Introduction: Both short sleep duration and obstructive sleep apnea (OSA) seem to be associated with insulin resistance. However, the majority of previous studies addressing the relationship between OSA and insulin resistance did not evaluate short sleep duration, and vice versa. In this study, we used a large-scale hospital-based cross-sectional dataset, including 5,447 participants, to examine 1) whether objectively measured short sleep duration and OSA are independently associated with insulin resistance, and 2) whether the presence of OSA modulates the association between sleep duration and insulin resistance.
Methods: Participants were consecutively enrolled from our sleep center during the period from 2007 to 2017. The index of homeostasis model assessment insulin resistance (HOMA-IR) was calculated from insulin and glucose. Sleep duration was determined by standard polysomnography. The associations between sleep duration and insulin resistance were estimated by logistic regression analyses.
Results: A total of 5,447 participants (4507 OSA and 940 primary snorers) were included in the study. In comparison to primary snorers, OSA combined with extremely short sleep duration (< 5 hours) increased the risk of insulin resistance by 34% (OR, 1.34; 95% CI, 1.01-1.77) after adjusting for confounding factors that are frequently associated with insulin resistance and OSA. In subgroup analysis stratified by sleep duration, the risk of insulin resistance in patients with a short sleep duration (5-6 hours or < 5 hours) was increased in those with OSA compared to primary snorers, but not in the other three sleep duration groups (6 - 7, 7 - 8, and > 8 hours).
Conclusion: OSA, but not short sleep duration, was independently associated with insulin resistance. It is worth noting that OSA combined with extremely short sleep duration showed a greater detrimental effect than OSA itself with regard to insulin resistance.
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ASSESSMENT OF SOFT PALATE MUSCLE FATIGUE AND ITS EFFECT ON VELOPHARYNGEAL UPPER AIRWAY (UA) MECHANICAL PROPERTIES

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Introduction: Soft palate muscles are crucial in the maintenance of UA patency. This study aimed to investigate the fatigability of soft palate muscles and to quantify its effects on velopharyngeal UA dynamic properties in OSA patients and control subjects.
Methods: 8 control (AHI ≤ 10 /h), 21 OSA patients (13 with mild/moderate disease: 10 /h < AHI ≤ 20 /h and 8 with moderate/severe: AHI > 20/h) were included in the study. Subjects were asked to develop repetitive intra-oral positive pressure during cheek-bulging maneuvers while wearing a mouth piece to keep the jaw opened. Subjects were asked to develop sustained maximal bulging pressure for 5 sec every 10 sec until the peak pressure could not reach 85% of baseline maximal pressure for 2 consecutive times. UA dynamic properties were assessed by measuring instantaneous airflow and velopharyngeal pressure in response to phrenic nerve magnetic stimulation (PNMS) performed before, immediately and every 3 minutes after the fatiguing protocol for a maximum of 30 minutes' recovery time. UA closing pressure (Pcrit) was estimated by modeling the flow/pressure relationship in response to PNMS.
Results: The sex, age, BMI and the soft palate mechanical properties (including the baseline strength, endurance time, total muscle work) did not significantly differ between the 3 groups. Maximal peak bulging pressure measured using cheek-bulging maneuver significantly changed following the fatigue task ($p < 0.05$). Baseline velopharyngeal Pcrit were less negative in moderate/severe OSA group compared to mild/moderate OSA (-6.5±2.6 vs. -11.9±3.2, $p < 0.05$). In mild/moderate OSA patients, PNMS-induced drop in maximal instantaneous airflow tend to increase 3 mins after the fatiguing trial compared to baseline (22.7±21.1 $l.s^{-1}$vs. 9.6±5.8 $l.s^{-1}$, $p < 0.1$), and their Velopharyngeal linear resistance 3 mins after the fatiguing trial tend to be higher than the moderate/severe OSA group (3.9 ± 5.0 $cmH_2O{\cdot}l^{-1}{\cdot}s^{-1}$ vs. 1.8 ± 1.1 $cmH_2O{\cdot}l^{-1}{\cdot}s^{-1}$, $p< 0.1$).
Conclusion: The cheek-bulging maneuver could induce soft palate muscle fatigue, with no difference observed in soft palate mechanical performances among patients with different OSA severity. The fatiguing maneuver could further alter velopharyngeal UA mechanical properties in patients with mild/moderate OSA.
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HYPOXIC BURDEN AND APNEA-HYPOPNEA DURATION IN PATIENTS WITH POSITIONAL OBSTRUCTIVE SLEEP APNEA

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Introduction: Recently, the measurement of the hypoxic burden and apnea-hypopnea duration has been shown to correlate with mortality in patients with obstructive sleep apnea (OSA). We hypothesized that in patients with mild positional OSA (apnea-hypopnea index [AHI] < 5 events/hr in the non-supine position) the hypoxic burden would be increased and apnea-hypopnea duration shortened and similar to patients with non-positional OSA.
Methods: Fourteen patients with positional OSA and 24 patients non-positional OSA with similar severity of OSA based on the respiratory event index (REI) were included. All patients had a home sleep apnea test for suspected OSA. The hypoxic burden was calculated by the multiplication of REI and the mean area under the desaturation curves.
Results: Thirty-eight patients [12 (35%) males, 50±12 yrs, BMI 35±7 kg/m^2, Epworth Sleepiness Scale (ESS) 11±8, REI 10±3 events/hr, apnea-hypopnea duration 19±4 sec, mean SaO_2 94±2%, lowest SaO_2 79±8%, % total sleep time (TST) SaO_2 < 90% 11±16%, hypoxic burden 30±17 %min/hr] completed the study. Fourteen patients [9 (64%) males, 46±14 yrs, BMI 31±6 kg/m^2, ESS 7±5, REI 9±3 events/hr, mean SaO_2 94±2%, lowest SaO_2 81±6%, %TST SaO_2 < 90% 4±6%] had positional OSA (supine REI 16±7 events/hr, non-supine REI 3±1 events/hr) and 24 patients had non-positional OSA [3 (13%) males, 52±10 yrs, BMI 38±7 kg/m^2, ESS 12±9, REI 10±3 events/hr, mean SaO_2 94±2%, lowest SaO_2 77±9%, %TST SaO_2 < 90% 14±19%]. The hypoxic burden was elevated in both the positional and non-positional OSA patients with no difference between the groups (26±19 %min/hr and 32±15 %min/hr, respectively, p=0.13). The apnea-hypopnea duration was