somewhat longer in comparison with the respective peaks of the primary evoked potential.

Photic responses recordable from other parts of the lateral surface of the hemisphere represent the third group of evoked potentials. The typical feature of these evoked potentials is the dominating negative phase. The initial positive wave is very seldom detectable. As far as the positive phase is recorded, its latency is from 11 to 16 msec. The onset of the following negative phase has a latency from 14 to 20 msec. The predominating negative phase peaks at 26 to 31 msec. On the ascending limb of this negative wave a small notch is detectable primarily within the sigmoidal gyri.

Discussion. Findings obtained during our experiments are comparable with results of KREINDLER's laboratory[9] and of other authors[10–13]. But it is obvious from our findings that the short-latency photic evoked potentials are quite well detectable from other cortical areas than have hitherto been defined.

There remain especially two questions to be answered. First, the question of the electrogenesis of these extraprimary evoked potentials. We are in agreement with KREINDLER's[13] point of view that the extraprimary evoked potentials are realized by thalamic afferences via the paucisynaptic chains and joining predominantly apical dendrites in the superficial layers of the cortex.

Secondly, the question of their thalamic relay. According to VASTOLA[14], there is a direct pathway passing from the lateral geniculate body into the paraoptic areas of the cortex. CHANG's[15] opinion is that the transmission of diffuse cortical photic responses beyond the primary visual area might start from the ventral part of the lateral geniculate body. Only continued investigations of this problem will reveal a definite answer.

Zusammenfassung. Die extraprimären optischen Potentiale, die in der somatosensorischen, akustischen und Assoziationsrinde der wachen Katzen mittels chronischer epiduraler Elektroden registriert wurden, zeigen fast dieselbe Latenz wie die primären Potentiale, unterscheiden sich aber von ihnen durch ihre Form.

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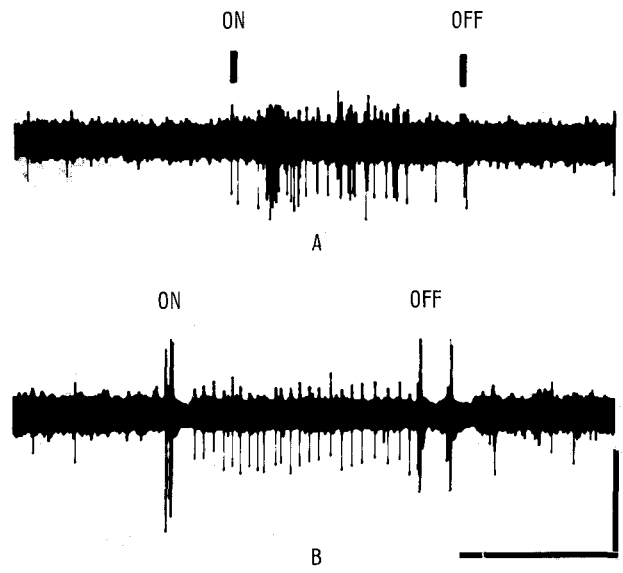
[9] M. STERIADE, M. DEMETRESCU, and J. KREINDLER-MANOLESCU, Stud. cercet. neurol., Bucharesti 7, 97 (1962).
[10] V. HAVLÍČEK, A. MEDEK, V. JANOUT, and J. HRBEK, Acta univ. olomuc. *33*, 211 (1963).
[11] J. BRUNER and R. SINDBERG, J. Physiol., Paris *54*, 303 (1962).
[12] L. I. LEUSHINA, J. Physiol., USSR *59*, 144 (1963).
[13] A. KREINDLER, Gagrskie besedy *4*, 369 (1963).
[14] E. P. VASTOLA, J. Neurophysiol. *24*, 469 (1961).
[15] H.-T. CHANG, cit. in [13].

