Fragments of the liver were fixed in isotonic formalin, neutralized with a buffer solution: then included in paraffin and stained with hematoxyline-eosine. Certain series were stained with Sudan III.

The most severe histological modifications were found in the liver of those animals which were first given carbon tetrachloride and only thereafter selenium (group I). The toxic modifications of the hepatic tissue consisted of the hydropic and lipid degeneration of the parenchymatous cells. The cells showing the most severe modifications were found in the peripheral areas of the lobules, the lipid substance appearing in the form of small fatty droplets. The confluence of these droplets gave rise to a large fatty drop, the cell becoming similar to an adipose cell. In the periportal spaces, and even in the interior of the lobules, we noted lymphocytic and plasmocytic infiltrations. Only a limited number of hepatic cells suffered necroses. In the animals of group II and III, the tissular picture of the liver was identical.

The lipid loading of hepatic cells proved to be slighter, manifesting itself only in the appearance of small droplets of fatty substance in the cytoplasm of a limited number of hepatic cells. It should be noted that the increase of binucleated cells amounted to 8–10%. Only small lymphocytic infiltrations were noted, particularly in the periportal spaces. The lesions were significantly slighter in the liver of animals having received selenium, either simultaneously with carbon tetrachloride (group II), or prior to its administration (group III). This fact allows us to conclude that selenium possesses a protective action towards the toxic effects exercised on the liver by carbon tetrachloride.

When, however, selenium is given later, it fails to stimulate in the same measure the degenerative processes of hepatic cells.

By observing the histopathological picture of the rat's liver, it appears that selenium administered prior to the initiation of intoxication accumulates in the organism and continues to exercise its protective effect also in the subsequent phases of carbon tetrachloride poisoning.

Résumé. Les auteurs ont étudié l'effet hépatoprotecteur du sélénite de sodium administré en doses de 1 γ/100 g du poids corporel, avant, pendant et après l'intoxication de rats avec du tétrachloride de carbone. Ils en ont conclu que, suivant un critère anatomo-pathologique, l'effet antitoxique du sélénite de sodium est de protéger le parenchime hépatique contre l'action du tétrachloride de carbone.

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Vestibular Influences on Vegetative Functions During the Rapid Eye Movement Periods of Desynchronized Sleep[1]

Transient vegetative changes parallel the appearance of the bursts of rapid eye movements (REM) during the desynchronized phase of sleep. At the time of the REM, phasic pupillary dilations appear[2,3] and two kinds of events affect the cardiovascular system: (i) a slight increase in heart rate and blood pressure, followed by (ii) a prominent slowing of the heart rate and fall in blood pressure[4]. These phasic vegetative changes seem to be an integral part of the constellation of events triggered by the mechanism which is also responsible for the episodes of REM. Previous experiments have shown that the medial and descending vestibular nuclei are responsible for the appearance of the REM[5]. The aim of the present investigation was to establish whether the vegetative changes characteristic of the desynchronized phase of sleep also depend upon the vestibular nuclei.

Methods. The experiments were performed on 10 unrestrained, unanaesthetized cats. The electroencephalogram, the electromyogram of the posterior cervical muscles, and the ocular movements were recorded through chronically implanted electrodes. The pupillary changes occurring during desynchronized sleep were observed in complete darkness with the aid of a sniperscope provided with an infrared source[2]. In order to prevent closure of the eyelids during sleep an apparatus made of plexiglass was applied to the cat before the experiment took place. The electrocardiogram was recorded by means of two electrodes placed subcutaneously on the limbs.

Results. The changes in pupillary diameter and heart rate that occur during desynchronized sleep, particularly those synchronous with the bursts of REM, were documented in 7 intact, unrestrained animals (Figure A, B).

Following a bilateral lesion of the vestibular nuclei in 3 cats, the bursts of REM characteristic of desynchronized sleep were abolished; only slow ocular movements and occasional isolated jerks of the eyes were noted. As in intact animals, the pupils constricted tonically in the periods of transition from wakefulness to drowsiness. The myosis gradually increased as synchronized sleep progressed. The pupils became fissurated when the cat reached the stage of desynchronized sleep. The most striking effect of this lesion was the abolition of the short-lasting and pronounced pupillary dilations which in the intact animal appeared in conjunction with the REM. During the episodes of desynchronized sleep, one only observed very slight variations in the tonus of the sphincter of the iris. At these times the transverse diameter of the pupil would vary from a width of 1 mm to complete fissuration. Even the phasic changes in heart rate, which occur simultaneously with the appearance of the outbursts of REM in the normal cat, were absent (Figure C, D).

