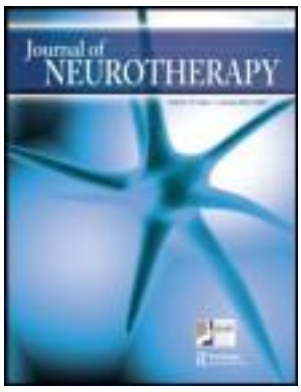

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Can LENS Neurofeedback Treat Anosmia Resulting from a Head Injury?

D. Corydon Hammond, PhD

ABSTRACT. Loss or reduction of olfactory acuity (anosmia) is a symptom associated with moderate to severe acceleration-deceleration traumatic brain injuries (TBI). Posttraumatic anosmia has been regarded as an irreversible symptom. It usually results in deficits in taste and is documented to negatively affect quality of life, safety, interpersonal relations, and nutritional intake. This paper presents the first two cases to be reported where there were spontaneous reports of the reversal of long-term anosmia following neurofeedback treatment with the Low Energy Neurofeedback System (LENS). These reports provide encouragement that neurofeedback may have potential to treat posttraumatic anosmia, as well as improving other TBI symptoms. Clinicians using neurofeedback to treat TBI are encouraged to evaluate patients for the presence of anosmia and to track possible improvements. doi:10.1300/J184v11n01_06

KEYWORDS. EEG biofeedback, neurofeedback, TBI, anosmia, LENS, Low Energy Neurofeedback System

INTRODUCTION

The olfactory nerve originates with the olfactory epithelium, which consists of about 5 million receptor cells on the roof and adjacent walls of the nasal cavities. The olfactory receptor neurons are small bipolar neurons with a slender dendrite coming from one end of its cell body and with an axon that emerges from the other end. Rather than being a brainstem nerve, the olfactory nerve is a complex pathway consisting of a collection of about 20 very fine axon bundles (olfactory fila) associated with the nasal nerve cells. This collection of olfactory fila compose the first cranial nerve which terminates in the olfactory bulb (Joseph, 1996; Nolte, 1999).

Uniquely, the olfactory nerve bypasses the thalamic relay station and connects directly with the ipsilateral hemisphere. At the posterior end of the orbital frontal cortex the olfactory tract attaches perpendicularly to the base of the brain, with the lateral olfactory tract moving up onto the surface of the temporal lobes where fibers then end in either the primary olfactory cortex or the amygdala. The receptor sites for olfactory data then project to the hypothalamus, limbic structures, hippocampus, and the thalamus which relays information to the olfactory association cortex (Nolte, 1999). Many people are unaware that much of our sense of taste is actually dependent on the aromas of our food. Thus someone who has lost their sense of smell from a head injury is likely to also complain of a decrease in their ability to taste.

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Perhaps interesting in light of the case about to be presented, the bipolar cells in the olfactory epithelium are composed of "very primitive cells that have a life span that lasts only days. New axons and new synapses are continually forming. These nerve fibers perforate and pass through the cribriform plate (making them very susceptible to injury and shearing . . . and project directly to the olfactory bulb" (Joseph, 1996, p. 379). Thus the receptor cells are being replaced throughout life. Joseph (1996) explained that "following a head injury the cribriform plate may fracture, the olfactory nerve may be severed, and the meninges may rupture . . . . However, in some cases, a head injury may cause these nerves to be severed although the cribriform plate remains intact. In these instances, loss of smell may result: anosmia" (p. 379). The process by which traumatic anosmia occurs was also summarized by Nolte (1999): "The olfactory fila may be torn loose from the olfactory bulb as a consequence of head trauma and be unable to regrow through a scarred cribiform plate, or olfactory areas at the base of the brain may be damaged by slight movement of the brain across the base of the skull" (p. 322).

Research with SPECT and PET neuroimaging (Eftekharie et al., 2006; Varney & Bushnell, 1998; Varney, Pinkston, & Wu, 2001) has documented that posttraumatic anosmia particularly corresponds with hypoperfusion in the orbital frontal cortex in a great number of patients. Olfactory problems have been found in 14%-27% of patients with moderate to severe head injuries (Costanzo & Becker, 1986).

