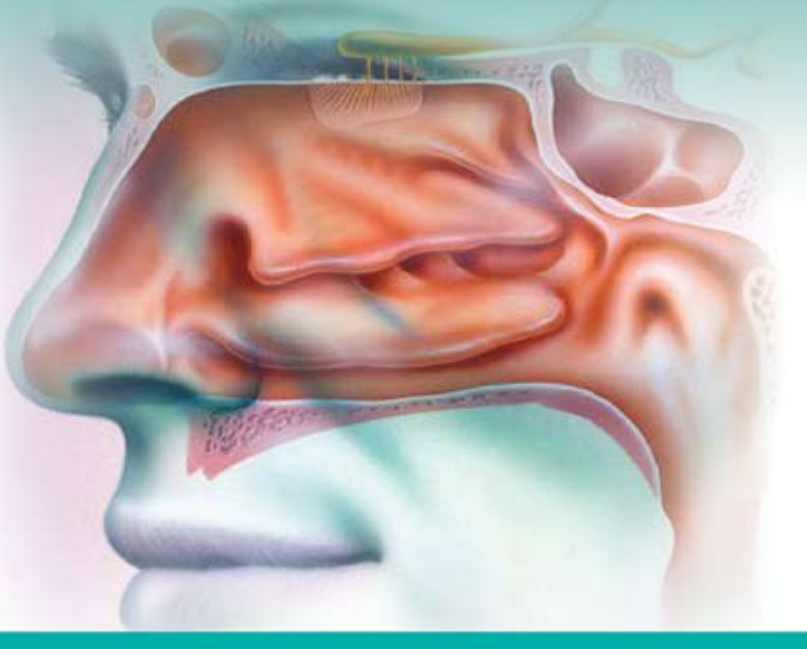
ology books, visit our website **www.jaypeebrothers.com,** for detailed information onotorhinolaryngology books

Now Available
With Complimentary App
Google play
App Store

ENT
Essentials



Elina M Toskala
David W Kennedy

Foreword
Michael J Ruckenstein

JAYPEE

ENT Essentials

Editors

Elina M Toskala MD PhD MBA
Professor
Department of Otolaryngology—
Head and Neck Surgery
Thomas Jefferson University
Philadelphia, Pennsylvania, USA

David W Kennedy MD FACS FRCSI
Professor
Department of Otorhinolaryngology—
Head and Neck Surgery
Perelman School of Medicine
University of Pennsylvania
Philadelphia, Pennsylvania, USA

Foreword

Michael J Ruckenstein MD MSc FACS

JAYPEE BROTHERS



JAYPEE BROTHERS MEDICAL PUBLISHERS
The Health Sciences Publisher
New Delhi | London

Contents

Section 1: The Ear

Section Editor: *Natasha Pollak*

1. **Ear Infections** 3
   Pamela C Roehm, Taha Ahmad Mur
2. **Hearing Loss** 17
   Kamil Amer, Pamela C Roehm
3. **Dizziness** 35
   Helen Xu, Yuan F Liu
4. **Tinnitus** 58
   Joseph Sciarrino, Michael Teixido
5. **Trauma to the Ear** 72
   Natasha Pollak
6. **Microbiology and Antimicrobial Therapy of the Ear** 86
   Purva Gumaste, Rafik Samuel

