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Advanced Parkinson disease patients have impairment in prosody processing

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ABSTRACT

Background: The ability to recognize and interpret emotions in others is a crucial prerequisite of adequate social behavior. Impairments in emotion processing have been reported from the early stages of Parkinson's disease (PD). This study aims to characterize emotion recognition in advanced Parkinson's disease (APD) candidates for deep-brain stimulation and to compare emotion recognition abilities in visual and auditory domains. **Method:** APD patients, defined as those with levodopa-induced motor complications ($N = 42$), and healthy controls ($N = 43$) matched by gender, age, and educational level, undertook the Comprehensive Affect Testing System (CATS), a battery that evaluates recognition of seven basic emotions (happiness, sadness, anger, fear, surprise, disgust, and neutral) on facial expressions and four emotions on prosody (happiness, sadness, anger, and fear). APD patients were assessed during the "ON" state. Group performance was compared with independent-samples t tests. **Results:** Compared to controls, APD had significantly lower scores on the discrimination and naming of emotions in prosody, and visual discrimination of neutral faces, but no significant differences in visual emotional tasks. **Conclusion:** The contrasting performance in emotional processing between visual and auditory stimuli suggests that APD candidates for surgery have either a selective difficulty in recognizing emotions in prosody or a general defect in prosody processing. Studies investigating early-stage PD, and the effect of subcortical lesions in prosody processing, favor the latter interpretation. Further research is needed to understand these deficits in emotional prosody recognition and their possible contribution to later behavioral or neuropsychiatric manifestations of PD.

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The ability to recognize and interpret emotions in others is a crucial prerequisite for individuals to update and tune their responses to others' emotional demands. Emotional processing involves an extensive neuronal network that includes the prefrontal cortices, insula, and basal ganglia (Vuilleumier & Pourtois, 2007; Wildgruber, Ackermann, Kreifelts, & Ethofer, 2006). The study of patients with primary disorders of the basal ganglia, like Parkinson's disease (PD), has the potential to clarify the role of those subcortical structures in emotional processing and contribute

to the understanding of emotional disturbances and behavioral disorders observed in PD. A significant association between deficit in emotion recognition in faces and some impaired social/behavioral tasks has been described—namely, by assessing empathy and theory of mind (Narme et al., 2013).

Behavioral disturbances have been increasingly recognized in advanced Parkinson disease (APD) and have a variety of manifestations ranging from depression and apathy (Robert et al., 2014; Starkstein, 2012) to dopamine dysregulation

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syndrome and psychosis, which have been largely associated with dopamine mesolimbic denervation (Thobois, Ardouin, & Schmitt, 2010). These disturbances tend to increase with disease severity and are a strong contributor to patients' disability and caregivers' burden in advanced stages of PD and a risk for institutionalization (Aarsland, Marsh, & Schrag, 2009; Coelho et al., 2015; Coelho et al., 2010).

Most studies of emotion recognition in PD have been performed in early-stage PD—that is, within the first five years of the disease—and were often tested in a single sensory modality: visual or auditory. Those studies have produced variable results. While some authors have associated PD with intact recognition of emotion in facial expressions (Adolphs, Scul, & Tranel, 1998; Pell & Leonard 2005), others reported selective deficits in disgust (Assogna et al., 2010; Sprengelmeyer, Young, & Mahn, 2003; Suzuki, Hoshino, Shigemasa, & Kawamura, 2006), fear and disgust (Kan, Kawamura, Hasegawa, Mochizuki, & Nakamura, 2002), fear and sadness (Ariatti, Benuzzi, & Nichelli, 2008), or anger (Lawrence, Goerendt, & Brooks, 2007). Disturbances of emotional prosody processing were also reported (Ariatti et al., 2008; Benke, Bosch, & Andree, 1998; Breitenstein, Van Lancker, Daum, & Waters, 2001; Dara, Monetta, & Pell, 2008; Lloyd, 1999; Pell & Leonard, 2003). Yip described a deficit of emotional recognition across different sensory modalities, which was considered evidence for the convergence of emotional pathways in subcortical structures and/or frontostriatal circuits (Yip, Lee, Ho, Tsang, & Li, 2003). A meta-analysis on the performance of emotion recognition tasks in PD also supported a robust link between this disorder and deficits of recognition, especially negative emotions (emotional faces and voices; Gray & Tickle-Degnen, 2010).

