

score (SGRQ) at baseline.  $K_{co}$  SR significantly deteriorated in both groups during follow-up ( $p < 0.001$  in both cases) but this was not true for FEV<sub>1</sub> SR. Group B had a significantly faster decline in  $K_{co}$  than Group A ( $p = 0.005$ ) resulting in all values falling below the LLN.

**Conclusion** About half of the A1AD patients with an isolated FEV<sub>1</sub> abnormality at baseline have a decline in  $K_{co}$  faster than expected. Whether exacerbations, treatment or emphysema distribution relates to faster decline remains to be determined.

## REFERENCE

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## NIV: the acute and domiciliary settings

### S65 INTERIM DATA FROM A RANDOMISED CONTROLLED TRIAL OF AVERAGE VOLUME-ASSURED PRESSURE SUPPORT (AVAPS) VERSUS SPONTANEOUS-TIMED (ST) PRESSURE SUPPORT IN OBESITY HYPOVENTILATION SYNDROME (OHS)

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**Introduction** The obesity epidemic is leading to an increase in respiratory morbidity including sleep disordered breathing (SDB). The optimal ventilatory strategy in OHS is yet to be characterised. Changes in the respiratory system compliance through sleep and positional variations could compromise the effectiveness of fixed level pressure support thus the idea of varying pressure support to maintain tidal volume is appealing. Published data show improvements in nocturnal control of SDB in OHS using AVAPS mode ventilation (Storre *et al*, 2006) but also concerns regarding sleep fragmentation (Janssens *et al*, 2009). Data from these studies have been difficult to interpret as patient set up has not been set out to minimise the differences between modes.

**Method** Consecutive patients admitted for setup of domiciliary NIV were randomised to either ST or AVAPS mode (BiPAP Synchrony, Philips-Respironics, Murrsville, USA). NIV was titrated to improve nocturnal oximetry-capnometry using a standard setup protocol. Patients had baseline investigations including anthropometrics, health related quality of life and gas exchange. A subset of patients had sleep disruption monitored via actigraphy (AW64, Philips-Respironics, Murrsville, USA) in the week following discharge.

**Results** 50 patients were enrolled, mean age  $55 \pm 11$  years, 53% male, BMI  $50 \pm 7$  kg/m<sup>2</sup>, PaCO<sub>2</sub>  $6.85 \pm 0.81$  kPa, PaO<sub>2</sub>  $8.91 \pm 1.55$  kPa, ESS  $12 \pm 6$  with preliminary data available at 6 weeks for 43 patients, 13 were initiated acutely and 30 were elective admissions. No significant between group differences (Abstract S65 Table 1). Actigraphy analysis (AVAPS n=16, ST n=17) during 1st week of NIV showed total sleep time of  $347 \pm 80$  min in AVAPS and  $348 \pm 78$  min in ST with sleep efficiency of  $81 \pm 6\%$  in AVAPS and  $79 \pm 12\%$  in ST. Arterial blood gas analysis at 6 weeks showed no between group differences. However, significant improvement occurred in PaCO<sub>2</sub> in AVAPS patients, mean difference 0.63 kPa ( $p = 0.013$ ), that did not reach significance in ST, mean difference 0.55 kPa ( $p = 0.053$ ).

## Abstract S65 Table 1

	AVAPS (n=22)	ST (n=21)
Age (years)	54±9	56±12
Gender (%male)	50	57
BMI (kg/m <sup>2</sup> )	48±7	51±7
ESS (/24)	11±6	14±6
Baseline PaCO <sub>2</sub> (kPa)	6.93±0.77	6.77±0.85
Baseline PaO <sub>2</sub> (kPa)	9.08±1.14	8.72±1.91
Mean delivered Ipap	23±5	23±4
4%ODI	23±17	20±15
Mean nocturnal SpO <sub>2</sub>	92±3	92±4
Mean tcCO <sub>2</sub>	7.0±0.6	7.2±1.1
ΔPaCO <sub>2</sub> (kPa)	-0.63±1.1	-0.55±1.2
ΔPaO <sub>2</sub> (kPa)	0.1±1.3	0.7±1.4

**Conclusion** The preliminary data indicate no significant sleep disruptive effect of AVAPS mode compared to ST following initiation of NIV for OHS. There was no clinically significant difference between groups in the primary outcome of change in PaCO<sub>2</sub>, although there was only a significant fall in PaCO<sub>2</sub> in the AVAPS group. 3 months data are awaited.

