The Preoperative Evaluation of the Patient Considering Microvascular Decompression for Hemifacial Spasm

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Introduction

Nonoperative treatment options for hemifacial spasm (HFS) include anticonvulsants and serial botulinum toxin injections, and operative treatment options include microvascular decompression (MVD). Unfortunately, anticonvulsants (e.g. carbamazepine, gabapentin, etc.) are ineffective in the treatment of HFS. Serial botulinum toxin injections are tolerated well by some rather than others, but essentially, botulinum toxin injections exchange weakness for cessation of spasms of the treated facial musculature. MVD of the facial nerve addresses the presumed cause of HFS, vascular compression of the facial nerve[3,6], and is effective and durable.[10] MVD, however, is associated with risks (e.g., cerebellar hematoma, cranial nerve injury, stroke, and death), albeit infrequent, not associated with serial botulinum toxin injections for HFS. Some practitioners argue that the benefits of MVD for HFS do not outweigh the risks.

In our center, we perform MVD of the facial nerve as the first-line procedure for HFS in patients able to undergo MVD regardless of age or prior history of failed MVD because a well executed MVD of the facial nerve provides the highest likelihood of success (i.e. spasm-free), the best quality of life (i.e. symmetric and normal facial function), and the lowest long-term recurrence rate of hemifacial spasms. Many of our patients have undergone an unsuccessful MVD in the past. Evaluation of the risk/benefit ratio for each patient before operation, however, is essential in optimizing care. With this in mind, this article is dedicated to the preoperative evaluation of the patient with HFS including appropriate supplemental testing in anticipation of MVD.

Step 1: Confirm the diagnosis of HFS

HFS is prevalent in 9.8 per 100,000 persons which means that approximately 30,000 Americans are affected by HFS at the present time.[7] The diagnosis of HFS is based on the clinical history and neurological examination. HFS, a syndrome of unilateral facial nerve hyperactive dysfunction, is a severe and disabling condition that causes impairments in the quality of life.[1] In most cases of hemifacial spasm, spasms begin insidiously in the orbicularis oculi muscle (i.e. the muscle about the eye) and spread over time to the muscles of the face with variable involvement of the frontalis (i.e. muscle of the forehead) and platysma (i.e. muscle of the neck) muscles. Ultimately, the patient may develop prolonged contractions of all the involved muscles causing severe, disfiguring grimacing with partial closure of the eye and drawing up of the corner of the mouth, the so-called "tonus phenomen."[4] Some patients will report

worsening of spasms with fatigue, situations of anxiety, and changes in position of the head (e.g. head to one side or the other on the pillow at night). Patients also frequently complain of new "noises" in the ear and a feeling of "fatigue" of the face of the affected side of the head as the day progresses.

Step 2. Rule out other disorders, which can be confused for HFS with an EMG

Although the diagnosis of HFS is made clinically, electromyography (EMG) (i.e. a n electrical needle test of the face) may help in distinguishing the disorder from other abnormal facial movement disorders such as blepharospasm, tics, partial motor seizures, synkinesis, Meige's syndrome, and neuromyotonia.[5,11] The electrophysiologic hallmarks of HFS consist of spontaneous, high frequency (as many as 150 impulses per second), synchronized firing on EMG and an abnormal motor response (AMR) elicited with the trigeminofacial or "blink" reflex. The AMR is the recording of a response in the orbicularis oris muscle to electrical stimuli applied over the supraorbital nerve (and sometimes a stimulus to a single facial motor branch) when the trigeminofacial reflex should be limited to the orbicularis oculi.

