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EEG Biofeedback: A Generalized Approach to Neuroregulation

By Siegfried Othmer, Susan F. Othmer, and David A. Kaiser

Overview

Many clinicians who have adopted EEG biofeedback are struck by the wide variety of clinical indications for which efficacy has been either observed directly, or claimed by others. This chapter presents a comprehensive overview of the current state of EEG biofeedback from the clinical perspective, but with an orientation toward model building. Specifically, the review covers the higher frequency training conventionally referred to as "SMR/beta" (nominally 12 to 19 Hz). Discussion of the lower frequency domain of "alpha/theta" (4 to 12 Hz), though of great interest as well, is left to others.

First, a conceptual model is proposed and discussed. Second, the research history of the field is drawn upon to illustrate the evolution of protocols and explain elements of the emerging model. Third, an overview of our clinical results is given that depicts the use of the proposed "generalized approach" for a number of mental disorders. These results were obtained with a relatively limited set of clinical protocols that evolved out of our extrapolation of new methods from the original research. From these results emerges a need to explain how such broad efficacy can be achieved. It is postulated that the EEG feedback technique not only promotes particular functional states of the brain, but more generally exercises neural mechanisms by which the fundamental functions of arousal, attention and affect are managed by the central nervous system (CNS). Rhythmicity in the EEG is seen as a key variable in the coordination of cortical activity, and clinical improvement is traceable to improved neuroregulation in those basic functions by appeal to their underlying rhythmic mechanisms. Current models of brain function are used to explain both the frequency and the spatial specificity of the EEG biofeedback process.

Introduction

EEG biofeedback is being established as a discipline at a time when psychiatry and psychology are caught up in a trend toward ever more refined differential diagnosis. At the same time, there is an intensive search for the physiological and even the genetic basis of behavior. This search focuses on a presumed structural basis for behavior to the exclusion of a more plastic, functional basis. A third characteristic of the age is a focus on the nervous system at the basic building block level of neurotransmitter function, receptor site properties, and membrane permeability; an orientation driven to a considerable extent by the needs of psychopharmacology. The connection between discrete, structural properties of the brain at the basic building block level and the higher order network or system properties of the functional level remains largely unexplored. In contrast, EEG biofeedback is a method by which the brain may be addressed directly at the functional or system level, and through the brain's ability to respond to operant conditioning of the EEG, the functional process may be altered and new patterns of behavior facilitated.

It will be demonstrated in the following that EEG biofeedback cuts across the bestiary of clinical diagnostic categories that has been devised over the last thirty years, demonstrating an ability to remediate a multiplicity of diagnoses with a limited set of protocols. It acts directly on underlying physiological mechanisms, and presupposes a considerable functional plasticity of the brain, a

concept that has only recently become a significant subject of inquiry within the research community. Such plasticity appears as "noise" in the search for the genetic basis of behavior, and as such has asserted itself primarily in its negative implications for such work. It will be argued that EEG biofeedback affects brain function at the network level, and a preoccupation with processes at the molecular, membrane, or even cellular level is not particularly illuminating for brain function at the higher levels. The implicit assumption of the bottom-up approach of the neurosciences seems to be that a viable conceptual model of the network cannot be constructed until we know the detailed workings of all the parts. Yet EEG biofeedback has proven to be a valuable clinical tool (as the data cited will show), and has stimulated the creation of a conceptual model based on such a top-down, systems approach.

EEG biofeedback can be best understood, and its relevant mechanisms discerned, by viewing the brain through the action of its web of inhibitory and excitatory feedback networks. Such networks require explicit mechanisms to manage them, integrate them, and assure their functional integrity. These networks must meet global stability criteria irrespective of what neurochemical implementation nature has, by evolutionary happenstance, devised. No doubt the technique impacts very specific neuromodulatory mechanisms, which remain undefined at this time. The clinical work can nevertheless proceed fruitfully on an empirical basis. Thus, EEG biofeedback is deemed to address itself to the core issue of control, with specificity at the network level, and yet with considerable generality in terms of clinical implications.

A Comprehensive Conceptual Model

In order for the field of EEG biofeedback to move forward and fulfill the promise that it has shown thus far, it is necessary to create a conceptual model that will explain the clinical results that have already been achieved in a way that will answer questions raised by skeptics, as well as facilitate a greater level of understanding and efficacy on the part of practitioners. The conceptual model presented here describes the characteristics of human neurophysiology upon which EEG biofeedback is based, how the process works, and why such wide-ranging efficacy can be gained by means of such a seemingly simple process.

Structure Versus Function

Before proceeding, it is necessary to clear some semantic underbrush: Though the process presented here is based on a "functional" approach, the hard distinction between structure and function survives in the tenacious tradition of the language of dualism. That is, structure and function are seen as the realization, if you will, of brain and mind, respectively. Every brain function, however, must have its structural underpinnings, so the more tangible distinction, and the one more accessible to experiment, is one based on the timescale of change and the ease with which change can be induced. Most of what we consider in terms of brain function involves typically rapid, transient changes in the electrical activity in the brain, activity which may leave little in terms of residual imprint. Most of what we consider in terms of brain structure is that which remains essentially unchanged over longer time constants. This is a continuum, and over much of the range in timescale, one can appropriately describe a phenomenon either in the vocabulary of structure or that of function. One analogy that comes to mind is the redefinition by David Bohm of a noun as a "slow verb".

Another way of looking at the structure/function duality is in the division between hardware and software in computers. On one hand, we have the true hardware, the semiconductor devices and ancillary items needed to service and operate them. On the other hand, we have the operating system software. Though this can be changed, it is generally modified only rarely and deliberately. At the next level is the applications software. A number of different modules may be drawn upon (brought to consciousness?) at a given moment, and there is in fact considerable "interaction" with the outside world which may make "functional" changes in the application software; and there may even be some adaptation to what the user typically wants. At the top level is the phenomenology of what is created with the applications software, which has typically

a very transient quality (e.g., imagery). One could argue that at each level we are dealing with physical electrons moving around from site to site (structure), but that would be cumbersome, and not really to the point. Similarly, one could talk about software failures in terms of "electron deficiencies" in certain memory locations. This is both true and absurd as a model for software failures. Every level has its appropriate terminology, referring progressively to structure, function, and objects (gestalts).

The categories distinguished here can find their analogues within the brain. However, the boundaries are not as discernible and the distinctions between structure and function even less definitive. Nevertheless, let us push the analogy forward a little further: A similarity can be drawn between our brain's neuromodulator systems and the operating system software of a computer. There is persistence in the workings of our neuromodulator systems that puts them on a different timescale than the applications software (which might involve the processing of a visual image, for example). Yet it would not be correct to regard the characteristics of a person's neuromodulator systems as immutable (even absent any drug intervention). Over time, it is clear that environmental influences, for example, can effect changes in neuromodulator function. A person may become more or less hypervigilant over time; he may become more depressed or anxious. He could also, however, achieve "spontaneous" recoveries from depression, which can be simply interpreted as autonomous normalization of neuromodulator functioning.

The distinction, therefore, between categories of structure and function is not based so much on issues of transience versus immutability, per se, but rather on a multiplicity of factors: the timescale of change; how matters have been historically viewed; and the level of abstraction which is appropriate to the discussion. This whole issue is currently very much in flux, and somewhat confused. We have, for example, the following from Michael S. Gazzaniga, director of the Center for Neuroscience at the University of California in Davis: "When someone remembers something, is there a structural-or discrete anatomical-change in neuronal synapses? Or is it functional change, which would simply reflect reprogramming of the pattern of neuronal discharges in the nervous system?" (Gazzaniga, 1995).

Here the posited "structural" change could equivalently be talked about in terms of function, and the posited "functional" change (which clearly must be sufficiently robust to persist long-term if it is to represent a memory) can be talked about in terms of structure (altered synaptic coupling strengths).

Brain Plasticity

If we are intent on maintaining the structure/function dichotomy, we are ineluctably in a semantic swamp. This is at least in part because the neurosciences are in the process of coming to terms with mounting evidence for what is collectively called "brain plasticity", and the old dualist terminology no longer serves us well. In its most general formulation, EEG biofeedback can be seen as the deliberate exploitation of 'functional brain plasticity'. More specifically, it depends upon plasticity in our neuromodulator systems. However, this concept is at best ambiguous, and a moving target. Simply put, brain plasticity refers to long- term alteration in brain systems that were historically thought to be static. Hence, the word tends to have a historical contextual reference, much like the word 'alternative health': once an intervention becomes mainstream, it is no longer "alternative". Similarly, once plasticity becomes accepted as an attribute of a particular brain system, the term tends to be discarded and future references may simply be to brain function. Hence the term brain plasticity tends to have only a transient utility, and to serve only

where the case for plasticity is still being made. However, making the case for EEG biofeedback on a model of brain plasticity may be the most accessible Ansatz.

To make the term maximally useful for our purposes, a review is in order. A remarkably prescient view of the model of brain plasticity is to be found in Brodal (1981, p. 259):

"Although our knowledge about the 'plasticity' of the nervous system is still in its beginnings, there is reason to believe that this plasticity is a general property of the central nervous system, and that it is a prerequisite for the capacity to learn (in general, be it motor patterns or pure intellectual capacities). Restitution after damage to the central nervous system may therefore in essence be likened to a learning process. Practical experience is in agreement with this."

In 1981, the evidence for brain plasticity was not being much attended to. Rather, the working assumption was that brain injury was relatively permanent after some period of "spontaneous remediation" lasting no more than 18 months. Hence, this reference to brain plasticity was unusual for the time. (It turns out that Brodal himself had previously suffered a head injury, and was probably speaking at least partly on the basis of his personal experience.)

A more modern view is summarized by Oliver Sacks in the popular book, An Anthropologist on Mars. (The views of neuroscientists are often more boldly expressed in their popular writings as opposed to their scientific ones, where they are compelled to be more reserved and circumspect.)

"Work in the last decade has shown how plastic the cerebral cortex is, and how the cerebral 'mapping' of body image, for example, may be drastically reorganized and revised, not only following injuries or immobilizations, but in consequence of the special use or disuse of individual parts. We know, for instance, that the constant use of one finger in Braille leads to a huge hypertrophy of that finger's representation in the cortex." (Sacks,1995, p.41)

Here the focus is on the long-term dendritic re-programming and/or regrowth, which has been shown to occur. However, there has still been little recognition of the obvious ability of the brain to accomplish significant reorganization on time scales much shorter than that of dendritic regrowth, which requires simply changes of state, and of regulatory function, that is, of functional rather than structural reorganization. This is now changing:

"Reorganization of somatic sensory receptive fields can appear within the dorsal column nuclei, the thalamus, and the cortex, within seconds of a peripheral manipulation. Similarly, motor cortical maps show dramatic shifts within hours of a peripheral nerve lesion or [within] minutes of a shift in arm configuration." (Donoghue, 1995)

When considering the reassignment of cortical neuronal resources within a time constant of seconds, one wonders if "plasticity" is the appropriate descriptor for the phenomenon. This is another case in point of the use of the term to describe as mutable something thought to be more permanently stable. If cortical resources can be so readily reassigned, then the mechanisms involved in stabilization must lie principally in the functional rather than structural realm. That is, there is less hard wiring than was thought! Thus we are likely to see the language of structure replaced over time by the language of function, and eventually we will see the disappearance of the term "plasticity" altogether in this connection.

With this in mind the term functional plasticity may be used to refer to all those processes by which brain functions thought to be relatively stable can be altered on a timescale short compared to that of dendritic regrowth, or the formation of new synaptic boutons. Functional plasticity is undoubtedly mediated, inter alia, via alteration of synaptic coupling strengths through the generation or attrition of receptor sites, and the alteration of neurotransmitter chemistry through changes in neuronal gene expression. The present interest will focus specifically on the neuromodulator systems and their regulation. Here the observed "functional plasticity" can have time constants short compared even to the above-postulated processes. For example, when we

are frightened, we are capable of changing our state of arousal within fractions of a second. The functional plasticity of neuromodulator systems clearly exists on all behaviorally relevant timescales. The claim of EEG biofeedback is that the dynamic range of neuromodulator system plasticity (flexibility) can be increased where it is deficient, and stabilized when it is unstable, by operant conditioning techniques.

Functional Plasticity: Implications of Recent Research

A number of developments over the past several years have prepared the ground for the claims we are now making for EEG biofeedback. First of all, the findings from functional magnetic resonance imaging (fMRI) are refocusing attention on collective neuronal activity; its time course, temporal interrelationships, and change as learning and habituation take place. Inevitably, these findings will raise questions about how such neuronal populations are organized and managed by the brain. Secondly, there is the ongoing research into the thalamocortical generation of rhythmic activity in the EEG by Mircea Steriade, David McCormick, and others. (Steriade, 1984; McCormick, 1990) Thirdly, there is the emerging interest in the binding problem, the mechanism for how the brain retains as a coherent phenomenon something that is parallel-processed at multiple neuronal sites (a visual image, for example, or a phoneme.) (von der Malsburg, 1995). We shall return to this critical theme below.

At another level, it may be said that much of psychopharmacology implicitly makes the case for the kind of functional plasticity required to explain the presumptive efficacy of EEG biofeedback. Quick-acting medications like stimulants can only operate by shifting the functional state of neuromodulator systems; there is no time for significant structural adaptation. The short-term effects of EEG biofeedback can be explained by similar shifts. The longer- acting medications such as anti-depressants and anti- psychotics work on the same timescale as the cumulative effects shown in many of the recoveries claimed for EEG biofeedback. The effect of Prozac administration, for example, can be discerned in the cerebrospinal fluid within hours, just as with stimulants, and yet its anti-depressant effects may take days or weeks to manifest. Such medications may work by means of longer-term adaptations that involve both functional and structural change. But it is not a large leap to argue that such changes can be induced over time by the challenge to the nervous system imposed by operant conditioning of the EEG. Both EEG biofeedback and pharmacological intervention can even be seen as a disequilibration of nervous system functioning to which the brain responds by long-term adaptation. In this view, their mode of action is seen to be uncannily similar. Regardless of whether or not this concept can survive further scrutiny, it is clear that the claims of EEG biofeedback are consistent with, and certainly not antithetical to, the implications of pharmacology.

