

Henry Ford Health

Henry Ford Health Scholarly Commons

Neurology Articles

Neurology

6-13-2021

Conversational Vocal Intensity in Parkinson's Disease: Treatment and Environmental Comparisons

Ramya Konnai

Henry Ford Health, RKONNAI1@hfhs.org

Meredith Van Harn

Henry Ford Health, mvanhar1@hfhs.org

Alice K. Silbergleit

Henry Ford Health, asilber1@hfhs.org

Follow this and additional works at: https://scholarlycommons.henryford.com/neurology_articles

Recommended Citation

Konnai R, Van Harn M, and Silbergleit A. Conversational Vocal Intensity in Parkinson's Disease: Treatment and Environmental Comparisons. *J Voice* 2021.

This Article is brought to you for free and open access by the Neurology at Henry Ford Health Scholarly Commons. It has been accepted for inclusion in Neurology Articles by an authorized administrator of Henry Ford Health Scholarly Commons.

Conversational Vocal Intensity in Parkinson's Disease: Treatment and Environmental Comparisons

*Ramya Konnai, †Meredith Van Harn, and ‡Alice Silbergleit, *‡West Bloomfield, and †Detroit, Michigan

Abstract: Background. Vibrotactile Feedback (VF) using wearable devices is an emerging treatment option for hypophonia in Individuals with Parkinson's disease (IwPD). Studies evaluating the effectiveness of VF in improving conversational vocal intensity in real-life environment in IwPD are limited.

Objective. To determine the effect of VF on conversational vocal intensity and compare vocal intensity between a) clinic and real-life environment b) VF and Lee Silverman Voice Treatment (LSVT LOUD®) vs. VF alone in IwPD using a portable voice monitor (VocaLog2).

Methods. Eight individuals with hypophonia secondary to PD were randomly assigned to two treatment groups- VF and LSVT LOUD® (Group 1) and VF (Group 2). VF was provided using VocaLog2 device. Duration of treatment was 4 weeks for both groups. Vocal intensity was measured in the real-life environment at baseline, during treatment, and at one-month follow-up.

Vocal intensity in clinic was obtained at baseline and one-month follow-up. Voice Handicap Index (VHI) questionnaire was administered at baseline and one-month follow-up.

Results. There was no significant difference in conversational vocal intensity between a) clinic and real-life environment at any point of time b) baseline and follow up for both treatment groups c) the two treatment groups at baseline, during each of the 4 weeks of treatment and at follow up d) VHI baseline and one month follow up scores.

Conclusion. VF, including when combined with LSVT LOUD®, is limited in improving conversational vocal intensity in real-life in IwPD. The effects of frequency and duration of VF on conversational vocal intensity must be systematically investigated using large scale studies in IwPD.

Key words: Vocal intensity—Parkinson's disease—Wearable device—Vibrotactile feedback—Hypophonia—LSVT LOUD®—Real-life environment.

INTRODUCTION

Parkinson's disease (PD) is estimated to affect over one million people across North America by 2030.¹ Speech is affected in about 90% of Individuals with Parkinson's Disease (IwPD) during the disease course.² Speech deficits in PD are associated with an altered recruitment of the main motor cerebral regions (orofacial motor cortex, cerebellum), and an increased involvement of premotor and prefrontal cortices.^{3,4} Hypokinetic dysarthria is a collective name for speech impairments in PD and is characterized by decreased vocal loudness, monotony of pitch, breathy, and harsh voice quality, reduced stress, variable rate, short rushes of speech, and imprecise consonants.⁵ Decreased vocal intensity or hypophonia can be an initial speech symptom in PD.⁶ Hypophonia is attributed partly to hypokinesia (reduced amplitude of movement) and rigidity caused by underlying dopaminergic deficiency.⁷⁻¹² Abnormalities in central sensory processing (reduced awareness of soft voice), internal cueing (difficulty self-generating increased loudness), and self-monitoring of speech output also contribute to

hypophonia.¹³⁻²² Both problems of sensory processing and internal cueing, in addition to the motor problems, may contribute to challenges in treatment.²³ Studies have compared the conversational vocal intensity of IwPD with healthy controls using portable voice monitors and reported 2-8 dB decrease in the PD group.^{24,25} Hypophonia contributes to significant difficulty communicating and participating effectively in a variety of daily speaking situations.

Voice and speech assessments are traditionally obtained in a controlled environment such as soundproof booths in a lab or clinic setting to ensure data reliability and replicability. Voice and speech produced in a lab setting may not necessarily represent how IwPD use their voice in their daily speaking situations such as in a restaurant, at work, or in the car. While "lab speech" is necessary to research certain aspects of speech which requires systematic experimental control (eg, vowel acoustics or aerodynamics), it is important to focus on functional tasks such as conversation in real-life environments when studying carry over of treatment effects. The absence of real-life environmental demands such as cognitive load, performance effects, background noise, and environmental cues in the clinic may produce results different than that of real-life environment.²⁶ Studies comparing voice use in clinic and real-life environments in IwPD are limited and the results are mixed.²⁵⁻²⁷ The differences in the findings of these studies is likely due to differences in methodology such as the number of participants studied, length of conversation samples, and technology used to monitor vocal intensity. More studies are

Accepted for publication April 8, 2021.

From the *Department of Neurology, Henry Ford Health System, West Bloomfield, Michigan; †Department of Public Health Sciences, Henry Ford Health System, Detroit, Michigan; and the ‡Department of Neurology, Henry Ford Health System, West Bloomfield, Michigan.

