

Facial nerve disorder: a review of the literature

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Abstract

Facial nerve disorders present with varying levels of facial dysfunction. Facial nerve reinnervation techniques aim to correct this by attempting to reestablish the connection lost between the facial nerve nucleus and its distal branches, or by using donor nerves to provide an alternate neural input to the facial nerve. Many facial nerve disorders exist; however, tumors and trauma to the facial nerve are the 2 causes that most commonly result in the patient being considered for reanimation procedures, as they most often result in facial nerve discontinuity. Reinnervation techniques are the first line surgical intervention for facial paralysis when a direct connection between the facial nerve cannot be reestablished, with the XII-VII nerve transfer being the most reliable and having the most predictable outcome when compared with the alternative VII-VII procedure. However, when the reinnervation time window is missed, other techniques of reanimation must be used in an attempt to best restore the normal symmetry and function of the face. The modifications to the XII-VII nerve transfer technique have made it the most popular of all methods; however, there are still many other nerves that may be considered as donors, giving the surgeon other options in the event of the hypoglossal (XIIth) nerve being unsuitable.

Keywords: Facial, Nerve, Disorder, Reanimation, Reinnervation, Techniques, Transfer, Surgery, Plastic

The term “facial nerve disorder” encompasses a wide range of conditions with a variety of causes. The extent of facial paralysis ranges from slight asymmetry to complete paralysis on one or both sides of the face. This has a great effect on the quality of life of the sufferer, with the subsequent weakness of facial muscles potentially causing corneal exposure, oral incompetence, and difficulty with articulation. Despite the clinical implications of these features, such as increased risk of corneal damage and infection, there is also a degree of social isolation due to the esthetic and functional impacts of facial paralysis, which in turn can greatly affect the mental health and well-being of the individual.

The main aim of facial reanimation procedures therefore, is to help the patient both physically and mentally by correcting the facial paralysis, thereby restoring facial symmetry and muscle function.

Treatment options initially focus on reestablishing the direct connection between the facial nerve nucleus and the distal

branches of the facial nerve. Failing that, several techniques can be used in an attempt to regain facial function, with reinnervation techniques being the first-line surgical intervention for facial paralysis.

Anatomy of the facial nerve

Embryology

The facial nerve (cranial nerve VII) is derived from the second pharyngeal arch, along with the (fetal) stapedia artery and the several structures that they supply, namely the posterior belly of the digastric muscle, the stylohyoid muscle, the stapedius muscle, and the muscles of facial expression.

Gasser^[1] stated that during the first 3 months of prenatal life, the course, branching patterns, and anatomic relationships of the facial nerve are already established. In the third week of gestation, a collection of neural crest cells gather to form the rudimentary basis of the facial nerve nucleus. In the fifth week, the chorda tympani and greater petrosal nerves begin to take shape and the motor nucleus of the facial nerve begins to form. In week 6, the branches to the posterior belly of the digastric and stylohyoid muscles are formed and in week 7, the geniculate ganglion and nervus intermedius are observed. In the eighth week of gestation, the stapedius nerve is seen and the temporofacial and cervicofacial parts of the extracranial facial nerve develop. All terminal branches of the facial nerve are seen at the end of week 8. Finally, from weeks 7–12 the muscles of facial expression develop.

The muscles of facial expression are split into 3 main groups—oral, nasal and orbital, and these lie within the subcutaneous tissue of the face, each taking their origin from either the bony surfaces of the skull or from the surrounding fascia.

The oral group contains the orbicularis oris muscle, which functions to purse the lips, the buccinator muscle, which pulls the

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This manuscript has been peer reviewed.

Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

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International Journal of Surgery Oncology (2018) 3:e65

Received 14 December 2017; Accepted 21 December 2017

Published online 25 September 2018

<http://dx.doi.org/10.1097/IJ9.000000000000065>

buccal surface of the cheek inwards against the teeth, and the upper and lower muscle groups, which act on the lips and mouth.

The upper muscle group elevates the upper lip and consists of the levator labii superioris, levator labii superioris alaeque nasi, levator anguli oris, zygomaticus major, zygomaticus minor, and risorius muscles. The lower muscle group depresses the lower lip and consists of the depressor anguli oris, depressor labii inferioris and mentalis muscles.

