elicited drinking preferentially of tap water and that a plateau was reached at 30 min. Eating was not induced. The injections of isotonic saline alone were without effect on the ingestion either of tap water or of saline.

Effect of atropine on drinking induced by carbachol. Figure 1 B shows that the intraseptal injection of 150 µg of atropine sulfate inhibited the effect of 1 µg of carbachol injected through the same cannula 80 min afterwards.

Effect of atropine on drinking induced by fluid deprivation. In Figure 2 A) the compensatory intake of water and saline by rats after 18 h of fluid deprivation is demonstrated. Figure 2 B) depicts the blocking effect on this ingestion by 150 µg of atropine injected into the septal area 80 min before the measurements of the intakes.

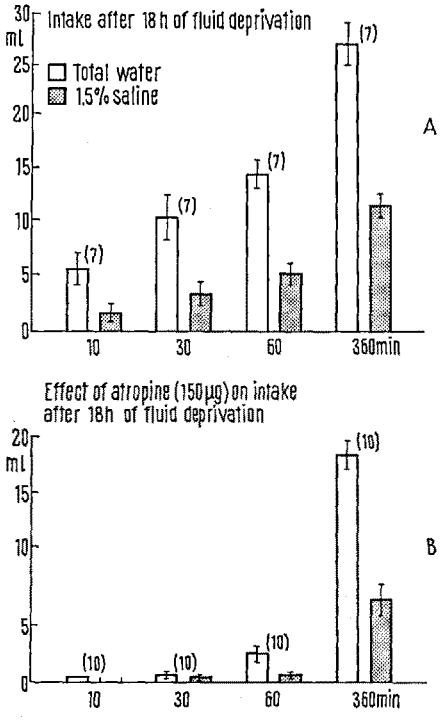

Fig. 2. A) Cumulative intake of total water and NaCl 1.5% in rats following 18 h of fluid deprivation. B) Inhibitory effect on this intake produced by 150 µg of atropine given 80 min before the measurements of the intakes. Same indications as Figure 1.

Discussion. Certain number of papers indicate that drinking mechanism involves neurons assembly which are sensitive to cholinergic stimulation (FISHER and COURY[4], COURY[5], GROSSMAN[6], CHIARAVIGLIO and TALEISNIK[7], ANTUNES-RODRIGUES and MCCANN[8]). In the present experiment, this event was strengthened regarding septal area. One interesting observation is based upon the fact that the augmented ingestion elicited by carbachol was preferential for tap water. It is known that electrolytic lesions of septal area in rats evoked an increase of NaCl solution and a diminution of tap water ingestion (NEGRO-VILAR et al.[9]). It appears that septal area normally restrains the intake of NaCl and stimulates the intake of water. This action could be made through the postulated circuit integrated by septal area, amygdala and hypothalamus.

Resumen. La inyección intraseptal de carbacol en ratas determinó la ingestión de agua. Este efecto, como asi tambien el provocado por 18 h de privación de liquidos, fué bloqueado por atropina. Estos resultados apoyan la hipótesis de que el mecanismo de la ingestión de liquidos en el área septal comprende neuronas colinérgicas.

J. ANTUNES-RODRIGUES and M. R. COVIAN

School of Medicine, Department of Physiology, Ribeirão Prêto (S.P., Brazil), 18 November 1970.

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Tooth-grinding During Sleep as an Arousal Reaction

On the basis of the responses to therapeutic procedures, several tentative interpretations[1] have been proposed on the generation mechanism of tooth-grinding during sleep. However, these hypotheses apply only to selected cases; most tooth-grinders are practically irresponsive to current therapeutic approaches. It is the aim of the present investigation to make some contribution to the elucidation of the central mechanism of tooth-grinding during sleep through studying it in the light of the physiology of sleep.

Thirteen all-night polygraphical recordings were performed on 8 male tooth-grinders of age between 19 and 41 who slept in a sound-attenuated chamber. 282 episodes of tooth-grinding were identified by hearing the sound through a microphone in front of the face of the subject and by recording the integrated potentials of the output of the microphone. 67% of the episodes were obtained on a background of lighter sleep stages, that is stages I and II assessed from the criteria of DEMENT and KLEITMAN[2]. This is comparable with the results of REDING et al.[3]. Long episodes of tooth-grinding were consistently followed by a considerable lightening of sleep stage or sometimes by awakening, while short episodes were usually not associated with any distinct shift of sleep stage. During paradoxical sleep 57 incidents of tooth-grinding were recorded. They were short in duration without exception and never observed during the bursts of rapid eye movements where arousal threshold is higher than during ocular-quiescent phase in the same sleep stage.

Trains of α-waves on EEG could be, in 49% of all incidents, recognized during intervals of characteristic rhythmic EMG activities of tooth-grinding which contaminated the simultaneously recorded EEG. K-complex[4], which is evoked on EEG by external and presumably also by internal arousing stimuli, preceded the

occurrence of tooth-grinding in 67 out of 105 cases obtained during sleep stage II where K-complexes make their appearance most obviously.

