Hemifacial spasm caused by vascular compression of the distal portion of the facial nerve

Report of seven cases

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It is generally accepted that hemifacial spasm (HFS) and trigeminal neuralgia are caused by compression of the facial nerve (seventh cranial nerve) or the trigeminal nerve (fifth cranial nerve) at the nerve’s root exit (or entry) zone (REZ); thus, neurosurgeons generally perform neurovascular decompression at the REZ. Neurosurgeons tend to ignore vascular compression at distal portions of the seventh cranial nerve, even when found incidentally while performing neurovascular decompression at the REZ of that nerve, because compression of distal portions of the seventh cranial nerve has not been regarded as a cause of HFS. Recently the authors treated seven cases of HFS in which compression of the distal portion of the seventh cranial nerve produced symptoms. The anterior inferior cerebellar artery (AICA) was the offending vessel in five of these cases. Great care must be taken not to stretch the internal auditory arteries during manipulation of the AICA because these small arteries are quite vulnerable to surgical manipulation and the patient may experience hearing loss postoperatively.

It must be kept in mind that compression of distal portions of the seventh cranial nerve may be responsible for HFS in cases in which neurovascular compression at the REZ is not confirmed intraoperatively and in cases in which neurovascular decompression at the nerve’s REZ does not cure HFS. Surgical procedures for decompression of the distal portion of the seventh cranial nerve as well as decompression at the REZ should be performed when a deep vascular groove is noticed at the distal site of compression of the nerve.

KEY WORDS • neurovascular compression • distal compression • facial nerve • hemifacial spasm

EMIFACIAL spasm (HFS) and trigeminal neuralgia are generally accepted as being caused by compression of the facial and trigeminal nerves (seventh and fifth cranial nerves, respectively) by blood vessels located at these nerves’ root exit or root entry zones (REZs).9,10 Neurosurgeons have performed decompression at the REZs of the fifth or seventh cranial nerve in such patients while ignoring vascular compression at more distal portions, even when noted as an incidental finding during surgery. This is particularly true in cases of HFS because the seventh and eighth cranial nerves are much more vulnerable to surgical manipulation than the fifth nerve, and efforts are made to avoid injuring these nerves when manipulating blood vessels that compress the distal portion of the seventh cranial nerve. We performed neurovascular decompression of the seventh cranial nerve during the period between July 1974 and December 1996 to treat HFS in 142 patients. In seven of these patients (5%) compression of a distal portion of the seventh cranial nerve combined with compression at its REZ or compression of a distal portion of the nerve alone played an important role in causing the HFS. We now report on these seven patients in whom HFS resolved after neurovascular decompression of the seventh cranial nerve distal to the REZ.

Selected Case Reports

Table 1 provides a summary of the seven patients in whom HFS resolved as a result of neurovascular decompression of the seventh cranial nerve at a site distal to the REZ. Four cases are presented in detail.

Case 4

This 51-year-old woman had undergone neurovascular decompression for left-sided HFS at another hospital in August 1988, but her symptoms remained unchanged. She underwent a second neurovascular decompression via a retromastoid approach in our neurosurgery department in November 1990. Located between the eighth cranial nerve and the anterior inferior cerebellar artery (AICA) was a small cottonoid prosthesis, which was carefully dissected and removed. The AICA, which was found to
compress the seventh cranial nerve at its REZ, was lifted away by inserting a small piece of Dacron felt (Meadox Medicals, Inc., Oakland, NJ) between the AICA and the prosthesis. There were no abnormal findings other than the HFS. Neurovascular decompression was found at the REZ and both the AICA and the prosthesis remained in their position. At a very distal portion of the seventh cranial nerve, near the internal auditory canal, the distal loop of the AICA was found to compress the nerve (Fig. 2 lower left). This loop was carefully lifted away from the seventh cranial nerve and attached to adjacent dura mater with a small piece of oxicellulose soaked in Biobond (Yoshitomi Pharmaceutical Industries, Ltd., Osaka, Japan) (Fig. 2 lower right). There was a deep vascular groove at the site of seventh cranial nerve compression. The patient’s ABRs were continuously monitored, but clear responses could not be obtained because of excessive noise that could not be avoided during surgery. The patient has been completely free of HFS, without facial paresis, since surgery. However, she has developed a marked hearing loss on the side that was surgically treated.

