Transient Somatosensory Evoked Potential Attenuation as a Warning Sign of Delayed Vertebrobasilar Infarction After Anterior Cervical Discectomy and Fusion

Jacob Chaisson,BS[1]; Jack Leoni, BS[1]; Ellery Hayden, MD[2]; Gabriel Tender, MD[2]; Amit Bhandutia, MD[3]
[1]School of Medicine, LSUHSC, New Orleans, LA
[2]Department of Neurosurgery, LSUHSC, New Orleans, LA
[3]Department of Orthopaedic Surgery, LSUHSC, New Orleans, LA

Introduction

- **Anterior cervical discectomy and fusion (ACDF)** is widely performed for cervical decompression and stabilization, with perioperative neurologic complications in only ~0.4 % [1].
- **Vertebral artery injury (VAI)** is rare (~0.07 %) [2], and **perioperative ischemic stroke** occurs in ~0.15 % of cases [3]—vertebrobasilar events being the least common.
- **Unrecognized vascular vulnerability** (atherosclerosis, tortuosity, intimal disease) may predispose patients to ischemia during multilevel ACDF but is often missed without **CTA-based risk assessment** [4–7].
- **Intraoperative neuromonitoring (IONM)** with SSEP/MEP can detect evolving neurologic injury [8].
- **Case objective:** Report a rare **delayed vertebrobasilar infarction** following multilevel ACDF with **transient unilateral SSEP attenuation**, emphasizing **vascular risk stratification** and **postoperative vigilance** [9–11].

Case

- **Patient:** 61-year-old man with hypertension and poorly controlled hyperlipidemia, presenting with 2-month history of progressive cervical myelopathy (bilateral paresthesia, impaired fine motor control, frequent object dropping).
- **Preoperative MRI:** Multilevel degenerative spondylosis (C4–C7) with severe canal stenosis and T2 cord signal consistent with myelomalacia *(Figure 1)*.
- **No preoperative vascular imaging** (CTA/MRA) was obtained, as standard ACDF protocols focus on spinal rather than vertebral artery anatomy.
- **Procedure:** Standard three-level ACDF (C4–5, C5–6, C6–7) via anterior approach.
- **Intraoperative neuromonitoring (IONM):**
  - Baseline somatosensory evoked potentials (SSEPs) symmetric and stable.
  - After first cage placement (C4–5) → transient unilateral attenuation of right upper and lower extremity (RUE/RLE) SSEPs.
  - Mean arterial pressure (MAP) maintained >85 mmHg; anesthetic depth (MAC) temporarily reduced (1.3 → 0.5).
  - SSEPs recovered fully within ~20 min and remained stable for the remainder of surgery *(Figure 2)*.

