Neuroanatomical correlation of the House-Brackmann grading system in the microsurgical treatment of vestibular schwannoma

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Avoidance of facial nerve injury is one of the major goals of vestibular schwannoma (VS) surgery because functional deficits of the facial nerve can lead to physical, cosmetic, and psychological consequences for patients. Clinically, facial nerve function is assessed using the House-Brackmann grading scale, which also allows physicians to track the progress of a patient’s facial nerve recovery. Because the facial nerve is a peripheral nerve, it has the ability to regenerate, and the extent of its functional recovery depends largely on the location and nature of its injury. In this report, the authors first describe the facial nerve anatomy, the House-Brackmann grading system, and factors known to be predictors of postoperative facial nerve outcome. The mechanisms and pathophysiology of facial nerve injury during VS surgery are then discussed, as well as factors affecting facial nerve regeneration after surgery.

Key Words • facial nerve • House-Brackmann scale • vestibular schwannoma • neuroanatomy • acoustic neuroma

Vestibular schwannoma is a benign CNS tumor arising from one or more constituent nerves of the eighth cranial nerve complex. Management options include observation, radiation, radiosurgery, microsurgery, and a combination of these modalities. Overall, microsurgical resection remains the best cytoreductive therapy and has been shown to be most effective for treating large lesions that cause mass effect and obstructive hydrocephalus. However, microsurgery cannot achieve the facial and cochlear nerve outcomes of radiosurgery. Because functional deficits of the facial nerve lead to physical, cosmetic, and psychological consequences for patients, avoidance of facial nerve palsy is a major goal of VS surgery. Therefore, it is critical to compare the pre- and postoperative facial nerve function in patients with VS, not only for effective care of these patients, but also to assess the likelihood of functional recovery. The House-Brackmann grading system was initially proposed in 1983 as a universal standard system for the assessment of facial nerve function, and it has since become a part of the standard of care for all patients with VS. This review will include discussions of the facial nerve anatomy, the House-Brackmann grading system, the factors known to predict postoperative facial nerve outcome, the pathophysiology of facial nerve injury during surgery, and finally, the mechanisms affecting facial nerve regeneration.

Facial Nerve Anatomy

The facial nerve has the longest and most tortuous course in the skull of any cranial nerve. The efferent component of the facial nerve mainly innervates the muscles of facial expression; in addition, it carries secretomotor fibers for the submandibular and sublingual salivary glands and the lacrimal glands. The afferent component of the facial nerve carries taste sensation from the tongue and the palate, as well as general sensation from the external ear.

The upper motor neurons of the facial nerve arise from the primary motor cortex anterior to the central fissure in the precentral gyrus (Fig. 1). The majority of the upper motor neuron axons descend through the corticobulbar tracts, cross the midline, and synapse in the contralateral motor nucleus in the pons; others descend without crossing to innervate the ipsilateral subnuclei responsible for the periorbital and frontalis muscles (Fig. 1). Thus, these subnuclei receive bilateral cortical innervation, whereas the subnuclei for the lower half of the face are only innervated by contralateral corticobulbar

Abbreviations used in this paper: CPA = cerebellopontine angle; IAC = internal auditory canal; VS = vestibular schwannoma.

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fibers. It is interesting to note that the motor pathways for emotion-induced facial movements, such as smiling and pursing the lips, contain additional synaptic junctions that have been found in the hypothalamus, basal ganglia, and midbrain tegmentum. Within the motor nuclei, subnuclei exist for each corresponding individual nerve branch, and those innervating the upper half of the face are ventral to those innervating the lower half. The fibers emerging from the facial motor nucleus initially loop around the abducens nucleus in a segment often referred to as the internal genu, in contrast to the external genu in the temporal bone.

The facial nerve consists of 2 roots: a larger medial motor root and a smaller lateral sensory root, also called the nervus intermedius, which contains fibers arising from both the sensory nucleus and the salivatory nuclei located in the rostral ventrolateral medulla. Both roots exit the lower lateral aspect of the pons between the inferior cerebellar peduncle and the olive. The average distance that the facial nerve travels from its point of exit in the brainstem at the CPA to its entrance at the IAC (the cisternal portion) is 15.8 mm, and this segment is covered by pia mater and bathed in CSF. Emerging from the brainstem, the facial nerve is accompanied by the nervus intermedius, and it runs anteriorly and superiorly adjacent to the vestibulocochlear nerves. Knowledge of the orientation of the nerves is critical during VS resection to preserve facial nerve function (Fig. 2). The anatomical proximity of the facial nerve with the nervus intermedius and the vestibulocochlear nerves at the level of the CPA and in the IAC increases the likelihood that lesions of the CPA can cause concurrent deficits of these nerves.

**Fig. 1.** Upper motor neuron connection from the cerebral cortex to the facial nuclei. The facial nuclei corresponding to the upper face are bilaterally innervated, whereas the facial nuclei targeting the lower face are only unilaterally innervated by the contralateral motor cortex. VII = cranial nerve VII; med. lemniscus = medial lemniscus.

**Tran temporal Bone Portion of the Facial Nerve**

The facial nerve traverses the entire temporal bone, and due to variation in the anatomy of the temporal bone, the course of the facial nerve within the bone is also variable (Fig. 3). Because the cisternal (intracranial) segment of the facial nerve from the brainstem to the fundus of the IAC is covered only by a thin layer of glia, it is quite vulnerable to surgical manipulation. However, it has been shown to be somewhat resistant to a slow process of stretching or compression, such as by a slow-growing VS. In the IAC, which is 7 mm long on average in adults, the motor root of the facial nerve lies in a groove on the anterior and superior surface of the auditory nerve, with the sensory root (nervus intermedius) between them (Fig. 3). Although the layer of meninges covering both the facial and auditory nerves usually ends at the fundus as the facial nerve pierces the dura, in some cases the dural coverings extend beyond the canal to as far as the geniculate ganglion.

