GLOTTOGRAPHIC MEASURES OF VOCAL FOLD VIBRATION: AN EXAMINATION OF LARYNGEAL PARALYSIS*†§

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ABSTRACT

Photoglottography and electroglottography were applied to groups of patients with recurrent laryngeal nerve paralysis, superior laryngeal nerve paralysis, and combined recurrent and superior laryngeal nerve paralyses of idiopathic causes. Individual patients with resection of the vagal nerve above the origin of the superior laryngeal nerve were also studied. Open Quotient and Speed Quotient were calculated from the photoglottography signals. Speed Quotient values significantly differentiated recurrent laryngeal nerve paralysis from idiopathic paralysis and superior laryngeal nerve paralysis, as well as from normal function. Data from patients with vagal resection clearly differentiated them from patients with recurrent laryngeal nerve paralysis. The measure of Open Quotient distinguished pathological phonation from normal, but was not as useful for separation of differing lesions. Electroglottography appeared to be less useful than photoglottography. The pathophysiology underlying the observed glottographic signals is discussed.

Most clinical methods for examination of the vocal folds are visual in nature. Visual techniques are somewhat difficult to quantify, and thus subjective factors, such as the experience of the examiner, may limit comparison of one examination to another. Although standard laryngoscopic techniques allow assessment of gross neuromuscular function and anatomy of the laryngeal structures, they do not reveal the vibratory movements that actually produce voice. Vocal fold vibrations are among the more rapid physiologic events that occur in the human body, and are not visible to the human eye. To view these movements, images must be recorded at high speed, and then slowed for display. Ultra-high-speed cine images filmed at thousands of frames per second, then projected at slow speed, have provided much of our current knowledge about phonatory physiology.[1-5] Although available since the 1930s, high-speed cinelaryngoscopy has not proven practical for general clinical applications.

Stroboscopy, an alternate method to view vocal fold movement, produces a composite of glimpses from many cycles, allowing a slow motion image of vibratory motion of the vocal folds. This technique was one of the first methods used to study vocal fold vibration.[6] The stroboscopic image may provide a relatively accurate picture for regular vibratory patterns. However, the validity of the strobe image may be questionable when there is irregular vibration.[7] For pathological phonation it may be difficult to adjust timing of the strobe flash to the actual periods of vocal fold vibration.

Digital technology has greatly enhanced the feasibility of accurate measurement of high speed events. Digital recording hardware and signal analysis software are now available for inexpensive micro computer systems. Thus, it has become practical to implement in the clinical setting some electronic methods of examining vocal fold vibration, which were previously available only to basic research laboratories. Glottographic techniques—measures that reflect the vibratory movements of the vocal folds during phonation—have been used in speech research laboratories since the 1950s[7-10] (*cf*, Kitzing[11] for a recent review of several such methods). It has been suggested that glottographic measures may be useful for study of pathological voices.[12-14]

Electroglottography, the most widely used glottographic technique, measures impedance to a low current flow across the neck in the vicinity of the vocal cords.[8,15] The dynamic impedance between two skin electrodes changes as the vocal folds open and close. Although data from the technique must be interpreted with caution, most authors agree that the Electroglottography (EGG) signal provides a useful estimate of vocal fold contact during the glottal cycle.[16-18] Figure 1 demonstrates the generally accepted interpretation of events seen in the normal EGG pattern.[17] It is less clear how to evaluate EGG patterns associated with pathological phonation.

Photoglottography (PGG) uses a light sensor to measure transillumination of light through the glot-

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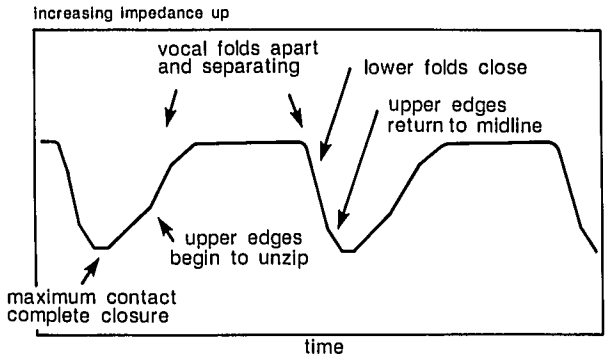

Fig. 1. Several events in the normal duty cycle of glottic vibration can be identified in normal EGG signals. (Idealized waveform modified from Rothenberg.[17])

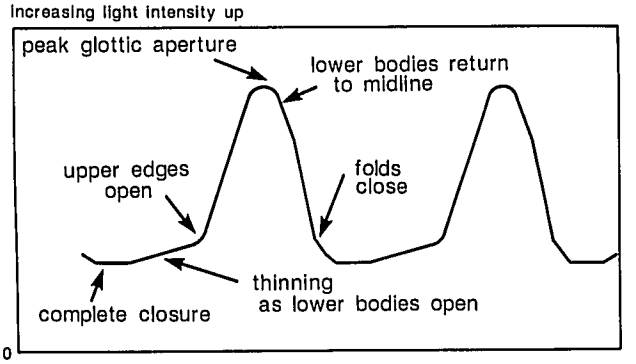

Fig. 2. The normal PGG signal offers complementary information to the EGG signal. In particular, peak glottal aperture is defined by the PGG waveform.