Efficacy of pharmacology for a variety of psychiatric disorders is often taken to imply that such chemical intervention is absolutely required for remediation, by analogy to the provision of insulin in the case of Type I diabetes. That this is not the case is demonstrated by the efficacy of electro-convulsive shock therapy for depression. Here the remediation may be long-term even absent any long- term pharmacological support. Additionally, spontaneous recovery from episodes of both mania and deep depression is the rule, not the exception, in even mature cases of bipolar disorder. Clearly, these brains have quite functional states within their inventory. The question of efficacy of EEG biofeedback (for the vast majority of applications) is then reduced to the relatively minor issue of whether a change in functional state can be induced, or at least promoted, by operant conditioning of the EEG, and the second issue of whether such a training can have lasting effects.

In the case of pharmacology, the challenge to the nervous system is provided by neurochemicals or their metabolic precursors, or other metabolic agents, or factors which modulate receptor site sensitivity or ion channel permeability. In the case of EEG biofeedback, the challenge is to the means by which brain function is organized and maintained in the time domain, which is reflected

in the EEG. It will be argued in the following that neuromodulator systems function to organize both general organismic arousal and more localized activation of collective neuronal activity by modulation of rhythmicity. The EEG is preferentially sensitive to such collective, periodic, activity.

The Bio-electrical Domain: The Role of Periodicity and the EEG

We must pause in the chain of argument to admit to a degree of circularity: The normal EEG in an activated state has the appearance of a noisy signal devoid of any dominant frequency. Hence, it is not obviously rhythmic (periodic). Nevertheless, the frequency decomposition of this signal manifests the bursts of rhythmicity referred to. However, one could decompose any such noisy signal (the noise from a waterfall, for example) and obtain band-limited (frequency-decomposition) data looking much like the EEG, with similar bursts of rhythmicity. Hence, the physical reality that is ascribed to these rhythms must be based on more than the EEG signal itself. That is, looking through green sunglasses (band- limiting visual data) does not allow us to proclaim the world to be green. In the present context, the most persuasive argument for the physical reality of these rhythmic bursts comes from the fact that they appear to respond in a frequency-specific manner to EEG biofeedback training! However, we should not assume the answer in order to help us prove it.

Historically, the EEG was first studied with a focus on its most obvious feature, the alpha rhythm. We now associate a prominent alpha rhythm in occipital cortex with idleness of the visual system. Similarly, the sensorimotor rhythm (14 Hz [Hertz]) so prominent in the cat (or in Stage 2 sleep in humans) is associated with stillness of the motor system (Chase 1971). Inactivation is associated with increased rhythmicity (increased amplitude), as neuronal populations coalesce to collective firing under their mutual influence in the absence of independent sensory stimuli or other inputs. When activation levels are increased, due to stimulation or processing, these neuronal populations desynchronize, to a point at which rhythmicity may no longer be readily observable in the raw signal. Hence, the normal activated EEG is seen as the relatively desynchronized extrapolation of manifest rhythmic activity, which has a defined physiological function: maintaining a state of inactivity, or perhaps of readiness. A noisy (desynchronized) EEG arises then from the superposition of many rhythmic generators of different frequencies, each undergoing its own rapid ebbing and flowing from rhythmicity to desynchronization. When any one of these generators reaches the extreme of low activation, it may begin to dominate the EEG record.

Next, it is necessary to make the case that whatever role the specific EEG frequencies play in cortical regulation, that role is invariant over cortex. One of the notable features of the neocortex is that it is morphologically and histologically fairly homogeneous. Moreover, the same set of neuromodulators, by and large, subserve a variety of functional subsystems, and are not unique to any one of them. Similarly, the natural parsimony which prevails in nature makes it likely that the general role of rhythmicity in activation and time binding'whatever that role may be in detail'is probably uniform across cortical regions, varying only quantitatively over cortex, not qualitatively.

Hence, operant conditioning of the EEG rhythmic activity can be seen as a general appeal to brain regulatory function, as it is manifested in the cortical EEG. Depending on scalp location, one may expect some influence on the specific thalamocortical projections to that region, and to the specific functions subserved by that cortical region. Also, one expects some influence on the nonspecific thalamocortical projections, for a general effect on activation and physiological arousal. Whether the effect is more localized or more generalized has to be answered by a review of the data. It is already clear, however, that the EEG training cannot be specific to one neuromodulator system, as might be the case for some medications. Recent findings with

fluoxetine (Prozac) make it apparent that even medications which impinge directly upon one neuromodulator system (serotonin), are behaviorally non-specific in their effects! (Kramer, 1993) We therefore have every reason to suppose that EEG training affects and hopefully promotes fundamental brain regulatory integrity, and that behavioral or other improvements are simply evidence of the heightening of such self-regulatory performance.

The Specific Role of Rhythmicity in Neuroregulation

It has been argued above that in the extreme cases of EEG synchronization and desynchronization, an obvious correlation with low and high activation and arousal, respectively, exists. It is also well known that arousal correlates with dominant frequency in the EEG. It falls readily to hand to argue that the degree of rhythmicity, together with changes in the EEG frequency spectrum, manages the entire range of activation and arousal in the bio-electrical domain. The EEG, then, reflects a parameter that the brain tightly constrains in the ordinary course of events. An appeal to dominant frequency or to the amplitude at a given frequency by operant conditioning could therefore be expected to serve as a powerful external forcing function on the brain's management of arousal. The whole matter of the role of frequency, however, bears further discussion.

One role advocated for rhythmic activity is that of time binding, the need for harnessing brain electrical activity which is spatially distributed while maintaining it as a single entity. The need for this kind of function is apparent when it is recognized that visual processing, for example, must occur by parallel processing over large areas of cortical real estate. The integrity and stability of the image must be maintained over time. Simultaneity of firing of the various neurons participating in the mapping of an image may be the relevant criterion of "belonging". The transient organization of such distributed, correlated neuronal activity may be the role of the thalamocortical rhythmic generators. At the lower frequency regimes, say less than 30 Hz, this organization ranges broadly over the cortex, and manages activation and arousal with relatively long persistence. At higher frequency regimes, above 30 Hz, and peaking in the 40-60 Hz regimes, the brain manages specific cognitive processes that are of a more transient nature, and more spatially localized.

A recent study beautifully exhibits both of these roles of rhythmic activity (Munk, 1996). In this study, a visual image was moved across the visual cortex under two conditions: normal, and under electrical stimulation of the mesencephalon (brain stem region in which the nuclei reside which source the neuromodulator substances that control attention and arousal.) With stimulation, a global coherence became prominent in which the firing rates of neurons in different regions became more coincident. This coherence was observed over the region of visual cortex that was involved in mapping the moving image. If the moving image was then changed into two images, moving in opposite directions, the coherence was still present, but was restricted to the neurons belonging to each moving target. This beautiful experiment illustrates the influence of global activating mechanisms directed from the brainstem. However, this mechanism was not sufficient to guarantee time binding. That requires augmentation by information derived from the image itself, and processed 'locally' in cortex, in order to define the specific cohort to which each participating neuron belonged. This is a process of which the brainstem remains ignorant. Hence, time binding requires both brainstem and cortical governance, and both may be mediated by thalamo- cortical networks, and may also be modulated by direct cortical-cortical interaction.

It must be kept in mind that most of the signal processing we do in the brain involves very transient events taking place on small time scales. The analogy to dynamic RAMs or to the refresh on your computer screen (every 17 milliseconds) comes to mind. Further, it is apparent that the real information content in neural signals (action potentials) relates in first order (and trivially) to the presence or absence of a particular signal, and, more significantly, to the actual timing of the signal. The magnitude of an action potential is not a function of the size of the stimulus that gives

rise to it. Only the timing matters. And even the timing gains significance only in the context of other events. All "mental activity" must ultimately have its basis in particular neuronal firing patterns that become discernible from the ambient noise background by virtue of timing coincidences or at least correlations. It is this timing which appears to be managed by thalamocortical circuitry. Rhythmicity may be one of the key ways in which such timing is organized. Recent research by Pfurtscheller (1990) and Sterman (1996), show that the brain's ability to locally desynchronize in a timely manner defines its capacity to process the next stage of an ongoing task. The ability to resynchronize quickly allows it to reenter a state of readiness for the next task. The process breaks down when synchronization or desynchronization of specific frequencies persists or is disregulated, decoupled from the demands of the moment. EEG biofeedback is then to be seen as a challenge to the mechanisms that underlie the management of this rhythmic activity, and in application to neuromodulation of arousal and activation its natural domain is the frequency range less than thirty Hz. Training is similar to stimulation, and constitutes a push that invokes the brain's capacity for restoring homeostasis. Over the longer term, this results in a long-term increase in stability. Training at a specific frequency is then a push in a very specific direction, which can be chosen in light of specific arousal disregulation or attentional deficits found in each case.

The Placebo Argument

Does EEG biofeedback, with all its instruments, bells and whistles, include a huge "placebo" component (for which we are not entitled to claim credit)? The placebo argument sometimes serves as a talisman which the scientist, comfortable in his paradigm, may use to ward off disagreeable new claims. However, the placebo effect is no more than the body's means of mobilizing self-recovery. The placebo effect is not a cause. It is not itself a mechanism of recovery, but it does imply a mechanism'though one which may seem featureless and devoid of testable properties when looked at through the prevailing structuralist paradigm. Hence, it can provide no help to our understanding. But EEG biofeedback is by its very nature self-remediation. The part we are entitled to take credit for cannot be experimentally distinguished from "other" aspects of the self-healing process.

For researchers attempting to prove the efficacy of medication, self-recovery represents the counter-hypothesis, which is wrapped up in the concept of "placebo effect" and need not be further discussed. It is not of interest to the designer of drugs. When the discussion is about self-induced recovery (such as EEG biofeedback) and the mechanisms thereof, then we must openly address the placebo effect and ask whether its self-healing properties are any different from what we are claiming. It is a moot point. The existence of the placebo effect proves the existence of self-remediation. Self- remediation cannot then be disproved by invocation of the placebo effect. The existence of a robust placebo effect in medical and mental health disciplines supports the claims of EEG biofeedback. It does not undermine them.

Still, if one cannot in the individual case determine what part of recovery is due to the specific effects of EEG biofeedback training and what part is attributable to non- specific effects, can one be sure that the effects aren't all in the latter category? The normal resolution to this question is by means of statistics. In the case of EEG biofeedback, however, we are not constrained to rely exclusively on statistics (although the statistical argument is favorable as well), as there are other proofs of its efficacy.

The placebo effect, seen here as stalking horse for nonspecific effects of the EEG biofeedback process, is not the explanation for the efficacy claimed for the following reasons:

1. The effects of the training are highly specific to electrode placement and to training frequency band.
2. Training protocols exist which can commonly elicit effects opposite to those desired.
3. The effects of training with one protocol can be reversed with another.
4. The effect of the training is cumulative, rather than fading with time, as is common with placebos. If EEG biofeedback were to be explained in terms of placebo phenomena, it would be the first time that placebos are dose- dependent (i.e., cumulative).
5. Training effects are in line with research from neuropsychology regarding localization of function.
6. Populations can be moved to levels of performance which exceed those of na‹ve populations
7. The effects of the training often lie outside the range of expectations for spontaneous recovery or placebo effects, not only with respect to the magnitude of the changes elicited but also with respect to the consistency with which they are produced, and the timescale over which they occur. (Curiously, the more striking and unusual the claims for EEG biofeedback, the more strenuously is the placebo hypothesis invoked by critics!)
8. EEG biofeedback was discovered in connection with animal research. It may be assumed that the test animals were not subject to the placebo effect. Moreover, the researcher was blind, since the discovery was by way of serendipitous connection to an unrelated experiment (Sterman, 1976).

The spatial and frequency specificity of the EEG training, as well as its reversibility, allow every subject to be their own control in the training. This is not to say that conventional controlled studies are entirely superfluous. We are just at the beginning of the scientific inquiry into this technique, much of which will require controlled paradigms. Rather, we are asserting that the epistemological assumptions operative in the clinical setting are already sufficient to demonstrate efficacy in the case of EEG biofeedback because of the above-enumerated features of the training. In view of the above, then, the recoveries, remediations, and performance enhancements claimed for EEG biofeedback may be regarded on their own merits, and cannot be gainsaid either by placebo factors or by the argument that they are not individually supported by blinded controlled studies.

Another prevailing perception must be examined before proceeding with review of the protocols and the clinical data. It is often asserted that the EEG biofeedback "trainee" is actually training his own behavior, and that the changed EEG is simply a manifestation of that altered behavior. Behavioral state and the EEG are clearly coupled, and a conscious redirection of one's physiological state can obviously be helpful in achieving the objectives of the training in the moment. This is the dominant theme in conventional biofeedback, which is dependent upon a great deal of deliberate engagement in the process by the subject. This is not a necessary condition for EEG biofeedback training to succeed, and in this sense it departs fundamentally from conventional, peripheral biofeedback.

The successful training of cats, of very young children, and even of people in mild vegetative states, demonstrates that the training can proceed without the subject being particularly aware of their behavioral state, or intent upon altering it, or indeed very conscious about what is going on at all. The training in this case consists in operant conditioning of the EEG, neither more nor less. For example, in the use of EEG biofeedback for the remediation of epilepsy and stroke, it is not "behavior" in any conventional sense that is being trained. In fact, we have observed that people can respond quite counter to their own desires, expectations, and motivations; with the expected effects (and even some that weren't expected by either the client or the therapist) arising out of the particular protocol selected. The resulting behavioral state may be concordant with the

protocol selected, and quite at odds with the participant's conscious goals. Finally, there is the compelling observation that sleep EEG is changed subsequent to EEG training in the waking state. (Sterman, 1970) All these observations are evidence for the proposition that it is 'brain behavior" that is being trained directly. And brain behavior may be non-specific with respect to overt organismic behavior.

