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Tinnitus

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Abstract. *Tinnitus.* This article is based on a review of the literature and the experience of some experts. Its goal is to present an overview of the physiopathology of tinnitus and perspectives of treatment based on recent publications. Tinnitus is a problem of society, affecting about 10% of the population. The causes of tinnitus are extremely diverse. Objective tinnitus is generally pulsatile and from arterial or venous origin; subjective tinnitus can be generated at any level of the auditory pathways. Approach to tinnitus includes qualification through anamnesis and specialized questionnaires, and thorough audiological characterization. Sometimes, imaging is indicated as it can reveal the cause of the tinnitus in case of a vascular abnormality or a retro-cochlear tumour. Among the various medications prescribed for tinnitus, only anti-depressants proved to be efficient when secondary depression is present. Hearing aids are useful for hearing impaired patients but the efficiency of tinnitus maskers is not proved. Tinnitus Retraining Therapy is very promising but results must be confirmed by future studies. Studies about neurostimulation are in progress. In the future, better understanding of the physiopathology of tinnitus will lead to new treatments.

Foreword

When one studies the literature of tinnitus, many different approaches are found but no study meets the EBM level I criteria. Therefore, any guideline on tinnitus management should be considered as a support rather than a strict protocol with information about what must be done to diagnose or treat.

Definition

Tinnitus (from the Latin *tinnire*, which means to ring or to tinkle) refers to the sensation of any sound perceived in the head or in the ears without an evident external stimulus.[1] Two main types can be identified: objective tinnitus which is caused by sounds generated somewhere in the body and subjective tinnitus, which is the perception of meaningless sounds without any physical sound being present.[2] Tinnitus should be differentiated from auditory hallucinations, which are perceptions of meaningful sounds, such as music or speech and are generally considered as a symptom of psychiatric or neurological disease.

Prevalence

Throughout life, most people occasionally experience tinnitus, and a high proportion of normal hearing subjects experiences tinnitus in a sound proof booth (64% in a group of 120 normal hearing volunteers). Studying the prevalence of tinnitus is difficult because tinnitus is subjective and therefore not amenable to straightforward quantification. Nevertheless, the epidemiological aspects of tinnitus are well established: tinnitus is highly prevalent throughout the world, and increases with age, amount of hearing loss, and amount of noise exposure.[3] According to the US National Centre for Health Statistics, tinnitus affects up to 30% of the adult population, with 6% of these individuals reporting incapacitating symptoms.[4] A British National Study of Hearing in 1978 found that at least 8% of persons with tinnitus, experience it as a moderate to severe annoyance or causing interference with sleep. Only 0.5% of them reported tinnitus as severely reducing their ability to lead a normal life and as causing depression and anxiety. A large scale Swedish study reported somewhat higher figures: 2.4% of the adults report that their tinnitus annoys continually. In 1996 the US National Centre for Health Statistics found that the prevalence of tinnitus was 3%, when all ages were considered. In fact the prevalence was 1% under the age of 45 and 9% over the age of 65.

Etiology

The possible aetiologies of tinnitus are extremely diverse. Table 1 gives an extensive listing of conditions that are possibly associated with tinnitus. In many of these diseases, tinnitus is not the major complaint. In a small number of patients, the tinnitus is a pulsatile sound that synchronizes to the heartbeat or the respiration. For these patients, tinnitus is likely to be caused by a vascular abnormality and can sometimes be cured. For most patients, however, tinnitus is associated with a (neuro)otologic disease. All pathologic states of the ear can actually be associated with tinnitus.

Physiopathology

Tinnitus most probably does not reflect a single entity but rather a diverse group of functional changes in the central auditory pathways. Different models have been proposed.[5]

One influential view stresses the analogy between tinnitus and chronic pain. As chronic pain, tinnitus is a phantom perception that can be caused by any lesion along the auditory pathways. Such lesions eventually cause down regulation of intracortical inhibition and tinnitus. For example, the loss of hair cells in a region of the cochlea will reduce the spontaneous neural activity of the corresponding auditory nerve fibres. This may result in a reduction of lateral inhibition at more central levels and lead to hypersensitivity and hyperactivity of a population of neurons at cortical level.[6]