tis during phonation.[19] As the vocal folds separate and come together during the vibratory cycles, a voltage is generated which reflects intensity of light transmitted through the cross-sectional area of the glottis. Several authors have reported that PGG signals closely parallel changes in cross-sectional area measured from simultaneously filmed high-speed-cine photographs.[20-22] PGG does not provide absolute values for glottic area, and may be affected by factors such as movement of the light source in relation to the glottis. Thus, PGG waveforms must also be interpreted with caution,[23] and are useful primarily for phonation during sustained vowel production. Figure 2 shows the generally accepted interpretation of events represented by the PGG signal during normal phonation in modal register.[22] Several authors have noted that simultaneous recording of EGG and PGG signals offered complementary information about vocal fold vibration that increased the usefulness of both techniques.[12,13,20] The relative advantages and disadvantages of various glottographic techniques have been detailed by other authors.[11,24]

Because PGG and EGG data reflect changes in cross-sectional area and three-dimensional configuration of the glottis, these measures allow identification of events related to opening and closing of the glottis. Thus, like high-speed cinelaryngoscopy, glottographic techniques can provide data for measures such as Open Quotient (OQ) and Speed Quotient (SQ).[4]

Our prior work suggested that the measure of SQ appeared to be sensitive to gross variations in neuromuscular tension of the vocal folds seen in specific neuromuscular disorders.[14] Others have reported that SQ appears to reflect variations in vocal effort or "strain."[11,12] Such observations have not been tested in a systematic study of patients with voice abnormalities caused by specific laryngeal pathologies.

The purpose of this study was to examine glottographic signals recorded from patients with specific abnormalities of neuromuscular function of the larynx. Paralyses of the laryngeal muscles were chosen for study because well-defined lesions of the neural supply to the larynx would be expected to cause predictable effects on vocal fold function that would be homogeneous within a group of patients. We wanted to use such groups of patients to determine how well glottographically derived measures could differentiate among various types of laryngeal paresis. We also wished to study the pathophysiology of specific lower motor neuron lesions to the larynx.

Paresis of the laryngeal muscles may occur from a number of causes.[25,26] However, the physiological effects of the paralysis depend on the site and degree of neural injury.[27] Motor fibers to the intrinsic laryngeal muscles are carried in the recurrent laryngeal nerve, which branches from the vagus nerve in the neck. Motor fibers to the cricothyroid muscles are carried in the superior laryngeal nerve, which branches from the vagus nerve at or just below its exit from the skull. For most patients with laryngeal pareses, the exact site and degree of injury to the nerves is not known for certain. This probably accounts for some confusion in the literature regarding the specific effects of various lesions. Even in the case of complete paralysis due to section of a motor nerve, the configuration of laryngeal structures changes over time due to muscle atrophy and effects of unopposed muscle tension.[27] Therefore, careful selection of lesions was necessary to be assured of similar laryngeal pathophysiology within patient groups.

MATERIALS AND METHODS

One hundred twenty consecutive patients with laryngeal pareses were evaluated. Male patients between the ages of 25 and 65 with laryngeal paresis who had no prior injury, disease, neurosurgery, or laryngeal surgery which might have influenced speech production were selected. Selected patients had no co-occurring neurological disorders which might contribute to voice or speech difficulty, and had no history of voice or speech disorder other than that related to the laryngeal paralysis.

Forty-nine patients met these selection criteria. The best estimates of site of lesion for these patients are shown in Table I. The precise location and degree of injury to specific laryngeal nerves was not certain in most cases. Location of the lesion was most clearly identified for patients with surgical injury. However, even in these cases, the exact degree of injury to the nerve was often not known. In addition, the duration of the paralysis varied

TABLE I.
Apparent Site of Lesion for Patients with Laryngeal Paresis.

Location	Number	Percentage
Central nervous system	12	25%
Recurrent laryngeal nerve	14	29%
Isolated superior laryngeal nerve	6	12%
High vagal nerve section	2	4%
Idiopathic	15	30%
Total	49	100%

among patients. A few individuals with well-documented surgical injury at specific points along the peripheral paths of the motor nerves to the larynx were identified. These patients had distinct changes in voice quality associated with a surgical procedure for which the operative report was available. The injuries had occurred more than 12 months prior to the time that the patients were studied.

A group of six patients who met the selection criteria had surgical section of the recurrent nerve. On video laryngoscopy examinations, these patients demonstrated compensated paralysis of one vocal cord. At the onset of phonation, the vocal processes approximated with the cords on the same level, and there was no rotation of the glottal midline in relation to other glottic structures.

A second group included four patients who had distinct change in voice after surgery of the neck along the course of the superior laryngeal nerve. These patients complained of loss of pitch range and difficulty with voice quality. Each of these patients had laryngoscopic examination shortly after his surgery and had intact abduction and adduction of both vocal cords. On video laryngoscopy, these patients demonstrated with the onset of phonation, distinct rotation of the orientation of the vocal folds in relation to the structures of the supraglottic larynx. The posterior commissure appeared to shift toward the side of the paralysis. During phonation the vocal cords were asymmetrical, with the vocal fold on the paretic side appearing shorter and more rostral. These patients demonstrated the symptoms and laryngeal appearance characteristic of isolated superior laryngeal nerve paralysis as described by Ward, *et al.*[28]

Several patients with complete vagal section were evaluated. Most had other cranial nerve abnormalities, had received Teflon® injections, or had other factors which did not meet screening criteria. One 30-year-old male with isolated intracranial resection of the vagus nerve was in otherwise good health and met all of the criteria. A second patient who had resection of a vagal neuroma met all criteria except that of gender.

