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Nerve injuries – A review

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Abstract
Surgeons like to prevent nerve injuries on limit them to the mildest type; however, this is something that is often beyond the control of the health care professional. If a more damaging injury is encountered preventing the degeneration of the distal segment of the nerve is undesirable because this developmental like environment allows peripheral nerve regeneration to take place at its best inherent abilities. The goal in the early management of nerve injuries is to decide as soon as possible which injuries will profit from early repair and which should be followed.

Keywords: Nerve anatomy, Nerve injury, Management of nerve injury

Introduction
The peripheral sensory innervation of the orofacial region may be injured by oral and maxillofacial surgical and general dental procedures (eg: local anesthetic injections, 3rd molar extractions, mandibular osteotomies, dental implants, endodontic procedures, and preprosthetic surgery), trauma (eg: facial fractures) or secondary to pathologic destruction and treatment (eg: oral squamous cell carcinoma, resection). A goal for surgeons who manage nerve – injured patients should be to develop methodology that enhances one or more of the processes that allow a peripheral nerve to regrow and function again.

Anatomy of Nerve
Nerve trunk is composed of 4 connective tissue sheaths. Mesoneurium – is a sheath analogous to the Mesentery of the intestine which suspends the nerve trunk with in the soft tissue. Epineurium – is the loose connective tissue sheath that defines the nerve. Trunk and protects against mechanical stress. Epineurium can occupy from 22 – 88% of the cross sectional diameter of nerves with greater cross – sect diameter are more capable of withstanding compressive forces than tensile forces. Epineurium has an epifascicular layer, which surrounds the entire nerve trunk, and lies superficial to the fascicles, and interfascicular layer, which occupies the space among the fascicles. The vasa nervorum, longitudinal blood vessels course through the epineurium and give off branches that penetrate the perineurium to connect with the endoneurial capillaries. Perineurium – Fascicles are delineated by the perineurium, which surrounds the axons and endoneurial sheaths. It is a continuation of the pia-arachnoid mater of CNS. Composed of two layers. An outer layer of dense connective tissue and an inner cellular layer made of a multilayered continuous sheet of flat squamous cells. Its functions are active transport of certain molecules, diffusion barrier, maintaining positive pressure inside the fascicle, providing structural support for the enclosed neural tissues, if the perineurium is breached, the intrafascicular million is altered and conduction disturbances may result, an intra-fascicular hemorrhage or edema results in an increased intra fascicular pressure that can alter axonal conduction. Three fascicular pattern have been described: Monofascicular pattern: is composed of one large fascicle surrounded by concentric layers of perineurium and epifascicular epineurium. The facial nerve at the stylomastoid foramen has a monofascicular pattern or digofascicular pattern with only a few fascicles [1]. Oligofascicular pattern: is characterized by 2 to 10 rather large fascicles, each surrounded by perinueurium and all held together by epifascicular and interfascicular epineurium. Polyfascicular pattern: consists of more than 10 fascicles of different sizes but with a prevalence of small fascicles. Both the IAN and lingual nerves have polyfascicular patterns. The polyfascicular pattern has a large amount

of nonfascicular tissue, which increases likelihood of misalignment of the fascicles during coaptation and epineurial fibrosis following injury. Endoneurium – Individual nerve fibers and their Schwann cells are surrounded by the endoneurium, composed of an external layer of collagen fibers and endoneurial fibroblasts and an internal layer of basal lamina and endoneurial capillaries. Nerve fiber is the functional component of the peripheral nerve. It is composed of axon; Schwann cell and myelin sheath in myelinated nerve fibers. Axon: is an extension of a neuron and can be characterized by morphology, conduction velocity and function. A-alpha fibers are the largest myelinated fibers and range in diameter from 7-16 μm conduction velocity – 70-120 m/sec. A-beta fibers are next largest myelinated axons. The diameter → 6-8 μm. its conduction velocity → 30-70 m/sec. Sensibility of touch is attributed to these axons. A- Delta fibers are smallest of the myelinated fibers. Diameter is 205-4μm. Conduction velocity is 0.5-2m/sec [2]. It transmit stimuli encoded for slow or second pain, temperature and efferent sympathetic fibers. Schwann cell is essential for axon survival, whether myelinated or unmyelinated. It surrounds several unmyelinated axon to form well-defined units that are interspersed among myelinated fibers is a fascicle. In myelinated nerve fibers multiple layers of the cell membrane of the Schwann axon to form a myelin sheath over a definable segment of axon, the inter node.

Terminology

Allodynia: Pain due to stimulus that does not normally provoke pain. Analgesia: Absence of pain in response to stimulation that would normally be painful. Anesthesia: Absence of any sensation in response to stimulation that would normally be painful 02 nonpainful. Anesthesia dolorosa: Pain in an area or region that is anesthetic. Causalgia: Burning pain, allodynia and hyperpathia after partial injury of a nerve. Dysesthesia: An unpleasant (usually painful) abnormal sensation, whether spontaneous or evoked. Special cases of dyesthesia include hyperalgesia and allodynia. Hyperesthesia: Increased sensitivity to stimulation, excluding the special senses. Hyperalgesia: A painful syndrome, characterized by increased reaction to a stimulus, especially a repetitive stimulus, as well as an increased threshold. Hypoesthesia: Decreased sensitivity to stimulation, excluding the special senses. Hypoalgesia: Diminished pain in response to a normally painful stimulus. Paresthesia: An abnormal sensation, whether spontaneous or evoked. It should be used to describe an abnormal sensation that is not unpleasant and dysthesia an abnormal sensation considered to be unpleasant [3].

Nerve Injury

Degeneration and Regeneration: When nerve is injured there are responses distal to the injury, at the site of injury, proximal to the site of injury and within the CNS. Neuron undergoes hypertrophic changes that begin on the third or 4th day following injury to the axon and peak between the 10th and 20th day. Total RNA content of the cell increases as the cell increases in size RNA migrates to the outer edges of the cell and break up in to smaller particles. Neuron is beginning an anabolic proteosynthetic state that is maintained as large as there are regenerative efforts – up to many years. Chromatolytic hypertrophic changes are more pronounced in more proximal injuries. Sometimes the neuron cannot meet these metabolic demands and cell death occurs. After regeneration is complete and conduction maturation has occurred, the neuron return to normal size and electrical activity. Proximal nerve trunk: 1hr after laceration of a nerve there is marked swelling proximally as far as 1 cm. The cross-sectional also may increase the three times normal and swelling persists for 1 week or more before slowly subsiding. By the 7th day there is vigorous sprouting of axons. Each axon may have as many as 50 collateral sprouts. It is not until the 28th day that axons cross the point of injury and the forty-second day before a sizable no. of axons occupy the distal segment [4]. Site of injury: Within hours of injury there is a proliferation of macrophages, perineurial fibroblasts, Schwann cells and epineurial fibroblasts. By the 7th day the Schwann cell is clearly the most active cell and assumes a phagocytic function of debridement. The Schwann cell response is proportional to the security of the injury. Distal nerve trunk: It undergoes Wallerian degeneration in preparation for the arrival of sprouting axons. Wallerian degeneration is initiated because all distal neural elements die. By 7th post injury day the majority of neural elements break down, facilitated by digestive enzymes present in the axons. Majority of cellular debris has been phagocytosed by Schwann cells by 21st day. At 42 days debridement is complete and parts of the fascicular anatomy persist. Endoneurial tubules either shrink or collapse. Tubules (bands of Bungner) composed of Schwann cells surrounded by collages guide axons toward the distal nerve trunk. Initially several axons may occupy a single distal tubule. But axonal numbers decrease during regeneration. With the arrival of new axons, Schwann cell again increase metabolic activity new myelin is layered around the axons by Schwann cells. New myelination is never as good as the original and the axon diameters are smaller. The endoneurial tubes are smaller in diameter and the nodes of Ranvier shorter, resulting in slower conduction velocities. The rate of axon regeneration varies during the process of repair. There is an initial delay until the axon has crossed the site of injury. The rate of axon advancement increases to 1 to 3 per day, followed by another slowing and delay as axons form new connections with sensory organs [5].

