History of neurofeedback

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History of the Development of Neurofeedback

By Siegfried Othmer

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The story begins with the discovery of the EEG by Hans Berger and moves quickly to the early research by M. Barry Sterman that led to the development of neurofeedback in application to seizure disorder. The second application area to be developed was ADHD, using similar methods. The subsequent proliferation of the method to cover a variety of psychopathologies will be traced: the anxiety-depression spectrum, pain syndromes, sleep disorders, traumatic brain injury, emotionally-based disorders, psychological trauma, and the dementias. Paralleling these clinical developments was necessarily an evolution of theoretical models. These had to become more general and inclusive over time to accommodate the clinical findings. Fortuitously, the emergence of neural network models generally, and of intrinsic connectivity networks in particular, provided a framework for understanding why particular approaches, even specific protocols, were differentiating themselves in terms of clinical effectiveness.

The development of infra-low frequency training is traced as a natural evolution of mechanisms-based training. The parallel development of the use of neurofeedback as an inducement to state shifts to facilitate the resolution of psychological trauma is also traced. This method has distinguished itself particularly in application to psychological trauma and to addictions.

The Scientific Antecedents of Neurofeedback

The discovery of EEG biofeedback in the 1960's was dependent on three principal antecedents. These were the discovery of the EEG by Hans Berger, the development of classical conditioning by Ivan Pavlov, and the establishment of operant conditioning under B.F. Skinner. The original discovery of the EEG was the result of determined effort by Hans Berger over the course of nearly two decades. He was set upon this path by a personal experience of mental telepathy, which redirected his career into medicine, where he was determined to find a physiological basis for the phenomenon. In this enduring side project, undertaken while serving as clinical director of a neurology and psychiatry clinic, he failed. But despite the limitations of primitive electronics he was able to find the miniscule EEG signal. In the face of his own uncertainties, it took another five years for him to publish his findings, which occurred in 1929, and then it was his colleagues' turn to be skeptical. It took another five years before Adrian and Matthews replicated his work in England, which served to change the conversation. These authors were also the first to observe the effects of synchronous visual stimulation on alpha band activity in the EEG. Berger had first identified this prominent EEG rhythmic bursting pattern, which subsequently came to be labeled the Berger rhythm.

Berger must be considered one of the pioneers of the nascent discipline of psychophysiology, and he is credited with coining the term. Although he did observe EEG phenomenology in epilepsy, he was not oriented toward the medical utilization of the EEG. For decades, as a matter of fact, the principal utility of the EEG in neurology remained with those features that were obvious on mere visual inspection of the clinical EEG. This had the effect of consolidating a rather modest appraisal of the utility of the EEG, one that would prove difficult to dislodge. The usefulness of the EEG in psychophysiology remained modest as well. The full exploitation of the EEG would have to await the availability of new tools of both measurement and of analysis.

Ivan Pavlov received his Nobel Prize for his work on the digestive system of dogs rather than for his work in classical conditioning. But one set the stage for the other. In studying salivation he observed that it often occurred well before the food arrived. The classical conditioning experimental design placed these observations in a rigorous framework. He called the anticipatory salivation the conditional response. Pavlov also studied the anticipatory reaction to aversive stimuli such as foot shocks, and in one design combined both food reward, signaled by one frequency, with the occasional delivery of a foot shock, indexed by another frequency. When the two frequencies were slowly brought together so that the dogs could no longer reliably distinguish between them, the dogs tended to give up on the whole experiment. Some of them even went to sleep.

The Foundations of Neurofeedback in Animal Studies

Six decades later, Pavlov's study inspired M. Barry Sterman, a research psychologist and sleep researcher at the UCLA School of Medicine and at the Sepulveda Veterans' Administration Hospital, in his own investigations of sleep onset. Pavlov's dogs had absented themselves from the task even in the face of the aversive foot shocks. Sterman took this as a paradigm for the voluntary withdrawal of behavioral responding that we all undertake once the head hits the pillow with the intention to fall asleep.

An operant conditioning design was set up in which cats were trained to expect food reward upon a bar press whenever a light came on in their experimental chamber. The trained cats were then taught that whenever a tone was present, food would not be available. They would simply have to bide their time until the tone went off. A behavioral state had thus been induced in which cats had to withhold their natural tendency to pounce on the bar whenever the light came on.

The cats had all been fully instrumented for EEG measurements with electrodes surgically implanted beneath the skull, but external to the dura. These measurements now revealed a bursting rhythm at sensorimotor cortex that Sterman named the sensorimotor rhythm. It was present only during periods of motoric stillness, and bore a strong resemblance to the sleep spindle that characterizes Stage II sleep in both cats and humans (Wyrwicka and Sterman, 1968). Just as the alpha spindle tends to occur during eyes-closed conditions, the SMR spindle occurred only during periods of de-activation of the motor system. It was centered at the slightly higher frequency of nominally 13 Hz.

Once this association was firmly established, Sterman made the food reward conditional on the appearance of such an SMR-burst. All the cat had to do was to compose itself in a state of quiet anticipation. As hunters, this comes naturally to them. Beautiful learning curves were acquired over a period of some weeks to months. Reinforcement on the waking SMR spindles was found to increase sleep spindle density and improve sleep efficiency (Sterman, Howe, and Macdonald, 1970). Controlled experiments followed, with both a balanced reversal design and an extinction design, to prove unambiguously that operant conditioning of the SMR bursting pattern had occurred.

The reversal design was the first to demonstrate that suppression of the SMR bursting response could also be trained. This must necessarily occur by an indirect pathway. The cat is fed at regular, brief intervals as long as SMR bursts are not observable. Even this task could be readily learned. The behavioral response to the training was that the cats became physically restless and twitchy, indicating a heightened level of motoric excitability, in contrast to the SMR-conditioned cats who exhibited a general calming. The sleep of the SMR-trained cats also changed. It became more efficient, and the sleep spindle density increased with the training. In fact the experiments relied upon the sleep spindle density as the salient index of change, simply because there was no chance that it might be affected by the cat's mood of the moment.

The extinction design made it clear that the conditioned SMR response had to be understood as a brain response rather than as a volitional response on the part of the cat. All the cat could contribute to the project of obtaining food was its motoric stillness, which was not sufficient by itself. This observation is foundational to the entire field of neurofeedback. The process must be understood in the perspective of the brain and of its engagement with the sensory environment, one that now also includes the feedback signal that bears directly on its own activity.

Subsequently, Sterman had the opportunity to research the low-level effects of the toxic rocket fuel monomethylhydrazine (MMH), for which his instrumented and well-characterized cats were highly suitable. The MMH effected the depletion of GABA, the primary inhibitory neurotransmitter, over the course of about an hour, after which the cats went into seizure. The animals responded with a high degree of uniformity until the end stage, at which point the population bifurcated, with a subpopulation showing substantially delayed seizure onset.

The seizure susceptibility was completely predicted by their assignment to cohort in the reversal design that had taken place many months earlier. Those who had received the SMR reinforcement last were selectively resistant to seizure onset. There must have been a carryover effect from the earlier training. Learning must have occurred that was not subject to the usual extinction that attends operant conditioning designs after reinforcement ceases. This experiment, which was quite unambiguous in its implications, was the signal experiment that established the direction and thrust of development in the field of neurofeedback.

