Complications and Cranial Nerve Rehabilitation

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Advances in microsurgical techniques have reduced the morbidity and mortality in surgery of the cerebellopontine angle (CPA) significantly. Nevertheless, surgical complications involving the intricate neurovascular structures or multiple cranial nerves of the skull base can occur. Surgeons can minimize complications with thorough knowledge of the relevant skull-base anatomy and its relationship to the targeted pathologic lesion, collaboration between members of a skull-base team, careful review of preoperative imaging, the use of intraoperative monitoring techniques, and well executed surgical techniques. Mortality for lesions of the CPA such as vestibular schwannoma (VS) in contemporary series is now very rare, well less than 1%, even for very large tumors [1–3]. This has shifted the focus of further complication prevention to other morbidities such as cerebrospinal fluid (CSF) leaks and the preservation and rehabilitation of cranial nerve dysfunction.

The management of complications begins with careful observation and recognition of common pitfalls in surgery and during the postoperative course. Anticipation of potential complications and prompt diagnosis can often limit the magnitude morbidities and allow for faster recovery. Different pathologic entities of the CPA and each surgical approach can be associated with particular complications. In a retrospective analysis of a large multi-institutional quality improvement database, overall complication rates of VS resection did not differ among transtemporal, retrosigmoid, or middle cranial fossa approaches [4]. While complications can be difficult for both the patient and the surgeon, continuing attentive care is important and may help avoid compounding an existing problem or missing secondary complications.

Cerebrospinal Fluid Leak and Meningitis

One of the most frequent complications of CPA surgery is postoperative CSF leak, which may manifest through different routes including through the primary wound, as otorrhea or as rhinorrhea. In a retrospective review of 300 surgical cases (100 of each surgical approach: translabyrinthine, middle fossa, and retrosigmoid), a leak rate of 10–13% was found, and neither approach nor tumor size was a factor in the rate of postoperative CSF leak nor the need for revision surgery [5]. A meta-analysis of 5964 cases similarly found no differences between surgical approaches, with rates of 9.5–10.6% reported [6]. A number of different surgical techniques have been suggested to help reduce the incidence of CSF leak after CPA surgery. Proposed techniques have all reported good outcomes, though preferences and surgical details vary significantly in different authors' hands—for example, in the degree of middle ear and eustachian tube manipulation, use of titanium mesh/hydroxyapatite cement, and obliteration in translabyrinthine approaches [7–10]. Abdominal fat grafting is a commonly used method that has reduced the rate of CSF leak in these surgical techniques, and care should be taken to meticulously close and monitor the donor site for rare but potential wound site morbidities, such as subcutaneous hematoma, which has been reported in approximately 3% of cases [2, 10, 11].

The management of a postoperative CSF leak often utilizes a graduated approach from conservative measures to surgical interventions [12]. Conservative measures may include bed rest, head of bed elevation, acetazolamide, wound oversewing, and pressure dressings. CSF diversion, such as with a lumbar drain, is an additional technique at times employed before pursuing surgical options that may include wound re-exploration, subtotal petrosectomy, or

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other transnasal approaches to eustachian tube closure [13, 14]. Infrequently, lumbar drain diversion in the setting of an active CSF leak can result in increasing pneumocephalus, an example of which is shown in a case in Fig. 15.1, which resolved after subtotal petrosectomy and plugging of the eustachian tube.

Meningitis can be a serious and threatening complication of skull-base surgery. Although uncommon overall, the incidence of meningitis seen in a systematic review assessing postoperative CSF leaks noted a significantly higher risk of meningitis in the presence of a leak, occurring in 14% of these cases [6]. Allen and colleagues reported on 508 lateral skull-base procedures, identifying meningitis in 3.1% with a median time from surgery to onset of 12 days and a much higher risk of meningitis in the presence of a CSF leak by a factor of 10.2 [15]. Higher rates of meningitis have been reported, including a 9.2% incidence seen in a series of 500 cases [16].

Many cases of meningitis postoperatively are likely chemical meningitis with symptoms of headache or fever but negative CSF Gram stain and cultures. In a study of 1146 patients investigating the differences between bacterial and chemical meningitis, an overall incidence of meningitis in 4.54% of cases was found [17]. Culture-proven bacterial meningitis was seen in 0.87% and, when combined with strongly suspected bacterial cases, had a total incidence of 1.92%, with the remaining 2.6% of cases suspected to be chemical. The bacterial meningitis cases had significantly higher CSF and serum white blood cell counts and lower CSF glucose. For CPA pathologies, notably epidermoid cysts of the skull base, cyst rupture and exposure of proteinaceous contents to the subarachnoid space may be particularly likely to trigger recurrent episodes of aseptic meningitis [18]. Care should be taken intraoperatively to avoid contamination of the CSF with byproducts such as bone dust from any drilling during skull-base approaches.

