Discovery of a new waveform for intraoperative monitoring of hemifacial spasms

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Abstract

Background Surgeons often rely on intraoperative electrophysiological monitoring to determine whether decompression is sufficient during microvascular decompression surgery for hemifacial spasms. A new monitoring method is needed when an abnormal muscle response is occasionally not available or is unreliable. This study was an observational clinical trial exploring a new waveform recorded from the facial muscles while the offending artery wall was electrically stimulated.

Methods Thirty-two patients with typical hemifacial spasm and 12 with trigeminal neuralgia as a control were included. The facial muscle response was recorded during microvascular decompression surgery while the offending artery wall was stimulated (2 mA×0.2 ms). The latency, amplitude, and effective refractory period were analyzed.

Results A waveform was recorded from the facial muscles of patients with hemifacial spasm when the offending artery wall was stimulated and was named the "Z-L response." The latency was 7.3±0.8 ms, the amplitude was 0.08±0.02 mV, and the effective refractory period was 3.5–4 ms. The Z-L response disappeared immediately after microvascular decompression. No waveform was recorded from the facial muscles of patients with trigeminal neuralgia while the anterior inferior cerebellar artery, which adheres to the facial nerve, was stimulated (2 mA×0.2 ms).

Conclusion We found a new waveform for intraoperative monitoring of hemifacial spasm. The Z-L response was useful when the abnormal muscle response was absent before decompression or persisted after all vascular compressions were properly treated. Particularly, the Z-L response may help neurosurgeons determine the real culprit when multiple offending vessels exist.

Keywords Hemifacial spasm · Facial nerve · Offending artery · Waveform

Introduction

Hemifacial spasm (HFS) is a neuromuscular disorder characterized by frequent, involuntary facial muscle contractions, resulting in impaired quality of life. HFS results from vascular compression of the seventh cranial nerve (the facial nerve), most likely at its root exit zone (REZ) [2, 9]. The compression is caused by such arteries as the anterior inferior cerebellar artery (AICA), posterior inferior cerebellar artery (PICA), or vertebral artery (VA) [2, 9, 15]. Microvascular decompression (MVD) is the standard surgical solution for this disorder [5, 15, 23]. Successful spasm relief after MVD ranges from 84 to 95% [3, 13].

Electrical stimulation of one branch of the facial nerve on the affected side can elicit a delayed response from the muscles supplied by other branches in patients with HFS, which has been called an “abnormal muscle response”
(AMR), also known as a lateral spread response. The presence of an AMR has been documented only in patients with HFS [22], so it is useful for intraoperative monitoring during MVD surgery [16, 22, 24]. Since Moller and Janetta [16] reported their AMR electrophysiological findings, many authors [4, 18, 26] have suggested that intra-operative monitoring of the AMR is useful for identifying culprit vessels and for confirming whether the facial nerve has been completely decompressed. According to a meta-analysis that included 855 cases [21], the chance of a cure if the AMR is abolished during surgery was 4.2-times greater than if the AMR persisted. Nevertheless, some authors [11, 12] have argued that the value of intra-operative AMR monitoring is questionable because some patients in a non-AMR-disappeared group obtained good spasm relief after MVD, and some patients in an AMR-disappeared group had persistent spasms. Furthermore, sometimes no AMR may be induced at all. Thus, an alternative method of intra-operative electrophysiological monitoring may help surgeons make a decision when AMR is unavailable or reliable. Ishikawa et al. [6–8] reported the use of F-waves during MVD operations for HFS. However, the F-wave is not specific to HFS. In fact, F-waves occur in normal control subjects, on the normal side of patients with HFS, and on the spasm side after decompression [8]. Therefore, we do not believe that an F-wave is a very good candidate.

We designed and conducted an observational clinical trial (ClinicalTrial.gov Registration number: NCT01271634) to develop a new intraoperative monitoring method by recording the facial muscle response while the offending arterial wall was electrically stimulated.

Material and methods

Patients

This observational clinical trial was approved by the ethics committee of XinHua Hospital, affiliated with Shanghai Jiaotong University School of Medicine. All patients gave informed consent according to the Declaration of Helsinki (BMJ 1991; 302: 1194).