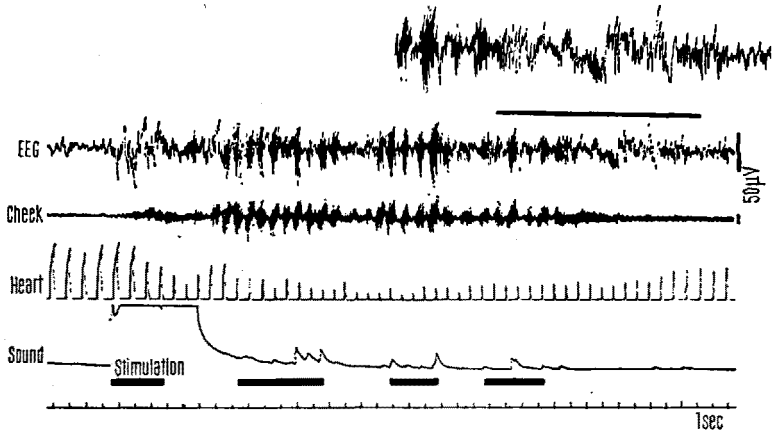
In all incidents, the heart rate never failed to be accelerated, and the finger-tip plethysmogram recorded photoelectrically indicated the sign of constriction of subcutaneous vessels during the tachycardia. The temporal relationship between the onset of tooth-grindings and that of the cardiovascular events was various; in some instances the former seemed to have prompted the latter, while in others the former followed the latter.

Electrodermal potentials of the forearm which can be evoked during sleep by arousing stimuli[5], were observed in 24 out of 44 episodes of tooth-grinding. Electrodermal potentials of the palm, which are outstanding during alertness and depressed in deeper sleep stages[6], were recorded in 5 out of 62 episodes.

suggested by the studies on the discharge pattern of single neurons[9]. On an occasion where the recovery of proper functioning of the motor center lags behind the arousal of the nigrostriatal system, tooth-grinding may be released.

BROUGHTON[10] has postulated that nocturnal enuresis, night-mare and somnambulism are manifested by the mechanism of 'carrying-over' of the state of slow-wave sleep into wakefulness. Although these disorders may appear to be based upon central mechanism similar to that of tooth-grinding, the occurrence of the latter is not restricted to slow-wave phase of sleep.

It has been reported that the administration of a therapeutic dose of dihydroxyphenylalanine, the precursor of dopamine, to a patient suffering from Parkinsonism has produced tooth-grinding[11]. This fact seems to be in favor of our assumption on the central mechanism of tooth-grinding.



Induction of tooth-grinding by a sonic stimulation applied to a subject in sleep stage II. From top, biparietal EEG on which a K-complex triggered by sonic stimulus is seen at the left and rhythmic EMG activities of tooth-grinding are superimposed, bilateral surface EMG of the cheek, heart rate showing tachycardia during the episode, and integrated potentials of the output of the microphone. Bars under the sound potentials indicate the sounds of tooth-grinding. The EEG under the line is enlarged at the upper right to show a train of α-waves.

The above results seem to point to a phenomenological feature common to all incidents of tooth-grinding during sleep, that is, it discloses itself as one of the peripheral manifestations of the central events occurring during transition from asleep to awake state. If tooth-grinding is not prescribed by the establishment of a particular state of sleep, but makes its appearance rather unspecifically as an arousal reaction, it must be able to be induced experimentally by arousing the sleeping subject.

Twelve episodes of tooth-grinding could be evoked by giving sonic stimuli of different strengths to subjects in different sleep stages. As shown in the Figure, sonic stimulation was immediately followed, in general, by a K-complex on EEG, and rhythmic muscular activities and sounds of tooth-grinding ensued with a latency of 11.6 ± 6.7 sec. The polygraphical pattern of induced tooth-grindings was indistinguishable from that of spontaneously occurring ones.

Recently it has been ingeniously worked out that dopamine-containing neurons in the nigrostriatal system of the brain stem are responsible for behavioral arousal, while noradrenaline-containing neurons in the midbrain reticular formation are important for the maintenance of the activated EEG pattern[7]. Tooth-grinding will manifest itself if, during transition from sleep to wakefulness, the dopamine-containing nigrostriatal system inflicts a particularly potent drive upon the brain areas directly concerned with the jaw movement of tooth-grinding, which have been localized in the rabbit[8]. On the other hand, upon arousal, the noradrenaline-containing midbrain reticular formation is considered to bring the disorganized functional state of the cerebral cortex during sleep to an organized one, as has been

Résumé. A cause du comportement des indices polygraphiques et surtout du fait de provocation par des stimuli d'éveil, on conclut que le grincement des dents pendant le sommeil se manifeste comme une sorte de réaction d'éveil.

T. SATOH and Y. HARADA

Department of Physiology, School of Dentistry, Aichi-Gakuin University, Nagoya 464 (Japan),
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