Volume Regulation in Parkinsonian Speech

Aileen K Ho[1]
John L Bradshaw[1]
Robert Iansek[3]
Robin J Alfredson[2]

[1] Neuropsychology Research Unit, Department of Psychology, Monash University, Clayton 3168, Australia.
[2] Department of Mechanical Engineering, Monash University, Clayton 3168, Victoria, Australia.
[3] Geriatric Research Unit, Kingston Centre, Warrigal Rd, Cheltenham 3192, Victoria, Australia.

ABSTRACT

This study investigated the ability to regulate speech volume in a group of six-volume impaired idiopathic Parkinson's disease (PD) patients and their age and sex-matched controls. Participants were asked to read under three conditions; as softly as possible, as loudly as possible, and at normal volume (no volume instruction). The stimuli consisted of a target sentence, easily read in one breath, embedded in a short paragraph of text. Mean volume and volume over time (intensity slope) for the target sentence were obtained. It was found that for all three conditions, patients' speech volume was less than controls' by a constant. Patients also showed a significantly greater reduction of volume (negative intensity slope) towards the end of the sentence, especially for the loud condition. The findings indicate that patients with Parkinsonian hypophonic dysarthria have significant difficulty maintaining speech volume in addition to the inadequate generation of overall speech volume.

1. INTRODUCTION

One of the clinical manifestations of Parkinsonian dysarthria is quiet speech[2,3,12]. Acoustic studies in Parkinsonian speech have typically employed mean intensity as an index of speech volume e.g. Ackerman et al[1] and Illes et al[9]. This measure of average, however, obscures the pattern of intensity change over the duration of the articulatory sequence.

Time-sensitive measures of movement amplitude in PD patients have shown a progressive reduction in extent over a sequence of movements such as progressively smaller step size in walking i.e. gait hypokinesia[10], and stroke length in handwriting i.e. micrographia[13]. Speech motor control, like handwriting and gait, is a highly automatic complex task heavily reliant on fronto-striatal mechanisms. Hence the disruption of the fronto-striatal motor circuit in PD may also underlie the progressive reduction of volume in speech sequences. Given the similarities between speech and other automatic limb movements, and the common mechanism, it would therefore be fruitful to adopt a time-sensitive measure in examining speech volume and control. The present study utilises such a measure to examine temporal patterns in volume control not indexed by the traditional average.

2. METHOD

2.1 Participants

Six idiopathic PD patients (mean age = 75;8, standard deviation = 6;8) with hypophonic dysarthria and six controls (mean age = 75;7, standard deviation = 2;7) with no neurological complications participated in this study. There were five males and one female in each group. Patients were stabilised on anti-Parkinson medication and remained on their usual medication regime when participating in the experiment, and were tested between one and three hours of receiving medication.

2.2 Apparatus

Reading stimuli consisted of Fairbank's Rainbow passage[6] in large print. The target sentence for analysis was the second sentence "The rainbow is a division of white light into many beautiful colours". This sentence was chosen because it was of moderate length and could easily be read in one breath. A Marantz tape recorder (PMD222) and microphone (David Clark) were used to record reading in a sound attenuated room. The target sentence was analysed using the KAY Elemetrics CSL 5.05 system.

2.3 Procedure

After participants had a practice trial to familiarise themselves with the Rainbow passage, they were asked to read according to three instruction conditions. The first condition was the Normal condition where participants where simply told to read the passage; no instruction on volume was given and participants read at their automatic self-selected default volume. The second condition was the Soft condition where they were told to read as softly as possible (but without whispering), as if there was a baby sleeping in the same room. In the third condition i.e. the Loud condition, participants were instructed to read as loudly as possible (but without shouting), as if they were at a very noise place such as a sporting event. The mouth-to-microphone distance was kept at a constant of 20cm.

3. RESULTS

The mean volume and linear regression slope of the energy waveform of the target sentence were analysed for group differences.

