Delta Activity at Sleep Onset and Cognitive Performance in Community-Dwelling Older Adults

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Study Objectives: Frontal intermittent rhythmic delta activity (FIRDA) has long been considered to be an abnormal variant in the electroencephalogram (EEG) among older adults. Prior work also indicates a predominance of slow wave EEG activity among patients with dementia. However, instability of state control occurring with aging generally and among many neurodegenerative diseases raises the possibility that FIRDA might represent the intrusion of sleep related elements of the EEG into the waking state. We examined delta activity at sleep onset (DASO) in community-dwelling, older adults without dementia, and examined whether this activity is related to poorer cognitive performance.

Methods: 153 community-dwelling, older adults without dementia underwent overnight polysomnography and measures of global cognition, delayed verbal memory, information processing speed, attention, inhibition, verbal naming, and visuospatial ability. Delta activity during sleep/wake transitions (scored either as Waking or N1) was analyzed visually.

Results: Participants were 83 women and 70 men, mean age 71.3 ± 0.6 y. DASO was present in 30 participants (19.6%). Age, years of education, sex, and body mass index did not differ between DASO (+) and (−) groups. Multiple regression analyses indicated faster reading of the Stroop color words in DASO (+) subjects (P = 0.007). None of the other cognitive domains differed between the two groups.

Conclusions: DASO was relatively common in our sample of community-dwelling, older adults without dementia. DASO was not associated with poorer performance on any cognitive domain. Instead, individuals with DASO demonstrated better performance on a simple reading task. Although these findings suggest that an abnormal EEG activity may represent normal variation, our study underscores the importance of distinguishing DASO from FIRDA when examining sleep in older adults.

Commentary: A commentary on this article appears in this issue on page 725.

Keywords: cognition, DASO, delta activity at sleep onset, FIRDA, sleep


INTRODUCTION

It is well known that overall increased slow wave electroencephalogram (EEG) activity is associated with dementia, especially dementia with Lewy bodies.1–4 Frontal intermittent rhythmic delta activity (FIRDA) also has long been considered to be an abnormal variant in the EEGs of older adults, with prior work indicating a predominance of FIRDA among patients with dementia.1,5,6 One complication in the literature is that in the transition to sleep in older adults, slow wave EEG activity may in fact reflect the intrusion of sleep related elements of the EEG into the waking state. As such, this type of slow wave EEG activity may reflect more the instability of wake sleep control that occurs with typical aging than a pathological process per se.7,8

FIRDA during sleep onset, which has also been called anterior bradyrhythmia, is characterized by sequences of rhythmic, bilateral anterior slow activity in the delta range (mostly 1.5–2.5/sec), with duration varying between 2 and 10 sec, and voltage output that may be considerable.9,10 Previous reports consistently show a higher prevalence of this type of delta activity at sleep onset (DASO) in older adults.6,9,10 Some investigators, e.g., Zurek et al.6 and Shiohama et al.5, have found DASO to be associated with a higher prevalence of altered mental status based on accumulated EEG data. Katz and Horowitz,9 however, found DASO in 16% of healthy older adults, suggesting it may represent a normal variant in this age range. The difficulty is that the majority of these studies reported on EEGs, which were not performed nocturnally, but which captured sleep onset in the context of daytime napping. Daytime napping may vary substantially from patient to patient, not occurring at all in some, and this may account for the mixed findings observed. Further, these studies did not investigate the relationship of DASO to more in-depth cognitive testing. As many as 20% of healthy, community-dwelling older adults have cognitive impairments indicative of mild cognitive impairment (MCI),11–13 placing them at increased risk for the development of dementia. In the absence of more in-depth cognitive testing, it is unclear if the 16% presenting with DASO in the Katz and Horowitz investigation are individuals with poorer cognitive functioning, who are at risk for the development of dementia. Here we propose to address this issue by examining whether DASO that is captured by nocturnal polysomnography (PSG) in healthy, community-dwelling older adults is associated with impairments in a range of cognitive domains. To this end, we examined the relationship of DASO, captured in nocturnal PSG, to cognitive performance in 153 community-dwelling, older adults without dementia.
METHODS
Participants were 153 community-dwelling older adults enrolled in an investigation of sleep and cognitive function. They were recruited through advertisements and local senior centers from 2005 to 2010. All participants provided informed consent in accordance with Stanford University Institutional Review Board regulations. An initial evaluation included demographics, self-reported current and past medical status, the Mini-Mental State Examination (MMSE) to screen for dementia, and Structured Clinical Interview for DSM-IV-TR (SCID-IV-TR) to screen for Axis I psychiatric disorders. Inclusion criteria were the following: being 50 y of age or older, the ability to give informed consent and sufficient visual and auditory acuity for the cognitive testing. Individuals were excluded if they had a MMSE < 26, a diagnosis of possible or probable dementia or a profile on the cognitive battery indicative of dementia, any serious medical illness or any Axis I disorders currently, or within the past 2 y on the SCID-IV-TR. Participants were also excluded if they were currently using psychotropic medication, short-acting anxiolytics, sedative hypnotic agents or medications with significant cholinergic or anticholinergic side effects, or any US FDA-approved medications for dementia. The following measures were administered.

