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How does one night of sleep deprivation affect the internal clock?

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ABSTRACT

Twelve subjects performed two temporal tasks, one explicit (Experiment 1) and one implicit (Experiment 2) after one night of sleep deprivation and after one night of normal rest. Experiment 1 involved a 1100-ms duration production task, and in Experiment 2 subjects performed a word identification task requiring implicit estimation of vowel duration (around 150 ms). One night of sleep deprivation had the same pattern of effect on explicit timing in the suprasecond range and implicit timing in the millisecond range. Specifically, sleep deprivation induced productions of shorter intervals in the duration production task and estimation of segmental durations as being longer in the word identification task. Both results are consistent with an acceleration of pacemaker rate.

Moreover, in both experiments, we found a correlation between the alertness level of participants and the size of the effect. Therefore, sleep deprivation, which physiologically manipulates cortical arousal level, produced similar performance modulation in suprasecond explicit and subsecond implicit tasks suggesting a common mechanism.

1. Introduction

Humans, as other animals, have the ability to perform behaviorally relevant time measurement across a wide range of intervals (for review, see Buhusi & Meck, 2005). The perception of time in the hundreds of seconds to minutes range is often referred to as interval timing and has to be differentiated from timing in the circadian range. For many years, the prevalent guiding theoretical framework for understanding how we measure the duration of intervals has proposed that we time intervals using an internal clock functioning as a stopwatch, with a clock stage composed of a pacemaker-counter device. An interval is specified by the accumulation of pulses emitted at a regular rate from a pacemaker. The more pulses that are accumulated, the longer the subjective estimation of duration.

One way of studying the clock component of the pacemaker-accumulator type is to attempt to change the rate at which the pacemaker runs. Different manipulations have already been mentioned to affect the speed of pacemaker (for review, see Wearden & Penton-Voak, 1995). In 1933, Hoagland was one of the first who reported that increases in body temperature yield to an acceleration of pacemaker rate. Later, Treisman and collaborators (Treisman, Faulkner, Naish, & Brogan, 1990; Treisman, Faulkner, & Naish, 1992) modeled a temporal pacemaker and proposed that external stimuli influence pacemaker rate, the more arousing the stimuli, the more the pacemaker rate increases. They presented participants with a train of repetitive stimulation (clicks or flashes), whilst they performed a time judgment task, and observed that participants behaved as if their internal clock had increased in speed. Similar results were later obtained by preceding stimulus presentation by a train of clicks in various tasks (Penton-Voak, Edwards, Percival, & Wearden, 1996). The same effect was found whatever the modality (visual or auditory) of the stimuli and it was more marked at longer stimulus durations, which was consistent with an acceleration of pacemaker rate. Burle and Casini (2001) demonstrated that presenting a train of clicks with strong intensity produced greater acceleration of pacemaker rate than presentation of clicks with weak intensity. In all of these experiments, it was supposed that increasing cortical arousal level with entraining sensory inputs speed up the rate of the pacemaker.

Another convenient way of modifying cortical arousal is to use sleep deprivation. Sleep deprivation is known to deteriorate a wide range of cognitive, behavioral and physiological measures (Dinges, 1992; Mertens & Collins, 1986; Pilcher & Huffcutt, 1996). For example, significant effects have been demonstrated on the Sternberg working memory task (Mu et al., 2005), Raven's matrices (Linde & Bergstrom, 1992), verbal fluency (Horne, 1988) and Tower of London (non-verbal planning) (Horne, 1988). Deteriorations in vigilance and activation, which frequently cause industrial or automobile accidents, are also often consecutive to

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sleep deprivation (Connor et al., 2002; Faireclough & Graham, 1999). Very recently, Vyazovskiy et al. (2011) demonstrated that as sleep deprivation increases in rats, the amount of cortical neurons switching from ON state (awake-like activity) to OFF state (NREM sleep-like activity) also increases. But on the contrary, some other recent studies performed in rats or in humans, suggest that instead of decreasing cortical arousal, moderate sleep deprivation increases the excitability of cortical neurons (Huber et al., 2012; Liu, Faraguna, Cirelli, Tononi, & Gao, 2010; Vyazovskiy et al., 2009).

A decrease in cortical arousal level, which slows the pacemaker rate, should manifest as underestimation of subjective time due to fewer accumulated pulses while an increase in cortical excitability should lead to the opposite manifestation, that is overestimation of subjective time due to more accumulated pulses. The objective of the present study was to investigate the effect of sleep deprivation on the internal clock.

