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## Assessment and treatment of acquired neurogenic stuttering: A case report

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### Abstract

Neurogenic stuttering is an acquired subtype of stuttering following brain damage such as cerebrovascular accident (CVA). This case involved a 51-year-old Malayalam-speaking male with a left parieto-temporal infarct causing speech disfluencies, right-sided weakness, and mild dysarthria. Assessment showed reduced verbal output, articulatory errors, intact comprehension, and mild stuttering (SSI-4 score 23). The individualized intervention included fluency shaping, prosodic training, counseling, and family involvement, delivered in three phases: establishment, transfer, and maintenance. Therapy improved disfluencies, intelligibility, and communication attitude, though setbacks occurred after a seizure. This highlights the effectiveness of evidence-based speech-language therapy for neurogenic stuttering post-CVA and the need for further comprehensive research to enhance clinical outcomes.

**Keywords:** Neurogenic stuttering, prolonged speech, severity ratings, case report

### Introduction

Stuttering is an interruption in speech flow characterized by sound repetitions, prolongations, and blocks (ASHA, 2021)<sup>[3]</sup>. Secondary behaviors include visible struggle and avoidance of speaking situations (Majic, 2021)<sup>[26]</sup>. Emotional consequences include embarrassment, anxiety, and reduced self-confidence, often causing social withdrawal (Craig *et al.*, 2009)<sup>[7]</sup>. Stuttering is classified into developmental and acquired types. Acquired stuttering, emerging after early childhood, includes psychogenic and neurogenic forms (Theys *et al.*, 2008)<sup>[33]</sup>. Neurogenic stuttering follows neurological events like stroke, TBI, or Parkinson's disease and involves repetitions, prolongations, and blocks throughout speech with minimal visible tension (DeVries, 2022)<sup>[11]</sup>. Lesions linked to neurogenic stuttering include the basal ganglia and frontal lobe (Helm-Estabrooks, 1999)<sup>[17]</sup>. Despite clinical relevance, neurogenic stuttering remains underdiagnosed with limited research and intervention protocols (Cruz *et al.*, 2018)<sup>[8]</sup>. This case report explores the clinical profile and therapy outcomes of neurogenic stuttering post-CVA, highlighting the value of detailed case studies in advancing understanding and management of this disorder.

### Case Presentation

**Participant details:** A 51-year-old Malayalam-speaking male from Calicut, Kerala, with a postgraduate degree in Business Administration and over ten years of experience as a sales manager, presented with speech difficulties following a CVA. He had no history of smoking or alcohol use and had been managing type 2 diabetes mellitus for 15 years and systemic hypertension for the past 3 years. On 11/10/2024, he experienced an acute infarct in the left parieto-temporal region, with right-sided weakness and signs of heart failure. Neuroimaging confirmed an infarct in the left front-opsiarietal region, with no haemorrhagic transformation. Following initial hospital-based care, he received two months of home-based speech therapy, which was later discontinued due to the unavailability of services. Subsequently, he was referred to the Association for the Welfare of the Handicapped (AWH) Special College in Calicut for a comprehensive evaluation and therapy.

The study utilised a single-subject case design that included a pre- & post-assessment phase and an intervention phase.

Assessment procedure: Pre-assessment phase starts with systematic data collection, focusing on gathering a comprehensive case history analysis and evaluating key communication skills. This phase serves as the foundation for clinical decision-making and intervention planning.

Following a comprehensive case history, which included both medical and non-medical background information, revealing that the client was on medications such as Clopilet A, Storvas 40 mg, Monit GNT 2.6 mg, Glimy M2, and Januvia 100mg - prescribed for the management of diabetes, cholesterol, and cardiac conditions and he is also attending physiotherapy from October 2024 as part of ongoing rehabilitation, further an in-depth assessment of the client's speech and language skills was conducted.

The following assessment tools were administered to evaluate different aspects of the client's communication

- Western Aphasia Battery (WAB) - Malayalam (Philip J E, 1992) <sup>[28]</sup>.
- Frenchay Dysarthria Assessment (FDA) -2 (Enderby, 2008) <sup>[13]</sup>.
- Stuttering Severity Instrument (SSI) - 4 (Riley, 2009) <sup>[29]</sup>
- GRBASI Scale (Hirano, 1981)
- Speech Intelligibility Rating Scale (AYJNHH, 1984)
- Modified Erickson Scale of Communication Attitudes – S-24 (Erickson, 1969) <sup>[14]</sup>.

