and olfactory bulbs and involved to some extent the following subcortical structures: septal nuclei, anterior commissure, caudate nucleus, globus pallidus, and preoptic area. The latter area is of particular interest because Andersson and Larsson (*7*) have reported that one dog with frontal lobectomy which involved the preoptic area did not drink when injected intravenously with hypertonic saline, but drank normally otherwise. This deficit lasted 3 weeks.

The above findings show that the neurological systems mediating drinking induced by cellular dehydration can be separated from volustatic mechanisms and are consonant with the earlier data of Fitzsimons (*2*) and Stricker (*2*), who demonstrated the physiological separation of the two controls, and with the more recent findings of Corbit (*3*) and Fitzsimons and Oatley (*3*), who found they acted independently of one another. These systems, which are independent structurally as well as functionally, may share a final common path originating in the lateral hypothalamus, because rats that have recovered from lateral hypothalamic lesions do not drink when challenged either with cellular dehydration (*8*) or extracellular hypovolemia (*9*).

The present preparation, in which only the volustatic control is operating, is of special interest because it allows an evaluation of the contribution of the extracellular component to drinking induced by reduction of both cellular and extracellular compartments, as occurs with water deprivation (*10*).

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15 October 1968

Sleep after Exercise

Abstract. *After moderate treadmill exercise, marked decreases in operant responding and in latency to onset of behavioral sleep occurs in cats. The sleep produced is characterized by enhancement of synchronized electroencephalographic activity with suppression of the desynchronized phase. The result is consistent with the theory that a function of synchronized sleep is to facilitate recovery from fatigue.*

A causal relation between fatigue and somnolence is strongly suggested by subjective experience. The corollary assumption, that sleep is restorative, is a tenet of common sense. Yet neither of these convictions about sleep has been clearly studied. Evidence showing that either somnolence or hypervigilance can follow exercise has been conflicting (*1–3*); evidence establishing the deleterious effects of sleep deprivation on psychomotor function and the relation between the quality and quantity of deprived sleep with sleep recovered thereafter, is indirect (*4–6*). I now present results of an attempt to clarify the relation of fatigue to subsequent sleep.

Ten adult male cats (3.5 to 5.0 kg) anesthetized with nembutal were operated upon, and electrodes were implanted for long-term electrographic recording. Screw electrodes were placed in the center of each superior orbital ridge for electrooculogram (EOG); screw electrodes were placed in the skull at A10, L5 and at P5, L3 bilaterally for electroencephalogram (EEG); stainless steel wires were fixed deep in the cervical musculature bilaterally for electromyogram (EMG). Fatigue was pro-

