

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.

This issue of the *Journal* features two editorial perspectives on whether obstructive sleep apnea is a risk for hypertension. Nine years ago, the Wisconsin Sleep Cohort published a landmark study demonstrating that there was a relationship between obstructive sleep apnea and the development of hypertension. This was the first long-term study demonstrating that obstructive sleep apnea was a risk factor for incident hypertension in a general population. Previously, studies linking hypertension and obstructive sleep apnea were cross sectional and thus causality could not be inferred. More recently, the prospective data from the Sleep Heart Health Study was published and on superficial review of this data, it would appear that obstructive sleep apnea was not an independent risk factor for the development of hypertension. In the first perspective, Dr. Paul Peppard from the University of Wisconsin, who was the first author of the paper from the Wisconsin Sleep Cohort, gives his explanation of why the results from these two studies differ in their outcomes. First, he points out that the baseline comparison category for the Sleep Heart Health Study was an apnea-hypopnea index less than five whereas it was zero in the Wisconsin Sleep Cohort. He indicates that recalculation of the data from the Wisconsin Sleep Cohort using the baseline definition of the Sleep Heart Health Study would have decreased their associations by approximately nine percent. Second, he argues that methods of determining the apnea-hypopnea index between the two studies are not necessarily comparable. In their sleep laboratory, comparisons between in-lab and in-home polysomnography found that in-home apnea-hypopnea indices were more than twofold higher than the in-lab measurements. As a result, the highest severity category in the Wisconsin Sleep Cohort, that is more than 15 events per hour, is more comparable to the severity category in the Sleep Heart Health Study of 30 events per hour. Finally, he notes that in order to determine the rate of incident hypertension in the Sleep Heart Health Study, subjects with current hypertension were excluded from the analysis. This was 51% of the total Sleep Heart Health Study cohort. In contrast, the same exclusion applied to Wisconsin Sleep Cohort, only eliminated

27% of the sample. He thus reasons that if obstructive sleep apnea is a cause of hypertension and that there is variability in the susceptibility to this risk factor, the Sleep Heart Health Study would have excluded a greater proportion of subjects at baseline who were relatively more susceptible to developing hypertension from sleep apnea than the Wisconsin Sleep Cohort.

In a second perspective, Dr. Susan Redline from the Sleep Heart Health Study and Case Western Reserve University provides an additional in depth discussion of the results from their recent analysis. First, she notes that the effect sizes from the incident hypertension analysis is almost identical to those estimates obtained from the previously published cross-sectional analysis performed in this cohort. Furthermore, the incident analysis was done in a population that was reduced by 40% in comparison to the cross-sectional analysis and thus if one were to look at the 90% confidence intervals and relax the P-value required for significance to less than 0.10, the results would be considered statistically significant. She argues that given the remarkable consistency of the point estimates for both the cross-sectional and longitudinal analyses, and the expected variability related to a smaller sample size, the results from the longitudinal and cross-sectional analyses combined point to an increased risk of developing hypertension as a result of obstructive sleep apnea. A second point made by Dr. Redline was that when the Sleep Heart Health analysis was restricted to those participants who were not obese, the odds ratio for development of hypertension was 2.71 with an apnea-hypopnea index exceeding 30 and this was statistically significant although the odds ratio is tempered by the relatively small numbers of participants. However, this sub-analysis provides evidence of an association between obstructive sleep apnea and hypertension that is not contaminated with obese individuals. She also notes that the odds ratio of 1.5 in the Sleep Heart Health Study for obstructive sleep apnea is quite similar to odds ratios for risk factors for cardiovascular disease reported in other cohort studies.

These two perspectives provide critical insight into the relationship between obstructive sleep apnea and hypertension. The results from both the Wisconsin Sleep Cohort and the Sleep Heart Health Study I believe provide strong evidence for a relationship between development of hypertension and the presence of obstructive sleep apnea. Support for this also can be found in smaller clinical case series as well as in some studies in which sleep apnea is treated and blood pressure is reduced in affected individuals.

