anthropometric (body mass index [BMI], neck circumference), comorbidities (atrial fibrillation, coronary artery disease, diabetes, hypertension, hyperlipidemia), echocardiographic and sleep-disordered breathing (apnea-hypopnea index [AHI], peak end-tidal CO2 [etCO2]) variables retrospectively examined. The echocardiographic visit closest to polysomnogram within two years was selected with missing values filled by available values within 6 months. Linear regression assessed the relationship of BMI, AHI, and etCO2 with left ventricular mass index (LVMI) after adjustment of demographics and comorbidities. Echocardiographic measures were logarithm transformed before regression analysis. Coefficients and 95% confidence intervals (CI) were calculated by exponential transformation. The analysis was performed based on an overall significance level of 0.05 using SAS software (version 9.4, Cary, NC).

Results: The total of 832 patients had 24% males, mean age 48.8±12, 60% white, and BMI:49.4±9.5kg/m2. Ejection fraction (%) was 60.0±7.0, and LVMI (g/m2): 80.9±23.7. In adjusted models, LVMI decreased by 2.1% for each 5kg/m2 increase in BMI (coefficient=0.979, 95%CI 0.961–0.997, p=0.022) and increased by 4.3% for each 5 mmHg increase in etCO2 (coefficient=1.043, 95%CI 1.013–1.073, p=0.005). Without adjustment, patients with AHI ≥ 5 had 15.3% higher LVMI than non-OSA group (coefficient=1.153, 95%CI 1.034–1.286, p=0.011) and moderate/severe OSA was associated with a 7.6% higher LVMI than those with AHI<15 (coefficient 1.076, 95%CI 1.003–1.153, p=0.040), but not statistically significant after adjustment.

Conclusion: In obese patients, nocturnal hypoventilation rather than obesity may have adverse influences on left ventricular morphology. Future studies should focus on clarifying whether obesity is truly protective in terms of LV mass, i.e. reflective of paradox versus a product of bias. The potential benefit of identifying/treating SDB in bariatric surgery candidates to mitigate cardiovascular risk also deserves further investigation.

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ASSOCIATION BETWEEN NOCTURNAL HYPOXEMIC BURDEN AND GLUCOSE METABOLISM

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Introduction: To evaluate the association between a novel integrated event-based and hypoxemia-based parameter of polysomnography (PSG), hypoxemic load or HL100, and fasting blood glucose (FBG) and hemoglobin A1c (HbA1c) levels.

Methods: Adult patients, who underwent an in-lab PSG at University of Iowa Hospitals and Clinics with FBG or HbA1c levels were included. Event-based parameter and hypoxemia-based parameter data were derived. HL100, defined as an integrated area of desaturation under 100% oxygen saturation curve during the total sleep time divided by the total sleep time, was calculated by Python software version 3.8.5. Demographic data and glycemic parameters within 1 year prior to PSG(FBG and HbA1c) were retrieved from chart review Spearman correlation analysis and stepwise backward regression analysis were performed to determine independent predictors of FBG and HbA1c levels.

Results: Of the 467 patients underwent an in-lab PSG, 385 had FBG levels and 239 had HbA1c levels. All event-based and hypoxemia-based parameter; including HL100, were significantly correlated to FBG and HbA1c levels. Stepwise backward regression analyses, adjusting for age, sex, body mass index and diabetes status, revealed that log HL100 was significantly related to FBG (B=20.8, p=0.015), and log oxygen desaturation index was found related to HbA1c levels (B=0.273, p=0.037). Other parameters (e.g. apnea hypopnea index, minimum oxygen saturation) were not independently associated with glycemic parameters.

Conclusion: HL100 showed a significant positive correlation with FBG and HbA1c levels and only log HL100 was an independent predictor for FBG levels. This might imply that any degree of desaturation below 100% could result in adverse glucose metabolism. HL100 might be useful for interpretation of sleep studies, risk stratification and patient management purposes in the future.

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THE RELATIONSHIP BETWEEN SLEEP DISORDERED BREATHING, MARKERS OF VENTRICULAR REPOLARIZATION AND CARDIOVASCULAR MORTALITY

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Introduction: Sleep disordered breathing (SDB) is associated with increased mortality. Obstructive apneas/hypopneas have been associated with an increase in both QTc duration and QT variability. These markers of ventricular repolarization are associated with arrhythmias and death. It is unknown whether SDB-related QTc changes are responsible for the relationship between QTc/QT variability and cardiovascular death (CVD).

Methods: From the Sleep Heart Health Study, we randomly selected 200 subjects in each of four groups based on overall apnea/hypopnea index: those with no SDB and those in either, mild, moderate or severe SDB at baseline, matched for gender, age and BMI. Respiratory-related channels and electrocardiograms (ECG) from each polysomnography were analyzed. QTc was calculated using Bazett's heart rate correction. The following measures of QT variability were obtained: i) standard deviation of QT intervals (SDQT) at 1- and 5-minute intervals and ii) short-term interval QT variability (STVQT) at 5-minute intervals. Cox proportional hazards regression models were used to evaluate potential predictors of CVD.