To this aim, two different timing tasks were used, involving either explicit (Experiment 1) or implicit (Experiment 2) temporal judgment. As proposed by Coull and Nobre (2008), "the crucial distinction between explicit and implicit timing is whether or not the task instructions require subjects to provide an overt estimate of duration (p. 137)." In the first experiment, participants performed a temporal production task where they had to explicitly time a 1100 ms interval. In the second experiment, participants perform a speech perception task. Speech perception, in which the role of segmental duration is of particular importance, is a good example of an implicit temporal task. Indeed, in many languages, it is observed that the variation in vowel duration depends on whether the following consonant is voiced or voiceless (Kingston & Diehl, 1994). For example, the /ɛ/ lasts longer in *bed* (voiced consonant) than in *bet* (*voiceless consonant*). This context-dependent variation in vowel duration has been shown to influence the perception of post-vocalic consonant voicing: following longer vowels, consonants are more often perceived as voiced than voiceless (Casini, Burle, & Nguyen, 2009; Fischer & Ohde, 1990). Moreover, the segmental durations involved in speech perception are in the range of tens to hundreds of milliseconds, whereas the temporal production task in Experiment 1 required estimation of a 1100 ms duration. In the field of the psychology of time, a distinction is often made between the processing of durations superior or inferior to one second. Some authors propose that time estimation of hundreds of milliseconds to seconds (supra-seconds durations) would be cognitively mediated whereas measurement of tens to a few hundreds of milliseconds (sub-second durations) is supposed to be of a highly perceptual nature and not accessible to cognitive control (Karmakar & Buonomano, 2007; Michon, 1985; Rammsayer & Lima, 1991). However, some behavioral data also suggest that common mechanisms are involved for both short and long durations (Rammsayer & Ulrich, 2005). As a consequence, the issue of timescale specificity is still debated and it appears relevant to check whether sleep deprivation affects the two duration ranges in a similar manner. As the segmental durations involved are around 150 ms, speech perception seems well-suited to tackle this question.

The duration production task and the speech perception task were carried out either after a normal night of rest (rest condition) or after one night of sleep deprivation (deprivation condition).

2. Experiment 1: Temporal production under sleep deprivation

Participants performed a temporal production task either after a rest night or after one sleep deprived night. Moreover, to investigate whether sleep deprivation specifically affects pacemaker rate, we also manipulated the attention level of participants. Since in a previous study (Burle & Casini, 2001) we have demonstrated that attention specifically acts on the switch device, our aim was to verify whether sleep deprivation and attention manipulations do not interact but rather are additive, which would suggest that sleep deprivation affects a different component of the internal clock, presumably the pacemaker rate. Participants therefore performed the temporal production task either as a single task or in a dual-task concurrent with a reaction time (RT) task. We also delivered click trains during duration production, as in Burle and Casini (2001). This would have allowed for the comparison of the effect of sleep deprivation, which tonically modifies brain activation level, to the effect of click trains, which are believed to phasically modify brain activation level. However, due to technical difficulties, the sound intensities applied did not match those intended. As a result, no effect of this variable was obtained even in the rest condition, and we will not refer to it further. Two factors were then crossed for results analysis in the present experiment: sleep conditions (rest or deprivation) and attentional resources (single or dual-task).

2.1. Material and method

2.1.1. Participants

Twelve participants were paid for their participation in the experiment (6 women and 6 men, mean age: 26 years, range: 21–37 years). All participants were volunteers and gave informed consent to the experimental procedure, following the Helsinki declaration (1964). The study was approved by the French Ethical Committee.

2.1.2. Procedure

Participants were seated comfortably in a dimly lit and sound-shielded room, facing a black video screen located 1 m away. A device with two response keys was available. The left index and middle fingers were used for the RT task and the right thumb was used on a single keypress for the duration production task (for a figure of the device, see Burle & Casini, 2001). All stimuli and responses were controlled by a computer running t-scope (Stevens, Lammertyn, Verbruggen, & Vandierendonk, 2006). Time production and RTs were recorded to the nearest millisecond.

Each participant performed two experimental sessions, one after one sleep deprived night and the other after a normal night's rest. Each of these sessions contained six blocks of 50 trials corresponding to two blocks of each task. The three tasks tested were: a 1100-ms duration-production task, a RT-only task, and a dual-task in which the participants simultaneously performed the duration-production task and the RT task. The order of blocks was counterbalanced across participants.

One day before the first experimental session (sleep deprived or rest night session, depending on participants), a training session for each task was performed by each participant in order to obtain reliable performance during experimental blocks. It was also important that subjects were trained on the standard duration and then build a representation of the standard duration before the sleep deprivation.

2.1.2.1. Training session

2.1.2.1.1. Duration-production training. The training session consisted of two parts. For the first five trials, a central red circle appeared on the screen indicating the beginning of the trial. Then a 600 Hz tone sounded during 1100 ms. At the end of the sound, participants reproduced the duration of the sound by pressing the keypress with the right thumb. When participants released the keypress, an auditory feedback was delivered. Five different feedbacks were used. If the produced interval was correct (less

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than 7.5% longer or shorter than the target), the word CORRECT was delivered. If the produced duration was too long or too short (7.5–22.5% longer or shorter than the target), either the words TROP LONG (too long) or TROP COURT (too short) were delivered. If the duration was excessively long or short (more than 22.5% longer or shorter), the words BEAUCOUP TROP LONG (much too long) or BEAUCOUP TROP COURT (much too short) were delivered.

After the first five trials, the word TEST appeared on the screen, indicating that no additional model of target duration would be delivered. During the remaining trials, once the red circle appeared on the screen, participants pressed the key for 1100 ms. As in the first five trials, a visual feedback was delivered following each response. The participants continued until they produced 12 correct durations through 15 successive trials. On average, 104 trials (SD=45) were necessary to reach the criterion.

2.1.2.1.2. RT-only and dual-task training. For the RT task and the dual-task, participants performed one training block of 50 trials. The RT-only and the dual-task conditions were always administered after the duration-production condition to ensure that during the duration-production condition the participants concentrated their attention on the temporal task.

