635

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PHYSIOLOGICAL BASES OF ACOUSTIC LRT IN NONSTUTTERERS, MILD STUTTERERS, AND SEVERE STUTTERERS

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The simple reaction time paradigm, incorporating a variable foreperiod, was used to investigate relative contributions of the respiratory and laryngeal systems to mild and severe stutterers' prolonged acoustic laryngeal reaction time (LRT) values. Prephonatory kinematic data were analyzed in terms of frequency of initiation, timing, and organization of events executed to attain the functional physiological targets of respiratory inflation during foreperiods and phonation onset after foreperiods. Acoustic data replicated a previously observed composite stuttering severity and foreperiod effect on stutterers' acoustic LRT values. Kinematic data revealed that, in general, the mild stutterers demonstrated delayed initiation of respiratory events and appropriate organization of respiratory and laryngeal events while the severe stutterers demonstrated delayed initiation of laryngeal events and inappropriate organization of respiratory and laryngeal events. That is, kinematic data both account for group differences in acoustic LRT values as a function of foreperiod and support the notion that differential respiratory and laryngeal deficits underly mild and severe stutterers' prolonged acoustic LRT values.

The hypothesis that stutterers have difficulty controlling rapid initiation and termination of voicing is shared by most physiologically based models of the disorder (Adams, 1974, 1978; Schwartz, 1974; Van Riper, 1982; Wyke, 1971; Zimmerman, Smith, & Hanley, 1981). Two observations support this hypothesis. First, stutterers show greater frequency of dysfluency and less adaptation when reading aloud passages containing both voiced and voiceless segments than passages containing only voiced segments (Adams & Reis, 1971, 1974; Adams, Riemenschneider, Metz, & Conture, 1975). Second, physiological data, based on fiberoptic viewing of the vocal folds (Conture, McCall, & Brewer, 1977) and recordings of electromyographic (EMG) signals from intrinsic laryngeal muscles (Freeman & Ushijima, 1978; Shapiro, 1980), reveal evidence of abnormal laryngeal activity during stutterers' dysfluent utterances.

Activity of the laryngeal system is not independent of activity of the respiratory and articulatory systems. Furthermore, the degree of interdependency is a function of phonetic environment. However, all of the studies cited above measured only laryngeal activity during connected speech tasks. No doubt this is related to difficulty associated with simultaneous monitoring of events in all three systems. However, the conclusion reached by several investigators (cf. Brenner, Perkins, & Soderberg, 1972; Hutchinson, 1976; Perkins, Rudas, Johnson, & Bell, 1976) is that dysfluency of a stutterer in tasks requiring production of connected speech is likely related to a lack of coordination of activity both within and between the three systems. Consequently, it is not clear to what extent data reported in studies analyzing laryngeal activity during samples of connected speech reflect potentially disruptive effects of (a) stutterers' attempts to coordinate timing of vocal fold adduction and abduction with respiratory and articulatory dynamics, or (b) deficits confined to independent control of the larynx.

One method of investigating laryngeal activity during voice initiation and termination, while minimizing the effects of respiratory and articulatory dynamics, is the reaction time paradigm. For example, a simple reaction time task (i.e., incorporating one stimulus and one known response) requiring production of isolated, voiced vowels has been used by several investigators to compare laryngeal function of stutterers with that of nonstutterers (cf. Adams & Hayden, 1976; Cross & Luper, 1979; Cross, Shadden, & Luper, 1979; Reich, Till, & Goldsmith, 1981; Venkatagiri, 1981; Watson & Alfonso, 1982, 1983). The metric for between-group comparisons in these studies is onset or offset latency of the acoustic signal corresponding to vocal fold vibration relative to an external signal, for example a tone or light. We refer to measures of this latency as acoustic laryngeal reaction time, or LRT.

LRT values are not independent of the interaction of respiratory and articulatory activities with the larynx. With respect to the former, variability in prephonatory chest wall posturing described by Baken, Cavallo, and Weissman (1979) and by Baken, McManus, and Cavallo (1983) may affect laryngeal activity. With respect to the latter, Cross and Olsen (1982) described the effects of delays in executing jaw opening on LRT values for a vocalic response in 4 of 10 stutterers. Measures of thoracic and abdominal wall kinematics permit examination of the effects of respiratory events on LRT values. Effects of movements of the supralaryngeal articulators on LRT values can be minimized by incorporating a variable foreperiod (e.g., interval between presentation of a warning cue and a phonate cue) in the design of reaction time experiments. A variable foreperiod can provide subjects time to attain appropriate vocal tract shapes before pre-

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sentation of the phonate cue, thereby minimizing the effects of supralaryngeal activity on laryngeal activity.

Using the comparative potential of the reaction time paradigm, we completed a series of studies whose goal was to describe laryngeal behavior in stutterers and its relation to between-group acoustic LRT differences (Watson & Alfonso, 1982, 1983). Results of our first study, in which we recorded LRT values from nonstutterers and stutterers rated as mild-to-moderately severe, led to the hypotheses that (a) sufficiently long foreperiods can provide subjects time to prepare for the known response before presentation of the phonate cue, and (b) acoustic LRT values are related to stuttering severity. Consequently, in our second study, comparing nonstutterers with both mild and severe stutterers, we expected LRT values for all groups to improve as the foreperiod increased. However, we observed a composite stuttering severity and foreperiod effect on stutterers' LRT values. That is, mild, but not severe, stutterers' LRT values approached nonstutterers' values as the foreperiod increased from 100 ms to 3,000 ms. This suggests that mild and severe stutterers may be distinguished on the basis of their ability to execute "preparatory" events during the foreperiod and "initiation" events after the foreperiod.

The distinction between mild and severe stutterers' abilities to execute prephonatory respiratory and/or laryngeal events during and/or after the foreperiod is described in terms of the differential deficit hypothesis (Watson & Alfonso, 1983). Briefly, this hypothesis suggests that mild stutterers may have deficits in executing prephonatory events occurring during the foreperiod, while severe stutterers may have deficits executing these events both during and after the foreperiod. Alternatively, both groups may have similar deficits, but the magnitude of the deficit is determined by severity of the disorder.

In addition to its usefulness in comparative studies, the reaction time paradigm provides a potentially valuable method for describing physiological details of coordination among the articulatory, laryngeal, and respiratory systems (Alfonso, Watson, & Baer, 1984; Baer & Alfonso, 1984). Prosek, Montgomery, Walden, and Schwartz (1979), in an investigation of stutterers' prephonatory events in a reaction time task, failed to observe significant differences between stutterers' and nonstutterers' response latencies of "laryngeal region" surface EMG. However, acoustic LRT values were significantly different for those groups. Baken et al. (1983) reported no significant differences between stutterers and nonstutterers with respect to onset latency or patterns of prephonatory respiratory adjustments. However, their experimental task was not designed to elicit minimal reaction times nor did they simultaneously monitor laryngeal adjustments. Peters and Boves (1984) compared prephonatory electroglottographic (EEG) and subglottal pressure signals of stutterers and nonstutterers and reported a significant between-group difference in the frequency of occurrence of "unusual" pressure build-up patterns. However, they did not observe group differences in EEG signals. We know of no other reaction time studies that have systematically investigated the relative contributions of deficits in the execution of prephonatory respiratory and laryngeal events to mild and severe stutterers' prolonged acoustic LRT values.

In the study reported here, we continue our investigation of mild and severe stutterers' abilities to initiate phonation for the production of isolated vowels by recording respiratory and laryngeal kinematic data in parallel with the acoustic signal. One method of analyzing kinematic data describes subjects' success in attaining functional physiological targets during and after the foreperiod (Watson, 1983). The proposed functional target of laryngeal and respiratory events occurring during the foreperiod is inflation of the respiratory system, while the proposed functional target of events occurring after the foreperiod is the initiation of phonation. In our analysis, kinematic properties of interest include the frequency of initiation, timing, and organization of events executed to attain functional targets.

