

SHORT REPORT

Regulation of parkinsonian speech volume: the effect of interlocuter distance

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Abstract

This study examined the automatic regulation of speech volume over distance in hypophonic patients with Parkinson’s disease and age and sex matched controls. There were two speech settings; conversation, and the recitation of sequential material (for example, counting). The perception of interlocuter speech volume by patients with Parkinson’s disease and controls over varying distances was also examined, and found to be slightly discrepant. For speech production, it was found that controls significantly increased overall speech volume for conversation relative to that for sequential material. Patients with Parkinson’s disease were unable to achieve this overall increase for conversation, and consistently spoke at a softer volume than controls at all distances (intercept reduction). However, patients were still able to increase volume for greater distances in a similar way to controls for conversation and sequential material, thus showing a normal pattern of volume regulation (slope similarity). It is suggested that speech volume regulation is intact in Parkinson’s disease, but rather the gain is reduced. These findings are reminiscent of skeletal motor control studies in Parkinson’s disease, in which the amplitude of movement is diminished but the relation with another factor is preserved (stride length increases as cadence—that is, stepping rate, increases).

(J Neurol Neurosurg Psychiatry 1999;67:199–202)

Keywords: Parkinson’s disease; speech volume; regulation; interlocuter distance

Perceptual studies^{1,2} and acoustic studies^{3,4} have described parkinsonian hypophonic dysarthria as being reduced in volume. Nevertheless, the regulation of this prevalent phenomenon in Parkinson’s disease speech⁵ has not yet been investigated using an experimental paradigm. Speech volume in normal subjects is adjusted appropriately according to the pragmatic demands of the communicatory setting.⁶ Physiological factors which underpin adjustment of speech volume are multiple and include respiratory effort; however, it is the

higher order regulation of these physiological factors that is more likely to be mediated by the basal ganglia,⁷ which is defective in Parkinson’s disease. This study investigated the organisational control of speech by examining the automatic modulation of speech volume in response to a situational cue (interlocuter distance). For healthy people, the speech volume of a speaker naturally increases with increasing interlocuter distance. This paper examined the effect of interlocuter distance in a volume deficient sample of patients with Parkinson’s disease to determine if regulation was impaired, over and above any general volume deficit. Conversation and the recital of sequential material were both examined on the pretext of examining the perception of speech volume at varying distances, and it was expected that controls would show the expected trend in regulating volume but that patients with Parkinson’s disease may differ.

Method

PARTICIPANTS

Twelve patients with Parkinson’s disease (mean age 75.1 (SD 6.2) years, mean Webster score 15.6 (SD 6.5)) with hypophonic speech, and 12 healthy age and sex matched controls (mean age 75.4 (SD 8.05) years) participated in this study. Patients were stabilised on antiparkinson medication and remained on their usual medication regime when participating in the experiment. They were tested between 1 and 3 hours of receiving medication, during their “on” state to ensure uniformity of patients and to control for the possible effect of medication on the speech task. All participants were free from respiratory diseases and were audiometrically screened using a hand held bell tone audiometer (Welch-Allyn) to ensure group homogeneity for hearing ability and thresholds.

APPARATUS

Two Marantz PMD222 audio tape recorders and players were used, one for playing stimulus speech and the other for recording the speech of participants. An additional tape player with an easily manipulable sliding volume control indicator was used by participants to make volume-perception responses.

PROCEDURE

The experiment was conducted in a large quiet room. Participants were seated at a table with a

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Received 14 July 1998 and in
final form
11 December 1998
Accepted 1 March 1999

