Risk and Severity of Motor Vehicle Crashes in Patients with Obstructive Sleep Apnea Hypopnea

Mulgrew AT\textsuperscript{1}, Nasvadi G\textsuperscript{2}, Butt A\textsuperscript{1}, Cheema R\textsuperscript{1}, Fox N\textsuperscript{1}, Fleetham JA\textsuperscript{1,3}, Ryan CT\textsuperscript{1,3}, Cooper P\textsuperscript{2}, Ayas NT\textsuperscript{1,3,4}

1. UBC Hospital Sleep Disorders Program, Respiratory Division, University of British Columbia, Vancouver, BC V5Z 1M9 Canada, 2. Insurance Corporation of British Columbia 151 West Esplanade North Vancouver, BC V7M 3H9 Canada, 3. Respiratory Division, Department of Medicine, University of British Columbia, Canada 4. Centre for Clinical Epidemiology and Evaluation, Vancouver Coastal Health Research Institute, Canada

Corresponding Author: Dr. Najib Ayas

Diamond Health Centre,

2775 Laurel St

Vancouver, BC, V6R 1M9

najib.ayas@vch.ca
Abstract

Obstructive sleep apnea hypopnea (OSAH) appears to be associated with an increased risk of motor vehicle crashes (MVC). However, its impact on crash patterns and particularly the severity of crashes has not been well described. We sought to determine whether OSAH severity influenced crash severity in patients referred for investigation of suspected sleep-disordered breathing. Objective crash data (including the nature of crashes) for patients with suspected OSAH for the 3 years prior to polysomnography were obtained from provincial insurance records and compared to an age and sex matched control group. Data were obtained for 783 patients with suspected OSAH and 783 matched controls. The patient group was 71% male, with a mean age of 50 years, a mean apnea-hypopnea index (AHI) of 22 events/hour and a mean Epworth sleepiness scale score of 10. There were 375 crashes, of which 252 were in patients and 123 in controls in the 3-year period. When compared to controls, patients with mild, moderate, and severe OSAH had an increased rate of MVC with relative risks (95% CI) of 2.6 (1.7, 3.9), 1.9 (1.2, 2.8), and 2.0 (1.4, 3.0) respectively, whereas patients with suspected OSAH and normal polysomnography (AHI 0-5) did not with a relative risk (95% CI) of 1.5 (0.9, 2.5 p =0.21). When we examined the impact of OSAH on MVC associated with personal injury, patients with mild, moderate, and severe OSAH had a substantially increased rate of MVC compared to controls with relative risks of 4.8 (1.8, 12.4), 3.0 (1.3, 7.0), and 4.3 (1.8, 8.9) respectively, whereas patients without OSAH had similar crash rates to control with a relative risk of 0.6 (0.2, 2.5). Very severe MVCs (head-on collisions or those involving pedestrians or cyclists) were rare but 80% of these occurred in OSAH patients (p=0.06).
Patients with OSAH have increased rates of MVC with disproportionately increased rates of MVC associated with personal injury.
Introduction

In a number of studies, drivers with obstructive sleep apnea hypopnea (OSAH) have a higher rate of motor vehicle crashes (MVC) than drivers without OSAH\cite{1-4}, however it remains uncertain whether all patients with OSAH share this risk. It is not surprising that patients complaining of excessive daytime sleepiness would have increased MVCs, indeed drowsiness is a contributing factor in 10% of all motor vehicle crashes (MVCs) and 12% of near-miss MVC\cite{5}. However in previous studies, subjective sleepiness frequently showed no correlation with MVC rates\cite{4,6-8}. Furthermore, whereas many studies show a dose-response relationship between OSAH severity and MVC risk\cite{3,9-11} an equal number do not\cite{4,7,8,12}. It is difficult to obtain driving records on large numbers of patients and this has limited some of the previous studies. Many studies have used self-reported as opposed to validated MVC (e.g., from insurance or police records)\cite{6,9,10,12-14}. Secondly, some studies had relatively few numbers of patients with OSAH, and few studies included many drivers with milder degrees of OSAH. Thirdly, some studies did not have information about important potential confounders (such as kilometers driven per week, body mass index, sedative medications, daily sleep duration, and nocturnal shift work). Other studies have lacked a control group\cite{8,15-18}. Perhaps most importantly there was often little information provided concerning the severity of the MVCs in this population, with no distinction made between MVCs resulting in minor property damage and those causing injury or death. This may be important as MVC’s related to sleepiness may be of
a more severe nature, as they are associated with lack of vigilance or even falling asleep at the wheel\textsuperscript{19-21}.

