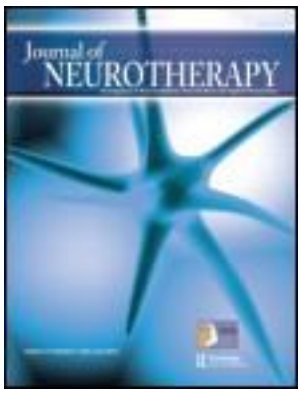

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Clinical Corner

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CLINICAL CORNER

D. Corydon Hammond, PhD, Editor

The purpose of the Clinical Corner is to provide responses to clinically oriented questions that may not, in many cases, have been evaluated yet by research. Therefore, the personal opinions expressed in the column are exactly that, the opinions of the individual authors, often based on their clinical experience. The opinions shared belong to the authors and are not necessarily those of the Society for Neuronal Regulation (SNR), or the Journal of Neurotherapy. Nonetheless, it is hoped that the diversity of opinion expressed in this column will stimulate thought and the further exchange of ideas.

Readers are invited to send questions for consideration to: D. Corydon Hammond, PhD, University of Utah School of Medicine, PM&R, 50 North Medical Drive, Salt Lake City, UT 84132. E-mail address: D.C.Hammond@m.cc.utah.edu

A recent Clinical Corner topic focused on the potential for occasional adverse reactions from neurofeedback training. This elicited a brief clinical case example from Dr. Dan Chartier, which illustrates the need for individualization and careful assessment prior to neurotherapy. In this month's Clinical Corner, we also have a response to a question about synchrony training in neurofeedback. Finally, we have three responses (one from an engineer, and one from a seasoned clinician, and an introductory response by myself) to a question raised by a past column about controlling for EMG contamination when doing 40 Hz neurofeedback training.

AN ADVERSE NEUROFEEDBACK REACTION, OR, THERE IS NO SUCH THING AS A NEUROFEEDBACK "DEMO"

Dan Chartier, PhD, Life Quality Resources, Inc., 8404-B Glenwood Avenue, Raleigh, NC 27612 (E-mail: DanChart@aol.com).

A number of years ago an experience occurred that forever changed my perspective on the potential impact of even brief neurofeedback training. That experience could be seen as an adverse neurofeedback reaction and it happened like this: A friend, a 43-year-old male, was visiting my office and wanted to try neurofeedback. He was a typical stressed out administrator so we elected to have him try about 20 minutes of Alpha-Theta enhancement (Peniston protocol) in an effort to promote rapid relaxation. The equipment used was the ROSHI by Chuck Davis, which has the unique feature of yoked dual channel training. We chose to do the training demonstration at P3 and P4 for 20 minutes. My friend proved very successful at increasing his levels of alpha and theta.

When I saw him again a week or so later he told me about an interesting and troubling side effect that his neurofeedback experience had produced. He said that when he got home after the session he felt very spacey. This condition resulted in a "kitchen accident." He went on to describe how he was using kitchen shears to cut up a plastic drink can holder from a six-pack of soft drinks, an environmentally respectful task that he had done many times before. He said that as he was using the shears some part of his consciousness alerted him to the fact that he was holding the plastic in a different way that could result in him cutting the skin between his thumb and index finger. Despite this internal warning he said he was "unable" to make the correction and did indeed give himself a nasty cut. He attributed this to the degree of mental spaciness he was experiencing and a sort of "it doesn't matter" state of mind.

When he told me about this experience I asked him to let me do a QEEG. The results showed that he, in fact, had an ADD (elevated slow frequency) type of dominant pattern. It was then apparent that his brief "demo" session of neurofeedback reinforced his already underlying potential for inattention and excessive slow brainwave activity, and thus, helped set the stage for the "accident."

The clinical lessons I learned from this experience are: first, there is no such thing as a simple neurofeedback demonstration (at least not with the ROSHI). Every experience of neurofeedback, even brief ones, can result in some shift in consciousness. Second, we should not operate on assumptions, but rather, do a QEEG to guide the training. Not know-

ing an individual's dominant frequency and brainwave pattern can result in reinforcing the wrong "direction."

SYNCHRONY TRAINING

QUESTION 1: "What is the value of synchrony training? When would it be indicated, what are the possible risks of using it, and when would it seem contraindicated?"

RESPONSE: Les G. Fehmi, PhD, Clinical Director, Princeton Biofeedback Center, 317 Mount Lucas Road, Princeton, NJ 08540 (E-mail: lesfehmi@ix.netcom.com).

