Facial Reanimation

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Facial nerve disorders encompass a broad spectrum of dysfunction, ranging from subtle dynamic facial asymmetry to dense flaccid paralysis. Following repair or grafting, the extent of facial nerve regeneration can greatly vary. Restricted movement may result from hypofunction (persistent weakness of facial muscles), hyperfunction (hypertonicity, spasm), aberrant regeneration (synkinesis), or a combination of these states. Facial nerve dysfunction may lead to numerous functional problems including corneal exposure, dry eye, epiphora, oral incompetence, poor manipulation of the food bolus, speech and articulation difficulties, nasal obstruction, and facial pain. Inability to convey emotion through facial expression and the accompanying psychological penalty as well as social isolation have a profound impact on a patient's quality of life. This chapter reviews anatomy of the facial nerve, grading of facial nerve injury, and contemporary surgical and adjunctive techniques for facial reanimation. An algorithm for zonal management of the paralyzed face is provided.

Anatomy of Facial Nerve

A neuron consists of a cell body with dendritic and axonal extensions enclosed by a plasma membrane. Axons are enveloped and supported by Schwann cells, some forming a myelin sheath serving to increase the velocity of action potential propagation. Individual myelinated axons are enclosed within a connective tissue network (the endoneurium) and arranged in groups of fascicles, each of which is surrounded by a distinct perineurium. The fascicles and surrounding perineurium are enclosed by loose areolar tissue containing multiple vascular channels, known as the internal epineurium. The external epineurium encloses all the fascicles and encases the entire peripheral nerve [1]. The mesoneurium consists of loose areolar tissue that extends from the external epineurium, through which segmental blood supply enters the nerve. Peripheral nerve sheaths are well vascularized and include two intracommunicative systems: (1) the perifascicular system, located in the internal epineurium, and (2) the intrafascicular system, located in the endoneurial network of perineurium-enveloped fascicles [2].

Voluntary facial motor function originates from upper motor neurons in the lower portion of the precentral gyrus. The frontotemporal cerebral cortex as well as limbic and basal ganglion sources house central neurons controlling involuntary expression. The facial motor nucleus is located in the ventral aspect of the caudal pons. The dorsal portion of the facial motor nucleus houses neuronal cell bodies controlling the upper third of the face and receives bilateral upper motor neuron input, while the ventral portion controlling the lower two-thirds of the face receives only crossed input [3]. Facial nerve fibers exit the motor nucleus, pass medioventral around the nucleus of the abducens (sixth) cranial nerve, and exit the lateral brainstem near the cerebellopontine junction (Fig. 16.1) [3]. The somatic motor component of the facial nerve exits the pontomedullary junction 1–2 mm anterior to the vestibulocochlear nerve together with the nervus intermedius, which carries the somatic sensory, special sensory, and visceral motor components of the facial nerve. The nerves are loosely joined, devoid of epineurium, and bathed in cerebrospinal fluid within this cisternal segment that spans the 17–24 mm gap between the brainstem and the porus acusticus at the cerebellopontine angle. The meatal segment spans 8–10 mm between the porus acusticus and the meatal foramen; the facial nerve occupies the anterosuperior quadrant of the internal auditory canal (IAC).

The facial nerve assumes its narrowest dimension within the IAC fundus at the meatal foramen, the entrance to the fallopian canal. Herein, the nerve is most susceptible to injury from trauma or inflammation. The fallopian canal,

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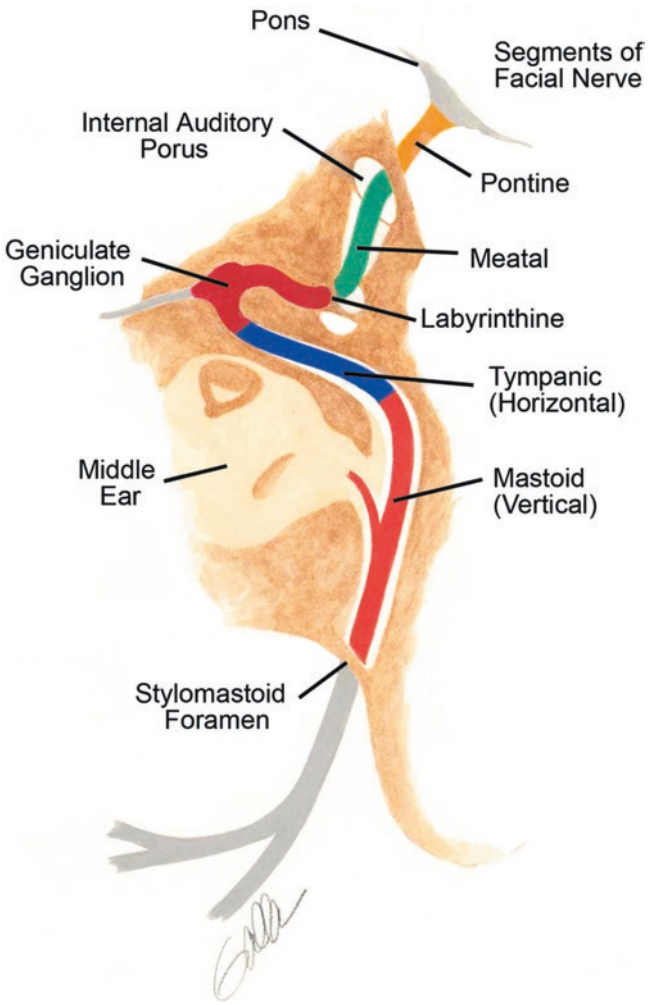

Fig. 16.1 The extramedullary segments of the facial nerve. (Modified with permission from BC Decker Inc., from Nadol JB Jr. [32])

which has the longest bony course of any cranial nerve, is divided into three segments (Fig. 16.1): labyrinthine (3–5 mm), tympanic or horizontal (8–11 mm), and mastoid or vertical (10–14 mm). The labyrinthine segment extends from the fundus of the IAC to the distal portion of the geniculate ganglion. The greater superficial petrosal nerve branches from the geniculate ganglion, carrying preganglionic parasympathetic secretory fibers to the nose, lacrimal gland, and palate. The tympanic segment spans the distal geniculate ganglion where the nerve sharply turns posteriorly (the first genu) to the pyramidal eminence where the nerve sharply turns inferiorly (the second genu). The nerve to stapedius muscle emerges near the second genu. The mastoid segment spans the distance between the second genu and the stylomastoid foramen and has two other branches: the ascending branch of the auricular branch of the vagus nerve (Arnold's nerve) and the chorda tympani. Arnold's nerve provides sensation to the ear canal, tragus, and auricle. The chorda tympani carries preganglionic parasympathetic secretory fibers of the submandibular and sublingual glands as well as sensory taste fibers from the anterior two-thirds of the tongue. The arachnoid pia-dura mater junction typically lies near the fundus of the IAC but may extend as far as the geniculate ganglion. Proximal to this juncture, the facial nerve is unsheathed and bathed in cerebrospinal fluid.

After exiting the stylomastoid foramen, the facial nerve gives off a posterior auricular branch, a motor branch to the posterior belly of the digastric muscle, and a motor branch to the stylohyoid muscle prior to the pes anserinus. The posterior auricular nerve carries motor fibers to the superior and posterior auricular muscles and occipitalis muscles and is thought to supply the skin covering the mastoid process and adjacent parts of the auricle. Though branching patterns are highly variable, the main trunk of the facial nerve often splits into upper and lower divisions that further branch to supply five zones: temporal, zygomatic, buccal, marginal mandibular, and cervical. Though these branches generally innervate separate facial regions, there is significant arborization and redundancy among neighboring territories, especially in the midface.

Nerve Injury Classification

The degree to which a nerve is injured may be classified according to the Sunderland system, which includes five levels of severity (Fig. 16.2) [4]. The Sunderland scale provides a conceptual framework that guides management of nerve injury.

First-Degree Injury

In first-degree injury or neuropraxia, nerve conduction is interrupted at the injury site. Axon continuity is preserved, and there is no Wallerian degeneration. Neuropraxia often results from compression and ischemic insults. The period of denervation is short, and function recovers rapidly and completely.

Second-Degree Injury

In a second-degree injury, axons are disrupted within their endoneurial tubules. The endoneurium, which consists of the basal lamina of the Schwann cells, and a fibrillar reticular lamina with surrounding collagen fibrils, is preserved. Wallerian degeneration occurs distal to the site of injury. The confines of the endoneurial tube accurately guide regenerating axons to their original targets. Functional recovery is complete or near complete.

