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REVIEW ON CONCEPTUAL STUDY OF KARNA NADA IN CORRELATION WITH TINNITUS

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ABSTRACT

Shalakya Tantra is one of the eight principal branches of *Aṣhtanga Ayurveda*, concerned with the diagnosis and management of diseases affecting structures above the clavicle, including the eyes, nose, throat, ear, and head. *Karna Nada* is described as one among the twenty-eight *Karṇa Rogas* in classical Ayurvedic texts such as *Susruta Saṃhita*, *Bhavaprakasha*, and *Yoga Ratnakara*. Clinically, *Karna Nada* can be correlated with Tinnitus as described in modern medical literature. According to Ayurvedic principles, when *Vata Dosha* becomes localized in the auditory channels of the ear, the individual perceives abnormal sounds resembling those of a *Bheri*, *Mṛudanga*, *Shankha*, and similar instruments; this condition is termed *Karna Nada*. The prevalence of tinnitus among the adult population is estimated to range between 6% and 19%, indicating its significant impact on quality of life. Various theories have been proposed in contemporary medicine, to explain the underlying mechanisms of Tinnitus. Ayurvedic management includes therapeutic procedures such as *Snigdha Virechana*, *Naḍi Sveda*, *Piṇḍa Sveda*, *Dhupana*, *Basti Karma*, *Navana Nasya*, *Abhyanga*, and *Karnapooraṇa*. In modern medicine, treatment modalities comprise medical and surgical interventions, pharmacotherapy, acoustic

therapy, counselling and patient education, Cognitive Behavioural Therapy (CBT), Tinnitus Retraining Therapy (TRT), and electrical stimulation.

INTRODUCTION

Ayurveda is a holistic and systematic approach to total healthcare. *Shalakya Tantra* is one among the eight branches of *Ashtanga Ayurveda*. *Shalakya Tantra* deals with diseases that affect part above the neck like eyes, nose, throat, ear, and head.

According to *Acharya Susruta*, there are 28 *Karna Roga's* and *Karna Nada* is one among them[1]. In modern literature it can be correlated with Tinnitus. The term Tinnitus derives from the Latin word 'Tinnire' meaning 'To ring'. Tinnitus is the perception of the sound in the ear or the head. Patients exhibit a wide range of reactions both physical and psychological to the presence of tinnitus. The psychological distress that are associated with tinnitus include worries, anxiety, depression, irritability, disturbance in social life, stress, tiredness, feelings of illness, disturbance in concentration, personality disorder and sleep disturbance. Different treatment methods like medical treatment, surgical treatment, palliative treatment, use of tinnitus maskers and hearing aid if there is accompanying hearing loss, electrical stimulation, psychotherapy, relaxation therapy etc. are used for tinnitus but with very little success.

Now days *Ayurveda, Yoga*, meditation (*Pranayama*) and relaxation therapy have been in use to help the patient to cope up with the irritation and distressing aspect of tinnitus.[2]

Karna Nada

Karna Nada is one among twenty eight *Karna Roga's* explained in *Susruta Samhita, Bhava Prakasha and Yoga Ratnakara*,the main Dosha are involved here is *Vata Dosha*. According to *Astanga Hridaya*, *Karna Nada* is one among the *Karna Srotogato Roga* and is a *Sadhya Vyadhi*.

Definition: The term *Karna Nada* is basically derived from two root words *Karna* and *Nada*. *Karna*–Means ear or organ of hearing. *Nada*–Sound or ringing in the ear.The term *Karna* refers to organs which are responsible for the perception of the sound (include external, middle and internal ear). The term *Nada* or *Ninada* refers to that which

produces rhythmic sounds in the ear. Diseases can be well known by these five measures, which are described in *Ayurveda Samhitas*.[3]

1.*Nidana* (etiology, causes)

2.*Purvarupa*(premonitory symptoms)

3.*Rupa*(signs and symptoms, clinical feature)

4.*Upasaya*(diagnostic test)

5.*Samprapti* (process of pathogenesis of disclosure)

Nidana: General etiological factors for *Karna Nada* are mentioned in Table 1.

