Disfluency: An Exploratory Study of the Effects of Subcortical Stroke

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Abstract: Purpose: Some stroke survivors exhibit disfluencies (e.g., repetitions, false starts, revisions, and filled pauses) in their discourse. Relatively little is known about the natural recovery trajectory of disfluencies following a subcortical stroke. The purpose of this study was to examine and describe the rate of occurrence of disfluencies in individuals with a history of subcortical stroke but absent a formal speech or language impairment diagnosis.

Method and Procedure: Discourse samples from 6 individuals with a history of subcortical stroke and 6 matched controls were examined for the presence of disfluencies at 3 intervals: 1, 6, and 12 months after a stroke. Nonparametric tests were completed on the individuals to analyze the presence of disfluency during their first year following a stroke.

Results: The total number of disfluencies and the disfluency rate were higher in the participants with subcortical stroke than in the controls. Disfluency rates were highly variable across the participants with subcortical stroke. Overall, the disfluency rate per minute declined in the first year following a stroke.

Conclusion: Disfluencies are a negative consequence of subcortical stroke and are disruptive to poststroke communication. Future studies are required to examine the relationship between disfluencies that occur after a subcortical stroke and the patient's communicative efficiency and effectiveness.

Keywords: disfluencies, speech, expressive language, stroke

Disfluency is a break in the continuous production of speech that is characterized by false starts, revisions, prolongations, hesitations, and/or repetitions. Disfluencies are associated with both normal and abnormal motor speech and linguistic functioning (American Speech-Language-Hearing Association [ASHA], 1999; Duffy, 2005). Hesitations, long pauses (filled and unfilled), and phrase repetitions are commonly regarded as normal disfluencies (ASHA, 1999). In contrast, whole-word repetitions, part-word repetitions, prolongations, false starts, and revisions are commonly believed to be abnormal disfluencies. The generally accepted rate of normal disfluency among adults as reported in previous studies is 3% of the words uttered (De Nil, Saisisekaran, Van Lieshout, & Sandor, 2005; Robb, Bloomgren, & Chen, 1998; Shames & Rubin, 1983) or three disfluencies (stuttered words) per minute (Ryan, 1974).

Neurologically based disfluency, or neurogenic disfluency, is disfluent speech resulting from a neurological disease or disorder (Duffy, 2005). According to Duffy, neurogenic disfluency is stuttering-like behavior that is similar to a disfluency of developmental causes and is characterized by similar sound or syllable repetition, blocking, prolongations, or hesitations. Neurogenic disfluency is associated with a number of neurologically based communication disorders, including aphasia, apraxia of speech, dysarthria, and cognitive–affective disorders. Parkinson’s disease, tumors, drug toxicity, progressive supranuclear palsy, and stroke are among the common etiologies of neurogenic disfluency (Duffy, 2005).

Neurogenic disfluency is not a homogeneous disorder (Helm-Estabrooks, 1993). Studies report pathologies that are cortical (Helm & Butler, 1980; Sahin, Krepis, Yilmaz, & Coban, 2005), subcortical (Alm, 2004; Ciabarra, Elkind, Roberts, & Marshall, 2000; De Nil et al., 2005; Van Borsel, Van Der Made, & Santens, 2003), unilateral
expressive language deficits that were largely different from patterns of language impairment emerged. Similarly, Copland, Nadeau and Crosson (1997) found that highly variable patterns of disfluencies following subcortical strokes. The basal ganglia also has a critical role as part of the motor speech programming mechanism (Duffy, 2005). Therefore, subcortical strokes can result in distinctive motor speech disorders that can be attributed to disruption of the cortical–basal ganglia pathways (Okuda et al., 1999). Duffy noted that subcortical strokes to the basal ganglia region are more likely to result in more profound deficits than are strokes in cortical areas. In summary, considerable evidence indicates that strokes to the subcortical mechanism are associated with language and motor speech deficits. However, less is known about the natural history of speech and language functions following a subcortical stroke.

