INTRODUCTION

Face is the interpreter of emotions, mirror of soul, powerhouse of senses and way for communication.

Facial nerve is the seventh cranial nerve which serves vital functions of lacrimation, salivation, taste, hearing and facial expression.It necessitates urgent measures to understand the cause and nature of nerve injury and undertake immediate steps for restoration and rehabilitation of facial symmetry. About 90% of lower motor neuron facial nerve disorders- inflammatory, traumatic or neoplastic, happens along its intratemporal course.

Over past two decades, many developments have been made. Newer technologies like radiology, electrodiagnostic study and emergence of intraoperative monitoring have been helpful. Complex course within the bony canal, congenital dehiscence, fine branching, interconnections, segmental blood supply, all these factors show significantly, heading towards causation and final result of insult to nerve. A list of etiologies commonly Bell's palsy, followed by Temporal bone fracture, Iatrogenic trauma, Herpes Zoster Oticus , Otitis Media (OM), CerebelloPontine (CP) angle or Intratemporal neoplasm (facial nerve Schwannoma), result in facial nerve paralysis.

The nerve testing depends on determining the scale of distal axonal degeneration (electrodiagnosis), function of branches of the nerve (topodiagnosis) and radiologic guidance in indicated cases[1]. The pathways of the facial nerve are variable, and knowledge of the key intratemporal and extratemporal landmarks is essential for accurate physical diagnosis and safe and effective surgical intervention.

EMBRYOLOGY

Facial nerve is second pharyngeal nerve arch . Gasser[2,3,4] and Sataloff [5,6] classified the development of facial nerve in embryos in accordance to length and denoted embryonic age in weeks. Gerhardt [7] explained caudal deviation of facial nerve in relation to facial ganglion as well as developmental malposition of nerve. The motor nucleus of facial nerve is medial to abducent nucleus. The facial ganglion forms common complex with vestibulocochlear ganglion(Figure 1).

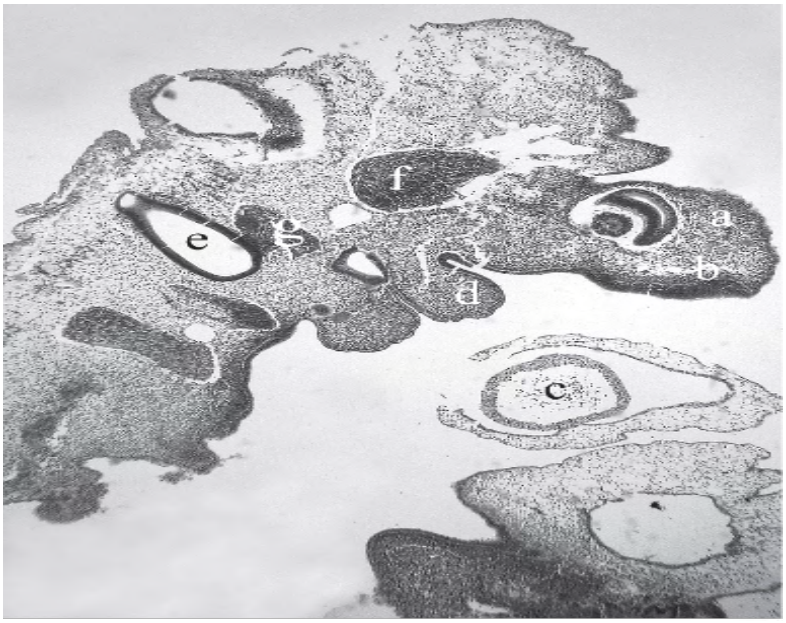


Fig 1. Sagittal section of human embryo at stage 13.
Cresyl violet, ×40; a — frontonasal eminence; b — olfactory crest; c — heart; d — first pharyngeal arch; e — otic vesicle; f -trigeminal ganglion; g — facial ganglion.

Ganglion presents a fusiform structure with cells in rows and is penetrated through nerve fibres (Figure 2). Ganglion is in contact with epipharyngeal placode of second pharyngeal arch and is located ventrally and anteriorly to the vestibulocochlear ganglion at level of the rhombencephalic neuromere 4 . The facial neural crest is visible. From trunk of the facial nerve originates the chorda tympani the first branch (Figure 3). It passes anteriorly whereas main trunk of the facial nerve courses ventrally and posteriorly along direction to second pharyngeal arch.

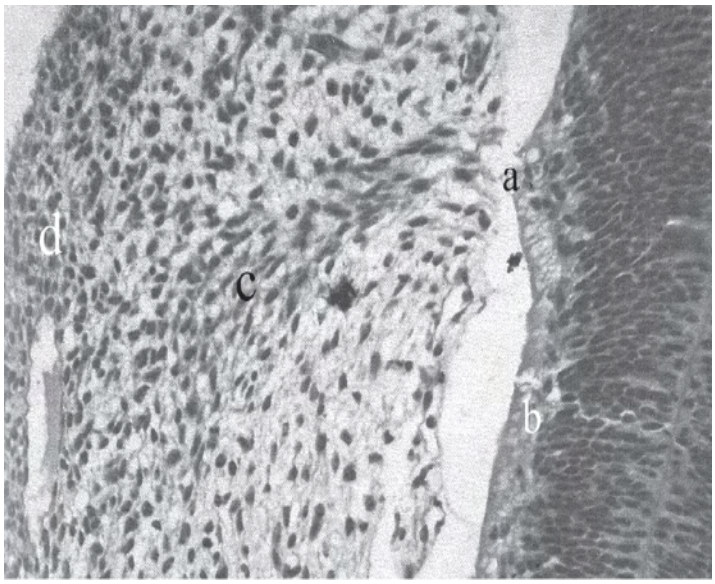


Figure 2. Sagittal section of human embryo at stage 13. Cresyl violet, ×400; a — roots of the facial nerve; b — rhombencephalon; c — facial ganglion; d — epipharyngeal placode of the second pharyngeal arch.

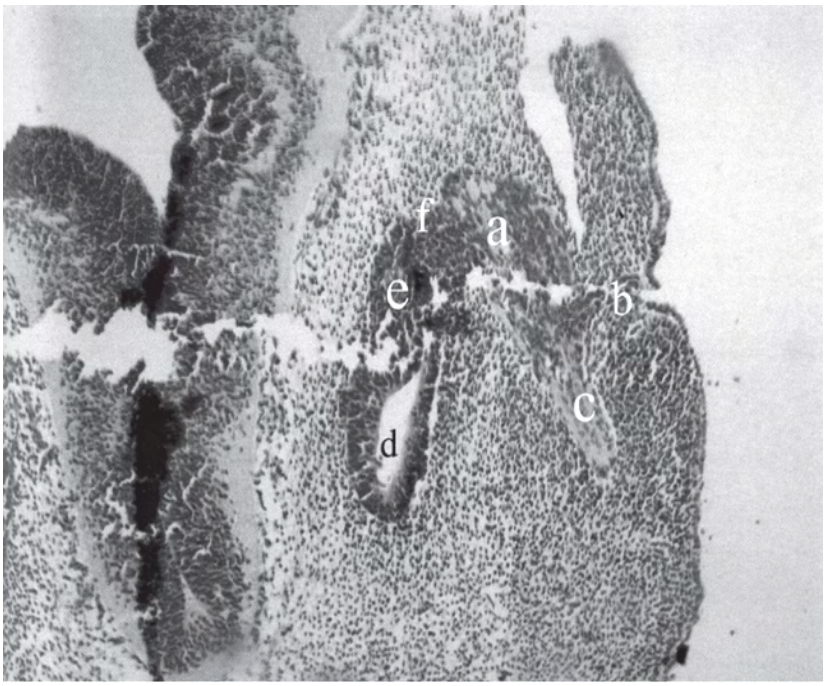


Figure 3. Frontal section of human embryo at stage 14. Bodian's protargol, ×100; a — facial ganglion; b — chorda tympani; c — trunk of facial nerve; d — otic vesicle; e — vestibular ganglion; f — cochlear ganglion

The motor nucleus of facial nerve lies medially to abducent nucleus in the future dorsomedialnuclear column. The intracranial part of facial nerve elongates. At the level of the facial ganglion arises greater petrosal nerve. It is directed anteriorly (Figs. 4, 5). Ventrally to greater petrosal nerve originates the chorda tympani that also passes anteriorly.The main trunk of facial nerve forms a slide arch bent anteriorly. This segment may be considered as the primordium of the horizontal part of the nerve.

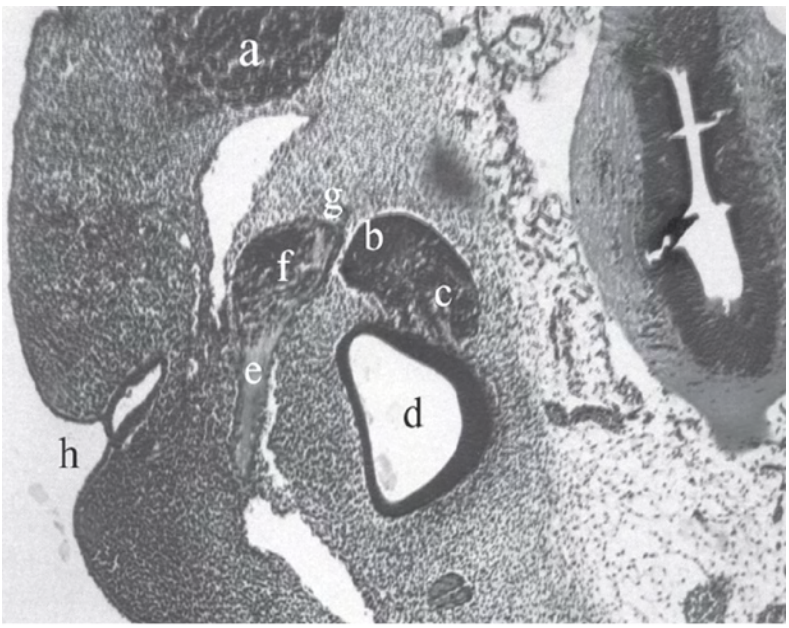


Figure 4. Horizontal section of human embryo at stage 15. Bodian's protargol, ×100; a — trigeminal ganglion; b — cochlear ganglion; c — vestibular ganglion; d — otic vesicle; e — trunk of facial nerve; f — facial ganglion; g — greater petrosal nerve; h — external auditory meatus

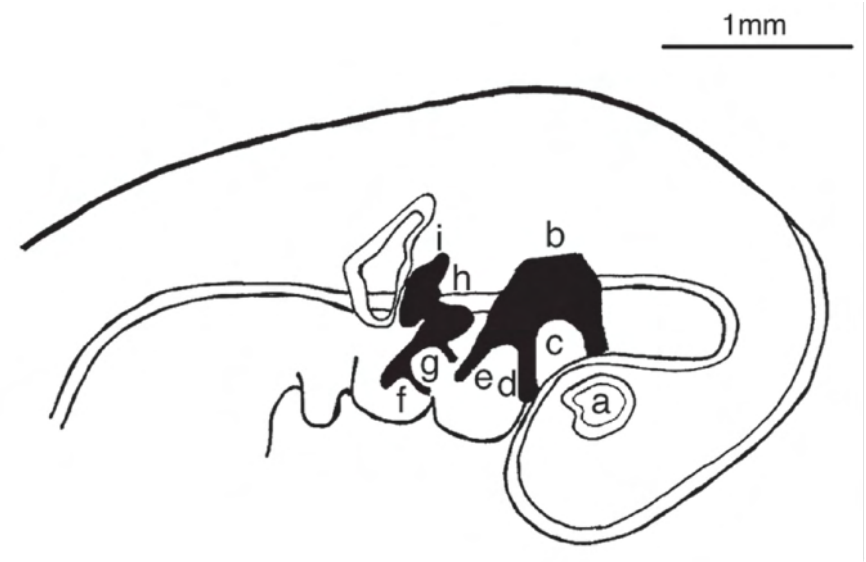


Figure 5. Reconstruction of brain in human embryo at stage 15; a — eye; b — trigeminal ganglion; c — ophthalmic nerve; d — maxillary nerve; e — mandibular nerve; f — chorda tympani; g — greater petrosal nerve; h — facial ganglion; i — cochlear and vestibular ganglia.

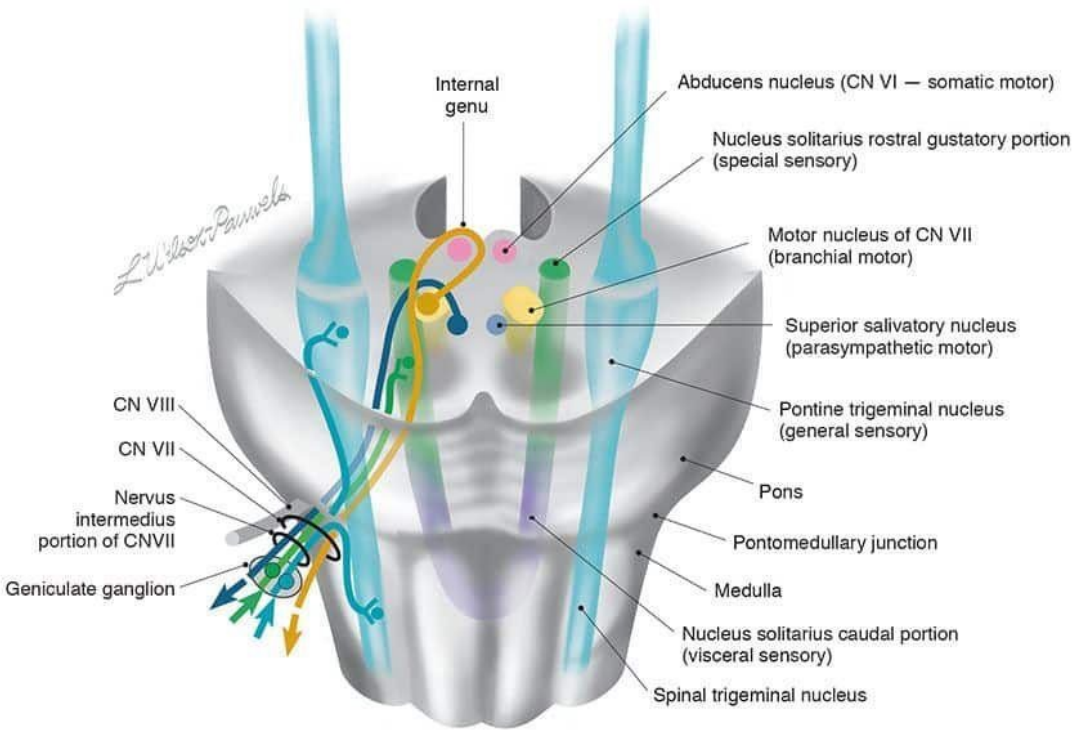
Embryos aged 5 weeks, the facial ganglion detaches from the vestibulocochlear ganglia and 2 branches of nerve appear . The chorda tympani at stage 14 and greater petrosal nerve at stages 15 [8] , Sataloff found facioacoustic primordium in embryo of 3 weeks and identified the chorda tympani in embryo of 4.8 mm long which corresponds to stage 13 and extra cranial branches of the facial nerve in embryos of 5 weeks . Gasser determined the age of embryos in accordance to length and development of the facial nerve into 4 stages. During stage I (embryos 4.2–6.5 mm) the ganglion forms common primordium with eighth nerve ganglion and the chorda tympani arises. In stage II (embryos 8.0–20.0 mm) appears the greater petrosal nerve which joins deep petrosal nerve. Gasser found the chorda tympani in embryos at stage 15. In embryos at stage 13 the facial nerve gives no branches.

ANATOMY OF FACIAL NERVE

The seventh cranial nerve contains somatic efferent (motor) fibres, visceral efferent (parasympathetic)fibres, general somatic sensory fibres, and special visceral sensory (taste) fibres [9,10] . Both sensory and motor roots have myelinated and unmyelinated axons.

The total number of myelinated axons ranges between 7000 to 9000 in motor root and 3000 to 5000 in the nerves intermedius [11,12] . Buskirk [9] found out that 58% of the fibres of facial nerve in human are motor, 24% are autonomic rest 18% are sensory. The intracranial segment of the facial nerve stretches from its exit at

posterior border of the pons in cerebellopontine angle to internal auditory meatus. From the brainstem the nerve emerges into two roots: motor and sensory. The motor root is larger than sensory root. The sensory root (intermediate nerve),contains sensory and parasympathetic fibres [13,14,15,16]. The sensory fibres of intermediate nerve comprise the larger part of the nerve. Within the internal auditory meatus, the intermediate nerve and motor root unite to form a single trunk which enters the facial canal.



Brancial motor (special visceral efferent)	Supplies the muscles of facial expression; posterior belly of digastric muscle; stylohyoid, and stapedius.
Visceral motor (general visceral efferent)	Parasympathetic innervation of the lcrimal, submandibular, and sublingual glands, as well as mucous membranes of nasopharynx, hard and soft palate.
Special sensory (special afferent)	Taste sensation from the anterior 2/3 of tongue; hard and soft palates.
General sensory (general somatic afferent)	General sensation from the skin of the concha of the auricle and from a small area behind the ear.

INTRACRANIAL PART (CISTERNAL PART)

Axons arise from neurons in the facial nuclear complex from the lateral part of Central tegmentum of pons rostral to pons-medulla transition [17] and run posteromedially through the pons, hooking over the abducens nerve nucleus to raise facial colliculus on the floor of fourth ventricle, before turning anterolaterally to leave brainstem.

When they pass through anterolateral aspect of pons , SVE motor axons are joined by general visceral efferent (GVE) preganglionic parasympathetic axons from neurons in superior salivatory nucleus that will finally synapse on postganglionic neurons in either pterygopalatine ganglion (PPG) or submandibular ganglion [18].

The GVE, SVA, GSA (and GVA) axons forms nervus intermedius which was firstly described by Heinricus Augustus Wrisberg [19].

This nerve usually lies between the motor root of facial nerve and the vestibulocochlear nerve as it exits the brainstem (**Figure 6**).The axons of motor root of facial nerve and the nervus intermedius mix within the substance of pons but separate onto the surface of the brainstem, deep in the pontomedullary sulcus.

The cisternal portion is 24mm long.

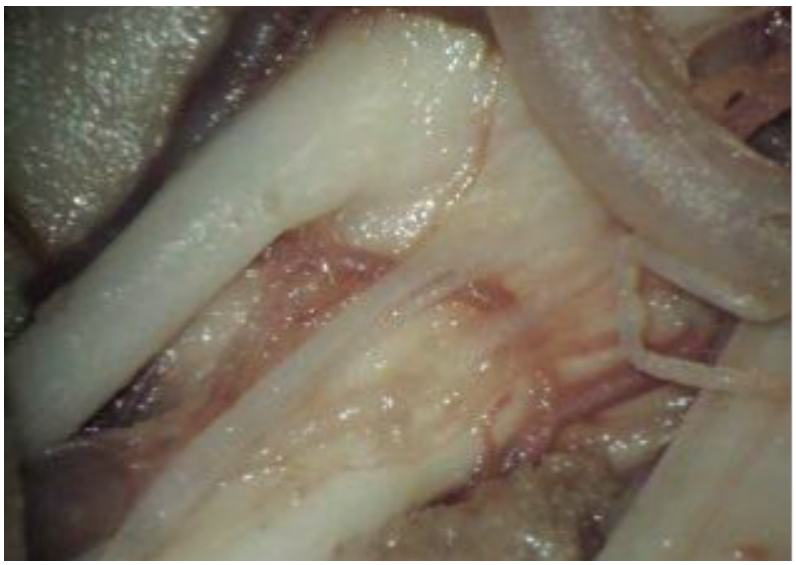


Figure 6. Right sided specimen showing the facial nerve (VII),the vestibulocochlear nerve (VIII), the nervus intermedius (NI), and a vein between cranial nerves VII and VIII. In this case, the NI (three bundles) arises separately from the brain stem (bs). The choroid plexus (pc) is located anteroinferiorly to the VII at the entry of the foramen of Luschka. From Alfieri et al.[20].

INTRATEMPORAL PART

The intratemporal portion is 30 mm long and further divided into meatal, labyrinthine, tympanic (horizontal) and mastoid (vertical) segments. The meatal segment goes in the IAM; the labyrinthine segment goes laterally to the first genu and the geniculate ganglion; the tympanic segment goes backwards from geniculate ganglion to second genu; the vertical segment goes from second genu to stylomastoid foramen. Extratemporal segment goes towards parotid gland where it divides into temporofacial and cervicofacial branches [21].

