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PREPHONATORY LARYNGEAL AND CHEST WALL DYNAMICS

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The timing of prephonatory movements of the larynx, rib cage, and abdomen was examined in order to gain insight into the contribution of the vocal folds to the posturing of the chest wall. A simple stimulus-response paradigm was used in eliciting brief utterances—/ɑ/ and /hɑ/—from six adult males. Chest wall movements were observed using mercury strain gages while simultaneous electroglottographic and airflow records provided information about vocal fold behavior. Independence of prephonatory laryngeal and chest wall behavior was demonstrated. Laryngeal adjustment preceded the start of vocal fold oscillation by a constant amount of time, whereas the time of onset of the chest wall adjustment varied as a function of the utterance type. The qualitative characteristics of prephonatory chest wall posturing were unaffected by altering glottal configuration requirements. Rib cage enlargement occurred during postural adjustment while the vocal folds were abducted (in preparation for /h/). This implies that rib cage enlargement during prephonatory chest wall posturing was not a passive response to abdominal compression.

The importance of rapid and precise neuromuscular coordination of the ventilatory and laryngeal structures for efficient sound production is well documented (Bouhuys, 1974; Gould & Tanabe, 1975; Lieberman, 1968; Proctor, 1974), yet the integrated preparation of these two systems for speech and phonation is not well understood. Even the simplest phonatory event requires coordination of respiratory and laryngeal musculature. The chest wall must be displaced in such a way that sufficient alveolar pressure is generated and adequately regulated (Bouhuys, 1974; Hixon, Goldman, & Mead, 1973; Hixon, Mead, & Goldman, 1976; Ladefoged, 1960; Mead, Bouhuys, & Proctor, 1968). At the same time the laryngeal musculature must be adjusted to produce the intended vocal product (Baer, Gay, & Niimi, 1976; Cavagna & Margaria, 1965; Faaborg-Andersen, 1965; Hirose & Gay, 1973).

Baken, Cavallo, and Weissman (1979) reported that speakers, in response to an auditory stimulus, used a stereotyped pattern of chest wall adjustment before phonation of the vowel /ɑ/. A sudden oppositional deformation of the chest wall—involving expansion of the rib cage and contraction of the abdomen—almost always occurred approximately 100 ms before phonatory onset. Baken and Cavallo (1981) postulated that this particular form of posturing optimizes chest wall biomechanics for speech. Since volume is shifted from the abdomen to the rib cage as a result of this adjustment, it most likely accomplishes the diaphragmatic "tuning" described by Hixon et al. (1973) while stabilizing the rib cage in preparation for the aerodynamic events of speech (Mead, Hixon, & Goldman, 1974). Deformation of the chest wall from its tidal configuration appears to increase the capability of the rib cage and diaphragm-abdomen for ventilatory pressure regulation during speech.

The contribution of laryngeal action to the posturing of the chest wall remains largely conjectural. Gould and Okamura (1974) concluded that "the abdominal musculature plays a key role in producing the subglottal pressure necessary for phonation" (p. 358). If a rapid rise in subglottal pressure does accompany contraction of the abdomen, the vocal folds are implicated as important participants in the chest wall adjustment because they would need to be closely approximated for some period of time before phonatory onset. Vocal fold adduction in opposition to abdominal compression is not an unreasonable strategy for quickly raising alveolar pressure and could explain the significant expansion of the rib cage that typically occurs before phonation. Mead and his coworkers (1974) have in fact concluded that

> the shift from the relaxation configuration to relative expansion of the rib cage is apparently the result of the dominant action of the abdominal muscles rather than of inspiratory activity of the muscles acting on the rib cage. (p. 64)

Considering these hypotheses and the conclusions of Gould and Okamura (1974, p. 359) that "a close reflex interconnection [exists] between the laryngeal mechanoreceptors and abdominal musculature," it appears that the larynx may play an important role in the accomplishment of a target prephonatory chest wall posture. Hixon et al. (1976) have suggested that the distortion of the chest wall during speech production in the upright posture is accomplished in a manner similar to the isovolume maneuver. They reported "the adjustment involved is somewhat analogous to an isovolume maneuver in which the major abdominal effort serves to displace the abdominal wall inward and the rib cage outward in the face of a lesser expiratory effort by the rib cage wall" (p. 349). This

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position implies that the vocal folds are closely approximated during the postural adjustment of the chest wall. Such a conclusion seems tenuous, however, since vocal fold involvement in the posturing of the chest wall would, of necessity, complicate the already complex laryngeal adjustments needed for efficient vocal fold oscillation. If the vocal folds were to close against the intrusion of the abdominal volume into the thorax before speech, phonatory function would most likely be compromised and the problems of neuromuscular control confronting the speaker might be enormous.

To evaluate the importance of laryngeal action for the posturing of the chest wall, vocal fold position at utterance onset was varied in this experiment. The syllable /hɑ/ was selected since both high-speed cinematography (Werner-Kukuk & von Leden, 1970) and fiberoptic endoscopy (Sawashima, 1968) have demonstrated that the glottis is open during initial /h/ production. In addition, it has been demonstrated that interarytenoid muscle activity is low and posterior cricoarytenoid muscle activity is high during initial /h/ production (Hirose & Gay, 1973). Production of /hɑ/, then, provided a means of exploring whether or not glottal closure is important to the oppositional displacement that seems to be such a regular feature of prephonatory chest wall behavior. By extension, the /hɑ/ condition also explored the likelihood that rib cage movement during this adjustment is actively generated. If a significant rib cage expansion is observed in the absence of complete glottal closure, then it is likely that such enlargement involves active contraction of the inspiratory rib cage musculature. If, on the other hand, a patent glottis abolishes or severely attenuates rib cage expansion, it is likely that such enlargement was mainly the passive response of the rib cage to intrusion of the abdominal volume into the thorax with the creation of a significantly positive intrapulmonary pressure.

