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Tracking pattern learning with single-trial event-related potentials

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Abstract

Objective: The main aim was to track the dynamics of pattern-learning using single-trial event-related potentials (ERPs). A new 'learning-oddball' paradigm was employed presenting eight random targets (the 'no-pattern') followed by eight regular targets (the 'pattern'). In total, six repetitions of the 'no-pattern' followed by the 'pattern' were presented.
Methods: We traced the dynamics of learning by measuring responses to 16 (eight random–eight regular) targets. Since this alternation of the 'no-pattern' followed by the 'pattern' was repeated six times, we extracted single-trial responses to all 96 targets to determine if learning occurred more rapidly with each repetition of the 'pattern.'
Results: Following random targets, ERPs contained a marked P3–N2 component that decreased to regular targets, whereas a contingent negative variation (CNV) appeared. ERP changes could be best described by sigmoid 'learning' curves. Single-trial analyses showed that learning occurred more rapidly over repetitions and suggested that the CNV developed prior to the decay of the N2-P3 component.
Conclusions: We show a new paradigm-analysis methodology to track learning processes directly from brain signals.
Significance: Single-trial ERPs analyses open a wide range of applications. Tracking the dynamic structure of cognitive functions may prove crucial in the understanding of learning and in the study of different pathologies.

Keywords: Learning; P3; N2; CNV; Single-trial; Wavelet analyses; Auditory ERPs; Oddball; Omitted stimuli

1. Introduction

1.1. Studies on pattern learning

Extracting patterns from the environment is central to human cognition. By identifying patterns, individuals are able to form predictions about upcoming events, helping them to function more efficiently within their environment (Friston, 2005; Huettel et al., 2002; Llinas, 2001). Behavioral research has revealed that recognition of patterns in event sequences occurs for the most part automatically and seems to rely on memory formation (Reber, 1967, 1989; Seger, 1994). When a sequence follows a repeating pattern, performance typically improves (i.e., becomes more efficient) (Eimer et al., 1996; Honda et al., 1998; Jentzsch and Sommer, 2001; Rüsseler et al., 2003; Salamon, 2002; Seger, 1994), often without conscious awareness (Honda et al., 1998; Reber, 1967, 1989; Schendan et al., 2003; Schlaghecken et al., 2000; Seger, 1994). This suggests that people acquire knowledge about patterns incidentally. Pattern learning is most often observed through changes in reaction times of overt motor responses (Reber, 1967, 1989; Seger, 1994). However, an important issue raised in incidental learning literature is the separate roles played by motor and perceptual processes (Eimer et al., 1996;

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Rüsseler et al., 2003). It is therefore preferable to employ a measure that allows to study pattern learning directly from neural responses, independent of motoric aspects that can hamper the interpretation of observed changes.

1.2. ERPs and pattern learning

Event-related potentials (ERPs) are time-locked voltage fluctuations in the EEG, resulting from neuronal responses to sensory, motor or cognitive events (Rugg and Coles, 1995). One of the major advantages of employing ERP measurements is that aspects of information processing can be instantaneously measured, without interference from e.g., motor skills, (Gaillard, 1988). Previous ERP (Baldwin and Kutas, 1997; Eimer et al., 1996; Lang and Kotchoubey, 2000; Rose et al., 2001; Rüsseler et al., 2003; Rüsseler and Rösler, 2000; Schlaghecken et al., 2000), PET (Berns et al., 1997; Honda et al., 1998) and fMRI (Aizenstein et al., 2004; Schendan et al., 2003) studies have focused on the study of learning patterned sequences by comparing performance before and after training (Schlaghecken et al., 2000). However, a caveat with these studies has been that the before and after comparison does not account for the instantaneous dynamics of the learning process. Though some of these studies have also attempted to track these dynamic processes by constructing consecutive sub-averages during a learning session (Honda et al., 1998), to our knowledge, this is the first study to track the dynamics of learning processes on a continuous trial-to-trial basis. The use of single trial evoked responses as markers of learning opens a wide range of new applications of ERPs. In particular, this information may prove crucial in understanding cognitive processes, and even in the study of different pathologies.

1.3. The "learning oddball" paradigm

In order to study the dynamics of pattern learning, it is necessary to track neuronal responses (using ERPs, PET or fMRI) on a single-trial basis throughout the experimental session, whereby the evoked neuronal response to every target tone is determined individually. This is a challenging task, owing to the low signal-to-noise ratio of the single-trial responses. A number of techniques for extraction of single-trial ERPs with optimum results have recently been proposed (Quian Quiroga and Garcia, 2003; Spencer, 2005). In particular, a de-noising procedure based on the Wavelet Transform (wavelet de-noising) has led to new insights concerning trial-to-trial changes in ERPs due to factors such as habituation (Quian Quiroga and van Luijtelaar, 2002; Sambeth et al., 2003), learning (Jongsma et al., 2004) and skill training (Atienza et al., 2005; Talnov et al., 2003).

In this study we present a new paradigm, the 'learning-oddball' paradigm, which exploits the possibility of obtaining single-trial responses using wavelet de-noising. Such analysis allows us to track the dynamic process of auditory pattern learning. The 'learning-oddball' paradigm has been developed as a variant on an auditory oddball paradigm. In a typical oddball experiment, frequent background stimuli are occasionally replaced (at random intervals) by infrequently occurring deviant stimuli – the 'oddball' or target stimuli. The most striking feature of ERPs elicited by these unexpected target stimuli is the appearance of a 'P3' component (also referred to as the 'P300' or 'P3b' component), a positive wave appearing between 300 and 600 ms after target presentation, and with a maximum amplitude over the central posterior region of the brain (Katayama and Polich, 1999; Picton, 1992, 1996; Pritchard, 1981). The P3 appears to have multiple underlying generators with involvement of the temporal and parietal lobes (Bledowski et al., 2004; Kiss et al., 1989). In addition, the thalamus (Horovitz et al., 2002) and hippocampus (Halgren et al., 1998; McCarthy et al., 1989; Tarkka et al., 1995) have also been found to contribute to P3 generation.

Though less studied, unexpected target stimuli give also rise to a 'N2' component (also referred to as the 'N2b'), a centrally distributed negative wave appearing before the P3 (ca. 200 ms after target presentation). The N2 is considered to be intimately linked to the P3 (Daffner et al., 2000; Nuchpongsai et al., 1999; Naatanen et al., 1981). Though it has been hypothesized that there also exist non-identical generators for the N2 and P3, at least activity in the supramarginal gyrus has been found to contribute to both the N2 and P3 component (Smith et al., 1990). In addition, (Karakas et al., 2000) found that an interplay of theta- and delta oscillations produced the morphology of both the P3 and N2(b) component (Karakas et al., 2000).

With the 'learning-oddball' paradigm, we studied responses to eight targets presented in a random oddball sequence, followed by responses to eight targets presented in a fixed oddball sequence (see also Fig. 1). This alternation of random targets and regular, or patterned, targets was repeated several times ($n = 6$).

The P3 amplitude has long been known to be sensitive to a wide array of manipulations, such as target probability, the inter-stimulus intervals and inter-target intervals (Croft et al., 2003; Fitzgerald and Picton, 1981; Gonsalvez et al., 1995; Gonsalvez and Polich, 2002). In addition, the P3 amplitude is also sensitive to sequence effects, independent of probability effects, which appears to be caused by confirmation or disconfirmation of expectancies (Jentzsch and Sommer, 2001; Squires et al., 1976). Despite the theoretical and empirical implications of these findings, systematic assessment of increased predictability due to learning has not been investigated. However, the P3 and N2 can be expected to increase as the unexpectedness of a target increases. Accordingly, when learning a regular sequence, the expectation of regular targets increases, resulting in the decreased P3 - and N2 - amplitude (Donchin, 1981; Jentzsch and Sommer, 2001; Jongsma et al., 2005; Polich and Kok, 1995).

