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Circadian amplitude and homeostatic buildup rate in postural control

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ABSTRACT

Postural control during quiet stance is a common everyday physical activity. Sleepiness is increasingly prevalent in our 24-h society. Yet, little research exists that quantitatively links the fluctuations in sleepiness and postural control. This study quantifies the circadian amplitude and homeostatic buildup rate in postural control. With a force plate we assessed postural control in 12 participants (21–38 years) every 2 h during 24 h of sustained wakefulness. The *sway area* was 1.39 ± 0.71 mm^2 at the circadian high around noon, and 4.02 ± 0.67 mm^2 at the circadian low around 6 am (a 189% change, $p = 0.02$). The circadian amplitude of the *sway area* was therefore 2.63 mm^2. The *sway area* was 1.92 ± 0.64 mm^2 at the start of the 24-h period and 4.42 ± 0.69 mm^2 at the end of the period (a 130% change, $p < 0.001$). The homeostatic buildup rate of *sway area* was 0.04 h^{-1}. The circadian- and homeostatic effects on *sway variability*, *sway velocity*, *sway frequency* and *fractal dimension* were smaller but still significant. This study found that the circadian amplitude and homeostatic buildup rate are quantifiable from posturographic data, and that they have significant impact on postural control. This finding is important because it means that one could apply the framework of the famous two-process model of sleep regulation (published by Borbély in 1982) to explain the previously reported sleepiness-related changes in postural control.

1. Introduction

The circadian amplitude A and homeostatic buildup rate ρ are free parameters in the two-process model of sleep regulation (see Eq. (1)) [1,2]. One determines A from the range between the circadian high and low in performance that the circadian process causes [1,2]. One determines ρ from the rate with which performance is impaired during wakefulness [1,2]. Sleep researchers often quantify and employ A and ρ in the two-process model of sleep regulation to explain impaired psychomotor vigilance [3] as well as traffic accidents [4–6].

Falls can cause injuries that range from cuts and bruises to bone fractures and head injuries [7,8]. Therefore, countermeasures against risk factors for falls have received increased attention during the last couple of decades [9–11]. Several studies show that sleepiness from circadian timing [12–15] and from sleep loss [16–24] influence postural control. In a literature search we found that postural control parameters can change by 21% during daytime hours [12,13], and that moderate sleep loss can change postural control parameters by 18% [16,17]. Importantly, recent research indicates that sleepiness is a risk factor for falls [25,26]. Predicting sleepiness-related changes in postural control could therefore facilitate countermeasuring some fall incidences [7,13,17].

Our 24/7 society has an ongoing need to develop different strategies that help to safeguard against sleepiness-related accidents. For example, work safety officials use mathematical models to predict task-specific performance when they design and compare the feasibility of different shift schedules [6,27]. Car makers are developing technologies to monitor car-based parameters of driving performance and warn the driver of impending sleepiness [28–30]. In fatigue risk management, which could include the aforementioned shift scheduling or warning technologies, knowing the circadian amplitude A and the homeostatic buildup rate ρ of the performance parameter S is relevant, because this allows predicting S with the two-process model of sleep regulation according to [1,2,6]:

$$S = A \sin\left(\frac{2\pi t}{24}\right) + \xi \exp(-\rho T), \quad (1)$$

where t is the clock time relative to midnight, ξ is the initial homeostatic level, and T is the time awake (see [2] for a review of the model). The model accounts for the circadian process, which causes a sinusoidal time of day dependent variation in sleepiness across the 24-h day such that it peaks between 02:00 and 08:00 [1,2,4]. The model also accounts for the homeostatic process, which causes sleepiness to accumulate (and saturate) exponentially with increasing time awake [1,2].

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The two-process model of sleep regulation [1] has successfully predicted sleepiness and psychomotor performance in laboratory- and occupational settings with acute- and partial sleep loss [3,5,31,32]. The model may also apply to postural control, because literature indicates that the circadian- and homeostatic processes, which are intrinsic to sleep regulation [1], are present in postural control as well [12–24]. If the model applies to posturographic data, it would provide a mathematical framework to explain the previously reported sleepiness-related fluctuations in postural control. However, this idea would require that the circadian amplitude and homeostatic buildup rate are quantifiable from posturographic data. As noted above, posturographic research has focused on, and succeeded in, detecting the circadian- and sleep loss-related changes in postural control [12–24]. We extend this work by examining the circadian amplitude and homeostatic buildup rate in postural control.

We set out to examine the circadian amplitude and homeostatic buildup rate in postural control. To do this we tested postural control every 2 h during 24 h of sustained wakefulness. Covering one circadian period was both necessary and sufficient to record the circadian high and low and determine the circadian amplitude [1,2,4]. The repeated testing was necessary to assess the homeostatic buildup rate [1,2,4]. To analyze the circadian effect one needs to regress the data on time of day [2], whereas to analyze the homeostatic effect one needs to regress the data on time awake [2], which we do in this work. From this work, we expect the most important result to be the quantification of the circadian amplitude and homeostatic buildup rate in postural control, thereby identifying a mathematical framework to explain sleepiness-related changes in postural control.

