off-Zentrum-Neurone, wie auch die Phasenverschiebung des Maximums der Aktivierung während der Dauer einer Sinuslichtperiode näherungsweise linear mit der Frequenz zu. Phasenverschiebungen bis zu 4 π wurden beobachtet. Die mittlere Entladungsrate nahm wie bei rechteckig moduliertem Flimmerlicht[2,3] mit steigender Sinuslichtfrequenz zunächst zu, erreichte zwischen 2–10 Sinuslichtreizen pro sec ein Maximum und nahm darüber hinaus wieder ab. Dieses Maximum lag um so höher, je heller das Sinuslicht war.

Die maximale oder «kritische» Sinuslichtfrequenz (CSF), auf die gerade noch eine regelmässige neuronale Antwort folgte, nahm mit Erhöhung der Leuchtdichte zu. Mit kombinierter Reizung des rezeptiven Feldzentrums und der Feldperipherie konnte die Phasenlage der Aktivierung relativ zum Sinuslichtreiz variiert werden[4].

Summary. The action potentials of optic nerve fibres in cats were stereotactically recorded (light pentobarbital anaesthesia, Curarin-immobilization). The activation of retinal neurons stimulated by light spots within the receptive field centre was a non-linear function of the sinusoidal intensity modulation of the stimuli.

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The Mode of Cerebral Activation of Red Nucleus Neurones

Histological investigations have revealed that the giant neurones in the cat's red nucleus (RN) receive projections mainly from two sources, i.e. the interposed nucleus of the cerebellum (IP) and the sensorimotor cortex of the cerebrum (SM). The mode of activation of RN cells by the former has been reported previously[1]. This article reports further on the latter projection which will be shown to be organized somatotopically and exerted upon the remote dendritic membrane of RN cells.

Cats were anaesthetized with pentobarbitone sodium. The procedure of dissection and the method of intracellular recording from RN cells were the same as those employed previously[1]. The ipsilateral sensorimotor cortex was stimulated by means of needle electrodes assembled at intervals of 2 mm. One set of 5 electrodes was placed on the pre-cruciate gyrus and another on the post-cruciate gyrus, their tips being inserted to a depth of about 1 mm below the cortical surface. A similar array of electrodes was inserted stereotaxically into the ipsilateral internal capsule. Pulses of 0.1msec duration were applied in turn between neighbouring poles in each set.

When the sensorimotor cortex (SM) was stimulated, an EPSP was produced in RN cells with a latency of about 2 msec. The EPSP rose slowly with a summit time of about 4 msec, and decayed within about 25 msec (Figure A, B). Its slow time-course is prominent when compared with that of the EPSP produced from the interposed nucleus (IP) (Figure E). The same slow EPSP could be induced from the internal capsule (IC) with a shorter latency of about 1 msec (Figure C, D). The SM and IC stimuli interfered with each other, indicating that both slow EPSPs were caused by the same fibres. The short latency from IC further indicated that the fibres impinge upon RN cells monosynaptically.

According to the histological observations[2,3], there is a cortico-rubral projection which is arranged in a somatotopical way: from the forelimb cortical area to the upper spinal segment through the dorso-medial, and from the hindlimb cortical area to the lower spinal segment through the ventrolateral part of the red nucleus. This mode of projection was demonstrated for the cerebral-induced EPSPs. The rubrospinal tract was stimulated at the second cervical and first lumbar segments. In neurones invaded antidromically from both levels (lumbosacral, LS-cells) EPSPs were evoked predominantly from the medial part of the pre- and postcruciate gyri, while in those activated only from the second cervical segment (cervico-thoracic, CT-cells), EPSPs were induced from the lateral part of the same gyri. The most effective cortical site for each RN cell is mapped in Figure J. The open circles for CT-cells fall in the forelimb area as defined by its motor effect, and the closed ones for LS-cells in the hindlimb area, there being no overlap between these two groups of circles. This agreement between the anatomical and physiological observations indicates that the cerebral-evoked EPSPs are produced through the cortico-rubral projection.

The cerebral-induced EPSP differed from the cerebellar-induced one in its sensitivity to alteration of the membrane potential. In Figure F these EPSPs are displayed on the same sweep for comparison, and in Figures G and H hyperpolarizing currents were passed through the impaled microelectrode. With currents of $4 \cdot 10^{-9}$ A (G) and $8 \cdot 10^{-9}$ A (H), the cerebellar-induced EPSP (IP) increased appreciably, as shown by the open circles in Figure I. On the other hand, cerebral-induced EPSPs were influenced neither in amplitude (closed circles in Figure I) nor in time-course. The insensitivity of the cerebral-induced EPSPs to alterations in membrane potential can be explained if it is assumed that cortico-rubral fibres make synaptic contact in the dendritic portion of RN neurones, in just the same way as has been suggested for the recurrent EPSPs in the frog motoneurones[4]. This view is in keeping with the histological

