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Comparison of the effects of sleep deprivation, alcohol and obstructive sleep apnoea (OSA) on simulated steering performance

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Patients with obstructive sleep apnoea (OSA) are reported to have an increased risk of road traffic accidents. This study examines the nature of the impairment during simulated steering in patients with OSA, compared to normal subjects following either sleep deprivation or alcohol ingestion.

Twenty-six patients with OSA and 12 normal subjects, either deprived of one night's sleep or following alcohol ingestion [mean (SD) alcohol blood level 71.6 mg dl^{-1} (19·6)], performed a simulated steering task for a total of 90 min. Performance was measured using the tendency to wander (SD), deterioration across the task, number of 'off-road' events and the reaction time to peripheral events. Control data for OSA, sleep deprivation and alcohol were obtained following treatment with nasal continuous positive airway pressure (nCPAP), after a normal night of sleep, and following no alcohol, respectively.

Patients with untreated OSA, and sleep-deprived or alcohol-intoxicated normal subjects performed significantly less well, compared to their respective controls ($P<0·01$ for all tests), with untreated OSA lying between that of alcohol intoxication and sleep deprivation. Alcohol impaired steering error equally throughout the whole drive, whilst sleep deprivation caused progressive deterioration through the drive, but not initially. Untreated OSA was more like sleep deprivation than alcohol, although there was a wide spread of data.

This suggests that the driving impairment in patients with OSA is more compatible with sleep deprivation or fragmentation as the cause, rather than abnormal cognitive or motor skills.

Key words: obstructive sleep apnoea; driving; automobile accidents; steering simulation; sleep deprivation; alcohol.

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Introduction

Patients with untreated OSA have an increased risk of motor vehicle accidents, but why? (1–8). In patients with OSA the repeated episodes of pharyngeal collapse during sleep cause recurrent arousal from sleep, usually accompanied by episodic hypoxaemia. OSA is associated with daytime somnolence and cognitive impairment (9–12), although the relative importance of the disrupted sleep architecture versus any brain hypoxaemia is still debated. This may have important implications, since one might expect the symptoms of sleep fragmentation to improve rapidly following treatment of the OSA (9), whereas hypoxic brain damage might not (13). It is also not clear what aspect of OSA causes patients to have difficulty in driving, and in particular whether it is simply due to reduced vigilance, or other factors such as cognitive or motor impairment that would also reduce the ability to respond appropriately to the incoming information.

Driving performance on simple simulators has been shown to be impaired in OSA, and improves following treatment with nasal continuous positive airway pressure (nCPAP) (14–18). These previous simulations have measured vigilance, reaction time, visual search and tracking, but have not used a realistic view of the road ahead. The complex task of placing a car correctly on the road whilst driving involves two distinct visual tasks, estimating and responding to the oncoming curvature (which allows for faster driving), and controlling the immediate position on the road with reference to the road edges (19). By not including a realistic perspective view of a road curving ahead, the previous simulators have therefore not been able to examine both of the visual tasks of driving.

Many factors, including alcohol consumption, sleep deprivation, certain drugs and previous driving experience will influence the ability to steer a vehicle. It is likely that these factors have a differential effect on the overall performance of driving. Driving performance on simulators

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has also been shown to deteriorate under conditions of sleep loss and alcohol consumption (14,20,21). We have constructed the hypothesis that alcohol will mainly influence cognition or motor skills, sleep deprivation will mainly alter vigilance, and as a consequence they will produce different patterns of performance on a driving simulator.

We have therefore compared the effect of sleep deprivation and alcohol consumption on performance during a simulated steering task, and examined whether in this respect the impairment due to OSA resembles more the effects of sleep deprivation or alcohol.

Methods

SUBJECTS

Twenty-six patients with untreated OSA, with $\geq 10\,h^{-1}$ of $>4\%$ oxygen saturation dips and an Epworth Sleepiness Score (ESS) ≥ 10, were recruited through the outpatient department of our sleep unit following a diagnostic sleep study (Visilab, Stowood Scientific Instruments, Oxford, U.K.). These subjects were the treated arm of a randomized placebo-controlled trial of nasal continuous positive airway pressure (nCPAP) published elsewhere (9). This study demonstrated considerable improvements in driving simulator performance in the nCPAP treated group, and essentially no change in the placebo arm. The more sophisticated driving simulator analysis software used in the current study was not available initially, and was applied to the data after the publication of the earlier study. Obstructive sleep apnoea was diagnosed from a one-night respiratory polygraphic study in a hospital single room decorated to resemble a normal bedroom. Patients' body movements, heart rate and pulse transit time (PTT) changes were recorded as measures of arousal from sleep (22). Arterial oxygen saturation measurements (SaO_2), snoring and respiratory effort [from the PTT (23)] were used as markers of breathing pattern and obstruction. The use of PTT to measure sleep fragmentation and characterize respiratory obstruction has been described elsewhere (24–27). The sleep study results are scored automatically with manual review as required and the all night video is also available for confirmation. The diagnosis of OSA syndrome was established from review of all the data and its severity simply quantified as the number of $>4\%$ SaO_2 dips, as this is one of the best predictors of symptomatic response to nCPAP treatment (26).

