

TINNITUS - A REVIEW

A independent project submitted in  
partial fulfilment for the

M.Sc, (Speech & Hearing)  
(Previous)

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UNIVERSITY OF MYSORE

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TO

THE

NEEDY

CERTIFICATE

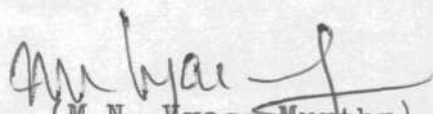
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CERTIFICATE

This is to certify that this independent project has been prepared under my supervision and guidance.

  
(M.N. Vyasa Murthy)  
GUIDE

DECLARATION

This independent project is the result of my own study undertaken under the guidance of Mr. M.N. Vyasa Murthy, Lecturer in Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier at any University or Institution for any other Diploma or Degree.

Mysore

Register No. 12

Dated:

## A C K N O W L E D G E M E N T

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## CHAPTER I

### INTRODUCTION

Tinnitus is a symptom in which the patient complains of perceiving irritant sounds inside the ear. Cryptogenic tinnitus is caused by abnormal excitation of acoustic apparatus or its afferent pathways or cortical areas and their interconnected nuclei; but cannot be recorded in terms of cochlear microphonics. Objective or secondary tinnitus is perceived if some internal head noises are present and it can also be appreciated by examiner.

Sometimes both types of tinnitus may be present.

The subjective tinnitus is not well understood because of lack of non-invasive techniques for objective observations. So is the treatment, and subjects are known to improve more on basis of reassurance than drug therapy.

The noises heard are of varying intensity, character, pitch and may be unilateral or bilateral.

It has been described as hissing, whistling, buzzing sounds and in some severe cases as noise of an engine.

It might be distressing as a consistent nuisance, leading to lack of concentration in work, difficulty in



hearing, disturbance of sleep and may lead to depression enough to attempt suicide.

It is mostly evident in silent environment when there are no distracting ambient sounds, but if it is due to a retrocochlear or central lesion/abnormality, it is not modified by ambient noise.

It may be seen in association with vertigo, and deafness as in Meniere's diseases or with neurological diseases. Its causes are various, for it could be because of wax in the ear canal, eustachian tube catarrh, acute otitis media, mesotympanitis, or any other external/middle ear pathology leading to conductive loss viz. CSOM, Otospongiosis, Otosclerosis, etc.

Tinnitus is an important symptom of auditory dysfunction and may provide the earliest diagnostic cue to conditions which include otosclerosis and 8th nerve tumors.

The patient may be able to localize his tinnitus to one or other ear, but sometimes both ears are affected or the tinnitus appears to arise centrally. The manner of the onset of the tinnitus may relate to the etiology. A gradual onset is uncommon except in otosclerosis. If questioned carefully the patient may be able to relate the onset of the tinnitus to an event such as an infection, trauma or a

cardiovascular or psychological upset. Trauma may be due to excessive noise, an injury or to iatrogenic causes which are most commonly related to ear syringing or impedance audiometry. The tinnitus due to these causes generally settles over the ensuing days or weeks but occasionally the tinnitus may continue unabated for years. Any person who undertakes ear syringing or impedance audiometry should ensure that sudden pressure changes within the external acoustic meatus do not occur.

The characteristics of the tinnitus may indicate the etiology. A pulsatile tinnitus may indicate a glomus tumor but more commonly it is related to some other cause of a conductive hearing loss. The patient hears his own pulse transmitted by bone conduction to the affected ear. A continuous tinnitus is more typical of a sensorineural condition. The pitch is often characteristic of the specific disorder (Douek and Reid 1968). A low pitched tinnitus is typical of Meniere's disorder, a pure tone corresponding to the frequency showing the maximum loss on pure tone audiometry is typical of acoustic trauma and a high pitched hissing noise is common in patients with ischemia affecting the cochlea. The description of the severity of tinnitus is most subjective and varies considerably according to the tolerance of the sufferer. Most patients notice the tinnitus when there is no background noise to mask it. A

description of very severe tinnitus is common in patients with syphilitic disorders and those with a total hearing loss especially when it follows a surgical procedure.

Occasionally some factor may be noticed by the patient which alters his tinnitus. Patients with auditory tube dysfunction may report tinnitus when their noses are congested. Workers subject to excessive noise levels may notice their tinnitus after finishing their work. Rarely a patient is encountered who can alter his tinnitus by twisting his neck into a certain position. More often patients will report that alcohol affects the intensity of their tinnitus.

It is important to detect any associated aural symptoms. The tinnitus of Meniere's disorder is often louder before or during the vertiginous attacks. In Lermoyez's syndrome, a variant of Meniere's disorder, the tinnitus actually decreases during the attacks.

Past inflammatory ear disease and previous otological operations may be relevant to both the etiology of tinnitus and to the interpretation of investigations. Occasionally patients with large mastoid cavities complain of a 'sea-shell' noise within the operated ear and this noise can be altered by filling the mastoid bowl with ointment. Acoustic trauma and ototoxic drugs are important causes of tinnitus

and may act synergistically together or with other factors which cause sensory (cochlear) damage. One third of all patients taking excessive doses of salicylates suffer from tinnitus . The tinnitus caused by salicylates is always reversible on stopping the drug, but the tinnitus caused by most other ototoxic drugs is irreversible. The presence of any allergies should be noted for occasionally a patient is encountered who develops tinnitus after eating certain foods.

The family history can be interesting. It may reveal the possibility of early otosclerosis. A peculiar objective whistling tinnitus can affect families of dachshund dogs and a similar condition has been reported in man (Glanville and Sullivan 1971). The whistling tinnitus in the patients could be heard clearly by an observer and was thought to have been transmitted down the cochlear duct from a valvular arteriovenous anastomosis.

