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Sleep: Physiology, Biochemistry, Psychology, Pharmacology, Clinical Implications.

Central and Peripheral Conditioned Responses
Induced by Intracerebral Conditioned Stimulus during Sleep

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It seems useful, for the understanding of dreaming from a physiological standpoint, to test during sleep the conditioned cerebral activity induced by an intracerebral conditioned stimulus (CS). In recent experiments [1, 2], we trained dogs to press a lever when an intracerebral CS was paired with food reward. The dogs were tested on the appearance of an EMG response in the lever-pressing forelimb (peripheral conditioned response [CR]); also, the change of the potentials evoked by the CS was measured before and after the occurrence of the EMG response. These evoked potentials (EP) at rest (control) seemed to be changed to the EP before the EMG by interference with the conditioned cerebral activity (central CR) which was responsible for discharging the forelimb EMG (fig. Ia). The motor potential (MP) was assessed by the following methods:
1. 20 or 25 EP in a series were sampled at each trial. The first 15 or 20 EP in this series had to include the last EP before the forelimb EMG. The control EP was determined by averaging the initial 5 EP in this series. Thus, the control EP was averaged when the animal was at rest. Using a computer, correlation coefficients (CC) between the control EP and each of the 20 or 25 EP were calculated, sampling at intervals of 4 msec. The CC curve of one trial is shown in figure 1a.

2. The arithmetic differences (fig. 1b) between the amplitude of the control EP (shown by dotted line in figure 1b) and each of the 20 or 25 EP (15 or 20 EP before and 5 EP after the EMG at a trial) are displayed on a map as contour lines (fig. 1c). The ordinate shows the order number of each EP, the abscissa shows time course of the amplitude difference for each EP.

As shown in figure 1a, the CC curve showed repetitive sharp decreases just before the EMG (in most cases three major decreases could be seen: they are indicated as a, b and c). These sharp decreases suggested the occurrence of different processes during the MP. Changes in the EP corresponding to these decreases of the CC were shown as elevations and depressions running parallel to the abscissa of the map (fig. 1c) (temporal pattern of the MP).

The MP maps of the sensorimotor cortex were rather simple, but those of the temporal and occipital cortices showed much more complicated patterns. To get the simple pattern of the MP map in subcortical areas, the site of CS must be selected. The lateral hypothalamus was the suitable site of the CS for this purpose. In this report, we shall show only the MP maps from sensorimotor cortex during awake and sleep states with the CS delivered to the nucleus ventralis posterolateralis of thalamus (VPL). When tested with intracerebral CS (VPL), the central CR (MP) was present in 100% during sleep, but the peripheral CR of EMG in the leverpress forelimb was observed in about 20% during slow sleep (especially spindle stage), and in about 70% during fast sleep. No lever-pressing mechanogram was obtained during sleep. The percentage appearance of the peripheral CR during sleep in dogs was very similar to the percentage recall of dreams during sleep in humans, suggesting that the peripheral CR may be a feedback for short-term memory, which may be interesting relative to the recall of dreams.

In this report, the MP induced by the intracerebral CS applied during sleep is compared with the MP observed in the state of internal inhibition during wakefulness, to disclose differences between the inhibitory mechanism
Fig. 1. Map of ‘Motor potential’ in sensorimotor cortex. CS: VPL 7.5 c/sec. A: Correlation coefficient curve. Marks a, b, and c show time points of decrease in the curve before EMG response. B: Three evoked potentials (stimulus No. 7, 13, and 15), which are selected from 15 EPs before the EMG. Dotted line is the control EP. C: Differences between amplitudes of the control EP and of the EPs No. 1 to No. 20, displayed with interpolated contour lines.