Actions of L-Glutamate, Acetylcholine and Dopamine on Single Neurones in the Nuclei cuneatus and gracilis of the Cat

The dorsal column nuclei (nucleus cuneatus and nucleus gracilis) are the major terminations of primary afferent fibres from body surface and joint receptors. Individual units in these nuclei are easily identified by means of electrophysiological techniques. It was tempting to examine this pool of secondary sensory neurones with respect to the action of microelectrophoretically applied drugs[1]. A similar study has been successfully completed on the vestibular nuclei[2].

The experiments were made on 21 adult cats under sodium pentobarbital anaesthesia. The medulla oblongata was exposed and the caudal parts of the cerebellum were removed. The activity of neurones in the dorsal column nuclei was recorded extracellularly with the central barrel (filled with 4 *M* NaCl) of a five-barrelled glass micropipette. The total tip diameter was 2–8 μ. One of the four remaining barrels was always filled with 1/6 *M* NaCl for current control and the other barrels contained strong solutions of acetylcholine-Cl (1–2 *M*), Na-L-glutamate (1 *M*), and dopamine HCl (1–2 *M*) respectively. Electrophoretic current of 20–120 nA for expelling the ions was used. For identification of neural elements, the following types of physiological stimulation were applied: hair movement, light skin touch, and joint movement.

Out of 250 cells 132 responded to adequate ipsilateral peripheral stimulation with an increase in the discharge rate and 6 with a decrease. *L-glutamate* activated 50% of the cells which had been influenced by ipsilateral stimulation



Response of a cell in the nucleus cuneatus to ipsilateral peripheral stimulation of the receptive field on the forearm (A) and to L-glutamate-microelectrophoresis [60 nA] (B). Time mark 1 sec Vertical calibration is 0.3 mV.

[1] G. C. SALMOIRAGHI and F. A. STEINER, J. Neurophysiol. *26*, 581 (1963).
[2] F. A. STEINER and G. WEBER, Helv. physiol. Acta *23*, 82 (1965).

(Figure) and 40% of those that remained indifferent to peripheral stimulation. The effect of L-glutamate occurred after a latency of 0.2–0.5 sec, and the discharge rate returned to control levels of activity after 0.1–0.3 sec. These findings appear to be in contrast with results obtained in other brain regions, where practically all cells were activated by L-glutamate[3,4]. However, considering that 40% of the cells recorded in the dorsal column nuclei are presumably presynaptic units[5] not responding to L-glutamate administration, the percentage of influenced units is likely to be considerably higher.

Acetylcholine acted only on a few neurones. The effect occurred after a relatively long latency of 15–50 sec, and when the microelectrophoretic current ceased, the activity change persisted for 10–50 sec. Two units localized in the dorsal column nuclei were inhibited by acetylcholine, one of these responded to peripheral stimulation. Three cells that had not been influenced by peripheral stimulation were activated by acetylcholine. These sparing effects suggest that the relay cells of the dorsal column are not themselves activated by cholinergic nerve transmission. The few acetylcholine-sensitive units may be related to other fibre systems which interact with the dorsal column system at the level of the nuclei gracilis and cuneatus (e.g. collaterals of the pyramidal tract, etc.).

Dopamine inhibited a majority of cells (8 of 11) responding to peripheral stimulation and only a minority of cells (3 of 13) that were indifferent to peripheral stimulation. The inhibitory effect occurred after a latency of 0.5–4.0 sec and vanished after the same delay. Dopamine consistently antagonized the facilitatory effect of L-glutamate.

Zusammenfassung. Von 250 Neuronen im Gebiet des Nucleus cuneatus und des Nucleus gracilis der Katze wurden 132 durch periphere physiologische Reizung aktiviert und 6 gehemmt. Mit Hilfe der Mikroelektrophorese wurden die folgenden Substanzen in die unmittelbare Nähe dieser Neurone gebracht: L-Glutaminsäure aktivierte 50% der Neurone. Acetylcholin war nur an wenigen Zellen im Kerngebiet wirksam, von diesen wurden 2 gehemmt und 3 aktiviert. Dopamin hemmte die Mehrzahl der durch periphere Reizung beeinflussbaren Neurone.

