Methods: Pdi,Sn and Pdi,Tw were measured in 91 and 101 healthy volunteers, respectively, to determine normal values, and in 453 patients referred for respiratory muscle assessment. Diaphragm weakness was defined as a Pdi,Sn and Pdi,Tw below the 5th centile.
Results: Mean (SD) Pdi,Sn was 131 cmH_2O (27) for men and 114 cmH_2O (24) for women. Mean (SD) Pdi,Tw was 27 cmH_2O (6) for men and 25 cmH_2O (5) for women. Age was negatively associated with Pdi,Sn ($r^2 = -0.27$) and Pdi,Tw ($r^2 = -0.39$). Height, weight and body mass index did not independently influence Pdi,Sn or Pdi,Tw. Taking into account age and gender, 204 patients were considered to have diaphragm weakness. The addition of the non-volitional Pdi,Tw to Pdi,Sn in the identification of diaphragm weakness improved specificity from 72% to 100% and positive predictive value from 82% to 100%.
Conclusions: Normal values for Pdi,Sn and Pdi,Tw have been established. The use of both tests of diaphragm function increases diagnostic precision.

S68 THE RELATIONSHIP BETWEEN NEURAL RESPIRATORY DRIVE AND HYPERCAPNIA IN PATIENTS WITH NEUROMUSCULAR DISEASE

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Background: Although respiratory muscle strength is a better predictor of hypercapnic ventilatory failure than poor spirometry in neuromuscular disease (NMD), more reliable tests to monitor progression to hypercapnic ventilatory failure are required. If the respiratory muscles are weak, neural respiratory drive must increase to maintain the balance between the capacity of the respiratory muscles and the load on the respiratory muscle pump. Neural respiratory drive can be measured by quantifying the diaphragm electromyogram recorded using a multipair oesophageal electrode.
Aims and Hypothesis: The aim of the study was to investigate the relationship between neural respiratory drive, measured by quantifying the diaphragm electromyogram (EMGdi) and carbon dioxide retention in NMD. We hypothesised that there would be a positive relationship between arterial pCO_2 and diaphragm electromyogram activity in patients with NMD.
Methods: 11 patients with NMD were studied (four neuralgic amyotrophy, two amyotrophic lateral sclerosis, one muscular dystrophy, one phrenic nerve trauma, one myotonic dystrophy, one polymyositis, one polyneuropathy; eight men; mean (SD) age 53.9 years (10.1); vital capacity 76.8% predicted (20.0); pH 7.43 (0.04), pO_2 11.0 kPa (1.5), pCO_2 5.1 kPa (1.0), HCO_3 24.9 mmol/l (3.5)). Sniff nasal pressure (SNIP), and mouth inspiratory pressure (PImax) were measured. EMGdi was recorded at rest using a multipair oesophageal electrode. Resting EMGdi was normalised by expressing EMGdi as a percentage of peak EMGdi recorded during maximum inspiratory manoeuvres. Normalised EMGdi activity/minute was then calculated (''EMGdi%index''). The maximum inspiratory manoeuvres were inspiration from FRC to TLC, PImax manoeuvres, maximum sniff manoeuvres and sprint maximum voluntary ventilation over 15 s. Relationships between variables were assessed using linear regression analysis.
Results: Correlations between EMGdi%index and each variable measured are shown in the table. There were significant correlations between EMGdi%index and pCO_2 ($r = 0.67$, $p = 0.03$) and HCO_3 ($r = 0.63$, $p = 0.04$) only. Mean (SD) PImax and SNIP were 63.1 cmH_2O (33.0) and 51.0 cmH_2O (35.7), respectively, and mean (SD) EMGdi%index was 575.6 au/min (312.6).
Conclusion: The significant correlations between EMGdi%index, pCO_2 and HCO_3 suggest that the EMGdi%index could potentially be used to monitor progression towards hypercapnic ventilatory failure in NMD. The value of the EMGdi%index over other respiratory muscle function tests requires further study.

