The Laryngoscope

"Lombard Effect" and Voice Changes in Adductor Laryngeal Dystonia: A Pilot Study

Maria Raffaella Marchese, MD, PhD; Ylenia Longobardi, SPL, MSc; Rosa Libero, MD; Gamze Yesilli-Puzella, SPL, PhD; Lucia D'Alatri, MD; Jacopo Galli, MD

Objectives: The aim was to describe the acoustic, auditory-perceptive, and subjective voice changes under the Lombard effect (LE) in adductor laryngeal dystonia (AdLD) patients.

Methods: Subjective perception of vocal effort (OMNI Vocal Effort Scale OMNI-VES), Maximum Phonation Time (MPT), and the perceptual severity of dysphonia (GRBAS scale) were assessed in condition of stillness and under LE in 10 AdLD patients and in 10 patients with typical voice. Speakers were asked to produce the sustained vowel /a/ and to read a phonetically balanced text aloud. Using the PRAAT software, the following acoustic parameters were analyzed: Mean Pitch (Hz), Minimum and Maximum Intensity (dB), the Fraction of Locally Unvoiced Frames, the Number of Voice Breaks, the Degree of Voice Breaks (%), the Cepstral Peak Prominence-Smoothed (CPPS) (dB).

Results: Under LE, the AdLD group showed a decrease of both G and S parameters of GRBAS and subjective effort, mean MPT increased significantly; in the controls there were no significant changes. In both groups under LE, pitch and intensity of the sustained vowel /a/ significantly increased consistently with LE. In the AdLD group the mean gain of OMNI-VES score and the mean gain of each parameter of the speech analysis were significantly greater than the controls' ones.

Conclusion: Auditory feedback deprivation obtained under LE improves subjective, perceptual-auditory, and acoustics parameters of AdLD patients. These findings encourage further research to provide new knowledge into the role of the auditory system in the pathogenesis of AdLD and to develop new therapeutic strategies.

Key Words: acoustic analysis, adductor laryngeal dystonia, audio-vocal feedback control, Lombard effect.

Level of Evidence: 4

***Laryngoscope*, 00:1–7, 2024**

INTRODUCTION

Adductor laryngeal dystonia (AdLD) is a focal task-specific dystonia characterized by irregular and uncontrolled voice breaks. AdLD is a rare disorder with an estimated prevalence of one in 100,000.[1] Over 65% of those affected are women and the average age of symptom onset is 45 years.[2,3] AdLD mainly results in strained-strangled and harsh voice quality with spasms and effortful speech production.[4]

The severity of the condition varies significantly depending on the speaker, with symptoms ranging from mild and barely noticeable to severe. Diagnosis can be challenging because few objective measures are clinically used to discriminate LD from other voice disorders. It is based on qualitative and phenomenological assessments carried out through the combined assessment of voice care specialists such as laryngologists or speech-language pathologists and neurologists with experience specifically in movement disorders. The pathogenesis is still unknown. To date, we know that it is a movement disorder: specifically, it is a functional and structural disorder of the neural network of the motor and sensory systems.[5]

Sensory system abnormalities have been described in many types of adult-onset focal dystonia. The sensory system is involved in the mechanisms underlying two clinical phenomena of LD: sore throat, discomfort and sensory tricks.[6] Recent studies have demonstrated that an abnormal sensory discriminatory process could be an endophenotypic marker of isolated dystonia.[7] The temporal threshold of visual and proprioceptive discrimination is increased in LD except for singer's LD.[7] On the other hand, it has been shown that the spatial-tactile discrimination and olfactory processing are normal in LD.[7] The interdependence between speech, voice, and hearing is well known.[8,9]

Indeed, the role of the auditory sensory domain in AdLD is emerging. Functional neuroimaging studies of speech production in speakers with AdLD have reported

From the Unità Operativa Complessa di Otorinolaringoiatria, Dipartimento di Neuroscienze, Organi di Senso e Torace (M.R.M., Y.L., R.L., L.D., J.G.), Fondazione Policlinico Universitario A. Gemelli IRCCS, Rome, Italy; School of Health Sciences, Speech and Language Therapy Department (G.Y.-P.), Cappadocia University, Ürgüp/Nevşehir, Turkey; and the Sezione di Otorinolaringoiatria, Dipartimento Universitario Testa-Collo e Organi di Senso (L.D., J.G.), Università Cattolica del Sacro Cuore, Rome, Italy.

Editor's Note: This Manuscript was accepted for publication on April 17, 2024.

The authors have no funding, financial relationships, or conflicts of interest to disclose.

Send correspondence to Maria Raffaella Marchese, Department of Neurosciences, Sensory Organs and Thorax, Otorhinolaryngology Unit, Fondazione Policlinico Universitario A. Gemelli IRCCS L.go "A. Gemelli", 8 – 00168 Rome, Italy. Email: mariaraffaella.marchese@policlinicogemelli.it

DOI: 10.1002/lary.31491

hyperactivation of the laryngeal and orofacial sensorimotor cortex, including premotor and motor regions, as well as auditory and somatosensory cortices that may interfere with typical sensorimotor processes.[10] Daliri et al.[11] demonstrated that the same pattern of hyperactivity was found even when auditory feedback control of speech was eliminated through noise masking, suggesting that the hyperactivation in the voice production network observed in speakers with AdLD is likely related to impairments in somatosensory feedback control and/or in the feedforward control mechanism.

