


# Otitis Media with Effusion in Children: A Review of Pathophysiology, Risk Factors, and Management

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## Abstract:

Otitis Media with Effusion (OME) is characterized by the presence of middle ear fluid without signs of acute infection and is primarily associated with Eustachian tube (ET) dysfunction. In children, ET immaturity, impaired middle ear ventilation, and inflammatory conditions contribute to negative pressure and fluid accumulation. Younger age, particularly under seven years, is a major risk factor due to anatomical and immunological immaturity. Additional predisposing factors include bottle feeding, pacifier use, daycare attendance, exposure to secondhand smoke, allergic rhinitis, and recurrent upper respiratory tract infections. Diagnosis is often subtle and relies on otoscopy, tympanometry, and audiological assessment. Tympanometry remains the gold standard for detecting effusion, while audiometry typically demonstrates fluctuating conductive hearing loss, predominantly affecting low frequencies. OME may negatively impact speech and language development, academic performance, and behavior, particularly when hearing loss is persistent or asymmetric.

Most cases resolve spontaneously within three months; therefore, initial management focuses on observation, hearing monitoring, and modification of risk factors. Surgical intervention with ventilation tube insertion is recommended for persistent OME lasting longer than three months when associated with significant hearing loss or documented speech and learning difficulties. Early identification and appropriate management are essential to prevent long-term auditory, cognitive, and linguistic consequences.

**Key words:** otitis media with effusion; children; ventilation tubes

## Introduction

Otitis media with effusion (OME) is defined as the presence of fluid within the middle ear (ME) in the absence of local or systemic signs of acute infection typically associated with acute otitis media (AOM), such as otalgia, fever, or overt inflammatory changes [1–5]. In this condition, fluid accumulates in the tympanic cavity without the characteristic clinical features of active infection, often resulting in a subtle or asymptomatic presentation. OME is widely regarded as the maximal expression of Eustachian tube (ET) dysfunction, as the likelihood of effusion formation increases proportionally with the degree of negative pressure within the ME [3].

Children younger than seven years are at increased risk for developing both OME and AOM, primarily due to immaturity of the immune system and ET dysfunction [6]. Following an episode of AOM, persistence of ME effusion is common, with fluid present in approximately 70% of cases after two weeks, 40% after one month, 20% after two months, and 10% after three months, until normal ME aeration is restored [7].

Immunological immaturity plays a central role in this susceptibility. Humoral immunity reaches near adult levels for immunoglobulin G (IgG) by approximately five to six years of age, whereas other antibody classes, including IgM and IgA, mature later, typically between 10 and 12 years [8]. Cell-mediated immunity, including T-cell function, stabilizes around six to seven years of age, although T-cell receptor diversity continues to develop throughout adolescence [9]. In addition, mucosal immunity—particularly secretory IgA—may not reach full maturity until late childhood, contributing to prolonged inflammatory responses and delayed resolution of ME effusion [10].

There is substantial clinical and experimental evidence supporting the critical role of ET function in maintaining normal hearing and ME homeostasis, and ET dysfunction is recognized as a key causal factor in the development of otitis media [11]. Gas exchange across the ME mucosa is the primary mechanism regulating ME pressure, while the ET acts as a dynamic valve, releasing excessive positive pressure and actively

equalizing negative pressure through muscular opening [12]. Inflammatory processes that alter mucosal thickness, vascular permeability, and blood flow impair gas diffusion across the ME mucosa, thereby disrupting pressure regulation and promoting effusion formation [10,13].

Failure of the ET to adequately perform its essential functions—pressure regulation, secretion clearance, and protection of the ME—leads to the development of negative ME pressure due to insufficient ventilation [13]. This negative pressure promotes transudation of intravascular fluid into the interstitial space and subsequently into the ME lumen, a process strongly associated with effusion formation [14]. However, effusion may also develop in the absence of markedly negative pressure, particularly following inflammatory insults mediated by pro-inflammatory cytokines [20]. Persistent negative pressure and chronic ET dysfunction may result in tympanic membrane (TM) retraction, ME atelectasis, and, in advanced cases, TM perforation with epithelial migration and cholesteatoma formation [15].

This progressive pathological sequence is described by the Continuum Theory, which proposes that otitis media evolves along a continuous spectrum from serous or purulent inflammation to seromucoid, mucoid, and ultimately chronic disease if spontaneous resolution or effective treatment does not occur [15]. Structural and histological changes in the epithelial and subepithelial layers of the ME are believed to drive this progression, with luminal effusion caused by sustained negative pressure representing one of the earliest markers of disease [21]. From this point onward, the subepithelium and later the epithelium respond to the adverse environment with histological modifications that may become irreversible and define the long-term pathological course [15].

