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Precise timing accounts for posttraining sleep-dependent enhancements of the auditory mismatch negativity

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Memory consolidation is a long-lasting dynamic process by which new acquired information is transformed at different analysis levels, from molecules to cognition, without additional practice. Results from a previous study on event-related potentials (ERPs) suggest that part of the neural events promoting changes in the electrophysiological correlates of enhanced automatization in a sound discrimination task occur during sleep. These data were reanalyzed in the present study at the single-trial level, and results indicated that the first night of sleep succeeding training is absolutely required to improve the timing consistency of cortical neural assemblies involved in automatic sound-change detection, as revealed by a significant reduction in the latency-jitter of the MMN response across trials. This change in the regularity of the brain response to previously trained sounds facilitated involuntary switch of attention towards the same sounds when they were task irrelevant, as reflected by the P3a emergence after posttraining sleep. Both responses were, however, prevented in subjects deprived of sleep the night following training in the sound discrimination task. We hypothesize that the reduction in the MMN latency-jitter, which, in turn, triggered an automatic shift of attention, might result from a change in synaptic efficacy and/or neural excitability, rather than from changes in firing synchronization and/or size of representation.

Keywords: Sleep; Memory consolidation; Perceptual learning; Event-related potentials; Single-trial analysis; Wavelet denoising; Humans

Introduction

Repetition is a frequent strategy by which the brain acquires and forms new perceptual and motor memories. When the brain experiences the same stimuli repeatedly, structural and functional changes evolve in local and widely distributed neocortical regions for improving perceptual and behavioral abilities. At first, new acquired information, as revealed by significant gains in performance across trials, remains in a labile state. With the passage of time, however, new memories become stronger and resistant to disruption without additional practice (Brashers-Krug et al., 1996; Shadmehr and Brashers-Krug, 1997). This dynamic process, which often results in further behavioral improvement when following a period of sleep (Gaab et al., 2004; Fischer et al., 2002; Gottselig et al., 2004; Graves et al., 2003; Karni et al., 1994; Mednick et al., 2003; Stickgold et al., 2000; Walker et al., 2002; see however Robertson et al., 2004), is known as the consolidation phase of learning. Thus, acquisition of memory is followed by two different stages of consolidation. While the first stage mainly develops during time awake and might be mediated by local cellular mechanisms, the subsequent stage develops during intervening periods of sleep and seems to be mediated by neural reorganization (Walker, in press).

Along with behavioral benefits, consolidation of procedural memory is often associated with a reduction of the attentional control required for the correct execution of the task (Schneider and Shiffrin, 1977; Shiffrin and Schenider, 1977). Learning-induced automatization has been also demonstrated as being impaired after one night of posttraining sleep deprivation (Atienza et al., 2004). In that study, subjects were presented with two sounds that were perceptually indistinguishable when organized into a single stream. After one training session, participants learned to separate high and low frequency tones into two independent sound streams. This change in the organization of sound influenced late stages of perception, as reflected by the training-induced enhanced performance, but also influenced the memory representation encoded in pre-perceptual stages of processing, as reflected by the emergence of the mismatch negativity (MMN)—one component of event-related potentials (ERPs) developing mainly in the auditory cortex in response to infrequent changes in previously registered regularities in acoustic sequences, even in the absence of attention (Näätänen and Winkler, 1999; Naatanen et al., 2001).

It has recently been demonstrated that the sound organization underlying the deviance-detection process reflected by MMN is not identical to that appearing in perception (Winkler et al., submitted

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for publication). So, changes in the MMN response do not necessarily correlate with changes in behavior because they reflect different stages of perceptual sound organization. In agreement with this notion, Atienza et al. (2004) found that learning was enhanced within the 48 and 72 h of training irrespective of whether subjects slept or not the night after training. On the contrary, changes in pre-perceptual sound organization processes were not evident in sleep-deprived subjects after one night of sleep recovery. In fact, only control participants showed an enhanced MMN followed by a large P3a component when sounds were gated out of the attentional focus. This MMN enhancement is thought to participate in triggering automatic shift of attention toward the task-irrelevant infrequent sounds embedded in the repetitive sequence (Näätänen, 1992), which is indexed by the subsequent P3a wave (Escera et al., 1998). Therefore, the results of our previous study can be interpreted in terms of enhanced automatization. From this point of view, sleep promotes, at least partially, qualitative changes in the memory representation encoded at early stages of sound organization–as reflected by the delayed enhanced MMN–which, in turn, facilitates involuntary switch of attention to the ignored sounds—as reflected by the delayed emergence of the P3a component.