Address correspondence and reprint requests to Ramya Konnai, Department of Neurology, Henry Ford Health System, West Bloomfield, MI 48322 E-mail: rkonnai@hfh.org

Journal of Voice, Vol. ■■■, No. ■■■, pp. ■■■–■■■
0892-1997

© 2021 The Voice Foundation. Published by Elsevier Inc. All rights reserved.
<https://doi.org/10.1016/j.jvoice.2021.04.019>

needed to investigate if IwPD use their voice differently in real-life environment compared to the clinic as real-life communication is important to their quality of life. Portable voice monitors are becoming available to monitor voice in real-life. One such monitor that is available for research is the Vocalog2 (VL2, Griffin Laboratories, Temecula, CA). The VL2 monitors vocal intensity and can provide vibrotactile feedback when an individual speaks above or below a threshold level. It does not measure fundamental frequency or environmental noise. The accuracy of VL2 in its measurement of vocal intensity was determined in reference to a head mounted microphone and there was good agreement with a mean error of -0.4 dB.²⁸ It should be noted that the beta version of VocaLog (*software version 1.2.4.2*) was tested in 3 different studies but the device has been modified since then.²⁹⁻³¹ The VL2 is different from the beta version in terms of the sensor used and calibration process (C. Griffin, personal communication, Nov 5, 2020). A recent study monitored IwPD in their real-life environment using a portable voice accumulator and found that IwPD use their voice 50%-60% less than their matched healthy controls in daily life.²⁵ Maintaining vocal volume in a patient's everyday life is crucial for continuing socialization and limiting communication isolation and the potential accompanying depression found in IwPD.³²

Traditionally, voice therapy for hypophonia is based either on behavioral treatment which involves training to strengthen muscles involved with coordination of respiration, phonation, and articulation or using devices that provide environmental cues or biofeedback or voice amplification.³³ Lee Silverman Voice Treatment, LSVT LOUD[®] (LSVT Global, Tucson, AZ) is a standardized and leading treatment of choice for hypophonia in IwPD.³⁴ This technique includes an intensive, high effort treatment with focus on increasing phonatory effort and vocal fold adduction as well as improving sensory perception of effort.²² Treatment is delivered 4 times a week over 4 weeks. Therapy tasks include a hierarchy of loud sustained vowel to functional phrases, reading, and conversational speech.²² Existing randomized control studies have shown that LSVT LOUD[®] can result in improved vocal intensity in monologue from 4.7 to 5.5 dB SPL and that the benefits of treatment can last for up to 2 years.^{22,35-37} Recalibration of internal cueing and self-regulation of vocal effort is a mainstay of LSVT LOUD[®].³⁵ However, reports regarding the carry-over of LSVT LOUD[®] treatment to real-life conversation are lacking with the exception of a single subject study where the IwPD increased voice intensity with 4.1 dB in real-life and 5.6 dB in the lab post-treatment.³⁸

Wearable devices may help with carryover of treatment effects for some IwPD or be an alternate option to treat hypophonia. The use of wearable devices for treatment of hypophonia in IwPD dates to the 1980.^{39,40} Some devices use biofeedback to improve hypophonia. Biofeedback is a "process of transducing some physiologic variable, transforming the signal to extract useful information and

displaying that information to the subject in a format that will facilitate learning to regulate the physiological variable."⁴¹ Biofeedback can be auditory, visual or vibrotactile in nature. Recently, VF was provided to a small group of six IwPD using Voxlog, a portable voice monitor and a 1.5 dB increase in conversational vocal intensity was demonstrated compared to no feedback condition and vocal intensity did not decrease significantly when feedback was removed.²³ Van Stan et al, (2015) point out that 1.5 dB may be statistically significant but may not represent a clinically significant change considering that IwPD speak approximately 10 dB lower than age matched controls.⁴² In a single case study using the Ambulatory Phonation Monitor (APM), a portable voice monitor, conversational vocal intensity decreased by 9 dB post-biofeedback when measured in the clinic.⁴³ Improvements in vocal intensity and generalization of treatment benefits could be related to the frequency, duration, type of biofeedback provided. A comparison of six different biofeedback configurations in IwPD showed that activating feedback when an individual speaks 3 dB below their mean vocal intensity study for at least 500 ms elicited optimal outcomes.⁴⁴ Also, intermittent biofeedback has been shown to be more effective than constant feedback.⁴⁵ Hence most studies in the literature investigating the effect of biofeedback on vocal intensity have used intermittent feedback but the nature of intermittent feedback varies within studies.^{23,46} In summary, VF by itself seems to be limited in improving vocal intensity in IwPD. Thus, a combination of treatments such as LSVT LOUD[®] and VF may be beneficial in improving vocal intensity in natural environments in IwPD compared to VF alone. This preliminary exploratory study aimed to address this using the VL2. Evidence also suggests that IwPD seem to need more time to achieve motor learning, especially to achieve automatization.⁴⁷ The previous studies that utilized VF in IwPD were limited to one week and this may not have been enough to improve vocal intensity in IwPD.^{23,43} Another rationale for undertaking this study was to provide VF for a longer duration (2 weeks) to facilitate motor learning. The aims and hypotheses of the present study are

1. To determine the effects of VF on conversational vocal intensity in IwPD in real-life environments and compare it with VF and LSVT LOUD[®] treatment combination.

We hypothesized that VF will produce higher vocal intensity at the end of treatment compared to baseline and conversational vocal intensity using LSVT LOUD[®] and VF treatments will be greater than using VF alone.

2. To compare conversational vocal intensity obtained in the clinic vs. real-life environments in IwPD using VL2 monitor.

We hypothesized that conversational vocal intensity in the clinic will be greater than the real-life environment.

3. To assess short-term carry over of LSVT LOUD[®] and VF treatments in real life environment.

We hypothesized that both treatment groups (VF combined LSVT LOUD[®] and VF) will carry over to real-life after a one-month time period.

METHODS

Participants

Twelve individuals (6 males) were initially enrolled in the study. They were recruited from an outpatient clinic in a hospital setting. Inclusion criteria included diagnosis of idiopathic PD by a neurologist specializing in movement disorders, complaints of hypophonia, and normal hearing based on audiological evaluation within the past year of enrollment. Exclusion criteria included DBS surgery, dementia as reported by the physician in the patient's medical record, LSVT LOUD® therapy in the last two years, and vocal fold pathology unrelated to hypokinetic dysarthria (such as vocal fold nodules). Study participants retained their regular medication schedule. Four IwPD dropped out of the study due to personal reasons. Therefore, eight IwPD completed the study, [Table 1](#). The study was approved by the institutional review board at Henry Ford Health System. All participants signed an informed consent form.

