WAKE-UP STROKE (WUS) is defined as neurological symptoms as noted on awakening, and it comprises 20% to 25% of all ischemic strokes.1,3 The characteristics and risk factors are not different between patients with WUS and those without WUS.1,2,4 Given that WUS occurs when patients are asleep, sleep disorders have been implicated in the development of WUS.5,6 Specifically, sleep-disordered breathing may be associated with the development of WUS.6,8 Nocturnal hypoxia may trigger ischemia10–9 or may increase the risk of ischemic event probably mediated by hypertension. Previous reports have suggested the possible association between WUS and sleep-disordered breathing. However, sleep-disordered breathing is usually diagnosed by polysomnography in highly selected patients, which limits its generalizability. More simple, but sensitive tools to diagnose sleep-disordered breathing are needed to better identify its association with WUS.

Stroke unit (SU) care is effective in improving outcomes in acute stroke. In the SU, the physiological parameters, including heart rate, blood pressure, body temperature, and oxygen saturation, are continuously monitored, which can detect nocturnal desaturation occurring at night even without polysomnography.10,11 Nocturnal oxygen desaturation (NOD) events can be evaluated using the oxygen desaturation index (ODI) based on oxygen saturation data obtained at the SU.12,13 Using high-resolution data, we compared the frequency of overnight NOD events between patients who were admitted for WUS and other patients with stroke. We hypothesized that nocturnal desaturation events in the SU are more common in patients with WUS.

Materials and Methods

Study Population
A total of 556 patients with acute ischemic stroke and transient ischemic attack (within 7 days from stroke onset) were screened during the periods from July 2013 to May 2015. Patients who stayed in the SU on the first night of admission were eligible (n=323). We excluded patients with the following conditions: lack of clinical information and patient death or discharge from the SU at night (PM7:00 to PM 7:00 AM) of the stroke unit admission, and nocturnal desaturation was defined as an oxygen desaturation index of 5 at least per hour. We compared the clinical characteristics and nocturnal desaturations between patients with and without WUS.

Results—Among all patients (age, 67.7±12.6 years; male, 54.4%), 26.5% patients had WUS. The proportion of nocturnal desaturation was significantly greater in patients admitted with WUS (29.1% versus 12.3%, P=0.001). The age, sex, risk factors except for hyperlipidemia, stroke severity, and stroke mechanisms were similar between the 2 groups. After adjustment for covariates, it was found that nocturnal desaturation was significantly more common in the WUS group (odds ratio, 3.25; 95% confidence interval, 1.63–6.46).

Conclusions—Nocturnal desaturation was more frequently observed in patients admitted with WUS during the first night in the stroke unit. This suggests that nocturnal desaturation is a possible modifiable risk factor for the occurrence of WUS. (Stroke. 2016;47:1748-1753. DOI: 10.1161/STROKEAHA.116.013266.)

Key Words: pulse oximetry ▪ risk factor ▪ sleep-disordered breathing ▪ stroke

Nocturnal Desaturation in the Stroke Unit Is Associated With Wake-Up Ischemic Stroke

Tae Jung Kim, MD; Sang-Bae Ko, MD, PhD; Han-Gil Jeong, MD; Ji Sung Lee, PhD; Chi Kyung Kim, MD, PhD; Yerim Kim, MD; Kiwoong Nam, MD; Heejung Mo, MD; Sang Joon An, MD; Huimahn Alex Choi, MD; Byung-Woo Yoon, MD, PhD

Background and Purpose—Wake-up stroke (WUS) represents a quarter of all ischemic strokes and may be a specific subgroup. Nocturnal desaturation secondary to sleep-disordered breathing is an independent risk factor for stroke, but the association between nocturnal desaturation and WUS remains unclear. We investigated the relationship between nocturnal desaturation using oxygen desaturation index and WUS in patients with acute stroke in the stroke unit.

Methods—A total of 298 patients admitted for acute ischemic stroke to the stroke unit between July 2013 and May 2015 were enrolled. The oxygen desaturation index was calculated using pulse oximetry data sampled every 1 minute during 9 hours on the first night (10:00 PM–7:00 AM) of the stroke unit admission, and nocturnal desaturation was defined as an oxygen desaturation index of 5 at least per hour. We compared the clinical characteristics and nocturnal desaturations between patients with and without WUS.