The role of audio-vocal feedback control in LD is of particular interest. Reflexive vocal behavior has been shown to be dysfunctional in a variety of disorders affecting both voice and speech, including vocal fold paralysis, Parkinson's disease, and cerebellar degeneration.[8–10,12–14]

Thomas et al.[15] showed that patients with LD exhibited increased sensitivity to altered audio-vocal feedback during sustained phonation (hyperactive pitch-shift reflex, approximately twice as high as typical) likely due to the alterations in central processing of sensory feedback or a change in sensorimotor coordination. The poor voice quality in AdLD could activate an auditory feedback overcontrol mechanism in order to correct the aberrant voice signal. Nevertheless, a dysfunction in self-monitoring of auditory feedback could contribute to the excessive muscle activation characteristic of the disorder.[15]

Another form of audio-vocal feedback control is the Lombard effect (LE). LE refers to the vocal adjustments that occur in typical speakers in response to background noise.[16–18] It is possible that the LE could be an innate or unlearned motor behavior controlled by a subcortical network that can be modulated by cortical brain areas.[19–22]

The acoustic components of "*Lombard speech*" differ from those of *neutral speech* (i.e., flatter spectrum with emphasis on high frequencies, longer duration of target phonemes, reduced speech speed, and formant frequency adjustments).[23–25] Due to LE, also, speakers increase their voice volume in the presence of loud noise as part of a compensatory mechanism.[26] This effect has also been demonstrated in people with Parkinson's disease[27,28] and in people who stutter whose disorder may result from abnormal processing of feedback.[29,30] Furthermore, the effect of Lombard test has been investigated in patients with hyperkinetic dysphonia compared with typical speakers showing similar change in aerodynamic and acoustic features and, in presence of background noise, a return to the baseline features only in typical speakers.[31] To date, findings about the audio-vocal feedback control provide interesting new insights into the pathophysiology of AdLD that reinforce the role of hearing in maintaining vocal control.

To date, botulinum neurotoxin is the gold standard treatment for AdLD due to the peripheral effect of temporary chemical denervation of vocal fold muscles and central modulation of sensory inputs.[13,32–35]

Nevertheless, in cases that respond to treatment, 51% of patients experience prolonged side effects that often interfere with breathing and swallowing.[36] The effectiveness of the treatment may gradually decrease over time as some patients develop resistance to BoNT and the injections are psychologically and financially burdensome because they are expensive and repeated injections are needed every 3 months, on average. The aforementioned findings relating to the auditory system may open up new potential therapeutic opportunities as has already happened for other movement disorders associated with speech impairment.

Overall, studies on this topic are scarce. The aim of this article was to describe the acoustic, auditory-perceptual, and subjective voice changes in patients with AdLD and in subject with typical voice as a control group, during rapid Lombard Test (LT) and to discuss the pathogenic hypotheses underlying our findings.

MATERIALS AND METHODS

The study was conducted with a prospective observational design on patients diagnosed with AdLD at Phoniatric Unit of the Fondazione Policlinico Universitario A. Gemelli IRCCS, Rome, Italy. AdLD was diagnosed by a specialized otolaryngologist (MRM or LD), expert in voice disorders, after voice evaluation, videolaryngostroboscopy, and audiometric examination. Additionally, patients with AdLD underwent neurological examination by a neurologist specializing in movement disorders.

Patients who met the inclusion/exclusion criteria were eligible for the study. The inclusion criteria were: Italian native language, hearing threshold <20 dB HL for frequencies from 0.5 to 4 KHz, age between 18 and 65 years. Exclusion criteria included: less than 3 months since last botulinum toxin injection, previous laryngeal surgery, ongoing speech therapy, ongoing dopaminergic therapy, inability to sustain phonation of sufficient duration (>3 s), or perform sufficient rehearsal to measure vocal outcomes. The research was conducted ethically in accordance with the Principles for Medical Research Involving Human Participants, and its later amendments or comparable ethical standards, contained in the Declaration of Helsinki developed by the World Medical Association (WMA) in 1964. Written informed consent was obtained from all participants enrolled in the study.

Control Group

Individuals of both sexes, aged between 18 and 65 years and with a typical voice, underwent an anamnestic interview by an otolaryngologist to exclude any cognitive, speech, voice, and neurological problems known at the time of the testing and an audiometric examination to exclude hearing problems.

Sound Signal Capture

For each speaker, we collected both sustained emission of the vowel /a/ and the connected speech by asking them to read a phonetically balanced text, in quiet and in noisy surroudings. Specifically, speakers were instructed to sustain the vowel /a/ for at least 4 s at a comfortable pitch and loudness and to read, at a conversational volume, a standardized phonetically balanced text ("Il Deserto"[37]). The duration of the reading was about 1 min. For the LT, each subject was exposed to speech white noise (SWN)[38,39] through worn open-air headphones. Noise intensity was 80 dB SPL.[40,41] Speech samples were recorded using a Samson Meteor microphone (Samson Technologies Corp, Hauppauge, New York) that was digitized at a sampling rate of 44.1 kHz with a resolution of 16 bits using the PRAAT software (version 6.2.01—November 17, 2021; Boersma P, Weenink D). Voice recordings occurred in a quiet room with ambient noise less than 35 dB SPL. The microphone was positioned at a distance of 10 cm from the lips at an angle of 45°–90° to the front of the mouth. The speech samples were saved in WAV format for later analysis.

Aerodynamics Measures, Auditory-Perceptual Assessment, and Acoustic Analysis

The maximum phonation time (MPT) was obtained by asking the patient to sustain the vowel /a/ as long as possible on a single breath. The longest of three attempts was calculated as the MPT (seconds).[42]

From both the middle 3 s of the sustained vowel /a/ and the speech sample, we obtained: Mean Pitch (Hz), Minimum and Maximum Intensity (dB SPL). Moreover, from each speech sample we extracted the following acoustics parameters reported in the Voice Report automatically calculated by the PRAAT software: the Fraction of Locally Unvoiced Frames (fraction of pitch frames analyzed as unvoiced), the Number of Voice Breaks (all inter-pulses intervals longer than 16.6667 ms), and the Degree of Voice Breaks (%) (the total duration of the breaks between the voiced parts divided by the total duration of the analyzed part of the signal[43–46]). Finally, we calculated the Cepstral Peak Prominence-Smoothed (CPPS) (dB) value from the most stable 3 s of the vocalization and from the initial 48 syllables of the reading task using the PRAAT script described by Maryn et al.[47] CPP is an acoustic measure of speech quality which represents the distance between the first rahmonic peak and the point with equal quefrency on the regression line through the smoothed cepstrum, where a cepstrum is is the Fourier Transform of the logarithm of the signal's power spectrum.[47] The more periodic a voice signal, the more well-defined the harmonic configuration in the spectrum (i.e., the more harmonic the spectrum), and, consequently, the more the cepstral peak will be prominent. Subsequently, the CPP signal was smoothed by averaging the cepstral magnitude across frequencies and time.[47] The smoothed measure of CPP referred to as the CPPS is strongly correlated with the overall dysphonia severity.[48] In our analyses, speech signals were broken into "voiced" and "voiceless" segments and CPPS values were derived only for the "voiced" periods.

Blind perceptual evaluation, using the Grade—Roughness—Breathiness—Asthenia—Strain (GRBAS) scale[49] was performed on recorded voice samples (reading task of the aforementioned text) by two speech therapists who were not involved in patients' voice care. Each recorded speech sample was made anonymous and distributed randomly to the raters.

The MPT, the perceptual evaluation (GRBAS scale), and the acoustic analysis were obtained in quiet condition and during LT (SWN at 80 dB SPL delivered through worn open-air headphones).

Self-Perceptual Assessment

All speakers were asked to fill the OMNI Vocal Effort Scale (OMNI-VES),[50] which is a validated tool for rating self-perceived voice-related exertion in people with AdLD. The OMNI-VES is a pictorial description of the scale to accompany the 0 to 10 equal intervals of gradual increase in vocal effort. The OMNI-VES was administered in quiet condition and during LT.

Gain

For Fraction of Locally Unvoiced Frames, Number of Voice Breaks, Degree of Voice Breaks, and OMNI-VES score we calculated the gain as the difference between each parameter obtained under quiet condition and during the Lombard test.

Statistical Analysis

Data were collected and analyzed using Excel® 2016 (Microsoft, Redmond, WA, USA). Statistical analysis was performed using XLSTAT software version 2021.1. Continuously distributed outcomes were summarized as the mean plus or minus (±) one standard deviation (SD) and categorical outcomes with frequencies and percentages. Quantitative data were compared using *t*-tests. The Chi-squared test was used for categorical variables. The significance level was set at 0.05.

RESULTS

Among 12 AdLD patients that met the inclusion criteria, two (16.7%) were excluded because of the inability to sustain phonation of sufficient duration. Therefore, final sample of patients with AdLD was composed of 10 patients (9 F: 1 M) 56.0 (SD = 10.3) mean years old.

The 10 control participants (9 F: 1 M) had a mean age of 52.1 (SD = 6.9) years. Statistical analyses showed no significant difference in the age between AdLD and control groups ($p > 0.05$).

In the AdLD group, the mean MPT in quiet condition was 6 s (SD = 3.5) and increased significantly to 10 s (SD = 6.4) in noise condition [$t\ (18) = -1.70$, $p = 0.007$]. In the control group the mean MPT was 11 s (SD = 6.9) and did not significantly change in noise [12 seconds (SD = 7.7) – $t\ (18) = -0.39$, $p = 0.22$].

In the AdLD group during the LT, perceptually the voice improved mainly because of the decrease in strain and roughness (Table I). The distribution of responses across scores was significantly different ($p < 0.05$) for the number of speech recordings with an overall severe

TABLE I.
Prevalence of the Scores for Each Parameters of GRBAS Scale Used to Assess the Speech Quality With and Without Auditory Masking in the Adductor Laryngeal Dystonia Group.

