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An acoustic investigation of Parkinsonian speech in linguistic and emotional contexts

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Abstract

The speech prosody of a group of patients in the early stages of Parkinson's disease (PD) was compared to that of a group of healthy age- and education-matched controls to quantify possible acoustic changes in speech production secondary to PD. Both groups produced standardized speech samples across a number of prosody conditions: phonemic stress, contrastive stress, and emotional prosody. The amplitude, fundamental frequency, and duration of all tokens were measured. PD speakers produced speech that was of lower amplitude than the tokens of healthy speakers in many conditions across all production tasks. Fundamental frequency distinguished the two speaker groups for contrastive stress and emotional prosody production, and duration differentiated the groups for phonemic stress production. It was concluded that motor impairments in PD lead to adverse and varied acoustic changes which affect a number of prosodic contrasts in speech and that these alterations appear to occur in earlier stages of disease progression than is often presumed by many investigators.

Keywords: Parkinson's disease; Prosody; Acoustic deficits; Speech production; Emotion; Linguistic stress

1. Introduction

It has long been noted that patients with idiopathic Parkinson's disease (PD) exhibit speech production impairments (among other motor deficits), as a result of reduced physiological support for speech (e.g., breath support) as well as lessened control of the

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musculature of the larynx and oral cavity (Canter, 1965a, 1965b; Critchley, 1981; Cummings, Darkins, Mendez, Hill, & Benson, 1988; Kegl, Cohen, & Poizner, 1999). Specifically, progressive deterioration in speech amplitude (i.e., loudness) and fundamental frequency (f0) (i.e., pitch), as well as abnormal rates of articulation have been noted in the speech of PD patients, in the absence of classical aphasic deficits (e.g., Canter, 1963, 1965a, 1965b; Harel, Cannizzaro, Cohen, Reilly, & Snyder, 2004; Illes, Metter, Hanson, & Iritani, 1988). These acoustic changes can be characterized as alterations of *prosody* and are likely to contribute to listener perceptions of Parkinsonian speech as lacking "acoustic contrast or detail" (Kent & Rosenbek, 1982).

Acoustic differences in the speech produced by adults with PD compared to normally aging adults have been reported after studying different speech phenomena varying in complexity and length. For example, studies that have focused on the ability to produce relatively "short" linguistic distinctions in speech, such as single consonants or syllables, have observed abnormalities in the modulation of several key acoustic parameters when these forms are produced by PD speakers (Canter, 1963, 1965b; Connor, Ludlow, & Schulz, 1989; Goberman, Coelho, & Robb, 2002). Producing the distinction between word pairs that differ only in stress placement (*phonemic stress,* e.g., *hot*dog versus hot *dog*) has also been found to be impaired in PD (Darkins, Fromkin, & Benson, 1988). When producing longer prosodic distinctions in speech, many PD speakers also have difficulties in the production of *contrastive stress*; that is, they show abnormalities in the ability to emphasize specific words within an utterance to underscore the salience of these word meanings in the verbal context (Blonder, Gur, & Gur, 1989; Penner, Miller, Hertrich, Ackermann, & Schumm, 2001). Many speakers with PD demonstrate abnormalities when required to modulate intonational features of full sentences (Canter, 1963; Cummings et al., 1988; Le Dorze, Dionne, Ryalls, Julien, & Ouellet, 1992; Ludlow, Connor, & Bassich, 1987) or to express emotions (Caekebeke, Jennekens-Schinkel, van der Linden, Buruma, & Roos, 1991). Finally, some studies have documented acoustic differences for reading aloud, engaging in conversation, or producing monologues by PD speakers (Canter, 1963; Goberman & Elmer, 2005; Goberman & Coelho, 2002a, 2002b; Van Lancker Sidtis et al., 2004). It is thus not surprising that healthy listeners experience difficulties with accurately perceiving prosodic meanings such as emotional statements when they are produced by speakers with PD as compared to healthy, aging adults (Pell, Cheang, & Leonard, 2006).

In these prior studies, there is some consensus that PD speakers display the following acoustic abnormalities, either in isolation or in combination, which impact on prosodic communication: shortened sustained phonation times, reductions in amplitude and amplitude range, anomalous f0 and restricted f0 range (e.g., Canter, 1963; Ho, Iansek, & Bradshaw, 1999; Illes, 1989). In addition, the duration or timing of Parkinsonian speech is frequently atypical, although the findings here are less uniform than with the amplitude and f0 parameters. PD speakers have been observed to produce utterances at higher or more variable rates of speech (Kent & Rosenbek, 1982; McRae, Tjaden, & Schoonings, 2002) or at slower rates (Illes et al., 1988). It is possible that these timing differences arise from differences in methodology; regardless, these studies reveal aberrant rates of speech among PD patients. In addition to identifying which acoustic parameters of speech are affected, some investigators have evaluated these acoustic changes as the disease progresses. Recent evidence suggests that pathologial f0 changes (i.e., markedly different changes that can affect intelligibility) occur very early in PD (Illes, 1989), possibly well before a formal diagnosis of PD has been made (Harel, Cannizzaro, & Cohen et al., 2004;

Harel, Cannizzaro, & Snyder, 2004). These new observations, while tentative, imply that early speech signs may have utility as a diagnostic or screening measure for PD, with the potential to influence the timing of clinical interventions with these patients. However, the acoustic features of these speech signs and their severity are not well understood.

The goal of the present study was to comprehensively evaluate the acoustic features of utterances produced by speakers in the early stages of idiopathic PD (without significant cognitive or depressive signs) and to compare these features to matched participants without the disease. We focused our analyses on how speakers produce different prosodic distinctions that are common to everyday interactions, such as the ability to produce stress, emphasis, and emotions through modulation of appropriate parameters of the voice. This approach will help determine the nature and extent of expressive difficulties experienced by Parkinsonian speakers during prosodic communication, while supplying objective acoustic measures to consider in light of our recent claim that *listeners* often fail to understand prosodic meanings when they are produced by Parkinsonian adults (Pell et al., 2006). Based on existing acoustic and perceptual data we expected that our PD speakers, relative to healthy controls, would display abnormal restrictions in f0 and amplitude in most if not all of our prosody conditions (e.g., Blonder et al., 1989; Caekebeke et al., 1991; Darkins et al., 1988; Pell et al., 2006), and possibly differences in duration measures as well (Illes et al., 1988; Kent & Rosenbek, 1982). While acoustic differences were expected to differentiate the groups in each of our prosody conditions, we especially predicted group distinctions in the production of emotional meanings which are expressed over longer time intervals (Caekebeke et al., 1991; Pell et al., 2006).

2. Method

2.1. Participants

There were 21 native English-speaking adults with idiopathic PD and 21 healthy control (HC) subjects closely matched on a one-to-one basis for sex, age, and education who took part in the study. These individuals were the same subjects who participated in our studies on how adults with PD perceive emotions in the voice and in the face (Pell & Leonard, 2003, 2005). The PD and HC groups were extremely similar in age (PD: $M = 61.7$ years, $sd = 8.6$; HC: $M = 61.9$ years, $sd = 8.5$) and formal education (PD: $M = 16.0$ years, $sd = 3.7$; HC: $M = 16.0$ years, $sd = 2.6$). Each group was composed of 11 male and 10 female speakers. All participants in the study had good hearing based on a puretone audiometric screening of both ears at frequencies critical to speech comprehension (minimum 30 db HL at 0.5, 1, and 2 KHz, for the better ear).

