methionine (WISEMAN[8]). The absorption mechanism may be influenced by several factors, as for instance by the preceding food intake (DONHOFFER[9]). In the case of protein deficiency, production of the protein-splitting enzymes in the pancreas may suffer an injury. For the absorption of S^{35}-methionine, no protein-splitting enzymes are necessary and so it is obvious that its absorption through the intestinal wall is not considerably affected by the protein-deficient condition of the organism.

Remarkable was the difference in the absorption of I^{131}-triolein after protein deficient diet had been given for a longer period. I^{131}-triolein is the ester of three oleic acid groups and one glycerine molecule. Each oleic acid group contains a double bond. According to its chemical nature the administered isotope is a fat, thus when investigating its absorption, one must consider the extensive digestive activity preceding fat resorption (VERZÁR and KUTHI[10], FRAZER[11]). For the changes in the absorption of I^{131}-triolein, the secretive activity of the intestinal epithelial cells, as well as of the bile and pancreas, may be responsible. To elucidate to what an extent these three factors take part in this action needs further investigation. Besides, it is possible that the energy stores of the protein deficient organism are too much decreased for the mechanism of fat resorption. This hypothesis is supported by our investigations, performed on similar animals, when, after double sugar loading in progression of protein deficiency, the protein and ATP content as well as the activity of the ATPase and the alkaline phosphatase gradually decreased.

Zusammenfassung. Die Wirkung der Eiweissmangeldiät auf die Resorption von S^{35}-Methionin und J^{131}-Triolein wurde untersucht. Die Resorption von Methionin wurde nicht wesentlich beeinflusst, während die Fettresorption nach 24tägigem Eiweissmangel signifikant vermindert ist.

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[8] G. WISEMAN, J. Physiol. *120*, 63 (1953).
[9] Sz. DONHOFFER, Pflügers Arch. ges. Physiol. *246*, 92 (1942).
[10] F. VERZÁR and S. KUTHY, Biochem. Z. *210*, 265 (1929); *225*, 267 (1930); *230*, 451 (1931).
[11] A. C. FRAZER, Physiol. Rev. *20*, 561 (1940); *26*, 103 (1946).

Transcallosal, Extracallosal, and Geniculo-Cortical Responses during Physiological Sleep and Wakefulness

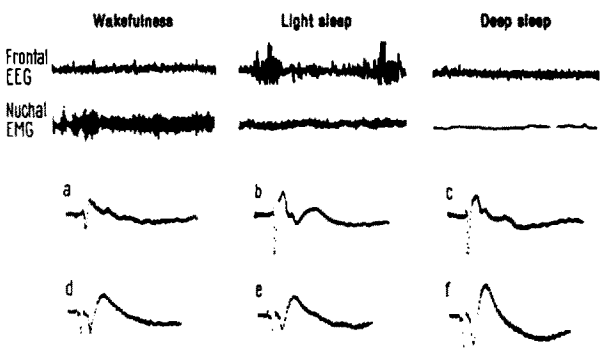
The excitability of neurons in the lateral geniculate body and visual cortex undergoes clear-cut variations during paradoxical sleep[1–4]. The aim of this investigation was the analysis of transcallosal (TCR), extracallosal (ECR) and visual cortex (VCR) responses and their interaction during the natural phases of sleep and wakefulness.

Experiments were made on cats, unrestrained, with chronic bipolar stimulating electrodes implanted in the lateral geniculate body of one side and in the white matter underneath the lateral gyrus of the opposite hemisphere. The responses were monopolarly recorded from the same cortical point in the lateral gyrus of the side where the lateral geniculate body was stimulated. A variable number of responses (50–300) was integrated in a CAT 400 Mnemotron and then photographed on an oscilloscope. The EEG (from frontal areas) and the EMG (from nuchal muscles), recorded with an inkwriter electroencephalograph, gave information on the degree of wakefulness and stage of sleep of the animal during the experiment.

Stimulation of white matter underneath the lateral gyrus, with single shocks (1/sec, 0.05–0.1 msec, 2–4 V) evoked in the homologous point of the opposite hemisphere a TCR with 2 msec latency followed by the ECR with the positive peak at 50 msec and the negative one at 90 msec from the stimulus (Figure b).

During *attentive wakefulness* the TCR fluctuated in amplitude and showed, when integrated, the lowest values observed. Usually its negative component was strongly reduced. The ECR was rarely present during wakefulness and when integrated was always very small in amplitude (Figure a).

In *light sleep* (with synchronized EEG), the TCR had the maximal amplitude. The ECR showed all its components. An integration of both responses revealed the highest values observed (Figure b).



Transcallosal, extracallosal and geniculo-cortical responses during wakefulness, light sleep and deep sleep. Unrestrained cat with chronic implanted electrodes. Each photogram represents an integration of one hundred responses. a, b, c: Responses of the lateral gyrus to stimulation of the homologous point of the white matter of the opposite hemisphere during attentive wakefulness (a), light sleep (b) and deep sleep (c). d, e, f: Responses of the same cortical point of the lateral gyrus to stimulation of the lateral geniculate body during the same episodes of wakefulness (d), light (e) and deep sleep(f). The sweep of photograms a, b and c lasts 125 msec and of d, e and f 31.25 msec.

[1] E. V. EVARTS, J. Neurophysiol. *25*, 812 (1962).
[2] J. MOURET, M. JEANNEROD, and M. JOUVET, J. Physiol. *55*, 305 (1963).
[3] E. FAVALE, C. LOEB, and M. MANFREDI, Arch. int. Physiol. *72*, 221 (1964).
[4] M. PALESTINI, M. PISANO, G. ROSADINI, and G. F. ROSSI, Exp. Neurol. *9*, 17 (1964).