Research History: Implications for Mechanisms of EEG Biofeedback

If EEG biofeedback training is indeed capable of promoting self-healing, its role is that of facilitating a process of change the capacity for which already exists in the human brain. But how is it that such an apparently simple tool is capable of such wide-ranging effects? What is it about the brain that allows it to be led to more functional states? And how can the operant conditioning process embodied in EEG biofeedback be applied systematically and predictably, to good effect?
The implications of our clinical findings are that the EEG training is not narrowly specific in its clinical effects, but that it impacts very basic regulatory mechanisms, the disregulation of which is responsible for causing or at least maintaining the disorders discussed. In the following, the case will be further made for such a simple underlying model. A connection will be made to current models of brain function, and the central role of rhythmic brain activity will be discussed in explaining the remarkable breadth of efficacy of this emerging modality.
The early model of efficacy proposed by Sterman is that the EEG training at sensorimotor cortex lowers the setpoint of the gamma motor system reactivity (Howe, 1972). As a result, cortical hyperexcitability is reduced. This manifests in higher threshold of onset of seizures, most particularly in the case of motor seizures (Sterman, 1984). Lubar initially worked only with those hyperactive children who were Ritalin- responsive, on the assumption that these were the ones whose hyperactivity was grounded in underarousal (Shouse & Lubar, 1979). So the early work already presaged our current perspective, that the principal mechanism of action of EEG biofeedback is to normalize autonomous management of arousal and to enhance overall nervous system stability. The intimate relationship between seizure susceptibility and arousal makes it plausible that efficacy for seizures is also at least partly attributable to normalization of arousal regulation.
On the basis of the early work, it was close to hand to consider all the conditions being treated in terms of their arousal dimension, and in terms of the stability/instability continuum. **Table 1** shows a classification of conditions with respect to the arousal axis, and with respect to the instability axis. In preparation of Table 1 it became obvious that this system of categorization represents an oversimplification, although it does provide a useful perspective. It is, for example, an oversimplification to talk about depression and anxiety as separate and distinct entities. It is a further oversimplification to appear to reduce these to merely arousal disorders. It is perhaps better to identify these as correlations or covariations. Then again, arousal itself is not a unitary concept. Moreover, the arousal dimension is very important in the conditions we have listed as instabilities (as already mentioned for seizures). It is hoped that the Table will prove useful in illustrating the connection between various conditions at the process level, and indeed the mechanisms by which EEG biofeedback can impact them.

Table 1. Classification of Common Disorders in Terms of Arousal and Instability

Underarousal

Endogenous Unipolar or Reactive Depression
Attention Deficit Disorder: Inattentive Subtype
Chronic Pain (Low Pain Threshold)

Insomnia (Frequent Waking)

Overarousal

Anxiety Disorders
Sleep Onset Problems/Nightmares
Hypervigilance
Attention Deficit Disorder: Impulsive Subtype
Anger/Aggression
Agitated Depression
Chronic Nerve Pain
Spasticity

Underarousal/Overarousal

Anxiety and Depression
Attention Deficit Hyperactivity Disorder: Combined Type

Instabilities

Endogenous Vulnerability

Tics
Obsessive-Compulsive Disorder
Aggressive Behavior
Episodic Rage Disorder
Bruxism
Panic Attacks
Hot Flashes
Bipolar Disorder
Migraine Headaches
Narcolepsy
Epilepsy
Sleep Apnea
Vertigo
Tinnitus
Anorexia/Bulimia
Suicidal ideation and behavior
PMS
Multiple Chemical Sensitivities
Dysglycemia; Diabetes (Type II); Hypoglycemia
Explosive Behavior

Exogenous Vulnerability

Just as depression has its arousal dimension, it also has its attentional dimension, and its affective dimension. Similarly for the other conditions listed. For present purposes, it is sufficient to argue that these are coupled systems. One of the most obvious implications of the biofeedback work is that it is not possible to intervene unilaterally with the brain. Impinging upon the arousal axis has implications for attention and affect, and vice versa. Moreover, challenging the brain with biofeedback tends to move the brain toward stability. The observation was made decades ago by Elmer Green that biofeedback in general moves the organism toward homeostasis and toward stability. This has been abundantly confirmed in the present work. Having said this, it is also possible to drive the brain toward any instability that may exist, with a powerful technique such as this. Skillful clinical application is still required.

Instabilities can be characterized by the degree to which they arise autonomously within the CNS or require an external trigger for initiation. An internal vulnerability is referred to as endogenous, and an externally triggered vulnerability is referred to as exogenous. The relevant instabilities are distributed along a continuum in this regard, and a case can be made that there is a natural progression for different instabilities from the exogenous domain to the endogenous over the course of a lifetime. This is known as the kindling model, and it is particularly applicable to seizures, Tourette's syndrome, OCD, depression, anxiety and panic, bipolar disorder, and migraines. A crude attempt has been made at an ordering along the exogenous/ endogenous axis in Table 1.

Arousal, Attention, and Affect

The conceptualization of brain function in terms of coupled systems was broached by W.R.Hess (1954). Experiments with electrical stimulation of regions of the diencephalon (thalamus and hypothalamus) in some instances led to very specific behavioral responses, and in other instances led to broad overall changes in behavior: arousal, quiescence, somnolence, torpor, and sleep. Hess subsumed these global changes in sympathetic and parasympathetic arousal in the terms ergotropia and trophotropia. The 'ergotropic shift' is characterized by a tendency toward higher sensory acuity, external focus, sympathetic arousal, high motor setpoint, etc. The 'trophotropic shift' is characterized, in contrast, by a tendency toward a more inward focus, less alertness, reduced sensory acuity, a shift toward vegetative functions, and a reduced motor system readiness. It is clear from our work that invoking either of these two shifts is possible with EEG biofeedback. What we refer to as "beta" training (15 to 18 Hz) is to be identified with a global ergotropic shift in organismic function, and that of "SMR" training (12 to 15 Hz) is to be identified with a trophotropic shift. The response of an individual to even a single session of EEG biofeedback training can make this quite obvious, an assertion which is independent of any claims for long-term efficacy of training.

Long-term EEG training has the effect of exercising and expanding the brain's ability to move freely along the continuum of ergotropic or trophotropic dominance, with all its implications for arousal, attentional state, and affect regulation. This brain exercise moves the individual into regions where he or she may not heretofore have been able to reside comfortably or stably. This is made possible not only by increased flexibility of state, but by an increased ability to maintain overall nervous system stability. The reason that two primary training regimens (higher and lower frequency) are sufficient is attributable to the fact that the ergotropic shift and the trophotropic shift are mutually inhibitory. To enhance the one is to suppress the other, as was already apparent to Hess. Gellhorn (1967) originally referred to the dynamic balancing of the ergotropic and trophotropic domains in terms of 'tuning' of the nervous system. The EEG biofeedback, by explicit appeal to rhythmic mechanisms, may be seen as a particularly efficacious agency of 'nervous system tuning.'

The brain's intrinsic bias toward homeostasis dictates that any training which evokes a brain response away from its then- prevailing equilibrium state will set in train forces to restore the original state. Thus, promoting an ergotropic shift will in first order tend to produce such a shift, and on the other, set in train compensatory mechanisms by which the brain restores the state it had intended for itself. Hence, even dis-equilibration can bring about improved equilibrium maintenance as a long-term consequence.

Hemispheric Specificity of Training: Spatial Dependence of Protocols

The clinical data reviewed below are supportive of the view that the training exercises the two hemispheres specifically, and differentially. Cumulative clinical evidence in our offices has also reinforced the view that referential training near C3 and/or C4 is generally the most effective. Small displacements from these sites laterally from the midline along the coronal plane seem to have a minor effect on the training. Small displacements in the horizontal plane, on the other hand, change the quality of the training more significantly in our experience. Hence the training sites have been determined by a process of local optimization (i.e., small spatial displacments), rather than of global optimization. For some applications (principally to the instabilities), T3 and T4 have been found preferable to C3 and C4, respectively.

Other clinicians have reached different conclusions with regard to training sites. Tansey has consistently recommended midline placement because he intentionally trains the supplementary

motor area (Tansey, 1990). Lubar trains predominantly on the midline because he finds the greatest excesses there in terms of low-frequency EEG amplitudes in ADD children (Lubar, 1995). Such excesses are deemed to reflect underlying cortical disregulation. Mann and coworkers, on the other hand, established in one recent study that the EEG desynchronizes more at C3 and C4 than Cz upon a motor challenge (Mann et al, 1996). Since the training impacts directly upon the synchronization/ desynchronization dynamics, the case may be made on fundamental grounds that C3 and C4 should be preferable to Cz. This proposition has been abundantly supported in our practice.

With a large amount of clinical data at our disposal (several thousand cases), a picture has emerged that the EEG training addresses the specific failure modes of each hemisphere. If a particular disorder could be associated more directly with one hemisphere than the other, it might give us a clue as to what part of the brain might require redress. Such a connection would then imply a unique, differential protocol. We found this to be true, and a number of disorders began to yield to assignment to one hemisphere or the other. Then, using a process of "local optimization" both in terms of spatial location and selection of the reward frequency band, a training strategy emerged which has gained considerable 'stability' from the effort at continual refinement, and what may have started out as mere clinical impressions have gradually been reinforced to the point at which they now constitute a defensible training strategy. The principal hallmarks of the strategy are as follows:

1. There appears to be a certain simplicity and directness attached to training along the sensorimotor strip.
2. Training away from the midline appears to yield stronger and more hemisphere-specific training effects, than training at Cz.
3. There is a distinct predominance of the need for up- regulation of the left hemisphere, using beta training (nominally 15-18 Hz), and a corresponding predominance of the need for down-regulation of the right hemisphere using SMR- training (nominally 12-15 Hz). Frequently, the need for both exist within the same individual. (This frequency dependence is addresses further below.)

The apparent advantage of training at the sensorimotor strip for most of the conditions discussed is consistent with the early Sterman hypothesis, since amply validated, that what is being trained is the degree of rhythmicity of the thalamocortical regulatory circuitry. And whereas the rhythmic EEG activity observable anywhere on cortex is traceable to these thalamically-mediated regulatory functions, the primary sensory areas of cortex are perhaps the most direct access we have to them. Specifically, the highest cortico-thalamic fibre-density is to be found in the primary sensory areas of cortex (and also in projections to the frontal lobe). Historically, most of the EEG biofeedback training that has been done has focused on the primary sensory regions.

Our continuing observation over a large clinical population of the need for up-regulation of the left hemisphere and down- regulation of the right can be explained in terms of the specific way in which the two hemispheres fail, or disregulate. The work of Malone, Kershner, and Swanson, et al, (1994), provides us with a detailed neurophysiological model which explains this hemispheric laterality in training effect. In this model, it is proposed that the left hemisphere (in collaboration with the frontal lobe) manages tonic activation for the conduct of intellectual and motor tasks, and for the maintenance of vigilance over time. This activity is preferentially under the management of the neuromodulators dopamine and to a certain extent acetylcholine. The right hemisphere, by contrast, manages phasic arousal for maintenance of sensory system readiness to receive and process new inputs from any source. This system is predominantly under the management of norepinephrine and to a certain extent serotonin.

The model, as applied to ADD, which will be discussed further in the coverage of our clinical outcomes, reveals ADD to be a problem of underactivation of the left hemisphere, principally involving dopamine, and of overarousal of the right hemisphere, principally involving norepinephrine. Hence, neither the sequential processing of intellectual or motor tasks, nor the deployment of resources responding to new incoming stimuli are well managed. The efficacy of Ritalin is attributed to a dual influence, the up-regulation of the dopamine system and the down-regulation of the norepinephrine system. In a kind of parallel or equivalent model, ADD of the inattentive subtype is addressed with higher frequency left hemisphere training (central and possibly frontal) and ADD of the impulsive subtype is addressed with lower frequency training of the right hemisphere (central and possibly the parietal region as well). A mutual consistency thus emerges between the claims of EEG biofeedback and psychopharmacology for ADD. The Tucker and Williamson (1984) model lays a credible foundation for the general claim that the two hemispheres need to be specifically and differentially addressed in the training, just as they are pharmacologically. Recent clinical work has led to further refinements of the principal protocols so that they now incorporate frontal and parietal training with bipolar placements that combine left central with prefrontal sites (e.g., C3-Fpz), and right central and parietal sites (e.g., C4-Pz).
These latter refinements specifically challenge communication loops between the selected sites. When a bipolar montage is used, then the reinforcement promotes an anti-phase relationship between the two sites. This may be counter- intuitive. It has been shown (Rappelsberger, 1994) that when distant cortical locations communicate with one another, they come into greater synchronization in the process. Why then would one wish to train these sites to reduce the prevailing degree of synchrony? The only justification that really counts is that this has been found effective empirically. The theoretical justification is to be found in the 'regulatory challenge' model of EEG biofeedback. The biofeedback reinforcement takes the brain momentarily out of its prevailing equilibrium, to which it then wishes to return. It may not matter in first order whether the disequilibration occurs in one direction or the other. Improved regulatory function may eventuate in either case.
It may now be possible to generalize the Malone model to other conditions. Just as there are left hemisphere and right hemisphere aspects of ADD, the same may hold for affective disorders of depression and anxiety (Goodwin,1990). The left hemisphere aspects of depression and anxiety may have to do with anticipatory activity, planning, ruminating, perseverating, worrying. The right hemisphere, by contrast, may harbor the non-rational, more catastrophic aspects of depression and anxiety, namely fear, panic, agitated depression, and suicidality (Heller, 1997). With a spatially localizable technique at our disposal, hemispheric specificities have been confirmed with EEG training not only for ADD, cognitive function, anxiety, and depression, but also for pain syndromes, sleep disorders, eating disorders, endocrine and immune system disorders. Laterality turns out to be one of the key organizing principles for the evolution of protocols.

The Protocols' Frequency Dependence

Protocols used for EEG biofeedback training of the 12-19 Hz band, are essentially derived from Sterman's seminal work with seizures. The 12-19 Hz region was originally identified as being prominent in the bursts of sensorimotor rhythm of the cat (Sterman, 1969). Subsequently, operant conditioning of the cat EEG was restricted to the peak frequency range of this distribution, 12-15 Hz (Sterman, 1970). As additional work was undertaken with human subjects, the 15-18Hz band was also investigated in one study (Sterman, 1978). In the following, we will

refer to training with the lower frequency (12-15 Hz) and higher frequency (15-18 Hz) bands. The lower frequency training has also been colloquially referred to as "SMR" training, for historical reasons, and the higher frequency as "beta" training. These terms have become commonplace through clinical usage, even though we are dealing with only a subset of the entire beta band, which extends from 12 or 13 Hz to 35 Hz.