Introduction

Webster's Collegiate Dictionary defines synchrony as events occurring at the same time–having the same period and phase. Using a differential amplifier in referential mode (that is, putting the reference sensor on a "relatively" electrically quiet location such as an earlobe, nose, or the seventh cervical vertebrae) one may monitor brain activity from a *local* region on the scalp with the active sensor. When the amplitude of monitored activity increases, then we know that more excitatory neuronal synchronization and electrical summing has occurred in that region. Thus, increases in amplitude reflect *local* brain synchrony, which improves figure-ground relationship, by increasing the signal-to-noise ratio (Fehmi, 1969; 1970; Fehmi, Adkins, & Lindsley, 1969; Fehmi, Lindsley, & Adkins, 1965). The greater the amplitude, the more clearly the synchrony in the *local* brain region stands out from the noise.

More *global* synchrony is monitored as we add widely separated sites of *local* synchrony (each monitored referentially) which have the same period and phase. When bipolar monitoring is used, a confusing recording is obtained because the differential amplifier subtracts the reference brain potentials relative to ground from the active brain potentials relative to ground (ground can be placed anywhere on the body). When the active and reference sensor potentials are in phase synchrony with each other, the synchronous brain activity of these two local regions is partially or totally subtracted out, depending upon their amplitude symmetry. On the other hand, 180-degree out-of-phase activity is amplified using bipolar recording and the associated feedback signal increases, thus rewarding the absence of synchrony. Problems in

recording *global* and *local* synchrony encountered by using bipolar versus referential recording are described in Fehmi and Sundor (1989).

Both synchrony and certain forms of asynchrony can be 100 percent coherent. In-phase synchrony is a special case of 100 percent coherence. Phase synchrony and coherence can be trained (Fehmi & Selzer, 1980).

Of the forms of neurotherapy of which I am aware, almost all training protocols depend upon local synchrony at various frequencies (e.g., C3 at 15-18 Hz or C4 at 12-15 Hz). My own research is oriented toward discovering the value of in-phase synchrony forms of coherence approaching 100 percent. I use only referential recording techniques and train for *global* synchrony by sampling brain activity from eight major areas of the brain, placing electrodes at Fpz, Cz, Oz, T3, and T4. Midline placements reflect increased bilateral in-phase activity. The remarks that follow are limited to *global* synchrony.

The Value of Synchrony Training

There is little research that relates directly to this question (McKnight & Fehmi, 2001). However, many years of observation supports the conclusion that training *global* synchrony at various frequencies has clinical value (Fehmi, 1978; Fehmi & Selzer, 1980; McKnight & Fehmi, 2001). Having the ability to increase and decrease *global* synchrony at will correlates with attentional options; respectively, to let go into an open and immersed awareness and alternatively to again grip experience with a narrow focus and objective awareness. This bi-directional control, in the event that it is frequently exercised, supports vibrant sensation and holistic, effortless perception. It represents the physiological underpinnings of the figure-ground relationship so necessary for attention as we know it. Equally important, the flexible control of EEG synchrony and associated attention manages the accumulation and resolution of stress. As fixated attentional biases give way to attentional flexibility, the accumulation of stress gives way to "on the spot" stress management and stress dissolution. The occurrence of stress within limits is not necessarily bad and, in fact, may provide a needed and healthy flexing of emergency functions. However, prolonged sympathetic, autonomic stimulation and chronic accumulation of the effects of stress can eventually exceed thresholds for a large variety of symptoms due to pervasive sympathetic autonomic anatomical innervations. The most important finding of this research, in my opinion, is the observation that various attentional parameters are reflected by EEG activity (Fehmi, 1978;

Fehmi & Selzer, 1980), amplitude, frequency, and *global* synchrony (e.g., *global* synchrony represents the physiological mechanism of open and immersed attention, Fehmi, in press).

Indications for Global Synchrony Training

If *global* brain activity does reflect attentional types, as I believe it does, then learning to increase and decrease *global* synchrony would enhance attentional flexibility which is necessary for stress dissolution. This is a most salutary and broad-reaching benefit of *global* synchrony training. As we observe, *global* synchrony training is a powerful aid for anyone interested in symptom remission or optimization of function.

Possible Risks and Contraindications of Global Synchrony Training

The neurofeedback provider must proceed cautiously with a client who is considered fragile or over-reactive to possible release phenomena, such as anxiety. One might begin by slowly teaching open attentional skills. Thus, in my experience, any unpleasant phenomena will be released into an expanded awareness containing many simultaneous sensations, causing these phenomena to be experienced as a small part of a large background experience. That is, any released noxious material is experienced as "small stuff." This is in contrast to seeing it as "big stuff," when this material is focused upon narrowly and is all that is in awareness, thereby eliciting a strong reaction. Along with *global* EEG synchrony training, a standard series of attention training tapes (Fehmi, 1977) which are taken home for twice-daily use, usually provide substantial support, and in my experience considerably speed the process of training. They also provide the necessary attentional strategies for dissolving physical and emotional pain.

