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Sleep disordered breathing may not be an independent risk factor for diabetes, but diabetes may contribute to the occurrence of periodic breathing in sleep.

Sanders MH, **Givelber R**.

Division of Pulmonary, Allergy and Critical Care Medicine, University of Pittsburgh School of Medicine, Pittsburgh, PA, USA. sandersmh@msx.upmc.edu

OBJECTIVE(S): (a) To determine if self-reported diabetes mellitus is independently associated with sleep-disordered breathing (SDB); (b) to determine if diabetes mellitus is specifically associated with central sleep apnea including periodic breathing (Cheyne-Stokes breathing pattern) during sleep. STUDY POPULATION: The study population reflected participants in the on-going Sleep Heart Health Study (SHHS). Analyses were conducted utilizing data obtained from 4872 SHHS participants without prevalent cardiovascular disease (CVD) and 1002 participants with self-reported CVD, defined as hospitalization for non-fatal coronary heart disease, congestive heart failure, myocardial infarction, coronary artery bypass graft, and stroke. METHODS: SHHS methodologies have been previously reported and include performance of overnight, in-home polysomnography (PSG), which recorded variables reflecting sleep architecture and breathing, permitting identification of obstructive and central apneas, hypopneas, periodic breathing and oxyhemoglobin saturation (SpO(2)). Anthropomorphic metrics as well as systemic blood pressure measurements were obtained at the time of PSG. Other health data were available from questionnaires and the data sets of the parent cohorts from whom SHHS participants were recruited. The investigators assessed and compared breathing parameters, sleep architecture and CVD variables in diabetic and non-diabetic participants. The relationships between diabetes and the various study parameters, independent of potential confounders, were examined by multivariable modeling. Linear regression modeling was employed to examine the relationship between continuously distributed variables such as respiratory disturbance index log (RDI). The relationships between diabetes and dichotomous outcome variables such as central apnea index (CAI), obstructive apnea index (OAI), periodic breathing and the percentage of time spent at various levels below SpO(2) 90% were examined by the logistic regression model. Age, gender, race, BMI and neck circumference were forced into all multivariable analyses since these factors are associated with both diabetes mellitus and SDB. RESULTS: The investigators reported that the prevalence of CVD risk factors including increased BMI, waist circumference, neck circumference, triglycerides, reduced HDL cholesterol and hypertension was greater in diabetic than non-diabetic participants. Native Americans represented a disproportionately high percentage of the diabetic population. Unadjusted data obtained from participants without prevalent CVD indicated that the mean RDI was higher in the diabetic participants. Moreover, there was a greater percentage of diabetic participants in the higher RDI categories (e.g. 23.8% of the 470 diabetics and 15.6% of the 4402 non-diabetics had RDI>15, P<0.001). Similarly, the unadjusted data indicated that a significantly greater proportion of the diabetic participants spent >5% and >10% of sleep time below SpO(2) 90%, compared with the non-diabetic participants. The unadjusted data from participants without prevalent CVD indicated that the diabetic and non-diabetic participants did not differ with regard to distribution by category of OAI severity (e.g. > or =2 events/h, > or =3 events/h, or > or =4 events/h). On the other hand, although the prevalence of central apneas was low, a significantly greater proportion

of diabetic participants were in the CAI categories (> or =2 events/h and > or =3 events/h) than non-diabetic subjects. There was no difference between diabetic and non-diabetic individuals with regard to CAI prevalence in the > or =4 events/h category. Of note, a greater percentage of diabetic patients exhibited periodic breathing (3.8% vs. 1.8%, diabetic and non-diabetic participants, respectively, P=0.002). Repeating the above analyses with inclusion of the participants with prevalent CVD did not change these relationships, and in fact, the differences between diabetic and non-diabetic participants with respect to central events and periodic breathing became more evident (the data forre evident (the data for this were not provided in the paper). Linear regression analyses demonstrated that BMI, age and male gender were independently related to increased RDI among participants without prevalent CVD. Furthermore, after adjusting for age, gender, race, BMI and neck circumference, there was no difference in geometric mean RDI between the diabetic and non-diabetic participants. The adjusted odds of having RDI> or =15 and the adjusted odds for spending> or =5% or > or =10% of sleep time with SpO(2) <90% did not differ between the diabetic and non-diabetic individuals. The investigators also examined sleep architecture in the study cohort. There were no differences between the diabetic and non-diabetic groups with regard to the adjusted proportion of time spent in non-REM sleep stages, although the mean percent time spent in REM sleep was 1.1% less in the diabetic individuals. The findings were the same with or without inclusion of participants with known CVD. Even after adjustment for potential confounders, in a sample without or with prevalent CVD, diabetic participants had increased adjusted odds for periodic breathing odds ratio (1.8, 95% confidence interval (CI) with a range of 1.02--3.15 in diabetic participants without prevalent CVD vs. 1.74, 95% CI with a range of 1.16--2.62 in diabetic participants with prevalent CVD). There was a suggestion of increased odds for CAI in diabetic subjects when analyzing populations with and without prevalent CVD. CONCLUSION: The authors concluded that diabetes mellitus is associated with sleep apnea but that this association is largely explained by risk factors in common for both disorders, most notably obesity. After adjusting for confounding factors there was no difference between diabetic and non-diabetic participants with regard to obstructive events. However, even after adjusting for potential confounders, there was a greater prevalence of periodic breathing in diabetic subjects. Although not reaching statistical significance, there was a suggestion of an increased prevalence of central events in the diabetic population, particularly when the sample included participants with known CVD. The investigators believed it unlikely that the findings were attributable to underlying congestive heart failure in as much as the diabetic subjects without prevalent CVD exhibited increased prevalence of periodic breathing and possibly increased central events. The authors proposed that diabetes mellitus might be a cause of SDB, mediated through autonomic neuropathy that may alter ventilatory control mechanisms. In this context, the authors commented that autonomic neuropathy may cause perturbations in ventilatory control by altering chemoreceptor gain or altering cardiovascular function (although the authors discounted underlying congestive heart failure as an explanation for the higher prevalence of periodic breathing in diabetic participants). To reinforce their conclusions, the authors cited the literature indicating increased prevalence of sleep apnea in diabetic patients with autonomic dysfunction, as well as the association between Shy--Drager syndrome, in which autonomic insufficiency is a constitutive element, and central sleep apnea.

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