Step 3. Obtain a brain MRI to exclude a structural problem

Some confusion exists amongst clinicians regarding the utility of MRI in predicting which patients may benefit from proposed microvascular decompression. This confusion exists, in part, because of conflicting reports regarding the ability to detect vascular compression of the facial nerve with MRI and also the significance of vascular compression by MRI. Occasionally, patients are not referred for MVD because the interpreting radiologist, neurologist or neurosurgeon does not note neurovascular compression of the facial nerve. This is a mistake. Lack of visible neurovascular compression can be attributed to technical inadequacies of MRI sequencing or simply the inability of even the most advanced MRI sequencing to detect certain neurovascular conflict (i.e. many compressing vessels are small and venous).[8] We frequently note apparent compression of a nerve on MRI preoperatively only to discover during the operation a different vessel causing the problem and/or multiple vessels in contact with the nerve. In summary, although we can often see the offending artery compressing the facial nerve by preoperative MRI, we primarily use MRI of the brain with gadolinium to exclude structural lesions including tumors, AVM, Chiari I malformation and other confounding diagnoses.[9]

Step 4. Obtain a comprehensive medical evaluation by your internist

The ultimate goals of preoperative medical assessment are to reduce the morbidity associated with operation, to reduce the need for prolonged perioperative care, and to return the patient to his or her life without hemifacial spasms. We routinely operate on patients classified as Grades I-IV according to the American Society of Anesthesiologists scale[2]:

I. A normal healthy patient

II. A patient with mild systemic disease

III. A patient with severe systemic disease

IV. A patient with severe systemic disease that is a constant threat to life

We work closely, however, with a patient's other physicians to best prepare a patient for the operation.

Step 5. Before proceeding with MVD, consider the risks of MVD carefully

Because MVD for HFS requires the surgeon to dissect about the lower cranial nerves, the risk profile of MVD for HFS (particularly facial, cochlear, glossopharyngeal, and vagus nerves) is different than the risk profile of MVD for other cranial neuralgias (e.g. trigeminal neuralgia). Cranial nerve injuries during MVD may result in facial weakness, hearing impairment, balance troubles, and swallowing difficulty and/or hoarseness, which can affect satisfaction with MVD despite the absence of hemifacial spasms postoperatively. Many clinicians (e.g. neurologists, neurosurgeons, ophthalmologists, plastic surgeons) who routinely treat patients with HFS are appropriately reluctant to refer patients for MVD because they feel that the risks of MVD outweigh the benefits, and they may have cared for patients who have suffered serious consequences of an MVD in their practice. One patient recently told me that their local neurosurgeon warned them "MVD is a bloody mess of an operation..stick with botulinum toxin injections."

Although MVD can be "a bloody mess of an operation", the operation can be routinely completed with no more than a teaspoon of blood loss in experienced hands. In this author's experience of more than 1000 MVDs for a variety of cranial neuralgias, only one patient has required a blood transfusion. Indeed, in recent years, the risk profile of MVD for HFS has improved with further refinements of the operative technique. In this author's experience of more than 250 MVDs for HFS in the past five years, 92% of patients have become spasm-free following an operation. In that same group of patients, only one patient suffered a stroke, one patient sustained a partial facial nerve injury, and three patients lost their hearing on the affected side of the head. Additionally, no patients sustained infections or cerebrospinal fluid leaks.

Step 6. Once a decision has been made to proceed with MVD, a few other tests are required to optimize MVD results

Once a patient has made the decision to proceed with an MVD of the facial nerve, audiometry (i.e. hearing test), acoustic middle ear reflexes, and brainstem auditory evoked potentials (BAEPs) testing should be completed. The audiometric tests are performed preoperatively to obtain a baseline for quantitatively determining deteriorations or improvement in hearing function following MVD. Additionally, preoperative BAEPs provide baseline information for the clinical neurophysiology team so that they may warn the surgeon of any deviations during

monitoring of the intraoperative auditory evoked potentials to preserve hearing during the EMG.

Conclusion

MVD of the facial nerve for HFS remains the only chance for a cure of HFS. Each patient must carefully consider the risk/benefit profile of such an operation versus continued serial injections with botulinum toxin. Patients should work closely with their physicians to develop a long-term plan for HFS and consult an appropriate neurosurgeon when considering MVD.

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