Tinnitus

Tinnitus, commonly referred to as “ringing in the ears”, is prevalent in 20% of any given population; 3 to 7% of those with tinnitus seek clinical intervention due to its debilitating effect on their lifestyle.

Tinnitus may also be accompanied by two additional conditions: hyperacusis and misophonia. Hyperacusis is a condition characterized by unusually strong behavioral responses to sound while misophonia is an extreme dislike of sound. Collectively, hyperacusis and misophonia make for a condition called *Decreased Sound Tolerance*.

Tinnitus may point to any number of potential underlying pathological conditions that must always be investigated. Tinnitus can be generated by certain anatomical structures in and/or adjacent to the ear emitting biological noise called somatosounds; vascular blood flow patterns, tumors, or benign conditions such as patent (open) eustachian tubes may generate somatosounds. However, in a great majority of cases, no identifiable pathology can be determined. This class of tinnitus is often called a “phantom auditory perception”; phantom tinnitus refers to an event that cannot be attributed to the presence of any physical acoustic stimulus.

While the cochlea plays a role in phantom auditory perception, a prominent theory called the Neurophysiological Model for Tinnitus suggests that clinically significant tinnitus *primarily* involves ascending (higher level) auditory and central nervous system centers and that the inner ear is involved only *secondarily*.

Generally, the model suggests that persons significantly affected by tinnitus at some point in time develop severe negative emotions associated with the tinnitus signal; in turn, a physiological-not psychological chain of events involving the brain’s limbic and autonomic nervous systems is activated as a defense mechanism to the signal.

Tinnitus Retraining Therapy has seen success in minimizing, if not completely eradicating, debilitating tinnitus cases. Extensive therapy protocols serve to retrain the brain’s physiological response to the troublesome tinnitus signals. This is possible due to the brain’s plasticity; humans are able to alter neuronal relays within the brain and reverse negative physiological reactions to the eliciting stimulus.

There remain many fallacies associated with tinnitus and its treatment. No single drug has been proven to systematically induce tinnitus in double-blind studies; however, a certain class of drug (benzodiazepines, i.e. Xanax, Valium, Lorazepam, etc.) may induce transient tinnitus and hyperacusis upon cessation. Caffeine can exacerbate existing tinnitus, but there is no scientific basis to eliminate “normal” caffeine intake. Aspirin does not induce or exacerbate tinnitus in small doses; one to five aspirins a day has no clinically significant

1781 S. Bell School Rd. | Cherry Valley, IL 61016
Phone: 815.332.3460 | Fax: 815.332.5175
Company Website: http://www.tkontheweb.com

If this model proves reliable, tinnitus is truly a noise born in one's brain, not one's mind!

Authored by: Robert Williams, Au.D. | Director Audiology | T K Group, Inc.

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