TINNITUS

Definitions:

The sensation of hearing a sound that originates involuntarily in the head of its owner, in the absence of an external stimulus

- May be **pulsatile** or **non-pulsatile**
- Pulsatile tinnitus may have an intracorporeal sound source
- **Subjective** or **objective**
- There is some ambiguity as the definition technically includes auditory hallucinations of mental illness, which is **not** considered to be a type of tinnitus
- Sound can take any form: usually simple (e.g. humming, whistling, ringing, notes), but may be complex (e.g. indistinct music)

Non-Pulsatile Tinnitus:

- Idiopathic Tinnitus = commonest form of non-pulsatile tinnitus
- *[Prevalence: 10.1% UK adults (spontaneous persistent tinnitus lasting ≥5min)*
    - *Prevalence increases with age, but decreases in severity*
    - *Male : Female = 1:1]*
- Predictors of Tinnitus:
    - Hearing loss (especially high frequency Hearing Loss)
    - Noise exposure (up to 3x increase in tinnitus compared to no noise exposure)
    - Advancing age (although tinnitus seems to plateau at 70 years of age)
- [Risk Factors:
    - Smoking and ETOH use
    - Previous Head injuries
    - Cardiovascular disease / Hypertension (HPT)
    - Otologic conditions: Meniere's disease, otosclerosis, vestibular schwannoma
    - Drugs: salicylates, quinine, aminoglycosides, platinum based anti-neoplastic drugs
    - Diet: higher caffeine consumption may be **protective against tinnitus]**
- Associated Comorbidities: Depression and Anxiety; Disorders of sound tolerance (eg. hyperacusis: 40% tinnitus sufferers report hyperacusis; 86% hyperacusis sufferers report tinnitus)

PATHOPHYSIOLOGY OF TINNITUS:

1. **Ignition site** = peripheral or central location in the auditory system where the initial tinnitus signal is generated
2. **Central promotion** = central auditory mechanisms which must be present for the generated signal to be misconstrued as a sound

[Numerous theories exist outlining mechanisms which lead to tinnitus:

- ***Peripheral mechanisms:***
    - *Discordant damage of cochlear hair cells: outer hair cells damage while inner hair cells remain intact*
    - *Calcium channel dysfunction: drugs (salicylates and quinine) and noise exposure affect intracellular calcium levels, which may cause depolarization*
- ***Central mechanisms:***
    - *Increased spontaneous neural activity in the auditory cortex*
    - *Central neural synchrony and reorganization of the cortical auditory map: Peripheral auditory damage results in increased synchrony of spontaneous cortical activity → may result in tinnitus*

- *Habituation: the central auditory system should habituate tinnitus over time, although this may not happen when there is high autonomic arousal (basis of tinnitus retraining therapy → working with autonomic nervous system, limbic system, reticular system)]*

WORK UP: INVESTIGATIONS

1. Audiometry with pure tone audiogram and tympanometry
2. Imaging: **Not for everyone.** Consider an **MRI** when concern of retro-cochlear pathology
    a. Unilateral tinnitus
    b. Asymmetrical sensorineural hearing loss (with no clear cause)
    c. Associated neurological symptoms (vertigo, cranial nerve fallout)
3. *[Tinnitus Questionnaires: useful in research and assessing response to management*
    a. *Tinnitus Handicap Questionnaire; Tinnitus Handicap Inventory (THI);* ***Tinnitus Functional Index***
    b. *Visual analogue scales*
    c. *Hospital Anxiety Depression Scale (HADS)]*

TREATMENT:

- Multiple options available, but poor evidence base supporting these due to a lack of RCTs (randomized control trials)
- Probably placebo effect involved
- There tends to be natural improvement in tinnitus over time due to habituation

1. Explanation and Reassurance
    - Avoid negative counselling i.e. "there is nothing we can do"
    - Education and information about the condition, natural history etc. supported by RCTs
2. Hearing Aids
    - Useful in cases where there is associated hearing loss (intervention of choice)
    - Amplification of external sounds may mask tinnitus
    - Indirect benefits: enhance communication, reduce associated anxiety and stress
3. Sound Therapies (part of TRT)
    - Sound can mask tinnitus in 95% patients in a clinic setting
    - Complete masking of tinnitus is counterproductive as it limits habituation
    - Low level sound (just below tinnitus threshold) may aid in habituation to tinnitus signal
4. *Ultrasound*
    - *High-frequency sound applied by a bone conduction transducer → meant to stimulate cochlea without interfering with hearing sounds within the normal auditory spectrum*
    - *Promising initial study results*
5. Combination Therapies: Tinnitus Retraining Therapy (TRT)
    - Combining sound therapies with counselling, cognitive behavioural therapy (CBT) promote habituation and seem to show good results
6. Alternative Medicine
    - Relaxation techniques have proven benefit: Acupuncture, Aromatherapy, Herbal medicine, Massage, Hypnotherapy, Meditation, Yoga, Thai Chi etc. (seem to promote relaxation)
    - Ginkgo biloba 480mg daily dose shows some promising results
    - Vitamin supplementation: Vitamin Bs (theoretically protect cochlea against noise trauma); zinc (highest concentration of zinc in the human body is in the cochlea → but limited evidence of response to supplementation)
7. Systemic Medication
    - Tricyclic antidepressant; Serotonin reuptake inhibitors: useful in cases with coexisting depression / anxiety
    - Benzodiazepines: limited use due to dependence. May have some benefit in treating tinnitus