Instrumentation

The VocaLog2 (VL2) system was used to measure participants' vocal intensity in the clinic and in real-life environments. The VL2 system consists of the VocaLog 2 activity monitor worn by the subject to monitor and record vocal activity, the computer software, and a calibrated Microphone (Samson Go mic model SAGOMIC). Using the VocaLog2 Desktop Application on a computer, the clinician downloaded the data recorded by the monitor. The VL2 monitor consists of a laryngeal sensor and a neck band and logs sound pressure level using the dBC scale. It was also used to provide VF when vocal intensity dropped below a pre-determined threshold for 500 ms. This feature can be disabled by turning a switch when VF is not required. Subjects were sized for either of the available sizes, VL2 28 or VL2 35, to ensure full contact of the laryngeal sensor with the neck.

Procedure

An initial speech evaluation was completed in the clinic consisting of case history, Consensus Auditory-Perceptual Evaluation of Voice (CAPE-V)⁴⁸ to determine the severity of hypophonia, Voice Handicap Index (VHI)⁴⁹, a patient reported outcomes measure that quantifies the impact of a voice disorder on a person's quality of life, the St. Louis University Mental Status (SLUMS) examination⁵⁰, and videostroboscopy by a speech-language pathologist (AS, third author). The videostroboscopy recordings were further reviewed by a laryngologist to rule out vocal fold pathology unrelated to hypokinetic dysarthria in all participants. Patients that were eligible and consented to participate in the research study returned to the clinic for a second visit to calibrate the VL2 and measure baseline conversational vocal intensity. Baseline vocal intensity was obtained in the clinic using the VL2 during 20-minutes of informal conversation with the clinician. For the purpose of this study, "conversational vocal intensity" was defined as vocal intensity measured during an unstructured dialogue between IwPD and clinician/communication partner (s). Conversation revolved around the participants' topics of interest. Following this clinic visit, baseline vocal intensity was also measured in the real-life environment. Real-life environment was defined as the natural environment where an individual is likely to communicate such as home, office, restaurant, etc. Participants wore the VL2 monitor for a total of four hours each day for three days for baseline measurement of vocal intensity in real-life. Participants were instructed to wear the VL2 monitor when they were likely to participate in conversation (eg, phone conversations, social outings). Participants were also instructed to maintain a daily log of when the device was turned on and off, type of speaking situation (home, restaurant, work, etc), number of people present, comments. The purpose of this daily log was to verify the date and time data that was displayed on the VL2 with the times entered by the participant. Following baseline

TABLE 1.
Subject Characteristics

Subject	Age	Gender	Yearsof PD	Severityof dysphonia (based on CAPE-V loudness scale)	CAPE-V loudness score	VHI score &Severity	Treatment Group	Cognition
1	51	M	8	Mild	14	60 (severe)	VF only	Normal
2	58	M	10	Moderate	46	70 (severe)	VF only	Normal
3	71	M	1	Mild-moderate	27	9 (normal)	VF only	Normal
4	63	F	7	Mild	15	65 (severe)	VF only	Normal
5	61	F	1	Mild	18	43 (moderate)	VF only	Normal
6	76	M	5	Mild	10	52 (moderate)	LSVT & VF	MCI
7	56	M	4	Moderate	30	90 (severe)	LSVT & VF	MCI
8	74	M	6	Moderate	13	58 (severe)	LSVT & VF	MCI

Abbreviation: CAPE-V, Consensus Auditory-Perceptual Evaluation of Voice; VHI, Voice Handicap Index; VF, Vibrotactile Feedback; LSVT, Lee Silverman Voice Treatment LOUD®; MCI, Mild Cognitive Impairment

measurements, participants were then randomly assigned to one of two treatment groups- Group 1 received VF via the VL2 (outside the clinic) and LSVT LOUD® (in-clinic) while Group 2 received VF only (outside the clinic). Group 1 consisted of five participants with normal cognition and group 2 had three participants with mild cognitive impairment (MCI) who lived independently and were able to follow directions. Group 1 received LSVT LOUD® treatment by a certified LSVT LOUD® trained clinician (RK, first author) according to the recommended protocol of 4 days per week for 4 weeks. Participants in Group 1 did not wear the VL2 during LSVT LOUD® therapy in the clinic but wore the monitor outside of the clinic to monitor their vocal intensity in real-life. Data from the VL2 was downloaded for Group 1 once a week. Participants in group 2 returned to clinic at the end of each week for four weeks to download the data from the VL2. Informal discussion took place with the clinician during data download for both groups where the clinician reminded them to check for placement of the VL2 sensor, to pay attention to the VF, and made sure that the daily log was being maintained. Both groups wore the VL2 outside of the clinic for 4 weeks during treatment, but VF was provided intermittently during weeks one and three for both groups. The VF feature was turned off by the clinician in the clinic for weeks two and four. During weeks one and three, VF was automatically initiated by the VL2 when participants spoke below the set threshold level for 500ms. The threshold level was set to 4 dB above the participant's average baseline vocal intensity obtained in the real-life environment. Participants were aware whether VF was on or off. Participants were instructed that the VF was a cue that their volume was dropping and that they had to speak louder when they felt the vibration. They were specifically instructed not to ignore the VF. At the end of 4 weeks of treatment, participants returned the VL2 monitor to the clinic. All participants returned to the clinic one month after the end of treatment for follow-up. Vocal intensity during conversation was monitored for 20 minutes in the clinic during the one-month follow-up session using the VL2 system. The VHI questionnaire was re-administered in the clinic during this follow-up visit. Participants were sent home with the VL2 to be worn for three days (four hours each day) to measure vocal intensity outside the clinic. They did not receive any VF during these three days and the goal was to monitor their vocal intensity to assess for short-term carry over. Participants returned to the clinic following the 3-days to return the VL2 monitor and downloading of the data. During this visit, participants provided feedback on the VL2 on various aspects such as comfort, nature of vibration. Figure 1 shows a flowchart of the data collection process. All analyses were performed using SAS 9.4 (SAS Institute Inc, Cary, NC, USA). A repeated-measures generalized estimating equations approach was used to account for the lack of independence inherent in this design. Data are presented using least-squared means and standard errors. Statistical significance was set at $P < 0.05$.