At the fundus of the IAC, the crista falciformis (transverse crest), a transverse ridge, divides the auditory canal into superior and inferior compartments, and the facial nerve passes across the top of this ledge and is separated from the superior vestibular nerve by a vertical bony structure called the Bill’s bar. After passing through the fundus, the facial nerve enters the fallopian canal and makes a Z-shaped course until it reaches the stylomastoid foramen. Its Z-shaped course within the fallopian canal is usually described in 3 anatomical sections that are nearly perpendicular to one another on 3 different 2D planes: the labyrinthine section, the tympanic or horizontal section, and the mastoid or vertical section (Fig. 3).

The labyrinthine segment of the facial nerve is its first segment in the fallopian canal and is also the shortest and the thinnest segment within the canal. It begins at the opening of the fallopian canal and runs laterally above the vestibule, passing between the vestibule and the cochlea until it reaches the medial wall of the middle ear cleft. The end point of this nerve segment is expanded by the geniculate ganglion containing the unipolar cells of the sensory root. Because the nerve fibers in the labyrinthine segment are loosely arranged, without epineurial covering, and pressed into the lateral aspect of the canal where it is narrowest at its entrance, the facial nerve can be easily damaged by any process that decreases this already narrow course. In addition, the blood supply to the nerve in this region is uniquely without anastomosing arterial arcades.

The tympanic portion begins at the geniculate ganglion where the nerve abruptly turns 90° to run posteriorly and inferiorly to form the external genu. The length of this section of the nerve is approximately 12 mm and is above the oval window and below the prominence formed by the horizontal semicircular canal.

The final segment of the facial nerve in the fallopian canal is the mastoid or vertical portion, which begins with a less abrupt bend, called the pyramidal turn, in which the nerve passes below the semicircular canal to descend behind the pyramid. This segment is roughly 15 mm in length and ends when it reaches the stylomastoid foramen. The mastoid segment lies immediately anterior...
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and medial to the lateral semicircular canal. On average, the facial nerve occupies approximately 25%–50% of the cross-sectional area of the fallopian canal.

After the facial nerve emerges from the stylomastoid foramen, it continues its course downward and forward, crosses the lateral surface of the styloid process and the external carotid artery, and reaches the parotid gland. Then, the nerve divides behind the neck of the mandible into 2 main branches, the temporofacial and cervicofacial trunks, which further subdivide to form the 5 terminal branches called the temporal, zygomatic, buccal, marginal mandibular, and cervical branches (Fig. 4).

Branches of the Facial Nerve

The facial nerve has numerous branches throughout its course, and they can be categorized as either branches of communication with other nerves or as branches of distribution to target tissues. The branches of the facial nerve can also be divided anatomically into 3 categories: branches within the temporal bone, branches in the neck, and branches in the face.

There are 2 branches of the facial nerve within the temporal bone: the nerve to the stapedius muscle and the chorda tympani nerve. The nerve to the stapedius muscle innervates the stapedius muscle; the chorda tympani nerve mainly carries fibers for taste sensation of the anterior two-thirds of the tongue, and also contains efferent fibers for saliva secretion.
secretomotor parasympathetic fibers for the submandibular and sublingual salivary glands. The temporal branch supplies anterior and superior auricular muscles, frontal belly of the occipitofrontalis muscle, and the orbicularis oculi and corrugator muscles. The zygomatic branch innervates the orbicularis oculi principally. The buccal branch supplies procerus, zygomaticus major and minor, levator labii superioris, levator anguli oris, depressor anguli oris, depressor labii inferioris, and mentalis muscle. The cervical branch mainly supplies the platysma muscle.

There are 3 branches of the facial nerve in the neck: the posterior auricular, digastric, and stylohyoid branches. The posterior auricular branch has an auricular division to the posterior auricular muscle and an occipital division to the occipital belly of the occipitofrontalis muscle. The digastric branch supplies the posterior belly of digastric muscle; the stylohyoid branch supplies the stylohyoid muscle.

There are 5 main branches of the facial nerve in the face with direct clinical relevance to the House-Brackmann scale (Fig. 4): temporal, zygomatic, buccal, marginal mandibular, and cervical branches. The temporal branch supplies anterior and superior auricular muscles, frontal belly of the occipitofrontalis muscle, and the orbicularis oculi and corrugator muscles. The zygomatic branch innervates the orbicularis oculi principally. The buccal branch supplies procerus, zygomaticus major and minor, levator labii superioris, levator anguli oris, levator labii superioris alaque nasi, orbicularis oris, and buccinator muscle. The marginal mandibular branch innervates risorius, depressor anguli oris, depressor labii inferioris, and mentalis muscle. The cervical branch mainly supplies the platysma muscle. In addition to the 5 branches in the face, the cutaneous fibers of the facial nerve accompany the auricular branch of the vagus, and these fibers are believed to innervate the skin over the conchal cartilage. It is worth noting that what has been described only represents a general pattern of innervation, and different patterns of innervation are possible because the terminal branches of the facial nerve anastomose in a plexus. Therefore, patients with different innervation patterns of facial nerve branches may recover from VS surgery with variable speed and pattern, resulting in differential outcome.

**Blood Supply of the Facial Nerve**

The facial nerve is supplied in the temporal bone by 3 arteries with overlapping territories, such that any 1 portion of the nerve has at least 2 supplying vessels, with the only exception being the labyrinthine segment (Fig. 5). The anterior inferior cerebellar artery feeds the nerve in the posterior fossa, and the internal auditory artery, which is a branch of the anterior inferior cerebellar artery, specifically supplies the nerve in the IAC. The petrosal branch of the middle meningeal artery mainly supplies the nerve in the fallopian canal. Within the canal the artery divides at the geniculate ganglion into a descending branch that runs distally with the nerve to the stylomastoid foramen and an ascending branch that supplies the region proximal to the geniculate ganglion. The stylomastoid branch of the posterior auricular artery enters the facial canal through the stylomastoid foramen, with an ascending branch running up with the nerve to the geniculate ganglion and a descending branch supplying the nerve down to the stylomastoid foramen, while running with the posterior auricular nerve. These arteries and their associated veins course within the loose connective tissue of the epineurium, between the peristeum of the canal wall and the nerve sheath proper. Within the temporal bone, not only do the branches of these arteries anastomose with each other and with the vascular plexus of the middle ear mucosa, but they also anastomose within the marrow spaces of surrounding bone. Finally, the extracranial portion of the facial nerve receives its blood supply from the branches of the stylomastoid, posterior auricular, superficial temporal, and transverse facial arteries, which send anastomosing branches to the nerve.