The only other caution that comes readily to mind concerning *global* synchrony training is one that is shared with local synchrony training, the possibility of triggering a seizure. Thus, for those trainees who have a history of seizure activity, avoiding low frequency training in the theta and delta ranges is advisable. In fact, the combined use of *global* EEG synchrony training at frequencies above 9 Hz, with home practice of attention training with audiotapes, has in my experience been effective in reducing seizure activity. It is my opinion that training bi-directional control (increasing and decreasing *global* synchrony) undermines the likelihood of the runaway forms of synchrony, which underlie seizure activity. That is, learning also to decrease synchrony provides a signifi-

cant measure of control over induction of aberrant forms of synchrony. Considering its value in enhancing information transport in the brain, its value as an attentional training aid, and its value as a means for personal integration and optimization of function and performance, any contraindications to *global* synchrony training are worth taking the trouble to circumvent, when possible.

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CONTROLLING EMG DURING 40 HZ TRAINING

QUESTION 2: A perfectly reasonable question was asked about a tough problem in a past Clinical Corner: "How can we do 40 Hz training without just increasing EMG?" Val Brown or Marvin Sams did not, in my view, adequately answer the question. The essence of what they asserted was that it's not a problem and enhanced EMG is probably inherently incompatible with 40 Hz. Both correctly suggested the use of

collateral EMG placements, but neither was specific about specifying an algorithm to remove 40 Hz. Dr. Brown said that the "time-frequency signature" of the 40 Hz and EMG are different. I'm not familiar with that phrase. If he is saying EMG does not reach down to 40 Hz and below, he is mistaken. Dr. Sams refers us to Dan Sheer's comparator circuits (no longer available). The question actually asked how one could ensure that EMG is not mistaken as 40 Hz by the hardware/software system. I would love to hear what some of the engineers say about that.

RESPONSE: D. Corydon Hammond, PhD, Professor, Physical Medicine & Rehabilitation, University of Utah School of Medicine, 50 North Medical Drive, Salt Lake City, UT 84132 (E-mail: D.C.Hammond@m.cc.utah.edu).

The seminal work on 40 Hz training by Daniel Sheer (1975) involved a sophisticated recognition that EMG contamination could be involved since this frequency overlaps with the muscle spectrum. He used bipolar leads at O1-P3 and created a "muscle comparator," with a bipolar recording from the neck and temporal muscles on the same side. He explained:

> For the EEG leads an anion gate circuit allows the 40-Hz output to trigger reinforcement only when it is not coincident with the 70-Hz output, which is used as an index of the polyphasic muscle. In addition, when a 40-Hz muscle signal from the muscle comparator coincides with a 40-Hz EEG signal, the slide projector [his reinforcement device] will again not trigger. When the output from the EEG comparator is neither coincident with the 70-Hz EEG signal nor with the 40-Hz muscle signal, it activates the stimulus control unit which triggers the slide projector. (p. 333)

Sheer's (1975) EMG inhibit was from 62-78 Hz with a center frequency of 70 Hz. He reinforced the range of 36-44 Hz. It was his experience that:

> . . . there were significant increases in the 40-Hz power bands for the normal children during problem-solving situations but not in the bordering 31.5 and 50 Hz bands set up as controls (Sheer & Hix, 1971; Sheer, 1974). There is no reason why polyphasic muscle, with a relatively higher amplitude at 50 Hz, should show up differentially in the 40-Hz band but not in the 31.5- and 50-Hz bands. (p. 334)

Sheer (1975) discovered that during eight sessions of training there was a 160% increase in 40 Hz activity, a 65% increase in beta (21-30 Hz) activity, and no significant increase (16%) in muscle activity. In contrast, after eight sessions of training to suppress 40-Hz, there was a 79% decrease in 40 Hz activity, an 18% decrease in beta, and a 15% reduction in EMG activity. All of this led him to conclude:

