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Laryngeal Paralyses: Theoretical Considerations and Effects on Laryngeal Vibration

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The neurological causes of vocal fold paralyses have been well documented. However, the effect of these disorders on laryngeal vibration is not well understood. A theoretical four-mass model of the larynx, based on the work of Ishizaka and Isshiki (1976) and Koizumi, Taniguchi, and Hiromitsu (1987), was developed and adapted to simulate laryngeal biomechanical behavior. The model was used to evaluate various states of asymmetric laryngeal vibration. Input parameters that relate observed laryngeal function and model simulation were developed. Laryngeal paralyses were simulated by their predicted effect on these parameters. Simulations were compared with available data on glottal vibration in laryngeal paralyses. Complex modes of vibration were seen with certain combinations of asymmetrical lower mass stiffness and initial glottal gap.

KEY WORDS: laryngeal vibration, laryngeal paralysis, theoretical models, diplophonia

Vocal fold paralyses occur frequently, and their neuropathology has been described in detail (e.g., Rontal & Rontal, 1986). However, the effects of such paralyses on laryngeal vibration are not well understood. In this study a well-known theoretical model of laryngeal function was used to investigate the vibratory and phonatory patterns associated with laryngeal paralyses.

Theoretical models of vocal fold vibratory behavior have been developed from knowledge of acoustics and laryngeal biomechanics and have been used to describe vocal fold vibration. The best known of these is the two-mass model of Ishizaka and Flanagan (1972), based on theoretical work of Ishizaka and Matsudaira (1972). The two-mass model is a mechanical simulation of the observed phase difference between the lower and upper margins of the vocal folds during phonation. Stevens (1980) described the significance of the two-mass model in advancing our understanding of laryngeal function. He pointed out that if the vocal fold is represented as a lumped mass model of coupled masses and springs, a significant transfer of energy from pulmonary air flow to acoustic vibration is achieved by motion of the upper and lower edges of the fold that are out of phase. This model has been used in speech synthesis and analysis of phonation, and has served as a standard of comparison for other phonatory models (Conrad & McQueen, 1988; Titze & Talkin, 1979). Further refinements of the two-mass model by Koizumi (Koizumi, Taniguchi, & Hiromitsu, 1987, 1989) to include vertical as well as horizontal displacements have yielded perceptually more natural-sounding synthetic speech, as well as a more realistic representation of vocal fold motion.[1]

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[1]In addition to "lumped mass" models like the one used here, a "continuum" model has been developed by Titze and Talkin (1979) The continuum model attempts to define and model tissue mechanics as well as

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Models of laryngeal vibration generally have been developed for use in computer speech synthesis applications. However, there have been a few applications to the study of pathological phonation. Ishizaka and Isshiki (1976) and Isshiki, Tanabe, Ishizaka, and Broad (1977) expanded the two-mass model to study vibration with asymmetrical variations in tension, resting glottal gap, and subglottal pressure. They described three basic vibratory modes on the basis of observations of laryngeal vibration made with a high-speed camera in excised canine and human larynges. At a small resting gap, the vocal cords vibrated at the same frequency, but the tense cord preceded the lax one in phase. This was termed Type I vibration. With larger resting gaps, the Type II pattern displayed alternating large and small amplitudes in an irregular or "dicrotic" pattern. At wide resting gaps, periodic vibration returned without the cords meeting at midline; this was termed Type III vibration. In these experiments, the resting glottal area was a crucial variable in determining the type of vibratory pattern, though other variables, such as subglottal pressure, tension factors, damping ratios, and stiffnesses of the vocal cords, were also important.

Although these three basic vibratory patterns have been described, they have not been well documented in humans with unilateral laryngeal paralysis. Three general categories of laryngeal paralysis have been distinguished on the basis of which nerve innervating the larynx is injured (Ward, Berci, & Calcaterra, 1977). *Recurrent laryngeal nerve* injury affects the endolaryngeal musculature, including adductors (thyroarytenoid muscle, lateral cricoarytenoid muscle, interarytenoid muscle) and abductors (posterior cricoarytenoid muscle). The affected vocal fold is immobile, in the "paramedian" position or just off midline. The voice is often breathy or hoarse initially. With time, some compensation generally occurs to yield a more normal speaking voice, but loss of ability to sing persists. *Superior laryngeal nerve* paralysis affects the cricothyroid muscle. Its manifestations are subtle: The voice generally sounds normal in the modal pitch range but is notably restricted at higher levels of F_0 (e.g., in singing). The glottis appears normal at rest; however, during phonation the affected side appears slightly flaccid, the anterior commissure rotates toward the intact side, and the posterior commissure rotates toward the affected side. If the internal branch of the superior laryngeal nerve is also injured, sensation is altered and aspiration may occur during swallowing. Injury to the *vagal nerve* supplying both the recurrent and laryngeal nerves generally results in more severe symptoms. Aspiration and swallowing difficulties may be present. The voice is weak and breathy. The affected vocal fold is immobile and laterally located, with a wide glottal gap present during attempts at phonation.

Some investigators (e.g., Woodson, 1990) have called into question theories relating the position of the vocal cord to the type of laryngeal paralysis, pointing to the wide variability in degrees of paralysis, vocal cord atrophy, and compensatory mechanisms. Only a few reports have specifically described vocal fold vibratory patterns associated with laryngeal paralyses. The Isshiki Type I vibratory pattern (Isshiki et al., 1977) and phase differences between cords were observed stroboscopically in humans with unilateral superior laryngeal paralysis (Arnold, 1961) and in the in vivo canine model (Tanabe, Isshiki, & Kitajima, 1972). However, vibration in more common recurrent and vagal (combined superior and recurrent) paralyses has not been well studied.

