Evidently, paradoxical sleep is not regulated by merely one type of neurons; rather there are quite important interrelations between the various adrenergic systems as shown by biochemical and by neurophysiological techniques.

Selective and Partial Deprivation of Sleep

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Because of limitations of time, space and competence, this review has concentrated on selective deprivation and partial deprivation of sleep by behavioral control and on the behavioral consequences of such control. By selective deprivation is meant the elimination of certain stages; by partial deprivation is meant limitations of length of the sleep period. The review specifically excludes suppression of sleep stages by pharmaceutical and surgical means and biochemical and physiological responses to the intervention.

My review will be subdivided into three major sections: rapid eye movement (REM) deprivation (RD); stage 4 deprivation (4D); and partial deprivation (PD) or reduced sleep.

REM Deprivation

Whether implicitly held or explicitly stated; whether derived from intuition, elaborations of Freudian theory or computer models, three questions form the substrata of research on the behavioral effects of RD: Does the absence of dream (REM) sleep result in personality disorders? Does the elimination of the REM result in enhanced generalized or specific drive states? Does a lack of REM sleep offset learning and/or memory?

To answer these questions, sleep researchers, beginning with the classical study of Dement reported in 1960, have in little over an ensuing decade yielded up at least 40 human studies and 40 animal studies which have involved literally hundreds of humans and a veritable horde of animals.
The following is, however, an evaluative summary.

Is REM necessary for the integrity of the personality or functional responsivity? There have been no clear-cut cases in which RD has resulted in a markedly disorganized personality response either in normal or in psychotic subjects who may be thought to be particularly susceptible to personality disorganization. Similarly, after prolonged deprivation, animals have failed to provide evidence of disruptive functioning. Where some signs of disorganization have been noted, these findings have been fraught almost inevitably with the dangers of subject or experimenter expectancies and/or stressful experimental conditions.

Does REM deprivation result in a higher need for ‘drive’ discharge? Again, in human subjects, there have been some subtle signs of variations in motivational states. However, these have not been found on objective testings and the most carefully controlled findings have been essentially negative. Animals have shown some evidence of increased hunger, sexual drive and aggression. However, the responses are similar to those noted in more generalized stress paradigms and, procedurally, stress is a necessary corollary of RD animal experiments. In almost all positive instances the ‘seek and ye shall find’ potential was high.

Is REM sleep critical in learning and memory processing? In the area of verbal learning, both in regard to learning of new material and retention of material learned prior to RD, the weight of evidence is to the contrary. The animal studies are severely compounded by methodological problems and the data highly conflictual.

In overall summary, I would feel that a jury would have to bring in a verdict of ‘not guilty’ while remaining worried about a considerable amount of circumstantial evidence.

A network of findings and phenomena outside the behavioral control matrix are supportive of the generally negative conclusions that I have drawn. To cite a few selected findings:

1. Drug suppression of REM sleep over long periods of time (up to 1 year) yields no positive support.
2. Partial sleep deprivation resulting in sharply reduced REM amounts for long periods of time similarly provide no supportive data.
3. REM amounts are not in any clear way concurrently responsive to wide variations in psychic changes. Schizophrenics differ little, if any, from normals in baseline REM amounts. The sharp learning and personality changes from the age of 12 to the 60s are associated with no parallel changes in REM characteristics. Geniuses and retardates without anomalies are
very similar in their REM amounts.

4. Across the phylogeny of animals there is no apparent relationship between the large ecological and behavioral differences which are present between species and the REM characteristics displayed by such animals. The primitive opossum has more REM than man (to maintain the integrity of its personality, and to consolidate its rich memories?); the cat, a subtle beast, has somewhat less.

5. REM, assigned the broadest functional rôle of all sleep stages, shows a most narrow range of individual differences.

6. REM appears to be the least responsive of sleep stages to presleep variations such as stress and time of prior wakefulness.

Let us assume that I am correct in my evaluation that there is a limited or no relationship between REM variations resulting from RD and behavioral consequences as we have measured them. There are three reasonable responses to such findings: (1) the relationship is indeed limited; (2) the relationship is subtle beyond our present visions and/or our measurement procedures, and (3) the independent variable, REM, has escaped our control.

I suspect that we neither can nor should accept as proven the first of these propositions. Intriguing positive findings dot the littered field of data: the varied REM rebound characteristics of schizophrenics; the positive responses of depressive patients; the subtle variations in emotional and drive responses; the positive rebound of REM to intensive learning. Certainly our measures have been crude and our hypotheses simple. Greenberg and his coworkers have, for example, varied the simple assumption of effects on verbal learning to consider more complex cognitive functions to be attributed to REM. Such continued efforts can only be encouraged. Increasingly there has been a re-evaluation of the RD process itself. This has taken three forms, a recognition of individual cases and conditions which do not show classical RD responses; a re-evaluation of the REM state itself, and questions about REM as a sleep-bound phenomenon.

First consider the ubiquity (or lack of ubiquity) of the REM rebound phenomenon. Cartwright and Monroe [1967] noting that two of Dement’s initial 7 subjects [1960] showed no rebound, purposefully studied this problem with 10 subjects over 2 nights of RD. Four of the subjects showed a substantial rebound, 2 subjects showed essentially no rebound and 4 subjects gave what the author described as ‘ambiguous’ results. The experimenters noted that in these 4 subjects the REM periods were frequently interrupted by short periods of stage 2 sleep. These interruptions prolonged
REM periods and were more often than not included in the scoring of REM. When these periods were carefully eliminated, the amount of REM in the 4 ambiguous cases was only slightly elevated or not elevated at all. Studies of two schizophrenic populations [Azumi et al., 1967; Zarcone et al., 1968] have given indications of a reduced rebound in active schizophrenics. Wide individual differences in all these studies were noted. For example, in one of Azumi’s three cases, REM rebound was, in fact, quite high. Nevertheless, these findings suggest that REM pressure may be dispersed or not developed in certain state conditions. Both of these findings, absence of strong rebounds in certain individuals or under certain circumstances, may explain some of the marginal character of the results of RD experiments when group data are considered and reported.

The idea of the REM period being a homogeneous event has been sharply questioned since the late 1960s. In 1969, Dement emphasized that the REM state was at least a two-phase process of tonic changes (such as the EMG suppression) and phasic changes (such as REM and muscular twitches). Within the phasic period, the presence of spikes emanating from the pons, oculomotor nuclei, lateral geniculate nuclei and the visual cortex (PGO) were identified as the sine qua non of REM sleep. More recently, Dement [1972] has written that the REM state may be simply a confluence of ‘separate processes which can readily operate, function, or discharge outside of REM periods and independently from one another’. Under such a conception ‘REM sleep deprivation may well be an entirely illusory process. It is possible that nothing at all is eliminated; only the temporal confluence is disrupted’. Dement suggests, then, that under RD the REM process may be redistributed into other sleep periods or ‘a more interesting possibility would be a shift to the waking state’.

A third line of research and reasoning certainly compounds the RD question. This is the notion that the REM period represents a continuous ultradian rhythm across the 24 h and occurs within the waking state [Webb and Agnew, 1967; Globus et al., 1971; Sterman et al., 1972]. Here it is suggested that REM is not a sleep-specific state but rather may be measured and displayed more readily in sleep where the ‘noise’ of behavior is reduced. From this line of reasoning, REM continues to occur throughout the waking state in its rhythmic cycle and the measurement of the REM state is simply suppressed. It would follow from this that ‘deprivation’ of REM by awakenings would merely be suppressing the measurement of REM. Further, this ‘deprivation’ or suppression would be minimal in the 8 h of sleep when its ‘deprivation’ is compared with the 16 h of
Dement [1971], who initiated the search that we have reviewed, recently drew the following conclusions: 'However, we must conclude that the selective deprivation of REM sleep is not likely to give us the enlightenment we crave. The answers to the questions posed by the existence of REM periods and the apparently unique activities that they facilitate must await a more factual (less metaphorical) description of their basic mechanisms. We need to know exactly what neuronal systems are responsible for phasic activity. We need to know whether or not phasic activity occurs all the time, but is recognizable only during REM periods because it is more synchronized. We need to know what neurotransmitters are involved in phasic activity, and so forth. Such knowledge may take a long time to accumulate, but when we have it, we may almost automatically understand the existence of REM sleep.'

