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The Effect of Subglottal Resonance Upon Vocal Fold Vibration

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Summary: An experiment with excised larynges was undertaken to investigate the interaction between acoustic pressures in a pseudotrachea and the amplitude of vibration of the vocal folds. Pressure was measured beneath the vocal folds at three specific moments of the vibratory cycle: (a) when the superior margin of the vocal folds began to separate, (b) when the vocal folds were maximally apart, and (c) when the inferior margin of the vocal folds began to touch. Results indicate that in half the larynges investigated, the maximum amplitude of vibration increased as a function of: (a) increased positive pressure at the moment of opening, and (b) reduced subglottal pressure when the vocal folds were maximally apart. The implications of these experiments regarding involuntary register transitions related to trachea resonance are discussed in light of a previously proposed register theory. **Key Words:** Voice—Vocal folds—Subglottal resonance—Vocal registers.

It has been proposed that resonance in the subglottal airway can influence the way the vocal folds vibrate. According to Large (1), Nadoleczny-Millioud and Zimmerman (2) concluded that the influence of resonances in the subglottal airway undoubtedly had some effect upon the vocal registers in singing. Van den Berg (3) suggested that register transitions are experienced at the frequencies of the subglottal resonances and therefore, those resonances must be causal. Even with very limited evidence, these ideas have been compelling enough to have influenced pedagogical theories of voice training. Vennard (4) described the importance of subglottal resonance in explaining why the chest-to-falsetto (or head) register transition occurs in both men and women at approximately the same frequency (~ E_4). He concluded that it could only be the similarity in the subglottal resonances that could account for this; suggesting that anatomical differences in the size and shape of the larynges between the sexes are too great for them to have this in common.

Acquiring reliable estimates or measurements of the resonances in the trachea has not been a trivial matter. Direct measurement requires either the placement of a transducer through the glottis or through a puncture in the trachea. Historically, estimates of subglottal resonances have been made on cadavers of large dogs, humans (5), and laryngectomies (6). However, the recent development of miniature pressure transducers has enabled investigators to measure subglottal pressures in normal subjects. Small transducers can now be mounted on the end of a catheter and placed in the subglottal airway via the posterior glottis (7,8). The high-frequency response of modern transducers makes it possible to measure pressure changes that occur within the vibratory cycle. This allows for accurate determination of resonant frequencies in the subglottic space. Subglottal formant frequency data gathered in this manner differ significantly from early estimates, as can be seen in Table 1.

Subglottal pressure has two components: (a) the mean pressure that results from expiratory effort and glottal resistance, and (b) a high frequency modulating pressure that results from standing waves created in the subglottal airway (9–11). The present study seeks to investigate

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TABLE 1. *Subglottal formant frequencies and bandwidths reported in the literature*

	F_1'	F_2'	F_3'	B_1'	B_2'	B_3'
van den Berg[8] (1960)	300	870	1,427	120	150	
Ishizaka et al.[9] (1967)	640	1,400	2,100	155	140	
Boves[10] (1984)	475	1,175	1,945	275	330	340
Cranen and Boves[11] (1987)	510	1,355	2,290	104	154	358

Values are reported in Hertz.

the effect of high-frequency pressure modulations (acoustic pressure) in the subglottal airway on the amplitude of vibration of the vocal folds.

INFLUENCE ON AERODYNAMIC DRIVING PRESSURE

Titze (12) proposed a hypothesis of vocal registers that considers the subglottal acoustic pressure as part of the driving pressure of the vocal folds during phonation. Titze (12) described conditions of constructive and destructive interference between oscillating subglottic pressures related to subglottal formants and vocal fold movement. A constructive interference would increase the amplitude of vibration, whereas the amplitude of vibration would be diminished for a destructive interference. Titze further suggested that the changes in amplitude could be of great enough magnitude to result in sudden shifts of vibratory mode of the vocal folds; thus causing involuntary changes of vocal register.

Figure 1 shows a graph from Titze (12) in which constructive and destructive interference are identified. The resonance of the first subglottal formant is represented as a decaying sinusoid at 510 Hz (solid curve). The dashed curve is an idealized glottal area curve with an open quotient of 0.5 for one period of vocal fold vibration. Glottal opening begins with the stippled shading. Maximum opening of the glottis occurs at the apex of the dashed curve.

Constructive interference should occur when the positive peak of the pressure waveform coincides with glottal opening. In this case, the positive acoustic pressure adds to the driving force of the mean subglottal pressure. If, in addition, the acoustic pressure is below the mean subglottal pressure when the vocal folds are returning to the midline, (region of vertical cross-hatching) then optimum reinforcement should occur. The optimum relationship should occur when $F_0 = 3/5F_1'$, or approximately 306 Hz, as shown in the middle of the figure.

Cases of destructive interference can be seen when $F_0 = F_1'$, or when $F_0 = 2/5F_1'$. The acoustic pressure in these cases is negative when the vocal folds are moving apart and positive when they are moving together. Several other interference relationships at higher and lower F_0 are presented in Fig. 1.

At the optimum constructive interference condition ($F_0 = 3/5F_1' = 306$ Hz), it was predicted that the amplitude of vibration could be increased by as much as 0.8 mm (12). This is a significant change when compared with a typical amplitude of vibration of 1.0 – 2.0 mm (14).

Thus, an experiment using excised canine larynges on an experimental bench was undertaken to investigate the interaction between subglottal acoustic pressure and vocal fold vibration. Excised larynges were used to allow for quantification of vibrational amplitude and subglottal pressure.

The analytical predictions discussed previously were based on relationships between a constant subglottal formant frequency and a broad range of F_0 in the human. Because of the limited frequency range of the excised larynx, it was not possible to recreate the same conditions. Therefore, the experiment was designed so that the F_0 of the excised larynx would remain relatively con-