We hypothesized that not only do patients with OSAH have an increased rate of MVC, but also that MVCs in OSAH patients are more likely to be of a severe nature. To better understand the relationship between OSAH and MVC, we have calculated rates of validated MVC categorized by severity, in the three years prior to polysomnography (PSG) in a large population of patients (matched to an age and gender matched control group) with suspected sleep-disordered breathing. We also collected extensive information on potential confounders to allow multivariable regression analysis to determine the independent effects of OSAH on rates and severity of MVC.

Materials and Methods

Study Sample: Consecutive male and female adult subjects (>18 years) referred for overnight PSG for suspected sleep disordered breathing were invited to participate. All patients were referred to the Vancouver Acute Hospital Respiratory Sleep Disorders program, the tertiary referral centre for sleep disorders in British Columbia, Canada. Patients with predominately non-respiratory sleep complaints (e.g. narcolepsy, insomnia) are routinely referred to another group of physicians at the clinic and are therefore not strongly represented in our patient population. Patients were seen by one of five academic respirologists with special interest in sleep-disordered breathing prior to PSG. Patients were excluded if they had symptoms of another sleep disorder known to cause daytime
sleepiness (e.g. periodic limb movement disorder), had another serious medical condition or overt psychiatric disease.

Patients were also excluded if they were already established on treatment for OSAH at time of referral or if they did not have a valid British Columbia driver’s license for the three years prior to PSG. The study was approved by the university ethics board and all patients provided written, informed consent.

Techniques

Self-administered Questionnaires:

Patients completed a variety of surveys on the night of their PSG. These surveys included questions about work history, medical history, psychiatric history, sleep habits, health habits (including smoking, caffeine, alcohol use), sleep symptoms, daytime sleepiness (Epworth Sleepiness Scale, (ESS)\textsuperscript{22}), and medications. For alcohol use, patients were asked to report their average use of: beer (1 glass, bottle, or can), wine (4 oz glass), or liquor (1 drink or shot) over the past year (11 gradations of response ranging from never or less than once per month, to more than 20 per day). Weekly alcohol use was calculated by summing the frequency of beer, wine, and liquor use. Weekly caffeine use was estimated by asking patients to report how many cups of coffee with caffeine they used over the previous year, using similar gradations.

Polysomnography (PSG)
Overnight PSG was performed using conventional instrumentation and analysis according to the recommendations on syndrome definition and measurement techniques published by the American Academy of Sleep Medicine\textsuperscript{23}. Sleep and its various stages were documented by standard electroencephalographic, electro-oculographic and electromyographic criteria. Apneas and hypopneas were recorded by oro-nasal flow cannulae attached to a pneumotachograph. Chest wall and abdominal movement were recorded using inductive plethysmography to document respiratory effort. Oxygen saturation was measured by pulse oximetry using a finger probe. Periodic limb movements were recorded from two surface electromyographic electrodes positioned 2-4 cm apart over the belly of the tibialis anterior muscle of each lower limb. Obstructive apneas were defined as absence of airflow for greater than 10 seconds; obstructive hypopneas as a 50\% decrease in airflow, or a clear but lesser decrease in airflow if associated with either a >3\% desaturation or an arousal in the context of ongoing respiratory effort. The complete record was scored manually for sleep stage, arousals, apneas and hypopneas.

**Motor Vehicle Crash Information**

All motorists in the province of British Columbia, Canada are insured by a single crown corporation (the Insurance Corporation of British Columbia (ICBC)). Objective crash data for subjects in the 3 years prior to PSG was obtained from ICBC records including details regarding crash severity type. All MVCs are categorised in the insurance database into one of 3 severity groups – 1. Crashes involving minor property damage (<$1000) 2. Crashes involving major property damage (>$$1000) including
damage to both the driver’s car and third party damage. Crashes that involved personal injury. More detailed descriptions of crash location and nature were also available e.g. serious crashes involving head-on collisions, cyclists or pedestrians.