A range of standardized assessment tools was administered to evaluate specific aspects of the client's communication profile. The WAB-M was used to assess overall language function and to rule out the presence of aphasia (Philip J E, 1992) <sup>[28]</sup>. FDA-2 evaluated the structure and function of the speech musculature to determine the presence and severity of dysarthria (Enderby, 2008) <sup>[13]</sup>. SSI-4 was employed to quantify the severity of stuttering and identify specific patterns of dysfluency (Riley, 2009) <sup>[29]</sup>. To assess voice quality, the GRBASI scale was used, which analyses six perceptual parameters: Grade, Roughness, Breathiness, Asthenia, Strain, and Instability (Hirano, 1981) <sup>[18]</sup>. The Speech Intelligibility Rating Scale measured the intelligibility of connected speech (AYJNHH, 1984), while the Modified Erickson Scale of Communication Attitudes (S-24) assessed the client's attitude towards communication and stuttering (Erickson, 1969) <sup>[14]</sup>. In this case, the absence of an apraxia component was inferred from the patient's consistent ability to respond spontaneously, comply with voluntary commands, and provide appropriate answers to questions, while the lack of speech discrepancies was further corroborated by multiple sources, including caregiver reports and analysis of earlier audio recordings. The pre- and post-assessment measures of the above tests are discussed in the discussion section.

Statistical analysis: Data are presented based on behavioural observations, test results, and treatment outcomes were analysed qualitatively through detailed narrative description and interpretation.

**Ethics approval:** Ethics approval was obtained from the institution's ethical committee

## Discussion

A comprehensive overview of the speech and language assessments administered to the client, the clinical findings, and the treatment interventions implemented is provided below.

## Clinical Features and Symptoms of Neurogenic Stuttering Following CVA

**Speech Assessment Findings:** The client exhibited dysfluencies such as initial syllable and word repetitions, interjections, filled and unfilled pauses, and blocks in all word positions. Articulatory errors including substitution, distortion, and omission were observed during conversational and reading tasks. Speech intelligibility was reduced, with increased rate and impaired prosody. Although neurogenic stuttering typically lacks an adaptation effect, the client demonstrated a clear presence, consistent with Tani and Sakai (2011) <sup>[31]</sup>, who reported positive adaptation in patients with basal ganglia lesions. While neurogenic stuttering has been described as resistant to fluency-enhancing conditions like singing or choral reading (Helm-Estabrooks, 1999) <sup>[17]</sup>, recent evidence shows some patients exhibit variability. Some stroke and brain-surgery patients showed positive adaptation during repeated readings and were more fluent in automatic speech or reading than spontaneous speech, indicating a minority may experience fluency benefits similar to developmental stuttering.

**Speech Subsystems:** Evaluation of the speech subsystems revealed reduced articulatory coordination and sequencing. This impairment results in difficulty transitioning smoothly between sounds or syllables, which likely contributes to dysfluencies such as repetitions and blocks. The client also exhibits challenges in producing rapid sequences of sounds, especially during complex or multisyllabic words, further reducing intelligibility, particularly at higher speech rates, as observed in the present case.

In addition, inadequate phonatory and respiratory sufficiency was noted, which results in fluctuations in voice quality, pitch, and loudness control, potentially impacting overall vocal effectiveness and prosody. Also, insufficient or poorly coordinated breath support makes it difficult to sustain speech during longer utterances or reading tasks. These respiratory deficits often compound difficulties with coordination and fluency, as speech may be forced or interrupted by the need to take breaths at inappropriate times.

Furthermore, an increased rate of speech combined with impaired prosody (including the melody, rhythm, and stress patterns of speech) can lead to monotonous, rushed, or unnatural-sounding speech, making communication less effective and expressive, as seen in this case.

**Language Assessment Findings:** The client demonstrated adequate comprehension of auditory verbal commands, yes/no questions, and connected passages. He participated in spontaneous, automatic, and responsive speech with only minimal difficulty. However, mild impairments were observed in generative naming and morphosyntactic skills. A detailed summary of test results is presented in Table 1 below.

