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Review

Ultradian and circadian modulation of dream recall: EEG correlates and age effects[☆]

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ABSTRACT

Dreaming occurs during non-rapid eye movement (NREM) and rapid eye movement (REM) sleep, which both are regulated by homeostatic, ultradian, and circadian processes. However, the magnitude of how ultradian REM and NREM sleep and its EEG correlates impact onto dream recall remains fairly unknown. In this review, we address three questions: 1. Is there an ultradian NREM–REM sleep modulation in successful dream recall, which is gated by the circadian clock? 2. What are the key electrophysiological correlates that account for dream recall during NREM and REM sleep and 3. Are there age-related changes in the ultradian and circadian regulation in dream recall and its electrophysiological correlates? Knowledge on the specific frequency and topography NREM and REM sleep differences prior to dream recall may pinpoint to the cerebral correlates that account for this cognitive process, and hint to their possible physiological meaning.

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1. Introduction

Dreaming is a universal human experience that offers one of the most unique views to consciousness and cognition, although there is much controversy on what exactly dreaming is. Theoretical definitions encompass simplistic perspectives as "any mental activity occurring in sleep" to specific frameworks as "dreaming represents the conscious awareness of complex brain systems involved in the reprocessing of emotions and memories during sleep" (Stickgold et al., 2001a) to narrower ones, such as "mental activity occurring in sleep characterized by vivid sensorimotor imagery that is experienced as waking reality despite such distinctive cognitive features as impossibility or improbability of time, place, person and actions; emotions, especially fear, elation and anger predominate over sadness, shame and guilt and sometimes reach sufficient strength to cause awakening; memory for even very vivid dreaming is evanescent and tends to fade quickly upon awakening unless special steps are taken to retain it" (Hobson et al., 2000). The functional importance of dreaming still remains a matter of intense debate. Chief among the hypotheses that may explain the relevance of dreaming are the mind–brain reductionism, which suggests that dreaming is a random by-product of underlying REM sleep physiology (Hobson and Mc Carley, 1977). More recently, dreaming has been argued as a means for reactivation and consolidation of novel and individually-relevant features of prior wake experience (Wamsley et al., 2010; Desseilles et al., 2011). Thus, dreaming may enable the replay of isolated elements (from their original context) for the integration of new features into existing cognitive representations linked to specific brain areas (Schwartz and Maquet, 2002). Another functional reasoning for dreaming to occur is that it may allow the enactment of affective processes by providing an internal activation, which is driven from an individual's affective and emotional history and serving an emotionality-regulation function (Cartwright et al., 1997a, 1997b; Nielsen and Chénier, 1999). Thus, dreaming may stimulate the resolution of emotional conflict and reduce next-day negative mood (Cartwright et al., 1997a).

In this review, we present electroencephalographic correlates of successful dream recall after NREM and REM sleep in healthy young and older volunteers and describe how NREM–REM sleep and circadian rhythmicity modulate dreaming, focusing particularly on our recent data on dream recall, and provide evidence on how it may change in healthy aging. Furthermore, we give an overview of neuroimaging data looking at the cerebral correlates that may account for dreaming, and draw, at the end, perspectives on what dreaming may imply.

2. Ultradian REM and NREM sleep modulation of dream recall and its EEG correlates

The neuroscientific basis of dreaming was firstly deemed to be modulated by the ultradian NREM–REM sleep cycle, particularly after the discovery that higher levels of dream recall were strongly connected to prior REM sleep (Aserinsky and Kleitman, 1953). Thus, it was plausible to believe that the specific brain mechanisms of REM

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sleep may hint to the origin of dream processing (Dement and Kleitman, 1957). This assumption leads to the "activation-synthesis" hypothesis, which described the brainstem generators of REM sleep as the basis of dreaming (Hobson and Mc Carley, 1977). This hypothesis triggered numerous studies thereof that aimed at understanding dream generation as a by-product of REM sleep (Cartwright et al., 1997b; Czaya et al., 1973; Foulkes, 1993; Casagrande et al., 1996). Nevertheless, this REM-centric belief has been challenged by compelling evidence of dreaming during NREM sleep (Cavallero et al., 1990; Nielsen, 2000, 2004). Initially, NREM dreaming was ascribed as a memory from a previous REM episode, as argued by the "covert" REM sleep theory, given that the great majority of NREM dream recall was observed during the second part of the night, most likely after a previous REM sleep episode (Nielsen, 2000). This type of assumption stemmed from the fact that NREM dreaming differs from REM dreaming on fundamental aspects, such that dream report lengths are 2 to 5 times longer in REM than NREM sleep episodes (Nielsen, 2004). Besides incidence and length, REM and NREM dreaming are substantially different with respect to its emotional salience. REM sleep dream reports are more emotional, anxious, with obvious visualized different scenes and more socially unacceptable content, while NREM reports are consistently more conceptual and thought-like (Hobson et al., 2000; Stickgold et al., 2001a).

