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DRIVING PERFORMANCE AND DRIVER STATE IN OBSTRUCTIVE SLEEP APNEA: WHAT CHANGES WITH POSITIVE AIRWAY PRESSURE?

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Summary

We evaluated naturalistic driving in 65 drivers with obstructive sleep apnea (OSA) before and after positive airway pressure (PAP) therapy and in 43 comparison drivers. Driving performance metrics included speed (mean, variability), and lateral, and longitudinal acceleration (g's). Driver state measures included sleepiness and attention to the driving task based on sampled trigger and baseline video clips. OSA drivers showed less variability in speed and lateral g's compared to control drivers before and after PAP treatment when vehicle speed was <45mph. There were no driving performance differences when vehicle speed exceeded 45 mph. OSA drivers remained less alert than comparison drivers before and after PAP. Average hours of nightly PAP-use predicted improved alertness and lower levels of sleepiness among OSA drivers. The findings suggest increased crash risk among OSA drivers may result from lower levels of attention to the driving task that result in performance lapses that may lead to crashes, rather than to clear and specific patterns of performance deficits in vehicle control.

OBJECTIVES

Meta-analytic studies indicate that OSA is associated with increased crash risk (Tregear et al., 2009). PAP, the standard treatment for OSA, appears to mitigate crash risk (Tregear et al., 2010). Crash statistics do not describe pervasive performance deficits of drivers with OSA that may be evident from electronic vehicle sensors such as average speed, variability in speed, lateral and longitudinal control compared to drivers without OSA. In contrast, controlled simulator studies provide more specific measures of performance deficits including poorer lateral and speed control as indicated by greater variability in vehicle sensor data, rate of off-road events, as well as delayed reaction time to peripheral events among OSA drivers. Consistent with crash statistics, these indices of simulator performance also improve following PAP treatment (Gosh et al., 2012; Hack et al., 2001; Orth et al., 2005; Risser et al., 2000; Turkington et al., 2004). However, controlled simulator can be limited by their generalizability to real-world settings. For example, simulator studies of driving safety in OSA or sleep deprivation in healthy controls are designed to induce sleepiness and lapses of attention even among healthy control drivers. They often employ monotonous drives that last an hour or more, with minimal visually stimulating cues in darkened rooms that differ from real-world driving environments.

Naturalistic driving studies offer advantages over simulator studies in two important ways. First, performance metrics are acquired in real-world settings that are visually more stimulating and variable than the typical monotonous drives in simulator environments. It is possible both pre and post-PAP performance differences obtained in simulator environments may have overestimated the effects of untreated OSA and PAP on safety in the real-world (Filtner et al., 2011; Thiffault & Bergeron, 2003). Second, drivers in their natural ecology choose many aspects of exposure to road risks including routes, frequency, timing, and duration of drives that differ from experimental protocols in simulators. It is likely drivers make choices to minimize drowsy driving related risks in their daily routines by choosing among a variety of countermeasures to combat drowsy driving (e.g. distractions, caffeine, napping) with varying degrees of effectiveness (Caldwell, 2001; MacLean et al., 2003). Better understanding of driving performance in untreated OSA and benefits of PAP in reducing risks in the real-world requires naturalistic studies.

We evaluated the effects of OSA on driving performance and driver state in a naturalistic driving study both prior to and after PAP-therapy spanning a total period of 3.5 months. Compared to previous work, this study: a) measured driving performance from electronic vehicle sensors and driver state including sleepiness and attention to the driving task from video, b) observed OSA drivers before and after PAP therapy, c) confirmed OSA diagnosis and non-OSA status with overnight polysomnography. We studied these drivers to address three broad questions:

1. Is untreated OSA associated with systematic differences in driver performance and driver state including sleepiness and attention to the driving task compared to drivers without OSA?
2. Is PAP-therapy associated with systematic changes in driver performance and driver state among OSA drivers? Does PAP-therapy improve OSA driver performance and driver state outcomes to control driver levels?
3. Does OSA driving performance and driver state outcomes in the post-PAP period depend on OSA disease severity and PAP-dose?

