Laryngeal Hyperfunction During Whispering: Reality or Myth?

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Summary: For years, otolaryngologists and voice therapists have warned voice patients that whispering causes more trauma to the larynx than normal speech. However, no large series of patients has ever been examined fiberoptically during whispering to test this hypothesis. As part of our routine examination, patients are asked to count from 1 to 10 in a normal voice and in a whispered voice. We reviewed recorded fiberoptic examinations of 100 patients who had voice complaints. We compared supraglottic hyperfunction and vocal fold closure during the normal and whispered phonation of each patient. Sixty-nine percent of the patients demonstrated increased supraglottic hyperfunction with whispered voice. Eighteen percent had no change, and 13% had less severe hyperfunction. The most common glottal configuration during whisper was an inverted Y, which resulted from compression of the anterior and middle thirds of the true vocal folds. However, 12 patients had no true vocal fold contact during whispered voice, despite having adequate glottic closure with normal voice. Although whispering involves more severe hyperfunction in most patients, it does not seem to do so in all patients. In some patients, it may be less traumatic than normal voice.

Key Words: Whisper—Whispering—Voice rest—Vocal fold—Vocal cord—Hoarseness—Dysphonia—Muscle tension—Voice abuse—Voice misuse.

INTRODUCTION

For years, otolaryngologists, voice therapists, and singing teachers have warned patients that whispering is more traumatic to the vocal folds than normal speech. To date, however, no sizable series of patients has ever been examined fiberoptically during whispering to test this hypothesis.

The basic paradigm that evaluates laryngeal hyperfunction is to look for compression of the supraglottic structures during phonation. This supraglottic constriction may occur as medial compression, with squeeze of the false vocal folds, or as anterior–posterior (A–P) constriction, with compression of the epiglottis and arytenoid cartilages toward each other. If whispering is more harmful to the vocal folds than normal speaking, it seems reasonable to

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assume that patients should demonstrate evidence of increased supraglottic hyperfunction, and the true vocal folds should make firm contact during whispering. Whispering involves increased airflow, in addition to a change in laryngeal resistance. An open channel through the larynx for air escape is necessary to produce a whisper. However, it is conceivable that glottal opening may be wider during whispering than during normal speech, and, despite an apparently less favorable supraglottic configuration, the true vocal folds might not touch during whispered speech.

MATERIAL AND METHODS

A review of recorded fiberoptic examinations of 100 patients evaluated by the senior author (RTS) from July 2003 to March 2004 was performed. All patients had vocal complaints. Pathologic diagnoses included muscle tension dysphonia, vocal fold paresis or paralysis, vocal fold mass, laryngopharyngeal reflux, arytenoid cartilage dislocation, vocal fold scar, and functional voice disorders. During routine fiberoptic voice evaluations, patients are asked to perform various vocal tasks to elicit evidence of hyperfunction and paresis. To assess for hyperfunction, in addition to other tasks, patients are asked to count from 1 to 10 in a normal (nonwhispered) and in a whispered voice. No specific instructions are given on how to whisper. Typically, the degree of hyperfunction in A–P and medial compression is assessed for each voice type. When paresis is suspected, laryngeal electromyography (LEMG) is performed by a neurologist who is board-certified in EMG and has performed over 4000 LEMGs. A monopolar electrode tests the cricothyroid, vocalis, and posterior cricoarytenoid muscle.

The normal and whispered voice "1–10" portions of each examination were recorded onto a CD-ROM without identifiers except a specific number assigned randomly to each patient. The examinations were reviewed by two otolaryngologists, currently laryngology fellows (ADR, VP), and a voice therapist (SG). Supraglottic hyperfunction was assessed as absent, mild, moderate, or severe for both the normal and whispered voice samples of each patient. Hyperfunction in the A–P direction was assessed as mild if there was slight squeeze in the A–P vector, but more than half of the length of the glottis was still visible; moderate if 50% or more of the length of the vocal folds was obscured in the A-P dimension; and severe if the vocal folds were completely obscured by compression of the epiglottis against the arytenoid cartilages (Table 1). Medial hyperfunction was defined as mild if there was compression of the false vocal folds, but more than 50% of the width of the vocal folds was still visible; moderate if 50% or more of the width of the vocal folds was obscured by the false vocal folds; and severe if the musculomembranous portion of the true vocal folds was completely obscured by the false vocal folds or if the false vocal folds made contact with each other (Table 1). In the latter case, the posterior portion of the true vocal folds may have been visible, despite contact of the anterior false vocal folds. This case still was considered severe supraglottic hyperfunction.

