HYPOTHALAMIC MODULATION OF BARORECEPTOR-INDUCED INHIBITION OF ELECTRICAL ACTIVITY IN THE RENAL NERVE

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UDC 612.819.916:612.187.35]-06:612.826.4

KEY WORDS: blood pressure, baroreceptor reflexes, sympathetic activity, stimulation of the hypothalamus.

Emotional stress or electrical stimulation of the "emotiogenic zones" of the hypothalamus leads to inhibition of the chronotropic component of the baroreceptor reflex (BR) [5, 7]. It is considered [6,9,11] that this mechanism depresses vagal tone and prevents the onset of tachycardia in emotional reactions. Data on the possibility of hypothalamic modulation of the vasomotor component of BR are more contradictory, and opinions vary between accepting [1, 3] and totally rejecting [2, 10] such modulation.

Since general anesthetics used in the above-mentioned experiments can substantially modify the intracentral organization of vasomotor control [4, 8], in the investigation described above the possibility of hypothalamic modulation of baroreceptor-induced inhibition of electrical activity was studied in a sympathetic nerve and under different conditions of anesthesia.

EXPERIMENTAL METHOD

Acute experiments were performed on 25 cats weighing 2.5-3.5 kg. The preparatory operations were carried out under the influence of sodium hydroxybutyrate (1 g/kg intravenously, after preliminary induction of anesthesia with ether) or of ketamine (30 mg/kg, intramuscularly). After the preparatory operation the animals were immobilized with suxamethonium and connected to an artificial respiration apparatus. The systemic arterial pressure (BP) was measured in the femoral artery. Sympathetic vasomotor activity was recorded in the left renal nerve in the usual way. Electrical activity was integrated with a time constant of 0.15 sec. Evoked responses were averaged on the F36 synchronized digital store. Baroreceptor afferents were activated by stimulation of the central end of the vago-aortic nerve (frequency 125 pulses/sec, pulse duration 0.5 msec, amptitude 0.5-15 V). The ipsilateral hypothalamus (ventromedial hypothalamic nucleus) was stimulated by a coxial electrode, inserted into the brain from the dorsal aspect (frequency 100 pulses/sec, pulse duration 1 msec, amplitude 0.5-7.5 V).

EXPERIMENTAL RESULTS

During scanning stimulation of the hypothalamus (duration of each stimulation 20 sec) tachycardia, a pressor reaction, and strengthening of activity in the renal nerve were always observed. During simultaneous stimulation of the hypothalamus and the vago-aortic nerve responses were obtained on the basis of which the points of hypothalamic stimulation could be divided into two zones. During stimulation of zone I, activation of vago-aortic afferents, which itself always evoked bradycardia, a depressor response, and inhibition of vasomotor activity, did not change responses to hypothalamic stimulation. During combined stimulation of zone II and vago-aortic afferent changes in heart rate, BP, and vasomotor activity which took place were the sum of the responses to their separate stimulation (Fig. 1).

Injection of chloralose (50 mg/kg, intravenously) did not change responses to stimulation of zone II of the hypothalamus and of the vago-aortic nerve but significantly modified hypothalamic modulation of BR to stmulation of hypothalamic zone I (Fig. 2).

In a separate series of experiments, after a study of the effect due to simultaneous stimulation of the hypothalamus and baroreceptor afferents, the carotid arteries were

Research Department of Experimental Cardiology, Leningrad Research Institute of Cardiology, Ministry of Health of the RSFSR. (Presented by Academician of the Academy of Medical Sciences of the USSR N. P. Bekhtereva.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 100, No. 12, pp. 645-647, December, 1985. Original article submitted December 3, 1984.

 0007-4888/85/0012-1612$09.50

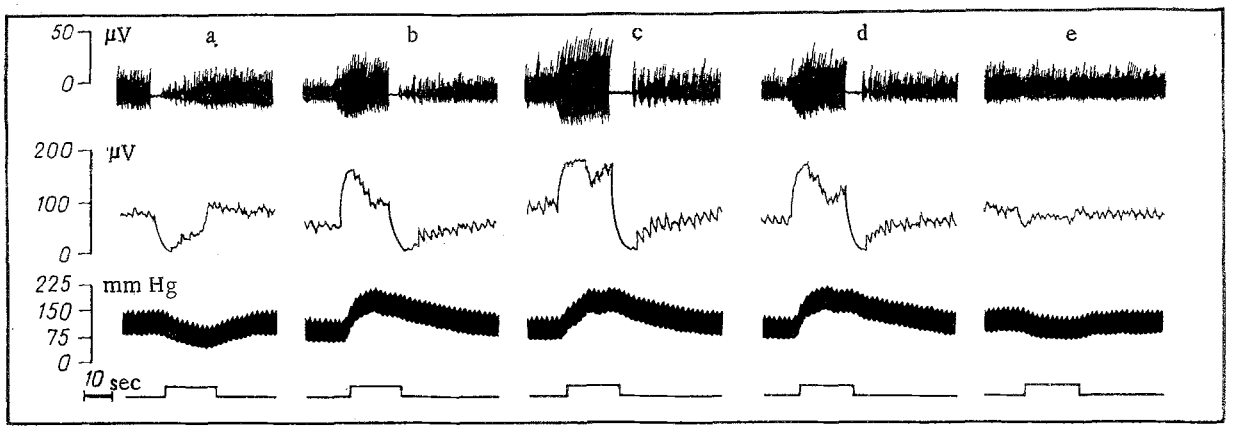

Fig. 1. Effects of separate stimulation of vago-aortic nerve (a), of different points of the hypothalamus (b, c), and of their combined stimulation (d, e). From top to bottom: electrical activity in renal nerve, integrated activity, BP, marker of stimulation (above).