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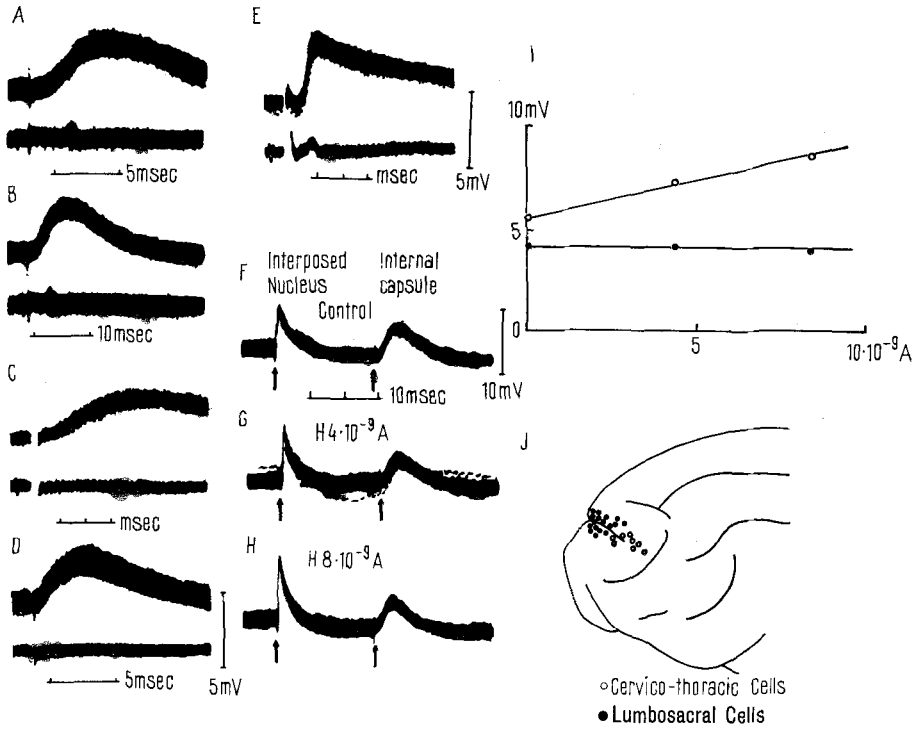

A–E, EPSPs recorded intracellularly from RN cell. A and B, EPSP induced from the sensorimotor cortex (upper traces). Lower traces are field potentials taken just after withdrawal. C and D, EPSP from the internal capsule. Voltage scale of 5 mV applies to A–D. E, EPSP from the interposed nucleus of the cerebellum. F–H, EPSPs in another RN cell induced from the interposed nucleus (IP) and the internal capsule (IC). Upward arrows mark the moments of their stimulation. F, control. G, during passage of hyperpolarizing currents of $4 \cdot 10^{-9}$ A. H, $8 \cdot 10^{-9}$ A. All records were taken by superposing about 20 faint traces. I, ordinates, amplitudes of the IP-evoked EPSPs (open circles) and IC-induced ones (closed circles). Abscissae, intensity of applied currents. J illustrates the sites from which the maximum EPSP was induced in each RN neurone.

finding that the cortico-rubral fibres end in the neuropil of the red nucleus where abundant axo-dendritic contacts exist[2]. The slow time-course of the EPSP is explicable through consideration of the cable properties of dendrites.

Zusammenfassung. Elektrische Reizung des sensorimotorischen Kortex ruft monosynaptische EPSPs, in den grossen Zellen des N. ruber der Katze hervor (RN-Zellen). Die kortikalen Punkte, von denen EPSPs hervorgerufen werden können, sind somatotopisch so angeordnet, dass sie mit der histologischen Projektion der kortikorubralen Fasern übereinstimmen. Membran-Hyper-Polarisierung, die stark genug ist, um EPSPs vom N. interpositus zu vergrössern, hat keinen Einfluss auf Amplituden oder Zeitfluss von cerebral hervorgebrachten EPSPs. Es wird daraus geschlossen, dass kortiko-rubrale Fasern synaptischen Kontakt mit den Dendriten von RN-Zellen machen.

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PRO EXPERIMENTIS

Bestimmung der freien Hexosamine in Serum und Harn

Glukosamin und Galaktosamin sind wichtige Bausteine von Glykoproteiden und Mukopolysacchariden. Ihre Bestimmung befasst sich meistens mit den proteingebundenen, durch saure Hydrolyse abspaltbaren Aminozuckern[1–3]. Der Erfassung der freien Hexosamine des Serums wird daher weniger Beachtung geschenkt, zumal man durch ihre relativ hohe Einbaugeschwindigkeit in die Glykoproteide eine verwertbare Messgrösse für den Aminohexosenmetabolismus zu haben glaubt[4]. Im diabetischen Stoffwechselgeschehen interessiert hierbei, inwieweit Glukose vermehrt in Glukosamin umwandelbar ist und hierbei besonders bei Glukosehaushaltsstörungen infolge Insulinmangel. SPIRO[5] nimmt an, dass der glykolytisch dekompensierte diabetische Stoffwechsel die Neubildung von Glukosamin bevorzugt, bzw. bei Blockade aller anderen Abbauwege die Glukoseutilisation nur noch in Richtung zum Glukosamin verläuft.

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