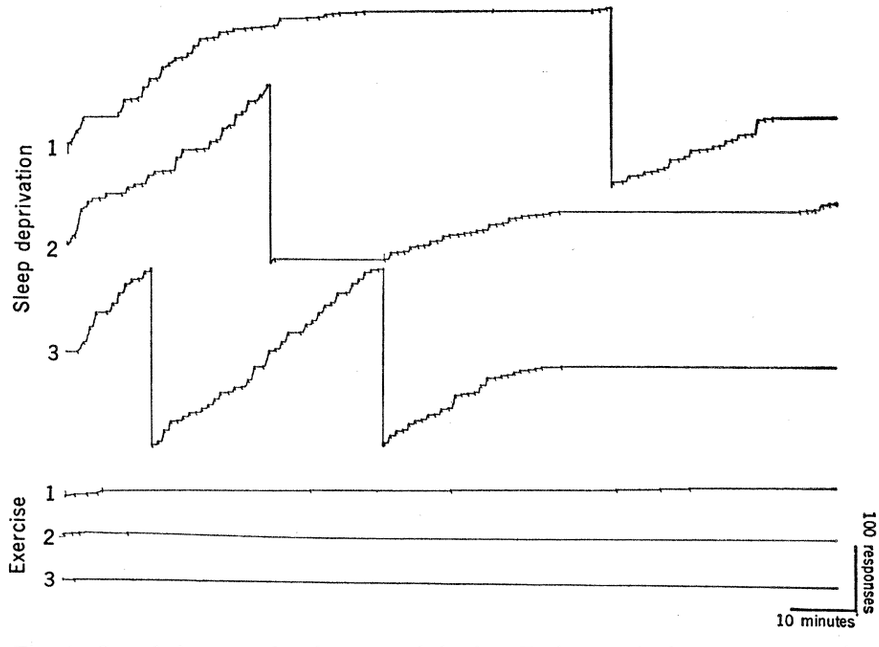

Fig. 1. Cumulative records of operant behavior. Each record plots response against time for the first 2½ hours of the six experimental sessions: a single vertical step is produced by one response, and 275 responses are required to move the pen through its full vertical range, after which it resets to zero; the diagonal hatch marks which are superimposed on the response record indicate reinforcements. Responding during this period was virtually eliminated by exercise.

Table 1. Behavioral and electrographic data. The total time is the first 5.5 hours of recording. Abbreviations are: S, synchronized sleep; and D, desynchronized sleep.

Condition	Recording days (No.)	Responses (No.)	Ratios			
			Awake/ total time	S/ total time	D/ total time	D/ time asleep
Sleep deprivation	12	131	0.53	0.41	0.06	0.14
Exercise	12	71	.35	.57	.07	.11
P diff.		0.001	.01	.01	—	.05

duced by exercise in a power-driven 30-inch (0.75 m) diameter wheel treadmill; controls were deprived of sleep by means of intermittent stimulation.

Six cats were studied in 38 pilot experiments to determine the behavioral and electrographic response to varying durations and intensities of exercise in a wheel-type treadmill in comparison with sleep deprivation and untreated controls. Two hours of exercise were sufficiently long to cause a full range of effects as a function of treadmill speed: rates below 2 rev/min (0.28 km/hour) were easily tolerated and produced no change in subsequent behavior; rates between 4 and 8 rev/min (0.57 to 1.13 km/hour) consistently produced hyperventilation and resistance to work, and were often followed by the sudden onset of sleep with EEG synchronization; speeds upward of 8 rev/min could not be maintained by cats and resulted in exhaustion with subsequent restless inactivity, hypervigilance, and EEG desynchronization.

Control conditions in the pilot experiments consisted of (i) no treatment, (ii) sleep deprivation by the swimming-pool technique (which allows the slow-wave but not the fast-wave phase of sleep), and (iii) subtotal sleep deprivation by means of intermittent stimulation of varied modality and strength under EEG control (which eliminated fast-wave and all but a small amount of slow-wave sleep activity). None of these conditions (which were studied because of the possibility that the exercise effect could be the result of sleep deprivation alone) ever produced the dramatic synchronized sleep often seen after moderate exercise, and there was actually very little difference in sleep parameters between control conditions when the duration was 2 hours or less. Subtotal sleep deprivation was selected as the most suitable control for subsequent experiments since it probably most nearly duplicates the sleep deprivation incidental to experimental exercise. Had either of the other control conditions been used the differences would have been as great or greater than those to be reported.

The apparent sleep-enhancing effect of moderate exercise (4 to 8 rev/min) was then studied systematically. In order to obtain more stable and sensitive baseline parameters than are possible when cats eat at will, four cats were trained to obtain food on a 1-minute fixed interval schedule of reinforcement. Operant behavior and the electrographically defined states of waking, synchronized, and desynchronized sleep were then monitored continuously except for 2 hours (2 to 4 p.m.) each day. After base lines were stable, exercise and subtotal sleep deprivation were then alternated for 6 days in each of four cats.

The cumulative records of operant behavior from one cat are shown in Fig. 1, and the corresponding electrographic record from the third day of exercise is illustrated in Fig. 2. After sleep deprivation, the animal responded almost continuously for 30 to 90 minutes; 50 to 75 percent of this period consisted of active waking behavior, and 300 to 600 responses were made. After exercise, the same animal responded for no more than 5 minutes and made a total of 1 to 10 responses. During this time, sleep behavior was observed, and the EEG was dominated by long runs of high-voltage, slow-wave activity. The EEG synchronization regularly preceded operant failure and the assumption of sleeping postures, suggesting a primarily central or neurogenic effect.