Cognitive Battery
Participants completed an extensive cognitive battery, with measures sensitive to milder cognitive impairments in older adults.14 For analytic purposes, to ensure the measures are independent and reduce those significantly correlated with each other, we conducted a principal component analysis (PCA), which identified four independent cognitive domains and associated assessments. We performed data reduction in cognitive domains utilizing PCA in the manner similar to that employed in previous reports.15,16 The cognitive domains identified were (1) delayed verbal recall (assessed by the Rey Auditory Verbal Learning Test (RAVLT)),17 (2) information processing speed and attention (assessed by the Stroop Color and Word Test (SCW)),18 (3) verbal naming (assessed by the Boston Naming Test (BNT))9 and (4) visuospatial ability (Judgment of Line Orientation).20 Prior work from our and other groups has consistently found lower performance on measures of delayed recall to be associated with increased age. Deficits in the other three domains are common in older adults and are associated with an increased risk of cognitive decline and onset of dementia. One outcome measure from each domain was selected. For the RAVLT, we chose the delayed recall component, that is, RAVLT7. For SCW we employed the time taken to complete the word, color, and color/word component. Total number answered correctly served as the outcome measure for the BNT, and for the Judgment of Line Orientation. The MMSE was included as a brief mental status examination to quantify global cognitive functioning. The selected measures are widely used and have proven reliability.

PSG and Sleep Related Questionnaires
Unattended, overnight PSG (Safiro Ambulatory PSG System; Compumedics, Charlotte, NC) was performed in all participants. Participants went to bed and arose as was their normal schedule. The PSG was performed as close as possible to the time they indicated that they typically went to bed. However, in all cases there was a period of wake recording both before the participant went to bed and after they got up. The standard recording montage included scalp electroencephalography electrodes (C3, C4, O1, O2, M1, M2) applied at positions according to the International 10–20 System of Electrode Placement), chin and bilateral anterior tibialis electromyography, electrooculography, electrocardiography, nasal pressure transducer and oral airflow (thermistor), abdominal and thoracic excursion (piezoelectric band), finger pulse oximetry, snoring sounds (microphone), and body position. All data were scored for sleep staging and respiratory events by a registered PSG technologist and were reviewed by a diplomat of the American Board of Sleep Medicine. An apnea is defined as a “cessation or near complete cessation (> 90% reduction) of airflow for a minimum of 10 sec,” a hypopnea is “a greater than 30% reduction of amplitude in airflow as compared to baseline with a greater 3% oxygen desaturation” and apnea-hypopnea index represents “the number of apneas and hypopneas divided by the number of hours of sleep.” Sleep related questionnaires, including Epworth Sleepiness Scale (ESS),21 Pittsburgh Sleep Quality Index (PSQI),22 Functional Outcomes of Sleep Questionnaire (FOSQ),23 and Morningness-Eveningness Questionnaire (MEQ),24 were administered.

A board-certified clinical neurophysiologist (MK) performed additional visual analysis of EEG parameters to identify DASO. The scoring of DASO was blind to any clinical parameters. DASO was defined as follows.