Table 1: *Nidana*

Sr. No	*Nidana of Karna Rogas*	*Sushruta Samhita*	*Ashtanga Hridaya*	*Yog Ratnakara*
1.	*Avashyaya*	+	+	+
2.	*Jalakreeda*	+	+	+
3.	*Karnakandooyana*	+	+	+
4.	*Mithya yogena Shastrasya*	+	-	+
5.	*Mithya yoga of Shabda*	-	+	-
6.	*Abhigaata*	+	+	+
7.	*Nitya Shira Snaana*	+	+	+
8.	*Pratishyaya*	-	+	-

The following are the etiological factors responsible for the causation of the clinical condition of *Karna Nada*

1. *Pratisyaya* (common cold) - *Pratisyaya* is said to be a cause of all the *Urdhwa Jatrugata Rogas*, it is causes by vitiated *Vata Dosha*.

2. *Avasyaayam* (exposure to cold wind) excessive contact of humid weather is *Avasyaaya*. Especially, during winter season it causes vitiation of *Vata Dosa* of its *Shitatva* properties. In Ayurveda, the function of *Vata* can be compared to that of functions of nervous system. Hence the vitiation of *Vata Dosa* due to excessive contact of *Avasyaaya* will affect the functions of auditory nerve. This leads to *Karna Nada*.

3. *Jalakrida* (swimming and play in rainy) due to entering of water into external ear by taking bath in sea, rivers fountains etc., may cause moistening of wax. The moistened wax swells up and occludes the meatus causing giddiness, vomiting, and noises in the ear.

4. *Karna kanduyana* (keeping matchsticks, pin, or other sharp edged objects in the ear).

5. *Sastra-mithyayoga* (*Avisodhita yantras* and *sastras* which are inserted in the *Karnam* for diagnostic and surgical procedures for removal of foreign body etc., will cause *Ksatam* and *Upasargam* in the *Karna* leading to *Karnasula*, *Karna Badhiryam*, *Putikarnam* etc.).

6. *Atiyoga, Ayoga, Mithya Yoga* of the *Shabda* (Incompatible correlation of sensation of sound (high, low, medium pitched sound) with the organ of hearing). *Karna Nada* is a *Vata Dosa Pradhana Vyadhi* and *Samanya Nidana* of *Vata Roga* can be applied to *Karna Nada. Vata Prakopa* is attributed as the prime etiological factor for the causation of *Karna Nada*.

The following factors which are responsible for the pathogenesis of *Vata Vyadhi* are

1. Excessive intake of unctuous, cold, scanty and light food.
2. Excessive sexual indulgence.
3. Remaining awake at night in excess.
4. Inappropriate therapeutic measures.
5. Administration of therapies which cause excessive elimination of *Dosha* and blood.
6. Keeping fast in excess.
7. Excess Swimming.
8. Resorting to wayfaring, exercise and other physical activities in excess.
9. Loss of *Dhatus* (tissue elements).
10. Excessive emaciation because of worry, grief and affliction by diseases.
11. Sleeping over uncomfortable beds, and sitting over uncomfortable seats.
12. Injuries to *Marmas* (vital spots in the body).
13. Riding over elephants, camel, horse or fast moving vehicles and falling down from the seats on these animals and vehicles.

Acharya Harita has mentioned the *Karna Nada* etiology and symptoms.[4] Those are the :

1. Stagnancy of water or entry of water in the ear.
2. Debris accumulation in ear.
3. *Karna Shoola.*
4. *Shwas Vega Dharana*
5. Injury and *Krimi*

Purvarupa

Purvarupa indicates the upcoming disease. Stage of *Sthanasamsraya* is term as *Purvarupa*. In stage of *Sthanasamsraya* vitiated gets aggravated and move to different parts and get accumulated is one of the region of the body. In classical literature there is no specific *Purvarupa* (prodromal symptoms) are mentioned in disease of *Karna Nada*. *Acharya Madhavakara* explain *Purvarupa* as precursor of *Rupa Laksanas* with less intensity, which

could be taken as less intensity of sound in ear, or shorter duration or intermittent frequency be considered as *Purvarupa* of *Karna Nada*.

