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Effectiveness of Speech Therapy in Neurogenic Stuttering: A Case Study

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Abstract

Introduction: Stuttering associated with acquired neurological disorders is an acquired disorder of fluency characterized by notable, involuntary repetitions or prolongations of speech that are not the result of language formulation or psychiatric problems.” Neurogenic stuttering has been reported following various lesions or degenerative disease conditions and as such does not appear to be associated exclusively with a particular neurological disorder or disruption in any particular brain area.

Aim: To compile the dysfluency characteristics of a subject with neurogenic stuttering and to study the efficacy of speech therapy.

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Method: A 40yrs old male came to the department with the complaint of recent onset of dysfluent speech, after his head hit against the wooden plank. Detailed fluency assessment revealed that he has increased rate of speech, repetition was observed in both content and in function words, filled and unfilled pause was seen, prolongations and some secondary behaviors (rapid blinking) were present. Presence of stuttering on non-initial syllables was noticed in his speech. There was no adaptation effect, situational variability, individual variability and sound or word fear observed. The stuttering severity instrument Riley (1972) was administered and the result revealed moderate degree of stuttering. The therapy was given for 14 sessions.

Result: After 14 sessions SSI was re-administered and the result revealed mild stuttering. During the course of therapy, rate of speech was reduced, fluency and speech intelligibility were improved.

Conclusions: This case study highlights the salient features of neurogenic stuttering which are most commonly mentioned in the literature. This case study also throws light on favorable outcomes of therapy. It also helps in differential diagnosis among neurogenic stuttering and developmental stuttering.

Introduction

Fluent speaking is, perhaps, the most refined motor act performed by humans requiring complex coordination of many different muscle groups. It can be sensitive to even small changes in neurological status, which may be why stuttering occurs in a wide range of neurological disorders from Parkinson's disease to close head injury (Helm Estabrooks 1999). Stuttering is classified as a communication disorder. It has been shown to be associated with increased incidence of mental health problems and lowered quality of life (Craig, Blumgart, & Tran, 2009).

Stuttering

According to the World Health Organization (WHO), stuttering consists of “disorders in the rhythm of speech in which the individual knows precisely what he wishes to say but at the time is unable to say it because of an involuntary, repetitive prolongation or cessation of a sound” (WHO, 1977, p. 202). The term neurogenic acquired stuttering denotes stuttering that appears to be caused by neurological disease or damage. It is typically acquired after childhood and its etiology may be stroke, head trauma, tumor, and disease process such as Parkinson's or drug toxicity.

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According to Helm-Estabrooks (1999) “Stuttering associated with acquired neurological disorders is an acquired disorder of fluency characterized by notable, involuntary repetitions or prolongations of speech that are not the result of language formulation or psychiatric problems.” Canter, (1971) suggested that neurogenic stuttering comprises 3 sub groups. One sub group is dysarthric stuttering which is seen in Parkinson’s disease or have a cerebellar lesion in which stuttering appears to emerge from lack of muscle control. The second sub group is apraxic stuttering in which stuttering may arise due to problem in motor planning. Both silent blocks and repetitions occur as the speaker struggle to sequence the appropriate speech movements. The third sub group is Dysnomic stuttering, which usually accompanies aphasia in which, the individual searches for a word he is having trouble to retrieve.

Causes for Stuttering

Acquired stuttering has various causes. Its different varieties can be psychogenic, or can arise from drug addiction or other causes. However, the most common cause of acquired stuttering is brain lesion (Helm-Estabrooks et al., 1986). Neurogenic stuttering has been reported following various lesions or degenerative disease conditions and as such does not appear to be associated exclusively with a particular neurological disorder or disruption in any particular brain area. In a survey study of hundred cases of patients with neurogenic stuttering, Market et al. (1990) reported that 38.3% started to stutter following head trauma and 37% following ischemic accidents, for a total of 75.3% of all patients analyzed in their study. The remainder of their participants acquired stuttering following a variety of disease and trauma conditions, including neurodegenerative disease (1.2%).