MEATAL SEGMENT

The meatal segment is 5–12 mm in length. The motor root of the seventh nerve and the nerve of Wrisberg run through the internal auditory canal from its porus (medial) to its fundus (lateral wall), along with eight nerve and the superior and inferior vestibular nerves and the labyrinthine artery (**Figure** 7). The bundles of nervus intermedius join the facial nerve within the internal Auditory Meatus, 3 mm from the porus [22]. Many degrees of rotation between the facial and vestibulocochlear nerves has been reported within the IAM [23]. The parts of the vestibulocochlear nerve rotate 90 degrees as they go from brainstem to the inner ear. The seventh nerve remains ventral in its course to the porus acousticus and in the IAM and also lies anterior to the superior vestibular nerve in the lateral end of the meatus [24]. On cross-section, fundus of IAM is segregated into four quadrants. It is compartmentized horizontally by a thin bony septum, the crista falciformis and vertically into anterior and posterior

compartments above the crista by a bony landmark , Bill's bar (named after **William Fouts House**). The motor root of seventh nerve and the nervus intermedius of Wrisberg lie anterior to Bill's bar in anterior superior quadrant; the superior vestibular nerve lies behind the Bill's bar in the posterior superior quadrant; the cochlear nerve and inferior vestibular nerve go below the transverse septum with cochlear nerve lying ventral to the inferior vestibular nerve (**Figure 7**).

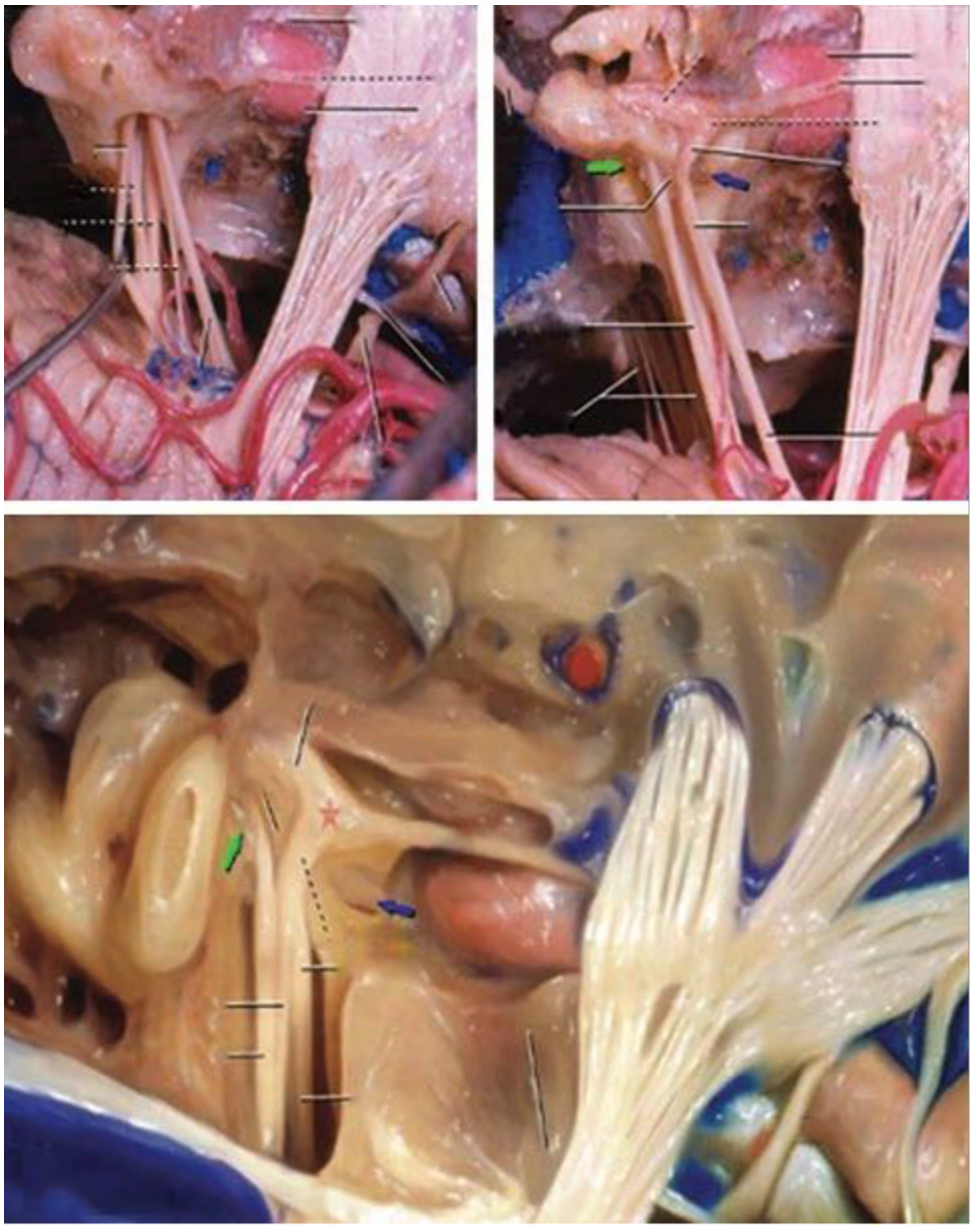


Figure 7 showing IAM and its contents [25]

LABYRINTHINE SEGMENT

The labyrinthine segment is shortest (3–5 mm) and narrowest part of the facial nerve (diameter of 0.68 mm). This segment of facial nerve lacks anastomosing arterial cascades and is likely to be affected by ischaemia in case of oedema following inflammation. The distal end of the labyrinthine segment, the geniculate ganglion forms a sharp 'hairpin' turn, first genu of the facial nerve and marks the beginning of the tympanic segment. At the genu, the nerve is related to the superior semicircular canal posteriorly and the cochlea anteroinferiorly. (**Figure 8)** Subarachnoid space may reach as far as the geniculate ganglion [26].

The geniculate ganglion is a sensory ganglion with no synapses. The central processes of the GSA neurons in first genu ganglion carry pain from external acoustic meatus and end somatotopically in spinal tract nucleus of trigeminal nerve. The central processes of SVA neurons in first genu ganglion carry taste from anterior two thirds of tongue via chorda tympani end somatotopically on second order neurons in rostral end of the solitary tract – gustatory nucleus . Trigeminal and glossopharyngeal sensitivity is thought to be modulated by chorda tympani [27,28] .Without synapsing in the geniculate ganglion, Preganglionic parasympathetic axons synapse on postganglionic neurons either in the PPG or submandibular ganglia. Those going towards PPG form the greater petrosal nerve(the first branch of the facial nerve)[29,30] . From geniculate ganglion , GSPN runs antereomedially and receives a branch from the tympanic plexus and grooves a hiatus on anterior surface of the petrous part of the temporal bone and enters the middle cranial fossa, where it goes forwards in a groove on the bone above the lesser petrosal nerve and then passes below the fifth nerve

ganglion to reach foramen lacerum. It is joined by the deep petrosal nerve carrying postganglionic sympathetic axons from internal carotid artery's sympathetic plexus, to become the nerve of the pterygoid canal and proceeds for the PPG. Axons going in the nervus intermedius pass to lesser petrosal nerve in the region of the first genu ganglion. The greater and lesser petrosal nerves usually separate in the area medial to the first genu ganglion [31].The fossa in which first genu ganglion lies is covered by a very thin layer of bone which separates it from floor of middle cranial fossa. Dehiscences here are common and when present they are in direct contact with the meninges. An enlarged fossa on HRCT temporal bone suggests a diagnosis of geniculate ganglion fossa (GGF) fracture [32].The ampullated ends of the superior and the lateral semicircular canals should be identified during translabyrinthine approach because the facial nerve lies anteriorly and is at a risk of injury. The labyrinthine segment of facial nerve is likely to be injured in temporal bone fractures as it gets compressed by bony spicules.

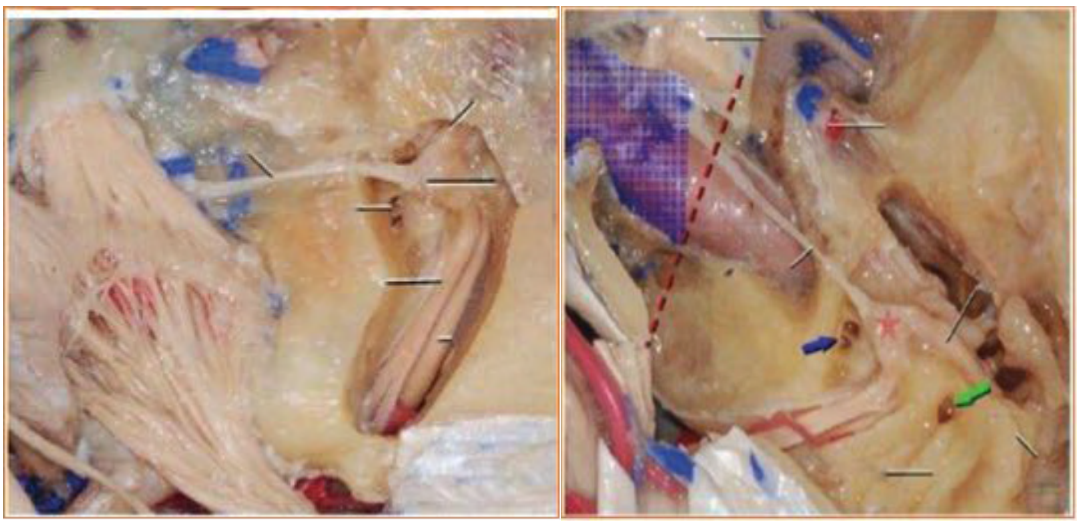


Figure 8 (a) The superior vestibular and the facial nerves has been exposed within the IAM along with the geniculate ganglion and cochlea.The bone over the tympanic segment of the facial nerve distal to the first genu ganglion has been removed. (b) The trigeminal nerve has been cut to expose the course of the GSPN and the petrous ICA. The interrupted red line corresponds to the posterolateral limit of the mandibular nerve and the GSPN passes 1.7 cm from this point to the first genu ganglion(marked with a red star).

The petrous segment of the ICA passes beneath the petrolingual ligament to enter cavernous sinus, as described by Tanriover et al [33].

TYMPANIC SEGMENT

The tympanic segment is 8–11 mm in length. It runs along the superior edge of medial wall of tympanic cavity, at right angles to long axis of the petrous temporal bone, taking a posterior and a downward inclining path. It extends laterally from the first genu ganglion to the second genu at the level of the pyramidal eminence [34].Proximally this segment passes just above and medial to posterior edge of the processes cochleariformis and the tensor tympani tendon. The cochleariform process

is a consistent and reliable landmark despite pathologies involving that area. Distally the nerve lies above the oval window niche, anteroinferior to the lateral semicircular canal prominence before taking a bend at the second genu to enter the styloid complex bone. Second genu hugs the inferior aspect of the lateral semicircular canal. The pyramidal eminence is a landmark for second genu. There are several small sinuses in retrotympanum around the facial canal: the sinus tympani lies anteromedial to the canal and facial and lateral tympanic sinuses lie posterolateral to the canal [35-37] .Via facial recess approach , the distal aspect of tympanic segment can be located . In the above approach ,the chorda tympani nerve and the fossa incudis can be used to safely identify the nerve. The second genu goes inferior and lateral to the horizontal semicircular canal. Behind the second genu is the posterior semicircular canal which demarcates the superior end of the retrofacial air cells, which delineates the medial aspect of the facial canal (**Figure 9**). Facial nerve is vulnerable during middle ear surgery at this level due to developmental dehiscences , especially around oval window niche [38-43] . In longitudinal fractures of the temporal bone , tympanic segment is vulnerable to traction injury.

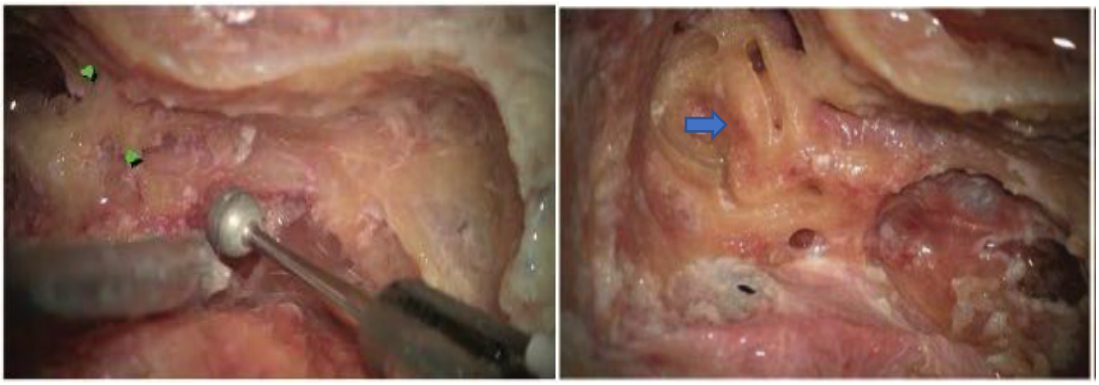


Figure 9 (a) The facial nerve has been exposed in the mastoid segment along an imaginary line from the digastric ridge(anterior part) to the short process of the incus tip in the mastoid antrum.(b) After skeletonizing the semi-circular canals, it has been opened to demonstrate the relationship of the posterior canal to the second genu (arrow).

MASTOID SEGMENT

The mastoid (vertical) segment is the longest measuring about 10–14 mm and is longest of all petrous segments. It runs downwards vertically in the posterior wall of the tympanic cavity and the anterior wall of the mastoid, distal to pyramidal process from the second genu to the stylomastoid foramen on the skull base. The digastric ridge is seen on well- pneumatized bones as the medial aspect of the mastoid tip which points to the lateral and inferior part of the mastoid segment. The path of mastoid segment is marked by a line drawn between the anterior end of the digastric ridge to the tip of short process of the incus .After removing the mastoid tip, the nerve is identified after skeletonizing the digastric ridge and outlining the inferior part of the vertical segment of the nerve.The nerve travels from the second genu to stylomastoid foramen from a posterior-medial to anterolateral direction. So expanding a posterior tympanotomy inferiorly would risk damage to nerve if

operating in the same plane. The facial nerve plane is always lateral to tympanic annulus , but it can cross it or lie medial in its lower half [44]. While elevating the tympanic annulus or lowering the mastoid ridge overlying the nerve, the nerve may be damaged. Failure to identify the mastoid antrum properly or being obscured by an overhanging tegmen is the common cause of iatrogenic injury. The positions of both the mastoid segment of facial nerve and tympanic annulus change during childhood [45] The mastoid segment of facial nerve gives three branches - the nerve to stapedius muscle, the chorda tympani and the sensory auricular branch.Given off behind the pyramidal eminence is the nerve to stapedius muscle which passes forwards through a small canal to supply the stapedius. Approximately 5 mm proximal to the stylomastoid foramen, the chorda tympani branches off and runs anterosuperiorly via posterior canaliculus to enter the tympanic cavity. It then travels across the Shrapnell's membrane between its mucous and fibrous layers, near to the posterior and anterior mucosal folds of the malleus, between the upper part of the handle or neck of the malleus medially and superior to the insertion of tensor tympani and lateral to the long process of the incus to reach the tympanic cavity in its anterior wall and it enters the anterior canaliculus (canal of Huguier) within the petrotympanic fissure's medial part [46] . After exiting the skull , it enters the infratemporal fossa where it is joined by the lingual nerve. A branch is given to the otic ganglion. Taste axons from anterior two thirds of the tongue and preganglionic parasympathetic axons are carried within chorda tympani nerve that will synapse on postganglionic neurons in the submandibular ganglion.The nerve serves as the lateral margin of the

facial recess in posterior tympanotomy. It is also used as the medial limit to lower the facial ridge when performing a modified radical mastoidectomy. Chorda tympani may arise extratemporally occasionally [47]. Thick fibrous tissue surrounds the facial nerve at the stylomastoid foramen. During anterior transposition of nerve, the segment is lifted en bloc together with fibres of posterior belly of digastric and slivers of bone around foramen. After leaving the stylomastoid foramen, the facial nerve lies inferior to the tympanic plate, lateral to styloid process base and the carotid sheath and posterior to the parotid gland. Posterior auricular nerve immediately branches off and runs upwards behind the ear between the parotid gland and the anterior border of sternocleidomastoid muscle and then in the notch between the external auditory meatus and the mastoid process to supply occipital belly of occipitofrontalis, auricularis superior and intrinsic auricular muscles. Posterior belly of digastric and the stylohyoid muscle are supplied by a second muscular branch.

EXTRACRANIAL SEGMENT

Extra cranial seventh nerve's main trunk enters the parotid gland high up on its posteromedial surface and runs forwards and downwards behind the mandibular ramus. Within the gland it divides, usually just behind and superficial to the retromandibular vein and external carotid artery (ECA) into an upper temporofacial trunk and a lower cervicofacial trunk. There is a varied relationship between the nerve and vein [48,49].The length of the facial nerve trunk from stylomastoid foramen to the intraparotid bifurcation is about 8 mm and 22 mm [50]. There are more axons in temporofacial trunk but fewer fascicles than cervicofacial trunk.

The trunks further divide to form a parotid plexus (pes anserinus) from which five main terminal branches arise namely temporal, zygomatic, buccal, marginal mandibular and cervical. These branches separate within the parotid substance and exit the gland medial to its anterior margin via its anteromedial surface. There are many individual variations in branching patterns and anastomoses in between branches, on the face as well as within parotid [51-55]. Plexiform pattern of nerves sustain surgical injury better than others, since significant facial weakness rarely occurs on account of accidental injury or division of a small plexiform nerve. Branching of the seventh nerve on the face is variable yet they maintain a relatively constant relationship to

soft tissue planes [56] . In parotidectomy, facial nerve injury is a significant complication [57] .To mitigate above issues intra-operative facial nerve monitoring can be done. A number of landmarks are used to identify the facial nerve trunk during surgical procedures. These include the posterior belly of digastric , tragal pointer, bony and cartilaginous junction of external ear canal, the tympanomastoid suture and the transverse process of the axis.1 cm medial and inferior to the tragal pointer (the medial aspect of the tragus that points inward, forming the anteroinferior wall of the external auditory meatus) is the main trunk of facial nerve. 6–8 mm below the inferior ‘drop off’ of the tympanomastoid fissure, the trunk can be identified or by dissecting along the posterior belly of the digastric muscle in a retrograde fashion towards mastoid process where it is inserted. In between the upper border of the muscle and the EAM, they form an angle which is bisected by the main trunk. A combination of these landmarks is the more consistent than a single landmark

[58,59].For identifying the marginal mandibular branch of the facial nerve , retromandibular vein is the commonly used landmark [60]. Other than retromandibular vein, facial vein can be followed proximally within the parotid tissue, where the nerve crosses lateral to the vein. 1 cm below the zygomatic arch, the buccal branch runs parallely and often along the inferior aspect of the parotid duct.There are many variations and anomalies in the course of facial nerve. Facial nerve is the nerve of the second branchial arch. Therefore one should be cautious while operating on patients with external ear malformations, congenital conductive ability to hear and craniofacial anomalies.

BLOOD SUPPLY OF FACIAL NERVE

Branches of the vertebrobasilar and ECA systems gives blood supply to these segments of facial nerve (**Figure 10**) [61,62]. Cisternal, meatal and labyrinthine segments are supplied by labyrinthine artery – internal auditory artery. It is a branch from AICA as it loops between the intracranial segments of the motor root , the nervus of wrisberg and the vestibulocochlear nerves, projecting into the IAM. Basilar, vertebral or superior cerebellar arteries can also supply it [63]. Petrosal branch of the middle meningeal artery supplies the greater petrosal nerve. The artery which usually passes through the bone enclosing the first genu ganglion and tympanic segment of facial nerve.Vessel and nerve are at risk when the dura is elevated from the MCF floor because artery can sometimes pass through the hiatus of greater petrosal nerve[64] .An anastomotic facial arch network formed by the middle meningeal artery branch – superficial petrosal artery and the stylomastoid branch derived from the

occipital or posterior auricular arteries, which courses the facial canal via the stylomastoid foramen supply the tympanic and mastoid segments. The stylomastoid artery collaterals supply the chorda tympani at the level of its origin. It is the lowest branch of stylomastoid artery to facial nerve. The bone marrow of the facial canal is supplied by facial arch anastomotic vessels with anterior and superior tympanic branches of first part of maxillary artery, inferior tympanic branch of the ascending pharyngeal artery and the posterior tympanic branch of the posterior auricular artery[65] From the stylomastoid foramen to the parotid gland , facial nerve is supplied by posterior auricular artery and occipital artery including its branches like stylomastoid artery. Collaterals of the superficial temporal, facial, transverse facial and maxillary arteries supply the temporofacial and cervicofacial branches that exit the parotid gland.Meatal arteries exclusively supply the labyrinthine portion and therefore more likely to suffer from ischaemic damage or hypoxia.