As we entered the field in 1985, we were aware only of the work of Barry Sterman, Joel Lubar et al., Michael Tansey, and Margaret Ayers with respect to the beta/SMR training. Joel Lubar et al. utilized both bands in the treatment of ADD (Lubar, 1984). Michael Tansey restricted himself to rewarding the frequency region centered on 14 Hz (Tansey, 1990), and Margaret Ayers used almost exclusively beta training (Ayers, 1993). In terms of electrode placement, Lubar et al. were typically using left-side training with bipolar placement near the sensorimotor strip, not deviating far from what Sterman had originally employed (C3-T3). Tansey exclusively used an electrode placement on the supplementary motor area, with a large-area contact that covered the space between Cz forward toward Fz, and also extending partially toward Pz. Margaret Ayers used C3-T3 placement almost exclusively, except when either symptomatology or EEG phenomenology indicated a need for right-side training at C4-T4.

All of the above protocols were accompanied by inhibition of low frequency activity, typically 4-7 Hz (called "theta" in the following). In the case of Michael Tansey, the information regarding excessive theta amplitudes was verbally communicated to the client. Additionally, Sterman and Lubar provided for inhibition of high-frequency activity in the region above 20 Hz.

Out of the work of these four pioneers, our protocols evolved in several stages. First, placement was changed from bipolar to referential to the ipsilateral ear, in line with a general trend within the field toward referential montage. Secondly, Cz placement was evaluated for the low frequency training on the basis of Tansey's work. For more than a year, most of the training was conducted at either C3 with the higher frequency band ('beta'), or at Cz with the lower frequency band (SMR), using an A1 reference. Excursions to C4 were, if needed, based on our early understanding of issues of laterality or in cases of localization of deficits to the right side (as in seizure disorders, head injury, and stroke). Over time, as we became more experienced and our understanding of the hemispheric specificity of certain aspects of cortical disregulation became clearer, it was observed that the C4 training was typically most effective with the lower frequency training, and that often stronger, more specific results were obtained than at Cz. Eventually, the predominant protocols became C3-beta and C4-SMR. Some frontal and parietal training was used as well to address specific issues.

Though early protocol selection was based upon the prior research work, it soon became necessary to devise methods of assessment (to be discussed later in this piece) that would assist us in teasing out which of these protocols were most appropriate for the client. But if the judgment turned out to be mistaken, then there was always the option to make an early change in protocol. If the choice was appropriate, then a different protocol might be used later to address residual issues.

It was observed also that if one persisted with the use of a single protocol, then eventually certain adverse symptoms could develop which called for compensatory training. Thus, with left-side training, ultimately client reports might indicate the need for right-side training, and vice-versa. Subsequently, more refined clinical skills led to an earlier integration of the secondary protocol into the training for optimization. This compensatory training led to the appreciation that in addition to addressing the specific failure modes of each hemisphere we really had to also achieve, or maintain, hemispheric balance. Symptoms could often be attributed to the inappropriate inhibition of one hemisphere by the other, or inappropriate disinhibition. This was

most directly demonstrated when a left-side seizure focus was also favorably influenced by training the contralateral placement. But the principle has proved to be valid broadly.
At the present stage of evolution of protocols, there has effectively been an integration of the C3-beta and C4-SMR protocols, which are both used with the majority of clients, generally within the same session, and the balance between them is titrated on the basis of symptom response. Assessment is then a matter of determining the client's physiological response characteristics, and the particular vulnerabilities expressed in their symptoms. In this appraisal, established clinical categories (from the DSM-IV) are only approximate guideposts. Whether diagnostic criteria are met in one respect or another is therefore irrelevant to the clinical burden. At least 80% of clients have been treated with this combination of protocols and this combination alone. The data reported in the following were obtained over the past eight years with the above protocols or derivations therefrom.

Clinical Evidence: Validating the Model

Clinical application is both the source and the destination of the theories and models proposed above. Without the surprises and inventiveness inherent in daily clinical practice, progress toward a comprehensive model for EEG biofeedback training would have been much slower, and the scope much narrower. By its very nature a research orientation must make certain choices and assumptions, and hold certain procedures invariant throughout the project. This does not allow for such a variety of approaches to be tried in such a short time. Yet, due to the volume of clients we were able to see since 1988, we have achieved significant depth of experience in a number of areas. It is now our goal to share this experience widely in order to allow it to be integrated with other approaches and perspectives, and subjected to more rigorous scientific evaluation and critique.
The list of conditions for which clinical efficacy of EEG biofeedback has been observed is given in **Table 2**, along with the nature of the qualifying evidence (controlled studies; published outcome studies; single case studies and conference presentations). Key references are indicated separately at the end of the chapter. The number of subjects that fall into each category are estimated as well. No systematic inquiry was under taken to flesh out this table, so we don't claim that it is complete. All entries relate only to data of which we have become aware through various means, and are therefore a lower limit in each case. In our own work, and that of our affiliates, we have acquired confirming evidence for all of the conditions listed, with the exception of Lyme disease.

Table 2. EEG Biofeedback Studies

CONTROL	OUTCOME	CASE HISTORY
ADHD		
Linden, Habib, & Radojevic (1996) Rossiter & LaVaque (1995) *Nash & Shakelford (1995)* *Cartozzo, Jacobs, & Gervirtz (1995)*	*Kaiser (1998)* *Kaiser & Othmer (1997)* *Thompson & Thompson (1997)* Lubar, Swartwood, Swartwood, & O'Donnell (1995) *Scheinbaum, Newton, Zecker, &*	Kotwal, Burns, & Montgomery (1996) Tansey & Bruner (1983)

	Rosenfeld (1995) Fenger (1995) *Toomin, Ibric, & Othmer (1994)* *Samples (1994)* Tansey (1991) Lubar (1985) Lubar & Lubar (1984) Shouse & Lubar (1979)	

LEARNING DISABILITIES

Linden, Habib, & Radojevic (1996)	Tansey, Tansey, & Tachiki (1994) Tansey (1991) Tansey (1990) Tansey (1985) Tansey (1984) Cunningham, & Murphy (1981)	*Kade (1995)* Tansey (1993)

DEVELOPMENTAL DELAY

		Fleischman (1997)

AUTISM

		Sichel, Fehmi, & Goldstein (1995) *Cowan (1994)*

TOURETTE'S SYNDROME

		Tansey (1986)

EPILEPSY

Lantz & Sterman (1988) Lubar, Shabsin et al (1981) Sterman & MacDonald (1978) Lubar & Bahler (1976) Seifert, & Lubar (1975)	Hansen, Trudeau, & Grace (1996) Andrews, & Schonfeld (1992) Tozzo, Elfner, & May (1988) Tansey (1986) Cott A, Pavloski RP, Black AH (1979) Quy & Hutt (1979) Kuhlman (1978) Sterman (1977) Kuhlman (1977) Wyler, Lockard, Ward, & Finch (1976) Sterman, MacDonald, & Stone (1974)	*Walker (1995)* Tansey (1985) Finley (1977) Finley (1977) Ellertsen & Klove (1976) Finley, Smith, & Etherton (1975)

	Sterman & Friar (1972)	

MILD TRAUMATIC BRAIN INJURY

Ayers (1993)	Walker (1998) *Salerno (1997)* Walker (1995)	Byers (1995) *Tansey (1994)* *Weiler, Schumann, & Brill(1994)*

STROKE

Ayers (1994)		Rozelle, & Budzynski(1995)

MULTIPLE SCLEROSIS

		Walker (1995)

CHRONIC FATIGUE SYNDROME (CFS)

Lowe (1994)	*Tansey (1994)* Tansey (1993)	James, & Folen (1996)

CHRONIC PAIN, MIGRAINES

	Othmer & Othmer (1994) Tansey (1991) Fehmi (1987)	

IMMUNE DISORDERS

	Schummer (1995)	

LYME DISEASE

		Brown (1995) *Kirk (1994)*

PRE-MENSTRUAL SYNDROME (PMS)

	Othmer & Othmer (1994)	

POST TRAUMATIC STRESS DISORDER

	Manchester(1995)	

BIPOLAR DISORDER

		Othmer & Othmer (1995)

CONTROL	**OUTCOME**	**CASE HISTORY**

Italics - Conference Presentation

This list is staggering in the variety of conditions responding to the training. A comprehensive treatment of the claims for these conditions cannot be undertaken here. Instead, a subset of conditions will be reviewed to indicate the breadth of the remediation accomplished with respect to types of symptoms, and to demonstrate that the remediation is non-trivial. That is, it may lie quite out of the range of what can be expected via spontaneous recovery or even, in some cases, with the standard interventions. Subsequently, an understanding of these findings will be sought by looking at underlying physiological mechanisms.

Before proceeding, it may be useful to make some more qualitative distinctions among the claims being made with respect to these varied conditions. Such an attempt is shown in Table 3. Here conditions are ranked according to the consistency with which remediation can be predicted; the completeness of the remediation; the duration of the training; and the simplicity or complexity of the protocols to be brought to bear. For entries in this table, the judgments are entirely our own, and are based on our own clinical experience.

A Review of Clinical Outcomes

In the following section, the categories listed in Table 3 will be reviewed in cursory fashion in terms of our own clinical experience (augmented by that of some other practices which have adopted the same protocols.) It goes without saying that such a cursory overview of such complex issues can only be unsatisfying to the critical scientist or the discerning clinician. We offer it only as kind of intellectual appetizer, in order to achieve a quick overview of the field that will motivate further engagement and inquiry.

Table 3. Rating our Effectiveness

CRITERIA: A) Consistency of Response
B) Completeness of Remediation
C) Duration of Training
E) Ambiguity of Protocol

-Strong, Consistent Results	Higher Variability of Outcome-
-Full Remediation of Symptoms	Partial Remediation of Symptoms-
-Short Duration of Training	Long Duration of Training-
-Simple, Standard Protocols	Complex, Variable and Multiple Protocols-

Depression ADD ADHD Oppositional Epilepsy
(inattentive) (combined) Defiant Disorder

Minor Traumatic ADD Sleep Disorders Conduct Disorder Stroke
Brain Injury (impulsive) Tourette Syndrome+OCD

Anxiety

PMS Migraines Bipolar Disorder Narcolepsy

Chronic Pain

Tension Headaches Panic Attacks Prenatal Substance Major Head Injury
Exposure

Bruxism Chronic Fatigue Syndrome
Hypoglycemia

Autoimmune
Dysfunction

Depression

It is noteworthy that depression is among the easiest conditions to treat with EEG biofeedback. These findings cover not only the mild depression that is frequently seen in connection with ADD, such as the dysthymia observed in childhood or the kind of low-grade pervasive depression for which Prozac has become the palliative of choice. They also cover episodes of deep depression, including some which are accompanied by episodes of suicidality, and even reactive depression.

The early effects of the training may be observed in the first few sessions. A person may recover from an excursion into suicidality in just one or two sessions. Full recovery from depression may, however, require on the order of twenty to forty training sessions. The recovery is seen as a restoration of a normal range of physiological arousal. The recovery is not characterized, however, by a numbing of feelings or constriction in affective state (in the event of reactive depression), nor does it interfere with a normal grieving process. The training is usually effective in disrupting patterns of chronic pain that are often seen in depression, although we are not dealing here with an anesthesia. Normal pain sensitivity is retained.

It is noteworthy that with SMR/beta protocols the greatest efficacy for unipolar depression is achieved with beta training on the left hemisphere at sensorimotor cortex. Since the left hemisphere is where language resides, one is aided by the fact that the patient can usually articulate very well the consequences of each training session for left hemisphere function and thus help to guide the process. Matters are different with respect to agitated depression or suicidality. These are attributed to disregulation primarily lodged in the right hemisphere, and require the calming and more stabilizing lower frequency training in the general case. The client may not be in a position to either properly appraise or to articulate his or her own state with respect to right hemisphere dysfunction.

These findings are so startling in their import that perhaps they stretch the credulity of the reader, and are entitled to some further discussion to make this plausible. First of all, this finding

of efficacy for depression is concordant with the belief among psychiatrists that depression is rather consistently responsive to electroshock treatment, as already mentioned in the introduction. In many clinical circles, ECT is considered the gold standard of treatment for depression. The severe side effects attendant to that procedure keep it from being employed except as a last resort, and in severe depression. However, the belief is firmly entrenched that depression is expected to respond to shock treatment in the general case. Shock treatment can be seen as a sudden change in the ambient electrical state of brain function. Existing reinforcement patterns of pathological arousal and affect are broken up, and a new homeostasis in terms of arousal level and affect can be quickly established and apparently sustained, often without continuing pharmacological support.

On the other therapeutic extreme, that of non-intervention, it is found that episodes of deep depression quite frequently result in spontaneous remission. Such remission is so commonplace in children and young adults, when deep depression is first observed, that anti-depressant medication has never been shown to be better than placebo (read spontaneous recovery) in children. (Just recently, a first study appeared in which statistical significance was achieved (Emslie, 1997). Yet no one would argue that the nervous system of a child is non-responsive to anti-depressants. The drugs clearly work there as well. It is simply that spontaneous recovery is so robust and commonplace that anti-depressants are not obviously superior statistically in a controlled research setting over a fixed time interval. The mechanisms are clearly in place for a natural recovery to occur in most individuals with a first experience with major depression.

Hence, the claim of efficacy of EEG biofeedback for depression would seem to have the same difficulty vis-a-vis spontaneous recovery that has confounded the drug studies. Not so. In fact, we assert that the mechanisms of spontaneous recovery and of EEG biofeedback are probably identical. The existence of a robust spontaneous recovery capability supports the claim; it does not undermine it. EEG biofeedback can simply induce a systematic re-normalization of arousal function which might also happen randomly all by itself. The difference is that when EEG biofeedback is employed, the response is prompt, predictable, relatively consistent, and more likely to be sustained over the longer term. Moreover, it tracks the specific protocols employed (in terms of electrode placement and reward frequency band). This proposition does not need to await statistical proof (although such proof would be salutary). Simple clinical observation is sufficient (just as it was for shock therapy).