	Lombard Effect OFF					Lombard Effect ON				
	G	*R*	*B*	*A*	*S*	*G*	*R*	*B*	*A*	*S*
Score 3	5 (50%)	0 (0%)	0 (0%)	0 (0%)	5 (50%)	1 (10%)	0 (0%)	0 (0%)	0 (0%)	1 (10%)
Score 2	5 (50%)	5 (50%)	0 (0%)	0 (0%)	4 (40%)	4 (40%)	2 (20%)	0 (0%)	0 (0%)	4 (40%)
Score 1	0 (0%)	5 (50%)	0 (0%)	0 (0%)	0 (0%)	5 (50%)*	6 (60%)	0 (0%)	0 (0%)	5 (50%)*
Score 0	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	2 (20%)	0 (0%)	0 (0%)	0 (0%)

*"Masking ON" versus "masking OFF," $p < 0.05$.

dysphonia (G score = 3) and for the ones with severe strain (for both 5 vs. 0 $-\chi^2 = 6.67$, $p = 0.009$). In the control group we included speakers with typical voice, then for all the participants the overall grade of dysphonia and each other parameter of GRBAS was 0 before the LT and did not change during LT.

The results of the acoustic analysis for AdLD and control group are shown in Tables II and III.

During the LT the sustained vowel /a/ was significantly louder and higher pitched in subject with typical voice and in patients with AdLD (Table II). For both groups we observed a significant increase in the mean pitch [t (18)= − 2.56 $p = 0.0065$ (case group); t (18) = − 1.30 $p = 0.0097$ (control group)], minimum intensity [t (18) = −3.47 $p = 0.0049$ (case group); t (18) = − 4.70 $p = 0.000057$ (control group)] and maximum intensity [t (18) = −4 $p = 0.0013$ (case group); t (18) = −4.90 $p = 0.000058$ (control group)]. The mean overall CPPS measure (dB) obtained from the sustained vowel /a/ under LE in the AdLD group was significantly higher than the one recorded in the quiet condition [14.6 (SD = 2.5) vs. 16.9 (SD = 1.9) − t (18) = −2.33 $p = 0.0011$]. In the control group the CPPS did not differ with and without audio vocal feedback [17.3 (SD = 1.1) vs. 18.2 (SD = 1.5) − t (18) = 0.084, $p = 0.45$]. The change of each acoustic parameter observed during vocalism under LE in patients with AdLD did not differ if compared with the gain recorded in subject with typical voice. In the AdLD group the mean gains of each parameter were significantly greater than the ones of the control group (Table III). In both groups the acoustic analysis performed on connected speech during LT showed a significant increase in the mean pitch [t (18) = −3.20 $p = 0.00053$ (case group); t (18) = −3.97 $p = 0.00025$ (control group)], minimum intensity [t (18) = −6 $p = 0.000079$ (case group); t (18) = −2.60 $p = 0.0047$ (control group)] and maximum intensity [t (18) = −4.30 $p = 0.00033$ (case group); t (18) = −5.60 $p = 0.000077$ (control group)]. On the other hand the Fraction of Locally Unvoiced Frames [t (18) = 1.99 $p = 0.0003$ (case group); t (18) = 1.50 $p = 0.014$ (control group)], the Number of Voice Breaks [t (18) = 1.89 $p = 0.001$ (case group); t (18) = 1.28 $p = 0.025$ (control group)], and the Degree of Voice Breaks [(t (18) = 2.14 $p = 0.0003$ (case group); t (18) = 1.49 $p = 0.018$ (control group)] decreased significantly while the mean overall CPPS measure [t (18) = −5.08 $p = 0.00015$ (case group); t (18) = −2.70 $p = 0.000017$ (control group)] was significantly higher than the one recorded in the quiet condition.

The mean OMNI-VES score obtained in the quiet condition was 5.4 (SD = 2.8), and the one during LT was 3.8 (SD = 2.4) with a mean improvement of 1.6 [t (18) = 1.37, $p = 0.00027$]. In the control group no patients complained of effort during the speech before LT and this result did not change during LT. The mean gain of OMNI-VES score calculated in the AdLD was significantly higher than the one recorded in the control group [1.6 (SD = 1.0) vs. 0 (SD = 0) − t (18) = 5.24, $p = 0.00027$].

DISCUSSION

This preliminary study demonstrates that the main acoustic effects of the Lombard test on the voice of patients with AdLD are those expected in subject with typical voice. Subjectively perceived effort and auditory-perceptual severity of dysphonia in AdLD patients under Lombard effect improved significantly. Furthermore, during the Lombard test we obtained a greater improvement in speech acoustic parameters in patients with AdLD compared to the change measured in the control group.

AdLD is more severe in connected speech as rapid transitions between voiced and unvoiced sounds are required.[51] To confirm this, our findings showed that, in the quiet condition, the average CPPS values obtained from the sustained vowel /a/ were higher than those extracted from the connected speech samples.

