



# How Parkinson's Disease Affects Non-verbal Communication and Language Processing

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## Abstract

In addition to difficulties that affect movement, many adults with Parkinson's disease (PD) experience changes that negatively impact on receptive aspects of their communication. For example, some PD patients have difficulties processing non-verbal expressions (facial expressions, voice tone) and many are less sensitive to 'non-literal' or pragmatic meanings of language, at least under certain conditions. This chapter outlines how PD can affect the comprehension of language and non-verbal expressions and considers how these changes are related to concurrent alterations in cognition (e.g., executive functions, working memory) and motor signs associated with the disease. Our summary underscores that the progressive course of PD can interrupt a number of functional systems that support cognition and receptive language, and in different ways, leading to both primary and secondary impairments of the systems that support linguistic and non-verbal communication.

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## Introduction

Knowledge of Parkinson's disease (PD) and its effects on cognition, language, and other aspects of communication have improved significantly since 1817 when James Parkinson first described the disease. Parkinson's disease is principally a disease of the elderly; it is a chronic, neurodegenerative disorder caused by progressive depletion of brain dopamine production in the basal ganglia, a set of subcortical structures with extensive connections to other parts of the brain (such as the prefrontal cortex and the limbic system). According to a report of the World Health Organization, an estimated four million people are living with PD, with 305,000 new cases diagnosed each year (WHO 1997). Most people recognize primary Parkinsonism, or *idiopathic* PD, by four cardinal motor signs: muscle rigidity, resting tremor, bradykinesia, and postural instability. However, new research tells us that many additional symptoms, motor and non-motor, typically emerge during the progression of the disease, although these symptoms tend to vary in nature and severity from one patient to another. In the case of motor symptoms, for example, some PD patients will show signs of dystonia or drooling, whereas others may present with speech and swallowing disturbances. A

wide range of non-motor deficits can also appear in the course of PD; these include mood alterations, such as depression, sleeping problems, and cognitive changes that can sometimes lead to a full-blown dementia (Caballol et al. 2007).

Even in PD patients who do not show evidence of broad intellectual decline or dementia, the majority of these individuals experience cognitive impairments that affect organization and planning ('executive functions') and/or working memory capacity (Taylor et al. 1986; Brown and Marsden 1991; Gabrieli et al. 1996; Lewis et al. 2003). While some studies suggest that the presence of executive dysfunctions in PD predicts which patients will later develop dementia (Levy et al. 2002), the idea that specific cognitive deficits observed in PD reflect a pre-dementia stage is far from being accepted (see Williams-Gray et al. 2007 for recent data and a discussion). It appears more certain that the pathophysiological changes that occur in PD progressively interfere with a variety of functional brain systems that support motor-related activities as well as critical aspects of cognition, emotion, and language processing, which is the focus of this chapter.

Based on anatomical and physiological evidence, we know that the striatal nuclei within the basal ganglia (caudate nucleus and putamen) receive projections from many cortical areas of the brain; these structures play a critical role in a set of functionally segregated fronto-striatal circuits (or 'loops') that act on motor and cognitive information (Alexander et al. 1986; Tisch et al. 2004). While it can be said that many of the clinical signs observed in the early course of PD are functionally related to dopamine-deficiency within the basal ganglia (e.g., cardinal motor signs), as the disease evolves these chemical changes have progressive and negative influences on the operation of the corticostriatal networks. These changes may explain why a broader array and distinct types of impairments are usually seen in more advanced stages of PD (Cools 2006).

Thus, given the strategic importance of the striatum and its connectivity with cortical (especially frontal and limbic) sites, many functions related to language and non-verbal communication may be considered vulnerable in the course of PD. In broad terms, auditory language processing is thought to engage a bilateral temporo-frontal network (Friederici 2002; Friederici and Alter 2004) and sentence processing is modulated in part by the fronto-striatal circuitry/basal ganglia during certain tasks (Friederici et al. 2003; Kotz et al. 2003). Similarly, the processing of non-verbal emotion cues, such as facial expressions or prosody ('speech melody'), relies on a distributed (partly right hemisphere dominant) network of brain structures that includes the superior temporal and inferior frontal cortices, amygdala, and the basal ganglia among other structures (Adolphs 2002; Schirmer and Kotz 2006). Given the pathophysiology of PD which affects major brain regions recruited during verbal and non-verbal tasks (e.g., the frontal lobes and basal ganglia), it is perhaps not surprising that many functions related to language and non-verbal processing decline in the context of PD.

As we attempt to summarize below, there is now indisputable evidence that PD patients in the early stages of the disease and without dementia have notable difficulties with language processing (e.g., to understand non-literal or implied meanings) and in the processing of non-verbal cues (e.g., facial expressions, emotional speech tone), which have a negative impact on the psychosocial abilities of those afflicted with the disease. Given the emphasis that many patients and their care-givers place on understanding motor components of the disease, the idea that PD is characterized by problems that affect cognition and receptive communication may initially come as a surprise; it is also likely that deficits in language processing and non-verbal communication are frequently under-diagnosed in PD or not consciously understood by those affected, which is one of the potential uses of this report.

We begin our summary by reviewing what is known about (auditory) language processing deficits in non-demented adults with PD, followed by an overview of non-verbal deficits that can co-occur in many individuals with PD. A concluding section will attempt to integrate knowledge from these different sources to establish a platform for further investigations into the nature of verbal and non-verbal processing impairments associated with PD.

### *How Parkinson's Disease Affects Language Processing*

It is long recognized that motor limitations in PD affect how well-affected individuals express themselves through speech, yielding a characteristic form of dysarthria (Critchley 1981). However, until recently it was widely believed that disorders of language comprehension were rare in patients with PD (see Cummings et al. 1988). The main language-related disturbance identified as common to this population involved difficulties with speech production; principally, naming (Huber et al. 1989; Cotelli et al. 2007) and verbal fluency (see Henry and Crawford 2004 for a recent meta-analysis on verbal fluency skills in PD). This narrow view is dispelled by recent studies that clearly associate PD with different forms of receptive language impairment (Berg et al. 2003, Grossman et al. 2003; Monetta and Pell 2007). For some researchers, the observation that language can be affected by PD has led them to evaluate PD patients, in part, as a means for understanding how the (intact) basal ganglia contribute to language processing and social cognition (Ullman et al. 1997; Friederici et al. 2003; Grossman et al. 2003; Kotz et al. 2003; Pell and Leonard 2003; Tettamanti et al. 2005). However, since neuropathological alterations in PD progress beyond the basal ganglia to affect the frontal lobe and fronto-striatal pathways (among other structures), many other researchers have investigated language processing deficits in the context of typical 'frontal lobe' deficits exhibited by PD patients, such as limitations in executive resources or working memory (see Grossman et al. 2003 for a general review on this topic).

Interestingly, findings to date imply that language comprehension in PD deteriorates most when patients are required to process sentences with

non-canonical (i.e., irregular or non-transparent) grammatical structures. Moreover, difficulties in 'pragmatic' language processing, that is, to process the intended or indirect meanings of language in context, tend to be most pronounced in complex social situations or when the individual patient's cognitive resource capacity is severely taxed (McNamara and Durso 2003; Monetta and Pell 2007). These observations have forced researchers to consider whether verbal language processing deficits in PD typically appear only in patients who show functionally related problems in executive control and working memory, which is a central topic of debate in the current literature. Stated another way, do many language processing difficulties in PD reflect a secondary consequence of more basic limitations in cognitive resource capacity, such as working memory needed to support certain language functions? Studies that inform this and related questions about the nature of language processing deficits in PD will be examined first, with a specific focus on sentence comprehension and pragmatic language processing skills.

