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Moderate sleep restriction in treated OSA participants: greater impairment during monotonous driving compared with controls

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ABSTRACT

Objectives: To examine the effect of monotonous driving on a realistic simulator, of sleep restriction in OSA patients treated by CPAP, compared with healthy controls.

Method: In a repeated measure counterbalanced design, 19 CPAP treated, compliant male OSA treated patients (OPs) age 50-75y were compared with 20 healthy age-matched controls. All participants completed a 2h afternoon, simulated, realistic monotonous drive in an instrumented car, following a normal night's sleep or sleep restricted to 5h (OPs remained on CPAP). Driving was monitored for sleepiness-related minor and major lane deviations. EEGs were recorded continuously and subjective sleepiness ratings taken frequently.

Results: After usual sleep there were no between groups differences in driving performance, nor in the ability to detect own sleepiness, when groups 'safely' drove without major deviation for approximately 90min. However, sleep restriction had a greater effect on OPs, whose 'safe driving' time was significantly shorter (65min), than the little change for controls. At this point, EEGs in the OPs indicated that apart from sleepiness, they were also applying more compensatory effort ostensibly to maintain alertness. This might explain their subjective underestimate of this enhanced sleepiness. Nevertheless, apart from this caveat, then for the OP group as well as the controls, there were generally close associations between subjective sleepiness, likelihood of a major lane deviation, and EEG changes indicative of sleepiness.

Conclusions: With adequate sleep, effectively treated people with OSA appear as safe as control drivers, but after shortened sleep and despite realising their sleepiness was greater, albeit still similar to controls, this seems to be an underestimate as their driving impairment was much worse than that for the controls.

Key words

Driving performance, driver sleepiness, obstructive sleep apnoea, perception of sleepiness, CPAP, sleep restriction

INTRODUCTION

Many patients with obstructive sleep apnoea (OSA) treated by continuous positive airway pressure (CPAP) are usually quite fit to drive, as reflected by post-treatment reductions of road traffic collisions (1,2), and at similar levels to those of healthy controls(3). Although some OSA treated patients (OPs) seem to have residual sleepiness (4,5), this may well be similar to that of healthy controls (6). Whilst road accident surveys comparing OPs with controls have equivocal findings, they tend not to match for age or annual mileage (7), or assess and compare the seriousness of the collisions (2).

There have been several laboratory-based driving simulator studies ascertaining driving ability in OPs. However, simulators vary in realism, from having to follow a simple winding road displayed on a standard computer screen and tracked by using a replica steering wheel, to the few advanced simulators utilising an instrumented, static real car with a full-size interactive road simulation viewed through the windscreen; clearly, the more realistic, the better. Other key variables are the time of day of driving and the extent to which the task is monotonous. Sleep-related collisions are most evident early morning and during the afternoon 'bi-circadian dip' (8-10), and associated with long, unstimulating driving conditions. Although, for example, the UK Department for Transport recommends car drivers take breaks from driving every 2h, and the European Union allows truck drivers to drive for 4.5h without a break, many of these simulator studies only last around 20-30 min (eg. 11). Moreover, if a study only provides a minimal practice beforehand, then even the most tedious tasks can have initial intrinsic novelty, thus motivating the participant to apply more effort. Other studies have taken place during the morning, when alertness is naturally on an incline. Thus, under these various experimental conditions it is possible for CPAP treated drivers to display normal driving behaviour, when the testing circumstances are inadequate to detect sleepiness under more realistic scenarios. Even those studies depending solely on driver reaction times can be misleading, as we have shown (12), and where drivers can be unduly distracted by this task and fail to anticipate hazards. Besides, reaction times do not generally 'slow down' in sleepy drivers, but are either normal or nil (as in a lapse or microsleep).

