Recognition of emotions from visual and prosodic cues in Parkinson’s disease

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Abstract Objective To assess whether Parkinson Disease (PD) patients are impaired at perceiving emotions from facial and prosodic cues and whether any putative defective performance concerns recognition of a particular emotion. Background Braak et al. [1] demonstrated that in different stages PD pathology involves the nigrostriatal system, the amygdala, and the insular cortex. Discrete brain lesions to these structures can cause selective deficits in recognising facial and prosodic stimuli expressing particular emotions. However, the investigation of facial and prosodic emotional processing in PD patients has lead to conflicting results. Materials and methods We compared 27 cognitively unimpaired PD patients with control subjects by means of the Facial Emotion Recognition Battery and the Emotional Prosody Recognition Battery. Results PD patients were impaired in recognising, selecting, and matching facial affects. In particular, the Facial Emotion Recognition Battery demonstrated a severe impairment in recognising sad and fearful faces. In the Emotional Prosody Recognition Battery PD patients demonstrated a diffuse impairment, including the recognition of emotional and propositional prosody. Conclusions Face emotion processing is impaired in PD patients, with a disproportionate deficit involving fear and sadness. The pattern of face expression processing impairment in PD patients might depend on the regional distribution of the pathology. The widespread involvement of both emotional and propositional prosodic processing parallels the aprosodic characteristics of Parkinsonian speech production.

Keywords Parkinson’s disease · Emotions · Facial expressions · Emotional prosody

Introduction

Both neuropsychological and neuroimaging data support the notion that cortical and subcortical regions are involved in processing emotions from facial and prosodic cues. A large number of different structures participate in recognising the facial expression of emotions: among them, the occipito-temporal cortex, the amygdala, the orbito-frontal cortex, the basal ganglia, and the right parietal cortex [2]. In contrast, studies on emotional prosody do not clearly indicate any similar neural network. In general, recognising emotions from prosody alone is more difficult than recognising emotions from facial expressions. Certain emotions, such as disgust, can be recognized only very poorly from prosody. The recognition of emotions from the voice draws on multiple prosodic cues, which are, in turn, processed by systems that in part are neuroanatomically segregated towards one or the other hemisphere. In numerous studies, the right fronto-parietal region has consistently emerged as critical for recognising emotional prosody [3–6], a role it might play in association with the basal ganglia [4]. Specific cerebral structures seem to be involved in the processing of specific visual and prosodic emotions. Neuropsychological [7, 8] and neuroimaging data [9] support this view. For instance, there is a wide agreement...
that amygdala mediates the recognition of fear across visual, auditory and gesture stimuli [10].

Functional imaging studies demonstrate the involvement of the basal ganglia, but also of the insular cortex, in facial disgust recognition [11–14]. The role of these structures in disgust processing is also supported by studies that have found that individuals with neurodegenerative diseases are impaired at recognising the facial expression of disgust. For instance, patients with symptomatic Huntington’s disease [15–18] and pre-symptomatic Huntington’s disease gene carriers [11, 19] showed an overall deficit in recognising emotions, with a particularly severe deficit for recognising expressions of disgust from both face and voice. Patients with Wilson’s disease [20], Tourette syndrome with comorbid obsessive compulsive behaviour, and people with obsessive compulsive disorder [21], all show selective deficits in facial disgust recognition.

