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10 November 1969

Facilitation of Spindle-Burst Sleep by Conditioning of Electroencephalographic Activity While Awake

Abstract. *A slow-wave electroencephalographic rhythm recorded from the sensorimotor cortex of the waking cat has been correlated behaviorally with the suppression of movement. Facilitation of this rhythm through conditioning selectively enhances a similar pattern recorded during sleep, the familiar spindle burst. The training also produced longer epochs of undisturbed sleep. The specific neural mechanism manipulated during wakefulness appears to function also in sleep and to be involved with the regulation of phasic motor behavior.*

Localized electroencephalographic (EEG) recordings from sensorimotor cortex in the cat show a very distinctive slow-wave pattern (12 to 14 count/sec), which occurs in brief trains periodically during the waking state. This activity has been termed the sensorimotor rhythm (SMR) (*1*). It is observed in the quiet, alert animal and has been related specifically to the suppression of previously trained motor responses (*1*, *2*). When the SMR in a naive hungry cat is reinforced with food, a conditioned EEG response associated with stereotyped motionless postures develops (*3*). Such training makes it possible to increase significantly both the occurrence of the SMR and the related suppression of movement.

The waking SMR is similar to EEG spindle-burst activity recorded during slow-wave or quiet sleep (*4*) both with respect to frequency-power spectrum and to cortical topography (*1*, *5*). This similarity (Fig. 1) suggested that the two phenomena are related. The objective of the present study was to test this hypothesis. We attempted to determine whether enhancement of the SMR in the waking animal, achieved through the use of EEG conditioning procedures, causes changes in the pattern of quiet sleep. Quantitative effects were sought in the amount of spindle-burst activity and in the duration of sustained quiet sleep epochs.

Eight adult cats were prepared surgically for localized EEG recording. The frontal sinus was opened bilaterally, and pairs of jeweler's screws were threaded into the frontal bone over lateral pericruciate cortex on both sides (Fig. 1). Leads from these, a pair of posterior cortical electrodes, and standard eye and neck muscle electrodes were attached to a connector which was fixed to the skull with dental cement. Later the animals were placed in a recording chamber, which was equipped with an automatic feeding device, and connected through a counterweighted cable system and slip-ring assembly to an electroencephalograph. After adaptation to this chamber, three independent records of sleep were obtained as controls in advance of experimental training. These consisted of continuous recordings of EEG, electrooculograph, and data from neck muscles through several sleep cycles. With localized EEG recordings in the cat, quiet sleep is easily identified by the simultaneous occurrence of recurrent spindle bursts from sensorimotor cortex and slow waves from posterior cortex (Fig. 1). This pattern is punctuated frequently by motor adjustments or spontaneous shifts in the EEG back to the patterns of the waking or drowsy states. These interruptions are usually brief and give way again to a sustained pattern of quiet sleep. The result is a series of quiet-sleep epochs which are terminated eventually by a period of active sleep. This sequence defines the sleep cycle in the cat and at least two such cycles were obtained from each animal during the three tests before training. Subsequently, the animals were assigned randomly to two groups of four each. Both groups were then trained to receive food by producing specific patterns of EEG activity from sensorimotor cortex. Group 1 was reinforced during daily sessions for producing SMR activity, while group 2 was reinforced for producing low voltage, fast (LVF) activity. A training session consisted of 60 such reinforcements. Most animals reached maximum performance after 2 to 4 weeks of daily training. On the final or test session, they were allowed to obtain unlimited reinforcement and remained in the chamber until several complete sleep cycles were registered. Training was then reversed for the two groups and a second sleep recording was eventually collected in a similar manner. A final sleep recording was obtained again 1 month after the termination of all training.

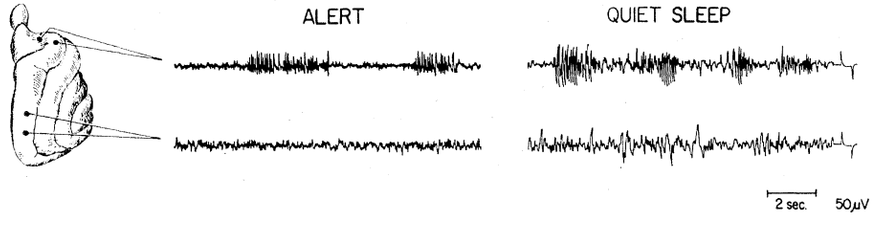

Fig. 1. Localized EEG recordings obtained from the cat during alert wakefulness and quiet sleep. Conditioned sensorimotor rhythm activity, shown as recorded in the waking animal, can be compared with the spindle-burst pattern which develops over this same cortical area during quiet sleep.

Instrumental EEG responses were established by previously described

techniques (*3*). Briefly, EEG signals from sensorimotor cortex were fed through a frequency filter, set at 12 to 14 count/sec, into a calibrated attenuation circuit and then to an integrator circuit. These units specified that a signal containing at least one-half second of the 12 to 14 count/sec SMR frequency, at a voltage 100 percent above background level, activated a relay. The closing and opening of this relay was displayed on the polygraph tracing. In the case of SMR conditioning, the relay activated the feeding device and thereby provided automatic reinforcement. For LVF conditioning, the closing of this relay precluded activation of the feeding device, which was operated manually by the experimenter during LVF activity. The apparatus for detecting 12 to 14 count/sec activity was operative during the collection of all sleep data and provided a quantitative measure of sleep spindle-burst activity. Analysis of sleep *duration* involved measurement of independent epochs of quiet sleep lasting no less than 20 seconds.

