Role of early voice therapy in patients affected by unilateral vocal fold paralysis

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Abstract

Objective: To evaluate the functional results obtained after voice therapy in patients with unilateral vocal fold paralysis caused by different aetiologies.

Design: Prospective analysis of the outcome of unilateral vocal fold paralysis cases treated at our speech and language rehabilitation service from November 2003 to January 2006. Thirty cases underwent behavioural treatment, between two and six weeks after unilateral vocal fold paralysis onset. A multi-dimensional assessment was carried out before, immediately after and six months after treatment.

Results: After behavioural therapy, the prevalence of complete glottal closure increased significantly (p < 0.05). Subjects' pre-therapy mean values for jitter, shimmer and noise-to-harmonic ratio were statistically significantly different from those taken both immediately and six months after treatment (p < 0.05). The mean values for voice turbulence index significantly improved only six months after therapy $(0.08 \ vs \ 0.04)$. At both post-treatment assessments, voice range profile analysis showed a significant decrease of lowest voice frequency and a significant increase of the number of semitones (p < 0.05). Mean values for grade, instability, breathiness, asthenia and voice handicap index scores were significantly decreased both immediately and six months after treatment, compared with pre-treatment values (p < 0.05).

Conclusions: Early voice therapy may enable significant improvement in vocal function, allowing the patient to avoid surgery.

Key words: Vocal Cord Paralysis; Voice Disorders; Recurrent Laryngeal Nerve; Speech Therapy

Introduction

Recurrent laryngeal nerve palsy may occur due to a variety of causes (e.g. tumour, trauma, vascular insult, viral or bacterial infection, and neurotoxic drugs). The mechanisms of these aetiologies of nerve damage differ, from temporary, partial neuropraxia to complete nerve disruption. The resulting laryngeal deficit may also vary, from a paramedian, tonic vocal fold to a lateralised, flaccid vocal fold. The position of the vocal fold is not diagnostic of the site of the nerve lesion; however, the width of the glottic gap affects the severity of clinical presentation.¹

Unilateral vocal fold paralysis may alter phonation, airway protection, breathing and stabilisation of the body core during physical activity. However, dysphonia is almost always the symptom that causes the patient to seek medical attention. The degree of voice complaint depends on the amount of glottal incompetence and on the type of compensatory behaviours that the patient may carry out in order to improve vocal intensity.¹ Usually, the voice is breathy and hoarse, with limited pitch and loudness variation and short phonation time. Patients cannot speak loudly, and diplophonia can occur as they increase effort to attain glottal closure. Compensatory hyperfunctional behaviours, such as anterior– posterior or lateral compression of the false vocal folds, can result in a rough, strained and low-pitched voice, while hyperfunction of cricothyroid muscles may lead to a falsetto register. Vocal fatigue, globus sensation and neck discomfort are other subjective symptoms that may be associated with unilateral vocal fold paralysis. This condition may have a negative impact on patients' quality of life.

The currently reported treatment of unilateral vocal fold paralysis comprises either conservative or surgical treatment.

The principal goals of voice therapy are: improvement of glottal closure and of intrinsic muscle strength and agility (without causing supraglottic hyperfunction); and development of abdominal support for breathing. The most commonly used

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behavioural approaches involve: hard glottal attacks and 'pushing';² half-swallow boom;³ abdominal breathing;⁴ vocal function;⁵ appropriate tone focus;⁶ accent method;⁷ and lip and tongue trill.⁸

Currently, the main surgical treatment options for unilateral vocal fold paralysis are: vocal fold augmentation (injection); laryngeal framework surgery (thyroplasty, arytenoid adduction); and laryngeal reinnervation.

