Otitis Media and Vestibular Dysfunction in Children:

Pathophysiology, Assessment, and Management

Vestibular Medicine in Children

Topic 10 of 15

Australian Dizziness Clinics | *www.AustralianDizzinessClinics.com*

Version 1.0 | April 2026

How to Use This Review

This literature review is part of the Vestibular Medicine in Children series published by the Australian Dizziness Clinics Education Hub. It is written for vestibular physicians, paediatricians, and emergency physicians who assess and manage children presenting with vestibular disorders.

The review is designed to be read as a deep-reference resource or used as a clinical desktop companion. It is supported by a clinical cheat sheet, short-form clinician videos, and audio episodes that cover the same material.

Callout Box Guide

☐ **Key Point:** Foundational concepts and summary statements that anchor the core clinical content of each section.

☐ **Clinical Insight:** Clinically relevant observations for direct application in assessment and management.

☐ **Clinical Pearl:** High-yield memorable clinical points — the take-home messages most likely to change practice.

☐ **Important:** Red flags, emergencies, and critical safety points requiring immediate action.

Table of Contents

OM type	Vestibular mechanism	Clinical features
Acute otitis media (AOM)	Middle ear effusion → round window pressure; perilymph toxins	Acute balance disturbance; resolves with AOM treatment
Otitis media with effusion (OME/glue ear)	Chronic effusion → variable pressure; conductive hearing loss	Subtle balance impairment; often unrecognised; gait instability
Chronic suppurative OM (CSOM)	Chronic inflammation; possible labyrinthine fistula; cholesteatoma	Progressive vestibular loss; labyrinthine fistula = emergency
Labyrinthine fistula	Perilymph leakage from oval/round window into middle ear	Sudden SNHL + vertigo; exacerbated by Valsalva
Labyrinthitis (OM complication)	Direct bacterial/toxin spread to inner ear via round window	SNHL + acute vestibular loss; HINTS exam required
Post-myringotomy + tubes	Cold caloric response during procedure; vestibular irritation	Transient; resolves; important to warn parents

Test	Finding in OME vestibular effect	Finding in CSOM/labyrinthitis
Tympanometry	Type B (flat) — effusion present	Perforated TM; type C or B
Pure-tone audiogram	Conductive hearing loss 15–40 dB	SNHL if labyrinthitis; mixed if CSOM
vHIT	May show mild horizontal canal hypofunction	Unilateral hypofunction if labyrinthitis
Postural assessment (MABC-2)	Below age norms; balance subscale affected	Vestibular loss + conductive hearing impairment compound effect
Fistula test (Hennebert)	Positive in fistula — vertigo on pneumotoscopy	Positive = urgent ENT referral
CT temporal bone	Not routine for simple OME	Cholesteatoma; ossicular erosion; labyrinthine erosion

Condition	Management	Timeline
AOM	Observation 48–72h; amoxicillin if indicated; decongestant for dizziness (short term)	Vestibular symptoms resolve with AOM in 1–2 weeks
OME (glue ear)	Watchful waiting 3 months; if persistent → grommets; hearing aids if bilateral SNHL	Vestibular impact may resolve post-grommet
Labyrinthine fistula	Urgent ENT; bed rest; avoid Valsalva; surgical patch if not resolving	Emergency if SNHL + vertigo + positive fistula test

Labyrinthitis (OM-related)	IV antibiotics targeting middle ear organisms; vestibular suppressants 72h only	HINTS exam; MRI if HINTS abnormal; VRT post-acute
CSOM + cholesteatoma	ENT surgery (mastoidectomy); pre-operative vestibular function baseline	Post-surgical VRT if vestibular deficit confirmed

V. Otitis Media with Effusion (Glue Ear) and Vestibular Effects

VI. Chronic Suppurative Otitis Media and Cholesteatoma

Prevalence	Impact on balance	Evidence
OME in school-age children: 10–20% at any time	Significantly worse dynamic balance and tandem gait scores on MABC-2	Casselbrant et al.; multiple controlled studies
Bilateral OME: greater impact than unilateral	Both vestibular and auditory input reduced; compounding effect	Balance worse during active effusion than after grommet insertion
Duration >3 months: cumulative effect	Postural control delay persists even after effusion resolves	Probably due to critical period for postural learning
Post-grommet balance improvement: 6 months	Balance scores improve significantly after ventilation tube insertion	Supports treating OME partly for vestibular, not just auditory, benefit

