Stuttering following acquired brain damage: A review of the literature

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Abstract:

Communication problems resulting from acquired brain damage are most frequently manifested as motor speech disorders such as dysarthria, syndromes of aphasia, and impairments of pragmatics. A much less common phenomenon is the onset of stuttering in adults who sustain a stroke, traumatic brain injury, or other neurologic events. When stuttering occurs in association with neuropathology, precise characterization and explanation of observed behaviors is often difficult. Among the clinical challenges presented by acquired stuttering are the problem of distinguishing this form of dysfluency from those associated with dysarthria and aphasia, and identifying the neuropathological condition(s) and brain lesion site(s) giving rise to this speech disorder. Another challenge to the precise characterization of acquired stuttering is the fact that some cases of acquired stuttering apparently have a psychological or neuropsychiatric genesis rather than a neuropathological one. In this paper we provide a review of the literature pertaining to the complicated phenomenon of acquired stuttering in adults and draw some tentative explanatory conclusions regarding this disorder.

Keywords: Neurogenic stuttering | Stuttering | Traumatic brain injury | Aphasia | Acquired stuttering

Article:

1. Introduction

Despite the fact that adult onset of stuttering behavior in previously fluent speakers has been discussed in the literature for more than 100 years, it remains unclear whether acquired stuttering is a distinct disorder or an epiphenomenon of other motor speech disorders such as apraxia of speech. Furthermore, conclusions regarding the nature of the underlying mechanisms of acquired stuttering remain speculative. A not insignificant factor contributing to the uncertainty surrounding acquired stuttering is the range of neurologic conditions that have been correlated with the syndrome: it has been reported in cases of single lesion strokes (e.g., Helm-Estabrooks, Yeo, Geschwind, Freedman, & Weinstein, 1986), multiple lesion strokes (e.g., Grant et al., 1999 and Helm et al., 1978), traumatic brain injury (TBI) (e.g., Helm-Estabrooks and Hotz, 1998, Lebrun et al., 1990 and Ludlow et al., 1987), seizure disorder (e.g., Lebrun, 1991 and Sechi et al., 2006), Parkinson's syndrome (Canter, 1971 and Koller, 1983), dialysis dementia (Rosenbek, McNeil, Lemme, Prescott, & Alfrey, 1978), and senile dementia (Quinn & Andrews, 1977). Additionally, various pharmacological agents have been reported to cause stuttering in individuals with no previous history of the disorder (see Brady, 1998 for review). In most of these cases, normal speech patterns returned after the drug was discontinued. A curious case of transient acquired stuttering in a young man with severe, chronic anorexia nervosa resulting in a significant hypoglycemic state was reported by Byrne, Byrne, & Zibin, 1993. All-in-all, given the many conditions that might give rise to acquired stuttering, one might well ask why it occurs so infrequently that it is deemed a phenomenon worthy of formal report. Indeed, the relatively rarity of acquired stuttering, itself, is in need of explanation.

Equally challenging with regard to acquired stuttering is the characterization of the speech output disorder. Few case reports provide comprehensive descriptions of the speech patterns that are labeled as “stuttering.” An exception is the report by Jokel, De Nil, and Sharpe (2007) who systematically assessed the speech characteristics of 12 individuals with neurogenic stuttering secondary to either TBI or stroke. These investigators assessed their cases in relation to the six principal characteristics of neurogenic stuttering often referred to in the neurogenic stuttering literature (see for example, Helm-Estabrooks, 1999,Ringo and Dietrich, 1995 and Rosenbek et al., 1978). These characteristics, which are generally understood as distinguishing acute onset, neurogenic stuttering from developmental stuttering, are as follows.