Mean volume data are depicted in Figure 1. A two-way repeated measures ANOVA with factors of Group (patients, controls) and Instruction (soft, normal, loud) showed significant main effects of Group ($F(1,10) = 14.33$, $p < .01$) and Instruction ($F(2,20) = 92.21$, $p < .001$), and the absence of a Group x Instruction interaction ($F(2,20) = .58$, $p = .569$). Therefore, PD patients' mean reading volume (mean = 56.87dB) was significantly lower than controls (mean = 61.69dB), and patients were always softer by a constant amount in all three conditions. Apart from the blanket volume reduction in patients, both groups were able to modulate speech volume to the same degree and showed similar increases in volume from the soft condition, to the normal and loud conditions.

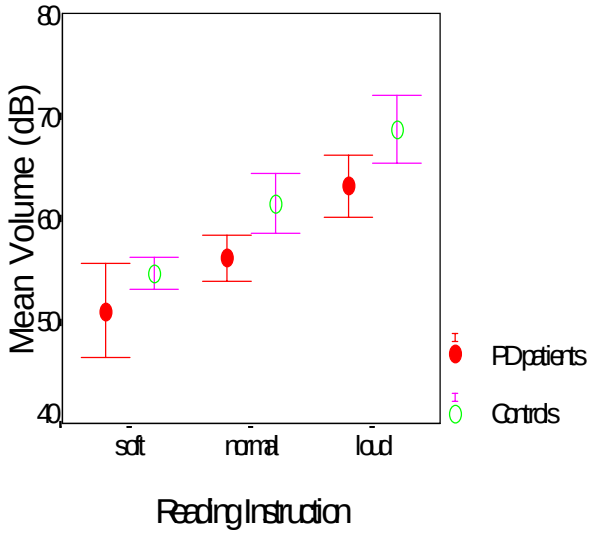

Figure 1: Mean volume of PD patients and controls according to reading instruction (soft, normal, loud).

Figure 2 shows an example of a patient and a control participant reading the target sentence under the normal condition. The energy contour of the target sentence has been subjected to a linear regression fit and a Lowess fit in order to summarise speech volume over the duration of the acoustic event. The linear regression lines show a negative slope in both cases (1a&b) illustrating the gradual decrease of speech volume over the time taken to read the target sentence in one breath. The patient (1b), however, shows a far greater diminishing of speech volume along the sequence.

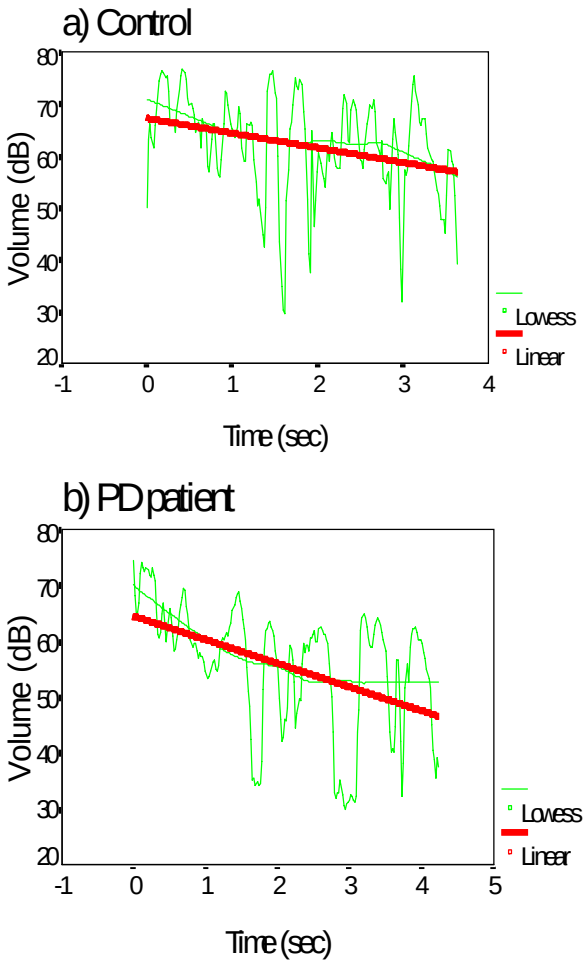

Figure 2: Linear regression slope (bold line) and Lowess fit (thin line) on the speech energy contours of the target sentence for a) one control participant and b) one PD patient.