In summary, it is possible to determine the contributions of respiratory and laryngeal activities to stutterers' difficulty initiating voicing rapidly by minimizing contributions of articulatory dynamics and analyzing simultaneous respiratory and laryngeal kinematic data. The isolated vowel condition of the simple reaction time paradigm is appropriate for this endeavor. In addition, this paradigm is appropriate for testing the hypothesis that differential physiological deficits underlie mild and severe stutterers' prolonged acoustic LRT values. Accordingly, the purpose of this study is twofold. First, we describe selected kinematic properties of prephonatory respiratory and laryngeal events (e.g., frequency of initiation, timing, and organization) executed by nonstutterers and by mild and severe stutterers in attaining the functional targets of respiratory inflation and phonation onset. Second, we use the kinematic data to test the differential deficit hypothesis.

METHODS

Subjects

Six adult male subjects participated in this study: two nonstutterers and four stutterers. Stutterers were classified as mild or severe using procedures developed by Watson and Alfonso (1983). Specifically, group assignment was determined by results of objective and subjective evaluation of each stutterers' dysfluencies during conversational speech and speech during reading of a standard passage (Fairbanks, 1960). Objective evaluations of frequency and types of dysfluency were completed using procedures described in the Stuttering Interview (SI) (Ryan, 1974) and the Stuttering Severity Index (SSI) (Riley, 1972). As defined by these instruments, stuttering severity is determined by analyses of rate of speech, overall frequency of dysfluency, frequency of specific types of dysfluency, and magnitude of secondary stuttering behaviors. Subjective judgments of stutter-

TABLE 1. Summary of the results of stuttering severity analyses of samples of reading and conversational speech.

ID	Age (years: months)	SI		SSI		Subjective		Group
		Reading	Conversation	Reading	Conversation	Reading	Conversation	
JK	39:11	Mild	Mild	Mild–moderate	Mild	Moderate	Mild	Mild
KG	25:11	Mild	Moderate	Mild	Mild	Mild	Mild	Mild
DA	33:06	Severe	Severe	Severe	Severe	Severe	Severe	Severe
BA	22:07	Moderate	Severe	Moderate	Moderate–severe	Severe	Severe	Severe

ing severity were obtained from two certified speech-language pathologists. Taken together, these evaluations provide a comprehensive description of several behaviors correlated with stuttering severity (Van Riper, 1982).

Results of analyses of stutterers' speech samples, as well as group assignments, are summarized in Table 1. Differences between severity ratings of stutterers' reading and conversational speech suggest that certain stutterers achieve greater control over the speech system in one modality than in the other. Consequently, to insure appropriate grouping, two additional criteria were established for classifying the experimental subjects. The first criterion was agreement on two out of three analyses of conversational speech. The rationale for this criterion is that the conversational modality is more relevant to the experimental task than is the reading modality. The second criterion was that there be no greater than one category difference between severity ratings of reading and conversational samples. This criterion served as a check on the internal consistency of stuttering severity classifications. Following these criteria, we classified the experimental group as two mild and two severe stutterers. Subjects in the control and the two experimental groups were matched with respect to age.

Stutterers participating in this study differed with respect to therapeutic history. Subject KG recently had completed the Precision Fluency Shaping Program (Webster, 1974). Subject BA was enrolled in a therapy program developed by Shames and Florance (1980). While both programs emphasize easy onset of phonation, acoustic analyses verified that all subjects used the soft mode of vocal attack. Subjects DA and JK had not received therapy for several years prior to the experiment.

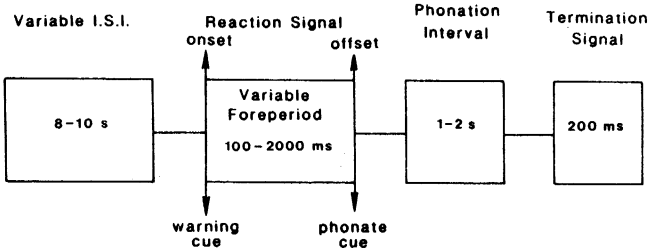

FIGURE 1. Schematic of the stimulus sequence used to obtain LRT data. Subjects were instructed to prepare for phonation at presentation of the warning cue and to initiate phonation at presentation of the phonate cue.

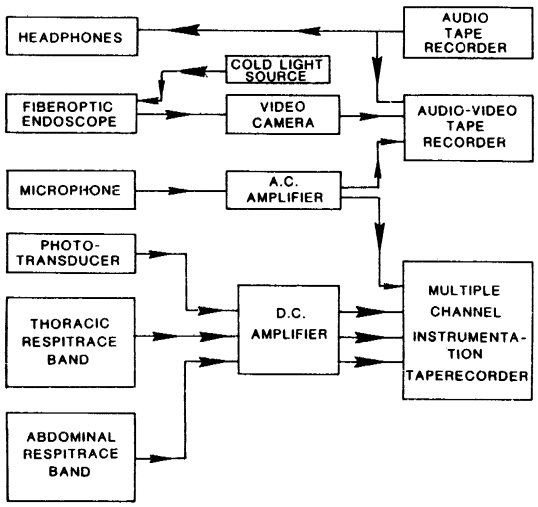

FIGURE 2. Schematic of instrumentation used to record simultaneous acoustic and kinematic data.

Test Stimuli

Figure 1 illustrates one sequence of stimuli used to obtain LRT responses. Each sequence was separated by an interstimulus interval (ISI) of 8 s to 10 s. ISIs of this duration encouraged subjects to breathe normally between responses. Thus, they may have been prevented from maintaining a voicing posture. The reaction signal consisted of the synthetic vowel /i/. Onset of the reaction signal served as the warning cue, and its offset was the phonate cue. Duration of the reaction signal (i.e., the foreperiod) varied from 100 ms to 500 ms in 100-ms increments, 700 ms to 1500 ms in 200-ms increments, and 1500 ms to 2000 ms in a 500-ms increment. Thus, LRT values were obtained at 11 foreperiods. Subjects phonated the isolated vowel /i/ during a response interval varying from 1 s to 2 s in 500-ms increments. A 200-ms "terminate phonation" signal, consisting of the synthetic vowel /a/, cued subjects to stop phonating. Each sequence was replicated two times. Four different randomized tests, consisting of two responses at 11 foreperiods, were output on audiotape using the Haskins Laboratories Pulse Code Modulation (PCM) system.

Instrumentation

Figure 2 is a schematic diagram of instrumentation used to obtain and record kinematic and acoustic data.

Subjects were seated in a dental chair with a support to restrict head movements. Prerecorded stimulus sequences were output from an audiotape recorder and presented simultaneously to the subject (binaurally through headphones at approximately 80 dB SPL), to an audio channel on the instrumentation tape recorder, and to one audio channel on the audio-video tape recorder. Subjects' acoustic responses were received by a unidirectional microphone and transmitted to a second audio channel on the instrumentation tape recorder and to the second audio channel on the audio-video tape recorder.

