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*Case Report*

# Dynamic changes of abnormal muscle response during decompression procedures in double compression-type hemifacial spasm

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## ABSTRACT

**Background:** Hemifacial spasm (HFS) is a neurovascular movement caused by vascular compression of the facial nerve in its root exit zone (REZ). Cases of HFS caused by double compression (DC) in both REZ and the cisternal portion (CP) have been sporadically reported. The nature of DC-type HFS is still not fully understood. Compression in CP is often overlooked, resulting in reoperation in DC-type HFS cases.

**Case Description:** A 48-year-old man with a 3-year history of left HFS was admitted to our department. Magnetic resonance imaging revealed that the vertebral artery (VA) passed around REZ of the facial nerve, and the anterior inferior cerebellar artery (AICA) was in contact with the facial nerve in CP. Microvascular decompression was performed while monitoring any abnormal muscle response (AMR). Although VA was dissected and detached from REZ, AMR showed only a transient decrease and the amplitude of the AMR wave soon recovered and subsequently increased. No other vessels compressing REZ beneath VA were found. AICA attached to the facial nerve in CP and was compressed upward by VA. When AICA was moved from the facial nerve in CP after the transposition of VA, AMR was immediately resolved. After surgery, the patient was completely free from HFS.

**Conclusion:** In DC-type HFS, precise preoperative diagnosis and intraoperative identification of the culprit vessel are difficult. In DC-type HFS, decompression of one side of a vessel may exacerbate the compression of the other side. In such a case, AMR helps us become aware of compressions in CP that we may preoperatively overlook. AMR is useful for identifying the exact culprit vessels and recognizing any compression changes caused by intraoperative manipulations.

**Keywords:** Abnormal muscle response, Anterior inferior cerebellar artery, Cisternal portion, Hemifacial spasm, Microvascular decompression, Vertebral artery

## INTRODUCTION

Hemifacial spasm (HFS) is usually caused by compression of the facial nerve by blood vessels in the root exit zone (REZ). Microvascular decompression (MVD) is widely known as a treatment for HFS. Good outcomes and low complication rates have been reported.[13] Jannetta stated that the compression findings in REZ should be focused on during treatment.[5] It has been reported that compression in any part of the facial nerve can cause HFS.[17,18] Compression in the cisternal

portion (CP) is the cause in 0.14–5.9% of cases undergoing MVD.[1,2,7,12,16] HFS due to distal neurovascular compression can be classified into two types: pure distal compression in CP by a single culprit without any culprits in REZ and double compression (DC) in both CP and REZ.[14] DC-type HFS is associated with a higher reoperation rate than the pure distal compression type.[14] Identification of the culprit vessels in both REZ and CP is difficult.[7,11,16,18] In DC-type HFS, the culprit vessels in CP are the anterior inferior cerebellar artery (AICA) or posterior inferior cerebellar artery (PICA). On the other hand, culprit vessels in REZ are AICA, PICA, a vein, or a combination of two vessels [Table 1]. However, there are a few reported cases of DC-type HFS associated with the vertebral artery (VA) as the culprit artery.[14,16,18]

Real-time abnormal muscle response (AMR) monitoring has been used to assess the adequacy of facial nerve decompression. AMR can predict postoperative symptom resolution.[8] Especially in DC-type HFS, identification of a culprit vessel is difficult; therefore, the effectiveness of AMR should be emphasized.[11,16]

Recently, we encountered a case of DC-type HFS in which AICA in CP was considered to be the direct culprit vessel. In our case, the AMR amplitude did not completely disappear but rather exacerbated after the release of nerve compression by VA in REZ alone. Translocation of the thick VA in REZ might exacerbate tandem compression of AICA by the distal VA in CP. This anatomical change of vessels might result in exacerbation of compression of the facial nerve by AICA in CP. We report a case of DC-type HFS caused by VA and AICA, showing dynamic changes in the AMR wave in which AMR was effectively utilized.

## CASE REPORT

A 48-year-old man with a 3-year history of the left HFS was admitted to our department. His facial spasms gradually increased in frequency and became persistent almost all day long. The spasm was particularly aggravated under stress, and he sometimes could not open his eyes. He had no previous medical history. Preoperative neurological examinations revealed no abnormal findings other than HFS. On magnetic resonance imaging, bilateral VAs were thick, and thicker right VA severely deviated to the left. The tortuous left VA passed around and compressed the REZ of the facial nerve. AICA originated from the basilar artery and ran along the left VA close to the proximal portion of the facial nerve. AICA was in contact with the facial nerve in CP [Figure 1]. VA was considered to be the cause of HFS, and MVD was performed.