Results—Among all patients (age, 67.7±12.6 years; male, 54.4%), 26.5% patients had WUS. The proportion of nocturnal desaturation was significantly greater in patients admitted with WUS (29.1% versus 12.3%, P=0.001). The age, sex, risk factors except for hyperlipidemia, stroke severity, and stroke mechanisms were similar between the 2 groups. After adjustment for covariates, it was found that nocturnal desaturation was significantly more common in the WUS group (odds ratio, 3.25; 95% confidence interval, 1.63–6.46).
(n=10) and oxygen therapy during SU care (n=15). A total of 298 patients (age range, 32–95 years) were included for analysis. The patients were divided into 2 groups: WUS (n=79, stroke during sleep) and non-WUS (n=219). This study was approved by the Institutional Review Board of Seoul National University Hospital (IRB NO H-1212-087-450).

Baseline and Clinical Assessment
Baseline characteristics, including demographic data (age and sex) and vascular risk factors (hypertension, diabetes mellitus, hyperlipidemia, smoking history [current or past regular smoking], body mass index [BMI], and a past history of stroke/transient ischemic attack) were evaluated. Hypertension was defined as a history of antihypertensive treatment, a systolic blood pressure ≥140 mm Hg, or a diastolic blood pressure ≥90 mm Hg. Hyperlipidemia was defined as a history of lipid-lowering medication, a serum total cholesterol level of ≥240 mg/dL, or a serum level of low-density lipoprotein cholesterol ≥160 mg/dL. Diabetes mellitus was defined as a hemoglobin A1C level of ≥26.5%, fasting blood glucose level of ≥7.0 mmol/L, nonfasting blood glucose level of ≥11.1 mmol/L, or the use of insulin or oral hypoglycemic drugs. All of the patients were initially evaluated for stroke severity based on the National Institutes of Health Stroke Scale (NIHSS) neurological examination at SU admission. Ischemic strokes were categorized as large-artery atherosclerosis (LAA), small-vessel occlusion, cardioembolism, other determined, and undetermined based on the Trial of Org 10172 in Acute Stroke Treatment criteria. The ischemic lesion locations were divided into anterior circulation (anterior cerebral artery and middle cerebral artery territories), posterior circulation (posterior cerebral artery and vertebrobasilar artery territories), and multiple artery territory. In addition, severity of white matter disease on fluid-attenuated inversion recovery magnetic resonance imaging was assessed using the Fazekas scale.

SU Monitoring Data and NOD
Patients in the SU were continuously monitored with General Electric DASH 4000 monitors for cardiac rhythm, heart rate, and oxygen saturation (pulse oximetry), which was sampled every 2 s and averaged >1 minute, and blood pressure (noninvasive automatic measurement every 15 minutes). A high-resolution data acquisition system (BedmasterEX, Excel Medical Electronics) was used to acquire organized SU digital data.

NOD was evaluated based on ODI during the first night after admission to the SU. We used pulse oximetry saturation data from 10:00 PM to 7:00 AM. Artifacts, about false desaturation because of sensing errors, were deleted after manual review, which was blinded to clinical information. We calculated the ODI, which was defined as the number of desaturation events per hour of sleep. A desaturation event occurred when the saturation level fell to ≤3% below the baseline saturation level. Baseline saturation was defined as the mean saturation of the previous minute. The ODI was the total number of desaturation events divided by the number of hours (9 hours). The patients were divided into 2 groups according to their ODI values as follows: normal, ≤5 ODI events per hour and NOD, ≥5 ODI events per hour. We also checked the amount of time during overnight sleep in which capillary pulse oxygen saturation (SpO₂) was <90%. We compared the mean change in mean heart rate between the baseline (5 minutes before desaturation) and during nocturnal desaturation in patients with NOD.