Another model of tinnitus, the model of Jastreboff,[7] (see appendix 1) integrates two fundamental properties of the human central

Table 1
Etiologies of tinnitus

OBJECTIVE TINNITUS

1. Pulsatile tinnitus

1.1. Arterial etiologies
- 1.1.1. Atherosclerotic carotid artery
- 1.1.2. Intracranial and extra cranial arteriovenous malformations
- 1.1.3. Arterio-venous fistulas
- 1.1.4. Atherosclerotic subclavian artery
- 1.1.5. Atherosclerotic occlusion of the contralateral common carotid artery
- 1.1.6. Fibromuscular dysplasia of the internal carotid arteries
- 1.1.7. Carotid artery dissection
- 1.1.8. Intrapetrous carotid artery dissection
- 1.1.9. Brachiocephalalic artery stenosis
- 1.1.10. External carotid artey stenosis
- 1.1.11. Ectopic intratympanic carotid artery
- 1.1.12. Persistent stapedial artery
- 1.1.13. Aberrant artery in the stria vascularis
- 1.1.14. Microvascular compression of the eighth nerve
- 1.1.15. Increased cardiac output (anemia, thyrotoxicosis, pregnancy)
- 1.1.16. Aortic murmurs
- 1.1.17. Paget's disease
- 1.1.18. Otosclerosis
- 1.1.19. Hypertension, antihypertensive agents
- 1.1.20. Vascular neoplasms of skull base and temporal bone
- 1.1.21. Tortuous carotid and vertebral arteries

1.2. Venous etiologies
- 1.2.1. Pseudotumor cerebri syndrome or benign intra cranial hypertension syndrome or idiopathic intra cranial hypertension in association with:
  - Obesity
  - Anemia (iron deficiency or pernicious)
  - Polycythemia
  - Steroids
  - Deficiency: Addison's disease, steroid withdrawal
  - Excess: Cushing's disease, iatrogenic
  - Hypoparathyroïdism
  - Hyperthyroidism
  - Pituitary adenoma
  - Uremia
  - Cystic fibrosis
  - Vitamins (deficiency of vitamin D, excess of vitamin A)
  - Medications: steroids, dilantin, chlorpromazin, lithium, tetracycline, trimethoprim-sulfamethoxazole, amiodarone, growth hormone, oral contraceptives, indomethacin, nalidixic acid
- 1.2.2. Jugular bulb abnormalities
- 1.2.3. Hydrocephalus associated with stenosis of the Sylvian aqueduct
- 1.2.4. Increased intracranial pressure associated with Arnold-Chiari malformation
- 1.2.5. Abnormal condylar and mastoid emissary veins
- 1.2.6. Venous hum or idiopathic or essential pulsatile tinnitus

2. Non pulsatile tinnitus
2.1. Patulous eustachian tube
2.2. Palatal myoclonus
2.3. Spasm of stapedius or tensor tympani muscle
2.4. Temporomandibular joint disorders (Clicking sounds)
2.5. Spontaneous otoacoustic emissions

Table 1
Continuation

SUBJECTIVE TINNITUS

1. Outer ear

1.1. Impacted cerumen/wax
1.2. External otitis
1.3. Foreign body
1.4. Exostosis or stenosis of the external auditory canal

2. Middle ear

2.1. Tuba dysfunction; barotrauma
2.2. Acute otitis media; glue ear
2.3. Chronic suppurative otitis media; cholesteatoma; perforated tympanic membrane; ossicular lesions
2.4. After middle ear surgery
2.5. Otosclerosis

3. Inner ear

3.1. Infection (labyrinthitis)
3.2. Autoimmune diseases
3.3. Trauma
  – Noise-induced hearing loss
  – Shock
  – Baro traumatism with perilymphatic fistula
3.4. Presbyacusis
3.5. Drugs ototoxic (aminoglycoside antibiotics, salicylates, loop diuretics, non steroidal, anti-inflammatory drugs, quinine, chemotherapy agents: e.g. platins, vincristin)
3.6. Intoxication with lead, mercury, carbon monoxide
3.7. Sudden deafness
3.8. Meniere's disease
3.9. Micro vascular causes: general vascular causes, hematologic factors, metabolic and endocrinian factors (diabetes, hyperlipidemy, hyperuricemy, hypo or hyper thyroïdism), cervical distortions and cervical osteoarthritis

