14 Monitoring Posterior Fossa Craniotomies

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Introduction

The base of the skull is divided into three regions: the posterior, middle, and anterior cranial fossae (Fig. 14.1). The posterior fossa is the deepest and largest and is enclosed by the occipital bone. Within the posterior fossa are the brainstem and cerebellum. The brainstem—consisting of the midbrain (mesencephalon), pons, and medulla—contains the nuclei of cranial nerves (CN) III–XII and is responsible for a vital autonomic nervous system function. The brainstem also contains afferent and efferent fiber tracts that connect the brain with the rest of the body. The cerebellum is responsible for movement, balance, and coordination. Due to the complex anatomy and close proximity of these vital structures to each other, the use of intraoperative neuromonitoring (IOM) during posterior skull base surgery can aid the surgeon in identifying neural structures at risk as well as verifying neural integrity once the decompression is complete. This chapter focuses on surgeries for microvascular decompression (MVD), vestibular schwannoma, and Chiari malformation and the modalities used to preserve the neurological function of cranial nerves and brainstem structures during these types of surgeries.

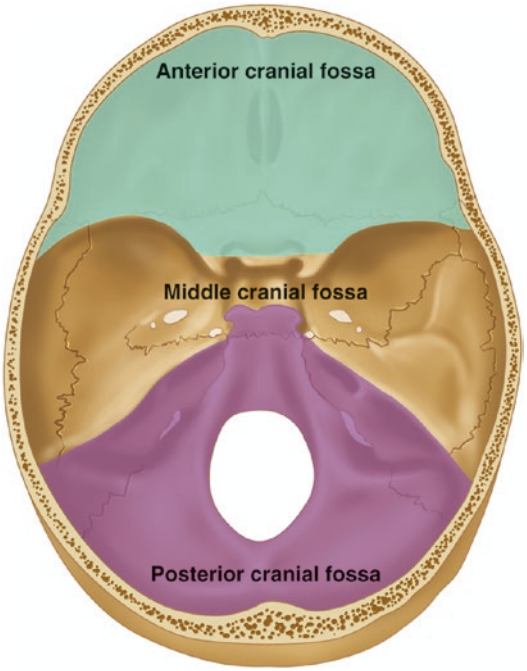

Fig. 14.1 Anterior, middle, and posterior cranial fossae

Microvascular Decompression

MVD is a procedure to relieve symptoms caused by vascular compression of a nerve. When medication does not provide relief, an

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MVD surgery is an option to treat syndromes such as trigeminal neuralgia, hemifacial spasm, and the less common glossopharyngeal neuralgia (not discussed in this section). To gain access to the offending vessel and the affected nerve, an incision is made behind the ear on the side of the head where the patient feels pain. A portion of the skull is removed and the dura is opened to expose the cerebellum. The cerebellum is moved out of the way, exposing the brainstem. Typically under a microscope, the arachnoid layer is dissected away allowing for visualization of the facial nerve (CNVII), the vestibulocochlear nerve (CNVIII), and finally the trigeminal nerve (CNV). The surgeon places a tiny Teflon sponge between the compressing vessel and the nerve, isolating the nerve from the pulsating effect and pressure of the blood vessel (Fig. 14.2).

During surgery to address cranial neuralgias, surgeons typically opt to monitor the trigeminal nerve (CNV) and the facial nerve (CNVII), using free-running EMG and triggered EMG. The trigeminal nerve is monitored by placing needle electrodes in the masseter or temporalis muscle. Facial nerve monitoring is accomplished by placing electrodes in the muscles of the five main branches of CNVII that control facial expression: temporal, zygomatic, buccal, marginal mandibular, and cervical. EMG monitoring is helpful in locating the cranial nerves and determining adequate decompression. A complication of MVD surgery is ipsilateral hearing loss from injury to the vestibulocochlear nerve. Brainstem auditory evoked potentials (BAEPs) are used to help prevent injury to CNVIII due to traction, ischemia, or cautery. BAEPS are also utilized when there