1. Sequences of rhythmic, bilateral anterior slow activity in the delta range (1–2.5 Hz)
2. Duration between 2 and 10 sec with amplitude more than 50 μV.
3. Occur within otherwise normal background activity in the transition period from awake to sleep
4. High-amplitude delta range activity in slow wave sleep is excluded.
5. Sleep transitions were defined as 30-sec epochs scored either as: (a) stage N1, (b) stage W intermixed with a short period of stage N1 sleep, the latter characterized by slow eye movement, disappearance of posterior dominant rhythm, vertex sharp wave transients or EEG activity in the range of 4–7 Hz with slowing of background frequencies by ≥ 1 Hz from those of stage W, or (c) stage W adjacent to epochs of any sleep stages.

Even though DASO can occur multiple times during overnight PSG, data were categorized initially as binary parameter of “present” [DASO (+)] if DASO occurred at least once and “absent” [DASO (−)] if no DASO was found in the overnight PSG record. Then continuous parameter of DASO, i.e., number of occurrences over night and mean duration of DASO, were obtained and analyzed.

Statistical Analysis
For our primary analyses, we employed multiple regression to examine the association of DASO to cognitive function. DASO, age, sex, year of education, and body mass index (BMI) served as independent variables with performance on the four cognitive measures serving as the dependent measures. For our measure of DASO, we first utilized a binary parameter (present
versus absent) for our analyses following the approach of other investigations of DASO.

For purposes of data reduction we employed PCA to identify our main cognitive measures that were independent of each other. Because none of the outcome measures were significantly correlated using PCA, we employed these as independent tests for the purposes of analyses, representing different cognitive functions well known to be affected in older adults. Thus, we conducted separate regression analyses for the four cognitive outcomes. For multiple regression analysis, as interactive effects were included, all independent variables were centered at the median to ensure interpretability of the regression coefficients. In additional analyses, we also employed continuous parameters of DASO, specifically the number of occurrences and the mean duration.

RESULTS
Participants were 83 women and 70 men of the age ranging from 52 to 90 y, with mean of 71.3 ± (standard error, SE) 0.6 y. Mean and SE of year of education were 16.5 and 0.2 y. DASO was present in 30 participants (19.6%). Mean and SE of number of occurrences over night and mean duration of DASO in these 30 participants were 4.1 ± 0.6 (times/night) and 3.8 ± 0.2 (sec). Typical DASO is shown as Figure 1.

Age, year of education, sex, and BMI were not different in the DASO (+) and DASO (−) groups by Mann-Whitney U test (see Table 1).

Table 1—Demographic characteristics of participants.

<table>
<thead>
<tr>
<th></th>
<th>DASO (+) n=30</th>
<th>DASO (−) n=123</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>71.4 ± 1.4</td>
<td>71.3 ± 0.7</td>
<td>0.74</td>
</tr>
<tr>
<td>Education (y)</td>
<td>16.3 ± 0.4</td>
<td>16.6 ± 0.3</td>
<td>0.61</td>
</tr>
<tr>
<td>Sex (Female:Male)</td>
<td>20:10</td>
<td>63:60</td>
<td>0.13</td>
</tr>
<tr>
<td>BMI</td>
<td>26.3 ± 0.7</td>
<td>27.1 ± 0.5</td>
<td>0.67</td>
</tr>
</tbody>
</table>

Comparison of demographic between DASO (+) and (−) group with Mann-Whitney U test. BMI, body mass index; DASO, delta activity at sleep onset.

Multiple regression analyses indicated that the reading condition of the Stroop performance was significantly faster (better in the DASO (+) group (48.1 ± 1.2 versus 53.3 ± 1.1 sec, P = 0.007; covaried with age, sex, year of education, and BMI). There was also a trend association for better global cognition in the DASO (+) group compared with the DASO (−) group (MMSE total scores = 29.3 ± 0.2 versus 28.8 ± 0.1, P < 0.058). None of the other cognitive measures in the DASO (+) group were significantly different than DASO (−) (Table 2). The multiple regression analysis for the reading condition of Stroop test showed DASO and male sex to be related to faster performance, whereas age were related to slower performance (Table 3). Age and sex distribution did not differ in the DASO (+) and (−) groups.
Among the DASO (+) group, multiple regression covaried for age, sex, years of education, and BMI indicated a relationship between our continuous measures of DASO, i.e., number of occurrences of DASO, and Stroop color word ($P < 0.048$, beta $= -0.36$) (Table 4). However, this is not statistically significant following Bonferroni correction. None of other cognitive measures showed an association with either number of occurrences or mean duration of DASO. In the DASO (+) group, DASO occurred in the first sleep onset in all 30 DASO (+) subjects.

