Intraoperative monitoring of the facial nerve during decompressive surgery for hemifacial spasm

STEPHEN J. HAINES, M.D., AND FERNANDO TORRES, M.D.

Departments of Neurosurgery and Neurology, University of Minnesota Health Science Center, Minneapolis, Minnesota

In 11 consecutive patients, intraoperative electromyographic (EMG) recordings were made from the facial muscles during microvascular decompression for hemifacial spasm. In one patient, recordings could not be obtained for technical reasons, and two patients had no abnormality. In the remaining eight patients, the abnormal response resolved before decompression in two, resolved immediately at the time of decompression in five, and failed to resolve in one. All patients were relieved of their hemifacial spasm. In the five patients whose abnormalities resolved at the time of decompression, there was a precise intraoperative correlation between decompression of the nerve and disappearance of the abnormal EMG response. In three cases, this was a useful guide to the need to decompress more than one vessel. These results confirm the findings of Moller and Jannetta, support the use of this technique for intraoperative monitoring of facial nerve decompression procedures, and provide strong circumstantial evidence that vascular cross-compression is an important etiological factor in hemifacial spasm.

KEY WORDS • hemifacial spasm • intraoperative monitoring • electromyography • facial nerve

MOLLER and Jannetta have reported a series of observations on intraoperative facial electromyographic (EMG) recordings obtained during microvascular decompression for hemifacial spasm. These observations have suggested that this method of monitoring provides a useful way of confirming complete facial nerve decompression intraoperatively. In addition, it shows evidence that the actual vascular compression of the nerve is an important factor in the production of hemifacial spasm. To our knowledge, independent confirmation of these observations has not been recorded. In this paper we report our experience with this technique in 11 patients.

Clinical Material and Methods

The technique is essentially that described by Moller and Jannetta. Anesthesia was induced with thiopental, and a single dose of a short-acting neuromuscular blocking agent (vecuronium bromide) was given. Stimulating needle electrodes were inserted intradermally over the marginal mandibular and temporal branches of the facial nerve. Recording needle electrodes were inserted into the mentalis muscle and the orbicularis oculi muscle on the same side. The absence of muscle relaxant was confirmed with a peripheral nerve stimulator. Square-wave stimuli of 100 μsec duration and a 3- to 5-mAmp current at a rate of 5.1 stimuli/sec were applied alternately to the marginal mandibular and the temporal branches of the facial nerve on the side of hemifacial spasm. Bipolar stimulation was performed with the two electrodes about 1 cm apart. Electromyographic potentials were recorded from both the orbicularis oculi and mentalis muscles with each stimulus. Recording was bipolar, with an interelectrode distance of about 1 cm. The recording electrodes were located at a distance of about 4 cm from the stimulating electrodes. Recordings were made at each stage of the decompressive operation.

Results

Recording has been attempted in 11 patients (Table 1). In one patient, equipment problems prevented satisfactory recording. In two patients, orthodromic responses to stimulation were recorded but no abnormal antidromic response could be identified. In the remaining eight patients, the abnormal response described by Moller and Jannetta was present. This consisted of a response in the muscles supplied by the unstimulated branch of the facial nerve. The latency between the stimulus and the response of the antidromically stimulated muscle is approximately 13 msec. In two patients, the abnormal response disappeared prior to decompression of the nerve: this occurred upon opening
Facial nerve monitoring in hemifacial spasm surgery

**TABLE 1**

Results of intraoperative neurophysiologic recording

<table>
<thead>
<tr>
<th>Result</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>technical failure</td>
<td>1</td>
</tr>
<tr>
<td>normal response</td>
<td>2</td>
</tr>
<tr>
<td>resolution of abnormal response</td>
<td></td>
</tr>
<tr>
<td>before decompression</td>
<td>2</td>
</tr>
<tr>
<td>after decompression</td>
<td>5</td>
</tr>
<tr>
<td>not achieved</td>
<td>1</td>
</tr>
</tbody>
</table>

