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Facial Emotion Recognition in Parkinson's Disease: A Review and New Hypotheses

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ABSTRACT: Parkinson's disease is a neurodegenerative disorder classically characterized by motor symptoms. Among them, hypomimia affects facial expressiveness and social communication and has a highly negative impact on patients' and relatives' quality of life. Patients also frequently experience nonmotor symptoms, including emotional-processing impairments, leading to difficulty in recognizing emotions from faces. Aside from its theoretical importance, understanding the disruption of facial emotion recognition in PD is crucial for improving quality of life for both patients and caregivers, as this impairment is associated with heightened interpersonal difficulties. However, studies assessing abilities in recognizing facial emotions in PD still report contradictory outcomes. The origins of this inconsistency are unclear, and several questions (regarding the role of dopamine replacement therapy or the possible consequences of hypomimia) remain unanswered. We therefore undertook a fresh review of relevant articles

focusing on facial emotion recognition in PD to deepen current understanding of this nonmotor feature, exploring multiple significant potential confounding factors, both clinical and methodological, and discussing probable pathophysiological mechanisms. This led us to examine recent proposals about the role of basal ganglia-based circuits in emotion and to consider the involvement of facial mimicry in this deficit from the perspective of embodied simulation theory. We believe our findings will inform clinical practice and increase fundamental knowledge, particularly in relation to potential embodied emotion impairment in PD. © 2018 The Authors. Movement Disorders published by Wiley Periodicals, Inc. on behalf of International Parkinson and Movement Disorder Society.

Key Words: facial emotion recognition; Parkinson's disease; basal ganglia; dopamine; embodied simulation

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The original description characterizing Parkinson's disease (PD) by motor symptoms^{1,2} has been updated because patients also experience cognitive and psychiatric symptoms,³⁻⁷ including emotional impairments. These lead to difficulties in describing bodily sensations, physiological arousal and feelings, expressing emotions, and identifying others' emotions from prosody and facial expression.⁸⁻¹⁰ Facial emotion recognition (FER) is one of the most basic aspects of emotional functioning and one of the most critical components of social behaviors. Aside from its theoretical importance, understanding FER disruption in PD is crucial for improving quality of life for both patients and caregivers. However, studies assessing FER in PD still report contradictory results. Although 2 reviews and 1 meta-analysis revealed an FER deficit in PD and outlined potential biasing factors,^{9,11,12} the origins of this inconsistency

are unclear, and several questions remain unanswered. For instance, what is the actual effect of dopaminergic medication on FER in PD? Does hypomimia affect FER in accordance with embodied simulation theory? From a more methodological perspective, are the tasks currently used sensitive enough to reveal impairments among patients? In short, we know that FER is impaired in PD but do not have sufficient information to fully understand the underlying mechanisms.

Building on a previous work,⁹ we therefore undertook a fresh review of FER in PD to enhance current understanding of this nonmotor feature while exploring significant potential confounding factors. Regarding probable pathophysiological mechanisms subtending impaired FER in PD, we looked at recent considerations about the role of basal ganglia-based circuits in emotion. We also reconsidered hypotheses about embodied simulation that merit further research, despite being mentioned in early studies. We end by discussing the clinic and social consequences of emotional dysfunctions in PD.

Methods

We conducted a detailed search of the literature to extend previous reviews.^{9,11,12} We searched PubMed and Web of Science services with the following key words: *Parkinson's disease, emotion recognition, facial expression, and decoding*. We also hand-searched relevant journals and examined the references of retrieved

articles. Articles were restricted to the English language and published between January 1983 and July 2016. This yielded 167 eligible articles. Some publications were excluded (eg, reviews, studies related to deep-brain stimulation, articles focused on emotion production/experience). A total of 59 articles reporting 97 comparisons between PD patients and healthy controls (HCs) during FER tasks were included. For each experiment, we checked whether significant differences between patients and HCs were highlighted and whether the presence of an emotion-specific deficit was investigated. We also looked at which errors were made by participants during the experiment to characterize the deficit in detail. Subsequently, we considered the impact of multiple confounding factors. We did not attempt to provide an exhaustive report on the substantial differences across experiments enrolling heterogeneous samples, which may explain inconsistent results but make comparisons across studies well nigh impossible. Rather, we discussed the factors that may play a role in FER impairment in PD and should be taken into account in future studies.

Facial Emotion Recognition in PD

Overview of Performances

Table 1 provides an overview of the reviewed studies. For each experiment, it indicates (1) the FER task used, (2) whether patients' performance were significantly lower than that of HCs, (3) whether the deficit

TABLE 1. Overview of performance on FER tasks (1 of 3)

	Task	Deficit	Anger	Disgust	Fear	Sadness	Surprise	Happiness	Neutral
Scott et al, 1984 ^a	MATCH	+	?	?	?	?	?	?	?
Beatty et al, 1989	ID	+	+	+	+	+	+	+	+
Blonder et al, 1989	ID	+	+	+	+	+	+	+	+
Blonder et al, 1989	MATCH	+	?	?	?	?	?	?	?
Borod et al, 1990	ID	-	-?	-?	-?	-?	-?	-?	-?
Borod et al, 1990	DISCRI	-	-?	-?	-?	-?	-?	-?	-?
Caekbeke et al, 1991 ^b	DESCRI	-	-?	-?	-?	-?	-?	-?	-?
Dewick et al, 1991 ^c	MATCH	-	-	-	-	-	-	-	-
Madeley et al, 1995 ^c	MATCH	-	-?	-	-	-?	-	-?	-
Madeley et al, 1995	ID	+	+	+	-	-	-	-	-
Haeske-Dewick, 1996 ^c	MATCH	-	-?	-	-	-?	-	-?	-
Jacobs et al, 1995	DISCRI	+	?	?	?	?	?	?	?
Jacobs et al, 1995	MATCH	-	-	-	-	-	-	-	-
Jacobs et al, 1995	IMAG	+	-	-	+	+	-	-	-
Jacobs et al, 1995	DESCRI	+	-	-	+	+	-	-	-
Breitenstein et al, 1998 (1)	ID	-	-	-	-	-	-	-	-
Breitenstein et al, 1998 (1)	MATCH	-	-	-	-	-	-	-	-
Breitenstein et al, 1998 (1)	DISCRI	-	-	-	-	-	-	-	-
Breitenstein et al, 1998 (1)	NAME	-	-	-	-	-	-	-	-
Breitenstein et al, 1998 (2)	ID	+	+	-	+	+	+	+	+
Breitenstein et al, 1998 (2)	MATCH	+	+	-	+	+	+	+	+
Breitenstein et al, 1998 (2)	DISCRI	+	+	-	+	+	+	+	+
Breitenstein et al, 1998 (2)	NAME	+	+	-	+	+	+	+	+
Adolphs et al, 1998	INT	-	-	-	-	-	-	-	-