**House-Brackmann Facial Nerve Grading System**

Various systems for assessing facial nerve function have been proposed since the 1950s, but none were universally adopted until House and Brackmann in 1983 systematically reviewed most that were in existence at the time. They proposed a new grading system intended to be an international standard that could be widely accepted and have sufficient reliability and validity. The grading system they proposed includes a 6-point scale, with Grade 1 representing normal and Grade VI representing total, flaccid paralysis (Fig. 6; Table 1). While they initially suggested that a grading score can be correlated with an 8-point scale from direct measurements of the movement of the eyebrow and corner of the mouth and comparing the results with those on the unaffected side, it was considered much easier to assign a grade based on the 6-point scale from simple clinical observation.
The implementation of the House-Brackmann grading system involves assessment of 3 components that contribute to the assignment of each grade: observations grossly, the face at rest, and motions of the facial muscles.\textsuperscript{48} When the 3 components of the grade do not fall into the same grade level, the usual clinical practice is to assign the most severe grade. Grade I represents normal facial movement in all areas with no weakness or synkinesis, which is the involuntary movement of a part of the face during voluntary movement of another part of the face. Grade II indicates mild dysfunction; one can observe slight asymmetry of facial movements with possible slight synkinesis, but normal symmetry and tone at rest. Grossly, there is slight weakness and asymmetry on close inspection, but the eye can achieve complete closure with minimum effort.\textsuperscript{48} In the published literature, a Grade II or less is often considered to indicate preserved facial nerve function.

In Grade III there is moderate dysfunction, with obvious but not disfiguring differences between the 2 sides; there can be noticeable but not severe synkinesis, contracture, and/or hemifacial spasm. In Grade IV, there is moderately severe dysfunction with obvious weakness and/or disfiguring asymmetry. Importantly, there is normal symmetry and tone at rest from Grades II to IV, but not Grades V or VI. In Grade III slight to moderate forehead movement remains evident, but in Grades IV and higher the forehead has no movement. In Grade III the eye can completely close with effort, but in Grade IV there is incomplete closure of the eye. The mouth is slightly weak with maximum effort in Grade III and is asymmetrical with maximum effort in Grade IV. In Grade V there is severe dysfunction, and grossly there is only barely perceptible motion, and one can observe asymmetry even at rest. The forehead has no motion, the eyelids cannot completely close, and the mouth can move only slightly with maximum effort. Finally, in Grade VI, there is total paralysis, and no movement of any kind is observed.

**Vestibular Schwannoma Surgery**

Surgical exposure of the facial nerve may be necessary for decompression, grafting, rerouting, or removal of lesions such as VSs, meningiomas, facial nerve neuromas, and cholesteatomas.\textsuperscript{12} Current microsurgical techniques make it possible to expose the entire course of the facial nerve without injuring the nerve and without disturbing hearing or vestibular functions.\textsuperscript{37}

Currently there are 3 established surgical approaches for VS resection (Fig. 7): the retrosigmoid, translabyrinthine, and middle fossa approaches.\textsuperscript{80} The rationale for the translabyrinthine approach is to gain lateral access to the IAC and the CPA lesions with no cerebellar retraction (Fig. 7D–F).\textsuperscript{80} The translabyrinthine approach also allows exposure of the neurovascular structures present in the CPA and thus enables removal of a VS of any size.\textsuperscript{80} However, unlike the middle fossa and retrosigmoid approaches, the translabyrinthine approach does not allow for hearing preservation.\textsuperscript{42} The retrosigmoid approach is a modified suboccipital approach that is performed more anterolaterally, just posterior to the sigmoid sinus (Fig. 7A and B). Unlike the translabyrinthine approach, this approach provides access to the CPA without sacrificing the labyrinthine, and the IAC is exposed by drilling its posterior wall (Fig. 7B and C).\textsuperscript{80} The middle fossa approach allows complete exposure of the IAC from the porus to the fundus and limited exposure of the CPA through the superior surface of the temporal bone (Fig. 7H and I);
thus, this approach allows for hearing preservation, while the translabyrinthine approach does not.\textsuperscript{80}

For small tumors, typically smaller than 20 mm, several studies have found that the preservation of facial nerve function was better achieved with translabyrinthine and retrosigmoid approaches compared with the middle fossa approach: preserved function was noted in more than 90% of cases for the translabyrinthine and retrosigmoid approaches, compared with 80% for the middle fossa approach.\textsuperscript{27,72} The worse outcome associated with the use of the middle fossa approach could be explained by the fact that the position of the facial nerve relative to the tumor, which most often arises from the inferior vestibular nerve, makes the nerve particularly vulnerable during the approach. Hillman et al.\textsuperscript{42} recently found similar results from comparing postoperative facial nerve outcomes of patients treated surgically using either the middle fossa or retrosigmoid approach; not only was the retrosigmoid approach associated with a higher percentage of preservation (80% for the middle fossa approach vs 90% for the retrosigmoid approach), but facial function recovered faster with the retrosigmoid approach, and there were more long-term House-Brackmann Grade I function results in the retrosigmoid group. This finding was confirmed by Rabelo de Freitas et al.,\textsuperscript{72} but they also found that the difference in facial nerve outcome appeared only for extrameatal tumors when they compared size-matched tumors (58.3% preservation in the middle fossa approach vs 98% in the retrosigmoid approach; p = 0.0006). Additionally, they found no difference in hearing outcome between the 2 approaches.\textsuperscript{72} However, Hillman et al.\textsuperscript{42} observed better hearing preservation in the middle fossa group and found that there were more recurrent and residual tumors in the retrosigmoid group.

