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PODCAST TRANSCRIPT

The First Podcast of the Journal of Clinical Sleep Medicine

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Welcome to the first Podcast of the Journal of Clinical Sleep Medicine. I am Dr. Stuart Quan, Editor of the Journal. It is my hope that the readership of the Journal will find that these Podcasts are a useful way to disseminate information contained in the Journal. The Podcasts will contain brief summaries of some of the key articles in each edition of the Journal, as well as from time to time, interviews with the principal authors of these papers. I welcome your feedback and you can feel free to provide comments by emailing them to me, care of amiller@aasmnet.org.

A pro-con debate in this issue of the Journal features Dr. Lee Brown, from the University of New Mexico, arguing that patients with mild sleep apnea require treatment and Dr. Michael Littner, from the greater Los Angeles VA Healthcare system and the University of California at Los Angeles, arguing that patients with mild sleep apnea do not require treatment.

In his arguments for treating mild sleep apnea, Dr. Brown summarizes data that mild sleep apnea can cause symptoms, can lead to adverse sequelae, can be treated and thus lead to improved outcomes. With respect to symptoms, he points out that the data from the Sleep Heart Health Study showed that sleepiness, as measured by the Epworth Sleepiness Scale, increased steadily with increasing OSA severity, and that the percentage of individuals who were sleepy with apnea-hypopnea indices between five and 15 was greater than those with apnea-hypopnea indices less than five. He also cites evidence from the Sleep Heart Health Study demonstrating that quality of life was worse in individuals with mild sleep apnea. With respect to adverse cardiovascular metabolic outcomes, Dr. Brown cites data from both the Sleep Heart Health Study and the Wisconsin sleep cohort, demonstrating that the prevalence of hypertension was greater in those with relatively mild sleep apnea in comparison to those who did not have this condition. Similar findings were also demonstrated by the Sleep Heart Health Study for cardiovascular disease prevalence, with increased odds of having cardiovascular disease even in those with apnea-hypopnea indices between 1.4 and 4.4 events/hour. Similar data are present for the risk of glucose intolerance with an increase in four year incidence of diabetes mellitus in subjects in the Wisconsin sleep cohort who had an apnea-hypopnea index between five and 15. Finally, Dr. Brown cites data demonstrating that mild sleep apnea can be treated with mandibular advancing devices, as well as surgery, and that clinical trials have seen improvement in treatment of these individuals with respect to their Epworth Sleepiness Scale, quality of life and performance on neurocognitive test batteries. He concludes that there is clear evidence that mild obstructive sleep apnea can be symptomatic, can lead to adverse consequences, and can be treated successfully.

In contrast, Dr. Michael Littner argues that mild sleep apnea does not require treatment. In his half of the pro-con debate, he points out that the American Academy of Sleep Medicine published evidence-based practice parameters in 2006 regarding the use of CPAP in obstructive sleep apnea. The practice parameters indicated that CPAP was recommended as treatment for mild sleep apnea but that this was optional, with optional implying inconclusive or conflicting evidence or conflicting expert opinion. He emphasizes that the evidence review behind this recommendation found that there was no change in blood pressure following treatment in mild sleep apnea comparing a placebo tablet to CPAP treatment. In addition, studies evaluating the impact of CPAP vs. placebo on heart rate have produced conflicting results. He also cites several studies indicating that the use of CPAP in mild to moderate sleep apnea reduced the apnea-hypopnea index but did not improve sleepiness or blood pressure. Furthermore, there were conflicting results for improvement in neuro behavioral performance, mood and quality of life. Moreover, he emphasizes that patients with mild sleep apnea are less adherent to treatment, specifically CPAP. Additionally, recent studies have demonstrated increased mortality only in those with an apnea-hypopnea index greater than 30, which is certainly not in the mild range. He summarizes his arguments by indicating the benefits of CPAP in the treatment of daytime sleepiness, in those with no daytime symptoms, for reduction in cardiovascular risks, improvement in quality of life and reduction in mortality are minimal or non-existent in those with mild sleep apnea.

I invite the listener to read both of these papers in the pro-con debate and come to their own conclusions.

