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Tinnitus

By Frank Pederick

Tinnitus is the term for noises which are heard "in the ears" or "in the head": buzzing, ringing, whistling, hissing, pulsing and other sounds which do not come from an external source. Emergence of tinnitus, usually lasting for a short period, is extremely common, e.g., after going to a disco, or listening carefully in a quiet room.

This is a natural phenomenon experienced by 75-90 percent or more of the population and goes away after a short time (even though loud music can damage your hearing). Persistent tinnitus occurs in about 10-17 percent of the population (up to a third of the elderly population), and is a result of the brain being concerned with analysing sound signals focusing on weak messages, which are part of normal ear function in most cases. There is no external sound corresponding to the patients' perception of sound; thus, tinnitus can be classified as a phantom auditory perception. The psycho-acoustical properties of tinnitus have basically no relation to the level of annoyance evoked by tinnitus. In other words, two people with an apparently very similar characteristics of tinnitus can differ dramatically in their level of annoyance. Some people who experience quite normal tinnitus emergence can easily get the idea that something dreadful has happened, e.g., damage to the ear, or brain, or that they are going to be stuck with the problem for life. This kind of negative and inaccurate thinking is frequently responsible for the focusing of the subconscious part of the brain on the small tinnitus signals coming from the ear. In some people a fear or phobia may develop for the perceived sound, creating feelings of annoyance, apprehension or even anxiety and depression. These emotions are generated in other areas of the brain outside the hearing mechanisms.

An epidemiological study by Axelsson¹ showed that the most common cause of tinnitus (37.8 percent of cases) was noise induced hearing loss. The majority of cases were attributed to many other causes, one being Meniere's disease (7.1 percent).

It is important to distinguish between emergence of tinnitus lasting a short time and persistent tinnitus which causes distress. Persistent tinnitus is often triggered or made worse by emotional events; bereavement, work and family stress, accident/ injury (not necessarily to the ear) retirement, or moving to a much quieter environment. It may start during a period of unrelated depression or anxiety. Persistence of tinnitus usually involves the development of feelings of hopelessness and despair that nothing can be done. In mild cases this may be reflected as a feeling of annoyance or disquiet. The traditional teaching on tinnitus was that it was caused by damage to the ear which couldn't be fixed. This ignores the fact that the majority of cases of hearing loss, noise exposure, etc., are NOT associated with tinnitus. Tinnitus is certainly twice as common in hearing impaired people, but not because the inner ear is below par! If you are hard of hearing, communication is often a strain. This 'straining to hear' focuses the subconscious brain to pick up anything coming from the inner ear. Everyone has background noise in the hearing system, just like a tape recorder with a blank tape playing. Hazell² says the perception and loudness of tinnitus depends on whether the brain suppresses these sounds, or whether it amplifies them because of their importance as a real or potential threat. Tinnitus is associated with the mechanisms of hyperacusis, recruitment and phonophobia.

Hyperacusis is due to an alteration in central processing of sound, and the cochlea is often completely normal. The traditional teaching involves only an understanding of "recruitment" due to cochlea damage. Since almost all people with hyperacusis can be helped by behavioural and "sound" therapy, it has become clear to some researchers that the symptoms cannot be the result of ear damage. Some people with hyperacusis also have phonophobia. This means they literally fear being exposed to a certain sound, sometimes because of the belief that it will damage the ear. Often these are normal environmental sounds (traffic, kitchen sounds, doors closing, or even loud speech) which cannot under any circumstances be damaging. In phonophobia certain complex sounds produce discomfort on the basis of their meaning or association, but other sounds which are enjoyed (music) can be tolerated at much higher levels. If there is a discrepancy in the level or different sounds which produce discomfort, then it is very likely that a degree of phonophobia exists. Phonophobia can lead to hyperacusis (by changes in central auditory processing), and a consequent persistence of abnormal loudness perception.

A normal ear is able not only to hear extremely quiet sounds (between 0 and 20 dB hearing level) but can also tolerate very loud sound without discomfort (up to levels of 115 dB hearing level). With hearing loss an inability to hear quiet sounds may be coupled with a paradoxical intolerance for loud sounds due to recruitment. An ear with recruitment might well be unable to hear sounds, particularly high frequency

sounds, below 50 dB, but find any sounds above 80 dB not only uncomfortable but liable to produce distortion. Recruitment is due to a reduction in neural elements in the inner ear (usually the hair cells), so that a small change in stimulus intensity produces a very big change in response of the ear. More nerve fibres are switched or 'recruited' for a corresponding sound stimulus. Another way to look at this problem is to consider the ear rather like a musical instrument. Think of the ear as an instrument 'playing sounds to the brain' where they are perceived in the auditory cortex. Most musical instruments have what musicians call a dynamic range. They are able to play very softly (pianissimo) or very loudly (fortissimo). A normal ear is one not only with good hearing but with a full dynamic range for different intensities of sound. A recruiting ear is one in which the dynamic range is narrowed or contracted. If sounds are heard at all they are heard in musical terms as fortissimo.

