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Trigeminal neuralgia, through the eyes of the medical illustrator

Brian D. McDermott

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A Thesis Submitted to the Faculty of The College of Fine and Applied Arts in Candidacy for the Degree of MASTER OF FINE ARTS

"Trigeminal Neuralgia, Through the Eyes of a Medical Illustrator"

By

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INTRODUCTION

Trigeminal neuralgia is perhaps the most painful disorder to affect mankind. The disorder is characterized by its "seizure-like" attacks of pain to designated areas of the face that are supplied by the trigeminal nerve. The individuals who suffer from trigeminal neuralgia describe its pain as unbearable, the equivalent of having a hot knife cutting through their faces down to the bone.

One of the earliest descriptions of Trigeminal Neuralgia was stated by Jurjani (1066-1136). He described the pain to affect the area of the teeth and jaw, and to occur in "spasm-like" attacks. Jurjani didn't designate specific anatomy, but concluded that the cause of the pain was due to the constriction of a nerve by an artery. His hypothesis is the earliest, most accurate theory proposed, thus supporting the presently known etiology of the disease.

Unfortunately, many other individuals have misdiagnosed the cause of trigeminal neuralgia. Crude attempts at eliminating the pain have been as extreme as extracting full rows of teeth, still leaving the patient to suffer the painful spasms.

However, thanks to the many advances in research and medicine, treatment of trigeminal neuralgia is now possible.
Specific medication and surgical procedures have given many individuals freedom from its painful attacks.
INCIDENCE AND ETIOLOGY

Since there are many types of facial pain due to abnormal dental, muscular, skeletal and neural abnormalities, trigeminal neuralgia is usually hard to diagnose. Such examples of facial pain that are common include: trigeminal neuritis, characterized by a persistent state of paresthesia; traumatic infraorbital neuralgia, similar to trigeminal neuritis; and traumatic dental neuralgia, paresthesias that occur after dental extraction. The duration of trigeminal neuritis is constant, so it is treated with continuous applications of analgesics; however, traumatic infraorbital neuralgia and traumatic dental neuralgia have ends to their cycles. Eventually, paresthesia ceases and normal sensation returns.

The incidence of individuals developing trigeminal neuralgia in a given population occurs in an estimated ratio of about 155 cases per 1,000,000 people. A further breakdown of distribution among each sex is roughly 107 cases per 1,000,000 males and 200 per 1,000,000 females. The age of acquiring the disease varies, but is usually found to affect adults past the age of 40; however there are a few childhood cases on record.

The pain attacks are usually experienced on the
right side of the face, though in some cases, bilateral tic douloureux may exist. According to statistics, the most frequently reported cases of the disease are said to be diagnosed in women over 40 years of age and the pain distribution is unilateral.

There seems to be a correlation among some people who develop multiple sclerosis and also develop trigeminal neuralgia. Multiple sclerosis is a neural disease that cripples millions of people each year. During the clinical course of multiple sclerosis, the myelin sheaths, which enclose the nerve fibers, degenerate and thus, oligodendria cease to function and oligodendrocyte production stops. As the myelin enclosing the epineurium degenerates, the nerve is no longer insulated, making it more vulnerable to irritation and injury. An important physiological trait of myelin is that it has the ability to aid in the transduction of neural impulses along axons, thus transmitting impulses quickly and effectively to peripheral target structures. During the course of multiple sclerosis, the outbreaks of demyelination may be followed by periods of remission. As in the case of trigeminal neuralgia, the cause of multiple sclerosis is unknown. The demyelin0tion process occurs in the white matter of the central nervous system. Areas often affected include the optic chiasm, optic nerves, cerebellum, brain stem and posterior cell columns in the spinal cord. As the
disease progresses, plaques form in regions of neural demyelination. The plaques are the by-product of the demyelination process and are macroscopic in size.

Although the causing factors of some cases of trigeminal neuralgia are unknown, certain discovered physical abnormalities can cause the condition. Vascular compression and tumor growth are the most frequent etiological factors. Vascular compression results from tension applied to a nerve, usually its root, from the constricting proximity of a blood vessel. Compression of the superior cerebellar artery to the trigeminal root is a common occurrence in patients with trigeminal neuralgia. The superior cerebellar artery, in this situation, is trapped beneath the trigeminal root. The blood flowing through the lumen of the vessel must build up enough pressure to flow through the area of constriction. As vascular pressure builds up inside the artery, the endothelium stretches, causing the the vessel to expand. This action triggers the pain receptors in the trigeminal root to fire. This condition can be corrected by surgery. The normal procedure would be to release the superior cerebellar artery from its constriction between the trigeminal root and the base of the pons.

Tumor growth can also be the cause of neurological problems. Trigeminal studies have shown that tumor growth along trigeminal pathways can cause pain, numbness and
paresthesia. Tumors can stretch, deform or compress the
nerve, thus sending afferent synapses of intense pain to the
thalamus, by way of the A-delta fibers. Tumors affecting
trigeminal pathways have been found in many regions
intracranially. These areas include the middle and posterior
cranial fossae, cavernous sinus, meckel's cave, foramen ovale
and areas around the tentorium cerebri. In many of these
sights of tumor compression, the tumor usually keeps growing
and will metastasize to surrounding tissues, unless surgery
or some other type of innervation is initiated.
The trigeminal nerve is the fifth and largest of the twelve cranial nerves. It is a compound nerve, meaning that it is capable of both sensory and motor innervation. The trigeminal motor pathway contains one nucleus, while the sensory pathway has three.

MOTOR PATHWAY: The motor nucleus of the trigeminal nerve is located interpontine, slightly lateral to the pontine medial plane and slightly inferior to the fourth ventricle. It receives cerebral stimuli initiated from the lower third of the precentral gyrus. These corticobulbar fibers pass through the layers of the corona radiata, internal capsule and cerebral puduncle and bury themselves in a decussating fashion into the pons, prior to their junction with the motor nucleus.

The motor root of the trigeminal nerve, portio minor, emerges in an anteriolateral fashion from the motor nucleus and projects itself through the tissue of the pons. As the Motor Root leaves the pontine tissue, it is orientated anteriomedially to the sensory root (portio major), which is the larger of the two. The portio minor continues anteriorly, passing through the region of the posterior cranial fossa and projects itself through the meningeal
tissue of the dura mater, inferior to the attachment of the
tentorium cerebri to the petrous portion of the temporal bone. Still traveling rostrally, it enters a cavity in the
dura mater, which is orientated superiorly to meckel's cave, a depression in the petrous ridge of the temporal bone, projecting under the semilunar (trigeminal) ganglion and exits the intracranial space through the foramen ovale (located medially in the middle cranial fossa). After the motor root exits the intracranial space, it joins with the mandibular division of the trigeminal nerve, thus forming the mandibular nerve. The mandibular nerve innervates the muscles of mastication: the masseter, the temporalis and the medial and lateral pterygoid muscles. Mandibular motor fibers also supply the tensor tympani, a muscle found in the osseous canal surrounding the eustacian tube; tensor veli palatini, an outer layer of muscle located on the lateral aspect of the posterior nares; and the mylohyoid and digastric muscles, "strap muscles" of the ventral side of the neck.

SENSORY PATHWAY: The sensory cell bodies of the trigeminal nerve are located near the apex of the petrous bone in the middle cranial fossa, within the semilunar ganglion. Sensory fibers from the semilunar (gasserian) ganglion travel in a dorso-medial fashion and enter the tissue of the pons.
After the fibers penetrate the pons, the sensory fibers distribute themselves to form three main nuclei within the brainstem. The three major nuclei of the trigeminal sensory pathway include the nucleus of the trigeminal spinal tract, the main (principle) sensory nucleus and the mesencephalic nucleus.

The spinal tract of the trigeminal nucleus is formed after the trigeminal sensory fibers enter the pons. They form a bundle-like mass extending to the caudal end of the medulla and descend into the spinal cord. The trigeminal spinal tract descends to the level of the third or fourth cervical vertebra, where it fuses with lisseuer's tract. As the spinal tract descends, it supplies fibers to the nucleus of the spinal tract of the trigeminal nerve. The trigeminal spinal nucleus has three subdivisions: the nucleus oralis, the interpolaris and the caudalis. Sensory fibers from the ophthalmic division descend to the second cervical level where the fibers are the most ventrally oriented fibers of the spinal tract. Fibers from the mandibular division descend to the most rostral level of the trigeminal spinal nucleus. The spinal tract pathway runs a course oriented more dorsally than other spinal tract fibers. Fibers from the maxillary division terminate in two different areas. Maxillary sensory fibers, supplying the medial portion of the face, nose and mouth, lead to the rostral aspect of the
spinal nucleus. Maxillary innervation to the lateral portion of the face leads a spinal tract pathway that terminates in the caudal end of the trigeminal spinal nucleus. Since the maxillary division of the trigeminal nerve has two different destinations in the afferent sensory pathway leading to the central nervous system, it accounts for a unique pattern for facial sensitivity. Spinal nucleus or spinal tract lesions can cause a peripheral sensory loss to occur in the face. The sensory loss occurs in "onion-skin" patterns and is found predominantly around the mouth or buccal area.

Afferent innervation from the face and mucous membranes send impulses of soft touch, pain and temperature to the spinal nucleus of the trigeminal nerve. From there, neurosynapses travel craniad via the trigemino-thalamic tract and are received in the ventral posteriomedial and intralaminar nuclei of the thalamus.

The main sensory nucleus of the trigeminal nerve is found posteriolateral to the trigeminal motor nucleus, inside the pons. Afferent fibers supplying this nucleus transmit messages conveying tactile stimulation and proprioceptive impulses. The trigeminal main sensory nucleus sends ascending impulses to the thalamus by way of the ventral crossed trigemino-thalamic tract. The specific destination of this nerve tract is in the thalamic ventral posteriomedial nucleus.
The mesencephalic nucleus ascends from the main sensory nucleus, cephalad, to the mesencephalic superior colliculus. It receives synapses of proprioception from muscles supplied by various motor nerves, as well as the trigeminal.

The regions of the face that are innervated by the trigeminal nerve are grouped into three different divisions. They are classified as the ophthalmic division (V1), maxillary division (V2) and the mandibular division.

**OPHTHALMIC DIVISION (V1)**

The ophthalmic division of the trigeminal nerve can also be classified as the ophthalmic nerve. The smallest of the trigeminal divisions, its mode of innervation is strictly sensory. The ophthalmic nerve supplies the nose, nasal fossa, forehead, integument of the eyebrow and regions of the orbital cavity including the lacrimal gland, ophthalmic mucosa and the eyeball itself. The ophthalmic nerve originates and branches off of the anterior superior aspect of the semilunar ganglion. It projects in an occular direction along the outer wall of the cavernous sinus. Before entering the orbital cavity, it divides into three branches within the sphenoid tissue. The subdivisions are the lacrimal, frontal and nasal branches of the ophthalmic
nerve.

The lacrimal nerve, the smallest division of the three subdivisions that make up the ophthalmic nerve, moves forward, entering the orbital cavity through the sphenoidal fissure. Its intraorbital path takes it along the superior aspect of the lateral rectus muscle and continues forward with the lacrimal artery to terminate into the lacrimal gland, the conjunctiva and the superior palpebral fascia. It also communicates with filaments of the facial nerve.

The frontal nerve is the largest of the three ophthalmic subdivisions. It enters the orbital cavity through the sphenoidal fissure and runs a superior and medially oriented pathway above the levator palpebrae muscle. Still running a frontal path, the frontal nerve bifurcates at the half-way point inside the confines of the orbital cavity. It forms the supratrochlear and supraorbital nerves. The smaller supratrochlear nerve passes above the "pulley-like" structure of the superior oblique muscle to enter the supraorbital foramen. After its exit from the supraorbital foramen, it takes an upward path along the forehead proximal to the frontal bone and beneath the corrugator, supercilli and frontalis muscles. Frontal nerve filaments also penetrate through these muscles to supply the integument of the forehead. The larger supraorbital nerve also projects forward and through the supraorbital foramen to supply the
upper eyelid and forehead. Supraorbital innervation terminates in subcutaneous integument and pericranial tissue of the frontal and parietal regions.