Membranous true vocal fold contact during both the whispered and modal portions of the examination was assessed independently. True vocal fold contact was assessed as none, anterior 1/3, middle 1/3, posterior 1/3, anterior and middle thirds, entire vocal fold, or could not detect. Typically, we could not evaluate vocal fold contact if the supraglottic hyperfunction was severe enough to preclude visualization of the true vocal folds.

All data were entered into an *Excel* 6.0 spreadsheet (Microsoft Inc., Redmond, WA) and cleaned. Data were imported into *SPSS* version 11.0 for analysis (SPSS Inc., Chicago, IL). Descriptive statistics and frequencies were calculated. Means were compared with *t* tests, and categorical data were compared with chi-square tests. A *P* value of .01 was considered statistically significant.

This study was approved for exemption status by the Institutional Review Board, Graduate Hospital, Philadelphia, PA.

RESULTS

One-hundred patients were evaluated, 32 men and 68 women. Their age range was 19 to 87 years, with a mean age of 48. Only 1 patient had no supraglottic hyperfunction during normal voice. Sixty-nine patients (69%) demonstrated worse supraglottic hyperfunction in one or more vectors when whispering was compared with normal voice, 13 (13%) had

TABLE 1. *Grading Scale of Supraglottic Hyperfunction*

	Normal	Mild	Moderate	Severe
Horizontal	No false vocal fold compression	More than ½ of width of true vocal folds visible	Less than ½ of width of true vocal folds visible	False vocal fold contact
Anterior-posterior	No compression of epiglottis towards arytenoid cartilages	More than ½ of length of true vocal folds visible	Less than ½ of length of true vocal folds visible	True vocal folds completely obscured (epiglottis and arytenoid cartilages touching)

improvement in supraglottic hyperfunction, and 18 (18%) had no obvious change in supraglottic configuration.

Of the 69 patients with worse supraglottic constriction, 37 (54%) demonstrated more severe hyperfunction in only one vector, 29 in both vectors (42%), and 3 (4%) had worse hyperfunction in one vector but improved hyperfunction in the other vector. Looking independently at medial and A–P hyperfunction, 60% of the patients had worse hyperfunction in the medial vector during whisper, whereas 38% had worse hyperfunction in the A–P vector (Tables 2 and 3).

The most common configuration of the true vocal folds during whispered speech was contact involving the anterior and middle thirds of the vocal folds (48%) (Table 4). Sixteen patients (16%) demonstrated contact along the entire vocal fold during whisper speech, and 11 (11%) had contact in only the anterior third; in 10 patients (10%), we could not visualize the true vocal folds because of severe supraglottic hyperfunction.

Of the 48 patients demonstrating contact in the anterior and middle thirds of the vocal folds during whispering, 14 (29%) had similar configurations during normal speech. However, 31 patients (65%) had contact of the entire vocal fold during normal speech (Table 4).

In 14 patients (14%), there was no vocal fold contact during whispering. Of these patients, 2 had no vocal fold contact during normal speech. These 2 patients had immobile vocal folds (1 arytenoid cartilage dislocation, 1 vocal fold paralysis). However, the remaining 12 patients had some vocal fold contact during normal speech. Eight of the 14 patients (57%) had contact of at least two thirds of the length of the musculomembranous portion of the true vocal folds during modal speech.

Of the 14 patients who had no true vocal fold contact during whispered speech, 5 (36%) showed a

TABLE 2. *Cross-Tabulation Comparing Supraglottic Hyperfunction During Normal and Whispered Voice in the Horizontal Vector*

		Whispered Voice					
		None	Mild	Moderate	Severe	Could not determine	Total
Normal Voice	None	2		4	3		9
	Mild		8	18	16		42
	Moderate		7	12	19	1	39
	Severe			2	8		10
	Total	2	15	36	46	1	100

Note: Numbers indicate number of subjects with corresponding degrees of hyperfunction in normal and whispered voice.

TABLE 3. *Cross-Tabulation Comparing Supraglottic Hyperfunction in the Anterior-Posterior Vector During Normal and Whispered Voice*

		Whispered Voice					
		None	**Mild**	**Moderate**	**Severe**	**Could not determine**	**Total**
Normal Voice	None	5	5		2		12
	Mild		23	21	4		48
	Moderate		8	21	6		35
	Severe			2	3		5
	Total	5	36	44	15	0	100

Note: Numbers indicate number of subjects with corresponding degrees of hyperfunction in normal and whispered voice.

reduction in supraglottic hyperfunction with whispering, 5 (36%) showed no change, and 3 (21%) had increased hyperfunction. One patient (7%) showed an increase in medial hyperfunction and a decrease in A–P hyperfunction.