#### SENTENCE COMPREHENSION DEFICITS IN PARKINSON'S DISEASE

The ability to process meaning from sentences revolves heavily around the shared syntactic competence of the speaker–listener; in gross terms, listeners must activate semantic knowledge about words and use word order and other syntactic relations to understand the meaning of a sentence. In the context of PD, it has generally been found that (automatic) semantic activation of words is intact in patients in early stages of the disease (see Copland 2003; Filoteo et al. 2003). Other studies have reported that lexical–semantic processing is abnormal in PD patients during sentence comprehension tasks (Lieberman et al. 1990; Grossman et al. 1991, 2002; Angwin et al. 2005). For example, it has been suggested that there is a delayed time course of semantic activation in participants with PD, which affects their sentence comprehension in a negative manner (Arnott et al. 2001; Grossman et al. 2002; Angwin et al. 2004a,b). Impairments in semantic knowledge appear to correlate with executive dysfunctions in some PD patients (see Portin et al. 2000). However, while these studies demonstrate that PD is likely associated with difficulties in sentence comprehension, little consensus has been reached about the exact origin of these semantic deficits (see Grossman et al. 2002).

Some researchers have argued that impairments in sentence comprehension are related to a specific grammatical or syntactic deficit produced by PD (Lieberman et al. 1992; Natsopoulos et al. 1993; Cohen et al. 1994). Lieberman and colleagues (1992) have shown, using the Rhode Island Test of Sentence Comprehension, that PD patients display longer response times and more errors in the comprehension of certain syntactic structures, suggesting that the basal ganglia help to regulate syntactic abilities. Alternatively, other researchers have pursued the hypothesis that sentence comprehension deficits in PD do not reflect difficulties with syntax, but rather,

emerge selectively in situations of limited cognitive resources to apply to sentence processing (Geyer and Grossman 1994; Waters and Caplan 1997; Kemmerer 1999). Of note, several studies that have examined syntactic comprehension in PD report that PD patients show greater comprehension difficulties when faced with syntactically complex sentences (Grossman et al. 1991, 1992; Natsopoulos et al. 1993). Based on these and other findings, Grossman and colleagues postulated that impaired sentence processing in PD is caused by underlying cognitive deficits, such as a deficient attentional system or slowed lexical processing (see Grossman et al. 2002, 2003). For example, in a functional magnetic resonance imaging study of sentence comprehension abilities, Grossman and colleagues showed that PD patients exhibit decreased activations in left frontal, right posterolateral temporal, and striatal regions that are associated with cognitive resources (see Grossman et al. 2003 for details). They associated these results with the interruption of a large scale network important for cognitive resources that can interfere with sentence processing. This argument is consistent with the idea that verbal language deficits in PD are secondary to more general deficits in cognitive resource capacity, which often occurs in many individuals with PD.

Along these lines, Friederici and her colleagues have investigated sentence processing in PD participants by measuring event-related potentials (ERPs, Friederici et al. 2003; Kotz et al. 2003). They suggest that observed differences in language processing abilities across PD studies may lie in the distinction between early, automatic structure-building syntactic processes and later, more conscious syntactic processes, such as the generation of different verb forms (Ullman et al. 1997), sentence-judgment and question-answering tasks (Grossman et al. 1991, 1993), and the comprehension of sentences demanding syntactic working memory resources (Natsopoulos et al. 1993). Friederici et al. (2003) argue that early automatic processes are intact in PD, whereas later integrational processes are more likely to be impaired, suggesting that the basal ganglia do not support early automatic processes during comprehension, but are implicated in the process of syntactic integration. Also, it has been shown that the P600 effect – an effect linked by many to processes of syntactic re-analysis and lexical-semantic integration – tends to be reduced in PD patients in a sentence processing task involving verb-argument structure violations (Kotz et al. 2003). These data supply further indications that only controlled aspects of syntactic processing are likely affected by basal ganglia dysfunction in PD patients (see also Friederici et al. 1999 for related data on patients with focal basal ganglia lesions). While these findings are being subjected to further study, they underscore ways that functional dysregulation of the basal ganglia may have a direct or primary impact on components of the language processing system in the context of PD.

In summary, it seems clear that sentence comprehension abilities are susceptible to the effects of PD under certain conditions, although no clear consensus about the origin of these deficits has been reached. The potentially

related observations that ‘automatic’ processes involved in sentence processing are largely retained in PD, and that limited cognitive resources such as attention tend to exacerbate sentence comprehension, represent the most promising avenues to explore in future research. These deficits may also bear a relationship to some of the difficulties experienced by PD patients in tasks of pragmatic language processing as will be discussed next.

#### PRAGMATIC LANGUAGE DEFICITS IN PARKINSON’S DISEASE

In addition to studies that have focused on sentence processing (i.e., syntactic), related investigations have explored whether PD patients encounter problems when interpreting the intended or ‘pragmatically-appropriate’ meanings of language as defined by its social context. Common situations that require sensitivity to pragmatic features of language include the processing of metaphorical expressions, paralinguistic behaviors, and discourse (Natsopoulos et al. 1991a,b, 1993, 1997; Berg et al. 2003; McNamara and Durso 2003; Monetta and Pell 2007).

In one study that conducted a broad survey of pragmatic language abilities in PD, Berg et al. (2003) concluded that PD patients exhibit ‘high-level’ language difficulties that affect the ability to generate inferences, to recreate sentences, and to comprehend metaphors and lexically ambiguous words. McNamara and Durso (2003) also evaluated PD patients using a general pragmatic communication skills protocol (see Natsopoulos et al. 1997); this battery examined the patients’ conversational and social skills with respect to verbal behaviors (e.g., speech acts, message specificity), non-verbal behaviors (e.g., facial expressions, gestures), and paralinguistic behaviors (e.g., fluency, prosody). Their results again demonstrated that pragmatic communication skills were impaired in the PD group under study, and interestingly, there was a significant relationship between the communication deficits observed and impairments on traditional tasks of ‘frontal lobe’ functioning for these patients (e.g., Tower of London, Stroop task). These findings suggest that efficient pragmatic language processing is dependent on intact cognitive functions that are often impaired in PD patients (i.e., executive functions or cognitive resources), as was also suggested by researchers who investigated ‘complex’ forms of sentence processing in PD patients (Grossman et al. 2003).

To expand on these observations, we recently tested a group of 17 PD patients using a more detailed task of understanding metaphorical language – the timed property-verification task described by Gernsbacher et al. (2001). Specifically, we tested whether there is a relationship between measures of metaphor comprehension and working memory capacity in the context of PD by evaluating patient subgroups who had normal or impaired (verbal) working memory. Our results showed that PD participants with unimpaired working memory processed metaphors, such as, ‘*That baby’s cheeks are roses*’, in a manner similar to healthy control participants, whereas PD patients with impaired working memory were notably slower and less accurate in their

responses to these metaphorical expressions (Monetta and Pell 2007). Our data appear to confirm that many pragmatic forms of language processing are susceptible to underlying deficits in working memory as suggested in related studies of pragmatic and complex sentence processing noted above.

Another pragmatic skill involves the ability to comprehend implied information from a discourse context. Using the Discourse Comprehension Task (Brookshire and Nicholas 1993), Murray and Stout (1999) tested how nine PD patients answer questions that probed their comprehension of main ideas versus implied information after listening to different stories. The authors found that PD patients responded more accurately to questions probing main ideas than to questions about story details, and that the patients were more accurate to questions when the information was explicitly stated than when it was implied. It can be inferred from these data that PD leads to difficulties for processing both detailed and implied information when compared with healthy controls (Murray and Stout 1999).