The trial recruited 32 patients with typical HFS and 12 patients with trigeminal neuralgia (as controls), all of whom underwent MVD surgery at the Cranial Nerve Disease Center of Shanghai. Typical HFS refers to a spasm that starts from the orbicularis occuli muscle and gradually progresses downward to involve the orbicularis oris muscle, buccinator muscle, and/or the platysma [10, 20]. This is different from an atypical HFS in which the spasm starts with the orbicularis oris and buccinator muscles and gradually spreads upward to involve the orbicularis occuli muscle. MVD procedures were carried out following previously described methods [14]. Intraoperative AMR was monitored as previously reported [14, 17] using an evoked potential system (Medtronic Keypoint 4, Dantec, Denmark), in which the stimulation intensity ranged from 10 to 30 mA. AMR was recorded from the mentalis muscle by electrically stimulating the temporal branch of the facial nerve to set a conduction time reference. The latency of the AMR was shorter than that of ZLR. When stimulation of the offending artery was increased to 10 mA, a giant waveform emerged similar to the AMR, with a latency of about 5 ms.

Fig. 1 Intraoperative electrophysiological monitoring findings during microvascular decompression surgery for hemifacial spasm. a Typical abnormal muscle response (AMR) was recorded before decompression. b AMR disappeared immediately after complete decompression. c The Z-L response (ZLR) is a waveform recorded from the facial muscles when stimulating the offending artery wall (2 mA × 0.2 ms). This waveform looks like an AMR but has a shorter latency. d The ZLR disappeared immediately after complete decompression. e The root exit zone (REZ) response (REZ-R) is a giant waveform recorded from the facial muscles when stimulating the REZ of the facial nerve to set a conduction time reference. The latency of the REZ-R was shorter than that of ZLR. f When stimulation of the offending artery was increased to 10 mA, a giant waveform emerged similar to the REZ-R, with a latency of about 5 ms.

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Recording a new waveform during MVD surgery

The same reference electrodes and recording electrodes were used to record the new waveform as were used for AMR recording. Briefly, the needle reference electrodes were inserted into the frontal muscle, and the needle recording electrodes were inserted into the orbicularis oculi, orbicularis oris, and mentalis muscles. However, the stimulating electrode was a non-invasive concentric electrode, which was used intracranially.

The entire course of the facial nerve and the offending arteries were exposed under a surgical microscope during the procedure.
MVD surgery. Before detaching the offending artery from the facial nerve, the stimulating electrode was placed on the offending artery wall near the compression site (within 5 mm), a square impulse (2 mA × 0.2 ms) was delivered, and the facial muscle response was recorded with the Medtronic Keypoint 4 system using the “F-Response” mode. This recording procedure was repeated in the same way after complete decompression of the facial nerve with Teflon sponges. Subsequently, the facial muscle response was recorded while the facial nerve root exit zone (REZ) was stimulated with the same parameters to provide a latency reference. As a control, the same recording procedure was performed in 12 patients with trigeminal neuralgia by stimulating the AICA, which is in close contact with the facial nerve.

After the new waveform was detected, its latency and amplitude were measured and compared with that of the AMR. At least five waveforms were measured from each patient to calculate the mean. Additionally, we also determined the effective refractory periods (ERPs) of the new wave, the AMR, and the facial nerve following the “double stimulation” method previously reported by Yamashita et al. [25] with interstimulus intervals of 0.5–7.0 ms.

**Results**

AMR (Fig. 1a) was found in 29 patients with HFS, except for cases 27–29. The AMR latency was 10.7±0.5 ms (Table 1). The new waveform was recorded from the orbicularis oculi, orbicularis oris, and mentalis muscles while stimulating the offending artery wall (Fig. 1c). For convenience of discussion, this new waveform was named the “Z-L response” (ZLR). The ZLR looked similar to an AMR. The ZLR latency was 7.3±0.8 ms (Table 1), which was significantly shorter than the AMR latency (P<0.01, Student’s t-test).

