Journal of Neuroscience Methods 178 (2009) 237–247

Contents lists available at ScienceDirect

Journal of Neuroscience Methods

journal homepage: www.elsevier.com/locate/jneumeth

Biocybernetics of attention in the tinnitus decompensation: An integrative multiscale modeling approach

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ARTICLE INFO

Article history:
Received 13 August 2008
Received in revised form 4 December 2008
Accepted 6 December 2008

Keywords:
Auditory evoked cortical potentials
Multiscale modeling
Auditory scene analysis
Auditory selective attention
Tinnitus decompensation

ABSTRACT

Tinnitus is one of the most common symptoms affecting people all over the world. In the absence of an established cure many individuals are not only faced with the need to adjust to the sensation of the tinnitus noise, but also with psychological comorbidities. In recent years, different studies have been directed to elucidate the psychophysiological mechanisms that are involved in the tinnitus decompensation. From these, special emphasis has been placed on studies related to attention and habituation, which accordingly play a crucial role in current tinnitus therapy approaches. In spite of such progress, the relationship between selective attention and the tinnitus decompensation with respect to large-scale neural correlates is still not well understood. In order to address this issue, we propose an integrative multiscale modeling approach for studying neural correlates of auditory selective attention in the tinnitus decompensation. Computational simulations based on our model confirmed electroencephalographic human data of both auditory selective attention and the tinnitus decompensation. It is concluded that the proposed methodology represents a promising approach to give insight into the neurodynamics of auditory selective attention in the tinnitus decompensation.

1. Introduction

The study of the tinnitus decompensation has acquired great relevance due to the increasing number of cases reported worldwide as well as the challenge it presents to medical researchers. In general consensus tinnitus is characterized by the perception of a phantom sound or noise in the ears or in the head without the presence of an external source. With respect to pathogenesis, tinnitus sufferers are classified as compensated and decompensated tinnitus patients. In the case of the earlier, the tinnitus noise is annoying but still these patients are able to habituate to such abnormality. In the case of the later, the tinnitus noise is troublesome and in some cases can lead to psychiatric symptoms such as insomnia and depression that can be a contributing factor to suicide (Delb et al., 1999; Turner et al., 2007).

Several conceptual models have been proposed to gain deeper understanding into the neurophysiological mechanisms of the tinnitus decompensation, among the more influential are (a) Jastreboff (1990), Jastreboff and Jastreboff (2006), (b) Hallam et al. (1988), and (c) Zenner et al. (2006). In particular, Jastreboff's seminal work suggests that habituation is prevented due to a patient's emotional response to the perceived threat represented by the tinnitus noise, in addition that the highly emotional connotation of the deteriorating tinnitus could lead to a higher degree of attention directed towards the tinnitus noise. These implications have also been the basis of the well-known tinnitus retraining therapy (TRT), in which a combination of sound therapy and counseling are used to achieve habituation toward the tinnitus. More specifically, patients are no longer aware of their tinnitus, except when they focus their attention on it, and even then tinnitus is not annoying or bothersome (Jastreboff and Jastreboff, 2006).

Zenner's model (Zenner et al., 2006) is based on the first, but rather advocates for a replacement of the acquisition of a conditioning reflex by a sensitization mechanism, which on the basis of brain plasticity suggests the involvement of cognitive mechanisms. In this respect, recent studies emphasize the role of enduring attention as a leading factor in the increase of the tinnitus perception, possibly sustained by human emotions (Zenner et al., 2006; Low et al., 2006; Husain, 2007). In such respect, neurobiological evidence of psychologically driven top-down interactions has been provided by examinations in mammals and humans (Ji and Suga, 2003; Penfield and Perot, 1963). In particular some studies

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doi:10.1016/j.jneumeth.2008.12.012

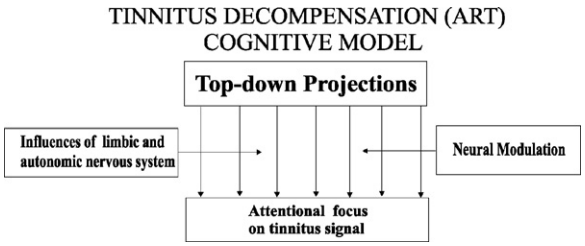

Fig. 1. Scheme of the Jastreboff's tinnitus model within the context of ART.

(Mahlke and Wallhäusser-Franke, 2004; Wallhäusser-Franke et al., 2003) have shown that the perception of a troublesome sound, e.g., a salicylate-induced tinnitus in the mongolian gerbil, leads to a significant alteration of cortical and subcortical plasticity due to an activation of the limbic system. More recently, other authors reported that attentional mechanisms in tinnitus patients are hampered in correlation with the patient's level of distress (Schlee et al., 2007). The latest statement is in agreement with the Jastreboff's tinnitus model (Jastreboff, 1990) in which decompensated tinnitus patients show not only an increased fixation of attention, but also a decreased habituation towards the tinnitus noise (Jastreboff, 1990).