The lead article in this edition of the Journal is entitled, "Association of Alcohol Consumption In Sleep-disordered Breathing in Men & Women" by Paul E. Peppard, Diane Austin and Richard L. Brown from the University of Wisconsin in Madison. This paper is an analysis of data from the Wisconsin Sleep Cohort. The Wisconsin Sleep Cohort is an ongoing epidemiologic cohort study of the natural history of sleep-disordered breathing. It was started in 1988 with the recruitment of middle-aged Wisconsin state employees. This analysis of the Cohort involved 1, 420 participants who had a total of 3,165 sleep studies. There were 775 men and 645 women. The purpose of the analysis was to determine the association between sleep-disordered breathing and usual alcohol consumption habits. Sleep-disordered breathing was assessed using in-lab polysomnography, with two different cut points of severity, an apnea-hypopnea index ≥ 5 defined mild or worse sleep-disordered breathing and an apnea-hypoponea index ≥ 15 defined moderate or worse sleep-disordered breathing. Alcohol

consumption was determined by questionnaire. The authors found that in comparison to men who consumed less alcohol, for each increment of one drink per day, men who consumed more alcohol had 25% greater odds of mild or worse sleep-disordered breathing. However, this increase in risk was not seen among the women. Interestingly in this Cohort, there were differences among the genders in the type of alcohol consumed. Beer accounted for 61% of drinking amongst men vs. only 32% amongst women. In addition, beer drinkers consumed more drinks per week than wine drinkers or consumers of hard liquor. Thus, beer drinking appeared to explain most of the increased risk of sleep-disordered breathing in this study related to alcohol, although there was more statistical power to detect associations among the beer drinkers. The finding that women did not have any increased risks of sleep-disordered breathing with alcohol was discussed by the authors. One explanation was that alcohol consumption amongst women was much more limited than in the men and, thus, the power of the study was not adequate to detect any association. Alternatively, it is possible that women may be more resistant than men to the effects of alcohol on worsening sleep-disordered breathing. Such an explanation would be consistent with other differences between men and women with respect to sleep-disordered breathing and ventilatory drive. The authors emphasize that the study controlled for several potential confounding factors, including age, cigarette smoking, body habitus, and medications. Furthermore, this data represented alcohol consumption during the entire day and not just limited to bedtime. Therefore, the authors suggest that men with sleep-disordered breathing or at risk for sleep-disordered breathing be advised to minimize alcohol consumption in general and not just at bedtime.

Another article I wish to highlight in this issue of the Journal is entitled, "Characteristics of Insomnia With Self-reported Morning and Evening Chronotypes", by Jason C. Ong, Jennifer Huang, Tracy F. Kuo and Rachel Manber from Stanford University. These authors examined the relevance of self-reported morning and evening chronotypes in a group of insomniacs presenting to a tertiary sleep disorders clinic. They studied 312 patients who underwent group cognitive behavioral therapy for treatment of insomnia. The patients were categorized as to morning, intermediate and evening chronotypes, based on scores from a morningness-eveningness composite scale. In addition to this scale, all participants also completed the Beck Depression Inventory, Functional Beliefs & Attitudes About Sleep Scale, and a one-week sleep diary. The authors found that evening chronotypes, in comparison to morning and intermediate chronotypes, reported more total sleep time, more time in bed, greater variability in the time out of bed and higher levels of distress on the Functional Beliefs & Attitudes About Sleep Scale and the Beck Depression Inventory. For example, evening chronotypes reported a total sleep time of 6.44 hours vs. 5.93 hours for the morning chronotypes and a time in bed of 8.71 hours vs. 7.94 hours for the morning chronotypes. Moreover, evening chronotypes had a mean Beck Depression Inventory score of 16. 99 v. 10.38 in the morning chronotypes. The authors emphasize that these findings have potential clinical implications for the evaluation and treatment of insomnia. They suggest that because of functional cognitions and poor sleep habits are worse among the evening chronotypes, more attention might be paid addressing these issues in the evening chronotypes. Furthermore, because there was greater night to night variability in the bedtime behavior of evening chronotypes, regulating sleep patterns might be a point of emphasis in this group of patients.

Next, we will proceed with an interview with Dr. Michael Bonnet from the Dayton Department of Veteran Affairs Medical Center in Wright State University School of Medicine, who with his co-author, Dr. Donna Arand from Wallace-Kettering Neural Science Institute, authored the paper, "EEG Arousal Norms By Age".

SQ: Dr. Bonnet, can you provide the audience with a brief summary of your findings?

MB: Yes, Stuart. In this paper, we actually were looking at EEG arousals in a wide range of people, ranging from less than 20 to individuals in their 60s to actually see if EEG arousals change as a function of age. There have been a couple of other studies that have looked at different age groups, previously, but none, I think, that really have looked at all the decades of life to see if there are significant changes. So we looked at a fairly large number of individuals in each age group to show that EEG arousals do increase as a function of age and that they sort of parallel changes that are also occurring in deep sleep and other sleep stages that are also age related.

SQ: Why do you think scoring arousals are important?