This investigation was designed to explore whether the prephonatory chest wall posturing previously observed is associated with preparation for *phonation* or whether it precedes any *speech* onset, including voiceless ones. It was also intended to provide data for drawing inferences concerning two other, closely related questions:

1. Must the glottis be closed in order to posture the chest wall?
2. Is the rib cage expansion during the chest wall adjustment the result of an active response of the rib cage musculature or is it a passive displacement of the rib cage caused by abdominal compression and diaphragmatic elevation?

METHOD

Subjects

The experimental subjects were six men between the ages of 23 and 30 years, mean age 26:2 (years:months). None had a history of respiratory, phonatory, or speech disorder and none had professional voice training. Pertinent subject characteristics are summarized in Table 1.

TABLE 1. Summary of pertinent subject characteristics.

Subject	*Age (yrs:mos)*	*Height (cm)*	*Weight (kg)*	*Vital capacity (l)*
1	26:4	184.7	78.3	5.51
2	23:10	174.5	75.6	4.70
3	23:4	176.5	69.8	4.96
4	30:0	182.5	96.3	5.59
5	28:11	182.3	84.6	5.47
6	24:11	174.5	74.3	4.60
Mean	26:2	179.2	79.8	5.14
(*SD*)	(2:8)	(4.5)	(9.4)	(0.44)

Experimental Tasks

The experiment had two phases. During one, subjects were required to produce /ɑ/, whereas in the other they produced /hɑ/ as quickly as possible after the onset of each of a series of 110-Hz stimulus tones. The /ɑ/ task provided baseline data on the temporal relationship of chest wall and laryngeal adjustments. The /hɑ/ task showed whether that temporal relationship was different when the vocal folds had to be kept separated for some time after speech onset. Comparison of the timing data from these two tasks was used to determine if chest wall posturing is related to speech onset or only to phonatory initiation. The qualitative aspects of chest wall movements during the two tasks served as the basis for inferences concerning the importance of glottal closure to chest wall adjustments.

Instrumentation

The instrumentation used in this study is diagrammed in Figure 1. Movements of the rib cage and abdomen were tracked by mercury strain gages using the same methods previously employed by Baken (1977), Baken and Cavallo (1981), Baken et al. (1979), and Baken, McManus, and Cavallo (1983). Laryngeal activity was monitored simultaneously by a Frøkjaer-Jensen EG-830 electroglottograph (EGG) that provided a dc output. This is the signal referred to by Fourcin (1974, 1981) as G_x. Airflow data were gathered to help resolve any ambiguities in the record of glottal activity (Rothenberg, 1981; Titze & Talkin, 1981). A Fleisch #3 pneumotachograph fitted to a Bennett "Benefit" anesthesia mask was the primary airflow transducer. The pressure drop across the pneumotachograph was sensed by a Validyne 2-cm H_2O differential pressure transducer and Validyne CD-15 carrier demodulator. A bias airflow of 350 ml/s was introduced into the mask to counter the effects of the increased ventilatory dead space. A Knowles XL-9073 probe microphone mounted perpendicular to the airflow at the distal port of the pneumotachograph transduced vocal responses.