The nasal nerve enters the orbital cavity, running between the two heads of the lateral rectus muscle, and passes across the optic nerve, inferior to the superior oblique and superior rectus muscles. It then enters the anterior ethmoidal foramen, which is orientated on the medial inner wall of the orbital cavity. After the nasal nerve passes through the anterior ethmoidal foramen, it enters the intracranial space, following a rostral path by passing downward in a transverse fashion through a groove located in the frontal aspect of the cribiform plate, part of the ethmoid bone. The nasal nerve then runs through a slit orientated proximal to the crista galli, also part of the ethmoid bone. Still following a rostral direction, the nasal branch of the ophthalmic nerve finally enters the nasal cavity and there, the nerve bifurcates into two additional branches, the internal branch and the external branch. The internal nasal nerve innervates the olfactory mucosa of the ventral aspect of the nasal septum. The external nasal nerve follows a medially oriented path inside the nasal bone. It descends to supply sensory innervation to the olfactory mucosa of the frontal part and outer walls of the nasal nares, including the inferior nasal spongy bone. The
external nasal nerve then exits the nasal cavity between the lower border of the nasal bone and lateral alar cartilage. It descends beneath the compressor nasi muscle and supplies the integumental tissue of the alar region of the nose and it also communicates with branches of the facial nerve.

The ciliary ganglionic nerve branches off the nasal nerve, oriented between the two heads of the lateral rectus muscle. It continues frontally, passing along the lateral side of the optic nerve and penetrating the neural tissue of the ciliary ganglion, entering its posterior superior aspect. It is sometimes joined by the superior division of the oculomotor nerve.

The long ciliary nerves, usually two or three branches, branch off the nasal nerve a short distance after the ciliary ganglionic nerve branches off the nasal nerve. The long ciliary nerves arise from their nasal nerve origin at the point where the nasal nerve crosses the path of the optic nerve. They join the short ciliary nerves which arise from the ciliary ganglion and penetrate the sclerotic tissue of the posterior aspect of the eye. The long ciliary nerves continue their path in an anterior direction, running between sclerotic and choroid tissues to finally reach their targets of primary innervation. They supply the meridional and circular fibers which make up the ciliary muscle group, the iris and cornea.
The infratrochlear branch arises from the nasal nerve prior to the point of entry of the nasal nerve into the anterior ethmoidal foramen. It runs anteriorly along the superior border of the medial rectus muscle and joins a filament from the supratrochlear nerve, located underneath the "pulley-like" structure of the superior oblique muscle. The infratrochlear nerve then runs to the inner angle of the eye, supplying innervation to the integumentary tissues of the eyelids, the conjunctiva, the lacrimal gland and the lateral aspect of the nose.

THE CILIARY GANGLION: The ciliary ganglion is very small, about the size of a pin-head. It is oriented in the posterior portion of the orbital cavity. The ganglion is embedded in fatty tissue and is situated between the optic nerve and the lateral rectus muscle. The ophthalmic artery usually lies medially to it.

The ciliary ganglion has three branches of communication: the sensory root, motor root and the sympathetic root. The ciliary sensory root arises from the nasal nerve at a short distance from the semilunar ganglion, and enters the ciliary ganglion inferiorly. The ciliary motor root, sometimes divided into two parts, originates from a branch of the oculomotor nerve, which supplies the inferior oblique muscle. It joins the ciliary ganglion's superior border. The third branch of communication of the
ciliary ganglion is the sympathetic root, formed from a single nerve branch originating in the cavernous plexus of the sympathetic nervous system.

The ciliary branches of distribution, the short ciliary nerves, are made up of six to ten fibers which branch off of the ventral aspect of the ciliary ganglion. They form into two bundle-like arrangements, with the lower bundle being the larger. Their path runs ventrally with the ciliary nerves and arteries, with one bundle above the optic nerve, and the other bundle below it. They too, like the long ciliary nerves, project through the ocular tissue of the sclera and run forward to reach their target structures: the iris, cornea and the ciliary muscles.

MAXILLARY DIVISION (V2)

The maxillary nerve is the intermediate of the three trigeminal subdivisions, because of its position and size. Its root originates from the ventral medial aspect of the gasserian ganglion. The root projects forward, ventrally, through the foramen rotundum, located along the inner ventral wall of the middle cranial fossa. After its exit from the foramen rotundum, the maxillary root passes the sphenomaxillary fossa to enter the infraorbital canal,
located inferiorly inside the orbital cavity. The maxillary nerve path continues inside the sphenomaxillary fissure, projecting in a slightly declined ventral angle to finally reach the infraorbital foramen. The nerve then passes through the foramen to the exterior. The major bulk of nerve lies beneath the quadratus labii superioris muscle; however, smaller branches of the infraorbital nerve travel to the lateral aspect of the nose, inferior ocular region and the upper buccal area. It communicates with branches of the facial nerve in these areas.

The maxillary nerve has four branches of distribution: intracranial, sphenomaxillary, infraorbital and facial.

The maxillary intracranial distribution consists only of the meningeal branch, stemming off of the maxillary nerve, as it originates from the semilunar (gasserian) ganglion. Its path, which leads to the meningeal dura mater, joins the pathway of the middle meningeal artery.

The sphenomaxillary distribution consists of three subdivisions: the temporomalar, orbital; the sphenopalatine, and the posterior superior dental branch.

The temporomalar branch originates within the sphenomaxillary fossa. It enters the orbital cavity via the sphenomaxillary fissure, where it bifurcates to form temporal and malar branches. The sphenomaxillary temporal branch runs a path along the lateral aspect of the orbital cavity,
communicates with a filament from the lacrimal nerve and passes through the malar bone via an osseous foramen to enter the temporal fossa. It projects itself cranial, running along the lateral aspect of the frontal bone to the temporalis muscle. It then pierces the tendonous tissue of the temporalis and fascia to innervate the integumental tissues of the lateral forehead and temporal area. In these regions, the nerve will communicate with some filaments of the facial and auriculotemporal branches of the inferior maxillary nerve. The sphenomaxillary-malar branch runs along the external inferior angle of the orbital cavity; the nerve then passes through to the face via a malar foramen and penetrates through the orbicularis occuli muscle to supply the skin of the cheek. In this external region, the malar nerve filaments also communicate with filaments from the facial and palpebral branches from the superior maxillary nerve.

The sphenopalatine division is made up of two branches which end their paths in the sphenopalatine ganglion.

The last subdivision of the sphenomaxillary branch consists of the posterior superior dental branches. These branches, which arise from the maxillary nerve prior to its entry into the infraorbital canal, are usually made up of two branches of nerves which give off fibers to supply the gums and mucous membranes of the cheek. These fibers also enter
the posterior dental canals of the maxillary bone and run ventrally within the osseous tissue, thus joining with filaments of the middle dental nerve. From here, fibers also will branch through the dental alveolar region to supply the molars and the pulp of the incisors.

The infraorbital distribution consists of two subdivisions: the middle superior dental and the anterior superior dental divisions.

The middle superior dental branch arises from the maxillary nerve, within the ventral aspect of the infraorbital canal. It descends ventrally, in the outer wall of the antrum to supply the bicuspids. The middle superior dental branch also communicates with the posterior dental branch at the ganglion of valentin and with the anterior dental branch at the junction called the ganglion of bochdalek. However, these are not proven to be actual ganglia, the nomenclature given is strictly reference areas for the separation points of the three dental branches.

The anterior superior dental division branches off of the maxillary nerve prior to its emergence from the infraorbital foramen. It descends through a canal in the anterior wall of the antrum to divide into sub-branches which supply the incisors and canine teeth. Being the most ventrally oriented of the dental branches, its only dental communication junction is with the middle dental nerve;
however, the anterior superior dental branch does give rise to a nasal branch, which follows a rostral pathway to supply the olfactory mucosa.

The facial maxillary distribution consists of three divisions: the palpebral, the nasal and labial divisions.

The palpebral branches originate from the maxillary nerve after its exit from the infraorbital foramen. These branches ascend between the zygomatic bone and the orbicularis oculi to supply sensory stimulation to the conjunctiva and integumental tissue of the lower eyelid. Malar and facial nerve fibers also communicate with the palpebral branches.

The nasal branches follow a path to the lateral aspect of the nose to supply its external integumental tissue. Here, they will communicate with the nasal branch of the ophthalmic (V1).

The maxillary labial branches are the most plentiful of the maxillary-facial distribution. These branches descend beneath the quadratus labii superioris to supply integumental tissue of the upper lip and oral mucosa. The labial branches also communicate with filaments of the facial nerve to form the infraorbital plexus which is located slightly inferior to the orbit.

THE SPHENOPALATINE GANGLION: The sphenopalatine ganglion, meckel's ganglion, sits slightly inferior to the maxillary
nerve root as it passes the sphenomaxillary fossa, proximal to the sphenopalatine foramen.

Branches of communication include a sensory, motor, and a sympathetic root.

The sphenopalatine sensory root arises from the two sphenopalatine branches which descend a short distance from the maxillary nerve. Descending through the sphenomaxillary fossa, some fibers terminate their pathway and enter the sphenopalatine ganglion while other fibers end their paths in the palat and nasal fossa. The sphenopalatine fibers that enter the sphenopalatine ganglion make up the sphenopalatine sensory root.

The sphenopalatine motor root of meckel's ganglion arises from the superficial petrosal branch of the facial nerve.

The sympathetic root of meckel's ganglion originates in the carotid plexus, via the deep petrosal nerve.

Both the superficial petrosal nerve motor root and the deep petrosal nerve sympathetic root join to form the vidian nerve prior to its entry into meckel's (sphenopalatine) ganglion.

The superficial petrosal branch arises from the geniculate ganglion of the facial nerve (VII), in the fallopii acqueduct. Its path runs ventrally, entering the cranial cavity. It continues frontally through a groove on
the anterior surface of the petrous region of the temporal bone, inferior to the dura mater. The superficial petrosal nerve then penetrates the cartilaginous tissue within the foramen lacerum and joins with the deep petrosal nerve branch to form the vidian nerve.

The deep petrosal branch arises from the carotid plexus and runs through the carotid canal, lateral to the internal carotid artery. It, being a tributary to the vidian nerve, joins the superficial petrosal branch after it enters the foramen lacerum. As the two branches join, they give rise to the vidian nerve.

The vidian nerve, after it is formed by the superficial petrosal branch and the deep petrosal branch, follows the path of the vidian canal. Arising from the otic ganglion, the sphenoid branch ascends to join the vidian nerve, prior to its entry into the sphenomaxillary fossa. The nerve then terminates in the sphenopalatine, meckel's ganglion.

There are four vidian branches of distribution: The ascending branches lead to the orbit, the descending branches lead to the palate, the internal branches lead to the nose, and the posterior branches lead to the nasal fossa and pharynx.

The vidian ascending branches comprising of two or three slender filaments, enter the orbit via the sphenomaxillary fissure, to supply the periosteum.
The vidian descending branches distribute themselves to the mucus membrane of the nose, tonsils, soft palate, and roof of the mouth. They are distributed into three branches: the anterior, the middle and the posterior.

The vidian internal branches supply the septum and the outer wall of the nasal fossa.

The vidian superior nasal branches enter the posterior region of the nasal fossa via the sphenopalatine foramen. These branches supply the mucous membrane tissue of the superior and middle spongy bones and the lining of the posterior ethmoidal cells.

The nasopalatine nerve enters the posterior region of the nasal fossa through the sphenopalatine foramen, as did the superior nasal branches. As they enter the fossa, their path runs medially across the roof of the nose, inferior to the sphenoid sinus, until it reaches the nasal septum. There, it then descends in an oblique fashion, anteriorly along the lower part of the septum of the anterior palatine foramen, between the periosteal tissue and the olfactory mucosa. The nerves project downward through the anterior palatine canal to the roof of the mouth. Two branches make up the nasopalatine nerve, and are situated in the intermaxillary suture. Within the mouth, the two branches become united and supply the mucous membrane behind the incisor teeth and also join the anterior palatine nerve. The
nasopalatine nerve also supplies filaments to the mucous membrane of the septum.