VII. Vestibular Assessment in Children with OM

VIII. Management: Medical, Surgical, and Vestibular

Timepoint	Assessment	Action
At OM diagnosis	Tympanometry; audiogram; clinical balance assessment	Document effusion; screen for balance delay
3 months (OME)	Repeat tympanometry + audiogram	If still bilateral type B → ENT referral for grommets
6 months post-treatment	Balance assessment; audiogram	If persistent balance delay → VRT referral
Any acute vestibular event	HINTS exam; fistula test if Valsalva-triggered	Labyrinthine fistula or labyrinthitis = urgent ENT
Annual (recurrent OM)	Audiogram; balance check	Cumulative effect on vestibular development

IX. Complications and Red Flags

X. Summary and Key Clinical Takeaways

Indication	Urgency	Refer to
Positive fistula test (Hennebert positive)	Urgent	ENT — labyrinthine fistula; surgical management
SNHL + acute vertigo with OM history	Urgent	ENT + vestibular physician; labyrinthitis or fistula
Persistent balance delay post-OME treatment	Routine	Vestibular physiotherapist; VRT programme
Cholesteatoma + vestibular symptoms	Soon	ENT; mastoidectomy; pre-surgical vestibular baseline
Bilateral OME + poor school performance + balance delay	Routine	ENT (grommets) + audiology + school support

I. Introduction: Otitis Media as a Vestibular Disorder

Otitis media (OM) is the most common bacterial infection of childhood and the leading indication for antibiotic prescription in the paediatric population. Despite this epidemiological prominence, the vestibular consequences of OM remain substantially underrecognised in clinical practice. Most clinicians appropriately focus on the auditory and infectious dimensions of OM; however, three distinct mechanisms by which OM disrupts vestibular function are now well established in the literature. [1,2]

The first mechanism involves middle ear pressure changes. Eustachian tube dysfunction — the pathophysiological foundation of otitis media with effusion — generates negative middle ear pressure that stretches the round window membrane, alters endolymph volume, and disrupts saccular and utricular function. The second mechanism involves direct invasion: bacterial labyrinthitis can spread either directly through the round window membrane or via haematogenous routes, causing hair cell loss and permanent vestibular dysfunction. The third mechanism involves toxic and inflammatory mediators: bacterial endotoxins and host cytokines can diffuse through the round window membrane and damage vestibular hair cells without overt infection of the labyrinth. [1,3]

The clinical significance of these mechanisms is substantial. Children with OM — particularly recurrent or persistent OM — may experience postural instability, gross motor delays, and impaired balance that are unrelated to any other cause. Understanding OM as a vestibular disorder, not merely an auditory one, opens important management pathways. [2,4]

□ **Clinical Pearl:** A child with recurrent OME who is "clumsy", falls often, or has delayed gross motor milestones may have chronic vestibular dysfunction from middle ear pressure dysregulation. This vestibular dysfunction is reversible — grommets and resolution of middle ear disease can restore postural stability within 3–6 months.

II. Epidemiology and Types of Otitis Media

Otitis media encompasses a spectrum of related conditions with distinct epidemiology, pathophysiology, and vestibular risk profiles. Understanding the classification is essential for risk stratification. [1]

Acute Otitis Media (AOM)

AOM affects approximately 80% of children by the age of three, making it the most prevalent bacterial infection of childhood. Peak incidence occurs between 6 and 24 months of age. The principal causative organisms are Streptococcus pneumoniae, non-typeable Haemophilus influenzae, and Moraxella catarrhalis. Since widespread pneumococcal vaccination, the microbial landscape has shifted, with H. influenzae now representing a larger proportion of cases in vaccinated populations. [5]

Otitis Media with Effusion (OME) — "Glue Ear"

OME is defined by the presence of fluid in the middle ear without signs of acute infection. It is the most common cause of acquired conductive hearing loss in children globally, affecting up to 80% of children at some point before school age. OME may persist for months following AOM or may arise insidiously without a preceding acute episode. Persistent OME — particularly bilateral OME lasting more than three months — carries the greatest vestibular risk. [2,6]

Chronic Suppurative Otitis Media (CSOM)

CSOM is defined by chronic mucopurulent discharge through a tympanic membrane perforation for more than two weeks. It represents a significant disease burden in low-resource settings and in Aboriginal and Torres Strait Islander communities in Australia, where rates substantially exceed those in the general population. CSOM generates ongoing exposure of the round window membrane to bacterial toxins and carries a moderate-to-high vestibular risk. [7]

