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Acquired stuttering following right frontal and bilateral pontine lesion: A case study

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Abstract

We report neurological information and experimental data regarding acquired neurogenic stuttering in a 57-year-old male following ischemic lesion to the orbital surface of the right frontal lobe and the pons. The experimental data consist of stuttering frequency measures under various conditions that are well known to enhance fluency in most individuals with developmental stuttering. Specifically, we report data for adaptation, unison reading, delayed auditory feedback (DAF), and frequency altered feedback (FAF). This work is the first published report of such a comprehensive examination of a variety of fluency-enhancing conditions in acquired stuttering. The patient read six 200-word texts under different conditions: Six solo readings (Text 1), five unison readings followed by five solo readings (Text 2), five readings with non-altered auditory feedback (Text 3), five readings with 50 ms delayed auditory feedback (Text 4), five readings with increased FAF (Text 5), and five readings with decreased FAF (Text 6). Results indicate that, unlike the typical situation for developmental stuttering, this individual with acquired neurogenic stuttering did not show increased fluency during an adaptation paradigm or under unison, DAF, and FAF conditions. We discuss possible implications of these findings and emphasize the need for further research on acquired neurogenic stuttering.

1. Introduction

Recently, sophisticated experimental methods have been applied (e.g., Salmelin, Schnitzler, Schmitz, & Freund, 2000) to examine some hypotheses regarding the neural bases of developmental stuttering that had been formulated several decades ago (Orton, 1927; Travis, 1931). Unlike the situation for developmental stuttering, the neural bases of acquired neurogenic stuttering are matter of certainty. The literature contains several case reports on acquired neurogenic stuttering following heterogeneous lesion sites in the left hemisphere (Orton, 1927; Canter, 1971; Carluer, Lambert, Defer, Coskun, & Rossa, 2000), the right hemisphere (Ardila & Lopez, 1986; Fleet & Heilman, 1985; Horner & Massey, 1983; Lebrun & Leleux, 1985; Soroker, Bar-Israel, Schechter, & Solzi, 1990), or both hemispheres (Balasubramanian & Hayden, 1996; Helm-Estabrooks, 1993). Although lesions of the network of brain structures supporting motor speech can lead to a variety of speech disorders, including acquired neurogenic stuttering, cases have also been reported in which neurological lesions led to the disappearance or reduction of stuttering (Andy & Bhatnagar, 1992) or to the re-emergence of childhood stuttering from which the person had recovered in early years (Helm-Estabrooks, Yeo, Geschwind, Freedman, & Weinstein, 1986; Mouradian, Paslawski, & Shauib, 2000).

Several investigators have observed striking similarities in symptomatology between developmental and acquired neurogenic stuttering. Van Borsel and Taillieu (2001) reported that even professionals were unable to differentiate between these two disorders on the basis of conversational speech samples. Van Borsel (1997) further pointed out that, based on such similarities, one may be tempted to conclude that developmental stuttering is also due to neurogenic dysfunction. In addition, some have argued that acquired stuttering may be influenced by the same variables that have been documented to have an influence on the symptoms of developmental stuttering. However, studies examining the role of such variables in acquired neurogenic stuttering have yielded inconsistent results (Van Borsel & Taillieu, 2001; Ringo & Dietrich, 1995). For example, most studies have reported an absence of the adaptation effect (i.e., decreased dysfluency on successive readings of the same text) in acquired neurogenic stuttering. However, the method of measuring adaptation in these studies was often unclear (Ringo & Dietrich, 1995). Similarly, one cannot generalize the available data regarding the effects of delayed auditory feedback (DAF) on acquired stuttering. DAF appears to have been applied only to four cases associated with traumatic brain injury and degenerative diseases (Van Borsel, 1997). Replicating such work, and extending it to other fluency enhancing conditions such as frequency altered feedback (FAF), may provide additional information regarding differences and similarities between developmental and acquired stuttering. One group of investigators has reported FAF to be very effective in reducing stuttering for most cases of developmental stuttering (Stuart, Kalinowski, Armson, Stenstrom, & Jones, 1996) although others have obtained less consistent findings (Ingham, Moglia, Frank, Ingham, & Cordes, 1997). To the best of our knowledge, FAF has not been investigated with acquired neurogenic stuttering.

