Evidence for the Effectiveness of NeuroField in a Three-Year-Old Boy with Insomnia

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Abstract

The objective of this study is to investigate the effect of NeuroField real-time Z-score training and low-intensity pulsed electromagnetic field (pEMF) stimulation on a three-year-old child diagnosed with Stickler Syndrome, Pierre Robin Sequence, and insomnia. Based upon the child's history, a NeuroField training plan was implemented to target the parents' primary complaint of insomnia. The boy had a total of 27 NeuroField training sessions. A pre-training, follow-up-training, and post-training Quantitative Electroencephalography (qEEG) was analyzed. Parent report of sleep and behavior was also recorded. The results show significant differences between the pre-test, follow-up, and post-test qEEG data. Symptoms of insomnia, restless sleep, and behavior problems improved over the course of the study. The data analysis also concluded that qEEG patterns showed continued improvements beyond the cessation of training.

Case Report

The subject is a three-year-old male who had been diagnosed by medical professionals with Stickler¹ syndrome and Pierre Robin Sequence². Prior to birth, the young child was diagnosed with intrauterine growth restriction (IUGR). At birth, the child required multiple resuscitations and suffered respiratory arrest, resulting in anoxia. He was placed on extracorporeal membrane oxygenation (ECMO). After stabilization, the child suffered from severe sleep apnea and wore an apnea monitor for several months. During the next three years of life, the child had undergone anesthesia for six major corrective surgeries.

At the age of three, the toddler's parents brought their child in for neurotherapy consultation. In desperation, the young parents explained that their toddler does not sleep. Per parent report, the child would not take naps and took four hours or more to fall asleep. During sleep, the parents reported that the toddler is exory, learning, attention, visuoperceptual and visuoconstructional abilities, generalized intellectual impairment, behavioral problems, and difficulties in executive functions. (Allen, Tranel, Bruss, & Damasio, 2006; Caine & Watson, 2000; Garcia-Molina et al., 2006; Lim et al., 2004; Myers et al., 2008; Pierro et al., 2005). No specific pattern emerges as a result of an anoxic event. Impairment observed in anoxic cases depends on a magnitude of factors, such as the nature and duration

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tremely restless. The child wakes in the mornings irritable and low in energy. The toddler presented as hyper-vigilant, hyperkinetic, and anxious. He also is small for his age group and has poor coordination and balance. The child has no history of seizure, and his MRI was within normal limits, though anoxia was noted. He regularly sees a pediatric physician and a nutritional therapist. He received most of his nutrition through a gastric tube.

Literature Review

Cellular injury occurs during an anoxic event. Deprivation of oxygen and metabolic substrates such as glucose causes a loss of energy and oxidative stress (Abramov, Scorziello, & Duchen, 2007). Anoxia is correlated with deficits in memof the anoxic event as well as the regions associated with neuronal degeneration (Bachevalier & Meuiner, 1996; Wilson, 1996; Wilson, Harpur, Watson, & Morrow, 2003). Abnormal EEG has been documented in several cases of anoxia, particularly, background slowing correlated with an encephalitic state (Thaler, Reger, Ringdahl, Mayfield, Goldstein, & Allen, 2012).

A growing body of recent research has suggested concerns that anesthetic agents may pose a risk in vulnerable brains of the very young (Zuccherelli, 2010). The current research is preliminary, but concern in the medical field has warranted the foundation of the Anesthesia Patient Safety Foundation to discuss long-term effects of surgery and anes-

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^{1.}Stickler syndrome is a

group of hereditary conditions characterized by a distinctive facial appearance,

eye abnormalities, hearing loss, and joint problems. These signs and symptoms vary widely among affected individuals.

