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PREPHONATORY CHEST WALL POSTURING IN STUTTERERS

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The possibility that prephonatory chest wall posturing is abnormal in stutterers was explored by observing rib cage and abdominal hemicircumference changes during the interval between the presentation of a stimulus and the production of /ɑ/ by a group of stutterers ($N = 5$). It was found that the patterns of chest wall adjustment for phonation were qualitatively identical in the stutterers and in a comparable group of normal men studied previously. There was, however, a significant difference in the way in which lung volume changed during the execution of the chest wall adjustment. This was considered to be indicative of delayed glottal closure among the stutterers rather than representative of a primary ventilatory disturbance.

In recent years research in stuttering has come to be characterized by a "retreat from the cortex" (Baken, 1975, p. 2). The affective and cognitive attributes of stutterers have received significantly less attention while brainstem and peripheral neuromotor mechanisms have become the focus of considerable research. As a result, it has become increasingly clear that disfluent speakers often demonstrate unusual patterns of activation of the intrinsic muscles of the larynx or peculiarities of ventilatory/vocal tract coordination.

A number of studies have shown, either directly or by inference, that abnormal behavior of the larynx, including inappropriate abductions and cocontraction of opposing muscles, characterizes many disfluent episodes (Adams & Hayden, 1976; Agnello, 1975; Conture, McCall, & Brewer, 1977; Freeman, 1975; Freeman & Ushijima, 1978; Wingate, 1969; Yoshioka & Löfqvist, 1980). Ventilatory discoordination also has been described as part of the stuttering complex. Not only has it been recognized historically that speech breathing is disrupted during the stuttering moment (Fletcher, 1914; Fossler, 1932; Robbins, 1919; Travis, 1927), but evidence that the timing of ventilatory events may be much less precise in stutterers than in fluent speakers also has accumulated (Blackburn, 1931; Metz, Conture, & Colton, 1976; Morley, 1937). Perkins, Rudas, Johnson, and Bell (1976) have interpreted their experimental findings to suggest that stuttering involves a disintegration of phonatory and ventilatory processes, while Metz et al. (1976) have demonstrated that cessations in chest wall diminution during stutterers' disfluencies are related to delays in laryngeal adductory activity.

We have shown recently that normal speakers use a stereotyped gesture to posture the chest wall just before phonation (Baken & Cavallo, 1980, 1981; Baken, Cavallo, & Weissman, 1979). The adjustment tends to modify certain biomechanical characteristics of the chest wall in a way that is likely to help optimize the ventilatory system for the speech to follow. Essentially, it consists of a contraction of the abdominal wall and an expansion of the rib cage just before phonation begins. The abdomen-to-rib cage volume transfer increases rib cage stiffness and "tunes" the diaphragm in a fashion consonant with the description of Hixon, Mead, and Goldman (1976). The result is to make the walls of the ventilatory system less prone to deformation by the rapid changes of airflow that are typical of speech. Therefore, the driving pressure will vary less as a function of articulatory events. The posturing behavior seems to be inborn and unlearned, since it is observable in the preverbal infant (Wilder & Baken, 1974) and in the congenitally deaf adult whose speech skills are only very poorly developed (Cavallo, Baken, Whitehead, & Metz, 1981).

Hypothetically, it is entirely possible that at least some of the ventilatory abnormalities seen during the stutterer's speech, such as the chest wall/laryngeal timing anomalies documented by Metz et al. (1976), represent responses to inadequate preparation of the ventilatory system for the utterance to be produced. By implication, then, it is not unreasonable to suppose that the chest wall preparatory adjustment of stutterers might be different in pattern from that used by normal speakers. The purpose of the research described in this report was to test that supposition by comparing clinically disfluent men to the group of normal subjects in whom the posturing gesture was originally characterized (Baken et al., 1979).

METHOD

Subjects

The normal men studied earlier by Baken et al. (1979) were compared to five adult male stutterers who served as research subjects for the present investigation. These five were selected from a larger group referred to the investigators by the professional staff of a major urban speech clinic at which all were clients. The subjects for the study were chosen to represent a broad range of stuttering severity as estimated by the referring speech-language pathologists (none of whom were otherwise involved in or aware of the purposes of the present study).

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No attempt was made to control for prior therapeutic experience. All the stutterers presented negative histories with respect to ventilatory or vocal tract pathology, with the exception of subject S3, who had experienced mild asthmatic episodes several years earlier. The gross chest wall structure of every subject was normal to visual inspection, and each subject's vital capacity was within 20% of the value predicted by his age and height (Kory, Callahan, Boren, & Syner, 1961).