Participants in the patient group were identified through a self-management clinic at the Baycrest Centre for Geriatric Care in Toronto, Canada. Presence of idiopathic PD was diagnosed by the residing neurologist on the basis of accepted motor criteria (Calne, Snow, & Lee, 1992). The PD participants were tested an average of 3.5 years post-diagnosis and each presented with a mild-moderate motor impairment according to Hoehn and Yahr and Unified Parkinson's Disease Rating Scale (UPDRS) motor criteria. Motor signs in the PD group were characterized as right dominant ($n = 7$), left dominant ($n = 11$), and bilateral ($n = 3$). All but one patient was medicated during testing, as follows: carbidopa/L-dopa ($n = 17$), d2-agonist ($n = 11$), monoamine oxidase-B inhibitor ($n = 1$), catechol-*O*-methyltransferase inhibitor ($n = 1$), amantadine ($n = 3$), and anticholinergics ($n = 1$).

One patient was simultaneously receiving an antidepressant (paroxetine). None of the patients had a history of substance abuse, major medical or neurological difficulties other than PD, or had undergone brain surgery of any sort.

Prior to testing, each PD participant was screened for language ability, cognition, and mood. The absence of formal language impairment (aphasia) was indicated by a speech-language pathologist, as determined from patients' medical records and through clinical observations during testing. An index of speech intelligibility for individuals within the PD group was obtained by eliciting a continuous language sample from each participant (a picture description task) and having these samples rated by an experienced speech-language pathologist and two student clinicians. Following De Bodt (De Bodt, Hernandez-Diaz Huici, & Van de Heyning, 2002), raters categorized the intelligibility of each Parkinsonian speaker on a four-point scale ranging from 0 (''normal–completely undisturbed'') to 3 (''clear and severe disorder''). Inter-rater consensus across the 21 speakers was high (85%). For all language samples, at least two of three raters were in agreement about the intelligibility rating assigned. Results indicated that all members of the PD group were highly intelligible, obtaining an average rating which fell between ''0'' (''normal–completely undisturbed'') and ''1'' (''slight but notable disorder''). Twelve patients were rated as ''completed undisturbed'' by all three raters, and seven additional patients were judged to have normal intelligibility by 2/3 raters, indicating that this dimension was not highly disturbed in the patient group overall.

The absence of dementia in each PD and HC participant was established based on results of the Mattis Dementia Rating Scale (Mattis, 1988). Both groups performed very well on this measure and no participant fit the criteria for dementia (PD: $M = 141.2$, $sd = 2.1$; HC: $M = 142.5$, $sd = 1.8$). All PD and HC participants also completed a battery of neuropsychological tests to assess their: perception of auditory and visual stimuli (Benton face, visual form, and phoneme discrimination subtests; Benton, Sivan, Hamsher, Varney, & Spreen, 1994); memory functions (forward digit span, auditory working memory span); and ''frontal lobe'' abilities of attention and mental set-shifting (Trail-Making Test, Wisconsin Card Sorting Test). Group differences were discerned only on the measure of auditory working memory (PD: $M = 36.2$, $sd = 3.6$; HC: $M = 39.7$, $sd = 2.3$, $t(40) = -3.67$, $p < 0.001$) and the phoneme discrimination subtest (PD: $M = 26.4$, $sd = 3.2$; HC: $M = 28.3$, $sd = 1.7$, $t(40) = -2.46$, $p = 0.02$). The presence/severity of depression was evaluated briefly in all 42 participants using the Hamilton Depression Inventory-Short Form (Reynolds & Kobak, 1995). A significant group difference was found (PD: $M = 4.7$, $sd = 3.4$; HC: $M = 2.3$, $sd = 2.9$, $t(40) = 2.45$, $p = 0.02$), although only three of the 21 PD participants and one of the HC participants were classified as ''mildly'' depressed on this inventory.[1] Table 1 provides a summary of the major characteristics of the two groups.

2.2. Production tasks

Each participant produced a series of utterances in three production tasks. Target utterances were captured onto digital audiotape by a high-quality, head-mounted

[1] While only mildly depressed, there is the (slight) possibility that even such levels of depression could impact the results of analyses. Secondary analyses with the data from these subjects (both PD and HC) were excluded, and the results in the grand majority of cases were the same as when the data were included. Hence, these results are not reported here.

Table 1
Demographic and clinical features of participants with Parkinson's disease (PD) and healthy controls (HC) (mean ± standard deviation)

Variable	GROUP	
	HC ($n = 21$)	PD ($n = 21$)
Sex (f/m)	10/11	10/11
Age (years)	61.9 ± 8.5	61.7 ± 8.6
Education (years)	16.0 ± 2.6	16.0 ± 3.7
Disease duration (years)	N/A	3.9 ± 1.9
Hoehn & Yahr rating score	N/A	2.0 ± 0.5
Motor UPDRS	N/A	14.5 ± 7.1
Mattis Dementia Rating Scale[a]	142.5 ± 1.8	141.2 ± 2.1
Hamilton Depression Inventory[b]	2.3 ± 2.9	4.7 ± 3.4
Hearing Threshold (Screening)	30 dB in better ear	30 dB in better ear

[a]Total score, maximum = 144.
[b]Short form, maximum = 33, increased scores indicate greater impairment.

microphone (sampling rate: 24 KHz, 16 bit). The complete text of all the utterances can be found in Appendix 1.

In all subsequent descriptions, the term "keyword" refers to words in any of the produced utterances that may be highlighted by participants to convey a specific meaning.

2.2.1. Phonemic stress production task

The ability to modulate prosody to mark the semantic distinction between word pairs was evaluated by eliciting sentences containing phonemic stress contrasts from each speaker. Participants viewed a series of (public domain, royalty-free) pictures depicting the noun compound or noun phrase meaning of word pairs that differed in stress only, e.g., *hot* dog (food) vs. hot *dog* (a dog that feels hot). The correct meaning of each picture was paraphrased for the speaker who then identified the picture verbally using the declarative form "this is a _ (e.g., *hot* dog)". Sixteen phonemic stress tokens were recorded from each speaker (2 noun types × 8 tokens).

2.2.2. Contrastive stress production task

To evaluate whether PD speakers could employ prosody to highlight the salience of a target word in their utterances, each speaker produced a series of utterances that varied the location of contrastive stress. The speaker heard a two-sentence narrative (e.g., The *girl jumped* on the *bed*. The *boy sat* on the *steps*) and was also shown the text of the narrative. Then the speaker responded to questions designed to elicit contrastive stress in one of three keyword locations (i.e., the italicized words in the example). An equal number of tokens required the speaker to emphasize the "first", "middle", or "last" keyword of the utterance. For example, the participant was asked, "Did the *boy* or the *girl* jump on the bed?" and then responded with the phrase, "The *girl* jumped on the bed". The order for eliciting stress in different keyword positions was counterbalanced across speakers. Eighteen contrastive stress tokens were elicited from each speaker (3 contrastive stress positions × 6 tokens).

2.2.3. Emotional production task

To evaluate how adults with PD express emotion through prosodic features of their voice, each participant produced sentences in six distinct emotional "tones": neutral, angry, disgusted, happy, sad, and surprised. There were six six-to-eight syllable sentences per emotion, and sentences were composed of basic vocabulary set to simple grammar structures (e.g., "They found it in the room"). To facilitate production of the different emotions, all sentences representing a given emotion were presented in a block before proceeding to another emotion; this order was randomized across subjects and matched between groups. In all cases, speakers were shown the sentence, printed in large black font on a white card, and were encouraged to read it silently and then produce it in a manner consistent with the target emotion (which was printed in small type at the bottom of the card). Subjects were instructed to produce the sentence in a manner "that if someone listened to their recording afterwards, that person would know what emotion the speaker was expressing". Each speaker produced 36 emotional utterances in total (6 emotions × 6 tokens).

2.3. Recording, acoustical, and analysis procedures

Recordings for each participant were obtained during a single session in a quiet testing room, beginning with a set of practice items which familiarized the speaker with task instructions and the recording procedure. No time limits were imposed during the experiment. All participants were tested individually, and were administered the production tasks in a fixed random order. Participants were asked to repeat their production of a trial when it resulted in erroneous production of the target sentence, and they could repeat their production at any time if not fully satisfied with their initial performance (this occurred rarely). The final production was always retained for acoustic analyses. Three distinct acoustic parameters were then extracted from each utterance using Praat speech analysis software (Boersma & Weenink, 2006): fundamental frequency (f0, in Hz), amplitude (in dB), and duration (in ms). It should be noted that several tokens had to be discarded from acoustic analysis as a result of recording error (e.g., tape hiss, unintentional noise caused by the movements of the participant). However, the grand majority of tokens was retained for evaluation.

The method for extracting each acoustic parameter varied according to the type of prosody. For the Phonemic Stress Production task, the target noun compound or noun phrase was isolated within the sentence, each constituent within the target was demarcated (e.g., *hot*-dog), and each constituent was analyzed for mean f0, mean amplitude, and total duration. With respect to the Contrastive Stress Production task, the word at each of the three-keyword locations in the utterance (first, middle, last) was isolated and then the same acoustic measures (mean f0, mean amplitude, duration) were computed for each of the keywords. Finally, acoustic measures were calculated for each utterance as a whole for the Emotion Production task (Banse & Scherer, 1996; Pell, 2001). For f0 and amplitude, the mean and variability of each parameter was computed automatically by Praat (using an autocorrelation algorithm method for f0). F0 and amplitude variability in each utterance were defined as the range between the minimum and maximum values of these respective parameters detected in each utterance. The speech rate of each utterance was computed by measuring the total duration between the onset and offset of the utterance (in s) and then dividing by the total number of syllables in the token.

Prior to statistical analysis, acoustical measures were re-examined by both authors and manual corrections were made to a subset of utterances for which the autocorrelation method produced obvious errors in pitch extraction (less than 5% of all tokens). All acoustic data collected from each production task were subjected to normalization prior to being entered into statistical analyses to allow appropriate comparisons between speakers and speaker groups and to correct for unavoidable differences in microphone distance and instrument recording levels that would have varied somewhat across testing sessions. Normalization should mitigate possible confounding effects of inter-speaker variability as well as voice differences of gender (or gender-related voice changes that arise from aging). Procedures used to normalize tokens varied according to the measure and the nature of the tokens being studied. All normalization procedures were undertaken for a given stimulus in reference to the same speaker within a single production task before any values were averaged across tokens or speakers.[2] Analysis of variance (ANOVA) was used to test differences in the acoustic measures between groups, and significant effects were explored with Tukey's HSD method ($\alpha = 0.05$). Despite our analyses of normalized data, only means of raw data will be presented to the reader in our summary tables to facilitate comparisons with previous reports.

3. Results

3.1. Phonemic stress production

Separate $2 \times 2 \times 2$ analyses of variance (ANOVA) involving the factors of SPEAKER GROUP (HC, PD), KEYWORD ORDER (initial, final), and NOUN TYPE (noun phrase, noun compound) were conducted on each acoustic parameter computed for the Phonemic Stress task. The un-normalized data are summarized in Table 2.

[2]Uniform normalization across token types was not attempted as the different types of tokens had distinct linguistic (and likely acoustic) characteristics; we selected normalization formulae that captured these particular characteristics.

For phonemic stress tokens, f0 data were normalized by dividing the difference between a keyword's mean f0 and the mean f0 of all keywords by the standard deviation of the f0 of all phonemic stress tokens (e.g.: [(mean $f0_{(\text{initial keyword})}$−mean $f0_{(\text{all keywords})}$)/(mean f0 standard $\text{deviation}_{(\text{all keywords})}$)]). The mean amplitude of a keyword was divided by the mean amplitude of all phonemic stress tokens to derive one standardized amplitude data point (e.g.: [(mean $\text{amplitude}_{(\text{initial keyword})}$)/(mean $\text{amplitude}_{(\text{all keywords})}$)]). Duration was normalized by dividing the length of a keyword by the total length of the token of which it was a constituent (e.g.: [$(\text{length}_{(\text{initial keyword})})$/$(\text{length}_{(\text{initial keyword + final keyword})})$]).

With respect to contrastive stress tokens, f0 and amplitude data of each keyword produced by a speaker were normalized by dividing the difference between the mean acoustic measurement of the keyword from the mean acoustic measurement of its utterance by the standard deviation of the acoustic measurement of the utterance (e.g.: [(mean $\text{value}_{(\text{initial keyword})}$−mean $\text{value}_{(\text{whole utterance})}$)/(standard deviation of $\text{value}_{(\text{whole utterance})}$)]). Duration was normalized by dividing the length of each keyword in an utterance by total duration of that utterance (e.g.: [$(\text{length}_{(\text{initial keyword})})$/$(\text{length}_{(\text{whole utterance})})$]).

For emotion tokens, both f0 and f0 range data were normalized by dividing individual data points by the averaged minimum f0 values of all the neutral tokens (e.g.: [mean $\text{value}_{(\text{one utterance})}$/mean minimum $f0_{(\text{all neutral tokens})}$]). Similarly, amplitude and amplitude range data points were standardized by using the mean amplitude value of neutral utterances as a divisor (e.g.: [mean $\text{value}_{(\text{one utterance})}$/mean $\text{value}_{(\text{all neutral tokens})}$]). Speech rate was standardized by dividing the total number of syllables in an utterance by the utterance's duration (e.g. [$\text{syllables}_{(\text{utterance1})}$/$\text{duration}_{(\text{utterance1}}$]).

