Understanding facial emotion perception in Parkinson's disease: The role of configural processing

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ABSTRACT

Parkinson's disease (PD) has been frequently associated with facial emotion recognition impairments, which could adversely affect the social functioning of those patients. Facial emotion recognition requires processing of the spatial relations between facial features, known as the facial configuration. Few studies, however, have investigated this ability in people with PD. We hypothesized that facial emotion recognition impairments in patients with PD could be accounted for by a deficit in configural processing. To assess this hypothesis, three tasks were proposed to 10 patients with PD and 10 healthy controls (HC): (i) a facial emotion recognition task with upright faces, (ii) a similar task with upside-down faces, to explore the face inversion effect, and (iii) a configural task to assess participants' abilities to detect configural modifications made on a horizontal or vertical axis. The results showed that when compared with the HC group, the PD group had impaired facial emotion recognition, in particular for faces expressing anger and fear, and exhibited reduced face inversion effect for these emotions. More importantly, the PD group's performance on the configural task to detect vertical modifications was lower than the HC group's. Taken together, these results suggest the presence of a configural processing alteration in patients with PD, especially for vertical, second-order information. Furthermore, configural performance was positively correlated with emotion recognition for anger, disgust, and fear, suggesting that facial emotion recognition could be related, at least partially, to configural processing.

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1. Introduction

Parkinson's disease (PD) is a neurodegenerative disorder characterized by a loss of dopaminergic neurons in the ventral striatum, subthalamic nucleus and other basal ganglia structures. Although its classical expression is characterized by motor disorders (for a review, see Lang & Lozano, 1998), PD is also frequently associated with cognitive deficits such as memory (for a meta-analysis, see Whittington, Podd, & Kan, 2000), visuo-spatial (Crucian & Okun, 2003), and executive disturbances (e.g., McKinlay, Grace, Dalrymple-Alford, & Roger, 2010). Behavioral and communicative disorders can also complicate the clinical presentation (Schneier et al., 2000). Indeed, impairment of emotional control (Dujardin et al., 2004; Yamamoto, 2001), apathy (Levy & Dubois, 2006; Pluck & Brown, 2002), anxiety disorders and, more rarely, hallucinations or psychosis (for a review see Aarsland, Marsh, & Schrag, 2009) may appear during the course of the disease.

Because of the evidence that the ventral striatum and subthalamic nucleus have connections with other brain regions, including the orbitofrontal cortex, the amygdala and the putamen (see Adolphs, 2002; Fusar-Poli et al., 2009 for reviews), emotional processing has been widely studied in patients with PD over the last two decades. Several studies (see Gray & Tickle-Degnen, 2010 for a meta-analysis) have shown specific impairments in patients' capacity to recognize emotions from facial cues (Clark, Neargarder, & Cronin-Golomb, 2008; Dujardin et al., 2004; Sprengelmeyer et al., 2003), prosodic cues (Pell & Leonard, 2003; Yip, Lee, Ho, Tsang, & Li, 2003) or both (Ariatti, Benuzzi, & Nichelli, 2008; Dara, Monetta, & Pell, 2008).

Given that the ability to interpret other people's emotional states from facial cues plays a crucial role in social behaviors (Darwin, 1965; Carton, Kessler, & Pape, 1999), facial emotional processing impairments could adversely affect the social functioning of patients with PD. Though obtaining a better comprehension of the mechanisms involved in such deficits may pose significant

clinical challenges, some of the uncertainties that remain must be addressed. First, it is not yet clear whether the emotion recognition deficit is specific for disgusted (e.g., Suzuki, Hoshino, Shigemasu, & Kawamura, 2006) or angry expressions (Lawrence, Goerendt, & Brooks, 2007) or whether it generally concerns negative emotions (e.g., Clark et al., 2008; Kan, Kawamura, Hasegawa, Mochizuki, & Nakamura, 2002; see Gray and Tickle-Degnen for a meta-analysis). Second, facial emotion recognition deficit appears not to be related to executive functions such as categorization (Clark et al., 2008) or working memory abilities (see Gray & Tickle-Degnen, 2010). Facial emotion recognition requires, however, the ability to discriminate facial features, and the visuospatial processing impairments reported in PD patients (Levin, Llabre, Reisman, & Weiner, 1991) could at least partially lead to a facial emotion recognition deficit.

Initially, Bruce and Young (1986) model of face recognition posited that processing facial emotion and facial identity were two independent functional components. Arguing against this hypothesis, more findings have actually suggested that identity and emotion recognition interact (see Vuilleumier & Pourtois, 2007 for a review). Both processes could, in fact, share some perceptual encoding mechanisms (Calder, Young, Keane, & Dean, 2000; Calder & Jansen, 2005). Configural processing, by which the brain understands the spatial relations between facial features (Carey & Diamond, 1977; Diamond & Carey, 1986), is crucial for identifying faces. It includes first-order relations (i.e. overall organization of facial features) and second-order relations (i.e., distances between features, e.g., inter-ocular distance; for a review, see Maurer, Le Grand, & Mondloch, 2002). The former refers to holistic information that is the integration of the facial features into a Gestalt, to identify the target as a face, while the latter is more relevant to facial identity recognition (Maurer et al., 2002; Rossion, 2008). Both information are altered when faces are presented upside-down, inducing a recognition decrement (Yin, 1969; for a review, see Rossion, 2008). This so-called 'Face Inversion Effect' (FIE) has been widely explained as a disruption of configural processing, inverted-face being insufficiently processed through its local elements (Freire, Lee, & Symons, 2000; George, Jemel, Fiori, Chaby, & Renault, 2005; Goffaux & Rossion, 2007; Leder & Bruce, 2000; see also Rossion, 2008). When individuals show a defect of processing configural information, for example in acquired prosopagnosia, upright recognition or discrimination of faces differing by second-order relations are decreased, while performance are less or not at all affected by inversion (Barton, Press, Keenan, & O'Connor, 2002). In such a case, the FIE is reduced or even suppressed.

