Introduction: Hypoxaemia consequent to sleep-disordered breathing, measured using pulse oximetry and quantified by the percentage of total sleep time SpO2 ≤90% (T90), increases the risk of all-cause mortality in men, but not women. However, SpO2 values ≤90% are rare in women. Here we tested the hypothesis that a greater percentage of total sleep time with SpO2 ≤93% (T93) increases mortality risk in women.

Methods: This project retrospectively analysed the Sleep Heart Health Study in which participants >39 years underwent home polysomnography at baseline, with mortality outcomes followed-up for ~11 years. Hypoxaemia was quantified by calculating T90 and T93. Cox’s proportionate hazard regression described the relationship between hypoxaemia (continuous variables) and mortality, adjusting for covariates (age, race, BMI, smoking status, hypertension, diabetes, cardiovascular disease). Data in women are presented.

Results: Studies for 2,773 women were available. The crude mortality rate was 19.5%. Cox regression showed that T93 improved the model relative to T90 alone (p < 0.05) and that younger vs. older women had greater hypoxaemia-related mortality risk (age × T93 interaction, p < 0.05). Age stratified hazard ratios (95% CI) for 45–60, 60–75 and 75–90 years were: 1.22 (1.11–1.33) per 10% increase in T93, 1.05 (1.01–1.09), and 1.03 (0.99–1.07) respectively. Thus, among women aged 45–60 years, those with T93 = 56% (90th centile of population) have 3 times the mortality risk compared with those with T93 = 0%.

Discussion: An increased proportion of sleep with relatively mild hypoxaemia (SpO2 ≤93%) is associated with increased risk of all-cause mortality in middle-aged women. Quantifying T93, rather than T90, may help identify middle-aged women whose sleep-disordered breathing places them at particular risk of adverse long-term outcomes.

Background: A recent systematic review regarding the association between obstructive sleep apnoea (OSA) and post-operative complications concluded that evidence in this area was lacking and that further well designed studies were needed. We designed a study to overcome several methodological limitations of previous studies by using subjective and objective tests to identify the presence and severity of OSA and linked these assessments to post-operative complications.

Methods: Patients referred to the pre-operative anaesthetic clinic were screened for OSA using the STOPBang questionnaire and a domiciliary Apnealink sleep study. Surgeons and anaesthetists performing the procedure were blinded to the results. The primary outcome was a composite of cardiac and/or respiratory complications or oxygen use in the post-operative period. Type of surgery, co-morbidities, use of opioid analgesia and hospital length of stay were recorded.

Results: 35 controls (mean AHI = 2, male = 31%, median age = 49, mean BMI = 29) and 104 OSA subjects (mean AHI = 31, male = 53%, median age = 66, mean BMI = 34) were included in this interim analysis. Subjects with OSA had higher rates of overall complications (41/104) than controls (7/35), p = 0.037. This was largely due to the higher oxygen requirements in the OSA group compared with controls (p = 0.046). Serious complications including atrial fibrillation, bradycardia and hypotension occurred in 5 subjects with OSA and no controls. There were no episodes of post-operative respiratory failure.

Conclusions: Interim analysis showed that subjects with OSA have higher rates of peri-procedural complications with serious events only occurring in those with OSA. These results confirm previous findings that OSA may be associated with increased cardio-respiratory complications. A strength of our study is the objective diagnosis of OSA in the pre-operative period and the blinding of anaesthetists and surgeons to this diagnosis.