The present work reports a case of acquired neurogenic stuttering following ischemic lesion of the right orbital frontal surface and bilateral attenuation of the pons. In addition to the neurological background, we present stuttering frequency data collected in each of the aforementioned fluency-enhancing conditions (i.e., adaptation, unison reading, DAF, and FAF).

2. Method

2.1. Subject

CP, a 57-year-old male with a medical history of noninsulin dependent hyperglycemia, hypertension, and ischemic disease, had a cerebro-vascular accident on 12-4-2000. A CT scan evaluation on 12-13-2000 revealed a new subcortical ischemia involving the right hemisphere and bilateral hypodensity of the pons (see Fig. 1). The subject was evaluated by a speech–language pathologist and was found to have dysphagia and occasional deficits in naming. Since onset of the CVA, CP's speech had reportedly become non-fluent. Family members reported that the symptoms resembled those of the childhood stuttering that the patient had shown, but outgrown, at a young age. CP and his brother claimed

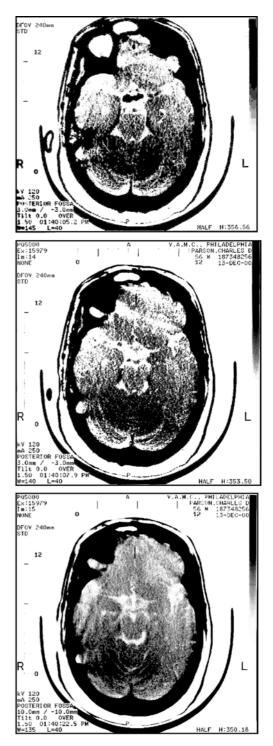


Fig. 1. CP's CT scans showing right hemisphere orbital surface lesion.

that CP had been free from symptoms of stuttering for well over four decades.

During oral reading of a 200-word passage, CP stuttered on 5% of the words. For this evaluation as well as all experimental conditions, stuttering was defined as part-word repetitions, audible and inaudible prolongations, broken words, and monosyllabic whole word repetitions.

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2.2. Materials

The present study used six different 200-word texts. These texts were chosen from current issues of Readers' Digest to have neutral content, and they were used for different oral reading tasks. Instrumentation involved in this study included a Korg digital signal processor used for FAF (Pandora 28086), a Kay Elemetrics Facilitator (Model 3500) used for DAF, a digital minidisk audio recorder (Sharp MD-MS722), and a digital video camcorder (JVC GR-DV2000).

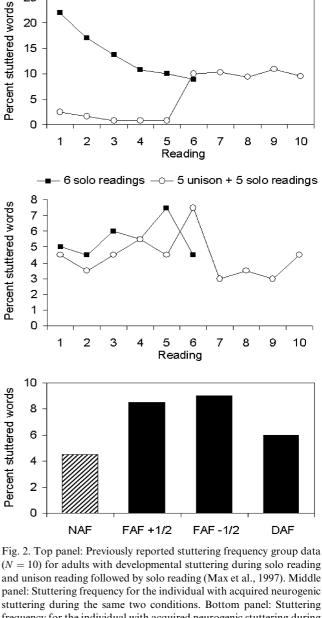
2.3. Procedures

CP was seen twice, with a one-month interval between the sessions. Each session lasted approximately one hour. In the first session, CP was tested in an adaptation/unison reading paradigm previously described by Max, Caruso, and Vandavenne (1997). This paradigm included an adaptation condition with six solo readings of a 200-word passage (Text 1) and a chorus reading condition in which the subject read another 200word passage five times in unison with a speech-language pathologist and then five times solo (Text 2).

In the second session, CP was tested using altered auditory feedback conditions. A non-altered auditory feedback condition served as a control condition. In this control condition, CP was wearing headphones while reading a 200-word passage (Text 3) but his speech was fed back to his ears without any modification. It was followed by a DAF condition during which CP's speech was fed back to his ears through the headphones with a delay of 50 ms. During this task, CP read another 200word passage (Text 4). For an increased FAF condition, CP's speech was altered by increasing the frequencies of his speech spectrum by 1/2 octave prior to feeding the speech signal back to his ears through the headphones. Another 200-word passage (Text 5) was read in this condition. For a decreased FAF condition, CP's speech was altered by decreasing the frequencies in the acoustic signal by 1/2 an octave before it was fed back to his ears through the headphones. As with all other conditions, CP read a different 200-word passage (Text 6). For each reading in each condition, both audio and video signals were recorded.