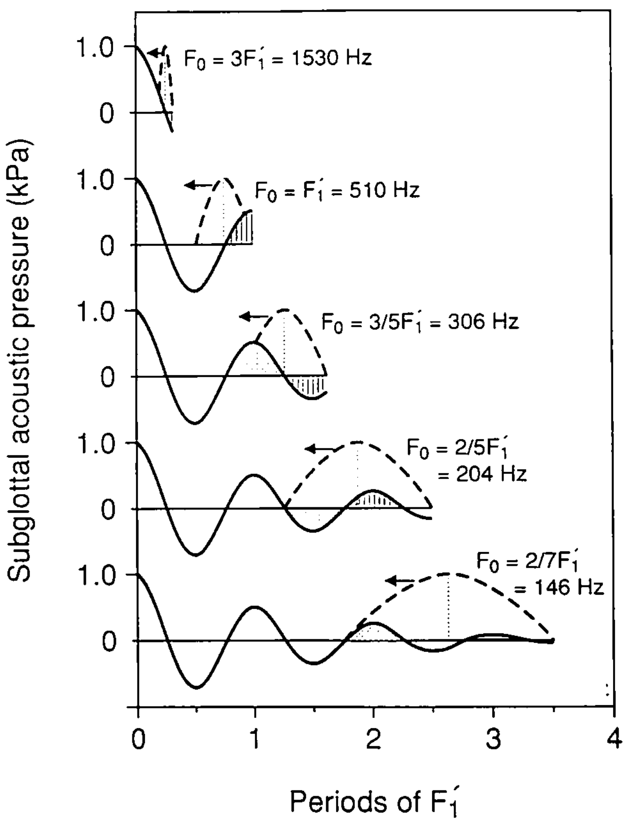

FIG. 1. Phase relationships between the acoustic pressure of a fixed first subglottal formant F_1' (solid lines) and the glottal area waveform (dashed lines) for systematically increasing fundamental frequency (bottom to top). (From Titze IR [12]).

stant, and the subglottal resonances could be tuned through a range of frequencies.

EXPERIMENTAL APPARATUS

A pseudosubglottal system was built using copper tubing acquired from a musical instrument manufacturer. The pseudotrachea consisted of straight and curved copper tubing similar to the slide mechanism on a musical brass instrument (Fig. 2). The length of the pseudotrachea was changed by sliding the telescoping lengths of tubing in or out of one another. Six different sets of slides of various length were necessary to provide a range of resonance frequencies wide enough to examine a sufficient range of fundamental frequency:subglottal-resonance frequency ratios. This tuning apparatus was integrated into a previously described bench set up for the experiment (15). The predicted frequency range for the first resonance of the pseudotrachea was estimated to be between 110 and 290 Hz. This was based on the assumption that the pseudotrachea functioned acoustically as a simple quarter-wave resonator.

PROCEDURES

Larynges

Excised canine larynges were procured from the cardiovascular research laboratory located in the Medical Research Laboratories at the University of Iowa. The dogs were typically of mixed breed and included both male and female animals weighing between 25 and 30 kg. Each animal had been used in cardiovascular research projects before the harvesting of the larynx. No animals were euthanized for the purposes of this experiment. The larynges were excised within a few minutes postmortem, placed in a 0.9% saline solution, and chilled for at least 24 hours before being used in the experiment. Each larynx was inspected for irregularities in anatomical structure. In particular, the medial surfaces of the vocal folds were carefully inspected for abnormalities. If any anomaly in structure was determined, the larynx was rejected. Preparation of the tissue for the experiment is described by Durham et al. (15), but a brief review of the procedures will follow.

Tissue mounting and data collection

The larynx was mounted by placing the tracheal tissue over the end of the pseudotrachea and securing it with a clamp. The larynx was stabilized with a customized mounting apparatus consisting of an aluminum framework with needle prongs attached to micrometers (Fig. 3). The needles attached to the micrometers were inserted into the muscular processes of the arytenoid cartilages and/or associated tissues, and were used to provide the adductory force to close the glottis.

The length of the vocal folds was adjusted by attaching a suture between the thyroid angle and a secure at-

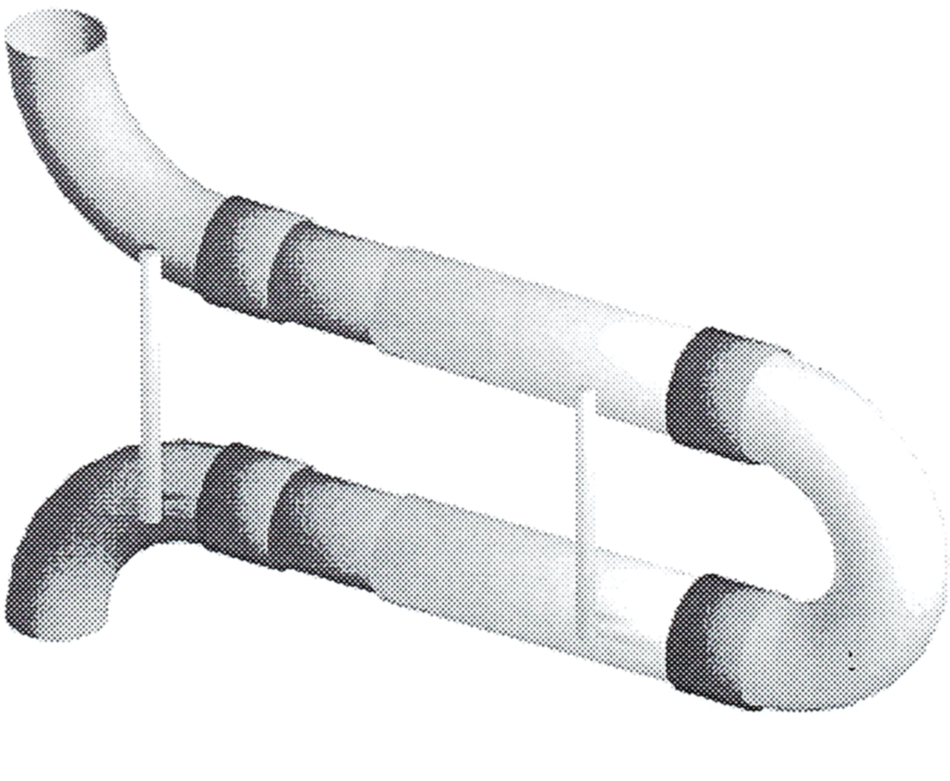

FIG. 2. Pseudotrachea consisting of a series of close-tolerance copper tubes and elbows that change the tube length by sliding in and out.