I cannot completely agree; such information will be interesting indeed. However, until the findings are related to the functional behavior of the human, I, as a psychologist, will not 'understand' REM sleep.

Stage 4 Deprivation

At the outset let me note my personal chagrin at the lack of 4D data. In the early 1960s, noting that all of the deprivation studies were RD, I formed the Society for Prevention of Cruelty to Stage 4, in hopes of drawing some of the heated attention to this prominent and potentially important stage of human sleep. Since I can find only 6 experiments that have utilized 4D and three of these were performed by my own laboratory, it is clear I have not been successful in spreading my concern.

The first experiment was essentially a demonstration experiment [Agnew et al., 1964]. Five subjects were stage 4 deprived by awakenings for two nights. A significant rebound effect was demonstrated, i.e. there was a significant increase in stage 4 sleep during two recovery nights when compared with the baseline levels of stage 4 sleep.

Three years later we reported a comparative study of 4D and RD [Webb and Agnew, 1967]. Two groups of 6 subjects each were deprived of either stage 4 or REM for 7 successive nights. The deprivation processes were strikingly different. Figure 1a shows the number of 1-min epochs in which
shock occurred for the two conditions on successive nights. Both show the classical increase in ‘pressure’ across the successive nights. Stage 4 rises more sharply and required significantly more stimulations for elimination. Within the night the concentration of shock requirements was differentially distributed. Figure 1b shows the percentage distribution of shocks across all nights of the experiment by thirds of the night. The shock requirements are suggestive of a ‘stronger need’ for stage 4. The recovery data suggest an opposite conclusion. Figure 2 shows the rebound and recovery characteristics of the two stages. REM rebound clearly exceeds stage 4 rebound and persists longer.

A battery of performance and personality tests were administered. Although there were no statistically significant differences, two psychologists, given the prior-post set of personality tests, and separated by groups, found that the 4D protocols were suggestive of a ‘depressed outlook’ whereas the RD protocols ‘resulted in a state of higher irritability and lability’.

Only two other considerations have been given to stage 4 involving behavioral consequences [Donnell et al., 1969; Johnson et al., 1972], In these experiments differential REM or stage 4 deprivation were combined with total deprivation. In the first experiment, 2 nights of total deprivation were followed by 5 nights of recovery sleep. One-third of the subjects was 4D during the first 2 nights of recovery, one-third was RD and one-third served as a control group. On a wide variety of performance tests, mood scales and personality tests, no differences were found in the recovery rates of the 3 groups. The authors stated that ‘these preliminary data indicate that REM sleep and non-REM sleep are equally recuperative and that performance on these tasks are more dependent on getting some sleep rather than a particular stage of sleep’. In the second experiment, the subjects were 4D or RD for 3 nights preceding 1 night of total sleep loss. Again a broad spectrum of psychological tests were used. The authors concluded as follows: ‘The single significant finding from an extensive and intensive battery of tests suggests the differential effect of deprivation of stage REM and stage 4 does not result in dramatic changes in waking behavior though both produced significant changes in sleep per se. The single significant finding cries for replication before a splurge into fantasizing and theorizing as to the role of stage 4 in long-term memory’.

In brief summary of the limited data regarding 4D, I believe that we
can certainly conclude that the deprivation procedures provide evidence that stage 4 sleep and REM sleep are two essentially different processes within sleep. There is no strong evidence that deprivation of stage 4 compared with stage REM results in clearly different behavioral consequences.

Partial Sleep Deprivation

Partial sleep deprivation (PSD) presents a compounded problem. When sleep is reduced from, say, 8 h to 4 h, there is not only an absolute reduction in sleep time but there is also a differential reduction in the stages of sleep. This stems from the differential distribution of REM and stage 4 sleep within the night. The reduction of sleep from 8 to 4 h results in a 66-percent reduction in REM sleep and only a 12-percent reduction in stage 4 sleep. In short, PSD is also differential REM deprivation.

