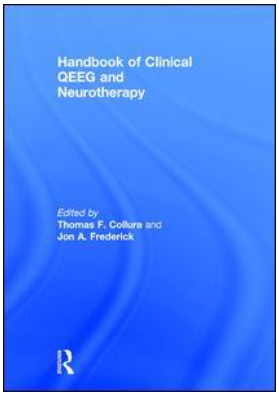

Handbook of Clinical QEEG and Neurotherapy

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Neurofeedback as a Treatment for Anxiety in Adolescents and Young Adults

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PART VII

Traditional Alpha/Theta/ Beta Protocols

23

NEUROFEEDBACK AS A TREATMENT FOR ANXIETY IN ADOLESCENTS AND YOUNG ADULTS

Cynthia Kerson

Abstract

Anxiety and its common comorbidities are disorders that rely upon psychological and medical intervention. Often anxiety is the symptom of underlying dysregulations. These dysregulations, developed over childhood, are both psychological and physiological. Thus, clinical intervention from a psychophysiological perspective may be the best approach and neurofeedback and biofeedback are often used in conjunction with psychological interventions. This chapter discusses the underlying mechanisms of anxiety as well as discusses a case in which both biofeedback and neurofeedback were provided.

Across the lifespan, diagnoses of anxiety and depression are on the rise, with the largest effected age group being teenagers and young adults (Blanco et al., 2008). The underlying issues facing this population are their need for excelled school performance, hormonal shifts, social pressures and changing family dynamics such as divorce, death or going off to college.

The 2008 study (Blanco et al.) found that close to 50% of all 19–25-year-olds has at least one diagnosed psychiatric disorder, with the highest of them being substance abuse. Depression and anxiety were also ranked high. (Note that these findings do not claim it is at this time in the young adult's life that the disorders may have begun.)

These alarming statistics might well be due to the marketing efforts of big pharma; after all, young adults drink. And they drink to relieve themselves from the pressures of evolving into adulthood. Normal age-related drinking aside, substance abuse is rarely a disorder unto itself; it is the self-medication of underlying psychological mechanisms, such as anxiety and depression as well as underlying physiological EEG mechanisms, which we will later discuss.

Medication is treatment as usual (TAU) for this population, who are prescribed SSRIs, anxiolytics and/or anti-seizure medications. However, of the alternative treatment options for them, neurofeedback has been shown to be one with the best outcomes with few adverse reactions or negative side effects. Neurofeedback is usually offered in complement to other treatments, which may be diet, exercise and peripheral biofeedback, such as heart rate variability, EMG or EDA, as well as EMDR and/or CBT. Thus, it is best when accompanied with life-style changes, which can be encouraged from these complementary and adjunct procedures.

Psychophysiological Bases of Anxiety

Evolutionarily, anxiety is a healthy behavior. It alerts and prepares for events that threaten safety and survival. Yet, it is not quite as useful when in constant exploit and in response to current, non-life-threatening stressors. Walter Cannon (1915), Philip Bard (1928), William James (1897), and more currently Robert Post (2007; Post & Weiss, 1998), Joseph LeDoux (1998) and Steven Porges (1995) have discussed the mechanisms underlying fear and subsequent behavioral presentations. During the past 100 or so years, we have learned, first from a behavioral perspective then from a physiological perspective, how chronic overarousal leads to systematic dysregulation.

These models look at the progression in the brain from stimulation to reaction. Kindling (Post & Weiss, 1996, 1998; Post, 2007) is a stress phenomenon, which occurs because of brain plasticity. Small appropriate brain responses to threatening stimuli lead to epileptiform spike behaviors if they become more abundant than is normal. On the cellular level, repetitive behavior strengthens neuropathways. When these behaviors are decontrolled they lead to poor regulation in important cortical structures, that then lead to deregulated brain behavior within the subcortex, then in the brain stem, and further to the autonomic nervous system, thus "kindling" regular events of anxiety, stress, depression and other pathologies that become chronic. This kindling action suppresses cognitive influence, making the behavior consistent, overriding normal, healthy reactions in the wake of distress.

