

Fernández-Morán and seems inescapable from a knowledge of these objects. An important unanswered question is simply, "How much?"—a question provoked in the open mind by such knowledge.

8) The concept of organelle isotopy is structurally correct from both physical and etymological standpoints. Plant and animal, microorganismal and metazoan cilia have basically the same form. This similarity does not impose the limitation of common evolutionary origin on the structure itself in the developmental sense.

9) On or off the earth, "non-atoms" represent the subatomic particles. The term stems from the well-known philosophical device of *differentia and negativa* and denotes a subset class that relates logically to the higher levels in the figure.

10) The lifetimes of such units as the enzyme-substrate complex are sufficiently short that ingenious instrumental measures, such as those conceived and brilliantly executed by Britton Chance, have been needed for their measurement. As pointed out in the article, the very organelles in or on which these fleeting events occur may be observed in vivo by cytologists at relative leisure. The qualification "relatively" was used by me deliberately and precisely to emphasize comparative lifetimes.

11) I cannot explain Nicholls' failure to find a need for a bridge or a link. A current key concern in physiological genetics and developmental embryology is the construction and testing of hypotheses that relate directly to the ascent from the macromolecular level that is experienced in the lifetime of every organism. If authoritarianism must be resorted to, it is well known that eminent experimentalists since Altmann and Hertwig have grappled with this problem along lines closely analogous to mine. That Nicholls' general accusation—that I have underestimated the contributions of biochemistry at the one extreme or of evolutionary theory at the other—is unfounded may be seen from a careful reading of my article and of the contextually relevant sections of the references cited.

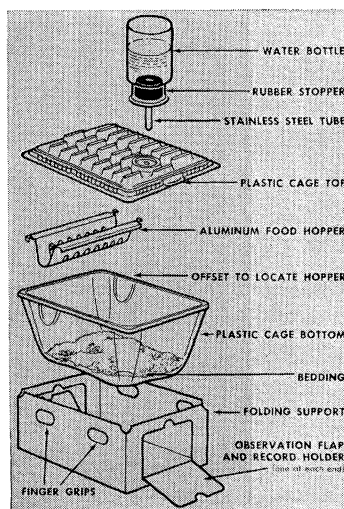
CHARLES F. EHRET
Laboratoire de Biophysique, Université
de Genève, Geneva, Switzerland

Dream Deprivation

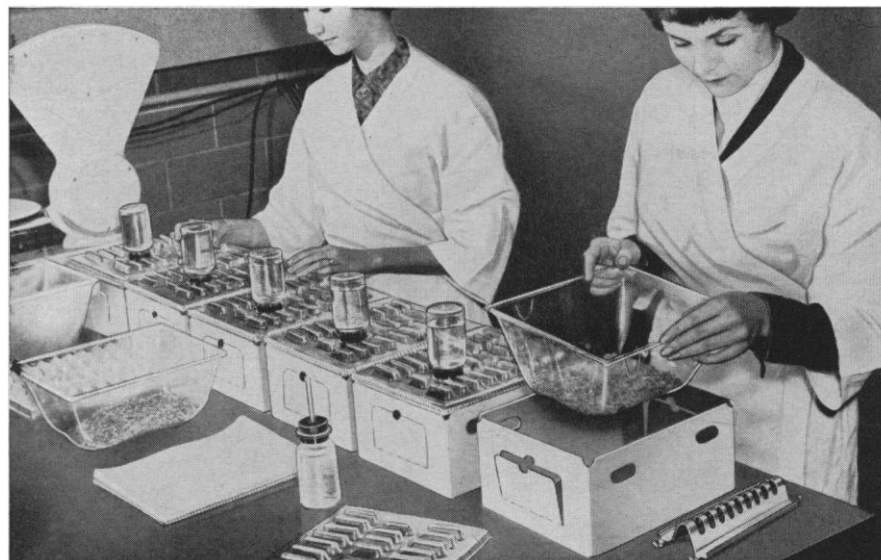
William Dement tentatively concludes from an interesting study of "The effect of dream deprivation" [*Science* 131, 1705 (1960)] that "a certain amount of dreaming each night is a necessity." A different interpretation of

