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Mechanisms of human attention: event-related potentials and oscillations

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Abstract

Electrophysiological and hemodynamical responses of the brain allow investigation of the neural origins of human attention. We review attention-related brain responses from auditory and visual tasks employing oddball and novelty paradigms. Dipole localization and intracranial recordings as well as functional magnetic resonance imaging reveal multiple areas involved in generating and modulating attentional brain responses. In addition, the influence of brain lesions of circumscribed areas of the human cortex onto attentional mechanisms are reviewed. While it is obvious that damaged brain tissue no longer functions properly, it has also been shown that functions of non-lesioned brain areas are impaired due to loss of modulatory influence of the lesioned area. Both early (P1 and N1) and late (P3) event-related potentials are modulated by excitatory and inhibitory mechanisms. Oscillatory EEG-correlates of attention in the alpha and gamma frequency range also show attentional modulation. © 2001 Elsevier Science Ltd. All rights reserved.

Keywords: Alpha; Attention; Brain; ERP; Gamma; Generator; N1; P1; P3; Oscillations

1. Introduction

1.1. Attention

Attention permits us to focus on a subset of the incoming sensory information, since we are not capable of processing all input. For example, at every moment of time, sensory information about the temperature of our left foot is available for processing by the brain. However, unless we focus our attention to our left foot, we will probably not evaluate this information unless something unexpected happens to our foot. Two different mechanisms of selective attention are responsible for this phenomenon. On the one hand, attentional mechanisms let us focus our attention to our left foot whenever we decide to do so. We will refer to this process as voluntary attention. On the other hand, if something unexpected happens to our left foot, let's say it suddenly gets cold, our brain automatically focusses attention to the sensory information of our left foot. One can then consciously perceive the changed sensory information and respond accordingly. We will refer to this mechanism of attention as automatic attention.

Such changes of attention can occur either within a modality or across modalities. These attentional mechanisms operate in all sensory modalities, but in the remainder of the text we will primarily discuss the auditory and visual domains. For the sake of simplicity we will use the term object for both visual and auditory events.

Attention can be directed either to one out of multiple objects, disregarding its spatial location, or to one out of multiple spatial locations, no matter which object is present at this location. Object attention refers to the process of attending to one object in the presence of other objects which are usually regarded as distractors, since they potentially distract our attention from the attended object. Spatial attention refers to attending to one out of multiple spatial locations, i.e. to attend to the right vs. the left half of the visual field.

Taken together, within the concept of attention, there are at least three differentiations of the involved mechanisms:

- Cross-modality switches vs. within-modality switches
- Automatic selection vs. voluntary selection
- Spatial-selective vs. object-selective

However, object-selective attention may be closely related to figure-ground-segregation. We will limit the scope of this review to the aspects of within-modality switches of attention. A thorough review on cross-modality switches of attention is given in this issue by Eimer.

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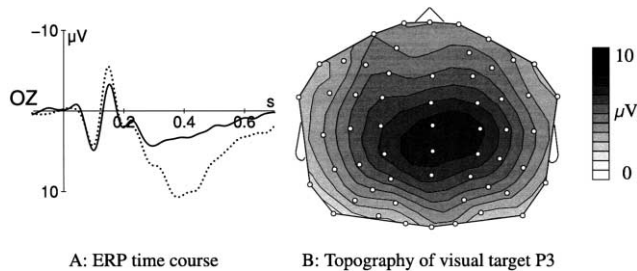


Fig. 1. (A) Typical time course of a visual target ERP (dotted) as compared to a non-target ERP (solid). P1 (ca. 100 ms), N1 (ca. 160 ms) and P3 (ca. 400 ms) deflections are visible. N1 and P3 are significantly larger for the target condition. (B) Topography (top view) of a visual target P3 with maximal amplitudes over central and parietal electrodes.

2. ERP correlates of attention

When the brain processes information attentively, differences in processing can be measured as compared to when one processes the same information unattentively. Electrophysiological correlates of attention are typically analysed using event-related potentials (ERPs). We will also discuss oscillatory brain activity and its relation to attentional mechanisms.

After event-related potentials have been recorded from the human scalp, the localization of the generators of these scalp potentials can be mathematically estimated. For this purpose, a model is assumed with a homogeneous volume conductivity inside the human brain and an inverse algorithm is used to compute possible dipole locations. Another way to localize ERP sources is to record the electric potential from specific locations inside the brain. This is only possible in animal studies or in patients where intracranial electrodes are used to plan brain surgery.

We will limit our discussion to three ERP components associated with the processing of different stages of attention: N1, P1 and P3. The mismatch negativity (MMN) which reflects pre-attentive mechanisms, which occasionally trigger switches of attention eliciting subsequent

P3 deflections in the ERP [1,2], will not be discussed here.