Section 2: The Face

Section Editor: *Oren Friedman*

7. **Wounds and Healing** 99
   Sarah A Gitomer, Anthony E Brissett

JAYPEE BROTHERS

8. **Facial Palsy** 109
Carly J Stewart, Andrew A Winkler

9. **Nasal Deformities** 118
Jarrod Keeler, Charles Woodard, Sam P Most

10. **Deformities of the External Ear** 127
Paul Sabini

11. **Facial Aesthetics** 141
Benjamin Marcus

12. **Facial Trauma: Soft Tissue Trauma and Bony Fractures** 145
Samuel Hahn

Section 3: Sleep Medicine

Section Editor: *Fredric Jaffe*

13 **Sleep Disordered Breathing** 159
Fredric Jaffe, Maria Elena Vega

Section 4: The Larynx

Section Editor: *Ahmed M S Soliman*

14. **Hoarseness (Dysphonia)** 179
Resha S Soni, Ahmed M S Soliman

15. **Dysphagia** 190
Gary Linkov, Barbara Ebersole, Nausheen Jamal

16. **Chronic Cough** 201
Resha S Soni, Nausheen Jamal

17. **Dyspnea** 210
Norman Chan, Ahmed M S Soliman

JAYPEE BROTHERS

Section 5: The Head and Neck

Section Editor: *Jeffrey C Liu*

18. Thyroid and Parathyroid Glands 231
Jennifer R Cracchiolo

19. Salivary Glands 253
Jennifer R Cracchiolo

20. Reconstruction 269
Christopher E Fundakowski

21. Oral Cavity and Oropharyngeal Cancer 278
Christopher E Fundakowski

22. Larynx and Hypopharynx Cancer 290
Jeffrey C Liu

23. Skin Cancer 299
Jeffrey C Liu

24. Nasopharynx Cancer 309
Jeffrey C Liu

25. Neck Dissection and Patterns of Metastasis 315
Jeffrey C Liu

Section 6: The Nose

Section Editor: *Elina M Toskala*

26. Rhinitis 323
Samir Ketan Bhandutia, Elina M Toskala

27. Acute Sinusitis 330
Samir Ketan Bhandutia, Elina M Toskala

JAYPEE BROTHERS

28. Nasal Obstruction 335
Derrick Tint, Elina M Toskala

29. Anosmia 349
Jennifer R Cracchiolo, Elina M Toskala

30. Epistaxis 357
Derrick Tint, Elina M Toskala

31. Endoscopic Sinus Surgery 366
Lori A Lemonnier

32. Septoplasty 375
Lori A Lemonnier

Section 7: Pediatric ENT

Section Editor: *Donald Solomon*

33. Embryology of Head and Neck Congenital Malforations 385
Glenn Isaacson

34. Congenital Hearing Loss 399
Gillian R Diercks, Michael S Cohen

35. Cleft Lip and Palate 415
Scott R Owen, Deborah S F Kacmarynski

36. Pediatric Tonsil and Adenoid Disorders 434
Sidrah M Ahmad, Gabriela Timoney, Donald Solomon

37. Neonatal Nasal Obstruction and Congenital Nasal Masses 442
Donald Solomon

JAYPEE BROTHERS

38. Pediatric Head and Neck Infections 454
Donald Solomon

39. Airway Foreign Bodies 480
Kristen Hurst

40. Pediatric Airway Disorders 496
George Harris, Mariah Pate

41. Pediatric Balance and Vestibular Disorders 510
Katherine M McKee-Cole, Robert C O'Reilly

42. Vascular Anomalies 524
Katherine M McKee-Cole, Douglas R Johnston

Index 539

JAYPEE BROTHERS

Chapter 3

Dizziness

Helen Xu, Yuan F Liu

Chief Complaint

"I feel dizzy, lightheaded, the room is spinning, unsteady, or off-balance".

DEFINITIONS

Vertigo: Illusion of oneself moving or one's environment moving without actual movement.

Central vertigo: Vertigo caused by disease of the central nervous system (CNS), including cranial nerve (CN) VIII.

Peripheral vertigo: Vertigo caused by disease of the peripheral vestibular system, within the membranous labyrinth.

ANATOMY AND PHYSIOLOGY OF VESTIBULAR SYSTEM

The vestibular system consists of five sensory organs within a bony labyrinth in the petrous portion of the temporal bone called the otic capsule. All vestibular end organs are lined by a membrane (membranous labyrinth) and contain endolymph. Perilymph flows between the bony and membranous labyrinths (Fig. 3.1).

1. Three semicircular canals
   a. Lateral (horizontal), superior (anterior), posterior canals
   b. Each semicircular canal is oriented at about 90° to the other two.