^{2.} Pierre Robin syndrome (or sequence) is a condition present at birth, in which the infant has a smaller-than-normal lower jaw, a tongue that falls back in the throat, and difficulty breathing.

thesia (Zuccherelli, 2010). Research on human brain development and anesthesia has suggested that children between birth and two to three years of age are at risk for developing anesthetic neurotoxicity (Sun, 2010). Anesthesia has also been studied for its effects on circadian rhythms. The research has shown that general anesthesia can alter circadian rhythms on the days following surgery (Dispersyn, Pain, Challet, Touitou, 2008).

Neurofeedback has long been studied for its effects on insomnia and other sleep disorders (Hammond, 2007; Hauri, 1981, Hauri, 1982). Cortoos et al. (2010) produced a study that demonstrated that SMR neurofeedback was superior to other forms of biofeedback in improving sleep behavior. Several studies have shown that patients with insomnia exhibit elevated levels of beta EEG activity (Merica, Blois & Gaillard, 1998; Perlis, Smith, Andrews, Orff & Giles, 2001).

Low frequency repetitive transcranial magnetic stimulation (rTMS) has been shown to decrease intracortical excitability and have inhibitory properties (Romero, Anschel, Sparing, Gangitano, & Pascual-Leone, 2002). These findings suggest that magnetic stimulation has implications for therapeutic use. NeuroField is a type of neurofeedback device that incorporates the use of low intensity pEMF stimulation. The NeuroField pEMF ranges from 1–50 microtesla, which is 10,000,000 times weaker than the stimulation generated by rTMS (Dogris, 2011).

Only a few studies which utilized NeuroField have been published. Publications that are available have studied the effects of NeuroField on Parkinson's disease, premenstrual dysphoric disorder, attention deficit disorder, anxiety, conduct disorder, and post-traumatic stress disorder (Dogris, 2011; Dogris, 2012). In addition to the available research, NeuroField has been rated by licensed practitioners to be clinically valuable. The effect of NeuroField pEMF appears to be very similar to the effects of rTMS and has been documented to have long-term
 Table 1: NeuroField training outline for three-year-old boy with insomnia.

| Week | Participant Received | NeuroField | Electrode | Coil Placement |
|--------|---|-------------|-----------|----------------------------------|
| Number | runcipartenecentea | Protocol | Placement | confideement |
| 0 | Pre-training qEEG | | | |
| 1 | 5 NeuroField RTZ sessions | 0.31–1Hz HD | C3, C4 | C3, C4, P3, P4 F3, F4, C3, C4 |
| 2 | 2 NeuroField RTZ sessions | 0.31–1Hz HD | C3, C4 | C3, C4, P3, P4 C3, C4, Cz |
| 3 | 2 NeuroField RTZ sessions | 0.31–1Hz HD | C3, C4 | |
| 4 | No training | | | |
| 5 | 2 NeuroField RTZ sessions | 0.31–1Hz HD | C3, C4 | F3, F4, P3, P4 |
| 6 | 1 NeuroField RTZ session | 0.31–1Hz HD | C3, C4 | C3, C4, P3, P4 |
| 7 | 3 NeuroField RTZ sessions | 0.31–1Hz HD | C3, C4 | C3, C4, Cz, Pz Fz, C3, C4, Pz |
| 8 | 3 NeuroField RTZ sessions | 0.31–1Hz HD | C3, C4 | Fz, C3, C4, Pz |
| 9 | 2 NeuroField RTZ sessions | 0.31–1Hz HD | C3, C4 | Fz, C3, C4, Pz C3, C4, Pz |
| 10 | Follow-up qEEG, 2 NeuroField RTZ sessions | 9–12Hz HD | P3, P4 | P3, P4 |
| 11 | 2 NeuroField RTZ sessions | 9–12Hz HD | P3, P4 | P3, P4 |
| 12 | 2 NeuroField RTZ sessions | 9–12Hz HD | P3, P4 | Fz, P3, P4, Pz |
| 13 | 1 NeuroField RTZ session | 9–12Hz HD | P3, P4 | Fz, P3, P4, Pz |
| 14–18 | No training | | | |
| 19 | Post-training qEEG | | | |

effects (Dogris, 2011; Dogris, 2012). Based upon the clinical and statistical findings, further investigation of Neuro-Field and its implications are warranted. The current study investigates the effect of NeuroField real-time Z-score training and low-intensity pEMF stimulation in the case of a three-year-old child.

