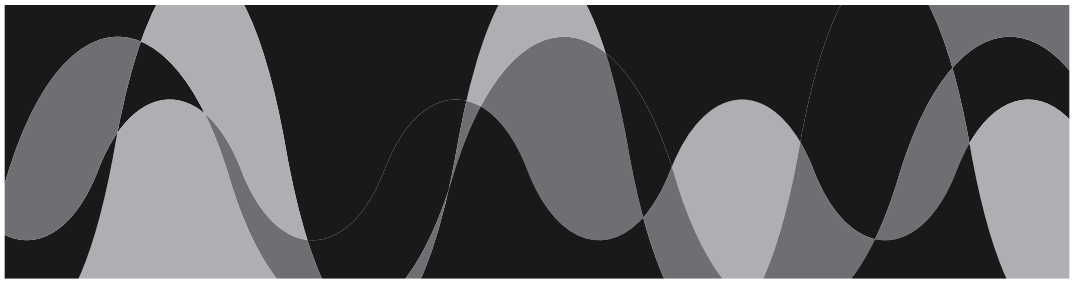

Ninth Meeting

Society for Research on Biological Rhythms

Program and Abstracts

SRS/SRBR

June 23, 2004

SRBR

June 24–26, 2004

Whistler Resort • Whistler, British Columbia

035

NASA workshop

TBA

036

REM Sleep Function: Maybe It's the Rhythm (Not the Tune)

Thomas J. Balkin*, Nancy J. Wesensten, Allen Braun, Gregory Belenky • Walter Reed Army Institute of Research

The physiological processes by which sleep restores alertness and cognitive performance are not known, but it is generally thought that these restorative processes require, or are subserved by, the relative brain deactivation that characterizes slow wave sleep. By EEG criteria, however, Rapid Eye Movement (REM) is a state of brain activation (at a level comparable to that seen during wakefulness). Clearly, if the restorative benefits of sleep accrue as a function of brain deactivation, REM sleep must serve a different purpose. The paradoxical nature of REM sleep, and the fact that most dream mentation occurs during REM, has captured the imagination of sleep researchers for the past 51 years÷and a spate of hypotheses regarding possible functions of REM sleep have been proposed. These have ranged from exotic psychoanalytic notions that dreams are somehow necessary for symbolically working through unresolved conflicts, to the mundane÷for example, that REM sleep is necessary for providing "practice" for conjugate eye movements. Currently popular is the notion that REM sleep enhances consolidation of some types of memory. Based on recent findings from functional brain imaging studies during sleep, an alternative function of REM sleep is proposed÷that it serves as a periodic probe to determine the status of the brain (with respect to its readiness for waking), and provides an internal stimulus for awakening when the brain is ready to do so.

037

Sleep Freely to Know Your DLMO

Burgess*, HJ and Eastman, CI • Rush University Medical Center

We analyzed baseline sleep times from 120 young healthy subjects to determine the phase relationship between the DLMO and sleep in subjects following fixed or free sleep schedules. "Free sleepers" (N=60) slept at times of their own choosing and "fixed sleepers" (N=60) slept on a fixed schedule similar to their weekday schedule. Subjects completed sleep logs that were verified by actigraphy. We averaged sleep times from 6 days before we measured the DLMO (threshold=mean+2SD of first 3 low daytime points). The fixed sleepers had earlier DLMOs and sleep times (fixed: DLMO 20:46±1.1 h, bedtime (lights out) 23:17±0.9 h, wake time (lights on) 7:06±1.0 h; free: DLMO 22:41±1.5 h, bedtime 00:56±1.1 h, wake time 9:25±1.3 h, all $p<0.001$). There was no difference in the DLMO to bedtime interval (fixed: 2.5±1.1 h; free: 2.3±1.2 h), but the wake time to DLMO interval was slightly longer in the fixed sleepers (fixed: 13.7±1.1 h, free: 13.3±1.1 h, $p=0.056$). The highest correlation between sleep times and the DLMO was with wake time. Surprisingly, the correlation between wake time and the DLMO was higher in the free sleepers (free: $r=0.70$; fixed: $r=0.44$, $p<0.05$). This is likely because fixed sleepers slept at times determined by social factors, whereas free sleepers slept at times promoted by the circadian clock. Thus maintaining a fixed sleep

schedule does not necessarily improve estimations of the timing of the DLMO. Support: RO1 NR07677, RO1 OH03954.

038

Differential Effect of Sleep Disruption on Menstrual Cycle Dynamics

Elizabeth B. Klerman*[1], Janet E. Hall[2] • [1]Division of Sleep Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston MA, [2]Reproductive Endocrine Unit, Massachusetts General Hospital and Harvard Medical School, Boston MA

We hypothesized that sleep disruption designed to mimic rotating shifts would alter menstrual cycle dynamics, but might have different effects depending on the phase of the cycle in which it occurred. Seven healthy young women (19-34 years) with regular menstrual cycles and on no medications affecting reproductive hormones or sleep were studied. The five-month study included a regular sleep-wake schedule and monthly assessments of day of menses and ovulation as well as a five-day inpatient stay in month 3 during either the follicular (FP; menses –1 to +1 days) or luteal phase (LP; ovulation + 3 to +5 days). The first night, subjects slept for 8 hours at their habitual bedtimes. They were then awake for 24 hours. For the following three days, they slept for 8 hours beginning at their habitual waketime and were awake for the remaining 16 hours. Sleep disruption in the FP resulted in a change in cycle length in all 3 subjects of greater than or equal to 2 days compared with the mean of cycles 1, 2 and 4. This difference was accounted for by an increase in FP length in two subjects and a decrease in FP length in one subject. Sleep disruption in the LP was not associated with changes in cycle length in the cycle of study. However, the following FP was increased in 2 of 4 subjects and decreased in one. Taken together these preliminary results suggest that sleep disruption in either the follicular or luteal phase may affect menstrual cycle dynamics. Support: NIH-R01-HD40291, NIH-NCRR-GCRC-M01-RR02635 and M01-RR01066.

039

Interactions Between Circadian Rhythms and Epilepsy

Mark Quigg • University of Virginia

Circadian rhythms have marked effects on expression of epilepsy, influencing the timing of occurrence of seizures and the characteristics of interictal epileptiform discharges (IEDs). Circadian rhythms are not a singular entity but involve many systems within and without the central nervous system. Therefore, a circadian influence is not likely a single, unified mechanism. Rather, circadian rhythms are better thought of as endogenously–mediated, excitatory and inhibitory influences that vary with time of day and dynamically compete with other seizure precipitants to elevate or depress seizure threshold. I review basic concepts of chronobiology, the organization of the circadian timing system, the effects of epilepsy and seizures on circadian rhythms, and the influences of circadian rhythms on the timing of seizures.