of intensity from maximum to complete darkness. This is shown by the figures in Table 3; 90.2 percent correct responses were recorded in five daily sessions involving 266 trials. One would not expect such a performance if the squirrel saw the blue port as gray. With performances such as these the more interesting question almost becomes: what is the cause of the incorrect responses?

We believe that the odor of the seeds is ruled out as an explanation, for the reward was always placed symmetrically with respect to the two ports. Unconscious clues by the human attendant seem to be ruled out by virtue of the fact that both of us have tested the male squirrel and have obtained the same results. Noises during the trials were completely random or else were symmetrical to the two ports. The female squirrel was as successful as the male in selecting the blue port. Moreover, attempts to train other animals to select green or orange ports have failed to elicit correct responses. The behavior of these animals toward green or orange light was random except possibly in relation to the blue port.

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Sleep: Changes in Threshold to Electroconvulsive Shock in Rats after Deprivation of "Paradoxical" Phase

Abstract. *Rats were deprived of paradoxical (radid eye movement or REM) sleep for a 6-day period but were allowed substantial non-REM (slow wave) sleep. Thresholds for electroconvulsive shock dropped significantly after deprivation in all these animals, but thresholds in control animals treated in a similar manner, but allowed REM sleep exhibited no change. Deprivation seems to heighten neural excitability.*

While REM sleep invariably occupies a substantial portion of the total sleep time in mammals, and has been closely associated with dreaming in man (*1*), little is known of its physiological function (*2*).

After a cat has been deprived of such sleep for 30 days or more, its first periods of REM sleep are dramatically enhanced by overt manifestations. The animal becomes completely flaccid, but against the background of muscular atonia there are episodes of violent facial and limb twitches interspersed with convulsive movements of the entire body so intense that the animal seems to be in the throes of a myoclonic seizure. The severity of these spasms appears to increase as the animal is increasingly deprived. During the recovery period, when the animal is allowed uninterrupted sleep, there is a rebound or compensation for the lost sleep that is roughly proportional to the length of period of deprivation (*2–4*), and the intensity of the phasic motor activity gradually subsides. In addition, Dewson *et al.* found accelerated auditory recovery in cats deprived of REM sleep for 5 or more days (*5*). The ratio betwen amplitudes of potentials evoked by a pair of clicks was greater in the nondeprived but equally disturbed animals than it was during a prior or subsequent period of deprivation. Hence, at a given time (25 to 75 msec) after neural discharge, more cells seem to be available for a second response in the deprived animal than are available in its nondeprived counterpart. This change was invariably reversed when the animals were allowed to make up the lost sleep.

These findings led us to hypothesize that an effect of the selective deprivation of REM sleep is a generalized increase in neural excitability. We have now tested this hypothesis in a study in which the threshold for electroconvulsive shock was used as a gross measure of change in excitability. The rat was chosen as the experimental animal because it displays the physiological features of REM sleep (*6*), (see Fig. 1), and there is an extensive literature on factors affecting electroconvulsive thresholds in this species (*7–11*).

Base thresholds for electric shock were determined for a group of 48 male Wistar rats, ranging in weight from 150 to 175 g. Thresholds were established with a constant-current stimulator. On-line current measurements were made with a Tektronix oscilloscope and a current-measuring probe. To ensure reliability, three determinations were made, by the method of Schwartzbaum *et al.* (*7*), for each rat; these did not vary by more than 2 percent of their original values.

Twenty-four of these rats (group 1) were then placed on inverted flower pots whose bottom diameter was 7 cm. The pots were placed in large tubs filled with enough water to just cover the pots' bottoms. Wire mesh was attached above the tubs and shaped to hold food and water bottles. The rats were able to rest on the pots and were even able to get non-REM sleep, but at the onset of REM sleep, with its ensuing muscular relaxation, they would either fall into the water and clamber back to their pots or would get their noses wet enough to awaken them. Twenty-four control animals (group 2), matched as closely as possible with those in group 1 for age, weight, and shock threshold, were placed in similar tanks. However, the bottoms of the flower pots in these tanks were wide enough (11.5 cm) to allow the animals to curl up and obtain REM sleep without falling into the water. The method is an adaptation of the method used successfully to deprive cats of REM sleep (*2, 3*).

Four animals in a third group, weighing 210 to 220 g each, were treated in the following manner in order to control nonspecific effects of sleep loss produced by the deprivation technique. These rats were placed in tanks identical with those used for the first group. However, they were removed from the tanks for a 6-hour period each day and allowed to sleep. Electromyographic and electroencephalographic potentials were recorded. Two animals were awakened at the first sign of loss of EMG potential and EEG activation; the remaining pair served as controls and were awakened from non-REM (slow wave) sleep with the same frequency as the deprived pair. Thus the control animals lost an amount of non-REM sleep comparable to the amount of sleep lost by

the deprived group and were subjected to the same environmental stresses as the deprived group, with the exception of REM deprivation itself. The animals were treated in this manner for 6 days; at the end of this time, thresholds were again determined.