Subgroup analysis was performed to investigate the prevalence of DASO in the population with cognitive function indicative of mild cognitive impairment (MCI). Fifty participants (33.6%) had cognitive performance indicative of MCI, using...
standard definitions.26 23.3% of DASO (+) group had MCI, compared to 36.1% of DASO (−) group had MCI. Chi-square analysis indicated no significant difference in the prevalence of MCI in the DASO (+), compared to DASO (−) groups (Chi-square = 1.76; df = 1; P < 0.18).

We also examined the PSG parameters (see Table 5), and Mann-Whitney U tests indicated shorter wake after sleep onset (WASO) (95.1 ± 11.5 versus 125.9 ± 7.1 min, P = 0.034), higher spontaneous arousal index (number of spontaneous arousal events divided by total sleep time) (2.3 ± 0.6 versus 1.5 ± 0.3 events/h, P = 0.03), and higher sleep efficiency (75.6 ± 2.2 versus 68.4 ± 1.4%, P = 0.02) in DASO (+) group compared with DASO (−) group. However, when we conducted multiple regressions that covaried for age, sex, and BMI, none of these parameters was statistically significantly associated with the presence of DASO. All sleep related questionnaires, including ESS, PSQI, FOSQ, and MEQ, did not show a significant difference between the groups (See Table 5).

**DISCUSSION**

A significant percentage of participants in our study had DASO (19.6%), similar to the Katz and Horowitz finding in their daytime EEG study of community-dwelling older adults (16%), suggesting DASO is relatively common in healthy older adults.9 Our study is the first to examine DASO based on nocturnal PSG, in which sleep is recorded in all participants, which may contribute to the greater prevalence than has been observed in previous studies of hospital EEGs.5,6 Older age may be one important factor contributing significantly to the prevalence of DASO,5,6,9 though our study did not find a statistically significant difference in age itself between DASO (+) versus DASO (−) group. Further, our participants were already selected based on age (older than 50 y) such that any effect of age and its association with DASO was possibly minimized.

With respect to our primary hypothesis, we found no evidence of worse cognitive performance being associated with DASO. Indeed, the reading condition of the Stroop Color Word test, a measure of simple processing speed, was faster in the DASO (+) group with statistical significance even after Bonferroni correction. Further, compared with DASO (−), the DASO (+) group demonstrated a trend toward better overall global cognitive function on the MMSE (P = 0.058). The inclusion of age and education in our model did not account for the observed differences in cognitive function between the DASO (+) and the DASO (−) groups. Although a positive association was observed with the presence of DASO and better performance on Stroop reading ability, we did not find any association with other, more complex and less

| Table 5—Polysomnographic parameters and sleep related questionnaires. |
|-----------------|-----------------|-----------------|-----------------|
|                 | DASO (+)        | DASO (−)        | P value by      |
|                 |                 |                 | Mann-Whitney U  |
|                 |                 |                 | test            |
|                 |                 |                 | P value by      |
|                 |                 |                 | multiple        |
|                 |                 |                 | regression      |
| AHI (events/h)  | 32.2 ± 3.6      | 32 ± 2          | 0.76            |
| AI (events/h)   | 23 ± 3.8        | 22 ± 1.8        | 0.83            |
| HI (events/h)   | 9.2 ± 1.4       | 9.9 ± 1.1       | 0.63            |
| Average SpO2 (%)| 94 ± 0.3        | 93.4 ± 0.8      | 0.70            |
| Minimum SpO2 (%)| 80.9 ± 3        | 80.6 ± 1.6      | 0.86            |
| PLMI (events/h) | 4.3 ± 1.6       | 11.1 ± 1.6      | 0.20            |
| Sleep latency (min)| 35.2 ± 8.7 | 31.6 ± 3.3    | 0.91            |
| TST (min)       | 352.6 ± 12.9    | 343.1 ± 7.4     | 0.59            |
| WASO (min)      | 95.1 ± 11.5     | 125.9 ± 7.1     | 0.03            |
| REM (%)         | 12.9 ± 1        | 13.5 ± 0.6      | 0.40            |
| Stage 1 (%)     | 15.3 ± 1.5      | 17.4 ± 1        | 0.42            |
| Stage 2 (%)     | 69.7 ± 1.7      | 68.1 ± 1        | 0.50            |
| Stage 3 (%)     | 1.3 ± 0.5       | 0.9 ± 0.2       | 0.41            |
| Stage 4 (%)     | 0.9 ± 0.5       | 0.2 ± 0.1       | 0.10            |
| Arousal index (events/h)| 2.4 ± 0.7 | 1.8 ± 0.3    | 0.10            |
| Spontaneous arousal index (events/h) | 2.3 ± 0.6 | 1.5 ± 0.3 | 0.03 |
| Sleep efficiency (%)| 75.6 ± 2.2 | 68.4 ± 1.4 | 0.02 |
| ESS             | 6.8 ± 0.7       | 7.6 ± 0.4       | 0.41            |
| PSQI            | 6.8 ± 0.7       | 7.1 ± 0.4       | 0.84            |
| FOSQ            | 18.8 ± 0.9      | 17.8 ± 0.2      | 0.25            |
| MEQ             | 58.5 ± 2        | 58.3 ± 0.8      | 0.84            |