During *deep sleep* (with desynchronized EEG and flattening of nuchal muscles), the TCR became quite stable while the EEG became desynchronized. The integrated value of the TCR was similar, or only slightly reduced, compared with the one seen during light sleep. The ECR was strongly reduced during paradoxical sleep and appeared very similar to the ECR observed during attentive wakefulness (Figure c).

The behaviour of visual cortex responses (VCR), evoked by geniculate stimulation (single shocks at 1/sec, 0.02 msec, 0.8–4 V), was quite different from that shown by TCR and ECR. During *attentive wakefulness*, the VCR markedly fluctuated in amplitude both in its presynaptic and postsynaptic components. An integration of a variable number of them showed an amplitude which was always higher than that seen in light sleep (Figure d).

During *synchronized sleep*, the VCR was very unstable; its integration revealed the lowest values observed (Figure e). As the animal went into an episode of *deep or paradoxical sleep*, the VCR became extremely stable. Its integration showed a dramatic increase in amplitude of both presynaptic and postsynaptic components up to 100% of the values seen during wakefulness and 130–150% of those observed in light sleep (Figure f).

The curve of interaction between transcallosal and geniculate stimulation (transcallosal stimulus preceding the geniculate one) showed a marked facilitation of VCR by callosal stimulation, which appeared always at an interval of 40–50 msec. This facilitation was present during both wakefulness and sleep, although it appeared maximal during deep sleep. During attentive waking and paradoxical sleep, facilitation was usually followed by a short inhibition at 70–90 msec stimulus interval, soon replaced by slight facilitation at an interval of 110–150 msec. During light sleep, on the other hand, the inhibition was much stronger and continued for several msec, reaching its highest value at an interval of 130–150 msec. Interaction was in no case present when geniculate stimulation preceded the transcallosal one. The results suggest that: (a) the excitability of cortical neurons activated by the transcallosal volley is higher during sleep (either slow or paradoxical) than during wakefulness. These results are slightly at variance with those reported by others[5]. (b) The ECR was very similar during both wakefulness and paradoxical sleep. Since the ECR has been shown to be due to activation of mesencephalic neurons[6], which are very active during wakefulness and deep sleep[7], the results obtained may be explained by reticular occlusion. (c) The transition from light to deep sleep was characterized by an augmentation of VCR indicating an increase of thalamic excitability as already reported by others[3,4]. (d) Interaction experiments indicate, moreover, that only during synchronized sleep can a long lasting inhibition of geniculo-cortical pathway by transcallosal volley occur. The inhibition appears to be very short during the most active wakefulness and the deepest stage of sleep.

Riassunto. La TCR è ampia nel sonno (lento e rapido) e ridotta nella veglia. La ECR è massima nel sonno lento e minima nella veglia e sonno profondo. La RCV, minima nel sonno leggero, raggiunge i valori massimi nel sonno desincronizzato. La curva d'interazione tra stimolo transcalloso e genicolato ha rivelato un processo inibitorio prolungato presente soltanto nel sonno leggero.

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[5] E. FAVALE, C. LOEB, and M. MANFREDI, Boll. Soc. ital. Biol. sper. *39*, 1513 (1963).
[6] L. T. RUTLEDGE and T. T. KENNEDY, Exp. Neurol. *4*, 470 (1961).
[7] P. R. HUTTENLOCKER, J. Neurophysiol. *24*, 451 (1961).

Excitability Changes during Paradoxical Sleep in the Rat

It was shown previously[1] that before the onset of paradoxical sleep (PP), a gradual decrease of the excitability of the reticular activating system (RAS) develops. Both direct reticular and external physiological stimulation become progressively less effective. Although the PP is characterized by decreased excitability in the RAS, it always ends with a sudden increase in reticular responsiveness, and, in the majority of sleep cycles, by a spontaneous behavioral (and EEG) arousal not precipitated by any new external stimulus. Thus it may be suggested that variations of activity in the RAS occur during the PP.

Bipolar electrodes were implanted in the frontal cortex, dorsal hippocampus, mesencephalic reticular formation and, in some cases, in the caudate nucleus and nonspecific thalamic region in 11 rats.

More than 100 sleep cycles were analyzed. It was found that after the beginning of the regular ϑ-activity (4–7/sec) in the hippocampus, which is a very typical EEG manifestation of the PP, the cortical EEG (Figure 1) usually exhibited well developed spindle activity (9–14/sec). The amount of spindling gradually decreased and almost disappeared by the end of the first minute of the PP (Figure 2). This spontaneous spindling was present also in cases where cortical spindles did not occur frequently in the slow wave sleep phase (SWSP). In these cases a gradual increase in the amount of cortical spindles was observed during the last minute of SWSP (Figure 2). However, when the cortical spindles were well developed during the whole SWSP it was difficult to determine whether the amount of spindling was increased before the end of SWSP. Often a few cortical spindles were observed during the last seconds of the PP before the behavioral arousal reaction (and hippocampal desynchronization in the EEG) following the PP. The frequency of the hippocampal ϑ-activity showed phasic ranges during the PP similar to those evoked in waking animals by changing the intensity

[1] E. ROLDÁN, T. WEISS, and E. FITKOVÁ, EEG clin. Neurophysiol. *15*, 775 (1963).