During the Lombard test we observed in both groups a significantly louder voice with higher pitch. It is known that the increase of loudness and pitch require medial compression of the vocal folds with consequent improvement in glottal closure and signal periodicity, as confirmed by a higher value of CPPS, a higher number of voiced segments and less voiced breaks. The improvement of CPPS in presence of background noise was significant in the AdLD regardless of the voice use. Furthermore, during the speech task the periodicity of the signal improved more in the patients with AdLD compared to subject with typical voice.

TABLE II.
The Mean Values of Acoustic Parameter Related to the Vocalism /a/ Recorded Without Auditory Masking and With Auditory Masking in Both Control and Case Groups.

	Control Group			Case Group		
Vocalism – /a/	Lombard Test OFF	Lombard Test ON	Gain	Lombard Test OFF	Lombard Test ON	Gain
Mean pitch (Hz)	208.4 ± 34.5*	233 ± 49.7	24.6 ± 27.4	199.8 ± 45.6*	244.3 ± 30.6	44.4 ± 45.6
Minimum intensity (dB)	73.1 ± 5.1*,†	82.9 ± 4.1†	9.7 ± 4.7	66.8 ± 6.3*	75.7 ± 5.1	12.8 ± 13
Maximum intensity (dB)	76.0 ± 4.7*,†	85.9 ± 4.2†	9.9 ± 4.8	73.0 ± 4.7*	82.1 ± 5.3	9 ± 6.9
Cepstral peak prominence-smoothed (dB)	17.3 ± 1.1	18.2 ± 1.9	0.06 ± 1.4	14.6 ± 2.5*	16.9 ± 1.9	1.65 ± 1.6

*Masking OFF versus ON in each group = $p < 0.05$.
†Significant difference between control versus case group ($p < 0.05$).

TABLE III.
The Mean Values of Acoustic Parameter Related to the Speech Obtained by Reading a Phonetically Balanced Text Without Auditory Masking and With Auditory Masking in Both Control and Case Groups.

SPEECH—Text Phonetically Balanced	Control Group: Lombard Test OFF	Control Group: Lombard Test ON	Control Group: Gain	Case Group: Lombard Test OFF	Case Group: Lombard Test ON	Case Group: Gain
Mean Pitch (Hz)	185 ± 17.9	218 ± 19.4	34.8 ± 19.7	188.7 ± 22.8	223.1 ± 25.6	35.5 ± 24.3
Minimum intensity (dB)	44.2 ± 2.3	47.7 ± 3.6	3.9 ± 3.3	12.1 ± 5.5	30.9 ± 8.15	18.8 ± 9.6
Maximum intensity (dB)	79.3 ± 2.7	86 ± 2.5	6.6 ± 3.4	77.5 ± 2.7	84.5 ± 4.3	7 ± 4.4
Fraction of locally unvoiced frames	18.9 ± 7.0*,†	14.7 ± 5.3†	−4.18 ± 5†	38.2 ± 10.7*	30.0 ± 7.3	−8.2 ± 5
Number of voice breaks	8.9 ± 2.4*,†	7.5 ± 2.5†	−1.4 ± 1.9†	133.5 ± 36.4*	108.4 ± 21	−25 ± 18.7
Degree of voice breaks (%)	20.7 ± 7.5*,†	16.0 ± 6.3†	−4.62 ± 5.9†	41.7 ± 11.6*	32.2 ± 7.8	−9.5 ± 5.8
Cepstral peak prominence-smoothed (dB)	12.5 ± 1.3*	14.0 ± 1.1	1.48 ± 0.6†	9.61 ± 1*	11.51 ± 0.7	2.3 ± 0.4

*Masking OFF versus ON in each group = $p < 0.05$.
†Difference between control versus case group = $p < 0.05$.

The multiparametric approach we adopted to assess dystonic voices was motivated by the multidimensional nature of voice quality and the fact that it is not related to a single physical variable or a single psychoacoustic determinant.[52] The inclusion of other outcome measures, such as GRBAS, provided an additional marker of the LT effect. In our cases, the overall severity of dysphonia and the vocal effort improved significantly.

Bond et al.[53] observed that Lombard test increases the vocal effort in four speakers with typical voice. Nevertheless, our controls reported no strain during the test. On the contrary, in the AdLD group, the presence of background noise seemed to effectively reduce self-reported vocal effort and similarly decrease the effort perceived by the expert listeners. This result is probably consistent with the typical feature of AdLD described by Ludlow et al.[54] for which the strain decreases at a higher pitch.

In summary, we can first assume that on the one hand an increase in background noise leads to LE (increase in vocal amplitude and pitch) which directly induces changes in acoustic parameters[55] and secondarily leads to changes in AdLD symptoms, as symptoms are reduced when speaking at higher pitches or at greater intensity.[56] Secondly, our results might be related to the alteration of auditory feedback due to the background noise with reducing the (peraphs maladaptive) effects on laryngeal motor control.

