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Original article

Effectiveness of a portable device and the need for treatment of mild-to-moderate obstructive sleep-disordered breathing in patients with cardiovascular disease

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KEYWORDS
Portable device;
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Summary
Background and purpose: In Japan, there are two sleep-disordered breathing (SDB)-related problems, which include diagnosing SDB using a portable device (PD) and treating mild-to-moderate SDB (mm-SDB) using continuous positive airway pressure (CPAP) for severe SDB (s-SDB) in obstructive sleep apnea (OSA) patients. Our aims were to evaluate the effectiveness of a PD in diagnosing SDB in patients with cardiovascular disease (CVD), and to assess the difference between mm-SDB [apnea-hypopnea index (AHI): 20—40 h^{-1}] and s-SDB (AHI: >40 h^{-1}) using brain natriuretic peptide (BNP) in OSA patients.
Methods and subjects: After their underlying CVD was treated, full-night sleep studies using polysomnography (PSG) and PD were performed on the same day.
Results: Eighty-three patients underwent full-night PSG simultaneously with PD. The average duration of the sleep study was 8.6 ± 6.2 days. There was a tendency for a higher AHI value obtained with PSG (PSG, $28.9 \pm 24.3\ h^{-1}$; PD, $22.3 \pm 16.7\ h^{-1}$; $p = 0.05$). However, the specificity and sensitivity of diagnosing SDB using PD were 86% and 81%, respectively. Using PD, twenty-nine OSA patients had mm-SDB and eleven patients had s-SDB. The BNP value was higher in the mm-SDB patients (318 ± 550 pg/ml) than in the s-SDB patients (202 ± 160 pg/ml).
Conclusions: The PD was effective in diagnosing SDB in patients with CVD. The BNP value was higher in the mm-SDB patients. Therefore, they need to be treated with CPAP to treat underlying CVD.

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Introduction

Sleep-disordered breathing (SDB) is closely related to cardiovascular disease (CVD), including heart failure (HF) and coronary artery disease (CAD) [1—4]. Furthermore, we reported that SDB is a strong risk factor for fatal cardiovascular events including acute myocardial infarction, fatal arrhythmias, and cardio-embolic stroke [5]. In future, it might be more important to diagnose and treat SDB in order to treat underlying CVD. However, there remain two problems related to SDB. These include diagnosing SDB using a portable device (PD), and treating mild-to-moderate SDB (mm-SDB) [apnea-hypopnea index (AHI): 20—40 h^{-1}] using continuous positive airway pressure (CPAP) for patients with obstructive sleep apnea (OSA) by Japanese health insurance rules. Full-night polysomnography (PSG) is a standard device used globally to diagnose the type and severity of SBD. Conversely, a PD is commonly used as a substitute to diagnose SDB. However, this device has several problems; i.e. it is impossible to assess patients' sleep and arousal periods, sleep stage, and electroencephalograms (EEG). Furthermore, there have not been many clinical trials evaluating the effectiveness of a PD in diagnosing SDB [6—13]. Our aims were to evaluate the effectiveness of a PD in diagnosing SDB in patients with CVD and to assess the difference between mm-SDB (AHI: 20—40 h^{-1}) and severe-SDB (s-SDB) (AHI: >40 h^{-1}) using brain natriuretic peptide (BNP) in patients with OSA.

Methods and materials

Study population

Between December 2007 and August 2008, 99 consecutive patients with CAD who were admitted to hospital because of anterior chest pain, or with HF who had symptoms defined as the establishment of New York Heart Association grade II or IV participated in the study. This study was conducted in accordance with the recommendations of the Helsinki Declaration of 1975, and the protocol was approved by our Medical Center Institutional Review Board. Informed consent was provided by each patient before entering into the study. In the course of our study, no adverse effects were observed and there was no change to the protocol after the study began. Sixteen patients without sufficient sleep study data were excluded.

Data collection

Baseline data included transthoracic echocardiography, medical history, physical examination, and biochemical examination of blood. After treating the underlying disease, transthoracic echocardiography was performed before the overnight sleep study. Venous blood samples were obtained on admission, after an overnight fast. BNP levels were estimated before the study. Arterial blood samples were obtained for blood gas analysis when the patients were supine and not receiving supplemental oxygen. Dyslipidemia was defined as the current use of cholesterol-lowering drugs, a triglyceride value of $\geq$150 mg/dl, a low-density lipoprotein cholesterol value of $\geq$140 mg/dl, and/or a high-density lipoprotein cholesterol value of <40 mg/dl. Hypertension was defined as the current use of antihypertensive drugs or a blood pressure of $\geq$140/90 mmHg. Diabetes mellitus was defined as the current use of insulin or antidiabetic drugs, fasting blood glucose value of $\geq$126 mg/dl, and/or a hemoglobin A_{1c} value of $\geq$6.5%.

Sleep study

All procedures were performed at our medical center. Patients receiving inotropic therapy were studied after this treatment was discontinued. After their underlying CVD was treated, full-night sleep studies using PSG and PD were performed on the same day. The average duration of the sleep study was 8.6 $\pm$ 6.2 days. During sleep evaluation, all patients underwent overnight PSG using a digital polygraph (P-Series Plus; Compumedics, Abbotsville, Australia). The assessment included EEG monitoring, an electrooculogram, a chin electromyogram, and monitoring of chest and abdominal movements using two bands (inductive respiratory bands). Airflow was assessed using a thermistor. Arterial oxygen saturation (SaO_2) was measured continuously using a pulse oximeter with a finger probe (Nonin 8000J Adult Flex Sensor, Plymouth, MN, USA) (sampling frequency: 1 s). Sleep staging and arousals were scored according to standard methods [14,15]. Apnea was defined as the complete cessation of airflow lasting $\geq$10 s. Hypopnea was defined as a $\geq$50% decrease in airflow for $\geq$10 s, accompanied by desaturation (3% decrease in SaO_2) and/or an EEG arousal response. AHI was defined as the total number of apneas and hypopneas per hour of sleep. An AHI of >20 h^{-1} was considered abnormal. The PD (Morpheus; Compumedics, Melbourne, Australia) was used to identify sleep apnea and sleep time, with proper monitoring by a physician. Airflow (using a thermistor), SaO_2, heart rate, and respiratory movements (using chest and abdominal bands) were continuously monitored. The AHI was calculated as the number of times per hour that pulse oximetry indicated an oxygen saturation decrease of more than 4% from the baseline oxygen saturation. Central and obstructive apnea were divided into forms of both airflow and movement of the chest and abdomen. To classify patients with OSA into two groups, the AHI score using PD was selected in this study.

Statistical analysis

Results are expressed as means $\pm$ standard deviations, or counts (expressed as percentage). Results of the sleep study were compared using paired Student's *t* test and clinical characteristics of the OSA population were compared using unpaired Student's *t* test for continuous variables and a chi-square test for categorical variables. A receiver operating characteristic (ROC) curve was drawn to show the sensitivity and specificity of each observed value of AHI. Significance was set at 5% and all analyses were performed using JMP software (JMP 6.0; SAS Institute Inc., Cary, NC, USA).