Table 2
Mean un-normalized values of fundamental frequency (f0), amplitude, and duration of Phonemic Stress Production Task tokens elicited from healthy control (HC) speakers and speakers with Parkinson's disease (PD)

NOUN TYPE	KEYWORD ORDER	f0 (Hz)		Amplitude (dB)[a]		Duration (s)[b]	
		HC	PD	HC	PD	HC	PD
Female speakers							
Noun compound	Initial	188.26 (31.94)	186.00 (26.61)	61.69 (1.84)	61.18 (3.64)	0.25 (0.02)	0.28 (0.03)
	Final	176.15 (29.96)	170.96 (32.70)	57.96 (2.16)	58.17 (3.13)	0.35 (0.05)	0.33 (0.05)
Noun phrase	Initial	182.74 (22.95)	181.44 (25.70)	61.51 (2.54)	60.61 (3.86)	0.29 (0.03)	0.31 (0.04)
	Final	174.19 (21.90)	171.25 (28.15)	59.20 (2.93)	58.03 (3.24)	0.40 (0.06)	0.38 (0.05)
Male speakers							
Noun compound	Initial	115.55 (25.38)	109.59 (20.85)	62.82 (3.54)	63.92 (3.37)	0.25 (0.02)	0.26 (0.04)
	Final	101.95 (14.57)	103.19 (18.34)	57.76 (4.31)	60.02 (3.02)	0.34 (0.03)	0.34 (0.09)
Noun phrase	Initial	111.06 (23.67)	110.83 (21.10)	62.11 (3.60)	63.45 (2.91)	0.28 (0.04)	0.29 (0.04)
	Final	107.28 (14.60)	104.07 (19.06)	59.01 (3.80)	60.56 (2.80)	0.39 (0.06)	0.39 (0.09)

Standard deviation values are in parentheses.
[a]Significant SPEAKER GROUP by KEYWORD ORDER by NOUN TYPE interaction for normalized amplitude measures.
[b]Significant SPEAKER GROUP by KEYWORD ORDER interaction for normalized duration measures.

3.1.1. Fundamental frequency effects

A main effect of KEYWORD ORDER was found ($F(1, 40) = 23.39$, $p < 0.001$); this effect was due to keywords in final position being lower in mean f0 than initial keywords. There was also a significant KEYWORD ORDER by NOUN TYPE interaction ($F(1, 40) = 4.5$, $p < 0.05$) that was accounted for by initial keywords being greater in mean f0 than final keywords regardless of noun type. No group differences were evidenced.

3.1.2. Amplitude effects

We found a main effect of KEYWORD ORDER ($F(1, 40) = 157$, $p < 0.001$) that was attributable to final keywords having lower amplitude than initial keywords. We also found a significant KEYWORD ORDER by NOUN TYPE interaction ($F(1, 40) = 27$, $p < 0.001$) that was due to initial keywords being greater in mean amplitude than final keywords regardless of noun type (note the similarity of this finding to the corresponding interaction in the f0 data). Additionally, initial keywords in noun compounds were greater in amplitude than initial keywords in noun phrases; final keywords from noun phrases were greater in intensity than those from noun compounds.

For amplitude, there was also an interaction involving SPEAKER GROUP, KEYWORD ORDER, and NOUN TYPE ($F(1, 40) = 5.0$, $p < 0.04$). Initial keywords were produced with greater amplitude regardless of NOUN TYPE for both speaker groups. Among HC speakers, the amplitude of final keywords of noun compounds (*hot*dog) was reliably lower than the corresponding keywords of noun phrases (hot *dog*), consistent with linguistic rules of English. While PD speakers displayed a qualitatively similar pattern of amplitude modulation, they did not achieve significant amplitude differences between token types like the HC speakers.

3.1.3. Duration effects

We found an effect of KEYWORD ORDER ($F(1, 40) = 218.7$, $p < 0.001$) that was attributable to final keywords being of longer duration than initial keywords.

There was also a SPEAKER GROUP by KEYWORD ORDER interaction ($F(1, 40) = 8.5$, $p < 0.01$). Post hoc elaboration showed that both PD and healthy speakers produced initial keywords that were shorter than final keywords. However, the difference between mean keyword durations was greater in the tokens spoken by our healthy participants than in those elicited from our PD speakers. Overall, the group difference suggests a reduced ability of PD speakers to accent phonemic distinctions in speech via amplitude and timing modulation.

3.2. Contrastive stress production

The normalized data representing each acoustic parameter were subjected to separate $2 \times 3 \times 3$ analyses of variance (ANOVA) involving the factors of SPEAKER GROUP (HC, PD), KEYWORD ORDER (initial, medial, final), and PLACE OF EMPHASIS (first, middle, last). Un-normalized contrastive stress data values are displayed in Table 3.

3.2.1. Fundamental frequency effects

There was an effect of KEYWORD ORDER ($F(2, 72) = 110.14$, $p < 0.001$) that was due to initial keywords being lower in f0 than all the other keywords. As well, we found a PLACE OF EMPHASIS effect ($F(2, 72) = 3.41$, $p < 0.05$) that can be explained by significantly decreasing mean f0 levels commensurate with the place of emphasis (i.e., first emphasis keyword f0 > middle emphasis keyword f0 > last emphasis keyword f0). Moreover, an interaction between these two factors was found ($F(4, 144) = 68.58$, $p < 0.001$). Tukey tests showed the interaction to be accounted for by higher or markedly elevated values in tokens that resulted from consistent (as opposed to inconsistent) pairing of levels of KEYWORD ORDER and PLACE OF EMPHASIS factors (e.g., when an initial word was required to be emphasized, the initial keyword had higher f0 values than medial or final words in this emphasis condition).

We further found an interaction involving SPEAKER GROUP, KEYWORD ORDER, and PLACE OF EMPHASIS ($F(4, 144) = 11.02$, $p < 0.001$). It is noteworthy that both PD and healthy speakers raised the mean f0 of initial, medial, and final keywords when concepts in each of these positions were supposed to be highlighted. While within group patterns of performance were similar, significant group differences arose when comparing the productions of words in medial and final positions; PD speakers produced sentence-final emphasized words with significantly lower f0 than healthy speakers. PD speakers also produced significantly higher f0 for sentence initial keywords when sentence-final words had to be stressed; HC speakers did not (see Fig. 1).

3.2.2. Amplitude effects

The effect of KEYWORD ORDER was significant ($F(2, 72) = 186.94$, $p < 0.001$) and is explained by consistently decreasing amplitude levels corresponding to the order in which the keyword appeared (i.e., initial keyword amplitude > medial keyword amplitude > final keyword amplitude). A KEYWORD ORDER by PLACE OF EMPHASIS interaction was found ($F(4, 144) = 35.52$, $p < 0.001$). As with the corresponding interaction in the f0 data, this interaction was explained by markedly elevated amplitude in tokens that resulted from consistent (as opposed to inconsistent) pairing of levels of KEYWORD ORDER and PLACE OF EMPHASIS factors.

