Effects of Short-Term Endotracheal Intubation on Vocal Function

By: Neal S. Beckford, MD; Robert Mayo, MS; Albert Wilkinson, III, MD; Michael Tierney, DO

Beckford, N.S., Mayo, R., Wilkinson, A., and Tierney, M. (1990). Effects of short-term endotracheal intubation on vocal function. *Laryngoscope* 100 (4), 331-336.

*****Note: Figures may be missing from this format of the document**

Abstract:
Transient voice change associated with endotracheal intubation has generally been attributed to vocal fold trauma. To assess the role of altered vocal fold function in transient voice change, a study was designed to evaluate the audio-acoustic, endoscopic, and laryngostroboscopic characteristics of the postintubation voice. Vocal function of 10 patients undergoing short-term outpatient surgical procedures using general anesthesia and endotracheal intubation were studied preoperatively and postoperatively. A second group of 10 patients that did not have surgery or general anesthesia was used as an age-matched control. Fundamental frequency, frequency perturbation, electroglottography, endoscopy (including laryngeal stroboscopy), and subjective speech analysis by experienced listeners were used to assess vocal function.

No consistent differences in fundamental frequency were observed, although patient-to-patient variation was marked. Statistically significant increases in cycle-to-cycle fundamental frequency variation (jitter) were found postoperatively in the majority of the postintubation patients ($P<0.05$). Electroglottography, laryngeal endoscopy, and stroboscopic laryngoscopy did not demonstrate consistent changes in glottic mucosal function. Listener judgments characterized the postintubation voice change by decreased intensity, increased roughness, and lowered affect without consistent changes in pitch. The perception of decreased affect in the voices (characterized by reduction in pitch variation, vocal stress, and increases in pause times) was a strong perceptual marker for change in the post-intubation voice. Objective measures of laryngeal function suggest that the glottic contribution to postintubation voice change is minimal and that this dysphonia is probably multifactorial.

Article:

INTRODUCTION

Patients often complain of transient hoarseness and dysphagia after undergoing procedures requiring endotracheal intubation; indeed, this complaint is so common that it is often considered part of the surgical experience. The morbidity associated with even short-term endotracheal intubation can be greater than that associated with the surgery. These symptoms are wholly unpredictable and usually resolve in 12 to 72 hours. Vocal changes associated with endotracheal intubation are generally assumed to be a result of vocal fold trauma.

Substantial work has been performed addressing the effects of long-term endotracheal intubation on the subglottic larynx and trachea.1-3 Postintubation contact ulcers, arytenoid dislocation, granulomas, and recurrent laryngeal nerve paresis are recognized clinical entities and have been thoroughly researched.4,5 Information regarding those temporary changes in vocal function associated with endotracheal intubation is limited. This study was designed to characterize vocal change after short-term endotracheal anesthesia using a number of accepted measures of vocal function.

MATERIALS AND METHODS

Subjects

Patients scheduled for outpatient gynecological procedures under general anesthesia were selected for the experimental portion of this study. Patients with histories of voice problems or laryngeal surgery were not considered for evaluation. Ten patients formed the experimental group (average age = 33 years; SD = 11.5). An age-matched group (average age = 34.6 years; SD = 10.41) of 10 patients was selected to serve as a control. These patients were not anesthetized and did not have surgical procedures performed.

Anesthesia

The experimental group underwent outpatient general anesthetic procedures (average intubation time = 66 minutes; SD = 19.7). A variety of anesthetic techniques were used, including different combinations of anesthetic medications and endotracheal tube sizes. To avoid experimental bias, the anesthesia staff was not made aware that vocal function assessments were being performed.

Laryngeal Function Evaluation

Laryngeal function studies were performed at two intervals for the experimental group: on the morning of surgery and immediately upon discharge from the outpatient facility. The control group was studied at 8-hour intervals, the approximate time between preoperative and postoperative studies in the experimental group. Preoperative and postoperative data were then compared. Four parameters of vocal function were assessed.