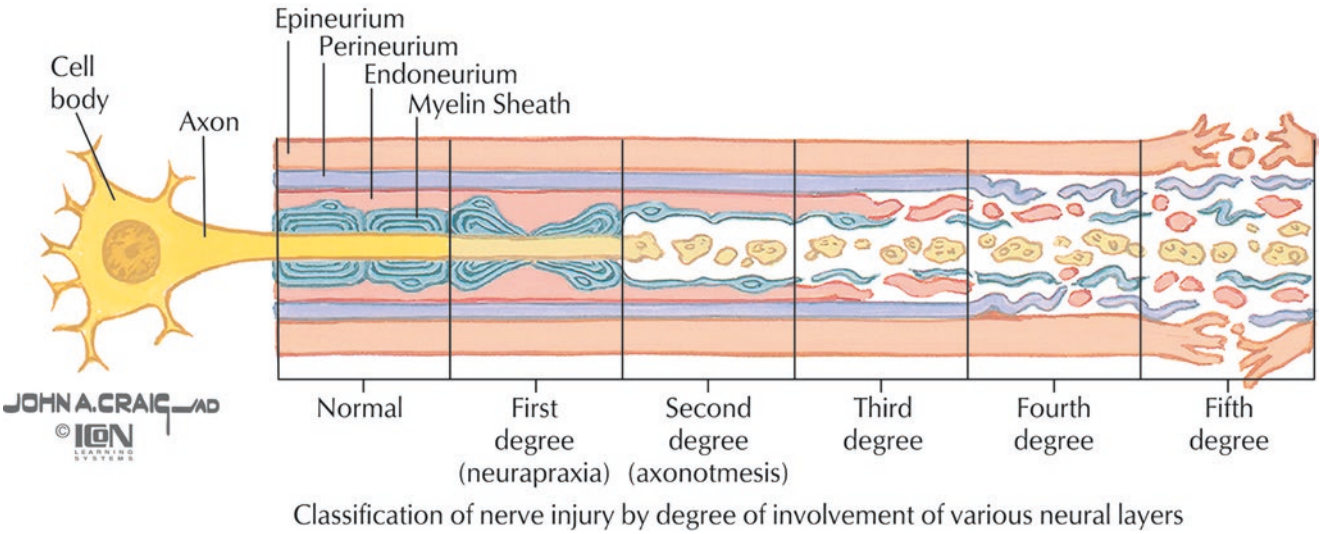

Fig. 16.2 Sunderland classification of degrees of nerve injury based on involvement of the neural layers. (Netter illustration used with permission of Elsevier Inc. All rights reserved)

Third-Degree Injury

At this level of severity, endoneurial disruption occurs without perineurial disruption. Continuity of the endoneurial tube is lost. However, fasciculi remain continuous and their arrangement in the nerve trunk is preserved. This level of injury may result from traction, hemorrhage, edema, vascular stasis, and ischemia. Resulting intrafascicular fibrosis severely impedes the process of regeneration, and extent of functional recovery varies. Endoneurial disruption may result in some aberrant axonal regeneration of axons or ephaptic communication with subsequent development of involuntary and uncoordinated facial movements, known as synkinesis.

Fourth-Degree Injury

This level of injury results in disruption of the endoneurium and perineurium. Only the external epineurium and mesoneurium remain intact. Intraneural fibrosis limits recovery, and pronounced aberrant interfascicular regeneration occurs causing disfiguring facial synkinesis.

Fifth-Degree Injury

At this highest level of injury, also known as neurotmesis, anatomic discontinuity is complete. Minimal to absent recovery occurs without surgical repair. Poor somatotopic fascicular organization of motor axons within the facial nerve results in significant aberrant regeneration and subsequent facial synkinesis despite meticulous repair of main trunk injuries [4–6].

Nerve Repair and Nerve Grafting

When facial nerve discontinuity is encountered, first-line therapy dictates reestablishment of neural continuity between proximal and distal facial nerve stumps. Direct end-to-end repair is preferred where nerve ends may be reapposed without tension; other interposition graft repair is favored [7]. In the setting of facial nerve defects of the fallopian canal, intratemporal rerouting of the nerve by means of mastoidectomy may be employed to facilitate repair and avoid need for interposition grafting [8]. The distal vertical segment of the nerve is amenable to suturing. A defect of the horizontal segment is typically addressed with simple approximation of nerve ends with reinforcement using fibrin glue, collagen, or hemostatic agents. During repair, the operating microscope should be used to evaluate the cut nerve endings. The stumps should be debrided and all devitalized tissue removed, followed by approximation and coaptation of the epineurial sheath. Interfascicular repair has not shown benefit over epineurial repair.

Nerve Grafting

When a tension-free neurorrhaphy cannot be achieved, an autograft is interposed between the proximal and distal endings of the facial nerve. Most often, donor nerve grafts are harvested from the great auricular, sural, or medial antebrachial cutaneous nerves (Fig. 16.3). The great auricular nerve is ideal for repairs that require grafts less than 6 cm long (Fig. 16.3a). A contraindication to its use is the presence of a neurotrophic malignancy. In such cases, the sural (Fig. 16.3b) or medial antebrachial nerve (Fig. 16.3c) is pre-