The vidian posterior branches are made up of the pharyngeal and upper posterior nasal branches.

The pharyngeal nerve is a tiny branch that originates from the posterior aspect of the pterygopalatine ganglion. The pharyngeal nerve descends dorsally, with the pterygopalatine artery, through the pterygopalatine canal to supply the pharyngeal mucosa located behind the eustachian tube.

The upper posterior nasal branches are few in number, originating from the posterior aspect of meckel's ganglion. They run dorsally inside the vidian nerve sheath to supply the mucous membrane of the nasal roof, the septum, the meatus and also the eustachian tube.

THE MANDIBULAR NERVE (V3)

The mandibular nerve is the largest of the three major divisions of the trigeminal nerve. It supplies the lower jaw, including the teeth and gums, the external temporal and ear region, the muscles of mastication and the tongue.

It has both a sensory and a motor root. The sensory root descends from the inferior aspect of the gasserian
ganglion. The motor root, the smaller of the two, also descends beneath the gasserian ganglion to join the sensory root after passing through the foramen ovale, as the roots leave the intracranial cavity. After it exits the foramen ovale, beneath the skull, the anterior and posterior trunks separate. Prior to this separation, a small meningeal branch arises from the mandibular nerve to supply the internal pterygoid muscle.

The recurrent branch arises from the mandibular nerve after its exit from the foramen ovale. The nerve passes back through the foramen spinosum to enter the intracranial cavity, following the path of the middle meningeal artery; the nerve bifurcates, forming an anterior and posterior branch. The anterior recurrent branch communicates with the meningeal branch of the mandibular nerve and supplies the meningeal dura mater. The recurrent posterior branch supplies the mucosa of the mastoid region and also supplies the dura mater.

The masseteric branch runs forward, between the outside border of the lateral pterygoid muscle (ventral to the tempomandibular articual disc) and beneath the temporalis tendon. It passes between the coronoid process and condyle of the mandible, via the sigmoid notch. At this point, the masseteric nerve shares the same route as the masseteric artery. Both, supply the masseter muscle and branch through
its tissue, some of the sub-branchings will travel as far forward as the anterior border. Occasionally, a few filaments may terminate at the mandibulotemporal articulation or at the temporalis.

The mandibular deep temporal branches supply the anterior and posterior borders of the temporalis along its medial surface. There are typically only two branches of the deep temporal division. The posterior temporal branch, the smallest, is found at the posterior region of the temporal fossa. Occasionally it will communicate with filaments from the masseteric division. The deep temporal anterior branch usually originates off of the buccal nerve. It ascends up along the pterygoid aspect of the sphenoid bone, leading to the frontal area of the temporal fossa. It terminates into the anterior border of the temporalis. Atypically, there exists a third deep temporal branch. It also supplies the inner surface of the temporalis, with its innervation region being the middle of the temporal fossa. The branch originates off the buccal nerve and reflects over the external surface of the lateral pterygoid muscle, up along the sphenoid bone to terminate in the temporalis muscle.

The buccal nerve runs forward through the separation between the superior and inferior heads of the lateral pterygoid muscle. The nerve descends in one of two paths at this point: either beneath the inner surface of the
mandibular coronoid process, or directly through the fibers of the temporalis. It will reach the buccinator muscle and bifurcate to form superior and inferior branches that ramify forward throughout the muscle.

During the buccal nerve's earlier descent, between the lateral pterygoid superior and inferior heads, tiny filaments are given off. Some will supply the pterygoid muscles and a few filaments will reach the temporalis. Occasionally, a filament or two will communicate with the deep temporal anterior division and the superior branch of the buccal nerve. The superior buccal nerve will communicate with VII and also supply the integumental tissue of the upper buccinator region. The inferior branch, reaching the lower integument and oral mucosa, also joins with the facial nerve.

The external pterygoid nerve commonly arises from the buccal nerve, however, it may originate directly from the maxillary (V2). It supplies the lateral pterygoid muscle, entering it on its inner surface.

The posterior part of the mandibular nerve is sensory; however, a few motor filaments supply it from its motor root. Its three divisions include the auriculotemporal nerve, the lingual nerve and the inferior dental nerve.

The auriculotemporal nerve branches off the mandibular nerve at the level of the otic ganglion. Descending backward beneath the lateral pterygoid muscle, the nerve then
protrudes outward over the mandibular condyle. At this point, the nerve climbs upward, under the parotid gland and ascends over the zygoma to finally bifurcate into two temporal branches.

The auriculotemporal nerve has two areas of communication: one, being the facial nerve and the other the otic ganglion. The facial nerve has two points of auriculotemporal connection. The auriculotemporal nerve, as it projects itself backward and out around the mandibular condyle, joins with the facial nerve at the posterior aspect of the masseter. Communication with the otic ganglion has four points of neural junction: the anterior auricular, the articular, the parotid and the superficial temporal branches.

The anterior auricular branch mainly supplies the anterior part of the pinna (external ear), and the integument of the tragus and helix.

The articular branch supplies the area of articulation between the temporal bone and the mandible.

The parotid branch supplies the parotid gland.

The superficial temporal branch supplies temporal integumental tissue. As well as its facial communication, the superficial temporal branch also joins a few filaments with the temporomalar branch of the maxillary nerve (V2).

The lingual nerve, also referred to as the gustatory (tasting nerve), supplies muscles and papillae of the
anterior two thirds of the tongue. It branches off of the mandibular nerve at the lingual-inferior alveolar bifurcation. The lingual path runs anterior to the inferior alveolar branch and descends beneath the lateral pterygoid muscle, with the inferior dental nerve medial to it. Occasionally filaments may communicate between the nerves. The chorda tympani nerve joins the lingual nerve, posteriorly at about a 45 degree angle. Still descending, the lingual nerve passes down between the medial pterygoid muscle and the inner surface of the mandibular ramus. Curving in at an oblique angle, the nerve passes over the styloglossus and superior constrictor muscles and also between the hyoglossus muscle and the submaxillary gland. The lingual nerve runs along wharton's duct (opening to the submaxillary gland) and to the apex of the genioglossus muscle, superior long muscle and inferior long muscle (all of which, make up the apex of the tongue) below the oral mucosa.

The lingual nerve has four branches of communication: the facial, the inferior dental, and the hypoglossal nerves and the submaxillary ganglion.

The inferior dental branch is the largest division of the mandibular (V3) nerve. Its path descends along with the inferior dental artery, running beneath the medial pterygoid muscle, then between the internal lateral ligament and medial surface of the mandibular ramus. The nerve then enters the
dental foramen and channels its path within the dental canal of the inferior maxillary bone, inferior to the teeth, to the mental foramen, where it divides into the mental and incisor branches.

The four branches that make up the inferior dental nerve include the mylohyoid, the dental, the incisive, and the mental branches.

The mylohyoid nerve originates off the inferior alveolar nerve prior to its descent through the dental foramen. The mylohyoid nerve descends along the medial surface of the mandibular ramus and runs under the mylohyoid to supply the mylohyoid and digastric muscles.

The dental branches of the inferior dental nerve supply the bicuspid and molar teeth. The nerve's fibers enter the pulp of the teeth.

The mental branch exits the mental foramen and subdivides beneath the depressor anguli oris. These divisions innervate the integument of the chin and lower lip. The dental branches also communicate with the facial nerve.

Two nerve ganglia are associated with the mandibular nerve (V3): the otic ganglion, with the maxillary trunk; the submaxillary ganglion, with the lingual nerve.

THE OTIC GANLION: The otic ganglion is located extracranially, inferior to the foramen ovale and medial to
the inner surface of the mandibular nerve. It is situated medial to the point of junction of the motor and sensory networks of the mandibular nerve. The ganglion is lateral to the eustachian tube and the origin of the tensor veli palati muscle, anterior to the middle meningeal artery.

The otic ganglion has a vast network of communication. It is exposed to sensory, motor and sympathetic modes of neural innervation. It shares communication with the external branch of the mandibular nerve by a few tiny filaments. These filaments supply the otic ganglion with a motor root and possibly a sensory root. Communication is established with cranial nerves seven and nine. Facial and glossopharyngeal impulses reach the otic ganglion via the superficial petrosal nerve, which arises in the tympanic plexus. The glossopharyngeal probably supplies a sensory root and the facial, a motor root. Sympathetic communication is established from the middle meningeal plexus. Other communication arises from the auriculotemporal and vidian nerves.

The otic distribution includes only two areas: the first runs backward along the lateral side of the eustachian tube to supply the tensor tympani muscle; the second runs a frontal path to supply the internal pterygoid muscle and a single branch to supply the chorda tympani.
SUBMAXILLARY GANGLION: The submaxillary ganglion is relatively small in comparison to the semilunar (gasserian) ganglion. It is "spindle-like" in shape and is located in close proximity to the posterior border of the mylohyoid muscle and superior to the submaxillary gland.

Its branches of communication include the lingual nerve, facial nerve and the sympathetic plexus. Lingual nerve communication is received by the submaxillary ganglion at its anterior and posterior aspects. Communication from the facial nerve travels via the chorda tympani. Sympathetic innervation arises from the sympathetic plexus, around the facial artery.

Branches of distribution originate from the inferior region of the submaxillary ganglion. They include approximately six branches. Target structures consist of the oral mucosa (including wharton's duct, with additional ramifications into the submaxillary gland), the lingual nerve and its connection with the frontal aspect of the submaxillary gland, the sublingual gland and the tongue.
PATHOGENESIS:

There have been many different theories derived throughout history regarding the pathogenesis of trigeminal neuralgia. However, two main hypotheses prevail: one theory states that tic doloureux is initiated from factors within the central nervous system, and the other theory bases the cause on peripheral factors.

Surgical intervention supports the "PNS (peripheral nervous system) theorists. Peripheral etiological causations include such occurrences as tissue inflammation, neoplastic growths and lesions, and vascular (compression) of the nerve.

However, therapeutic nonsurgical medical treatment has had an affect in suppressing pain in the trigeminal system. The introduction of carbamazepine and baclophen have had success in alleviating the abnormal pain associated with trigeminal neuralgia. These drug treatments support the theory of etiological factors within the CNS, specifically the region of the trigeminal nuclei, nucleus oralis, within the brain stem.

DATA SUPPORTING PERIPHERAL NERVOUS SYSTEM THEORY: The most common physical etiological factors discovered in patients suffering from the attacks of tic douloureux include vasuclar compression from arteries of the pons, tumor growths, and plaques deposited on the trigeminal root.
Vascular compression is most commonly caused by the aberrant location of the superior cerebellar artery, which arises from the circle of Willis, constricting the trigeminal root.

Neoplasms can metastasize with any tissue type, thus promoting the trigeminal root to be subjected to ever increasing neural compression unless the tumor is removed.

Plaques deposited on the trigeminal root are typically found if a patient has had a history of multiple sclerosis. Plaques are a by-product of the demyelination process. Plaques are dark and tarrish in appearance. Those who had suffered from the attacks of tic douloureux, during their lifetime commonly were discovered to have plaque accumulation when autopsies were performed. It is also clinically stated that individuals who suffer from multiple sclerosis, have a greater chance of developing trigeminal neuralgia.

Surgical sectioning of the trigeminal nerve has had lasting success in some patients, giving them months or years of pain free relief.

DATA SUPPORTING CNS (CENTRAL NERVOUS SYSTEM) THEORY:
Studies in animal laboratories, drug treatments and tests run on patients with trigeminal neuralgia (both, before and after surgery) have supplied valuable data regarding the neural-pathogenic actions of the trigeminothalamic system.