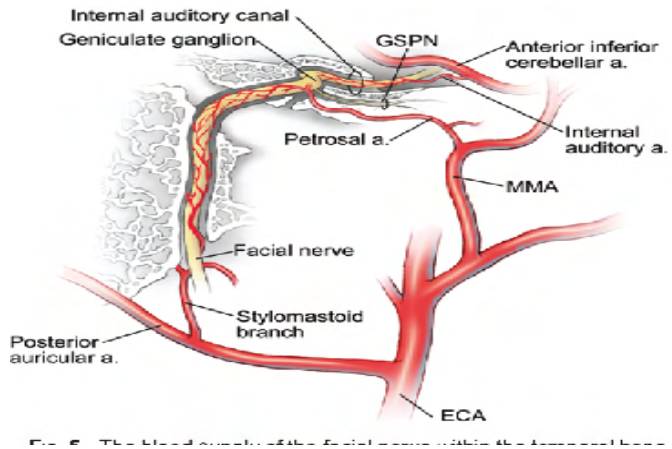


Figure – 10 showing blood supply to facial nerve

PATHOPHYSIOLOGY OF NERVE INJURY

Cranial and Spinal peripheral nerves can be injured in a number of ways. It can be classified as local which includes trauma both blunt or penetrating , compression like chronic traction or acute stretch , chemical or freeze injury. Systemic causes include autoimmune diseases ,diabetes mellitus or drug-induced. Ischaemia due to mild pressure with no structural changes may produce transient paraesthesia in which there is rapid recovery. In these cases surgical intervention is unwarranted. Immune-mediated attack can lead to prolonged ischaemia which may cause specific myelin loss (primary or segmental demyelination) without any loss of axonal integrity, which results in an increased refractory period of transmission and slowing of conduction and finally conduction block. If a bony spicule or haematoma is the causative agent , it is removed and remyelination occurs within 2–4 months with little residual functional loss for which surgical intervention is unwarranted. Injuries separating axons from neuronal cell bodies trigger a series of molecular and cellular events in and around the injury site neuron and target organs. Neurons will die when injury separates them from their nutrition which is the retrogradely transported neurotrophins. This is more evident if injury is close to neuronal cell body. Peripherally atrophy of chronically denervated muscles and sensory end organs may preclude their reinnervation. Augustus Waller termed 'Wallerian degeneration' which refers specifically to the degenerative process that takes place along a nerve distal to an injury. Acutely denervated distal stump facilitates regrowth of axons as it

provides a vascularised segment of longitudinally orientated, laminin- rich basal lamina tubes filled with axon-responsive Schwann cells (**Figure 11**).

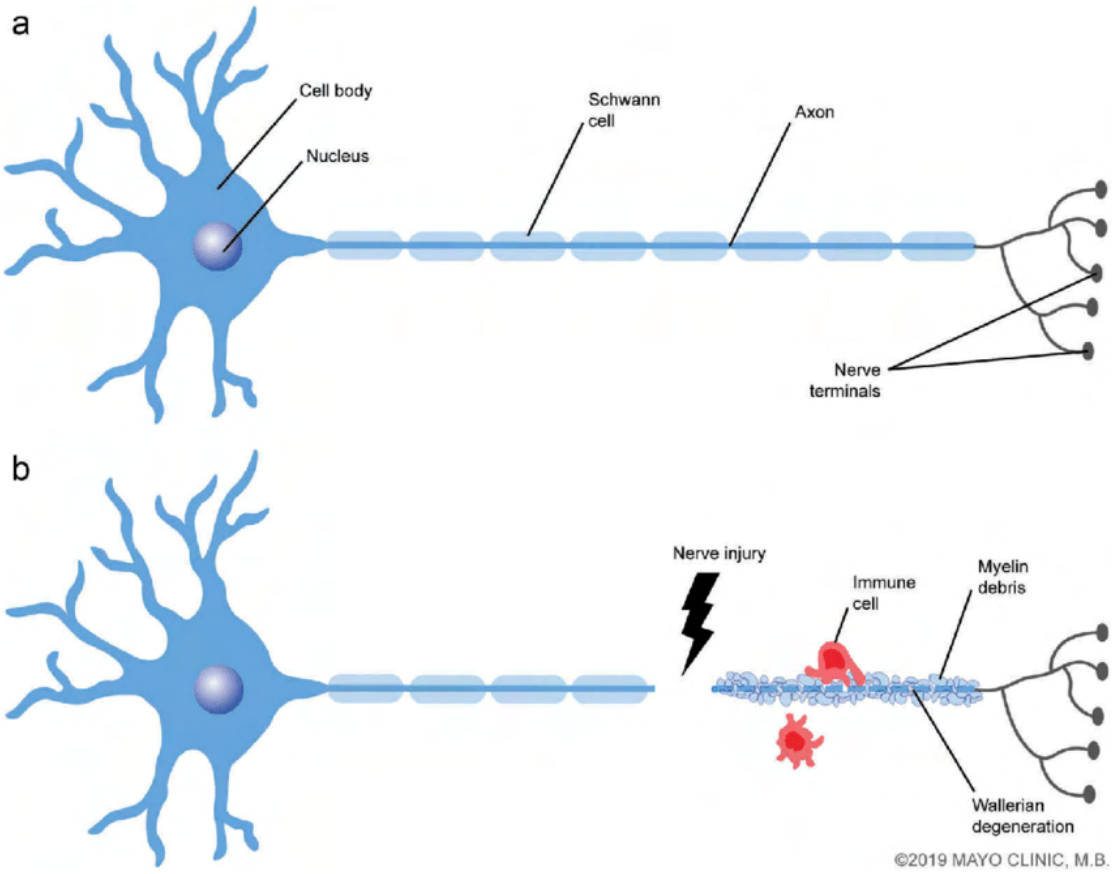


Figure 11 – a) Normal neuron (b) Injured neuron

Progressive endoneurial fibrosis and Schwann cell senescence interfere with axonal regrowth [68] .Early nerve repair gives an improved functional outcome [69,70]. Nerve repair can be done by direct coaptations of stumps by suturing or gluing , closing a long gap between stumps by either grafting a segment of nerve end-to-side or end to-end or by transfer of nerve. These methods are required to allow regrowing axons to interact with Schwann cells [71]. Sir Herbert Seddon introduced the terms neuropraxia, axonotmesis and neurotmesis to describe localised injuries to peripheral nerves [72].

Compression or stretch of nerve produces a physiological block of ion channel functions and axoplasmic transport along affected axons called neuropraxia. There is a temporary loss of function .Once the compressive agent is released it results in quick and full recovery of function and relief from pain. Prolonged ischaemia due to compression and traction produces demyelination. When a blunt injury to a nerve results in Wallerian degeneration with an intact connective tissue layer it is called Axonotmesis. Within few days of injury, conduction ceases throughout the distal extent of the nerve. Recovery usually progresses from proximal to distal at a rate of ~1 mm/day. When an axon reaches its target organ, subsequent reinnervation of the target is not a rule. Full functional recovery may not take place.

When a nerve is either completely cut or so badly disintegrated by an injury, which makes recovery difficult without some form of surgical intervention is called Neurotmesis. All the epi , peri , endoneurium of the nerve as well as its axons are divided at the site of injury. A wide inter-stump gap will be there.Following this type of injury, spontaneous axonal regeneration will be disordered and may not occur at all. Distance between stumps is a significant factor in recovery. Within days after denervation, changes in the damaged nerve fibre type and loss of striated muscle begin. 80% of volume of muscle may be lost by 4 months and irreversible muscle fibrosis with infiltration of fat occurs after 2 years [73-75] . Functional reinnervation is unlikely after 12–18 months. Muscles exhibit weakness, impaired coordination and reduced stamina even after repair of nerve. Cutaneous sensory receptors undergo degeneration post denervation and shall disappear after 3 years. Reinnervation post surgery tends to reverse these changes especially if the disruption occurs close to the

target organs and when the sensory nerves are involved. Sir Sydney Sunderland used Seddon's category of axonotmesis to describe progressively more invasive levels of injury to the endoneurial contents, perineurium and epineurium, respectively. Accurate prognosis of outcomes could be done in axonotmesis injuries while correlating with these new levels [76] . Sunderland's grade 1 which is equivalent to neuropraxia and grade 2 which is equivalent to axonal degeneration with intact endoneurium typically have a complete recovery.Recovery is partially seen in grade 3 injuries. Grade 4 and grade 5 which is equivalent to neurotmesis requires surgical intervention (**Figure 12**).

Table 1. Seddon and Sunderland Classification of Nerve Injury

Seddon	Sunderland	Injury	Spontaneous recovery	Nerve conduction study	Electromyography
Neurapraxia	Grade I	Focal segmental demyelination	Yes	Partial/complete conduction block proximally Preserved conduction block distally even after 2 weeks	Normal morphology and poor MUAP recruitment
Axonotmesis	Grade II	Damaged axon with intact endoneurium	Yes, slower than neurapraxia	Partial/complete conduction block proximally Preserved conduction block distally until Wallerian degeneration sets in	Abnormal activity
Axonotmesis	Grade III	Damaged axon and endoneurium with intact perineurium	Not very likely, surgical intervention may be needed		
Axonotmesis	Grade IV	Damaged axon, endoneurium, and perineurium with intact epineurium	Highly unlikely, surgical intervention is necessary		
Neurotmesis	Grade V	Complete nerve transection (disruption of myelin sheath, axon, endoneurium, perineurium and epineurium)	No, surgical intervention is necessary	Complete conduction block proximally and distally	Abnormal activity

Figure 12 – Sunderland's classification and Seddon's classification

Depending upon the site of tissue disruption, misdirection of regrowing axons into modality-inappropriate schwann tubes of the distal stump is inevitable [77,78].

Regrowing axons with their associated schwann cells can form a mechano sensitive neuroma at the proximal end of nerve stump or in-continuity with the distal stump. This phenomena compromises recovery. The distinction between conduction block and axon degeneration is important in prognosis.Other than sharp cut injuries, all other injuries or disorders causing facial nerve palsy will not have an all-or-none lesion. Mostly a mixture of all three types of injury will be seen.May and colleagues correlated Sunderland's classification with histological change, evoked electromyography, onset and final clinical recovery according to House and Brackmann's grading [79]. May suggested that the viral inflammatory immune disorders such as Bell's palsy and herpes zoster cephalicus show first three degrees of injury. Progress of facial nerve regeneration depends on various factors including the type and severity of injury and local microenvironment and patient general criteria such as age, nutritional status and comorbidities like diabetes mellitus.

FACIAL PALSY GRADING SYSTEMS

Clinical measurement of facial nerve function has been described by several systems. A six-point scale for reporting of outcomes and recovery from surgery for vestibular Schwannoma's was proposed by House in 1983. This system was called the House–Brackmann Staging System after some modifications. It has become the most widely used scheme and endorsed by the American Academy of Otolaryngology– Head and Neck Surgery [79]. In the House–Brackmann system, normal function is grade I , complete absence of motor function of facial nerve is grade VI and intermediate are grades II–V. (**Figure - 13**).But the House–Brackmann staging system have limitations in form of precision and inter-observer error. Two assessment domains are

used in grading facial nerve palsy. The first is gross observation and the second is movement of the forehead, eye and mouth.

The mixing of static and dynamic criteria lead to some disagreement between various observers. Other facial nerve palsy grading scores are Burres and Fisch [80] who described a method necessitating many measurements of movement in various parts of the face . Murty et al. [81] proposed and tested the Nottingham system of subjective estimation of movement at each of

several points on the face. This method correlated better with the House–Brackmann system than did the Burres– Fisch. It was easy to perform.Facial nerve dysfunction includes secondary effects like synkinesis, hemifacial spasm, contracture, crocodile tears, epiphora, dysgeusia, pain and hyperacusis. Both the Nottingham and Sunny brook scales have included these secondary effects in their overall score [82]. The ideal system of grading should be user-friendly, reliable and reproducible, should give information pertaining to pathophysiological events and correlate with self-assessment.

Grade	Description	Characteristics
I	Normal	Normal facial function in all areas
II	Mild dysfunction	Gross: slight weakness noticeable on close inspection; may have very slight synkinesis At rest: normal symmetry and tone Motion: Forehead – moderate-to-good function Eye – complete closure with minimum effort Mouth – slight asymmetry
III	Moderate dysfunction	Gross: obvious but not disfiguring difference between two sides; noticeable but not severe synkinesis, contracture, and/or hemifacial spasm At rest: normal symmetry and tone Motion: Forehead – slight-to-moderate movement Eye – complete closure with effort Mouth – slightly weak with maximum effort
IV	Moderately severe dysfunction	Gross: obvious weakness and/or disfiguring asymmetry At rest: normal symmetry and tone Motion: Forehead – none Eye – incomplete closure Mouth – asymmetric with maximum effort
V	Severe dysfunction	Gross: only barely perceptible motion At rest: asymmetry Motion: Forehead – none Eye – incomplete closure Mouth – slight movement
VI	Total paralysis	No movement

Figure – 13 House – Brackmann grading of LMN facial nerve palsy

TOPODIAGNOSTIC TESTS

Topodiagnostic tests refers to those tests that help to locate the anatomical level of injury.It is a test of function of nerve. These tests have a slightly limited correlation with the precise site of nerve damage.They do not have any prognostic value.

SCHIRMER'S TEST

Greater petrosal nerve is assessed here. Strips of paper are placed in the inferior conjunctival fornix for 5 minutes. Between both eyes , length of paper moistened is measured. Greater than 75% unilateral decrease in lacrimation or a bilateral decrease in lacrimation less than 10mm wetted for both sides at 5 minutes. Negative test is normal and positive test is abnormal.

Figure 14 – Schirmer's test

STAPEDIAL REFLEX

Nerve to stapedius is tested here. It is interpreted as present or absent.Loud sound 70 – 100 dB above the threshold of hearing of a particular ear causes bilateral contraction of stapedius muscle which is detected by tympanometry. Prognosis of facial nerve palsy can be assessed with this test.

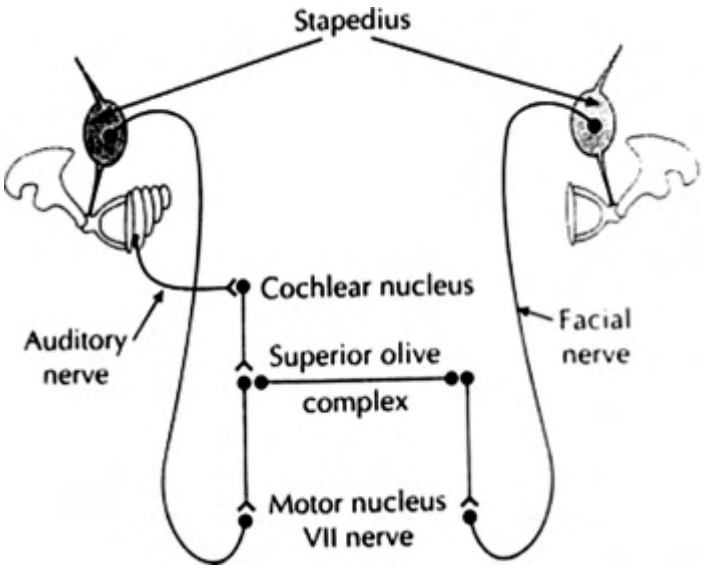


Figure 15 – Reflex arc of Stapedial reflex

ELECTROGUSTOMETRY AND SALIVARY FLOW TESTING

Chorda tympani nerve is tested here. In electrogustometry the tongue is stimulated electrically to produce a metallic taste and then both sides threshold are compared. In salivary flow testing , Wharton's ducts are cannulated and salivary flow is measured after gustatory stimulus. 25% reduction is abnormal.

ELECTROPHYSIOLOGICAL TESTS

Electrophysiological tests have been developed to provide prognostic information for recovery. The degree of dysfunction and assumed viability of the facial nerve can be determined. This will tell if any surgical intervention (facial reanimation) is needed or not. These tests are used in patients with complete paralysis and not on paresis patients where full functional recovery is possible. Facial nerve repair is contraindicated when the motor end plate muscle units are non functional. Reinnervation procedures are contraindicated when no muscle end plate activity is detected by electromyography as they will not be successful.Nerve excitability test (NET), and maximal stimulation test (MST), are rarely used nowadays. In both these tests normal side is compared to the palsied side and could be done at bedside using a Hilger Stimulator.Limitations of above are in terms of accuracy, reproducibility, inter-observer variation and prognostic value [83]. Nowadays in clinical practice are electroneuronography (ENoG) and electromyography (EMG) are only used.

ELECTRONEURONOGRAPHY (ENoG)

It is the most valuable prognostic indicator among electrophysiological tests. Indication for ENoG is acute onset complete facial nerve paralysis. Amplitude reduction percentage and rate of degeneration have been used as prognostic indicators of facial nerve recovery. In idiopathic paralysis, degeneration of > 90% within 14 days indicates a probable poor recovery in > 50% of cases [84] . Multiple sites of nerve involvement are seen in Ramsay Hunt syndrome and hence ENoG is not useful here. Fisch has suggested surgical intervention within 21 days in patients where there is

> 90% amplitude reduction by ENoG as a result of traumatic injury within 6 days of the injury [85,86] .

ELECTROMYOGRAPHY (EMG)

EMG records active motor unit potentials of the orbicularis oris and orbicularis oculi muscles during rest and dynamic action. EMG can be used to determine the volitional activity of nerve , fibrillatory potentials and polyphasic innervational potentials. Fibrillation potentials indicate Wallerian degeneration. Polyphasic innervation potentials indicate early signs of reinnervation .Following injury ,fibrillation potentials arise after 2–3 weeks and polyphasic reinnervation potentials may precede clinical signs of recovery by 6–12 weeks.In cases of acute onset complete facial nerve paralysis with > 90% degeneration on ENoG, the presence of active motor unit potentials indicates a favourable prognosis as some fibres are intact. Severe degeneration is unlikely to take place if some active motor units are recorded after 1 week of complete paralysis. EMG plays a key role in decision making regarding surgical intervention in long-standing paralysis. Presence of polyphasic motor unit potentials indicate regeneration and surgical intervention is unwarranted. Management is close follow up. Presence of fibrillation potentials indicate viable motor end plates but LMN denervation. Surgical exploration is warranted. Nerve continuity is achieved by either by end-to-end anastomosis, interposition grafting, re-routing or re-innervation techniques. No electrical output on EMG indicates that muscle is fibrosed. In this case facial reanimation is indicated.

LIMITATIONS OF ELECTROPHYSIOLOGICAL TESTING

- Normal or neuropraxic fibres are only stimulated by electrical impulse. Axonotmesis or neurotmesis injuries cannot be distinguished and both these have different prognosis.
- No information is provided in cases of incomplete facial paralysis.
- It fails to provide information within the first 72 hours post paralysis of nerve.

MANAGEMENT OF FACIAL NERVE PALSY

EYE CARE

In cases where eye closure is impaired , immediate corneal protection with lubrication and patching should be done. Large-lens sunglasses protects the eye from debris and dust on a windy day. During the daytime, eye drops have to be applied to corneal surface as frequently as possible whilst at night ointment is preferred. After the application of the ointment at night , eye should be taped closed. Over long period of time, there is unopposed action of levator palpebrae superioris which is supplied by the oculomotor nerve that results in upper eyelid shortening. Closing the eye manually by the patient using his/her finger as well as stretching the upper lid prevents this shortening. In long-standing cases of impaired eye closure, surgical weighting or levator lengthening procedures are done [87,88].

IDIOPATHIC/BELL'S PALSY

In 1959, Taverner [89] described the minimum diagnostic criteria for bell's palsy which included paralysis or paresis involving one side of the face including all muscle groups; acute onset; no signs of central nervous system disease; no signs of ear or Cerebellopontine angle disease. Patients present with periauricular pain , general malaise and other prodromal illness symptoms. They rapidly develop complete lower motor neuron facial weakness in 72 hours. The annual incidence of bell's palsy is ranging between 20 and 32.7 per 100,000 [90,91].The incidence peaks between the ages of 15 and 45 years. It is seen more in women younger than 20 years and in men older than 40 years. Bell's palsy is more common 3rd trimester of pregnancy. In later stages of pregnancy changes in fluid balance with increased ECF leads to oedema of the nerve and compression within the facial canal [92] .The aetiology of idiopathic palsy is said to be an infectious origin, which triggers an immunologic response that damages the facial nerve. Herpes simplex virus type 1 (HSV-1), herpes simplex virus type 2 (HSV-2), human herpes virus, varicella zoster virus (VZV), influenza B, adenovirus, Coxsackie virus and Epstein–Barr virus (EBV) [93]. Poor prognosis is seen in complete paralysis at onset , incomplete paralysis with late onset of recovery, old age, dry eye, decreased taste, absent stapedius reflex and post-auricular pain [94]. Normal function is regained in 3 months in most patients. After a period of 6 months , no further recovery is seen . Prednisone 1 mg/kg/day for 10 days and oral acyclovir (400 mg five times daily) for 10 days is a commonly agreed treatment regimen.

RAMSAY HUNT SYNDROME/ VZV INFECTION

Ramsay Hunt syndrome is a lower motor neuron facial nerve palsy characterised by an erythematous vesicular rash on the ear or in the mouth (**Figure 16**).It is due to reactivation of the latent VZV virus in the geniculate ganglion. Excruciating pain may precede the onset of palsy. Diagnosis is clinical [94]. Ramsay Hunt Syndrome prognosis is worse than idiopathic facial palsy. The recommended regime is prednisone 1 mg/kg/day for 5 days followed by a tapering 10-day dose, as well as oral acyclovir (800 mg five times daily) within 3 days of onset [134] . Robillard et al. have shown that patients with Ramsay Hunt syndrome treated with combination of steroids and acyclovir have reduced ear pain, vertigo and post-herpetic neuralgia [95].

