Non-Rapid Eye Movement (NREM) Sleep

Subjects: Neurosciences
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NREM is important part of the human sleep, and non-pharmacological interventions (acoustic, visual and other stimulations during sleep) are elaborated to modulate it.

healthy subjects NREM sleep delta wave power density acoustic stimulation visual stimulation

The two distinct states of non-rapid eye movement (NREM) and rapid eye movement (REM) sleep have their own neuroanatomic, electrophysiological, and behavioral characteristics. NREM is characterized by electroencephalography (EEG) that shows high-voltage K-complexes and sleep spindles in shallow sleep (stage N2) and high-voltage slow waves (0.5–2 Hz, amplitude above 75 μV) in deep, "slow-wave" sleep (stage N3)[1]. REM is characterized by low amplitude, mixed frequency EEG without K-complexes or sleep spindles[1]. NREM is followed by REM, with 4–5 of such cycles over the sleep episode, and the proportion of REM increasing towards the end of the night[2].

The voltage of the registered bioelectrical activity is normally highest in the first-cycle NREM sleep and then decreases exponentially[3]. A special parameter, EEG power density, calculated as square of the wave amplitude divided by wave frequency, namely in the frequency range between 0.5–4.5 Hz, is a recognized indicator of the effective dissipation of the 'sleep pressure' accumulated during daytime. The dissipation is often attenuated in depression (reduced delta sleep ratio), whereas increased NREM power density might be associated with better restorative sleep. EEG power density is denoted as “slow wave activity” (SWA) in the frequency band 0.5–4 Hz (subdivided further on slow oscillation 0.5–1 Hz and delta waves 1–4 Hz) and “spindle activity” in the 9–15 Hz band (subdivided further on slow spindle 9–12 Hz and fast spindle 12–15 Hz). The increase in SWA is accompanied by a decrease in spindle band power density that is also known to be characteristic for (deeper) sleep.

Stimuli studied for their potency to modulate sleep in a real-time manner include acoustic, visual, or olfactory stimuli, electrical or magnetic transcranial stimulation, vestibular, somatosensory stimuli (of peripheral nerves), or their combination. Repeated acoustic short (50 ms) stimuli increase slow wave activity and the number of K-complexes. Modern techniques allow anchoring the short stimuli to the up phase of endogenous slow waves to increase slow oscillatory activity (closed-loop in-phase stimulation), whereas stimulation time-locked to the down phase reduces slow waves. The behavioral and physiological consequences of acoustic enhancement of sleep slow waves may include an improvement of verbal declarative memory and immune status in healthy adults.

Sleep modulation properties of stimuli other than acoustic have been less investigated in humans. Visual pulsatile stimulation was reported to be not as potent, though there are no published studies found. However, in one article it was mentioned that a repeated light stimulation evoked more K-complexes than auditory stimuli did, probably due to the relatively high brightness of the stimuli presented from a light source positioned at three feet from the subject's face. Approximately 6% of light (5.6% of red, 0.3% of green, and 0.3% of blue light) passes through closed human eyelids, and even extremely low intensity light (0.002 lux) is sufficient to initiate a cascade of photochemical reactions in the open eyes of rats. It is also known that blue light, in comparison to red light, may immediately increase energy/alertness levels in humans (when acting via the open eyes).

A recent study showed that the electroencephalography (EEG) slow wave power density during human NREM sleep (stages 2 and 3) was not influenced by dim red light (short 50-ms signals presented via the eye mask), whereas auditory stimuli presented in the same closed-loop in-phase fashion (via headphones), did increase the EEG slow wave power density, as previously reported.

References

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