to allow cross-bridge attachment only when the muscle passes through its static mean length.

Zusammenfassung. Röntgenstrukturanalysen zeigen, dass die Beziehung zwischen sinusoidaler Kraftentwicklung und der Kinetik der Myosinbrückenzyklen von der Oszillationsgeschwindigheit abhängt.

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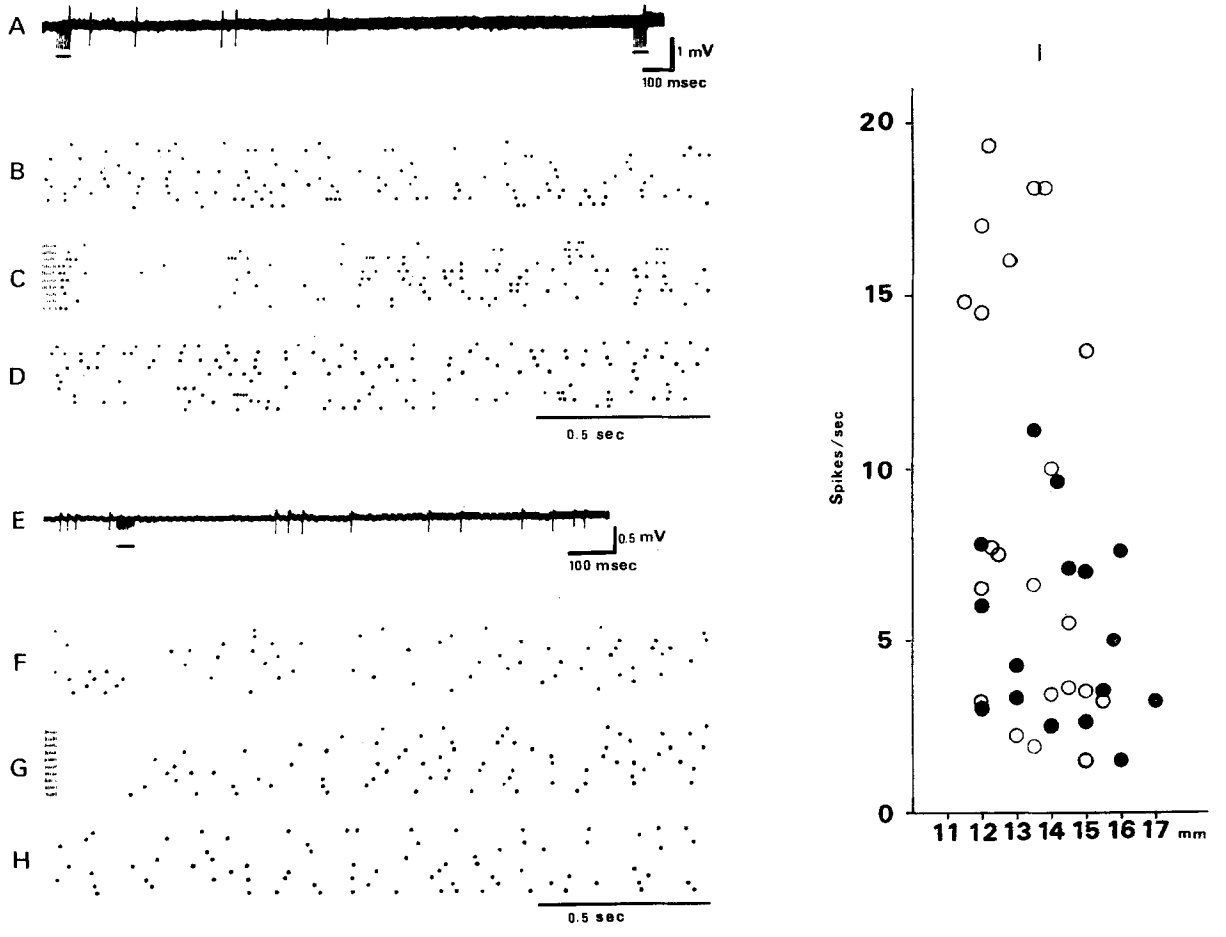
Excitation and Inhibition of Hypothalamic Neurons by Cerebellar Stimulation in Rabbits

A number of investigations have shown autonomic responses to be caused by stimulation of the cerebellum[1-3]. However, there is no agreement as to whether the hypothalamus is involved in the production of these responses[4-8]. The present experiment was undertaken to reveal influences of cerebellar stimulation on unitary activity in the hypothalamus.

Methods. 17 adult rabbits were used. They were anesthetized with i.v. injection of urethane (0.5 g/kg). After fixing the head of the rabbit in a stereotaxic apparatus, a bipolar electrode of stainless steel wire with a tip separation of 1.0 mm was inserted into the cerebellum. It was kept in place at a depth at which brief electrical stimulation at 100 Hz produced most prominent pupillar dilatations.

