Subclinical Epileptic Seizures

Impairment of Motor Performance and Derivative Difficulties

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• **Modern clinical observations have greatly expanded the conception of the characteristics of the various kinds of epilepsy. By simultaneously recording electroencephalograms and the performance of simple motor tasks, it has been possible to demonstrate the effects of epileptic seizures not detectable by unaided observation and not noted by the patient. The effects of these subclinical seizures have been manifested variously—by a lengthening of the time between stimulus and reaction, by inaccuracies of response to stimuli, or by total cessation of performance.**

From this study it is suggested that subclinical seizures probably play a role in producing some of the psychiatric conditions associated with the convulsive disorders, as well as primary behavior disturbances and undifferentiated mental deficiency. It is also suggested that such subclinical seizures may possibly contribute to the characteristics of some cases of criminality and antisocial reactions and schizophrenic reactions.

THE PURPOSE OF THIS PAPER is to present evidence that subclinical epileptic seizures may impair simple motor performance and to point out possible relationships of such impairment to more complex performance and ultimately to disturbed clinical behavior.

It is now well established that in focal seizures every gradation of impairment of consciousness may occur, from none at all to total loss of awareness. Symptoms of seizures are myriad and may include an almost limitless variety of involuntary motor acts, false sensory experiences and affective changes. In conjunction with the method used in the present study, it may be well to note at this point that, in some partial seizures, awareness of various sensory stimuli continues but the capacity to respond to them is lost. For example, it is not unusual for some patients with seizures, especially of the temporal lobe variety, to recall that they have been able to hear and understand the speech of others after they had lost the ability to respond to it.

By studies reported in 1941 and 1947, Schwab[18,19] opened new ways to study the effects of seizures on individuals. He tested the ability of patients with petit mal seizures to make motor responses to auditory and visual stimuli during attacks. Stimuli and responses were recorded simultaneously with the electroencephalograph so that the temporal relationship of each to the others could be ascertained. It was found that always if there was any response at all to a stimulus given during a seizure burst, the reaction time—that is, the interval between the stimulus and the response—was longer than it was to the same stimuli at other times. Degrees of prolongation varied roughly in proportion to the length of the seizure bursts as seen in the electroencephalograph; and often with the longer bursts there was no response to the stimuli. Schwab concluded that he had demonstrated six degrees of impairment of consciousness as reflected in the various degrees of prolongation of reaction times. Although Schwab did not say so, it would appear from the brevity of some of the bursts noted and the minimal effect they had upon the reaction time, that many of the seizures recorded would not have been detectable by ordinary clinical observations.

In 1953, Bates[1] reported the use of an apparently independent but similar technique in the study of a single case of petit mal epilepsy. By making synchronous recordings of electroencephalographic and sound impulses while the patient was reading, he demonstrated various degrees of impaired consciousness.

In the present study an apparatus like Schwab's was used, and the technique was somewhat similar. However, the effects of subclinical seizures upon spontaneously sustained performance were noted, as well as responses to stimuli. The present study also dealt with seizures other than petit mal lapses. Moreover, subclinical seizures were correlated with alterations of performance other than and in addition to the prolongation of reaction time. A final point on which the present study differed from Schwab's was that attention was given to the psychiatric implications of the effects of such seizures.

Figure 1 illustrates the technique employed in this study. The patient was prepared for a routine electroencephalograph which was recorded on the channels shown. A small magnet was attached to one

Presented before the Section on Psychiatry and Neurology at the 85th Annual Session of the California Medical Association, Los Angeles, April 29 to May 2, 1956.

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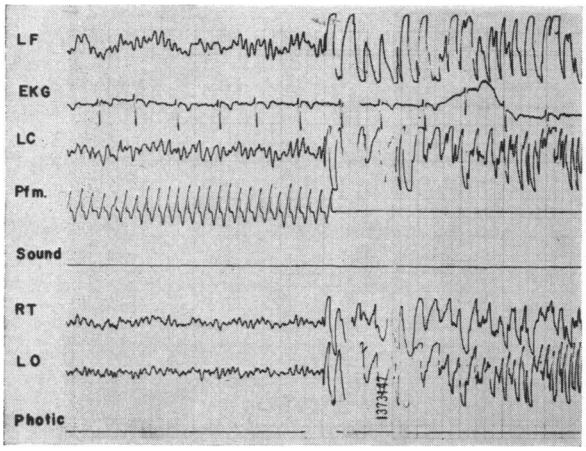

Figure 1.—Cessation of performance with grand mal seizure.

finger and a stationary induction coil was placed just in front of the magnet so that movements of the finger set up potentials in the coil, which were recorded on one channel of the electroencephalograph.* The patient was instructed to tap his finger at any rate he chose. As can be seen, the task was being performed steadily and rhythmically until shortly after the onset of an extremely high amplitude paroxysmal discharge, like that of grand mal seizure, which was inadvertently precipitated by intravenous injection of metrazol. When this occurred, finger movement stopped entirely.

