# A STUDY ON EFFICACY OF THE INTRATYMPANIC INJECTION OF STEROID IN THE TREATMENT OF SUBJECTIVE IDIOPATHIC TINNITUS

A dissertation submitted to the

TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY, Chennai

In partial fulfilment of the Regulations for the award of Degree of

MS BRANCH IV

(OTORHINOLARYNGOLOGY)

Reg No: 221914106

DEPARTMENT OF OTORHINOLARYNGOLOGY

MADURAI MEDICAL COLLEGE, MADURAI

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# CERTIFICATE I

This is to certify that the dissertation entitled **"A STUDY ON EFFICACY OF THE INTRATYMPANIC INJECTION OF STEROID IN THE TREATMENT OF SUBJECTIVE IDIOPATHIC TINNITUS"** is a bonafide record of work done by **Dr.E.SELVAPRIYA** in the Department of Otorhinolaryngology, Madurai Medical college and Govt. Rajaji Hospital, Madurai in partial fulfilment of the requirements for the award of the degree of M.S. Branch IV (Otorhinolaryngology), under my guidance and supervision during the academic period 2019 – 2022.

I have great pleasure in forwarding the dissertation to the Tamil Nadu Dr.M.G.R. Medical University.

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## CERTIFICATE II

This is to certify that this dissertation work **"A STUDY ON EFFICACY OF THE INTRATYMPANIC INJECTION OF STEROID IN THE TREATMENT OF SUBJECTIVE IDIOPATHIC TINNITUS"** of the candidate **Dr. E.SELVAPRIYA** with Registration Number 221914106 for the award of degree of M.S. Branch IV in the branch of Otorhinolaryngology.

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## DECLARATION

I, **Dr.E.SELVAPRIYA**, solemly declare that the dissertation entitled **"A STUDY ON EFFICACY OF THE INTRATYMPANIC INJECTION OF STEROID IN THE TREATMENT OF SUBJECTIVE IDIOPATHIC TINNITUS"** is a bonafide record of work done by me during the period of November 2020 – November 2021, at Madurai medical college and Government Rajaji Hospital, Madurai.

This dissertation is submitted to the Tamil Nadu Dr.M.G.R. Medical University for the examinations to be held in May 2022 in partial fulfilment of the requirements for the award of M.S. Branch IV (Otorhinolaryngology). I have not submitted this dissertation work previously for the award of any degree or diploma from any other University.

Date: 23.12.2021

Place: Madurai Dr. E.SELVAPRIYA

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Date: 23.12.2021

Place: Madurai. Dr. E.SELVAPRIYA

## LIST OF ABBREVIATIONS USED

| | |
|---|---|
| ABR | Auditory Brainstem Response |
| ACT | Acceptance and commitment Therapy |
| CBT | Cognitive Behavioural Therapy |
| CT | Computed Tomography |
| CNS | Central Nervous System |
| dB | Decibels |
| DCN | Dorsal Cochlear Nucleus |
| EAC | External Auditory Canal |
| ENT | Ear Nose Throat |
| GABA | Gamma Amino Butyric Acid |
| HRCT | High Resolution Computed Tomography |
| Hz | Hertz |
| IHC | Inner Hair Cells |
| IT | Intratympanic |
| kHz | Kilo Hertz |
| MD | Meniere's Disease |
| Mm | Millimetre |
| MRA | Magnetic Resonance Angiography |
| MRI | Magnetic Resonance Imaging |

| | |
|---|---|
| MRV | Magnetic Resonance Venography |
| NIHL | Noise Induced Hearing Loss |
| OAEs | Oto Acoustic Emissions |
| OHC | Outer Hair Cells |
| PTA | Pure Tone Audiogram |
| SD | Standard Deviation |
| SNHL | SensoriNeural Hearing Loss |
| SOAE | Spontaneous Oto Acoustic Emission |
| TMJ | TemporoMandibular Joint |
| THI | Tinnitus Handicap Inventory |
| TM | Tympanic Membrane |
| TRT | Tinnitus Retraining Therapy |

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## ABSTRACT

**Background:**

Tinnitus is a common and distressing symptom which is characterized by the perceived sensation of sound without any corresponding external stimulus. Seen in any age and sex, Subjective idiopathic tinnitus – defined as the experience of noises in the ears or head without both aberrant aetiology and external stimuli.

Intratympanic injection of steroid (Dexamethasone) is used for the treatment of subjective idiopathic tinnitus by introducing the drug through the tympanic membrane, resulting in reduced systemic toxicity and a higher perilymph steroid level.

**Aim of the study:**

The aim of the study was to evaluate the efficacy of Intratympanic steroid ( Dexamethasone) in the treatment of subjective idiopathic tinnitus.

**Materials and Methods:**

A total of 30 patients with subjective idiopathic tinnitus were subjected to Intratympanic injection of Dexamethasone, once per week for 3 weeks. Improvement in the symptom of tinnitus was assessed by means of subjective evaluation with the help of Tinnitus Handicap Inventory (THI) before and after 1 week , 1 month and 3 months of last injection.

**Results:**

The results of this study shows that, there was a significant improvement of tinnitus which is assessed by means of Tinnitus Handicap Inventory after the 3 doses of injections and found it in the subsequent evaluations - 1 month and 3 months after the treatment.

**Conclusion:**

Hence, the intratympanic injection of dexamethasone was found to be effective in the treatment of subjective idiopathic tinnitus. Intratympanic treatment was demonstrated to improve tinnitus scores in the study population.

**Keywords:**

Intratympanic Dexamethasone, Subjective Idiopathic tinnitus, Tinnitus Handicap Inventory.

# INTRODUCTION

The term “tinnitus” derived from the Latin word tinnire which means “to ring”. It is a phantom auditory perception, as sound perceived for more than 5 minutes at a time, arises due to aberrant spontaneous activity, from an altered state of excitation or inhibition within auditory system. Seen in any age and sex, it is one of the most common distressing problems causing various somatic and psychological disorders, affecting the ability of a person to lead a normal life. Tinnitus is not a well-defined disease, but a symptom of many pathologies, because of otologic or non-otologic causes. It may or may not be associated with hearing loss. Non otologic causes of subjective tinnitus may include neurologic, metabolic or psychogenic disorders.

Objective tinnitus usually due to vascular abnormalities of carotid artery or jugular venous systems, arteriovenous malformations, glomus tumours, palatal or tympanic myoclonus or Eustachian tube dysfunction.

**Subjective idiopathic tinnitus** – defined as the experience of noises in the ears or head without both aberrant etiology and external stimuli. Non otologic causes of subjective tinnitus may include neurologic, metabolic or psychogenic disorders. Several different options have been described for tinnitus treatment like tinnitus retraining, tinnitus masking, biofeed back therapy, various drug treatments.

## AIM OF THE STUDY

To examine the efficacy of Intratympanic steroid (Dexamethasone) injection in the treatment of subjective idiopathic tinnitus.

## REVIEW OF LITERATURE

### HISTORICAL REVIEW:

Shim et al. stated that the intratympanic steroid injection was effective in treating newly developed tinnitus. The efficacy of treatment decreases with prolonged tinnitus.

Sakata et al. treated 1,214 patients with intratympanic dexamethasone injections. They found that 77% of the patients with symptoms reduced immediately and 68% after six months.

In 2000, Shulman and Goldstein treated 10 patients with intratympanic steroid injection, reported that symptoms improved in about 70% of their subjects.

In 2002, Cesarani et al. following intratympanic steroids for tinnitus treatment reported complete resolution in 34% of patients, 40% shows significant improvement, and no change in 26%.Topak et al. following a three week intratympanic steroid injection, evaluated patients after 2 weeks and did not detect a significant difference between the steroid and placebo.

An et al. also reported that the mean Tinnitus Handicap inventory scores were significantly reduced at 3 months after Intratympanic dexamethasone injection.

The advantage of prednisolone over dexamethasone was explained by Parnes et al. who showed that methylprednisolone had a higher concentration and longer duration in the perilymph after intratympanic injection.

However, Hamid reported that dexamethasone is more effective, because it is absorbed faster than other steroids; he used a higher concentration of dexamethasone. He reported that steroids improve the stria vascularis ion transport system. Disturbance in the ion transport system or ion homeostasis affects the inner ear function, which includes hair cell function, endolymph and perilymph composition, nerve impulse conductance. Corticosteroids restore the ion homeostasis in the inner ear.

Mohamed and Hafize reported a significant difference between the study group and control group (tinnitus patients without intratympanic Dexamethasone injection) and concluded that tinnitus matching test could be used as a subjective evaluation of tinnitus and outcome measure of therapy. THI scores also improved after therapy. Most of the patients changed from severe, catastrophic handicap to slight, mild and moderate handicap.

## ANATOMY OF EAR:

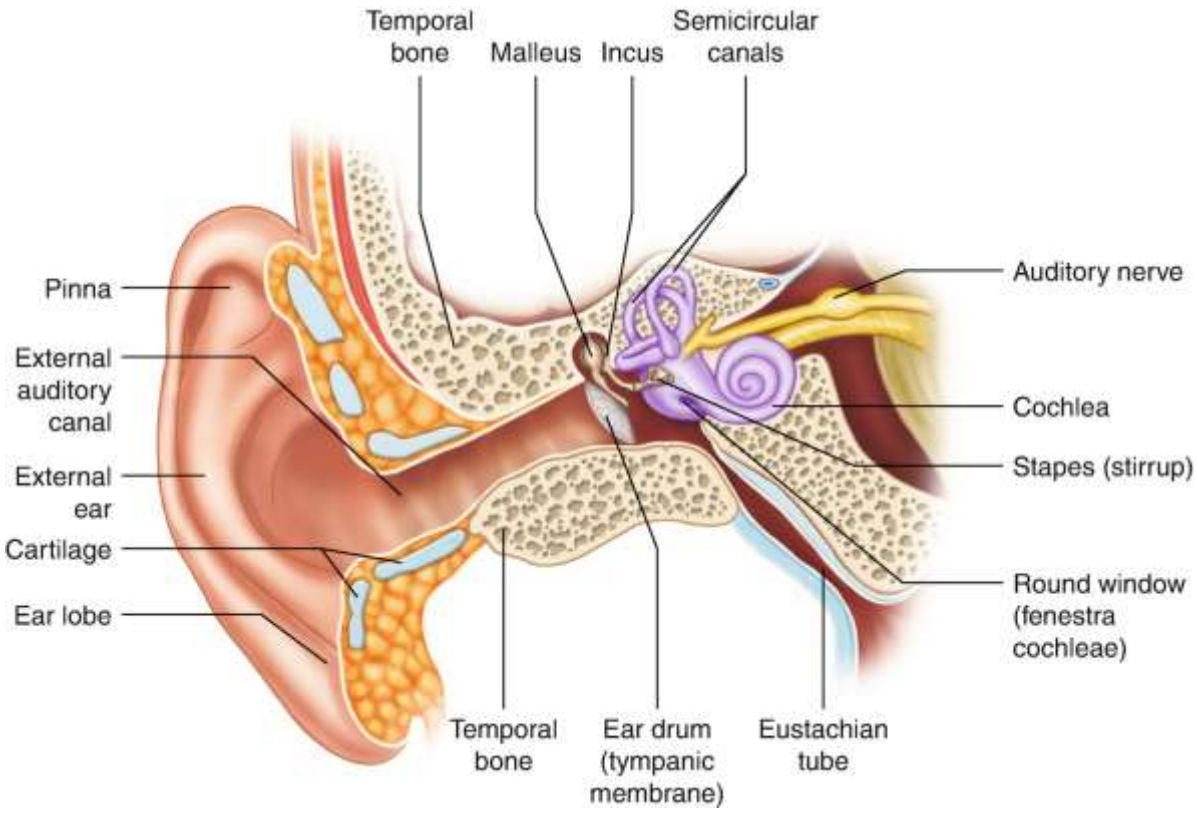



**FIGURE 1: ANATOMY OF EAR**

The Ear has Three parts which includes:

External Ear,

Middle Ear and

Inner ear.

## EXTERNAL EAR:

It Consists of Pinna and External auditory canal (EAC) and Tympanic membrane (TM).

Pinna captures sounds which travels along this canal to reach the middle ear. The length of S-shaped adult human EAC is approximately 24 mm and 5 to 10 mm in diameter contains fibrocartilaginous and bony parts. It also forms a resonance channel that amplifies the sound pressure up to 15-25 dB at frequencies between 2 and 5 kHz.

## TYMPANIC MEMBRANE:

The normal tympanic membrane (TM) is a cone shaped, shiny, pearly white in colour, thin (0.1 mm) membrane that separates the external ear from middle ear. And divided into 2 parts by anterior and posterior malleolar fold. Part above the fold is PARS FLACCIDA and part below the fold is PARS TENSA. Three layers of tympanic membrane: Outer epithelial, middle fibrous and the inner mucosal layers which is set obliquely, with the posterosuperior part more lateral than the anterosuperior part. A bright cone of light is seen on the antero-inferior quadrant of a normal TM.

**MIDDLE EAR:**

Bounded externally by the Tympanic membrane and internally by the inner ear. The middle ear together with the eustachian tube, Aditus, antrum and the mastoid air cells constitute the middle ear cleft which is lined by mucous membrane and filled with air. This air-filled cavity contains the ossicular chain

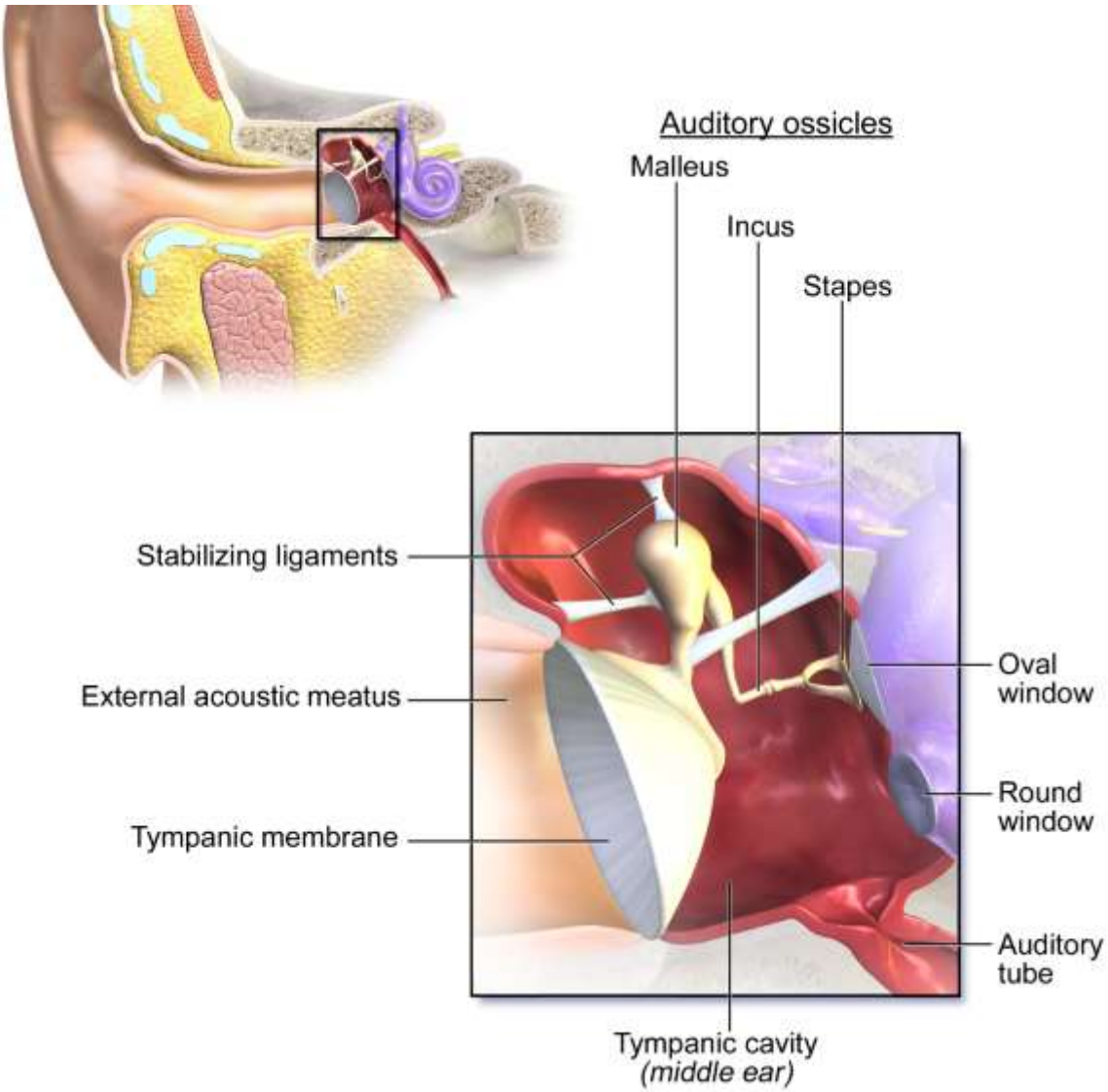



**FIGURE 2:ANATOMY OF MIDDLE EAR**

**EUSTACHIAN TUBE:**

Also called as Pharyngotympanic or the Auditory tube, connects middle ear to nasopharynx which is 36 mm long, contains a bony part and a fibrocartilaginous part. Under normal circumstances, this tube is closed. It can open to let a small amount of air through to prevent damage caused by pressure differences between the middle ear and the atmosphere.

**OSSICULAR CHAIN:**

Three interconnected bones: Malleus, Incus and Stapes. It is attached to one side of the tympanic membrane by the malleus, inserted into the oval window of the inner ear by the footplate of stapes. When a sound wave hits the TM, it transmits through the ossicular chain into the oval window of the inner ear.