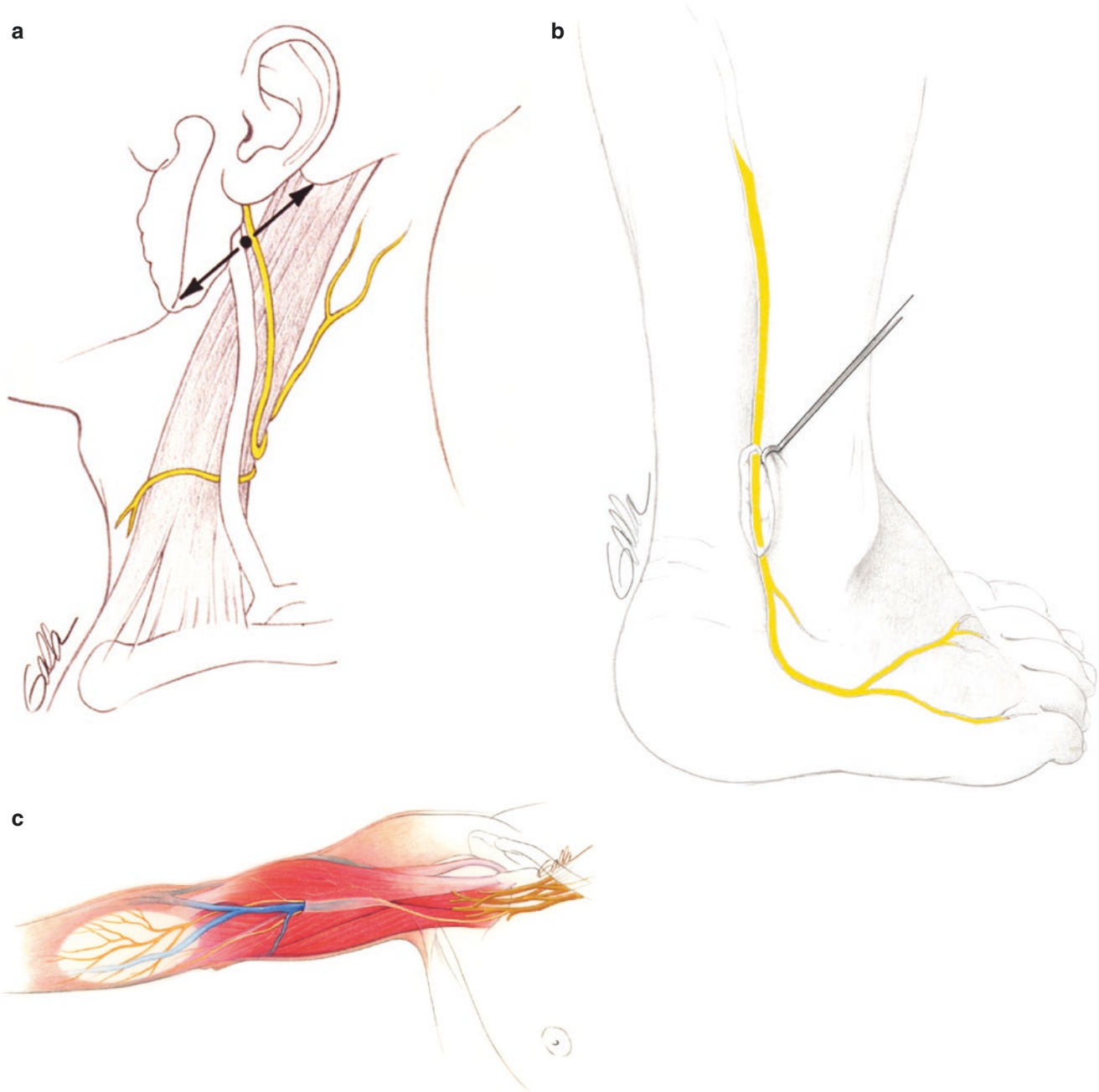

Fig. 16.3 (**a**) The great auricular nerve (yellow) is located on the lateral surface of the sternocleidomastoid muscle at the midpoint (dot) of a line (arrows) drawn between the mastoid tip and the angle of the mandible. (**b**) The sural nerve (yellow) is located in a subcutaneous plane posterior to the lateral fibular malleolus. The nerve runs parallel to the small saphenous vein. (**c**) The medial antebrachial cutaneous nerve arises primarily from the medial cord of the brachial plexus with contributions from the ventral rami of C8 and the first thoracic nerve. As the nerve enters the arm, it lies superficial to the brachial artery near the basilic vein. At the elbow, it divides into posterior and anterior branches that supply sensation to the ulnar aspect of both the flexor and extensor surfaces of the forearm. (Figure **a**, **b** from Nadol JB Jr. [32]. Figure **c** reproduced with permission from Cheney [22] © 2007 Mack L. Cheney, MD)

ferred. The latter is ideally suited for total nerve reconstruction to multiple distal branches. The results of cable grafting can be favorable [9]. The quality and quantity of axonal regrowth are best when repair is executed immediately following nerve injury or intentional resection. Under ideal circumstances, neural reconstruction proceeds in the same operative setting as sacrifice. Factors that lead to poor recovery include wound disruption, infection, or tension on the site of coaptation. In most cases, movement returns within 6–12 months following the reestablishment of neural continuity, depending on the location of the neural repair. Improvement continues to occur over several years.

Facial Reanimation

When neural continuity cannot be reestablished, either due to lack of a proximal stump at the brainstem or due to severe traumatic distortion of temporal bone anatomy, other methods of reestablishing facial balance and movement may be considered. Facial reanimation procedures refer to interventions that restore facial symmetry, resting tone, voluntary and involuntary movement, or a combination thereof. There are several broad categories of facial reanimation techniques, each appropriate to a specific set of clinical, anatomic, or outcome-related circumstances. These techniques include reinnervation techniques, muscle transfers, static procedures, and nonsurgical adjunctive therapy.

Nerve Substitution Techniques

Nerve substitution techniques, also known as nerve transfers, or reinnervation techniques, refer to procedures that provide neural input to the facial musculature via distal facial nerve branches using motor nerves other than the native facial nerve. Nerve transfers may be performed to the main trunk of the facial nerve, with the goal of restoring facial tone and some form of blink, or to more distal facial nerve branches, with the goal of restoring volitional control of specific facial movements such as smile. These techniques are indicated when the proximal facial nerve stump is unavailable but the distal facial nerve and facial musculature are present and functional. For example, resection of a skull base tumor requiring resection of the facial nerve at or near the brainstem may render neurorrhaphy technically infeasible. In this setting, immediate nerve transfer is indicated. Nerve transfers may be appropriate when no discernible facial function is noted 6–12 months following skull base surgery, intracranial injury, and traumatic facial paralysis, where the facial nerve is believed to be anatomically intact. While electrophysiologic demonstration of the lack of reinnervation potentials and the presence of fibrillation potentials at the 12-month mark may confirm persistent and complete denervation, lack of obvious functional recovery by this time period is a sufficient indication for intervention. These findings suggest insufficient regenerative potential from the proximal facial nerve stump and mandate alternative proximal axonal input to the distal facial nerve and facial musculature before atrophy and fibrosis become irreversible. Ongoing debate surrounds optimal timing for reinnervation in flaccid facial paralysis after skull-base surgery, though the most recent data suggest that complete flaccidity after 6 months portends a very poor smile prognosis. Nerve transfers are only indicated where facial muscles are likely to be receptive to reinnervation: typically 2 years from denervation in adults and possibly longer in children.

Hypoglossal–Facial Transfer (Cranial Nerve XII–VII Crossover)

The hypoglossal nerve was historically most often used to reinnervate the distal facial nerve. Its proximity to the extratemporal facial nerve, its dense population of myelinated motor axons, and the relative acceptability of the resultant hemi-tongue weakness made it a logical choice in the past [10–13]. In the classic cranial nerve XII–VII transfer, the entire hypoglossal nerve is transected and reflected upward for direct neurorrhaphy to the facial nerve stump (Fig. 16.4a). Several modifications have been described (Fig. 16.4b, c). In the "split" XII–VII transfer [14], the hypoglossal nerve is incised to a depth of approximately 30% of its caliber, and dissection is performed to elevate a segment that is several centimeters long for coaptation to the facial nerve or its branches (see Fig. 16.4b). This technique is inadvisable as the hypoglossal nerve is monofascicular in its proximal segment, without clear perineurial boundaries between groups of axons to guide dissection.

Another modification, the XII–VII jump graft, is designed to reduce tongue morbidity by avoiding the splicing away of a significant length of the hypoglossal trunk. An end-to-side neurorrhaphy between the hypoglossal nerve and a donor cable graft (usually the great auricular nerve) is sewn to the distal facial trunk (Fig. 16.4c) [15]. This modification is based on improved understanding of the microanatomy of the hypoglossal nerve, which has an interwoven fascicular architecture. Consequently, separating a 30% segment from the main trunk for several centimeters divides a significantly greater number of axons than if the fibers were oriented in parallel [15].

When the facial nerve can be mobilized from the second genu within the temporal bone and reflected inferiorly, removal of the mastoid tip allows direct coaptation of the facial nerve to the hypoglossal nerve, without the need for an interposition graft (Fig. 16.4d) [16]. Elimination of the cable graft provides a regenerative advantage by reducing the neurorrhaphies from two to one. Ongoing additional modifications to further simplify the delivery of axons from the hypoglossal to the distal facial nerve are continuously being devised, though lack of centralized or codified determination of outcomes hinders clear emergence of the superiority of one technique over others.

Surgical Technique

The classic XII–VII transfer is performed via a modified Blair parotidectomy incision. The parotid tail is elevated off the sternocleidomastoid muscle with preservation of the great auricular nerve, and the posterior belly of digastric is identified. Meticulous dissection in the plane between the perichondrium of the ear canal and parotidomasseteric fascia of the posterolateral aspect of the parotid gland is performed to identify the main trunk of the facial nerve, which is then