Guiry S et al.[57] interestingly demonstrated that in AdLD patients, the least affected types of vocal production were innate vocalizations, voiced and voiceless, including crying, laughing, and yawning. In contrast, learned vocal behavior, (i.e., speech), is selectively affected by the disease. This separation in AdLD symptomatology is explained by selective alterations of neural circuits that control learned but not innate vocal behaviors. One of the main interpretations of the LE is that it is a physiological audio-phonatory reflex.[58] Thanks to compensatory strategies involuntarily evoked by the LE, selective alterations of the neural circuits that control the learned vocal behaviors may be avoided, leading to a decrease in voice breaks during speech.

However, there is currently conflicting evidence regarding the contribution of subcortical and cortical networks to the Lombard effect. There is compelling data to suggest that cortical processes participate with a modulatory role in the LE.[31] Based on the state feedback control model the sensorimotor systems issue commands to activate muscle groups that result in behaviors such as speech production. Because sensory feedback is delayed and the external sensory environment can be noisy, motor control is primarily achieved through forward prediction.[59,60] In our sample, LE had a positive impact on the speech task, which is the one most affected by AdLD, due to the greater difficulty in controlling the laryngeal muscles.[61] The changes associated with Lombard speech observed in the patients affected by LD may suggest intrinsic differences in integrating auditory information with speech motor control with a transient modulation of the sensorimotor integration like it was a "sensory trick."

The mean baseline of MPT obtained in our AdLD group was lower than the typical, possibly due to the lower respiratory pressure employed as a strategy to improve voice quality. The presence of noise significantly improved the MPT duration in the patients with AdLD, suggesting the impact of self-monitoring in voice adjustment based on auditory feedback.

Overall, our results support the role of auditory feedback in the self-adaptation of voice in subjects affected by AdLD. We first observed a multiparametric beneficial effect of Lombard effect on voice quality in patients with AdLD. Previously only McColl et al.[62] studied the reaction of the AdLD speakers to the noise obtaining a reduction of perceptual speech intelligibility. The study included four patients assessed with a VAS scale, but it is possible that the noise affected the listeners. The listeners in our study did not hear the speech noise but only the recorded voice.

Based on our evidence, the research on the role of the auditory system in AdLD is of paramount importance to provide new knowledge on the pathogenesis of the disease that can guide conservative treatment, providing, for example, the basis for defining a specific vocal therapy protocol.

Despite its strengths, the limitation of this study is the small number of patients with AdLD to confirm the results. Moreover, further researches are necessary to compare the effects of alteration of the auditory feedback on different degrees of AdLD.

CONCLUSION

The increase in background noise provided a subjective, perceptual-auditory, and acoustic beneficial effect on voice quality in patients with AdLD, suggesting a possible role of auditory feedback in self-monitoring and voice regulation and prompting inspiration for future research on the pathogenesis of the disease and on new therapeutic perspectives in the rehabilitation field.

ACKNOWLEDGMENT

Open access funding provided by BIBLIOSAN.