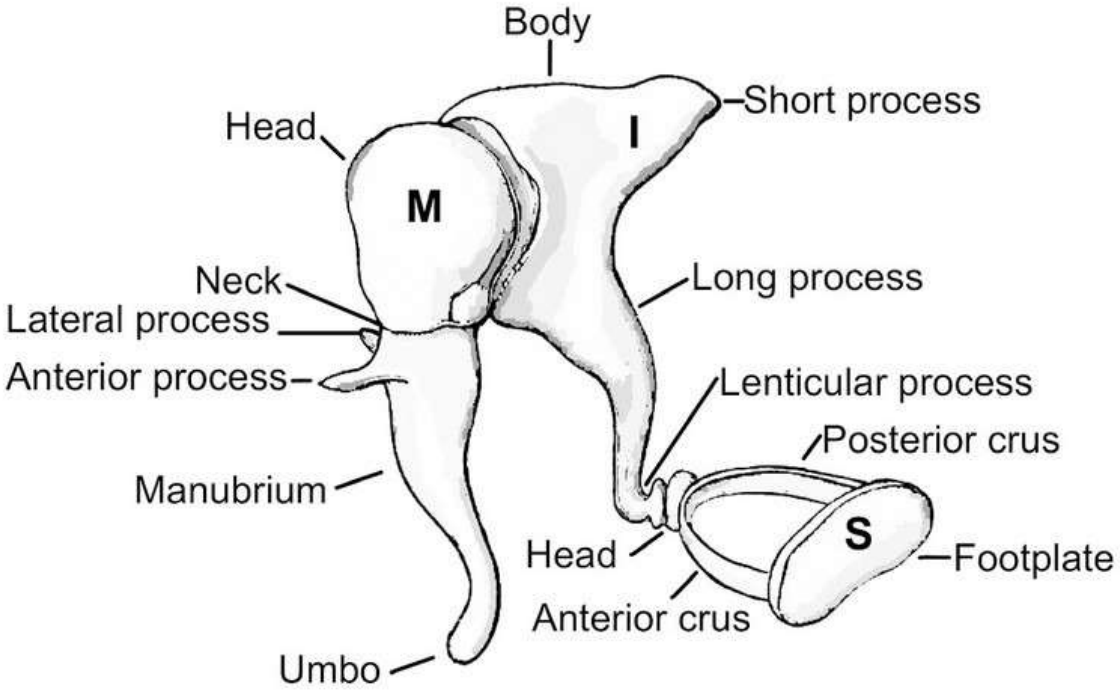



**FIGURE 3 : ANATOMY OF OSSICULAR CHAIN**

**MUSCLES:**

Tensor tympani and Stapedius. Tensor tympani attaches to the neck of malleus and tenses the tympanic membrane, while stapedius attaches to the neck of the stapes and helps to dampen very loud sounds, thus preventing noise trauma to the inner ear.

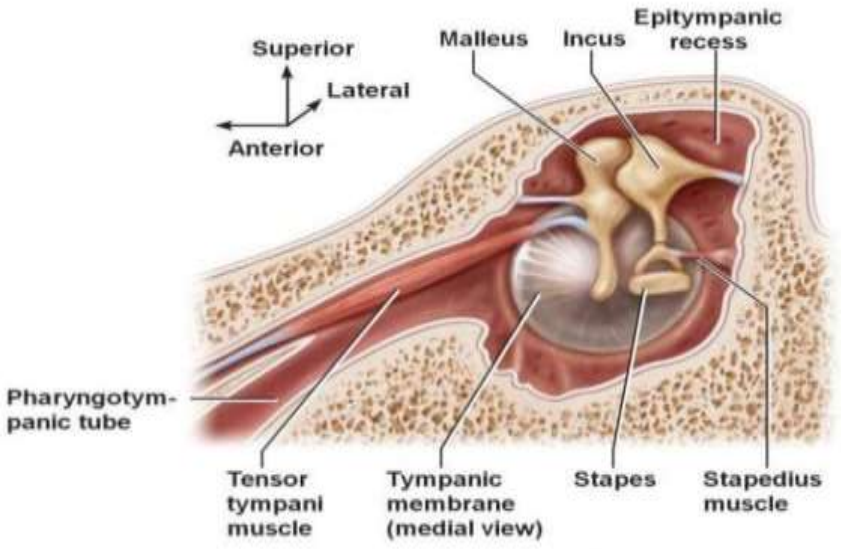



**FIGURE 4: MUSCLES OF MIDDLE EAR**

**INNER EAR**:

An important organ of hearing and balance. Composed of two separately functioning systems, the cochlea and the vestibular apparatus. Consists of a bony and membranous labyrinth.

The **bony labyrinth** consists of 3 parts:

Vestibule,

Semicircular canals and

Cochlea

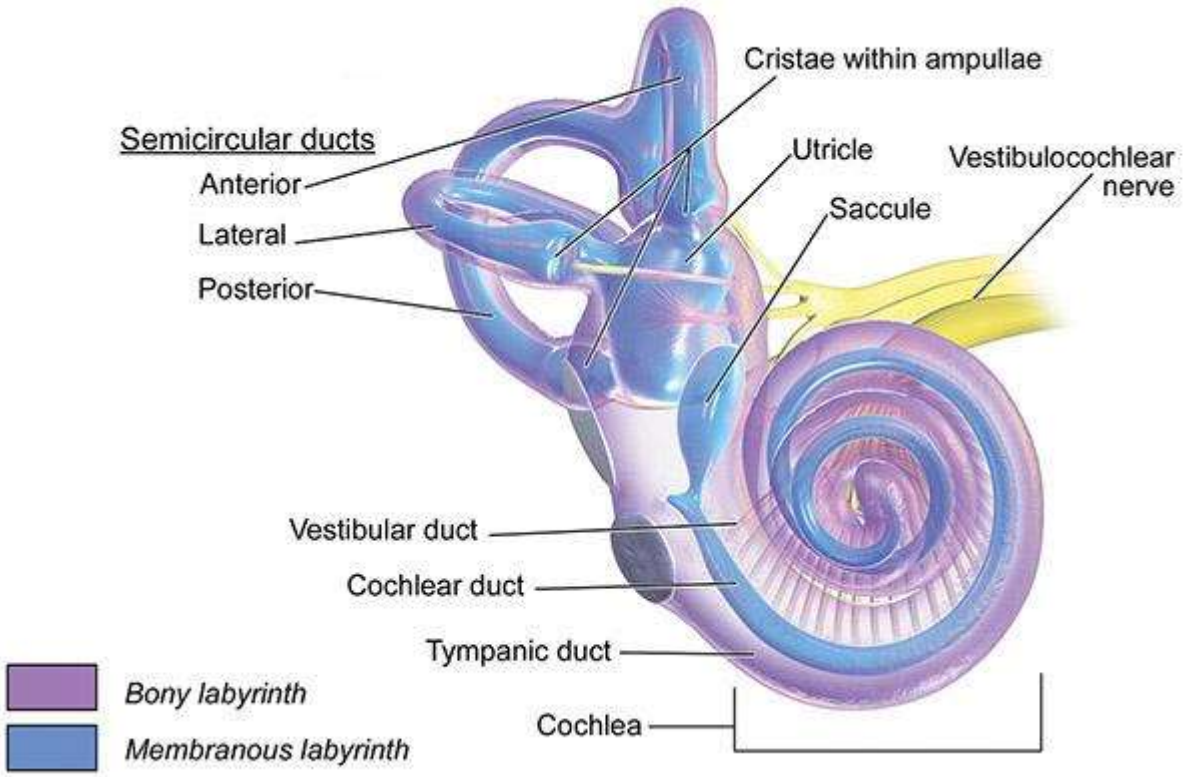



**FIGURE 5 : ANATOMY OF INNER EAR**

**Vestibule** is the central chamber of the labyrinth.

**Semicircular canals** are three in number: lateral, posterior and superior. Lies in planes right angles to each other.

**The bony cochlea** is an approximately 30 mm long coiled tube making 2.5 to 2.75 turns around a central pyramid called the modiolus. The base protrudes into middle ear as promontory of medial wall. The cochlea lying within the petrous bone, is divided by membranes into three chambers: the Scala vestibuli, Scala tympani and Scala media.

**The membranous labyrinth** consists of the cochlear duct, the utricle, the saccule, the three semicircular ducts, and the endolymphatic duct and sac. The cochlear duct is also known as Scala media, which is a blind coiled tube, triangular on cross-section. The union of two ducts one each from the utricle and saccule forms the endolymphatic duct passes through the vestibular aqueduct. Its terminal part dilates to form the endolymphatic sac lies between the two layers of dura on the posterior surface of petrous bone.

**The spiral organ of Corti** rests on the basilar membrane extends along the longitudinal length of the cochlea parallel to the basilar membrane. It contains sensory cells arranged in one row of approximately 3,500 inner hair cells (IHC) and 3 parallel rows of approximately12,000 outer hair cells (OHC), a tunnel of

corti containing cortilymph, between the inner and outer rods of Corti. And has supporting cells of Claudius, Dieter and Hensen.

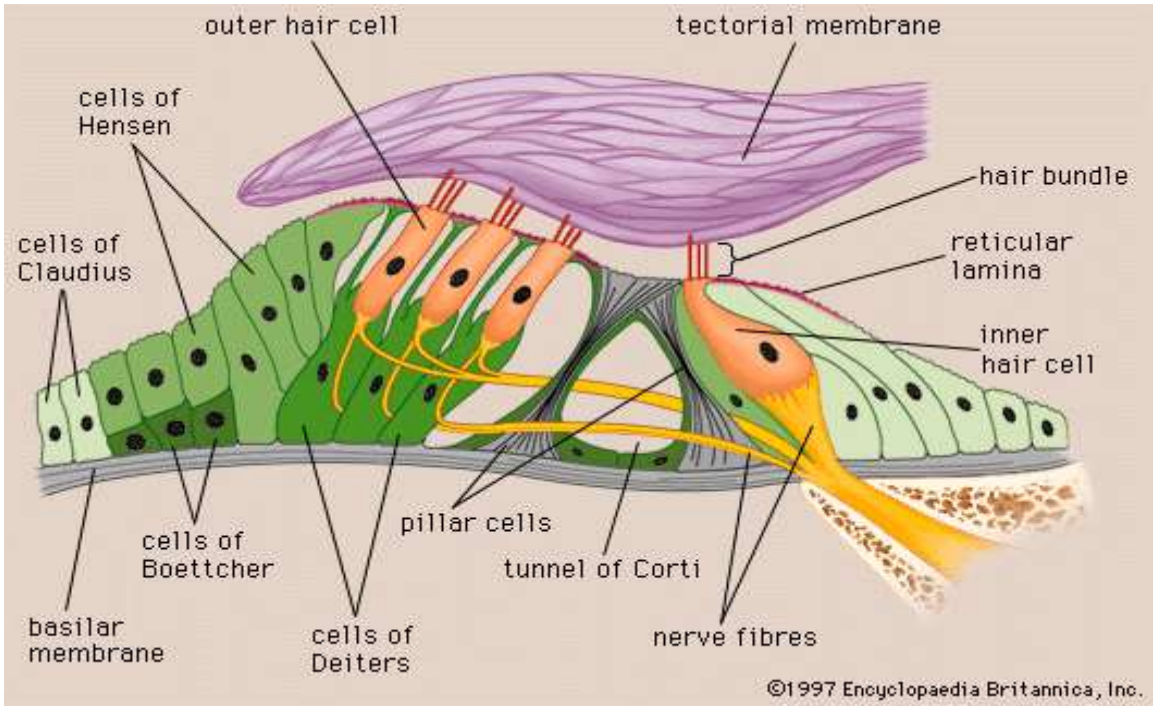



**FIGURE 6 : ORGAN OF CORTI**

Both the inner (IHC) and outer hair cells (OHC) lie on the basilar membrane and covered by the tectorial membrane. The apical portions of the hair cells are anchored in the cuticular plate, with the stereocilia (100-150 per cell) protruding through the cuticular plate.

**Hair cells**: The inner hair cells (IHC) are flask shaped, supported by interphalangeal cells and have stereocilia.

The outer hair cells (OHC) are columnar and supported by Deiters cells inferiorly and supported laterally by Hensen's cells. The tectorial membrane is anchored at the limbus medially and laterally attached to the Hensen's cells by a fibrous net.

They are sensory receptors in the auditory system. The sound energy creates a travelling wave delivered to the oval window, displaces the basilar membrane and the tectorial membrane vertically. They slide horizontally when stimulated, resulting in a shearing action between the tectorial plate and the cuticular plate. Displacement of stereocilia initiates an electrical event in the hair cells and are innervated by afferent and efferent neurons in a complex but in a orderly manner. The vibration is transferred by the hair cells to the auditory nerve; in this process, outer hair cells serve as amplifiers. The Inner hair cells transform the vibration into the fluids of the cochlea, where it is then transformed into an electrical signal.

The cochlear nerve contains a total of 30,000 afferent nerve fibers. The cell bodies of these fibers found in the Spiral ganglion located in the Rosenthal's canal of the bony modiolus.

The auditory branch of the vestibulocochlear nerve leaves the cochlea through the internal auditory meatus and enters the brainstem, with the afferent and efferent fibres running in an opposite direction.

The sole blood vessel that supplies the inner ear is the labyrinthine artery. This artery divides into the cochlear artery and the anterior vestibular artery, the cochlear artery further divides into the main cochlear artery and the vestibulocochlear artery.

**Central auditory nervous system:**

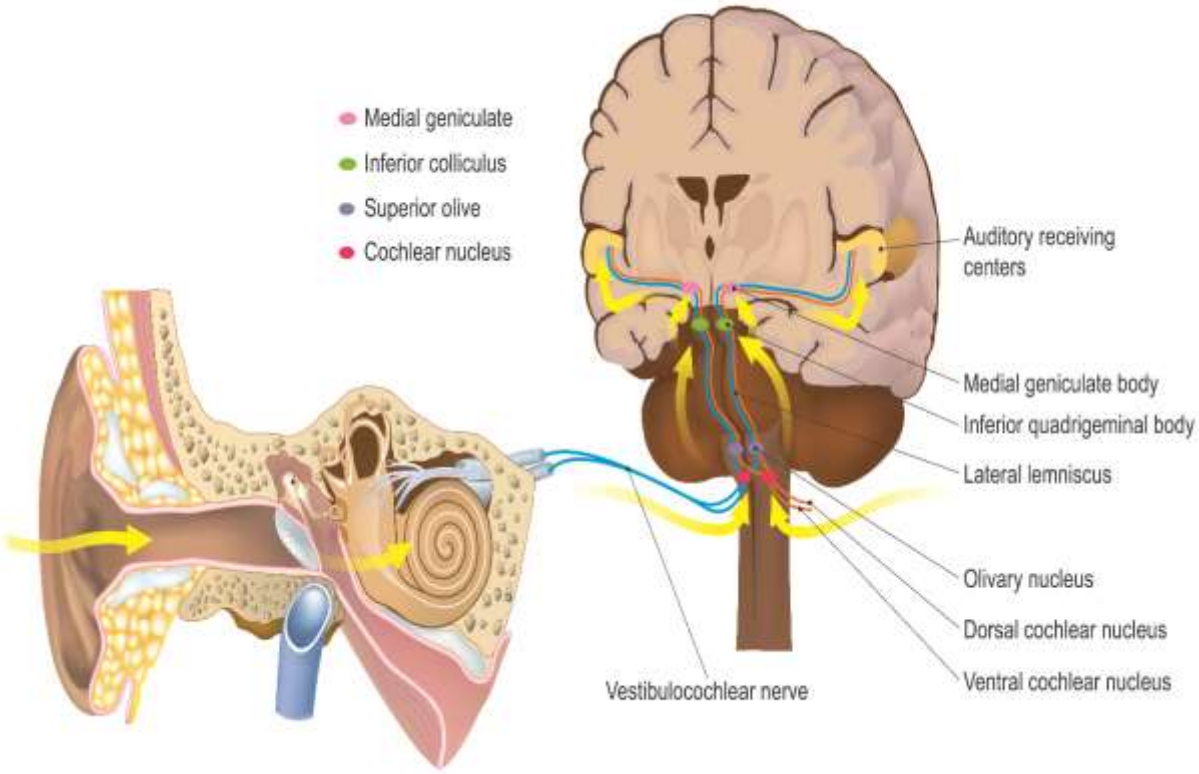



**FIGURE 7 : THE CENTRAL AUDITORY SYSTEM**

The ability to recognize and localize sound is dependent on processes and neural networks which are distributed throughout both hemispheres. The recognition of the environmental sounds are involved in bilateral regions in the temporal and prefrontal areas, and the sound localization seems to activate a large cortical area, especially the temporal, parietal and prefrontal cortices.

# TINNITUS

## EPIDEMIOLOGY

Tinnitus has likely troubled humanity and has been a common complaint in the global population. Patients experiencing tinnitus often describe it as a buzzing, ringing, hissing, whistling, chirping, squealing, clicking or roaring sensation inside the ears or head. Tinnitus can vary in loudness from a quiet background noise, to one that appears to mask external sounds.

Acute tinnitus can last for days to weeks, while chronic tinnitus is more persistent, lasting for greater than six months. From different studies with different age groups done in different countries, the approximate prevalence of tinnitus in adults is concluded to range between 10.1 and 14.5 percent. It is a positive function of age: 38 percent of patients < 40 years and 62 percent of patients > 40 years present with tinnitus.

The prevalence rate of tinnitus in children with normal hearing thresholds has been reported to range from 6 to 36 percent. Children do not see tinnitus as a problem and have learned to live with it without being concerned about it or are scared to talk about it; they lack the vocabulary to discuss tinnitus.