(Continued)

TABLE 1. Continued

	Task	Deficit	Anger	Disgust	Fear	Sadness	Surprise	Happiness	Neutral
St Clair et al, 1998	ID	-	?	?	?	?	?	?	?
Kan et al, 2002	ID	+	-	+	+	-	-	-	-
Tessitore et al, 2002	MATCH	-	-?	-	-?	-	-	-	-
Yip et al, 2003 (1)	ID	+	+	+	+	+	+	+	+
Yip et al, 2003 (1)	DISCRI	-	-?	-?	-?	-?	-?	-?	-?
Yip et al, 2003 (2)	ID	+	+	+	+	+	+	+	+
Yip et al, 2003 (2)	DISCRI	+	+	+	+	+	+	+	+
Sprengelmeyer et al, 2003 (1)	ID	+	+	+	+	+	-	-	-
Sprengelmeyer et al, 2003 (1)	HEXA	+	+	-	+	-	-	-	-
Sprengelmeyer et al, 2003 (2)	ID	+	+	+	+	+	-	-	-
Sprengelmeyer et al, 2003 (2)	HEXA	-	-	-	-	-	-	-	-
Dujardin et al, 2004 (1)	ID	+	+	+	+	+	-	-	-
Dujardin et al, 2004 (2)	INT	+	+	+	+	+	-	-	-
Yoshimura et al, 2005	SCREEN	-	-	-	-?	-	-?	-	-?
Pell and Leonard, 2005	ID	-	-	-	-	-	-	-	-
Pell and Leonard, 2005	DISCRI	-	?	?	?	?	?	?	?
Pell & Leonard, 2005 (1)	ID	-	-	-	-	-	-	-	-
Pell & Leonard, 2005 (2)	INT	+	+	-	-	+	-	-	-
Suzuki et al, 2006	ID	-	-	-	-	-	-	-	-
Suzuki et al, 2006	HEXA	-	-	-	-	-	-	-	-
Suzuki et al, 2006 ^d	INT	+	-	+	-	-	-	-	-
Lachenal-Chevallet et al, 2006	ID	+	-	+	+	-	-	-	-
Lawrence et al, 2007	ID	+	+	-	-	-	-	-	-
Ariatti et al, 2008	ID	+	-	-	+	+	-	-	-
Ariatti et al, 2008	MATCH	+	+	+	+	+	-	+	-
Clark et al, 2008	ID	+	+	-	-	-	+	-	-
Martins et al, 2008	ID	+	+	-	+	-	-	-	-
Delaveau et al, 2009 ^e	MATCH	-	-	-	-	-	-	-	-
Ibarretxe-Bilbao et al, 2009	ID	+	+	+	+	+	+	-	-
Assogna et al, 2010	ID	+	-	+	-	-	-	-	+
Cohen et al, 2010	ID	-	-?	-?	-?	-?	-?	-?	-?
Paulmann and Pell, 2010 ^f	ID	+	+	+	+	+	+	+	+
Clark et al, 2010	ID	+	+	-	-	-	+	-	-
Martinez-Corral et al, 2010 (1)	ID	-	-?	-?	-?	-?	-?	-?	-?
Martinez-Corral et al, 2010 (2)	ID	+	-	-	+	+	-	-	-
Narme et al, 2011	ID	+	+	-	+	+	-	-	-
Herrera et al, 2011	ID	+	+	+	+	+	+	+	-
Wieser et al, 2012	ID	-	-	-	-	-	-	-	-
Baggio et al, 2012	ID	+	+	+	+	+	-	-	-
Buxton et al, 2012 ^g	ID	+	-	+	-	+	+	+	-
Ventura et al, 2012 ^h	ID	-	-	-	-	-	-	-	-
Ventura et al, 2012 ^h	DISCRI	-	-?	-	-?	-?	-?	-?	-?
Ventura et al, 2012 ^h	MATCH	-	-?	-	-?	-?	-?	-?	-?
Garcia-Rodriguez et al, 2012 (1)	ID	-	-?	-?	-?	-?	-?	-?	-?
Garcia-Rodriguez et al, 2012 (2)	ID	+	+	+	+	+	+	+	-
Saenz et al, 2013	ID	+	-	-	+	+	-	-	+
Narme et al, 2013	ID	+	-	-	+	+	-	-	+
Alonso-Recio et al, 2013	ID	+	+	+	+	+	+	+	-
Alonso-Recio et al, 2013	DISCRI	-	-?	-?	-?	-?	-?	-?	-?
Hipp et al, 2014	ID	+	-	-	+	-	-	-	-
Alonso-Recio et al, 2014a	DISCRI	+	+	+	+	+	+	+	-
Alonso-Recio et al, 2014b (1)	ID	+	+	+	+	+	+	+	-
Alonso-Recio et al, 2014b (2)	DISCRI	+	+	+	+	+	+	+	-
Marneweck and Hammond, 2014 ⁱ	DISCRI	+	+	+	+	+	+	+	-
Marneweck et al, 2014 (1)	DISCRI	+	+	+	+	+	+	+	-
Marneweck et al, 2014 (2)	DISCRI	+	+	+	+	+	+	+	-
Marneweck et al, 2014 (3)	DISCRI	+	+	+	+	+	+	+	-
Marneweck et al, 2014 (4)	ID	+	+	+	+	+	+	+	-
Wabnegger et al, 2015 (1)	ID	-	-	-	-	-	-	-	-
Wabnegger et al, 2015 (2)	INT	+	-	-	+	+	+	+	-
Laskowska et al, 2015 ^j	EIS-F	+	+	+	+	+	+	+	-

(Continued)

TABLE 1. Continued

	Task	Deficit	Anger	Disgust	Fear	Sadness	Surprise	Happiness	Neutral
Enrici et al, 2015	ID	+	+	+	+	+	+	+	
McIntosh et al, 2015	ID	+	-	+	+	-	-	-	+
Ricciardi et al, 2015	ID	+	-	-	-	-	+	-	
Ille et al, 2016 (1)	ID	-	-	-	-	-	-	-	
Ille et al, 2016 (2)	INT	+	-	-	+	-	-	-	
Pietschnig et al, 2016	ID	+	-	-	-	-	-	-	
Albuquerque et al, 2016	ID	-	-	-	-	-	-	-	-
Albuquerque et al, 2016	DISCRI	-	-	-	-	-	-	-	-
Wagenbreth et al, 2016	ID	+	-	+	+	-	+	-	-
Lin et al, 2016 (1)	ID	+	-	-	-	+	-	-	
Lin et al, 2016 (2)	ID	+	+	-	-	+	+	-	
Argaud et al, 2016 ^k	ID	+	-	-	-	-	-	+	+
Quantitative summary:	n = 97	64%	44%	47%	54%	51%	30%	27%	42%