Therefore, the purpose of this investigation was to describe the presence of disfluencies and their rate of occurrence in the discourse of a cohort of individuals who sustained a subcortical stroke primarily in the area of the basal ganglia and were in their first year following a stroke. Many speakers complain of mild and intermittent “speech difficulties” after stroke; however, these difficulties do not consistently emerge during standardized assessments. Therefore, we examined the presence of disfluencies, which are a frequent complaint among stroke patients who have neither a history nor a diagnosis of a specific speech or language disorder. A second goal was to determine if the frequency of disfluencies changed across time in the first year following a stroke. The examination of disfluencies across time allowed us to explore the natural recovery patterns of disfluencies following a subcortical stroke.

METHOD

Participants

Twelve participants consisting of 6 adults with a history of a subcortical stroke and 6 matched adults with no stroke history were included in the study. All participants were male. The 6 participants with a diagnosed subcortical stroke were selected from a larger stroke recovery and caregiving study conducted by the Health Services Research and Development Service of the Department of Veterans Affairs (Rittman, 2001). Language samples selected for the analysis of disfluency were obtained from qualitative interviews that were completed as part of the stroke recovery and caregiving study. Each participant was interviewed at 1, 6, 12, 18, and 24 months following a stroke in his home. Language samples were obtained from the 1-, 6-, and 12-month interviews. Each semistructured interview included the same questions. Interviews examined a number of issues related to stroke recovery, including but not limited to descriptions of stroke onset, initiation of treatment, hospitalization and discharge, poststroke impairments, difficulties encountered after discharge, and socialization and adjustment issues. All data collectors were trained to minimally interrupt the participants in their attempts to elicit each stroke patient’s account of the stroke experience. Therefore, each interview was more likely to contain a representative sample of the participant’s typical daily communication in a relaxed
Participants gave written consent and approved by the institution review board and all participants for the stroke and caregiving study and this study were reviewed for rehabilitation services to enhance their recovery. The stroke participants were discharged home after their stroke, and none had received specialized speech/language evaluation or had a documented speech/language disorder. All stroke participants were discharged of English, and (c) no evidence of referral for speech-language evaluation or documented diagnosis of aphasia or general language impairment. Subcortical stroke was defined as infarcts primarily in subcortical structures (e.g., basal ganglia, internal capsule) in the left hemisphere. Neuroimaging scans of all participants included in this study were read and interpreted by a board-certified radiologist. Age and imaging results (MRI/CT) for each stroke participant are included in Table 1. The normal participants were matched with the stroke participants by age, gender, and educational level.

None of the stroke participants had participated in a speech/language evaluation or had a documented speech/language disorder. All stroke participants were discharged home after their stroke, and none had received specialized rehabilitation services to enhance their recovery. The stroke recovery and caregiving study and this study were reviewed and approved by the institution review board and all participants gave written consent.

Procedure

Each participant in the study completed a Mini-Mental Status Examination (MMSE; Folstein, Folstein, & McHugh, 1975) as a gross measure of cognitive ability. Speech samples for the stroke participants selected for analysis in this study were obtained from qualitative interviews that were completed as part of the larger study. Each stroke participant was interviewed in his home at 1, 6, and 12 months following his stroke. Interviews examined a number of issues related to stroke experience, including but not limited to descriptions of the stroke onset, initiation of treatment, hospitalization and discharge, poststroke impairments, difficulties encountered after discharge, and socialization and adjustment issues. Interviews included descriptions of a typical day in the life of a patient who had a stroke in addition to questions regarding the stroke experience. All interviews were audio recorded. Similar samples were collected from the normal control participants. Interviews for the normal controls were also designed to elicit descriptions of a typical day in their lives.

Five-minute speech samples for this study were extracted from the 1-, 6-, and 12-month qualitative interviews of the 6 stroke participants, for a total of 24 speech samples (18 from the stroke participants and 6 from the controls). The samples included the participants’ responses to interview questions regarding their typical day. Samples from the original audio-recorded interviews were downloaded into a Speech Tools 2.0 Speech Analyzer (2001) to separate the 5-min sample from the entire interview. Each 5-min sample included interviewer questions and prompts. Samples that are 5 min in length are typically adequate to measure behaviors occurring roughly three times per minute (Boles & Bombard, 1998). A comparison sample was collected from each of the 6 control participants. The samples from the controls were limited to a single audio-recorded description of a typical day, for a total of six speech samples.

