The Distribution of Phonated Intervals in the Speech of Individuals Who Stutter

Tara Godinho
Roger J. Ingham
University of California, Santa Barbara

Jason Davidow
University of Georgia, Athens

John Cotton
University of California, Santa Barbara

Purpose: Previous research has demonstrated the fluency-improving effect of reducing the occurrence of short-duration, phonated intervals (PIs; ~30–150 ms) in individuals who stutter, prompting the hypothesis that PIs in these individuals' speech are not distributed normally, particularly in the short PI ranges. It has also been hypothesized that this nonnormal PI distribution will be present during the stutter-free speech of affected persons.
Method: A comparison was made between the distributions of PIs during oral reading by adolescent and adult speakers who stuttered (*n* = 13; 11 males) and by age- and gender-matched, normally fluent control participants.
Results: The results did not support these hypotheses. The results showed that although there were significantly fewer PIs in the speech of the speakers who stuttered (probably because of their slower speaking rate), there was no significant difference between the PI distributions of both speaker groups. This was also true for comparisons between the stutter-free speech of the affected speakers and matched periods of speech produced by the control participants. The PI distributions from both groups were highly correlated.
Conclusion: The null hypothesis findings are discussed in relation to speech–motor- and neurologic-systems explanations for the fluency-inducing effects of reducing short PIs in the speech of individuals who stutter.

KEY WORDS: stuttering, phonation, stutter-free speech, adults

In a series of studies Ingham and colleagues (Gow & Ingham, 1992; Ingham et al., 2001; Ingham, Montgomery, & Ulliana, 1983) investigated the effect on stuttering of manipulating the frequency of occurrence of short phonated intervals (PIs; i.e., short voiced sounds produced by vocal fold vibration) during different speaking tasks. In general, the findings of those studies demonstrated that training individuals who stutter to speak with an approximately 50% reduction of 30–150-ms PIs (per minute of speaking time) results in substantial reductions in the frequency of stuttering. Furthermore, the resulting improvements in fluency do not necessarily involve concomitant increases in the frequency of longer duration PIs or a deterioration in speech naturalness (Gow & Ingham, 1992; Ingham et al., 2001). PIs in the 30–150-ms range represented approximately the lower 20th percentile of the distribution of PIs in the speech of affected speakers who participated in these studies.

Much of the research on PI modification has been prompted by efforts to identify the functional constituents of prolonged speech (Goldiamond, 1965; Ingham, 1984, Chapter 10), arguably the centerpiece of most current

1092-4388/06/4901-0161

treatments for developmental stuttering in adolescents and adults (see Bothe et al., 2003). Less emphasis has been placed on investigating the extent to which stuttering is characterized by an abnormal distribution of PIs or whether training to modify specific PI frequencies results in speakers' achieving a normalized PI distribution.[1] In this respect, PI variability is especially interesting because it is a speech–motor parameter that appears to exert functional control over stuttering. There are numerous speech–motor parameters within which differences between speakers who stutter and those who do not have been demonstrated (Bloodstein, 1995). However, relatively few of those differences (e.g., voice reaction time, short pauses) have been shown to be modifiable and exert direct functional control over stuttering (see Ingham, 1998); such a combination has clinical implications that have yet to emerge from investigations of differences in other speech–motor parameters. At the same time, researchers recognize that variations in the PI parameter likely involve concomitant variations in other speech–motor parameters.

Evidence that altering or modifying phonation will reduce stuttering has been derived mainly from studies conducted on the various so-called fluency-inducing procedures. For example, in a number of studies, researchers have shown that masking (Andrews, Howie, Dozsa, & Guitar, 1982), chorus reading (Andrews et al., 1982), singing (Colcord & Adams, 1979), rhythmic speech (Brayton & Conture, 1978) and, of course, prolonged speech (Goldiamond, 1965, 1967; Packman, Onslow, & van Doorn, 1994) may involve alterations in the proportions of phonated speech. The pattern of changed phonation is not consistent across these fluency-inducing procedures (see Ingham, 1990), but when this evidence of phonation change is related to the consistent finding that stuttering speakers are relatively slow to initiate and terminate phonation (see Bloodstein, 1995), it strongly suggests that phonation management or control is functionally related to stuttering.[2]

The apparent interaction between phonatory behavior and stuttering may also be related to evidence that adult speakers who stutter tend to display relatively longer intervals of phonation during stutter-free speech (Pindzola, 1987; Prosek & Runyan, 1982), a characteristic that is less evident in children who stutter (Healey & Adams, 1981; Zebrowski, Conture, & Cudahy, 1985). Onslow and Ingham (1987) speculated that this age effect may occur because older speakers have learned to manage their disorder, however imperfectly, by using slower voice initiation and longer intervals of phonation. Note, however, that research on phonation during affected speakers' stutter-free speech has been focused on extended rather than shortened intervals of phonation. Nonetheless, these findings highlight the importance of determining the extent to which PI production during the stuttered and stutter-free speech of affected individuals is significantly different than that that occurs during normally fluent speech production.

An equally important reason for learning the relationship between PI production in person who stutter and normally fluent speakers is that improved fluency produced by modifying the frequency of short-duration PIs may be associated with important neural changes in stuttering speakers. Ingham, Ingham, Finn, and Fox (2003) reported the results of an investigation using positron emission tomography (PET) with 17 adult speakers who stuttered (9 men), who had completed part of the Modified Phonation Interval (MPI; Ingham et al., 2001) therapy program, which trains for reduced PI production in the lower 20th-percentile-ms range of each speaker's base-rate PI distribution. The treatment was sufficient to enable the participants to be scanned while producing essentially stutter-free and natural sounding speech during brief monologues. A comparison with normally fluent controls showed that the stuttering speakers displayed normalized cerebral blood flow (CBF) activations in most regions that have been associated with normal speech production (Indefrey & Levelt, 2004; Jürgens, 2002). By contrast, the relatively normal fluency produced by adult speakers who stutter (n = 20; 10 men) during chorus reading was associated with CBF activations in significantly fewer of these same neural regions. This was not a straightforward comparison (monologues were compared with oral reading), but the findings suggest that, compared with chorus reading, the fluency induced during short-duration PI reduction activated neural regions that support normal speech. It is not clear, however, if MPI training resulted in the participants who stuttered achieving a normal pattern of PI production and thereby normalized neural changes, or if the normalized neural changes achieved by the MPI training required a nonnormal distribution of PIs. That information can only be obtained by comparing the distributions of PIs produced by normally fluent speakers and speakers who stutter, which was the purpose of the present study.