4. Auditory nervous system

4.1. Vestibular schwannoma (acoustic neuroma)
4.2. Other retrocochlear/cerebellar-pontine-angle tumours (meningioma)
4.3. After neuritis
4.4. Vasculo-nervous conflict (V/VIII) / vascular loop
4.5. Cerebral tumours;
4.6. Infectious: meningitis; encephalitis; sequel of Lyme disease; syphilis;
4.7. Closed Head injury
4.8. Multiple sclerosis
4.9. Vascular accident
4.10. Migraine (aura)
4.11. Lesion of the temporal lobe
4.12. Auditive hallucinations (psychiatry)

5. Others

5.1. Temporomandibular joint dysfunction and dental disorders
5.2. Cervical causes; whiplash
5.3. Psychological / psychiatric disorders (anxiety, depression, alcohol abuse, schizophrenia)
5.4. Inadequate emotional answer to stimulus
5.5. Hormonal cycle
5.6. Toxaemia

nervous system: the conditioned response paradigm (first demonstrated by Pavlov) and habituation. Habituation is the decrease in the strength of a behavioural response that occurs when a stimulus is repeatedly presented. This model suggests that the auditory pathways play a role in the initial generation of tinnitus, but the perception and the reinforcement of the phantom perception is maintained by extra-auditive pathways, especially the limbic system (playing an important role in our emotions) and the autonomic nervous system. The Tinnitus Retraining Therapy is based on the model of Jastreboff.

Until recently, fundamental knowledge on tinnitus was based on animal research.

Recently, it became possible to study the neurophysiology of tinnitus in humans in a non invasive manner with magneto encephalography, positron emission tomography (PET), and fMRI. Magneto encephalographic (scalp recorded variations of the magnetic fields in relation with neuronal depolarisation) studies on humans with tinnitus suggest a relation between tinnitus and brain plasticity.

They find altered cortical tonotopy in patients with tinnitus as is also observed in the somatosensory cortex of amputees who experience phantom pain. Those similarities are in favour of the hypothesis that tinnitus may be an auditory phantom phenomenon.[8]

PET studies measure the metabolic brain activity via a radiotracer and find that tinnitus and residual inhibition are related to networks of auditory higher–order processing, memory and attention.[9] It is a very sensitive technique that can also be used in

cochlear implanted patients. Disadvantages are the use of radioactive substances and the lack of equipped centres. Recently, also the fMRI technique, has been used to study tinnitus, and first reports are promising.[10] Our understanding of the neurophysiological mechanisms that lead to a phantom auditory perception and the associated debilitating consequences of this sensory experience is continuing to improve. Tinnitus appears to be significantly affected in a complex way by somatosensory, limbic and motor influences. Effective treatments will certainly emerge from these new insights.[11]

Diagnostic work-up

Questioning

A full diagnostic work-up for tinnitus includes extensive anamnesis. Table 2 lists a number of questions relevant to tinnitus: context of the tinnitus and specific aspects of tinnitus. Different groups developed, mostly for research purposes, specific questionnaires to estimate quantitatively the impact of tinnitus on daily living. Those questionnaires are of certain interest in the follow-up op the patient but in either case can not replace the anamnesis. The Tinnitus Handicap Inventory (THI) yields a psychometrically robust measure of tinnitus handicap based on self-report of the patient.[12,13] The higher the score, the more distressing the tinnitus. With a score between 0 and 16, the handicap is considered slight, being heard only in quiet environments and easily masked. In contrast, a score of 78 to 100 is considered catastrophic, with all tinnitus aspects experienced as severe. These patients need specialized counselling, and psychological problems are commonly associated. According to our experience, the THI questionnaire is most practical from an administrative perspective, and is used frequently (see Appendix 2). The subjective tinnitus severity scale (also available in French) consists of 16 questions to be answered with yes or no, yields a metric of tinnitus severity on a 16 point scale, and enables classification into mild, intermediate or severe tinnitus. In case of hyperacousis, the “questionnaire de sensibilité auditive” which measures the impact of hyperacousis on concentration, social life and emotional life can be used. The Hospital Anxiety and Depression scale is useful to explore anxiety and depression associated with tinnitus.[14,15]