Vascular Complications

A vascular injury or complication after skull-base surgery is a dreaded and potentially devastating outcome. Microsurgery in the CPA involves careful dissection of and around critical structures, including segments of the carotid artery, vertebrobasilar system, venous sinuses, jugular bulb, brainstem perforators, and other critical structures. Intraoperative bleeding from vessels or the venous dural sinuses can be controlled with gentle pressure using thrombin-soaked gelatin or other absorbable hemostatic agents external to the vessel, with the goal of controlling bleeding while avoiding vessel occlusion or complete thrombosis.

A postoperative hematoma within the CPA can occur in the early recovery period and can vary greatly from a minor collection to a rapidly evolving and compressive hemorrhage. Larger or threatening hematomas may require immediate surgical evacuation with a thorough inspection of the

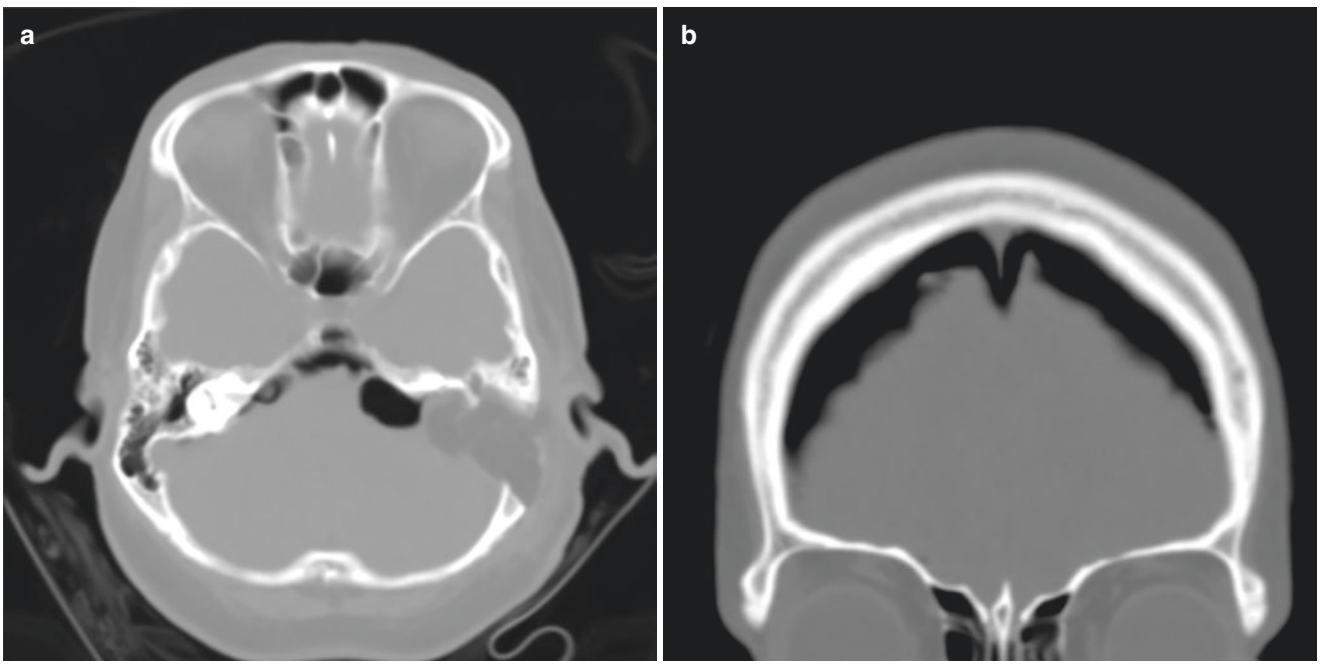

Fig. 15.1 (**a**) Axial and (**b**) coronal computed tomography images after left translabyrinthine approach to resection of a large left-sided vestibular schwannoma, showing worsening pneumocephalus after lumbar cerebrospinal fluid (CSF) diversion for postoperative CSF rhinorrhea. The patient underwent left subtotal petrosectomy with obliteration of the middle ear and plugging of the eustachian tube with resolution of the CSF leak and pneumocephalus

surgical field to identify a source of hemorrhage (Fig. 15.2). Postoperative hemorrhage or hematoma formation is rare, having been reported in approximately 1–3% of cases postoperatively [2, 16, 19].

