

# Recovery of Recurrent Transient Neurogenic Stuttering due to Functional Neuroplasticity

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## ABSTRACT:

Although epilepsy symptoms are well established, there are only several described cases of post seizure speech fluency impairments. Epileptic activity may interfere with speech but speech impairments have the ability to recover because of neural plasticity that has been widely investigated in epilepsy population. In the available literature there is only one case report of transient neurogenic stuttering described. In this case report we describe a recovery of a 33-year-old male patient with recurrent transient neurogenic stuttering after focal idiopathic seizures due to functional neuroplasticity.

**KEYWORDS:** adult-onset epilepsy, recurrent transient neurogenic stuttering, functional neuroplasticity

## SAŽETAK

OPORAVAK PONAVLJAJUĆEG PROLAZNOG NEUROGENOG MUCANJA ZBOG FUNKCIONALNE NEUROPLASTIČNOSTI

Iako su simptomi epilepsije poznati, postoji samo nekoliko opisanih slučajeva poremećaja tečnosti govora nakon epileptičkog napada. Epileptička aktivnost može ometati govor, ali govorna oštećenja imaju sposobnost oporavka zbog plastičnosti mozga koja je detaljno istražena u bolesnika s epilepsijom. U dostupnoj literaturi opisan je samo jedan slučaj prolaznog neurogenog mucanja. U ovom prikazu slučaja opisujemo oporavak 33-godišnjeg bolesnika s ponavljajućim prolaznim neurogenim mucanjem nakon fokalnih epileptičkih napadaja zbog funkcionalne neuroplastičnosti.

**KLJUČNE RIJEČI:** epilepsija u odrasloj dobi, prolazno neurogeno mucanje, funkcionalna neuroplastičnost

## OPEN ACCESS

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This article was submitted to RAD  
CASA - Medical Sciences  
as the original article

### Conflict of Interest Statement:

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

**Received:** 15 May 2023

**Accepted:** 01 June 2023

**Published:** 26 June 2023

### Citation:

Roje Bedeković M, Pilepić L. Recovery of Recurrent Transient Neurogenic Stuttering due to Functional Neuroplasticity 556=62-63 (2023): 82-84  
DOI: 10.21857/yvjrdcvjyy

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## INTRODUCTION

Epilepsy is one of three most common neurological disease that can occur in all age groups. According to a recent study, 38% of epilepsies starts between the age of 21 and 40, with 10% of them being idiopathic (1). Even though temporal lobe seizures occur in 75% of patients (2) and can cause a complete loss of speech, most evidence on speech fluency impairments after seizures are described in case reports (3). The term neurogenic stuttering is a subtype of acquired stuttering in which disfluencies are caused by acquired brain damage in a person who did not stutter before brain damage (3). Unlike developmental stuttering, which typically begins in childhood, neurogenic stuttering can appear at any age following a neurological event or injury. Neurogenic stuttering can take many forms and can vary in severity depending on the underlying cause and the extent of the brain damage. It may involve repetitions, prolongations, or blocks of sounds, syllables, or words. In some cases, patients with neurogenic stuttering may also experience other speech difficulties, such as slurred speech or difficulty pronouncing certain sounds or words. Patients with transient neurogenic stuttering can recover their speech fluency using speech language therapy because of early brain plasticity (4). Because most epileptic seizures occur in hippocampus where brain plasticity has been proven, patients can recover from postictal speech and language disorders with early rehabilitation.

In this case report we describe a case of transient neurogenic stuttering because of a focal seizure in a young male patient that recovered with early speech therapy.

## CASE REPORT

A 33-year-old male patient was hospitalized in a general hospital because of speech dysarthria and left arm weakness. Urgent brain computed tomography (CT) and computed tomography angiography (CTA) were normal. During the hospitalization an echocardiogram (ECG), transcranial doppler (TCD) and an electroencephalogram (EEG) were performed and showed no abnormalities. Magnetic resonance imaging (MRI) showed one small chronic vascular lesion in left frontal gyrus and one small oval hypodensity in frontal lobe centrum semiovale described as microhemorrhage. MRA showed no arteriovenous malformation or aneurisms. For the previous month before hospitalization, the patient had been working in a stressful environment and was sleep deprived. He was treated with acetylsalicylic acid. After discharge from the general hospital, only mild speech dysarthria persisted in his neurological status. He was referred to a speech language pathologist in Department of neurology – Sestre Milosrdnice University Hospital Center. Speech language pathologist did a thorough assessment for aphasia, dysarthria, speech apraxia and fluency disorders. Comprehensive language battery and audio-perceptual assessment showed no impairments. Fluency assessment included analysis of a representative

