

**American College of Radiology
ACR Appropriateness Criteria®**

Clinical Condition: **Vertigo and Hearing Loss**

Variant 1: **Sensorineural hearing loss, acute and intermittent vertigo.**

Radiologic Procedure	Rating	Comments	RRL*
MRI head without and with contrast	8		None
MRI head without contrast	7	High resolution internal auditory canal imaging.	None
CT head without contrast	6	For possible cholesteatoma with labyrinthine fistula. Consider thin section through temporal bone.	Low
CT head without and with contrast	3		Low
X-ray tomography skull	1		Min
X-ray skull	1		Min
CT cisternography head air/contrast	1		Low
<u>Rating Scale:</u> 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

Variant 2: **Sensorineural hearing loss, no vertigo.**

Radiologic Procedure	Rating	Comments	RRL*
MRI head without and with contrast	8		None
MRI head without contrast	7	High resolution internal auditory canal imaging.	None
CT head without contrast	5	Consider thin section through temporal bone.	Low
CT head without and with contrast	4		Low
X-ray skull	1		Min
X-ray tomography skull	1		Min
CT cisternography head air/contrast	1		Low
<u>Rating Scale:</u> 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

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Clinical Condition:**Vertigo and Hearing Loss****Variant 3:****Conductive hearing loss, rule out petrous bone abnormality.**

Radiologic Procedure	Rating	Comments	RRL*
CT head without contrast	8	Consider thin section through temporal bone.	Low
CT head without and with contrast	5		Low
MRI head without and with contrast	5		None
MRI head without contrast	5	MR may be helpful if dural extension is suspected.	None
X-ray skull	1		Min
X-ray tomography skull	1		Min
CT cisternography head air/contrast	1		Low
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

Variant 4:**Episodic vertigo, new onset (hours to days).**

Radiologic Procedure	Rating	Comments	RRL*
MRI head without and with contrast	7		None
MRI head without contrast	6		None
MRA head	6		None
CT head without and with contrast	5		Low
CTA head	5		Low
CT head without contrast	4	Consider thin section through temporal bone.	Low
X-ray skull	1		Min
CT cisternography head air/contrast	1		Low
X-ray tomography skull	1		Min
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

Variant 5:**Vertigo, no hearing loss, normal neurological exam.**

Radiologic Procedure	Rating	Comments	RRL*
MRI head without and with contrast	8		None
MRI head without contrast	7		None
CT head without contrast	5	Consider thin section through temporal bone.	Low
CT head without and with contrast	4		Low
X-ray tomography skull	1		Min
CT cisternography head air/contrast	1		Low
X-ray skull	1		Min
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

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Clinical Condition: **Vertigo and Hearing Loss**

Variant 6: **Total deafness, cochlear implant candidate, surgical planning.**

Radiologic Procedure	Rating	Comments	RRL*
CT head without contrast	9	Consider thin section through temporal bone.	Low
MRI head without contrast	5		None
MRI head without and with contrast	5		None
CT head without and with contrast	3		Low
X-ray tomography skull	1		Min
CT cisternography head air/contrast	1		Low
X-ray skull	1		Min
<u>Rating Scale:</u> 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

Variant 7: **Fluctuating hearing loss, history of meningitis or to rule out congenital anomaly.**

Radiologic Procedure	Rating	Comments	RRL*
CT head without contrast	8	Consider thin section through temporal bone.	Low
MRI head without contrast	7		None
MRI head without and with contrast	7		None
CT head without and with contrast	4		Low
X-ray tomography skull	1		Min
X-ray skull	1		Min
CT cisternography head air/contrast	1		Low
<u>Rating Scale:</u> 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

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VERTIGO AND HEARING LOSS

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Summary of Literature Review

Dizziness and Vertigo

Dizziness is a common clinical complaint that accounts for 1% of visits to U.S. office-based physicians. Vertigo is a form of dizziness in which there is an illusion of movement (rotation, tilt, or linear translation). The mechanism for vertigo is an imbalance of tonic vestibular signals. Thus, vertigo is a hallucination of movement and is a symptom of a disturbed vestibular system [1-3].

