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OTOLOGY: VESTIBULAR SCHWANNOMAS (RA FRIEDMAN AND M SCHWARTZ, SECTION EDITORS)

Advances in Facial Reanimation: Management of the Facial Nerve in the Setting of Vestibular Schwannoma

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Abstract

Purpose of Review While vestibular schwannomas are benign in nature, their treatment carries risk of facial nerve injury. Facial palsy causes significant physical and psychological morbidity. This article focuses on contemporary recommendations for the management of facial palsy in the setting of vestibular schwannoma.

Recent Findings Approximately 5–15% of patients experience long-term facial palsy following resection of vestibular schwannoma. Facial reanimation should be considered in patients with facial paralysis without evidence of recovery by 6 months post-operatively. Treatment of facial paralysis aims at restoration of facial symmetry and reduction of functional morbidity through wide range of treatment methods, from static procedures and injectable treatments to nerve transfers and regional or free muscle transfers.

Summary Facial palsy is a dreaded complication of vestibular schwannoma extirpation. Many treatment options exist, and early referral to a facial nerve specialist should be offered to patients with facial palsy for prompt evaluation and treatment.

Keywords Vestibular schwannoma · Facial nerve · Facial palsy · Facial paralysis · Facial reanimation

Introduction

Vestibular schwannomas may compress, adhere to, or distort the natural course of the facial nerve. The facial nerve is highly susceptible to injury from tumor compression, leading to Wallerian degeneration and prolonged conduction block[1, 2]. Clinical facial function is preserved when only 10% of functioning motor neurons are active due to collateral sprouting of the remaining motor axons [1]. As such, facial palsy is an uncommon presenting symptom of vestibular schwannoma, occurring in 1.8–2.4% of patients [3, 4]. As the vast majority of patients present with normal facial function, the preservation of the facial nerve is critical to the successful treatment of this benign pathology.

In surgical resection of vestibular schwannoma, the facial nerve may have already sustained significant damage and minimal additional insult by means of compression and stretch, or thermal injury may result in significant loss of facial function despite an anatomically intact facial nerve. While neuropraxia or axonotmesis resulting from trauma to the nerve is expected to recover at least partially, severe injury and neurotmesis (complete transection) carry high morbidity [5].

Management of Vestibular Schwannoma

The management of vestibular schwannomas has evolved over time due to the growing awareness of the reduction of quality of life and functional deficits from facial palsy [6]. The goals of treatment have shifted from radical resection to more conservative methods with prioritization of functional preservation of the facial nerve. While current practices vary considerably, treatment strategies include observation, stereotactic radiosurgery, and microsurgery.

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Observation with interval imaging for growth monitoring is often elected for small tumors in asymptomatic patients [7]. Growth patterns of vestibular schwannomas are variable [7, 8], but up to 45% of observed tumors ultimately receive treatment [8, 9]. The use of conservative strategies with intervention limited to those with progression of growth or symptoms has not increased the incidence of facial palsy [9]. Observation in large or rapidly growing tumors is not recommended [7, 10] as continued tumor compression may cause further deterioration of nerve function.

The use of stereotactic radiosurgery has increased with wider availability but remains highly dependent on the practices of the treatment facility. Long-term facial nerve preservation is seen in greater than 90–95% of patients treated with stereotactic radiosurgery [11, 12] but carries risk of trigeminal paresthesia in 10–20% of patients [11]. In young patients, stereotactic radiosurgery is less frequently utilized due to risk of long-term tumor regrowth, hearing loss, and other cranial nerve deficits.

In studies including all size tumors, post-operative rates of significant facial palsy (HB III–VI) and facial paralysis (HB V–VI) are 11–44% and 1–14%, respectively, which decreases to 5–17% and 0.5–2.4%, respectively, by 1 year. Ultimately, long-term HB I–II function is achieved in 86–95% of patients [3, 13–15]. In studies including only large tumors (> 2.5 cm), post-operative rates of significant facial palsy (HB III–VI) and complete paralysis (HB V–VI) are 36–64% and 12–17%, respectively, and decrease to 11–22% and 0.3–4%, respectively, by 1 year. In large tumors, 78–88% of patients ultimately achieved HB I–II function [16, 17•, 18, 19].