More recent reports on patients with longer PD duration (up to 10 years) showed disturbances in the facial recognition of different negative emotions (Alonso-Recio, Martin, Rubio, & Serrano, 2014; Baggio et al., 2012; Clark, Neargarder, & Cronin-Golomb, 2010; Narme et al., 2013; Saenz et al., 2013). Concerning the recognition of emotional prosody in PD again up to 10 years duration, studies seem to agree on an impairment of emotional prosody recognition in speech or music (Buxton, MacDonald, & Tippett, 2013; Lima, Garrett, & Castro, 2013), although not necessarily associated with congruent similar findings in emotional face recognition (Buxton et al., 2013).

As PD progresses further, pathological changes develop in anatomical structures other than the basal ganglia, such as the limbic system, insula, and frontal lobes (Braak & Del Tredici, 2008; Halliday, Hely, Reid, & Morris, 2008; Harding, Stimson, Henderson, & Halliday, 2002). In the long-term, this could be expected to be associated with more deficits of emotional processing, though possibly less selective of a particular emotion.

A subset of patients in later stages of PD, with severe levodopa-induced motor complications, and usually under 70 years of age, are now successfully treated by deep brain stimulation (DBS). In this group, acquired deficits in emotional recognition have been suggested as a possible consequence of DBS, although not confirmed (Albuquerque et al., 2014). The importance of baseline emotional deficits in the surgical candidates might assume a higher relevance after surgery, once motor factors of functional disability are corrected.

If indeed there is a progressive emotional compromise starting in early-stage PD, a study of a homogeneous group of pre-DBS patients seems relevant for a number of reasons: first because it is likely that impairment in emotional recognition will be more pronounced in later stages of disease; secondly because it may contribute to understanding the baseline emotional profile of DBS candidates; and thirdly because it will contribute to understanding the possible link with neuropsychiatric disturbances and disease evolution.

This study aims to analyze emotion processing in advanced Parkinson's disease candidates for surgery (APD) and, in particular, to characterize emotion recognition in two sensory modalities: visual and auditory.

Method

Study design

A cross-sectional study of APD patients and healthy controls, matched by gender, age, and educational level, was conducted in a tertiary academic hospital.

Participants

Selection, recruitment, and clinical evaluation

APD patients were consecutively recruited among those undertaking cognitive assessment for DBS selection in the Movement Disorders Clinic. All

cases fulfilled clinical diagnostic criteria of PD, according to the UK Brain Bank criteria (Hughes, Daniel, Kilford, & Lees, 1992).

APD subjects included correspond to a subset of PD patients with relatively preserved cognitive function, younger than 70 years of age, with levodopa-induced motor complications refractory to best medical treatment. Patients with a clinical diagnosis of dementia according to the attending neurologists had previously been excluded, as this is one of the exclusion criteria for surgery. Patients diagnosed with dementia (defined according to *Diagnostic and Statistical Manual of Mental Disorders—Fourth Edition, Text Revision, DSM-IV-TR*; American Psychiatric Association, 2000, criteria) after the formal neuropsychological assessment were also excluded from this study.

PD severity was rated by the Unified Parkinson's Disease Rating Scale (UPDRS), which comprises nonmotor symptoms (Part I), activities of daily living (Part II), motor examination (Part III), and treatment complications (Part IV; Fahn & Elton, 1987).

Controls were recruited among healthy familial members or caretakers of patients from the neurology outpatient clinic without previous neurological or psychiatric disorders. All controls undertook the Mini Mental State Examination (MMSE) using Portuguese cutoff values according to literacy (Morgado, Rocha, Maruta, Guerreiro, & Martins, 2010).

