

Cocaine-dependence and cocaine-induced paranoia and mid-latency auditory evoked responses and sensory gating[☆]

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Abstract

Cocaine-dependence has been shown to affect the amplitudes of the P50 mid-latency auditory evoked response (MLAER) as well as P50 sensory gating. The effects on subsequent MLAERs (N100 and P200) have not been examined. The objective of the current study was to further assess the effects of chronic cocaine use on the P50, N100, and P200 components. Thirty-four, at least three weeks abstinent, cocaine-dependent individuals and 34 age and gender matched healthy controls were examined. The amplitudes, latencies and gating measures were calculated and compared between the groups. The N100 and P200 were significantly smaller in patients as compared to control subjects. Sensory gating of the P50, the N100, and the P200 were deficient in cocaine-dependent subjects. Latencies of all measured components were prolonged in subjects who reported developing paranoia while intoxicated. Finally, a positive correlation was found between length of abstinence and evoked response amplitudes. We conclude that the effects of cocaine on sensory gating extend beyond the P50 to the N100 and the P200 components. The data also suggest that prolonged latency of the evoked potentials may be a correlate of cocaine-induced psychosis. Finally, the data suggest that some recovery of amplitude and gating occurs with abstinence.

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1. Introduction

Characterization of the effects of chronic cocaine use on brain function is important, as these effects may influence the ability of the patient to stay abstinent as well as the likelihood of developing psychiatric or

neurological complications with continued cocaine use. The mid-latency auditory evoked responses (MLAERs) occur between 10 and 250 ms from the auditory stimulus (Buchsbbaum, 1977). The early positive component, occurring between 35 and 90 ms after the stimulus (P50), has been shown to be resistant to alterations by attentional factors (Jerger et al., 1992; Cardinas et al., 1997), and may reflect a pre-attentive stage of information processing. The N100 (a negative component occurring between 75 and 150 ms) and the P200 (a positive component occurring between 150–250 ms) MLAERs have been associated with later

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(attentive) stages of information processing (Wilkinson and Morlock, 1967).

The effects of chronic cocaine use on the P50 have been examined in a number of studies. The P50 component is believed to play a significant role in the brain's ability to filter out incoming irrelevant sensory stimuli (sensory gating) thus protecting the higher cortical centers from being flooded with irrelevant input and preserving its ability to handle relevant stimuli (Freedman et al., 1983). Our prior work suggests that chronic cocaine use can result in decrease in the P50 amplitude and that cocaine effects on the P50 are persistent for at least two weeks of abstinence (Boutros et al., 1993; 2000). These findings were replicated in ten African American male cocaine-dependent subjects who have been abstinent for two weeks but not in a group of active alcoholics (Fein et al., 1996). The same finding was later confirmed in a more racially mixed group (Adler et al., 2001). On the other hand the effects of chronic cocaine use on the amplitudes or latencies of later MLAERs (N100 and P200) have not been well-characterized. The amplitude and latency of the P200 component were found not to differ from healthy controls when examined in an oddball paradigm in abstinent cocaine-dependent subjects (Biggins et al., 1997).

Similarly, the effects of chronic cocaine use on gating of the P50 were examined in a number of studies, while the effects on gating of the subsequent MLAERs (i.e., N100 and P200) have not been adequately examined. We have recently suggested that the N100 component may also be playing a role in mediating early attentive sensory gating functions (Boutros et al., 1999). Fein et al. (1996) were the first to report a significant decrease of the P50 gating in 10 African American cocaine-dependent subjects two weeks after their last cocaine use. The finding was later replicated in a more racially mixed group (Adler et al., 2001). More recently we provided further evidence of a deleterious effect of chronic cocaine on gating of the P50 response (Boutros et al., 2002). We have recently also provided evidence that the sensory gating deficit of schizophrenia patients is not limited to the P50 phase of information processing but extends to the N100 and P200 phases (Boutros et al., 2004). Utilizing stimulus trains with different inter-stimulus intervals (ISIs), we provided evidence that habituation of the N100 was decreased in cocaine-dependent subjects when short ISIs were used (Boutros et al., 2000).

Although the development of paranoid symptoms is common with cocaine use, not all cocaine users develop such symptoms despite prolonged and heavy exposure

(Satel and Edell, 1991). These observations suggest that the development of psychotic symptoms is not simply the result of excessive cocaine use and suggest that affected individuals may be predisposed to this drug-induced state (Cubells et al., 2000). Prior work by this group suggested an association between a P50 sensory gating deficit and the emergence of paranoid symptoms during cocaine use (Boutros et al., 2002).

In this study we sought to further characterize evoked potential abnormalities of abstinent cocaine-dependent individuals, examine sensory gating at the P50, N100, and P200 stages of information processing, and examine evoked potential correlates of cocaine-induced psychosis.

2. Methods

2.1. Subjects

Thirty-four (fourteen men and twenty women; a mean age of 35.9 and a range of 19 to 49 years of age), participants who met DSM-IV criteria for Cocaine Dependence (CD) during the past year, and who have been free of cocaine use for at least three weeks, were recruited to the study (mean length of abstinence 16 weeks with a range from three weeks to 18 months). Thirty-three age and gender matched healthy control (HC) subjects were also examined (mean age 35.8, range 21 to 52). The groups were not matched for ethnic backgrounds. The CD group contained 17 African American, six Hispanic and eight Caucasian individuals while the control group included only eight African American, two Hispanic and 20 Caucasian individuals. Also, education differed significantly between groups. While most CD subjects did not have a higher education, most control subjects did. All CD subjects were recruited from drug-rehabilitation or residential drug-treatment facilities in the New Haven area where random urine drug screening (UDS) is routinely performed. Based on the Cocaine Experience Questionnaire (CEQ) none of the subjects had psychiatric symptoms judged to be independent of cocaine use (Satel and Edell, 1991). The CEQ specifically inquires about the presence of paranoid or other psychotic symptoms during periods of abstinence. The CEQ assesses other use variables as well (age at onset of use, duration, intensity, route, etc.). These data were reported elsewhere (Floyd et al., *in press*). Use of alcohol within the context of moderating the effects of cocaine was permitted. Moreover, all subjects denied use of alcohol during the three weeks preceding recording of evoked potentials (EPs). This history was verified with the

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