Thus in a coup-contrecoup injury, particularly of moderate or greater severity, damage can be done to cranial nerve I as the brain moves within the anterior cranial fossa. This can result in either focal or diffuse injury in the orbitofrontal, and less frequently temporal areas. Posttraumatic anosmia can be either unilateral or bilateral, and it can also impair the ability to identify common odors after injury. Improvements have only been found in 36% of patients (Doty, Yousem, Pham, Kreshak, Geckle, & Lee, 1997), generally in the first 6-12 months (while 18% of cases worsen in this time period), and usually such injuries are regarded as permanent with no more than 10% of patients improving more than 2 years post-injury (Costanzo & Becker, 1986; Sumner, 1964). Posttraumatic anosmia has proven resistant to treatment with medication (Hirsch, Doughtery, Aranda, Vanderbilt, & Weclaw, 1996). These injuries may also impair olfactory naming and recognition (Levin, High, & Eisenberg, 1985), which is believed to impair learning in the non-dominant hemisphere that is associated with olfaction (Hirsch & Johnston, 1996).

BACKGROUND AND METHODOLOGY

Neurofeedback is an encouraging treatment that is being used to treat many problem areas (Hammond, 2006), including open and closed head injuries (e.g., Ayers, 1999; Hoffman, Stockdale, & Van Egren, 1996; Keller, 2001; Laibow, Stubblebine, Sandground, & Bounias, 2001; Thornton, 2000; Thornton & Carmody, 2005; Tinius & Tinius, 2001).

The Low Energy Neurofeedback System (Hammond, 2007; Larsen, 2006; Ochs, 2006) is a unique and passive form of neurofeedback which produces its effects through the introduction of a very tiny electromagnetic field which has a field strength of 10^{-18} watts/cm^2. This stimulation, which is far weaker than the input we receive from simply holding a cell phone to our ear, is delivered for one second at a time down electrode wires. The frequency of the electromagnetic stimulation is determined, moment-to-moment, by the dominant frequency of the EEG which is measured in hertz or cycles per second, and updated 16 times per second. The client sits eyes closed and the total time in which electromagnetic fields are received in a treatment session is usually only a few seconds at a small number of electrode sites on the head. This stimulation is believed to gently nudge the brain off of its stuck points, assisting it to become more efficient, flexible and self-regulating. Preliminary research and clinical experience have found that LENS rivals and in some cases surpasses more traditional forms of neurofeedback in the treatment of conditions such as traumatic brain injury (Schoenberger, Shiflett, Esdy, Ochs, & Matheis, 2001), fibromyalgia (Donaldson, Sella, & Mueller, 1998; Mueller, Donaldson, Nelson, & Layman, 2001), ADD/ADHD, depression, anger, and

other conditions (Larsen, 2006). LENS has even been used to treat animal behavior problems (Larsen, Larsen, Hammond, Sheppard, Ochs, Johnson, Adinaro, & Chapman, 2006).

This paper will report on two cases where patients were receiving treatment with LENS, but where the author was unaware that they had problems with posttraumatic anosmia. Both patients spontaneously reported improvements in their sense of smell after a relatively small number of LENS neurofeedback sessions.

CASE ONE

The patient was a 29-year-old man with a history post-concussion syndrome. He had experienced four mild head injuries which included two mild concussions associated with water skiing, and a whiplash in an automobile accident 6 years earlier. Nine and a half years prior to entering treatment he had experienced a moderate severity head injury when he was thrown from a four-wheeler in an accident in the mountains where a deer jumped across his path. He sustained a frontal lobe head injury and was unconscious for 10-15 minutes. He continued to go in and out of consciousness for some period of time afterwards. His period of loss of consciousness is congruent with a study (Swann, Bauza-Rodriguez, Currans, Riley, & Shukla, 2006) that found a significantly greater likelihood of patients with posttraumatic amnesia lasting for 5 or more minutes having posttraumatic anosmia, compared patients with amnesia for less than 5 minutes.

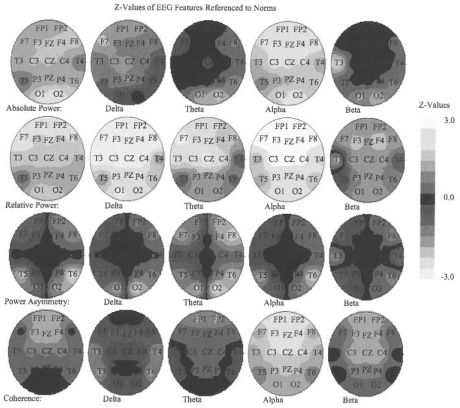
The patient was hospitalized for a week following his more serious head injury, and afterwards reported a change in personality, increased irritability, difficulties concentrating, explosiveness, problems with short-term memory, insomnia, anxiety, and mood swings. The only medication he was taking was testosterone, which had been necessary since the head injury just described.