Few studies have utilised advanced driving simulators to study post CPAP effectiveness on OPs. Using a variety of neuropsychological measures, as well as an advanced simulator, Orth et al (13) assessed the effects of CPAP on 31 male OPs. After a 15min practice, a one hour monotonous drive during either mid-morning or late afternoon, towards the circadian daily alertness peak, showed significant and marked improvements 42 days after initiation of CPAP, although there was no healthy control group. Driving ability bore no relationship with any of the neuropsychological tests, nor with polysomnographic determined sleep parameters, leaving the authors to conclude that, "car driving and its simulation is a comprehensive task which cannot be reflected in neuropsychological testing investigating specific aspects of attention, one at a time" (p 901)

Inasmuch that undiagnosed OSA sufferers usually experience chronic sleepiness, it is feasible that any acute addition to their sleepiness (eg. a curtailed night's sleep) is more likely to be masked than in the case of a healthy driver, and with less forewarning of an impending episode of falling asleep at the wheel; although, this is a matter that still requires further exploration. Whilst this may no longer apply to compliant OPs effectively treated by CPAP, whose excessive daytime sleepiness is largely eliminated (and their usually being permitted to drive), there will be occasions of a night's shortened sleep, maybe for work or family reasons when, like any healthy person, they are liable to experience increased sleepiness, particularly early afternoon. The issue then arises as to their ability to detect this transient increase in sleepiness, and whether it is sufficient for safe driving. No driving study has assessed curtailed sleep in treated OPs still maintaining CPAP, and this is the focus of the present study.

More specifically, the main aim of this study is to investigate the impact of modest sleep restriction on realistic driving ability in OPs compared with healthy controls and, secondly, to determine their state of subjective sleepiness when compared with objective EEG and driving performance measures. This will involve 2h periods of monotonous driving in a real-car interactive simulator, including continuous EEG measurement as a further bench-mark for sleepiness (an index seldom used in simulator studies), together with on-line subjective assessments of sleepiness.

METHOD

Participants

OPs were recruited from a local sleep apnoea patients association, comprising those diagnosed with OSA at the regional sleep clinic, who had received CPAP treatment, were pleased with this, and had joined this voluntary support network. Recruitment was as follows: 170 letters outlining the study, containing a preliminary questionnaire (including the Epworth Sleepiness Scale – ESS – ref 14), and asking for volunteers, were sent out to men in the association, aged 50-75y. 103 complied, and an initial screening was undertaken by phone. Seventy had ESS scores <10 (ie. not indicative of excessive daytime sleepiness- EDS), of whom 32 were apparently in good health, drove >3h per week and lived within 40km of our Centre, and were invited for screening and interview. All were being treated by CPAP and had been attending the clinic annually for check-ups. The structured interview comprised 39 questions further relating to health and medications liable to affect sleep and sleepiness (including CPAP usage), as well as habitual sleep characteristics, coffee and alcohol intake. Height, weight and percentage body fat (body composition analyzer TBF-300 - Tanita Corporation - Tokyo) were recorded. They also underwent a computer based driving hazard perception test, and a 30 min familiarisation drive in the simulator. On the basis of this screening, 10 decided not to continue (mostly for reasons of inconvenience), and two were asked not to participate as they were not sufficiently proficient at

driving the simulator. This left 20 remaining volunteers (another later withdrew for work reasons, leaving 19). All had been on CPAP treatment since their diagnosis, for an average of 7.5y.

Healthy, age matched male control participants were recruited via advert in local papers and 'Rotary Clubs'. Respondents were sent similar letters and questionnaires to those of the patients. Interested respondents underwent the preliminary screening by phone. Their sleep was screened for sleep disturbance by actimetry, but full PSGs were not undertaken. These participants were also selected for absence of risk factors associated with OSA: low BMI, non smokers, bed partners reporting infrequent snoring etc. Those with BMIs ≤28 then underwent the identical procedures to those for the patient group, and 20 were eventually recruited. Further details of both groups can be seen in Table 1.

The study met with the full approval of the University's Ethical Committee. For both experimental phases (see below), informed consent was obtained after full explanation of procedures. All participants were collected from and returned to home via taxi. All participants were given a small cash gift on completion.

Design & Procedure

All participants completed two conditions (normal night's sleep, and sleep restricted to 5h by delayed bed-time), in a counterbalanced design, with each condition 1-2 weeks apart. To ensure compliance with sleep instructions, participants wore wrist actimeters (Cambridge Neurotechnology, UK) for three nights prior to each experimental day, when they also kept daily logs of: estimated sleep onset, and morning wakening and rising times. No alcohol was consumed 36h prior to each drive, and nil caffeine after 18:00h the evening before. Participants refrained from eating after 10:00h on the morning of the drive, and had consumed only a light breakfast. On arrival at the laboratory, at 13:00h, they were given a light lunch of two cheese rolls and a glass of water. Actimeters were downloaded to verify that they had complied with the previous night's sleep requirements. At 13:15h electrodes (see below) were applied and they went to the simulator (see below) at 13:50h, and given 10 min to settle into the car. The 2h drive began at 14:00h and lasted 2h, with the shortened night's sleep having enhanced the afternoon dip. They were instructed to drive in the left hand lane (unless overtaking – in the UK we drive on the left) at a speed appropriate for the road and to enable full control of the vehicle. During the drive the investigator remained in the room, out of sight, but there was no communication between investigator and participant once the drive had begun.