For the purposes of this study, stuttering was defined as the presence of within-word disfluencies of the following types: part- or whole-word repetitions, sound prolongations, or broken words as defined by Conture et al. (1977). In addition, inappropriate between-word pauses were also considered disfluencies when terminated by explosive onset of the following phone.

At the time of testing, each subject was asked to read the first 1½ paragraphs (174 words) of the *Rainbow Passage* (Fairbanks, 1960). Two experienced evaluators (the first author and a judge not otherwise involved in the study) later listened to a recording of this reading and independently tabulated the frequency of the several stuttering types by each of the subjects. Interjudge reliability, evaluated by the agreement index of Sander (1961), was .92. The severity ranking of the subjects according to the frequency of disfluent episodes per 100 words differed somewhat from the clinicians' estimates of stuttering severity.

Pertinent characteristics of the stuttering subjects and of the normal speakers tested in the earlier study (Baken et al., 1979) are summarized in Table 1. The two groups differ somewhat in both age range and mean age, but it was not felt that the difference was great enough to affect the dependent variables of the study meaningfully. The groups were quite closely matched on measures of vital capacity and mean tidal volume.

Procedure

The specific experimental method and instrumentation used with the stuttering subjects are very similar to those employed with the fluent speakers (Baken et al., 1979) and therefore are only briefly reviewed here.

In normal circumstances, phonation is produced after an inspiration which is presumably planned to meet the speaker's estimate of the ventilatory requirements of the speech task. Any special posturing of the chest wall system for phonation therefore is likely to be integrated into the prephonatory inspiratory movement. For the purposes of the present study, isolation of the chest wall posturing gesture was achieved by requiring the subject to produce the vowel /ɑ/ immediately when stimulated at any point in the tidal breathing cycle. This had the effect of denying him the benefit of the usual prephonatory inspiration, thus forcing chest wall prephonatory preparation to be performed "out in the open" rather than as an integrated component of a complex inspiratory displacement. Subjects therefore were instructed to produce /ɑ/ as soon as possible when a 150-Hz stimulus tone (adjusted by the subject to comfortable loudness) was presented to the left ear. Although they were urged to phonate as quickly as possible, the intent of this instruction, as outlined above, was only to prevent assumption of a customary prephonatory lung volume. The task was not designed to evaluate minimal reaction time. Only productions of /ɑ/ that were perceptually judged by two experienced speech-language pathologists to be fluent (free of abnormal prolongation, repetition, or delayed and explosive onset) were considered for analysis.

A special monitoring circuit provided visual feedback that helped subjects maintain their phonations within 2.5 dB of a self-selected comfortable loudness. All subjects mastered the task during a practice session immediately prior to testing. For the testing of the normal subjects, stimuli were delivered by the experimenters according to a randomization schedule. For the stutterers, stimulus presentation was accomplished by an Altair 8800b microcomputer and Cromemco D+7A a/d converter that tracked the subject's ventilatory movements and presented a stimulus only when the ventilatory conditions randomly preselected by the computer program were

TABLE 1. Stuttering subjects' characteristics; also compared to normals[a] from an earlier study for mean and standard deviation.

Subject	*Age (yrs)*	*Vital capacity (liters)*	*Tidal volume (liters)*	*Clinician rating of severity*	*Disfluencies per 100 words (reading)*
Stutterers					
S1	27	5.1	.44	moderate	6.61
S2	40	5.4	.49	moderate/severe	40.51
S3	33	4.5	.53	moderate	3.45
S4	20	3.9	.34	severe	22.93
S5	41	4.3	.45	mild/moderate	2.01
$\bar{x}$	32.2	4.64	.450		15.01
SD	8.87	.61	.07		16.48
Normals					
$\bar{x}$	23.0	4.85	.421		
SD	2.6	.70	.08		

[a]Normal subjects tested in a study by Baken, Cavallo, and Weissman (1979).

met. In both cases, the intent was to obtain an adequate sample of all respiratory phases and lung volumes within each subject's eupneic tidal range. Sixty responses had been elicited from each of the normal speakers, but this number was reduced to 40 each for the stutterers to reduce the length of the test session, which was prolonged (sometimes greatly) by the need to obtain a reading sample. For the purposes of this experiment three ventilatory phases were recognized: inspiratory (I), expiratory (E), and static (S, when the chest wall was momentarily immobile during the transition from one ventilatory phase to the other). Lung volume was dichotomized into "high" (H) or "low" (L) when above or below 50% of the subject's mean tidal volume, respectively. Six ventilatory conditions thus were considered: I-H, I-L, E-H, E-L, S-H, and S-L.