The nasal group contains the nasalis muscle, which is split into 2 parts and is also the largest of the nasal muscles, with the transverse part closing the nares (nostrils) and the alar part opening them. The other 2 muscles in the group are the procerus muscle, which is the most superior of the 3, acting to pull the eyebrows downwards, and the depressor septi nasi, which pulls the nose inferiorly to assist the alar part of the nasalis muscle in opening the nares.

Finally, the orbital group is made up of 2 muscles, which act to close the eyelids and protect the cornea from damage (muscles involved in opening the eyelids, predominantly the levator palpebrae superioris and the superior tarsal muscle, are not classified as muscles of facial expression). The larger of the 2 orbital muscles is the orbicularis oculi muscle, which is also split into 2 parts. The outer orbital part is involved in forceful closing of the eyelid and the inner palpebral part is involved in gentle closing of the eyelid. The other, smaller muscle is the corrugator supercilii, which lies just posterior to the orbicularis oculi muscle and acts to pull the eyebrows closer together. Skeletal derivatives from the second pharyngeal arch include the stapes, the styloid process and part of the hyoid bone.

Origin, course, and distribution of the facial nerve

Functionally, the facial nerve is divided into 3 parts, with the motor part arising from the facial nerve nucleus in the pons, and the sensory and parasympathetic parts arising from the nervus intermedius or “nerve of Wrisberg,” located between the motor part of the facial nerve and the origin of the vestibulocochlear nerve (VIIIth cranial nerve). The cell bodies of the parasympathetic axons arise from the superior salivatory nucleus in the pontine tegmentum of the pons and the cell bodies of the sensory component are found in the geniculate ganglion.

Following its origin, the facial nerve leaves the cranial cavity via the internal acoustic meatus (along with cranial nerve VIII) to enter the petrous temporal bone. The facial nerve then travels through the petrous temporal bone in the “fallopian canal” where its pathway is separated into 3 segments: labyrinthine, tympanic, and mastoid.

The proximal, labyrinthine segment (so called because of its close, posterior proximity to the cochlea) is the shortest of the 3 segments and is also the narrowest, thus rendering the nerve susceptible to compression damage here from swelling of the nerve caused by infection. In this segment, the facial nerve is observed as running “obliquely forward, perpendicular to the axis of the temporal bone”^[2].

After crossing the labyrinthine segment, the nerve bends (first genu) into a collection of sensory cell bodies, otherwise known as the geniculate ganglion, which proceeds to give off the greater petrosal nerve. The greater petrosal nerve carries parasympathetic (secretomotor) fibers, running forwards in the petrous part of the temporal bone to emerge into the middle cranial fossa through the hiatus for the greater petrosal nerve. It then runs across the floor of the cranial cavity before exiting into the pterygoid canal in the

sphenoid bone, where it joins with the deep petrosal nerve (carrying sympathetic fibers from the periarterial plexus of the internal carotid artery). Both nerves then merge together to form the nerve of the pterygoid canal, which enters the pterygopalatine fossa (it is here where the parasympathetic fibers synapse in the pterygopalatine ganglion). The parasympathetic postganglionic and sympathetic fibers supply the lacrimal, nasal and palatine glands.

The next segment of the facial nerve is the tympanic (horizontal) segment, which runs from the geniculate ganglion to the horizontal semicircular canal, “measuring 8–11 mm in length”^[3]. Here the nerve runs behind the tensor tympani muscle and emerges from the middle ear cavity in between the posterior wall of the external auditory canal and the horizontal semicircular canal.

The nerve then makes another bend into the second genu, which marks the beginning of the third, mastoid segment, which is the longest of the 3. In this segment, the nerve runs straight down along the anterior wall of the mastoid process until its eventual emergence through the stylomastoid foramen. Two main branches are given off the facial nerve in this segment: the small nerve to stapedius muscle and the chorda tympani nerve. The chorda tympani nerve runs in the middle ear cavity between the incus and the malleus to eventually join with the lingual division of the trigeminal nerve. This union between the 2 nerves carries parasympathetic (secretomotor) fibers to the sublingual/submandibular glands and also special visceral afferent (taste) fibers from the anterior two thirds of the tongue back to the geniculate ganglion.

The nerve then leaves the fallopian canal by passing through the stylomastoid foramen, giving off branches to the stylohyoid muscle and to the posterior belly of the digastric muscle before entering the parotid gland (without innervating it). In the gland, the nerve divides at the pes anserinus into 2 major divisions and then 5 subsequent divisions: temporal (frontal), zygomatic, buccal, marginal mandibular, and cervical.

