Milestones in Development of the Othmer Method

By Siegfried Othmer, Ph.D.

Abstract

This monograph traces the evolution of Infra-Low Frequency Neurofeedback from its roots in the original SMR-beta training of Sterman and Lubar. It does so with a human interest flavor that reflects the emotional intensity and passion attending these developments over the years. The chapter also covers the theoretical perspectives that shaped the development trajectory from the original prescriptive neurofeedback to endogenous neuromodulation. This complements the companion monograph by Sue Othmer that takes a more clinical perspective on these same developments.

Key Words: Infra-Low Frequency Neurofeedback, SMR-beta training, Endogenous Neuromodulation, Intrinsic Connectivity Networks, Neural dynamics

Early Foundations

The early days of the field of neurofeedback are rich in ironies, human foibles, and stumbles in the darkness of ignorance, and were accompanied throughout by the specter of a resolutely oppositional—or uninterested—academic mainstream. When Sue and I got involved in 1985, the biofeedback field had just banished Joe Kamiya's and Barbara Brown's alpha training as unworthy of further scientific attentions. All the talk about altered states and transformative experience was just bringing discredit to serious academic inquiry. Researchers were still in charge of deciding what was to be taken seriously, versus what was just froth.

Scientifically respectable neurofeedback was to be found in Sterman's technique of operant conditioning on EEG variables in the low to mid-beta range. Being scientists ourselves, we were only too happy to have found ourselves on the right side of that divide. It didn't take long, however, for boundaries to blur and rigid divides to crumble. We pick up the story at the beginning of this new era. A first irony is that the discovery of the clinical import of SMR conditioning was completely fortuitous, and in retrospect met all of the conditions for a blinded, controlled design, the very thing that has always been insisted upon to qualify medical interventions. The birth of clinical SMR training had been by Immaculate

Conception, so to speak, with neither the cats nor the researchers aware of what experiment they were part of.

One experiment, intended merely to demonstrate the possibility of training a cat EEG via an operant conditioning procedure, had inadvertently 'contaminated' a subsequent experiment, conducted months later, to evaluate the susceptibility to chemically induced seizures. Here we had an inherently balanced design; yet there was no way for the placebo effect to play a role; there cannot have been any experimenter bias; and the results were not subtle. In Skinner's black box, figuratively speaking, some agent was disposing between live cat and dead cat. Whereas there were no 'hidden variables' in Erwin Schrödinger's black box, there was one here, and it was EEG conditioning.

One has the impression that Sterman, the meticulous animal physiologist, was yanked out of his comfort zone with this discovery. His colleague in arms, Polish expatriate scientist Wanda Wyrwicka, told him firmly: "Barry, this is too important. You have to do this with people." And that likely ended up sidelining his comfortable career and sullying his sterling reputation among his colleagues. In later reflection on the whole course of events, Sterman said wistfully: "I did everything the world of science expected of me to prove this out, and in the end it did not make any difference." He said this to all of us who were busy making a difference. But we were not his academic colleagues. I believe that all we were in a position to do, as far as he was concerned, was to sully his academic reputation even further! Where was the science, he asked plaintively, with all of your proliferation of methods?

Another irony is that Sterman had just obliterated B.F. Skinner's basic hypothesis by means of Skinner's own experimental technique, operant conditioning. Skinner said as late 1974: "It is a mistake to look for the origins of behavior within the brain instead of the outside world." Already, Joe Kamiya had whittled away at this hypothesis with his original alpha-band research, but Sterman's work demolished it wholesale. By 1972, the first paper on seizure control in a human subject had already been published.

The Sterman-Ayers-Othmer Triad

Our introduction to neurofeedback was by way of Margaret Ayers, who came to realize the power of the method while she was a student at Sterman's laboratory at the Sepulveda Veteran's Center. The mentor/student relationship deteriorated, however, and Sterman dismissed Ayers from his laboratory. Surreptitiously, she acquired an instrument from the lab engineer, Sidney Ross, and set up a private practice in Beverly Hills, just down the hill from Sterman's residence. Ross had secretly submitted a patent application on Sterman's instrument, installing himself as the inventor, and indeed also as the discoverer of the method! The patent was granted. When Sterman later got wind of this, according to Margaret, he

agreed not to make an issue of the fraud on condition that he, Sterman, could control sales of the instrument. He did not want the instrument getting loose outside of academia. After all, that is what had sunk alpha training. Ross instead made the system available to Margaret on a lease, and so the fox was loose among the chickens after all. Sterman's worst fears were being realized, with his approach now in the hands of someone he deemed unsuitable.