In addition to the patients with known surgical section of laryngeal innervation, patients who met the preliminary screening criteria were evaluated who had a history of sudden onset of hoarseness of unknown cause which had persisted for greater than 12 months. They demonstrated the laryngoscopic appearance of a unilateral combined recurrent and superior paresis. On documented laryngoscopic examination, these patients demonstrated paresis of one vocal fold in a paramedian position. Video laryngoscopy showed rotation of the posterior larynx toward the side of the paralysis at the onset of phonation. The vocal folds approximated on slightly different rostral/caudal levels with the fold on the paretic side appearing shorter and more rostral in position. All of these patients had otherwise unremarkable medical history, and normal head and neck anatomy. They also had normal CT or MRI scans of the brain stem, base of skull, neck, and chest. Five patients fit into this category and were considered to have idiopathic combined recurrent and superior paralysis.

For control data, adult male volunteers in good physical condition were screened for evidence of voice abnormality, and ten participants were selected who by history and examination had no current or past evidence of voice dysfunction. Each of these participants had normal findings on a careful examination of the head and neck, including video-documented, telescopic laryngoscopy. Table II shows the age distribution of patients and normal controls selected for the study.

TABLE II.
Distribution of Study Subjects and Patients by Diagnosis and Age.

Diagnosis	N	Age Range	Mean Age
Normal	10	36-65	56
Recurrent section	6	47-67	58
Superior section	4	36-68	55
Idiopathic paralysis	5	48-62	58

The participants were recorded in a double-walled, sound-treated (IAC) room. Following videolaryngoscopic documentation of gross laryngeal movement and anatomy, PGG and EGG signals were recorded during sustained phonation of the vowel /i/. This vowel was chosen because it is produced with the epiglottis in a forward position, thus obstruction of supraglottic light for PGG was minimal. EGG signals were recorded using a Synchrovoice Research Electroglottograph attached to the participant by an elastic collar. Two surface electrodes were located on either side of the thyroid alae, and a ground electrode was placed on the side of the neck. The unit provided a signal proportional to the dynamic impedance between the two electrodes located lateral to the thyroid cartilage. In addition, the device provided the first derivative of the dynamic impedance.

The photoglottography measurement system consisted of a Centronic, single-element photo-voltaic detector with an active area of 50 mm^2, followed by a preamplifier with a bandwidth of approximately 5 kHz. The photodetector and preamplifier were encapsulated in plastic and electromagnetically shielded. The photodetector was placed on the skin of the neck over the cricothyroid membrane. The larynx was illuminated by light projected from an Olympus flexible fiberoptic telescope inserted through a nostril and positioned so that the glottis filled approximately 50% of the viewing field. The illumination source for this unit was a 350-W xenon arc.

The EGG and PGG signals for three representative phonation samples were recorded simultaneously during steady state phonation of the vowel /i/, at comfortable pitch and loudness levels. Most of the laryngeal paralysis subjects were unable to match frequency or intensity targets. Thus, 1-second-long phonation samples were selected from the middle portion of the longest of three vowel productions for each patient at comfortable pitch and loudness. The glottographic signals were recorded for later analysis on a four-channel FM instrumentation recorder (Tandberg).

Samples of phonation at comfortable pitch and loudness were recorded in the same manner for adult males with normal voices who were matched for age to the paralysis patients. The recorded signals were low-pass filtered at 3 kHz and synchronously digitized at a sample rate of 20,000 points per second. Using a multipurpose signal analysis program, several points were marked for each glottal cycle. The point of opening of the glottis to airflow was defined by the method described by Gerratt, *et al.*[14] The point of peak opening was determined from the zero crossing of the velocity of the PGG signal. The amplitude of this peak was also measured. Baseline of the signal was determined by the PGG amplitude at the point that the electroglottographic signal indicated minimum impedance (maximum contact across the glottis). The point of closing was marked at the peak change of deceleration of the PGG signal, which corresponded most closely with the peak closing velocity of the EGG signal.

From these data, several measures were made for at least 50 cycles of representative phonation. The time-related measurements included: the period of each cycle; the closed phase; the open phase; the period during the open phase in which the glottic area was increasing; and the period during the open phase that glottic area was decreasing. From these values F0, OQ, and SQ were measured. OQ was obtained by dividing the duration of the open period by the duration of the entire period of the duty cycle. SQ was calculated by dividing the duration of increasing glottic area by the duration of decreasing glottic area.

In addition to the glottographic measures, high quality acoustic recordings were made during sustained vowel production by

TABLE III.
Glottographic Measurement Data.

