

REVIEW

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1 Department of Speech and Language Pathology and Audiology, Faculty of Education and Rehabilitation, University of Tuzla, Tuzla, Bosnia and Herzegovina

2 Medical Faculty, University of Tuzla; Sarajevo Medical School, University Sarajevo; School of Science and technology, Sarajevo; International Academy of Art and Science, Sarajevo, Bosnia and Herzegovina

3 Nurseries Public Institution „Ciciban“, Mostar, Bosnia and Herzegovina

Corresponding author: Lejla Junuzovic-Zunic, PhD., Full Professor, University of Tuzla, Faculty of Education and Rehabilitation, Department of Speech and Language Pathology and Audiology, Tuzla, Bosnia & Herzegovina. E-mail: lejla@gmail.com. ORCID ID: <https://orcid.org/0000-0003-0661-6335>.

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Neurogenic Stuttering: Etiology, Symptomatology, and Treatment

Lejla Junuzovic-Zunic¹, Osman Sinanovic², Blazenka Majic³

ABSTRACT

Background: Neurogenic stuttering is a subtype of acquired stuttering, and it is characterized by disfluencies associated with acquired brain damage. **Objective:** To provide an insight into pathophysiology, symptomatology, differential diagnosis, assessment, and treatment of neurogenic stuttering through a critical review of the literature. **Methods:** Studies published during the past and recent years were searched and analyzed on neurogenic stuttering. **Results:** Neurogenic stuttering is a complex disorder. The pathophysiological mechanism of neurogenic stuttering is not yet fully understood. It appears with several neurological diseases and conditions, and the use of some drugs. Differential diagnosis of neurogenic and psychogenic stuttering is a challenge for clinicians. Treatment usually requires a joint effort from speech therapists and doctors, most often neurologists. **Conclusion:** Although research on neurogenic stuttering can be found in the literature, the complexity of this disorder still requires detailed monitoring and studying to provide the best treatment for patients.

Keywords: acquired stuttering, neurogenic stuttering, psychogenic stuttering

1. BACKGROUND

Stuttering is an interruption of speech flow characterized by the occurrence of specific types of disfluencies including repetitions of sounds, syllables, and monosyllabic words, consonant prolongations, and blockages. These disfluencies can affect the rate and rhythm of speech and may be accompanied by negative reactions to speaking, avoidance behaviors, struggling behaviors, and physical tension (1). Stuttering usually occurs in children and these are usually cases of developmental stuttering which is the most frequent form of stuttering (2). However, some first-time occurrences of stuttering can also manifest in later life, usually in connection with neurological outbursts, as side effects of certain medications or due to psychological trauma. This kind of stuttering is referred to as acquired stuttering, although the term acquired stuttering is sometimes misused as a synonym of neurogenic stuttering (3).

Stuttering is usually classified into developmental and acquired stuttering. The term acquired stuttering is widely accepted and probably the most commonly used to denote fluency disorders of non-developmental origin. It points to the fact that disfluencies were not always present, as well as to the occurrence of disfluencies related to a certain age. The term neurogenic stuttering can be defined as a subtype of acquired stuttering in which disfluencies are associated with acquired brain damage in a person who did not stutter before brain damage (3). The neurological event itself is usually a head trauma or a disease that leads to disturbances in the normal functioning of the brain (4). Neurogenic stuttering can occur at any age as a result of neurological impairment (5). Additionally, there are reports of the onset of neurogenic stuttering in childhood in literature (4). There are known cases of three-year-old children whose neurogenic stuttering occurred due to damage caused by rotavirus, encephalitis, or trauma, as well as ninety-three-year-olds where neurogenic stuttering occurred as a result of stroke, neurodegenerative disease, and trauma. However, the highest incidence is in adults (5).

