



Modelling of auditory evoked potentials of human sleep–wake states

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ARTICLE INFO

Article history:

Received 5 February 2011
Received in revised form 19 July 2011
Accepted 24 October 2011
Available online 28 November 2011

Keywords:

Auditory evoked potentials
Sleep–wake states
Vigilance
Modelling
Positive and negative waves
Neuronal correlates

ABSTRACT

The shape of evoked potentials is influenced by the level of vigilance, varying with sleep–wake states. In this paper the shape of auditory evoked potentials is modelled by taking two factors, both modulating the underlying neuronal substrate, into account: ‘sensory gating’ and ‘neuronal firing mode’. Under low levels of vigilance sensory gating reduces the amount of neuronal activity reaching the cortical centres. Due to a rise in hyperpolarisations of thalamocortical neurons associated with an increasing depth of sleep, stimulus evoked primary and secondary excitations, seen as correlates of the N1 and N2 waves of the evoked potential, become smaller. Heightened hyperpolarisations also change the spontaneous activity of neurons from the ‘tonic’ firing mode of wakefulness into the ‘burst–pause’ firing mode of sleep. The large P220 complex together with the N350 and N550 waves in sleep are caused by the stimulus induced triggering of pauses and bursting of neurons. The results of this modelling experiment confirm the view that sleep-specific components such as P220, N350 and N550 are waves that facilitate and protect sleep, whereas the wake-specific components N1, P2–P3 and N2 have perceptual–cognitive functions. In particular the wake P2–P3 wave is sensitive to cognitive functions, such as attention. Based on the modelling results it is suggested that component negativities, expressed in N1, N2 and N350, reflect excitatory processes, whereas positivity in P2–P3 and P220 is a correlate of inhibitory processes. Hence, the large P3 in an attended condition is also interpreted as an inhibitory process suppressing irrelevant information, facilitation the saliency of relevant information.

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1. Introduction

Evoked potentials in the electroencephalogram (EEG) form a main research tool to study processes of vigilance during sleeping and waking, as well as the amount of information processing during these states. Shape and amplitude of these evoked potentials are considerably modulated by vigilance processes, indicating major variations in the processing of external information during sleep–wake states. Since evoked potentials are independent of behavioural responses and conscious awareness, they form an adequate tool to establish the extent of information processing during all states of vigilance. In this framework evoked potentials are frequently studied during sleep and wake states. Recent reviews of vigilance modulation of human evoked potentials are presented by Bastien et al. (2002), Campbell and Colrain (2002), Colrain and Campbell (2007) and Yang and Wu (2007). N1 is mainly controlled by the physical features of the stimulus, as well as by the general state of the subject's brain. N1 is largest under alert wake conditions and decreases at lower levels of vigilance (Nordby et al., 1996; Campbell and Colrain, 2002; Campbell, 2010). N1 is regarded as indicative of stimulus registration and detection, presumably underlying the perception of the stimulus

(Näätänen and Picton, 1987; Campbell and Colrain, 2002; Campbell, 2010). The P2 component (or P220) on the other hand shows an amplitude increase during sleep (Nordby et al., 1996). The amplitude is even larger in deep sleep than in light slow wave sleep. The P220 wave is a prominent sleep component of the evoked potential (Bastien et al., 2002), but its significance and function is still not fully clear (Crowley and Colrain, 2004). After this positive sleep wave a negative wave follows, in the time domain of 300 to 400 ms. and mostly indicated as N350. When subjects further descend into sleep later negativity start to increase getting its maximal amplitude between 500 and 750 ms. This negativity is also known as N550 or the ‘late negative wave’. The long latency of this component seems to indicate that processing of external sensory stimuli is delayed (Nordby et al., 1996). The shape of the evoked potentials constructed during REM sleep is mostly fairly similar to those of waking, although the amplitude of N1 is smaller compared to its waking level (Muller-Gass and Campbell, 2010).

In this paper it is tried to identify the factors responsible for the vigilance related changes in auditory evoked potentials. A main question is how the underlying neuronal substrates, producing and shaping the event-related potentials, change their characteristic activities depending on the diverging levels of vigilance. The shape of these evoked potentials is modelled starting with the assumed influence of the underlying neuronal substrates by processes of vigilance. Two factors are taken into account. Firstly, ‘sensory gating’ (Coenen and Vendrik, 1972; Steriade et al., 1993; Coenen, 1995; McCormick and

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Bal, 1997), involving the reduction of sensory information on the way from the senses to the perceptual cortical areas during descending levels of vigilance and, secondly, the 'neuronal firing mode' (McCormick and Bal, 1997; Weyand et al., 2001), meaning a gradual change in firing pattern of cortical neurons from the tonic firing mode into the burst–pause firing mode, when the state of the brain changes from wakefulness to sleep.

2. Methods

Twelve healthy, adult male and female subjects, with a mean age of 22 years were prepared for polysomnographic recordings. Ag–AgCl electrodes were placed on Fz, Pz, Cz, C4 and C3 and linked to the left mastoid, according to the 10–20 system. EOG and EMG electrodes were also placed. Subjects went to bed around 11.30 PM, without any specific instructions except 'sleep well'. Tones of 1000 Hz with a duration of 50 ms and a loudness of 40 dB were presented with intervals between 2 and 7 s through loudspeakers at approximately 1 meter distance placed right behind the subjects. Tones were presented during two entire, successive nights, starting when the subjects went to bed. The EEG signal was filtered between 0.1 and 40 Hz and recorded at a sampling rate of 1000 Hz. All electrophysiological recordings were stored and sleep–wake states were off-line scored by classic polysomnographic techniques. Auditory evoked potentials belonging to the several sleep–wake states, such as wakefulness, stage 1 to stage 4 of slow wave sleep, as well as REM sleep, were constructed off-line by averaging 50 single tone trials. Trials including eye movements exceeding 150 μ V in amplitude were removed. The resulting auditory evoked potentials were grand averaged over subjects. In the modelling study only the event-related potentials data recorded from Cz were taken into account.