Subjects	OQ	SQ	F0
Recurrent paralysis			
MS	1.0	.53	186
WL	.86	.44	144
OP	1.0	.38	78
BL	1.00	.40	148
CB	1.0	.52	150
GR	.99	.46	179
	$\bar{x}$=.98 SD=.05	$\bar{x}$=.46 SD=.06	$\bar{x}$=147 SD=38
Idiopathic paralysis			
KS	.99	.95	179
BG	.71	.92	137
WS	1.00	1.07	146
MK	.76	1.36	133
SL	1.00	1.10	138
HK	0.95	1.17	180
	$\bar{x}$=.89 SD=.13	$\bar{x}$=1.09 SD=.16	$\bar{x}$=152 SD=21
Superior paralysis			
DL	.98	.84	137
PM	.74	1.28	148
PA	.83	.99	152
HL	1.00	.98	160
	$\bar{x}$=.89 SD=.12	$\bar{x}$=1.02 SD=.18	$\bar{x}$=149 SD=9
Vagal paralysis			
DJ	1.00	2.44	130
EK	.91	1.70	260
Normal subjects			
BL	.527	1.02	117
DD	.520	.92	96
JS	.495	.88	105
DG	.529	.91	113
JI	.542	1.01	98
EK	.720	1.05	93
RC	.623	.84	113
GC	.531	.89	98
GC	.480	.89	151
JM	.525	.78	110
	$\bar{x}$=.549 SD=.071	$\bar{x}$=.92 SD=.09	$\bar{x}$=109.4 SD=16.81

each subject. Vocal tract resistance was calculated from airflow and from estimated subglottal pressure signals by a method similar to that reported by Netzell, *et al.*[29] Acoustic, perceptual, flow, and pressure data will be reported in a separate communication.

RESULTS

The data for OQ, SQ and fundamental frequency are shown in Table III. Open quotient provided a measure of the proportion of the glottal cycle during which there was flow of air through the glottis. Mean OQ for the normal speakers was 0.549 (SD=.071), indicating that the glottis was closed for approximately one half of the glottal cycle. In contrast, some of the pathological speakers who did not achieve closure of the glottis during the glottal cycle had OQ values of 1.0. Four of the six subjects with recurrent laryngeal nerve paralysis, two of the subjects with idiopathic paralysis, and one of the subjects with superior paralysis did not achieve any closure. The means of the three pathologic groups were all higher than the normal group demonstrating that the pathological speakers had a smaller duration of glottal closure than the normal subjects. Formal statistical evaluation of differences in OQ was not performed because of the ceiling effect of no glottal closure for many of the pathological subjects.

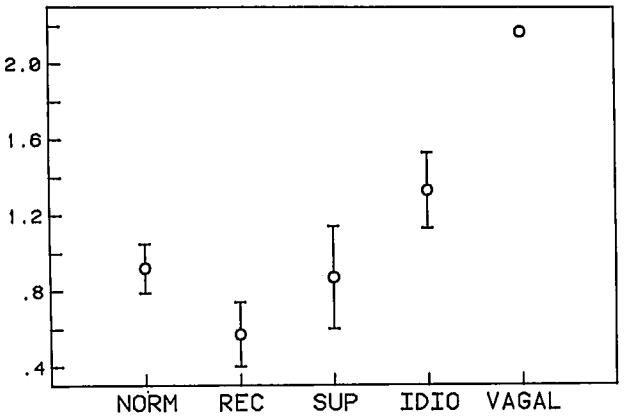

Fig. 3. Speed Quotient data for the groups listed in Table II represented by open circles for mean values and lines for standard deviations.

The relationship of the opening and closing movements of the vocal folds is reflected in SQ. The mean of SQ for the normal subjects was 0.919 (SD=.086), demonstrating that the duration of glottal opening was nearly equal to that of glottal closing. An analysis of variance of SQ, with one between-subjects factor revealed significant differences among groups ($F(3, 22)=34.083$, $p<.05$). Post hoc contrasts indicated significant differences ($p<.01$) between the normal and recurrent groups, between the normal and idiopathic groups, between the recurrent and idiopathic groups, and between the recurrent and superior groups. No other contrast was significant. Thus, the subjects with recurrent laryngeal nerve paralysis were significantly different from all other groups. Their SQ values were all lower than the other groups, indicating prolonged duration of glottal closing compared to opening. On the other hand, the subjects with idiopathic paralysis had SQ values which were higher than the normal group, demonstrating prolonged opening with respect to glottal closing. The subjects with superior paralysis had SQ values which were similar to those of the normal group. The individual patients with complete vagal section demonstrated SQ values greater than two standard deviations in excess of the mean of normal subjects. Figure 3 graphically represents the Speed Quotient values obtained for the patients and normal subjects.

The numerical data reflected visible differences in PGG signal patterns observed among the groups of patients and normal subjects. Normal phonation in modal range produced PGG and EGG signals that were similar to data reported by others.[20] An example of normal signals is demonstrated in Figure 4.