All patients were matched with an individual control matched for age, gender, type of license, and driving experience. Patients and controls were also matched by postal region since people residing in the same geographical area are more likely to experience similar travel patterns associated with urban/rural residency and socioeconomic status.

**Data Analysis**

All data are reported as mean ± standard deviation unless otherwise noted. A p value of 0.05 was considered statistically significant. Patients were divided into four groups of OSAH severity based upon the apnea hypopnea index (AHI)\(^2\). These included patients with normal PSG (AHI≤5 events per hour), mild OSAH (AHI>5 and ≤15), moderate OSAH (AHI>15 and <30), and severe OSAH (AHI>30). Patients were also divided into four quartiles of subjective sleepiness severity depending on their ESS.

Crash rates were compared for patient severity groups according to AHI or ESS. MVCs occur in a Poisson distribution; therefore, Poisson regression, in a forward-step model was used to assess the effect of potential confounders. Differences between rates of MVC in patients and controls were assessed for significance using McNemar’s test for discordant pairs.

**Control Group**
The control group was provided by ICBC from their database. Controls were individually matched to patients by age, sex and driver license type and residence. The number of claims in the matched control group was checked against mean figures from the database to ensure the group was representative. No medical data was available for controls and their OSAH status and levels of subjective sleepiness were unknown.

**Results**

**Patient Characteristic**

783 patients were identified and were matched with individual controls drawn at random from the ICBC database. The majority of patients were male (71%) with a mean age of 49.9±11.6 years. The mean AHI was 22.6±21.9 events per hour, mean BMI was 31.8±10.3 kg/m², mean ESS was 10.1±5.3 and average kilometers driven per week were 234±208. Patient demographics are shown in table 1.

<table>
<thead>
<tr>
<th>Total</th>
<th>AHI 0-5</th>
<th>AHI 6-15</th>
<th>AHI 16-30</th>
<th>AHI &gt;30</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n=783)</td>
<td>(n=140)</td>
<td>(n=235)</td>
<td>(n=206)</td>
<td>(n=202)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>49.9±11.6</td>
<td>46.9±11.2</td>
<td>49.7±11.0</td>
<td>49.6±11.4</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>31.8±10.3</td>
<td>31.2±19.9</td>
<td>31.1±6.4</td>
<td>31.4±6.4</td>
</tr>
<tr>
<td>Gender (% male)</td>
<td>71.1</td>
<td>59.7</td>
<td>66.5</td>
<td>72.3</td>
</tr>
</tbody>
</table>
MVC Characteristics

MVC data were collected for a period of 3 years extending back from the date of PSG. In the 3-year period, there were 375 crashes, 252 of which were in patients and 123 in controls. In the patient group there were 94 crashes causing minor property damage, 83 crashes causing major property damage and 74 crashes causing injury compared to 48, 52 and 23 in the control group. Crash breakdowns are shown in table 2 and figure 1.

Relationship between MVC and OSAH severity

The study group was stratified into groups according to sleep apnea severity. The rate of MVC over the 3 years prior to PSG evaluation was calculated for each severity group. When compared to controls, patients with OSAH had a significantly increased rate of MVC with a relative risk ranging between 1.9 and 2.6 (table 2) whereas patients who did not have OSAH (AHI 0-5) were not at increased risk compared to controls, with a relative risk (95% CI) of 1.5 (0.9, 2.5 p =0.21). A within patient analysis showed overall

<table>
<thead>
<tr>
<th></th>
<th>22.6±21.9</th>
<th>2.4±1.5</th>
<th>9.8±2.9</th>
<th>21.4±4.2</th>
<th>53.6±20.9</th>
</tr>
</thead>
<tbody>
<tr>
<td>AHI</td>
<td>10.1±5.2</td>
<td>10.2±5.1</td>
<td>9.9±5.3</td>
<td>9.3±5.1</td>
<td>10.9±5.2</td>
</tr>
<tr>
<td>ESS</td>
<td>234.3±208.3</td>
<td>195.3±201.9</td>
<td>237.7±211.2</td>
<td>223.3±201.7</td>
<td>266.6±206.9</td>
</tr>
<tr>
<td>Km/week driven</td>
<td>21.4±4.2</td>
<td>53.6±20.9</td>
<td>21.4±4.2</td>
<td>53.6±20.9</td>
<td>21.4±4.2</td>
</tr>
</tbody>
</table>
crash rate to increase as OSAH severity increased (figure 1) from 0.24±0.5 crashes/patient/3yrs in patients with an AHI of 0-5 to 0.35±0.7 crashes/patient/3yrs in patients with an AHI>30 however this difference was not significant.

