

1.5 The alpha band

1.5.1 Introduction

The alpha frequency band runs from 8 to 12 Hz. Oscillations in this band can have amplitudes large enough to be clearly seen in raw EEG traces, and were observed in the first EEG recordings. Interpretations of the alpha band initially centred around the basic idea that alpha-band oscillations reflect an absence of mental activity, or neural information processing. More recent findings and studies of thalamocortical interactions have suggested that alpha-band oscillations reflect specific kinds of inhibitory processes. Note that activity in this frequency band thus has a very different behavior in relation to experimental manipulations and hypothetical neural function than the theta-band activity discussed in 1.4.

1.5.2 Idling

Alpha band oscillations were for a long time taken to reflect an absence of mental activity [152] or, in later terms, a state of neural tissue in which it processes no information [187]. The idea of the presence of brain activity reflecting the absence of mental activity goes back to very early EEG studies. As described by Millett [152], the EEG was developed by Hans Berger (1873 - 1941) in the first decades of the twentieth century. Berger developed an interest in the physical form of mental events, that was triggered by the experience of a near-fatal accident. During a military exercise, he was thrown from his horse in front of a horse-drawn artillery gun. Berger escaped injury, but he later received a telegram from his father, sent at the urging of his sister who had felt that something terrible had happened to him. Such telepathic occurrences were not obviously in contradiction with the dominant scientific idea of that time: the conservation of energy [152]. Lehmann [129] had applied the conservation of energy idea to mental events by postulating the existence of psychic energy ("P-energy") as just another form of energy. Chemical energy, released by metabolism in the brain, could be converted into heat, electricity and P-energy, of which the P-energy was necessary for mental states. Thus, a subtraction logic could be devised: the difference between chemical energy and the sum of heat and electrical energy would be the amount of P-energy available for perception, feeling, emotions and conscious thought. Grounded in such ideas, the essential problem of Berger's psychophysiology was how to study the transformations of energy sources in the brain into P-energy. Berger's work on attempting to measure the non-P-energy of brain function seems to have to be understood from an additive energy model: as more total energy becomes available to the brain, more can be converted to P-energy; and given a certain amount of cerebral energy, a decrease of heat or electricity must be associated with an increase in P-energy, to preserve the conservation of energy. Berger performed both thermometric and electrical studies, his breakthrough occurring in 1924. In that year, Berger was stimulating the cortex of a patient named Zedel, when he switched the electrodes from a stimulator to a galvanometer used for electrocardiograms. The galvanometer recorded electrical oscillations from Zedel's cortex. In 1927, after some years of technical improvements, Berger was able to consistently produce EEG tracings measured from the scalp. This was before the approach of calculating ERP's, before digital storage and manipulation of signals were available at all. Nevertheless, two characteristics of the brain's electrical activity were visible: the 10 Hz, large-amplitude alpha and the 20 Hz, smaller beta waveforms, the latter of which were thought to reflect metabolic processes. Bursts of the alpha waveform appeared during mental rest, and disappeared - as a substrate for P-energy? - during mental activity and sensory stimulation.

Berger's framework for interpretation seems to suffer specifically from coming before information theory, lacking access to an idea that something non-physical (although still not specifically mental, or conscious) - information - can exist both in parallel with and systematically related to physical processes. Still, his interpretation of alpha-band activity as a negative indicator of mental activity remained dominant for a long period, in a somewhat different form. Neuronal spikes have become generally conceived of as events involving both physical processes and information processing, entwined together [230]. However, the negative relation between specifically alpha band oscillations and mental activity remained, only this became interpreted to mean that "idling" neuronal areas, i.e. areas that are not currently processing information, fire synchronously in the alpha band frequency range [187]. Berger's findings were taken as evidence for the idling hypothesis, as well as new data. One of the basic findings was that of 7 - 14 Hz sleep spindles, caused by thalamocortical interactions [222] discussed below. Alpha band effects were also found in awake subjects, extending early findings.

