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Research Paper

Sleep physiology in toddlers: Effects of missing a nap on subsequent night sleep

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ABSTRACT

The shift from a biphasic to a monophasic sleep schedule is a fundamental milestone in early childhood. This transition, however, may result in periods of acute sleep loss as children may nap on some but not all days. Although data indicating the behavioral consequences of nap deprivation in young children are accumulating, little is known about changes to sleep neurophysiology following daytime sleep loss. This study addresses this gap in knowledge by examining the effects of acute nap deprivation on subsequent nighttime sleep electroencephalographic (EEG) parameters in toddlers. Healthy children ($n=25$; 11 males; ages 30–36 months) followed a strict sleep schedule for ≥ 5 days before sleep EEG recordings performed on 2 non-consecutive days: one after 13 h of prior wakefulness and another at the same clock time but preceded by a daytime nap. Total slow-wave energy (SWE) was computed as cumulative slow-wave activity (SWA; EEG power in 0.75–4.5 Hz range) over time. Nap and subsequent night SWE were added and compared to SWE of the night after a missed nap. During the night following a missed nap, children fell asleep faster (11.9 ± 8.7 min versus 37.3 ± 22.1 min; $d=1.6$, $p=0.01$), slept longer (10.1 ± 0.7 h versus 9.6 ± 0.6 h; $d=0.7$, $p<0.01$) and exhibited greater SWA ($133.3 \pm 37.5\%$ versus $93.0 \pm 4.7\%$; $d=0.9$, $p<0.01$) compared to a night after a daytime nap. SWE for combined nap and subsequent night sleep did not significantly differ from the night following nap deprivation (12141.1 ± 3872.9 μV^2*h versus $11{,}588 \pm 3270.8$ μV^2*h; $d=0.6$, $p=0.12$). However, compared to a night following a missed nap, children experienced greater time in bed (13.0 ± 0.8 h versus 10.9 ± 0.5 h; $d=3.1$, $p<0.01$) and total sleep time (11.2 ± 0.8 h versus 10.1 ± 0.7 h; $d=1.4$, $p<0.01$). Shorter sleep latency, longer sleep duration, and increased SWA in the night following a missed nap indicate that toddlers experience a physiologically meaningful homeostatic challenge after prolonged wakefulness. Whether toddlers fully recover from missing a daytime nap in the subsequent night necessitates further examination of daytime functioning.

1. Introduction

Brain electrophysiology of recovery from sleep loss has been extensively characterized in adolescents and adults. Slow wave sleep (SWS) and slow wave activity (SWA; power density in the 0.75–4.5 Hz frequency range) of the sleep electroencephalogram (EEG) are well-known physiological markers of sleep pressure, exhibiting a saturating rise with duration of prior wakefulness and an exponential decline with time asleep (Daan et al., 1984). Following sleep loss, recovery sleep is marked by increased SWS and SWA, as well as shorter sleep onset latency and longer sleep duration (Brunner et al., 1993). Although the electrophysiology of adult sleep is predictable according to the two-process model of sleep regulation (Borbely, 1982), large inter-individual differences have been observed in the SWA response to sleep deprivation (Finelli et al., 2000; Rusterholz et al., 2010). This study extends the prior literature by quantifying the neurophysiological response to acute sleep restriction in toddlers.

Nearly all 2-year-olds fulfill part of their 24 h sleep need by taking a daytime nap (Weissbluth, 1995; Thorleifsdottir et al., 2002; Iglowstein et al., 2003; Crosby et al., 2005). In the next few

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years of life, napping declines. By age 5 years, the great majority of children sleep only at night. This shift from a biphasic to a monophasic sleep schedule in early childhood is a naturalistic context for studying sleep homeostasis, as children may nap on some but not all days of the week (Weissbluth, 1995; Crosby et al., 2005). Also, because young children's sleep habits are largely determined by caregivers and modulated by cultural and psychosocial factors (Crosby et al., 2005; Roffwarg et al., 1966; Komada et al., 2011), toddlers may experience daytime sleep restriction for reasons outside their control (e.g., daycare/preschool schedules, family demands). Although recent data show that habitually napping preschool-age children show decrements in behavioral processes (e.g., emotion expression, self-regulation strategies, declarative memory performance) after nap deprivation (Berger et al., 2012; Kurdziel et al., 2013; Giganti et al., 2014), less is known about changes to nighttime sleep neurophysiology following a missed nap in early childhood.

In the present study, we quantified recovery sleep neurophysiology following a missed nap with sleep EEG in a sample of healthy children ages 30–36 months. First, in order to quantify the physiological response to a missed nap, we compared sleep architecture, SWS, and SWA between a night following a nap and a night following a missed nap. We hypothesized that missing a nap would decrease sleep onset latency and increase sleep efficiency, sleep duration, SWS, and SWA. Second, we compared slow-wave energy (SWE; cumulative SWA over time) of a combined nap and subsequent night to that of a night following a missed nap. SWE was used to quantify neurophysiological recovery from a missed nap because it provides a comprehensive quantification of recovery sleep (Dijk and Beersma, 1989; Achermann and Borbely, 1990; Hekkens GAK and Rietveld, 1988) and has been used in similar experimental paradigms in adults (Werth et al., 1996; Campbell et al., 2005). We expected an increase in SWE following a missed nap and explored the extent to which toddlers neurophysiologically recover from a missed nap with one night's sleep (i.e., whether toddlers compensate for a missed nap with a commensurate increase in SWE during nighttime sleep).