Task: type of task used by the authors (see Table 2 for details). Deficit: “was the performance of patients with PD significantly lower than that of healthy controls?” A plus sign in a dark-shaded box indicates “yes, a facial emotion recognition deficit was highlighted according to a global score” (ie, whatever the displayed emotion). A minus sign in a white box indicates “no, there was no group difference.” When the authors investigated the groups’ performance as a function of the displayed emotion, the same symbols are used in the boxes corresponding to the specific emotions: a plus sign in a dark-shaded box indicates that the deficit affected this specific emotion, whereas a minus sign in a white box indicates that recognition of this specific emotion was spared. On the contrary, when the authors only examined the groups’ performance according to an overall score encompassing all the emotions, these signs are followed by a question mark. Question marks on their own mean that no information is available about the emotions displayed in the study. When the box relating to a specific emotion is empty, it means that the latter was not manipulated in the experiment. Quantitative summary: the percentage of the experiments we reviewed (n = 97) that highlighted a facial emotion recognition deficit in PD (general or specific) is indicated in the “Deficit” column. Likewise, for each specific emotion, we indicate the percentage of experiments that specifically showed that the emotion recognition deficit affected that emotion in particular (ie, when the authors submitted this emotion to the participant and examined the performance of recognition according to this emotion).

^aIn Scott et al (1984), participants were required first to describe a target facial emotion, then to match it with an affective sentence.

^bCaekebe et al (1991) also exposed participants to contempt (no group difference).

^cIn Dewick et al (1991), Madeley et al.(1995), and Haeske-Dewick (1996), participants were required to choose which facial expression in a pair of photographs of the same person’s face expressing different emotions matched a printed label. Breitenstein et al (1998) assessed FER among patients who met the criteria for (1) stage I or (2) stage II PD according to the Hoehn and Yahr classification system. Yip et al (2003) assessed FER (1) among patients with right-sided PD classified as stage I or II according to Hoehn and Yahr staging and (2) among patients with bilateral PD classified as stages II-V. Sprengelmeyer et al (2003) assessed FER (1) among patients in the early stages of the disease (mean score ± SD, 1.7 ± 0.5 on the Hoehn and Yahr scale) who had not yet received dopaminergic medication and (2) among patients in the more advanced stages (mean score ± SD, 2.6 ± 0.9 on the Hoehn and Yahr scale) under dopamine replacement therapy. Dujardin et al (2004) used a facial emotion rating task. The authors analyzed participants’ performance (1) in terms of the percentage of accurately identified expressions, where an expression was deemed to have been accurately identified if the emotion scale eliciting the highest intensity rating corresponded to the target emotion and (2) according to intensity scores. Pell and Leonard (2005) also used a facial emotion rating task. The authors investigated participants’ performance based on both (1) intensity ratings and (2) correlations between the intensity ratings that each patient assigned to a target face and the set of mean ratings assigned to that stimulus by HCs. This latter method did not allow the authors to run direct comparisons between groups but still highlighted deviations from normal patterns of sensitivity among patients for specific emotions.

^dSuzuki et al (2006) highlighted a specific FER deficit for disgust in PD during a facial emotion rating task, using a refined assessment method based on item response theory.

^eDelaveau et al (2009) conducted a study in which patients with PD and HCs were scanned both with and without levodopa medication. Regarding accuracy (correct responses), there were no differences between the groups (patients versus controls) or between pharmacological states (levodopa versus placebo) within these groups. The same data are presented in Delaveau et al (2010).

^fPaulmann and Pell (2010) highlighted a negative impact of PD on the recognition of emotions conveyed through different channels (lexical semantic/prosody/ facial expressions), either in isolation (unimodal) or in various combinations (bi- or multimodal emotion cues), with no significant effect of the communication channel. Martinez-Corral et al (2010) assessed FER among patients with PD (1) without and (2) with apathy.

^gBuxton et al (2012) examined FER abilities at 3 levels of difficulty (easy, moderate, and difficult). The authors highlighted deficits in the recognition of facial expressions of happiness at the moderate and difficult levels, disgust and surprise at the moderate level only, and sadness at the difficult level only. Patients had no difficulty identifying prototypical facial emotions (easy level).

^hVentura et al (2012) analyzed participants’ performance on an identification task and a discrimination task, based on a single composite score. In Garcia-Rodriguez et al (2012), the FER abilities of de novo patients with PD were tested under 2 conditions: (1) a simple identification task and (2) the same identification task concurrent with a secondary task (the Corsi Blocks tapping). Alonso-Recio et al (2014a) assessed FER abilities in PD during a one-back procedure: participants were required to indicate whether the current stimulus matched the one shown from one step earlier in a sequenced presentation of stimuli. They highlighted a deficit among the patients that was more pronounced in patients with higher disease severity according to the CISI-PD scale. In another study, Alonso-Recio et al (2014b) adapted a Stroop task to assess FER in PD taking into account inhibition abilities with an emotional version (ie, participants were required to identify the emotion portrayed on the presented face while ignoring the incongruent or congruent superimposed emotion category name) and a nonemotional version (traditional color-word Stroop task). They showed that patients were impaired in the emotional Stroop task but not in the traditional Stroop task; Likewise, to take into account the visual search abilities, the authors adapted the “face in the crowd” test with an emotional (ie, participants were required to decide whether the 24 faces showed the same emotion or whether any displayed a different one) and a nonemotional version. Here again, they showed that PD patients were impaired in the emotional version of the task but not in the classic version, the nonemotional task.

ⁱMarneweck and Hammond (2014) used 2 FER tasks in which participants were required to discriminate between neutral and angry faces. In one, they had to indicate whether the face expressed anger or neutrality by clicking the appropriate button. In the other, angry and neutral faces appeared successively on the screen, and participants were required to indicate the interval (first or second) containing the angry face. In both cases, the patients’ performance was lower than that of HCs. Marneweck et al (2014) used discrimination tasks in which participants were required to discriminate between (1) facial emotions and neutral faces, (2) facial expressions of the same emotion at different levels of intensity, and (3) 2 facial expressions of the same emotion and a different one. Wabnegger et al (2015) and Ille et al (2016) used a facial emotion rating task. They assessed participants’ performance (1) according to an index reflecting the response accuracy and (2) based on intensity ratings of the target emotion only.