Population statistics suggest a slightly higher prevalence of tinnitus among females (50-60 percent), especially below the age of 40 years, than males (40-50 percent). This relative difference appears to be greater in those reporting severe tinnitus and its resultant annoyance.

In case of severe tinnitus, the prevalence was 6.5% in females and 4.7% in males. In another study, similar rates of moderate or severe tinnitus (5.6% versus 7.4%) was reported among individuals randomly sampled with smaller gender differences.

Men have traditionally been more exposed to noise in the form of firecrackers, firearms (military), and noisy work environments. But females are affected more with psychological factors.

An increasing prevalence of tinnitus in unskilled, in comparison with the professional classes or higher socioeconomic groups, has been noted.

## CLASSIFICATION OF TINNITUS

Tinnitus can be classified as either vibratory or non-vibratory, as objective or subjective, further categorized by the hypothesized site of lesion.

**Vibratory tinnitus** is caused by transmission of vibrations to the cochlea from adjacent tissues or organs. It follows a rhythm and occurs because the patient is able to hear one's own muscle contractions, eustachian tube movements, blood flow within the vascular system.

**Non-vibratory tinnitus**, is non-rhythmic and random, attributed to a more central neural activity and is produced by biochemical changes in the nerve mechanism of hearing.

**Subjective tinnitus** can be vibratory or rhythmic in nature, but at a sound level experienced only by the patient. Most causes of tinnitus are subjective in nature. Yet, it is usually non-vibratory or irregular in pattern. Bilateral subjective tinnitus requires hearing assessment and may associated with presbycusis, noise-induced hearing loss, endolymphatic hydrops, and vascular labyrinthine lesion. Unilateral high-frequency hearing loss combined with poor speech discrimination suggests the presence of a tumour, usually a vestibular schwannoma or a meningioma.

**Objective tinnitus,** which can be recorded objectively by a microphone or can be heard by another listener. The sound can come from, for example, the carotid artery, auditory tube or temporomandibular joint. The perceived localization of the patient's tinnitus can be of diagnostic significance, especially because unilateral tinnitus may be a symptom of an underlying tumor like vestibular schwannoma. Pulsatile tinnitus is often associated with disorders of the vascular system.

Tinnitus prevalence rises with increasing hearing loss.

Symptoms may originate in several different places in the auditory system. It may have various causes:

i) Conductive (e.g., otosclerosis and infections in the middle ear),

ii)Sensori neural (most common) due to cochlear or retrocochlear impairment

iii) Mixed hearing loss (a combination of conductive and sensorineural hearing loss), and

iv) Central hearing loss due to damage to the central pathways.

The loudness of tinnitus sounds fluctuates in the majority of individuals. The volume can be altered by exposure to loud sounds, nerve tension, increased blood pressure, lack of energy and some chemical substances, such as drugs, alcohol, caffeine and tobacco. However, relaxation and good physical fitness can make the tinnitus less disturbing.

## MECHANISMS AND PROCESSES RELATED TO TINNITUS

The sensation of hearing occurs after sound waves travel through the 3 parts of the ear like outer, middle, and inner ear to the cochlea, the auditory nerve, to the brainstem, and into auditory cortex of the temporal lobe.

The exact cause of tinnitus is not known and it is a symptom. Knowledge of the pathological mechanisms and cellular events that surround tinnitus are insufficient to enable identification even in cases where the cause seems to be evident. When no sounds are present, random activity occurs in the neurons of the auditory pathway. The brain prioritizes all the sound information it receives, so the nervous system generally filters out this random neural activity thus no sound is perceived.

In a normal auditory system, external noise usually overrides any internal or random noise. However, tinnitus occurs when the internal noise is given higher priority and thus dominates or accompanies external noise. Regardless of the underlying pathology or pathophysiology behind tinnitus, the internal signal is processed by the central auditory nervous system and perceived as sound in the auditory cortex.

## PERIPHERAL AUDITORY SYSTEM

### Spontaneous Otoacoustic Emissions

Spontaneous otoacoustic emissions (SOAEs), are first discovered by Kemp, which are small acoustic signals presumed to be generated by the Electromotile activity of the outer hair cells of the cochlea and propagated into the external auditory canal and perceived as tinnitus.

Peripheral lesions or dysfunctions of different etiologies causes instability, making the inaudible SOAEs as audible. This occurs due to reduction in the afferent input and compensatory disinhibition within the proximal auditory pathway, altering spontaneous activity and causing plastic transformation of the brain, resulting in tinnitus.

According to **Jastreboff and Hazell** (2004), this imbalance of neural activity that can cause tinnitus-related changes which affects type I and type II fibres of the auditory nerve. These atypical SOAEs are much more prevalent in the higher frequency range and can appear at sound pressure levels up to 55 dB SPL in the ear canal. Tinnitus due to SOAEs is mild and more common in subjects with normal hearing and in those with only middle ear disorders. As hearing loss progresses, this SOAEs decreases and hence these otoacoustic emissions are not likely to cause tinnitus when a hearing loss of 35 dB or more is present.

**Edge Theory (Contrast Theory)**

This theory proposes that tinnitus is induced by increased spontaneous activity in the edge area. This area represents a transition from Outer Hair Cells in the organ of Corti with relatively normal morphology and function on the apical (i.e., low-frequency) side of a lesion to OHCs toward the basal side that are missing and poor functionality. Edge theory can be explained by discordant theory.

**Discordant Theory**

Inner Hair Cells are the receptor cells for sound transduction, and almost all afferent fibers in the auditory nerve (95%) innervate IHCs, while OHCs work as mechanical amplifiers, enhancing weak sounds by providing up to 50 dB, which can be evaluated by measuring otoacoustic emissions.

Intense noise and ototoxic agents initially damage Outer Hair Cells in the basal turn of the cochlea, and subsequently, if continued or repeated, affect Inner Hair Cells as IHCs are more resistant to such damage.

According to discordant theory, tinnitus is induced by the discordant dysfunction of damaged OHCs (seen in almost all situations) and IHCs of the organ of Corti which results in the disinhibition of neurons in the dorsal cochlear nuclei (DCNs).

Spontaneous activity is increased when neurons in the DCN receive excitation from IHCs but not from the damaged OHCs, and this is perceived as tinnitus.

Normally there is a small gap between the top of the cilia of the IHCs and the bottom of the tectorial membrane, but in the area in which OHCs are affected but IHCs are intact, the tectorial membrane might touch the IHC cilia, thus causing the IHCs to depolarize. The OHCs normally recover with a few days, but this can be delayed for up to a few months. Therefore, it is hypothesized that tinnitus represents a consequence of a central gain adaptation mechanism when the auditory system is confronted with a hearing loss.

Similarly, noise-induced tinnitus is caused by discordant damage between OHCs and IHCs. Two types of noise induced tinnitus have been identified: tonal and complex. Tonal tinnitus results from discordant dysfunction of OHCs and IHCs manifesting in a single area, whereas complex tinnitus results from multiple areas of discordance. When patients clearly have the central type of tinnitus, such as after transection of the auditory nerve, the OHC concept is not applicable and alternative mechanisms need to be considered.

## CENTRAL AUDITORY SYSTEM

### The Dorsal Cochlear Nucleus

The DCN has the tendency to become hyperactive due to the slow plastic readjustments in DCN caused due to outer hair cell damage on exposure to tinnitus-inducing agents such as intense sound and cisplatin. Hence this nucleus has been described as a possible site for generation of delayed onset tinnitus-related signals. Inner hair cell damage prevents hyperactivity in the Dorsal cochlear Nucleus. A reduction in auditory nerve input can lead to disinhibition of the DCN and an increase in spontaneous activity in the central auditory system, which manifests as tinnitus. This mechanism could explain the temporary ringing sensation that can follow exposure to loud sound.

### Auditory Plasticity

Damage to the cochlea enhances the neural activity in the central auditory pathway and this aberrant pathway results in auditory plasticity. Here, tinnitus might be considered to be the auditory system analog to phantom limb sensations in amputees. Tinnitus might be generated in the temporal lobe in the auditory association cortex and in the inferior colliculus. The plastic changes involving the development of aberrant connections between the auditory and sensory-motor systems in the brain which explains the ability of some individuals to modulate tinnitus by performing the voluntary somatosensory or motor actions.

**Crosstalk Theory**

According to this, when auditory nerve fibres are intact and some other cranial nerves are damaged, artificial synapses (crosstalk) can develop between the individual auditory nerve fibres, resulting in phase locking of the spontaneous activity of groups of the auditory neurons.

In the absence of external sounds, this creates a neural pattern that resembles patterns evoked by the actual sounds. These cranial nerves are sensitive to compression at the root entry zone, where these nerves are covered by myelin. Nerve compression causes crosstalk between nerve fibres, and the breakdown of the myelin insulation of the nerve fibres establishes ephaptic coupling between them.

This notion is applied to the vestibulocochlear nerve, which is covered by central myelin for most of its length and hence is vulnerable to compression from the blood vessels or tumors impinging upon the nerve (e.g., vestibular schwannoma). Such compression and consequent ephaptic coupling might lead to tinnitus if synchronization of stochastic firing in the cochlear nerve is perceived as a sound.

**Tonotopic Reorganization**

In the auditory cortex, all frequencies are tonotopically mapped to show coding of different frequencies at the level of basilar membrane. All the sequence of changes in relative levels of excitatory and inhibitory inputs to the primary auditory cortical neurons, following damage to the cochlea, induces the tonotopic reorganization of the receptor surface in the primary auditory cortex.

This in turn raises threshold sensitivity and broadens the frequency selectivity. The normal function of the neurons in the cortex is modified, which implies that these neurons do not respond to their own frequencies or to the frequencies generated at the non-affected area.

## SOMATOSENSORY SYSTEM

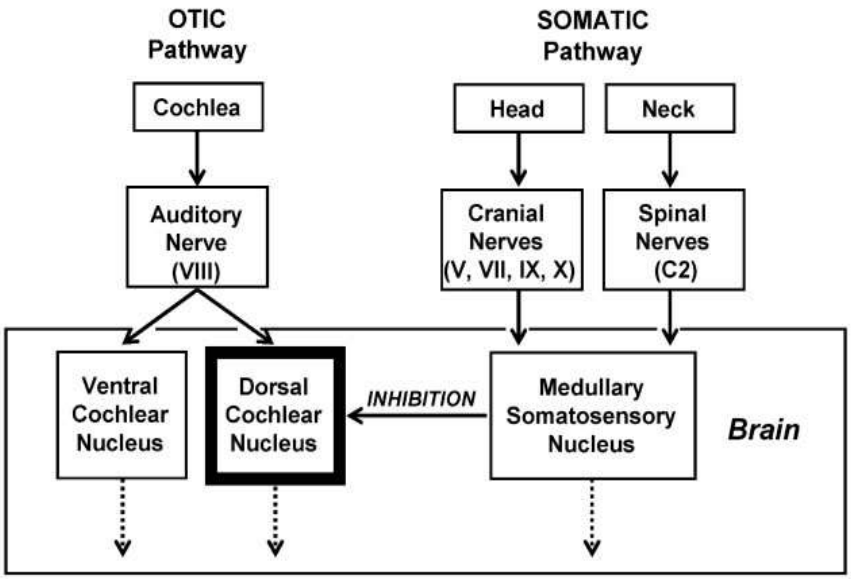



**FIGURE 8 : THE SOMATOSENSORY SYSTEM**

The activity of the Dorsal Cochlear Nucleus is also influenced by the stimulation of nonauditory structures and the somatosensory system is the only nonauditory sensory system that appears to be related to tinnitus (e.g., in temporomandibular-joint syndrome and whiplash).

Somatic tinnitus can develop from the activation of latent oto-somatic interaction. Somatic (craniocervical) tinnitus, like otic tinnitus, is caused by the disinhibition of the ipsilateral Dorsal cochlear Nucleus, which is mediated by nerve fibres whose cell bodies lies in the ipsilateral medullary somatosensory nuclei. These neurons receives inputs from the nearby spinal trigeminal tract, the fasciculus cuneatus, and facial, vagal, and glossopharyngeal nerve fibres innervating the middle and external ears.

Pain signals from the cochlea carried by the cochlear C fibres could also be interpreted by the Central nervous system as tinnitus. It is further hypothesized that somatic tinnitus is due to the central crosstalk within the brain, because certain head and neck nerves enters the brain near regions known to be involved in hearing.

## NEUROPHYSIOLOGICAL MODEL OF TINNITUS

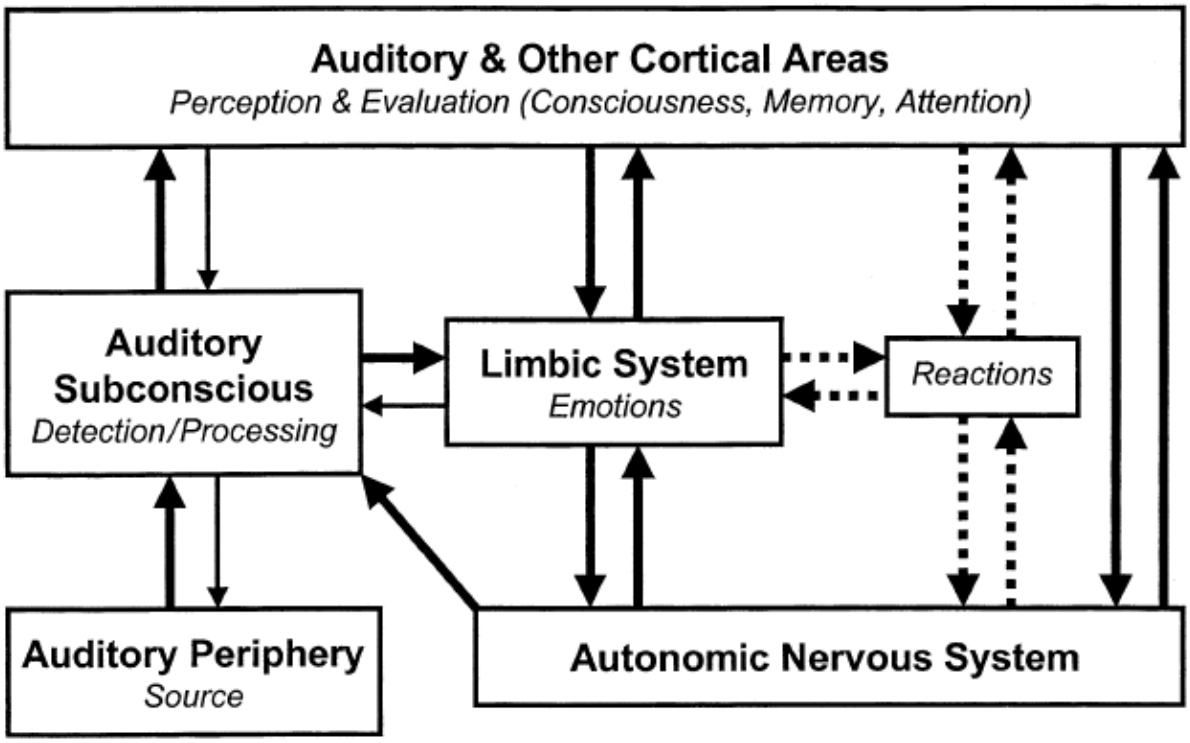



**FIGURE 9 : THE NEUROPHYSIOLOGICAL MODEL**

First described by P. J. Jastreboff (1990), this theory involves 3 systems such as auditory perception, emotional and reactive systems and a combination of peripheral and central dysfunction. It does not identify the mechanisms responsible for generating the neural activity that results in tinnitus, but rather the results of this neural activity.

This model presents three main postulates regarding clinically significant tinnitus.

**BIOCHEMICAL MODEL**:

A biochemical model of peripheral tinnitus has been recently proposed. This model is partly based on the clinical observation that adult humans with distressing tinnitus have experiences of agitation, stress and anxiety, and partly based on cochlear neurochemistry.

Endogenous dynorphins (associated with stress) potentiates the excitatory function of glutamate within the cochlea, mimicking the action of sodium salicylate in increasing the spontaneous neural activity. It also displays a highly neurotoxic effect, observed in various pathological conditions, including acoustic trauma.

Excessive noise exposure leads to excessive glutamate release and excitotoxic intracellular Ca ++ overload, which could be a basis for tinnitus. This neural hyperactivity and therefore tinnitus, is related to central processing, mediated by other neurotransmitters of the auditory pathways, such as glycine, acetylcholine and GABA. Disrupted or modified serotonin (5-HT) function might cause a reduction in auditory filtering abilities and in tinnitus habituation. This stresses on the need for investigation of the effect of selective serotonin re-uptake inhibitors upon tinnitus. The lateral olivocochlear efferents may play a role in modulating the sensitivity of the afferent receptors in the cochlea.

Dopamine, as a neurotransmitter, acts as a permanent gain control at the site of the action potential initiation.

The intracellular concentration of calcium is responsible for a number of functions of the OHC and IHC cells, including the fast and slow motility of the OHCs and transmitter release in the IHCs. Increases in the amount of calcium in the hair cells could lead to an amplified signalling to the brain.

If this signalling occurs in a dysfunctional cochlea, it would lead to increased neurotransmitter release from inner hair cells and increased activity in the auditory nerve fibers in the form of cascade signalling (burst firings).

The synchronization of activity in the small nerve fibers could cause the perception of the tinnitus sound.