3. Results

The grand averages of the auditory evoked potentials of the various sleep–wake states recorded from Cz are shown in the most right panel of Fig. 1. In the evoked potential of wakefulness are clearly visible: N1 (a negative wave between 50 and 200 ms), P2 (a positive wave around 125 and 275 ms) and N2 (a negative wave peaking between 200 and 350 ms) (Nordby et al., 1996; Colrain and Campbell, 2007; Folstein and Van Petten, 2008; Yang and Wu, 2007). The N1 wave is relatively

large during wakefulness and becomes smaller during stage 1 of sleep, while this component is not longer visible in other sleep stages. Just as N1, N2 is also decreasing during sleep, while a N350 component is appearing, ultimately growing and changing into N550. This large N550 wave is a typical sleep component with a maximal size in sleep stage 4. The P2 wave seems to grow and to shift in time with declining levels of vigilance, with a maximal size in stage 4 of sleep. It is then called P220. All evoked potentials as well as the change in the shapes of the potentials produced by the lowering of vigilance in sleep are in full agreement with the general waveforms described in the literature, perhaps with the exception of the shape of the potential of REM sleep. This shows a relatively flat pattern without characteristic components shape and also in the literature this REM sleep shape can vary considerably.

3.1. Modelling factor 1: Sensory gating

A stimulus evokes a train of action potentials in the sensory pathways. However, animal studies have clearly shown that on the way to the higher perceptual centres the peripheral activity is modulated on a thalamic level according to the level of vigilance (Coenen and Vendrik, 1972; Steriade et al., 1993; Edeline et al., 2000). This activity, the primary excitation, which ultimately arrives at the sensory cortex, declines with descending levels of vigilance. This is due to the process of 'sensory' or 'thalamic gating' (Coenen, 1995; McCormick and Bal, 1997). It is likely that the decrement in activity gives rise to a decrease in the amplitude of N1 during lower levels of vigilance. N1 is assumed to be associated with the primary excitation of cortical cells. This also fits with the assumption of Näätänen and Picton (1987) and Winter et al. (1995) that the locus of origin of N1 is in the cortex. Besides a primary excitation, a stimulus elicits also a secondary excitation. This secondary excitation, which is smaller than the primary excitation, is assumed to be expressed in N2. Just as N1, N2 declines during sleep. The primary and secondary excitations are separated by an inhibitory phase, which contributes to the formation of P2 (Fig. 2) (Coenen, 1995; Karmos et al., 1988).

In the leftmost panel of Fig. 1 the evoked potential components obtained during the several sleep–wake states are modelled according to the 'sensory gating' factor. The procedure for this part of the modelling process is based on single unit data expressed in post-stimulus time histograms' (PSTH) of sensory neurons responding to sensory stimuli. The data are obtained from animal work derived from Coenen and Vendrik (1972) and Karmos et al. (1988). The primary excitation in the PSTH (the direct response to the stimulus) is the underlying substrate for N1, while the inhibition following the primary excitation is the substrate for P2. After P2 a more tonic, secondary excitation, which is lower in activity of the primary excitation, follows, which is the substrate for N2. In waking, the primary excitation is maximal, thus also the amplitude of N1. The size of the modelled N1 is taken from the actual size of the waking evoked potential. The amplitude of the secondary excitation, expressed in N2, is also derived from single unit data, and set here on about two-third of the size of the N1. During lowering of vigilance both the amplitudes of N1, P2 and N2 are decreasing. The amplitudes of both the primary and secondary excitations decrease in an almost linear fashion with descending levels of vigilance from wakefulness to sleep stage 4. As estimated by Coenen (1995) about one-third of the original wakefulness-activity-amount is reaching the cortical centres during deep slow wave sleep. That fact is taken into account in the modelling, expressed in the sleep-amplitude of N1, taken as one-third of the waking amplitude. The valley between N1 and N2 is presumably due to an inhibitory phase directly following the primary excitation (Fig. 2) (Coenen and Vendrik, 1972; Karmos et al., 1988). As the amount of inhibition is directly dependent of the primary excitation, the P2 wave also declines with descending levels of vigilance.

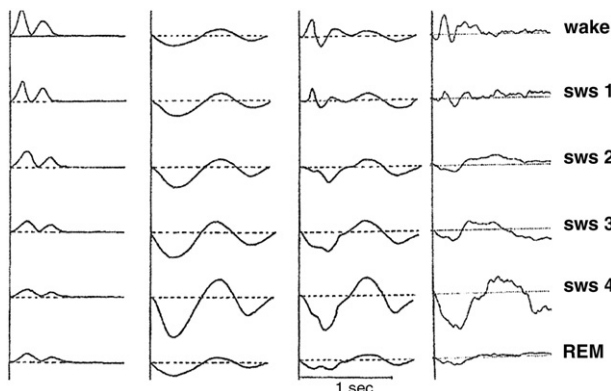


Fig. 1. In the most-right panel the grand averages of auditory evoked potentials of sleep–wake states (wake, SWS 1–SWS 4, REM) derived from position Cz are shown (voltage on the Y-axis is given in arbitrary units [negativity upward] and the time scale is indicated). In the most-left panel modelling according to the factor 'sensory gating' is presented, while in the middle-left panel modelling according to the factor 'neuronal firing mode' is indicated. Both modelling factors are described and discussed in the text. In the middle-right panel the results of the modelling with these two factors are combined, simply by addition. Note the remarkable resemblance between the constructed evoked potentials in this middle-right panel with the original recordings in the most-right panel.

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