For over 50 years we clinicians have administered electroconvulsive therapy with little to guide us in deciding whether or not a particular induced seizure is an effective treatment. At first we thought that piloerection or pupillary dilatation predicted the efficacy of a seizure, but these signs were difficult to assess and were never subjected to controlled experiments.

The duration of the motor seizure was examined next, and in evaluations of the seizures in unilateral and bilateral ECT, it seemed reasonable to opine that a minimum of 25 seconds defined a good seizure (Fink and Johnson, 1982). In studies of unilateral and bilateral ECT with threshold and suprathresh-old energy dosing, motor seizure durations were greater than 25 seconds, yet the threshold-unilateral condition yielded ineffective courses of treatment (Sackeim et al., 1993). Indeed, the new experience finds that longer seizures are not necessarily better for determining efficacy (Nobler et al., 1993; Krystal et al., 1995; McCall et al., 1995; Shapira et al., 1996). The occurrence of a prolonged, poorly developed, low-voltage seizure of indeterminate length and poor postictal suppression is a clear call for restimulation at a higher dose, with the expectation of inducing a shorter, better developed and clinically more effective seizure. The Seizure EEG

Modern brief pulse ECT devices provide the facility to monitor the seizure by an electroencephalogram, an electrocardiogram, and lately, an electromyogram. For a decade it has been feasible to examine the electrographic characteristics of the EEG seizure as well as its duration. The EEG usually develops patterned sequences consisting of high voltage sharp waves and spikes, followed by rhythmic slow waves that end abruptly in a well-defined endpoint. In some treatments, however, spike activity is poorly defined and the slow waves are irregular and not of particularly high voltage. It is also difficult to define the endpoint, with the record showing a waxing and waning period followed by an imprecise termination. Could these patterns be related to treatment efficacy? One suggestion was that bilaterally induced seizures were characterized by greater midseizure ictal amplitude in the two to five hertz frequency band than those induced by unilateral ECT (Krystal et al., 1993). Moreover, the seizures in bilateral ECT showed greater interhemispheric symmetry (coherence) during the seizure and more pronounced suppression (flattening) of EEG frequencies in the immediate postictal period. In other words, bilaterally induced seizures were more intense and more widely distributed throughout both hemispheres than seizures induced with unilateral stimulation.

The clinical relevance of these observations derives from the frequently reported therapeutic advantage of bilateral over unilateral ECT in the relief of depression (Abrams, 1986; Sackeim et al., 1993). The apparent validity of these observations led others to specifically examine the clinical predictive value of the described EEG patterns.

The EEG data of Nobler et al. (1993) came from studies of patients receiving either unilateral or bilateral ECT and energy stimulation either at threshold or two and one-half times threshold (Sackeim et al., 1993; 1996). The patients who received threshold unilateral ECT fared poorly compared to those who received bilateral ECT. Regardless of the electrode placement, however, those patients who exhibited greater midictal EEG slow-wave amplitude and greater postictal EEG suppression experienced greater clinical improvement and relief of depression (Nobler et al., 1993), confirming the observations by Krystal et al. (1993). Greater immediate post-stimulus and midictal EEG spectral amplitudes, greater immediate post-stimulus interhemispheric coherence and greater postictal suppression were reported with higher dose stimuli (two and one-half times threshold) compared to barely suprathreshold stimuli (Krystal et al., 1995). In another study, clinical improvement in depression correlated best with evidence for an immediate postictal reduction both
in EEG amplitude and coherence (Krystal et al., 1996). These analyses of the seizure EEG show promise of defining a clinically effective seizure. The available brief pulse ECT devices allow visual examination of the seizure record so that we can estimate the presence and duration of spike activity and the development of rhythmic high voltage slow wave activity, measure the duration of total seizure activity, and evaluate the endpoint of the fit (precise or imprecise).

In recent research studies, the methods of EEG analysis have been complex. Investigators often use sophisticated multichannel instrumentation recorders and EEG-analytic computer systems that are not usually available in clinical settings, but their elegant findings are consistent with the visual observations of the records provided by clinical ECT devices. **EEG Seizure Measurement**

ECT device manufacturers have sought some quantification of the EEG changes. The clinical Thymatron DGx device made by Somatics Inc. provides three quantitative measures of the seizure EEG: seizure energy index (integration of total energy of the seizure), postictal suppression index (degree of suppression at end of the seizure) and endpoint concordance index (a measure of the relation of the endpoints of the EMG and the EEG seizure determinations when simultaneously recorded).