Results for surgical management of unilateral vocal fold paralysis have been widely reported in the literature, but information regarding the efficacy of behavioural treatment is scarce.^{9,10} On the other hand, the improvement in voice which can occur with voice therapy, or sometimes spontaneously within one year of onset of paralysis, gives reason for corrective surgery not usually considered the primary treatment. Moreover, it is mandatory to validate the utility of voice therapy as a primary treatment (in order to avoid surgical treatment)-or as an adjunct to surgery.¹¹

The aim of the present study was to use multidimensional assessment, including objective and subjective measures, to analyse the functional outcomes achieved by a group of patients with recent onset of unilateral vocal fold paralysis who underwent voice therapy.

Materials and methods

Patients

Between November 2003 and January 2006, 91 subjects with unilateral vocal fold paralysis presented to our speech and language rehabilitation service in order to undergo voice therapy. When first assessed, none of them complained of dysphagia or had clinically demonstrated aspiration.

All patients commenced behavioural treatment between two and six weeks after the onset of unilateral vocal fold paralysis. Therapy was given for 30 minutes twice a week; the mean number of sessions was 24 (range eight to 35). The last follow-up consultation was planned six months after the end of voice therapy.

Recovery of vocal fold motion was detected endoscopically in 57/91 (62.64 per cent) patients, during treatment, within six months of onset of unilateral vocal fold paralysis.

After voice therapy, unilateral vocal fold paralysis persisted in 34/91 (37.36 per cent) patients (30 females and four males). These patients were followed up for between seven and 11 months from the onset of unilateral vocal fold paralysis.

The number of male subjects was too small (4/34; 11.76 per cent) to perform statistical analysis; therefore, we decided to include in our study group only the female patients (30/34; 88.23 per cent). These subjects' mean age was 41.56 years (range 31–68 years). The aetiologies of unilateral vocal fold paralysis for the study group are reported in Table I.

Each patient was evaluated objectively and subjectively before, immediately after and six months after voice therapy.

TABLE I UNILATERAL VOCAL FOLD PARALYSIS AETIOLOGY

Cause	Cases	
	<i>n</i> *	%
Thyroidectomy	24	80
Idiopathic	3	10
Endarterectomy	2	6.66
Oesophagoplasty	1	3.33

*Of 30.

Voice therapy

A functional diagnosis and a treatment plan were formulated on completion of the initial evaluation. Voice therapy was individualised, based on the degree of glottal incompetence as well as on the type and degree of the compensatory behaviours used by the patient. The first session comprised a broad-based vocal education and hygiene programme. Initially, training was directed toward progressive development of abdominal breathing support and control of the duration of utterance. Vocal exercises were designed to strengthen and coordinate the laryngeal musculature and to improve the interplay between airflow, vocal fold vibration and supraglottic structures.⁵ Patients were instructed to produce vocal exercises as softly as possible, without being 'breathy', and to maintain a relaxed, open vocal tract without postural tension changes. Pushing exercises and hard vocal attacks were always avoided. The half-swallow boom technique³ and lip and tongue trill⁸ were also used in some cases in order to improve glottal closure. Patients who used a falsetto register were helped to bring out the chest voice by moving the larynx to a lower position in the neck (manually or using techniques like deep inhalation and yawning). In the presence of ventricular hyperfunction, we used techniques such as speaking on inhalation¹² and nasal twang.¹³ Moreover, manual laryngeal muscle tension reduction tech-niques¹⁴ and laryngeal reposturing¹⁵ helped to decrease supralaryngeal muscle tension. Finally, training in auditory and proprioceptive biofeedback was instilled at every step of the behavioural treatment programme, in order to enable the patient to hear and feel the appropriate voice production.

Objective and subjective assessment

Strobovideolaryngoscopy. This was performed using a 70°, rigid laryngoscope (model RLS 9100 B, Kay Elemetrics, Lincoln Park, New Jersey, USA). Glottal closure was evaluated as: incomplete glottal closure of the entire glottis; incomplete closure of the membranous portion of the vocal fold; posterior glottal chink; or complete glottal closure.

Maximum phonation time. This was obtained by having the patient sustain the vowel /a/ for as long as possible on a single breath. The longest of three attempts was calculated as the maximum phonation time.

