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RESEARCH ARTICLE

The effect of extended wake on postural control in young adults

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Abstract The sleep–wake cycle is a major determinant of locomotor activity in humans, and the neural and physiological processes necessary for optimum postural control may be impaired by an extension of the wake period into habitual sleep time. There is growing evidence for such a contribution from sleep-related factors, but great inconsistency in the methods used to assess this contribution, particularly in control for circadian phase position. Postural control was assessed at hourly intervals across 14 h of extended wake in nine young adult participants. Force plate parameters of medio-lateral and anterior–posterior sway, centre of pressure (CoP) trace length, area, and velocity were assessed with eyes open and eyes closed over 3-min periods. A standard measure of psychomotor vigilance was assessed concurrently under constant routine conditions. After controlling for individual differences in circadian phase position, a significant effect of extended wake was found for anterior–posterior sway and for psychomotor vigilance. These data suggest that extended wake may increase the risk of a fall or other consequences of impaired postural control.

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Introduction

The sleep–wake cycle is the most conspicuous behavioural rhythm in humans. The major biological determinants of sleep–wake behaviour have been described in models, such as Borbély's 2-process model of sleep propensity (Borbely 1982), as an additive interaction between the core circadian rhythm and a homoeostatic drive to sleep. These components variably impact on a wide range of physiological processes (Duguay and Cermakian 2009), and on a wide range of performance tasks (Van Dongen and Dinges 2003). The two components can be manipulated through simple strategies such as increasing sleep deprivation (Lim and Dinges 2008), or through very sophisticated strategies such as forced desynchrony protocols (Dijk and Czeisler 1994) designed to differentiate the relative contribution of each component. The impact of the sleep–wake cycle, and particularly of extended wake periods, has been described for simple and complex reaction time tasks (Lisper and Kjellberg 1972; Cajochen et al. 1999), cognitive throughput tasks (Jewett and Kronauer 1999), and tasks of 'executive function' (Manly et al. 2002; Nilsson et al. 2005; Tucker et al. 2010). The general pattern of performance during extended wake, for example, on the psychomotor vigilance task (Dorrian et al. 2005), is curvilinear (Graw et al. 2004) with poorest performance typically around the circadian temperature nadir (Wyatt et al. 1999). The potential consequences of impairment in these skills may be seen, for example, in the high rate of severe road crashes associated with sleepiness (Connor et al. 2002); however, the peak in fatigue-associated accidents appears to occur

some hours before peak sleep propensity, suggesting differential impact on specific skills (Williamson et al. 2011).

There are strong indications of a link between disturbed sleep and impaired balance at the population level. For example, sleep disturbance and subsequent daytime sleepiness has been identified as a potential risk factor for falls in older adults (Brassington et al. 2000; Kawamoto and Doi 2002; Avidan et al. 2005; Stone et al. 2006; Hill et al. 2007), but the potential mechanisms underlying this relationship are uncertain. The impact of the sleep–wake cycle on fundamental mechanisms associated with postural control may be critical. A number of laboratory-based studies have examined the effects of sleepiness, extended wake, and circadian rhythm variation on specific components of postural control derived from computerised posturographic tests, typically utilising force plates (Schlesinger et al. 1998; Nakano et al. 2001; Gribble and Hertel 2004; Fabbri et al. 2006; Morad et al. 2007; Gomez et al. 2008; Patel et al. 2008; Ma et al. 2009; Bougard et al. 2010; Robillard et al. 2011), including a thorough series of studies by Forsman and colleagues (Haeggstrom et al. 2006; Forsman et al. 2007a, b, c, 2008, 2010a; b; Tietavainen et al. 2007). These studies support an association between time awake and specific indices of postural control, including centre of pressure (CoP) *velocity* (Gribble et al. 2007), CoP *area* (Bougard et al. 2010), CoP *anterior–posterior range* (Robillard et al. 2011), and others. While some studies have attempted to distinguish specific time-of-day effects (Bougard et al. 2010), in most cases they provide no experimental or statistical control for individual differences in circadian phase. This aspect may be important, as individual participants included in these studies may vary in their circadian phase position by several hours. Circadian phase position is associated with significant variation in a wide range of physiological processes, including core body temperature, hormone excretion, and some aspects of gross motor performance (Jasper et al. 2009). As such time-of-day, or clock time, may be less biologically meaningful than individual circadian phase position. Further, sampling resolution in previous studies has varied from 2-hourly across 36 h to repeat measurement before and after 24-h of wake (Ma et al. 2009) or more irregular sampling (Gribble et al. 2007; Morad et al. 2007). Despite these limitations, differential impact of sleep homoeostasis (Robillard et al. 2011) and circadian rhythm components (Haeggstrom et al. 2006) are suggested. A single forced desynchrony study has assessed postural control at different points of the circadian cycle after different durations of prior wake with CoP area during 1 min of quiet standing (Sargent et al. 2010). This study suggested that neither circadian phase nor duration of prior wake (up to 17 h) had a significant impact on postural control. Our aim was to assess the impact of extended wake on postural control, with sampling at hourly intervals to assess nonlinear variation in balance parameters, with regard to individual variation in circadian phase position, and with convergent measurement of objective vigilance with a psychomotor task.