Acharya Charaka explain are; *Avyakta Laksanam Tesam Purvarupama Iti Smrtama*. Ch. Chi. 28/19 *Avyakta Laksanas* (means unknown symptom) are the *Purvarupa* of *Vata Vyadhi*. [5]

Rupa: *Nanavidha Shabda*- Different kinds of sounds heard in *Shabdavaha Srotas*.[6] *Vividhan Sabda* is heard in *Shabdavaha Srotas* are as follow: *Bheri, Mrudanga, Samkha, Bhrungara, Kaunca, Mandura, Tantri, Vividha Shabda* etc.[7]

Samprapti: *Samprapti* is the process of manifestation of the disease by the morbid *Dosha* which are circulating all over the body. When the *Vata* gets localised in the channels of the ear the patient hears various types of sound like those of a *Bheri* (bulge/drum), *Mrudanga*, *Shankha* etc.[8]

Chikitsa* of *Karna Nada *:* The *Karna Nada* is a *Vataja Pradhana Dosa Vyadhi*. So our aims are to reduce Vata in the treatment of *Karna Nada*. *Acharya Susruta* mentioned the common treatment for *Karnasula, Karna Nada, Badhirya* and *Karna Kshweda Roga* (Table 1).[9]

Table 2: Specific Treatment According to Different *Acharya*.

Acharya Sushruta	***Acharya Vagbhata*[10]**	***Acharya Charaka*[11]**
• Administration of *Vata Hara Chikitsa*. • Administration of *Snigdha Aushadha*. • *Snigdha Virechana* • *Nadi Sweda* • *Pinda Sweda* •*Dhupana* with *Kshauma, Guggulu* and *Agaru* • *Basti Karma* • *Bala Taila* in the form *Murdha Basti, Nasya, Mastiska Parisechana* and for *Bhojana*.	• *Vataj Karnashoola Hara Chikitsa* • If there is an involvement of *Kapha Dosha* the first *Vamana Karma* is advised. • *Dhumpana* • *Navana* • *Abhyanga* • *Karnapurana* with *Sarshapa Taila*	*Vatahara Chikitsa* • *Pinasahara Chikitsa* • *Pradehakarma* • *Karnapurana* • *Nasyakarma* • *Vranahara Chikitsa*

Tinnitus

Tinnitus is defined as a phantom auditory perception- it is a perception of sound without corresponding acoustic or mechanical correlates in the cochlea. Tinnitus represents one of the

most common and distressing otologic problems, and it causes various somatic and psychological disorders that interfere with the quality of life.

Clinical Manifestation

Characteristics of Tinnitus : The sound perceived by those with Tinnitus can range from a quiet background noise to noise that is audible over loud external sounds. Tinnitus is generally divided into two categories: objective and subjective. Objective tinnitus is defined as tinnitus that is audible to another person as a sound emanating from the ear canal, whereas subjective tinnitus is audible only to the patient and is usually considered to be devoid of an acoustic etiology and associated movements in the cochlear partition of cochlear fluids.

The sounds associated with most cases of tinnitus have been described as being analogous to cicadas, cricket, winds, falling tap water, grinding steel, escaping steam, fluorescent lights, running engines, and so on. It is believed that these types of perception results from abnormal neuronal activity at a subcortical level of the auditory pathway. The pattern characterizing tinnitus is related to the library of patterns stored in auditory memory and also, via the limbic system, associated with emotional states.[12]

Incidence and Prevalence : The prevalence of tinnitus in the adult population is estimated to range from 6% to 19% of adults, depending on the population samples and the definition of tinnitus used in the survey.[13] Risk factors for tinnitus have been identified from large epidemiological surveys and include non -modifiable factors of gender (male) and ethnicity (non-Hispanic Whites), and modifiable factors including body mass index (≥30kg/m2), hypertension, diabetes mellitus, dyslipidemia, anxiety disorder, noise exposure, and smoking.[14]

Causes of Tinnitus : Main causes of subjective tinnitus Otologic problem and hearing loss: Loud noise, presbycusis, Meniere's disease, acoustic neuroma, external ear infection. Pharmacological Causes Analgesics, antibiotics, chemotherapy and anti-viral drugs, loop diuretics, anti-depressants, psychedelic drugs (5-MeO-DET, 5-Methoxydiisopropyltryptamine, diisopropyltryptamine, harmaline, N, N-dimethyltryptamine, psilocybin, salvinorin A.

