Conditional Reflexes and Sensory-evoked Epilepsy:
The Nature of the Therapeutic Process*

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Abstract—Studies of conditional reflex therapy for patients with sensory-evoked epilepsy are reviewed and experimental studies in animals reported. The behavioral methods employed in the therapy are reviewed. They are of a conditioning nature and are defined as clinical therapeutic conditioning.

Sensory-evoked epilepsy can be induced in animals by conditioning techniques, provided the animal brain has been rendered epileptogenic. Sensory-evoked epilepsy in patients can be made worse by conditioning, and can be changed from one type to another. However, sensory-evoked seizures in humans are not the result of conditional reflexes.

The possibility that sensory-evoked epilepsy in animals and humans might be a manifestation of conditional reflexes has often been considered. A recent volume edited by Servit (1963) considers the problem in detail, with particular emphasis on audiogenic seizures in animals. Critchley (1937) was the first to consider that musicogenic epilepsy in the human might represent a conditional reflex, but after careful consideration discarded the possibility. Shaw and Hill (1947) again raised the possibility.

The possible role of conditional reflexes in sensory-evoked epilepsy in animals and in humans has also been considered in a series of studies at the Epilepsy Center of the University of Wisconsin during the past eight years. (Forster et al., 1963; Forster and Chun [in preparation]; Forster and Campos, 1964; Forster, Ptacek et al., 1964; Forster et al., 1965; Booker et al., 1965; Forster, Klove et al., 1965a and b; Forster et al., 1967; Forster, 1967; Forster, 1966; Forster, Cleland et al. [in press]; Forster and Booker, 1968). This paper reviews and analyzes these and other studies.

Induction of Reflex Epilepsy in Experimental Animals by Conditioning

Forster et al. (1963) presented the results of conditioning studies using implanted electrodes in cats with intact brains. The US (electrical stimulation to the motor cortex of one hemisphere) produced focal motor seizures on the opposite side of the body. The CS (sound or intermittent light) was administered for a 4-second interval alone and then continued through a 2-second period coupled with the US. After a series of trials, usually not fewer than 25 nor more than 50, the animals developed behavioral and EEG changes upon presentation of the conditioning stimulus.

The behavioral responses consisted of changes in posture preparatory to the onset of the focal seizures. The exact nature of the preparatory response depended on the part of the body involved in the focal seizure and the personality of the cat. If the cat was docile, it tended to crouch, pull in its extremities, wrap its tail about itself and wait patiently. More aggressive cats would rise up on all fours and stand with their legs in a fixed position. If the hind leg was involved in the seizure, the animals tended to stand. Failure to present the US after conditioning resulted in surprise, and the animal would turn its head to look at the member of the body ordinarily involved in the focal seizure.

The brain-wave changes consisted of a generalization of the primary cortical evoked potential—e.g., the auditory-evoked spike occurring with the presentation of a sound (CS) at first appeared only in acoustic cortex but, as the conditioning process proceeded, could be elicited from all areas of cortex, even on bipolar recordings from the motor cortex. This generalization of evoked potential was followed by desynchronization and flattening of the brain waves from all areas until cessation of the conditioning stimulus. In cats with intact cerebral cortices, no seizure could be elicited by the CS alone. Both behavioral and EEG changes induced by conditioning could be removed by extinction, i.e., repeated presentation of the conditioning stimulus without reinforcement.

From these studies we concluded that, while it is possible by conditioning to produce behavioral and EEG changes, it is not possible to evoke seizures as a CR in cats with intact brains.

Studies were then undertaken to render the brain epileptogenic and to repeat these experiments (Forster et al., in preparation). We felt, however, that if the destructive and epileptogenic lesion were induced at the time of the original surgery, it would interfere with the conditioning process and, likewise, a second anesthesia and surgical procedure after the conditioning had been established.