Table 2

Context of the tinnitus

- Hearing loss? Hyperacusis? Vertigo? Otalgia? Otorrhoe? Ear surgery? Headache? Nasal symptoms?
- Noise exposure: (profession, hobby)
- Internal diseases (diabetes, dysthyroïdism, oncological)
- Vascular diseases (arteriosclerosis, hypertension, hypercholesterolemia ...)
- Psychiatric diseases
- Temporo-mandibular joint
- Cervical spine
- Drug abuse: tobacco, alcohol
- Medication (present and old); Ototoxic agents (quinine, aminoglycosides, loop diuretics, chemotherapy ...)
- Previous tinnitus treatments: hearing aid, acupuncture, homeopathy, hypnosis
- Personal and familial history

Specific aspects of tinnitus

Time course: onset, gradual or sudden, constant, intermittent, fluctuating
trigger
localisation: right ear – left ear – both ears – head
Pulsatile or not
Perceived quality: pure-tone, high or low frequency, humming, hissing...
Pitch
Loudness
Presence of real silence
Can the patient modulate her/his tinnitus?
Awareness of tinnitus in percentage of waking hours
Annoyance of the tinnitus
Impact on sleep
Impact on activities (work, concentration, reading, sports, social, other...)
Effect of noise on tinnitus (louder, softer)
Patient’s beliefs about tinnitus (will get worse, will go on for ever, there is no treatment, a tumour, become deaf)

Physical examination

An otological examination with otomicroscopy is required. Depending on the anamnesis, examination of the neck with carotid auscultation, of the temporal joint and otoneurological examination, particularly if complaints of dizziness are associated, are also indicated.

Audiological testing

Audiologic tests play a central role in the diagnostic work-up of tinnitus. Pure-tone hearing thresholds and speech discrimina-

tion should always be obtained, and guide further investigations. In many clinics, audiological characterization of tinnitus has become common practice. Results of these measurements reveal the variety of tinnitus, can sometimes be correlated with pure-tone thresholds, but are poorly correlated with the amount of tinnitus distress.[16]

Seventy-five per cent of the patients match their tinnitus pitch at or above 4000 Hz (median: 6000 Hz). 70% of the loudness matches are between 0 and 6 dB sound level (SL). Loudness matches are useful to confirm the genuine perception of tinnitus especially when compensation is sought for tinnitus in the context of occupational hearing damage or automobile injuries. In litigation cases, the loudness match of pretended tinnitus must be determined on at least five separate occasions. When the variation of the loudness matches does not vary by more than 3 dB, tinnitus perception is probably genuine, because it is extremely difficult to remember the loudness of a tone accurately.

Table 3 gives an extensive listing of tests that can be of some interest in the evaluation of tinnitus.

Laboratory tests

In our tinnitus working group, we could not obtain consensus concerning the laboratory tests that should be routinely performed in patients with tinnitus. However, considering Table 1 and all the pathologies that can be involved in patients complaining of tinnitus, some laboratory tests should be performed depending of the medical history, circumstances and other symptoms associated. For indication, Table 4 presents a series of tests that seem to be sometimes of interest in tinnitus evaluation.

Imaging

- Magnetic resonance imaging (MRI) should be considered in patients with unilateral tinnitus or/and those with asymmetric hearing loss.
- A CT scan of the temporal bone and the posterior fossa can still be useful in case MRI is not possible, and in case of suspected middle ear pathology.
- In case of unilateral pulsatile tinnitus, for detection of carotic artery stenosis, a duplex ultrasound is reliable. Others etiologies (aterio-venous malformations, jugular bulb and carotid abnormalities, chemodectome ...) are diagnosed by MRI, angio Mri or CT. Arteriography is seldom indicated.