Clinical data involving tic douloureux attacks has lead
to theorizing a CNS etiology. Such characteristics include: (a) a measurable period between the period of stimulation to a "trigger area" and the violent attacks of pain, (b) soft skin contact, which initiates the attacks, would normally be interpreted as light, gentle tactile stimulation, (c) The attacks are self-sustaining, following the initial "trigger-area" contact, (d) (the most astounding of all characteristics) the intense pain associated with the disease is experienced in regions of the face that are not in close proximity to the trigger areas.

Experimentation performed on laboratory monkeys and cats have lead to remarkable advancements in trigeminal nerve research. Trigeminal root demyelination, manually induced in the subjects, caused abnormal impulses to be experienced in peripheral regions of the trigeminal nerve. This neurophysiological phenomenon was alleviated after intravenous introduction of the drug phenotoin was administered, however, many of the subjects constantly rubbed and scratched at their faces. This condition typically occurs in people in treatment for trigeminal neuralgia. The direct application of "epileptogenic" agents to the spinal trigeminal nucleus, in laboratory subjects, induced a tactile state of hyperactivity in the faces of most of the animals. The animals frantically displayed an avoidance reaction to tactile stimulation. Some of the subjects reacted by
scratching and pawing at their faces.

Partial sectioning of the preganlionic trigeminal root resulted in abnormal afferent firing to peripheral branches of the face; however, this condition passed after a period of time, leaving the subject nonirritated. Total retrogasserian rizotomys, however, resulted in more intense afferent firing, with no point of duration.

Denervation of tooth pulps supplied by the maxillary and mandibular divisions, supplying afferent impulses, resulted in a notable short-term increase in "low threshold action potentials" in the spinal nucleus oralis. Tactile hypersensitivity also occurred.

Different degrees of trigeminal nerve injuries cause various types of perception. Typical mild injury or impact to the trigeminal nerve causes a short-term pain firing. High impact nerve damage results in constant A-delta (intense pain fibers) firing to occur. Neural injuries involved in trigeminal neuralgia are about in the middle, between soft impact and intense impact.

After the destruction of trigeminal afferent fibers, there is a definite increase in trigeminal efferent activity. This abnormal activity results in a sensory condition known as "dysesthesias", a burning itchy feeling which causes a patient to constantly scratch or rub at their faces. The hypothesis formed from this occurrence is that a decrease in
segmental (afferent) inhibition somehow causes the motor pathways to overact. The afferent inhibition factor is the primary hypothesis to the "trigeminal neuralgia, CNS pathogenesis theory".

PATHOGENIC CONCLUSION: Data from both schools of thought, the PNS theorists and the CNS theorists, have led to a pathogenic hypothesis involving a combination of both theories.

Continuous irritation to trigeminal afferent fibers causes abnormal afferent activity to be received by the CNS. This produces a state of low-threshold "neural-mechanical" activity to be lead to the trigeminal nuclei, thus producing the painful tic douloureux attacks.

This information is derived from the most recent, accurate scientific research performed. Basically, the most viable hypothesis regarding the cause of trigeminal neuralgia is that it has a peripheral cause, but, the pathogenesis occurs within the central nervous system.
CHARACTERISTIC PAIN PATTERNS:

Pathologic factors which could lead to trigeminal neuralgia are very complex. The condition has many viable explanations, each of which, is atypical of the normal anatomical and physiological characteristics of the trigeminal system.

During tic douloureux attacks, the trigeminal nerve's synaptic mechanism is altered. There occurs a failure in segmental inhibition of the afferent pathway. In other words, the afferent fibers continue to fire even though the initial nerve stimulation is no longer present.

The physical cause of the nerve vulnerability is usually due to a change of the nerve's myelin sheath. In some cases, it is due to demyelination (common in M.S.) while in other cases, physical damage (such as a lesion or tumor growth) may have occurred. Since one of main functions of myelin is to serve as protection to the nerve, the neural fibers are much more apt to be irritated, either physically or physiologically, if the tissue is damaged or degenerating.

The tic attacks could be the result of a reflex phenomenon because the impulse action is initiated by a stimulus and the pain outlasts the stimulus. This is followed by a short remission and then a period of
refraction. The whole reflex cycle ceases when the afferent impulses finally cease. This theory is based on ephatic or cross-stimulation of proximal nerve fibers as a result of demyelination or compression. The reflex phenomenon can continue as long as the axonal tissue is capable of conduction.

When a patient has a tic douloureux attack, there also occurs a charge within the brain stem in the trigeminal nuclei. In the spinal trigeminal nucleus, rostral aspect, there is a marked state of internuncial hyperactivity. Since there is a highly sensitized alteration in the neurochemistry, afferent signals (even though the initial stimulus was not a pain producer) are misinterpreted as pain impulses. The amazing thing about the disease is that there are times when trigger areas can be stimulated without a pain response.

If the specific nerves which convey the messages of abnormal perception are not too severely damaged in structural morphology, there may be hope within a period of time for the patient. Postganglionic nerve fibers of the peripheral nervous system possess remyelinating capability. If the vulnerable nerves are again insulated, by regenerated myelin, ephatic conduction is less likely to occur. Thus, pain-free remission is a strong possibility if vulnerable nerve fibers were the etiologic factor.
Even though the previous information is based on very viable theories and much of the stated data support actual clinical cases which lead to successful alleviation of patient suffering, there are no consistent trigeminal or brain stem pathologic findings that distinctly lead to tic douloureux. This statement is necessary because there have been many postmortem findings, such as demyelination, tumor growths and lesions in patients who had not experienced any history of trigeminal neuralgia.
SPECIFIC LOCATION OF THE PAIN:

Clinical data gathered, regarding a large number of case histories of trigeminal neuralgia, lead neurologists to the conclusion that there is no set pain location specifically found in every subject.

The disease is, however, commonly found to affect only one side of the face. There are some patients who do suffer bilateral pain, however this is a relatively small percentage. Bilateral tic douloureux is more common in family gene pools and those who suffer from multiple sclerosis, than in the general population. The time lapse for both sides of the face to be affected, if the pain becomes bilateral, usually takes a period of two or more years. It may, in some instances, take a few days or up to 60+ years.

The initial onset of pain experienced by a patient who develops trigeminal neuralgia is usually found to irritate a very small area. As the disease progresses, it may spread upward, downward, vertical or in an oblique direction. It may even affect more than one nerve track, but usually does not involve an entire dermatome.

Henderson (1967) concluded some distinguishing data from the treatment records of his patients. He designated two regions of the face as common sensitive areas for tic
tic douloureux attacks. The data gathered consisted of 650 clinical cases that he treated over a period of more than 17 years. He determined the most common area to be the "mouth-ear zone" and the less frequent area to be the "nose-orbit zone". The mouth-ear zone cycle usually originates in the superior and inferior dental areas, specifically, proximal to the canine and premolar teeth. Its progressive cycle of pain travels either toward the ear or from one gum to the other. The mouth-ear zone of pain was determined in 62% of his patients. The second region of pain, designated by Henderson, was found to occupy the nose-orbit zone and was diagnosed in 33 percent of his patients. The nose-orbit pain cycle emerges in the superior gum area much more often than in the orbit. Its pain travels upward, toward the nasozygomatic area or to the temporal or nasal canthus. Henderson found that tic douloureux pain very rarely originated behind the orbital cavity or would spread from that region toward the upper gum area. He never ranked this in his percentage data because of its extremely low incidence. Even though pain areas could move within a region, with time, in 95 percent of Henderson's case studies, the sensitized areas remained within the pain zone in which they originated (even after follow-up examinations of over 20+ years).

As far as general pain location is concerned, trigeminal
neuralgia is more commonly found to affect the right side of the face more than the left side by a 3:2 ratio. The trigeminal nerve divisions most commonly affected are the maxillary and mandibular (V2 and V3), either V2 or V3 alone (being the second most common) and the ophthalamic division (V1) being affected in the rarest of circumstances.

PAROXYSMAL PAIN: trigeminal neuralgia is classified as a "paroxysmal" disease. A paroxysm is a seizure-like attack of pain. The paroxysmal pain usually lasts between a few seconds to a few minutes in time. The painful spasms reoccur after brief periods of freedom. The cycle of pain and relief, pain and relief, can last over a period of minutes, hours, days or even months. If the disease is left untreated, the paroxysm attack periods last longer and painfree intervals become shorter. There have been cases of tic douloureux attacks lasting for years, until surgical intervention was initiated. Periods of paroxysm remission can last for a day, week or even up to twenty years or more.

Though the pain attacks commonly occur and diminish usually over short periods of time, the pain emitted during tic douloureux attacks is described as being the most excruciating pain known to man. Women who have suffered from trigeminal neuralgia have declared that "tic attacks" produce more pain than child bearing. There have been very few instances of tic douloureux cases where the patient
experienced pain at an extremely low level.

Upon cessation of a pain attack, it is not uncommon for the attacked area to become "reddened" for a short period of time. The patient frequently may experience a subtle warm or burning sensation which will shortly pass. Nasal congestion, salivation, watery eyes or even a runny nose may also be experienced post-paroxysm.

TRIGGER AREAS: Trigeminal neuralgia pain attacks are commonly elicited by light or gentle tactile stimulation to an area of the skin which is supplied by a sensory nerve with abnormal neurophysiology. Instead of transferring messages of light touch sensation, the disorder causes jolts of intense pain instead. The cutaneous sensitive areas on the surface of the face, which when stimulated provoke this reaction, are known as "trigger areas."

Common normal stimuli which can cause tic douloureux paroxysms are shaving, eating, talking, wind, nose blowing, brushing teeth, washing the face and movements of the tongue...ect., even hair combing can be a stimulus in a few instances (if the trigger area is supplyed by a temporal branch of the ophthalamic nerve).

The more frequent locations of trigger areas include the lateral alar area of the nose, upper eyelid and the upper and lower lip. Occasionally, trigger zones may develop within the oral cavity. In some studies a few trigger areas
in some studies, have been found to exist in gum and tongue tissue. Most tic douloureux patients can usually detect their trigger areas themselves, but in some cases testing must be used to determine them.

The agony experienced by patients with the disease is so intense, that for some, drastic measures are taken to avoid trigger area stimulation. Trigeminal neuralgia victims have been known to refuse to administer common hygiene practices such as face washing, tooth brushing, shaving and even hair combing. In very extreme cases, there have been patients who went as far as refusing to eat. Suicide cases due to the disease have even been recorded in chronic cases.

The amazing thing about trigeminal neuralgia is that even though pain attacks can be initiated by a subtle stimulation factor (and sometimes no stimuli at all), tic douloureux attacks generally do not occur during sleep. This fact is very puzzling, because, it is very common to move physically many times during sleep periods, especially the head, neck and facial areas.

Though trigger areas generally inhabit the same dermatome in which tic pain is experienced, trigger areas may occupy different dermatomes than the areas that experience pain.

The pain attacks of tic doulouruex leave patients virtually helpless against instantaneous pain alleviation,
until the paroxysms finally cease to initiate afferent activity to the brain. This is usually the case, however, in a very few instances. Patients may suppress pain by administering various forms of tactile stimulation directly to the area experiencing pain. Modes of stimulation may include furiously rubbing the area or direct applications of heat. The etiology of this physiological phenomenon is based on the principles of the gate control theory. The gate control theory states: if there are two different types of tactile sensation being experienced, one of the neural sensations may block the transmission of the other. On a physiological level, high frequency myelinated pain fibers are very small and touch-pressure fibers are anatomically larger in diameter. Therefore, the dominant transmission of tactile messages inhibit the pain transmission in the central nervous system. A common example of the gate control theory applied is instinctively rubbing a painful bruise, or rubbing your hands together when they are cold.

PHYSICAL CHARACTERISTICS: During tic douloureux paroxysm attacks there is relatively little to be said regarding visible physical characteristics.

If the diagnosis is of true (idiopathic) trigeminal neuralgia, one of two sensory phenomenons may accompany the tic. The first condition, a lowered sensitivity to ectopic pain, is known as hypalgesia. The second condition is just
the opposite. It involves an increased sensitivity to ectopic irritation (hypergesia). If one of these conditions becomes present, it will diminish once the pain attack has stopped.