Six Features of Neurogenic Stuttering

1. Dysfluencies occur on grammatical words at a similar rate of occurrence as substantive words,

2. Repetitions, prolongations, and blocks occur in all positions of words,

3. There is a consistency in stuttering behavior across speech tasks.

4. The speaker does not appear overly anxious about the stuttering behavior,

5. Secondary symptoms such as facial grimacing, fist clenching, and eye blinking are rarely observed,

6. An adaptation effect is not observed,

After examining their 12 cases vis a vis the extent to which their behaviors did or did not conform to these six oft-cited features of acquired neurogenic stuttering, Jokel et al. (2007) concluded that their cases did not comprise a homogeneous group. Van Borsel and Taillieu (2001) presented taped speech samples of acquired and developmental stuttering" to a panel of experienced professionals who regularly treated individuals with fluency disorders. The raters were asked to judge the severity of the dysfluency disorder, decide whether the disorder was consistent with the diagnosis of stuttering, and finally determine whether the disorder was neurogenic or developmental. Raters misidentified individuals with acquired stuttering as having developmental stuttering as often as they correctly identified individuals with developmental stuttering as having that form of dysfluency.

It would appear that sufficient level of uncertainty has been raised about the validity of the "six features of neurogenic stuttering" that they should be collectively regarded as a "rule of thumb" rather than pathognomonic indicators of neurogenic stuttering. In other words, if an individual with a history of a documented neurologic event shows all six behaviors associated with neurogenic, acquired stuttering, then that diagnosis can be made with greater certainty than If the individual does not display all these behaviors.

In this paper we review the literature related to neurogenic stuttering, discuss the complicated task of differential diagnosis and explore possible neuropathological correlates.

2. Differential diagnosis of acquired neurogenic stuttering

2.1. Acquired stuttering vis-à-vis aphasia and motor speech disorder

A review of the published cases of acquired neurogenic stuttering shows that many cases can be grouped into two categories, i.e., those whose stuttering was associated with aphasia and those whose stuttering was associated with a motor speech condition. Most of the cases have involved a relatively young group of individuals who developed dysfluent speech following a stroke or traumatic brain injury.

Luchsinger and Arnold (1965) described neurogenic stuttering as being either a core aspect of aphasia or as a psychological reaction to having aphasia. Indeed, stuttering or stuttering-like behaviors have often been described as an integral component of aphasia syndromes (Lebrun, Leleux, Rousseau, & Devreux, 1983). Among the types of aphasia associated with stuttering-like dysfluencies are amnestic aphasia (Arend, Handzel, & Wiess, 1962), Broca's aphasia (Trost, 1971), conduction aphasia (Farmer, 1975) and Wernicke's aphasia (Helm et al., 1978). Some of the dysfluencies manifested with aphasia may be the results of word retrieval or production

problems. In his description of conduction aphasia, for example, Goodglass (1993) pointed out that patients with this form of aphasia sometimes struggle with phonemic paraphasias that “may appear as stutter-like blocking” (p. 211) as the patients make repeated attempts to self-correct. It is important to note, however, that a significant number of cases of neurogenic stuttering have shown no signs of aphasia. Thus, while acquired dysfluencies may sometimes occur in association with aphasia, the two phenomena can and do exist independently.

Stuttering-like dysfluencies are sometimes a component of motor speech disorders. For example, apraxia of speech (“verbal apraxia”) is often associated with repetition of phonemes in a manner suggestive of neurogenic stuttering (e.g., Johns and Darley, 1970, Rosenbek et al., 1978 and Trost, 1971). Koller (1983) reported six cases of stuttering associated with extrapyramidal diseases such as Parkinson's disease. The speech dysfluencies of these individuals could be differentiated from both developmental dysfluency and the acquired dysfluencies observed secondary to stroke or traumatic brain injury. According to Koller, the speech dysfluencies resulting from extrapyramidal diseases were most frequently observed in spontaneous speech production. These dysfluencies included the repetition and prolongation of initial syllables of small grammatical and substantive words, and a positive adaptation effect. Another form of dysfluency associated with extrapyramidal diseases is palilalia, so it is important to distinguish between palilalia and stuttering. Stuttering is characterized as involuntary repetitions, prolongations or cessations of sounds produced at a relatively steady rate. In contrast, palilalia is manifested as whole word, phrase, or sentence repetitions that often are produced with increasing rapidity and decreasing distinctness, a phenomenon called “speech festination.” Thus, stuttering occurs at the phoneme level without changes in rate and articulatory precision while palilalia occurs at the word/phrase/sentence level with festination.