Apparatus

Car Simulator – This comprises an immobile car with a full-size, interactive, computer generated road projection of a dull monotonous dual carriageway; each having two lanes. The image was projected onto a 2.0m x 1.5m screen, located 2.3m from the car windscreen. The road had a hard

shoulder and simulated auditory 'rumble strips' (incorporated into white lane markings) either side of the carriageway, with long straight sections followed by gradual bends. 'Crash barriers' are located either side, beyond the rumble strips. Slow moving vehicles are met occasionally, which have to be overtaken. Lane drifting is the most common manifestation of sleepy driving, and a car wheel touching (or crossing) the left rumble strip or right side lane line was identified as a driving 'incident'. Split-screen video footage of the roadway and driver's face (filmed by an unobtrusive infrared camera) enabled the cause of the incident to be determined. Those caused by sleepiness (e.g. eye closure, eyes rolling upwards or vacant staring ahead) were logged as a 'sleep-related incident'. As a further check for the latter, the electroencephalogram (EEG) and electrooculogram (EOG - see below) were examined respectively for alpha/theta intrusions and confirmation of any "eye rolling." Non-sleep related incidents (driver distraction, fidgeting or looking around) were excluded.

Sleep-Related Driving incidents **-** After scrutinising all incidents to exclude non-sleepiness causes, two levels of lane drifting were identified: *Minor incident* – one or two car wheels touching a lane line. *Major incident* – all four wheels out of the driving lane.

Subjective Sleepiness – Every 200s during the drive, participants were verbally prompted by the computer system to report their subjective sleepiness, and their numerical response was based on the 9-point Karolinska Sleepiness Scale (KSS): 1=extremely alert, 2=very alert, 3=alert, 4=rather alert, 5=neither alert nor sleepy, 6=some signs of sleepiness, 7=sleepy, no effort to stay awake, 8=sleepy, some effort to stay awake, 9=very sleepy, great effort to keep awake, fighting sleep. The scale was located on the car's dashboard and continuously visible to the driver. This prompting and its response quickly became routine for the driver.

EEG and EOG - Electrodes were attached for two channels of EEG, with inter-electrode distances carefully maintained by using the '10-20 EEG montage' (main channel C_3-A_1, backup channel C_4-A_2). There were two EOG channels (electrodes 1cm lateral to and below left outer canthus and 1cm lateral to and above right outer canthus; both referred to the centre of the forehead). EEGs and EOGs were recorded using 'Embla' (Flaga Medical, Iceland) and spectrally analysed using 'Somnologica' (Flaga) in 4s epochs. EEG low and high band-pass filtering at >20Hz and <4Hz removed slow eye movements and muscle artefact. In these circumstances, greater EEG power in the alpha (8-11Hz) and theta (4-7Hz) ranges reflect increased sleepiness (15-17). We also measured beta EEG (13-20Hz) power as there is good evidence that this positively correlates with mental effort (18,19) and, in our case, suggestive of trying to stay awake (20-22). EEG power in the 4-11 Hz (theta+alpha) range was averaged in one minute epochs, as was beta activity. To accommodate for individual differences, and to allow comparisons between conditions, each individual's power in these ranges was standardised for all conditions, by taking the difference between each minute's epoch and the individual's mean value over the first 30min of baseline

data, and dividing this by the standard deviation around the mean of that 30min of data. This can mean that when 4-11Hz power is particularly low, values may appear initially to be negative. (23).