Comparison of polysomnography (PSG) parameters and sleep related questionnaires between DASO (+) and (−) groups. Numbers in left two columns represent mean and standard error of PSG parameters and scores in sleep related questionnaires. Multiple regression analyses were performed with DASO, age, sex, and body mass index as independent variables. *P < 0.05. #Not statistically significant after Bonferroni correction. DASO, delta activity at sleep onset; sleep onset frontal intermittent rhythmic activity AHI, apnea-hypopnea index; ESS, Epworth Sleepiness Scale; PSQI, Pittsburgh Sleep Quality Index; REM, rapid eye movement sleep; SpO2, oxygen saturation measured by pulse oximetry; TST, total sleep time; WASO, wake after sleep onset.
automatic cognitive abilities, including the Stroop Color Word Condition, which assesses speed of processing and inhibition of more complex information. It may be that DASO is associated with increased efficiency for particular overlearned tasks. No other significant differences for any of the cognitive measures were observed between the two groups.

Using our continuous measure of DASO, we found no significant association with any of our cognitive measures, including the reading condition of the Stroop test. However, it is important to note that DASO (−) subjects were excluded from the analysis for occurrences or duration of DASO. As such, we may not have had sufficient variabilty or statistical power to assess the effect of the number of occurrences and duration on cognitive function.

Because our sample ruled out for dementia, this also raised the possibility that we did not have sufficient levels of cognitive impairment to capture any association between DASO and cognition. However, our sample did have a broad range of cognitive function, with over 30% of participants having memory performance indicative of MCI. The distribution of those with MCI did not differ in the DASO (+) versus DASO (−) group.

There are other potential explanations for better cognitive performance on the reading condition of the Stroop in the DASO (+) group. Our additional analyses suggest that the DASO (+) group may have less dysregulated sleep. As such, more consolidated sleep in the DASO (+) group may result in better cognitive performance, particularly given the well-documented association between better sleep efficiency and better performance on measures of speed of processing and vigilance.

It is less clear, however, why DASO would be associated with better sleep.

At the least our findings do not support any negative effect of DASO on cognitive function in community-dwelling, older adults without dementia and our findings overall could be taken to support the view that the presence of DASO in healthy elderly may indeed represent normal variation. However, any definitive conclusion that DASO is a benign finding representing normal variation would require a longitudinal investigation of the relationship of DASO to cognitive impairment and decline. Further, although slow wave EEG activity in the context of sleep onset may reflect more the instability of wake sleep control that occurs with typical aging than a pathological process per se, it is crucial to note that slow wave EEG activity, not associated with sleep onset (such as FIRDA), do appear to indicate significant pathological findings in the older adults. For example, Watemberg et al. found intrahemispheric lesions or lacunar infarction in basal ganglia in the patients with FIRDA. Stam and Pritchard further suggested FIRDA in encephalopathy reflects a disturbance in the balance between excitation and inhibition of the neuronal populations in the cortical and subcortical region.

