Patterns of lung volume use during an extemporaneous speech task in persons with Parkinson disease

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Abstract

This study examined patterns of lung volume use in speakers with Parkinson disease (PD) during an extemporaneous speaking task. The performance of a control group was also examined. Behaviors described are based on acoustic, kinematic and linguistic measures. Group differences were found in breath group duration, lung volume initiation, and lung volume termination measures. Speakers in the control group alternated between a longer and shorter breath groups. With starting lung volumes being higher for the longer breath groups and lower for shorter breath groups. Speech production was terminated before reaching tidal end expiratory level. This pattern was also seen in 4 of 7 speakers with PD. The remaining 3 PD speakers initiated speech at low starting lung volumes and continued speaking below EEL. This subgroup of PD speakers ended breath groups at agrammatical boundaries, whereas control speakers ended at appropriate grammatical boundaries.

Learning outcomes: As a result of participating in this exercise, the reader will (1) be able to describe the patterns of lung volume use in speakers with Parkinson disease and compare them with those employed by control speakers; and (2) obtain information about the influence of speaking task on speech breathing.

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1. Introduction

General respiratory dysfunction has been reported to occur frequently in patients with Parkinson disease (PD), particularly in the advanced stages (Izquierdo-Alonso, Jiminez-Jiminez, Cabrera-Valdivia, & Mansilla-Lesmes, 1994). Especially affected are maneuvers that require speed, effort, and maximal range of ability (De la Torre, Mier, & Boshes, 1960; Hovestadt, Bogaard, Meerwaldt, van der Meche, & Stigt, 1989; Liker & Woolf, 1968; MacIntosh, 1977; Nakano, Bass, & Tyler, 1972; Neu, Connolly, Schwertley, Ladwig, & Brody, 1967). Many breathing impairments in idiopathic PD appear to be related to rigidity of the chest wall (MacIntosh, 1977; Nakano et al., 1972; Paulson & Tafrate, 1970). Generalization of these findings to breathing for speech purposes is difficult because the entire range of respiratory function is not used during speech production (Kent, Kent, & Rosenbek, 1987), and speech breathing requires different neural control than breathing at rest (Plum, 1974; Von Euler, 1982). In addition, other speech production subsystems (e.g., larynx, pharynx, and oral articulators) interact with the respiratory system during the production of speech (Netsell, 1983). Two primary methodologies have been used in studying speech breathing mechanics for normal and disordered speakers. The first method evaluates features of the air stream (e.g., pressure, flow, and volume expenditure) and the second examines measured chest-wall movements or kinematics.

1.1. Measurement of air stream

Lower than normal oral pressure during consonant production has been demonstrated in people with PD (Ewanowski, 1964; Mueller, 1971; Murry, 1983; Netsell, Daniel, & Celesia, 1975), especially when the disease is severe (Ewanowski, 1964; Marquardt, 1973). Smitheran and Hixon (1981) reported that oral pressure can be a good estimate of the pressure generated by the respiratory apparatus in specific speech contexts. This ''driving pressure'' is delivered to the larynx and upper-airway structures for sound generation. The influence of these structures on the air stream makes it difficult to determine whether the lower than expected oral pressure seen in speakers with PD is generated by the respiratory system or a problem with laryngeal and/or upper-airway valving. Solomon and Hixon (1993) reported oral pressure to be lower for speakers with PD, but estimated tracheal pressure did not differ between the PD and control speakers. This difference suggests that poor oral closure and/or velopharyngeal valving problems affect measures of oral pressure, thus it is not a good indicator of respiratory impairment.

Contradictory reports exist regarding lung volume expenditure per syllable and per second, as an indication of airflow during speech. Mueller (1971) measured flow at the airway opening for 10 men and women with PD on a sustained vowel task and reported no differences from control speakers. Smith (1964) reported on volume expenditure per syllable during rapid repetition of syllables in 23 men and women with PD. Participants used 0.070 L per syllable during syllable repetition tasks. At the time of publication no normal data for comparison were available; however, Smith suspected that the participants used more air per syllable than normal speakers. This conclusion may have been accurate based on data since published for the same task produced by healthy adults (0.040 and 0.053 L per syllable for men and women, respectively; Warren & Wood, 1969). Smith's

conclusions, however, may speak more to problems with laryngeal and articulatory valving than to respiratory performance. Solomon and Hixon (1993) reported no significant differences in lung volume excursion per syllable and per second for PD versus control groups.

In summary, air stream measures evaluate features, that are generated by the respiratory system and are then acted upon by the larynx and upper articulators. This interaction makes it difficult to comment directly on differences in respiratory behaviors that may affect speech production for speakers with PD. One additional concern regarding these types of measures is their limited implications for breathing during running speech. Speech tasks used to measure flow and pressure are highly constrained (e.g., syllable repetition or sustained vowels) and do not correlate with spontaneous speech production (Kent & Kent, 2000).

1.2. Measurement of kinematics

Measures of chest wall shape changes via kinematic assessment allow investigators look directly at the behavior of the respiratory system during speech production. Several case study reports of speech respiratory dysfunction can be found in the literature (Anthony & Farquharson, 1975; Hunker, Bless, & Weismer, 1981; Nakano, Zubick, & Tyler, 1973). In addition, five studies involving systematic measurements of chest-wall movements are available (Ewanowski, 1964; Huber, Stathopolous, Ramig, & Lancaster, 2000; Lethlean, Chenery, & Murdoch, 1990; Murdoch, Chenery, Bowler, & Ingram, 1989; Solomon & Hixon, 1993). These studies involved various speech tasks, such as sustained vowels, syllable-repetition tasks, sentence repetition, reading a standard passage and monologue production. Murdoch et al. (1989), and Lethlean et al. (1990) reported irregularities in chest wall movements in the tasks of vowel prolongation and syllable repetition. However, problems with measurement and interpretation make any conclusions drawn from these data tenuous (Hoit, 1994). Ewanowski (1964) measured rib cage circumference changes in 12 women with PD and 12 neurologically normal women. Results suggested no significant difference between extent and control of rib cage movement across participant groups. This study did not include simultaneous observation of abdominal movement, and therefore does not give a clear picture of speech breathing. Solomon and Hixon (1993) reported that during speech breathing, rib cage volume was smaller and abdominal volume was larger at the initiation of breath groups for speakers with PD than for healthy control speakers. Speakers with PD also produced fewer words, spent less time producing speech per breath group, and tended to have a faster interpause speech rate than did healthy control speakers. No differences were found in inspiratory behaviors between the two groups. These results provide indirect evidence of reduced relative compliance of the rib cage to the abdomen for speakers with PD. Huber et al. (2000) also reported increased reliance on the abdomen for changing lung volume compared to controls. Increased variability in respiratory movements compared to controls was also reported by Huber et al. (2000). This variability was found to increase when speakers with PD were cued to increase loudness. The control group did not demonstrate this increase in variability.