Also shown in table 2 are rates of MVC subdivided into 3 severity categories. MVCs causing minor property damage were increased in OSAH patients compared to controls whereas MVC involving major property damage were not. Within patients there is no evidence of a dose-response relationship between OSAH severity and MVC causing property damage. The presence of OSAH caused a disproportionate increase in rates of more severe MVC. When compared to controls (figure 2), patients with OSAH had between a 3.0 and 4.8-fold increased rate of MVC causing injury, whereas those without OSAH (AHI 0-5) had similar MVC rates to control. MVC involving personal injury represented 25.6% of the total. Within the patient group there appeared to be a dose-response relationship between OSAH severity and rate of MVC with injury, with the proportion of this type of MVC rising as OSAH severity increased (figure 3). In patients with an AHI of 0-5 MVC involving personal injury accounted for 9% of crashes compared to 37% in patients with an AHI >30. Compared to patients with an AHI of 0-5 the relative risk of MVC causing injury in patients with severe OSAH (AHI >30) was 6.1 (1.6,18.1). Patients with mild OSAH (AHI 5-15) were also at increased risk of MVC involving personal injury compared to those with an AHI of 0-5 with a 4.9-fold (1.5,16.1) increase in this type of MVC.

MVC felt to be of an extremely serious nature (head on collisions, or those involving pedestrians or cyclists) were rare, with only 10 such MVCs in the 3-year period. 80% of these MVC were in patients with OSAH. None of these MVC occurred in the patients
without OSAH (AHI 0-5), 3 occurred in patients with mild OSAH (3.6% of total accidents in this group), 2 in patients with moderate OSAH (3.1% of total accidents) and 3 in patients with severe OSAH (4.2% of total accidents). This was compared with 2 crashes of this nature in the control group. The mean rate of MVC of this type in patients was 0.01±0.1/3yrs compared with 0.002±0.05/3yrs in controls (p=0.057).

**Table 2**

<table>
<thead>
<tr>
<th></th>
<th>AHI 0-5 (n=140) Patient</th>
<th>AHI 6-15 (n=235) Patient</th>
<th>AHI 16-30 (n=206) Patient</th>
<th>AHI &gt;30 (n=202) Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>Control</td>
<td>Control</td>
<td>Control</td>
</tr>
<tr>
<td>Total MVC</td>
<td>33</td>
<td>22</td>
<td>83</td>
<td>32</td>
</tr>
<tr>
<td><strong>Relative Risk</strong></td>
<td>1.5</td>
<td>2.6**</td>
<td>1.9**</td>
<td>2.0**</td>
</tr>
<tr>
<td><strong>(95%CI)</strong></td>
<td>(0.9, 2.5)</td>
<td>(1.7, 3.9)</td>
<td>(1.2,2.8)</td>
<td>(1.4, 3.0)</td>
</tr>
<tr>
<td>MVC with minor property damage</td>
<td>17</td>
<td>10</td>
<td>33</td>
<td>15</td>
</tr>
<tr>
<td><strong>Relative Risk</strong></td>
<td>1.7</td>
<td>2.2*</td>
<td>1.7</td>
<td>2.2*</td>
</tr>
<tr>
<td><strong>(95%CI)</strong></td>
<td>(0.8, 3.4)</td>
<td>(1.2, 4.0)</td>
<td>(0.8, 3.3)</td>
<td>(1.1, 4.4)</td>
</tr>
<tr>
<td>MVC with major property damage</td>
<td>13</td>
<td>7</td>
<td>27</td>
<td>12</td>
</tr>
<tr>
<td><strong>Relative Risk</strong></td>
<td>1.85</td>
<td>2.3</td>
<td>1.5</td>
<td>1.2</td>
</tr>
<tr>
<td><strong>(95%CI)</strong></td>
<td>(0.7, 4.5)</td>
<td>(1.0, 4.4)</td>
<td>(0.8, 2.8)</td>
<td>(0.61, 2.2)</td>
</tr>
<tr>
<td>MVC with personal injury</td>
<td>3</td>
<td>5</td>
<td>24</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>21</td>
<td>7</td>
<td>26</td>
<td>6</td>
</tr>
</tbody>
</table>
Crashes are categorized into 1 of 3 severity groups. Relative risks are shown between patients and controls. P values were calculated using McNemar’s test.