^jLaskowska et al (2015) chose to use the Emotional Intelligence Scale-Faces (EIS-F), a more ecologically valid task featuring a mixture of basic and complex emotions (eg, tenderness, admiration, pride, disappointment, feeling of superiority, etc.). The authors based their analyses on signal detection theory, measuring decision-making strategy (response bias) and accuracy of stimulus detection (sensitivity) to determine whether the FER deficit in PD results from a decision-making impairment or from impaired sensory processes. In Lin et al (2016), 2 groups of patients were required to identify the valence (positive versus negative) of facial expressions: (1) patients with low motor dysfunction (<35 on UPDRS III; mean, 24; SD, mean, 48.73; SD, 14.58).

^kAs in Dujardin et al. (2004), Argaud et al (2016) used a facial emotion rating task and analyzed participants’ performance in terms of decoding accuracy, considering an expression to be accurately identified if the emotion scale receiving the highest intensity rating corresponded to the target emotion.

depended on the emotion, and (4) quantitative information about the number of experiments highlighting an FER deficit in PD for both overall performance and each specific emotion. Table 2 shows the different task types identified and which aspects of FER in PD are subject to a deficit. The quantitative summary in Table 1 should be considered with care, given that percentage rates reflecting the presence of a deficit for each emotion are based on the number of experiments in which authors examined FER as a function of the emotion displayed, when participants were exposed to that expression. Moreover, it is based on a qualitative review. See Gray and Tickle-Degnen¹² for an estimation of the deficit magnitude.

Impaired or Intact FER?

Although a nonnegligible number of authors failed to find any difference between patients and HCs, most of the studies investigating FER in PD highlighted lower performance among patients (Table 1). This is congruent with the meta-analysis by Gray and Tickle-Degnen.¹² Two years earlier, Clark and collaborators¹³ also underlined the deleterious impact of this deficit on patients' social relationships, highlighting a negative correlation between their FER difficulties and their level of interpersonal distress.

Is the Deficit Specific or General?

It is quite difficult to know if PD selectively impairs the recognition of specific emotions or leads to an overall deficit. Indeed, about 30% of the reviewed studies did not examine its effect on the recognition of specific emotions but calculated an overall score encompassing all the displayed emotions.^{4,14-29} Moreover, authors did not manipulate the same set of stimuli, and some only used a small subset.^{23,30-35} At last, some authors who explicitly investigated the recognition of specific emotions showed that although recognition was impaired for all the emotions they tested, some were more poorly recognized than others.^{24,32,34,36-39} Like Gray and Tickle-Degnen,¹² we noted that FER deficit in PD affected all the basic emotions but was greater for negative emotions (64% of studies highlighted a global deficit, 44% for anger, 27% for happiness; Table 1). This could echo the sub-cortical pathway involving the pulvinar, the amygdala, and the striatum, which may lead to a coarse but fast visual information processing.^{40,41} Indeed, because of its evolutionary relevance, this preserved route may induce a preattentive and autonomic bias toward threatening stimuli like angry faces.^{42,43} However, experimental data failed to fully support this "angry faces advantage" (whereby angry faces are detected more quickly than others). Some even supported the

TABLE 2. Description of the different types of tasks used in the literature and their conclusions regarding the presence of an FER deficit in PD

Task	Instructions	n	Deficit
ID	Identification task Participants were required to select the appropriate label for a given emotional expression.	53	39
DISCRI	Discrimination task Participants were required to determine whether the faces displayed simultaneously expressed the same or a different emotion.	16	9
MATCH	Matching task Participants were required to match a target facial expression with another facial expression and/or an affective prosodic sentence expressing the same emotion.	12	4
NAME	Naming task Participants were required to name the emotion displayed by the facial expression.	2	1
IMAG	Imagery task Participants were required to imagine a target facial emotion and to answer yes/no questions about the physical characteristics of that expression (eg, "Are the eyebrows drawn together?").	1	1
DESCRI	Description task Participants were required to answer yes/no questions about the physical characteristics of a displayed facial emotion (this task was designed as a perceptual control task for the imagery task).	2	1
INT	Intensity rating task Participants were required to assess the emotions portrayed and their intensity on visual analog scales ranging from "not at all" (ie, emotion absent from the expression) to "intensively expressed".	6	5
HEXA	Emotion hexagon task The emotion hexagon task is an identification task using morphed facial expressions that combines 2 closely related emotions (ie, the stimuli are morphed across a continua that lies around a hexagon: happiness-surprise-fear-sadness-disgust-anger-happiness).	3	1
SCREEN	Screening task Participants were required to press a button as soon as they perceived a target facial emotion.	1	0
EIS-F	Emotional Intelligence Scale- Faces Participants were required to indicate which emotions were expressed by a given facial expression and which were not, choosing between "shown", "not shown" and, as a last resort, "hard to say".	1	1

n: Number of studies that used this type of task out of a total of 97 reviewed experiments; Deficit: studies using the corresponding type of task highlighted a FER deficit in PD according to a global score.

reverse “happy faces advantage”⁴⁴ (see Ceiling Effect and Task Sensitivity section). In addition, there are several important caveats. First, these results were based on the subset of studies reporting participants’ performance as a function of emotion. Second, Gray and Tickle-Degnen¹² chose to pool data across modalities (face/voice). Third, we should bear in mind the greater diversity of negative as opposed to positive emotions in the literature. Surprise is ambiguous (pleasant, unpleasant, astonishment). Classifying it as a positive emotion is difficult without context. Therefore, of the 6 so-called basic emotions, there is only 1 prototypical and easily recognizable positive. Hence, the negative-emotion-specific FER deficit in PD is not so obvious. Future studies should shed light on this point by using a larger set of emotions including more than one positive affect.

What About the Errors Made?

Some authors conducted a deeper investigation of FER deficit in PD by examining the errors made. In an identification task, for instance, participants made an error if they did not select the appropriate label for a given expression. Instead of just counting this item as a misidentified expression, it can be quite more interesting to go further and examine the erroneous label. Did participants make an aberrant error (happiness versus sadness) or instead confuse 2 closely related emotions (surprise versus fear)? Only 5 studies reported this information.^{23,30,45-47} Three of them^{23,46,47} simply stated that patients and HCs made the same kind of errors. The two others^{30,45} statistically confirmed the presence of similar confusion patterns in PD patients and HCs. In Assogna et al,⁴⁵ patients mixed up negative emotions and assigned a neutral state to expressions they failed to recognize. In Argaud et al,³⁰ the most confusing emotion whatever the group was surprise.