Statistical Analyses

Mixed model, repeated measures analysis of variance (ANOVA) were utilised, with one between-subject (groups) and two within subject factors: i) Condition – two levels: normal sleep and sleep restriction; ii) Duration of driving – four levels: 0-30 min, 30-60min, 60-90min and 90-120min. Huynh-Feldt (ε) adjustments were used if sphericity was not assumed. Where appropriate square root transformations corrected for skewed driving incident raw data. For all measures, data was collapsed into 30 minute epochs for statistical analysis

RESULTS

Driving Incidents

Minor Incidents – (Figure 1) - There were significant effects for: between conditions [$F(1,37)=22.3, p< 0.001, \varepsilon = 1$], with both groups having more minor incidents following sleep restriction; and for duration of drive [$F (2.0,75.2)=26.9, p< 0.001, \varepsilon=0.68$]. There was no significant difference between the two groups, nor for any significant interactions. However, there was a trend for a greater number of minor incidents in the middle hour of the drive for OPs during sleep restriction.

Major Incidents – (Figure 2) Significant effects were found: between conditions [$F(1,37)=20.8, p< 0.001, \varepsilon=1$]; and for duration of drive [$F (1.7,63)=15.0, p<0.001, \varepsilon=0.57$]. Of particular importance was the significant group by condition interaction [$F(1,37)=9.4, p<0.05, \varepsilon=1$], with OPs more impaired by sleep restriction.

Time to first major incident (safe driving time) - After normal sleep the control group drove for an average of 97min (s.e. 8min) and the OPs for 89min (7min). Following sleep restriction these values declined to 91min (9min) and 65min (10min) respectively, resulting in a significant difference between conditions [$F(1,37)=4.0, p<0.05, \varepsilon=1$], and a significant group by condition interaction [$F(1,37)=4.2, p<0.05, \varepsilon=1$], with the OPs being more affected by the restriction.

Subjective Sleepiness

KSS – (Figure 3 - mean changes per 200 sec). The effect of condition (30min epochs) was significant as expected [$F(1,37)=19.9, p<0.001, \varepsilon=1$], as was time [$F(1.9,72.1)=70.0, p<0.001, \varepsilon=0.65$]. There were no significant differences between groups, or any interactions.

EEG

Theta+Alpha Power – There was no significant differences between groups or conditions (30 min epochs), but there was a significant time effect [F (1.7, 64.2)=35.0, p<0.001, ε=0.58], as can be seen in Figure 4, showing changes with time, plotted per minute. There was a significant group by time interaction [F (1.7, 64.2)=4.27, p<0.05, ε=0.58] with OPs having increasingly greater theta+alpha power during the last hour under both sleep conditions.

Beta Power – There was a significant effect with time (30 min epochs) [F (1.8, 65.6)=5.8, p<0.05, ε=0.59], but no significant overall effect between conditions or groups. However, there was a significant group by time interaction [F (1.8,65.6)=4.9, p<0.05, ε=0.59], with OPs having progressively more beta power than the control group, beyond the first 30min. This can be seen clearly in Figure 5 showing data per minute. More specifically, between minutes 60-65 of sleep restriction, and just before the mean onset of the first major incident for OPs (see above), beta power was significantly higher than at the same time for controls [t (37)=2.0, p= 0.029 – one tail] suggesting that OPs may have been increasing compensatory effort.

Associations between Measures

Comparisons between Figures 3 and 4 show that for all conditions there are close associations between subjective and objective sleepiness as reflected by the KSS and EEG alpha+theta respectively. When the EEG data are compressed into 200 sec epochs and compared with the corresponding KSS values within each of the four conditions, all 'Pearson's r' values are above 0.8, and very highly significant (df 34; p<001). That is, participants seem to have good self-insight into their physiological sleepiness as determined by the EEG. However, whilst both groups show similar KSS changes within conditions, especially with KSS scores for both groups being similar with sleep reduction, it is clear that reduced sleep in OPs impacts on major incidents to a to a far greater extent (Fig 2), indicating that they may be more sleepy than they realise, despite what might appear to be compensatory effort, as reflected by beta power. Figure 6 is a composite of Figures 2 and 3 (with KSS compressed into 30 min epochs), and clearly illustrates this point further. That is, despite the close parallels with KSS scores between the two groups within conditions, there is approximately a six-fold increase in major incidences following sleep reduction in the OP group.

DISCUSSION

Ours was a prolonged drive under monotonous but realistic conditions and at a time of day that, together, were designed to eke out potential driver sleepiness monitored by a variety of sensitive methods including EEG. After a normal night's sleep, driving ability in the OP group was comparable to that of the controls; thus adding to the evidence from previous reports of no significant difference between the driving performances of CPAP treated OSA participants and healthy controls (24-28).

