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Vasopressor and heart rate responses to systemic administration of bombesin in anesthetized rats

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Abstract:
The aim of the present study was to investigate the effects of aortic depressor nerve (ADN) transection, supranodosal vagi denervation (NG vagi cut) and adrenergic receptor blocker treatment on the cardiovascular responses evoked by systemic injection of bombesin.
The cardiovascular effects were studied in spontaneously breathing rats that were (i) bilaterally, midcervically vagotomized (MC vagi cut) and subjected to section of the aortic depressor nerves, (ii) midcervically vagotomized and subsequently vagotomized at the supranodosal level or (iii) midcervically vagotomized before and after pharmacological blockade of α- or β-adrenergic receptors with phentolamine and propranolol, respectively.
An intravenous bolus of bombesin (10 μg/kg) in midcervically vagotomized and ADN denervated animals increased mean arterial blood pressure (MAP) and heart rate (HR). An approximate 20% increase in blood pressure occurred immediately following bombesin injection and lasted for 2–3 min. Augmentation of the heart rate occurred 30–60 s after the bombesin challenge and persisted for more than 10 min. After section of the supranodosal vagi, bombesin failed to induce an increase in heart rate. Blockade of α-adrenergic receptors with an intravenous dose of phentolamine significantly reduced post-bombesin hypertension.
These results indicate that bombesin-evoked increases in blood pressure do not require aortic depressor nerves and supranodosal vagi and are presumably mediated by the activation of peripheral α-adrenergic receptors. Bombesin-induced tachycardia was dependent on an intact supranodose pathway and was amplified by activation of β-adrenoceptors.

Key words:
bombesin, α- or β-adrenergic receptor, aortic depressor nerve, nodosal vagal afferents