Computer modeling of laryngeal vibration, unlike studies in humans, allows systematic, independent manipulation of various parameters, so cause and effect relationships among variables can be examined in detail. Such modeling is a powerful tool for studying the effects of paralyses on laryngeal vibration. Wong, Ito, Cox, and Titze (1991) recently reported simulation of pathological phonation with a multiple-mass model, a hybrid of those developed by Ishizaka and Flanagan (1972) and Titze (1973). Wong et al. (1991) described perturbed vibrations resulting from both local and nonlocal parameter changes in stiffness and mass. This model revealed the existence of subharmonics in the output waveforms, due to complex oscillation that repeated periodically. This pattern is similar to the Type II vibration seen by Isshiki et al. (1977), which they termed "dicrotic" or "tricrotic." These vibratory modes were particularly manifest with decreases in longitudinal tension (model parameter T_{act}) to simulate "floppy" cords. Wong et al. attributed the observed vibratory patterns to nonlinear stiffness changes at large amplitudes of displacement.

The present study describes simulations of pathological laryngeal vibration generated with a currently available theoretical model of the larynx and compares results with observed pathological phonation in humans. Several limitations should be noted at the outset. First, model limitations may make it difficult to relate theoretical models directly to the pathological larynx. Second, comparison sets of objective measures and descriptions of vibratory patterns in the pathological larynx are not readily available from the literature. This study compares data from theoretical modeling and clinical investigation, given constraints of current knowledge. By systematically varying model parameters during simulations and then comparing results to vibratory patterns observed in patients with unilateral laryngeal paralyses, we attempted to determine which parameters are important for describing vibration occurring with asymmetrical laryngeal tension.

Method

Theoretical Model of the Larynx

We chose to use the Koizumi modification of the two-mass model of the larynx (Koizumi et al., 1987, 1989). In this model the upper mass is coupled solely to the lower mass, rather than doubly coupled to the side wall and lower mass (Figure 1). This arrangement appeared to be a more realistic representation of the structure and motion of the upper and lower margins of the vocal folds than the original two-mass model. The Koizumi model also allows for vertical displacement of the cords. Vertical cord position is believed to be of impor-

aerodynamics (Cooper, 1988), in order to follow Hirano's functional description of the "body-cover" relationship of the vocalis muscle and vocal ligament to the overlying mucosa (Hirano & Kakita, 1985)

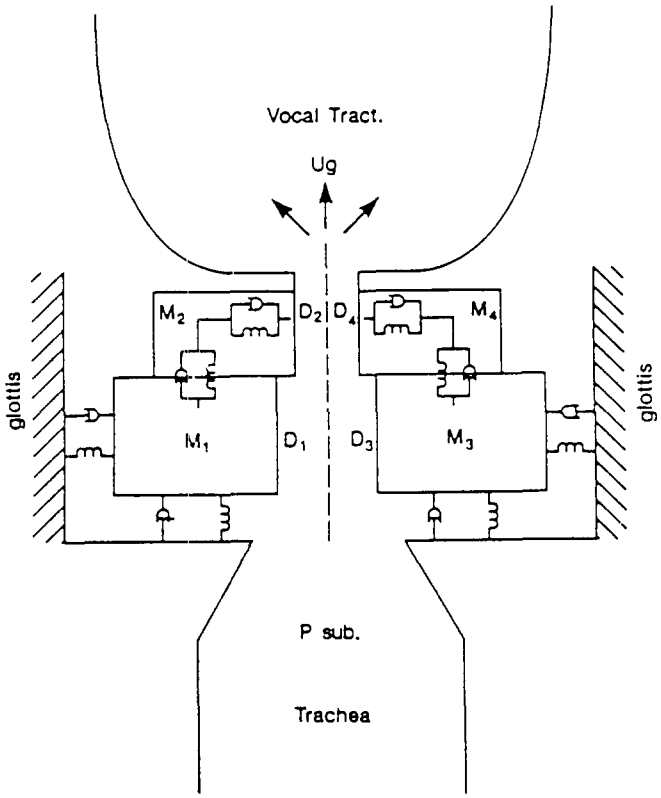

FIGURE 1. The four-mass model of larynx and vocal tract, after Koizumi et al. (1987, 1989). Note that each upper mass is coupled only to the lower mass, and not to the side wall (as is the lower mass). D = vertical distance; M = mass of each element; U_g = glottal flow; P_{sub} = subglottal pressure.

tance in evaluating laryngeal paralyses (Isshiki & Ishikawa, 1976).

The two-mass model is limited in certain respects for our purposes. It has been described as a "cover-only" model (Fujimura, 1988), which may limit the extent to which large tension asymmetries can be simulated. The glottal orifice is rectangular, and the masses are either in contact or apart. These restrictions limited the modeling of anterior-posterior defects in glottal closure, such as bowing. The current model has constraints in calculating pressure drops across the glottis and the resulting glottal air flow, because the interface between the upper and lower masses on each side must be on the same horizontal plane. This imposed vertical symmetry limited modeling of vocal folds on different levels and prevented the use of vertical dimension asymmetry simulation in the model. Such asymmetries may occur with superior and recurrent laryngeal nerve paralyses, for example.