LeDoux (1998) discussed the simple example of fearing a stick until we know it's not a snake; he describes a model in which the autonomic nervous system and limbic areas of the brain (specifically the amygdala) react before the cortex gets involved to save precious milliseconds when safety may be breached. This explains the flight/fight mechanism from a physiological perspective, which becomes overaroused (or kindled) the more the system is activated.

Porges (1995) described the freeze mechanism, which can replace the flight/fight as the preferred response behavior. He posits that the 10th cranial nerve, the unmyelinated vagal nerve (there are two; one myelinated and one that is not), which has deep connections to the other cranial nerves as well as to the heart and other viscera, will trigger an alternative to flight/fight, which presents as freeze, immobilization, passive avoidance, PTSD and the inability to move forward.

These three systems, kindling, flight/fight or immobilization, can be detrimental during the young adult time when life-long coping strategies are set up. How one responds to perceived threats is highly individual and involves any, or many, of the mechanisms described above. Regardless which, one's psychophysiological style of response/reflex is contingent on prediction of outcome, which is usually perfect at birth. However, with each negative outcome, each outcome that did not match the prediction, the belief structure becomes dysregulated. Enough incongruent outcomes and the system goes awry. Behaviors don't match expectations; tame situations spur wild and uncontrolled emotions.

For example, when a child is hungry the evolutionarily normal behavior is to bring attention to herself—maybe by crying or asking her mother for food. However, when this child brings attention to herself in this manner, she finds herself put into a room in isolation. Her expectation, or prediction, was that when she brought attention to herself, her mother would want to take care of her by bringing her food. However, this prediction failed to present itself and the child's emotional wellbeing (and possibly her nutritional health) becomes compromised. Now she no longer relies upon the instinctive behavior of attracting her mother to her needs. This cascades into a belief that most, or all, of her needs are not important to her mother. And as she matures, this becomes a deep-rooted dysregulation affecting many aspects of her life.

Finally, motivation plays a very important role with this population. The young adult has many obstacles to overcome and can easily get overwhelmed. Many have to fend for themselves for the first time. Life becomes exponentially harder and habits learned during this time will be set for life. Neurofeedback, because it is a long and somewhat slow process, requires a gauge of the level

of motivation, both during sessions and throughout the treatment process. When motivation is low the clinician must invoke engagement and commitment by creatively providing secondary rewards.

Neurofeedback Treatment Options

Adolescents and young adults tend to be very interested in their health care and often experience poor resolution from medication and other traditional treatments. Thus, they increasingly present to the neurofeedback practitioner. They are not quite as skeptical as their predecessors and are more open to new ideas. And over the past few years, the neurofeedback protocol and assessment options have increased, making it a more effective treatment modality than even 10 years ago.

The neurofeedback practitioner should look for three important brainwave patterns in the assessment that are shown to be specific to anxiety. These include (but are not necessarily limited to) excessive beta amplitude, which may include spindling; alpha asymmetry in the frontal lobes; and non-attenuating posterior alpha with eyes open based upon eyes-closed measure. These phenotypes are typically found in anxiety, substance abuse, PTSD and other disorders of hypervigilance (Johnstone, Gunkelman & Lunt, 2005). The authors discuss these and other phenotype patterns seen in the EEG, noting that often there is more than one present. While this population will surely present with at least one of these, others may also be present and I defer to Johnstone et al. (2005) for more information and suggest following the training recommendations specific to the pathologies.

Simply training the client to published protocols will often be a disservice. This author once worked with a client who did not want a QEEG and we proceeded to train with the alpha theta protocol. The client became more anxious and when finally convinced to do a 19-channel EEG and QEEG assessment, we found that there were alpha dysregulations at sites that were not being trained and that the alpha theta training was exacerbating them.

The clinician should also consider that only one training protocol might not be enough. Deciding which to train first depends on their severities and locations. This author has had better success training posterior pathologies first. Often they mitigate the frontal dysregulations simply due to network structures and the flow of neuronal communication. Basing protocol importance on symptoms and goals is also advised.