JAYPEE BROTHERS

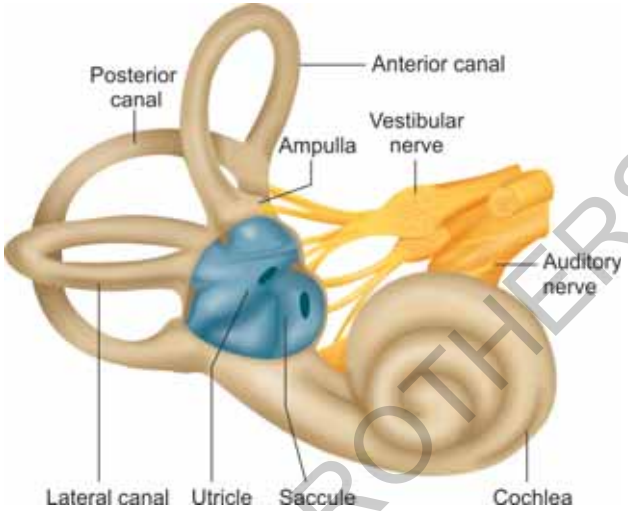


Fig. 3.1: The membranous labyrinth and vestibulocochlear nerve.

c. Each detects angular (rotational) acceleration.
d. Maximally sensitive to rotation in the plane of the canal.
e. *Functional pairs*: Each canal has a corresponding canal on the contralateral side that lies roughly in the same plane (coplanar).
   i. Left horizontal to right horizontal
   ii. Left superior to right posterior
   iii. Left posterior to right superior
f. Each canal has an ampulla, which contains the neuroepithelium with hair cells that detect motion.
g. *Vestibular hair cells*: Modified columnar epithelial cells with elongated microvilli on apical surfaces called stereocilia. Each hair cell has a single long kinocilium. Stereocilia are embedded in a gelatinous mass called cupula. When the head rotates, endolymph and cupula lag behind due to inertia, thereby deflecting stereocilia in the direction opposite of head movement. Deflection of

JAYPEE BROTHERS

stereocilia leads to depolarization or hyperpolarization of hair cells and initiates a change in the baseline rate of action potentials.

2. Two otolith organs
   a. Utricle (utriculus) and saccule (sacculus) are located in the vestibule: ovoid space within the bony labyrinth between the cochlea and the semicircular canals.
   b. Utricle is oriented in the horizontal plane and saccule in the vertical plane.
   c. Utricle and saccule detect linear acceleration. Saccule detects gravity in a standing position.
   d. Each contains a macula (analogous to ampulla).
   e. Each contains otolithic membranes (analogous to cupula), which contain embedded otoliths (otoconia). Otoliths are calcium carbonate deposits and have higher specific gravity than endolymph.
   f. Vestibular hair cells of the otolith organs work in a similar way to those of the semicircular canals.
3. Innervation
   a. The vestibulocochlear nerve (CN VIII) emerges from the brainstem at the cerebellopontine angle (CPA) and enters the internal auditory meatus (porus acusticus).
   b. Cranial nerve VIII branches into the cochlear, superior vestibular, and inferior vestibular divisions.
   c. Cell bodies of the vestibular divisions are located within the vestibular ganglion (Scarpa's ganglion), which sits within the internal auditory meatus. The vestibular ganglion is divided into superior and inferior ganglia. The superior ganglion gives rise to the superior vestibular nerve, which supplies the horizontal and superior semicircular canals, and the utricle. The inferior ganglion gives rise to the inferior vestibular nerve (IVN), which supplies the posterior semicircular canal and saccule.
   d. *Central vestibular pathway*: Cranial nerve VIII enters the brainstem at the pontomedullary junction and courses to the vestibular nuclei. Afferents to vestibular nuclei do

JAYPEE BROTHERS

not cross midline. About 70% of afferents terminate in cerebellar vermis, which contributes to coordination of head and eye movement. Efferents from the vestibular nuclei project to the spinal cord (that stabilizes head and shoulders and maintains postural balance), contralateral vestibular nuclei, thalamus (visual processing and conscious perception of vertigo). Bilateral projections from vestibular nuclei to other parts of brainstem contribute to vestibulo-ocular reflex (VOR).

e. Afferent fibers have a resting potential (tonic activity) and detect excitation or inhibition by increasing or decreasing the firing rate.
f. The CNS can compensate for acute deficits in the peripheral vestibular system, but this central compensation process can take days to weeks.
g. A person can function near normal with only one intact labyrinth.