Since Margaret Ayers died in 2008 at a relatively young age of 62, she can no longer tell her own story. She was the first person to have a comprehensive grasp of the clinical reach of Sterman's method, and she was alone in that position for many years. This breadth of understanding had come to her entirely through rich, cumulative clinical experience. It was trial and error, frankly, but that is what the moment called for. In her practice, she concentrated on closed-head injury and stroke recovery. There was no competition for such clients. She attracted them from around the world.

When our son Brian first started training on March 5, 1985 for his epilepsy, the rest of us were eager to experience the training also. My wife, Sue, had an unmistakable first-session "clear-windshield" effect. Nine-year-old son, Kurt, trained for a while to moderate his impulsivity. And I did four sessions, which resolved a number of my issues. They did even more, however, in terms of giving me an understanding of the process.

Ayers was working with the instrument that had been designed for Sterman's early research. There was a beep-tone and brief yellow light for the discrete rewards, and there was a little flickering green light that reflected the fluctuations in training band amplitude. A red light indicated that inhibit thresholds were exceeded. As it happens, my EEG amplitude in the low-beta band was among the lowest ever found in captivity. As such, it largely failed to trigger the threshold for the discrete rewards even on the most sensitive setting on the instrument. This meant that the bulk of the information my brain was receiving consisted of the fluctuations in low-beta amplitudes, punctuated only occasionally by the beep tone. It seemed clear to me that learning must be occurring largely on the basis of the fluctuating signal, i.e., the flickering green light.-There just weren't enough beeps to matter very much one way or the other. The implications of this early observation would take some time to be fully appreciated.

There was yet another complication that I did not sort out until later. Given the objective of producing beeps, the nervous system does whatever lies within its scope to make that happen. With such a low-amplitude EEG, it was easy to override the EEG amplitudes with a little muscle tension (EMG). Quite unbeknownst to me at the conscious level, I was turning my head slightly in order to produce sufficient muscle tension in my neck to boost the incidence of beeps. The resulting EMG activity is episodic, so it did not totally corrupt the training signal. But the actual discrete rewards now tended to be false rewards, so the formal training objective was being undermined. That recognition further strengthened the case for

believing the fluctuations in the ambient signal to be the significant feature that engages the brain.

When it came to our project of developing a new instrument for Margaret Ayers, effectively a computerization of Sterman's analog design, an inhibit for muscle activity was thoughtfully included. After months of conversation between the two of us, an initially reluctant Margaret Ayers agreed to form a three-way partnership with Edward Dillingham, a software engineer, and me in order to develop a new instrument for her entirely on spec. If she liked the instrument, then she would not only use it herself but teach to it and spread the knowledge. Ed's development took three years, for which he received no compensation. Ed was a family friend who had witnessed the changes in our son Brian, which was sufficient all by itself to persuade him of the significance of this work.

Ayers appeared thrilled with the new instrument, but she was reluctant to turn to teaching. She told us that she needed to be recognized for her knowledge before she was willing to spread it around. That of course had it entirely backwards, but she would not be persuaded. Ed Dillingham was devastated; he was counting on this instrument for his financial future. Ayers walked out of the partnership with the new instrument, and we had no Plan B. Surveying the wreckage, Ed and I turned to Sue, asking her to pick up the ball. After all, her own academic background as a neurobiology graduate student both at Cornell and at the Brain Research Institute at UCLA suited her eminently for this work. Her Ph.D. dissertation had close affinity to both Barry's cat research and that of Peter Rosenfeld—the other academic heavyweight in the biofeedback field who had an interest in EEG. It involved classical conditioning of attentional behavior in cats, as monitored by evoked potentials, an emerging field in 1968. And it had been roughly contemporaneous with Barry's critical early research.