Twelve normal subjects, with an ESS ≤ 10 and no history of snoring and no symptoms of OSA, were recruited for the sleep deprivation study from our nursing and student populations. Twelve similar normal subjects were recruited for the alcohol study.

STEERING SIMULATION

The steering simulator is a Q basic computer programme, run on an IBM-compatible PC, and is based on previous work to determine the correct relationship between turning a steering wheel and the effect on the car position (19). The subject steers using a standard computer game steering wheel (Grandprix 1, Thrustmaster, Hillboro, Oregon, U.S.A.). A computer-based image of the moving edges of a pseudo-randomly winding road is portrayed in front of the subject on a 17-inch video monitor, with the bonnet of the vehicle at the bottom of the screen.

The object of the test is to steer the vehicle as accurately as possible down the centre of the road. Each test is a maximum of 30 min and the road display can be the whole road ('all'), the near field ('near') or the distant field ('far'), although for this study these three tests are averaged to provide the results of up to 90 min of simulated steering. If the 'driver' left the road for more than 15 sec it was assumed that he had fallen asleep and the analysis of the run was terminated 60 sec prior to this point, even if subjects subsequently made it back onto the road to finish the drive. This was to stop contamination of the data by the 'off-road' period. Thus, some subjects 'drove' for less than the maximum 90 min.

A visual search task is included to produce a divided attention task. This is achieved by displaying single digits of between one to nine, which change pseudo-randomly at approximately 8–10 sec intervals, at each corner of the screen. The subject is required to scan the four corners while steering and identify a target digit by pressing a button on the steering wheel. All subjects received the same written and verbal instructions on the use of the simulator prior to commencing their training. The length of training was 18 min at each attendance and based on a previous study to determine the length of the learning effect (17). In this previous study, subjects with no previous knowledge of this simulator were given the standard explanation of the test and asked to perform a series of six drives each lasting 6 min. There was significant improvement in steering performance and reaction time between the first three runs, and no further improvement thereafter. This implies that the learning effect is eliminated by 18 min of practice.

ALCOHOL

The alcohol used for this study was 40% vodka, and the volume required was calculated by the equation (28): volume of 40% vodka (ml) = (0·65 × weight (kg)/0·324. The volume of vodka derived was expected to elevate the blood alcohol level to near the legal limit for driving in the U.K., $80\,mg\,dl^{-1}$. The alcohol was mixed with grapefruit juice to reach a volume of 400 ml. The control drink was 400 ml of grapefruit juice.

SLEEP DEPRIVATION

Subjects were studied in the morning, either after a first night on nursing nightduty with no prior daytime nap, or after a supervised night without sleep. This ensured 24 h with no sleep. The control study was after a normal night of sleep.

DESIGN

All untreated patients with OSA performed an 18-min training session followed by three 30-min drives on the steering simulator. The patients were admitted overnight to receive our standard nCPAP induction programme. A trained nurse specialist showed the patients a training video and taught them how to use nCPAP. They then underwent an all night nCPAP titration during which they were monitored in the same way as during their diagnostic study (Visi-Lab System). The therapeutic pressure was established with either the DeVilbiss Horizon or Sullivan Autoset-T auto-adjusting nCPAP machines. The optimal fixed pressure for subsequent use that controlled their OSA and snoring, was confirmed by a skilled technician (29). They were discharged home the following morning with telephone and outpatient support as required. The patients returned after 4 weeks treatment of nCPAP and repeated the training session and the three drives on the simulator.

The subjects in the sleep deprivation study were pseudo-randomized either to a normal night of sleep first, or no sleep for 24 h first, with the randomization balanced so that six subjects received sleep deprivation first, and six subjects normal sleep first. The following morning, subjects performed the usual training session followed by two 30-min drives on the simulator. Only two drives were done on each morning as the nursing staff coming off night duty were keen to get home and allowed us only 1 h of their time. Subjects thus attended on a total of four occasions at eight in the morning to complete 'all' and 'near', or 'all' and 'far' drives, following both a normal night's sleep, and following 24 h of sleep deprivation. The drives were separated by a median (5th/95th centile) of 24 days (10/151) and all four attendances were completed within 8 months.