The vocal tract was simulated by four concatenated cylinders with equivalent acoustical resistive, inductive, and capacitative losses (Ishizaka & Flanagan, 1972), shaped to simulate the vowel /ə/. The Koizumi model was expanded to simulate four-mass motion of the larynx (upper and lower margins of each vocal fold), rather than the usual two-mass motion (upper margins of both vocal folds and lower margins of both folds), allowing the independent variables of the model for each mass to be changed asymmetrically. Equations relating the interaction of forces during collision of the masses upon glottal closure were followed (Ishizaka & Isshiki, 1976). Pressure drops through the larynx were applied, following van den Berg (see Koizumi et al., 1987). Through each portion of the larynx, a drop in pressure was calculated by application of the Bernoulli equation, considerations of acoustical resistive and inductive losses through the glottis, and the conservation of momentum principle. With the subglottic pressure and glottal geometric dimensions known, these equations were solved to calculate the glottal flow. Equations of motion were then written for the masses using the known driving forces of the subglottic pressure and forces of the connected springs and dampers (Koizumi et al., 1989). The input variables included the masses and their associated spring and damping constants, subglottic pressure (P_{sub}), and the distance between the masses, termed the vibratory gap (A_{go} in previous studies). Initial values for these variables were taken from Koizumi et al. (1987). These differential equations were solved using the finite difference method, with a selected sampling interval to determine changes in displacement x and y for each mass. Mass displacement changes altered glottal geometry, leading to a new calculation of glottal airflow, and so on, in an iterative fashion.

The model was programmed in Fortran on an IBM-AT-compatible computer. Simulations were calculated for at least 400 msec to ensure the observation of steady-state oscillation.

Model Input Parameters

Model parameters are listed in Table 1. Parameters G, Q, P, and T served as input variable combinations for modeling laryngeal paralyses with the four-mass model. Parameter G measures the gap between the vibrating masses during phonation, previously termed A_{go} by some investigators. G corresponds well to glottal gap or chink, and an increase in this parameter has been associated with perception of breathy voice quality (Ishizaka & Isshiki, 1976; Isshiki, 1980).

Parameter Q was modified from the Ishizaka and Flanagan (1972) two-mass model. In previous studies this parameter was treated as analogous to cricothyroid tension (Ishizaka & Isshiki, 1976; Isshiki et al., 1977), and decreases in mass and thinning of vocal fold mucosa during cricothyroid contraction have been observed (Hirano, 1974). In our study, we applied Q to the Koizumi model much as Ishizaka & Flanagan (1972)

TABLE 1. Four-mass model simulation parameters.

Four-mass model parameters	Mathematical variables*	Laryngeal function paradigm
G	A_{go}	Vibratory gap
Q	$k_{x2,4}$, M (mass), D (vertical distance)	Cricothyroid contraction
P	$k_{x1,3}$, P_{sub}	Vocalis contraction and subglottal pressure
T	$k_{x1,3}$	Vocalis tension

*From Koizumi et al. (1987).

did, except that it was multiplied by k_x (spring stiffness) of only the upper, and not the lower, mass spring.

Parameter P is related to k_x of the lower masses and subglottic pressure P_{sub}. This is analogous to vocalis muscle contraction, so that as stiffness of the lower masses increased, subglottic pressure rose accordingly. Results of recurrent laryngeal nerve stimulation in the in vivo canine model support this subglottic pressure/stiffness relationship (Moore & Berke, 1988), although the canine model used a constant flow source.

Parameter T is related solely to lower mass spring stiffness k_x, without associated subglottal pressure rise. This reflects vocalis muscle tension exerted by longitudinal stretching, without changes in mass or stiffness that would affect subglottic pressure. Isshiki et al. (1977) observed that the stiffness and tension parameters of two-mass model simulations are not directly comparable to the biomechanical effects of laryngeal musculature. In our model, as in theirs, the stiffness parameters govern lateral displacement, not longitudinally oriented forces.

Objective Measures of Vibration

To study the output of our simulations, we used a number of objective measures of vocal fold vibration, including F_0 and mean airflow rate at the glottis. In addition, the open quotient and speed quotient were determined from the glottal area waveform, when possible. The location of collision of the lower masses relative to midline at the time of glottal closure was measured. A spectral analysis of the output flow derivative waveform (analogous to output sound pressure; Ishizaka & Flanagan, 1972) was performed as well. The H1 − H2 difference was calculated from the spectral analysis for each case. H1 − H2 is defined as the difference in energy between the fundamental frequency and the second harmonic, in dB (e.g., Huffman, 1987).

EXPERIMENT 1

Method

This experiment included five simulations that examined the extent to which varying the four input parameters (Table 1) mimicked different laryngeal paralyses.

The first simulation in this set represented normal symmetrical oscillation. Parameter G was set at .02 cm^2, P_{sub} was set to 8 cm H_2O, $k_{x1} = k_{x3}$ = 60,000 kdyne/cm, and Q was set such that $Q_r = Q_l = 1.1$. The second simulation modeled a right superior laryngeal nerve paralysis by decreasing Parameter Q on the right side (Q_r) from 1.0 to 0.7, representing reduced cricothyroid tension. The third simulation modeled a right-sided recurrent laryngeal nerve paralysis by increasing Parameter G (A_{g0}) from .02 cm^2 to .12 cm^2 to simulate an increased glottal gap, and by decreasing Parameter P (k_{x1} from 60,000 to 15,000 kdyne/cm, P_{sub} from 8.0 to 6.0 cm H_2O). These changes simulated reduced subglottal pressure and reduced vocalis stiffness on the paralyzed side. Finally, two types of right vagal nerve paralysis (combined superior and recurrent nerve paralysis) were simulated. An acute unilateral vagal paralysis was modeled with changes in G (A_{g0} to .12 cm^2), P (k_{x1} to 15,000 kdyne/cm, P_{sub} to 6.0 cm H_2O), and Q (Q_r to 0.7). Lastly, we modeled a unilateral vagal

TABLE 2. Model parameter settings for five simulated laryngeal states.