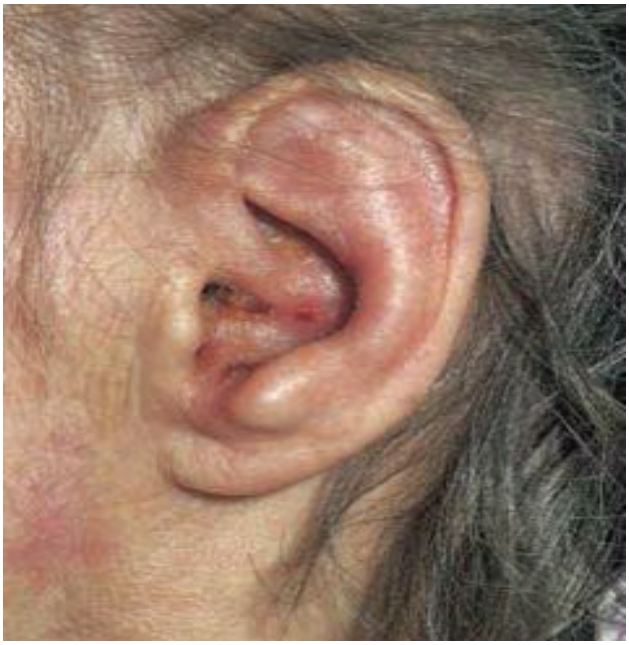


Figure 16- Vesicular eruption in Ramsay Hunt syndrome

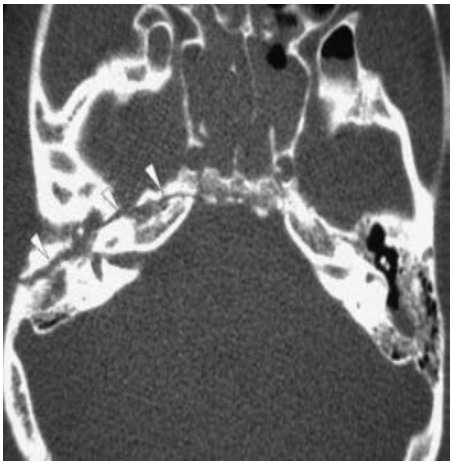
OTHER VIRAL CAUSES

Guillain–Barre syndrome (acute inflammatory demyelinating polyradiculoneuropathy) presents after an upper respiratory infection. Time period is usually 2-3 weeks.It is caused by an abnormal T-cell response against components of myelin. The salient features are symmetrically ascending muscular weakness and autonomic system involvement. Facial nerve paralysis is bilateral. Treatment is mainly focused on respiratory support and passive physiotherapy. There is no role for steroids or worse even may even delay recovery. The most effective form of treatment is intravenous immunoglobulin infusion [96] .Human immunodeficiency virus (HIV) infection can cause facial nerve palsy and can present at any stage of the disease. It infects the facial nerve or geniculate ganglion and sets off an inflammatory demyelinating neuropathy or secondary infection triggered by VZV, HSV or EBV [97]

FRACTURES OF THE TEMPORAL BONE

Temporal bone fractures are divided into longitudinal and transverse fractures. Incidence of complete paralysis is 20% in longitudinal fractures and usually involves the perigeniculate area.Incidence of facial nerve paralysis is 50% in transverse fractures and involves the labyrinthine and mastoid segments commonly. (**Figures 17** and **18**).Longitudinal fractures are more common than transverse fractures and is associated with better clinical outcome. Otic capsule involvement is characterised with increased incidence of complications [98]. There are approximately twice as likely to develop facial nerve paralysis, four times more likely to have a CSF leak and seven times more likely to sustain a profound hearing loss in an otic capsule

involvement. The use of HRCT of temporal bone and ENoG are important in decision-making regarding surgery. If surgical exploration is warranted, the aim is to to decompress the nerve ,remove bony spicules that impinge the nerve and re-establish continuity in case of complete cut injury.



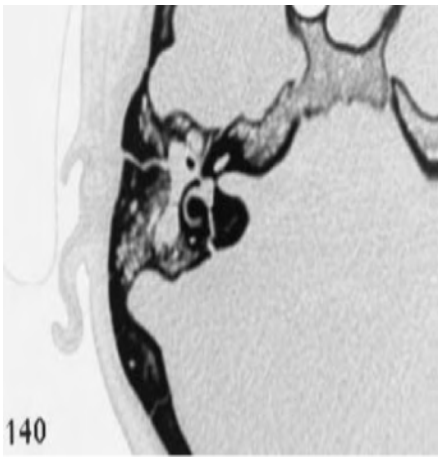


Figure 17 **Figure 18**

Figure -17 and 18 showing CT appearance in transverse and longitudinal fracture of temporal bone respectively.

Management in the early post-injury stage

Patients with normal facial nerve function or those with acute onset incomplete palsy without signs of worsening have an excellent prognosis and does not require any surgical intervention [99]. In cases of acute complete paralysis ,if ENoG shows more than 90% denervation within 6 days of onset of palsy, surgical intervention is warranted [100] . Lambert and Brackmann [101] have emphasised that surgery is indicated on the basis of ENoG findings and it can be done on unconscious patients as well.

Management in the late post-injury stage

The decision to treat is based on CT findings and electromyographic results. EMG findings suggesting long-term denervation, static or dynamic facial reanimation surgeries are done. A middle cranial fossa approach is preferred for longitudinal fractures in which preservation of hearing is possible. A translabyrinthine approach is used in severe sensorineural hearing loss as it is a less morbid surgery. Decompression, resection anastomosis, end to end anastomosis, interposition nerve graft from the great auricular or sural nerve should be used to repair the nerve.

IATROGENIC INJURY

The incidence of facial nerve palsy is between 0.6% and 3.6% in middle ear and mastoid surgery [102] . The tympanic segment distal end including the second genu, followed by the mastoid segment is the common site of injury [103] . If Intra-operative identification of facial nerve injury is done, exploration with decompression of proximal and distal segments of the facial nerve should be done. If facial palsy is observed immediately post op, a few hours of observation will allow for local anaesthetic-induced weakness to clear off. Tight mastoid dressing over an exposed facial nerve should be avoided . In incomplete paralysis, the patient is put on oral steroids and observed for progression of palsy. If progressing to full paralysis ,then exploration is warranted.Forceps delivery is associated with a slightly increased incidence of paralysis in neonates. The superficial part of the facial nerve at the stylomastoid foramen and the soft temporal bone makes it prone to injury by forceps assisted delivery, compression against the baby’s shoulder or maternal sacral

prominence in cephalopelvic disproportion [104] . Battle's sign and haemotympanum suggest trauma. EMG responses are seen at birth with progressive decrease in amplitude over time, as opposed to inherited disorders in which they are absent right from birth. Greater than 90% complete recovery is seen in this facial nerve injury.

OTITIS MEDIA

Facial nerve paralysis is seen in both acute otitis media and chronic otitis media. It is due to involvement of the facial nerve by infection spreading through Fallopian canal crevisces or physiologic canaliculi meant for neuro - vascular connections, osteitis of Fallopian canal, inflammatory oedema leading to compression and secondary vasa nervosum thrombosis with subsequent ischaemia and infarction of the seventh nerve. Sometimes demyelination of the facial nerve can be caused by bacterial toxins. The mainstay of treatment for above condition is antibiotic therapy. When spontaneous resolution by perforation is not there then tympanostomy tubes can be placed to relieve pressure. In chronic otitis media, mastoid exploration with facia nerve decompression is done.

MALIGNANT OTITIS EXTERNA

Malignant otitis externa is an invasive *Pseudomonas* or *Aspergillus* infection of the ear canal. It mainly affects the elderly, insulin dependent and poorly controlled diabetics. Facial palsy in malignant otitis externa indicates spreading infection and invasion through the fascial planes ,bony cartilaginous junction of EAC and the fissures of Santorini and posteriorly to the exit of facial nerve at stylomastoid

foramen. Ciprofloxacin (750 mg orally twice per day) is the antibiotic of choice and it is given for a minimum of 6–8 weeks [105] . Antipseudomonal β-lactam agents (ceftazidime, piperacillin, meropenem) with or without an aminoglycoside can be used in case of ciprofloxacin resistance . *Aspergillus* infection need systemic antifungal therapy. Surgical management is currently not done. Only diagnostic biopsy is done to exclude malignancy.

Venugopal et al [105] found that trauma (41.7%) contributed to the major proportion of cases. It may be Iatrogenic (20%) or Non-Iatrogenic (21.7%). A large number of road traffic accidents constituted 21.7% cases of facial palsy were accounted for trauma of which 16.7% showed fracture of temporal bone (longitudinal) and 5% showing blunt trauma to temporal region . The second most common aetiology was Bell's palsy (23.3%). Incidence of facial palsy secondary to Chronic otitis media is 67% of which one case was tuberculous otits media. Cholesteatoma was the most common infective cause . Malignant otitis externa was cause of facial palsy in diabetic patients in whom there was poor glycemic control. 5% of patients had Ramsay Hunt syndrome. Tumour was the cause of facial palsy in 13.3% of patients. Male preponderance of tumour to facial palsy was 83%. Majority of patients in their study was between 31 – 40 years. A slight male predominance was seen. Road traffic accident was common in young males and infectious pathologies were more in woman. Majority reported with House Brackmann grade III palsy (30%), followed by grade IV palsy (25%). Topodiagnostic tests showed Bell's palsy involving supragenicular region (44.4%). Iatrogenic trauma was common in infrastapedial region . Temporal bone fractures had supragenicular lesion.

Patel and Poole [106] in 1998 found out that high incidence of facial palsy was post superficial parotidectomy for benign parotid tumour.

Eftekharian A [107] in 2005 found out that while removing the cholesteatoma in epitympanum and supratubal recess, the 1st genu ganglion and proximal part of seventh nerve are at risk of damage.

Guerrisi [108] in 1997 found out that 17% of facial palsy was secondary to blunt trauma.

Nageris *et al* [109] described late onset seventh nerve palsy in fractures of temporal bone.

Altuntas *et al* [110] in 1998, found out that 1.7% incidence of facial palsy secondary to Chronic Suppurative Otitis Media in which 70% had Cholesteatoma.

Kirsch *et al.* [111] in 1996 found out that there was an increased incidence of Tuberculous Otitis Media in United states.

Yeo SW *et al.*[112] in 2007 found out complete revival only in 50% of Ramsay Hunt Syndrome. Bell's palsy recovered spontaneously in 96% patients.

Rosenberg [113] in 2000 proposed that Schwannoma of facial nerve caused facial palsy only in higher stages of disease that is in its advanced stage.

Ayala *et al.*[114] in 2007 found a male predominance for 60% of facial palsy patients . Road traffic accident was common in young men and infectious palsies were more in women.

Chow *et al* [115] in 2002 proposed that Electroneuronography helps to decide on what decision has to be taken after 48 hours of the onset of facial palsy symptoms.

Manish Munjal et al [116] conducted a retrospective study of 500 cases of head injury . 48 patients of facial palsy were taken to study the role of topodiagnostic tests in

localising the lesion site. In 48 patients of facial palsy, taste sensation was decreased in 67% (21 cases); acoustic reflex was absent in 86.8% (33 cases) and Schirmer's test showed decreased lacrimation in 29.1% (14 cases). He also concluded that topodiagnostic tests do not always localise the lesion site in head injury.

Dobie [117] in 1986, found out that stapedial reflex test was not useful in the majority of cases because temporal bone fractures more commonly produced a severe sensorineural hearing loss or conductive loss.

Tschiassny et al [118] popularised lesion site testing, of which Schirmer's test was thought to be the most accurate topodiagnostic test.

Lambert and Brackmann [119] in their review of 26 cases of longitudinal fractures of temporal bone, they used Schirmer's test as the main indicator of the site of facial nerve injury.

Gantz et al [120] demonstrated that Schirmer's test predicted the site of injury in 61% of the patients. Therefore these tests do not appear useful in deciding the injury site preoperatively.

Renou et al [121] using Schirmer's test and stapedial reflex test , carried out a topodiagnostic analysis of Bell's palsy in 45 cases. They used it as a guide for possible choice of surgical decompression of the facial nerve. They found geniculate or supragenicular lesion in 62% of cases.

Ide, Morimitsu et al [122] compared the stapedial reflex of 30 patients with peripheral facial nerve paralysis with the results of facial paralysis scores. The stapedial reflex test at 500 Hz for opposite ear stimulation was found to be correct for evaluating the degree of facial paralysis. With 500 Hz or 1,000 Hz opposite ear stimulation, the

stapedial reflex test seemed to be useful for predicting the prognosis for facial nerve paralysis. In these stimulations, positive reflex within 2 weeks showed full recovery within 12 weeks and positive reflex within 4 weeks showed recovery within 24 weeks.

Bharathi M et al [123] did a study in which 101 patients were analysed of which 25.7% were in third decade of age; 55.4% were males, and both right and left sides of the face are involved equally. Most patients (50.5%) had a history of post aural pain at presentation.Topodiagnostic tests showed majority of Bell's palsy patients had geniculate or supragenicular lesions (67.3%) in the study. 20.8% had lesion above the nerve to stapedius, and 11.9% had lesion below the nerve to stapedius.50.4% of patients had a House-Brackmann (HB) facial nerve grade IV while they presented to study.

In 1977, Chandler[124] reported a 32% incidence of facial nerve paralysis in patients suffering from malignant otitis externa.

The incidence of facial nerve paralysis appears to have decreased with the development of more effective medical therapy as shown by Franco-Vidal et al [125] who reported a 20% incidence of facial nerve paralysis in 46 treated patients.

In 1985, **Corey et al**[126] reviewed the findings of 83 patients with MOE reported in literature. Fifty-eight had facial paralysis (70%). The paralysis presented late in the course of the disease after 2 months to be precise and indicated progression of the disease process. However, outcome did not worsen.

Ben I. Nageris [127] studied 48 patients with Malignant otitis externa which included 31 men and 17 women . Facial paralysis was detected in 8 patients . Mean age at diagnosis was 78 years . Diabetes was the most common comorbidity.Diabetes and chronic renal failure were seen in older age group. The duration of patient complaints was 65 days.Clinical examination revealed edema of the ear canal in 7 patients (88%) and granulation tissue in 6 patients (75%). Average erythrocyte sedimentation rate was 77 mm/h .Bone scan findings were positive in all patients with facial nerve involvement There was a positive correlation between presence of ear canal edema and a positive bone scan finding .Computed tomography was performed in 7 patients in the facial palsy patients. Evidence of canal bone destruction and of mastoid, temporomandibular joint, parapharyngeal, or nasopharyngeal involvement was found in several patients. Only mastoid involvement was significantly more common in the patients with facial paralysis. Bone involvement was also significantly correlated with a finding of granulation tissue.

AIMS AND OBJECTIVES :

To know the prevalence and ascertain an etiology of peripheral facial nerve palsy in patients attending our hospital.

JUSTIFICATION FOR STUDY :

- Facial nerve is the seventh cranial nerve having important functions, and hence its paralysis can lead to a great deal of mechanical impairment and emotional embarrassment.
- Etiopathogenesis of lower motor neuron facial palsy is still a diagnostic challenge and the literature has shown varying results pertaining to the same.
- This study was designed to sketch out the prevalence of disease causation and the profile of peripheral facial palsy patients presenting to our hospital at Coimbatore.
- By doing so, disease burden to society , finding etiology earlier helps in early diagnosis, treatment and prognosis of the disease can be done promptly.

MATERIALS AND METHODS

STUDY TYPE : Cross - sectional study

STUDY PERIOD : January 2020 – June 2021

STUDY SETTINGS : Department of ENT

Department of General Medicine

Department of Neurology

STUDY POPULATION: All patients attending PSGIMSR with lower motor neuron facial weakness/asymmetry

SAMPLE SIZE : 40

INCLUSION CRITERIA: All patients with peripheral facial nerve paralysis in the age of 3 years to 80 years.

EXCLUSION CRITERIA: Infants

Toddler upto age of 3 years

Adults above 80 years.

METHODOLOGY:

After obtaining ethical committee approval, this study will be carried out in patients with lower motor neuron facial nerve palsy, attending our hospital PSG Institute of medical sciences and research , Coimbatore, during the period January 2020 to June 2021. After obtaining the patient information and filling it systematically, careful history will be taken regarding commencement, causation, duration and progression. Functions of facial nerve will be evaluated by Topodiagnostic level of voluntary movement present in facial musculature during clinical testing. Entire ENT evaluation including neurological examination will be done and further assessment included Schirmer's test, acoustic reflex, subjective taste sensation tests , routine laboratory investigations and audiological evaluation . We will undertake comprehensive radiology including High-Resolution Computed Tomography (HRCT) of temporal bone and Magnetic Resonance Imaging (MRI) of temporal bone if indicated. The ethical considerations are strictly followed and data obtained from the study will be kept confidential and disclosed only for scientific purpose(s).

FLOWCHART

All patients with peripheral facial nerve paralysis attending the hospital satisfying inclusion and exclusion criteria.

↓

Informed and Written consent shall be taken

↓

History and physical examination as in variables recorded

↓

Patient fit for study

↓

Data will be recorded

↓

Clinical examination , Topodiagnostic tests, Blood tests and Radiological Examination if indicated will be done.

RESULTS :

In this study we evaluated 40 patients presented with peripheral facial nerve palsy between the age group of 3 and 80 years and patients who were not willing were excluded.

All the statistical data entered in MICROSOFT EXCEL SHEET for master chart preparation.

The collected data were analysed with IBM SPSS Statistics for Windows, Version 23.0. (Armonk , NY : IBM Corp) . To describe the data descriptive statistics frequency analysis and percentage analysis were used for continuous variables.

Pie and bar graph have been used for pictographic representation .

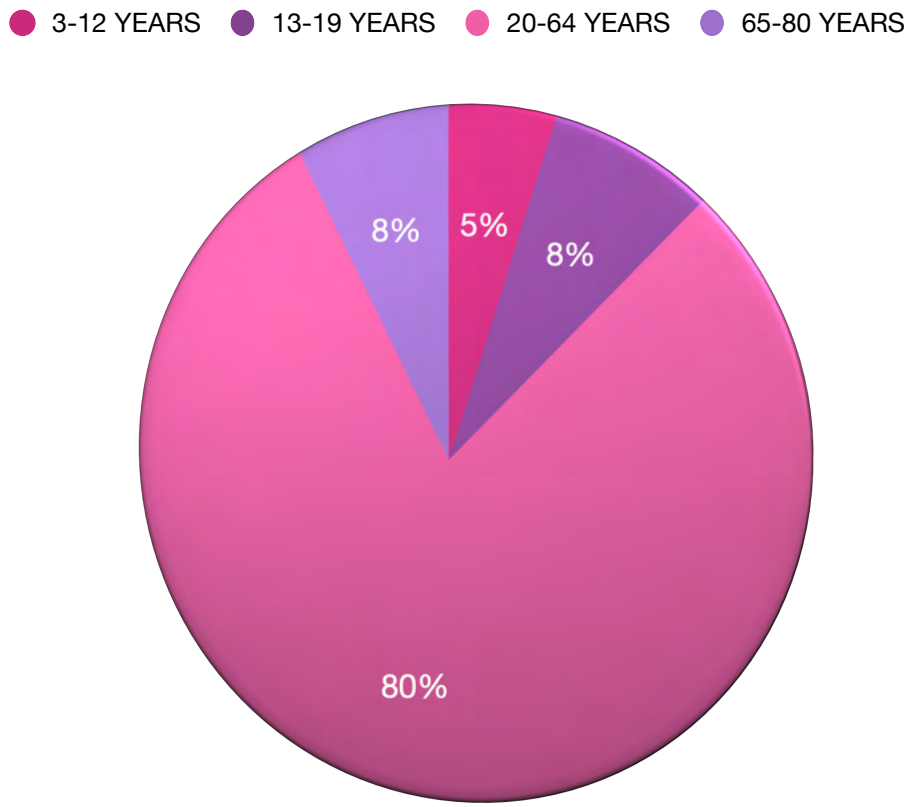
Values are expressed in numerical and percentage as %.

In the study process we found that ,

TABLE 1 - AGE INCIDENCE (N=40)

S.NO	AGE	FREQUENCY	PROPOTION
1	3-12 YEARS	2	5%
2	13-19 YEARS	3	8%
3	20-64 YEARS	32	80%
4	65-80 YEARS	3	8%

In our study we examined 5% of patients in 3-12 years , 8 % of patients between 13-19 years , 80% of patients between 20-64 years and 8 % of patients between 65-80 years.