The data for the first 5 hours of recording for all animals were pooled. The mean latencies to onset of sustained (at least 1 minute) sleep with EEG synchronization and of sleep with EEG desynchronization in the two conditions are shown in Fig. 3. Exercise was followed by earlier onset of synchronized sleep and a later onset of desynchronized sleep than sleep deprivation was. Both of these differences are statistically significant ($P < .01$).

Responding was markedly reduced after exercise (Table 1). The decrease in time spent awake (by electrographic criteria) and a corresponding increase in time spent in synchronized sleep were both statistically significant. Although there was no significant difference in the percentage of total time spent in desynchronized sleep, the relative amount of sleep time occupied by that phase was significantly reduced.

Episodes of synchronized sleep longer than 15 minutes were almost twice as frequent (34 compared to 20) and long-

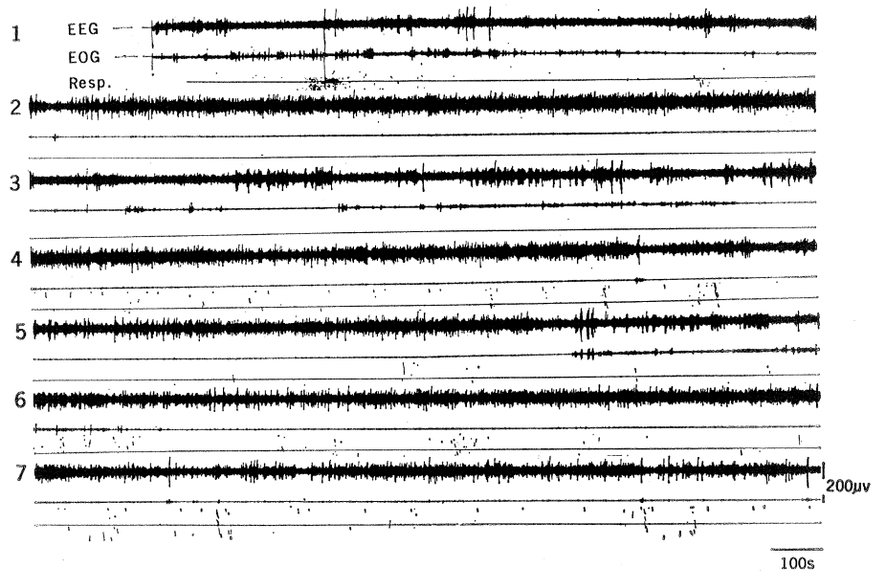

Fig. 2. Electrographic records after exercise in one cat (day 3 in Fig. 1). The seven consecutive three-channel records comprise the first 2 hours of the third exercise experiment. In each record *EEG* is biparietal electroencephalogram, *EOG* is the electrooculogram, and *Resp.* indicates response reinforcement (food delivery). The cat responded only once, then entered a prolonged period of synchronized sleep (lines 1 to 7) interrupted by brief arousals. There was no desynchronized sleep during this time.

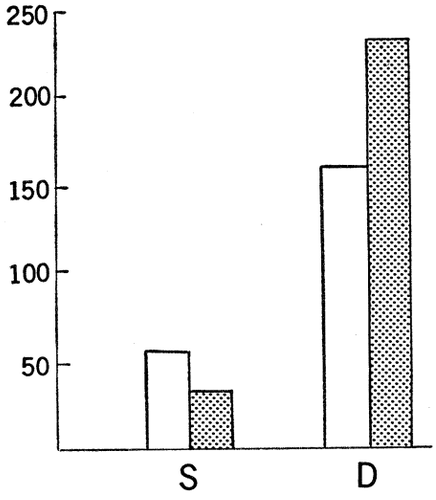

Fig. 3. Latencies (time in minutes as ordinates) of synchronized sleep (*S*) and desynchronized sleep (*D*) after sleep deprivation (open bars) and exercise (shaded bars). Exercise was followed by an earlier onset of synchronized sleep and a later onset of desynchronized sleep than sleep deprivation was.

er (32 compared to 28 minutes) after exercise. The six longest episodes occurred after exercise; one of these, lasting 59 minutes, was almost twice as long as the longest control episode (32 minutes). Thus, the increase in amount of synchronized sleep is a composite function of earlier onset, increased frequency, and increased duration of that state.