Results: Twenty-nine subjects were excluded either due to missing data or low quality ECG. The 771 subjects included were 68±10 years of age, half were female. During follow-up, 220 subjects (28.5%) died of CVD among whom, 67 (30.5%) had comorbid severe SDB, 45 (20.5%) had no SDB, and the remaining CVD deaths had mild (47, 21.4%) and moderate 61 (27.7%) SDB. The CVD patients were more likely to be older(p<0.001), hypertensive (p<0.001), diabetic(p<0.001), and had increased SDQT(p<0.001), STVQT(p<0.001) and QTc (0.017). After adjusting for covariates, the presence of mild (p=0.562), moderate(p=0.439) and severe SDB (p=0.912) did not moderate the association between QTc prolongation and CVD. Additionally, mild (p=0.486), moderate(p=0.478) and severe SDB (p=0.849) did not moderate the association between SDQT and CVD. Similarly, mild (p=0.144), moderate(p=0.594) and severe SDB (p=0.508) did not moderate the association between STVQT and CVD. However, QTc, SDQT, STVQT, mild and severe SDB were individually associated with CVD (p=0.004, 0.000, 0.000, 0.014, 0.022, respectively).

Conclusion: SDB was not a factor in the relationship between QTc prolongation/QT variability and CVD.

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THE ASSOCIATION OF QTC AND QT VARIABILITY WITH SEVERITY OF SLEEP DISORDERED BREATHING

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Introduction: The apneas and hypopneas that characterize sleep-disordered breathing (SDB) are associated with QTc prolongation and increased QT variability. There have been mixed results as to whether QTc and QT variability increase with increasing SDB severity. This study assesses whether QTc prolongation and QT variability are likely to increase with increasing severity of SDB in a large multi-center cohort.

Methods: 200 subjects with no SDB and approximately 600 with three levels of SDB (mild, moderate, severe) were randomly selected from the Sleep Heart Health study and matched by age, gender and BMI. SDB was defined as an apnea/hypopnea index ≥5. Respiratory and electrocardiograms (ECG) signals from polysomnography studies were analyzed. Bazett's heart rate correction was used to calculate QTc. QT variability was measured as standard deviation of QT intervals (SDQT) and short-term interval QT variability (STVQT), at 5-minute intervals. Subjects were excluded if there were missing data or low-quality ECG.

Results: Seven hundred and seventy-one subjects (age 68±10 years, 51% female, 92% Caucasian) were included. One hundred and sixty-five subjects had no SDB, 235 mild, 195 moderate and 176 had severe SDB. The mean (SD) QTc was 422(29), 411(26), 419 (34) and 418 (36) ms for the no SDB, mild, moderate, and severe SDB groups, respectively (p=0.017). The mean (SD) STVQT was 7 (9), 11 (16), 8 (9) and 9 (11) for the no SDB, mild, moderate severe SDB groups, respectively (p=<0.001). The mean (SD) STVQT was 3 (2), 4 (4), 4 (3) and 4(4) for the no SDB, mild, moderate severe SDB groups, respectively (p=<0.001). There was no statistically linear relationship between QT prolongation or QT variability and SBD severity.

Conclusion: QTc duration and QT variability were not increased with SDB severity.

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CARDIOVASCULAR AND METABOLIC RISK IN PATIENTS WITH SUSPECTED COMORBID INSOMNIA AND OBSTRUCTIVE SLEEP APNEA (COMISA)

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Introduction: Only few studies looked for a possible association of cardiovascular disorders (CVD), in comorbid insomnia with obstructive sleep apnea (COMISA) even though this is a relevant topic in order to prevent one of the major causes of morbimortality. The present study aimed to investigate the association of insomnia symptoms in patients at risk for obstructive sleep apnea in terms of prevalence and clinical interactions and to evaluate the risk of CVD in patients with a risk for COMISA.

Methods: This is a cross-sectional study. All medical records with data such as age, sex, height, weight and BMI, time to sleep, time to wake up, total sleep time, the Epworth Sleepiness Scale (ESS), STOP-BANG Questionnaires were studied. Insomnia and comorbidities were also investigated, and the patientsanswered yes or no to systemic arterial hypertension, diabetes, CVD.

Results: 685 patients were enrolled on the present study. We observed that the mild, moderate, and high risk for COMISA presented progressively increasing levels for the frequency of hypertension, diabetes, and CVD. A binary logistic regression was performed to assess whether risk for COMISA could be a predictor for CVD, and it was found that the model containing risk for COMISA was statistically significant: [x2(1)=5.273;p<0.021, R2 Negelkerke=0.014]. Risk for COMISA presented itself as a significant predictor for CVD (OR=1.672; 95% CI=1.079–2.592).

Conclusion: There was an increased frequency of associated comorbidities such as CVD, systemic arterial hypertension, and diabetes, according to the mild, moderate, or high risk. These findings highlight the need for a cardiometabolic evaluation in patients with this comorbid condition which may impact prognosis and therapeutic success.

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