Anatomical characteristics of the pediatric ET further contribute to this vulnerability. The ET undergoes significant growth throughout childhood, with a linear increase in cartilage volume of approximately 20 mm<sup>3</sup> per year until early adulthood [16]. Adult ET length is nearly twice that observed before seven years of age [17]. In young children, the ET is also more horizontally oriented, with angles as low as 10°, compared with 30–45° in adults, facilitating reflux of nasopharyngeal secretions and pathogens into the ME [18]. This anatomical configuration reduces the mechanical efficiency of the tensor veli palatini muscle and increases susceptibility to ME inflammation and infection. Additional features—including a narrower ET lumen, higher cartilage cell density with reduced elastin content, smaller Ostmann's fat pad, and increased mucosal folds—further compromise ET function in children and predispose them to ME pathology [17].

Chronic inflammatory conditions such as allergic disease, laryngopharyngeal reflux, or incomplete resolution of AOM may perpetuate ET dysfunction, maintaining a negative pressure environment within the ME [18]. Prolonged ET dysfunction promotes continued transudation of fluid from the mucosa and movement of intravascular fluid into the ME lumen, thereby sustaining effusion and increasing the risk of chronic disease [19].

### **Predisposing Factors for Acute Otitis Media and Progression to Otitis Media with Effusion**

Several environmental, behavioral, and inflammatory factors predispose children to acute otitis media (AOM) and may contribute to the persistence of middle ear effusion and progression to otitis media with effusion (OME). Artificial feeding is a well-established risk factor, as infants who are not breastfed or are exclusively bottle-fed present a higher incidence of both AOM and OME [21,22]. Bottle-feeding is commonly associated with a more horizontal feeding position, which facilitates

reflux of milk into the Eustachian tube (ET), increasing the likelihood of fluid and pathogen entry into the middle ear. In addition, the absence of protective immune components found in breast milk—particularly secretory immunoglobulin A (IgA)—reduces mucosal defense against respiratory and middle ear infections [22].

Oral habits, including pacifier use and digit sucking, have been associated with an increased risk of AOM through their impact on oropharyngeal pressure dynamics and ET function [23]. Pacifier use is linked to a 20–30% higher risk of AOM and may promote disease through several mechanisms, including bacterial contamination of pacifier surfaces, which favors nasopharyngeal colonization by pathogens such as *Streptococcus pneumoniae*, and reduced spontaneous swallowing, a physiological trigger for ET opening and middle ear pressure equalization [23]. Digit sucking may exert similar effects, although the available evidence supporting this association is less robust [23].

Early exposure to respiratory pathogens represents another important risk factor. Children attending daycare centers or group childcare settings experience two to three times more episodes of AOM during their first year of life compared with those cared for at home [24]. This increased susceptibility is closely related to group size and higher rates of transmission of common pathogens, including *Streptococcus pneumoniae* and *Haemophilus influenzae*, leading to recurrent upper respiratory tract infections and sustained nasopharyngeal colonization [24].

Passive exposure to tobacco smoke is strongly associated with both AOM and OME, increasing the risk by approximately 30–50% [25]. Secondhand smoke impairs ciliary function within the ET, compromising secretion clearance and middle ear ventilation, while simultaneously increasing nasopharyngeal colonization by pathogenic bacteria. In addition, tobacco smoke exacerbates inflammation of the respiratory mucosa, creating a favorable environment for recurrent infection and persistent effusion [25].

Inflammatory conditions of the upper airway, particularly allergic rhinitis, play a significant role in ET dysfunction and effusion development. Nasopharyngeal mucosal edema caused by allergic inflammation can obstruct the ET, impair middle ear ventilation, and promote fluid accumulation [26]. Children with allergic rhinitis have an approximately 1.8- to 2-fold increased risk of developing OME, especially in cases of persistent disease, due to chronic nasopharyngeal inflammation and ongoing ET dysfunction [26,27]. Chronic nasal obstruction, frequently associated with adenoid hypertrophy, may also lead to habitual mouth breathing, which alters oropharyngeal pressure patterns and further compromises ET function [29].

The association between swimming and OME remains controversial. Some evidence suggests that frequent exposure to chlorinated pool water may irritate the upper airways, particularly in atopic children, predisposing them to nasopharyngeal inflammation and subsequent ET dysfunction [28]. Finally, recurrent viral upper respiratory infections, including those caused by respiratory syncytial virus and rhinovirus, induce inflammation of the ET mucosa and facilitate secondary bacterial colonization of the nasopharynx, thereby increasing the risk of OME development [30].

