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Visual Vigilance in Drivers with Obstructive Sleep Apnea

Jon Tippin, MD[a,c,*], **JonDavid Sparks, MS**[b], and **Matthew Rizzo, MD**[a]
[a]Department of Neurology, University of Iowa, Iowa City
[b]Department of Biostatistics, University of Iowa, Iowa City
[c]Department of Veterans Affairs Medical Center, Iowa City

Abstract

Objective—To determine the effects of obstructive sleep apnea (OSA) on visual vigilance during simulated automobile driving.

Methods—Twenty-five drivers with OSA and 41 comparison drivers participated in an hour-long drive in a high fidelity driving simulator. Drivers responded to light targets flashed at seven locations across the forward horizon. Dependent measures were percent correct (hit rate, HR), and reaction time (RT). Self-assessment of sleepiness used the Stanford Sleepiness Scale (SSS) before and after the drive and the Epworth Sleepiness Scale (ESS).

Results—OSA drivers showed reduced vigilance based on lower HR than comparison drivers, especially for peripheral targets (80.7 +/− 14.8% vs. 86.7 +/− 8.8%, p = 0.03). OSAS drivers were sleepier at the end of the drive than comparison drivers (SSS = 4.2 +/− 1.2 vs. 3.6 +/− 1.2, p = 0.03), and increased sleepiness correlated with decreased HR only in those with OSA (r = −0.49, p = 0.01). Lower HR and higher post-drive SSS predicted greater numbers of driving errors in all subjects. Yet, ESS, pre-drive SSS, and most objective measures of disease severity failed to predict driving and vigilance performance in OSA.

Conclusions—Reduced vigilance for peripheral visual targets indicates that OSA drivers have restriction of their effective field of view, which may partly explain their increased crash risk. This fatigue-related decline in attention is predicted by increased subjective sleepiness during driving. These findings may suggest a means of identifying and counseling high-risk drivers, and aid in the development of in-vehicle alerting and warning devices.

Key terms

Attention; Driving performance; Driving simulators; Obstructive sleep apnea

INTRODUCTION

Sleepy driving has been estimated to cause more than 40,000 crash-related injuries and 1,500 deaths in the US annually [1]. Although sleep-deprived healthy drivers probably cause most of these crashes [2], many are the result of drivers with sleep disorders, such as obstructive sleep apnea (OSA). OSA drivers may be especially unsafe, as recent meta-analyses have shown

*Corresponding author: Department of Neurology, University of Iowa Hospitals and Clinics, 200 Hawkins Drive, 2RCP, Iowa City, IA 52242, USA, Tel. +1 319 356 8753, Fax: +1 319 384 7199, Email: jon-tippin@uiowa.edu.

a mean crash risk ratio of 2.72, indicating that these individuals have a 172% greater chance of a crash relative to the general population [3,4]. Motor vehicle crashes from improperly treated OSA lead to enormous suffering and annual costs that may exceed $11 billion [3].

Some sleepy drivers fall asleep at the wheel, but others have sleepiness-related cognitive impairments, which reduce driver performance and increase the likelihood of driver errors that result in crashes. Safe driving requires the continuous coordination of several cognitive processes, especially attention and perception [5]. Sustained attention (or vigilance) is often impaired in OSA patients [6,7,8,9] and reduced attention, especially to targets in the peripheral fields, is associated with increased crash-risk in drivers with cognitive impairments [5,10,11]. Hence, drivers with visual vigilance impairment due to OSA may be at increased risk for crashing without falling asleep. The potential dangerousness of this situation is heightened by the fact that many of these drivers are unaware of their impairment [12].

This study tested the hypothesis that drivers with OSA have impaired visual vigilance, especially to peripheral targets, compared to normal drivers. We also explored the relationships between visual vigilance, driving control, disease severity and self-reported sleepiness. Understanding these relationships may allow detection of high-risk drivers, and aid the development of in-vehicle alerting and warning devices.

METHODS

Participants

Sixty-six legally licensed drivers participated in this study. These included 25 drivers with OSA and 41 comparison drivers without neurological or sleep disorders. Participants with OSA were recruited from the Sleep Disorders Clinic in the Department of Neurology at the University of Iowa, and met accepted clinical criteria for the diagnosis [13]. Consecutive new patient referrals with a clinical suspicion of OSA were invited to participate in an attempt to minimize selection bias. None were being treated with positive airway pressure (PAP) therapy. Thirty-six subjects meeting clinical criteria for OSA underwent polysomnography (PSG, see *measures of disease severity*). Eleven subjects had an hypopnea-apnea index (AHI) less than 5 events per hour and were excluded from further analysis, leaving 25 subjects with confirmed OSA. Comparison drivers were recruited from visitors to the University of Iowa not seeking medical care, such as family members or friends of patients, and from the general community surrounding Iowa City, Iowa, by means of announcements in local newspapers or public service announcements. Participants in the comparison group were matched to the OSA drivers for gender and age within 5 years. These licensed drivers were screened with the same procedures as the OSA patients, including a complete medical history and physical examination, but did not undergo PSG. Potential comparison drivers were excluded if they had a history of neurological or sleep disorders, any symptoms of OSA [13], or an Epworth Sleepiness Scale (ESS) score greater than 10 (see *self-reported sleepiness*). All potential subjects were excluded if they were no longer driving, were acutely ill, had active, confounding medical conditions such as other sleep disorders, chronic obstructive pulmonary disease, congestive heart failure, dementia, major psychiatric and vestibular diseases, alcoholism or other forms of drug addiction, or used the following medications: stimulants, antihistamines, antidepressants, narcotics, anxiolytics, anticonvulsants and other major psychoactive medications. Potential subjects were also excluded if they consumed seven or more cups of coffee (or an equivalent amount of other caffeinated beverages) daily [14], currently smoked cigarettes [15], had an irregular sleep-wake pattern, or a habitual sleep duration of < 6 or > 9 hours [16]. However, as participants were studied on the same day that they were recruited, it was not possible to document sleep-wake cycles with the use of sleep logs or actigraphy. Individuals with diseases of the optic nerve, retina, or ocular media were excluded only if they had a corrected visual acuity of less than 20/50. Participants with visual field defects defined by Humphrey perimetry

[17] were also excluded. The study received prior approval by the University of Iowa Institutional Review Board and informed consent was obtained from each participant after full explanation of the study procedures. Subjects were compensated $50 (US dollars) for their participation.