In contrast, it is far from clear whether Parkinson’s Disease (PD) patients are impaired at perceiving emotional facial affects. Individuals with PD have a reduced ability in making spontaneous emotional expressions and have monotonous, flat, and poorly inflected speech. It has been suggested [22] that there is a strong link between perception and expression of emotions. Jacobs et al. [23] demonstrated that PD patients are impaired in imaging, perceiving, and expressing emotional faces. They explained the relationship between motor and perceptual-imaginative aspects with the notion of a central processor located in the basal ganglia or dependent upon neostriatal function. Indeed, the neostriatum receives input from the whole cortex, including projections from the inferior and the superior temporal gyri, which are involved in visual perceptual representations of face and facial emotions. Dujardin et al. [24] established that early in the course of PD, non-verbal emotional information processing is disturbed and that untreated PD patients are significantly impaired in decoding emotional facial expressions. They suggested that in PD nigrostriatal dopaminergic depletion leads not only to motor and cognitive disturbances, but also to emotional information processing deficits. Specifically this study showed that PD patients were less accurate than controls in decoding anger, sadness and disgust from facial expressions, regardless of the expression’s intensity level (low or mild). Suzuki et al. [25], using the facial expression hexagon, showed that PD patients were selectively impaired at recognising the facial expression of disgust. Wieser et al. [26] found that PD patients report less arousal compared to healthy controls during extended viewing of emotional pictures. They demonstrated that reduced arousal was not attributable to generally increased fatigue or an overall enhanced depression score.

Tessitore et al. [27] explored the neural basis of abnormal emotional behaviour in PD using fMRI. They found that, in non-medicated PD patients, an emotional task was not associated with bilateral amygdala activation as in normal subjects. Dopaminergic repletion, however, was shown to restore this response, at least partially. Absence of amygdalar response in PD patients was also found by Yoshimura et al. with visual event-related potentials (ERPs) related to viewing of fearful facial expressions [28].

Sprengelmeyer et al. [29] investigated the effect of dopamine medication and observed impaired recognition of disgust and anger from facial expressions. This deficit was more severe in non-medicated patients than in medicated patients with PD. The dopaminergic role on emotion perception and recognition was also emphasized by Salgado-Pineda et al. [30].

In contrast with this hypothesis, several studies failed to demonstrate any difference between patients and controls in facial emotion tasks [31–33]. Conflicting results have been also reported by studies investigating defective production and recognition of emotional and propositional prosody (e.g. the prosody involved in asking a question or stating an affirmation [34]) in PD subjects [33, 35–39]. Caekebeke et al. [40] demonstrated a deficit in producing, but not in recognising, prosody and facial expression, thus suggesting that the origin of the impairment may be the result of dysarthria and not necessarily related to defective encoding/decoding of affective behaviour. Kan et al. [41] found that PD patients are impaired in recognising fear and disgust from facial expression, but they did not find significant differences between PD patients and controls in recognition of emotion from prosodic and written verbal stimuli. Yip et al. [42] evaluated the emotion recognition abilities of a group of bilateral and right-sided PD patients. Results demonstrated that although patients with bilateral PD were impaired in recognising all six basic emotions, fear, followed by sadness, was more impaired than other basic emotions (disgust, surprise, anger and happiness). Bilateral PD patients showed defective performance in all tests used, irrespective of the modality and the type of experimental task. In contrast, patients with right-sided PD had difficulties with facial emotion identification and prosodic emotion identification only. Furthermore, the recognition of all emotions in patients with right-sided PD was significantly impaired and particularly the recognition of sadness and disgust.

Summing up, there is no clear evidence of either a general or a specific impairment of emotional processing in PD. When present, the impairment seems to involve negative facial affects such as disgust or fear. No specific deficit for prosodic emotion has been found. The present study addressed this issue evaluating the presence of a general or selective emotional impairment in PD across the visual and the auditory domain. We set up two batteries for the facial and the prosodic emotion evaluation using equivalent tasks within the two batteries. Our results suggest a specific impairment in recognising fear and sadness from facial cues in PD patients. In addition,
we found defective performances in PD patients both in emotional and non-emotional prosody processing.

Materials and methods

Subjects

Patients with diagnosis of idiopathic Parkinson Disease (PD patients) were selected through the outpatient Movement Disorder Clinic of the Modena University. They included 27 PD patients (15 males and 12 females). All patients had a negative CT or MR scan of the brain. With respect to the most affected body side, 13 patients were considered right-sided and 13 left-sided. This classification was not applicable for one patient (V. M.). Sixty-eight healthy volunteers (28 males and 40 females; mean age= 62.5 years) participated in the study as controls. All participants were free of alcoholism, psychiatric illness, head trauma, cerebrovascular disease, and other severe neurological conditions.