Similarly, Figure 2 shows a total cessation of performance during a petit mal lapse. In this record the patient was responding to sound stimuli recorded by way of a microphone on another channel of the electroencephalograph.†

Figure 3 presents the first example of what is here referred to as subclinical seizures. The patient was a 15-year-old boy with a diagnosis of "primary behavior reaction." He has never been known to have had any form of seizure. Symptoms had consisted essentially of shy, withdrawn behavior and difficulty in learning. The figure shows a total cessation of spontaneously sustained performance associated with a burst of paroxysmal theta activity on the electroencephalograph. It will be noted that at the termination of the paroxysm, performance was resumed. The patient was unaware that he had stopped. This patient showed numerous such bursts with impaired performance, but at no time was there a clinically observable seizure.

The present study grew out of an interest in the high incidence of abnormal electroencephalographic tracing, particularly the paroxysmal type, in children and adolescents with clinical evidence of a primary behavior disorder. That such electroencephalographic abnormalities are common is well supported by a number of studies,[2,3,10,14] beginning with that of Jasper, Solomon and Bradley[12] in 1938, although not every such study has demonstrated similar findings.[5] The relationship, however, of the electrical changes to disturbances of behavior is not known. That is to say, even after the discovery of an abnormality in

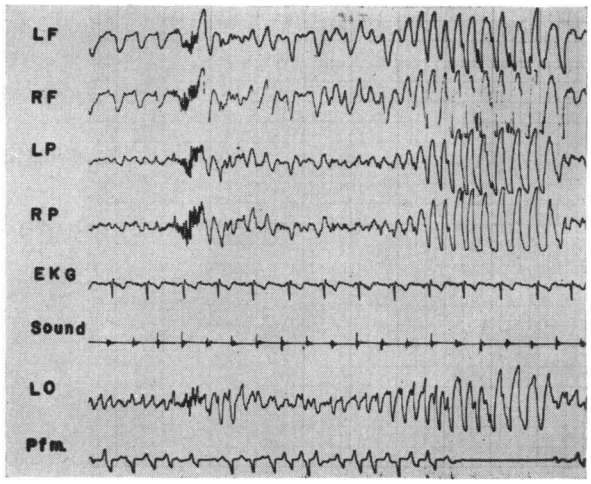

Figure 2.—Cessation of performance with petit mal seizure.

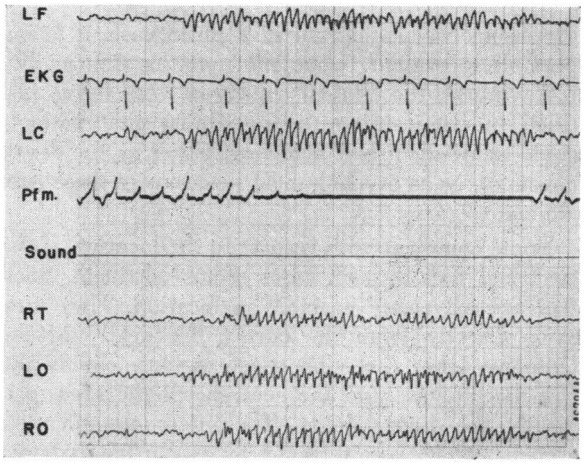

Figure 3.—Cessation of performance without clinical seizure.

*Reference may be made to a technical note made by Lipton[15] for a detailed description of this portion of the apparatus.

†When light was used as a stimulus it was recorded by means of a photoelectric cell through a channel of the electroencephalograph.

KEY TO ABBREVIATIONS ON MARGINS OF ILLUSTRATIONS

LF = Left frontal
RF = Right frontal
LC = Left central
RC = Right central
LT = Left temporal
RT = Right temporal
LO = Left occipital
RO = Right occipital
LP = Left parietal
RP = Right parietal
Photic = Stimulus by light
Sound = Stimulus by sound
Pfm = Performance

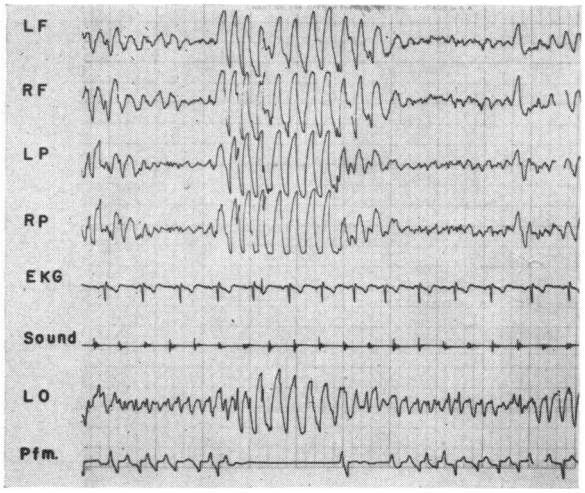

Figure 4.—Cessation of performance without clinical seizure.