For our primary hypothesis in this study, we proposed that the distribution of PIs (within the 30–1,000-ms range) during oral reading by adult and adolescent speakers who stuttered would be significantly different from that found during oral reading by age- and

---

[1] These studies have been with adolescents and adults; it has yet to be established that similar effects occur when PI frequencies are modified in the speech of children who stutter.

[2] It is of interest that among other putative, prolonged speech parameters (e.g., "soft contacts," "gentle onsets," or "easy onsets"), only acoustic rise time—an approximation to gentle onsets—has been investigated during the speech of speakers who stutter (Borden, Baer, & Kenney, 1985; Peters, Boves, & van Dielen, 1986). No study has reported on the effect on stuttering of modifying acoustic rise time.

gender-matched, normally fluent controls. To test whether an abnormal distribution of PIs in speakers who stuttered was a result of the presence of abnormal phonatory gestures produced during occasions of stuttering (Conture et al., 1977, 1985; Shapiro & De Cicco, 1982), we tested a secondary hypothesis that the distribution of 30–1,000-ms PIs during the stutter-free speech of affected speakers (i.e., when recorded intervals of speech containing stuttering were removed) would be significantly different from the distribution of 30–1,000-ms PIs during equivalently adjusted speech samples from normally fluent speakers. Support for this hypothesis would be consistent with evidence of unusual characteristics in the stutter-free speech of affected speakers (Finn & Ingham, 1989; Pindzola, 1987; Prosek & Runyan, 1982).

Method

Participants

Participants were 13 adult and adolescent individuals who stuttered (11 males) and 13 control participants who were matched for age (age range for speakers who stuttered = 14–48 years; age range for control participants = 15–50 years) and gender. Age matching was achieved by using a maximum age difference of 1 year between participants who stuttered and control participants aged 11–20 years, 2 years between participants aged 21–30 years, and so on. All speakers who stuttered reported a history of stuttering since childhood and had participated in a variety of treatment programs; however, none had been judged by the participant as effective. None of the participants were receiving treatment or had received formal treatment for stuttering in the previous 3 years. During oral reading tasks in this study, the affected individuals' mean percentage syllables stuttered (SS) ranged from 0.9% to 10.1% SS (see Table 1). The percentage of stuttered 5-s intervals (SI) during the participants' speech samples ranged from 6.5% to 95.4% (see Table 1). Ten of the participants had volunteered to be enrolled in an investigation of the MPI (Ingham, Moglia, Kilgo, & Felino, 1997) program being conducted at the University of California, Santa Barbara. Data for the additional 3 participants were obtained with permission from an ongoing research project at the University of Georgia. The protocol for participating in this project was approved by the Institutional Review Boards at the University of California, Santa Barbara, and the University of Georgia.

Control participants were volunteers from faculty, staff, and students at the University of California, Santa Barbara, and the University of Georgia. All participants had hearing within normal limits. All were successful professionals or high school or university students and were, therefore, assumed to have normal cognitive abilities.

Table 1. Speech performance data of the 13 speakers who stuttered and age- and gender-matched control participants.

	Speakers who stuttered				Controls
Participant no.	**SPM**	**SFSPM**	**%SS**	**%SI**	**SPM/SFSPM**
1	224.3	228.0	1.25	20.29	262.8
2	169.0	237.0	10.09	95.37	229.9
3	157.3	257.0	10.08	86.11	275.0
4	209.0	225.7	2.21	34.26	221.9
5	199.3	201.7	1.62	23.15	255.0
6	229.7	232.3	0.92	6.48	274.7
7	221.7	231.3	1.45	25.93	240.7
8	184.0	240.0	5.90	74.08	281.2
9	117.3	107.0	6.62	61.11	257.0
10	244.0	272.0	3.33	53.70	246.8
11	204.7	236.0	2.49	42.59	264.9
12	220.7	233.3	1.74	32.41	255.7
13	195.3	251.3	2.73	47.22	232.8
M	198.2	227.1	3.88	46.44	253.7

Note. Data are mean scores obtained from three 3-min oral reading tasks. SPM = syllables/min; SFSPM = stutter-free syllables/min; %SS = % of syllables stuttered; %SI = % of stuttered 5-s intervals.

Setting and Apparatus

During the experiment, participants sat alone in a sound-controlled room where their PIs were recorded using the MPI software program system. As described in Ingham et al. (2001), the MPI program uses customized software and is operated using Windows XP on a desktop personal computer. The MPI program uses two other hardware items: an accelerometer (ACH-01-04) and a customized preamplifier unit for conditioning the accelerometer signal. The accelerometer is a piezo-electronic transducer with no sensitivity relative to earth and a 2-Hz to 20-kHz frequency response. It is housed within a Velcro neckband and is wired to the preamplifier, which is, in turn, connected to the computer. Vocal fold vibration is registered by the accelerometer, which is positioned within the neckband so that its surface is just below the thyroid prominence. The accelerometer signal is routed to a bandpass (80–300 Hz) filter and to the preamplifier unit. The MPI system uses a Sound Blaster Live card (16-bit sampling) with a 10–44-kHz frequency response. The system sets the digitization rate for the sound card at 12 kHz and ignores 11/12 samples, providing an effective sampling rate of 1 kHz. The input signals are integrated and smoothed over a 10-ms window after which only alternate values are retained, thereby producing a 500-Hz sampling rate for the conditioned signal. Before each MPI session, the system noise floor is established. When the intensity of the input signal exceeds 10% above the noise floor, a PI signal is initiated, and it ceases when the signal recedes below the noise floor.

Before the start of each participant's experiment, preliminary steps established that the accelerometer signal during speech met prescribed intensity level criteria and that the system's noise floor when the participant was silent was within prescribed limits (typically between 62.5 and 75 mV). A presession PI recording accuracy test was also made with each participant using an oral counting task. This test requires the MPI system to record a consistent and predetermined number of PIs for the oral counting task. Last, each participant produced a series of voiceless utterances and swallowing and head movements consistent with normal utterances, each for 1 min, to ensure that the settings did not produce a false PI signal. During 3-min oral reading trials, the MPI system recorded all PIs using that same setting.

