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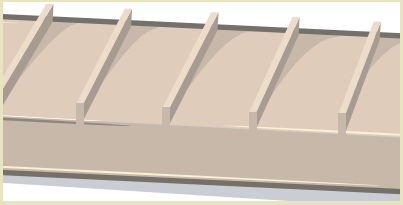

In addition to its widely recognized effects on gait, posture, balance, and upper limb coordination, Parkinson's disease (PD) can have a profound effect on speech and voice, within a cluster of speech characteristics termed hypokinetic dysarthria. Although dopaminergic therapy produces significant benefits in the early stages of PD, speech symptoms may show selective resistance to pharmaceutical therapy in patients with a disease history of more than 10 years. This article discusses the pathophysiology of PD as it relates to speech disorders and considers nonpharmaceutical therapeutic options for hypokinetic dysarthria.

Key words: *Parkinson's disease, speech pathology, dysarthria, treatment*

Nonpharmacological Management of Hypokinetic Dysarthria in Parkinson's Disease

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Introduction

Parkinson's disease (PD) is a degenerative illness whose cardinal symptoms include rigidity, tremor, and slowness of movement.[1] In addition to its widely recognized effects on gait, posture, balance, and upper limb coordination, PD can have a profound effect on speech and voice. Although symptoms vary widely from patient to patient, the speech symptoms most commonly demonstrated by patients with PD are reduced vocal loudness, monopitch, disruptions of voice quality, and abnormally fast rate of speech. This cluster of speech symptoms is often termed hypokinetic dysarthria.[2] While dopaminergic medications are typically effective in treating the voice or speech symptoms that present in the early stages of the disease,[3] some research has suggested that these symptoms may become selectively resistant to pharmaceutical treatment in the latter stages (10+ years) of the disease.[4] Despite the fact that some 60–80% of patients with PD may be expected to develop some voice or speech symptoms,[5,6] it has been estimated that only four percent of these patients receive speech therapy.[7] In this article, we will review the putative pathophysiology of PD as it relates to voice and speech disorders and discuss current trends in the nonpharmaceutical treatment of these symptoms.

The most common symptom of hypokinetic dysarthria is hypophonia, or reduced vocal loudness. Patients demonstrating this symptom may be unaware of the volume at which they are speaking and may require frequent requests to speak louder. Hypokinetic dysarthria also manifests as a lack of variability in pitch or loudness, wherein a patient may demonstrate monopitch, monoloudness, or reduced use of conversational inflection. Similarly, patients with PD often have disruptions of voice quality, in which their voice takes on an abnormal breathiness or hoarseness. Finally, hypokinetic dysarthria can, paradoxically, result in an abnormally fast rate of speech—not unlike the festination of gait that is often a symptom of PD. While it is difficult to attribute these symptoms to specific biological determinants, the pathophysiology of PD is specifically related to speech function in a number of key areas, most notably respiratory or aerodynamic function, laryngeal abnormalities, and motor control.[3]

PD seems to produce a consistent impairment of respiratory function. This impairment takes the form of an overall reduction in function[8] and an increased variability in air flow.[9] Although early research suggested that these abnormalities were caused by reduced movement of glottic and supraglottic structures,[9] more recent research has suggested that they may be more related to irregularities in muscle activation patterns within the chest wall[10] or reduced expansion of the rib cage.[11] Impairment of respiratory function has been implicated strongly in hypophonia and is targeted accordingly by many speech therapy paradigms.