Transcription and Segmentation

An independent transcriptionist was employed to transcribe the speech samples of all participants. The primary author checked all transcribed speech samples selected for the disfluency analysis for accuracy. If words were deemed unintelligible by the transcriber but understood by the first author, they were corrected. All words that were unintelligible to the primary author were excluded from the analysis.

Sample Length Analysis

To correct for the variability in sample length after extracting interviewer comments, and to limit the impact that length might have on the occurrence of disfluencies, a total word count was obtained for each conversational sample. Interviewer questions and comments were excluded from the word count. Contraction and dialectal variations such as wanna and gonna were counted as two words. Because speech rate can significantly influence fluency levels (Furquim de Andrade, Cervone, & Sassi, 2003), a words per minute (WPM) rate was also calculated for each sample.

Disfluency Analysis

All disfluencies were identified and circled on each transcript. Disfluencies included false starts, revisions, sound repetitions, part-word repetitions, whole-word repetitions, phrase repetitions, and filled pauses (uh, ah). Ideational

<table>
<thead>
<tr>
<th>ID</th>
<th>Age</th>
<th>MMSE</th>
<th>Education</th>
<th>Stroke type/location</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>73</td>
<td>28</td>
<td>12</td>
<td>L. basal ganglia</td>
</tr>
<tr>
<td>2</td>
<td>57</td>
<td>28</td>
<td>14</td>
<td>L. basal ganglia (caudate head)</td>
</tr>
<tr>
<td>3</td>
<td>60</td>
<td>26</td>
<td>14</td>
<td>L. basal ganglia (extension into internal capsule)</td>
</tr>
<tr>
<td>4</td>
<td>54</td>
<td>29</td>
<td>12</td>
<td>L. basal ganglia (extension into corona radiate)</td>
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<tr>
<td>5</td>
<td>52</td>
<td>29</td>
<td>12</td>
<td>L. basal ganglia</td>
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<tr>
<td>6</td>
<td>72</td>
<td>26</td>
<td>12</td>
<td>L. basal ganglia (caudate head; internal capsule)</td>
</tr>
</tbody>
</table>
participants were compared to the speech samples that were collected from the stroke participants at each time point (1, 6, and 12 months). Nonparametric two-group comparison tests (Mann Whitney U) were also completed to compare all variables of interest (total words, WPM, total disfluencies, disfluency rate, and DRPM) between the two groups. Group means for all variables are summarized in Table 2.

### RESULTS

Nonparametric group comparison tests (Mann Whitney U) were completed to compare age and MMSE scores for the two groups. The results did not reveal significant group differences in age between the two groups (Z = –2.08; p = .04) and 12 months (Z = –2.24; p = .03), but not at 6 months (Z = –1.92, p = .07). A Friedman’s test was completed to evaluate the total number of words produced by the stroke participants with time following onset (1, 6, and 12 months) as the within-subjects factor. This test revealed that the stroke participants did not exhibit a significant difference in the mean number of words that they produced across the three points in time (χ² = 2.33, p = .31).

#### Word Production

The control participants produced an average of 546.7 total words per sample compared to the stroke participants, who produced an average of 445.7 words at 1 month, 417.8 words at 6 months, and 375.5 words at 12 months. There were no significant group differences in the average total words produced by the stroke participants at 1 month (Z = –1.21, p = .24), 6 months (Z = 1.12, p = .31), or 12 months (Z = –1.44, p = .18). A Friedman’s test was completed to evaluate the total number of words produced by the stroke participants with time following onset (1, 6, and 12 months) as the within-subjects factor. This test revealed that the stroke participants did not exhibit a significant difference in the mean number of words that they produced across the three points in time (χ² = 2.33, p = .31).