2.1.2.2. Experimental session

2.1.2.2.1. Duration-production task. The participants' task was to maintain a keypress with the right thumb for 1100 ms. Trial onset was initiated by participants once a red circle appeared on the screen. Participants had to maintain the keypress as long as necessary to time the required duration. No feedback on performance was given. Two seconds after the release of the key, the red circle reappeared, indicating that the next trial could be initiated. During the duration production period a central blue circle appeared on the screen, serving as a fixation point (Fig. 1).

2.1.2.2.2. RT only condition. The task of the participants was to respond as quickly as possible to a green circle appearing on the video screen on the right or the left side of a central blue circle.

Trial onset was initiated by the participants, by a brief right thumb keypress whilst a red central circle was displayed on the video screen. As soon as they pressed the right key, the central blue circle appeared, serving as a fixation point, which later disappeared when participants responded. After a variable delay (ranging from 250 ms to 490 ms) following the appearance of the blue circle, one of the two green circles appeared. When the left circle appeared participants had to briefly press a button as quickly as possible with the left middle finger, when the right circle appeared they were required to respond with the left index finger (Fig. 1).

2.1.2.2.3. Dual-task condition. In the dual-task condition, participants were required to produce the 1100 ms target duration and to perform the RT task simultaneously. Each trial was similar to the duration-production condition described except that whilst participants were producing the required duration with the right thumb, one of the two green circles appeared and participants had to respond appropriately as quickly as possible as outlined in the RT-only condition.

2.1.3. Sleep deprivation and rested wakefulness procedures

Prior to the rest condition, participants had a normal night's rest at home. Prior to the deprivation condition, participants were kept overnight at the "Institut de Médecine Navale du Service de Santé des Armées". A research assistant remained with participants throughout the night to ensure they were awake. Access to television and games were provided and participants were aware of being filmed during the night. No food was allowed after midnight and caffeinated beverages were discontinued for 24 h prior to the study.

The two experimental sessions, corresponding to each condition, were separated by 2–4 weeks and each participant performed the two conditions on the same day of the week. Mondays were eliminated to avoid weekend effects. The order of conditions, rest-deprivation or deprivation-rest, was counterbalanced among participants.

To assess alertness level, we recorded electroencephalographic (EEG) activity from Ag/AgCl scalp electrodes during 5 min before

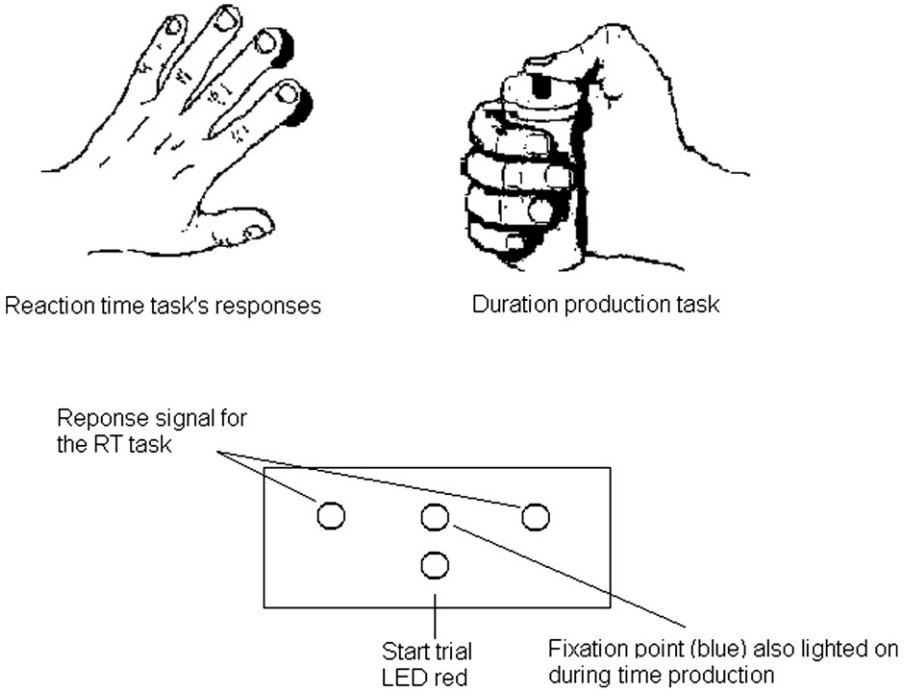

Fig. 1. Experimental apparatus. RT=reaction time; LED=light-emitting diode.

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the experimental session and we identified the sleep stages present in EEG activity of each subject to ensure the efficiency of the sleep deprivation procedure. The total recording time was divided into periods of 30 s and, for each participant, we noted the first period of time in which signs of sleep stages I–III were observed for more than 20 s. Sleep stages I–III were assessed according to the guidelines of the American Academy of Sleep medicine (2007; see also Silber, Ancoli-Israel, & Bonnet, 2007). Scoring was performed by an experienced operator by visual inspection of the whole EEG data set. This operator was not aware of the experimental condition he was scoring (blind scoring).

2.2. Results

2.2.1. Effect of sleep deprivation and rested wakefulness procedures

Table 1 reports the latency of the occurrence of different sleep stages. It can be observed that, except for one participant who presented no sign of sleep in both sessions (more exactly, in the deprivation condition, he presented some signs of sleep, but they never exceeded 20 s and, hence, were not reported in Table 1, whereas in the rest session, he never presented signs of sleep), all participants presented signs of sleep in deprivation condition whereas only two participants presented signs of sleep stage I in rest condition. Sleep stage data demonstrate that participants were clearly more sleepy in deprivation condition than in rest condition, confirming that the sleep deprivation procedure was therefore effective.