Figure 3 shows the mean intensity slopes of patients and controls on a negative scale for the three reading conditions. A two-way repeated measures ANOVA with factors of Group (patients, controls) and Instruction (soft, normal, loud) showed significant main effects of Group ($F(1,10) = 31.01$, $p < .001$) and Instruction ($F(2,20) = 5.94$, $p < .05$), and a Group x Instruction interaction trend ($F(2,20) = 3.14$, $p = .065$). Therefore, controls demonstrated a slight negative intensity slope (mean = -2.25), with intensity naturally diminishing toward the end of the target sentence and breath envelope. PD patients showed negative intensity slopes of even greater magnitude (mean = -4.46) as intensity diminished dramatically over the sentence, despite reading time not being any longer than controls.

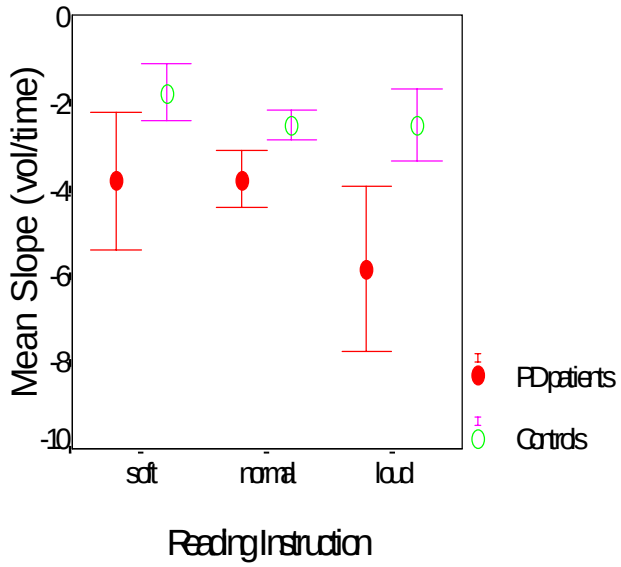

Figure 3: Mean linear regression slope on target sentence volume of PD patients and controls, according to instruction condition (soft, normal, loud).

The Group x Instruction interaction trend for intensity slope suggested that patients and controls may differ with regard to the extent of the intensity slope across the three instruction conditions. In order to locate the seat of this interaction trend, the soft and normal conditions were subjected to a two-way repeated measures ANOVA, and the normal and loud conditions to a separate two-way ANOVA. The latter ANOVA yielded a significant Group x Instruction interaction effect ($F(1,10) = 7.45$, $p < .05$) whilst the former did not ($F(1,10) = .92$, $p = .360$), showing that patients' and controls' negative intensity slopes differed in the normal condition compared to the loud condition. Paired sample t-tests on patients and on controls showed that there was a significant difference in the normal and loud intensity slopes for patients ($t(5) = 2.89$, $p < .05$) but not for controls ($t(5) = .02$, $p = .985$). Thus PD patients showed increased negative intensity slopes for the loud condition, compared to the soft and normal conditions, whereas controls did not show this. This indicates that whilst patients were able to increase mean volume for the loud condition, they were less able to maintain intensity when reading loudly rather than normally or softly.

4. DISCUSSION

This study investigated Parkinsonian speech motor control in PD by examining patients' ability to modulate their speech volume in response to reading instructions. There were two main findings from this study. Firstly, PD patients were able to regulate mean sentence volume as well as controls in response to explicit instructions, but were always softer because of a lower intensity baseline. Secondly, patients demonstrated a disproportionately higher than normal decay in speech volume towards the end of the sentence. These findings are consistent with Parkinsonian hypokinetic tendencies in the domain of skeletal motor control which have been explained in terms of reduced cortical motor set and motor instability.

Reduced cortical motor set refers to the disturbance in preparatory activity crucial for maintaining the motor programs comprising a complex movement sequence in readiness for execution[8]. Electrophysiological studies have shown reduced pre-movement or readiness potentials (Bereitschaftspotential) in the supplementary motor area (SMA) of Parkinsonian patients when performing automatic movements, thus resulting in deficient scaling of amplitude over the entire movement sequence[4,5]. The set related insufficiency in generating appropriate extent has been demonstrated in PD gait hypokinesia and micrographia. Morris[11] in particular showed that PD patients were able to regulate stride length in response to cadence (stepping rate) manipulation but that patients' stride length was always less than controls' by a constant amount. The present study provides similar findings in the area of speech volume regulation.