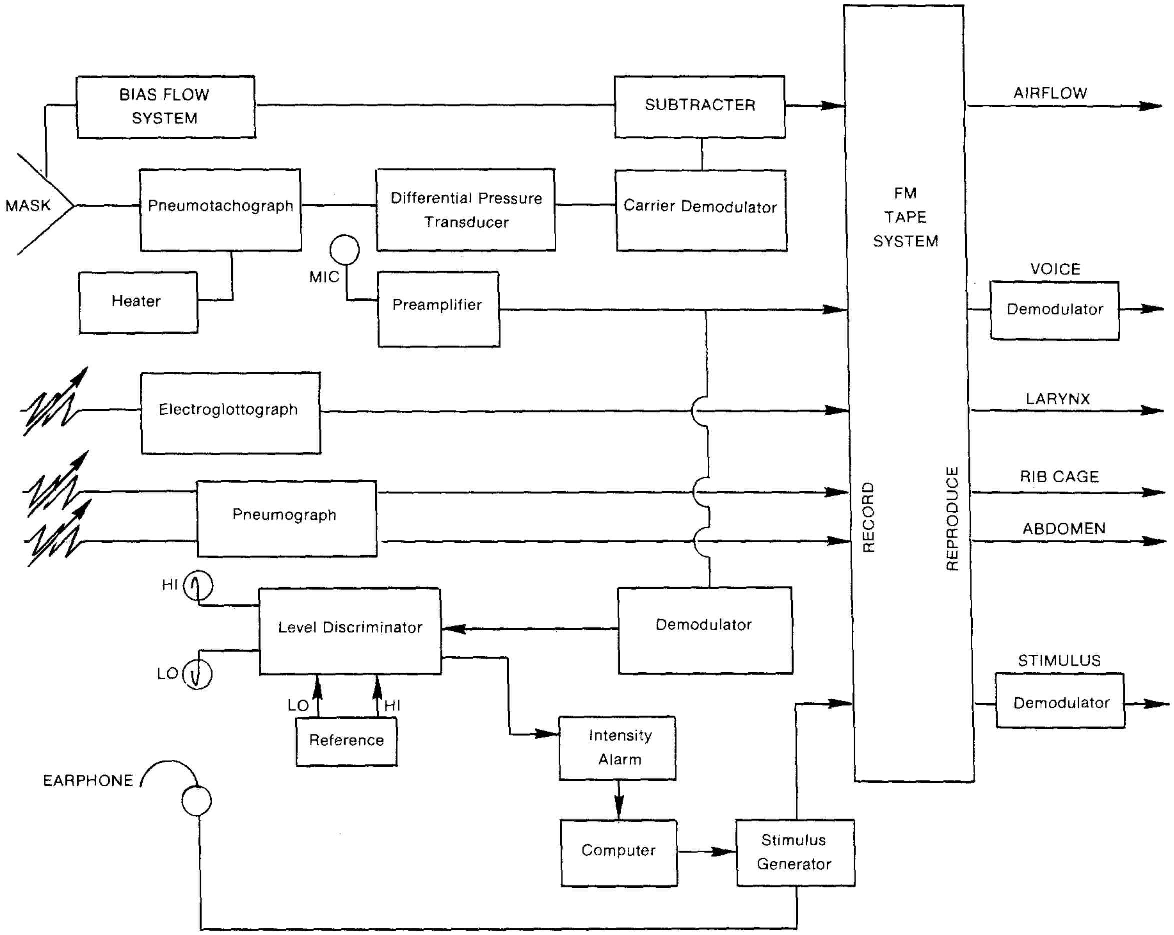

FIGURE 1. A diagram of the instrumentation used in this study.

Intensity control. A visual feedback system helped subjects maintain vocal intensity within a narrow range of the target intensity. The vocal signal was amplified, rectified, and smoothed before being applied to a level discriminator that compared it to two reference voltages representing ±2 dB SPL around the subject's comfortable loudness level. Vocal signals exceeding the upper reference voltage triggered a "too loud" light, whereas those below the lower reference voltage failed to turn off a "too soft" lamp. Subjects were instructed to keep both loudness indicators off during their phonation. Subject responses falling outside acceptable intensity limits activated a tone generator which marked the FM tape recording of the trial as unacceptable for analysis.

Procedure

Subjects were seated and the mercury strain gages were positioned across the anterior chest wall at the level of the umbilicus and nipples. EGG electrodes were placed approximately 1 cm lateral to the thyroid angle (Lecluse, Brocaar, & Verschuure, 1975). Adequate electrode placement was verified by observing the EGG signal during phonation of /ɑ/ on an oscilloscope. A head rest was used to stabilize subject position and minimize movement artifacts in the EGG record.

After an earphone was fitted to the subject's right ear, he was instructed to produce /ɑ/ at a comfortable loudness level (CL) as quickly as possible whenever he heard a stimulus tone. The amplitude of responses to 10 successive, randomly spaced stimuli was digitized by a Cromemco D+7A analog-to-digital converter and the results used by an Altair 8800b computer to determine the reference voltages representing the 2-dB SPL intensity tolerance limits for that subject for the entire experiment. Each subject then performed an isovolume maneuver (Hixon et al., 1973) from a relaxed (quasi-resting expiratory level) position with his mouth open. This provided information about relative displacements of the chest wall components, transducer output for zero airflow, and EGG representation of glottal closure.

Following the preliminary testing, the subject was instructed that he would hear a series of tones and that he was to produce the target utterance (/ɑ/ or /hɑ/, depending on which task was being examined) as quickly as possible after the onset of each stimulus. The task was demonstrated and the subject was given an opportunity to practice it using the intensity-feedback lamps. Order of testing of the /ɑ/ and /hɑ/ tasks was randomly determined for each subject.

Twenty stimuli, triggered on a random schedule by the computer, were presented for each task. The computer, via the A/D converter, also tracked airflow and was programmed to allow a minimum of three ventilatory cycles between stimuli to permit recovery of the ventilatory system after a phonatory response. Random spacing of the stimuli prevented a subject from predicting and preparing for the occurrence of the next stimulus. It also ensured that responses would be obtained throughout the eupneic tidal volume range. A 5-min rest period separated the /ɑ/ and /hɑ/ tasks.

Data Acquisition and Analysis

Stimulus tones, vocal responses, movements of the rib cage and abdomen, EGG, and airflow data were recorded on a Hewlett-Packard 3955 multitrack FM recording system at a tape speed of 19 cm/s. Data were played back at half-speed, and writeouts were prepared at a paper speed of 10 cm/s using a Narco Biosystems Physiograph-6 pen recorder. Effective writeout speed thus was 20 cm/s. A Houston Instruments Hi-Pad digitizer was used to determine the position along the time axis of the following points in the graphic record of each response: stimulus onset, voice onset, prephonatory change in rib cage hemicircumference, abdominal hemicircumference, and EGG output.

For the purposes of this study, these points were defined as a distinct change in slope of the appropriate trace during the period just before phonation. In a few cases the slope change was obvious but gradual, making the exact point in time of its occurrence difficult to establish by visual inspection alone. In these instances lines were drawn to extend the pre- and postinflection traces to a point of intersection then considered to be the temporal locus of the slope change. As a reliability check on the measurement procedure, a graduate student in speech pathology, who was unfamiliar with the experiment, used the same measurement techniques and independently remeasured the point of onset of rib cage, abdominal, and EGG changes in the period immediately following randomly selected stimuli. Pearson product-moment correlations of +.994 (rib cage), +.913 (abdomen), and +.997 (EGG) were obtained when the investigators' and student's measures were compared.