The enhanced MMN 48 h after completion of practice might result from a change in neural recruitment and/or neural synchronization. If so, such enhancement should not only be seen after averaging all brain responses to deviant sounds, but also in the single responses (i.e., single trial) to each one of these sounds. Alternatively, increases in the MMN amplitude might result from a reduction in its latency variability (latency-jitter) from trial to trial. We reanalyzed data from a previous study (Atienza et al., 2004) in order to determine if either one or several of these mechanisms account for automatization of learning reflected in the enhanced MMN/P3a responses following posttraining sleep.

Materials and methods

Subjects

Twenty university students (13 female, 24.2 ± 2.35 years, all non-musicians) participated in the present study. Medical illness, psychiatric/psychological disturbance, sleep disorders, substance abuse, and/or neurological disorders were criteria for exclusion. Subjects were instructed to maintain a normal sleep–wake schedule and refrain from alcohol, caffeine, medication, and/or drug consumption during the study duration. Sleep logs indicated neither variation in the length of sleep time in the nights proceeding the retest sessions nor in the diurnal naps.

Procedure

Subjects were trained in a single session to be able to discriminate two complex auditory patterns that differed in only one (the sixth tone) of their eight frequency tones. ERPs to standard and deviant (13% of stimuli) auditory patterns were recorded immediately before and after training (pre- and posttraining sessions), as well as before retesting performance at 48 and 72 h posttraining. Subjects were asked to ignore the sounds and read a book of their own choosing simultaneously with electroencephalographic (EEG) recordings. At the beginning and middle of each ERP recording session, subjects completed the Stanford Sleepiness Scale to discard changes in sleepiness as the main explanation of between-group differences. Ten of these subjects were deprived of sleep within 28 h of training, while the remaining ten control subjects slept at home (for more detail on the stimuli and procedure, see Atienza et al., 2004). EEG recordings were not performed while subjects slept. The experimental protocol is summarized in the diagram shown in Fig. 1.

Electrophysiological recordings

EEG activity was recorded from nine scalp electrodes: three along the midline (Fz, Cz, and Pz), two pairs of lateral electrodes referred to as L1/R1 and L2/R2 placed one-third and two-thirds of the distance from Fz to each mastoid, respectively, and two electrodes located at mastoids (L3/R3). The reference electrode was placed on the tip of the nose. Vertical eye movements were recorded with electrodes placed above and below the left eye, and horizontal eye movements with electrodes placed 1 cm lateral to the outer canthus of each eye. Signals were amplified and digitized at 250 Hz and low- and high-bandpass filtered at 0.1 and 40 Hz (−3 dB frequencies for a 24-dB/octave roll-off curve). Impedance for all electrodes was kept below 5000 Ω.

Data preprocessing

The first five trials of each stimulus block and the trial immediately following each deviant pattern were excluded as were trials with artifacts exceeding ±100 μV. For each artifact-free trial,

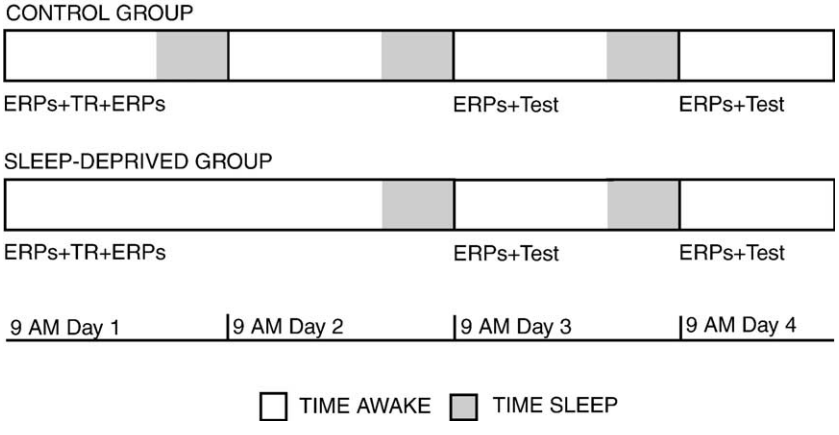

Fig. 1. Diagram of the experimental protocol followed by the control and sleep-deprived group. TR = training.

an epoch of 800 ms, including a 100-ms prestimulus baseline, was selected. Signals were digitally filtered in the range 0.1–30 Hz and drift artifacts corrected by applying linear detrending analysis to each trial. For each subject, ERP averages were computed for the standard and deviant sound pattern, separately in each experimental session (pretraining, posttraining, 48 h posttraining, and 72 h posttraining).