Data on the prevalence and incidence of neurogenic stuttering are still insufficient (6, 7, 8) because most published studies are based on a case report or a fewer number of cases (8, 9). Although the prevalence of neurogenic stuttering is thought to be low, this is not an uncommon disorder in clinical practice (4, 6, 8, 10). The frequency of neurogenic stuttering is higher in men (4). More accurate data on the incidence of neurogenic stuttering would help clinicians and researchers in better identifying these patients and thus in providing appropriate intervention (6).

2. OBJECTIVE

To provide an insight into pathophysiology, symptomatology, differential diagnosis, assessment, and treatment of neurogenic stuttering through a critical review of the literature.

3. RESULTS AND DISCUSSION

PATHOPHYSIOLOGY OF NEUROGENIC STUTTERING

The underlying pathophysiological mechanism of neurogenic stuttering is not yet fully understood. Contributing to this is the fact that neurogenic stuttering may be associated with multiple pathologies and with different lesion sites (8). During the analysis of the data on the etiology of neurogenic stuttering, it was found that neurogenic stuttering cannot be exclusively associated with damage to a particular part of the brain, but may involve various neurological structures that are part of the neural network for fluent speech production (11). Neurological structures that may be involved include four lobes of both hemispheres, cerebellum, subcortical white matter, basal ganglia, thalamus, and brainstem (12). Research shows that the left hemisphere of the brain is more commonly affected (6).

Neurogenic stuttering in most cases occurs as a result of a stroke. The occurrence of neurogenic stuttering as a result of a stroke is not limited to a lesion in a particular region of the brain but is the result of overlap with the cortico-basal ganglion-cortical network, which includes the lower frontal cortex, upper temporal cortex, intra- parietal cortex, basal ganglia, and their white matter interconnections through the superior longitudinal fasciculus and internal capsule. These areas are part of the neural sensory and motor network in speech production. Thus, one or more lesions in the said network may be a trigger for neurogenic stuttering, however, they are not directly related to the number of lesions in the brain and the severity of stuttering, which means that the severity of stuttering does not depend on the size of the lesion, but on the impact, it has on the neural network (13). Stuttering often begins shortly after a stroke in subjects with stroke, as the largest etiologic subgroup of neurogenic stuttering, and the lesions were less diffuse compared to other etiologies (6).

Traumatic brain injuries lead to a variety of long-term consequences, and one of the consequences that can occur is neurogenic stuttering. Neurogenic stuttering is most often the result of traumatic brain injury and stroke (14). When comparing the lesion site in a total of ten patients with neurogenic stuttering after brain injury (caused by a projectile from a firearm) with persons without injuries and neurogenic stuttering, it was determined that the outer capsules, frontal white matter, and striatum were significantly more affected in the group of patients with neurogenic stuttering. Symptoms in the conversational speech included uncontrollable speech rate, long pauses, accelerated speech rate between pauses, increased number of repetitions of syllables and words, prolongation, interjections, and rapid incomprehensible speech outbursts as a distinguishing feature

Name of drug	Research
Clozapine	Kumar, Kathpal & Longshore (24); Bar, Hager & Sauer (25); Murphy et al. (23)
Risperidone	Yadav (26); Rocha (27)
Olanzapine	Bar, Hager & Sauer (25)
Lithium	Netski & Piasecki (28)
Fluoxetine	Messiha (29)
Phenothiazines	Numberg & Greenwald (30)
Tricyclic antidepressants	Quader (31)
Alprazolam	Elliott & Thomas (32)

Table 1. Drugs associated with stuttering

(15). Difficulty finding words, language difficulties, and apraxia are often observed in patients with traumatic brain injury. In addition, problems in planning and executing narrative discourse may result in disfluencies which may indicate neurogenic stuttering (16). There was a case, in which a patient with neurogenic stuttering that occurred as a result of a traumatic head injury, had diffuse axonal damage with lesions in the right frontal and parietal lobes. Speech characteristics included slower pronunciation, word prolongation, repetition of the initial sound, pauses, and hesitation. What was interesting is that in addition to speech pathology, the patient also had uncontrolled leg movements, face, and neck tension, as well as twitching movements of the right side of the face, the lips, chin, and cheeks (17).