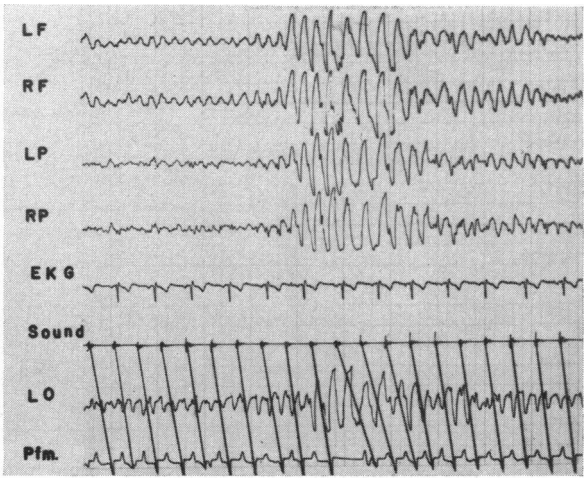

Figure 5.—Delay in response to stimuli.

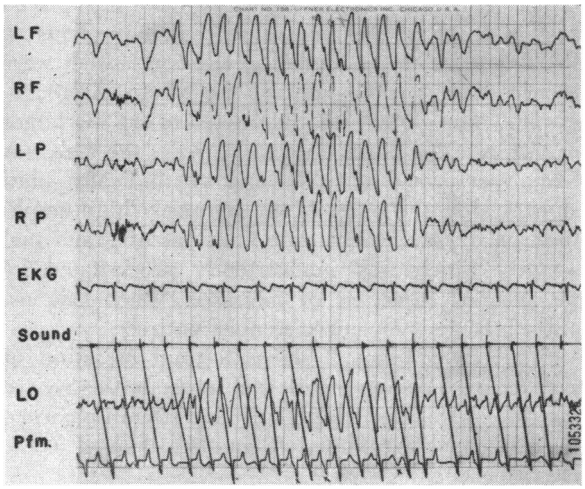

Figure 6.—Performance in excess of number of stimuli.

the electroencephalogram in a given case, it is impossible to evaluate what influence the abnormality is having on the clinical condition. One seems justified in entertaining the possibility that patients could show disturbed behavior, either as the direct effects of, or as psychological reactions to these subclinical seizures.

Some comment with respect to the location of the electrical foci in such cases seems pertinent. Such foci are extremely variable in location. They may arise directly from the cortex, frequently of the temporal lobe; the subcortical regions, chiefly the rhinencephalon; and the centrencephalic area, primarily the diencephalon. The intercommunicating systems which are referred to as cortico-thalamic sectors are significant in the mediation of the clinical manifestations of such seizures. In the case of the 15-year-old boy previously discussed, it was not possible from the record obtained to definitely localize the discharge more than to say that the paroxysmal bursts were of the type that are often associated with psychomotor automatisms, many of which are centrencephalic in origin.

In connection with one of these systems, namely the temporo-thalamic sector, it may be recalled that Hughlings Jackson recognized that uncinate fits frequently had affective components. More recently the report by Davidoff,[4] among others, showed that affectively charged ideas can occur as substantially the only symptoms in some temporal lobe seizures. Gibbs and co-workers[8,9] emphasized that the incidence of various psychiatric syndromes, including psychoses, psychoneuroses and severe personality disturbances is very high in persons with temporal lobe epilepsy. They also noted that the longer the interval between seizures the greater the emotional disturbances were likely to be. Conversely, the occurrence of frequent seizures tended to be accompanied by a reduction in the emotional disturbances. These observations were strongly supported by the studies of Ostow,[16] of Scott and Masland[20] and of Ervin and co-workers[6] (the last mentioned of whom included especially thorough psychological and psychiatric evaluations of their patients).

Until an adequate series is studied, it remains only a conjecture, but still a good possibility, that subclinical seizures in a patient of this type may be directly affecting the apparatus of emotions which the work of Penfield[21] showed to be activated by electrical stimulation of diseased temporal lobe cortices.