Acoustic voice analysis. This was performed via a multi-dimensional voice program (model 5105) and a voice range profile program (model 4326), using a computerised speech laboratory (model 4300B; Kay Elemetrics). Speech was recorded with a uni-directional, dynamic microphone (model SM48; Shure, Evanston, Illinois, USA), which was positioned at a 45° angle and at a distance of 20 cm from the patient's mouth. The microphone saturation input was fixed at 6/9 of channel 1 (CH1), and the environmental noise was <30 dB SPL.

Subjects were asked to sustain the vowel /a/ at a comfortable pitch and loudness level for at least four seconds. This sound was recorded with a sampling rate of 50 000 Hz for multi-dimensional voice program analysis. The quality of the signals was visually inspected and classified according to Titze's recommendation.¹⁶ We selected an interval of three seconds from the mid portion of each vowel in order to analyse the following parameters: mean fundamental frequency (Hz), jitter (per cent), shimmer (per cent), noise-to-harmonic ratio, voice turbulence index, and degree of sub-harmonics.

Patients undergoing voice range profile analysis were instructed to phonate using a sustained /a/vowel, as loudly and as softly as possible, from the lowest to the highest frequencies possible. An automated procedure was then used to obtain a voice range profile. The voice range profile program determined the lowest and highest vocal frequencies (Hz) and the number of semitones between these two measures. Patients' intensity ranges were not analysed, because a diplophonic sound or a falsetto voice (both commonly used) increased loudness.

Perceptual voice analysis and patient self-assessment. Blind perceptual evaluation, using the grade– instability–roughness–breathiness–asthenia–strain scale,^{17,18} was performed on recorded voice samples (i.e. conversational speech, a reading task and sustained vowels) by a team comprising one phoniatrician, one otorhinolaryngologist and two experienced speech therapists. The two speech therapists in the team did not treat any of the patients in the study. Finally, patient self-assessment of dysphonia was carried out using the voice handicap index.¹⁹

Statistical analysis

Statistical analysis was performed using a commercially available statistical software package (the Statistical Package for the Social Sciences version 10.0 for Windows[®]; SPSS Inc, Chicago, Illinois, USA). The chi-square test was used for categorical variables, while analysis of variance was used for continuous variables. A *p* value of less than 0.05 was considered as significant.

Results

Strobovideolaryngoscopy

Before commencement of voice therapy, complete glottal closure was found in six of 30 (20 per cent) patients, while glottal incompetence of the

membranous portion of the vocal folds and incomplete closure of the entire glottis were respectively visible in nine of 30 (30 per cent) and 15/30 (50 per cent) subjects. Following voice therapy (6 months later), complete glottal closure was observed in 15/ 30 (50 per cent) cases, posterior glottal chink in three of 30 (10 per cent), incomplete closure of the membranous portion of the vocal folds in six of 30 (20 per cent), and persistent, incomplete glottal closure of the entire glottis in six of 30 (20 per cent). After voice therapy (6 months later), the prevalence of complete glottal closure was significantly higher than before therapy, while the prevalence of incomplete glottal closure of the entire glottis was significantly lower (p < 0.05). Furthermore, lateral compression of the false vocal fold was observed in eight of 30 (26.66 per cent) patients before voice therapy but in only one of 30 (3.33 per cent) patients immediately after behavioural treatment (p < 0.05). Six months later, strobovideolaryngoscopic findings remained essentially unchanged.

Maximum phonation time

Compared with mean pre-therapy values $(6.50 \pm 3.22 \text{ seconds})$, patients' maximum phonation time significantly improved at the end of treatment $(11.15 \pm 4.2 \text{ seconds})$ and six months later $(11.40 \pm 3.98 \text{ seconds})$ (p < 0.05).