4. Vascular supply to the vestibular labyrinth
   a. Horizontal and superior semicircular canals, most of utricle, and some of saccule are supplied by the anterior vestibular artery, internal auditory (labyrinthine) artery, and anterior cerebellar, superior cerebellar, or basilar artery.
   b. Posterior semicircular canal, most of saccule, and some of utricle are supplied by the posterior vestibular artery, vestibulocochlear artery, common cochlear artery, internal auditory (labyrinthine) artery, and anterior cerebellar, superior cerebellar, or basilar artery.
5. Vestibular reflexes
   a. *Vestibulo-ocular reflex*: It has a role in retinal image stabilization. The VOR helps to maintain gaze by eliciting eye movements that are compensatory for head movement.
   b. *Vestibulocollic reflex (VCR)*: As the head moves, the VCR stabilizes the head through the activation of neck muscles.

JAYPEE BROTHERS

COMMON CAUSES OF DIZZINESS

Benign Paroxysmal Positional Vertigo (BPPV)[1]

Most common peripheral vestibular disorder (20–40%)

1. *Epidemiology*: It occurs at all ages, with peak incidence at ages 40–60, with a female-to-male ratio being 2:1.
2. Presentation
   a. Rapid onset of vertigo after head movement (e.g. rolling in bed, looking up, and bending forward).
   b. Vertigo lasts for a few seconds, always <1 minute.
   c. It may recur over months and may cluster in time.
   d. It may follow head trauma, ear surgery, and vestibular neuritis.
   e. Half of BPPV cases are without identifiable causes.
3. Pathophysiology
   a. Dislodged otoconia deflect the cupula of a semicircular canal on movement.
   b. Posterior canal (~85%), horizontal canal (10–15%), superior canal (~2%).
   c. *Cupulolithiasis*: Otoconia are deposited and adhere to the cupula of a semicircular canal.
   d. *Canalithiasis*: Free-floating otoconia in a semicircular canal.
4. Workup
   a. *Dix–Hallpike maneuver*: It is the diagnostic gold standard and can be used to elicit posterior and superior semicircular canal diseases. Lower the patient quickly from sitting to supine with the head turned 45° to one side and end with the head extended 20° over the edge. Observe for nystagmus (up-beating and torsional toward dependent ear) with latency of a few seconds, duration <1 minute, crescendo–decrescendo intensity. Repeat with the head turned to the opposite side. The side that elicits nystagmus is the affected side. Nystagmus fatigues with repetition. Benign paroxysmal positional vertigo can be rarely bilateral in cases of head trauma.

JAYPEE BROTHERS

b. *Supine roll test*: It can elicit horizontal semicircular canal disease. Perform the supine roll test when Dix–Hallpike is negative. The patient starts supine with the head resting on the table; then turn his/her head rapidly to the right. Monitor for nystagmus toward (geotropic) or away (apogeotropic) from the downward ear. Repeat with a head turn rapidly to the left. The affected ear is the side that elicits more intense nystagmus.

5. Management of BPPV
   a. Epley maneuver (canalith repositioning procedure)
      i. Start with Dix–Hallpike position of the head extended while supine; when nystagmus resolves, slowly roll 180° (90° at a time until nystagmus subsides) so that the affected ear is up, then sit upright.
      ii. Repeat until there is no more nystagmus.
      iii. It can unintentionally convert posterior to horizontal canal BPPV. After completing the Epley maneuver, the experienced physician will test for conversion of posterior BPPV to horizontal BPPV by performing the supine roll test.
   b. Horizontal canal BPPV usually spontaneously remits in a few days.
   c. Vestibular rehabilitation (physical therapy to help with habituation, adaptation, and compensation of balance disorder) and observation are options for initial treatment if the patient cannot tolerate the repositioning maneuvers.
   d. Reassess within 1 month.
   e. Surgery is an option if BPPV is disabling and not cured by repositioning maneuvers (posterior semicircular canal occlusion).

Vestibular Migraine

Vestibular migraine or migraine-associated vertigo is the most common cause for spontaneous episodic vertigo.