Nevertheless, following moderate sleep restriction, at a level likely to be encountered on occasions in everyday life by both groups, OPs had significantly more major driving incidents and a shorter 'safe' driving duration. Although no overall difference was found in subjective sleepiness, here, between the groups, EEG activity showed OPs to be physiologically sleepier (greater alpha and theta activity) under both normal and restricted sleep conditions in the second half of the drive, as was power in the beta range, suggesting they applied more effort to remain alert. The combination of these two somewhat antagonistic aspects of the EEG may be responsible for the apparent underestimate of subjective sleepiness by this group when their driving was clearly becoming impaired under sleep restriction.

The occurrence of 'major incidents' on our simulator does not imply that a collision was imminent, but that driving had reached a potentially 'dangerous level'. No driver actually collided with a slow moving car or hit the 'crash barriers' (see 'apparatus'). Of course, there was no danger to the driver, here, as would be the case under real conditions, and it is possible that both groups would have been able to drive more 'safely' for longer under real conditions. However, it remains likely that following sleep restriction the OP group would still have been more impaired than normal.

Why OPs should appear to be more vulnerable to sleep restriction in terms of sleepiness impaired driving ability, must remain an open question. However co-morbidities such as obesity or perhaps prior sleep fragmentation/hypoxemia may have induced enduring alterations to sleep mechanisms within the brain. Heightened daytime sleepiness, independent of OSA, and as a result of severe obesity, has been reported (29) for a group of adults having a mean BMI of 45. Although our OPs were generally obese, their mean BMI was much lower than this, averaging 34.5, whereas the controls were within the normal range, averaging 25.5. Despite BMI being a possible confounding factor with our study, when we compared in the OP group, their BMIs with increases in major incidents as a result of sleep restriction, there was no significant correlation.

In conclusion, our findings with a group of CPAP treated OSA patients, free of excessive daytime sleepiness and able to drive normally under monotonous conditions are, nevertheless, more susceptible to increased sleepiness when sleep is shortened, and they may misperceive the extent of this additional sleepiness. Thus, they should be advised accordingly.

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TABLE LEGEND

Table 1. Participant characteristics - mean and standard errors OSA group n=19; Control group n =20. ESS – Epworth Sleepiness Scale (see Method)

	OSA Group	Control Group	T	df	p
AGE (y)	63.8 (1.7)	66.6 (1.3)	1.12	37	0.27
BMI (m^2/kg)	34.5 (1.4)	25.5 (0.4)	5.80	37	<0.001
% FAT	32.4 (1.2)	23.9 (0.8)	5.62	37	<0.001
ESS	5.3 (0.7)	4.7 (0.6)	0.15	37	0.88
USUAL SLEEP (min by actimeter)	460 (8.1)	468 (10.1)	0.93	37	0.36
SLEEP baseline (min by actimeter)	462 (8.3)	477 (11.1)	1.09	36	0.29
SLEEP restricted (min by actimeter)	292 (6.1)	289 (5.9)	0.43	37	0.67