BIBLIOGRAPHY

1. Dejonckere PH. Criteres acoustiques de fluence pour l'evaluation desdysphonies spasmodiques. In: C Klein-Dallant (Ed.) *Voix Parleè et Chantè, Paris*; 2007:63-73.
2. Dejonckere PH, Bradley P, Clemente P, et al. A basic protocol for functional assessment of voice pathology, especially for investigating the efficacy of (phonosurgical) treatments and evaluating new assessments techniques. Guideline elaborated by the committee on phoniatrics of the European laryngological society (ELS). *Eur Arch Otorhinolaryngol*. 2001;258:77-82.
3. Hogikyan ND, Wodchis WP, Spak C, Kileny PR. Longitudinal effects of botulinum toxin injections on voice-related quality of life (V-RQOL) for patients with adductory spasmodic dysphonia. *J Voice*. 2001;15:575-586.
4. Marchese MR, D'Alatri L, Bentivoglio AR, Paludetti G. OnabotulinumtoxinA for adductor spasmodic dysphonia (ADSD): functional results and the role of dosage. *Toxicon*. 2018;155:38-42.
5. Dejonckere PH, Neumann KJ, Moerman MBJ, Martens JP, Giordano A, Manfredi C. Tridimensional assessment of adductor spasmodic dysphonia pre- and post-treatment with botulinum toxin. *Comparative Study*. 2012; 269:1195-1203.
6. Blitzer A, Mitchell F, Simonyan K, Ozelius LJ, Frucht SJ. Phenomenology, genetics, and CNS network abnormalities in laryngeal dystonia: a 30-year experience. *Laryngoscope*. 2018;128:S1-S9.
7. Frankford SA, O'Flynn L, Simonyan K. Sensory processing in the auditory and olfactory domains is normal in laryngeal dystonia. *J Neurol*. 2023; 270:2184-2190.
8. Gold T. Speech production in hearing-impaired children. *J Commun Disord*. 1980;13:397-418.
9. Smith CR. Residual hearing and speech production in deaf children. *J Speech Hear Res*. 1975;18:795-811.
10. Simonyan K, Ludlow CL. Abnormal activation of the primary somatosensory cortex in spasmodic dysphonia: an fMRI study. *Cereb Cortex*. 2010; 20:2749-2759.
11. Daliri A, Murray ESH, Blood AJ, et al. Auditory feedback control mechanisms do not contribute to cortical hyperactivity within the voice production network in adductor spasmodic dysphonia. *J Speech Lang Hear Res*. 2020;63:421-432.
12. Ali SO, Thomassen M, Schulz GM, et al. Alterations in CNS activity induced by botulinum toxin treatment in spasmodic dysphonia: an H215O PET study. *J Speech Lang Hear Res*. 2006;49:1127-1146.
13. Ludlow CL, Naunton RF, Sectary SE, Schulz GM, Hallett M. Effects of botulinum toxin injections on speech in adductor spasmodic dysphonia. *Neurology*. 1988;38:1220-1225.
14. Naunheim ML, Yung KC, Schneider SL, et al. Vocal motor control and central auditory impairments in unilateral vocal fold paralysis. *Laryngoscope*. 2019;129:2112-2117.
15. Thomas A, Mirza N, Eliades SJ. Auditory feedback control of vocal pitch in spasmodic dysphonia. *Laryngoscope*. 2021;131:2070-2075.
16. Brumm H, Zollinger SA. The evolution of the Lombard effect: 100 years of psychoacoustic research. *Behaviour*. 2011;148:1173-1198.
17. Zollinger SA, Brumm H. The Lombard effect. *Curr Biol*. 2011;21(16):R614-R615.
18. Hotchkin C, Parks S. The Lombard effect and other noise-induced vocal modifications: insight from mammalian communication systems. *Biol Rev Camb Philos Soc*. 2013;88(4):809-824.
19. Jarvis ED, Güntürkün O, Bruce L, et al. Avian brains and a new understanding of vertebrate brain evolution. *Nat Rev Neurosci*. 2005;6(2): 151-159.
20. Xiong XR, Liang F, Zingg B, et al. Auditory cortex controls sound-driven innate defense behaviour through corticofugal projections to inferior colliculus. *Nat Commun*. 2015;6:7224.
21. Liu B, Huberman AD, Scanziani M. Cortico-fugal output from visual cortex promotes plasticity of innate motor behavior. *Nature*. 2016;538:383-387.
22. Geangu E, Benga O, Stahl D, Striano T. Contagious crying beyond the first days of life. *Infant Behav Dev*. 2010;33:279-288.
23. Bou-Ghazale SE, Hansen JHL. A comparative study of traditional and newly proposed features for recognition of speech under stress. *IEEE Trans Speech Audio Proc*. 2000;8:429-442.
24. Garnier M, Henrich N, Dubois D. Influence of sound immersion and communicative interaction on the Lombard effect'. *J Speech Lang Hear Res*. 2010;53:588-608.
25. Lee J, Ali H, Ziaei A, Tobey EA, Hansen JHL. The Lombard effect observed in speech produced by cochlear implant users in noisy environment: a naturalistic study. *J Acoust Soc Am*. 2017;141:2788-2799.
26. Lane H, Tranel B. The Lombard sign and the role of hearing in speech. *J Speech Hear Res*. 1971;14:677-709.
27. Adams SG, Lang AE. Can the Lombard effect be used to improve low voice intensity in Parkinson's disease? *Eur J Disord Commun*. 1992;27:121-127.
28. Quedas A, Duprat Ade C, Gasparini G. Lombard's effect's implication in intensity, fundamental frequency and stability on the voice of individuals with Parkinson's disease. *Braz J Otorhinolaryngol*. 2007;73:675-683.
29. Armson J, Kiefte M, Mason J, De Croos D. The effect of SpeechEasy on stuttering frequency in laboratory conditions. *J Fluency Disord*. 2006;31: 137-152.
30. Armson J, Kiefte M. The effect of SpeechEasy on stuttering frequency, speech rate, and speech naturalness. *J Fluency Disord*. 2008;33:120-134.
31. Castro C, Prado P, Espinoza VM, et al. Lombard effect in individuals with Nonphonotraumatic vocal hyperfunction: impact on acoustic, aerodynamic, and vocal fold vibratory parameters. *J Speech Lang Hear Res*. 2022;65(8):2881-2895.
32. Jankovic J, Schwartz K, Donovan DT. Botulinum toxin treatment of cranial- cervical dystonia, spasmodic dysphonia, other focal dystonias and hemi- facial spasm. *J Neurol Neurosurg Psychiatry*. 1990;53(8):633-639.
33. Boutsen F, Cannito MP, Taylor M, Bender B. Botox treatment in adductor spasmodic dysphonia. *J Speech Lang Hear Res*. 2002;45:469-481.
34. Truong DD, Rontal M, Rolnick M, Aronson AE, Mistura K. Double-blind controlled study of botulinum toxin in adductor spasmodic dysphonia. *Laryngoscope*. 1991;101:630-634.
35. Blitzer A, Brin MF, Stewart C. Botulinum toxin management of spasmodic dysphonia (laryngeal dystonia): a 12-year experience in more than 900 patients. *Laryngoscope*. 2015;125:1751-1757.
36. Simonyan K, Barkmeier-Kraemer J, Blitzer A, et al. Laryngeal dystonia, multidisciplinary update on terminology, pathophysiology, and research priorities. *Neurology*. 2021;96(21):989-1001.
37. Schindler O. *Breviario di Patologia della Comunicazione*. Vol 2. Torino: Omega; 1985.
38. Coles RR, Priede VM. Masking of the non-test ear in speech audiometry. *J Laryngol Otol*. 1975;89(3):217-226.
39. Miller GA. The masking of speech. *Psychological Bull*. 1947;44(2):105-129.
40. Almeida K, Russo ICP, Momensohn-Santos TM. *A aplicação do Mascaramento em Audiologia*. São Paulo: Lovise; 2001:21-36.
41. Forrest K, Abbas PJ, Zimmermann GN. Effects of white noise masking and low pass filtering on speech kinematics. *J Speech Hear Res*. 1986;29(4):549-562.
42. Latoszek Ben BV, Watts CR, Schwan K. The maximum phonation time as marker for voice treatment efficacy: a network meta-analysis. *Clin Otolaryngol*. 2023;48(2):130-138.
43. Chernobelskii SI. The use of voicing in phoniatrics. *Vestn Otorinolaringol*. 2005;5:26-27.
44. Mezzedimi C, Di Francesco M, Livi W, Spinosi MC, De Felice C. Objective evaluation of presbyphonia: spectroacoustic study on 142 patients with praat. *J Voice.* 2017;31(2):257.e25-257.e32.
45. Swathy AA, Shetty PP, Mathew K. Acoustic analysis of indian teacher's voice: pre and post teaching circumstances. *Indian J Otolaryngol Head Neck Surg*. 2019;71(Suppl 1):S100-S103.
46. Tetsuji S, Yumoto E, Kodama N, Minoda R, Kumai Y. Long-term voice handicap index after type II thyroplasty using titanium bridges for adductor spasmodic dysphonia. *Auris Nasus Larynx*. 2014;41(3):285-289.
47. Maryn Y, Weenink D. Objective dysphonia measures in the program Praat: smoothed cepstral peak prominence and acoustic voice quality index. *J Voice*. 2015;29:35-43.
48. Hillenbrand J, Houde RA. Acoustic correlates of breathy vocal quality: dysphonic voices and continuous speech. *J Speech Lang Hearing Res*. 1996; 39:311-321.
49. Isshiki N, Okamura H, Tanabe M, Morimoto M. Differential diagnosis of hoarseness. *Folia Phoniatr (Basel)*. 1969;21:9-19.
50. Shoffel-Havakuk H, Marks KL, Morton M, Johns MM, Hapner ER. Validation of the OMNI vocal effort scale in the treatment of adductor spasmodic dysphonia. *Laryngoscope*. 2019;129(2):448-453.
51. Roy N, Mazin A, Awan SN. Automated acoustic analysis of task dependency in adductor spasmodic dysphonia versus muscle tension dysphonia. *Laryngoscope*. 2014;124:718-724.
52. Kreiman J, Vanlancker-Sidtis D, Gerratt BR. Perception of voice quality. In: Pisoni DB, Remez RE, eds. *The Handbook of Speech Perception*. Malden, MA: Blackwell Publishing; 2005:338-362.
53. Bond Z, Moore T, Gable B. Acoustic-phonetic characteristics of speech produced in noise and while wearing an oxygen mask. *J Acoust Soc Am*. 1989;85:907-912.