Based upon the subject's history and the available research, the following hypotheses were constructed: 1) Low-intensity, low-frequency pEMF will have an inhibitory effect on beta and high beta activity. 2) Regulation of beta and high beta activity can relieve symptoms of insomnia and restless sleep. 3) Low-intensity pEMF stimulation has a lasting effect on EEG. The null hypothesis in this study is that: 1) NeuroField pEMF will not reduce high beta, 2) there will be no changes in the infant's sleep patterns, and 3) 30-day follow up will show no changes in the EEG or the infant's sleep patterns.

Instrumentation and Methods

Quantitative EEG

Three qEEGs were acquired on three separate dates. A pre-training qEEG was recorded prior to the start of NeuroField sessions. A follow-up qEEG was recorded after receiving twenty low-intensity pEMF training sessions using the NeuroField X2000 and NeuroField Plus system. A third post-training qEEG was recorded six weeks after the last NeuroField pEMF training session. Each 19-channel qEEG was recorded using an Electro-Cap (Electro-Cap International, Inc). EEG acquisition was collected using a BrainMaster Discovery 24E amplifier. All qEEG recordings were acquired in the eyes open

condition, because of the age of the participant. All reference and cap electrodes were confirmed to have 5–10K Ohms resistance values using a Checktrode [®], model 1089NP impedance monitor. Impedance was measured to ensure the integrity of the data. QEEG data was analyzed using the NeuroGuide referenced normative database. Data was analyzed in both linked ear and Laplacian montages.

NeuroField

This study utilized the NeuroField X2000 with NeuroField Plus and 200 wound coil system. This system is a four-chan-

of EEG or heart rate variability.

The NeuroField system connects to a cable which is attached to four 200 wound coils. The 200 wound coils adhere to the NeuroField cap, which is worn on the cranium. The coils are then placed on the cap to target specific training areas determined by qEEG and presenting symptoms. Each of the four 200 wound coils creates an EMF intensity of 1–50 microtesla (or 1–500 milligauss).

Study Procedure

NeuroField sessions began shortly after the analysis of the baseline qEEG. Each

NeuroField RTZ is a feedback system which can be used to instantaneously measure the effect of each NeuroField pulse on brain activity.

nel frequency generator that is capable of generating low intensity pEMF from 0.31 to 300,000 Hz. The X2000 model has the ability to measure a participant's physiological response to each low-intensity pEMF stimulation through two channels session utilized NeuroField low-intensity pEMF. During this study, low-intensity pEMF was generated at 5V DC for 5,000– 10,000 milliseconds. NeuroField realtime Z-score thresholding (RTZ) was used throughout the course of training.



NeuroField RTZ is a feedback system which can be used to instantaneously measure the effect of each NeuroField pulse on brain activity. NeuroField RTZ measures and analyzes 4–32 seconds of EEG activity after each low-intensity EMF pulse. The EEG activity is then instantaneously analyzed using the NeuroGuide Z-score database. NeuroField RTZ repeats frequencies which meet the set training parameters which are guided by the results of the qEEG and presenting symptoms. The RTZ function can be set to threshold on a single or multiple frequency bands.

For the purpose of this study, the RTZ function was set to repeat frequencies which reduced high beta activity within ± 1 standard deviation. NeuroField pEMF with RTZ thresholding was given

for 40–60 minutes each session. The three-year-old participant had a total of 27 NeuroField RTZ training sessions. Table 1 illustrates the course of Neuro-Field training sessions, qEEG recordings and analysis, NeuroField protocol, electrode placement, and coil placement used throughout the duration of training. All training sessions and qEEGs occurred in the eyes open condition.