Measures of disease severity

PSG and multiple sleep latency test (MSLT) were performed according to standardized protocols [18,19]. Measures of disease severity were the AHI and minimum oxygen saturation from the PSG, and mean sleep latency (MSL) and presence of sleep onset REM periods (SOREMP's) from the MSLT. According to sleep laboratory protocol, patients with an AHI > 40 events per hour after a minimum initial sleep time of 2 hours are started on PAP therapy ("split night" protocol) and do not have an MSLT performed the following day.

Self-reported sleepiness

Self-reported sleepiness was assessed by the Stanford Sleepiness Scale (SSS) [20] and the Epworth Sleepiness Scale (ESS) [21]. The SSS is a standard assessment tool that asks a person to rate his or her current state of sleepiness on a 7-point scale (1 is most alert, 7 is almost asleep). The ESS assess global sleepiness by asking a person to rate on a 0–3 scale the chance that they would fall asleep in 8 situations (0 indicates no chance, 3 indicates a high likelihood). A score of 10 or less is considered normal.

Simulated driving performance

All subjects participated in an hour-long drive in SIREN (Simulator for Research in Ergonomics and Neuroscience), an interactive driving simulator creating an immersive, real-time virtual environment for assessing at-risk drivers in a medical setting [22]. SIREN comprises a 1994 GM Saturn with the running gear removed, embedded electronic sensors, miniature cameras for recording driver performance, a sound system and surrounding screens (150° forward FOV, 50° rear FOV), four LCD projectors with image generators, and computers for scenario design, control, and data collection. A tile-based scenario development tool (DriveSafety, Fort Collins, CO) permits selection of various road types populated with vehicles that interact with the driver and each other. Experimental performance data are collected digitally at 30 Hz and reduced to means, standard deviations, or counts for each virtual road segment. Simulator output includes steering wheel position (in degrees), normalized accelerator and brake position (i.e., scale of pedal depression from 0 to 100%), speed (in miles per hour, mph), and other variables such as position of the car in the lane, and longitudinal and lateral acceleration. Driving performance is captured using miniature cameras to record the scene observed by the driver and provide a backup record of the driver's lane-tracking. Synchronization of the digital and video data facilitates the inspection of artifacts and allows for review of potential driver safety errors.

Prior to beginning the experiment, a "warm-up and training" session lasting 5 to 10 minutes was held to familiarize the drivers with the vehicle controls. Afterward, drivers completed a brief checklist of vehicle knowledge and operations to assure a level of proficiency sufficient to proceed with the experiment. A simulator operator communicated with the driver by intercom during the drive to monitor the driver for signs of discomfort. Subjects drove on a simulated 2-lane highway comprising three identical drive segments. Each drive segment included two straight road types and two gradual curves (radii= 600 meters). There were approximately 24,800 meters of straight road and 5,600 meters of curved sections for each segment. The participants were instructed to drive at the posted speeds of 55 mph (~89 km/h). The drive scenario contained minimal traffic or distractions and was representative of drives that may induce drowsiness. Dependent measures were the standard deviation in lane position (SDLP), lane deviations and speed maintenance errors. The distance between the vehicle

midline and lane centerline indexed lane position error for determining SDLP. SDLP is a frequently used metric that indexes road tracking error or "weaving" [23]. Lane deviations were determined when any part of the vehicle contacted one of the lane boundaries (i.e., road shoulder or oncoming traffic lane). Speed maintenance errors were measured by calculating the number of deviations 10 mph or greater above or below the posted speed limit of 55 mph, as in studies of distracted drivers [24].

Vigilance during driving

Each driver responded to small light targets (200 ms duration) flashed at random intervals between 30 and 90 seconds (averaging one per minute) at seven locations (center; 12.5°, 50°, and 62.5° degrees to each side of center) across the forward horizon in the simulator (See Figure). Drivers responded by clicking the high beam control as soon as they detected the target. Dependent measures on the vigilance task were hits (defined as responses within 3 seconds after a target had appeared), misses (defined by failure to respond within 3 seconds after a target had appeared), false positives (defined as responses when no target had been presented within the previous 3 seconds), and reaction time (RT) in seconds. Hit rates (HR) were calculated from the number of hits divided by the total number of targets. Signal detection correction techniques were used to correct HR for false positive responses [25]. D-prime, the true sensitivity, was computed from the difference between the Z scores for HR (maximum score 99%) and the Z scores for the percent false positives (minimum score 1%). RT's were calculated only for hits.

Testing sequence

Potential OSA participants were recruited at the time of their clinic visit, which occurred between 8 a.m. and noon. Comparison drivers were also recruited and screened on the morning prior to testing. The simulator drive was performed at a fixed time in the afternoon (2 p.m.) to mitigate the confounding effects of circadian fluctuations of alertness [26]. ESS was administered during recruitment, and SSS was performed just before and after the drive. OSA participants underwent PSG that night and MSLT was performed the following day in those who did not undergo a "split night" protocol.