The decision to operate depends upon many factors including baseline hearing, tumor size, patient anatomy, and availability of experienced surgical teams [20]. While larger tumors are almost exclusively treated with surgical management, patients with smaller tumors may be surgical candidates. Patients and clinicians must weigh the risk of facial nerve injury, possible hearing loss, and surgical complications with the desire for a chance at total eradication.

Potential Preoperative Risk Factors for Facial Nerve Injury from Vestibular Schwannoma Resection

Age

While age is an important factor in treatment selection, no association between increased age and worse facial nerve outcomes has been demonstrated [21–25]. As increasing age is associated with poor tolerance of long periods of anesthesia, older patients, particularly those with smaller tumors, are more likely to be treated with observation or stereotactic radiosurgery [8, 20]. In those with large tumors, incomplete tumor resection is more frequently utilized to decrease surgical duration [20, 26–28], which in turn has resulted in better facial nerve outcomes in these patients[29].

Tumor Size

Many reports correlate increasing tumor size with poor facial outcomes [3, 14, 16, 17•, 29–31]. A systematic review revealed that 90% of 2890 tumors < 2 cm demonstrated HB I–II function compared to 67% of 6557 tumors > 2 cm. [31]. Incomplete resection has been used to mitigate risk in the setting of large tumors. Numerous studies have shown merit for near-total and subtotal resection with respect to facial nerve outcome with acceptable rates of tumor regrowth and retreatment [27, 28, 32–35], yet some variability remains [36•]. In gross total resection, recurrence is possible but rare [14, 33, 34]. In incomplete resection, the risk of tumor regrowth is correlated with the amount of unresected tumor [10, 14, 27, 28, 34], and regrowth occurs in 9–33% of patients, although the incidence of regrowth requiring retreatment is lower [3, 14, 28, 29, 32, 33, 35, 37].

The extent of resection continues to be a matter of debate. Inevitably, the use of less than total resection will result in some cases of recurrence requiring further treatment. Similarly, in some cases, the use of total resection will result in facial palsy, for which treatment may be more challenging. As such, neither technique can be universally applied, and the surgeon is faced with balancing these risks on an individual basis [36•, 38].

Surgical Approach

Bloch et al. found no difference between approaches in the treatment of 624 tumors of all sizes at a single institution [21]. Falcioni et al. evaluated 1151 tumors < 1 cm treated over 20 years at their institution and found that the middle cranial fossa approach was associated with worse facial nerve outcomes compared to retrosigmoid or translabyrinthine approaches [30]. On the contrary, Ansari et al. performed a systematic review of the literature including treatment of 850 tumors < 1.5 cm and found the middle cranial fossa had significantly better facial nerve outcomes than other approaches [20]. Ansari et al. found that the retrosigmoid approach was significantly better than the translabyrinthine approach in 1651 patients with tumors > 1.5 cm (although noted that translabyrinthine approach was used predominantly in the largest size tumors) [20], while in a systematic review of 1156 tumors >2.5 cm, Gurgel et al. found no difference between translabyrinthine and retrosigmoid approaches [35]. Ultimately, the choice of approach should be dictated by the tumor anatomy, hearing status, overall patient health, and surgeon experience [38].

Radiographic Characteristics

Anterior tumor extension is associated with worse facial nerve outcomes, presumably due to stretch or displacement of the facial nerve, leading to higher susceptibility to injury relative to tumors with posterior extension [5, 17•, 24, 39, 40]. Wong et al. reported that tumors extending > 1.5 cm anterior to the internal auditory canal are three times more likely to have post-operative HB III or higher [40] and patients are 16% more likely to have a higher post-operative HB grade for every 1-mm increase in the tumor anterior to the internal auditory canal [17•]. The extent of intrameatal tumor and bony changes of the internal acoustic meatus does not correlate with facial nerve outcome [24, 39]. Improvements in diffusion tensor tractography, a novel MRI technique that uses diffusion-weighted imaging data to reconstruct three-dimensional neural tracts, have allowed for the delineation of the facial nerve along the tumor in 80–100% of studied cases [41, 42]. Future use could improve nerve visualization and may ultimately enhance preservation.