Treatment

Guidelines for a constructive tinnitus treatment relationship (Recommendations Level C)

1. **Express sympathy and care**. Tinnitus patients often hope that cure is possible. Whatever you advise, you must not leave patients without hope. Never tell patients “There is nothing I can do” or “You must learn to live with it”. Because of the rapid

Table 3

Audiometry
- Pure tone audiometry (standard frequencies and high frequencies if possible), air and bone levels
- Loudness discomfort level
- Speech audiometry

Tympanometry
- Tympano ossicular compliance
- Acoustic reflexes: to be considered especially if suspicion of otosclerosis (because of the high intensity of the stimulation, there is a risk of majoration of tinnitus, particularly in patients suffering from hyperacusis)

Otoacoustic emissions
- Spontaneous otoacoustic emissions
- Sound evoked (clicks, two-tone)
- Transient otoacoustic emissions with controlateral masking

Auditory brainstem response (BERA)
In case of unilateral tinnitus, unilateral hearingloss, auditory neuropathy.

Vestibular evaluation
If complains of dizziness and / or abnormal neurotological examination

Tinnitus testing
+ Pitch match frequency (by adaptive method using supra liminar pure tones or narrow band stimulations)
+ Tinnitus loudness match (TLM) (ascending method in best hearing ear)
+ Minimum masking level (MML) (defined as the lowest sound intensity that renders tinnitus inaudible In the majority of the patients, MML occurs between 0 and 9 dB SL)
+ Residual inhibition testing (RI) (refers to the temporary disappearance of tinnitus after presentation of a masking sound, and can be complete or partial; e.g., MML plus 10 dB for 60 seconds).

Table 4

+ Complete blood count
+ Lipids
+ Ionogram
+ Uremia
+ Fluorescent treponema antibodies (FTA) ; Lyme disease
+ Thyroid hormones
+ Viral serology : EBV, CMV, herpes zoster and simplex

progress in tinnitus research, new therapies will emerge. You must convince patients that, even if current techniques fail, tinnitus research is daily making progress and that one day it will help them.

2. **Establish your interview as a dialog**. Do not simply see the patients, but interact with them. First, find out how tinnitus affects the individual and know the specific nature of their difficulties to select appropriate treatments. Second, encourage patients to ask questions and answer those questions honestly and completely. Third, be forthright in admitting incomplete knowledge when such exists.

3. **Develop a relax yet attentive interview format**. Do not distance yourself with unnecessary barriers (seating at a long distance, white coat). Encourage a spouse, a friend, or a family member to attend the session.

4. **Use an interviewing approach** that allows you to give your undivided attention to the patient. Provide adequate time for thorough interviewing, allowing large opportunities for patients to explain and comment their tinnitus history, and to ask questions.

5. **Provide clear explanations** of all your procedures in layman's language. Explain carefully the options of therapy. It is important to indicate both positive and negative aspects, using layman language but be careful to avoid any tone of condescension.

6. **Remember the quality of life.** Develop a broad concern about all aspects of the patient's problems (family difficulties, noisy work environment, signs of deafness or brain disorder).

Medications

No drug has proved effective in curing tinnitus (Evidence level III). In his review of 69 randomized clinical trials (RCTs), Dobie concluded that "no treatment can yet be considered well established in terms of providing replicable long-term reduction of tinnitus impact, in excess of placebo effects".[17] In randomized clinical trials, placebo effects are strong and are attributed in part to the medical attention provided to the patient.[18]

1. Tricyclic antidepressants are likely to be beneficial
An RCT with subjects suffering from depression and chronic tinnitus found that, when compared to placebo, the tricyclic antidepressant nortriptyline significantly improved tinnitus related disability, reduced tinnitus loudness, and symptoms of depression at 6 weeks, but found no significant difference in tinnitus severity.[19]

2. Benzodiazepines did not prove effective in controlling tinnitus.[20]
Prescription of benzodiazepines should be tempered because withdrawal intensifies tinnitus and causes supplementary distress.[21]

3. Antiepileptic drugs
One RCT found no significant difference between carbamazepine (150 mg 3 times daily for 30 days) versus placebo for tinnitus severity.[14] Three other studies also failed to show benefit.[22]

4. Nicotinamide, vitamine B3
One RCT found no significant difference between nicotinamide versus placebo for tinnitus severity at 30 days.[19]

