The Value of a qEEG Assessment, Especially Looking at Theta Unity

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After six unsuccessful treatments with five doctors, a 44-year-old male patient sat in the office desperate for a solution to his insomnia. Two and a half years before, he had moved into a new home and his sleep difficulties began. He found it difficult to fall to sleep, and at times even to feel tired, and when he finally would fall sleep he would wake up an hour later only to repeat the same cycle through the night. At most, his broken sleep would aggregate to a mere four hours.

He first sought treatment at a sleep recovery center where he received neurofeedback that rewarded the stability of his EEG. His sleep difficulties showed slight improvement, but his progress stagnated early on and the sleep center suggested he obtain a qEEG to better understand why his sleep was resistant to improvement. The qEEG results indicated his delta activity was insufficient, and his neurotherapist suggested they increase this frequency. The results were disastrous; the patient reported having suicidal ideation for the first time in his life and he was unable to sleep for four days. Becoming increasingly worried, the patient sought out another neurotherapist who used milder Z-score training. Although the patient reported his sleep returned to four hours per night, it began to feel like he was "conscious during sleep" and he could hear his "brain running." He then sought pharmaceutical help and was prescribed Ambien, Celexa, and Gabapentin. Unfortunately, the medications had a barely noticeable impact on his sleep, and the side effects were not worth continuing their use.

It was after this first bout of unsuccessful treatments that the patient came to our office for help. We took the data from his original EEG recording and analyzed it using SKIL software to evaluate the limbic and cortical contribution for 55 brain areas (Brodmann areas).

In the past, our analysis has been useful for evaluating the integrity of each brain area and the cortical networks involved: default-mode, dorsal attention, executive control, salience, and sensory networks. We have developed a handful of promising neuromarkers strongly associated with symptoms, behaviors, or life histories. For instance, an impairment in socioemotional awareness, a deficit found in 11 of 12 death row inmates, is associated with a disconnection of Brodmann area 44R (Kaiser et al., 2012). Childhood abuse commonly results in left or right auditory cortices being disconnected (Kaiser & Meckley, 2012). But in our case, we found a disconnected posterior cingulate, which we observed in all of our trauma cases to date. More than 30 studies have identified posterior cingulate problems in PTSD patients, and our data is consistent with this research. As a result, we feel comfortable calling a theta disunity of the posterior cingulate a neuromarker for trauma.

Our analysis of the patient's EEG revealed this trauma marker—his posterior cingulate cortex (PCC) exhibited what we call a corticolimbic dysrhythmia. Theta activity in the PCC was out of synch with theta activity in the adjacent cortex, suggesting another origin for its activity in the PCC. Given that the PCC anchors the default network (see text box, following), this deviation in shared activity indicates diminished functional integrity of the default mode network. This conclusion explains why the many sleep treatments he tried were unsuccessful—they were not treating the source of his sleep disruption, a dysfunction in his default mode network which made it difficult for him to transition from an awake to sleep state. Activity in the default-mode network must increase for sleep onset to occur (Picchioni et al., 2008).

In his intake, the patient denied he had experienced any trauma in his life. However, when we presented the findings to him, he revealed he had indeed suffered through three significant traumas in his life: emotional abuse by his mother throughout his childhood, the sudden death of his older brother in a car accident when the patient was 17, and the suicide of his fiancé when he was 21. Nevertheless, he insisted that he was "over these traumas" and left our office refusing to believe they had any relevance to his sleep problems.

Five months and two more unsuccessful medical treatments later, the patient called our office and was ready to consider that our analysis was relevant. He agreed to try an integrated trauma treatment that consisted of alpha-theta training combined with psychological trauma techniques, such as somatic experiencing, and a specialized mindfulness training that targets the dissociation often seen in trauma.

After 20 sessions of alpha-theta training at site Pz, directly over the PCC, the patient's sleep normalized. He became drowsy at night, was able to fall asleep within 20 minutes, and he was able to sleep for 6–7 hours without interruption. The patient was happy with his improvement and we agreed to terminate treatment. It is now three months after termination, and the patient still reports that his sleep difficulties did not recur. Without the EEG assessment, we did not know the patient's problems very well, nor did the previous practitioners who treated the symptom (sleep disruption) and not the mechanism (trauma).

ONLINE SUPPLEMENT

References are available in the supplement at: http://isnr.org/neurofeedback-info/neuroconnections-newsletters.cfm.