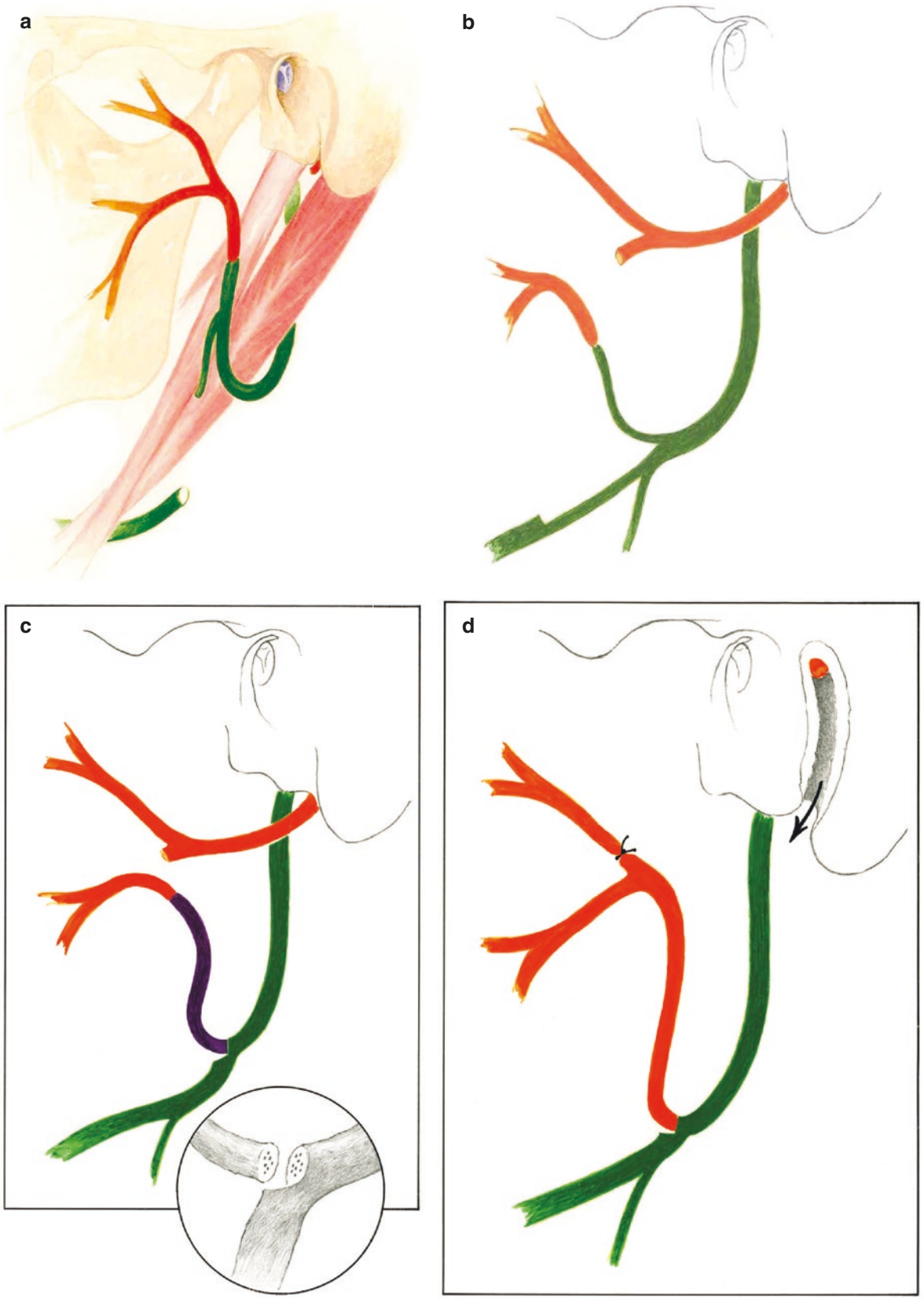

Fig. 16.4 Hypoglossal-facial nerve transfer. Hypoglossal nerve is shown in green; the facial nerve is shown in orange. (**a**) Classic procedure, with the entire hypoglossal nerve transected. (**b**) Modification with 40% segment of nerve secured to lower division. (**c**, left) Jump graft (purple) modification. (Inset) The graft is positioned to capture axons extending from the proximal aspect of the opened hypoglossal nerve. (**d**, right) Reflection of the facial nerve out of the mastoid bone (arrow) to meet the hypoglossal nerve in the neck. (Figure **a**, **b**, and **d** from Nadol JB Jr. [32] with permission from BC Decker Inc. Figure **c** published with permission © Tessa A. Hadlock, MD and Mack L. Cheney, MD)

traced to the pes anserinus. The ascending portion of the hypoglossal nerve is located deep to the posterior belly of the digastric muscle along the medial surface of the internal jugular vein and anterior surface of the internal and external carotid arteries. The nerve is mobilized distally to the branch point of the descendens hypoglossi, with ligation of occipital artery branches. The hypoglossal nerve is transected sharply and reflected superiorly to meet the facial nerve near the stylomastoid foramen. The main trunk of the facial nerve is sharply transected and secured to the hypoglossal nerve with five to seven 10-0 nylon epineurial microsutures under high stereoscopic magnification.

A modification designed to decrease mass movement of the face with XII–VII transfer involves coaptation of the hypoglossal nerve to selective nerve branches, or transection of selected facial nerve branches distal to the point of coaptation. For example, the hypoglossal nerve may be coapted to the inferior division of the facial nerve; alternatively, it may be coapted to the main trunk with transection of the upper division distal to the pes. Alternative reanimation techniques are then used to address the upper face.

The jump graft procedure is also termed the XII–VII side-to-end procedure. The great auricular nerve graft is harvested. The hypoglossal nerve is preserved in continuity once mobilized and an epineurial window made with transection of 30% of hypoglossal axons. Side-to-end coaptation of the graft is made in epineurial fashion, with end-to-end coaptation of the distal graft end to the facial nerve. Alternatively, the proximal facial nerve may be mobilized from the temporal bone, sectioned at the second genu, and transposed down into the neck by removal of the mastoid tip for direct side-to-end coaptation to the hypoglossal nerve.

With a XII–VII transfer, good resting facial tone is achieved in more than 90% of patients. When successful, the transfer allows deliberate facial movement with intentional manipulation of the tongue. However, results are variable. Time from denervation to transfer plays a key role in outcome. Reinnervation must occur within 2 years of injury; otherwise, neuromuscular fibrosis and atrophy progress to a point where meaningful tone and movement cannot be achieved [15].

Two significant drawbacks are associated with the procedure. Many patients experience mass facial movement, and the variable tongue dysfunction has been categorized as “severe” in as many as 25% of patients. Articulation and mastication difficulties are common. The modifications mentioned above are aimed at one or the other of these two problems. The procedure is contraindicated in patients who are likely to develop other cranial neuropathies (i.e., patients with neurofibromatosis type 2) or who have ipsilateral deficits of cranial nerve X. Combined cranial nerve X–XII deficits can lead to profound swallowing dysfunction.

Trigeminal-to-Facial Nerve Transfer

Over the past 5 years, the popularity of the masseteric branch of the trigeminal nerve as a donor source of axons for reinnervation of specific midfacial musculature driving smile has surged [17]. First popularized as a neural source for driving free muscle transfer for facial reanimation, it rapidly gained favor for its ease of access, abundance of motor axons, low donor-site morbidity, and ideal location for direct coaptation to relevant branches of the facial nerve driving smile. Rehabilitation of bite-driven smile is straightforward for most patients. Advances in surgical techniques now allow specific targeting of nerve-to-masseter transfer to distal zygomatic branches of the facial nerve driving smile, with preservation of facial nerve continuity elsewhere. This paradigm shift has led to earlier intervention to optimize outcomes in the setting of persistent flaccid paralysis at 6 months following skull base surgery where facial nerve continuity was preserved, while still allowing for the possibility of recovery of native function by avoidance of main trunk transection. Contrary to the hypoglossal nerve, the nerve-to-masseter does not provide resting tone. Though its use permits reanimation of a powerful smile, the midface remains flaccid at rest. In adults, the technique is best combined with concurrent nerve transfer of the hypoglossal nerve more proximally to the main trunk of the facial nerve to provide facial tone (which can be performed at a later date if native recovery is still possible) or alternatively with cross-face nerve grafting or static suspension of the midface. Results can be expected within 3–5 months and generally are superior to those achieved with muscle transfer (Fig. 16.5).

Surgical Technique

The procedure begins with elevation of a facial flap under the superficial musculo-aponeurotic system (SMAS), directly on the parotido-masseteric fascia. Branches of the facial nerve are identified as they emerge from the parotid gland, and a single large-caliber branch inferior to the zygomatic arch coursing toward the zygomaticus major and minor muscles is isolated. Retrograde dissection toward the upper division of the facial nerve through the parotid gland is carried out until the branching pattern of the upper division is fully appreciated and all other branches are left intact. Dissection proceeds deep to the parotid gland through the masseter muscle, where the nerve is located entering its deep surface. Once identified, the nerve is isolated using a vessel loop and dissected anteriorly as it courses more superficially into the muscle belly. When adequate length for primary coaptation to the recipient facial nerve is exposed, the nerve is transected distally and reflected out of the wound bed, and microsurgical coaptation to the recipient branch of the facial nerve is executed. When possible, proximal superomedial branches of the nerve-to-masseter are preserved during har-

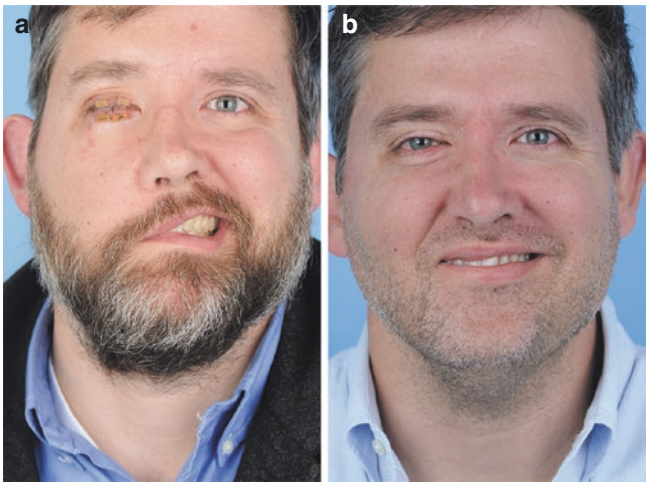

Fig. 16.5 (**a**) Preoperative and (**b**) postoperative view of patient attempting to smile, after complete facial nerve sacrifice at brainstem, with no grafting, followed 12 weeks later by five to seven transfer and two cross-face nerve grafts, one to the eye and one to a midface smile branch. Patient also underwent delayed static suspension

vest to lessen the risk of masseter muscle atrophy and subsequent hollowing of the cheek.