All participants were audiovisually recorded during each session using a digital video camera (Canon ZR 70) with shotgun microphone; the session recordings were stored in a retrievable digital format. All PI signals were stored in a temporal format that made it possible to retrieve all PIs in the 30–1,000-ms range. PIs ≤30 ms were excluded from analysis because tests conducted during previous studies (Gow & Ingham, 1992; Ingham et al., 2001) showed that PIs in this range are consistently confounded with head movement and swallowing. Only a few PIs from both groups exceeded 1,000 ms in the present study.[3]

Procedure

During the experiment, each participant sat alone in the sound-treated room in front of a table with a computer monitor and keyboard. Participants could choose to read from one of three books (Kagan, 1998; Linnea, 1995; Ridley, 1999) selected for general text homogeneity by being free of conversational content. Each participant completed three 3-min oral reading tasks, each separated by a 1-min data storage period. Participants were prompted to start and stop reading by tones emitted by the MPI computer program. During each reading task, a research assistant who was trained on the Stuttering Measurement System (SMS; Ingham, Bakker, Ingham, Kilgo, & Moglia, 1999) and Stuttering Measurement and Assessment Training (SMAAT; Ingham et al., 1998) programs recorded all syllables, stuttered and nonstuttered, using the computer mouse with the SMS system (SMS is built into the MPI program). The mouse-button–counted syllables were then converted into percentage SS and stutter-free syllables per minute (SFSPM). In addition, the MPI program recorded the number and percentage of 5-SI in which the judge recorded (from a mouse-button press) one or more stuttered events, including their duration.

The following procedure was used for the analysis of the data. For each participant, the millisecond duration of all PIs in each of the three 3-min oral readings was derived using the MPI program software. This software also superimposed a 5-SI template over each 3-min oral reading record. The PI data were then collated within Microsoft Excel files. The 5-SI in each participant's oral reading that contained a complete or partial stuttered event (identified from the mouse-button-press record) were then able to be isolated within the file. This made it possible to quantify each 3-min trial both with and without stuttered intervals. Each affected participant's 3-min trial PI record was then aligned with their age-matched control participant's PI record so that corresponding 5-SI could be removed from both PI records to test for differences between the groups when stuttered intervals were removed.

The three 3-min oral readings from each participant were collapsed into one data set so that all PIs could be ranked from the shortest to the longest duration. The 30–1,000-ms range was then subdivided into 50-ms ranges (except for the 30–50-ms range, which was left as a 20-ms subdivision) and the total number of PIs from each participant that occurred within each of these 20 subdivisions provided the raw data. These raw data were also converted into the percentage of each participant's total PIs within each of the 20 subdivisions. This conversion was necessary to control for the effect of different speaking rates within and between the speaker groups (see the Results section). The mean total number of PIs and percentage of total PIs within each of the 20 subdivisions were calculated for the following criteria: (a) the entire 9-min of the affected speakers' speech, (b) the matched control participants' speech, (c) the stutter-free speech of the speakers who stuttered, and (d) the matched control participants' speech minus those segments "matching stuttered intervals."

Calibration and Reliability

The accuracy of the MPI system's measurements of the duration of PIs was determined by comparing MPI-recorded PI durations with PI durations identified using an acoustic analysis program. These durations were obtained during two independent tests: (a) a trial test using a randomly selected recording of a participant's 3-min oral reading trial and (b) a nontrial test using oral reading of a phonetically balanced series of three-word combinations. The trial-test intervals were the first 50 PIs

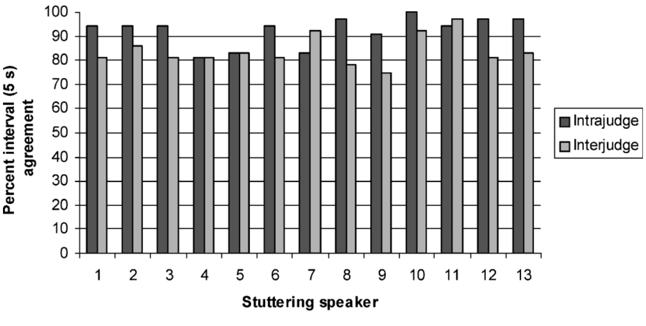
---

[3] The decision to use 50 ms was somewhat, but not completely, arbitrary. At the extremes, we could have used a 1-ms range or a much larger range (e.g., 250 ms). The 50-ms unit was selected because it had been used in initial investigations (Ingham, Montgomery, & Ulliana, 1983) and would subdivide the PI range found to be functional in previous studies (~30–150 ms; Gow & Ingham, 1992; Ingham et al., 2001). The ceiling of 1,000 ms was a post hoc decision that was based on the low frequency of PIs exceeding that size. Two participants from each group displayed zero data in 50-ms boxes beyond 1,000 ms, and only 15 PIs, evenly distributed across both groups, exceeded 1,000 ms.

recorded during an affected speaker's trial. They ranged from 18 ms to 870 ms. The PI durations, as measured by the MPI program, were then compared with the durations derived from the trial's acoustic recording using the PRAAT (version 4.2.07) acoustic analysis program (Boersma & Weenink, 2003). During the nontrial test, the MPI system measured PI durations while a normally fluent speaker was recorded producing a series of three-word utterances, each separated by approximately 5-s silent periods. The first 50 PI intervals recorded and measured by the MPI program were selected from this task for comparison with the durations derived from the PRAAT, as described above. They ranged from 198 ms to 624 ms. For the trial test, the 40/50 intervals measured by the MPI system and by PRAAT differed by 20 ms or less (five differed by 23–28 ms; five by 31–37 ms). For the nontrial test, 41/50 intervals measured by both systems differed by 20 ms or less (two differed by 22–23 ms; seven by 34–40 ms). The similarity between PI measurement accuracy obtained during both test conditions indicates that the MPI program's phonation-interval measures possessed satisfactory accuracy, irrespective of speaking task.

Interjudge reliability of stuttering judgments was assessed by a research assistant trained on the SMS and SMAAT programs and who was unfamiliar with the study. Using the SMS program, this assistant rated one of each affected speaker's 3-min audio-visually recorded speaking tasks. Interjudge and intrajudge agreement are displayed in Figure 1. The figure shows that the percentage of agreed stuttered intervals by the experimenter and research assistant ranged between 75% and 97% across the speakers. The experimenter's intrajudge reliability was calculated by arranging a "blinded" rerating of the same set of speaking task recordings at least 3 months after the experimental trials were completed. Intrajudge agreement for stuttered intervals ranged from 81% to 100% across the speakers. It should be noted that this exacting agreement metric was not inflated by the inclusion of the much higher number of nonstuttered intervals.