Abstract S68 Table Correlations between EMGdi%index and anthropometric/physiological variables

	r	p Value
Age	0.59	0.06
BMI	−0.03	0.9
pH	−0.42	0.2
pO_2	−0.51	0.1
pCO_2	0.67	0.03
HCO_3	0.63	0.04
Vital capacity %	−0.16	0.7
Sniff nasal pressure	−0.54	0.09
PImax	−0.61	0.1

BMI, body mass index; PImax, mouth inspiratory pressure.

Paediatric lung disease

S69 THE DEVELOPMENT OF THE BRONCHIAL SUBEPITHELIAL RETICULAR BASEMENT MEMBRANE

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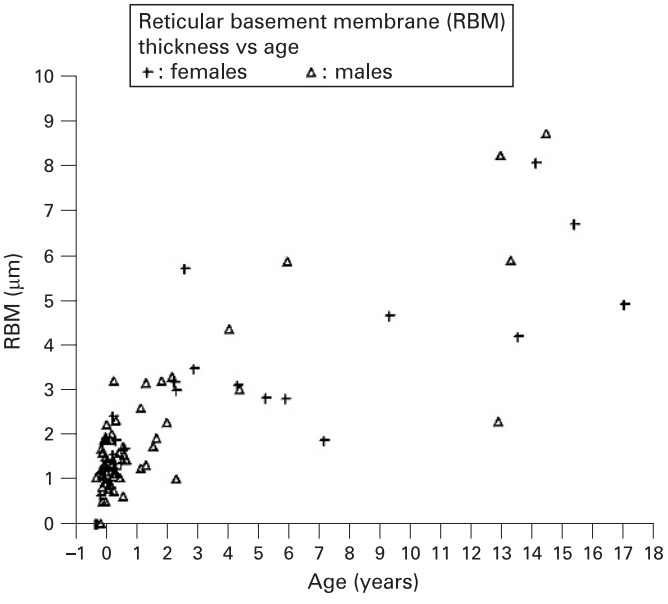
Background: Abnormal thickening of the bronchial subepithelial reticular basement membrane (RBM) is a recognised feature of airway remodelling.[1–3] However, even although the RBM is present in the airways of healthy children and adults, nothing is known about its normal development. We hypothesised that the RBM is first visible at birth and subsequently thickens normally with age for the first 3 years, when final adult thickness is reached.
Methods: Cartilaginous airways were studied in lungs obtained postmortem from 87 infants and children (22 weeks gestation to 17 years old) who had died from non-respiratory causes and had no history of asthma. RBM thickness was measured in haematoxylin and eosin stained paraffin wax sections using computer aided image analysis and a method previously validated in endobronchial biopsies.[4]



Abstract S69 Figure

Results: The RBM was first visible at 30 weeks gestation (28–34 weeks gestation, median (range) RBM thickness 1.1 μm (0–1.68)), and thickened rapidly during the first 3 years of life (fig). Subsequently it continued to thicken at a slower rate until 17 years (15–17 years old, median RBM thickness 5.78 μm (4.89–6.68)). From 6 years onwards, there was large biological variability in RBM thickness, which may explain why no plateau was seen as expected. RBM thickness was related to both weight and height of subjects (Spearman's r = 0.729, p<0.001 and r = 0.725, p<0.001) and increased with increasing airway size (Spearman's r = 0.357, p<0.001).
Conclusions: The RBM in healthy humans is first visible using light microscopy at approximately 30 weeks gestation and subsequently thickens with age. The large degree of biological variability in RBM thickness precluded the determination of an age when final adult thickness is reached. The influence of age, body and airway size on RBM thickness suggests that paediatric airway pathology studies should include controls matched for these parameters.