Informed written consent was obtained from patients and controls, and the research project was approved by the Institution Ethics Committee.

Emotion recognition tasks

APD and controls were evaluated by the Comprehensive Affect Testing System (CATS), a computerized battery licensed and adapted to the Portuguese population (Fernandes, 2006; Froming & Ekman, 2005). The battery evaluates the ability to recognize six basic emotions on facial expressions (happiness, sadness, anger, fear, surprise, and disgust), as well as neutral faces, and four prosody emotions (happiness, sadness, anger, and fear) or neutral stimuli conveyed by the prosodic intonation of sentences with neutral content. Faces and sentences without emotional content are designated as neutral. Emotion recognition was assessed using different tasks. In “emotional discrimination” tasks, patients were asked to choose whether

emotions of a pair of emotional stimuli (faces or sentences) were the “same” or “different.” In “emotional naming” tasks, patients were asked to listen (prosody) or attend (faces) to emotional stimuli and then to name the emotion presented, by choosing among several possible emotion labels depicted on the computer screen. Answers had no time limits, and correct responses were converted to a percentage of the total. The following subtests were chosen: “discrimination of identity” between two unfamiliar faces (22 pairs of neutral faces), “discrimination of facial emotions” (22 pairs of emotional faces), “naming of single facial emotions” (16 faces—4 neutral and 2 depicting each emotion, in sequence), “discrimination of prosody” between two sentences with neutral content (22 pairs of sentences), “naming of single emotional prosody” (18 sentences with neutral content and different prosodic intonations in sequence—4 happy and sad, 5 angry and fear). The aim of the discrimination of identity test was to control for primary deficits in discrimination of neutral/none-motional faces. The Portuguese adaptation of CATS used the same faces as those in the original battery, and sentences were translated to be read by a native actor. The final version described above retained stimuli scoring more than 50% correct by the healthy population, leading to the exclusion of only four stimuli of the test of prosody naming. APD patients displayed no subjective hearing or visual difficulties while dealing with the stimuli and were evaluated in the presence of one of the authors, in a quiet room isolated from external noises, under the effect of levodopa (in “ON” condition).

Cognitive assessment

Patients were evaluated by a comprehensive battery of cognitive tests: MMSE (Folstein, Folstein, & McHugh, 1975; Guerreiro et al., 1994), Dementia Rating Scale (Mattis, 1988), and several measures of executive function. Tests included working memory (Forward and Backward Digit Span, Wechsler Memory Scale—Third Edition, WMS-III; Guerreiro, 1998; Wechsler, 1987); abstract reasoning (Raven Matrices AB; Raven, Court, & Raven, 1993) and proverb interpretation; inhibitory control (the Stroop color–word test; Stroop, 1935); semantic (food items) and phonemic (letter p) verbal fluency (Martins, Maruta, Freitas, & Mares, 2013); episodic memory (Logical

Table 1. Demographic features.

Statistics	APD (<i>N</i> = 42)		CG (<i>N</i> = 43)		Statistics test (<i>df</i>)	<i>z</i> / χ^2	<i>p</i>
	Mean (<i>SD</i>)	Ratio	Mean (<i>SD</i>)	Ratio			
Age (years)	62.5 (7.0)		61.8 (10.3)		Mann-Whitney <i>U</i> (83)	-0.02	.98
Literacy (years)	7.8 (4.4)		8.8 (5.8)		Mann-Whitney <i>U</i> (83)	-0.26	.80
Gender (female:male)		22:20		25:18	χ^2	0.29	.59
PD duration (years)	14.6 (6.0)		NA		NA		NA
MMSE	27.7 (1.7)		28.5 (1.4)		Mann-Whitney <i>U</i> (76)	-2.3	.02*

Note. PD = Parkinson's disease; APD = advanced Parkinson's disease; CG = control group; NA = not applicable.

**p* < .05.

Memory-immediate recall of the stories from the Wechsler Memory Scale; Guerreiro, 1998; Wechsler, 1987); and visuomotor abilities (clock drawing).