5. Cinnarizine
One RCT found no significant difference between cinnarizine versus placebo for tinnitus severity.[19]

6. Zinc
One RCT found no significant difference between zinc versus placebo for tinnitus severity at 8 weeks.[19]

7. Baclofen
One RCT found no significant difference between baclofen versus placebo for tinnitus severity.[19]

8. Ginkgo Biloba is likely to be ineffective or even slightly harmful.
One systematic review and an RCT found no significant difference of tinnitus symptoms with ginkgo biloba versus placebo, and another study reported stomach upset, dizziness and dryness.[19]

9. Tocainide is likely to be ineffective or even harmful.
One RCT found no significant difference with tocainide versus placebo in improving symptoms, but found evidence that tocainide caused significantly more adverse effects after 30 days treatment (rash 25%, dizziness 12%, tremor 8%).[19]

Randomized clinical trials of flecainide and mexiletine were marked by adverse drug effects in up to 70 percent of the participants, and by dropout rates of about 50 percent.[17]

Hearing aids

When hearing impairment is present, hearing aids should be advised, as they mask the tinnitus through amplification of ambient sounds. Some patients attribute their hearing problems to tinnitus. For them, providing hearing aids often results in improvement of tinnitus distress[1] (Recommendation level C).

Tinnitus maskers

Maskers look similar to hearing aids and deliver broadband noise in the external auditory meatus. The instruments generate an external, constant sound that can be controlled by the patient, and allow concentration rather than distraction by sounds from televisions, etc. They reduce tinnitus distress by diverting attention, replacing tinnitus by another noise, under control.[23] The sound produced by a tinnitus masker is not a white noise. Although tinnitus maskers have been widely prescribed, no evidence supports their use. Only one randomized clinical trial (RCT) was found (75 people suffering from tinnitus but without hearing loss). The RCT compared two types of tinnitus masking devices versus a non-blinded control group and found that either type of device improved symptoms compared to no device[24] (Evidence level II).

The philosophy of tinnitus maskers is very different than the one of tinnitus retraining therapy (TRT®) where a white noise is used and where the intensity of the noise stimulation remains always under the level of masking.

Tinnitus Retraining Therapy

Regarding clinical evidence, this technique has to be covered in future updates. (Evidence level III). For Hazell *et al.*[7], TRT® is primarily a long-term treatment rather than a temporary palliative. This therapy exploits two approaches: psychological and prosthetic. The first is more important, and aims to remove inappropriate beliefs and fears about tinnitus, which enhance tinnitus intrusiveness. The role of the ENT doctor is fundamental, as he provides important information. The prosthetic approach used in TRT® consists of providing background sound (white noise's generators) that does not mask the tinnitus. Such sounds gradually decrease the brain's oversensitivity to auditory signals. The modalities of this treatment are quite different if tinnitus is isolated, or associated with hearing loss and/or hyperacusis. When tinnitus is associated with hearing loss, the white noise's generator can be built in a hearing aid.

<u>The retraining programme:</u>

- change patient's beliefs
- reduce signal detection
- lower arousal level
- increase plasticity and decrease contrast by using sound therapies
- face object of disaffection without reaction

It is important to use teaching material and stories to illustrate different aspects of the retraining model. These factors will tailor counselling to each individual patient and his level of comprehension. The first consultation is of fundamental importance.

Other treatments

Acupuncture
An analysis of six RCTs failed to demonstrate any efficacy.[25]

Low Power Laser
One RCT found no significant difference of tinnitus severity between low power laser and placebo after one month of therapy.[19]

Electromagnetic stimulation / ear canal magnets have unknown effectiveness.
Four small RCT found insufficient evidence for effects of magnetic and electromagnetic stimulation.[19]

Hyperbaric oxygen has unknown effectiveness
No systematic reviews or RCT were found.[19]

Psychotherapy has unknown effectiveness.
One systematic review found insufficient evidence in favour of cognitive behavioural treatment, relaxation therapy, counselling, education, hypnosis, biofeedback, or stress management compared with other or no treatment in people with chronic tinnitus.[19]

Hypnosis has unknown effectiveness*
One RCT found no significant difference of tinnitus severity after three months of hypnosis versus counselling.[19]
* However, the trial may have lacked power to exclude clinically important effects.

Surgical treatments remain controversial.

