The two lateral semicircular canals, one located in each inner ear, alert a person to the fact that his upright head is experiencing angular acceleration. Within the ampulla of the canal are several so called hair cells. The cilia of these cells protrude into the lumen of the ampulla where they are encased in a mass of jelly-like material (the cupula) which is attached to the opposite wall of the canal. As the head accelerates, the cilia are bent by an inertial force of the cupula and the viscous liquid in the canal lumen. The bending of the cilia excites hair cells which in turn excite afferent neurons; these then alert the brain that a change of position of the head has occurred. Similar events occur when the head stops moving. The result of a strong hair cell stimulus to the brain is a rapid eye movement, call nystagmus, a feeling of dizziness and disorientation, and a possibility of nausea and vomiting.

Normal hearing is in the range between the frequencies of 20,000 to 16,000 Hz with the optimal sensitivity for most people between the frequencies of 500 to 6000 Hz.

Mechanism to Produce the Desired Effects

Because the end organs for acoustic and vestibular perception are so closely related, intense acoustic stimulation can result in vestibular effects. The hypothesis is that the sound of normal intensity produces oscillations of the endolymph and perilymph, compensated for by oscillations of the round window. High intensity sound produces eddy currents, which are localized rotational fluid displacements. High intensity sound can also produce nonlinear displacement of the stapes, causing a volume displacement, the result of which can be a fluid void in the labyrinth. To fill the void, fluid may be displaced along the endolymphatic duct and/or block capillary pathways, which, in turn, could stimulate vestibular receptors. Stimulation of the vestibular receptors may lead to nausea and vomiting if the sound pressure level is high enough. Conclude that both eddy currents and volume displacement serve to stimulate vestibular receptors in humans, when exposed to high levels of noise.

One study found nystagmum in guinea pigs exposed to high levels of infrasound via stimulation of the vestibular receptors. However, the same lab was unable to produce nystagmus in human subjects at 5- and 10-second exposures to a pure tone at 135 dB, broadband engine noise, or a 100 Hz tone at 120 dB, pulsed three times/s or 2 minutes. The same research was unable to elicit nystagmus at levels up to 155 dB, and also equally unable to produce nystagmus using infrasound levels of 112-150 dB in guinea pigs, monkeys, and humans. However, research with audible components in the sound spectrum with guinea pigs and monkeys produced nystagmus. Other researchers report other vestibular effects in addition to nystagmus at the following thresholds: 125 dB from 200-500 Hz, 140 dB at 1000 Hz, and 155 dB at 200 Hz. Decrements in vestibular function occur consistently for broadband noise levels of 140 dB (with hearing protection).

Human subjects listened to very high levels of low-frequency noise and infrasound in the protected or unprotected modes. Two-minute duration as high as 140 to 155 dB produced a range of effects from mild discomfort to severe pressure sensations, nausea, gagging,