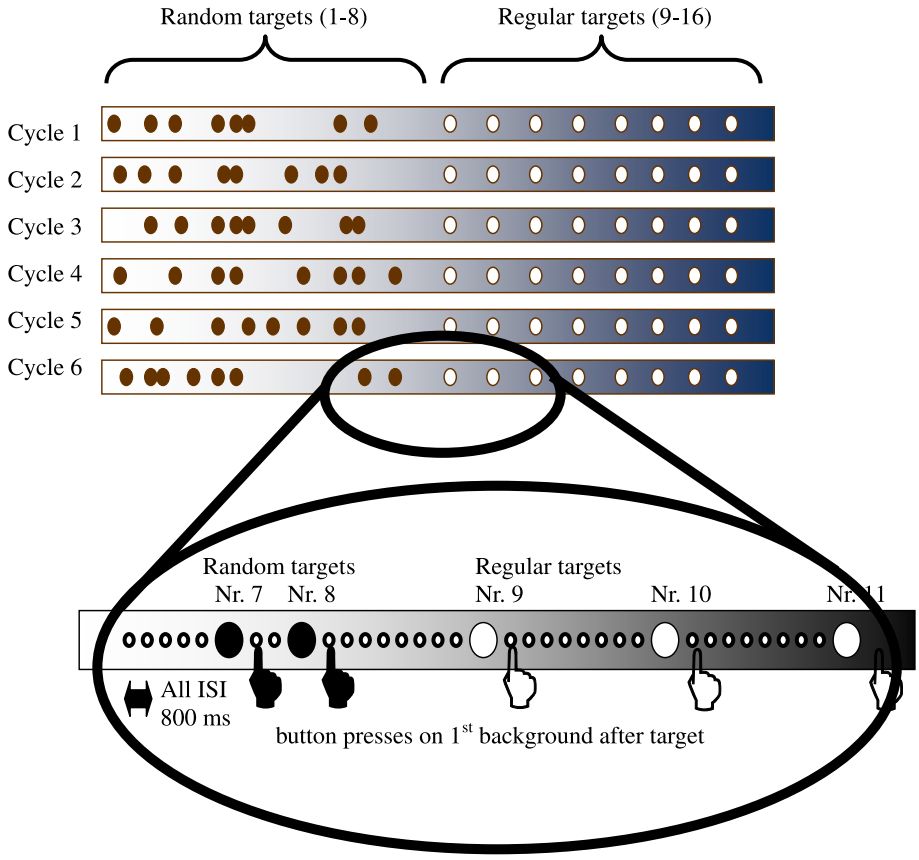

Fig. 1. This figure shows a diagram of the 'learning-oddball' paradigm. In this study eight targets were randomly (i.e., with 2–6 and 8–12 background tones in between to consecutive target tones) presented (depicted as black dots) within an ongoing steady sequence of auditory background tones (proportion targets:backgrounds, 1:8). Next, eight targets were presented at a regular position within the train of background tones (depicted as white dots). This alternation of random and regular target presentation was presented six times (cycles 1–6). Each cycle lasted 102.4 s, the total session lasted about 10 min. Part of the sequence has been magnified at the bottom. Targets were interspersed in an ongoing regular train of auditory background stimuli (small dots) with an 800 ms stimulus onset asynchrony (SOA). After eight targets, targets were presented regularly with seven background tones presented in between making the inter-target interval (ITI) 6.4 s. Participants had to give a mouse click after the first background stimulus following a target stimulus (depicted in pointing hands).

1.4. Hypotheses

Thus, in the current experiment we hypothesize that N2 and P3 responses to all random targets should be equal in case the target regularity is not perceived (i.e., the pattern is not learned). However, if pattern learning occurs, decreased single-trial N2 and P3 responses should be observed.

Additionally, if a target is expected, a slow negative shift in the ERP waveform should appear, starting about 300 ms before target stimulus onset – the 'Contingent Negative Variation,' or 'CNV' (Birbaumer et al., 1990; Walter et al., 1964), that is proposed to be generated by a network of cortical and subcortical structures (Bennett et al., 2004) including the basal ganglia (Zappoli, 2003). Although the CNV is ordinarily elicited in other paradigms than the oddball paradigm, the CNV appears to be sensitive to the expectancy of the target stimulus, and – like the P3 – has also been found to be sensitive to probability effects (Bauer et al., 1992; Korunka et al., 1993). We thus hypothesize that no CNV should be elicited in case the pattern is not learned. However, if pattern learning occurs, the elicitation of a CNV should be observed when target presentation becomes regularly spaced.

In this study, we first determined the conventional product of pattern learning, measuring the difference between the average performance to all random targets, compared to the average performance to all regular targets (see Fig. 2a). Second, we traced the dynamics of the learning process by measuring responses to all 16 consecutive targets (i.e., eight random targets, followed by eight regular targets; see Fig. 2b). Third, since the alternation of random and regular target presentation was repeated six times, we extracted single-trial responses to all 96 target stimuli individually. This allowed us to study whether pattern learning occurred more rapidly with consecutive repetitions (see Fig. 2c).

1.5. Main aim

Summarizing, we aimed to track the single trial-to-trial changes in the ERP CNV, N2, and P3 component

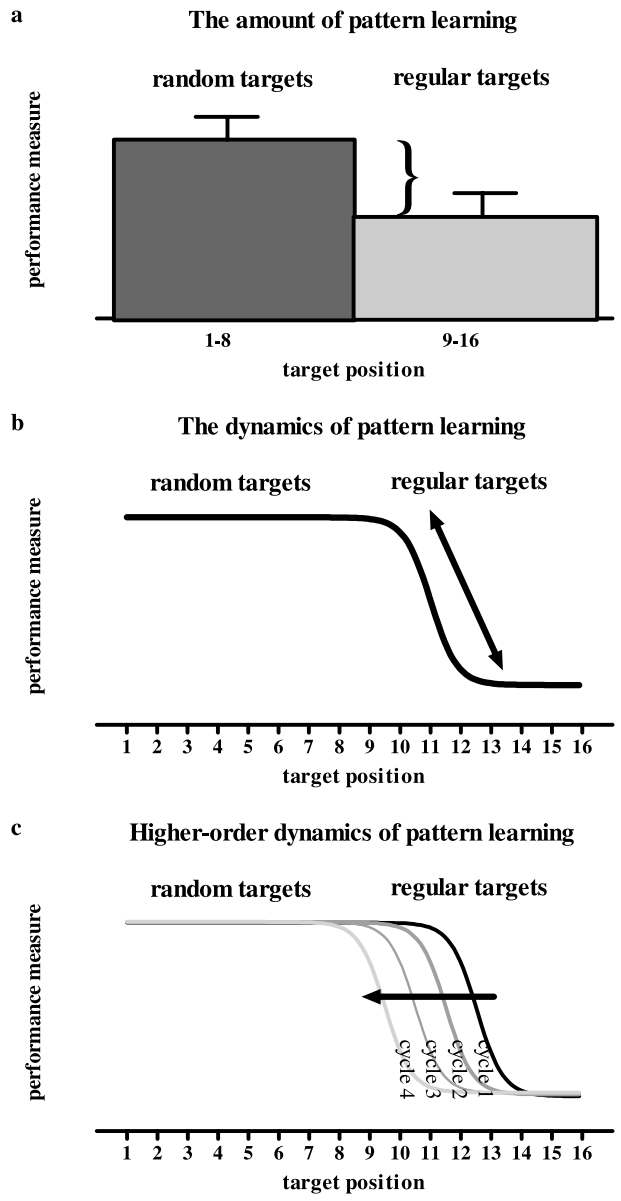

Fig. 2. This figure depicts the a priori defined hypotheses. The *x*-axes depicts target position (1–8 for random targets, 9–16 for regular targets) and the *y*-axes depicts the efficiency of performance (arbitrary units – lower values means better performance). (a) Depicts the alternative hypothesis regarding the average performance. (b) depicts the alternative hypotheses regarding the dynamics of pattern learning. (c) depicts the alternative hypotheses regarding higher-order dynamics of pattern learning. The H_0 hypotheses, namely, no difference between performances on random targets compared to regular targets are not depicted.

amplitudes, and to use them as dynamic markers of auditory pattern learning induced by switching from a pseudo-random to a fixed sequence of target stimuli. Though the N2, P3 and the CNV components have been under investigation for over 40 years, this is the first study – as far as we are aware – to investigate how the ERP components change from trial-to-trial during pattern learning.

2. Methods

2.1. Participants

Twenty-four participants (13 females, 11 males) took part in the experiment. Only right-handed healthy adults, not using medication and without a neurological or psychiatric history, were accepted. All participants signed a written statement of informed consent. They had a mean age of 27.4 ± 4.9 (mean $\pm$ SD) years. The participants sat comfortably in a recliner during the experiment and were instructed to keep their eyes closed and to sit as still as possible. This to avoid motor artifacts, in particular eye blinks. Both eye movements and blinking produce electric fields that overlap in time with the ERP of interest. Commonly, trials containing eye blinks are excluded from further analyses. However, since our aim was to track the trial-to-trial changes of ERP components due to pattern learning, no trials could be excluded. In addition, instructions to suppress eye blinking have also been found to affect the P3 amplitude (Ochoa and Polich, 2000). Thus – although this might have led to a general increase in ongoing alpha activity in the EEG – we measured ERPs during an eyes closed condition. Participants were tested in an electrically shielded, sound-attenuated, dark cubicle (inside dimensions: $2 \times 2.2 \times 2$ m). A computer mouse was placed under the participants' dominant hand to collect responses. The stimuli were presented through headphones. The sound consisted of woodblock sounds (duration: 200 ms with 5 ms rise/fall). All background stimuli had a center frequency at 2.45 kHz. Within the AEP session, target stimuli had a center frequency at 2.75 kHz. Within the OEPs session, targets consisted of missing stimuli. All stimuli were presented at a sound pressure level of 65 dB.