Statistical analysis

Wilcoxon Rank Sum Tests were used to examine the distributions between OSA and control drivers for vigilance, driving performance and subjective sleepiness measures. Performance on the vigilance task as a function of target location was examined using linear mixed models. The relationship between changes in vigilance performance over drive duration was also examined using a linear mixed model by comparing performance between the three drive segments. Residual plots were examined to verify that the normality assumptions of the linear mixed models were adequately met. Spearman correlations were used to determine the relationship between accuracy on the vigilance tasks and change in subjective sleepiness over the drive. ANOVA models were fit to explore relationships between measures of disease severity (AHI in 3 severity groups; 5–15, >15–30, >30, minimum oxygen saturation, MSL) and either of the vigilance measures (HR, RT). Poisson Regression models, which estimate the log of the mean value of the respective response variable, were fit to model the response variables (lane deviations, speed errors) against the predictors of interest; HR, RT, and a measure of sleepiness (SSS before the drive, SSS after the drive, and ESS). Over dispersion occurred in all models suggesting that there was more variability in the data than was accounted for in the model. To account for over dispersion, Pearson scaled standard errors were used. Data are reported as mean +/− SD.

RESULTS

Participants

Table 1 shows the demographic information for the participants. There were no statistically significant differences between OSA and comparison drivers in terms of gender or age. The mean AHI for OSA drivers was 21.2 +/− 19.9 (range 5.07 – 96.57), mean minimum oxygen saturation was 81.0 +/− 8.58% (range 58.0 – 94.0) and mean sleep latency was 10.6 +/− 4.0 (range 2.2 – 16.2). SOREMP's were seen in only one driver, who had 2 associated with an MSL of 4.8. Note that MSLT data were available for only 15 OSA subjects, as the remaining 10 underwent a "split night" protocol on the preceding PSG.

Self-reported sleepiness

OSA drivers were significantly sleepier than controls as evidenced by higher ESS scores (see Table 2). SSS did not differ significantly between groups immediately before the drive, but was significantly greater after in OSA drivers compared to controls. In each group, sleepiness increased, as SSS values were higher after the drive than before ($p<0.01$). However, OSA drivers had significantly higher SSS scores at the end of the drive than did comparison drivers ($p = 0.03$).

Vigilance during driving

Table 3 describes the outcomes on the vigilance task in OSA and comparison drivers. There was no difference in the number of targets presented to each group. Between group analyses showed that HR on the vigilance task was lower in drivers with OSA than in the comparison group ($p = 0.03$). The number of false positive responses was very small; a total of 25 subjects had false positives, but only 5 (7.58%) had more than one and there was no difference in the number of false positives between the two groups ($p = 0.79$). HR correlated with D-prime ($p = 0.03$), indicating that subject responses were not significantly influenced by false positives; consequently, HR was used as the measure of response accuracy in subsequent analyses. There was no significant difference in RT between the two groups ($p = 0.25$).

We also examined driver performance on the vigilance task as a function of target appearance in central versus peripheral locations of the simulator display. Preliminary results showed that the three central locations (center, 12.5 degrees left, and 12.5 degrees right) had similar outcomes for HR and RT ($p = 0.07$ and $p = 0.71$, respectively), as did the two far left locations ($p = 0.08$ and $p = 0.27$) and the two far right locations ($p = 0.15$ and $p = 0.80$). Consequently, we compared the 3 central locations with the 4 peripheral locations (see Table 4). The mean HR's for central and peripheral targets were 0.955 and 0.814 in the comparison group and 0.923 and 0.723 in the OSA group. Both groups performed similarly on the central targets ($p = 0.21$), but the OSA group performed more poorly than the controls on peripheral targets ($p = 0.02$). A mixed model aimed at assessing whether the two groups had similar differences in performance between central and peripheral targets, showed a trend towards different HR based upon target location ($p = 0.09$ for interaction). For RT, both groups performed in a similar fashion on central vs. peripheral targets ($p = 0.84$ for interaction). Adjusting for group status, subjects responded to central targets 0.14 seconds faster than peripheral targets ($p < 0.01$).

In terms of the relationship between changes in vigilance performance and drive duration, HR did not significantly differ between OSA and control drivers over the course of the drive ($p = 0.83$ for interaction), and did not significantly change over time for either group ($p = 0.19$ for main effect of time). Similarly, the trend in RT over the drive was not significantly different between OSA and control drivers ($p = 0.22$ for interaction). There was marginal evidence for a decrease in RT from the first to the last drive segment for both groups ($p = 0.08$). However, this amounted to a mean decline in RT of only 0.046 seconds.

Driving performance

There were no significant differences between OSA and comparison drivers in either the number of lane deviations (49.16 +/− 39.576 vs. 45.976 +/− 38.225, p = 0.72) or speed errors (2.96 +/− 4.996 vs. 5.098 +/− 8.139, p = 0.39), while there was a trend towards higher SDLP in the OSA drivers (0.3697 +/− 0.0920 vs. 0.3288 +/− 0.0827, p = 0.07).

Relationship between vigilance and sleepiness

There were no relationships between self-reported sleepiness and either vigilance measure for either OSA drivers or controls, except for HR, which was inversely correlated with ESS for the entire group (Spearman r = −0.28154, p = 0.02) (See Table 5). However, there was a relationship between changes in HR and sleepiness over time during the drive. In the OSA drivers, we found a negative correlation between change in SSS ("after" minus "before") and change in HR (from the first to the last drive segment) (Spearman r = −0.49, p = 0.01). In other words, the OSA subjects who became sleepier over the drive had lower HR's in the final segment. In contrast, there was no significant relationship between changes in SSS and HR for controls (Spearman r = 0.09, p = 0.59). No significant relationships were found for RT (Spearman r = .13, p = .45 for OSA drivers, Spearman r = −.11, p = .48 for controls).

