The link between sleep-disordered breathing and cognition in the elderly
New opportunities?

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Sleep disruption and cognitive impairments are not uncommon in the aging adult (affecting up to 50%) and there has been growing information to suggest a link between these conditions, which may lead to new opportunities in clinical care. In this issue of Neurology®, Haba-Rubio et al.1 highlight the association between sleep-disordered breathing (SDB) and cognitive impairment in the elderly. Such findings are consistent with recent studies.2–4 Identification of this relationship must be considered in the context of efforts to identify potentially treatable and modifiable factors that may contribute to the development of cognitive impairment in the older adult. Does this study suggest that we should become more aggressive in the management of SDB in elderly? Furthermore, should we be more thorough in our assessment of cognitive impairments in the elderly with SDB?

The association between sleep disruption and cognitive impairment can be viewed from several directions. Certainly, there is the immediate effect of sleep deprivation to reduce attention, executive function, and possibly memory. These changes may aggravate an underlying cognitive impairment. Sleep disruption, including SDB, may also be a factor in the development of other conditions. There has been growing interest, for instance, in the sleep-dependent clearance of toxic elements, such as β-amyloid.5 It is tempting, therefore, to consider that sleep disruption may contribute directly to the development of neurodegenerative disorders. On the other hand, causality may also be in the other direction. The sleep disorders, long considered to be associated with Alzheimer disease, may be related to the disruption of sleep and circadian rhythms by β-amyloid deposition.6–8 Haba-Rubio et al.,1 however, have placed a slightly different emphasis on this story. They present their findings from the large, well-characterized, population-based HypnoLaus Study. This study examined the results of polysomnographic recordings in a large sample of participants >65 years from the general population, with and without evidence of cognitive impairment. In their initial analysis, they found that the group with cognitive impairment had more time spent in N1 and less time in stage N3 or REM sleep, with lower sleep efficiency and more wake time after sleep onset. On further analysis, however, they noted that most of this could be accounted for by the presence of SDB. Although SDB can be associated with sleep disruption and hypoxemia, their data suggest that it is the hypoxemia and, in particular, the intermittent hypoxemia that accounts for most of the effect. These findings are consistent with the prospective substudy of the Study of Osteoporotic Fractures that demonstrated that prevalent SDB increased the risk of developing cognitive impairment.9 It should be noted that the Atherosclerosis Risk in Communities study, a community-based study, was not able to demonstrate a link between SDB and cognitive decline: they used a slightly younger population and did not specifically look at measures of intermittent hypoxemia.

It is important to remember that SDB may not only be associated with cognitive impairments, but also daytime sleepiness and an increase in cardiovascular risk factors. It is true that treatment with continuous positive airway pressure (CPAP) may improve daytime sleepiness and insulin resistance,10 with a modest reduction of blood pressure,11 but it may not be effective in the secondary prevention of cardiovascular events.12 One may not want to jump to the conclusion that an aggressive approach to the treatment of SDB in the elderly will or will not delay or prevent the development of cognitive impairment or dementia.13 However, there is reason to suspect that treatment with CPAP may be effective for immediate improvement of cognitive impairment and perhaps to delay the progression to dementia.

So where does this leave the clinician? On the one hand, there is growing evidence that SDB may be a treatable factor involved in the development of cognitive impairment in the elderly. There is now reason to believe that the link is more than the apparent relationship to sleep disruption; the intermittent hypoxemia of SDB may be the culprit. The available studies do not indicate whether aggressive treatment of SDB would delay or prevent the eventual development of cognitive impairment...
impairment and, perhaps, dementia in the elderly. Furthermore, we do not have a clear idea of the consequences related to the duration of untreated SDB. Would one need to start treating SDB in middle age to improve cognition in the elderly population?

Perhaps this is a study that leads to more questions. Indeed, there are some limitations to this study, as acknowledged by the authors. It is a cross-sectional study, which limits the ability to establish conclusions about causality. Only about a third of the available population had both the polysomnography and cognitive testing, and there was no pathologic confirmation of an underlying neurodegenerative disorder. Nevertheless, given the context of available research, it should heighten awareness of the clinician dealing with this population.

At the very least, the argument can be made for the clinician to look both ways. In other words, an evaluation of cognitive impairment and, possibly, early dementia in the elderly should include a screen for SDB. Similarly, an evaluation for SDB in the elderly should include a screen for cognitive impairment. Even if treating SDB does not prove to delay the onset of cognitive impairment, the association should be of concern. Otherwise, this may be a new opportunity for the clinician to improve the quality of life for the elderly patient.

STUDY FUNDING
No targeted funding reported.

DISCLOSURE
The authors report no disclosures relevant to the manuscript. Go to Neurology.org for full disclosures.

REFERENCES