2.2. Experimental design

A visual image of the 'learning-oddball' paradigm is presented in Fig. 1. Two sessions, each lasting for about 10 min, were recorded. One session employed deviant stimuli as targets and one session employed omitted stimuli as targets. The presentation order of the sessions was counterbalanced among the participants. In both sessions, targets ($n = 96$) with a 12.5% probability, interspersed within a train of backgrounds (SOA 800 ms), were presented in an eyes closed situation. Within one session, six blocks of 16 consecutive targets were presented as one continuous, ongoing train of stimuli (of 96 targets and 672 background tones). The first eight targets were presented in a random position (preceded by a semi-random (2–6 or 8–12) number of background tones), the following eight targets presented in a fixed position (all preceded by seven background tones). Thus, targets presented in fixed positions became predictable. The program E-Prime was used for presenting the stimuli. The program was set up in such a way that it generated eight strings containing a 'random' target. This was done by presenting a semi-random number of

background stimuli (2–6 or 8–12), followed by one target stimulus. This way, a random target was never preceded by seven background stimuli. However, occasionally two random targets were preceded by the same number of background tones. After generating eight random target strings, eight fixed target strings were generated by presenting seven background stimuli followed by one target stimulus. The program was started separately for each individual participant resulting in different random sequences. The task of the participants was to respond after the first standard tone following a target stimulus. This delayed response task was chosen in order to avoid motor activity closely locked to targets.

EEG recordings. EEG (band-pass: DC – 100 Hz, sampling rate 500 Hz) was recorded with a SYNAMPS amplifier (Neuroscan, Herndon, VA) from 27 Ag/AgCl electrodes (AF3, AF4, F7, F3, Fz, F4, F8, FC5, FC1, FC2, FC6, T7, C3, Cz, C4, T8, CP5, CP1, CP2, CP6, P7, P3, Pz, P4, P8, PO3, PO4) mounted in an elastic cap (EASICAP, FMS, Breitenbrunn, GER) at placements based on the International 10–20 recording system (American Encephalographic Society, 1994) and referenced to linked mastoids and stored on disk for offline processing. Vertical and horizontal eye movements were recorded by two additional bipolar channels placed above and below the right eye and on the outer canthi of each eye. Impedances of all electrodes was kept below 10 kΩ.

2.3. Data processing

First, epochs from −2048 ms to 2048 ms around all presented auditory target stimuli ($n = 96$) and all presented omitted target stimuli ($n = 96$) were off-line down-sampled to 250 Hz, de-trended and baseline corrected over the full 4.096 s length of each epoch. Second, these epochs were then collectively subjected to an independent component analysis, as implemented in EEGLAB (Delorme and Makeig, 2004), running in the MATLAB environment (The Mathworks, Inc., Natick, MA). Variance-components with activity attributable to artifacts, such as eye movement, fronto-temporal muscle activity and mains noise, were removed. We did not employ ICA components beyond the level of artifact reduction for several reasons. Within the current experiment the ICA output does not necessarily add to the clarity of the results. Apart from this, since we ran ICA separately for each individual, there would be a matching problem of ICA's across subjects.

Finally, all individual single-trial ERP responses were de-noised by means of a recently proposed algorithm based on the wavelet transform analysis method (Quian Quiroga and Garcia, 2003). The accuracy of this method in smoothing ERPs has been demonstrated with both simulated data and visual and auditory ERP data (Atienza et al., 2005; Jongsma et al., 2004; Quian Quiroga and Garcia, 2003; Quian Quiroga and van Luijtelaar, 2002; Sambeth et al., 2003; Spencer, 2005; Talnov et al., 2003). De-noising parameters were the same for all participants. Whole waveforms were additionally smoothed using a 3-point moving average across adjacent epochs. The above described data processing was applied to each channel independently. Under the assumption that scalp potentials are coupled to hemodynamic activity, single-trial ERPs can also be used for prediction of regional brain activation in functional magnetic resonance data (Eichele et al., 2005). Since some peaks diminished or appeared rapidly over consecutive targets, peak amplitudes were determined within a fixed latency window based on the grand average responses (of all subjects, for all targets, for AEPs and OEPs separately) and subsequently kept constant (Jongsma et al., 2005).

AEPs elicited by deviant target stimuli consisted, alongside an N1 and P2, of a prominent N2 and P3 component. In addition, a CNV-like component was observed. Mean component amplitudes in fixed latency windows (N2, 180–220 ms; P3, 350–430 ms; CNV, −300 ms to 0 ms) were further analyzed (Jongsma et al., 2005; Koelsch et al., 2004). In line with this, grand average OEPs elicited by omitted target stimuli also consisted of a N2, P3 and CNV-like component. Mean component amplitudes in the same fixed latency windows (N2, 180–220 ms; P3; 350–430 ms; CNV, −300 ms to 0 ms) were further included in analyses (Lang and Kotchoubey, 2000). In addition, since the N2 is considered to be intimately linked to the P3 (Daffner et al., 2000; Nuchpongsai et al., 1999; Naatanen et al., 1981), a P3–N2 component was constructed by subtracting the N2 amplitude from the P3 amplitude. This resulted in a more stable component, especially with regard to the single-trial ERP analyses.

2.4. Statistical analysis

Step 1. See also Fig. 2a. Component amplitudes of de-noised ERPs were determined as the average value within a fixed latency window (N2, 180–220 ms; P3, 350–430 ms; CNV, −300 to 0 ms). For each component (the AEP CNV, N2, P3 and P3–N2 component and the OEP CNV, N2, P3 and P3–N2 component) a two-within ANOVA analysis was performed: condition × electrode site, (condition, two levels: random targets vs fixed targets; electrode site, 27 levels). For EEG channels it is clear that nearby channels are generally more correlated than distant channels, thus leading to heterogeneous covariances. Therefore, the Geisser and Greenhouse correction was applied to the degrees of freedom. Post-hoc analyses applying Bonferroni correction were performed to determine condition effects per electrode site when appropriate. Reaction times of correct responses were analyzed. Correct responses were defined as responses within a time window of −400 ms to +400 ms around the point of optimal response (i.e., the first background stimulus after the target stimulus). Error rates were calculated and the RTs of the correct responses were analyzed with a *t*-test (random targets vs fixed targets).

Step 2. See also Fig. 2b. A priori defined hypotheses were tested by nonlinear regression analysis of the AEP

CNV, N2, P3 and P3–N2 and AEP RTs, as well of the OEP CNV, N2, P3 and P3–N2 component and OEP RTs, using the program GraphPad Prism 4. The over participants averaged RTs were smoothed using a 3-point moving average. For AEP and OEP component amplitudes, *F*-tests for best fit were obtained for all 27 electrode sites comparing:

H_0: a straight line with slope = zero. There is no learning effect.
H_1: a sigmoid-curve. With each cycle, 'learning' occurs more rapidly.

Step 3. See also Fig. 2c. Single-trial component amplitudes were determined at group level ($n = 24$). For the CNV component at Fz (maximal effect) and for the P3–N2 component at Pz (maximal effect). In addition, single-trial RTs were determined at group level ($n = 24$). Thus, each separate cycle ($n = 6$), containing 16 targets, was analyzed, resulting in six sigmoid-curves per session, per component and per RT. In addition, a regression analysis was performed with *F*-tests for best fit on found Tn50s – or the point where 50% of the amplitude modulation was reached.

Thus, regression analysis was applied on the estimated Tn50 values obtained in step 2 of the analysis, using the program: GraphPad Prim 4.03. The linear equations of the H_0 ($Y = \text{Intercept}$, there are no over-cycle effects) and the H_1 hypothesis ($Y = \text{Intercept} + \text{Slope} \times X$, with each cycle, 'learning' occurs more rapidly) were fitted to the data. An *F*-test determined whether the decrease of sum of squares for the H_1 was worth the loss of degrees of freedom. For the fitting and the *F*-tests three procedures were applied on each ERP component: the first most conservative procedure took into account only the number of cycles (i.e., 6) and consequently yielded five (H_0) or four (H_1) degrees of freedom. In the second procedure the significance was tested with 23 vs 22 degrees of freedom, reflecting the number of participants. In the last procedure the total number of measurement (i.e., 146, namely: subjects × cycles) was taken into account, resulting in 143 vs 142 degrees of freedom. An overview of these results together with the raw data in an Excel file are available from the author upon request.

3. Results

3.1. The amount of pattern learning

Fig. 3a shows the grand average ERPs to higher pitch targets, or auditory event-related potentials (AEPs) and to stimulus omissions, or omission event-related potentials (OEPs) at midlines electrode sites (Fz, Cz and Pz) for both random (dotted lines) and regular (solid lines) targets. Though all 27 electrode sites were included in the analyses, only results from midline sites (Fz, Cz and Pz) are depicted in the figures.

We observed a slow negative shift in ERPs elicited by the regular targets – the contingent negative variation (CNV) – starting about 500 ms before target onset. CNV was at a maximum in the central frontal region and showed larger negative amplitudes to regular targets than to random targets.

Random target stimuli elicited an N2, appearing between 180 and 220 ms after stimulus onset, with a maximum amplitude over the central region and a marked P3 component – a positive wave appearing between 350 and 430 ms after target presentation – with a maximum amplitude over the central posterior region of the brain (Picton, 1992; Polich, 1996; Pritchard, 1981; Sutton et al., 1965). A similar P3–N2 complex was also observed when the target consisted of an unexpectedly omitted stimulus (Besson and Faïta, 1995; Jongsma et al., 2004, 2005; Walter et al., 1964). Maximum P3–N2 was expressed at the central posterior region and had lower amplitudes (i.e., closer to baseline) with regular targets than with random targets.

Fig. 3b depicts the means and SEMs of concurrent ERP component amplitudes for midline sites in the form of bar graphs. Component amplitudes to regular targets are depicted as solid bars, and component amplitudes to random targets are depicted as dotted bars. Bar graphs are shown for the AEP session (on the left) and the OEP session (on the right). The *y*-axes show amplitudes (in μV). Bar graphs of the concurrent reaction times (RTs) are shown on the lower right-hand side, with *y*-axes showing time (in ms).

Table 1 summarizes all significant *F* and *p*-values from the ANOVA and *t*-test results for the AEP session (Table 1a) and the OEP session (Table 1b).

With respect to the AEP session, the CNV showed a significant effect of condition ($p < .0001$), electrode site ($p = .002$), and a condition × electrode site interaction effect ($p < .0001$). Post-hoc analyses revealed condition effect at frontal-central sites (AF3, AF4, F2, F3, Fz, F4, FC5, FC1, FC2, FC6, T7, Cz, C4, CP5 and CP1; with Bonferroni correction, all $p < .05$). On the N2 we found no main effects of condition or electrode site, but we did find a condition × electrode site interaction effect ($p = .016$). However, in the post-hoc analyses none of the electrodes showed a condition effect after Bonferroni correction. On the P3 a main condition ($p < .0001$) and electrode site effect ($p = .011$) was observed. The condition × electrode site effect did not reach significance ($p = .34$). The P3–N2 showed an effect of condition ($p < .0001$), electrode site ($p = .004$), and a condition × electrode site interaction effect ($p < .033$). Post-hoc analyses revealed condition effect at parietal sites (CP1, P3, Pz and P4; with Bonferroni correction, all $p < .05$).

With respect to the OEP session, the CNV showed a significant effect of condition ($p < .0001$), electrode site ($p < .001$), and a condition × electrode site interaction effect ($p < .0001$). Post-hoc analyses revealed condition effect at frontal-central sites (AF3, AF4, F3, Fz, F4, FC5, FC1, FC2, FC6, T7, Cz, C4, CP5, CP1, CP2 and CP6; with Bonferroni correction, all $p < .05$). On the N2 we found a main effect of electrode site ($p = .021$). No effect on

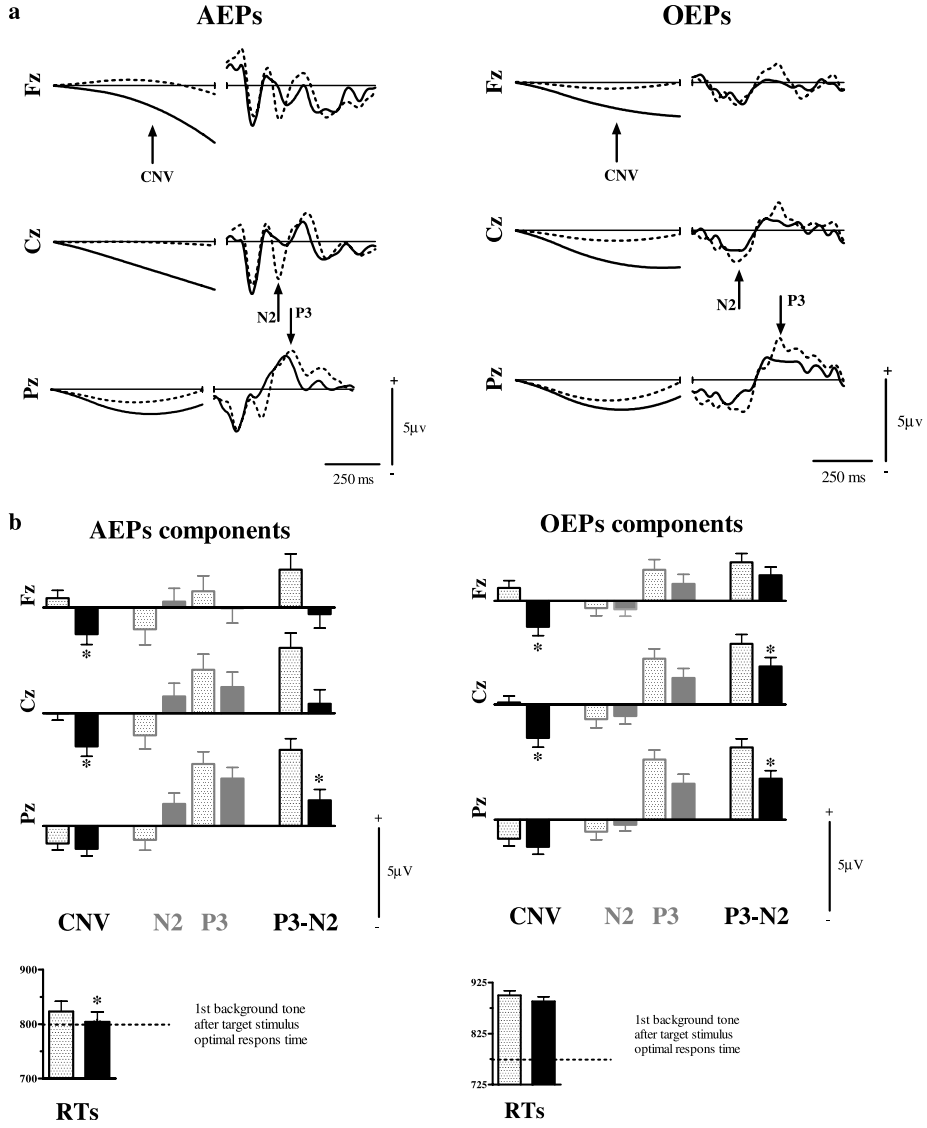

Fig. 3. (a) Shows grand average ERPs at midline sites. On the left side of each electrode panel, the tracings after the first wavelet-denoising solution, extracting the CNVs, are depicted. On the right side, tracings after the second wavelet-denoising solution, extracting the ERP N2, P3 and P3–N2 complex, are depicted. Grand averages of all 24 participants are given for all regular targets (solid lines) and all random targets (dotted lines). Tracings are given for the AEP session (on the left) and the OEP session (on the right). The *x*-axes show time (in ms) and the *y*-axes show amplitudes (in μV). (b) Depicts bar graphs (means and SEMs) of concurrent ERP component amplitudes at midline sites of ERP CNV, N2, P3 and P3–N2 complex. Group averages are given for all regular targets (solid bars) and all random targets (dotted bars). Bar graphs are given for the AEP session (left panel) and the OEP session (right panel). The *y*-axes show amplitudes (in μV). Bar graphs of the concurrent delayed response RTs are depicted at the lower right side with *y*-axes showing time (in ms).

condition, nor a condition × electrode site effect was observed. The P3 showed a main condition ($p < .0001$) and electrode site effects ($p = .013$). The condition × electrode site effect did not reach significance ($p = 0.10$). On the P3–N2 we found a significant effect of condition ($p < .006$) and electrode site ($p < .0001$). As for the P3, the condition × electrode site interaction effect did not reach significance ($p = 0.12$). An explorative post-hoc analysis suggested only condition effect at centro-parietal sites (Cz, CP1, CP2, CP6, P3 and Pz; with Bonferroni correction, all $p < .05$).

The error rates suggest that the task was more difficult within the OEP session than the AEP session (13.05% vs 5.15%). Also, error rates of the responses seemed slightly higher for random targets than regular targets (5.50% vs 4.80% within the AEP session; 13.30% vs 12.80% within the OEPs session). *T*-tests regarding the reaction times showed a significant effects in the AEP session ($p = .021$) and the OEP session ($p = .038$), with faster RTs during regular target sequences.

3.2. The dynamics of pattern learning

Fig. 4 shows component amplitudes and their best-fit curves (either a straight line or a sigmoid-curve) for the CNV, N2, P3 and P3–N2 component amplitudes at midline