Statistical analysis

A descriptive analysis of demographic and clinical variables was performed. Raw scores in each cognitive test were converted to age- and education-adjusted *z* scores, according to Portuguese norms (Guerreiro, 1998; Martins et al., 2013). Demographic, cognitive, and study tests of the CATS were compared between groups (APD and controls) by chi-square tests (categorical variables), or independent-samples *t* tests or Mann-Whitney *U* tests (continuous measures). Normality was assessed using the Kolmogorov-Smirnov test.

In addition, for emotions presented in both the visual and the auditory tasks (sad, happy, anger, fear, or neutral), we calculated a variable measuring the bias to attribute specific emotions, by dividing the number of times any emotion was chosen with the total number of items in which that emotion was correct. For example, if a participant chose the category "fear" 14 times, and there was a total of 7 "fear" stimuli (5 in the auditory modality and 2 in the visual modality), then this ratio would be $14/7 = 2$. Separate bias scores for each modality were also calculated (adding two other possibilities, disgust and surprise, in the visual presentation). We used this variable to compare APD and controls to investigate whether there was a particular tendency towards the choice of one particular emotion in the APD group. The association between CATS and MMSE and executive test performance (verbal fluencies, digit spans, and Stroop Test) was studied by Spearman correlations.

A significance level of .05 was assumed as statistically significant. Statistical analysis was

performed with SPSS Version 20.0 (IBM, SPSS, Chicago, Illinois).

Results

Forty-two APD and 43 controls were included in the study. Demographic features were similar in the two groups (Table 1). Patients performed cognitive tests within normal limits but, compared to controls, APD had a significantly lower MMSE score (Mann-Whitney *U*), $z(76) = -2.3$, $p = .02$ (Tables 1, 2).

Group performance in the emotion recognition tests is presented in Table 3. For visual stimuli, APD presented significantly worse results than controls in the discrimination of identity (Mann-Whitney *U*), $z(83) = -2.38$, $p = .02$. However, there were no group differences either in the discrimination of emotional faces or in visual emotion naming.

Concerning auditory stimuli, patients had significantly lower scores than healthy controls in all

Table 2. Mean scores of APD in UPDRS I, UPDRS III, and cognitive measures.

Test	Z-score/raw score
UPDRS IIIa	17.27 ± 7.5
UPDRS Ia	2.20 ± 1.7
MMSEa	27.7 ± 1.7
DRSa	131.7 ± 7.8
DS (Forward+Backward): 2	0.42
Phonemic Fluency (p)	-0.03
Logical memory	-0.27
Clock drawing	0.39
Stroop reading	0.73
Stroop color	-0.44
Stroop C-W	-0.35

Note. APD = advanced Parkinson's disease; UPDRS = Unified Parkinson's Disease Rating Scale; UPDRS III = Part III, motor score (normal score 0, maximum pathological score 108; scores during "ON" condition, in 34 patients); UPDRS I = Part I, motivation score (normal 0, maximum 16; in 34 patients); DS (Forward+Backward) = Forward and Backward Digit Span; MMSE = Mini-Mental Status Examination; DRS = Dementia Rating Scale; Stroop C-W = Stroop color-word interference.

^aMean raw score.

Table 3. Comparison of CATS scores: APD versus control groups.

CATS subtest	APD (%)	Controls (%)	Statistics test	<i>t/z</i>	<i>p</i>
Discrimination of Identity	87.01 ± 11.73	92.49 ± 8.81	Mann–Whitney <i>U</i>	–2.38	.02*
Discrimination of Emotional Faces	92.31 ± 7.93	93.23 ± 9.27	Mann–Whitney <i>U</i>	–1.1	.27
Naming of Emotional Faces	63.39 ± 17.49	66.72 ± 15.89	<i>t</i>	–0.92	.36
Discrimination of Prosody	92.21 ± 9.05	96.09 ± 6.47	Mann–Whitney <i>U</i>	–2.44	.015*
Naming of Emotional Prosody	49.99 ± 18.62	60.77 ± 14.52	<i>t</i>	–2.98	.004*

Note. APD = advanced Parkinson's disease; CATS = Comprehensive Affect Testing System.