The rates of CSF leaks between the 3 approaches were not significantly different as reported by Mangus et al.\textsuperscript{84} Previously, Brennan et al.\textsuperscript{14} reported that there was no difference in the leakage rate between translabyrinthine and retrosigmoid approaches (7.9% vs 10%, respectively; p = 0.46), although there were differences in the site of the leak: 56% of translabyrinthine leaks occurred through the wound, compared with only 8% of retrosigmoid leaks (p = 0.007), whereas otorrhea accounted for 9% of translabyrinthine leaks compared with 42% of retrosigmoid leaks (p = 0.02). Tumor size (maximum extracanalicular diameter) had a significant effect on the leakage rate overall: tumors in cases complicated by CSF leak were significantly larger than in those without a leak (mean diameter 21 vs 15 mm, respectively; p = 0.001), although significantly larger tumors were removed via the translabyrinthine approach compared with the retrosigmoid approach (22 vs 15 mm, respectively; p < 0.001). On subgroup analysis, it was found that this association of leak with tumor size was only significant for the retrosigmoid approach (p = 0.007), whereas otorrhea accounted for 9% of translabyrinthine leaks compared with 42% of retrosigmoid leaks (p = 0.02). Tumor size (maximum extracanalicular diameter) had a significant effect on the leakage rate overall: tumors in cases complicated by CSF leak were significantly larger than in those without a leak (mean diameter 21 vs 15 mm, respectively; p = 0.001), although significantly larger tumors were removed via the translabyrinthine approach compared with the retrosigmoid approach (22 vs 15 mm, respectively; p < 0.001). On subgroup analysis, it was found that this association of leak with tumor size was only significant for the retrosigmoid approach, not only that there was no significant relation between the extent of resection and the rate of tumor recurrence, but the extent of resection was highly correlated with patient age, tumor size, and surgical approach. However, using Cox regression analysis, the authors found that the approach used did not significantly affect tumor control when the extent of resection was controlled for.\textsuperscript{85}

### Prognosticators of Facial Nerve Outcome After VS Surgery

Prognostic factors such as age, tumor size, extent of

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#### TABLE 1: The House-Brackmann grading scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
<th>Characteristics</th>
<th>Estimated Function (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>normal</td>
<td>normal at rest; normal motion</td>
<td>100</td>
</tr>
<tr>
<td>II</td>
<td>mild dysfunction</td>
<td>normal symmetry &amp; tone; forehead: moderate to good</td>
<td>80</td>
</tr>
<tr>
<td></td>
<td></td>
<td>function; eye: complete closure w/ minimum effort;</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>mouth: slight asymmetry</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>moderate dysfunction</td>
<td>normal symmetry &amp; tone; forehead: slight to moderate</td>
<td>60</td>
</tr>
<tr>
<td></td>
<td></td>
<td>function; eye: complete closure w/ effort; mouth:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>slightly weak w/ maximum effort</td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>moderately severe dysfunction</td>
<td>normal symmetry &amp; tone; forehead: none; eye:</td>
<td>40</td>
</tr>
<tr>
<td></td>
<td></td>
<td>incomplete closure; mouth: asymmetric w/ maximum</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>effort</td>
<td></td>
</tr>
<tr>
<td>V</td>
<td>severe dysfunction</td>
<td>asymmetry</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td></td>
<td>forehead: none; eye: incomplete closure; mouth:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>slight movement</td>
<td></td>
</tr>
<tr>
<td>VI</td>
<td>total paralysis</td>
<td>no movement</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>asymmetry</td>
<td></td>
</tr>
<tr>
<td></td>
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Neuroanatomical correlation of the House-Brackmann scale

resection, and surgical approach have been implicated for predicting facial nerve function outcome after surgical removal of VS. In a meta-analysis of 296 studies involving more than 25,000 patients that included outcome data for facial nerve function of surgically treated VS patients, Sughrue et al. found that tumor size of less than 20 mm, the use of the middle fossa approach, and the use of neuromonitoring during surgery were associated with facial nerve preservation. However, others have found through retrospective review of patients presenting at single institutions that for small tumors, specifically those smaller than 20 mm, the preservation of facial...
nerve function was better achieved with translabyrinthine and retrosigmoid approaches compared with the middle fossa approach. Additionally, Brackmann and Barrs and Sanna and Caylan had previously suggested that the enlarged translabyrinthine approach produces the best facial nerve functional outcome; the lowest frequency of postoperative neurological sequelae was achieved, likely due to the absence of cerebellar retraction. Thus, there is a lack of consensus on the effect of surgical approach on preservation of facial nerve function.

In a recent multivariate logistic regression analysis of patients with VS from a prospectively collected database at the University of California San Francisco, Bloch et al. examined the effect of variables such as surgical approach, tumor size, patient age, and extent of resection on rates of facial nerve dysfunction after surgery. Only preoperative tumor size significantly predicted poorer facial nerve outcome for patients followed up for at least 6 months, as well as those followed up for at least 12 months. These investigators found no significant relationship between facial nerve function and any of the other factors they examined, such as extent of resection, surgical approach, and age.

Intraoperative continuous monitoring of evoked electromyography activity has been used by surgeons for more than a decade in preserving cranial nerve functions during VS resection. The usefulness of this procedure has been the subject of many recent studies. In a single institutional report of 477 surgically treated patients with VS, Sughrue et al. reported that elevated stimulation threshold exceeding > 0.05 mA is a highly specific (90%), but very insensitive (29%) finding in their cohort. The positive and negative predictive values of facial nerve electromyography for detection of permanent facial palsy reported were 68% and 63%, respectively. Additionally, they found that the negative predictive value decreased with increasing tumor size (72% vs 64% vs 53%) due to the increasing prevalence of postoperative facial nerve palsy in these patients. Whereas the findings by Sughrue et al., showed that the predictive value for facial nerve function remained to be determined, Amano et al. reported that the postoperative course of facial nerve function appears predictable using intraoperative monitoring. These investigators found that the amplitude preservation ratio correlated significantly with facial nerve function both immediately and 1 year after surgery.