Lateral suboccipital craniotomy was conducted. The left VA was thick and hard. The left VA ran beneath the facial nerve. VA was on REZ of the facial nerve and also compressed the trigeminal nerve. AICA runs along the VA and is in contact with the facial nerve [Figure 2a]. To move the thick VA, its translocation from the proximal to the distal side was necessary. Moving VA along its entire running course was difficult; therefore, VA was initially transposed by inserting Teflon felt between the brain stem and VA caudal to the facial nerve. At that time, the AMR wave transiently decreased in amplitude but soon recovered (date not shown). Then, the AMR waveform soon increased compared with that before the manipulation of the vessel [Figure 2b]. VA could not be sufficiently moved due to its stiffness and the existence of perforators from the VA to the brain stem. Furthermore, the VA attached to and compressed the trigeminal nerve. To move the VA and decrease the compression of the trigeminal nerve, the interposition of the VA was done by inserting Teflon felt between the VA and the trigeminal nerve on the cranial side of the facial nerve [Figure 2c, mid and right panels]. However, the AMR wave did not vanish after this procedure. Therefore, the size and location of Teflon felt were changed on the caudal side of the facial nerve. Teflon felt was placed caudal to REZ, not to compress REZ directly. By these procedures, VA could be sufficiently moved out of REZ of the facial nerve. Other vessels rather than VA were not found in REZ. However, AMR did not disappear, but further increased in amplitude [Figure 2c]. At that time, AICA was pushed up toward the facial nerve by the VA and strongly compressed the nerve in CP [Figure 2c]. After the translocation of VA, its distal part was considered to compress AICA more compared with the condition before translocation. Then, we tried to detach AICA

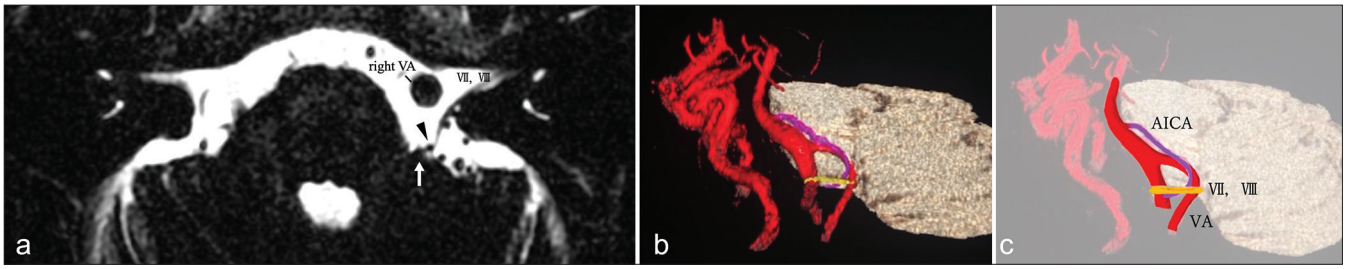


**Figure 1:** (a) Constructive interference in steady state imaging of magnetic resonance imaging (MRI) revealing vascular compression at the root exit zone by the left vertebral artery (VA) (arrow) and at the cisternal portion by anterior inferior cerebellar artery (AICA) (arrowhead). (b) MRI facilitates 3D confirmation of the relationships between vessels and nerves. (c) Schematic drawing of 3D MRI. VII: Facial nerve, VIII: Vestibulocochlear nerve.