Based on this group of studies, it is clear that individuals with PD exhibit respiratory behaviors during speech production, which differ from healthy control speakers.

Increased variability for an individual speaker across multiple trials as well as differences in performance related to the type of speaking task serves to highlight the need for further examination of the role of respiratory function for speech in individuals with PD.

1.3. Speech tasks

The majority of studies using kinematic measures to look at speech breathing in PD have employed speech tasks that were highly structured (except Solomon & Hixon, 1993 who included a monologue task). Research on normal speakers has demonstrated changes in performance related to the speech task. A number of authors have reported relationships between lung volume and length of the utterance during a reading task, such that a longer sentence spoken on one breath will result in a greater lung volume excursion (Hixon, Goldman, & Mead, 1973; Hodge & Rochet, 1989; Hoit & Hixon, 1986; Wilder, 1983; Winkworth, Davis, Ellis, & Adams, 1994, 1995). Differences have also been found for the type of utterance to be spoken and the location of that utterance within a paragraph. Acoustic and kinematic measures have been used to examine differences between structured reading tasks and spontaneous speaking tasks. Reading is associated with an increased speech rate, fewer dysfluencies, and a decreased range of initiation lung volumes and volume expired per breath group, compared to spontaneous speech (Hodge & Rochet, 1989; Solomon & Hixon, 1993). These differences are likely related to linguistic factors. For example, during reading inspirations are largely taken at sentence boundaries or other positions appropriate to the grammatical structure (Conrad, Thalacker, & Shonle, 1983; Henderson, Goldman Eisler, & Skarbek, 1965; Hixon et al., 1973). The association between linguistic factors and lung volumes suggests that the speaker anticipates the length or type of utterance. Winkworth et al. (1995) report a greater range of volume expired in spontaneous speech. They also report that an association between initiation lung volumes and breath group length in spontaneous speech suggests a relatively sophisticated degree of anticipation on the part of the speaker. Frequent perceptual reports of short rushes of speech, short phrases, reduced stress, and interruptions or pauses that characterize the dysarthria associated with PD suggest that speakers with PD may not be able to anticipate the demands of the task as healthy controls would. Difficulties associated with planning and control of the respiratory system for purposes of spontaneous speech production may contribute significantly to the perceptual presentation of speakers with PD. Given an increased therapeutic emphasis on functional communication and that spontaneous speech is predominant as a mode of human communication; it is of interest to explore respiratory behavior during a spontaneous speaking task for speakers with PD. Relating this behavior to the linguistic structure of the speech produced may uncover strategies used by these speakers to maintain communicative effectiveness in the face of an impaired speech production system.

The purpose of the current study was to examine speech-breathing behaviors in speakers with PD and normal controls during a spontaneous speaking task. Kinematic and acoustic measures were used to characterize lung volume excursions during rest and speech production. The linguistic structure of the speech sample and its relation to the location of inspirations was also considered.

2. Methods

2.1. Participants

Thirteen native speakers of English served as participants in the present study. Seven had been diagnosed with idiopathic PD and six served as controls. Participants with PD passed screening for dementia (Folstein, Folstein, & McHugh, 1975) and hearing and reported no communication disorders prior to their neurologic diagnosis. Participants were recruited from local Parkinson support groups, and were experiencing mild-moderate symptoms related to PD. All PD speakers were taking anti-Parkinsonian medications; no participants had evidence of dose-related dyskinesias during the recording session. All individuals with PD were felt to be optimally medicated by their neurologist and were at their self-perceived peak of the medication cycle at the time of recording. All participants had moderate-high single word intelligibility scores ($M = 87.5$) based on Kent, Weismer, Kent, and Rosenbek (1989) Intelligibility test. Two participants had received speech therapy services more than 3 years prior to the experiment (mean number of sessions = 5). Individual participant characteristics can be found in Table 1.

Six participants served as controls in the present study. The control group (CG) consisted of three men and three women. All were nonsmokers, and had no history of speech, hearing, or respiratory disorders. All control speakers passed screenings for hearing and dementia. The CG was age-matched to provide an appropriate comparison for the PD speakers.

2.2. Speech task

The speech task used in the present study was an extemporaneous monologue prompted by an investigator. Speech was elicited by an investigator asking an open-ended question, e.g., ''What brought you to Tucson?'' The task was designed to allow maximum freedom of expression, without extremes of emotion or cognitive load. For the duration of the speech

Table 1
Speaker characteristics

Speaker code	Gender	Age (years)	Intelligibility (%)	Years post-diagnosis	Medications
CG01	M	71	99.2		
CG02	M	69	98.5		
CG03	M	61	97.9		
CG04	F	64	97.96		
CG05	F	72	96.3		
CG06	F	75	94.5		
PD02	M	63	92.0	1.5	b
PD03	F	75	86.5	2	a, b
PD05	F	81	76.9	3	a
PD06	M	75	85.4	8	a, c
PD08	F	70	84.5	6	a
PD09	M	70	90.9	5	a
PD10	M	70	90.1	11	a, b

Blanks within the table indicate not applicable. Notes: a: Sinemet/L-DOPA; b: Deprenyl/Selegeline; c: Peroglide/Permax.

Table 2
The number of breath groups analyzed, mean duration, syllable counts, and percentage of breath group filled with speech for individual speakers

Speaker	No. of breath groups	Mean duration (s)	Syllables per BG	Syllables (s)	Percentage of speech per BG
CG01	36	2.08 (0.86)	10.5 (4.8)	4.06	95
CG02	47	2.51 (0.97)	11.2 (3.5)	4.18	94
CG03	46	2.88 (0.76)	11.4 (6.2)	4.36	96
CG04	48	2.16 (0.63)	10.2 (2.1)	3.97	97
CG05	33	2.29 (0.83)	11.2 (5.1)	4.38	95
CG06	33	2.69 (0.74)	10.9 (3.1)	4.01	95
PD02	44	2.53 (1.40)	10.1 (6.4)	3.79	79
PD03	46	2.74 (1.93)	10.9 (8.0)	3.88	92
PD05	48	2.23 (1.60)	9.9 (5.8)	3.00	61
PD06	46	1.84 (1.17)	11.4 (4.8)	3.93	81
PD08	48	2.46 (1.68)	9.3 (7.1)	3.75	80
PD09	40	1.93 (1.30)	8.3 (6.7)	3.95	91
PD10	42	1.73 (1.93)	8.5 (5.0)	3.50	91

Standard deviations are listed in parentheses.

recordings, the investigator was seated in front of the speaker. The first 3 min of the speech sample for each speaker was analyzed in the current study. This portion of the sample was judged to contain the most fluent speech. The speech samples contained between 33 and 48 breath groups ($M = 42.8$). The number of breath groups analyzed for each speaker is listed in Table 2.

2.3. Acoustic data

The audio signal was sensed by a microphone placed 8 cm from the participant's mouth, and was amplified and recorded simultaneously with the kinematic data onto one track of the data tape. The audio signal recorded on the tape provided a reference for analysis of the kinematic data. A high quality acoustic signal was recorded using a head-mounted microphone (AKG C410) and a Panasonic SV3500 digital audiotape (DAT) for acoustic analysis. A mouth to microphone distance of 5 cm was used for the head-mount microphone. Using CSpeech (Milenkovic, 2001), speech samples were filtered at 9.8 kHz and digitized at 22 kHz.

Based on a waveform/spectrogram display in CSpeech (Milenkovic, 2001), cursors were manually placed at the beginning and end the speech signal within each breath group. Breath group boundaries were identified using the kinematic data and reference audio signal (see below). Measures of duration of speech per breath group, pause time and syllable counts were recorded.

2.4. Kinematic data

Recordings were made of surface motions of the chest wall using respiratory magnetometers based on the method of Hixon et al. (1973). Linearized magnetometers (GMG Scientific, 1980) were used to assess antero-posterior changes of the rib cage and

abdomen for kinematic respiratory measures. The magnetometers incorporate two generator-sensor coil pairs, one for the rib cage and one for the abdomen. One member of the rib cage coil pair was attached to the rib cage at the level of the sternum, the other member of the rib cage pair was attached on the posteriorly at the same level. One member of the abdomen coil pair was attached to the abdomen at a level just above the umbilicus, with the other member attached posteriorly at the same level. Participants were seated with their hands resting in their laps and their feet on the floor. Magnetometer signals were displayed on-line using an oscilloscope (Tektronix 5111A). The two data channels and the reference audio signal were recorded on a Vetter 820 data recorder.

Data collection began with a 2-min period of rest breathing. For this task, participants were asked to sit quietly while ''we check the equipment.'' Participants' vital capacity (VC) was then measured. To do this, the participants, wearing a nose clip and coupled to a spirometer (Collins 9-L), inspired fully from tidal end expiratory level (EEL) and then expired fully. For volume calibration, participants inspired and expired from EEL approximately 1 L through a tube coupled to a spirometer while wearing a nose clip. The volume calibration was done over a series of trials with participants either inspiring or expiring on a single trial. Participants then performed several isovolume maneuvers at EEL. Participants were instructed to hold their breath at EEL and displace their lung volume back and forth between the abdomen and rib cage. Several isovolume maneuvers were completed at the beginning and end of the recording session. Details regarding calibration maneuvers can be found in Hixon et al. (1973) and Hoit and Hixon (1987). The participants then began the speech task protocol. The task utilized in this study was part of a larger speech protocol. The order of tasks within the protocol was randomized across speakers.

Customized software (LabView Software; National Instruments; Austin, TX) was used to digitize data to generate motion–motion and time–motion displays. The motion–motion display was constructed by displaying the magnetometer signals so that the rib cage was on the ordinate and the abdominal signal was on the abscissa. Using the calibration maneuvers, a template was created for each participant against which to measure the kinematic data.

To obtain measures of lung volumes at the initiation and termination of each breath group, cursors were placed manually and values recorded. Lung volume measures were recorded peak of inspiration (LVI), the initiation of speech (LVI-S) and at the termination of the both speech (LVT-S) and the onset of inspiration for the following breath (LVT). The location of the breath group was also marked on a transcript of the speech sample based on the reference audio signal display.

2.5. Linguistic data

A linguistic analysis was completed to examine the location of breath group boundaries. The analysis was based on the technique reported by Winkworth et al. (1994). Transcripts for each speaker were typed verbatim without punctuation. Two judges marked clause boundaries on the transcripts. The criterion for marking a clause boundary was that it contained a finite verb. Incomplete grammatical utterances, such as restarts, were not counted as independent clauses, but were included in following complete clause.