Movements of the rib cage and abdomen were tracked using Whitney gages (Baken & Metz, 1973), while vocal responses were transduced by an accelerometer just lateral to the thyroid ala. These signals were recorded, together with the stimulus tone, on an H-P 3955 tape system. Readouts of the data, including an estimate of lung volume change (Baken, 1977) and a timing signal, were prepared on a Narco Biosystems Physiograph-6 pen recorder. Quantification of the variables of interest was achieved with a Houston Instruments Hi-Pad digitizer. Resolution of the measurement system was no worse than 1.25 msec, and tape recorder jitter was measured at less than .3% worst case.

Where appropriate, resultant data were evaluated for statistical significance using mixed-model ANOVAs with two within-subjects (phase and lung volume change) and one between-subjects (normal vs. stutterer) factors (Dixon & Brown, 1979, p. 556).

RESULTS

Response Distribution

Measurable responses were elicited by 476 out of 480 stimuli (99%) presented to the normal subjects and by 198 out of 200 stimuli (99%) delivered to the stutterers. Subjects failed to phonate after presentation of the other six stimuli. Table 2 summarizes the relative distribution of responses as a function of the status of the ventilatory system at the time of stimulus presentation. The unequal distribution of responses among the several categories is consistent with documented asymmetries of the tidal breathing cycle (Peters, 1969; von Euler, 1974). The stutterers' sample is quite comparable to the one previously obtained from normal speakers.

TABLE 2. Ventilatory status at the time of stimulus presentation (percentage of all responses).

Parameter	*Mean % of responses* *Stutterers (198 stimuli)*	*Normals*[a] *(476 stimuli)*
Lung volume		
>50% mean tidal volume	43.9	40.8
<50% mean tidal volume	56.1	59.2
Phase		
Inspiratory	35.8	35.3
Expiratory	51.5	51.5
Static	12.6	13.2

[a]From Baken, Cavallo, and Weissman (1979).

Pattern of Chest Wall Movements

Qualitatively, three different patterns of chest wall adjustment were observed in both stutterers and normals: (a) oppositional, in which the rib cage enlarged while the abdomen contracted during the adjustment period; (b) expiratory, in which both rib cage and abdominal size diminished; and (c) inspiratory, in which both hemicircumferences increased. Figure 1 shows examples of readouts of all three patterns of chest wall adjustment by a stutterer. In Figure 1A (the oppositional pattern) the stimulus tone was presented while inspiration was in progress and lung volume was about 225 ml above the rest expiratory level. For about 220 msec after the stimulus, inspiratory movements of the rib cage and abdomen continued unaltered: This is the latency time (LT). Suddenly, however, there was an oppositional displacement of the chest wall components—The abdominal size diminished while the rib cage expanded. This represents the chest wall adjustment. The adjustment time (AT), from the start of the adjustment maneuver until the onset of phonation, was about 100 msec in this example. The examples of expiratory (1B) and inspiratory (1C) chest wall adjustments have been marked similarly.

Table 3 shows the relative frequency of occurrence of each of these patterns of adjustment in both the normal and the stuttering groups. The t tests on arcsine transforms of the frequencies of occurrence (Winer, 1962, p. 28, 221) failed to establish any significant ($\alpha = .05$) intergroup differences. There was, however, a perceptible tendency for the stutterers to use an expiratory pattern somewhat more often than the normal speakers, and subject S4 clearly did so. The oppositional pattern was by far the most prevalent in both groups, accounting for just over 90% of all responses. The range of frequency of occurrence of this pattern also was similar in the two groups: 72–100% for the stutterers and 71–100% for the normal speakers. In terms of the general pattern of movement, it was not possible to distinguish an adjustment maneuver by a stutterer from one by a fluent speaker.

