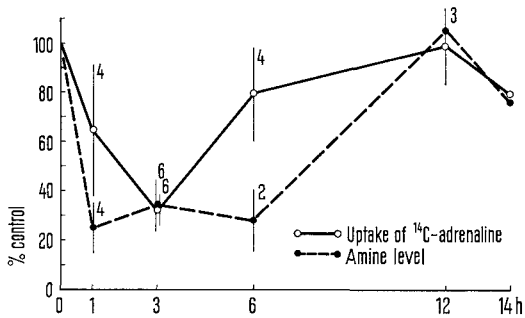
sariat de l'Energie Atomique, France, and stock solutions in 0.01 *N* HCl were stored at −30°C.

Results. In each experiment one or two prenylamine-treated and one control animal were used. In each experiment the amount of incorporated C^{14}-amines after prenylamine injection is given in % of the control value. The amine levels of the granules are also given in % of the control values.

Prenylamine caused a pronounced blockade of the uptake of adrenaline by the storage granules (Figure). The effect was observed 1 and 3 h after the injection. After 6–12 h the incorporation was restored. Also there was a pronounced reduction of catecholamine levels in the medullary granules. 3 h after prenylamine treatment the amine content was only 25% of normal. After 12 h the amine levels of the granules are restored.



Adrenaline level and uptake of C^{14}-adrenaline by adrenal medullary granules in vitro at various intervals following injection of prenylamine (5 mg/kg) i.v. in rabbits. The bars indicate s.e.m. and the figures the numbers of experiments.

Prenylamine thus resembles reserpine in blocking the storage function of the adrenal medullary granules not only when added in vitro but also after an intravenous injection of the drug to rabbits. The effect is not as long-lasting as after reserpine. After reserpine the storage function was restored within 48 h while the amine levels remained very low for a much longer time. After prenylamine both storage function and amine levels of the granules were rapidly restored. Experiments are in progress to study the effects in further detail[9].

Zusammenfassung. Es wurde der Speichermechanismus der Amingranula im Nebennierenmark von Kaninchen, in verschiedenen Intervallen, nach Injektion einer Dosis Prenylamin (5 mg/kg intravenös) untersucht. Prenylamin zeigt dabei einen reserpin-ähnlichen Effekt auf Nebennierenmarkgranula nicht nur in vitro, sondern auch nach intravenöser Injektion.

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Department of Pharmacology, University of Göteborg (Sweden), September 6, 1965.

[9] Acknowledgments: This work has been supported by grants from the Medical Faculty, University of Göteborg (Sweden). For the generous supply of prenylamine I am indebted to Hoechst Anilin AB, Göteborg, Sweden. For technical assistance I am indebted to Miss I. PETTERSSON.

Vestibular Origin of the Rapid Eye Movements During Desynchronized Sleep[1]

The neuronal mechanisms which are responsible for the appearance of the rapid eye movements (REM) during desynchronized sleep have been analysed. The importance of these REM, which have been described both in animals[2] and man[3], is stressed by the fact that in human sleep they are related to the visual content of dreams[3]. Attention was concentrated on the vestibular complex because (a) single vestibular neurones recorded from unrestrained, unanaesthetized cats during desynchronized sleep show bursts of rapid discharge associated with the REM[4], and (b) the second-order vestibular neurones control the oculomotor activity[5].

Methods. The experiments were performed on 15 unrestrained, unanaesthetized cats. Electrodes for recording electroencephalographic activity, the cervical electromyogram, and eye movements (electro-oculogram) were permanently implanted following a technique which has been previously described[6]. Electrolytic lesions of the vestibular nuclei were made using electrodes oriented with the Horsley-Clarke stereotaxic apparatus. The electrodes were inclined 30 degrees from the vertical axis, thus avoiding the bone of the tentorium. Recording sessions were made before and after the vestibular lesions.

Results. During the desynchronized episodes of sleep, two types of ocular movements were observed in the normal animals: (a) the well-known bursts of REM, which were binocularly synchronous, conjugate and grouped in clusters, and (b) slower, non-conjugate movements, which were present sporadically during the periods of quiescence intervening between REM. Occasionally isolated jerks of both eyes occurred. These phenomena were controlled for several days prior to making the lesions.

Bilateral lesions of the vestibular nuclei did not prevent the normal rhythm of sleep and wakefulness, nor did they substantially alter the phases of synchronized and desynchronized sleep.

The most remarkable change was the complete abolition of the bursts of REM typical of desynchronized sleep. Only slow ocular movements could be detected after such lesions. At times rare and isolated jerks of the eyes were observed. The phase of deep sleep in these

[1] This investigation was supported by PHS research grant NB-02990-04 from the National Institute of Neurological Diseases and Blindness, N.I.H., Public Health Service (USA).