Vocal fold movements were monitored using transillumination instrumentation (Lisker, Abramson, Cooper, & Schvey, 1969). A fiberoptic endoscope, positioned approximately 6 cm above the glottis, served as the supraglottal light source. The subglottal photo transducer was attached to the anterior midline surface of the neck at the level of the cricoid cartilage. Output from the transducer was routed to a DC amplifier and recorded on a DC channel on the instrumentation tape recorder. Use of a fiberoptic endoscope as the light source for transillumination permitted simultaneous videotaping of vocal fold movement. The videotape record was used to verify that supraglottal structures (i.e., the epiglottis) did not block the light source and, therefore, that the transillumination signal truly represented vocal fold movements. Use of the fiberoptic endoscope as the light source for transillumination provided an unanticipated benefit. Specifically, blockage of a nasal passage by the optic bundle encouraged subjects to breathe through the oral passage. Thus, it was necessary to maintain the jaw in a lowered position sufficient for respiration throughout the experimental session, essentially eliminating potential effects of delays in significant jaw lowering on acoustic LRT values, as described by Cross and Olsen (1982).

Thoracic and abdominal wall movements were monitored using Respitrace instrumentation (Cohn, Watson, Weisshaut, Stott, & Sackner, 1977). Voltage output from each of two Respitrace coils was transmitted to DC amplifiers and recorded on DC channels of the instrumentation tape recorder. Finally, octal codes recorded on both video and instrumentation tapes provided a calibration signal permitting identification of response tokens and temporal alignment of data channels.

Experimental Procedures

Because we were interested in documenting habitual prephonatory respiratory and laryngeal behaviors, no constraints were placed on these behaviors during the ISI. Subjects were instructed to begin any preparatory activities they felt necessary for production of the response (i.e., "inflate your lungs, move your vocal folds together, place your jaw and tongue in the appropriate position") when and only when they heard the onset of the reaction signal. Next, they were instructed to begin phonation for production of the isolated vowel /i/, at comfortable pitch and loudness levels, immediately at offset of the reaction signal and to continue phonating until they heard the terminate signal.

After placement of the Respitrace bands, subjects were given 10 training sequences, including both short and long foreperiods. Ten trials were sufficient for all subjects to learn this relatively simple task. Twenty-two responses were recorded before insertion of the fiberscope. This procedure was followed to determine whether presence of the optic bundle in the vocal tract differentially affected LRT values for stutterers and nonstutterers. The average difference between stutterers' LRT values with and without the fiberscope was 21.6 ms, while the average difference for nonstutterers' LRT values was 33.8 ms. These values suggest that presence of the optic bundle increases LRT values an average of 26 ms. However, it is important to note that the magnitude of this effect was similar for both groups.

Following insertion of the fiberscope, response sequences were presented in four tests separated by an optional 2- to 3-min rest interval. Each test elicited two responses per foreperiod. Thus, a maximum of eight responses was recorded at each foreperiod.

Fluency Analysis

Procedures developed by Watson and Alfonso (1983) were used to insure that only fluent responses were analyzed. Those procedures evaluate response fluency using both perceptual and acoustic criteria. Perceptual criteria require that responses be judged as fluent by both experimenter and subject. Acoustic criteria require that no abnormalities be observed in the acoustic signal (e.g., isolated pitch pulses before the onset of continuous phonation). As a result, we analyzed 163 of 176 total responses for the nonstutterers (92%), 103 of 132 total responses for the mild stutterers (78%), and 129 of 176 total responses for the severe stutterers (73%). The smaller total number of responses for mild stutterers reflects the fact that four responses per foreperiod were obtained for one subject in the group.

Data Analysis

Simultaneously recorded acoustic and kinematic data were processed and analyzed using computer facilities at Haskins Laboratories. Figure 3 shows a tracing of the computer display of one response. The top record displays reaction and termination signals, the next record displays the transillumination signal, the third and fourth display thoracic and abdominal Respitrace signals, and the bottom record displays the speech acoustic signal. Note that prephonatory thoracic and abdominal movements were in opposition for this response. Abdominal compression began a short time before onset of the speech acoustic signal while thoracic compression began after onset of the speech acoustic signal. This pattern was observed in 18% of the responses for all subjects.

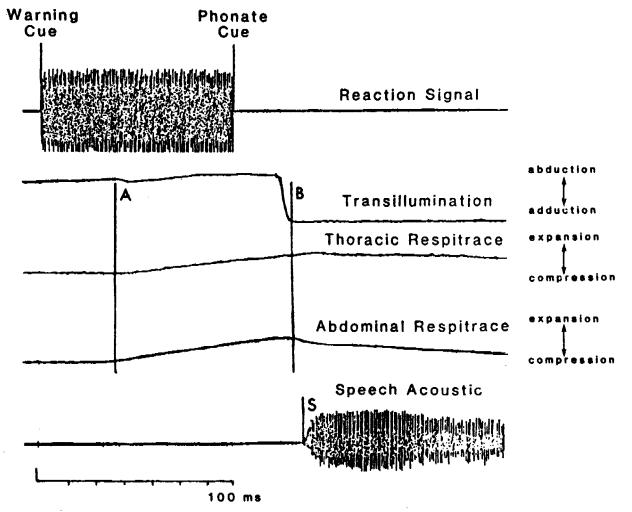

FIGURE 3. Example of simultaneous data channels for one response.

We evaluated both qualitative and quantitative kinematic properties of prephonatory respiratory and laryngeal events. The qualitative property, organization of those events, was evaluated in terms of their relative onset latencies during and after the foreperiod. Quantitative properties included frequency of initiation and onset latency of laryngeal and respiratory events during and after the foreperiod. The reference for temporal measures during the foreperiod was the warning cue, and the reference for measures after the foreperiod was the phonate cue.

Examples of moments used to obtain timing measures are illustrated in Figure 3. Acoustic reaction time was measured as the interval between reaction signal offset (labeled as the phonate cue) and onset of the acoustic signal (labeled point S). Onsets of respiratory expansion gestures were taken as the moment of marked upward deflection of the Respitrace signal. Onsets of respiratory compression gestures were taken as the moment of marked downward deflection of the Respitrace signal. Respiratory kinematic data were not reported for those responses in which deflections were questionable. An example of the onset latency of respiratory expansion during the foreperiod is illustrated as the interval between the warning cue and the vertical line A, corresponding to upward deflection of the Respitrace signals. An example of the onset latency of abdominal compression after the foreperiod is illustrated as the interval between the phonate cue and the vertical line B, corresponding to downward deflection of the Respitrace signal. All responses were analyzed twice. Only those moments of deflection consistently identified on both analyses were included in the final data set for temporal analyses. Note that, for this response, the interval between the phonate cue and line B also corresponds to the onset latency of vocal fold closure after the foreperiod as represented by a decrease in the transillumination signal to baseline.

The complete set of laryngeal and respiratory events observed between presentation of the warning cue and phonation onset is summarized in Table 2. Events associated with respiratory inflation during the foreperiod are vocal fold opening and expansion of the thoracic and/or abdominal cavities. Events associated with phonation onset after the foreperiod are vocal fold closure and compression of the thoracic and/or abdominal cavities. The differential deficit hypothesis suggests that there are systematic differences in the manner in which nonstutterers and mild and severe stutterers execute these events.

TABLE 2. Respiratory and laryngeal events observed during and after the foreperiod.

Events observed during foreperiod
Vocal fold opening
Vocal fold closing
Vocal fold closure
Thoracic expansion
Abdominal expansion
Events observed after foreperiod
Vocal fold closing
Vocal fold closure
Thoracic compression
Abdominal compression

To test the differential deficit hypothesis, we asked the questions that follow. In addressing these questions, we used nonparametric statistics because several of the assumptions required for parametric analyses were not met by the data (Siegel, 1956). Our analyses of respiratory and laryngeal kinematic data addressed three questions:

1. Are there significant between-group differences in the frequency of initiation of respiratory and laryngeal events executed in attaining functional physiological targets during and after the foreperiod?
2. Are there significant between-group differences in absolute onset latencies of respiratory and laryngeal events executed in attaining functional physiological targets during and after the foreperiod?
3. Do the groups differ with respect to relative onset latencies of respiratory and laryngeal events executed in attaining functional physiological targets during and after the foreperiod?