Recently, we attempted to replicate and extend this study by administering the Discourse Comprehension Task to 14 PD patients, half of whom displayed impaired working memory and half of whom whose working memory fell within the control group range. Our findings replicated those of Murray and Stout (1999) in that the PD group as a whole had significant difficulties understanding details as well as implied information from discourse. However, subsequent analyses showed that only the PD patients with impaired working memory performed abnormally on this task, whereas PD patients with normal working memory capacity processed pragmatic features of discourse in the same way as healthy participants (Monetta et al. forthcoming). These findings argue that pragmatic language deficits in PD are due to an underlying disruption in cognitive functions, such as working memory, which are normally supported by the (intact) fronto-striatal circuits. This argument echoes earlier claims by Copland et al. (2001); using an online task, they found that PD participants are unable to select appropriate meanings and to generate inferences on the basis of an integrated discourse context, a pattern that was attributed to fronto-striatal deficits in strategic information processing and working memory.

When combined with previous data on sentence and pragmatic processing, most studies argue that there is an important interaction between working memory capacity, or possibly other estimates of cognitive resource capacity (such as attention), and the ability to divert sufficient resources to engage in certain 'complex' forms of sentence processing and various forms of pragmatic language processing, including the ability to understand metaphors and implied discourse information. As claimed in a recent study, many language deficits in PD may be due to impairments of the 'phonological loop' connecting the pre-frontal cortex with the basal ganglia (see Bodis-Woliner and Jo 2006). It can thus be said that many of the receptive language processing difficulties witnessed in PD are secondary psycholinguistic deficits produced by more basic impairments of executive functions and/or

working memory; these cognitive functions rely critically on intact frontal-striatal systems that become functionally impaired over the course of PD, explaining the source of their language impairments, at least in large part. However, as noted earlier, some language-related deficits such as difficulties in lexical-semantic integration (Friederici et al. 2003; Kotz et al. 2003) may reflect more direct interruptions of the language processing system. These hypotheses continue to be debated actively in the literature and need to be studied further. In addition, the presence of language deficits in PD need to be interpreted in the context of potential co-existing deficits that affect processes critical to non-verbal information processing as reviewed in the section below.

### *How Parkinson's Disease Affects Non-verbal Communication*

While the importance of understanding verbal information for humans is obvious, it is often said that it is not what we say that is most important, but how we say it. This attitude reflects the significance of non-verbal cues, such as facial expressions, body position, gestures, and tone of voice, to human communication. For example, we typically recognize that our conversation partner is feeling angry or fearful from their tone of voice while speaking and/or from their facial expressions; these cues that often co-occur with language supply powerful indicators about a speaker's emotional state independent of what is communicated by the linguistic message. Non-verbal cues sometimes reveal a speaker's true attitude or emotional state in the face of an apparently conflicting message presented through language (e.g., a friend who claims '*I'm fine!*' in an obviously despairing tone of voice). As well, facial expressions and tone of voice often serve to qualify how the listener should interpret the explicit meaning of a sentence (e.g., when asking for directions, one might be cautious in interpreting '*You turn left at the lights*' when spoken in a hesitant or doubtful sounding voice). These examples emphasize the critical role of non-verbal cues for understanding the emotions of a speaker, for deriving the intended meanings of language, and, in general, for engaging in efficient and rewarding social interactions.

For some time, it has been known that focal lesions of the brain, especially strokes that occur in the right cerebral hemisphere, can interrupt the ability to understand non-verbal cues, such as facial expressions and tone of voice (Heilman et al. 1975; see Pell 2006 for a recent discussion). Evidence that PD is associated with difficulties of this nature is more recent and is still being elaborated by researchers on several fronts. Again, for many the notion that PD yields abnormalities in the comprehension of non-verbal signs, rather than their expression, may not be well known; since PD was first identified, it has been recognized that one sign of motor disturbance in PD is limitations in the range of possible movements of the face and vocal apparatus that can affect speech intelligibility and the ability to express emotions through these communication channels. For example, Parkinsonian adults often display

abnormal changes in articulation, voice quality, and the ability to modulate voice pitch and loudness while speaking (Canter 1965; Darkins et al. 1988). These changes are serious in that they affect how well PD patients are able to communicate with listeners (Pell et al. 2006; Miller et al. 2008) and may also contribute to the inaccurate impression that PD patients are 'cold' or less 'likeable' based on features of their voice (Pitcairn et al. 1990). Increasingly, the prospect that some PD patients experience difficulties in both the expression and comprehension of non-verbal cues seems likely (Pell and Leonard 2003; Cheang and Pell 2007).

In the developing literature on how PD affects the comprehension of non-verbal cues, a number of questions are currently being investigated. Principally, does PD lead to a broad-based deficit in the ability to process emotions from non-verbal cues? Or are the observed difficulties somehow more selective in nature, for example, are they more pronounced when processing specific emotions or specific non-verbal display types (e.g., face vs. voice)? As argued in the section on language processing, is it possible that non-verbal comprehension skills are affected only in a subset of PD patients who experience changes in other aspects of cognition (i.e., executive impairments) or who display specific clinical features (e.g., depression)? Research that attempts to address these questions is discussed below, focusing on how facial expression and tone of voice (speech prosody) may be affected by PD.

Although it cannot be covered in detail here, the possibility that PD is associated with general deficits in affective functioning and in the processing of various types of emotional events must also be considered. In a study of 48 PD patients with and without cognitive impairment, Benke et al. (1998) reported that the ability to produce and recognize emotional prosody as well as the ability to detect pictorial humor were impaired in PD patients with cognitive impairments. The authors concluded that emotional processing impairments in PD are relatively broad based and that these deficits correlate with cognitive variables, such as memory loss and psychomotor slowing. More recently, Wieser et al. (2006) observed differences in how PD patients explicitly rated pictures with high versus low arousing content when compared to healthy adults, although the patients demonstrated normal ERP patterns that reflect early, automatic processes of emotional/visual discrimination. These findings suggested to the researchers that PD is associated with a general 'blunting' or reduction in responses to emotional stimuli that may be related to the cognitive demands of subjective rating tasks.

In a similar vein, Casterner et al. (2007) presented individual words with a negative or neutral valence to adults with and without PD and found that (automatic) affective priming effects were preserved in the PD group, although the patients displayed abnormalities in their response time patterns to negative words in certain conditions (i.e., when the patients were off stimulation of the subthalamic nucleus). Collectively, these data exemplify some of the possible broader effects of PD on basic affective functions and emotional information processing, while highlighting that

such deficits can be modulated by the effects of levodopa (L-DOPA) and subthalamic nucleus stimulation (Funkiewiez et al. 2006; Casterner et al. 2007). Important research on these topics is ongoing but will not be consulted in detail in our discussion of non-verbal emotion processing in PD.

#### FACE PROCESSING IN PARKINSON'S DISEASE

A face reveals a variety of information about its owner: details about his or her identity, which may or may not be familiar to those who encounter it; cues about affect and emotion; speech-related information; and various cues that are a source of social inferences, whether correct or incorrect, about the person in question (e.g., whether they seem 'trustworthy' or 'kind'). Abnormalities in face processing are frequently cited as one of the cognitive changes that occurs in PD (Levin et al. 1989; Dewick et al. 1991), although the nature and source of these difficulties is still not perfectly clear.