If the disease is purely of symptomatic nature, physical signs may include a lowering of corneal reflex reaction.

In a few cases, patients have experienced paroxysm attacks with facial spasms. This condition is referred to as "tic convulsif". The facial convulsions and the tic attacks do not occur at the same time, but they do involve the same side of the face.

DIAGNOSIS:

Symptomatic trigeminal neuralgia is diagnosed when the characteristic pain paroxysms of tic douloureux exist and either lesions or areas of trigeminal nerve demyelination are the cause. The lesions may be a result of vascular compression, tumor growth or trauma. Demyelinated areas may be the result of progressing multiple sclerosis (M.S.).

Actual trigeminal neuralgia (etiologic) is diagnosed when the typical pain characteristics of tic douloureux occur and none of the causes of symptomatic tic douloureux are present. If the diagnosis is actual trigeminal neuralgia, the etiologic mechanism of the disease is a
dysfunction of normal nerve physiology.

PAIN OFTEN MISTAKEN FOR TRIGEMINAL NEURALGIA: Chronic facial pain disorders are often misdiagnosed, due to the complexity of factors that can cause irritation in the head and neck region. Actual diagnosis of trigeminal neuralgia is made after being examined by a trained neurologist, who are more familiar with specific pain neuropathies and neuromorphology.

Types of pain experienced in the face may often times be mistaken for tic douloureux. These disorders may include temporomandibular joint dysfunction (TMJ), atypical facial pain, mygrainous facial neuralgia, cluster headaches, postherapeutic neuralgia and glossopharyngeal neuralgia.

Temporomandibular joint dysfunction, whose etiological cause is an imbalance of temporomandibular joint movement produces a constant pain. Factors which contribute to this condition may include trauma, degenerative arthritis, bursitis and teeth grinding. T.M.J. can affect people of all ages. Masticatory movements primarily initiate the pain, the patients often experience recurring headaches; however, a very notable factor differentiates this condition from trigeminal neuralgia, because the pain associated with T.M.J. is not induced be tactile stimulation.

Atypical facial pain occurs commonly in individuals who suffer from chronic depression. Pain is usually
experienced over an entire side of the face, sometimes the whole head may become involved. Uncommon to tic douloureux, atypical facial pain may be experienced outside trigeminal innervation regions, trigger areas do not exist and emotion is the sole cause of the syndrome. The patients who develop atypical facial pain describe their experienced irritation as an aching, dull, burning sensation. Sometimes they may also experience a throbbing over the whole area experiencing paresthesia.

The pain, characteristic of migrainous facial neuralgia and cluster headaches, usually strike its victims during specific times of the day and usually in a consistent pattern. Unlike typical tic douloureux paroxysms, VII (facial nerve) neuralgia and cluster headache attacks usually last for periods of 15 to 30 minutes at the very least. They also are not provoked by tactile stimulation, and pain may be experienced on either side of the face at random.

Posttherapeutic neuralgia primarily involves the ophthalmic division of the trigeminal tract. The pain expresses itself as a constant burning sensation. Sometimes patients describe shock-like paroxysms whenever the eyebrow is touched, on the side of the face where the paresthesia is experienced.

Glossopharyngeal neuralgia is the closest in similarity to trigeminal neuralgia. Glossopharyngeal (IX) neuralgia
also possesses vital areas which can elicit pain attacks. Common causes of glossopharyngeal neuralgia attacks include coughing and swallowing. Since the pain of IX neuralgia is like tic douloureux and is triggered by tactile movement, it may at times be hard to diagnose. However, there is a difference between the two; glossopharyngeal neuralgia usually elicits pain in the tonsillar region and tonsillar pain in trigeminal neuralgia is practically unheard of.

ASSOCIATION WITH MULTIPLE SCLEROSIS: There seems to be a high incidence in those who develop multiple sclerosis to also become afflicted with trigeminal neuralgia.

M.S. is a progressive demyelinating disease of the central nervous system. The onset of the multiple sclerosis usually occurs between the ages of 15 - 30. The first periods of M.S. activity usually last a very short time, between a few days to a few weeks. Symptoms experienced during its clinical course include: clouded vision, loss of sight in part of the visual field, paresthesia and pain during eye movements, and a combination of numbness and paresthesia on the face and all affected parts of the body. After the original onset goes into remission, the patient seems relatively normal again, however, future periods of remission shorten and motor control and the affected parts of the body slowly degenerate. As the demyelination process continues, palsies develop throughout the periphery due to
lack of neuralconduction.

The by-product of the demyelination process of multiple sclerosis is black tarrish lessions called plaques. Plaques are produced only after proximal oligodendrocyte cells die. It is by necrosis of these cells that myelin is no longer produced.

The vulnerability of the sclerotic, non-myelinated nerve axon leaves it more susceptible to irritation. Thus, the possibility of developing symptomatic trigeminal neuralgia is heightened. There is a 1 to 8 percent incidence of patients who suffer from trigeminal neuralgia to also be afflicted with multiple sclerosis and vice versa.
CLINICAL TREATMENT

MEDICAL / MEDICINE-DRUG THERAPY: Due to the great intensity of pain experienced during tic douloureux paroxysms, preventative intervention must be taken. Pharmacological measures are first applied, and, if they are ineffective, surgical measures may be initiated.

CARBAMAZAPINE (TEGRETOL): Carbamazapine is the number one drug used in pharmacological treatment of tic douloureux. It is often used during diagnostic testing in determining if a patient has trigeminal neuralgia because, in most cases, tic douloureux paroxysms will diminish upon initial carbamazapine therapy. Carbamazapine commonly only suppresses two pain disorders of the face, trigeminal neuralgia and glossopharyngeal neuralgia. Since glossopharyngeal neuralgia can usually be distinguished from trigeminal neuralgia a specific pain diagnosis is more evident.

At a metabolic level, tegretol abolishes the failure of segmental inhibition in the trigeminal nerve, thus, causing the cessation of nociceptive impulses in the nucleus oralis. With segmental inhibition reestablished, normal circuiting of afferent and efferent pathways are once again maintained.
Upon initial administration of tegretol it usually takes approximately 12 to 48 hours to become effective in pain alleviation. If carbamazepine intake is ceased, pain paroxysm will again become active. After time, tolerance to the drug may occur, and after prolonged treatment, carbamazepine may reach potentially toxic levels in the bloodstream. Therefore, careful monitoring must be maintained.

Carbamazepine is initially introduced to the body in small amounts, beginning with doses ranging from 100 to 200 milligrams, twice a day. The medication is increased by 200mg every 48 hours, until an effective dose level (EDL) is established or side effects are encountered. The average EDL of carbamazepine varies between 600-800mg a day. If pain is not suppressed once a patient's blood tegretol level reaches maximum non-toxic levels, the drug is eliminated from treatment and drug therapy is ruled out as a means of tic douloureux suppression.

Side effects which may be experienced from carbamazepine include drowsiness, dizziness, gastrointestinal distress, nausea, anorexia, giddiness and ataxia. Some of these characteristics may only be temporary and cease as the body adjusts to the presence of the drug. It must also be stated that if tegretol therapy is discontinued, the patient must be slowly weened from the drug. Abrupt tegretol elimination
has been known, on rare occasions, to cause seizures.

BACLOFEN: Baclofen treatment has also had been successful in its application to trigeminal neuralgia therapy. Baclofen, like carbamazapine, takes an active role in reestablishing normal neurophysiology in the trigeminothalamic tract. Both drugs alleviate the nociceptive reflex action mechanism which provokes trigeminal A-delta firing.

Initial administration ranges between 5 to 10mg every 48 hours, added until paroxysms diminish or side effects occur. Stabilization of baclofen is usually achieved at an EDL between 50-60mg per day.

The possibility of experiencing side-effects with baclofen is much lower than with carbamazapine. Side effects which may be experienced with baclofen treatment include; drowsiness, disiness and gastrointestinal disturbances. Initial side affects experienced are usually temporary. If baclofen is discontinued, after long-term use, it must be eliminated at small doses at a time, or the patient may experience seizures or hallucinations.

PHENYTOIN: Phenytoin has the lowest metabolic action in trigeminal neuralgia pharmacotherapeutics. Its application in tic douloureux treatment is to be taken in conjunction
with baclofen. The synergistic action, created by the ingestion of the two drugs, is required if the patient cannot tolerate carbamazapine and pain suppression is not achieved by baclofen alone.

Average phenytoin dose intake varies between 150-200mg twice a day.

Side effects which may be experienced with phenytoin therapy include drowsiness, double vision (diplopia), dizziness and ataxia. If any of these side effects persist, they are usually only common in high doses. If phenytoin is used in high dosage over a long time frame, irreversible cerebellar damage may occur in rare occasions.

INITIATION OF MEDICATION: The objective of successful drug therapy is to achieve optimal therapeutic results while subjecting the patients to the least amount of risks and complications. Therefore, drugs that present the least amount of risks are the first to be employed upon treatment. If the drug is ineffective in reaching the therapeutic goal, other medications must be considered.

Typically, the first medication introduced during the treatment of trigeminal neuralgia is baclofen. If pain paroxysms do not cease, carbamazapine is the next administrated. If baclofen or tegretol do not suppress the paroxysms, an application of both drugs is the next attempt.
If the patient has a tolerance to carbamazapine, a combination of baclofen and phenytoin are then induced. If, after all three drugs are administered throughout testing and pain alleviation is not achieved, drug therapy is considered ineffective in treating the specific case. Surgical manipulation must now be considered.

SURGICAL INTERVENTION: If prolonged attempts at treating trigeminal neuralgia with medicine are unsuccessful, surgical intervention must be regarded as an alternative means of temporary or permanent pain alleviation.

Even in patients who measure a therapeutic response to drug therapy, surgery may present more beneficial results. Surgery may be a better alternative, because of the unpleasant side effects and possible irreversible damage which may occur with long-term pharmacotherapeutic treatment.

Some of the surgical techniques are so minor that they are performed routinely on an out-patient basis, and may require only a local anesthetic. However, in other clinical cases, a general anesthetic may be applied for up to 12 hours prior to the procedure and may be continued in diminishing doses of up to 48 hours post-operative time. The latter example is considered a major in-patient operation.
ALCOHOL BLOCK: The alcohol block technique, in the treatment of trigeminal neuralgia, is the quickest, easiest means of minor surgical intervention. Briefly speaking, this method involves a percutaneous (through the skin) ethanol injection directly into either the semilunar ganglion, or a trigeminal peripheral branch. Since the injection is of pure alcohol, the neural tissue will undergo partial necrosis. However, over time, like other peripheral nerves, the endoneurial fibers within the myelin sheaths, will be resupplied with viable, conductive tissue. Thus, tic douloureux attacks may once again return. Even though paroxysms usually reoccur, the technique is so quick and at such low risk to the patient, that it is an extremely popular choice by both patient and surgeon. If the alcohol block attempt is unsuccessful, the next procedure may be a rhizotomy or a microvascular decompressive operation. This Hypothesis is concluded from the alcohol block failure, since the neurophysiological paroxym-eliciting region is not distally peripheral, the cause must be proximal to the CNS.

MANIPULATION: Typically, the alcohol block is performed at the origin of the nerve which supplies the tic douloureux trigger areas. Therefore, all peripheral branches of the injected nerve will be completely without sensation, until, with time, regeneration occurs. For example, if trigger areas are located in the submandibular ganglion region, the
point of injection, typically, would be proximal to the lingual junction of the inferior alveolar nerve. Thus, the inferior alveolar, submandibular and submental regions would be without sensation.

The local anesthetic should be applied directly to the target area of the ethanol injection, because, it will prolong the post-operative state of numbness. There must be a reasonable time frame allowed between the anesthesia application and the alcohol injection. The anesthetic can mix with the ethanol, causing the alcohol to become diluted.