2.1. P300

2.1.1. Functional characteristics

The P3 is a positive deflection in the ERP which peaks around 300 ms after stimulus onset and is also called P300. It is the most prominent ERP component sensitive to cognitive processing [3]. The P3 is elicited when subjects attend to a stimulus and when they discriminate the stimulus features, e.g. to differentiate them from similar stimuli [4]. The P3 has first been found by Sutton and is typically evoked by infrequent, random stimuli (targets) embedded among frequent stimuli (non-targets) in the so-called oddball paradigm [5]. Rare stimuli evoke large P3 amplitudes (10–20 μV). The P3 is influenced by the probability of an event irrespective of the task-relevance [6]. Nevertheless, for stimuli with a 50/50 probability, the P3 amplitude will be higher for go trials (targets) than no-go trials (non-targets) in a go/no-go paradigm [7]. It has been argued that stimulus categorization is more important than stimulus probability for the P3 amplitude [8,9]. The amplitude of the P3 reflects the probability and task relevance of a stimulus while P3 latency reflects the duration of stimulus evaluation [10].

Fig. 1A shows the time course of a target P3 ($p = 0.25$) compared to a non-target P3 ($p = 0.75$) from a visual classification paradigm peaking around 400 ms [11]. In the above taxonomy, this P3 reflects the voluntary object-selective direction of attention within one modality. Fig. 1B shows the scalp topography of a visual target P3 from a similar visual classification paradigm with a centro-parietal maximum [12].

While early components like P1 and N1 in response to an auditory or visual stimulus will be generated in auditory or visual cortices, respectively, the later P3 is generated by multiple distributed generators [13]. The scalp topography of the P3 is largely independent of the input modality while its amplitude is larger and its latency is longer for the visual as compared to the auditory modality [14]. Therefore, it has been suggested that P3 generators depend in part upon the stimulus modality [15].

The term P3b has been coined for the target P3 in order to differentiate it from an earlier positive component in response to novel stimuli, the P3a or novelty P3 [16]. The P3a occurs slightly earlier in the ERP (about 50 ms) and has a more frontal scalp topography than the later P3b [17].

Fig. 2A shows the scalp topography of a P3a in response to auditory novel stimuli with a maximum over fronto-central electrodes from an auditory novelty task [18]. In contrast, Fig. 2B shows the scalp topography of a P3b in response to auditory target stimuli with a maximum over centro-parietal electrodes [19].

Numerous studies have tried to investigate the generators of the P3. Different methods can be applied to gain insights

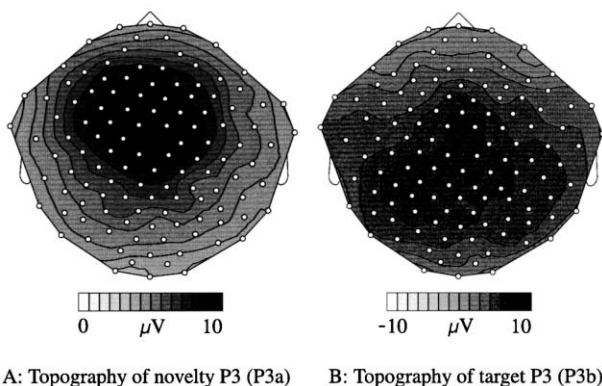


Fig. 2. Topographical distribution of P3a (left) and P3b (right). The earlier P3a has a more frontally distributed topography than the subsequent P3b (adapted from Ref. [19]).

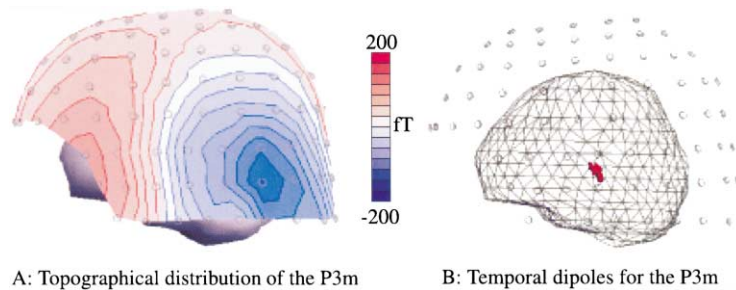


Fig. 3. (A) View onto the left side of a scalp topography of the P3m (brain occluded underneath). The topographical distribution reveals a dipolar pattern over each hemisphere (anterior positivity and posterior negativity). (B) One dipole in each medial temporal gyrus can explain these bipolar fields.

about the neural generators of ERP components including source localization of the scalp potential, intra-cranial recordings, functional magnetic resonance imaging (fMRI) or positron emission tomography (PET) and interpretation of lesion studies.

