Autobiographical memory impairment in obstructive sleep apnea patients with and without depressive symptoms

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SUMMARY

Obstructive sleep apnea is associated with memory impairments, and higher rates of depressive symptoms and major depressive disorder compared with community estimates. Autobiographical memory overgenerality, a behaviour characterized by difficulty recalling specific memories from one's own life, is recognized as a marker of depression. Previous studies have demonstrated the predictive quality of specific autobiographical memory recall on the course of depression in patients with obstructive sleep apnea. However, it remains unclear whether impaired autobiographical memory is simply a feature of depression, or whether it is also impaired in patients with obstructive sleep apnea without depression. This study aimed to investigate whether autobiographical memory impairments can be observed in patients with obstructive sleep apnea, independent of the severity of depressive symptoms. Twenty-one patients with obstructive sleep apnea symptomatic for depressive symptoms (mean age = 43.43 years, SD = 9.97), 17 patients with obstructive sleep apnea asymptomatic for depressive symptoms (mean age = 40.65 years, SD = 9.39), and 20 healthy controls without sleep-disordered breathing (mean age = 32.80 years, SD = 6.69) completed an Autobiographical Memory Test. Patients with obstructive sleep apnea symptomatic for depressive symptoms recalled significantly fewer specific memories when compared with healthy controls ($P = 0.010$). No difference in the recall of specific autobiographical memory was observed between symptomatic and asymptomatic patients with obstructive sleep apnea. With regard to valence, symptomatic patients with obstructive sleep apnea recalled significantly fewer negative specific memories when compared with controls ($P = 0.010$). Impairment in specific autobiographical memory recall can be observed in patients with obstructive sleep apnea, regardless of the severity of depressive symptoms; however, this effect may not be as prominent in younger patients with obstructive sleep apnea.

INTRODUCTION

Obstructive sleep apnea (OSA) has been linked to several neuropsychological deficits, such as memory, attention and executive functioning impairment (Jackson *et al.*, 2011). For instance, verbal memory deficits for both immediate and delayed recall of information are commonly found in untreated patients with OSA (Twigg *et al.*, 2010; Wallace and Bucks, 2013). In addition to cognitive impairment, OSA has also been associated with higher prevalence rates of major depressive disorder and self-reported depressive symptoms (Naqvi *et al.*, 2014; Ohayon, 2003). Depressive symptoms include cognitive-affective symptoms, such as depressed mood, loss of interest and feelings of guilt/worthlessness; and somatic symptoms, such as fatigue, excessive daytime sleepiness and appetite change (American Psychiatric Association, 2013).

Despite the overlap in symptoms such as fatigue and excessive daytime sleepiness, there is evidence that some depressive symptoms in patients with OSA are independent

and not solely attributable to the physical symptoms of OSA (Kingshott *et al*., 2000; Means *et al*., 2003). While it is challenging to determine the presence of clinical depression in OSA, self-rated depression questionnaires with a low proportion of overlapping symptoms, such as the Hospital Anxiety and Depression Scale, and the Center for Epidemiology Studies Depression Scale (CES-D), can be indicative of the severity of depressive symptoms in patients with OSA (Nanthakumar *et al*., 2015). Studies have investigated potential correlates of depression and OSA, such as the apnea–hypopnoea index (AHI), nocturnal oxygen desaturations, body mass index (BMI), age and gender (Aloia *et al*., 2005; Bardwell *et al*., 1999; McCall *et al*., 2006; Ong *et al*., 2009). While age, gender and BMI have been identified to be potential confounders to the relationship between OSA and depression, previous findings have generally failed to find correlations between depression and OSA indices (i.e. AHI and nocturnal oxygen desaturation; Bardwell *et al*., 1999; Harris *et al*., 2009). As nocturnal indices have been reported to be poor predictors of depression in OSA, further research is required to identify other possible correlates.