The temporal (frontal) division is made up of 3–4 branches, which run to supply the orbicularis oculi muscle, the corrugator supercilii and the frontalis muscle. It is susceptible to injury at the lateral border of the frontalis muscle, as there is little subcutaneous fat between the nerve and the skin. The surface anatomy used to locate this division is still under debate, with several surgeons suggesting different landmarks.

Pitanguy and Ramos^[4] line is currently the most commonly used landmark, which runs from ~0.5 cm below the tragus to 1 cm above the lateral edge of the eyebrow^[5].

However, Gosain et al^[6] discovered temporal (frontal) nerve branches at the lower border of the zygomatic arch, between 10 mm anterior to the external acoustic meatus and 19 mm posterior to the lateral orbital rim. This variation in the surface anatomy makes it all the more difficult for surgeons when attempting to accurately locate this division of the facial nerve. There are interconnections between the branches of the temporal (frontal) division, but there are none between this division and the other divisions of the facial nerve^[7].

After emerging from the parotid gland, the zygomatic and buccal divisions run forwards over the masseter muscle, under the deep facial fascia, which is subsequently pierced by both divisions at slightly different locations near the anterior border of the masseter muscle. The zygomatic branches pierce the fascia at ~4 cm anterior to the tragus, running anteriorly to supply the zygomaticus major muscle from its deep surface, while the buccal branches pierce the fascia at the anterior edge of the masseter.

The zygomatic and buccal branches together serve to supply the orbicularis oculi, orbicularis oris, and the buccinator muscles. There are several interconnecting branches between the 2 divisions, and as a result of this network between them, any injury to either of the divisions tends not to result in a noticeable defect of the face^[8].

The marginal mandibular division is the one most commonly damaged due to the significant variability in its course, often resulting in surgeons accidentally damaging the nerve during a procedure^[9,10].

The marginal mandibular division can also vary in the number of branches it has, with some surgeons recording one and others recording 3–4^[11,12].

In most individuals, after its emergence from the parotid gland, the marginal mandibular nerve runs in the deep facial fascia, downward toward the inferior border of the mandible (sometimes running 3–4 cm below) to enter the submandibular triangle^[11]. It then ascends into the face, with the main trunk of the nerve lying just superior to the border of the mandible^[12].

This division innervates the lower lip muscles, the depressor anguli oris, mentalis, and the upper platysma and any injury to this division generally produces an obvious defect in the face due to its lack of interconnections with other divisions^[13].

The cervical division emerges from the parotid gland and passes behind the angle of the mandible, after which it runs forwards 1–4.5 cm inferior to the border of the mandible. It often consists of >1 branch and may even communicate with the marginal mandibular nerve. It serves as the main supply to the platysma and due to its communications; injury to the division often goes unnoticed, as it does not cause any obvious deformity.

Fiber types and structures supplied

The facial nerve carries 4 different fiber types: special visceral afferent, general somatic afferent, special visceral efferent, and general visceral efferent. “Special” refers to fibers found specifically in the cranial nerves.

Special visceral afferent fibers carry taste sensation back to sensory cell bodies in the geniculate ganglion from the anterior two thirds of the tongue and the accessory palatine taste buds via the lingual division of the trigeminal nerve (V3). General somatic afferent fibers carry sensory fibers back from the external ear. The fiber type most relevant to this study are the special visceral efferent fibers, which run to supply the posterior belly of the digastric muscle, the stylohyoid muscle, the stapedius muscle and the muscles of facial expression. Finally, general visceral efferent fibers give parasympathetic (secretomotor) supply to the lacrimal, nasal, submandibular and sublingual glands via the first, second, and third divisions of the trigeminal nerve, respectively.

Facial nerve disorders

Disorders of the facial nerve are relatively common and have several possible causes, with each resulting in varying levels of paralysis of the structures innervated by the facial nerve. As stated above, the pathway of the facial nerve is long, passing through the petrous part of the temporal bone in the fallopian canal to emerge onto the surface of the face, and as a result there are a number of areas along this path where it is susceptible to damage.