As MMN shows maximum amplitude at Fz and inverts polarity at the mastoids as a result of its brain generator in the auditory cortex (Alho et al., 1998; Deouell et al., 1998; Kropotov et al., 2000), Fz and L3 (left mastoid) recordings for each sound pattern, experimental session, and subject were analyzed.

Single-trial analysis

Single-trial event-related responses were obtained by using wavelet denoising, a recently proposed algorithm based on the wavelet transform (Quian Quiroga, 2000; Quian Quiroga and Garcia, 2003). This method diminishes the contribution of background EEG activity, thus improving the visualization of the single-trial ERPs. Briefly, the method consists of the following steps: (i) the time–frequency decomposition of average ERP is obtained by using the multiresolution decomposition based on the wavelet transform; (ii) wavelet coefficients correlated with the ERP components are identified, with some additional coefficients also considered as allowing for latency variations, and (iii) single-trial ERPs are decomposed and then reconstructed from only those coefficients chosen in the previous step.

In this study, a five level multiresolution decomposition was used, thus having five scales of details (D1 to D5) and a final approximation (A5). Quadratic bi-orthogonal B-Splines were chosen as the basic wavelet functions due to their similarity to the evoked responses (therefore having good localization of the ERPs in the wavelet domain), as well as to their optimal time–frequency resolution (for details, see Quian Quiroga et al., 2001).

ERP analysis after denoising

The amplitude and latency of the MMN after denoising were measured in the difference wave obtained by subtracting the denoised ERP to the standard pattern from the denoised ERP to the deviant sound pattern. The amplitude was defined as the maximum negative and positive peak at Fz and L3, respectively, within the 300 ms following the onset of the sixth deviant tone, and the latency as the point where the maximum peaks were observed within the same time interval. The same was done for the P3a component, within a different time window ranging between 100 and 400 ms from introduction of the deviant tone within the sound pattern. Amplitude and latency variability of these components (amplitude- and latency-jitter) were defined as the standard deviation (SD) of the peak-amplitudes and peak-latencies of the single-trials for the standard and deviant sound patterns in the time interval of the MMN response. The same parameters were calculated for the P3a in those conditions where it was evident, i.e., at 48 and 72 h posttraining.

Statistical analysis

Differences in the amplitude and latency SD were evaluated with two-way ANOVAs for each ERP component and for each sound pattern, with session as the within-subject factor (pretraining, posttraining, 48 h posttraining, and 72 h posttraining) and group (control or sleep-deprived) as the between-subject factor. The degrees of freedom for the within factor were corrected with the Greenhouse–Geisser factor. The Newman–Keuls test was applied for the post hoc comparisons.

Results

Denoising of the event-related potentials

The gray curves in Fig. 2A show the decomposition of average ERPs to deviant sounds at Fz in the posttraining session for a control subject (SC_6). On the left side, we plot the wavelet coefficients and, on the right side, the actual reconstruction of the signal in the different frequency bands. The sum of all the reconstructions again gives the original signal (gray curve of the uppermost right plot). An example of the first 40 single trials before and after denoising is also shown in Fig. 2B.

The MMN is better seen in the difference wave resulting from subtracting ERPs in response to standard stimuli from those elicited by the deviant sound. Grand average difference waves at Fz after signal denoising are shown in Fig. 3 for both the control

