Anxiety, Anger, Depression, TBI and HEG

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Abstract:

This study revealed two candidate pathways from cortical receipt of a stimulus to activity of the amygdala. An excitatory pathway was found through the orbito-frontal cortex. Another emotional regulator pathway was found via the right inferior lateral prefrontal cortex.

The relative speeds and strengths of these pathways were expected to determine the anxiety and overly emotional responsiveness of anxiety, PTSD, depressive, and anger prone individuals.

It was hypothesized that HEG exercise of the regulatory and inhibitory pathways would strengthen them and bring overly emotional sufferers to remission.

One of two TBI Cases is reported here. Four examples of nine resolved anxiety cases with before and after QEEG or Loreta studies show EEG effects of HEG frontal cortex training

Introduction:

Anxiety, resulting from amygdala activation, has evolutionary advantages in rapidly preparing the individual for flight or fight in threatening situations. A brain exceptionally fast excitatory pathway has been found from the striatum that reaches the amygdala via the special branch of Broca. Another inhibitory pathway traverses the cortex before reaching the amygdala and is slower. This second pathway is inhibitory. A third regulator pathway in the right lateral inferior prefrontal cortex has been shown to regulate activation of the anterior cingulate cortex, a common route for pain and emotional activation. (Eisenberger N. et al. 2003). These pathways are illustrated in **Fig 1**.



Fig 1 Adapted from Baxter, L., et al, Lichter and Cummings (eds.) *Frontal-Subcortical Circuits in Psychiatric and Neurological Disorders*

The normal expectation is that cortical negation activity can subdue anxiety, fear, or negative emotional response. This experimental study exercises the inhibitory pathway. testing whether strengthening the cognitive response with HEG exercise can make it fast and strong enough to limit the norepinephrine release that activates direct uncontrolled amigdalar response.

Eisenberger et al. (Ibid), report brain areas that activate feelings of hurt and fear the anterior cingulate (ACC) and another near AF8 that inhibits this response. These areas, ACC and the right ventro-lateral prefrontal cortex, are shown to be in a relative strength relationship as diagrammed below in control of negative emotions such as anxiety and fear. Discovery of several such relationships (Davidson et al. 2000) raises the questions of where and how many agonist/antagonist pathways can be found in the brain and can their relative strengths be modified by suitable brain exercise.



Fig 2 Striatum from Brain Coloring Book Diamond M. and Scheibel A.

Some individuals are more prone to anxiety attacks than the average. Zubieta et al. (2003) studied disparity in pain tolerance. They found a gene, ComptVAL^{158met}, that regulates pain sensitivity. The gene has two alleles, methionine (met) and valine (val). Each allele is a dominant gene with equal opportunity for inheritance. One inherits a copy from each parent. Thus there are four genotypes: met-met (1), met-val (2a), val-met (2b), and val-val (3). The 2a and 2b genes have identical behavioral effects. There are thus three separable genotypes or grades of pain sensitivity 1,2, and 3. Type 1 and 3 each account for 25% of the population. Genotypes 2a and 2b account for 50% of the population. There are thus a low sensitivity (1) group, a high sensitivity (3) group and an intermediate sensitivity (2) group.

Aron E. in "The Highly Sensitive Person" (2000) provides a test to distinguish the high sensitivity group 3 members.

This is the first report using HEG to affect a mental state usually relegated to EEG or psychotherapy. This experimental result with HEG, a system that has demonstrated its physiological basis (Toomim, H. 2002), suggests that an underlying genetic or acquired physiological complex makes one prone to these devastating experiences. As Richard Davidson says: "Even though we all experience similar emotions, we respond to them in different ways" [Wisconsin University Communications News Release 2/19/2004; Zubieta et al. 2003 Science Feb. 21(299) 1240-1241; Toomim H. Neurofeedback with Hemoencephalography (HEG) Explore for the Professional 4(1) Nov. 2002].

Hypothesis:

The speed and strength of right and left lateral prefrontal cortical emotion regulators' of the orbitofrontal cortex can be developed to control the response of victims of anxiety and negative emotional experience.

Method:

Nine resolved Anxiety and a resolved traumatic brain injury (TBI) Case Reports with before and after QEEG and Loreta studies were completed. Here a sample of four of these Anxiety and the TBI cases are presented. They were trained with Hemoencephalography (HEG) at Fp1 and Fp2 or F8 to remission of anxiety or emotional TBI symptoms. Training with HEG was to increase the 10 minute average of one of the ratios: Fp1, Fp2 or F8 to AFz for 12 to 30 sessions.

Measurement with a dual HEG headband made rapid simultaneous recording of these responses convenient. The response to hyperventilation was clearly shown by the ratios of Fp1 to F7 or Fp2 to F8, the hypothesized controlling responses. The magnitude and timing of these responses were examined pixel by pixel of the saved recording.

Before and after QEEG or Loreta scans were obtained for each participant.

Case 1

65 year old male with a 40 year history of anxiety that included:

- 1. Profound sleep disturbance (unrelenting mental activity when he closed his eyes)
- 2. Severe exema
- 3. Heart palpitations
- 4. Shaky voice and stuttering
- 5. Mental Agitation
- 6.Ruminations

7.Multiple therapeutic interventions in past years including: Prozac, Effexor, Celexa, Zoloft, Xanax, Buspar, Ambien, Halcion.

