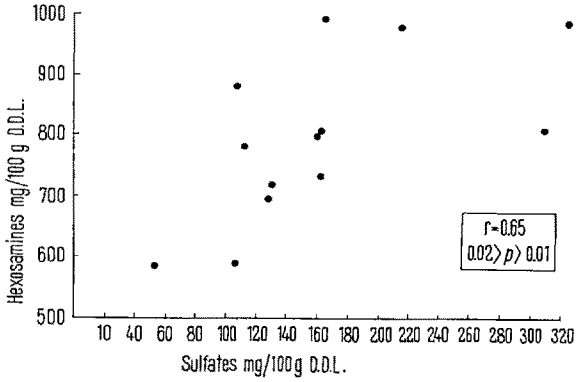
These results suggest that the accumulated mucopolysaccharide-like material is hexosamine-rich and sulphated, but devoid of uronic acids. SHETLAR et al.[8] have noted the relative lack of hexuronic acids in wound tissue from rabbits and dogs. The accumulation of sulphates in hepatic tissue following toxic injury is in accord with the findings of JUNGE HÜLSING and HAUSS[9] and PATRICK et al.[10], in which S^{35} incorporation by mucopolysaccharides was increased in rabbits and rats following liver injury. However, the former authors assumed an increased hepatic accumulation of chondroitin sulphates and did not specifically measure the hepatic uronic acid content.



Relationship between total hexosamine and protein-bound sulphate concentrations in fibrotic livers. (Dried defatted tissue.) r = correlation coefficient. p = significance level.

As our data were obtained on whole tissue, they do not allow conclusions regarding the intracellular location and chemical structure of the substance or substances. However, they strongly suggest that the sulphated hexosamine-rich material accumulated during liver fibrosis might possess a structure similar to the glycosaminoglycans described in different types of normal connective tissues[11].

The isolation and chemical determination of the substance is now under way.

Résumé. L'intoxication chronique au tétrachlorure de carbone produit dans le tissu hépatique du rat une forte augmentation des hexosamines et des sulfates liés sans modifier la teneur en acides uroniques. Ces modifications sont parallèles au processus de fibrilogénèse. Ces données indiquent l'accumulation pathologique d'un matériel de type glycosaminoglycan sulfaté.

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Unit Responses of the Lateral Geniculate Body to Light Flashes during Wakefulness and Synchronized Sleep

The existence of a reticular control upon the responsiveness of cells in the lateral geniculate body (LGB) is well established[1,2]. The enhancement during reticular[1,2] or natural[2] arousal of the geniculate responses evoked by electric shocks to the chiasm or optic nerve shows that the influence is facilitatory in nature. It is surprising, however, that the responses evoked by light flashes actually decrease during, or soon after, mesencephalic reticular stimulation[1]. The hypothesis[1] of an occlusion at the level of the geniculate, between reticular and retinal inputs, cannot easily account for this discrepancy.

This work is an attempt to re-examine the problem by a study of the evoked responses of single LGB units.

Experiments were performed on cats with the brain stem sectioned immediately rostral to the exit of the 5th nerve (midpontine pretrigeminal preparation[3]). This preparation characteristically shows EEG and behavioural patterns of wakefulness with some episodes of synchronized sleep. The pupils were paralysed with atropine. Spikes were recorded from the dorsal part of LGB with steel microelectrodes. In order to control the effect of eye movements during the waking state, some experiments were performed on curarized cats. The LGB cell firing rate was measured by a Beckman counter. A Sylvania 1160 glow tube was used to apply light flashes 300 msec in duration, the start of the flash being synchronized with the counter to facilitate detection of changes in rate. The response to the light pulse was calculated with respect to the average taken during 6 sec before the light pulse. EEG activity was continuously recorded by screws implanted in the skull.

The results can be summarized as follows: (1) During sleep the LGB cell activity occurs in bursts of high frequency (300–500/sec), while during wakefulness it shows random patterns. This confirms previous findings in the intact animal[4]. The average spontaneous frequency is also generally higher during wakefulness. A study of the standard deviation of the frequency in 1 sec compared to the average frequency shows a strong increase (up to 3–4 times) during sleep. (2) The firing rate of LGB units in