### S66 A RANDOMISED CROSSOVER TRIAL OF PRESSURE SUPPORT VENTILATION (PSV) VERSUS PRESSURE CONTROLLED VENTILATION (PCV) IN STABLE HYPERCAPNIC CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

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**Introduction** PSV, rather than PCV, is often the preferred mode of ventilatory support in stable hypercapnic COPD patients, although this is supported by limited data. We hypothesised that PCV would be equivalent to PSV with similar patient-ventilator synchrony, nocturnal ventilation, gas exchange and sleep quality with similar adherence to non-invasive ventilation and improvements in health related quality of life (HRQL).

**Method** Hypercapnic COPD patients (daytime PaCO<sub>2</sub> >6 kPa) were enrolled and randomised to PSV (low back up rate) or PCV for 6 weeks. Patients had baseline anthropometric, HRQL and gas exchange measurements, were re-assessed at 6 weeks and crossed over to the other arm with the final assessment at 12 weeks. Sleep quality was assessed using a sleep diary and nocturnal actigraphy (Actiwatch AW4, CamNTEch, Cambridge, UK) for 2 weeks prior to each assessment. Subjective patient-ventilator synchrony was assessed with a questionnaire (Score 0–3, 0=good synchrony). At 6 and 12 weeks, all measurements were repeated, including ventilator adherence.

**Results** 12 patients were enrolled. However, three were unable to tolerate NIV and 1 failed to adhere to the study protocol. Four commenced on PSV and 4 commenced on PCV. Mean (±SD) age  $72 \pm 8$  years, BMI  $32 \pm 9$  kg/m<sup>2</sup>, FEV<sub>1</sub>/FVC  $54 \pm 14\%$ , FEV<sub>1</sub>  $29 \pm 11\%$ , PaCO<sub>2</sub>  $8.6 \pm 1.7$  kPa and PaO<sub>2</sub>  $7.3 \pm 1.4$  kPa. Mean IPAP  $28 \pm 4$  cm H<sub>2</sub>O and mean EPAP  $5 \pm 3$  cm H<sub>2</sub>O. Backup rate for PSV

was 6 bpm and PCV was 16±1 bpm. The mean nocturnal respiratory rate was 14±3 bpm and 16±1 bpm with triggering compliance of 90±9% and 24±10% for PSV and PCV, respectively. According to the patient-ventilator synchrony questionnaire, patients reported an increased awareness of ineffective efforts (0.7±0.5 vs 0.1±0.3; p=0.03) and a short inspiratory time (0.7±0.7 vs 0.1±0.3; p=0.03) during PSV, but there was no difference in self-reported sleep comfort and quality. Furthermore, there were no between group differences in change in PaCO<sub>2</sub>, ventilator use or nocturnal actigraphy. Although there were similar between group improvements in HRQL, the respiratory symptom domain of the Severe Respiratory Insufficiency Questionnaire favoured PSV (p=0.02).

**Conclusion** Despite clinicians' perception that PSV has an advantage over PCV for the management of patients with stable hypercapnic COPD, these pilot data do not necessarily support this view.

### S67 PHENOTYPIC DIFFERENCES BETWEEN OBESE PATIENTS WITH EUCAPNIC AND HYPERCAPNIC SLEEP-DISORDERED BREATHING (SDB)

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**Background** Obesity-related SDB requiring domiciliary non-invasive ventilation (NIV) can present as (1) eucapnic obstructive sleep apnoea (OSA) (2) hypercapnic OSA (P<sub>a</sub>CO<sub>2</sub> >6 kPa) (3) hypercapnic OSA with obesity hypoventilation syndrome (OHS) and (4) lone OHS. We have adopted the term obesity-related respiratory failure (ORRF) to group these differing conditions. The aim of this study was to determine the clinical features prevalent in each of these conditions in order to guide respiratory management.

**Method** Data from patients initiated on domiciliary NIV at a tertiary referral unit, between August 2005 and December 2009, were obtained from a purpose-designed discharge summary database. Patients were categorised into four groups, as described above. Comparative analyses were performed between (1) eucapnic OSA and the hypercapnic groups (2) hypercapnic OSA and a group combining the OSA with OHS and the lone OHS groups (OSA & OHS and lone OHS) and (3) eucapnic and hypercapnic OSA. Logistic regression analysis was performed to determine factors associated with hypercapnia.

**Results** 163 patients were included in the analyses. Group mean (SD) age 54.3±14.2 years, weight 134.4±33.1 kg, body mass index (BMI) 48.4±2.5 kg/m<sup>2</sup> and Epworth sleepiness (ESS) score 14.8±5.8. Results are shown in Abstract S67 Table 1. The hypercapnic groups demonstrated a higher prevalence of diabetes. In addition, hypercapnic patients were overall, compared with eucapnic patients, more hypoxic with greater lung restriction, despite a non-significant increase in BMI in the hypercapnic OSA group. Compared with the hypercapnic OSA group, the combined OSA and OHS and lone OHS group had a higher BMI, ESS and greater hypercapnia. However, logistic regression analysis failed to demonstrate any factors that predicted hypercapnia.

