9 Facial Nerve Surgery

Core Messages

- Functional components of the facial nerve include:
  - General efferents to facial muscles
  - Secretomotor preganglionic efferents to the submandibular, lacrimal, and nasal glands
  - Special sensory to taste receptors in the tongue and palate/nasal pharynx
  - Somatic sensory to the skin of external auditory canal and concha
- The motor axons to regional facial muscle groups are mixed, not compartmentalized, through the temporal course of the facial nerve.
- The sensory ganglia of the facial nerve are the geniculate and meatal ganglia.
- Surgery is usually indicated for facial nerve paralysis secondary to chronic otitis media, transverse TB fracture, neoplasia, ear surgery, and pseudotumor.
- Surgery is usually not indicated in idiopathic facial paralysis (Bell's palsy) and longitudinal TB fracture.

9.1 Anatomy of the Facial Nerve

The gross anatomy and functional components of the facial nerve in its intratemporal course have been extensively described in texts of anatomy and otologic surgery. Recent additions to facial nerve anatomy and their impact on disorders of the nerve are briefly reviewed in this chapter.

9.1.1 Organization of the Facial Nerve

After emerging from the brainstem, the facial nerve (FN) travels with the vestibular division of the eighth cranial nerve the length of the internal auditory canal (Fig. 9.1). The FN then enters the labyrinthine segment of the fallopian canal, which conveys it throughout a tortuous course in the TB. The FN is derived from the second branchial arch and innervates structures that are derived from Reichert's cartilage. Four groups of functional neurons constitute the FN complex [15]:

1. The *special efferent* FN axons supply the striated muscles of facial expression, as well as the stapedius muscle, the stylohyoid muscle, and the posterior belly of the digastric muscle.

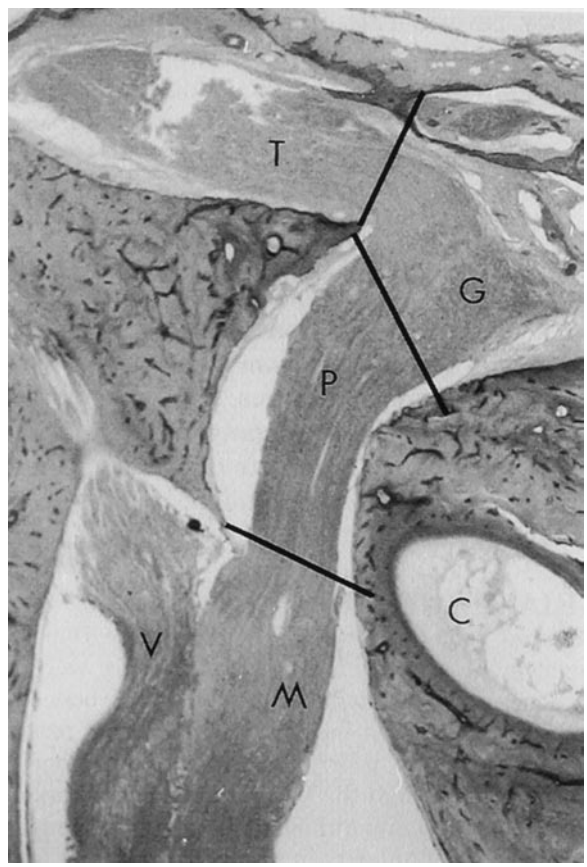

Fig. 9.1 Division of the FN as it enters the fallopian canal. *M* meatal, *P* petrosal, *T* tympanic, *G* geniculate ganglion, *V* vestibular nerve (superior division), *C* basal turn of cochlea

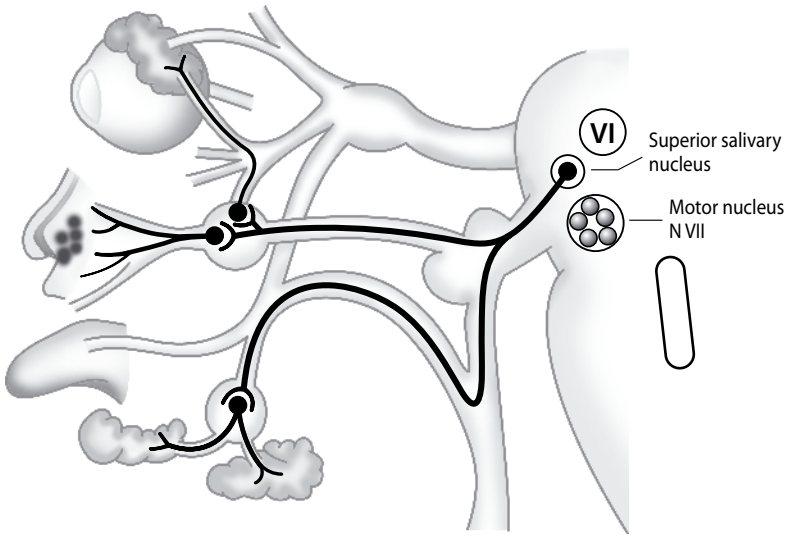

Fig. 9.2 Schematic of the efferent secretomotor pathways in the nervus intermedius