Following the grammatical markings, the locations of inspirations were marked on the transcripts based on data obtained from kinematic recordings. Breaths, not associated with speech, such as periods of rest or laughter were omitted from analysis. The location of inspirations was then compared to grammatical markings. Inspirations preceding the clauses marked on the transcripts were counted as occurring at structural boundaries. Inspirations occurring at other locations, such as those between lists of items or single words were categorizes as other. The number of inspirations occurring at structural boundaries and those classified as other were expressed as a percentage of the total number of inspirations in the speech sample. The two judges classified each breath group as occurring at a structural boundary or other location independently. Results were then compared. If the judges did not agree on a particular clause, they discussed differences and came to a mutual decision. Disagreement occurred on 4% of the total breath groups analyzed and all disputes were easily resolved.

2.6. Statistical analysis

Means and standard deviations were calculated for all participants for the following variables: breath group duration, syllable count, LVI, LVI-S, LVT, LVT-S. Two-sample *t*-tests were used to look at group differences for each of these variables. An alpha level of 0.05 was used to ascertain significance.

2.7. Reliability

For each speaker, 15 breath groups were randomly selected and were remeasured to estimate inter- and intra-judge reliability. The overall correlation coefficient between the first and second set of measurements was 0.93; the correlation coefficient between the two sets of measurements for each individual speaker was greater than 0.90. The average inter-judge reliability measurement errors for the temporal and lung volume measures were 15 ms and 2.6%, respectively. Average intra-judge measurement errors were 11 ms and 2.1%.

3. Results

Statistically significant differences between speakers with PD and the control group were found for the following measures: breath group duration, syllables per breath, duration of speech per breath group, as well as lung volume initiation and termination points. The amount of variability in these measures was also greater for the speakers with PD compared to the control group.

3.1. Acoustic

The total number of breath groups analyzed for each speaker, mean duration of each breath group, syllables per breath group, syllables per second, and percentage of time spent speaking per breath group are shown in Table 2. The mean duration of speech produced

within a breath group for the CG speakers was 2.37 s (S.D. = 1.31). For the PD group the mean was 2.22 s (S.D. = 1.74). A statistically significant difference in mean duration for the speaker groups was found ($t(9) = -2.25$, $p = 0.049$). Syllable count per breath group ranged from 1 to 31 for the CG ($M = 13.45$) and 1 to 24 for the PD group ($M = 8.7$). Large within speaker variability was noted for both the CG and PD speakers across the 3 min speech sample, as can be seen by the standard deviations for individual speakers listed in parenthesis in Table 2 (column 4). This was not unexpected given the unstructured speech task. Group differences in syllable count were not found to be statistically significant ($t(8) = -0.93$, $p = 0.38$). To further explore differences in production between the two speaker groups, a ratio of the number of syllables per second was computed. The ratio was lower for the speakers with PD compared to the CG, speakers with PD produced fewer syllables per second (CG: $M = 4.16$ (1.44), PD: $M = 4.076$ (3.75)). Group differences were statistically significant ($t(6) = 0.98$, $p = 0.02$). The final column of Table 2 showing the percentage of time spent speaking per breath group will be discussed below.

3.2. Kinematic

Lung volumes were measured at the end of the inspiratory portion of each breath cycle (LVI) as well as at the initiation of speech (LVI-S) on the expiratory phase. Measures were also taken at the termination of speech (LVT-S) on the expiratory phase and onset of the following inspiratory cycle (LVT). For the CG, LVI was highly correlated with breath group duration. Correlations ranged from 0.84 to 0.95. Longer breath groups had higher starting lung volumes and shorter breath groups had lower starting lung volumes. This strong correlation was not observed for the PD speakers. The range of correlations for these speakers was 0.17 to 0.73. The possibility of fatigue in the PD group as the session progressed was examined; however, there did not appear to be any systematic change in starting lung volume over time. The range of LVI-S volumes for each speaker is shown in Fig. 1 as a box plot. The CG initiated speech above EEL 94% of the time, with a mean value of 19.97% above EEL. The line extending below EEL for several of the CG speakers correspond to single word productions on the transcripts. For the PD speaker group 69% of speech events were initiated above EEL, mean initiation values for the PD group were
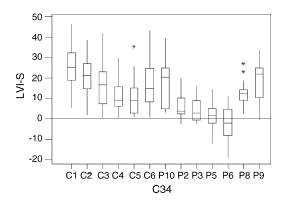

Fig. 1. Box plot range of LVI-S for individual speakers.

6.79% above EEL. Three of the speakers with PD had lung volume initiation (LVI-S) values that were comparable to the CG (PD08, PD09, and PD10), the mean values for this subgroup of speakers with PD was16.26% over EEL. The remaining four PD speakers, however, tended to initiate speech at lung volumes near or below EEL (PD02, PD03, PD05, PD06). Mean lung volumes at the onset of speech for this group were 3.64% above EEL. Statistically significant differences for both LVI and LVI-S measures were found between the speaker groups (LVI: $t(10) = 1.62$, $p = 0.014$; LVI-S: $t(5) = 4.45$, $p = 0.006$).

Lung volume termination measured at the offset of speech was at or slightly below EEL for the majority of utterances produced by the CG (81%). The mean for the group was 2.16% below EEL. There was some individual speaker variability noted in LVT. Two speakers (CG4, CG5) continued speaking below EEL more frequently than other speakers in the CG. Utterances which continued past EEL all ended at grammatical boundaries. LVT and LVT-S measures were nearly identical for the CG speakers, meaning that speakers began inspiring for the next breath as soon as they finished speaking. For the PD speakers, LVT values during periods of rest breathing were similar to the CG and ended at or above EEL (group mean = 3.10% above EEL). During the speaking task, however, lung volume termination for the PD speakers was measured below EEL (group mean = 4.09% below EEL). During the speaking task, only 17% of the utterances produced by the PD speakers were terminated at or only slightly below EEL (values comparable to the CG) while the remaining 83% of the utterances ended significantly below EEL ($M = -11.13\%$). A box plot showing individual LVT-S values relative to their EEL can be seen in Fig. 2. Differences between the groups were statistically significant ($t(10) = 4.10$, $p = 0.002$).