Acoustic voice analysis

Before voice therapy, nine of 30 (30 per cent) patients had a maximum phonation time of less than four seconds, and three of 30 (10 per cent) patients had type three spectrograms (according to Titze's classification);¹⁶ therefore, these patients were excluded from multi-dimensional voice program analysis. In addition, all 12 of these patients were unable to undergo voice range profile analysis. After voice therapy, almost all of these patients (11/12) had achieved good voice improvement, and so underwent both multi-dimensional voice analysis and voice range profile analysis; only one subject was still excluded from perturbation and phonetographic evaluation, because of low maximum phonation time (<4 seconds). In this study, statistical analysis of multi-dimensional voice program and voice range profile program parameters was carried out only for those patients who underwent a complete set of three assessments.

The results of multi-dimensional voice program and voice range profile program analysis are reported in Table II.

After voice therapy, mean fundamental frequency values were found to decrease, but this difference was not significant. However, the differences between pretherapy and early and late post-therapy mean values for jitter, shimmer and noise-to-harmonic ratio were statistically significant (p < 0.05). Voice turbulence index significantly improved, comparing pre-therapy and six months' post-therapy mean values (0.08 vs 0.04). There was no significant difference in sub-harmonics mean values, comparing pre-therapy and early and late post-therapy measurements.

TABLE II MDVP AND VRP ANALYSIS: PRE- AND POST-THERAPY RESULTS Parameter Pre-therapy Doct thorony

Parameter	Pre-therapy	Post-therapy		
		Early*	Late†	
F_0 (Hz)	176.77 ± 51.17	172.44 ± 47.47	187.63 ± 35.32	
Jitter (%)	2.00 ± 1.72	$0.91 \pm 0.69 \ddagger$	$0.75 \pm 0.55^{**}$	
Shimmer (%)	7.9 ± 3.45	$5.28 \pm 3.08 \ddagger$	$4.99 \pm 2.38^{**}$	
NHR	0.19 ± 0.08	$0.14 \pm 0.04 \ddagger$	$0.14 \pm 0.03^{**}$	
VTI	0.08 ± 0.05	0.06 ± 0.02	$0.04 \pm 0.02^{**}$	
DSH (%)	2.83 ± 4.87	0.86 ± 2.21	0.00 ± 0.00	
F_{low} (Hz)	195.85 ± 22.00	$171.31 \pm 21.68 \ddagger$	$167.66 \pm 16.22^{**}$	
F _{high} (Hz)	336.15 ± 69.24	355.80 ± 40.30	370.99 ± 53.46	
Semitones (n)	10.11 ± 2.47	$13.78\pm1.86\ddagger$	$14.67 \pm 2.12^{**}$	

Data are shown as mean ± standard deviation. *Immediately after therapy; [†]6 months after therapy. [‡]p < 0.05, comparing pre- and early post-therapy values; ^{*}p < 0.05, comparing pre- and late post-therapy values. MDVP = multi-dimensional pre- multi-dimensional voice program; VRP = voice range profile; F_0 = mean funda-mental frequency; NHR = noise-to-harmonic ratio; VTI = voice turbulence index; DSH = degree of sub-harmonics; $F_{low} = lowest voice frequency; F_{high} = highest voice frequency$

Voice range profile program analysis showed a significant decrease in the mean lowest voice frequency and a significant increase in the number of semitones (i.e., mean value) between the lowest and highest voice frequencies, comparing pre-therapy data with both post-treatment assessments (p < 0.05) (Table II).

Perceptual voice analysis and patient self-assessment

At both post-therapy assessments, mean values for grade, instability, breathiness and asthenia were significantly decreased compared with pre-therapy findings (p < 0.05) (Table III). Moreover, the same parameters showed a significant further decrease, comparing early and late post-treatment measurements.

Voice handicap index values before and after voice therapy are shown in Figure 1. Mean values for total scores significantly decreased, as did those for the physical, functional and emotional subscales, comparing pre-therapy with both post-therapy measurements (p < 0.05).