Statistical Analysis
Continuous variables and proportions of categorical variables were compared using Student t tests, paired t test, Pearson χ² tests, Mann–Whitney nonparametric test, or Fisher exact test, as appropriate. The association between WUS and NOD was analyzed using logistic regression analyses. Covariates with statistically significant differences (P<0.05) on univariate analysis and those with clinically important factors were adjusted for multivariate analysis. For all analyses, a 2-tailed P value of <0.05 was considered statistically significant.

Statistical analyses were performed using the SPSS program (version 21.0, IBM Statistics).

Results
Characteristics of Patients With WUS
Among a total of 298 patients (male, 54.4%; age, 67.7±12.6 years), 26.5% (n=79) were admitted for WUS (Table 1). The baseline characteristics of the patients with WUS were not different when compared with those with non-WUS, except that hyperlipidemia was more common in the WUS group (P=0.041). In general, NOD was identified in 16.8% (50/298) of patients. NOD occurred more frequently in the WUS group compared with the non-WUS group (29.1% versus 12.3%, P=0.001). The ODI number and total time of extreme desaturation (SpO₂ <90%) were not different between the WUS and non-WUS groups.

Factors Associated With NOD
To detect possible factors associated with NOD, patients’ clinical data were compared between patients with and without NOD (Table 2). Compared with non-NOD group, those with NOD were older (72.5±10.3 versus 66.8±12.8, P=0.001) and more obese (BMI, 25.0±4.4 versus 23.0±3.2, P<0.001). Vascular risk factors, stroke severity, stroke subtypes, stroke lesion locations, and degree of white matter disease were not different between patients with and without NOD (Table 2). We compared the proportion of stroke lesion locations affecting respiratory centers (eg, posterior circulating lesions) or large-size infarctions with mass effect in patients with WUS and non-WUS to exclude secondary respiratory problems related to acute stroke. However, there was no significant difference between the 2 groups (P=0.372).

As expected, ODI was higher in patients with NOD compared with those with non-NOD (7.2±2.5 versus 1.9±1.4, P<0.001). Baseline systolic blood pressure was not different between patients with and without NOD (162.5±30.6 versus 156.6±30.0, P=0.21). However, the mean heart rate during night was significantly higher in the NOD group (74.4±14.5 versus 69.1±13.8, P=0.015). In addition, the NOD group patients showed an increase in heart rate by 10.2% during the NOD event (baseline 74.0±14.2 versus during desaturation 81.4±15.6, bpm/min, P<0.001, paired t test; Figure). We further investigated whether the existence of hypertension, age, and BMI modified the effect of NOD on WUS. However, no significant interaction was found between NOD and hypertension, age, and BMI (P=0.11, P=0.424, and P=0.290 for interaction effect, respectively).

Association Between NOD and WUS
Using logistic regression analysis, it was found that NOD was associated with a higher risk of WUS (odds ratio, 3.2; 95% confidence interval, 1.63–6.46; P=0.001) after adjusting for relevant confounding variables (Table 3).

Discussion
The main finding of our study is that nocturnal desaturation in the SU was more common in patients admitted with WUS.
Patients with WUS had a 3× higher odds of having a nocturnal desaturation episode. Those with nocturnal desaturation were older and more obese compared with those without nocturnal desaturation, suggesting that WUS is possibly linked to breathing problems while asleep, either directly or indirectly, mediated by common risk factors of obstructive sleep apnea.

The association between sleep-disordered breathing and stroke has been studied previously, and sleep-disordered breathing has been shown to be associated with cardiovascular diseases. The hypoxia and carbon dioxide retention cycle during sleep-disordered breathing may disturb autonomic nervous activity, which affects heart rate and cardiac function and increases arterial pressure.22–24 We found that the mean heart rate was higher in the NOD group, and heart rate was significantly increased during NOD events in our patients, which may be mediated by high sympathetic nerve activity because of hypoxia.21,25,26 However, analysis of blood pressure changes was not possible because BP measurement was done every 15 minutes. In addition, recurrent apneic spells may be associated with decreases in cerebral perfusion and increased platelet activation, leading to a hypercoagulable state. These mechanisms could possibly trigger stroke in patients with sleep-disordered breathing.9,23,27,28 As we described above, we could not find the effect of nocturnal desaturation on WUS in patients with and without hypertension, suggesting that NOD effect was not different in patients with and without a history of hypertension. In addition, the effect of age and BMI was not found on nocturnal desaturation with WUS.