2. Materials and methods

2.1. Participants

Participants were 25 healthy habitually napping children ages 30–36 months (11 males; 33.5 ± 1.8 (SD) months) from Boulder, CO or surrounding areas. Details regarding recruitment and study exclusion criteria have been previously published (Berger et al., 2012; LeBourgeois et al., 2013). Briefly, all participants were screened for reported sleep problems/disorders, variable sleep schedules, chronic use of medications affecting sleep, alertness, or the circadian system, a personal or family history of sleep or mental health problems or personal history of developmental disabilities, head injury, chronic illnesses, or low birth weight. Of the 102 children screened, 45 met criteria, 35 were enrolled, and 25 were included in this analysis. Exclusions were due to discomfort with electrodes ($n=4$), EEG artifacts ($n=4$), or failure to complete all 3 sleep EEG assessments ($n=2$). Parents signed a consent form approved by the University of Colorado Boulder Institutional Review Board, and study procedures were performed according to the Declaration of Helsinki. Parents were compensated with $80 cash, and children received small non-monetary gifts throughout the study and a $200 U.S. Saving's Bond.

2.2. Training

Researchers performed ≥ 3 in-home trainings during the week before sleep EEG assessments. These trainings introduced children to study methods, such as wearing a wrist actigraph and attaching electrodes to the face and scalp.

2.3. Protocol

Fig. 1 illustrates a sample protocol for one participant. Children followed an individualized, stable sleep schedule for 5 days before completing each of 2 counterbalanced in-home 24 h sleep EEG recordings. Children did not attend daycare or preschool on these assessment days. The schedule required a minimum sleep opportunity of 12.5 h per 24 h day (including a daily nap opportunity of ≥ 45 min) and was employed to minimize sleep restriction and to optimize circadian entrainment before the sleep assessments. Schedule compliance was verified with actigraphy, sleep diaries, and daily contact with parents. Participants slept in their typical environment throughout the study (i.e., home, daycare, family care). Consumption of caffeine and medications affecting sleep, alertness, or the circadian system were prohibited throughout the study. Assessments were rescheduled if a child was ill within 24 h before the assessment or if their sleep timing deviated > 15 min from the prescribed schedule.

The experimental protocol included 2 counterbalanced sleep conditions: Baseline (afternoon nap and subsequent overnight) and Recovery (overnight following a missed nap). The Baseline Nap was recorded after 4 h of wakefulness (Fig. 1; e.g., Day 6), and both the Baseline and the Recovery Nights were performed 13 h after morning wake time (Fig. 1; e.g., Nights 6 and 13). Children were allowed to sleep until spontaneously waking. Children slept on their normal schedule for ≥ 5 intervening nights between conditions.

2.4. Measures

2.4.1. Sleep EEG

Sleep EEG from derivations C3A2, C4A1, OzA1, and FzA2 (Jasper, 1958), submental electromyogram, and electrooculogram were recorded using a Vitaport 4 EEG recorder (Temec Instruments, Kerkrade, Netherlands) with a sampling rate of 256 Hz. The high-pass filter was at 0.16 Hz and the anti-aliasing low-pass filter (2nd order) at 70 Hz.

2.4.2. Visual sleep stage scoring and sleep cycle definition

Sleep stages were visually scored in 30-s epochs according to standard criteria (Rechtschaffen and Kales, 1968). Sleep onset latency was defined as the duration (min) from lights off to the first epoch of stage 2 sleep. SWS latency and REM latency were defined as the duration from sleep onset to the first epoch of SWS and REM sleep, respectively. Sleep stage percentages were calculated in reference to the sleep period (sleep onset to last epoch of sleep). NREM and REM sleep episodes were defined according to standard criteria (Rechtschaffen and Kales, 1968; Feinberg and Floyd, 1979) and adapted when a “skipped” REM was observed after the first NREM sleep episode. As previously described in studies of children and adolescents (Kurth et al., 2010; Jenni and Carskadon, 2004), NREM episodes were manually divided if (1) the duration of the first NREM sleep episode exceeded 120 min and (2) SWS sleep in the first NREM episode was interrupted for at least 12 continuous min of stage 1 sleep, stage 2 sleep, wakefulness, or movement time. If both criteria were met, the NREM sleep episode was divided at the SWS midpoint. In total, there were 10 cases of skipped first REM episodes; 1 during a Baseline Nap, 3 during a Baseline Night, and 6 during a Recovery Night.

2.4.3. Protocol verification: sleep diary and actigraphy

Throughout the study, parents completed a daily 26-item sleep