RESULTS

There was no significant difference in conversational vocal intensity between a) clinic and real-life environment at any point of time ($P = 0.268$, Table 2) b) the two groups at baseline, during each of the 4 weeks of treatment and at follow up (Table 3). However, when data was pooled across the four weeks, group two had significantly higher conversational vocal intensity than group 1 ($P = 0.044$) c) VF on and off in group 2 ($P = 0.678$) d) baseline and one-month follow-up for both treatment groups in real life environment and in the clinic (Table 4) e) VHI baseline and one month follow up scores ($P = 0.082$). Figure 2 shows the average vocal intensity for each participant at various time points. Only participant eight with moderate hypophonia at baseline showed increase in vocal intensity post-treatment (LSVT LOUD & VF) compared to baseline. No improvement was noted in conversational vocal intensity between baseline and follow up for remaining seven participants.

DISCUSSION

Clinic vs. real-life environment comparison: In this study the average vocal intensity obtained in the clinic was approximately 2 dB higher than that obtained in the real-life environment, but this difference was not statistically significant. One of the reasons for this finding is possibly the duration of conversation sample obtained in the clinic (20 minutes). Previous studies comparing clinic and home environments have obtained monologue or connected speech samples in the clinic that are of shorter duration (30 seconds-three minutes).^{23,26,27,38} Twenty minutes of conversation in clinic was obtained in this study to be consistent with the LSVT LOUD® protocol.³⁷ A preliminary investigation into establishing initial benchmarks for obtaining robust estimates of long-term monitoring of voice recommended an hour-long monitoring of voice for an error rate of about 5% in the average sound pressure level curve.⁵¹ An hour-long monitoring of vocal intensity may not be feasible in a busy clinic environment. Accuracy level of the SPL measures obtained during a 20-minute sample may have an increased error rate more than 5% but is a practical time frame for clinical voice assessment. It is also possible that when recordings last for 20 minutes in the clinic, that “performance effect” or Hawthorne effect (defined as alteration of a subject's behavior due to their awareness of being observed)⁵² is less likely compared to the 30 second to 3 minutes of monologue samples. The correlation between recording duration in the clinic and conversational vocal intensity must be further investigated in IwPD. Since there was no difference in vocal intensity with 20 minutes of connected speech data obtained in the clinic and real-life environment, this duration may be recommended for clinical assessment for a good representation of conversational vocal intensity in the real-life environment.

Effects of treatment on conversational vocal intensity in real-life environments: This study's findings are in agreement with another similar study where the combined use of LSVT