2.2.2. Behavioral results

All variables measured in each task were cast in the form of a 2×2 repeated measures analysis of variance (ANOVA). The factors were sleep conditions (rest *versus* deprivation) and attentional conditions (single *versus* dual-task).

2.2.2.1. Duration production task. Mean produced duration and variance of produced durations were calculated for each subject in each of the four conditions. Mean produced duration reflects overall lengthening or shortening of produced time. To aid in the interpretation of results, it should be noted that a shortening of produced durations (also called underproduction) means that the criterion value corresponding to the duration learned during the training session was reached earlier. Conversely, a lengthening of produced durations (also called overproduction) means that the criterion value was reached later. The variance of produced durations provides information about individual participant's variability across trials.

2.2.2.1.1. Mean temporal production. The mean duration produced by each subject in each condition was computed. Fig. 2 shows mean temporal production as a function of sleep condition. Participants produced shorter durations in the deprivation (1424 ms) than in the rest (1565 ms) condition ($F1,11 = 6.52$, $p < 0.05$). Thus, although participants overproduced the target 1100 ms duration in both conditions, they underproduced duration after a sleep deprived night as compared to a night of rest. Participants produced longer durations in the dual-task condition (1586 ms) than in the single-task condition (1403 ms) ($F1,11 = 13.80$, $p < 0.005$), but as illustrated by Fig. 2, no interaction occurred between sleep condition and attentional condition ($F1,11 = 0.16$).

To determine whether the shortening of produced durations observed in deprivation condition was correlated to alertness level, we computed a duration shortening (DS) index and assessed the degree of duration shortening in deprivation condition for each subject. DS corresponds to the following ratio: (produced duration in rest condition − produced duration in deprivation condition)/produced duration in rest condition. For the analysis of correlation, we attributed the maximal L1 (300 s) to the participant who presented no signs of sleep, which corresponds to the less sleepy state. Fig. 3 shows DS as a function of L1 (latency of sleep stage I). We observe that DS tended to be larger for participants who presented a shorter L1 (with 12 participants, Spearman $r = -0.55$, $p = 0.06$), which means that the more sleepy the participants, the shorter the durations they produced.

2.2.2.1.2. Variance in temporal production. The variance of the durations produced by each subject in each condition was computed. Statistical analysis revealed no significant main effect of sleep condition or attentional condition (sleep condition: $F1,11 = 0.23$; attentional condition: $F1,11 = 0.34$), nor a significant sleep × attention interaction ($F1,11 = 0.2$). Sleep deprivation, as well as attention manipulation, did not affect participants' variability;

Table 1
Latency (in s) of sleep stage 1 (L1), sleep stage II (L2), and sleep stage III (L3) obtained for each subject from the EEG recorded before each of the two experimental sessions. No = no sign of sleep observed.

	L1	L2	L3
Deprivation			
1	No	No	No
2	30	60	120
3	30	90	300
4	180	300	No
5	90	150	No
6	90	180	No
7	30	180	No
8	30	90	No
9	60	150	No
10	120	180	210
11	270	No	No
12	0	30	No
Rest			
1	No	No	No
2	120	150	No
3	No	No	No
4	No	No	No
5	No	No	No
6	No	No	No
7	No	No	No
8	150	No	No
9	No	No	No
10	No	No	No
11	No	No	No
12	No	No	No

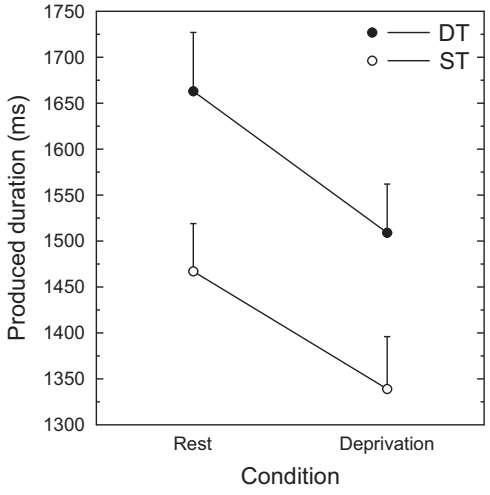

Fig. 2. Produced durations. Mean produced duration and standard errors of the mean (error bars) as a function of sleep condition (rest night or deprivation night) in the single-task (white circle) and dual-task (black circle) conditions.

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rather it only shifted the entire distribution of produced durations towards shorter (sleep deprivation) or longer (dual-task condition) durations.

2.2.2.2. RT task. Error rates and mean RTs were calculated for each subject in each condition. Fig. 4A shows the mean RT obtained when participants performed the task under single or dual-task conditions, as a function of sleep condition. Participants responded more quickly in the single (305 ms) than in the dual-task condition (398 ms), ($F1,11=18.13$, $p<0.01$) but there was neither a significant effect of sleep condition ($F1,11=1.71$), nor a significant sleep × attention interaction ($F1,11=0.1$).

The button press error rate obtained over the entire experiment was 1.84%. No effect of sleep deprivation was observed on error rate ($F1,11=2.01$), but a significant effect of attention revealed that participants made less errors in the dual-task (1.08) than in the single (2.6) condition (Fig. 4B) ($F1,11=5.16$, $p<0.05$).

