

duration (“on-time”) to ITI (“off-time”). PubMed and SCOPUS databases were searched through March 30, 2015 using the terms: “transcranial magnetic stimulation”, “TMS”, “rTMS”, “inter-train interval”, “inter-stimulus interval”, and “cortical spread”. Three hundred and one articles were identified comprising a total of 3359 patients. Clinical outcomes were reported as the proportion of patients achieving response defined as 50% reduction in baseline score on the primary outcome measure in each study. Treatment risk was assessed by the frequency of adverse events reported, and specifically considering the incidence of seizures.

RESULTS: This analysis confirms prior reports that the variables which impact treatment efficacy are the number of treatment sessions, the number of pulses per session and the percent motor threshold. Varying the train duration/ITI (on-time/off-time) ratio over a broad range from 2.0 to 14 did not impact efficacy or safety.

CONCLUSIONS: Shortening the ITI to 11 seconds does not impact the safety and antidepressant effectiveness of the NeuroStar TMS and would result in shortening of each treatment session from approximately 37.5 to 19 minutes.

FUNDING ACKNOWLEDGEMENTS: Neuronetics, Inc.

161

The NeuroStar Outcomes Registry

Miriam Mina¹; David G. Brock, MD²; W. Scott West, MD³; Todd Hutton, MD⁴; Kenneth P. Pages, MD⁵; and Timothy J. Kane, MD⁶

¹ Neuronetics, Inc., Malvern, PA

² no affiliation, Ambler, PA

³ ThriveLogic TMS + NeuroHealth, Vanderbilt University, Nashville, TN

⁴ Southern California TMS Center, USC Keck School of Medicine, Pasadena, CA

⁵ TMS of South Tampa, Tampa, FL

⁶ Comprehensive Health Systems, PLC, Fishersville, VA

ABSTRACT: The NeuroStar Outcomes Registry

OBJECTIVE: NeuroStar transcranial magnetic stimulation (TMS) is an effective acute treatment for patients with major depressive disorder (MDD). In order to further understand use of the NeuroStar in a clinical setting, Neuronetics has established a patient treatment and outcomes registry to collect and analyze utilization information on patients receiving treatment with the NeuroStar.

METHODS: Individual NeuroStar providers are invited to participate in the registry and agree to provide their de-identified patient treatment data. The NeuroStar has an integrated electronic data management system (TrakStar) which allows for the data collection to be automated. The data collected for the registry include Demographic Elements (age, gender), Treatment Parameters, and Clinical Ratings. Clinical assessments are: Clinician Global Impression - Severity of Illness (CGI-S) and the Patient Health Questionnaire 9-item (PHQ-9). De-identified patient data is uploaded to Registry server; an independent statistical service then creates final data reports.

RESULTS: Over 500 patients have entered the NeuroStar Outcomes Registry since Sept 2016. Mean patient age: 48.0 (SD ± 16.0); 64% Female. Baseline PHQ-9, mean 18.8 (SD ± 5.0.) Response/Remission Rate, PHQ-9: 61%/33% CGI-S: 78%/59%.

CONCLUSIONS: For the initial 500 patients in the Outcomes Registry, approximately 2/3 patients achieve response and 1/3 patients achieve remission with an acute course of NeuroStar. These treatment outcomes consistent with NeuroStar open-label study data (Carpenter, 2012). The TrakStar data management system makes large scale data collection feasible. The NeuroStar Outcomes Registry is ongoing, and expected to reach 6000 outpatients from more than 47 clinical sites in 36 months.

FUNDING ACKNOWLEDGEMENTS: Neuronetics, Inc.

162

Add-on Filgrastim During Clozapine Rechallenge Unsuccessful in Treating Benign Ethnic Neutropenia

Molly Britton, MD¹; Palanikumar Gunasekar, MBBS²; and Vithyalakshmi Selvaraj, MD³

¹ Psychiatry resident, Department of Psychiatry, Creighton University School of Medicine, Omaha, NE

² Post doctoral fellow, Department of Clinical Translational Science, Creighton University School of Medicine, Omaha, NE

³ Associate Professor, Department of Psychiatry, Creighton University School of Medicine, Omaha, NE