CHAPTER IIINCIDENCE

Fowler (1912) in a series of 2000 patients found tinnitus in "two thirds of all diseases of the ears, and in non-suppurative cases over 75% gave a history of tinnitus. Venters (1955) reported that 16% of 1466 cases of "deafness had associated tinnitus, while only 19 patients who reported tinnitus had no demonstrable deafness. Venters attributed the relatively low incidence in his study to progress in otology and the most common age of onset is 50-60 years (Venters 1953, Reed 1960). There is little question that tinnitus may be present in normal hearing ears (Fowler 1913, Seltzer 1947). Heller and Bergman (1953) reported tinnitus in 94% of the 80 apparently normal hearing adults, when placed in a testing situation having an ambient noise level no greater than 18 dBs. It appeared that tinnitus is present constantly, but is masked by the ambient noise which floods our environment. The ambient noise level is of sufficient intensity to mask physiological tinnitus which remains sub-audible. Graham and Newby (1962) found that 40% of 25 normal hearing subjects experienced tinnitus.

Kawamura (1968) examined 1865 cases of aural disturbances and found (1) tinnitus in 69.8% deaf group and 49.5% of normal hearing group. The frequency of continuous tinnitus

was reverse to that of intermittant tinnitus in deaf and the normal group. In so called chronic carbon anoxia poisoning, there was much more intermittant tinnitus than continuous tinnitus in both the groups. (2) The most frequent sound of tinnitus in both deaf and normal groups was sound 'ji' (53.2%) and sound 'ki' was next frequent. (3) Head noises were recognised in 7.9% of head trauma cases.

The American National Health Survey in 1968 found 36 million American adults (approximately 20% of the population) suffered from tinnitus which was severe in 7.2 million (4%). Almost every one can hear some tinnitus (usually white noise). In addition there is the common experience of transitory tonal tinnitus lasting only a few seconds and of which some people are more aware than others.

Nodar (1972) investigated tinnitus in school children in New York over 3 years and found an overall incidence of 15% which rose to 58% in the group which failed auditory screening. The ages between 13-15 years reported more tinnitus than younger or older children.

The fact that tinnitus is often completely absent in sensori-neural deafness, indicates that the auditory experience seems to be a pre-requisite. It is more surprising than finding sufferers amongst the normal hearing population in mens a recent random survey, 56% among deaf patients tinnitus was noted (Kimura, 1974).

### CHAPTER III

#### TYPES OF TINNITUS

Tinnitus aurium is the sensation of sound when there is no relevant external auditory stimulus present. Subjective tinnitus is audible only to the sufferer and usually indicates abnormality in the auditory system. Objective tinnitus is an interesting rarity, by comparison it can be heard by others and is often due to pathology outside the ear.

Dench in the latter part of 19th century felt that tinnitus often resulted from constipation, subacute gastritis or a pathological conditions in the pelvis by reflex action mediated through the vagus nerve.

1) Objective tinnitus is relatively a rare condition and represents only a small proportion of cases suffering from noise in the ears. Etiologically objective tinnitus is divided into two distinctive groups. First group where the tinnitus is caused by clonus of the palatal and pharyngeal muscles. Several different cases of myoclonus of palate have been described. Second group (minority group) is where the noise is due to vascular pathology (Engstrom, Graf 1950, 1952, Bonnal and Legri 1960, Wengray 1967). Graf (1952) enumerated several criteria for labelling objective tinnitus as essential. These were—

- 1) Sudden onset unrelated to other disease or injury
- 2) Persistence unchanged for more than a year
- 3) Definite objective tinnitus
- 4) Pronounced lateralization
- 5) Synchrony with the pulse
- 6) Certain relations to alterations in head position
- 7) No symptoms of raised intracranial pressure
- 8) No pulsating exophthalmos
- 9) No abnormalities of extra cranial arteries
- 10) A normal central angiogram

Atkinson (1947) listed the causes of objective tinnitus as follows:

- 1) Muscular spasms
- 2) Vascular bruits
- 3) Nasopharyngeal sounds
- 4) Clicking jaws.

The first report of clicking tinnitus due to palatal myoclonus came with the work of Politzer (1878), myoclonic clicks generally described as a myoclonus due to vascular spasm (Pulec and Simonton 1961, Mocckimmon 1969). This clonus is usually of the palatal, Salpingo pharyngeal and pharyngeal muscles. The cause of palatal myoclonus has been attributed to various lesions, among them vascular pathology, multiple sclerosis, aneurysm of verbal artery; different tumors involving the brain stem or cerebellum



(Bjark 1954; Parker 1956; Pulec and Simonton 1961). The interruption of the connection to the dentate nucleus, red nucleus and inferior olivary nucleus has been considered to be the pathological basis of palatal myoclonus by Swanson et al 1962. It is suggested that palatal myoclonus may represent another type of extrapyramidal disorder arising from disturbed neural mechanism in the brain stem including cerebellum (Hermannic, Crandall H.P., Harry 1957). On the other hand non-organic disturbances such as emotional conflicts, neurasthenia and neurosis have also been incriminated (Bjor 1954, Pulec and Simonton 1961).

Palatal myoclonus has been attributed in association with different neurological symptoms, such as hemiplegia, myasthenia, facial paresis in coordination of movements, diplopia, etc. (Northanson 1956).

The cause of sensory epilepsy as a possible mechanism in the production of tinnitus has been suggested by Core 1976. Further evidence of neuronal hyper activity occurring within the path auditory pathway has been provided by Gates and Chen 1975; Melding P.S., Goodey R.J. 1979), or it may be produced by central cysticerosis with nystagmus and hearing disturbances. (Jankinman, et al 1976). Palatal myoclonus was attributed to psychogenic disorder when it appeared as solitary symptom (Nodar 1972).

Hentzer (1968) further subdivided vascular bruits into type A in which angiography demonstrated an arterial anomaly and type B 'essential objective tinnitus' in which it has not been possible to demonstrate any vascular abnormality. He observed that essential tinnitus is usually synchronous with the pulse of the pulsation of the intracranial venous system.

Pulsatile tinnitus due to vascular abnormalities are sometimes audible (Frank and Horak 1976). A glomus jugularae tumor (House and Glasscock 1968) Glomus jugalae bulb abnormality (Hentzer 1968), Aneurisms or arterio venous fistulae (Tewfik 1974) may cause tinnitus. The occipital artery (Arenberg and McCreany 1971), superficial temporal artery (Zokrigewski, Kruk-zagajewskka and Konopaski 1971) and aortic arch (Schechier and Brownson 1970) have been implicated.

