Abstract

Electrical stimulation of the hypothalamus produces cardiovascular adjustments consisting of hypertension, tachycardia, visceral vasoconstriction and hindlimb vasodilation. Previous studies have demonstrated that hindlimb vasodilation is due to a reduction of sympathetic vasoconstriction and hindlimb vasodilation. However, the existence of a yet unidentified vasodilator mechanism has also been proposed. Recent studies have suggested that nitric oxide (NO) may be involved. The aim of the present study was to investigate the role of NO in hindquarter vasodilation in response to hypothalamic stimulation. In pentobarbital-anesthetized rats, hypothalamic stimulation (100 Hz, 150 µA, 6 s) produced hypertension, tachycardia, hindquarter vasodilation and mesenteric vasoconstriction. Alpha-adrenoceptor blockade with phentolamine (1.5 mg/kg, iv) plus bilateral adrenalectomy did not modify hypertension, tachycardia, or mesenteric vasoconstriction. Hindquarter vasodilation was strongly reduced but not abolished. The remaining vasodilation was completely abolished after injection of the NOS inhibitor L-NAME (20 mg/kg, iv). To properly evaluate the role of the mechanism of NO in hindquarter vasodilation, in a second group of animals L-NAME was administered before and after α-adrenoceptor blockade plus adrenalectomy. L-NAME treatment strongly reduced hindquarter vasodilation in magnitude and duration. These results suggest that NO is involved in the hindquarter vasodilation produced by hypothalamic stimulation.

Keywords
hindlimbblood&64258;ow, hypothalamus, electrical stimulation, defense reaction.