JAYPEE BROTHERS

1. *Epidemiology*: All ages are affected, but migraine-associated vertigo usually starts in early adulthood. The female-to-male incidence ratio is 2:1.
2. Presentation
    a. Dizziness or vertigo lasting from 5 minutes to 72 hours.
    b. Symptoms may be triggered by head position change or visual stimulation (e.g. moving subjects in a busy mall), or may be spontaneous.
    c. It may or may not have a history of headaches.
    d. It may be associated with migrainous features: hypersensitivity to light, sound, or smell.
    e. A history of motion sickness is common.
    f. Frequently, there is a family history of migraine.
    g. It may be accompanied by bilateral sensorineural hearing loss or tinnitus.
    h. It may overlap with BPPV, Ménière disease.
3. Pathophysiology
    a. *Unclear etiology*: Neurovascular involvement
        i. *Neuronal dysfunction hypothesis*: Activation of the trigeminovascular system, cortical spreading depression, and neuronal sensitization, which can lead to triggering of pain receptors.
        ii. Pain may be due to vasodilation and stretching of pain fibers in vessel walls (less popular with new evidence).
    b. *Basilar migraine*: Migraine attributed to basilar artery ischemia.
4. Workup
    a. Diagnostic criteria[2]
        i. *Definitive*: Vestibular symptoms, current or history of migraine, and migrainous features.
        ii. *Probable*: Vestibular symptoms plus either the history of migraine or the migrainous features.
    b. Exclude other causes.

JAYPEE BROTHERS

5. Management
   a. Lifestyle/diet modifications
      i. Strict migraine diet for 6–8 weeks. If it helps, add back favorite foods one at a time.
   b. *Prophylactic*: Tricyclic antidepressants, selective serotonin reuptake inhibitors, anticonvulsants, calcium channel blockers, β-blockers.[3,4]
      i. Start with low dose, increase dose slowly, stay at the dose with good effect for 6 months, then wean off.

Ménière Disease (Idiopathic Endolymphatic Hydrops)[5]

The second most common cause of spontaneous episodic vertigo:

1. *Epidemiology*: It occurs at all ages; peak age group 30–60. Ménière disease is more prevalent in Whites and slightly more prevalent in women.
2. Presentation
   a. Sudden onset of vertigo, recurring triad of vertigo, fluctuating sensorineural hearing loss, and tinnitus or aural fullness.
   b. Atypical Ménière disease may have symptomatic overlap with vestibular migraine, and distinguishing between the two entities may be difficult.
   c. Vertigo lasts from 20 minutes to 24 hours, usually 2–3 hours.
   d. No symptoms versus disequilibrium between attacks.
   e. Clusters of attacks with remission over years.
   f. Sometimes bilateral.
   g. About 10–20% familial (strongly associated with migraine).
3. Pathophysiology
   a. Ménière disease is caused by excess endolymphatic fluid (overproduction and/or underabsorption by endolymphatic sac). Etiology is unclear. There are

JAYPEE BROTHERS

possible autoimmune, viral, ischemic, traumatic, and other infectious, congenital, or multifactorial causes.

4. Workup
   a. There is no definitive diagnostic test for Ménière disease.
   b. Refer to American Academy of Otolaryngology—Head and Neck Surgery guidelines for detailed diagnostic criteria.[5]
   c. "Definitive" Ménière disease per guideline: two or more episodes of vertigo of at least 20 minutes, audiometric-proven hearing loss, tinnitus or hearing loss, and other causes excluded.
   d. Magnetic resonance imaging (MRI) is recommended to exclude other causes of vertigo and hearing loss, such as vestibular schwannoma.
   e. *Audiometry*: Sensorineural hearing loss, typically at low frequencies.
   f. Vestibular testing
      i. *Caloric testing*: Depending on the timing of test, it varies from normal response to significant reduction or complete absence of response.
      ii. *Vestibular evoked myogenic potential*: Elevated thresholds (nonspecific) or absent response.
      iii. *Electrocochleography*: Summating potential (SP) to action potential (AP) ratio increases (threshold for diagnosis ranges from 0.35 to 0.5) in Ménière disease, but sensitivity and specificity are low.
5. Management
   a. Vertigo treatment
      i. Spontaneous improvement occurs in 60–80% of patients.
      ii. Salt restriction—a low-sodium diet is recommended.
      iii. Diuretics (not enough evidence to show efficacy).
      iv. *Symptomatic*: Vestibular suppressants (antihistamine, benzodiazepine, and anticholinergic), antiemetics, sedatives, and antidepressants.