2.1.2. Source localization

Source localization approaches have claimed multiple brain regions to be involved in generation of the auditory P3b: the thalamus [20], the temporal lobe [20,21], the hippocampus/parahippocampal areas [21], and the insula have been implicated [20]. The same brain regions were also involved in generation of the visual P3b: the hippocampus/parahippocampal areas [22], the insula [23], the temporal lobe [24,25], and the thalamus [26]. An additional activity over occipital cortex was only found for the visual P3b [22].

The auditory P3a is probably also generated, at least in part, in the temporal lobe [27], but, an additional frontal dipole has been found during generation of this ERP correlate of auditory novelty which was not present for auditory targets [28].

Despite the diversity of results, temporal brain structures seems to be found in most studies. The influence of temporal brain areas can be seen in Fig. 3 which shows the topographical distribution of the magnetic equivalent of a target P3, also called P3m, from a visual discrimination task [29]. The dipoles in the two temporal lobes explain about 95% of this MEG pattern. Caution has to be paid to the depth of such temporal dipoles: low-pass filtering and averaging across subjects lead to artificial smoothing of potentials or fields. Dipoles close to the scalp result in steep potential- or field-gradients across the scalp while deeper dipoles generate shallower spatial slopes. Low-pass filtering or averaging may thus lead to deep dipole solutions.

Intracranial recordings found that the limbic system (hippocampus, parahippocampal gyrus and amygdala) generated potentials of up to 200 μ V during an auditory oddball task [30]. In contrast, the mid-brain recordings of Yingling and Hosobuchi were in line with thalamic but not with hippocampal involvement during auditory and visual P3b generation [31].

The medial temporal lobe including the hippocampus

contributes to P3b generation in human and monkeys during auditory as well as visual oddball experiments [32]. Even though deep brain structures probably do not contribute significantly to scalp-recorded potentials of more than 1–3 μ V due to their distance and small size [33], thalamic structures were found to be active during the time interval of the auditory P3b [34]. It may well be that thalamic structures are active during P3 generation and contribute in a modulatory way even if their potentials do not significantly contribute to the scalp potential by volume conduction.

An extensive study investigating multiple brain regions revealed that many cortical areas are involved in P3 generation: Intracranial recordings in epileptic patients during auditory and visual oddball tasks found the posterior cingulate and supramarginal gyrus to be involved in generation of the P3a while the superior parietal lobe was involved in generation of the P3b [35]. That study also found clear differences in the responses depending on the input modality. Moreover, they also found the middle temporal gyrus, the posterior parahippocampal gyrus and the fusiform gyrus to contribute to the P3a while the hippocampus and superior temporal sulcus contributed to P3b generation [36]. Even frontal brain structures were involved in the generation of both P3a and P3b with shorter latencies for novels: orbito-frontal cortex, anterior cingulate cortex and the inferior frontal sulcus show activation during P3 generation [37].

2.1.3. fMRI recordings

A further method to investigate the brain areas which participate in attention has emerged recently. fMRI and PET studies can display the hemodynamic response of brain areas involved in one task but not in another. PET results reported the expanded limbic system (hippocampal formation, parahippocampal gyrus, retrosplenial cortex, thalamus, subcallosal area, the border between areas 32 and 10, anterior and inferior cingulate cortex, putamen, and medial prefrontal cortex) to be activated by novel visual stimuli [38]. The posterior hippocampal formation, parahippocampal gyrus as well as lingual and fusiform gyrus also showed fMRI activation during the encoding of novel visual pictures [39]. Auditory target detection resulted in activation of the temporal–parietal cortex and anterior

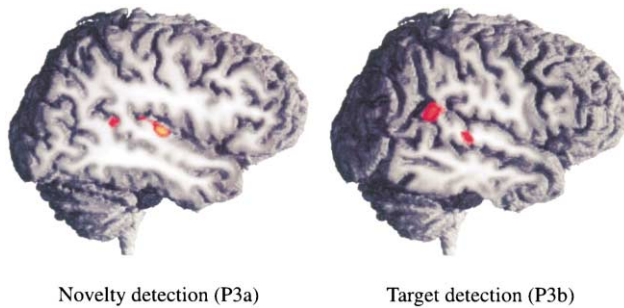


Fig. 4. fMRI results indicate that the temporal generators of the novelty P3a (left) reside about 2 cm more anterior within the superior temporal gyrus than those of the target P3b (adapted from Ref. [43]).

cingulate cortex [40]. By additional dipole modelling, Menon et al. demonstrated that the temporal–parietal cortex is probably the source for the simultaneously recorded P3 [40]. In a visual target detection task it was shown that, besides parietal and cingulate activity, the frontal lobe also was involved in visual target detection [41]. In order to differentiate the influence of stimulus and response modality, Linden et al. compared motor-responses and silent counting in auditory and visual target detection tasks [42]. They replicated the activation of frontal and temporoparietal cortex irrespective of input or response modality which indicated that these regions are engaged in attention rather than sensory coding or response preparation.