### **Middle Ear Ventilation and Diagnostic Assessment**

Middle ear (ME) ventilation is a dynamic process, as ME aeration conditions fluctuate over time rather than remaining constant. Accurate assessment of these variations is essential for the diagnosis and monitoring of otitis media with effusion (OME), particularly in children, in whom clinical manifestations are often subtle [3].

Clinical evaluation alone is frequently insufficient for diagnosing OME. The condition predominantly affects children under seven to eight years of age and is commonly associated with nonspecific symptoms, including attention and concentration difficulties, learning problems, hyperactivity, and behavioral changes. Subjective auditory complaints such as hearing loss, aural fullness, or tinnitus are uncommon in this age group [3]. Potential consequences of persistent OME include delayed speech and language development, articulation errors, and poor academic performance, underscoring the importance of objective assessment methods [3].

Otoscopy remains an essential component of the diagnostic evaluation, allowing visual inspection of the tympanic membrane (TM). Conventional otoscopy, pneumatic otoscopy, fiber-optic or video otoscopy, and microscopic examination may all be used to assess TM appearance and mobility. In OME, the TM typically appears opaque and thickened, with an absent or diffuse light reflex; it may be retracted and may show air bubbles or fluid levels ranging from clear to yellow or rust-colored [3]. Pneumatic otoscopy can provide indirect information regarding TM and ossicular chain mobility. However, otoscopy is inherently subjective and does not allow precise determination of ME pressure. Regardless of the technique used, otoscopic findings can only suggest normal or abnormal ME status and cannot accurately quantify ME aeration [3].

Audiological assessment is therefore a critical component of OME evaluation and should be tailored to the child's age and level of cooperation. Whenever possible, testing should be performed by an experienced speech-language pathologist or audiologist with expertise in pediatric assessment. Behavioral hearing evaluation may be used in younger children, while conditioned audiometry, speech detection thresholds, and air- and bone-conduction threshold testing with headphones can be performed in older children, including those as young as three to four years of age. Even small air-bone gaps should be carefully interpreted, as they may reflect clinically significant conductive hearing loss associated with ME effusion [3].

Among audiological tools, acoustic immittance testing (tympanometry) plays a central role. Tympanometry is a noninvasive, objective, and rapid examination and may be the only feasible test in very young children. It provides quantitative information about ME aeration by measuring ME pressure. Normal aeration is characterized by a Type A tympanometric curve (0 to -100 daPa), negative ME pressure by a Type C curve ( $\leq -100$  daPa), and the presence of effusion by a flat Type B curve, typically observed at pressures between -400 and -600 daPa [31]. By translating otoscopic impressions into numerical values, tympanometry serves as both a diagnostic and monitoring tool. Serial tympanometric measurements allow precise assessment of disease progression or resolution by documenting whether ME aeration remains stable, deteriorates, or improves over time [31].

Assessment by a speech-language pathologist is also fundamental, as it evaluates speech and language acquisition and helps guide therapeutic decision-making. A normal speech and language evaluation may suggest recent-onset OME that has not yet caused developmental impairment, allowing for a more conservative medical approach. In contrast, abnormal findings indicate interference with learning and communication processes and warrant timely intervention to minimize long-term consequences. Optimal hearing is critical for oral language acquisition, and early childhood represents a sensitive period during which auditory "windows of opportunity" open and close rapidly, making early diagnosis and appropriate management essential [31].

#### Prevention Priorities in Otitis Media with Effusion

Preventive strategies in OME focus on minimizing both local middle ear damage and the functional consequences of auditory impairment. Persistent negative ME pressure may lead to structural alterations, including horizontalization of the malleus handle, retraction pocket formation, mucosal changes within the ME, and, in advanced cases, cholesteatoma development secondary to TM retraction. Although less common, long-standing disease may also contribute to sensorineural hearing loss [31].

Equally important is the prevention of auditory dysfunction caused by sound attenuation and distortion. In OME, negative ME pressure is not static but fluctuates over time, resulting in variable and unpredictable hearing levels. For children in critical stages of oral language development, this auditory instability can be particularly harmful, leading to delayed speech and language acquisition, restricted vocabulary growth, and poor academic performance. Psychosocial and behavioral consequences may also emerge, including social withdrawal, reduced auditory attention, difficulty following instructions, poor concentration, and compensatory hyperactivity or restlessness. In some cases, motor coordination problems, such as clumsiness or frequent falls, may occur, potentially related to vestibular dysfunction or attentional deficits [31].