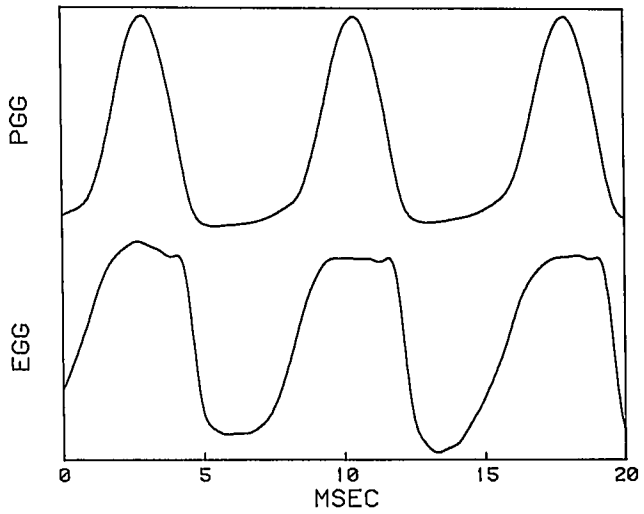

Fig. 4. Typical PGG and EGG signals for normal modal range phonation.

PGG and EGG signals representative of those recorded for patients with section of the recurrent laryngeal nerve are presented in Figure 5. The open period comprised most of the glottal cycle. Duration of the opening phase was less than half the duration of the closing phase. The EGG signal reached peak impedance at about the same time that projected glottal area was maximum. This differed from normal patterns in which the plateau of the EGG impedance occurred prior to peak glottal aperture. The plateau of the EGG signal waveform for these patients was less flat than in the normal pattern, and occurred relatively early in the duty cycle compared to normal signals.

The PGG signal patterns of patients with isolated superior laryngeal nerve paralysis were visibly different from those recorded from recurrent paralysis patients. Representative glottographic signals for

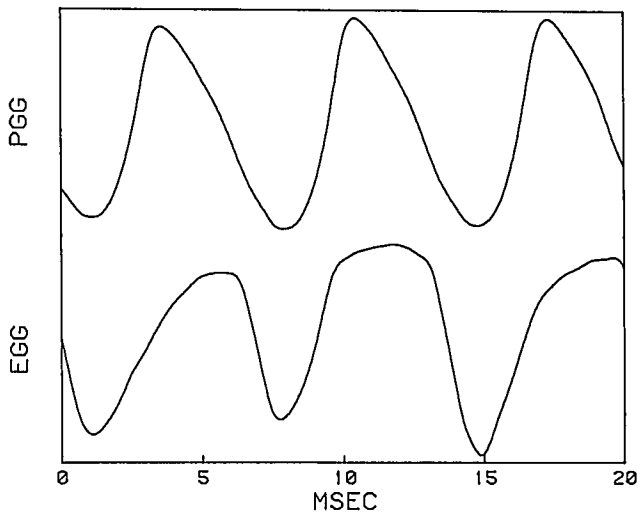

Fig. 5. These PGG and EGG signals from a patient who had resection of a recurrent laryngeal nerve are representative of the patterns observed in all patients with recurrent laryngeal paralysis.

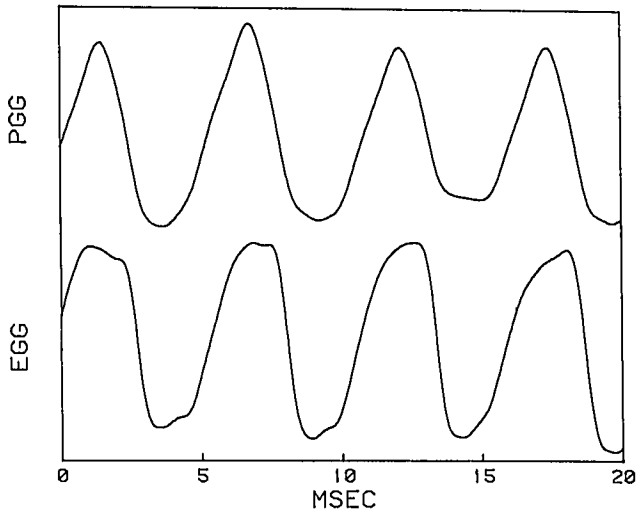

Fig. 6. PGG and EGG signals representative of the patterns seen in patients with superior laryngeal paralysis.

superior laryngeal nerve paralysis are shown in Figure 6. The baseline of glottic closure varied from cycle to cycle. The duration of opening was slightly longer than that of closing. In comparison to normal signals and to recurrent nerve paralysis, there was greater irregularity from cycle to cycle, often in a recurring pattern over several cycles. The EGG signals from this group of patients indicated that peak impedance (reflecting maximal separation of the folds) occurred relatively late in the cycle. In addition, the plateau of the EGG comprised less duration of the cycle in comparison to both normal and recurrent laryngeal nerve paralysis patterns.

