detrimental impact on patients (ERS 2010). Health service costs were not originally addressed but are now considered.

Methods One hundred consecutive patients were invited to have a pre-clinic telephone consultation; 49% (49/100) accepted; 51% (51/100) declined/failed to respond. Fifty-seven patients referred through the electronic Choose and Book (C&B) system during the same period formed a comparator group. The costs of delivery included the pre-clinic telephone consultations in the intervention group and first and follow-up clinic consultation in both groups. Two perspectives were taken, the purchaser (NHS Primary Care Trust) who incurs the cost of delivery, and the hospital who bear the financial burden for non-attendance.

Results In the intervention group, 98% (48/49) had a pre-clinic telephone consultation, 100% had an initial clinic consultation and 36.7% (18/49) had one or more follow-up appointments. In the C&B arm, 82% (47/57) of patients attended the first consultation and 49% (28/57) had one or more follow-ups. Taking the perspective of the purchaser there were 48 telephone patients, and 47 C&B patients. There was no cost for non-attendance from this perspective. The costs per patient for the telephone group were £340, and for the C&B group £359. This was not a significant difference (mean difference of £-20 (95% CI −74.60, 34.70, p=0.470)). From the hospital perspective there were 49 patients in the telephone group and 53 in the C&B group. The cost of non-attendance and rearranged appointments for 6 months from first planned contact was £19 per patient (telephone group) and £71 (C&B), mean difference £−52 (95% CI −97.24, −6.11, p=0.027).

Conclusion Pre-clinic telephone consultations have been shown to be cost-saving for hospitals, substantially reducing the financial burden of non-attendance. From the PCT perspective there was no statistical difference in the cost of delivery between the two groups. This study used observational data from a self-selected patient group, further work is needed to confirm findings.

Funding This work was supported by the Dunhill Medical Trust.

Assessing the impact of interventions in sleep-disordered breathing

S13 THE PRIMARY RESULTS OF THE MOSAIC TRIAL: DOES CPAP FOR MINIMALLY SYMPTOMATIC OSA REDUCE DAYTIME SLEEPINESS OR CALCULATED VASCULAR RISK?

doi:10.1136/thx.2010.150912.13

[1]S E Craig, [2]M Kohler, [1]D Nicoll, [3]D J Bratton, [3]A J Nunn, [1]R J O Davies, [1]J R Stradling. *[1]Oxford Centre for Respiratory Medicine, Oxford, UK; [2]University Hospital of Zurich, Zurich, Switzerland; [3]MRC Clinical Trials Unit, London, UK*

Introduction CPAP treatment for symptomatic OSA improves sleepiness, and reduces vascular risk by reducing blood pressure (BP) and cholesterol. Minimally symptomatic OSA is far more prevalent than symptomatic disease, and treatment of this group is contentious. This trial describes the effect of CPAP on sleepiness and calculated vascular risk in minimally symptomatic OSA.

Methods 391 patients from 10 centres, with proven OSA (sleep study ODI>7.5 h), but insufficient sleepiness for CPAP (based on established evidence), were randomised (minimisation by ODI, recruiting centre, and cardiovascular risk score (Pocock)), to either 6 months CPAP (ResMed Autoset S8 Spirit), or standard care. CPAP training and fitting was according to local clinical practice. Co-primary outcomes were the mean changes in Epworth Sleepiness Score (ESS) and the vascular risk score (comprising age, sex, systolic BP, smoking, diabetes, total cholesterol, height, creatinine, LVH on ECG, previous MI or stroke) from baseline to 6 months (intention to treat analysis). Home BP was measured in triplicate three times daily over 7 days at baseline and after 6 months, and the weekly average was used for further analysis.

Results Of 391 randomised, 14 withdrew or were lost to follow-up and have been excluded from the primary analysis. 347 patients attended for their 6 month visit within the predefined time window. The study groups were well matched at baseline. Median CPAP use was 3.25 h/night. Full data on ESS and the cardiovascular risk score components were available from 341 and 310 patients respectively.

Sleepiness outcome CPAP reduced daytime sleepiness (mean (SE) ESS change with CPAP −1.68 (0.24); control +0.32 (0.22), mean difference −2.00, 95% CI −1.37 to −2.64, p<0.0001), a cost effective outcome (UK NICE criteria).

Cardiovascular risk outcome CPAP did not reduce cardiovascular risk score (mean (SE) cardiovascular risk score change with CPAP +0.08 (0.17); control −0.37 (0.17), mean difference +0.45, 95% CI −0.03 to +0.93, p=0.064); the small increase with CPAP is not clinically significant.

Conclusions 6 months of CPAP in minimally symptomatic OSA is associated with a cost effective reduction in daytime sleepiness, but does not reduce calculated cardiovascular risk.

