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Feinberg I, Koresko R L & Heller N. EEG sleep patterns as a function of normal and pathological aging in man. *J. Psychiat. Res.* 5:107-44, 1967.
[Clinical Neuropharmacology Research Center, National Institute of Mental Health, Saint Elizabeths Hospital, Washington, DC and Department of Psychiatry, Downstate Medical Center, State University of New York, Brooklyn, NY]

This study documented electroencephalogram sleep patterns and their relationship to cognitive impairment in the normal and senile elderly. Several of the observations and hypotheses have since become the focus of renewed interest. [The *SCI*® and the *SSCI*® indicate that this paper has been cited in over 245 publications.]

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When I wrote this paper in 1967, I was working in the Section on Neurophysiology (headed by the late Edward V. Evarts) of Seymour Kety's Laboratory of Clinical Science at the National Institute of Mental Health. It was my first professional position, and I could not have been more fortunate. Evarts became and remained one of my closest friends and, throughout my career, was a role model for scientific integrity and creativity. Although best known for his fundamental studies on the control of movement,[1] Evarts had a deep interest in the problem of consciousness, and there was no aspect of my research that did not benefit from discussion with him. Kety was a remarkable lab chief; his scientific judgment and unselfish support created an environment that contributed to many careers, including that of Nobel Prize winner Julius Axelrod.

Most of my studies on sleep use human disorders to investigate basic as well as clinical issues. Here, with my research assistant (now PhD), Richard Koresko, and Naomi Heller, who managed the patients clinically, I selected senile elderly subjects to test the hypothesis that rapid eye movement (REM) sleep reflects the operation of memory mechanisms. Electroencephalogram (EEG) and eye movement were recorded for five nights in 15 normal and 15 senile elderly subjects; memory and other cognitive functions were tested independently.

This study is frequently cited probably because it outlined the main changes in sleep patterns that occur with age; between young adulthood and normal old age, sleep becomes increasingly interrupted by waking, and the amount of deep sleep and sleep spindles diminishes markedly. Senile subjects show a more extreme form of sleep fragmentation but do not differ from controls in deep sleep or spindle density. In both groups, subjects with "younger" sleep patterns, both REM and non-REM, showed significantly better cognitive function.

Several issues mentioned in this work remain of current interest. We included results on sleep EEG in young adults and children to expand the description of sleep ontogeny. We hypothesized that the enormous change in sleep patterns over adolescence underlies a decline in brain plasticity. I remained preoccupied with this question and later summarized evidence indicating that a major reorganization of the human brain takes place during adolescence, noting that a defect in this process might cause mental illness, especially schizophrenia.[2] In 1967 we noted the similarity of the ontogenetic curves for total sleep time and waking cerebral metabolic rate ($CMRO_2$); recent evidence[3] suggests that deep (slow-wave) sleep and $CMRO_2$ are closely correlated over the first two decades of life,[4] a relationship that may depend upon parallel changes found in cortical synaptic density.[5] Three of the 15 senile subjects originally studied repeatedly awoke from REM sleep in confusional states. This phenomenon, which was recently confirmed,[6] may cause the nocturnal wandering that often necessitates hospitalization in senile patients.

I continue to study the intimate relationship of sleep to age in humans because it holds clues to two basic problems: the biological function(s) of sleep and the physiology of brain aging.

1. **Evarts E, Shinoda Y & Wise S.** *Neurophysiological approaches to higher brain functions.* New York: Wiley, 1984. 198 p.
2. **Feinberg I.** Schizophrenia: caused by a fault in programmed synaptic elimination during adolescence? *J. Psychiat. Res.* 17:319-34, 1982/83.
3. **Chugani H T, Phelps M E & Mazziotta J C.** Local cerebral metabolic rates for glucose (LCMRglc) during human brain development studied with 2-deoxy-2[18F] fluoro-d-glucose (FDG) positron emission tomography (PET). *Soc. Neurosci. Abstr.* 12:1232, 1986.
4. **Feinberg I.** Letter to editor. (Adolescence and mental illness.) *Science* 236:507-8, 1987.
5. **Huttenlocher P R.** Synaptic density in human frontal cortex—developmental changes and effects of aging. *Brain Res.* 163:195-205, 1979. (Cited 45 times.)
6. **Schenck C H, Bundlie S R, Patterson A L & Mahowland M W.** Rapid eye movement sleep behavior disorder. *JAMA—J. Am. Med. Assn.* 257:1786-9, 1987.

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