

2.3 Decreased sound tolerance

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The model predicts that a relatively high percentage of tinnitus patients should exhibit increased sensitivity to an external sound, that is decreased sound tolerance. This is what one would expect from the increased auditory gain, or the combination of an increased gain and some even minor dysfunction of the inner ear. At the time the model was first presented (Jastreboff, 1990), this concept was not supported by most data in the published literature, which claimed that only a small percentage (0.3%) of tinnitus patients exhibited decreased sound tolerance (Vernon, 1987). When we started measuring sensitivity to external sounds in our population of tinnitus patients, both in the USA and the UK, it transpired that about 40% of patients showed a degree of increased sensitivity to environmental sound (Hazell & Sheldrake, 1992; Jastreboff, Gray & Gold, 1996). As our practice of TRT progressed and we systematically evaluated patients for any decreased sound tolerance, it became increasingly evident that a high proportion of patients (25–30%) were seeking help because of hyperacusis, which may be even more of a problem to them than their tinnitus. Decreased sound tolerance was even less recognized than tinnitus, and patients frequently were blaming tinnitus for the problems they experienced, being unaware of the true reason.

2.4 Relationship of tinnitus to hearing loss

Hearing loss and tinnitus are only indirectly related.

Another important implication of this model is that hearing loss and tinnitus are only indirectly related. As hearing loss increases sensitivity of neurons within the auditory pathways (Gerken *et al.*, 1985), it could be expected that people who have some hearing loss might be more prone to tinnitus. This observation is confirmed by epidemiological data. As people with sensory hearing loss typically have damage to OHC, there is a higher likelihood of an imbalance between IHC and OHC systems. The incidence of tinnitus in a hearing-impaired population is approximately twice that of the population with normal hearing (Coles, 1987). At the same time, 20% of people with tinnitus have normal hearing (Davis & El Refaie, 2000) and 27% who are totally deaf do not have any tinnitus (Hazell *et al.*, 1995). Furthermore, if we compare average audiograms from a population of people attending a tinnitus clinic with those from a normative population study, the audiograms are basically identical (Hazell & McKinney, 1996). The finding that hearing loss, specifically of a high frequency in the worse ear, approximately doubles the risk that a person will have tinnitus predicts that the ratio of tinnitus patients with normal hearing to

those without hearing loss should be about 2 to 1. The finding in clinical practice that about 30% of tinnitus patients do not have a hearing loss fits this prediction. The hypothesis of discordant dysfunction provides an explanation for this apparent paradox. If tinnitus emergence depends on a dysfunction of the OHC system, then this dysfunction can be very localized, very discrete, not reflected in the audiogram and not noticed by the patient.

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In the vast majority of those affected, tinnitus is not related to any on-going pathological process. It is not an indication of progressive hearing loss, nor a predictor that a person is more susceptible to hearing loss. In only a *very* small percentage will tinnitus be related to some medical problem, such as Ménière's Syndrome (disease), otosclerosis or compression of the auditory nerve by a slowly growing benign tumor. Such conditions should be screened for at the initial routine evaluation of a patient by the otolaryngologist and treated appropriately.

2.5 Phantom perception

The perception of tinnitus results from the detection of an activity within the auditory pathways without an external sound corresponding to the tinnitus. Consequently, tinnitus perception does not obey the same rules as perception of external sounds.

The very real perception of tinnitus results from the detection of modification of spontaneous activity within the auditory pathways, and there is no external sound corresponding to the tinnitus. In other words, tinnitus is a phantom sound, similar to phantom pain or the phantom limb phenomenon (Jastreboff, 1990; Moller, 1997; Muhlnickel *et al.*, 1998). Consequently, perception of tinnitus does not obey the same rules as perception of external sounds; for example, its suppression is governed by totally different principles. Even if the signal itself is very weak, it may be heard in the presence of high levels of environmental sound. It can be persistent and irritating, simply because it is an unusual signal, and unlike perception generated by external sounds.