While there are few data on the laryngeal abnormalities of patients with PD, the classic pathophysiology of hypokinetic dysarthria includes dysfunction of vocal fold kinematics[12] (i.e., slow opening and inadequate closing of the vocal folds), vocal fold asymmetry and bowing,[13] and vocal fold paresis.[14] These physiological changes may be responsible for much of the vocal hoarseness and hypophonia seen in hypokinetic dysarthria. It has also been suggested that, in some patients with PD, vocal hoarseness may result from dyskinesia within the laryngeal system.[15]

Given the primarily motoric sequelae of PD, it is not surprising that motor control has been implicated in speech dysfunction. As aforementioned, motor control deficits are related to speech breathing in PD.[10,11] Impaired motor control has also been shown to reduce the speed and amplitude of both jaw movements[16] and lip movements.[17] While it has been hypothesized that reduced amplitude of oral movements is the primary cause of reduced speech intelligibility in PD, the studies required to clearly establish this causal link are still lacking.[3,18]

Nonpharmacological Treatment

Treatment of parkinsonian speech disorders is, unfortunately, limited by the heterogeneity of the physiological determinants of the symptoms. Furthermore, evidence for the long-term efficacy of speech therapy in PD is equivocal, as few studies engage in longitudinal symptom assessment[19] or assess speech in everyday conversational contexts. Nonetheless, there are a number of promising treatments available for patients with PD that involve producing physiological change, compensating for perceptual dysfunctions that result from the disease, or both (Table 1).

One approach to improving outcomes among patients with hypokinetic dysarthria is respiratory effort therapy, a treatment technique that aims to change a patient's physiological capacity for producing higher intensity speech. Respiratory effort therapy focuses on increasing inspiratory and expiratory muscle activity in order to increase subglottal air pressure and respiratory volume. This, in turn, increases vocal loudness without requiring the patient to explicitly alter his/her perception of vocal loudness.[20,21] The most widely recognized behavioural treatment paradigm for patients with PD is, however, the Lee Silverman Voice Treatment (LSVT), a very intensive (16 individual 50-minute sessions within one month) and specific speech therapy program that focuses on ameliorating hypophonia, the most common feature of hypokinetic dysarthria. The LSVT program is based on five fundamental concepts: (1) thinking loud; (2) high speech effort; (3) intensive treatment; (4) recalibrating loudness level; and (5) quantifying improvements. The goal of the technique is to produce a physiological change, as well as a change in perceptual awareness (wherein the patient recognizes his/her own volume and learns to increase volume where necessary).[22,23] Ramig *et al.*[24] have found that this one-month treatment program can produce speech improvements that persist to a two-year follow-up.

Unfortunately, however, it is difficult to evaluate the true effectiveness of speech therapy in ameliorating the effects of hypokinetic dysarthria, given that performance on clinical/laboratory measures may be qualitatively different from everyday speech activities.[16,25–27] Furthermore, research on the cognitive sequelae of PD suggests that the procedural learning deficits demonstrated by this population may prevent the adoption of adaptive speech strategies within a patient's everyday speech habits.[28] Given the difficulties inherent in conducting longitudinal measurement (or even ecologically valid measurement), a promising line of intervention research relates to the use of assistive devices.

Perhaps the simplest assistive device is a personal amplification system, designed to augment the voice of the hypophonic patient by picking up his/her voice through a small microphone and playing it through a small

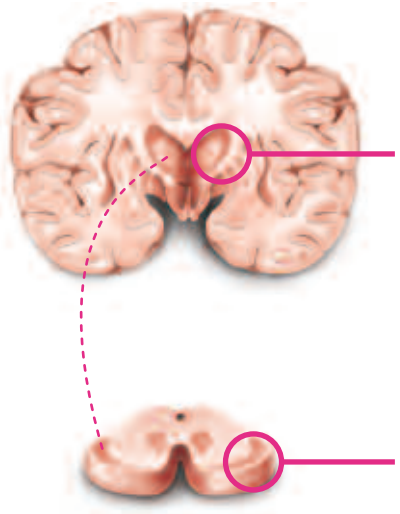
Table 1: Nonpharmacological Management Techniques for Reduced Vocal Loudness and Abnormally Rapid Speech