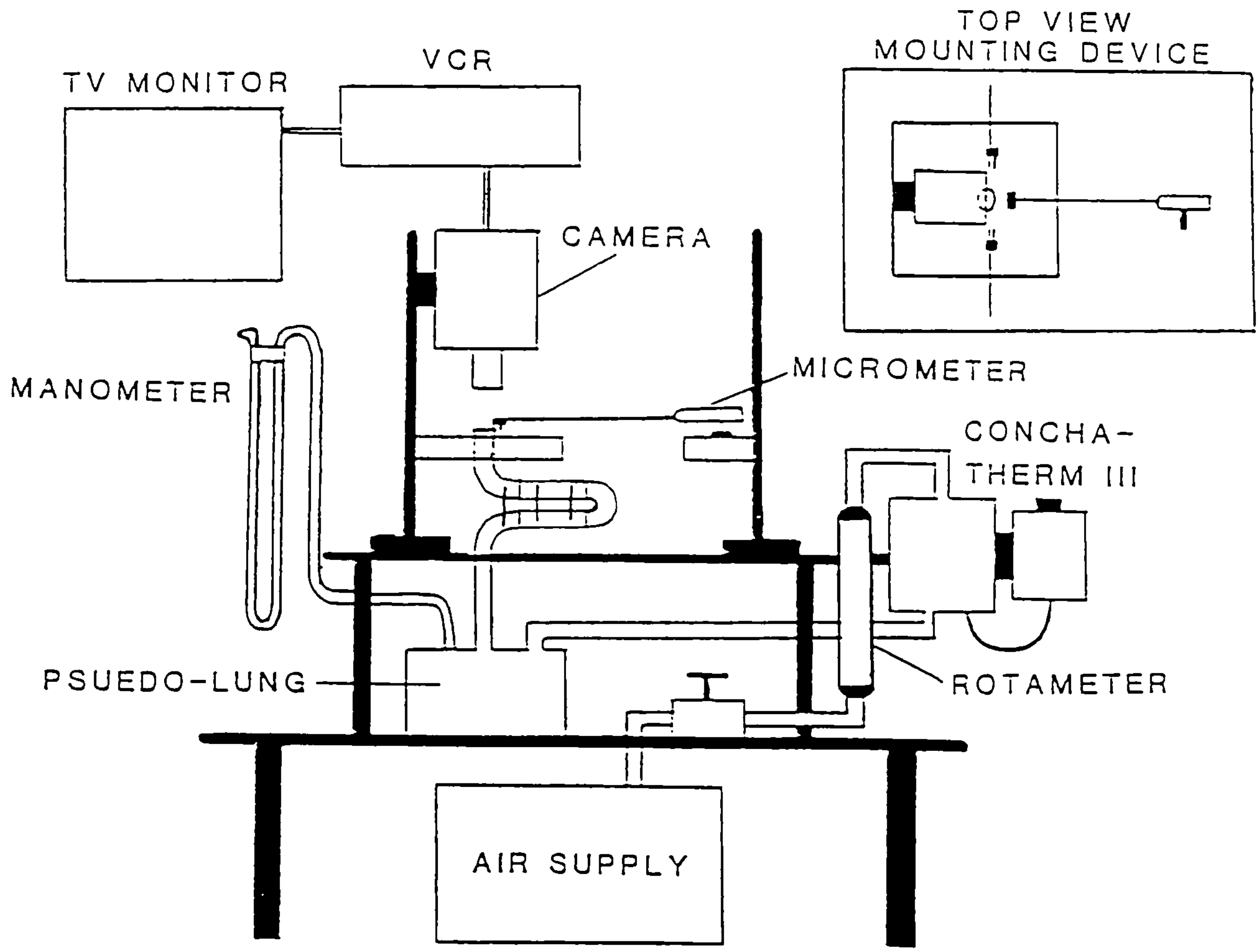

FIG. 3. Illustration of the excised larynx experimental setup.

tachment on the experimental apparatus. Optimum length of the vocal folds was subjectively determined for each larynx by judging the quality and stability of phonation. Optimum length was usually within 1 or 2 mm of the rest length (defined to be the *in situ* length of the vocal folds in the excised state). Length remained constant during the course of the experiment. Electrodes from a Synchrovoice Inc. electroglottograph (EGG) were attached to the thyroid lamina and held in place with small pins. The ground electrode was attached to the cricoid lamina.

A Micro Switch gauge–type pressure transducer (130PC) was used to record the pressure (P_{sg}) underneath the vocal folds. The housing of the transducer was fitted with a small extension tube for accessing the subglottal space. The transducer was mounted above the larynx, directly superior to the arytenoid cartilages (Fig. 4). The extension tube was placed between the arytenoid cartilages with the open end extending below the level of the vocal folds. (To avoid aberrant pressure readings that would result from the sensing membrane of the transducer being in the line of air flow, the distal end of the tube was sealed and a window was opened on the side of the tube for monitoring pressure.) (16) The tissue was sutured closed around the tube to prevent air leakage. The dynamic frequency response of the pressure transducer was measured to be flat to 1 kHz, and therefore well within the range of the predicted frequencies for the first formant.

Video images of the vocal folds were acquired with a Sony CCD video camera (model DXC-102) using a 90 mm Rokunar lens for measurement of glottal amplitude. The camera was mounted approximately 20 cm above the larynx and focused on the superior plane of the vocal folds.

The EGG signal was processed by a special phase delay circuit that was used to trigger a Pioneer DS330-ST stroboscopic light source (17). The phase of the

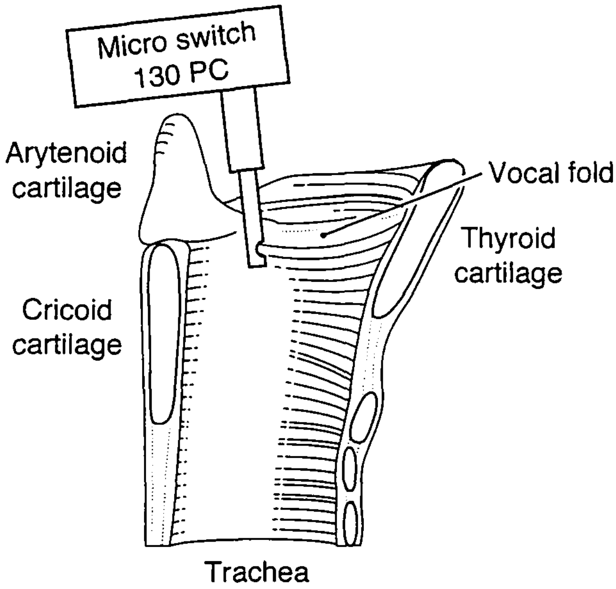

FIG. 4. Subglottal section of a larynx showing the placement of the sensing tube of the pressure transducer between the arytenoid cartilages in the posterior larynx.

strobe was controlled manually, which allowed the operator to visually "fix" the visual image at three specific points of the vibratory cycle: (a) maximum excursion from the midline of the glottis (henceforth referred to simply as *maximum*), (b) moment of separation of the superior margins of the vocal folds (*opening*) and (c) moment of first contact of the inferior margins of the vocal folds (*closing*). The image was 'fixed' at the three parts of the cycle by observing the video image on a TV monitor. Calibration of the image for measuring glottal dimensions was achieved by placing 1 mm grids on the superior surface of the vocal folds before the experiment. Video images were recorded on a Panasonic AG 1960 pro-line SVHS video cassette recorder, which has accurate frame by frame capabilities for analysis purposes. Maximum glottal amplitude was measured directly off the screen of a Sony Trinitron video monitor screen. Maximum glottal amplitude measurements were made when the glottis was rectangular, i.e. when the lower and upper lip of the vocal folds were vertically lined up.