The time course of alpha band activity has been shown to be related to experimental conditions and behavioral events; such effects have further been shown to be, in some cases at least, localized. In two cued-task experiments, the pre- and post-stimulus alpha-band power were compared and the time course of power in the upper (10 - 12 Hz) and lower (8 - 10 Hz) alpha band was studied [113]. In both experiments a warning tone was followed by a one, two or three second pre-stimulus interval. In the first experiment, the task was to silently read a word or a number. For these data, conclusions were based on the computation of event-related desynchronization (ERD) instead of the observation of alpha waveforms in the raw EEG. An ERD time course can be seen as an ERP based on decreases in instantaneous amplitude (in practice, the directly associated measure of power is used, but for consistency the term amplitude will be used throughout to describe amplitude-related parameters) relative to a baseline, positive values being given by reductions in amplitude [187]. Negative ERD values would reflect event-related synchronization (ERS). ERD values can be computed by band-pass filtering a signal, rectifying it, and averaging epochs time locked to repeated events of the same kind. In these data, ERD was greater post- than pre-stimulus, and increased post-stimulus with longer pre-stimulus intervals. The lower alpha band showed a roughly one second period of ERD following warning signals, which then dropped until stimulus presentation. An exception to this appears to occur at frontal sites, where the ERD persisted. The upper alpha band ERD did not respond to warning signals, and decreased pre-stimulus, increasing at posterior sites post-stimulus. The warning signals in this experiment were taken to increase alertness, or non-specific attention, but not expectancy, or the selection of specific processing pathways. The second experiment used conditions with blocked or randomized trial types to distinguish such general and specific forms or preparation. The task was to judge words as animals versus tools, and numbers as even versus odd. In blocked conditions subjects could prepare for the specific task, while in random conditions whether a word or number judgement was upcoming was unknown. For the blocked relative to the random condition, the upper alpha band showed a lower ERD, both pre- and post-stimulus, and the greatest increase in ERD at stimulus presentation. The latter result was interpreted as a reflection of a more relaxed state in the blocked condition. The upper alpha band showed greater differences over scalp locations (measured as a posterior - anterior difference) and the average ERD was greater in the lower alpha band. The results were interpreted to suggest that ERD in the upper alpha band reflects specific computational processes, due to its more specific scalp distribution and response to stimulus presentation, while ERD in the lower alpha band, showing a more generalized and stronger ERD, reflects overall alertness. These interpretations

were used in a later study on memory performance and ERD (described below), but do not seem to rest on strong evidence. For instance, could some kind of general but phasic alertness not peak post-stimulus, invalidating the assumption that activity arising post-stimulus must be assigned to specific processing? Nevertheless, these systematic effects on alpha band ERD provided a basis for further research. Whether the presented stimulus is (recognized as) an object or not has also been shown to influence alpha-band amplitude [234]. Using the MEG, the amplitude of an alpha rhythm originating from the parietal-occipital sulcus was measured following presentation of line drawing of objects, and of non-objects created by randomly rotating areas of the drawings. Subjects were instructed to indicate whether they perceived a coherent and meaningful object or not. Alpha desynchronization was greater following presentation of an object than a non-object. Missed objects showed an intermediate level of alpha activity. These differences were found in the period 300 - 1000 ms post-stimulus. When subjects did not have to respond, differences in alpha activity between objects and non-objects were lost. The results were interpreted in terms of disengaging the parieto-occipital sulcus from low level visual cortices, in which case it was described as resting as opposed to selecting a target for further processing.