2.2. Psychiatric considerations for acquired neurogenic stuttering

It has been reported that stuttering may occur in adulthood as a result of psychological trauma; a phenomenon referred to as “hysterical” stuttering by Bluemel (1935) and Freud (1966). Deal (1982) described eight speech characteristics associated with stuttering as a reaction to a psychological trauma. As can be seen, some of these characteristics overlap with those associated with neurogenic stuttering.

Deal's Eight Features of Psychogenic Stuttering

1. Sudden onset of stuttering behavior,
2. Onset related to a significant event,
3. Preponderance of repetition of initial or stressed syllables,
4. No apparent adaptation effect,
5. No apparent patterns of fluency,

6. No secondary symptoms,

7. No apparent concern about the stuttering behavior,

8. The pattern of dysfluency is similar in conversational speech and oral reading.

Other features of acquired psychogenic stuttering were provided by Roth, Anderson, and Davis (1989) who described 12 cases, 11 of which were attributed to a conversion disorder.

Roth, Aronson & Davis's Criteria for Psychogenic Stuttering

1. The disorder is manifest via the voluntary motor system (articulation and phonation),

2. Bizarre quality of dysfluencies and secondary behaviors,

3. History of stress prior to the onset of the stuttering behavior,

4. No permanent organic changes,

5. History of multiple somatic complaints,

6. Presence of primary or secondary gains,

7. A symbolic significance to the current disorder,

8. Previous exposure to someone with a neurological disease,

9. The presence of “la belle difference.”

Maher and Leith (1992) described four cases of psychogenic stuttering due to a conversion reaction that appeared to have “no organic causes.” Interestingly, however, all four cases suggest a neurological event or a series of neurological events that might have led to subtle neurological alterations, which in turn affected the speech system. For example, stuttering began in one case following a reported “mild respiratory depression” following surgery where a general anesthesia was administered. The patient awoke with mild speech hesitations and mild dysfluencies.

In a retrospective study, Baumgartner and Duffy (1997) examined the characteristics of 69 patients at the Mayo Clinic who were diagnosed as having psychogenic stuttering. (Note, however, that 20 cases had confirmed neurologic impairments with closed head injury and seizure disorders being the most common). After comparing the speech and language records of their 69 cases with the reported speech characteristics of neurogenic stuttering, Baumgartner and Duffy concluded that they could not distinguish neurogenic from psychogenic stuttering solely on the basis of the speech dysfluency. Features that did seem to distinguish the psychogenic group, however, were as follows.

Baumgartner and Duffy's Distinguishing Features

1. Rapid and favorable response to just one or two sessions of behavioral treatment,

2. Struggle behaviors and other signs of anxiety,

3. Intermittent or situational-specific episodes of stuttering,

4. Presence of unusual grammatical constructions, e.g., “Me get sick” and bizarre speech such as multiple repetitions of nearly all phonemes with simultaneous head bobbing, facial grimacing, and tremor-like arm movements.

According to Baumgartner and Duffy, rapid response to treatment was the feature that most often distinguished individuals with psychogenic stuttering from those with neurogenic stuttering. The most frequently occurring psychological diagnoses in their cases were conversion reaction, anxiety neurosis, and depression. Note that some patients had no prior history of psychiatric problems.

In summary, onset of stuttering in adults without a history of developmental stuttering has been described as being of either neurologic or psychologic origin with the forms having numerous overlapping features (see Table 1).

Table 1. Characteristics of neurogenic versus psychogenic stuttering.