Bell's palsy

The most common acute facial nerve disorder is Bell's palsy, accounting for 66% of unilateral facial nerve disorders^[14]. It is reported that 85% of patients begin to recover nerve function within 3 weeks from onset of symptoms without any medical or surgical intervention^[14,15]. The incidence of Bell's palsy is thought to increase with age, with the highest incidence in the 65–74 age group. In a study carried out by Morales et al^[16], 14,460 cases of Bell's palsy were recorded in the United Kingdom from 2001 to 2012 with an overall incidence of 37.7/100,000 person-years.

Although there is currently no definitive cause of Bell's palsy, it is thought to be associated with the herpes simplex virus (HSV-1), which causes the facial nerve to swell in the labyrinthine segment of the fallopian canal in the petrous part of the temporal bone. Bell's palsy should only be considered when other causes have been ruled out and it is therefore deemed a diagnosis of elimination.

The more persistent cases of Bell's palsy are often treated with corticosteroids to reduce any inflammation and swelling of the facial nerve. Failing this, the patient is considered for reanimation procedures.

Ramsay Hunt syndrome

Ramsay Hunt syndrome is another infective cause of facial palsy, which develops through reactivation of latent herpes zoster virus (HSV) in the geniculate ganglion of the facial nerve. A characteristic feature of the syndrome is the formation of vesicles within the ear canal, which is often associated with otalgia and varying levels of hearing loss. The incidence of Ramsay Hunt syndrome is much less than Bell's palsy, at ~5/100,000 person-years in the United Kingdom^[17]. Treatment involves the use of antiviral drugs and corticosteroids.

Otitis media

Otitis media is an infection of the middle ear cavity, which can disrupt the function of the facial nerve by causing inflammation and swelling of the nerve through a similar pathology to Bell's palsy. Acute otitis media is common in the United Kingdom, with around 30% of children under the age of 3 being diagnosed with the condition and 97% of them receiving antibiotics^[18]. The condition is much less common in adults.

Otitis media is most commonly secondary to bacterial infection with organisms such as *Streptococcus pneumoniae*, *Haemophilus influenzae* or *Moraxella catarrhalis*. This is routinely managed with the use of antibiotics and corticosteroids.

Trauma

Trauma to the facial nerve is the second most common cause of facial nerve palsy after Bell's palsy, and is divided into 3 types—temporal bone fracture, penetrating trauma, and iatrogenic trauma^[19,20]. Trauma to the side of the head resulting in a fracture of the temporal bone is highly likely to cause dysfunction of the facial nerve as it follows a path through this bone to the stylomastoid foramen. The vestibulocochlear (VIIIth) nerve may also be affected due to its close anatomic association with the facial nerve in this area, resulting in the patient presenting with vertigo and hearing loss along with facial paralysis, which can have sudden onset following trauma to the head. Cerebrospinal fluid leaking from the ear is an obvious sign of trauma and requires immediate action.

There are 2 main types of temporal bone fracture: longitudinal and transverse. A longitudinal fracture is the most common (70%–80%) and is caused by blunt force trauma to the temporoparietal region with a resulting fracture line seen running parallel to the long axis of the petrous pyramid. However, despite being the most common type of fracture, it rarely results in facial nerve paralysis unlike transverse fractures, with only 15% of cases showing a transected nerve in those with longitudinal fractures^[21].

Transverse fractures are caused by blunt force trauma to the frontal or occipital areas, making up 10%–20% of fractures to the temporal bone. The transverse fracture line is seen perpendicular to the long axis of the petrous pyramid, and resulting facial nerve paralysis with a transected nerve was recorded in 92% of cases^[21].

The second type of trauma is penetrating trauma and most often results in injury to the extratemporal segment of the facial nerve. The third type is iatrogenic, caused accidentally during a surgical procedure, most often due to an unknown variation in the branches of the facial nerve, and is most commonly seen following a parotidectomy or mastoidectomy.

Tumors

Tumors present the greatest threat to the integrity of the facial nerve, as they can compress and/or invade the nerve at any point along its path. In addition to this, there is also risk of iatrogenic damage during the resection of the tumour in the operating theatre, resulting in a greater level of paralysis. If the tumor is benign, then the facial nerve is ideally preserved when removing the tumor, but if it is malignant, then a wide excision margin is used to remove the tumor and any surrounding tissue, including the facial nerve.

Acoustic neuromas are a common cause of facial nerve disruption as well as causing problems with the vestibulocochlear nerve, as the course of tumor excision often requires the removal of the facial nerve and/or vestibulocochlear nerve.