Table 3
Mean un-normalized values of fundamental frequency (f0), amplitude, and duration of keywords from Contrastive Stress Production Task as functions of place of emphasis and keyword order for healthy control (HC) subjects and subjects with Parkinson's disease (PD)

	KEYWORD ORDER	HC			PD		
		PLACE OF EMPHASIS			PLACE OF EMPHASIS		
		First	Middle	Last	First	Middle	Last
Female speakers							
f0 (Hz)[a]	Initial	203.2 (20.8)	185.1 (26.7)	182.2 (21.0)	204.8 (15.0)	197.1 (18.9)	197.6 (20.1)
	Medial	177.1 (28.8)	200.4 (36.4)	176.0 (14.7)	173.3 (23.9)	193.0 (22.3)	178.6 (16.7)
	Final	158.7 (20.8)	157.7 (21.1)	183.5 (23.6)	166.9 (27.5)	163.8 (25.6)	177.8 (30.9)
Amplitude (dB)[b]	Initial	63.8 (2.1)	63.9 (2.1)	65.8 (5.0)	63.8 (3.1)	64.4 (3.4)	64.5 (3.6)
	Medial	59.8 (1.9)	63.9 (2.3)	63.2 (5.3)	59.9 (2.2)	63.2 (3.5)	61.0 (3.7)
	Final	59.3 (2.5)	60.2 (2.5)	61.7 (3.9)	59.2 (2.6)	59.4 (3.8)	59.9 (3.2)
Duration (s)	Initial	0.3 (0.0)	0.2 (0.0)	0.2 (0.0)	0.3 (0.0)	0.3 (0.0)	0.2 (0.0)
	Medial	0.2 (0.0)	0.3 (0.0)	0.3 (0.0)	0.2 (0.0)	0.3 (0.0)	0.3 (0.0)
	Final	0.2 (0.0)	0.2 (0.0)	0.4 (0.0)	0.2 (0.0)	0.2 (0.0)	0.4 (0.0)
Male speakers							
f0 (Hz)[a]	Initial	122.6 (33.8)	112.4 (24.8)	112.7 (24.5)	118.3 (22.9)	116.1 (22.6)	120.1 (23.6)
	Medial	104.0 (21.4)	118.7 (29.0)	108.7 (21.8)	105.1 (20.1)	116.9 (32.6)	112.0 (24.3)
	Final	98.4 (19.5)	97.6 (20.5)	115.7 (28.2)	100.9 (17.5)	106.6 (23.0)	107.6 (23.7)
Amplitude (dB)[b]	Initial	62.3 (5.0)	62.2 (3.0)	62.9 (2.9)	64.5 (2.7)	64.3 (2.4)	64.7 (2.3)
	Medial	58.2 (4.5)	62.1 (3.3)	60.3 (3.2)	61.9 (3.5)	63.4 (2.8)	62.0 (2.9)
	Final	57.0 (4.9)	58.2 (3.1)	58.5 (3.7)	60.9 (2.9)	60.8 (3.3)	60.2 (3.2)
Duration (s)	Initial	0.3 (0.1)	0.3 (0.0)	0.2 (0.0)	0.3 (0.1)	0.3 (0.0)	0.2 (0.0)
	Medial	0.2 (0.0)	0.3 (0.1)	0.3 (0.0)	0.2 (0.1)	0.3 (0.1)	0.3 (0.1)
	Final	0.2 (0.0)	0.2 (0.0)	0.4 (0.0)	0.2 (0.0)	0.2 (0.0)	0.4 (0.1)

Standard deviation values are in parentheses.
[a]Significant SPEAKER GROUP by KEYWORD ORDER by PLACE OF EMPHASIS interaction for normalized f0 measures.
[b]Significant SPEAKER GROUP by KEYWORD ORDER by PLACE OF EMPHASIS interaction for normalized amplitude measures.

An effect of SPEAKER GROUP was found ($F(1, 36) = 5.77$, $p < 0.05$), owing to PD speakers producing tokens that were significantly lower in intensity than tokens produced by healthy speakers. There was also a SPEAKER GROUP by KEYWORD ORDER by PLACE OF EMPHASIS interaction ($F(4, 144) = 3.28$, $p < 0.05$) that can be explained by the two speaker groups performing similarly with the exception of sentence-final words. When emphasis was required at the beginning of an utterance, PD speakers produced the sentence-final word with significantly greater intensity than when emphasis was required at the end of the utterance. The HC speakers correctly emphasized the sentence-initial word (i.e., the opposite pattern).

3.2.3. Duration effects

A PLACE OF EMPHASIS effect was found ($F(2, 72) = 62.50$, $p < 0.001$); this effect could be explained by last-emphasized words being longer than words emphasized in first and middle positions. The interaction between KEYWORD ORDER and PLACE OF

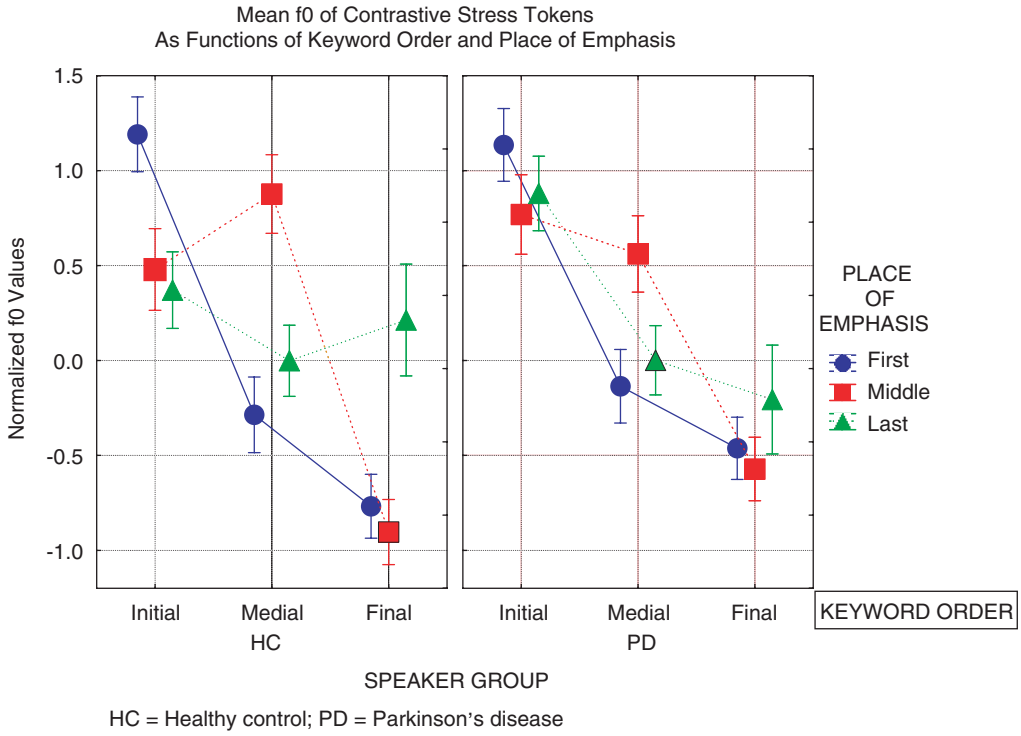

Fig. 1. Significant three-way interaction involving factors of SPEAKER GROUP, KEYWORD ORDER, and PLACE OF EMPHASIS on mean fundamental frequency (f0) measures for productions of tokens of contrastive stress.

EMPHASIS was also significant ($F(4, 144) = 282.80$, $p < 0.001$). Post hoc testing of this last interaction showed tokens arising from consistent pairing of levels of KEYWORD ORDER and PLACE OF EMPHASIS factors to be in greater in length. There were no significant effects involving SPEAKER GROUP for this measure.