The subjects in the alcohol study were pseudo-randomized to receive either a grapefruit drink with or without alcohol first, with the randomization balanced so that six subjects received the alcohol first, and six received the control drink first. The subjects performed the usual training session and were given the drink to finish within 10 min. Thirty minutes after finishing the drink the subjects had blood taken for a blood alcohol level, or performed breath analysis on an alcometer (Lion Alcometer S-300, Lion Laboratories, Ty Verlon, Wales, U.K.). This latter device only became available partway through the study and was introduced to avoid blood tests where requested. Subjects then performed three 30-min drives on the simulator in random order. All subjects returned on a separate occasion, separated by a median (5th/95th centile) of 49 (5–117) days to receive the other drink, have blood or breath tests, and perform the usual training and drives. All subjects were asked not to drink any tea or coffee 24 h prior to the drives.

ANALYSIS AND STATISTICS

The initial 60 sec of each test were excluded from the analysis to avoid including any 'settling time'. Position on the road relative to the centre was measured 20 times sec^{-1}. The standard deviation (SD) of this data was used to give a measure of the tendency to wander, or steering error, during the simulated drive. Mean reaction time for the detection of target digits was also measured. 'Off-road' events were also counted, which we defined as occurring when the centre of the vehicle's bonnet crossed the lateral border of the road. The number of these events was quoted per hour of driving to allow for different length drives. The driver 'crashed' if the vehicle remained off the road in this way for more than 15 sec. As mentioned above, in the event of a 'crash' the analysis programme terminated at this point, and the subsequent analysis excluded these last 60 sec to avoid over-estimation of overall tracking error.

In the first analysis we calculated results for steering error, reaction time, off road events, and the length of drive achieved in the patients with OSA before and after treatment, in the normal subjects with and without sleep deprivation, and in the normal subjects with and without alcohol ingestion. In the second analysis, a second set of derivatives was calculated to further characterise the drives. A slope was fitted to the steering error against time, in order to examine performance at the beginning of the drive and to assess deterioration in performance across the 30 min. These were characterized by looking at the SD extrapolated back to time zero (SD0, the impairment present at the beginning of the test), and at the slope of the steering error across the drive (SDslope, the deterioration over time). SD0 and SDslope were also calculated without the final 60 sec of the drive so as to remove the large changes of steering error occurring just prior to finally coming 'off the road' whenever the subject did this before the 30 min were completed.

The significance of any differences within the experimental groups was tested using the Wilcoxon rank test for non-parametric data, with P-values $<0{\cdot}05$ regarded as significant.

Results

The characteristics of the patients and subjects are shown in Table 1. The blood alcohol and breath test levels achieved were mean (SD) 71·6 (19·6) $mg\,dl^{-1}$ and 0·33 (0·14) $g\,l^{-1}$, respectively, with a correlation between the two of 0·79.

The performance of the OSA patients on the steering simulator showed significant improvements after 4 weeks of treatment with nCPAP in steering error, mean reaction time, length of drive achieved and off-road events (all $P<0{\cdot}03$). There were significant differences in these simulation performance parameters in normal subjects following sleep deprivation for 24 h, compared to a normal night of sleep (all $P<0{\cdot}006$). Subjects who had ingested alcohol showed significant differences, compared to their performance without alcohol, in steering error, mean reaction time, off road events and length of drive achieved (all $P<0{\cdot}05$; Table 2).

The steering performance of untreated patients with OSA lies approximately between that of normal subjects with alcohol ingestion, and normal subjects following 24 h of sleep deprivation.

TABLE 1. Characteristics of the study subjects

Median 5/95th centile	OSA patients $n=26$	Subjects to receive alcohol $n=12$	Subjects to be sleep-deprived $n=12$
Age (years)	50·0	20·6	21·0
	37·3/67·5	20·0/21·0	19·6/46·2
Body mass index	32·2	22·1	22.0
	26·3/42·8	20·6/24·4	17·1/34·1
Driving licence (years)	31·5	3·0	3·0
	11·3/49·8	2·0/3·7	1·8/13·5
Epworth sleepiness scale	15·0	7·8	6·0
	10·0/19·8	4·6/10·0	1·1/10·0
Steering error from centre (SD)	0·214 (on nCPAP)	0·128	0·154
	0·15/0·72	0·11/0·20	0·12/0·23

The second set of analyses to characterize the steering impairment also showed some impairment resulting from the three different experimental situations (Table 3). However the effect of the impairment on the intercept of the steering error at the start of the drive (SD0), and on the slope of the steering error across the drive (SDslope), was different between the three experimental groups. Alcohol significantly impaired SD0 ($P=0·005$) but not SDslope. On the other hand, sleep deprivation significantly impaired SDslope ($P=0·002$), but not SD0. OSA patients had significantly impaired SDslope ($P=0·03$), but not SD0, a similar pattern to the sleep deprivation results.

