expeditions. Indeed, even in 1993 Fiennes and Stroud needed to be picked up from an ice shelf before reaching its edge, having totally exhausted their energy reserves man-hauling across the Antarctic continent. Thus, even today, while technological advancements have engineered out certain weaknesses of the human condition, others remain as limiting factors that must be stretched to breaking point if explorers on foot are to return home alive from the South Pole.

Acknowledgements

We would like to thank Peter Clarkson, Adrian McCallum, Denis Wilkins, Tom Davies, Ben Edwards and Mark Hines for their comments on drafts of the essay. An especial thank you goes to Heather Lane at the Scott Polar Research Institute for her support during this project.

References

1. Massey, P. (1959). Finger numbness and temperature in Antarctica. J. Appl. Physiol. *14*, 616–620.
2. Zuntz, N. (1906). Höhenklima und Bergwanderungen in ihrer Wirkung auf den Menschen, (Berlin: Bong).
3. Stroud, M. (1998). The nutritional demands of very prolonged exercise in man. Proc. Nutr. Soc. *57*, 55–61.
4. Westerterp, K.R., Saris, W., van Es, M., and Ten Hoor, F. (1986). Use of the doubly labelled water technique in humans during heavy sustained exercise. J. Appl. Physiol. *61*, 2162–2167.
5. Marshall, E. (1943). Off the beaten track. In The Medical Press and Circular, Volume 8. pp. 359–362.
6. Rogers, A. (1981). The influence of diet in Scott's last expedition. In Starving Sailors. The Influence of Nutrition upon Naval and Maritime History, J. Watt, E. Freeman and W. Bynum, eds. (London: National Maritime Museum), pp. 163–173.
7. Tsintzas, O., Williams, C., Singh, R., Wilson, W., and Burrin, J. (1995). Influence of carbohydrate-electrolyte drinks on marathon running performance. Eur. J. Appl. Physiol. *70*, 154–160.
8. Speth, J., and Spielmann, K. (1983). Energy source, protein metabolism, and hunter-gatherer subsistence strategies. J. Anthropol. Archaeol. *2*, 1–31.
9. McArdle, W., Katch, F., and Katch, V. (2007). Exercise at medium and high altitude. In Exercise Physiology Energy, Nutrition and Human Performance, W. McArdle, F. Katch and V. Katch, eds. (Philadelphia: Lippincott Williams & Wilkins).
10. Grandjean, A., and Grandjean, N. (2007). Dehydration and Cognitive Performance. J.e Am. College Nutrition *26*, 5498–5548.
11. Wright, A. (1900). On the pathology and therapeutics of scurvy. Lancet *156*, 565–567.

[1]Roehampton University, Holybourne Avenue, London SW15 4JD, UK. [2]NIHR Biomedical Research Unit in Nutrition, Southampton University Hospitals Trust, Southampton SO16 6YD, UK.
*E-mail: l.halsey@roehampton.ac.uk

Correspondence

Rocking synchronizes brain waves during a short nap

Laurence Bayer[1*], Irina Constantinescu[1*], Stephen Perrig[2], Julie Vienne[3], Pierre-Paul Vidal[4], Michel Mühlethaler[1*] and Sophie Schwartz[1,5*]

Why do we cradle babies or irresistibly fall asleep in a hammock? Although such simple behaviors are common across cultures and generations, the nature of the link between rocking and sleep is poorly understood [1,2]. Here we aimed to demonstrate that swinging can modulate physiological parameters of human sleep. To this end, we chose to study sleep during an afternoon nap using polysomnography and EEG spectral analyses. We show that lying on a slowly rocking bed (0.25 Hz) facilitates the transition from waking to sleep, and increases the duration of stage N2 sleep. Rocking also induces a sustained boosting of slow oscillations and spindle activity. It is proposed that sensory stimulation associated with a swinging motion exerts a synchronizing action in the brain that reinforces endogenous sleep rhythms. These results thus provide scientific support to the traditional belief that rocking can soothe our sleep.