Sue finally agreed, but she did so somewhat reluctantly. It meant a total change in the life she had carved out for herself, and a shedding of the responsibilities that she had assumed. It had become clear to us that Margaret Ayers considered Sue a potential threat to her unique status in this emerging field, but it was equally clear to us that Sue had not given a single thought to "fishing in Margaret's waters." Quite inadvertently, it seems, Margaret had created the very thing that she most feared—an competing voice that knew what she knew and also had a grounding in neuroscience—yet another irony.

Ayers then launched both a State and Federal (patent) law suit in order to put the clamps on us legally. She had taken Ed Dillingham's instructional materials on the new system and submitted them verbatim to the patent office, claiming to be the inventor of the system. They reside there to this day, in as rough and incomplete form as they had been delivered to her. The Ross patent was never mentioned in the course of presenting the prior art. It was a case of fraud piled upon fraud. Scandal like this never makes it into the journals….

So to add to the pile of ironies, we now had Barry trying to shut Margaret down; we had Margaret trying to put us out of business with her patent; meanwhile, our new instrument was upstaging Barry's analog design. It was a case of 'stone, paper, scissors.' Legal affairs were not settled until 1991 in arbitration. The fake patent was declared invalid. Nevertheless, Ayers continued to flog potential competitors with her patent disclosure, even as we had become legally untouchable by virtue of the arbitration agreement.

It had been a matter of good intentions gone bad all around. Everyone was acting according to a deeply felt mandate, which just happened to be at cross-purposes with the others. It was a Greek tragedy. Ayers was looking for the recognition that was her due, and she now saw that threatened. She had rescued our entire family health-wise; we had worked together for three years in partnership, and our children regarded her almost as an aunt. There was pain on all sides among the former partners. But none of us were in a position to yield.

In the midst of our legal brouhaha with Margaret Ayers, our son Brian died in a nocturnal seizure. It had been six years to the day since starting neurofeedback. He was in his last year in college, doing beautifully in computer science and mathematics. He was also spending summers writing game software for our NeuroCybernetics system. Brian wrote the popular Mazes game, which emulated Pacman, and also the Boxes game.

Digital implementation of the Sterman protocol allowed the use of video screens for the feedback, which in turn made it a lot easier to display the dynamics of the training band signal. The amplifier was the only analog electronics that survived, and it was built for us by Tim Scully, who, incidentally, had made his name originally by manufacturing LSD for the Summer of Love in 1967. The bloom was off the LSD rose very quickly, however, and Tim turned his attentions to what was then called EEG biofeedback. The Feds had long memories, however, and he got twenty years in the penitentiary for his labor of love on LSD, of which he served ten. He got his Ph.D. while incarcerated, and continued his work on neurofeedback electronics development once he got out.

Affordable QEEG Analysis Arrives

By 1991 Barry Sterman had seized upon the new capability of doing quantitative analysis of the EEG within the scope of a private practice, and saw that as the path toward a truly scientific foundation for neurofeedback. He did not abandon his SMR-training by any means. That remained standard for the reward-based aspect of the protocol. But the QEEG capability would inform a complementary inhibit design. Like a dreadnaught coursing through a narrow shipping channel, its passage ripping all boats from their moorings, QEEG-based neurofeedback was compelling in its allure, particularly with Sterman as its cheerleader. Pining for recognition by a recalcitrant mainstream, many leaders in the field were confident

that marching under the flag of QEEG-informed feedback would lead to the breakdown of the walls of resistance and serve as the path to scientific respectability.

Unfortunately, the opposite happened. QEEG analysis was totally foreign to the field of psychiatry at the time; the field of neurology was using that technology only for the very limited purpose of localizing seizure foci and structural anomalies; and psychologists would be taken even further out of their comfort zone. Advocacy for neurofeedback now faced two major challenges instead of one.

Meanwhile, we saw no need to make radical changes in what we were doing. It seemed that our results were clearly better than anyone else's, as testified to by the fact that our reports were being rejected on all sides. The problem was this: our claims of rapid results in training were plainly incompatible with the operant conditioning model. Joel Lubar stated quite firmly that no one can credibly claim results in less than fifteen sessions. But instead of drawing the conclusion that the operant conditioning model needed refurbishing, it apparently was thought more plausible to just reject the claims of the new upstarts in the field.