Figure 1. Interjudge and intrajudge agreement for 5-s intervals (per 3 min) of speech that contained a stuttering event.



Interjudge reliability of the syllables per minute (SPM) and SFSPM data was assessed separately from the stuttering judgment measures by comparing the data obtained by the experimenter and those obtained by another research assistant also trained on the SMS and SMAAT programs. This assistant rated the 3-min audiovisual recordings from each of 6 speakers who stuttered and 6 control participants. Differences between the experimenter's and assistant's SPM scores ranged from 0.9 to 5.7 SPM, a difference of 0.4% to 2.4% between their scores. The corresponding SFSPM score differences ranged from 1.3 to 5.9 SFSPM or a 0.6% to 2.6% difference between their scores. These differences did not exceed the within- or between-group differences in the affected speakers' or control participants' speaking-rate scores.

Results

The principal metric used to quantify the distribution of PIs in this study was the percentage of total PIs produced by each participant. These data were obtained from the total PIs for 9 min of oral reading (3 × 3 min) that were then allocated to each of the 20 subdivisions covering the 30–1,000-ms PI range (except for the 30–50-ms interval; see above). As mentioned above, it was necessary to convert the raw data to percentage data to control for the effects of the within- and between-group speaking rates as shown in Table 1. There was a 21.8% mean difference in the overall SPM rates for both groups (speakers who stuttered, M SPM = 198.2, SD = 34.5, vs. control participants, M SPM = 253.7, SD = 18.5), $t(24) = 5.11$, $p < .01$, $d = 2.99$, which was only reduced to 10.5% by using stutter-free-interval SPM data (speakers who stuttered, M SFSPM = 227.1, SD = 39.9, vs. control participants, M SFSPM = 253.7, SD = 20.5), $t(24) = 2.18$, $p < .05$, $d = 1.30$. Predictably, these differences were reflected in the mean total PIs for both groups (speakers who stuttered, M PIs = 1029.2, SD = 166.6, vs. control participants, M PIs = 1,170.5, SD = 192.1), $t(24) = 2.003$, $p < .05$, $d = 0.85$, although the differences between the groups' PI means during stutter-free speech did not reach significance (speakers who stuttered, M PIs = 548.9, SD = 288.9, vs. control participants, M PIs = 632.5, SD = 204.0), $t(24) = 0.236$, $p > .05$.

We conducted log-linear chi-square analyses (Wickens, 1989) on the PI frequencies within the 20-PI-range subdivisions from the 26 participants (ns = 13 per group), that is, a total of 520 data cells. Wickens (1989) cautioned that chi-square tests require that "separate observations are probabilistically independent" (pp. 27–28), implying that each data cell observation (a PI) should derive from a separate participant (subject). He also noted, however, that a "series of trials from a single subject sometimes can be treated as independent events" (pp. 27–28), as in the case of psychophysical data. Thus, PIs were conceptualized as independent events because there is no evidence that the occurrence of a particular PI will modify the probability of occurrence of a different duration PI.

To test whether the relative frequency of responses in different subdivisions changes across groups, the relative subdivision changes were treated as estimates of the population distribution of such frequencies for a group or for an individual. Ideally, two hypotheses should be tested: H_1 = the distribution functions for group participants are identical, and H_2 (which assumes H_1 is true) = the distribution functions for the two groups are identical. However, absent such a test procedure, log-linear independence tests (see Wickens, 1989, pp. 33–36) were applied to tests for dependence between groups and subdivisions (frequencies in different PI ranges) and the dependence of participant effects and subdivision frequencies. Unfortunately, the theory for such an analysis does not help determine the dependence of participants nested within groups with other variables; indirect evidence about these effects must be obtained.

Another reason for performing the log-linear independence tests described by Wickens (1989) is that traditional chi-square tests are not applicable to three-dimensional or higher dimensional data structures. Thus, Wickens wrote, “One can extend the analysis to more than two classifications. The respondents in [a] poll can be classified by sex as well as region and opinion, to make a 4 × 2 × 3 table. Analyzing this multiway table allows more complicated relationships to be revealed” (p. 2). Wickens also noted objections to collapsing a three- or more-way table to a group of two-way tables, concluding, “All this is enough to demand a set of higher-order analysis techniques and an organized set of procedures to employ them” (p. 2). Log-linear analyses or, more generally, multiway contingency-tables analysis, are those techniques.

Complete Data Set Analysis

For the complete data set, which included stuttered intervals, we would have liked to segregate Participant × Subdivision effects within groups and Group × Subdivision effects within an overall analysis. Because we could not find a way to perform this segregation, we performed one log-linear analysis to assess Group × Subdivision independence, yielding a chi square of 86.83 ($p < .0001$, $N = 519$, $df = 19$). In the absence of Participant × Subdivision effects, this suggests that group membership produced a different distribution of relative PI frequencies in the different PI ranges. An additional log-linear analysis of the speakers who stuttered yielded a Participant × Subdivision independence chi square of 2,258.16 ($p < .0001$, $N = 259$, $df = 228$). In addition, a log-linear analysis of control participants yielded a Participant × Subdivision independence chi square of 3,123.63 ($p < .0001$, $N = 260$, $df = 228$). This indicates that, regardless of group membership, participants in any given group had different distributions of relative PI frequencies in different subdivisions. Hence, the log-linear analyses suggest that the Group × Subdivision interaction is likely an artifactual difference that is due to Participant × Subdivision effects. It is not clear how much residual effect of Group × Subdivision interaction remains.