Discrepancies in Results

Several confounding factors have been put forward to explain discrepancies in results. Some concern methodological aspects (study design, group size, instructions), stimuli (intensity, dynamism, emotion), task type and sensitivity, and analyses (categorical analyses based on rates of correct answers, continuous analyses based on intensity ratings, effect of emotion, covariables). Others could be linked to participants’ sociodemographic features (age, sex, personality) and/or patients’ clinical characteristics (disease severity, nonmotor symptoms, medication). In the following sections, we review evidence for the potential role of some of the most relevant factors when studying FER in PD.

Methodological Aspects

Stimuli

Although we are usually exposed to dynamic expressions in real-life situations, very few studies investigating FER in PD used dynamic stimuli.^{30,48-50} Because emotions are defined as transitory changes in several components of the organism, the dynamism of facial expressions plays a major role in emotion perception with a beneficial effect on FER accuracy.⁵¹ Moreover, neuroimaging studies have highlighted differential neural activity for static versus dynamic facial emotions, with higher activation in response to dynamic stimuli in regions processing socioemotional information, motion and faces and that belong to the mirror neuron system: superior temporal sulcus (STS), visual area, fusiform gyrus, inferior frontal gyrus, amygdala, and orbitofrontal cortex (OFC).⁵²⁻⁵⁴ However, only Kan et al⁴⁸ investigated the effect of stimuli dynamism on FER in PD. They found that patients’ performance clearly worsened when they had to recognize emotions from static rather than dynamic faces and concluded that this could artificially heighten the impact of PD on FER. In addition, the stimuli used in FER studies in PD generally depicted emotions at full-blown intensity. As emotional intensity has been shown to have a beneficial effect on FER,^{24,38,45} it is important to take this factor into account, especially knowing that patients could lose this positive effect.³²

Task Type and Difficulty

Task type variations across studies make consistent results less likely to emerge. In most cases, authors used identification tasks that require selecting the appropriate label for a given emotion. The remainder frequently chose discrimination/matching tasks that require judging whether or not 2 faces express the same emotion (Table 2). This may reflect different stages in the FER process.⁵⁵ Although both involve intact perceptual information processes occurring relatively early after stimulus onset, identification requires additional knowledge yielded by a categorization process. Thus, task type seems to determine task difficulty, and FER deficit in PD could be associated with a deficit in early perceptual and/or categorization processes. In the literature, identification tasks more frequently highlighted an FER deficit in PD (Table 2). However, Gray and Tickle-Degnen¹² showed a greater FER deficit during discrimination tasks. Furthermore, the level of difficulty can vary within the task. The forced-choice procedure of Cohen et al¹⁹ only featured 2 emotional labels, even though participants were exposed to 5 facial emotions. This considerably increased the probability of giving a correct answer by chance and then could explain the absence of a significant difference between patients and HCs. Last, the

above-mentioned problem of using prototypical stimuli and the differing levels of difficulty across emotions could result in a ceiling effect, thus biasing results (see below).

Ceiling Effect and Task Sensitivity

A lack of task sensitivity may have meant that significant differences between patients and HCs went unnoticed. In some studies, scores were very close to maximum, which suggests a ceiling effect that would have concealed any deficit.^{20,23,31,56,57} This could also induce a bias in favour of an emotion-specific deficit, especially when happiness recognition was compared with that of other emotions. Indeed, happy expressions are recognized more quickly and accurately than others. This could be related to its most distinctive configuration with a very salient feature, the smile, whereas other facial expressions show more overlapping, less distinctive features.⁵⁸⁻⁶³ Thus, it is not surprising that a specific FER deficit for negative emotions emerges, whereas happiness recognition elicits higher accuracy scores.^{35,47,48,64-68} One solution to avoid this bias would be to combine different task types within the same study^{15-17,23,24,28,36,37,56,69} or use more refined assessment methods.^{22,70} Alternatively, FER could be assessed in more detail using rating tasks in which participants assess emotions and quantify their intensity. More specifically, they rate a target expression on a set of emotional visual analog scales (VAS). Few studies used rating tasks,^{30,32,33,56,70-72} but all except one⁷¹ highlighted an FER deficit among patients. However, when intensity ratings were dichotomized as correct (when the scale corresponding to the target emotion had the highest intensity rating) versus incorrect, the deficit was not always reported.^{33,72} Surprisingly, only Dujardin et al³² analyzed response patterns according to the intensity scores on each VAS. They showed that patients rated the target emotion lower than HCs did and systematically rated surprise higher, whatever the emotion displayed. Thus, using VAS allows for a deeper analysis. Although this methodology is still rare in studies investigating FER in PD, it has been successfully used to characterize emotional bias in schizophrenia and depression when no impairment emerged from categorical judgments based solely on response accuracy.^{73,74}

Clinical Factors

Patients' characteristics varied significantly across and within studies. Some enrolled patients who had recently been diagnosed and were not yet receiving medication, patients under dopamine replacement therapy (DRT) with a more severe disease including nonmotor symptoms, or patients who had temporarily interrupted their treatment. Thus, disease duration and severity, medication,

cognitive/visuospatial impairments, and mood disorders are (quite interrelated) factors that could contribute to FER impairment in PD.

Disease Severity and Facial Hypomimia

Although an FER deficit has been highlighted in the early stages of the disease,^{21,32,66,69,70,75} it was greater in the most severely affected patients.^{15,28,29,36,38,68,76} That said, when the link between deficit magnitude and disease severity was investigated with correlations, results differed. Based on these statistics, Gray and Tickle-Degnen¹² suggested that the level of FER deficit is unrelated to the level of motor disability reflected by Hoehn and Yahr staging.⁷⁷ However, the average patients included in their meta-analysis exhibited mild to moderate motor disability. Their conclusions would have been different in more severe cases. Even when disease severity was measured with the Unified Parkinson's Disease Rating Scale, regarded as more sensitive than Hoehn and Yahr staging,^{78,79} results still diverged but seemed to be in favour of a positive correlation between disease severity and FER deficit.^{24,64,65,67,80,81} PD progression is very heterogeneous and depends on several factors. Patients with the same disease duration/severity may have different patterns of neuronal loss in striatal, limbic, and cortical regions.^{82,83} Thus, to examine the link between disease severity and FER deficit in PD, it might be useful to employ more sensitive markers (neuroimaging) of disease progression or subtype.⁸⁴ Furthermore, FER deficit could also be linked to motor asymmetries in PD, but the question of whether patients with left-dominant motor symptoms (LPD) showing relatively greater neural degeneration in the right hemisphere have a more severe deficit than patients with right-dominant motor symptoms remains open. LPD patients could be more likely to show FER impairments considering the relatively greater role of the right hemisphere in FER (at least for anger, fear, and sadness) and the prominence of visuospatial deficits in LPD patients.^{85,86} Last, since the earliest studies, emotional disorders in PD concern both expression and recognition, and some authors have even reported positive correlations between facial expression and FER impairment.^{17,37,87-89} These findings are in line with a peripheral component of the FER deficit arising from facial hypomimia in PD. This assumption is further developed in the Discussion section (see New Hypotheses Based on Embodied Simulation section).