In 1997, Somatics introduced a proprietary computer-assisted EEG analysis system for use with their ECT device to obtain the EEG power spectral and coherence analytic measures for routine clinical use.

In their new Spectrum 5000Q device, the Mecta Corporation makes available the EEG algorithms derived from research by Krystal and Weiner (1994) and licensed from Duke University to assist clinicians in better determining the quality and efficacy of individual seizures. The clinical significance of these measures has not been prospectively examined, yet the measures provide accessible quantitative indices of the seizure EEG which hold the promise of clinical application and provide the means for establishing their validity (Kellner and Fink, 1996). For immediate application, clinicians can visually examine the available EEG outputs for evidence of good seizure intensity and generalization. The present criteria for an effective seizure include a synchronous, well-developed, symmetrical ictal structure with high amplitude relative to baseline; a distinct spike and slow wave midictal phase; pronounced postictal suppression; and a substantial tachycardia response. These are reasonable criteria based on present experience. Another measure, that of interhemispheric coherence (symmetry), can be roughly estimated visually from a two-channel EEG recording when care is taken to position the recording electrodes symmetrically over both hemispheres.

Examples of inadequate and adequate seizures are shown in Figures 1, 2a and 2b. These samples are derived from an ongoing study involving energy dosing estimates in the first treatment of a 69-year-old man with recurrent major depression. In the first two stimulations, 10% (50 millicoulombs) and 20% (100 millicoulombs) energies were applied. In the third application, 40% (201 millicoulombs) energy was applied. Electrode placement was bilateral. **Interseizure EEG**

In patients receiving a course of ECT, EEG recordings made in the days after treatments showed profound and persistent effects. With repeated seizures, the EEG showed a progressive increase in amplitudes, a slowing and greater rhythmicity of frequencies, and the development of burst patterns. These changes in EEG characteristics were related to the number of treatments, their frequency, type of energy and electrical dosage, clinical diagnosis, patient age and clinical outcome (Fink and Kahn, 1957).

The improvement in patient behavior from the Fink and Kahn (1957) study (observed as a decrease in psychosis, lifting of depressed mood and decrease in psychomotor agitation) was associated with the development of high degrees of EEG change. The EEG characteristics predicted which patients had improved and which had not.

The association was quantitatively the greater the degree of slowing of EEG frequencies and the earlier that "high degree" slowing appeared, the earlier and more dramatic was the change in behavior. Elderly patients developed EEG changes early while younger adults were often slow in showing the changes. In some patients the EEG did not slow despite many treatments, except when the treatments were given more frequently during the week.

The association between ECT-induced interictal EEG slowing and improvement in depression was confirmed by Sackeim et al. (1996). EEG records were examined at different times during the treatment course in 62 depressed patients who received either unilateral or bilateral ECT at threshold or high-dose energies. ECT produced a marked short-term increase in delta and theta power, the former of which resulted from effective forms of ECT. The changes in the EEG were no longer present at two-month follow-up. The authors concluded that the induction of EEG slow-wave activity in the prefrontal cortex was tied to the efficacy of ECT.
An important clinical application of EEG methodology is in determining the adequacy of a course of ECT. When a clinical change does not occur in a timely fashion, the interseizure EEG can be examined visually or by computer analysis. Failure of the EEG from the frontal leads to show well-defined delta and theta activity after several treatments suggests that the individual treatments were inadequate. At such times, the treatment technique should be reexamined for adequacy (i.e., sufficient electrical dosage, choice of electrode placement, concurrent drug use), or the frequency of the treatments should be increased. If the patient fails to improve despite apparently sufficient EEG slowing, the diagnosis and treatment plan should be reexamined.

The renewed interest in the seizure EEG as a marker of seizure adequacy, and in the interseizure EEG as a marker of ECT course adequacy is likely to underlie the next phase of research into the physiology of ECT.

References:


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