2.6 Natural habituation

More than three-quarters of people who experience tinnitus naturally habituate to it.

Notably, more than three-quarters of people who experience tinnitus can naturally habituate to it (Davis & El Refaie, 2000; McFadden, 1982). The recognition of habituation can be linked to Pavlov (Konorski, 1948):

When a stimulus is repeatedly presented without being followed by any arousal-producing consequences, these effects are gradually attenuated and some of them may be eventually totally abolished. This phenomenon, originally denoted by Pavlov as “extinction of orientation reaction,” is now usually called “habituation.”

More recent wording offers the same meaning (*Stedman’s Concise Medical Dictionary*, 1997):

The method by which the nervous system reduces or inhibits responsiveness during repeated stimulation

or (*American Heritage Dictionary*, 1994):

The decline of a conditioned response following repeated exposure to the conditioned stimulus

Adaptation involves a peripheral sensory organ while habituation occurs within the central nervous system.

Contrary to adaptation, which involves a peripheral sensory organ, habituation occurs within the central nervous system.

Habituation of tinnitus means that the tinnitus-related neuronal activity is blocked from reaching the limbic and autonomic nervous systems and consequently there are no negative reactions to tinnitus (habituation of reaction). Moreover, the auditory system is capable of blocking this tinnitus-related neuronal activity, preventing it from reaching higher cortical areas and thus being perceived (habituation of perception).

Habituation of tinnitus means that the tinnitus-related neuronal activity is blocked from reaching the limbic and autonomic nervous systems and consequently there are no negative reactions to tinnitus (habituation of reaction). Moreover, the auditory system is capable of blocking this tinnitus-related neuronal activity, preventing it from reaching higher cortical areas and thus being perceived (habituation of perception). Individuals who naturally habituate are unaware of the presence of tinnitus, except at times when they consciously focus their attention on it. The role of natural habituation was noticed by Stephens, Hallam & Jakes (1986). These authors, however, did not propose a specific or effective protocol for inducing and sustaining habituation, which is necessary for clinical success of a treatment. Such a protocol, aimed at inducing and sustaining habituation, is an integral part of TRT.

2.7 The process by which tinnitus becomes a problem

A novel sound induces a new pattern of activity within the auditory pathways and subawareness centers allowing this activity to reach the cortex. After reaching the highest cognitive centers, sound will be perceived and further evaluated.

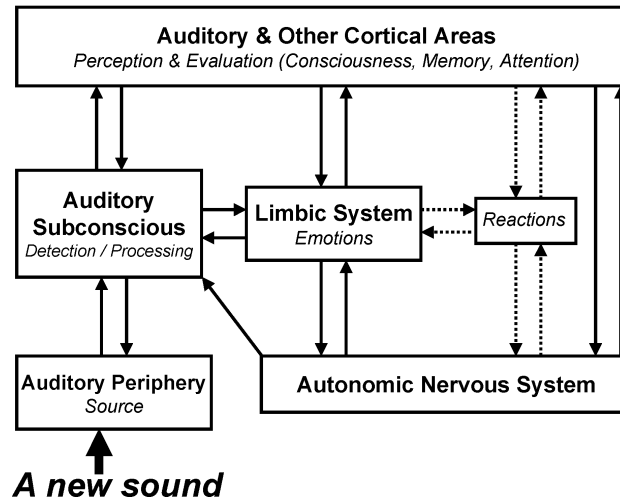


Figure 2.8 Activation of various systems evoked by a new sound. In this and subsequent figures, the thickness of the arrows and darkness of the boxes represent the strength of activation. Note a low-level activation evoked by a new sound.