2) High tone objective tinnitus Three cases of a father and his two sons of high frequency non-pulsatile objective tinnitus emitted at different pure tones were examined. The condition is believed to be inherited by a dominant characteristic that is most sex linked. The condition is believed to be harmless and due to vascular abnormalities at the base of the skull (Glanville J.D. Coles R.R.A., Brenda 1971).

In 1965, Pulue and Simonton demonstrated that the ticking was due to either a snapping shut or a snapping open of the enstachian tube. Myrharay 1964 also reported the use of an impedance device to study a patient with permanent objective tinnitus. He interpreted the impedance changes are due to spontaneous activity of the tumor of tympanic muscle.

Occasionally infestation of the external meatus may present an objective tinnitus such as Earwigs, Cockroaches (Snashall and Stephens 1975).

3) Subjective tinnitus is extremely common. The most common type of subjective tinnitus are like pure tones or kinds of white noise. However, it is quite common for multiple sound to be heard in different parts of the head. They can be complex bizzare defying description.

Etiology of idiopathic subjective tinnitus:- Different sites of pathology has been suggested by different authors. Cochlea (Fowler 1939), Neuronal resonater (Keurp and Martar 1976), small areasof demyelination in the acoustic pathway or non functioning nerve fibres causing an absence of spontaneous discharge.

CHAPTER IVETIOLOGY OF TINNITUS IN GENERAL

It can be classified as under:-

I. Based on Anatomic locations:

A. External auditory canal.

B. Tympanum

- 1) Membranous tympanum
- 2) Tympanum muscles
- 3) Ossicles
- 4) Tympanum plexus
- 5) Tympanum vessels
- 6) Eustachian tube
- 7) Round and oval windows

C. Cochlea

- 1) Perilymph
- 2) Endolymph
- 3) Organ of Corti

D. VIII nerve

- 1) Spinal ganglia
- 2) Trunk

E. Intracerebral

- 1) Ventral and dorsal cochlear nuclei
- 2) 2nd order neurons
- 3) 3rd order neurons
- 4) Cortex.

II. Based on pathological lesions: Various factors have been known to produce tinnitus or have been found in association. They are:-

- |                          |                         |
|--------------------------|-------------------------|
| 1. Anemia                | 2. Hyperemia            |
| 5. Edema                 | 4. Serocous exudate     |
| 5. Purulent exudate      | 6. Haemorrhage          |
| 7. Inflammation          | 8. Allergy              |
| 9.            Neuronitis | 10. Necrosis            |
| 11. Fibrosis             | 12. Tumor               |
| 13. Aneurysm             | 14. Capillary fragility |
| 15.            Stasis    | 16. Sludging            |
| 17. Vascular spasm       | 18. Vascular Sclerosis  |
| 19. Vasomotor paralysis  |                         |

(Jerome G. Alpiner 1968)

20. Middle ear and external ear conduction defects due to pathology, such as wax in ear, serous otitis media, otospongiosis, Eustachian tube atarrh.
21. Muscular murmors due to fistula's, aneurysms, etc.
22. Hemifacial spasms leading to stapedial spasms.
23. Myoclonus of tensor tympani muscle (Pulee, J.L. et al. June 1961)
24. Ischemia of the inner ear (Fowler E.P. and Fowler E.P. 1961) Jr. 1950.
25. Smudging of the small vessels in inner ear. Fowler E.P. and Fowler E.P. Jr. 1950.

26. Associated with Memiere's Syndrome, Ramsay Hunt.
27. Emotional Psychic factors (Fowler E.P. Jr. 1955)
28. Due to round trauma, ultrasonics (Moller P. et al  
May 1976)
29. Drugs Toxicity Streptomycin, (Cawthorne and Ranger, 1957)  
Gentamycin (Drake, T.E. Oct. 1974) etc.
30. Due to cerebral injury (Hackner Jr. 1976), Meningitis, etc.
31. Temporal lobe tumours (Frazier and Rowe, 1932)
32. Severe liver disturbances, duodenal ulcer, ulceratine  
Colitis (Fowler E.P. and Fowler E.P. Jr. 1950).
33. Decayed teeth.
34. Diseased tonsil
35. Presence of enlarged turbinate in the nose.
36. Faulty saw formulation
37. Head injury
38. Excessive use of tobacco
39. Digestive disturbance
40. Meningites
41. Glandular disturbances.

CHAPTER V  
CLASSIFICATION OF TINNITUS

Tinnitus aurium may be defined as noises in the ear whereas Tinnitus cerebri refers to head noises originating in the cerebral cortex.

Tinnitus is of two types, cryptogenic and vibratory. The cryptogenic variety is not well understood because of the relative inaccessibility of the sites of origin for neurophysiologic studies. It is not recordable in terms of microphonics and animals are known to have it. Hence Tinnitologic conclusions are to a large extent based on circumstantial evidence of a subjective nature.

Fowler (1939) classified tinnitus as vibratory and non-vibratory. He defined vibratory tinnitus as a result of actual vibrations arising from any source in the body and transmitted to the ear by the solid or fluid media of the body. Fowler stated that non-vibratory tinnitus was free of actual vibrations in the cochlea and suggested biochemical stimulation as a possible casual factor. In some cases an objective vibratory tinnitus may be heard by an individual other than the patient. Subjective tinnitus which can not be heard by an observer may or may not be vibratory (Nodar and Covering 1971).

According to Riddel (1956) tinnitus aurium may be defined as follows:

1. Objective:- A cortical perception of auditory sensations potentially audible to patient and examiner. It is vascular or muscular in origin. Vascular types are due to arteriovenous anomalies, causing a bruit which is audible with or without amplification. The muscular types are due to contractions of tympanic or tubal muscles in a typical rhythm. The mechanisms involved remain obscure but ties, metabolic and psychosomatic states may be contributory.

2. Subjective:- A subjective cortical perception of auditory sensations inaudible to anyone but the patient. It is due most commonly to auditory paresthesia arising from any portion of the auditory pathway.