Visuospatial Deficits and Other Cognitive Symptoms

From the earliest studies, authors controlled for visual functions with tasks such as the Benton Facial Recognition Test (BFRT).⁹⁰ Some found FER impairment among patients with no deficit in neutral faces recognition,^{15,16,23,29,68,69,76,91} whereas others showed

that face-processing impairment co-occurs with the deficit.^{24,46} Gray and Tickle-Degnen¹² did not report any significant differences in FER abilities between patients who performed normally on tests like BFRT and patients with abnormal performance. They concluded that FER deficit in PD exists beyond a general deficit in face processing. However, the BFRT may not be sensitive enough to highlight a deficit.³⁸ Based on more accurate measurements of visuospatial abilities including low-level visual functions (contrast sensitivity), some authors suggested that FER deficit could be related to visuospatial impairments in PD.^{28,38,47,75,92} For instance, showing that the ability to discriminate graded intensities of angry faces was positively correlated with the ability to discriminate unperfect/perfect circles (radial frequency patterns), Marneweck and Hammon indicated that impaired ability to perceive visual forms could contribute to FER deficit in PD.³⁸ It is noteworthy that visual and emotional systems are extensively interconnected; for example, the amygdala is connected to the superior colliculus via the pulvinar, to the OFC, and the anterior cingulate cortex, as well as to cortical visual regions in the temporal cortex.⁴¹ Furthermore, the idea that FER deficit in PD could be secondary to executive dysfunction is an old one. Working memory impairment could influence FER as the ability to manage, maintain, and operate with present and stored information is affected (particularly true for sequentially presented stimuli²⁹). Likewise, divided and selective attention is impaired in PD.⁹³ Thus, FER deficit in PD could be linked to attentional difficulty to process different sources of information at the same time. As authors who assessed cognitive abilities used different tests measuring different aspects of executive function, no consistent conclusion could be drawn regarding the link between cognitive impairment and FER in PD.^{21,32,36,45,48,56,94} In recent studies,^{21,22,26,29,45,95} however, an FER deficit was confirmed among patients after controlling for cognitive symptoms (including working memory and attention processes) but seemed to be influenced by their magnitude.

Mood Disorders

As emotional processes are disrupted in depression^{96,97} and knowing the high incidence of depression in PD,^{7,98} it has been suggested that FER deficit is not specific to PD but is linked to mood disorders. The studies we reviewed usually controlled for this aspect by selecting patients with normal depression scores and/or directly testing the effect of depression on performance. Here again, results were heterogeneous. When they compared the performance of relatively depressed patients with those with normal scores and assessed correlations between FER performance and depression score, Gray and Tickle-Degnen¹² concluded that FER deficit is not secondary to depression in PD.

Since 2010, only Baggio et al⁶⁴ and Linn et al⁷⁶ have reported a link between depression and FER in PD. Concomitant with depression, apathy and anxiety are also frequent in PD.^{3,99} However, these mood disorders have been taken into account less often, although some studies have highlighted their influence on FER (anxiety: Clark et al¹³ and Ille et al³³; apathy: Robert et al¹⁰⁰ and Martínez-Coral et al²⁵).

Dopamine Replacement Therapy

Some authors who assessed FER in patients who were not yet receiving medication or had temporarily withdrawn from DRT highlighted a deficit in the absence of treatment.^{32-34,94} Others failed to find any difference between patients without DRT and HCs.^{18,49} Only Sprengelmeyer et al⁶⁸ directly compared medicated and unmedicated patients on FER. They reported a deficit in patients whatever the treatment condition, but found a greater one among unmedicated patients. This is consistent with the positive correlation between patients' performance and daily levodopa-equivalent dose (LED) found by Assogna et al.⁴⁵ However, no other authors who looked for such correlations found this result.^{19,28,38,45,64,65,67,75} Otherwise, several studies have reported a beneficial effect of DRT on FER in PD. In Tessitore et al,¹⁰¹ both patients with and without DRT showed reduced amygdalar activation during angry and fearful faces processing. However, as evidenced by increased blood-oxygen-level dependent (BOLD) responses in the drug-on relative to the drug-off state, dopamine repletion partially restored the amygdala response. Similarly, Delaveau et al¹⁰² highlighted restoration of default mode network deactivation after levodopa administration in PD (posterior cingulate and lateral temporal cortices). In HCs, reduced activation of the mesocorticolimbic regions involved in emotional processing (with difficulties in recognizing anger¹⁰³) followed the administration of a dopamine antagonist.^{103,104} Conversely, Delaveau et al^{105,106} reported reduced amygdala activity after levodopa administration in HCs and patients. These results might appear contradictory, with DRT having a beneficial effect on FER in some cases and a detrimental one in others, but it would depend on disease progression. In the early stages, mesocorticolimbic pathways would be relatively spared compared with the motor pathway.^{82,107} Thus, the dose of levodopa needed to improve motor symptoms may simultaneously overdose mesolimbic projections to the amygdala. In more severe stages, DRT may have a beneficial effect, compensating for dopamine depletion, and blocking the responses of mesocorticolimbic structures in HCs by administering dopamine antagonists may mimic disease progression. The DRT overdose effect has already been highlighted in cognitive functions.¹⁰⁸ Regarding emotion recognition, only 1 study specifically examined the impact of

DRT in early PD¹⁰⁹ and found more pronounced difficulty recognizing emotional prosody among early patients under DRT versus the drug-off condition.

Discussion

A schematic diagram summarizing the review is shown in Figure 1.

What Can We Learn From This Review?

Recognition of the 6 basic facial emotions is impaired in PD, but the deficit seems to be greater for negative emotions. Because of high variability in results, several potential confounding factors need to be considered. Although some of them were highlighted by Assogna et al¹¹ and investigated by Gray and Tickle-Degnen,¹² no reliable conclusions could be drawn as to the effects of disease severity, depression, or cognitive/visuospatial symptoms or the impact of dopaminergic medication. Moreover, neither anxiety nor apathy was analyzed. Our review indicates that FER deficit in PD (1) depends on disease severity, while observable from disease onset; (2) is linked to visuospatial disturbances, notably low-level visual dysfunction; (3) is more pronounced in patients with cognitive impairment; (4) is not restricted to depression but seems to increase with the magnitude of mood

disorders including anxiety and apathy; and (5) fluctuates according to DRT, with an overdose effect in the early stages and a beneficial effect as PD progresses and treatment compensates for dopamine depletion.