Table 1
The amount of pattern learning

AEP	ANOVA results	*F* values	*p*-values
(a) Summary ANOVA's AEP component amplitudes			
CNV	Electrode site effect	$F(2.5, 57.9) = 16.2$	$p = .002$
	Condition effect	$F(1, 23) = 389.9$	$p < .0001$
	Interaction effect	$F(3.09, 71.0) = 4.3$	$p < .0001$
Post-hoc	Bonferroni corrected		
AF3	Condition effect		$p < .05$
AF4			
F2			
F3			
Fz			
F4			
FC5			
FC1			
FC2			
FC6			
T7			
Cz			
C4			
CP5			
CP1			
N2	Electrode site effect	$F(2.1, 48.9) = 26.6$	$p = .07$
	Condition effect	$F(1, 23) = 32.4$	$p = .23$
	Interaction effect	$F(3.16, 72.7) = 3.6$	$p = .016$
Post-hoc	Bonferroni corrected		n.s.
P3	Electrode site effect	$F(2.8, 64.7) = 12.4$	$p < .0001$
	Condition effect	$F(1, 23) = 7.6$	$p = .011$
	Interaction effect	$F(3.0, 68.7) = 11.6$	$p = .34$
P3-N2	Electrode site effect	$F(2.3, 53.8) = 9.8$	$p < .0001$
	Condition effect	$F(1, 23) = 10.6$	$p = .004$
	Interaction effect	$F(2.8, 64.6) = 3.2$	$p = .033$
Post-hoc	Bonferroni corrected		
CP1	Condition effect		$p < .05$
P3			
Pz			
P4			
	t-test		*p*-value
RT	Condition effect		$p = .021$
OEP	ANOVA results	*F* values	*p*-values
(b) Summary ANOVA's OEP component amplitudes			
CNV	Electrode site effect	$F(3.2, 74.4) = 6.32$	$p = .001$
	Condition effect	$F(1, 23) = 20.9$	$p < .0001$
	Interaction effect	$F(3.4, 79.1) = 7.4$	$p < .0001$
Post-hoc	Bonferroni corrected		
AF3	Condition effect		$p < .05$
AF4			
F3			
Fz			
F4			
FC5			
FC1			
FC2			
FC6			
T7			
Cz			
C4			
CP5			
CP1			
CP2			
CP6			
N2	Electrode site effect	$F(3.8, 86.7) = 3.1$	$p = .021$
	Condition effect	$F(1, 23) = 14.1$	$p = .27$
	Interaction effect	$F(2.9, 66.5) = 4.8$	$p = .40$
P3	Electrode site effect	$F(3.8, 88.0) = 12.91$	$p < .0001$
	Condition effect	$F(1, 23) = 7.2$	$p = .013$
	Interaction effect	$F(2.0, 45.5) = 31.0$	$p = .10$
P3-N2	Electrode site effect	$F(3.7, 84.5) = 11.6$	$p < .0001$
	Condition effect	$F(1, 23) = 9.1$	$p = .006$
	Interaction effect	$F(1.8, 41.1) = 52.0$	$p = .115.$
Post-hoc	Bonferroni corrected		
Cz	Condition effect		$p < .05$
CP1			
CP2			
CP6			
P3			
Pz			
	t-test		*p*-value
RT	Condition effect		n.s.

Summarizes *F* and *p* values of 3-within ANOVA results for the AEPs CNV, N2, P3 and P3–N2 amplitudes and the *t*-test result of the reaction times (RTs).

sites – as well as the RTs – for the AEP session (Fig. 4a) and the OEP session (Fig. 4b). Table 2summarizes *F* and *p*-values for the *F*-tests in terms of best fit for all learning curves at all 27 electrode sites. With respect to the AEP CNV, N2, P3, P3–N2 component all components showed 'learning' curves at most electrode sites that described the data significantly better than straight horizontal lines (levels of significance: $^{***}p < .0001$; $^{**}p < .01$; $^{*}p < .05$; n.s. = not significant). Significances for the CNV appeared to be more frontal-central orientated and for the N2, P3 and P3–N2 more central-parietal orientated. The RT data could also be better described by a 'learning' curve than a straight, horizontal line. Similar results were found with respect to the OEPs though the N2 'learning' curves only appeared to reach significance at more laterally orientated temporal-occipital sites.

For the AEP session, the CNV increased in the pre-stimulus period at all frontal central EEG electrode sites (see Table 2). In addition, the N2, P3 and P3–N2 amplitudes rapidly decreased after target presentation became regular (marked with a solid line), resulting in the hypothesized sigmoid learning curve at all central posterior EEG electrode sites (see Table 2). A corresponding effect was seen in the RTs that also decreased after target presentation became regular, though the effect was less clear, and appeared later.

In line, for the OEP session, the CNV increased in the pre-stimulus period at all frontal central EEG electrode sites (see Table 2). In addition, the P3 amplitude rapidly decreased after target presentation became regular (marked

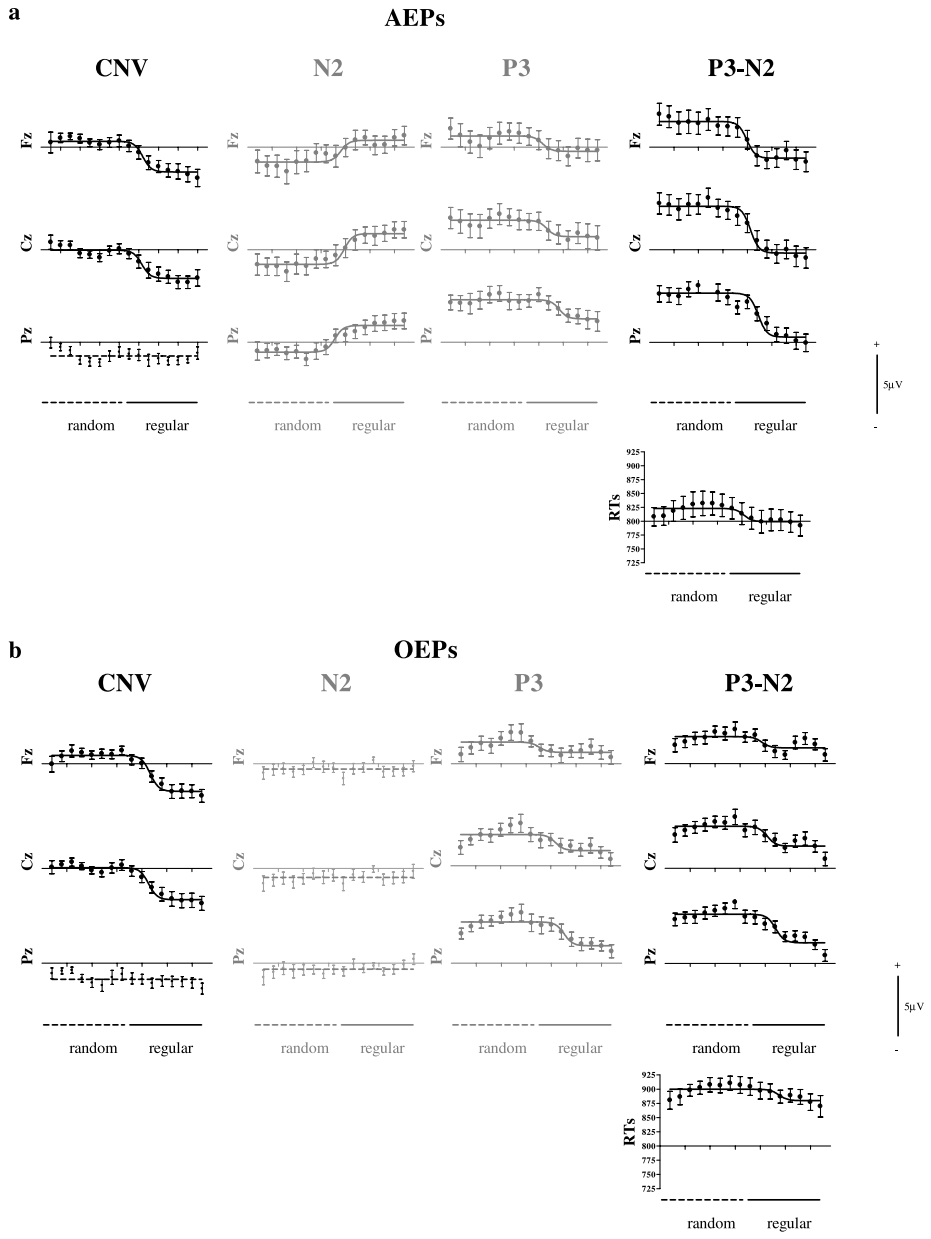

Fig. 4. This figure shows 'learning curves,' or s-curves, at all midline sites for the AEP CNV, N2, P3 and P3–N2 amplitudes and the RTs from the AEP session (a), and the OEP session (b). The *x*-axes show concurrent target position (1–16) with random targets falling on the left side (1–8) and regular targets on the right side. The *y*-axes depict ERP component amplitudes (in μV) and time (in ms) for the RTs. Only when a sigmoid-curve described the data significantly better then a straight line it was plotted with a black solid line. Non-significant sigmoid-curve solutions are depicted with a dotted thin line.

with a solid line), resulting in the hypothesized sigmoid learning curve at all central posterior EEG electrode sites (see Table 2). Although the N2 did not decrease at midline sites, the combined P3–N2 returned stronger and more stable decreases than P3 amplitudes alone. RTs failed to show a significant decrease.

