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Diffuse axonal injury due to traumatic brain injury alters inhibition of imitative response tendencies

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Abstract

It is well known that traumatic brain injury particularly affects the frontal lobes. Consequently, patients often suffer from executive dysfunction and behavioral disturbances. Accordingly, our study aimed at investigating patients after traumatic brain injury with two tasks involving different functional processes and structural networks supported by the frontal lobes. Two paradigms were applied: the Stroop color-word task and a task in which subjects had to inhibit imitative response tendencies. We selected a patient group solely with diffuse axonal injury, as this type of injury is homogenous and is correlated with cognitive dysfunction more than focal contusions. To evaluate long-term effects most relevant for rehabilitation, we selected a patient group whose brain injuries dated back several years. Our results show that patients with diffuse axonal injury inhibited imitative responses more successfully than control subjects, whereas executive processes examined with the Stroop task were unaltered. Interestingly, impairments were tightly correlated both with the length of the post-traumatic amnesia predicting outcome in traumatic brain injury and with behavioral disturbances. Impairments in the imitation-inhibition task may indicate alterations in an anterior frontomedian neural network even years after traumatic brain injury.

Keywords: Diffuse axonal injury; Executive functions; Inhibition; Imitation; Stroop; Traumatic brain injury

1. Introduction

Traumatic injuries represent the leading cause of death and disability in young adults in industrialized countries (Lucas, 1999). Injuries often involve the brain and cause neurological impairment, cognitive deficits, and alterations in consciousness and personality. Three main pathological pathways are involved in traumatic brain injury (TBI): (i) focal contusional damage resulting from local impact, (ii) diffuse axonal injury (DAI) resulting from head acceleration, especially rotational, and (iii) secondary damage, resulting, for example, from mass compressive effects through edema or hemorrhage (Fork et al., 2005; Wallesch, Curio, Galazky, Jost, & Synowitz, 2001). DAI, mainly caused by road traffic accidents (Scheid, Preul, Gruber, Wiggins, & von Cramon, 2003), results from damage to the axonal membrane and/or cytoskeleton (Fork et al., 2005; Wallesch et al., 2001). DAI is diagnosed indirectly by traumatic microbleedings on magnetic resonance imaging (MRI) (Fig. 1; Scheid et al., 2003). In comparison to focal contusions, it is anatomically more homogenous and more closely correlated with cognitive dysfunction (McDonald, Flashman, & Saykin, 2002). DAI affects predominantly frontal brain regions (Cicerone, Levin, Malec, Stuss, & Whyte, 2006; Okamoto, Hashimoto, Aoki, & Ohashi, 2007; Scheid et al., 2003; Stamatakis, Wilson, Hadley, & Wyper, 2002; Wilson, 2005). Accordingly, it is related to behavioral and cognitive symptoms of frontal lobe dysfunction, especially heightened susceptibility to interference and decreased semantic fluency (Wallesch et al., 2001).

In summary, it is well known that DAI due to TBI affects particularly the frontal lobes and consequently leads to executive dysfunction and behavioral disturbances. Hence, our study aimed at investigating performance in patients with DAI in

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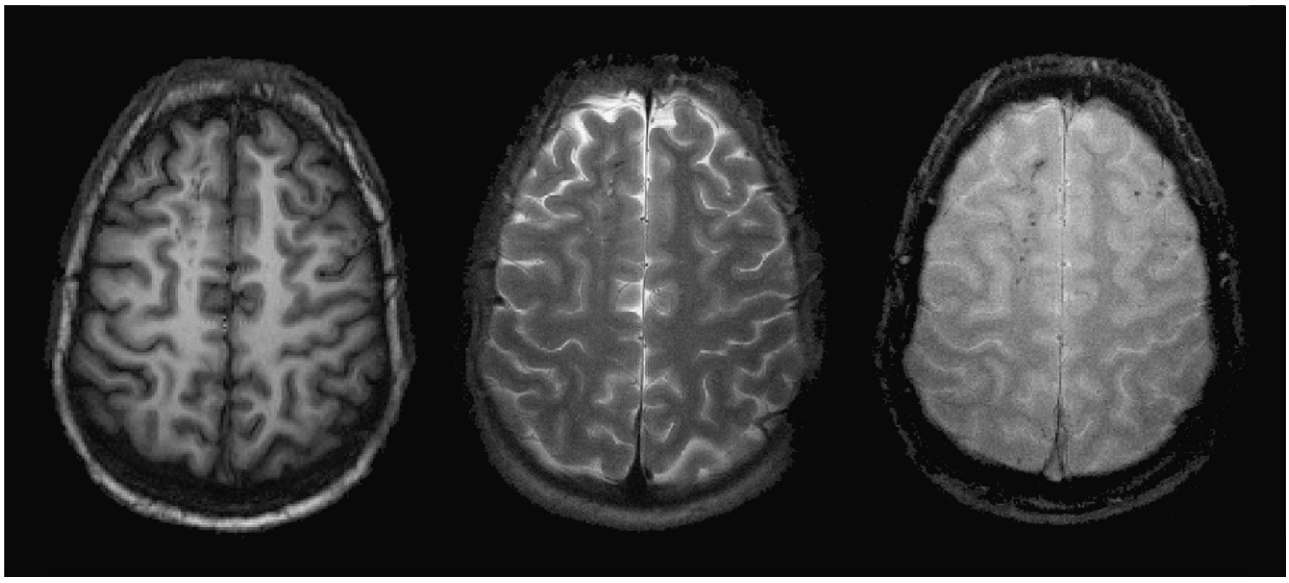