In summary, while there is a lack of consensus on the effect of surgical approach on preservation of facial nerve function, large tumor size and elevated stimulation threshold during intraoperative monitoring have been associated with poor preservation of facial nerve functional outcome. Conversely, small tumor size and the use of neuromonitoring have been associated with good facial nerve functional outcome, likely from the lower risk of nerve injury intraoperatively.

**House-Brackmann Correlation of Early Recovery Patterns in Facial Nerve Function After VS Surgery**

One of the important functions of the House-Brackmann grading scale is its ability to allow physicians to conveniently and precisely track the recovery of a patient’s facial nerve function after injury. Substantial evidence suggests that the most important determinant of successful clinical outcome of the recovery of a peripheral nerve is time to reinnervation. The faster the end organ is reinnervated, the less likely it is to undergo atrophy and permanent denervation. The facial nerve’s recovery after VS surgery can be similarly dependent upon the speed of immediate postoperative recovery.

Anecdotally, we have observed that VS patients with postoperative facial nerve dysfunction more often recover their upper face and especially eyelid function before their mouth or lower face function. House and Brackmann also explained that recovery of forehead movement indicates that there has not been total degeneration of the nerve, implying that the forehead branches are either more resistant to permanent injuries or are more likely to recover immediately after surgery. Thus, if frontalis motion is absent, facial nerve may have been sacrificed. Moreover, Brackmann and Barrs noted that if mouth movement is preserved, then the facial nerve is likely preserved, further indicating that the lower branches are more easily damaged.

Based on self-reported questionnaires, Brackmann and Barr’s analysis revealed that although patients’ ability to raise an eyebrow on the affected side postoperatively correlated well with their estimated extent of final recovery, their ability to move the corner of their mouth did not. This finding appears to suggest that the recovery of upper facial function directly correlates with the anatomy of facial nerve recovery and regeneration, and it further suggests the existence of a potential underlying physiological explanation for our experience of observing patients’ clinical course of postoperative facial nerve function.

**Physiological Basis of Facial Nerve Dysfunction**

The facial nerve is often distorted by VS, both in shape and in relationship to other anatomical landmarks. Thus, most surgeons use intraoperative electrical stimulation for both positive identification and as proof of preserved function even when the normal anatomy is preserved.

Several sources have been reported in the literature regarding the pressure-induced motor neuropraxia of the facial nerve, using physiological studies: primary pressure effects on saltatory nerve conduction, blockage of bulk and rapid axonal transport, and regional ischemia. It has been known that gentle pressure applied to the trunk of a peripheral nerve even for long periods of time, such as one produced by a slow-growing small VS, produces only minor anatomical change and no alteration of conduction; on the other hand, severe pressure, such as that of a surgical clamp, usually results in Wallerian degeneration and prolonged conduction block. Between the two ends of this pressure spectrum is a range of transient or reversible neuropraxias in which the onset, magnitude, and duration of the block after release of the nerve are roughly proportional to the magnitude and duration of the applied pressure. In real-world scenarios, however, the magnitude of force applied to the nerve can be more
important than the duration of the force, given the often observed differential functional outcome of surgical- versus tumor-derived pressure on the nerve. Because the nerve root central to the geniculate ganglion lacks the perineurium and epineurium, it is more vulnerable to compression injury. This central portion of the nerve also lacks the tensile strength of its peripheral counterpart and is more sensitive to traction injury than its peripheral counterpart. It is therefore possible that due to the mechanical nature of such injuries, the location of the injury site likely predicts functional outcome. During surgery, manipulation of the facial nerve proximal to the geniculate ganglion is thus more likely to injure the nerve fibers and produce functional deficits than if the nerve is manipulated in a similar fashion in the distal portions of the facial nerve. Moreover, the more central the injury site, the longer it takes the nerve fibers to regenerate to reach its innervating target.

**Somatotopic Basis of Physiological Manifestation of Partial Facial Nerve Injury: Microscopic Examination of Facial Nerve Anatomy and Spatial Orientation of Fibers in the Temporal Bone**

At the levels of the somatomotor cortex and the facial nucleus, the existence of somatotopic organization of neurons and their processes has been known for some time. The idea of a similar organization at the level of the facial nerve was suggested nearly a century ago, but it has not been definitively proven. There is substantial evidence, however, supporting a somatotopic organization of the facial nerve trunk. It is currently an accepted theory that somatotopic organization exists within at least some parts of the facial nerve and that the maintenance of this organization during regeneration is crucial for reinnervating correct targets (Fig. 8). While evidence suggesting the lack of such an organization also exists, the quality and amount of evidence is insufficient to disprove the existence of a somatotopic organization at this time.

Many of the findings demonstrating such an organization of the facial nerve were obtained initially using clinical observations, scalpel hemisections, and radiofrequency lesions; and as more modern techniques were developed, evoked electromyography activity, microdissection techniques, crush injuries, tease avulsions, and in many instances histopathological correlations have been used. Canuyt first reported that branches supplying the upper region of the face were surrounded superficially by those supplying the lower region. Canuyt’s initial findings were later confirmed by Eyries and Chouard.