Figure 4 shows total cessation of performance associated with a paroxysmal burst not accompanied by a seizure detectable by ordinary observation. This graph was taken from the electroencephalogram of a young woman who had frequent clinical petit mal seizures.

Figure 5 shows a frequently encountered change in performance associated with paroxysmal bursts. Lines have been drawn between the stimulus and response pips.‡ In this case a delay in response (prolonged reaction time of Schwab) resulted, although the patient continued accurately to give one response to each stimulus.

Figure 6 shows still another type of change in performance often seen with a subclinical seizure. Again lines have been drawn from the stimulus to the response pips. During a burst, three responses appeared in excess of the number of stimuli, whereas, in the absence of the paroxysmal activity, responses were accurate.

Figure 7 is shown to illustrate the relative accuracy of response which some individuals maintain during highly abnormal electroencephalographic activity. The patient was a 16-year-old boy who had idiopathic grand mal and petit mal seizures and whose electroencephalographic record shows the sort of severely abnormal activity evident in the figure shown, virtually all the time. Again lines have been drawn between the stimulus pips and response pips and although the rate of the stimulation was being varied at random, the patient responded to all but two stimuli. During this time the electroencephalogram was severely abnormal, showing high amplitude, slow, as well as spike and slow wave activity. It will be noted that at the point at which he missed two responses, the spiking activity had become predominant, which is in accord with an observation made by Schwab. It is interesting to speculate upon the possibility that over the long course of his severe disturbance the patient had learned to partially compensate for the accompanying impairment of function so that by the time the tracing shown in Figure 7 was made, he could perform accurately in the presence of bursts which earlier would have disabled him.

Although this patient showed largely accurate responses in this particular illustration, at other times he showed marked impairment of performance with subclinical seizures. In connection with this observation a segment of his history is pertinent. He had seemed normal and vigorous both mentally and physically until the onset of his convulsive disorder four years previously. Shortly after the onset of the illness, his intelligence quotient was 98. After four years of frequent convulsions, his intelligence quotient was 48 and his intellectual functioning was at the moron level. There is no doubt that Gastaut[7] expressed a current and widely accepted point of view when he remarked that impairment of intellectual functions in epilepsy results only from the brain lesions causing the epilepsy and not from the seizures per se. Idiopathic epilepsy is held never to cause such impairment. It occurs to the authors of this paper that the patient here under consideration had "deteriorated," probably on the basis of frequent—almost constant—subclinical seizures.* We cannot assert positively, of course, that he had no structural brain disorder, but careful clinical investigation, including pneumoencephalograms, failed to reveal any. It is thus suggested that in this patient deterioration was an ictus phenomenon consisting chiefly of subclinical seizures.

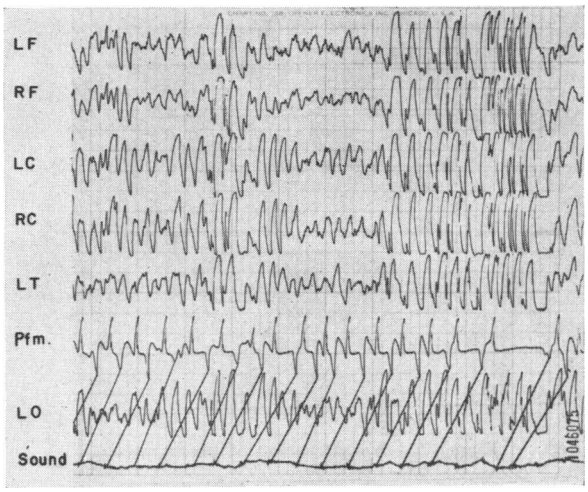

Figure 7.—Performance during constant paroxysmal activity.

The term *deterioration* is used in one sense as meaning organic degeneration, which probably does not occur in a case of this type. In another sense, apparent deterioration may be conceived of as occurring in either or both of two ways. The first would result from the taking over of facilitated pathways by a discharge which introduces patterns of positive but distorted perceptions through which memory patterns, which are not related to afferent stimuli, are called up. Examples of distortion of this kind are "forced thinking," delusional or hallucinatory experiences, anxieties, hysterical manifestations, or even schizophrenic reactions.

The second way "deterioration" might occur is by the interruption of normally functioning memory or thought events by the paroxysmal discharges which interfere with the natural continuity of thought sequences. The type of thought distortion will depend upon the area of interruption. In the light of this concept, one might conjecture that the level of intellectual functioning could be noticeably reduced by the simple but repetitive interruption of logical, unified thought sequences essential to reasoning. The intellectual deterioration noted in the present case could be explained in this way.