#### Severity of Hearing Loss and Psychosocial Impact in Otitis Media with Effusion

The hearing loss associated with otitis media with effusion (OME) is typically conductive, bilateral, and of mild to moderate degree, with average thresholds ranging from 18 to 35 dB HL at frequencies critical for speech perception [32]. Although often labeled as "mild," this level of hearing loss has clinically significant consequences, particularly in noisy domestic and educational environments. Children with OME frequently demonstrate impaired word recognition, especially in acoustically challenging settings such as classrooms, where background noise and reverberation are common [32].

Between the ages of 6 and 11 years, children with hearing thresholds within this range show significantly poorer cognitive, linguistic, and reading skills compared with peers who have better hearing sensitivity [33]. These findings highlight that the functional impact of OME-related hearing loss extends well beyond simple audibility deficits.

#### Functional Impact According to Degree and Configuration of Hearing Loss

Minimal hearing loss (16–25 dB HL) is associated with difficulty perceiving distant or soft speech, such as a teacher's voice at distances greater than one meter. In noisy classrooms, this degree of loss may result in the omission of approximately 10% of the speech signal, which is particularly detrimental during early school years when verbal instruction predominates [32].

Mild hearing loss (26–40 dB HL) leads to more pronounced speech perception deficits, with losses of approximately 25–40% of speech information at thresholds around 30 dB HL, increasing to as much as 50% at thresholds between 35 and 40 dB HL. High-frequency consonants are especially affected. In the absence of amplification, children with mild hearing loss often struggle during group discussions, particularly when speakers are not facing them or when the listening environment is reverberant or noisy [32].

Unilateral hearing loss, characterized by normal hearing in one ear and minimal to mild loss in the other, also carries important functional consequences. These children experience deficits in sound localization and significant difficulty understanding speech in noise, particularly in classrooms where the signal of interest may originate from different

directions. Speech presented on the affected side may be poorly detected even at normal conversational levels [32].

### Fluctuating Conductive Hearing Loss in OME

A defining characteristic of OME-related hearing loss is its fluctuation over time. Negative middle ear (ME) pressure resulting from Eustachian tube dysfunction leads to hearing loss that varies in severity, improving or worsening as ME aeration changes. In addition to attenuation, negative pressure causes sound distortion, which also fluctuates and further compromises speech perception. Importantly, negative ME pressure invariably produces some degree of conductive hearing loss [34].

Classic experimental work by Feldmann [34] demonstrated that a negative ME pressure of approximately  $-100$  daPa (or  $-100$  mmH<sub>2</sub>O) results in a conductive hearing loss of about 5 to 10 dB, predominantly affecting low frequencies below 1 kHz. This occurs due to reduced efficiency of sound transmission through the ossicular chain. When ME pressure reaches  $-400$  daPa, hearing loss becomes more pronounced, potentially ranging from 15 to 25 dB, again mainly at low frequencies. At this level, increased tension of the tympanic membrane and stiffness of the tympano-ossicular system further impair sound transmission [35].

Clinically, a child with bilateral hearing thresholds around 28 dB due to OME may struggle to hear soft speech, barely perceive normal conversational speech, or fail to detect salient sounds such as a crying infant [35]. These examples illustrate that the real-life impact of “mild” conductive hearing loss can be substantial.

### Developmental and Cognitive Consequences

The functional impact of mild hearing loss is consistently underestimated. Children aged 6 to 11 years with pure-tone averages between 15 and 30 dB HL demonstrate significantly poorer cognitive, linguistic, and reading abilities than children with better hearing thresholds [36]. The degree of hearing loss associated with OME may range from no measurable loss to moderate loss, reaching levels as high as 55 dB HL in some cases [36]. Although the average hearing loss in children with OME is approximately 28 dB, nearly 20% exhibit thresholds of 35 dB or greater [37,38].

The first and second years of life represent critical periods for speech, language, and cognitive development. Any degree of hearing loss during these sensitive windows may have long-lasting consequences, including poorer academic performance later in childhood [37–39]. Fluctuating hearing loss caused by OME during the first year of life degrades the quality of auditory input, particularly speech signals. Early auditory deprivation may lead to altered perception of the linguistic code, resulting in inadequate phonological representation and impaired early language acquisition. Additional consequences include atypical auditory electrophysiological and psychoacoustic processing, attention and behavioral difficulties, deficits in auditory selective attention, higher-order auditory processing disorders, and reading difficulties. These effects may be further compounded by genetic susceptibility and environmental factors such as parental interaction and stimulation [37,38].