Stutter-Free Intervals Analysis

For the data set excluding stuttered intervals, a log-linear analysis yielded a Group × Subdivision independence chi square of 34.05 ($p < .0001$, $N = 494$, $df = 19$). This suggests that group membership produced a different distribution of relative frequencies in different PI ranges. However, as before, the averaged Participant × Subdivision relative frequencies could account for some or all of the Group × Subdivision effect. An additional log-linear analysis of speakers who stuttered yielded a Participant × Subdivision independence chi square of 5,246.27 ($p < .0001$, $N = 245$, $df = 225$). In addition, a log-linear analysis of control participants yielded a Participant × Subdivision independence chi square of 4,048.31 ($p < .0001$, $N = 249$, $df = 227$). This indicates that regardless of group membership, participants in any given group had different distributions of relative PI frequencies in different subdivisions. Hence, the log-linear analyses suggest that, at least in part, the Group × Subdivision interactions reflect artifactual differences that are due to Participant × Subdivision effects. Again, it is not clear how much residual effect of Group × Subdivision interactions remains.

A second set of analyses involved tests of group effects on PI responses within each subdivision. Using *t* tests, we compared the relative PI frequencies within each PI range for the two groups, using each individual’s relative frequency (proportion per subdivision) and computing a mean for each participant. In all *t* tests, we used a two-tailed procedure and assumed unequal variance. For the data set that included both stuttered and nonstuttered intervals (see Figure 2), the 20 *t* tests for different PI ranges were inspected using a Bonferroni correction.[4] This required significant effects to be at the .0026 level or beyond. The nearest significant effect was .041. For the data set that included only nonstuttered

---

[4] A Bonferroni inequality correction for each set of 20 *t* tests is justified for the following reasons. For a given α criterion of significance for any single *t* test, the binomial theorem probability based on 20 independent tests of no significant results is $(1 - \alpha)^{20}$. For dependent tests, an inequality applies, yielding $1 - (1 - \alpha)^{20}$ as the maximum possible probability of one or more significant results. Therefore, given $.05 = 1 - (1 - \alpha)^{20}$, then $\alpha = .0026$. There is, however, a special issue here: Although we assumed the number of PIs in one subdivision to be independent of the number in any other PI range, an analysis using proportions in each PI range was constrained, with the total proportion being 1.0. Because the Bonferroni inequality takes an independent binomial result as its upper bound, the ipsative nature of these data does not invalidate the present conclusions.

Figure 2. Distribution of phonated intervals (PI; in 50-ms subdivisions) during speakers who stuttered's speech (n = 13) and control speakers' speech (n = 13).

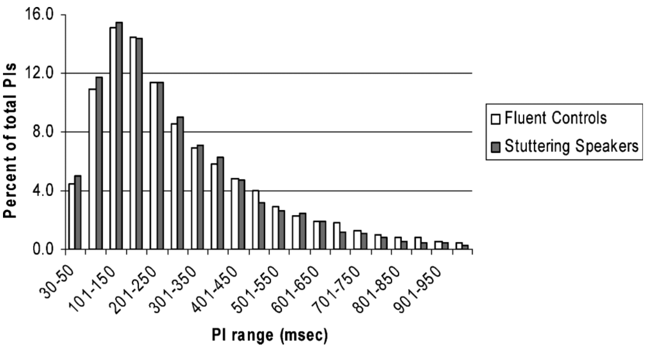


Table 2. Product–moment correlations between the proportion of PIs per subdivision for the complete speech samples from the speakers who stuttered (n = 13) and control participants (n = 13).

Variable	Speakers who stuttered	Controls (S-F)	Speakers who stuttered (S-F)
Controls	.997	.999	.997
Speakers who stuttered	—	.998	.997
Controls ("S-F")	—	—	.996

Note. PIs = phonated intervals. The correlations between PI proportions obtained from stutter-free (S-F) speech of the speakers who stuttered (stuttered segments deleted) and the control participants' with the corresponding and identical segments (S-F) are deleted.

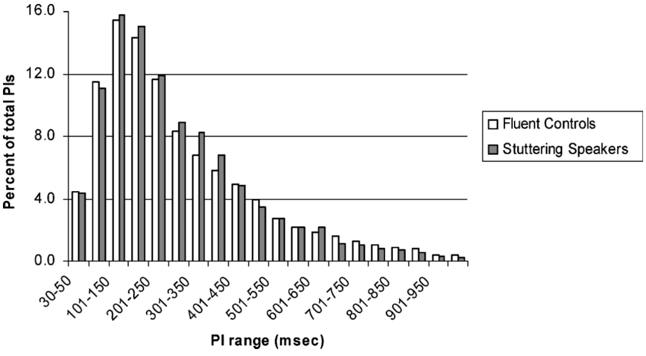
intervals (see Figure 3), the 20 t tests were again inspected using a Bonferroni correction, where significance would be obtained only at .0026 or less. The nearest significance level was .024.

Last, the level of the relationship between the distributions of total data sets from both groups was demonstrated by a product–moment correlation matrix (see Table 2). This table shows that the correlation between any pair of distributions of PIs, both between and within groups, was not less than .996. From this overall comparison, we conclude that not only was there no evident difference between the PI distributions for speakers who stutter and normally fluent controls, but there was also no evidence that removing stuttering intervals from the speech samples changed the PI distribution.

Discussion

The results discussed above indicate that adults and adolescents who stutter do not display an abnormal distribution of PIs in their speech, but their PI distribution is strikingly similar to that of normal speakers—at least this is the case during oral reading. It was of particular interest that no differences between the groups were found in the 30–150-ms PI range in either the stuttered or stutter-free speech of the speakers who stuttered compared with normal speakers (this range accounted for 16.7% of PIs in the present study, which is less than the approximately 20.0% reported in previously published studies on speakers who stutter). As mentioned in the beginning of this article, this finding is of interest because researchers have shown that reducing the number of PIs within this range dramatically reduces stuttering (Ingham et al., 1983; Gow & Ingham, 1992; Ingham et al., 2001). Furthermore, this reduction occurs without a concomitant increase in longer duration PIs (see Gow & Ingham, 1992). Previous studies (Andrews et al., 1982; Pindzola, 1987; Prosek & Runyan, 1982) have suggested that the stutter-free speech of adult speakers who stutter, particularly during some fluency-inducing procedures (e.g., masking, shadowing, delayed-auditory-feedback–prolonged speech and rhythmic speech) may involve concomitant increases in longer duration PIs. However, the focus of methodologies in these studies was on longer duration PIs and may have obscured equally important reductions in short-duration PIs. Nonetheless, these studies have led to the reasonable conclusion that speakers who stutter may be characterized by an abnormal distribution of PIs. The findings of the present study do not support that contention.

