

INFORMATION ABOUT NEUROFEEDBACK AND THE PHYSIOLOGY UNDERLYING ITS EFFECTS

Neurofeedback acts as a catalyst to release and restore the brain's own capacity for self-regulation. Once released, the brain has shown startling capacity to restore its own clarity, sense of ease, ability to initiate activities, to organize, to reduce muscle paralysis after stroke, and to regulate depression, explosiveness, and anxiety.

Neurofeedback is a form of biofeedback of brain wave (EEG) activity. It is a noninvasive procedure that involves monitoring and analyzing EEG signals, and using the EEG information to guide the feedback. While there are many kinds of neurofeedback, the kind of neurofeedback considered here delivers the feedback down the same EEG leads that carry the brain wave signals to the EEG and to the computer.

The feedback is not an electrical charge, but an electromagnetic field like the ones that carry radio signals to our radio antennas. The strength of the feedback signal, an FM-radio signal, is less than the FM-radio signals already surrounding us. This signal produces a measurable reaction in the EEG without conscious effort from the individual receiving the feedback. The EEG signals that are recorded at the scalp control the feedback. Neurofeedback may change the neurochemistry, which, in turn, changes the EEG.

Neurofeedback evokes behavioral and subjective reactions, as well as changes in EEG amplitude, specifically EEG slowing. Dr. Ayub Ommaya, George Washington University neuro-surgeon and TBI researcher concurs with current thinking about possible modes of action: the feedback disrupts the EEG activity that reflects dysfunction. This interruption of the brainwave state precedes the brain's reorganization of its own functioning, resulting in behavioral and symptomatic changes.

Neurofeedback uses a feedback frequency that is different from, but correlates with the dominant brainwave frequency. When exposed to this feedback frequency, the EEG amplitude distribution lowers in power, and the average dominant frequency shifts upward. The result is lower amplitude slow waves, and more flexible functioning. At other times, when the EEG amplitudes have too little variability, rises in amplitude are observed.

Shifts in EEG amplitude and reductions of symptoms are often observed as the brain is able to reorganize its own functioning. The more severe injuries require longer treatment. For severe damage requiring many treatments, families have been trained to use the equipment at home and administer it regularly. The average treatment time is 15 sessions across all conditions.

Barring a new head trauma, or engagement in painful bodywork or a somewhat premature overindulgence in life in an effort to make up for lost time, there have been few if any relapses or regressions after treatment. Two instances have been noted in which patients were very near the end of treatment and new traumas caused a return of symptoms. These were more easily treated than symptoms from their initial trauma; and both regained their former level of functioning.

The first published study of neurofeedback was for treating the EEG slowing in fibromyalgia. [Canadian Journal of Clinical Medicine, June 1998] An NIH-funded study applying neurofeedback to brain injury was published in the Journal of Head Trauma in June of 2001. [16(3): 260-274] Co-Principal Investigators were Nancy Schoenberger, Ph.D. of the Kessler Institute, Mary Lee Esty, Ph.D., Chevy Chase MD, and Len Ochs, Ph.D. developer of this form of neurofeedback. The spectral characteristics of neurofeedback energies were measured at Lawrence Livermore Laboratories, California, under a privately funded grant. A third study is just finishing at the Rush-Presbyterian-St. Luke's Medical Center, Chicago, IL, to research the effects of neurofeedback on the EEG slowing associated with fibromyalgia. We are finding a virtual remission of fibromyalgia (pain, energy, cognitive, mood, balance-coordination problems) in 79% of people who complete treatment. The Dana Foundation included coverage of Neurofeedback-TBI work on National Public Television.

Physiology underlying both the functional neurological problems, and their resolution

Many failures to function involving the brain, are thought to be structural. There may be a variety of neurochemical functional problems at the *cortical* level that prevent functioning, regardless of the existence of subcortical and/or cortical structural problems. Furthermore, assisting the cortex to function better can often compensate for many structural, and even subcortical, problems. It may be said that accompanying many neurological functioning problems is a permeability of the cortex to subcortical electrical activity, that does not appear when functioning is good.

Too much waking frontal Alpha, or too much Delta or Theta, may point to the failure of the cortex to inhibit these subcortical potentials. Another way to put this is that relatively large amounts of electrical activity are recordable at the scalp when the cortex fails to inhibit these subcortical potentials. When the cortex works properly, it does inhibit the subcortical activity, and it (Delta, Theta, frontal Alpha) can then be recorded only at miniscule levels at the scalp. Inhibition effected by the cortex sharply reduces the appearance of the subcortical activity at the scalp. It is as if the cortex *loses* its inhibitory powers and becomes *permeable* to the subcortical activity when traumatized, overloaded, and/or inadequately developed. The concept of cortical permeability becomes important in the strategic considerations about where to place time, money, and effort resources to rehabilitate a person suffering from neurological trauma. That is, although the locus of the problems may be subcortical, stimulation of the cortex toward better functioning may bring about amelioration of the problem.

