all experiments[7] as a result of electric stimulation of the thorax sympathetic chain (6 v, 20 imp/sec, 5 msec). The pattern of the neurogenic responses of resistance and capacitance vessels, recorded simultaneously in skin-muscle and splanchnic areas is illustrated in Figure 3.

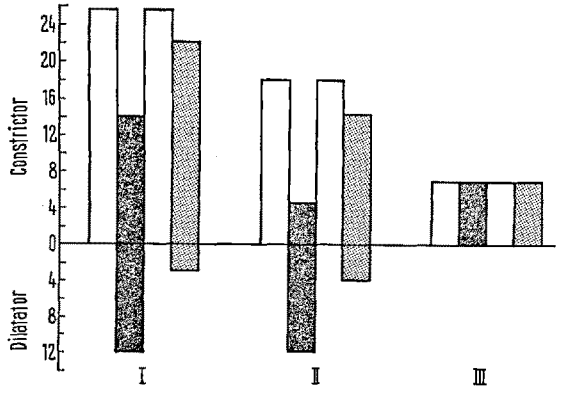

Fig. 3. Directivity of resistance and capacitance vessels responses in the skin-muscle and splanchnic regions under pressor synocarotid reflex (I), electric stimulation of the brachial nerve afferents (II), electric stimulation of the thorax sympathetic chain (III). Designations: on the ordinate, the quantity of experiments; white rectangles, responses of resistance vessels; black rectangles, responses of skin-muscle capacitance vessels; shade rectangles, responses of splanchnic capacitance vessels; above abscissa, constrictor response; below – dilatator response.

Conclusion. Electric stimulation of the thorax sympathetic chain resulted in a constriction of resistance and capacitance vessels in skin-muscle and splanchnic vascular zones. Simultaneous registration of resistance and capacitance vessel reflex responses in skin-muscle and splanchnic areas revealed resistance vessel responses to be always constrictor, while the capacitance ones might be either identical or different their direction being considered. Furthermore, the responses of capacitance vessels in the same vascular zone may differ directionally from the responses of resistance vessels.

Выводы. Электрическая стимуляция грудной симпатической цепочки вызывает констрикцию резистивных и емкостных сосудов в кожно-мышечной и спланхнической областях. Изучение рефлекторных реакций резистивных и емкостных сосудов одновременно в кожномышечной и спланхнической областях показало, что реакции резистивных сосудов всегда имеют однонаправленный констрикторный характер, тогда как реакции емкостных сосудов указанных областей могут быть как одинаковыми, так и различными по знаку. Кроме того, реакции емкостных сосудов одной и той же области могут отличаться от реакций резистивных по направленносси.

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Water and Sodium Chloride intake Following Microinjection of Carbachol into the Septal Area of the Rat Brain

Several studies have been published giving evidence that the activity of the septal area is mediated via cholinergic pathways, supporting the hypothesis that the septal area contains a cholinergic neuronal system (GROSSMANN[1], HAMILTON et al.[2], KELSEY[3]).

In the present study, this hypothesis was tested using the free water and sodium chloride intake as criteria of altered septal function. Adult male Wistar rats, of 200 to 300 g body weight, were kept in individual cages with a food cup filled with dry mixed diet and 2 graduated drinking bottles filled with 1.5% NaCl and unfiltered tap water respectively; daily readings were made of the intakes. After a control period of 2 weeks a stainless-steel cannula (O.D. 0.71 mm) was stereotaxically implanted into the septal area and after a further week injections were made through a dental stainless steel cannula (O.D. 0.31 mm) into the conscious and unrestrained rats. Carbachol (Carbamylcholine Chloride) and atropine sulfate were delivered by a 10-µl microsyringe in a standard volume of 2 µl of isotonic saline (0.15 *M*) by way of a polyethylene plastic tube connected to the inner cannula which was placed inside and advanced to the tip of the implanted cannula.

At the end of the experiment the rats were sacrificed and their brains were sectioned and stained. The end of the cannula was considered to represent the stimulation site. No particular localization was found and it was not intended to make any mapping, but a tendency was observed for more positive results with the cannula at an anterior placement.

Effect of carbachol. Immediately after the injection the rat was returned to its cage and the intake of fluids were measured after 10, 30, 60 and 360 min. Drinking started after a latency of between 3 to 5 min. Doses of 0.06, 0.125, 0.50, 1.0 and 2.0 µg were effective but routinely the dose of 1.0 µg was adopted. Figure 1 depicts the results obtained: it can be observed that carbachol

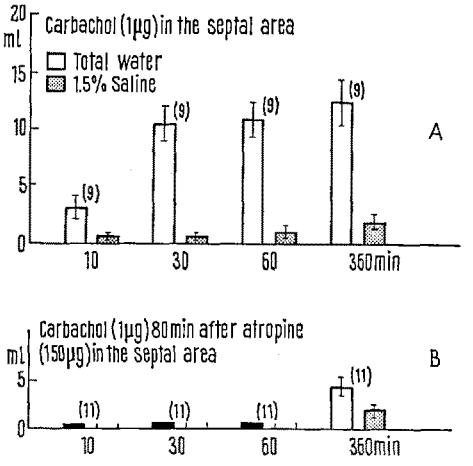

Fig. 1. A) Cumulative intake of total water and NaCl 1.5% following the injection of 1 µg of carbachol in the septal area. B) Inhibition produced by 150 µg of atropine upon the drinking effect of carbachol. The bars indicate the standard deviation of the mean; the number of rats are indicated between parentheses. Stereotaxic coordinates: F, 8.0; L, 0.3; H, +1.0. Total water: tap water plus water of saline solution.

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.

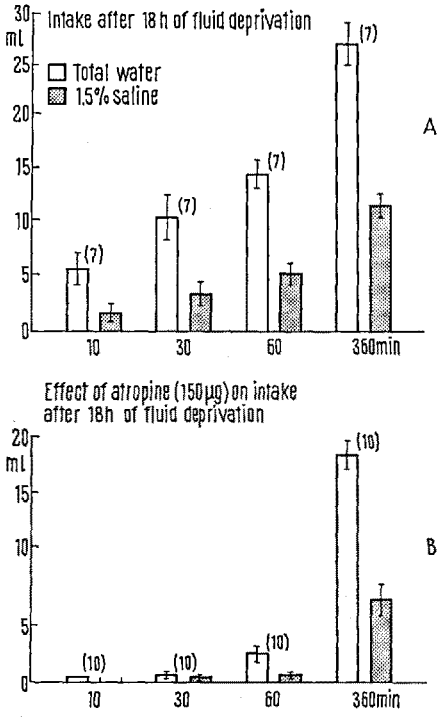

Fig. 2. A) Cumulative intake of total water and NaCl 1.5% in rats following 18 h of fluid deprivation. B) Inhibitory effect on this intake produced by 150 µg of atropine given 80 min before the measurements of the intakes. Same indications as Figure 1.

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.

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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