References

[1] Kashkoush A, Mehta A, Agarwal N, et al. *World Neurosurg.* 2019;128:e107–e115. **[2]** Ball JR, Shelby T, Mertz K, et al. *World Neurosurg.* 2024;181:e841–e847. **[3]** Ishak B, Abdul-Jabbar A, Singla A, et al. *Spine (Phila Pa 1976).* 2020;45(2):109–115. **[4]** Sano A, Hirano T, Watanabe K, et al. *Spine (Phila Pa 1976).* 2013;38(15):E960–E967. **[5]** Okano I, Salzmann SN, Winter F, et al. *J Neurosurg Spine.* 2021;36(2):261–268. **[6]** Caplan LR. *Curr Treat Options Cardiovasc Med.* 2003;5(3):251–256. **[7]** Brott TG, Halperin JL, Abbara S, et al. *J Am Coll Cardiol.* 2011;57(8):1002–1044. **[8]** Buhl LK, Bastos AB, Pollard RJ, et al. *J Intensive Care Med.* 2021;36(11):1237–1249. **[9]** Reddy RP, Chang R, Rosario BP, et al. *Spine J.* 2021;21(4):555–570. **[10]** Epstein NE, Danto J, Nardi D. *Spine (Phila Pa 1976).* 1993;18(6):737–747. **[11]** Phillips JL, Chalouhi N, Jabbour P, et al. *Neurosurgery.* 2014;75(5):560–567. **[12]** Kamel I, Zhao H, Koch SA, et al. *Anesth Analg.* 2016;122(5):1423–1433. **[13]** Sloan TB. *J Clin Neurophysiol.* 1998;15(3):217–226. **[14]** Anastasian ZH, Ramnath B, Komotar RJ, et al. *Anesth Analg.* 2009;109(3):817–821. **[15]** Noël P, Desmedt JE. *Brain.* 1975;98(1):113–128. **[16]** Guan Q, Chen L, Long Y, Xiang Z. *World Neurosurg.* 2017;106:715–722. **[17]** Cagnie B, Barbaix E, Vinck E, et al. *Surg Radiol Anat.* 2006;28(2):129–134. **[18]** Dickerman RD, Zigler JE. *Spine (Phila Pa 1976).* 2005;30(21):E658–E661. **[19]** Ishii D, Ishibashi K, Takeda K, et al. *Front Hum Neurosci.* 2021;15:761186. **[20]** Sameshima T, Morita A, Yamaoka Y, Ichikawa Y. *J Stroke Cerebrovasc Dis.* 2014;23(1):191–193. **[21]** Tran CT, Khoo LT, Martin NA, et al. *J Clin Neurophysiol.* 2012;29(1):17–22.

Postoperative Course

- **Immediate postop:** Uneventful; neurologically intact and extubated without issue.
- **POD 1 (7 AM):** Developed **right facial droop, dysarthria, hemiparesis, and tongue deviation** (NIHSS 7).
- **Imaging:**
  - **CT head:** Right cerebellar hypodensity with mild 4th-ventricle compression.
  - **CTA: Right vertebral artery thrombus** (V2 segment, C2–C6) *(Figure 3).*
  - **MRI brain: Acute right cerebellar and left pontine infarcts**, consistent with **embolic vertebrobasilar stroke** *(Figure 4).*
- **Management:** Neuro-ICU admission; **3 % hypertonic saline**, dual antiplatelet therapy (aspirin + clopidogrel); **no thrombolytics or decompression.** Required **intubation and tracheostomy** for airway protection.
- **Outcome:** Discharged to rehab on **POD 14**; at **1-year**, **full functional recovery** with 5/5 strength and resolution of dysarthria. Follow-up radiographs confirmed **solid C4–C7 fusion** with intact hardware.

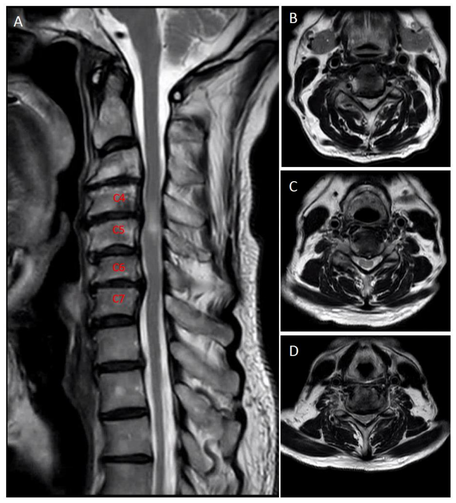

Figure 1. Preoperative MRI of the cervical spine demonstrating multilevel degenerative pathology. Sagittal T2-weighted imaging (A) shows severe canal stenosis from C4 to C7 with associated cord signal change. Axial images (B–D) show compressive pathology at the C4–5, C5–6, and C6–7 levels.

