'resting state' of subjects could be found. It should be recalled that when a sleep-deprived subject is resting or carrying out a task of low interest value with little or no motivation, physiological measures, such as heart rate and respiration rate, muscle tension, skin conductance were decreased below baseline. This suggested a deactivation of the ANS. However, in a highly motivated situation with the subject performing an interesting test, high levels of ANS measures were obtained. This would indicate a heightened arousal. Such relatively uniform patterns of either activation or deactivation of the ANS, however, are lost in a more prolonged SD. While the autonomic functions are coupled during wakefulness and during short SD, they become uncoupled during prolonged vigil. Naitoh et al. [1971] observed slowing of the heart rate and lower oral temperature (indicating deactivation) for up to 120 h of vigil; at the same time skin conductance was increased (indicating activation). With continuation of the vigil past 120 h many physiological parameters switched to an activated pattern. These included oral temperature, skin temperature, heart rate, EEG a-time, steroid output and subjective improvement [Naitoh et al., 1971; Johnson et al., 1965]. The autonomic arousal resulting from prolonged SD stands in contrast to a depressed CNS as judged from the decreased EEG a-time and behavior.

Sleep: Physiology, Biochemistry, Psychology, Pharmacology, Clinical Implications.
1st Europ. Congr. Sleep Res., Basel 1972, pp. 174-176 (Karger, Basel 1973)

Biochemical Effects of Sleep Deprivation in Rats¹

J. F. Pujol

Lyon

Dr. Pujol started by pointing out that the first working concept of his group was rather naïve. Using the neurophysiological model established by Jouvet and his coworkers, they modified paradoxical sleep and studied the

¹ An in extenso paper was not submitted. The following summary of Dr. Pujol's presentation was prepared by D. M. Loew, Basel.

Pujol Biochemical Effects of Sleep Deprivation in Rats 175

biochemical consequences of such manipulations. In particular, they were

interested in the effects on the noradrenergic, dopaminergic and serotonergic systems not the least because very much is known about these systems and their relevance to sleep. The method used in the present study was the so-called flower-pot method² for sleep deprivation. Thanks to EEG control (which, Dr. Pujol believes, is extremely important) it was found first that using different diameters of the flower-pots (4.5 versus 11.5 cm) quite different results are obtained. With 4.5-cm flower-pots total paradoxical sleep deprivation was obtained, whereas with the 11.5-cm flower-pots there was no total deprivation of paradoxical sleep. Thus, 11.5-cm pots were used for control studies.

During rapid eye movement (REM) rebound there was a significant increase of synthesis of noradrenaline as compared to those animals which remained under the deprivation condition. This increase of synthesis of noradrenaline was most pronounced in the mesencephalic structures. Furthermore, during rebound there was an increased disappearance of noradrenaline.

The serotonergic system was studied under identical experimental conditions using tagged tryptophan. During the initial period of 55 min after a deprivation of 96 h there was an increased capture of tryptophan, which was associated with an increase of radioactive serotonin in all the structures studied, most pronounced in the medulla oblongata. Evidently, deprivation stimulates synthesis in the serotonin (5HT) terminals. The comparison of deprived animals which were not allowed to recuperate showed that the disappearance rate of serotonin was higher in the former groups.

These in vivo effects were confirmed in an additional series of in vitro experiments.

The interpretation of these observations is still very difficult because, as already pointed out, it is hard to establish a specific functional deprivation of paradoxical sleep. Also, other results such as lesions of the raphe nuclei or the depletion of 5HT by the use of p-chlorophenylalanine show that there are specific changes in sleep related to serotonin and noradrenaline. Recently it was found that after lesions in the dorsal (NA-) fasciculus there is a marked increase of paradoxical sleep which lasts about 4 days : about 60 % of the recording time are occupied by paradoxical sleep.

² This well-established method for REM sleep deprivation involves placing experimental animals on inverted flower pots in a water tank. The animals are deprived of REM sleep because of their reluctance to fall into the water.

Symposia: D. Effects of Sleep Deprivation 176

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.

Sleep: Physiology, Biochemistry, Psychology, Pharmacology, Clinical Implications.
1st Europ. Congr. Sleep Res., Basel 1972, pp. 176-184 (Karger, Basel 1973)

Selective and Partial Deprivation of Sleep

Wilse B. Webb

Gainesville, Fla.

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