Fundamental frequency (F_o) and frequency pertubation (J') analysis. Patients were placed in an International Electro-technical Commission sound-treated chamber for recording of speech samples. Each patient read a phonemically-balanced text, “The Rainbow Passage,” and produced three groups of the sustained vowels /a/, /e/, and /oo/. Recordings were performed at comfortable volumes. The average of F_o and J' values from three examples of each behavior were used for data analysis. Recordings were made with an ElectroVoice® Dynamic Cardioid Microphone and recorded on a Revox® B77 MKII stereo reel-to-reel analog tape recorder. Using a Visipitch® 6087 DS (Kay Elemetrics) interfaced with a IBM® XT personal computer, F_o and J' were obtained from the audio recording.

Electroglottography (EGG). An indirect assessment of vocal fold contact was obtained by measuring changes in impedance across the vocal folds during phonation. Electroglottographic wave-forms were obtained using a Fourcin® laryngograph (Kay Elemetrics) and analyzed on the oscilloscope of the Visipitch 6087 DS. Waveforms were photographed using a Polaroid® oscilloscope camera. Symmetry, amplitude, changes in peak configuration, ascending slope, descending slope, and genu configurations were characterized.

Laryngeal videoendoscopy and videostrobolaryngoscopy. Laryngeal morphology was assessed in the experimental group by using an Olympus® Type PNF Flexible fiberoptic rhinolaryngoscope attached to a JVC® GX-N8U Color Video Camera. Findings were recorded on videocassette tape. Topical 4% lidocaine was used for nasal anesthesia. Dynamics of vocal fold vibration were evaluated with videostroboscopy, using a Bruel and Kjaer® Rhino Larynx Stroboscope, Type 4914. Amplitude of vibration, horizontal and vertical aspects of mucosal wave function, and periodicity were characterized. Examination of the vocal folds was made during sustained normal, soft, and loud vowel phonation /a/ and at the upper and lower extent of vocal range. Indirect laryngoscopy was used as a screen to assess laryngeal morphology of the control group. Videoendoscopy and videostroboscopy were not performed.

Subjective speech analysis. Two doctoral-level speech pathologists independently analyzed the recordings of subjects' voices. A series of seven-point rating scales examined voice properties in terms of pitch, roughness, breathiness, and tenseness. On each scale, "0" was assigned to normal or expected performance value, +1,

+2, and + 3 to greater than normal function, and -1, -2, and - 3 to less than normal function. An additional parameter of patient "affect" was rated before and after surgery using a similar seven-point scale.

TABLE I.
Pretest Fundamental Frequency in Hertz.

	Group A	Group B
Conversation	168.84	186.33
/a/	172.83	189.37
/e/	181.48	204.88
/oo/	185.43	196.14

TABLE II.
Fundamental Frequency Change in Hertz.

Patient	Continuous	/a/	/e/	/oo/
GROUP A				
1	−31.7	−12	33.3	10.1
2	0.3	−6.9	−8.7	1.8
3	−16.1	−18.1	8.6	20.7
4	17.6	11.7	31.6	17.3
5	−1.5	29.4	24.1	37.7
6	11.4	16.6	−4.5	3.3
7	8.9	−52.9	3.4	−53.6
8	−3.5	6.35	−13.4	−20.5
9	2.3	−44.9	−50.25	−28.1
10	0.3	−10.1	3.1	−17.7
GROUP B				
1	−14	−22	−27	−20.4
2	−9	−60.1	−64	−61
3	−3	−15	6	−20
4	7	−37	−31	−17
5	−1	9	12.3	8
6	−25	−15.3	−57.6	−11.7
7	8	3.7	23.9	6
8	2	−5	5.7	21
9	9	34.7	24.3	24.3
10	−3	−12.8	−10.2	−6.1

RESULTS

Analysis of F_o and J'