Questions 1 and 2 were examined through chi-square comparisons of frequency distributions of the initiation and absolute-onset-latency of relevant respiratory and laryngeal events during and after the foreperiod. Question 3 was examined through visual comparisons of the loci of peaks in absolute-onset-latency frequency distributions of relevant respiratory and laryngeal events during and after the foreperiod.

RESULTS

Acoustic LRT Values

Figure 4 displays mean acoustic LRT values and dispersions representing ± 1 standard deviation as a function of foreperiod duration. Mean LRT values at each

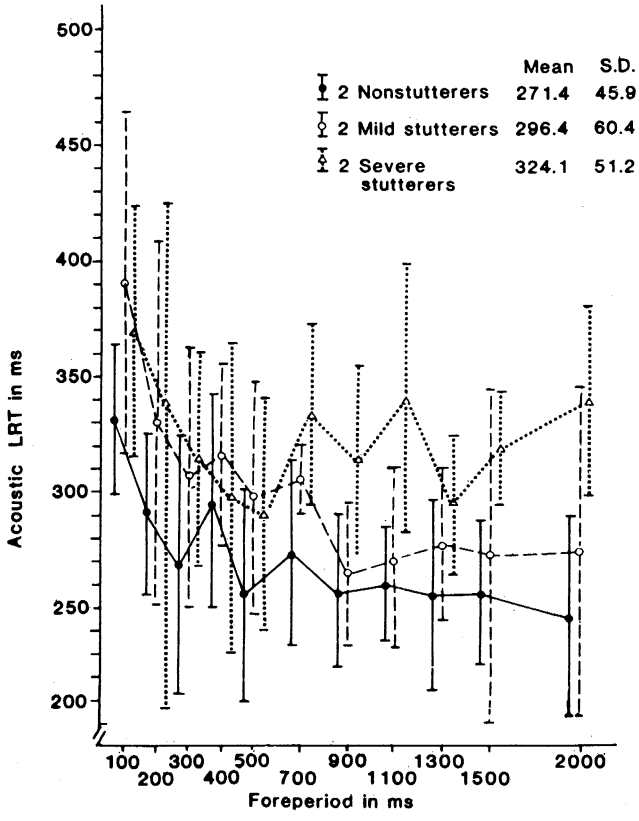

FIGURE 4. Acoustic LRT values as a function of foreperiod for 2 nonstutterers, 2 mild stutterers, and 2 severe stutterers. Vertical bars represent ± 1 standard deviation. Also shown are group means and standard deviations for acoustic LRT values collapsed across the 11 foreperiod conditions.

foreperiod represent data pooled across all fluent responses for both subjects in each group. Grouping of subjects was determined a priori by analyses of stuttering severity. In our discussion of the results of this study, we address the appropriateness of the grouping criteria. Standard deviations, rather than ranges, are reported to represent the dispersion of acoustic LRT values within each group for several reasons. First, the standard deviation explicitly takes into account values of all cases in the distribution (Downey, 1975), and use of the standard deviation statistic minimizes the effects of outlying cases on the distribution. Second, this statistic is standardized by the number of cases. Consequently, it is valid for comparing distributions of different sizes. Group mean acoustic LRT values and standard deviations collapsed across the 11 foreperiod conditions are also listed. These values were computed for the 163 valid (e.g., perceptually and acoustically fluent) responses recorded from the nonstutterers, 103 from the mild stutterers, and 129 from the severe stutterers.

Several features of these data are of interest and highlight their appropriateness for a test of the differential deficit hypothesis. First, the collapsed mean LRT value for the two nonstutterers in this study is nearly identical to the same value reported for five nonstutterers in a previous study (Watson & Alfonso, 1983). Thus, data for the nonstutterers in the present study appear consistent with, and representative of, LRT values recorded from a larger group of normal speakers. Second, acoustic data clearly demonstrate that LRT values vary as a function of stuttering severity and foreperiod. More important, these data replicate our second experiment (Watson & Alfonso, 1983) in which we reported a composite stuttering severity and foreperiod effect on acoustic LRT values for 5 mild and 5 severe stutterers. In both experiments, mild stutterers' LRT values tended to approach those of nonstutterers as foreperiod increased, while severe stutterers' LRT values remained longer than nonstutterers' at both short and long foreperiods. Thus, these data are appropriate for a test of the hypothesis that differential deficits may underlie acoustic LRT values recorded from these mild and severe stutterers.

Examination of Figure 4 reveals that the mild stutterers' LRT values were similar to the severe stutterers' values at foreperiods up to and including 700 ms, and they become similar to the nonstutterers' values at foreperiods beyond 700 ms. Consequently, in the following analyses of kinematic data, we divided responses into two categories. The first includes responses obtained at short foreperiods (e.g., from 100 ms to 700 ms), during which the mild and severe stutterers displayed similar acoustic LRT values. The second includes responses obtained at long foreperiods (e.g., 900 ms to 2000 ms), during which the mild stutterers and nonstutterers displayed similar acoustic LRT values. This division facilitates detailed analyses of between-group differences in the frequency of initiation, timing, and organization of laryngeal and respiratory events associated with between-group differences in acoustic LRT at short and at long foreperiods.

Frequency of Initiation of Prephonatory Events

Events during the foreperiod. Events occurring during the foreperiod were analyzed in terms of the presumed functional physiological target of respiratory inflation. Table 3 summarizes frequency values for initiation of vocal fold opening and of thoracic and abdominal expansion during short and long foreperiods. Frequency values are expressed as both the number of responses and the percentage of total responses during which a particular event was initiated. The last column shows chi-square (χ^2) values (Seigel, 1956) obtained from three-way group comparisons. Also shown is the total number of valid responses for each group at short and at long foreperiods.

Subjects were not instructed to assume a specific respiratory/laryngeal configuration before presentation of the warning cue. Furthermore, the 8-s to 10-s ISIs used in this study likely encouraged subjects to breathe normally between responses. Consequently, the relative degree of vocal fold opening and magnitude of lung volume at presentation of the warning cue could vary randomly for all subjects. That is, for certain responses, vocal fold opening and respiratory expansion sufficient to achieve the target of respiratory inflation may have occurred

TABLE 3. Summary of the frequency of initiation of respiratory and laryngeal events executed during short and long foreperiods. Frequency values are given as the number of responses as well as the percentage of valid responses during which an event was initiated.

Short foreperiods	*Nonstutterers*		*Mild stutterers*		*Severe stutterers*		χ^2
Vocal fold opening	25	28%	31	58%	22	30%	15.0[a]
Thoracic expansion	27	30%	16	30%	9	12%	8.4[b]
Abdominal expansion	19	21%	20	37%	7	9%	14.4[a]
Total Responses	89		53		73		
Long foreperiods							
Vocal fold opening	15	20%	37	74%	26	46%	35.3[c]
Thoracic expansion	43	58%	35	70%	11	19%	24.8[c]
Abdominal expansion	40	54%	30	60%	10	17%	23.6[c]
Total Responses	74		50		56		

[a] χ^2 (2, N = 215) = 9.21, $p < .99$
[b] χ^2 (2, N = 215) = 5.99, $p < .95$
[c] χ^2 (2, N = 180) = 9.21, $p < .99$

before presentation of the warning cue. In light of these factors acting on all groups, the most interesting result in these data was the significant relationship between subject group and frequencies of initiation of thoracic and abdominal expansion.