Hofmann proposed a different arrangement with a superior ramus and an inferior ramus, consisting of fibers innervating the upper and lower part of the face, respectively, that rotated slightly as the nerve exited the stylo mastoid foramen. confirmed Hofmann’s findings and reported that the upper ramus was located posterolaterally and the lower ramus anteromedially, although May pointed out that the anatomical dissection methods Hofmann relied upon could not adequately permit the conclusions he proposed. Additionally, Pollmann and Miehlke both independently reported that fibers within the tempo-
they concluded that this plexus formation and interbranching of funiculi disallowed spatial arrangement, although they admitted that it was possible for each peripheral branch to have a predominant representation in a particular sector of the facial nerve because they stipulated that they had only observed a gross anatomical intermingling and funicular redistribution. Additional evidence from Harris’ and Scoville’s attempts at partial transections of the facial nerve trunk in cats, resulting in no sparing of any of the peripheral branches, was interpreted by those authors as suggestive of a random arrangement of the facial nerve. However, the technical challenges of these experiments have caused others in the field to question the validity of a true hemisection of a nerve, and the lack of definitive data proving the randomization of fibers has allowed many investigators and clinicians to continue to believe the spatial arrangement concept. One critical flaw with Harris’ and Scoville’s reports, however, was that they did not document the extent of these lesions histopathologically. Thus, it is possible that they actually created near-total lesions. In contrast to the reports of Harris and Scoville, May’s landmark study demonstrated that the sparing of selective branches of the facial nerve was, in fact, a direct result of partial lesions as confirmed by histopathological changes specifically at the lesion site.

After May’s landmark study, subsequent investigations beginning in the late 1970s and early 1980s using retrograde nerve fiber labeling methods have again produced conflicting data. Using the horseradish peroxidase technique to retrogradely trace axons of the facial motor nerve in the rat and cat, Thomander et al. found that the intratemporal portion of the facial nerve was diffusely distributed. However, Crumley found that a definite spatial orientation was retained at least in the extratemporal portion of the nerve, also using the horseradish peroxidase labeling method. Subsequently, he found that the orbicularis oculi was represented in the posterolateral aspect of the facial nucleus, and facial muscle distribution between the human and the cat. Additional evidence from Harris’ and Scoville’s attempts at partial transections of the facial nerve trunk in cats, resulting in no sparing of any of the peripheral branches, was interpreted by those authors as suggestive of a random arrangement of the facial nerve. However, the technical challenges of these experiments have caused others in the field to question the validity of a true hemisection of a nerve, and the lack of definitive data proving the randomization of fibers has allowed many investigators and clinicians to continue to believe the spatial arrangement concept. One critical flaw with Harris’ and Scoville’s reports, however, was that they did not document the extent of these lesions histopathologically. Thus, it is possible that they actually created near-total lesions. In contrast to the reports of Harris and Scoville, May’s landmark study demonstrated that the sparing of selective branches of the facial nerve was, in fact, a direct result of partial lesions as confirmed by histopathological changes specifically at the lesion site.

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One of the potential concerns about the direct clinical relevance of these basic physiological studies conducted on animals is whether their findings, either supporting or refuting the existence of a spatial organization of the facial nerve, can be applied to the human facial nerve anatomy. Although many studies of the facial nerve anatomy were performed in cats, there is evidence to suggest that the spatial anatomy of the tympanomastoid portion of the facial nerve proposed for the cat model systems may be generally applied to man, a general assumption held in the field supported by the close resemblance of the spatial anatomy of the facial nerve in the motor cortex, pontine nucleus, and facial muscle distribution between the human and the cat.

Pathophysiology of Facial Nerve Lesion During VS Surgery

It has been well established that peripheral nerves can regenerate after nerve injury, and recovery of motor function following nerve damage is due to the ability of peripheral nerves to sprout and reinnervate denervated targets. The postoperative facial nerve function may thus be determined by the nature of the injury from the tumor growth, damage incurred during tumor resection, or the various factors affecting the recovery of the individual nerve branches. Compared with any potential damage incurred during a resection of a VS, the destructive effects that a slow-growing tumor has on the facial nerve are relatively minimal. In the case of attempted total resection of the tumor, there may be a danger of severe functioning nerve fibers that are microscopically embedded within the tumor.

Tumor-Nerve Interface During VS Surgery

During resection of a VS, the surgeon must find a cleavage plane between the facial nerve and the tumor, which can usually be achieved by fine dissection with the aid of the operating microscope. However, evidence from histological studies of the interface between a VS and the cochlear nerve from en bloc–resected VS tissue suggests that no well-defined connective tissue structure exists between the cochlear nerve and tumor tissue. The histological data on the facial nerve and tumor interface are much more scarce, however, due to efforts to preserve facial nerve function among surgeons. Nevertheless, studies exist that examine the interface between facial nerve and tumor, and all demonstrate lack of such interface in at least parts of the tumor, if not in all observable parts of the tumor histologically. Jääskeläinen et al. found that where the facial nerve trunk is attached to the surface of the tumor, nerve fibers in the contact area are either abutted directly against tumor cells or penetrated into the tumor tissue. Because these studies examined the histology of large VSs almost exclusively, they are particularly relevant to the current management paradigm for VS because the larger the tumor, the more likely it becomes a surgical candidate. Although there is no definitive evidence that the fibers embedded in the tumor are functional, immunostaining confirmed the existence of axoplasm with neurofilaments. Additionally, where the nerve fibers appeared intact, bundles of axons were sheathed by only a thin endoneurium that could be easily disrupted by the infiltrating tumor, often eliminating the distinct boundary between nerve fibers and tumor tissue, making it difficult to fully visualize the histological

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relationship of nerve fibers and tumor tissue through the operating microscope.  

The clinical implications of the lack of a tumor-nerve boundary are currently not fully understood, however. Data from a large series of VS resections published by Thomsen et al. suggest that during surgery not all fiber damage as noted by the surgeons leads to functional dysfunction, especially when taking into account the tumor size. In particular, in small- to medium-sized tumors, the postoperative outcome was much better than predicted based on nerve damage noted during surgery, but in large and giant tumors the postoperative outcome was much worse then predicted. Therefore, regarding the small- to medium-sized tumors, the histological questions about the nerve-tumor interface may appear less clinically relevant, because the current microsurgical technique in combination with intraoperative facial nerve monitoring can almost always achieve a de facto cleavage plane and a satisfactory facial nerve outcome. However, in the case of large tumors, unnoticed severing of these fibers can occur during surgery and result more often in immediate postoperative complications, perhaps due to embedding of nerve fibers within the tumor. Nevertheless, recovery of facial nerve function to normal (House-Brackmann Grade I) was shown to be achieved in 53.4% of large tumors and 31.8% of giant tumors, suggesting that not all large or giant tumors engulf all facial nerve fibers or that severing some portion of the fibers does not cause irreversible functional deficits.