speech sample, determination of disfluencies index, examination of secondary behaviors and assessment of speech rate. Neurogenic stuttering was diagnosed because of severe fluency impairments. After three therapies consisting of breathing techniques and speaking on a relaxed exhale, speech fluency improved to normal, and the patient didn't need any more rehabilitation. After two months he developed thrombophlebitis on his left arm and again was hospitalized in Sestre Milosrdnice University Hospital Center. As a part of a neurological examination an EEG was done that showed an epileptiform brain activity in temporal lobe and therefore oxcarbazepine treatment was introduced. During this time, no speech impairments were noticed. After five months patient was admitted to emergency department of Sestre Milosrdnice University Hospital Center because he woke up with left arm weakness and severe speech fluency difficulties. He didn't lose consciousness. CT and CTA were normal again. Speech language pathologist stated that speech fluency had the same type of impairment as seven months ago and left arm weakness was understood as Todd's paresis. He was treated with an intravenous dose of diazepam. Todd's paresis fully recovered in two hours, but speech fluency impairment remained. An MRI scan and control CT scan were performed after three and five days and showed no lesions. Patient was treated with intensive speech therapy for fluency impairments during hospitalization and was discharged with mild speech fluency disturbances and final diagnosis of idiopathic epilepsy. In a 2 week follow up no further speech fluency impairments were noted.

## DISCUSSION

In this case report we describe a young male patient with epilepsy that developed transient neurogenic stuttering. Several case reports described neurogenic stuttering after seizures but none of them described a transient form of neurogenic stuttering after seizures.

Epileptic activity can affect speech in a variety of ways, depending on the location of the seizure in the brain and the severity of the seizure (5). Some patients with epilepsy may experience speech difficulties such as slurred or slowed speech, difficulty finding words or expressing themselves, or changes in voice pitch or tone. In some described cases, focal seizures can cause brief interruptions in speech or periods of stuttering (6). Seizures can also cause sudden and unexpected changes in speech, such as speaking in a foreign accent or using inappropriate or nonsensical language (5). However, the connection between seizures and stuttering has mostly been investigated in children and some authors even suggest stuttering as a "relative of epilepsy" (6). Neurogenic stuttering occurs as a sudden onset adult subtype of stuttering with numerous neurological causes on record (7). This form of stuttering is significantly less common than the developmental type, and as a result, it has received much less attention in research (5). What is interesting, however, is that neurogenic

stuttering as a fluency disorder is heavily seen as a symptom rather than a diagnosis. One reason to explain this one-sided perspective in the literature is that the neurophysiology of stuttering is still unclear, as it is considered a complex process (5). Recovery of seizure symptoms is achievable because of brain plasticity (8) that has been widely investigated in epilepsy population. Brain plasticity in epilepsy population has been widely researched mostly because of a remarkable deficit recovery that hasn't been noted in other neurological diseases (9). The recovery is possibly due to a plastic reorganization in the central nervous circuitry as a result of neural damage (5). Hippocampus is one of the most common epilepsy center (9, 10). Abnormal electric activity in that brain area causes neuron damage but doesn't cause permanent neurological deficit because of high brain plasticity. The dentate gyrus which is a part of hippocampus is made of granulate cells that have been investigated for their ability to

recover and change after seizure (11, 12). Our patient developed recurrent transient neurogenic stuttering after two focal seizures as a consequence of idiopathic epilepsy. He luckily recovered speech fluency during a period of 2 weeks with early speech language therapy.

With this case report we emphasize the importance of early identification of fluency disorders in patients with epilepsy in order to differentiate them from other possible acquired speech and language impairments that occur with seizures. We also emphasize the importance of early speech language therapy stimulating early neural plasticity and shortening recovery period.

#### ACKNOWLEDGMENTS

We declare no conflicts of interest. The patient's consent was obtained. We did not receive any material support.

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