Data were stored and analyzed by the Altair microcomputer. Because the microphone was approximately 33 cm from the vocal folds, features of the acoustic signal were temporally offset from their correlates in the EGG trace by the time required for propagation of the acoustic wave through the vocal tract and face-mask system. Given the velocity of sound in air, the lag was estimated to be only about 0.9 ms. This constant discrepancy was considered negligible and was ignored.

RESULTS

/ɑ/ PRODUCTIONS

Pattern of Chest Wall Response

A typical prephonatory chest wall adjustment involving abdominal compression and rib cage expansion (Figure 2) was consistently observed in this investigation, confirming earlier reports (Baken et al., 1979; Baken & Cavallo, 1981) of a qualitatively invariant prephonatory chest wall postural adjustment. This chest wall movement pattern characterized the response to 99% of the stimuli delivered during the /ɑ/ condition. Subject 4 accounted for the other 1% of responses, in which diminution of both chest wall components was observed.

Temporal Relationship of Laryngeal and Chest Wall Adjustments

Rib cage, abdominal, and laryngeal adjustment times (ATs) were measured from the onset of adjustment to the moment of phonatory onset. The mean abdominal AT was 82.7 ms (SD = 22.9), whereas the corresponding mean rib cage AT was 87.4 ms (SD = 19.6). The small difference in the mean duration of the rib cage and abdominal adjustments was not statistically significant (t = .258). Figure 3(A) shows a subject response in which the onset of rib cage and abdominal adjustments occurred essentially simultaneously. The lack of a statistically significant difference in group means must not be allowed to obscure the importance of the numerous individual instances (75 of 116) in which the rib cage adjustment preceded movement of the abdomen, as in Figure 3(B), which shows a rib cage lead of about 38 ms. Occasionally, (27 of 116 instances) abdominal movement began before the rib cage adjustment (Figure 3C). Although simultaneous adjustment of the chest wall components was observed in only 14 of 116 instances, the onset of rib cage and abdominal movements occurred within ±2 ms for 48% of all /ɑ/ productions, demonstrating the tendency for an essentially simultaneous thoracoabdominal adjustment.

The mean laryngeal AT was 115.1 ms (SD = 36.1). The difference between mean laryngeal and rib cage or abdominal ATs was found to be statistically significant [$t = -4.08, df = 5, p < .01$ (rib cage); $t = -3.16, df = 5, p < .05$ (abdomen)]. Summarized in Table 2 are the mean onset time data for the laryngeal and chest wall adjustments.

/hɑ/ PRODUCTIONS

Pattern of Chest Wall Adjustment

The record shown in Figure 4 is typical of the data for /hɑ/ productions. Oppositional displacement of the rib cage and abdomen was again the predominant pattern,

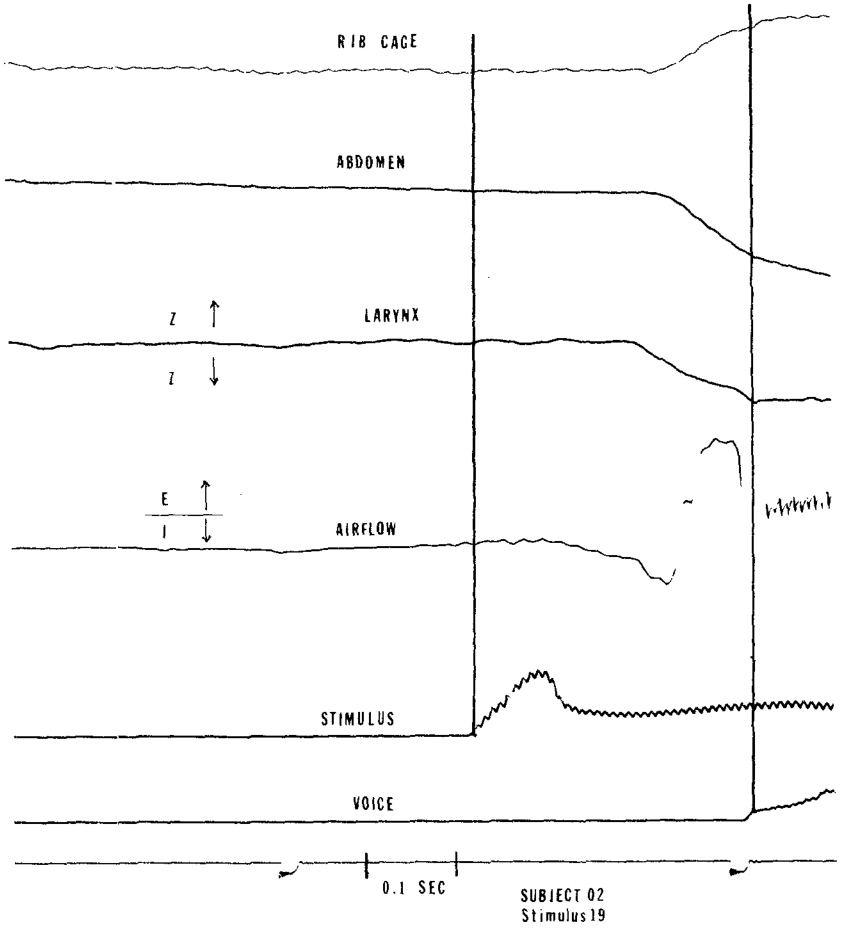

FIGURE 2. A typical oppositional chest wall movement pattern before /ɑ/ production. Upward deflection of the rib cage and abdominal traces represents increased thoracoabdominal size. Upward movement of the laryngeal trace indicates increased trans-neck impedance (Z). Zero airflow is represented by a horizontal line. Expiratory (E) and inspiratory (I) airflows are shown above and below zero, respectively.