Quite unintentionally the above experiment met all the criteria one would impose on a fully controlled design. For their part, the cats were not subject to a placebo response. And the researchers were obviously not biased in that they were totally blind-sided by the outcome. The bifurcation of the

response was problematic with respect to the objectives of the research. The experiment had been a totally placebo-controlled and fully blinded design. And the implications of the findings were not equivocal. In follow-up experiments, cats could be characterized in terms of their native seizure susceptibility, trained in the SMR paradigm, and then re-tested to evaluate their newly heightened tolerance. The results for sixteen cats (8 experimentals, 8 controls) were subsequently published (Sterman, 1976). Two of the eight cats remained entirely seizure-free, despite having shown all the prodromic symptoms. These results were subsequently confirmed by studies on rhesus monkeys as well (Sterman, Goodman, and Kovalesky, 1978).

Evaluation of SMR-Reinforcement with Human Subjects

Seizure Disorder

Sterman then turned his attention to human trials. An employee in his laboratory suffered from nocturnal seizures and offered to be the first trainee. During training for only 34 sessions over four months a near elimination of seizures was observed, which led promptly to a publication (Sterman and Friar, 1972). In the event, it took several years of training to render the person entirely seizure-free, to the point where she qualified for a California driver's license. An exploratory outcome study on four participants, all of whom benefited from the training, was published in 1974 (Sterman et al, 1974). By 1972, Sterman's research had attracted the interest of Joel Lubar, Professor of Psychology at the University of Tennessee in Knoxville. His research group published an outcome study on 8 participants, 7 of whom benefited from the training (Seifert and Lubar, 1975; Lubar and Bahler, 1976). Controlled studies followed, using either the reversal design or sham training as an ostensibly neutral control condition. All participants in this research were medically refractory seizure patients whose condition was stable. Whereas the initial target was motor seizures, eventually the scope encompassed complex partial seizures as well.

The first study utilizing an ABA design involved eight participants (Sterman & Macdonald, 1978). Two reinforcement bands were evaluated: 12-15Hz, and 18-23 Hz. For the reversal phase, Sterman used reinforcements in the 6-9 Hz band. This avoided the lower EEG frequencies which, if reinforced, could potentially aggravate seizures. Results were remarkable. 6 of 8 improved significantly in their seizure incidence, with an overall improvement of 74%. This despite the fact that half the time had been spent training in the wrong direction, where seizure incidence did indeed mostly get worse. One of the eight became entirely seizure-free, another very nearly so.

A second such ABA study was published by Lubar's group (Lubar, Shabsin, et al, 1981). It was the first to use a double-blind design. This study also involved eight participants. The reversal phase utilized a 3-8 Hz bandpass. As feared, seizure incidence could be exacerbated in this manner, and indeed one participant had to be withdrawn from this phase of the training for that reason. Average reduction in seizure incidence in the cohort was only 35%. Referenced to the 5 of 8 who were considered 'responders,' the mean reduction was 49%, a clinically significant improvement.

In Sterman's sham-controlled study, 24 participants with complex-partial seizures were divided into three groups: a passive seizure-tracking group, the sham-training group, and the veridical feedback group. After an initial six-week training program, at a rate of three sessions per week, the two control groups were given the chance to train for another six weeks. All were weaned off the training over the course of four weeks, and a six-week follow-up period was allowed for. An overall improvement in seizure incidence of 60% was found in post-testing (Sterman, 1984). The study also included extensive neuropsychological and neurocognitive evaluation, and these results were published subsequently (Lantz and Sterman, 1988). Those who improved the most in terms of seizure incidence also tended to improve more on the mental skills testing, as well as on the MMPI. Results also correlated with improvements observed in the clinical EEG. Of the 17 who exhibited typically abnormal EEG patterns, nine normalized their EEGs, and of this subset, three brought their seizures fully under control.

In this same time frame replications were also undertaken as far away as Scandinavia and Italy. There appeared to be a groundswell of interest in the method for a time. The results of all the early studies were reviewed by Sterman in a special issue of the Clinical EEG Journal focusing on neurofeedback (Sterman, 2000). Some 24 studies were evaluated, and these collectively involved some 243 participants. 20 of these were group studies, and 13 of those included competent controls. Collectively, 82% of all the participants improved their seizure incidence by at least 30%, with the average improvement being greater than 50%. A more recent reflection on the status of this clinical approach was published in 2006 (Egner and Sterman, 2006).

A meta-analysis of the epilepsy studies has brought the appraisal up to date (Tan, Thornby, Hammond et al, 2009). Some 63 studies were evaluated for inclusion, but only ten survived the screen. These involved some 87 participants. The analysis confirmed 'significant' reduction in seizure incidence. The review included one study that trained only on the slow cortical potential (Kotchoubey, Strehl, Uhlmann et al, 2001). Strictly speaking, then, the review consolidated the case for the use of EEG-derived cues in a behavioral strategy of seizure reduction. This relatively recent meta-analysis reflects the diversity of approach that came to characterize the field. This is described further below.

ADHD

A second principal area of clinical interest developed early around what is now called Attention Deficit Hyperactivity Disorder, but at the time was still thought of mainly in terms of hyperkinesis. This thrust was led throughout by Joel Lubar, Professor of Psychology at the University of Tennessee. This interest was initially kindled by the observation that a child undergoing training of seizure control also experienced a subsidence of his hyperactivity. (Lubar and Bahler, 1976) This observation was confirmed with other such cases. Since the training was targeting the motor strip, it seemed reasonable to assume that hyperactivity was being tamed as well. Formal evaluation followed with children who were not afflicted with a seizure condition.

The first case study was soon published (Lubar and Shouse, 1976). It was postulated that EEG training would be helpful with those children who were responsive to Ritalin, in which case the training would be expected to yield additive benefits. The study involved a 14-year-old who was placed on 10mg

of Ritalin for the duration. 6 no-drug and 6 drug-only baselines were obtained on behavioral and EEG assessments. A training sequence of 78 sessions of SMR reinforcement, combined with inhibition on excessive 4-7 Hz activity, was followed by 36 sessions of reversal phase training, which in turn was followed by another 28 sessions of the design protocol. Placement was bipolar on left hemisphere sensorimotor strip.

Learning curves were acquired for the incidence of rewards relative to baseline conditions, and electromyographic (EMG) activity at the chin was tracked as an index to muscular tone. Both reward incidence and EMG trends reflected the prevailing protocol to a statistically significant degree. Over the sessions, reward incidence increased by factors up to three or four over baseline. It regressed equally strongly during the reversal phase. Regression was also noted during a two-week break in the training.

Some 13 behavioral categories were tracked by two independent observers monitoring classroom behavior. Eight of these were found to be responsive to the training. Six of these categories had already shown improvement with Ritalin, but further improvement was observed with the EEG training, and countertrends were observed during the reversal phase. Five additional categories included four social behaviors plus self-talk, and these showed no improvement with the SMR training. In fact, benefit that had been observed with Ritalin was reversed with the training.

The above was the first of four cases treated using the same study design (Shouse and Lubar, 1979). Two children responded much like the first, and one was non-responsive to Ritalin as well as the EEG training. In a final phase added to the program, the three children were successfully weaned off the Ritalin while continuing to train, and behavioral gains were maintained. Years later, Lubar revisited a number of clients whom he trained during those early years, and found that the gains had been retained over the longer term (Lubar, 2003).

Other studies followed. Tansey was successful in remediating a case of hyperactivity, developmental reading disorder, and ocular instability with a sequential combination of EMG and SMR training (Tansey and Bruner, 1983). He utilized a placement on the midline. Normalization of highly elevated EMG levels was achieved, and substantial increases in SMR amplitudes were observed over the course of twenty training sessions. Success was achieved with respect to all three symptoms, and status was maintained over a two-year follow-up period.

Learning Disabilities and IQ

Lubar pursued the growing interest in application to learning disabilities with extended training of six boys (Lubar and Lubar, 1984). In this work, beta1 training (16-20Hz) was added for enhanced focus of attention and arousal regulation. All improved in their academic performance, and all exhibited learning curves with respect to SMR and beta amplitudes, EMG levels, theta-band amplitudes, and gross movement indicators.