Abstract S13 Table 1 Baseline values

	Standard care mean (SD)	CPAP mean (SD)
Age (years)	57.6 (7.5)	57.8 (7.2)
BMI (kg/m^2)	32.5 (5.6)	32.2 (5.6)
ESS	8.01 (4.15)	7.95 (4.42)
ODI (events/h)	12.7 (11.3)	13.8 (12.9)
Cardiovascular risk score	34.9 (7.9)	34.3 (7.5)

S14 CPAP IMPROVES ENDOTHELIAL FUNCTION IN MINIMALLY SYMPTOMATIC OSA PATIENTS: RESULTS FROM THE MOSAIC TRIAL

doi:10.1136/thx.2010.150912.14

[1]M Kohler, [2]S E Craig, [3]J C T Pepperell, [2]D Nicoll, [4]D Bratton, [4]A Nunn, [2]R J O Davies, [2]J R Stradling. *[1]Sleep Disorders Centre and Pulmonary Division, University Hospital Zurich, Zurich, Switzerland; [2]Oxford Centre for Respiratory Medicine, Oxford, UK; [3]Musgrove Park Hospital, Taunton, UK; [4]MRC Clinical Trials Unit, London, UK*

Background CPAP treatment for symptomatic OSA improves surrogate markers of cardiovascular risk, such as endothelial function and arterial stiffness, and may reduce actual cardiovascular events. Minimally symptomatic OSA is far more prevalent than symptomatic OSA but the effects of CPAP on endothelial function and arterial stiffness in minimally symptomatic patients are not known.

Methods In two centres taking part in the MOSAIC trial (Oxford and Taunton), 253 patients with minimally symptomatic OSA (ODI>7.5 h) were randomised to either 6 months of CPAP or supportive care. 245 patients had measurements of arterial stiffness by pulse wave analysis at baseline (augmentation index, AIx) and in 64 patients endothelial function was assessed by brachial artery flow-mediated dilatation (FMD, expressed as % change from baseline arterial diameter) measurements by ultrasonography. Multivariable analyses adjusting for baseline FMD or AIx, ODI and Pocock vascular risk score (age, sex, systolic BP, smoking, diabetes, cholesterol, height, creatinine, LVH, previous MI or stroke) were performed to assess the effect of CPAP treatment on FMD and AIx.

Results Of the 245 patients 8 withdrew or were lost to follow-up and in 8 patients pulse wave analysis was not possible at 6 months. All 64 patients who had FMD measurements at baseline attended

follow-up measurements. Baseline characteristics of 229 patients with complete data on pulse wave analysis are shown in the table (values are mean (SD) were applicable). CPAP improved endothelial function (FMD at follow-up +1.97% with CPAP compared to control group, 95% CI +0.84 to +3.09%, p=0.001), but there was no evidence of an effect on arterial stiffness (AIx at follow-up −0.56% with CPAP compared to control group, 95% CI −2.87 to +1.75, p=0.64). CPAP improved daytime sleepiness as assessed by the Epworth sleepiness score (mean (SE) change −1.91 (0.30) with CPAP; control group +0.08 (0.26), mean difference −1.99, 95% CI −2.77 to −1.21, p<0.0001, assessed using an unpaired *t*-test).

Conclusions 6 months of CPAP is associated with improved endothelial function, but does not reduce arterial stiffness in minimally symptomatic OSA. Thus patients with minimally symptomatic OSA may benefit from CPAP therapy in terms of cardiovascular risk reduction.

Abstract S14 Table 1

Variable	CPAP group	Control group
Age (years)	58.24 (7.21)	57.90 (7.55)
Male/females	97/19	97/13
BMI (kg/m^2)	32.69 (5.57)	32.58 (5.37)
ESS	8.39 (4.12)	8.55 (4.31)
ODI (events/h)	13.98 (14.14)	13.40 (11.38)
FMD (%)	3.41 (3.41)	3.42 (2.36)
AIx (%)	27.56 (9.32)	29.09 (10.54)

S15 AN OFFICE BASED ADVANCED DRIVING SIMULATOR TO ASSESS DRIVING PERFORMANCE IN OBSTRUCTIVE SLEEP APNOEA SYNDROME (OSAS): A PILOT STUDY

doi:10.1136/thx.2010.150912.15

[1]D Ghosh, [2]S Jamson, [1]M W Elliott. *[1]St James' University Hospital, Leeds, UK; [2]Institute for Transport Studies, University of Leeds, Leeds, UK*

Introduction Advising patients with Obstructive Sleep Apnoea Syndrome (OSAS) about whether they are safe to drive is challenging. Driving simulator studies have shown that OSAS patients perform poorly (Hack *et al*, 2001) but most simulators are simple, not realistic and in particular lack face validity, for example, multiple off road events during a short run. The Institute for Transport Studies, University of Leeds, host the UK's most sophisticated driving simulator but had also developed a PC based simulator (MiniSim) which incorporates the same realistic graphics and is much closer to 'proper' driving than most existing systems. It thus has the potential to be used in everyday clinical practice. We have investigated whether two parameters, proportion of high frequency steering activity (HFS) and standard deviation of lane position (SDLP), previously shown to be impaired in drivers suffering from fatigue, might predict drivers' behaviour in safety-critical scenarios.