A large voltage pulse created an event marker in the P_{sg} signal each time the strobe light flashed. Measurements were made on the pressure signal just before the superimposed pulse from the strobe at each of the three points during the vibratory cycle (as determined from the visual image).

A constant-flow air supply was provided by an Ingersoll-Rand Type 30 air compressor. An in-line flowmeter was used during the experiment to verify approximate flow rates at the beginning of each trial. All signals (EGG, P_{sg}, acoustic signal) except the video image were recorded with a digital Sony Instrumentation recorder (PC-108M) at a bandwidth of 5 kHz (10 kHz sampling frequency). With the use of the digital data recorder, it is estimated that the EGG and P_{sg} signals were essentially time synchronous within one or two sampling points. The air was conditioned for temperature and humidity by two Concha-therm III devices (Respiratory Care Inc.) to approximately 37–40°C and greater than 95% humidity downstream of the pneumotachometer but before entering the pseudotracheal tube system. Some cooling of the air took place in the tubing of the pseudotrachea between the pseudolung exit and the vocal folds.

After each larynx was mounted and the transducers were attached, a suitable subglottal pressure for stable phonation was determined and used throughout the experiment. A U-tube manometer was employed during the experiment to monitor the mean subglottal pressure for each condition. Phonation was initiated and all signals were recorded for each trial. The strobe was turned on

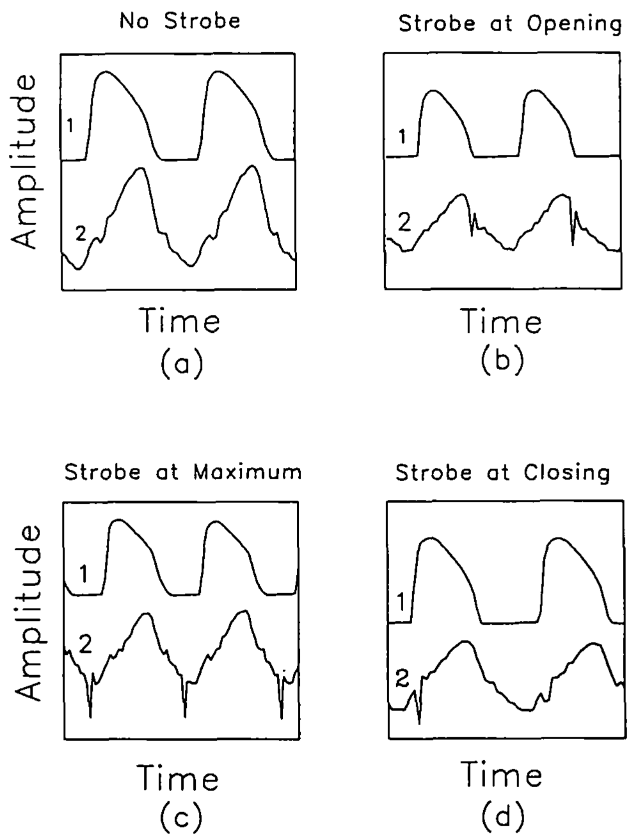

FIG. 5. Points of measurement on the subglottal pressure waveform. Top waveform (**1**) is the EGG signal and (**2**) is the pressure waveform; (**A**) without the strobe pulse, (**B**) strobe pulse superimposed on the pressure waveform at the moment of glottal opening, (**C**) strobe pulse at moment of maximum glottal width, and (**D**) strobe pulse at the moment of glottal closing.

and the video image used to tune the trigger–delay circuit to each of the three experimental moments of the vibratory cycle. Several seconds of all data signals were recorded at each of the three previously determined moments. Phonation was stopped and the next subglottal tubing length condition was set (using 1- cm increments in random order). The process was repeated until the full range of the pseudotrachea tubes had been used. The vocal folds were periodically moistened in a 0.9% saline solution to prevent desiccation of the tissue.

RESULTS

Figure 5 shows typical EGG and P_{sg} waveforms from the experiment. Part **A** is an example without the strobe pulse. The strobe pulse can be seen as the negative-going spike in the other three graphs. In part **B** the spike occurs at the moment of opening (as determined by the visual image), in part **C** during the maximum amplitude of vibration, and in part **D** at the moment of closure of the inferior margin of the vocal folds. The point on the P_{sg} waveform immediately before the spike occurred was taken to represent the pressure at that point in the vibratory cycle. By comparing the shape of the waveform with and without the strobe pulse, it was determined that the pulse introduced no significant distortion of the waveform before the point of occurrence.

A quantitative relation was sought between the amplitude of vibration and the effective acoustic driving pressure on the vocal folds. Vibrational amplitude is dependent on several things, including vocal fold length, the mean subglottal pressure (14), and the acoustic pressure discussed here. Given that mean subglottal pressure is the primary variable, the amplitude of vibration will be reported as a ratio of the maximum half-width of the glottis normalized to the mean subglottal pressure. This will be called the *glottal amplitude ratio* (R_a) defined as:

$$ {}_a = \frac{\text{½ maximum glottal width at midmembranous vocal fold}}{\text{mean subglottal pressure}} $$

Defining an effective acoustic driving pressure is less straightforward because subglottal pressure does not drive the vocal folds over the entire glottal cycle. In the open part of the cycle, the net driving pressure on the medial surface of the vocal folds can be idealized as (12):

$$ P \approx P_{in}\frac{a_2}{a_1} + P_{sg}\left(1 - \frac{a}{a_1}\right) \text{ where } a_1 > a_2 $$

where P_{in} is the input pressure to the vocal tract (supraglottal pressure), P_{sg} is the subglottal pressure, a_1 is the duct area at glottal entry, and a_2 is the duct area at glottal exit. For an excised larynx phonating into free space, $P_{in} \approx 0$ and the driving pressure is the subglottal pressure modified by the factor $1 - a_2/a_1$. When $a_1 > a_2$ (a highly convergent glottis), the full subglottal pressure is applied to the vocal folds. This occurs just after glottal opening (see the sketches at point **C** in Figs. 6 and 7). For a rectangular glottis, however, the driving pressure is basically zero because $a_2/a_1 = 1$. This occurs at the maximum glottal width (point **D** in Figs. 6 and 7). For a divergent glottis, the driving pressure is also near zero because the flow detaches from the wall and the glottal airstream is in the form of a jet (18). Thus, we conclude that the subglottal pressure has its main driving effect in the time interval between glottal opening and maximum glottal width. We therefore define

$$ \Delta P = P_{sg}(\textit{at glottal opening}) - P_{sg}(\textit{at maximum glottal width}) $$

to be the relevant acoustic pressure change that should influence the amplitude of vibration. Again, in Figs. 6 and 7, this pressure change occurs between points **C** and **D**. It is small and negative in Fig. 6, but large and positive in Fig. 7.