Fundamental frequency is the frequency of vocal fold vibration during phonation. The preintubation mean F_o values were 177.21 Hz in the experimental group and 194.18 Hz in the control group (Table I). There was a slight decrease in F. for the four vocal tasks in both groups during the postoperative (second) evaluation: 2.3 Hz, experimental; 8.7 Hz, control (Table II). The paired Student's *t* test and Wilcoxon signed rank test, however, showed that these intragroup changes were not statistically different ($P > 0.3$). Frequency perturbation is defined as the cycle-to-cycle variation in F_o and when excessive, can be perceived as a rough or dysphonic vocal quality. Consistent and significant increases in perturbation values were noted in the conversation speech sample for the experimental group (0.4114%, $P < 0.05$). Slight decreases in control group conversation speech perturbation values (0.1521%) were taken to reflect no significant change. Overall, when J' changes were averaged for continuous speech and sustained vowels, the experimental group (increase of 0.2450%) showed a greater than fivefold increase over the control group (decrease of 0.0438%) (Table III**).** Although these results were not statistically significant at traditional levels, parametric and nonparametric analyses supported the conclusion that a postintubation increase in J' had occurred ($P < 0.1$). Similar testing for the /oo/ vowel demonstrated a stronger and significant correlation ($P < 0.05$). Unpaired Student's *t* tests showed that in conversational speech, J' for the experimental group was significantly increased compared to that of the control group ($P < 0.5$).

TABLE III.
Percentage of Perturbation Change.

Patient	Continuous	/a/	/e/	/oo/
GROUP A				
1	2.038	1.391	2.157	0.484
2	0.834	1.016	1.236	1.754
3	0.108	−0.156	−0.156	0.327
4	−0.223	0.223	−0.613	0.395
5	−0.211	−0.141	−0.001	0.403
6	−0.051	−0.305	0.401	0.009
7	0.764	−0.205	−1.109	0.266
8	−0.075	−0.361	−0.422	0
9	0.531	−0.126	0.041	−0.059
10	0.399	−0.197	−0.015	0.06
GROUP B				
1	−0.039	0.26	0.03	−0.251
2	0.216	0.025	0.116	−0.392
3	0.062	−0.046	−0.375	0.262
4	0.004	2.066	0.086	0.081
5	0.01	0.221	−0.144	0.041
6	−1.382	−0.832	0.108	−0.552
7	−0.012	0.251	−0.813	0.161
8	−0.11	−0.17	−0.312	−0.296
9	−0.158	−0.123	−0.019	0.46
10	−0.211	0.097	0.055	0.126

TABLE IV.
Electroglottographic Change.

Patient	Peak Configuration	Periodicity	Opening Slope	Genu
1	+	−	NC	+
2	+	−	NC	NC
3	+	NC	NC	+
4	NC	NC	NC	NC
5	NC	NC	NC	NC
6	NC	NC	NC	NC
7	−	+	−	NC
8	+	NC	+	NC

NC = No change.

Electroglottography (EGG)

Two experimental-group patients were unsuccessful in generating adequate EGG tracings due to excessive soft tissue and neck girth. Most pronounced changes in the others were noted in peak configuration. Half of the remaining patients demonstrated increases in the width of the peak consistent with increased vocal fold contact during the open phase of the glottic cycle. One patient demonstrated a decreased peak width, with the remainder showing virtually no change. Two patients showed less waveform symmetry after intubation, but most demonstrated no change. One patient's waveform showed improved regularity after intubation. No clinically significant changes were noted with opening slope and genu configuration (Table IV). Waveform tracings in the control group exhibited consistent, symmetrical wave morphologies and did not demonstrate any of the changes noted in the experimental patients.

Laryngeal Videoendoscopy and Videostrobolaryngoscopy

Of the 10 patients in the experimental group, one refused laryngoscopic examination. Preoperatively, the larynges of the remaining patients were within normal limits. Postoperatively, all patients underwent laryngoscopic examinations. One patient manifested a traumatic vocal fold hematoma. No other trauma was noted in the glottis, posterior pharynx, pyriform sinuses, or supraglottic larynx.