Portions of the vertebrobasilar arterial system, brainstem perforators, and the anterior inferior cerebellar artery (AICA) in particular are often encountered in surgery of the CPA. The close relationship between branches of the AICA and the seventh and eighth cranial nerves make it a vulnerable structure for many pathologies of the CPA or in some rare cases of an aneurysm of AICA [20]. The labyrinthine or internal auditory artery can be injured, or potentially acute vasospasm may be initiated from surgery, leading to auditory and vestibular symptoms of hearing loss, tinnitus, and vertigo. Occlusion or injury to the AICA can also clinically manifest with other severe neurologic sequelae, including dysarthria, ipsilateral facial palsy, facial sensory loss, Horner's syndrome, dysmetria, contralateral loss of sensation, ipsilateral conjugate lateral gaze palsy, dysphagia, and ipsilateral motor weakness.

The dural venous sinuses and bridging veins are at risk during lateral skull-base surgery, particularly in combined middle and posterior fossa transpetrosal approaches [21]. The anastomotic vein of Labbé can have variable anatomy but bridges the lateral cortical temporal lobe to the transverse sinus and can potentially be the sole route of venous drainage for a large region of the temporal and parietal lobes. Vein of Labbé or other lateral temporal skull-base bridging vein injuries can cause severe neurologic consequences due to temporal lobe ischemia, edema, or infarction, including nausea, vomiting, aphasia, and hemiparesis [22]. Preoperative planning and careful division of the superior petrosal sinus and tentorium a distance from the variable venous bridging anastomoses near the transverse sinus can help to avoid injuries and complications. If the vein is severed intraoperatively, it can be reconstructed or revascularized, such as with a saphenous vein graft [23].

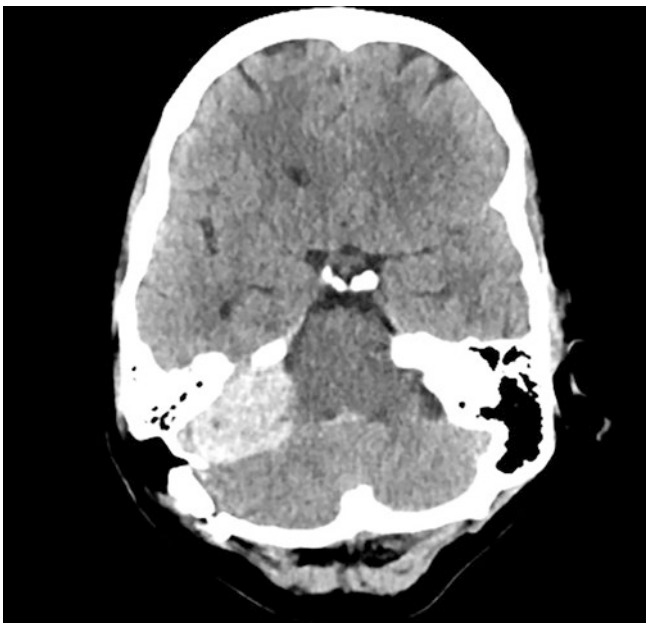

Fig. 15.2 Axial computed tomography image shows a post right-sided cerebellopontine hematoma, in this case requiring surgical evacuation without residual symptoms

Venous Sinus Thrombosis

Lateral skull-base approaches to the CPA involve careful surgical preservation and meticulous handling of the dural venous sinuses. Thrombosis of these venous sinuses resulting in morbidity or mortality is an uncommon but important complication for which to monitor carefully in the postoperative setting (Fig. 15.3). Occluding thromboses can cause slowly progressive symptoms of increased intracranial pressure, headaches, papilledema, visual disturbance, venous infarction, intracranial hemorrhage, seizures, or other focal neurologic deficits. In extremely rare cases, involvement of the superior sagittal sinus can lead to mortality [24].

Retrospective reviews of postoperative sinus thrombosis have reported an incidence ranging from 4.6% to 11.6%, presenting as early as the day of surgery to 40 days postoperatively [25–27]. Ohata and colleagues also noted that two cases of thrombosis were only noted on follow-up imaging 5.4 and 6.4 years postoperatively. Retrospective studies tend to report cases found due to clinical symptoms or intraoperative findings, likely underreporting the true incidence as many cases of smaller or partial thromboses may remain asymptomatic. In a prospective study with planned preoperative and postoperative magnetic resonance venography (MRV) in a series of lateral skull-base cases, 31.9% of patients showed some radiographic signs of at least a partial filling defect, though all of these patients were asymptomatic and none required anticoagulation [28].