Total breath group duration (time between LVI and LVT) was compared to speaking time (time between LVI-S and LVT-S) to examine the amount of time during the expiratory phase that each speaker spent producing speech. These findings are presented in Table 2 (column 6) as a mean percentage for each speaker. Results show that for the CG speakers almost the entire duration of the breath group was spent speaking (range 94–97%). Any silence during the breath group was located at the beginning of the breath group (a difference in LVI versus LVI-S) and lasted less than 0.15 s. For the PD speakers, the percentage of time spent speaking ranged from 61 to 91%. For three PD speakers (PD02, PD09, and PD10), a silent interval occurred at the beginning of the breath group and was
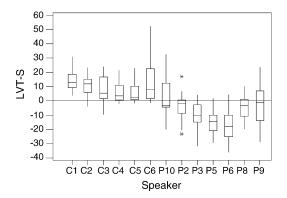

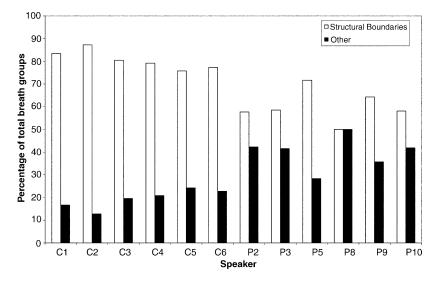Fig. 2. Box plot range of LVT-S for individual speakers.

Fig. 3. Results of linguistic analysis showing percentage of breath groups that ended at structural boundaries vs. other locations.

comparable in duration to the CG ($M = 0.11$ s). For the remaining 4 PD speakers differences between LVI and LVI-S were not observed. For these speakers, an audible exhalation was frequently heard following the termination of speech, before they initiated the next inhalatory cycle. This was calculated as the difference between LVT-S and LVT. The duration of this exhalation following speech ranged from 0.43 to 1.2 s.

3.3. Linguistic

Breath groups were labeled as occurring at a structural boundary or other locations. For each speaker, the percent of total breath groups that occurred at structural or other locations can be seen in Fig. 3. For the CG speakers between 75 and 87%, their breath group boundaries occurred at structural boundaries. The breath groups classified as other were typically restarts or repetitions. The number of breath groups for the PD speakers, which ended at structural boundaries, ranged from 50 to 71%. Of the boundaries that occurred at other locations, the majority occurred within a phrase (68%) followed by single words (20%) and repetitions (12%).

4. Discussion

The current study examined speech-breathing patterns in people with PD compared to an age-matched CG during an extemporaneous speaking task. Differences were found in the duration of breath groups, syllables per second, initiation and termination lung volumes associated with speech, as well as the linguistic patterns of the speech produced by the PD speakers compared to the CG.

4.1. Acoustic characteristics

Significant differences were found for speakers with PD compared to those in the CG on overall breath group duration as well as syllables per second. The PD speakers produced shorter breath groups ($M = 2.18$ s) than the CGs ($M = 2.42$ s). Durations for both groups are comparable to those reported by Bunton, Kent, Kent, and Rosenbek (2000) and Schlenck, Bettrich, and Willmes (1993) for speakers with dysarthria and age matched controls. Similar differences between speakers with PD and a CG on measures of breath group duration were reported by Solomon (1991, 1993). Larger intra-participant variability across breath groups was observed, the PD speaker group compared to the CG in the current study; with standard deviations for the PD group double that of the CG (Table 2). Similar variability during a monologue task was not found by Solomon (1991). Individual speaking style differences could account for differences seen in variability across the speaking task.

A ratio of syllable count and breath group duration showed statistically significant group differences; with speakers in the PD group produced fewer syllables per second than the CG. This finding is similar to data reported by Solomon and Hixon (1993) where PD speakers produced fewer syllables and spoke for less time per breath than the healthy controls. Increased airflow due to faulty laryngeal or velopharyngeal valving could be one reason syllable count differences were found across speaker group. Solomon and Hixon (1993), however, reported no group differences in lung volume excursion, an indirect measure of airflow. They did report a task effect related to airflow, with average airflow rates higher during a monologue task compared to paragraph reading. Hodge and Rochet (1989) have reported similar differences between speech tasks.

Based on examination of the transcripts for the PD speakers it appears that differences in syllable rate may be related to demands of linguistic formulation associated with the extemporaneous speaking task. Differences in the patterns of breath groups produced by the speakers were found for the PD group compared to the CG. The CG typically produced one longer breath group followed by a series of very short breath groups, which typically contained filler words. These speakers seemed to chunk the information they wished to express and then take a break, perhaps in an effort to formulate what to say about next. This pattern is similar to that reported by Winkworth et al. (1995) for healthy female speakers. The PD speakers produced several longer breath groups in succession and then followed this with a series of shorter breath groups. Unlike what was seen for the CG, the shorter breath groups produced by the PD speakers were typically content words produced in isolation. If we consider that speakers with PD frequently continued speaking below EEL, a pattern of several longer breath groups followed by shorter ones may relate to a physical need to rest or catch their breath. This is in contrast to the CG who appeared to use shorter breath groups for language formulation. When directly asked, PD speaker denied feelings of dyspnea. This question was asked at the completion of the entire experimental protocol, so isolated incidents of speaking related dyspnea may not have been accurately recalled.

The average LVI found for speakers in the CG, at a mean of 19.97% above EEL, was slightly higher than previous studies of women (13.3 and 10.49% VC above REL; Hodge & Rochet, 1989; Hoit, Hixon, Altman, & Morgan, 1989, respectively). The LVT volumes for the CG in the present study were 2.16% below EEL. This value is again comparable to values reported by Hodge and Rochet (1989) and Hoit et al. (1989), who reported average

LVT of 2.9 and 3.66% below REL, respectively. A strong link between breath group length and initiation or inspired lung volume was also found for the CG. In other words, longer breath groups were associated with higher LVI and shorter breath groups were associated with lower LVI. This relationship has also been reported by Winkworth et al. (1995). The longer breath groups initiated at higher lung volumes, which might represent a speakers need to convey a whole message without interruption, appear logical. Speakers in the CG most likely planned what they were going to say in advance and then took a larger breath to match the length of the intended message. This type of planning was not seen in the PD speakers, as lung volume initiation points were found to be highly variable for the speakers with PD, and were not correlated to the length of the breath group. Huber et al. (2001) have also reported large variability in LVI for speakers with PD, although their differences were associated with a sentence repetition task. For the PD speakers, 69% of speech events were initiated above EEL (compared to 94% for the CG). The mean LVI for the PD group was 4.79% above EEL. This value was found to be statistically different than the CG. Examination of individual data for the PD speakers, however, showed that four of the speakers (PD06, PD08, PD09, and PD10) had LVI values that were similar to the CG (mean = 16.26%) and data previously reported in the literature (Hodge & Rochet, 1989; Hoit et al., 1989). The three remaining PD speakers had mean LVI of only 4.64% above EEL. For these three speakers, in particular, no link between breath group duration and LVI was observed ($r < 0.18$). Terminating lung volumes for all 7 of the PD speakers fell further below EEL than the CG ($M = 2.16\%$ and $M = 3.10\%$ below EEL for CG and PD groups, respectively). Reporting a mean for the PD group is misleading as more than half the utterances produced continued well below the mean value (83%). The mode LVT-S value was 11.13% below EEL. Expiring this far below REL implies that PD speakers activated progressively more abdominal muscles to sustain the subglottic pressure as lung volume declined (Draper, Ladefoged, & Witteridge, 1959; Watson & Hixon, 1985). It appeared that the PD speakers were exerting considerably more ''effort'' than the CG speakers during the speech task.

Comparisons of the initiation and termination lung volumes for speech (LVI-S and LVT-S) versus the total breath group initiation and termination volumes (LVI and LVT) showed that for the CG the values were almost identical. In other words, CG speakers spent nearly the entire duration of a breath group producing speech (range 94–97%). The PD speakers were more variable in the amount of time per breath group that was spent producing speech. The range was 61–92% across speakers; with large intra-participant variability noted for all 7 speakers. Differences in amount of time spent producing speech and the total breath group duration were noted at the end of breath groups. The investigator frequently heard audible exhalations following the termination of speech. Even when an exhalation was not audible, it was detected on the LabView display as a difference in LVT-S and LVT. Solomon and Hixon (1993) reported that LVT-S and LVT values were nearly identical for both the CG and PD groups. The difference between LVT-S and LVT found for the PD speaker group and an accompanying audible exhalation after speech was terminated has not been reported previously in studies on speech breathing in PD. The strategy of exhaling speaking, seen in the present study, is costly in terms of effort, and therefore is difficult to explain. Speakers with PD, however, presumably need to contend with a rigid rib cage wall, so perhaps increasing abdominal activity below REL allows them two advantages. First, expiratory displacement of the abdomen forces the diaphragm in an optimal position for

inhalation. Second, the positive abdominal muscular pressures at low lung volumes may offer an additional efficiency gain in the form of mechanical tuning of the rib cage. Abdominal activity results in distortion of the chest wall toward a larger rib cage volume thus placing the expiratory muscles at greater and more optimal lengths for producing speech on the next breath group. This explanation has been presented for opera singing, where the demands of the musical piece are such that quick and forceful pressure changes are needed (Watson & Hixon, 1985). For the PD speakers, however, reasons for the behavior may not be related to the speaking task per se, but rather a way to deal with a disordered system. Solomon and Hixon (1993) reported a smaller contribution of the rib cage to lung volume change during speech which they related to increased rigidity of the rib cage as a result of PD. The abdomen, with a relatively greater compliance, appeared to contribute more than it would be expected to for lung volume changes during speech breathing. Therefore, in speakers with PD, greater abdominal effort may be related to their need to maintain and control an adequate air stream for speech production in the presence of a rigid rib cage.

4.2. Linguistic structure of the breath group

For the CG, the majority of inspirations preceded structural boundaries (M = 81%). The strategy of timing inspirations with these structures appears to be of benefit to the listener as disruptions to the flow of information are minimized or confined to appropriate breaks. These values are slightly higher than data reported by Winkworth et al. (1995) and Henderson et al. (1965). They reported, respectively, 63 and 69% of inspirations occurred at structural boundaries. The PD speakers produced fewer breath groups ending at structural boundaries compared to the CG in the current study (PD mean = 62%). Large inter-participant variability in the location of inspirations during spontaneous speech may be related to the individuals' characteristics speaking styles. Significant individual differences in pause distribution in spontaneous speech have been reported by Goldman Eisler (1968). Several researchers have suggested that breaths are taken at places that naturally segment the discourse, such as structural boundaries or points of hesitation. This notion of convenience, however, does not explain the pattern of inhalations, see for PD speakers in the current study. For the PD speakers, several breath groups contained only single content words. On possible explanation is that speakers were attempting to maintain lung volume adjustments that were not too costly in terms of energy and effort while keeping the listener engaged in the conversation. Thus, they produced single content words on these short breaths so as not to signal a turn exchange by pausing. This strategy may be effective in a true communicative interaction.

The speech task used in the present study requires special comment. This type of extemporaneous speech task might not have been representative of true communicatively functional speech, where there would be an interaction between communication partners. It is possible that lung volumes associated with spontaneous speech occurring in everyday situations would be different than those found in the present study. It is recommended that future work examining speech breathing in PD should include a variety of spontaneous speaking tasks, such as an interactive conversational exchange and event or story telling, in attempt to capture speech production that is comparable to "real-world" situations.

