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Deficits in decoding emotional facial expressions in Parkinson's disease

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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

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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

due to striatal dysfunction cannot be excluded, and it therefore seems important to control for the effects of medication.

The aim of the present study was thus to investigate the possible involvement of striatal structures in processing non-verbal, emotional information, by studying PD patients' ability to decode EFEs using ambiguous and difficult-to-decode expressions. Indeed, Hess, Blairy, & Kleck (1997) have shown that for all emotions except happiness, decoding accuracy varies greatly with the physical intensity of the expression. That is to say, decoding of an emotion is more accurate when the expressions are high-intensity, full-blown EFEs. Consequently, in the present study, participants were exposed to a series of moderately intense EFEs created and validated by Hess & Blairy (1995).

The PD patients were studied early in the course of the disease and prior to initiation of treatment with medication. Hence, at this stage of the disease, basal ganglia dysfunction is limited to striatal structures and mainly concerns the dopaminergic system. In light of recent approaches to the functional organization of the basal ganglia, the second aim was to investigate the possible interdependence of emotional and cognitive information processing disturbances in PD.

2. Methods

2.1. Subjects

Eighteen non-demented patients (12 men & 6 women) participated in the study: they were all early in the course of PD and had not yet received any medication. PD was defined according to the criteria of the United Kingdom Parkinson's Disease Brain Bank (Gibb & Lees, 1988). Motor symptom severity was assessed according to the Unified Parkinson's Disease Rating Scale (UPDRS) (Fahn et al., 1987) part III motor score (out of 108) in the off-drug condition. Their mean (S.D.) UPDRS part III motor score was 17.58 (6.16) [range: 8–28]. The mean (S.D.) disease duration was 11.33 (4.77) months [range: 6–24].

A group of 18 healthy control subjects (12 men & 6 women), chosen to match the patients group as closely as possible with respect to sex, age and education, also participated in the study. They had no history of neurological or psychiatric illness, and their family history was negative for PD or parkinsonian symptoms.

No participant received drugs which could potentially interfere with cognitive function. Participants were required to show a Mini Mental State score higher than 27 and a Mattis Dementia Rating Scale score higher than the lowest quintile of the reference population (Schmidt et al., 1994).

The characteristics of the patients and control subjects are shown in Table 1.

The groups did not differ with respect to age at testing ($t_{(34)} = 0.20$, $P = 0.841$) and duration of education ($t_{(34)} = 0.82$, $P = 0.417$).

Table 1
Demographical characteristics of the participant groups

Group	Number	Mean (S.D.) age (years)	Mean (S.D.) education duration (years)
PD patients	18	60.17 (10.31)	11.56 (7.79)
Healthy controls	18	59.50 (9.51)	12.28 (6.10)

2.2. Tasks

2.2.1. Emotional facial expression decoding

A series of emotional facial expressions constructed by Hess & Blairy (1995)[1] was used. Specifically, these authors selected facial expressions corresponding to happiness, anger, sadness, disgust and fear from a series of standardized EFEs produced by two male and two female Caucasian actors (JACFEE) (Matsumoto & Ekman, 1988). Based on the neutral face (0% emotional intensity) and the full-blown EFE (100% emotional intensity) from the same actor and using the Morph 1.0 computer program, the authors constructed a series of intermediate expressions differing in emotional intensity by steps of 10%. In the present study, a matrix of 2 (intensity: 30 and 70%) × 3 (emotions: anger, disgust and sadness) × 2 (sex: one male and one female actor) stimuli (i.e. 12 EFEs in total) constituted the material. These stimuli were presented in random order on a Apple Macintosh LCII computer screen. We selected a smaller subset of stimuli than those previously used in research into EFE judgement by clinical populations (e.g. Kornreich et al., 2001; Philippot et al., 1999), in order to reduce the patients' fatigability: under our original experimental conditions, patients were asked to decode a series of 16 expressions: 2 (intensity: 30 and 70%) × 4 (emotions: anger, disgust, sadness, and fear) × 2 (sex: one male and one female actor). Happiness expressions had already been excluded, since even at the very low level of intensity of 20%, they are recognized with a success rate close to 100% (Hess et al., 1997). Nevertheless, PD patients always reported fatigability, and so we chose to discard fear expressions, since they are less frequently displayed during everyday, social interactions.

The participants' task was to rate the emotion portrayed by each face and to quantify its intensity. In order to achieve this, they had to rate each expression on seven-point scales for each of seven basic emotions: happiness, sadness, fear, anger, disgust, surprise and shame.[2] These scales were presented in random order on the computer screen, below the

[1] More information on these stimuli are available in Hess et al. (1997) and in Blairy et al. (1999). Full technical details of the procedure for creating stimuli are available from Ursula Hess.