Treatment strategies for diagnosed postoperative thromboses vary greatly and are often tailored to the case based upon surgical findings and patient symptoms. Conservative measures including observation, hydration, corticosteroids, and acetazolamide have been used. A much larger body of evidence exists for spontaneous or trauma-induced dural venous thrombosis for which anticoagulation is recommended [29, 30]. However, in the setting of recent intracranial CPA surgery, treatment of dural sinus thrombosis with systemic anticoagulation is weighed against the risks of postoperative intracranial hemorrhage. In some cases with symptom deterioration, other management options available include endovascular therapies, such as thrombolysis or mechanical venous thrombectomy, and CSF diversion, such as a ventriculoperitoneal shunt [31].

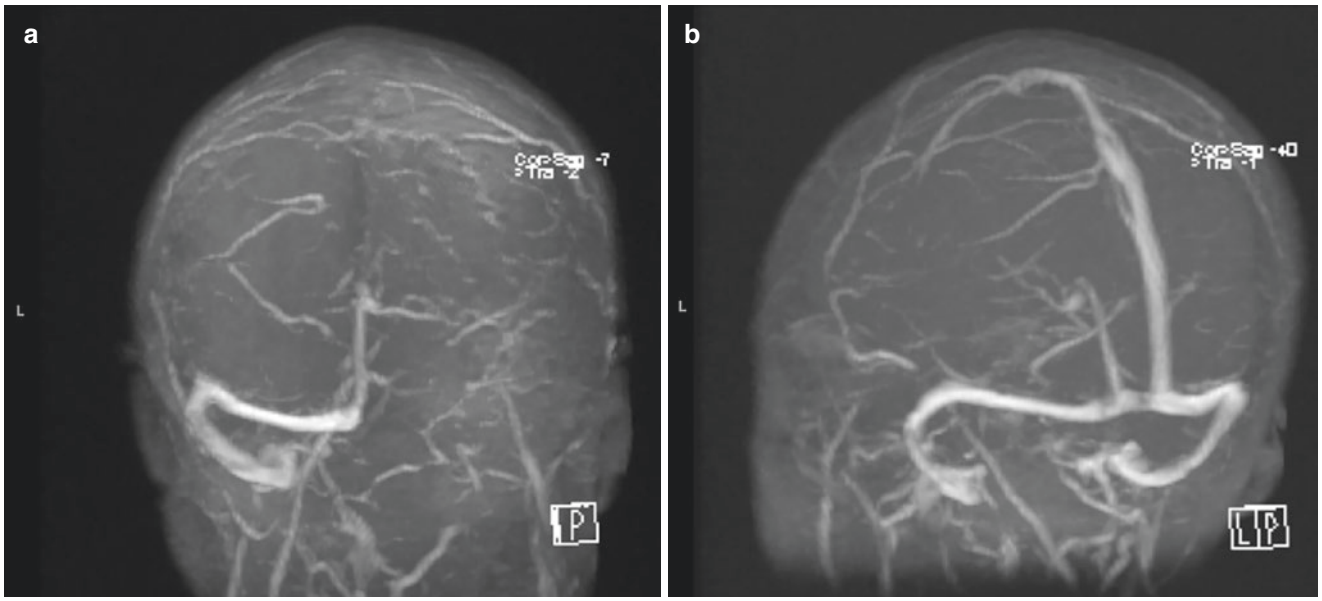

Fig. 15.3 (**a**) Magnetic resonance venography (MRV) after the resection of a right-sided vestibular schwannoma reveals a filling defect extending from a portion of the superior sagittal sinus into the right transverse sinus, sigmoid sinus, and jugular vein. (**b**) A follow-up MRV 1 year later revealed normal flow throughout the dural venous sinus system

Cranial Nerve Dysfunction

Microsurgical approaches to tumors within the CPA strive to provide complete removal of lesions while preserving the multiple cranial nerves that may be locally involved. These nerves may be already affected by the skull-base tumors preoperatively or altered by intraoperative dissection. Hearing loss is a very common presenting symptom of CPA lesions and, when feasible, hearing preservation approaches to save residual hearing and the cochlear nerve are undertaken. Other chapters of this text address hearing preservation and rehabilitation options after lateral skull-base surgery. A review of other potential cranial nerve neuropathies follows.

Vestibular Dysfunction

The vestibular nerves are commonly involved, depending on the nature of the pathologic lesion. In VS surgery, typically already dysfunctional vestibular nerves are sectioned resulting in complete loss of any residual ipsilateral vestibular function. In these cases, improvement in balance and dizziness may require adaptation and vestibular compensation over a significant period of time. Although hearing loss and facial nerve function are deservedly critical considerations in CPA surgery, in VS patients, ongoing dizziness was associated with the greatest reduction in quality-of-life measures [32].