METHOD

Subjects

Eighty-five OSA and 50 control drivers were recruited into a naturalistic driving study lasting 3.5 months. Control drivers were matched with OSA drivers at the group level on age within 5 years, education within 2 years, and distribution of gender, and county of residence for rural vs. urban driving. Patients met ICSD-2 clinical criteria for OSA (Kushida et al., 2006) and had a Respiratory Distress Index (RDI) > 15, while controls had no sleep complaints and an RDI < 5 as confirmed by overnight polysomnography.

Subject drop-out—Twenty-seven of these participants were excluded from the current report for a variety of reasons including: a) participant's car was incompatible with the instrumented vehicle data acquisition system (IV-DAS), or had technical issues that caused

large drop out in either drive or video data (e.g. < 20 days of useable driving data); b) participants dropped out of study too early because of other commitments; c) OSA participants did not provide data on their PAP-use to assess treatment status. The remaining participants each had at least 20 days of driving data and had 25% of driving days video clips evaluated for driver state. The final sample had 65 OSA (23 female; M-age = 46.43; M-education = 15.56 years) and 43 control (16 female; M-age = 43.93, M-education = 16.33 years) drivers. There were no systematic differences between those included or excluded in terms of disease severity, age, educational level (min $p = .117$). Group/disease status did not increase the likelihood of exclusion from this report, $p = .132$ Fisher's exact.

Study Procedures

The protocol called for observing OSA drivers with the IV-DAS for 2-weeks before beginning PAP-therapy and for 3-months after. Control drivers were evaluated on the same schedule to assure comparable data acquisition. Several OSA drivers were not compliant with the study protocol either starting PAP earlier or later than expected.

Procedures Pertinent to Driving Data Collection—IV-DAS contained four devices: an internal camera cluster, a GPS, OBD-II, and accelerometers. Two cameras were located beneath the rear view mirror. One pointed forward toward the road (i.e. driver's eye view). The other aimed at the driver face and upper body and car interior. Electronic drive files and associated video clips were transmitted to a remote server daily. Video data collection was triggered based on accelerometer exceedances (at least $35g$'s) and a baseline data collection schedule (see Aksan et al., 2011). Each ignition on-off cycle could be associated with three types of clips: one-minute ignition clips, 20-second clips when the driver reached at least $35g$, and 20-second "baseline" clips every 15-minute into a drive. Video data from ignition and 15-minute baseline clips were categorized as baseline clips in this report. OBD speed, lateral, and longitudinal g 's sampled at 1Hz in each ignition on to off cycle were used in this report.

Measures

Driving performance measures—In order to contextualize the electronic sensor data into likely-highway versus non-highway driving, each drive file was first segmented into consecutive sections where the OBD speed was less than 45 or greater than/equal to 45 mph. In each of these sections within a drive, mean OBD speed (in mph), variability in OBD speed, the standard deviation in OBD speed and standard deviation in lateral and longitudinal acceleration (in g units) were computed prior to producing averages across trips within a day as the primary vehicle-based performance measures.

Driver state measures—Clips were evaluated in 20-second segments in three broad domains: safety, exposure, and driver state (Aksan et al., 2011) based on prior work (Klauer et al., 2006; Neale et al., 2005; Wierville & Elsworth, 1994). Table 1 provides examples of coded dimensions used in this report. Coders were trained on sample clips illustrating the range of behaviors in each of the dimensions listed in Table 1 until they showed the following minimum levels of inter-rater reliability: on categorical scales a Kappa of 0.61 (e.g.

driving related gaze movements in Table 1) and on continuously distributed scales intra-class correlation of (71 for absolute agreement (e.g. magnitude of yawning in Table 1). All coders were blind to PAP-use and some coders were also blind to group status. Percentage of 20-second segments in which the driver made driving related gaze movements or appeared sleepy was computed separately for baseline versus trigger clips within a day.

PAP-adherence—Nightly PAP-use data were downloaded during monthly visits in the post-PAP phase. Average minutes of use per night were used in this report.

