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Editorial

The Role of OSAS on Metabolic Disorders

Patrizio Tatti*

Department of Endocrinology and Diabetes, ASL RMH-Roma, Italy

***Corresponding author**

Patrizio Tatti, Director, Endocrinology and Diabetes Unit, ASL RMH, Via Degli Scipioni 267- 00192, Roma, Italy, Tel:39 069 327 3094; E-mail: info@patriziotatti.it

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Obstructive Sleep Apnea (OSAS) is a frequent disorder in the general population, and even more so among diabetics, with incidence values ranging from 17[1] to 48% [2], although these figures are probably an underestimation. The disorder has been always considered a boring affection and easily dismissed. Patients themselves often omit mentioning this affliction to the doctor. Only in the extreme cases the Continuous Positive Air Pressure device (CPAP) is used, with scant results, mostly due to noncompliance. Surgical correction of the upper airway abnormalities is at best only partially effective.

Recent research puts this disorder in a quite different light. Breathing is a physiologic function that follows a circadian rhythm and is strictly related to the oscillatory pattern of the cardiovascular system. The heart rate is synchronized with the respiratory rate and the same is for the blood pressure changes. Furthermore the breathing function is capable of an immediate answer to cope with the different external circumstances that demand more or less oxygen delivery. Although not immediately evident, any system in the body requires oxygen, and all the metabolic functions depend strictly on oxygen to work. So it is no wonder that the respiratory function and the attendant oxygen delivery have an impact on blood glucose metabolism. In this line of thought a pioneering work [3] showed that OSAS disrupts blood glucose metabolism, insulin, TSH and Cortisol secretory pattern. Subsequent work confirmed these results, showing that the presence of OSAS is associated with high HbA1c values. A polysomnographyc study of 60 consecutive diabetic patients demonstrated that the severity of OSAS is inversely proportional to HbA1c level. [4] Furthermore a questionnaire based cross sectional analysis of more than 2500 non diabetic subjects found that OSAS was associated with high prevalence of prediabetes and incident diabetes, irrespective of obesity.[5] OSAS disrupts sleep, and many critical physiologic functions are apparently caused by short / disturbed sleep. Foremost among these is appetite. A recent met analysis demonstrated that adults who sleep less than 5 hours have 60% increase in the risk of obesity[6] Due to the well known effects of obesity on glucose metabolism it is clear that with this mechanism sleep disturbances can worsen blood glucose control. Sleep exerts its effects on appetite disrupting the secretion of Leptin, Ghrelin and other less well studied hormones in nondiabetic individuals[7]or inducing Leptin resistance in the diabetic population[8,9]. This condition is self perpetuating because the excess fat accumulates in the respiratory muscles, in the retropharyngeal space, in the abdomen, limiting the excursion of the diaphragm, thus further reducing the respiratory space, and increasing the burden of OSAS, which, in turn, impacts negatively on sleep. The accumulation of fat in the abdominal cavity causes increased secretion of inflammatory kinins and contributes to atherosclerosis [10]. A consistent positive linear correlation between visceral fat and one of the main indexes of OSA, the Apnea: Hypopnoea Index (AHI) has been demonstrated. [11] OSAS itself seems also capable of increasing the blood levels of the proinflammatory C/reactive protein, thus adding to the diffuse atherosclerotic damage [12]. On the basis of these data and others a critical role for OSAS in the high cardiovascular death rate of diabetics has been recently hypothesized. In brief OSAS and the attendant sleep disturbance may predispose to the appearance of Obesity and Diabetes in genetically predisposed individuals, worsen the inflammatory condition caused by these alterations, and precipitate a vascular catastrophe, depriving of oxygen for a critical time this atherosclerosis – prone individuals. [13].

There are few doubts on the role of OSAS on blood glucose metabolism, but the strict relationship of this respiratory disturbance with the sleep pattern is a matter of confusion. Sleep architecture is deeply disturbed in diabetes, as demonstrated by a large retrospective study.[14] An elegant study on 11 young men who underwent a period of sleep restriction to 4 hours and a subsequent recovery period of 12 hours, showed that, during sleep extension, Glucose Tolerance, Acute Insulin Response to glucose (AIR), Glucose Effectiveness (the capacity of glucose to enhance its own cellular uptake and suppress endogenous glucose production independent of insulin), and Insulin Sensitivity were significantly improved versus the sleep restriction period[15]. Another more recent study with selective suppression of the slow wave sleep with acoustic stimuli, without sleep interruption, confirmed this alteration [16] of great relevance is the increase in the AIR to glucose obtained improving the sleep pattern, because this response is characteristically suppressed in diabetes and prediabetes.

We definitely know that the unit OSAS / Sleep disruption affects the metabolic and cardiovascular environment, but there are many aspects still to be discovered. Is the altered pattern of sleep or the oxygen deficiency of OSAS the true culprit of these alterations in blood glucose control? A limited answer comes from a recently published study of 97 patients with type 2

diabetes treated with diet, metformin, or gliptins. In this study the day to day variability of the fasting blood glucose (fBGv) was evaluated over a period of seven days, and the respiratory pattern was studied for one night during this period. In these subjects the indexes of respiratory disturbance and the number of awakenings were strongly associated with the fBGv. However when the variability was mild the number of awakenings, a proxy for disruption of the sleep pattern, appeared to be the main driver of this condition. [17]

Other aspects of OSAS and the sleep pattern on glucose metabolism, like depth, duration, frequency of the awakening episodes, the role of the different phases of sleep when the OSAS / awakenings events occur; the role of the time when sleeping starts; and the role of the many drugs that diabetic patients take, are worth being explored. More important, what the term "awakening" really means has yet to be made clear,: what is the role of awakening, which can happen in more or less stressful conditions, and what is the role of the duration of each episode of awakening? These data should be related to the many aspects of blood glucose physiology that we know, like fasting blood glucose, postprandial blood glucose, HbA1c values and glucose variability. It is time to shed light on this net of missing information. There is much more to study in this fascinating more holistic approach to the derangements of metabolism. We hope that the pages of the Journal will host additional data to help "see through the glass."

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