**Table 1:** Summary of 35 patients with hemifacial spasm caused by double compression.

| Authors, year | Total cases | Age/sex | Confirmation of distal comp. | Reoperation | AMR | Culprit at REZ | The culprit at the cisternal portion | Treatment | Anatomical feature | Relief of spasm | Complication |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Nagahiro *et al.*, 1991 | 1 | N/S | 2nd MVD | + | N/S | AICA | AICA | Transposition | Cross type | Excellent | No major |
| Fukuda *et al.*, 1997 | 1 | 61/F | 2nd MVD | + | 1st no AMR 2nd + | PICA | AICA | Interposition | Cross type near IAM | Excellent | None |
| Ryu *et al.*, 1998 | 4 | 51/F | N/S | + | N/S | AICA | AICA | N/S | N/S | Excellent | Temporary VII weakness |
| | | 51/F | 2nd MVD | Cured on 3rd | N/S | AICA | AICA | Attached to dura mater | The mid portion of VII | Excellent | None |
| | | 33/F | 2nd MVD | + | N/S | AICA | AICA | Attached to dura mater | Very distal | Excellent | Hearing loss |
| | | 73/M | MRI before 2nd MVD | + | N/S | PICA | PICA | Attached to dura mater | The mid portion of VII | Excellent | None |
| Li *et al.*, 2010 | 1 | 51/M | 2nd MVD | + | Disappeared at 2nd MVD | AICA | AICA | Interposition | Cross type | Excellent | Mild facial weakness |
| Zhong *et al.*, 2010 | 7 | N/S | Intraop. | + | N/S | VA+AICA | AICA | Interposition | | Excellent | Temporary VII weakness |
| Zheng *et al.*, 2011 | 21 | 25–67/M: 6, F: 15 | N/S | - | Disappeared by decompression of REZ | AICA 6<br>AICA 15<br>PICA 1, vein 1<br>AICA+PICA 2<br>VA+PICA 1 | AICA 6<br>AICA 20 | Transposition or interposition | Cross type | Cure 17<br>Good 3 | No major |
| Present case, 2023 | 1 | 48/M | Intraop. | - | + | VA | AICA | Transposition and interposition | AICA attached to VA | Excellent | None |

AICA: Anterior inferior cerebellar artery, AMR: Abnormal muscle response, F: Female, IAM: Internal auditory meatus, Intraop.: Intraoperation, M: Male, MRI: Magnetic resonance imaging, MVD: Microvascular decompression, N/S: Not specified, PICA: Posterior inferior cerebellar artery, REZ: Root exit zone, VA: Vertebral artery, VII: Facial nerve

