ELSEVIER

Neuropsychologia 42 (2004) 239–250

NEUROPSYCHOLOGIA

www.elsevier.com/locate/neuropsychologia

Deficits in decoding emotional facial expressions in Parkinson's disease

Kathy Dujardin [a,b,*], Sylvie Blairy [c], Luc Defebvre [a], Stéphane Duhem [b], Yvonnick Noël [b], Ursula Hess [d], Alain Destée [a]

[a] *Neurology A Department, Neurology and Movement Disorders Unit, Salengro Hospital, EA 2683, Lille University Hospital, F-59037 Lille Cedex, France*

[b] *Psychology Department, Charles De Gaulle University, Lille, France*

[c] *Psychiatry Department, Brugmann University Hospital, Brussels, Belgium*

[d] *Psychology Department, University of Quebec at Montreal, Montreal, Que., Canada*

Received 18 October 2002; received in revised form 6 June 2003; accepted 23 June 2003

Abstract

Introduction: The basal ganglia have numerous connections not only with the motor cortex but also with the prefrontal and limbic cortical areas. Therefore, basal ganglia lesions can disturb motor function but also cognitive function and emotion processing. The aim of the present study was to assess the consequences of Parkinson's disease (PD) on ability to decode emotional facial expressions (EFEs)—a method commonly used to investigate non-verbal emotion processing.

Methods: Eighteen PD patients participated in the study, together with 18 healthy subjects strictly matched with respect to age, education and sex. The patients were early in the course of the disease and had not yet received any antiparkinsonian treatment. Decoding of EFEs was assessed using a standardized, quantitative task where the expressions were of moderate intensity, i.e. quite similar to those experienced in everyday life. A set of tests also assessed executive function. Visuospatial perception, depression and anxiety were measured.

Results: Early in the course of the disease, untreated PD patients were significantly impaired in decoding EFEs, as well as in executive function. The deficits were significantly interrelated, although neither was significantly related to severity of the motor symptoms. Visuospatial perception was not impaired, and the patients' impairment was related neither to their depression nor to their anxiety score. The PD patients' impairment in decoding EFEs was related to a systematic response bias.

Conclusion: Early in the course of PD, non-verbal emotional information processing is disturbed. This suggests that in PD, nigrostriatal dopaminergic depletion leads not only to motor and cognitive disturbances but also to emotional information processing deficits. The observed correlation pattern does not enable adoption of a clear-cut position in the debate over totally or partially segregated functional organization of the basal ganglia circuits.

Keywords: Basal ganglia; Emotion; Executive function; Limbic system

1. Introduction

In Parkinson's disease (PD), the dramatic loss of dopaminergic neurons in the substantia nigra pars compacta leads to dysfunction of the striatal structures innervated by those neurons (Parent, 1990). Even though motor symptoms dominate the clinical presentation of PD (Hoehn Yahr, 1967), several studies have shown that PD is associated with cognitive deficits which on the whole can be characterized as constituting a subcorticofrontal syndrome, since PD patients' cognitive impairments mainly concern tasks involving executive abilities (i.e. functions involved in the planning, shifting or sequencing of actions) (Dubois et al., 1994; Pillon, Dubois, & Agid, 1996). Only a small proportion of patients meets the criteria of dementia during the course of the disease, and this generally occurs late on (Hughes et al., 2000).

It has also been demonstrated that in addition to mood disorders (principally depression), a large proportion of PD patients suffers from anxiety disorders (Marsh, 2000).

The fact that motor symptoms are not the only symptoms observed in PD can be easily explained: indeed, anatomical and functional studies have demonstrated that the basal ganglia can be described as a group of "input structures" (the neostriatum and the ventral striatum, which receive direct input from the cerebral cortex) and "output structures" (the internal segment of the pallidum and the substantia

* Corresponding author. Tel.: +33-320-446-752; fax: +33-320-446-680.
E-mail address: dujardin@univ-lille3.fr (K. Dujardin).

0028-3932/$ – see front matter © 2003 Elsevier Ltd. All rights reserved.
doi:10.1016/S0028-3932(03)00154-4