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

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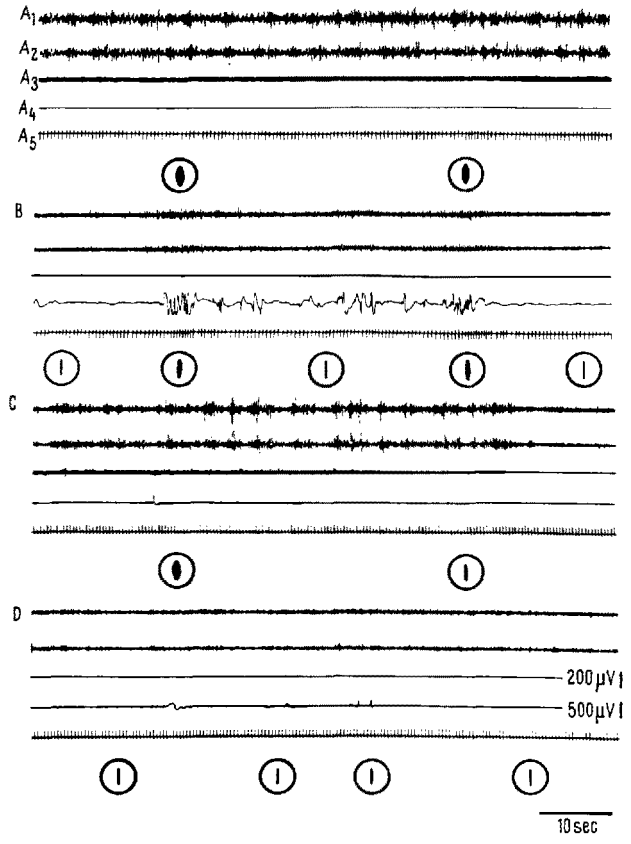
The critical region, whose destruction is responsible for the effects described above, includes the medial and descending vestibular nuclei. The lesion must be bilateral and affect completely these vestibular nuclei in their entire rostrocaudal extent.

The present experiments show that the medial and descending vestibular nuclei are critically involved in the phasic vegetative changes which appear simultaneously with the REM periods of desynchronized sleep. It has been suggested that the phasic mydriatic effect, which occurs during the bursts of REM, is due to phasic inhibition of the parasympathetic activity of the Edinger-Westphal nucleus[2] and that the transient acceleration of the heart rate occurring at the onset of the burst of ocular movements is possibly due to phasic inhibition of the cardioinhibitory centre[4]. These observations, coupled with the fact that the phasic vegetative effects of deep sleep are abolished by destroying the vestibular nuclei, support the concept that these nuclei are intimately concerned with brain stem mechanisms regulating vegetative functions.



Effects of vestibular lesions on vegetative changes characteristic of the desynchronized phase of sleep. Unrestrained, unanaesthetized cats. 1, left parieto-occipital; 2, right parieto-occipital; 3, posterior cervical muscles; 4, ocular movements; 5, electrocardiogram. The pupillary changes are indicated by the diagrams. A, B, intact animal showing regularity of the heart rate and of the pupillary diameter during synchronized sleep (A). During desynchronized sleep (B) the pupils become fissurated. Note the large pupillary dilation and the phasic increase followed by a reduction in heart rate synchronously with the bursts of REM. C, D, cat 3 days after bilateral lesion of the medial and descending vestibular nuclei. The tonic reduction in pupillary diameter during transition from synchronized to desynchronized sleep is still present (C). During the episode of desynchronized sleep (D), the large bursts of REM are completely abolished, as well as the related changes in pupillary diameter and heart rate. Note the regularity of the heart rate throughout the episode of deep sleep and the very slight decrease in tonus of the sphincter of the iris during occasional isolated jerks of the eyes.

Riassunto. Le modificazioni fasiche del diametro pupillare e della frequenza cardiaca sincrone coi REM caratteristici del sonno desincronizzato scompaiono dopo distruzione bilaterale dei nuclei vestibolari mediale e discendente.

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Radio-intervention sur animal vigile libre

Jusqu'à présent un certain nombre de travaux biologiques[1-4] ont utilisé la transmission radioélectrique, mais seulement pour recueillir des informations (radio-*réception*) à partir de potentiels d'action cardiaques[1] ou encéphaliques[2,3] ou encore pour connaître les allées et venues d'animaux sauvages comme le Blaireau dans son terrier[4]. La radio-*intervention* physiologique a été mise en œuvre dès 1959 pour procéder à la radiochirurgie simple sur animal immobilisé ou encore sur organe extirpé[5].

Or la physiologie sur animal arrêté par anesthésie ou autre moyen est une physiologie qui doit de toute nécessité être complétée par la physiologie sur animal libre, en état vigile, de façon à ce que les artefacts qui résultent des procédés d'immobilisation puissent être comparativement détectés, voire éliminés. Cette physiologie sur animal libre trouve un usage dans l'étude quantitative et objective de l'éthologie; grâce aux techniques radio-électriques, la psychophysiologie peut se développer en tant que cybernétique biologique précise. La présente Note indique comment le problème d'une radio-injection intramusculaire chez le Chien a été résolu.

L'*émetteur* mis en œuvre est de type «Handy» (caractéristiques: fréquence 27,12 MHz; modulation 1000 Hz; alimentation pile 9 V; consommation de courant 10 mA; antenne de 1 m de long; portée 150 m environ; poids total 50 g; dimensions 30 · 70 · 30 mm; équipé de 4 transistors).

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