In an early study, Dewick and colleagues (1991) examined a spectrum of different face processing skills that could be affected in non-demented patients with PD, including the ability to discriminate and match unfamiliar faces by their identity, to discriminate faces by sex or emotional expression, and to discriminate faces according to which speech sound was being produced (akin to speech or 'lip-reading'). It is widely assumed that the ability to structurally analyze and encode basic features of a face is a necessary precursor for extracting all types of 'code', such as identity and emotions, which occur at later stages of processing (Bruce and Young 1986). In Dewick et al.'s study, they found that PD patients experience significant problems on almost all face processing tasks when compared to a healthy control group; the authors concluded that PD is associated with a basic difficulty in the structural encoding of facial expressions that yields a range of face processing impairments at all subsequent stages of processing. The observation that visual-perceptual processing of faces is interrupted by PD has been reported elsewhere in the literature as well (Beatty et al. 1989; Haeske-Dewick 1996). The possibility that configural as opposed to componential processing of facial stimuli is largely affected by PD, and that facial expressions that are degraded in some way, for example, line drawings or black and white as opposed to color photographs, pose special difficulties for those with the disease, was raised in a recent study (Cousins et al. 2000).

However, it is increasingly clear from the research that PD does not always lead to a broad-based impairment for all aspects of face processing as suggested by a deficit at the level of basic structural encoding. Several investigations indicate that PD patients can accurately process and discriminate the identity of faces, but then fail to correctly process the emotional expression of faces in similar tasks (Jacobs et al. 1995; Sprengelmeyer et al. 2003). These findings imply that PD is associated with more selective deficits for understanding the emotional attributes of faces. Sprengelmeyer and colleagues (2003) further noted that PD patients were less sensitive to certain emotions in the face, particularly

'disgust', although these deficits appeared to resolve somewhat in more advanced PD patients who were receiving dopamine-replacement therapy.

The idea that PD may lead to rather selective difficulties in the recognition of specific emotions, such as disgust cannot be discounted and is still being investigated; to date, a number of studies now suggest that the basal ganglia, which is functionally suppressed in the context of PD, are critical for processing this emotion (see Sprengelmeyer et al. 1996; Gray et al. 1997, 2000; Suzuki et al. 2006). For example, Suzuki et al. (2006) argue that interruption of a basal ganglia-insula system for recognizing disgust is selectively compromised in PD leading to emotion-specific impairments that cannot be explained by general face recognition abilities. Alternatively, another recent report argues that facial expressions of 'anger' are selectively tied to operations of the basal ganglia and therefore most problematic for PD patients, a deficit that can be detected when these patients are removed from medication (Lawrence et al. 2007). These issues continue to be studied to determine how the comprehension of specific emotions is affected in PD. Irrespective of what details will be found, these data emphasize that many patients with PD are likely susceptible to impairments that affect how emotions are understood from facial expressions and possibly other cues such as prosody as discussed below.

There is further evidence that not all PD patients are hampered by a significant decline in face processing skills at all. We compared the ability of 21 adults with and without PD to structurally encode faces and to discriminate, identify, and rate emotions as well as speech-sound information portrayed by actors of the same identity (Pell and Leonard 2005). While our data confirmed the finding that 'disgust' was most poorly understood by the PD patients, we uncovered little evidence that PD patients as a group were impaired overall in face processing in the areas evaluated. This result confirms other studies that report that PD is not uniformly tied to face processing deficits (Borod et al. 1990; Adolphs et al. 1998; St. Clair et al. 1998). It is likely that the different conclusions arrived at in this literature are the result of several variables that still need to be reconciled. In the context of neurodegenerative pathology, one obvious factor to consider is how the severity of PD contributes to (emotional) face processing deficits. However, there is still no clear answer to this question as several studies indicate that advanced signs of PD (as measured by disease duration and/or the level of motor impairment) correspond with the emergence of face processing difficulties (Breitenstein et al. 1998; Yip et al. 2003), whereas a number of studies have found no evidence of such a relationship (Jacobs et al. 1995; Haeske-Dewick 1996).

One suggestion based on a review of this literature is that advanced stages of PD are more likely to affect skills at the level of structural face encoding, with subsequent effects on how various facial 'codes' are understood; however, more selective deficits in emotional face processing or for specific emotion types may occur at different, including relatively early, stages of the disease

(Pell and Leonard 2005). The likelihood that medication status and brain dopamine levels play an important role in how these deficits are characterized is also indicated, particularly for predicting when deficits for specific emotions such as disgust or anger are experienced by PD patients (Sprenkelmeyer et al. 2003; Lawrence et al. 2007).

#### VOCAL PROCESSING IN PARKINSON'S DISEASE

In most ways, information conveyed by tone of voice (speech prosody) can lead to a similar array of meanings about a speaker's identity, their emotions, and other interpersonal features than related information communicated by facial expressions. Unlike facial expressions, however, prosodic information is fully integrated with speech (i.e., linguistic) information in the auditory modality and cannot be studied in a 'static' form. Ongoing changes in pitch, loudness, and the rhythmic structure of spoken utterances provide clues about a speaker's emotions, and as well, these attributes are frequently used in conjunction with language to signal that utterances should be interpreted as ironic or humorous rather than for their literal meaning, for example (Cheang and Pell 2008). While the ability of PD patients to process speech prosody has been examined somewhat less than for facial expressions, available studies on prosody are more consistent in showing that PD patients are impaired for this type of processing.

Following initial studies that brought attention to the idea that prosodic processing is affected in PD (Scott et al. 1984; Blonder et al. 1989), investigations conducted in the past decade have sought to clarify the nature of these deficits. Based on this research, there are strong indications that PD patients can adequately process prosodic information of a linguistic nature when it serves a relatively localized function in the utterance, such as it is required to semantically differentiate stress cues in the words *HOT*dog (the food) versus hot*DOG* (the animal that is hot) (Pell 1996). However, prosodic distinctions that operate over longer time intervals to convey meaning, such as expressions of emotion in speech, tend to be a source of difficulties for many PD patients (Blonder et al. 1989; Pell 1996; Breitenstein et al. 1998).

Data from our laboratory strongly support the view that the processing of emotional information from prosody is particularly affected even in the early stages of non-demented PD. In our evaluation of 21 PD patients and 21 healthy control participants – the same participants who were relatively unimpaired for recognizing emotions from facial expressions (Pell and Leonard 2005) – we found that the PD patients were notably impaired on tasks of identifying and rating emotions from prosody when pseudo-utterances such as '*Such fector egzulling tuh boshent*' were spoken with different emotional inflections (Pell and Leonard 2003). Since these utterances did not contain meaningful linguistic cues for understanding emotions, this condition shows that PD patients fail to appropriately interpret prosodic aspects of speech,

whereas the same participants were unimpaired in the comprehension of sentences that contained an emotional semantic context, such as '*I didn't make the team.*' Another result of this study was that PD patients were most impaired in the ability to recognize vocal expressions of disgust, which is consistent with earlier claims that this emotion is likely affected in PD. Very recently, we have replicated the finding that vocal expressions of disgust are poorly understood in PD, in addition to other negative expressions of vocal emotion such as anger and fear, in a new study of 15 PD patients in which the patients were required to identify the emotion and rate the intensity and valence of a common set of vocal expressions (Dara et al. 2008).

When our data on face and vocal (prosody) processing of emotions are compared, it may be concluded that processes for rendering an interpretation of speech prosody are especially prone to disruption in the context of PD. This conclusion may also be inferred by the relative consistency of reports showing emotional-prosodic deficits in PD, whereas reports of emotional face processing deficits are somewhat mixed. One reason that prosody may be vulnerable to the neuropathological effects of PD centers on one of the presumed functional roles of the basal ganglia: the striatum has been implicated in a variety of timing operations, including the perception of time intervals and the ability to resolve meaning from sequential properties of sensory events (Harrington et al. 1998). The striatum has also been described as critical for responding to changes in stimulus relevance (Cools et al. 2006). As vocal expressions of emotion are composed of, and understood through, sequential changes in acoustic-perceptual parameters such as pitch, loudness, and duration that must be monitored for their relevance in an ongoing manner, it can be hypothesized that dysregulation within the basal ganglia leads to less efficient processing of prosodic meanings in speech owing to difficulties with sequential processing (Pell and Leonard 2003). This deficit may be responsible for many of the prosodic impairments exhibited by PD patients. If this is shown to be true, one can predict that deficits in the processing of cue sequences do not lead strictly to difficulties for understanding emotions from speech in PD but extend to other prosodic functions that require sequential integration as well.