FIGURE LEGENDS

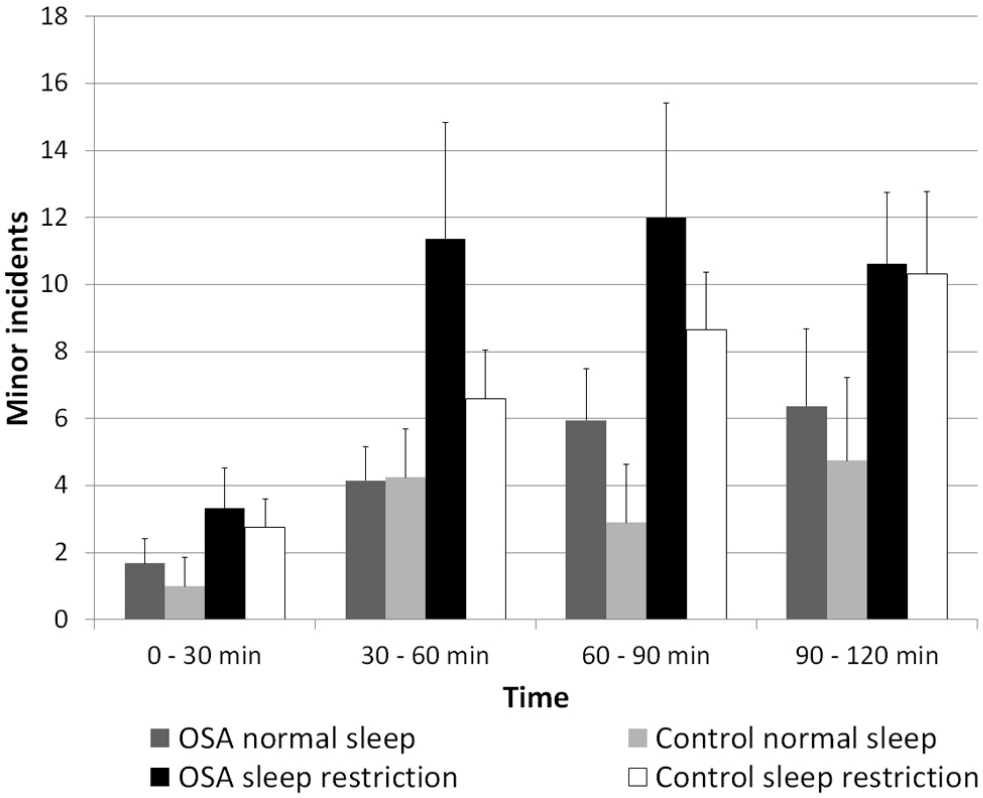

Figure 1 Minor incidents - per 30 min. There were significant between-condition effects.

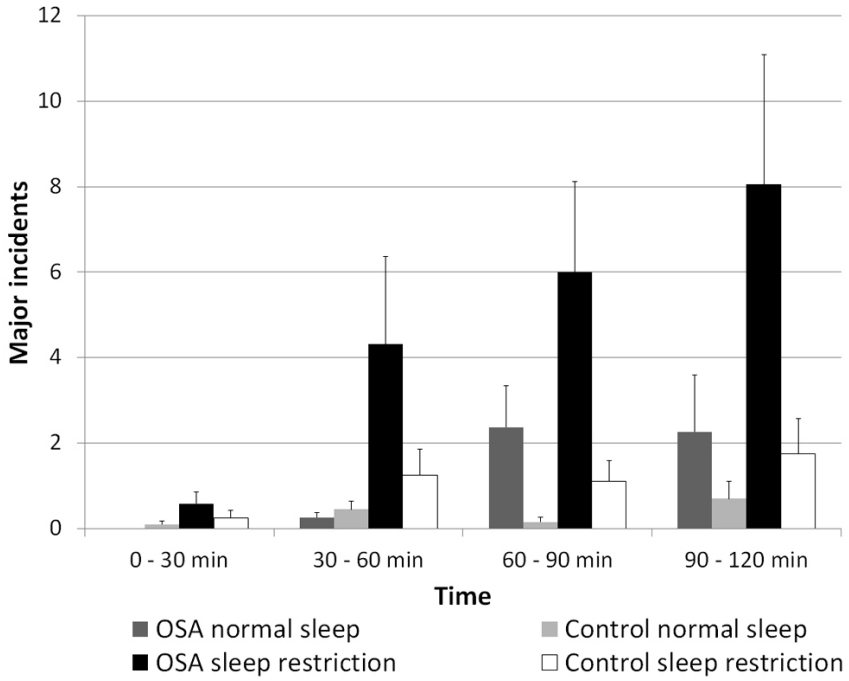

Figure 2 Major incidents - per 30 min. Significant effects were found between conditions, for duration of drive, and the for group by condition interaction, with OSA patients more impaired by sleep restriction.

