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.

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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?

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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

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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