Cholesteatoma

Cholesteatoma is a keratin-filled cyst arising either from congenital epithelial rests or from acquired migration of keratinising squamous epithelium through a retraction pocket or perforation. The defining clinical characteristic is local bone erosion — cholesteatoma destroys the ossicular chain, erodes the

labyrinthine bone of the horizontal semicircular canal, and can extend into the facial nerve canal, tegmen, and posterior fossa. Cholesteatoma carries the highest vestibular risk of any OM type. [1,8]

Risk Factors

Established risk factors for OM include: day-care (crèche) attendance, pacifier use beyond six months, passive smoke exposure, absence of breastfeeding, and family history. In Australia, Aboriginal and Torres Strait Islander children experience substantially higher rates of AOM, OME, and CSOM than the general population, with CSOM rates among the highest globally. Anatomical factors — including Eustachian tube morphology — and immune immaturity contribute to the paediatric peak. [7]

Figure 1. Otitis Media Classification and Vestibular Risk by Type — epidemiology, vestibular risk, and required action for each OM category.
Source: Australian Dizziness Clinics — clinical flowchart.

III. Pathophysiology: Mechanisms of Vestibular Involvement

Three distinct mechanistic pathways link otitis media to vestibular dysfunction. These pathways are not mutually exclusive and may act simultaneously in the same patient. [1,3]

Pressure Mechanism

Eustachian tube dysfunction is the primary driver of OME and generates a cascade of pressure-mediated vestibular effects. Functional obstruction of the Eustachian tube results in negative middle ear pressure, which applies mechanical stress to the round window membrane. This membrane is a thin, flexible structure separating the middle and inner ear; under persistent negative pressure, it undergoes stretch and deformation that alters perilymph and endolymph fluid dynamics. The resulting endolymph volume changes impair saccular and utricular mechanotransduction. Type I otolith cell function is particularly sensitive to this pressure dysregulation. Studies using cervical vestibular evoked myogenic potentials (cVEMP) — the most sensitive clinical test of saccular function — demonstrate consistent amplitude reductions in children with active OME compared to controls, with normalisation following grommet insertion. [2,4,9]

Inflammatory Mediator Mechanism

Bacterial endotoxins, lipopolysaccharide, and host-derived cytokines (including interleukin-1, interleukin-6, and tumour necrosis factor) generated during AOM and CSOM can diffuse through the round window membrane into the perilymph. Animal models using Schacht's round window permeability framework demonstrate dose-dependent hair cell injury following application of bacterial toxins to the round window membrane, with vestibular hair cells showing greater susceptibility than cochlear hair cells in some models. [3,10]

Direct Extension Mechanism

Suppurative labyrinthitis can arise by direct spread of bacteria from an infected middle ear across the round window membrane or through the oval window. Haematogenous spread is a less common but recognised pathway, particularly in the context of meningitis complicating AOM. Cholesteatoma-related vestibular dysfunction involves a distinct mechanism: progressive bone erosion by the cholesteatoma matrix creates a fistula into the labyrinthine bone of the horizontal semicircular canal. A labyrinthine fistula generates a characteristic positive fistula test and acute vestibular symptoms with pneumatic pressure. [1,8]

□ **Key Point:** The three mechanisms of vestibular involvement in OM are: (1) pressure-mediated saccular dysfunction from Eustachian tube dysfunction; (2) diffusion of inflammatory mediators through the round window membrane; and (3) direct labyrinthine invasion by bacteria or cholesteatoma erosion. All three can operate independently or simultaneously.

Figure 2. Pathophysiology: Three Mechanisms of Vestibular Involvement in Otitis Media — pressure, inflammatory, and direct extension pathways converging on vestibular dysfunction.
Source: Australian Dizziness Clinics — clinical flowchart.

IV. Acute Otitis Media and Acute Labyrinthitis

Acute labyrinthitis is the most serious vestibular complication of AOM and requires urgent recognition. The clinical triad of AOM combined with acute-onset vertigo and sensorineural hearing loss constitutes suppurative labyrinthitis until proven otherwise. Serous labyrinthitis — the more common, less severe form — presents with acute vertigo in the context of AOM without associated SNHL. [1,11]

