Chapter

Common Ear, Nose, and Throat Disorders in Childhood

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Abstract

This chapter provides a comprehensive overview of the most prevalent Ear, Nose, and Throat (ENT) disorders encountered in pediatric populations. Drawing insights from medical literature, the chapter delves into the multifaceted landscape of ENT disorders affecting children, exploring their etiology, clinical manifestations, diagnostic approaches, and contemporary management strategies. The discussion encompasses a wide spectrum of conditions, including otitis media, adenotonsillitis, and pediatric stridor. Emphasis is placed on the unique challenges posed by these disorders in the pediatric population, considering factors that affect physical and psychosocial development and age-specific treatment considerations. This chapter also highlights the importance of interdisciplinary collaboration between pediatricians, otolaryngologists, and other healthcare professionals in delivering optimal care for children with ENT disorders. Through a synthesis of evidence-based recommendations and practical clinical insights, this chapter aims to serve as a valuable resource for healthcare practitioners and medical students involved in the care of pediatric patients with ENT conditions.

Keywords: adenotonsillitis, acute otitis media, pediatric stridor, hearing loss, otitis media with effusion

1. Introduction

Children, with their unique physiology and evolving immune systems, are predisposed to a spectrum of Ear, Nose, and Throat (ENT) disorders that demand a specialized approach. Within the realm of pediatric healthcare, the intricate dynamics of ENT disorders play a significant role in shaping their overall health and quality of life. This chapter aims to illuminate the landscape of common ENT disorders in childhood, providing a nuanced understanding of the intricate interplay between pediatric anatomy and physiology and the myriad challenges that can arise in this vulnerable population. Etiopathogenesis, clinical manifestations, and contemporary management strategies of common ENT disorders affecting the pediatric population have also been discussed.

This chapter encompasses a diverse array of conditions, including acute otitis media, evaluation and management of pediatric stridor, and adenotonsillitis. A holistic and interdisciplinary approach between pediatricians, otolaryngologists,

audiologists, and other healthcare professionals is essential in navigating the intricate web of symptoms, diagnostic considerations, and treatment modalities.

2. Acute otitis media

2.1 Definition

Acute otitis media (AOM) is defined as the rapid onset of signs and symptoms, such as otalgia and fever, due to acute inflammation of the middle ear cleft. Symptoms include pain in the ear, ear discharge, irritability, insomnia, and anorexia. Signs include fever, otorrhea, erythema of the tympanic membrane (TM), full or bulging opaque TM, and impaired TM mobility [1].

It emerges as a widely prevalent condition in early childhood, often leading to referral to otolaryngologists. While the majority of primary healthcare providers effectively manage most AOM cases, children experiencing recurring episodes, severe symptoms, or AOM-related complications which may need swift evaluation by otolaryngologists, potentially leading to surgical interventions. Despite the widespread occurrence of AOM and the use of tympanostomy tubes, achieving a consensus on the optimal surgical approach for children with AOM remains an ongoing challenge.

As we delve into the key concepts in the management of AOM in children, we particularly focus on the evolving microbiological trend observed over the past 2 decades. Emphasis is placed on the crucial aspect of accurate diagnosis, employing stringent criteria and refining the selection of children deemed suitable for observation without immediate antibiotic intervention upon initial AOM diagnosis.

2.2 Epidemiology

AOM stands as a major health concern among children, with substantial implications for both public health and healthcare expenditure in many countries. Peak incidence is between 6 and 24 months of age [2, 3]. AOM incidence rates vary across different age groups, reflecting the susceptibility and prevalence within each demographic. Incidence rates range from 20 to 30% or higher in children less than 2 years of age, depending on geographical and socioeconomic factors [4]. AOM remains common in preschool-aged children, although the incidence tends to decrease slightly compared to infants. Incidence rates in this age group typically range from 15 to 25%, with variations influenced by factors such as exposure to environmental pathogens and daycare attendance [5]. The incidence of AOM tends to decline further in school-going children between 6 and 12 years of age, with incidence rates ranging from 5 to 15% [6]. AOM is less common in adolescents and adults compared to younger age groups. Incidence rates in adolescents and adults are generally lower, often falling below 5% [7]. Another postulated reason for the decline is that as children become older, maturation of their immune system occurs. It is important to understand that these incidence rates may vary based on geographical location, socioeconomic factors, access to healthcare, vaccination rates, and other variables. Additionally, modifications in antibiotic prescribing practices and the prevalence of bacterial strains resistant to antibiotics can influence AOM incidence rates over time.