the dura in one and after gentle cerebellar retraction was begun in the other. In one patient, the abnormalities did not disappear despite decompression of all visible compressing vessels. In the remaining five patients, the abnormality did not disappear until a complete microvascular decompression of the facial nerve had been accomplished. In three of these five cases, more than one vessel was cross-compressing the facial nerve, and complete disappearance of the abnormal EMG potentials did not occur until all vessels had been decompressed. Figure 1 shows progressive disappearance of the abnormal response as each of four compressing vessels were lifted off the facial nerve in one of these patients. Figures 2, 3, and 4 depict tracings correlated with intraoperative photographs showing compressing vessels. In several cases, the vessel was repeatedly separated from the nerve and then allowed to return to its original position. The abnormal responses disappeared immediately with decompression and returned each time the vessel was replaced (Fig. 5).

Hemifacial spasm has been relieved in all patients with a follow-up period of 3 months to 3 years. Two patients have complained of transient episodes of mild twitching in the orbicularis oculi muscle which has resolved spontaneously. The one patient in whom the abnormal response did not disappear, despite apparently adequate decompression, had persistent hemifacial spasm for 6 days postoperatively. The spasm then resolved completely and has remained absent. All other patients were free of spasm immediately after surgery.

**Discussion**

We have confirmed the findings of Moller and Janetta that, in patients with hemifacial spasm, there is

**FIG. 1.** Intraoperative electromyographic recordings from the orbicularis oculi muscle during stimulation of the marginal mandibular branch of the facial nerve. Progressive disappearance of the abnormal response of 1.3-msec approximate latency is shown as the operation progresses and all vessels compressing the facial nerve are mobilized.

**FIG. 2.** Upper: Intraoperative electromyographic recording after dural opening but before facial nerve decompression for hemifacial spasm. Lower: Drawing (left) and operative photograph (right) illustrate the compressing vessels as seen through the operating microscope.
an abnormality in the facial nerve or its nucleus which allows the stimulus applied to one branch of the facial nerve to spread to other branches of that same nerve. In most cases, this abnormality disappears immediately upon microvascular decompression of the facial nerve as it exits the brainstem. Møller and Jannetta have speculated that the vascular compressive lesion produces antidromic activity which alters the excitability of the facial nucleus. They believe that the fact that lateral spread is also seen after stimulation of the supraorbital nerve further implicates the facial nucleus in the disease. While the present observations do not provide

Fig. 3. Upper: Intraoperative electromyographic recording after mobilization of one of two cross-compressing vessels. There is actually an increase in the amplitude of the abnormal potential. Lower: Drawing (left) and operative photograph (right) showing the vessel held away from the facial nerve with a microdissector.

Fig. 4. Upper: Intraoperative electromyographic recording after nerve decompression is completed. No abnormality persists although the orthodromic response in the orbicularis oculi muscle is unchanged. Lower: Drawing (left) and operative photograph (right) showing both vessels held away from the nerve.
Facial nerve monitoring in hemifacial spasm surgery

The data presented by Møller and Jannetta\(^5\) suggest that failure to relieve the neurophysiological abnormality during surgery is associated with a relatively poor prognosis for relief of hemifacial spasm. In our patient in whom the abnormality was not relieved intraoperatively, although her spasm has resolved, it persisted unusually long postoperatively. It is too early to know if this relief will be long-lasting. This finding is consistent, however, with an association between the electrophysiological and clinical findings. We believe that these observations justify the use of this technique as a method for monitoring the completeness of decompression during microvascular decompression for hemifacial spasm. Further support to justify the use of this technique is given by the finding that in three of our cases it was a useful guide to the need to decompress more than one vessel.

Acknowledgments

We would like to acknowledge the secretarial assistance of Susan Whaley and technical assistance of Connie Erickson, R.EEGT, in obtaining the reports.

References


Manuscript received March 27, 1990. Accepted in final form August 13, 1990. Address reprint requests to: Stephen J. Haines, M.D., Department of Neurosurgery, 420 Delaware Street SE, Box 96 UMHC, Minneapolis, Minnesota 55455.