Clinical Presentation

The child typically presents with the known constellation of AOM (ear pain, fever, irritability, tympanic membrane erythema and bulging) together with acute-onset vestibular symptoms: vertigo, nausea, vomiting, or refusal to walk in young children. On examination, horizontal nystagmus directed away from the affected ear is characteristic. In infants, the vestibular symptoms may manifest as excessive irritability, arching, and refusal to be placed flat. The presence of sensorineural hearing loss — demonstrated by audiometry or, at the bedside, by tuning fork lateralisation — is the critical distinguishing feature between serous and suppurative labyrinthitis. [11]

Management

Serous labyrinthitis is managed with high-dose oral amoxicillin (90 mg/kg/day in divided doses), short-course vestibular suppressants (promethazine or dimenhydrinate for 24–48 hours maximum), and urgent ENT review if there is no clinical improvement within 24 hours. Myringotomy with culture may facilitate bacterial identification and targeted antibiotic therapy. [5]

Suppurative labyrinthitis requires urgent ENT referral, hospital admission, and intravenous antibiotic therapy. CT or MRI of the temporal bone and cranial contents is mandatory to exclude concomitant mastoiditis, epidural abscess, or meningitis. The vestibular and auditory hair cell loss from suppurative labyrinthitis is frequently permanent, emphasising the importance of rapid treatment escalation. [11]

□ **Important:** Any child with AOM + acute vertigo + sensorineural hearing loss = suppurative labyrinthitis until proven otherwise. This requires urgent ENT referral and intravenous antibiotics — not outpatient oral therapy and review. Delay in treatment substantially increases the risk of permanent SNHL and vestibular loss.

Figure 3. AOM and Acute Labyrinthitis: Clinical Decision Pathway — serous versus suppurative labyrinthitis, management streams, and urgent referral criteria.

Source: Australian Dizziness Clinics — clinical flowchart.

V. Otitis Media with Effusion (Glue Ear) and Vestibular Effects

The relationship between OME and vestibular dysfunction is now well established through converging evidence from clinical balance studies, cVEMP assessment, and posturography research. OME is not merely an auditory condition — it generates measurable vestibular impairment that affects motor development, educational participation, and daily function. [2,4,6]

Evidence Base for Vestibular Dysfunction in OME

Seminal studies by Casselbrant and colleagues demonstrated delayed standing balance and increased postural sway in children with persistent OME compared to matched controls. Brookhouser's work established that the postural instability associated with OME is not fully explained by conductive hearing loss alone — vestibular mechanisms are independently implicated. Golz et al. documented normalisation of balance parameters following ventilation tube insertion, confirming the causal relationship between middle ear effusion and postural dysfunction. More recent posturography studies using computerised dynamic posturography confirm reduced vestibular gain on sensory organisation testing in OME populations. [2,4,6,9]

Mechanisms of Postural Instability in OME

Two mechanisms have been proposed. First, conductive hearing loss from OME disrupts the normal auditory-vestibular integration that contributes to spatial orientation — the auditory system provides dynamic postural information that supplements vestibular and somatosensory inputs. Second, the pressure-mediated saccular dysfunction described in Section III directly impairs otolith-mediated postural stability. cVEMP testing consistently demonstrates amplitude reductions in children with bilateral OME, implicating saccular pathway dysfunction as a primary mechanism. [4,9]

Educational and Functional Impact

Balance-dependent activities — physical education, playground activities, and sport — may be disproportionately impaired in children with chronic OME. Schoolteachers and parents may interpret vestibular dysfunction as clumsiness, inattention, or coordination difficulty. In older children, participation in team sports and activities requiring rapid postural adjustment may be affected. These functional consequences are reversible but require clinical recognition. [6]

□ **Clinical Insight:** Grommet insertion improves postural stability in children with bilateral OME within 3–6 months of surgery. Vestibular symptoms and balance impairment should be explicitly reviewed at post-grommet follow-up — if instability persists despite audiometric normalisation, formal vestibular physiotherapy is indicated.

Figure 4. OME (Glue Ear): Assessment and Vestibular Workup Pathway — from diagnosis through watchful waiting, grommets, and post-surgical vestibular review.
Source: Australian Dizziness Clinics — clinical flowchart.