Figure 3. Distribution of phonated intervals (in 50-ms subdivisions) during speakers who stuttered's stutter-free speech and controls' speech using matched speech samples.



It should be recognized, of course, that in this study, the speakers who stuttered and the control participants differed with respect to speech rate and total number of PIs that they produced. The affected speakers, as expected, read aloud more slowly than the control participants, but they were also relatively slower during their stutter-free speech. As a result, the affected speakers' total PIs in all PI ranges were significantly less than the control speakers' PIs, although this was true only when PI frequencies were derived with stuttering present (the lower PI frequencies when stuttering was absent did not reach significance). In other words, the speakers who stuttered would be expected to produce relatively fewer short-duration PIs (~13%) simply because they produced fewer PIs overall. In this regard, the cohort of experimental participants in the present study was not unusual in speaking at slower than normal speaking rates (see Bloodstein, 1944). Previous researchers have shown that when intervals of speech containing stuttering are excluded from analysis, speakers who stutter may still display a perceptually tenuous fluency (Young, 1984) that may be characterized by unusual pauses (Love & Jeffress, 1971) and other unusual articulatory features (Di Simoni, 1974). These unusual features are most likely learned adjustments to chronic stuttering because they do not characterize the speech of young children who stutter (Colcord & Gregory, 1987; Krikorian & Runyan, 1983). Although these adjustments might be learned, it does not imply that the entire speech production system is maladjusted. This seems to be evident from an investigation of speech rate in adult speakers who stuttered by Smith and Kleinow (2000). They found that even when speech rate was manipulated, speakers who stuttered "can speak fluently at habitual and fast rates with articulatory kinematic parameters overlapping those of normally fluent speakers" (p. 534).

One interesting finding from the present study concerned the effect on the PI distribution of removing stuttered speech intervals from the affected speakers' samples. The very high correlation between the PI distributions of the complete speech samples and the stutter-free samples ($r = .997$) suggests that occasions of stuttering do not interact with the frequency of PIs in particular duration ranges. The present finding replicates a similar effect obtained in an unpublished investigation of the MPI by Gow (1998). At the least, this suggests that either the phonatory gestures that typify stuttering events (Conture, McCall, & Brewer, 1977; Conture, Schwartz, & Brewer, 1985; Shapiro & De Cicco, 1982) do not have abnormal features that alter the distribution of PIs within affected speakers' speech patterns or that the phonatory gestures are simply randomly distributed across the PI duration ranges.

The results of the present study imply that a disproportionate production of short-duration PIs is not part of the core problem in stuttering. However, it is possible that it is not the frequency of short-duration PIs that is problematic, but the affected speakers' ability to manage the production of short-duration PIs. The dominant theory behind the search for any impairment in affected speakers' speech production skills has been various versions of a speech–motor-deficit model (e.g., Zimmermann, 1980). It is not clear, however, that such peripherally focused models have advanced the understanding of the source of such a deficit (see Ingham, 1998). For that reason, there may be more to gain from a search that is framed by the nascent neural system models of speech production (Indefrey & Levelt, 2004; Jürgens, 2002). From that perspective, the present findings might be more productively interpreted by relating them to the findings of neuroimaging studies on speakers who stutter and normally fluent speakers.

The present findings may be related to those obtained by Ingham et al. (2003) in a PET study conducted with 17 adult speakers who stuttered (9 men) undergoing MPI treatment. One feature of the findings was the interesting role of putamen.[5] It is gradually becoming apparent from brain imaging research that putamen and specific regions of cerebellum either interact, or are involved in, managing the production of syllables of varying duration and rate (Jeffries, Fritz, & Braun, 2003; Riecker, Wildgruber, Dogil, Grodd, & Ackermann, 2002; Sidtis, Strother, & Rottenberg, 2003; Vanlancker-Sidtis, McIntosh, & Grafton, 2003; Wildgruber, Ackermann, & Grodd, 2001). In addition, researchers have found that during stuttered speech, the basal ganglia in general (Brown, Ingham, Ingham, Laird, & Fox, 2005) and right putamen in particular are abnormally inactive (Fox et al., 1996; Ingham et al., 2003). However, after receiving partial MPI treatment, when the participants' frequency of short PIs was reduced, right putamen was activated (Ingham et al., 2003). Activation in putamen was also reported during monologue tasks by 4 individuals who had recovered from stuttering (without formal treatment) after adolescence (see Ingham et al., 2003). These findings are particularly interesting when related to a recent PET study by Wildgruber et al. (2001). They found that when normally fluent speakers repeated a short phonated syllable (/ta/) at a rate of 4.0–5.5/s, there were consistent cerebellum and putamen activations. However, while repeating that syllable at a much slower rate (2.5–4.0/s), cerebellum was inactive, but putamen continued to be active. It would be expected that at the faster rate, the syllable required much shorter PIs than at the slower rate.[6] The reduced frequency of short PIs

---

[5] A recent review by Alm (2004) also suggested that this part of the basal ganglia may have an important role in stuttering. Alm's review, which draws attention to the role of putamen in stuttering, is derived from an analysis of only a portion of the current brain imaging research on stuttering. This may explain why the review overlooks the role of some important cortical regions that might interact with putamen and likely have equivalent prominence in any neural system that ultimately emerges as functionally related to stuttering. Foremost among these regions would be those in the temporal lobe that have been described by Braun et al. (1997), Fox et al. (2000), and Ingham et al. (2004) as displaying CBF activity that is negatively correlated with stuttering frequency.

[6] A series of trials were conducted in the second author's research laboratory at the University of California, Santa Barbara, using the procedure described by Wildgruber et al. (2001) and the MPI program. Wildgruber et al. showed that all PIs produced by 2 male and 3 female, normally fluent adults when uttering /ta/ at the faster rate (4.0–5.5/s) for 5 s were less than 100 ms, but at the slower rate (2.5–4.0/s for 5 s) they consistently exceeded 150 ms.

during post–MPI-treatment, stutter-free monologues in the Ingham et al. (2003) study would almost certainly have involved reduced PIs produced at a rate of 4.0–5.5/s but would have allowed for PIs to be produced at the rate of 2.5–4.0/s. In other words, although speakers who stutter may not engage putamen activity (as do normally fluent speakers) during their stuttered speech production, putamen activations do occur when their short PIs and stuttering are reduced. The obvious implication is that improved fluency in speakers who stutter may be obtained by reducing the use of short PIs that consistently fail to activate a neural region, putamen, which is necessary for normally fluent speech.