To confirm the diagnosis, the assessment results from various measures were compared with findings reported in existing literature (Table 2 below). The current case was provisionally diagnosed as neurogenic stuttering (Speech fluency disorder secondary to CVA) with mild dysarthria. The client demonstrated a non-aphasic language profile, mild dysarthria, and mild stuttering with an SSI-4 score of 23. GRBASI ratings indicated mild hoarseness and roughness, without signs of strain or instability. Intelligibility, as measured by the Speech Intelligibility

rating scale, was rated at 2, reflecting mostly intelligible speech with occasional repetitions. The prognosis for neurogenic stuttering is variable; some individuals achieve significant gains, while others experience only partial or minimal improvement, depending on various medical, neurological, and therapy-related factors.

### Speech-Language Therapy and Outcomes in Neurogenic Stuttering Following CVA

**Therapy Procedure:** Therapy was delivered in three phases Establishment, Transfer, and Maintenance through an individualized, evidence-based plan targeting fluency, articulatory coordination, prosody, intelligibility, and communicative confidence. The client attended 40 sessions: 13 in the establishment phase, 15 in generalization, and 12 in maintenance, which is ongoing. Sessions occurred about three times per week, each lasting 40 minutes.

During the Establishment Phase, therapy targeted awareness and physiological ease using techniques such as gentle onset, light articulatory contacts, and continuous phonation to reduce speech tension. Prolonged Speech (O'Brian *et al.*, 2003) <sup>[27]</sup> and reduced speech rate promoted smooth transitions and improved timing. Melodic Intonation Therapy (Albert *et al.*, 1973) <sup>[1]</sup> engaged alternative neural networks. Delayed Auditory Feedback (Yates, 1963) <sup>[37]</sup> altered feedback to reduce disfluencies. Voluntary stuttering and pullout (Van Riper, 1973) <sup>[36]</sup> improved control and reduced fear. Combining stuttering modification and fluency shaping improves fluency and emotional aspects (Langevin *et al.*, 2006) <sup>[21]</sup>. Articulatory training and language tasks targeted clarity, naming, and morphosyntax, with vocal modulation for pitch/loudness (Boone, 2013) <sup>[6]</sup>. Relaxation and anxiety-reduction strategies supported emotional communication (Guitar, 2014) <sup>[16]</sup>. Stuttering Severity Scale, adapted from the Camperdown Program, rated stuttering severity from 0 (none) to 8 (extremely severe). Clients were trained to self-rate, enabling ongoing evaluation and treatment adjustments (Cullinan & Prather, 1968) <sup>[9]</sup>.

In the Transfer Phase, fluency techniques were generalized through role-plays, monitored conversations, real-time feedback, and structured exposure to high-pressure speaking (Li *et al.*, 2024; Tichenor *et al.*, 2022) <sup>[22, 34]</sup>, emphasizing independent strategy use. The Maintenance Phase focused on sustaining gains with personalized fluency toolkits, family involvement, and gradually reduced sessions from twice weekly to monthly and beyond to quarterly or biannually for long-term follow-up.

A post-therapy evaluation was conducted to measure changes in fluency, intelligibility, emotional response, and speech control.

Therapy yielded significant improvements in fluency, as evidenced by a reduction in the SSI 4 score from 23 to 17, within the very mild range, which reflects a reduction in the frequency and severity of disfluencies, including initial syllable repetitions, word repetition, interjections, and blocks. This quantitative improvement was further supported by spontaneous speech observations during therapy, where the client used pull-out techniques and self-correction more effectively, especially in semi-structured conversations and reading aloud. According to GRBASI ratings for roughness and breathiness decreased from 1 to 0, reflecting improved voice quality. Speech intelligibility improved from 2 to 1, with the client being mostly understood and exhibiting fewer repetitions. Emotional adaptation was noted, with the S-24 score dropping to 10 and increased speech initiation and reduced anxiety observed. Maximum Phonation Duration (MPD) improved

from 12 to 20, 22, and 23 seconds for /a/, /i/, and /u/, respectively, indicating enhanced breath support and control. Articulatory errors diminished. The patient showed improvements in naming, morphosyntactic accuracy, and compensatory strategies in spontaneous and structured tasks. Although a gliotic seizure caused temporary motivation loss and MPD decline, therapy continued, facilitating fluency recovery and confidence. Session-wise data showed gradual technique improvement with some variability post-seizure. Counselling, relaxation, and family involvement supported emotional resilience and skill maintenance. Intensive therapy yielded notable improvements consistent with neuroplasticity (Lundgren, Helm-Estabrooks, & Klein, 2010) <sup>[24-25]</sup>.