2.3. Discussion

The main result of Experiment 1 is that participants produced shorter durations after sleep deprivation. Moreover, the more sleepy they were, the shorter the durations they produced. According to a timing model of the pacemaker-counter type, one hypothesis to explain underproductions is an acceleration of pacemaker rate. When pacemaker rate increases, pulses are emitted and accumulated faster. The number of pulses corresponding to the standard duration they have learned prior to sleep deprivation manipulations, would then be accumulated in a shorter time interval, leading to the production of shorter durations. The present data thus suggest that sleep deprivation could lead to an acceleration of the pacemaker rate. Moreover the lack of interaction between attention and sleep deprivation manipulations suggests that attention and sleep deprivation act at two different levels of temporal processing. Because attention is supposed to affect the switch component (Burle & Casini, 2001), the lack of interaction excludes a possible effect of sleep deprivation through attentional processes and reinforces the idea that sleep deprivation affects another component, very likely the pacemaker.

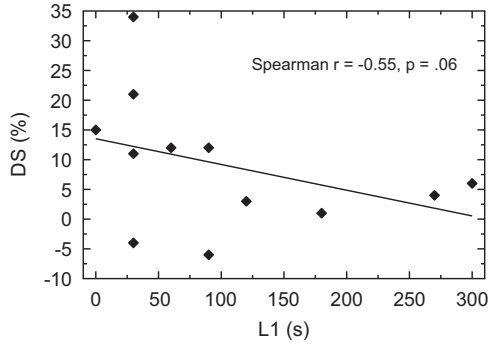

Fig. 3. Correlation between sleep and duration shortening. Correlation plots between the latency of sleep stage I (L1 in s) and the degree of shortening in duration produced in the deprivation condition, expressed as a percentage of duration produced in the rest condition (DS) (12 subjects).

This acceleration of pacemaker rate is consistent with an increase in cortical arousal, which could be consecutive to sleep deprivation, as reported in recent studies (Huber et al., 2012; Liu et al., 2010; Vyazovskiy et al., 2009). Indeed, several studies have already shown that increasing cortical arousal by different manipulations lead to an acceleration of the pacemaker rate (Burle & Casini, 2001; Penton-Voak et al., 1996). It has also been recently demonstrated that one night of sleep deprivation induces hyperstimulation of dopamine D2 (D2) receptors in the striatum (Volkow et al., 2008, 2009). Since Meck's seminal study (1986), showing that the drug dose needed to distort interval timing in rats was negatively correlated with the drugs affinity for the D2 receptor, data acquired in animals (see among others, Matell, Bateson, & Meck, 2006; and for an overview, Meck, 1996) and humans (Rammsayer, 1989, 1997, 1999) have consistently demonstrated that DA receptors play an important role in temporal information processing. DA antagonists (haloperidol) typically lead to behavioral performance that is consistent with a slowing of the clock, whereas DA agonists (methamphetamine or cocaine) produce the opposite effect (Drew, Fairhurst, Malapani, Horvitz, & Balsam, 2003; Meck, 1983, 1996). Therefore the underproduction observed here could also be due to hyperstimulation of D2 receptors consecutive to sleep deprivation leading to an acceleration of pacemaker rate.

Nonetheless, the underproduction of time intervals could also be explained by a deficit in memory. Indeed, during the training phase, the number of pulses that mark the durations to be learned are associated with this duration and stored in reference memory. During the test phase, samples from the accumulator are compared to those in reference memory in order to decide whether the current duration matches the previously learned target duration. If sleep deprivation deteriorates the representation of

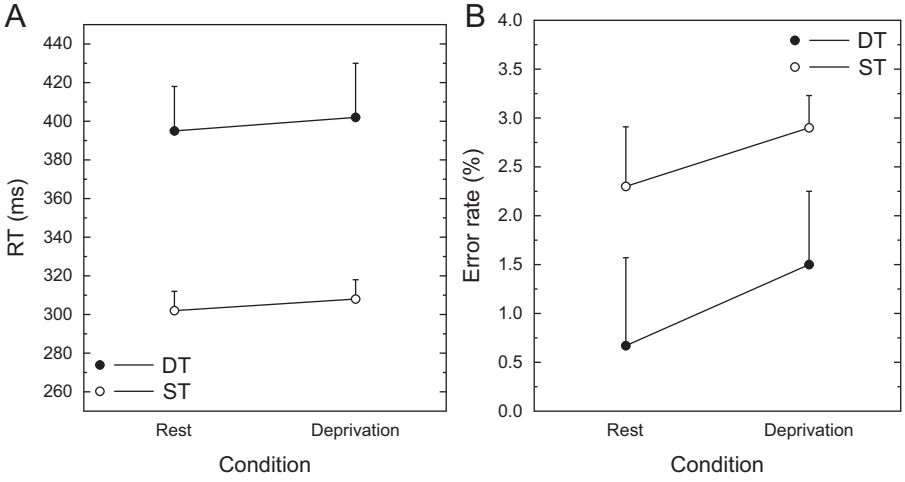

Fig. 4. Reaction Time and error rate: (A) mean reaction time (RT in ms) and (B) error rate as a function of sleep condition (rest night or deprivation night) in the single-task (white circle) and dual-task (black circle) conditions. Error bars correspond to standard errors of the mean.