P. Ghosh (1978) presented a comprehensive classification as shown in the table.

- I. Non vibratory (static, subjective, cryptogenic)
  - 1. Tinnitus aurium
    - a) Peripheral
    - b) Non-peripheral
  - 2. Tinnitus cranii
    - a) Central
    - b) Bilateral



## II. Vibratory (dynamic)

- a) Objective - subjective
- b) Objective

## III. Essential (unclassified)

1. Non-vibratory tinnitus (static, subjective or cryptogenic). The patient hears it, but the observer does not. It is due to auditory paresthesia (Goodhill, 1952). This is further sub-classified into tinnitus aurium and tinnitus cranii. Tinnitus aurium is further classified as either peripheral (the lesion is located anywhere from external ear to the osseous spiral lamina), which is maskable or non-peripheral (the lesion is located from the lamina to the cochlear nuclei), which is non-maskable, poorly maskable or maskable only with bone conducted sound. Tinnitus cranii is further classified as central (the lesion is beyond the nuclei upto the auditory cortex), which is non-maskable, or bilateral (identical to tinnitus aurium).

2. Vibratory tinnitus (dynamic). This is sub classified by its audibility. Objective-subjective vibratory tinnitus is heard by both the patient and the observer, with or without amplification. Objective vibratory tinnitus is heard only by the observer. Both forms may be due to intrinsic (intra-tympanic) or extrinsic (extra

tympanic) causes - (1) Vascular, due to some arteriovenous communication (intracranially such a lesion produces tinnitus cranii) and (2) muscular, due to contraction of tympanic, tubal, or other head and neck muscles. These are maskable ones.

3. Essential tinnitus (unclassified). Examples of this are "central memory tinnitus", analogous to "phantom limb syndrome", "mirror-image tinnitus"; and "occasional tinnitus". The second and third types may be tinnitus aurium or cranii, depending on the site of lesion and the psychophysiologic implications of the auditory system. P. Ghosh coined the phrase "occasional tinnitus" to indicate sudden, transient, unwarranted tinnitus without consequences. It may be present with a normal end organ, as is the case in itching.

Etiologic classification:- Tinnitus can also be classified on the basis of etiology as follows:-

1. Suprathreshold auditory paresthesia, as in peripheral neuritis.
2. Vasospasm, as in Meniere's disease, Lermoyez's syndrome, etc.
3. Irritation of the tympanic plexus in middle ear disease.
4. Hypersensitivity of chorda in the internal sonic system.

5. Intracellular edema in cort's cells, bringing them into steady contact with tectorial membrane.
6. Physical distortion of the cochlear sensory system.
7. Unmasked visceral tinnitus of Goodhill due to an impediment in sound conduction.
8. Vascular middle-ear lesion - eg. glomus tumor, oto-sclerosis, acute inflammation.
9. Clonic contraction of tympanic, pharyngeal and neck muscles.
10. Neural lesion - eg. acoustic neurinoma, multiple-sclerosis.
11. Toxic reactions - streptomycin, neomycin, kanamycin, gentamycin, quinine, salicylates, etc.
12. Cardio-vascular and hematologic conditions, including subclavian steal syndrome.
13. Metabolic and endocrine dysfunction - eg. hypo or hyperthyroidism, diabetes mellitus, premenstrum, pregnancy, defective or absent proteolysis, reactive hypoglycemia.
14. Trauma, including noise trauma.
15. Emotional state leading to blood sludging.
16. Miscellaneous effects of increased tension of intratympanic muscles on endo and perilymph, "occasional tinnitus", malocclusion syndrome, etc.

## CHAPTER VI

### PSYCHOLOGICAL IMPLICATIONS OF TINNITUS

Tinnitus means more to the patient than just a noise. In fact, many otologists have declared that when a patient comes for an interview, they do so because the tinnitus has proved too bothersome in their everyday life. Tinnitus is a manifestation of some disorder and the psychological implications presented may be manifestations of the tinnitus.

Venters (1949) stated that tinnitus rarely causes psychological difficulties for the patient, further the hearing impairment colored a patient's mental attitude toward tinnitus. Patients report because of the anxiety regarding tinnitus, some cases even do not realize that they have a hearing problem. Kennedy (1953) classified his cases with tinnitus at Durham University into 5 groups.

1. Depressive preoccupation with tinnitus - The patient is quite anxious and concerned and makes statements as "these noises are spoiling my life and driving me mad". In extreme cases patients have even mentioned suicide.

2. Tinnitus as a symptom of psycho neurotic escape - This attitude was manifested by those whose occupations carried the hazard of deafness. This was especially true before they became accustomed to the noise.

3. Anxiety states - There was much anxiety because of unsolved personal problems and frustration of ambition. It may be psychosomatic in showing itself in cardiac or gastric neurosis.

4. Presenile, senile and arteriopathie deterioration - There was distress in the early stages but decreased as deterioration advanced.

5. Malignant hypochondriasis - This is a deterioration of personality. The individual felt he never had the right to treatment and surgery which will make the whole difference in his life.

Goodhill (1950) stated that there was no correlation between actual intensity of tinnitus as measured audiologically and the amount of 'suffering' of the patient. He stressed, therefore, that it was important to assess the excitability level of the patient.

There are possibly two major factors to consider in this psychological area, (1) The premorbid personality of the individual gains strength with tinnitus allowing this personality to show itself. The study mentioned presented those groups of individuals who were concerned with other problems and anxieties other than tinnitus but that the noises gave them a sort of escape mechanism to bring these problems to the foreground.

(2) Here tinnitus has actually disturbed the individual and is the primary concern of the patient. In such cases supportive therapy should lessen his tension. However, with a patient suffering real stresses and strains, psychotherapy is warranted.



Vasomotor disturbance : hypertension \_\_\_\_\_  
 hypotension \_\_\_\_\_

Neurocirculatory asthenia \_\_\_\_\_

Otologic appraisal : Otoscopy\_\_\_\_\_Nasopharyngoscopy  
 Audiometry\_\_\_\_\_Tuning forks \_\_\_\_\_  
 Measurement of tinnitus \_\_\_\_\_  
 Vestibular tests : Spontaneous \_\_\_\_\_  
 Caloric\_\_\_\_\_Rotation\_\_\_\_\_

Psychological appraisal : Psychotic \_\_\_\_\_  
 Psychological overlay \_\_\_\_\_

John Ballantyne and John Groves (1977) also presented a brief outline for history of tinnitus. They claim that it is necessary, as it may have an aetiological significance. Some patients fear that they have developed a brain tumor and others fear that the tinnitus will drive them to insanity. Reassuring the patient is important, keeping in view the aetiological factors.