Relationship between vigilance and objective measures of disease severity (OSA drivers only)

AHI was divided into 3 categories; 5–15, >15–30, >30 events per hour. There were no significant relationships between AHI and either of the vigilance measures; HR (p = 0.11) and RT (p = 0.46). Minimum oxygen saturation did not correlate with RT (r = −0.03, p = 0.8986), but was significantly correlated with HR (r = 0.54, p < 0.01). There were no correlations between MSL and either vigilance measure (HR; r = 0.34, p = 0.22, RT; r = 0.05, p = 0.87). Too few SOREMP's were recorded to make any comment on their relationship to performance on the vigilance tasks.

Relationship between driving errors, vigilance and sleepiness

The main predictors of interest for driving performance were HR, RT, and a measure of sleepiness (SSS before the drive, SSS after the drive, and ESS). SSS after the drive was used as the measure of sleepiness as the other 2 did not have a significant relationship with the driving outcomes. The variables age, OSA status, and gender were looked at as possible confounders. SSS scores after the drive were grouped into 3 levels. Subjects with scores of 1–2 were termed "Awake", 3–4 "Marginally Sleepy", and 5–7 "Sleepy".

Lane Deviations

There were no relationships between lane deviations and age (p = 0.73), gender (p = 0.43) and OSA status (OSA vs. controls) (p = 0.75), so these variables were omitted from the final Poisson models. As there were no differences between OSA and comparison drivers, results below are for all 66 participants.

HR was a significant predictor of lane deviations (p = 0.01). A 10 % absolute decrease in HR yielded a 15.8% increase in the estimated mean number of lane deviations. RT was also a significant predictor of lane deviations (p = 0.03). A 0.1 second increase in reaction time yielded a 5.0% increase in the estimated mean number of lane deviations. However, the effect of RT was largely due to 2 outliers (1 OSA and 1 control). With these 2 subjects removed, RT was not a significant predictor (p = 0.21). SSS after the drive was a significant predictor of lane deviations (p < 0.01). The "Sleepy" subjects had an estimated 2.61 times the mean number of lane deviations as the "Awake" subjects and an estimated 1.62 times the mean number of lane deviations as the "Marginally sleepy" subjects. To determine the best predictors of lane

deviations between HR, RT, and SSS, a model was fit that included all three predictors. With all three predictors in the model, SSS remained significant ($p = 0.04$), while HR and RT did not, indicating that SSS after the drive was the strongest predictor of lane deviations.

Speed Errors

Few drivers had any low speed errors; the majority of speed errors were high-speed errors. Consequently, both low and high-speed errors were combined for subsequent analyses. Age was significantly related to speed errors ($p = 0.04$), so all results were adjusted for age. OSA status ($p = 0.23$) and gender ($p = 0.20$) were not significantly related to speed errors. As in the analysis of lane deviations, the results given below are for all 66 drivers.

HR was not a significant predictor of speed errors ($p = 0.44$) but RT was ($p < 0.01$). A 0.1 second increase in reaction time yielded a 19.4% decrease in the estimated mean number of speed errors. In other words, slower responses on the vigilance task predicted fewer high-speed errors. SSS after the drive was also a significant predictor of speed errors ($p < 0.01$). In order to determine the best predictor of speed errors, SSS and RT were modeled simultaneously. RT remained significant ($p < 0.01$) while SSS did not ($p = 0.28$). The results were very similar whether or not outliers were included in the analysis.

Standard Deviation of Lane Position

Gender was significantly related to SDLP ($p = 0.03$) and OSA status was marginally significantly related ($p = 0.07$). There was not a significant relationship between SDLP and age ($p = 0.93$). The following results are for all 66 drivers, adjusted for age and OSA status.

HR was a significant predictor of SDLP ($p = 0.02$). A 10% absolute decrease in HR yielded an estimated .022 increase in SDLP. RT was also a significant predictor of SDLP ($p = 0.04$). A 0.1 second increase in RT yielded a .006 increase in SDLP. SSS after the drive was a marginally significant predictor of SDLP ($p = 0.08$). The "Sleepy" subjects had an estimated SDLP that was .074 higher than the "Awake" subjects and .052 higher than the "Marginally Sleepy" subjects. To determine the best predictors of SDLP between HR, RT, and SSS, a model was fit that included all three predictors. With all three predictors in the model, HR ($p = 0.16$), RT ($p = 0.12$), and SSS ($p = 0.28$) did not remain significant.

DISCUSSION

We found that drivers with OSA have significantly impaired visual vigilance compared to drivers without neurological or sleep disorders. Vigilance is known to be impaired in many OSA subjects [6,7,8,9,27,28], but our finding that vigilance tends to be more impaired for peripheral visual targets, when studied in a naturalistic driving setting, is unique. These findings are important because safe driving is dependent upon the continuous coordination of several cognitive processes, of which sustained attention is critical [5]. Drivers with impaired attention are particularly at risk for making errors while following other vehicles, merging, negotiating intersections and curves, and while distracted. These driving behaviors are highly dependent upon executive control, which if impaired, does not allow for fast and accurate switching of the focus of attention [5]. Impaired vigilance, with resultant driving errors and increased crash risk, has been demonstrated in drivers with a variety of visual and neurological disorders [29, 30]. That vigilance is preferentially affected for peripheral visual targets may be construed as indicating that OSA drivers have a shrinking of their functional field of view, or a decrease in the efficiency with which they are able to extract information from a cluttered scene. This restriction in the field of view has been indexed in drivers with impaired attention by the useful field of view (UFOV) task, which has been shown to correlate with driving errors and motor vehicle crashes [10,11]. UFOV is operationalized as the visual area in which useful information

can be acquired in a brief time (typically less than 250 msec) without head or eye movements. However, safe driving requires continuous head and eye movements, and permitting these movements over an extended period of time, as in our study, provides an arguably more naturalistic assessment of visual attention. The area of visual attention when head and eye movements are allowed has been termed the "attended field of view" (AFOV), which has been studied in older drivers and those with visual field deficits [29,31].