The promise of benefit for learning disabilities eventuated in an investigation of SMR-training of eight mildly neurologically impaired children (Tansey, 1985). The average improvement in Full Score Wechsler IQ score (WISC-R) was found to be 19 points. If either verbal or performance IQ lagged the

other by more than 14 points, the deficited variable improved by an average of 40% more than the other. This epoch-making study was followed by one on 24 comparably impaired children. The average improvement in WISC-R score was found to be 19.75 (Tansey, 1990, 1991). Moreover, this was achieved with an average number of training sessions of 27, delivered at the rate of one per week. Both the number of sessions and the pace of training would now be considered sub-optimal, and yet the results were stunning.

Tansey's extraordinary findings led to a replication in our own clinic using a left-lateralized protocol and reinforcement at beta1 frequencies (15-18 Hz) rather than at 14 Hz on the midline. 15 children with ADD features and/or learning disabilities were trained just as if they had been in ordinary clinical practice. Independent testing and evaluation was relied upon exclusively (Othmer, Othmer, and Marks, 1992). Average improvement in WISC-R score was found to be 23 points. The profile of average change scores for the subtests of the WISC bore a remarkable similarity in the two studies. Strong improvements in performance were also registered on the Benton Visual Retention Test, the Wide Range Achievement Test, and the tapping subtest of the Harris Tests of Laterality. Not subject to quantitative assessment were the subsidence of oppositionality, general improvement in sleep quality among those where it was deficient, and the relief from persistent stomach and head pain.

Comparison of SMR-training with Stimulant Medication

Since stimulant medication was already recognized as a treatment for ADHD, the practical question faced by the medical practitioner is whether neurofeedback is in fact comparably effective. This essential question was answered in a study comparing 23 children in each arm (Rossiter and LaVaque, 1995). Comparison of outcomes was by means of the T.O.V.A. ® (Test of Variables of Attention), a continuous performance test (CPT). Results were comparable in both arms, with both methods showing essential normalization of inattention and impulsivity, accompanied by more modest improvements in mean reaction time, but with substantial improvement in variability.

Rossiter repeated the same design some nine years later, with 30 in each arm of the study. Results were better than they had been in 1995 (Rossiter, 2004, 2005). Both medication management and neurofeedback protocols had improved in the interim, and the two approaches were now even more closely matched. Most likely the neurofeedback strategies benefited from adding pre-frontal placements to the standard protocol. By now six such studies have been done, all showing essentially comparable outcomes for pharmacotherapy and SMR/beta neurofeedback (Fuchs, Birbaumer, and Lutzenberger et al, 2003; Monastra, Monastra, and George, 2002; Duric, Assmus, and Gundersen et al, 2012; Meisel, Servera, and Garcia-Banda et al, 2014).

The largest such study involved 100 participants (Monastra, ibid). The Lubar protocol was used. By 1991 Lubar had adopted the "theta-beta ratio" as a criterion of functioning in the ADHD population, based on the observation that the most prominent change observed in the EEGs of these children with training was a decline in theta-band amplitudes. An increase in SMR/beta amplitudes was not observed as consistently (Lubar, 1991). Lubar had also adopted the vertex, Cz, as a standard placement for the SMR training, on the basis that the theta/beta ratio was typically largest at this site.

Study participants were selected on the basis of the theta/beta criterion, among others. All were medicated to a level at which performance on the T.O.V.A. was optimized, with the result that both groups normalized their TOVA subtest scores. EEG training was undertaken with the neurofeedback cohort until the theta-beta ratio normalized. This required an average of 43 sessions. Over the course of training, behavioral variables normalized for the EEG training cohort, whereas they remained in the clinical range for the medication-only comparison group.

After a year of the above regimen, medication was discontinued for a week to allow the children to be evaluated under no-medication conditions. The neurofeedback contingent held gains on both the TOVA and the parent/teacher ratings, while the comparison group fell back into the clinical range on the TOVA, and remained in the clinical range on behavioral ratings. A clear advantage had been demonstrated for the neurofeedback approach using multiple independent criteria, and medication was not required to sustain the gains.

In 2009 a meta-analysis was published that reviewed the research status of neurofeedback in application to the ADHD spectrum (Arns, de Ridder, and Strehl et al, 2009). The unsurprising conclusion was that neurofeedback treatment for ADHD can be considered “Efficacious and Specific,” with large effect sizes for inattention (0.81) and impulsivity (0.69), and a medium effect size for hyperactivity (0.4). Fifteen studies were included in the evaluation, and these comprised some 476 participants in the controlled studies and some 718 in the pre/post evaluation (Kaiser, Othmer 2000).

Ironically, the Monastra study was excluded from the above compilation because the effect size was so large (2.22) that it failed the test for homogeneity of the sample of studies. Of course it did so for the best of reasons. The participants had been picked on the basis of an EEG criterion, the theta-beta ratio, that no doubt selected for those most likely to do well with a protocol tailored to that criterion.

By 2009, then, meta-analyses had confirmed the efficacy of SMR/beta neurofeedback in application to epilepsy and ADHD, respectively. Both reviews had also included studies employing slow-cortical potential training.

Neurofeedback in application to Addictions

A third major clinical interest emerged in the late eighties around neurofeedback in application to alcohol dependency. This topic also takes us back to the very beginnings of the field. The original discovery of EEG biofeedback had in fact been made by Joe Kamiya in the course of his research on the alpha rhythm of the EEG. Kamiya had originally intended to investigate the relationship of the EEG to psychophysiological states, and in that quest was testing whether an individual was able to discriminate the presence of an alpha burst in his occiput at a given moment. Fortuitously, he had in his chair someone who was able to learn that task perfectly in just four sessions, over which Kamiya gave him simple verbal feedback on whether or not he was correct whenever he was prompted by an occasional tone. By the fourth session, the trainee had an unbroken run of 400 successful trials. Kamiya never again had such a good subject, but the field was launched. This work took place in 1958 at the University of Chicago.

Within a few years operant conditioning of the alpha rhythm was undertaken in Kamiya's laboratory (Kamiya and Noles, 1970). Application to anxiety reduction followed (Hardt and Kamiya, 1978). Just as happened with the work of Sterman with epilepsy, as soon as therapeutic benefit of the training experience was insinuated, the whole procedure was subjected to heightened scrutiny. It did not help that the alpha training had been brought to broad public awareness through an interview with Kamiya that was published in Psychology Today in 1968. This resonated with the Zeitgeist of the psychedelic age, and alpha training became popular in certain circles as a way of inducing an LSD-like experience without the attendant risks. In the same timeframe, Barbara Brown worked diligently to bring alpha training to broader awareness within the culture through her books and lectures (Brown, 1974, 1977).

Several replications by teams of academics failed to support Kamiya's findings, and suddenly the nascent field of biofeedback, which had organized itself formally in 1989 around this new method of promoting self-regulation, found itself on the defensive. In particular, one controlled study used alpha-down-training as a control condition, and found that both challenges resulted in improved anxiety control (Plotkin & Rice, 1981). As the original hypothesis was not supported, alpha training was deemed a nullity. This is understandable in terms of the theories of the day. A more modern perspective would hold that both kinds of training can lead to learned self-regulation. Indeed, bi-directional training has become commonplace in biofeedback.

A second blow was struck with the report that subjects sitting in subdued lighting under eyes open condition were unable to increase their alpha amplitude with training above their own eyes-closed baseline (Lynch, Paskewitz and Orne, 1974). Since this served as a replication of an earlier such finding, the issue was considered settled, and this caused academics to abandon the research into alpha training. It was soon pointed out that the negative outcome studies had been riddled with methodological flaws. For example, Lynch et al. based their negative findings on the basis of a single training session. Further, most used a percent time feedback criterion rather than integrated amplitude. But by the time of the sober reappraisal by Kamiya, it was too late (Ancoli & Kamiya, 1978).