Results

The results of the pre-training, 20-session follow-up training, and six weeks' post-training qEEG were analyzed; summary Z-score analyses are presented in Figures 1, 2, and 3. Statistical Analysis was completed using the NeuroGuide normative database and NeuroGuide statistical software. Three one-way analysis of variance (ANOVA) comparisons were run on the qEEG data; pre-test vs. posttest, pre-test vs. follow-up and post-test vs. follow-up. All conditions showed significant changes in all EEG frequency ranges (p < 0.0001). The high beta range showed significant reductions in absolute power, hyper-coherence and phase in all conditions (p < 0.0001). (A comprehensive summary of results generated by the NeuroGuide statistical program is available upon request from the authors).

Parent report of average sleep latency and duration per night were recorded weekly. The results of this data indicated that the toddler required less time to fall asleep (Figure 4) and begun sleeping through the night (Figure 5). The parents reported that nighttime restlessness decreased. The child began taking rou-

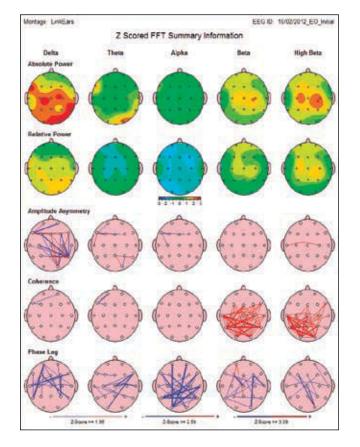


Figure 1: Summary Z-score Analysis of Pre-NeuroField training qEEG.

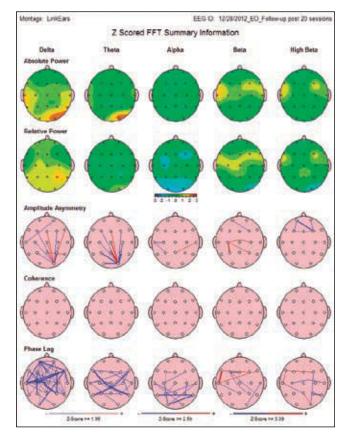


Figure 2: Summary Z-score Analysis of Follow-up qEEG, recorded after 20 NeuroField RTZ sessions.

tine naps by week three. Throughout the training, the parents also reported that the child had more energy, increased focus, and less anxiety.

This case clearly demonstrates neuroplasticity and adaptability of the brain. As the brain became more regulated, and excess high beta activity became more tory mechanism for the excess high beta. Whatever the root cause may have been, the NeuroField training plan significantly changed all EEG frequency ranges.

Discussion

The purpose of this study was to examine the effect of the NeuroField RTZ pEMF procedure on an infant with Stickler Syndrome, Pierre Robin Sequence, and insomnia. The results of this study support the hypothesis that pEMF training is able to reduce high beta and normalize sleep patterns. Furthermore, the data shows that high beta reductions continued after training was discontinued from follow-up to post-test qEEG. This observation supports the notion that the brain is able to continue regulating itself after the pEMF correction was implemented.

This case clearly demonstrates neuroplasticity and adaptability of the brain. As the brain became more regulated, and excess high beta activity became more normalized, other EEG activity also became more normalized.

normalized, other EEG activity also became more normalized. Delta training was not individually targeted in this case. The normalization of delta was a surprising clinical finding. Excess delta may have been present in the brain due to a multitude of reasons, including: sleep deprivation, history of anoxia, or as a compensaIt appears that NeuroField pEMF training has clinical value which warrants more research and study. Future research should focus on large subject samples with control groups and placebo/sham/ blind conditions. It would also be helpful to study each of the individual training programs that are available with the Neu-

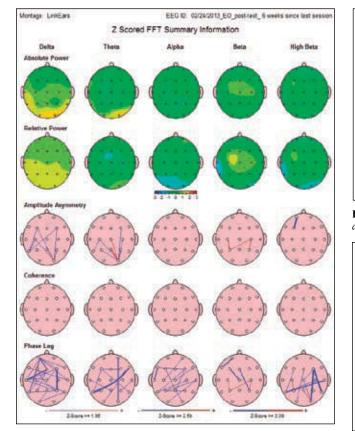


Figure 3: Summary Z-score analysis of post-NeuroField training qEEG. Post-qEEG was recorded after 27 NeuroField RTZ sessions.