Despite finding that OSA drivers have poorer vigilance than controls, we found little commensurate degradation in their simulated driving performance. This may have reflected the driving task design, which minimized challenges by incorporating a scenario with few distractions. Also, we used clinical assessment rather than PSG to exclude potential control subjects with sleep disorders. Absence of clinical features suggestive of OSA reasonably predicts those without the disorder [32,33], but it is possible that some of our control subjects were nevertheless affected as OSA is relatively common in the general population [34]. Perhaps most importantly, the majority of the OSA drivers we studied had mild to moderate disease; only 7 of the 25 OSA drivers had an AHI > 30 and the mean ESS score of the group was 12.5, just above the upper limit of normal. The mean MSL was normal, although more severely affected patients often underwent a "split night" protocol and did not have an MSLT. Patients with lower AHI's generally show less cognitive impairment than do those with more severe disease [7], which probably explains why we did not find worse simulated driving performance in our OSA drivers, as has been reported by others who have studied more severely affected patients [35,36]. The consecutive recruitment of OSA drivers in our study, however, suggests that our results are more representative of a typical clinic population. We did find that driving errors correlated with increased sleepiness and decreased performance on the vigilance task when OSA drivers and controls were combined, which implies that sleepiness and resultant vigilance impairment lead to more driving errors irrespective of whether or not drivers have a sleep disorder.

Clinicians treating OSA patients need to identify those with a high crash risk. Crash risk for most of these drivers is difficult to predict based upon ESS [37,38,39] and measures of disease severity, such as the AHI [37,38,39,40]. Similarly, performance in driving simulators has shown relatively weak association with objective measures of disease severity [35,41]. In this vein, we did not find correlations between vigilance performance and ESS, pre-drive SSS, AHI or MSL, although HR correlated with minimum oxygen saturation. The latter finding suggests that nocturnal hypoxemia may be a factor in causing vigilance deficits in OSA patients [42, 43], together with sleep fragmentation [7,9]. Although pre-drive SSS did not predict vigilance performance, SSS after the drive correlated with reduced vigilance in OSA drivers, and with poorer driving performance in the entire group. It may not be possible to identify high-risk drivers based upon subjective sleepiness before driving, but those who become sleepy while driving have vigilance and driving performance impairments that increase the likelihood of a crash. It has been shown that some OSA drivers are unable to perceive their impending sleepiness and subsequently increased crash risk [12]. Nevertheless, our drivers were aware that they were becoming sleepier, suggesting that they are potentially amenable to countermeasures, including education about the dangers of sleepy driving.

Do the vigilance impairments seen in our OSA drivers reflect changes particular to OSA or are they the result of non-specific effects of sleepiness? Roge et al [44] showed that sleep deprived healthy subjects have impaired visual attention during a simulated driving task similar to what we found in our OSA drivers. As we intentionally excluded potential control subjects with elevated ESS scores, our OSA drivers were consequently sleepier than the controls, although their mean ESS was only mildly elevated. Also, we found that increasing sleepiness over the drive was associated with greater impairment in vigilance for OSA drivers and more driving performance errors for the entire group. Hence, sleepiness, whether or not caused by

OSA, could explain the attentional deficits we observed. On the other hand, the fact that impaired vigilance correlated with hypoxemia, as indexed by the oxygen saturation minimum, and not AHI or MSL, suggests that sleepiness alone may not be the sole explanation for these findings. The extent to which cognitive impairments in OSA patients can be explained by either hypoxemia or disturbed sleep remains an unresolved question [7]. Perhaps more mildly affected patients, as we studied, have a different mechanism for their impaired performance than those with severe disease. Regardless of the mechanism by which these deficits occur, treatment of OSA with PAP appears to improve driving simulator performance [41] and crash risk [45].

The degraded vigilance found in our OSA drivers may suggest a means for identifying those with a higher crash risk. Driving simulators have been proposed as a method for determining driver fitness in patients with OSA [41], and the inclusion of vigilance tasks in the driving scenarios may increase the ability to detect impaired performance. Stand alone, off-road vigilance tests have also been proposed as a means for screening drivers [46] and on-board monitoring of vigilance may be useful for identifying drivers that are becoming potentially dangerous because of impending sleep [47]. In these ways, the ability to detect at-risk drivers and alert or warn those who are inattentive due to sleepiness may mitigate the suffering and financial impact of motor vehicle crashes in OSA and other sleepy drivers.