Neurologic disorders: Traumatic brain injury meningitis, encephalitis, strokes, multiple sclerosis, chiari malformation, auditory nerve injury.

Metabolic Disorders: Thyroid disorders, hyperlipidemia, vitamin B12 deficiency, iron deficiency, anemia.

Psychiatric Disorders: Anxious and depressive states.

Other: Tension myositis syndrome, fibromyalgia, head and neck muscle spasm, Temporomandibular joint disorders, thoracic outlet syndrome, lyme disease, hypnogogia, sleep paralysis, glomus tymapanicum, herpes infections.

Main causes of objective tinnitus

Pulsatile tinnitus: Altered blood flow or increased blood turbulence near the ear: Atherosclerosis, venous hum, carotid artery aneurysm, carotcarotid artery dissection. Muscle contraction that cause clicks or crackling around the middle ear.

Main Hypothesized Pathomechanisms of Tinnitus

Spontaneous Otoacoustic Emissions: Small acoustic signals perceived as tinnitus.

Edge Theory: Increased spontaneous activity in the edge area.

Discordant Theory: Discordant dysfunction of damaged outer hair cells and intact inner hair cells.

Main Central Auditory System and Somato Sensory Theories of Subjective Tinnitus

The dorsal cochlear nucleus: Hyperactivity/ plastic readjustment of DCN.

Auditory plasticity theory: Enhanced neural activity due to cochlear damage.

Crosstalk theory: Ephaptic coupling b/w nerve fibers.

Pathophysiology

The changes in the auditory nervous system, especially at the dorsal (DCN) and ventral cochlear nucleus (VCN) underpinning tinnitus are often represented by a reduction in the inhibitory rather than an excitatory input, resulting in a shift in the balance between inhibition and excitation. Deprivation of input may cause neural plasticity to change the relationship between inhibition and excitation and protein synthesis and finally impact on neurotransmission especially at the DCN, the inferior colliculus (IC), together with the primary and secondary auditory cortices eventually affecting the thalamic and dorsal cortex transmission, the amygdala, and other structures. The rerouting of information may cause structures of the central nervous system (CNS) that are not normally involved in processing auditory information to become activated by sound stimulation (i.e., the abnormal

involvement of the non-classical-non-specific/exrea lemniscalpathways). Yet to date, no univocal or exhaustive appreciation of tinnitus determining neural abnormalities have been reached; with its pathophysiological correlates still remaining a debated issue. Among others, the following evidence and hypotheses have been postulated.

Peripheral Auditory System Spontaneous Otoacoustic Emission: Small acoustic signals presumed to be generated by the electromotile activity of the outer hair cells (OHCs) of the cochlea and propagated into the external auditory canal may be abnormally perceived as tinnitus.

Edge Theory: Increased spontaneous activity in the edge area, which represents a transition from normal OHCs on the apical side of a lesion to OHCs toward the basal side that are missing or altered, may contribute to tinnitus perception.

Discordant Theory: The discordant dysfunction of damaged OHCs and intact inner hair cells (IHCs) may result in the disinhibition and increased spontaneous activity of neurons in the DCNs that receive excitation from IHCs, but with no inputs from the damaged OHCs, therefore playing a role in tinnitus phenomenon.

Central Auditory System

The Dorsal Cochlear Nucleus: Hyperactivity by disinhibition or plastic readjustments of the DCN, triggered for instance by OHC damage or a reduction in auditory nerve input have been supposed to concur in tinnitus genesis.