Discussion

This steering simulation detects impairments of performance in three different situations, due to OSA, alcohol ingestion and sleep deprivation, when compared to the relevant 'control situation'; i.e. the same subjects after nCPAP treatment, after no alcohol and after a normal night of sleep respectively. Patients with untreated OSA have significant improvement after nCPAP treatment with a change in the steering error towards normal. The performance of untreated OSA patients lies approximately between that of normal subjects with alcohol ingestion, and that following sleep deprivation. The mean blood alcohol level ($71·6\,mg\,l^{-1}$) and breath alcohol level ($0·36\,g\,l^{-1}$) in the subjects was less than the legal limit in the U.K. ($80\,mg\,dl^{-1}$ and $0·4\,g\,l^{-1}$, respectively) but steering impairment was still detected.

Further attempts at a comparison of the impaired performance data are difficult as the subjects were not matched, those in the alcohol and sleep deprivation arms of the study being younger. It might be expected that the patients with OSA would have more driving experience and would have performed better, making differences between groups harder to detect. On the other hand, performance may decline normally with age. The results of steering performance and reaction times for a control group of older subjects without OSA has been published by Juniper *et al.* (17), and are not significantly different to the values for the normal younger subjects without alcohol or sleep deprivation in this study.

Although the OSA patients were part of a randomized placebo control trial of nCPAP (18), and were blind to their treatment, they were nonetheless receiving an intervention. Reversing the order, treatment first and then withdrawing, would not have improved blinding. Clearly, sleep deprivation could not be blinded either. Motivation might be expected to be worse for the drives after the sleep deprived nights, however, all subjects continued to steer the simulator for the full 30 min even when in some cases they had been 'off-road' for 15 sec, sufficient to 'crash' and terminate the analysis programme earlier in the drive. It is also likely that subjects in the alcohol study could detect which drink contained the alcohol, although the use of vodka and grapefruit minimized the smell and taste. When asked after each experiment which drink they thought they had had, four out of the 12 subjects thought their placebo drink contained alcohol, and five out of 12 thought their alcohol drink was the placebo.

It must be acknowledged that for technical reasons the groups performed the tasks at different times of the day, although for each experimental group the times for the drives remained constant at their return visits. The OSA group performed their three test drives at 09.00, 12.00 and 15.00 hours. The normal subjects in the sleep deprived group were tested at 08.00 hours and only for a period of 4 h at a time (two drives) thus requiring four visits to complete the protocol. The normal subjects given alcohol performed all their three test drives at 14.00 hours. This makes it possible that there will be an influence of time of day on the absolute values. However, it is unlikely that there will be a time of day effect on the differences between the control and experimental situations. Thus in order to investigate the different patterns of steering impairment in the three groups, we used the change in performance

TABLE 2. Results of steering performance and reaction time in the three groups

	Effect of OSA				Effect of alcohol				Effect of sleep deprivation			
Median 5/95th centile	OSA nCPAP	OSA untreated	Δ	*P*	No alcohol	With alcohol	Δ	*P*	Normal sleep	Sleep deprived	Δ	*P*
Steering error (SD)	0·214 0·15/0·72	0·364 0·15/1·10	0·089 −0·06/0·91	0·002	0·128 0·11/0·20	0·289 0·14/0·99	0·139 0·01/0·84	0·003	0·154 0·12/0·23	0·476 0·22/0·92	0·308 0·06/0.74	0·002
Off-road events h^{-1}	10.1 0.17/75·7	17·8 0·35/248	10·8 −13·8/96·3	0·004	0·00 0·0/6·43	15·7 0·0/115·3	15·7 0·0/113·0	0·008	0·78 0·0/9·63	29·6 5·74/85·8	26·1 5·74/83·9	0·003
Drive length (min)	30·0 17·5/30·0	24·8 5·36/30·0	−2·53 −11·6/5·83	0·023	30·0 30·0/30·0	30·0 15·9/30·0	0·00 −14·1/0·0	0·043	30·0 30·0/30·0	27·3 20·3/30·0	−2·73 −9·66/0·0	0·008
Reaction time (sec)	2·19 1·47/3·55	2·58 1·75/4·80	0·37 −0·19/1·82	0·0002	2·04 1·41/3·23	3·14 1·65/4·90	0·86 −0·05/2·12	0·004	1·61 1·34/3·06	2·36 1·90/4·29	0·53 −0·15/1·97	0·005