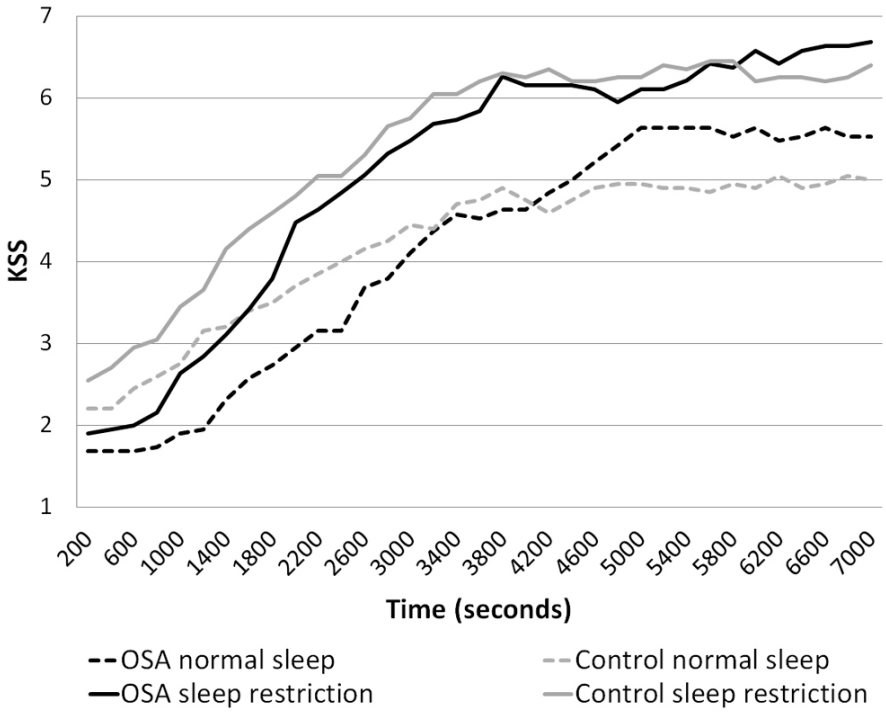

Figure 3 Mean KSS scores - every 200 seconds. There were significant effects for between conditions, and for time at task

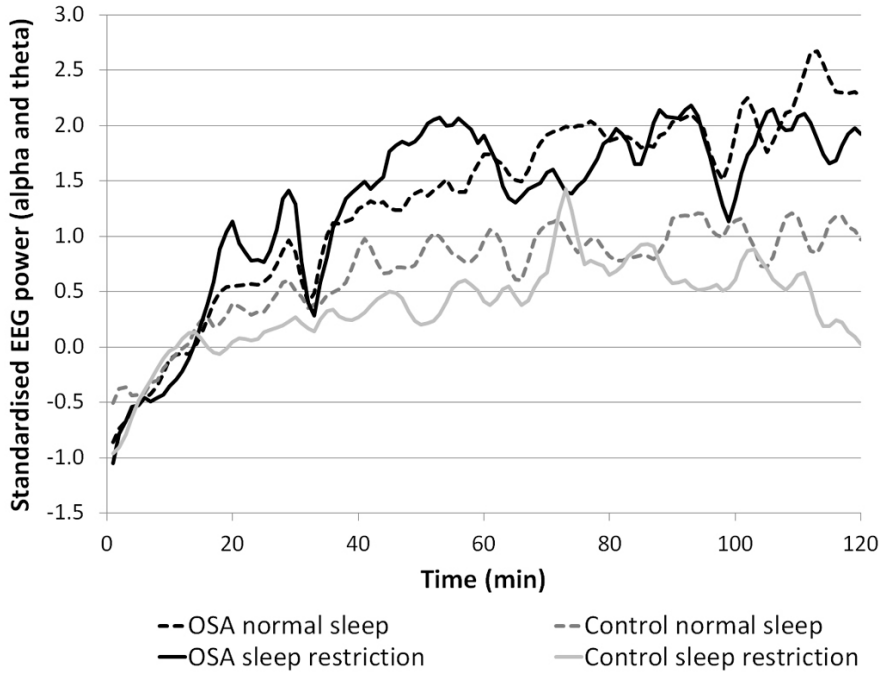

Figure 4 Mean standardised theta+alpha (4-11Hz) EEG power - in 1 min epochs (smoothed by 3 point running average). There were significant time, and group by time interaction effects, whereby OSA patients displayed greater power for the last hour under both sleep conditions.