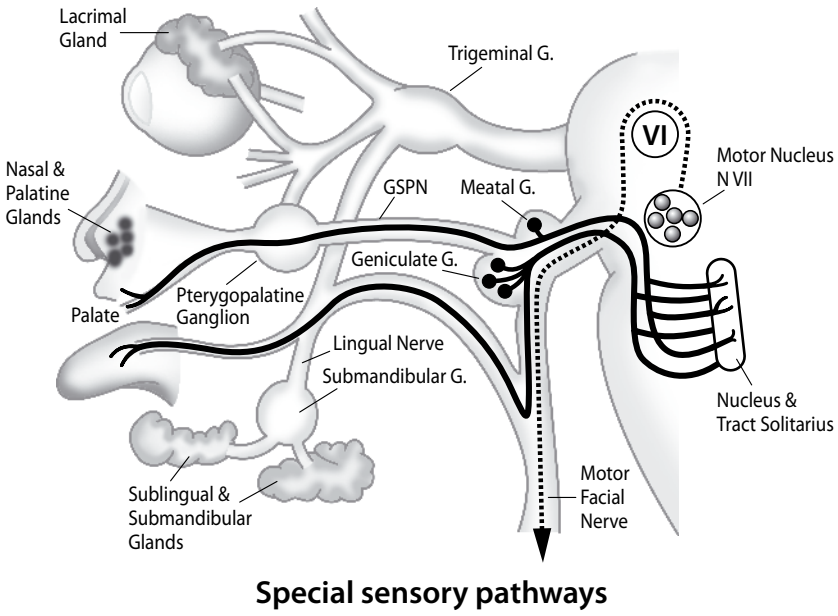

Fig. 9.3 Schema of the special sensory neural pathways serving gustatory function in the FN

2. *General visceral efferent fibers* represent the preganglionic portion of the autonomic pathway to glandular and vascular structures (Fig. 9.2). The main glandular structures are the submandibular, lacrimal gland, and the seromucinous glands in the nasal cavity. Some of these fibers travel in the greater superficial petrosal nerve (GSPN) to synapse in the sphenopalatine ganglion, which contains the postganglionic neurons providing secretomotor function. Secretory fibers are also carried by the chorda tympani nerve and synapse with postganglionic neurons in the submandibular ganglion innervating the submandibular and sublingual salivary glands.
3. *Special sensory fibers* (taste; Fig. 9.3) are carried over two pathways. The majority of the taste receptors inputting to the FN are located in the anterior two thirds of the tongue. Peripheral dendrites sup-

plying these sensory receptors in the chorda tympani nerve join their cell bodies in the geniculate ganglion (GG). A second group of taste receptors is located in the soft palate and nasopharyngeal mucosa and is innervated by fibers in the GSPN, which belong to ganglion cells (meatal ganglion [MG]) located in the meatal segment of the FN.

4. *Somatic sensory neurons* supply the skin of the external auditory canal and the concha.

The brainstem nuclei that give rise to FN axons are:

a. The motor nucleus of the FN, which is located in the caudal brainstem adjacent to the superior olivary nucleus of the auditory system; just caudal to the facial nucleus is the rostral limit of the nucleus ambiguus, which provides motor innervation to the intrinsic laryngeal musculature; the number of facial motor neurons has been estimated at approximately 10,000–20,000; the motor neurons for various facial muscle groups are topographically arranged in subnuclei within the facial nucleus [25]; however, the axons from these subnuclei intermix as they leave the facial nucleus in a dorsal direction to loop around the abducens nucleus near the floor of the fourth ventricle [13]; the axons converge at this point and then bend in a ventrolateral direction just medial to the vestibular nerve (VN) root before exiting the brainstem
b. The location of motor neurons for the stapedius muscle and the posterior belly of the digastric muscle are separately clustered in the brainstem; stapedius motor neurons are located in the interface between the facial nucleus and the superior olivary nucleus, where they are strategically located to receive stimuli from the afferent auditory pathway and carry out reflex contraction of the stapedius muscle (stapedius reflex) [19]; the motor neurons for the posterior belly of the digastric muscle are located along the course of the emerging FN root in the lateral brainstem region
c. The superior salivary nucleus is responsible for secretomotor (autonomic) neurons in the FN system; this nucleus is located dorsally to the motor facial nucleus and gives rise to the preganglionic parasympathetic secretomotor neurons entering the submandibular and the sphenopalatine ganglia
d. The nucleus of the solitary tract, also located in the medulla, receives taste input over sensory fibers of the FN.

The major portion of the FN is composed of motor axons to the facial musculature. Although arising from regional groups of motor neurons in the facial nucleus, these fibers intermix throughout the course of the FN in its intracranial and intratemporal segments [13]. After exiting the stylomastoid foramen, the motor axons gather in functional groups before forming the four to five branches that supply the regional facial muscle groups. For purposes of this discussion, the important divisions of the FN trunk are the meatal segment, the labyrinthine (petrosal) portion, the geniculate portion, and the tympanic part (Fig. 9.1). Except for the meatal portion that lies free in the internal auditory canal, the remaining segments of the FN are contained within a bony canal (fallopian). Accompanying the FN trunk is the nervus intermedius, which carries secretomotor of the preganglionic neurons in the superior salivary nucleus, as well as proximal axons of sensory neurons in the FN ganglia (geniculate and meatal), traveling to the nucleus solitarius in the brainstem.

The sensory ganglia of the FN (geniculate and meatal) are important to the subject of virus-mediated neuropathy (Fig. 9.4). A quantitative study [11] of 100 TB described these ganglionic masses (Fig. 9.5).

e. These two ganglia are derived from different embryologic anlagen, the GG from epibranchial placode (second branchial arch), while the MG develops from the neural crest primordium. In most TB (88%), the GG contains most of the sensory neurons in the FN, while the MG is very small (Fig. 9.6). In approximately 12% of FN, the MG may equal or exceed the number of ganglion cells in the GG. The study found that the number of neurons in the GG ranged from 66 to 4,017 (mean 1,713) while the MG contained from zero to 2,764 cells (448). Fourteen percent of the GG contained less than 1,000 cells, while 88% of the MG contained fewer than 1,000 cells. Sixty-four percent of the MG held fewer than 500 cells, and 34% had less than 200. In approximately 2% of the TB, the MG represents the entire ganglion associated with the FN.