Looking at the patients who had vocal fold contact during whisper, of the 48 patients who had contact of the anterior and middle thirds of their true vocal folds, 39 (81%) had worsened supraglottic hyperfunction with whispering. Of the 11 patients who had only anterior third contact with whisper, 7 had increased supraglottic hyperfunction (64%), 3 improved (27%), and 1 had no change (9%). Of the 16 patients who had contact of the entire lengths of the true vocal folds with whisper, 12 (75%) had worse supraglottic hyperfunction with whisper, 2 (12.5%) had improved hyperfunction, and 2 (12.5%) showed no change. Overall, 77% of patients who had vocal fold contact during whisper had worse supraglottic hyperfunction (significantly greater than the 21% of patients without vocal fold contact, $P < .001$). Eleven percent had improved supraglottic hyperfunction, and 12% had no change in supraglottic hyperfunction.

LEMG data were available for 8 of the 12 patients with bilateral vocal fold mobility who had no true vocal fold contact with whisper. Of these 8 patients, 4 (50%) had bilateral paresis and 4 (50%) had unilateral paresis. Of the 86 patients who had some contact of the vocal folds with whisper, 58 had LEMG data available, and 47% demonstrated evidence of bilateral paresis. Only 3 of the 14 patients with no vocal fold contact during whisper had undergone formal voice training.

DISCUSSION

The current, broadly accepted paradigm of laryngeal hyperfunction is evaluated by observing the

TABLE 4. *Cross-Tabulation Comparing True Vocal Fold Contact During Normal and Whispered Voice. Number Indicates Number of Patients with Corresponding Glottal Configuration During Normal and Whispered Voice.*

		Whispered Voice							
		None	**Anterior third**	**Middle third**	**Posterior third**	**Anterior and middle thirds**	**Entire vocal fold**	**Cannot detect**	**Total**
Normal Voice	None	2						1	3
	Anterior third	1	1						2
	Middle third		1						1
	Posterior third	3				3			6
	Anterior and middle thirds	3	2			14	2	2	23
	Entire vocal fold	5	6		1	31	14	5	62
	Cannot detect		1					2	3
	Total	14	11	0	1	48	16	10	100

degree of supraglottic constriction during laryngeal examination. Koufman describes four patterns of muscle tension dysphonia (MTD I –IV). Each pattern involves different degrees of compression of the false vocal folds or front-to-back foreshortening of the larynx via A-P contraction of the supraglottis. These patterns may be compensatory for organic voice disorders or characteristic of functional, nonorganic disorders.[1] Regardless, these patterns are associated with hyperfunction and increased vocal fold contact forces believed to be potentially harmful to vocal health and associated with the formation of vocal fold masses in some cases.

We evaluated both medial (false vocal fold) and A-P (epiglottis–arytenoids) compression during phonation and whispering. We used our rating system of mild, moderate, and severe during our routine voice examination as well; this allowed us to compare the degree of supraglottic hyperfunction during normal and whispered voices. Although it is a somewhat subjective evaluation, it more than suffices for clinical purposes and this study. In addition, it is a simple system to use.

We also evaluated true vocal fold contact, as it seems conceptually logical that, although supraglottic constriction might seem worse during whisper, the vocal folds might not come together completely. Obviously, some part must remain open for the escape of air to create a whisper.

Previous studies have tried to characterize changes in aerodynamics and laryngeal configuration during whispered voice.[2–5] Unfortunately, most studies were limited by small sample sizes and less sophisticated visualization techniques. Moreover, many studies were performed in subjects with no vocal complaints. Therefore, they might have been less likely to provide conclusions applicable to a population in which whispering might contribute to or be potentially helpful for voice problems and pathology.

Solomon et al[5] studied supraglottic and glottic configurations in 10 subjects without voice complaints. No subjects demonstrated supraglottic hyperfunction during modal speech. The investigators evaluated laryngeal configurations via fiberoptic endoscopy during repeated vowel tasks and running whispered speech for both “quiet” and “forced” whisper. They reported two predominant configurations of the glottis during whisper: a toed-in, inverted-Y configuration and one in which the edges of the true vocal folds remained straight. They could find no consistent relationships among glottal configuration, vowel type, and effort level. Although “forced” whisper often resulted in increased constriction at the supraglottic level, this did not necessarily result in increased true vocal fold contact. More often than not, subjects demonstrated increased glottal opening with high-effort whispering compared with a quiet whisper. Of course, “increased glottal opening” could mean compression of the true vocal folds anteriorly with a posterior chink. Four of the 10 subjects, however, had no vocal fold contact with either type of whisper. These 4 patients had a straight-edged configuration to the vocal folds.[5]

The only variable that had a significant effect on laryngeal configuration in the study of Solomon et al was the subject, which suggests that generation of a whispered voice depends on each speaker, and consistent patterns might not be discernible across a larger subject population. The authors concluded that whisper might not always be harmful and might be a useful technique for select patients.[5]

The generation of whisper is likely a consequence of changes in airflow in addition to changes in laryngeal configuration and resistance. Monoson and Zemlin[2] demonstrated increased peak flow rates with whisper compared with voiced speech. Quiet whisper resulted in airflow rates twice as great as normal speech, and forced whisper resulted in airflow rates three times as great.