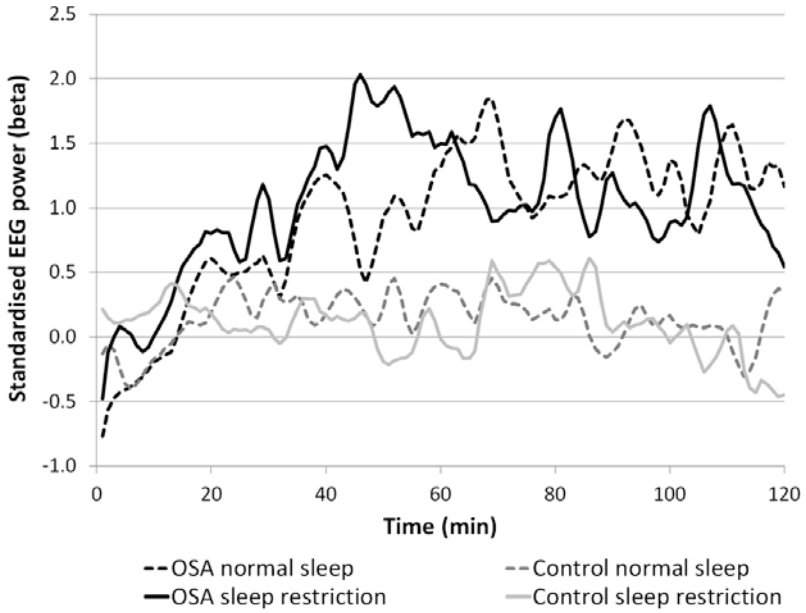

Figure 5 Mean standardised beta (13-20Hz) EEG power - in 1 min epochs (smoothed by 3 point running average). There were significant time, and group by time interaction effects, with OSA patients showing more beta power than the control group, beyond the first 30min.

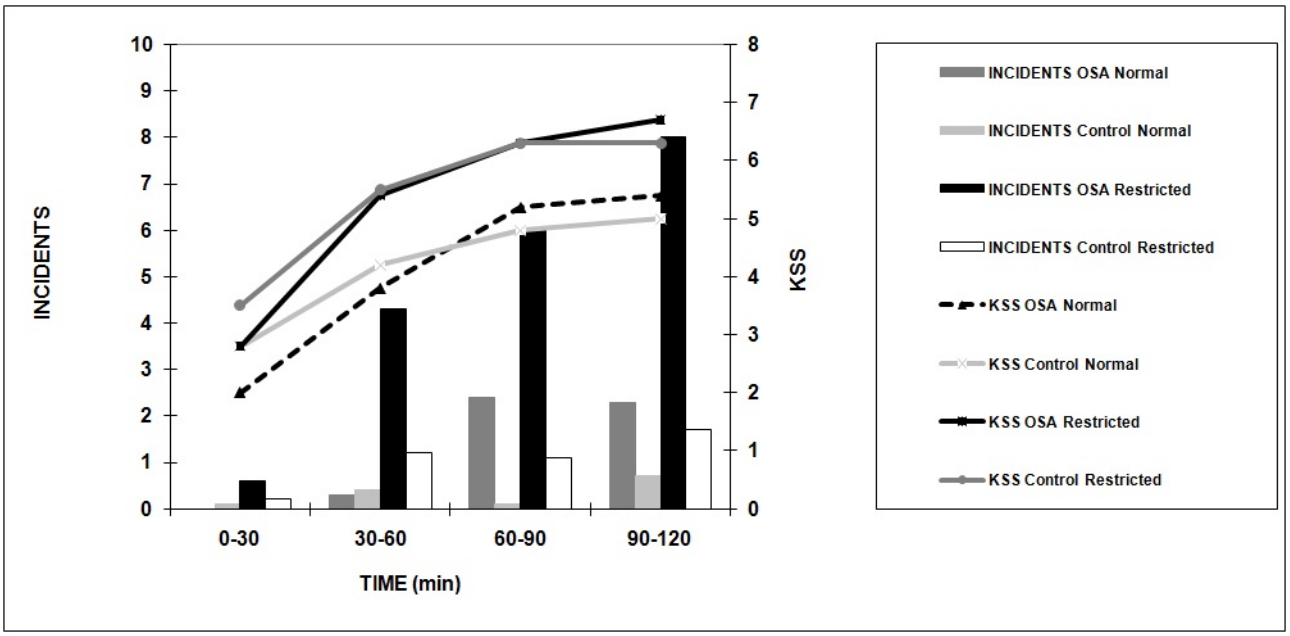

Figure 6 Comparisons of KSS with Major Incidents - in 30 min epochs. Compiled from Figures 2 and 3. Showing more clearly the disproportionately large increase of incidents for the OSA patients after sleep reduction, despite their having similar KSS scores to those of the sleep reduced control group (see Results).