It is currently no longer the goal of many VS surgeries to remove every last fragment of tumor that is adherent to the facial nerve, especially in large tumors, mainly to preserve facial nerve function. Moreover, these tumors are generally slow growing, and other adjunctive therapies such as Gamma Knife surgery are available for the residual tumors. In a comparison of 15 patients who underwent subtotal resection of tumors that exceeded 3 cm in diameter with the published results of patients with similarly larger tumors, Raftopoulos et al. found that facial nerve dysfunction was 0% in their patients compared with 20%–35% in patients from other published series. Following their suggestion that planned subtotal resections may be appropriate for large tumors, there is now a multicenter prospective trial to assess subtotal resection as treatment for tumors that are larger than 2.5 cm in diameter (www.clinicaltrials.gov; trial no. NCT01129687). Moreover, Esquia-Medina et al. found not only the expected association between degree of tumor adhesion and facial nerve dysfunction, but also that tumor displacement of the facial nerve predicted worse outcomes. Because both displacement and degree of adhesion were also associated with tumor size and location, they demonstrated that the combination of tumor stage, adhesion, and nerve displacement in a logistic regression model was highly predictive of postoperative facial function.

Based on the current evidence on tumor-nerve interface as well as the slow growth rate of VS, it is recommended that surgeons leave a fragment of the tumor behind if necessary and take the risk that the remnant may eventually grow large enough to cause symptoms. This risk, however, can often be reduced by postoperative Gamma Knife surgery, and it has been reported to be quite effective in the literature.

Ischemia of the Nerve Fiber During VS Surgery

It has been shown that during VS resection, preserving the blood supply of the cochlear nerve is crucial for hearing preservation. Similarly, it has been believed that preserving integrity of the vascular supply of the facial nerve is also crucial for preventing facial nerve dysfunction. Anecdotally, surgeons recognize that some blood vessels shared between tumor and the nerve may become damaged when the tumors are removed, and they have observed that local microvascular damage during surgery has been associated with facial nerve dysfunction postoperatively. Recent data suggest that ischemic injury of the facial nerve incurred during VS resection caused by disturbance of the microcirculation of the nerve can cause facial nerve paralysis. Moreover, it has been shown that the use of vasoactive treatments postoperatively can mask the onset of facial nerve dysfunction, and the termination of these treatments results in delayed onset of dysfunction.

Factors Affecting Regeneration of Facial Nerve After VS Surgery

During VS surgery, microscopic damage of nerve fibers can occur due to the nature of a tumor’s adherence to the fibers of the nerve. It has been known that there are inherent limitations of nerve regeneration limiting the speed and extent of regeneration. Additionally, several lines of evidence suggest that factors such as misguided axonal regeneration, excessive axonal branching, and lack of specificity of axonal guidance all contribute to the failure of precise regeneration. Recent evidence even suggests that it is possible for the regenerating axons to become aberrant throughout the length of the facial nerve, not only at the site of the lesion. Thus, successful peripheral nerve repair depends not only upon targeting of axons to the periphery, but also upon reestablishment of appropriate connections between the periphery and the CNS.

The consequences of nonspecific regeneration are usually clinically obvious. Incorrect localization of sensory stimuli has long been recognized as a hallmark of nerve regeneration. Misdirected motor axon regeneration can present with gross distortion of the face as the patient attempts to smile. Based on various retrograde labeling techniques, evidence has also emerged in recent years that suggests somatotopic reorganization of the facial nucleus after lesioning, surgical repair, and regeneration of the facial nerve. This somatotopic reorganization as a result of axon misguidance during regrowth is believed to underlie involuntary movement of a part of the face during voluntary movement of another part of the face (synkinesis). The results of these studies demonstrate the failure of nerve axons to make correct connections with their distal targets during regeneration. While the more recent findings of facial nucleus reorganization after facial nerve regeneration do not directly
N-cadherin can act as chemoattractants for axon growth and produce guidance molecules such as neural cell adhesion molecule and laminin. Soluble isoforms of cell surface adhesion molecules such as metalloproteinases and cathepsins may also have a role in axonal alignment and may be produced by targets to create a trophic gradient in the extracellular matrix.

The speed of peripheral nerve regeneration in mammalian nervous systems is largely limited by the intrinsic rate of axonal outgrowth, which is fairly constant across species. The axonal outgrowth is, in turn, limited by the rate of slow axonal transport, which is 1–4 mm/day. This rate, however, declines with aging and contributes to poor recovery in older adults. Given the restricted speed of axonal outgrowth, the time to full recovery clearly depends on the distance that the axons need to travel to reach their targets. Because the different branches of the facial nerve travel different lengths to reach their targets after the nerve exits the stylo mastoid foramen, it is very likely that muscles innervated by shorter branches will recover faster than muscles innervated by longer branches. This mechanism may underlie the difference in the rate of recovery between orbicularis oculi and orbicularis oris, which we have observed anecdotally.

Misguided Axonal Regeneration and the Need for Axonal Guidance

Successful recovery of facial nerve function from VS surgery not only depends on the speed of recovery but also on full and correct restoration of facial nerve function to prevent synkinesis caused by axon misguidance. The establishment of topographic and end organ specificity is necessary to prevent misguided axonal regeneration. Topographic specificity allows axons to return to the muscle or area of skin they served initially; end organ specificity can then match regenerating axons with end organs of the sensory modality or muscle type to which they were connected originally. Presuming the somatotopic organization of the facial nerve, the phenomenon of synkinesis, which can occur after nerve repair, can be easily explained by an injury-induced disorganization of the original somatotopy of the facial nerve.