Perhaps the majority of people who have hypersensitivity and hearing loss, have hyperacusis rather than recruitment, or may have some degree of both. Even if recruitment exists it is possible to retrain the brain (central processing) to alter its appreciation of loudness and accommodate the smaller dynamic range of the hearing impaired and recruiting cochlea. Where the hearing is normal, or near normal, hypersensitivity is always due to hyperacusis (plus or minus phonophobia) and NEVER due to recruitment. This concept represents a fundamental change of thinking to traditional views given by many health care professionals. The model is based on basic, well-established neurophysiological and psychological principles.

Mainly:

- i) The processing of information occurs on several levels for each sensory system, each level contributing to the final stage when a signal reaches the cortex.
- ii) The auditory system is closely connected with the part of the brain that controls emotions and the automatic response of the body to danger.
- iii) Connections within the nervous system are continuously modified, resulting in the enhancement of significant signals, and a decrease of neuronal response to irrelevant signals.
- iv) Sounds that are new, or associated with a negative experience, are treated as significant, evoke an emotional response that triggers the body to prepare for "fight or flight." The repetition of these sounds results in enhancement of their perception and in a resistance of their perception to be suppressed by other signals. The repetition of signals not associated with positive or negative reinforcement results in

the disappearance of a response to their presence, i.e., in habituation.

v) The detection of sound occurs on a pattern-matching principle, allowing for nearly complete perception of a signal even when it becomes highly distorted.

Consequently, the main point of the theory of tinnitus based on these neurophysiological principles is the postulate that nonauditory systems, particularly the limbic system (involved in emotion) and the autonomic nervous system, which controls all body functions and triggers the "flight or fight" reaction, are essential parts of each case of troublesome tinnitus. The auditory pathways play a secondary role. According to this model the annoyance of tinnitus is determined exclusively by the limbic and autonomic nervous systems. The information about what causes tinnitus distress is new, and not known to all health care professionals. It is difficult to understand because of the complex pathways within and outside the auditory system that are involved. Many professionals simply do not believe the new ideas and quite rightly question them. However the Jastreboff³ model has now been verified by human and (in the U.S.) animal research, and also by new techniques of brain scanning (positron emission tomography) which can in a research laboratory measure and pinpoint tinnitus perception and annoyance in tinnitus sufferers. Although ear damage may trigger the onset of tinnitus emergence (about 15 percent of cases), other factors determine whether tinnitus persists and causes annoyance.

Tinnitus, like "pain," is a subjective symptom. The problem is compounded because several different mechanisms must operate to cause the persistent sensation of tinnitus. Therefore, it is difficult to measure objectively any improvements in the condition. For example, it has been reported previously that sectioning the eighth cranial nerve does not abolish tinnitus in a majority of patients; therefore, central mechanisms must act to preserve the tinnitus. Finally, we know that tinnitus can occur in a host of conditions other than ototoxicity, aging, and noise exposure. Other conditions that may produce tinnitus are migraine headache with auditory aura, temporal lobe seizures, and head injuries. Therefore, it is naive to think of tinnitus as a disorder with a unitary origin and a unitary "cure."⁴

Arnold, et al., have shown increased metabolic activity in the left primary acoustic area in chronic severe cases of tinnitus⁵ Denk⁶ considers that the most common type of tinnitus is of cochlear-synaptic origin. According to results of inner ear research, new possibilities of pharmacologically influencing the cochlear synapse have been found. By intravenous application of the specific quisqualate antagonist glutamic acid diethyl ester (GDEE) 77.2 percent of patients with cochlear synaptic tinnitus noted a tinnitus reduction.

In a retrospective study of 1,002 chronic tinnitus sufferers Vernon⁷ concluded with a recommendation for TMJ referral for those tinnitus patients with unknown etiology who demonstrate any three or more of the 10 TMJ indicators undefined in his paper.