TABLE III				
GIRBAS SCALE SCORES				

Parameter	Pre-therapy	Post-therapy	
		Early*	Late [†]
Grade Instability Roughness Breathiness Asthenia Strain	$\begin{array}{c} 2.15 \pm 0.93 \\ 1.05 \pm 0.89 \\ 0.78 \pm 0.65 \\ 1.95 \pm 1.00 \\ 2.00 \pm 0.92 \\ 0.15 \pm 0.37 \end{array}$	$\begin{array}{c} 1.30 \pm 0.73 \ddagger \\ 0.55 \pm 0.60 \ddagger \\ 0.55 \pm 0.51 \\ 0.70 \pm 0.92 \ddagger \\ 0.90 \pm 0.79 \ddagger \\ 0.00 \pm 0.00 \end{array}$	$\begin{array}{c} 1.00\pm 0.79^{**\$}\\ 0.15\pm 0.37^{**\$}\\ 0.50\pm 0.61\\ 0.35\pm 0.75^{**\$}\\ 0.65\pm 0.67^{**\$}\\ 0.00\pm 0.00^{**} \end{array}$

Data are shown as mean ± standard deviation. *Immediately after therapy; [†]6 months after therapy. [‡]p < 0.05, comparing pre- and early post-therapy values; ^{*}p < 0.05, comparing pre- and late post-therapy values; [§]p < 0.05, comparing early and late post-therapy values. GIRBAS = grade-instabilityroughness-breathiness-asthenia-strain



Pre- and post-therapy voice handicap index scores. *p < 0.05, comparing pre- and early post-therapy values; $^{\dagger}p < 0.05$, comparing pre- and late post-therapy values. Pre = pre-treatment; early post = immediately after treatment; late post = 6 months after treatment

Discussion

This study highlights the importance of early voice therapy in patients affected by unilateral vocal fold paralysis. Published data on the efficacy of such therapy are scant. Two studies have compared the results obtained in patients with unilateral vocal fold paralysis treated by surgery and voice therapy.9,10 Both studies suggested that conservative therapy enables voice improvement and that aerodynamic measures are good predictors of the need for surgery.

Thirty female patients affected by recent unilateral vocal fold paralysis and undergoing voice therapy were studied using a multi-dimensional assessment. In spite of persistent unilateral vocal fold paralysis, all of the subjects showed significant improvement in endoscopic, aerodynamic, acoustic, perceptual and self-assessed measurements, following behavioural treatment. The late post-therapy follow-up consultation demonstrated stability of functional results.

Since voice is not a single, numeric outcome but a multi-dimensional function, evaluation of voice therapy efficacy required carefully selected assessment measures.²⁰ Morphofunctional laryngeal analysis cannot be directly related to the treatment outcomes of patients with unilateral vocal fold paralysis. In fact, strobovideolaryngoscopic findings do not always correspond to the degree of vocal impairment.

Posterior glottal chink is often found in a large percentage of normal women and in some men.²¹ We found persistence of incomplete glottal closure (entire or limited to the membranous portion of the vocal folds) after voice therapy in 12/30 (40 per cent) patients. Of these patients, only one did not show any improvement in vocal function and was therefore referred for phonosurgical treatment.

Behavioural training induces favourable compensatory strategies, such as extensive breath support and control and vocal tract postural adjustments, and minimises undesirable compensatory hyperfunctional behaviours, enabling functional improvement despite the persistence of a glottal gap. The increase in glottal closure observed after voice therapy may be due not only to adequate compensation of the contralateral vocal fold, but also to the paralysed vocal fold advancing to the midline, owing to the activity of the interarytenoid muscle or to passive lengthening induced by the cricothyroid muscle. Moreover, glottal closure may improve due to the compensatory activity of the inferior pharyngeal constrictor muscle.