To achieve full facial nerve recovery without synkinesis in adults, the facial nerve needs to reestablish somatotopy. Evidence suggests that the ability of neonatal rats to reestablish somatotopy of the facial motor nucleus after nerve lesion and repair is likely due to the influence of target-derived trophic factors in the neonate. Target-derived factors also regulate collateral axon sprouting and diffusible inhibitory factors may be produced by nontarget regions to repel developing axons away from incorrect paths. Additionally, extracellular molecules such as laminin and soluble isoforms of cell surface adhesion molecules such as neural cell adhesion molecule and N-cadherin can act as chemoattractants for axon growth cones and may be produced by targets to create a trophic gradient in the extracellular matrix.

It has been suggested that excessive axonal branching is a major factor contributing to the poor functional results of facial nerve repair due to aberrant projection within several nerve fascicles, such that the branches of 1 axon often synchronously reinnervate muscles with antagonistic functions and impair any coordinated activity. Additionally, often at the site of injury, there are multiple branches with growth cones that are trying to regenerate, and many neurotrophic factors appear to enhance this multiple branching behavior (such as nerve growth factor). However, directed longitudinal elongation of a main axonal branch is necessary for enhanced speed and success of regeneration.

The specificity of axon regeneration is determined primarily at the site of nerve repair; once a regenerating axon is confined to a Schwann cell tube in the distal nerve stump, it usually follows that tube to its peripheral termination. Neurotropism, neurotropism, and mechanical alignment are factors that may influence the specificity of distal Schwann cell tube reinnervation.

Effects of Facial Nerve Injury on the Primary Motor Cortex

Poor recovery of facial function after VS surgery of individual facial muscles may also occur if the organization of higher motor centers is changed after facial nerve lesion. This phenomenon has been demonstrated in patients with facial palsy, in a rat model, and in other peripheral nerve lesions. Recent studies in humans comparing ipsilateral and contralateral facial motor cortices showed that, while in healthy patients motor evoked potentials of perioral muscles elicited by transcranial magnetic stimulation of the contralateral hemisphere were always higher in amplitude than those of the ipsilateral cortical transcranial magnetic stimulation, in patients with unilateral peripheral facial paralysis a significant increase was found in the amplitudes of intact perioral motor evoked potentials to hemisphere stimulation contralateral to the paretic side, but not to stimulation ipsilateral to the paretic side. This finding suggests that patients with peripheral facial paralysis can more strongly activate their intact perioral muscles with their ipsilateral cortices. Moreover, it has been suggested that in acute and chronic nerve injuries in humans and in primates, cortical changes are accompanied by subcortical alterations at all levels of the somatosensory core.

Another factor affecting the recovery of facial nerve function that may explain the observed differences in the recovery rate of upper versus lower facial function may be attributable to the bilateral innervation of the upper face that can result in differential disruption of cortical input after facial nerve injury. Neurons in the cortex that project to the facial nucleus are CNS neurons and behave differently compared with peripheral neurons in the facial nerve after nerve injury. It has been shown that the survival of CNS neurons, unlike those in the peripheral nervous system, depends not only on the presence of trophic factors, but also on neural activity in the form of depolarization or increased levels of cyclic adenosine...
Monophosphat.35,64,83 Thus, perhaps the lack of signaling from facial muscle motor feedback loops after facial nerve injury alters the behavior and even survival of the CNS neurons in the facial area of the primary motor cortex, at least until function is restored via peripheral regeneration of the facial nerve. Given that the lower face is unilaterally innervated compared with the upper face, which is bilaterally innervated, the probability that lower face dysfunction affects corresponding ipsilateral cortical neurons is greater than the probability that upper face dysfunction affects bitemporal cortical neurons to the same extent. In other words, in upper face dysfunction, both sides of the cortex must be affected to disrupt cortical output, whereas for the lower face, ipsilateral lesions alone can interfere with cortical output. There is currently no conclusive evidence on the effect of peripheral neuropathy on CNS cortical neuron activity or survival, but given the difference in the anatomy of cortical output to the facial nucleus corresponding to the upper versus lower face, we are likely to discover yet unknown mechanisms regulating peripheral recovery.

Conclusions

Given the importance of preserving facial nerve function in VS surgeries, improved management algorithms regarding surgical approach, extent of resection, intraoperative monitoring, and postoperative rehabilitation can be implemented based on a combination of tumor size, location, growth rate, and intraoperative observations of tumor adhesiveness. For neurosurgeons planning VS surgeries, it is important to remember that attempted gross-total resection of the tumor may pose significant risks of injuring the facial nerve. For resection of large tumors, it may be harder to correctly discern the tumor—nerve interface, and thus care must be taken such as using neuromonitoring to avoid injuring the facial nerve. Anatomically, the portion of the facial nerve central to the geniculate ganglion is more susceptible to injury due to the lack of epineurium. Additionally, because the blood supply to the geniculate ganglion is mostly supplied by 1 artery, in comparison with the existence of overlapping supply from at least 2 arteries in the more distal facial nerve trunk, that region is also more prone to ischemic injury during surgery. Favorable prognostic factors for facial nerve outcome after VS surgery include tumor size of less than 20 mm and the use of neuromonitoring during surgery. Currently there remains a lack of consensus on the use of any particular surgical approach that reduces the risk of facial nerve injury.

The differential recovery speed of the upper face compared with the lower face after surgery can be explained by the presence of bilateral cortical innervation of the upper face, the somatotopic organization of the facial nerve, the distance needed to travel by regenerating axons, and intrinsic differences in regenerating capacity of different facial nerve fibers. Diligent follow-up postoperatively with frequent functional assessments using the House-Brackmann scale is needed to allow physicians to adjust treatment plans for individual patients to deliver optimal care. The recent increase in the use of stereotactic radiosurgery as an alternative to VS microsurgery presents a viable alternative for treating select types of VS, but its efficacy in tumor control and complication profile have yet to be established.

The landscape of VS treatment is changing. As we gain a deeper understanding of the biological behavior of VS and the effect of its treatment on facial nerve function, we may further optimize our management of this disease to provide the best clinical outcome in tumor control and facial nerve preservation.

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