A few previous studies have reported a correlation between sleep apnea and WUS using polysomnography or monitoring devices.6,8 Polysomnography is the gold-standard technique to diagnose sleep apnea and sleep-disordered breathing. However, analysis of blood pressure changes was not possible because BP measurement was done every 15 minutes. In addition, recurrent apneic spells may be associated with decreases in cerebral perfusion and increased platelet activation, leading to a hypercoagulable state. These mechanisms could possibly trigger stroke in patients with sleep-disordered breathing.9,23,27,28 As we described above, we could not find the effect of nocturnal desaturation on WUS in patients with and without hypertension, suggesting that NOD effect was not different in patients with and without a history of hypertension. In addition, the effect of age and BMI was not found on nocturnal desaturation with WUS.

A few previous studies have reported a correlation between sleep apnea and WUS using polysomnography or monitoring devices.6,8 Polysomnography is the gold-standard technique to diagnose sleep apnea and sleep-disordered breathing. However, it is not universally available and requires a sleep laboratory. In addition, polysomnography is often not feasible in acute stroke settings, and the diagnosis of sleep-disordered breathing is difficult during the acute period.7,13 Therefore, sleep-disordered breathing, diagnosed using polysomnography, has been studied in only highly selected patients among those with acute stroke, which has some limitation in external validity.
Our study analyzed organized high-resolution pulse oximetry data in the SU. Pulse oximetry is one of the most widely used, easy, and inexpensive tools to measure capillary oxygen saturation. It identifies intermittent hypoxia as the landmark consequence of sleep-disordered breathing. Therefore, monitoring NOD in SU is a simple and reliable tool for screening for sleep apnea in patients with acute ischemic stroke. NOD secondary to sleep-disordered breathing could be a modifiable risk factor for stroke and other cardiovascular diseases. Lifestyle modification, such as weight-loss intervention, may help improve sleep-disordered breathing symptom and may possibly lead to stroke prevention. In addition, long-term continuous positive airway pressure treatment, the representative treatment of sleep apnea, is considered to provide protection against stroke in patients with sleep-disordered breathing. Therefore, screening for NOD may suggest the causative clues in patients with WUS.