The biofeedback community reconstituted itself around the use of peripheral physiology to effect improved self-regulation. Fortunately the EEG work continued among a variety of independent groups that included in particular one at the Menninger Foundation that was organized originally by Elmer Green at the invitation of Karl Menninger himself. The interests in this group lay with the study of dimensions of the human experience, not with the amelioration of mental disorders. However, their work did inspire such pursuits by others. One early study yielded promising outcomes in application to alcoholism, and that kindled a new focus for the field (Passini, Watson, and Dehnel et al, 1977).

This initial study of the application of alpha training to addictions, among other influences, led Eugene Peniston to apply the method to his alcoholic veterans at the Fort Lyon Veterans Administration facility in Colorado (Peniston & Kulkosky, 1989). His first study involved ten Vietnam era veterans who had had a minimum of four prior treatment failures. The controls received only the regular VA treatment. The experimental participants received some initial exposure to temperature training, which was then followed up with thirty sessions of what was now called Alpha-Theta training. This training

utilizes reinforcements in both the alpha and theta bands. The results were stunning. Initially eight of the experimentals sustained abstinence after release from the program. The remaining two, having contemptuously dismissed the EEG training as a meaningless exercise, soon found out that they had lost their tolerance for alcohol, and perforce became abstinent as well. All the controls stayed true to their prior pattern, and all were re-admitted to treatment within eighteen months. The experimentals were followed up for more than eight years. All retained their sobriety.

This intriguing research was not welcomed by the biofeedback community when it was presented in 1990 because it threatened to rekindle the controversy about alpha training. Nevertheless, Peniston followed with several successful replications, and other groups did as well. He summarized his research in 1995 (Peniston &Kulkosky, 1995), and described his method in a book chapter (Peniston and Kulkosky, 1999). One major criticism was the limited size of the studies, even though they were clearly sufficient in light of the strong results.

A large-scale study was undertaken in 1994 at an addictions treatment center in Los Angeles. 121 participants were entered into a controlled study in which the control was the regular Minnesota Model in-patient treatment program (Scott, Kaiser, and Othmer et al, 2005). An SMR/beta protocol was inserted in place of the temperature training, but the emphasis again remained on the Alpha/Theta component. One year post-treatment it was established that the experimentals were sustaining sobriety at a rate three times that of the controls, at nominally 75%. After three years, the control group had mostly relapsed, while the experimentals had largely maintained sobriety---albeit with maintenance of group participation that was a key part of the 12-step-based program. Ongoing sobriety was highly correlated with continued participation in group, which in turn was highly correlated with the prior EEG training experience.

What came to be known as the Peniston Protocol was then also evaluated in a multi-faceted program in Houston to rehabilitate homeless crack users. This program was large-scale, and it was extraordinarily effective, with success in placing participants back into housing and into either an educational setting or a job in excess of 80%. While it is not possible to parse just how much the neurofeedback contributed to this outcome, those involved clearly saw it as the heart of the therapeutic dimension of this comprehensive program (Burkett, Cummins, and Dickson et al, 2005).

Slow Cortical Potential (SCP) Training

Whereas all of the above developments took place in the United States, a very different method of neurofeedback was being developed by a research group under Niels Birbaumer in Tuebingen, Germany. The discovery in 1964 of the Contingent Negative Variation (CNV), a transient negative excursion of the surface potential in preparation for a response, soon led to further observation that the CNV could be subjected to voluntary control. That in turn led to successful control of the baseline SCP through operant conditioning techniques (Lutzenberger, Elbert, and Rockstroh et al, 1979). As the SCP appeared to reflect cortical excitability directly, the method was applied to the management of medically uncontrolled seizures, migraine, and even schizophrenia. The emphasis in research, however, has been on ADHD, as in the case of EEG band training (Strehl, Leins, and Goth et al, 2006).

The clinical claims were not welcomed in Europe and more than they had been in the United States, so research focused increasingly on the use of this method in order to give locked-in patients the capability to communicate. Voluntary control of excursions in the SCP could be used in a selection scheme for letters of the alphabet. At a minimum, the success in this application proved the method to be quite capable of yielding real-time control of the SCP. Given the cognitive demand, the training could not be done with very young children, but in other respects the method was straight-forward. A single placement at Cz was universally used, and there aren't many nuances to the protocol. Bi-directional training of the SCP has been adopted in order to train for better regulation in general. At each trial during the session, the trainee is given the direction in which the SCP is to be moved.

New Departures in Neurofeedback

The Beginnings of QEEG-informed training

Up to the early nineties all of the principal neurofeedback protocols were grounded in physiological models, and hence could be referred to as mechanisms-based. SMR up-training was targeting the down-regulation of motoric excitability. The accompanying theta-band down-training was targeting the improvement of thalamocortical regulatory control. Alpha-band training moved trainees to lower arousal levels and to residence in calmer states. SCP training reduced cortical excitability directly.

However, the beneficial consequences could be observed over a variety of clinical conditions, ranging from migraines to insomnia to pain syndromes, that had not been specifically targeted. Cognitive function was often enhanced as well. The generality of effects could be best seen in application to minor traumatic brain injury (mTBI), where benefits could be observed for the entire range of symptoms characteristic of such injury. At the same time, there was no reason to assume that a physically-injured brain should necessarily respond in the same way as other brains.

In the early nineties it became practical and affordable to acquire a 19-channel digital EEG capability even in a private clinic, and to subject it to quantitative analysis for comparison against norms. This made it possible to tailor the training to the specific conditions prevailing in a particular brain. That in turn would for the first time yield the kind of protocol specificity that researchers would be looking for to validate the technique. And finally, this new capability would give neurofeedback the same 'deficit-focus' that characterizes psychiatry and psychology. This need was keenly felt, because neurofeedback still lacked general acceptance within the health professions.

One consequence of this orientation to the quantitative EEG is that it placed clinical considerations into the background. Deviation from norms provided the rationale for the protocol rather than any specific symptom or diagnosis. In practice, the observed deviations were often arbitrarily attributed to whatever diagnosis the person came in with, in order to make the case for neurofeedback to the client. Effectively the target was the deviation from norms. Training could now be done at any site and at any EEG frequency, and this resulted in a ramp-up of the collective body of experience and of the learning curve within the practitioner community. Aiding this endeavor further was the fact that this exploration was taking place in hundreds of individual clinics, without any central direction. The

downside was that this collective effort would not yield the kind of research that would be persuasive to academia. Hence neurofeedback would retain its outsider status even in the face of this aspiration to a scientifically grounded procedure.

There was yet another problem. Neither the field of neurology nor that of psychiatry had yet adopted digital EEG analysis to guide therapies, so the neurofeedback field had just multiplied its challenges of persuading the mainstream rather than reduced them. It did not help that individual neurofeedback clinicians were typically not credentialed in EEG diagnostics. Hence there was the messenger problem. Further, in its early days the whole field of digital EEG analysis was riven with controversies because many issues had simply not yet been resolved. Expert analysis of clinical cases rarely corresponded between experts. Then there was also a fundamental problem lying at the root of the whole enterprise, namely that EEG deviations sometimes reflect accommodations rather than deficits, which complicates targeting. And finally, EEG deviations were often so numerous that clinical judgment is required to establish the appropriate hierarchy of targets. Meanwhile, the promise of professional guidance toward reliable prescriptions for training protocols served to attract weak players into the field.