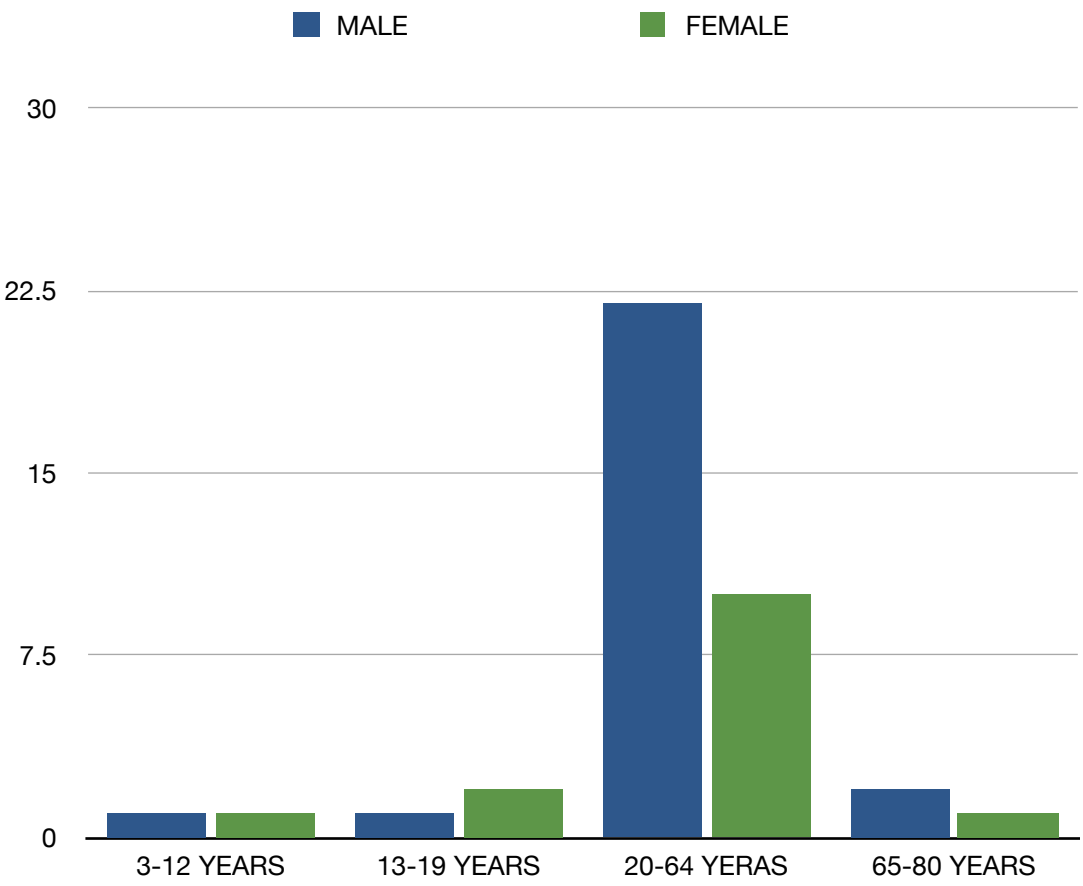


GRAPH 1 : AGE INCIDENCE

TABLE 2 - SEX INCIDENCE (N=40)

	3-12 YEARS	13-19 YEARS	20-64 YEARS	65-80 YEARS	PROPOTION
MALE	1	1	22	2	65%
FEMALE	1	2	10	1	35%

In our study out of 40 patients 65% were male and 35% were female and among then number of male patients were 1,1,22,2 in the age group between 3-12 years, 13-19 years, 20-64 years, 65-80 years respectively and female patients were 1,2,10,1 in the age group between 3-12 years, 13-19 years, 20-64 years, 65-80 years respectively.

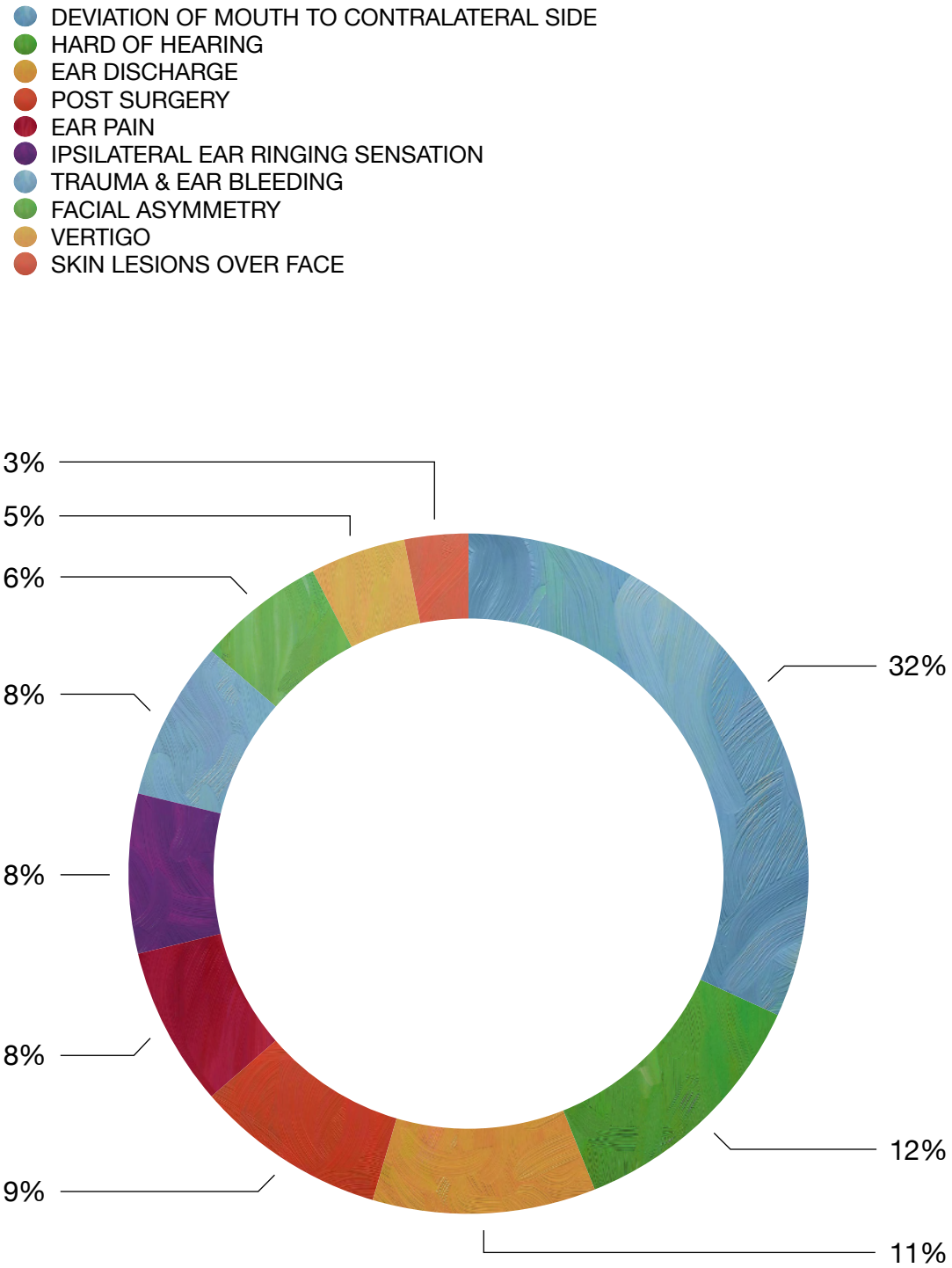


GRAPH 2 : SEX INCIDENCE

TABLE 3 - PRESENTING COMPLAINTS

COMPLAINTS	FREQUENCY	PROPOTION
DEVIATION OF MOUTH TO CONTRALATERAL SIDE	21	52.5%
HARD OF HEARING	8	20%
EAR DISCHARGE	7	17.5%
POST SURGERY	6	15%
EAR PAIN	5	12.5%
IPSILATERAL EAR RINGING SENSATION	5	12.5%
TRAUMA & EAR BLEEDING	5	12.5%
FACIAL ASYMMETRY	4	10%
VERTIGO	3	7.5%
SKIN LESIONS OVER FACE	2	5%

In our study 52.5% of patients presented with deviation of mouth to contralateral side of face and 20% with hard of hearing on same side and 17.5% and 10% of patients presented with ear discharge and 15% patients presented with peripheral facial nerve palsy post surgery . 12.5% patients presented with facial palsy following trauma which was associated with ear bleeding. 7.5% of the study patients presented with vertigo and vesicle like skin lesion were present in 5% of our study population.



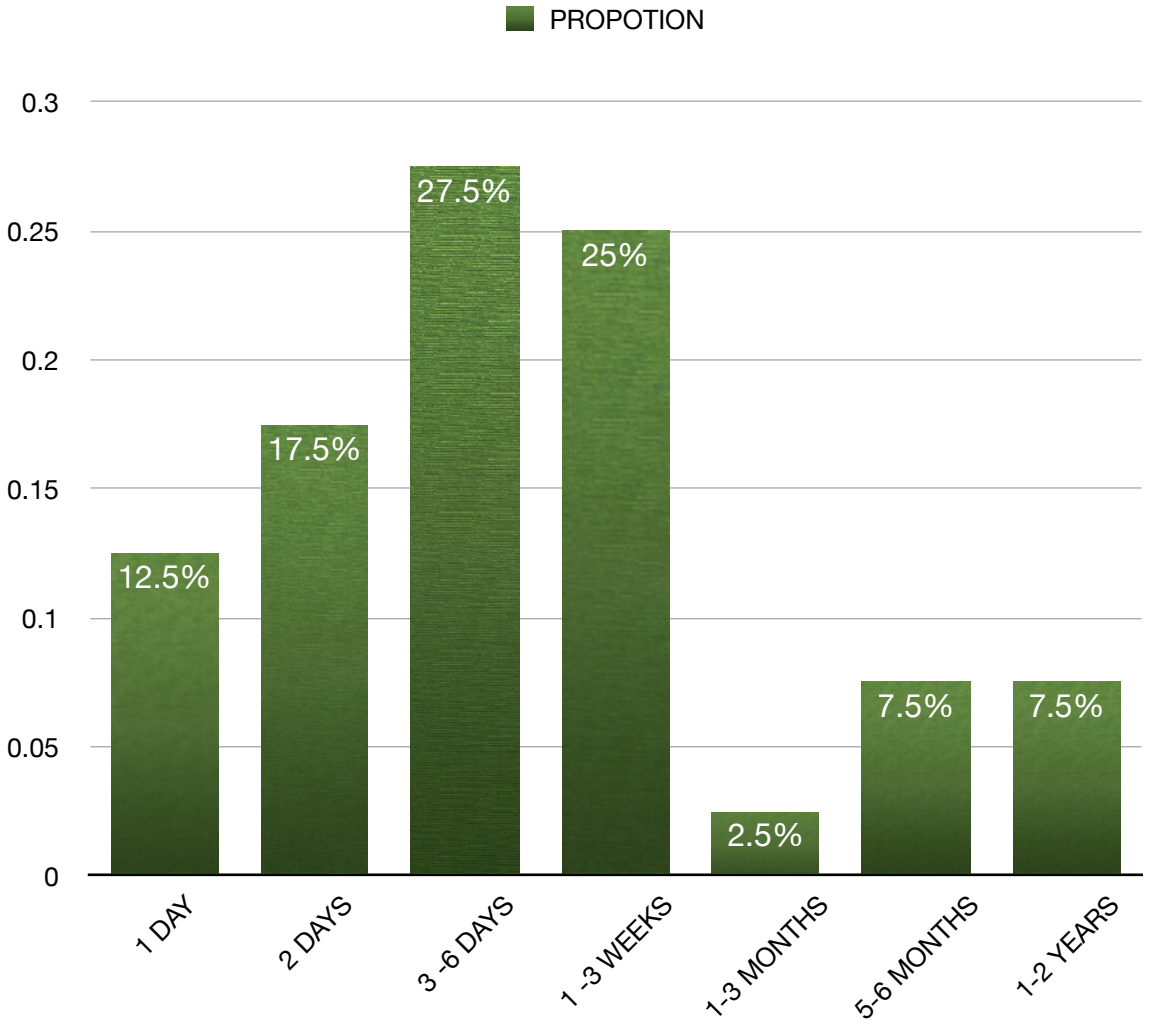
GRAPH 3 : PRESENTING COMPLAINTS

TABLE 4 - DURATION AT THE TIME OF PRESENTATION

DURATION AT THE TIME OF PRESENTATION	FREQUENCY	PROPOTION
1 DAY	5	12.5%
2 DAYS	7	17.5%
3 -6 DAYS	11	27.5%
1 -3 WEEKS	10	25%
1-3 MONTHS	1	2.5%
5-6 MONTHS	3	7.5%
1-2 YEARS	3	7.5%

In our study 27.5 % of the study population presented during 3-6 days of duration and 12.5 % presented on the very first day and 25% presented with duration of 1-3 weeks and 10 % presented between 1-6 months and 7.5 % presented during 1-2 years duration.

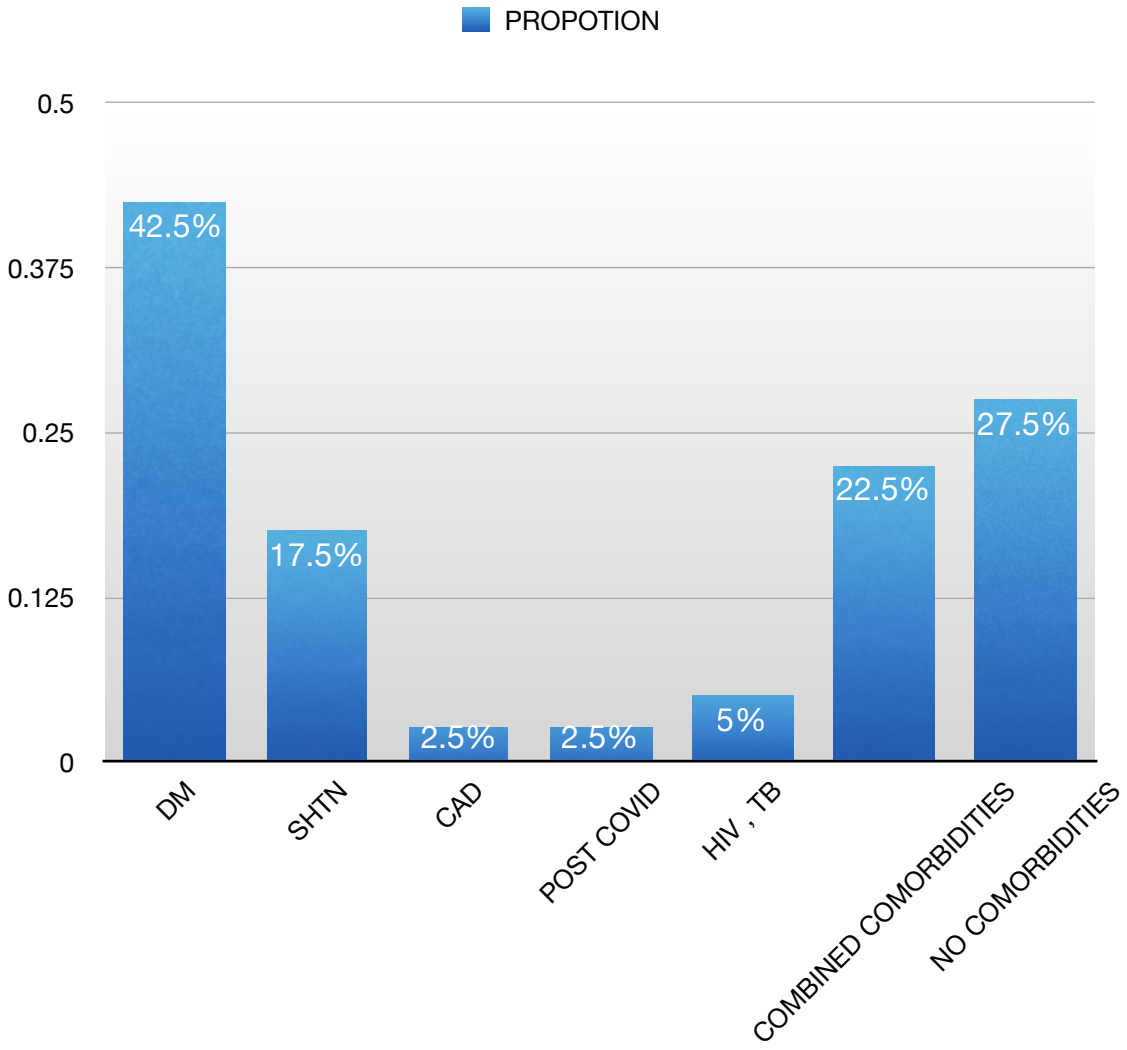
Most common duration at the time presentation was between 3-6 days .



GRAPH 4 : DURATION AT THE TIME OF PRESENTATION

TABLE 5 - COMORBIDITIES

COMORBIDITIES	FREQUENCY	PROPOTION
DM	17	42.5%
SHTN	7	17.5%
CAD	1	2.5%
CVA	1	2.5%
POST COVID	1	2.5%
HIV , TB	2	5%
COMBINED COMORBIDITIES	9	22.5%
NO COMORBIDITIES	11	27.5%



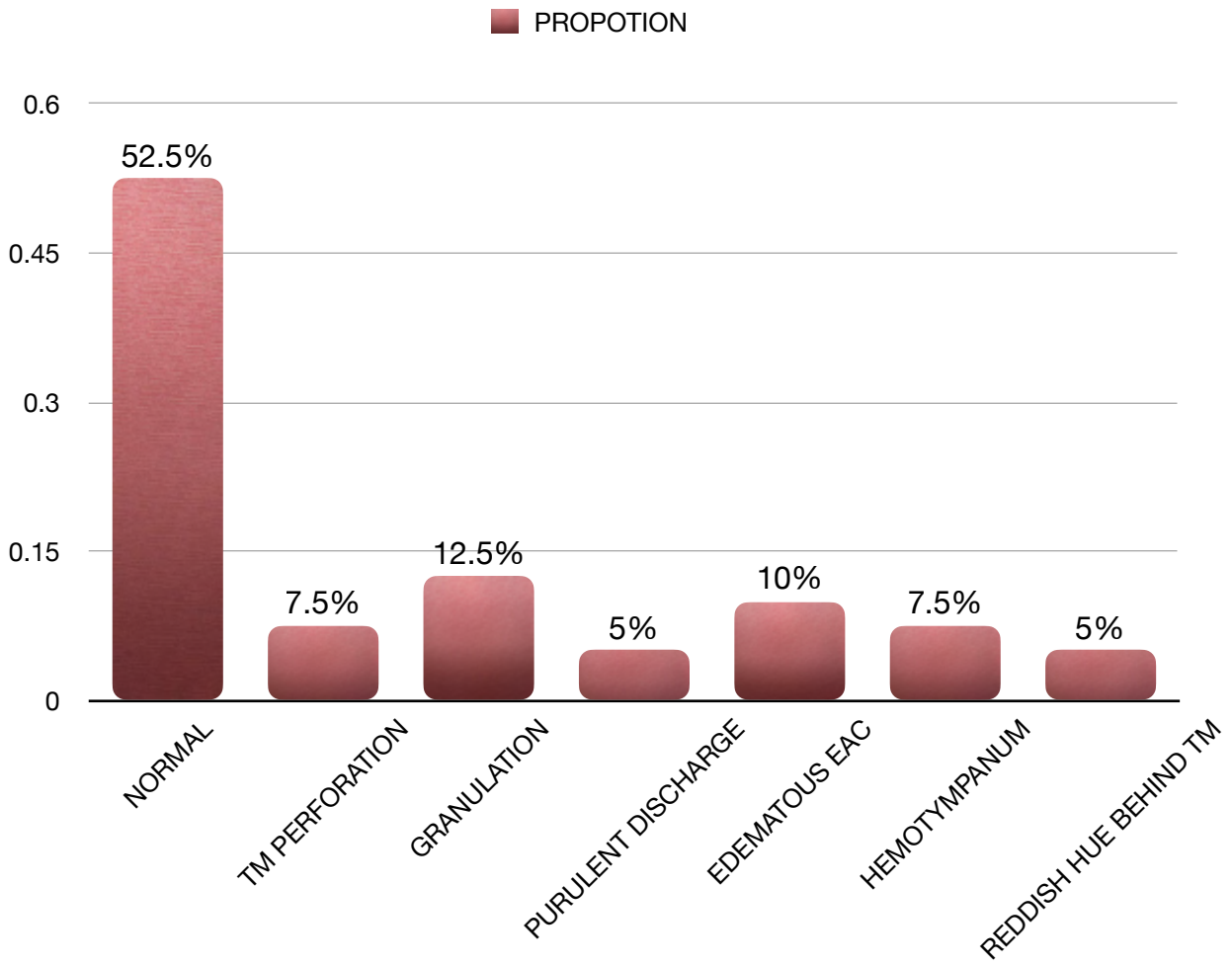
GRAPH 5 : COMORBIDITIES

In our study 42.5% of our study population were found to be diabetic on medications and among them 17.5% had associated systemic hypertension , and 2.5 % had CVA, CAD, 5% had HIV and TB which included 22.5% of combined comorbidities among 40 patients , and 27.5% had no comorbidities . And 1 patient was found to had post covid mucormycosis (5%) which involved multiple cranial nerve palsy .