In instances where the GG is absent and the MG represents the only sensory ganglion of the FN [11], TB specimens indicate that the GSPN inputs to the MG. This observation supports a conclusion that the afferent input from taste receptors in the soft palate and nasopharynx is carried over the GSPN to the MG, while the GG contains sensory neurons for taste receptors in the anterior two thirds of the tongue [6]. Furthermore, the MG loca-

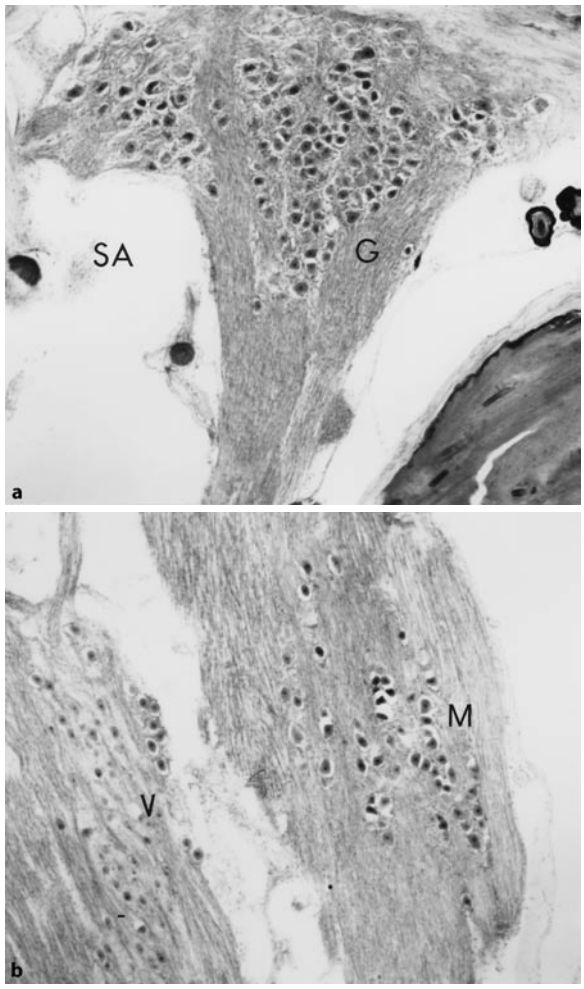

Fig. 9.4 **a** Photomicrograph of a horizontal TB section shows the GG (*G*) and a lateral extension of the subarachnoid space (*SA*). **b** Same TB section shows the facial and vestibular nerves in the internal auditory canal. *V* vestibular ganglion, *M* meatal ganglion

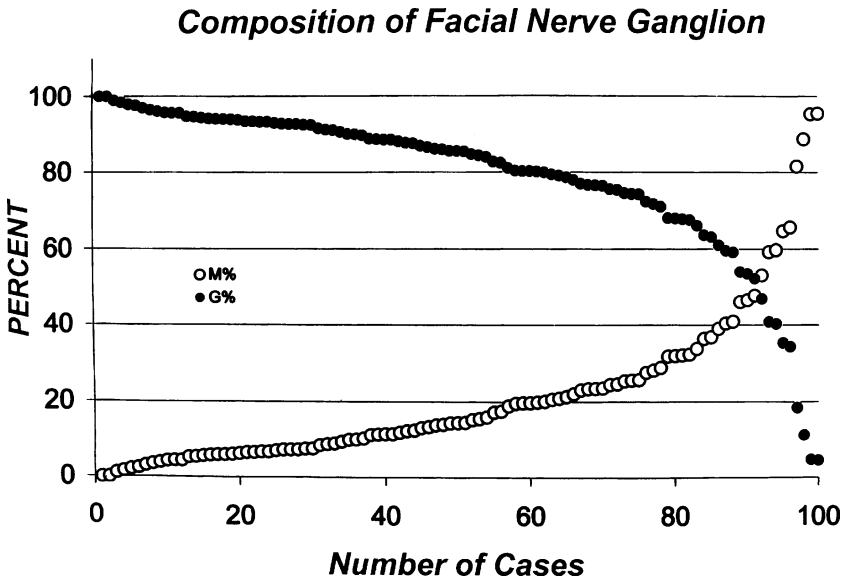

Fig. 9.5 Graph of 100 normal TBs, ordering the percentage composition of geniculate and meatal (*M*) ganglion calls in the FN (*G*)

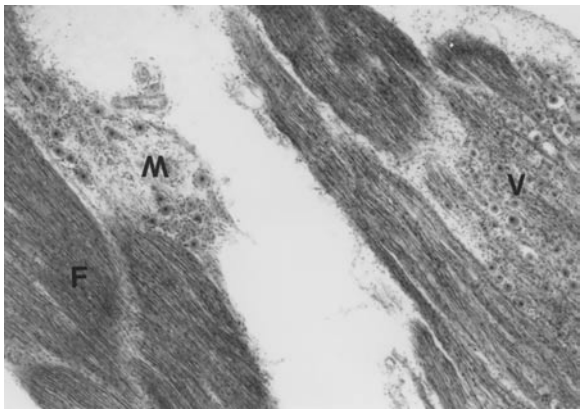

Fig. 9.6 The meatal ganglion (*M*) is usually small as shown in this photomicrograph. *V* vestibular ganglion, *F* FN

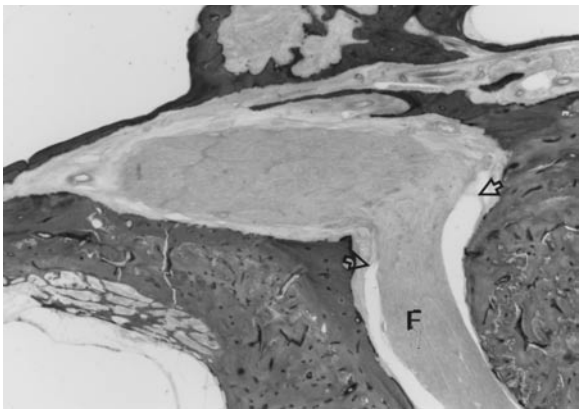

Fig. 9.7 The subarachnoid space in the petrosal facial canal usually is limited at the GG (*arrows*)

tion in the internal auditory canal portion of the FN is juxtaposed to the vestibular ganglion (Scarpa's ganglion). It is well known that part of the nervus intermedius travels within the vestibular nerve trunk before leaving by way of the vestibulofacial anastomosis. In this way, the nervus intermedius is collected before entering the FN trunk. The significance of this inclusion of a part of the nervous intermedius is that some MG cells are incorporated into the vestibular ganglion. Although these two ganglionic masses are derived from two separate embryologic sources, their intimate anatomic association permits a common involvement in inflammatory processes [12].