Subsequent pair-wise comparisons of frequency values for the initiation of abdominal and thoracic expansion revealed details of this relationship. First, the nonstutterers' and mild stutterers' frequency values for initiation of both thoracic and abdominal expansion were not significantly different during short or long foreperiods. Second, the nonstutterers' and severe stutterers' frequency of initiation values for thoracic expansion were significantly different during short [χ^2 (1, n = 162) = 6.64, $p < .01$] and long [χ^2 (1, n = 130) = 6.64, $p<.01$] foreperiods. Third, the nonstutterers' and severe stutterers' frequency of initiation values for abdominal expansion were also significantly different during short [χ^2 (1, n = 162 = 3.84, $p < .05$] and long foreperiods [χ^2 (1, n = 130) = 6.64, $p < .01$]. Finally, all comparisons between the mild and severe stutterers' frequency values revealed significant differences. (For comparison of frequency values for thoracic expansion, χ^2 (1, n = 126) = 3.84, $p < .05$ during short foreperiods, and χ^2 (1, n = 106) = 6.64, $p < .01$ during long foreperiods. For comparison of frequency values for abdominal expansion, χ^2 (1, n = 126) = 6.64, $p < .01$ during short foreperiods, and χ^2 (1, n = 106) = 6.64, $p < .01$ during long foreperiods.)

Table 3 also demonstrates that the nonstutterers and mild stutterers initiated inflation gestures more frequently during long foreperiods than during short foreperiods. Within-group differences for thoracic and abdominal expansion were significant for both groups. (For the nonstutterers: abdominal expansion, χ^2 (1, n = 163) = 3.84, $p < .05$; thoracic expansion, χ^2 (1, n = 163) = 6.64, $p < .01$. For the mild stutterers: abdominal expansion, χ^2 (1, n = 103) = 3.84, $p < .05$; thoracic expansion, χ^2 (1, n = 103) = 6.64, $p < .01$. Conversely, the severe stutterers' frequency of initiation of these movements was not significantly different between short and long foreperiods.

Events after the foreperiod. Events occurring after the foreperiod were analyzed in terms of the presumed functional target of respiratory inflation. Table 4 summarizes

TABLE 4. Summary of the frequency of initiation of respiratory and laryngeal events executed after short and long foreperiods. Frequency values are given as the number of responses as well as the percentage of valid responses during which an event was initiated.

Short foreperiods	*Nonstutterers*		*Mild stutterers*		*Severe stutterers*		χ^2
Vocal fold closure	88	98%	50	94%	67	91%	4.7
Thoracic compression	65	73%	31	58%	7	9%	67.8*
Abdominal compression	83	93%	49	92%	72	98%	3.2
Total Responses	89		53		73		
Long foreperiods							
Vocal fold closure	70	94%	25	50%	51	91%	43.9*
Thoracic compression	35	47%	36	72%	2	3%	49.1*
Abdominal compression	64	86%	46	92%	55	98%	5.7
Total Responses	74		50		56		

* χ^2 (2, N = 215) = 9.21, $p < .99$

frequency values for the initiation of vocal fold closure and of thoracic and abdominal compression after short and long foreperiods. The table also shows χ^2 values obtained from three-way group comparisons.

After short foreperiods, the only significant difference was obtained for comparison of frequency values for the initiation of thoracic compression. Subsequent pair-wise comparisons for thoracic compression revealed significant differences between the nonstutterers and the severe stutterers [χ^2 (1, n = 162) = 6.64, $p < .01$] and between the mild and severe stutterers [χ^2 (1, n = 126) = 6.64, $p < .01$]. The difference between the nonstutterers and mild stutterers was not significant. Thus, after short foreperiods, the nonstutterers and mild stutterers executed thoracic compression more frequently than did the severe stutterers.

After long foreperiods, significant differences were obtained for three-way comparisons of frequency values for the initiation of vocal fold closure and of thoracic compression. Subsequent pair-wise comparisons for vocal fold closure revealed significant differences between the mild stutterers and nonstutterers, [χ^2 (1, n = 142) = 6.64, $p < .01$] and between the mild and severe stutterers [χ^2 (1, n = 126) = 6.64, $p < .01$]. Further analysis revealed that significant between-group differences could be attributed to one of the mild stutterers who frequently executed vocal fold closure during long foreperiods. Pair-wise comparisons of frequency values for the initiation of thoracic compression revealed significant differences between the nonstutterers and mild stutterers [χ^2 (1, n = 142) = 6.64, $p < .01$], between the nonstutterers and the severe stutterers [χ^2 (1, n = 162) = 6.64, $p < .01$], and between the mild and severe stutterers [χ^2 (1, n = 126) = 6.64, $p < .01$]. Further analysis revealed that the severe stutterers, unlike the nonstutterers and mild stutterers, frequently initiated thoracic compression after phonation onset.

To summarize, the three most interesting results of analyses of the frequency of initiation of laryngeal and respiratory events during and after short versus long foreperiods revealed that groups were differentiated on the basis of respiratory movements. First, the nonstutterers and mild stutterers tended to initiate thoracic and abdominal expansion during the foreperiod more frequently than did the severe stutterers. Second, only the nonstutterers and the mild stutterers initiated thoracic and abdominal expansion more frequently as the foreperiod increased. Finally, the nonstutterers and mild stutterers generally compressed both the thoracic and abdominal cavities before phonation onset. The severe stutterers, on the other hand, more often compressed the abdominal cavity before phonation onset and compressed the thoracic cavity after phonation onset.

Timing and Organization of Laryneal and Respiratory Events

The timing of prephonatory laryngeal and respiratory events was evaluated in terms of absolute onset latencies relative to presentation of warning or phonate cues. Because variability is an important measure of performance in a reaction time task, we based our analyses of timing on comparisons of absolute-onset-latency frequency distributions. Latency values were categorized into 100-ms intervals following the warning cue and into 20-ms intervals following the phonate cue. Differences in interval durations reflect the fact that events following the phonate cue occurred during a shorter time period than did events following the warning cue. That is, average LRT values for all groups were shorter than the average foreperiod duration. Categorization procedures resulted in a frequency distribution of onset latency values for each event. The frequency value in each temporal interval was then placed in one cell of a contingency table for subsequent between and within group χ^2 analyses.

Events during all foreperiods. Nonsignificant χ^2 values were obtained for within-group comparisons of the timing of events occurring during short versus long foreperiods for all groups. Consequently, onset-latency data were collapsed across all foreperiods for between-group comparisons. Figure 5 displays absolute-onset-latency-value frequency distributions for vocal fold opening and for thoracic and abdominal expansion for each group during all foreperiods. The only significant finding in comparisons of these distributions was the difference between the mild and severe stutterers for vocal fold opening [χ^2 (15, n = 232) = 30.58, $p < .01$]. The mild stutterers demonstrated onset of vocal fold opening early during the foreperiod more often than did severe stutterers. Although the difference between nonstutterers and severe stutterers was not significant, visual inspection of the figures suggests that the nonstutterers tended to initiate vocal fold opening earlier in the foreperiod than did severe stutterers.

Comparisons of the loci of peaks in the three frequency distributions for each group reveal the organization of events leading to respiratory inflation. The nonstutterers demonstrated a peak in the distribution for vocal fold opening 100 ms before the peaks of thoracic and abdominal expansion. The mild stutterers demonstrated peaks in all three distributions during the 100- to 199-ms temporal interval. Nonstutterers' and mild stutterers' organization of these events was appropriate for attaining respiratory inflation. Conversely, the severe stutterers demonstrated peaks in the onset-latency distributions for thoracic and abdominal expansion 100 ms before the peak for vocal fold opening. Only the severe stutterers began respiratory expansion movements before onset of a vocal fold opening gesture.