‡The term *pips* is applied to the pinnacles or nadirs in tracings which indicate the extremes of action that is being recorded.

*He has clinical seizures too, but no one has ever contended that intellectual functioning was unimpaired during clinical seizures of a major type.

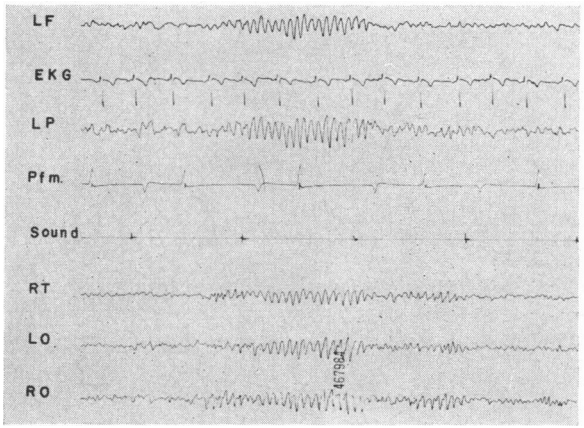

Figure 8.—No alteration of performance.

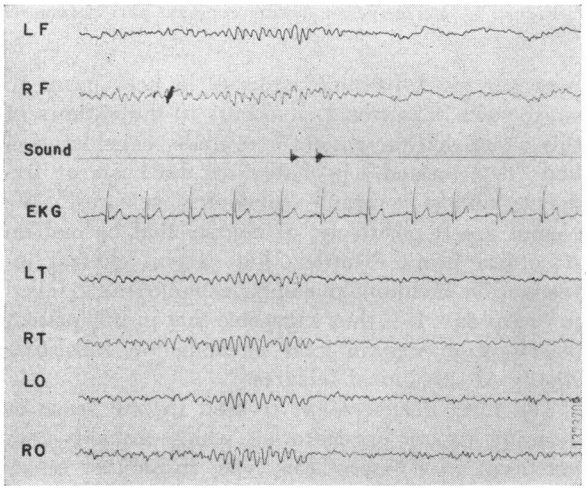

Figure 9.—Suppression of paroxysmal burst.

Figure 8 is taken from the record of the same patient as Figure 3 and is presented to show that sometimes (but uncommonly) no change in performance is detected even with the appearance of a clear-cut paroxysmal burst. In this case two stimuli occurred during the burst and no alteration of response was noted. It seems reasonable to expect that by varying the complexity of the performance response, precise measurements of degrees of impairment might be made. It appears probable that delays in responses and inaccurate (missed and extra) responses are owing to intermediate degrees of impairment between none at all and impairment so severe as to cause total cessation of performance. Specifically, it should be expected that with lesser degrees of impairment more complex responses could be executed without error, and with greater impairment more errors would be evident with increasing complexity of the performance task. Thus it is possible that this patient, who performed well as tested, might have shown impairment if he had been responding to more rapid stimulation or, for example, had been asked to respond to alternate stimuli rather than to consecutive ones.

Conversely, some preservation of the ability to do deliberate acts might possibly be demonstrated in some of the patients who showed complete cessation of response, provided that an even simpler test than movement of a finger could be devised. Finally, in order to test more fully the total reaction or response of a person, the simple motor output must be equated with functional disturbances that appear in response to discharges in other areas of the brain which have only remote influence or none at all upon the motor systems. Further research is being directed toward such problems.

Figure 9 is shown simply to illustrate one of the technical difficulties which may occur in testing of this type. It shows a paroxysm being suppressed in response to auditory stimulation. Such suppression as this has been described previously by Schwab and others.

There are certain limitations to this method. As was just shown, it cannot be used to study the effects of activity which it suppresses. Application to extremely brief, transient activity is also difficult. Finally, of course, it can be used only with patients able and willing to cooperate.

Another limiting factor is that only motor responses are recorded and, as suggested earlier, ability to make such responses may not be impaired unless some influence is brought to bear upon the motor systems. This method does not test for all possible effects of subclinical seizures and some may take forms which cannot be studied in this way. In those cases, other techniques for studying cerebral dysfunction will have to be devised.

Three other clinical groups for which we do not have illustrative material should be mentioned. As recently shown by Walter, Yeager and Rubin,[21] paroxysmal activity is found in an enormously high proportion of patients with undifferentiated mental deficiency. Hill[11] and others have shown similar but less pronounced changes in psychopathic and criminal persons. Much less decided changes have been reported by a number of investigators, most recently Kennard,[13] in schizophrenia. The possibility that subclinical seizures contribute to the impaired intellectual functioning of such mental defectives is certainly an excellent one, and some role in the other clinical groups mentioned seems possible.

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