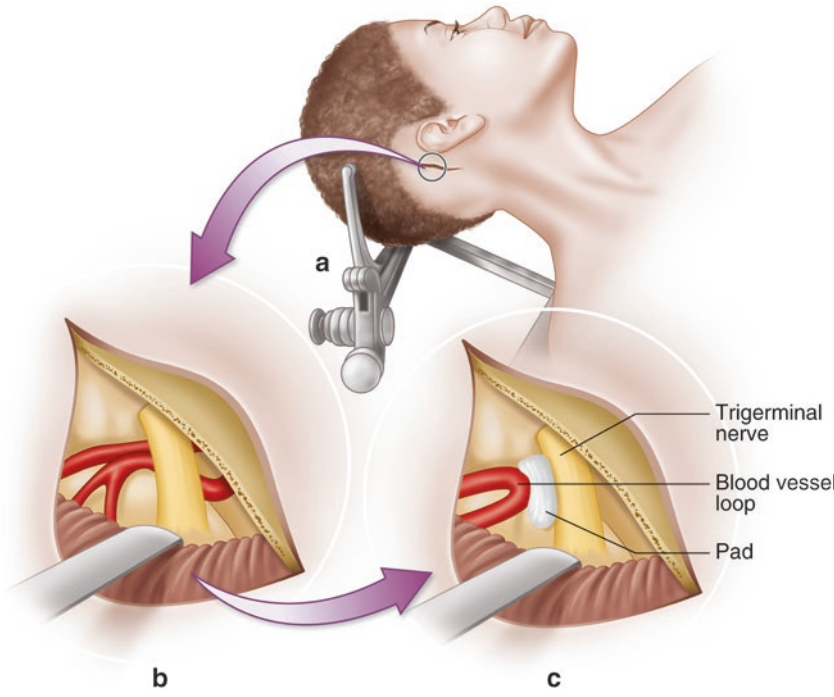

Fig. 14.2 Microvascular decompression. (**a**) Access to the trigeminal or facial nerve is accomplished through a posterior fossa craniotomy. (**b**) The cerebellum is retracted exposing the nerve and the offending blood vessel. (**c**) A Teflon pad is placed between the nerve and vessel, decompressing the nerve

is a risk of brainstem ischemia associated with manipulation of the cerebellum.

Trigeminal neuralgia, also known as *tic douloureux*, is an inflammation of the trigeminal nerve causing extreme pain and muscle spasms in the face. The trigeminal nerve functions in sensing facial touch, pain, and temperature, as well as controlling muscles used for chewing. Attacks of intense, electric shock-like facial pain can occur without warning or be triggered by touching specific areas of the face. The trigeminal nerve has three major branches. The ophthalmic, or upper, branch supplies sensation to most of the scalp, forehead, eye, and eyebrow. The maxillary, or middle, branch passes through the cheek, upper jaw, top lip, teeth and gums, and to the side of the nose. The nerve's mandibular, or lower, branch passes through the lower jaw, teeth, gums, and bottom lip. More than one nerve branch can be affected by the disorder. The superior cerebellar artery (SCA) is the vessel most often responsible for neurovascular compression of the trigeminal nerve root, although other arteries or veins may be the culprit vessels [1]. BAEPs and EMG for CNV and CNVII are typical modalities used for monitoring of MVD to relieve trigeminal neuralgia. The t-EMG response for CNV can easily be confused with CNVII responses. The latency of a t-EMG response from the trigeminal nerve should be around 5 ms, while a facial nerve response is seen around 7 ms when stimulated near the exit point from the brainstem. This should be very easy to remember!