3.3. Higher-order dynamics of pattern learning

Fig. 5 shows the group-averaged single-trial data with the best-fit curves per cycle presented (either a straight horizontal line or a sigmoid-curve) for the CNV at the Fz electrode site (Fig. 5a left panel for AEPs; Fig. 5b left panel for OEPs), and P3–N2 at the Pz electrode site (Fig. 5a middle panel for AEPs, Fig. 5b middle panel for OEPs), along with the RTs (Fig. 5a right panel for AEPs session; Fig. 5b right panel for OEPs session). Table 3 summarizes the significance of *F*-tests for best fit of group-averaged AEPs and OEPs component amplitudes and RTs per trial over six consecutive cycles. With respect to both the AEP and OEP components, the single-trial CNV amplitudes (at Fz) and P3–N2 amplitudes (at Pz) resulted in significant 'learning' curves for all six cycles (all $p < .05$). Moreover, turning point of these 'learning' curves (the Tn50s),

Table 2
The dynamics of pattern learning

Site	CNV	N2	P3	P3–N2	RT
AEP					
(a) Summary of group statistics of fitting 'learning' curves on AEP component amplitudes					
AF3	***	n.s.	n.s.	**	
AF4	***	n.s.	n.s.	***	
F7	***	n.s.	n.s.	n.s.	
F3	***	**	*	***	
Fz	*	***	*	***	
F4	***	***	*	***	
F8	***	n.s.	n.s.	*	
FC5	***	**	*	***	
FC1	***	***	*	***	
FC2	***	***	*	***	
FC6	***	***	n.s.	***	
T7	***	***	n.s.	***	
C3	***	***	*	***	
Cz	***	***	*	***	*
C4	***	***	*	***	
T8	***	***	n.s.	***	
CP5	***	***	**	***	
CP1	***	***	**	***	
CP2	***	***	**	***	
CP6	***	***	*	***	
P7	n.s.	***	***	***	
P3	**	***	***	***	
Pz	n.s.	***	***	***	
P4	*	***	***	***	
P8	n.s.	***	***	***	
PO3	n.s.	***	***	***	
PO4	n.s.	***	***	***	
OEP					
(b) Summary of group statistics of fitting 'learning' curves on OEP component amplitudes					
AF3	***	n.s.	n.s.	n.s.	
AF4	***	n.s.	n.s.	n.s.	
F7	***	n.s.	n.s.	n.s.	
F3	***	n.s.	n.s.	n.s.	
Fz	*	n.s.	*	**	
F4	***	n.s.	*	**	
F8	***	n.s.	n.s.	n.s.	
FC5	***	n.s.	n.s.	*	
FC1	***	n.s.	**	***	
FC2	***	n.s.	**	***	
FC6	***	n.s.	**.	***	
T7	***	**.	n.s.	**	
C3	***	n.s.	***	***	
Cz	***	n.s.	***	***	n.s.
C4	***	n.s.	***	***	
T8	***	***.	n.s.	***	
CP5	***	***	***	***	
CP1	***	n.s.	***	***	
CP2	***	n.s.	***	***	
CP6	***	***	***	***	
P7	n.s.	***	***	***	
P3	***	***	***	***	
Pz	n.s.	n.s.	***	***	
P4	***	*	***	***	
P8	**	n.s.	***	***	
PO3	n.s.	*	***	***	
PO4	n.s.	**	***	***	

F and *p*-values of *F*-tests for best fit of AEPs and OEPs component amplitudes at all electrode sites and RTs from both AEP and OEP session (***$p < .001$; **$p < .01$; *$p < .05$).

appeared to occur earlier with each repetition Thus, additionally, a regression analysis was performed with *F*-tests for best fit on the mean and 95% confidence intervals of these Tn50s – or the point where 50% of the amplitude modulation was reached – from each of these curves, comparing:

H_0: a straight line with slope is equal to zero. There are no over-cycle effects.
H_1: a straight line with slope is not equal to zero. With each cycle, 'learning' occurs more rapidly.

Table 3 (bottom) summarizes the significant results from the *F*-tests for best fit of Tn50s from six consecutive cycles. Tn50s of all 'learning' curves were taken separately for AEP and OEP component amplitudes and RTs per cycle.

Though all separate cycles showed significant learning curves at the single-trial ERP CNV and P3–N2 component amplitudes, some cycles showed decreases even before the introduction of target regularity; possibly due to increased variability of the single-trial data. However, in general, these learning curves showed progressively earlier Tn50s suggesting that the regularity of the pattern was detected more rapidly with each consecutive cycle (see Fig. 5, bottom panels). RTs did not show significant learning curves for all cycles (only for cycles 3 and 4 for AEPs, cycles 2, 3 and 4 for OEPs). Therefore, no *F*-test for straight lines with either slope is equal to zero vs slope is different from zero on the RTs' Tn50s could be performed.

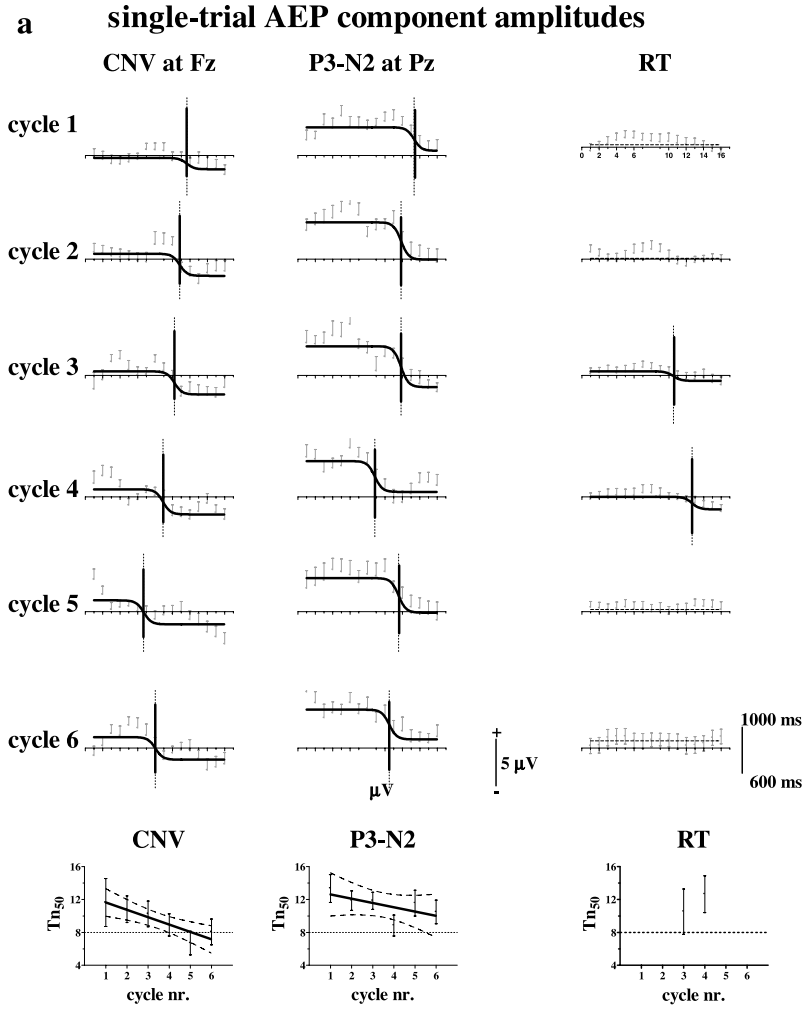

Fig. 5. This figure shows the six sigmoid-curves for the six consecutive cycles, thus showing ERP component amplitudes of all 96 single-trials. The sigmoid-curves for the ERP CNV are derived from the Fz electrode site and the sigmoid-curves for the ERP P3–N2 are derived from the Pz electrode site. Only sigmoid-curves that described the data significantly better then a straight line are depicted in solid black lines. For each 'learning' curve, the Tn50 is marked with a solid vertical line. The *x*-axes plot target position (1–16 for cycle 1; 17–32 for cycle 2; 33–48 for cycle 3; 49–64 for cycle 4; 65–80 for cycle 5; 81–96 for cycle 6). The *y*-axes depict ERP component amplitudes (in µV) and time (in ms) for the RTs. Single-trial 'learning' curves are given for the AEP session (a) and the OEP session (b). In addition, Tn50s (means and 95% confidence intervals) are plotted for the ERP CNV, P3–N2 and the RTs for both the AEP session (a, bottom) and the OEP session (b, bottom). The *x*-axes depict cycle nos. (1–6) and the *y*-axes depict values of the Tn50 (as expressed in target position, 1–16) per cycle. A regression analysis was performed comparing a straight line with slope is equal to zero with a straight line with slope is different from zero.

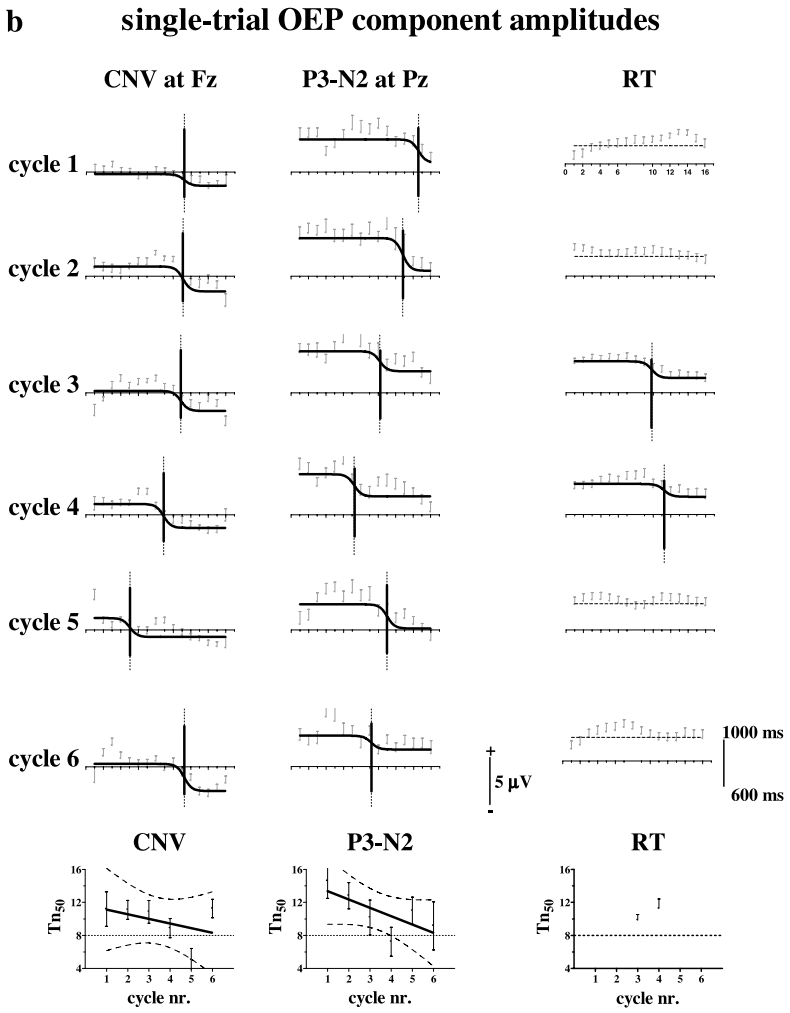

Fig. 5 (*continued*)

