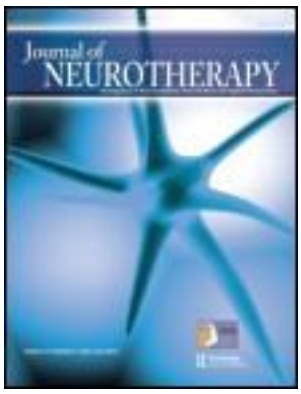

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Neurofeedback Overtraining and the Vulnerable Patient

Thomas V. Matthews, PhD

ABSTRACT. Neurofeedback overtraining in vulnerable patients can cause transient, site-specific functional decline that may be distressing to the patient and trainer. Susceptible patients can be identified before training with a checklist, and overtraining then avoided by close observation of training response. Procedures are described and a possible mechanism is offered.

KEYWORDS. Blood glucose, brain injury, EEG Biofeedback, IQ, neurofeedback, overtraining, prescreening, task fatigue

INTRODUCTION

I have found that when conducting neurofeedback training it is possible to overtrain in a session, which can cause a transient decrease in the cognitive functioning of the patient. The reduced functioning has been found to be specific to the location trained, with a reduction of the capacities that the training would be expected to enhance. This unwelcome outcome may last several days, much like the time course of treatment-benefit from a single session. This effect has been observed in EEG and Hemoencephalography (HEG) training.

A small fraction of general neurofeedback patients appear to be exceptionally vulnerable to overtraining. It seems that most of those may be identifiable through a simple checklist prescreening. In addition, paying close attention to the within-session treatment response early in the course of training seems to be a very useful way to avoid overtraining. I would speculate based on my experience that such vulnerable patients may contribute a large percentage of treatment failures (including dropouts) in settings where their vulnerability is overlooked. Particularly where the clientele have significant medical and brain-injury conditions, it is important that neurofeedback clinicians screen for and adapt to these needs.

Overtraining is a well-recognized phenomenon in athletics, an analogy that may be a useful heuristic when it comes to designing neurofeedback training.[1] Athletic overtraining can cause severe muscle and systemic fatigue, actually reducing the competitor's athletic capability until there is recovery (Jansen, 2001, p. 151). It is my observation in a 15-year neurofeedback practice that certain patients are dramatically more vulnerable to overtraining. They may tolerate—and benefit from—less than 5 minutes of training, whereas most would respond well to half an hour. They have the same vulnerability across diverse protocols

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including those based on a quantitative EEG evaluation. They generally do not overcome the vulnerability with training experience, though tolerance may increase marginally. Such patients have comprised perhaps 5% of my practice.

So far we have found two ways to identify such patients before they have an adverse response. A brief inquiry asking about blood glucose abnormality, brain injury, and IQ identifies most patients. Close attention to within-session treatment response by examining the minute-by minute microvolt changes (or watching for a drop in the HEG measures) early in the treatment course is also very important in order to identify fatigue on the specific treatment task.

INQUIRY FOR VULNERABLE PATIENTS

Vulnerable patients will show task-fatigue much earlier in a session than usually would be expected. Initial observations in this clinic have identified vulnerable patients as those who display three risk factors: any blood glucose abnormality, serious brain injury, or high IQ (meaning those with a high IQ before a brain injury, or when they train). It is speculated that possibly high IQ is related to rapid learning with neurofeedback. The strongest predictor has been blood glucose abnormality in the patient or a first-degree relative, including either diabetes mellitus, or hypoglycemia, whether diagnosed or merely suggested by appropriate symptoms. The greater the glucose difficulty, the greater the likelihood of overtraining vulnerability, and successful medical treatment of the glucose problem may reverse the vulnerability. In this context brain injury refers broadly to traumatic, anoxic, toxic, fever, infectious, or other serious damage. The three predictors seem to be cumulative.