The PGG signals from the patients with idiopathic paralysis also contrasted to those seen in patients with recurrent laryngeal paralysis. Representative glottographic signals for a patient with idiopathic paralysis are shown in Figure 7. The glottis was open for most of the cycle. However, in contrast to the patterns seen in recurrent laryngeal nerve pa-

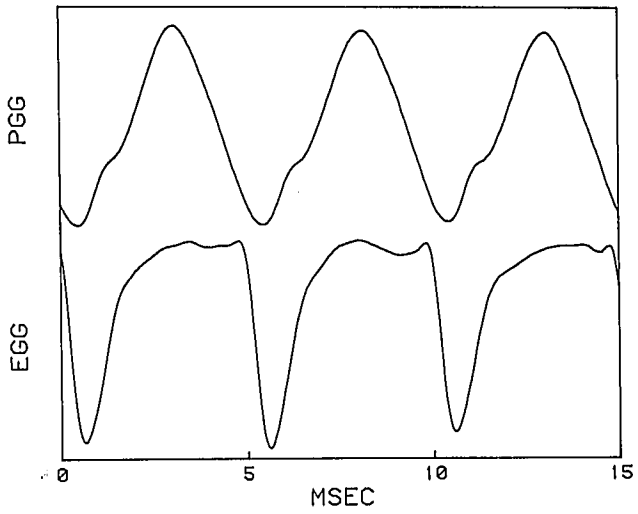

Fig. 7. PGG and EGG signals representative of the patterns seen in patients with idiopathic paralysis.

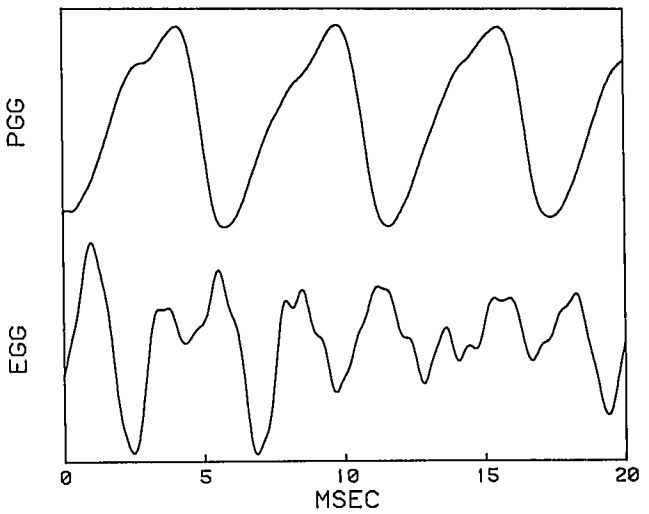

Fig. 8. PGG and EGG signals from a 30-year-old man 1 year after resection of a vagal neuroma above the foramen magnum.

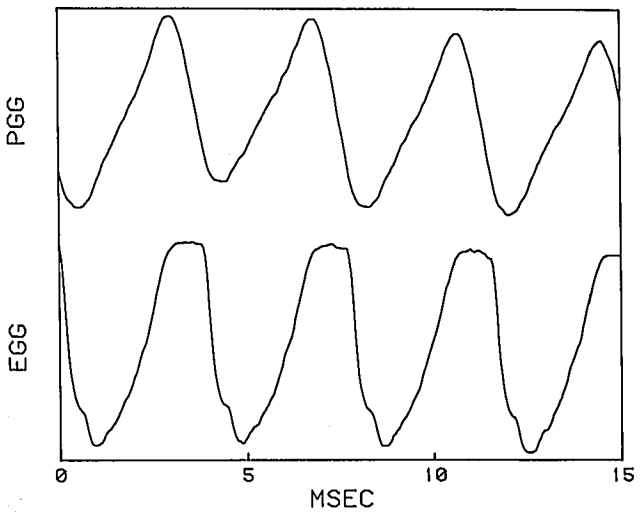

Fig. 9. Glottographic signals recorded from a 62-year-old woman 18 months after resection of a vagal neuroma.

ralysis, patients with idiopathic laryngeal paralysis had prolongation of the opening phase in comparison to the closing phase. There were often changes in the velocity of the opening slope of the PGG. EGG signals from the idiopathic paralysis patients indicated that maximal impedance (*i.e.*, least contact of the folds occurred in different patients at variable relationships with the peak of the PGG).

Figure 8 shows glottographic signals from a 30-year-old man 1 year after high vagal nerve resection. There was no closed period in the glottal cycle, reflected in variation of the baseline. The duration of opening in the PGG was prolonged in relation to closing with a brief change in velocity in the opening slope. The EGG signal reflected lack of vocal fold approximation with a low signal-to-noise ratio.

Glottographic data from a 62-year-old woman after vagal resection is shown in Figure 9 for comparison. This patient had better vocal fold approximation than the patient described above and had a relatively normal speaking voice. The PGG signal indicated a prolonged opening phase compared to the closing phase. The EGG signal showed a marked shift of the plateau of impedance in relation to the peak of the PGG signal, indicating maximal separation of the folds relatively late in the glottal cycle in comparison to normal patterns. The PGG signals from both vagal paralysis patients showed prolongation of the opening phase in comparison to the closing phase, with irregularity in waveform shape from cycle to cycle. The irregularity was most apparent in the slope of opening.

DISCUSSION

These studies of patients with carefully selected lesions of the laryngeal nerves indicate that there are specific variations in vibratory pattern for different lesions to laryngeal innervation. For comparison of the patterns, Figure 10 demonstrates examples of PGG signals for each type of paralysis. Measures of Open Quotient and Speed Quotient were sensitive to pathological phonation caused by lesions to the innervation of the larynx. Speed Quotient significantly distinguished particular pathological patterns among the neural lesions (except superior laryngeal paralysis compared to idiopathic paralysis).

