COMMENTARY

Slow Waves and Learning: Beyond Correlations

Commentary on Landsness et al, Sleep-dependent improvement in visuomotor learning: a causal role for slow waves. SLEEP 2009;32:1273-84.

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ELECTROENCEPHALOGRAPHIC (EEG) SLOW WAVES ARE THE EPITOME OF DEEP SLEEP. THE PREVALENCE AND AMPLITUDE OF SLOW WAVES IN THE SLEEP EEG are typically quantified as slow-wave activity (SWA; power density in the 0.75 - 4.5-Hz range). SWA appears to be a marker of sleep need and to be an expression of a homeostatic sleep-regulatory process.[1] But it is unclear whether SWA is simply an EEG epiphenomenon or whether it directly serves important functions. In this issue of SLEEP, Landsness et al[2] tested the hypothesis that SWA is responsible for the consolidation of visuomotor learning.

Many recent studies have linked SWA to learning and synaptic plasticity. SWA is not the only aspect of sleep that has been related to such processes, but it has specifically been implicated in declarative memory,[3] as well as in overnight gains in perceptual[4] and visuomotor performance.[5] SWA appears to be use dependent such that cortical circuits that were particularly active during wakefulness produce more SWA during subsequent sleep,[5,6] whereas underused circuits produce less SWA.[7] Increases in SWA that were induced either by practicing a learning task during prior wakefulness[5] or by transcranial direct-current stimulation during sleep[3] were found to correlate positively with the magnitude of sleep-dependent learning effects.

Whereas much of the earlier evidence was correlative, a recent study using an acoustic SWA-suppression paradigm provided support for a causal relationship between SWA and sleep-dependent gains in perceptual learning.[8] The power of the SWA-suppression paradigm is that— if applied carefully—it allows for a sizable reduction of SWA, while total sleep time and REM sleep remain unaffected. Using this type of paradigm, Landsness et al[2] examined the role of SWA in rotation adaptation, a well-characterized form of visuomotor learning. The authors show that reduction of SWA by an average of 31% prevented overnight improvement in visuomotor performance and that changes in SWA over the right parietal cortex, as measured with high-density EEG, correlated with changes in performance. Despite its strength, the acoustic SWA-suppression paradigm also has limitations: it is difficult to know whether the behavioral effects of the stimuli are attributable solely to the decrease in SWA or additionally to an increase in arousals. Well aware of these limitations, Landsness et al[2] systematically pursued several new strategies to overcome them.

First, they included a control acoustic stimulation condition (CAS), in which subjects were exposed to stimuli during sleep but only when slow waves were absent. In contrast to slow wave deprivation (SWD), subjects exhibited significant learning in the CAS condition, thus ruling out the possibility that the acoustic stimulation procedure per se prevented learning. Next, the authors compared the number of clinically defined arousals. Although they found that the arousal index was slightly higher in the SWD than in the CAS conditions, multiple regression analysis with SWA and arousal index as independent variables revealed that only SWA predicted next-day visuomotor performance. Finally, the authors introduced a new automated arousal index that was based on an event-related spectral perturbation analysis of the EEG response to auditory stimuli. The basic idea was to identify EEG responses to stimuli that may not meet the criteria for clinically defined arousals but may represent a more subtle kind of sleep fragmentation that may differ between the SWD and CAS conditions. At the end of an exhaustive analysis, it was demonstrated, however, that it was indeed SWA and not a subtle type of "EEG lightening" or sleep fragmentation that predicted visuomotor performance.[2] The analysis validated the acoustic SWA-suppression paradigm and strengthened its interpretational power.

In summary, Landsness and colleagues[2] provide strong evidence for a causal role of SWA in visuomotor learning. Despite the clarity of these results, one should keep in mind that some other studies have failed to find effects of SWA suppression on learning[9,10] or on other waking functions.[11,12] It is possible that methodologic differences may have played a role, and, therefore, it will be important in the future to examine such differences more systematically. It is also reasonable to assume that waking functions differ in the degree to which they depend on SWA. The investigation of such differences may reveal clues about the functional role of SWA. The fact that the largest behavioral effects of acoustic SWA suppression so far have been found in learning[2,8] is consistent with a role for SWA in plasticity.

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