The complete vestibular system comprises the end organs in the temporal bone, the vestibular components of the VIIIth cranial nerve, and the central connections in the brainstem. The end organs in the temporal bones are the cristae of the three semicircular canals that respond to movement of the head and the macula of the utricle, which records the position of the head. The semicircular canals record dynamic actions and the utricle records static function. Vertigo is subdivided into peripheral vertigo (due to failure of the end organs) or central vertigo (due to failure of the vestibular nerves or central connections to the brainstem and cerebellum) [2,4].

Benign Positional Vertigo, Ménière's Disease, and Peripheral Vestibular Disorders

Patients with benign positional vertigo describe episodic vertigo lasting less than a minute, brought on by movements of the head, and without other associated symptoms. There are no radiological findings in patients with benign positional vertigo [1,4].

In Ménière's disease, paroxysmal attacks of whirling vertigo are usually accompanied by nausea and are

transient, lasting a few hours but not days. The severe episodic vertigo is accompanied by tinnitus, fluctuating hearing loss, and a feeling of fullness in the affected ear or ears. Typically, hearing decreases and tinnitus increases during the attack. Hearing may improve between attacks in early stages of the disease. Generally, the hearing loss begins unilaterally and affects the lower frequencies primarily; mid and high frequencies are affected in later stages of the disease [1,2,4].

Ménière's disease is most common in middle age and may become bilateral in up to 50% of the affected patients. The etiology of Ménière's disease is a failure of the mechanism regulating the production and disposal of endolymph, resulting in recurrent attacks of endolymphatic hydrops. Since the endolymphatic duct and sac are the sites of resorption of endolymph, these structures play an important role in the pathogenesis of endolymphatic hydrops. The success of various surgical procedures in relieving Ménière's disease symptoms has led to great interest in using computed tomography (CT) or magnetic resonance imaging (MRI), or both, to evaluate the vestibular aqueduct, endolymphatic duct, and sac [4-7].

Unfortunately, there is no unanimity on the value of imaging in cases of Ménière's disease. Some investigators have used CT or MRI to predict results of shunt surgery, based on showing patency of the vestibular aqueduct [3,5]. Other investigators, however, report that the size, shape, and patency of the vestibular aqueduct are of no value in predicting surgical results in shunt procedures or in predicting occurrence of bilateral disease [4]. MR imaging, with its ability to detect the endolymphatic duct and sac separate from the bony vestibular aqueduct, may offer more useful information than CT [5]. The value of CT and MRI rests in their ability to rule out associated infectious or neoplastic disease [2,4,8,9].

Vestibular neuritis is a clinical diagnosis based on an aggregate of symptoms. The disease is characterized by an acute onset of severe vertigo, lasting several days, followed by gradual improvement over several weeks. Hearing is typically unaffected. The history includes onset of vertigo following an illness such as an upper respiratory infection. Most patients become completely symptom free following resolution of the primary disease [4,10]. Vestibular labyrinthitis is similar, because the disease presents with the acute symptoms of vertigo but is always associated with hearing loss. Labyrinthitis is usually viral in origin but may result from acute or chronic bacterial middle ear infections. Unlike viral labyrinthitis, labyrinthitis associated with suppurative ear disease may progress to partial or complete occlusion of the lumen of the affected labyrinth [2,4]. Early on, the

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obstructed lumen may be detected on MRI because of loss of the signal intensity of the fluid contents. Later on, more complete obliteration of all the labyrinthine structures occurs, with an end result of labyrinthitis obliterans, which is readily diagnosed on high resolution CT [11].

With MRI, there may be gadolinium enhancement of the labyrinthine structures or vestibular nerves during the acute or subacute stages of vestibular neuritis or labyrinthitis, or both [12,13]. Such results must be interpreted with care, because sudden labyrinthine dysfunction may be caused by spontaneous hemorrhage or injury, which results in abnormal signal intensities within the labyrinthine structures secondary to the blood products [14].

Superior semicircular canal dehiscence syndrome is a pathologic condition in which sound or pressure transmitted to the inner ear may inappropriately activate the vestibular system. The diagnosis of superior semicircular canal dehiscence syndrome can be made by high resolution coronal CT imaging of the temporal bones [15-17].

Sound-induced vertigo or nystagmus has been reported in perilymphatic fistulas, syphilis, Ménière's disease, congenital deafness, chronic otitis, and Lyme disease.