In a study of 48 patients operated on for VS and assessed with vestibular testing and the Dizziness Handicap Inventory, 71% of patients had no change or reported improved equilibrium postoperatively [33]. Preoperative serviceable hearing, cystic transformation, normal cervical vestibular evoked myogenic potentials (cVEMPs), diplopia, or other vestibular syndromes were predictive of having worse equilibrium postoperatively, while having preoperative vestibulopathy (caloric weakness >75%) was a good prognostic factor. Similarly, in another study of 81 patients who had undergone surgery for VS, abnormal preoperative vestibular testing measures—such as caloric weakness, abnormal positional nystagmus, and abnormal vestibulo-ocular reflex (VOR) asymmetry on rotary chair testing—correlated with lower postoperative disability [34]. Patients with preoperative dysfunction may have already completed some degree of compensation compared to patients with normal function who suffer an abrupt change and complete loss of ipsilateral vestibular function in the acute postoperative period. In a review of 210 patients operated on for VS via a retrosigmoid approach, 31% were noted to have dysequilibrium lasting greater than 3 months postoperatively, based upon a retrospective chart review of documentation for recorded symptoms [35]. In a follow-up study by the same group, completed by surveying the patients, 65% reported some degree of persistent dysequilibrium postoperatively [36].

Vestibular rehabilitation is considered beneficial to reduce the duration of symptoms and for overall equilibrium after CPA surgeries that may disturb balance. However, existing reports may not always show the benefit, with noted limitations in selection bias as typically patients with greater struggles with equilibrium are prescribed or choose to pursue

vestibular rehabilitation programs while those with no symptoms do not [33]. The use of brief periods of vestibular exercises even in the very early stages of recovery after CPA surgery may be beneficial to postoperative patients [37]. The use of simple VOR exercises and education has also been studied and shown to help improve the rate of compensation in the absence of a formal physical therapy program [38]. Patients receiving therapy have reported a longer time period to ambulate independently, though this may be due to accentuated symptoms in the acute period due to exercises, and 89% of patients reported they felt the exercises to be helpful in regaining their balance [34]. In a prospective randomized controlled trial comparing general instructions to a customized vestibular rehabilitation protocol for 12 weeks, younger patients <50 years improved significantly with either intervention, but patients over the age of 50 years receiving customized vestibular rehabilitation showed improved balance test results compared with those only given general instructions [39].

Facial Nerve

Preservation of facial nerve function is a critical goal in lateral skull-base surgery as facial nerve paresis can be a disfiguring and difficult complication. The facial nerve is most commonly injured at the porous region, and careful avoidance of trauma caused by suction on the nerve is important [40]. Microsurgery for vestibular schwannoma has a varied rate of reported facial nerve preservation. In a series of 162 consecutive patients with small tumors undergoing excision through a middle cranial fossa approach, House–Brackmann (HB) grade I or II was achieved in 97% of cases [41]. In a retrospective review of 410 cases operated on with a variety of approaches, good facial nerve function (HB I or II) was found in 86% of patients immediately postoperatively, and 58.9% of those with initially poor function would go on to improve during follow-up [42]. In 1052 patients with anatomically preserved facial nerves and total tumor removal, 65% maintained HB grade I or II, with another 29.4% with HB grade III [43]. In a prospective cohort including tumors of all sizes, 73% of patients had good facial function at postoperative day 180 [44]. Even for large vestibular schwannomas undergoing surgical resection, good facial nerve function has been reported in up to 88% of patients [45, 46]. In a systematic review of the literature that included an analysis of 11,873 patients, facial nerve preservation (HB I or II) was found in 78–85%, varying by approach. Although rare, in some complex skull-base lesions with significant vascularity, preoperative angiogram, and embolization help reduce blood loss and facilitate surgical resection but carry a small risk of cranial nerve dysfunction, including facial palsy [47, 48].