Events after short versus long foreperiods. Figures 6 and 7 display absolute-onset-latency-value frequency distributions for vocal fold closure and for abdominal compression for each group after short and long foreperiods, respectively. Because the severe stutterers rarely executed thoracic compression before phonation onset (see Table 4), frequency distributions for thoracic compression were omitted from these figures.

Correlation analyses were conducted to determine the relationship between onset latencies of vocal fold closure

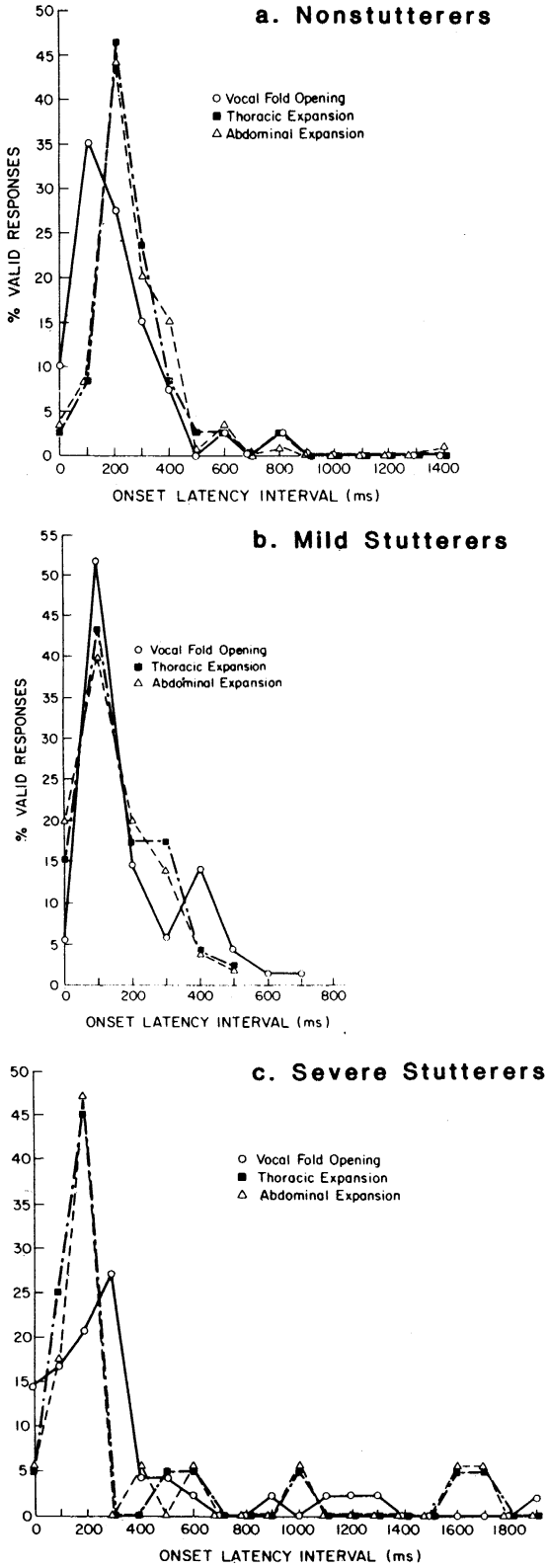

FIGURE 5. Frequency distributions of absolute-onset-latency values for events occurring during all foreperiods as a function of 100-ms intervals following presentation of the warning cue. Frequency values represent the percentage of valid responses in which an event began during a specific temporal interval.

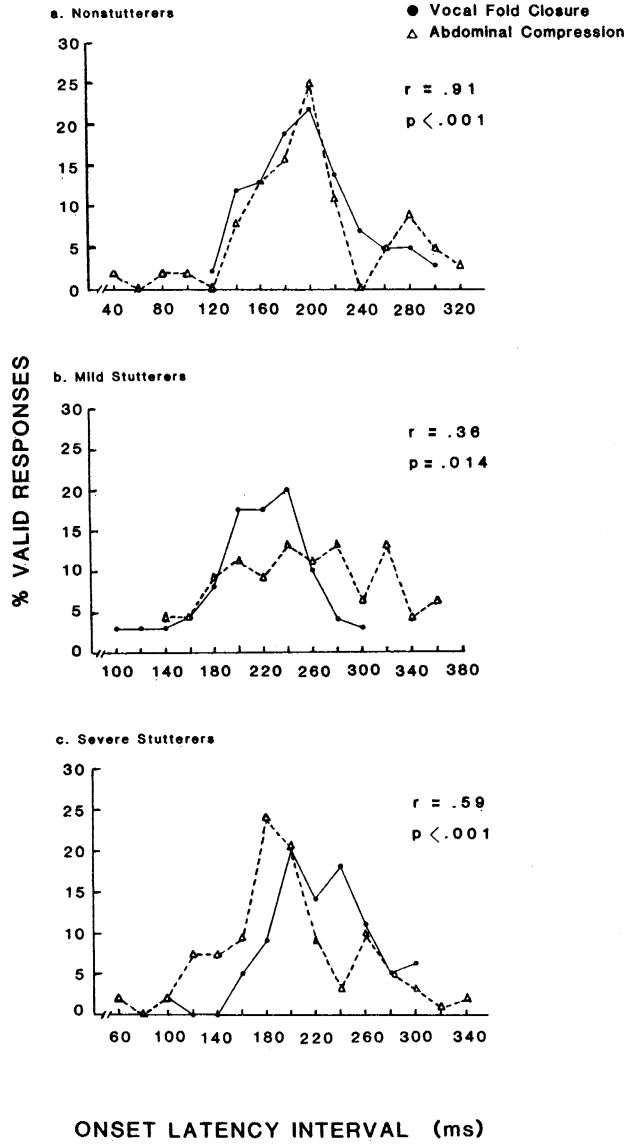

FIGURE 6. Frequency distributions of absolute-onset-latency values for events occurring after short foreperiods as a function of 20-ms intervals following presentation of the phonate cue. Frequency values represent the percentage of valid responses in which an event began during a specific temporal interval.

and abdominal compression for each group at short and long foreperiods. Significant *r* values would indicate that short vocal fold closure latencies are associated with short abdominal compression latencies. Figures 6 and 7 contain *r* values for all comparisons. Although varying in strength, all *r* values were significant at probability levels ranging from .028 to less than .001. Significant *r* values indicated that laryngeal and respiratory onset-latency frequency distributions were temporally interdependent and could be used to examine the organization of prephonatory events in terms of relative onset latency values.

Within-group comparisons of frequency distributions shown in Figures 6 and 7 revealed several findings of

