

# Brain Electrical Activity Mapping in Treatment Resistant Schizophrenics

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## Introduction

Quantitative studies of EEG and evoked response, including spectral analysis and topographic mapping, are becoming increasingly popular in clinical practice. Recent studies have shown the techniques to be useful in cerebrovascular disease (Ahn et al, 1987; Nuwer et al, 1987), epilepsy (Kowell et al, 1987; Lombroso and Duffy, 1982; Nuwer, 1988), mass lesions (Nagata et al, 1985), and head injuries (Houshmand, et al, 1987). Perhaps the most important application has been in documenting organicity in patients with psychiatric syndromes or learning disabilities (Duffy, 1985, 1986a,b).

Brain electrical activity mapping (BEAM) has also demonstrated subtle neurological abnormalities in patients with schizophrenia (Daniels et al, 1988; Stoudemire et al, 1983; Morstyn et al, 1983; Karson et al, 1987), depression (in press), Parkinsons and Alzheimers (Duffy et al, 1984), toxic exposures (Bemad, 1989; Knoll et al, 1984), and more. Brain mapping is also useful in analyzing and assessing the function of drugs which are given to patients (Saletu et al, 1987). In many situations the quantitative/topographic approach may be more sensitive than conventional test modalities, such as standard EEG, CT scanning, MRI, and neuropsychiatric testing (Duffy et al, 1988; Fisch et al, 1988; Nuwer, 1988). Moreover, brain mapping may also be useful in monitoring neurosurgery and cardiovascular surgery patients (John et al, 1989a,b).

Treatment resistant schizophrenia is a very difficult problem that faces the clinician. It has been defined as failing as many as four different anti-psychotics. Others say a treatment resistant schizophrenic is one who just failed Haldol and Prolixin. Numerous studies have been written

about individual schizophrenics who do not respond to conventional anti-psychotic drugs.

I commonly see these type of patients in the clinical setting. Particularly in association with a clinic called Earth House in Belle Meade, New Jersey. I have treated dozens of treatment resistant schizophrenics based on reports of benefits of various drugs in this patient population, i.e. Reser-pine, Xanax, Prednisone, Clozapine, Lithium, and B-Blockers. None of these drugs have been particularly satisfactory, many other reports suggest that the anticonvulsants such as carbamazepine, divalproex sodium, and phenytoin may be particularly useful. BEAM provides clues as to why anti-convulsants can treat some symptoms of schizophrenia. Already many reports of anti-convulsants useful in schizophrenia have been identified (McElroy et al, 1989; Sramek et al, 1988; Dose et al, 1987; Hakola et al, 1982; Kalinowsky et al, 1943; Kidron et al, 1985; Klein et al, 1984; Kubanek et al, 1946; Lautin et al, 1980; Post et al, 1986; Puzynski et al, 1984; Stramek et al, 1988; Wolkowitz et al, 1988; Diamond et al, 1971; Haward 1969; Pinto et al, 1975; and Bellak, 1976).

## Methods and Results

I studied the BEAMs of twelve treatment resistant schizophrenic patients as defined above. Among these twelve it was particularly striking that only two of the twelve had an abnormal EEG with a mild generalized slowing and background disorganization probably due to medication effects. In essence all EEGs were normal. The spectral analysis which is often called a quantified EEG was abnormal in seven of the twelve patients. The cortical auditory evoked potentials (AER) were abnormal in eleven of the twelve. The cortical visual evoked potentials (VER) were abnormal in eleven of the twelve. This gave the first indication

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that these patients might have electrical or neurobiochemical abnormalities and that they might be more responsive to anticonvulsants than any other medication.

In the spectral analysis there were four abnormalities in theta, three individuals with abnormalities in delta, three with abnormalities in beta, and one with abnormalities in alpha. This is of particular interest since theta slowing and delta slowing are dominant. We know that anti-psychotics do increase the speed of delta and theta waves (Saletu et al, 1987).

In the AER five individuals had temporal lobe abnormalities, four individuals had parietal lobe, three had frontal lobe, and one occipital lobe involvement. Others had delayed wave forms throughout the entire brain region. Some so called schizophrenics probably have atypical temporal lobe epilepsy as a basis of these hallucinations.

#### Conclusion

In summary, treatment resistant schizophrenics have grossly abnormal brain maps, and may respond better to single and multiple anti-convulsants more than any other agent for this problem. All of these patients had at least some partial response to anti-convulsants with many having the best functions in years. Response of psychotic patients to anti-convulsants may be more widespread than previously thought. Treatment resistant schizophrenics probably have a higher frequency of anti-convulsant responsiveness.

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