Direct trauma to the nerve and tumors are the 2 most likely pathologies that result in patients being considered for facial reanimation procedures as they often cause facial nerve discontinuity (a separation of the facial nerve nucleus from its distal branches), whereas the other causes can often be treated medically without surgical intervention, as the nerve remains intact^[22]. Patients with congenital onset facial paralysis, developmental (craniofacial microsomia) or genetic (eg, Moebius syndrome), are also common candidates for surgery^[23,24].

Before any surgical intervention can occur, there should be a clear diagnosis of the disease or incident responsible for the facial paralysis. The site and level of the nerve lesion should be accurately located and the time between the onset of paralysis and diagnosis should be established, as this is of great importance to the prognosis, (discussed later in further detail). A neurological examination is performed initially, with computed tomography and magnetic resonance imaging scans being utilized when necessary. If the nerve is found to be intact, every opportunity must be given for spontaneous regeneration, with up to 12 months of regular assessments suggested before any surgical intervention is considered.

The House-Brackmann facial nerve grading system

A number of standardized scales are used to objectively describe and quantify levels of facial nerve dysfunction, the most popular of which

is the House-Brackmann facial nerve grading system. This is a simple and accurate system which grades the level of facial nerve function in 6 stages, from HB I (normal) to HB VI (total facial paralysis).

Facial reanimation procedures

When facial nerve discontinuity is appropriately diagnosed and the nerve lesion has been accurately located, the first approach is to attempt to restore the direct connection lost between the facial motor nucleus and the distal facial nerve, otherwise known as neurorrhaphy.

Direct repair of the facial nerve

Primary end-to-end neurorrhaphy is the only technique that will enable the patient to regain spontaneous mimetic (involuntary) facial expression and is ideally carried out within 30 days of initial discontinuity^[25,26].

The 2 ends of the separated facial nerve are identified, after which they are trimmed and the epineurium is peeled back to expose the endoneurial surface. Methylene blue staining is used on both ends of the exposed nerve to distinguish them from other surrounding structures. A blue background paper is then placed under the nerve and 2 perineurial sutures are used to join the 2 ends back together.

May^[27] reports that procedures carried out within thirty days have excellent results, with all patients yielding mimetic movement, the ability to close the eye with minimal mouth movement, and normal facial tone and symmetry restored at rest.

After ~30 days, however, the biology of the facial nerve nucleus is reported to alter unfavorably and the facial nerve distal to the lesion becomes subject to fibrosis and atrophy. Results from this procedure when it has been performed 1 year after discontinuity have been very poor, so much so that reinnervation techniques are preferred at such a point in time^[27].

When direct repair is not possible, reinnervation techniques are the first line of surgical intervention in an attempt to best restore facial symmetry and function. Second line procedures are muscle transposition or static transfer procedures; however, these will not be covered in great detail in this paper.

Reinnervation techniques

Reinnervation techniques provide an alternate neural input to the distal facial nerve when the 30-day time window for direct repair of the facial nerve has been missed or when the proximal facial nerve has been removed during tumor resection. These techniques should ideally be carried out between 30 days to a year after initial injury to the nerve; however, procedures performed up to 2 years after injury can still be successful. Unlike in direct repair of the facial nerve, involuntary (mimetic) movement is not possible with these techniques, but voluntary movement with good facial symmetry and tone is still achieved.

Also termed nerve substitution techniques, the procedure involves the transfer of either the hypoglossal (XIIth) nerve, the contralateral facial nerve, or, less commonly, the glossopharyngeal (IX), trigeminal (V) or spinal accessory (XI) nerves. These donor nerves are transferred to the distal facial nerve and a connection is established to provide alternate motor input to the structures of the face.

