



Original Article

Obstructive sleep apnea exaggerates cognitive dysfunction in stroke patients



Yan Zhang ^{a, b, *, 1}, Wanhua Wang ^{a, b, 1}, Sijie Cai ^c, Qi Sheng ^c, Shenggui Pan ^d, Fang Shen ^{a, b}, Qing Tang ^{a, b}, Yang Liu ^e

^a Department of Neurology, The Kunshan Affiliated Hospital of Jiangsu University, 215300 Kunshan, China

^b Department of Neurology, The First People's Hospital of Kunshan, 215300 Kunshan, China

^c Department of Respiratory Medicine, The Kunshan Affiliated Hospital of Jiangsu University, 215300 Kunshan, China

^d Department of Rehabilitation Medicine, The Kunshan Affiliated Hospital of Jiangsu University, 215300 Kunshan, China

^e Department of Neurology, Saarland University, 66421 Homburg, Germany

ARTICLE INFO

Article history:

Received 19 October 2016

Received in revised form

20 November 2016

Accepted 28 November 2016

Available online 4 February 2017

Keywords:

Cognition

Obstructive sleep apnea

Prospective memory

Sleep and stroke

ABSTRACT

Background: Obstructive sleep apnea (OSA) is very common in stroke survivors. It potentially worsens the cognitive dysfunction and inhibits their functional recovery. However, whether OSA independently damages the cognitive function in stroke patients is unclear. A simple method for evaluating OSA-induced cognitive impairment is also missing.

Methods: Forty-four stroke patients six weeks after onset and 24 non-stroke patients with snoring were recruited for the polysomnographic study of OSA and sleep architecture. Their cognitive status was evaluated with a validated Chinese version of Cambridge Prospective Memory Test. The relationship between memory deficits and respiratory, sleeping, and dementia-related clinical variables were analyzed with correlation and multiple linear regression tests.

Results: OSA significantly and independently damaged time- and event-based prospective memory in stroke patients, although it had less power than the stroke itself. The impairment of prospective memory was correlated with increased apnea–hypopnea index, decreased minimal and mean levels of peripheral oxygen saturation, and disrupted sleeping continuity (reduced sleep efficiency and increased micro-arousal index). The further regression analysis identified minimal levels of peripheral oxygen saturation and sleep efficiency to be the two most important predictors for the decreased time-based prospective memory in stroke patients.

Conclusions: OSA independently contributes to the cognitive dysfunction in stroke patients, potentially through OSA-caused hypoxemia and sleeping discontinuity. The prospective memory test is a simple but sensitive method to detect OSA-induced cognitive impairment in stroke patients. Proper therapies of OSA might improve the cognitive function and increase the life quality of stroke patients.

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1. Introduction

Obstructive sleep apnea (OSA) is characterized by repeated collapses of the upper airway during sleep, and is common in the general population. Its mean prevalence is 22% and 17% in men and

women, respectively [1]. In stroke patients, the prevalence can rise to 72% [2]. OSA is an independent risk factor of stroke [3,4]. It also contributes to the occurrence of stroke through exaggerating hypertension, diabetes mellitus, and cardiovascular disease [5–7]. Moreover, OSA has been shown to inhibit the recovery of stroke patients. Stroke patients with OSA showed lower functional capacity and spent longer time in the rehabilitation than non-OSA stroke patients [8,9]. Fortunately, OSA is treatable with continuous positive airway pressure (CPAP) and maxillomandibular advancement [10–12]. Thus, proper OSA therapy not only prevents stroke, but also optimizes the stroke rehabilitation. Since there are still more than 10 million major strokes every year in the world

DOI of original article: <http://dx.doi.org/10.1016/j.sleep.2016.12.014>.

* Corresponding author. Department of Neurology, The Kunshan Hospital Affiliated to Jiangsu University, Qianjinxi Road, No. 91, 215300 Kunshan, Jiangsu Province, China. Fax: +86 512 57501112.

E-mail address: 2573560757@qq.com (Y. Zhang).

¹ These two authors contributed equally to this study.

<http://dx.doi.org/10.1016/j.sleep.2016.11.028>

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[13], a timely diagnosis of OSA in stroke patients and the following recovery training has become extremely important.

Cognitive dysfunction, shown as a variety of deficits in attention, executive function, memory, language and visuospatial ability, is a common consequence of stroke [14,15]. Prospective studies have shown that impaired visuospatial construction and memory, or inattention and perceptual disorders within one month after stroke, predicts a poor functional outcome after six months [16,17]. Recently, OSA has been shown to worsen the attention, executive functioning, visuospatial perception, psychomotor ability, and intelligence of stroke patients, which causes difficulty in the rehabilitation of stroke patients [18]. In the follow-up study, CPAP therapy was able to significantly improve the cognitive function of stroke patients, although the effect of CPAP on functional recovery is still under investigation [19]. Thus, OSA potentially damages the cognitive function, which subsequently mediates the negative effects of OSA on stroke recovery. The improvement of cognitive function can be used to evaluate the efficacy of anti-OSA therapy in stroke patients.