### Psychosocial and Neurodevelopmental Effects

From a neuropsychological perspective, Chase [41] evaluated infants with otitis media and observed their interactions with caregivers. The study demonstrated that children with more frequent episodes of otitis media were less attentive, less responsive during structured parent–child interactions, and more likely to exhibit irregular sleep patterns, feeding difficulties, and delayed sphincter control. Parents were also affected, experiencing poorer sleep quality, which reduced their capacity to provide

structured learning activities and to sustain their child’s attention during interactions.

Longitudinal data from Hall, Munro, and Heron [42], derived from the Avon Longitudinal Study of Parents and Children (ALSPAC), further elucidate the developmental impact of OME. This large cohort study assessed developmental changes in speech recognition thresholds (SRT) between 2 and 5 years of age in children with different otitis media statuses. In children without otitis media, mean SRT improved by approximately 5 dB between 31 and 43 months and between 31 and 61 months, reaching an average of 23 dB at five years of age and improving further to a mean adult SRT of 18.6 dB. The total gain in SRT from age two to adulthood was approximately 11 dB [42].

Children with unilateral OME demonstrated SRTs that were 4 to 5 dB worse than those of children without otitis media, likely reflecting the loss of binaural hearing advantages, which normally confer an approximate 3 dB benefit for speech perception. In contrast, children with bilateral OME showed significantly impaired SRTs at all ages ( $p < 0.001$ ), with an average deficit of 15 dB compared with children without otitis media, independent of age. The greatest effect was observed at 31 months, reflecting poorer baseline auditory development at this stage. Functionally, a five-year-old child with unilateral OME performed similarly on speech perception tasks to a three-year-and-seven-month-old child without otitis media. Importantly, children who had normal tympanometric findings at five years of age did not exhibit long-term SRT deficits, underscoring the importance of early resolution of middle ear dysfunction [42].

### Surgical Management: Ventilation Tube Insertion

Ventilation tube (VT) insertion remains the most widely used surgical intervention for otitis media with effusion (OME) in children, particularly in cases of persistent disease associated with hearing loss or developmental risk. Evidence consistently demonstrates that VT placement provides meaningful short-term benefits in middle ear aeration, hearing thresholds, and quality of life.

The landmark study by Hellström, Groth, Jørgensen et al. (2011) [43] showed that VT insertion is associated with significant short-term improvements in children’s health-related quality of life. Meta-analytic data indicate that VT placement reduces the prevalence of OME by approximately 32% during the first postoperative year and improves average hearing thresholds by 5 to 12 dB [44]. In addition, a non-randomized study demonstrated improved caregiver perception of speech and language outcomes following VT insertion, particularly among children with pre-existing developmental delays, highlighting the potential benefit of early auditory rehabilitation in vulnerable populations [45].

### Complications Associated with Ventilation Tubes

Complications related to VT insertion are relatively common but are generally transient or of limited functional significance. The most frequently reported adverse event is otorrhea. Approximately 16% of children experience otorrhea within the first four postoperative weeks, and up to 26% develop otorrhea at some point while the VT remains in place [46]. In the United States, most ventilation tubes remain functional for 8 to 18 months, during which approximately 7% of children experience recurrent otorrhea episodes [46].

A prospective Dutch study reported a higher otorrhea incidence (52%) when children were followed closely and systematically; however, only 3.9% of cases evolved into chronic otorrhea lasting three months or longer. Identified risk factors included younger age, VT placement for



recurrent acute otitis media (RAOM), the presence of older siblings in the household, and frequent upper respiratory tract infections [47].

Other complications occur less frequently and include obstruction of the VT lumen (7–10%), granulation tissue formation (approximately 4%), premature tube extrusion (4%), and medial displacement of the VT into the middle ear cavity (<0.5%) [46,48]. Structural tympanic membrane (TM) sequelae, such as tympanosclerosis, focal atrophy, or retraction pockets, are often detected on follow-up but are usually non-progressive and clinically insignificant.

### Persistent Tympanic Membrane Perforation

Persistent TM perforation is among the most clinically relevant long-term complications of VT insertion. The reported incidence varies from 1% to 6% of ears following tube extrusion [49]. Large population-based data from a Medicaid cohort demonstrated a persistent perforation rate of approximately 3% seven years after VT insertion [50]. Similarly, O'Neil, Cassidy, Link, and Kerschner [51] reported a 1% rate of persistent perforation after spontaneous VT extrusion, with 2.6% of children requiring surgical removal of retained tubes that failed to extrude spontaneously. Repeat VT insertion and older age at the time of surgery were identified as significant risk factors for persistent perforation. When spontaneous closure does not occur, surgical repair with myringoplasty or tympanoplasty may be required.

