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CPAP increases exercise tolerance in obese subjects with obstructive sleep apnea

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Summary
Obese subjects commonly suffer from exertional dyspnea and exercise intolerance. Preliminary evidence suggests that treatment with nocturnal continuous positive airway pressure (nCPAP) may improve dyspnea in obese patients with obstructive sleep apnea (OSA), but the effect on exercise tolerance is unknown. This study sought to investigate whether nCPAP improves exercise tolerance and exertional dyspnea in obese patients with OSA.

Obese patients prescribed nCPAP for moderate/severe OSA and without cardiopulmonary disease were recruited. Patients completed a constant-load exercise test and Baseline and Transitional Dyspnea Index questionnaires (BDI/TDI) at baseline and after one and three months of nCPAP. Primary outcome was change in constant-load exercise time from baseline to one and three months. Secondary outcomes included changes in isotime dyspnea, isotime leg fatigue and BDI/TDI score at one and three months.

Fifteen subjects (body mass index = 43 kg m^{-2}, apnea-hypopnea index = 49·hr^{-1}) were studied. Constant-load exercise time increased by 2.0 min (40%, $p = 0.02$) at one month and 1.8 min (36%, $p = 0.04$) at three months. At one and three months, isotime dyspnea decreased by 1.4 ($p = 0.17$) and 2 units ($p = 0.04$), and leg fatigue decreased by 1.2 ($p = 0.18$) and 2 units ($p = 0.02$), respectively. BDI/TDI scores were 2.7 ($p = 0.001$) and 4.5 points ($p < 0.001$) at one and three months. Peak oxygen consumption and static pulmonary function were unchanged.

Nocturnal CPAP improves exercise tolerance and dyspnea in obese patients with OSA. Effects on exercise time and chronic dyspnea were seen after one and three months of nCPAP, while exertional dyspnea was only improved at three months.

Abbreviations: AHI, apnea-hypopnea index; ANOVA, analysis of variance; BDI/TDI, baseline dyspnea index/transitional dyspnea index; BMI, body mass index; IC, inspiratory capacity; MRC, Medical Research Council; nCPAP, nocturnal continuous positive airway pressure; OSA, obstructive sleep apnea; SD, standard deviation; VO_2, oxygen consumption.

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Introduction

Obesity is associated with multiple medical conditions, including metabolic, cardiopulmonary and musculoskeletal diseases,[1] and has been identified as an independent risk factor for premature death.[2] Obesity is an established risk factor for obstructive sleep apnea (OSA)[3] and has a number of profound effects on the cardiac and respiratory systems. It is well documented that obesity reduces static chest wall compliance and increases airway resistance due to respiration at lung volumes closer to residual volume.[4,5] These mechanical problems are worsened in the supine position.[6] Additionally, the hypercapnic ventilatory response is reduced in obese subjects compared to non-obese subjects.[7] Obesity also independently affects cardiovascular function by elevating pulmonary artery pressure and impairing left ventricular diastolic function.[8,9] These cardiopulmonary changes may have important implications for exercise intolerance in obese individuals.

Obesity is commonly associated with both a decreased exercise capacity[10] and an increase in oxygen cost of breathing during exercise.[11] Exertional dyspnea is also increased in obese subjects[12] and is a prime determinant of functional impairment.[13] The inability to sustain exercise due to dyspnea results in worsening obesity, deconditioning, and an increased risk of cardiovascular disease.[14,15]

The effects of OSA on exercise are variable. Although reductions in aerobic capacity have not been consistently demonstrated,[16–20] patients with OSA may have impaired cardiac stroke volume, inefficient ventilation during exercise and muscle metabolic impairment due to nocturnal hypoxemia.[21–23] Chronic nocturnal continuous positive airway pressure (nCPAP) has been shown to improve exercise capacity in small studies,[24,25] but the effect on dyspnea is unknown.

Clinical observation by our group suggests that obese patients with exertional dyspnea as a presenting symptom of OSA report an improvement in dyspnea after treatment with nCPAP, without a change in body mass index (BMI) or activity level. This reduction in dyspnea may translate into improved exercise tolerance; however, to date no study has addressed this interesting question. The aim of the current study was to test the hypothesis that nCPAP would reduce exertional dyspnea and improve exercise tolerance in obese patients with OSA.

Methods

Additional detail on the methods is provided in an online data supplement.

Patients

Subjects were recruited from referrals to the Foothills Medical Centre Sleep Centre in Calgary, Alberta, Canada. Inclusion criteria were: age 18–65 years; BMI $\geq$ 30 kg m^2; a new diagnosis of moderate to severe OSA (Apnea-Hypopnea Index (AHI) $\geq$ 15 h^{-1}); decision by the patient and sleep physician to use nCPAP for treatment of OSA; and Medical Research Council (MRC) class III or greater dyspnea. Exclusion criteria were: known or suspected airways or parenchymal lung disease; cardiac disease including pulmonary hypertension; active smoking within the last 6 months; previous treatment for OSA; inability to tolerate nCPAP; another sleep disorder; inability to perform cycle ergometry; or refusal or inability to provide informed consent. All subjects signed an informed consent that had received approval from the University of Calgary Conjoint Health Research Ethics Board.

Study design

This pilot study used a prospective, single group design. At baseline, subjects completed the dyspnea and physical activity questionnaires before performing a symptom-limited incremental exercise test. On a separate day, patients underwent constant-load exercise testing. The subjects were then started on nCPAP at a pressure determined by polysomnography. After one and three months of nCPAP, patients repeated both incremental and constant-load testing on separate days and completed the dyspnea and physical activity questionnaires. Pulmonary function measurements were repeated after three months. Investigators were blind to the results of all measurements until the conclusion of the study.

Specific methodology

Incremental cardiopulmonary exercise test

Incremental cardiopulmonary exercise testing to symptom limitation was performed according to American Thoracic Society guidelines.[26]

Constant-load exercise trials

A symptom-limited constant-load exercise trial was performed at 85% of maximal workload achieved in the baseline incremental exercise trial. Exertional symptoms using the modified Borg scale,[27] and repetitive inspiratory capacity (IC) maneuvers were performed every 2 min.

Measurements of dyspnea and physical activity

The Baseline Dyspnea Index/Transitional Dyspnea Index questionnaire (BDI/TDI) was used to assess changes in chronic dyspnea. The BDI is a baseline measure of chronic dyspnea, while the TDI quantifies the change from baseline. The Godin Leisure Time Exercise questionnaire was also used to account for activity as a possible contributor to increased exercise tolerance.[28]

Outcomes

The primary outcome was the change in constant-load exercise time from baseline to one and three months after the initiation of nCPAP. Secondary outcomes included the changes in isotime exertional symptoms and chronic exertional dyspnea from baseline to one and three months. Isotime was defined as the exercise time at symptom limitation during the shortest of the three constant-load trials.

Statistical analysis

One-way repeated measures analysis of variance (ANOVA) was used to compare primary and secondary outcomes at