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the learned duration stored in reference memory, for example by decreasing the number of pulses, the criterion value corresponding to a smaller number of pulses will be reached earlier also yielding to shorter produced durations. Effects on temporal memory have already been observed in animals injected with cholinergic agonists such as physostigmine. Administration of cholinergic agonists in rats caused the duration stored in, and retrieved from memory, to be less than the real duration, whereas cholinergic antagonists produced the opposite distortion (Meck, 1983,1996). Therefore, from this first experiment, we can also hypothesize that values stored in reference memory are impaired by sleep deprivation. This would be congruent with data reporting that sleep deprivation impairs reference memory in rats (Kalomia, Bishnoi, & Kular, 2008; Smith, Conway, & Rose, 1998) as well as data from insomniacs who often complain of memory deficits, both suggesting that insomnia severity is related to a decrement in delayed recall (Schmidt, Richter, Gendolla, & Van Der Linden, 2010).

To conclude, our data suggest that pacemaker rate accelerated after sleep deprivation but the hypothesis of a memory deficit deserves to be more deeply investigated before being discarded.

3. Experiment 2: Speech perception after one sleep deprived night

We designed the second experiment – speech perception involving the implicit measurement of segmental duration (Casini et al., 2009) – to further investigate the effect of sleep deprivation on pacemaker rate. The experiment aimed to verify whether results from Experiment 1 could be generalized to another temporal task that differs from the previous one in three major ways. First, it taxes temporal processing implicitly. Second, segmental durations involved in speech perception are in the range of tens to hundreds of milliseconds, whereas the temporal production task in Experiment 1 required estimation of a 1100 ms duration. And finally, since the perception of voiced or voiceless consonant does not require temporal reference memory but does require an accurate estimate of segmental duration, an effect of sleep deprivation on segmental duration judgments should therefore confirm the hypothesis that sleep deprivation does not cause reference memory impairment but actually affects the internal clock.

3.1. Material and methods

3.1.1. Participants

The same 12 participants took part in the experiment. Two participants were excluded from the study because they were unable to correctly perform the word identification task during the training session.

3.1.2. Procedure

Participants were seated in a dimly lit, sound-proof room. Two response keys were available. The experiment was controlled by a computer running t-scope (Stevens et al., 2006). Auditory stimuli were delivered to participants through headphones and participants were required to indicate whether the presented stimuli corresponded to the beginning of words *cache* (/ka∫/, voiceless final consonant) or *cage* (/kaʒ/, voiced consonant) by pressing the appropriate response key with the index or middle finger of the right hand, depending on the word.

The auditory stimuli were synthesized using the HLSyn speech synthesis system (Sensimetrics). The HLSyn control parameters were derived from the word *cache* /ka∫/ recorded beforehand by a French male speaker and submitted to a detailed acoustic analysis. A series of 11 stimuli was generated from this synthetic sequence. Each stimulus contained the sequence/ka/ immediately followed by white noise that replaced the final consonant (/∫/ or/ʒ/). Vowel durations varied from 150 ms to 310 ms in 16 ms steps. It has already been shown that subjects clearly perceived the beginning of the word *cache* for the/a/ duration of 150 ms and the beginning of the word *cage* for the/a/ duration of 310 ms (Casini et al., 2009). For more linguistic and acoustic details, see Casini et al. (2009).

The experimental block contained 110 trials corresponding to 11 different auditory stimuli, each delivered 10 times (inter-trial interval=2 s). As in the previous experiment, each participant completed two experimental sessions, rest and deprivation conditions, in counterbalanced order across participants, with one training session one day before the first experimental session.

In the training session, we only used the shortest (/a/ duration of 150 ms) and longest (/a/ duration of 310 ms) stimuli. The participants were first presented with this pair of stimuli four times and were instructed just to listen to the stimuli with no response required. Next, they performed the word identification task with each of these two stimuli randomly presented ten times. In average, the participants correctly identified the word *cache* when the vowel was short and the word *cage* when the vowel was long (mean percentage of correct responses=82.94).

3.2. Results

For each participant, we computed percentage of *cache* responses for each vowel duration and after we fitted a logistic function to individual performance in each condition, deprivation or rest, separately. This function allowed us to estimate two dependent variables: the standard deviation (SD) and the point of subjective equality (PSE). The standard deviation reflects perceptual variability: larger SD indicates that a greater difference was required between the two vowel durations in order to identify the two different words, which also means that participants were more variable in their judgment. The PSE corresponds to the value at which participants were equally likely to respond *cache* or *cage*. It reflects the perceptual boundary between *cache* and *cage* along the vowel duration scale. An increase in the PSE (corresponding to a rightward shift of the curve) means that, for intermediate durations, participants chose to respond *cache* more often, inversely a decrease in the PSE (corresponding to a leftward shift of the curve) means that participants chose to respond *cage* more often. To aid in the interpretation of results, it should be noted that a shift towards responding *cache* means that participants perceived the/a/ as shorter and then it reflects an underestimation of the vowel duration. Conversely, a shift towards responding *cage* means that participants perceived the/a/ as longer and then it reflects an overestimation of the vowel duration.

Statistical analysis were only carried out on seven participants because three participants were not able to perform the task in the deprivation condition, although were able in the rest condition. In the deprivation condition, these participants obtained a percentage of *cache* responses close to 50% whatever the vowel duration, preventing the estimation of the two variables, SD and PSE.