Missy

Recall the many reasons for anxiety in adolescents and young adults from the beginning of this chapter. You'll find most of them in this case. Missy is a 26-year-old woman who first came to me when she was 22 years old to help with ADD symptoms. She is very creative and was attending the art academy at the time and finding it hard to complete her studies. We did many sessions then to reduce theta frontally and then alpha posteriorly as indicated by her brain map. After finishing neurofeedback for the first time, she almost completed college, but was recruited by a leading movie producer before she finished her last semester. During this time, she found she could not control her alcohol consumption. As she stated, "I did not have an off switch, I would come home from work and drink a glass, then another glass and finally a full bottle of wine ending up too drunk to function." She had completely stopped drinking but proceeded to become dependent on pain medications for hip pain that she now knows was stress induced. During the last few months, she has been weaning herself off them, and was currently taking a very low dose of Vicodin every four hours.

Because it was successful for her in 2010, Missy thought she would revisit neurofeedback after she was laid off from her position, her dog and constant companion of many years died and she was about to be married. She had nearly chronic anxiety and pain. She would wake everyday at least an hour earlier than necessary, prepared to manage the inevitable morning anxiety event. She would listen to relaxation tapes, take her time eating, do her stretching exercises for the hip pain and walk her new

Table 23.1 Results of 2-channel pre-assessment.

Frequency	*Location*⋆	*μV*⋆⋆
Theta (5–8)	Frontal	6
	Central	6
	Parietal	9
	Occipital	7
Alpha (8–12)	Frontal	6
	Central	7
	Parietal	7
	Occipital	8.5
Beta (15–19)	Frontal	6
	Central	6
	Parietal	6
	Occipital	5
High Beta (20–30)	Frontal	10
	Central	10
	Parietal	8
	Occipital	9
Electrodermal Activity	Left hand	0.66–2.15
Heart Rate	Left middle finger	~ 70

⋆ F3/F4; C4/C4; P3/P4; and O1/O2
⋆⋆ Magnitude is the average of the homologous sites

pup while weathering her concerns about her future, her upcoming wedding and her aging parents, with whom she is very close.

I did not do a full 19-channel EEG because she was concerned about the expense. Therefore, I did a 2-channel assessment looking at frontal, central, parietal and occipital homologous sites. The results of that assessment can be found in Table 23.1. Note that within the 20–30 Hz range the 20–24 Hz range presented as mostly true EEG, often associated with worry and rumination, while the 25–30 Hz range represented muscle artifact as based upon the wave morphology and the presentation of Missy (when she tensed, this EMG was represented in the 25 Hz and higher range).

One particular pattern, "low-voltage fast," as described by Johnstone, Gunkelman and Lunt (2005), was found. I did not observe spindles. In their paper, they recommend posterior alpha suppression to remediate this behavior. However, we also saw that the alpha and theta were lower in amplitude than is considered normal, so we didn't want to train alpha down any further. I decided to do alpha (8–12 Hz) theta (4–7 Hz) training while suppressing 20–24 Hz at Pz. I monitored electrodermal activity (EDA) and heart rate (HR) to forewarn any shift in state that helped with "tweaking" the thresholds.

With alpha theta training, the client has her eyes closed so the sound reward is very important. They are usually water sounds, bells or deep tones that are conducive to relaxation. Feeling this to be intuitive, I usually set the alpha tone to be a bit higher than the theta tone, being sure they are distinct enough from each other. Also, while it's usually appropriate to make changes during the session and to inform the client of the changes, it is sometimes best to not disturb the client or you might compromise the deeper state that the training incites. It will be apparent whether to inform or not based upon your client's breathing and EEG.

To address the muscle artifact seen in the 24–30 Hz range, in the first session we trained trapezius muscle relaxation (electromyography or EMG). The active and reference sensors were placed on the belly of both the left and right trapezius muscles (upper back) (the ground was on the spine at about C3) and Missy was prompted to tense and release at differing intensities to help her differentiate how tense and relaxed muscles feel. She was able to reduce the muscle tension from approximately 8μV to 3μV and remain relaxed for at least three minutes at a time. So, with just this one session of EMG training, we felt she was ready to start the neurofeedback training.

For the first three neurofeedback sessions only, I monitored the EMG and Missy was able to maintain a low (approximately 3μV) level for extended periods of time throughout the session. If her HR and/or EDA became aroused, generally the EMG measure also did, however usually delayed (within a minute). HR and EDA levels reduce when relaxed. There is no optimal level for either because body type, age, gender, level of fit, medications and many other factors play a role; a full explanation of this is beyond the scope of this chapter. Mostly, we look for trends. Did the HR and EDA go up? Or did they go down? What if one went up and the other down? If both rise, the body is more aroused. Conversely, if both go down, the body is more relaxed. These trends are easy to follow. However, opposite trending, or fractionating, reflects more complicated issues that are determined as you interact with the client. If you're not sure, gently ask if your client needs a break, is OK—whatever feels right.