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Abteilung für experimentelle Medizin, F. Hoffmann-La Roche & Co. AG Basel und Institut für Hirnforschung der Universität Zürich (Switzerland),
August 2, 1965.

[3] D. R. Curtis, Brit. med. Bull. *21*, 5 (1965).
[4] P. B. Bradley and J. H. Wolstencroft, Brit. med. Bull. *21*, 15 (1965).
[5] D. L. Winter, J. Neurophysiol. *28*, 48 (1965).

Storage Function and Amine Levels of the Adrenal Medullary Granules at Various Intervals after Prenylamine Treatment

Independent work by Hillarp et al.[1] and Kirshner[2,3] has shown that the amine granules of the adrenal medulla are able to take up and concentrate monoamines *in vitro* by a Mg^{++}-ATP dependent storage mechanism. The storage mechanism is blocked by low concentrations of reserpine. In a second paper Hillarp et al.[4] analysed the uptake mechanism in further detail. Apart from reserpine, prenylamine [N-(diphenylpropyl)-amphetamine] proved to be the most potent inhibitor of the uptake mechanism. In higher, but still very low, concentrations it caused complete release of the granule amines. Similar effects were found in adrenergic nerve granules[5,6].

In a previous paper the storage function of the adrenal medullary granules was studied at various intervals after reserpine treatment[7]. In the present work adrenal medullary granules were examined in essentially the same way at different intervals following injection of a single dose of prenylamine to rabbits.

Methods. Rabbits weighing about 1.5 kg were injected with prenylamine (5 mg/kg) intravenously. At different intervals following injection (1–12 h) the rabbits were sacrificed by an injection of air intravenously. The adrenals were immediately removed and chilled with ice. The medulla with some adhering cortical tissue was rapidly dissected and homogenized with a loose-fitting plastic pestle for about 20 sec in 7 ml of 0.3 *M* sucrose. To remove unbroken tissues and cells, but at the same time to prevent loss of amine granules, the homogenate was centrifuged at 800 *g* for 5 min. The supernatant was centrifuged at 20,000 *g* for 20 min. The sediment was suspended in 0.5 ml 0.3 *M* sucrose. The granule suspension was transferred to 1.0 ml of an incubation mixture (at 0°C) containing 0.31 *M* glycyl-glycine (pH 7.3 with NaOH), 0.0025 *M* ATP and $MgCl_2$, 25 μg unlabelled adrenaline, 4.5 μg C^{14}-labelled adrenaline.

Incubation was performed without shaking at 31°C for 30 min, after which the suspension was chilled to 0°C, diluted 30 times with cold 0.5 *M* sucrose and – after about 1 h at 0°C – centrifuged at 74,000 *g* for 30 min. After thorough rinsing of the tubes with 0.5 *M* sucrose, the granule sediment was extracted with 5.0 ml of 0.01 *N* HCl in 98% ethyl alcohol. The catecholamine content of the extracts was determined spectrophotofluorimetrically[8]. The C^{14}-amine content was determined directly in a liquid scintillation counter. Pure nucleotides from the Pabst laboratories and pure prenylamine-lactate (Segontin) generously supplied by Hoechst, Frankfurt a. M. were used. *dl*-Adrenaline-7-C^{14} was purchased from Commis-

[1] A. Carlsson, N.-Å. Hillarp, and B. Waldeck, Med. exp. *6*, 47 (1962).
[2] N. Kirshner, Science *135*, 107 (1962).
[3] N. Kirshner, J. biol. Chem. *237*, 2311 (1962).
[4] A. Carlsson, N.-Å. Hillarp, and B. Waldeck, Acta physiol. scand. *59*, Suppl. 215 (1963).
[5] U. S. v. Euler, L. Stjärne, and F. Lishajko, Life Sci. *3*, 35 (1964).
[6] U. S. v. Euler, L. Stjärne, and F. Lishajko, Life Sci. *11*, 878 (1963).
[7] P. Lundborg, Exper. *19*, 479 (1963).
[8] Å. Bertler, A. Carlsson, and E. Rosengren, Acta physiol. scand. *44*, 273 (1958).