Patients with OSA with depressive symptoms have been shown to have notably greater neural injury when compared with patients with OSA asymptomatic of depressive characteristics (Cross *et al*., 2008; Kumar *et al*., 2009). As a result of the increased neural injury, depressive symptoms may exacerbate cognitive impairments observed in patients with OSA. In non-OSA samples, studies have previously identified correlates of cognitive impairments in depression, and one such correlate is specific autobiographical memory (AM) recall. AM is defined as 'memory for events of one's life' (Conway and Rubin, 1993, p. 103), while the retrieval of a specific AM is described as a memory of a single event that occurred at a particular time and place, with a time span of less than a day. Accordingly, a memory that reflects repeated activities (categoric), or a memory that describes an event that lasted longer than a day (extended), would be categorized as an overgeneral AM (Williams, 2005).

Autobiographical memory overgenerality was first recognized by Williams and Broadbent (1986), who showed that depressed individuals with suicidal behaviours had difficulty recalling specific memories, and were more inclined to retrieve overgeneral memories than healthy controls when given emotional cue words (e.g. happy, interested, clumsy, angry). Most studies have since used the Autobiographical Memory Test (AMT; Williams and Broadbent, 1986) to elicit specific memories, and congruent findings have been observed across several clinical populations (Brewin *et al*., 1998; Croll and Bryant, 2000; McNally *et al*., 1994).

Although numerous studies have established a link between AM and depression, limited studies have been conducted to examine this relationship in the OSA population. Svaldi and Mackinger (2003) and Mackinger and Svaldi (2004) evaluated the predictive power of AM recall as a marker for the course of depression in patients with OSA. Patients with OSA who recalled a greater number of specific memories to positive cue words at baseline showed better recovery from depression after 6–9 weeks of continuous positive airway pressure therapy (Svaldi and Mackinger, 2003). On the other hand, negative cue words were shown to be poor predictors of the course of depression in patients with OSA. In the second study (Mackinger and Svaldi, 2004), it was reported that the recall of specific AM to positive cue words predicted the cognitive-affective, but not the somatic, symptoms of depression. However, both studies did not clearly distinguish between patients with and without depression, thus it is still unclear if overgeneral AM can be observed in patients with OSA without depression.

Given that patients with OSA exhibit a range of memory disturbances (Twigg *et al*., 2010), and may have neuronal damage in regions associated with memory processing (Bartlett *et al*., 2004; Morrell *et al*., 2010), it is also possible that impairments in AM may occur in patients with OSA due to aspects of the sleep disorder itself, such as sleep fragmentation or intermittent hypoxia, rather than depression. Thus, it is unclear whether impairment in specific AM recall is associated with OSA or is specifically a feature of depression. The primary aim of this study was to determine if patients with OSA, both symptomatic and asymptomatic for depressive symptoms, recall fewer specific AM when compared with healthy controls. To examine the effect of OSA independent of depression on specific AM recall, a healthy control group was included for comparison (i.e. patients with OSA asymptomatic for depressive symptoms versus controls). Second, as specific AM recall to positive cue words was shown to be a better predictor for the course of depression (Mackinger and Svaldi, 2004; Svaldi and Mackinger, 2003), the study also aimed to determine if the valence of cue words affected the overgenerality of memory recall in patients with OSA.

MATERIALS AND METHODS

Participants

The study population consisted of 58 patients with OSA (age range: 26–59 years; 14 females) and 20 healthy individuals (age range: 23–45 years; six females) with no sleep disorder. Based on the CES-D cutoff score ($\geq$ 16; Weissman *et al*., 1977), the patients with OSA were divided into two groups: patients with OSA symptomatic for depressive symptoms (OSA-s, $n = 21$); and patients with OSA asymptomatic for depressive symptoms (OSA-a, $n = 17$). The patients with OSA were recruited from the Austin Health sleep clinic and from community advertising, and the healthy controls were recruited through advertisements placed around the University of Melbourne campus. Written informed consent was obtained and the study was approved by the University of Melbourne Human Research Ethics Committee.