A more explicitly functional role than a reflection of idling was assigned to alpha-band activity in a study that compared tasks requiring the observation of external stimuli (sensory intake tasks, e.g. searching for Mooney faces, pictures of faces which are reduced to black and white areas, making them hard to recognize) or internally directed attention (sensory rejection tasks, e.g. mental rotation of a geometric figure). In both conditions, tasks were such that cortex was not expected to be idling, regardless of the intake - rejection distinction. Performing tasks that required sensory information to be processed decreased right-hemisphere parieto-occipital alpha-band activity, relative to tasks involving only internal representations [194]. Alpha band activity is also related to memory performance [115]. A "difference based on later memory performance", or Dm, effect, was found for high- and low-performing subjects in an incidental memory task. Subjects first performed a task in which they made semantic judgements on words, and were then confronted with an unexpected memory task in which they had to recall as many of the words presented in the judgement task. Finally, subjects were presented with a cued-recall task in which the six categories from which the presented words were taken were provided (e.g. birds, vehicles, weapons). Upper and lower alpha band ERD was measured during semantic judgement trials, and compared between trials for which the presented word would be remembered or forgotten at memory testing. Trials consisted of a warning signal, followed after a delay of around one second by a word, followed after 1500 ms by a signal indicating that the response could be given. The subjects were divided into a good- and a bad-memory group based on a median split of the free-recall scores. Alpha-band ERD was greater for bad performers, most strongly at parieto-occipital sites. A post-stimulus difference between frontal-central upper and lower alpha ERD occurred, that was described as stronger ERD in the lower alpha band. However, inspection of the figures shows that it is ERS in the upper alpha band, relative to baseline, that causes the difference, which may be relevant in terms of later results and hypotheses on the alpha band described below. Good and bad performers both showed a memory-dependent effect, but in different frequency bands. Good performers showed a peak in lower alpha ERD 200 - 600 ms post-stimulus for remembered words, while bad performers showed a more gradual increase in upper alpha ERD for remembered than non-remembered words. Based on the earlier study concerned with lower and upper alpha activity, lower and upper alpha activity was interpreted to reflect general alertness and specific processing (semantic encoding), respectively. Good performers were assumed to more easily encode the stimuli while being more alert

during stimulus presentation, and given these interpretations and assumptions, the ERD could be explained. As argued above, the grounds for this interpretation did not seem to be strong, and no specific mechanisms underlying the various effects or an explanation for the strong ERS relative to the baseline in the upper alpha band was provided.

Motor activity evokes alpha-band event-related desynchronization over cortical areas associated with the moved limb [188] (in the context of motor activity, the alpha frequency range is termed μ). Foot, hand and tongue movements result in ERD topologies that could be related to the underlying sensorimotor homunculi [185] [186]. For instance, hand movements are associated with μ ERD at electrodes C3 and C4, while Cz responds to foot movements. A related phenomenon called focal ERD / surround ERS has been demonstrated, which refers to ERS occurring at areas around a desynchronized region [186]. In a study comparing self-paced ballistic movements, sustained contractions, and the release of a contraction of the wrist, μ -band activity was shown to decrease at central and parietal sites from around a second prior to movement to a minimum around half a second to one second after movement onset [1]. Around contraction releases, the reduction in alpha amplitude was weaker than around ballistic movements and sustained contractions. Motor imagery has been shown to result in similar patterns of desynchronization as actual movement, both showing lateralization depending on real or imagined movement of the left versus right hand [147]. Desynchronization was, however, weaker for imagined movements. Anticipatory hand-switching results in increased alpha-band coherence [208]. Subjects either performed 30 s of flexion - extension movement with their right hand, or switched to left-hand movements after 15 s following a verbal cue. Subjects knew whether a switch would occur. In the first 15 s, subjects showed higher μ -band coherence in switch blocks, bilaterally but mostly within the hemispheres (importantly for later discussion, no effects in the beta band were found). No differences in power were found. Results using directed transfer functions showed higher directed coherence from frontal to more posterior areas (motor and parietal) in the right hemisphere (i.e. the hemisphere of the hand to be switched to), and these results were interpreted to reflect a frontal-to-central flow of information. The left-hemisphere directed transfer results were not reported. The results were taken to reflect a communicative function of between-signal alpha-band coherence, as opposed to the idling "null-function" of within-signal alpha band activity.