The average volume of ethanol used in the alcohol block technique is approximately 0.5cc. Dosage may vary, but it never exceeds 1.5cc per injection site. If more than one injection target is required, the consecutive injection should be initiated after at least 24 hours pass. All injections should follow this rule, regarding alcohol block therapy. For example, if 3 injections are required for treatment, at least 72 hours should lapse between the first injection and the third injection. This time period is necessary because some patients experience "tic-like" pain for at least 24 hours prior to pain alleviation.

During surgical manipulation, care must be taken during the injection, because, if an artery (which supplies the nerve) is injected, unwanted complications may occur. Since the artery usually runs the same path of the nerve, and
continually branches off more tributaries to the nerve, the excess alcohol will keep flowing through distal parts of the nerve, too. The potency of the ethanol may initiate an endothelial break down and additional lesions may result.

Periods of pain freedom after alcohol block therapy vary in clinical studies. Some freedom lasts only a very short time, other cases have had remissions lasting over thirty years. It seems that the outcome of the duration is principally based on the regenerative properties of the manipulated nerves. Therefore, the faster the growth rate, the shorter the absence of pain.

Paresthesia may be experienced after treatment but usually subsides with time (common in nerve manipulation). Other complications that may also pass include ocular muscle palsies and general weakness of the face. In some cases, herpes blisters may break out around the buccal-oral area.

The major change that patients have to adjust to is desensitized facial regions. Since the manipulated nerve's (or nerves') conductability has been erradicated, the priorly innervated areas will be completely numb.

RADIOFREQUENCY GANGLIOLYSIS (THERMAL DESTRUCTION): Like the alcohol block treatment, radiofrequency gangliolysis therapy is a relatively low-risk procedure. The objective of the operation is to destroy nerve fibers within the
semilunar ganglion or the trigeminal sensory root by using direct applications of heat. An advantage that this procedure has over the alcohol block technique is that not all nerve fibers within the nerve sheath have to be destroyed. In fact, in many cases of radiofrequency treatments, the patient may be pain free and may still possess sensation in the effected trigeminal regions. Another important factor favoring this therapy is that its thermal application is limited to the proximal target structure, where in the alcohol block technique, the risk of ethanol spreading to ectopic viable tissue is relatively high.

MANIPULATION: The patient is given a local anesthetic while laying in a supine position. Once the anesthetic is well in effect, a 19-guage, hollow needle is applied percutaneously to the foramen ovale. Once the needle is proximal to the foramen, the patient is given between 30-50mg of methohexitol, for an additional anesthetic reaction. The needle is then moved further, until it is situated superiorly to the basilar groove of the sphenoid bone (clivis). The needle is then retracted a bit, in order not to puncture the carotid artery and to also initiate the flow of cerebral spinal fluid.

When entering the area of the foramen ovale, all three trigeminal posterior root divisions are in close proximity.
During this procedure, a fluoroscope is used in order to determine exact positioning of the needles applied. Placement of the fluoroscopic mechanisms are lateral, in reference to the head.

Once the 19 gauge needle is in the exact location, it is then withdrawn and replaced, by a radiofrequency electrode. The end of the electrode needle is capable of producing be between 5-10Hz. The thermally manipulated area, may include either all three trigeminal divisions, or just one. The electrode needle is curved in order to give the surgeon precise control upon gasserian penetration (since the nerve roots run a curved path within the ganglion) and in order to affect one division at a time.

Exact intraneural electrode placement is achieved (with observable reactions of the face) during slight electrical stimulation from the electrode. For example, if the radio frequence needle tip is in contact with an ophthalmic (V1) fiber, the ocular muscles may twitch, if the needle contact is with a sensory fiber of the mandibular division (V3), either paresthesia or "tic" pain may be expressed somewhere within its innervation region. This surgical testing determines not only morphographic intraneural location, but, which areas must be manipulated during the procedure. Throughout the operation, the needle is moved only 3-5mm at a time. During this process, contacted fibers (eliciting pain
to the face) are heated with radiofrequency stimulation, until the expressed "tic" is ceased. During gasserian ganglion penetration, needle depth must not exceed 5mm posterior to the clivis. When thermal stimulation is initiated, the surgeon should take care to avoid motor fiber contact. It is often inevitable to have some motor contact because the objective of R.F. therapy is to achieve pain relief without muscular palsies.

After the thermal applications are ceased, methohexital is again injected and the patient is awakened. The surgeon does tactile testing to the face in order to determine if additional R.F. treatments are necessary. The patient is carefully monitored for at least 15 minutes once hypesthesia is achieved. If the patient is stable, hospital release is usually the day following the procedure.

Successful percentages range from 78%-100% of treated patients. Cases of reported failures usually result from morphologic complications of the foramen ovale region.

Undesirable effects of radiofrequency gangliolysis may include muscle palsies, corneal hypesthesia, anesthesia dolorosa and neuroparalytic keratitis.

PERIPHERAL NEURECTOMY: The peripheral neurectomy approach to the surgical treatment of trigeminal neuralgia is a relatively low-risk procedure. Even though the mode of
anesthetic application is general, the dosage is so low that this therapy is widely recommended, even to elderly patients and those who have chronic health problems. Another reason for this procedure's popularity is that unpleasant side effects, such as ocular motor palsies, anesthesia dolorosa and corneal anesthesia (which are possible side effects from other neuralgia treatments) have not been reported in peripheral neurectomy follow-up studies.

The basic approach to this procedure is to treat the nerve eliciting the trigger, within the trigger zone and not the area where pain paroxysms are experienced. The objective of surgical manipulation is to completely cut the nerve or nerves that innervate the trigger zone and whose path leads to the region of paroxysm attacks.

The only notable draw-back of the peripheral neurectomy procedure is that since post ganglionic peripheral nerves possess regeneration capabilities, the nerve fibers will again grow and paroxysms will eventually return. Therefore, this technique will need to be repeated as often as pain reoccurs unless alternative treatment methods are considered.

MANIPULATION: Surgical manipulation is achieved by incising the skin, reflecting tissue to expose the trigger nerve, and making a complete sectioning. In cases where the trigger area is proximal to a foramen, the nerve should be sectioned as far back within the foramen as possible. This
measure is taken to prolong the regeneration of the nerve to its usual length. For example, if the affected nerve is the supraorbital nerve, the site of sectioning will occur within the supraorbital foramen. If the affected nerve is within osseous tissue, a hole may be drilled in order to expose the nerve for proper cutting.

This procedure relieves pain for approximately 2-3 years (average), however, some patients exceed 5 years of paroxysm freedom. If the pain attacks are still experienced after the neurectomy procedure, the administration of "pharmacotherapy" may suppress pain even if the medications were ineffective before surgical measures were initiated. If paroxysms do not cease following surgery and drug therapy, the neurologist must consider the etiologic factor to be within the trigeminal sensory root. If this is concluded, the following surgical alternatives may include a rhizotomy, a tumor removal or a vascular decompression procedure.

RHIZOTOMY (SECTIONING OF THE SENSORY ROOT): In cases where the etiological instrument of a given case of trigeminal neuralgia is not defined through the use of medication, radiofrequency gangliolysis, alcohol block therapy, M.R.I. (magnetic resonance imaging) screening and sometimes an M.D.V. (microvascular decompression) operation, the next treatment approach (if neoplastic formations are not
discovered) is directed at the trigeminal root.

Even though the rhizotomy approach is 80%-90% effective, the procedure is not a common means of trigeminal neuralgia treatment. The cases where the rhizotomy method is applied results from a failure to find a vascular or other type of neural compression during a MVD procedure. The incidence of the preceeding condition ranges from approximately 14-21 percent in microvascular decompression procedures.

MANIPULATION: Two approaches to the trigeminal rhizotomy technique exist. These surgeries include the subtemporal procedure and the posterior fossa approach. Both types of trigeminal neuralgia treatments require a general anesthetic.

Subtemporal entry begins with an 8cm incision and a 2cm deep cut ventral to the auditory canal (externally). Once the superficial tissue is reflected, an incision is also made in the temporalis following the same direction as the muscle fibers. When the temporal bone is exposed and a circular hole is drilled, a 4cm craniotomy is performed. Dissection is performed extradurally. The foramen spinosum is protected by applying either cotton or wax directly into its opening, and then the middle meningeal artery is divided. The procedure continues to the foramen ovale. After some dural tissue is dissected off the semilunar ganglion, the three trigeminal branch origins are exposed. The order of branch
encounter begins with the mandibular root (V3), the maxillary (V2) root and finally the ophthalmic (V3). It is very important not to apply traction in the region around the gasserian ganglion because injury to the facial or the greater superficial petrosal nerve may result. Next, an incision is made in the dural tissue (posterior to the gasserian ganglion) to expand the area of neural manipulation. As the incision is made, some cerebral spinal fluid (of the dural cistern) will be released. The following and most critical step of the rhizotomy procedure is the actual root fiber sectioning. The sectioning is initiated proximal to the lateral aspect of the semilunar (gasserian) ganglion. As the fibers are sectioned, the ophthalmic fibers are left unmanipulated, unless the tic douloureux paroxysms are experienced within the ophthalmic division. It is important that the motor root be not sectioned because palsies and atrophy, especially of the temporalis and other masticatory muscles, may occur. After proper sectioning is complete, the dura is resectioned and the remaining incisions are also closed.

Posterior fossa entry entails a retrogasserian approach to sensory root sectioning (the same entry approach as M.V. D.). When the root is exposed, it is cauterized then sectioned. Like the subtemporal approach, the ophthalmic nerve is usually spared and the V motor root is avoided.
Since, in both procedures, the entire sensory root system is not always destroyed, tactile sensation may be saved in many areas of the face.

Clinical data regarding the subtemporal approach shows that the percentage of success is approximately 85%, deaths from the procedure ranges from 0.8%-1.6%, facial atrophy is 7%-8%; mild paresthesias at 56%; chronic (severe) paresthesia 5% and neuroparalytic keratitis ranks at 15%.

Clinical data surveys of posterior fossa approach: 80%-90% success, 1%-2% death caused by surgery, 28% corneal reflex weakness (50% minor facial herpes), 8% severe dysthesias, 20% mild paresthesias, 3%-8% minor facial atrophy, 10% hearing impairment (in 50% percent of these patients, the acoustic loss is permanent). In rare instances, the patient may be more prone to hematomas and vascular complications.

Both procedures have very similar data results and the objective of the two techniques is the same. Only a few post-operative effects differentiate the subtemporal from the posterior fossa surgeries. In subtemporal entry cases, hearing impairment, or loss, has not been recorded. Patients who have undergone the retrogasserian procedure have no reported cases of postoperative keratitis.
MICROVASCULAR DECOMPRESSION OF THE TRIGEMINAL ROOT: The MVD operation has brought success in treating patients with symptomatic trigeminal neuralgia. The procedure may be initiated if the patient is refractory to pharmacotherapeutic measures and minor surgical techniques. Another factor considered, in many cases, is the health and age of the patient being considered as a candidate for surgery. Since the microvascular decompression operation is a major surgery, the mortality rate is higher to those of high health risk and the elderly.

Pre-operative testing involves roentgenography, CT scanning and angiograms. The major objective of these tests is to study the morphological terrain of the posterior cranial fossa. The goal is to define any vascular, neural or osseous abnormalities which, if they exist, may be the etiologic cause of the tic douloureux. There are instances, however, that these anomalies may occur and be undiscovered during testing. This occurs in a very low percentage.

MANIPULATION: The objective of MVD is to release a blood vessel which is compressed between the trigeminal sensory root (portio major) and the pons (the vessel must be released to end the pain firing in the portio major).

Twelve hours before operation time the patient is administered 10mg of dexamethasone (an anesthetic), in an intramuscular injection. The anesthetic is continued
throughout the procedure and 48 hours post-operative time with dosages being tapered by 4mg every six hours. The patient is situated in a lateral decubitis position. The legs are secured with ace bandages, intubation is as well as a central venous line and bladder catheter. The head is also secured in a "pin head holder". To monitor the heart properly, ultrasound is applied above the cardiac region. The head is then bent until the chin is in close proximity to the sternum. The head is turned slightly to the side. A 15-20 minute I.V. line of 50g of mannitol is administered shortly after intubation is stable.