Antibiotics, prescribed more frequently for AOM than any other childhood illness, play a central role in its management [8]. The epidemiology of AOM has witnessed

noteworthy changes in trends over the past decade. Despite the prevalence of the condition, there has been a decrease in physician visits for suspected AOM globally and in the United States [5, 9]. The reasons for this decline are multifaceted, potentially influenced by public awareness campaigns regarding the viral etiology and mode of spread of Upper Respiratory Tract Infections (URTI) and the launch of vaccines such as the 7-valent pneumococcal vaccine (PCV7) and influenza vaccines [10]. The dissemination and adoption of best clinical practices and guidelines may also have contributed to the observed decrease in clinician visits.

2.3 Pathophysiology

AOM frequently, though not always, arises following a viral URTI [11]. Inflammatory processes cause edema in the nasal cavities and nasopharynx, leading to blockage of the eustachian tube. This obstruction leads to the development of negative pressure in the middle ear, followed by middle ear effusion. Microbe-laden secretions from the nasal and nasopharyngeal mucosa migrate into the middle ear due to the pressure differential, becoming entrapped and potentially fostering bacterial replication and infection. Young children face an elevated risk for AOM due to heightened viral exposure, immunological naivety, and inherent eustachian tube dysfunction [12]. Recurrent attacks of sore throat, URTIs, and exanthematous fevers like measles, diphtheria or whooping cough, adenotonsillitis, chronic rhinosinusitis, allergic rhinitis, cleft palate can act as predisposing factors.

The 2013 American Academy of Pediatrics (AAP) guidelines advocate for various preventive health interventions to decrease the incidence of AOM. Over the past 2 decades, the microbiology of AOM has transformed and been influenced by the widespread adoption of pneumococcal vaccination programs. Despite *Streptococcus pneumoniae*, nontypeable *Haemophilus influenza*, and *Moraxella catarrhalis* persisting as the major bacterial culprits, the introduction of the heptavalent PCV7 in 2000 led to a decline in the recovery of *Streptococcus pneumoniae* in AOM cases. However, nonvaccine pneumococcal serotypes have emerged in tympanocentesis and nasopharyngeal colonization studies, equating the incidence of *Streptococcus pneumoniae* with that of *Hemophilus influenza*, while *Moraxella catarrhalis* becomes less frequent. The subsequent licensing of the 13-valent *Streptococcus pneumoniae* vaccine (PCV13) in 2010 is expected to further modify the microbiological landscape of AOM. PCV7 vaccination led to a decline in physician visits for AOM, though subsequent serotype replacement emerged, necessitating consideration for PCV13 vaccination [13–16]. Furthermore, the influenza vaccine is advised for children above 6 months of age. Given the association between influenza and AOM, particularly due to upper respiratory mucosa inflammation, the influenza vaccine can significantly decrease the frequency of AOM with approximately 55% efficacy.

2.4 Pathological stages of AOM and clinical features

2.4.1 Stage of tubal occlusion

The initial phase involves tubal occlusion, marked by nasopharyngeal edema obstructing the eustachian tube, resulting in negative intratympanic pressure. TM retraction and subtle effusion characterize this stage, accompanied by minor symptoms of deafness and earache without notable fever.

2.4.2 Stage of presuppuration

Advancing to the presuppuration stage, prolonged tubal occlusion invites pyogenic organisms, instigating inflammation in the middle ear. Intense earache, deafness, and tinnitus become prominent, with a feverish and restless child. Signs include a congested TM and a cart-wheel appearance due to blood vessel prominence. Tuning fork tests confirm conductive deafness (**Figure 1**).