The present study strongly suggests that the PI distribution in people who stutter is not different from that of people who do not stutter. This has important implications for future research into the treatment and cause of this disorder. The findings tend to suggest that the production of phonation per se is not abnormal in speakers who stutter, a finding that is consistent with the results of many investigations of speech–motor activity among speakers who stutter (Ingham, 1998). We speculate that the part of the source of difficulty in managing this particular speech–motor activity in a way necessary for fluent speech production will be located in a neural system dysfunction related to basal ganglia.

Acknowledgments

This study was conducted with support of a faculty research grant awarded to the second author by the Faculty Research Committee of the University of California, Santa Barbara. We thank Allison Grant, Irene Seybold, and Melanie Gomez for their assistance with the stuttering and phonated intervals measurement reliability analysis. We are also grateful for the advice given by Rebecca Zwick and Thomas Wickens with respect to the chi-square analyses. The responsibility for the analyses themselves is ours alone. Last, thanks are given to Janis Ingham for her careful editing of the manuscript.

References

Alm, P. A. (2004). Stuttering and the basal ganglia circuits: A critical review of possible relations. *Journal of Communication Disorders, 37,* 325–369.

Andrews, G., Howie, P. M., Dozsa, M., & Guitar, B. E. (1982). Stuttering: Speech pattern characteristics under fluency-inducing conditions. *Journal of Speech and Hearing Research, 25,* 208–216.

Bloodstein, O. (1944). Studies in the psychology of stuttering: XIX. The relationship between oral reading rate and severity of stuttering. *Journal of Speech Disorders, 9,* 161–173.

Bloodstein, O. (1995). *A handbook on stuttering* (5th ed.). San Diego, CA: Singular.

Boersma, P., & Weenink, D. (2003). PRAAT (Version 4.2.07) [Computer software]. Institute of Phonetics Sciences, University of Amsterdam, The Netherlands. Retrieved October 12, 2004, from http://www.praat.org

Borden, G. J., Baer, T., & Kenney, M. K. (1985). Onset of voicing in stuttered and fluent utterances. *Journal of Speech and Hearing Research, 28,* 363–372.

Bothe, A. K., Davidow, J. H., Ingham, R. J., Crowe, B. T., Bramlett, R. E., Levy, J., & Taylor, K. (2003, November). *Systematic review of the stuttering treatment literature.* Paper presented at the Annual Convention of the American Speech-Language-Hearing Association, Chicago, IL.

Braun, A. R., Varga, M., Stager, S., Schulz, G., Selbie, S., Maisog, J. M., Carson, R. E., & Ludlow, C. L. (1997). Altered patterns of cerebral activity during speech and language production in developmental stuttering. An $H_2{}^{15}O$ positron emission tomography study. *Brain, 120,* 761–784.

Brayton, E. R., & Conture, E. G. (1978). Effects of noise and rhythmic stimulation on the speech of stutterers. *Journal of Speech and Hearing Research, 21,* 285–294.

Brown, S., Ingham, R. J., Ingham, J. C., Laird, A. R., & Fox, P. T. (2005). Stuttered and fluent speech production: An ALE meta-analysis of functional neuroimaging studies. *Human Brain Mapping, 25,* 105–117.

Colcord, R. D., & Adams, M. R. (1979). Voicing duration and vocal SPL changes associated with stuttering reduction during singing. *Journal of Speech and Hearing Research, 22,* 468–479.

Colcord, R. D., & Gregory, H. H. (1987). Perceptual analyses of stuttering and nonstuttering children's fluent speech. *Journal of Fluency Disorders, 12,* 185–195.

Conture, E. G., McCall, G. N., & Brewer, D. W. (1977). Laryngeal behavior during stuttering. *Journal of Speech and Hearing Research, 20,* 661–668.

Conture, E. G., Schwartz, H. D., & Brewer, D. W. (1985). Laryngeal behavior during stuttering: A further study. *Journal of Speech and Hearing Research, 28,* 233–240.

Di Simoni, F. G. (1974). Preliminary study of certain timing relationships in the speech of stutterers. *Journal of the Acoustical Society of America, 56,* 695–696.

Finn, P., & Ingham, R. J. (1989). The selection of "fluent" samples in research on stuttering: Conceptual and methodological considerations. *Journal of Speech and Hearing Research, 32,* 401–408.

Fox, P. T., Ingham, R. J., Ingham, J. C., Hirsch, T., Downs, J. H., Martin, C., Jerabek, P., Glass, T., & Lancaster, J. L. (1996). A PET study of the neural systems of stuttering. *Nature, 382,* 158–162.

Fox, P. T., Ingham, R. J., Ingham, J. C., Zamarripa, F., Xiong, J.-H., & Lancaster, J. (2000). Brain correlates of stuttering and syllable production: A PET performance-correlation analysis. *Brain, 123,* 1985–2004.

Goldiamond, I. (1965). Stuttering and fluency as manipulatable operant response classes. In L. Krasner & L. P. Ullman (Eds.), *Research in behavior modification* (pp. 106–156). New York: Holt.

Goldiamond, I. (1967). *Supplementary statement to operant analysis control of fluent and non-fluent verbal behavior.*

Report to U.S. Department of Health, Education, and Welfare (Public Health Service Application No. MH-8876-03).

Gow, M. L. (1998). *Modifying phonation interval distributions during solo and chorus reading: The effect on stuttering*. Unpublished doctoral dissertation, University of California, Santa Barbara.

Gow, M. L., & Ingham, R. J. (1992). The effect of modifying electroglottograph identified intervals of phonation on stuttering. *Journal of Speech and Hearing Disorders, 35,* 495–511.

Healey, E. C., & Adams, M. R. (1981). Speech timing skills of normally fluent and stuttering children and adults. *Journal of Fluency Disorders, 6,* 233–246.

Indefrey, P., & Levelt, W. J. M. (2004). The spatial and temporal signatures of word production components. *Cognition, 92,* 101–144.

Ingham, R. J. (1984). *Stuttering and behavior therapy: Current status and empirical foundations*. San Diego, CA: College-Hill Press.

Ingham, R. J. (1990). Stuttering. In A. S. Bellack, M. Hersen, & A. E. Kazdin (Eds.), *International handbook of behavior modification and therapy* (pp. 599–631). New York: Plenum.