#### Tinnitus history - a brief outline

Localization of Tinnitus	- Right ear/Left ear/Both ears/ inside head
History of present complaint	- Duration onset - sudden or graded - any events related to Progression - Getting better/ same/worst Fluctuation
Characteristics of the tinnitus	- Subjective description Continuous, pulsatile, clicking, etc. hissing noise, pure tone, etc. Pitch of tinnitus Severity - aggravating factors alcohol, exercise, neck twisting etc. - Relieving factors drugs, yawning, etc.



- Associated aural symptoms - hearing disorder  
vertigo
- Associated general symptoms - especially headache,  
visual disturbances
- Past history - Otological - Past inflammatory disorders  
Previous otological operations, trauma  
excessive exposure to noise  
Ototoxic antibiotics and  
other drugs or agents
- General - metabolic disturbances  
eg. thyroid disorders  
Cardiovascular disease  
and hypertension allergies
- Family History - hearing loss (especially  
otosclerosis)  
Tinnitus
- Psychiatric history-
- Social history - Occupation  
alcohol and tobacco.

## CHAPTER VIII

MEASUREMENT OF TINNITUS

Assessment of the severity of tinnitus or its response to treatment is normally left to the patients subjective report. This is notoriously unreliable and relates as much to the individual's personality and mood as to the actual loudness of tinnitus. The major efforts of the investigators in this direction are many. They are:

1. Masking by pure tones - Wegel (1931)
2. Measurement by loudness balance - Fowler (1939)
3. Freefield matching of tinnitus - Mortuner (1940)
4. Masking in central and peripheral tinnitus - Fowler (1944)
5. Identification using taped sound effects - Goodhill (1952)
6. Audiometric study of 200 cases - Reed (1960)
7. Diagnostic value of tinnitus pitch - Donek and Reid (1968)
8. Classification of tinnitus by masking - Fieldman behaviour (1971)

The acoustical characteristics have been studied by Reed (1960), Graham and Newby (1952) and Nodar and Graham (1965). They used sweep frequency audiometers and reported a higher pitch of tinnitus in patients with sensorineural deafness than in those with conductive deafness, except for a few cases in Meniere's disease. Donek and Reid (1968) in

their study of 'The diagnostic value of tinnitus pitch' concluded that (1) the pitch of tinnitus has been shown to be of some diagnostic value. (2) adequate pitch matching can be achieved with an ordinary clinical audiometer and (3) an audiometric artifact due to tinnitus was also noted.

Fieldman's technique (1971) uses the narrow band masking of a clinical audiometer. Slowly increasing levels of noise are presented through headphones until the tinnitus is no longer audible. The point of masking the tinnitus is measured for different frequencies and a masking audiogram can be plotted.

Hazell (1977) used a commercially available music synthesizer which is theoretically capable of infinite variation and which often can produce an exact match for subjective tinnitus. Apart from being able to match cases of multiple and bizarre tinnitus pulsatile signals can be presented for the correct identification of haemodynamic abnormalities. Although it may take a considerable time to achieve a good 'match' with this technique, it is clear that many forms of tinnitus simply described as pure tone are in fact more complex.

Attempts to measure the loudness of tinnitus by a loudness balance technique often appear to show severe tinnitus as relatively quiet. Fowler used this fact to reassure

his patients that they were worrying ever nothing. However, this technique does not take into account the difficulties of comparing the loudness of a pure tone with a complex sound, or the reduced dynamic range that may be present in an ear with recruitment . If only ordinary acoustic equipment is available Feldman's tests are probably the most useful and repeatable.

In tonal tinnitus a 'dip' may be seen on the pure tone audiogram at the frequency of the tinnitus (Sedlacek 1948, Donek and Reid, 1968). Whether this is due to 'masking' of the threshold as part of the pathology is not clear. Some patients have no change in their speech discrimination, despite loud tinnitus. Others complain bitterly that they can hear nothing because of it.

Sayed Tewfik (1974) used phonocephalography. Phonocephalography is a new term introduced for the systematic auscultation, amplification and recording of sounds from both the surface of the head and its cavities, such as the nose, external auditory meatus and the nasoprimarynx.

Technique - Auscultation of the surface of the head was done by using the 'Lithman Stethoscope'. The nasopharynx was auscultated by using a modified stethoscopic end in the form of a polyethylene tube attached to a nasopharyngoscope. Amplification and recording of the sound is done by the

'Helige Phonocardiographic apparatus'. The microphone was applied to the area to be tested. The sound was amplified, heard by the apparatus stethoscope and recorded by its recording system. The recorded sound tracing was called 'Phonocephalogram' (PCG). The amplified sound can be fed back to the patient to see whether it is the sound subjectively heard by him. Phonocephalography is useful in localizing, recording and aiding in the diagnosis of the lesion.

Killer, A.P., (1974) used oscillographic study for an young girl with objective tinnitus (audible click). The sound was recorded, analyzed by oscilloscopic methods and found to be of a ringing pattern with a pulse lasting about 4 milliseconds. The results showed that

1. The ratio of signal to noise level is approximately 5:1
2. The duration of single pulse to be about 4 m. second.
3. The sound pulse has a ringing patten and confirms the duration of about 4 m.seconds.
4. The sound varied in intervals and on tape ranged from about 1-2 pulses/second.
5. The small spikes represent the noise level.

Pulse volume recordings in outer ear canal in pulse synchronous tinnitus. L. Andreasson et al (1978) with the aid of a volume flowmeter recorded pulse synchronous volumetric

changes in the outer ear canal. In 7 ears with glomus tumor in the tympanic cavity and in 5 with serous otitis media, such changes were larger than in 125 persons with a normal middle ear. By changing the ambient pressure in a pressure chamber and instructing the patients not to swallow, the drum can be pushed inward or outward. In all the cases of glomus tumour studied, the pulse volumetric change was considerably affected, when the drum was pushed inward or outward.

Tinnitography!- Tinnitogram and differential tinnitogram help in the study of tinnitus (Ghosh, P., 1978). Both depend on the maskability of tinnitus. Maskability and additional phenomenon of attempted masking (due to synchronization of tinnitus and masking sound) indicate peripheral lesions, since in lesions more cranially situated acoustic perceptive elements are not available for masking or additive effects.