TABLE 6 - CLINICAL EAR FINDING

EAR CLINICAL FINDING	FREQUENCY	PROPOTION
NORMAL	21	52.5%
TM PERFORATION	3	7.5%
GRANULATION	5	12.5%
PURULENT DISCHARGE	2	5%
EDEMATOUS EAC	4	10%
HEMOTYMPANUM	3	7.5%
REDDISH HUE BEHIND TM	2	5%

In our study 52.5% patients had a normal tympanic membrane on clinical examination, 12.5% had granulation and 10 % had oedematous EAC , 7.5 % had TM perforation and 5% had purulent ear discharge on ipsilateral ear following chronic otitis media and 7.5% had haemotympanum following trauma and 5% reddish hue behind TM due to glomus jugulare.



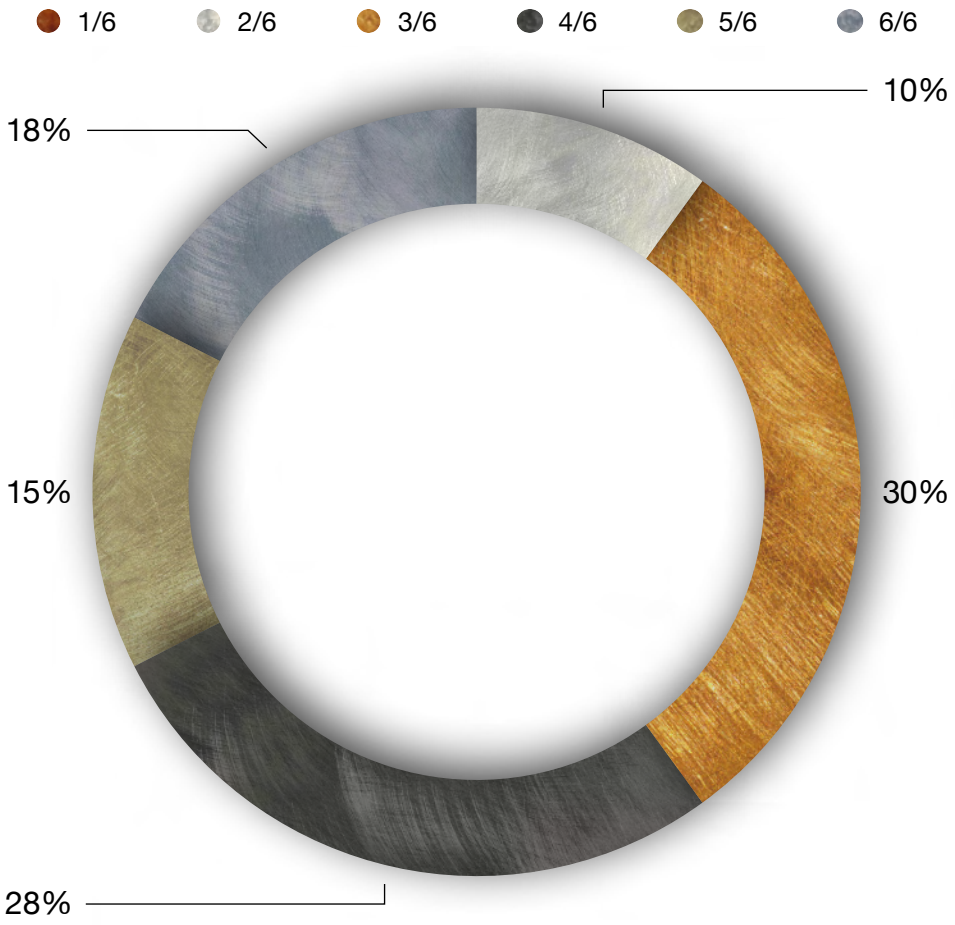
GRAPH 6 : CLINICAL EAR FINDING

TABLE 7 - HOUSE BRACKMANN GRADING ON DAY OF PRESENTATION

HOUSE BRACKMAN GRADING ON DAY OF PRESENTATION	FREQUENCY	PROPOTION
1/6	0	0%
2/6	4	10%
3/6	12	30%
4/6	11	27.5%
5/6	6	15%
6/6	7	17.5%

In our study 30% of the study population presented with House Brackmann grading of 3/6, 27.5% had 4/6, 17.5% had 6/6, 15% had 5/6, 10% had 2/6.

Most common HOUSE BRACKMANN grading during presentation was 3/6.

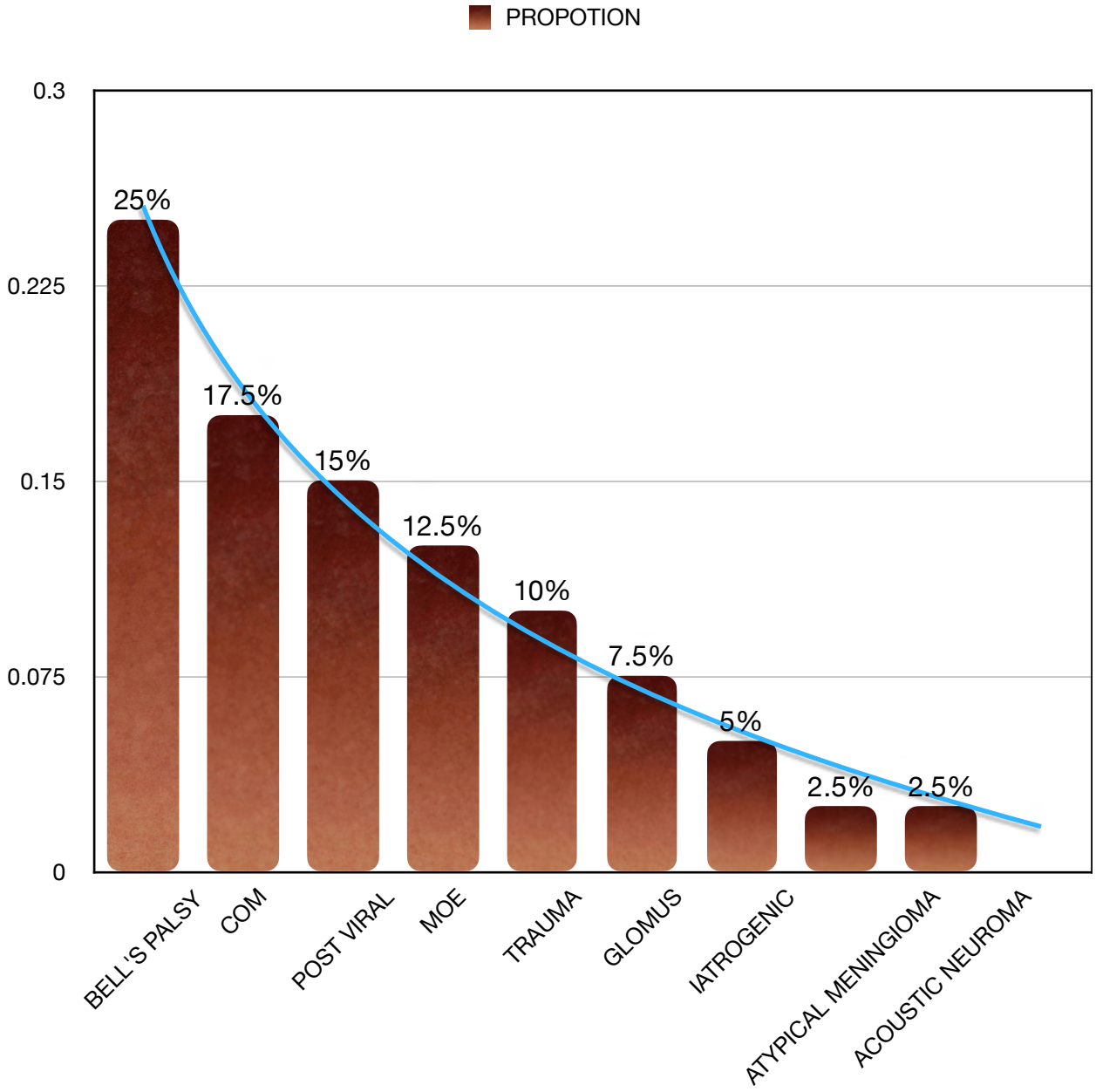


GRAPH 7 - HOUSE BRACKMANN GRADING DURING PRESENTATION

TABLE 8 - ETIOLOGY OF LMN FACIAL NERVE PALSY

ETIOLOGY	FREQUENCY	PROPOTION
BELL'S PALSY	10	25%
COM	7	17.5%
POST VIRAL INFECTIONS	6	15%
MALIGNANT OTITIS EXTERNA	5	12.5%
TRAUMA	4	10%
GLOMUS	3	7.5%
IATROGENIC	3	7.5%
ATYPICAL MENINGIOMA	1	2.5%
ACOUSTIC NEUROMA	1	2.5%

In this study it is found that 25% of the study population had Bell's Palsy ; 17.5 % had COM ; 15% had following post viral infections . 12.5% had MOE ; 10% had trauma ; glomus as a cause for LMN facial nerve palsy in 7.5% . 5% of the patients developed LMN facial nerve palsy iatrogenically following MRM, cortical mastoidectomy and submandibular gland resection surgeries . Other causes like atypical meningioma and acoustic neuroma leading to facial nerve palsy were only 2.5%

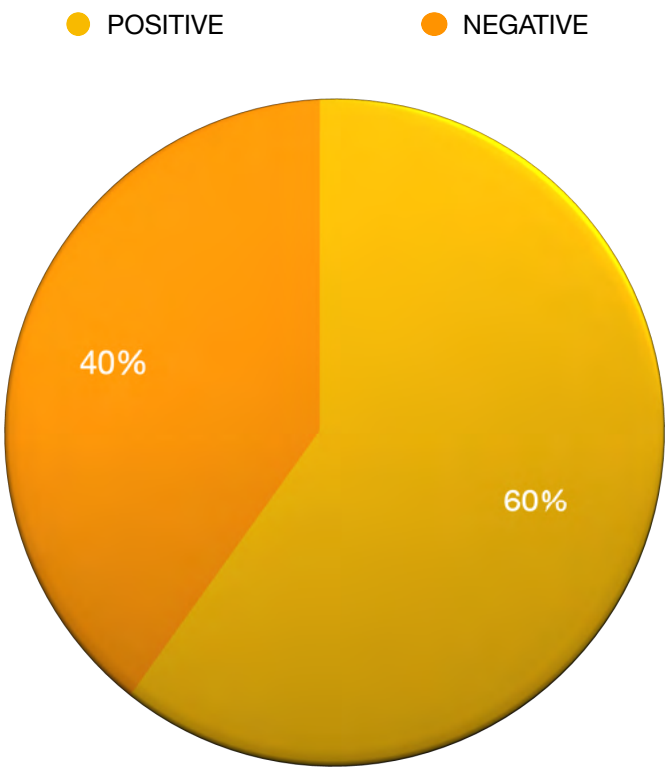


GRAPH 8 : ETIOLOGY OF LMN FACIAL PALSY

TABLE 9 - SCHIRMER TEST

SCHIRMER TEST	FREQUENCY	PROPOTION
POSITIVE	24	60%
NEGATIVE	16	40%

In our study we found 60% of the patients tested positive and 40% negative for Schirmer test at the time of presentation with LMN facial nerve palsy.

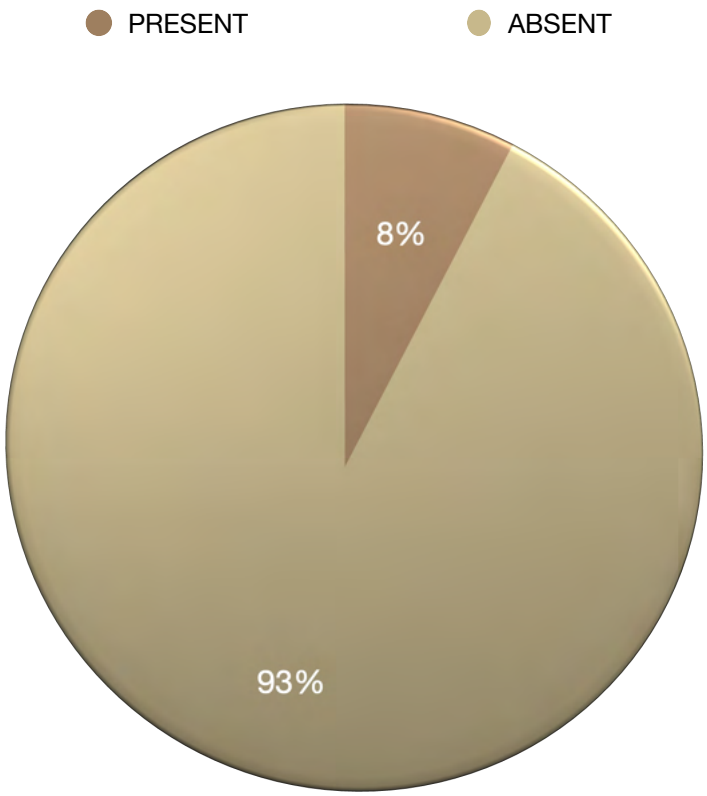


GRAPH 9 : SCHIRMER'S TEST

TABLE 10 - ACOUSTIC REFLEX

ACOUSTIC REFLEX	FREQUENCY	PROPOTION
PRESENT	3	7.5%
ABSENT	37	92.5%

In our study acoustic reflex was absent on 93% of patients and preserved in only 3 % patients who were diagnosed either with post RTA , or post submandibular gland excision with marginal mandibular nerve palsy or retro positive patient presenting with acute clinical features.

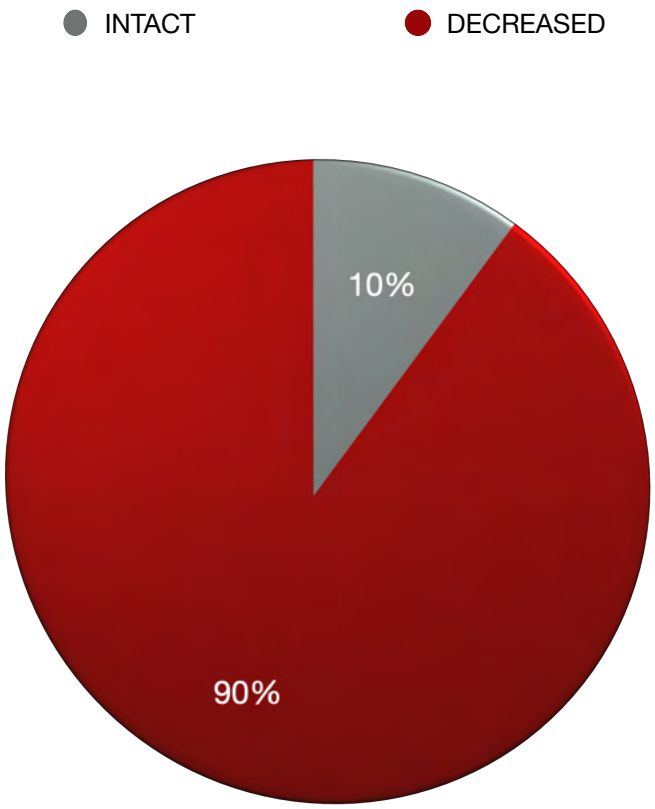


GRAPH10 : ACOUSTIC REFLEX

TABLE 11 - TASTE SENSATION

TASTE SENSATION	FREQUENCY	PROPOTION
INTACT	3	7.5%
DECREASED	37	92.5%

In our study , 92.5% of the patients had reduced taste sensation and it was found intact in only 7.5% (3 patients) who are diagnosed with LMN facial palsy post RTA, post submandibular gland excision with marginal mandibular nerve palsy , retroviral disease patient in whom acoustic reflex and taste sensation were preserved

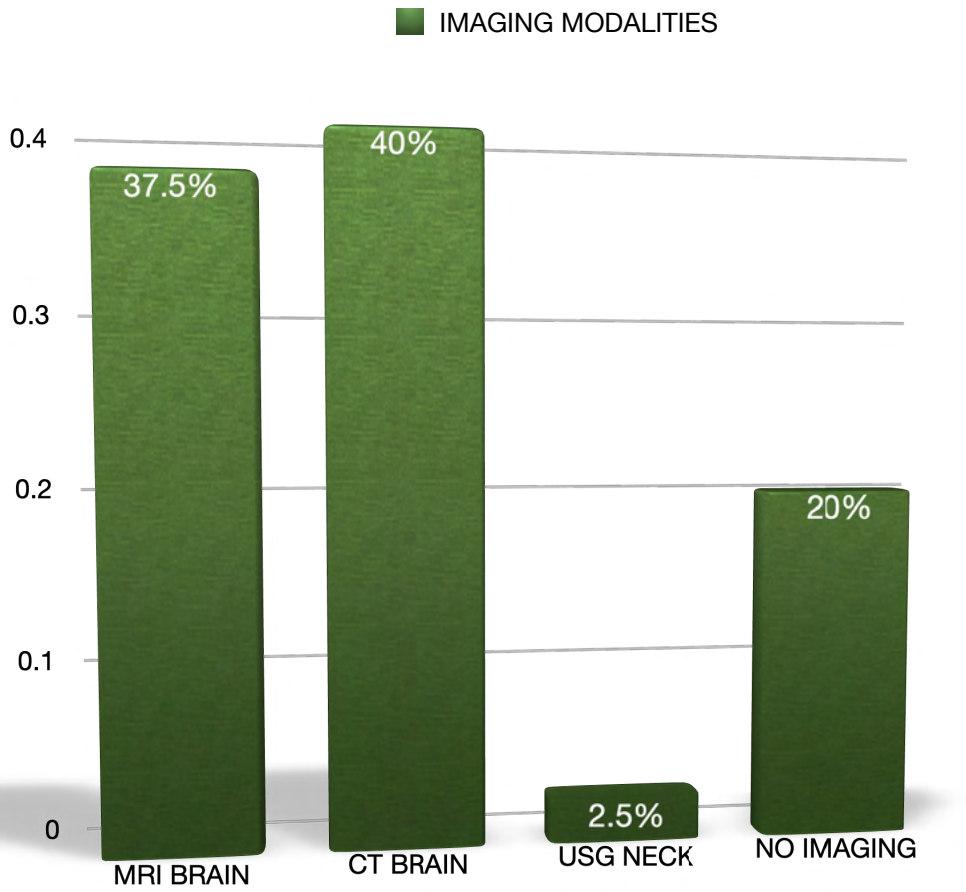


GRAPH 11 : TASTE SENSATION

TABLE 12 - IMAGING MODALITIES

IMAGING	FREQUENCY	PROPOTION
MRI BRAIN	15	37.5%
CT BRAIN	16	40%
USG NECK	1	2.5%
NO IMAGING	8	20%

In our study MRI Brain has been used as an imaging modality in 37.5 % of patients in whom clinical disease involving soft tissues and vascular structures in brain and 40% patients required CT brain as an imaging modality where skull , petrous and temporal bone were involved . 1 patient (2.5%) required USG neck for confirming parotits . 20% patients have been diagnosed clinically and required no imaging facilities.



GRAPH 12 : IMAGING MODALITIES

DISCUSSION :

The study was conducted among 40 participants , with preponderance of male patients (65%) and females (35%) between 4 - 80 years. The most common presenting complaint was deviation of mouth to contralateral side (52.5%) followed by hard of hearing (20%) and were diagnosed with peripheral facial nerve palsy at most common duration of presentation at 3-6 days (27.5%) .

Among our study population 42.5% were found to be associated with diabetes mellitus and associated comorbidities which include SHTN , CVA, CAD , HIV , TB , MUCORMYCOSIS (22.5%).

The most of our study population had normal ear finding (52.5%) on clinical examination . The other clinical findings included , granulation in EAC (12.5%), oedematous EAC (10%) ,TM perforation, haemotympanum (7.5%) .

We found 30% of the study population presented with HOUSE BRACKMANN grade of 3/6 .

The most common cause of peripheral facial nerve palsy was found to be BELL'S PALSY (25%) followed by the second most common cause being chronic otitis media (17.5%) . The other causes included post viral infection (15%) malignant otitis externa (12.5%), trauma (10%), glomus jugulare (7.5%) iatrogenic (7.5%) atypical meningioma and acoustic neuroma (2.5%).

Venugopal et al [105] found that trauma (41.7%) contributed to the major proportion of cases. It may be Iatrogenic (20%) or Non-Iatrogenic (21.7%). A large number of road traffic accidents constituted 21.7% cases of facial palsy were accounted for trauma of which 16.7% showed fracture of temporal bone (longitudinal) and 5% showing blunt trauma to temporal region . The second most common aetiology was Bell's palsy (23.3%). Incidence of facial palsy secondary to Chronic otitis media is 67% of which one case was tuberculous otits media. Cholesteatoma was the most common infective cause . Malignant otitis externa was cause of facial palsy in diabetic patients in whom there was poor glycemic control. 5% of patients had Ramsay Hunt syndrome. Tumour was the cause of facial palsy in 13.3% of patients. Male preponderance of tumour to facial palsy was 83%. Majority of patients in their study was between 31 – 40 years. A slight male predominance was seen. Road traffic accident was common in young males and infectious pathologies were more in woman. Majority reported with House Brackmann grade III palsy (30%), followed by grade IV palsy (25%). Topodiagnostic tests showed Bell's palsy involving supragenicular region (44.4%). Iatrogenic trauma was common in infrastapedial region . Temporal bone fractures had supragenicular lesion.