4. Discussion

4.1. The amount of pattern learning

We found slightly faster RTs (ca. 20 ms) following regularly presented targets, compared to RTs following randomly presented targets. Other ERP studies found more marked RT effects (ca. 60 ms difference) with pattern learning (Baldwin and Kutas, 1997; Eimer et al., 1996; Rüsseler and Rösler, 2000; Schlaghecken et al., 2000). This is probably due to the fact that in these studies, responses were given immediately following targets (Baldwin and Kutas, 1997; Eimer et al., 1996; Rüsseler and Rösler, 2000; Schlaghecken et al., 2000) whereas in our study participants responded to the background stimulus following 800 ms after target presentation.

In addition, a central anterior distributed CNV was observed in ERPs elicited by the regularly presented targets, but not in ERPs elicited by randomly presented targets. Although such a CNV is commonly elicited in paradigms in which a warning stimulus precedes a target stimulus – thereby making the appearance of the target stimulus expected (Bennett et al., 2004; Birbaumer et al., 1990; Walter et al., 1964) – the P3 and CNV have been strongly associated in the classical ERP literature due to their common relationship to expectation and expectation-related constructs (Donchin, 1981; Korunka, 1993; Sutton et al., 1965) and their common sensitivity to target probability (Korunka, 1993).

In a similar experiment, Eimer and colleagues (1996) observed a slow negative shift preceding targets – the 'Lateralized Readiness Potential' (LRP) – which became more pronounced with learning. They assumed that this component reflected the acquired knowledge, and stated that participants may have learned the stimulus sequence and that expectations were communicated to the motor system at a very early stage of processing, namely, prior to target presentation. However, their participants had to respond directly after a target stimulus was presented, whereas in the current experiment a delayed response was required, thus eliminating the motoric component of response preparation. Because a delayed response task was

Table 3
Higher order dynamics of pattern learning

cycle	CNV	N2	P3	P3–N2	RT
(a) Summary of the Tn50s (turning point) of 'learning' curves per cycle for AEP component amplitudes (mean ± 95% confidence intervals)					
1	11.6 (8.86–14.4)	13.3 (11.3–15.4)	n.s.	13.3 (11.7–14.9)	n.s.
2	10.8 (9.28–12.3)	11.8 (10.7–12.9)	n.s.	11.9 (10.7–13.0)	n.s.
3	10.2 (8.76–11.7)	9.68 (8.61–10.8)	n.s.	11.9 (10.9–12.8)	10.5 (7.93–13.1)
4	8.92 (7.64–10.2)	6.05 (4.06–8.04)	10.1 (8.61–11.6)	8.84 (7.64–10.0)	12.7 (10.5–14.8)
5	6.71 (5.35–8.06)	9.36 (7.16–11.6)	12.2 (10.5–14.0)	11.6 (10.1–13.0)	n.s.
6	8.05 (6.57–9.53)	n.s.	7.96 (6.27–9.65)	10.5 (9.12–11.9)	n.s.
Higher order					
F(Dfn, Dfd) based on no. of cycles	20.5 (1,4) $p = 0.010$	4.82 (1,3) $p = 0.12$ n.s.	–	2.77 (1,4) $p = 0.17$ n.s.	–
F(Dfn, Dfd) based on no. of subjects	18.7;(1,22) $p = 0.0003$	14.3 (1,18) $p = 0.0014$	–	7.01 (1,22) $p = 0.01$	–
F(Dfn, Dfd) based on no. of measurements	18.4 (1,142) $p < 0.0001$	21.4 (1,118) $p < 0.0001$	–	9.84 (1,142) $p = 0.002$	–
(b) Summary of the Tn50s (turning point) of 'learning' curves per cycle for OEP component amplitudes (mean ± 95% confidence intervals)					
1	11.2 (9.22–13.2)	n.s.	n.s.	14.6 (12.6–16.6)	n.s.
2	11.1 (10.0–12.2)	n.s.	12.3 (11.3–13.3)	12.8 (11.3–14.3)	n.s.
3	10.9 (9.56–12.2)	n.s.	10.3 (7.91–12.6)	10.2 (8.18–12.2)	9.84 (8.54–11.2)
4	8.88 (7.77–10.0)	n.s.	13.1 (12.3–13.9)	7.25 (5.59–8.91)	11.3 (9.03–13.5)
5	5.04 (3.74–6.34)	n.s.	12.1 (10.6–13.6)	11.0 (9.42–12.6)	n.s.
6	11.3 (10.2–12.3)	n.s.	9.67 (7.21–12.1)	9.16 (6.40–11.9)	n.s.
Higher order					
F(Dfn, Dfd) based on no. of cycles	0.913 (1,4) $p = 0.39$ n.s	–	0.495 (1,3) $p = 0.53$ n.s.	4.48 (1,4) $p = 0.1$	–
F(Dfn, Dfd) based on no. of subjects	3.74 (1,22) $p = 0.07$	–	1.11 (1,18) $p = 0.30$ n.s.	11.6 (1,22) $p = 0.003$	–
F(Dfn, Dfd) based on no. of measurements	8.96 (1,142) $p = 0.003$	–	1.41 (1,118) $p = 0.24$ n.s.	16.6 (1,142) $p < 0.0001$	–

Summarizes the over participants averaged Tn50s (means and 95% confidence intervals) of learning curves. (Target 9 being the first regular target) for the AEP session (a) and the OEP session (b).

employed in the current experiment, no lateralized readiness potential was elicited by the target stimulus. Instead, regularly presented target stimuli started to function as a kind of warning stimulus, after which a response was then acquired. Interestingly, a CNV-like potential was observed preceding these 'warning' targets. Although conventionally the CNV develops before an expected motor response, it also develops before an expected stimulus that does not require a motor response (Hohnsbeim et al., 1998). Thus, the CNV is assumed to reflect the cognitive preparation of the next trial, or the facilitation of specific brain area, which are relevant for the next trial (Hohnsbeim et al., 1998). The CNV has also been shown to develop during the temporal interval between two events. This wave would reflect anticipatory processing of a temporally expected stimulus. The CNV is thus the main ERP correlate of the estimation or production of a time interval and has been shown to increase during the learning of a temporal interval (McAdam, 1966; Pfeuty et al., 2003). This is in line with our observations, where the temporal interval between regular targets (6.4 s) was learned.

Finally, we found that the central posterior distributed P3–N2 complex was smaller in response to regularly presented targets as compared to randomly presented targets in both the oddball and omission sessions. The CNV was maximal for the 'regular' condition over frontal sites. As expected, we did observe a condition × electrode site interaction effect revealing a CNV condition effect over frontal sites for both the AEP and OEP session. Since the P3–N2 was maximal for the 'random' condition over centro-parietal sites, an interaction effect was also expected. We did observe an interaction effect within the AEP session revealing a P3–N2 condition effect over parietal sites. For the OEP session the interaction effect was not significant. This might be due to the fact that the OEP P3–N2 is a very broad and smeared component that is visible over most electrode sites, with exception of some frontal sites. Compared to the current experiment, Eimer and colleagues (1996) described similar N2 effects due to implicit pattern learning. In addition, others have reported decreased P3 due to learning (Rose et al., 2001), high expectancy (Sutton et al., 1965) and regularity (Lang and Kotchoubey, 2000). Correspondingly, in a previous study we found that the P3–N2 complex rapidly disappeared when omitted target stimuli could be expected (Jongsma et al., 2005).