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REFERENCES

1. Knipling, RR.; Wang, J-S. Research Note from the Office of Crash Avoidance Research. Washington, DC: U.S. DOT/National Highway Traffic Safety Administration; 1994. Crashes and fatalities related to driver drowsiness/fatigue. p. 1-8.
2. McCartt AT, Ribner SA, Pack AI, Hammer MC. The scope and nature of the drowsy driving problem in New York State. Accid Anal Prev 1996;28:511–517. [PubMed: 8870778]
3. Sassani A, Findley L, Kryger M, Goldlust E, George C, Davidson T. Reducing motor-vehicle collisions, costs, and fatalities by treating obstructive sleep apnea syndrome. Sleep 2004;27:453–458. [PubMed: 15164898]
4. Tregear, SJ.; Tiller, M.; Greenberg, MI.; Rizzo, M.; Hegmann, KT.; Phillips, B., et al. Sleep Apnea and Motor Vehicle Crashes – A Systemic Review and Meta-Analysis. 2008 National Occupational Injury Research Symposium; Pittsburg, PA. October 13–21;
5. Rizzo, M. Safe and unsafe driving. In: Rizzo, M.; Eslinger, PJ., editors. Principles and Practice of Behavioral Neurology and Neuropsychology. Philadelphia: WB Saunders; 2004. p. 197-222.
6. Bedard M-C, Montplaisir J, Richer F, Rouleau I, Malo J. Obstructive sleep apnea syndrome : pathogenesis of neuropsychological deficits. J Clin Exp Neuropsychol 1991;13:950–964. [PubMed: 1779033]
7. Aloia MS, Arnedt JT, Davis JD, Riggs RL, Byrd D. Neuropsychological sequelae of obstructive sleep apnea-hypopnea syndrome: a critical review. J Int Neuropsychol Soc 2004;10:772–785. [PubMed: 15327723]
8. Mazza S, Pepin JL, Naegele B, Plante J, Deschaux C, Levy P. Most obstructive sleep apnoea patients exhibit vigilance and attention deficits on an extended battery. Eur Respir J 2005;25:75–80. [PubMed: 15640326]
9. Verstraeten E, Cluydts R, Pevernagie D, Hoffmann G. Executive function in sleep apnea: Controlling for attentional capacity in assessing executive attention. Sleep 2004;27:685–693. [PubMed: 15283003]

10. Clay OJ, Wadley VG, Edwards JD, Roth DL, Roenker DL. Cumulative meta-analysis of the relationship between useful field of view and driving performance in older adults: current and future implications. Optom Vis Sci 2005;82:724–731. [PubMed: 16127338]
11. Owsley C, Ball K, McGwin G, et al. Visual processing impairment and risk of motor vehicle crash among older adults. JAMA 1998;279:1083–1088. [PubMed: 9546567]
12. Engleman HM, Hirst WSJ, Douglas NJ. Under reporting of sleepiness and driving impairment in patients with sleep apnoea/hypopnoea syndrome. J Sleep Res 1997;6:272–275. [PubMed: 9493528]
13. American Academy of Sleep Medicine (AASM). Diagnostic and Coding Manual. Vol. 2nd edition. Westchester, Illinois: American Academy of Sleep Medicine; 2005. International Classification of Sleep Disorders; p. 51-55.
14. Walsh JK, Muehlbach MJ, Humm TM, Dickens QS, Sugerman JL. Effect of caffeine on physiological sleep tendency and ability to sustain wakefulness at night. Psychophysiology 1990;101:271–273.
15. Wetter DW, Young TB. The relation between cigarette smoking and sleep disturbance. Prev Med 1994;23:328–334. [PubMed: 8078854]
16. Aeschbach D, Postolache TT, Sher L, Matthews JR, Jackson MA, Wehr TA. Evidence from the waking electroencephalogram that short sleepers live under higher homeostatic sleep pressure than long sleepers. Neuroscience 2001;102(3):493–502. [PubMed: 11226688]
17. Anderson AJ, Johnson CA. Mechanisms isolated by frequency-doubling technology perimetry. Invest Ophthalmol Vis Sci 2002;43:398–401. [PubMed: 11818383]
18. Kushida CA, Littner MR, Morgenthaler T, Alessi CA, Bailey D, Coleman J, Friedman L, Hirshkowitz M, Kapen S, Kramer M, Lee-Chiong T, Loube DL, Owens J, Pancer JP, Wise M. Practice parameters for the Indications for Polysomnography and Related Procedures: An Update for 2005. Sleep 2005;28:499–521. [PubMed: 16171294]
19. Arand D, Bonnet M, Hurwitz T, Mitler M, Rosa R, Sangal RB. The clinical use of the MSLT and MWT. Sleep 2005;28:123–144. [PubMed: 15700728]
20. Hoddes E, Zarcone VP, Smythe H. Quantification of sleepiness: A new approach. Psychophysiology 1973;10:431–436. [PubMed: 4719486]
21. Johns MW. A new method for measuring daytime sleepiness: The Epworth Sleepiness Scale. Sleep 1991;14:540–545. [PubMed: 1798888]
22. Rizzo, M.; McGehee, D.; Jermeland, J. Design and installation of a driving simulator in a hospital environment. In: Brookhuis, K.; DeWaard, D.; Weikert, C., editors. Human System Interaction: Education, Research and Application in the 21st Century. Maastricht, The Netherlands: Shaker Publishing; 2000. p. 69-77.
23. Summala H, Nieminen T, Punto M. Maintaining lane position with peripheral vision during in-vehicle tasks. Human Factors 1996;38(3):442–451.
24. Rizzo M, Stierman L, Skaar N, Dawson JD, Anderson SW, Vecera SP. Effects of a controlled auditory-verbal distraction task on older driver vehicle control. Transportation Research Record: Journal of the Transportation Research Board 2004;1865:1–6.
25. Green, DM.; Swet, JA. Signal Detection in Theory and Psychophysics. Huntington, NY: R E Krieger Publishing; 1966. p. 34-40.
26. Carskadon MA, Dement WC. MSLT's during the constant routine. Sleep 1992;15:396–399. [PubMed: 1455121]
27. Bedard M-C, Montplaisir J, Malo J, Richer F, Rouleau I. Persistent neuropsychological deficits and vigilance impairment in sleep apnea syndrome after treatment with continuous positive airways pressure (CPAP). J Clin Exp Neuropsychol 1993;15:330–341. [PubMed: 8491855]
28. Feuerstein C, Naegele B, Pepin J, Levy P. Frontal lobe-related cognitive functions in patients with sleep apnea syndrome before and after treatment. Acta Neurol Belg 1997;140:529–530.
29. Coeckelbergh TRM, Brouwer WH, Cornelissen FW, van Woffelaar P, Kooijman AC. The effects of visual field defects on driving performance: A driving simulator study. Arch Ophthal 2002;120:1509–1516. [PubMed: 12427065]
30. Uc EY, Rizzo M, Anderson SW, Shi Q, Dawson JD. Unsafe rear-end collision avoidance in Alzheimer's disease. J Neurol Sci 2006;251:35–43. [PubMed: 17049360]