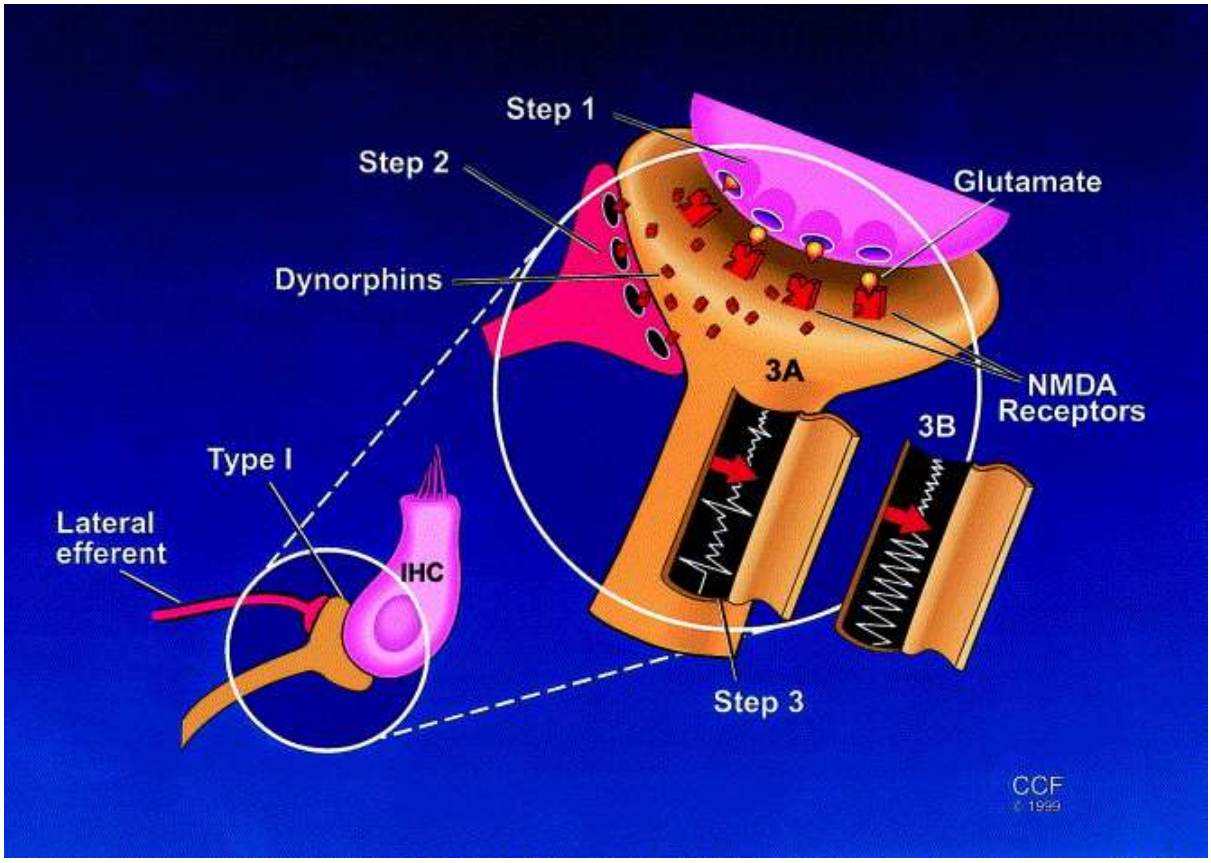



**FIGURE 10 : THE BIOCHEMICAL MODEL**

## ETIOLOGY OF TINNITUS

Tinnitus does not represent a disease itself but instead is a symptom of a variety of underlying diseases.

**Otologic causes**: Noise-induced hearing loss, presbycusis, otosclerosis, otitis, impacted cerumen, sudden deafness, Meniere's disease, and other causes of hearing loss.

**Neurologic causes:** Head injury, whiplash, Multiple sclerosis, vestibular schwannoma (acoustic neuroma), and other cerebellopontine- angle tumours.

**Infectious causes** : Otitis media and sequelae of Lyme disease, meningitis, syphilis, and other infectious or inflammatory diseases.

**Drug induced**: Salicylates, nonsteroidal anti-inflammatory drugs, aminoglycoside antibiotics, loop diuretics, and chemotherapy agents (e.g., platins and vincristine). Temporomandibular-joint dysfunction and other dental disorders can also cause tinnitus.

**Causes of conductive hearing loss that may lead to tinnitus** include impaction of cerumen, otitis media, otomycosis, eustachian tube dysfunction, otosclerosis, and other ossicular diseases. Cerumen impaction may produce a low pitched, intermittent non-vibratory sound that is resolved once the cerumen was removed. Some dental disorder and temporomandibular-joint (TMJ) dysfunction have also been documented to be associated with tinnitus.

**Sensorineural tinnitus**, a form of peripheral tinnitus, would be the most common form of tinnitus as sensorineural hearing loss (SNHL) is the most common form of hearing loss. The idea that tinnitus originates in the inner ear has been supported by the idea that patients often localize tinnitus to one ear versus the other.

Sensorineural tinnitus has four subclasses.

1) Tinnitus related to the OHC also referred to as motor tinnitus,

2) Tinnitus related to the IHC also referred to a transduction tinnitus,

3) Tinnitus related to the auditory nerve also referred to as transformation tinnitus, and

4) Tinnitus related to the "Extrasensory" sources referred to as objective tinnitus.

Schaette and Kempter (2009) correlated tinnitus to increased spontaneous firing rate of central auditory neurons. The increase in neural response gain counteract the decreased auditory activity due to hearing loss and in effect restores the mean firing rate, but also leads to hyperactivity in the central auditory neurons. The hyperactivity patterns are strongest at frequencies close to perceived tinnitus pitch which also correlates to patient's audiogram in terms of hearing loss. Thus they suggest tinnitus can be caused by cochlear damage.

**Central tinnitus** relates to tinnitus originating anywhere in the central auditory pathways. Retrocochlear lesions or neural lesions that are located beyond the cochlea on the vestibulocochlear nerve or in one of the auditory areas of the brain, causing degeneration of the nerves, making them unable to convey neurochemical information, resulting in retrocochlear hearing loss. The types of retrocochlear hearing loss are divided into two groups: central and vestibular nerve diseases.

1. Auditory diseases (central) are disorders of auditory perception resulting from diseases of the central auditory pathways or auditory associated cortical areas, such as cortical deafness. Above the level of the pons, bilateral lesions are usually required to produce auditory dysfunction.
2. Vestibulocochlear nerve diseases are the diseases that can damage the vestibular and/or cochlea nerves. A rather common cause of retrocochlear hearing loss is the growth of a benign tumour (eg: vestibular schwannoma) that presses on the auditory nerve. Finally, patients with acoustic neuromas often report unilateral tinnitus.

**Presbycusis,** a common type of sensorineural hearing loss which is caused by normal, biological changes in the cochlea and degeneration of the spiral ganglion. The first and defining sign of presbycusis is loss of threshold sensitivity in the high frequency region of the hearing spectrum. Changes in this region are usually first detected at 60 years if no other factors are involved. Over time, the threshold increasingly progresses to lower frequency areas. Pure presbycusis is more often a combination of several factors, including genetics, life style and noise exposure factors.

Perry and Gantz (2000) cautions that other medical factors like vascular disease, diabetes, hypertension, autoimmune disorders, and neural disorders also increases with age and could contribute to tinnitus. Likewise, medical conditions may lead to the increase use of medications which could also be linked to tinnitus.

**Noise-induced hearing loss (NIHL),** Intense noise exposure sometimes leads to a temporary threshold shift in the cochlea but can also lead to permanent hearing loss that may be accompanied by other auditory disorders, such as tinnitus and hyperacusis. NIHL is generally bilateral and symmetric and affects higher frequencies first, followed by lower frequencies. This cochlear tinnitus is most commonly described as a high pitched tonal or hiss-like sound.

**Meniérè's disease (MD)** is a disorder of the inner ear, characterized by repeated spontaneous and episodic vertigo over time, fluctuating hearing loss and low-pitched roaring tinnitus. The cause is still unknown, but it is believed that endolymphatic hydrops is a pathophysiological condition closely associated with the disease causing the symptoms or just its side effects. Several studies have suggested that the symptoms originate from a change in the volume/pressure relationship, due to fluid over production or decreased resorption of the endolymph, leading to hair cell damage.

**Idiopathic Sudden Sensorineural Hearing Loss (ISSNHL)** is described as a symptom rather than a disease and can be accompanied by tinnitus and/or vertigo. It may be a result of local autoimmune processes that affects the cochlea, or a consequence of infection or a vascular disorder. There are different types of treatments, including steroids, that are based on the immune theory.

**Hearing loss induced by ototoxic drugs** can be temporary or permanent. These can lead to high-frequency hearing loss and are associated with a loss of OHC in the cochlea and also often reported as a high-frequency ringing sensation. Alcohol, caffeine, and tobacco have also been linked to an increase in tinnitus symptoms.

**Cochlear Otosclerosis** is an uncommon disease and usually presents in young patients. This disease often occurs between 30 and 50 years of age and is known to worsen during pregnancy. The stiffening of osseous bone causes a mixed hearing loss (sensorineural and conductive hearing loss) that is occasionally combined with tinnitus.

**Vascular abnormalities:** Tinnitus in these cases is usually soft, rushing, pulsatile synchronous with the heartbeat, which shows an increase in rate with exercise. It is rare, experienced by lesser than 10% of patients who have tinnitus. An audible bruit is heard in only a small proportion of these patients. Causes include arteriovenous shunts (intracranial or preauricular, congenital or acquired, with or without decreased hearing), arterial bruits, and venous hums.

**Congenital arteriovenous shunts**: Interconnections between the occipital artery and the transverse sinus, the internal carotid and the vertebral vessels, or the middle meningeal artery and the greater superficial petrosal artery, have all been reported. Diagnosis is by exclusion and by magnetic resonance imaging (MRI) or angiography (MRA).

**Acquired arteriovenous shunts:** The most common acquired tinnitus secondary to arteriovenous shunting is caused by glomus tympanicum or glomus jugulare tumor. The tinnitus in this condition is described as a soft or blowing sound, and often associated with diminished hearing. The diagnosis is made by computed tomography (CT) of the middle ear, demonstrating erosion of the caroticojugular spine, or by appearance of the lesion lying within the middle ear space.

**Arterial bruits** are transmitted from the carotid or prevertebral vessels and are detected in aberrant carotid positions. Other types of arterial bruits are those coming from vascular loops within the internal auditory meatus pressing on the eighth cranial nerve. Diagnosis generally is made by air-contrast CT, MRI, or magnetic resonance angiography (MRA). A carotid bruit with a pulsatile tinnitus merits treatment, including endarterectomy.

**Venous Hums:** Heard in patients who have hypertension or abnormal high placement of the jugular bulb. The tinnitus of a dehiscent jugular bulb is often unilateral and often gives a soft, low-pitched venous hum, which can be altered by head position, activity, or pressure over the jugular vein. The diagnosis is based on CT evaluation. MRA or magnetic resonance venography (MRV) usually can define the abnormality. Treatment is fraught with great risk, however, there is no defined specific management.

**Patulous Eustachian tube:** occurs frequently in patients who have had radiation therapy involving the nasopharynx. Patients, in this case, often complain of ocean roar sounding tinnitus, generally synchronous with nasal respiration. The symptoms are usually absent on arising but rapidly occurs thereafter and diminished by lying down with the head in a dependent position This is a useful diagnostic clue. Patients also may complain of some reverberation and have abnormal awareness of their own voice (i.e., autophony).

Fluttering motion of the tympanic membrane associated with respiration is diagnostic. Tympanography may be helpful for diagnosis by having the patient breathe sharply or quickly through his or her nose while being tested. Treatment includes caustics to the nasopharynx, mucosal irritants, saturated solution of potassium iodide, and Teflon or Gelfoam injections around the torus tubarius.

**Palatomyoclonus:** It is caused by the mucous membranes of the eustachian tube snapping together in response to the movement of the palatal musculature. Patients frequently give histories of postoccipital headache and temporomandibular joint pain. Tinnitus here is described as an irregular clicking sound. The sound is rapid (40 to 200 beats per minute) and occurs intermittently. Diagnosis is made by observation or hearing the clicking using a Toynbee tube.

The tympanogram is useful by recording movement synchronous with the contractions. Electromyographic studies of the palatal musculature may be necessary to confirm the diagnosis. The differential diagnosis for palatal myoclonus includes idiopathic stapedial muscle spasm, multiple sclerosis, cerebral vascular disorders, and tumour. Treatment relies on medication, including muscle relaxants (e.g., clonazepam, diazepam). Botulinum toxin injection into the active muscle helps in severe cases. Only rarely is surgery indicated.

. **Idiopathic stapedial muscle spasm**: This rough rumbling or crackling noise of idiopathic stapedial muscle spasm may be accentuated by external sound (e.g., water faucets, musical tones, certain voices). Diagnosis can be made on examination if tympanic membrane movement is synchronous with the noise. Treatment has consisted of division of the stapedius muscle and the tensor tympani muscle. Occasionally, muscle relaxants (e.g., diazepam) may relieve this symptom. Patients should be advised to avoid stimulants such as nicotine, coffee, and decongestants.

## EFFECTS OF TINNITUS

About 6-17 percent of the general population experience tinnitus lasting for a period of at least 5 minutes. About 15 percent have spontaneous tinnitus with no obvious temporary cause. In at least 3-8 percent, interferes with sleep or causes moderate or severe annoyance and in 0.5-2.5 percent, tinnitus severely affects the ability to lead a normal life.

The prevalence of 'clinical tinnitus', those seeking medical advice is about 7.2 percent. Reactions to tinnitus can vary in severity from mild irritation to suicidal ideation.

Han et al. estimated that approximately 20 to 30 percent of patients with tinnitus experience its most common psychosocial distresses like anxiety, depression, irritability, annoyance, anger, insomnia, difficulty in concentration and speech, with these causing disruption of everyday activities that they seek medical intervention.

Due to the above, children may have learning and writing difficulties and decrease in lingual capacity affecting their academic development. Psychological attributes such as depression and concentration can be attributed more to tinnitus severity than audiometric thresholds across all populations. Patients suffering from depression are vulnerable to developing distress at a relatively low degree of tinnitus, while stress-tolerant individuals can endure higher degrees of tinnitus before seeking help.

## TREATMENT:

The course of treatment for each tinnitus patient will be varied depending on factors like patient characteristics, cause, symptoms and severity.

Tinnitus treatment is even further complicated by the idea that the exact causes of tinnitus have not been identified and it being a subjective symptom, it is difficult to do epidemiologic and efficacy studies for treatment. Despite the existence of a large variety of treatment models, there is no permanent cure for tinnitus.

**Medical/Surgical Treatment:** Treating an underlying medical or otologic cause of tinnitus can provide relief.

De Ridder et al., (2007) summarize the most relevant pathologies that may be treatable with surgical intervention like: vestibular schwannomas and other cerebellopontine angle lesions, arachnoid cysts, Ménière's disease, otosclerosis, brain tumours along the auditory pathways, Chiari malformations, microvascular compressions of the vestibulocochlear nerve, benign intracranial hypertension, arteria carotid stenosis, glomus tumours, vascular lesions of the petrous bone and skull base, ateriovenous malformations, aneurysms, and vascular loops inside the internal auditory canal.

Inconsistent results have been obtained regarding stapedectomy surgery for patients whose tinnitus is associated with otosclerosis. Some patients report tinnitus symptoms improve greatly while others notice no difference. If an acoustic neuroma or glomus tumor is found, surgical removal may help alleviate tinnitus. In severe cases of Meniere's disease surgery can help improve both tinnitus and vertigo symptoms.

Dental treatment for temporomandibular joint (TMJ) problems may help patient who have tinnitus associated with their TMJ. Impacted cerumen causing tinnitus is usually resolved once the cerumen is removed with the help of aural syringing. Infectious causes of tinnitus may all be treated with specific pharmacotherapy.

**Diet/Lifestyle:**

Certain drugs, such as aspirin and NSAIDs, are known to exacerbate or cause tinnitus in some patients. Patients should limit or avoid these medications if they can be linked to tinnitus onset.

Stimulants such as caffeine contained in coffee, tea, and pop may increase the tinnitus symptoms and patients with tinnitus are often told to avoid ingesting large amounts of caffeine. Avoiding smoking and nicotine in all its forms also helps improve tinnitus. The distress caused due to tinnitus may also be diminished with the help of adequate relaxation and physical fitness.

**Pharmacology :**

**Lidocaine :** A local anaesthetic also used in cardiac arrhythmias, lidocaine was found to suppress tinnitus following nasal administration. It is not an option for long-term treatment due to its short living effects arrhythmogenic effects.

**Benzodiazepines** The different benzodiazepines differ in their half-life and are used as sedatives, anxiolytics, anticonvulsants and muscle relaxants.

**Alprazolam**, a short-acting drug, reduced tinnitus loudness.

**Diazepam**, a longer acting benzodiazepine, with a very similar mechanism of action as alprazolam, had no effect on tinnitus loudness.

**Clonazepam**, a long acting benzodiazepine taken at a dose of 0.5–1 mg/day for 60– 180 days for vestibular or cochleovestibular disorders revealed improvement of tinnitus in 32% in a large retrospective study. It was found to significantly reduce tinnitus loudness and annoyance relative to the control group in a prospective study. Clinical use of benzodiazepine is limited by their side effects such as high risk of drug dependency and personality changes.

**Antidepressants :** Beside their antidepressant properties, tricyclics have been shown to be highly efficient for the treatment of chronic pain, which has etiological similarities between tinnitus and neuropathic pain.

**Amitriptylin**e inhibits the reuptake of serotonin and noradrenaline almost equally. 6 weeks with amitriptyline at 100 mg per day significantly reduced tinnitus complaints and tinnitus loudness.

The results suggest that tinnitus patients with depression and anxiety may benefit from antidepressants, mostly due to the beneficial effect of antidepressants on comorbid depression and anxiety than its direct effect on tinnitus severity.