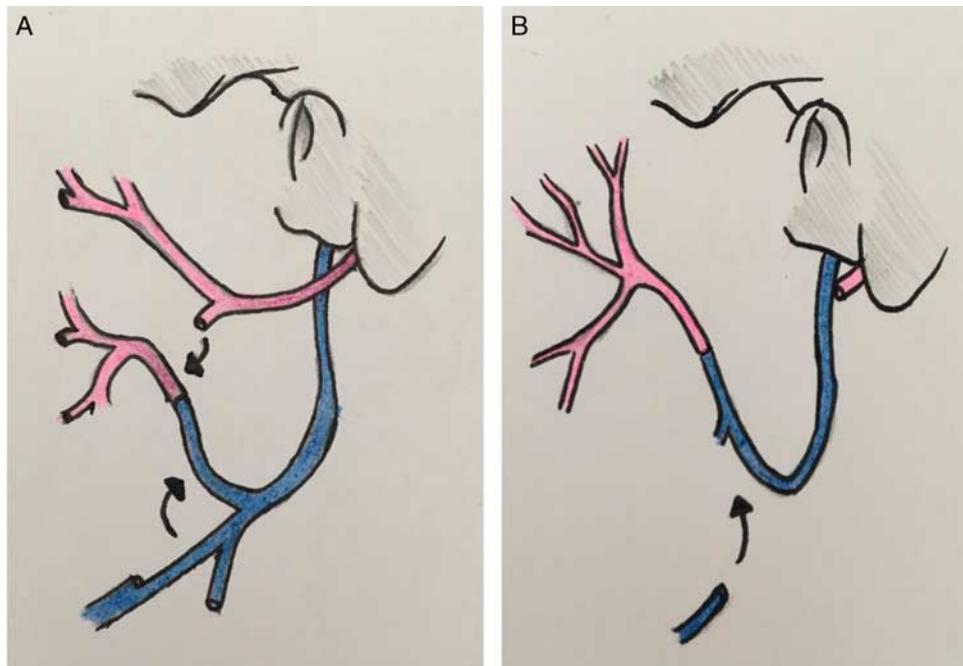


Figure 1. A, The “classic” XII-VII transfer. B, The “split” XII-VII transfer. The hypoglossal nerve is shown in blue and the distal part of the facial nerve in pink^[28].

XII-VII transfer

The hypoglossal nerve was first suggested for use in transposition in 1901 by Körte and Bernhardt and is the nerve most often utilised for reinnervation due to its relatively close proximity to the facial nerve but more importantly because of its highly dense population of myelinated motor axons, which significantly increases the chances of

successful reinnervation. However, as the hypoglossal nerve is the sole cranial nerve responsible for the motor supply to the intrinsic and extrinsic muscles of the tongue, it is highly likely that the procedure will result in hemitongue weakness; a consequence most often accepted by surgeons and patients as the lesser of 2 evils.

The “classic” XII-VII transfer, in which the entire hypoglossal nerve is utilized, is one with an excellent prognosis in terms of

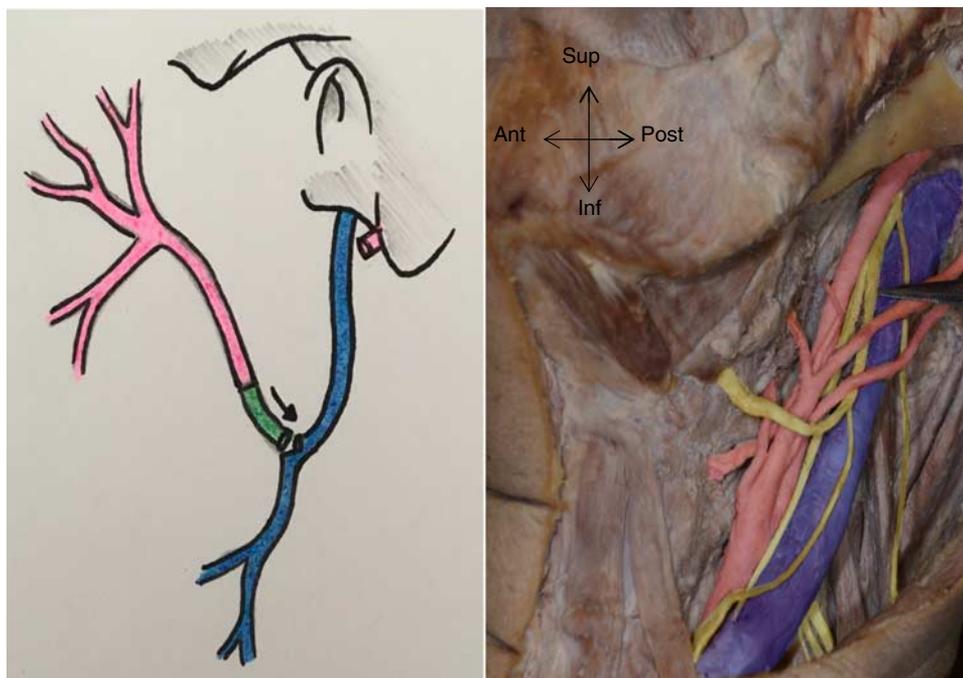


Figure 2. The XII-VII “jump” graft. The great auricular nerve graft is shown in green^[28].