One important aspect underlined is the potential lack of task sensitivity when assessing FER and other forms of processing. Some studies that failed to find behavioral differences nevertheless observed deficits at the cerebral level.^{57,72,81,101} Wieser et al⁸¹ reported diminished early visual discrimination of facial emotions among patients at the electrocortical level, but no differences in affective ratings or recognition accuracy. Here, behavioral assessments were not sufficiently sensitive to detect subtle impairments already present at the neural level. Intensity rating tasks offer an alternative method for accurately studying FER in PD. Analyses of response patterns distinguishing between target and nontarget VAS allow both FER accuracy (qualitative information) and emotion discrimination (quantitative information) to be appraised. So far, however, only Dujardin et al³² have assessed FER among patients using intensity ratings per se. They reported a weaker level of emotion discrimination in PD, leading to noisy responses, with lower ratings on target scales and higher ratings on nontarget scales. In addition, such more refined methodologies should be used to avoid a potential ceiling effect. In the same vein, future studies should use dynamic

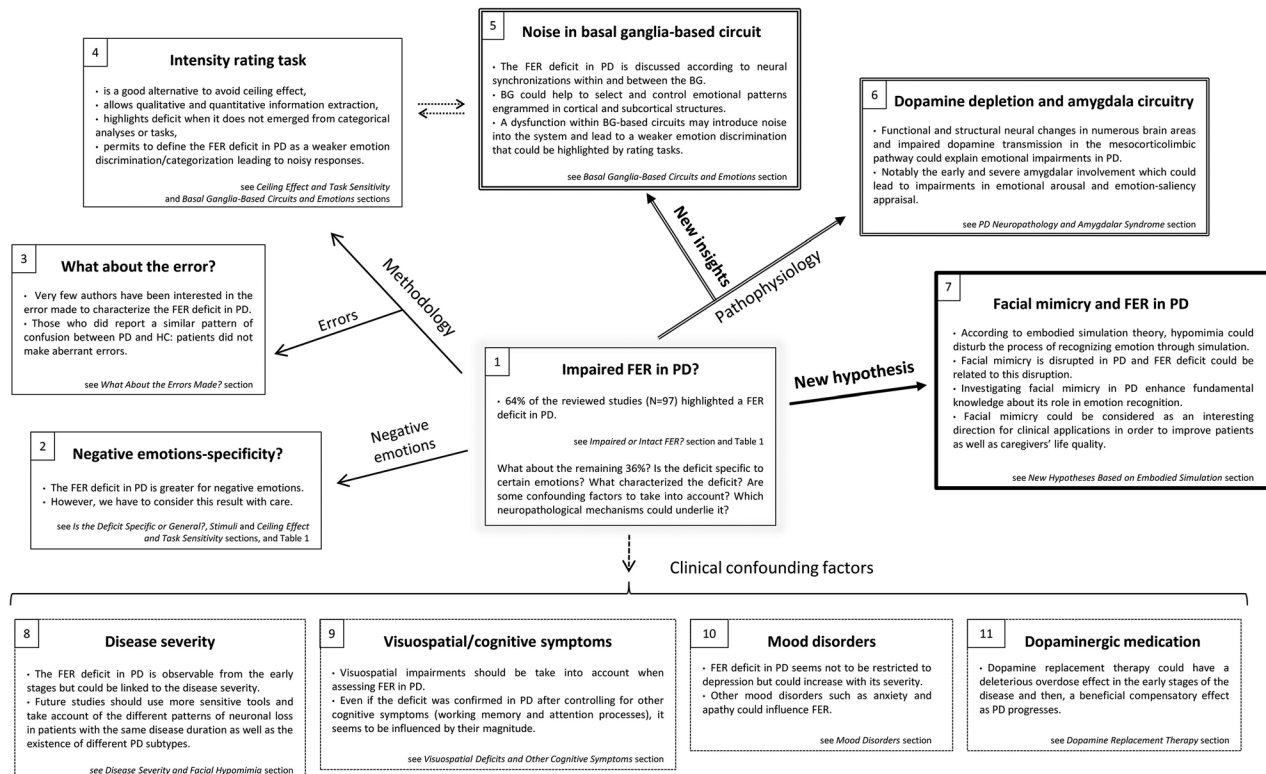


FIG. 1. Facial emotion recognition in Parkinson’s disease: review and discussion. (1) level of impairment, (2) emotions-specificity, (3–4) methodology, (5–6) underlying pathophysiological mechanisms with (5) recent considerations about basal ganglia-based circuits in emotions, (7) new hypothesis related to hypomimia, (8–11) potential clinical confounding factors.

stimuli portraying more subtle/complex emotions such as tenderness, disappointment^{22,34} (with more than one positive emotion) to investigate the deficit consequences in conditions closer to everyday reality.

Furthermore, this review encourages future studies to adopt a precautionary approach as far as possible avoiding the effects of clinical confounding factors and/or evaluating their impact on FER. Although it could increase study duration, patients should undergo an extensive neuropsychological/psychiatric interview investigating numerous aspects of cognitive functioning and mood-related features. Thus, patients with mood and/or cognitive disorders could be excluded or test results could be correlated with FER outcomes. Likewise, knowing the clear influence of DRT on FER, future studies should take into account the LED calculated for each patient according to common guidelines¹¹⁰ or enrolled patients not receiving medication yet or assessed off medication. At last, as far as disease duration/severity is concerned, more sensitive markers should be privileged. Future studies will surely favour neuroimaging techniques rather than only clinical scales that despite scoring symptoms severity, do not convey much information about disease progression or subtype.