9.1.2 Sheath of the Facial Nerve

The FN benefits from having a dense, collagenous sheath surrounding it throughout its course through the TB. Since the dural lining of the internal auditory canal blends with the perineurium of the FN at the GG, the subarachnoid space extends to the ganglion in the majority of TB (Fig. 9.7). Thus, in most TB, the dural lining of the labyrinthine (petrosal) fallopian canal permits an extension of the subarachnoid space to the GG [10]. Here the dural sheath becomes closely applied to the perineurium of the main nerve trunk. This dense nerve sheath extends throughout the temporal course and joins the periosteum at the stylomastoid foramen. The extra temporal FN is surrounded by a normal epineurium. However, various factors in development such as increased subarachnoid pressure may displace the lateral extent of the cerebrospinal fluid space into or distal to the GG (Fig. 9.8). Therefore, in a small number of TB the subarachnoid space may extend beyond the GG and into the tympanic segment of the fallopian canal. With continuous pulsatile subarachnoid pressure, such extension may fistulize into the middle ear space (Fig. 9.9). Such a preformed pathway may be responsible for spontaneous CSF otorrhea.

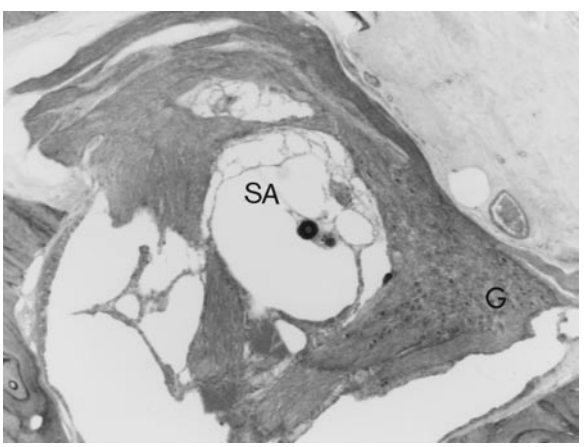

Fig. 9.8 Extension of the subarachnoid space (*SA*) into the GG (*G*) may displace the FN laterally

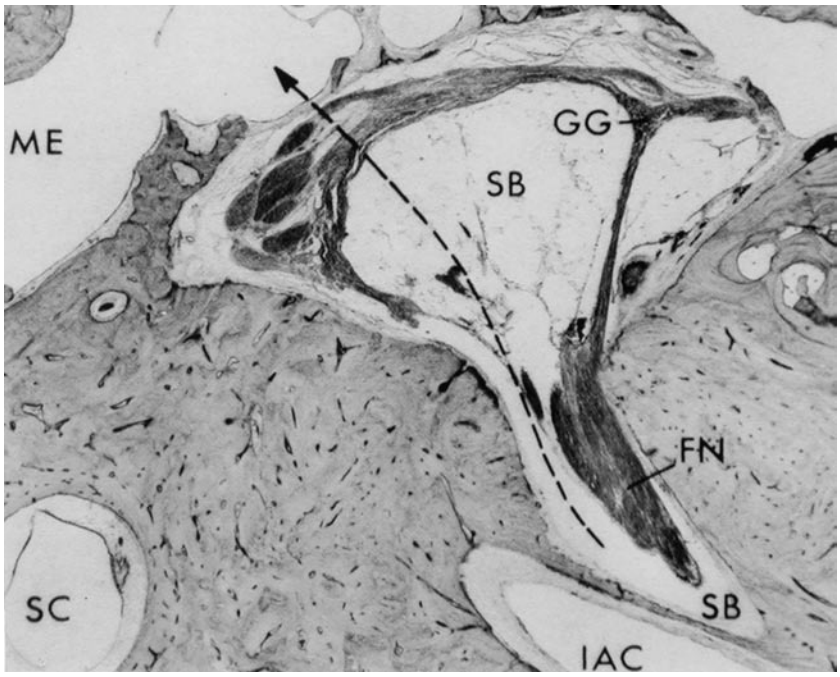

Fig. 9.9 Horizontal temporal bone section demonstrates near fistulization (*dashed line*) of the subarachnoid space (*SB*) into the middle ear (*ME*). Compare with Fig. 8.1 (Chap. 8). *IAC* internal auditory canal, *SC* superior semicircular canal ampulla