Minor Traumatic Brain Injury

A second category in which rapid, substantial recovery is observed is minor traumatic brain injury. The principal symptoms associated with MTBI are listed in **Table 4**. Many of these symptoms relate to disregulation of arousal, and of these the majority is depressive in character: depression, inattention, irritability, effort fatigue, chronic pain, and frequent waking. Some relate to overarousal: mania, impulsivity, anxiety and fear, anger, and sleep onset problems. Others relate to cognitive function: dyslexia, loss of short-term memory, articulation problems, word retrieval problems. Other problems relate more to frontal lobe function: behavioral disinhibition, obsessive-compulsive disorder, exacerbated motor and vocal tics, perseveration.

Table 4: Characteristic Symptoms of Minor Traumatic Brain Injury

Headache
Chronic Pain

Dizziness-Vertigo
Difficulty Concentrating
Difficulty with Attention
Difficulty Planning
Effort Fatigue
Anxiety and Depression
Sleep Disturbances
Irritability
Mood Swings
Personality Changes
Hemiparesis
Palsies
Aphasia
Visuospatial impairments
Changes in appetite
Sensitivity to hot and cold
Seizures

Characteristically much of the whole spectrum of MTBI symptoms may be manifested in any one head injury victim. And characteristically also, essentially all of these symptoms remediate with the training at least to some significant degree, although at different rates. The recovery of energy, the restoration of the ability to sleep properly, and the stabilization of mood, are the early markers for EEG training. Subsequently, there is recovery of cognitive function, diminution of pain syndromes, and ultimately even recovery of memory function.

Efficacy of the biofeedback for MTBI is probably largely attributable to three factors: 1) restoration of appropriate regulation of arousal level; 2) increase in the stability of brain function; and 3) increase in the flexibility of brain function. Commonly in MTBI the EEG exhibits paroxysmal activity, or elevated low frequency activity. Typically also, significant deviations in temporal coherence may be seen between brain regions. These deviations may be in either direction. Too low a coherence would indicate insufficient coupling or communication between brain regions, and too high a coherence would indicate too tight a coupling. It is easy to explain low coherence in terms of axonal shearing or other structural injury attributable to the original trauma. However, that may be too facile.

EEG deviations tend to normalize with the training, as would be expected. However, that is not always the case. Nor does such normalization closely track the recovery of function. Hence the EEG is of limited utility as a measure of recovery of function. Diminishing of paroxysmal activity is attributed to an increase in cortical stability with a strengthening of thalamic regulatory control. Elevated low frequency amplitude could simply be a manifestation of functional disengagement, of low activation and arousal. It can also result from inappropriate cortical-cortical coupling, attributed to insufficient subcortical regulation. The recovery could therefore again be attributed to the strengthening of thalamo-cortical regulatory mechanisms. Finally there are the deviations in coherence themselves. The fact that coherence is likely to recover with training regardless of whether it is low or high indicates that we are dealing largely with functional disorganization rather than structural impediments to function. Again, it is postulated that reassertion of thalamic control of brain rhythms is sufficient to restore appropriate coherence. However, direct cortical-cortical communication surely also plays a role in normalization of coherence.

Recovery from depressive symptoms is attributed to the first factor, renormalization of arousal control. Restoration of cognitive function and short-term memory is attributed to an increase in

continuity of brain function, to which the diminution of paroxysmal activity and delta and theta amplitudes are testimony. Paroxysmal activity is very likely to disrupt the temporal relationships by which images, concepts and gestalts are bound together as coherent entities and retained for processing. The subjective experience of this disruption is an inability to organize activities, to make plans, to weigh several competing ideas, to carry mental challenges through to their resolution, and to reliably retain a memory. Finally, the restoration of appropriate coherence leads to recovery of the person's original behavioral flexibility.

MTBI has been listed as responding very quickly and reliably to EEG biofeedback training. This is indeed the case, in the sense that there can be significant recoveries of function even in the first few sessions. A more complete resolution may require as many as 50-100 training sessions, although 20 ' 30 sessions are adequate in most cases. A representative sampling of 16 such cases was reported by Jonathan Walker, of the Neuroscience Centers in Dallas. The results are summarized in **Table 5**. The average recovery with respect to premorbid functioning, by self-report, was 83%, and the median improvement was 85%. The average number of training sessions was 32, and the median was 30. The EEGs changed in line with the protocol to a statistically significant degree (decrease in theta amplitudes, and an increase in beta amplitudes).

Table 5. Recovery by self-report from symptoms of Minor Traumatic Brain Injury.

Client	Baseline av.pwr Beta/Cz	Post training av.pwr Beta/Cz	Baseline av.pwr q/Cz	Post-training av.pwr q/Cz	Percent Improvement	Number of Sessions
K.R.	5.1	8.3	12.1	7.2	100	14
R.M.	6.5	16.9	11.3	10.8	80	12
M.M.	7	9.3	14	11.9	95	18
J.M.	15.1	18.6	10.5	6.5	90	40
C.G.	4.8	5.6	22.6	19.5	80	43
A.D.	14.4	20.4	10.6	7.8	50	46
S.A.	4.4	5.2	13.9	15.7	90	13
T.G.	5.8	12.8	13.1	13	80	35
P.K.	6.1	11.7	24.7	17.8	50	86
M.D.	8.6	12	18.4	15	80	30
E.S.	9	9	17.4	17.1	100	30
C.H.	10.1	8.1	13.1	11.9	90	20
S.S.	7.7	9.5	27.8	23.1	100	42
S.B.	8.2	11	14.6	9.3	75	23
G.C.	4.7	5.1	12	9.4	98	22
S.B.	9.3	13	25.8	16.9	75	30

Data courtesy of Jonathan Walker, MD

There may also be obvious deficits remaining that relate to organic (morphological, structural) injury. In these cases significant recovery is possible as well, but the rate- limiting mechanism is presumably some dendritic regrowth or rearborization. Hence the pace of progress is only partly conditional on the schedule of training. The trainee may continue to make gains by returning to the training episodically, to exploit any new learning opportunity. This phase of training is similar to the experience of Bernard Brucker (1985) in his EMG training for spinal chord injury, where it is found that a limb which did not yield to training on one occasion may readily respond a year later.

When specific organic injury has occurred, it seems more appropriate to include this in the category of major head injury. However, the latter distinction is reserved for those head injuries in which skull fracture or major organic loss has occurred. This is a less meaningful distinction,

and often uncorrelated with the severity of deficits incurred. Paradoxically, some head injury that involves skull fracture can be less severe than minor head trauma. Conceivably, this could be due to the fact that the skull fracture, by yielding somewhat on impact, can reduce the g-forces sustained by the brain and the brainstem. For present purposes, organic injury is lumped along with major head injury, and as such appears in the last column of the chart.

The intimate connection of head injury symptomatology with disregulation of arousal seems to have been under-recognized by clinicians, who have by and large retained both a structuralist perspective as well as a focus on the cortex as the locus of injury. When such techniques as CAT scans and structural MRI scans failed to confirm injury, the victim was often declared to be a malingerer and his symptoms discounted. Thus the person became a victim a second time, in this instance of the clinician's myopia. In fact, most head injury involves severe jostling of the head upon its spindly neck, resulting in trauma to the brainstem, from whence arousal is managed. Fortunately, such injury consists more likely of compressional effects such as anoxia rather than of actual axonal shearing. As such, the injury is functional in nature, rather than structural, and turns out to be eminently remediable with our techniques.

Premenstrual Syndrome

Another indication for which EEG biofeedback is very helpful is Premenstrual Syndrome (PMS). This condition is not recognized as a distinct disorder in the DSM-IV, but that is probably at least partially in recognition of societal sensibilities. In its severe form, it is known as Premenstrual Dysphoric Disorder, which is conditionally listed in the Appendix of the DSM-IV (DSM-IV, p. 715). The difficulties with such a listing are, among others, that the symptoms of PMS are so diverse, so highly variable, so subject to 'psychosomatic" influences, so frequently seen simply as an exacerbation of other existing disorders, and so devoid of discernible organic basis. One wishes to blame hormonal shifts, but these are not usually out of line in those suffering PMS symptoms.

The weight of evidence is that PMS is a matter of brain sensitivity to ordinary shifts in hormonal levels. PMS can even be considered as the defining condition for the functionally based "brain disregulation model" of psychopathology. That is, disregulation is the defining characteristic of PMS, and the remedy offered by EEG training is to return brain function to homeostasis and to stability, i.e. to a restored capacity for neuroregulation. Almost no condition remediates as completely and consistently as does PMS with EEG training, and few conditions entail such a breadth of symptomatology. Yet PMS in all its clinical variety is successfully addressed with little more than this straight- forward training. PMS symptoms which have been identified are shown in **Table 6** (O'Brien, 1987), and the symptoms which have been observed in our practice, and which have been subject to remediation, are shown with an asterisk. We have no relevant experience with the symptoms that are not marked.

Table 6. Representative Symptoms of PMS.

Those symptoms which have been observed to respond to EEG training are shown with an asterisk. Other symptoms were either not reported or not verified, or not deemed to have been primarily PMS-related.

Physical	Behavioral
*Drowsiness	*Aggression
*Fatigue	Anorexia
Thirst	*Decreased Alertness
*Proneness to Accident	*Decreased Libido

Acne	*Food Craving
Asthma	*Hunger
Bloatedness (actually)	*Bloatedness(feeling)
*Blurred Vision	*Hypersomnia
*Breast Swelling	*Impulsive Behavior
*Breast Tenderness	*Increased Libido
*Clumsiness	*Insomnia
*Constipation	*Lack of Volition
*Diarrhea	*Lethargy, Listlessness
Dizziness	*Loss of Judgment
Epilepsy	*Loss of Self-Control
Finger Swelling	*Social Isolation
*Flushes	*Suicidal Tendency
Formication	*Tension
*Headache, Migraine	*Violent Behavior
Weight Increase (actual)	*Weight
Increase (feeling of) Vertigo	
Sinusitis	
*Pelvic Pain	EMOTIONAL
Edema	
*Nausea	*Agitation
*Muscle Pain	*Anxiety
*Joint Pain	*Contentiousness
*Vomiting	*Depression
*Hypoglycemia	*Emotional Lability
	*Hopelessness
	*Irritability
COGNITIVE	*Loss of Confidence
	*Malaise
*Confusion	*Moodiness
*Loss of Concentration	*Pessimism
*Proneness to Accident	*Sadness
*Poor Coordination	

The above results are also non-trivial. PMS symptoms can be disabling in their severity for a significant fraction of women. Yet is has been possible to remediate even the most severe cases encountered. Individual case histories cannot not be reviewed within the prevailing limitations in space, but one example may be given for concreteness: It has been observed that a woman who had a lifetime history of severe PMS, with frequent episodes of suicidality, and with a litany of failed interventions, was able to reach a point within forty sessions of training where she was unaware when her period approached. At the initiation of training, the woman was scheduled for surgery for fibroid tumors. The surgery was never performed. The failure rate in training is on the order of five percent or less for those who follow through with the training until meaningful milestones (20 or 40 sessions) are reached. Some of these failures probably relate to ongoing emotional issues that compromise or sabotage the training. Other cases of PMS are likely sustained by histories of early sexual or physical abuse, and might not remediate with high- frequency training alone, but rather would require alpha-theta training as well.

Medically, PMS is typically managed with anti-depressants such as Wellbutrin. Such pharmacological approaches remain deficient, since the condition is so volatile and variable that no unilateral, long-acting shift in neuromodulator function can offer remedy. It is noteworthy, however, that the EEG biofeedback protocol most commonly employed is also used for depression, and is probably the closest EEG training analog to an antidepressant. Clearly, the EEG

training can not only shift the "operating point" of the nervous system in terms of arousal, but also increase the "operating range" over the continuum of behavioral states.
The training does not have to be done during the symptomatic phase of the cycle. This makes it apparent that the training promotes a nervous system capability rather than a particular state. On the other hand, if the training is done during the symptomatic phase, the trainee may experience changes in symptoms literally from session to session, or even during a single session. If at least six sessions of training are accomplished between periods, then substantial relief will typically already be experienced by the time of the subsequent period.

Headaches

Finally, in the category of the conditions most readily remediated we have what are colloquially referred to as tension headaches. Such headaches typically subside within thirty minutes of the appropriate training. Conversely, they can get worse with the wrong protocol selection. With repeated training sessions, susceptibility toward tension headaches can be abated and a person rendered essentially headache-free. Curiously, tension headaches tend to respond to the higher frequency training, as opposed to the lower frequency training that is thought to be more calming. The training in this instance is probably best thought of in terms of increased control of brain states, as opposed to a "relaxation" model.

Attention Deficit Disorder

Next in the order of difficulty and complexity we have ADD, migraines, panic attacks, bruxism, and hypoglycemia. Within the diagnostic category of ADD we also have to distinguish the combined type from the inattentive and impulsive subtypes. The combined type is slightly more complex to deal with, and therefore is placed in the next level of complexity.
Although the dominant application of EEG training is to ADD, it is by no means the easiest to deal with. Perhaps this is due to the fact that the condition is clearly not a unitary phenomenon. ADD is a "dirty" diagnosis. It is so riven with comorbidities that its essence can be obscure. (This is particularly true in the children likely to be referred for EEG training, who have typically already failed to respond to conventional remedies.) The case has even been made that ADD is a composite of more fundamental disorders, including affective disorders, specific learning disabilities, and a primary disorder of vigilance. (Weinberg, 1992, 1993). In an explicit investigation of comorbidities, less than half of ADD was found to be uncomplicated by diagnoses of major depressive disorder, anxiety disorder, or conduct disorder. (Biederman, 1991) Oppositional-defiant disorder alone overlaps 60% with ADD. And when one also considers Tourette's Syndrome, dysthymia, bipolar disorder (Biederman, 1996), specific learning disabilities, elimination disorders, pain syndromes, sleep disorders, and PTSD, then there remains very little which is not compromised in a significant way by comorbidities which have their own specific implications for EEG training.
Consistent with our model that much of the phenomenology of ADD and its comorbidities is traceable to a modest set of underlying failure modes, it is appropriate to assess the remedy by a means of an evaluation tool which focuses attention at that level. This caused us to eschew the conventional behavior rating scales. Instead, we relied upon a continuous performance test, a computerized test which assesses sustained attention, vigilance, and impulsivity. We chose the Test of Variables of Attention, or TOVA(r) (Greenberg, 1987). This test was favored because it had a demonstrated lack of practice effect, and it has been in common practice for titration of

fast-acting stimulant medications for ADHD because of its sensitivity. Thus, if the evidence surfaced by this test was accepted for assessing medications, then it would clearly have to be accepted as a measure of EEG biofeedback as well.

