The P50 evoked potential component and mismatch detection in normal volunteers: implications for the study of sensory gating

Nashaat N. Boutros*, Michael W. Torello, Brandy A. Barker, Patricia A. Tucting, Shu-Chieh Wu, Henry A. Nasrallah

*Department of Psychiatry, West Haven VA Medical Center (116A), 950 Campbell Avenue, West Haven, CT 06516, USA
Department of Psychiatry, Ohio State University, 473 West 12th Avenue, Columbus, OH 43210, USA

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Abstract

Sensory gating is a complex, multistage, multifaceted physiological function believed to be protecting higher cortical centers from being flooded with incoming irrelevant sensory stimuli. Failure of such mechanisms is hypothesized as one of the mechanisms underlying the development of psychotic states. Attenuation of the amplitude of the P50 evoked potential component with stimulus repetition is widely used to study sensory gating. In the current study, we investigated the responsiveness of the P50 component to changes in the physical characteristics of ongoing trains of auditory stimuli. Forty normal volunteers were studied in a modified oddball paradigm. At all cerebral locations studied, P50 amplitudes were higher in response to infrequent stimuli. We postulate that the increase in P50 amplitude reflects the system's recognition of novel stimuli or "gating in" of sensory input. The ratio of the amplitude of the responses to the infrequent stimuli to those of the frequent stimuli was significantly higher for the posterior temporal regions. This finding provides further evidence that the temporal lobes may be significantly involved in sensory gating processes. Although this study only included normal subjects, the data generated contribute to the understanding of sensory gating mechanisms that may be relevant to psychotic states.

Keywords: Electrophysiology; Auditory stimuli; Temporal lobes; Sensory inhibition

1. Introduction

The study of sensory gating processes is of major interest to behavioral neuroscientists because a failure of these processes has been implicated in the pathophysiology of schizophrenia and other psychotic conditions (Freedman et al., 1983; Baker et al., 1987; Boutros et al., 1991). Sensory gating is traditionally defined in terms of inhibitory processes. This definition is reflected in the three paradigms commonly used to study sensory gating. Erwin et al. (1991) used trains of auditory stimuli at varying interstimulus intervals to study the effects of stimulus repetition on the amplitudes of mid-latency auditory evoked potentials (MLAEPs). The MLAEPs are a series...
of brain waves that are recorded at the scalp following auditory stimulation and usually occur between 10 and 250 ms poststimulation. The MLAEPs are known to decrease in amplitude with repetition of identical stimuli at short interstimulus intervals (Fruhstorfer et al., 1986). Sensory gating has also been studied with a paired click paradigm. In the paired click condition, the first of a pair of stimuli ($S_1$) is followed by an identical stimulus ($S_2$) 500 ms later. The inhibitory capability of the brain is measured as the ratio of the amplitude of the P50 component of the MLAEPs to the $S_2$ stimulus to the amplitude of the $S_1$ response ($S_2/S_1$). The lower ratios are presumed to reflect better inhibition or more intact sensory gating (Freedman et al., 1983). Lastly, a paradigm examining the inhibitory effect of low amplitude warning signals on the magnitude of the auditory startle response (prepulse inhibition or PPI) has also been used to study sensory gating (Braff and Geyer, 1990). In a recent report, the PPI measure correlated well with the amplitude of the P50, but not with the $S_2/S_1$ ratio in normal volunteers (Schwarzkopf et al., 1993). This finding suggests that the different paradigms may be measuring related, but not identical aspects of sensory gating mechanisms.

Processing of sensory input seems to require at least two stages: a stimulus-identification stage (a stimulus is present) followed by a stimulus-evaluation stage. Evidence from the literature suggests that while schizophrenic patients can identify the stimuli in their environment, they may have difficulty in evaluating stimulus input (Freedman et al., 1991). In addition, poor performance by schizophrenic patients on the continuous performance test points to a deficit in the immediate discrimination of letters from numbers over extended periods of time (Nuechterlain and Dawson, 1984). A diminished amplitude of the P300 component of the AEP may also indicate a decreased capacity to distinguish between two tones with certainty on a task requiring maintained attention (Pfefferbaum et al., 1984). It is possible that such difficulty in continuously discriminating between stimuli in a vigilance task could be a reflection of sensory gating difficulty.

Physiological theories developed to explain a decrease in amplitude of the P50 component of the EP with repetition are diverse and problematic. Direct evidence in humans for an active sensory gating function is lacking. The active gating hypothesis suggests that an incoming stimulus will create a local neuronal inhibitory activity that will specifically inhibit (and thus gate or filter out) the response to a second identical stimulus. The second stimulus, in being similar to the first one, carries no new information and thus is inhibited so as not to flood higher cortical centers with irrelevant information.

Another possible theory to explain the attenuating effect of a previously presented stimulus involves refractory periods. If refractoriness were responsible, the decreased amplitude of the response to the second stimulus would depend on the recovery status of the neuronal pool stimulated by the first stimulus. Evidence supporting the refractory theory comes from the fact that the amplitude of the second response shows better recovery progressively as the time between the two stimuli increases (Zouridakis and Boutros, 1992). Most neurons, however, need only a few milliseconds to reset their ionic equilibrium and regenerate their internal energy (Freedman et al., 1991). Thus, refractoriness is an unlikely explanation of the attenuation of the P50 responses seen at 500-ms repetition intervals. Elucidation of the mechanisms of amplitude attenuation with stimulus repetition is crucial to the understanding of the neurobiological meaning of the decreased capacity to attenuate responses with stimulus repetition seen in schizophrenia.

The oddball paradigm provides a measure of the difference in the brain’s response to two different stimuli presented at two different rates. Our hypothesis is that if the attenuation effect is a simple lack of recovery, we should see no increase in amplitude to the infrequent stimulus. If, on the other hand, it is an active and specific gating inhibitory effect, we should expect the brain to recognize the different stimulus and respond to it in as little as 50 ms after stimulus presentation. Our preliminary data from nine normal volunteers support the second hypothesis (Boutros, 1993).
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