Effects of unilateral vocal fold paralysis on formant frequencies: Evidence for vocal tract modifications in severely breathy voice

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ABSTRACT

Unilateral vocal fold paralysis (UVFP) affects the characteristics of the voice source by causing phonatory air leakage resulting in a breathy voice quality. Our aim was twofold. First, to evaluate the changes in formant resonance frequencies for two patients in 3 conditions : (1) normal voice before onset of UVFP, (2) voice one week after onset of UVFP caused by thoracic surgery, and (3) one week after vocal fold medialization using the technique of autologous fat injection. Second, to investigate two possible causes of formant modifications – glottal air leakage and supraglottic constriction – using an acoustic simulation of the vocal tract, to determine the effects of these two different phenomena on formant frequencies. To our knowledge, this is the first report on formant frequencies to use the patient's own normal voice before paralysis as the reference.

1. INTRODUCTION

According to the acoustic theory of speech first described by G. Fant [1], the vocal tract acts as a filter to modify the sound produced at the glottic source. Formants are resonances produced in the vocal tract and determine the different characteristics of the sounds of speech. In this study, we will be concerned with the first two formants which we will refer to as F1 and F2. In a general sense and in normal subjects, each formant frequency results from the entire vocal tract shape, but F1 is mainly affected by the mandibular position and with the cranio-caudal position of the tongue (F1 is lowered by opening the jaw and/or by lowering the tongue). F2 is affected by the anterior-posterior position of the tongue constriction (F2 is lowered by an oropharyngeal constriction as opposed to an anterior oral constriction). Both formants are lowered by a lengthening of the vocal tract [2].

Further modifications of formant frequencies are provided by the addition of other resonant cavities, such as the nasal sinuses, which "side branch" from the main pharyngeal-oral vocal tract. In breathy voice, the trachea has also be alikened to a "branch" of the vocal tract [3]. Resonances from these branches interact with the resonances from the main vocal tract (this is called acoustic coupling) to modify the frequency of the formants before coupling and also to give rise to new formants originating in the coupled cavity [3].

UVFP provokes a glottal air leak on phonation due to the absence of adduction of the paralyzed vocal fold, leading to a breathy voice quality. In UVFP, supraglottic constriction, also called ventricular dysphonia [4,5], may be observed, and may be present to some extent in all cases, possibly as a means of providing additional resistance in a attempt to increase transglottic pressure across the glottal gap. For these reasons, some modification of formant frequencies seems to be expected after onset of UVFP.

The object of this study was twofold : i) to demonstrate modifications of formant frequencies after onset of UVFP and to test whether surgically correcting the phonatory glottal gap corrects these modifications; and ii) using and acoustic simulator of the vocal tract, to determine if a change in glottal opening area or in supraglottic constriction size could account for the formant modifications observed in our patients.

2. MATERIALS AND METHODS

The isolated vowels [a] and [i] in comfortable phonation and in a read text ("Grand-mère raconte," a French equivalent of "The Rainbow Passage") were recorded for two patients in 3 conditions : (1) normal voice before onset of UVFP, (2) voice one week after onset of UVFP caused by thoracic surgery and (3) voice one week after vocal fold medialization using the technique of autologous fat injection [6]. Recordings were made in a quite room on a SONY DTC-60ES Digital Audio Tape deck with a sampling rate of 48 kHz. Mouth to microphone distance was 15 cm. Fiberoptic transnasal videolaryngoscopy was performed for all cases. After onset of UVFP, patient 1 presented with type I muscle tension dysphonia [4], which consisted in a medialization of the ventricular folds. After vocal fold medialization, this supraglottic constriction was no longer observed. Patient 2 had no visible muscle tension dysphonia at any time.

For each condition and for each patient, F1 and F2 for [a] and [i] in isolation were measured manually from linear prediction coding (LPC), based on an average of 25 short-time Fourier spectra (for a total duration of 200 ms)

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calculated with a hamming window after resampling at 16 kHz. For [a] in running speech a 40 ms mid-vowel segment from the French word "village" was analyzed in the same way. For the vowel [i] a mid-vowel point was chosen in the French word "tic." Snorri, a signal processing software designed by Y. Laprie and V. Colotte was used for all of the spectral calculations [7].

One acoustic model of the vocal tract [8] was then used to calculate F1 and F2 for [a] and [i] in a "standard" vocal tract configuration, corresponding to a normal vocal tract and closed glottis. Then varying constrictions at 1 and 2 cm from the glottis were applied and the new values of F1 and F2 noted. A second acoustic model of the vocal tract was employed, first with the glottal area set at 0 cm² and then with the glottal area set at .2 cm² , to simulate an open glottis situation. F1 and F2 for each vowel were noted in each case.

3. RESULTS

The results are shown in tables 1 through 4. F1 increased in all cases after onset of UVFP, of an average of 217 Hz. F2 increased in the stationary vowel setting and for [a] in the text for patient 1. These changes were improved by the surgical injection of autologous fat in most cases. The simulated effect of the supraglottic constriction was an increase in F1 and F2 for [a] only. The effect of increased glottal area was an increase in both formants for both vowels.