The test is a go-no go challenge that requires only up-down discrimination (which even plants can manage). The test conditions remain invariant for 11 minutes, at which time they change from a stimulus-infrequent to a stimulus-frequent condition. This monotony is a feature of the test, and serves as a challenge to sustained attention. The length of the test helps to assure reasonable statistics on errors of omission, which are taken as a measure of inattention. Errors of commission, which are typically more frequent, are taken as measures of impulsivity. Average response time is measured, as well as variability in response time. The latter is taken as the most revealing measure of ADHD. Results of TOVA testing for 342 subjects are shown for the four subtests in **Figure 1**.

Mean pre- and post-training results are shown in terms of standard scores for the four dependent measures of the TOVA. The data are segregated by severity of initial deficit for each measure. Standard scores of less than forty are not deemed to be meaningful, and are arbitrarily set at forty for this analysis (four standard deviations below the mean). For inattention and for variability, the data show that the most impaired group (starting score of 40) improved by two standard deviations. In the case of impulsivity, the most impaired group improved by three standard deviations. The effect size is seen to be quite significant. Data are not shown for those whose starting values were >100. Thus the actual number of subjects comprising each graph (as shown) is less than 342.

It is revealing to look at the individual data comprising the data of **Figure 1**. This is shown for impulsivity in **Figure 2**. The individual data reveal the consistency with which positive results are obtained. Some 84% of the data points are positive-going despite any test-retest variability, and even though the data include those subjects who test within the normal range. This Figure is proof that we are not dealing with a regression to the mean, if any doubt remained. The entire population moves upward, irrespective of starting point in terms of standard score. This observation demonstrates that essentially everyone is capable of responding to this training. This, combined with the fact that subjects can be readily moved to function above na‹ve norms, disposes of any residual placebo arguments. Some of the small number of cases in which scores declined significantly (beyond expected test-retest variability of perhaps half a standard deviation, or 7 points) may very well have done so in response to the training, as opposed to being "non-responders." The decline may be attributed to choice of training protocol, which may in these cases have been driven by issues other than impulsivity. A different choice of protocol might well have effected a recovery in those cases, but that opportunity does not always present itself in a clinical setting. Some declines in score of course have trivial explanations, such as illness on retest.

ADHD of mixed type requires a combination of approaches used for training the inattentive and impulsive subtypes, and these need to be properly titrated in order to achieve optimal results. For this reason, ADHD of mixed type is considered more of a challenge than the simpler subtypes, and is therefore listed in Table 2 as being of greater difficulty. It is appropriate, however, to incorporate it into this discussion. In addition to evaluations with the TOVA, IQ tests and other tests of cognitive function have been found useful in the past. In an early study of ADHD by our group that has not previously been published in a professional journal, Wechsler IQ scores were measured pre-post. The results are reproduced in **Figure 3**. The tests reveal the classic pattern for ADHD, namely depressed scores for Information, Arithmetic, Digit Span, and Coding. After the training, the same characteristic pattern is still recognizable, but at a much higher level. The

increase in mean arithmetic scores, for example, is quite astounding. Since nothing in the training conferred arithmetic skills, one must attribute the gains to something like increased working memory. These children knew the rules of arithmetic all along. However, they failed in execution. The training allowed them to persist to completion, and to retain in working memory the task they were about. The increase in coding score is more modest. However, closer inspection reveals that a number of subjects did not change at all on the coding test; others made substantial changes. This study was performed early in our work (1990-1991), when a single protocol predominated. It is possible that the lack of progress in some scores is attributable to that paucity of approaches.

The general impression one has from these data is that the training improved level of function broadly. The average improvement in IQ score was 23 points. This change is much too large to be attributable to a test-retest effect, particularly since the retests were done typically nine months after the pre-test (with a six-month minimum interval). Verbal and Performance IQs changed comparably in most subjects. The largest improvements were seen in Picture Completion, which is not seen as a measure in ADD. The least change was seen in Block Design. Verbal and Performance IQs changed comparably in most subjects. This is noteworthy, because in most of them only left-side training was performed. The results imply that the training impinges on inter-hemispheric communication pathways as well.

The three categories of Arithmetic, Coding, and Digit Span together constitute a measure called 'Freedom from Distractibility.' All three also depend on sequential processing skills. The view commends itself that EEG biofeedback increases the 'continuity of mental states,' which manifests itself behaviorally in terms of reduced distractibility, and cognitively in terms of improved sequential processing ability and improved working memory.

In support of the contention that the training influences function broadly, there is the additional evidence of the Benton Visual Retention Test. Whereas the IQ test showed the group to have been of above-average IQ (107) even before the training, they were in significant deficit with respect to visual retention, as shown in **Figure 4**.

After the training, some six subjects rated superior, having tested at average or less before the training. Everyone improved with the training, and one subject moved all the way from a defective to a superior rating. This subject unambiguously experienced an improvement in his level of functioning that cannot be explained by non-specific factors. The change is so startling that it does not require the weight of statistical evidence to prove the point.

Improvements were also noted in the tapping subtest of the Harris tests of lateral dominance. These results are shown in **Figure 5**. When these results are plotted up in terms of the ratio of right-to-left hand performance, an intriguing result obtains. We observe a depletion of mixed dominance and a loss of scatter in the data, as shown in **Figure 6**. Laterality normalizes. This test is unequivocal testimony to the fact that the training produces change in neurophysiological functioning. First of all, this test is unambiguously scoreable. There is typically 95% concordance between different testers. Secondly, the result was neither expected nor even wished for. (Inclusion of this test in the battery was almost an afterthought.) Thirdly, laterality presumably is not affected by non-specific aspects of the training, such as motivational factors. Fourth, the training itself does not involve any movement of the hands. Improvement in this regard must be ascribed to "central" effects of the training.

Some years ago, it was found that childbirth trauma significantly altered patterns of laterality. The study, published in Nature, examined fetal thumb-sucking and found that before birth, 95% of fetuses preferred their right thumb (Hepper, 1990). After birth, only 85% did so. The shift can be interpreted as an effect of birth trauma, which may bring about a compensatory shift to opposite

hemisphere dominance or to mixed dominance when the natively dominant hemisphere has been injured. An appealing suggestion is that the EEG training remediates the functional injury. In this view, the tie-in to ADD becomes more apparent. The functions of vigilance and sustained attention have their own hemisphere-specific mechanisms. When birth injury disturbs hemispheric function to the degree that it impacts handedness, then perhaps it could also impact the management of vigilance and attention. Hence head injury in general, and birth injury in particular, is another confounding variable in the diagnosis of ADHD. This is not surprising. The original research in hyperactivity considered it to be grounded in minimal brain injury.

Attention Deficit Disorder-Combined Type

ADHD of the combined subtype has been ranked of slightly greater difficulty than the inattentive and the impulsive subtypes. And even though we are presenting issues in the order of difficulty as shown in Table 2, it is appropriate to take up the matter of ADHD here. The issues are just slightly more complex, and require a somewhat more complicated protocol management. The additional complexity is partially attributed to the comorbidities of ADHD previously mentioned. The prominence of significant comorbidities in the clinical population makes research problematic in that setting.

Protocols may involve training at C3 and C4 (and, historically, Cz) on the sensorimotor strip, with both SMR and beta reward frequencies. They may also involve frontal training at Fz or Fpz, as well as parietal training at P4 or Pz. Left hemisphere and frontal training are more likely to involve the higher frequencies (nominally 15-18 Hz), whereas Pz and right-side training are more likely to involve the lower frequencies (nominally 12-15 Hz). This is consistent with a lower degree of localization in right-hemisphere functions. It is also consistent with current theories of activation and arousal, as previously discussed (the Tucker- Williamson and Malone, Kershner, and Swanson models). This is consistent with the strategy that has emerged in EEG training, namely high frequency training for improved control of activation on the left hemisphere (sometimes with a frontal bias with bipolar montage), combined with lower frequency training on the right hemisphere (sometimes with a parietal bias with bipolar montage).

Migraines

One of the remarkable findings of the past few years is that migraine headaches respond readily to EEG biofeedback training. Efficacy has also been demonstrated for migraines with conventional biofeedback, but there seems to be particular merit in training the brain directly for this vulnerability. Ongoing migraines can sometimes be aborted, or more typically significantly lessened in severity, in thirty minutes of training with the appropriate protocol. In the case of migraines, there are two principal protocols. Choice of the wrong one may often lead to increased migraine pain within a matter of minutes, which motivates a change of course. It is also found that migraines will move from one place in the head to another in response to the training. It may be advantageous to respond to the movement of pain locus for the most effective training.

These prompt responses to the training are concrete evidence that the training is having a specific effect. However, these are the least interesting effects. If the training is pursued long-term, then a propensity toward migraines can be arrested relatively permanently. On the order of twenty to forty training sessions may be required to achieve this objective (absent complicating issues). Moreover, such an outcome is highly predictable. Migraines are extraordinarily responsive to this training. Follow-up data indicate that these gains may be held for several years (that is, for as

long as follow-up has been conducted). Barring the happenstance of further trauma, the effects seem permanent. Also, the training efficacy does not appear to depend a great deal on what kind of migraine one is dealing with, classic or common. It is interesting to speculate how this might occur.

Migraines can be seen as a particular form of collective activity of neuronal populations. It is fundamentally a matter of the brain rather than of the vasculature. After all, migraines can be triggered by light stimulation. It is assumed that the effect of such stimulation is on neuronal systems, not on the vascular system. Hence, what happens to the vasculature is consequence, not cause. The light stimulation of a vulnerable brain is assumed to unleash a cascade of collective activity that alters neuromodulator function (serotonin in particular) at the brainstem level. The time constants of such changes in neuromodulator function may be long, but not long enough to account for the duration of migraines. The latter requires some kind of self-reinforcement of the adverse state.

Migraines are characterized by disregulation of central arousal function, which also impinges upon sympathetic and parasympathetic balance. The problem is fundamentally one of instability, for which typical pharmacological agents are not a good answer. The remedy is to increase fundamental stability in the brain, so that the excursion into migraines cannot be as readily triggered. In its role in aborting active migraines, the EEG training may be compellingly promoting a particular state of arousal that stabilizes against the ongoing excursions in arousal level. Even in the case of training at the higher EEG frequencies, reinforcement of an increased EEG amplitude is in effect to reward quiescence. We will return to this theme later.

In training to remediate migraines, sessions are of course preferably conducted during an asymptomatic period. The obvious signposts of whether the correct protocol has been selected may then be unavailable. A general pattern has emerged, however, in which migraines generally require both the higher and lower frequency training to improve stability, with a bias toward the lower-frequency (SMR) training unless the migraines are PMS-related, in which case a bias toward higher-frequency (beta) training prevails. A client may need to keep records of their migraine incidence in order to document the improvement as early as possible to confirm the choice of protocol. Also, migraines are not usually the only symptom affected by the training. The individual will respond favorably to the correct training in other ways, such as improved sleep and mood regulation. In general, if there are adverse consequences in any of a number of areas, an adjustment in protocol is called for.

Panic Attacks

Panic attacks may be considered another paroxysmal brain state in which inappropriate collective activity is subjectively perceived as a panic reaction. It arises out of a matrix of vulnerability to anxiety. Stabilizing the brain against excursions such as panic attacks is quite readily achievable with EEG biofeedback training, and protocol selection is generally straightforward. As in the case of migraines, such stability is difficult to achieve with pharmacological means.

One striking and illustrative case must be mentioned. A woman who had been in treatment for panic anxiety and agoraphobia for ten years, with repeated hospitalizations, long-term psychotherapy, and extensive pharmacological intervention, was eventually given EEG training by the same psychologist who had worked with her for ten years. After only eight sessions, she was able to vacation with her husband in Las Vegas, mixing easily in crowds, and declaring later that she felt anxious only once. On the basis of cases such as these, panic attacks are seen as fundamentally issues of brain instability rather than of psychological state. There may have been

psychological underpinnings, but panic susceptibility takes on a life of its own. Onset of panic excursions appear to be chaotic in character, and in its mature form need not have a behavioral antecedent.

Bruxism

Bruxism often responds quite readily to EEG biofeedback training in the beta and SMR domains. Intuitively, bruxism would appear to be a stress reaction, one for which relaxation training might be the appropriate remedy. However, the fact that bruxism is also commonly observed during general anesthesia makes it more reasonable to regard it simply as a consequence of disregulation of arousal, or even of underarousal.
Sterman has proved the direct connection of SMR-training at sensorimotor cortex with motor inactivity in cats, and the identification holds true in primates as well (Sterman, 1978) Hence, SMR-training would appear to be the appropriate remedy. This is generally true, but sometimes an instability in arousal requires beta training also. In the present instance, it is still preferred to regard the process as a normalization of arousal, with whatever frequency training is required to accomplish that objective in a particular instance, and that in consequence of such normalization motor system activation will normalize as well.
In clinical experience, it has been found possible to normalize nocturnal bruxism behavior as it is commonly observed in children with attentional disorders, as well as long-standing conditions in which major restorative dental work has been mandated by the persistent bruxism. This fairly general clinical success supports the hypothesis of bruxism as having a central nervous system origin as opposed to being primarily a disorder attributable to such factors as malocclusion (Parker, 1990, McNeill, 1990).