Although some reports claim improvement of tinnitus after micro vascular decompression of the auditory nerve, the use of surgical treatments, including nerve section, remains controversial.[26,27]

Conclusion

Research has progressed our comprehension of the physiopathology of tinnitus. Nevertheless, many factors remain unclear. What has to be stressed is certainly the necessity of audiological investigation and to provide time enough for a good anamnesis. Tinnitus is a very frequent problem of which the etiology remains often uncertain, and the possible presence of a VIIIth cranial nerve schwannoma must be kept in mind, particularly in cases of unilateral tinnitus. In those cases, an MRI scan has to be done. When pulsatile tinnitus is present, vascular pathologies should be searched for. It is now well accepted that tinnitus reflects an unlucky brain adaptation to many possible disturbances within or outside the auditory pathways. Therefore the therapeutic approach has to take into account auditive and psychological aspects. The TRT, at present, seems to be one form of treatment giving certainly good results in some cases. A lot of treatments have been proposed but in the majority, correct evaluations disprove their efficiency. In case of depression related to tinnitus, antidepressive drugs have been proved useful. In the future, refined comprehension of tinnitus will certainly lead to new approaches of treatment.

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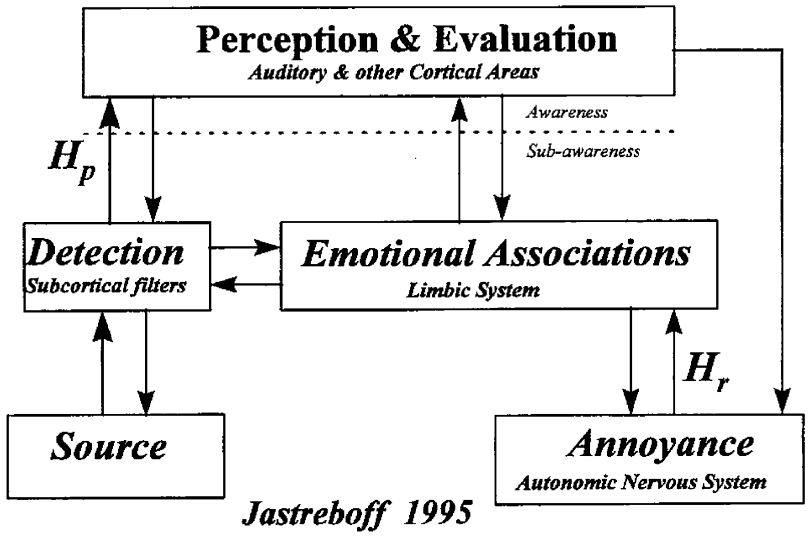
APPENDIX 1
Schema Jastreboff's Model



APPENDIX 2

Tinnitus Handicap Inventory
The purpose of the scale is to identify the problems your tinnitus may be causing you. Check "Yes", "Sometimes" or "No" for each question.

F1. Because of your tinnitus, is it difficult for you to concentrate?
F2. Does the loudness of your tinnitus make it difficult for you to hear?
E3. Does tinnitus make you angry?
F4. Does your tinnitus make you feel confused?
C5. Because of your tinnitus, do you feel desperate?
E6. Do you complain a great deal about your tinnitus?
F7. Because of your tinnitus, do you have trouble falling to sleep at night?
C8. Do you feel as though you cannot escape your tinnitus?
F9. Does your tinnitus interfere with your ability to enjoy your social activities such as going out to dinner, to the movies, etc?
E10. Because of your tinnitus, do you feel frustrated?
C11. Because of your tinnitus, do you feel that you have a terrible disease?
F12. Does your tinnitus make it difficult to enjoy life?
F13. Does your tinnitus interfere with your job or household responsibilities?
E14. Because of your tinnitus, do you find that you are often irritable?
F15. Because of your tinnitus, is it difficult for you to read?
E16. Does your tinnitus make you upset?
E17. Do you feel that your tinnitus problem has placed stress on your relationship with members of your family and friends?
F18. Do you find it difficult to focus your attention away from your tinnitus? And on other things?
C19. Do you feel you have no control over your tinnitus?
F20. Because of your tinnitus, do you feel tired?
E21. Because of your tinnitus, do you feel often distressed?
E22. Does your tinnitus make you feel anxious?
C23. Do you feel you can no longer cope with your tinnitus?
F24. Does your tinnitus get worse when you are under stress?
E25. Does your tinnitus make you feel insecure?