* p < .05

** p < 0.005

† p < 0.001

### The Relationship Between Motor Vehicle Crashes and Subjective Sleepiness

MVC rates were calculated according to degree of subjective sleepiness (quartiles) as measured by the Epworth Sleepiness Scale. There was no significant difference in MVC rates among the quartiles (figure 4). When subsets of MVC (minor property damage, major property damage or personal injury) were examined, ESS had no effect on crash rate. In addition, ESS did not influence the rate of extremely serious crashes.

### Multivariable Modeling:

Multivariable Poisson regression was performed to control for the effect of a variety of potential confounders. The following variables were included in the model (table 3): AHI group, ESS, BMI, gender, age, kilometers driven, and use of alcohol, sedatives or caffeine. After controlling for confounders, the presence of OSAH increased the rate ratio (95% CI) for any motor vehicle crash to 1.22 (0.92, 2.01) but this was not
statistically significant with a p value of 0.12. Of note, BMI and kilometers driven were both significant in the model with p values of 0.008 and 0.003 respectively. However, the presence of OSAH increased the rate ratio for crashes causing personal injury to 3.67 (1.30, 10.3), p value= 0.014. Kilometers driven was also nearly significant in the model with a p value of 0.053. Of note, gender was not significantly associated with increased MVC rates in patients.

In control subjects a simplified regression model including only age and gender was used. Male gender was associated with an increased rate of any MVC, p<0.001 whereas age was not a significant predictor in the model for all crashes. Increased age was associated with a significantly reduced rate of more serious crashes.

Table 3

<table>
<thead>
<tr>
<th>Patients</th>
<th>Unselected Motor Vehicle Crashes</th>
<th>Motor Vehicle Crashes with Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rate ratio (95% CI)</td>
<td>p value</td>
</tr>
<tr>
<td>Presence of OSAH</td>
<td>1.22 (0.92,2.01)</td>
<td>0.12</td>
</tr>
<tr>
<td>ESS</td>
<td>1.01 (0.99, 1.04)</td>
<td>0.35</td>
</tr>
<tr>
<td>Male Gender</td>
<td>0.91 (0.66,1.25)</td>
<td>0.55</td>
</tr>
<tr>
<td>Age</td>
<td>1.00 (0.99, 1.01)</td>
<td>0.69</td>
</tr>
<tr>
<td>BMI</td>
<td>1.01 (1.00,1.02)</td>
<td>0.008</td>
</tr>
<tr>
<td>Per 100 km driven per week</td>
<td>1.09 (1.03, 1.16)</td>
<td>0.003</td>
</tr>
<tr>
<td>Alcohol use*</td>
<td>1.01 (1.00,1.03)</td>
<td>0.12</td>
</tr>
<tr>
<td>Sedative Use</td>
<td>0.79 (0.49,1.28)</td>
<td>0.34</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>--------------------------</td>
<td>-----</td>
<td>-----</td>
</tr>
<tr>
<td>Caffeine use**</td>
<td>0.99 (0.98,1.00)</td>
<td>0.15</td>
</tr>
<tr>
<td>Controls</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male Gender</td>
<td>1.55 (1.00,2.41)</td>
<td>0.049</td>
</tr>
<tr>
<td>Age</td>
<td>0.99 (0.98,1.01)</td>
<td>0.38</td>
</tr>
</tbody>
</table>

* for every one increase in alcohol use per week (i.e., bottle/can/glass of beer, 4 oz wine, or drink/shot of liquor)

** for every one cup increase in coffee use per week
Discussion

We found that compared to controls, patients with OSAH had an increased rate of MVC. Importantly, the relative risk was much greater for crashes associated with personal injury than for those involving property damage only. Our large sample allowed us to perform regression analysis for a number of confounding variables. After multiple regression the presence of OSAH did not significantly increase the odds of unselected MVCS but was associated with a more than 3-fold increase in the odds of MVC associated with injury.