It also transpired that in the new regime, in which normalization of EEG parameters became the objective, the up-training of presumptive deficits in the EEG band amplitudes was found to be much more problematic than the inhibition of excesses. The latter either worked or it didn't, but it rarely caused a problem. The promotion of higher EEG amplitudes, on the other hand, often led trainees into further distress. In consequence, quantitative EEG-based (QEEG-based) training came to be focused largely on inhibiting excesses. For the reward-based training, clinicians typically defaulted back to the standard protocols that had carried the field to its initial success. In this manner, the information yielded up from the full-head EEG was more clearly additive to what could be done with only single-channel derivation.

How is this differential effectiveness of reward- and inhibit-based training to be understood? The inhibit-based training is typically accomplished with threshold-based withholding of the rewards. Nothing is actually being inhibited. This kind of cueing elicits a rather non-specific response on the part of the brain. The reward-based training, on the other hand, is much more specific in its appeal to the brain, and the specifics matter. In our own implementation of the SMR/beta protocols, for example, we consistently observed a preference for higher frequency training on the left hemisphere than the right. This finding came about in an interesting way. We had started out with left-hemisphere training in the beta1 band, 15-18Hz, following Margaret Ayers, who had been a graduate student of Barry Sterman.

Ayers had found the higher band to be more consistently helpful for her head-injury and stroke patients (Ayers, 1999). We then added SMR-band training at Cz, following Tansey and Lubar, for the ADHD children. A move from Cz to C4 then led to much more hemisphere-specific effects. Thus our standard protocol became a combination of "C3beta and C4SMR," with the two protocols titrated as needed. This approach became broadly popular within the field, and was adopted by several thousand practitioners over the years. This was the protocol employed in our large practitioner survey on ADHD (Kaiser & Othmer, 2000).

The Evolution of Mechanisms-Based Training

By the mid-nineties, even our standard mechanisms-informed, protocol-based training was subjected to evolution. It was observed that trainees who happened to be particularly sensitive to the training responded in a highly frequency-specific manner. The training needed to be individualized with respect to the rewards as well as the inhibits. First the intermediate frequency of 15 Hz was provided. Then adjustment in 0.5 Hz steps was provided for. Eventually, even finer frequency divisions turned out to be advantageous. Such a high frequency-specificity has never been adequately explained.

This surprising finding led to our adoption of an optimization strategy for each client, one in which the best reward frequency needed to be determined through sequential A/B comparisons. The optimization had to be accomplished by tracking the sometimes subtle responses of the client through the sessions and from session to session. It could not be done by merely inspecting the EEG. This finding sheds light on the difficulties that had been encountered earlier with the QEEG-based training, because the requisite discrimination of the optimum target frequency is just not possible on the basis of the EEG.

The discovery of the frequency-specificity of reward-based training gave permission for the migration of the reward frequency beyond the standard bands in pursuit of the optimum training frequency. In fact, for many individuals it became mandatory. This, together with the liberation from the standard training sites that had already been accomplished with the adoption of digital EEG analysis, led to yet another period of rapid clinical progress. Looking back on this period, however, it is apparent that every step into the unknown was undertaken cautiously. Every incremental step forward was thoroughly consolidated in empirical support before additional steps were taken.

With the availability of 19-channel data for the display of spatial maps, attention shifted to site-specific data, and away from the bipolar derivation that had been customary in clinical EEGs. And in the conceptual frame of targeting EEG anomalies it had also become obligatory to undertake single-site training. This is referred to as referential placement. That means only a single active electrode is placed over cortex, with the other active electrode, referred to as the reference, placed on a quasi-neutral site such as an ear lobe. This meant the abandonment of the bipolar montage that had been standard for Sterman and Lubar---even for the standard Sterman and Lubar protocols. We had found the Sterman and Lubar protocols to be quite adequate for our purposes. With the impetus to move beyond the sensorimotor strip we later returned to bipolar montage so that we could at once explore new sites while keeping one foot planted, so to speak, on the familiar turf of the sensorimotor strip. As Sterman felt the need to point out, the sensorimotor rhythm is only observable on the sensorimotor strip. By moving the other electrode off the strip, we were training the relationship between the two sites. Training effect was enhanced nicely by virtue of the incorporation of frontal and pre-frontal sites, but possibly also by virtue of the return to bipolar montage.

The increase in sensitivity of the training that was purchased with bipolar montage brought about a heightened awareness of the frequency specificity. This was particularly an issue with those who responded very sensitively to the training, such as migraineurs and fibromyalgia patients. It was the challenge of sensitive responders that led us progressively to new placements and new regions of the

EEG spectrum. Over the course of some years, the entire conventional EEG spectrum, from 0.5 Hz to 40 Hz, was eventually encompassed. During this initial exploration, we found the distribution of optimum target frequencies to cover the entire spectrum.

The Migration to Lower Frequencies

The greatest challenge we confronted in that time frame (1999 - 2004) was the progression to ever lower frequencies. We proceeded into this terrain with some trepidation because of the known hazards of training at low EEG frequencies that Lubar and Sterman had already exposed. The difference was that we were now armed with the knowledge that training had to take place under very specific conditions. We therefore actually had complete consistency between the new and the old findings. The problem that had been identified by Lubar was that up-training toward greater EEG synchrony was potentially a risk, particularly in conditions such as epilepsy where excess synchrony is a known hazard. Under specific conditions, however, that could now be managed. The use of bipolar montage biases the training toward desynchronization of the target frequency between the two active sites (Putman and Othmer, 2006). Lubar had used bipolar montage as well, but that was not enough to render the training benign in his early study on seizure disorder. With careful adjustment of the training frequency, however, the effects were not only favorable but rather more powerful than we had been accustomed to. This led to breakthroughs with clinical conditions that had not yielded to the earlier higher-frequency training. These positive developments encouraged further exploration, and that led eventually to the exploration of the infra-low frequency regime, the clinical approach that is featured in many of the chapters of this book.

Status of the Principal Approaches to Neurofeedback

Before that topic is taken up, however, a perspective on the context in which this development took place is in order. Progress was being made in all of the principal ways of doing neurofeedback that have been described. QEEG-based training evolved in the direction of targeting the coherence between two sites rather than amplitudes at a single site. This made for more dynamic, more impactful training, just as we had found when moving from referential back to bipolar training in our own approach. Representative studies that reflect the state of the art of this approach exist for migraine (Walker, 2011), TBI (Thornton & Carmody, 2008), and schizophrenia (Surmeli, Ertem, and Eralp, 2012).

Site-specific targeting also evolved further. This approach could be aided with the use of LORETA, a program that constructs a source distribution of virtual dipoles that is consistent with the prevailing EEG at the nineteen sites. With the application of physically realistic constraints, the infinity of such solutions that exists in principle can be reduced to just one. This procedure can be used to refine targeting and to focus on sources at depth within the cerebrum. It can also be useful in pre-post evaluation of training procedures.

Inhibit-based training also matured as a stand-alone approach in two principal manifestations. In the first, the brain is regarded as a non-linear dynamical system, and the EEG is seen to reflect those properties. This means that the EEG cannot be properly analyzed in terms of Gaussian distributions. On the contrary, EEG parameters are known to exhibit scale-free statistics, or very broad distributions that

may be well approximated by power laws. In the Gaussian perspective that we continue to press into service, it would be said that there are long tails. In essence, stationarity of the EEG cannot be assumed. What is measured at one moment is not necessarily replicable at another moment. Hence fixed thresholds are contra-indicated. Excursions into disregulation are detected dynamically at two bilateral sites (C3 and C4) on the sensorimotor strip and the brain is cued with respect to these excursions. All the drama that attends reward-based training because of the decision-making involved is avoided. Since no clean boundary between function and dysfunction is discernible in the EEG, the tactical choice is made to inhibit only the extrema in the tracked variables. This increases the likelihood that the fouls being called on the brain are indeed episodes of disregulation. A study was recently published in which this method was used in the recovery from “chemo-fog” (Alvarez, 2013).