Bharathi M et al [123] did a study in which 101 patients were analysed of which 25.7% were in third decade of age; 55.4% were males, and both right and left sides of the face are involved equally. Most patients (50.5%) had a history of post aural pain at presentation.Topodiagnostic tests showed majority of Bell’s palsy patients had

geniculate or supragenicular lesions (67.3%) in the study. 20.8% had lesion above the nerve to stapedius, and 11.9% had lesion below the nerve to stapedius.50.4% of patients had a House-Brackmann (HB) facial nerve grade IV while they presented to study.

But we found that in our study most of the patients with facial nerve palsy at the time of presentation was found to have 3/6 .

Topodiagnostic tests among our study population showed 60% positive Schirmer's tests , absent acoustic reflex in 92.5% , compromised taste sensation in 92.5%.

Manish Munjal et al [116] conducted a retrospective study of 500 cases of head injury . 48 patients of facial palsy were taken to study the role of topodiagnostic tests in localising the lesion site. In 48 patients of facial palsy, taste sensation was decreased in 67% (21 cases); acoustic reflex was absent in 86.8% (33 cases) and Schirmer's test showed decreased lacrimation in 29.1% (14 cases). He also concluded that topodiagnostic tests do not always localise the lesion site in head injury.

In our study we have considered MRI in 37.5% patients, CT in 40 % of the patients for localisation of the pathology .

At the end the study we found that , Peripheral facial nerve palsy is associated with male preponderance presenting with deviation of mouth to contralateral side on 3-6 days of presentation with most common HOUSE BRACKMANN GRADE 3/6 & etiology being BELL'S PALSY and the site of lesion were localised with the help of topodiagnostic tests and imaging modalities (MRI/CT/USG).

CONCLUSION:

On the basis of current clinical prospective cross sectional study we conclude that , the peripheral facial nerve palsy has male preponderance with most common etiology being BELL'S PALSY presenting with contralateral side deviation of mouth & HOUSE BRACKMANN grade 3/6 and site of lesion localised with the help of topodiagnostic tests and non invasive imaging modalities .

LIMITATION OF THE STUDY :

- This study did not include follow up or recovery of the patients.
- Topodiagnostic tests are less reliable and limited correlation with precise site of nerve damage.
- This study did not use electrophysiologic tests for prognostication.

ETHICAL CLEARANCE : cleared

FUNDS : self / patients

SUMMARY:

- The study was conducted among 40 participants , with preponderance of male patients (65%) and females (35%) between 4 - 80 years.
- The most common presenting complaint was deviation of mouth to contralateral side (52.5%).
- The most common duration of presentation at 3-6 days (27.5%) .
- Among our study population 42.5% were found to be associated with diabetes mellitus.
- The most of our study population had normal ear finding (52.5%) on clinical examination.
- We found 30% of the study population presented with HOUSE BRACKMANN grade of 3/6.
- The most common cause of peripheral facial nerve palsy was found to be BELL' PALSY (25%).
- Topodiagnostic tests among our study population showed 60% positive Schirmer's tests , absent acoustic reflex in 92.5% , compromised taste sensation in 92.5%.
- In our study we have considered MRI in 37.5% patients, CT in 40% of the patients for localisation of the pathology .

- We conclude that , the peripheral facial nerve palsy has male preponderance with most common etiology being BELL'S PALSY presenting with contralateral side deviation of mouth & HOUSE BRACKMANN grade 3/6 and site of lesion localised with the help of topodiagnostic tests and non invasive imaging modalities .

BIBLIOGRAPHY

1. Venugopal M, Rajan S, Suma R, Thomas S. Etiopathogenesis of lower motor neuron facial palsy : Our experience. Indian J Otol 2011; 17:58-62
2. Gasser RF (1967) The development of the facial nerve in man. Ann Otol Rhinol Laryngol, 76: 37–56.
3. Gasser RF (1970) The early development of the parotid gland around the facial nerve and its branches in man. Anat Rec, 167: 63–78. 16. Gasser RF, May M (1986) Embryonic development of the facial nerve. In Facial nerve. Thieme Inc, New York, 3–20
4. Gasser RF, May M (1986) Embryonic development of the facial nerve. In Facial nerve. Thieme Inc, New York, 3–20.
5. Sataloff RT (1990) Embryology of the facial nerve and its clinical applications. Laryngoscope, 100: 969–984.
6. Sataloff RT (1991) Embryology and anomalies of the facial nerve. Raven Press, New York.
7. Gerhardt HJ (1981) The intratemporal course of the facial nerve and its influences on the development of the ossicular chain. Acta Otolaryngol, 91: 567–573.
8. M. Weglowski, W. Woźniak, A. Piotrowski, M. Bruska, J. Weglowska, J. Sobański, M. Grzymisławska, J. ŁupickaEarly Development of the facial nerve in human embryos at stages 13–15 (Folia Morphol 2015; 74, 2: 252–257)
9. Buskirk C van (1945) The seventh nerve complex. J Comp Neurol, 82: 303–333.
10. Foley JO (1960) Quantitative studies of the functional components of the facial nerve. Am J Anat, 107: 237–244.
11. Thurner KH , Egg G, Spoendlin H, Schrott – Fischer A. A quantitative study of nerve fibres in the human facial nerve. Eur Arch Otorhinolaryngol 1993;250:161 – 7
12. Captier G et al . Organization and microscopic anatomy of the adult human facial nerve: Anatomical and histological basis for surgery. Plast Reconstr Surg 2005;115:1457- 65.

13. Alfieri A, Strauss Ch, Prell J, Pesche E (2010) History of the nervus intermedius of Wrisberg. Ann Anat, 192: 139–144.

14. Bruyn GW (1984) Nervus intermedius neuralgia (Hunt).Cephalalgia, 4: 71–78.

15.Oh CS, Chung IH, Lee KS, Tanaka S (2003) Morphological study on the rootlets comprising the root of the intermediate nerve. Anat Sci Int, 78: 111–113.

16. Rhoton jr AL, Kobayashi S, Hollinshead WH (1968) Nervus intermedius. J Neurosurg, 29: 609–618.

17. Van Buskirk C. The seventh nerve complex. *J Comp Neurol* 1945; **82**: 303–33.

18. Haines DE. *Fundamental neuroscience for basic and clinical applications*, Ch.14 Cambridge, MA: Elsevier Inc. 2013.

19. Alfieri A, Strauss C, Prell J, Peschke E. History of the nervus intermedius of Wrisberg. *Ann Anat* 2010; **192**: 139–44.

20. Alfieri A, Fleischhammer J, Strauss C, Peschke E. The central myelin-peripheral myelin transitional zone of the nervus intermedius and its implications for microsurgery in the cerebellopontine angle. *Clin Anat* 2012; **25**: 882–8.

21. Tubbs RS, Steck DT, Mortazavi MM,Cohen-Gadol AA. The nervus intermedius:A review of its anatomy,function, pathology, and role in neurosurgery. *World Neurosurg* 2013; **79**(5–6):763–7.

22. Alfieri A, Fleischhammer J, Strauss C, Peschke E. The central myelin-peripheral myelin transitional zone of the nervus intermedius and its implications for microsurgery in the cerebellopontine angle. *Clin Anat* 2012; **25**: 882–8.

23. Tian GY, Xu DC, Huang DL, et al. The topographical relationships and anastomosis of the nerves in the human internal auditory canal. *Surg Radiol Anat* 2008; **30**:243–7.

24. Bernardo A, Evins AI, Visca A, et al.2013. The intracranial facial nerve as seen through different surgical windows:An extensive anatomosurgical study. *Neurosurgery* 2013; **72** (2 Suppl Operative): 194–207.

25. Tanriover N, Sanus GZ, Ulu MO, et al.Middle fossa approach: Microsurgicalanatomy and surgical technique from the neurosurgical perspective. *Surg Neurol* 2009*;* **71**: 586–96.

26. Lysek MC. Anatomy of the facial nerve.In: Tubbs RS, Rizk E, Shoja MM et al, (eds). *Nerves and nerve injuries,* Vol 1.Philadelphia: Academic Press, Elsevier,2015, Ch. 24, pp. 357–64.

27. Bartoshuk LM, Snyder DJ, Grushka M, et al. Taste damage: Previously unsuspected consequences. *Chem Senses* 2005; **30** (suppl 1): i218–i219.

28. Berteretche M-V, Eloit C, Dumas H, et al.Taste deficits after middle ear surgery for otosclerosis: Taste somatosensory interactions. *Eur J Oral Sci* 2008; **116:**394–404.

29. Tanriover N, Sanus GZ, Ulu MO, et al.Middle fossa approach: Microsurgical anatomy and surgical technique from the neurosurgical perspective. *Surg Neurol* 2009*;* **71**: 586–96.

30. Lan MY, Shiao JY. Using greater superficial petrosal nerve and geniculate ganglion as the only two landmarks for identifying internal auditory canal in middle fossa approach. *Eur Arch Otorhinolaryngol* 2010; **267**: 1867–71

31. Kakizawa Y, Abe H, Fukushima Y, et al.The course of the lesser petrosal nerve on the middle cranial fossa. *Neurosurgery* 2007; **61**(3 Suppl): 15–23.

32. Mu X, Quan Y, Shao J, et al. Enlarged geniculate ganglion fossa: CT sign of facial nerve canal fracture. *Acad Radiol* 2012; **19**: 971–6.

33.Tanriover N, Sanus GZ, Ulu MO, et al.Middle fossa approach: Microsurgical anatomy and surgical technique from the neurosurgical perspective. *Surg Neurol* 2009*;* **71**: 586–96.

34. Nikolaidis V, Nalbadian M, Psifidis A,et al. The tympanic segment of the facial nerve: Anatomical study. *Clin Anat* 2009;**22**: 307–10.

35. Donaldson JA, Anson BJ, Warpeha RL Rensink MJ. The surgical anatomy of the sinus tympani. *Arch Otolaryngol* 1970; **91**:219–27.

36. Nitek S, Wysocki J, Niemczyk K, Ungier E.The anatomy of the tympanic sinus. *Folia Morphol* 2006; **65**: 195–9.

37. Marchioni D, Alicandri-Ciufelli M,Grammatica A, et al. Pyramidal eminence and subpyramidal space: An endoscopic anatomical study. *Laryngoscope* 2010;**120**: 557–64.

38. Moreano EH*,* Paparella MM*,*Zelterman D, Goycoolea MV. Prevalence of facial canal dehiscence and of persistent stapedial artery in the human middle ear: A report of 1000 temporal bones.*Laryngoscope* 1994; **104**: 309–20.

39. Barnes G, Liang JN, Michaels L,et al. Development of the fallopian canal in humans: A morphologic and radiologic study. *Otol Neurotol* 2001;**22**: 931–7.

40. Di Martino E, Sellhaus B, Haensel J, et al.Fallopian canal dehiscences: A survey of clinical and anatomical findings. *Eur Arch Otorhinolaryngol* 2005; **262**: 120–6.

41. Isaacson B, Vrabec JT. The radiographic prevalence of geniculate ganglion dehiscence in normal and congenitally thin temporal bones. *Otol Neurotol* 2007; **28**:107–10.

42. Trinidade A, Yung MW. The intra-operative incidence of Fallopian canaldehiscence during surgery for cholesteatoma: A prospective case-control study and review of the literature. *Clin Otolaryngol* 2014; **39**: 138–44

43. Pauna HF, Monsanto RC, Schachern PA,et al. The surgical challenge of carotid artery and Fallopian canal dehiscence in chronic ear disease: A pitfall for endoscopic approach. *Clin Otolaryngol* 2017;
42(2): 268–74.

44. Zaghal ZA, Raad RA, Nassar J, et al.Anatomic relationship between the facial nerve and the tympanic annulus. *Otol Neurotol* 2014; **35**: 667–71.

45. Dahm MC, Shepherd RK, Clark GM. The postnatal growth of the temporal bone and its implications for cochlear implantation in children. *Acta Otolaryngol* Suppl 1993;**505**: 1–39.

46. McManus LJ, Dawes PJD, Stringer MD.Clinical anatomy of the chorda tympani:A systematic review. *J Laryngol Otol* 2011;**125**: 1101–8.

47. Kulczynski B, Wozniak W. Variation of the origin and course of the chorda tympani. *Folia Morphol* 1978; **37**: 237–41.

48. Laing MR, McKerrow WS. Intraparotid anatomy of the facial nerve and retromandibular vein. *Br J Surg* 1998; **75**: 310–12.

49. Piagkou M, Tzika M, Paraskevas G, Natsis K. Anatomic variability in the relation between the retromandibular vein and the facial nerve: A case report, literature review and classification. *Folia Morphol (Warsz)* 2013; **72**: 371–5.

50. Pather N, Osman M. Landmarks of the facial nerve: Implications for parotidectomy. *Surg Radiol Anat* 2006; **28**: 170–5.

51. Davis RA, Anson BJ, Budinger JM, Kurth LR. Surgical anatomy of the facial nerve and parotid gland based upon a study of 350 cervicofacial halves. *Surg Gynecol Obstet* 1956; **102**: 385–412.

52. Kwak HH, Park HD, Youn KH, et al. Branching patterns of the facial nerve and its communication with the auriculotemporal nerve. *Surg Radiol Anat* 2004; **26**: 494–500.

53. Gosain AK. Surgical anatomy of the facial nerve. *Clin Plast Surg* 1995; **22**: 241–51.

54. Lineaweaver W, Rhoton A, Habal MB. Microsurgical anatomy of the facial nerve. *J Craniofac Surg* 1997; **8**: 6–10.

55. Kochhar A, Larian B, Azizzadeh B.Facial nerve and parotid gland anatomy. *Otolaryngol Clin North Am* 2016; **49**:273–84.

56. Roostaeian J, Rohrich RJ, Stuzin JM.Anatomical considerations to prevent facial nerve injury. *Plast Reconstr Surg* 2015; **135**: 1318–27.

57. Sood AJ, Houlton JJ, Nguyen SA,Gillespie MB. Facial nerve monitoring during parotidectomy: A systematic review and meta-analysis. *Otolaryngol Head Neck Surg* 2015; **152**: 631–7.

58. Rea PM, McGarry G, Shaw-Dunn J. The precision of four commonly used surgical landmarks for locating the facial nerve in anterograde parotidectomy in humans. *Ann Anat* 2010; **192**(1):27–32.

59. Muhleman MA, Wartmann CT, Hage R, et al. A review of the tragal pointer:Anatomy and its importance as a landmark in surgical procedures. *Folia Morphol*
(Warsz) 2012; **71**: 59–64.

60. Zhong W, Ashwell K. A cadaveric study of surgical landmarks for retrograde parotidectomy. *Ann Med Surg (Lond)* 2016; **9**: 82–5.

61. Blunt MJ. The blood supply of the facial nerve. *J Anat* 1954; **88**: 5206.

62. Hendrix P, Griessenauer CJ, Foreman P, et al. Arterial supply of the lower cranial nerves: A comprehensive review. *Clin Anat* 2014; **27**: 108-17.

63. Mortazavi MM, Latif B, Verma K, et al. The fallopian canal: a comprehensive review and proposal of a new classification. *Childs Nerv Syst* 2014; **30**:387–95.

64. El-Khouly H, Fernandez-Miranda J, Rhoton AL Jr. Blood supply of the facial nerve in the middle fossa: The petrosal artery. *Neurosurgery* 2008; **62**(5 Suppl 2):ONS297–303.

65. Anson BJ, Donaldson JA, Warpeha RL, et al. Surgical anatomy of the facial nerve *Arch Otolaryngol Head Neck Surg* 1973; **97**: 201–13.

66. Hall SM. The biology of chronically denervated Schwann cells. *Ann N Y Acad Sci* 1999; **883**: 215–33.

67. Sulaiman OA, Gordon T. Role of chronic Schwann cell denervation in poor functional recovery after nerve injuries and experimental strategies to combatit. *Neurosurgery* 2009; **65**(4 Suppl): 105–14.

68. Poppler LH, Ee X, Schellhardt L, et al.Axonal growth arrests after an increased accumulation of Schwann cells expressing senescence markers and stromal cells in acellular nerve allografts. *Tissue Eng Part A* 2016; **22**(13–14): 949–61.

69. Mackinnon SE. New directions in peripheral nerve surgery. *Ann Plast Surg* 1989;**22**: 257–73.

70. Birch R. *Surgical disorders of the peripheral nerve*s. 2nd ed. Springer; London;2011.

71. Grinsell D, Keating CP. Peripheral nerve reconstruction after injury: a review of clinical and experimental therapies. *Biomed Res Int* 2014; 698256.

72. Seddon HJ. A classification of nerve injuries. *BMJ* 1942; **2:** 237–9.

73. Bowden REM, Gutmann E. Denervation and re-innervation of human voluntary muscle. *Brain* 1944; **67**: 273–313.

74. Menorca RMG, Fussell TS, Elfar JC.Peripheral nerve trauma: Mechanisms of injury and recovery. *Hand Clin* 2013; **29**: 317–30.

75. Lee SK, Wolfe SW. Peripheral nerve injury and repair. *J Am Acad Orthop Surg* 2000;**8**: 243–52.

76. Sunderland S. A classification of peripheral nerve injuries producing loss of function. *Brain* 1951; **4**: 491–516.

77. Allodi I, Udina E, Navarro X. Specificity of peripheral nerve regeneration: interactions at the axon level. *Prog Neurobiol* 2012;**98**: 16–37.

78. Hall SM. The biology of chronically denervated Schwann cells. *Ann N Y Acad Sci* 1999; **883**: 215–33.

79. House JW, Brackmann DE. Facial nerve grading system. *Otolaryngol Head Neck Surg* 1985; **93**: 146–7.

80. Burres S, Fisch U. The comparison of facial grading systems. *Arch Otolaryngol Head Neck Surg* 1986; **112**: 755–8.

81. Murty GE, Diver JP, Kelly PJ, et al. The Nottingham system: objective assessment of facial nerve function in the clinic. *Otolaryngol Head Neck Surg* 1994; **110**:156–61.

82. Ross BG, Fradet G, Nedzelski J.Development of a sensitive clinical Facial grading system. *Otolaryngol Head Neck Surg* 1996; **114**: 380 –6.

83. May M, Harvey JE, Marowitz WF,Stroud M. The prognostic accuracy of the maximal stimulation test compared with that of the nerve excitability test in Bell's palsy. *Laryngoscope* 1971; **81**: 931–8.

84. Gantz BJ. Idiopathic facial paralysis.In: Gates G (ed.). *Current therapy in otolaryngology – head and neck surgery*.Philadelphia: BC Decker; 1987, pp. 62–6.

85. Fisch U. Facial paralysis in fractures of the petrous bone. *Laryngoscope* 1974; **84**:2141–54.

86. Fisch U. Management of intratemporal facial nerve injuries. *J Laryngol Otol* 1980;**94**: 129–34.

87. Guillou-Jamard MR, Labbe D, Bardot J, Benateau H. Paul Tessier's technique for the treatment of paralytic lagophthalmos by lengthening of the levator muscle: evaluation of 29 cases. *Ann Plast Surg* 2011; **67**(6): S31–5.

88. Hayashi A, Yoshizawa H, Natori Y, et al. Levator lengthening technique using cartilage or fascia graft for paralytic lagophthalmos in facial paralysis. *J Plast Reconstr Aesthet Surg* 2016; **69**(5): 679–86.

89. Taverner D. The prognosis and treatment of spontaneous facial palsy. *Proc R Soc Med* 1959; **52**: 1077–80.

90. Yanagihara N. Incidence of Bell's palsy. *Ann Otol Rhinol Laryngol* Suppl 1988; **137**: 3–4.

91. Peitersen E. Bell's Palsy: The spontaneous course of 2,500 peripheral facial nerve palsies of different etiologies. *Acta Otolaryngol* Suppl 2002; **549**: 4–30.

92. Hussein, A, Nduka N, Moth P, Malhotra R. Bell's facial nerve palsy in pregnancy: A clinical review. *J Obstst Gynaecol* 2017; **31**: 1–7.

93. Mutsch M, Zhou W, Rhodes P, et al. Use of the inactivated intranasal influenza vaccine and the risk of Bell's palsy. *N Eng J Med* 2004; **350**: 896–903.

94. Murakami S, Honda N, Mizobuchi M, et al. Rapid diagnosis of varicella zoster virus infection in acute facial palsy. *Neurol* 1998; **51**: 1202–5.