Cranial Nerve VII–VII Cross-Facial Grafting

Another potential source of axons for facial reinnervation is the healthy contralateral facial nerve [18]. It is the only donor source with the potential to reanimate mimetic function, such as spontaneous blink and emotive smile. Because it manifests significant distal arborization, several branches may be sacrificed for use in cross-facial grafting without adversely affecting healthy-side function. Donor branches contain far fewer motor axons than the hypoglossal or masseteric nerves to power the paralyzed side [19]. The use of contralateral facial nerve branches strictly for reinnervation of native facial musculature has largely been replaced by cross-face nerve grafting in conjunction with other reinnervation techniques or in preparation for free-muscle transfer; direct coaptation to branches of the paralyzed side is now thought to be useful only when performed within 3 months of proximal facial nerve sacrifice because of the time required for axons to traverse the face and the sensitivity of paralyzed muscles to denervation time.

Surgical Technique

A preauricular incision is made on the nonparalyzed side. A flap is raised on the parotid-masseteric fascia until the anterior border of the parotid gland is identified. With the guidance of electrical stimulation, the masseter fascia is dissected to identify 5–10 branches of the facial nerve. The 1–3 branches yielding isolated smile or blink movement are

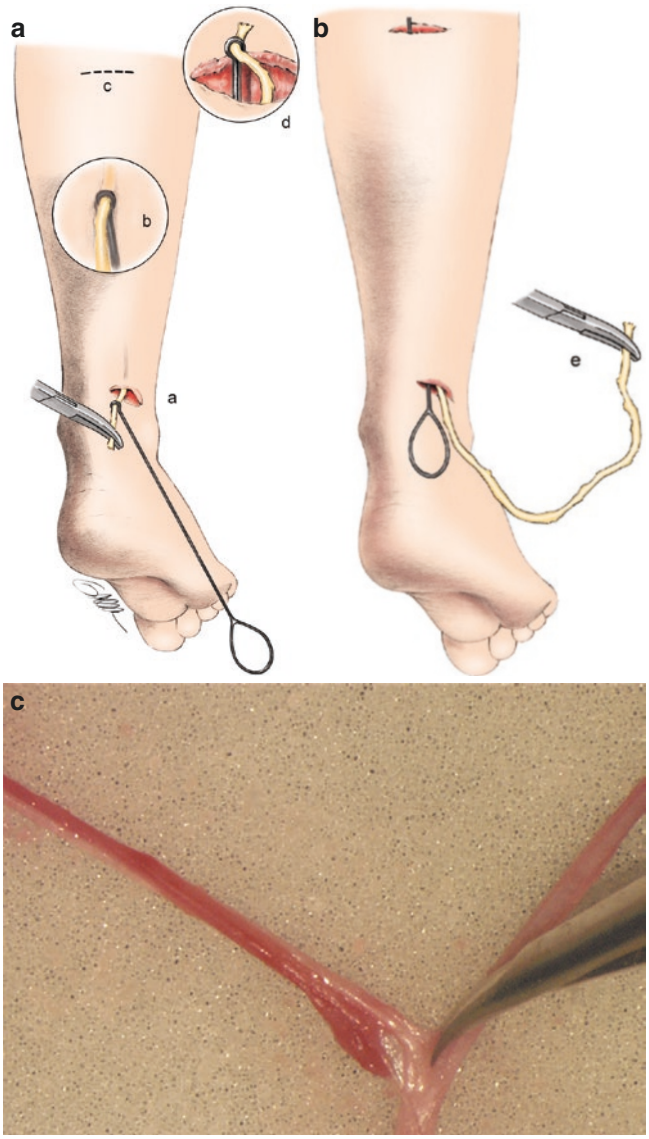

Fig. 16.6 Steps involved in minimally invasive sural nerve harvest. (**A**, a) The sural nerve is identified 1 cm lateral to the lateral malleolus, divided distally and inserted through the islet of the nerve stripper. (b) The nerve stripper is passed superoposteriorly, freeing the nerve from the surrounding subcutaneous fat. (c) When an appropriate length of nerve has been freed from surrounding tissues, a second skin incision is made by palpating the eyelet through the skin. (d) The eyelet of the stripper is delivered through the incision, and the proximal end of the sural nerve is cut, preparing it for removal through the distal incision. (**B**, e) Removal of a 15–20 cm sural nerve graft through the distal incision. Note the avoidance of a long skin incision. (**C**) Intrafascicular dissection of a single sural nerve into two strands, using a microscissors and microscopic magnification. (Published with permission © Tessa A. Hadlock, MD and Mack L. Cheney, MD)

selected as donor branches and transected sharply. A sural nerve graft is harvested from the leg through an open, endoscopic or minimally invasive approach (Fig. 16.6) [17]. The nerve is used either singly or divided into two strands under microscopic guidance if both blink and smile are targeted.

The strands are then tunneled subcutaneously from the donor branches either to the gingivo-buccal sulcus on the paralyzed side (if free tissue transfer is planned) or across into the paralyzed face for direct coaptation, in which case an identical facial nerve dissection is executed on the paralyzed side (direct hookup). In direct hook-up cases, end-to-end neurorrhaphy is performed to recipient smile and/or blink branches. On the donor side, nerve coaptation between the sural nerve and the donor facial nerve branches is performed with 10-0 nylon sutures. Clinically, the growth of axons into the graft is followed by tapping on the graft (Tinel's sign); tingling indicates the presence of regenerating axons. When required, a second-stage free-muscle transfer is performed 6–9 months later (see section "Free-Muscle Transfer" later in this chapter).

Regional Muscle Transfer Techniques

When the distal facial nerve or facial musculature has atrophied or become significantly fibrotic, the delivery of viable motor axons will not yield adequate excursion to create meaningful facial expression. In such cases, transfer of functional innervated musculature into the face offers the only reliable possibility of providing meaningful facial movement. A segment of innervated muscle can be transposed into the appropriate segment of the face from the temporalis, masseter, digastric, or other regional muscles. Alternatively, a muscle segment can be transferred as a free flap from a distant site (gracilis, pectoralis minor, serratus anterior, latissimus dorsi) and reinnervated locally.

Effective rehabilitation requires training and physical therapy to achieve optimal function. The literature supports the concept of neural plasticity; after a certain training period, some patients with trigeminally driven muscle transfers are believed to achieve movement without consciously clenching their teeth [20].

Temporalis Muscle Transfer

The temporalis muscle has historically been employed for reanimation of the smile in patients whose face is chronically paralyzed. The procedure is also useful as an interim therapy when the regenerative potential of the facial nerve is in question (i.e., after skull-base surgery) and during the waiting period for regeneration because it does not interfere with potential facial nerve regeneration [21].

Before proceeding, it is imperative to establish that the muscle and its nerve and vascular supply are intact. Some neurotologic procedures affect these structures, and several congenital facial palsy syndromes are associated with other cranial nerve abnormalities that may affect function of the temporalis muscle. Severe atrophy of the musculature, such as in an edentulous patient, is also a contraindication to temporalis transfer.

Surgical Technique

In the classic description of the procedure (Fig. 16.7), dissection is performed through an incision from the superior temporal line to the attachment of the lobule [22]. Scalp flaps are raised both anteriorly and posteriorly in the subdermal plane, just under the hair follicles. Care is taken to preserve the superficial temporal artery and veins so that the temporoparietal fascial flap (TPFF) can be used to obliterate the donor-site defect. The TPFF is incised posterior to the course of the temporal branch of the facial nerve and reflected from the true temporalis muscular fascia, leaving the TPFF pedicled on its vessels. A flap is raised deep to the subdermal plane from the zygomatic arch to the oral commissure. Some fat is left on the skin flap to avoid direct apposition of the transferred muscle to the overlying skin, which can produce tethering. The skin flap extends medially to the modiolus necessary for adequate coaptation to the transferred muscle.