Diseases of the internal auditory canal and cerebellopontine angle are generally not characterized by severe attacks of vertigo, but rather with intermittent dizziness or periods of exacerbated dizziness, or both [1,4]. A variety of benign or malignant tumors of the petrous temporal bone, such as paragangliomas, carcinomas, or metastatic tumors, may directly involve the labyrinthine structures, causing vertigo. Such processes are readily evaluated with modern imaging techniques.

Central Vestibular Disorders

Lesions of the brainstem or cerebellum that result in central vertigo can be readily diagnosed by MRI. Vascular insufficiency in the vertebrobasilar circulation is a common cause of vertigo in patients older than age 50. Thrombosis of the labyrinthine artery or infarction of the lateral medulla from vertebral or posterior inferior cerebellar artery (PICA) insufficiency may cause severe vertigo. Subclavian steal syndrome can cause a variety of symptoms, including vertigo [2,18,19]. Such conditions can be carefully evaluated with MR angiography or conventional angiography of the posterior fossa vasculature.

A variety of other central nervous diseases may produce vertigo or dizziness. These include seizure disorders, multiple sclerosis, ataxic diseases, head injuries, or any

cause of increased intracranial pressure. Vertigo may result as a sequela of stroke, and transient ischemic attacks may present as episodic dizziness [4].

Various metabolic disorders may result in dizziness. These include thyroid disorders, hyperlipidemia, diabetes, and hypoglycemia. Autoimmune diseases or diseases that affect the proprioceptive system may cause vertigo. In many cases, the possibility of functional neurotic symptoms must be considered in patients in whom no disease can be found. Finally, cervical spondylosis is thought to cause vertigo by disc degeneration and narrowing of the disc space, which affects nearby nerves, or by osteophyte formation, which compresses the blood vessels. In such cases, CT may be helpful [2,4,11].

Hearing Loss

Hearing loss is typically classified as conductive, sensorineural, or mixed. Conductive hearing loss results from pathologic changes of either the external or middle ear structures preventing the sound waves from reaching the endolymph of the inner ear. Sensorineural hearing loss (SNHL) results from the pathologic changes of inner ear structures such as the cochlea or the auditory nerve and prevents neural impulses from being transmitted to the auditory cortex of the brain [3].

Sensorineural Hearing Loss

SNHL may be sudden, fluctuating, or progressive. Sudden SNHL is a manifestation of viral infections, vascular occlusive diseases, or inner-ear membrane ruptures [20-24]. Vertigo may be associated with these conditions, which can help define whether the lesion is peripheral or central [25]. To discriminate among idiopathic, viral infections and other causes of SNHL, auditory brainstem responses and gadolinium-enhanced MR imaging are used [20-22,26]. Patients with cochleitis or cochlear nerve neuritis typically have abnormal auditory brainstem responses and may be helped by a tapering course of oral corticosteroids [21,22]. Whether or not gadolinium enhanced MR imaging shows enhancement of the cochlear nerve or cochlea does not reliably guide corticosteroid therapy. However, some authors suggest that MRI positive sudden deafness is more difficult to cure with steroid therapy than MRI negative sudden deafness [21].

Fluctuating SNHL is difficult to evaluate. The audiometric examination would indicate the level of dysfunction, but not the likely cause. Patients who are noted to have large vestibular aqueducts (apertures greater than 4 mm), may have a congenital cause for fluctuating hearing loss [27-30]. Such patients with large vestibular aqueducts have high frequency loss more often than low frequency loss. Fluctuating SNHL due to an enlarged vestibular aqueduct appears to be more common in children and young adults, an important point in

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differentiating this disease from Ménière's disease, in which most patients are middle aged or older. Of interest is that the vestibular aqueduct of patients with Ménière's disease may be small, rather than large [2,4].