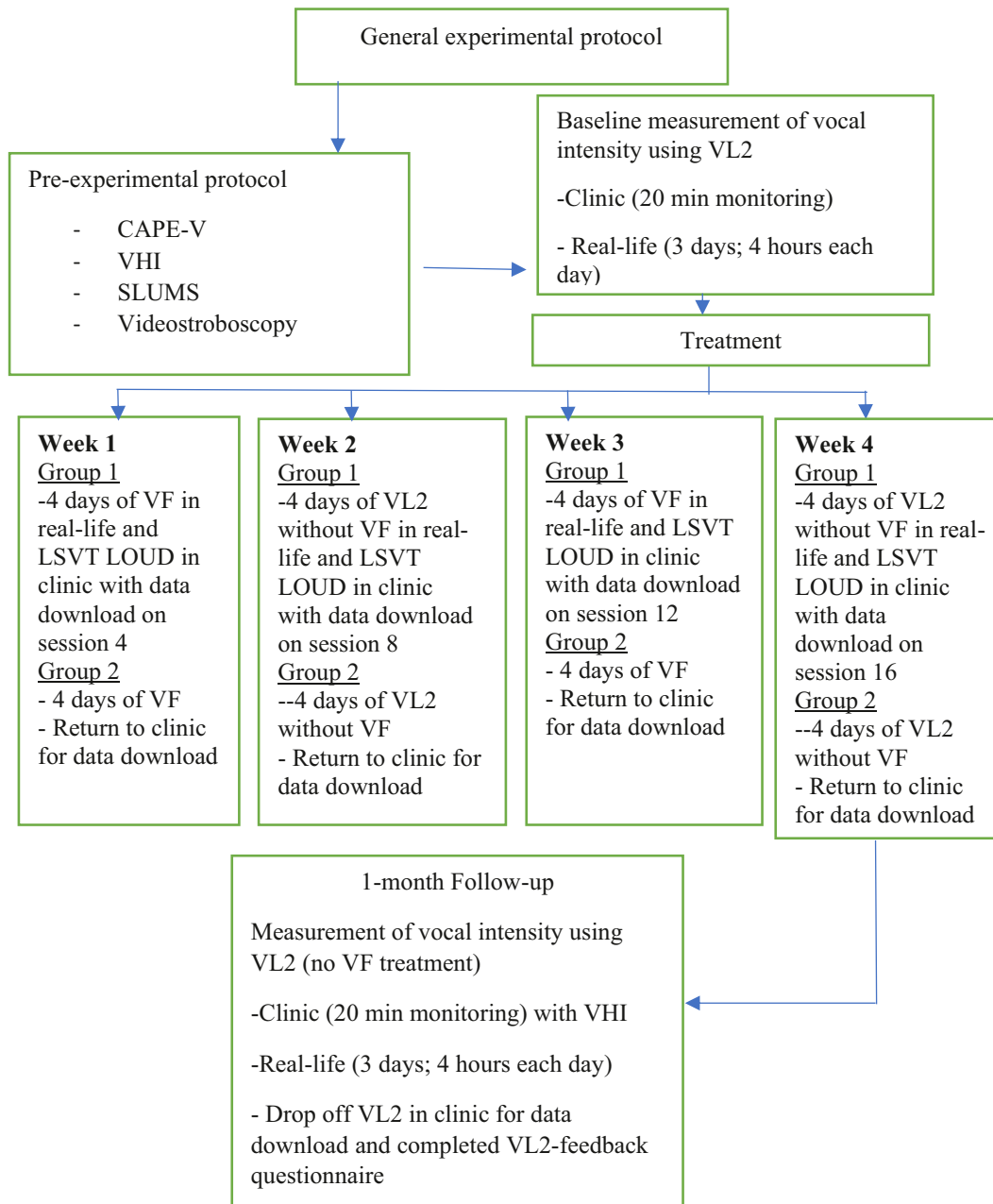


FIGURE 1. Flowchart of data collection process

LOUD[®] and VF in IwPD did not demonstrate better maintenance of target loudness levels than LSVT LOUD[®] alone, as well as no reports of consistent increase in vocal intensity over the course of LSVT LOUD[®] therapy.⁵³ Treatment effects of LSVT LOUD[®] has been mostly studied in the clinic and using various tasks such as sustained phonation, reading, monologue. However, the monologue measured in clinic has been brief ranging from 30 to 60 second samples.³⁵⁻³⁷ Improvements reported in the conversational measures of speech intensity in clinic are inconsistent across studies. For example, maintenance of increased speech intensity for conversation at follow-up was found in some studies^{36,37,54} while other studies^{35,55} failed to find evidence for improvements in speech intensity

for conversation at follow-up. An audit by Wight and Miller (2015) replicated this finding. Their clients continued to have louder sustained vowel production two years post LSVT LOUD[®] but did not maintain increased loudness in reading and monologue tasks during the 12 month and 24-month follow up.⁵⁶ In another single case study, the IwPD increased conversational voice intensity in daily life by 4.1 dB post- LSVT LOUD[®] treatment and 1.4 dB at one-year follow-up compared to before treatment.³⁸ While this finding is promising, it cannot be generalized to the PD population. More studies are needed to determine if treatments such as LSVT LOUD[®] and/or biofeedback improve carry over of conversational loudness from clinic to real-life environments.

TABLE 2.
Least Square Mean with Standard Errors of Vocal Intensity in Clinic and Real-life Environment at Baseline, End of Treatment and During One-month Follow-up

Variable (Environment)	Response	LS Mean (SE) of dB	P-Value
Baseline	Clinic	71.7 (1.5)	0.251
	RLE	69.6 (0.8)	
Group 1 at end of treatment	Clinic	71.9 (3.3)	0.341
	RLE	67.6 (1.0)	
Group 2 at end of treatment	Clinic	70.8 (1.5)	0.735
	RLE	69.5 (1.7)	
Group 1 at follow up	Clinic	72.7 (3.0)	0.447
	RLE	69.5 (1.7)	
Group 2 at follow up	Clinic	69.5 (1.5)	0.611
	RLE	68.5 (0.9)	
All data combined	Clinic	71.2 (1.6)	0.268
	RLE	69.2 (0.5)	

Group 1- LSVT LOUD® and VF; Group 2 - VF only

Abbreviation: RLE, Real life environment; VF, Vibrotactile feedback

In this study, VF did not result in improvement in vocal intensity at the end of treatment and at follow-up. This finding is similar to another study where tactile biofeedback did not improve vocal intensity in an IwPD.⁴³ Although our study findings are in contrast with Schalling et al, (2013) which resulted in statistically significant improvement in vocal intensity with tactile biofeedback in IwPD, the improvement was only 1.5 dB which has limited clinical significance.²³ Improvements in vocal intensity and generalization of treatment benefits could be related to the frequency and duration of biofeedback provided. This study was carried out for a total of 4 weeks to be consistent with the LSVT LOUD® protocol while other studies have taken 1-3

weeks.^{23,43} VF was provided for a total of 8 days in this study while other studies in the literature have provided feedback for only for 3-5 days.^{23, 43} Also, the frequency of feedback varied among these studies.^{23,43} In future studies, the frequency of vibration should be tailored to the individual, for example, some IwPD may need more feedback than others. In this study, VF was provided when vocal intensity dropped below the set threshold for 500ms. The delay was set to 500ms based on the recommendation by Gustaffson et al, (2016).⁴³ Despite this, some IwPD in this study felt that the VF was constant. The VL2 does not provide information on number of times the VF feature was activated. To draw a comparison, in another study that used the Ambulatory Phonation Monitor (APM) to provide tactile biofeedback to improve vocal intensity in one IwPD, the APM vibrated 377-1138 times on a single day.⁴³ Such increased frequency of feedback can make it challenging for

TABLE 3.
Comparison of Conversational Vocal Intensity between Treatment Groups in Real-life Environment at Baseline, During Treatment and at Follow-up

Variable	Response	LS Mean (SE) of dB	P-Value
Baseline only	Group 2	71.1 (0.8)	0.134
	Group 1	68.8 (0.9)	
Week 1 only	Group 2	71.2 (1.1)	0.086
	Group 1	67.5 (1.4)	
Week 2 only	Group 2	71.6 (1.2)	0.320
	Group 1	69.6 (1.4)	
Week 3 only	Group 2	70.3 (2.6)	0.182
	Group 1	63.9 (3.3)	
Week 4 only	Group 2	69.0 (0.8)	0.429
	Group 1	67.9 (1.0)	
Weeks 1-4 only	Group 2	70.5 (0.8)	0.044*
	Group 1	67.2 (1.0)	
Follow up only	Group 2	68.5 (1.1)	0.582
	Group 1	69.5 (1.3)	

* Statistically significant ($P < 0.05$) difference in vocal intensity between groups 1 and 2

Group 1- LSVT LOUD and VF; Group 2 - VF only

TABLE 4.
Comparison of Conversational Vocal Intensity between Baseline and Follow-up for Both Treatment Groups in Clinic and Real-life environments

Variable	Response	LS Mean (SE) of dB	P-Value
Group 2	Baseline (RLE)	71.1 (0.8)	0.134
	Follow-up (RLE)	68.8 (0.9)	
Group 1	Baseline (clinic)	72.1 (2.5)	0.454
	Follow-up (clinic)	69.5 (2.3)	
Group 2	Baseline (RLE)	68.4 (1.3)	0.400
	Follow-up (RLE)	70.3 (1.3)	
Group 1	Baseline (clinic)	71.0 (3.0)	0.712
	Follow-up (clinic)	72.7 (3.0)	