**p* < .05.

measures: discrimination of emotional prosody (Mann–Whitney *U*), $z(83) = -2.44$, $p = .015$, and naming emotional prosody, $t(83) = -2.98$, $p = .004$.

We tested whether participants had a bias to name particular emotions and found no significant differences between APD and controls. For instance, the answer “fear” was equally frequent in patients and controls. This was true for all emotions within each modality and for each emotion in both modalities (all $p > .05$).

No significant correlations were found between discrimination or naming of emotional prosody and MMSE or executive test scores.

Discussion

This study showed that patients with APD (fulfilling criteria for subthalamic nucleus-DBS therapy) performed significantly worse than healthy controls in discriminating and naming emotional speech prosody, but had a similar performance in visual discrimination and naming of emotional faces. This finding in APD was not explained by cognitive dysfunction.

Few studies performed in less advanced stages of PD tested both faces and speech prosody in the same group of patients (Ariatti et al., 2008; Buxton et al., 2013; Yip et al., 2003). The current study of APD corroborates those studies by reporting deficits in prosody processing, but it disagrees concerning the visual recognition of emotions. Yip et al. (2003) described a combined deficit of emotion recognition in visual (56 patients) and auditory (11 patients) presentations in PD patients with a mean disease duration of 7 years. Ventura et al. (2012) found that facial emotion recognition was preserved in a group of 24 PD; however, when comparing motor asymmetry, PD participants with predominant left limb involvement had specific impairment in prosodic emotion recognition, especially for sadness. In a series of 27 cases reported by Ariatti (Ariatti et al., 2008), with a mean PD duration of 6 years, patients disclosed impaired

recognition in visual emotion only for fear and sadness, in contrast with a diffuse deficit in recognizing emotional and propositional prosody. A diffuse impairment in emotional prosody recognition was also supported by Buxton et al. (2013), who further referred similar deficits in processing subtle facial expressions of happiness. Taken together, these results all seem to imply that deficits in the prosody domain are robust and general. Conversely, findings reporting deficits in the visual domain are often specific to certain emotions, are variable across studies, and seem to depend on the kind of analysis performed and on the population tested.

Our results may have two possible interpretations. Either APD patients have a general deficit in prosody processing (emotional and nonemotional) or there is a selective dysfunction in auditory emotional processing networks. The first hypothesis is favored by the fact that impaired emotional prosody discrimination and recognition were found, but there was a normal performance in the visual presentation of the same emotions and an absence of biased responses towards choosing any specific emotion. However, we cannot fully prove it because, like many other studies, we did not include a task assessing recognition of general/propositional prosody. Conversely, the second hypothesis seems unlikely as it would imply a complete segregation of all emotion networks depending upon the sensorial modality.

Our a priori hypothesis that dysfunctional basal ganglia in PD would have a determinant role in impairing emotion processing was therefore not confirmed, and a deficit in emotional prosody recognition was found instead, which leads to the discussion of the role of basal ganglia in prosody processing.

Prosody recognition encompasses emotional and propositional prosody. Its impairment has been described after focal lesions of the basal ganglia (Cancelliere & Kertesz, 1990; Starkstein, Federoff, Price, Leiguarda, & Robinson, 1994).

While selective disturbances of emotional prosody have been associated to lesions in the right hemisphere, propositional prosody disorders were related to damage of either hemisphere (Heilman, Bowers, Speedie, & Coslett, 1984; Pell, 2006; Ross & Monnot, 2008; Starkstein et al., 1994). In this respect, PD is a model of bilateral dysfunction of basal ganglia, although asymmetric, and thus disturbances in both emotional and propositional prosody would be expected. Studies in early PD have shown either a disturbed recognition of propositional or global (emotional and nonemotional) prosody (Blonder, Gur, & Gur, 1989; Lloyd, 1999) or disturbances affecting only recognition of emotional prosody (Breitenstein et al., 2001; Dara et al., 2008; Pell & Leonard, 2003), although in the latter studies nonemotional prosodic categories were not tested.