MB: That's an excellent question and one of the reasons we actually did this paper was because we recently did a review of the arousal literature. I participated in that, with other members of the AASM. So we did a systematic review of the literature to see if arousals indeed were relevant and were worth scoring and that's also going to be published in the Journal of Clinical Sleep Medicine in the real near future, I believe. We found that there is a large amount of data showing that there is a relationship between arousals and daytime function. There are a number of empirical studies that have caused arousals during the night and, when you do that kind o f paradigm, the findings that have a number of arousals per hour increases, the subjects become increasingly sleepy during the day. We think that's probably the underlying mechanism of why patients with sleep apnea, for example, become significantly sleepy because they have lots and lots of EEG arousals during the night. We think there probably also is a relationship between arousals and other sleep pathology that produce sleepiness, like periodic limb movements. So the idea is that when sleep medicine professionals are evaluating patients who are sleepy, it is also important to look at the number of arousals that they have, in hopes that the therapy that they are using is going to reduce those arousals and it should improve their level of daytime alertness. The problem with that is that there aren't really good norms out there so the idea here was to actually start to develop some age-related norms of arousals in people who weren't sleepy during the day so the clinicians will know when they are looking abnormal numbers of arousals so that they have an idea that they have a pathology and when they do see those patients they have an idea of when those patients have returned to normal again.

SQ: Well that's very good. I just might add for the listeners that the article to which Dr. Bonnet refers has been published in the March 2007, special issue of the Journal. So, Mike, how do your findings compare with other studies that have quantified arousals in the normal population?

MB: As I have referred to before, there have been between three or four other studies that have published norms and in either one age group or two age groups or in a broader range of individuals. In the paper, I actually plot some of those data. I think

that the gratifying thing is that I think our data agrees actually really well with previous studies at various levels. I think the major other study that had some age dispersion in their subjects found a kind of a similar increase with age that was kind of parallel to the increase that we found in our paper. It was an arousal or so an hour off, but that's actually very close as these things go. So I think that the data that we plotted was generally pretty consistent with what the newer data that we have presented. I think there is a fairly consistent idea now that what arousal levels are at various decades, in addition to the fact that they are going to increase with age.

SQ: You may have answered this question already, to some extent, but what implications do your findings have for the sleep medicine practitioner?

MB: My hope is that I think the real reason behind this paper was that we are going to give the practitioner a new tool. The tool is that when they have patients that come and see them and there's a question about whether they're having more arousals during their sleep than they should have, they will actually have a norm to look at now to judge those patients by. It may help in some more unusual patients. Obviously, someone who already has sleep apnea, you're going to worry about treating the apnea and the arousals should go away. But some patients, for example, who may upper airway resistance syndrome may have lots of arousals but it may not be apparent that they really are having a significant respiratory problem and if you look at their arousals, compared to the norms, they might be elevated. That might give you a hint that that might be a place where you might want to proceed with treatment. There are some other medical problems, rheumatoid arthritis for example, where there might be a large number of arousals and this will help identify those patients compared to a normal group. This may then allow practitioners to institute treatment, in the hope that their arousals will come down some. Again, that should give people an idea of when they have normal numbers of arousals, as well.

SQ: What additional research needs to be done in this area?

MB: Actually, this is an area where I think a lot of research does need to be done. The major area that I have been trying to encourage people for a long time is to actually understand the neural physiology of these brief arousals. We as sleep people, knew a lot about sleep but I think we know relatively little about the process of arousals. There are a couple of groups that are just starting to look at this now. There is actually going to be a symposium on this at the sleep meeting in Minneapolis this summer to try and understand what actually happens in one of these arousals, how that relates to the sleep process, how that disturbs the sleep process. I think that's going to tell us a lot about how sleep works when we get a little bit further down the line. The other area of research that is already ongoing is looking for other correlates of arousal. We primarily have scored these by changes in EEG frequencies but when you have an EEG arousal there are also physiological changes, increases in heart rate, blood pressure and several other physiological measures. There is a lot of active research now trying to determine if just those changes, those physiological changes, changes in heart rate, are actually the same as the EEG arousals that we have looked at in this paper. I think that they are probably not but I think that that is something that I think is an active area of research still. I think that trying to differentiate or understand how EEG and how changes in heart rate, for example, are different from EEG arousals I think is going to tell us more about the underlying physiology of sleep.

SQ: Thank you very much. This has been Dr. Michael Bonnet, answering questions regarding his paper, "EEG Arousal Norms By Age", which has been published in this issue of the Journal of Clinical Sleep Medicine. Thank you again, Dr. Bonnet.

MB: Great. Thanks, Stuart.