Results on alpha-band activity during the presentation of probe stimuli do not seem to be fully explainable by the idling hypothesis. A Sternberg study in which lower and upper alpha-band ERD was measured showed differences in alpha band responses to memory set presentation and probe stimuli [121]. Auditory stimuli consisting of four vowels were presented, after which a target vowel was presented. Subjects had to determine whether the target was in the probe set. During the memory set presentation, mostly from 1 - 3 sec into the presentation, the lower and upper alpha bands showed synchronization, the lower and upper bands showing predominantly occipito-parietal and broad distributions, respectively. Following the probe, ERD occurred in both bands. In the lower band, the ERD had an occipito-parietal distribution and was significant from 500 to 3000 ms after the probe. The upper band ERD had a parietal, central and occipital distribution and was significant at two time points, around 1 s and 2 s following the probe. However, the ERS during probe presentation is difficult to explain in terms of idling - are neural areas doing less while probes are being perceived and memorized than during a baseline resting period? A similar finding of ERS during a pre-stimulus period of activity was found in a delayed response task [18]. Subjects were shown a target location which either remained visible or disappeared during a delay, of one or four seconds, preceding a required response (touching the target location). During the four second

delay, both lower and upper alpha band amplitude over posterior electrodes rose above baseline. Again, an interpretation purely in terms of idling does not seem able to explain these results. Why should areas be expected to systematically process less than during baseline? As described below, alpha-band activity appears to reflect not so much the reduction of cortical information processing, but what makes such a reduction possible in the face of constant afferent input.

1.5.3 Inhibition

More recent EEG studies [58] [61] [245] have found further results that do not fit with an interpretation of alpha band activity as an index of idling. These studies suggest that it reflects active inhibition. In an intermodal attention study [58], subjects were cued to direct their attention to either the visual or the auditory modality. Alpha-band effects were found in the one second delay between the cue (the word BEEP or FLASH) and the compound visual - auditory stimulus. Stimulus targets were defined by a mismatch between two component stimuli in the cued modality. Mismatches concerned the tone frequency of binaurally presented tones or the position of bilaterally presented circles. Starting around 500 ms post-cue up to stimulus presentation, alpha-band amplitude began to diverge for the two cue types over parieto-occipital sites. Following cues indicating an auditory task, alpha-band amplitude increased in this interval, while it decreased when the visual task was cued. Similar effects were found using an auditory instead of a visual cue [61]. Auditory cues were single or double clicks, succeeded after 1200 ms by, randomly, a unimodal or compound audio-visual stimulus. Subjects had to detect targets in the cued modality. Again, around 500 ms post-cue, parieto-occipital alpha-band amplitude diverged, increasing when the auditory task was cued and decreasing when the visual task was cued. Spatial-cueing effects on alpha-band amplitude have been found within the visual modality using a go - no go task [245]. Cues indicated whether stimuli would appear, following a 1000 ms delay, in the left or right hemisphere. Stimuli consisted of rotated T's in one of four rotations or dots moving in one of four directions, one of each stimulus type being the target for a block. Stimuli were presented in either the upper or lower visual field in different blocks. Following the cue, amplitude dropped to a minimum after around 400 ms, and subsequently increased to a maximum before and around stimulus presentation. Pre-stimulus, the electrodes over occipital cortex ipsilateral to the cued side showed a greater increase in amplitude than those over contralateral cortex. Amplitude increases were highest over parietal-occipital areas. The topology of amplitude increases was also dependent on whether stimuli would be presented in the upper or lower visual field, with more medial - dorsal distribution for the upper visual field. The focality of the amplitude increases and the attention-dependent shifts in location suggested that the alpha-band activity reflects active inhibitory process as opposed to a passive idling state.

