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Neurotherapy and Clinical Science

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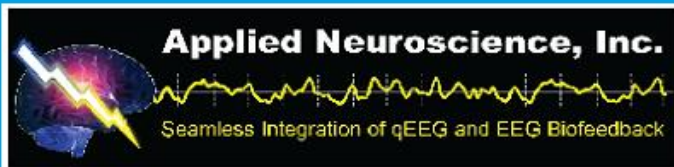
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Current Concepts In Neurotherapy: Neurotherapy and Clinical Science

T.J. La Vaque, Ph.D.

The use of operant procedures which utilize electrophysiological signals from the human brain for therapeutic purposes (“EEG biofeedback” or “neurotherapy”) has received increasing attention in recent years. It is generally assumed that “abnormal” electrophysiological values reflect underlying functional abnormalities in brain activity, and, further, those functional abnormalities are in turn responsible for the clinical presentation of emotional, cognitive, and/or behavioral pathology. It is also generally argued that “normalization” of the electrophysiological signals via operant procedures (“increase SMR”, “decrease theta”, etc.) occurs as a result of the “normalization” of underlying brain function, thus effectively correcting the pathological condition of concern. Since it is assumed that operant procedures which manipulate a particular electrophysiological brain signal will result in a correction or “normalization” of an abnormal functional brain state, a logical corollary to the argument would be that an associated change in the electrophysiological signal toward “normalcy” should be demonstrable.

It seems to be patently clear that, given the current state of technology, many of the assumptions cited above give rise to testable hypotheses, and further suggest clinical protocols and measures that serve to:

1. Document the existence of the abnormal electrophysiological activity and associated clinical pathology and,
2. Document the “normalization” of the electrophysiological signal consequent to neurotherapy with recovery from the clinical pathology for which treatment was sought.

If neurotherapy as an intervention is to gain wide acceptance in psychology and medicine, it will be mandatory to demonstrate that there exists a *necessary* relationship between the operant procedures, changes in the electrophysiological signal

in the reinforced direction, and changes in the clinical symptoms. Unless a necessary (not just sufficient) relationship between those variables can be established, multiple legitimate alternative explanations are still possible (placebo, spontaneous recovery, etc.) no matter how strongly the clinician may feel that the operant intervention was the necessary active component of the treatment. Mere correlation between treatment and clinical change is notoriously weak evidence for efficacy, and, from a scientific point of view, barely exceeds the status of anecdotal evidence (Bauer, 1994).

Some neurotherapy procedures have developed as a result of rationally developed hypotheses derived from empirical observation (Lubar & Shouse, 1979; Lubar, 1997; Lubar & Shouse, 1976; Lubar & Shouse, 1977; Rosenfeld, 1997; Sterman, 1984) while other treatment protocols seem to arise in the absence of clearly articulated theoretical underpinnings. There is, for instance, considerable traffic on the internet interest lists which recommend a broad variety of “treatment protocols” for which no substantive justification is presented and for which no data in support of the claims of efficacy is presented. With repetition, however, such communications create the façade of legitimate clinical science without ever having had the benefit of peer review.

A growing body of evidence suggests that there is an electrophysiological “signature” characteristic of attention deficit disorder (ADD) (Chabot, Merkin, Wood, Davenport, & Serfontein, 1996; Chabot & Serfontein, 1996; Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992; Monastra et al., 1999; Suffin & Emory, 1995) and depression (Rosenfeld, 1997; Rosenfeld, Baehr, & Baehr, 1995). Similarly, there is a growing number of published studies regarding the efficacy of EEG operant procedures for treating ADD (Linden, Habib, & Radojevic, 1996; Lubar, Swartwood, Swartwood, & O'Donnell, 1995b; Rossiter, 1998; Rossiter & La Vaque, 1995; Thompson &

Thompson, 1998). While those studies report significant improvement in behavioral and psychometric measures of ADD symptoms following neurotherapy, none provide pre- and post-treatment brainwave analyses showing an associated change in the presumed electrophysiological abnormality. One notable exception is that of Lubar's interesting report showing different operant learning EEG profiles for subjects who exhibit improvement in their ADD symptoms vs. those that did not (Lubar, Swartwood, Swartwood, & Timmermann, 1995a).

Technology which permits quantitative analysis of the EEG (quantitative EEG or qEEG) has the potential for contributing significantly to the development of neurotherapy as a mature clinical procedure based upon well established principles of clinical science. Quantitative EEG information can be an invaluable resource for the design and evaluation of neurotherapy and its efficacy (Thatcher, 1998). Other cutting edge technologies such as LORETA (Low Resolution Brain Electromagnetic Tomography) have the potential for powerful contributions to neurotherapy in coming years.

Quantitative analysis is certainly not new. Hans Berger's laboratory performed the first quantitative analysis of primitive EEG using Fourier analysis (Berger, 1932). Later workers developed automatic "frequency analyzers" for EEG (Gibbs & Grass, 1947; Walter, 1943), and the prototype of the modern topographic brainmap (the "toposcope") was demonstrated by Gray Walter in a popular science magazine in 1954 (Walter, 1954). Computer technology now permits us to perform complex mathematical and statistical analyses almost instantly. There are now over 36,000 journal articles utilizing qEEG analysis in behavioral, pharmacological, and neurological studies. One qEEG company (Neurometrics) recently received FDA approval of its software for the "post hoc analysis of EEG". The current technologies that permit noninvasive examination of brain function are being cross-validated (i.e., qEEG with functional magnetic resonance imaging—fMRI, and quantitative magnetic resonance imaging—qMRI, (Thatcher, Biver, McAlaster, Camacho, & Salazar, 1998; Thatcher, Hallet,

Zeffiro, John, & Huerta, 1994). In short, qEEG technology is readily available and is the method of choice by which brain electrophysiological activity can be analyzed and compared to known norms (John & Prichep, 1993; Thatcher, 1998). The same technology has produced discriminant function analysis for psychopathology, attention disorders, and closed head injury.

Thus, the natural wedding of neurotherapy with qEEG can permit documentation of abnormal electrophysiological activity prior to initiating neurotherapy, and should permit documentation of the "normalization" of the electrophysiological signal as a result of neurotherapy. These developments will provide significant impetus for the development of standard diagnostic procedures, commonly accepted treatment protocols, and a recognized method for analysis of the electrophysiological results of neurotherapy. In the absence of such rigorous clinical procedures, neurotherapy will have great difficulty in gaining broad recognition as a valid clinical activity.

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