An hour-by-hour analysis indicated that the synchronized sleep-enhancing effect diminished through the first quarter of the 22-hour recording period and reversed itself in the second and subsequent quarters. The time course of these changes in one cat is indicated in Fig. 4. Reciprocal rebound effects for waking and synchronized sleep are apparent; exercised animals sleep first and awaken later, whereas the controls do the opposite. For this reason, the total quantity of sleep and of its component phases during the entire recording period was not significantly different for the two conditions despite marked differences in its distribution within that time. This result would seem to mean that environmental effects contribute relatively little to the total quantity of sleep, implying in turn that this quantity is somehow set by endogenous, probably neural, mechanisms.

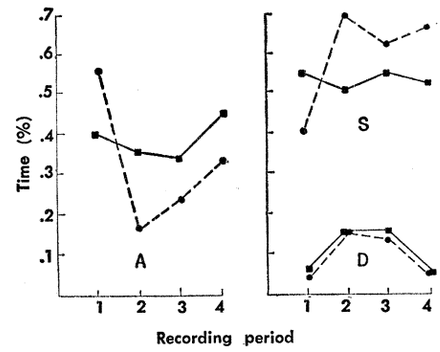

Fig. 4. The amounts of time (expressed as percentages on ordinates) spent by cat number 2 awake (*A*); asleep with synchronized EEG (*S*); and asleep with desynchronized EEG (*D*); after exercise (dots) and sleep deprivation (squares) in each successive quarter of the recording period (abscissas). In the first quarter after exercise, waking was lower and synchronized sleep was higher than the control, whereas the opposite was true thereafter. Desynchronized sleep does not change its distribution in time.

The results suggest an inverted U-shaped relation between fatigue and subsequent sleep. Exercise demands within critical limits, that may be set by the animal's metabolic capacities and previous experience, produce somnolence. When these limits are exceeded sleep disturbance is the result, and there is probably an intermediate region where the two effects are in balanced opposition. This observation and interpretation can account for apparent discrepancies between other studies of this problem (*1–3*).

The mechanism and functional significance of these opposite effects are presumably different. The arousing effect has not here been systematically documented or analyzed, but might reasonably be the result of activation of emergency or stress mechanisms, such as the pituitary-adrenocortical system. The adaptive advantage of vigilance after severe stress, as in flight from a predator, can be imagined.

This study has been more sharply focused on the sleep-enhancing effects of moderate exercise. The mechanism of this effect has not been fully clarified but increased body temperature may be a mediator; the effects of heat in synchronizing the EEG have been demonstrated in other contexts with hypothalamic structures implicated (*7*). This interpretation could also explain the observed suppression of desynchronized sleep which results from increased body temperature (*8*). The contribution of chemical mediation cannot be ruled out, however.

Whatever the mechanism, a functional role of synchronized sleep in recovery from fatigue is strongly suggested. Such a function has been previously suggested by these observations: (i) synchronized sleep is concentrated in the first half of the night of human sleep (*6*) as if it were responsive to the fatigue of day-long vigil and activity; (ii) this tendency is increased by total deprivation of sleep after which the synchronized phase is recovered first and in amount proportional to the time spent awake (*4*); and (iii) exercise increases the relative amount of sleep with intense EEG synchronization in man (*2*).

Current theory of the function of the desynchronized phase of sleep accords sleep a different, and in some respects an opposite, function—control of excitability of cells within the nervous system through the spontaneous and intense discharge of some nervous elements (*9*). These apparently disparate views are, however, reconciled by the hypothesis, indirectly supported by cellular data (*10*), that the synchronized and desynchronized phases of sleep are continuous manifestations of disinhibition, and hence permit a unitary functional interpretation of the function of sleep for the brain—rest of inhibitory nerve cells. Whatever the significance of desynchronized sleep for the brain, it is like the synchronized phase of sleep in forcing rest, through inactivity, on the somatic musculature.

Note added in proof: Since submitting this report, I have read the paper by Matsumoto *et al.* (*11*). Comparable experiments in rats produced results identical with those reported here.

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