**Anticonvulsants :** Carbamazepine binds to voltage-gated sodium channels and stabilizes the sodium inactivation state, thereby reducing neural firing. It has shown mixed results for tinnitus.

**Gabapentin**, which acts on voltage-gated calcium channels, is used for the treatment of neuropathic pain and migraine. Several large, randomized clinical trails failed to demonstrate benefit of gabapentin over placebo in treating tinnitus while few other pilot studies showed positive results

**Antiglutamatergic agents** : Excitotoxicity mediated by NMDA receptors has been proposed as a mechanism for cochlear tinnitus. Cochlear application of a selective NMDA antagonist in the first four days following noise exposure also reduced the probability of developing noise-induced tinnitus.

**AM-101**, an NMDA antagonist failed to significantly reduce minimal masking levels, but its higher dose showed substantial and statistically significant reduction in tinnitus loudness, sleep impact and tinnitus impairment in patients suffering from acute tinnitus with established cochlear origin.

**Dopaminergic/Antidopaminergic agents:** Both dopaminergic and antidopaminergic drugs have been proposed for treating tinnitus. Dopamine is an inhibitory transmitter in the cochlea; antidopaminergic drugs improve thalamic filtering of sensory signals. Furthermore, dopaminergic pathways in limbic and prefrontal areas may be involved in mediating emotional aspects of tinnitus.

**Sulpiride**, an antipsychotic drug that selectively blocks dopamine D2 receptors significantly reduced subjective ratings of tinnitus and tinnitus visual analogue scores in one double-blind, placebo-controlled study.

## OTHER COMPOUNDS

**Intratympanic gentamycin** has been used successfully to treat Meniere's Diseases and the tinnitus associated with it.

**Ginkgo biloba** contains bioactive flavonoids and terpenes with vasoactive and antioxidant properties and has been proposed for the treatment of a wide range of disorders including tinnitus.

**Betahistine** is an H3 antagonist blood receptor and an H1 agonist receptor which improves inner ear microcirculation, promoting and facilitating central vestibular compensation. It has been tried in the treatment of Meniere's Disease in combination with other drugs to have shown significance in reducing tinnitus.

**Antihistamines** were found to increase inner ear vascularity and metabolism and hence, reduce tinnitus. But studies have shown this decrease in tinnitus not to be its direct effect and may be due to its sedative property causing a reduction in the effect of tinnitus. Cinnarizine, dimenhydrinate and betahistine have been tried for tinnitus.

**Vitamins and Minerals** have been used ascomplementary and alternative pharmacologics in tinnitus and these include vitamins A, B1, B3, B6, B9, B12, C, E, Magnesium, Calcium, Potassium, Manganese, Selenium and Zinc.A statistically significant increased incidence of vitamin B12 deficiency was seen in patients with chronic tinnitus and noise-induced hearing loss with reported improved symptoms after replacement therapy.

**Zinc**, an essential trace metal plays an important role in numerous biological signalling functions and is widely distributed in the auditory pathway. While positive results have been reported in some patients with hypozincemia, zinc therapy did not result in tinnitus improvement in patients with normal zinc levels.

**Antioxidants** may be helpful as a supplemental treatment for patients undergoing therapy with ototoxic drugs, though it may not be a therapy for tinnitus as such.

**Botulinum toxin** has been investigated in a small pilot study, where it has demonstrated superiority over placebo. Botulinum toxin has also been shown to be highly effective in the rare cases where tinnitus is caused by palatal tremor.

## Tinnitus Retraining Therapy (TRT)

Tinnitus Retraining Therapy is a multi-step therapy developed by P. J. Jastreboff and his colleagues and is based on the neurophysiologic model of tinnitus. The hypothesis associated with TRT is that two different processes of non habituation create tinnitus perception and tinnitus annoyance. Hence, the goal of TRT is to habituate the patient to the emotional reactions surrounding tinnitus, such as fear and anxiety, and by doing so to habituate to tinnitus. The goal here is not to mask the tinnitus, but to help reduce the stress associated with tinnitus for a life comfortable even with tinnitus.

## Cognitive Behavior Therapy (CBT)

CBT is one of the more recent forms of psychotherapy and is characterized by a focus on how thoughts, behaviors and reactions affect each other. It is a treatment approach used to identify and modify behaviors, thoughts and cognitions that are disruptive for the individual. It does not focus on reducing the patient's perception of tinnitus in any form but aims to reduce the psychological distress associated with tinnitus by eliminating some of the negative attitudes regarding tinnitus.

A successful treatment should eliminate the disturbance caused by the tinnitus and aid patients in accepting and dealing with their tinnitus.

## Hearing Aids

Hearing aids has been a useful treatment for patients suffering from both tinnitus and hearing impairment. Hearing aids allow patients to become less aware or completely unaware of tinnitus by providing external auditory stimuli that can mask the tinnitus (Jastreboff & Hazell, 2004) and thus reducing its negative effects. Also Hearings aids removes the sensation that sounds are being "masked" by tinnitus by reinforcing sounds that patients have difficultly hearing due to their hearing loss and due to the masking of the sounds by the tinnitus.

## Sound Therapies

Sound therapies are believed to increase the background neuronal activity in the auditory system via sound stimulation, therefore masking tinnitus, distracting attention away from tinnitus, lowering stress associated with tinnitus, or lessening the tinnitus sound level. The idea of using music therapy with customized thresholds is the basis the Neuromonics.

## Neuromonics

The Neuromonics Tinnitus Treatment program combines acoustic stimulation with a structured program of counselling and support administered by clinician trained in tinnitus rehabilitation.

This form of treatment focuses on both the theorized cause of tinnitus, auditory deprivation resulting from hearing loss, and the role of the limbic system in creating a negative cycle of awareness and negative emotions surrounding the tinnitus. The Neuromonics Tinnitus Treatment is designed to stimulate the auditory system while positively engaging the limbic system.

**Biofeedback**

Biofeedback is based on the theory that tinnitus is induced or worsened by stress. As experiencing tinnitus can be stressful, tinnitus patients often find themselves in a cycle of stress, as tinnitus can cause stress and be exacerbated by stress. It is a relaxation process that can reduce the severity of tinnitus by reducing hyperarousal therefore facilitating habituation and helping patient foster a sense of physiologic control over tinnitus and thereby the distress associated with it.

**Alternative Treatments**

The list of alternative therapies for tinnitus includes acupuncture, music therapy, various herbal therapies, and relaxation. Relaxation therapy, yoga, visualization, and hypnosis can also be used to help reduce the stress associated with tinnitus.

## Acupuncture

Although acupuncture is commonly used to control pain, its use in tinnitus treatment has gained some recent popularity. Acupuncture employs needles to stimulate specific points on the human anatomy.

## Existing Therapy Plans

With an increasing number of treatment options for patients suffering from tinnitus, care givers have been searching for further rehabilitation alternatives that may have a greater impact on patients' sensitivity to their tinnitus; acceptance and commitment therapy (ACT) is one of these options. The goal of ACT is to increase the quality of life rather than to try and remove the annoyance or pain sensation.

In the Stepped Care approach the first step is that the patients see an audiologist with special training for counselling and diagnosing patients with tinnitus and hearing problems. Step number two is a multidisciplinary information meeting for those patients who did not feel they got enough help from the first step. As a final step, the patients who are not satisfied with step two are offered individual therapy that could include CBT, physiotherapy, counselling, physiotherapy etc

## INTRATYMPANIC THERAPY

Intratympanic treatment with various agents is an emerging treatment option currently. Although the mechanism of action of Intratympanic steroid injection in tinnitus is unclear, the mechanisms are considered to be the suppression of irritability or hypersensitivity of the sensory cell in the inner ear, the reduction of the inflammation caused by immune mediated/autoimmune dysfunction, and the direct effect on the inner ear neuroepithilium.

The therapeutic effect of intratympanic drugs is thought to occur by diffusion through round window, annular ligament of oval window capillaries or through the lymphatics of inner ear.

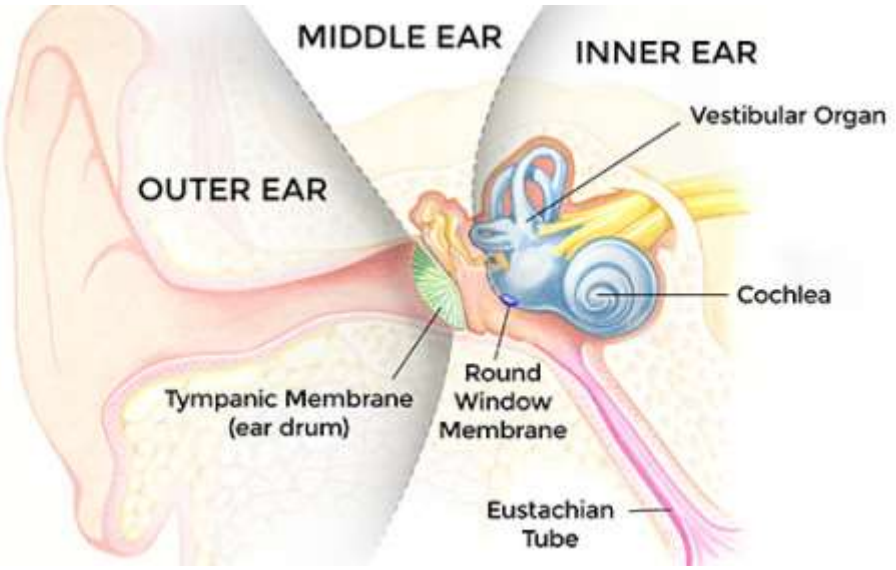



**FIGURE 11: RELATIONSHIP OF TYMPANIC MEMBRANE WITH ROUND WINDOW MEMBRANE**

**Advantages of intratympanic steroid injections :**

- High perilymph levels are attained as a result of providing a direct passage through the oval window membrane
- Inducing high concentrations in inner ear without systemic side effects,
- Also it reduces tinnitus perception significantly without any effect on hearing level.

For that we had selected Dexamethasone for intratympanic injection. It is relatively safe when compared to other drugs.

## PHARMACOLOGY OF DEXAMETHASONE

Dexamethasone is a synthetic fluorinated, long-acting, high-potency, anti-inflammatory and immunosuppressive corticosteroid with low mineralocorticoid activity. It has approximately 7 times the anti-inflammatory potency of prednisolone or prednisone and 30 times that of hydrocortisone: thus, 750 micrograms of dexamethasone is roughly equivalent in its glucocorticoid effects to 5 mg of prednisolone or 20 mg of hydrocortisone.

Each ampule of dexamethasone contains 3.3 mg dexamethasone (as sodium phosphate) which is equivalent to 4 mg dexamethasone phosphate or 4.3 mg dexamethasone sodium phosphate.

Dexamethasone is metabolized by the liver and excreted in the urine mainly. Dexamethasone has a half-life of approximately 3 hours.

Dexamethasone is more effective than other steroids, because it is absorbed faster than other steroids.

Dexamethasone and other glucocorticoids bind to cytoplasmic glucocorticoid receptors, and can modulate gene expression directly, or by an interaction with various transcription factors. This inhibits the synthesis or expression of numerous substances associated with inflammation, including cytokines, chemokines, arachidonic acid metabolites, and adhesion molecules. They may also upregulate the production of some anti-inflammatory mediators.

# MATERIALS AND METHODS

## STUDY DESIGN:

This study is a prospective observational study.

## STUDY AREA AND STUDY PERIOD:

The study is conducted in 30 patients, who were treated in the ENT Ward at Madurai Medical college and Hospital prospectively during the time period of 1 year from November 2020 to November 2021.

## INCLUSION CRITERIA:

1. Patients who can give informed consent
2. Patients with subjective idiopathic tinnitus
3. Patients between 15 to 50 years
4. Patients who are refractory to medical treatment

## EXCLUSION CRITERIA:

1. Patients having otitis media
2. Patients with Tympanic membrane perforation
3. Patients with pulsatile tinnitus
4. Patients with other systemic diseases who are contraindicated for steroid injection.

**METHODOLOGY:**

Clinical history to be taken for every patient and complete ear, nose and throat (ENT) examination with careful otoscopic, oto endoscopic, otomicroscopic examination.

Routine blood tests are required to rule out any systemic or metabolic disorders. Pure tone audiometry, can reveal the state of the hearing threshold levels and indicate the location of the probable cause.

Impedence audiometry to rule out middle ear pathologies.

Speech audiometry, auditory brainstem response (ABR) measurement that may be performed according to the demands of the condition.

Radiological investigations like HRCT Temporal bone was done to exclude the causes of pulsatile tinnitus.

Various questionnaires have been designed to grade the tinnitus to measure the degree of tinnitus severity as tinnitus is often subjectively perceived. **Tinnitus Handicap Inventory (THI)** is among the most validated and useful method to measure the impact of tinnitus on patients life.

After complete evaluation, the patient can be taken for procedure of intratympanic injection by endoscopic method under local anesthesia. The therapeutic efficacy was determined by the patients symptoms before and after intratympanic injection therapy.

For that THI is used which is to be performed before the therapy and at first week, first month and three months of treatment.

## TINNITUS HANDICAP INVENTORY(THI)

**Name:** **Date:**

| | | | |
|---|---|---|---|
| 1.Because of your tinnitus, is it difficult for you to concentrate? | Yes | Sometimes | No |
| 2.Does the loudness of your tinnitus make it difficult for you to hear people? | Yes | Sometimes | No |
| 3.Does your tinnitus make you angry? | Yes | Sometimes | No |
| 4.Does your tinnitus make you feel confused? | Yes | Sometimes | No |
| 5.Because of your tinnitus, do you feel desperate? | Yes | Sometimes | No |
| 6.Do you complain a great deal about your tinnitus? | Yes | Sometimes | No |
| 7.Because of your tinnitus, do you have trouble falling to sleep at night? | Yes | Sometimes | No |
| 8.Do you feel as though you cannot escape your tinnitus? | Yes | Sometimes | No |
| 9.Does your tinnitus interfere with your ability to enjoy your social activities (such as going out to dinner, to the movies)? | Yes | Sometimes | No |

| | | | |
|---|---|---|---|
| 10. Because of your tinnitus, do you feel frustrated? | Yes | Sometimes | No |
| 11.Because of your tinnitus, do you feel that you have a terrible diseases? | Yes | Sometimes | No |
| 12.Does your tinnitus make it difficult for you to enjoy life? | Yes | Sometimes | No |
| 13.Does your tinnitus interfere with your job or household responsibilities? | Yes | Sometimes | No |
| 14.Because of your tinnitus, do you find that you are often irritable? | Yes | Sometimes | No |
| 15.Because of your tinnitus, is it difficult for you to read? | Yes | Sometimes | No |
| 16.Does your tinnitus make you upset? | Yes | Sometimes | No |
| 17.Do you feel that your tinnitus problem has placed stress on your relationships with members of your family and friends? | Yes | Sometimes | No |
| 18.Do you find it difficult to focus your attention away from your tinnitus and on other things? | Yes | Sometimes | No |

| | | | |
|---|---|---|---|
| 19.Do you feel that you have no control over your tinnitus? | Yes | Sometimes | No |
| 20.Because of your tinnitus, do you often feel tired? | Yes | Sometimes | No |
| 21.Because of your tinnitus, do you feel depressed? | Yes | Sometimes | No |
| 22.Does your tinnitus make you feel anxious? | Yes | Sometimes | No |
| 23.Do you feel that you can no longer cope with your tinnitus? | Yes | Sometimes | No |
| 24.Does your tinnitus get worse when you are under stress? | Yes | Sometimes | No |
| 25.Does your tinnitus make you feel insecure? | Yes | Sometimes | No |

**4 Points for YES**

**2 Points for SOMETIMES**

**0 Points for NO**

**The sum of all response is the THI SCORE.**

## TINNITUS HANDICAP INVENTORY SEVERITY SCALE

| GRADE | SCORE | DESCRIPTION |
|---|---|---|
| 1 | 0 – 16 | Slight |
| 2 | 18 – 36 | Mild |
| 3 | 38 – 56 | Moderate |
| 4 | 58 – 76 | Severe |
| 5 | 78 – 100 | Catastrophic |

With the THI Score, patient's symptom has been graded as Grade 1 to Grade 5.

## OUTCOME VARIABLE:

Clinical improvement assessed by means of subjective evaluation.

**TECHIQUE OF INTRATYMPANIC DRUG ADMINISTRATION:**

Informed consent from the patient was obtained after explaining the nature of the treatment proposed and the study.

The patient was placed in supine position with the head turned about 45 degrees away from the surgeon. Topical anesthesia of the external canal and tympanic membrane was administered using 10% lignocaine spray. Complete clearance of which will be done using suction after 2 – 3 minutes.

Using a 2 ml syringe and a spinal needle of size 26 gauge, the dexamethasone solution of dose 4 mg/ml was administered under direct vision using an endoscope in the posteroinferior quadrant of the tympanic membrane.

About 0.5 ml of Dexa was injected in to the middle ear and the patient was asked to remain in the same position for about 20 minutes and is told to avoid any swallowing movements or yawning during this period to prevent drainage of medication through the Eustachian tube.

The patients were under follow up. And totally 3 injections were performed, 1 injection per week for 3 weeks.

After finishing the treatment, the patients answered a questionnaire about the status of their tinnitus.

