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Research report

Caudate nucleus and social cognition: Neuropsychological and SPECT evidence from a patient with focal caudate lesion

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ARTICLE INFO

Article history:
Received 20 May 2011
Reviewed 01 August 2011
Revised 3 October 2011
Accepted 9 January 2012
Action editor Stefano Cappa
Published online 15 January 2012

Keywords:
Caudate nucleus
Theory of Mind
Social cognition
Emotion

ABSTRACT

Most studies in social cognition have focused on developmental diseases or analyzed the consequences of acquired frontal lesions on the integrity of Theory of Mind (ToM), but, to our knowledge, none to date has addressed the eventual consequences of damage to the basal ganglia on ToM. To investigate the possible consequences of such lesions on social cognition, we tested a selected patient, MVG, a 44-year-old man with a focal caudate nucleus (CN) lesion following stroke. In the aftermath of this stroke, MVG shows loss of empathy and difficulties recognizing emotions in others. The dual aims of this study were first, to evaluate the implications of CN on ToM and recognition of emotion, and second, to discuss these results as a consequence of a disconnection of the sub-cortical orbito-frontal (OF) loop due to caudate damage. We performed a complete neuropsychological assessment of MVG, as well as different tasks evaluating social cognition, such as the Faux-Pas Test and the Reading the Eyes in the Mind Test. No deficits were found in the neuropsychological tests. However, on tasks assessing social cognition, MVG showed impairments in the "warm" or "affective" part of ToM as well as in the ability to recognize negative emotions (i.e., sadness and fear). These results indicate that damage to the head of the left CN can lead to impairment of ToM and emotion recognition. Furthermore, the data shows that, in MVG, such impairment appears to be due to a disconnection of the sub-cortical OF circuit resulting from damage to the CN. Neuro-imaging data tends to confirm this hypothesis by bringing out a hypo-perfusion in both, the territory of his left CN and prefrontal (i.e., ventromedial) brain areas.

1. Introduction

The basal ganglia (BG) (i.e., striatum, globus pallidus, substantia nigra, sub-thalamic nucleus) are involved in the regulation of motor functions, motivation and procedural learning (Graybiel et al., 1994; Yin and Knowlton, 2006), as well as in cognitive functioning, behavior and personality (Abdullaev et al., 1998; Laplane et al., 1989), with the striatum, an important part of

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0010-9452/$ – see front matter © 2012 Elsevier Ltd. All rights reserved.
doi:10.1016/j.cortex.2012.01.004

the BG, involved in affective processes. The striatum can be divided into the ventral striatum, which contains the nucleus accumbens, and the dorsal striatum, which contains the caudate and putamen (Utter and Basso, 2008). Lesions in the caudate nucleus (CN) are most likely to cause behavioral alterations (Bhatia and Marsden, 1994), as well as cognitive losses in Huntington's disease (Lawrence et al., 1998) and Parkinson's disease (Rinne et al., 2000), and possibly also in obsessive compulsive disorder (Baxter, 1992). Lesions in the CN have been associated with a wide range of behavioral alterations, including abulia (Trillet et al., 1990), disinhibition, a variety of affective and personality disorders (Kumral et al., 1999) and depression (Caplan et al., 1990), suggesting that the sequelae of CN damage are not always homogeneous among patients. However, a consensus has been established concerning the behavioral and cognitive consequences of CN damage, which appear commonly in the foreground.

The BG are thought to be connected to well delimited regions of the frontal cortex by five separate and parallel loops (Alexander et al., 1986). Two of these loops connect the CN to frontal neocortical regions of the brain, which have been implicated in higher cognition; these loops are the dorsolateral (DL) loop, which regulates executive functions and involves the DL part of the head of the CN, and the orbitofrontal (OF) circuit, which mediates affective processes and involves the ventro-medial part of the caudate. On the basis of the model of these fronto-sub-cortical circuits, detailed predictions have been made regarding the causal relationship between specific behavioral consequences and the disruption of a particular loop (Lawrence et al., 1998; Mega and Cummings, 1994). Behavioral disturbances following damage to the CN seem to result from an impairment of function in cortical areas, due to a loss of striatal efferences from the CN (Kumral et al., 1999). That is, these behavioral disturbances result from the disturbances in connectivity among cortico-sub-cortical networks due to damage to the BG. A description of two patients supports this hypothesis (Benke et al., 2003). Indeed, from a phenomenological perspective, the observed behavioral (i.e., abulia) and cognitive (i.e., executive functions) modifications since stroke would be consistent with a disruption of the anterior cingulated-prefrontal and the DL prefrontal circuits, respectively.