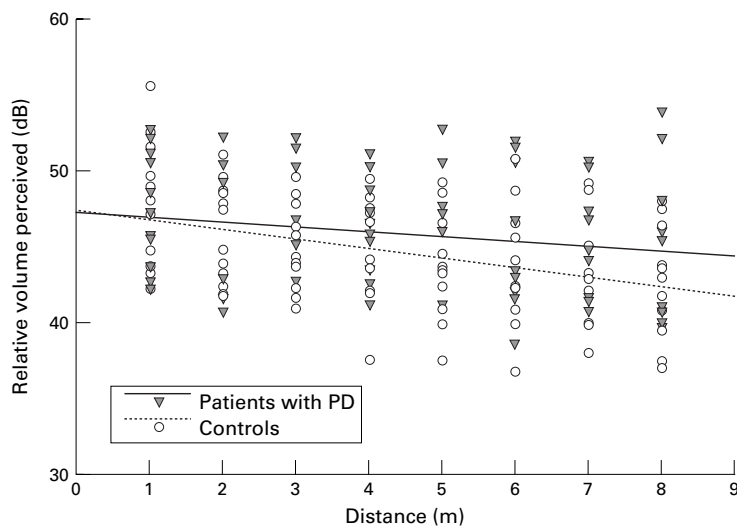


Figure 1 Volume perception (dB) of 12 patients with Parkinson's disease (PD) (filled triangles) and 12 controls (unfilled circles) at 1 to 8 m from interlocuter; plotted with lines of best fit for patients with Parkinson's disease (solid line) and controls (dotted line).

tape player while the experimenter carried a portable tape player to play taped speech at 1 m intervals ranging from 1 to 8 m (scrambled sequence) directly in front of them. Participants were instructed to listen carefully to about 15 seconds of taped speech. Immediately after this, they were required to match the volume of the speech heard by adjusting the volume control slide of the tape player (which played an identical tape) near them until it sounded identical to what they had previously heard. This whole procedure was conducted with participants' eyes closed to avoid estimates based on visual distance information rather than pure loudness judgements. Later, the mean volume corresponding to volume readings were analysed.

Participants were also told that the entire procedure would be tape recorded, and led to think that it was to ascertain the real loudness of the taped speech from where they sat. After each trial, they were instructed to open their eyes and take a quick break, supposedly to prepare them for the next volume perception trial by removing any possible carryover effects from the previous trial. During this time, they conversed with the experimenter or recited simple sequential material (for example, days of the week, counting, spelling) to the experimenter who stood at various distances (1 to 8 m) from them. This covert means of obtaining speech samples directed to the experimenter at various distances was necessary to elicit speech output free of any artificial and conscious modulation of speech volume. Later, the mean volumes of speech samples (after editing out pauses) were obtained using the Kay Elemetrics CSL 400 system. Conversational speech was analysed separately from sequential speech.

Results

Perception data over distance are shown in fig 1, which shows the individual data points of the 12 patients with Parkinson's disease and controls over eight distances, with lines of best fit for each participant group. A two way repeated

measures analysis of variance (ANOVA) was then performed with factors of group (patients with Parkinson's disease, controls) and distance (1 to 8 m). There was a main effect of distance ($F(7,154)=16.72$, $p<0.001$) as both groups perceived a decline in speech volume as distance between experimenter and participant increased. There was no group main effect ($F(1,22)=0.82$, $p=0.374$), but there was a group \times distance interaction ($F(7,154)=2.38$, $p<0.05$). This showed that the groups behaved differently with respect to increasing distance, patients with Parkinson's disease perceived speech to be increasingly louder than controls with increasing distance—that is, there was minimal difference between patients' and controls' perception at 1 m but as distance increased, patients with Parkinson's disease increasingly overestimated volume relative to controls. Alternatively, the perception of volume by the controls decreased more rapidly than the perception of volume by the patients as a function of increasing distance.

The mean volumes of conversational speech are depicted in fig 2 (A), which shows the individual data points of the 12 patients with Parkinson's disease and controls over eight distances, with lines of best fit for each participant group. A two way repeated measures ANOVA showed significant main effects of group ($F(1,22)=5.98$, $p<0.05$) such that patients with Parkinson's disease spoke more softly than controls, and distance ($F(7,154)=16.11$, $p<0.001$) such that speech volume increased when distance increased. There was no group \times distance interaction effect ($F(7,154)=0.56$, $p=0.791$) indicating that patients with Parkinson's disease spoke consistently more softly than controls by a constant amount across all distances. Thus patients with Parkinson's disease started softer than controls at 1 m, and maintained this constant difference over increasing distances up to 8 m.