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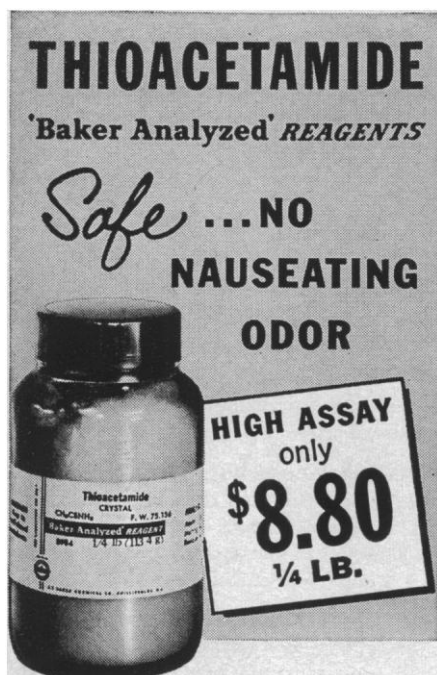
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the data, however, appears equally plausible. Dement's procedure, in brief, was as follows: to determine the subject's base-line total nightly dream time and sleep pattern, eye movements and brain waves were recorded during a variable number of undisturbed nights of sleep. During the subsequent nights the subjects were awakened each time the electromyographic (EMG) eye-leads indicated that they were dreaming (and the electroencephalographic [EEG] leads presumably indicated that they were in light sleep). Immediately after, when allowed a number of undisturbed nights of sleep ("recovery nights"), the EMG showed a significant elevation in the nightly dream time as compared with the base-line levels. This finding is open to the following interpretation: The psychological disturbance caused by the abrupt awakenings (as indicated by "anxiety, irritability . . . difficulty in concentrating . . . apparent panic") was closely correlated with a disturbance in the sleep pattern which included an increase in the amount of time spent in light sleep. Since evidence previously presented by Dement and Kleitman [*Electroencephalog. and Clin Neurophysiol.* 9, 673 (1957)] strongly suggests that dreaming occurs during periods of light sleep, the "progressive increase in the number of attempts to dream" during the "dream deprivation" nights and the increased percentage of dream time during the "recovery nights" may have been a secondary consequence of the increase in light sleep time during these periods.

After a varying number of nights off, the same subjects returned to the experimental room and were awakened when the EMG indicated that they were *not* dreaming (and the EEG presumably indicated that they were in deep sleep). Subsequently, when permitted a number of undisturbed nights of sleep ("recovery nights"), the EMG did *not* show an increase in the amount of dream time over the base-line levels. This finding can be interpreted as follows: By this time the subjects had become adjusted to the procedure. Since they were now habituated and undisturbed, they slept normally; consequently, as compared with the base-line levels, they did not show a significant change in the percentage of light sleep time or the percentage of dream time during the "recovery nights" (or during the preceding nondream awakening nights).

It would be inappropriate to accept either Dement's interpretation or the above interpretation until a second experiment is completed in which subjects are *first* awakened when the EMG indicates that they are *not* dreaming and, after an intervening "recovery" period, are awakened when the EMG indicates that they are dreaming. Such an experiment may give the opposite results of

those reported by Dement: Subjects may show an increased amount of dreaming during "recovery nights" which follow nondream awakenings and no significant increase in the amount of dreaming during "recovery nights" which follow "dream deprivation."

THEODORE XENOPHON BARBER
Worcester Foundation for Experimental
Biology and Medfield State Hospital,
Harding, Massachusetts

I wish to comment briefly on William Dement's tentative interpretations of the effects of his experimental studies in dream deprivation. Over two decades ago Nathaniel Kleitman put forth the notion of a subcortical wakefulness center which influenced the course of events in the sleep-wakefulness cycle. The more recent studies on the reticular activation system appear to have borne out some of these earlier ideas. When these data are considered in connection with the elucidation of the existence of the corticofugal fibers linking the cortex with the reticular system, and thereby creating a reverberating circuit, we have the beginnings of a neurophysiological model elucidating the dreaming process. We know as a result of the recent studies of Kleitman and his associates that repetitive bouts of partial arousal associated with dreaming occur universally as a nightly process. At certain times during the night it would appear as if the threshold in the reticular system is low enough for partial cortical arousal to occur. The events associated with this are experienced subjectively as the dream; but objectively these events further influence the threshold in this system. It is in this manner that it has been suggested that the cortex participates in its own arousal.