In addition to the set deficit in PD, it has been found that movement amplitude deteriorates progressively down a sequence of complex automatic (well-learnt) movments. This sequencing effect has been termed 'motor instability' referring to the inability to maintain the preset amplitude for each sub-movement[8]. The phasic activity in the globus pallidus which acts as a cue to trigger in turn each sub-movement in the motor sequence has been hypothesized to underlie the progressively diminishing amplitude of each component of a movement sequence. Hence the smaller and smaller step size and stroke length in walking[10] and hand-writing[13] respectively. Other upper limb tasks e.g. Georgiou et al[7] also clearly show this cue deficit superimposed on already insufficient movement resulting from reduced preparatory (overall) set.

The present study used the measure of intensity slope to successfully demonstrate the sequencing deficit in PD, in addition to reduced overall mean volume. The intensity slope was an important index because (unlike the mean) it was sensitive to the exaggerated decay in the volume of PD speech over the duration of the breath envelope relative to controls. This sequencing deficit was especially pronounced in the most effortful loud condition where the task demands were greatest. Therefore, while patients were capable of achieving (to the same degree as controls) loud volume relative to normal, patients had great difficulty maintaining vocal intensity.

The findings of the present study are consistent with current concepts of defective fronto-striatal mechanisms[8] in Parkinson's disease and extend these principles to the area of speech motor control.

5. REFERENCES

1. Ackermann H., Zeigler W. Articulatory deficits in Parkinsonian dysarthria: An acoustic analysis. *Journal of Neurology, Neurosurgery and Psychiatry* ***54,*** 1093-1098,1996.

2. Canter G. J. Speech Characteristics of Patients with Parkinson's Disease: I. Intensity, Pitch, and Duration.

*Journal of speech and hearing disorders 28***,** 221-229,1963.

3. Critchley E. M. R. Speech disorders of Parkinsonism: a review. *Journal of Neurology,Neurosurgery and Psychiatry 44***,** 751-758,1981.

4. Cunnington R., Iansek R., Bradshaw J. L., Phillips J. G. Movement related potentials associated with movement preparation and motor imagery. *Experimental brain research 111***,** 429-436,1996.

5. Dick J. P., Rothwell J. C., Day B. L., et al. The Bereitschaftspotential is abnormal in Parkinson's disease. *Brain 112,* 233-244,1989.

6. Fairbanks G. *Voice and articulation drillbook.* 2nd ed. New York: Harper, 1960.

7. Georgiou N., Bradshaw J. L., Iansek R., Phillips J. G., Mattingley J. B., Bradshaw J. A. Reduction in external cues and movement sequencing in Parkinson's disease. *Journal of Neurology, Neurosurgery and Psychiatry 57,* 368- 370,1994.

8. Iansek R., Bradshaw J. L., Phillips J. G., Cunnington R., Morris M. E. Interaction of the basal ganglia and supplementary motor area in the elaboration of movement. In *Motor conrol and sensory integration: Issues and directions,* D. J. Glencross, J. P. Piek, eds. 37-59, New York: Elsevier, 1995:

9. Illes J., Metter E. J., Hanson W. R., Iritani S. Language production in Parkinson's disease: Acoustic and linguistic considerations. *Brain and Language 33,* 146-160,1988.

10. Morris M. E., Iansek R., Matyas T. A., Summers J. J. The pathogenesis of gait hypokinesia in Parkinson's disease. *Brain 117,* 1169-1181,1994.

11. Morris M. E., Iansek R., Matyas T. A., Summers J. J. Stride length regulation in Parkinson's disease: Normalising strategies and underlying mechanisms. *Brain 119,* 551-568,1996.

12. Ramig L., Gould W. J. Speech characteristics in Parkinson's disease. *Neurologic Consultant 4,* 1-8,1986.

13. Selby G. Parkinson's disease. In *Handbook of Clinical Neurology,* P. J. Vinken, G. W. Bruyn, eds. pp 173-211, North Holland, 1968.