The mean volumes of sequential speech data are depicted in fig 2 (B), which shows the individual data points of the 12 patients with Parkinson's disease and controls over eight distances. A two way repeated measures ANOVA showed significant main effects of distance ($F(7,154)=4.25$, $p<0.05$), such that speech volume increased for sequential material as distance increased. There was neither a group main effect ($F(1,22)=2.32$, $p=0.142$) nor a group \times distance interaction ($F(7,154)=1.07$, $p=0.386$). Therefore patients with Parkinson's disease and controls produced sequential material at similar volumes at all distances.

A two way repeated measures ANOVA on the data from patients with Parkinson's disease with factors of task (conversation, sequences) and distance (1 to 8 m) showed no task main effect ($F(1,11)=0.25$, $p=0.630$), indicating that volume in patients with Parkinson's disease over increasing distance was similar for both conversation and sequential material. The two way repeated measures ANOVA on control data, however, showed a significant task main effect ($F(1,11)=9.36$, $p<0.05$), such that the volume of controls was louder for conversation

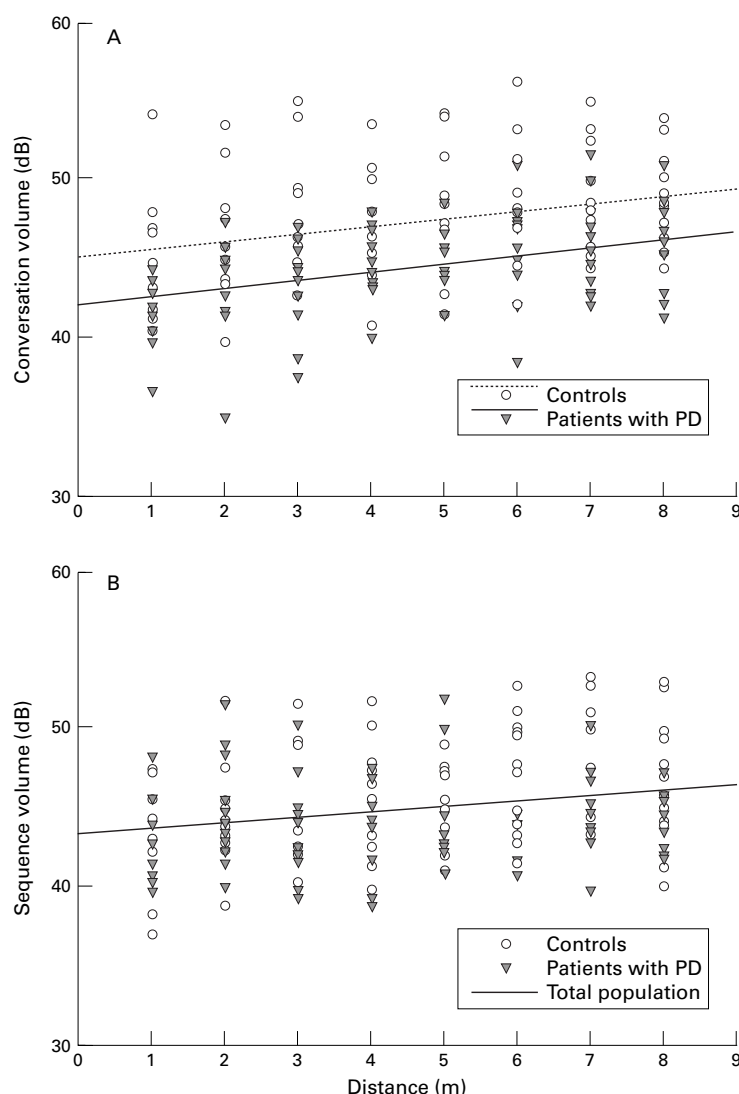


Figure 2 Conversation (top) and sequence (bottom) volumes of 12 patients with Parkinson's disease (PD) (filled triangles) and 12 controls (unfilled circles) at 1 to 8 m from interlocuter; plotted with appropriate lines of best fit.

than for sequential material. Neither patients ($F(7,77)=1.52$, $p=0.173$) nor controls ($F(7,77)=0.41$, $p=0.895$) showed a task \times distance interaction.

