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Cognitive strategy usage in long-term survivors of severe traumatic brain injury with persisting impulsive aggression

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Abstract

Impulsive aggression (IA) is a relatively common phenomenon in the general nonpatient population and IA, among other forms of impaired self regulatory behavior, is common sequelae of traumatic brain injury. Impaired self regulatory behavior has been associated with dysfunction of orbital frontal cortex. This study examined strategy development and usage in a group of survivors of traumatic brain injury (TBI) who exhibited persisting problems with impulsive expressions of aggression. All participants were administered the Revised Strategy Application Task (R-SAT) and a series of personality questionnaires. The results indicated that the subjects with IA selected and used strategies that were significantly less efficient than those of the nonaggressive controls. The present study confirms, using an explicit and direct measure, previously reported deficits in strategy usage in IA and supports the notion of inefficient cognitive processing indicated by event related potential (ERP) studies. Additionally, as impaired performance on the R-SAT has been associated with lesions of ventromedial prefrontal cortex, this study provides the first concrete neuropsychological data implicating this region in IA. Further study is necessary to clarify the nature of the apparent orbital frontal dysfunction in IA and to delineate the role of impaired strategic processing and related deficits in the genesis and expression of impulsive aggression.

Keywords: Impulsive aggression; Cognition; Frontal lobes; Traumatic brain injury

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1. Introduction

"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" (Barratt, Stanford, Kent, & Felthous, 1997, p. 1046). Research into the personality and neurocognitive features of persons who exhibit impulsive aggression has revealed significantly higher impulsiveness and hostility (irritable impulsiveness; Coccaro et al., 1989; Stanford, Greve, & Dickens, 1995), decreased verbal abilities (Barratt et al., 1997), and deficient strategic planning and problem-solving (Stanford, Greve, & Gerstle, 1997). There are also consistent changes in event-related potentials (ERP) indicative of cognitive inefficiency (Barratt et al., 1997; Mathias & Stanford, 1999). These findings have been replicated across a variety of populations including incarcerated prisoners (Barratt et al., 1997), neuropsychiatric patients (Mungas, 1988), and college students (Stanford et al., 1997). Further, impulsive aggresion (IA) or similar phenomena are remarkably common in the general population with incidence estimates ranging as high as 25 (Fava et al., 1991; Stanford et al., 1995).

It has been suggested that behavioral dyscontrol, like that seen in IA, results from a loss of frontal lobe inhibition over the subcortical limbic structures involved in the facilitation of aggression and other primitive impulses (Grafman, 1989, 1994a, 1994b; Grafman et al., 1996). Impulsive aggression (whether verbal or physical in nature) and other forms of impaired self regulatory behavior are common sequelae of traumatic brain injury (TBI; Eslinger, Grattan, & Geder, 1995). Such deficits contribute to poor community and vocational reintegration and may also be an important stimulus for long-term institutional placement, posing a serious challenge to rehabilitation (Eslinger et al., 1995). Recently, Greve et al. (2001) attempted to replicate the research concerning personality and cognitive correlates of IA in a traumatic brain injury sample. Consistent with previous research on IA, the survivors of TBI with persisting IA were more impulsive, irritable, and antisocial than the nonaggressive control participants. They also had a higher incidence of premorbid aggressive behavior, were younger, and had shorter tenure in the program. Unlike past research, however, no differences were found in performance on a variety of neuropsychological measures.

The failure of Greve et al. (2001) to find clear neurocognitive differences between the IA and nonaggressive control (NC) participants is not entirely surprising for three reasons. First, their patients were generally more cognitively impaired and more variable in their performance than the non-brain injured samples previously studied. Second, the deficits observed in IA on measures of higher order cognition (e.g. Stanford et al., 1997) were often quite subtle. Third, it is often the orbitofrontal or ventromedial (terms often used interchangeably) frontal cortex which is implicated in disorders of self-regulation (Eslinger et al., 1995) such as IA and it is well known that most traditional neuropsychological measures are relatively insensitive to lesions or dysfunction of this region (e.g. Cicerone & Tanenbaum, 1997; Eslinger & Damasio, 1985). This may also be one reason the neurocognitive deficits observed in other samples have often not been particularly robust.

Based on subtle qualitative differences on several standard clinical neuropsychological measures, Stanford et al. (1997) inferred a strategic processing deficit in IA. Deficits in strategic processing and efficient action planning are commonly seen with orbital/ventromedial frontal