2.4.3 Stage of suppuration

The subsequent suppuration stage is characterized by the formation of pus in the mastoid air cell system and middle ear, causing excruciating otalgia, increased hard of hearing, and potential fever. TM bulging indicates imminent rupture with a visible yellow spot.

2.4.4 Stage of resolution

Resolution follows TM rupture, leading to symptom relief as pus is evacuated. Patients may present with blood-stained discharge, progressing to mucopurulent, with a small perforation visible. The hyperemia diminishes, returning the membrane to a normal appearance.

2.4.5 Stage of complications

Complications may arise if the infection persists, extending beyond the confines of the middle ear and mastoid air cell system. Intratemporal complications such as acute mastoiditis, subperiosteal abscess, facial paralysis, labyrinthitis, petrositis, and

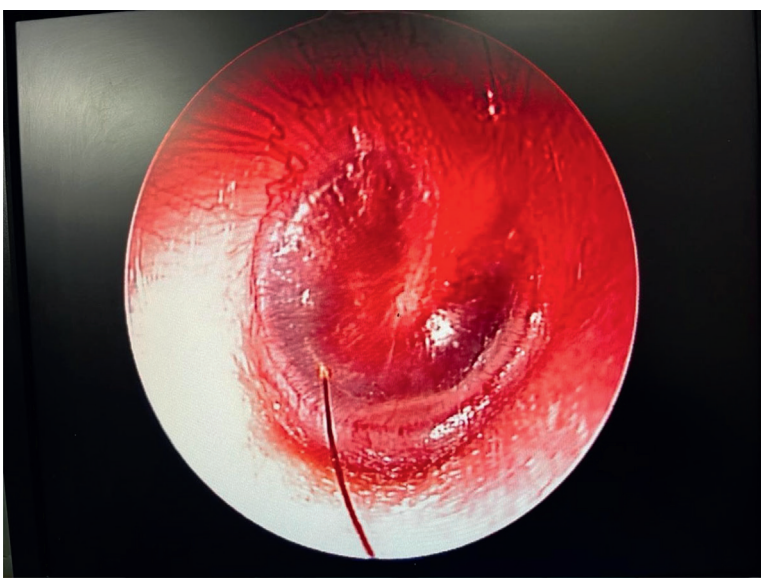

Figure 1.
Stage of presuppuration of AOM—signs of congested tympanic membrane with prominent blood vessels are shown.

intracranial complications such as extradural abscess, meningitis, brain abscess, or lateral sinus thrombophlebitis can manifest in cases of high virulence or poor patient resistance. Vigilant management is crucial to prevent such complications.

2.5 Diagnosis

The diagnosis of AOM poses significant challenges owing to the absence of a definitive gold standard diagnostic method apart from tympanocentesis and culture of middle ear fluid. The lack of a diagnostic benchmark fuels ongoing debate regarding the most accurate clinical means of identifying acute middle ear infections. The complexity arises from the diverse array of clinical presentations that evolve throughout the disease course, compounded by practical difficulties in examining the ears of young children, potentially uncooperative children, particularly when cerumen occlusion is present. Furthermore, the symptomatology of AOM, including fever, otalgia, irritability, and insomnia, often overlap with manifestations of other conditions, such as viral illnesses, further complicating diagnosis. In light of these diagnostic challenges, it becomes imperative to adopt a nuanced approach that integrates clinical judgment with available diagnostic tools and considers individual patient characteristics.

The initial step in diagnosing otitis media involves a thorough physical examination, including the use of an otoscope, preferably a pneumatic otoscopy. Laboratory studies are typically unnecessary. Imaging studies are generally not recommended unless there are concerns about intratemporal or intracranial complications. However, if complications are suspected, high-resolution computed tomography (HRCT) of the temporal bones can identify various conditions, such as mastoiditis, meningitis, or sigmoid sinus thrombophlebitis. Additionally, contrast-enhanced CT of the brain can be combined to rule out intracranial complications. Tympanocentesis, though reserved for extreme or refractory cases, may be employed to confirm the presence of middle ear fluid and to culture pathogens. Additionally, tests like tympanometry and acoustic reflectometry can aid in evaluating middle-ear effusion.

