

July 26, 1965

Richard P. White, Ph.D.
Associate Professor of Pharmacology
Department of Pharmacology
University of Tennessee
Memphis, Tennessee 38103

Dear Dick:

I am most grateful for your careful review of the report on cholinergic mechanisms in induced convulsions. Your comments are helpful and the cited references augmented my review.

You suggest that this data indicates epilepsy to have a different biochemical substrate than induced convulsions and trauma, and I would concur. The data is not so clear, however, and further study will be needed to clear up this point. For my purposes, I am more interested in the data that implicates acetylcholine in all three processes, and in their similarities than their differences. The reference to Aird and seizures is only partly true. Trypan red experiments, in seizures and in myopathies, were therapeutic failures and soon interest waned. In an early study in my residency we administered trypan red to a group of neurological subjects, without any clinical effects (except the most startling skin colors!).

I was most interested in your reference to Malhotra and Pundlik, which suggests yet another mechanism whereby elevated CSF and tissue acetylcholine can affect cellular activity and behavior. I didn't know their work.

At one time I collected the data relating to other catechol amines and induced convulsions. The data is sparse and not convincing. I elected, therefore, to focus on cholinergic mechanisms, as these provide a more reasonable view of the process.

Parenthetically, I want to congratulate you on your latest article in the EEG Journal. It is an excellent piece of work, and while the answers are not as simple as we would like the nervous system to be, the data and figures are convincing. It is the clearest evidence that the CNS evoked response follows different pharmacological laws than spontaneous cortical activity.

July 26, 1965

In my readings recently I came across the Australian studies of Curtis and Eccles (e.g., Pharmacology of Cholinergic and Adrenergic Transmission, ed Koelle, G. B. et al, McMillan, 1965) which clearly show that different cells and cell complexes can be stimulated or inhibited by acetylcholine, and that these differences may be basic to the differences in spontaneous and evoked responses. (Our own evoked response studies are much less elegant, and we have recently embarked on a study of simultaneous stimuli and the evoked response.)

Again many thanks for your kindness in reviewing my report. I hope we can arrange to meet together this fall.

Sincerely yours,

Max Fink, M.D.
Professor of Psychiatry

MF:jm

MAX FINK, M.D.

A graduate of the New York University College of Medicine in 1945, Dr. Fink obtained extensive clinical training in neurology and psychiatry from 1946 to 1952. After qualifying as a certified specialist in both disciplines, he accepted the position of Director of Research in the Department of Experimental Psychiatry at the Hillside Hospital in New York.

In that position, for ten years, he developed an extended research program studying the neurophysiological, psychological, social and clinical effects of the various somatic treatments in psychiatry.

In 1962 he was invited by the Governor of Missouri to establish the Research and Training Center at the Missouri Institute of Psychiatry, a part of the Division of Mental Diseases of the State of Missouri. Concurrently, he was appointed Research Professor of Psychiatry at Washington University.

In 1960, after attending the first Computer Conference on EEG Methods at U.C.L.A., he was stimulated to study the applications of digital computers to the field of specialization, EEG and Psychopharmacology. When the opportunity to develop the Missouri Institute of Psychiatry was given him, he created a Digital Computer Research Unit specifically designed to satisfy the special studies which seemed important in psychiatric research. The present computer unit at the Missouri Institute of Psychiatry is the outgrowth of those interests.