There is little information regarding the normal range of Open Quotient and Speed Quotient values for male voices. Unpublished data indicate that, unlike OQ, SQ does not change significantly with variations in fundamental frequency or intensity.[30] Our studies of frequency and intensity effects on Speed Quotient indicate that differences of frequency and intensity among the phonation samples of normal speakers were not sufficient to explain the relatively large differences in Speed Quotient values among the various patient and normal groups.

The measures of F0, OQ, and SQ were all determined from PGG signals. The patterns of the PGG signals for the normal subjects and patient groups

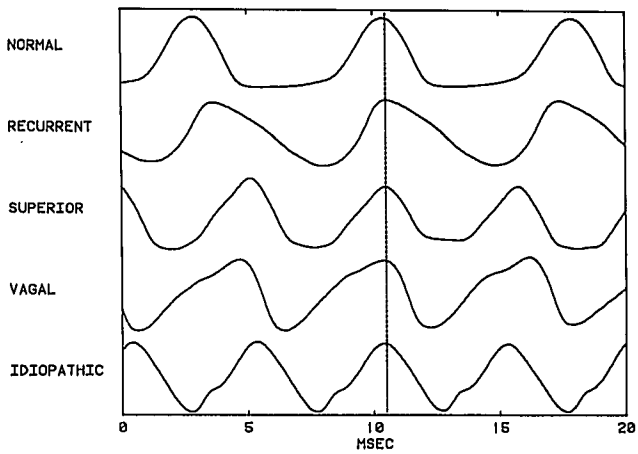

Fig. 10. A comparison of PGG patterns seen for the different lesions to laryngeal innervation.

were quite consistent for each diagnostic category. However, EGG signals in the patient groups showed a greater variation in patterns than the PGG signals. In part, variation in EGG signal patterns reflected the degree of approximation of the vocal folds. As approximation of the vocal folds decreased, the ratio of signal to noise in the EGG signal deteriorated. The EGG waveform was also affected by mucous stringing across the glottis.

To understand better the pathophysiological significance of these observations, an in-vivo canine model of vocal fold vibration (reported by Berke, *et al.*[31] and Moore, *et al.*[32]) was examined. Lesions of recurrent nerve, superior laryngeal nerve, and combination of the two were produced in the canine model. The PGG and EGG waveform patterns recorded in the canine models were similar to those documented for normal human subjects as well as for human patient data for respective types of paralysis. Simultaneous stroboscopy, triggered from a sharp negative spike in the velocity of the EGG signal, allowed observation of the vibratory movements that were associated with the glottographic waveforms.

In modeled normal phonation, it appears that the velocity of the opening slope of the PGG was determined by movements of the upper edges of the vocal folds. Closing of the glottal aperture reflected movements of the lower bodies of the folds. The increasing impedance of the EGG signal preceded the opening movements of the projected glottic aperture reflected in PGG signal because the folds opened from below, and the lower bodies of the cords reached maximal separation before the upper edges unzipped.

In modeled recurrent nerve paralysis, the flaccid cord did not vibrate in the normal two-mass mode, but rather was pushed laterally as one mass by the subglottal pressure. This was associated with relatively rapid opening of the glottis toward the side of the flaccid fold. The midline of the glottal area shifted toward the flaccid fold. Closure occurred with the upper edge of the normally innervated fold crossing the midline. This resulted in a picture that was very similar to data reported from the few high-speed cinelaryngoscopic studies of recurrent nerve paralysis in humans.[33] The pattern of PGG and EGG signals recorded in the model was similar to that seen for the recurrent laryngeal nerve paralysis patients. The glottographic signals recorded from these patients appear to reflect a pathologic vibratory pattern in which the flaccid vocal fold offers little resistance to subglottal pressure. When one fold is paralyzed, the upper edge of the flaccid fold moves laterally relatively early in the cycle, and, therefore, the opening slope of the PGG reflects primarily the movements of the flaccid cord. The flaccid cord also appears to be delayed in return to the midline, and glottic closure does not occur until the upper edge of the normal vocal fold crosses the midline to approximate the paretic fold. This results in a relatively decreased velocity of the closing slope of the PGG signal. The EGG signal also reflects the rapid separation of the folds early in the sequence of glottal opening.

In the canine model of superior laryngeal nerve paralysis, the upper edges of the folds vibrated 90° out of phase with the normal pattern. There was a clear shift of the posterior commissure toward the side of paralysis with the onset of phonation. On viewing from below, the vocal fold on the side of cricothyroid paralysis first moved laterally. As the subglottal air pulse reached the upper edges of the folds, the midline of the glottic aperture moved markedly toward the side of paralysis. The mucosal wave of both vocal folds then rolled toward the side of intact cricothyroid function. The picture from above showed the epithelial coverings of both folds rolling first to one side then to the other with each glottal pulse. The visible glottic aperture viewed from below the folds during stroboscopy traveled from the paretic side to the more tensed side as opening progressed toward the upper edges. Viewing from below, it was apparent that the cross-sectional area of the glottic aperture, as represented in the PGG signal, resulted from complex interactions of the movements of the lower and upper portions of two folds which, although vibrating at the same frequency, were vibrating out of normal phase with each other. The discontinuity in velocity of the opening slope of the PGG signals and the irregularity of glottographic signal patterns reflect these complex vibratory interactions. Such complex interactions were predicted by Ishiki, *et al.* in models of asymmetrical vocal fold tension.[34]