A 1.5-cm–wide strip of temporalis muscle with its underlying pericranium is elevated from the calvarium. The segment is chosen so that reflection over the zygomatic arch pulls the commissure in a vector appropriate to the patient's smile pattern. The muscle is reflected into the midface and secured with polyglactin sutures to the orbicularis oris. Secure muscle-to-muscle contact is necessary to promote potential neurotization of the orbicularis fibers (Fig. 16.7a). The commissure is deliberately overcorrected so that with relaxation, an appropriate position is achieved. The TPFF is placed into the donor defect, and the incision is closed over a drain (Fig. 16.7b) [21].

Over the past decade, antidromic temporalis transposition has been largely supplanted by orthodromic approaches, where the tendinous attachment of the temporalis muscle is transferred from the coronoid process to the modiolus and nasolabial fold area. This approach avoids the characteristic bulge of the muscle over the zygomatic arch, which is accentuated by the hollowing in the temporal fossa, created by absence of muscle in the original position.

Irrespective of maneuver, the temporalis muscle has been shown to lead to significantly less oral commissure excursion than free muscle, thus in centers where microvascular surgery is widely performed, regional muscle transfer is reserved for patients who are extremely high surgical risks or who have a poor survival prognosis.

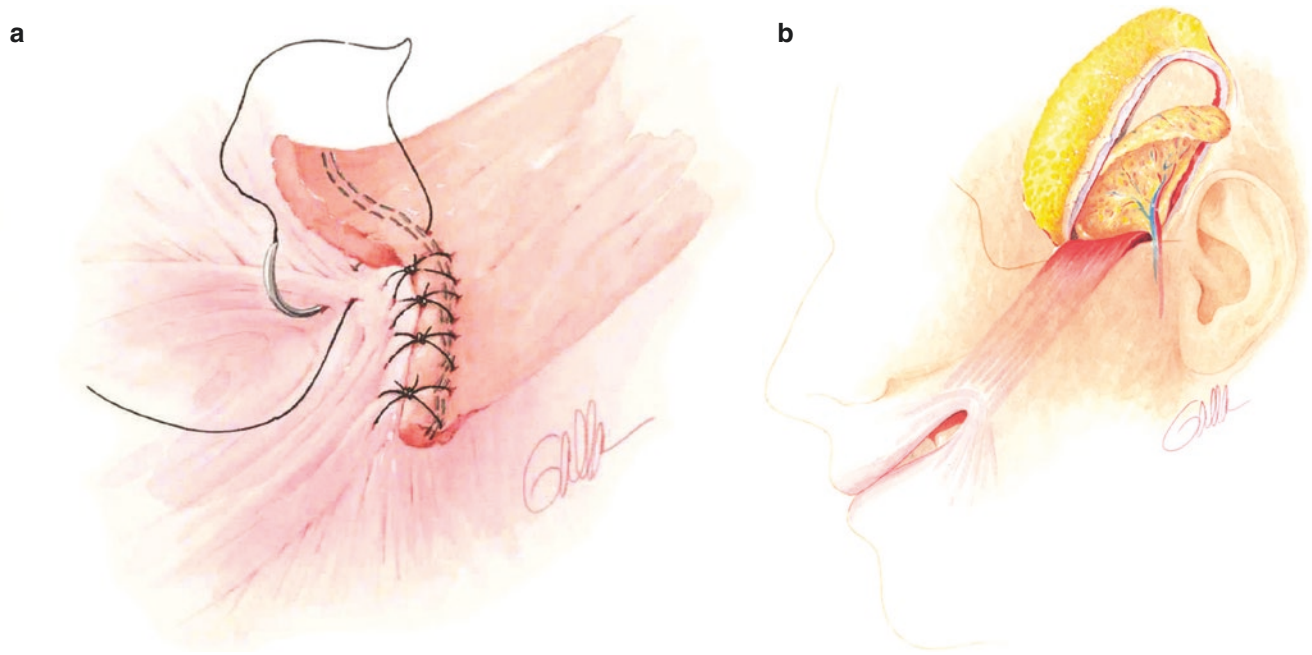

Fig. 16.7 Temporalis muscle transposition. (**a**) Securing the transposed muscle to the orbicularis oris. Note the double staple line and mattress sutures from the temporalis muscle to the modiolus. (**b**) Note the overcorrection of the commissure so that the first molar is visible. (From Nadol JB Jr. [32] with permission from BC Decker Inc.)

Other Regional Muscle Transfers

The masseter muscle transfer, popularized by Rubin and Baker and Conley, can also provide excursion at the oral commissure [23, 24]. The muscle is freed from its mandibular attachments and secured to the lateral aspect of the orbicularis oris, in the same fashion as the temporalis muscle. However, its more lateral vector pull, significant resulting contour defect, and availability of superior options have rendered the procedure obsolete.

The digastric muscle transfer has been described as useful in isolated marginal mandibular nerve injuries for lower lip reanimation. However, it may compromise oral competence in patients with total facial paralysis. In appropriately selected patients with an isolated injury of the marginal mandibular nerve, the procedure may effectively restore depressor function to the lower lip. The procedure involves sectioning the digastric muscle at the junction of the posterior belly and its tendon. The anterior belly and tendon are then freed from surrounding structures and secured to the orbicularis oris along the ipsilateral inferior border (Fig. 16.4). Mylohyoid nerve innervation to the anterior belly can be maintained [25], or the muscle can be driven by a cross-face nerve graft [26].

Other local muscle flaps have been suggested (for example, the innervated platysma musculocutaneous flap [27]) but insufficient numbers of treated patients leave their ultimate utility in question.

Free-Muscle Transfer

Free-muscle transfer may be employed for facial reanimation in several different clinical scenarios. If the proximal facial nerve stump is available but the facial musculature has been resected, a free-muscle flap can be placed in the face and driven by the ipsilateral proximal facial nerve. Patients with congenital facial palsy and those with longstanding facial paralysis or resected or myopathic facial muscle may likewise be good candidates for a free-muscle transfer. The procedure is performed in one or two stages. In the single-stage procedure, the muscle is driven by a branch of the ipsilateral trigeminal nerve (masseteric or deep temporal), while in the two-stage procedure, the muscle is powered by a cross-facial graft (with the first-stage operation performed as described above in the reinnervation section) or is dually innervated by both a cross-facial graft and trigeminal input. The waiting period for axonal extension through the cable graft before muscle transplantation is 6–9 months.

The gracilis muscle was the first muscle used in successful facial reanimation and remains the most popular choice for this purpose [28]. Although modifications involving subsegments of the muscle and alternative neural sources for the graft have been described [29, 30], the muscle implantation procedure is described below.

Surgical Technique

Preoperatively, the vector of the smile on the healthy side (if present) is noted so that it can be emulated on the affected side. The procedure (Fig. 16.8) is begun by harvest of the gracilis muscle from the medial aspect of the thigh. A curvilinear incision is made 1.5 cm posterior to a line connecting the pubic tubercle to the medial condyle of the tibia. The soft tissues are divided until the belly of the gracilis muscle is identified. The vascular pedicle is located entering the deep surface of the muscle, 8–10 cm distal to the pubic tubercle, and followed proximally for 6 cm. The obturator nerve is identified 1–2 cm proximal to the vascular pedicle and similarly traced. The pedicle, nerve, and approximately 40% of the width of the muscle belly are isolated, using a running locked suture at one or both ends to create pseudotendons useful to subsequent inset. The muscle is then thinned *in situ* by removal of approximately 40% of its superficial surface and removed from the surgical bed (Fig. 16.8a) [31].

A preauricular incision is made and extended to immediately below the mandible to identify the facial vessels for microvascular anastomosis. A thick skin flap is raised, exposing the zygomatic arch and malar eminence, and extends medially to expose the orbicularis oris. In the two-stage procedure, the stump of the cross-face nerve graft is identified in the gingivobuccal sulcus for later neurorrhaphy. In single-stage procedures, the masseteric nerve is identified exactly as described in the reinnervation section above. The gracilis muscle is then secured to the modiolus, stretched to its resting tension length, and secured to the temporalis fascia in the appropriate vector. The microvascular anastomoses and neurorrhaphy are performed, and the incisions are closed in layers over suction drainage (Fig. 16.8b). Movement is expected between 3 and 9 months following muscle transfer (Fig. 16.8c, d).