Timing

The absolute and relative durations of the stutterers' latency and adjustment periods are shown in Table 4. The mean latency time was very close to that of the normal speakers. While the stutterers' adjustment times were longer on the average than those of the normal

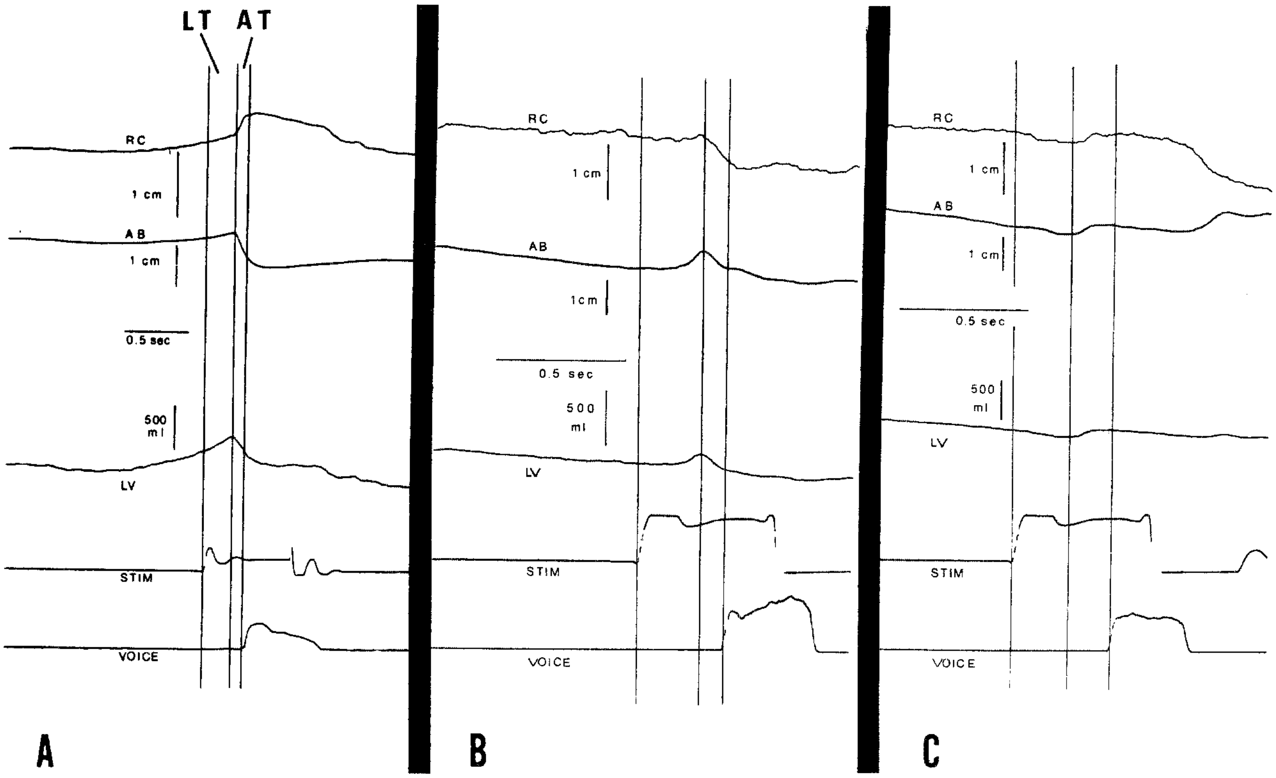

FIGURE 1. Chest wall adjustments. In each case the stimulus-response interval is divisible into two periods. Following presentation of the stimulus (leftmost vertical line), ventilatory movements continue essentially undisturbed. This is the latency time (LT), which ends with a fairly abrupt change in chest wall movement (center vertical line). The adjustment period (AT) follows and ends with phonatory onset (rightmost vertical line). The examples shown are by a stutterer but are not qualitatively different from readouts of normal speakers. A: oppositional displacement pattern; B: expiratory pattern; C: inspiratory pattern.

speakers, an analysis of variance (Dixon & Brown, 1979, p. 556) showed that the difference was not significant at the .05 level [$F(1, 11) = 4.02$, $p = .07$].

Lung Volume Adjustments

Typically, lung volume changed during the adjustment maneuver. In Figure 1A, for example, the lung volume diminished by about 130 ml while the chest wall adjustment was carried out. It had been shown in the earlier study of normal speakers (Baken & Cavallo, 1981) that a lung volume change was to be expected and that its direction and magnitude would be related to the ventilatory conditions prevailing at the time of stimulus presentation. This is, among the normal subjects, when inspiration was in progress during stimulus presentation,

TABLE 3. Relative frequencies of adjustment patterns, in percentages.