The next paper featured in this podcast is entitled, “Effects of Heated Humidification and Topical Steroids on Compliance, Nasal Symptoms, and Quality of Life in Patients With Obstructive Sleep Apnea Syndrome Using Nasal Continuous Positive Airway Pressure,” by Silke Ryan, Liam Doherty, Geraldine Noland, and Walter McNicholas from the St. Vincent’s University Hospital and the School of Medicine & Medical Science University College in Dublin, Ireland. This study randomized patients with obstructive sleep apnea to use of CPAP with humidification, use of nasal fluticasone, or use of a heated humidifier. After four weeks of therapy, there was no difference in compliance between all three groups. Moreover, quality of life and subjective sleepiness improved in all groups. However, nasal symptoms were less frequently reported in the humidifier group in comparison to the non-humidifier group and those receiving fluticasone. Fluticasone also increased the frequency of sneezing. Nasal symptoms are frequently encountered in patients using nasal CPAP without humidification. Topical nasal steroids are sometimes prescribed for these patients. These results would suggest that topical nasal steroids have limited efficacy in the treatment of nasal symptoms in those individuals who have rhinitis from the use of nasal CPAP.

Another interesting paper published in this issue of the Journal is entitled, “Poor Long-Term Patient Compliance with a Tennis Ball Technique for Treating Positional Obstructive Sleep Apnea,” by James Bignold, Georgina Deans-Costi, Mitchell Goldsworthy, Claire Robertson, Douglas McEvoy, Peter Catcheside, and Jeremy Mercer from Adelaide, Australia. Positional obstructive sleep apnea is commonly observed among patients with this condition. Advising patients to sleep only on their side is a frequent prescription. Many practitioners advise patients to sew tennis balls or similar objects on the back of their night clothes so that the patient does not sleep on their back. However, the efficacy of this treatment has not been well studied. In this study, 77 patients out of a potential 108 patients were surveyed by mail to determine whether they were complying with using the tennis-ball technique for treatment of their positional obstructive sleep apnea. The average follow-up time was 2.5 years. The results suggest that the tennis-ball

technique is not effective therapy. 80.6% of respondents were neither using the tennis-ball technique nor avoiding the supine position. Although there are obvious problems with using survey techniques to determine compliance with treatment, these data would suggest that a better method of trying to keep patients from sleeping on their back is needed to treat positional obstructive sleep apnea.

The final article to be highlighted in this podcast is entitled, “The Tongue-Retaining Device: Efficacy and Side Effects in Obstructive Sleep Apnea Syndrome,” by Diane Lazard, Marc Blumen, Pierre Lévy, Pierre Chauvin, Dorothée Fragny, Isabelle Buchet, and Frédéric Chabolle from the Hôpital Beaujon in Clichy, France. Although oral appliances are now increasingly used for the treatment of obstructive sleep apnea, most appliances are mandibular advancement devices. However, these devices are not practical for those individuals with poor dentition or absent teeth. The tongue-retaining device does not require presence of natural teeth and has been demonstrated in some studies in small populations to be efficacious in the treatment of obstructive sleep apnea. However, data from larger cohorts are not available. In this study, the tongue-retaining device was studied in a population of 84 patients with obstructive sleep apnea. The authors found that a complete or partial response was obtained in 71% of cases with a decrease in the apnea-hypopnea index from 38 to 14 events per hour. An age greater than 60 years and a mandibular protrusion distance less than or equal to seven millimeters was predictor of a poor response. In addition, nasal obstruction was a negative predictor of good compliance. These data suggest that the tongue-retaining device may be a viable alternative for some individuals with obstructive sleep apnea. This opinion was supported in an editorial that followed this paper by Dr. Rosalind Cartwright from Rush University Medical Center in Chicago, Illinois.

This concludes the regular podcast of the *Journal of Clinical Sleep Medicine*. The listener is encouraged to read the contents of the *Journal* for additional information regarding each of the papers summarized in this podcast, as well as other papers published in this issue of the *Journal*.