Ingham, R. J. (1998). On learning from speech-motor control research on stuttering. In A. K. Cordes & R. J. Ingham (Eds.), *Treatment efficacy for stuttering: A search for empirical bases* (pp. 67–101). San Diego, CA: Singular.

Ingham, R. J., Bakker, K., Ingham, J. C., Kilgo, M., & Moglia, R. (1999). *Stuttering Measurement System (SMS)* [Computer software and manual]. Santa Barbara: University of California, Santa Barbara.

Ingham, R. J., Cordes, A. K., Kilgo, M., & Moglia, R. (1998). Stuttering Measurement Assessment and Training (SMAAT) [Computer software and CD]. Santa Barbara: University of California, Santa Barbara.

Ingham, R. J., Fox, P. T., Ingham, J. C., Xiong, J.-H., Zamarripa, F., Hardies, L. J., & Lancaster, J. L. (2004). Brain correlates of stuttering and syllable production: Gender comparison and replication. *Journal of Speech, Language, and Hearing Research, 47,* 321–341.

Ingham, R. J., Ingham, J. C., Finn, P., & Fox, P. T. (2003). Towards a functional neural systems model of developmental stuttering. *Journal of Fluency Disorders, 28,* 297–318.

Ingham, R. J., Kilgo, M., Ingham, J. C., Moglia, R., Belknap, H., & Sanchez, T. (2001). Evaluation of a stuttering treatment based on reduction of short phonation intervals. *Journal of Speech, Language, and Hearing Research, 44,* 1229–1244.

Ingham, R. J., Moglia, R., Kilgo, M., & Felino, A. (1997). *Modifying Phonation Interval (MPI) stuttering treatment schedule* [Computer software and manual]. Santa Barbara: University of California, Santa Barbara.

Ingham, R. J., Montgomery, J., & Ulliana, L. (1983). An investigation on the effect of manipulating phonation duration on stuttering. *Journal of Speech and Hearing Research, 26,* 579–587.

Jeffries, K. J., Fritz, J. B., & Braun, A. R. (2003). Words in melody: An $H_2{}^{15}O$ PET study of brain activation during singing and speaking. *Neuroreport, 14,* 749–754.

Jürgens, U. (2002). Neural pathways underlying vocal control. *Neuroscience and Biobehavioral Review, 26,* 235–258.

Kagan, J. (1998). *Three seductive ideas*. Cambridge, MA: Harvard University Press.

Krikorian, C. M., & Runyan, C. M. (1983). A perceptual comparison: Stuttering and nonstuttering children's nonstuttered speech. *Journal of Fluency Disorders, 8,* 283–290.

Linnea, A. (1995). *Deep water passage*. Boston, MA: Little, Brown.

Love, L. R., & Jeffress, L. A. (1971). Identification of brief pauses in the fluent speech of stutterers and nonstutterers. *Journal of Speech and Hearing Research, 14,* 229–240.

Onslow, M., & Ingham, R. J. (1987). Speech quality measurement and the management of stuttering. *Journal of Speech and Hearing Research, 52,* 2–17.

Packman, A., Onslow, M., & van Doorn, J. (1994). Prolonged speech and modification of stuttering: Perceptual, acoustic, and electroglottographic data. *Journal of Speech and Hearing Research, 37,* 724–737.

Peters, H. F. M., Boves, L., & van Dielen, I. C. (1986). Perceptual judgment of abruptness of voice onset in vowels as a function of the amplitude envelope. *Journal of Speech and Hearing Disorders, 51,* 299–308.

Pindzola, R. H. (1987). Durational characteristics of the fluent speech of stutterers and nonstutterers. *Folia Phoniatrica, 39,* 90–97.

Prosek, R. A., & Runyan, C. M. (1982). Temporal characteristics related to the discrimination of stutterers' and nonstutterers' speech samples. *Journal of Speech and Hearing Research, 25,* 29–33.

Ridley, M. (1999). *Genome: The autobiography of a species in 23 chapters*. New York: Harper Collins.

Riecker, A., Wildgruber, D., Dogil, G., Grodd, W., & Ackermann, H. (2002). Hemispheric lateralization effects of rhythm implementation during syllable repetitions: An fMRI study. *Neuroimage, 16,* 169–176.

Shapiro, A. I., & De Cicco, B. A. (1982). The relationship between normal dysfluency and stuttering: An old question revisited. *Journal of Fluency Disorders, 7,* 109–121.

Sidtis, J. J., Strother, S. C., & Rottenberg, D. A. (2003). Predicting performance from functional imaging data: Methods matter. *Neuroimage, 20,* 615–624.

Smith, A., & Kleinow, J. (2000). Kinematic correlates of speaking rate changes in stuttering and normally fluent adults. *Journal of Speech, Language, and Hearing Research, 43,* 521–536.

Vanlancker-Sidtis, D., McIntosh, A. R., & Grafton, S. (2003). PET activation studies comparing two speech tasks widely used in surgical mapping. *Brain and Language, 85,* 245–261.

Wickens, T. D. (1989). *Multiway contingency tables analysis for the social sciences*. Hillsdale, NJ: Erlbaum.

Wildgruber, D., Ackermann, H., & Grodd, W. (2001). Differential contributions of motor cortex, basal ganglia, and cerebellum to speech motor control: Effects of syllable repetition rate evaluated by fMRI. *Neuroimage, 13,* 101–109.

Young, M. A. (1984). Identification of stuttering and stutterers. In R. F. Curlee & W. H. Perkins (Eds.), *Nature and treatment of stuttering: New directions* (pp. 13–30). San Diego, CA: College Hill Press.

Zebrowski, P. M., Conture, E. G., & Cudahy, E. A. (1985). Acoustic analysis of young stutterers' fluency: Preliminary investigations. *Journal of Fluency Disorders, 10,* 173–192.

Zimmermann, G. (1980). Stuttering: A disorder of movement. *Journal of Speech and Hearing Research, 23,* 122–136.

Received December 27, 2004

Accepted May 3, 2005

DOI: 10.1044/1092-4388(2006/013)

Contact author: Roger J. Ingham, Department of Speech and Hearing Sciences, University of California, Santa Barbara, Santa Barbara, CA 93106. E-mail: rjingham@speech.ucsb.edu