Base thresholds to electroconvulsive shock for the rats in group 1, prior to deprivation, ranged from 21 to 40 ma (mean, 26.2 ma). After 6 days of deprivation, the thresholds ranged from 17.2 to 27 ma (mean, 20.2 ma); the drop in the mean was 6 ma, or 23 percent of baseline. In every case, each individual animal's threshold was lowered by at least 5 percent of the value it had before deprivation. Values were often lowered by 20 to 30 percent. Statistical tests (t tests) for differences between correlated means were applied to pre- and post-deprivation values; The differences were found to be highly significant ($t = 5.0$, $P < .001$).

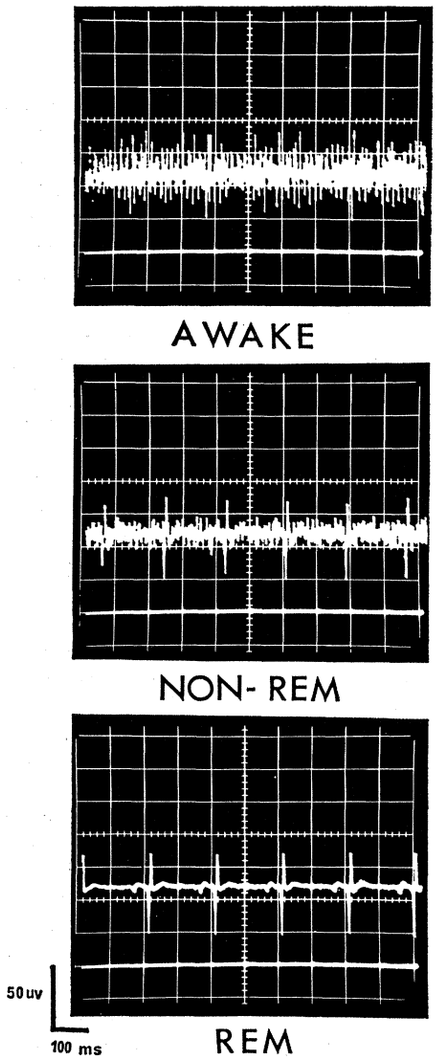

Fig. 1. Electromyographic recordings (EMG) from neck muscles of a REM-deprived rat. Tracings are typical of those obtained from rats during wakefulness, slow-wave sleep, and REM sleep. Note the complete loss of EMG potential in REM sleep; only a heartbeat artifact remains.

Table 1. Mean shock thresholds 24 REM-deprived rats and 24 nondeprived controls, before and after 6 days in deprivation tanks. Controls were allowed REM sleep in tanks, while deprived rats were not. Ranges are shown in parentheses.

Group	Threshold (ma)	
	Baseline	Experimental
Deprived	26.2 (21–40)	20.2 (17–27)
Control	24.0 (22–27)	26.1 (23–28)

Base thresholds for the control group ranged between 22 and 27 ma (mean, 24.0 ma). After 6 days in the tank (with big flower pots), the thresholds ranged from 23 to 28 ma, (mean, 26.1); the rise of the mean was 2.1 ma. These data are summarized in Table 1. While animals in group 1 did not show any significant loss in weight, those in the control group showed a mean gain of 10 g.

When animals were deprived by both deprivation tanks and hand awakenings (group 3), and controls were kept awake on platforms of the same size as those provided for the deprived rats but were allowed REM sleep, the results were essentially the same as those obtained from groups 1 and 2. One of the deprived rats showed a drop of 26 percent in shock threshold, while the second deprived rat showed a drop of 18 percent in threshold; both changes were statistically significant ($t = 3.7$; $P < .05$). One of the animals which served as a control showed no change in threshold, while the other showed a 4-percent drop. Each of the four rats lost 18 ($\pm$ 1) g of body weight over the 6-day period. After post-deprivation thresholds had been determined, rats were allowed to sleep without interruption.

Shock thresholds for all deprived animals were determined during recovery. They remained low for the first 2 days and began to rise on the 3rd day. By recovery day 5, the thresholds of 23 of the 26 deprived rats had returned to the base levels.

The results demonstrate a drop in threshold to electroconvulsive shock during REM sleep deprivation which is reversed when the animals are allowed to recover their lost REM sleep time. These changes cannot be attributed to weight loss (*8*) because the animals in group 1, which showed large drops in threshold, did not lose any weight at all. In group 3, all of the animals lost the same amount of weight, yet only those awakened from REM sleep showed a significant drop in threshold.

The exact physiological reasons for the drop in threshold remain unspecified. Many factors, among them food intake (*8, 9*), metabolic acidosis (*8*), plasma electrolytes (*10*), adrenocortical steroids, and adrenocorticotrophic hormone (*11*), influence shock threshold.

Since it is not yet known whether any of these physiological factors can be influenced by REM sleep loss, the possibility remains that REM deprivation affects an intermediary factor which leads to a lowering of to electroconvulsive shock. However, our working hypothesis is that REM deprivation directly produces certain neurochemical changes and that the cumulative effects of these changes may be responsible for the neurophysiological and behavioral changes observed in animals selectively deprived of REM sleep.

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