Necessary head shaving is done and the head piece of the operating table is moved into a horizontal position in order to provide a rest for the surgeon's elbows.

A ventral retromastoid incision is produced, approximately 7cm in length, 2cm medial to the mastoid process of the temporal bone. The incision is cut in a superior frontal path lateral to the hairline. The frontal part penetrates through to the calvarial tissue and the posterior part will penetrate deeper muscles. These muscles are cauterized as they are incised. Muscles and fascia are reflected from the calvarium, to prepare for a craniectomy.

The craniectomy, 3cm by 3cm, is then performed in the area of the superior lateral region of the posterior cranial fossa. The mastoid air cells are exposed from this removal
and are coated with wax for protection. Now that the dura mater is exposed efficiently, a U-shaped incision is cut. The incision runs from the superior angle of the lateral sinus down toward the periphery of the inferior aspect of the calvarial craniectomy, back in an occipital direction, then up again, still following the direction of the craniectomy. The dura is then sutured in the two inferior lateral aspects of the cut tissue. This flap is now pulled back, in a craniad fashion to expose the brain and cerebellum. If there is not enough room for surgical manipulation, more bone may be removed.

The cerebellum is now retracted, from the lateral aspect of its superior surface. A binocular microscope, using a 250 focal length objective, is now applied for the rest of the procedure.

In cases involving trigeminal neuralgia of the mandibular (V3) nerve, the target for vascular manipulation is usually the superior cerebellar artery (see surgical illustration). Its course usually follows a craniad direction around the pons, and bifurcates. One of the branches, either the medial or lateral branch, is caught beneath the trigeminal root's pontine entry. This aberrant artery location causes the compression of the portio major, which in turn, initiates the paroxysm reactions of tic douloureux.
With the cerebellum retracted, the area of the pontine-trigeminal sensory root junction is cleaned by careful dissection, until the morphologic structures are clearly denoted. Next, the trigeminal root is stretched off the pons by running a tiny piece of surgical tubing directly around it. Just enough tension is drawn against the root to allow for the trapped artery to be removed by micro-operative surgical forceps. With the superior cerebellar artery still withdrawn, the trigeminal root is then released from its restrain, to its original position. A piece of tiny teflon is next applied to the lateral side of the root. The superior cerebellar artery is then placed on the lateral side of the portio major with the teflon material laying between the two structures. The teflon acts as a shock buffer and absorbs some of the vascular pressure form the artery.

If the tic douloureux pain paroxysms occupy an area which is innervated by the maxillary (V2) division, the target vessel of manipulation is the superior petrosal vein. The operative approach is basically the same as the superior cerebellar technique.

Trigeminal neuralgia within the ophthalmic (V1) division, requires vascular manipulation of the inferior cerebellar artery. This procedure follows the same basic guidelines of the other M.V.D. techniques. The success rate of trigeminal microvascular decompression is approximately
88%-89%. Successful outcome depends on the surgeon finding the neural-vascular constriction and effectively releasing it. If pain is still present following the operation, small administrations of diphenylhydantoin usually suppress it. In most cases, the irritation will cease in about 6-8 weeks. This diminishing pain syndrome may occur in up to 40% of clinical cases.

Post-operative surgical complications, which may arise, include air embolism (usually not serious) 29%, facial herpes 21%, temporary hearing loss 15%, permanent hearing loss 3%, serous otitis media 7%, aseptic meningitis 6%, stitch abscess formation 6%, cerebellar hematoma 1%, subdural hematoma of the posterior fossa 1%, hemispheric subdural hematoma 1%, cerebellar infarction 2%, pneumonia 2%, pseudomeningocele 2%, bacterial meningitis 2%, and death 1%.

Trigeminal neuralgia post-operative recurrences are possible. This may result from improper manipulation, or in some cases, failure of the surgeon to discover a tiny compression. Tic douloureux recurrences require a second operation. Target vessels manipulated in the second attempt include: the superior cerebellar artery in 27.7%, anterior inferior cerebellar artery 11.11%, anterior inferior cerebellar vein 11.11%, superior cerebellar artery and vein 16.66%, superior cerebellar and anterior inferior cerebellar arteries 5.55%, anterior inferior cerebellar vein and
arachnoid cyst 5.55%, a prothesis caused irritation 5.55%, slipped prosthesis 5.55% and a reactive prosthesis at 5.55%.
PROGNOSIS:

If a patient experiences extreme pain in spasm-like attacks which fail to respond to analgesics and are not caused by pain-evoking stimuli the pain paroxysms cease and within minutes return again until the cycle finally diminishes. A non-painful, proprioceptive sensation (possibly a tingling feeling) is experienced somewhere on the face prior to the pain attacks. These conditions may indicate a probability of trigeminal neuralgia, as the diagnosed syndrome.

If, after proper neurological diagnosis, the patient truly has trigeminal neuralgia, the clinical longterm outcome may at times vary. In most cases, however, the pain paroxysms last longer when they occur and pain-free periods shorten.

Since its prevalence in patients who suffer either nerve trauma or a demyelinating disease is high, the patient's neuromorphological viability status plays a major factor in the outlook, if of the trigeminal neuralgia is caused by the vulnerability or injury of the trigeminal nerve.

If nerve demyelination or plaque deposits are the cause of the disease, areas of neural short-circuiting will spread throughout the trigeminal periphery to future areas of
demyelination.

If trauma is the causing agent, there may be some means of surgical intervention applied to alleviate the condition. The condition may also be produced by a nerve compressing structure, such as a tumor growth or vascular impinging. If tumor proximity is the cause, the tissue may be removed. After vascular surgical procedures, vascular compression may be relieved. If these symptomatic trigeminal neuralgia initiating causes are not abolished, the tic douloureux paroxysms will continue.

These prognostic data are based on the vast majority of cases. However, there have been reported cases of "symptomatic" and "idiopathic" trigeminal neuralgia patients who have experienced short-term or long-term pain-free periods.
CONCLUSION:

The treatment of trigeminal neuralgia initially involves drug therapy. If pharmacotherapy is ineffective, or unbearable side-effects are encountered, surgical manipulation is next step taken and is usually at least 80%-90% effective.

Even though trigeminal neuralgia is not a fatal disease, its pain producing paroxysms are unbearably intense. They have driven some patients, in extreme cases, to commit suicide. Since the exact pathogenic and etiologic mechanisms are not entirely known, continuing research is required to hopefully put this pain syndrome to clinical extinction.
PROFILE OF THESIS WRITING:

Due to the fact that I am very new to writing a large body of literature and scientific research-writing, the thesis report was an extreme challenge for me. Thanks to the guidance of Dr. Richard Doolittle, scientific advisor and thesis consultant, I was able to develop a workable thesis outline for the preceding body of writing. Advise from Glen Hintz, medical illustration professor and "chief" thesis consultant, and Professor Robert Wabnitz, medical illustration advisor and thesis consultant regarding medical illustration literature, was essential feedback as well.

CONCEPTION OF THE TEXT: The objective of the "Trigeminal Neuralgia, Through the Eyes of a Medical Illustrator" text was designed to give an insight to the neural disease, trigeminal neuralgia. The "audience" that the scientific writing is designed for is medical students. The writing, following the scientific text, explains the thesis writing and illustration factors applied, as well as my personal statement and conclusion.

LITERATURE RESEARCH APPLIED: My literature research began during the summer of 1989. I was able to gain access
to Falk Library of Health and Sciences, at the University of Pittsburgh. While at Falk, I utilized books pertaining to peripheral and cranial nerve research. The biggest influence to my research and writing was a text written by University of Pittsburgh professor, from the Department of Neurology, Dr. Gerhard H. Fromm, M.D.. The text is entitled "The Medical and Surgical Management of Trigeminal Neuralgia". I read Dr. Fromm's book, which gave me a good overview of the condition of trigeminal neuralgia. But still, there was much information that was very foreign to me, regarding neurophysiology and neuronomenclature. I was determined to contact Dr. Fromm to request an interview with him. After I made a few contacts with University of Pittsburgh personnel, I was granted an interview by Dr. Fromm.

During the interview, I asked Dr. Fromm many questions regarding trigeminal neuralgia. He was very patient, and, explained things to better help me understand some of his literature, on a level that I could comprehend. He was very kind and stimulating to talk to. I owe him a great deal of thanks.

Among the other sources of text that I utilized while doing my research were "The Physiology of Peripheral Nerve Disease", Dr. Austin J. Sumner (University of Pittsburgh); "Peripheral Neuropathy, Volume II", Dr. Peter James Dyck, Dr. P.K. Thomas, Dr. Edward H. Lambert, and Dr. Richard
Bunge; "The Cranial Nerves", Dr. M. Samii and Dr. Peter Janetta (University of Pittsburgh). A great deal of the "Neuromorphology" section of the scientific writing was researched from "Gray's Anatomy" by Dr. Henry Gray. Netter's "Atlas of Human Anatomy" was also a great help.
Ophthalmic nerve: $V_1$
Maxillary nerve: V₂
Mandibular nerve, V₃
PROFILE OF THESIS ILLUSTRATIONS:

CONCEPTION OF IDEAS: Artistically speaking, portraiture of the human head, throughout history, is regarded as a major discipline among artists. Da Vinchi had the "Mono Lisa"; Rembrandt had his self-portrait series; Rubens had the portrait studies of Elizabeth Brandt; and modern master Andrew Wyeth made the subject of "The Helga Studies" a memorable face among modern art collections. Though I am not the caliber of these great masters, I too share their interest in the artistic expression of human face and figure.

In this project, my objective was to use the human head in a medical context, with neuroanatomy applied. I also chose to incorporate a self portrait into the series as a personal challenge.

OBJECTIVE OF THE RENDERINGS: My goal for the series of illustrations was to indicate neural pathways and specific anatomy in a visually pleasing manner and to use four approaches: portraiture, figure drawing, surgical illustration and anatomy plates.

Plate 1: The self portrait illustration also indicates the orientation of the brain, brain stem, semilunar ganglion, cerebellum, and the trigeminal root.
Plate 2: The neural pathways of the ophthalmic division of the trigeminal nerve (V1).

Plate 3: The neural pathways of the maxillary division of the trigeminal nerve (V2).

Plate 4: The mandibular division of the trigeminal nerve (V3).

Plate 5: The conceptual illustration, denoting the orientation of the figure, cranium, spinal cord and posterior cranial fossa anatomy, including reference to cranial nerves.

Plate 6: The pen and ink surgical illustration illustrates the microvascular decompression technique, common in trigeminal root manipulation, involving vascular impingament. This procedure is perhaps the most popular major surgery involving tic douloureux.

MEDIA USED: Artistic media incorporated into this project are what I would classify as quick media, among other forms of medical illustration applications.

Media which would require much more time in the actual rendering applications may include the air brush, watercolor, acrylic painting and the carbon dust technique.

The applications used in this project include pastel, colored pencil, cartoon color paint and the pen and ink technique.

My goal was to develop quick and competent rendering
techniques for successful full-color illustration. I believe that this was a good objective for me to undertake because my portfolio showed a weakness in my color work. It was missing the punch that it needed to stand alone as color illustration.

I also chose pen and ink for one of the illustrations. My drawing technique tends to lend itself to a linear style, so it would be a more natural media to adopt. Since the pen and ink illustration denotes a complex surgical procedure, I used a clear and simple way to show it. Pen and Ink is a fast media and when done effectively, form can be understood very easily.

The pastel-colored pencil illustrations (Plates 1-5) were rendered using Prismacolor colored pencils, Berol pastel pencils, Nupastel color sticks, HB mechanical pencils, cartoon color paint and touches of titanium white acryllic paint.

TECHNIQUES APPLIED: The self-portrait (brain, cerebellum, brain stem, trigeminal root orientation) illustration shown on plate #1, was rendered in a series of steps.