Criteria of patient selection included absence of general and severe cognitive deterioration (MMSE ≥23), severe depression as evaluated by the Beck Depression Inventory (BDI <23 [44]) and obsessive-compulsive disorder assessed by the reduced version of the Maudsley Obsessional-Compulsive Questionnaire (MOCQ-R) <75 percentile [45]. All subjects were fully independent in everyday life activities and they all gave their informed consent to take part in the study. The study was approved by the Ethics Committee of the University of Modena and Reggio Emilia. Table 1 shows demographic, clinical and neuropsychological data of PD patients included in the study.

Table 1 Demographic, clinical, and neuropsychological data of PD patients

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<tr>
<th>Patient</th>
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MMSE, Mini Mental State [43]; BDI, Beck Depression Inventory [44]; MOCQ/R, Maudsley Obsessional-Compulsive Questionnaire [45]; MCST, Modified Card Sorting Test; H.E. Nelson, 1976 [64]; FAB, Frontal Assessment Battery [65]; UPDRS, Unified Parkinson’s Disease Rating Scale [66]; H.Y., Hoehn & Yahr Scale [67]. Scores are corrected for age and education (MMSE, FAB) and age, sex and education (Raven).

* Categories achieved and perseverative errors

Stimuli and experimental design

The Facial Emotion Recognition Battery used face pictures from the Ekman and Friesen [46] series showing the following expressions: neutral, happiness, sadness, disgust, fear and anger. The Battery included the following tasks:

1. Face Matching (FM) is a control task consisting of 14 trials. It requires selecting from a vertically arranged
set of four faces with neutral expression the identical photograph of the stimulus face. Distracters are photographs of different persons of the same sex.

2. Facial Identity Recognition (FIR) measures the ability of recognising one person’s identity despite different expressions. It consists of 14 trials. The subject is requested to select the target person among a vertically arranged set of four faces with different expressions.

3. Facial Affect Naming (FAN) requires the subject to select the name of the emotional expression of a face among five alternatives printed vertically below the stimulus face. The test consists of 25 trials.

4. Facial Affect Selection (FAS) requires the participant to select from a vertically arranged set of five faces the one that bears the expression corresponding to a target label. The test consists of 25 trials.

5. Facial Affect Matching (FAM) requires the subject to select among a vertically arranged set of five faces the one bearing the same expression as the stimulus face.

The Emotional Prosody Recognition Battery consisted in a visual and auditory presentation of Italian sentences arranged in four different tasks:

1. Vocal Identity Discrimination (VID) requires indicating whether two sentences were pronounced by the same individual. The test consists of 16 pairs of neutral (aprosodic) stimuli.

2. Prosodic Discrimination (PrD) requires indicating whether two sentences presented were pronounced with the same prosodic intonation. The test consists of 16 pairs of sentences expressing four different intonations: interrogative, declaratory, exclamatory, and imperative.

3. Prosodic Affect Naming (PrAN) requires the subject to select among five alternatives the emotion conveyed by the intonation of a sentence. The five alternatives are presented on the computer screen. The test consists of 25 trials, 5 for each emotion (happiness, fear, disgust, anger, and sadness).

4. Prosodic Affect Discrimination (PrAD) requires indicating whether two sentences presented were pronounced with the same emotional prosody. It consists of 45 pair of sentences.

Only 11 out of 27 patients (7 right-sided and 4 left-sided) performed the Emotional Prosody Recognition Battery.

Results

Emotion recognition battery

The proportion of correct responses of PD patients and controls was compared for each of the different subtests by means of separate non-parametric analyses (Mann-Whitney U tests). Level of statistical significant was adjusted for multiple comparison with Bonferroni correction (alpha= 0.0102 for each test).