Simulation	Parameter	Value
Normal phonation	G	0.02 cm^2
	P_{sub}	8 cm H_2O
	k_{x1}	60,000 kdyne/cm
	k_{x3}	60,000 kdyne/cm
	Q_r	1 0
	Q_l	1 0
Superior laryngeal nerve paralysis	G	0.02 cm^2
	P_{sub}	8 cm H_2O
	k_{x1}	60,000 kdyne/cm
	k_{x3}	60,000 kdyne/cm
	Q_r	0 7
	Q_l	1.0
Recurrent laryngeal nerve paralysis	G	0 12 cm^2
	P_{sub}	6 0 cm H_2O
	k_{x1}	15,000 kdyne/cm
	k_{x3}	60,000 kdyne/cm
	Q_r	1 0
	Q_l	1 0
Acute vagal paralysis	G	0.12 cm^2
	P_{sub}	6.0 cm H_2O
	k_{x1}	15,000 kdyne/cm
	k_{x3}	60,000 kdyne/cm
	Q_r	0 7
	Q_l	1 0
Chronic "compensated" vagal paralysis	G	0.12 cm^2
	P_{sub}	6.0 cm H_2O
	k_{x1}	15,000 kdyne/cm
	k_{x3}	60,000 kdyne/cm
	Q_r	0 7
	Q_l	1.3

TABLE 3. Objective measures of pathologic phonation from four-mass model simulations.

Simulation	F_0	OQ	SQ	MFR	H1−H2
Normal	144	.68	1.12	219	−0.45
SLN paralysis	128	.67	1.45	259	1 8
RLN paralysis	98.5	77	0.88	389	6 1
Acute vagal paralysis	68	.80	1.05	433	2.6
Chronic vagal "compensated" paralysis	66	81	1.81	447	−4.8

Note. SLN = superior laryngeal nerve; RLN = recurrent laryngeal nerve, F_0 = fundamental frequency in Hz; OQ = open quotient; SQ = speed quotient; MFR = mean flow rate in cc/sec; H1 − H2 = spectral difference in dB

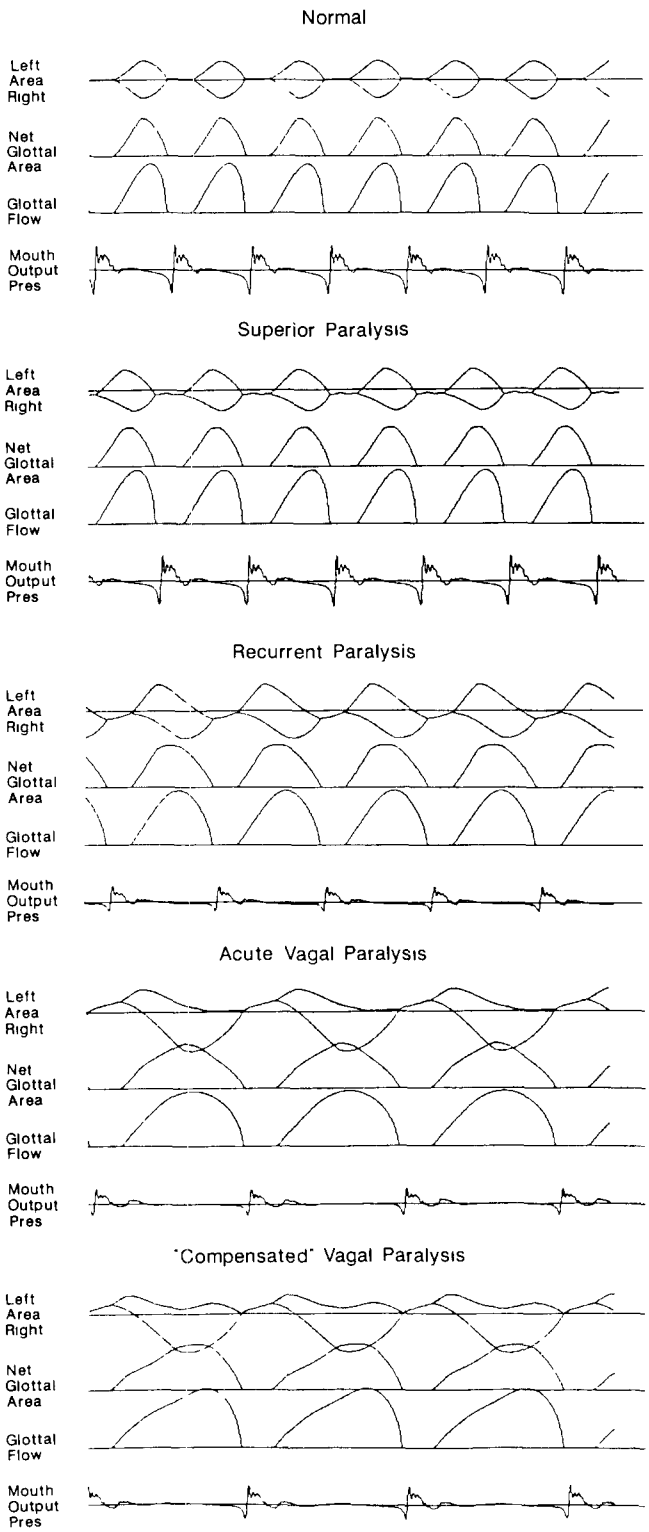

FIGURE 2. Model simulations of laryngeal paralyses with time domain display of glottal area with left and right (paralyzed side in the simulation) mass displacements, net glottal area and glottal flow, and oral output pressure. Top tracing: Normal phonation. Second tracing: Simulated superior laryngeal nerve paralysis. Third tracing: Simulated recurrent laryngeal nerve paralysis. Fourth tracing: Simulated acute vagal paralysis. Bottom tracing: Simulated chronic vagal paralysis with compensation by the unaffected side. See text for discussion.

paralysis with attempts at compensation on the intact side. In this case, Q was increased on the left side (Q_l to 1.3), corresponding to an attempt by the intact cricothyroid muscle to compensate for the flaccid paralysis. These parameter settings are summarized in Table 2.