Fig. 1. Images of subject 3, who was the driver of a motorbike involved in a traffic accident. Initial score on the Glasgow coma scale was 10. Multiple traumatic microbleeds are particularly shown in the white matter of the left superior frontal gyrus. T1-, T2- and T2*-weighted images. Axial view sections obtained from the identical location. Neurological orientation (left on left).

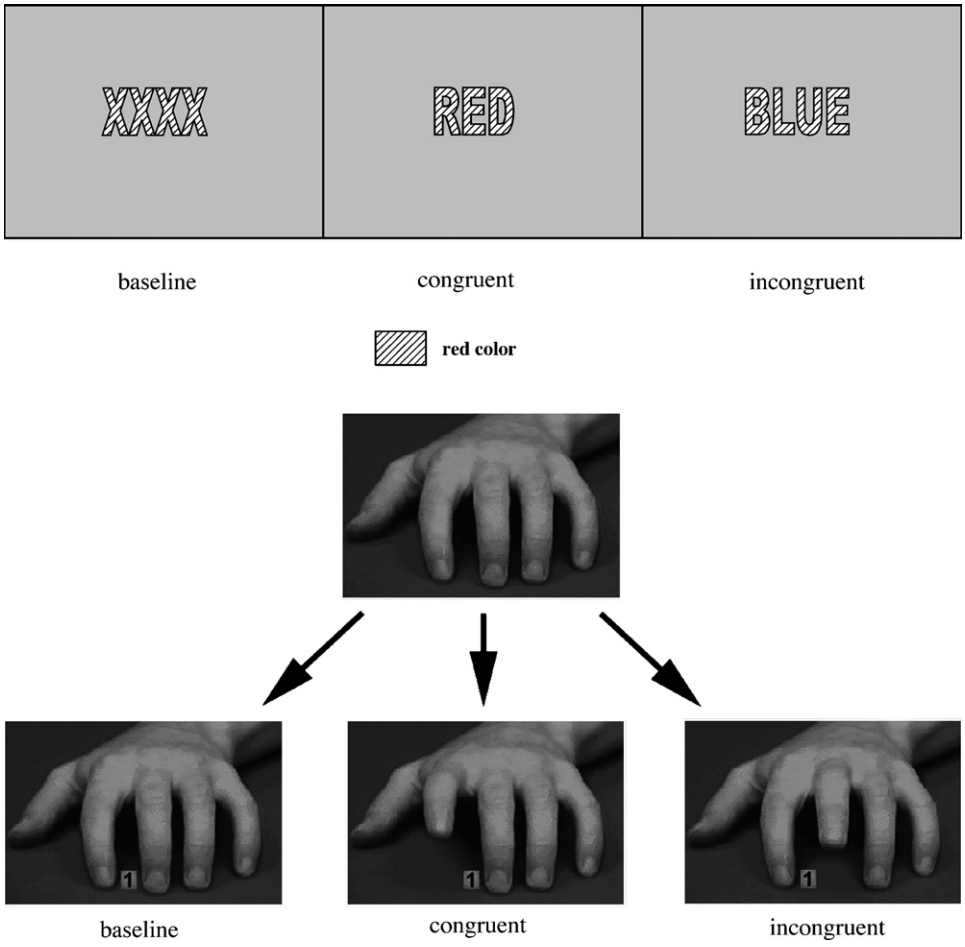

Fig. 2. Schematic drawings of the Stroop task (top) and the imitation-inhibition task (bottom).