Blunt force trauma to the neck frequently results in damage to the cervical spine, either through direct assault or from whiplash. Whiplash most commonly occurs in rear-end automobile crashes but may also result from sport injuries, fall accidents, and violent head shaking. This type of trauma may cause audiovestibular system damage and additional neurosensorial disorders (Pain Pract 2008;8[1]:65). The symptoms of neck trauma may be transient and resolve in a few weeks to months, or they can develop into ongoing neck pain, headaches, hearing loss, tinnitus, or vertigo, with other injuries that can last for years.

Whiplash injury is a result of rapid acceleration and deceleration of the head. The head is first extended backward (hyperextension) and is then whipped forward (hyperflexion; Int Tinnitus J 1995;1[2]:105). Even at 5 mph, which is bumper car speed, the positive acceleration of the head is 8.2 Gs (J Forensic Leg Med 2009;16[2]:53). The reentry of Apollo 16 was measured at 7.19 Gs.

This backward-and-forward action results in damage to cervical microstructures, including ligaments, facet joints, discs, muscles, and nerves (Man Ther 2004;9[2]:60). While other mechanisms are likely involved, vertebrobasilar artery insufficiency or sudden changes in blood flow hemodynamics; labyrinthine, cerebellar, or brain stem concussion; and possibly inner ear vascular damage are primarily responsible for the audiovestibular complaints.

Whiplash-associated disorders can be classified into five categories, ranging from no neck pain or physical signs to significant neck injury with fracture or dislocation (Man Ther 2004;9[2]:60). Risk factors for chronic impairment after whiplash injury include preexisting degenerative disc disease, older age, female gender, head position at the time of the accident, concomitant head trauma, and prior psychological distress.

In a study by Segal et al that selected 227 cases of neck trauma without other conditions such as noise exposure, tinnitus was reported within the first three months in 55 percent of cases, and hearing loss in 52 percent (Otol Neurotol 2003;24[5]:734). Any type of hearing loss is possible, but the typical pattern was a high-frequency sloping loss in one or both ears, with a noise-induced notch.

In another piece of research, 60 percent of subjects tested within 15 days of whiplash injury had ongoing complaints of vestibulopathy, including cervical vertigo, benign paroxysmal positional vertigo, or ocular motor system abnormalities, especially when neck and associated minor head trauma were present (Acta Otorhinolaryngol Ital 2011;31[6]:378). In cases of trauma, the high mental burden related to such an event often produces a more severe tinnitus reaction compared with non-trauma cases (PLoS One 2012;7[9]:e45599).

Even without head trauma, blunt force neck injury may result in physiological changes and psychological distress. More severe symptoms and long-term consequences are common for trauma patients who develop hearing loss, tinnitus, and hyperacusis. I always treat the hyperacusis first in these cases. Once it is manageable, I start with amplification and change the sound therapy regimen for tinnitus retraining. I am careful, however, when selecting compression, output, and noise reduction characteristics to avoid reintroducing hyperacusis and stimulating the tinnitus.

The use of hearing instruments alone does not provide sufficient care in this situation because amplification without a specialized treatment program may misdiagnose additional symptoms, delay recovery, and misdirect the patient (Laryngoscope 2003;113[5]:821). Patients who have debilitating tinnitus and hyperacusis are special cases, and mis-handling the therapy can be disastrous to the patient’s quality of life.

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