Results and Discussion

Table 3 lists objective measures of phonation for the five simulations, and Figure 2 shows tracings taken from the simulations. For all five conditions, the tracings indicate right and left displacement of the upper and lower masses, glottal area, glottal flow, and oral output sound pressure. The first tracing in Figure 2 represents normal symmetrical oscillation. The second tracing displays results for the simulation of a right-sided superior laryngeal nerve paralysis. A slight drop in F_0 and shift from midline in the displacement curves were seen, but the glottal area and flow waveforms were not noticeably different from (modeled) normal phonation. The third tracing represents a right-sided recurrent laryngeal nerve paralysis. Note the shift in closure to the right of midline in the displacement curves, a drop in frequency, a shift in peak area to the left, and less two-mass motion of the right side.

The final two tracings in Figure 2 represent the two types of simulated right vagal nerve paralysis (combined superior and recurrent nerve paralysis). The fourth (an acute unilateral vagal paralysis) shows marked motion of the "flaccid" fold across the midline and poor vibration of the "intact" side. This pattern of vibration in vagal paralysis compares well to findings for unilateral vagal paralysis in the in vivo canine model (Trapp & Berke, 1988). In contrast, the fifth tracing shows a simulated unilateral vagal paralysis with attempts at compensation on the intact side. The frequency of oscillation of the left masses increased with Parameter Q. This resulted in a net glottal area change that shifted the peak glottal area to the right and increased the speed quotient. This increased speed quotient is similar to findings in patients with a vagal section (Hanson, Gerratt, Karin, & Berke, 1988). The mean flow rates were similar for both vagal paralysis simulations but were elevated relative to the normal case. The low F_0 observed in both cases is probably related to the effect of Parameter Q: As cricothyroid tension decreased, vocal cord mass per unit area increased, markedly influencing the speed of oscillation (because F_0 is proportional to $(k/m)^{1/2}$). Note that simulation of cricothyroid function by the four-mass model may be imprecise: As mentioned above, model parameters govern lateral, but not longitudinal, forces. Thus, had we modelled a compensation that closed the large resting gap (for example, by increasing lateral cricoarytenoid contraction on the unparalyzed side), our results would have shown an increased F_0.

During these simulations with the four-mass vocal tract model, different steady-state oscillation patterns appeared, during which the vibratory pattern repeated itself over two or more cycles. The output resembled the Type II glottal vibrations described by Isshiki et al. (1977) and simulated by Ishizaka & Isshiki (1976). It contained several fairly symmetrical cycles of oscillation followed by an intervening abnormal

compensating cycle, with the entire pattern repeated periodically. When viewed in the time domain, we call these patterns *supraperiodic,* because the repeating vibratory pattern (viewed as a waveform) consists of more than one glottal cycle. [Other authors (e.g., Wong et al., 1991) have noted these patterns in the frequency domain and have labelled them *subharmonic.*]

Supraperiodic vibratory patterns have been previously observed in superior laryngeal nerve paralysis (Hirano, 1975; Hanson et al., 1988), in approximately 20% of patients with dysphonia associated with Parkinson's disease (Gerratt, Precoda, & Berke, 1990), in peripheral neuropathy (Hanson, Gerratt, & Ward, 1983), and in vocal hoarseness (Monsen, 1981). Supraperiodic vibration has also been observed in normal speakers. For example, Klatt and Klatt (1990) reported "diplophonic double pulsing" as a temporal variant pattern in more than a quarter of the normal speakers they evaluated; and Rose (1988) reported that "diplophonia" (dicrotic and tricrotic vibration) is used phonemically in certain Chinese dialects. Experiment 2 examined the conditions underlying these vibratory patterns in more detail.

EXPERIMENT 2

Method

In a second set of simulations we attempted to systematically determine the initial conditions eliciting supraperiodic patterns of vibration. These simulations examined the effect of initial glottal gap (Parameter G) on a simulated recurrent nerve paralysis. G was increased incrementally from an initial value of 0.02 cm^2 until the masses were completely apart (open quotient = 1.0). Vibratory patterns were evaluated for two values of P_{sub} (4 and 6 cm H_2O). Other model parameters were set as for the simulated recurrent nerve paralysis described above.

Results and Discussion

Figures 3 and 4 show the results of changes in G (the initial glottal gap) on a simulated recurrent nerve paralysis, for P_{sub} = 6 cm H_2O and P_{sub} = 4 cm H_2O, respectively. For P_{sub} = 6 cm H_2O, when G was small, symmetric oscillation occurred with collision of the masses to the side of the lower stiffness. This is similar to the Isshiki Type I oscillation. As G increased, the frequency decreased, speed quotient decreased (peak glottal area shifted to the left), open quotient increased as expected, and the collision point shifted toward the midline. At a certain initial glottal gap (about 0.088 cm^2), the oscillation became supraperiodic. Objective measures of glottal vibration could not easily be taken in these cases, but the output display resembled the Isshiki Type II vibratory pattern. Just beyond this point, as G increased further, symmetrical oscillations resumed. The speed quotient increased, with a discontinuity at this point. Further increases in G resulted in a slight increase in fundamental frequency, increased open quotient, and decreased speed quotient. When G became large enough, the masses no longer touched when oscillating, corresponding to Isshiki's Type III pattern. Additional simulations at a lower P_{sub} (4 cm H_2O; Figure 4) showed a similar pattern, with supraperiodic vibration around G = .08 cm^2, followed by resumption of symmetrical vibration with increased G.