As you evaluate your client's needs based upon physiological measures during an alpha theta training session, the important issue is to determine whether he still feels safe. Because alpha theta training leads to deeper states and thus to exposure to suppressed emotional baggage, the physiological measures will inform you even before your client is aware of any state change. Making appropriate real-time changes to the neurofeedback protocol hinge upon these readings.

After nine weekly 20- to 30-minute sessions in which we trained the theta and alpha up and the beta and high beta down at PO3, Missy was having trouble with beta (15–19 Hz) (monitored, but not trained) and theta, which remained low regardless of training parameters. (See Figures 23.1a and 23.1b). At minute 6 the HR was 87, the EDA was 2.02 uSiemens and the alpha magnitude was

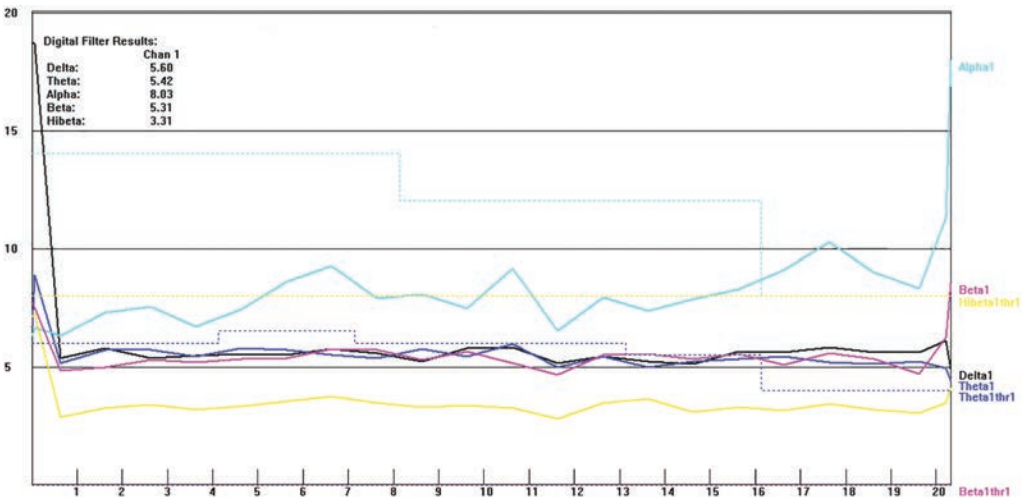

Figure 23.1a By session 9, you can see somewhat more typical magnitude levels except for theta and beta.

Note: this image shows magnitude at the following frequencies: delta (2–5 Hz), theta (5–8 Hz), alpha (8–10 Hz), beta (15–19 Hz) and high beta (20–24 Hz). Solid lines are magnitude levels averaged per minute; dotted lines are manually changed threshold levels. Y-axis is magnitude and X-axis is time in minutes.