The first step was to draw a self portrait for reference. This was done from direct observation by using two mirrors, at angles, in order to capture a profile view.
Drawing from direct observation, "life", is a common discipline that I feel not only enhances draftmanship ability, but also enables the artist to observe three-dimensional form. If using a photograph, the artist would see form on a two-dimensional image. This is an essential practice of an illustrator, since models are not always available; however, the traditional practice of "life drawing" is a rudimentary and necessary discipline.

The original self portrait study was rendered using a 4B pencil. It was drawn on news print, sprayed with a fixative and transferred by using tracing paper and pencil tracings onto a piece of peach-flesh Canson paper. Once the graphic image was on the paper, a flesh colored pastel pencil was used to apply an even tone of dark flesh color to the face. The tone was then smoothed, using a sable brush, until a flat medium-flesh color was achieved over the whole area of the face. Next, lighted areas were manipulated into the drawing, using an eraser to erase the dark flesh and to leave the light color flesh of the Canson paper to show through. Shadows of the nose, eye, chin, lip and cheek area....ect., were achieved by applying very subtle tone with a #2 graphite pencil. The graphite was mixed with the pastel color. Care must be taken when using the graphite application because if too much graphite is applied, the paper texture will become waxy and pastel applications will not adhere to it.
As the drawing took shape, the Prismacolor colored pencils were used to reinforce the form of the facial anatomy and to make the illustration stand out better. The colored pencils were also used in the finishing touches of the illustration. A bold contour line was drawn around the portrait and certain anatomical regions were also emphasised. This technique was a new approach for me, but I found it created a more dynamic image.

Many achievements of an artist are developed by trial and error when drawing throughout his or her life. Accidental discoveries are also a major contributor to an artist's technique. I owe much of the success of the thesis illustration series on trial and error and "happy accidents".

The next step of the self portrait was to add the hair. This was done by first applying a brown pastel which was smoothed to create an even brown tone, then brown colored pencils were used to create variations in dark and light.

Titanium white was then applied to add a reflective quality to the eye. After this process, a "fixative" was sprayed onto the illustration.

The final objective of the self portrait was to create an acetate overlay to designate specific anatomy. Wet media acetate was used because the rendering would be done using the pen and ink technique. Once the brain, cerebellum, brain stem and trigeminal root were denoted in the drawing, parts
of the illustration were introduced to the stipple technique, in order to show shadow. "Stipple" is creating dots, which, when successful, will read as tone. More conglomerated areas of the dots will create darker areas, for shadows and darker regions of a illustration.

Plates #2-4: The ophthalmic, maxillary and mandibular divisions of the trigeminal nerve are illustrated in this series.

This series of drawings was a tremendous big break-through for my knowledge of color application. The previously discussed artistic growth through the trial and error philosophy went into full play. I recall roughly eight to ten unsuccessful illustrations during my attempts to devise a successful color formula for bone color and the background color. A major problem was encountered when I tried mixing extravagant colors for bone. This technique does work for some artists, but for me, it was an out of control mess. Being very frustrated, I sought the advise of my fellow classmates and instructors. Sometimes when working continuously on a project, it is good to get feedback from other sources because they may see more clearly any flaws or defects in an illustration. My intuition was correct, because, after evaluation of my flops by my colleagues: Joseph Bloch, Dean Vigyikan and further evaluation from professors Glen Hintz and Robert Wabnitz, I was stimulated
with fresh solutions to the problem solving process for the trigeminal nerve anatomy illustrations.

Advice such as "Keep it simple" from Professor Wabnitz and from Professor Hintz; "Apply the basics of analogous color theory" was essential to solving my problem. It is very easy to become overwhelmed with something that you labor over, sometimes in frustration, and to overlook the simple basics of success. This statement may be applied to many things in life. The conversations that I had with my colleagues, as well as friends, Bloch and Vigyikan, were equally valued. They were more on the humorous side, which was exactly what I needed - a light hearted way to view the challenge and boosted my morale.

After careful personal evaluation and the gathering of ideas from my peers and instructors, a successful color theory was developed. To keep the color composition of the series consistent, the same color background which was used for the self portrait illustration was incorporated into the nerve branch illustrations. The bone color formula was rendered in yellow ochre, for the base color and burnt umber for the shadows and contour lines. Yellow ochre was first applied by using a Berol pastel pencil. Burnt umber prismacolor pencil was added to the yellow ochre decreasing its value. This mixed media application was very successful and possible complications such as the waxy texture, which
can be encountered using graphite and pastel did not arise. Using the same color background color was a wise choice because it kept the orientation drawing (plate #1) and the nerve anatomy illustrations (plates #2-4) consistent. Yellow cartoon color, an opaque bright paint, was very effective in making the nerve branches stand out. For the trigeminal nerve branches that run underneath bone, the burnt umber contour lines were applied in a dotted pattern.

Plate #5, the conceptual illustration, illustrates the orientation of the figure – cranium, spinal cord, cerebellum, view of the posterior cranial fossa and reference to the cranial nerves.

The first step was to render the figure. This, like the self portrait, was drawn from "life" or direct observation. Prismacolor colored pencils were used in blue-violet for the darks and white for the lights. My objective was to keep the figure drawing more consistent with its blue background and render the two other illustration insets in color in order to make them stand out.

The posterior cranial view and the craniectomy, showing the posterior region of the brain, cerebellum, cranial nerves...ect., were rendered in ochre, burnt umber, yellow, beige, pink and white. Pastel pencils were used for the ochre, beige and pink colors, pastel pencils and white charcoal were used for the white color, and cartoon color
paint was used for the yellow color.

I chose a blue background because I wanted this illustration to stand apart from the first four plates in the series. It was a different approach.

The final illustration (plate #6), the surgical procedure, was rendered using, perhaps, my favorite technique, pen and ink. Pen and ink is a very straightforward approach to medical illustration. When applied properly it is very traditional and effective in producing easy to read imagery. It is probably my most natural media, due to the fact that much of the tonal areas that I render, while sketching and drawing, are achieved using linear variations.

This illustration designates the microvascular decompression procedure, as it applies to trigeminal root manipulation. The orientation drawing (upper left of plate #6) shows the initial incision and craniectomy targets for this "retromastoid approach". Rendering "A" denotes the dura matter exposed after the craniectomy is performed. Rendering "B" illustrates the dura matter incised in a U-shaped manner and reflected upward to expose the brain tissue, while the cerebellum is in the action of being reflected backward, in order to make the trigeminal root at the base of the pons visible. Renderings "C-E" show a dissecting microscopic view.

Rendering "C" illustrates the vascular impingament of
the superior cerebellar artery. The dotted line pattern is effective in making the artery read as being under the trigeminal root.

"D" illustrates the manipulation process of lifting the trigeminal root with a retractor and the actual removal of the artery from beneath the root by using forceps.

Rendering "E" indicates the final objective of the procedure. The superior cerebellar artery is placed on top of teflon material which serves as a vascular shock absorber between the nerve root and artery. The tonal looking area, teflon material, of the illustration was achieved using zippatone film, a frisket-like, transparent-adhesive plastic which can be cut to any two dimensional shape and applied to an image. It is used to represent a dark or shadowed area. The film has dot patterns on it and the conglomeration of the dots create an overall area of tone.

All lettering used on the illustrations were applied using rub-off lettering.
ILLUSTRATION RESEARCH: Research done for this project included many illustration references: The "AMI Sourcebook" various "Journal of Biomedical Communication" manuals, advertising "Black Books", pharmaceutical advertisements and studies done by "mentor" illustrators, including Karen Vissor, a personal friend and an inspiration.
ARTIST'S STATEMENT

Becoming a medical illustrator has been my goal for the past eight years. Before I set my mind on making medical illustration a major part of my life, I was very frustrated. Even though drawing had been a hobby since my childhood, something was missing in my spirit, especially during my sophomore year as a Fine Art's major at Edinboro University of Pennsylvania, in 1984.

I became discouraged, because I knew that I was missing something in my life. Though I was talented in the fine art area, my art work no longer served its purpose in satisfying me. Dismayed with laboring over still-life drawings and design problems, and no intellectual stimulation, I finally decided to get out of art all together and use my art as a hobby.

I approached my advisor, Professor James Vredovogue, (Edinboro University, fine art professor) and proclaimed, "I want to change my major". He was concerned because I had made much progress under his instruction. He was not only my trusted teacher, but was also a friend. He advised me not to change my major just then, but, to take a semester off from art courses and take other types of classes, just to see if I could gain an interest in any other field of study.
During the following semester, I took two general biology courses called "Man and Nature" and "Health and Human Sexuality". That 15 weeks stimulated my interest in learning more about the human body and how it worked.

It was also during that particular semester that I became friends with Scott Williams. We were fellow athletes on the "Edinboro University, Fighting Scotts" swim team. Scott was an outstanding athlete and I heard that his reputation as an Edinboro fine art student was phenomenal. I discussed Scott's future artistic plans with him and found that he planned on pursuing a career in medical illustration. This was a new field to me.

A very short period of time passed during the semester and I came to realize that I did have artistic blood in me and even if I tried escaping, I could not let go of my love for drawing. Since I did not have any art courses that semester, I would stay up half the night drawing for my own enjoyment.

My interest continued to grow in biology and the study of the human body. I routinely visited Scott in his studio, asking him more about medical illustration. The following semester Scott and two of his fellow classmates had an art show. When I saw Scott's anatomical and figure studies, I knew then that this was what I wanted to do. I sought Scott's advise many times. He was very helpful and
inspirational.

Another person who was very influential in my interest in human anatomy was Edinboro University art professor, James McMurry. Professor McMurry is, by far, one of the most incredible artists that I have ever encountered. His instruction was very precise, articulate, strict and intellectually challenging. He would challenge your artistic skills to the limits of creativity and accuracy. His figure drawing instruction was excellent. He, like Scott, gave me the much needed academic guidance and advise.

During my medical illustration training at the Rochester Institute of Technology, Professor Robert Wabnitz and Professor Glen Hintz greatly influenced me. Robert Wabnitz is well known among medical illustrators across the country, having done illustrations in Gray's Anatomy and various orthopaedic surgery atlases just to name a few. Professor Wabnitz was very patient with me upon my initial attempts in medical illustrating. I was new to the techniques that he had mastered and he was able to see my perspective as a beginner and worked with me often individually. His most accomplished medical media is the carbon dust technique. The most valuable technique that I learned from Professor Wabnitz is the ability to improvise shadow and light, to make illustrations tonally dynamic and more volumetric. Professor Hintz introduced me to very versatile ways to render medical
illustrations. His accomplishments with air brush, pen and ink, water color, and schematics are very motivating for students to see. Professor Hintz has the ability of making you see things in illustrations that you normally would not see. During my studies under Glen, he was able to see the strengths and weaknesses of my artistic ability. He helped me capitalize on my strong points and improve my flaws. The best thing that Professor Hintz taught me was that I did not have to completely change my drawing technique to do medical art. Unlike many medical illustration colleagues of mine, I tend to favor rendering dark or shadowed areas with line variations instead of solid tone. I thought that I would have to change my whole drawing approach to achieve more success, but Glen encouraged me to use my line style to develop a stronger pen and ink ability.

Professor Wabnitz and Professor Hintz were very helpful in stimulating class-room morale. Their humor also helped to break the ice during a rough day. I was very grateful to become their medical illustration graduate assistant during the 1990-1991 school year.

This "Artist's Statement" is devoted to all those who helped me grow artistically, many are unnamed, but certainly not forgotten.
The "Trigeminal Neuralgia, Through the Eyes of a Medical Illustrator" project has been a very draining experience for me to undertake. I am pleased that I chose a neurological topic to research, because the subject of neurophysiology always intimidated me. Completion of this research gives me a sense of pride.

It is my goal to continue my growth as a medical artist. Learning more about art enhances my creative lust, learning medical information stimulates my ego, being a medical illustrator gives me integrity.
BIBLIOGRAPHY