54. Ludlow CL, Adler CH, Berke GS, et al. Research priorities in spasmodic dysphonia. *Otolaryngol Head Neck Surg*. 2008;139:495-550.
55. Brockmann-Bauser M, Van Stan JH, Carvalho Sampaio M, Bohlender JE, Hillman RE, Mehta DD. Effects of vocal intensity and fundamental frequency on cepstral peak prominence in patients with voice disorders and vocally healthy controls. *J Voice*. 2021;35(3):411-417.
56. Izdebski K. Symptomatology of adductor spasmodic dysphonia: a physiologic modhel. *J Voice*. 1992;6:306-319.
57. Guiry S, Worthley A, Simonyan K. A separation of innate and learned vocal behaviors defines the symptomatology of spasmodic dysphonia. *Laryngoscope*. 2019;129:1627-1633.
58. Le Lombard E. signe de l'elevation de la voix. *Ann Maladies l'Oreille Larynx Nez Pharynx*. 1911;37:101-119.
59. Luo J, Hage SR, Moss CF. The Lombard effect: from acoustics to neural mechanisms. *Trends Neurosci*. 2018;41(12):938-949.
60. Roy S, Miller CT, Gottsch D, Wang X. Vocal control by the common marmoset in the presence of interfering noise. *J Exp Biol*. 2011;1(214):3619-3629.
61. Pick HL Jr, Siegel GM, Fox PW, Garber SR, Kearney JK. Inhibiting the Lombard effect. *J Acoust Soc Am*. 1989;85(2):894-900.
62. McColl D, McCaffrey P. Perception of spasmodic dysphonia speech in background noise. *Percept Mot Skills*. 2006;103(2):629-635.