Lu et al. emphasize the role of the basal ganglia-thalamocortical circuit in speech fluency, emphasizing the difference in its functioning between non-stuttering and stuttering individuals. The results of the study show that in stuttering individuals there are deficiencies in the connection between the basal ganglia, thalamus, and cerebral cortex (including the frontal motor cortex and the temporal auditory cortex), which affects temporal control in speech production (18). From an anatomical point of view, the characteristics in stuttering individuals include increased gray matter volume of the left putamen and decreased gray matter volume of the left medial frontal gyrus and anterior superior temporal gyrus, and decreased white matter volume in the left posterior superior temporal gyrus within the basal ganglia-thalamocortical circuit (19). In practice, there is also a known type of stuttering that is unsystematically classified as neurogenic stuttering–thalamic stuttering, where the thalamus is part of the basal-ganglion thalamotor system, therefore the lesion on any part can lead to disturbances in impulse transmission to the cerebral cortex (3). Individuals who stutter show a stronger projection from the putamen to the thalamus than individuals who do not stutter (20). The basal-ganglion thalamotor system may play a particularly important role in a possible focal stuttering disorder (Alm, 21).

There are other causes of neurogenic stuttering such as cysts and other types of neoplasm, degenerative diseases (Parkinson's disease and multiple sclerosis), diseases such as meningitis, Guillain-Barre syndrome, and HIV, as well as causes related to the overuse of certain

Psychogenic stuttering	Neurogenic stuttering
- sudden onset	- sudden onset
- unusual forms of fluency, such as multiple repetitions of all phonemes, followed by facial grimaces, nodding, and tremor-like movements	- repetitions, prolongation, blockade in all positions in words
- consistency of stuttering through different speech tasks	- consistency of stuttering through different speech tasks
- the symbolic significance of the current disorder	
- the disfluencies occur during speech anywhere in a word or utterance	- disfluencies occur during speech anywhere in a word or utterance
- a person can be indifferent to their speech	- a person is often unaware of the disorder but can be frustrated by their speech
- bizarre voice quality	
- anamnestic data indicate a history of emotional problems (personality disorder, post-traumatic stress disorder, drug addiction, anxiety, or depression)	
- diagnosis of psychopathology is not necessary	
- the person gives the impression of "sticking" to a certain pattern of disfluencies and continues to stutter in conditions that improve fluency and during the imitation of mimic movements;	
- after expressing emotional information, there is a sudden improvement in fluency;	
- after a short period of therapy rapid and satisfactory progress is noted	
- worsening of symptoms when performing simpler tasks	
- worsening of stuttering during re-reading the same text	
- bizarre movements (for example head and eyes) and signs of anxiety unrelated to speech production	
- unusual grammatical constructions	
- the existence of an occasional episode of stuttering or stuttering in specific situations	

Table 3. Stuttering characteristics indicating possible psychogenic/neurogenic stuttering

drugs (22). The onset of stuttering due to the use of drugs refers to the occurrence of stuttering-like disfluencies (SLD) due to the use of drug therapy, which is also called pharmacological stuttering (3). Drugs used for neurological or some other specific pharmacological interventions can cause stuttering (23). Some of them are listed in Table 1.

Studies show that there is a connection between epilepsy and neurogenic stuttering. That is because, in practice, several cases have been identified in which there was an onset of stuttering immediately after or sometime after the epileptic seizures. It is important to emphasize that the cause and effect relationship between epilepsy and stuttering has not been fully clarified (33). Stuttering can be the result of brain injury as well as abnormal electrical activity, or the result of a lesion or paroxysmal changes within the mechanism of speech production. There are two directions of connection, i.e. epilepsy and stuttering can occur at the same time as a consequence of the same neurological damage and be mutually independent or stuttering can develop as a result of altered electrical activity caused by neurological damage (19). In that case, if epileptic seizures are eliminated, there is a possibility to eliminate the symptoms of stuttering (33).