Hemifacial spasm (HFS) is characterized by intermittent, involuntary twitching of the muscles in one side of the face, which lasts from a few seconds to several minutes. Spasms occur spontaneously and without warning. They are often exacerbated by stress or fatigue but can also be triggered by stimuli like sunlight, touch, chewing, and talking. Spasms do not cause pain, but can cause discomfort, impaired vision, social distraction, and embarrassment. HFS is most often caused by a branch of the posterior inferior cerebellar artery (PICA) or anterior inferior cerebellar artery (AICA), pulsating against the facial nerve root as it leaves the brainstem, resulting in hyperactivity of the facial nerve [1, 2]. Similar to the treatment for trigeminal neuralgia, in order to relieve HFS symptoms the facial nerve must be moved away from the offending vasculature.

To adequately monitor the facial nerve, electrodes are placed in muscles corresponding to the extracranial branches that control facial expression. For example, electrodes can be placed in the orbicularis oculi (temporal branch), nasalis (zygomatic branch), orbicularis oris (buccal branch), mentalis (mandibular branch), and if requested, the platysma (cervical branch). Free-running EMG responses in any of these muscles can indicate surgical manipulation [3]. Triggered-EMG responses can assist the surgeon in verifying the degree of decompression of the nerve. In patients with HFS, stimulation of a branch of the facial nerve may result in delayed muscle activity recorded from myotomes of adjacent branches. This is known as a lateral spread response (LSR). Current understanding is that compression of the nerve causes antidromic signals to travel back to the facial nerve nucleus within the brainstem where the nucleus becomes hyperactive and sends signals to all branches, resulting in abnormal facial movements [2–5]. Stimulation of a branch of the facial nerve may have the same effect. For example, stimulating the marginal mandibular branch and seeing a delayed response in the orbicularis oculi is evidence of a lateral spread response (Fig. 14.3). Once the offending vessel is isolated and adequate decompression has been achieved, this abnormal muscle response usually disappears. If it still persists, an additional vessel that was not apparent during visual inspection may be compressing the nerve. Monitoring the lateral spread response decreases the incidence of re-operation.

The close proximity of CNVIII puts hearing at risk during surgery for MVD. BAEPs are monitored during HFS surgery to protect hearing. In addition, BAEPs offer protection against ischemia to the brainstem.

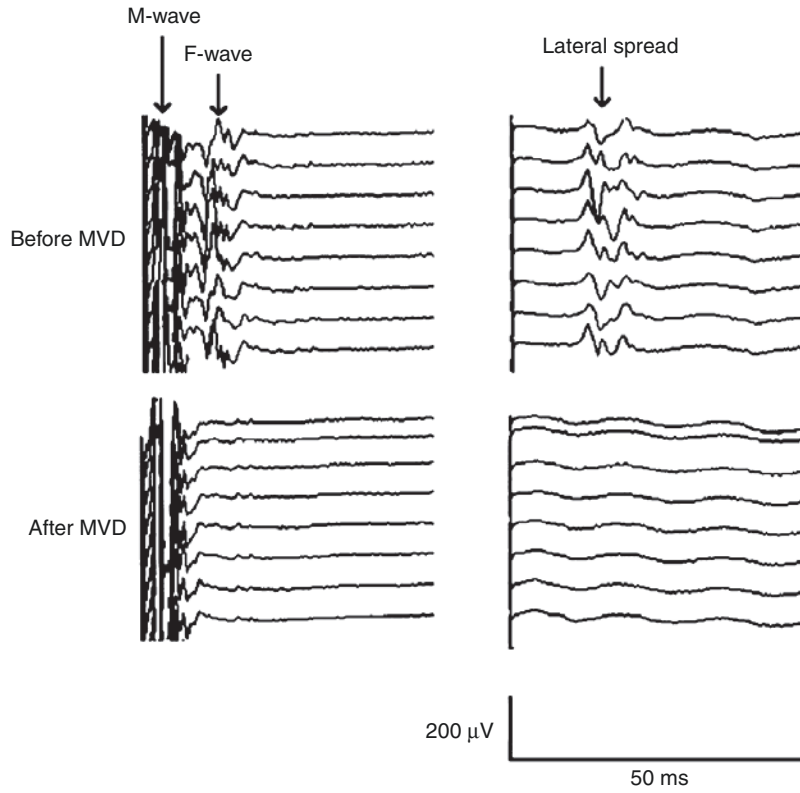

Fig. 14.3 Recordings of LSR and F wave were obtained after direct stimulation of marginal mandibular branch (*left column*) showing LSR to the orbicularis oculi muscle (*right column*). Simultaneous disappearance of the LSR and F wave after MVD was achieved. (Reprinted with permission from Fernández-Conejero et al. [16])

Vestibular Schwannoma

A vestibular schwannoma, also referred to as an acoustic neuroma, is a benign slow-growing tumor that arises from the Schwann cells covering the vestibulocochlear nerve. The vestibulocochlear nerve is the eighth cranial nerve (CNVIII) and is a sensory nerve that facilitates hearing and balance. Symptoms caused by a vestibular schwannoma correlate with the size and growth of the tumor. The most common early symptom is hearing loss. Small tumors can cause hearing loss, tinnitus, and dizziness. As the tumor expands into the cerebellopontine angle—the anatomic space between the cerebellum and the pons—hearing loss may worsen, facial weakness can occur, and balance problems may worsen. Large tumors can compress the brainstem, with severe compression causing all of the above symptoms as well as headaches and visual problems [6]. While small tumors or those causing few symptoms can be observed, surgical removal is the most common treatment for large tumors. The goal of surgery is to (1) maintain facial nerve function, (2) preserve socially useful hearing in the affected ear, and (3) remove as much tumor as possible. Total tumor removal carries a higher risk of hearing loss and facial nerve damage so surgeons often opt for partial or near-total tumor removal in order to preserve neurological function [7].

There are three main approaches to remove a vestibular schwannoma based on tumor size, location, and hearing status [8]. With a suboccipital (retrosigmoid) approach, an incision is made behind the ear and through the occipital bone to expose the internal auditory canal and the tumor. With a translabyrinthine craniotomy, the approach is through the ear in the mastoid bone. The semicircular canals are removed to expose the tumor resulting in complete sensorineural hearing loss in the ipsilateral ear. A middle fossa approach is above the ear in the temporal bone, exposing the internal

auditory canal and the tumor. This approach can be used for small tumors and when hearing preservation is optimal.

During any of these approaches, the use of intraoperative monitoring can further assist the surgeon in locating and protecting cranial nerves. Surgeons often choose to utilize EMG for CNVII to protect from surgical manipulation damage to the facial nerve or if the facial nerve is being directly affected by the tumor. A very large tumor may require EMG for CNV as well. Once the tumor is removed, the integrity of the facial nerve can be tested by electrically stimulating at points proximal and distal to the site of tumor resection. A good prognosis for facial nerve function is if low-intensity proximal and distal muscle responses are the same [9]. Additional studies suggest a low threshold post-resection response of 0.05 mA or lower with response amplitudes >240 μV is indicative of preserved facial nerve function [10, 11].

CNVIII is monitored using BAEPs, not only watching the risk to the nerve associated with stretching or compression but also detecting changes in the function of the brainstem. With a translabyrinthine approach, hearing is sacrificed but monitoring BAEPs on the contralateral side can help protect brainstem integrity. According to Angelo and Møller, recording of the BAEP makes it possible to detect insults to the brainstem before changes in cardiovascular function become apparent [12].

Chiari Malformation

A Chiari malformation (CM) is a condition in which the cerebellum herniates through the lower part of the skull and down into the spinal canal. The herniated tissue compresses the brainstem and blocks the normal flow of cerebrospinal fluid (CSF) causing a build-up of CSF in the spinal cord. This can result in a fluid-filled cavity in the surrounding white matter called a syrinx, and the condition is known as syringomyelia (Fig. 14.4). Chiari malformations are found in both children and adults and are often difficult to diagnose. Symptoms can be variable from one patient to another and are not always related to the size of the herniation. Treatment options depend on the type of malformation and the severity of the symptoms, which can range from headaches, neck pain, and vertigo to numbness in extremities, vision problems, hearing loss, fatigue, and depression.