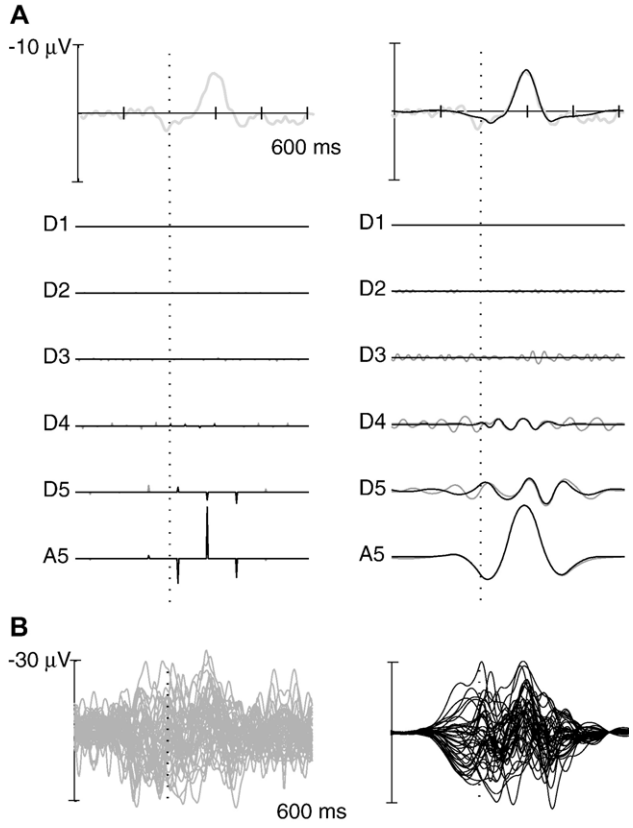

Fig. 2. (A) Multiresolution decomposition and reconstruction of averaged potentials to the deviant sound pattern in a control subject. Gray curves: original decomposition and reconstruction; black curves: denoised decomposition and reconstruction; vertical dashed line: moment at which the frequency change was introduced within the sound pattern. D1–D5 are the details at different scales and A5 is the last approximation. (B) Original (gray curves) and denoised (black curves) overlapped single trials (first 40 trials) to the deviant sound pattern.

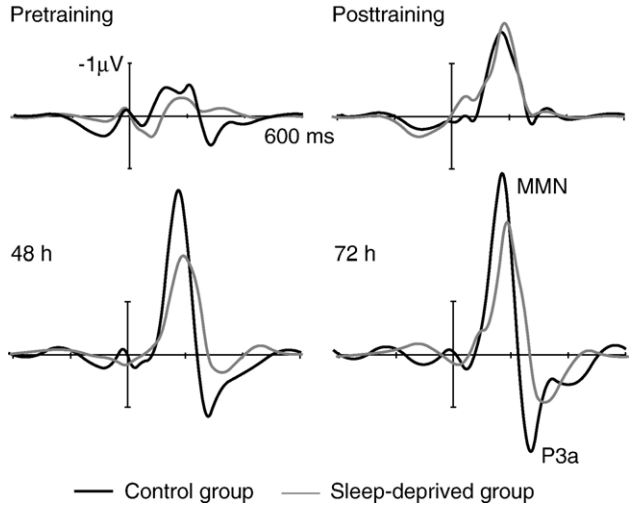

Fig. 3. Grand averaged difference waves obtained at Fz in the control and sleep-deprived group for all experimental conditions after denoising the single trials.

and sleep-deprived group in all experimental sessions. As in our previous analysis without implementing denoising (Atienza et al., 2004), only control subjects showed a significantly enhanced MMN at 48 and 72 h posttraining re-tests (interaction effect: $F(3,51) = 3.25$, $P < 0.05$, $\varepsilon = 0.7$; post hoc test for the control group: $F(3,36) = 11.4$; $P < 2 \times 10^{-5}$). The enlargement of the MMN was however not seen at the left mastoid. The slight difference observed in the MMN amplitude between the two groups at the pretraining session was not significant. Differences seen in Fig. 3 are mainly due to the results from one subject who already showed a clear MMN before training.

The P3a wave only emerged at 48 and 72 h posttraining (see Fig. 3), its amplitude being significantly higher in the control group ($F(1,17) = 14.6$, $P = 0.001$), as it was before implementing denoising.

Amplitude- and latency-jitter of the event-related negativity within the MMN time window

Fig. 4 (top panel) shows the amplitude-jitter (SD mean) of the deviant-related negativity amplitude at Fz within the MMN time window per group and experimental session for standard and deviant sound patterns. Two-way ANOVAs of repeated measurements for each sound pattern yielded neither significant effects of the session and group nor interaction effect on the inter-trial amplitude consistency of the event-related negativity. On the contrary, the latency-jitter significantly decreased during posttraining sessions when subjects were allowed to sleep the first night after training (interaction effect: $F(3,51) = 3.1$, $P < 0.05$, $\varepsilon = 0.8$; post hoc test for the control group: $F(3,36) = 14.2$, $P < 3 \times 10^{-6}$). This effect was only observed for the deviant sound at Fz as can be seen in Fig. 4 (bottom panel). This effect was not seen at LM (data not shown).