Delayed Facial Paralysis

In a subset of patients in whom the facial nerve is anatomically preserved and who awaken with good facial nerve function, delayed onset palsy may occur. A wide range of rates of delayed palsy has been reported, with varying definitions regarding the initial postoperative facial function and timing of worsening. A bimodal distribution of cases of delayed onset has been reported—those within the first 2 days and another group a week or more later [49]. In a series of 129 patients undergoing VS resection, a 29% incidence of delayed facial nerve dysfunction was found and primarily occurred within the first few postoperative days [50]. In this series, neurophysiologic stimulation parameters did not differ between patients with delayed palsy and those without. Those with delayed palsies had an excellent prognosis, with 89% recovering to HB grade I or II by 1 year. Similarly, a large series of 255 consecutive patients was evaluated to identify a 24.3% rate of delayed facial paresis occurring at an average of 3.65 postoperative days [51]. The vast majority (90%) ultimately recovered to their initial postoperative HB grade. In a review of 314 consecutive patients, excluding cases of early onset weakness (within 48 h) and counting only delayed facial weakness >72 h postoperatively, a 4.8% rate of delayed facial palsy was found [52]. These patients also had excellent recovery over time, with 93% recovering to HB grade I or II by 1 year. In a review of 489 patients that focused on facial deterioration of at least two HB grades from postoperative days 5 to 30, 16% of cases experienced a delayed weakness.

Viral reactivation is a proposed mechanism for the delayed palsies. In a prospective study of 20 patients undergoing vestibular schwannoma surgery, seven patients developed postoperative delayed facial weakness. Serum IgM titers of herpes simplex 1, herpes simplex 2, and varicella zoster virus were found to be significantly more elevated postoperatively in those with delayed palsies than those without, and IgG titers did not differ significantly [53]. However in another small series in which three patients had serology, IgG had a significant increase without IgM changes [54]. Perioperative administration of famciclovir has been suggested to reduce the risk of delayed facial palsy. Patients undergoing VS surgery who received famciclovir showed potential benefit compared to a historical group without pretreatment [55].

Facial Paralysis and Eye Complications

In cases of facial nerve weakness or paralysis, ocular complications can arise due to weakened musculature as well as potential lacrimal dysfunction from nervus intermedius dysfunction. Lagophthalmos causes poor distribution of tears that

may lead to exposure keratitis and corneal injury. In some cases of larger CPA lesions, trigeminal nerve dysfunction with resultant corneal hypoesthesia can further contribute to eye complications. In one review, up to 44% of patients undergoing VS surgery required some form of ophthalmologic procedure postoperatively, though in that series a high rate, 70%, of cases had facial palsy [56]. Patients with trigeminal involvement and corneal hypoesthesia are at highest risk to develop corneal pathology [56, 57]. A more contemporary review of 174 patients undergoing VS resection reported a much lower incidence of the need for ophthalmologic referral (7.6%) and only 5.3% of patients requiring an ophthalmic procedure [58]. Treatment for the eye most commonly includes an upper eyelid loading procedure to address lagophthalmos and potentially a lower eyelid tightening procedure.

Contemporary care of facial nerve paralysis after skull-base surgery has incorporated a wide range of advancements in techniques: free muscle transfer, masseteric to facial nerve transfer or as a source of innervation for microvascular free flaps, cross-face grafting, static procedures, and botulinum toxin injections [59–61]. Advanced techniques in microsurgical excision of lateral skull-base tumors have led to improved facial nerve results. However, in the cases of complete facial paralysis postoperatively, even in the setting of an anatomically preserved facial nerve, early assessment of recovery and consideration of intervention by 6 months may be considered [62]. Facial nerve reanimation is discussed in greater detail in another chapter of this text.

For patients with facial paralysis and a challenging path to recovery, facial nerve rehabilitation therapy in addition to medical and surgical treatment is often utilized. Physical therapy can be applied in various forms including mime therapy, neuromuscular retraining, massage therapy, and mirror feedback for coordination of exercises. These approaches have been utilized to promote function and inhibit or control synkinesis [63, 64]. Specialized therapy can be a useful adjunctive therapy in cases of postoperative facial paralysis and improves patients' quality of life [65, 66].

Lower Cranial Nerves

Extensive lesions of the CPA, such as those involving the jugular foramen, can place the lower cranial nerves at risk during surgery. Pathologies such as meningiomas of the CPA or jugular foramen and other tumors of the jugular foramen, such as paragangliomas or schwannomas of the cranial nerves themselves, may present preoperatively with neural dysfunction and may be challenging or, at times, impossible to excise without changes to lower cranial nerve function (Fig. 15.4).