Figure 4: The average amount of time (hours) the three-year-old child took to fall asleep over the course of NeuroField training.

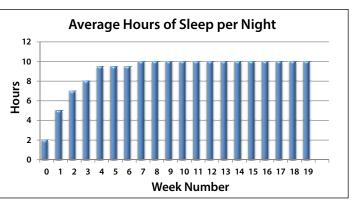


Figure 5: The average amount of time (hours) the three-year-old child slept per night over the course of NeuroField training.

roField software.

This case study offers support for the idea that early intervention is very important. Neurotherapy does work for very young children. Sleep loss can impair performance, cognition, and have other health and behavioral implications. A child's sleep patterns may also affect other members of the family. Unfortunately, clinicians, researchers, and even parents often ignore sleep disorders in children. This case study demonstrates that when you change a brain, you change a life. And when you change a child's life, you

A note from the parents: "Before receiving training at Integrated Neurotherapy, our son typically fell asleep four hours after being put to bed for the night, despite going without a nap during the day. He would wake up in the middle of the night and have trouble falling back to sleep by himself. Since being seen at Integrated Neurotherapy, our son now falls asleep routinely within 30 minutes. Moreover, he sleeps longer and rarely wakes up in the middle of the night. The improvements in our son's sleeping habits have allowed our whole family to sleep and function better! Our son's prior insomnia had affected our family's health, work efficiency, and social life. We are thankful for the resolution of our son's insomnia, as it has restored our lives to a more normal and positive routine.

About the Authors

Jamie Moore, RN, BCN, has been a Registered Nurse for over 33 years. He graduated from Saint Joseph's School of Nursing in 1979, and has continued to study the brain and Autism Spectrum Disorder ever since. Jamie has studied under Leslie Sherlin, and completed training by John Anderson, a pioneer in the field of neurofeedback. He completed his internship/mentoring under Nicholas Dogris, inventor of the NeuroField system. Jamie plans to continue his education in neurotherapy and nursing, and to carry on practicing from his heart. Erica Kube, BS, is a graduate from the program of neuroscience and biology at the University of Nebraska, Omaha, where she received recognition for her achievement and outstanding performance. She has authored award-winning work and practices neurotherapy full-time.

Erica has completed extensive training with experts in the field of neurotherapy including John Anderson and Nicholas Dogris. She is currently finishing a Master's of Science in Clinical Counseling. This advanced training and passion for learning the latest approaches have increased the efficiency and effectiveness of neurotherapy, from which her clients achieve maximum health and quality of life.

Disclaimer: The authors of this study have no affiliation with any of the brands or businesses mentioned in this paper. The participant and parents were not compensated for their participation in this study.

References

- Abramov, A. Y., Scorziello, A., & Duchen, M. R. (2007). Three distinct mechanisms generate oxygen free radicals in neurons and contribute to cell death during anoxia and reoxygenation. *The Journal of Neuroscience*, 27(5), 1129–1138.
- Allen, J. S., Tranel, D., Bruss, J., & Damasio, H. (2006). Correlations between regional brain volumes and memory performance in anoxia. *Journal* of Clinical and Experimental Neuropsychology, 28, 457–476.
- Bachevalier, J., & Meuiner, M. (1996). Cerebral aschemia: Are the memory deficits associated with hippocampal cell loss? Hippocampus, 6, 553–560.
- Caine, D., & Watson, J. D. G. (2000). Neuropsychological and neuropathological sequelae of cerebral anoxia: A critical review. *Journal of the International Neuropsychological Society*, 6, 86–99.
- Dispersyn, G., Pain, L., Challet, E., & Touitou, Y. (2008). General anesthetics effects on circadian temporal structure: An update. *Chronobiology International*, 25(6), 835–850.
- Dogris, N.J. (2011). NeuroField: Three case studies. Journal of Neurotherapy, 15, 75–83.
- Dogris, N.J. (2012). The effect of NeuroField pulsed EMF on Parkinson's disease symptoms and qEEG. *Journal of Neurotherapy*, 16, 53–59.
- Garcia-Molina, A., Roig-Rovira, T., Ensenat-Cantallops, A., Sanchez-Carrion, R., Pico-Azanza, N., et al. (2006). Neuropsychological profiles of per-