The supraperiodic oscillation pattern can be seen in the waveforms in Figure 5, with data corresponding to the region of speed quotient discontinuity displayed in Figures 3 and 4. Recall that the model simulated right recurrent nerve paralysis with asymmetric tension (k_{x1} = 15,000 kdyne/cm) and lowered P_{sub} (6 cm H_2O). For G = .12 cm^2 (top waveform), the oscillation pattern was symmetrical with collision to the right of midline, the "flaccid" side. At G = .13 cm^2 (middle tracing), oscillations became supraperiodic; that is, a pattern of six fairly symmetrical cycles was followed by a long compensatory cycle, a pattern that repeated itself periodically. The output sounded rough, and the spectrum (on the right of the figure) showed subharmonics below the fundamental frequency. At G = .14 cm^2 (lower tracing), symmetrical oscillation resumed, with the collision point shifting to midline. Supraperiodic oscillation modes were also simulated in bicyclic or tricyclic patterns (the patterns most commonly found in dysphonic voices; see Gerratt et al., 1990) by increasing the upper mass stiffness relative to the lower mass stiffness with other parameters set asymmetrically, as described above.

General Discussion

In recent years, much discussion and review of laryngeal models has appeared (Cooper, 1988; Fujimura, 1988; Stevens, 1980). These exchanges have centered around the advantages and limitations of current models of vocal fold vibration, including their ability to accurately predict results of direct physiological experimentation and the ease with which they can be applied to clinical research (Cooper, 1988). In this regard, results of model simulations should be considered in light of the limitations of a particular model's ability to simulate both normal and pathological vocal fold vibration.

The two-mass model of Ishizaka and Flanagan (1972) has several advantages as well as some disadvantages relevant to our experimental design. It is conceptually simpler than other models because it uses a small number of variables to describe the mass motion. It has been applied in a wide variety of laryngeal and speech research settings. Because a prior version of the two-mass model had been used in simulation of pathological phonation by Ishizaka and Isshiki (1976), the present simulations could be compared with existing data. Recent improvements by Koizumi et al. (1987, 1989) allow for vertical cord motion, which we felt was relevant for simulation of pathological phonation. Unfortunately, this feature could not be investigated because of limitations in aerodynamic modeling. The two-mass model also assumes the dimensions of the glottal gap are the same throughout the vertical height of the simulated glottal duct. Model simulations with convergent and divergent duct configurations have resulted in different oscillation threshold pressures (Titze, 1988), although it is not known how these configurations affect vibratory patterns. Also, the duct config-

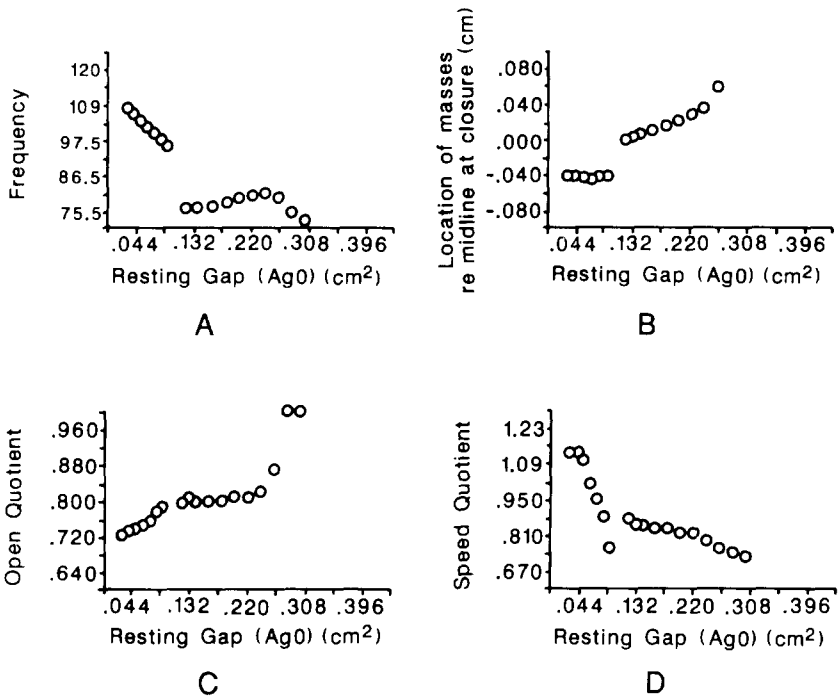

FIGURE 3. Results of simulations of unilateral recurrent paralysis varying initial glottal gap (A_{g0}) from .02 cm² until the masses were completely apart (open quotient of 1.0). For these simulations, P_{sub} = 6 cm H_2O. Negative values indicate closure to the paralyzed side of midline, and positive values indicate closure to the nonparalyzed side of midline. When the opposing masses collided at midline, the value of this position was .000 cm. A: Changes in F_0 as a function of resting glottal gap. B: Changes in the location of closure for the lower mass as a function of resting glottal gap. C: Changes in open quotient as a function of resting glottal gap. D: Changes in speed quotient as a function of resting glottal gap.