Electrical stimuli, consisting of 0.1 msec square waves repeated at 100, 200 or 300 Hz, were delivered once per 2 sec for a 30 to 50 msec period. ECG recording showed that this cerebellar stimulation caused no changes in heart rate. At the end of each experiment, an electrolytic



A) A sample record showing excitation. Cerebellar stimulations are marked by short horizontal bars. Single spikes were evoked toward the end of stimulus train. Positivity, downward. B), C) and D) Dot displays of spike discharges from another neuron. B) and D) control records taken before and after C), respectively. C) Early excitations followed by long-lasting suppressions due to cerebellar stimulation taken with 8 pulses at 200 Hz. E) A sample record showing inhibition. F), G) and H) Dot displays of spike discharges from another neuron. F) and H) Control records taken before and after H), respectively. H) Suppressions of spike discharges, immediately consequent upon cerebellar stimulation with a train of 8 pulses at 300 Hz. I) A plot of spontaneous firing rates of hypothalamic neurons as a function of the recording depths measured from the cortical surface. Open circles, excitation (number of units, 22). Filled circles, inhibition (16).

coagulation was produced by passing a direct current through the cerebellar electrode. Paraffin sections were made and stained by cresylecht violet. Electrolytic lesions were found in the anterior vermal cortex or within or around the fastigial nucleus.

Glass capillary microelectrodes filled with 3 *M* KCl were inserted into the medial anterior hypothalamus according to SAWYER's atlas[9]. Extracellular spikes were recorded from an area extending from the ventromedial nucleus to the medial preoptic area.

After spontaneous discharges were recorded for 20 sec, effects of cerebellar stimulation were examined. The stimulus was repeated 10 times at intervals of 2 sec. Then the baseline discharges were again recorded for 20 sec.

Results. In 62 units recorded, the rate of spontaneous discharge in the control stage ranged from 0.5 to 38.7 spikes/sec with an average of 8.5 spikes/sec. The cerebellar influence was excitatory in 22 units and inhibitory in 16 units. The remaining 24 units did not respond.

Effects of cerebellar stimulation were evaluated as excitatory by the following criteria: 1. Unitary spikes were evoked by stimulation. 2. The spontaneous firing rate increased by 50 to 100% of control, while cerebellar stimulation was repeated, and returned approximately to the control rate after stimulation was stopped.

Figure A is an example of excitation. In this unit a single spike was evoked in a latency of 32 msec measured from the first stimulus pulse. Figures B, C and D are dot displays of spike discharges from another unit. B and D are control records. In C it is seen that single or repetitive spikes were evoked in a latency of 10 msec upon cerebellar stimulation and that this excitatory effect was followed by a long inhibition.

Cerebellar stimulation was judged as inhibitory when the spontaneous firing pattern changed in the following way: 1. An almost complete cessation of the spontaneous discharges followed cerebellar stimulation immediately and lasted for several hundreds msec. 2. The spontaneous discharge rate decreased to less than 50% of control for a 20 sec period during cerebellar stimulation and returned approximately to the control rate after stimulation.

Record of Figure E is to exemplify the cerebellar inhibition. In this unit, spike production was suppressed for 250 msec from the beginning of cerebellar stimulation. Figures F, G and H are dot displays spike of discharges of another neuron. Suppresion of spike discharges following cerebellar stimulation (G) was judged as significant, taking the baseline discharges of F and H as control. The mean duration of cerebellar inhibition measured from the last stimulus pulse was 494 $\pm$ 176 msec (mean $\pm$ S. E., $n = 10$).

In Figure I the spontaneous discharge rates of the hypothalamic units (ordinates) are plotted against the recording depths measured from the cortical surface (abscissae): Empty circles denote excitation and filled ones inhibition. Excitation is distributed over a wide range of discharge rates, whereas inhibition occurs preferentially in neurons with low discharge rates.

Discussion. The excitation of hypothalamic neurons by vermal lobe stimulation is in accord with the findings of SAWYER et al.[7] in rabbits. They found that stimulation of the vermis caused EEG arousal in VMH and the preoptic area. Stimulation of the vermal cortex causing an increment of spontaneous discharges of hypothalamic neurons seems to contradict the current theory of Purkinje cell action. However, ITO et al.[10] recorded the late facilitatory depolarization from cells of the medullary reticular formation following stimulation of the vermal cortex. Similar potentials may be evoked in cells of the mesencephalon by stimulation of the vermal cortex. Excitatory impulses produced there may then be conducted to the hypothalamic neurons.

The cerebellar inhibitory influence on hypothalamic neurons is not likely to be due to direct inhibitory impulses arriving at these neurons. It appears to be due to a decrease or cessation of excitatory impulses to the hypothalamus[11].

Zusammenfassung. Der Einfluss elektrischer Kleinhirnreizung auf die spontane Neuronenaktivität des medialen Hypothalamus wurde untersucht. Nach Reizung des Vermis kam es zur Erhöhung und Depression der Spontanaktivität.

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Intraaxonal Iodate Inhibits Sodium Inactivation[1, 2]

Recent experiments on single Ranvier nodes undertaken with Prof. J.F.W. KEANA[3] to test the action of specific chemical reagents on ion-specific potential and time dependent pathways across the excitable membrane showed, among other results, a considerable lengthening of the action potential if KIO_3 diffused into the axon from the cut ends. Superfusion of the nodal membrane with Na-IO_3 had no striking action, either on action potentials or on Na, K and leak currents. I have now done some more experiments to check the origin of the lengthening of the action potential by intraaxonal application of IO_3 ions.

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