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Editorial

OSAS, Sleep, Dreams and Metabolic Disorders. Is there enough Light on this Array?

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Editorial

The role of sleep on human health has been largely underplayed till the 60' when the first papers appeared, but the true advancement in this field came at the end of the past century when the connection with most metabolic and endocrine disorders was made with the pioneering works of Spiegel et al [1].

After a period of sleep restriction the authors observed disruption of the secretory pattern of many hormones and a reduction of the peripheral Insulin sensitivity. Of even more interest was the finding of a reduction of the first peak of Insulin secretion in young non diabetic individuals. The reduction of this peak is one of the first manifestations of diabetes, even in predisposed individuals before the full blown picture of the disease. This study however was aimed at the effects of sleep shortening, not to the role of the interruptions, and the subjects under study were not affected by OSAS. On the other hand many independent studies have explored the role of Sleep Disturbance (SD) on the control of glucose metabolism. One of the most interesting is by Punjabi and Beamer [2]. These authors studied the effect of Sleep Disordered Breathing in 118 non diabetic subjects on many parameters of Glucose Metabolism using Polysomnography and the Frequently Sampled Intravenous Glucose Tolerance Test (FSIGTT) according to the Minimal Model Technique of Bergman [3]. In this paper the Authors classify SDB according to the Apnea-Hypopnea Index and report a reduction in insulin sensitivity of 44%, 36.5% and 27% according to the a classification of SDB as severe, moderate and mild, independent of sex, age and degree of obesity. This Study has many merits, but using the Apnea-hypopnea index as a proxy for Sleep-Disturbed Breathing does not help shed light on the intricacies of the matter. Sleep disordered breathing is an umbrella term of which OSAS, as evaluated with the Apnea-Hypopnea Index is just one component, although the most relevant. A more appropriate term for the patients of this study could be Sleep Related breathing Disorders (SRBD), as reported in the CD-10 diagnostic coding. Thus, from these two landmark studies we know that sleep restriction and OSA cause metabolic disturbances. What is the connection between them? Which one has the most relevant effect? Are they additive? The two phenomena are really interchanged. We attempted to have some insight into this relationship with a study on 24 noninsulin treated subjects with Type 2 Diabetes Mellitus (14 males, 10 females) who were put on Continuous Glucose Monitoring (CGM) for 7 days and an ambulatory Polysomnographic recording on the 3rd day, when the Monitor was stabilized [4]. The main focus of the study was on one parameter of glucose control, the "glucose variability", which is most probably one of the major culprits of cardiovascular disease and death [5,6]. We tried to obtain an answer to two questions, (1) if there is any relationship of OSAS and SD with Glucose variability, and this was the case according to the highly significant negative correlation with the time spent in REM sleep and the negative correlation with the severity of the OSA indexes. (2) Which is the most powerful driver of glucose variability, OSAS or the sleep disturbance? According to the data when the Glucose variability is low the effect of REM sleep seems to prevail, while as the Glucose variability becomes more marked the effect of OSAS seems to prevail. Although the data should be confirmed with a more numerous study, the possibility of an additive effect seems likely, with severe OSAS added upon the stressful SD to increase the glucose variability. This however leaves many unanswered questions. Is the same tenet also true for the other parameters of glucose control, like HbA1c, fasting blood glucose, postprandial blood glucose? What maybe the effect on diabetes micro and micro complications?.

SD has a disruptive role, as proven by the fore mentioned studies, and can probably interfere negatively on metabolic functions, so we should look for other causes of interrupted/short sleep than OSAS, like prostate hypertrophy, environmental causes like noise, allergic conditions, a restless partner, child, pet, whenever we are investigating a subject for prevention or cure of a metabolic disorder. If the cause of DS is OSAS we should expect a more severe condition and be more aggressive. And what if a subject is a snorer without OSAS? Snoring has been traditionally treated at home and seldom mentioned at the physician consultation. Home remedies are commonplace, like having a bowl of vinegar on the bedside table, nudging the partner, blowing to induce him/her to change position, sewing a tennis ball in the pajama to force the sleeping person to lie on one side. Doubtless snoring is a marker of disturbed breathing, but there is no proof that this can disrupt the sleep pattern, and less so it can cause metabolic derangements.