Instrumental conditioning of SMR activity in the waking cat produced statistically significant changes in both of the parameters of sleep measured here. A reliable facilitation of spindle-burst activity occurred specifically in all sleep samples obtained after SMR conditioning (Fig. 2). This facilitation was still apparent 1 month after the termination of all training for animals who received the LVF-SMR sequence (group 2), but was not sustained with the reversed training sequence, where intervening LVF conditioning was given (group 1). The increase in the percentage of spindle-burst activity after SMR conditioning resulted from differences in both the number and duration of spindle bursts. In addition to this modification of the EEG, the mean duration of quiet-sleep epochs was also significantly increased immediately after SMR conditioning, but this effect was not sustained in either group. The change reflected a decrease in the number of motor adjustments and spontaneous pattern shifts which led to fewer brief and more protracted periods of quiet sleep (Fig. 3). Statistical evaluation indicated that these alterations in the duration of sleep were reliable. No systematic differences were found between the SMR and LVF conditioning test sessions in number of instrumental EEG responses or latencies of sleep onset.

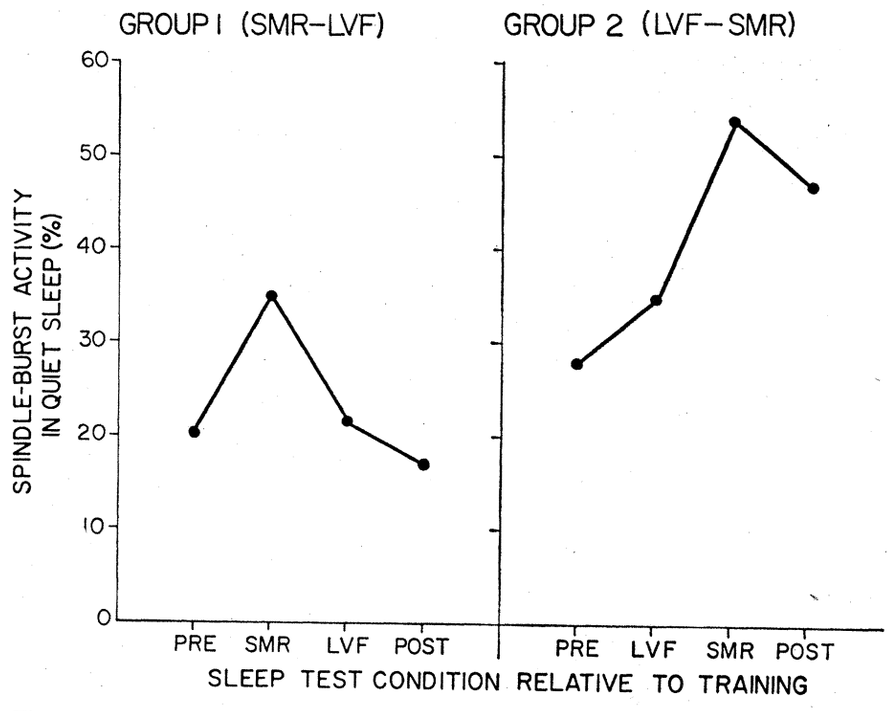

Fig. 2. Comparisons of the percentage of spindle-burst activity in the EEG of quiet sleep before (*Pre*), during (*SMR, LVF*), and 1 month after (*Post*) instrumental EEG conditioning in the waking cat. Mean data for the two test sequences are shown separately, since a significant order effect was noted. Student *t*-tests for correlated means showed a selective facilitation ($P < .01$) of spindle-burst activity during sleep following SMR training for group 1 ($t = 4.55$) and group 2 ($t = 8.61$). This facilitation was sustained 1 month after training in group 2 only ($t = 5.69$).

Sensorimotor EEG activity identical to the SMR was observed periodically during active sleep. No reliable changes in the amount of this activity were found in the limited samples of active sleep collected in this study. Invariably, however, this pattern occurred during those portions of active sleep devoid of any of its characteristic phasic phenomena. Thus, phasic motor behavior, including rapid eye movements, was never observed in the presence of 12 to 14 count/sec activity from sensorimotor cortex, either during wakefulness or during both quiet and active sleep.