sons with anoxic brain injury: Differences regarding physiopathological mechanism. Brain Injury, 20, 1139–1145.

- Hammond, D. C. (2007). What is neurofeedback? Journal of Neurotherapy, 10(4), 25–36.
- Hauri, P. (1981). Treating psychophysiologic insomnia disorder with biofeedback. Archives of General Psychiatry, 38(7), 752–758.
- Hauri, P. J., Percy, L., Hellekson, C., Hartmann, E., & Russ, D. (1982). The treatment of psychophysiologic insomnia disorder with biofeedback: A replication study. Biofeedback and Self Regulation, 7(2), 223–235.
- Lim, C., Alexander, M. P., LaFleche, G., Schnyer, D. M., & Verfaellie, M. (2004). The neurological and cardiac sequelae of cardiac arrest. *Neurobiology*, 69, 1774–1778.
- Merica, H., Blois, R., & Gaillard, J. (1998). Spectral characteristics of sleep EEG in chronic insomnia. *European Journal of Neuroscience*, 10(5), 1826– 1834.
- Myers, C. E., Hopkins, R. O., DeLuca, J., Wolansky, L. J., Gluck, M. A., Sumner, J. M., et al. (2008). Learning and generalization deficits in patients with memory impairments due to anterior communicating artery aneurysm or hypoxic brain injury. *Neuropsychology*, 22, 681–686.
- Perlis, M., Smith, M., Andrews, P., Orff, H., & Giles, D. (2001). Beta/gamma EEG activity in patients with primary and secondary insomnia and good sleeper controls. Sleep, 24(1), 110–117.
- Pierro, M.M., Bollea, L., Di Rosa, G., Gisondi, A., Cassarino, P., Giannarelli, P., et al. (2005). Anoxic brain injury following near-drowning in children. Rehabilitation outcome: Three case reports. Brain Injury, 19, 1147–1155.
- Romero, J. R., Anschel, D., Sparing, R., Gangitano, M., & Pascual-Leone, A. (2002). Subthreshold low frequency repetitive transcranial magnetic stimulation selectively decreases facilitation in the motor cortex. *Clinical Neurophysiology*, 113(1), 101–107.
- Sun, L. (2010). Early childhood general anaesthesia exposure and neurocognitive development. *British Journal of Anaesthesia*, 105, (suppl 1), i61–i68.
- Thaler, N.S., Reger, S.L., Ringdahl, E.N., Mayfield, J.W., Goldstein, G., & Allen, D.N. (2012). Neuropsychological profiles of six children with anoxic brain injury. *Child Neuropsychology*, 1–16.
- Wilson, B. (1996). Cognitive functioning of adult survivors of cerebral hypoxia. *Brain Injury*, 10, 863–874.
- Wilson, F., Harpur, J. J., Watson, T. T., & Morrow, J. I. (2003). Adult survivors of severe cerebral hypoxia: Case series survey and comparative analysis. *Neurorehabilitation*, 18(4), 291–298.
- Zuccherelli, L. "Long term effects of anaesthesia: neurotoxicity at the extremes of age." Southern African Journal of Anaesthesia and Analgesia 16.1 (2010): 70–74.