Videostroboscopy was performed on 6 of the 10 experimental patients. One subject refused videostroboscopic examination. Supraglottic tension (purse-string-like constriction of the supraglottic structures) precluded sustained observation of the true vocal folds and subsequent preoperative stroboscopic laryngoscopy in two experimental patients. One patient developed a large amount of postoperative supraglottic tension, preventing successful stroboscopic examination. No mucosal abnormalities were apparent on initial evaluations. Postoperative examination of the five remaining patients revealed no gross changes in vocal fold mucosal function. Mucosal wave propagation and amplitude were decreased in 40%. Mucosal wave asymmetry was noted in the patient who sustained the postintubation hematoma. No evidence of vocal fold edema was noticed in any of the patients. In general, these changes did not appear to be the result of trauma and were considered minimal. These small alterations in stroboscopic presentation may have been the result of functional factors such as intensity of phonation and glottic tension, causing changes in mucosal wave characteristics and not due to direct glottic trauma.

Subjective Speech Analysis

Little difference in speaking pitch was noted between preoperative and postoperative recordings (Table V). Postoperative changes were characterized by decreased intensity, reduced affect, and increased roughness, generally amounting to one-scale value. Three patients demonstrated a +1 increase in breathiness. Most of the patients displayed increased constriction or tightness in voice quality.

DISCUSSION

Endotracheal intubation was first described in 1878 by MacEwan, but was not advocated for general anesthesia until 1910 by Elsberg. It has since become one of the most common procedures performed in modern medical practice. Although many studies have characterized the acute and long-term complications of endotracheal tube placement, very little attention has been paid to the attendant vocal dysfunction. Objective assessments of vocal function impairment secondary to endotracheal tube placement are scant in the literature.

TABLE V.
Preoperative and Postoperative Subjective Speech Findings.

		Pitch			Roughness			Breathiness			Tenseness			Affect		
Patient	Age	Pre	Post	Change	Pre	Post	Change	Pre	Post	Change	Pre	Post	Change	Pre	Post	Change
1	36	0	1−	−	0	2+	++	0	1+	+	1+	2+	+	0	2−	−
2	54	0	1−	−	0	1+	+	0	1+	+	1+	2+	+	0	1−	−
3	36	1	1+	NC	0	0	NC	1+	2+	+	0	0	NC	1+	0	−
4	22	0	0	NC	0	0	NC	1+	1+	NC	0	0	NC	0	0	NC
5	18	0	0	NC	0	0	NC	1+	1+	NC	0	0	NC	0	0	−
6	27	0	0	NC	0	0	NC	1+	1+	NC	0	0	NC	0	1−	−
7	50	0	0	NC	0	1+	+	1+	1+	NC	1+	1+	NC	0	1−	−
8	31	0	1−	−	0	0	NC	0	0	NC	1+	2+	+	1+	1−	−
9	25	1+	1+	NC	1+	1+	NC	1+	1+	NC	0	1+	+	0	0	NC
10	31	0	1−	−	2+	2+	NC	1+	1+	NC	1+	2+	+	0	0	NC

NC = No change.

Gleeson and Fourcin6 used EGG to study changes in vocal fold vibration secondary to endotracheal intubation in children. This noninvasive measure of glottic impedance change generates an indirect measure of vocal fold contact. A frequency distribution histogram was developed from the initial waveforms in order to compare changes in laryngeal frequency distribution after intubation with similar studies in other disease states. It was hoped that insight into the nature of glottic damage and its effect on vocal fold vibratory patterns would be obtained. Changes in postoperative F_o distribution and the subsequent resolution of these changes over a 7-day period were demonstrated. Initial postoperative recordings showed a bimodal distribution pattern with an increased low-frequency accentuation and increased E. Clinically, an increased frequency of pitch breaks were noted. All patients' histograms returned to normal after 48 hours.

