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COMMENTARY

Perceiving the Misperception

Commentary on Saline et al. Sleep fragmentation does not explain misperception of latency or total sleep time. *J Clin Sleep Med* 2016;12(9):1245–1255.

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There is no truth. There is only perception.
—Gustave Flaubert

The International Classification of Sleep Disorders, Third Edition (ICSD-3)[1] defines paradoxical insomnia, or sleep state misperception, as follows:

> Paradoxical insomnia, which has previously been called sleep state misperception, is described as a complaint of severe sleep disturbance without corroborative objective evidence of the degree of sleep disturbance claimed.

As is the case with all primary insomnias in ICSD-3, paradoxical insomnia has been reduced to a subtype of "chronic insomnia disorder." The definition is purposely vague. Despite knowledge of its existence for decades, sleep state misperception remains difficult to identify and characterize. Does it exist in all insomniacs to varying degrees as some studies suggest?[2] Is it a specific subtype of insomnia that requires its own form of diagnosis and treatment?[3] Do physiologic correlates exist, and if so, are they clinically relevant?

Investigators have been unable to identify specific EEG characteristics on routine PSG analysis that correlate with a patient's tendency to mistake sleep for wake.[3–5] Research on belief constructs and comorbid behavioral health diagnoses haven't shown consistent associations.[4] Lastly, and perhaps most importantly, sleep state misperception is not defined in ICSD-3 using any of the basic tools clinicians have at their disposal—sleep logs, actigraphy, or PSG.

In this issue of the *Journal of Clinical Sleep Medicine*, Saline and colleagues venture back into the "mine-field" that is sleep state misperception.[6] They performed a retrospective analysis on 643 patients who had PSG performed in their lab. The morning after their sleep study, all subjects were asked to estimate their total sleep time (TST). Patients with obstructive sleep apnea (OSA) were included, as were those with insomnia complaints. The study was done to test definitions for sleep-onset latency (SOL) and TST misperception, on the assumption they represent two different processes. Once each was defined and measured separately, the authors hypothesized they'd find the relationship between fragmentation on PSG and misperception that has eluded them in the past.[5]

To start, they coin a new term: sleep during subjective latency (SDSL). SDSL refers to all objective sleep that occurs prior to subjective sleep onset. As an example, let's say a patient had a subjective SOL of 30 minutes. His SDSL would be the amount of objective sleep that occurs during the first 30 minutes of the PSG. If he immediately has 10 minutes of sleep followed by 20 minutes of wake, his SDSL would be 10 minutes. If the first 20 minutes are wake followed by 10 minutes of sleep, his SDSL would still be 10 minutes.

Per the authors, SDSL removes two important barriers to standardizing misperception across studies. First, it eliminates the need to define objective SOL, which has no gold standard. Second, SDSL can be subtracted from objective TST to account for the presumed patient tendency to anchor their subjective TST to their subjective SOL. This allowed them to calculate the latency-adjusted TST (LA-TST), defined as objective TST minus any sleep that occurred before subjective SOL. LA-TST ensures SOL misperception is not "double counted" as part of TST misperception.

To evaluate subjective SOL, the authors split patients into two groups—those with > 20 and those with 5–20 minutes of SDSL. For both OSA and non-OSA patients they found that > 20 minutes SDSL was associated with less N1%, more N3%, and fewer transitions. They had hypothesized that higher SDSL would correlate with measures of fragmentation, like lighter stages (N1) and more sleep-wake transitions. What they found was the opposite—fragmented sleep was associated with less misperception. They dichotomized misperception during LA-TST at 60 minutes and the findings were similar. If anything, more fragmentation, as measured by transition frequency or stage duration, was associated with less misperception.

This was a negative study, and there are a host of possible reasons why. To start, most patients presumably had PSG to rule out OSA. Although 70% had at least one insomnia symptom, this was not a group of insomniacs per se. Subjects reported taking hypnotics, antidepressants, and anxiolytics, and may have taken them the night of the study. Medications in these classes alter sleep architecture and affect cognitive processes, thus complicating the interpretation of the independent (EEG characteristics) and dependent (misperception) variables.[7–9] While the authors went to great lengths to assess

the predictive capacity of different EEG characteristics, some have suggested different metrics are more important.[10]

Lastly, there's the ever-present problem of measuring misperception. The authors outline logical arguments for using SDSL and LA-TST, and they should be commended for their work. They've pushed us one step closer to standardizing our perception of the misperception. Had they proven their hypothesis that fragmentation is associated with SDSL and subjective LA-TST, the reader could be more confident that the new definitions have value. As it stands though, SDSL and LA-TST will have to be studied again to prove their worth.

CITATION

Holley AB. Perceiving the misperception. *J Clin Sleep Med* 2016;12(9):1211–1212.

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DISCLOSURE STATEMENT

Dr. Holley has indicated no financial conflicts of interest.