PD patients compared with controls, irrespectively of the most affected side, were significantly impaired at the Facial Affect Name Recognition (U=538.0; \( p < 0.001 \); mean correct responses = 81.04% in PD patients and 89.35% in controls), and at the Facial Affect Matching tasks (U=450.5; \( p < 0.001 \); mean correct responses = 72.59% in PD patients and 87.06% in controls; Fig. 1). PD patient impairment was close to significance at the Facial Affect Selection (U=628; \( p = 0.016 \); mean correct

![Fig. 1 Mean correct identification rate in the Emotion Recognition Battery across the two experimental groups. * represent statistical significance at \( p = 0.0102 \); whiskers are SE](image-url)
responses = 84.74% in PD patients and 92.23% in controls). There was no significant difference between PD patients and controls at the Face Matching and at the Facial Identity Recognition tasks.

We then considered the proportion of responses for each of the five emotions at the Facial Affect Name Recognition. Adjusting the level of statistical significance for multiple comparisons with the Bonferroni correction (alpha=0.0102), PD turned out to be impaired on fear (U=582.5; p=0.005; mean correct responses = 56.29% in PD patients and 73.82% in controls) and sadness recognition (U=608; p=0.01; mean correct responses = 79.26% in PD patients and 93.52% in controls), but they appeared to perform as well as control subjects with faces expressing happiness and there was no significant difference between PD patients and controls with faces expressing anger and disgust. In particular, with respect to controls, right-sided PD patients were impaired in recognition of fearful faces (U=225.5; p=0.004; mean correct responses = 50.77% in right-sided patients and 58.46% in left-sided patients) whereas left-side patients were impaired in recognition of sad faces (U=188; p<0.001; mean correct responses = 84.61% in right-sided patients and 72.31% in left-sided patients; Fig. 2).

Emotional prosody recognition battery

The proportion of correct responses of PD patients and controls was compared for each of the different subtests by means of separate non-parametric analyses (Mann-Whitney U tests). Level of statistical significance was adjusted for multiple comparison with Bonferroni correction (alpha=0.0127 for each test).

Compared with controls, PD patients were significantly impaired at the Vocal Identity Discrimination (U=198.5; p=0.003; mean correct responses = 69.79% in PD patients and 80.61% in controls), at the Prosodic Discrimination (U=153.5; p<0.001; mean correct responses = 73.96% in PD patients and 86.4% in controls) and at the Prosodic Affect Naming task (U=149.5; p<0.001; mean correct responses = 53% in PD patients and 77.53% in controls). There was no significant difference between PD patients and controls at the Prosodic Affect Discrimination task.

With respect to controls, right sided patients were significantly impaired at the Vocal Identity Discrimination (U=85.5; p=0.003; mean correct responses = 66.96% in right-sided patients and 68.75% in left-sided patients), at the Prosodic Discrimination (U=93; p=0.006; mean correct responses = 73.21% in right-sided patients and 75% in left-sided patients) and at the Prosodic Affect Naming task (U=49.5; p<0.001; mean correct responses = 53.14% in right-sided patients and 55% in left-sided patients; Fig. 3).

Adjusting the level of statistical significance for multiple comparisons with the Bonferroni correction (alpha=0.0102), we considered the proportion of correct response for each of the five emotions at the Prosodic Affect Naming task. PD patients were impaired in recognising happy intonation (U=218; p=0.005; mean correct responses = 60% in PD patients and 88.25% in controls). In particular, right-sided PD patients were significantly impaired in recognising the intonation expressing disgust in comparison both with control subjects and with left-sided PD patients (U=67; p<0.001; mean correct responses = 11.43% in right-sided patients and 55% in left-sided patients Fig. 4) while the performance of left sided PD patients was significantly different from con-
trols in recognising happy intonation (U=21; p=0.002; mean correct responses = 68.57% in right-sided patients and 35% in left-sided patients; Fig. 4).

Patients appeared to perform as well as control subjects in sadness, fear, and anger recognition.

Lastly, we did not find any significant correlation between the two emotional battery scores, the Beck Depression Inventory, the reduced version of the Maudsley Obsessional-Compulsive Questionnaire, tests exploring executive functions (FAB, Raven and MCST), the motor score at the UPDRS, and disease duration.