- No strong evidence: anticonvulsants, antispasmodics, betahistine, hyperbaric oxygen therapy
- Intravenous Local Anaesthetic: invasive but provides temporary central suppression of tinnitus (not practical long-term solution)

8. ***Regional Drugs***
    - *Botulinum toxin: has shown non-paralytic benefits in treating migraines and neuropathic pain (blocks acetylcholine release as well as other neurotransmitters in the autonomic pathway). One small study showed benefit to Botulinum toxin injection around the ear (not statistically significant as only 26 participants)*
9. ***Intratympanic Drugs***
    - *Direct labyrinthine drug absorption with theoretically improved labyrinthine metabolism - but will only target the peripheral pathways (i.e. cochlear pathology)*
    - *Potential use in cases of:* ***sudden onset sensorineural hearing loss, acute noise trauma, acute otitis media***
    - *Drug options: Steroids, local anaesthetic, non-ototoxic NMDA antagonists]*
10. Surgery
    - For specific conditions associated with tinnitus:
        - otosclerosis (stapedectomy eradicates associated tinnitus in 80-88% patients)
        - Cochlear implantation for profound hearing loss (86% tinnitus improvement on implanted side; 67% on contralateral ear)

Pulsatile Tinnitus:

- Refers to sound that is not continuous (clicking, pulsation, fluttering)
- Classified as: **Synchronous** or **Non-synchronous** (based on timing with patient's pulse)

Synchronous Pulsatile Tinnitus

Causes of Synchronous Pulsatile Tinnitus		
PATHOLOGY TYPE		EXAMPLES
Vascular	**Arterial**	Carotid artery atherosclerosis / dissection / stenosis Arteriovenous malformation / fistula Intracranial aneurysm Vascular anomalies of the ear Vascular compression of CNVIII (lateral)
	Venous	Jugular bulb abnormalities Dural venous sinus stenosis / diverticulum Abnormal emissary veins of mastoid Idiopathic tinnitus
Microvascular		Glomus tumour Paget's disease Cholesterol granuloma of middle ear Meningioma of middle ear Cavernous haemangioma
Circulatory		Increased Cardiac Output: anaemia, pregnancy, thyrotoxicosis

Perceptual	Conductive hearing loss Cochlear trauma
Other	Benign Intracranial Hypertension Superior Semicircular Canal Dehiscence

INVESTIGATION:

- Bloods: FBC; Thyroid function; bHCG (especially when bilateral)
- Imaging: depends on underlying aetiology
    - Contrasted CT temporal bone, brain: if retrotympanic mass seen
    - Duplex Carotid Ultrasound: Atherosclerosis
    - MRA/MRV "Time of Flight": Intracranial venous and arterial anomalies
    - Gold standard = formal angiography but this is invasive, therefore decision of imaging depends on clinical picture

TREATMENT:

- Supportive
    - Counselling and Reassurance
    - Sound therapy
- Surgery
    - Targeted at identified cause: Microvascular decompression of vascular loops; decompression of sigmoid sinus dehiscence; coiling / tying off vascular anomalies
    - Variable results: 40-77% improvement of tinnitus

Non- Synchronous Pulsatile Tinnitus

= buzzing / fluttering sounds that are not related to the patient's pulse

PATHOLOGY	FEATURES
Middle Ear Muscle Myoclonus	- Always subjective tinnitus - May have impedance changes on audiogram over time - Tensor tympani / Stapedius muscle

Palatal Muscle Myoclonus	- Can be objectively audible to others
	- Irregular clicking
	- Associated with involuntary palatal muscle movements which can be seen (transorally or transnasally)
	- 2 forms:
	  - Symptomatic palatal myoclonus = associated with brainstem lesions
	  - Essential palatal myoclonus = idiopathic, isolated condition

Otologic: Middle ear	- Patulous eustachian tube - Ossicular chain pathology; Middle ear effusion - Otosclerosis - Semicircular canal dehiscence
Joint Disorders	- Temporomandibular joint disorder

- ***INVESTIGATION:***
    - *Audiogram to assess impedance changes over time (middle ear myoclonus)*

    - *MRI if palatal myoclonus: to exclude brainstem lesions*
- ***TREATMENT:***
    - *Conservative therapies as for non-pulsatile tinnitus*
    - *Pharmacotherapy: Benzodiazepine, Orphenadrine; Carbamazepine; Botulinum toxin*
    - *Failed above: can consider surgical division of stapes and tensor tendons]*

SUMMARY: