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Increased vocal intensity due to the Lombard effect in speakers with Parkinson's disease: Simultaneous laryngeal and respiratory strategies

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ABSTRACT

Purpose: The objective of the present study was to investigate whether speakers with hypophonia, secondary to Parkinson's disease (PD), would increases their vocal intensity when speaking in a noisy environment (Lombard effect). The other objective was to examine the underlying laryngeal and respiratory strategies used to increase vocal intensity.

Methods: Thirty-three participants with PD were included for study. Each participant was fitted with the SpeechVive™ device that played multi-talker babble noise into one ear during speech. Using acoustic, aerodynamic and respiratory kinematic techniques, the simultaneous laryngeal and respiratory mechanisms used to regulate vocal intensity were examined.

Results: Significant group results showed that most speakers with PD (26/33) were successful at increasing their vocal intensity when speaking in the condition of multi-talker babble noise. They were able to support their increased vocal intensity and subglottal pressure with combined strategies from both the laryngeal and respiratory mechanisms. Individual speaker analysis indicated that the particular laryngeal and respiratory interactions differed among speakers.

Conclusions: The SpeechVive™ device elicited higher vocal intensities from patients with PD. Speakers used different combinations of laryngeal and respiratory physiologic mechanisms to increase vocal intensity, thus suggesting that disease process does not uniformly affect the speech subsystems.

Learning outcomes: Readers will be able to: (1) identify speech characteristics of people with Parkinson's disease (PD), (2) identify typical respiratory strategies for increasing sound pressure level (SPL), (3) identify typical laryngeal strategies for increasing SPL, (4) define the Lombard effect.

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1. Introduction

Speech production is continuously modulated during everyday activities to meet the needs of the communicative environment, such as personal conversations, speaking to a small group, and conversing in noisy environments. When confronted with these diverse tasks, speakers must be able to adjust and control their vocal intensity.

Typical speakers change their breathing patterns in order to more efficiently produce the subglottal air pressures (P_s) needed to increase vocal intensity (Finnegan, Luschei, & Hoffman, 2000; Huber, 2007; Huber, Chandrasekaran, & Wolstencroft, 2005; Winkworth & Davis, 1997). In healthy individuals, combined passive and active forces are used to finely control P_s to regulate vocal intensity (Stathopoulos & Sapienza, 1997). Specifically, typical speakers expand the thorax (inhale to higher lung and rib cage volumes) to take advantage of greater elastic recoil forces, thereby limiting the amount of thoracic muscle contraction needed to increase P_s (Hixon, Goldman, & Mead, 1973; Huber, 2007). Further, typical speakers may increase their abdominal muscle contraction, as seen by smaller abdominal volumes during loud speech, to help maintain thoracic resistance and alveolar pressures (Hixon et al., 1973; Hoit, Plassman, Lansing, & Hixon, 1988), but this has not been reported consistently (Huber, 2007; Stathopoulos & Sapienza, 1997). Hereafter, the use of higher lung and rib cage volume ranges and potentially smaller abdominal volumes during increased vocal intensity is considered to be "improved respiratory support."

