Loss of auditory function in microvascular decompression for hemifacial spasm

Results in 143 consecutive cases

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Auditory function was studied before and after surgery in 143 consecutive patients who were operated on for hemifacial spasm by microvascular decompression of the intracranial portion of the facial nerve. The acoustic middle ear reflex was abnormal preoperatively in 41% of the patients, indicating that the vascular abnormalities that caused the hemifacial spasm also affected the auditory nerve. Three patients suffered a profound hearing loss in the ear on the operated side, and one lost hearing function totally. In addition, 24 patients had a moderate elevation in the pure-tone threshold at one or more octave frequencies. Of these, 16 patients experienced a hearing loss at only one frequency (8000 Hz), while eight had a threshold elevation of no more than 20 dB in the speech frequency range (500, 1000, and 2000 Hz). Two patients were deaf on the side of the spasm before the operation. Three patients were not tested postoperatively, and one patient was tested only after surgery. Thus, in this series of 143 patients, only 2.8% suffered a significant hearing loss as a complication of facial nerve decompression to relieve hemifacial spasm.

KEY WORDS • hemifacial spasm • microvascular decompression • auditory nerve • hearing loss

Classical hemifacial spasm is a disorder characterized by a hyperactive dysfunction of the facial nerve leading to progressive involuntary twitching of the muscles of the face. The spasm is most often unilateral, occurs more commonly in women than in men, and affects the left side more often than the right.

There is now extensive evidence that the spasm is usually caused by cross compression of the seventh cranial nerve at its root entry zone, and a study of a large series of patients has shown that microvascular decompression of the seventh cranial nerve at its root entry zone can result in permanent relief of the symptoms. It has been proposed that this cross compression causes crosstalk at the site of the lesion and that this crosstalk causes the spasm and the synkinesis. Another hypothesis suggests that the facial nucleus is involved. Recent electrophysiological studies seem to favor the latter hypothesis.

The close anatomical relationship between the facial and auditory nerves makes it likely that a vascular abnormality compressing the facial nerve might also influence the auditory nerve and cause measurable changes in auditory function. It is known, particularly from studies of patients with acoustic neuromas, that injuries to the auditory nerve not only cause elevation of the pure-tone threshold but also result in elevation of the threshold of the acoustic middle ear reflex and in changes in the pattern of auditory brain-stem responses. Also, it has recently been shown that in a large number of patients with hemifacial spasm the pure-tone threshold and the threshold of the acoustic middle ear reflex are elevated (MB Møller and AR Møller, in preparation, 1985). In addition, patients with hemifacial spasm show changes in the auditory brain-stem responses similar to those observed in patients with acoustic nerve tumors. Vascular abnormalities which affect the vestibular nerve in such a way as to cause vertigo have also been shown to affect auditory function.

Since hearing loss is a potential complication of microvascular decompression operations involving the facial nerve, we routinely test hearing pre- and postoperatively. We present here our findings in 143 consecutive patients who underwent this operative procedure to relieve hemifacial spasm.
Summary of Cases

From June, 1980, to December, 1983, 143 patients with hemifacial spasm underwent microvascular decompression at this institution. We have included in this study only those patients who had not had previous surgery to relieve the spasm. Ninety-five patients were women aged 29 to 77 years (mean age 50.6 years) and 48 were men aged 20 to 68 years (mean age 51.7 years). Eighty-four patients had spasm on the left side and 59 had spasm on the right side. One patient was operated on in 1980 for spasm on the left side and then 2 years later developed spasm on the right side for which she underwent surgery in 1982.

The microvascular decompression procedure used in this series has been described previously. The cause of hemifacial spasm was confirmed at operation to be cross compression from either arteries or veins in all the patients included in this study.

Auditory Testing

The hearing test included the determination of pure-tone thresholds using standard audiometric techniques (air and bone conduction for octave frequencies 250 to 8000 Hz), speech reception threshold and speech discrimination scores for each ear, recorded standardized speech materials, and thresholds for the crossed acoustic middle ear reflex response obtained at 500, 1000, and 2000 Hz. Otoneurological evaluation was conducted in all patients prior to the operation, with special emphasis on detection of such symptoms as tinnitus, vertigo, dysequilibrium, presence of middle ear pathology, and history of noise exposure. The hearing tests as well as the otoneurological evaluation were repeated 5 to 10 days after the operation and again at a 3- and/or 6-month follow-up examination.

We have previously described a technique for monitoring auditory functions during microvascular decompression operations on the facial nerve at its root entry zone to relieve hemifacial spasm. In this procedure, compound action potentials are recorded directly from the eighth cranial nerve during the intradural part of the operation and auditory brain-stem evoked potentials are recorded from scalp electrodes before the dura is opened. Direct monitoring of auditory compound action potentials was introduced in October, 1982, and was performed in 39 of the patients included in the present study.

Pure-Tone and Speech Audiometry

Figure 1 shows the distribution of the differences between pre- and postoperative pure-tone thresholds for six frequencies. Three patients experienced a pronounced hearing loss as a consequence of the surgery. These patients' audiograms are shown in Fig. 2. One patient suffered a complete loss of hearing on the operated side (audiogram not shown). Two patients were deaf on the affected side (spasm side) prior to the operation, and one patient was deaf on the unaffected side. Some of the patients in the higher age range had varying degrees of hearing loss at high frequencies (presbyacusis) and some had noise-induced hearing loss. Both conditions were associated with symmetrical hearing loss in both ears. Only three patients were not evaluated postoperatively, but none of them had no-
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ticed any subjective hearing loss at discharge from the hospital. One patient underwent only postoperative testing which showed normal hearing. Thus, there were 137 patients with preoperative hearing who had complete pre- and postoperative testing.