My reason for asserting the above is based on the following observation: Potentials of relatively sizeable magnitudes are recordable at the scalp in the form of "EEG slowing" when an individual shows functional impairments of mood, cognition, movement, energy, and pain-without-local or regional pathophysiology. ("EEG slowing" refers to the presence of high amplitude, low frequency brain waves.) On the other hand, potentials of much smaller amplitude are recordable at the scalp when the person functions well. In other words, sizeable scalp potentials accompany poor functioning, while they are detectable in much smaller amounts during good functioning. The

evidence used to consider the cortex as the site of the problem is the *absence of higher functioning usually attributed to the cortex*, and, in particular, the ability of the cortex to exercise inhibition. Cortical inhibition is one of the last of our evolutionary powers to develop, as it is one of the last powers to develop through an individual's life span. Absent these powers after some trauma(s), and the individual appears immature in his or her developmental abilities.

Because EEG slowing is inhibited under good and/or wakeful conditions doesn't mean that it goes away. Delta, Theta, and frontal Alpha are all perfectly recordable with high amplitude all the time, *subcortically*. This is visible through the use of indwelling electrodes. Activity in these bands is always present, if not visibly so via surface recording. It is functional cortical failure of one kind or another that makes activity in one or more of these bands visible at the scalp.

When the cortex fails, then one, some, or all bands of activity do show up much more significantly at the scalp. It may be that a particular layer or structure in the cortex is responsible for the inhibition of a particular band, and that the appearance of substantial amplitude in one band reflects a disabling of a particular inhibitory cortical network. For example, when Alpha cortical inhibitors fail, Alpha shows in the recordings, and as a result of this kind of failure, anxiety-type disturbances are observable in the behaviors of the person. In a similar fashion, if the Delta or Theta cortical inhibitors fail, activity in these bands shows up in the recordings.

It is reasonably well understood that the brain attempts to protect itself by secreting inhibitory neurotransmitters such as GABA. It is hypothesized that much stimulation from ordinary activities and states such as trying, anxiety, learning, long neurofeedback sessions, or proprioception may evoke large potentials measurable at the scalp, and perpetuate the problems. It is further hypothesized that the inhibitors designed to protect the cortex and cortical functioning actually prevent the cortex from receiving the useful stimulation that it needs to function. The cortex is thus inhibited in its functioning, leading to the behavioral, energetic, cognitive, motor, mood, and pain dysfunctions outlined above.

Deprived of its ability to function as an inhibitor of subcortical activity, higher function is impaired. Neurofeedback may disturb and remove the inhibition *upon* the cortex, and thus permit the cortex once again to function, which means, to promote inhibition *by* the cortex, and the return of higher and more adaptive functioning.

When we look at topographic maps, each which shows the amplitude activity within certain frequency band ranges, we may actually be looking at the degree of failure of the Delta, Theta, frontal Alpha, etc. inhibitors, respectively, each of which releases a specific kind of cortical permeability. As each one of these inhibitory subsystems returns to functioning, we also see the return of energy, clarity, flexible and appropriate mood, motor integration, and system quietness.

I believe at this point, that neurofeedback works by inducing perturbations and disturbances in the neurochemical blockades of the cortex, by creating a different kind of

conduction demand in the tissues of the cortex. And when conduction and communication is facilitated, the inhibitory blockade of the cortex is released and we see a return of function.

The intensity, or dose, of the feedback becomes critical in at least three instances. The first instance is when the patient is extremely reactive to stimulation of many kinds, from food, to medication, to atmospheric conditions, to social slights from others. The second instance occurs in the second phase of head injury rehabilitation work using neurofeedback. The patient becomes very much more responsive to the feedback in the second phase of treatment; consequently smaller treatment doses are needed. The third concerns fatigue syndrome patients with profoundly low surface EEG amplitudes. The neurofeedback course of treatment for these patients is not well understood. In each of these cases dosages must be drastically reduced in the early stages of treatment, and increased very slowly as the person becomes hardier and can tolerate them.