Using experimental manipulations designed to have subjects direct their attention to external stimuli or internally, further arguments were given for the hypothesis that alpha-band activity reflects inhibition of task-relevant areas, as opposed to idling or intake-rejection [40]. Subjects were given either easy or more demanding questions to answer about upcoming sequences of sensory stimuli (e.g., no question, the number of stimuli presented, the number of different stimuli presented). Visual, tactile and auditory stimuli were used. After the blocks in which these sequences were presented (called the externally directed attention condition), subjects performed the same tasks but now with imagined stimuli (called the internally directed attention condition). Alpha-band activity was lower during sequence presentations than during sequence imagining, while following more demanding questions alpha band activity increased, either significantly or as a trend for all modalities. No interaction between the task demand and internal - external factors was found. The

findings were taken to argue against the idling and intake-rejection hypotheses, due to the either reversed or indifferent predictions, respectively, concerning effect of task demands. The inhibition hypothesis was argued to provide a parsimonious explanation, as it would predict the inhibition of task-irrelevant areas regardless of whether such inhibition is necessary due to the protection of internal processing or higher task demands. Referring back to the difficulties in explaining results in terms of the idling hypothesis described above, inhibition of task-irrelevant neural areas, or processes, may provide a better, although unspecific, kind of explanation. As probes are presented or a response-goal must be both delayed and maintained, the inhibition of potentially interfering effects may be necessary. It may, as suggested by the results above, be the case that whether such interference comes from external stimuli or memory processes is irrelevant. It does seem possible that the effects of stimulus presentation will require the greatest inhibition. Ecologically, it would make sense to remain sensitive to at least some classes of potentially dangerous or advantageous events in the outside world. This somewhat gray relation between sensory events and internal processes, as well as the link between inhibition of the effects of such events and processes and alpha band activity, can be mapped quite directly onto the consequences of thalamocortical connectivity.

1.5.4 Alpha rhythms, augmenting responses and the thalamus

Cortical alpha-band activity is strongly linked to thalamocortical interactions [162]. All input to the neocortex from sense organs, as well as low level brain areas such as the cerebellum and mamillary bodies, is relayed via the dorsal thalamus (hereafter referred to as the thalamus). The thalamus sends excitatory output to all regions of the cortex (layers IV and VI), as well as to the striatum and amygdaloid complex, from so-called relay cells, and receives cortical feedback from layers V and VI. Input is also received from the brain stem and the thalamic reticular nucleus. Interactions between the inhibitory cells in the reticular nucleus and the thalamic cells that relay peripheral information are the basis of thalamocortical alpha rhythms, due to a recurrent inhibition pattern (see paragraph 1.1.4) discussed in more detail below. The thalamus consists of nuclei of cells that relay a specific type of information - e.g. visual, thermal, auditory etc, resulting in maps of sensory space (e.g. the position on the retina) on the thalamus. The nuclei contain two kinds of relay cells, first- and higher-order, which receive input from the characteristic peripheral input type and from the cortex, respectively. The input to first-order relays is termed driving afferent input, and is communicated via the thalamus in parallel, that is, without direct interactions, with the information being transmitted in other nuclei. Higher-order relays provide a means of interaction between cortical areas, as one cortical area may project to another's relaying thalamic nucleus. The effect of such corticothalamic connections is modulatory: it cannot change what driving afferents will be relayed by a certain nucleus, but can change how they are relayed.

The modulation of the behavior of relay cells concerns their firing mode: tonic firing, synchronous bursting and asynchronous bursting [162]. Given a constant excitatory input, relay cells with a membrane potential that is more depolarized than around -65 mV respond with the production of single spikes, at a low frequency (around 10 Hz, but these single spikes are not the source of the alpha rhythm measurable in the EEG). More hyperpolarized relay cells temporarily open so-called transient-type calcium channels; this results in a low threshold Ca^{2+} conductance. When these channels are open, excitatory potentials are increased, as the channels do not close immediately as the opening hyperpolarization is lost (the response lag of these channels to membrane voltage lies around 100 ms). This mechanism underlies the post-inhibitor rebound described below, and results in a transient high-frequency burst of spikes. Such bursting can occur synchronously