Item are classified as pertaining to the functional (F) subscale, emotional (E) subscale or catastrophic (C) subscale. Answers of "yes" are scored 4 points, "sometimes" 2 points and "no" receives O points.

APPENDIX 3[28,29,30]

Algorithm : Tinnitus

Il all cases, history, microscopic otoscopie, audiological testing

UNILATERAL TINNITUS

Non pulsatile

– With conductive hearing loss → etiological treatment- if necessary, temporal bone TDM

– With neurosensoral hearing loss → cerebello- pontine MRI

- normal: see tinnitus + hearing loss treatment (*)
- pathologic: etiological treatment + **

– Without hearing loss → cerebello- pontine MRI

- normal: see tinnitus treatment (**)
- pathologic: etiological treatment and (**)

Pulsatile

1) Arterial pressure measure and thyroid test control

2) Tympanoscopy

– Retro tympanic mass → temporal bone TDM
→ if indicated, surgery

– Normal → Neck Duplex ultrasound
Cerebral Angio-IRM
Neck TDM

Bilateral tinnitus

Non pulsatile

If normal hearing → **

If conductive hearing loss → etiological treatment - if necessary, temporal bone TDM

If neurosensoral hearing loss

– Symetric hearing loss → *

– Asymmetric hearing loss → cerebello- pontine MRI
  - normal: see tinnitus + hearing loss treatment (*)
  - pathologic: etiological treatment + **

Pulsatile

1) Arterial pressure measure and thyroid test control
2) Neck Duplex ultrasound and Cerebral Angio-IRM

*: Hearing aid trial (except in cases of hyperacusis).
**: See different therapeutic approaches described in the text in the paragraph treatment.

CME QUESTIONS

1. What is the most pertinent exam for a patient with a persisting unilateral tinnitus without hearing loss?

   A – OAE
   B – BERA
   C – Temporal bone CT scan
   D – MRI
   E – Vestibular testing

2. Which of the drugs has Evidence Based efficacy?

   A – Cinnarizine
   B – Benzodiazepines
   C – Tricyclic antidepressants
   D – Ginkgo biloba
   E – Zinc

3. What is the worst thing to say to a patient suffering from tinnitus?

   A – I am not competent in this kind of therapy
   B – There is nothing that can be done for you. You will have to learn to live with it
   C – We will treat you but we do not know if it is curable
   D – This medical problem is not heavy
   E – Be quiet, I will probably cure you

4. What is the prevalence of incapacitating tinnitus in the population?

   A – 1 to 3%
   B – 3 to 7%
   C – 7 to 10%
   D – 10 to 16%
   E – 16 to 30%

5. Recruitment:

   A – Is responsible for hyperacusis
   B – Causes any discomfort to the patient
   C – Should always be treated with sound generators
   D – Is independent from hyperacusis
   E – Is the basis of phonophobia

6. Sound used in TRT®

   A – Must be provided by sound generators
   B – Must be set at the threshold of hearing
   C – Should not mask tinnitus
   D – Must be heard only one hour a day
   E – Must not be heard each day

7. Audiological testing should always include

   A – Otoacoustic emission
   B – Acoustic reflexes
   C – Auditory brainstem response
   D – Pure-tone audiometry
   E – Speech audiometry

8. The following therapies are advisable to supplement TRT®

   A – Xanax
   B – Anti-depressants
   C – Dietary constrains with suppression of alcohol, caffeine, salt
   D – Techniques of relaxation
   E – Zinc

9. In the different Jastreboff categories, which therapeutic element is always essential?

   A – Relaxation techniques
   B – White Noise Generators
   C – Directive counselling
   D – Psychological aid
   E – Hearing aid

10. In a pluridisciplinary medical team, which association is most appropriate?

    A – ENT, audiologist, psychotherapist
    B – Audiologist, psychotherapist
    C – ENT, dietician, psychotherapist
    D – ENT, psychotherapist
    E – ENT, audiologist

Answers: 1D; 2C; 3B; 4C; 5D; 6C; 7D; 8D; 9C; 10A