JCSM
Journal of Clinical Sleep Medicine

LETTERS TO THE EDITOR

Dissociative Identity Disorder and Obstructive Sleep Apnea

Response to Gupta and Pur. Obstructive sleep apnea severity in dissociative identity disorder can vary significantly depending upon the autonomic activation status of the personality that has executive control. *J Clin Sleep Med*. 2018;14(9):1633.

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In their letter, Gupta and Pur[1] provide much to agree with and logic is presented related to the premise of personality influencing sleep pathophysiology. Gupta and Pur also provide two references that we missed that indicate that when awake, patients with dissociative identity disorder (DID) can have autonomic nervous system (ANS) physiologic differences across personalities. In the patient we describe in our case report,[2] we had only one value for the diagnostic apnea-hypopnea index (AHI) and no personality type was reported in the diagnostic study or follow-up until the causes for adherence were investigated as we did. Please refer to Figure 1 in our original paper.[2] The other personalities, when combined, had the same data for AHI on continuous positive airway pressure (CPAP) therapy even though the personality (if we can equate to frontal lobe and ANS differences) was that of a pre-pubertal female and the upper airway was that of a mature woman. The patient was on a fixed CPAP of 9 cmH_2O. We, however, cannot address the premise with the information we have.

We considered what data there might be to test the hypothesis suggested by Gupta and Pur—that personality, ANS type, or frontal lobe function cause severity of obstructive sleep apnea (OSA). Are there clues in the literature? Cross-sectional epidemiological studies, that presumably contain many personalities, have physiological outcomes of AHI at baseline. For instance, an elevated AHI results in a 5-fold increase in hypertension at 5 years in normotensive individuals, yet there is a wide range of blood pressure (ANS) outcomes.[3] Other studies reported in this review also show a wide range of OSA severity in the community, both across the range of AHI and within each severity category (mild, moderate, severe). The reference to Eckert et al.[4] is to phenotype causes of OSA through physiologic mechanisms; however, none are directly tied to a predisposing personality feature. The presentation of OSA also has five optimal clusters: three previously reported in Iceland (labeled disturbed sleep, minimal symptoms, and upper airway symptoms with sleepiness) and two new, less symptomatic clusters (labeled upper airway symptoms dominant and sleepiness dominant)[5] but no personality subtypes were collected. What Gupta and Pur suggest, however, is a prospective trial that captures personality in the DID spectrum and follows through on the response to therapy, using both presenting symptoms and mechanisms.

CITATION

Gandotra K, Rosenberg C, Strohl K. Dissociative identity disorder and obstructive sleep apnea. *J Clin Sleep Med*. 2018;14(9):1635.

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SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication July 10, 2018
Submitted in final revised form July 10, 2018
Accepted for publication July 10, 2018
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DISCLOSURE STATEMENT

All authors have seen and approved the manuscript. Work for this study was performed at MetroHealth Medical Center, Cleveland, OH. The authors report no conflicts of interest.