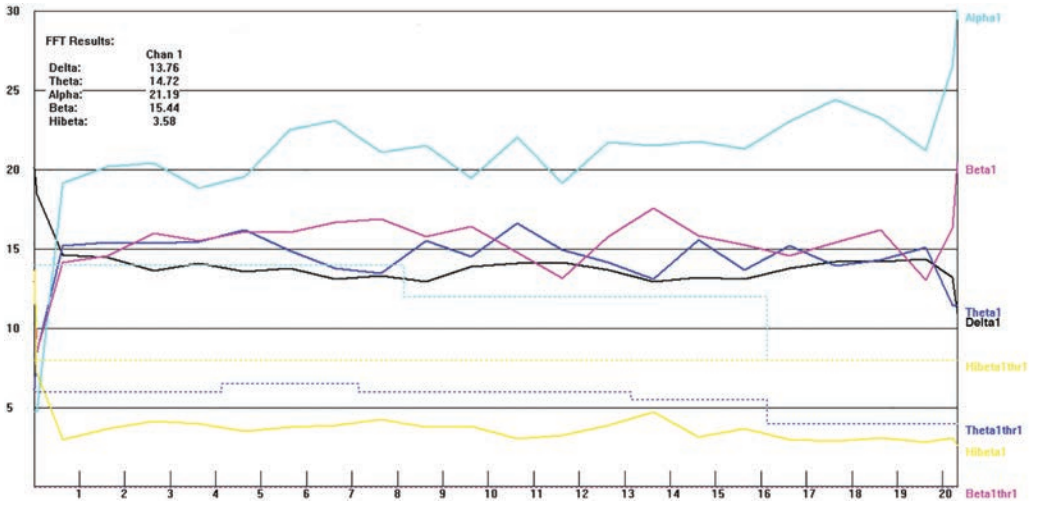

Figure 23.1b The slower frequencies are at what may be considered as too low magnitude, despite the proper proportion to the overall energy.

Note: some of these frequencies are changed from the assessment due to training needs. However, the conclusions from them are still relevant. Solid lines are relative power levels averaged per minute; dotted lines are manually changed threshold levels. Y-axis is % power and X-axis is time in minutes.

slipping, so I changed the threshold of the alpha range, as displayed by the dotted lines (from 14 to 11.5). Having checked in with her, and she realized she began thinking about unimportant things, I felt the change in threshold would enable her to transition back to the deeper state. (Interestingly, the beta and high beta increases slightly during this time as well). This is an example of how monitoring the HR and EDA allowed me to observe change in her state that may not have been as obvious by just looking at her or her brainwave patterns alone.

Importantly, by this time, the delta (2–5 Hz), theta (5–8 Hz) and alpha (8.5–11.5 Hz) frequency spans are reduced from those during the baseline assessment to accommodate changes in the EEG as the neurofeedback training progressed. I have found that reducing the frequency span increases the chance of success with most neurofeedback protocols, including with alpha theta training. However, this makes reporting cases difficult. So, please bear with me. Reporting on cases such as these tend to be more subjective in nature. To the hard-pressed scientist, I hope I don't offend.

Changing thresholds to make any of the criteria easier or harder depends on what is needed at the time and is very individualized. Recall, I am also monitoring HR and EDA, which help to reveal the client's internal state, especially when coupled with the EEG measures. For example, when the client is going into a deeper state, I may remove the alpha reward to better distinguish the theta feedback. At this point, the HR would be slower and the EDA level would be reduced from baseline measures (taken at the beginning of each session). This may also provoke the crossover (described below).

Adhering to the true operant conditioning model, I do not use auto-thresholding. I do not leave my client alone because I feel these micro on-the-fly changes are extremely useful in coaching and coaxing the client to attain the neurofeedback goals. (Staying with the client during alpha theta training is especially important because of the possibility of abreaction, which may occur if the client is not ready to revisit underlying trauma that is likely to resurface when they achieve the deeper states.)

Session 16 was an anomaly, and presented here to show that they can happen—that not all session results are expected. Missy was unable to attend to the training. On that day, she reported feeling concern about a prospective job offer and had a hard time "getting into" the training. The beta and alpha were within expected ranges, yet the delta, theta and high beta were not (see Figures 23.2a