The differences between both conditions were significant for SD as well as for PSE. Fig. 5a shows an increase in mean SD under the deprivation condition (39.3 ms) compared to the rest condition (18.5 ms) ($t_6=2.1$, $p<0.03$). After one night of sleep deprivation, a larger difference between vowel durations was needed for participants to detect the difference between *cache* and *cage*. On Fig. 5b, we observe that the mean PSE was inferior under the deprivation condition (223 ms) compared to the rest condition (243 ms) ($t_6=3.71$, $p<0.01$), revealing a leftward shift in the perceptual boundary between the two words under sleep deprivation. This means that for intermediate durations, participants identified the

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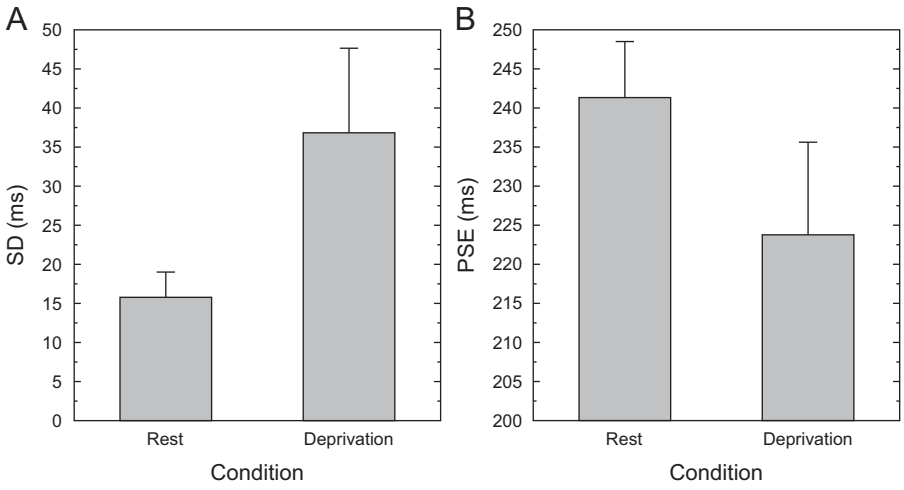

Fig. 5. Speech perception task: (A) mean standard deviation (SD in ms) and (B) mean point of subjective equality (PSE in ms) obtained in the word identification task performed after a rest night or after one sleep deprivation night.

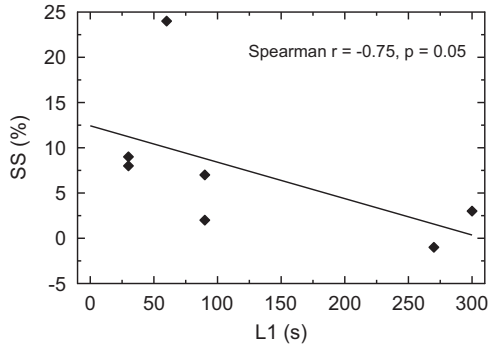

Fig. 6. Correlation between sleep and PSE shift. Correlation plots between the latency of sleep stage I (L1 in s) and the shift in PSE in the deprivation condition, expressed as a percentage of shift obtained in the rest condition (DS). (12 subjects).

masked word as *cage* more often than *cache* after one night of sleep deprivation.

To determine whether the leftward shift of the PSE observed in deprivation condition was correlated to alertness level, we computed a size of shift index (SS) and assessed the size of PSE shift in deprivation condition for each subject. SS corresponds to the following ratio: (PSE in rest condition − PSE in deprivation condition)/(PSE in rest condition). Fig. 6 shows SS as a function of L1 (latency of sleep stage I). We observe that SS was larger for participants who presented a shorter L1 (with 7 participants, Spearman $r=-0.75$, $p=0.05$), which means that the more sleepy the participants, the larger the probability to identify the masked word as *cage*.

3.3. Discussion

The present data show that speech perception was affected by sleep deprivation. Firstly, the word identification task was more difficult for participants after one sleep deprivation night. This is shown by the increase of SD after sleep deprivation and is confirmed by three participants being unable to perform the task at all after sleep deprivation. Second, and most importantly, perceptually ambiguous words were more frequently perceived as ending in a voiced consonant (*cage*) after sleep deprivation. Since voiced consonants are associated with longer vowels (Casini et al., 2009; Fischer & Ohde, 1990), this result suggests that participants perceived longer/a/ durations after sleep deprivation, which would mean they overestimated perceived durations. Duration overestimations (as motor underproductions observed in Experiment 1) can be explained by an acceleration of the pacemaker rate. If the pacemaker rate increases, more pulses are accumulated during an equal interval of time leading participants to overestimate vowel duration. As a consequence, the present data again suggest that sleep deprivation may speed up pacemaker rate. Moreover, since the association between segmental duration and consonant voicing is established during childhood and not during the training session, the results refute the hypothesis that the sleep deprivation effects observed in the first experiment are due to impairments in temporal reference memory. The overestimation of segmental duration found in the speech perception task is more likely to be explained by an effect of sleep deprivation on clock speed. Therefore, these results would again support the hypothesis that sleep deprivation produces an acceleration in pacemaker rate.

