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 hypoventilation leads to intermittent nocturnal hypoxia and increased arterial pCO₂ (hypcapnia). 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 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 hypoxic-hypercapnic gas mixture compared to severe OSA patients (AHI: 55.31 ± 18.25) (3.26 ± 1.64 l 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 min⁻¹ x BSA⁻¹) to the hypercapnic-hyperoxic gas mixture.

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

Introduction A brisk ventilatory response to carbon dioxide (CO₂) is integral to the development of central sleep disorders. Modern treatments for HF enhance cardiac function and may improve central SDB. The role of hypercapnic ventilatory responses (HCVR) in central SDB in HF patients managed according to modern guidelines is unclear. For example, adaptive servventilation (ASV), used to treat central SDB, both improves cardiac function and reduces hypercapnic ventilatory responses (HCVR), suggesting that heightened chemosensitivity in HF may relate to cardiac impairment.

Objective To test the hypothesis that there would be no difference in HCVR between optimally treated patients with HF and central SDB (HF-CSA), and those with HF alone (HF-noSDB).

Method Measurements of resting expired CO₂ (awake) and evening and morning HCVR, using the Read rebreathe technique, were undertaken in patients optimally treated for HF. Patients also underwent overnight polysomnography. Sample size calculations (using data from Javaheri, NEJM 1999) showed 10 patients were needed in each group. Statistical analyses were undertaken using SPSS. The study received ethical approval.

Results Twenty-six HF patients were studied (11 with HF-CSA: median (IQR) age 68 (58–78) and 15 with HF-noSDB: age 72 (67–78) years). Left ventricular ejection fraction was: HF-CSA 32.0 (20–40)% and HF-noSDB 40.0 (27–47)%. The apnoea hypopnoea index was: HF-CSA 14.6 (12.9–37.1)l/hr and HF-noSDB 5.0 (3.2–6.0)l/hr. The HF-CSA group had lower median resting expired CO₂ than the HF-noSDB group (end tidal CO₂: 30.6 (28.6–37.3) vs. 36.2 (35.2–40.4)mmHg, p = 0.02). There was no significant difference between the HF-CSA and HF-noSDB in evening HCVR (2.15 (1.70–2.74) vs. 1.99 (1.60–3.33)l/min/mmHg ET CO₂, p = 0.53) or morning HCVR (2.71 (1.43–4.88) vs. 2.20 (1.00–3.00)l/min/mmHg ET CO₂, p = 0.23). Resting expired CO₂ in the total study population correlated negatively with morning, but not evening, HCVR.

Conclusion The results of this small study suggest that modern HF management may have an effect on ventilatory stability via changes in HCVR. The timing of the HCVR tests may be a factor. We speculate that overnight disturbances in breathing may promote ventilatory instability in the morning, rather than the evening.
corresponding to diffusion path lengths of up to 8mm. We aimed to determine the microstructural correlates of $S_{\text{cond}}$ and $S_{\text{acin}}$ in patients with asthma, using $^3$He-MRI.

**Methods** Twenty-nine patients with asthma underwent MBW using sulphur hexafluoride as the inert tracer gas, and the parameters $S_{\text{cond}}$ and $S_{\text{acin}}$ were calculated. $^3$He-MRI was performed and the ADC was calculated at both short (14ms) and long (1.5s, 3s and 6s) timescales. $^3$He-MRI data was also fitted to a previously reported geometrical model of the acinus (Yablonskiy DA et al, J Appl Physiol. 2009;107(4):1258–65), and estimates of the alveolar duct outer radius (R) and alveolar sleeve width (h) were derived.

**Results** Correlations between MBW and $^3$He-MRI parameters are shown in Table 1. The approximate length scales probed by short and long timescale ADC are also indicated for reference. Significant positive correlations were observed between $S_{\text{acin}}$ and ADC at 14ms, 1.5s and 3s, but not 6s. In a stepwise linear regression model, ADC at 1.5s was the only significant determinant of $S_{\text{acin}}$, with a model $R^2$ of 0.334. $S_{\text{cond}}$ did not correlate significantly with any of the MRI parameters. Yablonskiy model estimates of alveolar sleeve width and alveolar duct outer radius did not correlate significantly with either $S_{\text{cond}}$ or $S_{\text{acin}}$.

**Conclusion** $S_{\text{acin}}$ in patients with asthma is associated with an elevated ADC at 1.5s, corresponding to length scales of the order of 4mm. This suggests that DCDI in asthma is associated with structural asymmetries at the level of the distal acinar airways and/or collateral ventilation between parallel intra-acinar airways.

<table>
<thead>
<tr>
<th>Abstract S120 Table 1. Correlations between multiple breath washout and $^3$He-MRI parameters</th>
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<tr>
<td>ADC 14 ms (0.5mm)</td>
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<td>ADC 1.5s (4mm)</td>
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<td>ADC 3s (5.5mm)</td>
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<td>ADC 6s (9mm)</td>
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<td>Alveolar duct outer radius (R)</td>
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<td>Alveolar sleeve width (h)</td>
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*ADC = apparent diffusion coefficient. Significant correlations are indicated *(p < 0.05) or **(p < 0.01).*

**Keywords** Bronchoalveolar lavage, COPD, MBW, $^3$He-MRI, collateral ventilation.

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**S121 ASSESSMENT OF INTERLOBAR-COLLATERAL VENTILATION PRIOR TO ENDOBRONCHIAL VALVES TREATMENT FOR SEVERE EMPHYSEMA**

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Bronchoscopic lung volume reduction (BVLR) with one-way endobronchial valves are employed to reduce hyperinflation to improve symptoms and lung function. The clinical benefits of Zephyr (PulmonX) valves in achieving target lobe volume reduction are most marked in those without collateral ventilation (CV) between ipsilateral non-target and target lobes. The presence of CV can be assessed by visual assessment of fissure integrity on CT scan and by measurement of flow and resistance following balloon catheter occlusion of the target lobe bronchus (Chartis; PulmonX). We have compared these two techniques for assessing collateral ventilation in patients with severe emphysema referred for EBV treatment.

**Pulmonary infection: clinical studies**

**S122 STATIN USE IS ASSOCIATED WITH IMPROVED LONG TERM OUTCOMES AFTER COMMUNITY-ACQUIRED PNEUMONIA**

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**Introduction** Long term outcomes after community-acquired pneumonia (CAP) are poor, with high rates of readmission, cardiovascular events and mortality. No intervention has previously been shown to alter the excess morbidity and mortality associated with CAP. Statins are effective in preventing cardiovascular disease, but may also have beneficial anti-inflammatory effects. We tested the hypothesis that statin users would have improved long term outcomes following CAP.