The inclusion criteria for the healthy control group were an Epworth Sleepiness Scale (ESS) score of 10 or less, a Pittsburgh Sleep Quality Index (PSQI) score of 10 or less, a

Multivariable Apnea Prediction Index (MAPI) score of 0.5 or less, and a CES-D score of 15 or less. Participants in the control group underwent a night of polysomnography (PSG) screening to confirm the absence of any sleep disorders. The inclusion criterion for the OSA groups was a clinical diagnosis of OSA via PSG, with an AHI of more than 10. Participants were excluded if they had a history of drug or alcohol dependence, learning disability, brain injury, and had been or were currently involved in shift work. Eight patients from the OSA-D group reported taking antidepressants.

Materials

Autobiographical Memory Test

The cue words for the AMT were drawn from a sample of cue words used by Williams (2005). These were grouped according to the emotionality ratings and Kucera–Francis written frequency, with the positive and negative cue words being high in positive or negative emotional valence, respectively, and high-frequency words. The cue words consisted of 12 test items: six positive words (e.g. happy, relieved, sunny) and six negative words (e.g. guilty, hopeless, failure). The words were presented in the same randomized order for each participant. Three practice words were presented initially to ensure the participants understood the instructions. Participants were given 1 min to recall a memory.

All the responses were tape-recorded, transcribed in detail and scored. Each response was scored as specific, overgeneral or no response, and 1 point was given for each specific memory recalled and zero for each overgeneral or no response, with a maximum total score of 12 (six for negative and six for positive cue words). The outcome measures from the AMT include the number of positive and negative specific memories recalled, and the total specific memories recalled. Twenty per cent of the transcripts were scored by a second scorer for inter-rater reliability.

Actigraphy and sleep diary

The actigraph watch (Actiwatch-2, Philips Respronics) is an ambulatory monitory device with a photometer and a linear accelerometer that scores sleep and wakefulness from recorded activity data. Together with the actigraph watch, participants were also asked to keep a daily sleep diary that reported sleep period and quality as well as daytime naps and sleepiness. The Actiwatch-2 data were analysed with the Actiware software (Philips Respironics Inc., Murrysville, PA, USA).

Polysomnography

A standard clinical PSG assessment was performed using a Compumedics E-Series system on the control participants to confirm absence of a sleep disorder. Data were collected on Compumedics ProFusion PSG 3 V3.3. The PSG recordings included standard placements, based on the international 10-20 system, for continuous monitoring of central and occipital electroencephalogram (F3/F4, C3/C4 and O1/O2) with A1 and A2 as reference. Also, horizontal electrooculogram, submental and anterior tibialis electromyogram and electrocardiogram were collected. Nasal airflow was sensed by measurement of end tidal CO_2 using a thermistor (Compumedics Reusable Airflow Sensor), while the thoracic and abdominal excursion were obtained by Respiratory Inductance Plethysmography (Compumedics Respiratory Effort Belt). Vibration associated with snoring was recorded by a snore sensor (Sleepmate Snore Sensor), and continuous arterial oxygen saturation was recorded by a finger oximeter probe. Leg movements were recorded using a limb movement sensor.

All variables were recorded on a 17-channel PSG, and PSG data were analysed by a trained sleep technician according to the AASM Manual for the Scoring of Sleep and Associated Events (American Academy of Sleep Medicine, 2007) in 30-s epochs. The outcome measures included the AHI (events per hour), arousal index, sleep efficiency, total sleep time and percentage of sleep time when oxygen saturation was below 90%.

All OSA participants had completed a clinical PSG prior to enrolment.

Procedure

All participants completed a telephone screening session to ensure they met the initial inclusion criteria, and then attended a general screening session at the University of Melbourne Sleep Laboratory. They were informed of the study and gave written informed consent. The participants were then required to complete a set of screening questionnaires: the MAPI, ESS, PSQI and CES-D.

Participants who met the inclusion criteria, based on their responses to the screening questionnaires, were scheduled for a testing session approximately 1 week after the general screening session, and were asked to maintain a regular sleep schedule throughout the week. To verify their sleep–wake cycle, all of the eligible participants were required to wear an actigraph watch and record their sleep periods in a sleep diary for at least 5 days prior to the testing session.