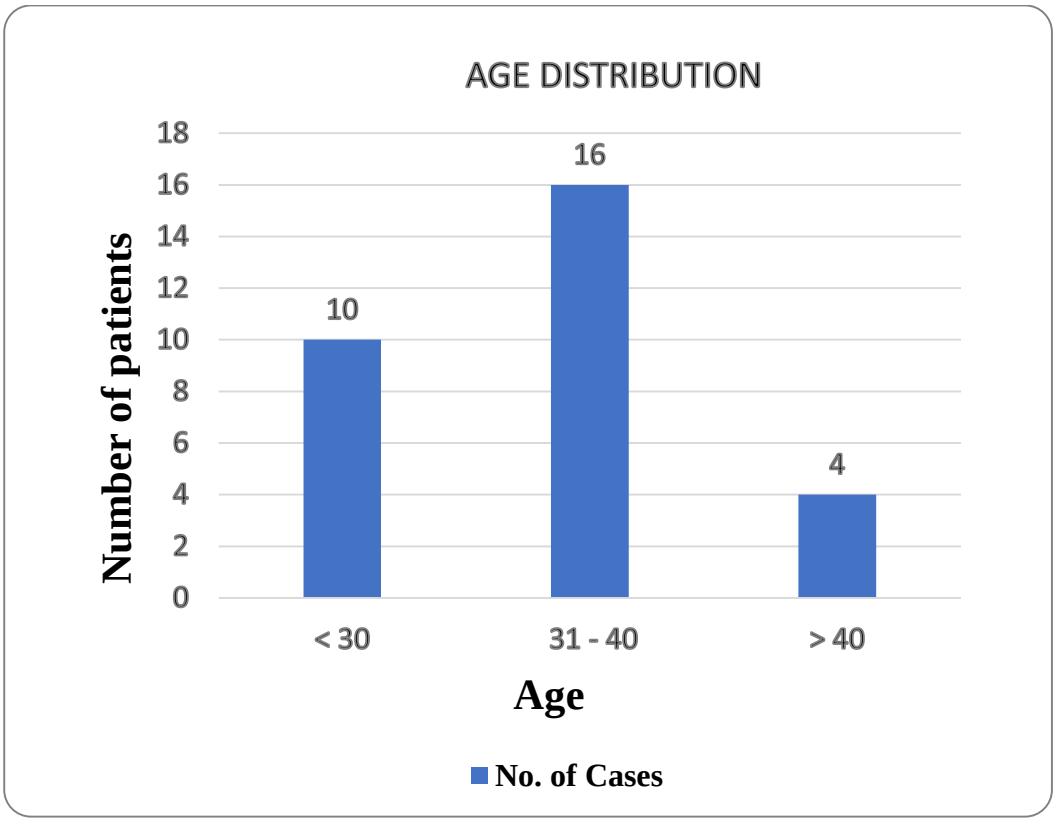
**FIGURE 12 : INTRATYMPANIC INJECTION PROCEDURE IN PROGRESS**

## OBSERVATION AND RESULTS

## AGE DISTRIBUTION:

| Age | No. of Cases | % |
|---|---|---|
| < 30 | 10 | 33.33 |
| 31 – 40 | 16 | 53.33 |
| > 40 | 4 | 13.33 |
| Total | 30 | 100.00 |
| Mean | 34.7 | |
| SD | 6.035 | |

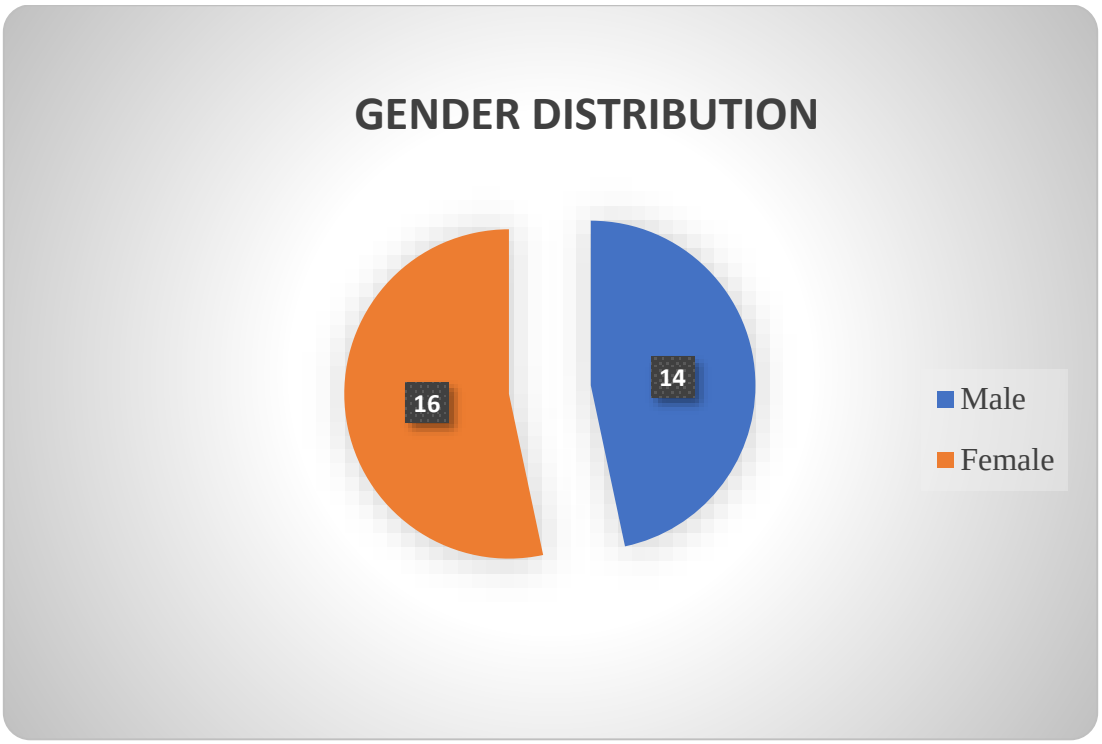
Majority of the population lies in the age group of 31 – 40 (53.33%).



## GENDER DISTRIBUTION:

| Gender | No. of Cases | % |
|---|---|---|
| Male | 14 | 46.67 |
| Female | 16 | 53.33 |
| Total | 30 | 100.00 |

In our study there is slight female preponderance ( 53.33%).

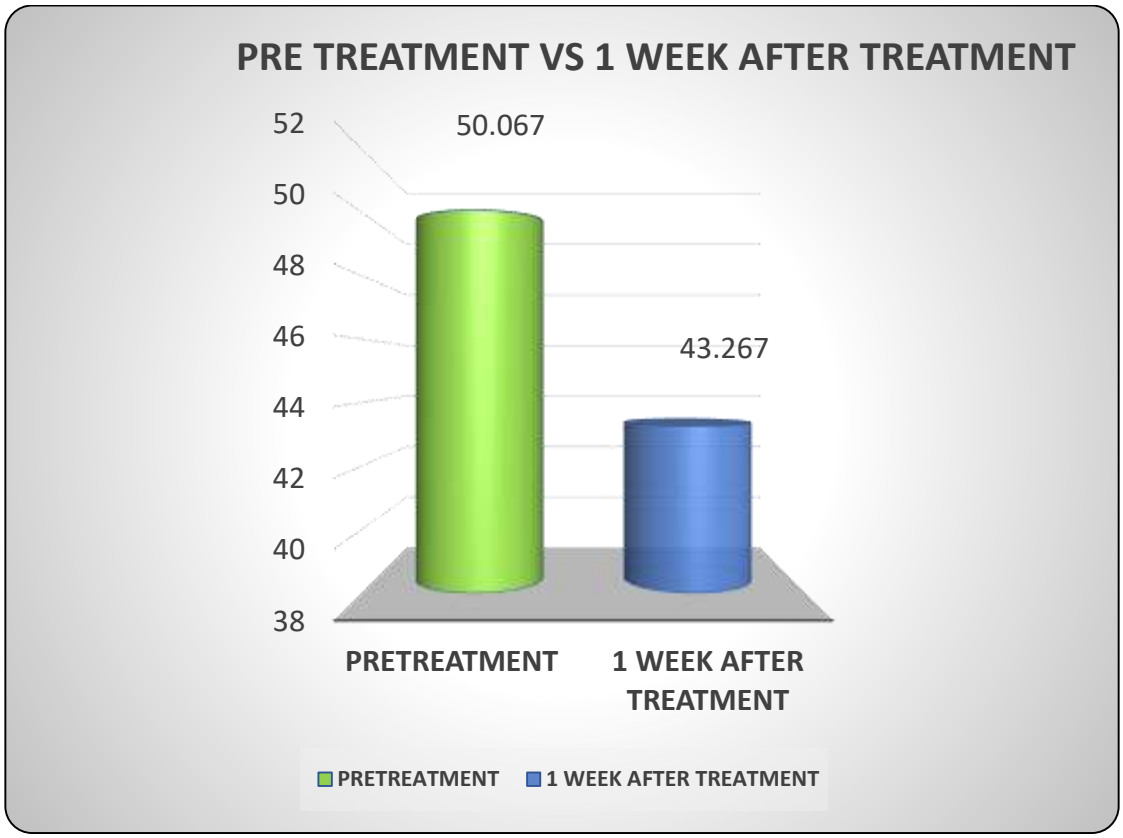


**INVESTIGATION:**

**COMPARISON OF THI SCORES - PRETREATMENT AND 1 WEEK AFTER TREATMENT :**

| PRETREATMENT VS 1 WEEK AFTER TREATMENT | | |
|---|---|---|
| THI SCORE | MEAN | SD |
| PRETREATMENT | 50.067 | 16.942 |
| 1 WEEK AFTER TREATMENT | 43.267 | 14.971 |
| t' value | 1.647 | |
| p' value | 0.105 Not significant | |

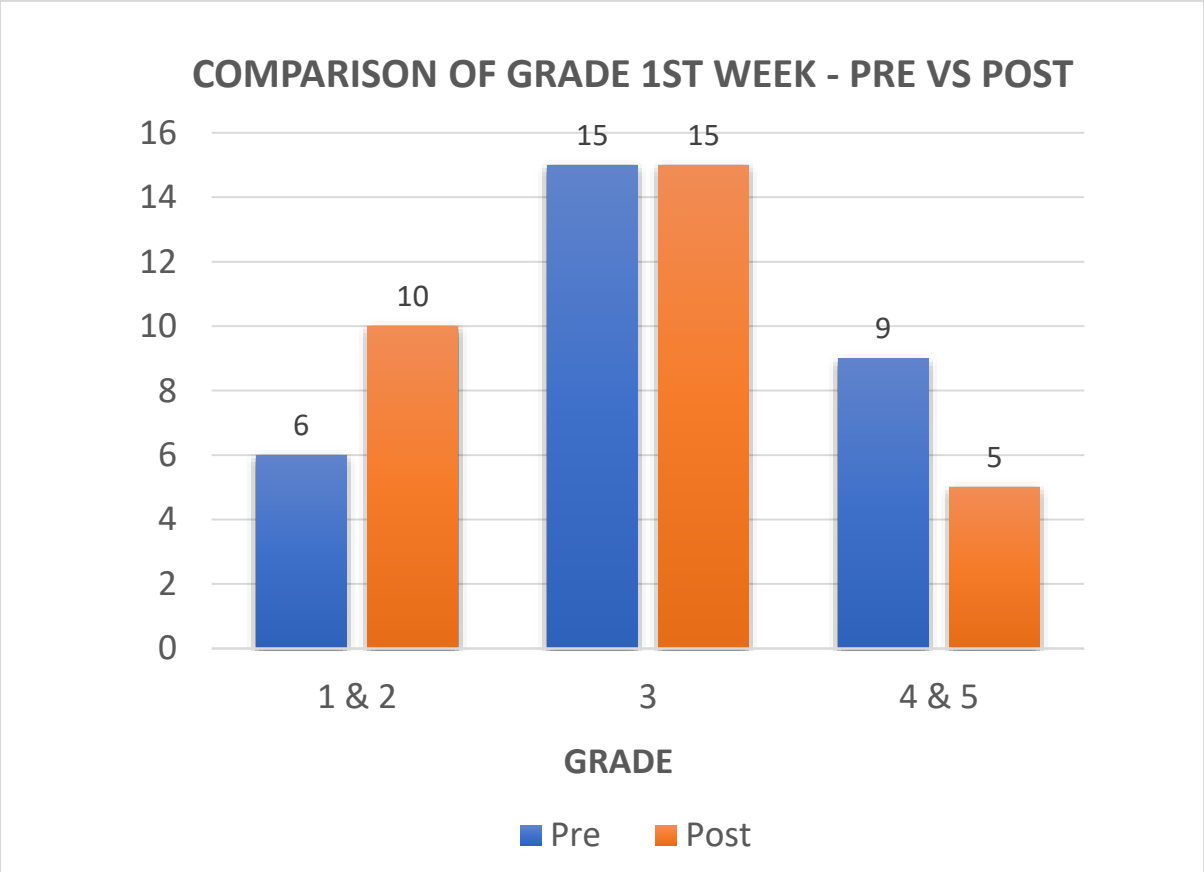
When comparing the THI Score before treatment and 1 week after the doses of intratympanic therapy, the results were not significant.



## COMPARISON OF GRADING OF THI SCORE PRETREATMENT AND 1 WEEK AFTER TREATMENT:

| PRETREATMENT VS 1 WEEK AFTER TREATMENT | | |
|---|---|---|
| GRADE | MEAN | SD |
| PRETREATMENT | 3.067 | 0.907 |
| 1 WEEK AFTER TREATMENT | 2.767 | 0.817 |
| t' value | 1.346 | |
| p' 'value | 0.184 Not significant | |

When comparing the Grading of THI Scores before treatment and 1 week after the doses of intratympanic therapy, the results were not significant.



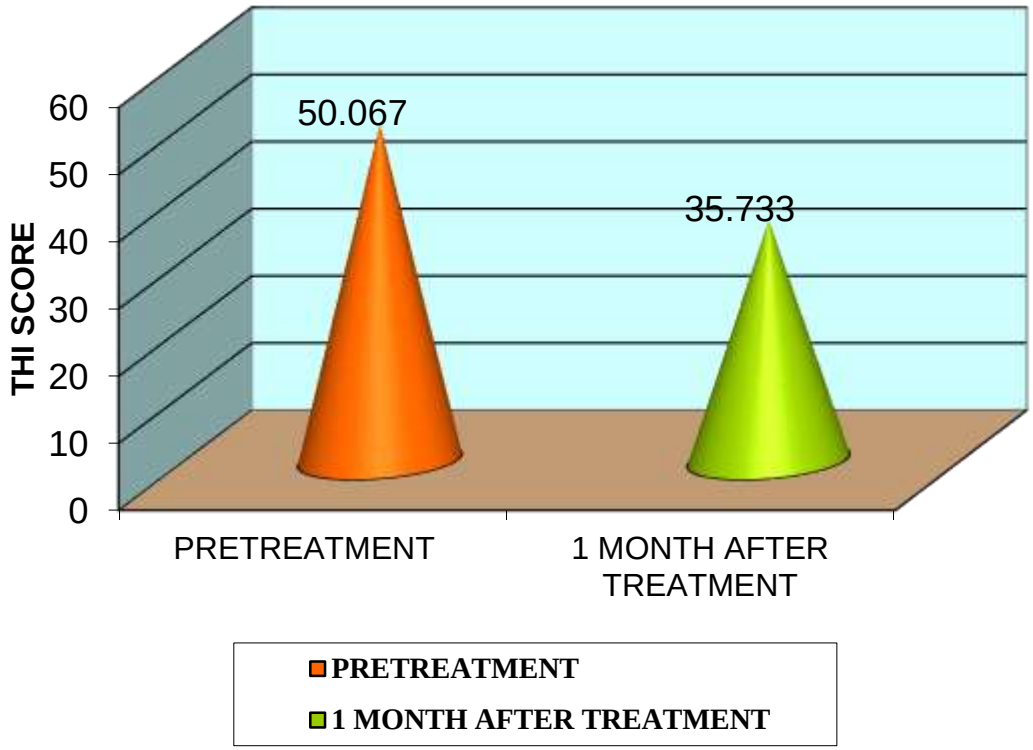
Before treatment, Number of patients in grade 1 & 2, grade 3, grade 4 & 5 were 6, 15,9 respectively. After 1 week of intratympanic therapy, among total of 30 patients, only 4 patients improved to grade 1 & 2. So the results after 1 week were inconclusive.