Disease severity—In addition to RDI, Apnea Hypopnea Index (AHI) and SpO₂ Nadir were used as indices of disease severity from the overnight sleep study.

Data reduction—Each night's PAP-use was linked with the following day's driving data from both video (e.g. relative frequency of driving related gaze movements) and electronic vehicle sensors (e.g. lateral control). These daily measures were summarized with the average function across pre and post-PAP periods.

RESULTS

Only targeted between and within group differences were examined with weighted least squares to ensure variation in available data across participants did not unduly influence inferences. Participant specific weights were based on number of electronic drive file and coded video days that contributed to the respective summary measures shown in Table 2 (unadjusted raw values are shown in the table).

The p-values associated with the critical comparisons for study questions, pre-PAP differences between OSA and control drivers, and post-PAP changes among OSAs, and persistent group differences in the post-PAP period are shown in Table 2. Table 2 shows that 2 of 4 focused comparisons for electronic sensor measures were significant when vehicle speed was below 45mph (lower speed segments) but none of the corresponding measures were significant when vehicle speed was greater than 45mph (higher speed segments). OSA drivers showed lower variability in both speed and lateral g's in low speed segments prior to PAP-therapy. Average speed, lateral and longitudinal variability in low speed segments increased from pre- to post-PAP for OSAs. However, in the post-PAP period differences persisted between the groups in lateral control and speed variability (the latter at the trend level). OSA drivers also showed increased variability in lateral g's in high speed segments from pre to post-PAP. Regarding driver state measures, only driving related gaze movements showed significant differences between OSA and control drivers in both baseline and trigger clips prior to PAP, consistent with poorer attention to the driving task among OSA compared to control drivers. Other than trend-level increases in driving related gaze movements in baseline clips for OSAs, there was no significant change in these measures from pre to post-PAP. Finally, OSA drivers continued to show poorer attention to the driving task compared to control drivers in the post-PAP phase.

Table 3 shows the Pearson correlations of driving outcomes with average nightly PAP-use and disease severity indices. Out of 48 correlations, 3 were significant at the conventional level, at chance levels. When correlations were significant, PAP-use appeared to be correlated with driver state measures rather than electronic driving performance metrics. OSA drivers who used PAP for longer hours on average appeared to be less sleepy in both baseline and trigger segments, showed better attention to the driving task in the post-PAP phase. Disease severity showed weak/chance level associations with driving outcomes in the post-PAP period and the corresponding associations in the pre-PAP period were similarly at chance levels. Of 36 correlations in the pre-PAP period (not shown in Table 4), only one was significant at $p < .05$, SpO2 Nadir was associated with higher variability in lateral control in high speed segments, $r(60) = .30, p < .02$.

CONCLUSION

Driving performance measures acquired from electronic vehicle data in this naturalistic study indicated few differences between OSA drivers with a range of disease severity and matched control drivers prior to PAP therapy. Importantly, none of these differences clearly indicated that OSA drivers were less safe than control drivers. For example, OSA drivers showed less variability in speed and lateral g's compared to control drivers in low-speed segments prior to PAP. Furthermore, while changes from pre to post-PAP in OSA drivers made their average speed, average variability in lateral and longitudinal g's in low speed segments more similar to control drivers, group differences persisted in speed variability and lateral control in the post-PAP period. There were no within or between group differences in performance metrics from vehicle sensors during high speed segments. With regard to differences in driver state, OSA drivers did not appear to be more sleepy than control drivers either pre or post-PAP. However, their attention to the driving task was poorer both in baseline and trigger clips compared to control drivers and those differences persisted in the post-PAP period. Average nightly PAP-use was associated with better attention to the driving task and lower levels of sleepy appearance in the post-PAP period but not driving performance measures from vehicle sensors.