	Reduced Vocal Loudness	
Behavioural	**Biofeedback**	**Assistive Device**
vocal pushing	sound level metre	portable amplifier
increased vocal effort	intensity feedback display	telephone amplifier
"think loud"	speech intensity monitor	white noise generating system (Lombard effect)
deeper, more forceful breaths		
improved posture		
large, forceful oral movements		
	Abnormally Rapid Speech	
imitation of slower speech	computerized rate feedback	pacing board (Figure 1)
stretching out vowels	computerized rate pacing	portable delayed auditory feedback devices (Figure 1)
longer, more frequent pauses	metronome rate pacing	

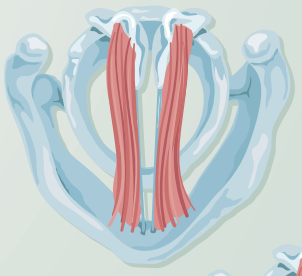
Figure 1:
Management of Hypokinetic Dysarthria in Parkinson's Disease

Hypokinetic Dysarthria

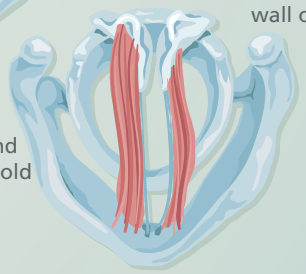
Hypokinetic dysarthria is a motor speech disorder associated with basal ganglia control circuit pathology. It is due to a lesion in the substantia nigra where dopamine is produced by neurons. In order to have normal muscle movement dopaminergic and cholinergic pathways must be in balance.

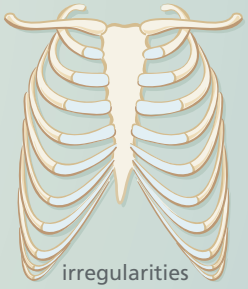


muscles involved with vocal loudness



dysfunction of vocal fold kinematics, vocal fold asymmetry and bowing, or vocal fold paresis

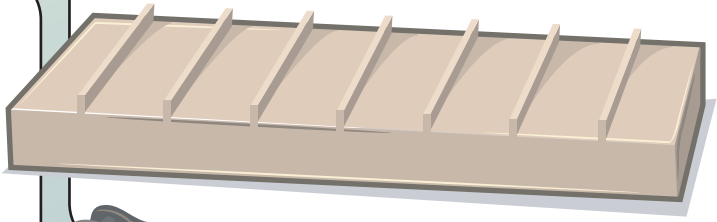




irregularities in muscle activation patterns within the chest wall or reduced expansion of the rib cage

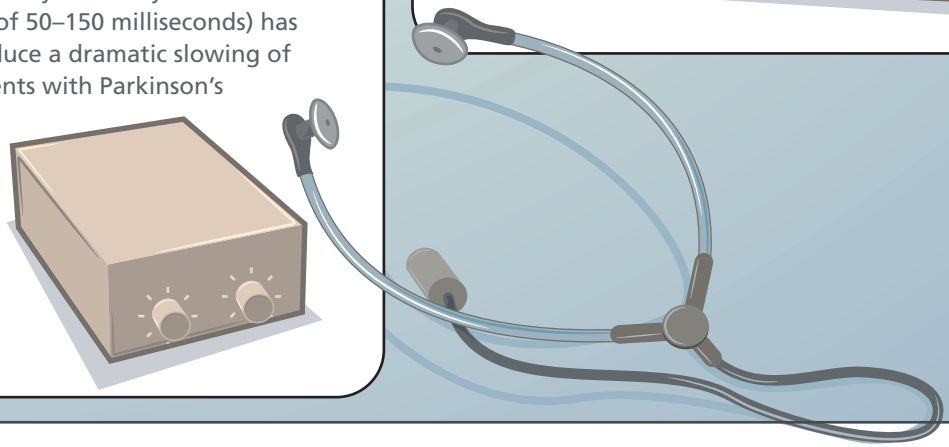
pacing board

A pacing board is used for Parkinson's patients suffering from dysarthria — a condition exhibiting an abnormally fast rate of speech. Divided into seven equally-spaced dividers, the patient taps one section, from left to right, every time a syllable is pronounced. As the fingers cannot move as quickly, this should slow the rate of speech. Once a reduced rate is achieved using the board, the patient can count syllables on their fingers, and eventually will be able to speak at a normal rate without any means of aid.



portable delayed auditory feedback device

This instrument is a powerful assistive speaking device. The advantage of the delayed auditory feedback device over the pacing board is its portability and simplicity. Auditory feedback delay (in the range of 50–150 milliseconds) has been shown to produce a dramatic slowing of speech among patients with Parkinson's disease for a period of at least two years.