Etiology of Nerve Injuries

Meyer listed the procedures most commonly associated with trigeminal nerve injuries seen the most common procedure associated with a trigeminal nerve injury was the removal of impacted teeth followed by osteotomies, fractures and dental implants, alveolar ridge augmentation with hydroxyapatite, root canal therapy, tumor resection and genioplasty other miscellaneous causes of trigeminal nerve injuries included salivary gland excision, vestibuloplasty. Biopsies, gunshot wounds, and bony cyst excision. Some of the etiologic factors such as trauma and tumor surgery are unpreventable. A nerve may be injured by open wound such as cuts, gunshots. Closed injury as by external pressure, compression, stretch, fracture and chronic irritation. Effect of nerve injury Depends on the type of nerve, pure motor, pure sensory or mixed. Motor effect, Paralysis of muscles**,** Atrophy of muscles and replacement of muscle fibers by fibro fatty tissue**,** Due to free over play of intact antagonist muscles. A change in position of joint or attitude results. Reaction of degeneration: this means certain electrical changes in muscles supplied by injured nerve. Normally a muscle responds to faradic and galvanic current and cathodal closing contraction (KCC) is more than anodal closing contraction (ACC). After injury muscles responds only to galvanic current for about a week and after 10 days there is no response to either galvanic or

faradic current. And there is polar reversal i.e. ACC is more than KCC. Fibrillation: In partially injured nerves or during recovery state, the affected muscle exhibits irregular twitching contractions known as fibrillations. Sensory effects includes anesthesia where there is loss of pain, touch, pressure & temp. Sensation**,** Pain – due to the partial injury – also known as causalgia, Pseudomotor effects**,** Hyper-hydrosis – increased sweating due to partial injury**,** Anhydrosis – absence of sweating and complete dryness due to total nerve injury. Vasomotor effect **t**here is vasodilatation in the denervated skin due to paralysis of vasoconstrictor fibers so that skin is red and warm. There is loss of both superficial and deep reflexes if the injures nerve is a part of reflex arc. Nutritional changes like skin become smooth and inelastic loses pits and wrinkles, decreased resistance to trauma leading to indolent ulcers**,** loss of subcutaneous tissues**,** loss of hair**,** and distortion of nails. The squeal is contractures, ankylosis of joint**,** decalcification of bones [6].

Classification of Nerve Injuries

The appropriate and logical management of nerve injuries is based on the accurate description and classification of the nerve injury. A variety of classification schemes have been proposed, the most common of these being the Seddon and Sunderland Classification.

Seddon Classification: Described 3 types of nerve injuries – based upon the severity of tissue injury, prognosis for recovery and the time frame for recovery. Neuropraxia, Axonotmesis and Neurotmesis. Neuropraxia is a common block resulting from a mild insult to the nerve trunk. There is no axonal degeneration. Sensory recovery is complete and occurs in a matter of hours to several days. There is physiological paralysis of conduction and the nerve fibers otherwise intact. The cause of injury is usually a stretch or distortion – there may be segmental demyelination. The magnitude of sensory deficit is usually mild and consists of a paresthesia with some level of stimulus detection but poor discrimination and disturbed stimulus interpretation. Axonotmesis is a more severe injury than neuropraxia. Afferent fibers undergo degeneration but the nerve trunk is grossly intact with variable degrees of tissue injury. There is rupture of nerve fibers in an intact sheath. The cause of such as injury is an abnormal stress such as in fracture, traction injuries, contusion of nerve with extensive hemorrhage into its sheath and finally compression by tourniquets, splints etc or by scar tissues. Sensory recovery is good but incomplete. The time course for sensory recovery is dependent on the rate of axonal regeneration; it is usually several months. The sensory deficit is usually a severe paresthesia. The incidence of neuropathic regeneration is very low, but a neuroma in continuity may rarely develop. Neurotmesis *i*s a severance of the nerve and is the most severe injury in the classification. There is partial or complete division of nerve fibers and sheath caused by penetrating wounds. Sensory recovery is not to be expected when the nerve courses through soft tissue. Nerves that travel within a canal may exhibit some degree of sensory recovery because of the guiding influence of the canal. The incidence of neropathic regeneration is high with this type of nerve injury. The sensory deficit of neurotemesis is either an anesthesia or a dysesthesia [7].

Sunderland Classification*:* The Sunderland classification is based on the degree of tissue injury, there is considerable overlap with the seddon classification. First-degree injury Sunderland classification is the same as the neuropraxia in the Seddon classification. Axonal conduction is temporarily blocked and all the tissue components of the nerve are intact. Axonal conduction blockade may be the result of ischemia or mechanical demyelination. There are 3 types of first-degree nerve injury based in the proposed mechanism of conduction block. First degree, Type I injury results from nerve trunk manipulation. Mild traction or mild compression such as occurs during sagittal split osteotomy, Inferior Alveolar nerve repositioning or Lingual nerve manipulation during sialadenectomy of the sublingual or submandibular gland. The mechanism of the conduction block is presumed to be anoxia from interruption of the segmental or epineural blood vessels, but there is no axonal degeneration or demyelination. Normal sensation or function returns within several hours (less than 2 hours) following the restoration of circulation. A first degree, type II injury results from moderate manipulation, traction or compression of a nerve. Intrafascicular edema from trauma of sufficient magnitude to injure the endoneurial capillaries results in a conduction block. Normal sensation or function returns within 1 –2 days following the resolution of intrafascicular edema which generally occurs within 1 week following nerve injury. First degree, type III nerve injuries results from severe nerve manipulation, traction or compression. Pressure on the nerves causes segmental demyelination or mechanical disruption of the myelin sheaths. Sensory and functional recovery is complete within 1 –2 months. The psychophysical response to this type of injury is paresthesia. Microconstructive surgery is not indicated for first-degree nerve injuries unless there is a foreign body irritant. The second, third and fourth degree injuries of Sunderland overlap with Seddon's axonotmesis. The afferent or efferent fibers (axons) damaged the undergo degeneration. The remaining tissue components of the nerve trunk, endoneurium, perineurium and epineurium remain intact. The signs and symptoms usually associated with second-degree injuries include a generalized paresthesia with a localized area of anesthesia. Surgical intervention is not necessary unless there is a foreign body irritant [8].

In third degree injury the intrafascicular tissue components, the axons and endoneurium are damaged. Generally there is some degree of intrafascicular fibrosis blocking the path of degenerating axons. This results in fair to poor sensory recovery, with some degree of persistent paresthsia, synhestheisa and increased two-point discrimination. The incidence of neuroma in continuity is low because the perineurium and epineurium remain intact. The severity of the sensory disturbance is directly related to the severity of the mechanical insult. The signs and symptoms can range from paresthesia to dysthesia to anesthesia or any combination. These injuries must be monitored carefully, and surgical intervention must be dictated by the nature of the sensory disturbance the recovery pattern, the presence of foreign body irritant and suspected severity of the injury. In fourth degree injury fascicular disruption is the characteristics of this injury, the perineurium, endoneurium and axons are all damaged. The incidence of enuropathic sensory impairment is high because of extensive internal fibrosis and only the epineurium remains intact. The prognosis for sensory recovery is poor. Sequelae of IV degree injury include anesthesia, dysesthesia, synesthesia and severe paresthesia. Generally requires surgical intervention to upgrade the injury and improve the prognosis for favorable sensory recovery and to minimize the potential for neuropathic recovery. Sunderland's fifth degree nerve injury is characterized by transection or rupture of the

entire nerve trunk. This results in loss of nerve conduction at the level of the injury and within the distal nerve segment. Intraosseous fifth degree injury may undergo spontaneous recovery of some degree of sensibility of this canal is intact. Soft tissue of fifth degree nerve injuries have poor prognosis for recovery and require surgical adaptation and coaptation. The prognosis depends on the nature of the injury, as well as on local and general factors. The sixth degree injury pattern was added by Macjinnon and Dellon, to describe the combination of Sunderlands' 5 degrees of injury. Within the same nerve trunk, some fascicles may exhibit normal function and others will have various degree of nerve injury. [First through fifth degree injuries]. This injury pattern presents the surgeon with the greatest challenge. The first, second and third degree nerve injuries will undergo spontaneous recovery superior to that provided by surgical neurography or grafting, whereas the fourth and fifth degree nerve injuries require surgical reconstruction [9].