### Conclusion

This case report illustrates that neurogenic stuttering following CVA benefits from intensive speech-language intervention combining fluency, articulation, voice, prosody, language skills, and counseling. Tailored therapy reduced disfluencies, improved intelligibility, and fostered positive communication attitudes. The patient's seizure highlighted the need for continuous support and adaptive strategies to sustain fluency. Findings emphasize holistic, individualized therapy addressing speech-motor, linguistic, and emotional dimensions of neurogenic stuttering. Larger studies are needed to refine intervention protocols and support evidence-based care. Unique successes or failures in a single patient serve as pilot data, demonstrating potential new therapeutic approaches for systematic testing. Future research should expand sample size, examine targeted therapy effectiveness on speech motor control, cognitive-linguistic factors, and emotional regulation, and include communication partners to understand broader impacts on speech behaviors, emotions, and participation, improving intervention efficacy (Baxter *et al.*, 2016) <sup>[4]</sup>.

### Conflicts of interest

The authors declare that there are no conflicts of interest regarding the publication of this article.

### Consent to participate

Written informed consent was obtained from the patient for publication of this case report.

### Funding declaration

This case report received no funding.

### Authors' contributions

P.S.S (second author) conceptualized the study, designed the methodology, performed the literature search, contributed to data interpretation. R.M. (first author) conducted the assessment and treatment, was responsible for data collection, analysis, and initial manuscript drafting. The original manuscript draft was prepared by R.M., while P.S.S critically reviewed, revised, and approved the final version.

### References

- Albert ML, Sparks RW, Helm NA. Melodic intonation therapy for aphasia. *Archives of Neurology*. 1973;29(2):130-131. <https://doi.org/10.1001/archneur.1973.00490260074018>
- Alm PA. Stuttering and the basal ganglia circuits: a critical review of possible relations. *Journal of Communication Disorders*. 2004;37(4):325-369.
- American Speech-Language-Hearing Association. Fluency disorders. ASHA; n.d.

