Facial nerve: Anatomical revision
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Citation

Abstract
This article describes the observations collected from the dissection of nonformalinized cadavers analyzed in respect to the anatomical relation variations of facial nerves. Other findings reported on the medical literature are analyzed.

INTRODUCTION
We manage patients with facial nerve (FN) problems secondary to upper motor neuron or lower motor neuron lesions on every single day and those problems do not have the same characteristic in spite of to be secondary to the same etiology or under similar pathophysiological process therefore we have hypothesized about some anatomical variations of the FN from one patient to another to explain our clinical findings.

There are many reports about malformation of the FN and its anatomical variations, since racial differences in mastoid morphology exist, there may be racial differences in the surgical anatomy of the FN in the mastoid. Low, dissected thirty Chinese adult temporal bones and he found that the facial nerve in the mastoid coursed vertically (60.0%), anteriorly (33.3%) and posteriorly (6.7%) in the sagittal plane. In the coronal plane, it coursed vertically (46.7%) and laterally (53.3%). The chorda tympani was found to have extra temporal origin in 53.3% and for the rest, the distance of its origin from the stylomastoid foramen averaged 3.17 mm (range 0.5-6.0 mm). The mean dimension of the extended facial recess was 4.40 mm (range 3.0-6.0 mm). The FN was at a mean of 3.15 mm (range 2.0-5.0 mm) posterior to the most posterior point of the tympanic annulus and partially crossed the tympanic annulus from medial to lateral at this point. In conclusion, the typical surgical anatomy of the mastoid FN as described in the Western literature may not apply to the Chinese.

The FN is a mixed peripheral nerve being the cranial nerves most commonly affected in neurology. Its main function is motor (Facial expression muscle, buccinators, posterior digastrics, Stylohyoid, Stapeius), also has sensory function (Taste – Ant. 2/3 tongue) and autonomic (Parasymp. –Lachrymal, Sub Maxillary and Sub Lingual glands) we test FN functions on bedside activities very easy (Smile, screw eyes, whistle, show teeth, taste ant 2/3 rd). Main branches of FN are: A. Posterior auricular branch to occipital belly of occipitofrontalis and auricular muscles, B. Branch to posterior belly digastrics and stylohyoid muscle. C. Temporal branch. D. Zygomatic branch. E. Stylostomatoid foramen. F. Bucal branch (buccinators). G. Marginal mandible branch. H. Cervical branch (Platysma).

In order to get a better idea about variations of FN we reviewed the investigation done by Häusler and his collaborators. He performed 595 stapedotomies between 1992 and 1999 and found 40 cases (6.7%) where the facial nerve had an abnormal course. In 32, a partial nerve prolapsed over the oval window was noted with (6 cases, 1 being a duplicated nerve around the oval window) or without (26 cases) dehiscence in the long bony canal. In 8 cases, there was a total prolapsed of the nerve over the oval window, with 2 special cases: facial nerve having an inferior course over the oval window and the promontory; facial nerve being widely spread over the oval window and the promontory. Concomitant anomalies of the stapes were seen and several patients had dimorphic syndromes with conductive hearing loss since early childhood. Stapedotomy was performed in 39 patients. In the 32 cases of partial nerve prolapsed, a small piston (0.4 mm) was placed in the lower part of the oval window which was sometimes enlarged towards the promontory, except when the nerve was duplicated: the prosthesis was placed into the footplate between the nerve branches. In the 8 patients with total facial nerve prolapsed, the prosthesis was either placed directly in a burr hole into the promontory just below the oval window (6 cases), or, when the nerve ran over the
promontory and over the oval window, the prosthesis was placed above the oval window at the site where the facial nerve is usually located (1 case). In the case where the nerve was spread widely over the oval window and the promontory, no prosthesis was placed. In the 39 patients where a stapedotomy was performed, the average hearing level gain ranged from -15 dB to 40 dB (average: 18 dB) at 0.5, 1, 2 and 4 kHz. The average residual air-bone gap was <30 dB in 36 patients (92.3%), < 20 dB in 30 (77%) and < 10 dB in 16 (41%). A post-operative additional hearing loss >= 10 dB occurred in 3 cases (10, 12.5 and 12.5 dB). There were no cases of post-operative deafness or facial palsy. This analysis shows that in many cases with an aberrant course of the FN stapedotomy using adequate and sometimes non-conventional techniques can give post-operative hearing improvement.

The FN is the most frequently injured of all the cranial nerves, causing paralysis of the muscles of facial expression. It is a mixed nerve but the motor component is the most important. The pathways follow by the facial nerve and the relation are very important and carry great significance for anatomist, neurosurgeon and clinicians in order to made accurate diagnosis and effective surgical intervention. The main anatomical consideration will be described in order to understand some clinical implication.