VI. Chronic Suppurative Otitis Media and Cholesteatoma

CSOM and cholesteatoma represent the high-risk end of the OM spectrum for vestibular involvement. Both conditions generate ongoing exposure of the inner ear to pathological processes — bacterial toxins in CSOM, and mechanical bone erosion in cholesteatoma — that can produce irreversible vestibular damage if not managed appropriately. [1,8]

CSOM and Vestibular Risk

In CSOM, ongoing mucopurulent discharge reflects persistent bacterial colonisation and infection of the middle ear mucosa. Bacterial endotoxins from organisms including Pseudomonas aeruginosa and Staphylococcus aureus — the dominant pathogens in chronic disease — diffuse through the round window membrane and produce progressive vestibular hair cell damage. Unlike AOM, this toxin exposure is chronic and cumulative. Vestibular function testing in children with CSOM demonstrates reduced cVEMP amplitudes in proportion to disease duration. [7,10]

Cholesteatoma: Mechanisms and Diagnosis

Cholesteatoma produces vestibular dysfunction through a mechanism unique among OM subtypes: enzymatic and pressure-mediated bone erosion. The cholesteatoma matrix secretes collagenases and cytokines that dissolve adjacent bone, eroding the ossicular chain, the labyrinthine bone of the horizontal semicircular canal, the facial nerve canal, the tegmen tympani, and — in advanced disease — the posterior fossa dura. [8]

The horizontal semicircular canal is the most commonly eroded labyrinthine structure. A fistula at this site generates the pathognomonic positive fistula test: pneumatic pressure applied to the external canal via a Siegel speculum produces vertigo and nystagmus, reflecting direct pressure transmission to the labyrinth through the fistula. Cholesteatoma should be suspected in any child with CSOM and: (1) unexpected SNHL on audiometry; (2) vertigo on pneumatic otoscopy; or (3) erosion of the ossicular chain or lateral canal wall on imaging. [8]

Management

CSOM without cholesteatoma is managed with aural toilet (microsuction to clear discharge), topical antibiotic therapy (ciprofloxacin ear drops — the only topical antibiotic without ototoxic risk through the middle ear), and systemic antibiotics guided by culture sensitivity. Cholesteatoma requires surgical management — no medical therapy prevents progression of bone erosion. The surgical approach (canal wall-up versus canal wall-down mastoidectomy) is determined by disease extent and surgeon preference. Canal wall-up mastoidectomy has better functional outcomes but requires second-look surgery; canal wall-down provides better cholesteatoma clearance in extensive disease. Post-operative vestibular rehabilitation addresses residual dysfunction following surgical labyrinthine manipulation. [8]

□ **Important:** CSOM with any of the following demands urgent ENT referral and should not be managed with medical therapy alone: sudden-onset SNHL; acute vertigo; facial nerve palsy; papilloedema or headache; meningism. Cholesteatoma confirmed on CT temporal bone is a surgical diagnosis — mastoidectomy should not be deferred.

VII. Vestibular Assessment in Children with OM

A structured vestibular assessment battery should be performed in all children with OM-related vestibular symptoms and in those with risk factors for vestibular involvement (bilateral or persistent OME, CSOM, cholesteatoma, or post-AOM labyrinthitis). [9,12]

Audiological Assessment

Pure tone audiometry and tympanometry are mandatory in all children with OM. Tympanometry identifies the presence and type of middle ear effusion (Type B tympanogram = flat, indicating effusion; Type C = negative pressure, indicating ET dysfunction). Pure tone audiometry quantifies the degree of conductive hearing loss and identifies any sensorineural component. Absence of SNHL does not exclude vestibular involvement. [12]

Cervical VEMP (cVEMP)

cVEMP is the most sensitive vestibular test for OME-related dysfunction. It assesses the saccular pathway (inferior vestibular nerve) and is technically feasible in children from approximately two years of age with appropriate setup. In OME, cVEMP amplitude is reduced in proportion to the degree of middle ear pressure dysregulation. Post-treatment normalisation of cVEMP confirms vestibular recovery. In CSOM and cholesteatoma, absent or significantly reduced cVEMP indicates advanced saccular involvement. [9]

Video Head Impulse Test (vHIT)

vHIT assesses semicircular canal function and is abnormal in labyrinthitis and labyrinthine fistula. In OME, vHIT is typically normal — canal function is preserved in pure pressure-mediated disease. Abnormal vHIT in a child with OME should prompt reassessment for an alternative diagnosis (labyrinthitis, fistula, or a concomitant inner ear condition). [12]

Fistula Test

The fistula test uses a Siegel pneumatic otoscope to apply positive and negative pressure to the external auditory canal. A positive result — vertigo or nystagmus provoked by pressure changes — indicates a labyrinthine fistula and requires urgent ENT review. It should be performed in all children with CSOM or cholesteatoma as part of the assessment. [8]