By combining electrophysiological and hemodynamic measures, Opitz et al. showed that the target P3b in an auditory oddball paradigm (Fig. 2B) can be modelled by two dipoles in the superior temporal gyri where fMRI activation was found in the same task [19]. These dipoles were active during the time interval of the P3 (200–400 ms) and explained 80–90% of the P3 topography while they

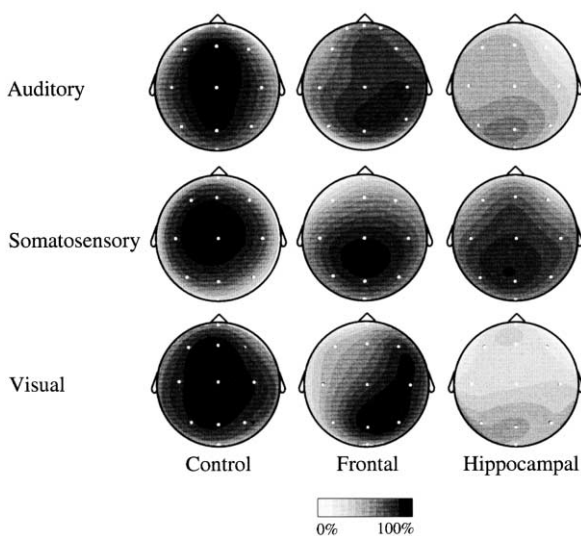


Fig. 5. Circumscribed brain lesions and their influence on P3a topography in an auditory (top), somatosensory (middle), and visual (bottom) task. P3a topographies of patients with frontal and hippocampal lesions are significantly suppressed as compared to control subjects.

explained only 30–40% of the earlier N1 topography. In an auditory novelty task, Opitz et al. [18] also found the superior temporal gyri to be active, indicating that this region is involved in P3a generation as well (Fig. 2B). Comparing the two results revealed that the generators of the novelty P3a are located about 2 cm more anterior within the temporal lobe than the generators of the target P3b [43]. This is illustrated in Fig. 4.

Kirino et al. used a visual oddball paradigm where identical stimuli served either as target or novel stimuli and reported that the frontal cortex was only activated for targets but not for novels [44]. This indicates that the task-relevance of a stimulus is important for involvement of the frontal lobe but the lack of frontal activation by novel events is not supported by other evidence.

2.1.4. Lesion studies

Investigations of patients with circumscribed brain lesions provide important insights into ERP generators. By studying brain lesions and their influence on ERPs, excitatory and inhibitory effects can be dissociated.

2.1.4.1. Excitatory modulation. Johnson studied patients in whom the amygdala as well as the hippocampal and anterior temporal lobe had been resected unilaterally [45]. Since he did not find an asymmetry for P3b topography nor a significant difference in P3b amplitude or latency between patients and healthy subjects, he doubted the importance of these structures for the scalp recorded P3b generation. Similar findings from amnesic patients with bilateral hippocampal damage also question the contribution of the hippocampus to the scalp P3b [46].

Studying patients with unilateral frontal disease, Knight was able to show that lesions of frontal cortex affect the auditory novelty P3a but not the target P3b in a simple detection task [47]. Investigating patients with lesions of the temporal and parietal lobes, Knight et al. showed that lesions of the lateral parietal lobe had no effect on the auditory P3 while lesions of the temporal–parietal junction eliminated auditory P3a and P3b at posterior scalp sites [16]. Lesions of the posterior hippocampal formation had a differential effect upon P3a and P3b: while auditory, visual and somatosensory target P3bs are unaffected, novelty P3as are significantly reduced in all sensory modalities in hippocampal patients [48]. In a comparison of frontal, temporal–parietal and lateral parietal lesions, Knight found that neither of the lesions significantly affected visual P3b amplitude at posterior scalp sites [49]. The P3a and frontal scalp P3b amplitude, however, were significantly affected by frontal as well as posterior lesions. Fig. 5 shows the topographical distribution of the P3a in normal control subjects and patients with lesions of their dorsolateral prefrontal cortex or hippocampus.

2.1.4.2. Inhibitory modulation. The above results demonstrate that dorsolateral prefrontal cortex has an excitatory

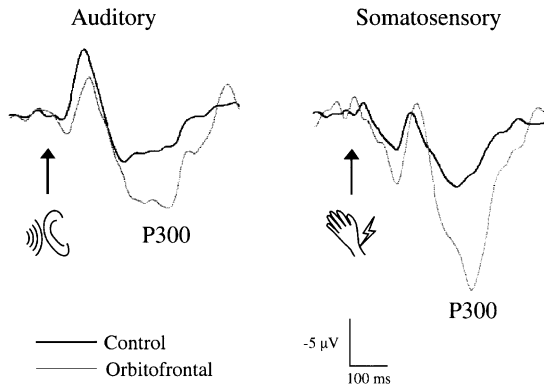


Fig. 6. Orbito–frontal lesions (dotted) result in a disinhibition of P3a at electrode Pz in response to auditory and somatosensory stimuli as compared to normal control subjects (solid) (adapted from Ref. [50]).

influence on P3a generation. Orbito–frontal cortex, however, has been shown to have an inhibitory effect on P3a amplitude. When patients perceive mildly aversive auditory or tactile stimuli (i.e. computer generated bizarre sounds or electric shocks, respectively), their novelty P3a is significantly enhanced as compared to normal control subjects [50]. This is shown in Fig. 6, where the P3a is disinhibited in patients with orbito–frontal lesions.