The second approach seeks to enhance the reliability in the discrimination of disregulation by force of large numbers. By tracking a number of EEG parameters (band amplitudes and site-to-site coherences) for several sites over time, advantage is taken of the fact that correlation between the measures increases as the brain enters a state of disregulation. Superposition of all the measures then yields a collective index to the instantaneous state of disregulation of the whole system. The reliability of this index increases further as the brain reaches extremes of disregulation. In practice, the trainee is simply exposed to the time course of this index, which serves as an incremental guide to more regulated states, and ultimately to clinical success. Since this method is typically based on deviations from normative behavior, it is referred to as Z-score training.

The bulk of the work in the clinical realm is still being done with variations on the standard SMR/beta protocols, combined with one inhibit strategy or another. Placement is either on the midline at Cz, straddling Cz on the midline in bipolar montage, or using the traditional lateralized placements such as “C3beta/C4SMR”, which likely remains the most common choice.

Alpha-theta training has also been adopted quite broadly as a complement to the SMR/beta training. The thrust here is very different. Whereas the intellectual frontier in this field has all along been the challenge of functional normalization, there has remained a persistent need to redress the psychological residue of prior traumatic experiences or of a traumatic early childhood. This can be accomplished with Alpha-Theta training, and more effectively after a measure of physiological stabilization has been achieved. The Alpha-Theta training quietens the outer-directed faculties that keep one rooted in the present moment, and calms the fear-driven self. Hence it allows the journey to the interior realm, where historical traumas can be resolved while the person is resident in a perceived calm and safe place. The intent in this training is entirely experiential.

For many who encounter this training, the effect is transformational. In particular, it is those who come with a trauma history who find this experience healing. And that can go a long way toward explaining the early reports of dramatic spiritual experiences with alpha training that were so off-putting to the academic community originally. Having fled their families, those youngsters who were drawn to Haight-Ashbury and the drug experience were likely also those who would respond powerfully to Alpha-Theta training. By the same token, those same experiences would not easily be replicated with research

subjects plucked from among engineering students. In retrospect, it is apparent that all the early 'magical' claims for alpha training have since been validated in clinical experience.

Alpha training has also played a large role in training toward functional normalization and the enhancement of our attentional capacities. This is the work of Lester Fehmi, which also had its origins in the early days of discovery of alpha training. Fehmi relies heavily on the language of attention, thus recruiting the individual actively into the task of self-regulation practice. The objective is to move from narrow and objective focus to a broader, more inclusive focus, and from a separate to an immersive presence. This shift is accompanied by a lowering in arousal level, albeit with maintenance of alertness. Reinforcement on alpha-band activity supports this shift, and it does so even more strongly when the training promotes whole-brain synchrony (read coherence) of the alpha signal explicitly (Fehmi and Robbins, 2007).

Jim Hardt has also relied on the promotion of alpha synchrony for most of his work. His engagement with this field extends all the way back to his days as a graduate student of Joe Kamiya, given impetus by his own impactful first alpha training experience. And he had been an engineer. Hardt offers intensive programs in alpha training that also incorporate group therapy to support personal transformation. More advanced training is offered with emphasis on the theta band (Hardt, 2007).

Yet another approach that had an early start is one that emphasizes 40-Hz training. This training was first investigated by Bird, targeting cortical function (Bird, Newton, and Sheer et al, 1978). The 40-Hz region is one where EEG synchrony is commonly observed in the engaged brain, meaning that bursts of such narrow-band activity are readily discernible above background. As with other synchrony training, the training is experienced as both calming and alerting. But the subjective response is typically distinct for each of these frequencies, in that different network configurations are being appealed to in each case.

Toward a New Departure in Feedback

The range of clinical conditions that are presently addressed with one or more of these methods now extends to nearly the entire spectrum of psychiatric conditions, plus many neurological conditions. The best evidence indicates that neurofeedback can be far more effective than pharmacotherapy alone, or psychotherapy alone. The best evidence in this case is to be found in the clinical realm, where clients typically come to neurofeedback late in their clinical trajectory, and are already getting treatment to current standards.

The implications are clear. Psychiatric and neurological conditions involve learned brain behavior that cannot readily be undone by means of pharmacotherapy or talk therapy alone. And yet this learned behavior is accessible to us through methods of re-learning. It is appropriate, therefore, to think of neurofeedback as a modality of rehabilitation, by analogy to physical rehabilitation. And just as there is a complete continuum between physical therapy and training for optimum functioning in the athletic realm, there is complete continuity between feedback-based rehabilitation and optimal mental capability---not only in the cognitive realm but the affective realm as well.

This core reality has been somewhat obscured by the fact that neurofeedback has had to find its space within the framework of our disease- and disorder-focused health care model. Within that model, problems need to rise to a given level of severity before they get any attention. In line with that model, neurofeedback strategies have been favored that maximally emulate the prevailing focus on the discrete disorders. This becomes obvious in the focus on features of the EEG that manifest the disregulation status. The immediate target is not the disorder itself, but rather the landmarks of disorder, implicitly the disregulation status per se. Without question this approach has borne abundant fruit, and therefore its validity is not in question. But the approach remains incomplete. Alas, some of the most disturbing and intractable mental disorders cannot be discerned in the EEG at all by current methods. The personality disorders are a case in point. These are also the conditions that first take root in the course of early development. Disregulation status is not reducible to what can be readily detected in the EEG. A different approach is needed to complement what we already have.

Throughout our quarter century of work with neurofeedback, we had always been stymied by the most intractable end of the distribution of severity, almost irrespective of the particular diagnosis involved. This might involve migraines, fibromyalgia, depression, anxiety, Tourette Syndrome, Obsessive-Compulsive Disorder (OCD), or substance dependency. And then there were the conditions that had remained relatively intractable to remediation, such as chronic pain syndromes, the developmental disorders of childhood, borderlines, and Dissociative Identity Disorder, and addictions.

Since 2006 inroads have been made with a new clinical approach that allows us to address even the most challenging cases that are seen in psychiatry outside of institutional settings. This method is a radical departure from all existing approaches. It cannot be understood in terms of the existing models. It is not deficit-focused; it is not prescriptive. It is not a close-order drill to micro-manage the brain and teach it how to behave. This method allows the brain to acquire new capacities for self-regulation in the same way it learns other skills. Even in the face of all that has already been accomplished with various methods of neurofeedback, this new approach is deserving of special treatment, and for that reason this book is largely devoted to this one approach. We now turn to the further elaboration of the development of this method.

The Development of Infra-low frequency training

By 2004 we had explored the EEG spectrum across the entire ‘conventional’ range, from 0.5 Hz to 40 Hz, and found individuals who optimized in all regions of this spectrum. We were constrained, however, by our software, which limited us to a 3-Hz bandwidth. This meant that the lowest center frequency we could dial in was 1.5 Hz. Over time, client data piled up to the point where it was obvious that the lowest frequency was preferred by more clients than any other. This motivated the investigation of yet lower frequencies. This transpired in 2006.

We found software that allowed us to extend the range to 0.1 Hz, expecting of course that those who had optimized at 1.5 Hz would now distribute themselves over this wider range. Instead we observed that the new distribution was even more strongly skewed toward the new lowest value than had been the case before. In fact, some two-thirds of all of our clients exhibited a preference for the

lowest frequency of 0.1 Hz. This frequency was too low for our conventional methods of signal-handling, and accommodations had to be made.