The length and complexity of the anatomical course of the facial nerve explains the difficulty of its accurate morphologic evaluation. Anatomical variations exist in length or thickness of all intrapetrous segments or as frequent dehiscence's which can lead to false positive results or at the opposite falsely negative diagnoses. Close relations with the antero-inferior cerebellar artery in the intracisternal and intracanalicular segments must be known. Gadolinium enhancement is usual in the fallopian canal with variable intensity and thickness and should be differentiated from pathological enhancement. Finally the intrapetrous course of the chorda tympani can be precisely displayed on CT in the intra-osseous canal and in the middle ear near the ossicles.

Facial nerve is known to have considerable variations more so in the temporal bone. An otologist with inadequate familiarity with facial nerve usually has a tendency to do incomplete surgery in chronic suppurative otitis media. The tympanomastoid segment of FN has variations in length and in its relation with various middle ear structures. Further the nerve, in Indians is also at variance as compared to Japanese and Americans probably because of different racial configuration of the skull.

A rare facial nerve anomaly was incidentally discovered whilst performing a tympanoplasty and ossicular reconstruction on a patient with an acquired unilateral conductive hearing loss. The nerve was seen to bifurcate and straddle a normal stapes superstructure as it ran posteriorly through the middle ear, a unique and as yet unreported combination. This case highlights the importance of vigilance regarding FN anatomical variations encountered during middle-ear surgery thus avoiding inadvertent damage.

Abnormalities of the facial nerve may occur in conjunction with malformations of the ear, in isolation without associated anomalies, or in conjunction with a variety of syndromes that include abnormalities elsewhere in the body. In the newborn, the otolaryngologist evaluating a facial paresis or facial palsy must decide whether it is congenital or acquired. One in 2000 live births has a unilateral facial palsy, with a 90% spontaneous recovery rate. Approximately 75-80% of palsies in newborns are related to birth trauma. A history of forceps delivery, prolonged labor, ecchymosis over the mastoid, or hemotympanum raises suspicion for birth trauma.

The presence of bilateral facial paralysis, other cranial nerve deficits, or other anomalies suggests a developmental etiology. Early accurate diagnosis is important if the etiology is traumatic. In rare cases, surgery and facial nerve repair may be required in the newborn if the etiology is determined to be traumatic.

Evaluation of FN paralysis includes evoked electromyogram (EEMG), CT scan, and electromyogram (EMG). If the etiology is traumatic, the nerve can be stimulated for 3-5 days postnatal; fibrillation potentials on EMG develop 14-21 days after birth. If the cause is not traumatic, treatment generally is delayed. Eye protection rarely is required in congenital facial paralysis.

In patients with congenital malformations, eliciting the fetal age at which development was arrested is usually possible. This allows for elucidation of the anatomy of the malformed structure based on its normal course of embryological development. Furthermore, if anomalies are present in other organ systems (in particular the kidney), they often reflect arrested development at the same time during development. In this way, the surgeon should be able to predict the location of the facial nerve, particularly in the case of middle ear malformation.
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Most hereditary conditions that include facial paralysis are manifest at the time of birth. However, a few hereditary syndromes are associated with the development of facial paralysis later in life. In addition, many hereditary and congenital malformations are associated with abnormal facial nerve anatomy in the presence of normal nerve function. The otolaryngologist must be familiar with these conditions because abnormal development may place the nerve at increased risk of injury during otologic surgery.

Many abnormalities of the facial nerve canal in the petrous temporal bone have been documented. The most common is congenital bony dehiscence of the CN7 canal that occurs in up to 55% of otherwise normal temporal bones, predominantly involving the tympanic portion (91%) [1]. With this high prevalence, it is more accurately described as a variation in normal anatomy [6,7]. Anomalies of the CN7 canal are infrequently found in normal temporal bones and are usually seen in association with middle and inner ear dysplasias [4]. Anteromedial displacement of the labyrinthine segment of CN7 has been described in association with cochlear malformations, and an anteriorly displaced CN7 mastoid segment is often noted with congenital aural atresia [9,10]. An anomalous course of the tympanic segment has also been reported in association with oval window atresia [8,11].

Bifurcation and trifurcation anomalies of CN7 have been previously described in the otolaryngology literature. These have been reported to involve all portions of the nerve from the intracanalicular segment to the mastoid segment, with the most common anomalies occurring along the tympanic segment [9,10,11,12,13]. Bifurcation of the intracanalicular CN7 has been reported both within one canal and with a double internal auditory canal containing a facial nerve in each. In both case reports, there was a strong facial response to stimulation of each facial nerve segment [13,14].

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It has three nuclei of origin located in the Pons. Two motors and one sensory nucleus.

1. Facial motor nucleus: the axons of these cells carry special visceral efferent to supply the muscle of facial expression.

2. The superior salivatory nucleus: Contain presynaptic parasympathetic neurons to supply the lachrymal and salivary glands.