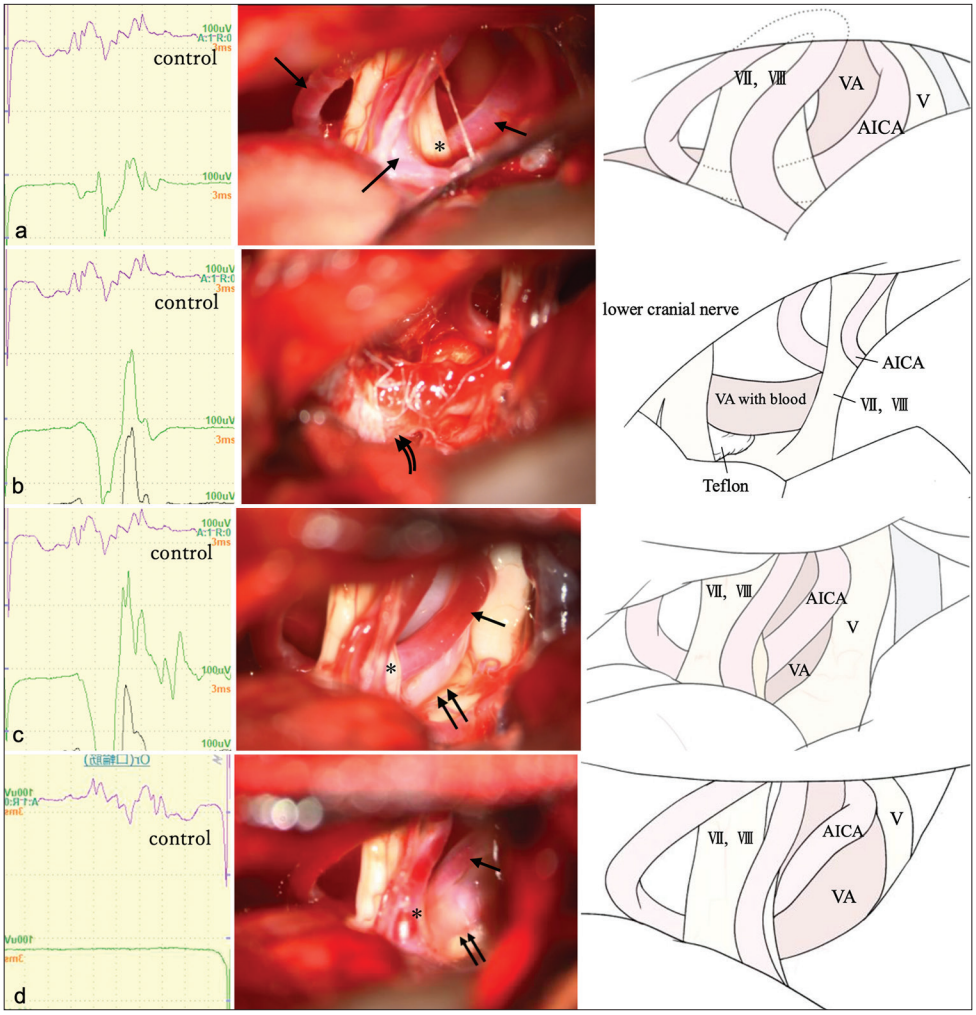


**Figure 2:** Abnormal muscle response (AMR) waves (left panels), intraoperative photographs (mid), and schematic illustrations (right). (a) Intraoperative findings before microscopic manipulation. The anterior inferior cerebellar artery (AICA) (arrow) on the vertebral artery (VA) is in contact with the facial nerve (asterisk). (b) AMR waves (left panels), intraoperative photographs (mid), and schematic illustrations (right). Teflon felt (curved double arrows) is inserted between the brain stem and VA. AMR is amplified after decompression of the root exit zone. (c) AMR waves (left panels), intraoperative photographs (mid), and schematic illustrations (right). Compression of the facial nerve by AICA (arrow) is exacerbated after further decompression of both sides of the facial nerve (asterisk). AMR is further amplified. (d) AMR waves (left panels), intraoperative photographs (mid), and schematic illustrations (right). Compression of the facial nerve by AICA (arrow) is completely resolved by transposition using fibrin glue (asterisk). AMR remains absent after surgery. double arrows: VA, V: Trigeminal nerve, VII: Facial nerve, VIII:Vestibulocochlear nerve.

manually from the facial nerve and found that AMR was attenuated. This phenomenon indicated that AICA and VA at CP were direct and indirect culprits, respectively. Therefore, the transposition of AICA was performed by sticking AICA to the anterior surface of the deep existing VA with fibrin glue. AMR waves immediately disappeared after these procedures [Figure 2d]. Continuous monitoring of auditory brain stem responses during surgery revealed no abnormal findings. His postoperative course was uneventful, and HFS has been completely resolved since the operation.

## DISCUSSION

HFSs are basically due to the compression of nerves caused by beating blood vessels in REZ.[5] However, HFS can be induced by compression of any part of the facial nerve.[17,18] The problem with HFS caused by compression in CP is the difficulty of precise preoperative recognition of a culprit vessel, especially in DC-type HFS. In a previous report, 51.6% of cases of distal compression were classified as DC-type HFS.[14] Although vessels attach to the nerve in both

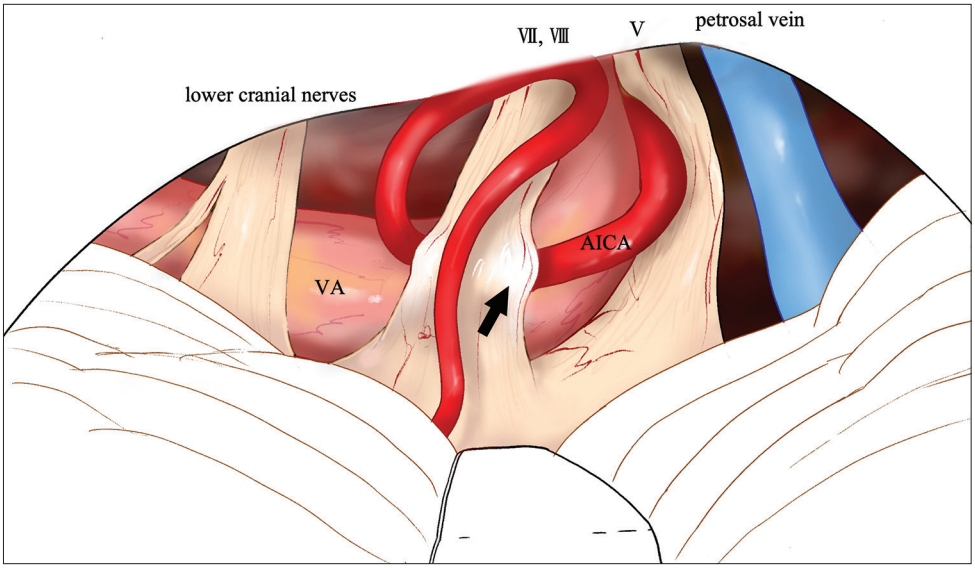


**Figure 3:** Overview illustration representing compression of the facial nerve by vertebral artery (VA) and anterior inferior cerebellar artery (AICA). Transposition of VA and decompression of the root exit zone exacerbated facial nerve compression at the cisternal portion through the AICA (arrow). V: trigeminal nerve, VII: Facial nerve, VIII: Vestibulocochlear nerve.