31. Coeckelbergh TRM, Cornelissen FW, Brouwer WH, Kooijman AC. Age-related changes in the functional visual field: further evidence for an inverse age x eccentricity effect. Journal of Gerontolgy: Psychological Science 2004;59B(1):11–18.
32. Deegan PC, McNicholas WT. Predictive value of clinical features for the obstructive sleep apnoea syndrome. Eur Respir J 1996;9:117–124. [PubMed: 8834344]
33. Young T, Shahar E, Nieto FJ, Redline S, Newman AB, Gottlieb DJ, Walseben JA, Finn L, Enright P, Samet JM. Predictors of sleep-disordered breathing in community-dwelling adults. Arch Intern Med 2002;162:893–900. [PubMed: 11966340]
34. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. N Eng J Med 1993;328:1230–1235.
35. George CFP, Boudreau AC, Smiley S. Simulated driving performance in patients with obstructive sleep apnea. Amer J Respir Crit Care Med 1996;154:175–181. [PubMed: 8680676]
36. Risser MR, Ware JC, Freeman FG. Driving simulation with EEG monitoring in normal and obstructive sleep apnea patients. Sleep 2000;23:393–398. [PubMed: 10811383]
37. Teran-Santos J, Jimenez-Gomez A, Cordero-Guevara J. The association between sleep apnea and the risk of traffic accidents. NEJM 1999;340:847–851. [PubMed: 10080847]
38. Horstmann S, Hess CW, Bassetti C, Gugger M, Mathis J. Sleepiness-related accidents in sleep apnea patients. Sleep 2000;23:383–389. [PubMed: 10811382]
39. Young T, Blustein J, Finn L, Palta M. Sleep-disordered breathing and motor vehicle accidents in a population-based sample of employed adults. Sleep 1997;20:608–613. [PubMed: 9351127]
40. George CFP, Smiley A. Sleep apnea and automobile crashes. Sleep 1999;22:790–795. [PubMed: 10505825]
41. Orth M, Duchna H-W, Leidag M, Widdig W, Rasche K, Bauer TT, Walther JW, de Zeeuw J, Malin J-P, Schultze-Werninghaus G, Kotterba S. Driving simulator and neuropsychological testing in OSAS before and under CPAP therapy. Eur Respir J 2005;26:989–903. [PubMed: 16319326]
42. Findley LJ, Barth JT, Powers DC, Wilhoit SC, Boyd DG, Suratt PM. Cognitive impairment in patients with obstructive sleep apnea and associated hypoxemia. Chest 1986;90:686–690. [PubMed: 3769569]
43. Roehrs T, Merrion M, Pedrosi B, Stepanski E, Zorick F, Roth T. Neuropsychological function in obstructive sleep apnea syndrome (OSAS) compared to chronic obstructive pulmonary disease (COPD). Sleep 1995;18:338–388.
44. Roge J, Pebayle T, El Hannachi S, Muzet A. Effect of sleep deprivation and driving duration on the useful visual field in younger and older subjects during simulator driving. Vision Res 2003;43(13): 1465–1467. [PubMed: 12767314]
45. George CF. Reduction in motor vehicle collisions following treatment of sleep apnoea with nasal CPAP. Thorax 2001;56(7):508–512. [PubMed: 11413347]
46. George CFP. Driving simulators in clinical practice. Sleep Med Rev 2003;7:311–320. [PubMed: 14505598]
47. Desai AM, Haque MA. Vigilance monitoring for operator safety: a simulation study on highway driving. J Safety Res 2006;37:139–147. [PubMed: 16626742]