Case 6

This 73-year-old man had experienced left-sided HFS for 1 year. Preoperative neurological examination revealed no abnormal findings other than the HFS. Neurovascular decompression was performed via a retromastoid approach in May 1991. The REZ of the facial nerve was compressed by the posterior inferior cerebellar artery (PICA), which was lifted away from the nerve by inserting a small piece of Dacron felt between the PICA and the pons. The patient’s postoperative course was uneventful; however, 3 years later the HFS recurred. Magnetic resonance imaging provided findings that suggested PICA compression of the seventh cranial nerve at its midportion. In August 1994 a second operation was performed, in which it was demonstrated that there was no vascular compression at the REZ of the seventh cranial nerve, and that a more distal loop of the PICA was compressing this nerve at its midportion in the cerebellopontine cistern. There was marked adhesion between the artery and the pons (Fig. 2 lower right). Special care was taken not to stretch the meatal loop, which sends internal auditory arteries into the internal auditory meatus. There was a deep vascular groove at the site of compression of the seventh cranial nerve. Continuous monitoring of auditory brainstem responses (ABRs) during surgery revealed no abnormal findings. The patient’s postoperative course was uneventful and he has been completely free of HFS ever since surgery.

Case 7

This 36-year-old woman had experienced atypical left-
sided HFS for 6 months before admission to the neurosurgery department. Her facial spasm originated from the musculus orbicularis oris and buccinator and gradually progressed to involve the musculus orbicularis oculi, although there was less involvement of the latter. A preoperative neurological examination revealed no abnormal findings other than the HFS. Surgery was performed in November 1995 and revealed no neurovascular compression at the REZ of the left seventh cranial nerve (Fig. 3 left). The meatal loop of the AICA was found to run parallel and very close to, but not in contact with, the seventh cranial nerve. However, the vessel did compress the posterior surface of the seventh cranial nerve near the internal auditory meatus as it passed between the seventh and eighth cranial nerves (Fig. 3 center). The artery was carefully lifted from the seventh cranial nerve and attached to adjacent dura mater with fibrin glue (Fig. 3 right). Care was taken not to stretch the internal auditory arteries arising from the meatal loop. Continuous monitoring of ABRs showed no abnormal findings during the procedure. The patient has been free of HFS, with no complications, since surgery.

Discussion

When Dandy in 1934, Campbell, et al., in 1947, and Gardner in 1962 reported that vascular compression of the fifth and seventh cranial nerves were the possible mechanisms of trigeminal neuralgia and HFS, they did not mention the sites of compression of these nerves. It was Jannetta, et al., in 1977, who clearly referred to vascular compression at the REZ of the seventh cranial nerve as the cause of HFS for the first time. They stated:

It must be emphasized that the vascular compression must be cross-compression at right angles to the nerve and must be at the root exit zone of the nerve from the brain stem. Peripheral vessels crossing the facial nerve, and vessels running parallel to and distorting the nerve, have not been shown to cause hemifacial spasm in this series. These latter vessels are left undisturbed.

The oligodendroglia that comprise the myelin sheath of each axon of the seventh cranial nerve in the brainstem are replaced by Schwann cells at the REZ, which is located within 1 mm of the site where the seventh cranial nerve emerges from the brainstem, the same as in other cranial nerves. Jannetta postulated that the REZ of the seventh cranial nerve, as well as that of the fifth cranial nerve, is the location at which the nerve is most easily irritated by mechanical stimulation such as vascular compression. His basic concept has been accepted worldwide, although there has been some controversy. Neurosurgeons perform neurovascular decompression at the REZ of the seventh cranial nerve to treat HFS, often ignoring vascular compression of this nerve at other sites, and this surgical procedure has indeed yielded excellent results.

We recently treated seven patients in whom vascular compression distal to the REZ of the seventh cranial nerve caused HFS and in whom neurovascular decompression performed at those sites resulted in resolution of the patients’ symptoms. No vascular compression was found at the REZ of the seventh cranial nerve during the first operation in two of the seven patients. In the remaining patients, neurovascular decompression performed at the REZ of the seventh cranial nerve temporarily improved symptoms, but HFS recurred after varying periods. The second (or third) operation in these patients revealed compression of the seventh cranial nerve distal to the REZ, and neurovascular decompression performed at that site resulted in complete cure of the HFS. The site of compression was the distal portion of the seventh cranial nerve.
around the internal auditory canal in two patients, and the midportion of the nerve located between the REZ and the internal auditory canal in five patients. Six of the patients had vascular compression at the anterior surface of the seventh cranial nerve, as is usually seen, and all of these had typical HFS. Only one patient (Case 7) had vascular compression at the rostral and posterior surface of the nerve near the internal auditory canal; this resulted in atypical HFS. In all seven cases the compression sites were definitely not at the REZs.