A similar technique was used by Lesser, *et al.*[7] in adults. He also showed an increased low-frequency distribution. All of his patients demonstrated an F. increase with a characteristic "harsh, creaky or metallic" vocal quality. These changes were confirmed with subjective speech evaluations by experienced listeners. Both of these studies are important in that they objectively document the change and subsequent resolution of a measure of vocal function after short-term intubation. No attempts were made to directly attribute these findings to morphologic changes in the vibrating portion of the vocal folds. Other methods, including complex acoustic wave analyses and strictly subjective studies, have been used to document reversible change in vocal patterns after short-term intubation.[8,6]

In the present study, an attempt was made to measure various parameters of vocal function in order to better characterize and possibly identify the sites responsible for postintubation voice change. Since voice is a function of the coordinated interaction of the respiratory system, glottic larynx, and supraglottic vocal tract (supraglottic larynx, pharynx, and oral cavity) as well as higher central nervous system functions, changes in the input of any of these areas can affect the vocal function to varying degrees. The lungs provide the aeromechanical energy that displaces certain vocal tract structures, generating pressures behind valves and generating flows through constrictions within the vocal tract. Changes in that air flow as it meanders through the vocal tract constitute acoustic speech. Pulmonary function is an important factor in the regulation of important parameters of speech such as intensity, fundamental frequency (pitch), lingual stress, and the division of speech into various units (syllables, words, and phrases).[10] Restricted ventilation after general anesthesia can result from splinting due to the musculoskeletal tenderness incurred with paralyzing agents or pain from abdominal or chest procedures. Impaired gas exchange resulting from atelectasis in dependent lung regions during general anesthesia with mechanical ventilation can alter respiratory function in the immediate postoperative period. These changes increase after administration of muscle paralyzing agents and can be reduced by use of intraoperative positive-end expiratory pressure." Clinically important ventilatory depression may persist for significant periods after the intraoperative administration of barbiturates and narcotics.[12,13] Postoperative administration of pain medications also have been associated with decrease in respiratory function. These anesthetic-related changes in pulmonary function may be responsible for some of the changes in perceived vocal function associated with general anesthesia.

The vocal-tract pain associated with endotracheal intubation can be attributed to traumatic endotracheal tube placement in the larynx, irritation of the posterior glottis and arytenoids from tube motion during respiration, and oral cavity/oropharyngeal trauma. Compensatory vocal tract configurations are often assumed to alleviate some of this discomfort.

This posturing may lead to alterations in the normal resonance characteristics of the pharynx and oral cavity, with resultant changes in voice quality.

Changes in cortical function associated with the administration of anesthetic agents may affect the fine vocal tract neuromuscular activity necessary to coordinate normal speech production. Narcotic analgesics are known to cause central nervous system changes characterized by drowsiness, inability to concentrate, difficulty in mentation, decreased fine motor control, apathy, and lethargy. These effects are dose-dependent and subject to individual variance, depending on the type of narcotic used. Even in situations where normal cognitive function is present, compromise in psychomotor testing has been demonstrated.[14] Inhalation anesthetics have also been known to cause similar symptoms, with longer durations. Changes in sensorium, often encountered for variable periods after administration of these agents, may be manifested by changes in phonation secondary to cortical input.

Findings from our study differ from those of other investigators in that the changes seen in F_o, although present, are not consistent. Whereas Gleeson and Fourcin[6] and Lesser, *et al.*[7] have shown consistent increases in Fa after intubation, our study does not reflect similar trends. Positive and negative changes in F_o were common and unpredictable, with the average group decreases in F_o (2.3 Hz in the experimental group and 8.7 Hz in the control group) not considered to be clinically significant. These changes may represent normal diurnal variations and may have nothing to do with the effects of intubation. In studying daily F_o fluctuations, Garrett and Healey[15] found a moderate range of variation in an individual's habitual pitch over the course of a day.

Consistent changes in the regularity of vocal fold vibration were reflected throughout the various measures used, albeit more convincingly with some tests than others. Acoustic analyses of voice recordings in the intubated group showed uniform increases in the cycle-to-cycle variation of F_o (J') with continuous speech and sustained vowels. The average increase in perturbation of conversational speech 0.4110% was significantly higher than that of the control ($P < 0.05$). This degree of change is felt to be clinically perceptible. The acoustic changes observed on objective analyses were confirmed by subjective assessment of audio recordings by our speech pathologists. EGG and stroboscopic laryngoscopy, however, did not reflect these changes.