At approximately 17:00 hours, the AMT was conducted in a quiet testing room. Subsequently, participants in the OSA group were discharged while individuals in the control group underwent PSG on the night of the testing session. All control participants had at least 7 h of lights out, and the sleep studies were scored by a trained sleep technician. One control participant was excluded on the basis of their PSG study, which indicated mild sleep-disordered breathing.

Statistical analysis

All statistical analyses were carried out using IBM SPSS Statistics 21 (SPSS, Chicago, IL, USA). An alpha level of

0.05 was considered to be of statistical significance. The total number of specific AM recalled was analysed using a one-way analysis of variance (ANOVA) for comparison between the groups (controls, OSA-N, OSA-D). To examine the effect of valence on AM retrieval, a mixed design ANOVA was applied to evaluate the group (control, OSA-N, OSA-D) by cue valence (positive, negative) interaction. Significant group effects were analysed *post hoc* using Tukey's Honestly Significant Difference (HSD) tests.

RESULTS

Sample characteristics

Demographic and PSG data of each group (control, OSA-a and OSA-s) are presented in Table 1. A chi-squared test indicated no significant difference in gender distribution among the three groups [$X^2(2, n = 58) = 1.02, P = 0.600$]; however, the groups significantly differed on BMI and age. While the OSA-a and OSA-s groups did not significantly differ on objective sleep measures (i.e. AHI and arousal index), they differed significantly on subjective sleep measures (i.e. ESS and PSQI), with the OSA-s group scoring notably higher in both measures. The OSA-s group scored significantly higher on the ESS and PSQI when compared with controls, but no difference was observed between the OSA-a group and controls.

Autobiographical Memory Test

A one-way ANOVA revealed a significant main effect of group for the number of specific AM recalled ($P = 0.008$). A *post hoc* Tukey's HSD test showed that both the OSA-s group ($P = 0.010$) and OSA-a ($P = 0.042$) group recalled significantly fewer specific AM than the controls, while the difference between OSA-a and OSA-s groups was not significant ($P = 0.91$; Table 1).

There was a significant group difference in negative specific memories recalled ($P = 0.008$), but there was no significant difference between groups for positive memories ($P = 0.094$). A *post hoc* Tukey's HSD test showed that the OSA-s group recalled significantly fewer negative specific AM than the controls ($P = 0.010$). Although not significant, the *post hoc* tests revealed a trend towards significance between the OSA-a and controls ($P = 0.063$). There was no significant group difference in positive specific memories recalled ($P = 0.112$; Table 1).

Due to the significant difference in age between the groups, a subset analysis for the AMT was conducted with younger patients with OSA and controls between the ages of 25 and 49 years. The OSA-s group recalled notably fewer specific AM than the controls ($P = 0.007$), and the difference between OSA-a and OSA-s groups remained not significant. The difference between the OSA-a group and controls ($P = 0.091$) was also not significant. Regarding valence, similar findings were observed. A *post hoc* Tukey's HSD revealed that the OSA-s groups recalled notably fewer negative AM than controls ($P = 0.007$), but no significant difference were observed between the groups in positive specific memories recalled (Table 2).

DISCUSSION

Consistent with previous studies (Mackinger and Svaldi, 2004; Svaldi and Mackinger, 2003), the current study

Table 1 Demographic and polysomnography data of the study sample ($n = 58$)