3.3. Emotion production

Five distinct acoustic parameters were analyzed for each token in the emotion production task: mean f0, mean amplitude, speech rate, f0 variability, and amplitude variability. Each normalized measure was considered in a separate 2×5 ANOVA involving SPEAKER GROUP (HC, PD) and EMOTION TYPE (anger, disgust, happiness, sadness, surprise)[3] (see Table 4 for un-normalized data).

[3]While the rank ordering of some of the acoustic data for our speakers' simulated emotions is not fully consistent with some descriptions (e.g., compare our "anger" speech rate data with Banse and Scherer's (1996) findings), we have confidence that the intended emotions were accurately portrayed for most of the tokens since they were accurately recognized well above chance levels in a concurrent study involving naïve listeners (Pell et al., 2006). Acoustic differences across studies are likely to reflect differing methods for eliciting the emotions and collecting the speech samples.

Table 4
Mean un-normalized values of fundamental frequency (f0), amplitude, speech rate, f0 range, and amplitude variability of Emotional Production Task tokens for healthy control (HC) subjects and subjects with Parkinson's disease (PD). Standard deviations are in parentheses

EMOTION	f0 (Hz)[a]		Amplitude (dB)[b]		Speech rate (syl/s)		f0 range (max–min)		Amplitude range(max–min)	
	HC	PD	HC	PD	HC	PD	HC	PD	HC	PD
Female speakers										
Anger	192.8 (23.4)	214.8 (27.6)	61.2 (2.4)	60.3 (3.2)	4.0 (0.7)	3.9 (0.3)	123.3 (38.2)	158.6 (36.9)	35.3 (5.1)	39.0 (5.3)
Disgust	174.2 (18.2)	191.1 (32.1)	58.5 (2.8)	57.9 (2.4)	3.8 (0.7)	3.8 (0.4)	91.1 (20.7)	102.0 (25.1)	30.4 (3.5)	32.8 (4.5)
Happiness	222.5 (28.4)	228.7 (31.8)	61.1 (2.9)	59.6 (3.0)	4.6 (0.6)	4.5 (0.5)	161.9 (32.4)	182.5 (58.8)	31.6 (2.4)	35.1 (5.4)
Sadness	186.0 (19.0)	184.2 (23.9)	57.7 (2.8)	56.5 (2.6)	4.1 (0.7)	4.0 (0.4)	79.9 (18.8)	72.9 (13.2)	30.7 (3.4)	34.4 (4.6)
Surprise	259.1 (41.7)	271.8 (44.1)	62.3 (2.6)	60.6 (3.7)	4.0 (0.7)	3.9 (0.3)	254.4 (55.6)	258.3 (84.5)	30.9 (3.8)	32.2 (5.0)
Male speakers										
Anger	129.1 (23.9)	123.2 (22.5)	60.4 (4.0)	61.0 (2.4)	4.2 (0.6)	4.3 (0.6)	82.2 (34.4)	73.1 (20.4)	35.6 (5.3)	36.3 (3.5)
Disgust	119.7 (21.4)	112.2 (24.4)	58.4 (3.5)	58.7 (3.3)	4.2 (0.6)	4.2 (0.7)	68.9 (24.5)	58.4 (23.2)	31.5 (5.0)	35.5 (3.9)
Happiness	142.2 (28.6)	130.7 (25.8)	60.5 (4.0)	60.4 (3.2)	5.0 (0.7)	4.8 (0.7)	103.8 (38.7)	82.7 (19.5)	32.1 (4.7)	35.2 (3.9)
Sadness	113.9 (22.6)	108.3 (19.5)	57.0 (4.3)	57.8 (3.3)	4.3 (0.6)	4.6 (0.7)	55.3 (28.3)	47.5 (16.3)	32.3 (4.5)	34.7 (3.2)
Surprise	173.6 (30.1)	145.8 (31.1)	62.9 (4.3)	62.9 (3.4)	4.5 (0.6)	4.4 (0.6)	158.3 (50.9)	114.7 (33.1)	29.1 (5.5)	30.5 (3.1)

[a]Significant SPEAKER GROUP by EMOTION interaction for normalized f0 measures.
[b]Significant SPEAKER GROUP effect for normalized amplitude measures.

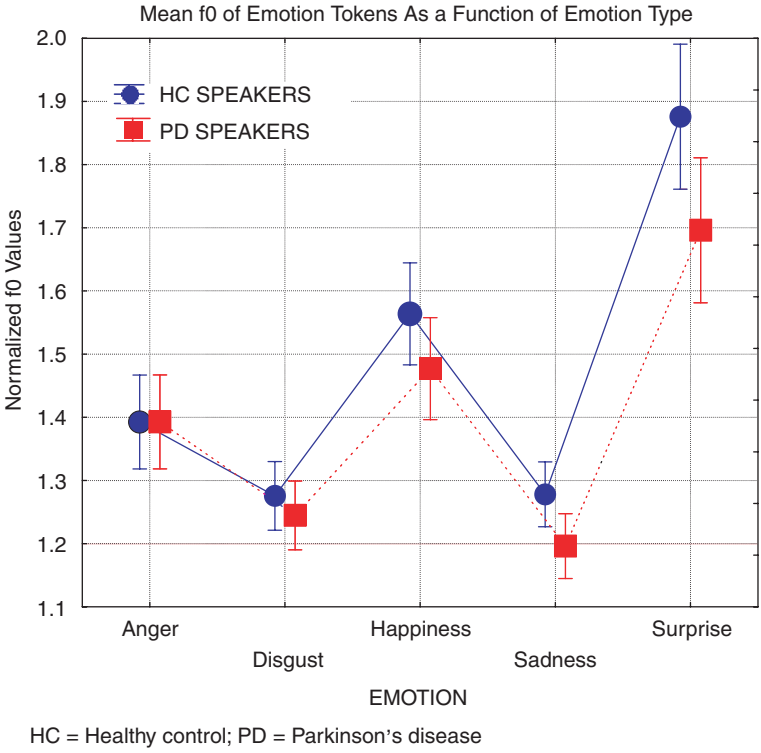

Fig. 2. Significant two-way interaction involving factors of SPEAKER GROUP and EMOTION on mean fundamental frequency (f0) measures for productions of emotional tokens.

3.3.1. *Fundamental frequency effects*

With respect to mean utterance f0, EMOTION TYPE was found to be significant ($F(4, 160) = 145.79$, $p < 0.001$). Expressed as ranks of emotion types, f0 varied as follows: surprise > happiness > anger > (disgust, sadness). There was a significant SPEAKER GROUP by EMOTION interaction ($F(1, 40) = 3.39$, $p < 0.05$) for mean utterance f0 (see Fig. 2). A specific cross-group difference was found for the expression of surprise wherein Parkinsonian speakers conveyed this emotion with a significantly lower f0 than the healthy participants. The patterns of within group differences were distinct as well. Upon analysis of HC speakers' elicited speech, it was found that surprise tokens were produced with significantly greater f0 than all other tokens. Happiness tokens, with the next highest mean f0 values, were also significantly greater in f0 than tokens of anger, disgust, and sadness; these last three tokens were not different from each other in terms of f0. A qualitatively different pattern was in evidence during perusal of Parkinsonian speech. PD speakers produced surprise tokens with the highest f0 in the emotion production task. Happy- and angry-sounding tokens were statistically similar with respect to f0, and these two groups of tokens were, as a group, higher in mean f0 than the statistically equivalent sad- and disgusted-sounding tokens. These within group differences can be summarized in the following manner: HC speakers: surprise > happiness > (anger, sadness, disgust); PD speakers: surprise > (happiness, anger) > (sadness, disgust).