[2] In order to reduce the likelihood of correct answers being recorded by chance, decoders were provided with seven emotion scales—even though only three types of emotion were employed as stimuli. These particular emotions were chosen because they are defined as "basic" or "fundamental" emotions by a majority of theorists (for more information on the topic of "basic emotion", see Stein & Oatley (1992) or Kirouac (1995), for example).

facial expression, 3 s after each face was first displayed. The face was maintained on the screen until all the scales had been answered. After completion of the emotion scales, participants were also required to rate the task difficulty (i.e. how difficult they found it to deduce the emotion portrayed by that specific facial expression). All the scales were anchored by "not at all" at one extremity and "very intensely" at the other. There was a 2 s inter-trial time. Decoding accuracy was defined as the participant's ability to correctly infer the posed emotion. An expression was considered to be accurately identified if the emotion scale receiving the highest intensity rating on the emotion profile corresponded to the target emotion. Accurately identified or misidentified expressions were scored by 1 and 0, respectively. Participants' performance was expressed as the percentage of accurately-identified expressions.

To enable familiarization with the procedure and use of the computer, participants completed two practice trials with the experimenter prior to completion of the procedure individually.

2.3. Executive function

During the same session, participants also completed a set of tasks assessing executive function.

2.3.1. Word fluency tasks

Three different word generation tasks were administered. In the phonemic task, participants were instructed to name as many words beginning with the letter 'P' as they could in 1 min. In the semantic task, participants were instructed to name as many animal nouns as they could in 1 min. In the alternating task, they were instructed to alternatively name a word beginning with the letter 'T' and a word beginning with the letter 'V'. For all tasks, people's names and proper nouns were not allowed, as well as words from the same root.

Scores for each task were analysed separately. Performance was assessed by the number of different words named in 1 min.

2.3.2. The Stroop word-colour test (Stroop, 1935)

A 50-item version of the test was applied, and comprised two trials. In the first phase (the simple condition), a 21 cm × 29.7 cm (A4) sheet of paper with 50 strings of five dots was presented to participants. Each string was randomly printed in one of three colours (red, blue, green). Participants were instructed to name the colour of each string of dots as quickly as possible without error. In the second phase (the interference condition), a 21 cm × 29.7 cm (A4) sheet of paper with 50 colour names printed in a colour different from the word itself was presented. Three colour names were used to construct the list (red, blue, green). Each word was pseudorandomly printed in one of these three colours. The only restriction was that the name had to be different from the ink in which it was printed. Participants were instructed to name the colour of the ink of each word as quickly as possible and without error.

Performance was assessed by the time in seconds needed to complete each phase, the number of errors in the interference condition and an 'interference cost' index (the difference between the time needed to complete the interference condition and the time needed to complete the simple condition).

2.3.3. Letter and number sequencing task (Dujardin, Krystkowiak, Defebvre, Blond, & Destée, 2000)

In this task, participants were first instructed to recite the alphabet. Next, they were instructed to count forward from 1 to 26. In a third phase, they were instructed to alternatively give a letter and a number in the right order, beginning with "A" as the first letter and "1" as the first number until 26 items were produced, i.e. an ideal sequence of 'A, 1, B, 2, . . . , M, 13'. The time to complete each phase was measured.

Performance was evaluated by the number of alternation errors in the third phase and by an alternation cost index corresponding to the time increase due to alternation: TB–TA, where TA is the mean time of the first two phases ((alphabet + simple counting)/2) and TB is the time of the third phase.

2.3.4. Crossed tapping test (Godefroy et al., 1992)

Participants were given a stick and were instructed to listen to a sound recording. When they heard a single, brief sound, they had to tap twice on the table with the stick. To be sure that the instruction was understood, five practice trials were run. Participants were then instructed that when they heard two, consecutive, brief sounds, they had to tap once on the table with the stick. Five practice trials were run. Thereafter, both kinds of sounds were mixed. Ten practice trials were run before starting the actual task, which comprised 40 trials.

Performance was assessed by the number of errors.

2.3.5. Backward digit span

The WAIS-R backward digit span subtest was administered.

Performance was assessed by the subtest score (marks out of 14).

2.4. Control measures

Patients and control subjects were also assessed with respect to several control parameters, in order to prevent or minimize the influence of variables such as depression, anxiety or visuospatial perception deficits. Depressive mood was assessed using the short version of the Beck Depression Inventory (Beck, Steer, & Garbin, 1988; Bouvard & Cottraux, 1996) and anxiety by the AMDP-AT anxiety scale (Bobon, von Frenckell, Troisfontaines, Mormont, & Pellet, 1985). A Pillon's 15 object test (Pillon et al., 1989) was also administered as a measurement of visuospatial perception.