The pathophysiology of Parkinson's disease (PD) substantially affects the function of motor systems in the body (Wichmann & DeLong, 1996). In addition to the limb motor system, the disease process affects the respiratory motor system. It has been demonstrated that both inspiratory and expiratory muscle impairments in patients with PD, more specifically decreased strength or poor coordination of respiratory muscles (de Bruin, de Bruin, Lees, & Pride, 1993; Haas, Trew, & Castle, 2004), may contribute to decreased expiratory flows (Bogaard, Hovestadt, Meerwaldt, Meche, & Stigt, 1989). The speech and voice deficits commonly reported in PD can also be attributed to deficits in neuromuscular function (Darley, Aronson, & Brown, 1969; Duffy, 2005). For example, muscular rigidity, a hallmark symptom of PD, has been suggested to contribute to the observed reduction in pulmonary function (Solomon & Hixon, 1993). These authors suggest that a breakdown in the synergistic force of the muscles of the rib cage and abdomen could contribute to inefficient breathing patterns during the speech production of individuals with PD. For example, individuals with PD have shown evidence of oppositional movement of rib cage and abdomen during expiration (Solomon & Hixon, 1993), and more variable lung volumes than healthy speakers (Huber, Stathopoulos, Ramig, & Lancaster, 2003). In addition, speakers with PD often have more difficulty controlling their vocal intensity (Sadagopan & Huber, 2007), and also planning in advance to support longer utterances (Bunton, 2005), particularly during extemporaneous speech (Huber & Darling, 2011). In summary, due to muscle rigidity, reduced muscle strength, and difficulties coordinating respiratory movements when speaking, individuals with PD are likely to be challenged when responding to everyday communicative demands, in particular increasing vocal intensity.

While the respiratory system has been identified as an important contributor to increased vocal intensity, it does not act in isolation. The laryngeal mechanism also plays an important role in adjustments of intensity. Laryngeal strategies to increase vocal intensity are achieved through changes to glottal shape which in turn affects the resistance to glottal airflow (Isshiki, 1964). Changes to glottal shape can be observed by utilizing glottal airflow measures (Holmberg, Hillman, & Perkell, 1988; Stathopoulos & Sapienza, 1997). Using a noninvasive circumferentially-vented pneumotachograph mask, the oral airflow (V_o) waveform during speech can be inverse filtered to obtain a derived glottal airflow waveform (Rothenberg, 1973). During typical speech production by a broad age range of males and females, glottal aerodynamic events were shown to change with increased vocal intensity (Stathopoulos & Sapienza, 1997). Specifically, open quotient (OQ) decreased reflecting that the vocal folds were in a closed state for a greater part of the entire cycle. Peak-to-peak glottal airflow increased, reflecting that the amplitude of vocal fold vibration increased. Minimum glottal airflow decreased during the "closed" part of the cycle, indicating that there was increased vocal fold adduction. Last, maximum flow declination rate (MFDR), which reflects speed of vocal fold closure, increased at higher vocal intensity levels (Stathopoulos & Sapienza, 1997). These changes in laryngeal function help to achieve greater levels of P_s thereby improving laryngeal "support" at higher vocal intensity levels. In summary, adjustments in vocal intensity are regulated by simultaneous changes to both the respiratory and laryngeal systems (Stathopoulos & Sapienza, 1997).

Laryngeal muscle function during speech and non-speech activities is also affected by the pathophysiology of PD. For example, a recent EMG study showed that during rest and vocalization, 73% of participants with PD presented with increased laryngeal muscle activity, thus possibly reflecting hypertonicity and muscle rigidity of the laryngeal musculature (Zarzur, Duprat, Shinzato, & Eckley, 2007). Increased levels of laryngeal muscle activity have also been associated with vocal fold bowing (Gallena, Smith, Zeffiro, & Ludlow, 2001).

Differences in glottal shape offer insight into problems with motor function of the laryngeal muscles. Videostroboscopic studies have confirmed the presence of vocal fold bowing, increased glottal opening over the entire period of vibration, abnormal phase closure, and asymmetric vibratory patterns during phonation by individuals with PD (Dromey, Ramig, & Johnson, 1995; Hanson, Gerratt, & Ward, 1984; Perez, Ramig, Smith, & Dromey, 1996; Smith, Ramig, Dromey, Perez, & Samandari, 1995). These changes to the laryngeal mechanism are likely to impact the ability of individuals with PD to increase the vocal fold resistance and adduction forces necessary for producing high intensity speech.

Despite problems with muscle function, some patients with PD increased vocal intensity after voice treatment (LSVT®), showing improvements in the speech mechanism. In a single subject study of one male with PD, acoustic and aerodynamic data indicated increased vocal intensity and improved laryngeal function following one month of treatment (LSVT®)