95. Robillard RB, Hilsinger Jr RL, Adour KK. Ramsay Hunt facial paralysis: Clinical analyses of 185 patients. *Otolaryngol Head Neck Surg* 1986; **95**: 292–7.

96. Terada K, Niizuma T, Kosaka Y, et al. Bilateral facial nerve palsy associated with Epstein-Barr virus infection with a review of the literature. *Scand J Infect Dis* 2004; **36**: 75–7.

97. Belec L, Georges AJ, Bouree P, et al. Peripheral facial nerve palsy related to HIV infection: Relationship with the immunological status and the HIV staging in Central Africa. *Cent Afr J Med* 1991; **37**: 88–93.

98. Dahiya R, Keller J, Litofsky SN, et al. Temporal bone fractures: Otic capsule sparing versus otic capsule violating. Clinical and radiographic considerations. *J Trauma* 1999; **47**: 1079–83.

99. Brodie HA, Thompson TC. Management of complications form 820 temporal bone fractures. *Am J Otol* 1997; **18**: 188–97.

100. Quaranta A, Campobasso G, Piazza F, et al. Facial nerve paralysis in temporal bone fractures: Outcomes after late decompression.*Acta Otolaryngol* 2001; **121**: 652–5.

101. Lambert PR, Brackmann DE. Facial paralysis in longitudinal temporal bone fractures:A review of 26 cases. *Laryngoscope*1984; **94**: 1022–6.

102. Wiet RJ. Latrogenic facial paralysis. *Otolaryngol Clin North Am* 1982; **15**: 773–80.

103. Green JD, Shelton C, Brackmann DE.Iatrogenic facial nerve injury during otologic surgery. *Laryngoscope* 1994; **104**: 922–6.

104. Maw JL, Kartush JM. Traumatic and neoplastic disorders of the facial nerve.In: Canalis RF, Lambert PR (eds). *The ear: Comprehensive otology*. Philadelphia: Lippincott Williams & Wilkins; 2000, pp. 735–50.

105. Grandis JR, Branstetter BF IV, Yu VL. The changing face of malignant (necrotising) external otitis: Clinical, radiological, and anatomic correlations. *Lancet Infect Dis* 2004; **4**: 34–9.

106. Patel N, Poole A. Recurrent benign parotid tumours. The lesson not learned yet. Aust N Z J Surg 1998;68:562-4.

107. Eftekharian A. Facial nerve in middle ear and mastoid surgery. Otol Neurotol 2005;26:1093.

108. Guerrissi JO. Facial nerve paralysis after intratemporal and extratemporal blunt trauma. J Craniofac Surg 1997;8:431-7.

109. Nageris B , Hansen MC, Lavelle WG, Van Delt FA Temporal bone fracture Am J Emer Med 1995;13:211-4.

110. Altuntas A, Unal A, Aslan A, et al. Facial nerve paralysis in chronic suppurative otitis media: Ankara numune hospital experience. Auris Nasus Larynx. 1998;25(2):169–172.

111. Kirsch CM, Webner JH, Jensen WA, Kagawa FT, Campagna AC. Tubeulous otitis media. South Med J 1995;88:363-6.

112. Yeo SW, Lee DH, Jun BC, Chang KH, Park YS. Analysis of prognostic factors in Bell's palsy and Ramsay Hunt syndrome Auris Nasus Larynx 2007;34:159-64.

113. Rosenberg SI. Natural history of acoustic neuromas. Laryngoscope 2000;110:498-508.

114. Ayala Mejias A, Casqueiro Sanchez JC, Durio Calero E, Sanz Fernandez R. Peripheral facial palsy. Descriptive study at the university hospital in Getafe - Acta Otolaringol - Esp 2007;58:52-511.

115. Chow LC, Tam RC, Li MF. Use of electroneurography as a prognostic indicator of Bell's palsy in Chinese patients Otol Neurotol 2002;23:598-601.

116. Munjal M, Badyal HL, Mehndiratta A, Munjal S, Bharadwaj I, Kalra S, Mehta A. Role of topodiagnostic tests in evaluation of facial nerve palsy in head injury. Int J Otorhinolaryngol Head Neck Surg 2020;6:1874-7.

117. Dobie RA. Electrical and topognostic tests of the facial nerve. In: CW, Cummings, JM Fredrickson eds. Otolaryngology Head and Neck Surgery. 2nd ed. New Jersy: Wiley;1986:2821-7.

118. Tschiassny K. Eight syndromes of facial paralysis and their significance in locating the lesion. Annals of Otology. Rhinology & Laryngology. 1953;62(3): 677-91.

119. Lambert PR, Brackmann DE. Facial paralysis in longitudinal temporal bone fractures: A review of 26 cases. The Laryngoscope. 1984;94(8):1022-6 120. GantzBJ, Gmür A, Fisch U. Intraoperative evoked electromyography in Bell's palsy. American journal of otolaryngology. 1982;3(4):273-8.

121. Renou G, Psaume T. Diagnostic topographique de la paralysie faciale a frigore topographical diagnosis of facial paralysis (Bell's palsy) (author's transl). Ann Otolaryngol Chir Cervicofac. 1978;95(12):769-75.

122. Ide M, Morimitsu T, Ushisako Y, Makino K, Fukiyama M, Hayashi A. The significance of stapedial reflex test in facial nerve paralysis. Acta Otolaryngol Suppl. 1988;446:57-63.

123. Bharathi, M. & Unisa, Thanzeem & Chandresh, Swathi & R., Venkatesh & S., Harsha. (2019). Clinical Study of Bell's Palsy at Tertiary Care: Our Experience. Annals of Otology and Neurotology. 02. 10.1055/s-0039-1693096.

124. Chandler JR. Malignant external otitis: further considerations. *Ann Otol Rhinol Laryngol*. 1977 Jul-Aug. 86(4 Pt 1):417-28.

125.Franco-Vidal V, Blanchet H, Bebear C, Dutronc H, Darrouzet V. Necrotizing external otitis: a report of 46 cases. *Otol Neurotol*. 2007 Sep. 28(6):771-3.

126. Corey JP Levandowski RA Panwalker AP Prognostic implications of therapy for necrotizing external otitis. *Am J Otol* 1985;6 (4) 353- 357

127. Soudry E, Joshua BZ, Sulkes J, Nageris BI. Characteristics and Prognosis of Malignant External Otitis With Facial Paralysis. *Arch Otolaryngol Head Neck Surg*. 2007;133(10):1002–1004. doi:10.1001/archotol.133.10.1002

OP NO/IP NO	AGE	SEX	COMPLAINTS	DURATION	COMORBIDITIES	EAR	DIAGNOSIS WITH HB SCALE	ETIOLOGY	SCHIRMER S TEST	ACOUSTIC REFLEX	TASTE SENSATION	IMAGING AND OTHER
O21056301	45	M	RIGHT EAR PAIN. DEVIATION OF MOUTH TO LEFT	1w 3d	NIL	NORMAL	4/6	RIGHT BELL'S PALSY	POSITIVE	ABSENT	DECREASED	NIL ; INC NEU;DEC LYMP; MP; TFT NORMAL
O210325111	21	M	K/C/O B/L CSOM WITH ACTIVE LEFT EAR DISCHARGE AND DEVIATION OF MOUTH TO RIGHT	1d	NIL	B/L TM PERFORATION - CP	4/6	B/L COM - LEFT 7TH NERVE PALSY	NEGATIVE	ABSENT	DECREASED	NIL
O20003740	51	F	DEVIATION OF MOUTH TO RIGHT , RIGHT EAR RINGING SENSATION	2d	SLE, RA, SHTN, DM , OLD CVA, HYPOTHYROID	NORMAL	3/6	RIGHT BELL'S PALSY	POSITIVE	ABSENT	DECREASED	MRI - NORMAL;INC NEU; EU;DEC LYMP
O21007502	19	F	DEVIATION OF MOUTH TO LEFT , RIGHT HARD OF HEARING, EXPOSURE TO COLD	3d	NIL	NORMAL	3/6	RIGHT BELL'S PALSY	POSITIVE	ABSENT	DECREASED	NIL
I20030754	32	M	POST RTA - LEFT EAR DISHARGE	2d	NIL	LEFT OTITIS EXTERNA	2/6	RIGHT PALSY- POST RTA	NEGATIVE	PRESENT	NORMAL	THIN SDH IN LEFT FPT WITH INTRAPARENCHYMAL HE
O20021819	72	F	DEVIATION OF MOUTH TO RIGHT, LEFT TINNITUS	1d	DM;SHT	NORMAL	5/6	LEFT BELL,S PALSY	POSITIVE	ABSENT	DECREASED	HBA1C - 8.2,LYMP DEC,CHRONIC SMALL VESSEL ISCHAEMIC CHANGES
O20030234	41	M	LEFT EAR ACHE,DISCHARGE X1 MONTH;FACIAL WEAKNESS X1W	1w	NIL	GRANULATIONS IN EAC	3/6	LEFT COM , LEFT 7TH NERVE PALSY	NEGATIVE	ABSENT	DECREASED	CHOLESTEATOMA WITH HORIZONTAL SEGMENT EROSION
I21033191	40	F	DEVIATION OF MOUTH TO LEFT ; WATERING OF EYE X 1 DAY, IMMEDIATE POST CORTICAL MASTOIDECTOMY	1d	NIL	POST SURGERY	3/6	IATROGENIC RIGHT 7TH NERVEL PALSY	NEGATIVE	ABSENT	DECREASED	NIL
O21034824	9	M	DEVIATION OF MOUTH TO RIGHT; IMMEDIATE POST MRM	1d	NIL	POST SURGERY	3/6	IATROGENIC LEFT 7TH NERVE PALSY	NEGATIVE	ABSENT	DECREASED	VERTICAL SEGMENT - CHOLEASTEATOMA EROSION INTRAOP
O16016650	15	F	WORSENING RIGHT FACIAL ASYMMETRY , POST R - GLOMUS TUMOR EXCISION	1w 3d	NIL	RIGHT REDDISH HUE BEHIND NEOTM	4/6	RIGHT 7TH NERVE PALSY , RECURRENT GLOMUS	POSITIVE	ABSENT	DECREASED	TUMOR INVOLVING EXPANDING VII NERVE CANAL AT ANTERIOR GENU AND TYMPANIC SEGMENT (MRI)
O18051507	57	M	EAR DISCHARGE RIGHT SIDE MOUTH DEVIATION X 1 YEAR , DCOMP CRANIOTOMY	1 year	DM/SHT/OLD CVA	LEFT SUBTOTAL PERFORATION	6/6	LEFT COM , LEFT 7TH NERVE PALSY	POSITIVE	ABSENT	DECREASED	GRANULATIONS IN MIDDLE EAR , LEFT VERTEBRAL ARTERY NOT VISUALSED;SMALL BASILIAR ARTERY (MRI)
O20057845	61	M	LEFT FACIAL PAIN AND EYE SWELLING X 3 MONTH	3 months	DM;POST ESS+ORBITAL DECOM	NORMAL	6/6	LEFT COM , LEFT 7TH NERVE PALSY, MUCORMYCOSIS	POSITIVE	ABSENT	DECREASED	PETROUS BONE AND SKULL BASE OSTEOMYELITIS ; III,IV,V1,V2,VI,VII,VIII CN PALSY
O20036093	36	F	LEFT DEVIATION OF ANGLE OF MOUTH POST RIGHT SUBMANDIBULAR GLAND EXCISION - RIGHT	1d	NIL	NORMAL	2/6	RIGHT MARGINAL MANDIBULAR NERVE PARESIS	NEGATIVE	PRESENT	NORMAL	NIL
O20056979	45	M	VESICLES,PAIN EAR X 5DAYS;DEVIATION OF MOUTH TO RIGHT X 3DAYS	3d	NIL	LEFT EAR VESICLES IN CC,EAC	6/6	LEFT 7TH NERVE PALSY , RAMSAY SYNDROME	POSITIVE	ABSENT	DECREASED	NIL; LYMP DECREASED
O21055885	61	M	RIGHT NOCTURNAL EAR PAIN X FACIAL ASYMMETRY X 5YEARS	5 years	DM/OLD CVA	R EAC EDEMA WITH GRANULATION	3/6	RIGHT MALIGNANT OITIS EXTERNA	POSITIVE	ABSENT	DECREASED	ANTERIOR TYMPANIC SEGMENT EROSION ON RIGHT(CT)
O20057663	58	M	L EAR DISCHARGE X 1ODAYS; MOUTH DEVIATION TO R X 3DAYS	3d	DM	L AURAL POLYP AND GRANULATION	6/6	LEFT MALIGNANT OTITIS EXTERNA	POSITIVE	ABSENT	DECREASED	LEFT SKULL BASE,PETROUS APEX SOFT TISSUE (CT)
O20061914	35	M	DEVIATION MOUTH LEFT X 8YEARS; RIGHT EAR DISCHARGE X 5DAYS	5d	NIL	RIGHT EAC - GRANULATION;CSF LEAK	5/6	RIGHT ATYPICAL MENINGIOMA	POSITIVE	ABSENT	DECREASED	LARGE RIGHT CP ANGLE WELL DEFINED LESION WITH TEGMEN EROSION AND MIDLINE SHIFT (MRI)
O19080785	15	M	DEVIATION MOUTH TO LEFT; RIGHT EAR BLEED,BLOCK,HOH	5d	NIL	HEMOTYMPANUM	4/6	RIGHT POST TRAUMATIC 7TH NERVE PALSY	NEGATIVE	ABSENT	DECREASED	RIGHT SQUAMOUS AND PETROUS TEMPORAL BONE FRACTURE(CT)
O18020171	12	F	RIGHT EAR AND EYE PAIN X 5DAYS;FEVER,VOMITING, LEFT 7TH NERVE PARALYSIS LEFT MRM;MENINGITIS OLD	5d	NIL	R - CHOLESTEATOMA ,L NARROW EAC	4/6	RIGHT COM , LEFT 7TH NERVE PALSY	POSITIVE	ABSENT	DECREASED	SUBDURAL EMPYEMA COMMUNICATING TO R EAC WITH CEREBRITIS;MENINGITIS;LST(CT)
O17049881	25	F	RIGHT FACIAL ASYMMETRY X 2 YEARS ; PREOP RIGHT GIANT CYSTIC ACOUSTIC NEUROMA - RS APPROACH	2years	NIL	NORMAL	4/6	RIGHT 7TH NERVE PALSY, RIGHTCYSTIC ACOUSTIC NEUROMA	POSITIVE	ABSENT	DECREASED	RIGHT V,VII,VIII,IX,X PALSY AND LEFT HEMIPARESIS(CT), (MRI)
O21056710	45	F	FEVER,HEADACHE,SEIZURES	5months	DM;CAD;CVA	NORMAL	4/6	LEFT 7TH NERVE PALSY, TB MENINGITIS	POSITIVE	ABSENT	DECREASED	LEFT X PALSY (CT)
O20013664	60	M	RIGHT EAR DISCHARGE,PAIN,DEVIATION MOUTH TO LEFT X 5MONTHS	5 months	DM	SMALL CP-RIGHT	4/6	RIGHT COM , RIGHT 7TH NERVE PALSY	NEGATIVE	ABSENT	DECREASED	R SCLEROSED MASTOID,SOFT TISSUE ANTRUM,MIDDLE EAR,HBA1C -8.7(CT)
O19081690	40	M	POST RTA - EAR, NOSE,ORAL BLEED	2d	NIL	NORMAL	3/6	RIGHT POST TRAUMATIC 7TH NERVE PALSY	NEGATIVE	ABSENT	DECREASED	RIGHT SQUAMOUS TEMPORAL BONE FRACTURE ,FISSURE TO MASTOID PORTION(CT)
O20030517	62	M	RIGHT EAR PAIN,DISCHARGE,HOH X6 MONTHS	1w	DM	RIGHT EAC EDEMA WITH GRANULATION	6/6	RIGHT MALIGNANT OITIS EXTERNA	POSITIVE	ABSENT	DECREASED	VI,VII,VIII,IX,X,XI,XII PALSY (CT)
O18075842	28	M	UNABLE TO CLOSE BOTH EYES	2d	NIL	NORMAL	6/6	B/L 7TH NERVE PALSY, GBS	POSITIVE	ABSENT	DECREASED	MRI GBS
O19016323	58	F	VERTIGO, RIGHT TINNITUS, HOH X5DAYS	5d	DM;2014 OPERATED POST OP PALSY	R REDDISH HUE BEHIND TM	3/6	RIGHT 7TH NERVE PALSY , GLOMUS	POSITIVE	ABSENT	DECREASED	NIL
Z1000921	42	F	FEVERX 1 MONTH,HOH,TINNITUS B/L,VERTIGOX 15DAYS	2w 1d	DISS TB,HIV POS	NORMAL	2/6	LEFT VIRAL 7TH NERVE PALSY	NEGATIVE	PRESENT	INTACT	GENEXPERT - TB POSITIVE,PANCYTOPENIA (MRI)
O20053586	53	M	RIGHT EAR PAIN ,HOH,H/O EXANTHEMATOUS FEVER X 2 WEEKS	2w	NIL	RIGHT EAR VESICLES IN EAC,CC	5/6	RIGHT 7TH NERVE PALSY, RAMSAY SYNDROME	POSITIVE	ABSENT	DECREASED	
O20027065	28	M	RTA, RIGHT EAR BLEED	2d	NIL	HEMOTYMPANUM	3/6	POST RTA RIGHT 7TH NERVE PALSY	NEGATIVE	ABSENT	DECREASED	FRACTURE RIGHT SQUAMOUS TEMPORAL BONE TO PETROUS APEX,FLOOR ME INVOLVED(CT)
O20004940	38	M	LEFT SIDED WEAKNESS AND VERTIGO	2d	DM,HIV,NEW CVA- RIGHT POST CIR STROKE	NORMAL	4/6	RIGHT MALIGNANT OITIS EXTERNA, RIGHT 7TH NERVE PALSY	NEGATIVE	ABSENT	DECREASED	RIGHT ACUTE PARAMEDIAN LOWER PONS AND UPPER MEDULLA STROKE SITE(CT)(MRI)
O19041323	70	M	RIGHT EAR PAINX 6 MONTHS	6m	DM/SHT	R EDEMATOUS EAC WITH GRANULATION	6/6	RIGHT MALIGNANT OITIS EXTERNA	POSITIVE	ABSENT	DECREASED	ALTERED MARROW SIGNAL IN CLIVUS,PM TEMPORAL BONE,OCCIPITAL CONDYLE,JUGULAR FORAMEN; 7-12 CN PALSY(MRI)
O19074520	45	M	PAROTID SWELLING ,DEVIATION MOUTH TO LEFT X 3DAYS	3d	RIGHT MRM/DM/SHT	DISCHARGE IN RIGHT EAC	5/6	LEFT PAROTITIS , LEFT 7TH NERVE PALSY	NEGATIVE	ABSENT	DECREASED	USG - PAROTITIS
O20091970	50	M	DEVIATION OF MOUTH TO LEFT X 2DAYS	2d	DM/COVID POSITIVE STATUS	NORMAL	3/6	RIGHT POST VIRAL 7TH NERVE PALSY	POSITIVE	ABSENT	DECREASED	MRI BRAIN NORMAL
O19087543	36	F	DEVIATION OF MOUTH TO LEFT X 5DAYS	5d	SHT	NORMAL	3/6	RIGHT BELL'S PALSY	NEGATIVE	ABSENT	DECREASED	MRI BRAIN NORMAL
O20087164	54	F	DEVIATION OF MOUTH TO RIGHT X 6DAYS	6d	DM	NORMAL	4/6	LEFT BELL,S PALSY	POSITIVE	ABSENT	DECREASED	LYM - DEC
O20007654	43	F	DIFFICULTY IN CLOSING RIGHT EYE X 7DAYS	1w	DM	NORMAL	5/6	RIGHT BELL'S PALSY	POSITIVE	ABSENT	DECREASED	MRI BRAIN NORMAL
O21007592	28	M	DEVIATION OF MOUTH TO LEFT X 4DAYS	4d	NIL	NORMAL	3/6	RIGHT BELL'S PALSY	NEGATIVE	ABSENT	DECREASED	MRI BRAIN NORMAL
O21023094	72	M	DEVIATION OF MOUTH TO RIGHT X 8DAYS	1w 1d	SHT	NORMAL	4/6	LEFT BELL,S PALSY	POSITIVE	ABSENT	DECREASED	LYMP - DEC
O17864570	44	M	DEVIATION OF MOUTH TO LEFT X 15DAYS	2w 1d	DM	NORMAL	5/6	RIGHT BELL'S PALSY	POSITIVE	ABSENT	DECREASED	MRI BRAIN NORMAL
O21058970	50	M	VERTIGO X 20DAYS;HOARSENESS,ASPIRATION;L TINNITUS/HOH	2w 6d	NIL	REDDISH HUE BEHIND INTACT TM	2/6	LEFT 7TH NERVE PALSY , LEFT GLOMUS	POSITIVE	ABSENT	DECREASED	MRI - GLOMUS JUGULARE - ENHANCING LESION LEFT JUGULAR FOSSA; L VII,VIII,IX,X,XI,XII PALSY