No non-emotional face recognition test was included because deficits in this ability are very uncommon in early PD (Sprengelmeyer et al., 2003; Taylor, Saint-Cyr, & Lang, 1986) and are usually related to cortical lesions as in Alzheimer or Lewy bodies dementia (Gnanalingham, Byrne, Thornton, Sambrook, & Bannister, 1997; Mori et al., 2000); yet, each of our patients had a magnetic resonance examination in order to discard the presence of such lesions.

2.5. Data analysis

Univariate (ANOVA) and, when appropriate, multivariate (MANOVA) analyses of variance were used to test for the effect of group on the whole set of dependent measures. A 5% significance level was adopted.

Regression analysis was carried out to investigate the possible interdependence of emotional and cognitive information processing disturbances in PD, and Student's *t*-tests were performed to test for significant effects of group on control measures. Spearman correlation coefficients were also calculated, in order to investigate the relation between depression, anxiety, visuospatial perception and EFE decoding accuracy. The significance level was set at 0.05 for all parameters.

3. Results

Since only main effects or interactions involving group are of interest in the context of the present work, our presentation and discussion will thus be restricted to these aspects. None of the control measures were significantly correlated with the cognitive and emotional parameters, and hence their effect was not checked (for example by covariance analysis) in the subsequent analyses. Likewise, no significant main effect or interaction involving the participants' sex emerged, and thus all subsequent analyses were collapsed across this factor.

3.1. Decoding accuracy

Mean (S.D.) overall decoding accuracy and the decoding accuracy in function of emotion and intensity level are presented in Table 2.

Table 2
Accuracy scores (mean (S.D.)) according to emotion type and intensity level

Decoding accuracy (%)	PD patients	Healthy controls
Global score	24 (3)	48 (2)
Disgust (30)	0	14 (5)
Disgust (70)	47 (10)	75 (10)
Sadness (30)	22 (15)	50 (12)
Sadness (70)	39 (13)	56 (17)
Anger (30)	0	25 (13)
Anger (70)	36 (17)	67 (18)

Table 3
F-values for the MANOVA on EFE decoding accuracy scores

Sources	d.f.	*F*-values	Power
Group	1.34	18.42***	0.993
Emotion	2.68	1.75	0.343
Intensity	1.34	52.86***	1
Emotion × group	2.68	0.26	0.083
Intensity × group	1.34	0.08	0.059
Emotion × intensity	2.68	11.54***	0.996
Group × emotion × intensity	2.68	0.98	0.206

*** $P < 0.001$.

In order to assess whether PD patients were impaired in decoding EFEs, a MANOVA was carried out on the decoding accuracy scores with emotion (anger, sadness, and disgust) and intensity level (30 and 70%) as within-subject factors, and group (PD patients and healthy controls) as a between-subjects factor. The results of this analysis are presented in Table 3 and reveal significantly lower decoding accuracy scores in PD patients when compared to healthy controls ($F_{(1,34)} = 18.42$, $P < 0.001$). There was no significant interaction between group and the other factors.

3.2. Intensity scores

A MANOVA with emotion (anger, sadness, and disgust), intensity (30 and 70%) and scales (happiness, sadness, fear, anger, disgust, surprise and shame) as within-subject factors and group (PD patients and healthy controls) as between-subjects factor was carried out on the intensity scores, in order to investigate other qualitative and quantitative differences in the judgement of emotional facial expressions. This analysis (whose results are presented in Table 4) revealed a significant group × scales interaction ($F_{(6,204)} = 5.67$, $P < .001$) qualified by a significant "group × scale × emotion type" interaction ($F_{(12,408)} = 4.542$, $P < .001$), as shown in Fig. 1.

Table 4
F-values for the MANOVA on intensity judgement scores

Sources	d.f.	*F*-values	Power
Group	1.34	0.01	0.051
Emotion	2.68	12.99***	0.999
Intensity	1.34	42.03***	1
Scale	6.204	40.90***	1
Group × emotion	2.68	1.17	0.239
Group × intensity	1.34	2.77	0.35
Group × scale	6.204	5.67***	0.998
Emotion × intensity	2.68	0.39	0.108
Emotion × scale	12.408	33.47***	1
Intensity × scale	6.204	38.36***	1
Group × emotion × intensity	2.68	0.37	0.105
Group × emotion × scale	12.408	4.54***	1
Emotion × intensity × scale	12.408	12.96***	1
Group × emotion × intensity × scale	12.408	1.27	0.72