Concerning the pathogenesis of those impairments, different cognitive mechanisms have been proposed to explain why basal ganglia lesions might lead to impairments of prosody processing. First, there may be a general disorder of time processing (Coelho et al., 2004) in sequential auditory stimuli (e.g., speech). Secondly, there may be a more specific inability in the comprehension and discrimination of lexical stress in speech. Finally, an executive dysfunction due to the progressive deterioration of the frontal–striatal circuits might compromise tasks requiring inhibitory control and working memory, as in the case of prosody judgments (requiring the analysis of emotional prosodic information that is not in agreement with the neutral content of the sentence) and prosody discrimination (requiring the simultaneous judgment of a large piece of information online; Breitenstein et al., 2001; Lloyd, 1999; Paulmann & Pell, 2010; Pell & Leonard, 2003). Our study was not directed to test these mechanisms, but the fact that our patients scored within the normal range in cognitive tests involving frontal-executive networks (digit span backwards, verbal fluencies, Stroop test color–word interference) and more general cognition (MMSE, Dementia Rating Scale), and the absence of correlations between cognitive and emotional performances rather excludes the latter hypothesis.

The basal ganglia may also play a role in the production of prosody, as suggested by Benke et al. (1998) and Ariatti et al. (2008), who have pointed to a parallel between disturbed recognition and production of aprosodic speech in PD. Dysarthria per se is not sufficient to give rise to dysfunction in emotional prosody recognition, as studies in

amyotrophic lateral sclerosis show (Zimmerman, Eslinger, Simmons, & Barrett, 2007), although slight dysarthria after cerebellar stroke has been associated with deficits in the recognition of emotional prosody but sparing propositional prosody (Adamaszek et al., 2014). Altogether the basal ganglia may have a unique and determinant role in the neuronal networks both for the production and for the judgement of prosody, through its connections with higher associative temporal and prefrontal cortices (Pichon & Kell, 2013).

Finally the absence of deficit in visual recognition of different emotions in the current APD may seem to contrast with: (a) their difficulty in identity discrimination of neutral faces; and (b) previous reports of impairment in facial emotion recognition (Alonso-Recio et al., 2014; Clark et al., 2010; Gray & Tickle-Degnen, 2010; Herrera, Cuetos, & Rodriguez-Ferreiro, 2011; Narme, Bonnet, Dubois, & Chaby, 2011).

These findings may have been caused by a limited number of stimuli and differences in task sensitivity within the visual tasks (identity versus facial emotion) and between the visual and auditory tasks, and were discussed elsewhere for the same tasks (Albuquerque, Coelho, Martins, & Martins, 2014; Martins, Moura, Martins, Figueira, & Prkachin, 2011), or by the fact that in our subset of APD amenable to surgery, cognitive decline and/or executive dysfunction were absent, contrarily to previous series (Alonso-Recio et al., 2014; Clark et al., 2010).

Another limitation of this study is the lack of evaluation of other forms of prosody in APD. Emotional and propositional prosody need to be compared in order to confirm the relative integrity of emotion networks in comparison to those involved in general prosody processing. Results on the recognition of music emotions may also differ from speech findings (Lima et al., 2013). Another caveat might be the role of medication versus disease itself, since patients were tested under dopaminergic medication. Two recent studies testing memory of emotional faces described impairments in PD that were improved by dopaminergic medication (Halbig et al., 2011; Subramanian, Hindle, Jackson, & Linden, 2010).

In summary, nondemented APD DBS candidates in the “ON” condition disclosed a clear impairment in recognizing emotional prosody. However, there was no deficit in emotion recognition in the visual presentation and no deficit in the processing of particular emotions. Taken

together, available data support some impairment in prosody recognition, along with relative sparing of emotional networks in APD. Although these results need to be confirmed in larger clinical samples with a more comprehensive assessment of prosody processing, the identified difficulty of emotional prosody recognition and its impact in social interaction or behavior should be explored.

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