As part of the evaluation a quantitative EEG was gathered on the patient. Analysis through the Nx Link database revealed that his discriminant scores were outside normal limits for his age ($p < 0.025$) and predicted the presence of post-concussion syndrome ($p < 0.025$). As seen in Figure 1, there was a very significant excess in absolute power alpha, especially in frontal areas where there was a 2.0-2.2 Z-score excess of alpha above norms. There was also a mild right frontotemporal and occipital excess in absolute power theta. Relative power alpha was also very excessive, especially in frontocentral areas, ranging from 2 to over 3 Z-scores in excess for his age. Further analysis through the NeuroGuide database showed excess occipital activity primarily from 5-9 Hz, and excess frontal absolute power alpha activity at 8-9 Hz. This frontal slow activity found in this case correlates with literature just reviewed involving posttraumatic anosmia.

After discussion and informed consent with the patient we agreed to use LENS for treatment. An offset assessment was conducted (with 4 seconds of stimulation) for his first treatment session, and an offset frequency of 5 Hz faster than his dominant frequency was determined to be the most effective frequency for reducing his slow EEG activity. His second treatment session consisted of 19 seconds of stimulation at the 19 standard electrode sites while we gathered a LENS map. He experienced no side effects from the treatment, and following the second session he reported that he felt "clearer in the head." Subsequent treatment sessions consisted of providing 1 second of stimulation at 7 electrode sites.

Before treatment began the patient was asked to rate his 8 most prominent symptoms (fatigue and lack of motivation, depression, anxiety, anger/explosiveness, insomnia, im-

FIGURE 1. NxLink QEEG of posttraumatic anosmic patient prior to treatment



pulsiveness, short-term memory problems, and poor concentration) on a 0-10 scale, where 0 represents no problem, and 10 a severe problem. He provided these symptom ratings at the beginning of each session. His mean pre-treatment symptom baseline was 9.0. At the end of 12 LENS treatment sessions his mean symptom rating was 5.1.

When the patient arrived for his 13th treatment session he indicated that after his previous treatment he had begun to smell things for the first time in 91/2 years. He first noticed a smell when he was outdoors on a construction site. At first he was confused and could not tell what he was experiencing, and then he finally realized that he was smelling sagebrush. It was at this interview that he also indicated for the first time that his sense of taste was "coming back." Previous to these spontaneous reports, neither of these problems had ever been mentioned. It was in this interview that he indicated that following the more serious head injury he had completely lost all sense of smell and his neurologist had told him that it would never return.

After 15 treatment sessions the patient reported that his sense of smell was "rapidly improving now," along with his sense of taste. He described smelling perfume, foods, and his dog. After 19 treatment sessions the patient indicated that he believed that most smells had now returned. He said, "I'm not used to it," and he indicated that occasionally he had been awakened in the night by a smell. We know that olfactory input is conveyed to the hippocampus, and thus it was fascinating that the patient said that as he was becoming aware of more smells they elicited "lots of memories." For example, when he was aware of smelling the scent of pine once more, it evoked memories of many camping trips. He further indicated that "food tastes *so* good now."

After 22 sessions the patient's mean symptom rating had been reduced from 9 to 3.75 and he indicated that his sense of smell and taste seemed completely normal. Two more treatment sessions were completed and then the patient had to interrupt treatment due to work demands, although he plans to come for further sessions in the future. Several contacts in the ensuing three months have found that his symptoms continue to be rated low and his sense of smell and taste remain normal.

A LENS map does not compare EEG data to a normative database. The EEG data is gathered sequentially at all 19 electrode sites, rather than simultaneously, and simply provides information on EEG amplitudes and variability. In an effort to understand the improvements in this case, the patient's LENS mapping data was examined. Because posttraumatic anosmia appears most related to frontal lobe damage, data on the frontal lobes was deemed particularly relevant for examination. This was also congruent with the findings in this particular case because the areas where the patient's QEEG showed deviations of 2 Z-scores or more in absolute power on the Nx Link database were primarily in frontal electrode sites (as well as at C3 and Cz). It was decided to compare the changes in LENS map data obtained after the first session of treatment with the data after 15 sessions, when the patient's olfactory and gustatory senses had substantively improved (although slightly greater improvements continued to be found in frontal areas after another LENS map following 22 treatment sessions). Table 1 displays amplitudes in delta, theta, and alpha frequency bands after 1 and 15 sessions, respectively. It can be seen that mean amplitudes decreased across frequency bands. The mean microvolt level decreased in delta by 0.32, in theta 0.31, and in alpha 0.9 (mean across frequency bands, 0.51μV).