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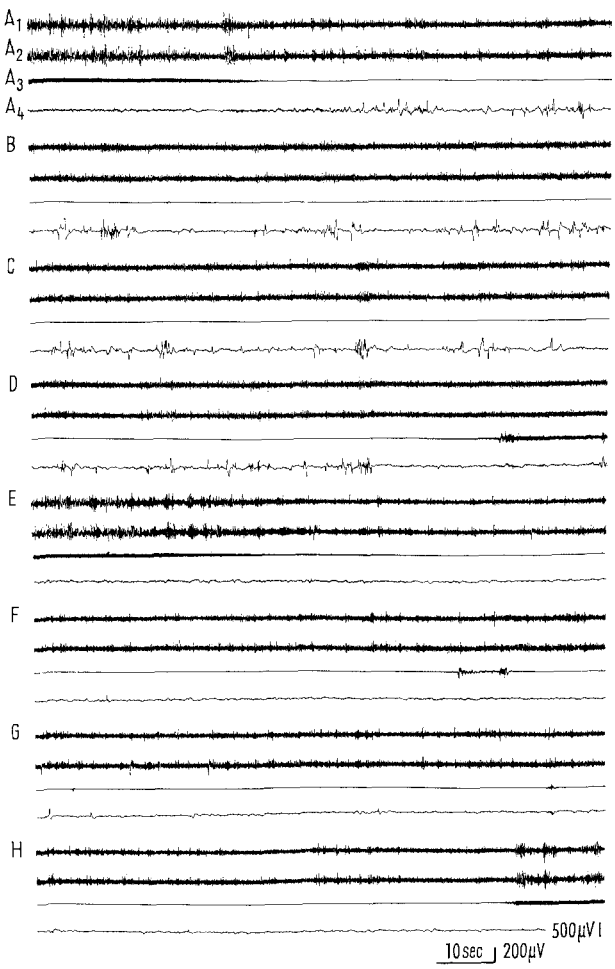

Abolition of the bursts of rapid eye movements during desynchronized sleep following vestibular lesions. Unrestrained, unanaesthetized cat. 1, left parieto-occipital; 2, right parieto-occipital; 3, EMG from posterior cervical muscles; 4, ocular movements (electro-oculogram). A–D, episode of desynchronized sleep recorded in the intact animal 4 days following chronic implantation of the electrodes. Note the occurrence of large bursts of rapid eye movements when the EMG becomes silent. E–H, episode of desynchronized sleep recorded in the same animal 2 days following a chronic bilateral lesion of the medial and descending vestibular nuclei. Calibration of 500 μV applies only to channel 4, calibration of 200 μV applies to channels 1–3.

preparations was characterized simply by desynchronized electrocortical activity and by complete relaxation of the posterior cervical muscles. The abolition of the REM during desynchronized sleep was not a transient phenomenon, but persisted throughout the survival period (up to 23 days).

Control experiments showed that the REM were still present following complete cerebellectomy and/or bilateral section of the VIII nerve. The changes which have been observed are therefore due to destruction of second-order vestibular neurones.

The effects described above were seen when the lesion was symmetrical and affected completely the vestibular nuclei of both sides. Bilateral electrolytic lesions limited to the medial and descending vestibular nuclei were also equally effective (Figure). Unilateral lesion of the vestibular nuclei or bilateral lesion limited to the superior and lateral vestibular nuclei, however, did not prevent the appearance of the bursts of REM.

It is of interest that in the intact animal the activity of the units recorded from the superior and lateral vestibular nuclei remains unmodified during desynchronized sleep, while the units in the medial and descending vestibular nuclei show bursts of rapid discharge synchronous with the bursts of REM[4].

The present experiments show that the medial and descending vestibular nuclei are of critical importance for the appearance of rapid eye movements during desynchronized sleep.

Riassunto. I movimenti rapidi oculari caratteristici del sonno desincronizzato nel gatto dipendono dall'integrità anatomica e funzionale dei nuclei vestibolari.

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August 30, 1965.

PRO EXPERIMENTIS

A Method for Revealing Inhibition of Virus-Induced Synthesis of RNA

In the search for virus-specific inhibitors various methods of testing are applied, a plaque assay method being most often used[1]. This method enables the determination of the rate of inhibition by a given compound of virus production, which is composed of cell-specific and virus-specific synthetic processes. It is, however, important to measure the inhibition of virus-induced synthesis, particularly the synthesis of viral nucleic acids.

It was established that the synthesis of cellular RNA is dependent on DNA while the synthesis of viral RNA in one-stranded RNA viruses is determined by viral RNA itself[2]. However, the revelation of the synthesis of viral RNA is difficult, because the rate of the latter is considerably lower than the rate of the synthesis of cellular RNA. Therefore actinomycin D (C_1), which inhibits the synthesis of DNA-dependent RNA and does not inhibit the synthesis of viral RNA[3], can be used in experiments

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