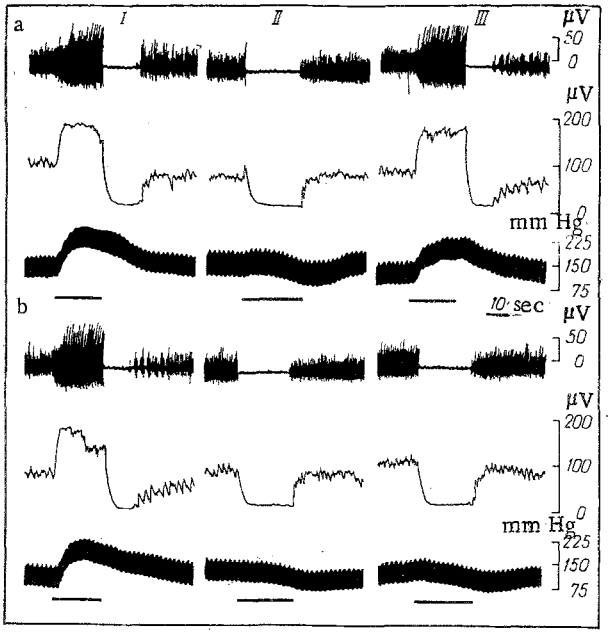

Fig. 2. Effect of chloralose on response to combined stimulation of hypothalamic zone I and vago-aortic nerve: a) before, b) after injection of chloralose. I) Stimulation of hypothalamus; II) stimulation of vago-aortic nerve; III) simultaneous stimulation of hypothalamus and vago-aortic nerve. Remainder of legend as to Fig. 1.

compressed and the second vago-aortic nerve divided. After barodeafferentation, the completeness of which was confirmed by absence of change in vasomotor activity during an artificial rise of BP caused by intravenous injection of phenylephrine (0.1 mg/kg), stimulation of hypothalamic zone I led in four experiments to a fall of BP, amounting on average to 64% of the original pressor response, and only in two experiments were pressor responses observed, which on average exceeded the original levels by 27%. Stimulation of hypothalamic zone II after barodeafferentation led in half of the experiments to an increase in the pressor response on average 53%, and in half of the experiments there was no change in BP or a depressor response was observed.

The effect of barodeafferentation on changes in sympathetic activity in response to

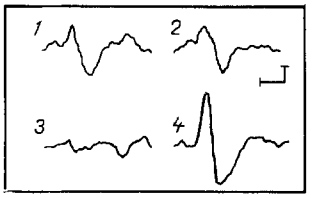

Fig. 3. Evoked response in renal nerve to stimulation of different hypothalamic zones before and after deafferentation of mechanoreceptor zones. 1, 2) Stimulation of zones I and II respectively before compression of carotid arteries and division of right vago-aortic nerve; 3, 4) the same after compression and division. Averaging of 32 responses. Time of stimulation coincides with beginning of sweep. Calibration: amplitude 10 μV, time 100 msec.

stimulation of the various hypothalamic zones also differed. In response to stimulation of hypothalamic zone I after barodeafferentation potentiation of the response by 19% was obtained in only one experiment, and in the remaining five cases activity in the renal nerve either was unchanged or was reduced on average by 29%. Stimulation of hypothalamic zone II after compression of the carotid arteries and division of the vago-aortic nerve in four experiments either did not change the response of vasomotor activity or increases on average by 20%. In two experiments the response of vasomotor activity was reduced on average by 19% of its initial value. In some experiments a response was recorded in the renal nerve to single stimulation of the different hypothalamic zones before and after barodeafferentation. During hypothalamic stimulation, when BR was depressed in response to repetitive stimulation, the evoked response was reduced in amplitude and duration after barodeafferentation. Single stimulation of hypothalamic zone II after barodeafferentation led to an increase in the response (Fig. 3).

The experiments thus showed that stimulation of certain points in the "protective zone" of the hypothalamus leads to depression of the vasomotor component of BR. General anesthetics considerably inhibit hypothalamic modulation of BR, a fact which explains data in the literature [2, 10] showing that baroreflex regulation of vascular tone is maintained in the period of hypothalamic stimulation. Meanwhile the absence of changes in BP and in electrical activity in the renal nerve during stimulation of hypothalamic zone I after baroreceptor deafferentation confirms the view [12] that depression of BR is one of the mechanisms of the elevation of BP during emotional stress.

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