- Unilateral vocal fold paralysis may alter phonation, airway protection, breathing and stabilisation of the body core during physical activity. However, dysphonia is almost always the symptom that causes the patient to seek medical attention
- Little research on this topic has been published; this study highlights the importance of early voice therapy in patients affected by unilateral vocal fold paralysis
- Early voice therapy may enable significant improvement in vocal function, which could avoid the need for surgery

The post-treatment acoustic parameters suggest a greater stability of vocal fold vibration, with a consequent improvement in the harmonic structure of the spectrum. It is important to note that, after voice therapy, acoustic voice analysis was reliable in 11/12 patients initially excluded from multi-dimensional voice program and voice range profile program analysis because of low maximum phonation time and strongly aperiodic signals. Thus, the reliability of acoustic voice analysis after treatment constitutes in itself a significant index of treatment outcome.

Concerning acoustic analysis, the main result of behavioural treatment was the significant increase in the number of semitones in the voice range profile, revealed by a significant lowering of the mean minimum frequency while the mean maximum frequency remained unchanged. To our knowledge, this is the first published report of such data. Patients with unilateral vocal fold paralysis often develop compensatory laryngeal behaviours, such as an elevated position of the larynx in the neck, causing a high pitched voice; the lower minimal frequency of the voice range profile observed after voice therapy may be due to better use of the chest register.

In the clinical practice of a speech therapist, assessment tools such as strobovideolaryngoscopy and acoustic voice analysis are rarely available. Therefore, perceptual evaluation of voice quality remains an essential component of clinical examination and of treatment outcome assessment. Regarding the use of the grade-instability-roughness-breathiness-asthenia-strain scale, it has already been reported that, in untreated patients, grade, breathiness and asthenia are the most sensitive parameters for paralytic dysphonia.²² In our study, these parameters improved significantly after voice therapy.

Treatment outcome addresses the physical, mental and social well-being of a patient. Self-assessment measures provide outcome data from the patient's perspective, and are particularly sensititive to quality of life changes caused by dysphonia.²³ Moreover, the patient's perception of dysphonia severity may influence the treatment type and results. The voice handicap index has been shown to have testretest reliability and to be sensitive for a wide variety of voice disorders.¹⁹ It is interesting to note that, when monitoring success (or lack thereof) during and after treatment, changes in the patient's voice handicap index score may be more important than the absolute score.²⁴ The voice handicap index score of patients with untreated unilateral vocal fold paralysis tends to reveal greater perceived vocal dysfunction, particularly in the physical subscale, compared with patients with dysphonia from other causes.²⁴ This finding is probably due to severe voice change suddenly experienced by patients. They (the patients) generally preserve a good auditory and proprioceptive biofeedback which enables good treatment compliance, which is important to the success of voice therapy.

In this study, electromyographic (EMG) data were not reported. Our patients commenced voice therapy within six weeks of onset of unilateral vocal fold paralysis. It is recognised that EMG studies become useful at approximately six weeks to three months after paralysis.²⁵ During this period, our patients had already begun behavioural treatment, with positive initial effects. The patients, asked to undergo EMG analysis refused to participate in this invasive procedure.

Based on our experience, we believe that the use of early and appropriate behavioural voice treatment could avoid the need for surgery in many unilateral vocal fold paralysis patients, without exposure to unnecessary risks. Surgical treatment may be considered after failure of conservative rehabilitation, or for those cases with additional symptoms besides dysphonia, especially dysphagia and aspiration.²⁶ Moreover, the need for intervention depends on many factors, the most important of which is the patients' vocal requirements, based on their occupational and social demands.

Conclusion

Early voice therapy may enable significant improvement of vocal function in patients affected by unilateral vocal fold paralysis. On the other hand, residual innervation²⁷ and unpredictable reinnervation and synkinesis,^{28–30} which maintain vocal bulk and tonus, may sometimes lead to spontaneous vocal recovery even in the absence of restored vocal fold motion. For this reason, further research is required in order to establish the contribution of these factors to the functional outcome of patients with unilateral vocal fold paralysis receiving behavioural treatment.

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