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Effects of increased homeostatic sleep pressure on postural control and their modulation by attentional resources

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HIGHLIGHTS

- First evidence that increased sleep pressure has a direct destabilizing effect on postural control without any manipulation of attentional or sensory resources.
- Supports the hypothesis that attentional resources modulate the relation between sleep and postural control.
- Results suggesting that psychomotor speed mediate the influence of sleep deprivation and cognitive load on postural control.

ABSTRACT

Objective: This study aimed to determine how increased sleep pressure interferes with postural control according to the availability of attentional resources and visual input.
Methods: Thirteen healthy young adults performed a psychomotor vigilance task and postural tasks after a night of sleep and after 25 h of sleep deprivation. Primary outcome variables were calculated from the center of pressure (CoP) displacement measured by two force plates in various cognitive load and visual state conditions.
Results: Sleep deprivation increased CoP anterior–posterior range in the no cognitive load condition and decreased CoP mediolateral range and velocity in the high cognitive load conditions. Sleep deprivation effects on the mediolateral range in the eyes open high cognitive load condition were significantly correlated with its effects on the psychomotor vigilance task.
Conclusions: Sleep deprivation destabilizes postural control when attentional and sensory resources are not challenged. In high cognitive load condition, sleep loss induces a general freezing effect that seems to be modulated by the degree of impairment in psychomotor speed.
Significance: This study demonstrates that sleep pressure has a destabilizing effect on postural control independently of circadian factors, therefore suggesting that sleep debt may be a significant risk factor for falls.

1. Introduction

To maintain its balance, the body constantly produces reaction forces under the feet to counteract the movements of the center of mass (Gage et al., 2004). The center of pressure (CoP) is the central application point of the reaction forces. As it constantly moves around the center of mass to maintain the body's balance, dynamic parameters of the CoP displacement are commonly used to characterize postural control. Importantly, different configurations of these parameters reflect different postural states. For instance, wide, fast, and disorganized CoP displacement increases the likelihood of crossing postural stability boundaries, and is therefore commonly interpreted as reflecting a more unstable state. Accordingly, increased range, speed, and variability of CoP displacement during quiet standing have all been associated with increased risks of falls (Berger et al., 2005; Fernie et al., 1982; Maki et al., 1994). On the other hand, CoP displacement that is slow, narrow, and having low variability indicate an overly rigid or stiff postural control that is likely to reduce sensory feedback and the ability to adjust for external disturbances. Postural control is thought to operate through distinct mechanisms in the anterior–posterior (AP) and mediolateral

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(ML) direction. CoP displacement along the AP axis is controlled mainly by the ankle muscles, whereas displacement along the ML axis is controlled mainly by the hip muscles (Day et al., 1993; Winter et al., 1996). Studies also suggest that unchallenged quiet stance relies mainly on the AP muscle group (Winter, 1995).

It has been proposed that extended wakefulness impairs postural control (Avni et al., 2006; Fabbri et al., 2006; Gribble and Hertel, 2004; Liu et al., 2001; Nakano et al., 2001; Patel et al., 2008; Schlesinger et al., 1998). Sleep debt may therefore be a risk factor for accidents caused by loss of balance. Contemporary models propose that sleep–wake cycle regulation operates through two interactive but distinct processes: the circadian process, which induces the rhythmic variation of sleep propensity over 24 h, and the homeostatic process, by which sleep pressure accumulates with increasing time awake and dissipates during sleep (Borbély, 1985). Some studies showed that prolonged wakefulness increases the variability, speed, and area of CoP during upright stance (Fabbri et al., 2006; Gribble and Hertel, 2004; Liu et al., 2001; Nakano et al., 2001). However, these studies assessed postural control at different times during a night of sleep deprivation without including a baseline postural evaluation after a normal night of sleep. Thus, it was not possible to evaluate whether the effects of constant wakefulness on postural control were produced by higher circadian or homeostatic sleep pressure. Many studies showed circadian variations in postural control mechanisms (Avni et al., 2006; Kohen-Raz et al., 1996; Nakano et al., 2001; Patel et al., 2008), but only a few evaluated the effects of higher homeostatic sleep pressure on CoP displacement while controlling for circadian time. Those studies reported a significant effect of sleep deprivation, but only when attentional resources or sensory inputs were modulated (Schlesinger et al., 1998; Patel et al., 2008). Unfortunately, these studies used different postural parameters and experimental manipulations, making their results difficult to reconcile.