*** $P < 0.001$.

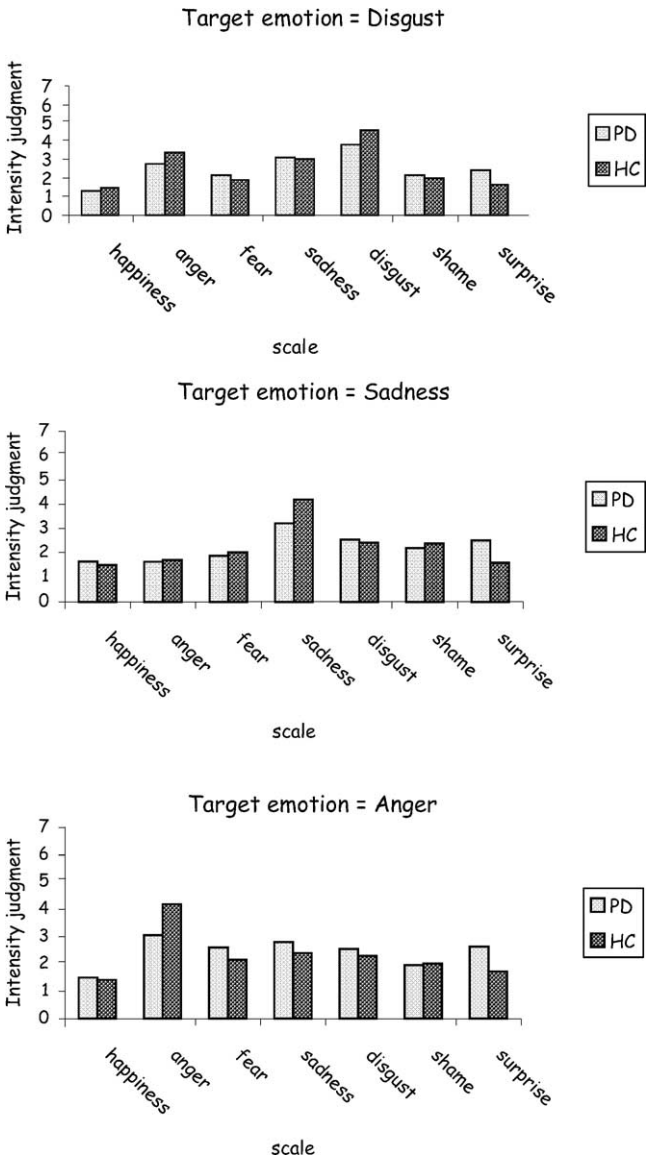

Fig. 1. Group × emotion × scale interaction with intensity judgement.

For disgust expressions, post-hoc analyses revealed a significant group effect on the surprise scale ($F_{(1,34)} = 6.154$, $P = .009$), related to the fact that the PD patients scored higher on this scale than the healthy controls (mean (S.D.) = 2.41 (1.00) and 1.65 (0.83), respectively). A group effect also emerged on the disgust scale ($F_{(1,34)} = 5.837$, $P = .011$), since the PD patients scored lower on this scale than the controls (mean (S.D.) = 3.76 (1.10) and 4.54 (0.80), respectively). No other significant group effects were detected.

For sadness expressions, post-hoc analyses revealed a significant group effect on the surprise scale ($F_{(1,34)} = 8.261$, $P = 003$), again due to the fact that the PD patients scored higher on this scale than the healthy controls (mean (S.D.) = 2.5 (1.21) and 1.58 (0.80), respectively). A group effect also emerged on the sadness scale ($F_{(1,34)} = 6.554$, $P = 007$)—our PD patients scored lower on this scale than the controls (mean (S.D.) = 3.20 (1.01) and 4.18 (1.25), respectively). No other significant group effects were detected.

Table 5
F-values for the MANOVA on task difficulty rating scores

Sources	d.f.	*F*-values	Power
Group	1.34	1.17	0.173
Emotion	2.68	1.47	0.292
Intensity	1.34	7.62***	0.774
Emotion × group	2.68	0.35	0.102
Intensity × group	1.34	4.23*	0.504
Emotion × intensity	2.68	3.71*	0.659
Group × emotion × intensity	2.68	0.76	0.168

* $P < 0.05$.
*** $P < 0.001$.

For anger expressions, post-hoc analyses again showed a significant group effect on the surprise scale ($F_{(1,34)} = 9.317$, $P = 002$), since the PD patients scored higher on this scale than the healthy controls (mean (S.D.) = 2.62, (0.95) and 1.72 (0.81), respectively). A group effect also emerged on the anger scale ($F_{(1,34)} = 8.604$, $P = 003$) with PD patients scoring lower on this scale than the healthy controls (mean (S.D.) = 3.04 (1.02) and 4.18 (1.28), respectively). No other significant group effects were noted.

In summary, for each EFE judged, the PD patients rated the target emotion lower than the healthy controls, while evaluating the expressed surprise more highly.

3.3. Difficulty ratings

As an impairment in decoding EFEs can be related to the perceived task difficulty, a MANOVA with target emotion (anger, sadness, and disgust) and intensity (30 and 70%) as within-subject factors and group (PD patients and healthy controls) as a between-subjects factor was carried out on the difficulty rating scores. The results presented in Table 5 revealed a significant group × intensity interaction ($F_{(1,68)} = 4.22$, $P = 0.047$), which is illustrated in Table 6.

Post-hoc analyses showed that healthy controls rated 70% intensity EFEs as easier to decode than 30% intensity EFEs ($t_{(17)} = 3.07$, $P = .006$) while no differences emerged for the PD patients ($t_{(17)} = 0.32$, n.s.). This suggests that in contrast to healthy controls, the PD patients were not aware of how easy it is to decode 70% intensity EFEs.

3.4. Executive function

Mean (S.D.) results for each executive function task are presented in Table 7.

Table 6
Participant ratings of the difficulty of the EFE decoding task (mean (S.D.)) as a function of the intensity level

	Intensity level	
	30%	70%
PD patients	3.56 (0.09)	3.45 (0.23)
Healthy controls	3.50 (0.18)	2.70 (0.51)

Table 7
Participant performance scores (mean (S.D.)) for the executive function tasks

	PD patients	Healthy controls	$F_{(1,34)}$
Word fluency			
Phonemic	13.44 (5.10)	18.74 (3.96)	12.10**
Semantic	18.89 (5.19)	23.72 (4.18)	9.46**
Alternating	12.17 (4.85)	17.33 (3.76)	12.75**
Stroop word-colour test			
Time to complete phase 1 (s)	34.50 (8.72)	29.72 (5.62)	3.37*
Time to complete phase 2 (s)	64.67 (18.00)	52.28 (8.92)	3.95*
Errors in phase 2	2.72 (4.01)	0.72 (1.02)	2.05*
Interference cost index	30.17 (12.42)	22.55 (5.05)	1.73
Letter number sequencing task			
Errors	0.72 (1.02)	0.00 (0.00)	9.06**
Alternation cost index	21.61 (12.52)	15.64 (3.90)	0.74
Crossed tapping test			
Errors	2.94 (4.45)	0.28 (0.58)	6.35*
Backward digit span			
Score (14)	6.39 (1.91)	7.39 (1.50)	1.74

* $P < 0.05$.
** $P < 0.01$.

The MANOVA performed on executive function measures using group as a between-subjects factor showed a significant effect of group (Wilks' lambda $= 0.427$, $P < 0.001$). Thus, untreated PD patients early in the course of the disease were significantly impaired in their capacity to decode EFEs and to perform a set of tasks evaluating different aspects of executive function. Post-hoc analyses revealed that PD patients performed less well than healthy controls for all the parameters studied here, with the exception of the Stroop word-colour interference index, the alternation cost index of the letter and number sequencing task and the backward digit span.

3.5. Regression analysis

Regression analysis was carried out to investigate the possible interdependence of emotional and cognitive information processing disturbances in PD. Before pursuing the regression analysis, we first examined the predictors' co-linearity in order to select the most pertinent set from the executive function measures as a whole. Indeed, even though these measures assess different aspects of executive function, they are all clearly related to the concept of "cognitive control", which refers to effortful and intentional processes involved in guiding and coordinating flexible information processing (Gray, 2001). One approach could consist of calculating a mean "z" score to resume performance across all the executive function tasks, as previously reported in other studies (Dibley et al., 1987; Jason et al., 1997; Kiernan & Matthews, 1976). However, an alternative approach consists of performing a principal component regression analysis, i.e. a regression on the principal components extracted from the set of predictors using principal component analysis (PCA). In the present case, PCA showed that all the predictive variables could be summarized by one, principal factor: as can be seen in Table 8, this component accounted for 61% of the total variance of the data.

Table 8
Principal component analysis: proportion of the variance accounted for by the different components

Components	Proportion of variance
1	0.606
2	0.138
3	0.099
4	0.072
5	0.043
6	0.022
7	0.018

The eigenvalue graph represented in Fig. 2 shows that all the other components had an eigenvalue lower than one—suggesting a clear, unifactorial structure.

These results supported our choice of considering only one overall executive score. Based on the PCA's properties, we thus know that amongst all the possible choices of an overall score that can be obtained by linear combination of the standardized predictor variables, the subjects' projections on the first principal component do provide the best fit. This in turn implies than these projections offer the best inter-individual discrimination, and so this measure was thus used in subsequent analyses. Thereafter, the linear regression of EFE decoding accuracy against executive score was calculated. The results of this analysis showed that EFE decoding accuracy was significantly related to the overall executive score ($F_{(1,34)} = 11.95$, $P = 0.0015$). This linear model was further validated by a Shapiro–Wilk test of normality on the residuals distribution ($P = 0.746$), and a Durbin–Watson test of residuals autocorrelation (D–W $=$ 1.76, $P < 0.49$). Introduction of a quadratic term did not significantly improve the model's fit ($P < 0.43$). The regression plot in Fig. 3 shows that the relationship between

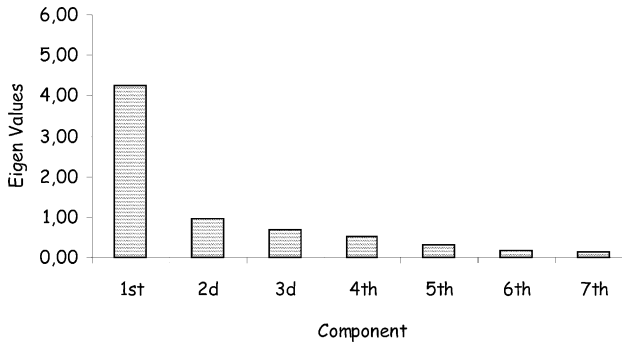

Fig. 2. Principal component analysis: eigenvalues.