Auditory Plasticity Theory: Damage to the cochlea enhances neural activity in the central auditorpathway, such as the IC and the temporal lobe of the auditory association cortex (similar to the phantom limb sensation in amputees).

Crosstalk Theory: The compression from blood vessels or a tumor on auditory nerve causes ephaptic coupling between nerve fibers, which may result in the phase-locking of spontaneous activity of groups of auditory neurons that may be perceived as sound.

Somatosensory System: Abnormal DCN activity could also be influenced by the stimulation of the somatosensory system. Furthermore, pain signals from the cochlea carried by the cochlear C fibers may be interpreted by the CNS as tinnitus.

Neurotransmitters: It has been postulated that numerous cochlear neurotransmitter play a role in tinnitus sensation.

GABA: It is known that the number of gamma aminobutyric acid (GABA) immunoreactive neurons in the auditory nuclei decreases with age; together with its concentration and release, the glutamatergic acid decarboxylase activity (the ratelimiting enzyme in the formation of GABA) releasing terminals availability. This trend has been postulated to be a higher risk of noise-induced hearing loss as an important tinnitus etiological factor, especially in older men, and has promptted both researchers and clinicians to consider GABAergic modulators as a candidate therapy for tinnitus patients. Serotonin: Serotonergic (5-HTergic) modulation (eg, by salicylate administration) has been reported to provoke tinnitus, although human genetic studies on the regulatory region of the 5-HT transporter (5- HTTLPR) SLC6A4 gene failed to show any significant effect on the susceptibility to chronic tinnitus, thus indicating an indirect, rather than a direct, modulating role of 5-HT in tinnitus etiology and CNS-adaptive mechanisms. Modulation of 5-HT is a core mechanism of a very large number of routinely prescribed drugs including the selective serotonin reuptake inhibitor (SSRI) antidepressants, thus their putative role in increasing the sensation of tinnitus should therefore be taken into account.

Dopamine: Tinnitus perception takes place in prefrontal, primary temporal and temporoparietal associative areas, as well in the limbic system. Dopamine (DA) neurotransmissions acts through prefrontal, primary temporal, temporoparietal associative areas and the limbic system. Tinnitusperception and DA-ergic pathways share the same cerebral structures, which control attention, stress, emotions, learning, memory and motivated behaviour.

Glutamate: N-methyl d-aspartate (NMDA) glutamate receptor is most likely involved in tinnitus, as it is involved in many forms of central neuropathic pain. Specifically, it has been shown that acetylsalicylic acid activates cochlear NMDA receptors and that the use of NMDA-antagonists at the round window abolishes tinnitus, while NMDA receptor agonists may induce tinnitus-like behaviours. Salicylate amplifies cochlear NMD Amediated responses but has little or no effect on αamino-3-hydroxy-5-methyl-4-isoxazolepropionic (AMPA) glutamate receptor and glutamatergic kainite-mediated responses.

Ion Channel and Others: The inner cells of the cochlea have only the L-type of calcium (ca2+) channels that are involved in the regulation of in situ glutamate release and are blocked by salicylate. In the IC, the current through the L-type channels, although unable to directly trigger the neurotransmitter release, contributes to GABAergic transmission by activating the second messenger system and/or by increasing the intracellular Ca2+ connection; while salicylate also blocks the outward and delayed rectifier potassium (K+) channels in rat IC, hence decreasing the GABAergic transmission. Glycine receptor (whose number significantly decreases with age in DCN) along with acetylcholine muscarinic and nicotinic receptors (whose number also decreases with age in the VCN and IC) antagonism has also been investigated for their implication in the genesis of tinnitus.[15]

Evaluation: Laboratory, Otologic, and Neurotologic Testing

Complete audiometric assessment has primary importance for evaluating the patient with tinnitus. Testing should be

- Pure-tone threshold assessment by air and bone conduction
- Speech discrimination, and
- Tympanometry
- Distortion product otoacoustic emission are useful when evaluating patients with tinnitus and a normal audiogram with pure tone thresholds < 25 dBHL. Demographic studies have shown the association b/w tinnitus and systemic diseases (diabetes, hypertension, hypercholesterolemia, and thyroid dysfunction).[15] Laboratories testing for specific metabolic, infectious, or inflammatory markers is indicated only if there are signs and symptoms that rise suspicious for specific primary cause associated with tinnitus.