Materials and methods

Participants

The sample consisted of 3 male and 6 female university students aged 18–25 years ($M = 21.56$, $SD = 2.51$). Height and weight ranged from 164 to 175 cm and 51–70 kg ($M = 169.33$ and 59.78, $SD = 3.97$ and 7.58), respectively. The participants were recruited through word of mouth, with fourth-year psychology students asked to participate.

Exclusion criteria

Participants were excluded if they were currently sick, consumed drugs that could impede balance abilities (e.g. drugs with a sedative effect), engaged in transmedian travel and/or shiftwork 1 week prior to the study, excessive daily caffeine consumption (>4 cups), sleep difficulties (Pittsburgh Sleep Quality Index Score >5 (Buysse et al. 1989), sleep disorder diagnoses, and self-reported balance problems, physical conditions, and/or injuries to feet, ankles, legs, and arms (which could impede balance).

The study was approved by the Queensland University of Technology ethics committee (EC00171; approval 0900000337), and informed consent was provided by all participants.

Measures

Standing balance

Participants stood quietly, their barefeet positioned with heels 15 cm apart at 30° angle, arms at their sides on a force plate (HUR laboratory force platform, Model BT4, Tampere Finland) with eyes open and closed. Centre of pressure changes in the anterior–posterior (AP) and medial–lateral (ML) directions were recorded over a duration of three minutes at a sampling rate of 100 Hz. The CoP changes in motion were used to derive additional balance parameters: trace length (total distance of CoP change during a trial), C90 area (mm^2; fitted ellipse in which 90 % of the trace length points fit into), and velocity. Higher values reflect greater balance instability. Data were acquired for analysis with Finsole Orthotic Analysing suite, version 2.0.

Perceptual psychomotor vigilance task (PPVT; Mueller 2010)

The computer-based PPVT (version 0.07) from the PEBL psychological test battery (version 0.1) was a visual attention task used to measure arousal and sleepiness. In line with conventional sleep deprivation research (Dinges and Powell 1985), the intervals between stimuli presentation ranged from 1 to 10 s. A block approach was used for this study. Each inter-interval stimulus interval (ISI) was presented eight times, was sampled without replacement and randomly presented, and resulted in 80 RTs for each trial block. This task required participants to press the space bar as quickly as possible when the stimulus (red circle) appeared on the computer screen. RTs in milliseconds were shown on screen following each response. A false start was recorded if the space bar was pressed prior to the presentation of the stimulus, and this was counted as a 'too fast' trial. Reaction times exceeding 5 s were considered to be a lapse. Lapses in particular are sensitive to the effects of sleep deprivation and circadian effects on performance (Rajaraman et al. 2008).

Actigraphy

Habitual sleep–wake behaviour was assessed using Actigraphy (wristwatch style recording accelerometers; Minimitter-Respironics Actiwatch II) for 1 week prior to attending the laboratory. Sleep indices, including daily sleep onset and wake times, were identified from the objective activity count data (Actiware 5.2 software). Actigraphy typically has good concordance with polysomnographically determined sleep (Chesson et al. 2007), but has an advantage for longer-term behavioural assessment.

Procedure

Participants attended the laboratory to complete the screening measures and study induction. They were fitted with an actigraph on their nondominant wrist and took the Pittsburgh Sleep Diary (*PghSD*; Monk et al. 1994) home with them to record sleep–wake times for a 6-day period prior to an extended wake testing day in the laboratory. Participants awoke at 5:30 am on that day, arrived at the laboratory at 5 p.m. in the evening, and commenced hourly testing of balance and PVT from 6 pm (12½ h since wake time) until 7 am the next morning (22 ½ h since wake time).

During the extended 14-h wake period, hourly measures were made of postural sway and the 10-min computer-based PVT task. Food consumption was restricted to hourly 500 kj aliquots, and lighting was standardised in both the testing and waiting areas to <40 lux at eye level (Gossen Mavolux 5032B digital luxmeter), similar to other constant routine protocols (Smith and Eastman 2009). Caffeine and alcohol consumption was not permitted, and participants watched DVDs when not being tested. Screen brightness was reduced to minimum settings on the monitors, and illumination from this source was measured at <15 lux at eye level from the normal viewing distance of approximately 1 metre.

