compared to subjects without, have lower lung volumes (upright and supine), a greater airway impedance (seen in FOT at FRC, both upright and supine), and a larger fall in their ERV on lying down. All these differences may be due to the higher BMI, and in particular differences in distribution of fat in subjects with early chronic respiratory failure. However, these obesity differences were not reflected in large differences in the AHI or ODI between the groups.

**Introduction**

Obstructive Sleep Apnea (OSA) is a condition defined by the collapse of the upper airway and cessation of respiration during sleep. The resulting hyperventilation leads to intermitent nocturnal hypoxia and increased arterial pCO2 (hypercapnia). Control of ventilation is largely dependent on two interactive pathways, the central and peripheral chemoreceptors. Both chemoreceptors respond to H+ ions liberated as a result of the presence of CO2. However, the peripheral chemoreceptors also respond to hypoxia particularly when PaO2 falls below 70 Torr. Hypoxia has a synergistic effect on the ventilatory response to CO2. Our study therefore investigated the ventilatory response to CO2 with and without the presence of moderate hypoxia to determine whether the central and/or peripheral chemoreflex response to CO2 is modified with the development of OSA.

**Methods**

The ventilatory response to CO2 amongst 33 newly diagnosed male OSA patients (AHI: 32.6 ± 24.8; age: 53.1 ± 10.3 years, BMI: 36.4 ± 6.8 kg/m²) was measured whilst breathing four different gas mixtures balanced with N₂ (Mixture 1: ambient air; mixture 2: 25% O₂/6% CO₂; mixture 3: 13% O₂; mixture 4: 13% O₂/6% CO₂). The minute ventilation of each participant, normalised by their body surface area (BSA), was recorded with subjects blinded to the order of test gases. Additionally, fasted venous blood samples were taken to assess plasma leptin concentrations.

**Results**

Mild and moderate OSA patients (AHI: 13.76 ± 7.01) revealed a significantly (p < 0.01) greater change in their ventilatory response to the hyperoxic-hypercapnic gas mixture (5.22 ± 1.95 l x min⁻¹ x BSA⁻¹) compared to severe OSA patients (AHI: 55.31 ± 18.25) (3.26 ± 1.64 l x min⁻¹ x BSA⁻¹). There was no significant change in ventilation between the hypoxic and hypoxic-hypercapnic conditions in both groups. A significant negative correlation (r = -0.39; p < 0.05) was found between AHI and ventilation change (l x min⁻¹ x BSA⁻¹) to the hyperoxic-hypercapnic gas mixture.

**Conclusion**

These findings suggest the reduced ventilatory response to hypercapnia amongst severe OSA patients is likely a result of adaptation to the central chemoreceptors.