Physiologic Conduction Block -Type "A" conduction block. The pathophysiologic basis for a type "A" conduction block is intraneural circulatory arrest or metabolic (ionic) block with no nerve fiber pathology. This conduction block is immediately reversible. This type of injuries best managed by therapies that improve or restore circulation to the nerve trunk such as sympathetic blockade, decompression or the use of agents to decrease edema or reverse vasospasm. Intraneural edema resulting in increased endoneural fluid pressure or metabolic block with little or no nerve fiber pathology is the basis for type "B" conduction block. This type of conduction block is reversible within days or week. Therapies to decrease edema and promote venous drainage are most appropriate in managing the type "B" conduction block [10].

Symptomatic Classification: Patients with sensory disturbances following nerve injury present with subjective complaints of numbness, which can be broadly classified, into anesthesia, paresthesia or dysesthesia. A thorough clinical examination is necessary to describe and classify the sensory disturbance resulting from nerve injury. First it is necessary to understand the components of sensibility. Sensibility id the sum of stimulus detection, stimulus localization and stimulus interpretation or perception. Stimulus detection is simply the ability to determine whether or not something is contacting the skin or mucosa. The ability to accurately and precisely locate the point or area of stimulus contact is stimulus localization. The ability to describe the stimulus, such as hot, pricking, pressure, tickles and so on stimulus interpretation or perception. Some examples of abnormal stimulus perceptions include pulling, swollen, tight and tingling. Anesthesia is the complete lack of any stimulus detection and stimulus reception, including mechanoreceptive and nociceptive stimuli – this is usually associated with a severe injury of the nerve interrupting the integrity of the axons. Sensory recovery following anesthesia is slow and unpredictable. The timing of the surgical intervention must be balanced against the potential for distal nerve trunk atrophy if the endoneural tubes are not invaded by regenerating axon in a timely pattern. Depending on the mechanism and circumstances of injury, early surgery may be indicated. Acute or early repair is indicated for observed transection injuries of nerves located within soft tissue, observed transection injuries of nerve located within a bony canal when the canal has been disrupted (Eg. Laceration of inf. alveolar nerve during sagital split osteotomy). Anesthesia that persists for 3 months with a high index of suspicion for severe nerve injury and poor prognosis of spontaneous sensory recovery (Crush injury of a nerve located within either soft tissue or bony canal) presence of a foreign body irritant with a poor or uncertain prognosis for spontaneous regeneration and sensory recovery (endosseous implant compressing the inf. Alveolar nerve). Paresthesia is an alteration in sensibility in which there is abnormal or occasionally normal stimulus detection and stimulus perception that may be perceived as unpleasant but is not painful. Stimulus detection may be normal, increased, or decreased and may affect wither mechanoreception or nociception. Decreased touch and pressure stimuli detection is called hypoesthesia, whereas increased perception of these stimuli is called hyperesthesia. Decreased nociceptive stimuli detection is called – hypoalgesia. Increased perception is called – hyperalgesia. Patient may additionally complaint of a constant abnormal background perception, which they describe as numb, tingling, itching, swollen, fat tight, heavy, drawing and son on. These abnormalities of stimulus detection and perception may be the result of conduction disturbances. Ischemia and alterations in protein transport occur, along the axon to the peripheral receptor, and not necessarily from disruption of axons. Difficulty in quickly and accurately localizing the point of stimulus of application is called synesthesia. Synesthesia is probably the result of misdirection of the axons during the process of degeneration and is a common finding following neurography. Protopathia is the inability to distinguish or differentiate between two distinctly different stimuli, such as sharp and dull. Distal anatomy is not a concern with paresthesia therefore no urgency for surgical exploration and repair that there is with anesthesia. If there is a foreign body irritation causing paresthesia surgical intervention is indicated to remove the foreign body.

Dyesethesia is the alteration in sensibility in which there is abnormal stimulus detection and stimulus perception may be perceived as unpleasant and painful. Dysesthesia shares all the features of a paresthesia but has the additional features of pain, which may be spontaneous, or triggered. Allodynia is a specific type of dysesthesia characterized by a sharp, first pain perception elicited by a light touch stimulus. Hyperpathia is another type of dysesthesia characterized by a dull, second pain elicited by a pressure stimulus. The pain of hyperpathia lingers or has an after make that persists even after the pressure stimulus is removed. Alodynia and Hyperpathia are signs associated with neuromas, entrapment, compression and sympathetically maintained pain. Initially dysesthesia must be managed non-surgically with supportive therapies to prevent or minimize sensitization of wide dynamic range neurons that result in central mediated pain or sympathetically mediated pain. If the mechanism of pain is determined to be due to a peripheral neuropathy such as neuroma, entrapment or compression urgent surgical intervention is indicated to upgrade the nerve injury and convert dysesthesia to a parasthesia with an improved prognosis for sensory recovery [11].

Anatomic Classification: Nerve injuries can be classified anatomically as intraosseous and soft tissue. This distinction is important because the management and prognosis for one differ from the other.

Intraosseous Nerve Injury: Osseous canals provide protection from mechanical trauma unless the integrity of the canal is breached. The closed space of the osseous canal

predisposes the enclosed nerve trunk to compartment syndrome, which starts a cascade of events in the acute phase: compression increased vascular permeability, edema, increased endoneurial fluid pressure, ischemia and nerve fiber dysfunction. The chronic effect of compression are fibreblast invasion, scarring, fiber deformation – degeneration and nerve fiber dysfunction. Generally no acute surgical intervention is necessary for mechanical injuries if the canal remains intact and if the nerve is not compressed by a foreign body or edema within the canal. Foreign bodies such as implants, tooth roots or displaced bony fragments must be removed acutely to alleviate compression and prevent an unfavorable cascade of events results from compression. Chemical injuries are unique and generally require acute surgical intervention to remove or neutralize the agent followed by delayed micro reconstructive nerve surgery as indicated by progressive assessment of sensory recovery. The environment of a nerve has a bearing on the suceptibility to injury prognosis for recovery and timing of surgical intervention osseous canal provide protection from mechanical trauma unless the integrity of the canal is breached, conversely closed space of the osseous canal predisposes the enclosed nerve trunk to compartment syndrome, which starts as cascade of events in the acute phase, compression, increased vascular permeability edema and nerve fibre dysfunction the chronic effects of compression are fibroblast invasion, scoring fibre deformation degeneration and nerve fibre dysfunction [12].

Soft Tissue Nerve Injury: Nerves located within soft tissue are not afforded the protection from mechanical trauma that are intraossesous counterpart are within guiding influence of an osseous canal, lacerations and transactions of nerve located within soft tissue are more likely to form neuromas (either symptomatic or asymptomatic) and are less likely to undergo spontaneous regeneration because of the formation of the scar tissue between the injured ends. The lingual nerve because of tissue close proximity to the lingual cortex of the mandible seems to be very susceptible to entrapment injury that prevent guiding of the nerve [11].

