

The impact of altered sleep on memory consolidation in Parkinson's disease

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Introduction

Sleep disturbances are a common non-motor symptom of Parkinson's disease but their direct impact on cognition is not known [1].

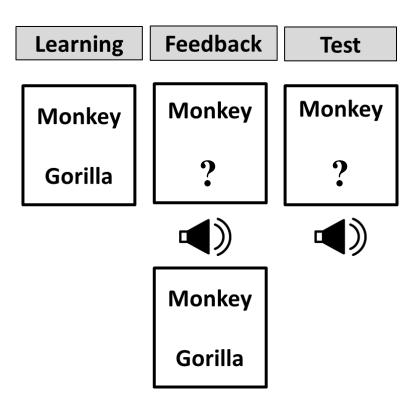
Alterations in sleep architecture [2], in particular, non-REM sleep, which is crucial to memory consolidation [3-5], are common in Parkinson's disease.

Obstructive sleep apnea (OSA), which causes sleep fragmentation and overall reduced sleep quality, is also very common in Parkinson's disease [6].

- Are alterations in non-REM sleep associated with impaired memory consolidation in Parkinson's disease?
- Is obstructive sleep apnea associated with memory consolidation in Parkinson's disease patients?

To address these questions we recorded overnight sleep and tested memory before and after sleep.

Experimental Task

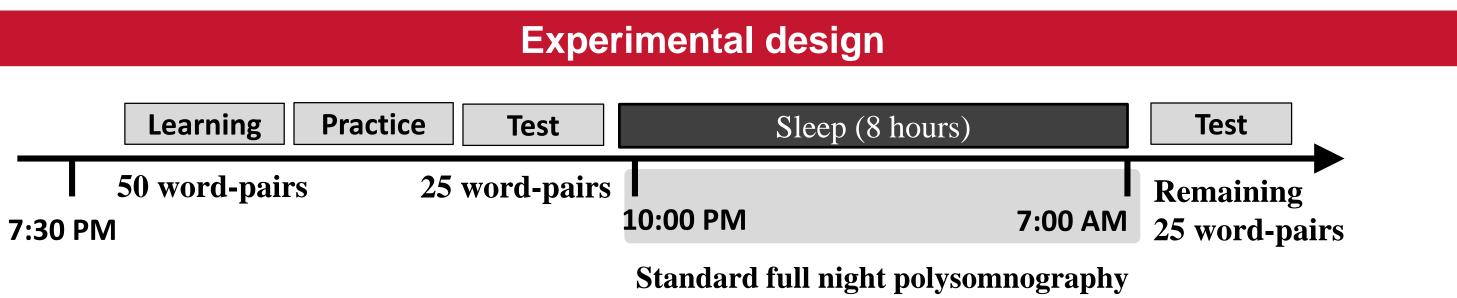


The task consisted of 3 phases:

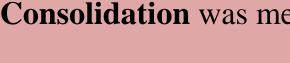
Learning: 50 word-pairs appeared one-by-one on the screen and patients were told to remember them.