4. General discussion

The question addressed in the present study was the effect of sleep deprivation on the internal clock. In the production task, participants underproduced time intervals and in the word identification task they overestimated vowel duration. In the framework of a pacemaker-counter clock model, both behavioral data may be explained by an acceleration of pacemaker rate. This acceleration of the pacemaker rate could be due either to an increase in cortical arousal due to sleep deprivation as proposed by recent studies (Huber et al., 2012; Liu et al., 2010; Vyazovskiy et al., 2009), or to a hyperstimulation of striatal DA D2 receptors consecutive to one sleep-deprived night (Volkow et al., 2008, 2009). On the other hand, these results can neither be explained by attentional effects (see experiment 1) nor by a memory effect (see experiment 2). The last component of the internal clock which could also be affected is the decision stage, but if we consider that sleep deprivation could bias the decision stage, there is no reason to think that this effect would be proportional to the duration range. Indeed, it seems that sleep deprivation proportionally affected temporal judgments in the two different duration ranges: subjective duration shortening in the sleep deprived condition compared to the rest condition was of 6.43% in Experiment 1 and the size of the shift of the PSE between conditions was of 7.49% in Experiment 2 ($t_6=0.93$, non-significant). This observation provides a reasonable argument in favor of the idea that sleep deprivation taps on the pacemaker rate.

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Moreover, in both experiments, we observed that the degree of duration shortening tended to be correlated to the alertness level of participants: the more sleepy the participants, the faster the pacemaker. If we consider that changes in pacemaker rate could be due to a hypersensitization of striatal DA D2 receptors, this correlation is consistent with the idea proposed by Volkow and colleagues that striatal DA D2 would increase with the sleep state of the participants, as if the aim of this D2 hyperstimulation was to maintain brain arousal as the drive to sleep increases. This seems partly achieved in Experiment 1 since RT task performance were not impaired in the sleep deprivation condition, but not in Experiment 2 in which participants presented greater difficulty in performing the speech perception task after sleep deprivation. Concerning RT task performance, it has already been reported that several factors could modify the effects of sleep deprivation, among others the length of the period of sleep deprivation, the duration of the task, and the size of the preparatory period (delay between the warning stimulus and the imperative stimulus) (for review, see Oken, Salinsky, & Elsas, 2006). The effects of sleep deprivation on RT are partially reversed when the task is short (Steyvers & Gaillard, 1993) and when the preparatory period is short (Cochran, Thorne, Penetar, & Newhouse, 1992) as in our case.

In addition, and very importantly, our results suggest that sleep deprivation affects timing across different duration ranges: In the speech perception task, segmental durations varied from 150 to 310 ms whereas in the duration production task, participants were required to produce intervals of 1100 ms. These results disagree with the often-mentioned idea that the measurement of sub-second durations, such as those involved in speech, is not accomplished using the same mechanisms as used for longer durations (for review, see Buhusi & Meck, 2005). More specifically, in 2008, Meck and colleagues proposed a role for DA only for timing of supra-second durations. If we accept the idea that changes in pacemaker rate are due to dopaminergic hypersensitization of striatum, our data provide indirect arguments to suggest that segmental durations could also be sensitive to DA D2 level.

Finally, sleep deprivation affected vowel duration judgment in the same manner it affected temporal production. First, this result suggests that explicit and implicit temporal judgments are similarly affected by sleep deprivation which seems good arguments to propose that they share common mechanisms. This is not consistent with data reported by Zelaznik, Spencer, and Ivry (2002) who found no significant correlation in timing performance between the continuation phase of a paced finger-tapping task (explicit timing) and continuous circle drawing (implicit timing), suggesting engagement of discrete functional mechanisms. However, more recently, Piras and Coull (2011) proposed that the same representational mechanism was involved in implicit and explicit timing.

Nonetheless, it should be noted that in the explicit temporal task (Experiment 1), sleep deprivation affected the timing accuracy of participants but not their precision since there was no effect of sleep deprivation on subject's variability, whereas in the implicit task (Experiment 2), the variability of judgments was also affected. Therefore, even if temporal production and speech perception have common mechanisms that affect clock, explicit and implicit tasks may still be differentiated on the basis of their timing precision even if it is also simply possible that speech identification task provided more sensitive measure to variability. This is also congruent with data reported by Piras and Coull (2011) who observed that timing variability was greater during implicit timing, at least for durations greater than 200 ms. It should also be noted that according to scalar expectancy theory, if the pacemaker rate is increased, then the variability should decrease and not increase as found in Experiment 2. This suggests that even if the pacemaker rate is affected by sleep deprivation, some other processes involved in this judgment may also be disrupted.

Second, this similar effect of sleep deprivation on temporal production and speech perception also suggests that estimation of speech segment duration relies on mechanisms similar to those implicated in duration estimation for non-speech stimuli. In Casini et al. (2009) we demonstrated that the perception of segmental duration was sensitive to attention, as has previously been shown for explicit time estimation (Brown, 1997; Casini & Macar, 1997; Macar, Grondin, & Casini, 1994; Zakay, 1989). In the current research we found that it is also sensitive to sleep deprivation, possibly *via* DA brain levels. This provides evidence that the perceptual estimation of segmental duration in speech is governed by a general timing system and favors the idea of a central supramodal and context-independent internal clock. It also enables a better understanding of why patients with Parkinson's disease present deficits in speech perception (Gräber, Hertrich, Daum, Spieker, & Ackermann, 2002). The dopaminergic disorder associated with Parkinson's disease could modify pacemaker rate and hence disrupt segmental duration perception involved in speech perception.

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