![Fig. 2](https://example.com/f2.png) An abnormal muscle response (AMR) was absent in this patient (case 27) during the entire operation. The root exit zone (REZ) of the facial nerve was compressed by two branches of the anterior inferior cerebellar artery (AICA). The Z-L response (ZLR) was identified from branch 1 (a, e) but not from branch 2 (b). The ZLR disappeared after decompression (c, d, f). The patient achieved immediate “excellent” resolution of spasms after surgery.

We also recorded a waveform from the facial muscles while stimulating the facial nerve REZ to set a reference for the facial nerve conduction time. This giant waveform was referred to as the “REZ response” (REZ-R) (Fig. 1e). The REZ-R latency was 5.0±0.1 ms (Table 1), which was significantly shorter than the ZLR latency (P<0.01, Student’s t-test). Thus, the ZLR latency was longer than that of the REZ-R but shorter than that of the AMR.

As all parameters except for the stimulating site were the same when recording the ZLR and REZ-R, we think that the ZLR and REZ-R amplitudes are comparable. The ZLR amplitude was 0.08±0.02 mV (Table 1), which was only approximately one-fifth of the REZ-R amplitude (0.44±0.09 mV, Table 1), and the difference was significant (P<0.01, Student’s t-test).

When stimulation of the offending artery wall was increased to 8–10 mA, a 5-ms-latency giant waveform (Fig. 1f) emerged. Although its stimulating site was the same as the ZLR, its wave shape and latency were more like the REZ-R than the ZLR.

The new waveform was recorded from the orbicularis oculi, orbicularis oris, and mentalis muscles while stimulating the offending artery wall (Fig. 1c). For convenience of discussion, this new waveform was named the “Z-L response” (ZLR). The ZLR looked similar to an AMR. The ZLR latency was 7.3±0.8 ms (Table 1), which was significantly shorter than the AMR latency (P<0.01, Student’s t-test).

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When stimulation of the offending artery wall was increased to 8–10 mA, a 5-ms-latency giant waveform (Fig. 1f) emerged. Although its stimulating site was the same as the ZLR, its wave shape and latency were more like the REZ-R than the ZLR.
After complete decompression of the facial nerve, the AMR and ZLR disappeared simultaneously in 24 patients (cases 1–24; Table 1; Fig. 1b, d), and they achieved good or excellent surgical outcomes (Table 1).

When the offending arteries were decompressed in two patients (cases 25 and 26), the ZLR disappeared immediately, but the AMR persisted. After repeated thorough exploration of the entire course of the facial nerve, no more neurovascular conflicts were found; thus, we determined that the decompression was sufficient. After surgery, these two patients obtained immediate and complete spasm relief (Table 1).

The AMR was absent in three patients (cases 27–29) during the whole operation course due to unknown reason. However, ZLR was successfully induced, and disappeared after decompression. These patients achieved excellent or good outcomes (Table 1).

In another three cases (cases 30–32), a typical AMR was detected at the beginning but disappeared when we dissected the arachnoid mater and elevated the cerebellum (before the neurovascular compression was exposed). ZLR was not induced for these three patients, probably because the neurovascular contact had changed slightly. Thus, MVD for these three patients was performed without electrophysiological reference, and one patient failed the surgery (Table 1).

Multiple compressions were found in three cases (nos. 23, 24, and 27). The AMR was not available in case 27; we found that two branches of the AICA compressed the REZ. ZLR was recorded from one branch but not from
tion (Fig. 2). In case 24, we found that the PICA was the other branch. The ZLR disappeared after decompression. In case 22, we found that the PICA was compressing the cisternal portion of the facial nerve. ZLR was detected from the AICA but not from the PICA. First, we transposed the PICA with Teflon sponges, but the AMR and ZLR did not disappear. Then, we decompressed the AICA, and the AMR and ZLR vanished immediately (Fig. 3). In case 23, the AICA compressed the cisternal portion, and the VA compressed the REZ. A ZLR was identified from the AICA but not from the VA. The AMR and ZLR were eliminated only after the AICA was decompressed.