In the present study, we asked twelve healthy male volunteers (22–38 years old) to nap on a bed that could either remain stationary or rock gently (0.25 Hz; Figure 1A). All participants were good sleepers, non-habitual nappers with no excessive daytime sleepiness and had low anxiety levels. Sleep quality and quantity were assessed by questionnaires and actimetry recordings. The experimental procedure involved taking two 45-minute afternoon naps (2:30 to 3:15 PM): one with the bed stationary, and one with the bed put in motion (condition order randomized). The motion parameters were set to stimulate vestibular and proprioceptive sensory systems, without causing nausea or any entrainment of cardiac rhythm. In both conditions the naps were spent in complete darkness in a controlled room temperature (21 ± 1°C) and the level of auditory stimulation was around 37 dB. During both sessions, polysomnography data were recorded continuously. Sleep stages and sleep spindles were visually identified by two experienced scorers, blind to the experimental conditions. We also performed spectral analysis (FFT routine) using the midline frontal (Fz) and parietal (Pz) derivations. The data from two participants were excluded from the final analyses (see the Supplemental Information).

Over the three consecutive nights preceding each experimental day, all participants had a good quality and quantity (mean ± s.e.m.; 7.32 ± 0.78 h) of sleep as assessed by self-rated sleep questionnaires, with no difference for these measurements between stationary and swinging conditions. Similarly, wrist actimetry recorded during these same nights did not show any difference in sleep efficiency between conditions (mean ± s.e.m.; swinging: 86.63 ± 1.95%; stationary: 86.71 ± 1.23%). For both conditions, participants were more alert (on visual analogue scale) after napping than before ($F(1,9) = 8.4$, $P = 0.018$). Eight participants rated the swinging condition as 'more pleasant' than the stationary condition; for one participant both sessions were equally pleasant and for one participant the stationary condition was more pleasant.

We found that rocking accelerated sleep onset, as evidenced by a shorter duration of stage N1 sleep and a reduction of stage N2 latency, compared to the stationary condition (Supplemental Table S1). Rocking also affected deeper sleep stages by increasing the duration of stage N2 sleep and the mean spindle density per 30-s epoch (Supplemental Table S1, Figure 1B). Spindle density increased significantly from the second half of the nap (Figure 1C) and persisted throughout the entire duration of stage N2 (Supplemental Figure S1A). All these modifications were observed in each and every participant (all $P < 0.009$; Supplemental Table S1). In the only previous study investigating the effect of rocking on sleep, Woodward *et al.* [1] found no consistent modulation for the percentage of stage 1 sleep and an overall reduction of the percentage of stage 2 sleep during the motion condition. In contrast to our present

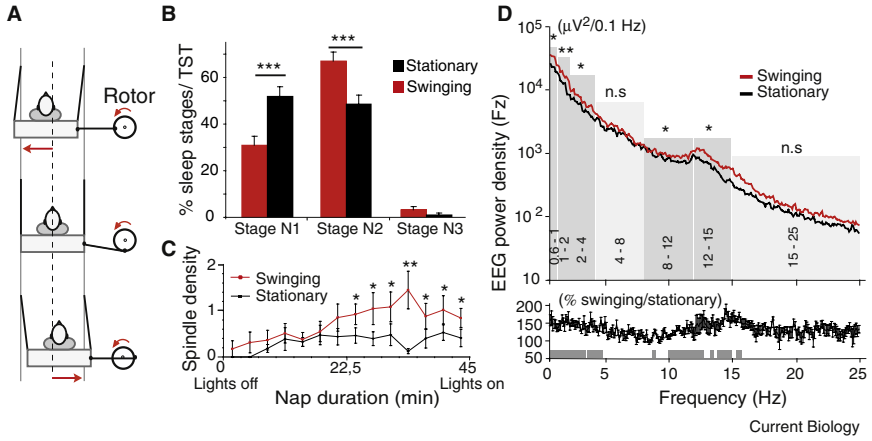


Figure 1. Experimental set-up and results.
(A) Schema of bed rocking. (B) Decreased stage N1 and increased stage N2 during rocking compared to stationary condition. ***P < 0.001. (C) Spindle density (mean number of spindles per 30-s epoch) against time from lights off until lights on. The total nap period is divided in 14 equal parts for each individual recording. Paired t-tests are performed at each time point. **P < 0.005, *P < 0.05. (D) Fast Fourier analysis during N2 at Fz derivation. Top: average power spectrum for the swinging and stationary condition: **P < 0.005, *P < 0.05. Bottom: relative power density (swinging/stationary x 100) for successive frequency bins of 0.1 Hz from 0.6 to 25 Hz. Horizontal gray bars indicate the significant bins, P < 0.05.

study, however, these data were computed over whole nights of sleep recordings, and did not address the question of whether vestibular/somatosensory inputs influence the transition from wakefulness to sleep (stage 1 and 2 sleep early in the night after sleep onset).