In general, minimal doses of feedback may help re-tone the brain's reactions systems, and, at the same time, be small enough that they by-pass the brain's triggers to defensively overreact, allowing the feedback signals to stimulate the cortex. If the dose were too high, the brain could over-react in a defensive way, block useful stimulation to the cortex, and thus perpetuate the inhibition of the cortex.

Feedback dose is a function of both the intensity of the feedback field strength and the duration of the exposure to the feedback signal. If the dose does not match the sensitivity and responsiveness of the individual receiving it, the feedback will affect the individual the way most of life affects him or her: it becomes an overdose and degrades the functioning of the person. The most familiar side effects of overdose of neurofeedback are (1) irritability, (2) fatigue, or (3) no change. These effects are temporary, and may last from a few hours to a week, depending on the sensitivity and hardiness of the individual. If the dose is *small* enough, the feedback does not cause an over-reaction, and stimulates the cortex to function at a higher level. This is measured by a decrease in EEG slowing, as well as obvious increases in performance ease, complexity, functioning flexibility, and adequacy. Dose is regulated by duration of exposure to feedback. Typical doses range from 1 to 60 seconds per session, and averaging 7 seconds per session. Treatments administered in 45-minute sessions, most of which is used to assess patient progress, provide a context for their experience of change, answer questions, and to talk about expectations for the upcoming week.

The process of recommencement of self-regulation continues after treatment.

Treatment considerations with special populations and conditions

Neurofeedback only re-starts the self-regulatory process for the brain. Initially the changes are brief, but become more durable and lasting as the self-regulation is restarted in more areas of the brain, and longer-term problems (e.g., chronic frontal Alpha) are managed. While a number of conditions are mentioned below, this form of neurofeedback is not specifically a treatment for condition or symptom. It is a general process that allows the brain to function more flexibly and to communicate better with itself. The brain is able to accomplish the following *by itself*, between sessions:

Population

Self Regulation Effects

Fibromyalgia

Reductions in symptoms such as pain, headaches, depression, as well as cognitive, mood, and energy problems. Treatment success is 77% if there are no active structural problems.

Doses usually are 1 second/session at first until they start to improve. Each time there is further improvement the dose may be doubled.

Stroke

Recovery of sensation, movement, muscle strength, motor control, and of clarity to various degrees, depending on the severity of any damage.

Wherever there is spastic paralysis there will initially be approximately a week of pain in the affected muscles, followed by spastic jerking, until sensation and controlled movement reappear, usually commencing after a week.

Patients become angry and push away caregivers and therapists for a few days as they clear from medication reductions.

Traumatic Brain Injury

Recovery of ability to take in information, improve short-term memory, organization, sequencing, prioritizing, sensory discrimination, initiation, confidence, assertiveness, and of sense of humor.

Reduced depression and explosiveness. Better stamina during the day as well as better sleeping at night.

Average treatment of previously high functioning patient is 6 sessions.

Autism

Less fearful. Reduced explosiveness. More social. Better communication skills. Better academic skills. More graceful and balanced: less autistic gait. Transient incontinence in young children passes in a week. Parents find their children continue to improve for years, using a system on a child at home. Autistics have been shown to be able to usefully accept much larger doses, up to 20 minutes per session.

Depression	Reactive depression average treatment is 3 sessions. Endogenous depression has a 15-session average treatment length.
ADD/ADHD	Average treatment length is 15-sessions. Improved ability to initiate, move from task-to-task at the appropriate time, and to complete tasks. Reduced irritability and opposition. Increased ability to rest.
Migraines	Average treatment is 3 sessions unless status migrainous and opiate detoxification is necessary.
Seizures/Tourette's	Reduction or elimination of seizures and tics, and reductions or elimination of medication. Some transient disinhibition of motor tics may occur, leaving the person at higher risk for falling and breaking bones.
Early-Stage Progressive Dis.	Early-stage Parkinson's, MS, and Alzheimer's show greater steadiness, balance, and strength, and less depression. Monthly brush-ups needed. Goal: to enhance independence and functioning until the disease takes its course
Adolescents	Increased displays of closeness and affection toward parents, reduced negativism and opposition, improved ability to initiate, change, and complete tasks; greater cooperativeness

It is important to remember that each of these specific improvements in functioning comes about because it is in the nature of the brain to function in this way. The improvements are neither non-specific, nor micromanaged. They are, in fact, multi-specific, because the brain is a multi-specific organ. Improvement in the functioning of this multi-specific organ reflect improved responsiveness in the systems of the brain, more differential reactivity, greater inhibition exercised by the cortex, and an improved ability to idle with less background neuronal-electrical activity, i.e., EEG slowing.