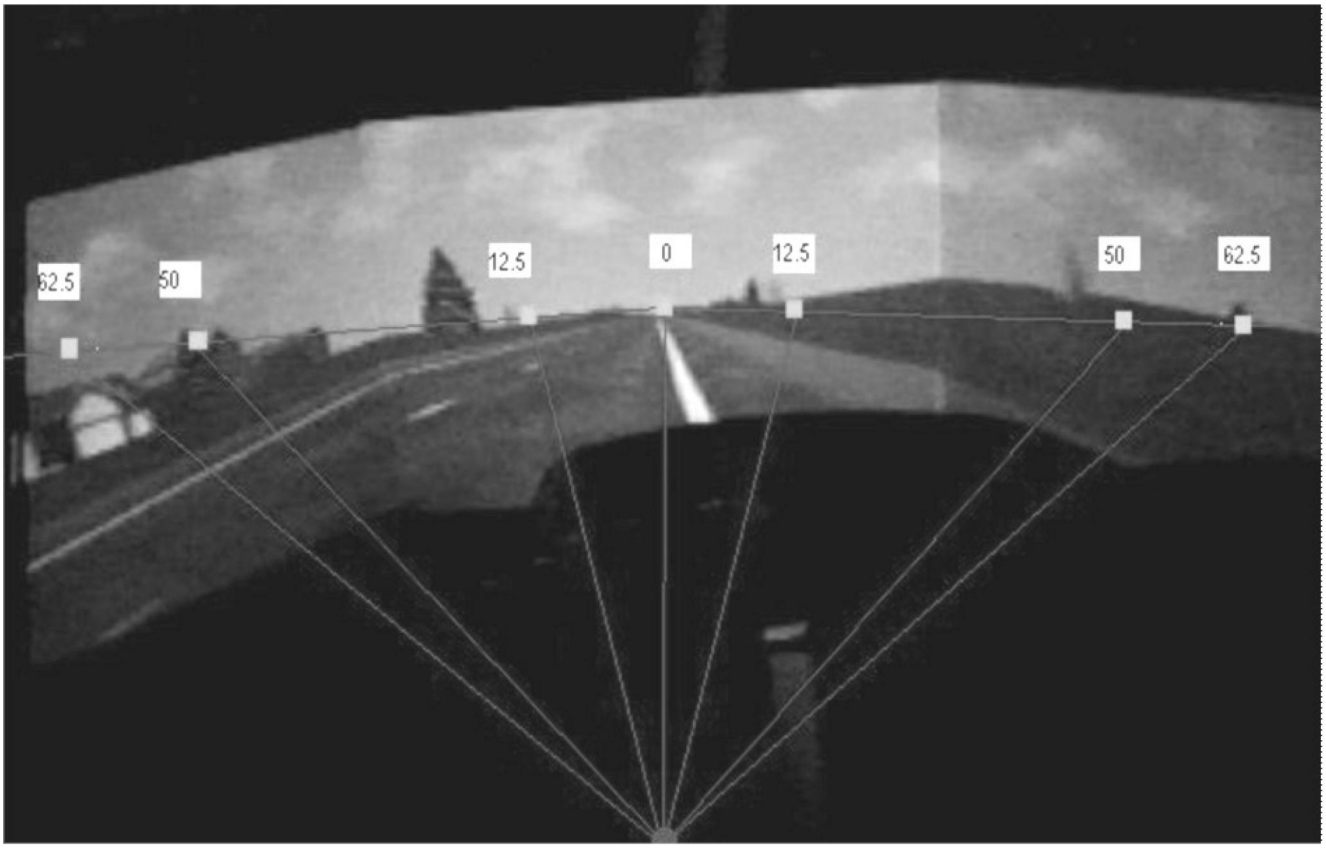

Figure 1.
Vigilance task during simulated driving in SIREN. Squares show the locations of potential targets (values are in degrees from center).

Table 1

Participant demographics

	OSA n = 25	**Controls n = 41**	**p**
Age (years)	44.96	40.56	0.18
SD	9.676	9.378	
Range	29 – 65	23 – 55	
Sex ratio (M/F)	18/7	21/20	0.10

Table 2

Subjective sleepiness measures by OSA status. ESS = Epworth Sleepiness Scale; SSS = Stanford Sleepiness Scale.

		Mean	Std	Median	Min	Max	p
ESS	Control	6.098	2.567	6	0	10	**<0.01**
	OSA	12.417	5.141	12.5	1	20	
SSS before	Control	2.22	0.852	2	1	5	0.51
	OSA	2.36	0.907	2	1	4	
SSS after	Control	3.561	1.205	3	1	6	**0.03**
	OSA	4.2	1.155	4	2	7	

Table 3

Results of the vigilance task by OSA status. Targets (#), HR = hit rate (%), FP = false positive responses (#), RT = reaction time (seconds);

		Mean	Std	Median	Min	Max	p
Targets	Control	58.927	4.962	59	45	70	0.74
	OSA	59.400	5.123	60	50	68	
HR	Control	0.867	0.088	0.895	0.645	0.968	**0.03**
	OSA	0.807	0.148	0.828	0.203	0.964	
FP	Control	0.390	0.542	0	0	2	0.79
	OSA	0.800	1.354	0	0	5	
D-prime	Control	3.4491	0.4235	3.5013	2.5140	4.2546	**0.03**
	OSA	3.0997	0.6803	3.1988	0.5739	3.8651	
RT	Control	1.371	0.374	1.335	0.764	2.956	0.25
	OSA	1.449	0.367	1.419	0.813	2.712	

Table 4

Results of the vigilance task as a function of target location. HR = hit rate (%), RT = reaction time (seconds).

		Mean	Std	Median	Min	Max	p
HR Peripheral	Control	0.814	0.126	0.864	0.45	0.971	**0.02**
	OSA	0.723	0.198	0.75	0.026	0.969	
HR Central	Control	0.955	0.059	0.968	0.75	1	0.21
	OSA	0.923	0.101	0.957	0.567	1	
RT Peripheral	Control	1.438	0.465	1.424	0.817	3.71	0.32
	OSA	1.539	0.57	1.485	0.811	4	
RT Central	Control	1.29	0.274	1.239	0.717	1.954	0.26
	OSA	1.407	0.389	1.35	0.686	2.636	

Table 5

Relationship between vigilance and subjective sleepiness measures. Spearman Correlation Coefficients with p-values in parentheses. HR = hit rate (%); RT = reaction time (seconds); ESS = Epworth Sleepiness Scale; SSS = Stanford Sleepiness Scale; OSA = obstructive sleep apnea syndrome.

	SSS before	SSS after	SSS before-after	ESS
All subjects				
HR	−0.177	−0.21221	−0.14185	−0.28154
	(0.1551)	(0.0872)	(0.2559)	**(0.02)**
RT	0.13506	0.11216	0.09259	0.01572
	(0.2796)	(0.3699)	(0.4597)	(0.90)
OSA				
HR	−0.1694	−0.13412	−.0.5243	−0.13581
	(0.4182)	(0.5227)	(0.8034)	(0.53)
RT	0.05998	0.04122	0.02539	0.18158
	(0.7758)	(0.8449)	(0.9041)	(0.40)
Controls				
HR	−0.14891	−0.15823	−0.09992	−0.16582
	(0.3528)	(0.3231)	(0.5342)	(0.30)
RT	0.16349	0.15185	0.13098	−0.05476
	(0.3071)	(0.3433)	(0.4143)	(0.73)