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In normal male volunteers, intravenous infusions of the alpha 1-adrenergic agonist methoxamine stimulated the secretion of prolactin, thyroid-stimulating hormone (TSH), and adrenocorticotrophic hormone (ACTH), and the effects were abolished by pretreatment with the alpha 1-antagonist prazosin. To investigate the site of action of methoxamine, its effects were compared with those of equipotent doses of **norepinephrine, an alpha 1-agonist that reaches the pituitary gland and the median eminence after an intravenous infusion but, unlike methoxamine, does not cross the blood-brain barrier.**

Norepinephrine did not stimulate secretion of prolactin, TSH, or ACTH, suggesting that the stimulant alpha 1-adrenoceptors are located in the central nervous system and not directly on the pituitary gland or in the periphery. The alpha 2- and betaadrenoceptor agonist properties of norepinephrine could not account for the differences from methoxamine, as pretreatment with prazosin did not modify hormone concentrations after norepinephrine. Methoxamine had no behavioral stimulant effects, as judged by visual analog scales that were sensitive to physiological changes in behavioral arousal. In four patients with hypothalamic dysfunction but responsive pituitary corticotrophs, methoxamine had no stimulant effect on the secretion of ACTH, confirming that the alpha 1-adrenoceptors that stimulate ACTH secretion are not located directly on the pituitary. None of the drugs had an effect on the secretion of growth hormone or the gonadotrophins.