Discussion

This study examined the ability of patients with Parkinson's disease to regulate volume in response to distance for two types of speech output (conversation and sequential material). Any difference between patients with Parkinson's disease and controls in speech perception over distance was also investigated.

The production results are discussed firstly in terms of overall volume and secondly, the regulation of volume over distance; then perception data are discussed.

Sequential data showed that patients with Parkinson's disease and controls were similar in overall loudness; however conversation data showed that controls were significantly louder than patients with Parkinson's disease. These different results on overall volume were clearly related to the different type of speech output

elicited by the two tasks. For the (more attentional) sequential production task, participants were asked to recite routine, predictable, and overlearned sequences of numbers or letters. By contrast with this, the (less attentional) conversation task incorporated a secondary cognitive element to speech involving the production of meaningful content material such as participants' personal and unique experiences or descriptions. The reduction in overall loudness (or gain deficit—that is, intercept reduction) for patients with Parkinson's disease was evident for conversation, when less attention was devoted to the production of speech than to the construction of speech content. Furthermore, patients with Parkinson's disease seemed unable to increase their overall speech volume in accordance with the pragmatics of the communicatory function of the conversation task. Indeed, this lack of volume increase for conversational speech may relate to patients' generalised deficit in the appreciation of pragmatics. That patients with Parkinson's disease, unlike controls, failed to increase volume appropriately for conversation suggests that preset speech amplitude is essentially reduced in Parkinson's disease. This reduction in gain has been previously described⁷ and attributed to reduced set production by the basal ganglia in Parkinson's disease. It is also consistent with the findings of upper⁸ and lower⁹ limb movement studies in which the amplitude of movement is miniaturised overall, due to insufficient cortical motor set.¹⁰ This similarity suggests that speech may share a similar mechanism with that of limb movement control.¹¹

For the regulation of volume, patients with Parkinson's disease were still able to gradually and appropriately increase volume over increasing distance (slope similarity). This was seen for both conversation and sequential material. Once again, this finding for speech mirrors the limb movement literature,¹² in which the relation between stride length and cadence (stepping rate) was maintained, although stride length was generally reduced overall, and supports the suggestion that patients with Parkinson's disease are still able to regulate movement amplitude despite an overall dampened baseline level.

Thus far, the findings for speech volume in Parkinson's disease seem consistent with the hypothesis of decreased motor set in movement control in the disease. However, perceptual aspects may also influence resultant speech volumes in Parkinson's disease. Patients' estimation of an interlocuter's speech volumes at increasing distances decreased less rapidly than that of controls; they exhibited overconstancy, perceptually, in this regard. This did not affect the volume regulation of patients disease, which was normal, and may or may not have contributed to the set deficit in overall volume in Parkinson's disease. Nevertheless, the abnormal reduction in range of perceived loudness is interesting given other studies which have demonstrated perceptual difficulties in patients with Parkinson's disease^{12,13} and indicates that perceptual aspects cannot be divorced from motor consequences under investigation. Thus

the perceptual feedback of one's own speech loudness is probably critical in volumetric scaling, and should be examined in future research.

In summary, this paper has found normal regulation of speech volume in response to interlocuter distance (slope similarity) in Parkinson's disease, but overall reduction of gain across all distances (intercept difference). The influence of medication on this gain loss and how rehabilitative strategies¹⁴ in speech volume regulation may influence this deficit will require further investigation in future research.

We acknowledge the assistance of the Linguistics Department of Monash University for the use of the CSL system, and the Australia for Christ Fellowship (ACF) for the use of their premises for testing.

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