Table 2. Patient Profile According to NOD

<table>
<thead>
<tr>
<th></th>
<th>NOD (n=50)</th>
<th>Non-NOD (n=248)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean±SD), y</td>
<td>72.5±10.3</td>
<td>66.8±12.8</td>
<td>0.001</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>24 (48.0)</td>
<td>138 (55.6)</td>
<td>0.322</td>
</tr>
<tr>
<td>BMI (mean±SD), kg/m²</td>
<td>25.0±4.4</td>
<td>23.0±3.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>37 (74.0)</td>
<td>172 (69.4)</td>
<td>0.513</td>
</tr>
<tr>
<td>Diabetes mellitus, n (%)</td>
<td>13 (26.0)</td>
<td>61 (24.6)</td>
<td>0.834</td>
</tr>
<tr>
<td>Hyperlipidemia, n (%)</td>
<td>20 (40.0)</td>
<td>77 (31.0)</td>
<td>0.218</td>
</tr>
<tr>
<td>Smoking, n (%)</td>
<td>11 (22.0)</td>
<td>81 (34.7)</td>
<td>0.137</td>
</tr>
<tr>
<td>Previous stroke/TIA, n (%)</td>
<td>16 (32.0)</td>
<td>75 (30.2)</td>
<td>0.806</td>
</tr>
<tr>
<td>Atrial fibrillation, n (%)</td>
<td>15 (30.0)</td>
<td>52 (21.0)</td>
<td>0.163</td>
</tr>
<tr>
<td>Initial NIHSS, median (IQR)</td>
<td>4 (2–8.25)</td>
<td>3 (1–7)</td>
<td>0.564</td>
</tr>
<tr>
<td>Discharge NIHSS, median (IQR)</td>
<td>2 (1–6.5)</td>
<td>1 (0–4)</td>
<td>0.140</td>
</tr>
<tr>
<td>Stroke mechanism, n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAA</td>
<td>18 (36.0)</td>
<td>61 (24.6)</td>
<td>0.223</td>
</tr>
<tr>
<td>SVO</td>
<td>7 (14.0)</td>
<td>40 (16.1)</td>
<td></td>
</tr>
<tr>
<td>CE</td>
<td>14 (28.0)</td>
<td>75 (30.2)</td>
<td></td>
</tr>
<tr>
<td>Other determined</td>
<td>3 (6.0)</td>
<td>23 (9.3)</td>
<td></td>
</tr>
<tr>
<td>Undetermined</td>
<td>7 (14.0)</td>
<td>23 (9.3)</td>
<td></td>
</tr>
<tr>
<td>TIA</td>
<td>1 (2.0)</td>
<td>26 (10.5)</td>
<td></td>
</tr>
<tr>
<td>WMD Fazekas score, median (IQR)</td>
<td>1 (1–2)</td>
<td>1 (1–2)</td>
<td>0.188</td>
</tr>
<tr>
<td>Lesion location, n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior circulation</td>
<td>25 (51.0)</td>
<td>136 (61.3)</td>
<td>0.372</td>
</tr>
<tr>
<td>Posterior circulation</td>
<td>16 (32.7)</td>
<td>53 (23.9)</td>
<td></td>
</tr>
<tr>
<td>Multiple territory</td>
<td>8 (16.3)</td>
<td>33 (14.9)</td>
<td></td>
</tr>
<tr>
<td>ODI (mean±SD)</td>
<td>7.2±2.5</td>
<td>1.9±1.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SBP (mean±SD), mmHg</td>
<td>162.5±30.6</td>
<td>156.6±30.0</td>
<td>0.207</td>
</tr>
<tr>
<td>DBP (mean±SD), mmHg</td>
<td>87.2±16.9</td>
<td>85.4±15.2</td>
<td>0.438</td>
</tr>
<tr>
<td>Mean HR (mean±SD), bpm/min</td>
<td>74.4±14.5</td>
<td>69.1±13.8</td>
<td>0.015</td>
</tr>
</tbody>
</table>

BMI indicates body mass index; CE, cardioembolism; DBP, diastolic blood pressure; HR, heart rate; IQR, interquartile range; LAA, large artery atherosclerosis; NIHSS, National Institutes of Health Stroke Scale; NOD, nocturnal oxygen desaturation; ODI, oxygen desaturation index; SBP, systolic blood pressure; SVO, small vessel occlusion; TIA, transient ischemic attack; and WMD, white matter disease.
we estimate the length of sleep. These factors may underestimate the true prevalence of sleep-disordered breathing. NOD was observed in 16.8% in our study, which was lower than the reported prevalence of sleep apnea after stroke using polysomnography.\(^6\)\(^,\)\(^7\)\(^,\)\(^9\)\(^,\)\(^23\) In addition, we excluded patients treated with oxygen to minimize bias. This could have affected the lower rate of sleep-disordered breathing in our patients, despite the absolute numbers being small. Second, we calculated ODI using continuous pulse oximetry data. The data resolution of \(\text{SpO}_2\) is lower than polysomnography; \(\text{SpO}_2\) was measured in every 2 s and averaged >1 minute, which might have affected the lower sensitivity of sleep-disordered breathing in our patients. Moreover, pulse oximetry data could be influenced by poor peripheral perfusion, which could have influenced the lower prevalence of nocturnal desaturation in our study, although the probability is low. Third, we could not evaluate secondary medical conditions, such as central apnea, aspiration, or respiratory muscle weakness, which could induce unexpected nocturnal hypoxia in patients with acute stroke.\(^3\) However, the stroke lesion size and location, which possibly have an effect on respiration, were not different in patients with WUS and non-WUS. Fourth, no patient underwent sleep studies before stroke onset, and it is impossible to find a causal relationship between sleep-disordered breathing and WUS. In addition, we could not evaluate sleep apnea–related symptoms, such as habitual snoring, apneic episodes observed by the bed partner, or daytime sleepiness. Fifth, our study was performed in a retrospective design based on prospectively collected data from the SU. Patients with severe stroke (median NIHSS, 13) were excluded, partly because they were admitted directly to the neurointensive care unit if they had airway issues or partly because they were treated with oxygen therapy from the beginning. Therefore, patients with minor strokes were more likely to be included in our study. Taken together, some sort of selection bias was inevitable. Despite these limitations, our study has more external validity in patients with acute ischemic stroke because we evaluated NOD using universally monitored \(\text{SpO}_2\) data in all patients admitted to the SU without a need of additional monitoring devices during the first night after admission.