The abovementioned results suggest that the enhanced MMN observed in the control group at 48 h results from an increase in the precise timing of neural responses. Indeed, when latency-jitter of the single-trial potentials to the deviant sound was corrected, control and sleep-deprived subjects showed no differences in the amplitude of the MMN in all posttraining sessions as illustrated in Fig. 5.

Amplitude- and latency-jitter of the P3a

Unlike MMN, the amplitude- and latency-jitter of the P3a to the deviant sound at Fz were neither affected by the experimental

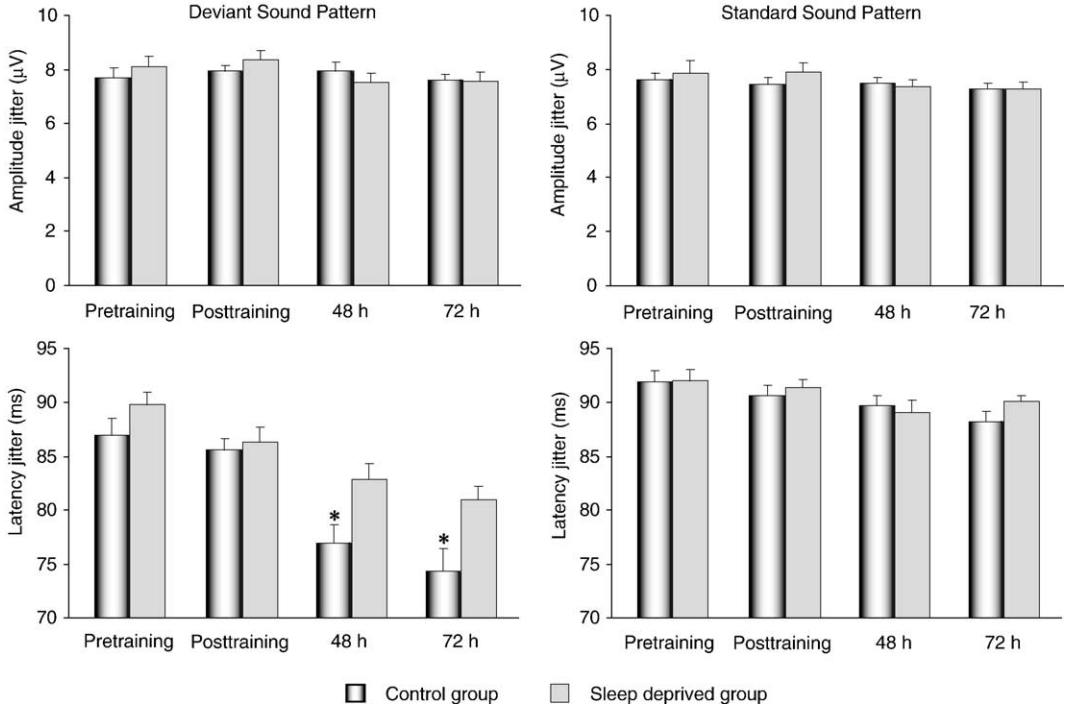

Fig. 4. Mean standard deviation for the amplitude and latency of the event-related negativity measured in the single trial within the MMN time interval for the standard and deviant sound in all experimental conditions.

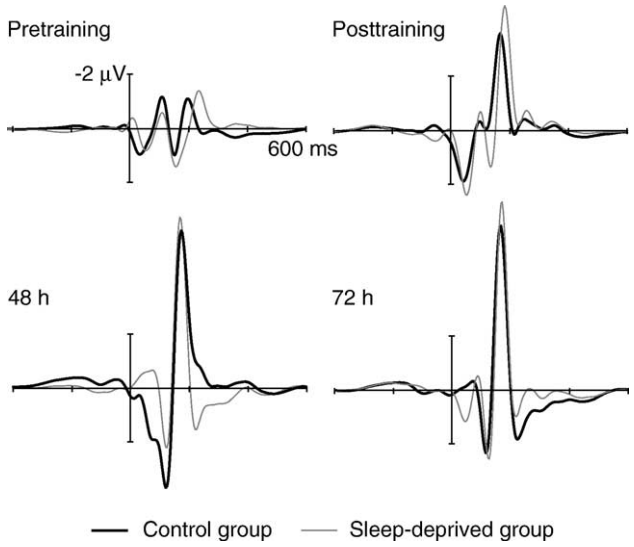

Fig. 5. Grand averaged difference waves obtained at Fz in the control and sleep-deprived group for all experimental conditions after correcting latency-jitter of the brain response obtained within the time interval of the MMN.