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speaker integrated in a portable amplifier.[29] Another method that shows promise for improving hypophonia is the presentation of white noise to the dysarthric individual, thereby disrupting his/her perception of vocal loudness and causing him/her to speak more loudly to compensate. This device is based on the Lombard effect and has been shown to produce positive changes among patients with PD.[30,31]

Both of these assistive devices are, however, focused on the improvement of hypophonia, which is only one speech symptom in PD. As aforementioned, PD can also produce an abnormally fast rate of speech. One device that may be applied to the treatment of this symptom is a pacing board, a narrow board with seven equally spaced dividers along its length (Figure 1). Patients are trained to tap from left to right between the dividers, making one tap per syllable, producing a significant reduction in rate of speech.[29,32] Another device that has been used to treat rapid speech is the portable delayed auditory feedback device (Figure 1). The advantage of this device over the pacing board is, of course, its greater portability and its low demands for patient instruction. Auditory feedback delay (in the range of 50–150 milliseconds) has been shown to produce a dramatic slowing of speech among patients with PD that persists for at least two years.[18,33]

Conclusion

As the overall clinical management of PD has improved, we have seen the mortality rate of this population decrease to approximately that of the normal population. To the extent that pharmaceutical treatments become less effective (in general) as the disease progresses, nonpharmaceutical treatment options become increasingly important for maintaining patients' quality of life. Fortunately, there are a number of such options available for the clinical management of hypokinetic dysarthria, ranging from speech therapy to wearable assistive devices. Although further research is needed on the long-term effectiveness of these therapies, current findings suggest that the positive effects derived from these treatments are not easily extinguished with proper management within a speech clinic.