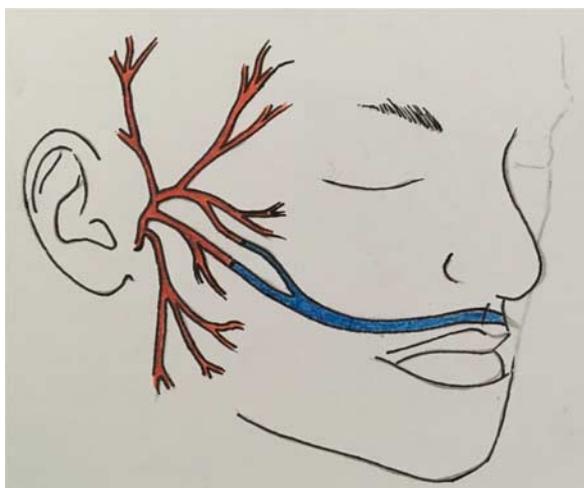


Figure 3. The VII-VII transfer with the sural nerve graft^[28].

restoring facial function and symmetry. In short, the procedure begins with a modified Blair parotidectomy incision, after which the main trunk and pes anserinus of the facial nerve are located and the hypoglossal nerve is located in its ascending portion, deep to the posterior belly of the digastric muscle. The hypoglossal nerve is then freed of its attachments and is reflected upwards to join the distal trunk of the facial nerve.

However, despite the initial success of this procedure, due to the certainty of tongue morbidity, there have since been several modifications to the procedure in an attempt to reduce this.

The first modification to the “classic” procedure is the “split” XII-VII transfer (Fig. 1), which involves dividing 30%–40% of the hypoglossal nerve from its main trunk to be joined up to the distal trunk of the facial nerve, thereby leaving behind some input to the intrinsic and extrinsic tongue muscles^[29].

The second modification to the “classic” procedure, the XII-VII “jump” graft (Fig. 2), aims to reduce tongue morbidity further by avoiding the removal of a significant amount of the hypoglossal nerve and by using an “end-to-side” graft between the hypoglossal nerve and the great auricular nerve (from the cervical plexus, C2–C3), which is then sewn to the facial nerve^[30].

Contraindications to the procedure include those who have an existing problem with the vagus nerve (Xth cranial nerve), as a combined X and XII palsy would severely affect the individuals ability to swallow. A thorough history and examination of the patient before considering for surgical intervention should be sufficient to prevent this issue from occurring.

VII-VII transfer

Cross-facial nerve grafting was first described by Scaramella in 1968 and is carried out in 2 stages, involving the use of a sural nerve graft from the leg, which is attached to the healthy distal branches of the contralateral facial nerve and then most commonly drawn across the philtrum to supply the damaged facial nerve^[31] (Fig. 3).

The first stage of the procedure begins by removing the sural nerve graft from the leg, which is then anastomosed to the buccal and marginal mandibular branches of the healthy facial nerve. Selection of the healthy nerves is achieved through the use of a

nerve stimulator. The nerve anastomoses is performed using the Millesi technique, which first involves separating the fascicles of the sural nerve graft from each other for a distance of 1–2 cm, after which, each is brought into contact with the end of the facial nerve branch to lie in a small loop, without tension, thereby removing the need for sutures^[32].

Six to 12 months later, in the second stage, the graft is tested using Tinels sign (tapping on the graft to evoke a tingling sensation), which if positive, demonstrates that there are regenerating axons growing into the graft^[33]. An incision is then made over the zygomatic arch, anterior to the parotid gland on the affected side of the face, to easily identify the branches of the affected facial nerve, after which the healthy graft is drawn across the philtrum to the contralateral side of the face and attached to the branches of the affected, contralateral facial nerve^[31].

Ethical approval

Ethical approval is not required as this is a review of the literature.

Sources of funding

None.

Author contribution

All authors equally contributed to this article.

Conflict of interest disclosure

The authors declare that they have no financial conflict of interest with regard to the content of this report.

Research registration unique identifying number (UIN)

Not applicable.

Guarantor

None.

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