	Control	*OSA-a*	*OSA-s*	*F*	*P*	*Effect size*
n	20	17	21			
Gender (women/men)	6/14	4/13	9/12			
Age (years)*,‡	32.80 (6.69)	40.65 (9.39)	43.43 (9.97)	7.94	0.001	0.23
BMI ($kg \cdot m^{-2}$)*,†,‡	23.72 (3.43)	29.43 (5.24)	33.92 (5.46)	23.31	< 0.001	0.46
ESS (/24)*,†	4.15 (3.05)	5.84 (2.52)	9.05 (4.61)	9.93	< 0.001	0.27
PSQI*,†	4.45 (2.06)	6.42 (2.29)	10.76 (3.18)	32.03	< 0.001	0.54
CES-D*,†	6.70 (5.09)	8.82 (3.41)	29.86 (8.21)	90.06	< 0.001	0.77
AHI (/h)*,‡	0.09 (0.23)	23.81 (6.91)	27.95 (8.21)	29.92	< 0.001	0.52
Arousal index (no. per h)*,‡	8.48 (3.50)	24.51 (6.99)	26.31 (18.37)	13.56	< 0.001	0.33
Sleep efficiency (%)*	87.62 (9.03)	78.88 (13.7)	80.22 (8.15)	3.99	< 0.05	0.13
Total sleep time (min)‡	429.00 (50.31)	359.57 (66.86)	393.61 (95.04)	4.07	< 0.05	0.13
Time oxygen saturation below 90% (%)	0.33 (0.59)	2.39 (2.82)	4.08 (9.71)	1.97	0.15	0.07
Specific AM score						
Total*,‡	9.80 (1.82)	7.88 (2.37)	7.57 (2.73)	5.29	0.008	0.16
Positive	5.10 (1.07)	4.24 (1.39)	4.24 (1.67)	2.47	0.094	0.08
Negative*	4.70 (1.03)	3.59 (1.77)	3.33 (1.53)	4.98	0.010	0.15

AHI, apnea–hypopnoea index; AM, autobiographical memories; BMI, body mass index; CES-D, Center for Epidemiologic Studies – Depression scale; ESS, Epworth Sleepiness Scale; OSA-a, patients with obstructive sleep apnea asymptomatic for depressive symptoms; OSA-s, patients with obstructive sleep apnea symptomatic for depressive symptoms; PSQI, Pittsburgh Sleep Quality Index.
*$P < 0.05$ between control and OSA-s groups.
†$P < 0.05$ between OSA-a and OSA-s groups.
‡$P < 0.05$ between control and OSA-a groups.

Table 2 AM test data of younger participants (n = 46)

	Control(n = *17)*	*OSA-a (*n = *14)*	*OSA-s (*n = *15*)	F	P	*Effect size*
Age	33.53 (6.12)	37.50 (6.87)	38.77 (7.71)	2.54	0.090	0.10
Specific AM score						
Total*	9.94 (1.47)	8.07 (2.49)	7.20 (3.075)	5.47	0.008	0.20
Positive	5.18 (0.80)	4.14 (1.40)	4.00 (1.85)	3.39	0.043	0.14
Negative*	4.76 (0.97)	3.86 (1.66)	3.20 (1.52)	5.12	0.010	0.19

AM, autobiographical memories; OSA-a, patients with obstructive sleep apnea asymptomatic for depressive symptoms; OSA-s, patients with obstructive sleep apnea symptomatic for depressive symptoms.
*$P < 0.05$ between control and OSA-s groups.

supports the findings of an AM overgenerality effect in OSA individuals symptomatic for depressive symptoms when compared with healthy controls, and extends these findings by demonstrating that the recall of specific AM does not differ between patients with OSA symptomatic and asymptomatic for depressive symptoms.

As the current findings indicated no distinction between asymptomatic and symptomatic patients with OSA in specific AM recall, it is possible that AM impairment in patients with OSA is not solely mood state-dependent, but rather may reflect a long-standing impairment; potentially a result of the underlying neurological impairment reported in patients with OSA or other co-morbidities that were not controlled for in this study. Similarly, previous studies have reported verbal episodic memory impairments in patients with OSA across a range of OSA severities (Twigg *et al*., 2010; Wallace and Bucks, 2013). The retrieval of more detailed memory has been shown to correlate with an increase in hippocampal activity (Addis *et al*., 2004) and, in conjunction, magnetic resonance imaging studies have reported significant impairment in grey matter concentration within the left hippocampus in patients with OSA (Greenberg *et al*., 2005; Morrell *et al*., 2003). However, due to the nature of the present study, the neurological involvement can only be speculated, and future studies could explore the potential role of hippocampal impairment in the development and maintenance of AM impairment in patients with OSA.