Conclusions

A number of studies have reported that PD patients are impaired in recognising emotional facial expressions [23–25, 35, 47] and emotional prosodic information [7, 35, 41, 42]. However, other neuropsychological studies failed in demonstrating differences between patients and controls in several emotional tasks [26, 31–33, 40].

These conflicting results may be attributed to selection bias (inclusion of patients with depression, cognitive impairment, different disease stages, medication, disease...
duration and/or different most affected side). Also, it is possible that different findings depend on different tasks for assessing emotional processing.

With this study we have evaluated emotional processing across both the visual and the auditory domain in cognitively unaffected PD patients. The different tasks included in the two emotional batteries aimed at detecting generic or specific emotional deficit across the two sensory modalities.

The results of the Emotional Prosody Recognition Battery demonstrated that PD patients are impaired in processing not only emotional but also propositional prosody. This finding seems to parallel the apropoic characteristic of speech produced by PD patients. In addition, the data from controls and patients confirmed that recognising emotions from prosody is more difficult than recognising emotions from facial expressions, and certain emotions such as disgust can be recognized only very poorly from prosody. Furthermore, only 11 out of 27 PD patients enrolled in this study were also examined with the Emotional Prosody Recognition Battery. For these reasons it is difficult to disentangle a possible emotional processing component from the mere general impairment in processing prosody. However, a more general impairment in processing prosodic intonation cannot explain the finding of a double dissociation between most affected side and most impaired emotion (with right-sided PD patients being impaired in processing intonation expressing disgust while left-sided PD patient are more impaired in processing happy intonation).

Emotion recognition impairment could not be explained by a more general cognitive deficit. Indeed, we excluded from this study cognitively impaired PD patients. In addition, those that were examined turned out to be unimpaired at the Face Discrimination, a test aimed at assessing perceptual processing of individual characteristics of faces. Moreover, lack of correlation between our tasks and the Beck Depression Inventory rules out the hypothesis that the emotion recognition impairment of PD patients can be attributed to depression.

UPDRS motor score did not correlate with the ability to recognize emotions from facial and prosodic cues, thus suggesting that the facial emotion recognition impairment does not simply follow the motor disease and is probably due to the damage of brain structures different from those causing the motor impairment.

The results of the Emotion Recognition Battery also suggest the possibility of a selective deficit in the facial expression recognition which depends on the side most affected by the disease, with right-sided PD patients being impaired in recognition of faces expressing fear whereas left-sided patients are impaired in recognition of sad expression. A number of clinical [48] and neuroimaging studies [49, 50] have shown the role of the amygdala in processing the facial expression of fear [51–53]. In particular, neuropsychological [54, 55] and neuroimaging studies [56–58] have consistently found that the right amygdala is involved in processing fearful facial expressions. Activation of the left amygdala in processing faces expressing sadness have also been found in normal subjects [59]. Moreover, morphological and functional abnormalities in left amygdala have been found in psychiatric diseases and in particular in depression [60], in schizophrenia [61] and in the Asperger syndrome [62, 63].

A number of studies have described mild executive dysfunction in patients with early Parkinson Disease and the role of impaired cortico-basal connections in determining them [68, 69]. However the fact that we did not find any correlation between tasks exploring frontal dysfunctions and scores at the two emotional batteries argues against ascribing the emotional deficit to impaired cortico-basal connections. In contrast, our results are in line with data indicating that the amygdala is damaged early during the course of Parkinson’s disease. Braak et al. [1] suggested that Parkinson’s disease is characterized by a progressive pathological process marked by the presence of α-synuclein-immunoreactive inclusions, affecting circumscribed regions of the brain in a topographically predictable sequence, during which components of the olfactory, autonomic, limbic, and somatomotor system become progressively involved.

According to this view, the amygdala (and particularly its central subnucleus) is damaged early during the course of the disease, possibly even before substantia nigra/pars compacta, which is responsible for the motor impairment in PD.

Evidence about performance at emotional processing tasks in atypical Parkinsonism is still lacking. However, it is possible that when carefully investigated, the impaired emotional processing of PD patients might turn out to be a useful complement in the differential diagnosis of Parkinsonian syndromes.

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References


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