Alternative free-muscle flaps for facial reanimation, including the pectoralis minor, latissimus dorsi, serratus anterior, and abductor hallucis, have been described. The choice of which muscle to employ is based on surgeon experience.

Static and Adjunctive Facial Reanimation

Patients with facial paralysis are best rehabilitated through combinations of static and dynamic modalities. A treatment algorithm (Fig. 16.9) outlining the evaluation and ongoing management of patients with facial paralysis helps to prevent overlooking and thus undermanaging a specific facial zone. Static procedures are directed at specific functional and cosmetic issues, almost all of which are performed as office procedures under local anesthesia. Management options for the eye, which are paramount, include thin-profile, platinum-weight placement (Fig. 16.10) and unilateral correction of brow ptosis (Fig. 16.11). Nasal obstruction from collapse of the nasal valve caused by paralysis of the dilator nares can be addressed with standard or fascia lata nasal valve repair (Fig. 16.12). If oral incompetence is a significant complaint, resection of the lateral lower lip, fascia lata slings, or both can improve cosmesis and competence in this area. Subtle alteration of the nasolabial fold region to correct effacement or hyperprominence can be performed via minor suture-suspension techniques. Chemodenervation with botulinum toxin and aggressive neuromuscular retraining via physical therapy are vital to optimize facial balance.

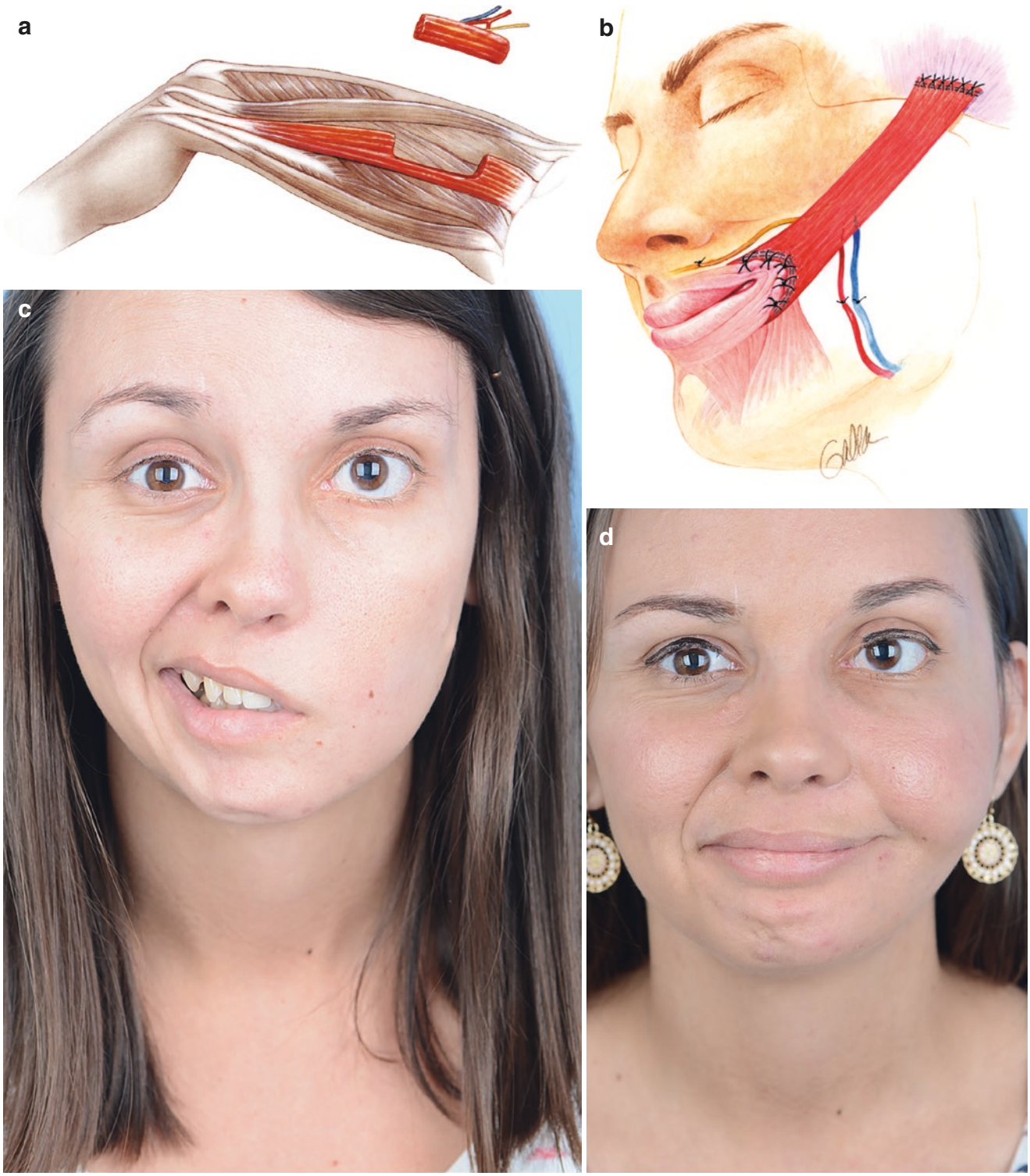

Fig. 16.8 (**a**) The branching pattern of the anterior division of the obturator nerve allows the gracilis muscle to be separated into at least two functional muscular units. A single fascicle usually supplies the anterior 25% of the muscle; the remaining nerve fascicles supply the rest of the muscle. A small portion of the muscle can be harvested with the main vascular pedicle and the fascicle from the anterior branch of the obturator nerve. (**b**) The suture lines can be seen at both ends of the transferred portion of the gracilis muscle. (**c**) Preoperative and (**d**) postoperative views of a patient smiling after gracilis muscle transfer with concomitant static facial suspension. (Figure **a** reproduced with permission from Urken with permission from Cheney et al. [30] © 1995 Mack L. Cheney, MD. Figure **b** from Nadol JB Jr. [32] BC Decker Inc.)