If symptoms worsen or medications are no longer effective, a posterior fossa decompression may be necessary to create room for the cerebellum and the brainstem.

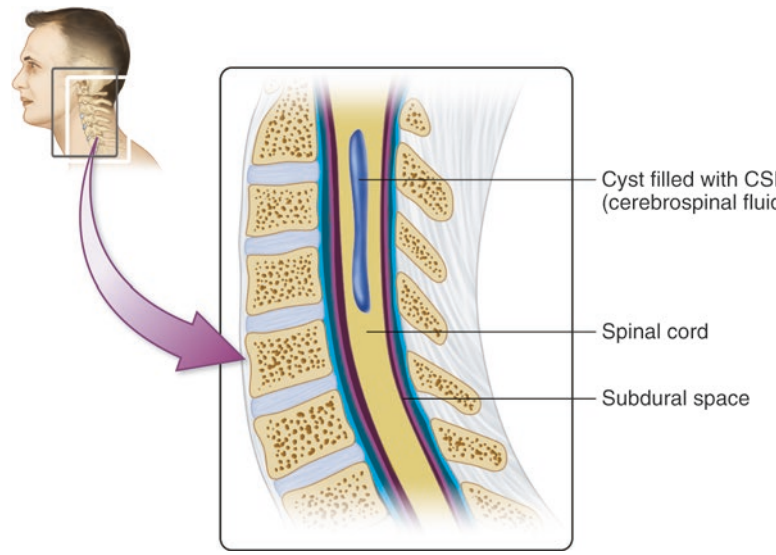

Fig. 14.4 Syrinx

There are four grades of Chiari malformations (CM Type I–CM Type IV) [13]. In Type I, the lower part of the cerebellum, called the cerebellar tonsils, extends into the foramen magnum. Type I is the most common form of CM and may not always cause symptoms. It is usually discovered during later childhood or early adulthood. Type II is where both the cerebellum and brain stem tissue are extending into the foramen magnum. Patients often have symptoms that are more severe than Type I and appear during infancy or childhood. Also seen with Type II is a form of spina bifida called myelomeningocele, where the backbone and spinal canal have not closed properly [14]. CMIII and CMIV are the most severe and are discovered at birth or with intrauterine ultrasound. This section discusses only the pathology of Chiari malformation Type I.

Beyond the cerebellar tonsils being displaced, a high incidence of patients with CM Type I will develop syringomyelia, which can cause irreversible damage to the spinal cord [15]. In addition to symptoms resulting from the cerebellar herniation, a patient's myelopathic symptoms may be attributed to an expanding syrinx. Compression of the brainstem and cranial nerve nuclei can occur as well leading to issues with sleeping, breathing, facial pain and numbness, and hearing loss.

In order to stop the progression of the herniation or if symptoms are worsening, a posterior fossa decompression is performed to reduce pressure on the cerebellum and spinal cord and restore the normal flow of CSF. An incision is made down the back of the neck, exposing the bottom of the skull and the top of the spine. A suboccipital craniotomy removes a small section of the skull. A C1 laminectomy may also be required for full decompression. Bony decompression will relieve pressure on the herniated tissue, but to fully restore CSF flow, the dura may need to be opened and then replaced with a larger autologous or synthetic dural patch. Shunting of the syrinx may also be necessary to drain CSF and relieve compression of the spinal cord.

Multiple structures and neurological functions can be at risk during surgery for a Chiari malformation. IOM is typically chosen to protect the brainstem, cerebellum, and spinal cord. As with other posterior fossa surgeries, BAEPs are used to monitor the integrity of the brainstem and effects of retraction on the cerebellum. The ascending and descending pathways of the spinal cord and brainstem are also at risk. The use of somatosensory evoked potentials (SSEPs) and transcranial motor evoked potentials (TcMEPs) will provide monitoring of the dorsal column pathway and corticospinal tract, respectively, as they pass from the brainstem into the spinal cord.