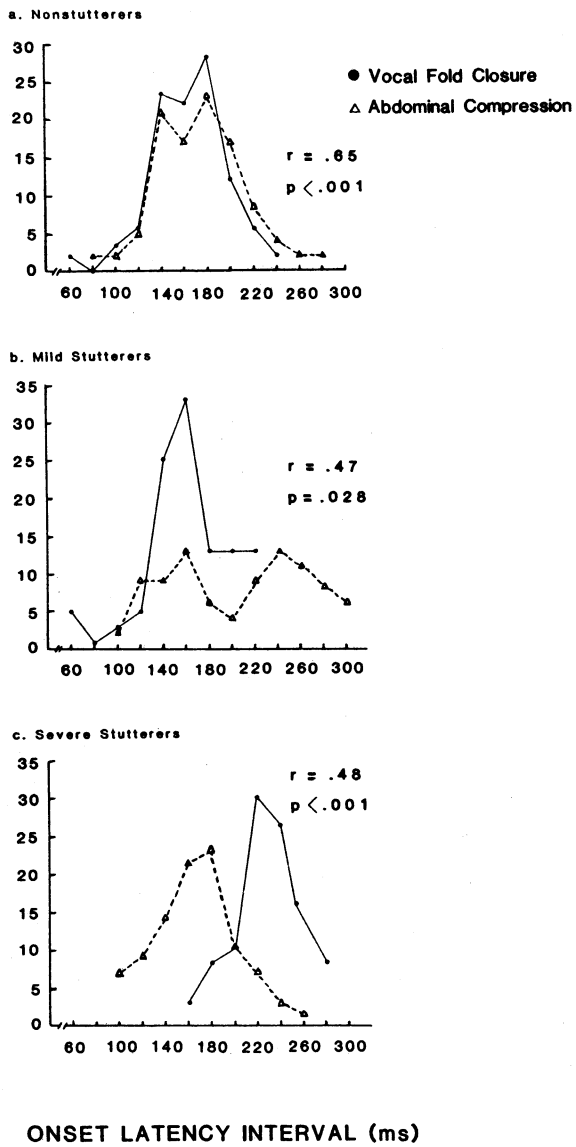

FIGURE 7. Frequency distributions of absolute-onset-latency values for events occurring after long foreperiods as a function of 20-ms intervals following presentation of the phonate cue. Frequency values represent the percentage of valid responses in which an event began during a specific temporal interval.

interest. First, the nonstutterers demonstrated shorter onset latency values for vocal fold closure after long foreperiods than after short foreperiods [χ^2 (12, n = 163) = 26.22, $p < .01$]. Mild stutterers also demonstrated this difference [χ^2 (15, n = 103) = 30.58, $p < .01$]. Second, although comparison of the mild stutterers' latency values for abdominal compression after short versus long foreperiods was nonsignificant, this event occurred earlier after long foreperiods than after short foreperiods. Finally, the severe stutterers demonstrated no improvement in onset latency values for laryngeal or respiratory events as a consequence of foreperiod duration.

Three-way group comparisons of frequency distributions in Figure 6 revealed significant differences in the onset latency of vocal fold closure [χ^2 (28, n = 215) = 41.34, $p < .05$] and of abdominal compression [χ^2 (28, n = 215) = 39.9, $p < .07$]. Pair-wise comparisons of onset-latency distributions for vocal fold closure revealed significant differences between the nonstutterers and mild stutterers [χ^2 (15, n = 142) = 25.00, $p < .05$] and between the nonstutterers and severe stutterers [χ^2 (16, n = 162) = 32.0, $p < .01$]. The difference between mild and severe stutterers was not significant. Pair-wise comparisons of onset-latency distributions for abdominal compression revealed significant differences between the nonstutterers and mild stutterers [χ^2 (15, n = 142) = 30.58, $p < .01$] and between the mild and severe stutterers [χ^2 (16, n = 126) = 32.0, $p < .01$]. The difference between nonstutterers and severe stutterers was not significant. This nonsignificant temporal difference is related to the organizational difference described below.

Comparisons of the loci of peaks in the frequency distributions for each group in Figure 6 revealed that both the nonstutterers and mild stutterers generally attained vocal fold closure simultaneously with, or immediately before, onset of abdominal compression. This organization of laryngeal and respiratory events is appropriate for the goal of a soft mode of vocal attack. Conversely, the severe stutterers generally executed a poorly organized sequence of events characterized by onset of abdominal compression before attaining vocal fold closure. Summarizing, after short foreperiods, the mild stutterers demonstrated delayed onset of well organized laryngeal and respiratory events, while the severe stutterers demonstrated delayed onset of vocal fold closure and a poorly organized sequence of events.

Three-way group comparisons of frequency distributions in Figure 7 also revealed significant differences in the onset latency of vocal fold closure [χ^2 (22, n = 180) = 40.29, $p < .01$] and of abdominal compression [χ^2 (22, n = 180) = 40.29, $p < .01$]. Pair-wise comparisons of onset-latency distributions for vocal fold closure revealed significant differences between the nonstutterers and severe stutterers [χ^2 (11, n = 130 = 24.72, $p < .01$] and between the mild and severe stutterers [χ^2 (8, n = 106) = 20.09, $p < .01$]. Pair-wise comparisons of onset-latency distributions for abdominal compression revealed significant differences between the nonstutterers and mild stutterers [χ^2 (12, n = 124) = 26.22, $p < .01$] and between the mild and severe stutterers [χ^2 (11, n = 106) = 19.68, $p < .05$]. The nonsignificant difference between the nonstutterers and severe stutterers apparently reflects severe stutterers' execution of abdominal compression before the moment of vocal fold closure.

Comparisons of the loci of peaks in the frequency distributions for each group in Figure 7 revealed that the nonstutterers and mild stutterers, once again, demonstrated appropriate organization of laryngeal and respiratory events for the soft mode of attack. With respect to the mild stutterers, the bimodal distribution for abdominal compression reflects the fact that one mild stutterer initiated this gesture simultaneously with the amount of vocal fold closure, while the other mild stutterer initiated abdominal compression after vocal fold closure. The

severe stutterers continued to demonstrate poor organization. To summarize, after long foreperiods, the mild stutterers demonstrated delayed onset of only abdominal compression and well-organized events, while the severe stutterers continued to demonstrate delayed onset of vocal fold closure and poorly organized events.

Two final comparisons of onset latency differences after the foreperiod are of interest. First, while the nonstutterers and mild stutterers differed in onset latencies of vocal fold closure and abdominal compression after short foreperiods, these groups differed only in the onset latency of abdominal compression after long foreperiods. Second, the nonstutterers and severe stutterers differed in the onset latency of vocal fold closure after short and long foreperiods. Thus, as foreperiod increased, mild, but not severe, stutterers' onset latency of vocal fold closure became similar to that of nonstutterers.

DISCUSSION

Due to the relatively invasive nature of fiberoptic endoscopy, it is difficult to perform this procedure on a large number of naive subjects. However, we successfully recorded Respitrace and transillumination signals from 9 stutterers. Data from 5 of these subjects were not reported in this study because they did not meet fluency criteria, did not represent the soft mode of vocal attack, or did not unambiguously distinguish the subject as either a mild or severe stutterer. Recall that acoustic LRT values for the 4 stutterers reported here are nearly identical to the values for 10 stutterers reported in a previous study (Watson & Alfonso, 1983). Therefore, we assume that the 2 mild and 2 severe stutterers described in this experiment are representative of larger groups of stutterers with similar severity ratings. As is often the case in physiologically based studies, we are forced to draw what conclusions we can from a relatively small sample of the population.

Using the magnitude of acoustic LRT values as an indicator of subjects' success in executing prephonatory respiratory and laryngeal events, we present a two-part discussion of the kinematic data reported in this study. Part one examines kinematic properties of events associated with the relatively stable boundary conditions of acoustic LRT values illustrated in Figure 4. The lower (i.e., rapid onset) boundary is represented by the nonstutterers' acoustic LRT response function while the upper (i.e., prolonged onset) boundary is represented by the severe stutterers' acoustic LRT response function. Part two examines kinematic properties of events associated with the mild stutterers' improving acoustic LRT response function from short to long foreperiods. Examination of kinematic properties of prephonatory respiratory and laryngeal events associated with fast and slow acoustic LRT values can provide insights into both the coordination of these events in stutterers and nonstutterers and the nature of differential deficits in the mild and severe stutterers.