#### Disfluencies

The control participants produced an average of 3.2 disfluencies per sample compared to the stroke participants, who

| Table 2. Values for total words, words per minute (WPM), total disfluencies, disfluency rate, and disfluency rate per minute (DRPM) for the two study groups. |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Variable        | Stroke participants | Controls | Stroke participants | Controls | Stroke participants | Controls | Stroke participants | Controls | Stroke participants | Controls |
| Total words     | M (SD) Range      | M (SD) Range   | M (SD) Range      | M (SD) Range   | M (SD) Range      | M (SD) Range   |
| WPM             | 69.10 (35.30) 46.4–128.4 | 83.60 (49.50) 15.8–130.2 | 75.10 (43.20) 23.6–128.0 | 135.70 (37.80) 82.0–194.9 |
| Total disfluencies | 13.00 (9.50) 1–27 | 11.80 (8.30) 1–26 | 7.80 (7.10) 1–20 | 3.20 (2.60) 0–20 |
| Disfluency rate | .03 (.03 .003–.078) | .05 (.05 .002–.152) | .04 (.07 .003–.169) | .01 (.01 .000–.169) |
| DRPM            | 2.60 (1.90 20–5.40) | 2.40 (1.70 20–5.50) | 1.60 (1.40 20–4.00) | .63 (.51 00–1.20) |

*p < .05 compared to the control group.
produced 13.0 disfluencies at 1 month, 11.8 at 6 months, and 7.8 at 12 months. There were significant group differences in the average number of disfluencies produced by the stroke participants at 6 months \((Z = -2.17, p = .03)\), but not at 1 month \((Z = -1.77, p = .09)\) or 12 months \((Z = -1.46, p = .18)\). A Friedman’s test to evaluate the mean number of disfluencies that were produced by the stroke participants with time following onset as the within-subjects factor revealed that the stroke participants exhibited declining but nonsignificant \((\chi^2 = 2.80, p = .25)\) average numbers of disfluencies from 1 month to 12 months.

**Disfluency Rate**

The disfluency rate for the control participants was .01 compared to a disfluency rate for the stroke participants of .03 at 1 month, .05 at 6 months, and .04 at 12 months. There were significant group differences in the stroke participants’ disfluency rates at 1 month \((Z = -2.18, p = .03)\) and 6 months \((Z = -2.25, p = .03)\), but not at 12 months \((Z = -1.53, p = .13)\). A Friedman’s test to evaluate the disfluency rate for the stroke participants with time following onset as the within-subjects factor revealed that the stroke participants produced a variable disfluency rate across time points. The disfluency rate was not significantly different across the three points in time, \(\chi^2 = 1.73, p = .42\).

**DRPM**

The DRPM for the control participants was .63 compared to a DRPM of 2.6 for the stroke participants at 1 month, 2.4 at 6 months, and 1.6 at 12 months. There were significant group differences in the stroke participants’ DRPM at 6 months \((Z = -2.17, p = .03)\), but not at 1 month \((Z = -1.53, p = .090)\) or 12 months \((Z = -1.46, p = .13)\). A Friedman’s test to evaluate the stroke participants’ DRPM with time following onset \((1, 6, 12\) months) as the within-subjects factor revealed a declining but nonsignificant \((\chi^2 = 2.80, p = .25)\) change across the three time points.

**DISCUSSION**

In this study, our goal was to examine the presence of disfluencies in the discourse of individuals who had a history of subcortical stroke. We measured the presence of disfluencies at three time points in the first year following stroke among participants who were absent a diagnosed speech or language impairment. Our primary findings were that the participants’ total number of disfluencies, disfluency rate, and DRPM were significantly higher than those of the control group at 1 and 6 months while approaching a similar nonsignificant rate at 12 months. In general, the mean total number of disfluencies and DRPM declined across the three time points in the stroke participants’ first year following a stroke. In contrast, the stroke participants’ observed disfluency rate was slightly higher at 6 months than at 1 month following a stroke. However, the 12-month rate was lower than the 6-month rate even though it was slightly higher than the 1-month rate. Finally, the average number of words produced by the stroke participants remained generally constant across the three time points and was not significantly different than that of the matched controls.