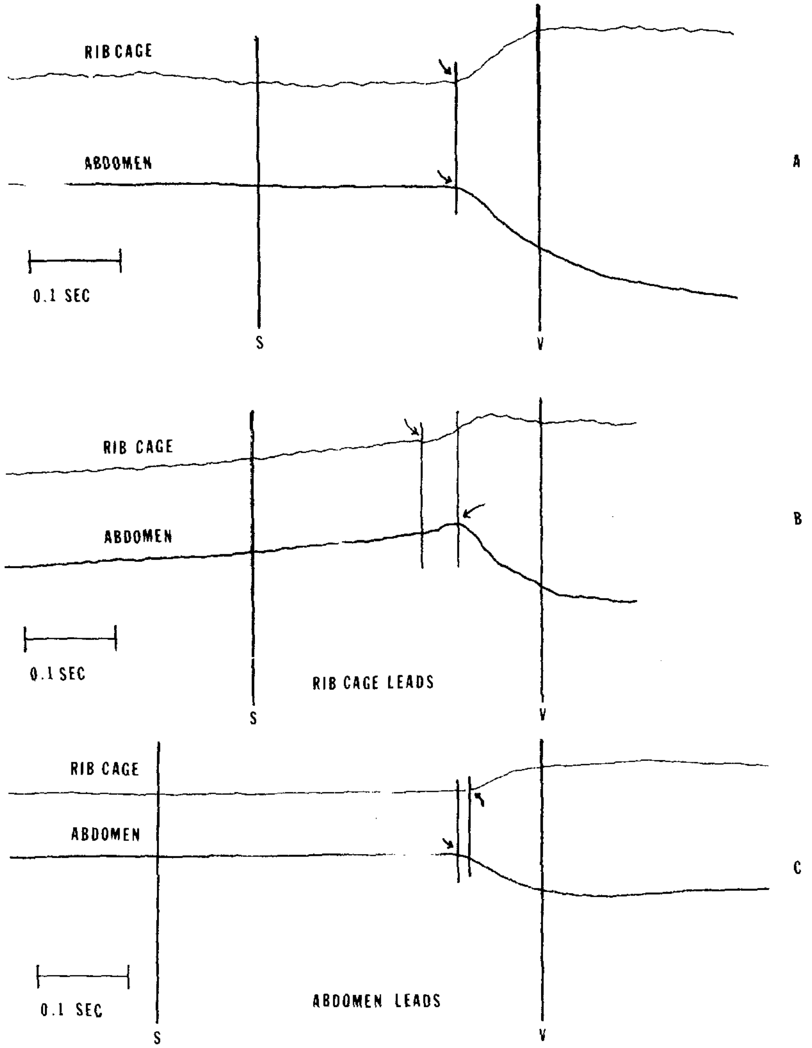

FIGURE 3. Representative examples of prephonatory oppositional chest wall adjustments in which (A) rib cage and abdominal movements occurred essentially simultaneously, (B) rib cage movement preceded the adjustment of the abdomen, and (C) rib cage movement occurred after the onset of abdominal movement.

TABLE 2. Mean rib cage, abdominal, and laryngeal adjustment times: /ɑ/ condition.

	Mean prephonatory adjustment times (ms before voice onset)		
Subject	*Rib cage*	*Abdomen*	*Larynx*
1	76.8	69.0	128.5
2	96.0	89.7	125.9
3	122.8	124.9	172.0
4	71.2	59.9	65.5
5	85.0	75.0	101.6
6	72.6	77.6	96.8
Mean	87.4	82.7	115.1
(*SD*)	(19.6)	(22.9)	(36.1)

occurring in response to 92.5% of all stimuli. Although this pattern was observed less frequently than before /ɑ/ production for these subjects, the value is in close agreement with that obtained for /ɑ/ productions by Baken et al. (1979). The qualitative nature of the chest wall adjustment before /hɑ/ was not different from that of chest wall movements before /ɑ/. Of particular interest in Figure 4 is the observation of rib cage enlargement in the presumed absence of significant vocal fold adduction, since a slight increase in transelectrode impedance is evident from the electroglottographic trace.

Temporal Relationship of Laryngeal and Chest Wall Adjustments

Individual mean ATs for the rib cage, abdomen, and larynx before /hɑ/ production are presented in Table 3. The mean abdominal AT was 99.5 ms (*SD* = 19.8) and the mean rib cage AT was 106.7 ms (*SD* = 15.0). The mean laryngeal AT observed was 106.8 ms (*SD* = 28.2). Although the grand means of the rib cage and laryngeal data are essentially identical, the adjustment of the rib cage preceded the onset of a laryngeal adjustment for Subjects 3, 4, and 6. Even Subjects 1, 2, and 5, whose mean ATs did not betray this tendency, demonstrated individual instances of rib cage precedence.