Rocking also increased EEG power of slow wave activity (SWA: 0.6–5 Hz; Figure 1D), predominantly during the last third of stage N2 (Supplemental Figure S1B; P < 0 .005). A significant increase of EEG power within spindle frequency bands was also observed for the frontal derivation (P < 0.05; Figure1D and Supplemental Figure S1C), but not for the parietal derivation (P > 0.07; Supplemental Results) [3]. Together these results show that rocking induces a speeded transition to an unambiguous sleep state, and may enhance sleep by boosting slow oscillations and spindle activity.

How can we explain that rocking may accelerate wake-sleep transition and promote sleep consolidation? Three mechanisms could explain these effects of rocking on sleep. First, because vestibular/somatosensory pathways have anatomical links with structures implicated in emotions such as the amygdala [4] and because the amygdala affects the regulation of sleep-wake states [5], faster sleep onset could be due to a 'relaxing' feeling associated with the rocking condition, which most of our participants (8 out of 10) found pleasant. Second, rhythmic vestibular/somatosensory inputs associated with rocking may modulate sleep-wake centres via direct or indirect connections between sensory systems and hypothalamic [6] or brainstem areas [7]. Third, sensory inputs could affect the synchrony of neural activity within thalamo-cortical networks because both somatosensory and vestibular inputs send direct projections to thalamic nuclei [8]. Consistent with this view, slow rhythmic cortical stimulation was recently found to increase EEG slow oscillations and spindles [3,9], which are both hallmarks of deep sleep. The latter hypothesis of an influence on neural synchrony fits best the present observation that rocking does not only facilitate sleep onset but has a persistent effect on brain oscillations and spindles. Recent evidence that increased spindle activity protects sleep against disruptive stimuli is in agreement with this interpretation [10]. Follow-up experiments could assess whether sleep changes triggered by rocking have beneficial functional consequences on post-sleep performance or on memory consolidation processes [3].

We suggest that rhythmic rocking may enhance synchronous activity within thalamo-cortical networks, which in turn could promote the onset of sleep and its maintenance. The use of rocking to soothe the sleep thus belongs to our repertoire of adaptive behaviours in which a natural mechanism of sleep (thalamo-cortical synchronization) has been harnessed in the simplest manner since immemorial times.

Supplemental Information

Supplemental Information includes one figure, one table, Supplemental Results and Supplemental Experimental Procedures, and can be found with this article online at doi:10.1016/j.cub.2011.05.012.

Acknowledgments

This work was supported by the Swiss National Science Foundation. We thank A. Borbély, P. Franken, C. Frith, B.E. Jones, C. Leonard, and M. Tafti for their comments.

References

1. Woodward, S., Tauber, E.S., Spielmann, A.J., and Thorpy, M.J. (1990). Effects of otolithic vestibular stimulation on sleep. Sleep *13*, 533–537.
2. Krystal, A.D., Zammit, G.K., Wyatt, J.K., Quan, S.F., Edinger, J.D., White, D.P., Chiacchierini, R.P., and Malhotra, A. (2010). The effect of vestibular stimulation in a four-hour sleep phase advance model of transient insomnia. J. Clin. Sleep Med. *6*, 315–321.
3. Marshall, L., Helgadottir, H., Molle, M., and Born, J. (2006). Boosting slow oscillations during sleep potentiates memory. Nature *444*, 610–613.
4. Carmona, J.E., Holland, A.K., and Harrison, D.W. (2009). Extending the functional cerebral systems theory of emotion to the vestibular modality: a systematic and integrative approach. Psychol. Bull. *135*, 286–302.
5. Chou, T.C., Bjorkum, A.A., Gaus, S.E., Lu, J., Scammell, T.E., and Saper, C.B. (2002). Afferents to the ventrolateral preoptic nucleus. J. Neurosci. *22*, 977–990.
6. Horowitz, S.S., Blanchard, J., and Morin, L.P. (2005). Medial vestibular connections with the hypocretin (orexin) system. J. Comp. Neurol. *487*, 127–146.
7. Jones, B.E. (2003). Arousal systems. Front. Biosci. *8*, s438–s451.
8. Moruzzi, G. (1972). The sleep-waking cycle. In Reviews of Physiology: Biochemistry and experimental pharmacology, R.H. Adrian, E. Helmreich, H. Holzer, R. Young, K. Kramer, O. Kreayer, F. Lynen, P.A. Miescher, H. Rasmussen, A.E. Renold, et al., eds. (Berlin, Heidelberg, New York: Springer-Verlag), pp. 1–165.
9. Massimini, M., Ferrarelli, F., Esser, S.K., Riedner, B.A., Huber, R., Murphy, M., Peterson, M.J., and Tononi, G. (2007). Triggering sleep slow waves by transcranial magnetic stimulation. Proc. Natl. Acad. Sci. USA *104*, 8496–8501.
10. Dang-Vu, T.T., McKinney, S.M., Buxton, O.M., Solet, J.M., and Ellenbogen, J.M. (2010). Spontaneous brain rhythms predict sleep stability in the face of noise. Curr. Biol. *20*, R626–R627.