Statistical analyses

Circadian phase position for each participant was estimated from their individual sleep–wake times recorded by Actigraphy and cross-checked with the Pittsburgh Sleep Diary reports (Monk et al. 1994). The mean of habitual wake time (but not sleep onset time) provides robust prediction of dim-light melatonin onset (DLMO) and hence the circadian core body temperature nadir (Martin and Eastman 2002; Burgess et al. 2003; Crowley et al. 2006; Bjorvatn and Pallesen 2009). Mean wake time for each sleep period was therefore calculated for the prior 7-day period for each participant. Participant data were then re-binned into hours since mean habitual wake time (from 10 to 23 h posthabitual wake) for further analysis.

To test whether postural sway parameters would vary across the 14 h of extended wake, a 2 × 14 repeated-measures ANOVA was conducted for each of the five dependent variables (ML sway, AP sway, trace length, C90 area, and velocity). To control for family-wise error, a Bonferroni adjustment was applied. There were two levels of the eye condition factor (open or closed) and 14 levels of the time factor. The main effects of eye condition and time are reported in Table 1, with conservative Greenhouse–Geiser-corrected estimates of degrees of freedom.

Results

A significant main effect of eye condition (open or closed) was found for the majority of the balance metrics (apart from AP sway; Table 1), in the direction of increased deviation with eyes closed. A main effect for the time condition was found only for AP sway. The pattern of this variation in AP sway was assessed with planned contrasts (simple), with each hourly score compared to the baseline start score. Planned contrasts revealed significant increase in AP sway with increasing wake duration for both the eyes open and eyes closed conditions, with the pattern of change described in Fig. 1a.

Participants varied in their habitual sleep duration, and the fixed wake time of 5:30 am also meant that sleep duration on the night prior to the study was restricted to a varying degree between individuals (from 4.2- to 7.1-h sleep), depending on their habitual sleep onset and wake

Table 1 Repeated-measures ANOVA: main effects of eye condition and time

Source	*df*[a]	*F*	*p*	EF (partial η^2)
ML				
Eye condition	1	0.98	0.76	0.01
Time	4.62	1.06	0.39	0.11
AP				
Eye condition	1	3.8	0.086	0.323
Time	3.21	3.9	**0.017**	0.331
Trace length				
Eye condition	1	45.26	**<0.001**	0.85
Time	2.3	0.51	0.64	0.06
C90 area				
Eye condition	1	15.14	**0.005**	0.65
Time	1.5	1.94	0.19	0.19
Velocity				
Eye condition	1	45.3	**<0.001**	0.85
Time	2.3	0.51	0.63	0.06
PVT lapse				
Time	13, 104[b]	8.56	**<0.001**	0.59
PVT false start				
Time	13, 104[b]	1.73	0.071	0.22

Bold values indicate statistical significance of $p < 0.05$

[a] Greenhouse-Geiser adjusted

[b] Sphericity assumed

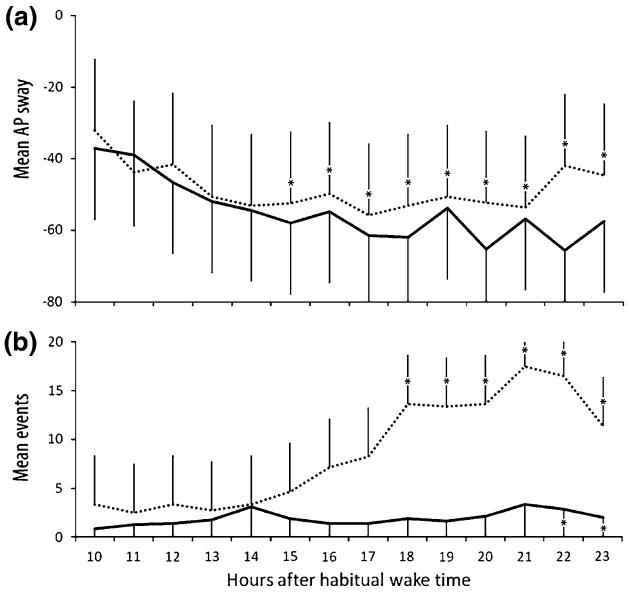

Fig. 1 **a** Variation in mean AP sway (mm from centre) across 10–24 h extended wake for eyes open (*solid line*) and eyes closed (*dashed line*) conditions. Note reversed scale. **b** Variation in the mean number of false starts (*solid line*) and lapses (*dashed line*) across the same period. *Error bars* represent standard error of the mean. Significant differences from baseline are indicated with *asterisks*

times. The relationship between duration of prior sleep and balance variables was assessed with Pearson correlation to determine eligibility as covariates. Correlation coefficients (r^2) varied between 0 and 0.28, all nonsignificant, and prior sleep was not included in further analyses as a covariate.