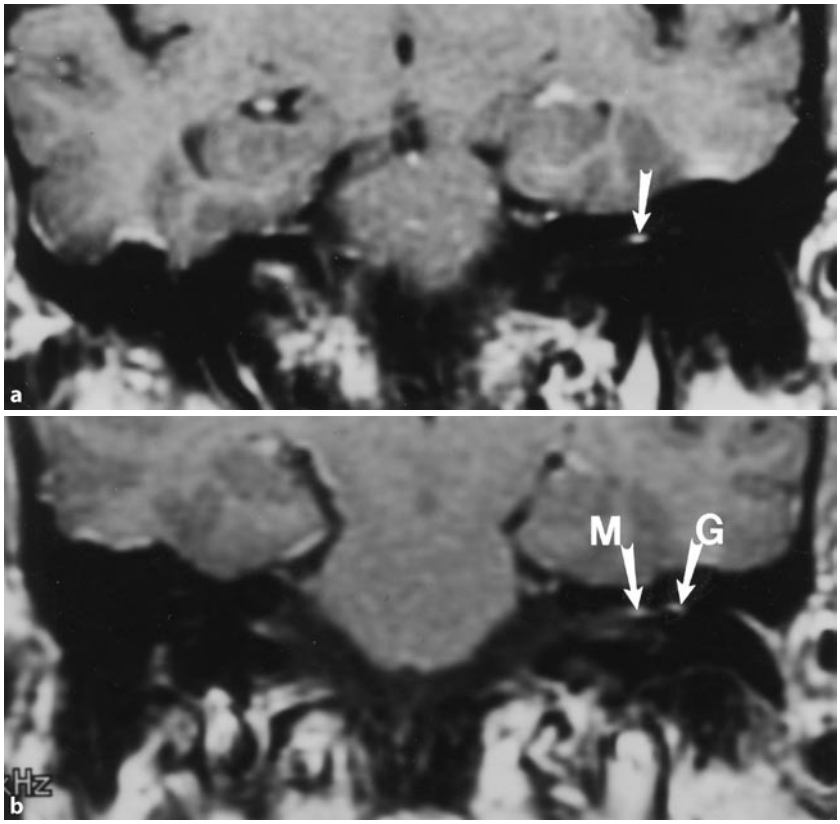

Fig. 9.10 **a** Idiopathic facial paralysis (Bell's palsy) shows enhancement of the meatal ganglion (*arrow*) early. **b** Later in IFP enhancement is seen in the geniculate (*G*) as well as the meatal (*M*) ganglion

9.2 Surgery of the Facial Nerve

9.2.1 Idiopathic Facial Paralysis (Bell's Palsy)

Idiopathic facial paralysis (IFP) is by far the most common cause for peripheral facial paralysis (>70%). Clinical and experimental evidence supports a viral neuritis caused by reactivation of neurotropic viral agents (HSV I and HZV) as the responsible pathology [1, 3, 17, 20–22, 27]. Other viral agents such as mumps, Epstein Barr, and Cytomegalo-inclusion virus as well as Lyme disease have been reported as causative factors. The treatment therefore is primarily—if not entirely—medical, not surgical. Excellent spontaneous resolution can be expected in >85% of patients with IFP [23]. Resolution with minimal-to-moderate residual deficit can account for all but a few percent of the remainder. Oral administration of steroids and antivirals has been shown to significantly improve the quality of return in IFP [2].

Surgery has been advocated in a small number of patients with electrical monitoring signs of impending nerve axonal degeneration [7]. Such surgical intervention is based on FN swelling proximal to the meatal foramen in the internal auditory canal. Swelling in this segment of the FN can be explained by virus reactivation in the meatal ganglion. Numerous reports describing early enhancement with contrast MRI [4, 5, 14, 18, 26], agree that the lateral end (fundus) of the internal auditory canal is characteristic in this viral neuropathy (Fig. 9.10). Histopathology in TBs from patients with a history of IFP revealed degenerated ganglion cells in the meatal ganglion but not the GG (Fig. 9.11) [14]. However, decompression of the meatal foramen may pose a risk of further reactivation of latent virus in the meatal ganglion by virtue of surgical stress, thus aggravating the pathogenesis of IFP. Therefore, the benefits of such surgical intervention must be balanced against the risk of worsening the end result by virus reactivation.

9.2.2 Chronic Otitis Media

Facial neuritis as a complication of chronic middle ear infection with or without cholesteatoma is the next most common cause of facial paralysis. Although

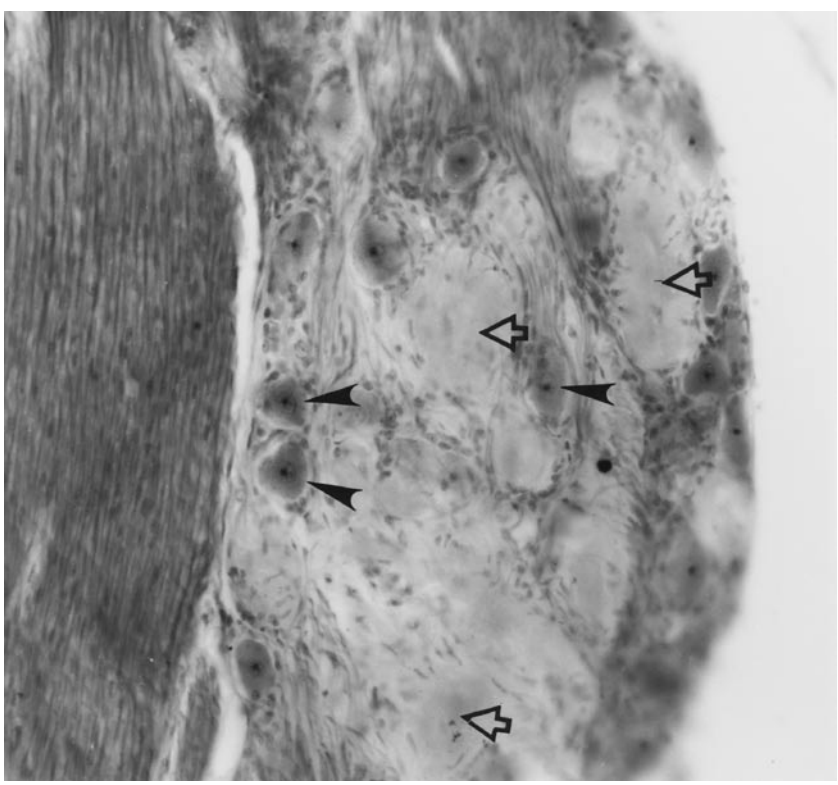

Fig. 9.11 Photomicrograph of the meatal ganglion in a case of IFP shows many degenerated (*arrows*) and a few intact ganglion cells (*arrowheads*)

the inflammatory effect of cholesteatoma membrane is the usual pathology responsible, chronic mucous membrane disease with granuloma formation may also cause FN neuritis. This complication represents an urgent indication for surgical intervention to ensure a satisfactory recovery of FN function. Thorough exenteration of mastoid and middle ear disease with exposure of the FN proximal and distal to the location of nerve disease interface is necessary.