Cystic Vestibular Schwannomas

Cystic vestibular schwannomas represent 12–50% of all vestibular schwannomas [43]. These tumors are unpredictable and may undergo rapid expansion of cystic components [43, 44]. Cystic vestibular schwannomas are frequently larger than solid tumors at presentation [45, 46], and surgical excision is the standard therapeutic approach [43, 44]. A meta-analysis including 821 cystic and 2253 solid vestibular schwannomas demonstrated that cystic tumors were associated with a higher rate of facial palsy and lower rate of anatomic preservation of the facial nerve than solid tumors [43]. Particular caution should be applied in tumors with thin cystic walls, large extrameatal diameter, and cystic structures along the medial or anterior aspect of the tumor [44]. Success is related to surgeon familiarity with cystic tumors and use of subtotal resection when necessary [44].

Surgeon and Institution Experience

Complications rates, including facial nerve injury, decrease as volume of vestibular schwannoma treatment increases at a single institution [47, 48]. Surgical learning curves reveal higher rates of facial palsy occurring in the first 50–56 operations, with gradual improvement and stabilization of outcomes with additional experience as surgeons and their teams acquire sufficient experience to maintain optimal rates of preservation of facial function [49, 50].

Intraoperative Considerations

Intraoperative Neuromonitoring

Intraoperative facial nerve monitoring was developed to provide real-time feedback through continuous electromyography monitoring, typically of the orbicularis oculi and oris muscles. Auditory feedback occurs when distal compound action motor potentials are detected when the facial nerve is inadvertently irritated or directly stimulated.

Various metrics assessing degradation of electric response have been used to predict facial function [32, 51, 52]. In multivariate analysis, patients with electromyographic response of ≥ 100 μV to low-amplitude stimulation (0.05 mA) were eight times more likely to have a good long-term facial nerve outcome [38]. The presence of "A trains," the electromyographic pattern of prolonged neurotonic discharge, may be related to potential axonal loss and is correlated with poor post-operative and long-term facial nerve dysfunction [53–55]. The loss of electromyographic signal in an anatomically intact facial nerve is correlated with immediate post-operative facial palsy; however, recovery is possible as shown by a case series of 11 such patients with HB VI function post-operatively who recovered to HB III or better (64%) by a median time of 9.4 months [56].

No single metric can be used as a perfect predictor of final facial nerve outcome. Patients with normal nerve monitoring may develop long-term facial palsy, while others with concerning neuromonitoring findings may ultimately experience good function [38, 56]. Consideration should be given to the use of intraoperative neuromonitoring as a type of warning sign that may guide intraoperative decision-making. Concerning findings such as increased presence of A trains using electromyography or increased thresholds for stimulation of motor-evoked potentials may indicate higher risk of injury, and aborting further tumor excision should be considered [55, 57].

Facial Nerve Injury Identified at Time of Tumor Resection

Facial nerve interruption or transection occurs in 1–5% of resections of any size tumor [14, 30, 56, 58•, 59] and up to 7–14% when including only large (> 3 cm) tumor resections [18, 29, 37, 60]. When the nerve is transected, and the site of injury is clearly visible, immediate repair is indicated. If the two ends of the transected nerve can be re-approximated without tension, end-to-end anastomosis is preferred using two to three 10-0 nylon sutures and fibrin glue [59, 61, 62•, 63••].

When tensionless repair is not possible, cable interpositional graft repair should be performed with harvest of the sural, greater auricular, or medial antebrachial cutaneous nerve. The absence of epineurium in the intracranial

segment of the facial nerve makes anastomotic suturing challenging [2, 5, 64•], particularly given the depth of the working field and the background pulsation of cerebrospinal fluid [64•]. A stitch-less fibrin glue technique has been described to facilitate anastomosis, and outcomes are similar to traditional microsuture methods [61, 64•]. Recovery to HB III is seen in 50–68% of patients with cable graft repair, although paucity of details regarding cable graft length and follow-up time renders the interpretation of ultimate facial nerve recovery challenging [30, 64•, 65, 66]. When the nerve is transected in the cerebellopontine angle and the proximal stump cannot be identified at the brainstem, no intervention can be performed immediately. Instead, subsequent surgical reanimation with nerve transfer or other techniques should be discussed with the patient.