Fig. 2. Maps of ‘MP’ in sensorimotor cortex during internal inhibition. Positive CS: VPL, 7.5 c/sec; Negative CS: Hippocampus, 100 c/sec. A: Control; lever-pressing (+), EMG (+). B: ‘Inhibition-1’ type; positive CS was preceded by negative CS; lever-pressing (—), EMG (+). C: ‘Inhibition-2’ type; positive CS was preceded by negative CS; lever-pressing (—), EMG (—). D. ‘Inhibition-3’ type; experimental extinction of positive CS; lever-pressing (—), EMG (—). In the case of EMG (—), decreases in the correlation coefficient curve are marked on the ordinate as (a), (b), and (c).

of sleep and that of the internal inhibition of conditioning. Figure 2 shows four MP maps obtained from the sensorimotor cortex in the awake state. Map A is the same as figure lc and shows the control MP generally observed during positive CS (VPL, 7.5 cps). Maps B, C and D were recorded in different states of internal inhibition. Map B shows the MP when the animal lifted the forelimb (EMG positive) but did not press the lever (lever-pressing negative) during the positive CS when it was preceded by the negative CS which was 100 cps electrical pulses to the hippocampus. Map C shows the MP when the positive CS was preceded by the same negative CS, but when neither lifting the forelimb (EMG negative) nor pressing the lever was observed. Map D shows the MP during experimental extinction; neither EMG nor mechanogram of the lever-press was recorded. The main differences between control map A and the other three inhibited MP maps obtained from the same area consisted in an increase of amplitude of the late component of the EP from 30 to 100 msec just before the EMG. In control map A, an increase of amplitude of the late component appeared at about 80 msec in the EP, while in map B it was found about 20 msec earlier. In map C, another increase of amplitude of the late component, which appeared in control map A between 30
and 60 msec in the EP and disappeared several hundreds of milliseconds before the EMG, was continuously present in every EP and was not extinguished when the EMG was not produced in the lever-press forelimb.

The former increase of late component with the time-lag of about 80 msec was scarcely observed. Therefore, a characteristic pattern running parallel to the ordinate was visualized. In map D, the MP pattern was much changed: no remarkable pattern parallel to the ordinate was seen. Those three inhibited patterns of MP of sensorimotor cortex represent examples of ‘inhibition 1’, ‘inhibition 2’ and ‘inhibition 3’, respectively. The strongest inhibition is ‘inhibition 3’: during experimental extinction, the animal did not press the lever but lifted the forelimb in the ‘inhibition 2’ state of MP, while the animal did not even lift the forelimb in ‘inhibition 3’.

Though there were remarkable differences between the patterns of the MP maps, no characteristic differences were found in the temporal pattern of the CC curves. Examination of the MP maps from the sensorimotor cortex suggests that two inhibitory mechanisms exist in the brain: one is mainly related to the changes in late component of the EP (from 30 to 100 msec) induced by the VPL stimulation, and the other (which is stronger) is related to the disappearance of the spatial pattern of the MP.

Figure 3 shows four MP maps obtained from the sensorimotor cortex during sleep of the same animal. Figure 3a and 3b were obtained during slow sleep and figure 3c and d during fast sleep. EMG was detected in the lever-press forelimb in a and c, but not in b and d. Figure 3a is rather similar to the MP map of ‘inhibition 1’ in awake state (in fig. 2) with the appearance of the late component increase in the EP just before the EMG, while figure 3b is the pattern of ‘inhibition 2’ (in fig. 2). MP maps during fast sleep (fig. 3c and d) are more complicated than those during slow sleep: most elevations and depressions run parallel or diagonally to the abscissa, and those parallel to the ordinate are few or very short. They are rather similar to ‘inhibition 3’ (in fig. 2d).

It is difficult to predict correctly whether the forelimb EMG would
appear or not, only from examining the pattern in the MP map of sensorimotor cortex, especially during fast sleep. In the sleeping animal, data on subcortical MP may be helpful for correct prediction, because differences exist between MP maps with positive and negative EMG. However, these data are omitted from this report.

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Changes in Sleep and Waking in the Rat after Lesion of Ascending Noradrenaline Pathways by 6-Hydroxydopamine

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After the description of monoamine-containing nerve cells in the lower brain stem [4, 5] and subsequent delineation of ascending axons to the forebrain [15, 18] it has become possible to perform selective lesions