When the total amplitudes across frequencies were averaged for the 7 frontal electrode sites, the mean at the beginning of treatment mean was 12.04, and the mean at the comple-

TABLE 1. Frontal EEG amplitudes after 1 and after 15 LENS sessions

Sites	Delta Pre	Delta Post	Theta Pre	Theta Post	Alpha Pre	Alpha Post
Fp1	3.24	2.69	5.30	5.72	9.41	9.69
Fp2	2.96	3.11	8.03	5.52	15.14	9.68
F3	2.04	2.80	5.19	7.12	8.52	10.26
F4	3.26	2.65	7.23	6.34	11.68	10.16
Fz	3.11	1.79	5.39	4.68	7.91	8.05
F7	2.51	2.04	4.75	3.65	8.38	5.93
F8	2.28	1.82	4.04	4.71	7.77	8.74
Means	**2.73**	**2.41**	**5.7**	**5.39**	**9.83**	**8.93**

tion of 15 sessions was 11.1. Thus it does appear that mean amplitudes decreased across frequency bands frontally, but most significantly in frontal alpha. We then examined the sum of the total amplitude plus variability (standard deviations) at all 19 standard electrode sites and averaged this sum in each frequency band. Delta across the entire scalp was found to have decreased in amplitude + variability by only .01, theta decreased .38, but alpha decreased 1.09. When the total amplitude combined with variability was then averaged across all electrode sites for all the combined frequency bands they were found to have changed from 15.73 to 15.03 (between sessions 1 and 15). It therefore appears that amplitude and variability as a whole decreased at all electrode sites, but primarily in the alpha frequency band.

CASE TWO

A second case has provided further encouragement that LENS neurofeedback may hold potential for overcoming anosmia. A 43 year old professional woman was referred for problems with anxiety. The patient did have a history of a concussion in an automobile accident where her head went through the windshield at age 15. She also experienced a whiplash almost 7 years earlier. A quantitative EEG was done and the data analyzed in the NxLink database. It showed a pattern of diffuse excess beta activity and the patient's discriminant scores suggested the probability that she suffered with post-concussion syndrome ($p <= 0.05$). Ten sessions of LENS neurofeedback were provided, during which time the anxiety steadily improved and energy increased.

After 9 LENS sessions, as part of casual conversation, the author mentioned to her the improvement in smell in the case of the patient described above. She indicated that this was fascinating because in the last few weeks she had noticed that her sense of smell had noticeably improved, and she had told her companion about this during the previous two weeks. Having been unaware that she even had a problem with anosmia, further inquiry was made. It was learned that she had 4 sinus surgeries in the 1990's. Ever since that time her sense of smell had been quite impaired, but not completely absent. It is unknown, of course, whether her whiplash in 1999 could have contributed to her diminished sense of smell, or if the sinus surgeries did damage to the transit between the sinuses and the brain.

DISCUSSION AND CONCLUSIONS

Anosmia is a symptom that may accompany an acceleration-deceleration traumatic brain injury, particularly of moderate or greater severity. Impairment in taste commonly accompanies anosmia. Posttraumatic anosmia has commonly been regarded as a permanent condition and has proven resistant to medication treatment. A literature review by Hummel and Nordin (2005) found that anosmia has a very severe negative effect on quality of life, safety and interpersonal relations, as well as eating habits and nutritional intake.

However, two cases that were treated with LENS neurofeedback have shown an unexpected reversal of anosmia, to the delight of the patients and the complete surprise of the author. These uncontrolled case reports provide encouragement that posttraumatic anosmia may be reversible in some cases through the use of neurofeedback. How such changes can occur remains uncertain, but in the first case it was documented that EEG amplitudes decreased, particularly in the alpha frequency band frontally where the greatest excess was found in QEEG evaluation. We might speculate that neurofeedback may possibly produce improvements in the orbitofrontal cortex and olfactory bulb that were damaged in acceleration-deceleration head injuries. Since we know that new axons and synapses are continually forming in areas associated with olfaction (Joseph, 1996, p. 179), is it possible that new axonal growth may be occurring in the olfactory fila or that damaged cranial nerve I fibers may be positively influenced by neurofeedback?

Clinicians working with TBI cases are encouraged to routinely inquire during evaluation concerning anosmia, and to ask about improvements in smell during the course of neurofeedback.

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