To test whether prosodic difficulties in PD extend beyond the so-called 'basic' emotions, we recently evaluated whether a group of 15 PD patients can recognize different attitudes from prosody, such as the confidence or politeness of a speaker, by listening to prosodic features of pseudo-utterances (Monetta et al. 2008). We found that PD patients could normally recognize the intended politeness of speakers, which tends to be signaled in a relatively categorical manner through prosody by adopting a high or low voice pitch. However, the same patients were impaired relative to control participants when rating how confident/doubtful a speaker sounds, which is signaled in a more complex manner by modulating multiple prosodic cues over the duration of an utterance. These data, while in need of replication and extension, appear to support the idea that prosodic difficulties in PD

relate to how cue sequences are processed for their significance over longer time intervals of speech. However, note that this explanation would not account for why specific emotions are sometimes affected by PD (for both face and voice); these difficulties may instead be due to the fact that the basal ganglia are simultaneously part of specialized neural circuitry for understanding certain emotions, such as disgust, anger, or fear (see Dara et al. 2008 for a recent discussion). In addition, electrophysiological data indicating that the pre-attentive processing of emotional prosody may be abnormal in PD (Schröder et al. 2006) will need to be explored further in the context of the growing literature on this topic.

With the exception of emotion-specific deficits that may have a common source for both face and voice, it is noteworthy that current accounts of why prosodic deficits emerge in the course of PD (i.e., due to problems with sequential information) do not fit neatly with data showing that static facial expressions are recognized poorly by these patients under certain conditions. These discrepancies imply that the source of face and prosody deficits in PD may overlap to some extent, but is largely independent. More research is clearly needed. The fact that several distinct impairments have been proposed to account for the non-verbal and verbal deficits observed in PD patients may simply reflect the current immaturity of this research field. However, it is equally possible that these different proposals confirm that progressive changes in the brain of PD patients, with increasing effects on 'high-level' cortical functions, can disrupt a variety of functional systems that support critical aspects of cognition and communication, as elaborated below.

### *Toward an Integrated Account of How Parkinson's Disease Affects Receptive Communication*

When the various findings on non-verbal communication are compared to what is known about language processing deficits in PD, a rather complex picture emerges of how PD affects communication and language comprehension more generally. Some of the communication deficits associated with PD seem to reflect influences of the functionally impaired basal ganglia on communication processes, whereas other communication deficits emerge as one of the sequelae of cognitive restrictions that emerge at certain stages of the disease, which can be linked to deterioration of the fronto-striatal pathways. An attempt to structure the collective findings on PD in a constructive manner will be important for future research.

One of the prominent themes emerging from studies of receptive communication in PD is the notion of 'resource capacity' or resource-allocation during online language processing. Many of the verbal and non-verbal deficits observed in PD patients may be explained, or at least influenced to some degree, by conditions in which individual PD patients do not have sufficient cognitive resources demanded by these communication processes. According to Taylor and St-Cyr (1995), 'when the problem to be solved require(s) the

subject to plan and to execute a strategy using only internal resources to guide behavior, PD patients experience difficulty.' Although it is still unclear how to precisely characterize the 'internal resources' that PD lacks with respect to communication, the fact that difficulties with language and pragmatic processing are often confined to the most demanding conditions and that they frequently correlate with working memory capacity (e.g., Breitenstein et al. 2001; Grossman et al. 2003; Monetta and Pell 2007) represents a good platform for additional studies on receptive communication in PD.

In addition, to fully understand the difficulties faced by individuals suffering from PD, one must always keep two concepts in mind: the clinical picture of PD and its motor and cognitive sequelae are often variable from patient to patient; and, communication deficits are progressive or evolving. For example, while PD is usually categorized as a 'motor' disorder and most patients retain their intellectual capacity, a minority of PD patients develop a full-blown dementia; even in most instances when dementia is absent, the majority of PD patients nonetheless present with impairments in working memory, planning, and frontal-executive functions, although the presence and severity of these signs also vary (Dubois and Pillon 1997). Even hallmark motor signs of PD (e.g., tremor, bradykinesia) are variable across patients; in fact, the prominence of particular motor signs in individual PD patients has been linked by some researchers to particular profiles of cognitive decline. For example, a weak relationship has been reported between tremor and cognitive deterioration in PD, whereas bradykinesia and gait appear to be most strongly correlated with problems in cognitive functioning, such as executive control, verbal learning, and visual memory (Mortimer et al. 1982; Fleischman et al. 2005). On the other hand, a negative correlation has been found between rigidity and cognitive functioning (Reid et al. 1989).

Another issue of probable importance to understanding communication deficits in PD (and their treatment) is the patient's medication status. With rare exceptions, the data reported in this overview have been derived from investigations of PD patients who are optimally medicated during language testing using L-DOPA. This medication is well-known to improve the motor symptoms of PD but its effects on cognitive functions are either unknown or complex; according to Cools (2006), depending on the nature of the task and basal dopamine levels in different parts of the striatum, L-DOPA can produce either beneficial or detrimental effects on cognitive capacities, such as working memory or cognitive flexibility. Based on our review, the manner in which medication status affects these basic cognitive abilities – whether positive or negative – is critical to document since the status of these cognitive abilities is believed to have important secondary effects on language functions. Further evidence that L-DOPA levels and certain surgical interventions to alleviate motor symptoms of PD, such as subthalamic nucleus stimulation, can have diverging effects on cognitive versus affective functions in many PD patients (Funkiewiez et al. 2006) will also need to be examined closer in relation to the communication skills of PD patients.

Perhaps one of the main conclusions that can be drawn from our discussion is that PD should not be treated as a single disease entity with predictable consequences on receptive communication. Rather, it may be more useful to specify PD subgroups that satisfy hypothetical conditions (e.g., high versus low working memory or some measure of executive functioning), which tend to influence particular communication difficulties at different stages of the disease. In this way, it is likely that the psychosocial effects of PD can be studied in a more principled manner and in more detail by researchers or clinicians who work with these patients, and ultimately, the underlying nature of these deficits can be better determined. Other clinical variables that have at times been associated with a degradation of cognitive and communicative functions, such as medication status, depression, disease duration, and age of PD onset, may also prove useful in constructing PD subgroups that illuminate the effects of PD on language and communication in future investigations. In time, this knowledge will undoubtedly translate into improvements in clinical practice that help adults with PD to retain their quality of life and the functional independence afforded to those who can communicate effectively in society.

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### *Short Biographies*

Marc Pell has a broad interest in how humans communicate their emotions, attitudes, and other intentions during normal social interactions and in the context of acquired diseases of the brain (e.g., stroke, PD). Much of his research has studied how speech prosody, or a speaker's tone of voice, is used to convey different intentions in speech and how these cues provide a source of information for understanding the emotions and cognitive disposition of others in the context of spoken language. Current research uses methods from social psychology, cognitive neuropsychology, and neuroimaging to explore inter-related topics of emotional and pragmatic aspects of communication and to link these forms of processing to the brain. He holds grants from Canadian and Québec funding agencies and his work was recognized by McGill University in the form of a junior research chair awarded in 2003 (William Dawson Scholar). Since 1997, Marc has been teaching in the School of Communication Sciences and Disorders at McGill University, Montréal, Canada. He holds a BA in Linguistics and Slavic Studies from the University of Ottawa and an MSc and PhD degree in Communication Sciences and Disorders from McGill.