Two years ago we used a simple checklist to screen some 30 patients for the risk factors just described. At the same time they completed Aron's (2000) questionnaire that seemed to have high face-validity for a purported syndrome called ''highly sensitive people'' (HSP). We did not say why they should take the questionnaire, except to indicate that doing so might help us provide better treatment to future patients. The great majority of patients had none of the risk factors, and during the course of treatment they routinely tolerated a half-hour of neurofeedback easily. Those few who displayed overtraining effects in treatment seemed to be easily identified by the checklist, and comprised maybe 5 to 10% of our patients. In my casual, informal assessment of the survey results it seemed clear that the risk factors were quite good in predicting vulnerability. On the other hand, the HSP questionnaire seemed to have no value in predicting vulnerable patients. Most of our clinic patient population have brain injuries, many rather severe. In my clinical experience, these conclusions have remained valid over the 2 years since the initial survey, identifying approximately 20 vulnerable patients.

Other observations may offer a potential mechanism that is involved. The connection between training and blood glucose was brought to my attention by a very bright head-injury survivor with diabetes. He discovered that eating so as to assure that he had adequate blood glucose levels before the session helped him tolerate more training in the session. He still tolerated only a few minutes but had a good treatment response overall. I noticed later that another traumatic brain injury survivor with diabetes was prone to utter exhaustion after an ordinary session, until he started insulin therapy. These observations led me to notice more closely when clients had less success in HEG training. When poor response was intermittent, it seemed to often occur after the patient missed a meal, and eating some candy in the session seemed to improve performance. If the problem was not intermittent it generally occurred in patients with blood glucose problems. Thus it may be the case that aberrant blood glucose contributes directly to an inability to sustain the brain metabolic activity required by neurofeedback or HEG training. Alternately it may be the case that training has caused a shift of the targeted physiology (or some related measure) from being deficient to excessive. If such is the case, then the risk factors

may mediate the tendency to respond rapidly or to overrespond in a brittle manner.

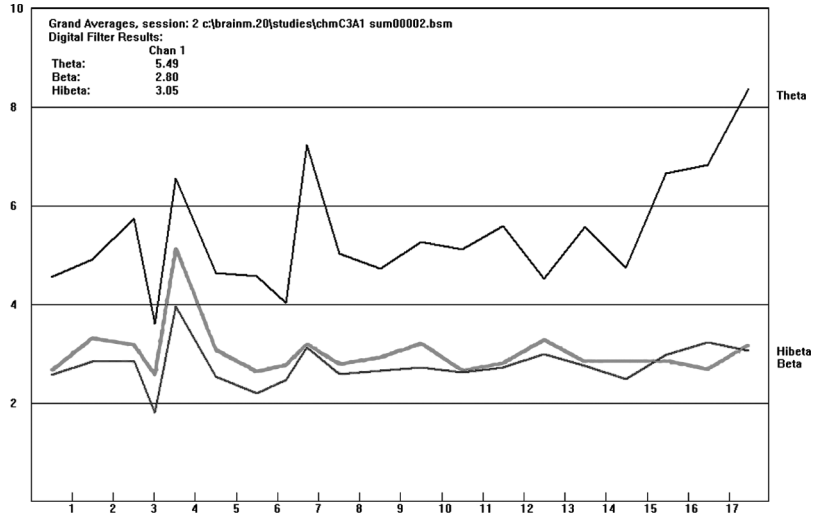
NEUROFEEDBACK TASK FATIGUE

Close attention to within-session treatment response early in the treatment course is a useful way to avoid overtraining. By tracking task performance results during training, one can identify early fatigue on the specific treatment task. Fatigue shows up as a shift to failure at the specific task. For example, when required to reduce excess slow or fast activity in EEG neurofeedback training, the client initially succeeds, but progress then slows and their performance may then gradually become worse, with microvolt levels deteriorating compared with the levels at the beginning of training.