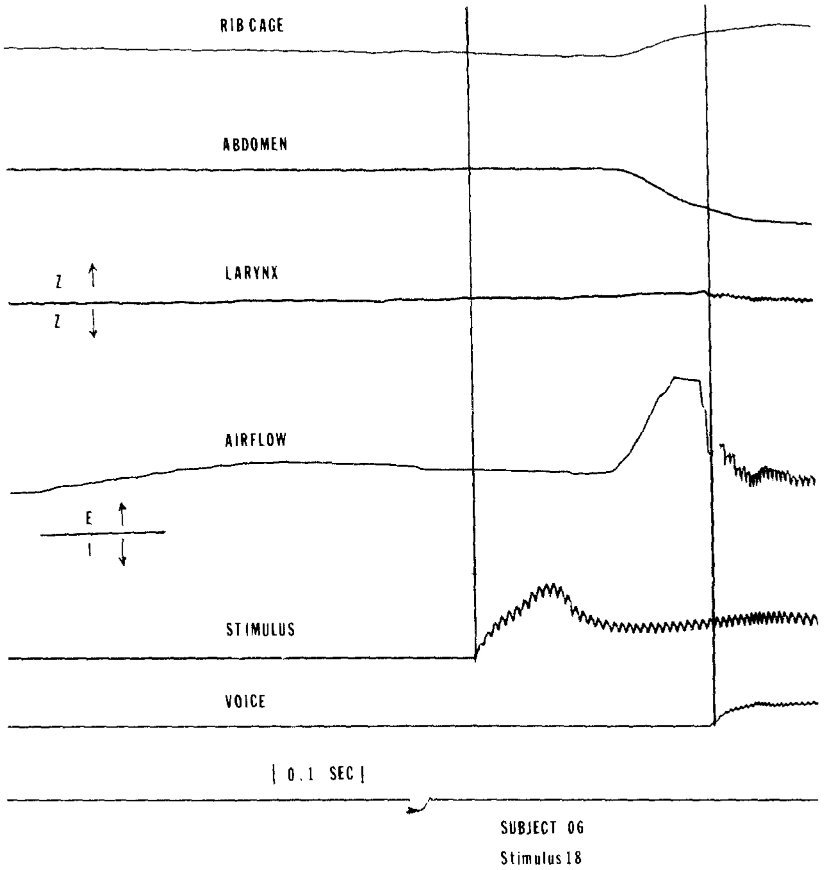

FIGURE 4. A typical chest wall response before /hɑ/ production. Upward deflections of the rib cage and abdominal traces represent increased thoracoabdominal size. Upward movement of the laryngeal trace indicates increased trans-neck impedance (Z). Zero airflow is represented by a horizontal line. Expiratory (E) and inspiratory (I) airflows are shown above and below zero, respectively.

TABLE 3. Mean rib cage, abdominal, and laryngeal adjustment times: /hɑ/ condition.

	Mean prephonatory adjustment times (ms before voice onset)		
Subject	*Rib cage*	*Abdomen*	*Larynx*
1	94.7	80.0	131.5
2	119.6	119.9	150.5
3	127.3	124.9	86.3
4	111.9	99.2	88.5
5	92.4	76.9	103.7
6	94.3	95.9	80.4
Mean	106.7	99.5	106.8
(*SD*)	(15.0)	(19.8)	(28.2)

Timing of Laryngeal and Chest Wall Adjustments as a Function of Utterance Type

An evaluation of the effect of phonetic context on the onset of prephonatory laryngeal and chest wall adjustments was made by comparing the mean AT values for the rib cage, abdomen, and larynx before /ɑ/ and /hɑ/ productions (Tables 2 and 3). Comparable mean laryngeal ATs were observed for the production of /ɑ/ (115.1 ms, *SD* = 36.1) and /hɑ/ (106.8 ms, *SD* = 28.2). The small difference in the mean laryngeal ATs for the two tasks was found to be statistically nonsignificant (t = .493). Such temporal stability was not observed for the two chest wall components across utterance tasks, however. The mean onset of the rib cage (87.4 ms, *SD* = 19.6) and abdominal adjustments (82.7 ms, *SD* = 22.9) before voice onset under the /ɑ/ condition occurred considerably later than under the /hɑ/ condition in which mean preutterance rib cage and abdominal ATs of 106.7 ms (*SD* = 15.0) and 99.5 ms (*SD* = 19.8) were observed, respectively. These differences in mean AT across utterance tasks were statistically significant [$t = -3.64$, $df = 5$, $p < .05$ (rib cage); $t = -2.63$, $df = 5$, $p < .05$ (abdomen)].

DISCUSSION

Independence of Laryngeal and Chest Wall Adjustments

Change in the EGG trace preceded onset of phonation by an interval that was relatively consistent for all productions. On the other hand, onset of chest wall adjustments occurred significantly sooner (with respect to phonatory initiation) for /hɑ/ than for /ɑ/. The stability of laryngeal adjustment onset and the difference in the start of chest wall posturing across the "speech" tasks was a direct consequence of designating voice onset as the anchor point for the temporal measures of this study. Had utterance onset been chosen as the timing anchor point instead, the temporal stability of laryngeal adjustment onset would not have been observed. Chest wall and laryngeal preparation for phonation seem to be independent phenomena performing distinct functions. The laryngeal adjustment is likely to be phonation-specific. The adjustment of the chest wall, on the other hand, probably serves to prepare the ventilatory system for the onset of any speech expiration, phonated or not. For the vowel /ɑ/, the onset of expiratory flow is at most only slightly earlier than the onset of phonation. The syllable /hɑ/, however, requires a much longer delay between the start of expiration (for the generation of turbulence within the glottis) and the start of vocal fold oscillation. In effect, the /hɑ/ task was only a convenient means of separating phonatory and utterance onset. The finding that the chest wall adjustment began substantially earlier for the production of /hɑ/ than for /ɑ/ indicates its importance for the onset of the expiratory event rather than for the phonatory process.