Post-operative Considerations

Anatomically Intact Facial Nerve and Post-operative Facial Palsy

The rate of significant post-operative facial palsy (HB III–VI) is highly variable and is reported in 11–44% of patients following vestibular schwannoma resection [3, 13–15]. In two large retrospective reviews of patients with an anatomically intact facial nerve and post-operative facial palsy of HB III or IV, recovery to HB I–II was seen in 70–98% and 50–88% of cases, respectively, when followed for a year or more [15, 67•]. While up to 75% of patients with immediate post-operative facial palsy (HB V) and 50% of those with complete facial palsy (HB VI) may improve, full recovery is less likely [15, 58•, 67•, 68••]. The rate of recovery and earlier onset of recovery, particularly in the first 3–6 months, are associated with better final outcomes [14, 67•].

Predicting likelihood of facial nerve recovery is difficult, and timing of facial reanimation intervention has historically been a source of debate. Traditionally, patients were observed for 12 months prior to discussing facial reanimation procedures to avoid intervention in patients who may spontaneously recover [69]. This delayed approach negatively affects those who fail to spontaneously recover and decreases their chance of success after facial reanimation surgery due to prolonged neural and muscular degeneration. Instead, patients should be considered for reanimation surgery in the absence of any detectable recovery by 6 months [67•, 68••].

This recommendation is supported by a recent study by Albathi et al., which demonstrated that occult facial reinnervation is unlikely if no evidence of facial recovery has occurred within 6 months following vestibular schwannoma resection. These patients are unlikely to recover function without surgical intervention. In their cohort of 62 patients with facial paralysis and an intact facial nerve, 35 patients spontaneously recovered, while 27 patients showed no recovery by 6 months. Of these 27 patients, 10 underwent nerve transfer by 12 months, 9 underwent nerve transfer after 12 months, and 8 patients had no intervention. All patients who underwent nerve transfer had no signs of occult reinnervation on direct intraoperative facial nerve stimulation at the time of nerve transfer, and the patients who declined intervention demonstrated HB V or VI at 18 months of follow-up. Earlier reanimation surgery resulted in reduction in the total duration of paralysis, and masseteric nerve transfer resulted in earlier recovery relative to hypoglossal nerve transfer [68••].

Care of Facial Paralysis Post-operatively

When facial palsy is noted immediately post-operatively, thorough physical examination should be performed and documented with close follow-up after the patient is discharged. Photo and video documentation including appearance at rest and with voluntary facial movements should be tracked at baseline and interval follow-up [63••]. Facial palsy exists on a spectrum ranging from flaccid palsy, with complete absence of tone and movement, to nonflaccid palsy, with variable degrees and patterns of hyperkinetic and synkinetic movement. Several clinician-based assessments are used to characterize divergent patterns of facial palsy, including the Sunnybrook [70] and eFACE [71•] grading systems. However, the House–Brackmann (HB) grading system is most commonly reported in neurotology literature [72].

When incomplete eye closure is noted, ocular care should be initiated immediately. Frequent use of daytime eyedrops, nighttime lubricating ointment, and moisture chambers is recommended to prevent corneal irritation and exposure keratopathy. For patients with significant lagophthalmos or symptoms of exposure keratitis, platinum weight placement in the upper lid is recommended and can be performed under local anesthesia [73, 74•]. Patients with a poor Bell's phenomenon (the upward and outward roll of the globe with voluntary attempt at eye closure), impaired trigeminal nerve sensation, or diminished tear production are at higher risk for exposure keratitis, and expedited weight placement is indicated [74•]. Platinum is preferred over gold due to lower allergenicity and thinner profile [75].

Early referral to a facial reanimation surgeon and multidisciplinary team is recommended for prompt assessment and intervention. Surgical treatment options are dependent upon duration of paralysis (Table 1), but supportive measures can be initiated at any time. Specialized facial nerve physical therapy programs play a critical role in patient education and facial rehabilitation; although, unfortunately, physical therapy maneuvers have not been shown to accelerate recovery. Treatment is aimed towards the reduction of compensatory hyperactivity of the healthy hemiface and reduction of synkinetic movements on the affected side using a