**COMPARISON OF THI SCORES - PRETREATMENT AND 1 MONTH AFTER TREATMENT:**

| PRETREATMENT VS 1 MONTH AFTER TREATMENT | | |
|---|---|---|
| THI SCORE | MEAN | SD |
| PRETREATMENT | 50.067 | 16.942 |
| 1 MONTH AFTER TREATMENT | 35.733 | 12.247 |
| t' value | 3.755 | |
| p' 'value | <0.001 Significant | |

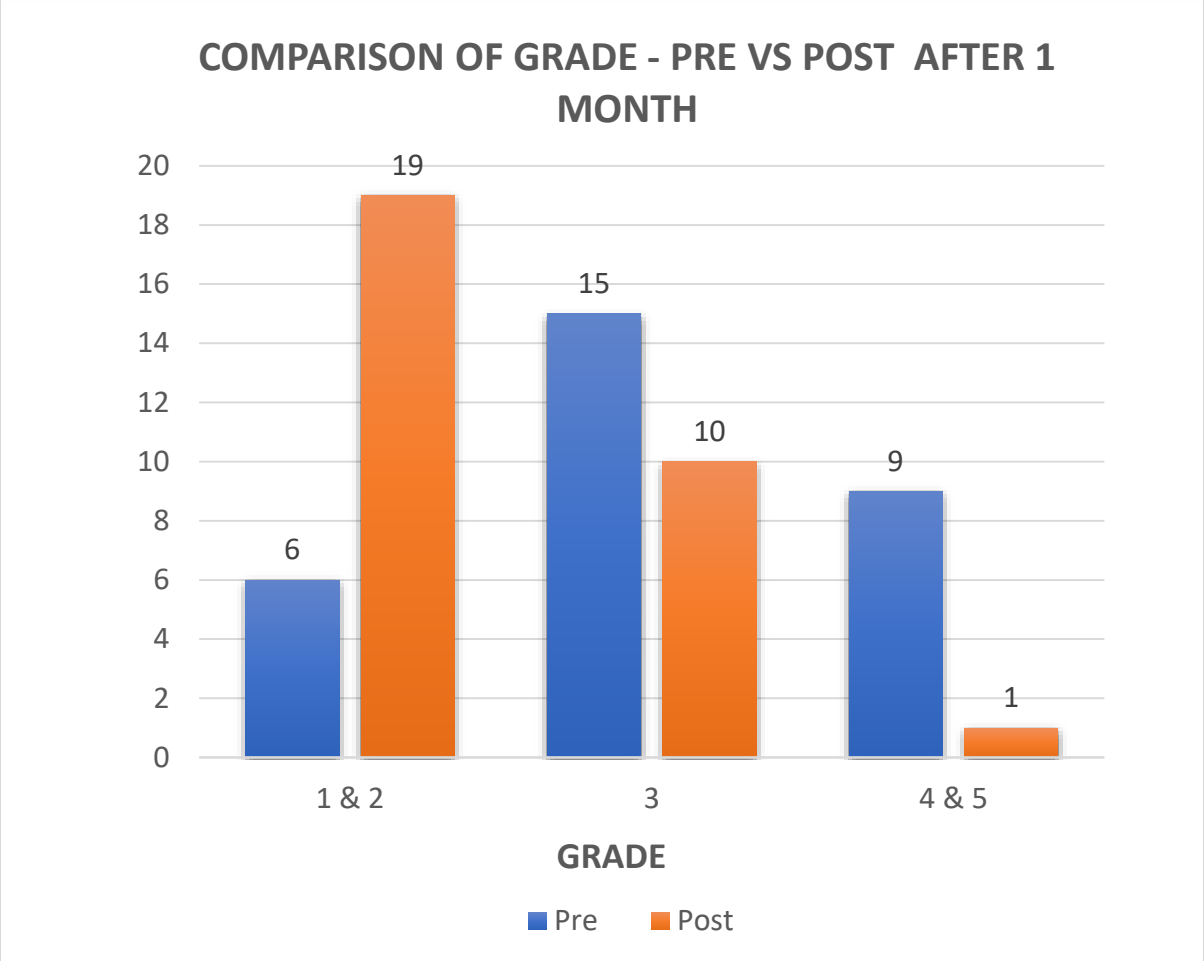
When comparing the THI Score before treatment and 1 month after the doses of intratympanic therapy, Both the mean and the standard deviation were significantly reduced and hence the results were significant.

**MEAN THI SCORE - PRE VS 1 MONTH AFTER TREATMENT**




## COMPARISON OF GRADING OF THI SCORE PRETREATMENT AND 1 MONTH AFTER TREATMENT:

| PRETREATMENT VS 1 MONTH AFTER TREATMENT | | |
|---|---|---|
| GRADE | MEAN | SD |
| PRETREATMENT | 3.067 | 0.907 |
| 1 MONTH AFTER TREATMENT | 2.333 | 0.661 |
| t' value | 3.579 | |
| p' 'value | <0.001 Significant | |

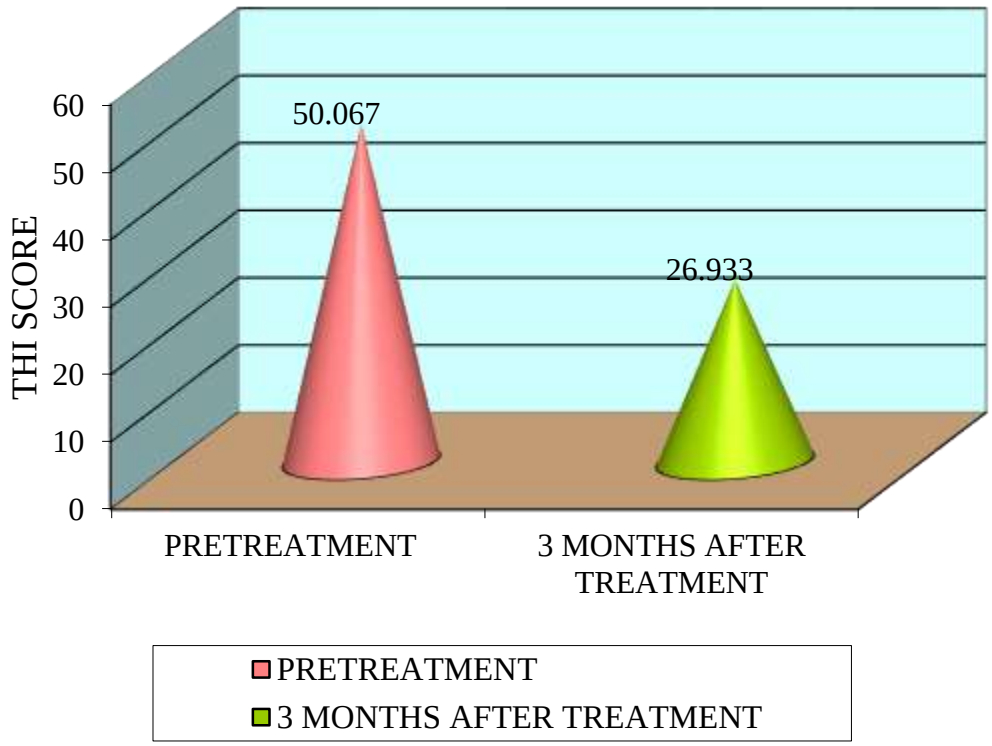



When comparing the results of pre treatment and 1 month of the doses of intratympanic therapy, there are significant number of people were under grade 1 & 2 which implies that grading of tinnitus is improved and the results were much significant.

## COMPARISON OF THI SCORES - PRETREATMENT AND 3 MONTHS AFTER TREATMENT:

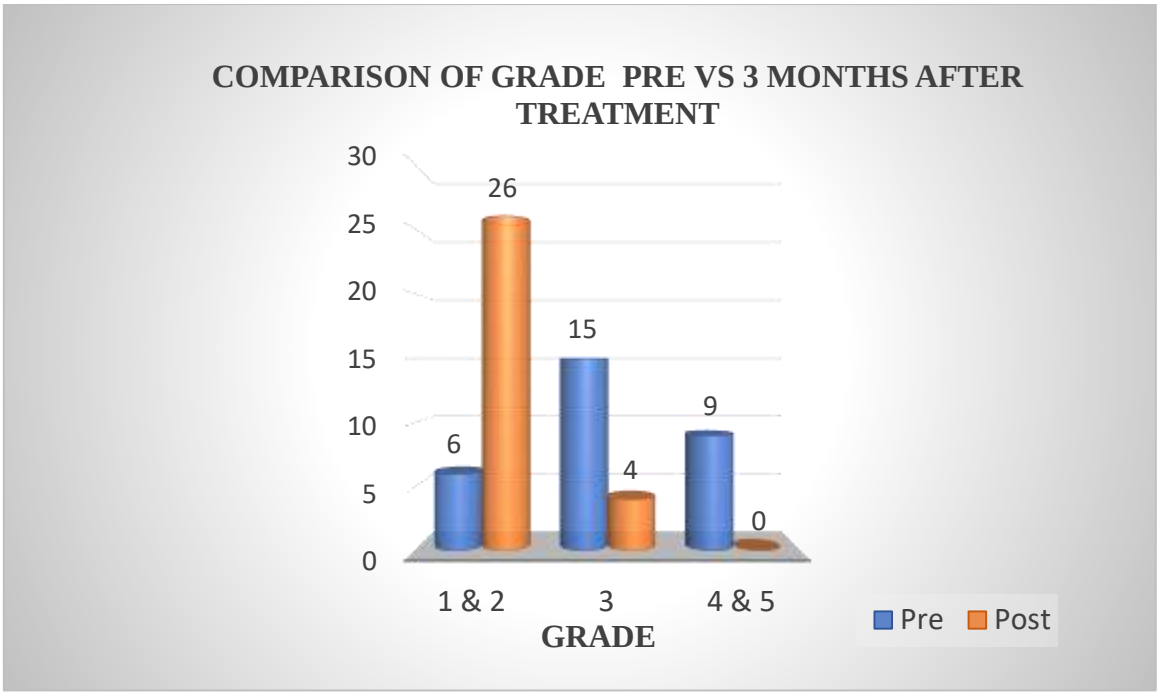
| PRETREATMENT VS 3 MONTHS AFTER TREATMENT | | |
|---|---|---|
| THI SCORE | MEAN | SD |
| PRETREATMENT | 50.067 | 16.942 |
| 3 MONTHS AFTER TREATMENT | 26.933 | 11.335 |
| t' value | 6.216 | |
| p' 'value | <0.001 Significant | |

**MEAN THI SCORE - PRE VS 3 MONTHS AFTER TREATMENT**



## COMPARISON OF GRADING OF THI SCORE PRETREATMENT AND 3 MONTHS AFTER TREATMENT:

| PRETREATMENT VS 3 MONTHS AFTER TREATMENT | | |
|---|---|---|
| GRADE | MEAN | SD |
| PRETREATMENT | 3.067 | 0.907 |
| 3 MONTHS AFTER TREATMENT | 1.9 | 0.607 |
| t' value | 5.853 | |
| p' 'value | <0.001 Significant | |

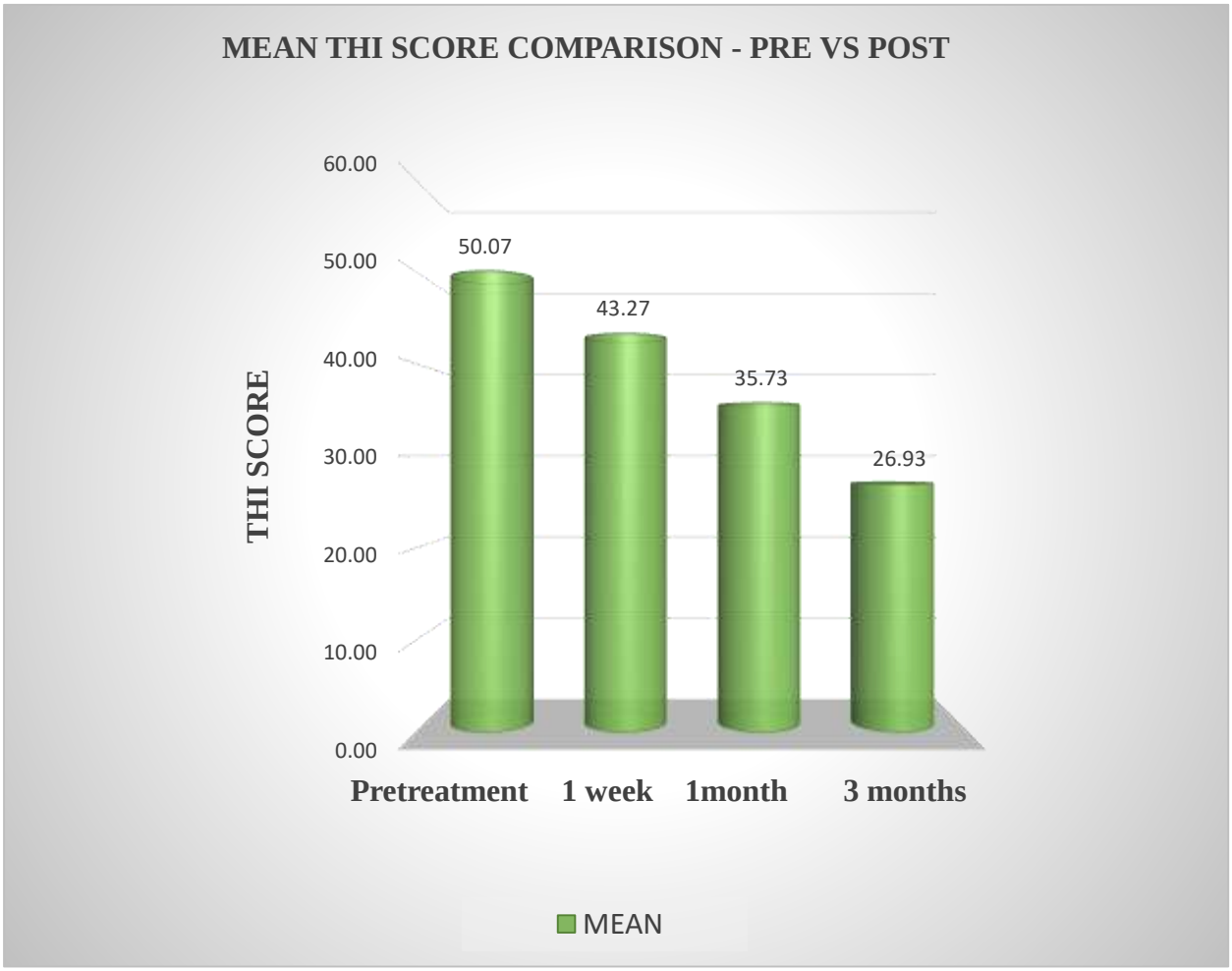



When comparing the results of pre treatment and 3 months after the doses of intratympanic therapy, the results were much significant.

Over all comparison of the mean values of THI SCORES before treatment and after 1 week, 1 month and 3 months of intratympanic therapy.

| THI SCORE | MEAN |
|---|---|
| PRETREATMENT | 50.07 |
| 1 WEEK AFTER TREATMENT | 43.27 |
| 1 MONTH AFTER TREATMENT | 35.73 |
| 3 MONTHS AFTER TREATMENT | 26.93 |

From the above table, the mean THI SCORE has been significantly reduced in 1 month and 3 months after the treatment. Hence the patient has been clinically improved from the symptom of tinnitus.



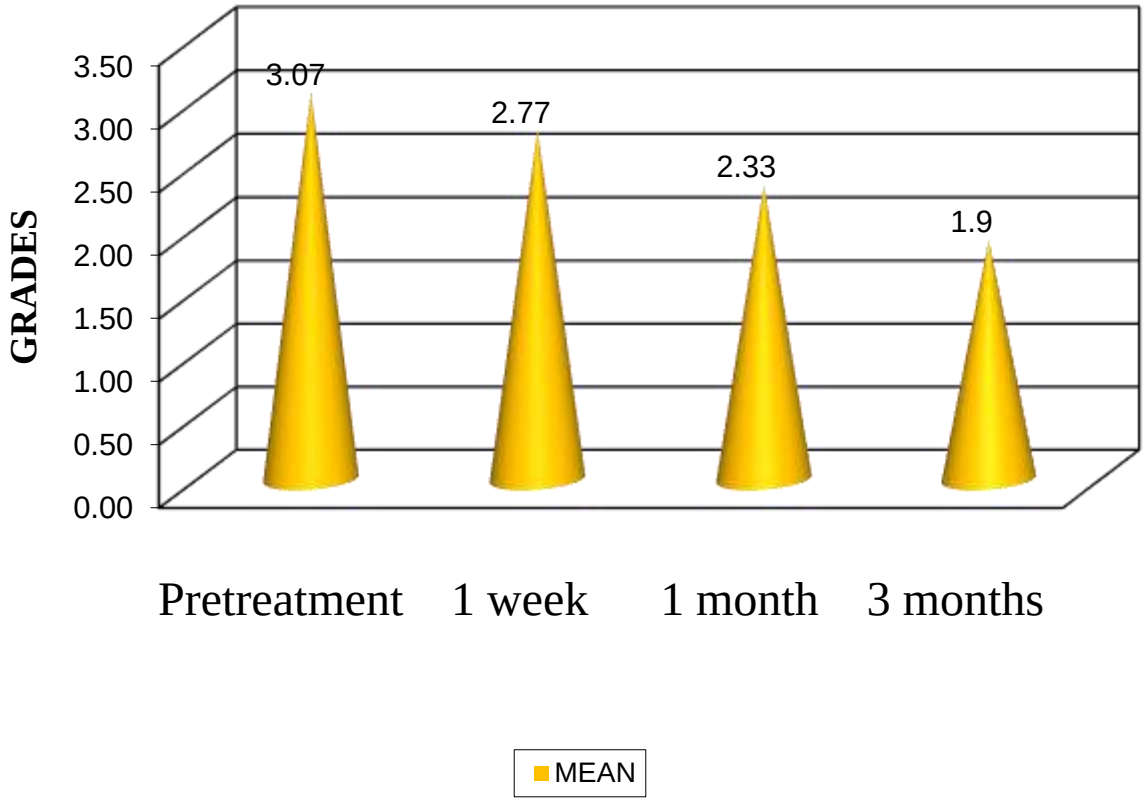

The above graphical representation concludes that the mean THI SCORE values has been significantly reduced after 3 months of the intratympanic dexamethasone therapy.

Over all comparison of the mean values of GRADES OF THI SCORES before treatment and after 1 week, 1 month and 3 months of intratympanic therapy.

| GRADE | MEAN |
|---|---|
| PRETREATMENT | 3.07 |
| 1 WEEK AFTER TREATMENT | 2.77 |
| 1 MONTH AFTER TREATMENT | 2.33 |
| 3 MONTHS AFTER TREATMENT | 1.9 |

From the above table, there was a statistically significant improvement in the GRADES OF THI SCORE after of the treatment. Hence the patient has been clinically improved from the symptom of tinnitus.

**MEAN GRADE COMPARISON - PRE VS POST**




The above graphical representation concludes that the mean values of GRADES OF THI SCORE values has been significantly reduced after 3 months of the intratympanic dexamethasone therapy.