Hence, if impairment of one of the BG pertaining to one of these circuits induces deficits similar to those caused by a lesion of the cortical area belonging to that loop, there may also be consequences of this impairment in another domain, namely social cognition. Indeed, the prefrontal cortex (PFC), comprising the orbito-frontal cortex (OFC), has been shown to play an important role in social cognition (Adolphs, 1999).

Social cognition alludes to the processes by which we make sense of ourselves, the social environment in which we live and the individuals around us (Fiske, 1993; Macrae and Bodenhausen, 2000) and the ability to represent internal somatic states, knowledge about the self, perceptions of others and interpersonal motivations which are carefully orchestrated to support skilled social functioning (Fiske and Taylor, 1991). It refers to a relatively large number of constructs. The most representative mechanism of social cognition is Theory of Mind (ToM), which alludes to a skill that underlies humans' ability to engage in complex social interactions: the ability to make inferences about others' mental states. Within ToM, there is both a cognitive or "cold" component and an empathic, affective or "warm" component (Coricelli, 2005). Cognitive ToM refers to the ability to make inferences about the cognitive states, beliefs, thoughts, intentions and motivations of other people (Brothers and Ring, 1992; Coricelli, 2005), while affective ToM refers to the ability to infer the feelings, affective states and emotions of others (Brothers and Ring, 1992). Furthermore, the neuronal bases underpinning affective and cognitive ToM seem to be different, suggesting that cognitive and affective mentalizing abilities are partly dissociable (Shamay-Tsoory and Aharon-Peretz, 2007).

Empathy, a second component of social cognition, is mainly implicated in prosocial behavior, morality and the regulation of aggression (Eisenberg and Eggum, 2009). Moreover, the term empathy is applied to a broad spectrum of phenomena, from feelings of concern for other people that create a motivation to help them, experiencing emotions that match another's, knowing what the other is thinking or feeling, to blurring the line between self and other (Hodges and Klein, 2001). Empathy is not a passive affective resonance phenomenon with the emotions of others. Rather, goals, intentions, context and motivations play feed-forward roles in how emotions are perceived and experienced. According to a recent model, empathy can be separated in two systems, which are affective empathy (i.e., the capacity to *experience* affective reactions to the observed experiences of others) and cognitive empathy (i.e., *making inferences* regarding the other's affective and cognitive mental states) (Shamay-Tsoory, 2010), which can be impaired selectively [e.g., (Dziobek et al., 2008)]. The term "empathy" as used in this study only alludes to affective empathy, since cognitive empathy refers to ToM.

The recognition of emotions in faces is also an essential component of social cognition. Facial expressions signal important information about the internal states of others and the external events that may have elicited those expressions (Ekman, 1997). Individuals use this information to guide their social behavior.

Many studies have enabled identification of the cerebral areas implicated in ToM. The social cognition tests used in this study have frequently been used in functional neuroimaging and lesion studies in order to enable identification of the cerebral areas implicated in ToM.

For example, a popular paradigm of ToM assessment in neuro-imaging studies consists in using false-belief tasks [inspired by (Happé et al., 1998) task; see below for a task description] during functional magnetic resonance imaging (fMRI) measures. Indeed, this paradigm has shown that the medial PFC [e.g., (Saxe and Wexler, 2005)], more precisely Brodmann areas 8 and 9, plays a crucial role in this ability in healthy adults [e.g., (Fletcher et al., 1995; Russell and Sharma, 2003)]. Implication of other brain areas such the right lateral rostral PFC has also been objectified by this type of paradigm [e.g., (Sommer et al., 2007)]. A study using event related potentials provided further evidence for the involvement of the left frontal areas in achieving this task (Sabbagh and Taylor, 2000). Furthermore, the Faux-Pas Test is a popular test of ToM assessment in lesion studies. Indeed, it has highlighted impairment of this ability in patients with lesions