Freud had no neurophysiological model to serve as a guide in evolving his own dream theory. He was forced to see the dream as psychologically inspired by a disturbing unconscious impulse and as taking shape through the mechanism of dream work oriented towards disguising the impulse and maintaining sleep. The neurophysiological model, by comparison, suggests that the level of arousal varies during the night in a rhythmically patterned manner and that states of partial arousal are associated with more or less extended periods of dreaming.

Dement's study is concerned with the question of what happens when these events are interfered with in a specific way, namely, by eliminating that aspect of the cycle associated with dreaming. Both direct effects relating to dreaming and indirect, more distant effects relating to generalized behavioral disturbances were noted.

With the above considerations in mind, one can then move in two pos-

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sible directions in evaluating the direct results of dream deprivation experiments. One can minimize all factors other than the occurrence of dreaming and thus focus mainly on the subjective aspects of the experience. One then begins to talk as Dement does: "It is as though a pressure to dream builds up . . ." and ". . . a certain amount of dreaming each night is a necessity."

A second approach would emphasize the fact that the experimenter is interfering with states of partial arousal which presumably may have significance for the organism apart from dreaming. If these states of repetitive partial arousal are, for example, associated with vigilance operations during sleep, then the direct effects noted—namely, the increased frequency of dream attempts on the experimental nights and the increased nightly dream time on the recovery nights—may represent states of heightened vigilance of which dreaming is but one manifestation.

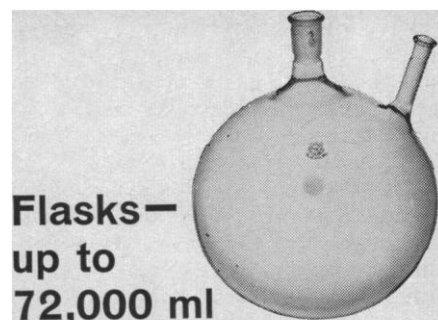
MONTAGUE ULLMAN

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Theodore Barber's alternative interpretation of the results is based mainly on his perception of a methodological weakness in the experimental procedure. He feels that the multiple awakenings, in and of themselves, could have been responsible for the rise in dream time seen on recovery nights after all, because, as he points out, the control awakening series always followed the dream deprivation series and "by this time the subjects had become adjusted to the procedure." He feels that if the control awakening series had been carried out *before* the dream deprivation awakening series, one might have seen a dream time rise following the control awakenings and little or none following dream deprivation.

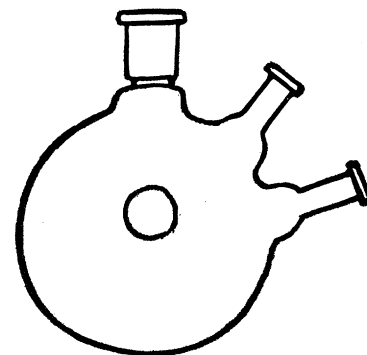
Although we did not consider such a possibility "equally plausible" at the time the article was in preparation, it was obvious that it could not be entirely ruled out. Happily, there is no need now for an exhaustive documentation of the reasons for our original judgment. We have recently concluded experiments along the line independently suggested by Barber in his letter; and we have found that a second dream deprivation series in the same subject is as effective as the first, that no habituation is apparent, and that when the control series is carried out first, a dream time rise still follows the later dream deprivation series.

There are, however, several points in Barber's letter that warrant further comment. First, one of his contentions seems to be that abrupt awakenings caused the observed psychological disturbances. In all our work in this area,



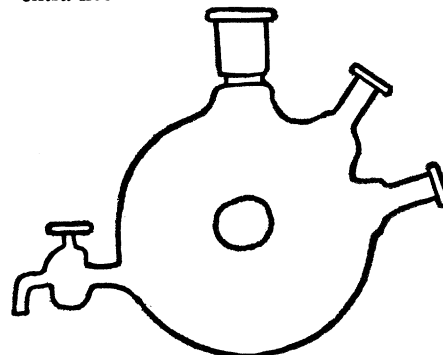
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one thing that has continued to impress us is how well subjects tolerate various disturbances of their sleep. When we were waking subjects many times a night to elicit dream recall in our early studies, we never observed any sort of psychological disturbance. To be sure, until the dream deprivation experiments were made, we had never done 20 to 30 awakenings in a single night. However, with the support of our more recent experiments, we are still inclined to view the psychological disturbances reported in the article as a *secondary* consequence of the reduction in dream time and not as a primary result of awakenings per se.