Traditionally, upright stance was thought to be controlled mainly by automatic reflexes. However, studies in the mid-eighties challenged this idea by revealing the close interplay between the attentional and postural control processes (Kerr et al., 1985; Maki and McIlroy, 2007; Woollacott and Shumway-Cook, 2002). According to limited resources theories of attention (Kahneman, 1973; Neumann, 1996; Woollacott and Shumway-Cook, 2002), when two tasks relying on the same attentional resources are performed simultaneously, their individual attentional demands are summed. When the summation exceeds the global available attentional capacity, interference occurs, causing decreased performance in at least one of the tasks. This theory implies that if performance is lowered in either one of the tasks when someone is simultaneously performing a postural and a cognitive task, both the postural and cognitive processes compete for the same attentional resources. Accordingly, studies showed that young adults make more errors and perform more slowly on cognitive tasks with increasing postural challenge (Kerr et al., 1985; Lajoie et al., 1993) and regulate their posture more stiffly when performing cognitive tasks (Dault et al., 2001a,b; Siu and Woollacott, 2007). Studies that evaluated postural parameters in the AP and ML directions reported that CoP range and frequency (i.e., proportional to CoP Speed) along the AP axis are particularly sensitive to the effects of cognitive tasks (Dault et al., 2001a,b; Pellecchia, 2003). Because sleep loss impairs attention (for a review, see Lim and Dinges, 2008), it might reduce the attentional resources available for postural control. This hypothesis is supported by a small sample study suggesting that the effects of sleep deprivation on CoP displacement variability appear only when a concurrent cognitive task is performed (Schlesinger et al., 1998).

Sensory inputs also play an important role in the regulation of upright stance. Vision allows online processing to adjust information about body movements and the representation of spatial environments. Two main mechanisms of visual feedback to the postural control system were recently defined (Guerraz and Bronstein, 2008). During upright stance, the constant body oscillations generate subtle movements of the visual scene in the retina. A copy of the motor commands allowing the extraoccular muscles to adjust for these movements is also sent back to the central nervous system. Visual input is thought to be a primary source of sensory information that is integrated into the postural control system, and to have a stabilizing effect on postural control (Travis, 1945). The range, speed, and RMS amplitude of CoP displacement have all been shown to increase in conditions of visual deprivation, especially in the AP direction (Paulus et al., 1984; Day et al., 1993; Prieto et al., 1996; Nougier et al., 1997; Uchiyama and Demura, 2007). Interestingly, these effects are gradual, increasing with the degree of visual impairment, and the AP axis is more sensitive to gradual reductions in visual acuity (Edwards, 1946; Paulus et al., 1984). Because it alters the visual system (Clarke and Warren, 1939; De Gennaro et al., 2000; Horne, 1975), sleep deprivation might impair visual information integration in postural control. Accordingly, a recent study suggested that postural control may be more sensitive to alterations in visual input after sleep deprivation (Gomez et al., 2008).

Additionally, sleep deprivation effects on postural control could result from alterations in muscular and nerve functions. Notably, sleep deprivation is known to induce musculoskeletal stiffness (Moldofsky et al., 1975) and to reduce muscle sympathetic nerve activity (Kato et al., 2000), two factors that could potentially affect the range, speed, and variability of CoP displacement.

The present study had two main objectives: (1) to evaluate the effects of increased homeostatic sleep pressure at the same circadian phase on the main components of postural control dynamics (scale, speed, and variability); and (2) to assess, using a within-subject design, possible interactions between sleep deprivation, attentional resources, and the integrity of visual input. We hypothesized that: (1) in unchallenged quiet stance condition, sleep deprivation would have a destabilizing effect on postural control by increasing CoP scale, speed, and variability; (2) this effect would be amplified when visual input is altered for all three postural parameters; and (3) in high cognitive load condition, sleep deprivation would increase CoP range and speed.

2. Methodology

2.1. Subjects

Thirteen healthy subjects participated in this study (6 women and 7 men; 25 ± 2.7 years). All of them spoke French. Exclusion criteria were uncorrected visual impairment; use of medication known to influence sleep or postural control; sleep complaints; history of auditory, postural, vestibular, psychiatric, or neurological disorder; night work or transmeridian travel three months prior to the study; or body mass index higher than 30. All subjects had a score lower that 10 on the Beck Depression Scale (long version; Beck et al., 1996). Blood sample analysis (complete blood count, serum chemistry comprising hepatic and renal functions, and prolactin levels) and urinalysis results were checked by a certified physician to rule out significant medical conditions. Prior to data acquisition, subjects underwent a polysomnographic (PSG) adaptation and screening night. The presence of sleep disturbances such as sleep apnoeas and hypopnoeas (index per hour >10), periodic leg movements (index per hour >10), prolonged sleep latency (>30 min), or low sleep efficiency (<80%) resulted in the participant's exclusion. All subjects signed an informed consent form and received monetary compensation. All research studies in which they participated were approved by the Hospital's Ethical Committee.