



Emotion and object processing in Parkinson's disease

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ABSTRACT

The neuropsychological literature on the processing of emotions in Parkinson's disease (PD) reveals conflicting evidence about the role of the basal ganglia in the recognition of facial emotions. Hence, the present study had two objectives. One was to determine the extent to which the visual processing of emotions and objects differs in PD. The other was to assess the impact of cognitive load on the processing of these types of information. Thirty-one patients with idiopathic PD (IPD) under dopamine replacement therapy (DRT) were compared to 30 control subjects on emotion and object recognition tasks. Recognition of objects was more accurate and faster than recognition of facial expressions of emotion, for both groups of subjects. In a second experiment using an N-back procedure with the same stimuli—a more demanding task with a higher cognitive load—patients with IPD were as accurate as control subjects in detecting the correct sequential presentation of stimuli, but were much slower in their decision responses. This indicates that IPD patients under DRT are not impaired in encoding emotion or object information, but that they have difficulty with the processing demands of the N-back task. Thus, patients with IPD appear to be more sensitive to cognitive load than to type of information, whether facial emotions or objects. In this perspective, one must consider that a deafferented dopaminergic system has problems processing more complex information before one can posit the existence of deficits affecting a specific type of information.

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1. Introduction

The involvement of the basal ganglia in the processing of emotion has been widely studied for nearly two decades. Studies of patients with lesions in or dysfunction of the basal ganglia have reported deficits in the processing of emotional prosody, in both the perceptive and expressive modalities (Breitenstein, Van Lancker, Daul, & Waters, 2001; Dara, Monetta, & Pell, 2008; Weddell, 1994). In line with these findings, neuroimaging studies have also shown neural activation within these subcortical structures during the response to emotions from vocal expressions (Kotz et al., 2003; Wildgruber et al., 2005). Also consistent with the involvement of the basal ganglia in emotional processing is an fMRI study by Phillips et al. (1997), who found activation of the right putamen for the processing of intense disgust.

Patients in the early stages of Huntington's or Parkinson's disease are impaired in the recognition of facial expressions of emotions (Dujardin et al., 2004; Lawrence, Goerendt, & Brooks, 2007;

Sprengelmeyer et al., 1997, 2003). Specifically, patients with Huntington's disease (HD), who suffer from degeneration of medium-sized spiny striatal neurons, are severely impaired in the ability to recognize facial expressions of disgust, while their interpretation of other emotions is relatively preserved (e.g., Sprengelmeyer, Schroeder, Young, & Eppelen, 2006). This impaired recognition of disgust has also been found with vocal expressions of disgust (Hayes, Stevenson, & Coltheart, 2007).

In Parkinson's disease (PD), the evidence as to whether emotion processing is negatively affected and, if so, which specific emotions—if there is such a selective disturbance as that found in HD—is not clear. Sprengelmeyer et al. (2003) examined emotional information processing in PD patients under different conditions: while unmedicated PD patients presented with higher deficits in the recognition of disgust, this specific impairment was not observed in medicated PD patients, even at a more advanced stage of the disease. In these authors' view, the administration of dopaminergic treatment exerts a corrective effect on the performance of patients with PD in the recognition of facial emotions. Dujardin et al. (2004) presented evidence of a broader impairment in the recognition of facial emotions in PD, not limited to expressions of disgust. These authors reported that patients with PD had an impairment in decoding the facial expressions of sadness and anger. Paradoxically, injection of levodopa in healthy subjects leads

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to an impairment in the recognition of emotions (Delaveau, Salgado-Pineda, Wicker, Micalef-Roll, & Blin, 2005).

A few studies, however, present a more complex picture of the involvement of dopaminergic neuromodulation in the recognition of disgust in PD. In their first study, Lawrence, Calder, McGowan, and Grasby (2002) found that acute dopaminergic blockade in healthy subjects temporarily triggers an impairment in the recognition of anger, but not of disgust or other emotions. These authors therefore hypothesized that PD, characterized by a loss of dopaminergic nigrostriatal neurons, is associated with impaired anger recognition. Consistent with these findings, Lawrence et al. (2007) found that PD patients who were suddenly withdrawn from dopamine replacement therapy performed worse than healthy controls in the recognition of anger only, with no difference between the performance of patients with PD and control subjects for the processing of disgust or other emotions. Finally, the severity of the disease appears to determine how subjects respond to emotional stimuli. Yip, Lee, Ho, Tsang, and Li (2003) found that patients with bilateral disease performed worse on an extensive emotion recognition battery (facial and prosodic recognition and identification) than patients with right-sided disease, who only had difficulty with the identification tasks.