One interpretation of the mechanism of attention in relation to the tinnitus decompensation, can be provided by the Grossberg's adaptive resonance theory (ART) (Grossberg, 2005). In this framework, incoming sensory stimuli activate stored top-down expectations originating from learning background processes, such expectations are thus compared with bottom-up sensory signals so that a match between them results in an attentional shift to the incoming signal. The mapping of this framework to the case of the tinnitus decompensation suggests that the activity in response to stimuli other than the tinnitus noise is relatively suppressed or desynchronized, in other words that the attentional focus is on the tinnitus signal (Fig. 1). Neural correlates of such top-down processing could thus be reflected in auditory evoked cortical potentials (AECPs).

In this paper we propose a model of neural correlates of auditory selective attention in the tinnitus decompensation. Such framework lies under the light of our recent findings related to an objective quantification of the tinnitus decompensation by electroencephalographic correlates (Strauss et al., 2008). The organization of the paper is as follows: Section 2.1 provides a brief overview of neural correlates of selective attention and the tinnitus decompensation. Section 2.2 introduces the proposed auditory selective attention model. Section 2.3 discusses about the subjects and experimental paradigms. Section 3 introduces our numerical results. Section 4 presents the discussion. Finally, Section 5 presents our conclusions.

2. Materials and methods

2.1. EEG correlates of selective attention and tinnitus decompensation

Selective attention refers to the brain's outstanding ability to select specific information from a vast amount received at any time (Mackintosh, 1965). Numerous studies have addressed the effects of selective attention at the level of electroencephalograms (EEG) and late evoked responses (ERPs). In what follows we provide a brief overview of such findings, although for the purpose of our model we focus on the selective attention effects on the prominent auditory evoked cortical potential (AECP) components N1 (latency at about 80–100 ms) and the P2 (latency at about 160–200 ms).

2.1.1. Oscillatory EEG activity

It is widely accepted, that attentional processes can be monitored by the observation of EEG alpha- and gamma-band (Doesburg et al., 2008). Phase stability is stated to correlate highly with the power of a given EEG frequency band, thus a drop in the power of the bands spectral peak is considered a desynchronization of the underlying generators. In 2007 Birbaumer et al. (2008) could demonstrate an enhanced synchrony in gamma-band (30–50 Hz) and theta-band (4–7 Hz) phase, representing top-down and bottom-up activity, respectively, when a target stimulus is attended. Similar findings were made by several authors including the study by Fries et al. (2001) who monitored an increase in gamma band activity in macaque monkey visual cortex (V4), while attending a behavioral relevant task. But a diminished synchrony in low-frequency bands ($<$17 Hz) monitored during this task is inconsistent with an increase of theta-band phase synchrony as demonstrated in Birbaumer et al. (2008). Similar mechanisms may be assumed for auditory processing.

According to several authors, focal attention generates a central area featuring a higher synchrony in a given EEG frequency band, surrounded by areas in which the phase synchrony in the same frequency band is lowered. Suffczynski et al. (2001) demonstrated this event-related desynchronization in the alpha band in focal cortical areas accompanied by a synchronization of the alpha band in neighboring areas following a stimulus event.

2.1.2. Auditory evoked cortical potentials (AECPs), the N1 and P2 components

In the auditory modality, Hillyard et al. (1973) showed differential processing of attended and unattended auditory stimuli at the level of the N1 component. Subsequent studies have since furthered this finding by showing specific processing characteristics of the N1 component, e.g., amplitude enhancement in relation to selective attention and increasing stimulus intensity (Knight et al., 1981; Hillyard, 1981; Mangun, 1995; Ritter et al., 1998), and inter-stimulus interval (ISI) (Hari et al., 1982). Analogously, some studies have emphasized the amplitude enhancement of the P2 component in regard of auditory selective attention and stimulus characteristics (Hillyard et al., 1973; Garcia-Larrea et al., 1992; Näätänen, 1990). Within the auditory modality, the P2 component often occurs together with N1 (referred to as the N1–P2 complex) and shares many of characteristics, yet the two components can be dissociated experimentally and developmentally (Oades et al., 1997). The maximum amplitude of the P2 component can span a broader latency range (150–275 ms) when compared to the N1 component (Dunn et al., 1998) and can be double-peaked (Hyde, 1997; Ponton et al., 1996). The N1 component has been suggested to be generated by sources in primary auditory cortex in the temporal lobe, while the generators of the P2 component are thought to be located mainly in the primary and secondary auditory cortices (Zouridakis et al., 1998). With regard to attention, some authors (Woldorff et al., 1993; Woldorff, 1999) have suggested that the electrophysiological attention effects, that originate in the primary auditory cortex, take the form of an amplitude modulation of the early sensory evoked components and are co-localized with the sources of these sensory evoked components. This in turn provides support for the existence of an attention-modulated sensory gain control of auditory input channels at or before the initial stages of cortical processing. In support of this hypothesis, some studies have shown evidence of the existence of an intracranial homologue to the scalp N1 attentional effect (Neelon et al., 2006).

2.1.3. Tinnitus decompensation correlates

At the level of the N1 and P2 components, we recently identified large-scale neural correlates of the tinnitus decompensation by means of analysis of phase clustering between sweeps (Dobie and