SYMPTOMATOLOGY OF NEUROGENIC STUTTERING

The onset of neurogenic stuttering in many patients is not easy to determine. Neurogenic stuttering is usually

- Repetition of syllables and sounds, while blockades are less frequent
- Disfluencies are almost as common with substantive words as with non-substantive words;
- The speaker may seem worried about stuttering, but does not show anxiety in terms of repetitions or prolongation, and the blockages appear anywhere in a word or utterance, as opposed to the initial word position in developmental stuttering
- Secondary symptoms rarely occur even if facial grimaces, blinking, and clenching of the fists do occur, it is not tied to moments of disfluencies
- There is no adaptation effect
- There is consistency in stuttering in different speech tasks (conversation, explanation, repetition, and reading)
- People often show additional signs of aphasia and dysarthria

Table 2. The most common symptoms of neurogenic stuttering

associated with cerebrovascular insults, or, in some cases, they occur several months after a medical problem has been diagnosed (4). The often described characteristics of neurogenic stuttering in the literature list the symptoms presented in Table 2 (8, 9).

Assessment of general symptomatology is an important step in the assessment of neurogenic stuttering. Headache, left or right hemiparesis, tremor, or coordination difficulties are symptoms that allow for easier diagnosis because they are more often associated with neurogenic stuttering rather than some other type of stuttering (6). Due to communication difficulties, neurogenic stuttering can also cause social incompetence, negative self-perception, and negative perception of

communication, leading to social isolation which negatively affects the quality of life (10). Accurate and expeditious recognition of neurogenic stuttering can reduce or even prevent such communication difficulties (34).

DIFFERENTIAL DIAGNOSIS OF PSYCHOGENIC AND NEUROGENIC STUTTERING

The unavailability of neuropsychological literature describing speech and language abnormalities of psychogenic origin results in a lack of guidelines for distinguishing psychogenic and neurogenic types of speech and language disorders (35). Differential diagnosis in the case of neurogenic and psychogenic stuttering presents a challenge for clinicians due to the manifestation of similar symptoms or their overlap (11, 36). Another aggravating circumstance for the precise differentiation of acquired stuttering is the fact that some cases of acquired stuttering clearly have a psychological or neuropsychiatric genesis rather than a neuropathological one. Therefore, it is important to exclude the existence of neurological impairments during a speech-language assessment, based on anamnestic data (34). Some of the characteristics of psychogenic and neurogenic stuttering that may be useful in making a differential diagnosis are shown in Table 3 (12, 34, 36, 37).

Table 3. Stuttering characteristics indicating possible psychogenic/neurogenic stuttering

The indistinguishability between neurogenic, developmental, and psychogenic stuttering could lead to ineffective assessments and generally inappropriate interventions, which in turn would prevent adequate advances in stuttering therapy (38).

Neurogenic stuttering can be associated with other communication disorders, and the most common are aphasia, dysarthria, apraxia of speech, palilalia, anomia, and confusion (39, 40, 41). Tani and Sakai state that 35% of cases of individuals with neurogenic stuttering, which are known in the literature, are associated with aphasia or dysarthria to some degree (9). Due to the presence of comorbid speech and language disorders, it can be difficult, and sometimes even impossible, to distinguish certain types of speech and language difficulties and stuttering (42). Diagnosis of neurogenic stuttering is a complex process and requires knowledge of other speech and language disorders. Therefore, it is not always easy to establish clearly defined boundaries between neurogenic stuttering and other speech-language disorders of neurological origin (43). The direction of the treatment of neurogenic stuttering is determined by comorbid symptomatology. Therefore, systematic identification of symptoms or disorders present during the assessment is important in order to apply appropriate therapy (6).