There is speculation on the causes of a sudden drop in hearing in patients with large vestibular aqueducts. Two possible causes are reflux of hyperosmolar fluid from the endolymphatic sac to the inner ear and rupture of the membranous labyrinth or a perilymphatic fistula due to transmission of intracranial pressure to the inner ear through the enlarged vestibular aqueduct. It is well recognized that patients sustaining relatively minor head trauma or who are subjected to extreme barotrauma (scuba diving) may aggravate their episodes of hearing loss. In such cases, it may be worthwhile to image the temporal bones to detect enlarged vestibular aqueducts and thus advise the patients or their parents of the dangers of contact sports or activities that entail extreme barometric pressure changes [28,29]. The imaging findings must be correlated with audiometry, because the fluctuating SNHL of patients with large vestibular aqueducts does not resemble the low frequency changes characteristic of Ménière's disease, which may also be associated with fluctuating hearing loss [28,29]. Patients with isolated large vestibular aqueducts may have a different pathophysiologic basis than patients whose large aqueducts are associated with other inner-ear malformations. Cases with complex inner ear malformations may be subject to recurrent episodes of meningitis, or the "gusher" syndrome, or both, resulting in a dead ear at the time of surgical intervention such as a stapedectomy [24,28].

Asymmetric SNHL or gradually declining unilateral SNHL is a common symptom that may be ascribed to many different pathologic processes. Initial evaluation is geared to localizing the site of the lesion, (ie, cochlear [31] or retrocochlear [32]). Most retrocochlear lesions are associated with an abnormal auditory brainstem response, which is often obtained before an imaging study. Whether auditory brainstem response testing should be eliminated, as a cost saving measure is a subject of considerable debate. It seems unlikely that clinicians will refer patients directly to MRI without at least preliminary audiometric or auditory brain response testing, or both [20,22,33,34].

Patients with retrocochlear localization should have a complete MRI study of the head in addition to the studies of the internal auditory canal and temporal bones. The MRI examination should include complete evaluation of the central nuclei in the brainstem as well as the auditory pathways extending upward into the cerebral hemispheres [35]. Whether gadolinium contrast enhancement is routinely used depends on many factors, including coil size, field of view, field strength, and pulse sequences. CT is sometimes diagnostic in lesions 1.5 cm or greater in

diameter when dedicated techniques are used, but it does not readily detect small brainstem lesions such as infarctions of demyelination [33,35-41].

In general, most coclear disorders such as otosclerosis are evaluated by high resolution CT imaging. Similarly, preoperative assessment for cochlear implants is usually best accomplished using thin section CT with reformatted multiplanar images. In patients with congenital etiologies for hearing loss, recent reports suggest that high resolution MRI is more useful for surgical planning [42].

Conductive Hearing Loss

CT is an excellent technique for demonstrating even small abnormalities of the bony structures of the middle ear. For this reason it is the modality of choice in the study of conductive hearing loss. However, not every patient complaining of conductive hearing loss requires a CT study. Established indications encompass conditions such as the complications of acute and chronic otomastoiditis, the postoperative ear following surgery for chronic otomastoiditis, the postoperative localization of prosthetic devices, and the assessment of congenital or vascular anomalies. Particularly, the precise extent of bone erosion associated with cholesteatoma is correctly demonstrated by high resolution CT. Conversely, although fistulization through the tegmen tympani of the temporal bone is usually detected by CT, the actual involvement of the meninges and veins is better assessed by MRI. MRI is also indicated when complicated inflammatory lesions are suspected to extend into the inner ear or towards the sigmoid sinus or jugular vein. Neoplasms arising from or extending into the middle ear require the use of both techniques, as their combined data provide essential information. The most important data for surgical planning concern the destruction of thin bony structures and the relationships of the lesion to the dura and surrounding vessels. Vascular imaging should be performed when there is suspicion of a paraganglioma extending into the middle ear [43].

Trauma

Temporal bone trauma has numerous manifestations, many of which are detected by current high resolution CT scanning techniques. CT is used extensively to identify fractures, ossicular dislocations, fistulous communications, hearing loss, and facial nerve injury [44].

Congenital and Childhood Hearing Loss

The ideal imaging method for children with unilateral or asymmetric sensory neural hearing loss is still controversial. Several authors suggest that all children with unilateral or asymmetric sensory neural hearing loss should have a high-resolution temporal bone CT scan and that brain and temporal bone MRI be obtained in select cases. In general high resolution CT has been shown to be

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efficacious for the preoperative workup for congenital hearing loss due to aural dysplasia, congenital ossicular anomalies, large vestibular aqueduct syndrome, congenital absence of cochlear nerve, and labyrinthitis ossificans [45-53].

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