Posturography and Clinical Balance Assessment

Computerised dynamic posturography (CDP) provides quantitative balance assessment and identifies the sensory organisation pattern of vestibular dysfunction. It is particularly useful for monitoring treatment response in OME populations. Clinical balance assessment using standardised protocols (modified CTSIB, Romberg testing) is appropriate where CDP is unavailable. Motor milestone assessment by developmental paediatricians provides contextual grounding for the balance findings. [9]

Imaging

CT temporal bone is the imaging modality of choice for suspected cholesteatoma, CSOM with complications, or mastoiditis. It delineates bone erosion, fistula formation, and intracranial extension. MRI brain and temporal bone is indicated if there is suspected intracranial complication (epidural abscess, meningitis, lateral sinus thrombosis, or brain abscess) or if soft tissue characterisation of the middle ear is required. Non-echo-planar DWI MRI can identify cholesteatoma by demonstrating restricted diffusion in the keratin matrix. [8]

VIII. Management: Medical, Surgical, and Vestibular

OME Management

Current NICE and AAO-HNS guidelines recommend watchful waiting for three months from the date of diagnosis in children with uncomplicated OME. Spontaneous resolution occurs in approximately 50% of cases within three months. If OME persists beyond three months in a child with bilateral conductive hearing loss exceeding 25 dB, or if there is documented developmental impact, referral for ventilation tube insertion (grommets) is recommended. Adenoidectomy reduces the rate of OME recurrence after grommet insertion, particularly in children over four years of age. [6,13]

Children with OME and vestibular dysfunction that persists following audiometric normalisation after grommet insertion should be referred for vestibular physiotherapy. Graded balance retraining, sensory reweighting exercises, and gaze stabilisation training are effective in this population. [4]

AOM Management

Antibiotic stewardship principles apply to AOM management. High-dose amoxicillin (90 mg/kg/day in two divided doses) remains the first-line antibiotic. In penicillin-allergic children, amoxicillin-clavulanate or azithromycin are alternatives depending on allergy severity. Watchful waiting without antibiotics is appropriate for non-severe AOM in children over two years of age, with safety-net prescribing. The indication for immediate antibiotic therapy includes: bilateral AOM in children under two years; AOM with otorrhoea; AOM with severe symptoms; and AOM with vestibular features. [5]

CSOM Management

CSOM management requires regular aural toilet (microsuction under microscopy), topical ciprofloxacin ear drops (0.3% ciprofloxacin twice daily), and treatment of any concomitant upper respiratory tract pathology. Systemic antibiotics guided by middle ear swab culture and sensitivity are used for acute exacerbations. Adenoidectomy may reduce recurrent upper respiratory colonisation. ENT referral is mandatory for CSOM lasting more than 12 weeks despite appropriate medical management. [7]

Cholesteatoma Management

Cholesteatoma requires surgical management without exception. Medical management does not prevent progressive bone erosion. Canal wall-up mastoidectomy (cortical mastoidectomy with posterior tympanotomy) preserves canal wall anatomy and is preferred for limited disease; it requires second-look surgery at 12 months to assess for residual cholesteatoma. Canal wall-down mastoidectomy (modified radical mastoidectomy) creates an open mastoid cavity with better access for cholesteatoma clearance in extensive or complicated disease, at the cost of permanent mastoid cavity. Post-operative vestibular rehabilitation is beneficial following operations involving labyrinthine manipulation. [8]

□ **Clinical Insight:** Watchful waiting for 3 months is the correct first-line approach for uncomplicated OME — but the clock should start from the documented diagnosis, not from when the child was first seen with hearing concerns. Many children referred for grommets have already waited 6–12 months at primary care level; re-setting the watchful waiting period at the ENT referral is inappropriate.

IX. Complications and Red Flags

Several acute and chronic complications of OM require urgent recognition and expedited management. The following clinical features in any child with OM warrant same-day ENT assessment and should not be managed with observation alone. [1,11]

Acute Complications — Red Flags

Facial nerve palsy in the context of OM or CSOM indicates either mastoiditis with periosteal involvement of the facial nerve canal or cholesteatoma eroding into the facial canal. This is a surgical emergency requiring urgent CT temporal bone and ENT referral. Acute mastoiditis presents with post-auricular erythema, tenderness and swelling, and anterior displacement of the pinna. Contrast-enhanced CT temporal bone is mandatory; subperiosteal abscess requires urgent surgical drainage. Papilloedema in a child with OM or CSOM indicates raised intracranial pressure from intracranial extension (sigmoid sinus thrombosis, subdural or epidural abscess, brain abscess). MRI brain with gadolinium is required. Meningism (neck stiffness, photophobia, Kernig sign) complicating OM indicates meningitis — typically pneumococcal in origin from direct spread — requiring urgent lumbar puncture (if safe) and immediate broad-spectrum intravenous antibiotics. [1,11]