Characteristics	Acquired stuttering	Psychogenic stuttering
Sudden onset	+	+
Repetitions, prolongations, and blocks occurring in all positions of words	+	–
Anxiety associated with the disorder	–	+
Consistent stuttering across fluency tasks (reading, speaking, repetition)	+	+
Secondary symptoms	–	–
Bizarre quality to the dysfluencies	–	+
A symbolic significance to the current disorder	–	+
Key: + present − absent		

The differential diagnosis of neurogenic from psychogenic stuttering, based solely on perceptual features of speech characteristics, has been shown to be unreliable. It appears, instead, that it is the rapid, favorable response to the treatment that best differentiates the psychogenic from the neurologic group. It is important to note again, however, that many of the cases of acquired stuttering described in the literature as being psychogenic in origin, may have had a neurogenic component Newer imaging technology may identify neurological changes in cases where the stuttering was initially considered to be psychogenic.

3. Neuropathological correlates of acquired neurogenic stuttering

Arguably, the most complex aspect of acquired neurogenic stuttering is the issue of its neuropathological correlates. A review of case reports with lesion information shows that various areas within the neuraxis have been implicated in this disorder. It would appear that acquired stuttering is not correlated with a lesion in one discrete area of the nervous system. Instead, it appears that lesions causing acquired stuttering have been located in all lobes of the both cerebral hemispheres and the cerebellum, deep white matter, the thalamus, and the brainstem. Most reported cases involve strokes causing definable lesions. For example, case studies by Grant et al. (1999) include a 68-year-old, right-handed man with a vascular lesion in the left frontotemporoparietal region; a 59-year-old, right-handed man with infarcts in the left posterior temporal lobe and bilateral cerebellum; a 59-year-old man, who was left-handed as a child but forced to use his right hand when he entered school, with a lesion in the right parietal cortex, and a 55-year-old, right-handed man with a medial left occipital lobe lesion. Note that two of these cases had resolved developmental stuttering that re-emerged after strokes; an occurrence reported by other investigators such as Mazzucchi, Moretti, Carpeggiani, Parma, and Paini (1981) and Helm-Estabrooks et al. (1986).

Several reports of acquired stuttering involved subcortical lesions. Ciabarra, Elkind, Roberts, and Marshall (2000) described a 53-year-old, right-handed man with a pontine infarct also affecting the cerebellum, a 54-year-old, right-handed woman with a left basal ganglia lesion (putamen, caudate, and corona radiata), and a 63-year-old, left-handed woman with a left corona radiata, putamenal and subinsular infarct. Fawcett (2005) described the case of an 84-year-old, left-handed woman with a left basal ganglia infarct. The 57-year-old patient of Balasubramanian, Max, Van Borsel, Rayca, and Richardson (2003) had bilateral pontine strokes that also involving the right orbital frontal area. Doi et al. (2003) reported the case of 60-year-old man who developed dysarthria and a “repetitive speech disorder” following a stroke involving the midbrain and upper pons. The resulting symptoms included repetition of the first and last syllable of a word, a gradual increase in the speed of speech production, and a gradual decrease in loudness. Doi and colleagues attributed the onset of stuttering to changes in the reticular network secondary to acute multiple brain infarcts to the brainstem.

Van Borsel, Van Der Made, and Santens (2003) proposed a unique clinical stuttering subtype of “thalamic stuttering” in a 38-year-old, right-handed man who experienced a stroke in the

left ventrolateral thalamus. The patient also displayed symptoms consistent with thalamic aphasia. The stuttering behavior was unique in that the patient was less fluent during propositional speech tasks. Although this is the first reported case of thalamic stuttering following a stroke, others have reported the appearance and disappearance of stuttering following thalamic stimulation (Ciabarra et al., 2000 and Ojemann and Ward, 1971).

Compared with the number of individuals whose stuttering was associated with strokes, fewer reported cases were associated with traumatic brain injury. Ludlow et al. (1987) discussed 10 cases of acquired stuttering in men with traumatic brain injury from missile wounds sustained in combat. The investigators compared the lesion sites in these men to those of a group of men who did not stutter following the same kind of injuries. Ludlow and colleagues found that the corpus callosum and the basal ganglia were impaired in the neurogenic stuttering group but not in the group of individuals with TBI who did not stutter. This finding contrasts with that of Lebrun et al. (1990) who described a 23-year-old man with persistent stuttering following a penetrating shrapnel wound to the right parietal lobe and the mesial aspect of the left parietal lobe.