Our study certainly suggests that, in most cases (69%), whispering seems to cause more severe supraglottic hyperfunction. In addition, 86% of patients had some degree of true vocal fold contact during whispering and normal voice. Increased supraglottic function in such patients is likely to be traumatic to the vocal folds.

The most common glottal configuration during whisper in our study was contact involving the anterior and middle thirds of the glottis, which included the striking zone. This configuration is equivalent to the inverted-Y or “toed-in” configuration described in earlier reports.[2,5] Most subjects in our study had contact along the entire glottis during normal

speech. Regardless of the normal speech configuration, however, 81% of these subjects had worse supraglottic hyperfunction during whisper concurrent with the toed-in glottis. Only 6% had decreased supraglottic hyperfunction, and 13% had no significant change in supraglottic configuration. The inverted Y typically seems "pressed" anteriorly and most likely results from excessive anterior compression during glottic closure.

Overall, 11% of patients with true vocal fold contact during whisper demonstrated an improvement in apparent supraglottic constriction, whereas 12% had no change. The effect of these behaviors on vocal fold contact forces remains unknown (particularly if the true vocal fold configuration is no different than with normal speech), but the possibility that whispering is beneficial in these patients merits consideration.

Fourteen percent of patients had no vocal fold contact during whisper. A total of 79% of these patients had either no change or an improvement in supraglottic hyperfunction. This result is in stark contrast to the rest of the subject population in which 77% had more severe hyperfunction. If the vocal folds do not make contact with each other, particularly when there is less or the same amount of supraglottic constriction, whispering might not be more harmful than normal voice and might result in less trauma to the true vocal folds.

We obtain LEMG on all patients in whom we suspect paresis on physical examination. Although one might assume that the vocal folds of patients with paresis might be more likely to remain apart during whisper, this was not the case. Although 2 patients had an immobile vocal fold, the other 12 without vocal fold contact during whisper had only mild-to-moderate paresis. Six of these 12 patients (50%) had paresis of both superior laryngeal nerves. However, this result did not differ significantly from the rest of the subject population (48%) with bilateral paresis. The severities of the pareses were similar to those found in the rest of the subject pool.

The placement of patients on voice rest is not benign. Many patients find it difficult and frustrating. Many patients try to mouth words or do "silent talking," which often results in forced adduction of the vocal folds despite not generating sound. If a patient does not make vocal fold contact during spontaneous whispering, allowing him or her to whisper during vocal rest might be reasonable. This action could cause the patient less emotional strain and protect the vocal fold edges. Studies have suggested that whispering can be an effective communication tool.[6–8] Emotions can be conveyed through a quiet whisper.[8]

Whispering could conceivably even be therapeutic for selected hyperfunctional voice disorders. If a patient naturally has less supraglottic hyperfunction during whispering, exercises could be developed to try to help him or her recognize differences in technique and sensation, and to try to "mimic" the apparently healthier whispering technique with the spoken voice. Others have suggested the therapeutic potential of whispering in previous studies.[5,9]

Of course, further research is needed to establish the safety and efficacy of whispering before deeming it safe and to determine criteria that indicate that this approach is appropriate. Much remains to be learned. For example, lack of vocal fold contact might not necessarily result in healthier true vocal folds. Turbulent airflow might be harmful to the vocal fold edges. We did not control for effort or intensity of whisper, which certainly could affect glottal airflow, glottal resistance, and other factors that might have implications for the therapeutic potential of whisper.

Another limitation of this study was that all subjects had laryngeal pathology. Most had received more than one diagnosis (eg, paresis, laryngopharyngeal reflux, and mass). There were not enough patients with different, specific pathology to draw conclusions about whisper configurations between different subsets of patients. It would be interesting to compare whisper configurations with those of a control group, with no laryngeal pathology.

CONCLUSIONS

In most cases, whispering seems to result in laryngeal configurations that are probably more traumatic to the vocal folds than normal speech. However, in some patients, whispering does not seem to be more traumatic. In fact, in some patients, it might be healthier to the true vocal folds than normal speech. Comparisons with soft speech and "confidential

voice" have not been made, but they seem worthwhile subjects for future study. Observing how a patient whispers might be useful as part of the routine voice examination, and whispering could be considered in lieu of absolute voice rest for select patients. Further investigation is necessary.

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