Steward and Rowley (1996) reported that 69.2% of the subjects in their sample had a neurogenic origin, which presumably combine stroke, trauma and neurodegenerative diseases. No further differentiation was made within this group. In the case of neurogenic stuttering following stroke several brain sites have been implicated, including sub-cortical regions such as thalamus and brain stem (Abe, Yokoyama, and Yorifugi, 1993; Van Borsel and Taillieu, 2000), basal ganglia (Nass et al., 1994), and cerebellum (Van Borsel and Taillieu, 2000), as well as cortical regions including temporal and parietal lobe (Ardila and Lopez 1986; Bijleveld, Lebrun,

and Dongen, 1994; Helm- Estabrooks et al., 1986), Supplementary motor area (Van Borsel, et al., 1997), Frontal cortex (Van Borsel and Taillieu, 2000), similar to findings by Ludlow et al.(1987) in trauma patients neurogenic stuttering following stroke can occur with either unilateral (left or right) or bilateral lesions.

Although much of the literature on neurogenic stuttering consists of single case studies (Example Billeveld, Lebrun & Vandongen, 1994), there have been several attempts by clinician researchers to summarize their findings on multiple cases and thereby develop a clearer picture of the disorder. Dietrich (1995), Helm-Estabrooks (1999), and Ludlow and Loucks (2003) listed general characteristics of neurogenic stuttering as follows.

1. Dominant characteristics are syllable and sound repetition, blocks are less frequent.
2. Dysfluencies occur on grammatical words nearly as frequently as on substantive words.
3. The speaker might be annoyed, but does not appear anxious.
4. Repetitions, prolongations, and blocks do not occur only on initial syllables of words and utterances.
5. Accessory symptoms such as facial grimacing, eye blinking, and fist clenching are not associated with moments of dysfluency.
6. No adaptation effect exists.
7. Stuttering occurs consistently across speech tasks of various types.
8. Patients tend to show additional signs of aphasia and dysarthria.

Neurogenic Stuttering vs. Developmental Stuttering

It has been suggested that neurogenic stuttering can be differentiated from developmental stuttering by the findings that neurogenic stuttering do not become fluent with rhythmic speech, masking or speaking slowly, paradoxically these conditions may be therapeutically useful for some patients (Guitar, 2006). Pacing is essentially a technique of speaking one syllable at a time, so that each syllable is spoken separately, without the usual co articulation across syllables. As a result the speech is produced more slowly and with a regular, staccato rhythm. This treatment was developed by Helm (1979) for patient with palalalia but has been used for neurogenic

stuttering as well. Rentschler et al (1984), Marshall and Starch (1984), and Helm Estabrooks (1999) reported that masking and DAF can be used as therapeutic tool to induce fluency, and in some cases, the fluency can be generalized. Market et al (1990) conducted a survey of clinicians who had worked with acquired stuttering and found that many of them reported success with fluency- shaping tools such as slow rate and easy onset. Helm Estabrooks (1986) and Rubow, Rosenbek and Schumaker (1986) suggested that training patients to relax muscles with the help of biofeedback can be effective in reducing Neurogenic stuttering. As there is a dearth of study profiling the characteristics of dysfluency as well as its therapeutic effectiveness in Indian scenario, an attempt has been made to compile the dysfluency characteristics of a subject with neurogenic stuttering and also to study the effectiveness of speech therapy for a subject with neurogenic stuttering.

Aim of This Study

1. To compile the dysfluency characteristics of a subject with neurogenic stuttering
2. To study the efficacy of speech therapy.

Method

A 40yrs old male came to the department with the complaint of recent onset of dysfluent speech, after his head hit against the wooden plank. The subject reported that there was loss of consciousness and blurring of vision at the time of head injury and after which the speech became dysfluent. Detailed fluency assessment revealed that he has increased rate of speech, repetition was observed in both content and in function words, filled and unfilled pause was seen, prolongations and some secondary behaviors (rapid blinking) were present. Presence of stuttering on non-initial syllables was noticed in his speech. There was no adaption effect, situational variability, individual variability and sound or word fear observed. The stuttering severity instrument Riley (1972) was administered and the result revealed moderate degree of stuttering. Neurologic evaluation revealed no motor sensory deficits and weakness. CT scan report revealed small, Focal, irregular hypodense lesion including the right medial temporal lobe. By considering the above characteristics, the case was diagnosed as neurogenic stuttering. The

therapy was given for 14 sessions. The goals taken for the therapy was to reduce the rate of speech and to decrease the dysfluency.