Variation in the PVT metrics of lapses and false starts across the extended wake period was assessed with one-way repeated-measures ANOVA with a Bonferroni adjustment. There was a significant main effect of time for PVT lapses (Table 1). To examine the pattern of this variation with time, post hoc simple contrasts were conducted for both (Fig. 1b). A significant increase in both lapses and false starts was observed in the hours 20–24 h after habitual wake (approximately 4–7 am for most participants).

In summary, an effect of extended wake was found only for AP sway, with increased AP sway observed relative to the baseline measure after 15 h of extended wake. A main effect of eye condition was observed for trace length, c90 area, ML sway, and velocity. Except for AP sway, higher values of all the other balance measures, indicative of decreased stability, were found when participants had their eyes closed. PVT lapses, but not PVT false starts, demonstrated a main effect for an increase across 14 h of extended wake.

Discussion

Postural control is a complex physiological process that requires preserved function across a range of specific brainstem and cortical structures (Winter et al. 1990). This process certainly changes abruptly with sleep onset and may be impaired with increased sleepiness associated with extended wake. The impact of extended wake demonstrated in this study was restricted to a single control

parameter (AP sway), but with a potentially large effect on this parameter observed in the small sample. This finding is consistent with the previous findings of time-of-day effects, in particular those of Robillard et al. (2011), and suggests that these effects may be robust. The relationship between time and both PVT measures and AP sway suggests a general effect on psychomotor control. The variation in the PVT measures was also consistent with that observed in other extended wake studies (Smith et al. 2007). The findings partly contradict the findings by Sargent et al. (2010), at least for the single index of AP sway. The duration of posturographic assessment and duration of prior wake used in that study may not have been sufficient to identify degraded postural control, despite very careful manipulation of sleep–wake and circadian function. Conversely, it is recognised that control for circadian phase outside of a forced desynchrony methodology is partly confounded by time awake (as in the current study). Nevertheless, these two factors appear to interact to produce variation in postural control.

Several large prospective studies have identified that postural sway is associated with falls in older people (Lord et al. 2003) and in people with neurological disease (Kerr et al. 2010). While lateral sway has the strongest association with falls risk in these populations, oscillations in the AP plane are consistently reported (Lajoie and Gallagher 2004), including higher AP speed with eyes open (Topper et al. 1993). These measures form the basis of clinical assessment tools for predicting falls in these populations. However, it has been noted that methods used to measure postural sway and analyse sway parameters in clinical populations differ widely (Piirtola and Era 2006), making comparison of the magnitude of effects very difficult. The clinical significance of increased AP sway in young sleep-deprived subjects remains to be determined.

It remains possible that more subtle variation in other postural control indices could be observed in a larger sample, after more extreme sleep restriction (e.g. chronic partial sleep deprivation), or in special populations such as older adults. It is also possible that some participants in the current study could 'rally' to the relatively brief performance tasks, especially as standing itself has a transient alerting effect (Bonnet and Arand 1999). The light levels experienced during the study may have been sufficient to suppress or delay expression of melatonin in the evening (Gooley et al. 2011; Zele et al. 2011) with concomitant increase in alertness relative to normal dim-light expression (Cajochen et al. 2005). Assessment of postural control with melatonin expression either controlled (e.g. in a constant dim-light protocol) or manipulated (e.g. with exogenous melatonin or bright short-wavelength light) may be necessary to determine these effects. Continuous assessment of postural control (e.g. via measurement of gait) could provide a more sensitive measure with specific workplace validity, as could a 'dual-task' paradigm (Woollacott and Shumway-Cook 2002) requiring effort to be oriented to an executive function task. Higher resolution of homoeostatic and circadian contributions could be achieved with a forced desynchrony protocol or other unmasking strategies, or with more refined measurement with direct melatonin or core body temperature measurement.

A recently recognised issue in sleep and circadian rhythm research is that of individual differences in sensitivity to sleep loss (Galliaud et al. 2008). This variation may be due to a range of factors, including differences in clock genetics, body mass, and age. Intra-individual differences in muscle tone and 'fitness' may also contribute to individual difference in postural control, even in a healthy young sample. Multi-level modelling approaches have been proposed to assess such differences (Kristjansson et al. 2007), but studies using this approach would require quite large samples. Despite these considerations, this study provides further support for the impairment of postural control after extended wake and is consistent with circadian variation in this control.

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