ROLE OF THE α- AND β-ADRENERGIC SYSTEMS IN THE REGULATION OF HEAT PRODUCTION BY SHIVERING IN CATS

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Experiments on cats in which adrenergic drugs (phenylephrine, phentolamine, isoproterenol, and propranolol) were injected into the lateral ventricle showed that α-adrenergic receptors are included in the system activating cold shivering and β-adrenergic receptors in the system inhibiting it.

KEY WORDS: adrenergic drugs; cold shivering.

The role of adrenergic mediators in the central regulation of body temperature is insufficiently clear because of qualitatively contradictory results obtained even in animals of the same species following injection of noradrenalin into the brain [2, 3, 5-8]. In the light of modern views regarding the duality of adrenergic reception [1, 10] it must be considered that one or other action of noradrenalin on the central mechanism of thermoregulation may be due to its action predominantly on α- or β-adrenergic receptors [4, 9, 11].

The results of an investigation of the functional role of α- and β-adrenergic receptors in the mechanism of central regulation of heat production by shivering in cats are described below.

EXPERIMENTAL METHOD

Experiments were carried out on 34 cats anesthetized with chloralose and urethane (50-500 mg/kg), after oxyphenonium premedication (2.5 mg/kg). Cold shivering was induced by lowering the ambient temperature. The brain temperature and subcutaneous temperature on the dorsum of the left and right feet of the hind limbs were measured by thermocouples and recorded on an N-3020 five-channel automatic writer. Parallel recordings on the same writer were made of the integral EMG of the left and right sartorius muscles. The amplifier of the "Medicor" electromyograph with an integrator was used as the preliminary EMG converter. The α-adrenomimetic phenylephrine, the α-adrenolytic phentolamine hydrochloride, the β-adrenomimetic isoproterenol (Novodrin), and the β-adrenolytic propranolol (Obsidan) were used for the analysis. The drugs were injected through a cannula into the external jugular vein or injected, diluted in pyrogen-free bidistilled water, through a cannula implanted stereotaxically into the right lateral ventricle in accordance with coordinates from Maršala's atlas ($AP_{+4}\,L_3V_{+6}$) in a volume of 0.15 ml.

EXPERIMENTAL RESULTS

Propranolol, injected intravenously in a dose of 5 mg/kg against the background of cold shivering, had no effect on the dynamics of shivering or on the subcutaneous temperature in all 12 experiments. Phentolamine (5 mg/kg, intravenously) completely inhibited cold shivering in 13 of 14 experiments 1-3 min after injection; the subcutaneous temperature meanwhile was increased by 3-6°C. The inhibitory action of phentolamine on cold shivering cannot, however, be attributed to its effect on central mechanisms, because of the marked peripheral effect on the cutaneous blood flow.

Blocking central β-adrenergic receptors by injection of propranolol into the lateral ventricle (Table 1) led in all experiments to an increase in the intensity of cold shivering; this effect was particularly marked if

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TABLE 1. Effect of Intraventricular Injections of α- and β-Adrenergic Drugs on Cold Shivering

Drug	Number of experiments	Dose, μg	Effect on cold shivering	Time of onset of effect, sec
Propranolol	7	200	Potentiation	40—70
Isoproterenol	12	50	Abolition	20—40
Propranolol after isoproterenol	12	200	Restoration	40—150
Phenylephrine	11	100	Potentiation	20—90
Phentrolamine	14	500	Abolition	30—150