Changes in J' can be a result of alteration of the intrinsic vibratory characteristics of the vocal fold mucosa or associated vocal tract systems, *eg* pulmonary, that participate in voice production. Increased J' can result from changes in the normal vibratory pattern of vocal fold vibration. These effects can result from deficits in vocal fold mobility, changes in glottic tension, and alterations in the integrity of the mucosa on the leading edge of the vocal fold. In our series, endoscopic assessment of the glottis after intubation showed gross evidence of vocal fold injury significant enough to disrupt normal vocal fold vibration in only one patient. Stroboscopic laryngoscopy, which provides a very sensitive and physiologic measure of vocal fold vibration, did not reveal changes that could account for the increases in perturbation appreciated with the acoustic studies. Perioperative inhalation of gaseous substances, *eg,* inhalation anesthetics and oxygen, could theoretically dessicate laryngeal mucosa and impair regular vocal fold vibration. Finkelhor, *et* al.[16] studied the effects of changes in vocal fold viscosity on vocal fold oscillation. Using a canine model, they demonstrated that the subglottic pressure necessary to initiate and sustain vocal fold vibration varies inversely with the amount of vocal fold hydration. This increase in hydration or decrease in viscosity was also associated with the impression of decreased pitch. Although no means of direct, in vivo measurement of human vocal fold hydration or viscosity are available, Bless and Shaikh[17] showed that effects of atropine-induced vocal fold dessication could be demonstrated by stroboscopic laryngoscopy. Vocal fold trauma stimulates a reactive edema that should result in greater vocal fold contact during the glottic cycle. Increased vocal fold contact, usually reflected by a broader peak on the EGG wave tracing, was not consistently evident in our study population.

It can then be assumed that either postintubation changes in vocal function are not solely a result of changes at the level of the glottis or the measures used to quantitate vocal fold function are not sensitive enough to detect the changes reflected in the acoustic and perceptual studies. Electroglottography has been found to be a useful instrument in demonstrating vocal fold mucosal abnormalities, including laryngeal polyposis, neoplasms, and glottic tension. Reproducible recordings, however, are dependent on electrode placement and can be difficult to obtain in patients with short, thick necks and ill-defined landmarks.

The character of EGG waveform can be altered by factors other than changes in vocal fold morphology. Changes in breath support and cerebral control of vocal function can lead to perturbation, intensity, and frequency differences that will be reflected in the glottograph. It is felt that the changes demonstrated with EGG may well have been a result of postintubation trauma. However, given the many variables known to affect the electroglottographic tracing, it cannot be unequivocally stated that the changes observed are a result of forces acting on the vocal folds or due to morphologic changes in the folds themselves.

Similar problems are encountered with the use of the stroboscopic laryngoscope. Extraglottic factors that affect the stroboscopic image are often related to changes in vocal stability and glottic and supraglottic tension. Producing a tone that exhibits excessive amounts of cycle-to-cycle variation will not allow the laryngeal stroboscope to flash at a rate that will produce representative stroboscopic images. Increases in glottic tension for a given frequency will decrease the amplitude of vibration and the horizontal and vertical phase characteristics of the glottic wave. Laryngeal tension is often reflected by posterior displacement of the pediole, collapse of the aryepiglottic folds, apposition of the vestibular folds, and to a lesser extent, posterior tilting of the epiglottis. These changes obscure the glottis and can make observation of the vocal folds difficult. Once again, these changes can be independent of any intrinsic change in vocal fold morphology, and may depend primarily on breath support and cortical control. Developing a study design that would control for all the variables that may influence vocal fold vibration would be most difficult.