Overall, although VT-related complications are not uncommon, the majority are self-limited and must be weighed against the potential benefits of improved hearing, auditory stability, and developmental outcomes in appropriately selected children.

### Conclusion

Otitis media with effusion is a highly prevalent pediatric condition primarily resulting from Eustachian tube dysfunction and influenced by anatomical, immunological, and environmental factors. Although often self-limiting, OME may lead to fluctuating conductive hearing loss with significant consequences for speech and language acquisition, academic performance, and psychosocial development, particularly during critical periods of early childhood.

Accurate diagnosis, with emphasis on tympanometry and age-appropriate audiological assessment, is essential to identify children at increased risk of developmental impairment. Management should be individualized, incorporating careful observation, mitigation of modifiable risk factors, and timely surgical intervention when indicated. Tympanotomy with ventilation tube insertion remains an effective treatment for persistent OME associated with documented hearing loss or developmental impact, offering meaningful short-term improvements in hearing and quality of life.

Early recognition and appropriately timed intervention play a pivotal role in minimizing long-term auditory, cognitive, and educational sequelae, underscoring the importance of a structured, evidence-based approach to the management of otitis media with effusion.

### References

1. Stool SE, Berg AO, Berman S, et al. (1994). Otitis Media with Effusion in Young Children: Clinical Practice Guideline No. 12. Rockville, MD: Agency for Healthcare Research and Quality; AHCPR publication 94-0622.
2. Berkman ND, Wallace IF, Steiner MJ, et al. (2013). Otitis Media With Effusion: Comparative Effectiveness of Treatments. Rockville (MD): Agency for Healthcare Research and Quality (US)
3. Rosenfeld RM, Shin JJ, Schwartz SR, Coggins R, Gagnon L, Hackell JM, et al. (2016). Clinical practice guideline: otitis media with effusion (update). *Otolaryngol Head Neck Surg*;154:S1–41.
4. NICE. Otitis Media with effusion in under 12s: surgery Guidance and guidelines; 2008
5. SFORL. Prise en charge thérapeutique des OSM de l'enfant. Recommandations de pratiques cliniques; 2016 Pediatric Group. ENT society of CMA. *Chin J Otorhinlaryngol Head Neck Surg* 2008.
6. Bluestone CD, Swarts JD. Human evolutionary history: consequences for the pathogenesis of otitis media. *Otolaryngol Head Neck Surg*. 2010;143(6):739-744.
7. Teele DW, Klein JO, Rosner BA. Epidemiology of otitis media in children. *Ann Otol Rhinol Laryngol Suppl*. 1980 May-Jun;89(3 Pt 2):5-6.
8. Nelson WE, Kliegman R, W J, Behrman RE, Robert Charles Tasker, Shah SS, et al. Nelson textbook of pediatrics. 21st ed. Philadelphia: Elsevier, Cop; 2019.
9. Murphy K, Weaver C. Janeway's Immunobiology. 9th ed. New York, NY, USA: Garland Science, Taylor & Francis Group, LLC; 2017.
10. Manual de Normas e Procedimentos para Vacinação / Ministério da Saúde, Secretaria de Vigilância em Saúde, Departamento de Vigilância das Doenças Transmissíveis. – Brasília: Ministério da Saúde, 2014.176 p.
11. Canali, Inesângela. Estudo da função da tuba de eustáquio em pacientes com retrações da membrana timpânica e em indivíduos normais. Tese de Mestrado. Orientador: Sady Selaimen da Costa. Dissertação (Mestrado). Universidade Federal do Rio Grande do Sul, Faculdade de Medicina, Programa de Saúde da Criança e do Adolescente, Porto Alegre, BR-RS, 2013.
12. Levy D, Herman M, Luntz M, Sadé J. Direct demonstration of gas diffusion into the middle ear. *Acta Otolaryngol*. 1995 Mar;115(2):276-278.
13. Licameli GR. The eustachian tube. Update on anatomy, development, and function. *Otolaryngol Clin North Am*. 2002 Aug;35(4):803-809.
14. Cinamon U. Passive and dynamic properties of the eustachian tube: quantitative studies in a model. *Otol Neurotol*. 2004 Nov;25(6):1031-1033.
15. Yoon TH, Paparella MM, Schachern PA, Lindgren BR. (1990). Morphometric studies of the continuum of otitis media. *Ann Otol Rhinol Laryngol Suppl*. Jun;148:23-27.
16. Takasaki K, Sando I, Balaban CD, Ishijima K (2000) Postnatal development of eustachian tube cartilage. A study of normal and cleft palate cases. *Int J Pediatr Otorhinolaryngol* 52:31–36.
17. Canali I, Petersen Schmidt Rosito L, Siliprandi B, Giugno C, (2017). Selaimen da Costa S. Assessment of Eustachian tube function in patients with tympanic membrane retraction and in normal subjects. *Braz J Otorhinolaryngol*. Jan-Feb;83(1):50-58.
18. Bluestone CD (2005). Eustachian tube structure, function, role in otitis media. BC Decker, Hamilton.
19. Doyle WJ, Seroky JT, Alper CM. (1995). Gas exchange across the middle ear mucosa in monkeys. Estimation of exchange rate. *Arch Otolaryngol Head Neck Surg*. Aug;121(8):887-892.
20. Bluestone CD, Hebda PA, Alper CM, Sando I, Buchman CA, Stangerup SE, Felding JU, Swarts JD, Ghadiali SN, Takahashi H. (2005). Recent advances in otitis media. 2. Eustachian tube, middle ear, and mastoid anatomy; physiology,