In the conventional frequency range, the narrow-band signal was rectified and smoothed in order to yield the magnitude of the EEG in the particular frequency band. At 0.1 Hz, that process is too slow for good feedback, so instead we simply had the trainee watch the EEG signal go up and down with its periodicity of ten seconds. This actually worked quite well. That was not entirely unexpected, however, because we had already been feeding the continuous band magnitude back to the client over all these years to accompany the discrete rewards. The intent all along had been to promote engagement of the client with the process. But the brain was clearly also deriving information from the ongoing signal stream, so it was more engaged with the process for that reason as well. The continuous signal was information-rich by comparison to the discrete rewards, and as such was responsible for the exquisite frequency sensitivity of the training that we had observed. The same thing was now happening with the actual signal. The brain got all the information it needed for the low frequency training from the time course of the continuous signal. This struck us as remarkable on first encounter, but at another level it was also not unexpected. After all, we had undertaken this initiative in the expectation of success.

In the low-frequency region, the simple expedient of tracking the actual signal instead of the amplitude envelope meant that the discrete rewards no longer made sense. Thresholds had lost their meaning in the new context. We would have threshold crossings once every cycle, and they would not convey significance. With the abandonment of discrete rewards, it also became clear that we had entirely cut our moorings to the operant conditioning model that had been a central pillar of the entire development of neurofeedback. The operant conditioning model had been our lodestone. Indeed, Sterman's cat data stands as an elegant exemplar of that kind of learning. Now another explanation was clearly needed. We will return to this conundrum later.

In the clinic the path was clear. With clients piling up at the lowest frequency, the range obviously needed to be extended further. It was extended another order of magnitude, to 0.01 Hz, early in 2008. We were now dealing with a rather slowly changing signal, and yet the brain seemed to handle it much as it had before. And over time the same pattern we had observed before was once again repeated. About two-thirds of the clients optimized at the lowest frequency. The range was hastily extended yet another order of magnitude to 1 milliHertz, or 1mHz, later in 2008. Yet the same pattern emerged over time as we became acquainted with this new regime: cases piled up at the lowest frequency available. Finally, as the range was extended in 2010 to 0.1mHz, a similar pattern once again developed. Some two-thirds of clients eventually ended up preferring the lowest target frequency.

0.1mHz implies a period of 10,000 seconds, or a period of 2.8 hours. We did not have to be told that training a frequency this low is an absurdity on its face. And yet the brain was responding as promptly as ever. In fact, the overall training process was more demanding than it had been before, quite simply because it was stronger in its impact. It needed to be done right, which meant in particular that the choice of target frequency was critical. But just how did the frequency enter the picture? In the conventional view, an outside observer would have to track the signal for a good part of a whole cycle in

order to know the precise frequency being represented. And yet the brain was responding quickly, and in a way that was very frequency-specific. And on top of everything else, the signal did not even appear very sinusoidal, which meant that the signal unfolding on the screen was not necessarily the target frequency, i.e. the center frequency of the filter. The laws of nature—or at least the principles of signal processing---appeared to be violated.

The Search for a Mechanism

At this point I would hate to deprive a certain kind of reader of the challenge of figuring this out, so for these people it is recommended that they stop reading at this point and see if they can puzzle this out on their own. For everyone else, and for those who gave up trying to figure this out, what follows is the explanation for what is happening in this process. It is best to start with an anecdote that makes matters concrete: At a professional training course an attendee has just been wired up to experience the training for the first time. After just a few minutes of training she finds herself feeling distinctly different than before, but has no idea why. She is urged to report how she feels, because this is the beginning of the process of hunting for the optimum response frequency. Everything was happening as expected, except of course for the specifics that related to this person. But a psychologist in the audience who was thoroughly steeped in the operant conditioning model leapt out of his chair and blurted out with finality: “This is not operant conditioning.”

Indeed it is not, but we do have something to explain here. The brain had gone into action on the basis of the information that was being provided, and we had nothing to do with the specifics of that. To understand this process, matters have to be regarded from the perspective of the engaged brain, not that of an outside observer. The low-frequency filter shapes the information going back to the brain about the EEG so that it contains only low-frequency information. This would appear to preclude a rapid response on the part of the client. But even though the frequency response is slow, the transient response is not. The transient response is merely attenuated by the filter, but it is still present. Most significantly, it is still present in real time. Even slow brain rhythms must respond to environmental demands, both internal and external, and they must do so promptly. The signal processing does not obliterate that information. It does not delay the signal; It merely attenuates it.

The brain is able to detect the subtle changes in the signal because it is expecting them, and therefore is looking for them. As soon as the brain recognizes that the signal on the screen is somehow reflecting an aspect of its own activity, it ceases to be the naïve observer of an innocuous signal. The brain then relates to the signal as its own output. The particulars of the relationship remain obscure, at least at the outset. The brain goes into a hypothesis-testing mode. It assumes command of the situation, if you will, and attempts to anticipate, control, and manipulate the signal. This is just how the brain deals with information about its more direct interaction with the world through movement. Movement that the brain cares about is always the execution of an intention, and then the brain renders an ongoing judgment about whether its intentions are being successfully executed. Finally, it takes corrective action such as fine adjustment of the activity. All the elements are there: prediction, comparison to template, and correction. The ‘movement’ of the EEG signal is treated analogously to the manner in which the brain treats actual movement of the body.

A fruitful way to regard brain function is to see it as organized for the regulation of movement. Clearly that has become a highly refined skill on the part of our brain. All that is required to understand infra-low frequency neurofeedback is to realize that this entire repertoire of refined regulatory control can now also be applied to the brain's internal activity, as reflected in the EEG. And that capability extends to every function that is subject to regulatory control by the brain and can be made 'visible' to the brain through its EEG. The brain relates to the outside world as agent, and the feedback allows the brain to encounter its own EEG also as agent.

The EEG is a correlate of brain activity, and so is the dance reflected in the mirror in the dance studio. The brain is directly in charge only of its own neuronal activity, but it adjusts its responses on the basis of the correlates that implement or reflect that intrinsic activity. Much of cortical activity with respect to its axonal communication is regulatory in nature. And that aspect is distilled for us in the EEG in large measure. Finally, the refinement of regulatory role that results from the brain attending to itself then plays through the entire hierarchy of regulation.

Infra-low frequency training has shown us that the brain can enhance its own self-regulatory capacities through conventional skill learning. It is not necessary to install an operant conditioning paradigm. This also means that volitional engagement is not required, nor does the process impose high cognitive demand. This makes the training available even for working with infants. Even the infant brain is doing its best to come to terms with its environment, and is quite capable of becoming entranced with its own EEG. And when it comes right down to it, if we have given the method an appropriate interpretation, should that same explanation not also serve to explain operant conditioning? Indeed it does.

With the point of departure that the brain is to be understood in first instance in its role as agent rather than in its role as observer, we can also view the operant conditioning paradigm in that perspective. Once the brain is exposed to a sequence of discrete rewards that begins to look like a pattern, it will simultaneously register those events that are correlated with it. These can be external events, or internal activity, or external events that reflect internal activity. The brain will then resort to its predictive algorithm to do hypothesis-testing on the various correlations. Eventually only a single correlation will survive the screening. But the hypothesis-testing continues. This explains both the extinction phenomenon and the observation that a program of partial reinforcement is most resistant to extinction. Under the latter, it is most difficult to be certain that circumstances have indeed changed.