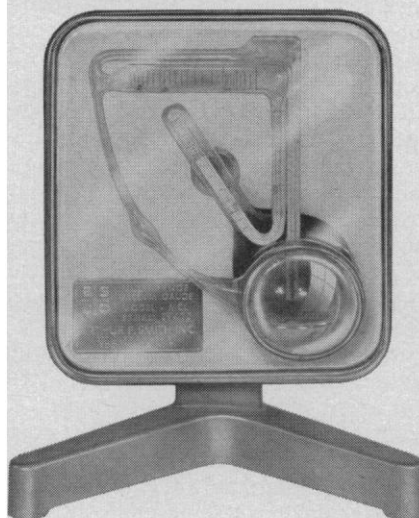
Second, taking his cue no doubt from my own statement in the article which referred to earlier work, "the eye movement periods were observed to occur regularly throughout the night in association with the lightest phases of a cyclic variation in depth of sleep as measured by the electroencephalogram," Barber uses the term, *light sleep*, saying, "the increased percentage of dream time during the recovery nights may have been a secondary consequence of the increase in light sleep time during these periods." It should be made very clear that what is meant by "light sleep" in this context can only be the so-called rapid eye movement period. The term, *eye movement period*, refers to a discrete interval of sleep during which rapid eye movements are seen in association with a characteristic low voltage, nonspindling EEG pattern (called stage 1 by us) which persists unchanged throughout the entire period although the amount of the eye movement may vary widely. Dreaming, as shown by earlier work, very likely occurs throughout the entire eye movement period, its presence being specifically indicated by the appearance of the stage 1 EEG pattern. The rapid eye movements are also related to dreaming, but more precisely to the visual imagery involved, and accordingly may vary in quantity from virtually none to many depending on what the dreamer is doing in the dream. Thus, for all practical purposes, the terms *eye movement period*, *stage 1 period*, *dream period*, and Barber's *light sleep* are synonymous. It should now be apparent that one cannot say, as Barber does, that increased dream time is a secondary consequence of an increase in light sleep time. The light sleep (stage 1 EEG) stage is simply the physiological concomitant of dreaming. Until more is known about mind-brain interaction, it might just as well be said that the increase in light sleep time was a secondary consequence of the increase in dream time.

Perhaps Barber had some other attributes in mind when speaking of light sleep which made his proposition seem

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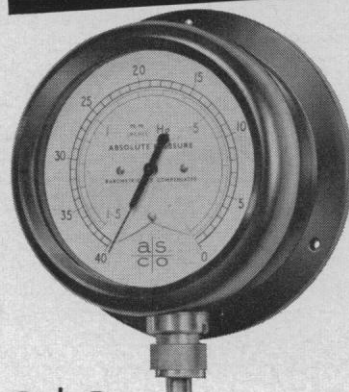
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more reasonable. However, these should be specified because light sleep (or deep sleep) is a concept that can have objective meaning only in terms of the variable or variables used to describe it. One may speak of light sleep in terms of a low voltage EEG (as we have), or a low arousal threshold, or a high incidence of body movement, or a high heart rate, and so forth. Unfortunately for the concept, the various measures of lightness of sleep do not correlate well with each other. For example, the incidence of gross body movement is lower during stage 1 (dream) sleep than during adjacent periods of stage 2 (deeper) sleep. I personally feel that if depth of sleep must be measured, the most meaningful criterion is the arousal threshold. Even in this instance dreaming fails to qualify as light sleep. Current work in several laboratories seems to indicate that subjects are often more difficult to arouse while dreaming than at certain other times. The use of the EEG, or any other variable, as *the* criterion of depth of sleep is merely a matter of taste or convenience, and probably depends to some extent on how well the chosen variable fits the experimenter's personal subjective notion of this elusive characteristic.

A final point is that the eye movement potentials are not muscle potentials and hence the eye movement record

is not an EMG (electromyogram). There is a more or less steady potential difference existing across the eyeball (cornea positive—retina negative). The eye movement potential is a consequence of the spatial change of the electrical field of the eyeball dipoles with reference to the fixed periorbital electrodes. Passive movement of the eyes will elicit such a potential with reference to fixed recording points, and retinal destruction will abolish it. There is no evidence of EMG potentials in the recordings that we routinely obtain, presumably because the extraocular muscles are too far from the recording electrodes.