over multiple nuclei, under which circumstances peripheral information is not being relayed (as nucleic output then communicates the event of synchrony as opposed to events that are transmitted by driving afferents). Asynchronous bursting also occurs, however, and in this case bursting has been argued to still convey information about driving afferents, but different information than transmitted by single spikes. The difference is based on linearity relations between stimulus intensity (or afferent input) and the response in spikes per second. In tonic mode, spikes increase somewhat linearly with increased intensity, while in burst mode, showing more all or none than gradual responses, the intensity - response relation is flatter. In other words, the response is more sensitive to the intensity in tonic firing mode. If excitatory input causes a burst, the event of an increase in intensity in the relayed, e.g. sensory, variable may be more strongly communicated than by the singles spikes in tonic firing mode. This would be at the cost of more subtle coding of intensity. The burst mode of firing is associated with thalamic alpha rhythms, especially synchronous rhythms which would result in alpha-band waves of cortical excitation at scales that would be measurable in the EEG. Burst firing and thalamic alpha rhythms are related via the augmenting response described below.

When thalamic and cortical neurons are stimulated in the alpha frequency range, their response increases, in terms of depolarization and spiking, from around the first to the fifth stimulus [223] [21]. This augmenting response involves a secondary depolarization that appears at the second stimulation, with a relatively late onset in the order of 10 ms. The augmenting response is thus a form of short-term plasticity that depends on an increased tendency to depolarize at a certain point in time following a previous neuronal event. Thalamic stimulation leads to stronger augmenting responses in the cortex than stimulation of the cortex directly, due to spike bursts generated within the thalamus. A mechanism for generating augmenting responses is the post-inhibitory rebound [21]. Such rebounds occur after (recurrent) inhibition which leads to hyperpolarization, which leads to the deinactivation of low-threshold Ca^{2+} currents, which leads to a greater response to subsequent excitatory inputs as described above. So, if the inhibition is time locked to the arrival of the next stimulation, e.g. due to recurrence between thalamic relay and reticular cells, augmentation can occur. Computational modelling has been done on augmenting responses in thalamocortical networks [21]. Both the thalamus and cortex layers in the model contained excitatory (thalamocortical cells in the thalamus and excitatory cortical cells) and inhibitory neurons (thalamic reticular cells and inhibitory interneurons). Only AMPA and GABA receptors were included in the model, as related experimental data had been acquired under an NMDA-blocking anesthesia. The simplest thalamocortical model contained one of each of the four neuron types listed above, with recurrent inhibition within the thalamus and the cortex, and with the excitatory cells projecting onto all other cells. Thalamic stimulation that arrived after recurrent inhibition from reticular cells resulted in an augmented burst of spikes in thalamocortical neurons. This burst subsequently evoked EPSP's in the cortical layer. More complex models showed that further interactions could occur between thalamic augmenting responses and cortical activity. One result, that may be especially important to understanding cognitive control, was that cortico-thalamocortical feedback could induce augmenting responses in other cortical areas than the area initially targetted by the stimulated thalamus cells.

Connections within the thalamic reticular nucleus may be essential for the ability to direct attention. In a computational thalamocortical model built up from modules which contained the basic relay - reticular, excitatory - inhibitory connection, inhibitory intrareticular interactions were shown to be able to reproduce the focal ERD / surround ERS finding [225]. In this model, a

cholinergic neuromodulatory input (which is the type provided by the brain stem) was included that excited relay and inhibited reticular cells. When this input was low, cells achieved sufficient hyperpolarization for rebound spikes and thus bursting alpha activity to occur. When the modulating input was applied to only one of two mutually inhibitory reticular nucleus cells in a two-module model, the modulated module lost its alpha activity while the other module showed an increase in alpha activity. The mean hyperpolarized state associated with alpha activity was suggested to result in the withholding of sensory information. In this model this state was taken to underlie a resting state, but is also compatible with an interpretation in terms of internal versus external attention.

