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Mid-latency evoked potentials in self-reported impulsive aggression

Rebecca J. Houston, Matthew S. Stanford*

University of New Orleans, Department of Psychology, New Orleans, LA 70148, USA

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Abstract

The present study was conducted to examine psychophysiological differences in arousability among individuals who display impulsive aggressive outbursts. Amplitude and latency for the mid-latency evoked potentials (P1, N1 and P2) were obtained at scalp electrode sites. The evoking stimuli were three intensities (low, medium, high) of photic stimulation. Compared to non-aggressive controls, impulsive aggressive subjects showed significantly reduced P1 amplitude, which is indicative of an inefficient sensory gating mechanism. In addition, these subjects exhibited significantly larger N1 amplitude implying an enhanced orienting of attention to stimuli. Impulsive aggressive subjects also exhibited shorter P1, N1 and P2 peak latency. These results suggest that impulsive aggressive individuals may display quicker orienting and processing of stimuli in an attempt to compensate for low resting arousal levels. Finally, impulsive aggressive subjects augmented the P1–N1 component more frequently than controls, which is consistent with previous studies examining impulsivity and sensation seeking. Together, these findings extend previous work concerning the underlying physiology of impulsive aggression. It has been suggested that impulsive aggressive individuals may attempt to compensate for low resting arousal levels by engaging in stimulus seeking behaviors. Accordingly, the present findings imply similar physiological compensatory responses as demonstrated by heightened orienting of attention, processing and arousability. In addition, a compromised sensory gating system in impulsive aggressors may exacerbate such circumstances, and lead to later cognitive processing deficits.

Keywords: Aggression; Evoked potentials; Augmenting/reducing; P1; N1; Arousal; Attention

* Corresponding author. Tel.: +1-504-280-5525; fax: +1-504-280-6049.
E-mail address: mstanfor@uno.edu (M.S. Stanford).

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1. Introduction

Violence is a widespread problem in today's society, and individuals who commit such acts often display impulsive aggressive behavior out of proportion or inappropriate to the eliciting situation or stimuli. According to Barratt et al. (1997) impulsive aggression is defined as 'a hair trigger response to a stimulus that results in an agitated state and culminates in an aggressive act; during the agitated state, interpersonal communication is non-adaptive and information processing appears to be inefficient.'

In order to develop treatments and interventions for this type of problem behavior, psychophysiological research has been conducted with violent and impulsive persons (Barratt et al., 1997; Gerstle et al., 1998; Mathias and Stanford, 1999). The primary focus of these studies has been to identify differences in the neural function of those individuals who display impulsive aggressive behaviors. Accordingly, physiological measures in these individuals have indicated a low level of arousal during resting conditions (Fishbein et al., 1989; Convit et al., 1991; Barratt et al., 1997; Mathias and Stanford, 1999). However, it has also been suggested that impulsive aggressors display a greater magnitude of change in arousal in reaction to a stimulus, when compared to controls (Mathias and Stanford, 1997). In conjunction with low arousal levels, there is some question as to the efficiency of attentional processes in aggressive individuals as well (Raine, 1997).

1.1. Augmenting / reducing

One paradigm that aggression research has rarely utilized is that of augmenting–reducing. Augmenting–reducing is a measure of sensory inhibition that was developed through investigations of individual differences. Petrie (1978) classified individuals as 'augmenters' or 'reducers' based on their style of response to stimulation in a kinesthetic figural after-effects (KFA) task. Augmenters tended to judge the magnitude of a standard stimulus as larger after kinesthetic stimulation while reducers judged the magnitude as smaller. Petrie hypothesized that the KFA was an index of a general central nervous system mechanism regulating the intensity of incoming sensory stimulation. This idea of a mechanism moderating sensory input led to psychophysiological investigations utilizing the evoked potential (EP). Reliable individual differences in the way evoked potentials change (i.e. change in amplitude) with different stimulus intensities can be easily determined, and have been applied in the same manner as Petrie's KFA hypothesis. This measure is referred to in the literature as augmenting–reducing or A/R. Therefore, two types of individuals who regulate sensory input differently have been hypothesized; augmenters, who tend to increase the perceived intensity of a stimulus, and reducers, who tend to reduce this perceived intensity.

Buchsbaum and Silverman (1968) developed the visual EP technique for measuring A/R. Typically, different intensities of light flashes are presented in randomized blocks (Buchsbaum et al., 1973). The magnitude of change from the P1 component to the N1 component forms the bases for the variable of EP augmenting–reducing. Typically the P1, N1, and P2 are visually identified for each of the intensities for each session. The P1 and P2 are positive in polarity and occur approximately 76–112 and 168–240 ms after stimulus presentation, respectively. The N1, however, has a negative polarity and occurs approximately 116–152 ms after stimulus presentation. The amplitude, as measured from peak to peak (P1 to N1), is examined from one stimulus level to the next. Increases in amplitude represent an increased cortical response with increasing stimulus intensity which is considered augmenting. Decreases or no changes in amplitude represent reducing, and are produced by reductions in response at the higher stimulus intensities. In other words, these individuals reach a threshold beyond which the amplitude begins to decrease.

It has been pointed out by several researchers (Sales, 1971; Barnes, 1976; Davis et al., 1984) that the A/R dimension defined by Petrie is very similar to the aspect of Pavlovian neurological typology known as 'strength of the nervous sys-