The results of this pilot study indicate that some individuals who sustain a subcortical stroke may produce disfluencies at a rate that is more frequent than is typically observed in nonneurologically impaired speakers. Our participants on average produced disfluencies that represented at a minimum 3% of their total output. Their disfluency rate at 1 month (3%) was very similar to the reported average rate of nonneurological populations; however, the stroke participants’ disfluency rate at 6 months and 12 months, and the range of their disfluency rates, suggests significant variability in the level of communication impairment (disfluencies) among the subcortical stroke participants.

The range of disfluency rates suggests a greater deficit level among the subcortical stroke participants than the 3% average actually conveys. The percentage disfluency in this patient population was on average 3%–5% of their total output compared to 1% for the control sample. This value represents a disfluency production rate that is at the cutoff (3%) for classifying individuals with an excessive percentage of disfluent output. We note here that the 3%–5% average was derived from a range of <1% disfluencies to a high of 15%–17% disfluencies for one of our participants at both 6 months and 12 months. These high values suggest an excess disfluent output and the presence of undiagnosed disfluency of neurogenic origin due to subcortical stroke. Interestingly, overall, the participants with a stroke history exhibited a general decline in disfluencies over time in the absence of specialized speech-language interventions.

Reports of disfluencies occurring after a subcortical stroke are not new to the literature (Carluer, Marie, Defer, Coskun, & Rossa, 2000; Ciabarra et al., 2000; Kono, Hirano, Ueda, & Nakajima, 1998; Ludlow, Rosenberg, Salazar, Grafman, & Smutok, 1987). Ciabarra and colleagues examined the disfluency patterns of 3 patients, 2 of whom had a lesion to the basal ganglia. The patients with basal ganglia lesions exhibited initial prolongations of consonants, and repetitions of initial syllables during spontaneous speech. The authors’ explanations for the disfluent output included (a) disruption of intrahemispheric connections due to basal ganglia lesions or (b) disruption of the timing of speech motor control following a subcortical stroke. In summary, multiple mechanisms were likely in play following the complex variety of lesions that can occur in subcortical structures (Ciabarra et al., 2000). Similar patterns have been reported in case reports of individuals with lesions to the basal ganglia (Carluer et al., 2000; Kono et al., 1998; Ludlow et al., 1987) and other subcortical structures such as the thalamus (And & Bhattachar, 1992; Heuer & Sataloff, 1996).

Although there are neuroanatomical explanations for the observed disfluency patterns (Alm, 2004), the purpose of this study was only to describe the presence of disfluencies in the discourse of speakers with a history of subcortical stroke but absent specific speech and language diagnoses. It was not our intent to identify the specific underlying
mechanisms that may have caused the disfluency pattern that was observed in the study participants, particularly given our small sample size and the absence of standardized speech and language assessments. Our goal was primarily to highlight the presence, nature, and natural history of disfluencies in poststroke speakers during the first year of recovery.

Although the general trend observed in these patients who had a stroke suggests declining but persistent disfluency rates across time points (1, 6, 12 months), considerable within-group variability was observed at each time point. Therefore, within-patient variability must be considered when analyzing the observed results (Nespoulous, 2000). The observed changes in rates (increases or decreases) were potentially an artifact of health status (i.e., unrecognized second stroke) or behavioral changes (depression) that were unknown to the authors. Additionally, a degree of task variability may have resulted from the speech samples being collected across time points (Armstrong, 2002; Nespoulous, 2000) or other issues associated with discourse production.