Group 1- LSVT LOUD and VF; Group 2 - VF only

Abbreviation: RLE, Real life environment; VF, Vibrotactile Feedback

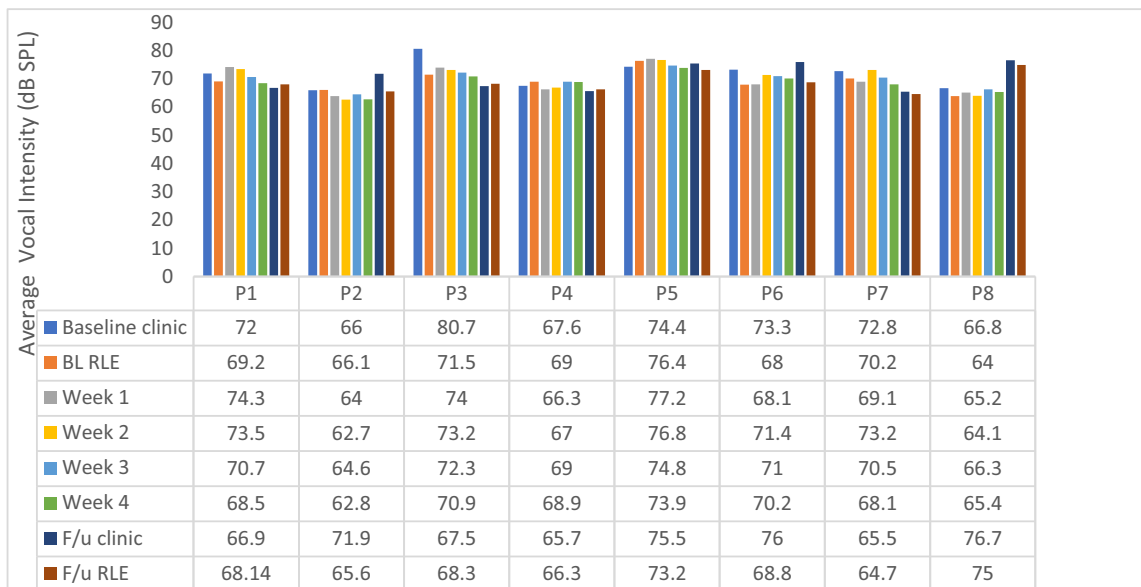


FIGURE 2. Average vocal intensity as measured by Vocalog2 in clinic and real-life environment (RLE) for each participant.

IwPD to facilitate motor learning of a new behavior. Future technology should allow for individual control over when to activate the VF as all situations may not require the use of a loud voice. Also, tailoring the frequency and duration of VF to the individual may be more meaningful. IwPD can become dependent on sensory cues with constant feedback and find it hard to achieve automatization.⁴⁷ The VL2 does not measure environmental noise and hence the effect of background noise on vocal intensity could not be determined in this study.

Methodological considerations and limitations: Results of this study must be interpreted with caution due to the small sample size and disproportionate number of participants in each treatment group. In addition, all the participants in Group 2 had MCI and those in Group 1 did not. Although, prevalence of MCI is common in PD without dementia⁵⁷, the MCI could have influenced how the IwPD paid attention to the VF. Hypophonia was rated by the clinician on the CAPE-V to be mild or mild-moderate in severity for all participants except two of them. However, self-perception of voice problem on the VHI was rated as “severe” by 5 participants, “moderate” by 2 participants and “normal” by one participant. It is important to include IwPD with varying severity of hypophonia as the motivation and “room for improvement” may vary in treatment. The average baseline vocal intensity of our participants was 71.7 dB in clinic and 69.3 dB in real-life. This is close to the normal limit of about 70 dB for connected speech.⁵⁸ These close to normal values may have contributed to a lack of significant difference in the results in addition to the small sample size. The baseline vocal intensity values in this study are consistent with literature.^{23,36,37} Also, all study participants were referred to the Speech Pathology clinic as they complained of hypophonia to the Neurologist.

Device-related limitations: All eight participants wore the VL2 for four days at least each week as instructed. Some participants wore the monitor for longer than four days on certain weeks or longer than four hours on a given day since they felt that they do not have to strictly monitor the hours. In that case, data from the first four days of each week and first four hours of each day were analyzed. Just like any technology, the VL2 had its fair share of challenges. If technical malfunction occurred such as the device did not vibrate or data was not registered, the registration period was prolonged within each week. Although the monitor was worn for four hours, the vocal activity varied for each subject and during each day within a subject. The VL2 monitor picks up the voiced sounds and records it as vocal activity. It is impossible to control the amount of vocal activity for everyone. Another limitation is the design of the vocal monitor. The vocal monitor of the VL2 system is a neckband. Although the neckband was adjustable and came in various sizes and was fitted to the IwPD, participants provided feedback that it did move at times and that they had to adjust it. Also, the vibratory response of the monitor must be improvised since participants felt that it vibrated occasionally for non-vocal sounds.

Communicative environment also could influence results and needs to be further investigated. In the clinic environment, only two people were present (the clinician and the IwPD). However, in real-life environments, the number of communication partners present varies, the distance between the communication partners varies, and as a result the motivation of IwPD to speak louder may vary. Medication ON-OFF effects were also not monitored. We did not control for these factors since the goal was to capture speaking in a natural environment over a period. These factors, however, should be explored in further studies with larger

sample sizes to further understand the effect of environment on conversational demands in IwPD.

CONCLUSION

This study investigated the effects of hypophonia treatment on conversational vocal intensity of IwPD in real-life environments and compared vocal intensity between the clinic and real-life environment. While no significant differences in vocal intensity were discovered between the two environments and therapy approaches, the findings imply that clinicians use 20 minutes of dialogue in clinic to get a good representation of patients' vocal intensity in the real world. With advancing technology, the nature of vibratory feedback on motor-learning of a new behavior such as speaking loudly needs further investigation. Large scale studies including a placebo device with a variety of wearable devices are needed to assess carry over of hypophonia treatment to real-life and to determine the most appropriate device for a patient. Monitoring the conversational vocal intensity of IwPD in real-life environments is a growing area of interest and further studies investigating a standard methodology for functional speech assessment and monitoring treatment progress are warranted.