Mechanisms Responsible for Rib Cage Expansion

The results of this study failed to support the implication of the research of Mead et al. (1974), Gould and Okamura (1974), and Hixon et al. (1976) that rib cage enlargement during the prephonatory interval represents passive expansion in response to glottal closure at the time of abdominal compression. If the glottis can be open and if rib cage expansion can occur—as it often did—before the onset of abdominal contraction, the enlargement of the rib cage can only result from active contraction of inspiratory muscles. Supporting this assertion are the data alluded to in the caveat regarding the group mean differences between rib cage and abdominal ATs for the /ɑ/ task. As noted earlier, rib cage expansion usually preceded movement of the abdomen. In addition, the observations of rib cage movement before a laryngeal adjustment as well as rib cage expansion in the presumed absence of vocal fold approximation support the conclusion that active muscle forces are responsible for increasing the dimensions of the rib cage.

Since the latency of laryngeal adductor EMG activity in response to a sudden displacement of the abdomen in humans is on the order of 30–40 ms (Baer, 1979) and the mechanical response time of the laryngeal muscles is around 15 ms (Atkinson, 1978), a typical latency between abdominal contraction and rib cage expansion of approximately 45–55 ms would be expected if rib cage enlargement could be explained entirely as the response to a sudden abdominal contraction against a closed glottis. The results of this investigation demonstrate that this was not the case. Active rib cage expansion appears to be an essential part of prespeech chest wall dynamics. This is not to say that some amount of rib cage expansion might not be a passive response to abdominal-volume intrusion into the thoracic space, but it seems clear that rib cage displacement is very likely to be primarily an active process.

From a mechanical point of view it is well known that the dual-component chest wall system presents two degrees of freedom of movement during any ventilatory flow (Agostoni, Mognoni, Torri, & Saracino, 1965; Bergofsky, 1964; Konno & Mead, 1967). The finding of active rib cage expansion during chest wall posturing provides evidence that the two degrees of freedom of movement of the rib cage and abdomen are preserved during this adjustment. This finding has implications regarding the neural control of chest wall posturing. Specifically, a chest wall adjustment accomplished by exercising the two degrees of freedom of movement represents a relatively complex neuromuscular event requiring parallel and carefully coordinated active contraction of the rib cage and abdominal musculature.

Function of the Chest Wall Adjustment

Posturing of the chest wall for speech appears to be primarily a means of achieving a more effective biomechanical state for the efficient load-regulation of pressure during speech production, rather than simply a pressure generating maneuver. As Mead et al. (1974) have suggested, speakers most probably solve the pressure and configurational problems separately. If the chest wall adjustment were designed expressly for generating the subglottal pressure needed to support speech, it is unlikely that such a significant rib cage expansion would occur. A more efficient strategy for creating a rapid increase in subglottal pressure might involve diminution of both the rib cage and abdomen. However, since speech production involves sudden and extreme changes in glottal and supralaryngeal impedances, simply compressing both chest wall components would prove to be inefficient in the long run. Speech characteristically requires rapid alternations of high and low vocal tract impedance. Expansion of the rib cage stiffens the upper walls of the ventilatory system, whereas contraction of the abdominal wall similarly tunes the diaphragm (Hixon et al., 1976). The system is therefore much more efficient in compensating for the rapid changes in flow that occur during speech utterances—collapsing more quickly to maintain pressure during high flow events and resisting displacement when flow suddenly stops (as during plosives).

Of perhaps greater importance are the potential biomechanical benefits that accrue from an oppositional adjustment of the chest wall. The advantages of increasing rib cage size include the generation of increased recoil forces and elongation of the expiratory rib cage musculature which has the effect of increasing their contractile force. In addition, shifting volume from the abdomen to the rib cage probably accomplishes the diaphragmatic tuning described by Hixon and his co-workers (1976). The results are (a) a stiffened chest wall making the rib cage, abdomen, and diaphragm less prone to perturbation in the presence of the ballistic pressure pulses associated with speech; and (b) a system that is maximally prepared for the rapid shifts in expiratory flow that characterize speech production. In effect, rib cage enlargement before speech and phonation optimizes the "capacitance" of the air supply, demonstrating its importance to the regulation of a stable pressure supply during speech production.

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REFERENCES

AGOSTONI, E., MOGNONI, P., TORRI, G., & SARACINO, F. (1965). Relation between changes of rib cage circumference and lung volume. *Journal of Applied Physiology, 20,* 1179–1186.

ATKINSON, J. E. (1978). Correlation analysis of the physiological factors controlling fundamental voice frequency. *Journal of the Acoustical Society of America, 63,* 211–222.

BAER, T. (1979). Reflex activation of laryngeal muscles by sudden induced subglottal pressure changes. *Journal of the Acoustical Society of America, 65,* 1271–1275.

BAER, T., GAY, T., & NIIMI, S. (1976). Control of fundamental frequency, intensity and register of phonation. *Haskins Laboratories: Status Report on Speech Research, SR-45/46,* 175–185.