Figure 6 presents waveforms for a case in which the length of the pseudotrachea was approximately 38 cm. The measured F_1' (first subglottal formant) was 361 Hz and the F_0 was 181 Hz ($F_0/F_1' = 0.5$). The calculated one-quarter wavelength F_1' of the pseudotrachea was 230 Hz. The discrepancy in the predicted and measured subglottal formant frequencies indicates that the subglottal system was not behaving as a simple quarter-wave resonating tube, probably because the terminations at both ends were nonideal. The upper trace (**A**) is the EGG signal and **B** is the P_{sg} signal. Coronal images of the vocal folds are placed along the pressure waveform to identify specific events during the vibratory cycle. Point **A** marks the moment that the inferior margin of the vocal folds make contact, which is the beginning of the closed phase. At **B** the superior and inferior margins of the vocal folds are in full contact, **C** is the moment just before opening of the superior margin of the vocal folds, and **D** indicates the moment of maximum open glottis (where the superior and inferior margins are vertically aligned). Placement of the coronal images were estimated by the timing markers from the strobe pulses. Figure 6 is a case of destructive interference between subglottal acoustic pressure and vocal fold amplitude. Specifically, subglottal pressure is lowest before glottal opening and rises too late in the open phase to drive the vocal folds outward. The folds are already returning toward the midline when

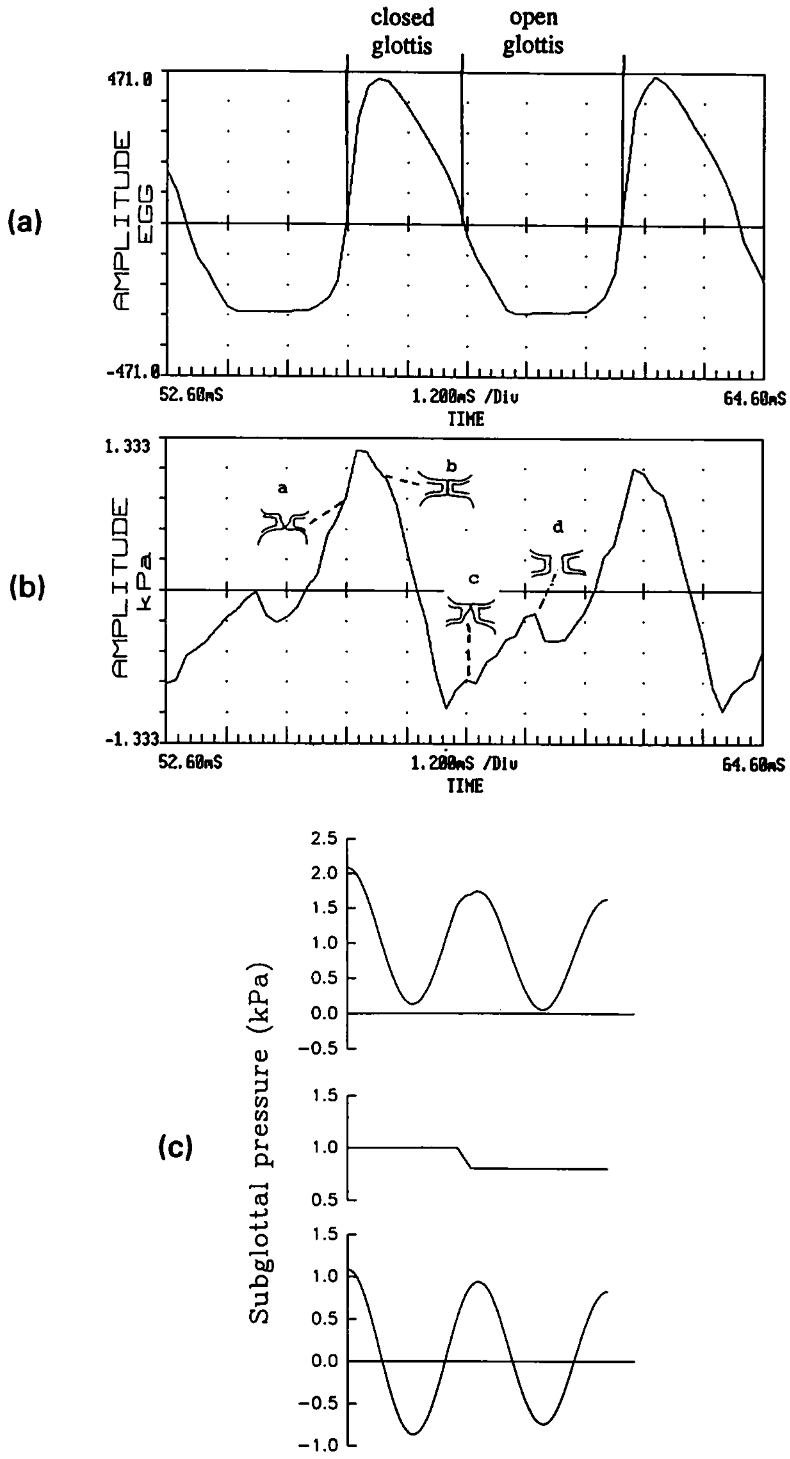

FIG. 6. Waveforms from electroglottograph (A) and subglottal pressure transducer (B) with schematic drawings of coronal sections of the vocal folds at four points in the vibratory cycle. This case represents destructive interference between subglottal acoustic pressure and vocal fold movement. Subglottal pressure is low at glottal opening and rises during the open glottis phase. (C) A damped sinusoid of the same frequency as the measured first subglottal formant (bottom waveform) and a modeled mean subglottal pressure waveform (middle waveform) are added together. The resulting waveform (top) resembles the shape of the measured subglottal pressure signal.

the pressure reaches its second peak. Thus, the acoustic pressure does not provide any desirable driving force to the vocal folds in their lateral excursion. The F_0/F_1' ratio here is 0.63, close to the 3:5 ratio suggested in Fig. 1 for constructive interference, but it seems that there are delays in the acoustic pressure that prevent a favorable phase relation.