Hypoglycemia

Hypoglycemia can be regarded for present purposes as disregulation of blood glucose level, which is presumptively also under the management of the central nervous system. One of the common failure modes of a feedback regulatory system is that it can go into damped oscillation. A small stimulus can send the system in to near oscillation, from which it recovers only slowly. In this case, the small stimulus may be a sugar challenge, or even a challenge with a sugar substitute.
It has been found quite generally that conditions of hypoglycemia can be normalized with EEG training in the higher frequency domains of SMR and beta. The measure in this case is simply behavioral. No studies of glucose level after EEG training have been done, to our knowledge. However, it is observed that the cognitive, behavioral, and mood aspects of hypoglycemia remediate with the training, and dietary restrictions can often be abandoned after the training reaches completion. Thus, either the glucose levels have been stabilized through improved regulatory function, or the brain has been made more tolerant to the fluctuations in ambient glucose level.
A case can be made that glucose regulation is directly affected by the training on the basis of comparison with Type I diabetics undertaking the training. A reduction in insulin requirement may be observed in these cases (in which of course the glucose level is being actively monitored). A more stable blood sugar level is implied. It has been observed in some Type II diabetics, for whom dietary management was becoming insufficient, that EEG training could delay indefinitely the onset of insulin replacement therapy.

Sleep Disorders

The field of SMR-beta EEG biofeedback training got its start in the context of sleep studies (Sterman, 1970). Since the early years, it has become clear that one of the first observable data consequent to EEG training relates to the quality of sleep. The EEG training has emerged as a powerful tool in the management of ordinary sleep disorders such as sleep onset difficulties, frequent waking, nightmares and night terrors. (More challenging sleep disorders such as sleep apnea, narcolepsy, and nocturnal myoclonus are not included here.) A relationship has been observed between pattern of sleep disturbance and affective disorders. Thus, sleep onset difficulties correlate with anxiety, and difficulty in staying asleep are correlated with depression. The inability to find one's way to bed, and sleeping only a few hours each night, is associated with mania. The protocols used in these cases are identical to the approaches used for anxiety, depression, and mania, respectively.

Nightmares can be seen as an anxiety phenomenon for purposes of protocol selection. Night terrors, on the other hand, are presumably a paroxysmal event. They generally respond to a combination of higher- and lower frequency training. Nocturnal elimination disorders (enuresis, encopresis) generally respond readily to the training in the young. However, enuresis which survives into adulthood may require a greater variety of protocols and a greater number of training sessions, and may even be entirely refractory to training with any protocol we have devised. Enuresis can be considered a concomitant of disregulation of arousal during sleep. Encopresis, on the other hand, could be a paroxysmal phenomenon, possibly requiring more extended training.

Efficacy for common sleep disorders can be invoked in support of the model that improved regulation of arousal is the primary mode of action of EEG biofeedback training. It can also be used to exclude the hypothesis that overt behavior may be trained as opposed to control mechanisms. After all, rehearsed behavioral strategies are not likely to be relevant as the brain manages its own sleep state transitions. Sometimes aborting a pattern of enuresis or nocturnal bruxism can be accomplished within one to four training sessions. The person most surprised may be the child himself, unaware of having made any behavioral adjustment whatsoever.

Anxiety

In principle, anxiety responds exceedingly well to EEG biofeedback training. In this respect, it is similar to peripheral biofeedback. In practice, however, just as with peripheral biofeedback a considerable problem with patient compliance may be observed. Whereas the competence of the technique in remediating anxiety is now beyond question, there are other factors that can affect compliance adversely. The dynamics of the training can elicit performance anxiety; the anxious person may have difficulty abandoning the perceived'but ambiguous''comfort zone' of the anxiety state (to wit, my vigilance is keeping me alive). By the same token, the anxious person may have difficulty perceiving a more relaxed and controlled state as being desirable. In some individuals, compliance can be increased if the lower- frequency alpha training is also employed early on in training. However, the latter is not the focus of this survey.

It is primarily for reasons of compliance that we have ranked anxiety as more problematic, on the whole, than panic disorder. This probably contrasts significantly with what is found with other therapies. We consider panic disorder as a paroxysmal condition. The EEG training is manifestly quite competent in stabilizing the brain against the minor paroxysmal events such as panic

disorder and night terrors previously discussed. Obtaining such stabilization may, curiously, be quicker and easier than comprehensive management of an anxiety susceptibility.

Chronic Pain

Chronic pain is not a unitary phenomenon. We subsume under this topic all such pain that persists over the long term and does not appear commensurate with the apparent cause. The categorization includes lingering post-operative pain, persistent post-trauma pain, lower-back pain, fibromyalgia, causalgia, sciatica, and Reflex Sympathetic Dystrophy. Apparently, such inappropriate pain reaction is sustained by a reinforcement of pain sensory signals through central nervous system gating mechanisms. EEG biofeedback training can frequently disrupt this escalation of pain sensitivity and restore normal pain thresholds. Sometimes these results are achieved quite quickly, even in persons who have been quite resistant to standard interventions, including in particular conventional peripheral biofeedback.
It is this rapid response to relatively simple protocols in many cases that caused us to categorize chronic pain in this column of the table. However, many other cases require quite comprehensive care and do not respond quickly to EEG training. Such a resistance to the high-frequency training should be considered a marker for a trauma history. These persons require not only the higher frequency training but also alpha- theta training. In truth, then, chronic pain could equally well be listed along with the most challenging conditions on the Table.

Oppositional-Defiant Disorder and Conduct Disorder

It is quite satisfying to observe that the disruptive behavior disorders of ODD and CD are profoundly remediable with EEG training. This indicates that these disorders are significantly neurophysiologically driven. And this in turn means that when the physiological dimension of the problem is addressed, there may be very little residual that needs to be addressed with other modalities such as talk therapy (that is, talk therapy directed to the behavior problem, as opposed to issues of family dynamics). The training is not intrinsically more difficult than with pure ADHD. However, compliance is clearly an issue, since the children are of an age where they have to be brought by parents to the training, and parental relations are likely to be problematic. It is for this reason that ODD and CD are listed on the more difficult side of the spectrum. On the other hand, there is a clear advantage of EEG biofeedback vis-a-vis talk therapy since there need be no discussion of the fact, or even understanding by the child, that the training is intended to deal with a behavior problem. The EEG protocols employed suggest an association of ODD with left-hemisphere function and with depression, whereas conduct disorder is more of a right-side issue in which the aggressive child is not well-coupled to the source of his own emotional states.

Bipolar Disorder

One of the most remarkable findings of the past several years is that EEG biofeedback training can effectively remediate even medically refractory cases of bipolar disorder. In retrospect there is a remarkable parallelism here to developments in psychopharmacology, where it was found (Post, 1989) that anticonvulsants could be helpful in stabilizing against bipolar excursions that were refractory to lithium administration. Thus an initial finding by Sterman of efficacy for seizures led eventually to our discovery of efficacy for bipolar disorder as well.
Initially, this came about through our work with depression. In the course of training clients out of depression, typically with left-side beta training, it was sometimes noted that they manifested

manic propensities. These could in turn be controlled with right hemisphere SMR training. Thus a protocol gradually emerged for addressing end-stage bipolar disorder. It involved moving the client toward a more appropriate arousal level from whatever starting point (manic or depressive) prevailed at the time. Clients could be so responsive to the training that an adjustment of the protocol might need to be made several times during a single training session. (Any thought that EEG biofeedback may involve a large placebo component dissipates as one watches a bipolar client train.) Fortunately, bipolar clients are usually able to articulate their state very well in order to guide the training. It is in application to bipolar disorder that the 'exercise' model of EEG biofeedback, or the 'regulatory challenge' model, is most clearly illustrated.

These findings have been replicated in several clinical settings with the above protocols. Even rapid-cycling bipolar disorder (24-hour cycle) was found to stabilize in one individual in 22 sessions. In fact, bipolar disorder responds to this training quite consistently. Bipolar disorder illustrates an apparent paradox: some conditions that are quite refractory to pharmacological management and/or psychotherapeutic intervention nevertheless yield quite readily to EEG biofeedback. This puts into relief what is perhaps the most significant role of this modality: increasing brain stability and enhancing continuity of states.

Prenatal Substance Exposure

It is our clinical experience that children who had been subject to prenatal substance exposure are quite readily responsive to this training. In this category, we are seeing children who are not institutionalized. Hence, our experience is restricted to those children who have less severe impacts. The symptoms being addressed are those of ADHD, emotional disturbance, mild mental retardation, pervasive developmental delay, etc.

One of the significant findings is that even children with low IQ's (about 70) are intellectually capable of responding to this training, and their brains may have significant recovery potential. In one instance (not in our own practice) a child of 70 IQ was remeasured at 112 after one year of training. Our experience with prenatally substance exposed children extends down to the age of three. Clearly, the mental capacity to respond to this training is present even in severely impacted children of very young ages. The condition is listed in this column because of the large variety of symptomatology one must confront with children thus at risk.

Epilepsy

Epilepsy is listed among the most difficult conditions to address with EEG training because of the variety with which epilepsy manifests, because of the ongoing structural deficits which may underlie the condition, and also because historically it has been the most intractable cases which have been referred for EEG training. This historical circumstance introduces a bias into how matters are viewed, since in fact there are many types of seizure that respond quite readily to EEG training. Since training was done at the sensorimotor strip, and was deemed to address the motor system specifically, Sterman argued initially that the training could be expected to be beneficial only for seizures with a predominantly motor symptomatology. Subsequently, however, a controlled study was successfully accomplished with primarily temporal lobe or complex-partial seizures (Lantz, 1988).

The Sterman protocol was replicated for seizures in a number of laboratories and by a number of groups (See References for Table 1.) The technique failed to be acknowledged at the time, however, because of confounding issues regarding anticipated changes in the EEG (Quy, 1979).

The cat EEG had manifested a countable increase in incidence of bursts of SMR rhythmic activity with training. The human EEG does not exhibit such bursts except during Stage 2 sleep. And whereas there was in fact an increase in sleep spindles with training in epileptic subjects, the various studies which were intended to replicate Sterman's findings did not yield consistent EEG changes in the waking state (Kaplan, 1975). We now understand that this is not a contradiction. The human EEG remains more desynchronized in the waking state than the cat EEG, and observable bursts would now be considered anomalous. Some individuals did in fact show increased amplitudes of the EEG in the SMR subsequent to long-term reinforcement; others tended toward normalization of their EEG characteristics, which in many instances meant an overall decrease in EEG amplitudes, even within the training band. At the relevant time, however, during the 1970's and 1980's, the lack of consistent EEG changes accompanying the training was thought to be fatal to the hypothesis that EEG training had taken place. The behavioral benefit of EEG biofeedback training that had been replicated in all of the studies was therefore attributed instead to non-specific factors.

Subsequent developments (in our clinical setting) extended the seizure work to absence seizures as well. These generally require higher-frequency training of 15-18Hz in addition to the SMR-training. It is important to make the distinction that in the use of EEG training with seizures, no claim is made that the seizure focus is in any sense extinguished or annihilated. Rather, it is claimed that by enhancing stability conditions in the surrounding healthy brain tissue, the irritable focus will no longer as readily lead to spreading of paroxysmal activity and hence to focal or generalized seizures. The effect of enhancing stability can often be additive to the effect of anti-convulsant medication. It may also lead to the reduction or even elimination of such medication.

More than half of seizures occur at night, and most of these are closely associated with sleep transitions, particularly with falling asleep and waking. This association suggests an intimate connection of seizure susceptibility with stability of arousal. Similarly, about half of all seizures are associated with identifiable events of brain trauma. This of course suggests a connection between the seizure susceptibility with the specific organic loss suffered in the brain injury. However, an equally compelling case can be made that the association is in fact with arousal disregulation here as well. As already indicated in our discussion of brain injury, the predominant symptomatology associated with such injury relates not to the specific location of injury, but to generalized function, in particular the management of arousal. Hence, a brain with an intrinsic seizure vulnerability could simply have been pushed over the edge by a minor head injury.

The hypothesis that efficacy for epilepsy is traceable in large measure to improved regulation of arousal comes from an unusual quarter. A Swedish study has demonstrated some 60% seizure reduction in children by behavioral methods alone (Dahl, 1992). The strategies typically involve deliberate changes in arousal level when the subject anticipates a seizure. As it happens, the 60% reduction is also the average seizure reduction obtained using the Sterman protocol in the various published studies. The nexus with arousal dysfunction helps us to address the structuralist objection that the seizure focus should be impervious to such an intervention as EEG biofeedback. (Whether articulated or not, it is this structuralist objection that has resulted in neurologists dismissing this technique out of hand for thirty years.) It is in fact quite sufficient to argue that only healthy brain tissue is affected by the training in order to explain the clinical findings.

Stroke

The application to stroke recovery is one of the most unambiguous demonstrations of the power of the technique. Most stroke victims who have found their way to EEG training have already been

living with the consequences of the stroke for a number of years. Hence, their other therapies have typically terminated by the time they start the training. Further spontaneous recovery is no longer expected after about 18 months. Hence, any significant improvement in function achieved after that time must in fairness be attributed to the EEG training.
The recovery of function in the case of stroke can be divided into three stages. The first involves recovery from the generalized symptoms attributable to the stroke. These may include depression, irritability, effort fatigue, sleep problems, and attention problems. These symptoms are often predominantly depressive in character, are therefore related to regulation of arousal, and are typical of anyone who has suffered any kind of brain trauma, not specifically stroke.
The second stage involves recovery of the specific functions that were impacted by the stroke. Thus, there may be improvement in gait, in spasticity, in hand movement, in speech articulation, in word finding, etc. Remediation in this second stage may require a period on the order of a year, and up to 100 training sessions. It is assumed that in this second stage one is taking advantage of residual functional brain matter in the region of the organic injury, and that the training helps to reintegrate that region through improved functional connectivity to other parts of the brain.
The third stage involves longer-term training. A person may find it advantageous to return for additional training periodically. In this stage, one is taking advantage of any dendritic regrowth into the region of organic injury, or of continuing changes in assignment of function of remaining functional areas. The time constant of such regrowth is long'years! By training periodically, one can take advantage of any such regrowth that may have occurred in the interim. One presumes that the advantage of such episodic training may continue for as long as it is undertaken. The combination of all three stages of training can yield a continuing increase in the level of function, and a significant recovery from even quite severe initial deficits, even after the initial post- trauma window of opportunity has already closed for other interventions.