To understand why the perception of tinnitus might create a problem, we can start by studying what is happening in the brain when it is exposed to any new sound. The novel sound induces a new pattern of activity within the auditory pathways. This activity in groups of nerve fibers will increase and become regular, rather than being random as nerve activity is in the absence of signal. While the pattern of activity is new and not previously experienced, subawareness centers allow this activity to reach the cortex. These centers are also responsible for our selective hearing by blocking unimportant auditory information. The new sound-induced pattern will be compared with that stored in memory patterns representing other sounds. If this pattern does not find a match in auditory memory, then it is passed to the highest cognitive centers, where it will be perceived and evaluated further (Fig. 2.8).

Sounds can be classified in three general categories: neutral (not significant), having some positive (pleasant) meaning, and having a negative (unpleasant) association or meaning. During this process, with every new sound, the limbic and the autonomic nervous systems are activated to some extent (Fig. 2.8). This results in an orientation reaction (startle reflex⁵) in which the head may be turned in the direction of a sound to learn more about it. Meanwhile, our autonomic nervous system is preparing our body to an appropriate reaction should it be needed.

⁵ The startle reflex involves a quick, involuntary movement, frequently contraction of the limb and neck muscles, in response to some sudden and unexpected stimulus.

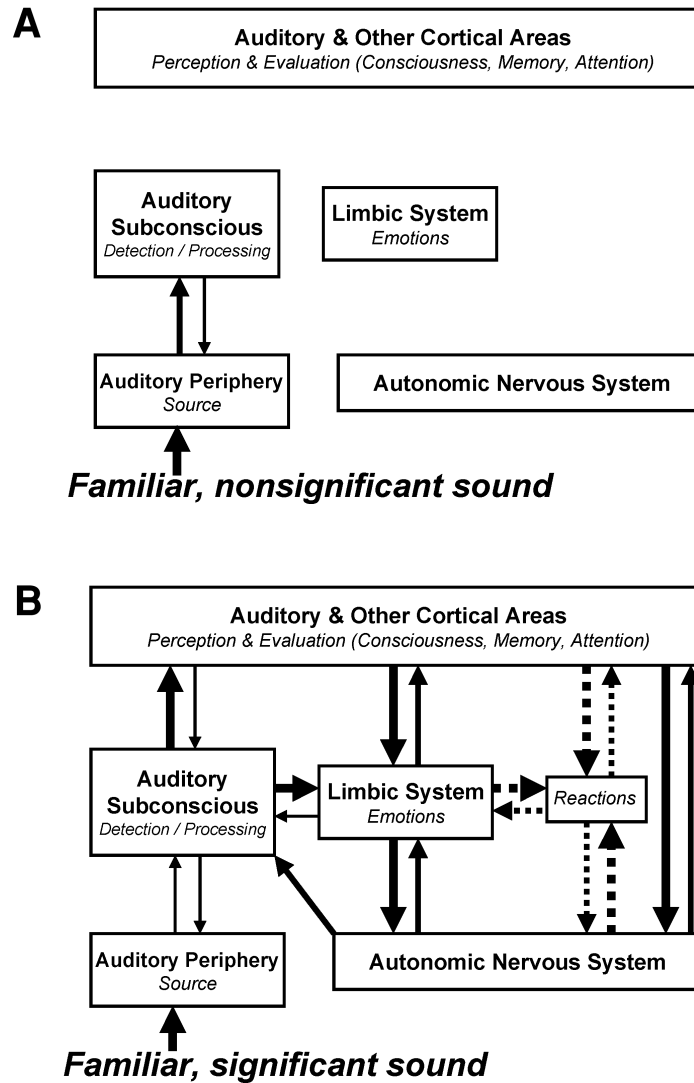


Figure 2.9 Activation evoked by a new sound. (A) An unimportant non-significant, familiar sound does not activate the limbic and autonomic nervous systems nor the higher cortical areas responsible for sound awareness. These signals are not inducing reactions (habituation of reaction) and are not perceived (habituation of perception). (B) By comparison, a familiar, significant sound will result in a high level of activation of the limbic and autonomic nervous systems and of higher level cortical areas.