Histopathology Classification: Neuromas are characterized by disorganized microsprouting and formation of a disorganized mass of collagen and randomly oriented small neural fascicles. Peripheral neuromas can be classified as amputation or stump, central, and eccentric. The amputation or stump neuroma is a knobby, disorganized mass of axons and collagen associated with the proximal nerve stump and completely separated from the distal nerve stump. This type of neuroma is the result of a Sunderland fifth-degree injury. The cental or neuroma in continuity is a fusiform expansion or fibrotic narrowing of the nerve with variable degrees of fasciculoar disruption and disorganization. There is no breach of the epineurium. This type of neuroma is the result of a Sunderland fourth-degree injury or fifth-degree injury in which continuity between the proximal and distal stumps was re-established. The lateral entrapment neuroma of the lingual nerve is a specific type of neuroma in-continuity. This neuroma is characterized by a neurofibrous union between the epineurium and lingual peristeum of the mandible usually in the third molar region. The neuroma in-continuity can also result from scarring at the site of microsurgical coaptation. There are two types of eccentric neuromas lateral exophytic and stellate neuromas of the inferior alveolar nerve. The lateral exophytic neuroma is an outgrowth of axons and collagen, forming a terminal knob-like structure on an otherwise intact nerve. Only a few superficial fascicles are disrupted because of an incomplete transection of the nerve or a poor coaptation of the distal and proximal nerve stumps. There is a recognizable breach of the epineurium at the site of the lateral exophytic neuroma. The stellate neuroma has two or more branches at the site of injury ending in adjacent soft tissue or mucosa. This type of neuroma has been identified with the inferior alveolar nerve in the third molar area. These collateral branches penetrate the lingual or buccal cortex and end in the adjacent soft tissue. In contrast to the lateral exophytic neuroma, the epineurium is intact and the branches terminate in soft tissue. Stump neuromas are managed by excision and preparation of the nerve ends, which are trimmed until a definite fascicular pattern can be identified and axonal mushrooming is observed at the proximal stump. A direct neurorrhaphy or graft is performed as indicated. The neuroma in-continuity is most difficult to manage surgically. The clinical appearance of the neuroma in-continuity is usually an enlargement or bulging of the nerve with a relatively normal or slightly enlarged proximal trunk diameter and reduced distal trunk diameter. The problem is to determine whether the sensory deficit is due to epineural fibrosis or fascicular disruption, or both. The first step is to perform an epifascicular epineurotomy and palpate the nerve to determine whether there is interfascicualr scarring. If there is, an epifascicular epineurectomy and interfascicular epineurectomy are performed. The nerve is again palpated to determine whether there is intrafascicular scarring. If there is, the scarred segment is excised and the nerve ends prepared for neurorrhaphy or graft reconstruction. If the instrumentation is available, evoked action potentials can be recorded intraoperatively across the neuroma or site of injury to determine whether or not there is axonal continuity. This is a great help in deciding whether or not to excise the damaged segment. Generally, if the sensory deficit is that of paresthesia, neurolysis is sufficient. Excision and neurorrhaphy or graft reconstruction are generally indicated for anesthesias and dysesthesias. FIBROSIS: There are various degrees of reactive fibrosis that occur following trauma to a nerve. These have been classified by Millesi. Type a fibrosis involves the epifascicular epineurium and is associated with a good prognosis for recovery. Type B fibrosis involves the interfascicular epineurium. The prognosis is guarded and depends on the original damage. Internal neurolysis is indicated for the surgical management of type A and type B fibrosis. Type C fibrosis extends into the endoneurium and has a poor prognosis. Type C fibrosis requires excision of the scarred segment and neurorrhaphy or graft reconstruction. Types A, B, and C fibrosis can be used as modifies with the Sunderland classification: IA and IB, IIA and IIB, IIIA, IIIB, and IIIC. Type N fibrosis is a Sunderland class IV injury in which epineural connective tissue maintains continuity and is infiltrated by a neuroma. Type S fibrosis is a Sunderland class IV injury that is maintained only by scar tissue band and neurorrhaphy or graft reconstruction [12].

Classification of damage by location of the reactive fibrosis:

Designation	Location	Prognosis
A*	Epifascicular epineurium	Generally good
B*	Interfascicular epineurium	Depends on original damage
C*	Endoneurium	Poor
N	In a Sunderland class IV injury, the epineural connective tissue that maintains continuity can be infiltrated by a neuroma (IVN)	Poor
S	Continuity in class IV injury maintained only by scar tissue	Poor

Pathophysiologic Classification

Compression: Compression injury to a peripheral nerve may produce a nerpoathic pain syndrome. The acute response of compression is inflammation and edema. The first stage in the pathogenesis of nerve compression is blood nerve barrier changes resulting in subperineural and endoneurial edema. This leads to connective tissue changes that include perineurial and epineurial thickening. Next stage in pathogenesis is the localized nerve fiber changes. Some fibers within he nerve trunk function normally where as others will exhibit segmental demyelination. As the degree or duration of compression increases, Wallerian degeneration will be apparent. The peripheral fasciles and nerve fibers will be more affected first where as more centrally located fascicles and nerve fibers may be spared. The mechanism of compression injuries includes both mechanical deforming forces and ischemic factors.

Compartment Syndrome: The nerve injury associated with compartment syndrome is similar to that seen with nerve compression except that the effects are largely due to ischemia of the nerve caused by diminished flow within the compartment. The compartment syndrome may apply to the inf. alveolar, descending palatine and infraorbital nerves. Increased venomous pressure results from increased local tissue pressure, such as from edema and inflammation of the nerve trunk. This leads to a decrease in the local arterioneous gradient diminished blood flow, decreased oxygen delivery to the nerve. The reversibility of these changes will be determined by the amount of tissue pressure and the duration of the pressure. As with the compression injury, the large A β nerve fibers are most susceptible to the ischemic changes. Clinically the first sign is abnormal vibration and touch perception. The management is by – immediate decompression of the compartment and VSE of pharmacological agent to alleviate inflammation and edema of the nerve trunk, such as steroids and anti-inflammatory drugs.

Stretch Injury: Stretch or fraction injuries truly demonstrate a three dimensional injury pattern. The degree of injury varies not only from fascicle to fascicle but also in a longitudinal manner along the length of the nerve. Surgical exploration of stretch injuries mandates exposure of a considerable length of nerve so as not to miss a damaged area. The sequence of changes associated with nerve stretch is not completely understood. Sunderland feels the sequence of tissue rupture begins with axonal rupture (second-degree injury) followed in sequence by rupture of the endoneurial tubes (third-degree injury), perineurium (fourth-degree injury), and finally endoneurial rupture (fifth-degree injury). Early surgical exploration of a stretch injury is warranted to establish the diagnosis and rule out an avulsion but surgical reconstruction should be delayed in all cases. Early reconstruction of a stretch injury may result in missing or "skipping" a lesion, unnecessarily exiting a damaged segment or failing to excise a damaged segment [13].

Transection, Laceration, Repture and Avulsion: This constitutes Sunderlands 5th degree injury. Recovery depends on the approximation and co-adaptation of the nerve ends without tension. The prognosis depends on the nature of the injury and local factors. Transection and laceration injuries are generally associated with less tissue destruction than rupture or avulsion injuries and therefore have a better prognosis. The scar tissue within the between the nerve ends interferes with the advancement of the axonal growth cone. Those factors that minimize scar formation will improve the prognosis. The prognosis for Sunderland fifth-degree injuries is better for intraosseous nerves than for soft tissue nerves. Surgical reconstruction of infraosseous nerve should be delayed when the neuro vascular canal is intact. The reconstruction should be predicated on the functional recovery of the nerve as determined by sequential sensory evaluations. Surgical reconstruction of soft tissue nerve should be performed early because the prognosis is poor and the local factors unfavorable [12]

Chemical Injuries: Most often the result endodntic therapy dry socket packing or chemical neurolysis. Very little information is available regarding the pathophysilogy of these injuries. Some of the offending agents include eugenol, alcohol, phenol, and paraformaldehyde-containing endodontic filling materials. The greatest variable is the depth of intraneural penetration of any of these chemicals. The initial response following exposure of a nerve to a chemical is inflammation. This may initiate a compartment syndrome for intraosseous nerves. The severity of fibrosis and nerve fiber dysfunction will depend on the duration of exposure, depth of intraneural penetration, and toxicity of the chemical. The management should begin with identification of the chemical and determination of the neurotoxicity. Chemical exposure of an intraosseous nerve should be managed as a compartment syndrome. Surgical reconstruction of chemical nerve injury should be delayed because of the variability and unpredictability of the injury pattern [13].