Subject	*Frequency of occurrence (%)* Oppositional	Expiratory	Inspiratory
Stutterers			
S1	98	2	0
S2	92	8	0
S3	93	5	2
S4	72	28	0
S5	100	0	0
$\bar{x}$	91.0	8.6	.4
SD	11.1	11.2	.9
Normals[a]			
$\bar{x}$	90.9	5.1	4.0
SD	12.4	6.9	7.0

[a]From Baken, Cavallo, and Weissman (1979).

TABLE 4. Mean latency (from onset of stimulus to start of adjustment) and adjustment times (from start of adjustment to onset of phonation), in msec.

Subject	*Latency* msec	% of total	*Adjustment* msec	% of total	*Total* msec
Stutterers					
S1	194.3	66	98.7	34	293.0
S2	264.0	72	100.2	28	364.2
S3	316.6	74	109.9	26	426.5
S4	273.1	57	202.9	43	476.0
S5	217.9	63	129.1	37	347.0
$\bar{x}$	253.2	66.4	128.2	33.6	381.4
SD	48.1	6.9	43.5	6.9	
Normals					
$\bar{x}$	244.8	72.0	93.3	28.0	338.1
SD	22.9	5.6	22.0	5.6	

[a]From Baken, Cavallo, and Weissman (1979).

lung volume increased during the adjustment maneuver. Conversely, if expiration had been under way, lung volume decreased. Data for the stutterers therefore were evaluated according to the six categories of ventilatory status, as had been done with the data of the normal subjects.

In Table 5 two differences between the groups are immediately apparent. First, the normal speakers' lung volume changed in conformity with the ventilatory phase in progress when the stimulus was delivered. If the subject had been expiring, lung volume change was negative (expiratory); if inspiring, however, it was positive (inspiratory). The stutterers, on the other hand, almost always lost lung volume immediately before phonating, as shown in Figure 1A. Thus, while the effect of prestimulus ventilatory phase on lung volume was highly significant [$F(2, 22) = 25.45, p < .001$], so was the phase × group interaction [$F(2, 22) = 11.65, p = .001$]. Second, the stutterers always lost more lung volume than the normal speakers. Ignoring the ventilatory phase effect, the difference in mean magnitudes of expiratory volume changes was significant [$F(1, 11) = 6.81, p = .025$]. It should be noted that lung volume at the time of stimulation also had a significant influence [$F(1, 11) = 21.70, p = .001$] on the lung volume adjustment, and there was a significant volume × group interaction [$F(1, 11) = 9.73, p = .01$]. Volume × phase and volume × phase × group interactions were not statistically significant.

DISCUSSION

The most prominent feature of prespeech chest wall posturing by both normal speakers and stutterers is an oppositional displacement of the rib cage and abdominal walls. This stiffens the chest wall system, tunes the diaphragm, and elongates the expiratory muscles of the rib cage, increasing their contractile efficiency. It was this feature that was of central interest in the present study. Given the suggestions in the literature that the general motor integration and nonspeech breathing patterns of stutterers might be different from normals (e.g., Murray, 1932; Schilling, 1960; Snyder, 1958), it seemed possible that the prespeech movements for preparation of the chest wall also might be different. The consequences of a failure to posture the ventilatory system efficiently could be very serious because of the diminished regulation that would result. In fact, however, there was no observable difference between the stutterers and the normal speakers in the organization of chest wall movements preparatory to production of isolated vowels, at least fluent ones.

It seems indisputable, on the basis of the literature (Fletcher, 1914; Fossler, 1932; Hill, 1944; Robbins, 1919; Seth, 1934; Travis, 1927; Van Riper, 1936) and common clinical observation, that peculiar ventilatory activity often occurs immediately before or during stuttering moments. Adams (1974, 1975) saw such ventilatory breakdowns as a primary manifestation of stuttering, implying that there may be an abnormality of neural control of chest wall structures. The results of the present study show that stutterers set up the mechanical characteristics of the chest wall for phonation (at least for isolated vowel production) in the same way that normal speakers do. There is no general disorganization of this function, and it seems unlikely that ventilatory abnormalities associated with stuttering episodes represent intrusive compensations for abnormal chest wall onset conditions.