[1]Department of Neuroscience, University of Geneva, Switzerland. [2]Sleep Laboratory, Geneva University Hospital, Switzerland. [3]CIG, University of Lausanne, Switzerland. [4]CNRS, UMR 8194-Université Paris Descartes, France. [5]Swiss Center for Affective Sciences, University of Geneva, Switzerland.
*These authors contributed equally to the work.
E-mail: sophie.schwartz@unige.ch; michel.muhlethaler@unige.ch

Supplemental Information:

Rocking synchronizes brain waves during a short nap

Laurence Bayer, Irina Constantinescu, Stephen Perrig, Julie Vienne, Pierre-Paul Vidal, Michel Mühlethaler, Sophie Schwartz

---

Supplemental Experimental Procedures

Participants

Twelve healthy male volunteers gave informed consent to participate in this study according to the ethical regulations of the Geneva University Hospitals. Female participants were not included because of the effects of menstrual cycle on EEG and sleep [1]. All participants included in the study were good sleepers, with normal and regular sleep-wake habits, and were non-habitual nappers (taking a short nap less than once per week in the last two years). None of them suffered from excessive daytime sleepiness as assessed by Epworth sleepiness scale [2]. They had no psychiatric or neurological history, had never suffered from any vestibular disorder, and did not take any medication during the whole experimental period. The data from two participants had to be excluded before performing the analyses: one because of elevated anxiety before one experimental session preventing him from falling asleep (in the stationary condition), and the other one because of technical problems with the EEG recording. The remaining 10 participants had a mean age of 30.1 (range of 22-38 years) and had low anxiety levels, as assessed by State Trait Anxiety Inventory [3] (mean score ± s.d.; 32.56 ± 3.68). Sleep quality and quantity were assessed by self-rated sleep questionnaires over 3 consecutive nights before each experimental session. Wrist actimetry was also recorded during the last night preceding each experimental session.

Protocol

The experimental procedure consisted of a 45-minute afternoon nap spent in a custom-made bed that could either remain stationary or rock gently (Figure 1A in main text). This bed was suspended by four metal rods to a metallic frame and connected to an electrical motor that produced sinusoidally-modulated horizontal accelerations. The electrical engine and the experimental bed were built to be silent (adding only 2.5 dB to background noise). During pilot testing, we selected a set of motion parameters that generated stimulation while minimizing physical discomfort. The optimal parameters were obtained for total lateral excursion of 10.5 cm amplitude at the level of the bed and a swinging frequency of 0.25 Hz. Data recorded with an accelerometer (sampling frequency: 500 Hz; 3D motion tracker; Xsens MTx, Netherlands) on the bed and on the participants' head measured a peak horizontal acceleration of 0.1 m/s^2 (g load = 0.01) and confirmed a negligible (non-detectable) vertical acceleration.