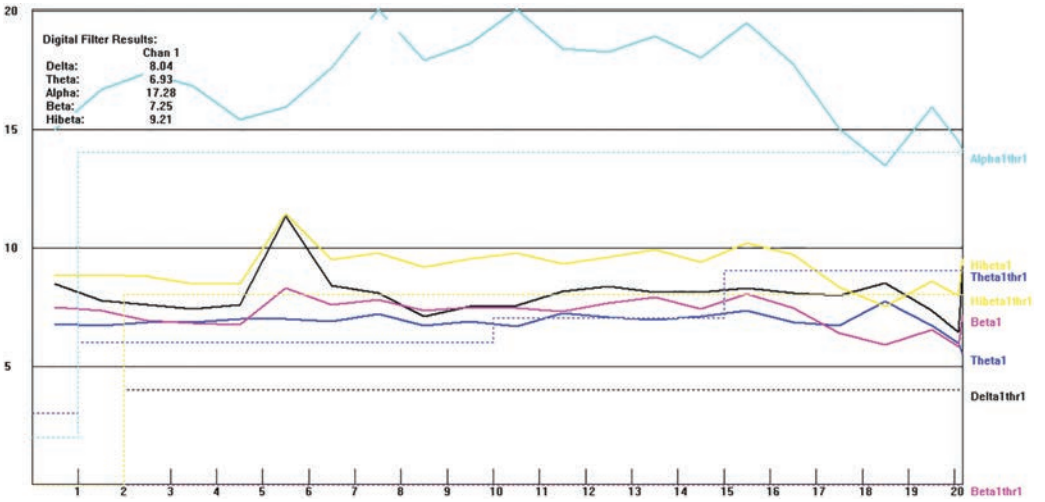

Figure 23.2a 20-minute eyes-closed session number 16. Peak-to-peak graph (magnitudes). Note high alpha (8–12), beta (15–19) and high beta (20–30 Hz) magnitudes as compared to low theta (5–8 Hz) and delta (2–5 Hz) magnitudes. Solid lines are magnitude levels averaged per minute; dotted lines are manually changed threshold levels. Y-axis is magnitude and X-axis is time in minutes.

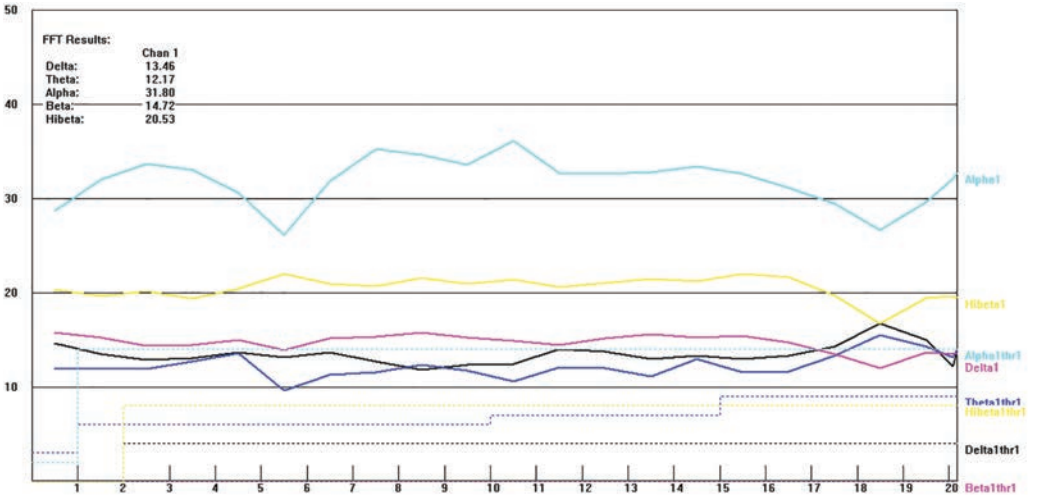

Figure 23.2b 20-minute eyes-closed session number 16. FFT graph (relative power). Solid lines are relative power levels averaged per minute; dotted lines are manually changed threshold levels. Y-axis is % power and X-axis is time in minutes.

and 23.2b). At first glance, one might think that since the alpha was out of range with the others, that it was too high. But in fact, it remained within normal range and the delta and theta magnitudes were depressed. So we started by training the theta frequency up and added alpha suppression training just to be sure it wouldn't rise along with the theta. As you can see, we were not completely successful until the end. HR and EDA were also somewhat elevated, averaging between 72 and 84, and 1.93 and 2.46, respectively.

Starting at session 21, Missy began experiencing intermittent "crossovers," where the theta magnitude becomes higher than the alpha magnitude for a period of time. In the literature (Kerson & Martins-Mourao, in press) this represents confirmation that the client is experiencing a deeper state—the state that we hope to achieve with alpha theta training. HR averaged approximately 68 and EDA at 1.75 uSiemens during these times, approximately 20% lower than when not in the crossover state. Note in the Figures 23.3 and 23.4 that only theta is being reinforced. At this time, Missy also began working with a psychologist to work through some issues that now began to resurface.