ACKNOWLEDGMENTS

This study was supported by a Harris grant to Henry Ford Health System and the authors would like to thank Mr. Mort Harris for his generous financial support.

The authors wish to thank the individuals with Parkinson's disease that participated in the study. We wish to acknowledge Cliff Griffin and Mark Robertson from Griffin Laboratories for their technical support with the Vocalog2 device.

REFERENCES

- Marras C, Beck JC, Bower JH, et al. Prevalence of parkinson's disease across north america. *NPJ Parkinsons Dis*. 2018;4:21.
- Duffy JR. *Motor speech disorders: substrates, differential diagnosis, and management*. 3rd Edn St. Louis, MO: Elsevier Mosby; 2013:6.
- Liotti M, Ramig LO, Vogel D, et al. Hypophonia in Parkinson's disease: neural correlates of voice treatment revealed by PET. *Neurology*. 2003;60:432–440.
- Pinto S, Thobois S, Costes N, et al. Subthalamic nucleus stimulation and dysarthria in Parkinson's disease: a PET study. *Brain*. 2004;127:602–615.
- Darley FL, Aronson AE, Brown JR. Clusters of deviant speech dimensions in the dysarthrias. *J Speech Hear Res*. 1969;12:462–496.
- Logemann JA, Fisher HB, Boshes B, et al. Frequency and cooccurrence of vocal tract dysfunctions in the speech of a large sample of Parkinson patients. *J Speech Hear Disord*. 1978;43:47–57.
- Atkinson-Clement C, Sadat J, Pinto S. Behavioral treatments for speech in Parkinson's disease: meta-analyses and review of the literature. *Neurodegener Dis Manag*. 2015;5:233–248.
- Darley FL, Aronson AE, Brown JR. Differential diagnostic patterns of dysarthria. *J Speech Hear Res*. 1969;12:246–269.
- Darley FL, Aronson AE, Brown JR. Clusters of deviant speech dimensions in the dysarthrias. *J Speech Hear Res*. 1969;12:462–496.
- Fox CM, Ramig LO. Vocal sound pressure level and self-perception of speech and voice in men and women with idiopathic Parkinson disease. *Am J Speech Lang Pathol*. 1997;6:85–94.
- Baumgartner CA, Sapir S, Ramig LO. Voice quality changes following phonatory-respiratory effort treatment (LSVT®) versus respiratory effort treatment for individuals with Parkinson disease. *J Voice*. 2001;15:105–114.
- Midi I, Dogan M, Koseoglu M, et al. Voice abnormalities and their relation with motor dysfunction in Parkinson's disease. *Acta Neurol Scand*. 2008;117:26–34.
- Sewall GK, Jiang J, Ford CN. Clinical evaluation of Parkinson's-related dysphonia. *Laryngoscope*. 2006;116:1740–1744.
- Sapir S. Multiple factors are involved in the dysarthria associated with Parkinson's disease: a review with implications for clinical practice and research. *J Speech Lang Hear Res*. 2014;57:1330–1343.
- Sapir S, Ramig LO, Fox CM. Intensive voice treatment in Parkinson's disease: Lee Silverman voice treatment. *Expert Rev Neurother*. 2011;11:815–830.
- Ho AK, Bradshaw JL, Iansek R. Volume perception in parkinsonian speech. *Mov Disord*. 2000;15:1125–1131.
- Kwan LC, Whitehill TL. Perception of speech by individuals with Parkinson's disease: a review. *Parkinson's Disease*. 2011;2011.
- Mollaei F, Shiller DM, Gracco VL. Sensorimotor adaptation of speech in Parkinson's disease. *Mov Disord*. 2013;28:1668–1674.
- Arnold C, Gehrig J, Gispert S, et al. Pathomechanisms and compensatory efforts related to Parkinsonian speech. *Neuroimage Clin*. 2014;4:82–97.
- Liu H, Wang EQ, Metman LV, et al. Vocal responses to perturbations in voice auditory feedback in individuals with Parkinson's disease. *PLoS one*. 2012;7:e33629.
- Houde JF, Nagarajan S, Heinks T, et al. The effect of voice therapy on feedback control in Parkinsonian speech. *Movement Disorders*. 19. Div John Wiley & Sons Inc, 111 River St, Hoboken, NJ 07030 USA: Wiley-Liss; 2004. S403-S403.
- Ramig LO, Countryman S, Thompson LL, et al. Comparison of two forms of intensive speech treatment for Parkinson disease. *J Speech Lang Hear Res*. 1995;38:1232–1251.
- Schalling E, Gustafsson J, Ternström S, et al. Effects of tactile biofeedback by a portable voice accumulator on voice sound level in speakers with Parkinson's disease. *J Voice*. 2013;27:729–737.
- Donovan NJ, Boudreaux DM, Savage MC. Objective measurement of vocal parameters in older people with and without parkinson's disease in their natural environments: a pilot study. *Contemp Issues Commun Sci Disord*. 2015;42:100–109.
- Gustafsson JK, Södersten M, Ternström S, et al. Voice use in daily life studied with a portable voice accumulator in individuals with parkinson's disease and matched healthy controls. *J Speech Lang Hear Res*. 2019;62:4324–4334.
- Rountrey CE. *Speech function in persons with parkinson's disease: effects of environment, task and treatment*. Virginia, USA: James Madison University; 2015... Unpublished doctoral dissertation Retrieved from: <https://commons.lib.jmu.edu/cgi/viewcontent.cgi?article=1038&context=diss201019>.
- Searl and Dietch. *In-clinic vs. at-home voice intensity estimates in parkinson disease*. SanDiego, CA, USA: Presented at the American Speech and Language Hearing Association convention; 2011.
- Bottalico P, Ipsaro Passione I, Astolfi A, et al. Accuracy of the quantities measured by four vocal dosimeters and its uncertainty. *J Acoust Soc Am*. 2018;143:1591–1602.
- Searl J, Dietsch A. Testing of the VocaLog vocal monitor. *J Voice*. 2014;28:523–e27.
- Searl J, Dietsch AM. Tolerance of the vocalog™ vocal monitor by healthy persons and individuals with parkinson disease. *J Voice*. 2015;29:518–e13.
- Van Stan JH, Gustafsson J, Schalling E, et al. Direct comparison of three commercially available devices for voice ambulatory monitoring and biofeedback. *Perspectives Voice Voice Disord*. 2014;24:80–86.
- Miller N, Noble E, Jones D, et al. Life with communication changes in Parkinson's disease. *Age ageing*. 2006;35:235–239.