session nor sleep deprivation. Grand average difference waves after correcting the latency-jitter for each experimental group at posttraining sessions are shown in Fig. 6. Subjects who slept the night following training showed a much larger P3a at 48 and 72 h than subjects deprived of sleep (interaction effect: $F(3,51) = 3.8$, $P < 0.03$, $\varepsilon = 0.8$; post hoc test for the control group: $F(3,36) = 8.57$, $P < 0.0002$). These results suggest that the sleep-dependent emergence of the P3a, unlike the enhanced MMN, did not result from changes in the precise timing of cortical neural assemblies generating P3a.

Discussion

Reduction of MMN latency-jitter accounts for changes in sound organization after sleep

A few previous studies have trained subjects to discriminate complex sound patterns that were indistinguishable when integrated into one single stream but became perceptually differentiated when decomposed into two independent sound streams (i.e., segregation of low and high frequencies; Atienza and Cantero, 2001; Atienza et al., 2002, 2004; Gottselig et al., 2004; Naatanen et al., 1993; Schroger, 1994; Schroger et al., 1992). These studies further showed that, after training, subjects were able to automatically detect the infrequent change introduced in the low auditory stream, as indicated by the MMN to deviant sounds when attention was directed elsewhere. This suggests a reorganization of the auditory scene at both pre-perceptual and perceptual stages of information processing. Even though integration before training seems to be the default sound organization, after training the primed representation is segregation.

One of the abovementioned studies has further shown that auditory memory representations underlying MMN evolve after acquisition, at least partially, during sleep (Atienza et al., 2004). In that study, sleep deprivation did not prevent behavioral improvement in the auditory discrimination task while attention was focused on the relevant features of the sound. On the contrary, it prevented the delayed enhanced MMN and the P3a emergence shown by control subjects within 48 and 72 h of training when attention was directed elsewhere. In the present study, these data were reanalyzed at the single-trial level and went a step further, demonstrating that the MMN enhancement resulted from a reduction in its latency-jitter rather than from a true increase in its amplitude. This finding shed some light on possible neural mechanisms underlying automatization of learning.

Neural mechanisms underlying reduction of MMN latency-jitter after sleep

Changes in firing synchronization as well as in size and locus of the representation have been the main neural mechanisms reported as subserving consolidation of procedural memory (for a review, see Gilbert et al., 2001). However, results of the present study suggest that a different neural process is responsible for sleep-induced automatization, another aspect of memory consolidation (Atienza and Cantero, in press; Kuriyama et al., 2004). Control subjects showed higher MMN amplitudes than sleep-deprived subjects because the brain response latency to unpredicted changes in the sound pattern was more regular from trial to trial when subjects were permitted to sleep during the posttraining night. In fact, differences in the MMN amplitude between the two groups disappeared once latency-jitter of the deviant-related negativity at single-trial potentials was corrected.

Näätänen (1992) proposed changes in sharpening of tuning and cortical recruitment as main accounts of the experience-induced MMN-amplitude enhancement shown by subjects trained in the same auditory discrimination task as our participants (Naatanen

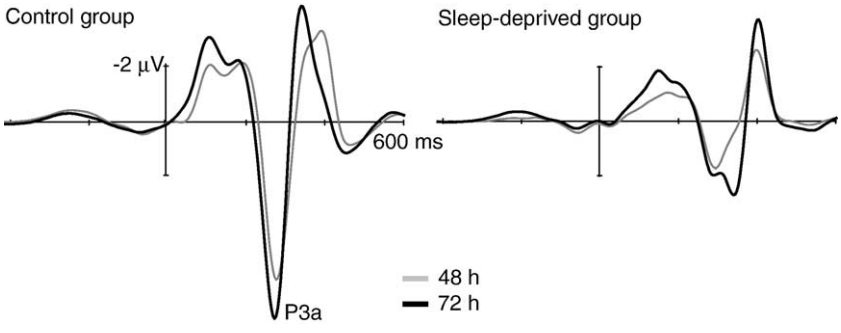

Fig. 6. Grand averaged difference waves obtained at Fz in the control and sleep-deprived group at 48 and 72 h posttraining sessions after correcting latency-jitter of the brain response obtained within the time interval of the P3a component of ERPs.

et al., 1993). Concomitant effects of both mechanisms on stimulus representation after intensive practice in different discrimination tasks have been demonstrated in animal studies (Daly et al., 2004; Recanzone et al., 1993; Weinberger et al., 1990; Zohary et al., 1994). This might be true for the increased MMN seen immediately after training in both groups and for the additional increase observed 2–3 days later at the single-trial level.