PD Neuropathology and Amygdalar Syndrome

Neural changes in numerous areas and impaired dopamine transmission in the mesocorticolimbic pathway are invoked to explain FER deficit in PD. Indeed, not only putaminal but also orbitofrontal and amygdalar presynaptic dopaminergic functions were altered in early PD.¹¹¹ In addition, brain responses recordings during FER tasks have revealed decreases in striatal, amygdalar, and orbitofrontal activity and lower activation in analytic temporal facial recognition areas (STS and fusiform gyrus).^{57,72,101,112} Lotze et al¹¹² have shown that the less dopamine transporter availability (DAT) present in the putamen, the lower the ventrolateral prefrontal cortex activation in response to emotional gestures and highlighted a positive correlation between the putaminal DAT reduction and the number of errors in emotional gestures recognition. Similarly, morphometry analyses reported decreased gray-matter volume in numerous limbic, paralimbic, and neocortical associative temporo-occipital areas^{64,66,113,114} and showed that the atrophy could be associated with the deficit, as gray-matter volume in these regions correlated with patients' FER performance.^{64,66} More specifically, impairment of the amygdala, observed since the early stages and worsening with disease progression,^{107,115,116} has often been invoked to explain emotional deficiencies in PD, as we know that the amygdala circuitry is involved in multiple behavioral functions including emotional arousal and emotion-saliency appraisal.^{117,118} In their recent

review, Diederich et al¹¹⁹ clearly reported the behavioral consequences of amygdalar dysfunction in PD and depicted this amygdalar syndrome as both a "failing doorman" who struggles to identify emotional contents of sensory inputs and a "failing disk jockey" who cannot orchestrate emotional outputs adequately anymore. They also specified that dopamine could induce hyperactivity of the amygdala, a finding that fits with the hypothesis of an overdose effect on FER in early PD and that compensatory mechanisms could occur as well (see New Hypotheses Based on Embodied Simulation section).

Basal Ganglia-Based Circuits and Emotions

FER deficit in PD could also be discussed in light of neural synchronizations within and between the basal ganglia (BG).¹²⁰ The BG may be involved in emotion processing in the same way they are in motor and cognitive functions. Cortico-BG-thalamocortical loops may inhibit nonrelevant information (nontarget emotions/related facial features) and activate relevant ones (target emotions/related facial features) just as they select a specific movement by inhibiting competing programs and disinhibiting the selected one.¹²¹ Moreover, BG-based circuits are involved in automated *chunked* representations of action/cognitive sequences and contribute to the suppression of goal-directed behavioral control when it becomes habitual.¹²² The progressive loss of rapid habitual processing and the replacement of automatic control by effortful processing may make PD patients more vulnerable to interference and lead to difficulty in performing even well-known procedures. The BG may perform similarly when it comes to processing emotional information efficiently. Regarding FER, the BG might help to select and control emotional patterns engrammed in cortical and subcortical structures (face fusiform area for face perception, amygdala for relevance detection, OFC for evaluation-driven emotion processing) by inhibiting competing patterns and coordinating the whole process.¹²⁰ Studies investigating subthalamic nucleus deep-brain stimulation in PD suggest that the BG recruit and synchronize the activity of the face fusiform area, amygdala, and OFC.^{123,124} Dysfunction within BG-based circuits may therefore introduce noise into the system, disrupt the synchronization process, and lead to biased emotional judgments characterized by a weaker emotion discrimination (noisy responses) that could be highlighted by rating tasks.

New Hypotheses Based on Embodied Simulation

The link between facial expression and FER impairment highlighted since the earliest studies on FER in PD^{17,37,87} recall embodied simulation theory, suggesting that disturbed motor processing can lead to

emotion recognition deficiency. According to this theory, emotion recognition is facilitated by internally generated somatosensory representations triggered by the simulation of a perceived facial expression that partially activates the corresponding emotional state in the observer.^{55,125-127} This is subtended by somatosensory-related cortices and could be linked to facial mimicry, the tendency to replicate others' facial expressions.^{128,129} Hence, in HCs, facial electromyography (EMG) could highlight congruent facial muscle responses to facial expressions, which could foster emotion recognition.¹³⁰⁻¹³² However, one of the most frequent and distinctive Parkinsonian motor symptoms is hypomimia.¹ Thus, in addition to central disorders, emotional symptoms in PD may be induced by peripheral dysfunction associated with impaired facial mimicry. To our knowledge, only 2 studies tested this hypothesis.^{30,133} By recording EMG responses during an FER task, authors showed that FER impairment was accompanied by disruption of facial mimicry in PD. During the experiment, facial expressions were mimicked as expected, but emotion-specific EMG variations were disturbed among patients with weaker-than-normal corrugator and medial frontalis reactions in response to angry and sad faces respectively, and almost no reactions from the orbicularis and the zygomaticus in response to happy faces. These facial reactions could be linked to emotion-decoding accuracy and response time: the weaker the responses, the lower the performance was. Neuroimaging and electrophysiological studies bring some very interesting elements here, suggesting a rearranging of the brain mechanisms underlying FER in PD.^{57,72,112,134} Yoshimura et al⁵⁷ have shown that event-related potential recordings in response to fearful faces were generated within the parietal somatosensory cortex among patients instead of the amygdala and the visual temporal cortex as in HCs. Similarly, in accordance with Anders et al,¹³⁴ who highlighted a ventrolateral premotor cortex compensatory activity during emotions processing in Parkin mutation carriers, Wabnegger et al⁷² showed a stronger activation in somatosensory regions among patients that was positively correlated with their recognition performance. The somatosensory recruitment could be considered a compensatory mechanism following dopamine depletion and/or pathological changes in the amygdala, which may overcome emotional deficits in PD. Finally, those results raise the question whether facial mimicry and its feedback to neural systems are a necessary part of the process of recognizing emotion through simulation. An alternative assumption stipulates that emotion recognition could only lie on sensorimotor simulation without measurable facial mimicry.¹²⁷ In this view, FER impairment in PD could stem not from a disturbance of facial mimicry but from incongruent feedback that

comes into conflict with the internal simulation of the observed facial expression. In any case, facial mimicry may not be mandatory but could constitute an interesting therapeutic lever to counteract FER deficit in PD when patients are asked to consciously imitate to accurately recognize.

Conclusion

Hypomimia has considerable repercussions on patients' (often perceived as bored, anxious or cranky¹³⁵⁻¹³⁷) and relatives' quality of life, damaging interpersonal relationships and gradually increasing social isolation.^{1,138} Patients, caregivers, and clinicians can break this vicious cycle. It starts with awareness of the patients' difficulties in decoding, expressing, and mimicking emotions, along with their attendant social consequences. Research on these issues could also improve medical management, as therapeutic strategies can be adapted to patients' symptoms, especially knowing that there are several PD subtypes with 3 separate phenotypes: mainly motor/slow progression, intermediate, and diffuse/malignant.⁸⁴ Patients with the latter are more likely to exhibit nonmotor symptoms, including cognitive and mood disorders, but patients with the main motor form may also develop emotional impairments as a consequence of impaired facial mimicry. Finally, these findings open up a new line of inquiry into patients' masked face and its impact on socioemotional communication among both patients and their caregivers. ■

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