The nap protocol consisted of two sessions: one with the bed stationary, one with the bed put in motion. The order of the experimental conditions was randomized across participants and the two sessions were at least one week apart. Time in bed from lights off (2:30 PM) to lights on (3:15 PM) was controlled by the experimenters. The naps were spent in complete darkness and the temperature of the sleep room was controlled (21° ± 1° C). During the stationary condition, the motor was turned on, but disconnected from the bed, so that both conditions included the same level of auditory stimulation (37 dB in each condition). In these conditions, auditory input can be excluded. Visual input can be discounted since in both controls and experimental conditions subjects closed their eyes closed and the lights were off. During both

sessions, polysomnography data were recorded continuously with a sampling rate of 1024 Hz, a high-pass filter at 0.5 Hz, and low-pass at 70 Hz (Vitaport3, TEMEC, Netherlands). The montage included 10 scalp electrodes placed according to International 10-20 system (Fz, Cz, C3, C4, Pz, Oz, O1, O2, A1, A2), plus electrooculogram and electromyogram contacts. To keep vestibular inputs due to the swinging motion constant across participants, each participant had his head placed on a soft and comfortable pillow that adopted the shape of the head and slept in a supine position. This setting also minimized movement artifacts. Before the experiment, the participants were told that they should bring comfortable clothes for the nap. Bedcovering consisted of a bed sheet and a blanket. Prior pilot experiments were performed to ensure that the experimental conditions were comfortable. During the main experiment, a careful debriefing of each subject at the end of the experiment confirmed that the sleeping conditions, including the imposed sleeping position and swinging parameters, were judged as comfortable and pleasant by all the subjects. One subject who experienced anxiety during one of the session was not included in the analysis (see above). None of the subjects reported difficulties falling asleep, as also supported by short sleep latencies in both conditions.

EEG data analyses

Sleep polysomnography was scored over 30s epochs, according to standard criteria [4], by two experienced scorers blind to the experimental conditions. Several sleep parameters during the naps were determined: latencies to stage N1 (from lights-off) and N2 (from first N1 period), time and percentage of each sleep stage, total sleep time (TST; sum of the time spent in different sleep stages), total sleep period (TSP; total time from sleep onset to final awakening, including intra-sleep wake intervals),

sleep efficiency and number of intra-sleep awakenings (Table S1). Sleep efficiency was defined as TST/TSP x 100. Sleep spindles were visually quantified at Cz contact referenced against mastoid channels, based on their typical fusiform morphology, frequency of 11-16 Hz, minimum duration of 0.5 s, and minimal amplitude of 10 μV [4] (Table S1). Scoring agreement for both sleep stages and spindles was greater than 95%, and points of disagreement were resolved by mutual agreement between scorers.

EEG spectral analysis was applied at the midline frontal and midline parietal sites (Fz and Pz) on stage N2. Fast Fourier transform was performed on average, 25% overlapping, 10 s windows, with a Hanning window, free of artefacts resulting in a frequency resolution of 0.1 Hz. Values below 0.6 Hz and above 25 Hz were omitted. Analyzes were performed using the Cartool software by Denis Brunet (http://brainmapping.unige.ch/Cartool.htm). Mean power in the following bands was calculated: slow oscillations (0.6-1 Hz), delta 1 (1-2 Hz), delta 2 (2-4 Hz), theta (4-8 Hz), slow spindles (8-12 Hz), fast spindles (12-15 Hz), and beta (15-25 Hz). Comparisons between rocking and stationary conditions were performed using paired 2-tailed t-tests.

Supplemental Results

Spindle power and spindle characteristics

Marshall et al. (2006) found that slow oscillation stimulation simultaneously enhanced slow oscillation, spindle counts, as well as EEG power within the slow spindle frequency range (8–12 Hz) at the frontal location (Fz), but within the fast spindle frequency range (12-15Hz) at the parietal location [5]. We investigated this issue in two ways. We applied the same FFT approach than Marshall et al. (2006). We found

that while Fz showed significant effect of swinging on the slow spindle band (8-12 Hz, $P<0.05$), no such effect was observed on Pz neither in slow (8-12 Hz) nor fast (12-15 Hz) spindle frequency ranges (all $P>0.07$). These results are therefore consistent with those found by Marshall et al. (2006) when applying slow oscillation transcranial stimulation.

