

Inhibitory attentional control in patients with frontal lobe damage

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Abstract

The performance of a group of frontal lobe lesion and a group of frontal lobe dementia patients was compared with the performance of their respective matched normal control groups on two tests of inhibitory attentional control—the stop-signal reaction time task and a negative priming task. Both patient groups responded significantly slower than their respective normal control groups, but they showed only marginally significant selective impairments on the measures of inhibition. The data suggest that the specific inhibitory processes evaluated by these two tests are, in general, spared in patients with focal frontal lobe lesions or frontal lobe degeneration.

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1. Introduction

It is widely accepted that frontal lobe damage causes impairments in inhibition. However, the term inhibition (and frontal disinhibition, respectively) is often used to describe two different aspects of this cognitive function. The first one is manifested in the realm of social conduct as social disinhibition, profanity, impulsivity, tactlessness, loss of social responsibility, and lack of respect for social conventions. The most famous case to illustrate these effects of prefrontal lobe lesions on social behavior is that of the landmark patient Phineas Gage (Harlow, 1848, 1868). Modern imaging techniques, which were used to reconstitute the accident and to determine the location of Gage's lesion, as well as the observations of other patients with similar anatomical and behavioral patterns, led to the hypothesis that social conduct regulation depends on the orbitofrontal cortex, or more

specifically, on the ventromedial prefrontal cortex (Damasio, Tranel, & Damasio, 1990; Dimitrov, Phipps, Zahn, & Grafman, 1999; Eslinger, 1998; Saver & Damasio, 1991).

The other type of inhibition is a component of the process of selective attention and is manifested in the suppression of goal irrelevant stimuli. There are numerous lesion and neuroimaging studies suggesting the critical involvement of the frontal lobes in this type of inhibitory control but their findings are different and sometimes inconsistent with respect to the frontal regions found to subservise the particular attentional and inhibitory processes. Glosser and Goodglass (1990), Wilkins, Shallice, and McCarthy (1987), Woods and Knight (1986), and Rueckert and Grafman (1996) reported impairments in sustained attention in patients with right frontal lobe lesions (FLL). Stuss, Benson, Kaplan, Weir, and Della (1981) found that FLL patients were not impaired compared to matched normal control (NC) subjects on several attentional measures including the Stroop. Vendrell (1995) reported that the right prefrontal lateral region appeared to be the most important

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region for maintaining correct Stroop performance, that left lobectomies did not impair Stroop performance, and that lesions in the anterior cingulate cortex (ACC) did not produce selective changes in the Stroop effect, but simply resulted in increased reaction times in the non-interference condition. Two neuroimaging studies with normal subjects (Bench et al., 1993; Pardo, Pardo, Janer, & Raichle, 1990) concluded that the right ACC plays a role in the attentional aspects of the Stroop task. Deficits in selective attention in patients with right frontal lobe lesions were observed in several studies (Alivisatos & Milner, 1989; Knight, Hillyard, Woods, & Neville, 1980, 1981), whereas Lee, Wild, Hollnagel, and Grafman (1999) reported that the cognitive processes underlying visual selective attention and response competition as measured by a task based on space and target feature identity were, in general, spared in patients with frontal lobe lesions. In a neuroimaging study by Corbetta, Miezin, Dobmeyer, Shulman, and Petersen (1991), it was found that selective attention during visual discrimination of shape, color, and speed tasks induced activation in the basal ganglia, the lateral orbitofrontal (OF) cortex, and the premotor cortex. Effects of right and left OF lesions on different interference measures were reported in several studies (Fuster, 1985; Posner, Early, Reiman, Pardo, & Dhawan, 1988; Stuss et al., 1982), while Milner, Petrides, and Smith (1985) reported an effect of dorsolateral prefrontal lesions on interference. The lack of specificity and the inconsistency in some of these results might be due to the different etiologies, different lesion localization, and different ways in which the attentional and inhibitory processes were tested and measured (Stuss et al., 1999).

Stuss et al. (1999) suggested that clearly distinguishing among possible anterior inhibitory attentional processes would facilitate research in attention. In the present study we also adopted the approach of fractionation of the inhibitory mechanisms of selective attention in order to evaluate several specific aspects of inhibitory control in patients with frontal pathology and to try and identify the precise anatomic correlates of any observed cognitive impairments. We studied a homogeneous group of patients with well-documented focal frontal lesions and a group of patients with frontal lobe dementia (FLD)—a progressive bilateral fronto-temporal cortical degenerative disease in which cognitive deficits, including distractibility, impulsivity, and disinhibition, often accompany personality and behavioral changes (Elfgren, Ryding, & Passant, 1996; Filley, Kleinschmidt-De Masters, & Gross, 1994; Frisoni et al., 1995; Gregory & Hodges, 1996; Miller, 1997; Moss, Albert, & Kemper, 1992; Talbot, 1996) that are similar to, but generally more severe than the ones observed in patients with focal frontal lobe lesions (Damasio, 1996; Grafman, 1989; Hecaen & Albert, 1978). We employed two tasks: a stop-signal reaction time task, developed by

Logan and colleagues (Logan, Cowan, & Davis, 1984; Schachar, Tannock, Marriott, & Logan, 1995; Williams, Ponesse, Schachar, Logan, & Tannock, 1999), and a lexical negative priming task, extensively used by Hasher and colleagues (Kane, Hasher, Stoltzfus, Zacks, & Connelly, 1994).

The inhibitory control functions of human and non-human primates with frontal damage have been evaluated predominantly using the go/no-go task. Damage to the DLPFC has been found to impair response inhibition in monkeys in the studies of Iversen and Mishkin (1970), Butters, Butter, Rosen, and Stein (1973), and Sasaki, Gemba, and Tsujimoto (1989). A number of other studies reported that patients with frontal pathology were impaired on the go/no-go task (Decary & Richer, 1995; Leimkuhler & Mesulam, 1985). The involvement of frontal areas in response inhibition has been also documented in several neuroimaging studies, which employed the go/no-go task in humans. DLPFC activation during mixed go/no-go trials minus go trials was observed in a PET study by Kawashima et al. (1996) and in a blocked fMRI study by Casey et al. (1997). The right inferior prefrontal cortex was found to be involved in inhibition during no-go trials in an event-related fMRI study by Konishi et al. (1999). Right DLPFC dominance for inhibitory function was observed in an event-related fMRI study by Garavan, Ross, and Stein (1999), in which a paradigm similar to the go/no-go one was utilized.

The stop-signal task we employed is a computerized measure of inhibitory control similar to the go/no-go tasks, but designed to isolate the inhibitory processes more effectively. The stop-signal type of inhibition is conceptualized as one of several internally generated acts of control in the repertoire of a higher order executive system that regulates behavioral execution (Goldman-Rakic, 1987; Shallice, 1982). The stop-signal procedure is a laboratory analogue of a situation that requires an individual to stop a planned or prepotent response. On the stop-signal task, a subset of trials from a series of regular choice reaction time trials is interrupted by a stop signal (Logan et al., 1984), which instructs the subjects to withhold the response that was in preparation. It becomes harder to suppress a response as the stop signal is presented closer to the go-signal induced moment of responding. A profile of inhibitory efficiency over time is derived by manipulating the stimulus onset asynchrony between the go-signal and stop signal. The stop-signal reaction time indicates the speed of the inhibition process.

Negative priming refers to the slowing of responses to targets which were distractors in immediately preceding trials (prime trials followed by probe trials) and could be viewed as an indication of inhibition, which is a normal component of selective attention. Metzler and Parkin (2000) reported reversed negative priming following

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