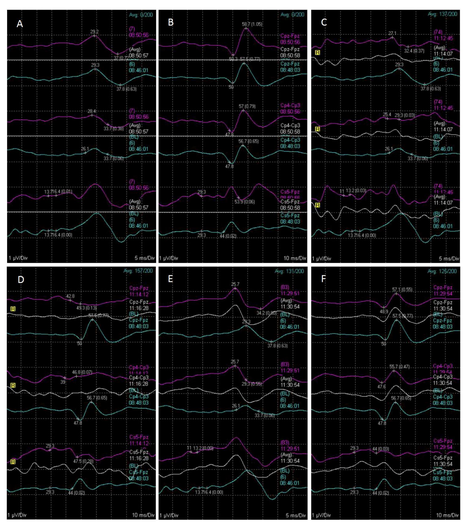

Figure 2. Intraoperative SSEPs during ACDF. Panels A–B: Baseline right upper (A) and lower extremity (B) SSEPs with symmetric waveforms and stable amplitudes across subcortical (Cs5–Fpz) and cortical (Cpz–Fpz, Cp4–Cp3) channels. Panels C–D: Attenuation of right-sided SSEPs after graft placement at C4–5 (C: RUE; D: RLE), most pronounced cortically. Panels E–F: Recovery of amplitudes following anesthetic reduction and positional adjustment. Blue = baseline; white = cumulative average; magenta = most recent trace.

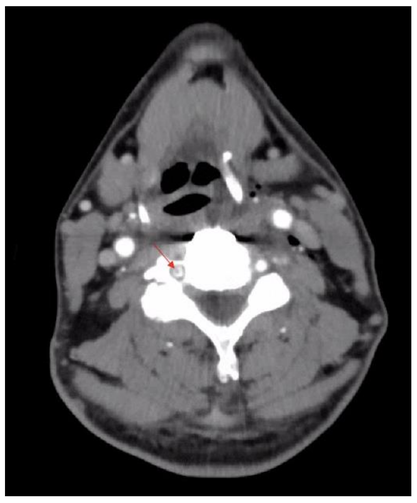

Figure 3. Axial CTA of the neck showing a focal thrombus in the right vertebral artery (V2 segment, red arrow). This finding correlates with the patient's delayed posterior circulation infarcts, supporting a vertebrobasilar embolic mechanism following ACDF.

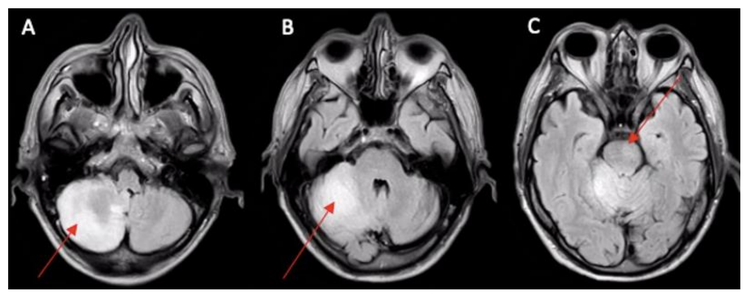

Figure 4. Axial T2-weighted MRI demonstrating multifocal posterior circulation infarcts. (A) Right cerebellar hyperintensity at the level of the inferior cerebellar peduncles; (B) right cerebellar hyperintensity at the middle cerebellar peduncle; (C) left paramedian pontine hyperintensity at the rostral pons. Red arrows indicate areas of abnormal T2 signal consistent with embolic vertebrobasilar ischemia.

Discussion & Conclusion

- **Transient unilateral SSEP changes**, especially involving both upper and lower extremities, may reflect **vertebrobasilar hypoperfusion** rather than anesthetic or positional artifact [12–15].
- **Proposed mechanism:** Multilevel exposure, retraction, or cage insertion may cause **intimal stress or kinking** of a diseased vertebral artery, leading to **delayed thromboembolism** [6,7,16–18].
- **Pattern interpretation:** Simultaneous **RUE/RLE attenuation** indicates disruption of **brainstem or subcortical sensory pathways** supplied by the vertebrobasilar system [19–21].
- This appears to be the **first reported case** of **delayed vertebrobasilar infarction** following ACDF associated with **transient lateralized SSEP changes**.
- **Preventive measures:** Maintain **MAP >85 mmHg**, minimize retraction, and perform structured **postoperative neuro checks** to detect evolving vascular injury early.
- **Limitations:** Single-case design; larger studies needed to validate mechanisms and refine IONM response protocols.