	F1			
	Patient	**NL**	**UVFP**	**FAT**
[a] isolated	1	583	802	671
	2	551	761	567
[i] isolated	1	258	319	251
	2	277	371	330
[a] text	1	442	610	390
	2	563	751	453
[i] text	1	311	655	270
	2	390	839	282

Table 1: First formant frequencies (Hz) measured from isolated vowels [a] and [i] and from the same vowels in text. NL : normal voice. UVFP : voice after onset of unilateral vocal fold paralysis. FAT : voice after injection of autologous fat into the paralyzed vocal fold.

	F2			
	Patient	**NL**	**UVFP**	**FAT**
[a] isolated	1	1196	1259	1188
	2	1156	1172	1187
[i] isolated	1	2080	2353	1967
	2	2180	2218	2178
[a] text	1	1478	1645	1635
	2	1692	1631	1525
[i] text	1	2043	1961	1842
	2	2270	1875	1960

Table 2: Measured second formant frequencies (Hz). NL : normal voice. UVFP : voice after onset of unilateral vocal fold paralysis. FAT : voice after injection of autologous fat into the paralyzed vocal fold.

	Area at 1 cm above glottis	**Area at 2 cm above glottis**	**F1**	**F2**
Standard [a]	**2.3**	**1**	**681**	**1238**
	1	.5	681	1238
	1	1	681	1300
	.5	1	715	1300
	.5	.5	712	1331
Standard [i]	**3**	**2**	**248**	**2260**
	1	2	248	2260
	3	1	248	2229
	1	1	248	2321
	.5	.5	248	2352

Table 3: Formant calculations (Hz) with varying supraglottic constrictions (area in cm²).

Vowel	**Area of glottis (cm²)**	**F1**	**F2**
[a]	0	664	1240
[a]	.2	880	1480
[i]	0	232	2250
[i]	.2	300	2352

Table 4: Formant calculations (Hz) with varying glottal area.

4. DISCUSSION

Our results confirm that formant modifications occur after the onset of UVFP in the absence of voluntary or imposed vocal tract modifications, and that surgically correcting the phonatory glottal gap corrects these modifications. These results along with the results of the acoustic simulations imply that the increased glottal gap itself may be implicated in the observed formant changes, as shown in table 4. These results also imply that supraglottic constriction may also intervene, as seen in table 3. The measured second formants did not undergo a large modification for the vowel [a], which may be expected as F2 for [a] is affiliated with the configuration of the front or oral cavity, whereas for the [i] in isolation the increase in

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F2 with onset of UVFP is compatible with a modification of the pharyngeal cavity by a supraglottic constriction (tables 2 and 3). The vowel [i] in the text did not undergo the same modification. This may be due to effects of coarticulation, to vowel undershooting in the text or to a larger test-retest variability for formant frequencies in text.

According to the acoustic theory of speech first described by G. Fant, the vocal tract acts as a filter to modify the sound produced at the glottic source [1]. Formant frequencies thus depend on the vocal tract configuration : its length, the patient's age, height, weight, sex, and certain cultural and linguistic settings [9]. From an acoustic standpoint, based on the model of linear propagation of sound within the vocal tract, other physical attributes such as the addition of nasal (or tracheal) "side branches" or vocal tract wall rigidity intervene. Finally, formant frequencies are subject to the same within-subject day-to-day variability as other vocal characteristics.

Our study was constructed in such a way as to eliminate the variability due to differences between individual patients: each patient was his own "control." Test-retest variability was unavoidable in this case, however, and may have influenced the results.

Other vocal tract modifications could be in play. A higher base of tongue tongue position used to counter the high phonatory airflow in UVFP may explain the decrease in F2 for the vowel [i] in the text setting. An additional "tracheal formant" has been described [3] situated at around 2100 Hz. This supplementary formant could affect F1 and F2 by raising their frequencies (in the case of /a/) or by lowering F2 (in the case of /i/). Tracheal coupling may vary in amplitude and frequency depending on the glottic gap in UVFP and on the anatomical characteristics of the trachea itself. Modifications in vocal tract wall rigidity, as a reaction to high airflow in the vocal tract, could also modify acoustic absorption and reflexion and thus modify the resonant frequencies of the vocal tract. Thus, the resonant modifications observed are probably due to a complex combination of physiologic and aerodynamic phenomena including supraglottic constriction, tracheal coupling, pharyngeal and oral cavity configurations and eventually modifications of the vocal tract wall rigidity.

These observations lead us to conclude that measurements based on formant frequencies or on inverse filtering in UVFP may give erroneous results. Other forms of dysphonia may also provoke involuntary vocal tract modifications or source-tract interactions. Further studies of different types of dysphonia, before and after treatment, are warranted.

5. CONCLUSIONS

Modifications of the voice source by UVFP lead to changes in measured formant frequencies. These formant modifications can be primarily explained by an incomplete glottal closure and, to a lesser extent, by a change in the vocal tract configuration by supraglottic constriction. The formant modifications were restored by correction of the glottal gap using the technique of vocal fold injection of autologous fat. Interaction between voice production at the glottal level and the vocal tract itself affect speech formant frequencies in UVFP. These modifications need to be characterized more fully and taken into account when analyzing objective measurements of speech and voice in UVFP and possibly in other types of breathy dysphonia.

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