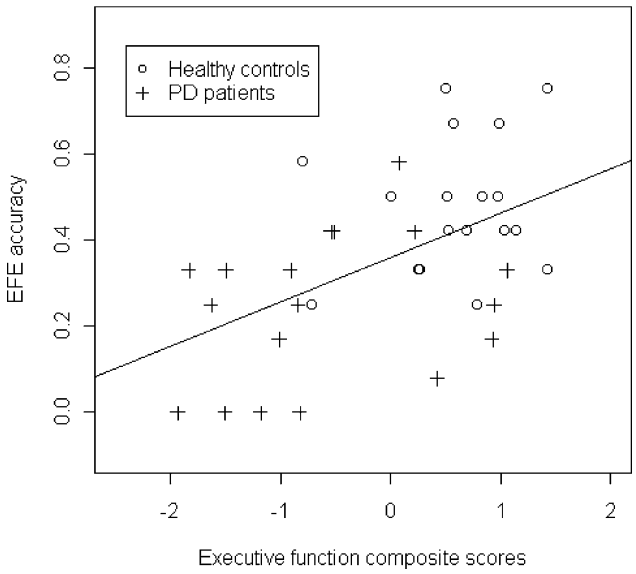

Fig. 3. Regression plot.

executive function and EFE accuracy is well described by a simple, linear model.

In the patient group, correlations between the UPDRS part III motor score and EFE decoding accuracy (as well as the executive score) were also considered. None was significant (UPDRS part III motor score and EFE decoding accuracy: $r = -0.448$, $P = 0.062$; UPDRS part III motor score and executive score: $r = -0.424$, $P = 0.079$).

3.6. Control measures

Mean (S.D.) scores are presented in Table 9.

Student's *t*-tests revealed a significant group effect on the Beck Depression Inventory score ($P = .002$). Although none of the PD patients met the depression criteria, the group's mean depression score was significantly higher than that of the healthy controls. No group effect was observed for the two other control measures. The correlations between the EFE decoding accuracy, depression score, anxiety level and time needed to identify 12 objects in Pillon's 15 object test are presented in Table 10. They were not significant in either group.

Table 9
Participant scores (mean (S.D.)) for the control measures

	PD patients	Healthy controls
Depression		
BDI score (/63)	6.22 (4.45)	2.22 (2.13)*
Anxiety		
AMDP-AT score (/68)	0.92 (0.77)	0.94 (0.56)
Pillon's 15 objects test		
Time (s) to identify 12 objects	39.35 (16.19)	46.83 (10.97)

* Signals significant group effects: $P < 0.01$

Table 10
Correlation values (Spearman ρ) between EFE decoding accuracy and depression (BDI), anxiety (AMDP-AT) and visuospatial perception (Pillon's 15 object test) in both participant groups

	BDI score (/63)	AMDP-AT score (/68)	Pillon's 15-object test
Healthy controls			
Decoding accuracy (%)			
Disgust	0.03	0.28	−0.23
Sadness	−0.15	−0.15	0.14
Anger	−0.26	−0.32	0.35
PD patients			
Decoding accuracy (%)			
Disgust	−0.06	−0.07	−0.004
Sadness	−0.21	0.03	−0.30
Anger	0.00	−0.23	−0.23

4. Discussion

By using a standardized, quantitative task of facial expression recognition, the present results support the notion that non-verbal, emotional information processing is disturbed early in the course of PD. Specifically, the results showed that PD patients were less accurate than healthy controls in decoding angry, sad and disgusted EFEs, regardless of the expression's intensity level (low or mild). Analysis of the response's qualitative aspects also revealed significant group differences with respect to the judgement of intensity. In healthy controls, the scale corresponding to the current emotion always corresponded to the most intense judgement. However, even though PD patients also judged the current emotion with the highest intensity when it corresponded to anger, sadness and disgust, their judgement was always less intense than that of the healthy controls, and the differences with scores on the other emotion scales were not so clear-cut. Furthermore, the PD patients always scored the surprise scale more intensely than the healthy controls, regardless of the expressed emotion. It is possible that the PD patients are more frequently exposed to surprised expressions in their social circle (due to manifestations of the disease symptoms) and so tend to emphasise this emotion in response to non-verbal cues. Further research investigating impairments in EFEs recognition associated with interpersonal relationships could verify such an hypothesis.