Nerve Injection Injury: Unexplained sensory disturbances following an intra-oral surgical procedure, especially odentectomy may be blamed on the local anesthetic injection. Histologic studies in animal shows that needle denetration to an nerve trunk causes minimal nerve injury and results in no long lasting alteration histologic or physiologic character of the nerve However, the intraneutral injection of drugs and chemical does result in severe and irreversible nerve damages. Recent evidence suggests that enzymatic hydrolysis of L.A. agent into a drug metabolite and alcohol may cause a persistent neurosensory deficit. Any symptoms of paresthesia when injecting the awake patient should alert the physician that the needle may have penetrated the nerve. The needle should be immediately withdrawn until the paresthesia has subsided. The diagnosis of an infection injury relies on the history. Generally the patient may complain of severe immediate pain that radiates into the sensory field of the nerve being blocked

Central Neuropathy: Nerve injuries may results in debilitating central neuropathies such as a typical facial pain and pain resulting from deafferentation. The diagnosis of the central neuropathies can be quite difficult but extremely important because surgical reconstruction of the injured nerve rarely offers improvement and in some instances may exacerbate the pain. No single therapy has proved effective in managing this type of injury. Supportive measure include – antidepressants, anti-convulsants, transcutaneous neurostimulation, physical therapy, biofeedback, acupuncture, relaxation therapy, psychotherapy and management in the setting of a comprehensive multidisciplinary pain clinic.[14]

Clinical Evaluation of Nerve Injuries

Basic clinical neurosensory examination of the TN consists of 4 tests: Static light touch, Brush directional discrimination, Two-point discrimination, Pin pressure nociceptive discrimination. The patient is comfortably seated in the semireclined position in a quiet, temperature-controlled room [14].

Static light touch detection: Assesses the integrity of the Merkel cell and Ruffini ending, which are innervated by myelinated afferent axons of 5-15 μm in diameter (A-beta). These receptors adapt slowly, and their putative sensory modality is pressure. Test is performed using Weinstein – Semmes filaments, which are nylon filaments of identical lengths but variable diameters mounted in plastic handles. Keep the filament vertically oriented and above the cutaneous area to be stimulated. Slowly place the tip of the filament on the skin continue the pressure without movement for 1 second and 1 to 1.5 sec slowly raise the filament. If patient reports touch during the application period, discard this approximation trial and select a filament two to three steps lower on the scale and repeat the procedure for a filament to which there is no response, continue with filaments of increasingly greater stiffness until the patient responds. Record this filament value as the first ascending determination. Then select a filament two to four steps above the one felt by the patient apply this in the same fashion to the same spot on the skin. If there is no response, select filaments two steps above that one and repeat the procedure. If the patient reports 'touch' continue applying filaments of decreasing stiffness in the same manna until there is no response. Record as the descending value the last filament that was perceived by the patient [14].

Brush directional discrimination: This is a test of proprioception and assesses the integrity of large A-alpha and A-beta myelinated axons that innervate the lonceolate endings and pacini and meissner corpuscles. The putative sensory modalities for these receptors are vibration, touch and flutter. With the patients eyes closed, the least stiff Weinstein-Semmes filament detected from the static light touch thresholds or a 00 Camel's – Hair brush is gently stroked over a 1-cm area of skin at a constant rate. The patient is asked if any sensation is detected and in which direction the filament or brush moved. The correct no. of responses for the total no. of trials is recorded. It is important to note whether or not any sensation was detected. No correct responses because no sensation was detected a more ominous. Finding than no correct responses because the direction of most could not be accurately determined in spite of stimulus detection.

Two-point discrimination: This is a test of tactilegnosis which assesses the quantity and density of functional sensory receptors and afferent fibers. If sharp points are used, the small myelinated A-delta and unmyelinated C-afferent fibers of 0.5-7 μm diameter are assessed. If blunt points are used, the larger myelinated Alpha afferent fibers of 5-15 μm diameter are assessed. Campbell *et al* reported that normal measures for two-point discrimination in the TN distribution vary from 7 to 14 mm, it is considered diminished at from 15 to 20 mm and absent above 20 mm. ECG calipers or a Boley gauge works well for this test with the patient eyes closed the test is initiated with the points essentially touching. So that the patient is able to discriminate only one point. The distance between the points is increased in 2-mm increments until the patient is able to discriminate two distinct and separate points in at least four or five trials with the points widely separated, the points are moved closer together until the patient is able to discriminate only one point. Record the separation of the two patients, whether or not the patient could distinguish two points at any time and the patient's perception of the stimulus [14].

Pin pressure nociception: This test assesses the free nerve endings and the small A-delta and C-fibers that innervate the free nerve endings responsible for nociception. For this test a pressure algesimeter is used. This instrument is easily made from a no. 4 Taylor's needle and an orthodontic strain gauge (15-150 gm). Needle is applied perpendicular to the skin of an unaffected area and the force is increased over 1-2 secs until the desired level is reached, held constant for 1-2 secs and slowly removed over 1-2 sec period. First the needle is applied with sufficient force so the patient precious → pricking sensation (15-25 gm). Some force is applied to affected region and the patient is asked to choose one of 4 adjectives (touch, pricking, stinging, stabbing) that best describes his perception. The nociceptive threshold for the affected area is determined. The force is gradually increased on the affected areas until the sharpness of the pin is identical to that of the unaffected area just tested. The magnitude of force necessary to equal the sharpness of the unaffected area is recorded as the nociceptive threshold for the affected area. If there is no response at100 gm pressure, the area is considered to be anesthetic.

Thermal Discrimination: Thermal discrimination is a useful adjunctive test of sensation but is not essential. This is a test of small-diameter myelinated and unmyelinated fibers. Similar to those tested for pin pressure. Warmth sensation → A-delta fibers. Cold sensation → C-fibers. Various instrumentation available for thermal testing: thermodes and Minnesota Thermal Disks (MTD), ice, ethylchloride sprays, acetone and water. Cotton swab saturated with either ethyl chloride or acetone is applied to the skin and the patient is asked to mark on a visual analogue scale the magnitude of temperature perceived and whether or not the stimulus was painful or uncomfortable. Minnesota thermal disks (MTD) can be used for thermal discrimination. There are four disks made of copper (C); stainless steel (S) glass (G), and polyvinyl chloride (P). Copper is the coldest stimulus and S, G and P are progressively less cold. Normally, C is readily recognized as cooler than P. Most normal individuals can recognize C as cooler than G and few can recognize C as cooler than S. The difference between C and P is there order magnitude, C and G two and C and S one. Testing is performed by applying or series of paired MTD to the face and asking the patient to determine for each sever which of two MTD is cooler. There are 10 possible pairs of thermal stimuli, but for convenience only thus pairs are used; C and P; C and G; C and S. Factors influencing the timing and results of nerve repair. There are several well identified factors that

have a definite bearing on the timing of nerve repair and the results of motor and sensory returns following repair. Age: A number of authors have reported on the increased rate and quality of recovery following nerve repair in children and even in young adults, repair in children under 6 years of age there is excellent recovery of sensibility in all cases, after the age of 6 years there is a steady decrease in functional return with the two point discrimination value expressed in millimeters being about the same as the age of the patient upto 20-31 years of age the sensory recovery varied but tended to be poor above 31 years of age all the results were poor with a 2 PD near or above 30mm. Type of trauma: Gunshot wounds, high speed missile wounds and any trauma that stretches a nerve cause severe nerve injuries both at the site of wounding and at a distally from this point, this type of trauma also causes gross destruction of nerve and other tissues, which leads to scarring and a poor bed for the repaired nerve, in contrast there are the sharp knife and glass nerve injuries that occur most commonly in civilian life, sharpely divided nerves most commonly caused by glass or a knife, involve the nerves only at the site of injury and extend a few millimieters proximally and distally. Level of nerve injury: The rapidity and quality of sensory and motor function following high nerve injury is not as good as following lower injury, reasons for this are the greater loss of nerve cell mass as discussed under the heading of metabolic changes of nerve cell the more equal proportion of motor and sensory fibers at higher levels and thus the greater chances of cross innervation, particularly in the median nerve, the greater distance the axons must travel to reach muscle in high lesions, which gives more time for the denervation changes in muscle that limit restoration of function and the more destructive types of trauma that tend to occur at higher levels. Primary versus secondary repair: It can be stated that peripheral nerves severed by gunshot wounds, high speed missiles or streching are best treated by secondary repair approximately 2-3 weeks after injury, it can also be stated that peripheral nerve severed by sharp glass or knives or other well localized trauma at a lower level are best treated on the day of injury or within first ten days. Technique of nerve repair: Grabb has shown in a controlled study in primary repaired median and ulnar nerves in monkeys that funicular suture with alignment of funiculi by their size and position gave a significantly greater degree of return of motor function than did nerves sutured by epineural technique, he further showed that alignment of the funiculi on the cut ends and position was as satisfactory as aligning the funiculi by the more complex method of identifying the more motor and more sensory funiculi by their electrical properties, the ability of the surgeon performing the nerve repair undoubtedly has a direct relationship on the results, although this has not been studied in a scientifically controlled manner [15]

Management of Nerve Injuries

The first step is to classify the injury as observed or unobserved. Treatment of the observed injury may be initiated immediately, whereas the unobserved injury may need to be monitored for a period of time before definitive treatment is initiated. The timing of nerve repair may be classified as-primary, delayed primary or secondary. Primary nerve repairs are completed within hours of the injury, delayed primary repair 14-21 days following injury and secondary repair more than 3 weeks following surgery [8].