2.2. P1 and N1

2.2.1. Functional characteristics

It has been assumed that early ERP components like P1 and N1 reflect mainly exogenous processes which are modulated by physical stimulus attributes but not by cognitive processes [51]. Visual P1 and N1 are, for example, both affected by the brightness of a visual stimulus [52].

A fundamental question about attention is whether it operates at an early stage (e.g. before 200 ms) and can influence stimulus encoding or rather at a later stage (e.g. after 200 ms) which has no influence on the encoding [53]. Hillyard et al. demonstrated that attending to tones presented to one of two ears enhances auditory N1 amplitude [54]. This indicated that spatial-selective attention can operate as early as about 100 ms after stimulus presentation. It was shown later that even the visual P1 amplitude reflects spatial-selective attention when attending to one half of the

visual field [55]. But note that the visual P1 occurs later than the auditory P1 (at about 120 ms). Luck et al. differentiated the attentional effects of visual P1 and N1, arguing that P1 might reflect a facilitation of early sensory processing for stimuli presented at an attended location (spatial-selective), whereas N1 might reflect the orienting of attention towards task-relevant stimuli [56]. Similar findings for the visual domain were demonstrated by Hillyard et al. [57].

The above-cited literature assumes that attention modulates the amplitude of an ERP component, such as P1 and N1. An alternative model supposes the existence of an additional processing negativity which is computed by subtracting the ERP of the unattended from that of the attended condition [58,59]. In case of short inter-stimulus-intervals (ISIs) of less than 200 ms, this processing negativity may overlap the N1 [60].

2.2.2. Source localization

Since the early evoked potentials P1 and N1 are generated in primary cortices, one has to differentiate between ERPs for different input modalities. Here, we will review visual and auditory P1 and N1 components.

2.2.2.1. Visual. Fig. 7 shows the topographical distribution of the visual P1 and N1 of the ERP in Fig. 1. Comparing dipole localizations and positron emission tomography (PET) revealed that the generators of the visual P1 probably lie within the fusiform gyrus [61]. The N1 is probably generated by distributed dipoles in lateral extrastriate cortex [62]. It was suggested that dorsal occipito–parietal as well as ventral occipito–temporal areas are also involved in N1 generation [63]. Clark et al. argued that the frontal positivity during the N1 time interval is not merely a projection of posterior generators, but results from frontal generators [64].

2.2.2.2. Auditory. Since, in the auditory domain, the N1 has been the earliest component to reliably be sensitive to manipulations of attention, we will focus on this component. Dipole source analysis of the N1 revealed activity in the primary auditory cortex, i.e. Heschl's gyrus on the supra-temporal plane [65]. Localization of the magnetic counterpart, the N1m, also found the auditory cortices to be responsible for N1m generation [66]. The N1 component

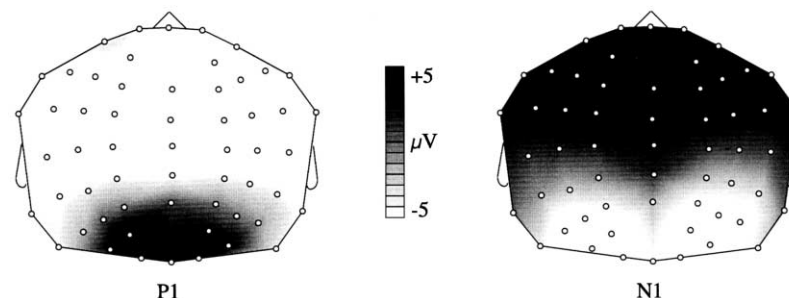


Fig. 7. Topographic distributions of visual P1 and N1 indicate generators in fusiform gyrus (left) and extrastriate occipital cortex (right), respectively.

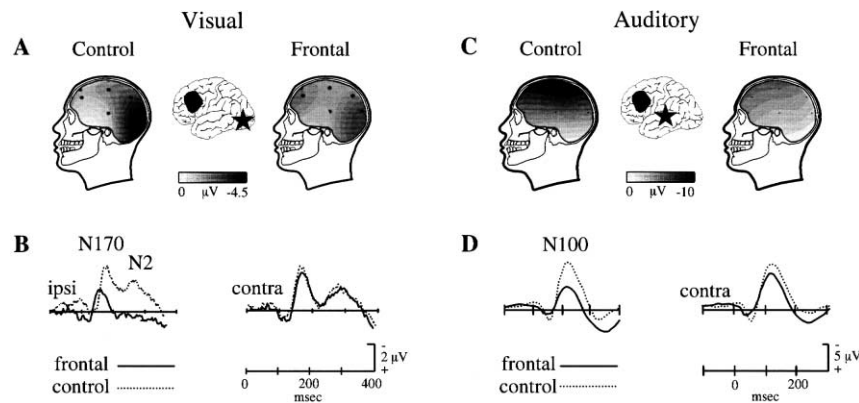


Fig. 8. Frontal brain lesion and their influence on the topography of the visual N170 (A). The lesioned area is indicated in black and a star denotes the approximate generator of the N170. Ipsilateral to a frontal brain lesion, N170 and N2 in response to targets are significantly suppressed while contralateral there are no significant changes (B). The topography of the auditory N100 is also affected by prefrontal brain lesions (C, lesion and generator indicated as above). Prefrontal lesions significantly suppress the auditory N100 (D) (adapted from Refs. [71,73]).

shows a tendency to lateralize to the hemisphere contralateral to the ear of stimulation [67]. Some authors even differentiate a number of different auditory N1-like ERP components between 50 and 150 ms [68,67].

2.2.3. Lesion studies

2.2.3.1. Excitatory modulation. Knight et al. showed that patients with lesions of temporal–parietal cortex have reduced auditory N1 amplitudes while lesions of frontal cortex had no significant effect on the N1 amplitude at midline electrodes [69]. In a subsequent study, these authors conducted an experiment employing selective attention and were able to show that frontal brain lesions affected the attentional modulation of the auditory N1 while the unattended amplitude remained unaffected [70]. However, frontal cortex also provides an excitatory intrahemispheric input to auditory N1 generators [71]. Frontal cortex also modulates the amplitude of the occipitally generated visual P1: the standard-P1 ipsilateral to a unilateral frontal lesion is suppressed in amplitude while the contralateral P1 amplitude does not differ significantly from control subjects [72]. For responses to target-stimuli, also visual N1, N2 and P3 are suppressed in amplitude ipsilateral to the frontal lesion.

Fig. 8 shows how a frontal brain lesion affects the occipitally generated visual N1 and N2 in response to target stimuli: ipsilateral to the frontal lesion, N1 and N2 are suppressed in amplitude while the contralateral ones are not [73]. Similarly, lateral frontal cortex provides a facilitatory input into ipsilateral auditory association cortex [71].

2.2.3.2. Inhibitory modulation. It is worth mentioning that prefrontal cortex can have also inhibitory influences on ERP generation. Dorsolateral prefrontal cortex was shown to have an inhibitory effect on middle-latency auditory evoked potentials. The Pa component (25–35 ms) in response to randomly presented auditory clicks was enhanced in

patients with unilateral lesions of dorsolateral prefrontal cortex as compared to normal subjects [74]. Responses to somatosensory stimuli were also enhanced in patients with dorsolateral prefrontal lesions: P26, N28, P45 and N67 all showed significant increases for patients as compared to healthy controls [75].

3. Oscillatory brain activity reflecting attention

When considering oscillatory activity, it is important to differentiate between activity which is independent of stimulation (spontaneous), tightly correlated in phase with the time of stimulus onset (evoked), or elicited by a stimulus but not tightly phase-locked (induced) [76]. A simulation showing evoked and induced oscillatory activity is shown in Fig. 9. When multiple epochs are averaged, evoked activity is clearly visible in the average while induced activity cancels out. Special analysis methods are necessary to compute the induced activity [77].

3.1. Alpha activity (8–12 Hz)

When recording EEG from human subjects at rest, a 10 Hz oscillatory activity is the most prominent feature of the ongoing signal [78].

When primary visual cortex receives no or little input, it in turn oscillates predominantly in the alpha frequency range at relatively high amplitude which is considered to reflect cortical idling [79]. The high-amplitude alpha oscillations at the scalp are due to a high degree of synchronicity of the underlying generators. The synchronized alpha activity desynchronizes in response to stimulation, leading to a decrease of the total alpha power. At the same time as alpha power decreases, the randomly distributed phase of the alpha activity during baseline is reset, leading to an increase of phase-locked (evoked) alpha activity [80]. This simultaneous increase of phase-locked alpha and decrease of alpha power after a stimulus was described as the alpha