5. Summary

Results of the present study were somewhat unexpected given previous publications on speech breathing in PD (e.g., Solomon & Hixon, 1993). Speakers with PD began speaking at lower lung volumes and had an increased variability in starting lung volumes across the speech sample than the CG. Speakers with PD also continued speaking below EEL. Speaking below EEL implies greater muscular effort and is therefore, difficult to explain. However, if we consider effort required to produce speech as a combination of that exerted for inspiration plus expiration, it may be that expending more effort during one part of the cycle will minimize effort required for another and will have the benefit of allowing a speaker to maintain a high level of intelligibility. Therefore, one possible way to deal with the increased rigidity of the rib cage seen in PD was for speakers to increase abdominal effort to aid both the rib cage and diaphragm in their primary functions. From the linguistic analysis, we see a mismatch between location of inspirations and the grammatical structure of the utterances produced for speakers with PD. This may reflect a strategy to maintain their conversational turn while responding to a physiologic need to inspire. Findings in this study should be considered preliminary in nature, as further studies of speech breathing behavior in a variety of spontaneous speech tasks are needed to replicate current findings and strengthen an explanation of why speakers with PD continue past EEL.

Acknowledgements

Continuing education questions

1. General respiratory dysfunction in PD:
    a. has never been reported
    b. does not change as the disease progresses
    c. ultimately leads to the need to be put on a ventilator
    d. affects maneuvers of speech, effort, and maximal range
    e. is not a problem
2. The following air stream measures can be used to assess respiratory function:
    a. pressure
    b. flow
    c. volume
    d. all of the above
    e. none of the above
3. Based on the findings of this study which of the below are true?

   a. All speakers continue speech production below EEL.
   b. Speakers with PD produced fewer syllables per breath group than controls.
   c. The location of inspirations always occurs at a linguistic boundary.
   d. Some speakers with PD begin speaking below EEL.
   e. Speech is never produced below EEL.
4. The results of the current study suggest that:
   a. Treatment of dysarthria should emphasize strategies to coordinate speech breathing with the linguistic structure of the intended message.
   b. Patterns of speech breathing during interactive communicative situations need to be studied.
   c. Speech breathing should not be addressed in treatment.
   d. Both (a) and (b) above.
   e. None of the above.
5. Patterns of lung volume use can be affected by:
   a. speaking task
   b. speaker age
   c. disease process
   d. problems with upper-airway valving
   e. all of the above

References

Anthony, J. K., & Farquharson, I. (1975). Clinical research in speech pathology. *The Journal of Laryngology and Otology, 89*, 1169–1184.

Brown, L. K. (1994). Respiratory dysfunction in Parkinson's disease. *Clinics in Chest Medicine, 15*, 715–727.

Bunton, K., Kent, R., Kent, J., & Rosenbek, J. (2000). Perceptuo-acoustic assessment of prosodic impairment in dysarthria. *Clinical Linguistics and Phonetics, 14*, 13–24.

Conrad, B., Thalacker, S., & Shonle, P. (1983). Speech respiration as an indicator of integrative contextual processing. *Folia Phoniatrica, 35*, 220–225.

De la Torre, R., Mier, M., & Boshes, B. (1960). Studies in parkinsonism: IX. Evaluation of respiratory function—Preliminary observations. *Quarterly Bulletin, Northwestern University Medical School, 34*, 580–584.

Draper, M., Ladefoged, P., & Whitteridge, D. (1959). Respiratory muscles in speech. *Journal of Speech and Hearing Research, 2*, 16–27.

Ewanowski, S. J. (1964). Selected motor-speech behavior of patients with parkinsonism. *Dissertation Abstracts International, 24/11*, 4680 (University Microfilms No. AAC6407081).

Folstein, M., Folstein, S., & McHugh, P. (1975). "Mini-Mental State"—A practical method for grading the cognitive status of patients for the clinician. *Journal of Psychiatric Research, 12*, 189–198.

Goldman Eisler, F. (1968). *Psycholinguistics: Experiments in spontaneous speech*. London: Academic Press.

Henderson, A., Goldman Eisler, F., & Skarbek, A. (1965). Temporal patterns of cognitive activity and breath control in speech. *Language and Speech, 8*, 236–242.

Hixon, T., Goldman, M., & Mead, J. (1973). Kinematics of the chest wall during speech production: Volume displacements of the rib cage, abdomen, and lung. *Journal of Speech and Hearing Research, 16*, 78–115.

Hodge, M., & Rochet, A. (1989). Characteristics of speech breathing in young women. *Journal of Speech and Hearing Research, 32*, 466–480.

Hoit, J. D. (1994). A critical analysis of speech breathing data from the University of Queensland. *Journal of Speech and Hearing Research, 37*, 572–582.

Hoit, J. D., & Hixon, T. (1986). Body type and speech breathing. *Journal of Speech and Hearing Research, 29*, 313–324.

Hoit, J. D., & Hixon, T. (1987). Age and speech breathing. *Journal of Speech and Hearing Research, 30*, 351–366.

Hoit, J. D., Hixon, T. J., Altman, M. E., & Morgan, W. (1989). Speech breathing in women. *Journal of Speech and Hearing Research, 32*, 353–365.

Hovestadt, A., Bogaard, J. M., Meerwaldt, J. D., van der Meche, F. G., & Stigt, J. (1989). Pulmonary function in Parkinson's disease. *Journal of Neurology, Neurosurgery, and Psychiatry, 52*(3), 329–333.

Huber, J. E., Stathopolous, E. T., Ramig, L. O., & Lancaster, S. L. (2000). Respiratory function and variability in individuals with Parkinson disease: Pre- and Post-Lee Silverman Voice Therapy. *Journal of Medical Speech-Language Pathology, 11*(4), 185–201.

Hunker, C., Bless, D., & Weismer, G. (1981). *Respiratory inductive plesthysmography: A clinical technique for assessing respiratory function for speech*. Paper presented at the annual convention of the American Speech-Language-Hearing Association, Los Angeles. Available from Ambulatory Monitoring, Inc. (Manufactureres of Respitrace systems), Ardsley, NY.