Unlike previous studies that have reported lesser recall of positive specific memories in individuals with depression (Puffet *et al*., 1991; Williams and Dristchel, 1988), the current study found that patients with OSA with depressive symptoms tended to recall fewer negative specific memories when compared with healthy controls. This lack of specificity was not observed with memories prompted by positive cue words. One explanation is that in order to avoid the possibility of being confronted by negative memories that may be distressing and painful, depressed individuals may adopt an avoidance strategy by repressing the memory and providing a non-specific response to the negative cue words (Williams, 2007).

To take into account the age difference between the groups and potential memory impairment observed in older adults, a subset consisting of younger patients with OSA age-matched to the control group was examined. Similar findings were observed in that a lack of difference in the recall of specific AM between asymptomatic and symptomatic patients with OSA was observed. While younger patients with OSA symptomatic for depressive symptoms recalled notably fewer specific AM when compared with the control, the difference between the controls and asymptomatic patients with OSA did not reach statistical significance (P = 0.09). This suggests that there may be an age-related effect on AM, in that younger patients with OSA do not show such impairments. It is likely that younger patients with OSA have had the disease for a shorter period of time, and therefore may have not developed cognitive and/or neural changes. It is also possible that younger patients with OSA have a higher 'cognitive reserve', which may allow them to retain memory function more than older patients. The lack of a significant result could also be the smaller sample size of 14 asymptomatic patients with OSA, which may reduce potential statistical significance. Given that impairments were only seen in the symptomatic group relative to the controls, this finding could suggest that depression may have a bigger impact on overgeneral AM recall in younger patients with OSA.

Other potential confounding factors in the current study include obesity and medical co-morbidities such as hypertension and diabetes. Six patients with OSA reported hypertension, which was balanced across the two OSA groups, and two symptomatic patients had diabetes. Higher BMI has been associated with greater cognitive decline and poorer cognitive performance in older adults (Cournot *et al*., 2006; Sabia *et al*., 2009). As obese individuals have been reported to have smaller whole brain volume and total grey matter volume when compared with normal weight and overweight individuals (Gunstad *et al*., 2008), it is possible for high BMI scores to contribute towards the impairment in specific AM recall. It was proposed that cardiovascular diseases and impaired insulin regulation may be potential underlying mechanisms for the memory deficits seen in individuals with elevated BMI (Gunstad *et al*., 2006, 2008). Despite the difference in BMI scores between the groups in the current study, the study population consists of a fairly young OSA sample with minimal medical co-morbidities.

A limitation of the current study is that antidepressant intake in patients with OSA with symptomatic depressive

symptoms was not controlled for. Eight patients from the OSA-s group were on antidepressants. Past studies have reported that depressed individuals on antidepressants still demonstrated impairment in specific AM recall (Brittlebank *et al*., 1993; Goddard *et al*., 1997), therefore these participants were included in the study. Interestingly, they all reported high depressive symptoms and were classified in the symptomatic group despite their current depression treatment. A further limitation is the younger age of the control group. The memory performance of the control group was similar to older healthy individuals, therefore we do not believe that the difference found between patients and controls was solely due to age but, rather, similar findings would be expected when comparing older healthy adults with age-matched patients. With regards to gender effects, the current study did not have sufficient sample size to examine differences between male and female patients with OSA on depression scores and AM overgenerality. Future research would benefit from examining both age and gender differences in depression in OSA and the potential impact on AM.

In summary, the present study demonstrated that patients with OSA have difficulties recalling specific AM when prompted with cue words, regardless of the severity of their depressive symptoms. As AM impairment has been linked with the loss of identity, depression and a decrease in perception of quality of life in various clinical populations, AM deficit in these patients may have a significant impact on well-being (Jetten *et al*., 2010; Kenealy *et al*., 2000). Future studies would benefit from examining whether the impairment in specific AM recall has neurological underpinnings, and whether these deficits are reversible with treatment.

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CONFLICT OF INTEREST

The authors have no conflicts of interest to disclose.

AUTHORS' CONTRIBUTIONS

All authors contributed to the study conception and design, and the drafting of the manuscript. V. L. and M. L. J. collected the data and performed the analysis.

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