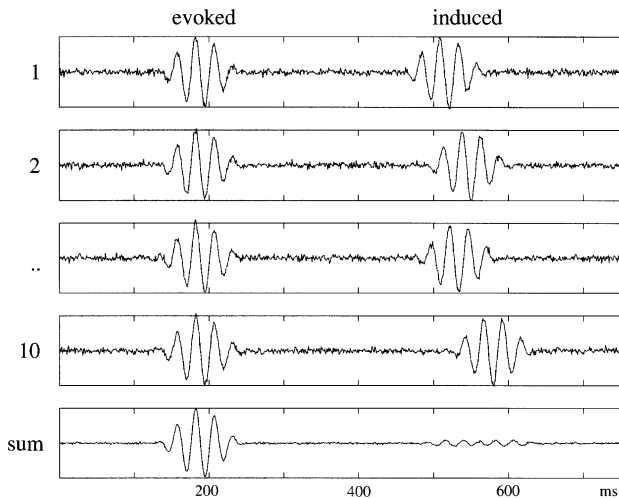


Fig. 9. Oscillatory EEG activity has to be differentiated into evoked (left) and induced (right) activity according to its phase locking to a stimulus. The simulation shows four of ten averaged trials and the resulting sum (bottom). While evoked activity adds up, induced activity cancels out almost completely.

paradox [81]. Differentiating between evoked and induced alpha activity reveals this phenomenon.

3.1.1. Functional characteristics

Changes of induced alpha power after stimulation have been associated with attention, since targets lead to a decrease of induced alpha activity in a visual oddball paradigm [82]. An auditory oddball experiment revealed that event-related desynchronization (the decrease of induced alpha activity after stimulation) is coupled with the amplitude and latency of the auditory target P3b, again indicating a correlation with attentional processes [83]. Comparing a passive listening condition with an auditory oddball paradigm showed an increase of evoked alpha for the targets which was interpreted as a correlate of attention [84]. Shifts of evoked alpha activity from temporal to occipital regions also reflect attentional shifts from the auditory to the visual modality [85,86].

3.1.2. Source localization

Even though cortical neurons generate the scalp-recorded alpha EEG, they are probably driven by thalamic neurons [87]. Oscillatory sleep spindles can be recorded both from thalamus and cortex, but after disconnecting the two, only the thalamus remains oscillating [88]. Even though sleep spindles and waking alpha activity are not the same, the neural generators are probably identical [89]. In a model, thalamo-cortical feedback loops through the nucleus reticularis have been proposed to serve as the driving source for cortical alpha generators [90].

A combination of electric and magnetic recordings of the human alpha EEG indicated the generators of the posterior alpha to be in the calcarine fissure [91]. Fitting dipoles into the alpha activity of 22 normal subjects resulted in sources

near the midline in the basal parts of the occipital lobe [92]. A combination of MEG recordings and magnetic resonance imaging located dipoles in the calcarine fissure, the parieto-occipital sulcus and the surrounding occipital and parieto-occipital areas [93]. These findings suggest that alpha activity is generated in cortex but can be modulated or driven by thalamic structures.

3.2. Gamma activity (30–80 Hz)

A phenomenon similar to the alpha paradox was also observed for gamma activity in an auditory oddball paradigm: while evoked gamma activity increased after stimulation, induced activity was reduced [94]. This indicates that the phase of the gamma activity (with respect to baseline) rather than the amplitude is modulated by stimulation. Therefore, the differentiation into evoked and induced activity is necessary [95].

When a stimulus is presented to a subject, the high-amplitude alpha oscillations are replaced by oscillations of higher frequency but lower amplitude. Induced activity around 40 Hz (gamma frequencies) has been especially associated with cognitive processing [77].

3.2.1. Functional characteristics

According to the temporal correlation hypothesis, the simultaneous firing of neurons indicates that they code features of the same object [96,97]. Electrophysiological studies on animals show strong evidence that synchronized brain activity in the gamma frequency range could be the correlate of feature binding [98]. Human experiments found induced gamma activity to correlate with this binding mechanism [99]. Induced gamma activity has not only been correlated with binding, but there is also evidence that the same type of activity correlates with attention [100–103]. Induced gamma activity was found to accompany the P3 in cats suggesting that these oscillations are related to attentional processing [104]. Also, single cell recording in monkeys report enhanced 40 Hz responses when the animals attended to a stimulus among distractors [105].

When somatosensory targets are detected among distractors, dorsolateral prefrontal and parietal EEG electrodes show a coherence in the gamma band restricted to the hemisphere which perceives the stimuli [106]. A study using somatosensory targets also identified prefrontal cortex as the source of target gamma activity [107]. In auditory oddball tasks, unattended deviants elicited larger induced gamma activity during the time interval of MMN than standard stimuli [108,109]. During the time interval of the P3, attended deviants (targets) elicited less induced gamma activity than the standard stimuli.