Treatment: Medical and Surgical Current medical treatments for tinnitus include pharmacologic therapy, aacoustic-based therapy, counselling and education, cognitive behavioural therapy (CBT), and treatments based on complementary and alternative medicine.

Psychopharmacological Treatment

Many drugs, including psychopharmacological agents, are routinely employed in the treatment of tinnitus.

Main Pharmacological Agents for Tinnitus[16]

Drugs	Rationale	Evidence	
Anti-depressants TCAs	NA- and 5HT-ergic transmission modulation, anticholinergic action, BDNF synthesis enhancement	Nortriptyline	Superior to placebo (RTC) (higher doses of TCAs and SSRIs appear work for tinnitus patients who exhibit depression and anxiety or insomnia).
		Amitriptyline	Some success. Low dose sometimes related to tinnitus onset.
		Trimipramine	Less effective than placebo (RTC)
		Clomipramine	High doses associated with tinnitus onset
SSRIs	5HT-ergic transmission modulation, BDNF synthesis enhancement.	Paroxetine	No significant improvement for nondepressed patients (RTC)
		Sertraline	More effective than placebo for severe refractory tinnitus causes (RTC)
		Fluoxetine	Worsening of most of the tinnitus cases
SNRIs	NA- and 5HT-ergic transmission modulation, BDNF synthesis enhancement.	Duloxetine	Withdrawal has been seldom associated with tinnitus
		Venlafaxine	Some success (case report); withdrawal has been seldom associated with tinnitus
NDRIs	NA- and DA-ergic transmission modulation, BDNF synthesis enhancement.	Bupropion	Limited sample reports indicated its potential induction of tinnitus
Heterocyclics		Mianserin Lack of evidence Trazodone Lack of evidence	Lack of evidence
		Trazodone	Lack of evidence
Sedative-Hypnotics Benzodiazepines	GABA-A-ergic transmission modulation (alprazolam, clonazepam, midazolam: short half/average-life and pro 5HT-ergic features).	Alprazolam	Superior to placebo in the treatment of severe disabling tinnitus of predominantly cochlear origin (effective especially for those patients presenting high levels of comorbid anxiety)
		Clonazepam	Superior to placebo (effective also for pulsatile tinnitus when associated with beta-blocker agents)
		Midazolam	Superior to placebo
GABA-B agonists Mood stabilizers.	Muscle relaxing and antispastic action.	Baclofen	Effective for pulsatile tinnitus.

Effective surgical treatments for Tinnitus are those that address a specific underlying disorder. Tinnitus that occurs with middle ear effusion and tympanic membrane perforation resolves with treatment of the conductive hearing loss. Curiously, elimination of tinnitus with stapes surgery is successful in only 50% of patients.[17] Tinnitus related to acoustic neuroma will frequently resolve if hearing is preserved after tumor resection.[18]

Surgery treatment for primary tinnitus is limited. Historically, the cochlea and eighth nerve were thought to be the source of tinnitus and cochlear nerve section for severely disturbing tinnitus was a treatment option. Nearly 50% of patients reports unchanged or worse tinnitus after labyrinthectomy performed for vestibular symptoms.

Transcranial Magnetic Stimulation

Transcranial magnetic stimulation (TMS) is a non-invasive neuromodulation technique that has been adapted from the neuropsychiatric field and applied to the treatment of acute and chronic tinnitus. Pulsed current is passes through a coiled wire held over the cranium. The current generates a strong magnetic field including an electric field which painlessly stimulates the underlying brain area of interest. The affected brain area is either hyperpolarized (excited) or inhibited, depending on the coil size, shape, skull orientation, current pulse characteristics, and stimulation frequency. Lowfrequency (sparked after low-frequency stimulation of the left temporoparietal cortex using repeated (rTMS) reduced auditory hallucinations in schizophrenic patients.[19]