Laura Monetta's research interests focus on pragmatic communication disorders in brain-damaged populations. Current projects are looking at the study of metaphor, verbal irony, and inference comprehension in patients with PD. She is also collaborating in a longitudinal project aiming to compare the evolution of cognitive and motor signs as PD progresses using neuropsychological evaluation and functional magnetic resonance imaging. She has held fellowships from the Canadian Institutes of Health Research and the McGill Centre for Research on Language, Mind and Brain. She gives different lectures at the University of Montreal on cognitive aging and acquired languages deficits for speech-therapist, ergo-therapist, and physio-therapist students. She also works at a clinician in a rehabilitation center for adults with neurological disorders in Montreal. She holds a BA in speech pathology and audiology from the University of San Luis, Argentina, and a PhD in Biomedical sciences from the University of Montreal, Canada. She is currently an Assistant Professor at Laval University, Quebec, Canada.

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### Works Cited

- Adolphs, R. 2002. Neural systems for recognizing emotion. *Current Opinion in Neurobiology* 12.169–77.
- Adolphs, R., R. Schul, and D. Tranel. 1998. Intact recognition of facial emotion in Parkinson's disease. *Neuropsychology* 12.253–8.
- Alexander, G., M. DeLong, and P. Strick. 1986. Parallel organization of functionally segregated circuits linking basal ganglia and cortex. *Annual Review of Neuroscience* 9.337–81.
- Angwin, A. J., H. J. Chenery, D. A. Copland, W. L. Arnott, B. E. Murdoch, and P. A. Silburn. 2004a. Dopamine and semantic activation: an investigation of masked direct and indirect priming. *Journal of the International Neuropsychological Society* 10.15–25.
- Angwin, A. J., H. J. Chenery, D. A. Copland, B. E. Murdoch, and P. A. Silburn. 2004b. The time course of semantic activation in Parkinson's disease. *Brain and Language* 91.145–6.
- . 2005. Summation of semantic priming and complex sentence comprehension in Parkinson's disease. *Cognitive Brain Research* 25.78–89.
- . 2007. The speed of lexical activation is altered in Parkinson's disease. *Journal of Clinical and Experimental Neuropsychology* 29.73–85.
- Arnott, W. L., H. J. Chenery, B. E. Murdoch, and P. A. Silburn. 2001. Semantic priming in Parkinson's disease: evidence for delayed spreading activation. *Journal of Clinical and Experimental Neuropsychology* 23.502–19.
- Beatty, W. W., D. E. Goodkin, W. S. Weir, R. D. Staton, N. Monson, and P. A. Beatty. 1989. Affective judgments by patients with Parkinson's disease or chronic progressive Multiple Sclerosis. *Bulletin of the Psychonomic Society* 27.361–4.
- Benke, T. H., S. Bösch, and B. Andree. 1998. A study of emotional processing in Parkinson's disease. *Brain and Cognition* 38.36–52.
- Berg, E., C. Bjornram, L. Hartelius, K. Laakso, and B. Johnels. 2003. High-level language difficulties in Parkinson's disease. *Clinical Linguistics and Phoniatric* 17.63–80.

- Blonder, L., R. Gur, R. Gur, A. Saykin, and H. Hurtig. 1989. Neuropsychological functioning in Hemiparkinsonism. *Brain and Cognition* 9.244–57.
- Bodis-Woliner, I., and M. Y. Jo. 2006. Getting around and communicating with the environment: visual cognition and language in Parkinson's disease. *Journal of Neural Transmission* 70.333–8.
- Borod, J. C., J. Welkowitz, M. Alpert, A. Z. Brozgold, C. Martin, E. Peselow, and L. Diller. 1990. Parameters of emotional processing in neuropsychiatric disorders: conceptual issues and a battery of tests. *Journal of Communication Disorders* 23.247–71.
- Breitenstein, C., D. Van Lancker, I. Daum, and C. Waters. 2001. Impaired perception of vocal emotions in Parkinson's disease: influence of speech time processing and executive functioning. *Brain and Cognition* 45.277–314.
- Breitenstein, C., I. Daum, and H. Ackermann. 1998. Emotional processing following cortical and subcortical brain damage: contribution of the fronto-striatal circuitry. *Behavioural Neurology* 11.29–42.
- Brookshire, R. H., and L. E. Nicholas. 1993. Discourse comprehension test. Tucson, AZ: Communication Skill Builders.
- Brown, R. G., and C. D. Marsden. 1991. Dual task performance and processing resources in normal subjects and patients with Parkinson's disease. *Brain* 114.215–31.
- Bruce, V., and A. Young. 1986. Understanding face recognition. *British Journal of Psychology* 77.305–27.
- Caballol, N., M. J. Martí, and E. Tolosa. 2007. Cognitive dysfunction and dementia in Parkinson disease. *Movement Disorders* 22.S358–66.
- Calder, A., F. Keane, F. Manes, N. Antoun, and A. Young. 2000. Impaired recognition and experience of disgust following brain injury. *Nature Neuroscience* 3.1077–8.
- Canter, G. J. 1965. Speech characteristics of patients with Parkinson's disease: III. Articulation, diadochokinesis, and over-all speech adequacy. *Journal of Speech and Hearing Disorders* 30.217–24.
- Casterner, J. E., H. Chenery, D. Copland, T. Coyne, F. Sinclair, and P. Silburn. 2007. Semantic and affective priming as a function of stimulation of the subthalamic nucleus in Parkinson's disease. *Brain* 130.1395–407.
- Cheang, H. S., and M. D. Pell. 2007. An acoustic investigation of Parkinsonian speech in linguistic and emotional contexts. *Journal of Neurolinguistics* 20.221–41.
- . 2008. The sound of sarcasm. *Speech Communication* 50.366–81.
- Cohen, H., S. Bouchard, P. Scherzer, and H. Whitaker. 1994. Language and verbal reasoning in Parkinson's disease. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology* 7.166–75.
- Cools, R. 2006. Dopaminergic modulation of cognitive function—implications for L-DOPA treatment in Parkinson's disease. *Neuroscience and Biobehavioral Reviews* 30.1–23.
- Cools, R., R. B. Ivry, and M. D'Esposito. 2006. The human striatum is necessary for responding to changes in stimulus relevance. *Journal of Cognitive Neuroscience* 18.1973–83.
- Copland, D. 2003. The basal ganglia and semantic engagement: potential insights from semantic priming in individuals with subcortical vascular lesions, Parkinson's disease, and cortical lesions. *Journal of the International Neuropsychological Society* 9.1041–52.
- Copland, D., H. Chenery, and B. E. Murdoch. 2001. Discourse priming of homophones in individuals with dominant nonthalamic subcortical lesions, cortical lesions and Parkinson's disease. *Journal of Clinical and Experimental Neuropsychology* 23.538–56.
- Cotelli, M., B. Borroni, R. Manenti, M. Zanetti, A. Arevalo, S. F. Cappa, and A. Padovani. 2007. Action and object naming in Parkinson's disease without dementia. *European Journal of Neurology* 14.632–7.
- Cousins, R., J. Hanley, A. Davies, C. Turnbull, and J. Playfer. 2000. Understanding memory for faces in Parkinson's disease: the role of configural processing. *Neuropsychologia* 38.837–47.
- Critchley, E. M. R. 1981. Speech disorders of Parkinsonism: a review. *Journal of Neurology, Neurosurgery, and Psychiatry* 44.751–58.
- Cummings J. L., A. Darkins, M. Mendez, and M. A. Hill. 1988. Alzheimer disease and Parkinson's disease: comparison of speech and language alterations. *Neurology* 38.680–4.
- Dara, C., L. Monetta, and M. D. Pell. 2008. Vocal emotion processing in Parkinson's disease: reduced sensitivity to negative emotions. *Brain Research* 1188.100–11.