Conclusion

It is an understatement to say that multiple vital structures are in close proximity to one another in the posterior fossa. The anatomy of this area is very complex, and these vital structures can be difficult to identify, especially if a tumor has altered the anatomy even further. Neurosurgical procedures of the posterior fossa can involve the cranial nerves, brainstem, cerebellum, and the spinal cord. The use of multimodality IOM—using EMG and evoked potentials (SSEPs, TcMEPs, and BAEPs)—assists the surgical team in identifying structures at risk, as well as verifying structural integrity at the close of the procedure.

References

1. O'Donovan CA, Kuhn S. Cerebellopontine angle surgery: microvascular decompression. In: Husain AM, editor. A practical approach to neurophysiologic intraoperative monitoring. New York: Demos; 2008. p. 195–211.
2. Thirumala PD, Shah AC, Nikonow TN, Habeych ME, Balzer JR, Crammond DJ, et al. Microvascular decompression for hemifacial spasm: evaluating outcome prognosticators including the value of intraoperative lateral spread response monitoring and clinical characteristics in 293 patients. J Clin Neurophysiol. 2011;28(1):56–66.
3. Ying T, Li S, Zhong J, Li XY, Wang XH, Zhu J. The value of abnormal muscle response monitoring during microvascular decompression surgery for hemifacial spasm. Int J Surg. 2011;9:347–51.
4. Møller AR. Intraoperative neurophysiological monitoring. 2nd ed. Totowa: Humana; 2006.
5. Møller AR, Jannetta PJ. Monitoring facial EMG responses during microvascular decompression operations for hemifacial spasm. J Neurosurg. 1987;66:681–5.
6. Roland JT, Fishman AJ, Golfinos JG, Cohen N, Alexiades G, Jackman AH. Cranial nerve preservation

in surgery for large acoustic neuromas. Skull Base. 2004;14:85–91.
7. El Kashlan HK, Zeitoun H, Arts HA, Hoff JT, Telian SA. Recurrence of acoustic neuroma after incomplete resection. Am J Otol. 2000;21:389–92.
8. Lee SH, Willcox TO, Buchheit WA. Current results of the surgical management of acoustic neuroma. Skull Base. 2002;12:189–95.
9. Goldbrunner RH, Schlake H, Milewski C, Tonn JC, Helms J, Roosen K. Quantitative parameters of intraoperative electromyography predict facial nerve outcomes for vestibular schwannoma surgery. Neurosurgery. 2000;46(5):1140–6.
10. Neff BA, Ting J, Dickinson SL, Welling DB. Facial nerve monitoring parameters as a predictor of postoperative facial nerve outcomes after vestibular schwannoma resection. Otol Neurotol. 2005;26(4):728–32.
11. Mandpe AH, Mikulec A, Jackler RK, Pitts LH, Yingling CD. Comparison of response amplitude versus stimulation threshold in predicting early postoperative facial nerve function after acoustic neuroma resection. Am J Otol. 1998;19(1):112–7.
12. Angelo R, Møller AR. Contralateral evoked brainstem auditory potentials as an indicator of intraoperative brainstem manipulation in cerebellopontine angle tumors. Neurol Res. 1996;18:528–40.
13. "Chiari Malformation Fact Sheet," NINDS, Publication date June 2017. NIH Publication No. 17–4839.
14. Stevenson K. Chiari type II malformation: past, present and future. Neurosurg Focus. 2004;16(2):E5.
15. Milhorat TH, Chou M, Trinidad EM, Kula RW, Mandell M, Wolpert C, et al. Chiari I malformation redefined: clinical and radiographic findings for 364 symptomatic patients. Neurosurgery. 1999;44(5):1005–17.
16. Fernández-Conejero I, Ulkatan S, Sen C, Deletis V. Intra-operative neurophysiology during microvascular decompression for hemifacial spasm. Clin Neurophysiol. 2012;123:78–83.