3. Results and discussion

All adaptation/unison and altered auditory feedback results for CP are shown graphically in the middle and bottom panels, respectively, of Fig. 2. For comparison, the top panel of the figure also shows the stuttering frequency group data for adults with developmental stuttering during solo reading and unison reading followed by solo reading as previously reported by Max et al. (1997).



----- 6 solo readings ----- 5 unison + 5 solo readings

and unison reading followed by solo reading (Max et al., 1997). Middle panel: Stuttering frequency for the individual with acquired neurogenic stuttering during the same two conditions. Bottom panel: Stuttering frequency for the individual with acquired neurogenic stuttering during reading under normal auditory feedback (NAF), 1/2 octave up frequency-altered auditory feedback (FAF +1/2), 1/2 octave down frequency-altered auditory feedback (FAF -1/2), and 50 ms delayed auditory feedback (DAF).

As shown in the middle panel of Fig. 2, CP showed no evidence of an adaptation effect during six repeated solo readings. Moreover, it can be seen that unison reading, a strong fluency-enhancing condition for individuals with developmental stuttering, had no beneficial effects on the stuttering frequency of CP. As shown in the bottom panel of Fig. 2, DAF and FAF also failed to enhance fluency in CP's oral reading. In fact, in all three altered feedback conditions, CP's stuttering frequency was slightly higher than that observed during the control condition.

Clearly, the results for CP, an adult with acquired neurogenic stuttering, are inconsistent with those reported by Max et al. (1997) for adults with developmental stuttering. Comparing the lack of adaptation in CP's solo reading with the presence of such an effect in most cases of developmental stuttering, it may be the case that individuals with developmental stuttering but not individuals with acquired neurogenic stuttering benefit from repeated practice (i.e., resulting in motor learning) of a limited set of speech utterances. This may suggest that the stuttering symptoms seen in acquired neurogenic and developmental stuttering are not based on the same neural dysfunction (Van Borsel, 1997). In particular, this finding is in keeping with the hypothesis that developmental stuttering may involve, at least in part, a problem with the process of developing and refining internal models for speech motor control (i.e., the neural mapping between motor commands and the sensory consequences of those commands) during speech development (Max, in press). It would seem reasonable to speculate, then, that such a problem would allow individuals with developmental stuttering to obtain some benefits from repeated motor practice. In acquired neurogenic stuttering, on the other hand, the dysfluencies are the result of an actual lesion and shortterm practice such as provided during an adaptation paradigm may be insufficient to result in improved speech fluency.

The lack of enhanced fluency under DAF and FAF conditions also suggests that the mechanisms underlying CP's non-fluent speech are different from those involved in individuals with developmental stuttering given that the speech of the latter group typically becomes more fluent under these conditions. Most certainly, CP's lesion in the right hemisphere orbital surface does not suggest a causal role for the auditory system in this case. However, the effects of DAF and FAF need to be further explored in acquired neurogenic stuttering to replicate the present findings. Indeed, individuals with developmental stuttering also vary in terms of which delay and frequency shifts are most optimal, and they do not respond uniformly to FAF conditions (Ingham et al., 1997). Hence, other DAF durations and FAF frequency shifts should be investigated in future studies on acquired stuttering. There is clearly a need for further study in this regard.

Interestingly, the role of right hemisphere in speech production has become increasingly clear in recent research (Goulet, Joanette, & Van Dongen, 1997; Kurowski, Blumstein, & Mathison, 1998). The present work documents a case of acquired neurogenic stuttering following lesions in both the right hemisphere and the pons. Of course, this case study and the accompanying experimental data do not allow us to attribute the patient's stuttering specifically to either of these two lesions. In order to gain a better understanding of the differences and similarities between developmental and acquired stuttering, and the neurological mechanisms underlying each of these two disorders, more cases with a variety of etiologies/sites of lesion resulting in acquired neurogenic stuttering need to be studied using a large number of tasks. We hope that the tasks and analyses used in the present study will form a first step in this direction.