The following behavioral treatments were used to achieve the targeted goals.

1. Pacing: This treatment was developed by Helm(1979).In this the subject was instructed to speak one syllable at a time, so that each syllable was spoken separately, without the usual coarticulation across syllables. As a result speech was produced more slowly with a regular, staccato rhythm. A pacing board was used for this purpose.
2. Easy Onsets - first sound needs to be very gentle. 'Sigh' the first word
3. Precision fluency shaping program: In this program, the skills to be achieved were stretched syllables, smooth transition between syllables, slow change within syllable, diaphragmatic breathing and gentle onset. These skills were to be practiced first in simple syllables and then in longer words and phrases.
4. Airflow therapy: in this program the case was trained to control a wide range of aspects of airflow. These aspects involve smooth breathing, exhalation prior to speech, blending words into exhalation pattern, continuing exhalation until the last utterance, pausing at natural juncturing points, smooth inhalation during the pre speech pause.
5. Soft Contacts -tongue, lips etc need to make soft touches. 'Delicate' mouth
6. Continual Phonation -one breath for each sentence/idea, pausing at the right times

Overall Improvement

The client attended the therapy regularly. The progress observed in the case was as follows

- The rate of speech was reduced.
- Till 7th sessions of therapy, repetition of words and filled pauses were not reduced.
- From 8th to 10th session, the repetition of words and filled pauses were occasionally present.
- From 11th session onwards, none of the dysfluencies were present.
- The overall speech intelligibility improved.

- On 14th session, SSI was re-administered. Scores were below mild severity (less than 10). During the course of therapy, rate of speech was reduced, fluency and speech intelligibility were improved.

Discussion

Stuttering associated with acquired neurological disorders is an acquired disorder of fluency characterized by notable, involuntary repetitions or prolongations of speech that are not the result of language formulation or psychiatric problem. (Helm-Estabrooks, 1999). The nature of onset for neurogenic stuttering is usually sudden and the most common cause of acquired stuttering is brain lesion (Helm-Estabrooks et al., 1986). The present case also exhibited a sudden onset of dysfluency followed by head trauma and the lesion was found to be a Focal, irregular hypodense lesion including the right medial temporal lobe. Similar result was reported by (Van Borsel and Tallieu 2000) who reported onset of dysfluency followed by lesion in the cortical region such as temporal and parietal lobe.

The present case exhibited irregular and rapid rate of repetitions on function words as well as content words which are the typical characteristics of neurogenic stuttering as reported by Canter (1971). In neurogenic stuttering, the stuttering is not restricted to initial syllables in words (Bloodstein, 1995). The present case also had a similar trend in which the dysfluency was observed in non-initial syllables. The case also exhibited absence of adaption effect, situational variability, individual variability, sound or word fears. Bloodstein (1995); Van Riper (1982) reported individual with neurogenic stuttering has no adaptation effect and fear and anxiety.

The present case attended the therapy program for 14 sessions. An individualized rehabilitation program was planned which included the following therapy techniques - pacing, easy onset, precision fluency shaping program, Delayed Auditory Feedback, Airflow therapy, Soft contact and continual phonation. Post therapy Perceptual evaluation revealed a marked improvement in his fluency. SSI scores also revealed a remarkable improvement in his fluency. Market et al (1990) conducted a survey of clinicians who had worked with acquired stuttering

and found that many of them reported success with fluency- shaping tools such as slow rate and easy onset.

Conclusion

Neurogenic stuttering, although relatively rare, is increasing a well recognized disorder in the clinical case load of speech language pathologist. Much of the information about neurogenic stuttering, however, is still based on case studies of single patient. This case study highlights the salient features of neurogenic stuttering which are most commonly mentioned in the literature. The features of neurogenic stuttering exhibited by the present case were absence of Adaption effect, situation variability, and individual variability and sudden onset of stuttering. Features like repetition and pauses were noticed. In addition other characteristics like tension and struggle reaction were seen and restricted tongue movement was also present. This case study also throws light on favorable outcomes of therapy. It also helps in differential diagnosis among neurogenic stuttering and developmental stuttering.

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