JAYPEE BROTHERS

v. Intratympanic injection of dexamethasone or gentamicin.
   - *Dexamethasone (limited evidence on effectiveness)*: It possibly suppresses autoimmunity and can maintain residual hearing.[6]
   - Gentamicin can result in a chemical labyrinthectomy. Gentamicin has high vestibulotoxicity, but it is also cochleotoxic and can cause severe hearing loss.[7]
   - When titrated properly (e.g. gentamicin 40 mg/mL, one injection every 4 weeks, up to three injections as needed), gentamicin intratympanic injections can result in selective vestibulotoxicity with high successful control of vertigo (90%) without significant cochleotoxicity.[21]
   - Gentamicin is also shown very effective (95%) in control of drop attacks.[21]

vi. Surgery (insufficient evidence for benefit)[8]
   - Endolymphatic sac decompression (controversial efficacy).
   - Vestibular neurectomy
     - Invasive and difficult neurosurgical procedure requiring a craniotomy.
     - Up to 85–95% vertigo control with 80–90% hearing maintenance.
     - It is less favored today because of the high efficacy of chemical ablation with gentamicin.
   - Labyrinthectomy
     - It is most destructive and results in complete unilateral vestibular and hearing loss.
     - It may be offered to patients with no serviceable hearing and intractable vertigo.
     - It has higher vertigo control rate than vestibular neurectomy.
     - It is less favored today because of the high efficacy of chemical ablation with gentamicin.

JAYPEE BROTHERS

Vestibular Neuritis[9,10]

1. Epidemiology
   a. Middle age.
2. Presentation
   a. Dramatic, sudden onset of vertigo, nausea, vomiting.
   b. Spontaneous, usually horizontal nystagmus suppressed with visual fixation.
   c. It may last hours to days with gradual improvement.
   d. Balance complaints related to head movement may last for months after resolution of the acute episode.
   e. Benign paroxysmal positional vertigo during recovery period is common.
   f. It can recur over years (unusual).
   g. There is no hearing loss or focal neurologic symptoms. There is sometimes a prior or concurrent upper respiratory infection (less than half).
3. Pathophysiology
   a. Vestibular nerve inflammation.
   b. The mechanism of dizziness is unclear: possibly viral, vascular, and immunologic causes.
   c. Herpes simplex virus 1 is responsible in some cases, but there is no identifiable cause in most cases.
   d. It affects the superior vestibular nerve more frequently, possibly due to the longer and narrower canal encasing the superior nerve (more susceptible to compression); however, vestibular neuritis involving the IVN can occur.
   e. Benign paroxysmal positional vertigo may occur infrequently following disease.
   f. Bilateral disease is possible.
4. Workup
   a. Physical examination
      i. Postural instability; the patient may be leaning toward the affected ear, but can walk without falling.
      ii. Slow phase of nystagmus beats toward the affected ear (dominant excitation from the unaffected side).

JAYPEE BROTHERS

iii. Nystagmus is intensified when looking away from the affected side and attenuated when looking toward the affected side (Alexander's law).
iv. Head thrust test helps to determine which side is affected.

b. Vestibular testing
i. *Caloric testing*: Reduced response on the affected side.
ii. *Vestibular evoked myogenic potential*: Response attenuated or absent on the affected side.

c. Rule out central vertigo
i. Magnetic resonance imaging scan with thin cuts to evaluate brainstem and cerebellum if there are risk factors for stroke, neurological abnormalities, or no improvement in 48 hours.

5. Management
a. Corticosteroids (insufficient evidence for benefit).[11]
b. Supportive
i. Vestibular suppressants (antihistamine, benzodiazepine, and anticholinergic).
ii. Antiemetics.
c. Stop medications when possible to allow central vestibular compensation to take place.
d. Vestibular rehabilitation (physical therapy to help with habituation, adaptation, and compensation of balance disorder) if the patient experiences residual symptoms.

Superior Semicircular Canal Dehiscence Syndrome[12]

1. Epidemiology
a. Middle age.
2. Presentation
a. Vertigo with loud sounds, pressure changes (e.g. tragal compression, nose-blowing, Valsalva, straining).
b. Nystagmus (vertical and torsional) in the plane of the superior semicircular canal (SSC).
c. Possible autophony (increased loudness of own voice), conductive hearing loss, pulsatile tinnitus, aural fullness.

JAYPEE BROTHERS

d. Spectrum from asymptomatic to auditory only to vestibular-only symptoms.