Table 1 Facial nerve management timeline

Nerve interruption at time of vestibular schwannoma resection
• End-to-end anastomosis
- Requires tensionless coaptation
• Cable graft
- Requires harvest of nerve graft
- 10-0 nylon sutures or fibrin glue technique
Short-term facial paralysis (up to 18-24 months)
• V-VII transfer
- Masseteric branch of trigeminal nerve transfer to distal facial nerve branch innervating smile
• XII-VII transfer
- Hypoglossal nerve transfer to main trunk of facial nerve
Long-term facial paralysis (greater than 2 years)
• Single-stage surgery:
- Temporalis muscle transfer (orthodromic or antidromic)
- Gracilis free-muscle transfer innervated by ipsilateral masseteric branch of trigeminal nerve
• Two-stage surgery:
- Gracilis free-muscle transfer innervated by cross-face nerve graft from contralateral intact facial nerve branch
• Static Interventions
- *Upper face*: brow lift, upper eyelid weight placement, lateral tarsorrhaphy, lateral tarsal strip suspension, canthoplasty, fascia lata sling
- *Midface*: nasal valve correction, nasolabial fold suspension (fascia lata or suture)
- *Lower face*: oral commissure suspension (fascia lata or suture)

combination of biofeedback, neuromuscular retraining, and soft tissue mobilization[63••, 76].

Delayed Facial Palsy

Delayed facial palsy wherein patients with normal facial function immediately post-operatively develop facial nerve weakness in the days to weeks after surgery is reported in 4–26% of patients following vestibular schwannoma extirpation [14, 17•, 26, 77–82]. The mean onset of delayed weakness is reported between 10 and 12 days post-operatively [77, 78, 80], although may begin as early as the first post-operative day. Recovery rates are encouraging; the majority of patients return to baseline post-operative facial nerve function [14, 77–80, 82]. The etiology of delayed facial palsy is thought to be related to vascular spasm or edema [26, 77]. Treatment with steroids are frequently utilized based on Bell's Palsy Clinical Guidelines [83], although data supporting efficacy specifically for skull base surgery is lacking [77]. Surgical approach may play a role as the use of the retrosigmoid approach is associated with greater rates of delayed facial palsy relative to the translabyrinthine approach, potentially due to the greater length of facial nerve that is decompressed in the translabyrinthine approach, which may mitigate the effect of edema [77].

Surgical Options for Facial Reanimation in Short-Term Facial Palsy (< 2 Years)

Reinnervation of native facial musculature is feasible within 18–24 months from onset; longer periods of facial paralysis lead to chronic denervation and irreversible muscle fibrosis [63••]. Smile restoration and midfacial function can be restored through a trigeminal to facial nerve (V–VII) transfer. The masseteric branch of the trigeminal nerve is favored due to its close proximity to the facial nerve, similar diameter, and minimal donor-site morbidity [63••, 84, 85]. The masseteric nerve is reliably found in the subzygomatic triangle and is coapted to branches of the facial nerve innervating midface and smile (Fig. 1) [68••]. Advantages of the masseteric nerve transfer include low rates of complication, early recovery of facial movement, good resting tone, volitional smile, increased dynamic symmetry, and commissure excursion [63••, 68••, 85–90, 91•]. As only one distal facial nerve branch is used for a trigeminal to facial nerve transfer, concurrent recovery through the native facial nerve remains possible. Dual facial reanimation techniques using both cable grafting at the skull base and masseteric nerve transposition have been published in 13 cases and may improve dynamic movement [92, 93]. Careful assessment of masseter function and a thorough cranial nerve exam should be performed upon discussing facial reanimation surgery options with the patient.

Hypoglossal to facial nerve (XII–VII) transfer differs from the masseteric to facial nerve transfer in that the trunk of the