ABSTRACT: Clozapine is an atypical antipsychotic approved by the Food and Drug Administration for treatment-resistant schizophrenia and also indicated for the reduction in risk of recurrent suicidal behavior in patients with schizophrenia or schizoaffective disorder. The most serious side effect of clozapine treatment is agranulocytosis, which is defined as an absolute

neutrophil count (ANC) $< 0.50 \times 10^9$ per L. Benign ethnic neutropenia (BEN) is a condition found in members of African or Middle Eastern descent that is characterized by ANC $< 1.50 \times 10^9$ per L in the absence of other causes. Filgrastim is a granulocyte colony-stimulating factor (G-CSF) that has shown efficacy in reducing the duration of agranulocytosis in some patients who develop clozapine-induced agranulocytosis. It is currently unknown whether filgrastim is beneficial in the treatment of neutropenia due to BEN. We here, for first the time report a case of a patient with BEN who developed agranulocytosis both during the first clozapine trial for schizophrenia and during the rechallenge, despite early stabilization with filgrastim treatment, which highlights the failure of filgrastim in treating BEN.

FUNDING ACKNOWLEDGEMENTS: No funding.

163

Treatment of Odor-Induced Anxiogenesis With Odor-Induced Anxiolysis

Muktiben M. Patel¹; Nigam D. Patel, MB²; Angela Rekhi, MD³; and Alan R. Hirsch, MD⁴

¹ Aureus University School of Medicine, Aruba, West Indies

² Nanjing Medical University, Nanjing, China

³ Aureus University School of Medicine, Aruba, West Indies

⁴ Smell & Taste Treatment and Research Foundation, Chicago, IL

ABSTRACT: Study Objective: To understand the effects of odor on anxiety.

INTRODUCTION: Reduction of odor-induced anxiety through a presentation of an odor has not heretofore been described.

METHOD: Case report: A 69-year-old right-handed male with a five year history of generalized anxiety disorder, presented with a one and a half month history of hypersensitivity to odors of multiple synthetic chemicals manifest by the perception that these odors were more intense and unpleasant inducing nausea, abdominal cramping, coughing, a need to “get away from the smell”, and panic with intense anxiety. These symptoms would occur whenever he was exposed to these smells, 20 to 25 times a day, and would persist for 10 to 15 minutes after the exposure. When odors induced the above symptoms, exposure to the aroma of cinnamon immediately alleviated these symptoms. He now continues using cinnamon odor whenever the odor induced anxiety and associated symptoms arise. This remedy has been effective over the course of treatment, for almost two years.

RESULTS: Abnormalities on examination: Three per second titubation. Archimedean Spiral Test: Saw tooth pattern with macrographia. Anxious, circumstantial, overly inclusive. Unable to determine how to put on shoe covers. Impaired voluntary upward gaze, but intact vertical doll's eyes. Left torticollis. Bilateral finger to nose dysmetria. Low amplitude, high frequency tremor on extension of both upper extremities. Areflexic. Olfactory Testing: hyposmic. MRI of brain with and without infusion: mild generalized volume loss.

CONCLUSIONS: There are myriad mechanisms whereby odor may have reduced the odor-induced anxiety. Since aroma induced anxiogenesis is usually confined to a specific odor, it does not preclude other odors from acting in an anxiolytic manner. The combination of exposure simultaneously of anxiolytic and anxiogenic odors may have acted to increase the threshold of the anxiety producing odor, inhibiting perception of the anxiogenic odor and thus precipitation of anxiety. The two odors could have combined in an additive fashion, changing the olfactory characteristics of the anxiety provoking odor such that it no longer was perceived as the same odor and thus no anxiety. The anxiolytic/anxiogenic odor mixture could have overwhelmed the anxiogenic odor, thus creating the perception of only anxiolytic odor. On a central basis, the anxiolysis and anxiogenesis may have been induced to occur coincidentally with anxiolysis superseding anxiogenesis. Alternatively, the odors may have acted as a distractor, changing the focus of attention from anxiogenic odor to a different odor which does not have the same anxiety provoking effect. Maybe because the patient already has demonstrated a heightened odor emotion linkage, he may be more susceptible to any other odor emotion effects. Trial of odors in those with odor induced anxiety warrants consideration.

FUNDING ACKNOWLEDGEMENTS: No funding.

170

Prospective Evaluation of the Economic Utility of Combinatorial Pharmacogenomics in Generalized Anxiety Disorder and Major Depressive Disorder

Nathan Roe, PhD¹; Catherine Passariello, PhD¹; Lisa Brown, PhD¹; James Li, MS¹; Michael Jablonski, PhD¹; and Bryan M Dechairo, PhD¹

¹ Assurex Health, Mason, OH

ABSTRACT: Mental illness is one of the leading causes of disability, with direct and indirect costs posing a significant financial burden. Previously, a large prospective economic utility study (n > 13,000) showed that the GeneSight® test, a psychiatric pharmacogenomic