4. Baxter S, Johnson M, Blank L, Cantrell A, Brumfit S, Enderby P, *et al.* Non-pharmacological treatments for stuttering in children and adults: a systematic review. *Health Technology Assessment*. 2016;20(2):1–302.
5. Blomgren M. Behavioral treatments for children and adults who stutter: a review. *Psychology Research and Behavior Management*. 2013;6:9–19.
6. Boone DR, McFarlane SC, Von Berg SL, Zraick RI. *The voice and voice therapy*. 9th ed. Boston: Pearson; 2013.
7. Craig A, Blumgart E, Tran Y. The impact of stuttering on the quality of life in adults who stutter. *Journal of Fluency Disorders*. 2009;34(2):61–71. <https://doi.org/10.1016/j.jfludis.2009.05.002>
8. Cruz C, Amorim H, Beca G, Nunes R. Neurogenic stuttering: a review of the literature. *Revista de Neurologia*. 2018;66(2):59. <https://doi.org/10.33588/rn.6602.2017151>
9. Cullinan WL, Prather EM. Reliability of live ratings of the speech of stutterers. *Perceptual and Motor Skills*. 1968;27:403–409.
10. De Nil LF. Neurogenic stuttering. In: Murdoch BE, editor. *Acquired speech and language disorders*. London: Routledge; 2019. p. 243–262.
11. DeVries N. Neurogenic stuttering: exploring potential emotional and life impact [master's thesis]. Western Michigan University; 2022.
12. Duffy JR. Motor speech disorders: substrates, differential diagnosis, and management. 2nd ed. St. Louis: Elsevier Mosby; 2005.
13. Enderby PM. *Frenchay Dysarthria Assessment – Second Edition (FDA-2)*. Pearson; 2008.
14. Erickson RL. Assessing communication attitudes among stutterers. 1969.
15. Grant AC, Bioussse V, Cook AA, Newman NJ. Acquired stuttering associated with acute infarction of the left middle cerebral artery territory. *Neurology*. 1999;52(6):1206–1209. <https://doi.org/10.1212/WNL.52.6.1206>
16. Guitar B. *Stuttering: an integrated approach to its nature and treatment*. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2014.
17. Helm-Estabrooks N. Stuttering associated with acquired neurological disorders. In: Curlee RF, editor. *Stuttering and related disorders of fluency*. 2nd ed. New York: Thieme; 1999. p. 321–334.
18. Hirano M. Clinical examination of voice. In: Daniloff GA, editor. *Speech evaluation in voice disorders*. San Diego: College-Hill Press; 1981. p. 81–84.
19. Iverach L, Rapee RM. Social anxiety disorder and stuttering: current status and future directions. *Journal of Fluency Disorders*. 2014;40:69–82. <https://doi.org/10.1016/j.jfludis.2014.02.001>
20. Jokel R, De Nil LF, Sharpe KS. Speech disfluencies in adults with neurogenic stuttering: a case study. *Journal of Communication Disorders*. 2007;40(4):334–351. <https://doi.org/10.1016/j.jcomdis.2006.12.002>
21. Langevin M, Huinck WJ, Kully D, Peters HF, Lomheim H, Tellers M. A cross-cultural long-term outcome evaluation of the ISTAR Comprehensive Stuttering Program. *Journal of Fluency Disorders*. 2006;31:229–256.
22. Li J, Wu S, Leshed G. Re-envisioning remote meetings: co-designing inclusive and empowering videoconferencing with people who stutter. *Proceedings of the Designing Interactive Systems Conference*. 2024:1926–1941. <https://doi.org/10.1145/3643834.3661533>
23. Ludlow CL, Rosenberg J, Salazar A, Grafman J, Smutok MA, Hallett M. Site of penetrating brain lesions causing chronic acquired stuttering. *Annals of Neurology*. 1987;22(1):60–66. <https://doi.org/10.1002/ana.410220112>
24. Lundgren K, Helm-Estabrooks N, Klein R. Stuttering following acquired brain damage: a review of the literature. *Journal of Neurolinguistics*. 2010;23(5):447–454. <https://doi.org/10.1016/j.jneuroling.2010.03.002>
25. Lundgren K, Helm-Estabrooks N, Klein R. A fluency treatment program for chronic neurogenic stuttering. *Seminars in Speech and Language*. 2010;31(4):287–299. <https://doi.org/10.1055/s-0030-1265764>
26. Majic B, Junuzovic-Zunic L, Sinanovic O. Neurogenic stuttering: etiology, symptomatology, and treatment. *Medical Archives*. 2021;75(6):456.
27. O'Brian S, Onslow M, Cream A, Packman A. The Camperdown Program. *Journal of Speech, Language, and Hearing Research*. 2003;46(4):933–946. [https://doi.org/10.1044/1092-4388\(2003/073\)](https://doi.org/10.1044/1092-4388(2003/073)
28. Philip JE. *Test of Aphasia in Malayalam*. Mysore: University of Mysore; 1992.
29. Riley GD. *Stuttering Severity Instrument – Fourth Edition (SSI-4)*. Austin: Pro-Ed; 2009. Available from: <https://www.proedinc.com/Products/13025/ssi4-stuttering-severity-instrument--fourth-edition.aspx>
30. Rosenbek J, Messert B, Collins M, Wertz RT. Stuttering following brain damage. *Brain and Language*. 1978;6:82–96.
31. Tani T, Sakai Y. Analysis of five cases with neurogenic stuttering following brain injury in the basal ganglia. *Journal of Fluency Disorders*. 2011;36(1):1–16. <https://doi.org/10.1016/j.jfludis.2010.12.002>
32. Theys C, Van Wieringen A, De Nil LF. A clinician survey of speech and non-speech characteristics of neurogenic stuttering. *Journal of Communication Disorders*. 2013;46(2):159–172. <https://doi.org/10.1016/j.jcomdis.2013.01.001>
33. Theys C, Van Wieringen A, De Nil LF. A clinician survey of speech and non-speech characteristics of neurogenic stuttering. *Journal of Fluency Disorders*. 2008;33(1):1–23. <https://doi.org/10.1016/j.jfludis.2007.09.001>
34. Tichenor SE, Herring C, Yaruss JS. Understanding the speaker's experience of stuttering can improve stuttering therapy. *Topics in Language Disorders*. 2022;42(1):57–75. <https://doi.org/10.1097/tld.0000000000000272>
35. Van Borsel J, Taillieu C. Neurogenic stuttering versus developmental stuttering: a case study. *Journal of Communication Disorders*. 2001;34(5–6):385–395. [https://doi.org/10.1016/S0021-9924\(01\)00049-2](https://doi.org/10.1016/S0021-9924(01)00049-2)
36. Van Riper C. *The treatment of stuttering*. Englewood Cliffs (NJ): Prentice-Hall; 1973.
37. Yates AJ. Delayed auditory feedback. *Psychological Bulletin*. 1963;60(3):213–232. <https://doi.org/10.1037/h0044155>