If sleep EEG activity during dreaming reflects the activation or deactivation of particular anatomical pathways related to this process, it may unravel processes related to dreaming (Desseilles et al., 2011). Previous data on EEG spectral power and dream recall have provided mixed results. For instance, EEG beta activity has been shown to increase in NREM sleep stage 2, at the transition from epochs without dream recall to those with distinct recall (Williamson et al., 1986). Furthermore, less EEG power density in the theta range (5–8.5 Hz) has been reported for successful recall subsequent to NREM sleep (Cajochen et al., 2003). With respect to REM dreaming, alpha activity (Esposito et al., 2004) and widespread 40-Hz oscillations have been associated to increased dream recall (Llinás and Ribary, 1993), as both may induce large functional states for cognitive processing. Faster oscillations during REM sleep may thus turn out to be an "electrophysiological signature" for dream processing (Llinás and Ribary, 1993). Interestingly, there is also evidence for an inverse relationship between EEG slow-wave activity and dream recall, with higher levels of delta activity associated to no recall conditions during NREM sleep and, particularly, REM sleep (Esposito et al., 2004). Indeed, the inverse association was more robust for REM sleep, indicating that delta power may be a negative index of dream recall. Early studies found similar inverse relationships for more broadly defined frequency bands, such as 4.0–14.0 Hz (Lehmann et al., 1981). More recently, successful dream recall, after morning awakening from REM sleep, was associated to higher frontal theta (5–7 Hz) activity, while dream recall following morning awakening from NREM sleep stage 2 resulted in less alpha (8–12 Hz) activity of the right temporal area (Marzano et al., 2011). These results may possibly hint to a neurophysiological mechanism that underlies encoding and recall of episodic memories, which may remain similar across different sleep states (Marzano et al., 2011). However, the precise EEG spectral correlates of dream recall are still unclear. One likely candidate is the use of different EEG approaches, such as which sleep stages are taken into account (only NREM or REM sleep or both), and which power spectra are computed for the analysis (NREM sleep delta or REM sleep alpha power, and so forth) (Armitage et al., 1989; Esposito et al., 2004). Another possible explanation is the use of different approaches accounting for dreams, such as qualitative (dream content) or quantitative methods (dream recall and number of dreams) (Stickgold et al., 2001b). Another aspect that also remains largely unknown is the role of the circadian timing system on the generation of dreaming. In the next section we address studies that focused on the impact of the circadian timing system on dreaming.

3. The Tick-tock of dreaming: circadian activation of dream recall

Circadian-driven changes in cortical activity are a very likely candidate to promote dreaming during NREM sleep and, particularly, during REM sleep (Wamsley et al., 2007). Compelling evidence for circadian influences on dreaming arise from a study in which relationships between circadian factors and dreaming were out of sync (Antrobus et al., 1995). In this study, sleep onset and offset were delayed by 3 h to create a phase delay of dreaming and to test the hypothesis of circadian influences. REM and NREM dreaming would then presumably occur 3 h later than usual, thus coinciding with the hypothetical rising phase of core body temperature and with temporally close to the peak of REM sleep propensity. Dream reports were then compared from the phase-delayed condition to control reports from non-delayed sleep (Antrobus et al., 1995). Delayed dream reports were longer and visually more intense, particularly during the latter part of the night as compared to reports collected at the beginning of the night. While REM and NREM sleep differences were observed, both REM and NREM dream reports were affected by the circadian factor independent of sleep state differences. In particular, for visual imagery the circadian effect size (0.23 or small) was about 30% of the ultradian NREM/REM effect size (0.70 or large). These findings suggest that the ultradian NREM-REM sleep and circadian sources of cortical and subcortical activation can be independent, but additive, in their effects on dreaming (Antrobus et al., 1995). Further support for the circadian-driven activation of dreaming derives from an ultra-short multiple-nap protocol study (20/40 min sleep/wake schedules) (Suzuki et al., 2004). Dreaming scores from NREM sleep reports were sinusoidally distributed across the 24-h day, with maxima at 08:00 h. REM sleep report scores were high during the diurnal period of 06:00–16:00 h and then dropped. Of particular interest, the NREM sleep dream score peak coincided with the peak of REM sleep stage duration. One likely explanation could be that human dream experiences in NREM sleep are influenced by the REM sleep-generating mechanisms (Nielsen, 2004). Ponto-geniculo-occipital activity, a robust factor activating the visual cortices that generates dream experiences, and a hallmark of REM sleep (Hobson and Mc Carley, 1977), may occur in human NREM sleep, particularly in the morning hours when REM sleep is most likely to occur, thus resulting in dream experiences during NREM sleep (Suzuki et al., 2004). REM sleep propensity is under tight circadian control, and exhibits a clear peak in the early morning hours (Dijk et al., 1997). Under this assumption, one may hypothesize that the coincident NREM sleep dream peak with the peak of REM sleep stage duration described in Suzuki et al. (2004) suggests that the dreaming propensity from both REM and NREM sleep is influenced by the circadian oscillator.

In the next section, we address recent studies on how dreaming is modulated by the NREM–REM sleep regulation at an electrophysiological level, and what is the role of the circadian system on dream recall.

4. The NREM-REM sleep and the circadian signatures of dreaming

Recently we investigated the electrophysiological NREM and REM sleep EEG correlates prior to dream recall and age-related effects on dream recall (Chellappa et al., 2009, 2011, 2012). EEG power density during sleep associated with and without dream recall was investigated in both young and older participants. Furthermore, we focused on how the topographic distribution of EEG activity during NREM and REM sleep can underpin potential cortical networks related to dream recall. The methodological rationale was to access dream recall using the sleep mentation questionnaire (Chellappa et al., 2009), which was carried out immediately after awakenings from scheduled 75-min naps during a 40-h multiple nap paradigm (150/75 min of wake–sleep schedule; 10 naps in total), under constant routine conditions (Cajochen et al., 2006). This enabled the estimation of dream

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