2. Methods

2.1. Participants

Ten men and two women (21–38 years, mean = 26.6) participated in the study after giving their written informed consent. Exclusion criteria were smoking, alcohol abuse, regular medication, and diagnosed musculoskeletal-, postural control-, or sleep disorders. The participants were asked to maintain a regular sleep–wake schedule during the three days preceding the experiment. This assured that they arrived rested to the experiment. Sleep diaries were kept (mean bedtime 23:29 hours (SD 00:55 hours); mean wake-up time 07:20 hours (SD 01:13 hours); mean sleep per night 07:51 hours (SD 01:04 hours)). The bedtimes and wake-up times of two participants were missing.

2.2. Postural control testing

Postural control was measured with a custom-made force plate [33]. During the measurements the participants stood unshod on the plate with their feet together (feet touching from heel to toe) keeping their arms crossed over the chest. Measurements were performed eyes open: the participants were asked to look at a fix point (visual reference) in front of the plate. The experimenter checked the stance at each test occasion. During the 30-s measurements the plate sampled the body center of pressure (COP) excursions at 1 kHz. The data were downsampled to 50 Hz by averaging 20 adjacent datapoints. The postural control parameters were computed from the anteroposterior and lateral directions of the COP-excursions (Section 2.2.1).

2.2.1. Postural control parameters

The COP-excursions were centered by removing the arithmetic means of the time series. Eight postural control parameters were computed (Table 1). The parameters *sway area*, *sway velocity*, and *sway variability* evaluate the stability of postural control [34,35] and were chosen because increased area, velocity, and variability have been associated with an increased risk of falling [9–11,36,37]. The parameters *sway frequency* and *fractal dimension* of sway were chosen because they evaluate the activity of postural control [34,35]. Hereafter ML refers to the centered mediolateral signal, whereas AP refers to the centered anteroposterior signal.

Table 1
Parameter definitions and equations. $N = 1500$ data points, $n = 1, \ldots, N$, $T = 30$ s, k = ML, AP, and $z_{0.5} = 1.645$. Refer to [34,35] for detailed explanations of the equations. $r_n = \sqrt{ML_n^2 + AP_n^2}$, SD denotes standard deviation, d denotes maximum distance between any two points.

Postural control parameter (unit)	Equation
Sway variability, root mean square amplitude (mm)	$RMS_k = \frac{1}{N}\sqrt{\sum_n k_n^2}$
Sway velocity (mm/s)	$VELOCITY_k = \frac{1}{T}\sum_{n=1}^{N-1}\sqrt{(k_{n+1} - k_n)^2}$
Sway area, 95% confidence circle (mm^2)	$AREA_{CC} = \pi(\frac{1}{N}\sum_n r_n + z_{0.5}SD_r)^2$
Sway frequency (Hz)	$FREQ_k = VELOCITY_k/(\frac{4\sqrt{2}}{N}\sum_n k_n)$
Fractal dimension (unitless)	$FD = \log(N)/\log(Nd/(T \cdot VELOCITY_r))$

2.3. CFF testing

Cortical arousal was assessed with a 5-min critical flicker fusion (CFF) test [38]. The CFF test that assesses a person's ability to distinguish discrete sensory data is a simple test of sleepiness: when sleepiness increases, the CFF scores decrease [38]. The subject sat, chin supported, 30 cm in front of a red light emitting diode (LED, ∅ 5 mm). One test comprised 18 sequences, with a 1-min rest after every 6 sequences. During one sequence the LED blinked at decreasing frequencies from 40 to 30 Hz in 10 s. The subject watched the LED and pressed a button when perceiving the LED flicker. CFF scores were expressed in Hz, calculated from the 30% trimmed average of the 18 button pushes (obtained by discarding the three highest and the three lowest CFF scores before calculating the average of the remaining 12 button pushes). The filtering reduces the influence of lapses, micro sleeps, and decision-stage impairments [39]. We used the CFF scores as a reference test of sleepiness to compare with the postural control parameters.

2.4. Protocol

On the Thursday preceding the experiment the participants attended a practice session lasting less than 1 h. During the experiment the subjects were kept awake for 26 h; they slept until 06:00 hours on Friday morning, the experimental protocol began at 08:00 hours on Friday, and lasted until 08:00 hours on Saturday. Every 2 h the participants took a postural control- and a CFF test, 13 sessions in total. Between the sessions they were allowed to read magazines, listen to music, and converse with each other in the common room of the laboratory. They were prohibited from going outdoors. Meals were allowed every 4 h, immediately after a session. Caloric intake was not standardized; the participants chose from the open breakfasts, lunches, and dinners in the laboratory canteen. When the canteen was closed they were allowed rye bread and fruits. They had free access to water but were prohibited from consuming alcoholic-, caffeinated-, and sugary beverages.

2.5. Data analysis

We used the Kolmogorov–Smirnov test with Lilliefors correction to test each parameter for normality. A $p \leq 0.05$ indicated that the parameter was not normally distributed.