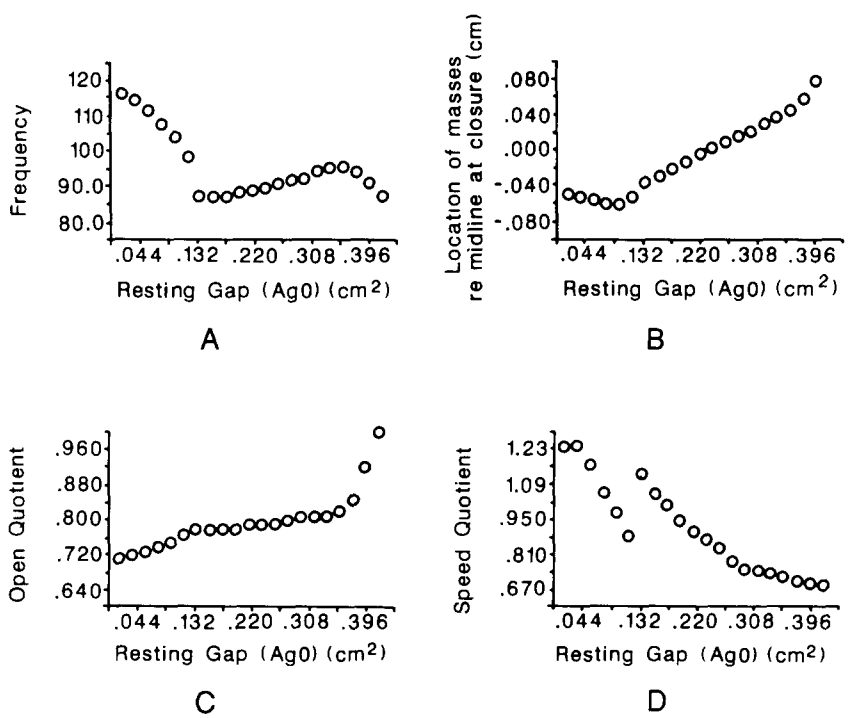

FIGURE 4. Results of simulations of unilateral recurrent paralysis, with P_{sub} = 4 cm H_2O. Other details are as in Figure 3.

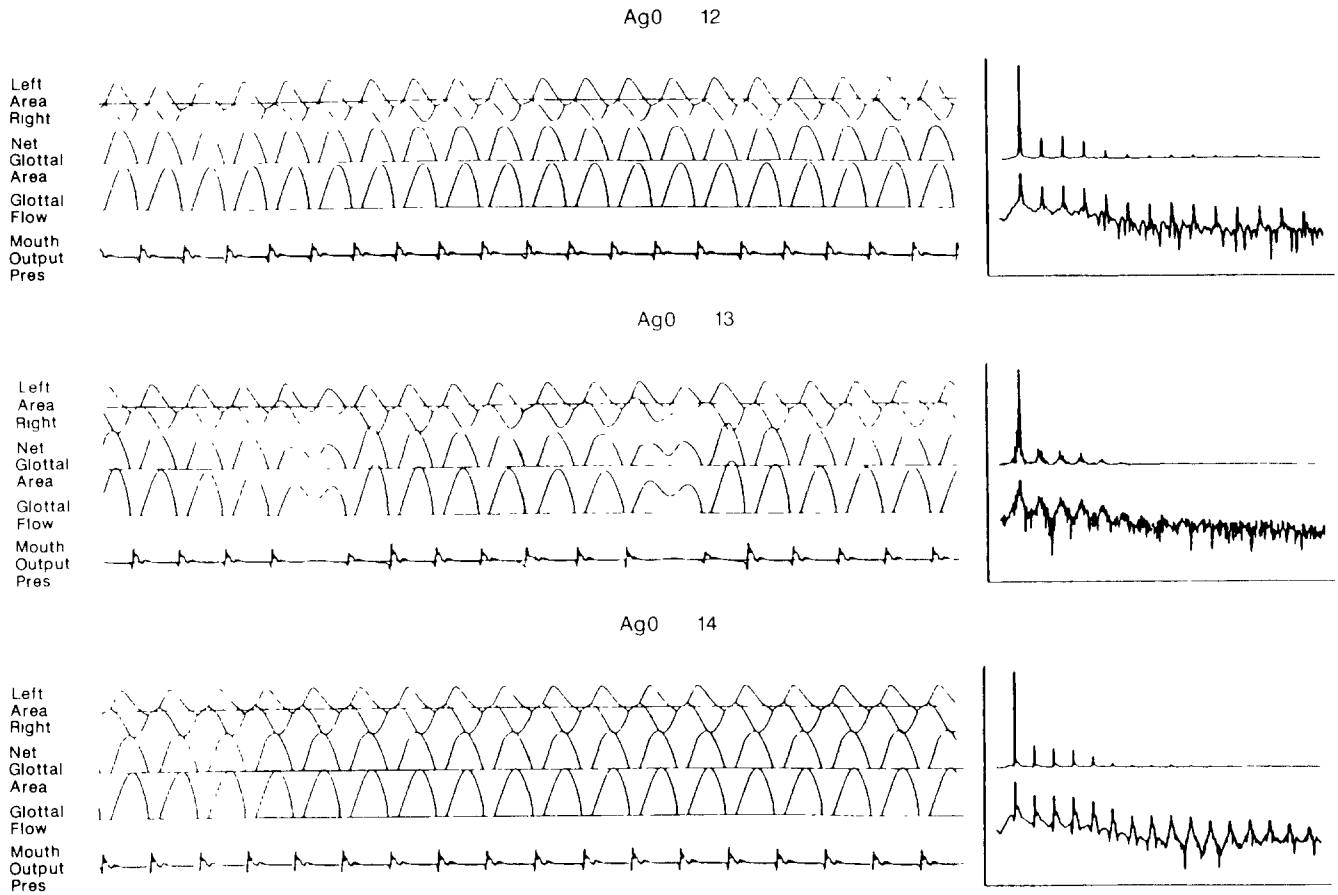

FIGURE 5. Time and frequency domain displays of recurrent paralysis simulations taken at A_{g0} of .12, .13, .14 cm^2, corresponding to the region of speed quotient discontinuity (P_{sub} = 6 cm H_2O side) in Figure 3.

urations of larynges in states of paralysis have not been well described. For purposes of comparison with other models we assumed a straight (not convergent or divergent) prephonatory vertical duct configuration.