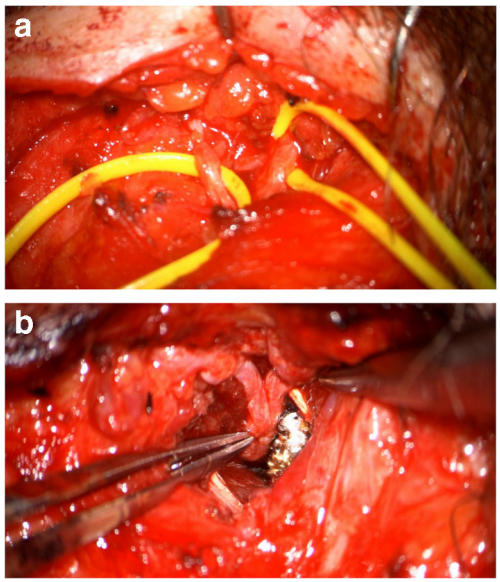

Fig. 1 Masseteric to facial nerve transfer. **a** Facial nerve branches innervating smile identified with vessel loops in the subzygomatic space upon exit from the anterior border of the parotid gland. **b** Subzygomatic triangle dissection of masseteric branch of the trigeminal nerve with forceps at site of neurorrhaphy to facial nerve branches. In this case, two midfacial branches were coapted to the proximal masseteric branch as the patient had a transected facial nerve at the brainstem, which was concurrently repaired with an interpositional cable graft

facial nerve is typically sacrificed and thus no native spontaneous facial movement will result. Historically, the XII–VII transfer involved greater post-operative morbidity as the entire hypoglossal nerve was sacrificed for coaptation with the facial nerve. While excellent facial tone can be achieved and newer techniques sparing complete hypoglossal transection have been described to reduce tongue morbidity [68••, 94, 95], any injury to the hypoglossal nerve may be complicated atrophy of the ipsilateral tongue resulting in difficulties with speech, mastication, and swallowing [94, 95]. Caution should be used in patients with multiple cranial nerve deficits; diagnosis of neurofibromatosis II is a contraindication for hypoglossal to facial nerve transfer due to a risk of future dysphagia and dysarthria.

Future advances in nerve identification may augment our ability to identify distal nerve branches in facial nerve reanimation procedures. The use of a fluorescent peptide binding probe has been shown to improve intraoperative visualization of chronically denervated (up to 9 months) nerves in a live mouse model [96]. The development of this technology is ongoing in a multidisciplinary clinical trial.

Treatment of Longstanding Facial Palsy

After 18–24 months, reinnervation of native facial musculature with nerve transfer is no longer a viable option due to chronic denervation and muscle fibrosis. Alternative muscle sources such as regional or free muscle transfers should be considered for smile reanimation.

Regional Muscle Transfer

Temporalis muscle transfers are a good option for restoration of a volitional (non-spontaneous) smile in longstanding facial palsy. In the orthodromic approach, the temporalis tendon is elevated off the mandible and a portion of the coronoid process is transected to release the superior attachment of the tendon. The tendon is pulled inferiorly and inset at the modiolus (fusion of lip elevator and depressor muscles lateral to the oral commissure) [97–99]. The antidromic approach involves reflecting the middle third of the temporalis muscle either over the zygomatic arch or following resection of the zygoma but may leave a depression in the temporal fossa and a bulge over the zygomatic arch [97]. While temporalis transfer provides support to the midface and oral commissure, volitional smiling is bite-activated and requires facial rehabilitation for optimal outcomes.[97]

Free Muscle Transfer

Gracilis free muscle transfer is widely regarded as the gold standard smile reanimation procedure for longstanding or irreversible facial paralysis[100, 101] and may be employed in patients without major medical comorbidities who can tolerate microvascular surgery. A reliable procedure with low failure rates, the gracilis free muscle transfer will produce a meaningful smile (at least 3 mm of excursion with smile) in 84% of recipients when driven by the contralateral facial nerve via a cross-face nerve graft and 94% of recipients when driven by the ipsilateral trigeminal nerve [100].

A single-stage gracilis free muscle transfer is driven by the ipsilateral masseteric nerve and produces a volitional smile. As with any trigeminal nerve–driven smile reanimation (similar to the 5–7 nerve transfer or temporalis transfer), facial rehabilitation is key to successful smile outcomes [98].

Spontaneous smile reanimation typically involves a two-stage procedure, whereby a donor branch of the contralateral healthy facial nerve is coapted to a cross-face nerve graft (usually sural nerve) without a distal coaptation. After 6–9 months or advancing onset of a Tinel's sign (elicited by tapping on the distal end of the cross-face nerve graft with a referred sensation to the neurorrhaphy site), the gracilis free muscle transfer surgery can take place wherein the distal nerve graft is coapted to the donor flap. Patients should be informed that the onset of movement may take up to 12–18 months following the second stage and may strengthen over time [102].