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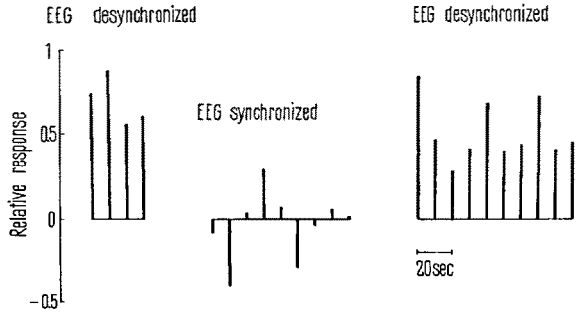
response to light pulses is always greater during wakefulness. (3) When the light pulse is decreased by means of neutral density filters, a stimulus evoking a clear-cut discharge during wakefulness will become ineffective during synchronized sleep. The Figure shows an example of this. It can be seen that during synchronized sleep any correlation between light stimulus and cell firing response is lost. (4) These effects are never seen when the responses of retinal cells are studied.



Flashes of light 300 msec in duration, of constant intensity, were applied every 10 sec. Relative rate response (vertical scale) is given by $(x - \bar{x}) / \bar{x}$, where x is cell firing at the time of light pulse (the time of measure is 1 sec), and $\bar{x}$ is average rate during 6 sec prior to pulse. The synchronization of the EEG occurred spontaneously, while the second state of desynchronization was provoked by olfactory stimulation.

Our results lead to the conclusion that LGB units are strongly facilitated or disinhibited during wakefulness. This effect is *tonic* in nature, since it is constantly observed independently of the transition from synchronized sleep to wakefulness. Hence it appears to be related to a state of wakefulness rather than a phasic phenomenon of arousal[5].

Riassunto. Registrazione microelettrodica eseguita in gatti pretrigeminali dalla parte dorsale del nucleo geniculato laterale (LGB). Si dimostra che le cellule del LGB hanno una risposta maggiore in frequenza a brevi lampi di luce durante la veglia anzichè durante il sonno sincronizzato.

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[5] This research was sponsored jointly by the Office of Scientific Research, EOAR, through the European Office, Aerospace Research, United States Air Force, under Grant AF EOAR 64-37.

Disparition de protéines urinaires spécifiques dans le syndrome néphrotique à la Puromycine[1]

Les protéinuries pathologiques chez l'homme ont fait l'objet de nombreux travaux de recherches[2-6]. Les auteurs ont tous remarqué la présence d'une quantité élevée de protéines sériques dans l'urine des néphrotiques. Des lésions causées au filtre rénal semblent être à l'origine de ce phénomène[2,3].

Quant aux protéines urinaires spécifiques, qui ne proviennent pas du sérum, leur présence n'a été rapportée que sporadiquement dans l'urine humaine normale[3,7,8]. La mucoprotéine de TAMM et HORSFALL[9] et les deux estérases urinaires migrant électrophorétiquement dans la zone des α- et γ-globulines sont apparemment d'origine rénale[3]. Dans le présent travail, nous voulions suivre, étape par étape, les changements dans la composition et la nature des protéines urinaires au cours du développement de la néphrose expérimentale provoquée chez le rat par des injections de Puromycine[10]. Notre attention a été spécialement attirée vers l'étude des modifications immunochimiques des protéines urinaires spécifiques dans ce syndrome néphrotique.

Matériel et méthodes. Des rats mâles de souche Sprague-Dawley, pesant environ 100 g ont été divisés en deux groupes: témoins et néphrotiques; les premiers ayant reçu quotidiennement une injection sous-cutanée de 0,3 ml de solution physiologique (NaCl à 0,9%), les derniers 0,3 ml de Puromycine (contenant le 6-diméthylaminopurine, 3-amino-*d*-ribose, un aminonucléoside) à 0,5% pendant 14 jours consécutifs[10]. Aux 7e et 14e jours depuis le début de l'expérience, la moitié des animaux de chaque groupe ont été placés dans des cages à métabolisme. Les urines de 24 h ont été recueillies et filtrées à travers le papier filtre Whatman no 1. Leur taux protéique a été ensuite mesuré au biuret[11]. Elles ont été alors dialysées contre de l'eau distillée pendant 24 h et lyophilisées. Les animaux ont été ensuite sacrifiés par décapitation; leurs reins ont été prélevés et soumis à des examens histologiques.

L'analyse immunoélectrophorétique de l'urine normale et des urines pathologiques a été effectuée avec la poudre lyophilisée, à l'aide de l'appareil LKB, suivant la méthode décrite par GRABAR et WILLIAMS[12]. Les protéines totales ont été colorées par le Rouge Ponceau S tandis

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