However, studies investigating the relationship between OSA and cognitive impairment in stroke patients are rare. In few published studies, there was not a control group of non-stroke patients with OSA, which can be used to investigate the independent effects of OSA and stroke on cognitive dysfunction [18,19]. The underlying pathophysiological mechanisms through which OSA impairs the cognitive function of stroke patients are unclear either. To address these questions, our study recruited both stroke and non-stroke patients and carefully examined both respiratory parameters and the structure of sleep. In order to find a method with which the clinicians (especially in local hospitals and without extensive training for psychological analysis), can also successfully examine the cognitive status of stroke patients, we used the prospective memory test to evaluate cognitive function; the patient was simply requested to perform a planned action at a certain future time or after a certain event occurred [20].

2. Materials and methods

2.1. Patients

In this study, all 65 stroke patients (ischemic, hemorrhagic and subarachnoid hemorrhage) admitted to the Department of Neurology, Kunshan affiliated Hospital of Jiangsu University, China, from June 2013 to June 2015 were invited, and 44 patients were enrolled into this study according to the following criteria: (1) stroke confirmed by a neurologist, (2) age between 30 and 65 years old, (3) admission at six weeks after stroke, (4) able to participate in the sleep study and neuropsychological assessment, and (5) sufficiently fluent in lingual communication. The exclusion criteria was as follows: (1) severe, unstable medical conditions, respiratory failure, or history of severe congestive heart failure, (2) traumatic brain injury, (3) severe aphasia, confusion, or severe psychiatric comorbidity, (4) central sleep apnea, or (5) any previously diagnosed sleep diseases including narcolepsy, periodic limb movement disorder and Parkinson's disease-related sleep disorders. In order to investigate the relative contribution of OSA to cognitive dysfunction of stroke patients, we also recruited 24 non-stroke patients, who were over 18 years old and came to our outpatient service due to snoring (they had no previous diagnosis of neurological, psychiatric and/or respiratory disease). All subjects received overnight polysomnography. Before polysomnography, the subjects were evaluated for sleeping and cognitive function by using Epworth sleepiness scale (ESS), Mini-mental state examination (MMSE), and prospective memory test. The study was approved by the Ethics Committee of Jiangsu

University and written informed consent was obtained from each participant.

2.2. Polysomnographic study

All stroke patients underwent one night (≥ 8 h) of polysomnographic study at the sleep laboratory of our department six weeks after stroke onset. Non-stroke patients participated in the polysomnographic (PSG) test without drinking alcoholic or caffeinated drinks in the last two days. The overnight sleep polysomnograph (Alice 5 Diagnostic Sleep System; Philips Healthcare, Andover, USA) included electroencephalography (C3/A2, C4/A1, O1/A2, and O2/A1), electrooculography, submental electromyography, bilateral anterior tibialis electromyography, electrocardiography, nasal airflow measurement (sensed by both thermistor and pressure transducer), monitoring of thoracoabdominal movements, oxygen saturation, snoring and body position.

Sleep stages and respiratory events were analyzed against the Sleep Medicine Criteria (American Academy, 2007) [21]. Apnea was defined as a reduction of airflow of $\geq 90\%$ for at least 10 s and hypopnea was defined as a reduction of airflow of $\geq 50\%$ for at least 10 s followed by an oxygen desaturation of $\geq 3\%$. Apneas with thoracic motion, without thoracic motion, and with initial lack of motion followed by respiratory effort, were classified as obstructive, central, and mixed apneas, respectively. OSA was diagnosed when at least 50% of the respiratory events were of the obstructive type. The apnea-hypopnea index (AHI) was defined as the mean number of apneas and hypopneas per hour in bed. Patients with AHI < 5 were referred to as non-OSA patients and enrolled in the control group of this study. Other OSA-associated respiratory parameters, including minimum of peripheral oxygen saturation (SpO₂), mean of SpO₂ and percentage of time with SpO₂ at $< 90\%$ were recorded. Sleep architecture variables were also examined, such as sleep efficiency, sleep stages, and microarousal index. Sleep efficiency is percentage of sleeping duration divided by the total time spent in the bed. Microarousal index was the number per hour of microarousals which last 3–15 s.

2.3. Epworth sleepiness scale (ESS)

The ESS is a self-administered questionnaire that is widely used for subjective assessment of daytime sleepiness. It contains eight items involving eight daily-life scenarios, with each item being assessed on a zero-to-three point scale. The total scores of ESS range from 0 to 24. The cutoff point for excessive daytime sleepiness is set at > 10 [22].

2.4. Mini-mental state examination

The Chinese version of the MMSE questionnaire consists of several subscales: orientation, immediate and short-term memory recall, attention/calculation, language, and visuospatial skills. Cognitive deficit on the MMSE is defined as a score less than 27 [23].

2.5. Prospective memory test

The prospective memory test was assessed with a validated Chinese version of Cambridge Prospective Memory Test (CAM-PROMPT) [24]. The CAM-PROMPT comprises six prospective memory tasks which are either cued by events (event-based prospective memory tasks; $n = 3$) or by time (time-based prospective memory tasks; $n = 3$). In this test, participants were asked to remember carrying out three time- and three event-based prospective memory tasks while working on a number of other activities (pencil and

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