A common observation, particularly in the earlier literature (e.g., Hill, 1944; Seth, 1934), is oppositional displacements of chest wall components at the time of disfluencies. If the glottis is closed during these events, then the oppositionality represents an isovolume maneuver, and rib cage expansion may be the passive result of abdominal contraction. If, however, the vocal folds are abducted, as they well may be during at least some types of disfluencies (Conture et al., 1977), then rib cage movement is due to active intercostal muscle contraction, as it has recently been shown to be in the prephonatory chest wall posturing of normal speakers

TABLE 5. Mean lung volume change (in milliliters) during the adjustment maneuver as a function of ventilatory status at the time of stimulus + = inspiratory; - = expiratory).

Group	*Inspiratory* High	*Inspiratory* Low	*Expiratory* High	*Expiratory* Low	*Static* High	*Static* Low	(Phase) (Volume)
Stutterers							
S1	23.6	52.6	10.0	−1.6	7.1	19.3	
S2	−230.3	−200.9	−228.8	−265.4	−108.3	–	
S3	−63.5	−89.3	−115.7	−72.0	−83.7	−69.4	
S4	−40.7	−20.2	−32.8	−42.1	−23.3	−41.0	
S5	−79.6	−83.7	−106.1	−91.1	−61.8	−104.2	
$\bar{x}$	−78.10	−68.30	−94.68	−94.44	−70.00	−48.82	
SD	93.70	93.79	91.30	101.36	74.79	52.25	
Normals[a]							
$\bar{x}$	+33.2	+95.6	−59.6	−22.5	−48.6	+51.0	
SD	35.7	43.4	40.8	43.0	26.4	71.6	

[a]From Baken, Cavallo, and Weissman (1979).

(Cavallo & Baken, 1982). If the rib cage muscles are active during the oppositional chest wall movements that accompany disfluent episodes, the maneuver is qualitatively the same as the chest wall preparation for normal phonation. It would be tempting to view it as an attempt (however inappropriate) to reset the system. Further research, especially simultaneous observation of laryngeal and chest wall behavior during disfluencies (on the order of the study done by Metz et al., 1976), may be useful.

The differences between the two groups in the magnitude and direction of lung volume change in the course of the adjustment maneuver do not necessarily signal a difference in the basic organization of prephonatory chest wall control. The chest wall is a dual-component system having two degrees of freedom of movement (Agostoni, Mognoni, Torri, & Saracino, 1965; Bergofsky, 1964; Konno & Mead, 1967). Therefore, differences in lung volume alteration could be due to differences in the relative contribution of the rib cage and abdominal components during oppositional movement. It is conceivable that, despite the gross normality of an oppositional pattern, stutterers might always use relatively more abdominal contraction than normal speakers do and thereby lose lung volume during the adjustment, even under conditions in which the normal speakers tend to gain lung volume. This study did not explore the relative magnitude of the chest wall movements, and thus this possibility cannot be tested with the available data. Inspection of the readouts, however, made it seem an unlikely explanation.

There is a more parsimonious hypothesis that explains the intergroup differences in lung volume change during the adjustment maneuver. Stutterers may not achieve glottal closure as quickly or as effectively as normal speakers, even before fluent utterances (Adams & Hayden, 1976; Agnello, 1975; Agnello & Wingate, 1972; Starkweather, Hirschman, & Tannenbaum, 1976). The mean adjustment time of the stutterers in this study was slightly longer than that of the fluent speakers. This may not reflect a sluggishness of the chest wall but rather a delay in readiness of the larynx for voice initiation. The mean time difference was too small to be statistically significant, but its effect may have been magnified in the lung volume change data. Because the expiratory driving pressure is presumably increasing to a phonatory level during the adjustment period, each additional unit of time that glottal closure is delayed may have a disproportionately large effect on the amount of air lost. The stutterers might expire enough to obliterate any lung volume increase they may have gained at the start of an adjustment due to the lingering influence of an inspiration in progress at the time of stimulation (which caused the normal speakers to gain volume during the adjustment period). Thus, the net lung volume change of the stutterers during the adjustment period was always expiratory. This explanation gains support from the observation that lung volume decrease is smaller after inspiratory-phase stimuli than after those delivered during expiration.

The findings of this study, then, tend to indicate that stutterers do not suffer a primary disorder that results in disorganization of the posturing of the walls of the ventilatory system for fluent phonation. Rather, the stutterers' loss of lung volume during chest wall adjustment, irrespective of the preadjustment status of the chest wall, argues in favor of the view that stutterers do not mobilize their vocal folds for phonation as quickly as fluent speakers do. This supports those models of stuttering that focus on the larynx.

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