Recall that our analysis of kinematic properties of prephonatory laryngeal and respiratory movements was based on a division of the range of foreperiods into two regions. Specifically, we identified short foreperiods (from 100 ms to 700 ms), during which mild and severe stutterers' acoustic LRT values are similar, and long foreperiods (from 900 ms to 2000 ms), during which mild stutterers' and nonstutterers' acoustic LRT values are similar. However, note that LRT values for all groups improve across the 100-ms through 300-ms foreperiods. Improvement in LRT values for all groups during these foreperiods may be a function of increased time available to overcome mechano-inertial constraints associated with laryngeal and respiratory movements. For example, peaks in the nonstutterers' onset-latency frequency distributions for abdominal and thoracic expansion occurred during the interval from 240 ms to 260 ms. This finding is consistent with the 244-ms average latency for respiratory movements in nonstutterers reported by Baken et al. (1979).

Lower Boundary of LRT Values

Acoustic LRT values for the nonstutterers generally improve during the first three foreperiods (e.g., 100 ms through 300 ms) and then plateau. As noted above, initial improvement in the nonstutterers' LRT values during very short foreperiods is likely related to inherent response times of the respiratory and laryngeal systems. Thus, the presumed functional target of respiratory inflation is seldom achieved during these foreperiods. A likely consequence of failure to inflate the respiratory system adequately during the foreperiod is delayed onset of events after the foreperiod. For example, onset latency of the nonstutterers' respiratory and laryngeal events after the foreperiod was faster when events associated with attaining the functional target of respiratory inflation were initiated during the foreperiod. To summarize, improvement in the nonstutterers' LRT values seems to reflect the increased time available to overcome possible mechano-inertial constraints on the onset latency of laryngeal and respiratory movements. Once foreperiods were sufficiently long to permit respiratory inflation, nonstutterers frequently demonstrated rapid onset of well-organized respiratory and laryngeal events leading to the functional targets of respiratory inflation and phonation onset.

Upper Boundary of LRT Values

The severe stutterers' acoustic LRT values also improve during the first three foreperiods and then plateau. However, their LRT values remain significantly longer than the nonstutterers' values throughout the range of short to long foreperiods. Analyses of kinematic data reveal three factors that can account for the severe stutterers' consistently long acoustic LRT values at all foreperiods. First, the severe stutterers showed delayed

onset of vocal fold opening and infrequent initiation of respiratory expansion during short and long foreperiods. Consequently, it is likely that the functional target of respiratory inflation was rarely attained. Second, the severe stutterers consistently showed delayed onset of laryngeal events after the foreperiod. Third, the severe stutterers frequently demonstrated poorly organized respiratory and laryngeal events at both short and long foreperiods.

Summarizing the first part of this discussion, fast LRT values demonstrated by the nonstutterers reflect frequent and rapid execution of well-organized respiratory and laryngeal events. Conversely, slow LRT values demonstrated by the severe stutterers reflect infrequent and delayed initiation of poorly organized respiratory and laryngeal events. We now turn to an examination of kinematic properties of events underlying improvement in mild stutterers' acoustic LRT values from short to long foreperiods.

Mild Stutterers' LRT Values

The mild stutterers' acoustic LRT values can be classified into two intervals. First, they are similar to the severe stutterers' values at foreperiods up to 700 ms. Second, the mild stutterers' LRT values are similar to nonstutterers' values at foreperiods beyond 700 ms. As with the nonstutterers and severe stutterers, improvement in the mild stutterers' LRT values across very short foreperiods (up to 300 ms) is assumed to be a function of increased time available to overcome mechano-inertial constraints on laryngeal and respiratory movements. Improvement in the mild stutterers' LRT values from short to long foreperiods appears to be related to four factors. First, mild stutterers initiated vocal fold opening as well as thoracic and abdominal expansion more frequently during long foreperiods than during short foreperiods. Second, these subjects achieved vocal fold closure more rapidly after long foreperiods than after short foreperiods. Third, although consistently slower than the nonstutterers, mild stutterers tended to begin respiratory compression more rapidly after long foreperiods than after short foreperiods. Finally, the mild stutterers consistently demonstrated well-organized prephonatory events during and after both short and long foreperiods. Thus, the mild stutterers' delayed acoustic LRT values at short foreperiods likely reflect (a) infrequent initiation of thoracic and abdominal cavity expansion during the foreperiod and (b) delays in both achieving vocal fold closure and initiating abdominal compression after the foreperiod. Conversely, the mild stutterers' improved acoustic LRT values at long foreperiods likely reflect (a) frequent initiation of thoracic and abdominal cavity expansion during the foreperiod and (b) more rapid vocal fold closure and shorter delays in initiating abdominal compression after the foreperiod. Thus, because the mild stutterers always executed well-organized events during and after all foreperiods, improvement in their acoustic LRT values as a function of increasing foreperiod can be related to improvements in the frequency of initiation and timing of prephonatory events.

CONCLUSIONS

Kinematic data reported in this study support the differential deficit hypothesis for a subject classification scheme based on stuttering severity. While severity is used frequently to identify subgroups within the stuttering population, it is possible that characteristics other than severity could account for observed group differences in prephonatory physiological events. For example, stutterers may be classified as a function of anatomical locus of stuttering (Shapiro, 1980) or prominent type of dysfluent behavior (e.g., repetitions versus prolongations and tonic blocks [Stromsta, 1984]). Implicit in the former classification scheme is the assumption that a "laryngeal stutterer" would generate different prephonatory physiological events in a reaction time task than would a "lingual stutterer." Implicit in the latter scheme are the assumptions that relative frequencies of repetitions and prolongations vary across stutterers and that physiological bases for repetitions and prolongations are distinct. Responses analyzed in this study were judged to be fluent. Therefore, to account for between-group differences in prephonatory physiological events observed in this study, it would be necessary to assume further that distinguishing physiological characteristics representative of "locus" or "type" are present during "fluent" speech production. Given that our subjects did not meet grouping criteria other than severity, the possibility that between-group differences can be attributed to "locus" or "type" of dysfluency appears rather remote.

Observation of physiological deficits in both the respiratory and laryngeal systems suggests that abnormal laryngeal function is not the sole, nor perhaps the primary, factor associated with the stuttering disorder (cf. Adams, 1974; Peters & Boves, 1984). In contrast to this view, Baken et al. (1983), using a somewhat different experimental task, concluded that expenditure of a significantly greater prephonatory lung volume by stutterers than by nonstutterers during the production of isolated vowels reflected stutterers' delays in attaining vocal fold closure. That is, based on their analysis of respiratory data alone, they suggested that stutterers demonstrate laryngeal system dysfunction while the respiratory system functions like that of nonstutterers. Our observations of simultaneous respiratory and laryngeal kinematics, however, indicate that, at least for the severe stutterers in this study, deficits exist in both systems.

With the exception of insights gained from recent papers such as those by Shipp, Izdebski, and Morrissey (1984), who analyzed laryngeal electromyographic and subglottal pressure data; Peters and Boves (1984), who analyzed electroglottographic and subglottal pressure data; and Baken et al. (1983), who analyzed respiratory movements, less is known about neuromuscular and kinematic properties of rapid responses in a reaction-time task than about the acoustic response. Thus, data reported

in this study are significant in at least three respects. First, the data represent simultaneous measures of prephonatory respiratory and laryngeal events in stutterers as well as nonstutterers and add to our understanding of physiological bases of stutterers' delayed phonation onset. Second, these data suggest that physiological events leading to phonation appear to differ as a function of stuttering severity. Thus, mild and severe stutterers attain, or fail to attain, functional prephonatory targets in qualitatively and quantitatively different ways. Third, these data imply that, at least for severe stutterers, physiological deficits underlying slower acoustic LRT values may occur in both the respiratory and laryngeal systems. Taken together, these data support multiple anatomical loci models of stuttering and suggest that these models account for physiological differences that characterize mild and severe stutterers.

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