Understanding Infra-Low Frequency Training

On the one hand we have the well-established operant conditioning model, and on the other we have what we call the prediction model, one in which the brain reacts to the ongoing signal on the basis of its own interpretation of the signal and the resulting projecting of the signal into the future. If an equivalence between the two methods can be drawn, what then is the advantage conferred by the infra-low frequency training? First of all, there is the matter of information density. In the ILF training, the signal is continuous rather than discrete. There is more information per unit time, first of all, and

secondly, the continuous signal offers greater traceability to the underlying activity that the brain is trying to regulate. There is more subtlety and precision, which leads to finer control.

But it has already been said that we have had the continuous signal as part of the feedback all along. So there must be yet another advantage to training in the infra-low frequency regime to compensate for the fact that there is less 'information' forthcoming per unit time than at higher frequency. That may have to do with the fact that in this region the signal reflects more purely what the brain is trying to manage---cortical activation. At higher frequencies, the EEG signal is much more complex, and reflects many influences, only one of which is local cortical activation.

Once the advantages of training at infra-low frequencies are apparent, it is also clear that at these frequencies there's very little choice about how training must take place. We know of no viable alternative to the "waveform-tracking" approach to training. But even if that is accepted, there remains the question as to why there is such a strong preference for the extreme low frequency training. What advantage do we derive from training at such a specific frequency? At this point, we are reduced to mere speculation. The most common target frequency of nominally 0.1mHz falls into the range of our Basic Rest-Activity Cycle of 90-120 minutes (the BRAC). This may be the lowest periodicity governing our cerebral activation that is dynamically managed. The circadian rhythm, by contrast, is under tighter control by a number of clock genes.

It seems only too likely that we are interacting with the mechanisms that govern this periodicity of tonic cortical activation. The periodicity of the BRAC suggests that the governing mechanisms are organized as a resonant system, and act to maintain the system in resonance status. If that is the case, then we would expect to encounter some of the properties of a resonant system (Othmer, 2008). One feature is that the behavior of the system is most strongly frequency-dependent within the vicinity of the resonance frequency. Another is that the system behaves most benignly at the resonance frequency. A third is that the behavior can be very different in the near neighborhood of the resonance frequency. All these observations hold true for this kind of training.

Exploiting Infra-Low Frequency Training

The direct engagement with the regulation of cortical activity ties us directly into arousal regulation governed by the brainstem. That in turn can be seen as the foundation of our entire regulatory hierarchy. By suitable site selection, the training can then be focused on higher rungs of the regulatory hierarchy, autonomic regulation and affect regulation being the first among them. Surprisingly, perhaps, a wide variety of rather intractable symptoms fall away with just one two key placements that address the bottom of the regulatory hierarchy.

The early work with SMR/beta protocols had already given evidence of system-wide impacts that appeared to have no particular connection to what was going on at the sensorimotor strip. The conclusion was already inescapable at the time that we are interacting with a highly integrated regulatory regime. With infra-low frequency training this system was being engaged at its most foundational level, and that yielded immediate clinical benefits. One is tempted to draw the conclusion that the principal problems in brain regulation lie at the foundations rather than—to pick an example---

in the specifics of higher cognitive function and the subtleties of attentional failure. By facing into the problem of learning disabilities early on we were tackling the issues in the wrong order. We were starting at the wrong end of the regulatory hierarchy.

Infra-low frequency training has now given us access to the infant brain in its age-appropriate stages of development. And by the same token it has given us access to all those disorders that could be traced to early childhood developmental lacunae or misdirections---traumas of neglect and abuse; physical injury to the brain; birth trauma; high fevers and brain infections; emotional crises in early childhood; etc. It has been clear for a long time that the most intractable disorders encountered in psychiatry are traceable to disruptions in the normal developmental trajectory for one reason or another, or because the psyche had simply been under prolonged siege during its formative stages. We finally have a way of reaching back, effectively, into those early developmental years and redressing the malformations of regulatory networks. The success of ILF training made it unambiguously apparent that even though the behavioral consequences appeared therapeutically intractable, the causal chain lay largely in the functional realm and was therefore accessible to us for remediation.

Indeed, we know that "structure follows function," but we can take that in both directions. We can understand the intractable disorders in terms of the equivalent dictum, "structure follows dysfunction," but with a persistent appeal to function the process can now be substantially walked back. Having finally 'discovered' brain plasticity in the mid-nineties, neuroscientists came to see it mainly in positive terms. However, one can also understand mental disorders in terms of brain plasticity mediating accommodations under duress. In the case of shock trauma, for example, such accommodations can even be appropriate in the immediate context, and yet be detrimental over the longer term. The term "plasticity diseases" has been invoked to classify this phenomenology.

In conventional therapies, the dysfunctional network status is the point of departure (irrespective of whether that is part of the operative model). If the particular therapy does not resolve the core disregulation, however, then the resolution can only be partial. Symptom relief may be obtained, but the core dysfunction remains, and may even be further consolidated. With infra-low frequency training, the appeal is made directly to the core issue of the regulation of cortical activation and of central and autonomic arousal. Once it is firmly established that this is possible, it follows also that it is obligatory on the therapeutic community. The ineluctable reality of the domain of mental disorders is that the most intractable among them are trauma-based. If the potential exists to remediate the consequences of physical and emotional trauma of early childhood, and even in early childhood, then that must become the therapeutic priority. We know by now that these calamities can be remediated in young children, and they can also be remediated later in the adult stage. It is for this reason that a relatively new therapeutic approach, arriving late upon the scene, deserves to be treated as its own entity in this book.

Over the last decade, new findings in the realm of brain functional imaging have provided new theoretical support for the above claims. This is the discovery of our "resting state networks." The term has its basis in the historical development of functional magnetic resonance imaging (fMRI). For years comparison was being made between activated states and the baseline state, when at some point it was

realized that the baseline state was itself active. In fact, its activity totally dominates what the brain does at any moment. Engagement with the outside world is never more than a perturbation on baseline activity.

It seems reasonable to propose, therefore, that our good function hinges largely upon the organization of our baseline state, which is called the Default Mode Network (Raichle and MacLeod, 2001; Raichle, 2010). A secondary concern is then the smooth integration of the task-negative network with the task-positive networks. This coordination is mediated by the salience network (Sridharan, Levitin, and Menon, 2008). In a ground-breaking paper, Menon has made the case that much of psychopathology is traceable to dysregulation in the coordination of the Default Mode and the Central Executive Networks, as mediated by the Salience Network (Menon, 2011).

As it happens, the empirically derived electrode placements of our key protocols match up with those key nodes of the Default Mode Network that are accessible to us at the cortical surface. It appears that our primary pathway of intervention is to restore the internal functional connectivity of the Default Mode Network. Subsidiary protocols then address the smooth integration with the Central Executive Network, the key task-positive control network, and the Salience Network that mediates between them Recently, evidence derived from fMRI measurements has been furnished to document the up-regulation of the salience network with neurofeedback (Ros, 2013).

With infra-low frequency training we restore the proper hierarchy to the therapeutic agenda. This hierarchy recapitulates the original developmental sequencing. This sets the stage for other therapies to follow, if necessary. Surprisingly, however, if the proper hierarchy is respected, a lot gets done with as few as four basic protocols. That statement is given substance in the remainder of this book.

Summary

By virtue of all the research and clinical work that has been done over the last forty years, the following propositions have been established beyond doubt among reasonable men: 1) The brain is responsive to information about its own EEG; 2) the brain is capable of utilizing this information to enhance self-regulatory control; and 3) the new capability is a learned response that is then reinforced through ongoing activities of living. The evidence alluded to in the remainder of this volume testifies to the utility of infra-low frequency training in addressing the physiological basis of mental disorders in considerable generality. In this chapter it is proposed that the benefits of ILF training can be explained in terms of altered functional connectivity of our intrinsic connectivity networks. The information made available to the brain on cortical activation at infra-low frequencies appears to be quite sufficient to mobilize this renormalization.

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