References

- Andy, J. O., & Bhatnagar, S. C. (1992). Stuttering acquired from subcortical pathologies and its alleviation from thalamic perturbation. *Brain and Language*, 42, 385–401.
- Ardila, A., & Lopez, M. V. (1986). Severe stuttering associated with right hemisphere lesion. *Brain and Language*, 27, 239–246.
- Balasubramanian, V., Hayden, P. A., 1996. Acquired stuttering following bilateral parietal lobe lesion: A case report. In: C. W. Starkweather & H. F. M. Peters (Eds.), *Proceedings of the first* world congress of fluency disorders (pp. 617–620). Nijmegen: University Press, The International Fluency Association.
- Canter, G. (1971). Observations on neurogenic stuttering: A contribution to differential diagnosis. *British Journal of Disorders of Communication*, 6, 139–143.
- Carluer, L., Lambert, J., Defer, G. L., Coskun, O., & Rossa, Y. (2000). Acquired and persistent stuttering as the main symptom of striatal infarction. *Movement Disorder*, 15, 343–346.
- Fleet, W. S., & Heilman, K. M. (1985). Acquired stuttering from a right hemisphere lesion in a right hander. *Neurology*, 35, 1343– 1346.
- Goulet, P., Joanette, Y., & Van Dongen, H. (1997). Word fluency after right hemisphere lesion. *Neuropsychologia*, 35, 1565–1570.
- Helm-Estabrooks, N. (1993). Stuttering associated with acquired neurological disorders. In: R. Curlee (Ed.), *Stuttering and related disorders of fluency*. New York: Thieme Medical Publishers.
- Helm-Estabrooks, N., Yeo, R., Geschwind, N., Freedman, M., & Weinstein, C. (1986). Stuttering: Disappearance and reappearance with acquired brain lesions. *Neurology*, 36, 1109–1112.
- Horner, J., & Massey, E. W. (1983). Progressive dysfluency associated with right hemisphere disease. *Brain and Language*, 18, 71–85.
- Ingham, R. J., Moglia, R. A., Frank, P., Ingham, J. C., & Cordes, A. K. (1997). Experimental investigation of the effects of frequencyaltered feedback on speech of adults who stutter. *Journal of Speech, Language, and Hearing Research*, 40, 361–372.
- Kurowski, K. M., Blumstein, S. E., & Mathison, H. (1998). Consonant and vowel production of right hemisphere patients. *Brain and Language*, 63, 276–300.
- Lebrun, Y., & Leleux, C. (1985). Acquired stuttering following right brain damage in dextrals. *Journal of Fluency Disorders*, 10, 137– 141.
- Max, L. (in press). Stuttering as a neuromotor disorder: Identifying the mechanisms. In: B. Maassen, R. Kent, H. F. M. Peters, P. Van Lieshout & W. Hulstijn (Eds.), Speech motor control in normal and disordered speech. Oxford University Press.
- Max, L., Caruso, A. J., & Vandavenne, A. (1997). Decreased stuttering frequency during repeated readings: A motor learning perspective. *Journal of Fluency Disorders*, 22, 1–17.
- Mouradian, M. S., Paslawski, T., & Shauib, A. (2000). Return of stuttering after stroke. *Brain and Language*, 73, 120–123.
- Orton, S. T. (1927). Studies in stuttering: Introduction. Archives of Neurology and Psychiatry, 18, 671–672.
- Ringo, C. C., & Dietrich, S. (1995). Neurogenic stuttering: An analysis and critique. *Journal of Medical Speech Language Pathology*, 3, 111–122.

- Salmelin, R., Schnitzler, A., Schmitz, F., & Freund, H. J. (2000). Single word reading in developmental stutterers and fluent speakers. *Brain*, 123, 1184–1202.
- Soroker, N., Bar-Israel, Y., Schechter, I., & Solzi, P. (1990). Stuttering as a manifestation of right hemisphere subcortical stroke. *European Neurology*, 30, 268–270.
- Stuart, A., Kalinowski, J., Armson, J., Stenstrom, R., & Jones, K. (1996). Fluency effect of frequency alterations of plus/minus onehalf and one-quarter octave shifts in auditory feedback of people

who stutter. Journal of Speech and Hearing Research, 39, 396-401.

- Travis, L. E. (1931). *Speech pathology*. Englewood Cliffs, NJ: Prentice-Hall.
- Van Borsel, J., & Taillieu, C. (2001). Neurogenic stuttering versus developmental stuttering: An observer judgment study. *Journal of Communication Disorders*, 34, 1–11.
- Van Borsel, J. (1997). Neurogenic stuttering: A review. Journal of Clinical Speech and Language Studies, 7, 16–33.