Penetrating head wounds are more focal than closed head injuries which often result in multiple discrete lesions and diffuse axonal injury. Helm-Estabrooks and Hotz (1998) described a 30-year-old woman who began stuttering following closed head injury resulting from a motor vehicle accident. In addition to the diffuse axonal injury, results of magnetic resonance imaging (MRI) showed right frontal/parietal brain lesions.

The connection between epilepsy and neurogenic stuttering emerges repeatedly in the stuttering literature (Van Riper, 1971 and West, 1958) and is discussed in depth in an article by Lebrun (1991). Lebrun reviewed a number of cases where individuals either began to stutter following a seizure or ceased stuttering following the administration of seizure medications or following surgery for seizures (Manders and Bastijns, 1988 and Terzano et al., 1983).

Metabolic factors with global central nervous system effects have also been described as a causative factor in stuttering. Byrne et al.'s (1993) case of stuttering during a hypoglycemic state resulting from severe, chronic anorexia lends support to this notion. Also, Brady (1998) reviewed 16 reports (22 patients) of drug-induced stuttering that abruptly stopped when their medications were discontinued only to reappear when the same medications were readministered. The medications used affected cholinergic systems (tricyclic antidepressants), dopaminergic systems (neuroleptics), noradrenergic systems (propranolol, theophylline), and serotonergic systems (selective serotonin reuptake inhibitors). Additionally, stuttering has been associated with lithium and benzodiazepines. Although these cases suggest a compelling cause-and-effect relationship between the drugs and the stuttering, such a wide range of medications have been cited that the reports do not lead to a single, unifying biochemical explanation for acquired stuttering.

In sum, there appears to be no one neuropathological correlate that can be considered a reliable norm for acquired neurogenic stuttering. Perhaps, however, different lesions or different neurotransmitter systems may converge in some yet undetermined, final common pathway to produce a similar clinical picture. Alternatively, acquired stuttering may not represent one clinical entity but may be able to be further subclassified into more discrete types with distinct localizations, much as there are subtypes of aphasia. A factor that further complicates the issue of lesion localization and acquired stuttering, however, is that stuttering can follow traumatic brain injury. The identification of causative lesions in TBI is difficult due to the fact that diffuse axonal injury is typically superimposed on multiple focal lesions.

Clearly, elucidation of neuromechanisms underlying acquired stuttering requires more scientific investigation. Perhaps the next step is to conduct functional magnetic resonance imaging (fMRI) studies of individuals with this disorder. Such studies have been conducted with groups of individuals with chronic developmental stuttering and individuals who do not stutter. Blombergren, Nagarajan, Lee, Li, and Alvord (2003), for example, found that when carrying out the same verbal tasks, both groups showed activation in motor areas (primary motor cortex, premotor cortex, supplementary motor area, Rolandic operculum, lateral cerebellum) and auditory areas. Individuals who stuttered, however, showed overactivation in primary motor cortex, supplementary motor area, cingulate motor area, cerebellar vermis. Other areas showed right laterality (i.e., frontal operculum, Rolandic operculum, anterior insula). Recently, the study of Watkins, Smith, Davis, and Howell (2008) contributed additional information concerning differences between individuals who stutter and those who do not. One new finding was that during speech tasks, individuals who stuttered showed overactivation in the midbrain with extension into basal ganglia structures. Interestingly, their group of those with stuttering showed *underactivation* of cortical motor and premotor areas associated with speech articulation/production. Thus, fMRI investigations of developmental stuttering are addressing the underlying brain mechanisms of this disorder. While findings from these studies cannot be generalized to acquired stuttering in adults, this fMRI work may serve as a model for further exploration of mechanisms associated with neurogenic stuttering and may assist in differentiating a neurologic from a psychologic origin.

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