Maskability of tinnitus with air-conducted sound in the absence of temporary threshold shift and recruitment gives a fair measure of loudness of tinnitus. In the presence of temporary threshold shift and recruitment, the available varieties of masking character yield the phenomena that are the basis of the tinnitogram and differential tinnitogram, graphical representations of tinnitus. Partial maskability of tinnitus with bone conducted sound indicates a non-peripheral lesion very near to the cochlea (ventro-

coehlear). Non-maskable tinnitus with AC sound having tonal quality subjectively or objectively suggests the presence of a lesion in the non-peripheral sigment (immediate retro-cochlear). The tinnitus even when monotone, can be masked with multiple tones (AC), and the values can be plotted on an audiograph, yielding a tinnitogram.

In the absence of recruitment or temporary threshold shift , the masking tone need not be much higher than the loudness of tinnitus (which is usually 5-10 dB). Several factors may play important roles (1) busy-wire effect (2) a biochemical fatiguing effect causing sensorineural damage with or without retrograde degeneration and (3) generation of the recruitment phenomena. Hence the tinnitogram at times is much higher than the AC threshold in comparing the loudness of tinnitus. An inordinate discrepancy between the AC threshold and the tinnitogram is suggestive of the presence of a temporary threshold shift and/or incomplete recruitment.

Differential tinnitogram - is based on the differential auditory perception of tinnitus between the stages of initial and complete masking. The masking tone is presented in a gradually increasing loudness, and the intensity at which the tinnitus is just masked is noted, as is that at which it is completely masked or almost completely masked. Similarly the values are obtained at

different frequencies are plotted on an audiograph. The difference between the two is calculated which represents the differential tinnitogram. If this is less than the loudness of tinnitus, it is indicative of presence of recruitment, if more, it is suggestive of temporary threshold shift.



## CHAPTER IX

### TREATMENT

Tinnitus in the late 1970's is in the position of fever a century ago. It is something which can be quantified and there is a plethora of treatments advocated for this entity. It is not essential to consider the entity in itself but rather the underlying cause. While the search of treatment is symptom oriented rather than disease oriented little can be expected in the way of significant advances. Determination of the underlying cause, therefore, has inevitably to be indirect methods and by extrapolation. An extensive multi-disciplinary approach to the problem is essential, if there is to be any further understanding of the basic disorder underlying the symptom. This is a pre-requisite if any meaningful treatment is to be achieved. Till now little consideration has been given to the possibility that a particular treatment may be valid for tinnitus of a certain aetiology and another for that caused by a different lesion.

The current approach to the treatment of tinnitus has two orientations, the abolition or reduction of the symptom from an absolute point of view and secondly, the alleviation of the psychological effects arising from it.

Goodhill (1954) said that the treatment of tinnitus as a disease is an illogical dream, but the management of the patient with tinnitus is an everyday necessity. Many efforts have been made over the centuries to cure tinnitus.

The various kinds of treatment attempted are:-

1. Medical treatment:- Various drugs attempted are -
  1. Antidepressants (Yosuda, Nishida & Keda 1971)
  2. Antihistamines
  3. Amylobarbitone (Donaldson I.J. 1978)  
Bitahistine
  4. Carbamazepine (Shea J.J. et al 1978)
  5. Cocaine (topical) (Crishani and Lovino 1948)
  6. Cortisone (topical) (Schroer 1955)
  7. Curare (Atkinson M. 1946)
  8. Dimethyl sulphoxide (Cazo 1975)
  9. Diphenyl hydantoin (Melding P.S. et al 1979)
  10. Ergor (Tanner 1955)
  11. Heparin (Breu 1956)
  12. Lidocaine (Gejrot T. et al 1963)
  13. Hydergion (Tanner K. 1955)
  14. Lignocaine (intravenous) (Lewy 1937, Englesson  
Larsson and Dindquist 1976, Rahm et al 1962)
  15. Metoprolol (Camara 1957)
  16. Nicotinic acid (Wilens 1975; Flottorp 1955)
  17. Nucleic acid (Makishima, Yosuda and Miyahara 1971)
  18. Procaine (Fowler E P. Jr. 1953)

19. Prostigmine (Judge 1942)
20. Thiamine (Shanbaugh 1942)
21. Traiqidlizers (Seltzer 1947)
22. Vitamin A (Anderson 1950; Lobel 1951)

Barany (1933) described relief from tinnitus with intravenou-s procaine as did. Fowler (1953) when treating Menier's disease I.V. Lignomine has been found to relieve severe tinnitus in some patient (Engelson et al,1976) and to relieve tinnitus associated with Minier's disease in some patients (Gejrot 1963, 1976).

I.V. Lignocaine and its usage was a diagnostic tool was emphasized (Melding P.S. et al 1978). As the effect of I.V. Lignocaine known to be are (Grollman and Grollman 1971)

1. Primarily used as a local anaesthetic
2. Anti arrythmic
3. Anti convulsant (Bamhand and Bohn 1954)
4. Vasodilator effect
5. Sedativ-e and Traquilizers
6. Anti allergic (mild)
7. Anti cholinergi (Wielding 1959)

Its convulsant effect was emphasized by Melding P.S. et al as the primary action in treatment of tinnitus and was supported by work of Shea J.J. et al 1978. Sakatal et al 1976.

No double blind studies were conducted by them for the response to these treatment and role of psychological factors and reassurance was not ruled out.

Martin F.W. and Colman B.H. (1980) did a double blind cross ones trial to evaluate the use of Lignowine. By their study it was shown that intravenous lignowine is effective in reducing tinnitus although the duration of such effect is short. Their findings indicated that psychological factors play a less significant role in tinnitus than previously thought or that lignowine is more effective on peripheral lesion.

Melding P.S., Goodey R.J. and Thome P.R. (1978) studied 78 cases. The results of these study indicated that lignocaine is strikingly effective in suppressing tinnitus in some patients. The irresponsible groups of patients include those in whom the hearing loss is predominantly conductive . Those will skislope S.N. hearing loss which Schuknclcht (1964) suggested may be due to softening of the basillar membrane and those with cervical dysfunction.