In contrast, our results point out that neither changes in size of representation nor changes in neural synchrony are crucial for triggering an automatic shift of attention. In support of this idea, the enhanced MMN response, shown by sleep-deprived subjects in single trials at 48 h posttraining, was not sufficient to elicit a P3a component in these participants. Instead, it was the higher probability of automatically detecting the sound change within a narrower time window which increased probability of activating P3a generator mechanisms and, hence, also increased the probability of initiating attention switching. So, P3a emergence was dependent on a change in MMN generating mechanisms. The reduction in the MMN latency-jitter might result from a change in synaptic efficacy and/or neural excitability, which is in agreement with the notion that automatic attention switching, reflected in the P3a wave, would only occur once neural excitability exceeds some threshold (Näätänen, 1992). Nevertheless, we have to be cautious when extracting conclusions on the cortical activity from changes in the latency-jitter because no study to date has investigated this issue.

All together, the present results suggest that the first night of sleep might be required for increasing automatization of learning by improving the response timing consistency of cortical neural assemblies involved in automatic sound-change detection; that is, by raising regularity of neuronal response in the auditory cortex or its neighborhood.

Long-term effects of learning on latency-jitter for single-trial potentials have previously been reported in musicians (professional drummers and bass guitarists). They were asked on demand to detect unexpectedly omitted parts of percussion sounds, these results later being compared to those of musically untrained subjects (Jongsma et al., 2004). The fact that musicians showed less latency-jitter of the omission evoked potentials and more constancy in their behavioral responses than untrained participants supports the crucial role of long-lasting training on internal representation of rhythm in the auditory scene. Our study takes this notion a step further demonstrating that intervening sleep following training is required to influence the timing consistency mechanism of perceptual learning after a single training session, without additional practice, as well as attention mechanisms.

How does sleep promote automatization of perceptual learning?

During training, participants not only learned to segregate the stimulus into two independent sound streams. Likely, they also learned to automatically weigh both streams differently because the change was always introduced in the low auditory stream. This change in the specific weight of one stream over the other implies a qualitative change in the sound organization at pre-perceptual stages of information processing. On the basis of our results, it seems that sleep facilitated this qualitative change which, in turn, promoted automatic shift of attention toward the ignored stimuli. The neural correlate of this qualitative change in pre-perceptual sound organization processes was an increase in the timing consistency of cortical neural assemblies involved in automatic sound-change detection and triggering of involuntary attention switching (seen in the MMN enhancement).

We are unaware of neuronal computations involved in the automatization of perceptual learning promoted during sleep and far from knowing the plasticity mechanisms underlying such neuronal computations. However, growing evidence suggests that neural consolidation events may develop during both slow-wave sleep (SWS) and REM sleep (Giuditta et al., 1995; Ribeiro and Nicolelis, 2004; Stickgold, 1998). While the former seems to be an ideal brain state for memory recall (neuronal reverberation), REM sleep would provide optimal conditions for memory storage (plasticity-related gene expression). Consistent with this idea, neuronal reverberation during SWS may be the way to increase the weight of the trained stream (low frequencies) over the untrained one. Subsequently, the same neuronal reverberation during REM sleep may facilitate sustained neuronal depolarization, a necessary and perhaps sufficient condition to induce reprogramming of gene expression (Ribeiro and Nicolelis, 2004). Learning-induced brain reactivation during REM sleep would then help synaptic efficacy and enhanced neural excitability that might be responsible for the reduction of the MMN latency-jitter following posttraining sleep.

Conclusions

Results derived from analysis of ERPs at the single-trial level suggest that sleep deprivation after training decreases probability of including potential meaningful stimuli within one's focus of attention. This conclusion is supported by two findings: (i) sleep makes automatic brain responses to trained stimuli more consistent in time as reflected by the delayed decrease of the MMN latency-jitter after sleep; and (ii) this sleep-induced change in the time organization of the brain response seems to be a prerequisite to generate an involuntary switch of attention to unexpected changes in the unattended environment, as revealed by the P3a emergence after posttraining sleep.

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