

## Podcast of the Journal of Clinical Sleep Medicine

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Welcome to the regular Podcast of the Journal of Clinical Sleep Medicine. I am Dr. Stuart Quan, Editor of the Journal. These Podcasts are a regular feature of each issue of the Journal and can be downloaded at the Journal's website. Each Podcast features summaries of important articles published in the current issue of the Journal, as well as occasional interviews with authors of these papers.

The lead article in this issue of the journal is entitled, "Does CPAP Lead to a Change in BMI?" by Rachel Redenius, Carli Murphy, Erin O'Neil, Majed Al-Hamwi and Sarah Nath-Zallick from Illinois Neurologic Institute Sleep Center, Peoria, IL and University of Illinois College of Medicine at Peoria. How many of us clinicians have had a patient ask, "Gee, if I use CPAP will I lose weight?" This is an important question in counseling patients in CPAP adherence because the more incentives one can provide for a patient to use CPAP, the more likely it will be that they will actually adhere to the therapy. In this study, clinic patients were divided into those who were adherent to CPAP more than four hours/night and using it at least 70% of these nights, and control subjects who did not use CPAP or were less compliant with CPAP usage. BMI was determined at the time of diagnosis and at follow up after approximately one year. There were 183 subjects in the treatment group and 45 subjects in the control group. The average age of the subjects was 55.4 years and the average initial BMI was significantly obese at 35.9 kg/m<sup>2</sup>. There were no differences in the initial BMI between the treatment subjects and those in the control group. After approximately one year of treatment with CPAP, or no CPAP, there was no change in the BMI in either the treatment or the control groups. Moreover, when men were analyzed separately from women, there was no change in the BMI in the men in the treatment group. However, the women actually gained, on average, 0.5 kg/m<sup>2</sup>—a statistically significant change. Furthermore, when subjects were selected as being highly compliant with CPAP, in comparison to the control subjects, those in the treatment group also gained weight. This study provides evidence that patients who are starting out on CPAP therapy for obstructive sleep apnea should not be told that they necessarily will experience weight loss without actually actively trying to increase their physical activity and reducing their caloric intake. However, this study was retrospective in nature and does require some further confirmation. A prospective study on this topic is currently being conducted in the APPLES clinical trial, as noted in the accompanying commentary entitled, "Is There A Bi-Di-

rectional Relationship Between Obesity and Sleep-Disordered Breathing" by Drs. Quan, Budhiraja and Parthasarathy.

Portable monitoring is gaining increasing recognition as a possible diagnostic tool for identifying individuals with obstructive sleep apnea. Currently recommended portable monitors utilize sensors that measure airflow, respiratory effort and oximetry. However, there is increasing interest in developing portable monitors that are simpler and require less technology. In this issue of the Journal, Drs. Heneghan, de Chazal, Ryan, Chua, Doherty, Boyle, Nolan, and McNicholas describe a paper in which an electrocardiogram recording is utilized as a screening tool for sleep-disordered breathing. They applied a previously developed automatic algorithm to a single channel ECG obtained during a standard overnight polysomnogram in 92 subjects to obtain an estimate of the apnea-hypopnea index in these individuals. Using apnea-hypopnea index thresholds of less than 5 and greater than or equal to 15 events/hour to define the presence or absence of sleep-disordered breathing, they calculated the likelihood ratios that their algorithm would detect sleep-disordered breathing. Their technique achieved positive and negative likelihood ratios at 2.16 and 0.08 and there was a high correlation between the estimated and actual apnea-hypopnea index of 0.88. These data, therefore, provide promising preliminary findings to suggest that use of a simple recording technology with a sophisticated analysis algorithm can identify sleep-disordered breathing in a clinical population. However, the reader should be cautioned that these subjects were derived from a sleep-disorders clinic and not from the general population. Thus, the applicability of this technique as a screening tool for the general populace cannot be determined.

There are a few studies of hypnotics, which compare the effects of one hypnotic to another. Most hypnotic studies compare the designated hypnotic to a placebo. In this issue of the Journal, Drs. Erman, Zammit, Rubens, Schaefer, Wessel, Amato, Caron, and Walsh present a paper entitled, "A Polysomnographic Placebo-Controlled Evaluation of the Safety and Efficacy of Eszopiclone Relative to Placebo and Zolpidem In the Treatment of Primary Insomnia." It should be noted that support for this study was provided by Sepracor, the makers of eszopiclone. This study was a multi-center randomized crossover study of adults with primary insomnia who received two-nights treatment, each with placebo, four doses of eszopiclone and zolpidem. Efficacy was determined by polysomnography with the primary end-point latency to persistent sleep. A sec-

ondary end-point was sleep efficiency. All active treatments, that is eszopiclone, 1 mg, 2 mg, 2.5 mg or 3 mg, and zolpidem, 10 mg, showed significant differences in improvement in latency to persistent sleep and sleep efficiency in comparison to placebo. There were no differences among all the active treatments. However, the incidence of central nervous system adverse events was greater for zolpidem than for the eszopiclone doses and placebo. The data suggests that although all treatments were equally efficacious in the treatment of primary insomnia for this short-term study, there were slightly greater incidences of side effect using zolpidem. The reader should be cautioned, however, that this was a short-term treatment study and effects for more than two nights cannot be inferred and the incidence of all adverse events, including the unpleasant taste that has been associated with eszopiclone, was not different among all treatment groups including placebo.

The final study to be highlighted in this Podcast is entitled, "Correlation Between Severity of Obstructive Sleep Apnea and Prevalence of Silent Cerebral Vascular Lesions" by Drs. Nishibayashi, Miyamoto, Miyamoto, Suzuki, and Hirata from

Dokkyo Medical University in Tochigi, Japan. This was a cross-sectional study of 192 polysomnographically confirmed individuals drawn from a sleep-disorders clinic who had magnetic resonance imaging of the brain. Silent lacunar infarction was identified in 21.1% of control subjects, 12% of patients with mild obstructive sleep apnea, 48.6% of those with moderate sleep apnea and 54% of those with severe sleep apnea. Moderate sleep apnea was defined as those individuals with an AHI of greater than 15 events/hour and severe sleep apnea were those individuals with AHI greater than 30 events/hour. The data suggests that there is a high prevalence of silent cerebral vascular lesions in individuals with moderate to severe obstructive sleep apnea. These data, along with other studies suggesting a linkage between sleep apnea and stroke provide more evidence to indicate that individuals with moderate to severe sleep apnea should be aggressively treated.

This concludes the regular Podcast of the June 15, 2008, issue of the *Journal of Clinical Sleep Medicine*. The listener is encouraged to read the articles summarized in their entirety, as well as other papers published in this issue of the *Journal*.