

Understanding Labyrinthine Concussion

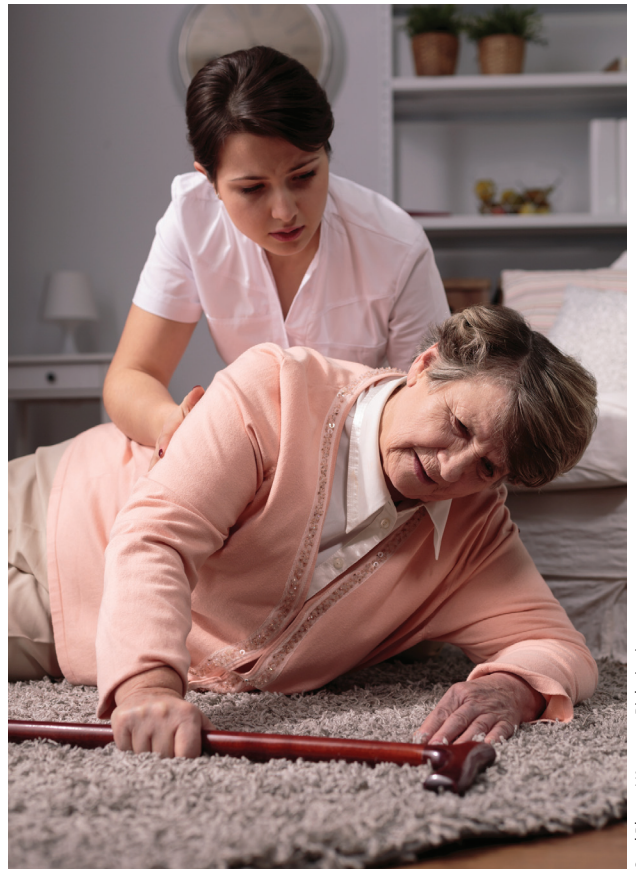
By Dennis Colucci, AuD, MA

Labyrinthine concussion is defined as damage to the inner ear due to head trauma with no well-defined injury or skull fracture, resulting in sensorineural hearing loss with or without vestibular symptoms (*Korean J Audio.* 2013;17[1]:13). Because of the damage to the inner ear structures, tinnitus is also frequently reported. It has been suggested that designations for cochlear and vestibular concussions should be prescribed to delineate the organs involved in these different conditions. A labyrinthine concussion is typically a unilateral injury to the ipsilateral side, although it can be bilateral, and in rare cases, its symptoms and test result can present as contrecoup. Symptoms of hearing loss, dizziness, and tinnitus are usually reported to occur immediately, within hours after the injury, or a couple of days later.

CAUSES OF THE CONCUSSION

Although the exact cause of this concussion is not clear, it is assumed that the injury occurs because of the acceleration-deceleration movement of the membranous labyrinth against the bony labyrinth, or the compression and vibration forces generated by a blunt force trauma. It is suggested that these actions result in "hemorrhaging sites and microcirculation disturbances in the cochlea, destroying the sensory epithelium due to rupture of vessels in the membranous labyrinth" (*Case Rep Otolaryngol.* 2016;2016:2123182). Furthermore, there can be structural changes that create avulsions or through the recovery process, sclerosing labyrinthitis that can be seen on MRI (*Neuroradiol J.* 2013;26[1]:52). In some cases, a labyrinthine concussion may also result in posttraumatic endolymphatic hydrops.

Changes in hearing or the observance of nystagmus must be present to make the diagnosis of labyrinthine concussion with any reasonable degree of certainty. The hearing loss may range from mild to profound with greater hearing loss in the high frequencies. In some cases, what would appear to be noise-induced hearing loss with a distinctive notch at approximately 4,000 Hz and recruitment may be observed. If a vestibular component is suspected, vision-denied, recordable nystagmus is present, initially beating towards the irritated ear. Nystagmus is typically horizontal or torsional and direction-fixed. However, during recovery, nystagmus changes direction, with the fast phase beating towards the intact ear. Positional and spontaneous nystagmus would be consistent with Ewald's Second Law and

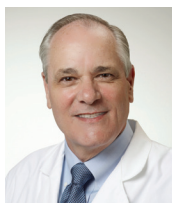


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Alexander's Law. There should be no central findings with caloric response within normal limits or a directional preponderance. Active head rotation would reveal reduced gain, with a phase lag and asymmetry. If the otolithic structures are involved, the observance of benign paroxysmal positional vertigo (BPPV) can be recorded in one or multiple semi-circular canals on Hallpike and horizontal canal testing (*Hearing Journal.* 2014; 67[7]).

Labyrinthine concussion may occur in conjunction with post-concussion syndrome or whiplash syndrome, making the differentiation more difficult to discern. The primary differences between these conditions are based on the examination, history, predispositions, diagnostic testing, and psychological state of the patient. For example, in post-concussion syndrome, the primary complaints are headaches, migraines, dizziness, and mental discomfort. These may also be the same primary complaints of patients with whiplash association disorder, with the addition of chronic neck and shoulder pain, tinnitus, and possibly also hearing loss. All three groups may present with BPPV in more than one ear canal.