The results of the above-mentioned studies of PD patients suggest that the basal ganglia are involved in the processing of emotions. However, this does not appear to be a uniform observation, as some studies failed to show any such impairment. For instance, Adolphs, Schul, and Tranel (1998) found that PD subjects were similar to healthy controls in the recognition of emotions, but suggest that the nature of the dysfunction of the basal ganglia is not sufficient to compromise the processing of this type of information in PD. Pell and Leonard (2005) found little evidence of impairment in processing facial expression of emotions in PD. These authors, however, considered the role of the basal ganglia in the processing of emotion from static faces relative to speech prosody to be more limited. These findings suggest that brain circuits involving subcortical brain areas highlight an adaptive intentional control of behavior, where representations of emotional content are generated in a task-dependent mode (see Elmer & Holmes, 2006).

In sum, the neuropsychological literature on the processing of emotions in PD reveals conflicting evidence about the role of the basal ganglia in the recognition of facial expressions. One interpretation for such divergent results is that corrective dopaminergic treatment (DRT) may compensate for some of these deficits, as the findings of impaired recognition of facial emotions included unmedicated or off-medicated patients (e.g., Dujardin et al., 2004; Lawrence et al., 2002, 2007; Sprengelmeyer et al., 2003;). From this perspective, dysregulation of dopaminergic striatal afferentation is sufficient to reveal impairment in the recognition of emotions.

DRT also appears to modulate the visual recognition of living objects. For example, the performance of parkinsonian patients in the identification of animals is significantly poorer in patients off medication than when they are under DRT (Righi, Viggiano, Paganini, Ramat, & Marini, 2007). These deficits in the recognition of living objects, revealed in the absence of medication and silenced by dopaminergic treatment, suggest that dopaminergic modulation plays a critical role in visual recognition. Until now, all studies have focused on how patients with PD processed emotions, without contrasting their performance in response to another type of information. Thus, it is not clear whether the impairment observed is specifically caused by a disturbance in the processing of emotions or whether it is a reflection of more generalized and nonselective companion deficits in visual information processing.

A related question pertains to the nature of the tasks used in these studies. Rating facial emotions along a continuum, identifica-

tion or recognition of facial emotions were the tasks of choice in most studies cited. These tasks may not be sensitive enough to reveal problems in emotion processing, when patients with PD are taking DRT. Cognitive deficits in memory, executive function and spatial abilities are often observed in the early stages of the disease (e.g., Amick, Schendan, Ganis, & Cronin-Golomb, 2006; Dubois & Pillon, 1997; Taylor & Saint-Cyr, 1995). It is thus conceivable that tasks that tap into higher processes relying on more complex analysis of information could detect deviant cognitive patterns.

Hence, the aim of the present study was twofold. One objective was to determine the extent to which the visual processing of emotions and objects differs in PD. If emotions and objects are treated similarly, it would be difficult to argue that PD patients are specifically impaired in the processing of emotions. The other was to assess the impact of cognitive load, or task difficulty, on the processing of these types of information, using a more ecologically valid approach to the study of visual processing of emotions. Such an approach would help tease out whether it is the difficulty of the task or the type of information which is implicated in the observed impairment, if any.

2. Experiment 1

As mentioned above, it remains unclear whether patients with PD are selectively impaired in their ability to recognize facial expressions of emotions. It is possible that categories of visual information other than emotional information are also affected, thus reflecting a more generalized impairment of visual recognition in PD. The aim of this first experiment was to provide an initial comparison of the recognition of two types of visual information: facial emotions and objects. To do this, we compared patients with idiopathic PD (IPD) to a matched group of healthy controls on tasks of emotion and object recognition.

2.1. Methods

2.1.1. Participants

Thirty-one individuals with a diagnosis of IPD (15 women, 16 men) participated in the study. The patients were recruited from Clinique Sainte Anne, Quebec, Canada. The participants were French-Canadian individuals who had received a diagnosis of probable IPD from a movement disorder specialist (E.P.), using the Hughes Diagnostic Criteria for PD (Hughes, Daniel, Kilford, & Lees, 1992). Patients with a significant comorbid vascular medical history not related directly to their PD (e.g., diabetes, coronaropathy, high blood pressure) or with focal sensory or perceptual deficits were not included in the study. Patients who scored 24 or lower on the Mini-Mental State Examination (Folstein, Folstein, & MacHugh, 1975) were also excluded. The severity of clinical symptoms was assessed according to the Hoehn and Yahr (1967) five-point rating scale. Hoehn and Yahr ratings ranged between 1 and 2.5. All patients had been receiving stable medication doses for at least 2 months and were not affected by motor or affective fluctuations during their waking day. Subjects were not depressed and not demented, according to classical cut-off scores (e.g., Schrag, Barone, Brown, Leentjens, & McDonald, 2007) on the Beck Depression Inventory (Beck & Steer, 1987) and the Mattis Dementia Rating scale (e.g., Kulisevsky & Pagonagabarra, 2009), respectively.

The patients with IPD were compared to 30 healthy controls (16 women, 14 men) who were recruited from among the patients' relatives. There was no difference in age or level of education between the two groups of participants (all p 's > .05). The study was carried out according to the principles laid out in the Helsinki declaration and was approved by the Clinique Sainte Anne's ethics committee. The demographic and clinical characteristics of IPD patients and healthy controls are presented in Table 1.

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