepinephrine-induced vasoconstriction was unaffected after atropine, propranolol or pyridium-β-blocker, and it was antagonized by CGRP, but not by CGRP receptor antagonist.

In this study, several lines of evidence suggest that antimuscarinic drugs (atropine, scopolamine, and propantheline bromide), α-adrenoceptor antagonists (prazosin, phentolamine, and propranolol), and dihydroergotamine (Fritz et al., 1991) did not affect the norepinephrine-induced vasoconstriction. To the best of our knowledge, no study has been performed to evaluate the effect of CGRP on the vasoconstrictive action of norepinephrine in the systemic circulation. It is interesting to note that the CGRP receptor antagonist, CGRP8–37, did not affect the vasoconstrictive action of norepinephrine in the systemic circulation. This suggests that the CGRP receptor antagonist does not have a significant effect on the vasoconstrictive action of norepinephrine in the systemic circulation.

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