Dual innervation using both methods has also been described [100, 103–105]; the optimal neural source, number of stages, and coaptation patterns remain a source of debate [84, 105, 106]. In patients with suboptimal esthetic outcomes from midfacial bulk or oral commissure, malposition may be safely addressed in a small revision surgery without endangering the neurovascular pedicle [107].

Management of Upper Facial Paralysis

Brow ptosis can obstruct vision and worsen ocular irritation. Static correction can be performed with a brow lift using a minimally invasive approach in the office under local anesthesia [108, 109]. Of note, a direct brow lift [63••] may be insufficient to bear the facial soft tissue weight in facial paralysis patients, and the power brow technique with suture suspension to a titanium mini-plate is preferred for optimal outcomes [108]. Treatment of lagophthalmos can be achieved by eyelid weight placement or tarsorrhaphy [73, 74•]. Ectropion caused by lower eyelid laxity can be treated with tarsal strip suspension, lateral or medial canthoplasty [74•], or a fascia lata lower eyelid sling [63••].

Management of Midface Paralysis

Due to the weight of the facial soft tissue in facial paralysis, rhinoplasty techniques to correct external nasal valve collapse may be insufficient [63••]. Static suspension of the nasal accessory cartilages within the nasal ala to the true temporalis fascia with fascia lata provides superior relief of nasal

obstruction and improves symmetry of the nasolabial fold, supporting the midface and oral commissure [63••, 76, 110•]. Patients with an overprominent nasolabial fold related to hyperkinetic or synkinetic movement can undergo nasolabial fold effacement procedures [76]. Smile reanimation should be addressed through regional or free muscle transfers as detailed above.

Management of Lower Facial Paralysis

Paralysis of the oral depressor muscles leads to an asymmetric smile as well as deficits in oral incompetence, speech, and emotional expression [111]. In flaccid facial paralysis, the lip and oral commissure are typically inferiorly malpositioned, and philtrum skew may occur in response to pull from the healthy hemiface; this can be corrected with either fascia lata or suture static suspension [63••]. In nonflaccid facial paralysis, hyperkinetic contraction of the oral depressors can be disfiguring. Patients with a prior history of satisfactory response to local anesthesia or botulinum injection of the oral depressors may undergo selective myectomy of the affected depressor anguli oris, mentalis, or buccinator muscles for a permanent effect[112].

Injected Chemodenervation

Injected chemodenervation agents such as botulinum toxin represent a valuable component in the management of facial palsy and may be used on either side of the face to balance facial asymmetry [63••, 113, 114•, 115, 116]. Effects are temporary, but injections can be performed routinely in clinic, are well tolerated, and show significant improvement in facial symmetry and quality of life [114•, 116–118]. Complications are rare, including ptosis or facial droop, and are related to larger doses. However, symptoms are often mild and self-resolving as the effect of the toxin decreases [113, 118].

In nonflaccid facial paralysis, injection is used for hyperkinetic or overactive movement of facial muscles and synkinesis. Chemodenervation in appropriately targeted synkinetic muscles has been reported to improve facial symmetry and reduce involuntary muscle contractions [113, 114•, 116, 118]. Assessments on subjective morbidity in nonflaccid facial palsy prior to and after botulinum treatment show significant improvement in quality of life, social function, facial comfort, and perception of self-image [118]. The optimal results of facial function can be gained by timing facial rehabilitation training after chemodenervation and should be coordinated with a physical therapy team specialized in facial nerve management.

Conclusion

Conservative treatment approaches to vestibular schwannoma have been developed and accepted in an effort to prioritize preservation of facial nerve function in the management of this benign condition. The rates of post-operative facial palsy are highly variable, but recovery is expected in the majority of patients over the months following surgery. The absence of any detectable improvement in facial paralysis by 6 months is independently predictive of permanent paralysis, and reanimation procedures should be considered [68••]. Early referral to a facial reanimation surgeon is recommended for prompt assessment and intervention, followed by long-term monitoring for the evolution of symptoms and refinement of individualized treatment plans. The management of facial palsy entails physical therapy, injectable treatments, and various surgical reanimation procedures. Treatment is aimed at optimization of facial symmetry and restoration of smile and has been effective in improving esthetic appearance, functional deficits, and quality of life in patients with facial palsy.

Declarations

Conflict of Interest The authors declare no competing interests.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- • Of importance
- •• Of major importance

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