A high vagal lesion impairs the larynx and pharynx both by the loss of motor function and a loss of sensory innervation. This combination leads to ipsilateral vocal fold paralysis and associated dysphonia as well as dysphagia and a high risk of aspiration. Voice and swallowing dysfunction in the

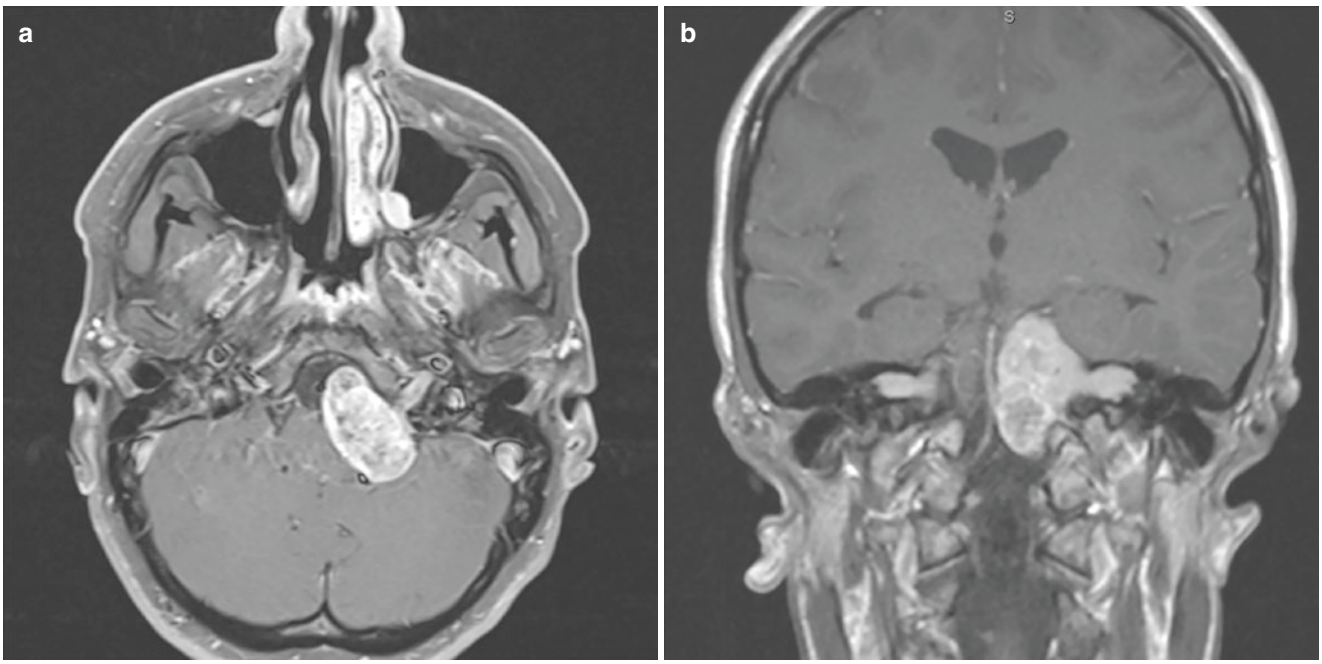

Fig. 15.4 T1 postcontrast magnetic resonance images in the (**a**) axial and (**b**) coronal planes of a patient with neurofibromatosis type 2, with multiple cranial nerve schwannomas, hearing loss, left vocal fold paralysis, and hypoglossal nerve paralysis due to collision tumors involving the left vestibular nerve and lower cranial nerves

immediate postoperative period likely varies based upon particular CPA tumor characteristics and extension but was found in 10% of one series of CPA surgeries [67]. CPA tumor size was found to be an independent risk factor for postoperative vagal palsy. In a series of 50 patients with giant VS, 10% of patients had lower cranial nerve dysfunction preoperatively and 6% developed new dysfunction postoperatively, though most recovered over time [1]. Although the severity of symptoms for a vagal lesion can vary greatly, it appears the timing of onset plays a role. Sudden onset high vagal paralysis, such as that induced as a result of skull-base surgery, is associated with a higher rate of tube feeding in comparison to a more gradual onset of paralysis, such as from progressive tumor effect [68]. Patients with normal function preoperatively and sudden loss of vagal function postoperatively have no time to adapt or modify their swallowing behavior, leading to abrupt dysphagia and the potential need for an alternative route of feeding or other interventions. Palsies induced slowly by tumor growth provide a longer period for compensation and swallowing modification. Surgical resection affecting additional combinations of the lower cranial nerves, including the glossopharyngeal and hypoglossal nerves, or other neurologic changes, such as in cognitive status, may further impair the patient's ability to compensate.