Methods After a practice run, 63 patients (age 53±10, ESS 11±3, ODI 39±19) completed 50 min motorway driving on the MiniSim. Two situations were programmed that required evasive action to avoid a crash. A 'fail' was determined by an outright crash or veering completely out of lane. We compared HFS and SDLP in subjects with 'pass' or 'fail' and with Oxygen Desaturation Index (ODI) and Epworth Sleepiness Score (ESS).

Results (Abstract S15 Table 1) 'Fail' was more likely with worse sleep disordered breathing, but was not affected by subjective sleepiness. Subjects who 'failed' had significantly worse HFS (0.27 vs 0.34, p=0.03) & SDLP (0.58 vs 0.41, p=0.002). Both HFS & SDLP worsened with time. There was also a strong correlation between HFS & SDLP (r=0.51, p<0.0001).

Abstract S15 Table 1 Comparison between patients passing & failing the simulator run (Mean HFS=mean for the proportion of high frequency steering activity in epochs 3,6,7 of the simulator run, Mean SDLP=mean for the standard deviation of lane position in epochs 3,6,7 of the simulator run)

	ODI (Desaturations/h)	ESS	Mean HFS 3,6,7	Mean SDLP 3,6,7
'Pass'	34.97±3.47	12.13±1.02	0.26±0.012	0.41±0.02
'Fail'	46.40±4.38	12.23±1.40	0.31±0.018	0.57±0.05
p	0.043	0.95	0.0406	0.0025

Conclusion These data show that HFS and SDLP have promise as objective markers of poor driving in OSAS patients. They relate to measures of disease severity and to an event which has face validity as an indicator of poor driving in the real world. The patient will not be aware that they are being measured and therefore they have potential for repeated use.

S16 DETECTION OF SLEEP-DISORDERED BREATHING IN CHRONIC HEART FAILURE PATIENTS: UTILITY OF HEART RATE VARIABILITY VERSUS PULSE OXIMETRY?

doi:10.1136/thx.2010.150912.16

[1]N R Ward, [2]M R Cowie, [2]S D Rosen, [1]V R Roldao, [2]J Hooper, [2]T A McDonagh, [1]A K Simonds, [1]M J Morrell. *[1]Clinical and Academic Unit of Sleep and Breathing, Royal Brompton Hospital and National Heart and Lung Institute, Imperial College, London, UK; [2]Clinical Cardiology, Royal Brompton Hospital and National Heart and Lung Institute, Imperial College, London, UK*

Introduction and Objectives Sleep-disordered breathing (SDB) is a frequent comorbidity in chronic heart failure (CHF). Patients are often asymptomatic and sleep studies may be required for SDB diagnosis. Our department has previously reported that %VLFI component of Heart Rate Variability (HRV) is correlated with apnoea-hypopnoea index (AHI) in CHF (r=0.52). Thus, we tested the hypotheses that %VLFI component of HRV, or pulse oximetry, can be used to rule out SDB in patients with CHF.

Methods Stable CHF patients attending cardiology clinics were enrolled, irrespective of cause or severity of CHF. Patients were studied using polysomnography, simultaneous ambulatory electrocardiography and pulse oximetry. SDB was defined as AHI ≥15.0/h, measured by polysomnogram. Fourier analysis of the electrocardiogram was used to measure %VLFI component of HRV, with a cutoff ≥2.23% to indicate SDB. The oxygen desaturation index (ODI) ≥3% was measured by pulse oximeter, with a cutoff >7.5 desaturations/h to indicate SDB. Diagnostic performance of %VLFI and ODI≥3% were calculated, with the polysomnogram as reference standard for SDB diagnosis.

Results 180 CHF patients were studied, seven were excluded due to insufficient sleep (<200 min). In 173 CHF patients (mean (SD) age 66.9 (13.0) years; 86% male; Epworth Sleepiness Scale 7.6 (4.3); NYHA 2.1 (0.6); median (IQR) BNP 118 (55–239) pg/ml), SDB was present in 77 (45%) patients with mean AHI 32.4 (18.2)/h. %VLFI was measured in 77 (45%) patients: in CHF patients with SDB (n=36), mean %VLFI was 3.13% (2.4) compared to 3.25% (2.6) in patients without SDB (n=41). Cardiac pacing, atrial fibrillation and frequent ectopy prevented %VLFI measurement in the remainder. ODI ≥3% was measured in 171 patients: in CHF patients with SDB (n=76), mean ODI ≥3% was 29.2 (17.2)/h compared to 10.2 (6.4)/h in patients without SDB (n=95).

Conclusion The %VLFI component of HRV has no utility to screen for SDB in patients with CHF. Moreover, it could not be measured in more than half of this cohort of patients. In contrast, the high sensitivity and negative predictive value of the ODI ≥3% suggest pulse oximetry is a valuable tool to rule out SDB in CHF patients.