Since chronic inflammatory changes camouflage the bony structures in the middle ear, the fallopian canal may be difficult to identify in surgery for this complication. A reliable landmark to the location of the FN canal in the epitympanum is the shelf of bone that separates the epitympanum from the anterior epitympanic space [8] (Fig. 9.12). This partition, located anterior to the head of malleus, may be entirely bony or have a membranous portion (Fig. 9.13). It is a reliable structure for the FN canal because it develops from the bony partition that envelops the FN, forming the fallopian canal (Fig. 9.14).

Incision of the sheath with release of edematous nerve is controversial because even atraumatic incising of the sheath is accompanied by axonal degeneration. However, when herniation of nerve tissue has already occurred through the sheath, incision of the sheath is recommended. Preservation of motor axons by incision of the lateral aspect of the sheath is favored by the lateral location of the sensory component in the temporal course of the FN.

Facial paralysis in acute middle ear infection requires a staged approach. Generally, intravenous antibiotic administration with sustained drainage of the middle ear (tube) is sufficient to reverse the middle ear infection responsible for FN neuritis. However, poor response of the infection to such treatment supported by radiologic evidence of mastoid bone demineralization mandates surgical exploration with decompression of the fallopian canal (tympanic and mastoid).

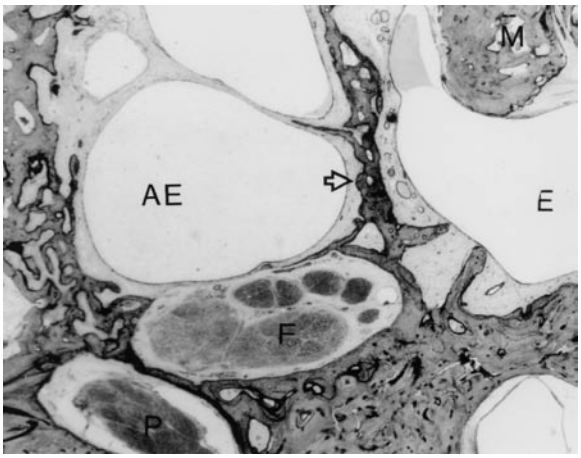

Fig. 9.13 Photomicrograph of the same area as in Fig. 9.12. The bony partition (*arrow*) forms part of the fallopian canal by enveloping the FN (*F*). *AE* anterior epitympanum, *E* epitympanum, *M* head of malleus, *P* petrosal segment of the FN

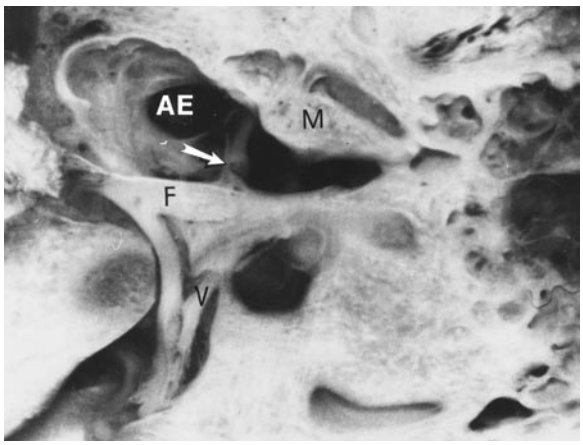

Fig. 9.12 Celloidin embedded TB demonstrates the FN (*F*) in the anterior epitympanum. The partition separating the anterior epitympanic cell (*AE*) from the epitympanum is indicated by arrow. *M* head of malleus and body of incus, *V* vestibular nerve (superior division), *P* petrosal segment of FN

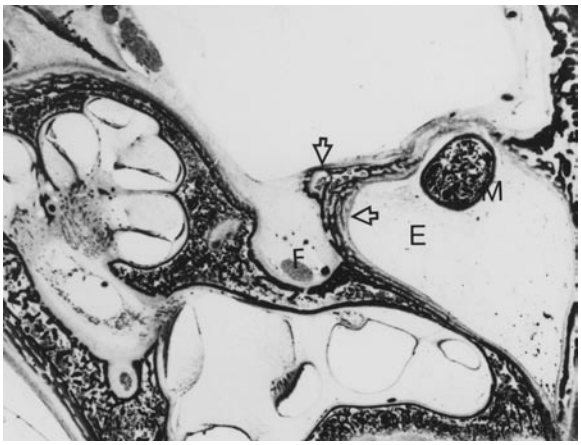

Fig. 9.14 Similar section through a fetal TB shows development of the fallopian canal in the epitympanum (*arrow*). *M* head of malleus, *F* FN

9.2.3 Trauma: Temporal Bone

9.2.3.1 Longitudinal Fracture

TB fractures represent another common cause for facial paralysis. Longitudinal fracture of the TB results from a force to the lateral aspect (squamous) of the TB. In such injury, the fracture line passes through the juncture of petrous bone with the floor of middle cranial fossa, injuring the FN at the GG [28]. The type of injury produced is contusion without transection of nerve. Surgical exploration is not usually indicated as nerve function recovers with observation. Rarely, the fracture may be complex and shear the vertical segment of the nerve near the bony external auditory canal. CT scan of the TB will reveal such involvement and prompt surgical exploration. A conductive hearing loss (hemotympanum) also usually improves with time. A residual conductive loss when the ear has returned to a normal state suggests ossicular dislocation. Cerebrospinal leak may occur with this injury, but usually resolves spontaneously.

