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Multi-Watt Near Infrared Phototherapy as a Treatment for Traumatic Brain Injury

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ABSTRACT: Background: Depression treatment is hampered by low efficacy of antidepressant medications and concerns about alternative modalities. Animal studies of treatment with low-level (0.5 Watt or less) near infrared (NIR) light from diodes has shown some benefit in models of traumatic brain injury (TBI) with evidence of reduced lesion size, increased neurotrophin production, synaptogenesis, fewer apoptotic cells, and improved neurological function. Two small case series have demonstrated transient clinical improvement with low-level NIR treatment given on a daily basis over several weeks. We have previously shown marked and persistent clinical improvement in a case series of patients with chronic mild-to-moderate (m-MTBI) after treatmentwith NIR at a power of 9 Watts or greater. We also have published a review of the potential for application of NIR for the treatment of depression. The current study explores NIR efficacy in a proof-of-concept study as a treatment for depression.

METHODS: Thirty-nine sequential patients treated for TBI between March 2013 and May 2017 provided depression self-assessment data and/or were administered the Hamilton Depression Rating Scale. Each completed the Quick Inventory of Depression Symptomatology-Self Reports (QIDS) before and after treatment. Patients received transcranial multi-Watt near-infrared light treatment (NILT) using near-infrared lasers (810/980 nm at 8-15 Watts) applied to forehead and temporal regions bilaterally for 9-12 minutes to each area.

RESULTS: For 36 of the 39 patients, after 16.82 + 6.26 treatments, QIDS scores indicated a robust response (decrease of QIDS total score by > 50%). For 32 of 39 patients post-treatment QIDS scores indicated a remission from depression (decrease of QIDS total score < 5). Overall, the QIDS score fell from 14.10 + 3.39 to 3.44 + 3.39 SD (p = 6.29 X 10-19). With 12 or fewer treatments, QIDS score dropped from 14.83 + 2.55 to 4.17 + 3.93. Patients receiving 13 or more treatments showed a change in QIDS score from 13.67 + 3.64 to 3.11 + 3.14. Those (N = 15) who received the entire treatment course within 8 weeks or less (5.33 + 1.72 weeks) showed a change in QIDS score from 13.86 + 3.14 to 4.5 + 3.94. Suicidal ideation resolved in all, but two patients. The non-responsive patients are described in detail. Patients remained in remission for up to 55 months after a single course of treatment.

CONCLUSION: This is the first report of high-powered NILT showing efficacy for depression. Patients saw benefit often within 4 treatments and some had resolution of depressive symptoms in as little as 4 weeks. These data raise an intriguing possibility – that NILT may be a safe, effective, and rapid treatment for depression. A double-blind, placebo controlled trial is warranted to verify these proof-of-concept data.

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A New Syndrome: Phantogeusia-Induced Phantosmia

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ABSTRACT: Case Objective: While phantosmia-induced phantogeusia has been described (Ahmed, 20173), the reverse, phantogeusia-induced phantosmia, has not heretofore been described. Such a case is presented.

METHODS: Case Study: A 39-yr-old left-handed (pathological) male, six years prior to presentation, noted a sudden onset of phantogeusia of roast cooking, pizza, fruit, strawberries, or a sour taste, and shortly thereafter he would develop unpleasant phantosmias which would sometimes combine with the ambient aroma. These would occur 3-10 times per week and would last for the duration of the phantogeusia, for as long as 1-2 hours. Occasionally the phantosmia would occur first and then induce the phantogeusia of asour taste.

RESULTS: Abnormalities in Neurologic examination: Mental status examination: Immediate recall: Digit span: 6 digits forward and 3 digits backwards. CN XI, X: Decreased gag bilaterally. Motor Examination: Drift: left pronator drift with right abductor digiti minimi sign and right cerebellar spooning. Gait Examination: Tandem Gait: unstable. Cerebellar Examination: Holmes Rebound Phenomena: bilaterally positive, left greater than the right. Sensory Examination: Ipswich Touch Test: decreased in left lower extremity. Temperature: decreased in left lower extremity. Rydel-Seiffer Vibratory Test: bilateral upper extremities 5, bilateral lower extremities 3. Reflexes: upper extremities 1+, absent lower extremities. Neuropsychiatric Examination: Go-No-Go Test: 6/6 (normal). Animal Fluency Test: 15 (normal). Clock Drawing Test: 3 (abnormal). Center for Neurologic Study Lability Scale: 16 (Pseudobulbar affect).

CONCLUSION: Close connection of the tertiary smell and taste integration areas, where smell and taste converge, in the posterior orbitofrontal cortex, anterior to the insular taste cortex, and posterior to the granular orbitofrontal cortex may have allowed activation of memory engrams connecting these two (Rolls, 19944). Alternatively, electrical discharge from the primary taste area may have spread to involve the cortical representation of smell. Since the cortical area involved in the interpretation and hedonics of taste co-localize with the area involving olfactory hedonics, spread from one area to the other area may occur. As a result of electrical discharge (from an epileptiform focus) or as a result of well-connected and developed memory engrams with associated hedonics, phantom tastes may induce phantom smells. Alternatively, this may represent a distorted retronasal smell whereby the olfactory component of the gustatory hallucination causes a discharge of the olfactory epithelium (a pseudoretronasal smell).
Given the above, treatment of those with both phantosmia and phantogeusia may respond to treatment of phantogeusia alone. Under this construct, the phantosmia is the slave of the phantogeusia whereby management of the taste hallucination will thus eliminate the smell hallucination.

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