ASSESSMENT OF NEUROGENIC STUTTERING

Assessment of neurogenic stuttering involves anamnestic data, i.e. medical, social, and speech anamnesis (44), and neurological examination (39). Key diagnostic questions include knowledge of the intervals of neurological and psychological trauma, information on environmental factors, and the time of initiation of medical therapy, if administered (6). Standard assessment in-

cludes analysis of a representative speech sample, determination of disfluencies index, examination of secondary behaviors, assessment of speech rate, assessment of opinions and attitudes related to stuttering. Assessment of motor speech performance is indispensable to determine whether there are additional motor speech disorders that affect speech fluency (44). Stuttering needs to be analyzed in different speech tasks. It is necessary to determine the position of disfluency in words and the occurrence of disfluency in substantive and non-substantive words (43), as well as the adaptation effect (9). An important diagnostic marker in the assessment of neurogenic stuttering is the appearance of symptoms at the same time as brain damage (45). If the scans do not show the presence of neurological impairment, cognitive abilities may be an indicator of brain damage and they should be assessed. Afterward, cognitive testing should be performed if a neurological impairment is suspected and scans do not show that is the case (11).

NEUROGENIC STUTTERING THERAPY

Previous research has characterized neurogenic stuttering as persistent and time-resistant to speech therapy (11, 43). However, individual cases and advances have been reported when it comes to the use of speech therapy, which includes counseling, and the use of fluency modification and fluency shaping techniques. The results of neurogenic stuttering therapy depend primarily on the neuropathology of neurogenic stuttering. If the damage can be reduced or removed, the outcome in therapy will be more successful. On the other hand, if cases of neurological impairment require a long recovery period, progress is rarely seen in the early stages of therapy (11). Considering there are multiple conditions that can cause neurogenic stuttering and affect the frequency with other communication disorders with which it coexists, it cannot be said that there is only one treatment approach that is successful in alleviating neurogenic stuttering. Treatment usually requires a joint effort from speech therapists and doctors, most often neurologists (39). Treatment methods traditionally used in the treatment of developmental stuttering are also used in neurogenic stuttering (8). Speech therapy remains the mainstay in the treatment of stuttering. Multiple strategies to improve fluency can be used, including facilitation of word pronunciation, speech rate reduction, fluency modification-modeling mechanisms, choral speech, metronome speech, voice pitch change, white noise (8, 40). Stuttering therapy most commonly involves speech therapy using behavioral and cognitive methods (46).

Neurogenic stuttering can also be treated with medication, although it is not the first choice in the treatment of neurogenic stuttering. When it comes to medications, haloperidol is the most commonly used drug and has potential beneficial effects on the treatment of stuttering (46). Other antipsychotic medications used in the treatment of stuttering, that have also been mentioned in the studies, include chlorpromazine, trifluoperazine, thioridazine, and atypical antipsychotics such as risperidone and olanzapine, and antiepileptic drugs such as carba-

mazepine, sodium valproate, and levetiracetam (47). Although stuttering is most often treated with speech therapy and psychotherapy, research shows that several antipsychotic medications also present a possible way of treatment (46).

Therapy for neurogenic stuttering may include counseling or education about the impairment if the person shows anxiety or concern about the impairment present. Counseling also includes assistance to families and caregivers. There is no consensus on which is the most effective among the various possible methods available. All of these methods can be used alone, sequentially, or combined (39).

4. CONCLUSION

The pathophysiology of neurogenic stuttering is not fully understood due to the occurrence of neurogenic stuttering concerning different lesion sites. Also, the spectrum of symptoms caused by different lesions leads to difficulty in understanding and defining neurogenic stuttering. The use of a complete battery of tests to assess neurogenic stuttering facilitates the diagnostic process and timely diagnosis. For professionals dealing with this issue, the list of symptoms of neurogenic stuttering facilitates differential diagnostics. With an accurate diagnosis, it is possible to make an appropriate individualized treatment plan that will be effective in eliminating or reducing neurogenic stuttering. The complexity of neurogenic stuttering requires further study to provide patients with the best intervention based on scientific evidence, as well as guidelines for the work of professionals dealing with these patients.

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