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3. **Payne DN,** *et al*. *AJRCCM* 2003;**167**:78–82.
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S70 RELATIONSHIP BETWEEN NON-INVASIVE INFLAMMATORY MARKERS AND THE CURRENT LEVEL OF CLINICAL CONTROL IN CHILDHOOD ASTHMA

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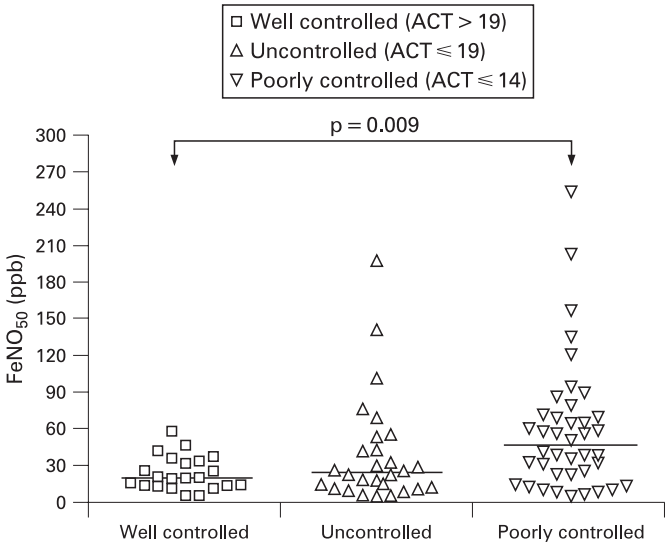
Background: Exhaled nitric oxide ($FeNO_{50}$), sputum analysis and exhaled breath condensate pH (EBC pH) are recognised non-invasive markers of airway inflammation. The asthma control test (ACT) is a validated tool that can be used in clinical practice for monitoring asthma control.
Aim: To investigate whether the measurements of non-invasive markers of inflammation ($FeNO_{50}$, sputum eosinophils and neutrophils and EBC pH) reflect asthma control in children with a range of asthma severity.
Methods: Sputum induction and EBC were performed and $FeNO_{50}$ was measured in 55 children with severe asthma and in 35 children with mild–moderate asthma. The ACT was used to evaluate current symptom control over the previous 4 weeks.



Abstract S70 Figure $FeNO_{50}$ (ppb) in asthmatic children with different level of control

Results: $FeNO_{50}$ was significantly increased in the poorly controlled compared with the well controlled group (p = 0.009) (fig). However, there was a marked overlap between the groups. Sputum eosinophils (p = 0.183), sputum neutrophils (p = 0.518), and EBC pH (p = 0.136) did not differ between the three different control groups.
Conclusion: $FeNO_{50}$ may reflect recent poor control better than sputum cytology and EBC pH.

S71 EFFECTS OF IL-13 ON NORMAL AND ASTHMATIC PAEDIATRIC BRONCHIAL EPITHELIAL CELLS: IL-13 AS A THERAPEUTIC TARGET IN CHILDHOOD ASTHMA

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Background: Goblet cell hyperplasia and decreased ciliogenesis are characteristic features of chronic asthma and this may be influenced by exposure to Th2 cytokines (eg, IL-9 and IL-13). IL-13 may cause goblet cell hyperplasia, increased mucus production and decreased ciliogenesis.
Aim: We studied in vitro basal mucociliary differentiation and looked for differences (between normal and asthmatic individuals) when paediatric epithelial cells were exposed to IL-13, IL-9 and IL-9 plus IL-13.
Method: Blind non-bronchoscopic bronchial brushings obtained from children (normal and asthmatic) are differentiated at the air–liquid interface for 28 days. Cells are treated with 20 ng/ml IL-13, 2 ng/ml IL-13, 20 ng/ml IL-9 and 20 ng/ml IL-9 and 20 ng/ml IL-13. Transepithelial resistance (TER), number of ciliated (anti α_1-acetylated tubulin antibody) and goblet cells ($Muc5AC^+$) are assessed as a measure of tissue differentiation.
Results: Both asthmatic (n = 4) and normal (n = 5) cell cultures formed well-differentiated pseudostratified epithelium (TER >500 Ω/cm^2). Asthmatic cultures expressed significantly more goblet cells (50.6%, SD 15.9) when compared with non-asthmatic cultures (23.6%, SD 6.3) under basal culture conditions (p = 0.036). Significantly more goblet cells are seen in asthmatic cultures when chronically exposed to IL-13 (20 ng/ml and 2 ng/ml), IL-9, IL-9 plus IL-13 when compared with identically treated non-asthmatic cultures (p<0.05). Asthmatic cultures expressed significantly less ciliated cells (15.1%, SD 2.4) when compared with non-asthmatic cultures (24.7%, SD 5.8) under basal culture conditions (p = 0.0381). Chronic treatment with IL-13, IL-9, IL-9 plus IL-13 produced less ciliated cells in asthmatic cultures when compared with non-asthmatic individuals but these results were not statistically significant.
Discussion: Asthmatic cells differentiate at basal conditions with a higher proportion of goblet cells and a lower number of ciliated cells when compared with non-asthmatic cultures. Chronic treatment with IL-13, IL-9, IL-9 plus IL-13 increases the number of goblet cells.
Funding: Sponsored by the Irish Thoracic Society.