Missy had gotten married during this time, which relieved some of her anxiety. However, she still becomes highly anxious whenever an important event emerges. We leave off with her as she attempts to reenter the academic community to complete her Bachelor's degree and continue to do alpha theta

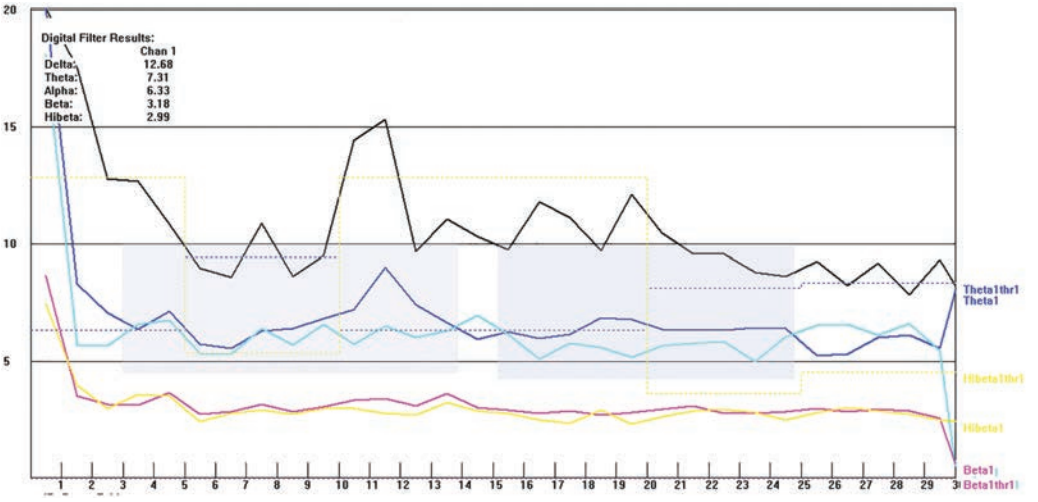

Figure 23.3 Session 21. Instances of the theta/alpha crossover. Y-axis is magnitude and X-axis is time in minutes.

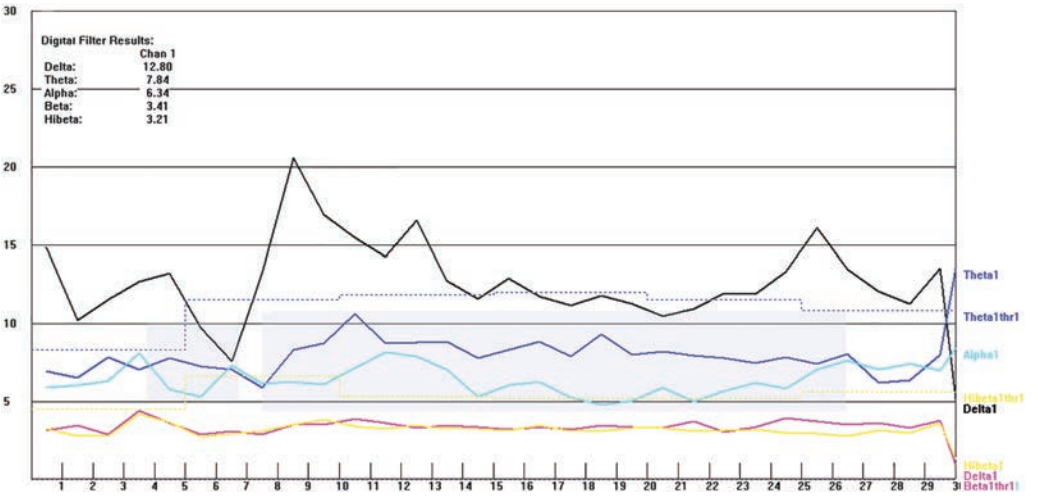

Figure 23.4 Session 24. Longer instances of the theta/alpha crossover.

neurofeedback sessions once weekly coupled with the psychotherapy sessions. She finds the neurofeedback sessions very helpful to remain calm for longer and longer periods of time. She states that she can almost always stop and refocus whenever a stressful event presents itself and that she has fewer mornings when she wakes up with unsettling anxiety. Missy still experiences some hip pain, although it is not as severe. She has reduced the dose of Vicodin further, and, along with her prescriber, planned to stop taking it within the next month. While feeling "50% better" she is fearful that the pain will reemerge and that the anxiety will never subside and she discusses these issues with her therapist.

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