There are some reports in the ear, nose, and throat literature of HFSs being caused by compression of the extracranial portion of the seventh cranial nerve by hemangiomas and benign parotid tumors. There have been no such reports in the neurosurgical field, although neurosurgeons have surely encountered some cases of HFS that are caused by compression located at a distal portion of the seventh cranial nerve instead of at the REZ in the cerebellopontine cistern. In a few reports, it can be assumed that compression of a distal portion of the seventh cranial

Fig. 2. Case 5 (right side of patient). Intraoperative photographs reconstructed from a videotape. Upper Left: The AICA is compressing the seventh cranial nerve at its REZ (first operation). Upper Right: A small prosthesis of Dacron felt (asterisk) has been inserted between the AICA and the pons to decompress the seventh cranial nerve. Lower Left: A distal loop of the AICA is compressing the distal portion of the seventh cranial nerve around the internal auditory meatus. Lower Right: The distal loop of the AICA has been lifted away from the seventh cranial nerve and attached to the dura mater with glue. F = facial nerve; N = nerve.

Fig. 3. Case 7 (left side of patient). Intraoperative photographs reconstructed from a videotape. Left: There is no neurovascular compression at the REZ of the seventh cranial nerve (asterisk). A small branch (a) of the AICA does not touch the seventh cranial nerve, even after removal of the retractor. Center: The distal portion of the AICA is compressing the posterior aspect of the seventh cranial nerve in its distal portion (arrowhead). Right: The distal portion of the AICA compressing the seventh cranial nerve has been carefully lifted away from the nerve and attached to the dura mater with fibrin glue (arrowheads). F = facial nerve; N = nerve.
Distal compression of the facial nerve

nerve was present in some cases of HFS, despite not having been clearly described by the authors.10,13,15 We believe that the present report is the first in the neurosurgical field in which a clear description is given of HFS due to compression of a distal portion of the seventh cranial nerve rather than compression of the REZ in the cerebellopontine cistern.

The present findings demonstrate that distal compression of the seventh cranial nerve by blood vessels as well as compression at the REZ play an important causal role in HFS. The mechanism of HFS caused by compression of distal portions of the seventh cranial nerve is unknown. Partial demyelination and axonal degeneration of the seventh cranial nerve in HFS due to neurovascular compression have been reported,14 and these changes may be necessary to produce the hyperactivity of the facial motor nucleus that causes HFS.15 The same mechanism may apply to cases of HFS due to compression of a distal portion of the seventh cranial nerve. Vascular compression at distal portions of the seventh cranial nerve, however, causes HFS much more rarely than does compression at the REZ, possibly because Schwann cells are more resistant to vascular compression causing demyelination of the seventh cranial nerve.12

We believe that the REZ is the primary site of vascular compression causing HFS in most cases, as Jannetta7 postulated, and that this portion should be decompressed initially. However, neurosurgeons should keep in mind that compression of a distal portion of the seventh cranial nerve may be responsible for HFS in cases in which neurovascular compression at the REZ is not confirmed during surgery and in cases in which neurovascular compression performed at the REZ does not result in cure of the HFS. Distal decompression of the seventh cranial nerve combined with decompression at the REZ should be performed when a deep vascular groove is noticed at the site of distal compression of the nerve.

It is quite important to ascertain that the AICA is the offending vessel in most cases of compression of the distal portion of the seventh cranial nerve. The offending vessel was the AICA in five of our seven cases. One must be very careful in manipulating the AICA because it gives rise to internal auditory arteries that enter the internal auditory canal. These internal auditory arteries are quite vulnerable to stretching, which readily causes hearing loss3 as occurred in one of our own cases (Case 6). We therefore recommend that manipulation of these vessels be performed with great care and that neurovascular decompression be performed by highly experienced neurosurgeons with continuous monitoring of the patient’s ABRs during the procedure.

References


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