# Effects of Electric Stimulation and Destruction of Caudate Nucleus on Short-Term Memory

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The effects of stimulation and destruction of the caudate nucleus on the cat short-term memory were studied by the method of classical delayed reactions. Short-term memory improved, if electrical stimulation of the caudate nucleus before presentation of the conditioned signal caused desynchronization of the electrocorticogram in the prefrontal and temporal cortex. Unilateral destruction of the caudate nucleus leading to the development of hyperactivity and attention disorders deteriorated short-term memory.

**Key Words:** *caudate nucleus; delayed reaction; short-term memory; motor hyperactivity*

Many brain structures are involved in memory regulation. The caudate nucleus (CN) with its presumable modal specific functions attracts special attention [2,3,5,6]. On the other hand, some scientists claim that CN destruction does not cause memory disorders [4] or inhibits some cortical areas.

We studied the effects of CN and its destruction on temporal characteristics of the classical delayed reaction (CDR).

## MATERIALS AND METHODS

Experiments were carried out on adult cats (3.0-3.5 kg). Short-term memory (STM) was studied by indirect method of CDR in a special shielded box consisting of the start and experimental sections divided by a movable wall. Food-getting conditioned reflex was trained: the animals ran to the right feeding rack in response to 250 Hz tone and to the left rack in response to a series of acoustic clicks. Twenty tests were presented during a day. Alternation of racks was performed using Helerman's table. Small pieces of meat of the same size and weight served as the unconditioned stimuli. When discrimination of acoustic signals attained 90-100%, the training of delayed reactions was started. The animals were divided into 2 groups: cats with strong unbalanced nervous system (group 1, $n$=4) and cats with strong balanced nervous system (group 2, $n$=5).

Monopolar constantane electrodes (0.2-0.3-mm tip diameter) were stereotactically implanted implanted into CN (coordinates: A — 17, L — 5, H — 5) under nembutal narcosis (40-45 mg/kg intraperitoneally). CN was stimulated for 1.2-1.4 sec (230-300 μA current, 10-12 Hz frequency, pulse duration 0.2 msec). Electrocorticogram was recorded in the right prefrontal and temporal cortical areas via bipolar silver chlorinated electrodes 0.6 mm in diameter on a Nihon Kohden electroencephalograph. The CN was stimulated with an ESU-1 stimulator. For unilateral destruction of CN, electric current (5-10 mA) was passed through the CN for 30-45 sec using an alternating to direct current converter (TIP AGP-33). The destruction process was controlled on the VC-7A cathode oscillograph monitor (Nihon Kohden). Electric stimulation and destruction of CN was right-sided with a 7-day interval between stimulation and destruction.

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For morphological control of CN destruction (Fig. 1), the animals were sacrificed with high nembutal doses (120-140 mg/kg intraperitoneally), the brain was removed and put into 10% formalin. The area of CN destruction was evaluated on serial sections stained with cresyl violet after Nissl.

## RESULTS

The maximum of CDR (the period during which the animals correctly performed 90% tests) was determined for each animal. In groups 1 and 2 this period was 45-55 and 125-135 sec, respectively. If preliminary electrical stimulation of CN caused desynchronization in the prefrontal and temporal cortex and under these conditions, the animals were offered conditioned acoustic signals causing primary responses in the temporal area, the maximum CDR increased by 10-15 sec in group 1 animals and they correctly performed the food-getting task. Prolongation of CDR by 10-20 sec in response to the threshold electrical stimulation of CN was also noted in group 2 cats. If CN stimulation caused no desynchronization of electrocorticogram in the prefrontal area, the animals did not demonstrated correct CDR despite the appearance of primary responses to conditioned signals.

Electrical stimulation of CN in the presence of electrocorticogram desynchronization was as a rule paralleled by a significant prolongation of CDR (Fig. 2).

Destruction of CN in group 1 cats during the delay caused motor hyperactivity in the start compartment of experimental box, with attempts at getting free and significant deterioration of CDR. The duration of the maximum delay decreased 10-fold (from 50 sec normally to 5 sec; Fig. 3). In group 2 animals, destruction of CN did not lead to so pronounced changes: in contrast to group 1 animals, they were much more quiet and STM deteriorated not so drastically.

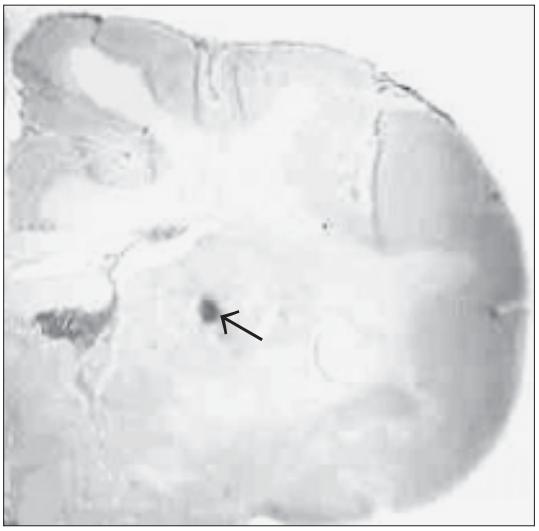

**Fig. 1.** Section of cat brain through CN area, ×10. Arrow shows destroyed site of CN head. Cresyl violet staining after Niessle.