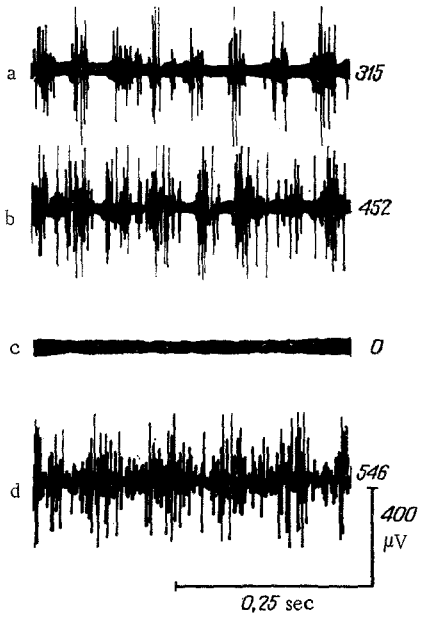

Fig. 1

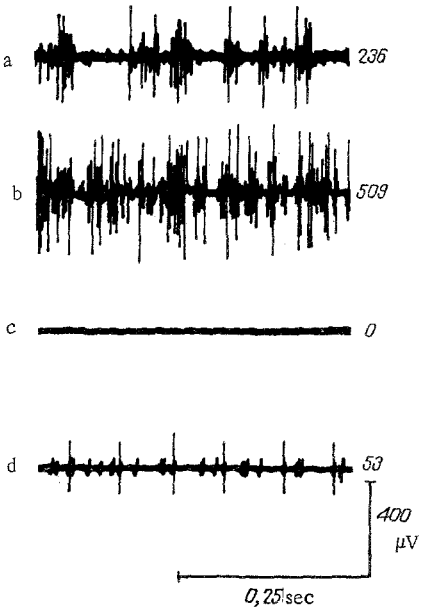

Fig. 2

Fig. 1. Dynamics of interference EMG of sartorius muscle during cold shivering together with activation and blocking of central β-adrenergic receptors: a) beginning of shivering induced by general cooling; b) regular shivering; c) 5 min after intraventricular injection of isoproterenol (50 μg); d) 10 min after intraventricular injection of propranolol (200 μg). Here and in Fig. 2, numbers on right denote number of pulses of integrator per minute.

Fig. 2. Dynamics of interference EMG of sartorius muscle during cold shivering with activation and blocking of central α-adrenergic receptors: a) shivering induced by general cooling; 2) 12 min after intraventricular injection of phenylephrine (100 μg); c) 5 min after intraventricular injection of phentolamine (500 μg); d) 16 min after injection of phentolamine – beginning of restoration of shivering.

the drug was injected against the background of an irregular tremor. Activation of central β-adrenergic receptors by isoproterenol in 11 of 12 experiments caused complete cessation of cold shivering for 1 min. The increase in subcutaneous temperature observed after injection of isoproterenol occurred in most experiments later, after 4-7 min. In response to a subsequent injection of propranolol the shivering, inhibited by isoproterenol, was restored (Fig. 1). Activation of central α-adrenergic receptors by phenylephrine, on the other hand, led to an increase in the intensity of cold shivering for 8-15 min in all 11 experiments without any substantial change in the subcutaneous temperature. When central α-adrenergic receptors were blocked by phentolamine, cold shivering ceased in all the experiments for 1-1.5 min, including shivering activated by phenylephrine (Fig. 2). Elevation of the subcutaneous temperature by 3-8°C after intraventricular injection of phentolamine developed much later than inhibition of cold shivering, namely after 5-12 min.

The differences in the times of these effects on shivering and on the subcutaneous temperature suggest that the changes in cold shivering do not take place through thermoreceptive reflexes from the integument of the body, but that they are the results of the direct action of the drugs on the central mechanism of thermoregulation. Usually all the effects of intraventricular injection of adrenergic drugs on cold shivering and on the subcutaneous temperature arose bilaterally and simultaneously, but in some experiments the effect appeared sooner on the contralateral side.

The results suggest that noradrenalin in cats can behave as the mediator in the activating and inhibitory neuronal systems responsible for the central regulation of cold shivering. The activating effect of noradrenalin in this case is mediated through α-adrenergic and its inhibitory effect through β-adrenergic receptors.

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DEPENDENCE OF ELECTRICAL ACTIVITY OF A MUSCLE ON ITS LENGTH DURING DEVELOPMENT OF A CONSTANT FORCE

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The dependence of the integral electromyogram (EMG) on the length of the muscle during the development of constant force was investigated in relation to the human biceps brachii muscle, while performing flexion at the elbow in a horizontal plane against a constant force of resistance. Both with a static load and during movement the integral EMG is increased several times during shortening of the muscle. The causes of the increase in electrical activity are discussed.

KEY WORDS: biceps brachii muscle; integral electromyogram; change in length.

In so far as the force developed by a muscle during contraction is a linear function of its excitation, the surface electromyogram (EMG), which reflects the level of excitation, can also characterize its strength. In most investigations [1, 4, 9] during isometric contraction of the muscle a near-linear relationship was obtained between the integral EMG and force for small loads. Attempts to extend this result to movements with a change in length of the muscle showed that the relationship is influenced in particular by the character of the movements (shortening or lengthening of the muscle) [3, 11]. The dependence of electrical activity of the

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