The fact that behavioral manipula-

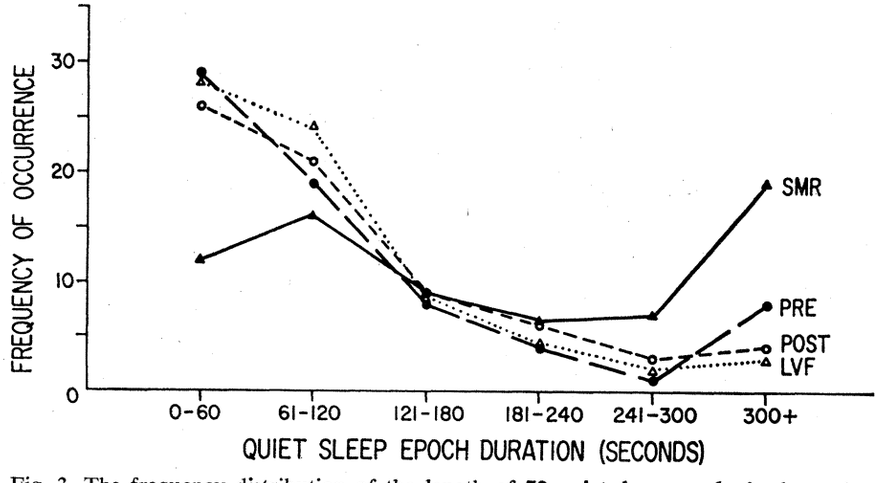

Fig. 3. The frequency distribution of the length of 72 quiet-sleep epochs is shown here for each of the conditions of sleep tested. This represents data from nine sustained periods of quiet sleep in each animal tested, measured backward from the second or third period of active sleep. Following SMR training only, sleep epochs were significantly prolonged ($t = 4.20$, $P < .01$).

tions which facilitated SMR activity in the waking animal led to a selective and sustained increase in spindle-burst activity during quiet sleep suggests a common neural mechanism for these two EEG phenomena, with functional continuity across wakefulness and sleep. This suggestion is supported further by the observation that phasic motor behavior, which is specifically suppressed in relation to conditioned SMR activity in the waking state, is significantly reduced, also, in sleep following SMR conditioning. It is possible, therefore, that some aspects of the physiology of sleep are determined by the nature of waking experience, through the modification of common neural mechanisms by environmental contingencies. This conclusion is consistent with our previous observation that different behavioral conditions before sleep, such as fatigue from prolonged work and frustration from the extinction of a previously rewarded response, can produce significant differences in the configuration of the EEG during subsequent sleep periods (*5*). Thus, the immediate factors preceding a particular episode of sleep as well as the general history of the animal are important determinants of the physiological characteristics of that sleep. In fact, the complex EEG configurations during sleep of higher mammals may result from the operation of a number of functionally specific neural mechanisms. The effective combination of these mechanisms could be responsible for the particular physiological pattern of sleep at any given moment.

As suggested previously, the neural mechanism common to the SMR and the sleep spindle burst is concerned with the suppression of movement, as indicated by the absence of phasic motor behavior when these rhythms are present in wakefulness and in sleep. Electrocortical rhythms, such as the spindle burst, are thought to result from the summation of synaptic prepotentials gated by thalamocortical feedback networks (*6*). There is evidence that removal of sensory input to specific thalamic nuclei can abolish thalamocortical discharge along specific afferent pathways, and set these networks free to generate slow-wave activity in appropriate cortical projection areas (*7*). We have noted SMR-like electrical activity in the ventral posterolateral and ventral lateral nuclei of the thalamus which, although lacking a direct correspondence to the cortical rhythm, was clearly associated with it (*8*). Thus, afferent discharge, possibly proprioceptive, may be involved in the generation of this rhythm. Hongo *et al.* (*9*) have found that spontaneous and electrically induced spindle bursts in the cortex of cats were directly correlated with decreases in the rate of discharge from muscle spindle afferents in both flexor and extensor muscles. However, they concluded that the decreased gamma motor activity and the corresponding sensorimotor spindle bursts were both the result of a common central regulatory mechanism. We agree with this interpretation, for it is clear that these two phenomena bear no causal relationship to one another. Hongo *et al.* found that complete ablation of the sensorimotor cortex did not interfere with the depression of gamma motor activity elicited by caudate or thalamic stimulation at parameters which induced cortical spindles in the intact cat. Conversely, the sensorimotor rhythm is neither abolished nor increased in paralyzed cats (*10*).

This central neural mechanism, which is responsible both for the development of sensorimotor cortex spindle activity and for the suppression of phasic motor behavior, involves, most likely, structures of the extrapyramidal motor system. We have found preliminary evidence of an important contribution from the cerebellum. The caudate nucleus has been implicated also. In addition to the extensive work in this area by Buchwald and Hull (*11*), Liles and Davis (*12*) recently reported suppression of cortically induced movements with stimulation of the rostral and medial portions of the head of the caudate nucleus. Stimulation of these same points elicited slow-wave EEG activity over sensorimotor cortex.

Our findings appear to lend new significance to the EEG as an index of physiological activity. With certain configurations, such as 12 to 14 count/sec high voltage activity over sensorimotor cortex, the operation of specific neural mechanisms can be inferred. These findings provide, also, for a more comprehensive approach to the sleep process, which undoubtedly has many of its roots in waking behavior.

MAURICE B. STERMAN
RICHARD C. HOWE
LORRAINE R. MACDONALD
Veterans Administration Hospital, Sepulveda, California 91343, and *Departments of Anatomy* and *Physiology, School of Medicine, University of California, Los Angeles 90024*

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