Some of the above considerations may also apply to Ullman's comments. He refers to the dream periods as states of "partial arousal" perhaps "associated with vigilance operations during sleep." It seems to me that the term, *partial arousal*, is as ambiguous as the term *light sleep*. If by "partial arousal" is meant only the occurrence of the stage 1 EEG pattern, then the term is redundant. If the state of partial arousal is thought to have some other attribute (for example, we might logically assume it would include a lowered arousal threshold), again, we must ask, is this additional attribute in fact associated with the stage 1 EEG period and dreaming?

The periodic occurrence of a low

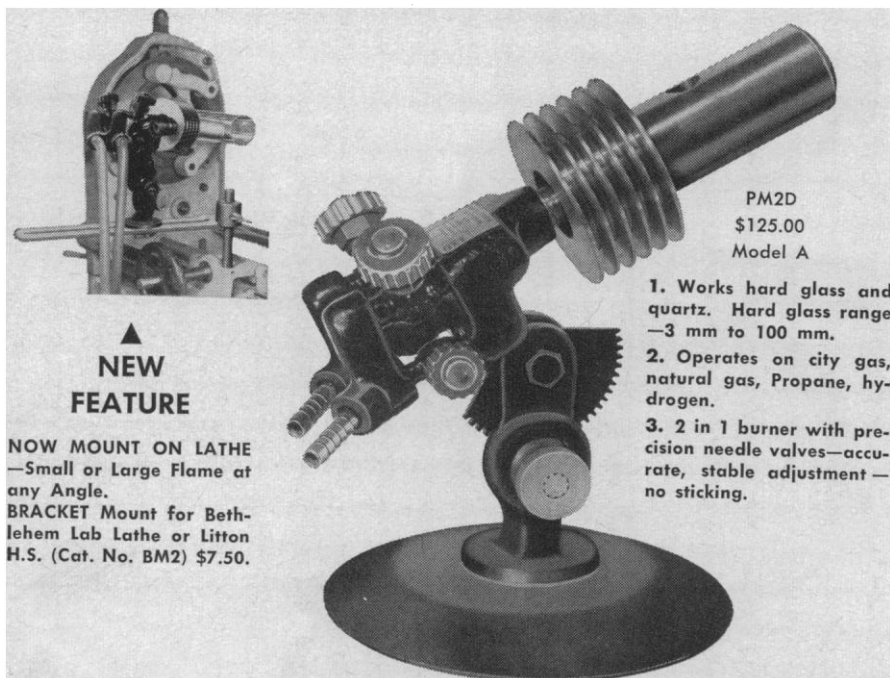
voltage, fast EEG stage during normal sleep has also been observed in cats by myself [*Electroencephalog. and Clin. Neurophysiol.* **10**, 291 (1958)] and Jouvet *et al.* [*Compt. rend. soc. biol.* **153**, 1024 (1959)]. Since we can do no more than speculate about dreaming in cats, and since it seems unlikely that cats dream in the manner of humans, we might assume that the occurrence of a low voltage, fast EEG stage in this animal is a pure example of a vigilance operation. It might follow that during these periods the cat is more alert to various stimuli, and that the frequent occurrence of such periods during sleep greatly enhance his chances for survival. However, even here, where the issue of dreaming does not complicate the picture, the cat is, if anything, less vigilant. Jouvet reports a definite *increase* in arousal threshold during these low voltage periods as opposed to the slow wave stage of sleep, and, in a less refined study, I found that it was difficult to differentiate the two phases in terms of arousal threshold. I think that Ullman must recast his definition of vigilance with an eye to some of the more recent findings regarding the functional significance of the various EEG patterns during sleep. I think that he might also ask himself how much of the current neurophysiology of the brain stem really applies to the physiology of sleep in a more than speculative way, especially since there are still large gaps in a purely descriptive picture of sleep physiology.

WILLIAM DEMENT

Department of Psychiatry,
Mount Sinai Hospital, New York

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This statement contains several errors. First, it may give people the incorrect impression that apochromats were produced empirically (by craftsmen), whereas it ought to be well known that they were designed entirely mathematically by the famous professor