**RESULTS:**

The results of this study show that the intratympanic injection of dexamethasone was found to be effective in the treatment of subjective idiopathic tinnitus. Intratympanic treatment was demonstrated to improve tinnitus scores in the study population.

# DISCUSSION

Our study was a prospective observational study that involved 30 patients with complaints of tinnitus.

Most of our study patients presenting with tinnitus belonged to the age group of 31-40 years. Because children tend to notice less about tinnitus and they seem to be less bothered by it such that they hardly complain of such a symptom.

In our study, females showed a slight preponderance (53.3%) compared to males (46.6%) in the presentation of tinnitus. Working males may ignore their symptoms, while females tend to get more affected and annoyed by the symptom to a greater extent. Psychological comorbidities are also found more commonly among females, contributing to tinnitus.

Among the various types of tinnitus, in our study, we included the patients who had tinnitus which is subjective in nature.

A detailed ENT examination which included an otoscopic and otoendoscopic examination was done for examining the ear to help assess the status of the external and middle ear.

The status of the tympanic membrane was assessed. Most of our study patients who presented to us with tinnitus were found to have normal tympanic membrane on otoscopic examination. And we excluded the patients who have perforation in the tympanic membrane.

Pure tone audiometry (PTA) was performed in all patients and hearing loss was assessed quantitatively and qualitatively. In our study, we included the patients had hearing within normal limits.

Impedance audiometry was also performed and it helped confirm middle ear pathologies like serous otitis media and otosclerosis.

Routine blood investigations were done along with radiological investigations were also done to confirm many of the diagnosis and to evaluate the extent of the disease.

After making the diagnosis, the patients who solely presented with tinnitus with no other otological conditions and systemic diseases were considered to be Tinnitus of Idiopathic in origin. And those patients were included in the study.

Our questionnaire THI helped us to evaluate the various characteristics of tinnitus.

This questionnarie helped to direct towards the diagnosis and also helps to assess the severity of tinnitus to decide on the modality of treatment to be considered. The severity of tinnitus was graded in our questionnaire based on the degree of tinnitus.

A step-wise individualized treatment protocol was followed for treating our study patients considering various factors like age, presence of comorbidities, presence of associated factors, severity of tinnitus etc. Specific causes were diagnosed and referred for specific treatment.

The various classes of drugs tried in patients who were diagnosed to have **subjective idiopathic tinnitus** like Labyrinthine sedatives like Betahistine and Cinnarizine, Microvasodilators, Antipsychotics including Antidepressants and Antianxiety drugs, Neurotropics and Antioxidants, Antihistamines.

Treatment of tinnitus currently includes hearing aids with masking, Tinnitus retraining therapy and oral medications which have limited efficacy.

Hence, Intratympanic treatment with steroid is an emerging treatment option for the idiopathic tinnitus. The most important factors that determine the efficacy of the intratympanic therapy of tinnitus is time and drug dose.

Intratympanic perfusion time and frequency is one of the key factors determining the effectiveness of treatment.

Patients who failed to respond to the above medical treatment were selected for intratympanic drug injection.

In our study, 30 patients with subjective Idiopathic tinnitus, We found that after completing three doses of intratympanic injection of Dexamethasone,

At the first follow up that is 1 week after the treatment, there was no significant improvement in the tinnitus.

At the second follow up that is 1 month after the last injection, there was a mild improvement in the tinnitus.

At the third visit, that is 3 months after the last injection, there was statistically significant improvement in the tinnitus.

The advantage of intratympanic steroid injection is adverse effects of the systemic administration of the drug was avoided.

Local side effects: may include injection site pain, dizziness, infection, persistent perforation of the membrane. In our study, no significant side effects were noted.

Sufficient warming of the drug, the use of fine needles and appropriate local anesthesia , a gentle rate of injection and avoidance of excessive volume of injection are the key factors for good local tolerance.

# CONCLUSION

Tinnitus is a global burden known to mankind since old age. It is not a disease but is a symptom of any possible underlying pathologies which may be of mild to even life-threatening forms.

Each of the characteristics of tinnitus may signify a underlying pathology, hence it should not be ignored even if it is not found to cause distress or affect the lifestyle. But in most of the cases, patients are present to hospitals or clinics with tinnitus only when it starts to bother or disturb them.

Reaching the correct diagnosis itself may take long and demands a proper procedure involving a detailed history taking and examination, along with audiometric tests and adequate investigations to substantiate.

Every possibility of a life-threatening pathology needs to be excluded before arriving at the final diagnosis. Tinnitus may be prevented by attaining preventive measures to most of the underlying causes.

Improving lifestyle by attaining good dietary habits and avoiding all the addictive habits and excessive noise exposure, controlling systemic illnesses by having regular check-ups and follow ups helps to reduce the occurence.

There is no single treatment modality for the cure of tinnitus.

Keeping in mind the etiological diagnosis and the goal of attaining a maximum symptomatic relief, different modalities should be considered.

In cases where the causes are treatable, specific medical or surgical mode of treatment should be adopted.

In cases, where the causes are non-treatable, symptomatic relief is aimed in the form of reducing the distress associated with tinnitus and decreasing its intensity and improves the quality of life.

Intratympanic injection of Dexamethasone could be simple and effective method for controlling the Subjective Idiopathic Tinnitus. The tinnitus will be alleviated, thereby enabling the patient to cope more easily with the disease and reducing the patient's handicap and improving the quality of life.

In our study, the treatment efficacy of the Intratympanic injection of Dexamethasone on tinnitus severity was statistically significant ($p < 0.001$). The patients in our study, experienced no side effects after the treatment.

Hence that Intratympanic steroid injections could be considered as a viable option for the treatment of tinnitus.

## SUMMARY

Our study done in Madurai Medical college and Hospital, Madurai for a period of 1 year was with the aim of studying the efficacy of Intratympanic Injection of steroid for treatment of Subjective Idiopathic tinnitus.

The patients included in our study were both males and females between the age of 15 and 55 years. The patients excluded from our study were those who failed to comply and follow up.

All our study patients were evaluated with a detailed history, a questionnaire which was in reference with the THI, general physical examination, complete ENT Examination which included otoscopic and otoendoscopic examination, audiometric tests and the required investigations like radiological imagings.

Based on the above, patients were diagnosed and the etiologies were identified. And the patients with Subjective Idiopathic Tinnitus were identified and included in our study.

Tinnitus may not be completely curable in all cases.

But in most of the patients, we were able to achieve symptomatic relief in the form of reduction in intensity and severity of tinnitus leading to an improvement in quality of life.

Intratympanic injection of Dexamethasone is gaining popularity as the treatment of choice for the treatment of Subjective Idiopathic Tinnitus.

For that patients were selected appropriately, and intratympanic injection of Dexamethasone was given as three injections for 3 weeks and the patients were followed up for 3 months.

Symptomatic relief and clinical improvement of tinnitus assessed by means of subjective evaluation with the help of a Questionnarie called TINNITUS HANDICAP INVENTORY.

Hence the results of this study demonstrates that weekly administration of Intratympanic dexamethasone injections in controlling the subjective idiopathic tinnitus and it improves the quality of life.

We convey the message from our study, is that the importance of educating the public about the importance of attaining a good and healthy lifestyle and protecting the ears for helping in the prevention of the onset of tinnitus. Good physical fitness and relaxation helps reduce the distress associated with tinnitus.

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## ANNEXURE

## PROFORMA

Name:

Age: Address:

Sex: Ip.no:

Occupation: Socio economic status:

**Chief complaints:**

Side Duration

Ringing sensation in Ear

**H/O Presenting Illness:**

Ringing sensation Side

Duration

Progress

Character

High pitched/low pitched voice

Sleep disturbances

H/o drug abuse

H/o Noise exposure

H/o Ear discharge

H/o Hard of Hearing

H/o Itching sensation

H/o Ear pain

H/o Headache

H/o Giddiness

H/o Fever

H/o Recurrent cold and cough

H/o Nasal discharge

H/o Nasal obstruction

H/o Nasal Bleeding

H/o Throat pain

H/o Difficulty in swallowing

H/o change in voice

**Past History:**

H/o DM/ SHT/ PTB/ Epilepsy/ Cardiovascular diseases/ Thyroid disorders/ Exanthematous fever/Bronchial asthma

H/o Trauma

H/o Previous surgeries

H/o Blood transfusion

**Personal History:**

Mixed diet

Bowel and bladder disturbances

H/o Smoking/ alcohol/ occupation

**Treatment History:**

H/o any ototoxic drugs intake

**General Examination:**

Conscious, Oriented, Built, Nourishment

Pallor Clubbing

Cyanosis Pedal edema

Icterus Generalised lymphadenopathy

**Vitals**:

Pulse rate:

Blood pressure:

Respiratory rate:

Temperature:

**Local Examination:**

**EAR** **Right** **Left**

Pinna:

Size/Shape/Position

Congenital/Acquired deformity

Preauricular region

Postauricular region

External Auditory canal

Tympanic Membrane

Pars Tensa

Pars Flaccida

Facial Nerve

Three finger Test

Fistula Test

Tuning fork Tests:

Rinne

Weber

ABC Test

**Examination of Nose:**

External Nasal Contour

Anterior Rhinoscopy:

Nasal septum –

Turbinate-

Nasal mucosa-

Posterior Rhinoscopy:

Cold spatula test:

Cotton wool test:

**Examination of Throat:**

Oral cavity: Lips/ Gingiva/ Teeth/ Gingivobuccal sulcus/ gingivolabial sulcus

Buccal mucosa/Hard palate/Floor of mouth

Oropharynx: Anterior pillar

Tonsillar fossa

Posterior pillar

**Examination of Neck:**

No palpable lymph nodes

**Provisional Diagnosis:**

**Investigations:**

Examination under microscope:

Aural swab – culture & sensitivity

Routine blood investigations

Pure Tone Audiometry

Diagnostic Nasal Endoscopy

X ray both Mastoids – lateral oblique view

Tinnitus Handicap Inventory (THI):

**Treatment:**

Initial conservative management:

Intratympanic Injection:

**Post-operative Follow up:**

Subjective evaluation by Tinnitus Handicap Inventory:

# MASTER CHART

| S.NO. | AGE | SEX | PRE TREATMENT | | 1WEEK AFTER TREATMENT | | 1 MONTH AFTER TREATMENT | | 3 MONTHS AFTER TREATMENT | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | THI SCORE | GRADE | THI SCORE | GRADE | THI SCORE | GRADE | THI SCORE | GRADE |
| 1 | 36 | M | 48 | 3 | 48 | 3 | 40 | 3 | 34 | 2 |
| 2 | 45 | F | 60 | 4 | 54 | 3 | 50 | 3 | 46 | 3 |
| 3 | 29 | F | 34 | 2 | 32 | 2 | 32 | 2 | 32 | 2 |
| 4 | 34 | M | 14 | 1 | 10 | 1 | 10 | 1 | 8 | 1 |
| 5 | 28 | M | 62 | 4 | 58 | 4 | 50 | 3 | 46 | 3 |
| 6 | 35 | F | 48 | 3 | 44 | 3 | 36 | 2 | 30 | 2 |
| 7 | 27 | M | 56 | 3 | 48 | 3 | 36 | 2 | 16 | 1 |
| 8 | 39 | F | 74 | 4 | 58 | 4 | 54 | 3 | 48 | 3 |
| 9 | 40 | F | 46 | 3 | 42 | 3 | 34 | 2 | 16 | 1 |
| 10 | 42 | F | 60 | 4 | 56 | 3 | 48 | 3 | 36 | 2 |
| 11 | 38 | M | 56 | 3 | 48 | 3 | 34 | 2 | 24 | 2 |
| 12 | 28 | M | 80 | 5 | 74 | 4 | 58 | 4 | 36 | 2 |
| 13 | 32 | F | 62 | 3 | 52 | 3 | 40 | 3 | 16 | 1 |
| 14 | 30 | M | 66 | 3 | 50 | 3 | 36 | 2 | 24 | 2 |
| 15 | 38 | F | 72 | 4 | 66 | 4 | 54 | 3 | 50 | 3 |
| 16 | 42 | M | 36 | 2 | 34 | 2 | 30 | 2 | 16 | 1 |
| 17 | 36 | F | 74 | 4 | 60 | 4 | 56 | 3 | 36 | 2 |
| 18 | 28 | F | 44 | 3 | 38 | 3 | 32 | 2 | 26 | 2 |
| 19 | 40 | F | 54 | 3 | 42 | 3 | 34 | 2 | 14 | 1 |
| 20 | 38 | M | 64 | 4 | 56 | 3 | 40 | 3 | 36 | 2 |
| 21 | 39 | M | 56 | 3 | 42 | 3 | 34 | 2 | 28 | 2 |
| 22 | 22 | F | 14 | 1 | 10 | 1 | 10 | 1 | 10 | 1 |
| 23 | 28 | M | 38 | 3 | 32 | 2 | 28 | 2 | 22 | 2 |
| 24 | 26 | F | 60 | 4 | 52 | 3 | 38 | 3 | 32 | 2 |
| 25 | 32 | F | 46 | 3 | 38 | 3 | 32 | 2 | 24 | 2 |
| 26 | 38 | M | 20 | 2 | 18 | 2 | 18 | 2 | 18 | 2 |
| 27 | 44 | F | 40 | 3 | 36 | 2 | 30 | 2 | 20 | 2 |
| 28 | 38 | M | 40 | 3 | 34 | 2 | 28 | 2 | 22 | 2 |
| 29 | 40 | F | 36 | 2 | 30 | 2 | 20 | 2 | 18 | 2 |
| 30 | 29 | M | 42 | 3 | 36 | 2 | 30 | 2 | 24 | 2 |

## PATIENT CONSENT FORM

Participation Name: Address:

**Title of the project**: **A STUDY ON EFFICACY OF THE INTRATYMPANIC INJECTION OF STEROID IN THE TREATMENT OF SUBJECTIVE IDIOPATHIC TINNITUS**

The details of the study have been provided to me in writing and explained to me in my own language. I confirm that I have understood the above study and had the opportunity to ask questions. I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without the medical care that will normally be provided by the hospital being affected. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purposes. I have been given an information sheet giving details of the study. I fully consent to participate in the above study.

Signature of the participant: Date:

Signature of the Witness: Date:

Signature of the Investigator: Date:

# ஆராய் ச்சிஒப்புதல்படிவம்

ஆராய்ச்சியின்தலைப்பு: **A STUDY ON EFFICACY OF THE INTRATYMPANIC INJECTION OF STEROID IN THE TREATMENT OF SUBJECTIVE IDIOPATHIC TINNITUS**

பெயர்: தேதி:

வயது:

நோயாளி எண்:

ஆராய்ச்சி சேர்க்கை எண்:

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு விளக்கபட்டது.

எனக்கு விளக்கபட்ட விஷயங்களை நான் புரிந்து கொண்டு எனது முழுமனதுடன் சம்மதிக்கிறேன்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில்தான் பங்குபெறுகிறேன். மற்றும் நான் இந்த ஆராய்ச்சியில் இருந்து எந்த நேரமும் பின்வாங்கலாம் என்றும் அதனால் எந்த பாதிப்பும் எனக்கு ஏற்படாது என்பதையும் புரிந்து கொண்டேன்.

நான் என்னுடைய சுயநினைவுடன் மற்றும் முழுசுதந்திரத்துடன் இந்த மருத்துவ ஆராய்ச்சியில் பங்குகொள்ள சம்மதிக்கிறேன்

**INSTITUTIONAL ETHICS COMMITTEE**
**MADURAI MEDICAL COLLEGE & GOVT. RAJAJI HOSPITAL, MADURAI**
**CDSCO:Reg.No.ECR/1365/Inst/TN/2020 &**
**DHR Reg.No.EC/NEW/INST/2020/484**

| | | |
|---|---|---|
| **Study Title** | : | A study on efficacy of the intratympanic injection of steroid in the treatment of subjective idiopathic tinnitus. |
| **Principle Investigator** | : | Dr.Selvapriya .E |
| **Designation** | : | PG in MS., Otorhinolaryngology (2019 – 2022) |
| **Guide** | : | Dr.N.Dhinakaran<br>Professor & HOD of ENT |
| **Department** | : | Department of ENT<br>Govt. Rajaji Hospital & Madurai Medical College,<br>Madurai. |

The request for an approval from the Institutional Ethics Committee (IEC) was considered on the IEC meeting held on **03.03.2021** at Auditorium, Govt. Rajaji Hospital, Madurai at 10.00 AM.

The Members of the committee, the Secretary and the Chairman are pleased to inform you that your proposed project mentioned above is **Approved.**

You should inform the IEC in case of any changes in study procedure, methodology, sample size investigation, Investigator or guide or any other changes.

1. You should not deviate from the area of work for which you had applied for ethical clearance.

2. You should inform the IEC immediately, in case of any adverse events or serious adverse reactions. If encountered during from study.

3. You should abide to the rules and regulations of the institution(s)

4. You should complete the work within the specific period and if any extension is required, you should apply for the permission again for extension period.

5. You should submit the summary of the work to the ethical committee on completion of the study.

**MEMBER SECRETARY,**
IEC, Madurai Medical College,
Madurai.

Dr.K.RAADHIKA, M.D(Pharm)
Associate Professor
Member Secretary
IEC - Madurai Medical College
Madurai.

- 5 MAR 2021

**CHAIRMAN,**
IEC, Madurai Medical College,
Madurai

**Prof. Dr. V. Nagaraajan**
MD.,MNAMS.,DM.,DSC(Neuro).,DSC(Hon)
**CHAIRMAN**
**IEC Madurai Medical College**
**Madurai**

Curiginal

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## Sources included in the report

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