TABLE 3. Results of steering error at the start, and the slope (deterioration) of the steering error

	Effect of OSA				Effect of alcohol				Effect of sleep deprivation			
Median 5/95th centile	OSA nCPAP	OSA untreated	Δ	*P*	No alcohol	With alcohol	Δ	*P*	Normal sleep	Sleep deprived	Δ	*P*
Steering error at time 0 (SD0)	0·109 0·06/0·35	0·116 −0·09/0·41	0·009 −0·16/0·12	NS	0·079 0·06/0·13	0·123 0·08/0·34	0·044 −0·02/0·22	0·005	0·079 0·06/0·13	0·117 0·06/0·17	0·028 0·00/0·25	NS
Slope of SD deterioration (SDslope, SD h^{-1})	0·059 −1·02/0·39	0·175 −1·14/30·3	0·103 −1·41/30·3	0·03	0·008 −0·05/0·04	0·065 −0·54/0·22	0·080 −0·58/0·21	NS	0·051 0·01/0·08	0·389 0·10/2·12	0·350 0·04/2·05	0·002

between 'abnormal' and 'normal', rather than the absolute abnormalities in the affected state. In this way we hoped to reduce the confounding effects due to having unmatched groups and thus explore the effects of the specific abnormality alone.

Normal subjects with alcohol ingestion showed significant impairment in the steering error at the start of the drive (SD0) when compared to no alcohol, which remained unchanged across the drive, showing no further deterioration. This could be interpreted as global impairment caused by the alcohol which is present throughout the drives and thus not affected by the time through the drive. Normal subjects with sleep deprivation show significant impairment across the drive (SDslope) when compared to normal sleep, but are not impaired at time zero, when fresh to the task. This could be interpreted as reduced vigilance caused by lack of sleep which comes on during the drive and progresses, as one would expect (30). Overall, the patients with OSA had performance characteristics which better resemble sleep deprived normal subjects, with the steering error at the start of the drive not being significantly different on or off nCPAP, but with a significant difference in the slope. However, there was a considerable spread of data, with some patients having impaired values for SD0 that improved on nCPAP.

This suggests there is likely to be a different pathophysiological mechanism influencing the simulator performance following alcohol, compared to sleep deprivation, and that on the whole the impairment due to OSA resembles more the effects of sleep deprivation than alcohol. However, the larger spread of data in the patients suggests that they may not be a homogeneous group, and that some of them may have impairments not necessarily resulting simply from sleep deprivation. Possible supporting data for this is that following treatment the patients with OSA do not return their driving error to the low values found for age- and sex-matched normal subjects reported in an earlier publication from this department using the identical simulator (17); driving error (measured as the SD) in treated OSA, median (5th/95th) 0·21 (0·15/0·72), driving error in matched controls, 0·16 (0·13/0·31), $P<0·05$. This might mean there is a residual defect not improved by correction of sleep fragmentation. However, the interpretation of performance data following treatment is difficult given the variation in compliance with nCPAP (31), although the overall compliance in our 26 patients was good, with a median of 5·6 (100%, range 2·5–7·8) h per night.

There is data now available to show that not only do patients with OSA have more car accidents than would be expected (2,3,6,32), but that the accident rate falls following treatment (7,33,34). For example, Findley *et al*. showed a significant fall in 36 patients using nCPAP for OSA, from 0·07 accidents per driver per year to no accidents at all in the 2 years after therapy was commenced ($P=0·03$) (34). Others have also shown that the average number of accidents per patient per year decrease, for real accidents (from 0·17 to 0·07, $P<0·01$) and for near-miss accidents (from 1·2 to 0·1, $P<0·01$) (33). However, it is difficult to know whether the accident rate on adequate nCPAP therapy truly returns to normal, or whether it remains slightly raised; this will require further trials.

In conclusion, steering simulator performance is impaired by 24 h without sleep, by blood alcohol levels a little below the U.K. legal limit for driving, and by moderate to severe OSA. Although the degree of impairment overall is approximately similar, the pattern of impairment is not. On average, alcohol produces impairment across the whole drive with no deterioration with time. On average, sleep deprivation does not affect steering at the start of the drive, but causes progressive deterioration with time. On average, OSA affects steering performance in a way that more resembles sleep deprivation, with clear deterioration across time. Thus, it seems that much of the steering impairment in patients with OSA is likely to be due to poor vigilance, rather than a general cognitive or motor defect, as presumably occurs with alcohol intoxication.

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