In conclusion, NOD, measured in the SU is associated with WUS. Therefore, prospective studies may be required to find the causal relationship between screening and diagnosing sleep apnea and WUS in the general population.

### Table 3. Association Between NOD and Wake-Up Stroke

<table>
<thead>
<tr>
<th></th>
<th>Unadjusted OR (95% CI)</th>
<th>(P) Value</th>
<th>Adjusted OR (95% CI)</th>
<th>(P) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>NOD</td>
<td>2.92 (1.55–5.49)</td>
<td>0.001</td>
<td>3.25 (1.63–6.46)</td>
<td>0.001</td>
</tr>
<tr>
<td>Age</td>
<td>1.00 (0.98–1.02)</td>
<td>0.934</td>
<td>0.99 (0.97–1.01)</td>
<td>0.318</td>
</tr>
<tr>
<td>BMI</td>
<td>0.98 (0.91–1.06)</td>
<td>0.562</td>
<td>0.94 (0.87–1.02)</td>
<td>0.158</td>
</tr>
<tr>
<td>HL</td>
<td>1.78 (1.04–3.03)</td>
<td>0.035</td>
<td>1.74 (1.00–3.01)</td>
<td>0.049</td>
</tr>
</tbody>
</table>

Adjusting for NOD, age, BMI, and history of HL. BMI indicates body mass index; HL, hyperlipidemia; and NOD, nocturnal oxygen desaturation.
References


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Stroke Unitにおける夜間の酸素飽和度低下と起床時の虚血性脳卒中発症の関連性

Nocturnal Desaturation in the Stroke Unit Is Associated With Wake-Up Ischemic Stroke

Tae Jung Kim, MD; Sang-Bae Ko, MD, PhD; Han-Gil Jeong, MD, et al.
Department of Neurology, Seoul National University Hospital, Seoul, Republic of Korea

背景および目的：起床時発症脳卒中（wake-up stroke: WUS）は，全虚血性脳卒中の1/4を占める特定のサブグループである。睡眠呼吸障害による夜間の酸素飽和度低下は脳卒中の独立危険因子であるが，夜間の酸素飽和度低下とWUSの関連はいまだに不明である。本研究では，Stroke Unitに入院した急性脳卒中患者における酸素飽和度低下指数による夜間の酸素飽和度低下とWUSの関連を調査した。

方法：2013年7月～2015年5月に急性虚血性脳卒中でStroke Unitに入院した患者合計298例を対象とした。Stroke Unitに入院後，最初の夜の9時間（PM 10:00～AM 7:00）に1分間隔で記録したパルスオキシメーターデータから酸素飽和度低下指数を算出し，1時間の酸素飽和度低下指数が5以上の場合は夜間の酸素飽和度低下と定義した。WUS患者と非WUS患者の臨床的特徴と夜間の酸素飽和度低下を比較した。

結果：全患者（年齢67.7±12.6歳，男性54.4%）のうち26.5%がWUSであった。WUSで入院した患者は夜間の酸素飽和度低下が生じる率が有意に高かった（29.1% vs. 12.3%，P = 0.001）。高脂血症，脳卒中の重症度，脳卒中の発症機序を除く2群の年齢，性別，危険因子はほぼ同様であった。共変量の調整後，夜間の酸素飽和度低下はWUS群で有意に多いことがわかった[オッズ比3.25，95%信頼区間1.63～6.46]。

結論：WUSでStroke Unitに入院した患者では，最初の夜に夜間の酸素飽和度低下が低下する率が高かった。このことから，夜間の酸素飽和度低下は修正可能なWUSの危険因子である可能性が考えられる。