So, the thalamus generates alpha-band activity that, just as in the EEG results described above, is not restricted to any one simple function. Sensory rejection seems likely to be one function, but corticocortical interactions via the thalamus can also occur, with unknown but potentially important computational results. Further and perhaps relatedly, it does not seem that thalamic alpha-band activity necessarily reflects the lack of information transmission. It may serve as a "wake-up call" [211], transmitting a strong interruption to ongoing cortical processing. This may be part of corticocortical interactions, for instance if the result of some cortical computation is the overruling of whatever another area is doing.

1.5.5 The alpha band and task switching

The EEG results and the anatomical and physiological properties of thalamocortical interactions suggest hypothetical roles of alpha-band activity in task switching. Recall that backwards inhibition of task sets occurs, as shown by a difficulty in returning to a task previously switched away from [142]. This inhibition was described as an automatic processes, but the initial activation of such a process (i.e. setting up the triggering conditions) could nevertheless be the result of cognitive control aimed at achieving a target task set. The use of tasks involving different sensory modalities provides an opportunity to study the role of inhibition in a task switching context. If two tasks differ on their stimulus sets, then switching between them may be achieved by inhibiting the undesired set of stimuli, and thereby effectively the stimulus - response mapping [200]. For instance, let task A map a square and a circle to the right and left index finger, respectively. Then let task B map a high and a low tone to the right and left index finger, respectively, and let all stimuli be compound: a visual and auditory stimulus are presented together, so that only the internal state of the subject can determine which of the two tasks will be performed. In this case, when subjects switch from the visual to the auditory task, the relay of visual information might be inhibited, which would, as described above, be associated with an increase in cortical alpha-band activity prior to and during stimulus presentation. Such behavior would basically replicate the general findings on anticipatory occipito-parietal inhibition: subjects are simply cued to perform their next task, which involves certain inhibition demands. Preparing for hold trials would not be expected to be essentially different, perhaps showing somewhat smaller amplitudes because of the decay of previous-task interference to be inhibited. Consideration of backwards inhibition would seem to lead to the expectation of high parieto-occipital alpha amplitude when switching to the visual task, relative to when repeating the visual task.

In a similar way, alpha-band activity may turn out to reflect other limitations of preparation when switching between and repeating task sets. One way to view switch costs is as a failure to inhibit interference from the previously active task set. If subjects use inhibition to control their behavior, inhibition of the visual modality should be stronger preceding auditory than visual trials.

However, if this control is limited preceding switch trials, leading to switch costs, anticipatory, inhibitory alpha-band activity might not be able to occur prior to auditory switch trials. It would then be expected to be strongest while preparing to repeat an auditory trial.

Finally, cortico-thalamocortical connections open the possibility that changes in thalamocortical activity may not be involved directly with the relay of sensory information but with changes in cortical processing. If such changes plays a role in preparation in a task switching task, they would be expected to occur prior to stimulus presentation, as opposed to being part of an adjusted state determining how driving stimulus information is transmitted, which would have to persist at least up to stimulus presentation.

Similarly to shifts between input modalities, certain task combinations may allow switching to be achieved by shifts in output modalities. By using response sets which contain only movements all made by either the left or the right hand, measures of response-hand related lateralization of, e.g., mu-band ERD could be used to follow preparatory processes involved in switching between and repeating tasks. This kind of amplitude lateralization would also be expected for the beta band, and will be discussed further in section 1.6.

Finally, the anatomical distinction between driving first-order afferents and modulating, corticothalamic higher-order afferents to the thalamus may be part of the reason why switch costs are confined to the first trial following a switch, that is, why they disappear after a single trial performed using a new task set. One of the two clear differences between preparing for an upcoming trial and actually responding to a stimulus is that only in the latter case is there driving peripheral input from an external stimulus. The second difference is the analagous occurrence of an actual motor response after stimulus presentation. This difference will be discussed in the following paragraph in relation to beta-band activity.