In addition to this increase in induced gamma activity, the phase-locking of gamma activity also seems to play a role in attention. Auditory targets resulted in a higher degree of phase-locking than non-targets [110,111]. Herrmann et al.

showed that an early evoked gamma EEG response in a visual discrimination task was larger for target stimuli than for non-target stimuli [11]. Another experiment with the identical stimuli but a different instruction for the subjects (different target stimulus) revealed that the early evoked gamma activity changed with task requirements: just like the P3, it was larger for targets than non-targets, indicating that the 40 Hz response reflects mechanisms of attention [112]. In an MEG experiment, the topography of this early evoked gamma activity was located over prefrontal brain areas [29].

3.2.2. Source localization

Neocortical excitatory–inhibitory circuits have been thought to underlie the visual gamma oscillations [113]. Data which indicated the synchronization of striate and extrastriate cortices and interhemispheric synchronizations of left and right area 17 lead to the interpretation that gamma oscillations are generated in cortex [114,115]. But, magnetic field tomography revealed cortical and subcortical 40 Hz oscillations to be coherent, suggesting also a thalamic source for auditory gamma activity [116]. Electrical stimulation of different thalamic nuclei showed that gamma oscillations in auditory cortex can be inhibited by the medial geniculate nucleus and evoked by the posterior intralaminar nucleus [117]. Therefore, a thalamo–cortical network seems most plausible for the generation of gamma oscillations [118,119].

Intracranial recordings during an auditory discrimination task revealed temporal areas to generate an increase of induced gamma activity after stimulation which was much more focused than the simultaneous decrease of induced alpha in the same areas [120].

4. Conclusions

Even though some experiments revealed contradictions concerning the origins of ERP components, we will try to summarize those brain areas which resulted in converging evidence for their involvement in attentional processes.

4.1. Sensory cortices

Concerning the question whether attention can operate already at early stages of sensory processing [53], ERP results clearly demonstrate that early attentional modulation is possible, as indicated by amplitude modulations of P1 and N1 components. Also hemodynamic responses show that attention reliably modulates neural activity in primary and secondary cortices [121,122]. However, initial visual input to striate cortex is not modulated by attention suggesting that the hemodynamic changes in primary sensory cortex (i.e. V1) may be due to a re-entrant signal [123,124]. In support of this notion, single unit activity in monkey V4 showed the first robust modulation of attention

100–300 ms after stimulation while it occurred later in V2 and still later in V1 [125].

During early stages of stimulus encoding, processing takes place in primary sensory cortices and is specific to the input modality. Here, spatial-selective attention modulates early ERP components like the visual P1, leading to larger amplitudes if a stimulus appears at an attended location as compared to an unattended location [56].

Directing attention towards certain features of objects rather than their spatial location leads to later effects, starting at about 150 ms [57]. Selecting target-relevant stimuli only starts after about 300 ms and is reflected in N2 and P3 amplitudes which are generated in distributed neural systems [57].

4.2. Posterior association cortices

During later processing stages, as indicated by P3 potentials, processing is not limited to sensory cortices: the scalp-recorded P3, the main electrophysiological correlate of late attention processes, is mainly generated by neocortical generators: the temporal–parietal junction (superior posterior temporal plane and superior temporal sulcus) have been shown to be critical for P3b generation by lesion studies [16,17]. While the anterior part of the temporal lobe is involved in P3a generation, the posterior part of the temporal lobe participates in P3b generation [43].

4.3. Subcortical structures/hippocampal formation

The hippocampal formation also plays an important role in P3 generation. It was found to be active during P3b generation in a variety of tasks [21,22,30]. However, the scalp P3b peaks 30–50 ms before intracranial field potentials [126] and lesions of the hippocampal formation fail to significantly reduce P3 amplitude [48]. This indicates that the scalp-recorded P3 is not due to volume conduction of hippocampal activity. Instead, the hippocampal formation probably has a modulatory influence on P3 generation.

Also the thalamus plays an important role for mechanisms underlying P3 generation. Thalamic sources have been found to be active during P3 generation with MEG and MRI [20,26]. However, thalamic structures probably do not contribute significantly to scalp-recorded potentials via volume conduction due to their distance, small size and closed field geometry [33].

While also oscillatory EEG activity reflecting attentional mechanisms is generated cortically, it is probably driven by neurons in the thalamus [87,117–119].

4.4. Prefrontal cortices

Even though early sensory coding is performed in sensory cortices, prefrontal cortex can have both excitatory and inhibitory influence on the neural generators of early ERP components [127]. This indicates that not only sensory cortices are involved in these early processing stages, but

they are supported by frontal areas. Structural equation modelling of fMRI responses revealed prefrontal cortex to modulate activity in the visual cortex [128] and the top-down modulation of frontal cortex during visuo-spatial attention operates even without visual stimuli [129].

In addition, subregions of lateral and orbital prefrontal cortex can have excitatory as well as inhibitory influence on later P3 generation [49,50].

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