Acoustic Therapy

Environmental sounds have been used to mask tinnitus and decrease subjective awareness of the sensation for millennia. Hippocrates is credited with the observation that an external sound can mask of inhibit tinnitus.[20] Itard recognized the value of matching tinnitus with specific masking sounds to obtain relief and improve disturbed sleep.[21]

Hearing aids are an ideal technology for providing sound therapy when treating tinnitus associated with hearing loss.[22,23] The consistent stimulation may reverse central auditory pathway changes that occurs in response to hearing loss and auditory deprivation. Clinically significant improvement in tinnitus severity has been reported in the large majority of patients fitted with bilateral digital hearing aids.[24] The benefit of hearing aids combined with counselling over counselling alone has been demonstrated in a group of chronic tinnitus sufferers.[25]

Tinnitus retraining therapy (TRT) uses acoustic stimulation combined with directive counselling to facilitate habituation to the tinnitus perception and decrease the emotional reaction to the tinnitus. Acoustic stimulation can be achieved with enrichment of environmental sounds, hearing aids, combination instruments, or most commonly, white noise generators.

A variation of the technique of counselling combined with sound therapy to treat tinnitus uses customized sound therapy to promote habituation, relaxation, and desensitization.

Electrical Stimulation

Non-auditory Electrical Stimulation

Early attempts to improve tinnitus using electrical stimulation of the face, ear, of neck met with mixed results.[26] Herraiz et al. hypothesized that transcutaneous electrical stimulation (TENS) of the periauricular region could augment the inhibition of the dorsal cochlear nucleus through somatosensory inputs.[27]

Auditory Electrical Stimulation (Cochlear Implants)

Cochlear implantation for rehabilitation of bilateral hearing loss with either single-channel or multichannel devices improves tinnitus in the majority of patients, with a small percentage of patients reporting worse tinnitus after implantation.[28]

DISCUSSION

Karna is one of the most important *Gyanendriya* in body. It is important function is hearing. It is also important organ in maintaining balance of body. According to *Acharya Sushruta*, there are 28 *Karna Roga* and *Karna Nada* is one among them. In *Karna Nada* i.e. Tinnitus is a *Vata Dosha Pradhana Vyadhi*. When the *Vata* gets localised in the channels of the ear the patient hears various types of sound like those of a *Bheri* (bulge/drum), *Mrudanga, Shankha* etc. In Ayurvedic literature various treatment modality are applicable in treating *Karna Nada* including administration of *Vatahara Cikitsa*, administration of *Snigdha Ausadha, Snigdha Virecana, Nadisweda, Pinda Sweda, Dhupana* with *Kshouma, Guggulu* and *Agarau, Basti Karma, Navana, Abhyanga, Karnapurana* etc. In modern science different treatment methods like medical treatment, surgical treatment, palliative treatment, use of tinnitus maskers and hearing aid if there is accompanying hearing loss, electrical stimulation, psychotherapy, relaxation therapy etc. are used for tinnitus but with very little success. Thus the Ayurveda procedures and medicines are to have very effective treatment without side effects and

economical drug on it. In modern science various pharmacological drugs are used, but there effect is found to be less.

CONCLUSION

Concluding the study, any complaints related to the ear is to be treated early. Degeneration conditions of auditory nerve and other structures of the ear, disturbs the day today activity of a person. In Ayurveda, Tinnitus, hearing loss and different ear infections managed with following treatment procedures, *Snigdha Ausadha, Snigdha Virecana, Nadi Sweda, Pinda Sweda, Dhupana* with *Kshouma, Guggulu* and *Agarau, Basti Karma, Navana, Abhyanga, Karnapurana* etc., treat disease at the root cause level and also help to correct *Dosha* using diet and lifestyle habits. *Ayurveda* drugs reduce risk of adverse drug reactions. Thus we can conclude that the *Karna Nada* is a condition for which modern science treatment is with very little success, and Ayurvedic approach of treatment in *Karna Nada* is treated to the systemically and alleviates the root cause of the *Dosha*.

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