3.3.2. Amplitude effects

There was a main effect of EMOTION TYPE ($F(4, 160) = 52.58$, $p < 0.001$). This effect arises from the following rank ordering of emotions: surprise > (anger, happiness) > disgust > sadness. A SPEAKER GROUP effect was found in amplitude analyses ($F(1, 40) = 4.36$, $p < 0.05$) because HC speakers produced emotional utterances with greater mean amplitude than PD speakers.

3.3.3. Speech rate effects

EMOTION TYPE was found to be significant ($F(4, 160) = 37.06$, $p < 0.001$), Speech rates across the emotions can be summarized as follows: happiness > (sadness, surprise, anger) > disgust. There was no interaction or main effect involving SPEAKER GROUP.

3.3.4. Fundamental frequency variability effects

A main effect of EMOTION TYPE was found ($F(4, 160) = 118.12$, $p < 0.001$). As with f0, the effect is accounted for by this ranking of emotion types: surprise > happiness > anger > (disgust, sadness). SPEAKER GROUP did not achieve significance from this analysis.

3.3.5. Amplitude variability effects

We found an EMOTION TYPE effect: ($F(4, 160) = 26.11$, $p < 0.001$). This main effect can be accounted for by the following amplitude variability differences in emotion: anger > (sadness, disgust, happiness) > surprise. This acoustic parameter did not differentiate between speaker groups.

4. Discussion

Multiple significant group differences or group interactions were found upon analyzing the acoustic parameters across production tasks, and group amplitude differences were the most robust. However, our hypotheses were not fully borne out in that differences in all acoustic parameters across groups were not found in every prosodic production task. Tokens elicited from PD speakers were lower in amplitude than the tokens spoken by healthy participants, or lower in contexts that required emphasis to convey meaning in all three production tasks. Meanwhile, f0 was found to be aberrant among the PD speakers in producing only some contrastive and affective distinctions, and duration measures revealed anomalies between HC and PD speakers merely in phonemic stress production. These collective findings reveal a differential pattern of deteriorating control in speech production among PD patients as a likely source of the attenuated acoustic precision and suggest that PD is associated with varied changes at the acoustic level that cannot always be easily generalized to all speech contexts.

The most salient conclusion supported by our results is that objectively quantifiable decline of acoustic qualities of Parkinsonian speech begins early in Parkinson's disease progression, a situation that previously had only been hinted at in limited linguistic and acoustic contexts by exploratory studies (Harel, Cannizzaro, & Cohen et al., 2004; Harel, Cannizzaro, & Snyder, 2004). Furthermore, despite being tested while medicated at a time of day when their symptoms were presumably least severe, and despite having no drastic impairments to primary cognitive domains or language deficits, our PD speakers experienced acoustic alterations in their speech tokens. Importantly, their speech

productions were found to be less well understood than those of the healthy speakers by normal listeners in a concurrent study (Pell et al., 2006). We arrived at these results in spite of the fact that expert listeners rated the Parkinsonian speech as generally intelligible and unimpaired, further evidencing the prominent impact of PD on speech in functional contexts. Our data can be interpreted as evidence for a sensitivity advantage that autocorrelatory methods have over employing trained listeners to quantify the physical qualities of speech. A secondary conclusion is that, even among a single group of PD speakers, speech production patterns vary according to the linguistic attributes of what had to be produced (Goberman & Elmer, 2005; Ho, Bradshaw, Iansek, & Alfredson, 1999). Thus, to better understand speech and language mediation in PD and to design more appropriate interventions, it is important to identify both the types and extent of communication impairments among PD patients.

4.1. Characteristics of Parkinsonian speech

Previous work has suggested that (subclinical) acoustic alterations occur in the speech of persons whose PD has not been diagnosed and that these symptoms continually worsen until the first administrations of drugs designed to increase dopamine levels, although such speech signs appear to vary across speakers (Harel, Cannizzaro, & Cohen et al., 2004; Harel, Cannizzaro, & Snyder, 2004). What has been found is not particularly amenable to generalization however, given that many of the speech samples evaluated were obtained under uncontrolled contexts and from very few individuals. Some of Harel et al.'s (Harel, Cannizzaro, & Cohen et al., 2004; Harel, Cannizzaro, & Snyder, 2004) PD patients used their voices professionally, further distinguishing their patterns of speech from the typical speaker with PD. The present results clearly verify early onset of speech symptoms in PD and are more robust and potentially more representative, given our large number of comparable PD speakers and our careful construction and elicitation of speech tokens.

With respect to the substance of our findings, it is noteworthy that only the analysis of amplitude revealed overall similarity with previous data (e.g., Canter, 1963; Ho, Iansek, & Bradshaw, 2001), i.e., generally pervasive reductions in mean amplitude among PD speakers when they attempted to distinguish shades of meaning or convey affect. There was a more subtle pattern of group differences for the other acoustic parameters, suggesting that early stages of PD have lesser impact on other acoustic parameters in speech, a situation which could account for the unexpected lack of overall changes in f0 or duration in Parksinsonian speech (e.g., Goberman & Elmer, 2005; Penner et al., 2001). The early stage of PD progression among our PD speakers may account for our results diverging somewhat from previous findings (of a greater variety of acoustic alterations in speech production); at the time of testing, our PD participants were at earlier stages than subjects in previous studies (e.g., Illes et al., 1988).

The acoustic profiles of the PD speakers are of interest for a further reason: note that when acoustic deficits were evident, overall patterns of speech production among the PD participants matched healthy speech patterns (review Figs. 1 and 2). F0 and duration group differences were accounted for by distinctions in specific contexts rather than uniform discrepancy in all production tasks (e.g., lower f0 in PD relative to HC speakers' conveying of surprise; reduced duration gaps between keywords differing in phonemic stress by PD speakers as compared to healthy controls). One has to question the degree to which the representations and motor plans for the various linguistic forms were damaged

in the PD patients. Our data do not permit an evaluation of this notion. Rather, it is worth considering in future studies.

Another issue arises from inspection of the emotional sentences. Specifically, we found far fewer overall or even specific group differences in the production of emotional tokens than was expected from the existing literature. Both clinical and empirical observations have cited "flattened" or "less intense" affect in Parkinsonian speech (Caekebeke et al., 1991; Kent & Rosenbek, 1982; Pell et al., 2006). Given our present findings and those of our already published study (Pell et al., 2006), it is possible that general reductions in speech amplitude as well as declines in f0 in traditionally "intense-sounding" emotions (e.g., surprise) were enough to contribute to the perception of "flattened" affective speech. By extension, the observed changes may contribute to the notion that PD patients have reduced pragmatic ability (McNamara & Durso, 2003) or lessened adaptability of volume in speech (Ho, & Iansek et al., 1999; Ho, & Bradshaw et al., 1999). Our results do not refute the possibility that PD patients have actual difficulties in discerning how to interact appropriately in conversation, but they do highlight the possibility that listeners may be construing the speech production restrictions of PD speakers as signs of pragmatic inadequacy rather than considering the speech restrictions as difficulties stemming from predominantly motor limitations (e.g., PD speakers may not wish to produce utterances at an amplitude that is inappropriately low for a given conversational context but may have no choice in the matter).