nigra pars reticulata, projecting back to the cerebral cortex via the thalamus). As initially proposed by Alexander, De Long, & Strick (1986), the basal ganglia are classically viewed as participating in various functional loops with the cerebral cortex. At present, the most generally adopted view is that of Parent and Hazrati (Parent & Hazrati, 1995), who considered that motor, associative and limbic cortical areas project in a segregated manner onto three distinct, striatal subregions referred to as motor, associative and limbic striatal territories. The motor striatum (dorsolateral putamen and dorsolateral region of the caudate) is innervated by the primary motor cortex and the supplementary motor area. The associative striatum (consisting of most of the head, body and tail of the caudate nucleus, and significant parts of the putamen rostral to the anterior commissure) receives input from associative areas of the cortex—mainly the prefrontal cortex's 8, 9, 10 and 46 areas. The limbic striatum (nucleus accumbens and the most ventral parts of both the putamen and caudate nucleus) receives input from limbic structures: the hippocampus and amygdala and the prefrontal areas involved in limbic and autonomic functions, i.e. the orbitofrontal, infralimbic and prelimbic cortices (Joel & Weiner, 1994; Parent & Hazrati, 1995). It has been shown that this tripartite principle of organization is maintained at the pallidal and subthalamic levels (Joel & Weiner, 1997; Parent & Hazrati, 1995). The absolute segregation of these circuits is currently the object of wide debate, and some recent models suggest that a certain degree of interaction between the circuits is essential for producing coherent behaviour, as well as for understanding the variety of symptoms associated with basal ganglia dysfunction (for a review, see Joel & Weiner, 2000).

Few studies have addressed the issue of PD-related emotional information processing deficits (Adolphs, Schul, & Tranel, 1998; Breitenstein, van Lancker, Daum, & Waters, 2001; Jacobs, Shuren, Bowers, & Heilman, 1995a; Sprengelmeyer et al., 2003). One procedure commonly used to assess the ability to process emotional information is the recognition of emotions portrayed by a facial expression. Deficits in this ability have been observed not only after focal lesions of the basal ganglia but also in basal ganglia dysfunction related to neurodegenerative diseases such as Huntington's (Jacobs, Shuren, & Heilman, 1995b; Sprengelmeyer et al., 1997) and Parkinson's diseases (Blonder, Gur, & Gur, 1989; Jacobs et al., 1995a; Sprengelmeyer et al., 2003). For example, Jacobs et al. (1995a) compared 12 PD patients to 30 healthy controls, and observed that although the PD patients showed impaired performance of a task assessing emotional facial imagery, they showed normal levels of performance when object imagery was examined. The PD group was also impaired with respect to tasks probing emotion expression and the perception of emotional faces. Recently, Sprengelmeyer et al. (2003) showed that compared to healthy controls, untreated PD patients early in the course of the disease were impaired in identifying emotion in facial expressions. This deficit was not observed in a group of treated PD patients, despite a greater severity of the disease. The main difference between the patient groups concerned disgust, the identification of which was significantly more impaired in the untreated group. However, Adolphs et al. (1998) pointed out that the results of the previous studies should be interpreted with caution since they used tasks that (for example) required participants to match facial expressions with verbal descriptions or tones of voice, and thus did not provide a valid measure of emotional retrieval. Hence, Adolphs et al. (1998) were the first researchers to expose PD patients to an emotional facial expression (EFE) recognition task which provided a more precise assessment of the participants' abilities to recognize emotion than a simple, face label matching task. Specifically, participants were asked to judge each EFE on a series of five point scales (from 0 = not at all to 5 = very much so) labelled as "happy", "sad", "disgusted", "angry", "afraid" and "surprised". This type of task enabled judgement of related emotions that may be portrayed by the facial expression at the same time as the target emotion. The authors used EFEs identified by normal individuals at success rates of over 80% (Ekman & Friesen, 1976). When comparing the performance of 18 PD patients to that of 13 healthy controls, Adolphs et al. did not find any significant group differences and concluded that the basal ganglia structures damaged in PD cannot be considered to be critical components of the neural systems involved in EFE decoding. However, a lack of significant difference has to be interpreted very cautiously. Indeed, with such a small sample size (subjects <20 per group), the lack of a significant result may result from inadequate power: as pointed out by Stevens (Stevens, 1990), the danger of low power studies is that they may stifle or cut off further research in an area where effects do exist but are perhaps subtle. Moreover, the control group and patient group sample sizes used by Adolphs et al. were different, leading to an unbalanced design which could also have reduced the likelihood of detecting group effects. Furthermore, as Adolphs et al. (1998) used facial expressions (from Ekman & Friesen, 1976 series) that are correctly identified by a large proportion of individuals (i.e. with a success rate of over 80%), it may well be that the emotions portrayed by the facial expressions in question are not sufficiently ambiguous to enable detection of differences between the judgements of PD patients and normal controls. Indeed, it is important to note that everyday life, EFEs are more spontaneous and less prototypical than the posed expressions usually employed in experiments (Motley & Camden, 1988). Finally, Adolphs et al. studied PD patients receiving dopaminergic medication, and even though they found no evidence to suggest that their results could be explained by a medication effect, they did underline the fact that medication effects cannot be controlled in most studies, especially as "*a large range of dosages and of different drugs are typically presented in the PD sample of any study*". Consequently, the possibility that the dopaminergic treatment might attenuate the impairment in EFE decoding