S72 NON-INVASIVE MARKERS OF INFLAMMATION AS PREDICTORS OF A SEVERE EXACERBATION IN CHILDREN WITH PROBLEMATICAL ASTHMA

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Background: Exacerbations of asthma are associated with an increase in airway inflammation. The ability to predict a severe exacerbation could improve management if doses of maintenance treatment were altered appropriately.
Aim: To evaluate the ability of non-invasive markers of inflammation (sputum eosinophils and exhaled nitric oxide ($FeNO_{50}$)) and clinical symptoms to predict an exacerbation requiring a course of oral steroids in children with severe asthma within 2 weeks.

Abstract S72 Table

	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Area under ROC curve
Sputum eosinophils					0.565
⩾0.1%	88.2	19.3	12.1	92.9	
>2.5%	64.7	48.1	13.6	91.5	
$FeNO_{50}$					0.565
⩾22 ppb	68.4	39.1	10.4	92.3	
>30 ppb	57.9	46.7	10.1	91.5	
Asthma control test					0.726
<15	73.3	62.0	17.5	95.5	
<20	100	36.5	14.7	100	

NPV, negative predictive value; PPV, positive predictive value; ROC, receiver operator characteristic.

Methods: At four routine clinic visits over a one-year period sputum induction was performed and $FeNO_{50}$ measured in 55 children (7–17 years) with severe asthma. The asthma control test (ACT) was used to evaluate current symptom control (total score 25, lower scores reflect worse control).
Results: 203 study visits were completed. 14 children had a total of 19 exacerbations requiring a course of oral steroids within 2 weeks of the clinic assessment. The sensitivity, specificity, positive and negative predictive values for the inflammatory markers and ACT are shown in the table.
Conclusion: In children with severe asthma non-invasive markers of inflammation are poor positive predictors of an acute severe exacerbation of asthma, but better negative predictors, because low levels of inflammatory markers ($FeNO_{50}$ or sputum eosinophil count) or a high ACT score (good symptomatic control) indicate that an acute exacerbation of asthma within the next 2 weeks is unlikely.

S73 DISCORDANCE BETWEEN SPUTUM EOSINOPHILS AND EXHALED NITRIC OXIDE IN CHILDREN WITH ASTHMA

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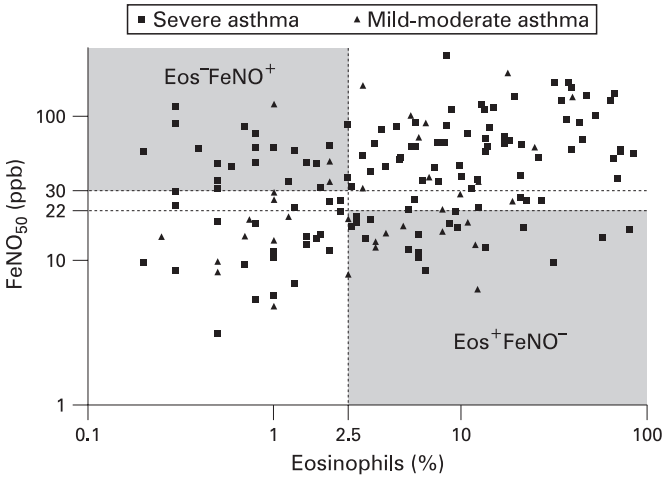
Introduction: It has been suggested that management of patients with asthma can be improved by using two non-invasive markers of inflammation, sputum eosinophils and exhaled nitric oxide ($FeNO_{50}$) and that these two measures are positively related.
Methods: Sputum induction was performed and $FeNO_{50}$ measured at four routine clinic visits over a one-year period in 55 children with severe asthma (taking ⩾500 μg fluticasone or equivalent per day) and at two routine clinic visits over a 6-month period in 35 children with mild–moderate asthma (taking <500 μg fluticasone or equivalent per day). Sputum differential cell counts were expressed as a percentage of 400 cells. The relationship between sputum eosinophils and $FeNO_{50}$ was defined as outlined in the table.