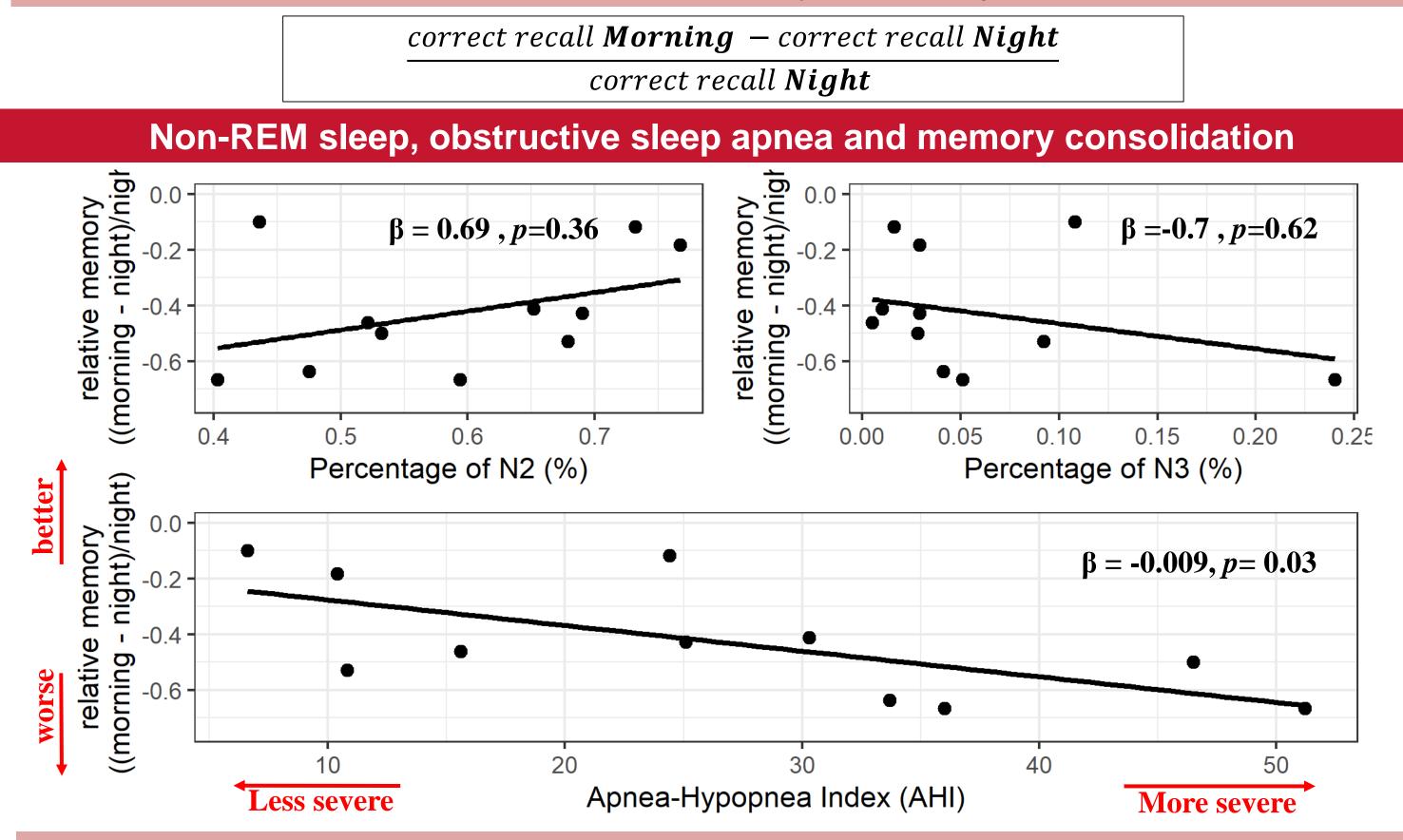
Practice: Only one word from a word-pair was shown, patients had 3.5 seconds to recall the second word before the tone. After the tone, the second word appeared on the screen.

<u>Test:</u> Only one word from a word-pair was shown, and patients had to correctly recall the second word before the tone. Half the word pairs (25) were randomly chosen to be tested at Night, and the remaining half were tested in the **Morning**.



Parkinson's disease patients without REM-sleep behavioral disorder (N=11), as assessed by the single-question RBD questionnaire [7], were tested on their memory and their sleep was recorded overnight using polysomnography. Signals included 6 EEG channels (F3, F4, C3, C4, O1, O2), EOG, ECG, chin and leg EMG, respiratory monitoring and blood oxygenation. Data was manually scored by a certified technician using standard American Academy of Sleep Medicine (AASM) criteria. To control for possible effects of dopaminergic drugs on encoding and retrieval, all patients took their usual dopaminergic medications 45 minutes before both the night and morning sessions. No patients took overnight dopaminergic drugs.





Percentage of time spent in non-REM stages 2 or 3 was not associated with overnight memory consolidation, controlling for time spent in other stages (stage 2: $\beta = 0.69$, *p*=0.36; stage 3: $\beta = -0.7$, *p*=0.62).

Higher AHI score (more severe obstructive sleep apnea) was associated with greater decay of memory overnight, controlling for total sleep time ($\beta = -0.009, p = 0.03$).

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Consolidation was measured as the relative difference between number of words correctly recalled in the morning and numbers of words correctly recalled at night.



Clinical and sleep characteristics				
	Mean	St. Dev	Min	Max
AHI	26.4	14.8	6.6	51.2
TST	319.4	62.3	217.0	421.5
N1%	21.6	8.3	10.0	34.0
N2%	58.9	12.4	40.3	76.7
N3%	5.9	6.8	0.5	24.0
Age	59	6.7	48	68
MoCA	26.6	3.3	18	30

Our sample has a moderate apnea-hypopnea index with on average 26.4 apnea or hypopnea events per hour. Our sample slept on average 5 hours (319.4 minutes).

Summary

- In this preliminary sample, severity of obstructive sleep apnea was associated with overnight memory consolidation, even controlling for total sleep time.
- This is important as treatments for OSA do exist and will have to be evaluated for their impact on sleep-dependent cognitive processes.
- Surprisingly, percentage of time spent in non-REM sleep was not associated with overnight memory consolidation. However, it is important to note that these gross measures of sleep architecture do not fully reflect the underlying electroencephalographic activity. Additional analyses will focus on spectral activity and on sleep spindles and their relationship to memory consolidation. This may also reveal an explanation for the relationship between OSA and memory consolidation.

References

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