Recovery from prolonged barbiturate sleep in the rat[1]

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Prolonged sleep was produced by periodic injections of pentobarbital. This sleep consisted predominantly of slow waves and spindles. No paradoxical sleep waves were observable in the cortex during the drug treatment. Following discontinuation of the drug, the amount of slow wave sleep decreased and paradoxical sleep increased when compared to a control period, suggesting a partial satiation of slow wave sleep and deprivation of paradoxical sleep by the treatment.

This experiment is concerned with the need for sleep; in particular with the susceptibility of sleep to satiation, with special reference to the two phases of sleep. Two phases of sleep thought to be qualitatively different have been delineated both in the human (Dement, 1965) and the rat (Swisher, 1962). The two phases in the rat are slow wave sleep (SWS) consisting of 1-3 c/sec. activity with spindling, and paradoxical sleep (PS) consisting of 6-8 c/sec. activity (Swisher, 1962). The paradoxical phase is thought to be analogous to Rapid Eye Movement (REM, dream) sleep in humans (Dement & Kleitman, 1957).

The purpose of this experiment was to determine the effect of a 24 hr. period of pentobarbital induced sleep on subsequent sleep.

Method

Bipolar cortical electrodes (right frontal to occipital) were surgically implanted on the dura of four adult male Long-Evans rats. One week elapsed between surgery and beginning the experiment. EEG was recorded with a Grass III-D unit. The Ss were placed in individual cages in the experimental room 24 hr. prior to the beginning of recording; the first 6 hr. of EEG data were discarded as an additional adaptation period. The room was sound deadened, temperature was maintained at 67 to 69°F., and the lights were on 12 hr. and off 12 hr. each day.

Waking, SWS, and PS were scored according to the criteria described by Swisher (1962). The control sleep cycle was recorded by EEG for 24 hr. Following this control period, sleep satiation was attempted by producing a prolonged sleep period lasting 24 hr. Pentobarbital sodium (40 mg/kgm IP) was injected at the beginning of the drug period and then further injections were given whenever four consecutive waking minutes were present in the record. Injections were required approximately every 3-4 hr. The drugged period was followed by 24 hr. of post-drug recording.

Results

The EEG during the drug period (satiation period) consisted almost entirely of slow waves and spindles similar to normal SWS in the rat, but with a higher frequency of spindles; no evidence of PS was present in the record. Small amounts of waking activity, amounting to less than 10 per cent of the total record were present.

Table 1 summarizes the data from this experiment. During the satiation period the amount of waking and PS is significantly less than pre-and post-treatment and the amount of SWS is greater ($p < .001$ for all 6 comparisons).

When the pre-treatment day is compared to the post-treatment day, the decrease in SWS is significant ($p < .05$), but the increases in amounts of waking and PS do not reach statistical significance. However, there was a significant increase in PS expressed as a percentage of total sleep during the post-treatment (recovery) period ($p < .01$).

Discussion

The supression of PS during barbiturate induced sleep is in agreement with experiments using human Ss, which have found barbiturates to depress REM sleep (Oswald, Berger, Jaramillo, Keddie, Olley, & Plunkett, 1963). It is likely that the total depression found in this experiment in contrast to the limited depression in human Ss is due to the dosage differences (a sedative dose in the human vs. an anesthetic dose in the rat).

The finding of an increase in PS post-treatment is consistent with the supression of this phase during the satiation period. This increase in PS following its' deprivation has been found by other investigators (Dement, 1960; Siegel & Gordon, 1965). Since barbiturate sleep does not mimic normal sleep but actually results in the deprivation of PS, a question would seem to be raised about this means of provoking sedation.

The idea that sleep functions as a need is supported both by the rebound increase in PS following its' deprivation and the decrease in SWS following satiation. The finding that barbiturate induced sleep consists of only one sleep phase and that SWS and PS respond

Table 1. A summary of the sleep cycle changes produced by pentobarbital – induced sleep.

	Pre-Treatment	Satiation-Period	Post-Treatment
Minutes			
Waking	790	135	838
PS	81	0	106
SWS	569	1305	496
	1440	1440	1440
PS as a per cent of total sleep	12	0	18

Mean values for the four rats.

differentially to this treatment lends support to the suggestion that there are at least two kinds of sleep; qualitatively distinct and subserving different functions (Dement, 1965; Jouvet, 1960).

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Notes

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