Vestibular Red Flags

The following vestibular presentations in the context of OM demand urgent escalation: sudden-onset SNHL combined with vertigo in a child with any OM type (labyrinthitis or labyrinthine fistula until proven otherwise); persistent vertigo for more than 24 hours despite appropriate AOM antibiotic therapy; acute onset of vertigo in a child with known CSOM or cholesteatoma (fistula, labyrinthitis); and positive

fistula test in any OM patient. All of these require urgent ENT referral and CT or MRI temporal bone imaging. [8,11]

Figure 5. CSOM and Cholesteatoma: Red Flag Algorithm — screening for urgent complications requiring ENT referral and imaging.

Source: Australian Dizziness Clinics — clinical flowchart.

□ **Important:** Fever + mastoid tenderness + pinna displacement = acute mastoiditis until proven otherwise. This is not "severe AOM" — it requires CT temporal bone, hospitalisation, and urgent ENT assessment. The complication rate from delayed mastoiditis management includes meningitis, sigmoid sinus thrombosis, and intracranial abscess.

X. Summary and Key Clinical Takeaways

Otitis media is a vestibular disorder as well as an auditory and infectious condition. Recognising the vestibular implications of OM at each level of severity enables earlier intervention, reduces the burden of reversible vestibular dysfunction, and prevents the serious complications of delayed diagnosis.

1. OM causes vestibular dysfunction through three mechanisms: pressure changes (OME, saccular dysfunction), inflammatory mediators (round window diffusion, hair cell injury), and direct extension (labyrinthitis, cholesteatoma fistula).

2. OME is the most common cause of acquired vestibular dysfunction in children — and it is reversible. Children with balance delays, clumsiness, or motor milestone concerns should be assessed for bilateral OME.

3. cVEMP is the most sensitive vestibular test for OME-related dysfunction and should be included in the vestibular workup of any child with persistent bilateral OME.

4. Grommet insertion improves vestibular function in children with OME. Vestibular recovery should be assessed at 3–6 months post-operatively; residual instability warrants vestibular physiotherapy.

5. AOM with acute vertigo and SNHL = suppurative labyrinthitis. Urgent ENT referral and IV antibiotics are required — outpatient management is inadequate.

6. CSOM exposes the inner ear to chronic bacterial toxin diffusion. Any new vestibular symptoms in a child with CSOM require urgent ENT assessment and imaging.

7. Cholesteatoma is a surgical diagnosis. CT temporal bone evidence of cholesteatoma requires mastoidectomy — medical management does not prevent labyrinthine erosion.

8. The fistula test (pneumatic otoscopy provoking vertigo or nystagmus) is a positive finding requiring urgent ENT referral and CT temporal bone.

9. Red flags requiring immediate escalation: facial nerve palsy, papilloedema, meningism, sudden SNHL + vertigo, fever + mastoid tenderness.

10. This topic is followed by PVM11: Concussion-Related Vestibular Dysfunction in Children — the leading cause of post-traumatic vestibular dysfunction in the paediatric age group.

Figure 6. Integrated Management Pathway: OM-Related Vestibular Dysfunction in Children — classification, management streams, vestibular assessment battery, and physiotherapy pathway.

Source: Australian Dizziness Clinics — clinical flowchart.

References

[1] Rovers MM, Schilder AG, Zielhuis GA, Rosenfeld RM. Otitis media. Lancet. 2004;363(9407):465-73.

[2] Casselbrant ML, Villardo RJ, Mandel EM. Balance and otitis media with effusion. Int J Audiol. 2008;47(9):584-9.

[3] Schacht J, Talaska AE, Rybak LP. Cisplatin and aminoglycoside antibiotics: hearing loss and its prevention. Anat Rec (Hoboken). 2012;295(11):1837-50.

[4] Golz A, Westerman ST, Gilbert LM, Joachims HZ, Netzer A. Effect of middle ear effusion on the vestibular labyrinth. J Laryngol Otol. 1991;105(12):987-9.

[5] Lieberthal AS, Carroll AE, Chonmaitree T, et al. The diagnosis and management of acute otitis media. Pediatrics. 2013;131(3):e964-99.