Analysis of the results showed that if preliminary electrical stimulation of CN had a facilitating effect on the electrocorticogram in the prefrontal and temporal areas, the maximum CDR was prolonged by 10-15 sec and the prefrontal area and CN participated in integrative activity as a system [3,6]. Electrical stimulation of CN had a desynchronizing facilitating effect not only on visual and auditory cortex, but also on the prefrontal areas. Areas of electrolytic destruction of the central part of the CN head were detected in the right hemisphere (Fig. 1), which led to reduction of CDR maximum, more pronounced in animals with less balanced nervous

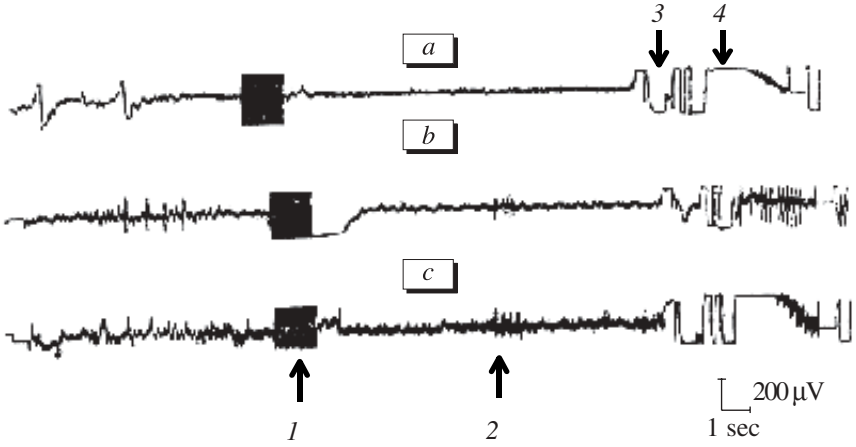


**Fig. 2.** Effect of electric stimulation and acoustic signals on electrocorticogram in the prefrontal area (*a*), electrical activity of the dorsomedial nucleus (*b*), and electrocorticogram of the cortex (*c*). *1*) electric stimulation of CN; *2*) stimulation with series of acoustic clicks; *3*) motor reaction during CDR; *4*) chewing reaction during CDR and food getting.

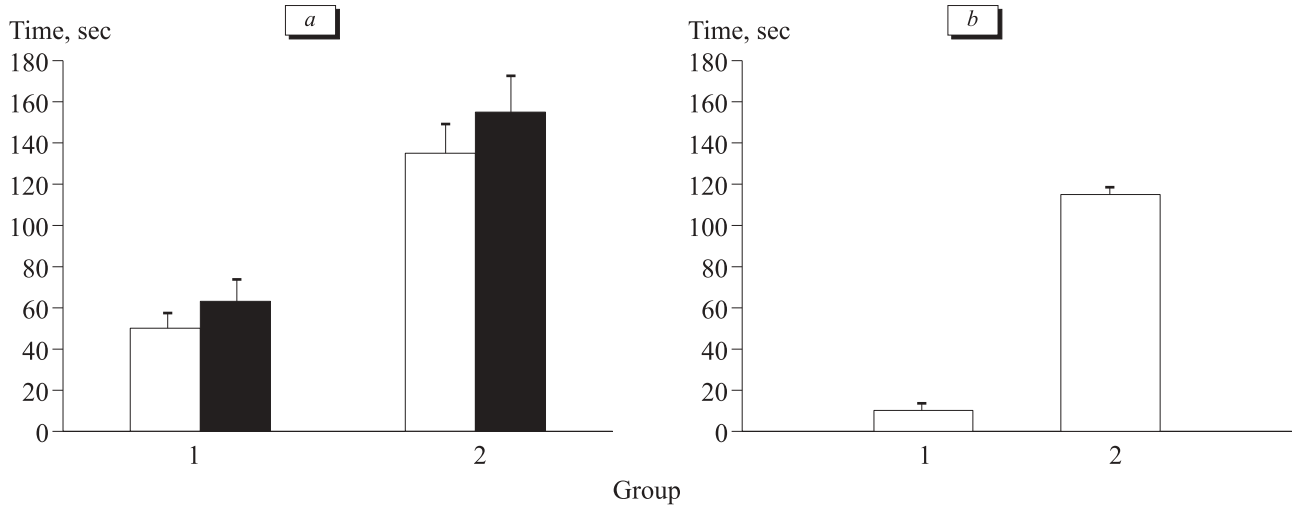


**Fig. 3.** Effects of electrical stimulation (*a*) and destruction of CN (*b*) on the duration of CDR in cats. *a*: Light bars: before stimulation; dark bars: after stimulation.

system. This can be attributed to motor hyperactivity after disappearance of the inhibitory effect of CN on the sensorimotor cortex during the delay and loss of the orientation reaction, which reduced the CDR maximum and deteriorated STM.

Hence, electrical stimulation of CN caused desynchronization of summary bioelectrical activity of the prefrontal and auditory cortex. This effect was paralleled by a synchronizing suppressive effect mainly on the sensorimotor area of brain hemispheres, which contributed to the formation of purposeful orientation and exploratory behavior in response to conditioned acoustic stimulus [1], maintaining, in turn, the perception and fixation of conditioned signals and playing the key role in realization of CDR and STM.

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