In Japan, Ryu⁸ has shown neurovascular compression of the eighth cranial nerve is one of the causes of tinnitus. Lemaire and Beutter⁹ have shown that disturbances of brainstem evoked responses caused by tinnitus affect two types of brain waves. They considered an hypothesis of possible involvement of the efferent systems could be proposed. The findings of Attias et al.,¹⁰ point to a cortical information processing dysfunction, i.e., increased time required to respond in chronic tinnitus patients associated with visual and primarily with auditory stimuli. Event-related potentials may provide an objective electrophysiologic measure of tinnitus. Hiller, et al.,¹¹ discuss three possible conclusions : (i) tinnitus may be somatoform symptom; (ii) the findings may indicate a substantial comorbidity of two different conditions; (iii) tinnitus and somatization may be linked through common mechanisms of arousal and somatic anxiety. Miller¹² has suggested there is considerable evidence that both chronic pain and some forms of tinnitus are caused by changes in the central nervous system, and that the anatomic location of the physiologic abnormality causing the symptoms of chronic pain and some forms of tinnitus is not the same location to which the symptoms are referred, i.e., the ear for tinnitus and the location of injury pain. Such changes in the central nervous system may have been induced by peripheral processes such as tissue damage, but the changes can persist a long time after complete healing of a peripheral lesion. Seligmann¹³, et al., say tinnitus and hearing loss, both reversible and irreversible, are associated both with acute intoxication and long-term administration of a large range of drugs. The mechanism causing drug-induced ototoxicity is unclear but may involve biochemical and consequent electrophysiological changes in the inner ear and eighth cranial nerve impulse transmission. Over 130 drugs and chemicals have been reported to be potentially ototoxic. The major classes are the aminoglycosides and other antimicrobials, anti-inflammatory agents, diuretics, antimalarial drugs, antineoplastic agents and some topically administered agents. The vestibular system has many anatomical features in common with the acoustic system. Disturbances of the vestibular system may result in motion sickness. A newer explanation,¹⁴ known as the neural mismatch or sensory conflict theory, describes motion sickness as one's response to discordant motion cues to which one is not yet adapted. Motion sickness can occur when there is a discrepancy between the external stimuli gathered by the senses and the internal expectations associated with the body's actual state of movement. There could be an analogous situation in the acoustic system . The ear has the ability to "fill in " sounds which it does not actually hear. A discrepancy between detected sounds and expected sounds may also be a

factor in tinnitus.

The perilymph, which is separated from the endolymph by a membrane inside the acoustic and vestibular labyrinths, is connected to the cerebrospinal fluid. Variations in CSF pressures are in synchrony with normal perilymph pressure variations or by pressure applied to the thorax or the atlanto-occipital membrane (in the rabbit, and the human too, presumably).^{15,16}

The acoustic sensory organ rests in part on the basilar membrane, a part of the membranous lining of the labyrinth and consequently may be affected by CSF pressure variations. Four cranial nerves carry parasympathetic fibres to smooth muscles and glands cranial nerves III, VII, IX and X. There are four autonomic ganglia in the head ciliary, pterygopalatine, otic and submandibular; each receives three types of fibres:

Sensory: from a branch of the trigeminal nerve.

Parasympathetic: from cranial; nerves III, VII, or IX. These nerves synapse in the ganglion.

Sympathetic: from the sympathetic trunk from the wall of the closest artery.

Sense organs, including the cochlea, have been shown to be influenced by sympathetic impulses.¹⁷ The effect of increased impulse traffic in sympathetic fibres is to exaggerate their discharge causing them to report greater intensity of stimulation than is actually occurring or when none is occurring. The SNS affects the reticular formation and the reticulospinal system and the superior cervical ganglion affect cortical and subcortical activity.¹⁸ Carrick¹⁹ has undertaken research to show how cervical adjusting could affect cortical function and on blind spots. The nerves supplying the tympanic membrane are the auriculotemporal br. of the mandibular n., the auricular br. of vagus. and the tympanic br. of the glossopharyngeal nerve.

The use of ginkgo biloba²⁰ has been demonstrated to be effective in treating tinnitus in clinical double blind studies, and has been reported to improve recovery in cases of acute cochlear deafness, e.g., due to pressure changes or sound. Ginkgo biloba has been shown in many studies to be effective in rectifying cerebral vascular insufficiency and thereby influencing mental performance.

Chiropractic Approaches to Tinnitus

Clearly from a theoretical basis chiropractic approaches to tinnitus are worth trying. Clinically improvements are often observed.²¹ Adjusting the cervical spine, especially upper cervical and cranium,²² could be a starting point. These methods have the potential to restore sympathetic/parasympathetic balance and influencing cortical function. If, as has been suggested by one researcher, there is a parallel between chronic pain and tinnitus, full spinal adjusting, extremity adjusting and soft tissue techniques also have a role to play. Fascial release techniques and trigger point techniques concentrating on paraspinal areas, suboccipital and mastoid muscle insertions and the TMJs would be a good beginning. Exercises and relaxation techniques to help to maintain tone of tissues could also play a role. Supplementation with ginkgo biloba may be useful.

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