We are now ready to accept that OSAS is a cause of some health problems, like diabetes, obesity [7], hypertension [8], metabolic syndrome [9] and serious car crashes [10] or other accidents due to reduced attention, but does this negative impact on the organism reach up to causing death? There are some proofs that OSAS per se can cause CV disease and death. The Busselton study [11] of 295 non diabetic subjects reported a Risk Ratio for all cause mortality 6.24 (p=0.002) in subjects with moderate/severe sleep apnea. The Wisconsin Sleep Cohort Study [12] demonstrated that the adjusted HR for all cause mortality with severe Sleep Disordered Breathing was 3.8 (1.6-9.0). On the basis of these data we hypothesized that OSAS may add a serious burden on the cardiovascular death rate in diabetic patients. We proposed a "three steps route to premature

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death [13]". 1-OSAS precipitates or worsen obesity, Diabetes and hypertension; 2-once diabetes is manifest worsens the HbA1c, and as we demonstrated increases glucose variability; the third step occurs when an OSAS episode in a diabetic with already established vascular damage abruptly reduces the Oxygen supply causing arrhythmias or vascular occlusion in any district. This dramatic event has been demonstrated also for the non diabetic subjects [14], but due to the precocious and extensive vascular damage may probably occur more frequently in the diabetics.

Although there is now much more attention to the presence of OSAS other aspects of the disturbance are still to be evaluated. Although we have some insight the different effect of OSAS and sleep disturbance are not very clear under different pathophysiological conditions. Furthermore we do not know if the respiratory/sleep disturbance has a threshold of risk, if the episodes occurring during the different phases of sleep have a different health impact, if the duration has a role, if the recovery phase may be even more dangerous [15], if there is any interaction with other diseases. There is a long way before we can state we know what really is going on through the night.

References

1. Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. Lancet. 1999; 354: 1435-1439.
2. Punjabi NM, Beamer BA. Alterations in Glucose Disposal in Sleep-disordered Breathing. Am J Respir Crit Care Med. 2009; 179: 235-240.
3. Pacini G, Bergman R. MINMOD: A computer program to calculate insulin sensitivity and pancreatic responsively from the frequently sampled intravenous glucose tolerance test. Comput Methods Programs Biomed. 1986; 23: 113-122.
4. Tatti P, Strollo F, Passali D. Sleep Disturbances and OSAS have an additive role in glucose variability. Endocr Disorders. 2014; 1: 1-4.
5. De Vries JH. Glucose variability: where it is important and how to measure it. Diabetes. 2013; 62: 1405-1408.
6. Bergenstal RM. Glycemic Variability and Diabetes Complications: Does It Matter? Simply Put, There Are Better Glycemic Markers! Diabetes Care. 2015; 38: 1615-1621.
7. Romero-Corral A, Caples SM, Lopez-Jimenez F, Somers VK. Interactions between obesity and obstructive sleep apnea: implications for treatment. Chest. 2010; 137: 711-719.
8. Zhang W, Si LY. Obstructive Sleep Apnea Syndrome (OSAS) and Hypertension: Pathogenetic Mechanisms and Possible Therapeutic Approaches. Ups J Med Sci. 2012; 117: 370-382.
9. Calvin AD, Albuquerque FN, Lopez-Jimenez F, Somers VK. Obstructive sleep apnea, inflammation, and the metabolic syndrome. Metab Syndr Relat Disord. 2009; 7: 271-278.
10. Tregear S, Reston J, Schoelles K, Phillips B. Obstructive sleep apnea and risk of motor vehicle crash: systematic review and meta-analysis. J Clin Sleep Med. 2009; 5: 573-581.
11. Marshall NS, Wong KK, Liu PY, Cullen SR, Knuiman MW, Grunstein RR. Sleep apnea as an independent risk factor for all-cause mortality: the Busselton Health Study. Sleep. 2008; 31: 1079-1085.
12. Young T, Finn L, Peppard PE, Szklo-Coxe M, Austin D, Nieto FJ, et al. Sleep disordered breathing and mortality: eighteen-year follow-up of the Wisconsin sleep cohort. Sleep. 2008; 31: 1071-1078.
13. Pack AI, Platt AB, Pien GW. Does untreated obstructive sleep apnea lead to death? A commentary on Young et al. Sleep 2008; 31:1071-1078.
14. Tatti P, Strollo F, Politi L, Passali D. Why OSAS may be regarded as the real hidden killer in diabetes. J Endocrinol Diab. 2014; 1: 6.
15. Gami AS, Olson EJ, Shen WK, Wright RS, Ballman KV, Hodge DO, et al. Obstructive sleep apnea and the risk of sudden cardiac death: a longitudinal study of 10,701 adults. J Am Coll Cardiol. 2013; 62: 610-616.

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