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 $PaCO_2$, 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_aCO_2 >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^2 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^2)	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_aO_2 (kPa)	10.4±1.6	8.0±1.5*	8.0±1.5*	7.8±1.3*
P_aCO_2 (kPa)	5.4±0.4	7.0±0.7* ‡	7.6±1.3* †	7.9±1.2* †
HCO_3^- (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, FEV1 <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_2 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

patient deteriorated in Group A and was withdrawn. The use of accessory muscles of respiration (AMR) reduced significantly in group B at 3, 6 and 7 h ($p<0.01$), BORG dyspnoea score improved significantly ($p<0.01$) in group B after 1 h. There was no difference between two groups in terms of improvement in RR, HR, FEV1 and ABG. In group B, the mean IPAP and EPAP used was 14.32±0.945 and 7.16±0.472 cm of water, respectively.

Conclusion The use of NIPPV in patients with acute severe asthma though found to be useful in terms of faster resolution of dyspnoea and decrease in use of AMR but did not improve pulmonary functions significantly.

S69 HOT HMV UK: PREVALENCE OF PERSISTENT SIGNIFICANT HYPERCAPNIA FOLLOWING ACUTE EXACERBATION OF COPD (AECOPD) REQUIRING NON-INVASIVE VENTILATION (NIV)

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Introduction Acute NIV is now standard therapy for hypercapnic respiratory failure (AHRF) complicating AECOPD. However, although chronic hypercapnia is an acknowledged poor prognostic factor in COPD and some data suggest a survival advantage with domiciliary NIV compared with oxygen alone, its use for chronic hypercapnic respiratory failure (CHRF) remains controversial.[1] Patients with CHRF who have recently been treated acutely with NIV for AECOPD may be a particularly appropriate group in whom to consider long term domiciliary NIV.

Method We are conducting an RCT (HOT HMV UK) of domiciliary NIV plus oxygen against domiciliary oxygen alone in CHRF (defined as $PaCO_2$ >7 kPa), focussing on patients who have recently received acute NIV for AECOPD, and we have evaluated the likely uptake of such treatment after resolution of the acute episode. All patients seen over a 5-month period at two tertiary centres for consideration of domiciliary NIV following acute NIV were assessed at least 2 weeks following resolution of AHRF and arterial blood gas analysis was performed.

Results 38 patients received acute NIV for an AECOPD. Mean (±SD) age was 69±17 years and 50% were male, with $PaCO_2$ of 9.3±2.4 kPa at acute presentation. Eight (21%) patients died prior to review (six during initial admission, two shortly following discharge); four patients whose diagnosis was clarified as an overlap of obstructive sleep apnoea and COPD were treated on clinical grounds with NIV and one patient was already receiving domiciliary NIV. Of the remaining 25 patients, nine had $PaCO_2$ <7 kPa at 2 weeks, leaving 16 (42%) with CHRF at 2 weeks post recovery. Importantly, seven (44%) of these did not wish to be considered for a clinical trial involving domiciliary NIV, the principal reason being poor tolerance during the acute episode.

Conclusion More than 40% of patients requiring NIV for AHRF complicating AECOPD had CHRF with a $PaCO_2$ >7 kPa, when assessed 2 weeks after discontinuation of acute NIV. However, nearly half of these reported negative experiences of acute NIV that made them reluctant to consider long term treatment.

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S70 NON-INVASIVE VENTILATION (NIV) IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) EXACERBATIONS WITH ACUTE HYPERCAPNIC RESPIRATORY FAILURE (AHRF) WITH PH

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Introduction Recent British guidelines on NIV suggest patients with AHRF and pH <7.26 on arterial blood gases (ABG) should be managed by critical care (ITU) depending upon local circumstances with a low threshold for intubation, unless NIV is deemed to be the ceiling of treatment. The 7.26 pH cut-off was derived from subgroup analysis of in-hospital mortality assessment from the study by Plant *et al* (*Lancet* 2000; 355:1931–5). The use of NIV as an alternative to endotracheal intubation in more severely acidotic COPD patients (pH<7.26) has been controversial but there is increasing evidence that the outcomes in such patients may not be any worse if treated with NIV.

Methods Analysis of initial ABG (pre-commencement of NIV) for AHRF secondary to COPD between 1 August 2004 and 31 December 2009 was performed. NIV was undertaken in a dedicated unit on a respiratory ward. The admission episodes were stratified by initial pH ranges (predictor variable) and in-hospital mortality was recorded (outcome variable).

Results Out of 728 (505 unique patients) admissions with COPD requiring NIV for AHRF, 282 admissions had a pH <7.26. Of these, 224 admissions survived to discharge (mortality 20.6%). Stratifying the admissions by pH categories of 0.05, there is no significant difference in the mortality with pH ranges 7.2–7.25 and 7.25–7.30 (p value=0.845). If all COPD admissions requiring NIV (n=728) are stratified into two groups above and below 7.2, the ratio of odds of survival below pH 7.2 drops sharply (p=0.000000088): Abstract S70 Table 1.

Abstract S70 Table 1

pH	Survived	Not Survived	n
<7.2	110 (73.3%)	40 (26.7%)	150
≥7.2	517 (89.4%)	61 (10.6%)	578
Total	627	101	728

Conclusion Mortality in the group with pH 7.20–7.25 was similar to the group with pH 7.26–7.30, suggesting that NIV on a dedicated respiratory ward can manage COPD patients with AHRF and pH <7.26. Our findings suggest that it is probably justified to recommend that the initial pH cut-off is modified from pH >7.26 to pH >7.20 for ward-based NIV in COPD exacerbations with AHRF. This has important resource implications, given that more patients could be managed in a ward-based dedicated NIV unit rather than the utilising ITU beds in the UK National Health Service.

Novel outcomes and interventions in pulmonary rehabilitation

S71 AMBULATORY OXYGEN IMPROVES THE EFFECTIVENESS OF PULMONARY REHABILITATION (PR) IN SELECTED PATIENTS

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Rationale The acute effect of supplemental oxygen on exertion is well documented although its use in PR has not yet been clearly