Abstract S73 Table

	Eosinophils %	$FeNO_{50}$ ppb
Concordant		
Eos^+FeNO^+	⩾2.5	⩾22
Eos^-FeNO^-	<2.5	⩽30
Discordant		
Eos^+FeNO^-	>2.5	<22
Eos^-FeNO^+	<2.5	>30

Eos, eosinophils; FeNO, exhaled nitric oxide.



Abstract S73 Figure

Results: 200 paired sputum and $FeNO_{50}$ samples were obtained in 79 subjects. 37 subjects (47%) were found to have a discordant relationship on at least one occasion (59 paired samples). In 26 paired samples the $FeNO_{50}$ was high despite low numbers of eosinophils (Eos^-FeNO^+), in keeping with previous findings; however, in 33 paired samples, despite high sputum eosinophils, the $FeNO_{50}$ was found to be low (Eos^+FeNO^-). The relationship was not consistent; 21 discordant subjects also demonstrated a concordant relationship and two showed discordance in both directions. There were no significant differences in the proportions of discordant samples between severe and mild–moderate asthmatic patients (see fig).
Conclusion: Many children with asthma do not show the expected relationship between sputum eosinophils and $FeNO_{50}$. The $FeNO_{50}$ measurement may under or overestimate sputum eosinophilia and is inconsistent over time. The clinical usefulness of $FeNO_{50}$ to measure underlying eosinophilic inflammation is low and the two measurements cannot be used interchangeably in paediatric asthma.

S74 PEPSIN MEASURED IN INDUCED SPUTUM: A TEST FOR PULMONARY ASPIRATION?

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Background: Pepsin is only produced in gastric mucosa. Pepsin measured in bronchoalveolar lavage fluid is a useful marker of pulmonary aspiration secondary to gastro-oesophageal reflux (GOR) but obtaining samples is invasive. Induced sputum is a potential non-invasive method of obtaining samples. It is possible that the induced sputum procedure could cause GOR and invalidate the interpretation of results.
Aim: We assessed pepsin concentrations in induced sputum (N = 20) and in pre and post-induced sputum saliva (N = 19) samples from 20 normal children. Our hypothesis was that induced sputum and saliva after the induced sputum procedure contains no detectable pepsin.
Methods: Children, aged 4–16 years, with no respiratory or gastrointestinal disorders were recruited. Induced sputum samples were obtained after nebulisation with 3% saline. Samples of saliva were collected pre and 15 minutes post-sputum induction. Pepsin concentrations were measured using an "in house" ELISA with antiporcine pepsin antibodies. Samples were classified as detectable if pepsin concentration was above our lower level of detection (1.19 ng/ml).

Results: Of 20 induced sputum samples 17 (85%) were pepsin positive. Saliva pepsin pre-induced sputum was detected in 17 cases and from a further two cases post-induction. In addition, we observed a statistically significant increase in saliva pepsin concentration after the induced sputum procedure (median increase 2.9 ng/ml, $p = 0.004$).
Conclusion: Pepsin appears to be detectable in the saliva and induced sputum in asymptomatic children and is therefore not useful as a method of detecting GOR-related aspiration. We have shown some evidence that the induced sputum procedure could cause GOR. The frequent finding of pepsin to be detectable in baseline saliva suggests that mild asymptomatic GOR events occur and that pepsin clearance from saliva may not be rapid.
Funding: This work was funded by The Royal Belfast Hospital for Sick Children and Northern Ireland Chest, Heart and Stroke Association.