> With proper controls the conditioning of 40-Hz EEG can be dissociated from muscle activity. There is a significant but low degree of common variance between 40-Hz and beta (21 to 30 Hz). The distribution of correlations between 40 Hz and beta for the different sessions, combining conditioning and suppression, generally ranged from .35 to .45, which indicates about a 20 percent common variance. It is understandable that there should be a significant common variance–perhaps larger if error variance were reduced–because beta and 40 Hz represent different aspects or functioning of a common arousal process, diffuse and focused. At the same time it should be recognized that the different functions must have other parameters that are distinct and significant because there is a considerable variance, which is not common. (p. 345)

More recently, Davidson and his colleagues (Davidson, Jackson, & Larson, 2000) have discussed their procedure for controlling EMG when simply investigating gamma band activity in research. They advocate something that no one to my knowledge has done in the field to neurofeedback–deriving a measure of power in an EMG band from every EEG lead that is used. They then "regress this value on power in the traditional EEG bands to derive measures of EEG power with the component accounted for by EMG removed" (p. 36). They sample at 250 Hz and use the band of 70-100 Hz as their EMG band to extract power from, then deriving EMG power separately for each electrode site. Taking into account Sheer's work and adapting Davidson's EEG research procedures to neurofeedback, it seems to me a very sound idea to control for EMG activity above an established initial baseline level with a built-in inhibit in the 70-100 Hz range.

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RESPONSE: Frank Deits, Engineer, Focused Technology, P.O. Box 367, Ridgecrest, CA 93556 (E-mail: frank@focused-technology.com).

From an instrumentation perspective we can certainly measure activity in a band around 40 Hz. The width of the band will determine the response delay with a narrower response producing a longer delay. This is known information that applies to any band we chose to instrument.

Signals in the 40 Hz band can originate from both EMG and EEG sources. Whether there is any characteristic that can distinguish one from the other is for the neurofeedback types to argue. Sheer (1975) has done a good job of discussing this issue in his article.

Use of signals in some other range to control for EMG is subject to the same conditions I noted above. A signal at 70 Hz as used by Sheer (1975) could originate from EMG or possibly from EEG activity as well. I personally would suggest using a relatively high frequency band such as 100-500 Hz to control for EMG. This should be out of the range of known EEG activity and is very responsive to EMG in the scalp muscle.

Use of a different site for EMG control requires making the assumption that the entire scalp produces homogeneous EMG activity. I have informally observed the ability of a subject to locally control EMG in the scalp muscles. In designing methods to use an EMG control signal one should keep in mind that non-synchronous signals do not add algebraically, but rather combine as the sum of the square root of the squares of the individual signals. As an example, a 2 μV EEG and a 4 μV EMG will combine as 4.47 μV, not 6 μV as might be expected. This has implications for designing an EMG artifact control.

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RESPONSE: Michael Thompson, MD, ADD Centre, 50 Village Centre Place, Mississauga, Ontario, Canada, M9A 3S4 (E-mail: LandMthompson @cs.com).

Though I cannot comment from an engineering viewpoint, here is a clinician's way of ensuring that EMG artifact is not mistaken for 40 Hz activity associated with cognitive processes. The way we try to do this in North America is to place a very severe inhibit on 45-58 Hz activity. (In Australia and Europe we use an inhibit across 44-48 Hz). The microvolt level for the inhibit is determined after measuring the average activity in that range when EMG activity is minimal. This, of course, can only be viewed on instruments that show the spectrum up to at least 60 Hz.

With these inhibits in place, if the client's activity is around 39-41 Hz (frequencies we term the Sheer Rhythm after Daniel Sheer's work, 1975) and is due to muscle activity, they will not receive rewards. The rationale is that the EMG artifact will affect the higher frequencies even more than activity around 40 Hz. Most of the activity generated by EMG will have an influence above 60 Hz, though it does sweep down and it can increase activity even below 20 Hz, albeit to a lesser extent.

Clinicians are familiar with the principle behind a 45-58 Hz inhibit because it is the same principle behind using 24-35 Hz inhibits to get valid Beta readings; namely, a slight rise in 24-35 Hz activity due to EMG will raise microvolt levels of Beta (16-20) or even SMR (13-15) more than it affects Alpha and Theta. The 40 Hz problem is greater because the true amplitudes in that range are smaller to begin with, plus you are closer to the EMG range, so increases due to EMG have relatively greater effects. Thus, many clinicians feel you cannot do accurate, artifact-free feedback in the 40 Hz range.

The bottom line is that you have to be very careful in interpreting activity around 40 Hz because it is hard to measure accurately. It would be very helpful to see research produced on the question of whether encouraging this activity gets better/different results than feedback done at lower frequency ranges. Regardless of whether you work at the 40 Hz range, it is a good idea not to include the 38-42 Hz range in your inhibit frequency band for any work you are doing.

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