The results observed for the self-reported task difficulty mirrored those observed for the decoding accuracy: for healthy controls, the more intense the expressions, the less difficult the decoding task was perceived to be—our results agree with those from previous studies (Hess et al., 1997). In contrast, the PD patients evaluated the more intense expressions as being as difficult-to-decode as the less intense ones. This could therefore explain the low accuracy of the patients' decoding, which applied to even the more intense expressions. Such a result could be expected, since both decoding accuracy and self-reported task difficulty measure essentially the same variable, i.e. decoding difficulty.

Nevertheless, it is interesting to note that the PD patients are aware of their decoding deficits, in contrast to other psychiatric populations suffering from cognitive impairments, for example alcoholics (e.g. Kornreich et al., 2001, 2002).

The overall decline in emotional information processing here observed appears in opposition with a recent study of Lawrence, Calder, McGowan, Grasby (2002) reporting that in healthy males, acute administration of a dopamine antagonist leads to a selective disruption in the recognition of facial expressions of anger with preserved recognition of other emotions. However, the fact that Lawrence et al. (2002) administered a dopamine D2-class receptor antagonist (sulpiride) can explain such a discrepancy. Indeed, in healthy subjects, it is not surprising that the selective disruption of one dopamine receptor family leads to a very selective impairment. Yet, PD not only disrupts the D2 receptor family but also other ones, including the D1–D5 receptors which, as the D2 receptors are localized in the ventral striatum participating in the nigro-striato-prefrontal limbic functional circuit (Missale, Nash, Robinson, Jaber, & Caron, 1998; Smith & Kieval, 2000). This could thus explain the larger impairment we observed.

Despite using very similar methods, our results do not concord with those of Adolphs et al. (1998). As mentioned in our introduction, the lack of significant results in Adolphs et al. (1998) study might be due to inadequate statistical power. Another possible explanation may be related to the type of stimuli used. Indeed, decoding accuracy and difficulty varied greatly with the physical intensity of the expressions used (Hess et al., 1997). In the present study, participants were exposed to EFEs that were probably less intense than those used by Adolphs et al. (1998) and in the majority of studies investigating decoding accuracy. It is thus possible that deficits do indeed appear for low-intensity emotion expressions, in contrast to the situation with more intense, full-blown ones. Further studies using low-intensity EFEs are needed to clarify this point.

Another difference between the studies concerned the characteristics of the PD patients. Adolphs et al. (1998) examined PD patients with quite a broad range of disease severities (Hoehn & Yahr scores ranging 2–5, disease duration ranging from to 2 to 20 years) and a great variety of dopaminergic treatments. Thus, even though Adolphs et al. (1998) considered that there is no evidence from their results of a medication effect, they noted that the use of "*large ranges of dosage and of different drugs*" constituted a methodological limitation in most studies. In the present study, we decided to control this aspect by only including treatment-naive PD patients early in the course of the disease. Consequently, our results do not suffer from a possible drug effect, and the homogeneity of the patients group was rather better (disease duration ranging from 0.5 to 2 years, UPDRS motor score ranging from 8 to 28). The choice of this type of patient group was driven by our wish to determine whether dopaminergic nigrostriatal depletion involves an early disturbance of the limbic part of the nigro-striato-prefontal functional circuits. Indeed, the results of a group of previous studies with which our results concord had suggested an involvement of the basal ganglia—and especially the striatum—in EFE decoding (Blonder et al., 1989; Jacobs et al., 1995a,b; Sprengelmeyer et al., 2003), as well as in production and decoding of emotional prosodies (Benke, Bösch, & Andree, 1998; Caekebeke, Jennekens-Schinkel, van der Linden, Buruma, & Roos, 1991). Since converging evidence has indicated the involvement of several limbic system structures (especially the amygdala (Adolphs, Tranel, Damasio, & Damasio, 1994) and the orbitofrontal cortex (Blair, Morris, Frith, Perrett, & Dolan, 1999; Hornak, Rolls, & Wade, 1996) in processing facial expressions, it thus seems that dysfunction of this system appears very early in the course of PD and is likely to be associated with dopaminergic nigrostriatal depletion. Indeed, as the patients were not medicated, the possible interfering role of dopaminergic medication can here be excluded. A role of the dopaminergic system is also suggested by the recent results of Sprengelmeyer et al. (2003) who monitored face label matching performance in untreated PD patients early in the course of the disease, in treated patients suffering from more advanced PD and in healthy controls. These authors reported that compared to the healthy controls, only the untreated PD patients showed impaired identification of sadness, anger and disgust on morphed facial expressions. The treated PD patients showed no impairment, and the main difference between the early-stage and advanced patient groups concerned disgust and anger. This suggests thus that in PD, nigrostriatal dopaminergic depletion leads not only to motor and cognitive disturbances but also to non-verbal, emotional information processing deficits. The three nigro-striato-prefrontal functional circuits proposed by Parent & Hazrati (1995) could thus be affected very early.