Observed Injury: Transected nerves: Sutured primarily but nerve grafted primarily. Following initial exploration or repair → weekly sensory examinations are performed for 1 month, then manly for 5 months. If neurons or neuropathic pain. First line of treatment – nerve blocks, analgesis and TENS. If pain dies not resolve → after 3-4 week treatment → microsurgical exploration and repair. Compression and stretch injury: Immediate decompression and alternation of stretch to prevent ischemic and mechanical trauma. Serial sensory examination weekly for 1 month and then once a month for 5 mos [7].

Unobserved Injuries: Offers the greatest diagnostic challenge because exact anatomic nature of injury is usually not known. Monitor area of sensory loss/disturb for 1 month. Neuroplaxia or Sunderland 1st degree injury resolves within 1st month. The monitor monthly basis after 1 mo Sunderland law 2 w and Sunderland degree show the first sign of sensory recovery within 2-5 mos. If sensation fails to improve and paresthesia persists, microsurgical exploration and repair indicated only if there is a strong suspicion of an extraneural irritant is compressing the nerve. Sunderland 2 w and 3 w degree injury rarely develops sign of dysesthesia. 4th and 5th degree injuries have poor prognosis for sensory recovery and are more likely to develop signs of dyesthesia. Dysesthesia should be managed immediately. If anesthesia persists for 3-5 months after nerve injury and this is unacceptable to the patient, then microsurgical exploration and repair are indicated. Indications for microreconstructive surgeries: Painful conditions with a peripheral locus characterized by allodynia and hyperpathia. Prevention of available post-traumatic sensory disturbances. Anesthesia that is objectionable to the patient. Surgical management of lingual nerve injuries: Injury to lingual nerve can occur during routine oral surgical procedures such as 3rd molar odontectomy, orthognathic surgery and even local anesthetic block injections, the most common procedure asociated with lingual nerve injury is the removal of impacted mandibular 3rd molars, in those instances in which the lingual nerve is positioned high in the alveolar crest and first medial to the lingual plate, the lingual nerve is particularly vulnerable to injury, cadaver studies have established that this is normal variation in position of lingual nerve is present in 10% of humans. Surgical management: Exploration and repair of the lingual nerve can only be accomplished through an intraoral approach. The incision should begin laterally at the base of the ramus and continue anteriorly to the midportion of the distal and buccal region of the mandibular 2nd molar. The incision carried around the distal aspect of the second molar to lingual side and continued anteriorly within the lingual gingival sulcus to the canine region. A periosteal elevator is then used to elevate a subperiosteal lingual flap from the medial aspect of the ramus to canine region, because of scar formation, the elvation of the soft issue flap in the region of recent 3rd molar extraction is typically difficult and requires more careful dissection, dissection of the gingiva along the posterior and lateral aspect of the 2nd molar should be avoided because this will provide a secure fixation point during closure the lingual flap is gently retracted medially to expose the lingual nerve that is located just beneath the lingual periosteum, dense scar tissue at the site of soft tissue trauma makes identification of the nerve difficult it is usually helpful to identify a normal segment of lingual nerve at a point proximal and distal to the site of injury and dissect toward the injury site. Once the nerve has been identified, gentle retraction applied to the nerve with vessel loops or umbilical tape to facilitate a complete dissection, bleeding should be addressed with a fine bipolar cautery, the integrity

of nerve is assessed with magnifying loupes, can be more assessed by incising epineurium and exposing the nerve fasicles, microdissection is then used to remove any intrafascicular scar tissue if there is physical disruption of the nerve or if a large neuroma is present, resection of the damaged segment is necessary resection of nerve tissue at the proximal and distal stumps is continued until normal, viable fascicles are observed. A tension free primary anastomosis is surgical goal. The proximal segment is exposed and mobilized posteriorly through the pterygoid space, the distal segment is mobilized anteriorly to the point where the nerve crosses the submandibular duct; if further mobilization of the distal segment is necessary then the small sensory branches of lingual gingiva may be sacrificed when maximal mobilization of the nerve has been achieved the nerve gap is then assessed. Nerve gaps measuring upto 1.5cms can usually be approximated with minimal tension following maximal mobilization. The nerve is stabilized during suturing process by using a microvascular clamp or 7-0 prolen traction suture placed within the epineurium at a point proximal and distal to the anastomosis the anastomosis of the proximal and distal stumps is now achieved by placing three or four 8-0 nylon sutures within the epineurium, sutures should not be placed deeper than the epineiurium which could cause additional intraneural scarring, if the proximal and distal segments cannot be reapproximated without tension, then an interpositional graft is indicated, the graft length should be several milimeters larger than the size of the gap to assure that a tension free repair is achieved. The lingual flap is repositioned against the mandible and sutured in place, the first and most important closing suture is placed at the distal aspect of the second molar in the lateral mucoperiosteum that has not been mobilized. This secures the lingual flap and prevents it from overdoing the occlusal surface of the posterior mandibular teeth. The sutures in the dentate portion of the mandible should be placed interdentally through the dental papilla, although patients can quickly resume normal activity following this procedure, they are instructed not to open their mouth maximally for 2-3 weeks so that tension across the suture line can be minimized [13].

Surgical Management of Inferior Alveolar Nerve Injuries

Etiology and Incidence: The goal is to present different methods of approaching repair of the inferior alveolar nerve after injury prior to implant placement by nerve repositioning. Data for incidence of inferior alveolar nerve injury suggest an overall risk of 0.5% to 5% in most cases the injured nerve recovers spontaneously, but the rate of permanent injury ranges from 0 to 0.9%. The most importnat consideration in assessing the risk of injury is the association of the roots of the impacted tooth with the inferior alveolar canal considerations of the usual two dimensional nature of the radiographic interpretation has led to few guidelines to help the surgeon better inform the patient on the likelihood of post-operative paresthesia, the usual complication, or course, anesthesia due to severance of the neurovascular bundle. Surgical approaches: There are three potential approaches to the IAN: one extraoral and two intra oral methods (one through the socket itself and the other a sagittal split of the mandible to gain accesses. The use of surgical microscope or high power loupes is highly recommended. Going through the socket represents the most direct approach its main advantage would be during the initial procedure. Thus an IAN bundle runing through roots would manifest as an “object on a string” using the same approach as for the odontectomy, the tooth might be split to permit the nerve bundle to escape its anatomy it would be difficult to attempt to surgically sever and reanastomose a nerve to free such a tooth. A sagittal split type of approach was advocated early in the course of nerve repair because of the ability of this method to expose not only the IAN at its area of primary injury, but also the distal nerve extent it was originally described using the transoral approach. The proximal nerve exposure was less of a problem, but exteriorization of the IAN distal to the third molar socket is what made this method attractive. Freezing the distal nerve, including the mental nerve, added considerable surgical length, thus aiding in the primary repair of the nerve. The problem with this approach was its overall morbidity and difficulty interestingly, this is the equivalent of gaining length of the lingual nerve by dissection of the proximal nerve, with the limiting factor being the branch to the lingual gingiva. One very good indication for sagittal approach or other transoral method was for the repositioning of the IAN in implant cases in which the length of the implant and its potential position put the nerve at risk. The indications for this technique have lessened due to - (1) The use of computerized imaging method to better describe the mandible in 3 dimensions, (2) The use of improved shorter implants and (3) The evidence that nerve repositioning carries with it the same incidence of paresthesia as posterio implant placement without nerve repositioning about 6%. An extra-oral approach for IAN is simple, direct and gives good access. Direct access to the injured nerve is achieved by cutting out a cortical window of the bone after a standard Risdon approach to the mandible. The best method uses a power drill to outline the area, taking the cut down to bleeding medullary bone, small curved osteotomes are then placed at an angle into the cut, and the separation of inner cortex from medullary bone is then carefully performed. Once the window of bone is lifted free, the marrow bone is dissected carefully using a small straight dental curette experience helps in identifying the canal and freeing both the pathology and normal nerve present, the actual extent of the cortical window can be enlarged as needed. The bone overlying the canal is moved with curettes and the bundle exposed [14].

