You May Be Losing Your Hearing As You Sleep

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The National Institute on Deafness and Other Communication Disorders (NIDCD) estimates that 30%-35% of adults over 65 years old and 40%-50% of adults over 75 years old will suffer some hearing loss. Nevertheless, it is possible to avoid hastening hearing loss as one ages. One solution may be as simple as treating a bed partner’s sleep apnea.

Two types of hearing loss that commonly occur in adults are presbycusis (age-related hearing loss) and noise-induced hearing loss. Presbycusis typically affects one’s ability to hear high-pitched sounds such as the ring of a phone, ticking of a watch, and children’s or women’s voices. Noise-induced hearing loss initially affects one’s ability to hear a certain range of high-pitched sounds (e.g., 2.0-4.0 kilohertz [2,000-4,000 cycles/second]) but a person is still able to perceive other high pitched sounds but to a lesser degree than normal. Later, as noise-induced hearing loss progresses, a person becomes unable to perceive low-pitched sounds. Both presbycusis and noise-induced hearing loss involve injury to the cochlea. In presbycusis, cochlear structures are destroyed. In noise-induced hearing loss, cochlear structures are damaged but not destroyed.

The cochlea is a tapered cone-like structure which coils upon itself giving it the appearance of a sea shell. The basilar membrane runs the length of the cochlea. It supports the organ of Corti which contains various types of receptor cells involved in the neurological aspect of hearing: inner hair cells, outer hair cells, inner and outer phalangeal cells, border cells, and Hansen’s cells. Sound waves cause cochlear fluids (perilymph and endolymph) to flow back and forth within the cochlea. The hair cells, which project into endolymph, sway in conjunction with the fluid’s flow. Each movement of the hair cells transmits a signal to the cochlear nerve and from there the signal travels to the cochlear nuclei in the brain to be interpreted as sound.

In noise-induced hearing loss, the hair cells — particularly the outer hair cells — move about excessively in response to loud noise. This causes them to swell, weaken, and twist. In this condition, the hair cells can not transmit their signals accurately to the cochlear nerve resulting in diminished hearing.

In presbycusis, the hair cells die off, the organ of Corti atrophies, the basilar membrane thickens, and the stria vascularis (a layer of vascular tissue lining the cochlear duct that secretes endolymph) atrophies. Scientists are not sure why these changes occur but have looked to genetics, diet, and external factors (e.g., ototoxic drugs, noise) as a cause.

A normal healthy human ear begins to perceive sound at 0 decibels (dB). Loud noise begins to cause pain at 125 dB (about the loudness of a car horn if you were standing less than 4 feet away). Damage to inner ear structures begins at 160 dB (about the loudness of a jet engine at less than 100 feet). Destruction of inner ear structures occurs at 180 decibels. (The loudest sound possible to measure is 194 dB.) The government agency Occupational Safety and Health Administration (OSHA) recommends a person wear ear protection (such as ear plugs) at 85 dB and requires workplaces to provide ear protection to workers at 90 dB.

A light snorer snores at about 38 dB. Most snorers snore at about 60-70 dB. Very loud snorers can snore as loud as 80 decibels — nearly the level at which OSHA recommends ear protection. Even though light and moderate levels of snoring are below the level considered damaging to the ear, snoring may still play a role hearing loss.

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In 1973, Yugoslavian scientist M. Prazic was the first to examine whether snoring contributed to hearing loss in snorers. He expected that snorers would have an increased incidence of presbycusis since snorers are exposed to loud noise repeatedly for many years. He examined the audiograms of 17 snorers all of whom were 60 years or older and found that each had presbycusis. He concluded that their snoring had contributed to their presbycusis.

Only within the last six years has Prazic’s experiment begun stimulating other researchers to examine snoring’s effect on hearing loss. In 1999, Victor Hoffstein et al. looked for an association between snoring and presbycusis. They were to conclude that no association existed.

Hoffstein’s study involved 219 subjects; 182 of these were snorers. They compared each subject’s hearing threshold (i.e., the lowest signal a person can hear) with his maximum snoring noise level. A subject had a hearing loss if he could not hear high-pitched sounds greater than 4,000 cycles/second (4.0 kilohertz). Hoffstein et al. found that the hearing threshold of the subjects as a group remained in the normal range (i.e., below 4.0 kilohertz) throughout the snoring noise range (50-100 dB). When they compared the hearing threshold of mild snorers with that of loud snorers, they found no statistical difference in threshold. Hoffstein et al. concluded that snoring does not contribute to presbycusis since the hearing threshold did not increase with increasing snoring loudness (as would be expected if snoring were causing hearing loss) and since there was no difference in hearing threshold between loud and mild snorers.

However, noise-induced hearing loss caused by snoring may be a different matter. Noting that studies such as those of Prazic and Hoffstein focused only on presbycusis, Canadian doctors Maya G. Sardesai et al. examined whether snoring could cause noise-induced hearing loss. Of particular interest to them was the impact of snoring on a bed partner’s hearing.

They used four couples (i.e., eight subjects) in their study. Each couple was composed of a “snorer” and a “non-snorer.” All eight participants were given a behavioral audiogram and an otoacoustic emissions (OAE) test. A behavioral audiogram tests the function of a person’s hearing. It is used to determine speech perception (i.e., word recognition), hearing threshold, and the function of the auditory nerve and brain pathways involved in hearing. An OAE test measures the cochlea’s ability to emit a signal (i.e., the otoacoustic emission) in response to a test signal. Hearing loss has occurred if the cochlea does not emit a signal in response to a 30 dB test signal.

Like Hoffstein, Sardesai et al. could find no correlation between snoring noise and hearing loss in the snorers. All of the bed partners, on the other hand, had high frequency noise-induced hearing loss in the ear next to the snorer during sleep. Because of this consistent pattern, Sardesai et al. concluded that loud snoring can result in noise-induced hearing loss in the bed partners.

Snorers often suffer from sleep apnea (the cessation of breathing during sleep) which occurs when pharyngeal tissue collapses into and blocks the airway. As a result of air blockage, a person will abruptly arouse for a few seconds to take some deep breaths. It is during the arousal when snoring occurs. During snoring, pharyngeal tissue partially blocks the airway and flutters with each breath.

Sleep apnea can have potentially serious consequences for a sufferer. Sleep apnea suffers have an increased risk of gastrointestinal reflux disease; an increased risk of cardiovascular problems (e.g., hypertension, stroke, congestive heart failure); and increased difficulty with controlling obesity and associated obesity problems. Additionally, frequent nocturnal arousals from sleep apnea can result in excessive sleepiness during the day which in turn can jeopardize one’s ability to function at work or in social situations — a person may find himself inadvertently dozing at work, in social settings, or at dangerous times such as while driving. Sleep apnea treatment can counteract these consequences and, as a double benefit, protect the hearing of a bed partner.

References

About the Author
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