However, in their investigation of normal older adults, Katz and Horowitz did not find either focal or diffuse slow wave activity in any of their participants consistently. They concluded that focal or diffuse slow wave activity should be considered abnormal. Yet, utilizing the same database, Katz and Horowitz reported in another investigation that delta activity considered during sleep onset occurred in 16% of normal older adults without focal slow wave activity in the baseline wake recording. In line with the findings of our own study, they suggested that this activity thus represents a normal phenomenon. Yet, Shiohama et al. found that 80% of the patients with slow wave EEG activity at sleep onset during the day (likely napping) have dementia, and Zurek et al. reported that cerebrovascular disorder represented the most frequent etiology for slow wave EEG activity at sleep onset during the day.

It may be that the distinction between FIRDA and DASO is more difficult during daytime studies rather than when captured at the onset of nocturnal sleep. Overall, the field may benefit from increased clarification and distinction between DASO on the one hand and pathological slow wave EEG activity (such as p) on the other hand. For DASO, the slow wave EEG activity should only occur in the period of sleep onset and the wake EEG background activity should be normal. The mixed findings in the consideration of the clinical import of slow wave EEG activity in older adults to date may reflect a confabulation of DASO with FIRDA. We suggest that delta activity seen at sleep onset in healthy older adults with otherwise normal EEG background activity should not involve the term “FIRDA” to avoid confusion.

Our study has several limitations, including the cross-sectional design, lack of baseline full-montage EEG, only a single observer responsible for determining absence or presence of DASO, and lack of frontal EEG electrodes due to utilizing traditional PSG montage before the issue of the American Academy of Sleep Medicine manual. The cross-sectional design limits the extent to which we can identify developmental or mediational relationships of DASO in longitudinal change of cognition decline in older adults.

For detecting DASO, our study depended on visual analysis of EEG parameter and did not quantify by spectral analysis, which may improve the accuracy of our measure of DASO. However, because DASO is a paroxysmal finding on EEG and usually occurs in a very limited time of transition phase from wake to sleep state, we believe the classification by presence or absence, number of occurrences, and mean duration of DASO is appropriate for examination of its relationship to cognitive function.

We performed visual analyses of PSG even in the wake period before lights out and did not find abnormality in any participants. However, without baseline full montage EEG, there is a possibility that we missed mild EEG abnormality during the wake period, especially focal slow wave activity in frontal or temporal regions.

In the DASO (+) group, DASO occurred in the first sleep onset in all 30 DASO (+) subjects. In addition, as we showed in Table 3, the statistical significance of spontaneous arousals in association with presence and absence of DASO disappears in the multiple regression when we covaried for age, sex, years of education, and BMI. This led us to conclude that fragmentation of sleep is not a major contributor to the presence and absence of DASO.

The lack of frontal EEG electrodes may decrease the sensitivity of detecting DASO, although we note that the activities we identified as DASO in this study are similar to the activity previously defined as sleep onset FIRDA or anterior...
bradyrhythmia in terms of the amplitude, frequency, rhythmic character, timing of occurrence in the sleep onset, and duration.\textsuperscript{5,6,9,33} We believe the influence from the lack of frontal electrodes did not limit our ability to detect DASO, because, as it is shown in Figure 1, the amplitude is usually high enough to present in the central electrodes, and the prevalence in our study is equivalent to (even higher than) the one in previous study performed with standard EEG recording.\textsuperscript{8}

Despite these limitations, this is by far the largest study to date to examine the relationship of DASO to cognitive function among community-dwelling, healthy older adults in the United States. Moreover, this study benefits from a comprehensive assessment of neurocognitive function that objectively establishes the absence of dementia as well as full objective neuropsychological test battery and polysomnography. We believe that describing the lack of effect of DASO on cognitive function in healthy older adults could help clinicians avoid misinterpretation of DASO in EEG or PSG and thus provide better management of sleep and cognition for older adults.

In conclusion, unlike FIRDA arising from slow EEG background representing either encephalopathy or structural lesion, the lack of association of DASO with cognitive dysfunction or worsening of PSG parameters suggests its presence in healthy elderly may represent normal variation. Nevertheless, further longitudinal follow-up is necessary to examine whether or not DASO in community-dwelling older adults may presage brain impairments that are sufficient to negatively affect cognitive performance.

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