4.2. Basal Ganglia and speech production

PD is characterized by progressive degeneration of brain structures which include, but are not limited to, the basal ganglia. It may thus be reasonable to assume, given our present and past investigations, that features of motor and communicative dysfunction can be linked (at least in part) to basal ganglia deterioration. Consider that we have found in a large single sample of PD patients significant acoustic deficits in their production of ubiquitous linguistic devices and modes of expression; these differences decreased the accuracy with which healthy naïve listeners perceived the Parkinsonian speech in our previous study (Pell et al., 2006). Finally, the present PD patients have presented some difficulties in comprehending emotional prosody (Pell & Leonard, 2003). It appears that *overall communication*, and not simply speech production, can be problematic for patients at the early stages of PD; thus, this problem is greater and begins earlier for patients with PD than was generally recognized by clinicians or interlocutors. The basal ganglia may impact on communication through its multiple connections with cortical and subcortical structures known to be involved in information processing across a number of (debated) cognitive domains (Breitenstein, Van Lancker, Daum, & Waters, 2001; Cohen, Branch, & Hynd, 1994; Tettamanti et al., 2005).

4.3. Limitations of the present study

Our results do not speak to the potential acoustic deficits that may characterize other parts of speech across different groups of PD patients. With the exception of amplitude, other acoustic parameters were not impacted consistently among our PD patients across different linguistic and affective speech forms. Thus, there is a need to study an even wider variety of language behaviors among PD patients and healthy matched controls.

Our investigation also does not address specific long-term speech deficits that arise from PD. However, we can assume that greater acoustic differences and deficits will present themselves over time as a function of PD progression. Longitudinal studies that examine multiple language forms (in a manner similar to our approach) will inform this issue. Also, as we examined individuals who have not as yet received speech therapy (having only been administered medication), we are unable to ascertain the potential effects of early speech and language intervention on Parkinsonian speech.

It also bears noting that our present speech samples were derived from non-spontaneous conditions; the prosodic alterations that we found may thus not be fully comparable to changes found in the conversational speech of PD patients (Brown & Docherty, 1995; Kempler & Van Lancker, 2002). Two patterns of findings have bearing on this matter. First, acoustic changes have been found in both non-spontaneous Parkinsonian speech samples collected from reading or repetition tasks (e.g., Forrest, Weismer, & Turner, 1989) and in spontaneous speech tokens (e.g., Illes, 1989). Second, in studies where both types of speech tokens were collected from a single sample of patients, the spontaneous speech tokens were found to be even more significantly acoustically altered than non-spontaneous tokens (Brown & Docherty, 1995; Kempler & Van Lancker, 2002). Thus, while the prosodic shifts in the present elicited Parkinsonian speech may not be equivalent to (potential) changes in the unprompted verbalizations of the PD speakers, there is nonetheless a good possibility that the acoustic distinctions we found reflect genuine alterations in the PD patients' spontaneous speech.

4.4. Clinical implications

Together with previous data (Harel, Cannizzaro, & Cohen et al., 2004; Harel, Cannizzaro, & Snyder, 2004; Illes, 1989; Illes et al., 1988), our data strongly suggest that acoustic differences in Parkinsonian speech can be detected at the stage of diagnosis of PD. Speech deterioration can occur early in PD progression and can impact listener comprehension for basic linguistic and emotional forms (Pell et al., 2006); it would likely be of benefit to PD patients and their interlocutors to verify this possibility by objective means. Brief mention must also be made regarding medication. While it is impossible within the confines of the present study to discern whether medication had any positive or appreciable effect on the speech production of the PD speakers (as we did not test speakers both in medicated and unmedicated states), the current medicated PD speakers nonetheless presented significant acoustic alterations in their speech. Our findings are inconsistent with some previous suggestions (Goberman et al., 2002; Goberman, Coelho, & Robb, 2005; Harel, Cannizzaro, Cohen et al., 2004; Harel, Cannizzaro, & Snyder, 2004); given that the effects of any type of intervention on PD are still not well understood, discrepancies regarding treatment results are unavoidable in the literature. Nevertheless, the sum total of our present and past findings (Pell et al., 2006; Pell and Leonard, 2003) argues for early evaluation of PD patients for speech and communication symptoms.

The current data also suggest that it is important to examine different contexts of spoken language in patients with PD rather than any one single language form or acoustic parameter since different parts of the speech elicited in the current study were characterized by different acoustic features. Despite some evidence that the length of materials to be produced verbally may be linked to prosodic abnormality and our finding that group differences occurred across different measures of amplitude for all production

Table A1

Text of phonemic stress tokens sequence: noun phrase/noun compound	Text of contrastive stress tokens		
This is a black *board*/*black*board.	Brenda lost her *keys*.	*Jack* works in Paris.	*Sue* wrote the letter.
This is a blue *"J"*/*blue* jay.	*Jane* bought a wallet.	Bob *lives* in London.	Mary read the *book*.
This is a dark *room*/*dark*room.	Jane *bought* a wallet.	Jack works in *Paris*.	Sue *wrote* the letter.
This is a green *house*/*green*house.			
This is a high *chair*/*high*chair.			
This is a hot *dog*/*hot*dog.	The dog is *sitting* under the table.	The girl *jumped* on the bed.	The teacher *arrived* late.
These are white *caps*/*white*caps.	The cat is sleeping on the *chair*.	The boy sat on the *steps*.	The *student* left early.
This is a white *house*/*White* House.	The *dog* is sitting under the table.	The *girl* jumped on the bed.	The teacher *arrived* late.

Text of emotion tokens

"Anger" tokens	"Disgust" tokens	"Happiness" tokens	"Neutral" tokens
Stop telling lies.	He has terrible breath.	I can't wait to see you.	I'm leaving at five o'clock.
That car just splashed me.	There are worms in my cereal.	Let's go to the beach.	She grew up on a farm.
You burnt me with your cigarette.	We found the rotten food.	We're going on vacation.	The hat was brown.
"Sadness" tokens	"Surprise" tokens		Tokens common to all conditions
I didn't make the team.	Look at all these gifts.		Jane is coming tomorrow.
No one survived the accident.	We won the lottery.		They found it in the room.
They won't be back for a long time.	You're all here.		They're going to the movies.

Text of all tokens elicited and recorded from all speakers. Italicized font denotes words upon which speakers were predisposed by experimenter to place emphasis.

tasks, there was no single acoustic parameter that was consistently affected in the PD speakers across all prosody contexts. These findings are consistent with trends in the literature that show variable patterns of speech impairment and differential treatment outcomes to be more likely across patients with PD (see Goberman et al., 2002; Goberman & Coelho, 2002a, 2002b; Schulz & Grant, 2000 for a review). Such trends counsel a more prudent course of action, i.e., intervention that is more specific to the characteristics of individual linguistic or communicative forms that is commenced at early stages of PD progression.

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Appendix 1

For a text of all tokens elicited and recorded from all speakers, see Table A1.

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