Differences in how patients recover also exist. For example, patients with labyrinthine concussion have faster recovery



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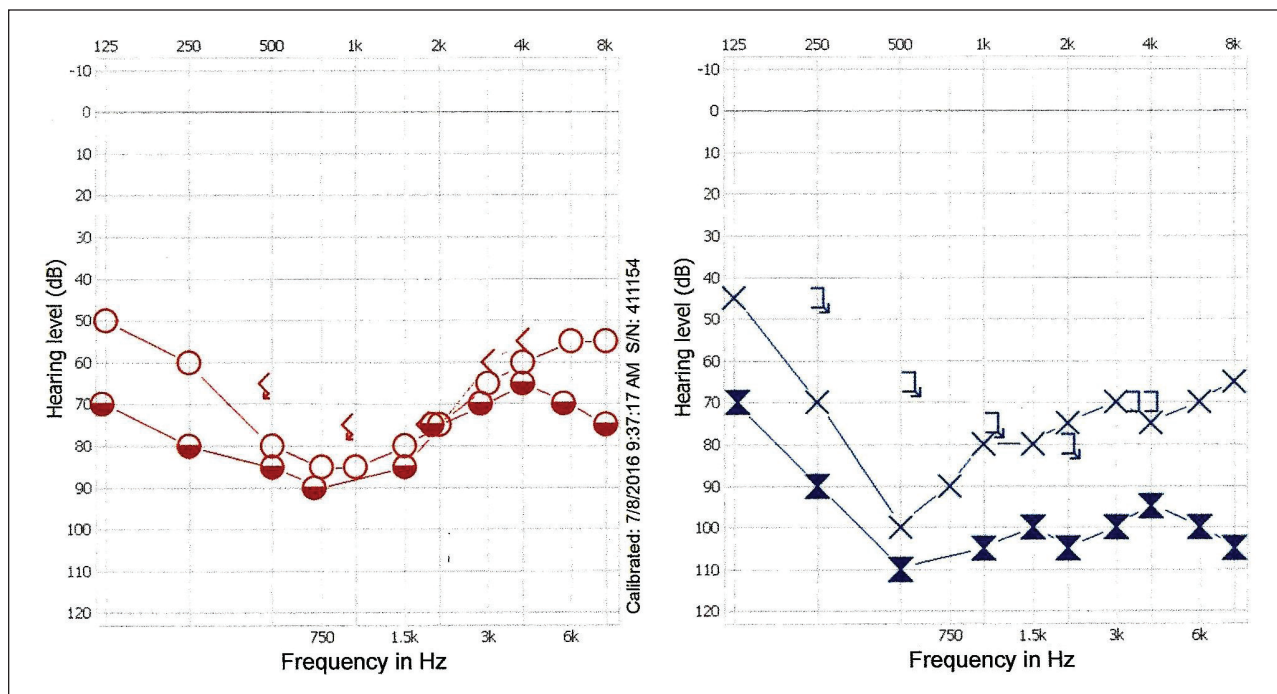


Figure 1. Pre- and post-head trauma audiograms for the 80-year-old female patient. X's and O's are pre-trauma hearing loss for the left and right ears, respectively. The hourglass and half-filled circles are post-trauma audiograms at one year.

times compared with those who are syndromic because of established comorbidities.

The Centers for Disease Control and Prevention reported that about 800,000 people a year are seen in emergency rooms for falls, resulting in bone fractures or head trauma (CDC, 2017). Among older adults aged 65-75 years without major health or prior balance problems, 25-35 percent report a dangerous fall annually (WHO, 2007). For those over 75 years of age, the rate of falls increase to 32-42 percent. In the senior population, falls are the number one cause of head trauma. In some of these cases, labyrinthine concussions can result in severe to profound hearing loss requiring cochlear implantation.

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CASE HISTORY

The patient is an 80-year-old woman with a history of moderate to severe sensorineural hearing loss in both ears prior to falling backwards and striking her head on the floor. At the time of the accident, the patient complained of vertigo and increased tinnitus. The patient was rushed to the emergency room where it was determined that she did not have a skull

fracture, hygroma, or hematoma. She was seen by her ENT physician who referred her to physical therapy and an audiological evaluation. She was seen for hearing evaluation within a week of the accident and mild changes in hearing were observed, bilaterally. Within a month, the hearing loss increased dramatically and by the end of the first year, she had severe to profound hearing loss in both ears (Fig. 1). Because the left ear had greater hearing loss and poor discrimination (32%), the patient received a cochlear implant. Although the patient was a candidate for bilateral implantation, she preferred to use amplification on a bimodal configuration. Her vertigo was resolved within six months following vestibular and physical therapies without recurrence at one year. The tinnitus remains at pre-concussion levels and is essentially habituated.

TIPS FOR THE AUDIOLOGIST

The occurrence of head trauma in the general population is approximately five percent. Audiologists should be aware of the greater fall risk of elderly patients and the need for follow-up hearing testing when a fall occurs, even in the absence of vestibular complaints. In some cases, a labyrinthine concussion may result in post-traumatic endolymphatic hydrops, and because a differential diagnosis can be difficult, the evaluation may include testing for a post-traumatic perilymphatic fistula, superior canal dehiscence, or an eight-nerve injury. When treating patients with labyrinthine concussion, the objectives are to identify changes in hearing, treat any hearing loss and tinnitus, extinguish BPPV if present, and ensure that the vestibular impairment is centrally compensated and without any symptoms. 