Here we have yet another irony. It was Barry Sterman's own instrument design that led us to discover the critical importance of rendering the dynamics of the signal visible to the trainee. But, Sterman himself never credited that finding, insisting for all his remaining professional life upon the standard Skinnerian operant conditioning model. This early rejection of our claims led to a divergence between our own subsequent development and that of the "official" standard model, namely QEEG-based training. Implicitly, it also led to a divergence between the standard operant conditioning model and what later came to be known as endogenous neurofeedback.

For a while in our training we continued to give lip service to the operant conditioning model, in the interest of maintaining a united front within the field to the degree that was still possible. But at some point Val Brown simply announced that we were not really doing operant conditioning, and that forced our hand to acknowledge that he was quite right. That represented a final breach with conventional thinking.

Without getting too much into the weeds here, it might also be useful to recall that with real-time computer analysis of the EEG using the Fast Fourier Transform, it became standard within the field to do feedback on transform-based data. This meant windowing of the data, which in turn meant an even further suppression of the dynamics of the signal. It got to the point where people on the two sides of this divide no longer even communicated very much.

Optimal Response Frequency and Infra Low Frequency Training

The subtlety in the feedback achieved by including the dynamics of the signal made it possible for Sue to discern the frequency-specificity of the training effects. Operating at what we termed the Optimal Response Frequency (ORF) of the client further accelerated the training, and also expanded the clinical reach to previously intractable conditions. The response could be so prompt that adjustments had to be made within session, sometimes within minutes of beginning the training. Colloquially, this has also been called "sweet-spot" training, which unfortunately tends to trivialize the concept, and to obscure the theoretical significance of the existence of such special frequencies.

Another major milestone in development of the present Othmer method was Sue's discovery of the importance of training infra-low frequencies, i.e., below 0.1 Hertz. Once we entered the infra-low frequency regime, discrete rewards fell away completely, and the last vestige of the operant conditioning design was jettisoned from the 'reward' aspect of the protocol. In one sense, then, it could be said that we had cut our moorings completely from our launch point within Sterman's original model. But in another sense, our approach retained more of the essentials of Sterman's original method than any other within the field.

Our entire approach was based on what is called "mechanisms-based training". We placed our electrodes on the basis of functional neuroanatomy and neural dynamics, the very same rationale that was the basis of Sterman's choice of placement and frequency for the SMR training on the sensorimotor strip. Some frequencies were indeed special; they just differed among people. Everybody got trained with the same basic placements and similar training frequencies. Electrode placement with our core protocols is invariably bipolar, in line with all of Sterman's and Lubar's early research. And the accompanying inhibit design is also familiar from the early days of Sterman and Lubar.

So, what was accomplished with the gradual migration to training in the infra-low frequency region? Our clinical reach or footprint got ever larger as we went lower, and that led us to the realization that we were reaching the foundations of the cerebral regulatory hierarchy. At the same time we were also migrating to a priority for right-side electrode placements. This meant that we were approaching the foundations of the developmental hierarchy. A unitary model emerged in which the developmental hierarchy, the regulatory hierarchy, and the frequency hierarchy were simply different frames in which to regard the same underlying reality.

Emergence of a New Theoretical Model

We are far removed at this point from specific diagnoses and conditions. The question for us is much more about the basic competence of the nervous system to manage its primary

role, which is to regulate its own affairs. The brain must be regarded in the frame of a control system. Its primary burden is to maintain its own stability—the capacity to sustain function—through all circumstances. The principal issue here is neuronal hyper-excitability—i.e. at the membrane level—and excitability at the network level. The latter is accessible to us in training. The brain's secondary burden is to sustain homeostasis—or homeodynamics--with respect to core functions of arousal regulation, affect regulation, autonomic regulation, and interoception. Finally, there is the matter of preparedness to engage with the outside world volitionally, the realm of intentionality and of responsivity.

It is now understood that these core functions are not localizable in the brain, but they are spatially differentiated. Considering the brain as a unitary entity, the critical regulatory variable is core physiological arousal. The primary spatial segmentation is the hemispheric divide. Consistent with their divergent, complementary responsibilities, the two hemispheres have distinct training requirements. The right hemisphere has primary responsibility for the foundational regulatory functions, whereas the left has the primary responsibility for engagement with the outside world. We have found that inter-hemispheric coordination is critically involved in the maintenance of global stability.