To our knowledge, the greater impact of OSAH on crashes involving injury has not previously been demonstrated. However, studies looking specifically at severe MVCs have been performed and support the concept that decreased vigilance and/or falling asleep at the wheel are associated with a more severe spectrum of crashes, perhaps because sleepiness-related crashes may be related to failure to break/reduce speed, driving off the road, or higher speeds.24 25 In our study, any degree of OSAH was associated with an increase in MVC rate. This contrasts with the findings of George and associates who noted an increased MVC rate only in patients with an AHI >40 but is consistent with data from Young and colleagues which showed an adjusted odds ratio of 4.2 for MVC’s in patients with an AHI of 5-158. We did not find significant differences in MVC rates when patients with mild OSAH were compared to patients with severe OSAH. Interestingly, OSAH had little effect on MVCs involving major property damage. Although insurance database commonly include severity cut-offs for property damage our impression was that inclusion in this category was more influenced by the cost of the
vehicle rather than by the actual crash impact. Consequently it is not unexpected that OSAH did not influence this category of MVC.

We adjusted for several other confounding variables, including age, kilometers driven, gender, and substance use. MVC rates were related to kilometers driven and BMI. Although we identified a significant association between alcohol use and injury associated crashes, we found no relationship between MVCs and sedatives or stimulants. There is no clear reason why BMI should have increased the rate of unselected MVC in our regression, but there are potential explanations. First, obese patients may have difficulty moving easily in the drivers’ seat, leading to reduced ability to see traffic approaching from behind or the side. Second, adipose tissue increases levels of inflammatory mediators (such as tumor necrosis factor) which may result in reduced vigilance independently of sleep apnea severity. 26 Third, obesity may be a marker of subtle sleep-disordered breathing events that may not be captured by the standard definitions of AHI as assessed by polysomnography. Given that the severity of sleep apnea (as assessed by AHI) does not correlate well with sleepiness and did not correlate well with crash risk in our study, we may need to search for other metrics of sleep-disordered breathing that could be better indicators of crash risk. 27

Another interesting finding from our regression analysis was that gender did not significantly influence rate of MVCs among patients with OSAH. The actual MVC rate in female patients with OSAH was 0.27 crashes/3 yrs compared to 0.36 crashes/3 yrs in male patients. In contrast our regression showed gender to be a significant predictor of MVCS in control patients (0.11 crashes/3 years in females and 0.18 crashes /3 yrs in
males). OSAH appears to have an ‘equalizing’ effect between the genders in terms of driving risk.

We did not find a significant relationship between subjective sleepiness and rates of MVC. This is somewhat surprising given the findings in a recent large naturalistic driving study\(^5\) which indicated that drowsiness was a contributing factor in 10\% of all crashes and 12\% of near crashes. However, our study is consistent with other studies involving patients with OSAH. In a recent systematic review of papers examining rates of MVC in patients with OSAH, ESS was not predictive of MVC in 8 of 15 studies\(^28\). Several factors may contribute to this counterintuitive finding. First, ESS is not a good predictor of OSAH severity and correlates poorly with multiple sleep latency testing\(^27\). Second, patients who score highly on the ESS are by definition aware of their propensity to fall asleep and thus have potential to adapt their behavior accordingly. Patients who are less aware of impairment may be at much higher risk. This possibility was underlined in a study by Connor and coworkers, where acute sleepiness measured on the Stanford Sleepiness Scale\(^29\) was associated with increased risk of death or injury in MVCs, but chronic sleepiness as determined by the ESS was not, underlining the possibility of adaptive strategies by drivers who are aware of their own levels of sleepiness\(^24\). We suspect that many patients with OSAH may not be effective in assessing their impairment and that this confers increased risk. Third, patients may intentionally misrepresent their levels of sleepiness because of concerns related to exclusion form driving. Fourth, our study is a retrospective analysis of a referral population as opposed to a population based study. As such, it may be possible that patients may be referred because they perceive a problem with their driving (whether or not they complain of sleepiness per se) thus
minimizing the relationship between subjective sleepiness and crash risk. Future prospective studies may clarify this issue. Fifth, for many of the variables (e.g., caffeine use, kilometers driven) we have relied on self-reports of patients. For some of these, self-report may be inaccurate (e.g., sedatives, coffee) which might be a potential explanation of the lack of significant association between these variables and crashes.