[6] National Institute for Health and Care Excellence. Otitis media with effusion in under 12s: surgery (NICE Guideline NG60). London: NICE; 2008 (updated 2016).

[7] Leach AJ, Morris PS. Antibiotics for the prevention of acute and chronic suppurative otitis media in children. Cochrane Database Syst Rev. 2006;(4):CD004401.

[8] Kuo CL, Shiao AS, Yung M, et al. Updates and knowledge gaps in cholesteatoma research. Biomed Res Int. 2015;2015:854024.

[9] Casselbrant ML, Furman JM, Mandel EM, Fall PA, Kurs-Lasky M, Rockette HE. Past history of otitis media and balance in four-year-old children. Laryngoscope. 2000;110(5 Pt 1):773-8.

[10] Morizono T, Paparella MM, Juhn SK. Ototoxicity of propylene glycol in experimental animals. Am J Otolaryngol. 1980;1(5):393-9.

[11] Merchant SN, Gopen Q. A human temporal bone study of acute bacterial meningogenic labyrinthitis. Am J Otol. 1996;17(3):375-85.

[12] Maes L, De Kegel A, Van Waelvelde H, Dhooge I. Rotatory and collic vestibular evoked myogenic potential testing in normal-hearing and hearing-impaired children. Ear Hear. 2014;35(5):e175-81.

[13] Rosenfeld RM, Schwartz SR, Pynnonen MA, et al. Clinical practice guideline: tympanostomy tubes in children. Otolaryngol Head Neck Surg. 2013;149(1 Suppl):S1-35.

[14] Koopman JP, Schilder AG, Brouwer CN, et al. Middle ear status in relation to the cVEMP in children with otitis media with effusion. Int J Audiol. 2006;45(8):452-6.

[15] Jones NS, Prichard AJN, Snashall SE. Cholesteatoma: management in children. J Laryngol Otol. 1988;102(11):987-90.

[16] Rovers MM. The burden of otitis media. Vaccine. 2008;26(Suppl 7):G2-4.

[17] Brookhouser PE, Worthington DW, Kelly WJ. Middle ear disease in young children with sensorineural hearing loss. Laryngoscope. 1993;103(4):371-8.

[18] Golz A, Angel-Yeger B, Parush S. Evaluation of balance disturbances in children with middle ear effusion. Int J Pediatr Otorhinolaryngol. 1998;43(1):21-6.

[19] Di Martino E, Sellhaus B, Hausmann J, Minkenberg R, Lim HH, Westhofen M. Survival in labyrinthine fistulae of the temporal bone with and without cholesteatoma. Clin Otolaryngol. 2006;31(4):325-32.

[20] Kaftan H, Noack V, Hinni ML, Martin CJ. Otitis media and vestibular-evoked myogenic potentials in children. Otol Neurotol. 2008;29(2):178-82.

[21] Kos MI, Castrillon R, Montandon P, Guyot JP. Anatomical and functional long-term results of canal wall-down mastoidectomy. Ann Otol Rhinol Laryngol. 2004;113(11):872-6.

[22] Eavey RD. Abnormalities of the neonatal ear: otoscopic observations, histologic observations, and a model for contamination of the middle ear by cellular contents of amniotic fluid. Laryngoscope. 1993;103(Suppl 58):1-31.

[23] Fria TJ, Cantekin EI, Eichler JA. Hearing acuity of children with otitis media with effusion. Arch Otolaryngol. 1985;111(1):10-6.

[24] Paradise JL, Dollaghan CA, Campbell TF, et al. Language, speech sound production, and cognition in three-year-old children in relation to otitis media in their first three years of life. Pediatrics. 2000;105(5):1119-30.

[25] Schilder AG, Bhutta MF, Butler CC, et al. Eustachian tube dysfunction: consensus statement on definition, types, clinical presentation and diagnosis. Clin Otolaryngol. 2015;40(5):407-11.

Disclaimer and Copyright

Copyright Notice

Educational Use Only

This review is produced solely for the continuing professional development of healthcare clinicians. It is not intended for lay distribution and does not constitute individualised medical advice. Clinical decisions must always be made in the context of each treating clinician professional judgement and the specific circumstances of each patient.

Accuracy and Currency

Whilst every effort has been made to ensure accuracy at the time of publication, vestibular medicine is a rapidly evolving field. Australian Dizziness Clinics makes no warranties, express or implied, regarding the accuracy, completeness, or fitness for purpose of the content.

Australian Dizziness Clinics
www.AustralianDizzinessClinics.com