Sleep and ventilatory support

S75 FACTORS CONTRIBUTING TO RESIDUAL DAYTIME SLEEPINESS IN PATIENTS WITH OBSTRUCTIVE SLEEP APNOEA SYNDROME TREATED BY CONTINUOUS POSITIVE AIRWAYS PRESSURE

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Background: Although continuous positive airways pressure (CPAP) is a highly effective treatment for obstructive sleep apnoea syndrome (OSAS) some patients complain of persistent daytime sleepiness, the reasons for which are not well understood.
Aim: To investigate the factors contributing to residual daytime sleepiness in a cohort of patients with OSAS compliant with CPAP treatment.
Method: Prospective data collection from patients established on previously titrated, fixed pressure CPAP for at least 12 months. Entry criteria: OSAS with an apnoea/hypopnoea index (AHI) of 15/h or greater; CPAP compliance more than 4 h/night for at least the preceding 12 months. Patients were categorised by their current Epworth sleepiness score (ESS): non-sleepy group (ESS $\leqslant$10); sleepy group (ESS >10). Patients in group 2 underwent re-titration studies using an autotitrating machine (RespMed Autoset) to ensure optimal CPAP usage and identify central apnoeas.
Results: 59 patients were included, 19 sleepy and 40 non-sleepy. 48 (81%) were men. Mean age was 49.4 years (range 23–67). The groups are compared in the table. No significant differences were seen in: age; smoking status; frequency of relevant co-morbidities and previous or current shift working. 16 patients with an ESS greater than 10 have completed re-titration studies with the following results: CPAP set too low (three); CPAP too high (one); significant mask leak (one); central apnoeas noted (one); CPAP pressure to overcome apnoeas intolerable (three); no issue noted (seven; 44%). In addition, three had co-morbidity, which may have contributed to sleepiness (severe chronic obstructive pulmonary disease; multisystem atrophy; ischaemic heart disease and diabetes).
Conclusions: Patients with residual daytime sleepiness were sleepier before treatment with a trend to more severe disease. They were more likely to be current snorers using higher CPAP pressures and to have had empirical pressure increases. The use of medications that cause drowsiness was more prevalent. Re-titration enabled the identification and management of specific issues in most patients. This represents and is a useful tool for assessing persistent sleepiness in patients compliant with therapy. There remains a significant proportion in whom no cause of residual daytime somnolence is identifiable.

S76 ARTERIAL STIFFNESS IN THE OBSTRUCTIVE SLEEP APNOEA/HYPOPNOEA SYNDROME

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Introduction and Objectives: The obstructive sleep apnoea/hypopnoea syndrome (OSAHS) is an independent risk factor for cardiovascular disease. One marker of cardiovascular dysfunction is increased arterial stiffness. Arterial stiffness can be measured non-invasively using aortic pulse wave velocity (PWV) and the augmentation index (AIx), which is a measure of the augmentation of central aortic pressure due to pulse wave reflection. We aimed to determine whether there was a relationship between OSAHS severity and arterial stiffness in patients without known cardiovascular disease.
Methods: We recruited consecutive patients presenting to the sleep clinic with symptoms suggestive of OSAHS and an AHI of 15 or greater on polysomnography. All patients were continuous positive airways pressure (CPAP) naive and had no history of cardiovascular disease, hypertension or diabetes. Radial pulse wave analysis and aortic (carotid–femoral) PWV were carried out by a single operator using applanation tonometry (Sphygmocor). The AIx, corrected to a heart rate of 75 (AIx 75) was derived from radial pulse wave analysis. Analysis was undertaken using standard methods (SPSS, version 14). Pearson or Spearman's rank correlations were also performed. A p value of 0.05 or less was considered statistically significant.
Results: 48 patients (mean age 46 years (SD 8.6), 65% male) were recruited. Baseline characteristics were: body mass index 30.7 kg/m^2 (SD 4.3), fasting cholesterol 5.2 mmol/l (SD 0.9), blood pressure systolic 129 mm Hg (SD 13), diastolic 76 mm Hg (SD 9), mean arterial pressure 94 mm Hg (SD 9), AHI 35 h/sleep (SD 21), ESS 11 (SD 5). Mean aortic PWV was 7.6 m/s (SD 1.4) and AIx75 18% (SD