Abstract S67 Table 1

	Eucapnic OSA (n=50)	Hypercapnic OSA (n=38)	OSA+OHS (n=64)	Lone OHS (n=11)
Male	36 (72)	22 (57.9)	29 (45.3)*	3 (27.3)*
Age (years)	50.0±13.5	56.1±14.0*	56.1±13.8*	57.1±17.4
Diabetes mellitus	10 (20)	17 (48.6)*	32 (50.0)*	4 (36.4)
Hypertension	21 (42)	24 (66.7)	33 (51.6)	5 (45.5)
BMI (kg/m <sup>2</sup> )	44.0±11.4	46.11±10.0‡	53.72±13.9* †	48.25±8.9
ESS	14.2±6.3	13.1±5.4‡	16.4±5.0*	12.7±10.6*
FVC (L)	3.0±1.1	1.9±1.0*	1.9±0.9*	1.5±0.6*
pH	7.41±0.02	7.39±0.03*	7.38±0.03*	7.39±0.06
P <sub>a</sub> O <sub>2</sub> (kPa)	10.4±1.6	8.0±1.5*	8.0±1.5*	7.8±1.3*
P <sub>a</sub> CO <sub>2</sub> (kPa)	5.4±0.4	7.0±0.7* ‡	7.6±1.3* †	7.9±1.2* †
HCO <sub>3</sub> <sup>-</sup> (mmol/l)	25.2±1.9	30.8±3.0*	33.0±4.5* †	33.5±5.2†
Length of stay (days)	2.6±2.8	4.3±2.7*	5.1±4.2*	6.5±6.0*
1-year mortality	0 (0)	1 (2.6)	2 (3.1)	1 (9.1)

Data presented as n (%) or mean±SD.

OSA, obstructive sleep apnoea; OHS, obesity hypoventilation syndrome; BMI, body mass index; ESS, Epworth sleepiness score; FVC, forced vital capacity.

\*Significant difference compared to eucapnic OSA group.

†Significant difference compared to hypercapnic OSA group.

‡Significant difference between hypercapnic OSA group and combined group of OSA+OHS and lone OHS.

**Conclusion** Demographic, anthropometric, spirometric and clinical features allow the different ORRF conditions to be distinguished. BMI, daytime symptoms and degree of chronic respiratory failure distinguished between hypercapnic OSA from obesity-related hypoventilation. Although we hypothesise that ORRF is a disease spectrum, from eucapnic OSA progressing to hypercapnic OSA to OSA with OHS, we were unable to identify factors that predicted hypercapnia. From these data, we propose that more detailed physiological assessment, including neural respiratory drive and pulmonary mechanics, is required.

### S68 EVALUATION OF NON-INVASIVE VENTILATION IN MANAGEMENT OF ACUTE SEVERE ASTHMA

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**Objectives** To study the role of non-invasive positive pressure ventilation (NIPPV) in management of acute severe asthma.

**Study design** Open randomised controlled trial.

**Methods** 50 patients of acute severe asthma having asthma for at least 1 year duration with exacerbation of less than 7 days duration, FEV<sub>1</sub> <50% of predicted, respiratory rate of >25 breaths/min and pulse rate >110/min after half hour of 5 mg nebulised salbutamol were included in the study over 1 year. Patients with known COPD, history of smoking >10 years, HR >140/min, systolic BP <90 mm Hg, facial deformity, pulmonary oedema, pneumonia and pregnancy were excluded. Patients were divided into two groups A and B. All patients received nebulisation with salbutamol 5 mg and ipratropium bromide 0.5 mg and hydrocortisone 100 mg IV at zero hour and later 5 mg salbutamol with small volume oxygen driven nebuliser @ 6 l/min at 1, 2, 3, 5 h of the study. Group B patients were given NIV support in addition to medical therapy for 6 h. All patients received O<sub>2</sub> at 6–8 l/min for 6 h. NIV and medical treatment were stopped after 6 h. Spirometry, ABG, respiratory rate (RR), accessory muscles of respiration (AMR) and Borg dyspnoea score were assessed at 0, 1, 3, 6 and 7th hour of study.

**Results** Out of 308 patients 246 were excluded because of non-fulfilment of inclusion criteria. Eight patients refused consent, three had pneumonia and claustrophobia to mask respectively. One