- Darkins, A. W., V. A. Fromkin, and D. F. Benson. 1988. A characterization of the prosodic loss in Parkinson's disease. *Brain and Language* 34.315–27.
- Dewick, H. C., J. R. Hanley, A. D. M. Davies, J. Playfer, and C. Turnbull. 1991. Perception and memory for faces in Parkinson's disease. *Neuropsychologia* 29.785–802.
- Dubois, B., and B. Pillon. 1997. Cognitive deficits in Parkinson's disease. *Journal of Neurology* 244.2–8.
- Filoteo, J. V., F. J. Friedrich, L. M. Rilling, J. D. Davis, J. L. Stricker, and M. Prenovitz. 2003. Semantic and cross-case identity priming in patients with Parkinson's disease. *Journal of Clinical and Experimental Neuropsychology* 25.441–56.
- Fleischman, D., R. Wilson, J. Bienias, and D. Bennett. 2005. Parkinsonian signs and cognitive function in old age. *Journal of the International Neuropsychological Society* 11.591–7.
- Friederici, A. D. 2002. Towards a neural basis of auditory sentence processing. *Trends in Cognitive Sciences* 6.78–84.
- Friederici, A. D., and K. Alter. 2004. Lateralization of auditory language functions: a dynamic dual pathway model. *Brain and Language* 89.267–76.
- Friederici, A. D., D. Y. von Cramon, and S. A. Kotz. 1999. Language related brain potentials in patients with cortical and subcortical left hemisphere lesions. *Brain* 122.1033–47.
- Friederici, A. D., S. A. Kotz, K. Werheid, G. Hein, and D. Y. von Cramon. 2003. Syntactic comprehension in Parkinson's disease: investigating early automatic and late integrational processes using event-related brain potentials. *Neuropsychology* 17.133–42.
- Funkiewiez, A., C. Arduin, R. Cools, P. Krack, V. Fraix, A. Batir, S. Chabardès, A-L. Benabid, T. W. Robbins, and P. Pollak. 2006. Effects of levodopa and subthalamic nucleus stimulation on cognitive and affective functioning in Parkinson's disease. *Movement Disorders* 21.1656–62.
- Gabrieli, J. D. E., J. Singh, G. T. Stebbins, and C. G. Goetz. 1996. Reduced working memory span in Parkinson's disease: evidence for the role a frontostriatal system in working and strategic memory. *Neuropsychology* 10.322–32.
- Gernsbacher, M. A., B. Keysar, R. R. W. Robertson, and N. K. Werner. 2001. The role of suppression and enhancement in understanding metaphors. *Journal of Memory and Language* 45.433–50.
- Geyer, H. L., and M. Grossman. 1994. Investigating the basis for the sentence comprehension deficit in Parkinson's disease. *Journal of Neurolinguistics* 8.191–205.
- Gray, J. M., A. W. Young, W. A. Barker, A. Curtis, and D. Gibson. 1997. Impaired recognition of disgust in Huntington's disease gene carriers. *Brain* 120.2029–38.
- Grossman, M., A. C. Cooke, C. DeVita, D. Alsop, J. Detre, W. Chen, and J. Gee. 2002. Age-related changes in working memory during sentence comprehension: an fMRI study. *Neuroimage* 15.302–17.
- Grossman, M., A. Cooke, C. DeVita, C. Lee, D. Alsop, J. Detre, J. Gee, W. Chen, M. B. Stern, and H. I. Hurtig. 2003. Grammatical and resource components of sentence processing in Parkinson's disease: an fMRI study. *Neurology* 60.775–81.
- Grossman, M., S. Carvell, S. Gollomp, M. B. Stern, G. Vernon, and H. I. Hurtig. 1991. Sentence comprehension and praxis deficits in Parkinson's disease. *Neurology* 41.1620–6.
- . 1993. The sum and substance of it: the appreciation of mass and count quantifiers in Parkinson's disease. *Brain and Language* 44.351–84.
- Grossman, M., S. Carvell, M. B. Stern, S. Goliomp, and H. I. Hurtig. 1992. Sentence comprehension in Parkinson's disease: the role of attention and memory. *Brain and Language* 42.347–84.
- Haeske-Dewick, H. C. 1996. Are perception and memory for faces influenced by a specific age at onset factor in Parkinson's disease? *Neuropsychologia* 34.315–20.
- Harrington, D., K. Haaland, and N. Hermanowicz. 1998. Temporal processing in the basal ganglia. *Neuropsychology* 12.3–12.
- Heilman, K. M., R. Scholes, and R. Watson. 1975. Auditory affective agnosia: disturbed comprehension of affective speech. *Journal of Neurology, Neurosurgery, and Psychiatry* 38.69–72.
- Henry, J. D., and J. R. Crawford. 2004. Verbal fluency deficits in Parkinson's disease: a meta-analysis. *Journal of the International Neuropsychological Society* 10.608–22.