Driving is a complex task, and driving performance is subject to a wider range of influences. The relationship between OSAH severity, sleepiness and the ability to perform intricate tasks shows considerable variability. Simulated driving performance is certainly impaired by OSAH and improved by CPAP, however whether simulators can be used to gauge MVC risk is unclear. In addition, our data indicates that the increased risk of MVC occurs at all levels of OSAH severity confirming the findings of Barbe and colleagues that conventional makers of OSAH severity do not discriminate those patients at higher risk of MVC. In any event, caution should be exercised when assessing patients driving risk and all patients with OSAH should be advised of potential risk.

Our study had a number of strengths compared to previous studies in this area. First, this is the largest study of this kind to date, with a large range of OSAH severity. Second, we used a validated measure of MVC rates (provincial insurance records) rather than self-reports. Third, we collected information about a variety of potential confounders, including use of alcohol, sedatives and stimulants. Finally, our study is the first to examine the relationship between MVC severity and OSAH, and can confirm directly the previous figures from George’s data. However, our study also had limitations. First, our study was retrospective in design in that PSG information was collected after the
MVC. As such, we cannot exclude the possibility that the occurrence of MVC was a reason for referral to the sleep laboratory. This could have artificially increased the difference between rates of controls and patients, and be a potential explanation of the slightly increased rate of MVC in the 0-5 AHI group compared to controls (odds ratio=1.5) that was nearly significant. However, this should not have affected the relative risk between patients with and without OSAH. Second, our study was based on patients referred to a sleep disorder clinic with suspected sleep disordered breathing; our results may thus not be applicable outside this type of patient population. Patients may have been partially referred on the basis of sleepiness, and this could help explain the increased rate of crashes in our patients who did not have OSAH (i.e. they had causes of sleepiness other than OSAH). This could also explain the relatively poor correlation between AHI and ESS in our study (r=0.13, p<0.0001).

In summary, patients with OSAH have an increased rate of MVC compared to patients without OSAH, and age/gender matched controls. Patients with OSAH are particularly prone to MVC’s associated with personal injury and this increased risk occurs even in patients with mild OSAH. Subjective daytime sleepiness does not appear to be particularly helpful in terms of risk prediction for MVC which indicates a need for alternative strategies to assess driving performance. Treatment with continuous positive airway pressure has been shown to normalize crash rates and improve vigilance in OSAH patients. Motor vehicle crashes associated with OSAH are potentially preventable. Increasing public awareness of the symptoms and risks of OSAH along with improving access to diagnosis and treatment is likely to confer major cost savings to society in addition to benefiting individual patients.
Figure Legends

Figure 1
Mean Crash rates are shown over 3 years for patients separated by OSAH severity and their respective controls. Error bars represent standard error of the mean.

Figure 2
Mean rates of crashes causing injury are shown over 3 years for patients grouped by OSAH severity and their respective controls. Error bars represent standard error of the mean.

Figure 3
Proportion of crashes with injury and crashes causing property damage only are shown for the patient group.

Figure 4
Mean crash rates are shown over 3 years for patients separated into quartiles of subjective sleepiness bases on the Epworth Sleepiness Scale. Error bars represent standard error of the mean.
References


Crash Breakdown in OSA Severity Groups

% of Total

AHI 0-5  AHI 6-15  AHI 16-30  AHI >30

Injury  Property Damage
Risk and Severity of Motor Vehicle Crashes in Patients with Obstructive Sleep Apnea Hypopnea

alan mulgrew, Glenyth Nasvadi, Arsalan Butt, Rupi Cheema, Nurit Fox, John A Fleetham, Frank Ryan, Peter cooper and Najib Ayas

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