The next spatial segmentation in the hierarchy of regulation is the front-back axis, the input-output divide. We have ended up with a protocol sequence that gives priority to this functional differentiation by quadrants. The training of each quadrant is associated with particular functions, for which we have specific expectations in the training process. These expectations guide the optimization procedure.

With respect to the intrinsic connectivity networks (ICNs), our priority lies with the Default Mode Network and the Salience Network. We have to access these networks wherever possible at the cortical surface. With respect to the Default Mode Network, the relevant sites happen also to be the multi-modal association areas, the areas known for the greatest effective functional plasticity, the areas that are the last to reach maturity in the nervous system, and the areas of greatest vulnerability on aging. It is important to observe, however, that nearly all of our standard electrode placements emerged out of our clinical work well before ICNs were first identified in fMRI studies.

More recently, our approach has been refined on the basis of further elaboration of neural network dynamics within the framework of the ICN model. And indeed, an entirely new core protocol has emerged directly out of the ICN model, combined with the clinical experience of David Kaiser that is based on his SKIL analysis program.

Our developmental thrust over the past three decades has mainly been on the core protocols that have just been discussed. They do not exhaust the clinical agenda, however. First of all, these basic protocols are not restricted to the ILF regime, but can be deployed

within the conventional EEG spectrum as well, with application of appropriate frequency rules. All the basic protocols were solidly established in the EEG range, after all, well before we entered the ILF regime. These protocols are complemented by synchrony training within the EEG range, and with Alpha-Theta training. And, we do rely on the QEEG when faced with particular ambiguities; but that happens only rarely, and never at the outset of training. It is the training process itself that reveals to us what a particular nervous system needs, and it does so very efficiently. This is endogenous neurofeedback.

An Overview

An approach to brain-training has been developed that is wholly distinct from other forms of neurofeedback. The latter are generally clinician-centered in the sense that the clinician decides what a particular brain needs for its recovery, based either on QEEG analysis or other considerations. The specific techniques, whatever they may be, are targeting the remediation of deficits. They are deficit-focused, and therefore emphasize the identification of deficits in the EEG before the training process even begins.

Our approach, by contrast, is client-focused, and it is function-focused. The objective is the restoration of functional integrity---globally, and from the bottom up. It is the brain's response to its own activity that effectuates the remediation, and simultaneously reveals what remains to be accomplished. The neurofeedback signal can be seen as a kind of 'augmented reality' for the brain to facilitate the restoration or enhancement of self-regulatory capacity. The process is entirely self-referential, hence endogenous. The response, however, may well be idiosyncratic. The clinician functions as a knowledgeable collaborator who keeps matters on track and decides on the optimal path forward, subject to mid-course correction by virtue of subsequent experience.

Our training process is layered, sequential, progressive, and cumulative, with early stages setting the table for subsequent protocols—a kind of scaffolding process. The method is obviously needed the most by those whose state of dysregulation is profound and pervasive. That includes the sequelae of early childhood maltreatment trauma or trauma grounded in early neglect, as well as early mild brain injury. It includes the autism spectrum, the personality disorders, the addictions, and developmental delays of all kinds. It includes the eating disorders, the movement disorders, and the dementias.

We have found that nearly everyone benefits from exposure to these basic protocols, often to a surprising degree. People generally have a normalcy bias, the 'healthy illusion' that they are actually OK. They may well be surprised by how strongly they react to these protocols, an indication of sub-optimal self-regulatory control. More than that, we say that no

one really knows the inherent functional competence of their nervous system until it is trained with ILF neurofeedback.

Yet more irony lies in the realization that ILF neurofeedback is much closer to traditional biofeedback than Sterman's operant conditioning procedure. Biofeedback training signals are usually continuous; there are typically no thresholds; there is no obvious divide between the functional and the dysfunctional realms; and the entire approach is function-focused rather than dysfunction-focused: improved regulatory competence is the goal. It could be said the ILF neurofeedback reunites the fields of peripheral biofeedback and neurofeedback in regard to that fundamental orientation. But there are key differences as well. Whereas the biofeedback field succeeded in getting the conscious mind and volition into the discussion, we've had to take it out again.

