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in a subgroup of experiments, we found that low concentrations of the noradrenergic agent isoproterenol (0.1 $\mu\text{mol/l}$) directly applied to the brainstem preparation could activate the locomotor cpg and drive rhythmic activity in the spinal ventral roots. however, the increase in the respiratory frequency induced by this treatment was partially attenuated by the combined exposure to atropine (1 $\mu\text{mol/l}$) and guanethidine (10 $\mu\text{mol/l}$) that eliminated neural and muscular sympathetic cholinergic tone, respectively. the effects of the noradrenergic agonist were also abolished by the combined exposure to tetrodotoxin (5 $\mu\text{mol/l}$) and d-tubocurarine (1 $\mu\text{mol/l}$). these findings suggest that the effects of isoproterenol on spinal rhythms involve an increase in the cholinergic drive to the motoneurons and probably involve the activation of muscarinic receptors and the inhibition of nicotinic receptors in spinal cord. the contribution of the cholinergic system to the modulation of respiratory frequency after activation of the noradrenergic system was confirmed in another set of experiments where the ventral roots were recorded after systemic treatment with the cholinergic agonist carbachol (1 $\mu\text{mol/l}$) alone or in combination with tetrodotoxin (5 $\mu\text{mol/l}$). in both conditions, an increase in the respiratory burst frequency, similar to that elicited by isoproterenol, was observed. these results suggest that noradrenergic activation of motoneurons, particularly those that belong to the vmrf, can modulate the activity of spinal respiratory rhythm generators via an increase in the cholinergic drive. 5ec8ef588b

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