Carbamazepine (Goodman and Gillman 1977) is known to have the same following effects.

1. Anti convulsant
2. Neurologic analgesia
3. Sedative

3 cases with objective tinnitus caused by palatal myoclonus were treated effectively with carbamazepine by Rahko and Hakkinen V. (1979).

Carbamazepine was reported to have cured tinnitus owing to its anticonvulsant properties and it was again a subjective assessment (Melding P.S. et al 1979).

Bitahistine may have some effect in reducing the tinnitus in Meniere's disorder. No consistent relief of tinnitus in general has been reported in properly controlled studies (Still P.M. 1980).

Amylobarbitone may be of help in treating tinnitus of varied causes (Donalson J. 1978).

But the results achieved from treatments of drugs has been inconsistent and disappointing.

## II Surgical treatment.

1. Eighth nerve section (Antoli candela, Alvares de cozar and Anotoli Candden - 1975); Malnros Elbrand and Anderson 1966)
2. Carotid ligation
3. Cochorda tympani section - Roser 1952
4. Labyrinthectomy (Pedersen & Soreneer 1970; Pulic 1974)
5. Ligation of Internal jugular Vein
6. Pre-frontal leucotomy - Elithorn and Beck (1955)  
Beard (1965)

7. Stellate Ganglion block - Adlington (1971)

8. Tympano Sympathectomy (Lempert 1946; Portman 1948)

66 patients with tinnitus were treated by stellate ganglion block. 26 being Meniere's disease and 40 of unknown etiology or due to certain otologic conditions. A marked reduction in the loudness of tinnitus was obtained in 56% of Meniere's disease and a significant reduction in 27% of other cases. It is suggested that the tissue displacement technique is very safe and high proposition of successful blocks may be expected.

Temporary stellate ganglion block with novacine applied to 18 tinnitus patients resulted in improvement in 56%, no change in 53% and worst condition in 11% (Vijay A. Shah and Bindu).

For the treatment of objective tinnitus ligation of internal jugular vein has been suggested (Ward H.P., Babin R. Thomas 1975).

R. Gibson (1973) reported a case of Pageti, disease with pulsating tinnitus. The external carotid artery was ligated with subsequent improvement for 4 months after which tinnitus recurred.

Acupuncture is of little use for tinnitus (Felix Mann 1974).

Tympanosympathectomy gave relief in more than half patients in some studies. Even the section of the entire 8th nerve gave relief in about 33% cases. The sections of vestibular portion gave relief to only 27% of patients. Prefrontal leucotomy resulted in good improvement in half the number of patients.

Labyrinthectomy or acoustic nerve section probably results in relief or improvement in tinnitus in about 50% of cases. Unfortunately some cases are made worse, and others are quite unchanged, showing how little is known about the aetiology of this condition. Useful hearing must not be destroyed. In the really suicidal tinnitus patient it is often justified to take such extreme measures. As a diagnostic procedure local anaesthesia may be introduced into the middle ear to produce temporary anaesthesia of the cochlea (Mitani and Tanaka 1975). This technique may be used with iontophoresis for added control. If tinnitus is not abolished despite temporary vestibular and facial paresis, destructive surgery is unlikely to be helpful.

The most drastic form of treatment entails surgical intervention which has in the past range from cochlear nerve section to prefrontal leucotomy. The seriousness of these procedures reflects the dire effects which tinnitus might have on sufferers and indeed they should be regarded only as last resort procedures. Even with such interventions complete tinnitus relief cannot be generated.

This leads us to the psychological treatment of tinnitus.

Psychotherapy: Information and reassurance remains the mainstay of management in idiopathic subjective tinnitus. Proper reassurance takes time and needs to be supported by proper examination. Patients must be encouraged to avoid listening intently for small changes in pitch and intensity, but to involve themselves as much as possible in extrovert actively. The great majority of patients will within a year learn to accept tinnitus as part of their 'environment' and will not seek further advice.

When anxiety or depression co-exist those should be treated, but it should be made clear than any therapeutic agents prescribed will not affect the level of tinnitus itself.

What is needed is our frank explanation of our present state of knowledge of this condition. Many patients have difficulty getting to sleep and mild hypnotic in this situation is very valuable.

Biofeed back training: It was originally attempted because of the apparent relationship of severe tinnitus with emotional or physical stress. Group of patients who are extremely aggravated by their tinnitus and have found that it causes difficulty with their everyday activities, they respond well to bio-feed back training (House J.W. 1978).



The bio-feed back training consists of approximately 12 one hour sessions. Patients are monitored by electromyograph and skin temperature machines which constantly inform them of their progress and thus teach them to understand the effects their mind can have on their muscle tension and vasospasm. Both during the therapy and at home the patients were given specific relaxation exercises to do.

Bio-feed back is not a new modality of treatment. For approximately 20 years, bio-feed back has been used for pains and stress related physical problems. Basically bio-feed back is a method of teaching one to learn exert conscious control over areas of the body which were thought previously to be outside of the control of the conscious mind. Instruments are used, which will sense and amplify impulses coming from parts of the body, which one wishes to alter, which the individual can modify and alter the specific physiologic processes.

When bio-feed back is coupled with various relaxation exercises, the patient is able to learn to discriminate and alter physiologic functions. The patient eventually develops a relaxed conscious state in which, he learns to recognize uses for altering his body processes. Inherent in this treatment are the accepted principles that the mind can reduce the perception of the physical experience and that establishing conscious control over physiologic states provide the patient with the ability to alter the function of that process.

For patients with tinnitus feed back of information from the frontalis muscle and skin temperature of the fingers are used. The patients are taught to relax the frontalis muscle and at the same time to increase circulation through the peripheral vascular system. Results with the patients show that in addition to improvement in tinnitus, they reported an overall improvement in their general sense of well being and enjoyment of life (House J.W. 1978).

#### Electrotherapy:

Suppression of tinnitus by electrical stimulation has been known for over a century. Politzer in the 1800s described the use of galvanic currents. Hulton also found that galvanic stimulation attend the tinnitus. William F. House and J.W. House also used electrical stimulation in an attempt to alter tinnitus. But suppression of symptom occurs during passage of current (usually PC) (Graham and Hazell 1977). It is possible that for some cases continuous stimulation with an implanted electrode, on the round window, may provide an answer.