Laryngeal procedures to help address dysphonia and dysphagia include laryngeal framework surgery, such as medialization thyroplasty, or vocal fold injection using a variety of materials. In a review of 35 patients with high vagal lesions due to a variety of causes in addition to skull-base surgery, 14 patients had tracheotomies before any laryngeal interventions, but 11 of these 14 patients were able to be decannulated after laryngeal framework surgery [69]. The procedures included medialization thyroplasty with or without arytenoid adduction or cricopharyngeal myotomies. Most of those patients (94%) had some improvement in aspiration, though 19% continued to require a feeding tube. Fang and colleagues reviewed high vagal lesions also of various causes, including CPA surgery, and found the feeding tube dependency rate improved from 27% to 5.9% after laryngeal interventions. Medialization thyroplasty has been previously suggested to be performed primarily at the time of skull-base surgery to provide immediate improvement and avoid the need for tracheotomy, though other authors note final vocal cord status is difficult to judge immediately and suggest injection laryngoplasty for short-term improvement, with further surgery determined when the cord position is constant [68, 70]. Early arytenoid adduction as the sole method of medialization by postoperative day 2 has also been reported in a series of 26 patients as another option for early rehabilitation. In that group, 62% of patients required a percutaneous feeding tube after skull-base surgery, though all but one was able to resume nutrition by mouth with removal of the feeding tube by 1 year, and 33% of patients required tracheotomy, all of whom were decannulated by 3 months [71].

Headache

Patients with CPA lesions such as VS, even untreated, are more likely than controls with tumors to have severe headache disability, and 60% overall report some degree of headache before treatment [72]. Headache disability outcomes have not been shown to be statistically different between the treatment modalities of observation, stereotactic radiosurgery, and microsurgery.

In studies of CPA surgery approaches, retrosigmoid craniotomy has frequently been identified as having a greater risk of postoperative headache in comparison to other lateral skull-base approaches. In a systematic review of surgery for VS, the retrosigmoid approach was significantly more likely to result in persistent postoperative headache (17.3%) compared to the translabyrinthine (0%) approach but statistically not significantly more than middle cranial fossa cases (8%) [73]. Although most reports describe short-term postoperative headaches, it is possible that long-term outcomes at 1 year may not show significant differences between approaches [74]. Potential causes of this postoperative headache include cervical muscle adhesion and subsequent irritation to exposed dura at the craniotomy site, dural tension, intradural bone dust initiating aseptic meningitis from intradural drilling, and injury to either the lesser or greater occipital nerves from the incision and approach.

To address potential dural adhesions, several authors have reported utilizing replacement of a bone flap or cranioplasty techniques to reduce the incidence or severity of postoperative headache [75–77]. A retrospective review of 565 retrosigmoid approaches to the CPA reported significant differences between the incidence of postoperative headache based on if patients were treated for vestibular nerve section (5%) or removal of VS (54%), suggesting internal auditory canal drilling and dispersed bone dust as the primary difference between those approaches being a significant contributor to the headaches [78]. In addition to intradural drilling, fibrin glue as a cause for aseptic meningitis and tight dural tension from closure has also been considered as factors [79]. The use of Gelfoam to trap residue or bone dust has been advocated as a method to reduce postoperative headaches [80]. Modifications of surgical technique using a curvilinear incision design has also been suggested as a method to avoid occipital nerve injury and headache [81].

For most cases of postoperative headache, simple analgesics such as NSAIDs, COX-2 inhibitors or paracetamol/acetaminophen are effective and used as first-line medical therapy [82]. Sumatriptan has also been described as an

effective treatment in a small study that suggested some cases may have pain mediated by the trigeminal nerve [83]. A subset of the patients with postoperative headaches exhibit occipital neuralgia with occipital neuromas or nerve entrapment and may benefit from procedures targeting these pathologies [84]. Many large tumors of the CPA can also cause direct compression or vascular compression of the trigeminal nerve with associated neuralgia. Microsurgical resection of tumors improves trigeminal neuralgia in the majority of these cases and can alleviate facial numbness and paresthesia in some cases [85, 86].

Conclusion

Although microsurgery within the CPA has seen remarkable advancements in surgical technique and intraoperative monitoring, perioperative complications still occur. Management of potential morbidities often begins during preoperative counseling when the risks and possibilities for a given lesion and surgical plan are fully discussed so that patients understand their risks and may be prepared for the unlikely event that complication is encountered. Preoperative dysfunction of cranial nerves can be recognized, and abrupt postoperative neuropathies, such as in the vestibular system and swallowing mechanisms, can benefit from therapy and compensation. Knowledge of the broad range and likelihood of various complications allows for early identification and treatment. Advancements in rehabilitation techniques and improved options for surgical rehabilitation have led to a trend toward early management of some cranial neuropathies, such as facial paralysis and laryngeal dysfunction, to facilitate the return of function and quality of life for patients.

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