As visuospatial information processing deficits have been described in PD (Doyon, Bourgeois, & Bedard, 1996; Dubois, Boller, Pillon, & Agid, 1991; Finton, Lucas, Graff-Radford, & Uitti, 1998; Ogden, Growdon, & Corkin, 1990), it could thus be suggested that the PD patients' deficit in decoding EFEs is related to a decline in perception. Such a hypothesis can probably be eliminated here, since visuospatial perception abilities were monitored, and no significant, inter-group difference was found. However, as underlined by one of the reviewers of this article, only one task assessing visuospatial abilities was used in our study, and it did not examine the specific ability to recognize faces. Consequently, although face recognition impairment is very uncommon in early PD, the possibility that the emotional information processing deficits reported here are partly due to a deficit in face recognition cannot be completely discounted.

The potential involvement of secondary factors such as anxiety and depression (shown to influence recognition of EFEs, see Moretti, Charlton, & Taylor, 1996) can also be excluded: both groups had comparable anxiety scores, and even though PD patients scored slightly higher than the controls in the Beck Depression Inventory, their score was very

low and none could be suspected of suffering from depression. Moreover, neither score was correlated with the emotion decoding accuracy, regardless of the emotion and the intensity with which it was presented.

The correlation study showed that EFE decoding and executive function are significantly related. This is in agreement with a previous study of Breitenstein et al. (2001) showing that impaired perception of vocal emotions in non-demented PD patients is significantly related to working memory deficits. Nevertheless, a recent study using transcranial Doppler sonography to investigate middle cerebral arteries blood flow velocity changes when viewing emotional slides showed disturbed patterns in PD patients although they performed as well as the controls at a set of cognitive tasks (Troisi et al., 2002). However, the tasks used in the latter study less specifically assessed executive function.

In our PD patients, the severity of motor symptoms was neither significantly related to the EFE decoding accuracy nor to the executive score. Yet, even though they do not reach significance, these latter correlations are rather large and suggest that motor symptoms severity, executive dysfunction and emotional information deficits are probably related in PD. Such a pattern does not enable the adoption of a clear-cut position in the debate over a totally or partially segregated functional organization of the basal ganglia circuits. As the patients were early in the course of the disease and had not received any treatment, it can be suggested that the striatal dopaminergic depletion does lead to all the observed deficits but that the observed correlation pattern is related to the fact that in the disease's initial stages, the striatal territories are differentially affected. Nevertheless, although the raw data show that the patients' dysexecutive syndrome were significant but mild, the EFE decoding deficit was quite important: it thus seems difficult to explain the results by a less severe dysfunction of the associative and limbic territories compared to the motor territory. Another hypothesis is that other neurotransmission systems are involved. A dysfunction of the cholinergic system could contribute to the dysexecutive syndrome, since several studies have demonstrated its involvement in the cognitive decline of PD patients (Bedard, 2001; Bedard et al., 1999). Although noradrenergic and serotoninergic deficits are commonly related to affective and emotional disturbances in movement disorders (Ring & Serra-Mestres, 2002), the neurotransmitters participating in emotional information processing have yet to be identified.

In conclusion, by supporting the notion that not only motor disturbances but also cognitive and limbic dysfunctions may be present very early in the course of PD, this study emphasizes the importance of considering all such dysfunctional aspects when dealing with PD patients. Indeed, since it has been shown that executive function deficits can appear in first degree relatives of patients with familial PD (Dujardin et al., 1999; Holthoff et al., 1994; Vieregge, Hagenah, Heberlein, Klein, & Ludin, 1999) as well as in subjects exposed to 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) (Stern, Tetrud, Martin, Kutner, & Langston, 1990), suggesting that the first symptoms of the disease may be cognitive, the possibility that impairment in non-verbal emotional information processing is the first sign of the disease to appear—i.e. before the motor symptoms—cannot be ruled out. However, the present study used negative emotions only since it has been shown that even at very low levels of intensity, the expression of happiness is recognized with a success rate close to 100% (Hess et al., 1997). Nevertheless, so as to reduce the duration of the experiment, fear expressions were not included in the present study, despite the fact that they have been included in many previous neuropsychological studies (for a review, see Adolphs (Adolphs, 2002)). Moreover, the present study did not explore vocal emotion. These factors therefore constitute a certain limitation, since we cannot exclude the possibility that PD involves changes in the recognition of positive EFEs, of other negative EFEs (such as fear expressions) and vocal emotion. Further studies in PD are necessary to answer such questions and thus to help clarify the striatum's role in emotional information processing.

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