Surgical Treatment of Maxillary Nerve Injuries: The infraorbital nerve: The oral and maxillofacial surgeon is likely to encounter patients with nonpainful altered sensation or dysesthesia secondary to injury to the infraorbital nerve, a distal brand of the second division of trigeminal nerve. Patients may note abnormal sensations, most commonly in cheek and upper lip, typical sensory complaints include numbness, tingling, becoming, itching, crawling, swelling, shocking and temporary or persisatent pain. Behavior complaints might relate to undesirable changes in kissing, speaking biting, chewing or drinking. Etiology and Incidence of infraorbital nerve injury: Infraorbital nerve dysfunction ranges from complete anaesthesia to a lesser degree of altered sensation and affects 25-80% of trauma patients with zygoma fractures, fractures of zygoma may impact the infraorbital nerve along the floor of the orbit either through an adjacent to the infraorbital foramen of the maxilla, resulting in altered neural conduction due to ischemia compression or transection, severe fractures with more displacement or communication are associated with a higher incidence of ION injury-edema and ischemia localized to the course of the ION, even in the absence of bony impingement, has the potential to alter normal nerve function. The lefort I maxillary osteotomy a relatively common procedure performed by the oral and

maxillofacial surgeon can be associated with postsurgical altered sensation in the distribution of the ION. Several other sources of injury to ION or patients complaints have been documented. Orbital decompression for ophthalmopathy has been associated with distressing hypersthesia in the infraorbital region upto 15% of patients reconstruction of the orbital floor or infraorbital rim after trauma may damage the ION pathology such as squamous cell carcinoma in the vicinity of the ION, may likewise lead to a numb cheek and upper lip in rare instances the subperiosteal face lift procedures can injure the ION. Dental procedures ranging from L.A. infections to endodontic treatment also can result in ION damage. Surgical approaches to the infra-orbital nerve. Following the surgical management of ION injury if not managed properly long term pain may develop or if pain is present prior to surgery, it might returns and possibly worsen perioperative antibiotics should be administered coverage of micro-organisms from the oral cavity, maxillary sinus and skin may be desirable, pre-operative intravenous steroids help to reduce post-operative swelling and might aid in nerve regeneration. If indicated, extraoral surgical access to the infra orbital rim allows the surgeon to expose not only the rim but he infra orbital foramen and the orbital floor, the surgeon choice of techniques to expose the infra-orbital rim which are similar to the cutaneous approaches to this area for open reduction of zygomatic orbital complex fractures infra orbital, lower eyelid, blephoroplasty, or transconjunctival type incisions are satisfactory options for surgical exposure surgeon experience and preference in conjunction with knowledge of advantages and disadvantages of each technique should guide the practitioner in making a final decision. However, the lower eyelid or infraorbital incisions may provide more direct access to and wider exposure, the foramen and orbital floor. The infra-orbital and lower lid surgical incisions can be placed within skin lines to minimize in esthetic postoperative scars. A transfacial approach to the ION as described by Ziccardi *et al* may provide the greatest access to this area, a modification of the ferguson incision is reported in which an incision from the nasolabial fold is extended along the alar groove to the medial canthal area and connected to a lower lid incision, the reported benefits include maximum visibility of bony and soft tissue components and no oral communication. Nonetheless a scar will result that might be undesirable for some patients. Following soft tissue dissection for access to the ION it should still be necessary to remove the bone from around the nerve if injury to the nerve is localized at or within the foramen, or is localized to the floor of the orbit and the nerve is covered by bone, then osteotomy will be necessary maintenance of the infraorbital rim, if possible is preferred for cosmetic concerns. A small fissure or round bur and small osteotomies, can be used to accomplish this procedure, copious irrigation and core to avoid damaging the nerve with the bur must be used. Visualization of the nerve may indicate compression, stricture, transection or neuroma formation surgical procedure to the nerve itself may include neurolysis, resection of neuroma or scar tissue, primary anastomosis or potentially a nerve graft, any suturing should be done with a small diameter nylon monafilament with 3-4 sutures, circumferentially placed through the epineurium if a graft is needed, the greater auricular nerve with a fascicular area of 0.82 mm^2 better approximates the size of the infra-orbital nerve than other nerves [15].

Surgical management of facial nerve injuries: Surgeons involved in the comprehensive management of complex craniomaxillofacial trauma are destined to encounter injuries to the facial nerve. However in patients with head injuries or in the multisystem injured patient, delay in diagnosis and therefore treatment of facial nerve injury can result from inability to visualize the patients facial expression caused by altered consciousness or the need for neuromuscular blockade or sedation, facial nerve compression. This topic is not relevant and therefore focused on the management of extracranial facial nerve injuries. Facial nerve injury related to soft tissue laceration or avulsion make up the majority of extracranial facial nerve injuries. Location of the injury must be determined accurately because it can significantly influence management. Parotid injury is commonly associated with extracranial facial nerve injuries.

Neurorrhaphy: Direct repair of the facial nerve should be accomplished by a skilled microsurgeon under optimal conditions. Partial parotidectomy is often required to gain adequate exposure for nerve anastomosis bipolar cautery is used where necessary within close proximity to the nerve, a bloodless field is required to achieve appropriate visualization of the nerve ends. The proximal nerve must be identified by visual inspection, orientation and depth are they features used to identify the nerve. High levels of magnification and trimming of the nerve allow characterization of the structure at the proximal end. These same procedures and electrical stimulation can be used within 3 days of transection for confirmation of the native of the distal segment. Direct neurorhaphy is especially indicated when sharps precise lacerations of the facial nerve have occurred such as one could see following razor blade, knife or plate glass injury. The identified segments must be minimally dissected to preserve blood supply to the remaining nerve segments, the nerve ends are carefully and completely trimmed direct simple perineural sutures are used to achieve approximation. Careful cooptation is necessary to prevent scar tissue in growth during the time of axonal regeneration, nylon or prolene sutures (10-0) are typically used. A surgeon's knot and 2 additional square throw are adequate the ideal anastomosis uses a minimal number and volume of suture to limit the inflammatory response to these materials. A toper cut needle has the advantage of being easy to pass while causing minimal trauma to the nerve. Graft neurorrhaphy: The procedure of nerve grafting is identical to that of direct nerve repair with the exception of requiring an additional anastomosis for each nerve branch treated, autogenous for each nerve branch treated, autogenous nerve graft remain the standard against which all other treatment are compared, tubulization with alloplastic materials remains a procedure most applicable and appropriate to the experimental microsurgical procedure, generally nerve grafting is required following avulsive type of injuries. Nerve graft donor sites commonly used include greater auricular nerve, sural nerve and more recently the antebrachial cutaneous nerve. The great auricular nerve is most commonly used when the total length of nerve graft required is small, its proximity to the operative field and its exposure in the course of performing superficial parotidectomy are major contributing factors the size and shape of the nerve is most compatible to the more proximal segments of the facial nerve the several nerve is generally used following extensive injuries or resections of multiple facial nerve branches its abundant length is certainly advantage in such cases, the antebrachial cutaneous nerve has advantage in that its structured in the proximal forearm involves many branches of the facial nerve in such instances a

single proximal anastomosis is combined with multiple distal anastomosis to critical facial nerve branches the reproducible result of nerve graft reconstruction of facial nerve defects can be best appreciated by comparing the procedure to immediate grafting of the facial nerve in radical parotidectomy [15].