With modeled combined paralysis of recurrent laryngeal and superior laryngeal nerves, a combination of the effects of flaccid paralysis with asymmetry of longitudinal tension was observed in the vibratory pattern. The body of the intact, more stretched fold, viewed from below, appeared to vibrate very little, remaining near the midline. Viewing from above, the opening of the upper edges of the vocal folds was distinctly prolonged. Opening progressed from posterior to anterior. Lateral and medial movements of the upper edge of the flaccid fold lagged the movements of the more stretched fold. When the lateral excursion of the epithelium of the upper mucosal cover was maximal, the lower body of the intact vocal fold had already returned to midline. Closure of the glottic aperture then occurred very rapidly as the lower body of the intact vocal fold approximated the flaccid fold. The pattern of the PGG waveform seen for vagal paralysis reflected the effects on cross-sectional glottal aperture of prolonged unzipping of the upper edges and phase lag of the lateral and medial movement of the fold on the side of paresis. The PGG and EGG signal patterns and their relationship seen in the canine model of vagal paralysis approximated the appearance seen

for our vagal paralysis patient's data seen in Figure 9. This pattern of the EGG signal in relation to the PGG signal appeared to be associated with lack of firm approximation of the bodies of the folds so that the EGG signal reflected primarily the degree of contact of the upper edges. Because of the prolonged opening phase of the upper fold edges, maximal separation of the epithelial edges occurred relatively late in the cycle at a time when medial movement of the body of the intact fold was already reducing cross-sectional glottal aperture. Contact of the lower bodies of the folds occurred soon thereafter. Thus the plateau of the EGG occurred late in the duty cycle and was of relatively short duration.

The differing patterns seen in the PGG and EGG signals from patients with specific lesions of the laryngeal nerves are consistent with effects predicted by animal model studies. The data from the patient group with idiopathic paralysis are most consistent with the conclusion that these individuals had a lesion that affected motor innervation to both the intrinsic laryngeal muscles and the cricothyroid muscles on the same side of the larynx. The glottographic patterns of vibration were most similar to those seen in patients with vagal section. Previous reports have suggested that the lesion in idiopathic paralysis of the larynx is usually a high vagal, or central lesion.[35]

An additional factor that must be considered in the interpretation of these data is the possible influence of afferent receptor feedback reflexes on vocal fold tone. The exact effects of asymmetrical tension in the folds on afferent reflex control of vocalis contraction are not known. Presumably, stretch results in increased muscle tone to muscles that have intact motor innervation. Although the nature of the stretch-reflex arcs affecting the laryngeal muscles have been somewhat controversial, it is likely that such reflexes do exist.[36] This could affect both vocal folds if motor innervation were intact, as in superior laryngeal nerve paralysis, and may contribute to increased tension in the intact cord in vagal paralysis. The degree of motor fiber damage in idiopathic paralysis is not known and may vary among patients. The state of sensory fiber and reflex feedback innervation to the larynx in idiopathic paralysis is also not known.

CONCLUSIONS

It appears that glottographic measures can provide useful information regarding pathological phonation. Photoglottography provided essential information about patterns of vocal fold movement in the subjects we studied. The two measures — OQ and SQ — require knowledge of maximal glottal area which is not easily derived from the EGG waveform. Thus, electroglottography alone could not provide the information necessary to calculate these measurements. The simultaneous use of PGG and EGG signals provided complementary information, as has been previously noted. However, when there was lack of normal approximation of the folds, decrease in signal to noise ratio made the EGG signal less useful. Our experience with these patient studies indicated that PGG may provide useful information in cases of deviant phonatory function when EGG cannot.

Glottographic waveform configurations and the time-related measurements of OQ and SQ distinguished between normal and pathological phonatory function. Furthermore, measurements made from glottographic recordings distinguished between different neural lesions to the laryngeal musculature. In particular, SQ data clearly differentiated between recurrent laryngeal nerve paralysis and idiopathic laryngeal pareses. This observation may have some clinical applications, since the separation of these lesions on the basis of current examination techniques may be difficult. For most laryngeal paralyses, the extent and location of the neural damage are less well defined than were the lesions included in this study. Thus the validity of these measures requires further evaluation before glottography can be advocated for clinical application. Generalization of these results should be made with caution, considering the relatively small number of subjects studied.

Since current practice of both laryngology and speech pathology suffer from a lack of clinically applicable objective measures of phonatory function, these observations have implications beyond the application of glottography to laryngeal paralysis. Glottographic techniques may offer a means for better understanding of phonatory pathophysiology, and for better quantitative documentation of phonatory function in patients with voice disorders. It appears reasonable to propose that other laryngeal pathologies may demonstrate glottographic characteristics that would be useful in diagnosis and for documentation of the effects of therapy. These studies indicate that application of photoglottography and electroglottography to patient populations is feasible, likely to result in useful information, and should be pursued on a clinical research level.

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