Vulnerability to overtraining has been found to vary in degree. Some patients tolerate only a very few 2- to 3-min training periods in an hour-long treatment session, with 5- to 10-min breaks between periods during which they can rest or we talk. What may be even more remarkable, these patients often show good treatment response to this small amount of training. In contrast, patients with lower risk factors show proportionately more tolerance for training and an increase in tolerance as treatment progresses. With vulnerable patients, the time remaining in a session between periods or after the cessation of neurofeedback may or may not be devoted to supportive counseling. However, given the severe brain-based conditions that we commonly see responding to treatment in our clinic, it is difficult to attribute treatment success to mere counseling, particularly where prior counseling has already failed to produce improvements. Despite their sensitive nature, these patients have been found to show the same kind of location-specific positive response to briefer duration training that we observe with hardier patients undergoing neurofeedback treatment. Although they may require more sessions, they seem to show more benefit from each session than would be expected based strictly on the minutes of training. That is, it seems that there is a shift in the usual dose–response relationship.

Figure 1 displays the results of the second session with a young man who entered neurofeedback treatment following a year of inpatient and outpatient therapies for his severe head injury. He had postconcussive diabetes during his hospital course, which later resolved, and a family member with diabetes. Premorbid IQ was average. To avoid overtraining we used brief training periods with several-minute rest breaks

FIGURE 1. Illustration of task fatigue.



between periods, and we stopped training in this session after 18 min. Neurofeedback training consisted of seven periods: 0–1, 1–3, 3–6, 6–9, 9–12, 12–15, and 15–18 min. Theta decreased (improved) in the second, third, and fourth periods, maintained in fifth and sixth, and then in the last period theta increased to above the initial level, showing task-fatigue. The peaks at the fifth and ninth data points include artifact related to resuming the task.

The client in Figure 1 displayed only a moderate level of risk factors. In subsequent sessions, he showed treatment tolerance that gradually increased across sessions as we slowly incremented task demands in various ways, carefully tracking his tolerance and avoiding overtraining. Generally task demand may be gradually increased by lengthening training periods, with shorter pauses between periods, eliminating off-task activity during pauses in training (such as conversation or even brief naps), and other factors such as adding more complex reinforcement.

It may be useful to comment that the phenomenon described here as task-fatigue is apparently distinct from the usual fatigue and drowsiness commonly observable as an early response in training, particularly when a patient has insomnia, and from the profound relaxation observed in other patients specifically related to the type of training protocol. In those situations the patient gets drowsy but recovers if roused. In contrast, a patient showing task fatigue will progressively decline in task performance regardless of being coached or provided with a stimulating secondary activity, such as squeezing a stress ball—interventions that may work well with a drowsy client.

Patients who experience declines because of overtraining should be shifted to another, dissimilar protocol or away from training entirely for a few days or even a couple of weeks until the adverse response remits. It may be necessary to reverse the training effect with an appropriate contrasting protocol, but this probably will not be well tolerated in the same session at the same scalp location. It is recommended that during any hiatus from treatment, the therapist remains in close contact with the patient because the transient negative effect may frighten the patient. Such patients may be expected to display overtraining reactions to other protocols as well. Therefore, proceed cautiously and observe treatment response in the sessions.

CONCLUSIONS

The transient negative reactions from overtraining can be quite disconcerting to the client and to the unsuspecting clinician. There may initially have been a good response to training, followed after a session or two with adverse response to precisely the same protocol when the client's tolerance has been exceeded. The resulting loss of trust can be profound and lead to either aborted treatment efforts or dropout from treatment. Just knowing that overtraining may occur can bolster the clinician to explain such potential effects to vulnerable clients who are deemed to be at risk, and to then search for and remedy any overtraining reactions, preventing premature termination. Given the dramatic distress overtraining may produce, it may account for a large percentage of treatment failures and dropouts where this vulnerability is overlooked. Particularly in clinics where the clientele have significant medical and brain-injury conditions, it is important to screen for and adapt to these needs.

NOTE

1. Peter Janssen, MD, provided an explanation of the physiology of overtraining specific to athletic performance. Naturally the mechanisms including lactate acidosis are probably not relevant to neurofeedback overtraining. Further, no effects lasting over a few days have been observed with neurofeedback in contrast to those with severe athletic overtraining.

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