Acceleration of recovery after nerve inujury to the peripheral nervous system using ultrasound and other therapeutic modalities

Peripheral neuropathy caused by injury or disease is a common clinical problem often associated with significant motor or sensory deficits, pain and other unpleasant sensations recovery can be slow and incomplete leading to personal hardship for patient and significant costs to society at large. The severity, or grade, of a peripheral nerve injury is one of the most important correlates with recovery for example, in a neuraproxia grade of injury such as those that arise as a result of mild to moderate acute injuries and chronic entrapment neuropathies, there is usually demyelination of the nerve with preservation of the axons. Acute and chronic injuries of greater magnitude or produce degeneration of nerve fibers distal to the site of trauma, these axonotmetic grades of injuries require not only axonal remyelination but axonal regeneration for recovery to occur. If the cellular and extracellular components that form a suitable substrate for axonal regeneration are available, as in crush injury, nerve fibers regenerate at an average rate of approximately 1mm/d injuries that require axons to traverse long distances before reaching their target muscles or sensory receptors can take upto 2 years for recovery of function current treatments for disabling nerve injuries involve surgical decompression for entrapment neuropathetis and surgical exploration and repair after severe trauma, trials of drugs for peripheral neuropathies, including various neurotrophic factors, have been unsuccessful to date, despite advances in medicinal and surgical management of peripheral nerve injuries recovery is often incomplete major impediments to a full recovery include long delay and associated tissue atrophy and imprecise reinnervation of the targets of the motor and sensory nerves it would be clinically beneficial to develop new treatment to accelerate and improve the recovery process [16].

Recent Research on Nonsurgical Treatment of Injured Peripheral Nerves

Recent research on treatment of peripheral nerve injury in addition to surgery includes laser treatment, electromagnetic stimulation and ultrasound. Laser: Rochkind and Ovaknine reviewed the literature on the use of low-power laser irradiation to promote healing in a rat model of acute peripheral nerve injury that often results in damage to the neuronal cell body. They applied the light transcutaneously every day for several minutes/session immediately after surgery, they have worked with a rat model of acute peripheral nerve damage that often result in damage to neuronal cell body, they have shown that direct laser treatment nerve tissue eventually restores the electrophysiologic activity of the injured peripheral nerve and prevents degenerative changes in neurons of the spinal cord interestingly, they note that treatment must occur within about 3 days of injury or there is no discernible effect. This implies that the laser stimulation acts on healing processes at work early in neuron recovery, perhaps by accelerating degeneration or the early stages of axon or myelin regeneration. They correlate these therapeutic benefits with the production by the laser of singlet oxygen, which has been shown in vitro to low concentrations to modulate intra cellular biochemical processes associated with mitosis.

Electromagnetic Stimulation: Almajed and coleagues showed in a rat model of a cut and reapproximated femoral nerve that as little as 1 hour of electric stimulation proximal cut immediatley after reconnection caused all the motor axons to progress down their regeneration pathway a given distance in 3 weeks rather than the 8-10 weeks required by the control animals, their histologic analysis is consistent with electric stimulation causing the axons to grow more quickly than they would without stimulation, as determined by counting the axons with a black labeling technique, their data are also consistent with the hypothesis that electric stimulation accelerates the progressive reinnervation of appropriate axonal pathways by the motor neurons at least moreover when tetrathodoxin was applied to the proximal portion of the cut nerves at a sufficient dose to block electric signals evoked in the cell body by the electric stimulation, the stimulation had no therapeutic effect, the tetradotoxin assay indicates that the beneficial action of the electric stimulation starts in the cell body of the regenerating axons, perhaps by accelerating protein synthesis [14].

Ultrasound: Recent studies demonstrate ultrasound's efficacy in treating chronic and acute peripheral nerve injuries, in human patients suffering from carpal tunnel's syndrome, for example, local application of ultrasound 20 times over a 2 week period reduced symptoms are improved nerve conduction compared with results in a placebo control group, even 6 months after the end of treatment similarly in an animal study, the application of ultrasound for 1mm 3 days per week over 1 month enhanced the recovery of normal nerve conduction velocity after partial crush injury to the tibial nerve of rats, in particular all electrophysiologic measures improved in a statistically significant fashion for the ultrasound group relative to the control group. The work by Hong *et al* motivated Mourod *et al*, who showed that local application of ultrasound for 1 min. 3 days per week over 1 month acceleration the recovery of sciatic nerve function after complete crush injury. Mourad *et al* found a statistically significant acceleration in recover of gait after complete crush injury for 2 ultrasound protocols relatively to controls. How does ultrasound create the effects observed by Hong *et al* and Mouard *et al*, as reviewed by mourad, ultrasound has been succesfully used to treat a variety of medical problems, including flesh wounds, bone fractures and several tendons, typically through the augmentation of protein synthesis; other studies have shown an increase in macrophage activity after exposure to ultrasound and still others have shown ultrasound to accelerate angiogenesis - ultrasound therapeutic effect on peripheral nerves may be the result of (1) acceleration of remyelination or axonal regeneration (2) acceleration of the macrophage led portion of the entire degeneration and regeneration phases by accelerating the flux of nutrients into and toxins out of injury site. The signal transduction mechanism that translates the acoustic energy into a biologic effect is unknown, mortime and Dyson have shown that ultrasound alters Ca++ influx in fibroblasts and changes in intracellular Ca++ are often involved in signal transduction [15].

Surgical Therapy for Traumatised Peripheral Nerves

Peripheral nerves of the face that have undergone neruopaxia or axonotmesis generally spontaneously recover, however

there are nerve injuries for which treatment by an oral maxillofacial surgeon trained in micro vascular surgery is warranted. External or internal nerve compression. Intentional or accidental severance of a nerve. Appearance of a traumatic neuroma and/or pain syndrome after nerve injury. External compression of a nerve trunk may result from impingement of a bone, tooth root or broken instrument. This external compression may be visible with special radiographs imaging techniques but frequently depends on explanatory surgery for establishing a diagnosis. Internal compression usually caused by growth of scar tissue into the nerve trunk, while it was regenerating. Surgical decompression of the treatment of choice for either external or internal nerve compression [16].

Prognosis of Nerve Injuries

Likelihood of spontaneous recovery: The likelihood of spontaneous recovery depends on the condition of the nerve, the severity of nerve injury and the site of injury a divided intraosseous nerve within a well aligned and approximated canal is more likely to recover spontaneously than is a divided extraosseous nerve because of the protection and guidance provided by the canal, divided extraosseous nerves are at risk for neuroma formation and dysesthesia or paralysis. Nerves that are chronically compressed or stretched will never achieve maximal regeneration, therefore exploration and neurolysis are indicated for maximal recovery the consequences of chronic compression and stretch are fibrosis, blocked axonal regeneration neuroma formation, dysesthesia, parasthesia, anaesthesia or paralysis intact nerves that have sustained a severe compressive crush or stretch injury have a poor prognosis for spontaneous recovery because of disruption of the fascicles and intraneural hemorrhage [17]. Clinically these patients will have anesthesia, dysesthesia or paralysis in the distribution of the injured nerve these injuries are best managed secondarily and even then the surgeon is presented with the dilemma of whether neurolysis or resection reconstruction is the appropriate treatment. Conditions limiting regeneration: Conditions that will limit or prohibit regeneration include chronic compression or stretch, divided nerves in which the nerve ends are not approximated and excessive scarring of either the nerve or tissue bed. Conditions that contribute to excessive scarring and fibrosis include infected or contaminated wounds, poorly vascularized wounds and wound closure or neurorhaphy under tension. Uncertainties: Clinical decisions are made more difficult as the number of uncertain variables increases, uncertainities regarding the condition of the nerve and wound are common. Paresthesia and incomplete paralysis are consistent with an intact nerve and carry a favourable prognosis for spontaneous recovery. Anesthesia and complete paralysis are associated with a severe compressive or stretch injuries and division injury and have a poor prognosis [18].

Conclusion

The comprehensive management of nerve injuries requires a surgeon with a number of available methods of reconstruction at his or her disposal. Comprehensive evaluation or documentation of injury is required to determine the most appropriate timing and method of surgical intervention. Accurate diagnosis and timely intervention are critical in achieving acceptable outcomes. Multidsciplinary collaborationis occasionally necessary for extremely proximal injuries. Delayed facial reconstruction by facial reanimation requires subspecialty skills and necessitates referals to centers experienced in such procedures.

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