3. Nucleus of solitary tract: Containing gustatory afferents fibers from the anterior two third of the tongue.

The parasympathetic motor fibers from the superior salivatory nucleus are grouped with the gustatory fibers to form the nervus intermedius. (See picture 1)

The fibers from the motor nucleus remain alone. These fibers after leaving the motor nucleus situated in the inferior part of the Pons beneath the four ventricle pass around the nucleus of the abducens nerve forming an elevation in the rhomboid fossa called facial colliculus. This bend around the
abducen nucleus with the ascending part is known as the genu. (See picture 1)

Sites of emergence:

The two roots, exit in the cerebellopontine angle between the Pons and the olive, medial to the exit of cranial nerve VIII. The nerves intermedius as the name imply exit between the motor root and cranial nerve VIII. (See picture 2)

Those nerves travel together in the posterior cranial fossa into the internal acoustic meatus, where Facial nerve is located on top. The average length of those nerves between the exit in the brainstem and the entrance in the internal acoustic meatus is 15.8 mm. (See picture 3)

The facial nerve travels through the petrous part of the temporal bone, in which four segment are described: meatal, labyrinthine, tympanic and mastoid segments.

The labyrinthine segment is the narrowest and shortest part (3.5-4mm). At the end of this part the nerve change direction and forming a genu that marks the location of the geniculate ganglion.

One branch originates from this segment:

The greater petrosal nerve.

This nerve carries parasympathetic fibers from the superior salivatory nucleus and make synapses in the pterigopalatine ganglion. The postganglionic fibers supply the lachrymal and nasal glands. (See picture 5)

The tympanic segment extends from the geniculate ganglion to the horizontal semicircular canal, (8-11mm in length), it then bend backward and arches downward in a vertical direction to the stylomastoid foramen (mastoid segment). Its length is approximately 10-14 mm.

Two branches are given in this segment:

The nerve for the stapedius m.

The chorda tympani nerve. (See picture 5)

This nerve unites the lingual nerve and conveys:

1- Presynaptic parasympathetic fibers that synapse in the submandibular ganglion, them the postganglionic fibers supply the submandibular, sublingual and small salivary glands in the anterior two third of the tongue.

2- Gustatory fibers from the anterior two third of the tongue. The first order neuron is in the geniculate ganglion, and the central axon makes synapses in the nucleus of the solitary tract.

Once the nerve exits the stylomastoid foramen give of branches for:

Posterior auricular m

Stylohyoid muscle.

Posterior belly of digastic muscle.

After given these branches the main trunk pass lateral to the styloid process and penetrates in the parotid gland forming the intraparotid plexus, from which the following branches are given: (temporal, zygomatic, bucal, Mandibular and cervical) for the muscles of facial expression. (See picture 4)

Pathway for voluntary movement of facial expression's muscles. (See picture 6)

1- Upper motor neuron: Primary motor cortex (Precentral gyrus)

The axons of these neurons enter the Corticonuclear fiber bundle to reach the second lower motor neuron in the Pons.

2- Lower motor neuron: Facial motor nucleus.

The facial nucleus is divided into two parts:

The upper part receives bilateral innervations, and supplies the muscles of the forehead and eyebrows (temporal branches).

The lower part receives innervations mainly from the contra lateral hemisphere, and supplies the muscles of the lower part of the face through the facial nerve.

This arrangement allows the examiner to differentiate central (supranuclear) paralysis from the peripheral (infranuclear) paralysis.

In the central paralysis (loss of the upper motor neuron) the patient will present clinically with paralysis of the contra lateral muscle of the facial expression in the lower half of the face, the contra lateral forehead and extra-ocular muscles remain functional.

Peripheral paralysis (loss of lower motor neurons) is characterized by complete paralysis of the ipsilateral muscles. The patient cannot wrinkle the forehead, the corner
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of the mouth sags, articulation is impaired, and eyelid can not be fully closed (Bell phenomenon), additional deficits may be present such as decreased lacrimation and salivation or loss of taste sensation of the anterior two third of the tongue.

Figure 1
Picture 1: Anterior view of the brainstem. The origin of the facial nerve in the pentane nuclei is represented. (Dissection done by G Milanes-Rodriguez from WSU)

Figure 2
Picture 2: Base of the brain. Exit of the cranial nerves V, VII and VIII from the brainstem. (Dissection done by G Milanes-Rodriguez from WSU)

The upper part of the facial motor nucleus receives from both sides of the cortex and supplies the muscles of the upper part of the face. The lower part of the nucleus only receives contra lateral and supply the muscle s of the lower part of the face.

CONCLUSION

Anatomical variations of FN can be observed on persons from different origin and among people from the same ethnical group and it can explain some clinical variations in patients affected by the same pathological process, but to probe this theory large series should be investigated.

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