- Huber, S., D. Freidenberg, E. Shulteworth, G. W. Paulson, and J. A. Christy. 1989. Neuropsychological impairments associated with severity of Parkinson's disease. *Journal of Neuropsychological Clinical Neuroscience* 1.154–8.
- Jacobs, D. H., J. Shuren, D. Bowers, and K. M. Heilman. 1995. Emotional facial imagery, perception, and expression in Parkinson's disease. *Neurology* 45.1696–702.
- Kemmerer, D. 1999. Impaired comprehension of raising-to-subject constructions in Parkinson's disease. *Brain and Language* 66.311–28.
- Kotz, S. A., S. Frisch, D. Y. von Cramon, and A. D. Friederici. 2003. Syntactic language processing: ERP lesion data on the role of the basal ganglia. *Journal of the International Neuropsychological Society* 9.1053–60.
- Lawrence, A. D., I. Goerendt, and D. Brooks. 2007. Impaired recognition of facial expressions of anger in Parkinson's disease patients acutely withdrawn from dopamine replacement therapy. *Neuropsychologia* 45.65–74.
- Levin, B., M. Llabre, and W. Weiner. 1989. Cognitive impairments associated with early Parkinson's disease. *Neurology* 39.557–61.
- Levy G., M. X. Tang, E. D. Louis ED, L. J. Côté, B. Alfaro, H. Mejia, Y. Stern, and K. Marder. 2002. The association of incident dementia with mortality in PD. *Neurology* 59.1708–13.
- Lewis, S. J., R. Cools, T. W. Robbins, A. Dove, R. A. Barker, and A. M. Owen. 2003. Using executive heterogeneity to explore the nature of working memory deficits in Parkinson's disease. *Neuropsychologia* 41.645–54.
- Lieberman, P., E. Kako, J. Friedman, G. Tajchman, L. S. Feldman, and E. B. Jiminez. 1992. Speech production, syntax comprehension, and cognitive deficits in Parkinson's disease. *Brain and Language* 43.169–89.
- Lieberman, P., J. Friedman, and L. S. Feldman. 1990. Syntax comprehension deficits in Parkinson's disease. *Journal of Nervous and Mental Disease* 178.360–36.
- McNamara, P., and R. Durso. 2003. Pragmatic communication skills in patients with Parkinson's disease. *Brain and Language* 84.414–23.
- Miller, N., E. Noble, D. Jones, L. Allcock, and D. J. Burn. 2008. How do I sound to me? Perceived changes in communication in Parkinson's disease. *Clinical Rehabilitation* 22.14–22.
- Monetta, L., and M. D. Pell. 2007. Effects of verbal working memory deficits on metaphor comprehension in patients with Parkinson's disease. *Brain and Language* 101.80–9.
- Monetta, L., H. S. Cheang, and M. D. Pell. 2008. Understanding speaker attitudes from prosody by adults with Parkinson's disease. *Journal of Neuropsychology* 2.
- Monetta, L., C. M. Grindrod, and M. D. Pell. forthcoming. Effects of working memory capacity on inference generation during story comprehension in adults with Parkinson's disease. *Journal of Neurolinguistics*.
- Mortimer, J. A., F. J. Pirozzolo, E. C. Hansch, and D. D. Webster. 1982. Relationship of motor symptoms to intellectual deficits in Parkinson's disease. *Neurology* 32(2).133–7.
- Murray, L. L., and J. C. Stout. 1999. Discourse comprehension in Huntington's and Parkinson's diseases. *American Journal of Speech-Language Pathology* 8.137–48.
- Natsopoulos, D., G. Grouios, S. Bostantzopoulou, G. Mentenopoulos, Z. Katsarou, and J. Logothetis. 1993. Algorithmic and heuristic strategies in comprehension of complement clauses by patients with Parkinson's disease. *Neuropsychologia* 31.951–64.
- Natsopoulos, D., Z. Katsarou, S. Bostantzopoulou, G. Grouios, G. Mentenopoulos, and J. Logothetis. 1991a. Strategies in comprehension of relative clauses by Parkinsonian patients. *Cortex* 27.255–68.
- Natsopoulos, D., G. Mentenopoulos, S. Bostantzopoulou, Z. Katsarou, G. Grouios, and J. Logothetis. 1991b. Understanding of relational time terms before and after in Parkinsonian patients. *Brain and Language* 40.444–58.
- Natsopoulos, D., Z. Katsarou, A. Alevriadou, G. Grouios, S. Bostantzopoulou, and G. Mentenopoulos. 1997. Deductive and inductive reasoning in Parkinson's disease patients and normal controls: review and experimental evidence. *Cortex* 33.463–81.
- Pell, M. D. 1996. On the receptive prosodic loss in Parkinson's disease. *Cortex* 32.693–704.
- . 2006. Cerebral mechanisms for understanding emotional prosody in speech. *Brain and Language* 96.221–34.
- Pell, M. D., and C. L. Leonard. 2003. Processing emotional tone from speech in Parkinson's disease: a role for the basal ganglia. *Cognitive, Affective and Behavioral Neuroscience* 3.275–88.

- . 2005. Facial expression decoding in early Parkinson's disease. *Cognitive Brain Research* 23.327–40.
- Pell, M. D., H. S. Cheang, and C. L. Leonard. 2006. The impact of Parkinson's disease on vocal-prosodic communication from the perspective of listeners. *Brain and Language* 97.123–34.
- Pitcairn, T. K., S. Clemie, J. M. Gray, and B. Pentland. 1990. Impressions of Parkinsonian patients from their recorded voices. *British Journal of Disorders of Communication* 25.85–92.
- Portin, R., S. Laatu, A. Revonsuo, and U. K. Rinne. 2000. Impairment of semantic knowledge in Parkinson Disease. *Archives of Neurology* 57(9).1338–43.
- Reid, W. G., G. A. Broe, M. A. Hely, J. G. Morris, P. M. Williamson, D. J. O'Sullivan, et al. 1989. The neuropsychology of de novo patients with idiopathic Parkinson's disease: the effects of age of onset. *International Journal of Neuroscience* 48(3–4).205–17.
- Schirmer, A., and S. A. Kotz. 2006. Beyond the right hemisphere: brain mechanisms mediating vocal emotional processing. *Trends in Cognitive Sciences* 10.24–30.
- Schröder, C., J. Möbes, M. Schütze, F. Szymanowski, W. Nager, M. Bangert, T. Münte, and R. Dengler. 2006. Perception of emotional speech in Parkinson's disease. *Movement Disorders* 21.1774–8.
- Scott, S., F. Caird, and B. Williams. 1984. Evidence for an apparent sensory speech disorder in Parkinson's disease. *Journal of Neurology, Neurosurgery, and Psychiatry* 47.840–3.
- Sprenkelmeyer, R., A. W. Young, A. J. Calder, A. Karnat, H. Lange, V. Homberg, D. I. Perrett, and D. Rowland. 1996. Loss of disgust: perception of faces and emotions in Huntington's disease. *Brain* 119.1647–65.
- Sprenkelmeyer, R., A. W. Young, K. Mahn, U. Schroeder, D. Woitalla, T. Buttner, W. Kuhn, and H. Przuntek. 2003. Facial expression recognition in people with medicated and unmedicated Parkinson's disease. *Neuropsychologia* 41.1047–57.
- St. Clair, J., J. C. Borod, M. Sliwinski, L. J. Cote, and Y. Stern. 1998. Cognitive and affective functioning in Parkinson's disease patients with lateralized motor signs. *Journal of Clinical and Experimental Neuropsychology* 20.320–7.
- Suzuki, A., T. Hoshino, K. Shigemasa, and M. Kawamura. 2006. Disgust-specific impairment of facial expression recognition in Parkinson's disease. *Brain* 129.707–17.
- Taylor, A. E., and J. A. St-Cyr. 1995. The neuropsychology of Parkinson's-Disease. *Brain and Cognition* 28(3).281–96.
- Taylor, A. E., J. A. St-Cyr, and A. E. Lang. 1986. Frontal lobe dysfunction in Parkinson's disease: the cortical focus of neostriatal outflow. *Brain* 109.845–83.
- Tettamanti, M., G. Buccino, M. C. Saccuman, V. Gallese, M. Danna, and P. Scifo, F. Fazio, G. Rizzolatti, S. F. Cappa, and D. Perani. 2005. Listening to action-related sentences activates fronto-parietal motor circuits. *Journal of Cognitive Neuroscience* 17.273–81.
- Tisch, S., P. Silberstein, P. Limousin-Dowsey, and M. Jahanshahi. 2004. The basal ganglia: anatomy, physiology, and pharmacology. *Psychiatric Clinics of North America* 27.757–99.
- Ullman, M. T., S. Corkin, M. Coppola, G. Hickok, J. H. Growdon, W. J. Koroshetz, and S. Pinker. 1997. A neural dissociation within language: evidence that the mental dictionary is part of declarative memory, and that grammatical rules are processed by the procedural system. *Journal of Cognitive Neuroscience* 9.266–76.
- Waters, G., and D. Caplan. 1997. The role of working memory in sentence processing: evidence from Parkinson's disease. *Brain and Language* 60.64–6.
- Wieser, M. J., A. Mühlberger, G. W. Alpers, M. Macht, H. Ellgring, and P. Pauli. 2006. Emotion processing in Parkinson's disease: dissociation between early neuronal processing and explicit ratings. *Clinical Neurophysiology* 117.94–102.
- Williams-Gray, C. H., T. Foltynie, C. E. G. Brayne, T. W. Robbins, and R. A. Barker. 2007. Evolution of cognitive dysfunction in an incident Parkinson's disease cohort. *Brain* 130.1787–98.
- World Health Organisation: Press Release WHO/71, 14 October 1998.
- Yip, J. T. H., T. M. C. Lee, S.-L. Ho, K.-L. Tsang, and L. S. W. Li. 2003. Emotion recognition in patients with idiopathic Parkinson's disease. *Movement Disorders* 18.1115–22.