M. Fortmann et al (1979), while investigating the possibilities of electrically induced hearing in cases of profound deafness, observed that very often an appropriate stimulation of the cochlea could result in tinnitus cancellation, but as long as the cochlea was stimulated. It was hypothesized that this phenomenon acts through an inhibition

of abnormal activity of sensorineural elements of the electrically stimulated cochlea.

It is interesting that House (1976) reporting on 13 patients with cochlear implantation showed that 11 had marked improvement of their tinnitus. In five of these the improvement occurred only after one month of electrical stimulation.

K. Terkildsen documented that corneal reflex stimulation with either tactile stimulation or with a fold air jet will elicit a tensor tympani reflex. This also travels through the mandibular division with its direct branch to the tensor tympani muscle. Quarry J.G. (1972), with the probe from the impedance bridge, still in the left ear, a cool air jet from an air hose was directed on the cornea of the left eye. At that moment, the ticking noise stopped immediately and permanently.

Sataloff has reported that breath holding will stop the tinnitus temporarily.

Residual inhibition J. Veron and Alexander Schleuning (1978) found that the removal of a masking sound did not immediately reinstate the tinnitus. Instead there was usually a brief period of quiet followed by a period of gradual recovery of the tinnitus. Josephson was the first to discover the temporary disappearance of tinnitus after a period of mask-

ing. Feldman considered masking to be a form of inhibition, hence residual inhibition was coined to describe the silent period and the return of tinnitus. The authors state that residual inhibition may indicate how well a patient will respond to relief procedures.

Tinnitus masking entails the provision of an alternative noise source to either distract the patient from his tinnitus or to physically mask it. Various noise sources have been used in this respect for many years (P.M. Stell, 1980). Often patients discover for themselves that radios, running water, etc. will mask their tinnitus and find these controllable noise sources preferable to their own relentless tinnitus.

Ear level masking devices have been available in U.S.A. (Veron 1977) and a high degree of sophistication has been reached in adjusting the masking noise to the user's requirement. Very high frequency tonal tinnitus is very difficult to mask at all. In U.K. a similar but simpler masking device is available. Early results are encouraging but long term evaluation is needed. Vernon in U.S.A. has even combined a hearing aid and masking device with good effect in some cases.

Currently there are 3 ways of masking (1) by a hearing aid (2) by the tinnitus masker and (3) F.M. Masking.

1. The hearing aid for the relief of tinnitus: Since 1940s hearing aids have been fitted to some patients to amplify external noises and mask or provide distraction from the tinnitus. While the internal noise of some of these hearing aids may have accidentally provided additional masking, in the past few years, noise generators have been deliberately incorporated into aids to act as tinnitus maskers. It is assumed that amplification of ambient environmental noises will provide masking sound which will cover up the tinnitus patients are fitted with an open tube configuration utilizing a free field ear mould. Some advocates of this approach report 70-80% success rates in tinnitus relief, but others report perhaps a more realistic effectiveness with some 20-30% of patients being helped. Most of the effect of tinnitus maskers is by psychological relief from tinnitus, but cases have been reported in which the tinnitus is abolished after the masker has been removed.

2. The tinnitus masker for relief of tinnitus: Masking as a relief for tinnitus was mentioned by Hippocrates, as early as 400 B.C. It was proposed by Delvees and Vernon in 1975. Tinnitus masker is worn like a behind the ear hearing aid and generates a band of noise or a tone. In essence, it is the substitution of an external relatively pleasant bearable noise for an internal unpleasant, unbearable noise. The intent is to provide a band of noise

starting at frequencies above the speech frequencies and extending to as high frequency region as possible. Technical limitations of equipment have restricted the tinnitus masker to an upper limit of 7000 Hz. Tinnitus masker is equipped with a volume control ranges from 40-80 dB SPL. The noise band produced by the tinnitus masker is capable of masking in this region, while at the same time it does not interfere with speech reception. It is likely that the masker produces a sound which not only masks the tinnitus, but a sound which is more acceptable than that of tinnitus. A band of noise is less pleasant than the screech of a high pitched tone. Also the masker is an external sound and as such perhaps it can be more easily suppressed or ignored than internally generated sounds. Thus it can be rare that a tinnitus patient objected the sound produced by the masker.

3. Feldman reported that external sounds presented via earphones mask the tinnitus in 89% of 200 patients mostly with sensori-neural hearing loss.

3. F.M. Masking for the relief of tinnitus - For some patients the major complaint about tinnitus is its disruptive effect upon sleep. Their tinnitus was such that routine daily activity could mask it. At night tinnitus becomes a problem. In such cases F.M. masking is recommended, i.e. detuning an F.M. radio so as to produce static which occurs

between stations. The patient is instructed to increase the intensity of the FM static until it masks the tinnitus and to arrange a bedside radio, so that it is available throughout the night. F.M. masking has the disadvantage of producing wide band noise extending into the speech frequencies, so that it disrupts speech reception. Some patients, however, use it for periods of concentration or work.

## CONCLUSION

The problem and ubiquitous nature of tinnitus is well known. Tinnitus is the most common symptom associated with hearing impairment and also prevalent where there is no demonstrative hearing impairment. Many things conspire to complicate and confuse the matter of tinnitus and is a subjective experience for which objective indicators are lacking. It is also confusing that it can arise from a great variety of insults ranging from concussion of the brain, whiplash and excessive noise to disease states such as otosclerosis, diabetes, hypertension, Meniere's disease and the like. A very high proportion of noise and drug induced hearing loss is accompanied with tinnitus and yet the extent of hearing loss is not clearly related to the severity of the tinnitus. It is also observed that many tinnitus patients deny any hearing loss only to have testing reveal extensive high frequency losses. It is believed that tinnitus indicates structural physiological dysfunction and further that it is amenable to treatment. Wide variety of medical, surgical, psychotherapeutic, bio-feed back training, relief of tinnitus through hearing aids, tinnitus maskers, F.M. masking techniques have been applied with varying results. More research towards diagnostics, evaluation and treatment procedures can only unfold the poorly understood problem of tinnitus for the application of suitable way of treatment (medical, surgical, psychological or through masking aids).



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