Izquierdo-Alonso, J. L., Jiminez-Jimenez, F. J., Cabrera-Valdivia, F., & Mansilla-Lesmes, M. (1994). Airway dysfunction in patients with Parkinson's disease. *Lung, 172*, 47–55.

Kent, R. D., & Kent, J. F. (2000). Task-based profiles of the dysarthrias. *Folia Phoniatria et Logopaedica, 52*, 48–53.

Kent, R. D., Kent, J. F., & Rosenbek, J. (1987). Maximum performance tests of speech production. *Journal of Speech and Hearing Disorders, 52*, 367–387.

Kent, R. D., Weismer, G., Kent, J., & Rosenbek, J. (1989). Towards phonetic intelligibility testing in dysarthria. *Journal of Speech and Hearing Disorders, 54*, 482–489.

Lethlean, J., Chenery, H., & Murdoch, B. (1990). Disturbed respiratory and prosodic function in Parkinson's disease: A perceptual and instrumental analysis. *Australian Journal of Human Communication Disorders, 18*, 83–97.

Liker, E. S., & Woolf, C. R. (1968). Pulmonary function in Parkinson's syndrome: The effect of thalamotomy. *Canadian Medical Association Journal, 99*, 752–757.

MacIntosh, D. J. (1977). Respiratory dysfunction in Parkinson's disease. *Primary Care (Symposium on Parkinson's disease), 4*(3), 441–445.

Marquardt, T. P. (1973). Characteristics of speech production in Parkinson's disease: Electromyographic, structural movement, and aerodynamic measurements. *Dissertation Abstracts International, 34/08B*, 4113 (University Microfilms No. AAC7402217).

Milenkovic, P. (2001). *Cspeech (Computer program)*. University of Wisconsin-Madison.

Mueller, P. B. (1971). Parkinson's disease: Motor-speech behavior in a selected group of patients. *Folia Phoniatrica, 23*, 333–346.

Murdoch, B. E., Chenery, H. J., Bowler, S., & Ingram, J. C. (1989). Respiratory function in Parkinson's participants exhibiting a perceptible speech deficit: A kinematic and spirometric analysis. *Journal of Speech and Hearing Disorders, 54*, 621–626.

Murry, T. (1983). The production of stress in three types of dysarthric speech. In W. B. Berry (Ed.), *Clinical dysarthria* (pp. 69–83). San Diego: College-Hill Press.

Nakano, K. N., Bass, H., & Tyler, J. (1972). Levodopa in Parkinson's disease: Effect on pulmonary function. *Archives of International Medicine, 130*, 346–348.

Nakano, K. N., Zubick, J., & Tyler, J. R. (1973). Speech defects of parkinsonian patients. *Neurology, 23*, 865–870.

Netsell, R. (1983). Speech motor control: Theoretical issues with clinical impact. In W. R. Berry (Ed.), *Clinical dysarthria*. San Diego: College-Hill Press.

Netsell, R., Daniel, B., & Celesia, G. G. (1975). Acceleration and weakness in Parkinsonian dysarthria. *Journal of Speech and Hearing Disorders, 40*, 170–178.

Neu, J. C., Connolly, J. J., Schwertley, F. W., Ladwig, H. A., & Brody, A. W. (1967). Obstructive respiratory dysfunction in parkinsonian patients. *American Review of Respiratory Disease, 95*, 33–47.

Paulson, G. D., & Tafrate, R. H. (1970). Some "minor" aspects of parkinsonism, especially pulmonary function. *Neurology, 20*(Part 2), 14–17.

Plum, F. (1974). Cerebral control of breathing. In B. Wyke (Ed.), *Ventilatory and phonatory control systems* (pp. 208–223). London: Oxford University Press.

Schlenck, K.-J., Bettrich, R., & Willmes, Z. (1993). Aspects of disturbed prosody in dysarthria. *Clinical Linguistics and Phonetics, 7*, 119–128.

Smith, L. L. (1964). *Restricted motility of the speech articulators, lung function, amount of air expired per unit of speech and speech defectiveness in adults with Parkinson's disease.* Unpublished master's thesis. State University of Iowa, Iowa City.

Smitheran, J. R., & Hixon, T. J. (1981). A clinical method for estimating laryngeal airway resistance during vowel production. *Journal of Speech and Hearing Disorders, 46,* 138–146.

Solomon, N. P. (1991). *Speech breathing in Parkinson disease.* Unpublished Doctoral Dissertation, University of Arizona, Tucson, AZ.

Solomon, N. P., & Hixon, T. (1993). Speech breathing in Parkinson's disease. *Journal of Speech and Hearing Research, 36,* 294–310.

Von Euler, C. (1982). Some aspects of speech breathing physiology. In S. Grillner, B. Lindblom, J. Lubker, & A. Persson (Eds.), *Speech motor control* (pp. 95–103). New York: Pergamon Press.

Warren, D., & Wood, M. (1969). Respiratory volumes in normal speech: A possible reason for intraoral pressure differences among voiced and voiceless consonants. *Journal of the Acoustical Society of America, 45,* 466–469.

Watson, P., & Hixon, T. (1985). Respiratory kinematics in classical (opera) singers. *Journal of Speech and Hearing Research, 28,* 104–122.

Wilder, C. (1983). Chest wall preparation for phonation in female speakers. In D. M. Bless, & J. H. Abbs (Eds.), *Vocal fold physiology—Contemporary research and clinical issues* (pp. 109–123). San Diego: College-Hill Press.

Winkworth, A., Davis, P., Ellis, E., & Adams, R. (1994). Variability and consistency in speech breathing during reading: Lung volumes, speech intensity, and linguistic factors. *Journal of Speech and Hearing Research, 37,* 535–556.

Winkworth, A., Davis, P., Ellis, E., & Adams, R. (1995). Breathing patterns during spontaneous speech. *Journal of Speech and Hearing Research, 38,* 124–144.