Overall these findings did not support large differences on vehicle-based performance metrics or driver state between OSA and control drivers either prior to PAP therapy or changes post-PAP in the real-world, at odds with inferences from previous studies, mostly based on simulator studies that induce sleep even among healthy, non-sleep deprived drivers. While it is possible differences in the environmental culture are partly responsible for lack of strong evidence in favor of lower safety among OSAs, it is unlikely those differences account for changes that were observed pre to post-PAP. OSA is an insidious onset disease that goes undiagnosed for years (Rahagi & Basner, 1999). Initial symptoms of the disease including increased fatigue and low energy likely lead to slow changes in driving habits and risk tolerances of OSA drivers. Our findings raise the possibility that to understand the time course of real-world improvements in OSA, drivers need to be followed up for longer periods in real-world contexts.

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Table 1

Coded dimensions in 20-second segments from each video clip in three domains of interest

Domain	Dimension	Examples
Driver State	Sleepiness	Slow eye lid closure, fixed gaze, rubbing eyes, yawning, low facial and bodily muscle tone, leaning/ holding neck/head (each scored from 1 (low) to 4 (high))
	Attention to Driving	Driving related gaze movements (e.g., checking mirrors, scanning the road ahead)

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Table 2

Means, standard deviations for OSA and controls pre & post-PAP on driving outcomes.

Outcome Measures	OSA		Control		Pre-PAP Group p-value	Post-PAP in OSA p-value
	Pre-PAP M(SD)	Post-PAP M(SD)	Pre-PAP M(SD)	Post-PAP M(SD)		
Speed < 45mph:						
M-speed	15.61 (5.01)	16.25 (4.98)	17.79 (3.19)	17.55 (4.33)	.103	.001
SD-speed	8.84 (2.11)	8.84 (2.21)	9.82 (1.24)	9.51 (2.01)	.042 ^a	.220
Lat-g SD	.0493 (.011)	.0508 (.011)	.0557 (0.009)	.0566 (.011)	.013 ^a	.004
Long-g SD	.0582 (.013)	.0594 (.012)	.0614 (.0094)	.0621 (.011)	.240	.016
Speed >=45 mph:						
M-speed	53.35 (4.22)	52.77 (3.81)	51.7 (3.38)	52.6 (2.97)	.120	.187
SD-speed	2.07 (0.91)	2.03 (0.84)	2.1 (0.76)	2.31 (.65)	.612	.580
Lat-g SD	.0224 (.006)	.0234 (.006)	.023 (.006)	.024 (.006)	.612	.027
Long-g SD	.0246 (.006)	.0253 (.006)	.0248 (0.005)	.0255 (.005)	.314	.061
Sleepiness						
Baseline clips	.15 (.17)	.13 (.09)	.10 (.09)	.09 (.08)	.341	.549
Trigger clips	.11 (.18)	.10 (.10)	.08 (.10)	.08 (.08)	.860	.426
Attention to Driving						
Baseline clips	.58 (.13)	.61 (.12)	.68 (.15)	.69 (.13)	<.001 ^a	.077
Trigger clips	.43 (.26)	.47 (.24)	.54 (.29)	.54 (.25)	.013 ^a	.140

M = Mean SD= Standard Deviation Lat-g= Lateral g, Long-g = Longitudinal g, N's range from 61 to 65 for OSAs and from 41 to 43 for controls.

^a designates persistent post-pap group differences.

Table 3

Pearson correlations of PAP-use and disease severity with driving outcome measures in the post-PAP period.

Outcome Measures	Mean PAP-use	AHI	RDI	SpO2Nadir
Speed < 45mph:				
M-speed	.18	.07	.14	.17
Var-speed	.05	.13	.17	.01
Lat-g SD	.09	.14	.26*	.13
Long-g SD	.05	.16	.22⁺	.09
Speed >=45 mph:				
M-speed	-.02	-.15	-.17	.08
Var-speed	-.06	-.06	-.01	.10
Lat-g SD	.05	-.10	-.02	.22⁺
Long-g SD	.05	.18	.17	-.03
Sleepiness				
Baseline segments	-.31*	.16	.22⁺	.16
Trigger segments	-.22⁺	.08	.12	.17
Attention to Driving				
Baseline segments	.30*	.01	.02	.10
Trigger segments	.15	.11	.14	.09

⁺ p < .10,

** p < .05

N's range from 63 to 65. SD= Standard Deviation Lat-g= Lateral g, Long-g = Longitudinal g

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