Tourette Syndrome

Tourette Syndrome is defined by the presence of persistent but variable motor and vocal tics. However, it should not be discussed without consideration of Obsessive-Compulsive Disorder. Both may have essentially the same physiological mechanisms, and they are usually both observed in the same subjects (Rapoport, 1990). The extent of possible remediation of these symptoms with EEG training varies widely depending on the severity and the length of time over which symptoms have existed. The passage of time does appear to make this condition more intractable.
One of the confounding issues in addressing TS is the fact that the individual is often aware of considerable benefits that he or she derives from this 'disorder.' Hence, it is not so much a matter of 'curing' Tourette's as it is a matter of dealing with some of its disagreeable attributes. Tourette Syndrome is best regarded as a spectrum disorder where a basic neurophysiological hyperexcitability (in the orbito-frontal cortex-to cingulate gyrus-to caudate/striatum-circuit [Stein, 1996]) may manifest in a variety of symptoms including ADHD, addictive propensities, thrill-seeking behavior, hypersexuality, rage behavior, in addition to the defining tics, and the usually comorbid OCD (Comings, 1990). However, the person may also be aware of heightened mental acuity, may command considerable resources in terms of creativity, and may be quite conscious of the positive aspects of obsessive behavior in terms of reaching personal goals. He may even experience the tics as intrinsically rewarding release experiences.

Hence, a therapeutic approach to Tourette Syndrome is inherently problematic. The typical client, therefore, is a child whose parents are concerned about the tic behavior or other problems such as precocious sexual behavior. And in the early phases of Tourette Syndrome, the condition appears to be quite remediable with respect to tics, OCD, and the correlated symptoms listed above. As the natural course of the condition unfolds over the years, it becomes progressively more difficult to deal with, and this is probably due as much to the conflicts within the subjects respecting the goals of the training as to the inherent intractability of mature complex tic behavior.

Narcolepsy and Sleep Apnea

Whereas most ordinary sleep disorders respond quite dramatically to the EEG training, narcolepsy and sleep apnea remain a considerable challenge. One is tantalized by the fact that the training can clearly have a favorable effect, but the outcome is highly variable, and presently unpredictable. In both instances, however, the benefit likely to be derived is such that it justifies the attempt to train.

Sleep apnea is currently recognized in two forms: central sleep apnea, and obstructive sleep apnea. Central sleep apnea is recognized to be a problem of sleep regulation attributable to the central nervous system, whereas obstructive sleep apnea is thought to be more peripheral in origin, being often associated with an excess of weight in the individual. From the standpoint of EEG biofeedback training, the distinction between the two types of sleep apnea loses a good deal of its significance. Clearly there is no actual physical obstruction in the airway passages, since the person can breathe perfectly well during the day. The obstruction arises at night because of relaxation of the relevant musculature in the back of the throat. The latter however does not operate autonomously as supposed, but is clearly also under the management of the central nervous system. Hence it is also accessible to operant conditioning of the mechanism which governs motor tone.

The training has been found to be helpful with both kinds of sleep apnea, but only a small number of cases have been seen to date in our practice. No systematic studies, supported with all night polysomnography, have been done. The reports of improvement would be considered anecdotal at this point. However, these findings do not stand alone. They are generally supportive of, and in turn supported by, the view that sleep apnea is one of the vulnerabilities of ADHD-residual type. The remediation of arousal disregulation in adult ADD subjects apparently involves, among its various benefits, also a heightened threshold of onset of apnea episodes and better maintenance of muscle tone during sleep. Confirming evidence is to be found in the rather commonplace finding that snoring may respond to EEG training. Snoring involves the same issues of management of muscular tone as are at issue in sleep apnea.

Narcolepsy is a tough clinical challenge. We have seen a small number of cases in our practice over the years. It is regarded in our model as an instability in brainstem-regulated arousal mechanisms, much like migraines. In fact, considerable migraine comorbidity exists among those with narcolepsy. The problem can often be traced to brain-stem injury such as whiplash. Training is done with a protocol that is grossly similar to that for migraines and other instabilities. However, the training may need to be nuanced very carefully. At the outset, training for narcolepsy can actually trigger migraines in those who are susceptible. Eventually, greater stability against both can be achieved with the training.

Major Head Injury

The distinction between major and minor head trauma is a medical one. It is a question of whether there was a skull fracture or other major organic injury such as a hematoma. In the absence of such gross organic injury, it is referred to as minor head trauma, irrespective of symptom severity, as already mentioned. The reason, therefore, that major head injury is listed in the category of our most difficult challenges, relates to the variety in which major head injury manifests as a result of the specific organic injury, and because such organic injury may limit the extent of the recovery.

By virtue of such specific loss of function, much of the work needs to be directed to the evaluation and remediation of the specific deficits traceable to such injury. Many of the issues, however, are identical to those that predominate in minor head injury, and respond just as readily and quickly. A hierarchy emerges in which the general effects of head injury'major or minor'are treated with the standard protocols as a first order of business. The residual specific effects traceable to the organic injury are usually addressed last.

Also subsumed under this category are cases of near- drowning, of meningitis, of major birth trauma, of prolonged anoxia due to any cause, and other such major impacts on the brain. In such instances, there can be a significant loss of function: loss of motor control; loss of visual field; loss of urinary and bowel control; impacts on language function; etc. It is our observation that the training can be beneficial in all these respects; however, the extent of the recovery is highly unpredictable as to its rate and extent, and as regards the variety of training protocols required to achieve them.

Chronic Fatigue Syndrome

EEG biofeedback training has been found to be helpful with Chronic Fatigue Syndrome, or Chronic Fatigue Immune Deficiency Syndrome (CFIDS), as well as with its diagnostic cousin, Fibromyalgia. The benefits of the training are the most dramatic in those who are not totally disabled by CFIDS. However, nearly everyone can benefit to a certain extent from the training, particularly in the context of a multi- dimensional program of recovery.

The emerging understanding of CFS from our standpoint is that it is a "burnout syndrome" to which ADHD subjects, and Type A personality, are especially vulnerable. The particular trigger that finally manifests in chronic fatigue is variable, which is frustrating to those looking for specific medical causation. The subjective observation is that chronic fatigue and fibromyalgia sufferers often try their best to motor on despite their disability. Angrily, they rail against their disability, and by their persistent effort, may inadvertently militate against its remediation.

Herein lay the clue for EEG biofeedback training. CFS sufferers did not so much need beta training for higher energy level and a higher level of functioning. Beta training did indeed confer those benefits, but they were usually transitory, and often met with an adverse rebound later. CFS was not an ordinary depressive syndrome, although it had depressive features. There appeared to be in many CFS sufferers a considerable efforting in spite of their condition. Thus, EEG training became a matter of getting them to ease up on themselves and learn to work out of a more relaxed state. Hence, lower frequency (SMR) training was introduced, and now predominates in our approach to CFS.

Fibromyalgia has already been discussed in terms of chronic pain. It is observed that the pain component of fibromyalgia responds primarily to the higher-frequency (beta) training, to which it often responds quite readily. Other aspects of fibromyalgia such as fatigue and anger require the lower- frequency training, and may have more ambiguous outcomes.

In summary, chronic fatigue and fibromyalgia should be addressed in the context of a multi-disciplinary approach involving medical management, nutritional support, and other interventions. In such a context, EEG biofeedback can be a significant aid in recovery. Nevertheless, it should be made clear that there is no suggestion that EEG training addresses the core issues of CFS or fibromyalgia, which remain obscure.

Autoimmune Dysfunction

It is been a fairly consistent though quite remarkable observation that EEG biofeedback training can be helpful in the management of autoimmune diseases such as Crohn's disease, lupus, multiple sclerosis, Type I diabetes, and in some cases rheumatoid arthritis. There is no implication that EEG biofeedback in any sense addresses the core issue here of autoimmune disease. However, the training does frequently improve the level of function in afflicted individuals. Thus, in Type I diabetes we have seen reductions in symptoms traceable to peripheral neuropathy in long-term diabetics; we have seen reduction in the incidence and severity of lupus episodes; we have seen essentially complete symptom regression in Crohn's disease; other researchers using the same methods have observed significant remediation of M/S symptomatology in some cases; and we have observed diminution of pain in some cases of rheumatoid arthritis.
The level of clinical experience from which the above has been drawn is indicated in **Table 7**. The categories here may be overlapping. That is, a person may be counted in the ADHD category and also in the migraine category. Also, reference is to the key symptomatology that the person manifests, irrespective of whether established clinical diagnostic criteria are met. This means that in our clinical practice no such threshold is applied as to whether a person qualifies for training; hence there is no need to make a specific determination regarding diagnostic threshold criteria. In actual experience, it is found that the persons referred for training manifest rather severe forms of these various disorders. For many, coming to biofeedback is the end of a very long road of unsatisfactory remedies. Hence, there is usually very little question about their meeting clinical criteria. More than likely, it would be a matter of multiple diagnoses.

Table 7. Conditions Impacted Favorably with EEG Biofeedback Training.

Entries are ordered by the amount of experience we have had with each condition.

400	Attention Deficit Disorder
	Childhood sleep disorders
200	Childhood depression: Dysthymia
	Anxiety Disorders and Panic Attacks
	Chronic headache; migraines and tension headaches
	Specific Learning Disabilities; Dyslexia
	Hypoglycemia; Dysglycemia, Type II Diabetes
100	Attention Deficit Disorder: Residual Type
	PMS; menopause
	Chronic Pain
	Conduct Disorder; Oppositional-Defiant Disorder
	Minor traumatic brain injury

	Adult sleep disorders
50	Bruxism
	Primary Unipolar Depression
	Tourette Syndrome; Tics; OCD
	Chronic Fatigue Syndrome; Fibromyalgia
25	Epilepsy
	Addictions
	Prenatal Substance Exposure
	Major Head Injury
	Tinnitus
	Autoimmune Dysfunction
	Bipolar Disorder
	Eating Disorders
10	Stroke
	Chemical Injury; Multiple Chemical Sensitivities
	Autism; Asperger's Syndrome
	Cerebral Palsy
	Post-traumatic Stress Disorder
<10	Nocturnal Myoclonus
	Alzheimerþs and Non-Alzheimer's dementia
	Rumination Syndrome
	Multiple Sclerosis
	Reflex Sympathetic Dystrophy
	Narcolepsy; Sleep Apnea; Restless Leg Syndrome

Summary

In the above we have made a case for global efficacy of EEG training for a broad variety of brain-based disorders. By building on early models of this work by Sterman and Lubar, a comprehensive view has emerged in which EEG training influences fundamental rhythmic timing mechanisms by which the brain manages activation, arousal, and affect. By challenging EEG activity in specific frequency regions in a training paradigm, normalization of activation and arousal mechanisms can be brought about, and cortical stability enhanced. Broad benefit for the organismic functioning has been demonstrated. Once the power of this technique is fully appreciated, the comprehensive reach of this 'new' intervention will result in a reframing of psychopathology in terms of a few key deficits in basic regulatory functioning which, by virtue of the centrality of rhythmic mechanisms, is amenable to redress with EEG biofeedback training to promote neuroregulatory capacity.

A case has been made for a very parsimonious set of protocols by which most of these objectives can be achieved. These protocols have been found to address the specific failure modes of the left and right hemispheres, and to address problems of inter-hemispheric communication. Consistency with the implications of pharmacological interventions for common disorders is indicated. The results alluded to here in cursory and summary fashion portend a significant new

capability for remediating brain-based disorders which have been refractory to other interventions, and which represent a staggering loss of human potential, as well as being a considerable drain on health care resources.

Epilogue

The above review of various clinical conditions is too cursory to satisfy the discerning scientific mind, and raises more questions than we attempt to answer. For this we apologize. The prevailing constraints do not allow us to be both comprehensive and thorough. The intent is to draw the interest of the researcher as well as the clinician to this fascinating new field. If the reader's credulity has been challenged too severely, and if the above clinical findings were therefore to be collectively rejected, that would certainly be understandable, but still unfortunate. In any event, the field has grown in the face of a prevailing skepticism, and will continue to do so. The technique of EEG biofeedback is most humane in its implications, because it offers help with those mental disorders which interfere most severely with our human capacities. It deserves a full measure of attention from both the clinical and research communities.

It is unarguably a tragedy that in our adversarial health care system a new intervention is seen first and foremost as a nuisance and an intrusion, if not an outright fraud, by third- party payers; that the scientific community is so utterly invested in its prevailing system of thought as to be unable to appraise the new claims objectively; and that those who are in the greatest need of this new intervention are unable to afford it.

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Biographical Material on Authors

Siegfried Othmer received his Ph.D. in physics at Cornell University in 1970. After a first career in aerospace, he and his wife Susan were drawn to the field of EEG biofeedback in 1985 because it had been profoundly helpful to their epileptic son. Siegfried Othmer is Chief Scientist at EEG Spectrum, a clinical service delivery organization and network of EEG biofeedback clinicians which has been in existence since 1988. Susan F. Othmer received her B.A. in physics at Cornell, and pursued her Ph.D. in neurophysiology there and at the Brain Research Institute at UCLA. Her Ph.D. work was aborted because of their epileptic son, but eventually this son drew her back to the field of brain research. Susan Othmer is Clinical Director at the home office of EEG Spectrum. David A. Kaiser obtained his Ph.D. at UCLA in research psychology, with a special interest in EEG phenomenology. He is a cognitive neuroscientist at EEG Spectrum.

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Figure 1. TOVA test results for four dependent measures for 342 subjects. A large effect size is indicated for those in severe deficit on any of the four subscales.

Figure 2. Individual pre-post test data for the impulsivity measure, for those with starting values of less than 100 in standard score, are shown rank-ordered in terms of starting value. A general tendency toward improvement, irrespective of pre-training status, is noted.

Figure 3. Average pre and post WISC-R Scores for all 15 study subjects.

Figure 4. Pre and post data for the Benton Visual Retention Test.

Figure 5. Pre and post test data for the Tapping Subtest of the Harris Tests of Lateral Dominance. The average increase in tapping performance is 20%; the median increase is 40%; and three subjects increased tapping speed by over 100%.

Figure 6. Pre and post values of right/left ratio in tapping performance. We observe a tightening of the distribution and a depletion of mixed dominance.