The waveforms in Fig. 7 are from the same larynx but with a different subglottal resonance frequency. In this case the length of the pseudotrachea was approximately

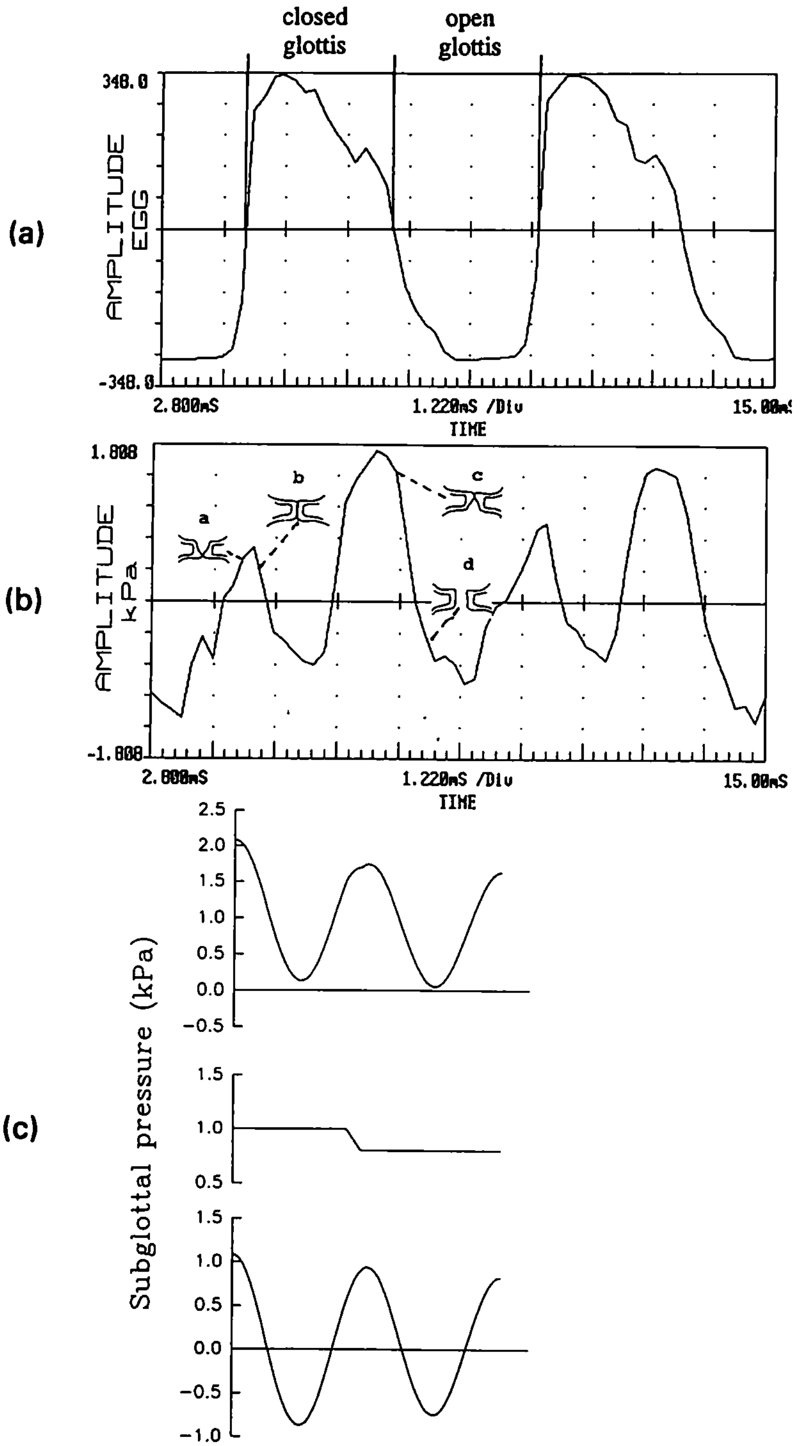

FIG. 7. Waveforms from electroglottograph (**A**) and subglottal pressure transducer (**B**) with schematic drawings of coronal sections of the vocal folds at four points in the vibratory cycle. This case represents constructive interference between subglottal acoustic pressure and vocal fold movement. Subglottal pressure is high at the glottal opening and decreases during the open phase. (**C**) A damped sinusoid of the same frequency as the measured first subglottal formant (bottom waveform) and a modeled mean subglottal pressure waveform (middle waveform) are added together. The resulting waveform (top) resembles the shape of the measured subglottal pressure signal.

80 cm, with a measured F_1' of 350 Hz. The measured F_0 was 171 Hz, giving an F_0/F_1' ratio of 0.49. In this case the pressure was highly positive at the moment of glottal opening. Thus, the conditions existed for the acoustic pressure to drive the vocal folds in their natural movement. Glottal amplitude ratio for this case was .113, compared with 0.06 for the less constructive case in Fig. 6.

In both Fig. 6C and Fig. 7C the gross shape of the pressure waveform has been modelled by adding a damped sinusoid at the same frequency as the measured subglottal resonance frequency (bottom waveform) to an idealized mean subglottic pressure waveform (middle waveform). When added together (top waveform) the shape resembles that of the measured P_{sg} waveform. However, it is obvious from viewing the pressure waveforms in Figs. **6B** and **7B** that the actual subglottal pressure signal is more complex than the hand-drawn illustrations suggested in Fig. 1.

Of particular interest in much of the pressure data is the fact that the initial pressure maximum does not coincide with the moment of closure. A complete explanation of this delay is not available at this time. It does not appear to be related to a delay imposed by the instrumentation. The occurrence of the pressure maxima is dependent on the length of the pseudotrachea, therefore eliminating artifact of the instrumentation as the source of the delay.

Scatter plots for data gathered on six larynxes are presented in Fig. 8. Glottal amplitude ratio R_a is plotted as a function of the driving pressure ΔP as the pseudotrachea was elongated in 1-cm increments. Each data point represents a different length of the pseudotrachea and is the averaged value of at least 20 consecutive cycles from the same trial. Mean subglottal pressure was constant throughout the experiment for each larynx. The lines through the data indicate the best-fit first-order linear regression. The regression coefficient (r) is indicated in each plot. For the three larynges on the left side (L10, L12, and L15), the data indicate no strong relationship between acoustic driving pressure and the amplitude ratio. Correlation coefficients are generally low except for the case in L12 where the mean subglottal pressure was 1.4 kPa where $r = 0.60$. A positive relationship is seen between the glottal amplitude ratio and the acoustic driving pressure ΔP for the three larynges on the right side of Fig. 8. Correlation values are modest in significance, but physiologic data of this kind cannot be dismissed as meaningless: L11 ($r = .53$), L14 ($r = .67$), and L16 ($r = .85$).