Simulation of a right-sided superior laryngeal nerve (SLN) paralysis resulted in a shift from midline in glottal displacement curves, with little change in F_0 and no change in glottal area and flow relative to normal phonation. In the present study, SLN paralysis was simulated by decreasing Parameter Q (i.e., reducing spring stiffness) for the right side to represent reduced cricothyroid tension. Though the Q parameter developed by Ishizaka and Flanagan (1972) may possibly reflect the effects of cricothyroid tension, Isshiki et al. (1977) observed that Q- and k-imbalances had similar effects. In our modified Koizumi model, Q was defined so as to affect only upper mass stiffness and mass and thickness of both masses. The results appeared similar to those of Isshiki et al. (1977), who described vibratory patterns resulting from unilateral paralyses by using excised larynges with weights to simulate asymmetric muscle tension. Results are also analogous to findings reported in superior laryngeal paralysis in humans (Arnold, 1961). Clinically, vibration in unilateral superior paralysis generally appears symmetrical, and glottal gap is not significantly altered.

Simulation of a unilateral recurrent laryngeal nerve paralysis produced a shift in closure to the right of midline, a drop in frequency, a reduction in speed quotient, and less two-mass motion of the paralyzed side. A similar frequency drop was also reported in simulated recurrent laryngeal nerve paralysis in the in vivo canine model (Trapp & Berke, 1988; Trapp, Berke, Bell, Hanson, & Ward, 1989). In contrast, in a sample of patients with recurrent laryngeal nerve paralysis studied by Hanson et al. (1988), average frequency was higher than for an unmatched group of normal speakers. This suggests that patients with laryngeal nerve paralysis may use compensatory mechanisms (e.g., high cricothyroid muscle tension) to increase contact and tension of the vocal folds to compensate for the paralyzed vocal fold. This compensating increase in frequency was also seen in the in vivo canine model when recurrent laryngeal nerve paralysis was simulated with the addition of high cricothyroid tension (Trapp & Berke, 1988). Photoglottographic (PGG) waveforms for various laryngeal neurological lesions have demonstrated shifts in the peak of the PGG and related speed quotient changes (Hanson et al., 1988). The present model showed similar shifts in glottal area waveform with simulated compensation of the intact side (see Figure 2). The shifts in PGG peaks and speed quotient measures may reflect an individual patient's degree of laryngeal compensation of the intact musculature, as well as the vocal fold asymmetry resulting from the neurological lesion itself, such as the combined effects of

atrophy, changes in stiffness, and changes in phonatory position.

Our results also demonstrated supraperiodic vibration, which Isshiki et al. (1977) described as Type II phonation. As discussed above, supraperiodic vibration has been reported for pathological and normal speakers (e.g., Hirano, 1975; Monsen, 1981; Klatt & Klatt, 1990; Rose, 1988) and in other simulation studies (Wong et al., 1991; Ishizaka & Isshiki, 1976). Supraperiodic vibration patterns were induced by stiffness asymmetries combined with certain subglottal pressure and initial glottal area conditions. Spectra from the vibratory patterns in our study and from that of Wong et al. (1991) displayed subharmonic acoustical energy in addition to noise energy between peaks. Although the output was not perceived as different pitches, it sounded rough.

These supraperiodic vibration patterns may be explained as follows. The glottal gap (how far apart the masses are when they begin to oscillate) influences the amplitude of lateral displacement of the masses. When the masses oscillate, their amplitude of displacement is also affected by the stiffness of the springs to which they are attached. The spring stiffness has been empirically determined to be nonlinear; as the amplitude of displacement increases, the spring stiffness increases exponentially (Ishizaka & Flanagan, 1972; Ishizaka & Kaneko, 1968) according to the relation $f = -(kx + nkx^3)$, where f is the restoring force exerted on the masses, x is the mass displacement, k is the spring stiffness, and n is a coefficient describing the nonlinearity of the spring. The greater the displacement amplitude, the larger the restoring force. At a small initial glottal gap (G), the displacement amplitude is small, and the frequency of vibration is determined by the mass with greater stiffness. The system oscillates at a higher frequency, and the stiffer mass crosses midline to contact the other mass on the flaccid side. At large G, the displacement amplitude is large, and the oscillation is determined by the side with the least stiffness, with the stiff side aerodynamically coupled to it. The flaccid mass crosses midline to contact the stiff side, until G becomes so great the masses no longer touch. The lateral excursion of the stiff side is limited at large G by the increased nonlinear stiffness forces exerted on the mass at large displacement amplitudes (at large displacement x, the x^3 term dominates the stiffness equation; Ishizaka & Flanagan, 1972; Wong et al., 1991). A transition point exists between these two states; at that point, the stiffness characteristics of the masses and their coupled aerodynamic forces are such that the two sides appear to compete for control of oscillation of the system. The flaccid and stiff sides are not fully aerodynamically entrained with each other; thus irregular, supraperiodic oscillation results.

The key role that initial conditions of P_{sub}, G, and stiffness parameters play in determining the vibratory mode in states of asymmetrical laryngeal tension is a significant finding of this study. The present results suggest that even small changes in initial glottal gap exert a strong influence on the resulting glottal vibration. Two patients with laryngeal paralyses and similar-appearing laryngeal exams may have differing stiffnesses, as well as slightly different resting gaps, and thus may require very different treatments. Research is ongoing to measure P_{sub}, G, and stiffness in humans, and to examine how these parameters interact in vivo to govern vibration (e.g., Berke, in press; Sercarz, Berke, Arnstein, Gerratt, & Natividad, 1991). Combined with findings from laryngeal modeling, such studies will provide an improved theoretical framework for describing the vibratory modes associated with laryngeal paralyses. Treatment outcomes for these disorders will also certainly improve with a better understanding of which variables govern laryngeal vibration and of how those variables interact.

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