As a result of the operation, 24 patients had a decrease in hearing postoperatively, amounting to a 15-dB or greater increase in hearing threshold at one or more octave frequencies. (A change of 10 dB or less is within the accepted test-retest variability.) Sixteen of these patients had hearing loss only at the 8000-Hz frequency level. In eight of the 24 patients the hearing threshold was increased by 15 to 20 dB at one or more frequencies within the speech frequency range (500, 1000, and 2000 Hz). Ten patients had an elevation of their threshold at 250 Hz by 15 dB or more. In three patients the pure-tone thresholds in fact improved postoperatively by 15 dB in the speech frequency range, and seven patients had an improvement in their pure-tone thresholds at 8000 Hz of 15 dB or more.

An effusion may often be noted in the middle ear after posterior fossa operations, and leads to a conductive hearing loss. In patients who had middle ear effusions after the operation we used the bone conduction threshold as the measure of the postoperative pure-tone hearing threshold. Speech discrimination did not change significantly as a result of the operation except in the four patients who suffered a profound hearing loss. Thus, in this series of 143 consecutive patients who underwent the procedure, only four (2.8%) suffered a significant hearing loss as a complication of microvascular decompression to relieve hemifacial spasm. Of the 39 patients in whom intraoperative monitoring of auditory function was performed using direct recording from the eighth nerve, only one patient suffered a profound hearing loss as a complication of the operation. The change in hearing in those patients in whom this type of intraoperative monitoring was used is shown by shaded areas of the histograms in Fig. 1.

**Acoustic Middle Ear Reflexes**

Preoperatively, the threshold of the acoustic middle ear reflex, when elicited from the spasm side, was normal in 81 patients; the responses were elevated or absent in 56 patients. When the reflex was elicited from the unaffected side and the response was recorded in the ear on the side of the spasm, 77 patients had normal thresholds and 60 had elevated thresholds or absent responses, indicating that this parameter had been affected by the facial nerve dysfunction. In six patients, the acoustic middle ear reflex could not be evaluated because the patients were unilaterally deaf (three patients) or had middle ear pathology (three patients).

**Discussion**

In this series of 143 consecutive patients, only four (2.8%) had a pronounced hearing loss as a result of microvascular decompression for hemifacial spasm. One of these patients lost his hearing completely on the operated side, and three other patients had varying degrees of hearing loss on pure-tone audiometry but with severely reduced speech discrimination scores (Fig. 2). This reduction in speech discrimination is consistent with a pronounced loss of hearing. Three of these patients who suffered hearing loss did not undergo intraoperative monitoring of nerve compound action potentials and the fourth patient, despite monitoring during the operation, lost his hearing instantaneously during decompression of the facial nerve.

The 16 patients whose hearing declined 15 dB or more at a frequency of only 8000 Hz experienced no subjective hearing loss, since an elevation of the hearing threshold above 4 kHz has very little effect on speech discrimination both in quiet surroundings and against background noise. Most of the 10 patients whose hearing had decreased postoperatively at 250 Hz noticed a moderate change in hearing and complained of a

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**FIG. 2.** Pre- and postoperative audiograms on the side of the spasm of three patients who suffered hearing loss after microvascular decompression. Recordings in a 52-year-old man with right hemifacial spasm (upper), a 48-year-old man with left hemifacial spasm (center), and a 68-year-old woman with left hemifacial spasm (lower). PTA = pure tone average; SRT = speech reception threshold; MASK = non-tested ear covered by a masking noise to insure testing of the other ear only; DISCR = discrimination; LE = left ear; RE = right ear; CNT = could not test.
change in sound, despite the fact that speech discrimination remained the same as it had been preoperatively. It is of interest that postoperatively in three patients hearing had improved in the mid-frequency range, whereas in seven patients hearing improved 15 dB or more at 8000 Hz. These changes in hearing threshold may reflect a true improvement in hearing. Many patients with hemifacial spasm also have tinnitus on the affected side, which can influence hearing and which is often relieved by the operation. Postoperative headaches and tiredness, as well as tenseness prior to the operation, could also have caused hearing thresholds to vary more than in a normal population.

The fact that the threshold of the acoustic middle ear reflex was elevated or absent before the operation in 56 patients (41%), whereas 81 patients had a normal threshold in the ear on the side of the spasm, is a further indication that the vascular compression causing hemifacial spasm also causes objective changes in the auditory nerve. This is to be expected, because the afferent pathways of the acoustic middle ear reflex involve the primary auditory nerve fibers, neurons of the ventral cochlear nucleus, and the superior olivary complex, while the efferent pathways are the ipsi- and contralateral motoneurons of the nucleus of the facial nerve and of the facial nerve itself.

It is possible to distinguish with great sensitivity between cochlear and auditory nerve lesions by examining recordings of the acoustic middle ear reflex, because the threshold of the reflex is elevated when a lesion is in the auditory nerve but is normal in patients whose lesions are cochlear in origin. Thus, 70% to 100% of patients with cerebellopontine angle tumors have elevated or absent middle ear acoustic reflex responses, depending upon the criteria used. We consider a threshold higher than 95 dB at any one of the test frequencies to be abnormal and indicative of eighth nerve involvement. Forty-one percent of patients with hemifacial spasm and the facial nucleus. References

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