- pathophysiology, and pathogenesis. *Ann Otol Rhinol Laryngol Suppl.* Jan;194:16-30.
21. Bowatte G, Tham R, Allen K, Tan D, Lau M, Dai X, et al. (2015).Breastfeeding and childhood acute otitis media: a systematic review and meta-analysis. *Acta Paediatrica* [Internet]. Nov 4;104(S467):85–95.
  22. Posenato Garcia L. (2016).The Lancet: série sobre amamentação. *Epidemiologia e Serviços de Saúde.* Jan;25(1):1–10.
  23. Hanafin S, Griffiths P. (1995).O uso de chupeta causa infecções de ouvido em crianças pequenas? 2002. Em: Banco de Dados de Resumos de Revisões de Efeitos (DARE): Revisões avaliadas pela qualidade [Internet]. York (Reino Unido): Centro de Revisões e Disseminação (Reino Unido);
  24. Lieberthal AS, Carroll AE, Chonmaitree T, Ganiats TG, Hoberman A, Jackson MA, et al. (2013).The diagnosis and management of acute otitis media. *American Academy of Pediatrics* [Internet].;131(3):e964–999.
  25. Office on Smoking and Health. The Health Consequences of Involuntary Exposure to Tobacco Smoke [Internet]. Nih.gov. Centers for Disease Control and Prevention (US);.
  26. Scadding GK, Kariyawasam HH, Scadding G, Mirakian R, Buckley RJ, Dixon T, et al. (2017).BSACI guideline for the diagnosis and management of allergic and non-allergic rhinitis (Revised Edition 2017; First edition 2007). *Clinical & Experimental Allergy.* Jul;47(7):856–889.
  27. Juszcak HM, Loftus PA. (2020).Role of Allergy in Eustachian Tube Dysfunction. *Current Allergy and Asthma Reports.* Jul 9;20(10).
  28. Bernard A, Nickmilder M, Voisin C. (2008).Outdoor swimming pools and the risks of asthma and allergies during adolescence. *European Respiratory Journal.* May 14;32(4):979–988.
  29. Cassano P, Gelardi M, Cassano M, Maria Luisa Fiorella, Fiorella R. (2003).Adenoid tissue rhinopharyngeal obstruction grading based on fiberendoscopic findings: a novel approach to therapeutic management. *Dec 1;67(12):1303–1309.*
  30. Chonmaitree T, Trujillo R, Jennings K, Alvarez-Fernandez P, Patel JA, Loeffelholz MJ, et al. (2016).Acute Otitis Media and Other Complications of Viral Respiratory Infection. *PEDIATRICS* [Internet]. Mar 28;137(4):e20153555–5.
  31. Rosenfeld RM, Shin JJ, Schwartz SR, Coggins R, Gagnon L, Hackell JM, Hoelting D, Hunter LL, Kummer AW, Payne SC, Poe DS, Veling M, Vila PM, Walsh SA, Corrigan MD. (2016).Clinical Practice Guideline: Otitis Media with Effusion (Update). *Otolaryngol Head Neck Surg.* Feb;154(1 Suppl):S1–S41.
  32. Anderson, K. L.(1991.).Hearing conservation in the public schools revisited.Seminars in Hearing, 12(4), pp. 361-363
  33. Adaptado por Bernero, R.J. & Bothwell, H.,(1996).Relationship of Hearing Impairment to Education Needs.
  34. Moore DR, Zobay O, Ferguson MA. (2020).Minimal and mild hearing loss in children: association with auditory perception, cognition, and communication problems. *Ear Hear.*;41(4):720-732.
  35. Feldmann H. (1971).Homolateral and Contralateral Masking of Tinnitus by Noise-Bands and by Pure Tones. *International Journal of Audiology.* Jan;10(3):138–144.
  36. Rabinowitz WM. (1981).Measurement of the acoustic input immittance of the human ear. *The Journal of the Acoustical Society of America.* Oct;70(4):1025–1035.
  37. Rosenfeld RM. (2005). A Parent’s Guide to Ear Tubes. PMPH-USA;