PSD may also be further compounded by a circadian effect. To stay up longer (say until 0300 h) and get up at a regular awakening time (say 0700 h) is quite different from going to bed at 2300 h and awakening at 0300 h. Although the restricted regimes are equal in length, performance at 0300 h is quite likely to be different from performance at 0700 h.

Prior to the early 1930s there were a few, primarily single subject, experiments involving 1-2 nights of restricted sleep. Between the 1930s and the new era of sleep research of the 1960s, the literature is silent. However, in review of the literature [Webb, 1969], I was able to cite 5 studies which had imposed restricted regimes: Sampson [1965], a 21/2-hour sleep regime for 3 consecutive nights; Webb and Agnew [1965], a 3j/2-hour regime for 7 nights; Dement and Greenberg [1966], a 4-hour regime for 3-6 nights; Rush et al. [1968], a 4-hour regime for 7 nights; and Wilkinson [1969] who imposed regimes of 1, 2, 3 and 5 h for 2 nights each with recovery nights in between.

Three of these studies reported the effect of PSD on sleep stages. In brief summary, REM sleep, as expected, was sharply restricted and there was some evidence that the deprived stage REM sleep compensatorily ‘moved forward’ in time into the restricted periods. Stage 4 sleep showed some increase. There is a REM rebound effect and a curious, but consistent, stage 4 rebound (since there is only a limited reduction) during recovery nights.

Three of the experiments used performance batteries. Sampson [1965] found no changes on brief digit span or complex serial subtraction tasks. Webb and Agnew [1965] reported ‘evidence from the behavioral tasks...
that performance had begun to deteriorate on the seventh and eighth nights... These decrements were neither uniform nor fully consistent.' The Wilkinson [1969] experiment used a vigilance task. His findings were summarized in the earlier review as follows: utilizing signal-detection theory in the interpretation of his data, Wilkinson found 'the "capacity" to perform discrimination on the vigilance task was not affected on the first night until sleep had been reduced to less than three hours.' Because stage 4 is not materially decreased until that point, he inferred that stage 4 might be related to the capacity to perform. On the other hand, 'willingness or motivation' to perform was affected when there was less sleep reduction but REM deprivation was occurring.

Two studies of chronic restricted regimes have been reported. Johnson and MacLeod [1972] using 3 subjects, reduced sleep from 7 1/2 h by 1-hour units at 2-week intervals to 4 h of sleep and maintained the 4-hour regime for 3 weeks (one subject withdrew at the 5-hour period). At 5 1/4 h, the REM latency decreased significantly and there was a reduction in total REM sleep. In the 4-hour regime, REM latency was sharply reduced but, in spite of this, total REM time was sharply curtailed. The amount of stage 4 sleep was generally undisturbed. Performance and personality measures were not strikingly affected.

In a recent study by our laboratory [Webb and Agnew, in press] 15 of 16 initial subjects maintained a 5-hour sleep regime for 8 weeks. REM latency was sharply reduced compared to baseline. In spite of this there was a 20-percent reduction in absolute REM time. This reduction was present across the 8 weeks. Slow-wave sleep showed an increment in absolute amount in the first week about 15% above baseline with a gradual drift across weeks to the baseline amounts. We also found no dramatic changes in performance and no changes in personality functioning as displayed on objective personality tests. Our one positive finding was a decline in vigilance detections on a 1/2-hour vigilance task. This was accompanied by a decline in 'attempts' and hence may have been a motivational rather than a capacity change.

In summary, the reduction of the typical sleep amount will result in a change in the kind of sleep obtained; primarily a reduction of REM sleep. There are strong suggestions that reductions in the 4-hour range and below, limit the capacity to maintain performance on vigilance tasks. However, it does appear that more limited chronic PSD regimes may be maintained voluntarily without dramatic personality or performance variations. This certainly appears to be true for reduction of sleep to approximately 5 h.