BAKEN, R. J. (1977). Estimation of lung volume change from torso hemicircumferences. *Journal of Speech and Hearing Research, 20,* 808–812.

BAKEN, R. J., & CAVALLO, S. A. (1981). Prephonatory chest wall posturing. *Folia Phoniatrica, 33,* 193–203.

BAKEN, R. J., CAVALLO, S. A., & WEISSMAN, K. L. (1979). Chest wall movements prior to phonation. *Journal of Speech and Hearing Research, 22,* 862–872.

BAKEN, R. J., MCMANUS, D. A., & CAVALLO, S. A. (1983). Prephonatory chest wall posturing in stutterers. *Journal of Speech and Hearing Research, 26,* 444–450.

BERGOFSKY, E. H. (1964). Relative contributions of the rib cage and the diaphragm to ventilation in man. *Journal of Applied Physiology, 19,* 698–706.

BOUHUYS, A. (1974). *Breathing: Physiology, environment and lung disease.* New York: Grune & Stratton.

CAVAGNA, G., & MARGARIA, R. (1965). An analysis of the mechanics of phonation. *Journal of Applied Physiology, 20,* 301–307.

FAABORG-ANDERSEN, K. (1965). Electromyography of laryngeal muscles in humans: Technics and results. *Aktuelle Probleme der Phoniatrie und Logopadie, 3,* 1–72.

FOURCIN, A. J. (1974). Laryngographic examination of vocal fold vibration. In B. D. Wyke (Ed.), *Ventilatory and phonatory control systems* (pp. 315–333). New York: Oxford University Press.

FOURCIN, A. J. (1981). Laryngographic assessment of phonatory function. In C. L. Ludlow & M. O. Hart (Eds.), *Proceedings of the Conference on the Assessment of Vocal Pathology* (ASHA Reports No. 11, pp. 48–53). Rockville, MD: American Speech-Language-Hearing Association.

GOULD, W. J., & OKAMURA, H. (1974). Interrelationships between voice and laryngeal mucosal reflexes. In B. D. Wyke (Ed.), *Ventilatory and phonatory control systems* (pp. 347–360). New York: Oxford University Press.

GOULD, W. J., & TANABE, M. (1975). The effect of anaesthesia of the internal branch of the superior laryngeal nerve upon phonation: An aerodynamic study. *Folia Phoniatrica, 27,* 337–349.

HIROSE, H., & GAY, T. (1973). Laryngeal control in vocal attack. *Folia Phoniatrica, 25,* 203–213.

HIXON, T. J., GOLDMAN, M. D., & MEAD, J. (1973). Kinematics of the chest wall during speech production: Volume displacement of the rib cage, abdomen and lung. *Journal of Speech and Hearing Research, 16,* 78–115.

HIXON, T. J., MEAD, J., & GOLDMAN, M. D. (1976). Dynamics of the chest wall during speech production: Function of the thorax, rib cage, diaphragm and abdomen. *Journal of Speech and Hearing Research, 19,* 297–356.

KONNO, K., & MEAD, J. (1967). Measurement of the separate volume changes of rib cage and abdomen during breathing. *Journal of Applied Physiology, 22,* 407–422.

LADEFOGED, P. (1960). The regulation of sub-glottal air pressure. *Folia Phoniatrica, 12,* 169–175.

LECLUSE, F. L. E., BROCAAR, M. P., & VERSCHUURE, J. (1975). The electroglottography and its relation to glottal activity. *Folia Phoniatrica, 27,* 215–224.

LIEBERMAN, P. (1968). Vocal cord motion in man. In A. Bouhuys (Ed.), *Sound production in man. Annals of the New York Academy of Science, 155,* 28–38.

MEAD, J., BOUHUYS, A., & PROCTOR, D. F. (1968). Mechanisms generating subglottic pressure. In A. Bouhuys (Ed.), *Sound production in man. Annals of the New York Academy of Science, 155,* 177–181.

MEAD, J., HIXON, T. J., & GOLDMAN, M. D. (1974). The configuration of the chest wall during speech. In B. D. Wyke (Ed.), *Ventilatory and phonatory control systems* (pp. 58–67). New York: Oxford University Press.

PROCTOR, D. F. (1974). Breathing mechanics during phonation and singing. In B. D. Wyke (Ed.), *Ventilatory and phonatory control systems* (pp. 39–57). New York: Oxford University Press.

ROTHENBERG, M. (1981). Some relations between glottal airflow and vocal fold contact area. In C. L. Ludlow & M. O. Hart (Eds.), *Proceedings of the Conference on the Assessment of Vocal Pathology* (ASHA Reports No. 11, pp. 88–95). Rockville, MD: American Speech-Language-Hearing Association.

SAWASHIMA, M. (1968). Movements of the larynx in articulation of Japanese consonant. *Annual Bulletin of the Research Institute of Logopedics and Phoniatrics* (University of Tokyo), *2,* 11–20.

TITZE, I. R., & TALKIN, D. (1981). Simulation and interpretation of glottographic waveforms. In C. L. Ludlow & M. O. Hart (Eds.), *Proceedings of the Conference on the Assessment of Vocal Pathology* (ASHA Reports No. 11, pp. 48–53). Rockville, MD: American Speech-Language-Hearing Association.

WERNER-KUKUK, E., & VON LEDEN, H. (1970). Vocal initiation, *Folia Phoniatrica, 22,* 107–116.

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