33. Schulz GM, Grant MK. Effects of speech therapy and pharmacologic and surgical treatments on voice and speech in Parkinson's disease: a review of the literature. *J Commun Disord.* 2000;33:59–88.
34. Fox CM, Morrison CE, Ramig LO, et al. Current perspectives on the lee silverman voice treatment (LSVT) for individuals with idiopathic parkinson disease. *Am J Speech Lang Pathol.* 2002;11:111–123.
35. Ramig LO, Countryman S, O'Brien C, et al. Intensive speech treatment for patients with Parkinson's disease: short-and long-term comparison of two techniques. *Neurology.* 1996;47:1496–1504.
36. 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.
37. Ramig L, Halpern A, Spielman J, et al. Speech treatment in Parkinson's disease: Randomized controlled trial (RCT). *Mov Disord.* 2018;33:1777–1791.
38. Gustafsson J, Södersten M, Ternström S, et al. Long-term effects of lee silverman voice treatment on daily voice use in parkinson's disease as measured with a portable voice accumulator. *Logoped Phoniatr Vocol.* 2018;44(3):1–10.
39. Scott S, Caird FI. Speech therapy for Parkinson's disease. *J Neurol Neurosurg Psychiatry.* 1983;46:140–144.
40. Rubow R, Swift E. A microcomputer-based wearable biofeedback device to improve transfer of treatment in Parkinsonian dysarthria. *J Speech Hear Disord.* 1985;50:178–185.
41. Rubow R. Role of feedback, reinforcement, and compliance on training and transfer in biofeedback-based rehabilitation of motor speech disorders. *The dysarthrias: Physiology, acoustics, perception, management.* 1984;58(3):207–230.
42. Van Stan JH, Mehta DD, Hillman RE. The effect of voice ambulatory biofeedback on the daily performance and retention of a modified vocal motor behavior in participants with normal voices. *J Speech Lang Hear Res.* 2015;58:713–721.
43. Swaminathan S. *Role of Therapeutic Devices in Enhancing Speech Intelligibility and Vocal Intensity in an Individual with Parkinson's Disease.* Johnson City, Tennessee, USA: East Tennessee State University; 2012... Unpublished thesis Retrieved from: <https://dc.etsu.edu/cgi/viewcontent.cgi?article=2668&context=etd>.
44. Gustafsson J, Ternström S, Södersten M, et al. Motor-learning-based adjustment of ambulatory feedback on vocal loudness for patients with Parkinson's disease. *J Voice.* 2016;30:407–415.
45. Schmidt RA, Lee TD. *Motor control and learning: A behavioral emphasis.* 5th ed. Champagne, IL: Human kinetics; 2011.
46. Van Stan JH, Mehta DD, Sternad D, et al. Ambulatory voice biofeedback: Relative frequency and summary feedback effects on performance and retention of reduced vocal intensity in the daily lives of participants with normal voices. *J Speech Lang Hear Res.* 2017;60:853–864.
47. Nieuwboer A, Rochester L, Müncks L, et al. Motor learning in Parkinson's disease: limitations and potential for rehabilitation. *Parkinsonism relat disord.* 2009;15:S53–S58.
48. Zraick RI, Kempster GB, Connor NP, et al. Establishing validity of the consensus auditory-perceptual evaluation of voice (CAPE-V). *Am J Speech Lang Pathol.* 2011;20:14–22.
49. Jacobson BH, Johnson A, Grywalski C, et al. The voice handicap index (VHI) development and validation. *Am J Speech Lang Pathol.* 1997;6:66–70.
50. Szcześniak D, Rymaszewska J. The usefulness of the SLUMS test for diagnosis of mild cognitive impairment and dementia. *Psychiatr Pol.* 2016;50:457–472.
51. Mehta DD, Listfield RW, Cheyne II HA, et al. *Duration of ambulatory monitoring needed to accurately estimate voice use.* September. Portland, OR: Paper presented at the Interspeech conference; 2012... Retrieved from: <http://web.mit.edu/dmehta/www/docs/MehtaInterspeech2012%20Duration%20of%20ambulatory%20monitoring%20needed%20to%20accurately%20estimate%20voice%20use.pdf>.
52. Franke RH, Kaul JD. The hawthorne experiments: first statistical interpretation. *Am Sociol Rev.* 1978;43(5):623–643.
53. Hauser CW. *Use of ambulatory biofeedback to supplement Lee Silverman voice treatment in patients with idiopathic Parkinson's disease.* Boston, USA: MGH Institute of Health Professions; 2005. Unpublished thesis Available from ProQuest Dissertations & Theses Global Database.
54. Ramig L, 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;71:493–498.
55. Spielman J, Ramig L, Mahler L, et al. Effects of an extended version of the lee silverman voice treatment on voice and speech in parkinson's disease. *Am J Speech Lang Pathol.* 2007;16:95–107.
56. Wight S, Miller N. Lee silverman voice treatment for people with parkinson's: audit of outcomes in a routine clinic. *Int J Lang Commun Disord.* 2015;50:215–225.
57. Litvan I, Aarsland D, Adler CH, et al. MDS task force on mild cognitive impairment in parkinson's disease: critical review of PD-MCI. *Mov Disord.* 2011;26:1814–1824.
58. Baken RJ, Orlikoff RF. *Clinical measurement of speech and voice.* 2nd Edn. Cengage Learning; 2000:108–109.