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References

1. Waters CH. Diagnosis and management of Parkinson's disease. Los Angeles: Professional Communications, Inc, 1998.
2. Darley FL, Aronson AE, Brown JR. Motor speech disorders. Philadelphia: W.B. Saunders Company, 1975.
3. McNeil MR, Ed. Clinical management of sensorimotor speech disorders. New York: Thieme Publishing, 1997.
4. Bonnet AM, Loria Y, Saint-Hilaire MH, et al. Does long-term aggravation of Parkinson's disease result from nondopaminergic lesions? Neurology 1987;37:1539–42.
5. Mutch WJ, Strudwick A, Roy SK, et al. Parkinson's disease: disability, review, and management. BMJ 1986;293:675–7.
6. Streifler M, Hofman S. Disorders of verbal expression in parkinsonism. Adv Neurol 1984;40;385–93.
7. Ramig LO. Treatment of speech and voice problems associated with Parkinson's disease. Top Geriatric Rehab 1998;14:28–43.
8. Mueller PB. Parkinson's disease: motor-speech behavior in a selected group of patients. Folia Phoniatrica 1971;23:333–46.
9. Vincken WG, Gauthier SG, Dollfuss RE, et al. Involvement of upper-airway muscles in extrapyramidal disorders: a cause of airflow limitation. N Engl J Med 1984;311:438–42.
10. Murdoch BE, Chenery HJ, Bowler S, et al. Respiratory function in Parkinson's subjects exhibiting a perceptible speech deficit: a kinematic and spirometric analysis. J Speech Hear Disord 1989;54:610–26.
11. Solomon NP, Hixon TJ. Speech breathing in Parkinson's disease. J Speech HearRes 1993;36:294–310.
12. Gerratt BR, Hanson DG, Berke GS. In: Baer T, Sasaki C, Harris K. Laryngeal function in phonation and respiration. Boston: College Hill Press, 1987: 521–32.
13. Hanson DG, Gerratt BR, Ward PH. Cinegraphic observations of laryngeal function in Parkinson's disease. Laryngoscope 1984;94:348–53.
14. Schley WS, Fenton E, Niimi S. Vocal symptoms in Parkinson's disease treated with levodopa: a case report. Ann Otol Rhinol Laryngol 1982;91:119–21.
15. Ludlow CL, Bassich CJ. In: McNeil MR, Rosenbek JC, Aronson AE. The dysarthrias: physiology, acoustics, perception, management. San Diego: College-Hill Press, 1984: 163–92.
16. Connor NP, Abbs JH, Cole KJ, et al. Parkinsonian deficits in serial multiarticulate movements for speech. Brain 1989;112:997–1009.
17. Forrest K, Weismer G, Turner GS. Kinematic, acoustic, and perceptual analyses of connected speech produced by parkinsonian and normal geriatric adults. J Acoustic Soc Amer 1989;85:2608–22.
18. Adams SG. Motor speech disorders. In: Till JA, Beukelman DR, Yorkston KM, eds. Advances in assessment and treatment. Maryland: Brookes Publishing, 1993.
19. Stemple J, Glaze L, Gerdemann B. Clinical voice pathology: theory and management. San Diego: Singular Publishing Group, 2000.
20. Baumgartner CA, Sapir S, Ramig TO. Voice quality changes following phonatory-respiratory effort treatment (LSVT) versus respiratory effort treatment for individuals with Parkinson disease. J Voice 2001;15:105–14.
21. Ramig LO, Countryman S, Thompson LL, et al. Comparison of two forms of intensive speech treatment for Parkinson disease. J Speech Hear Res 1995;38:1232–51.
22. Ramig LO. How effective is the Lee Silverman voice treatment? American Speech-Language Hearing Association 1997;39:798–807.
23. Ramig LO, Sapir S, Fox C, et al. Changes in vocal loudness following intensive voice treatment (LSVT) in individuals with Parkinson's disease: a comparison with untreated patients and normal age-matched controls. Mov Disord 2001;16:79–83.
24. Ramig LO, Sapir S, Countryman S, et al. Intensive voice treatment (LSVT) for patients with Parkinson's disease: a 2 year follow up. J Neurol Neurosurg Psychiatry 2001;71:493–8.
25. Connor NP, Abbs JH. Task-dependent variations in parkinsonian motor impairments. Brain 1991;114:321–32.
26. Countryman S, Hicks J, Ramig L, et al. Supraglottal hyperadduction in an individual with Parkinson's disease: a clinical treatment note. Amer J Speech-Lang Pathol 1997;6:74–84.
27. Stemple J. Voice therapy: clinical studies. San Diego: Singular Publishing Group, 2000.
28. Saint-Cyr JA, Taylor AE, Lang AE. Procedural learning and neostriatal dysfunction in man. Brain 1988;111:941–59.
29. Yorkston KM, Beukelman DR, Bell KR. Clinical management of dysarthric speakers. Boston: College-Hill Press, 1988.
30. Adams SG, Haralabous O, Dykstra A, et al. Effects of multi-talker background noise on the intensity of spoken sentences in Parkinson's disease. Canadian Acoustics (in press).
31. Adams SG, Lang AE. Can the Lombard effect be used to improve low voice intensity in Parkinson's disease? Eur J Dis Comm 1992;27:121–7.
32. Lang A, Fishbein B. The "pacing board" in selected speech disorders of Parkinson's disease. J Neurol Neurosurg Psychiatry 1983;46:789.
33. Downie AW, Low JM, Lindsay DD. Speech disorder in parkinsonism; use of delayed auditory feedback in selected cases. J Neurol Neurosurg Psychiatry 1981;44:852.