Both the P3 and CNV appear to be sensitive to (local) probability effects (Bauer et al., 1992; Croft et al., 2003; Fitzgerald and Picton, 1981; Korunka et al., 1993). Therefore, we wanted to explore whether our results could be

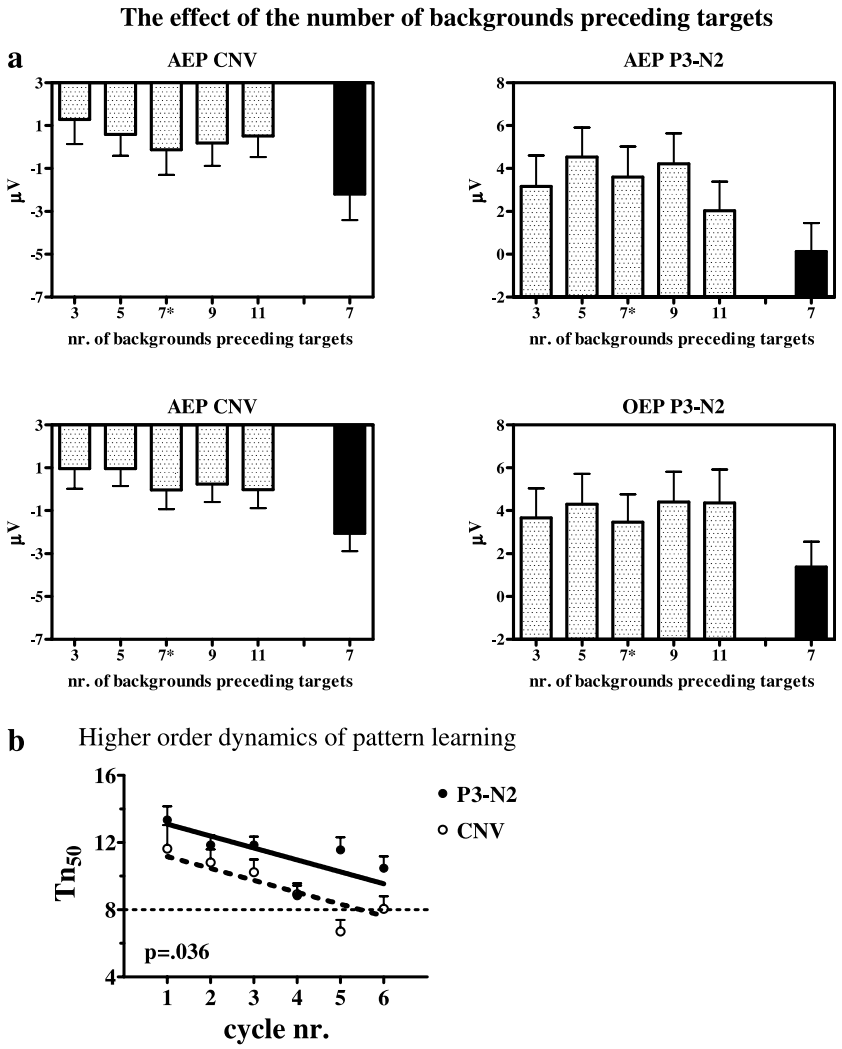

Fig. 6. (a) Shows a bar graph estimating the amplitudes of the ERP CNV (on the left) and the ERP P3–N2 (on the right) for both the AEP session (top) and OEP session (bottom). Component amplitudes to random targets (preceded by 3, 5, 7*(taken as the first target from regular series), 9 or 11 background tones) are depicted in dotted bars, component amplitudes to regular targets (2nd–8th within each regular cycle preceded by seven background tones) are depicted in black bars. (b) Shows the linear regression of the Tn50s for the ERP CNV and P3–N2 as combined from both the AEP and OEP session. The *x*-axes depict cycle nos. (1–6) and the *y*-axes depict values of the Tn50 (as expressed in target position, 1–16) per cycle. As can be seen in the graph, the appearance of the ERP CNV preceded the decay of the ERP P3–N2 for each cycle. An *F*-test comparing the linear regression of the ERP P3–N2 with the linear regression of the CNV gives a significant difference of intercept.

(partly) ascribed to probability effects. Although during the total experiment global probability was kept constant (12.5% for both random and regular targets), local probability varied with respect to only the random targets which could be interspersed with 2–12 background tones. Although not statistically tested, and for explorative purposes only, we estimated probability effects of the CNV and P3–N2 component amplitudes by grouping targets preceded by 3, 5, 7 (though in random series), 9 or 11 background stimuli and compared them to targets preceded by seven background stimuli (from regular series). Fig. 6a depicts these results suggesting that the current findings cannot be ascribed due to (local) probability effects as described by others (Croft et al., 2003; Fitzgerald and Picton, 1981). Thus, when learning a regular sequence, the expectation, or predictability, of regular targets alone results in the increase of the CNV and the decreased of the P3–N2 amplitude (Donchin, 1981; Jentzsch and Sommer, 2001; Jongsma et al., 2005; Polich and Kok, 1995).

Thus, different ERP components may serve as markers for pattern learning. Moreover, these specific components (CNV, P3–N2) mapping pattern learning seem to be modality independent, since they appear in response to both deviant and omitted targets.

4.2. The dynamics of pattern learning

We observed that ERPs in response to randomly presented target stimuli contained a marked P3–N2 complex, possibly reflecting processes related to target evaluation. However, when target presentation became regular, the P3–N2 complex decreased and a marked CNV component

appeared. It is likely, that regular target presentation gives leads to a memory formation of the target-to-target interval. Such a memory formation could give rise to the development of the CNV component occurring ca. 300 ms before target presentation, possibly reflecting processed linked to target anticipation (Nobre, 2001).

Learning curves started at a plateau that remained stable during random target presentations, before falling rapidly following introduction of the regular targets and bottoming out after approx. 4 regularly presented targets. Detecting changes in discrete events requires spanning of the temporal interval between the events. The neural systems involved must create a representation of the event that can be retained for some interval of time (Hughes et al., 2001). For the detection of regularity, at least two consecutive targets with the same number of preceding background tones (7) are needed in order to perceive the regularity. In the current experiment, the time window for temporal integration of the detection of the regularity was thus ca. 13 s. These findings are in line with the instance theory of Logan (2002) – a memory-based theory of learning. Logan (2002) states that the formation of a memory trace for a stimulus, that is predictive for a subsequent stimulus, spans over the whole period collapsing between the stimuli at hand. The process of learning is then accompanied by a shift of attention towards those stimulus features that remain constant.

Interestingly, a previous study measuring ERPs in response to regularly presented targets found that an early (ca. 150 ms after target onset) central anterior distributed component detecting automatic change, the 'Mismatch Negativity' (MMN) disappeared when using short stimulus onset asynchronies (100 ms), but remained intact when targets were presented at regular positions when using longer stimulus onset asynchronies (1300 ms) (Sussman et al., 1998). The authors of this study argued that a slow presentation rate – leading to similar inter-target intervals as in our study (i.e. 6.5 s compared to 6.4 s) – exceeds the limits of the acoustic memory trace underlying this automated MMN. The MMN depends on a short-lived sensory memory, which appears to last about 6 s (Sussman et al., 1998). Therefore, in the current experiment, a longer-lasting auditory memory template must have been formed, spanning at least the full inter-target interval, which probably does not rely on fully automated and involuntary processes.

4.3. Higher-order dynamics of pattern learning

With respect to higher order dynamics of implicit learning, we observed that – within the first presentation cycle – the CNV starts to develop between the 3rd and 4th regularly presented target, and the P3–N2 complex decreases between the 5th and 6th regularly presented target; however, with each repetition of the presentation cycle (cycle length 92 s), both the CNV and P3–N2 effect seems to occur earlier (see also Fig. 6b and Table 3). 'Learning' curves still described the data better than straight, horizontal, lines. Nevertheless, some of these curves returned improbable Tn50s, namely, at points before introduction of the target regularity (e.g., at cycles 4 and 5), probably due to an increase in variability in the single-trial data. The following observations are therefore somewhat speculative.

We observed that the CNV seems to develop prior to the decay of the P3–N2 complex. Thus, after two or three regular targets, a marked CNV develops – apparently expressing target anticipation. Initially, the P3–N2 complex remains intact at the 3rd and 4th regular target presentations. However, when the CNV is fully developed, the P3–N2 complex starts to decrease. It is likely that the CNV early in the regular sequence follows from a 'guess' that some regularity has installed and that it is worth preparing to detect a target. The continuing P3–N2 would reflect evaluation of the situation to confirm the speculation, while later, when the anticipation has been confirmed, the stimulus evaluation decreases. We suggest that the brain seems to be set to finding information in the environment that might lead to target anticipation rather than target evaluation, even without awareness on the part of the participant.

With respect to the delayed response RTs, only significant 'learning' curves were observed for the cycles 3–4 within the auditory session and cycles 3–4 in the omission session. Apparently, delayed response RTs provide a more variable, less robust measure than the ERP components on a single-trial level. Also, in the current experiment, a delayed response task was employed. Participants did not respond directly to the target stimulus, but to the background stimulus following the target. Therefore, effects of target detection would have affected RTs less. This is in Line with Jentzsch and Sommer (2001) who also reported higher variability in RTs than P300 amplitudes. This higher variability might be ascribed to the fact that delayed response RTs are sensitive to more factors – besides the expectancy of the target – for example response strategy and motivational aspects of the participant.

4.4. Conclusions

Our findings imply that dynamic cognitive processes like pattern learning can be studied with the aid of methods such as single-trial ERP measurements. Whereas ERPs have classically been measured within paradigms that avoid systematic trial-to-trial variations (thus allowing averaging procedures), the 'learning-oddball' paradigm described in this study allows us to specifically study systematic trial-to-trial variations. Thus, single-trial ERP research can add to one of the most fascinating and basic subjects of cognitive research, namely learning. Implementing similar single-trial ERP paradigms could also lead to clinically useful tools assessing the ability or speed of pattern learning in different patient groups.

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