3. Pathophysiology
   a. A type of perilymphatic fistula between the SSC and epidural space.
   b. Absence of bone over the SSC, creating a "third window" that results in abnormal movement of endolymphatic fluid.
   c. It is possibly due to congenital or developmental abnormality.
   d. Conductive hearing loss may be due to dissipation of air-conducted acoustic energy from the third window.
4. Workup
   a. High-resolution computed tomography (CT) temporal bone without contrast.
   b. Physical examination
      i. *Tullio phenomenon*: Nystagmus elicited by sound.
      ii. *Hennebert's sign*: Nystagmus elicited by ear canal pressure.
   c. *Audiometry*: There may be a conductive hearing loss in the affected ear.
   d. Vestibular testing
      i. *Vestibular evoked myogenic potential*: Lower threshold or increased amplitude for response in the affected ear is a typical finding in superior semicircular canal dehiscence (SSCD) syndrome.
5. Management
   a. Treatment is based on the severity of symptoms. Observation alone is acceptable if symptoms are mild.
   b. Surgery
      i. Repair of dehiscence
         - Middle cranial fossa approach with or without obliteration of the SSC lumen is the standard surgical approach.
         - Transmastoid approach can be used if the anatomy of the middle cranial fossa allows access to the SSC.[13]

JAYPEE BROTHERS

ii. Transcanal reinforcement of round window ± oval window.[14]
- Dampens hypercompliance.
- Less invasive.

iii. Tympanostomy tube placement may help with aural pressure symptoms.

Neoplasms

Neoplasms may cause compressive vestibular symptoms due to space-occupying lesions in the brainstem, cerebellum, CPA, or temporal bone.

Cerebellopontine Angle Tumors

The CPA is the most common location for posterior fossa neoplasms.

1. Epidemiology
   a. *Common primary CPA lesions*: Vestibular schwannoma (>90%), meningioma (~3%), facial nerve neuroma (1%), other CN neuromas, glomus tumors, arachnoid cyst, hemangioma.
2. *Presentation*: Presenting symptoms may include progressive sensorineural hearing loss, tinnitus, dysequilibrium (mild imbalance, vertigo uncommon because of slow progressive loss of vestibular function with compensation), facial hypesthesia, increased intracranial pressure (headache, nausea, and vomiting), ataxia.
3. Pathophysiology
   a. Compression of the following:
      i. Cranial nerves VIII, VII, and V.
      ii. Fourth ventricle leading to obstructive hydrocephalus.
      iii. Brainstem, possible cerebellar tonsillar herniation.
   b. Vestibular schwannoma
      i. Benign schwannoma of CN VIII.
      ii. It usually arises from the vestibular nerve, superior or inferior portion.
      iii. Slow-growing tumor, 1–2 mm per year.

JAYPEE BROTHERS

iv. It usually arises within the internal auditory canal, but can develop in the CPA.
v. It is associated with neurofibromatosis type 2 (frequently bilateral schwannomas).

4. Workup
   a. Audiometry
      i. Asymmetric sensorineural hearing loss.
      ii. Impaired speech discrimination, worse than expected for the given extent of hearing loss.
   b. Auditory brainstem response testing
      i. Adjusted interaural latency for wave *V* > 0.2 milliseconds.
      ii. Auditory brainstem response is less used due to sensitivity of MRI.
   c. Magnetic resonance imaging with gadolinium.[15]
   d. Computed tomography with contrast: Computed tomography is not sensitive for detecting small tumors. This is used if MRI is contraindicated (e.g. pacemaker and cochlear implant).
5. Management[16]
   a. Surgical approach to vestibular schwannoma is based on the location and size of the tumor and hearing status.[17]
      i. Translabyrinthine
      ii. Retrosigmoid (suboccipital)
      iii. Middle fossa
   b. Stereotactic radiation.
   c. Serial imaging and observation.

Vascular Causes

Vertigo may occur with decreased blood flow through any of the three branches of the vertebral or basilar arteries: posterior inferior cerebellar artery (PICA), anterior inferior cerebellar artery, and superior cerebellar artery.