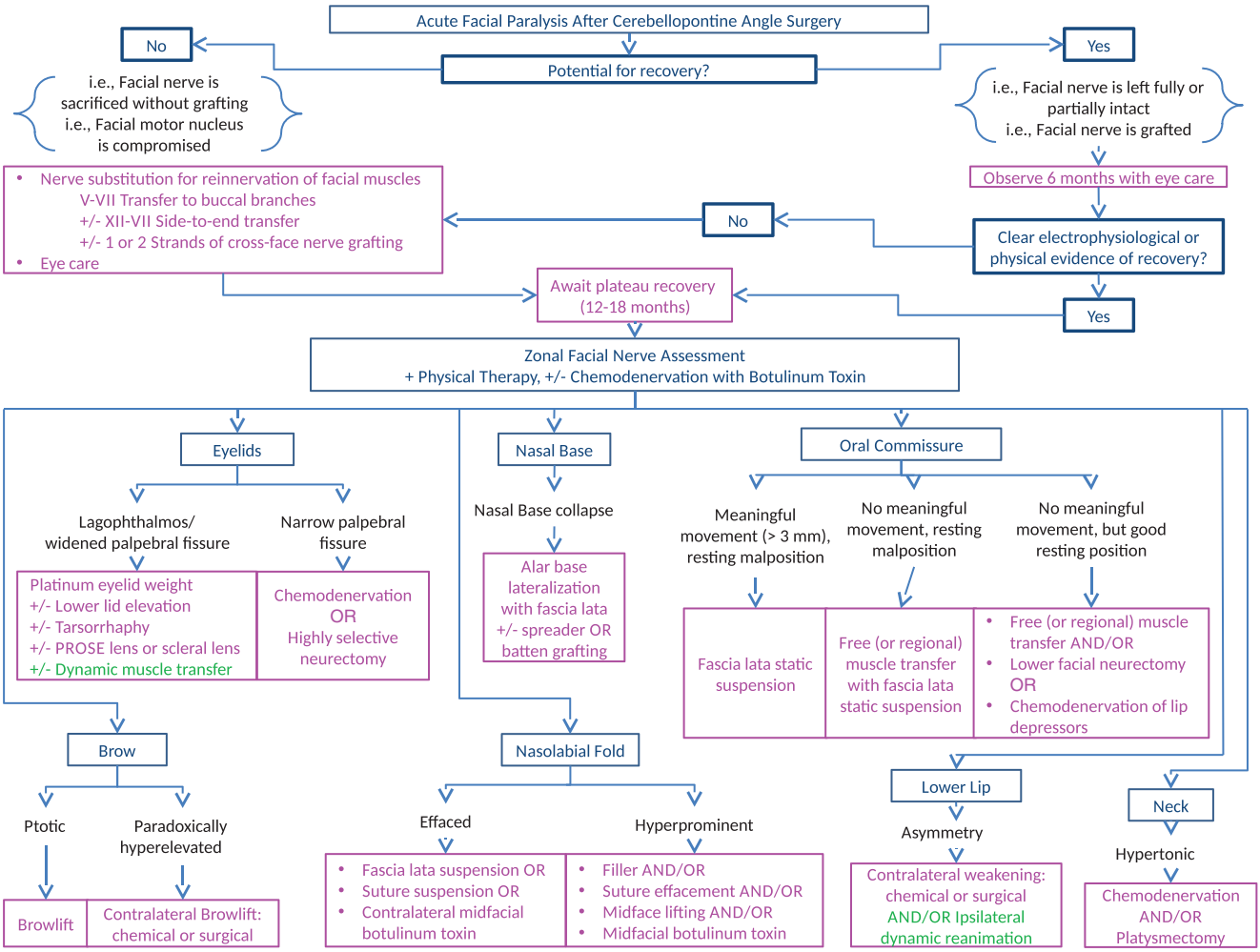

Fig. 16.9 Algorithm for acute facial paralysis after cerebellopontine angle surgery. Blue indicates broader categories, black indicates decision points, pink indicates action and interventions, and green indicates therapy that is not yet mainstream. These interventions are still experimental and describe future directions of treatment

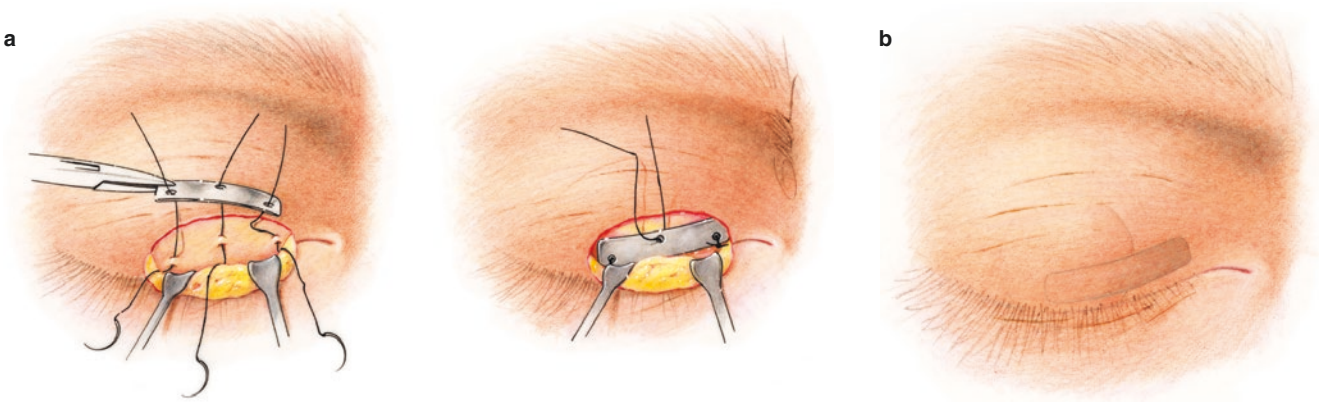

Fig. 16.10 (**a**, left) Implant positioned over tarsal plate. (**a**, right) Implant sutured onto tarsal plate. (**b**) Implant centered between medial and canthal tarsus on eyelid. (Published with permission, © Tessa A. Hadlock, MD and Mack L. Cheney, MD)

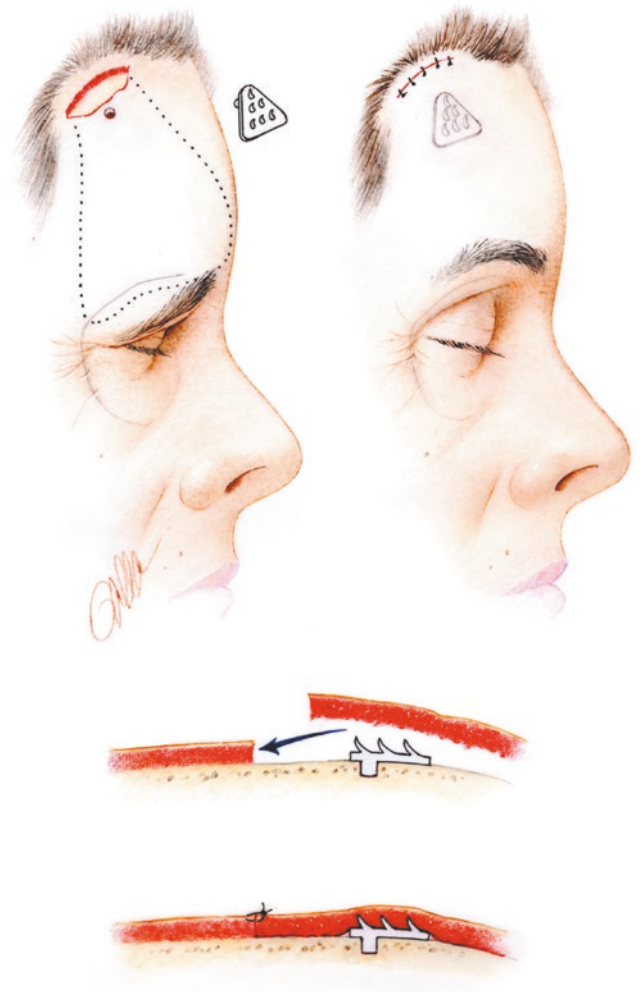

Fig. 16.11 Technique for correction of brow ptosis. Schematic showing lateral views (top) and close-up views of intraoperative (middle) and postoperative (bottom) positions of the tissue with the tine depicted. Note the intradermal position of the tines in the postoperative view. (From Hadlock et al. [33] with permission from Lippincott Williams & Wilkins)

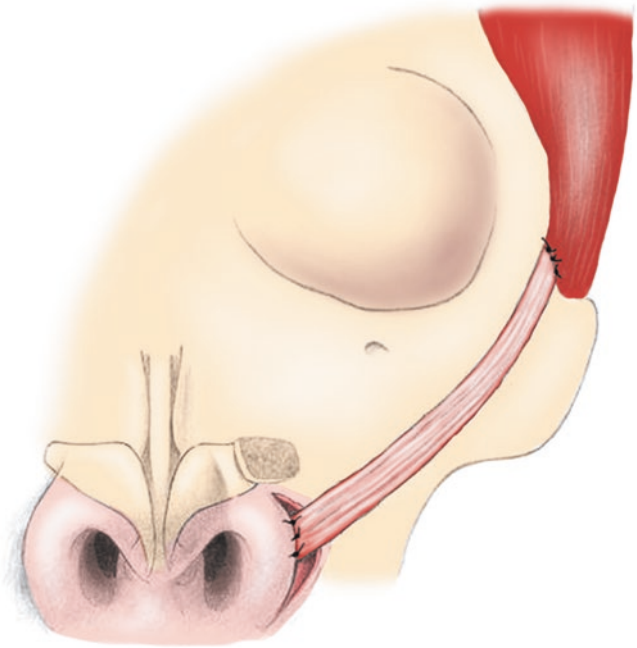

Fig. 16.12 External nasal valve repair via a fascia lata sling from the alar base to the zygoma. (Published with permission © Tessa A. Hadlock, MD and Mack L. Cheney, MD)

Summary

Facial paralysis is a disfiguring and debilitating condition, with its management determined by a large set of clinical variables. A systematic approach to the problem is mandatory. Each facial zone must be evaluated carefully at initial intake as well as longitudinally as the patient's recovery progresses. The complementary roles of reinnervation techniques, muscle transfer, static rehabilitation procedures,

adjunctive physical therapy, and chemodenervation must be appreciated. Optimal comprehensive management is most easily accomplished in a multidisciplinary, high-volume setting.

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