Conclusion

Disfluencies resulting from neurologic causes are by nature a heterogeneous disorder that represents one of many consequences of neurological disease or injury (Helm-Estabrooks, 1993). Therefore, neurogenic disfluencies can occur after a variety of neurological conditions, including stroke, head trauma, extrapyramidal disease, dementia, tumors, and drug use (Shapiro, 1999), and a diagnosis of neurogenic disfluency requires that psychiatric causes are also ruled out (Sapir & Aronson, 1990). Our interest in these data are primarily related to the persistent presence of disfluencies following a stroke and their potential relationship to mild speech or language deficits. Identifying abnormal disfluencies is straightforward, but characterizing the relationship of disfluencies to a specific motor speech or expressive language disorder or determining their functional impact poses greater difficulty. If the disfluencies we observed represent true neurogenic disfluency, a number of questions emerge: (a) What is the incidence of speech disfluency in poststroke individuals who have no history or diagnosis of speech disorder? (b) What is the functional impact of disfluencies on communicative ability? and (c) Do disfluencies consistently influence communicative efficiency in a negative fashion? For some poststroke speakers, low disfluency rates (3–5 DRPM) in some contexts are probably impossible to differentiate from normal disfluency rates. We calculated a disfluency rate and DRPM, including multiple disfluency types, without assumptions regarding the type or cause in order to measure the frequency at which disfluencies occur.

Disfluencies can occur within, between, across, among, before, after, and throughout speech (Cordes & Ingham, 1995). Distinguishing disfluencies due to language formulation deficits (e.g., mild aphasia) from speech-based disfluencies is beyond the scope of this study but should be an emphasis of future studies of poststroke speakers. Standardized definitions, descriptions, and assessments are sorely needed to better understand disfluencies of both types (Cordes & Ingham, 1995). Further studies of disfluencies are also needed to characterize the relationship between speech production errors and communicative effectiveness.

We recognize a number of weaknesses of this study. First, our samples were not originally collected for speech analysis, and standardized measures were not completed. Second, some heterogeneity of the subcortical lesion locations existed among our participants, which may have contributed to our results (Nespoulous, 2000). Disfluency following subcortical damage has been described in the literature, but the recovery pattern is not well understood. The heterogeneity of lesion locations among the participants of this study also precludes a uniform explanation for the observed reductions in fluency. A third issue is the validity of using control comparisons from samples that were collected at one time point as a comparison to multiple time points among the participants with a subcortical stroke. We realize that subsequent study designs derived from theoretical frameworks of mild motor speech deficits and normal speech production variability are required to adequately address these questions. Future studies should also consider the relationship between disfluencies and measures of communicative efficiency and effectiveness.

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APPENDIX A. EXAMPLES OF DISFLUENCIES INCLUDED IN THE ANALYSIS

False Starts
Depends on what you gonna call going in public
I’ve taken her to Publix (grocery store chain)
I stay out in the car while she goes in, [but-----] (delay followed by interviewer presentation of next question)

Revisions
Uh get up uh (name) and my older son
He uh they uh, iron my clothes
Help the little one get in, get his teeth brushed and take a bath and prepare him to go

Sound Repetitions
When I tell me t-[t]-[t]-[t]-[t] down, kept the TV down, that’s the worst part

Word Repetitions
It’s [it’s] comin’ along pretty good
I’ve go the grandsons helpin’ me
And the daughters and my wife
They’re makin’ it
I tell’em what I need and all the things
I show em’ what I gotta do
And then, they’ll [they’ll] do it
So, pretty good kids

Phrase Repetitions
It feel like, [it feel like] your froze in it and I got some puff up in my chest in it
It’s something else

APPENDIX B. EXAMPLES OF DISFLUENCIES EXCLUDED FROM THE ANALYSIS

Interviewer: So let’s talk about how you spend your day.

(Participant #1)
Close round. Close round
Right at home
Right round here in this mission
Yes, right at home
I stays right at home because see don’t nobody but me and her and if I get off too far from the house and get down, she worries about me

(Participant #9) Discussing work; taking a break
It’s not just a break, it’ just that, say like I’m gone let it go and they’ll work it out
I mean like, uh they’ll work it out
I mean like they’ll get, they’ll get it (louder with emphasis)

Interviewer: How are your evenings after work?

(Participant #5)
After work I come home
Most of the time I cook
Like most of the time I cook like two meals
Like if I cook something on Monday, I have something on Tuesday too
There is nothing wrong with leftovers