1. Permanent
   a. Arterial occlusion by atherosclerotic plaques.
   b. Hemorrhagic or ischemic infarction.

JAYPEE BROTHERS

2. Transient
    a. Vascular spasm.
    b. Low perfusion pressure.
    c. Vascular steal syndromes
        i. *Subclavian steal*: Retrograde flow through the vertebral artery due to stenosis of the subclavian artery proximal to branching of the vertebral artery (decreased vascular resistance through distal subclavian branches leads to siphoning of blood from vertebral artery). Subclavian steal syndrome manifests as vertigo and other neurologic symptoms with arm exercise.

Vertebrobasilar Insufficiency

1. *Epidemiology*: It occurs in the elderly, often with known vascular risk factors.
2. *Presentation*: Abrupt onset of dizziness, lasting minutes. There may be vertigo, headache, nausea, vomiting, diplopia, ataxia, numbness, and weakness.
3. Pathophysiology
    a. Atherosclerosis in the vertebrobasilar system.
    b. Hypertension.
4. Workup
    a. Neurology consult.
    b. Computed tomography to rule out hemorrhagic event.
    c. Magnetic resonance angiography with contrast to identify vertebrobasilar artery stenosis.
5. Management
    a. Control diabetes, hypertension, and hyperlipidemia.
    b. Anticoagulation (aspirin or warfarin).
    c. Consider stenting or endarterectomy.

Lateral Medullary Syndrome (Wallenberg's Syndrome)

1. *Epidemiology*: It usually occurs in middle-aged patients.
2. Presentation
    a. Contralateral sensory deficits to trunk and extremities.

JAYPEE BROTHERS

for detailed information on otorhinolaryngology books, visit our website **www.jaypeebrothers.com,** for detailed in

ENT
Essentials

Salient Features

- The most important information for medical students, residents and primary care physicians
- Symptom-based for easy access, important diagnostic steps and differential diagnosis
- Available in electronic tablet/smart phone format for easy bedside and clinical access
- Multi-author format from leading academic otolaryngologists in each field, cooperating with residents and fellows to ensure that the most important information is included
- Bullet point chapters are linked to multiple choice questions
- The essential otolaryngologic information at your fingertips.



Elina M Toskala MD PhD MBA is a nationally and internationally recognized researcher and teacher. She currently works as Professor in Otolaryngology–Head and Neck Surgery in Thomas Jefferson University, Philadelphia, USA. She has held academic appointments internationally as well as within the US and is past President of Finnish Rhinologic Society and Finnish Society of Allergy and Immunology. She lectures world-wide at CME courses and meetings, and has done so for the last 20 years. Her main area of research has been focusing on chronic rhinosinusitis pathophysiology and genetics. Her other research interest include epidemiological and occupational studies of allergic airway disease, allergen immunotherapy and the connections between upper and lower airway diseases. She has supervised numerous PhD students in Helsinki University, Finland and in the Karolinska Institute in Stockholm, Sweden. She has published over 130 articles and chapters and serves as an Associate Editor for the International Forum of Allergy and Rhinology. She enjoys teaching residents and medical students on a daily basis in her clinic and in the OR, and supervises their research activities.



David W Kennedy MD FACS FRCSI is best known for his work in pioneering endoscopic sinus surgery and minimally invasive endoscopic skull base surgery nationally and internationally. He has served as Director of Resident Training at Johns Hopkins Hospital, Chair of the Department of Otorhinolaryngology–Head and Neck Surgery and Vice Dean at the University of Pennsylvania where he also continues clinical practice as Rhinology Professor. With a long history of medical student, resident and continuing medical education teaching, he is also a past President of the American Academy of Otolaryngology–Head and Neck Surgery, the American Rhinologic Society, the International Rhinologic Society and the ISIAN. Dr Kennedy has been elected to National Academy of Medicine for his contributions to the medical field. He is a prior recipient of the AAO-HNS Board of Governors Practitioner Excellence Award and was awarded the 2010 National Physician of the Year for Clinical Excellence by Castle Connolly, Publishers of America's Top Doctors. He is Editor-in-Chief of the International Forum of Allergy and Rhinology and serves on the editorial boards of numerous other journals. He has published approximately 250 articles and chapters and received a number of international awards.

Available at all medical bookstores
or buy online at www.jaypeebrothers.com



JAYPEE BROTHERS
Medical Publishers (P) Ltd.
www.jaypeebrothers.com

Join us on facebook.com/JaypeeMedicalPublishers

Shelving Recommendation
OTORHINOLARYNGOLOGY