Yet, a similar FIE has been reported using facial emotion recognition tasks (Calder & Jansen, 2005; McKelvie, 1995; Prkackin, 2003). Turning pictures of facial expressions upside-down has been found to impair recognition performance (McKelvie, 1995), especially for angry, disgusted and fearful faces (Prkackin, 2003). These results suggest that (i) configural processing is also used to encode facial expressions and (ii) expressions of anger, disgust, and fear more heavily tax a person's configural processing resources. Thus, as was suggested many years ago (Ekman, Friesen, & Ellsworth, 1972; McKelvie, 1973), facial emotion recognition requires configural processing rather than a simple inspection of various facial features. For example, in an expression of anger, the shape and position of the mouth may be coded relative to the shape and position of other features (e.g., the eyebrows, Calder et al., 2000). Arguing for this hypothesis, functional neuroimaging studies have shown that fusiform gyri, involved in the structural encoding of faces, are activated when facial expressions are presented (Vuilleumier & Pourtois, 2007). In addition, although dissociations exist, face recognition impairments and facial emotion recognition deficits have been found to correlate in various diseases, such as bilateral amygdala lesions (Young, Hellawell, Van de Wal, & Johnson, 1996),

autism (see Behrmann, Thomas, & Humphreys, 2006 for a review) and schizophrenia (Chambon, Baudouin, & Franck, 2006).

Few studies have investigated facial processing skills in PD patients. Using various perception tasks (unfamiliar matching task, perception of age, gender or gaze direction), previous studies have shown a relatively spared facial identity processing in PD patients (Clark et al., 2008; Lawrence et al., 2007; Sprengelmeyer et al., 2003, see also Gray & Tickle-Degnen, 2010 for a meta-analysis). In contrast, some reports argue that PD patients demonstrate a general impairment in their ability to recognize or perceive faces (Beatty et al., 1989; Dewick, Hanley, Davies, Playfer, & Turnbull, 1991; Haeske-Dewick, 1996; Levin et al., 1991), especially when tasks used more directly involved configural processing (Cousins, Hanley, Davies, Turnbull, & Playfer, 2000). For example, configural processing as assessed by identification of degraded faces was specifically disrupted in PD patients, while part-based object processing was intact (Cousins et al., 2000). Thus, PD could be associated with more basic defects in the structural encoding of faces, and more specifically in configural processing (Beatty et al., 1989; Cousins et al., 2000; Dewick et al., 1991; Haeske-Dewick, 1996). Some evidence have been provided supporting this hypothesis: (1) visuospatial processing impairments are common in PD (Crucian & Okun, 2003; Crucian et al., 2010; Ey et al., 2005); (2) the global processing may be altered in PD, even whether it seems dependent of the body side of motor onset (Schendan, Amick, & Cronin-Golomb, 2009; see also Cronin-Golomb, 2010); (3) a configural processing impairment has been reported in PD while viewing degraded neutral faces (Cousins et al., 2000); and (4) basal ganglia are connected to fusiform regions (Geday, Ostergaard, & Gjedde, 2006) which are involved in face processing (Kanwisher, McDermott, & Chun, 1997). Moreover, configural processing seems crucial for facial emotion recognition (Calder & Jansen, 2005; Calder et al., 2000; McKelvie, 1995; Prkackin, 2003). Thus, we hypothesized that the deficit in facial emotion recognition in patients with PD could be accounted for by a deficit in their configural processing abilities. Nevertheless, although recent studies on the emotional processing deficits in PD patients do exist, to the best of our knowledge, no study has investigated the structural encoding abilities of PD patients by directly manipulating facial configuration yet (e.g. by changing distances between facial features), in relation to emotion processing.

To assess this hypothesis, three tasks were proposed: (i) a facial emotion recognition task, (ii) an upside-down facial emotion recognition task to assess the face inversion effect, and (iii) a configural task to assess the processing of second-order information in neutral faces. We hypothesized that performance on the facial emotion identification task would be lower for the PD group than for the healthy controls, especially for negative emotions, and that the PD group would show a reduced classic FIE for emotion recognition. Whether configural processing is impaired, detection of feature displacements should also be more difficult for patients than for healthy controls. Finally, whether facial emotion recognition impairment in PD is linked to changes in configural processing, performance on these two tasks should be correlated.

2. Method

2.1. Participants

Twelve patients suffering from idiopathic Parkinson's disease (PD) took part in the study. All patients met the clinical criteria of the United Kingdom Parkinson's Disease Society Brain Bank for Idiopathic PD (Hughes, Daniels, Kilford, & Lees, 1992) and received their diagnosis from a movement disorder specialist (AM.B) in the Pitié-Salpêtrière Hospital (Paris, France). The severity of the disease in each participant was rated using the Hoehn and Yahr (1967) scale. All of the participants were within the stage I–stage III (mild unilateral to moderate bilateral disability) at the time of testing. Nine patients had right body side onset of motor symptoms and three had left side onset. All of the patients were undergoing dopamine replacement therapy and were tested while being administered their anti-parkinsonian medication (i.e.,