  38. Gravel JS. (2003).Hearing and auditory function. In: Rosenfeld RM, Bluestone CD, eds. *Evidence-Based Otitis Media.* 2nd ed. BC Decker Inc.;342-359.
  39. Fria TJ, Cantekin EI, Eichler JA. (1985).Hearing acuity of children with otitis media with effusion. *Arch Otolaryngol.*;111(1):10-16).
  40. Kaplan GJ, Fleshman JK, Bender TR, Baum C, Clark PS.( 1973). Long-term effects of otitis media: A ten-year cohort study of Alaskan Eskimo children. *Pediatrics*;52(4):577-585.
  41. Sak RJ, Ruben RJ. (1981).Recurrent middle ear effusion in childhood: Implications of temporary auditory deprivation for language and learning. *Ann Otol Rhinol Laryngol*;90(6 pt 1):546-551.
  42. Teele DW, Klein JO, Chase C, Menyuk P, Rosner BA. (1990).Otitis media in infancy and intellectual ability, school achievement, speech, and language at age 7 years. Greater Boston Otitis Media Study Group. *J Infect Dis*;162(3):685-694.
  43. Chase C. In: Iavey RD, Klein JO, (1992).Hearing Loss in Childhood: A Primer. Report of the 102nd Ross Conference on Pediatric Research. Columbus, Ohio: Ross Laboratories; 88-94.
  44. Hall AJ, Munro KJ, Heron J. (2007).Developmental changes in word recognition threshold from two to five years of age in children with different middle ear status. *Int J Audiol.* Jul;46(7):355-361.
  45. Hellström S, Groth A, Jo`rgensen F, et al.( 2011). Ventilation tube treatment: a systematic review of the literature. *Otolaryngol Head Neck Surg.*;145(3):383-395.
  46. Browning GG, Rovers MM, Williamson I, Lous J, Burton MJ. (2005).Grommets (ventilation tubes) for hearing loss associated with otitis media with effusion in children. *Cochrane Database Syst Rev.* 2010;(10):CD001801; Rovers MM, Black N, Browning GG, Maw R, Zielhuis GA, Haggard MP. Grommets in otitis media with effusion: an individual patient data meta-analysis. *Arch Dis Child.*;90(5): 480-485.
  47. Rosenfeld RM, Jang DW, Tarashansky K. (2011).Tympanostomy tube outcomes in children at-risk and not at-risk for developmental delays. *Int J Pediatr Otorhinolaryngol.*;75(2):190-195.
  48. Kay DJ, Nelson M, Rosenfeld RM. (2001).Meta-analysis of tympanostomy tube sequelae. *Otolaryngol Head Neck Surg.*;124(4):374-380.
  49. van Dongen TM, van der Heijden GJ, Freling HG, Venekamp RP, Schilder AG. (2013).Parent-reported otorrhea in children with tympanostomy tubes: incidence and predictors. *PloS One.* 8(7):e69062.
  50. Conrad DE, Levi JR, Theroux ZA, Inverso Y, Shah UK. (2014).Risk factors associated with postoperative tympanostomy tube obstruction. *JAMA Otolaryngol Head Neck Surg.*;140(8):727-730.
  51. Hellström S, Groth A, Jørgensen F, et al.( 2011).Ventilation tube treatment: a systematic review of the literature. *Otolaryngol Head Neck Surg.*;145(3):383-395.
  52. Alrwisan A, Winterstein AG, Antonelli PJ. (2016).Epidemiology of persistent tympanic membrane perforations subsequent to tympanostomy tubes assessed with real world data. *Otol Neurotol*;37(9):1376-1380.

53. O’Niel MB, Cassidy LD, Link TR, Kerschner JE.(2015).  
Tracking tympanostomy tube outcomes in pediatric patients

with otitis media using an electronic database. Int J Pediatr  
Otorhinolaryngol. 79(8):1275-1278.



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