CONCLUSION

Experienced listeners convincingly demonstrated that after short-term general anesthesia with endotracheal intubation the voices of patients were characterized by decreased intensity, increased roughness, and decreased affect. Acoustically, this postintubation dysphonia was characterized by an increase in the cycle-to-cycle variation of F_o (pitch) without consistent changes in sustained vowel or conversational E. Various standard measures of glottic function were unable to pinpoint the vocal folds as the site of this glottic instability. General anesthesia has multisystem effects, some of which can disturb the fine harmony necessary for normal voice production. Given that voice production is a function of laryngeal and extralaryngeal factors, it is safe to assume that changes in vocal quality may be a function of any one or all of these factors. These factors are potentially responsible for some of the changes in phonation commonly seen after administration of a general anesthetic with endotracheal intubation procedures. This is not intended to belittle the contribution of intrinsic vocal fold trauma, but to emphasize that other factors are at work and may be more significant in the perception of vocal quality than alterations in vocal fold morphology.

Bibliography:

1. McGovern, EH., Fitz-Hugh, G.S. and Edgemon, L.J.: The Hazards of Endotracheal Intubation. *Ann Otolaryngol,* 80:556, 1981.
2. Strong, R.M. and Passy, V.: Endotracheal Intubation: Complications in Neonates. *Arch Otolaryngol*, 103:329, 1977.
3. Hedden, M., *et al.:* Laryngotracheal Damage After Prolonged Use of Orotracheal Tubes in Adults. *JAMA*, 207:703, 1969.
4. Ward, P.H., *et al.:* Contact Ulcers and Granulomas of the Larynx: New Insights Into Their Etiology as a Basis for More Rational Treatment. *Otolaryngol Head Neck Surg*, 88:262-269, 1980.
5. Hahn, F.W., *et al.:* Vocal Cord Paralysis With Endotracheal Intubation. *Arch Otolaryngol*, 92:227, 1970.
6. Gleeson, M.J. and Fourcin, A.J.: Clinical Analysis of Laryngeal Trauma Secondary To Intubation. *J Royal Society Med*, 76:928, 1983.
7. Lesser, T.H.J., Williams, R.G. and Hoddinott, C.: Laryngographic Changes Following Endotracheal Intubation in Adults. *Br J Dis Comm*, 21:239-244, 1986.
8. Priebe, HZ., *et al.:* Effects of Tracheal Intubation of Laryngeal Acoustic Waveforms. *Anesth Analg*, 67:219-227, 1988.
9. Ishida, T, *et al.:* Quantitative Analysis of Tracheal Damage.*Crit Care Med*, 11:283, 1983.
10. Hixon, T.J.: *Respiratory Function in Speech and Song.* Little, Brown, Boston, p. 27, 1987.
11. Tolics, M.D., *et al.:* Lung Collapse and Gas Exchange During General Anesthesia: Effects of Spontaneous Breathing, Muscle Paralysis, and Positive End-Expiratory Pressure. *Anesthesiology*, 66:157, 1987.
12. Cartwright, M.B., *et al.:* Ventilatory Depression Related to Plasma Fentanyl Concentrations During and After Anesthesia in Humans. *Anesth Analg*, 62:966-974, 1983.
13. Goodman, L.S. and Gilman, A.: *The Pharmacological Basis of Therapeutics.* The Macmillan Company, New York, 1970.
14. Cohen, R.L. and MacKenzie, A.I.: Anesthesia and Cognitive Functioning. *Anesthesia*, 37:47-52, 1982.
15. Garrett, K.L. and Healey, E.C.: An Acoustic Analysis of Fluctuations in the Voices of Normal Adult Speakers Across Three Times of Day. *J Acoust Soc* Am, 82:58-62,1987.
16. Finkelhor, B.K., Titze, I.R. and Durham, P.L.: The Effect of Viscosity Changes in the Vocal Folds on the Range of Oscillation. *J Voice*, 1:320-325, 1988.
17. Bless, D. and Shaikh, A.: The Effect of Atropine on Voice Production. Presented at the State Medical Society of Otolaryngology, Milwaukee, WI, 1986.