ERPs were detected in five patients using double stimulation (Table 2). The ERP of ZLR and that of AMR were 3.5–4 ms, whereas the ERP of REZ-R was only 1.5–2 ms. To test the specificity of ZLR in HFS during the MVD surgery for trigeminal neuralgia in 12 patients, we stimulated (2 mA×0.2 ms, within 5 mm of the adhesion site) the AICA, which adhered to the facial nerve, but no waveform was recorded from the facial muscles. However, a 5-ms-latency giant waveform similar to a REZ-R emerged when the intensity of arterial stimulation was increased to 8–10 mA, indicating that without the pathophysiological basis of HFS abnormal cross-transmission, ZLR cannot be induced even if an artery adheres to the facial nerve.

Discussion

As HFS is not life-threatening, it is very difficult for patients to decide whether to undergo a posterior fossa operation; hence, they are extremely unwilling to accept the possibility of a single failed surgery. Thus, although the curative effect of MVD (84–95%, immediate plus delayed resolution) seems satisfactory, there is still much room for improvement. However, new technology is needed to further improve the curative effect. Our findings indicate that the combination of AMR and ZLR provides more useful information than does the AMR alone. In most cases in this series, ZLR was consistent with AMR, but surgeons had to rely on the ZLR to confirm that the facial nerve was sufficiently decompressed in five patients (cases 25–29). We suppose that ZLR is useful in those patients whose AMR was absent from the beginning or persisted after the offending artery was transposed and in those whose AMR disappeared for a while but reappeared when the posterior cranial fossa was refilled with physiological saline.

We found that when more than one artery compressed the facial nerve, only one artery was “ZLR-positive,” but the AMR disappeared (cases 23 and 24) only when the ZLR-positive vessel was detached. Thus, we suppose that ZLR might help neurosurgeons to determine the real culprit when multiple offending vessels exist.

The essential of AMR is different branches of facial nerve for stimulating and recording. However, the stimulation of ZLR is not on a “branch” of the facial nerve, so it is basically different from the AMR. The F-wave is a waveform that always follows a direct compound muscle potential (M-wave) [7, 8]. In contrast, no M-wave formed prior to the ZLR, so the ZLR is not a variant of the F-wave. Therefore, we consider the ZLR a novel waveform. The exact circuit for ZLR is unknown. We propose two hypotheses. One hypothesis is that the electrical stimulus of the arterial wall may directly spread to the facial nerve and activate it; a small amount of current (2 mA) might only depolarize some of the slower conducting fibers (latency, 7.3 ms), whereas when more current (10 mA) reaches the nerve, faster conducting fibers are depolarized (latency, 5.0 ms). The other hypothesis is that the sympathetic fibers on the surface of the arterial wall [1] might connect to the demyelinated facial nerve fibers; thus, stimulating the arterial wall activates sympathetic fibers, and action potentials pass from sympathetic fibers to facial nerve fibers via ephaptic transmission. However, morphological evidence should be obtained by autopsy to confirm this hypothesis.

Our findings revealed that the ERP of ZLR and that of AMR are much longer than that of REZ-R, indicating that the ZLR and AMR circuits may share a part of a longer ERP than that of the facial nerve fibers.

Conclusions

We found a new waveform for intraoperative monitoring of HFS. Z-L response monitoring is useful when an AMR is absent before decompression or persists after all vascular

### Table 2 Effective refractory periods of the AMR, Z-L, and REZ responses

<table>
<thead>
<tr>
<th>Case no.</th>
<th>ERP of AMR (ms)</th>
<th>ERP of Z-L response (ms)</th>
<th>ERP of REZ response (ms)</th>
</tr>
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<tbody>
<tr>
<td>6</td>
<td>4</td>
<td>4</td>
<td>1.5</td>
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<tr>
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<tr>
<td>19</td>
<td>4</td>
<td>4</td>
<td>2</td>
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</table>

AMR: stimulating the zygomatic branch of the facial nerve and recording from the mentalis muscle

Z-L response: stimulating the offending arterial wall and recording from the mentalis muscle

REZ response: stimulating the REZ of facial nerve and recording from the mentalis muscle

ERP effective refractory period, AMR abnormal muscle response, REZ root entry zone
compressions are properly treated. Particularly, the Z-L response may help neurosurgeons determine the real culprit when multiple offending vessels exist.

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Conflicts of interest  None.

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