Our process involves the brain interacting with information about itself, and the brain's owner is irrelevant to that aspect of the process. Clients cannot help by means of cognitive engagement because the signal itself does not reveal where good function lies. The signal only makes sense to the brain that gave rise to it, and even then it makes sense only when it is being observed in real time. The brain's interaction with the signal is firstly a matter of recognition. The brain is witnessing an aspect of its own dance, which invites its immediate engagement in the same manner as a real dance.

Concluding Observations

Finally, a reflection on what has transpired over the past thirty years. Maturation of the field has taken place according to two paths: one is the top-down, theory-driven, prescriptive, sequential path. Related research is typically narrow in scope, tightly defined, and hypothesis-driven. The objective is to rule out the null hypothesis as a pre-condition for moving forward. Studies are frequentist in character, with group size sufficiently large to yield adequate power to resolve the issue of significance. A lot of effort is expended for modest results.

The other path is bottom-up. It is naturalistic and observational in character. Typically there are several working hypotheses being entertained at the same time, each informed by prior findings, and all of them provisional. Client data bearing on hypotheses are obtained episodically and somewhat randomly. Confidence in the hypotheses consolidates via a process of multimodal Bayesian inference that is based on strength of evidence. Additionally, one looks for 'multiple, independent determinants' in support of the hypotheses, which by this process then either gain or lose support with the passage of time.

Over the 34-year span of development of our method, this clinically-driven process critically involved the contributions of top instrument designers, software developers, experts

in signal processing, and game designers, all in collaboration in a feedback loop with scientifically schooled clinicians. Three generations of instruments were developed: NeuroCybernetics, EEGer, and Cygnet, each with its own distinguished designer at the helm: Edward Dillingham, Howard Lightstone, and Bernhard Wandernoth—each with extensive prior history in aerospace research that involved the development of instrumentation and software for real-time applications.

The past three decades can be seen as an experiment of nature in which the two paths toward acquiring scientifically validated knowledge have co-existed, and to an extent co-habited. Bayesian inference is always in play, officially or unofficially, and frequentist analysis is often relied upon, sooner or later, to firm up clinically-derived findings. The bottom-up approach is the more suitable for exploratory development, and that has proved to be particularly valuable in the case of neurofeedback. This approach is even more likely to dominate in the future. We are entering the age of healthcare progressing on the timescale of software development. With a competent, versatile platform in place, the feedback loop between design, execution, and validation of new initiatives is shortening. We are optimistic. After all, we are still just at the beginning of the age of the brain.

Key to the Literature

The broad clinical footprint achieved with our SMR-beta training protocols by the late nineties is covered in Othmer et al. (1999). Outcomes achieved with our SMR-beta protocol are best illustrated quantitatively using results obtained with a continuous performance test. Training outcomes were evaluated for 1089 participants at 32 clinical practices using the method. Impulsivity was diminished overall, and substantially normalized in the deficited population (Kaiser and Othmer, 2000).

In the largest controlled study performed to date in neurofeedback, our SMR-beta and Alpha-Theta protocols were utilized in a replication of the Peniston protocol for the treatment of drug and alcohol dependency and addiction in the mid-nineties. In a four-year study that involved three-year follow-up of sobriety and abstinence, 121 participants in two matched groups compared standard addictions treatment with and without the neurotherapy component. Outcomes favored neurofeedback by a factor of three after one year. Sobriety was largely sustained among the neurofeedback cohort in three-year follow-up, while continuing to attrition among the controls (Scott et al, 2005).

Our study on application of neurofeedback to Complex Regional Pain Syndrome (Type 1) took place just as we were approaching the threshold of Infra-Low Frequency training (Jensen et al, 2007). Application of ILF neurofeedback to Post-Traumatic Stress Disorder is presented in a clinical perspective in Othmer et al. (2011). ILF neurofeedback is discussed comprehensively, in both clinical and theoretical perspective, in Othmer et al. (2013). The history of the development of neurofeedback is reviewed in Othmer (2015), and the milestones of development of the Othmer Method are covered more formally in Othmer (2017).

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