9.2.3.2 Transverse Fracture

The fracture line here results from a blow to the occiput and passes through petrous bone where the labyrinth and the internal auditory canal (IAC) represent a weakness (Fig. 9.15). The marked displacement of fracture fragments frequently disrupts the FN, which is securely anchored in its bony canal. Surgical exploration is indicated when the patient's neurological status permits [6]. Because of the loss of labyrinth function, there is opportunity for rerouting of FN segments through the vestibule and repair with primary anastomosis (Fig. 9.16). Total obliteration of the ear to control CSF leak is used for closure of the defect.

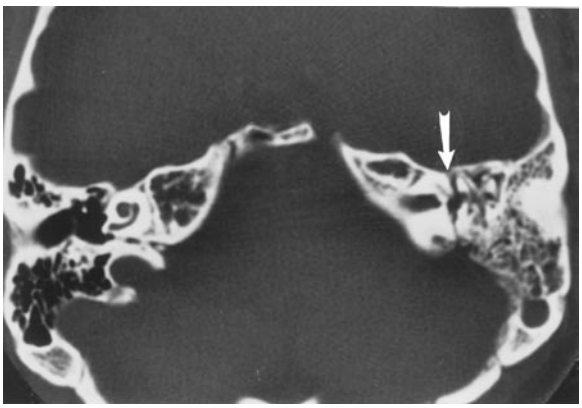

Fig. 9.15 CT scan of a transverse TB fracture (*arrow*) shows wide separation of the bone fragments of the petrous bone

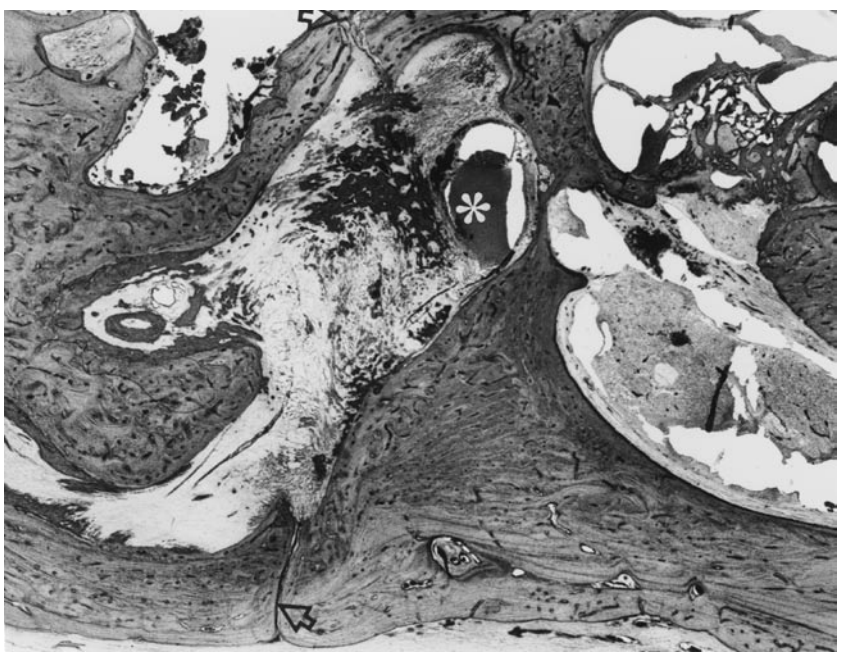

Fig. 9.16 The histological changes produced by a transverse TB fracture (*arrow*) are shown in the photomicrograph. Replacement by fibrous and osseous tissue is seen in the labyrinth (*)

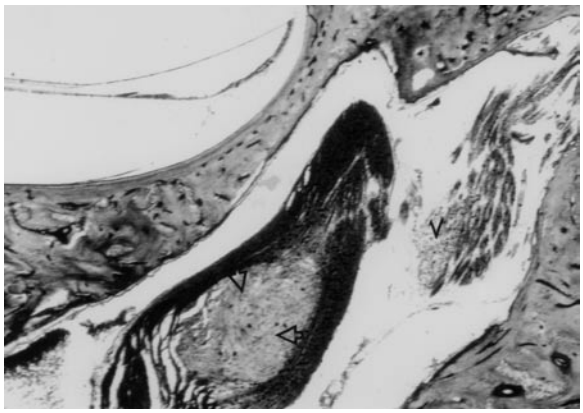

Fig. 9.17 Photomicrograph of a small FN schwannoma (*arrow*) shows displacement of adjacent FN axons. *V* vestibular nerve

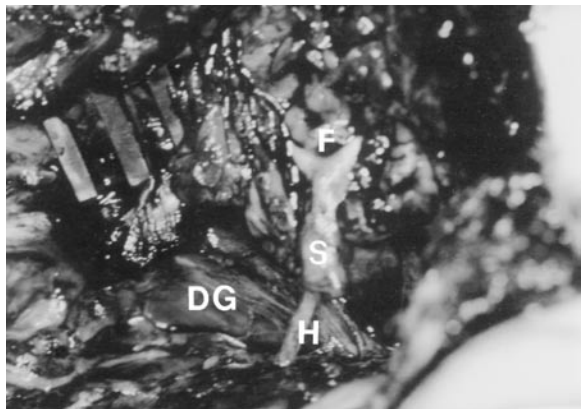

Fig. 9.19 Photo of hypoglossal (*H*)-to–facial (*F*) nerve anastomosis in the neck. A vein sleeve (*S*) covers the anastomosis. *DG* posterior belly of digastric muscle