Abstract S75 Table Comparison of characteristics of two groups

	Non-sleepy group	Sleepy group	p Value
Years from diagnosis (mean)	6.07 ± 0.63	5.47 ± 0.98	ns
ESS at diagnosis (median)	13 (3–22)	18 (7–24)	0.0015
Change in ESS	−7.5 (−21–5)	−3 (−11–7)	0.04
Mean BMI (kg/m^2) at diagnosis	35.1 ± 1.04	36.3 ± 2.12	ns
Change in BMI	−1 ± 0.47	−0.7 ± 0.31	ns
Mean AHI/h	45.1 ± 3.37	57.9 ± 6.3	0.06
Mean compliance in last 12 months (h/night)	5.99 ± 0.34	6.58 0.35	ns
Empirical change from initial CPAP pressure, n (%)	13/40 (32.5%)	10/19 (52%)	0.055
Median current CPAP pressure (mm Hg)	10 (6–15.6)	12 (8–16)	0.054
Snorer on CPAP, n (%)	2/40 (5%)	9/19 (47%)	0.006
Patient on at least 1 drug causing drowsiness, n (%)	11/40 (27.5%)	11/19 (57.8%)	0.049

AHI, apnoea/hypopnoea index; BMI, body mass index; CPAP, continuous positive airways pressure; ESS, Epworth sleepiness score.

Corrections

doi:10.1136/thx.2008.102947corr1

O'Driscoll B R, Howard L S, Davison A G on behalf of the British Thoracic Society. BTS guideline for emergency oxygen use in adult patients. *Thorax* 2008; **63(Suppl 6)**:vi1–vi68.

The Guideline Development Group regret that no advice was offered for the management of status epilepticus. As this is a life-threatening condition where a patient may suffer from cerebral hypoxia (and oximetry may not be possible), patients with status epilepticus should be treated in accordance with table 1 (reservoir mask until clinically stabilised). This advice also applies to other rare conditions that may cause life-threatening hypoxaemia that are not listed specifically in table 1.

Roberts JA. Benchmarking chronic obstructive pulmonary disease across an inner city primary care trust: one year on. *Thorax* 2008;**63(Suppl vii)**:A8 (abstract S12).

The correct authors for this abstract are as follows: Roberts JA, Bakerly ND.

Roberts JA. Should chronic obstructive pulmonary disease service delivery in an inner city primary care trust be targeted at general practice or practice-based commissioning cluster level? *Thorax* 2008;**63(Suppl vii)**:A8 (abstract S13).

The correct authors for this abstract are as follows: Roberts JA, Bakerly ND.

Tsartsali L, Fleming L, Regamey N, *et al*. Relationship between non-invasive inflammatory markers and the current level of clinical control in childhood asthma. *Thorax* 2008;**63(Suppl vii)**:A33 (abstract S70).

This abstract has been withdrawn.

Baird S, Ashish A, O'Connor J, *et al*. Respiratory assessment centre: does it increase the number of patients taken home with the early supported discharge team? *Thorax* 2008;**63(Suppl vii)**:A67 (abstract S154).

It has come to the attention of the Scientific Committee that this abstract was not seen by all the authors prior to submission and is therefore withdrawn.

doi:10.1136/thx.2007.088831corr1

Creagh-Brown B C, Nicholson A G, Showkathali R, *et al*. Pulmonary veno-occlusive disease presenting with recurrent pulmonary oedema and the use of nitric oxide to predict response to sildenafil *Thorax* 2008;**63**:933–4.

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