REZ and CP in DC-type HFS, the symptoms are not relieved by decompression of the nerve only in REZ. AMR disappears or symptoms are improved after decompression of the nerve in CP in such cases. To the best of our knowledge, a total of 35 cases of DC-type HFS have been described in 6 reports [Table 1]. In DC-type HFS, vessels existing at REZ are variable, such as AICA, PICA, veins, and VA, along with other vessels [Table 1]. However, in REZ of DC-type HFS, VA compression alone in the absence of other vessels has not been reported. In our case, only VA compressed the facial nerve in REZ, but it was not the culprit artery. In cases of common HFS caused by VA in REZ, a small vessel is often present.[6,9,10,15] Therefore, even if VA is suspected as the culprit on radiological examinations, it is important to carefully search for other vessels in addition to VA in REZ initially.[9] If other small vessels are not found in REZ, and decompression of VA in REZ does not result in AMR improvement, the existence of other vessels at other locations should be explored.

In DC-type HFS, the vessel most frequently compressing CP is AICA (97%). AICA being compressed by VA in CP was considered to be the cause of HFS in our case. VA was found in REZ and simultaneously contributed to the compression of AICA in CP. This means that the thick VA contacted AICA in CP and contributed to the compression of the facial nerve. A translocated VA in REZ might exacerbate compression of AICA in CP after the decompression procedure in REZ [Figure 3]. Therefore, the AMR wave did not disappear even after sufficient decompression at REZ. Other small vessels anatomically interacting with VA should be carefully searched for. There is a possibility that a small vessel in CP is a direct culprit vessel indirectly compressed by another large vessel such as VA. Nagahiro *et al.* reported that proximal decompression of the nerve exacerbated distal compression.[11] In our case, VA was atherosclerotic, thick, and hard. In such a case, the translocation of the vessel at one location may affect the position of the vessel in another location as a lever. This might be one of the reasons why the reoperation rate for DC-type HFS is as high as 40% [Table 1]. The reoperation rate for DC-type HFS is reportedly higher than that for the pure distal compression type.[14] Even if typical compression is found in REZ, we should be aware that there might be another compression even in CP.[16]

AMR has been used in MVD for HFS to assess whether adequate decompression has been achieved. Intraoperative AMR monitoring exhibits high specificity for predicting a spasm-free status after MVD. AMR is also useful in cases of compression in CP. Zheng *et al.* reported that if AMR had not been used, at least 17 of 20 cases of multi-compression would have been missed.[16] In our review, the disappearance of AMR was confirmed in all patients who underwent nerve decompression in both REZ and CP [Table 1]. Hirono *et al.* reported that AMR exhibited dynamic and diverse changes, such as transient, sudden, gradual, or componential disappearance during decompression.[4] However, to the best of our knowledge, exacerbation of the amplitude of AMR was only mentioned in one report.[7] The authors summarized 13 patients with no improvement or worsening of HFS after initial MVD. Among them, AMR disappeared once but then recurred at the same or a higher amplitude in seven patients. In our case, AMR amplitude increased after the translocation

of VA in REZ, and it immediately disappeared after the translocation of AICA following VA translocation in CP. These phenomena suggested that the transposition of VA as the suspected culprit vessel in REZ resulted in further compression of the facial nerve in CP via AICA. AMR might have keenly facilitated the detection of intraoperative change in the degree of nerve compression. The principle of leverage might induce these phenomena. In our case, AMR did not disappear but rather amplified after the transposition of VA in REZ, which was initially considered to be the compression point. Therefore, we could suspect that AICA in CP being compressed by VA was the direct culprit vessel. Pre- and intraoperative diagnoses and identification of the culprit vessel are difficult, especially in DC-type HFS. Therefore, AMR is mandatory to confirm the identity of culprit vessels in such cases. Furthermore, AMR is useful to detect any change in the compression status due to anatomical changes of vessels by intraoperative manipulations.

## CONCLUSION

In DC-type HFS, decompression of the vessel on one side may exacerbate compression on the other side as a lever, especially in cases with thick and atherosclerotic vessels. AMR is useful to indicate the existence of a culprit vessel in CP that may have been preoperatively overlooked. It is also helpful to recognize anatomical changes regarding the relationship between nerves and vessels.

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### Ethical approval

All procedures performed in this study involving human participants were conducted in accordance with ethical standards of the Institutional and/or National Research Committee and the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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### Conflicts of interest

There are no conflicts of interest.

### Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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