We also tested for any change in the peak frequency of the spindles themselves by extracting the frequency for each spindle in each condition for Fz and for Pz. We performed an ANOVA with condition (swinging, stationary) and electrode (Fz, Pz) as repeated measures and found an effect of electrode ($p=0.011$), reflecting slower spindles at Fz, but no effect of condition and no interaction. From these results we conclude that swinging affected the frequency power (at frontal electrodes) but did not lead to a shift of the peak spindle frequency.

Supplemental References

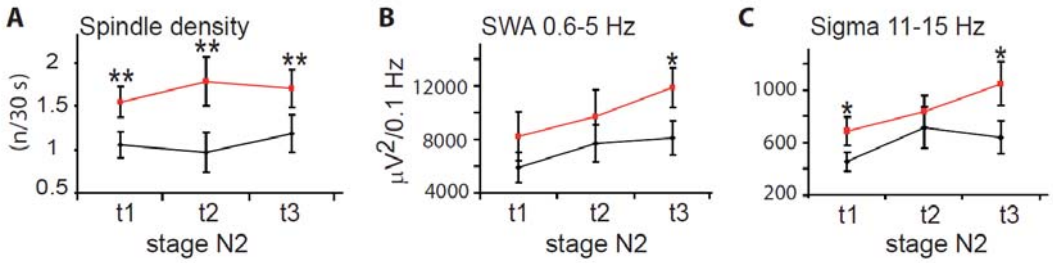
1. Baker, F.C., and Driver, H.S. (2007). Circadian rhythms, sleep, and the menstrual cycle. Sleep Med *8*, 613-622.
2. Johns, M.W. (1991). A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep *14*, 540-545.
3. Spielberger, C.D. (1983). Manual for the State-Trait Anxiety Inventory, (Palo Alto, California: Consulting Psychologist Press Inc.).
4. Iber, C., Ancoli-Israel, S., Chesson, A., and Quan, S.F. (2007). The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology

and Technical Specifications, (Westchester, Illinois: American Academy of Sleep Medicine).

5. Marshall, L., Helgadottir, H., Molle, M., and Born, J. (2006). Boosting slow oscillations during sleep potentiates memory. Nature *444*, 610-613.

Supplemental Figure

Fig. S1



Supplemental Figure Legend

Fig. S1. Time-course of (A) spindle density (mean of spindle per 30 s epoch) during N2, as well as (B) SWA (0.6-5 Hz) and (C) sigma activity (spindle range, 11-15 Hz) at the midline frontal electrode (Fz) during N2. Data from each subject were split into 3 successive equi-duration periods (T1-T3) of time spent in N2 and t-tests comparing the swinging and stationary conditions were performed at each time point (N=10 for each time-point). * $P < 0.05$ ** $P < 0.005$

Table S1. Sleep parameters in each experimental condition (n=10, mean ± s.e.m)

	Bed stationary	Bed swinging
Latency to stage N1 (min) [a]	8.75 ± 2.45	7.75 ± 1.48
Latency to stage N2 (min) [b] *	8.85± 2.05	5.35± 1.56
Stage N1 (min) *	12.4 ± 1.61	7.9 ± 1.17
Stage N2 (min) *	12.2 ± 1.94	16.9 ± 1.04
Stage N3 (min)	0.3 ± 0.3	0.87 ± 0.52
Total sleep time (min)	24.9 ± 3.07	25.67 ± 1.31
Total sleep period (min)	34.05 ± 2.61	34.85 ± 1.74
Awakenings nb	5.45 ± 1.08	4.85 ± 0.9
Sleep efficiency	73.1 ± 5.74	73.65 ± 2.78
Spindles :		
- Total number*	29.15 ± 6.27	57.5 ± 9.27
- Density (per 30s)*	1.20 ± 0.57	1.64 ± 0.70
- Frequency (Hz)	12.41 ± 0.49	12.96 ± 0.69
- Amplitude (μV)	36.38 ± 3.6	35.48 ± 3.48
- Duration (s)	1.06 ± 0.19	1.09 ± 0.42
- Proportion of spindles preceded by a K-complex	0.17 ± 0.03	0.24 ± 0.03

All measurements are reported as mean ± s.e.m.

* significant difference between bed stationary and swinging, t-student, d.f.=9, all $P < 0.001$ except for latency to stage N2 $P < 0.01$

[a] calculated from lights-off

[b] calculated from first N1 period

Note that mean alpha frequency of the population was within normal ranges: 9.68 +/- 0.77 Hz