DISCUSSION

The data presented here indicate that the subglottal pressure in excised larynges contains not only a mean (dc) component, but also an acoustic component owing to resonance in the subglottal airway. The resonance frequencies were, at least in part, under experimental control and dependent on the length of the pseudotrachea. But the F_0/F_1' ratios predicted by Titze (12) were not verified. For one case, a constructive interference was observed for $F_0/F_1' = 0.49$ instead of 0.60; in another, a case of destructive interference was observed for $F_0/F_1' = 0.63$ instead of 1.0. These smaller ratios may be because the open quotients were not 0.5 as assumed in the theoretical treatment. A larger open quotient would require a higher F_1' (and hence a smaller F_0/F_1') for positive pressure to occur at glottal opening. The discrepancy may also be related to the fact that the initial peak acoustic pressure occurred slightly after the point of closure.

The magnitude of the peak-to-peak pressures in our data were similar to those reported elsewhere. Peak-to-peak values in the literature vary from 40% to 50% of the mean value (19) to as much as 100% of the mean pressure (20,21). In the present study, the pressure at a specific moment in the vibratory cycle varied over a range equal to or greater than the mean subglottal pressure.

It is common with excised larynges to have considerable variability in the data. Consistency among different larynges is difficult to quantify before the experiment. Conditions for stable oscillation were established according to the response of the individual larynx; no attempt was made to control the adductory force, longitudinal tension, or any other dimension of the vocal folds in any uniform manner among all larynges.

At least half of the data presented seem to support the general theory that acoustic pressures in the subglottal vocal tract can have an influence on amplitude of vibration of the vocal folds. An increase in the glottal amplitude ratio occurred when the vocal folds were exposed to higher pressures at the moment when they were opening, and lower pressures when they were maximally apart.

Application to human data

At this point we may ask, do these data on excised canine larynges apply in any way to human phonation? Fig. 9 shows data from Miller and Schutte (22) indicating subglottal pressure variations (labeled P_{sub}) in a human subject. The pressure signals were recorded in the vocal tract with miniature pressure transducers. The EGG signal was used to mark closing and opening of the glottis (vertical lines). The waveforms show the pressure events associated with the subject singing /ibi/ at an F_0 of 225 Hz. The marked segment on the left is before the plosive consonant and during the initial vowel. A pressure maximum in the P_{sub} waveform occurs just after glottal closure and is followed by several negative and positive pressure peaks, each of decreasing amplitude (caused by damping). The point marked as the opening moment (O) occurs coincidentally with the second positive pressure peak and indicates a low pressure on the P_{sub} waveform, which the authors identify as 2.6 kPa. The mean value for subglottal pressure was not given for these data, but it appears from the overall shape of the waveforms that

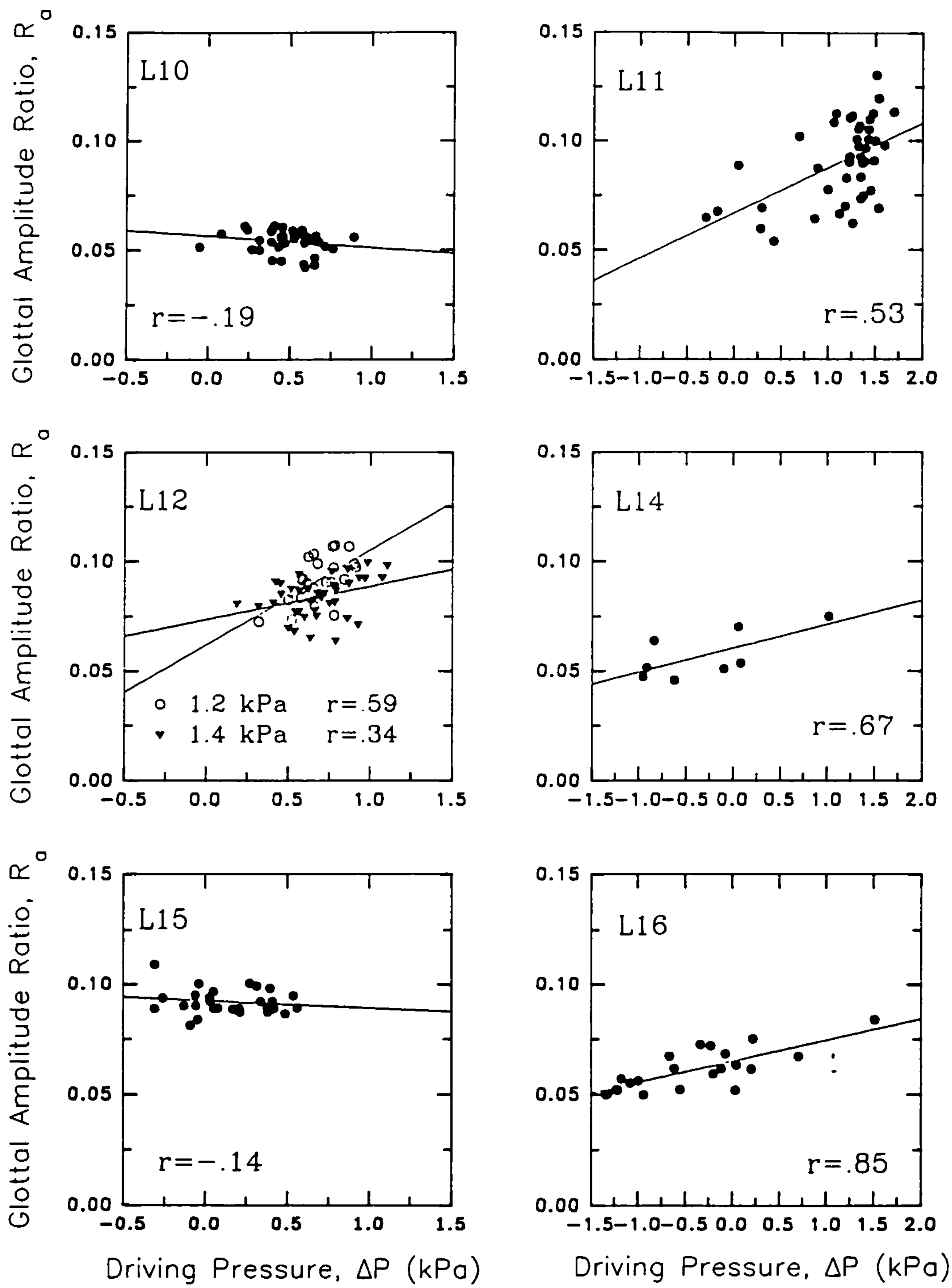

FIG. 8. Glottal amplitude ratio R_a as a function of the acoustic pressure Δp measured below the vocal folds for six larynges. The glottal amplitude ratio is defined as the ratio of the maximum half-width of the glottis normalized to the mean subglottal pressure.

the pressure at this point is approximately the same magnitude as the mean pressure. This frequency (225 Hz) in the male singing voice does not represent one of the major involuntary register transitions. No interference, either constructive or destructive, between P_{sub} and vocal fold vibration would be anticipated here.