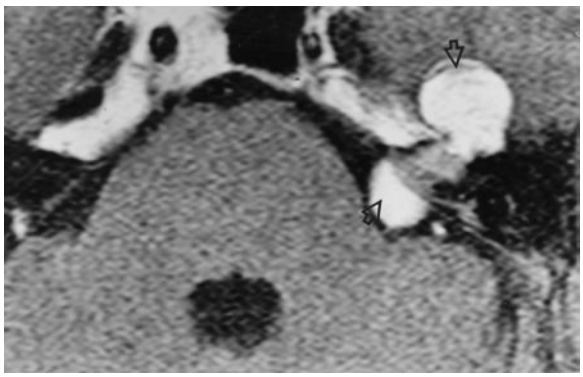

Fig. 9.18 MRI demonstrates the middle and posterior fossa extension of a large FN schwannoma (*arrows*)

9.2.4 Neoplasia

Benign tumors arising in the FN present clinically as progressive facial weakness. The most common tumors are hemangioma and schwannoma. Hemangiomas are usually found at the geniculate bend or in the meatal segment of the FN where the sensory ganglia are located [24]. These tumors are thought to arise from the vascular plexus associated with sensory ganglia. Schwann cell tumors may arise at any level distal to the glial–Schwann cell interface of the nerve near the porus acousticus in the internal auditory canal.

Hemangiomas have an expansile growth from within the ganglionic mass to engulf the nerve. Complete removal requires resection of the segment of nerve containing the neoplasm. Repair with a free nerve graft is necessary. Schwann cell tumors arise from a discrete group of sensory axons and displace intact fascicles of motor nerve fibers as they enlarge (Fig. 9.17). Frequently they may be removed while preserving some intact FN bundles [16]. However, when they reach large dimensions, the nerve segment must be resected and repaired with a free nerve graft (Fig. 9.18). Malignant tumors involve the FN secondarily as spread from a nearby primary. Typical primary tumors are squamous cell carcinoma of the ear canal or middle ear, and parotid gland carcinoma. Resection of the FN with serial frozen sections taken proximally is necessary for tumor control. Facial rehabilitation may be achieved with a cable graft to peripheral main branches or facial–hypoglossal nerve anastomosis depending on the length of remaining nerve segments distally (Fig. 9.19).

9.2.5 Pseudotumor of the Facial Nerve

Pseudotumor refers to an exuberant hyperplasia of the dural sheath surrounding the FN in the TB, simulating a neoplasm of the nerve [9]. Since the usual preceding history includes multiple mastoid surgeries, the stimulus for this hyperplastic response is likely an inflammatory one. Characteristically FN function is normal, and the pseudotumor is encountered at revision surgery for recurrent chronic infection. These signs indicate that the mass represents a pathologic alteration of the sheath rather than the nerve bundles. The mass is resected by excision of the FN sheath with preservation of the FN (Fig. 9.20).

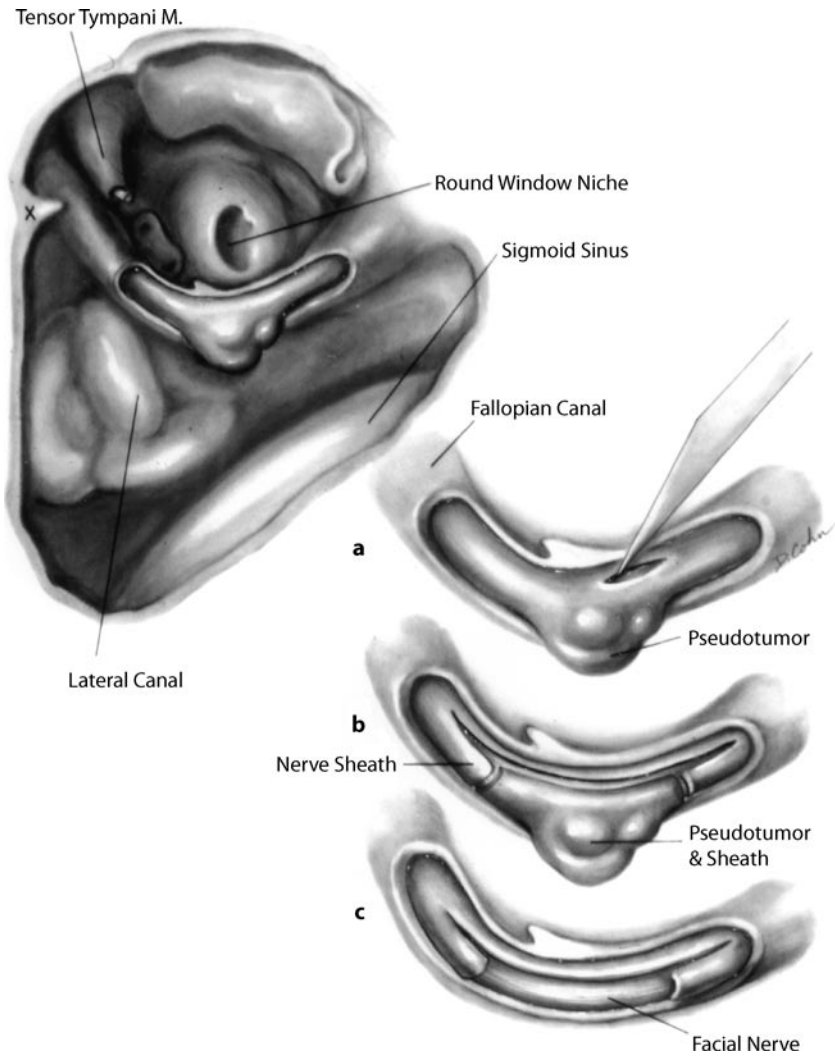

Fig. 9.20 Drawing of the surgical management of FN pseudotumor

COMPLICATIONS TO AVOID

1. Identification of the FN in chronic otitis media starts with exposure of the nerve in a normal segment of the fallopian canal to avoid injury to the nerve.
2. Pseudotumor of the FN sheath requires sharp dissection of the sheath to avoid nerve injury.
3. Cable nerve grafts in FN surgery should be longer than the defect to avoid poor regeneration of nerve fibers.

Pearl

- The meatal ganglion of the FN is equal to or larger than the GG in 15% of TBs.

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