All medications exacerbated his symptoms.

In addition to anxiety he was severely dyslexic and believed he was probably ADHD as a child. He received 12 HEG sessions

Note this was not global treatment!

Case 2

56 old caucasian female, Married 39 yrs. No children History:

- 1. Panic attacks since 1978
- 2. Multiple outpatient interventions through the years with several different anti-anxiety agents
- 3. Several attempts at outpatient psychotherapy
- 4. History includes dissociative experiences (diagnosed as MPD in mid 80's).
- 5. Restlessness and agitation
- 6. Shortness of breath
- 7. Palpitations
- 8 "feeling pressured to get things done"
- 9. Dissociative feelings.

Currently taking Prefaced for stomach problems but no other medications.

Pretreatment QEEG showed greatest signal at f8. Patient underwent 14 HEG treatments with optodes at F7

Case 3

22 Year old caucasian female.
Married; no children;
4th Year of college studying marketing.
Anxiety symptoms began during freshman year of college.
Symptoms included:

- 1. Shortness of breath
- 2. Dizziness
- 3. Insomnia
- 4. Chest pains

Medications included: Effexor, Paxil, Celexa.

She experienced short term relief from each but symptoms quickly reappeared and all medications were discontinued approx. 3 months prior to her presenting for treatment. She Trained every other day for 24 Sessions. Sessions were 30 minutes each, divided into 3 ten-minute segments. Alternated treatment sites daily from Fp1 to Fp2.

Case 4

18 Year old white male History:

- 1. Insomnia
- 2. Tics
- 3. Ruminations
- 4. Difficulty sleeping.
- 5. LD (math, reading).

Senior in high school symptoms worsened at times of increased academic demands.

Case 5 (TBI)

A 48-year-old Caucasian male suffered a traumatic brain injury in performance of his profession as a marine engineer. He was released from hospital at the end of the day of injury with diagnosis of "Moderate Concussion"

For two years following injury he suffered:

- 1. Episodes of recurrent dizziness
- 2. Impaired working memory
- 3. Inability to remember task sequences
- 4. Unable to read mail or follow customer assignments
- 5. Profound depression
- 6. Insomnia
- 7. Inability to work

Twenty-two HEG targeted treatments at Fp1 and F7 designed to improve working memory were implemented.

Three HEG treatments at F8 were then applied following publication of Eisenberger et al. (ibid)

Results:

Case 1

His post-treatment Q showed global changes.

Following treatment he was sleeping approximately 6 hrs. per night with no difficulty falling asleep. He was less agitated, had less vocal tremor, and his eczema was dramatically improved. At 6 months follow-up the improvements were maintained and he was medication free.

Case 2

Post-treatment Q topogram showed major increase at the treatment site F7 matching the pretreatment value at F8. Following treatment at 6 months follow up she reported falling asleep more easily, no further dissociative experiences and was "very productive without feeling pressured". She also had left her husband and decided to go back to school in the health care field.

Case 3

Following treatment at 6 months follow up this young woman was reporting relief from all of her symptoms. At 6 months follow-up she had maintained all gains and said "I really never even think about having gone through all of that."

Case 4

Following treatment at 6 months follow up, he reported he was sleeping well, was free from ruminative thinking and he was most pleased because his tics were nearly totally eliminated.

Case 5 (TBI)

Significant incremental improvement of working memory followed the initial course of HEG treatments. He became able to read again and his depression was somewhat improved.



F8 targeted sessions were begun in November '03 following the first presentation of this report at the ISNR meeting. A dual HEG headband initially monitored Fp2 while training Fp1. After 22 sessions the dual HEG array was changed to monitor F8 while activating the AFz position.



During the first Afz training session a significant 3-minute 30% rise at F8 was recorded. At the end of the 10-minute segment, asked to describe the significant rise "I felt like crying" was his response.

Thus was confirmed the use of the F8 position for treatment of Eisenberger's 'emotion regulator'. Subsequent HEG sessions targeted this position. During the third HEG F8 targeted session he suddenly stopped the session with the recognition: "I feel like I used to. My brain is working again!"

Conclusion:

Pre-frontal treatment with HEG may have great utility in treating anxiety. Anxiety can be associated with both 'high beta' and 'high alpha' states.

HEG training at the lateral prefrontal locations on the hypothesized pathways resulted, after 12 to 30 sessions, in significant symptom improvement in four samples of 9 similar cases.

These results support Targeted Location theory and the role of the amygdale in anxiety/panic Further support is provided that the targeted pathway to the amygdale can be influenced with pre-frontal HEG training.

This is a preliminary test that opens a possible pathway for analysis and training of florid emotional (anxiety, PTSD, anger, and TBI) cases toward more controlled responses. Although the hypothesized results happened according to prediction, so much is still unknown about brain responses to stimuli that these results must be approached with caution. The interconnectivity of neural populations is so large that almost any brain area can be expected to make a contribution to path length and effective time of action.

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