Figure 10 shows data from the same subject with the F_0 at 307 Hz. Glottal opening is now coincident with the

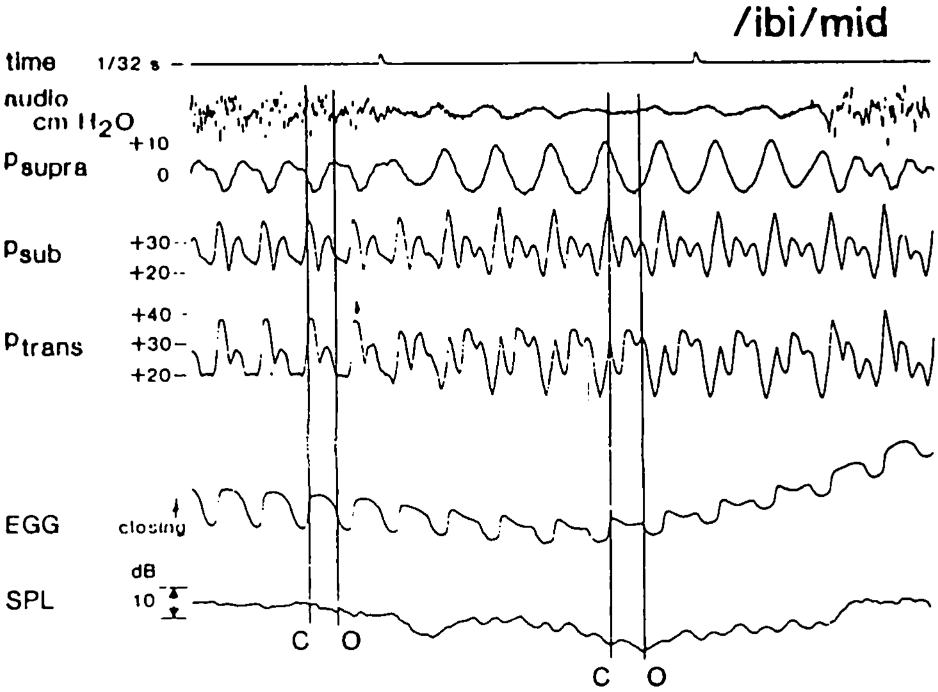

FIG. 9. Pressure signals recorded at several points along the vocal tract including the subglottal space in a human subject. EGG signal is used to mark moment of opening and of closure. F_0 is approximately 215 Hz. (From Miller DG and Schutte HK[22])

secondary pressure peak after the maximum at closure. The pressure is reported as 5.3 kPa, significantly higher than the pressure at opening in Fig. 9. The F_0 is approaching the range at which an involuntary register adjustment occurs (especially in the untrained voice). If we assume that the subglottal formant frequency was 510 Hz (13), the ratio between the F_0 (307 Hz) and the subglottal formant is approximately 0.6 (F_0 = $3/5F_1'$). This is the

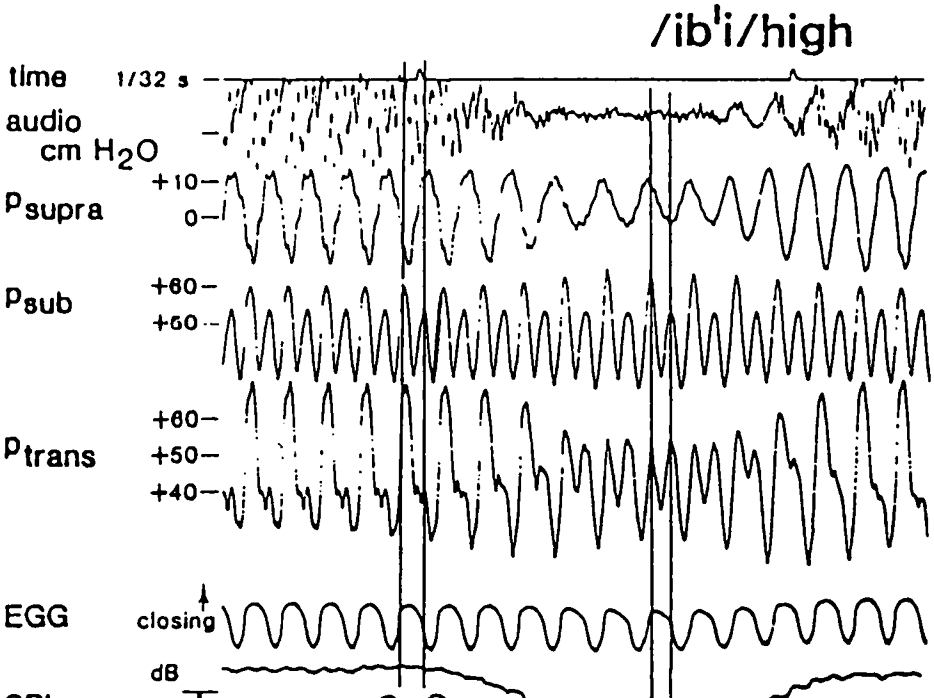

FIG. 10. Pressure signals recorded at several points along the vocal tract including the subglottal space in a human subject. EGG signal is used to mark moment of opening and of closure. F_0 is around 307 Hz.
From: Miller, D.G. and Schutte, H.K. (1991). Effects of downstream occlusions on pressures near the glottis in singing. In Gauffin J, Hammarberg B, eds., *Vocal Fold Physiology*. San Diego: Singular; 91–98. Used with permission.

ratio that Titze describes as the condition for optimum positive interference.

CONCLUSION

Interactions between acoustic pressures in the subglottal airway and amplitude of vibration of the vocal folds have been quantified with the use of excised larynges. Changes in amplitude of vibration on the order of 50% to 100% were observed in a subset of the data presented. The increase was apparently the result of high pressures below the glottis just as the vocal folds were opening, followed by low pressures in the glottis during the opened phase. This agrees with theoretical predictions made by Titze (12). The modest support of the data leads to the conclusion that: (a) more excised larynx experiments of this nature should be done with greater understanding of the acoustic environment in the subglottal space; (b) a greater attempt must be made to control abduction quotient, longitudinal tension and prephonatory half-width; and/or (c) a physical vocal fold model should be used to further test the veracity of the theory.

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