Employing otoscopy, preferably pneumatic otoscopy, and tympanometry, in conjunction with a thorough assessment of symptoms and medical history can improve diagnostic precision. The 2013 AAP guidelines on managing an AOM underscored diagnostic criteria centered on otoscopic examination. This marked a more stringent approach compared to the 2004 guidelines, with a focus on achieving greater diagnostic accuracy. According to the 2013 guidelines, diagnosing AOM is crucial and justified in children presenting with *moderate to severe bulging of the TM, new onset of otorrhea, or mild TM bulging accompanied by recent ear pain or notable TM erythema*. Importantly, the guidelines specify that AOM should not be diagnosed unless middle-ear effusion is present [17–21]. The guidelines also suggested various terminologies of AOM based on severity:

- *Uncomplicated AOM*—AOM without otorrhea
- *Severe AOM*—AOM with the presence of moderate to severe otalgia *or* fever equal to or higher than 39°C
- *Nonsevere AOM*—AOM with the presence of mild otalgia and a temperature below 39°C [17]

2.6 Treatment

2.6.1 Antibiotics of choice and duration

Every infant under 2 years old showing clinical features of AOM should be administered an antibiotic effective against the three primary bacterial pathogens that are implicated in AOM, *S. pneumoniae*, *H. influenzae*, and *M. catarrhalis*. Children above the age of 2 years who have "severe" AOM (i.e., moderate to severe otalgia or fever ≥39°C) should also receive antimicrobial therapy. The option of observation may be considered for a child older than 2 years who has "nonsevere" AOM (i.e., mild otalgia and fever <39°C in the past 24 hours) (**Table 1**).

High-dose amoxicillin, administered at a dosage of 80–90 mg/kg per day, stands as the recommended first-line treatment in the 2013 guidelines for AOM.

For individuals sensitive to penicillin, second- or third-generation cephalosporins, including intramuscular ceftriaxone, represent viable alternatives. Additional agents, particularly relevant for penicillin-sensitive patients or instances of amoxicillin failure, include second and third-generation cephalosporins and clindamycin. Patients exhibiting no improvement within 48–72 hours of their initial treatment regimen should undergo reassessment, with consideration given to alternative therapy. In these situations, the primary treatment choices typically include amoxicillin-clavulanate and intramuscular or intravenous ceftriaxone. Second-line options may involve clindamycin alone or combination therapy using clindamycin along with a third-generation cephalosporin. In challenging cases, tympanocentesis may be contemplated for drainage and culture-directed therapy [17, 22].

2.6.2 Supportive therapy

Analgesics: Once the diagnosis of acute otitis media is established, the goal of treatment is pain management and antibiotics to curb the infectious process. Nonsteroidal anti-inflammatory drugs (NSAIDs) such as acetaminophen and ibuprofen can be used to achieve pain control.

Decongestant nasal drops and antipyretics with antihistaminics are added to reduce edema and congestion of the Eustachian tube and tackle pain.

Tympanostomy tubes or ventilation tubes, also known as grommets, are tubes inserted in the TM following myringotomy (radial incision made in the anteroinferior

Age (months)	Certain diagnosis*	Uncertain diagnosis
<6	• Antibiotics	• Antibiotics
6–24	• Antibiotics	• Antibiotics if severe • Observe if not severe
>24	• Antibiotics if severe • Observe if not severe**	• Observe

*Certain diagnoses: middle-ear effusion, rapid onset, and middle-ear inflammation.
**Nonsevere: mild otalgia; temperature <39°C orally or <39.5°C rectally in past 24 hours.
Adapted from Refs. [17, 18].

Table 1.
Guidelines for management of acute otitis media—American Academy of Pediatrics and American Academy of Family Physicians.

quadrant of TM) and are meant to improve middle-ear ventilation. These are specifically recommended for children "at risk" of developing acute otitis media such as children with craniofacial syndromes, cleft palate, and Down's syndrome [23].

3. Pediatric stridor

3.1 Definition

Stridor, a high-pitched sound during breathing, is a clinical sign rather than a diagnosis, often signaling partial airway obstruction in children [24].

Stridor warrants careful evaluation to determine its cause and severity. In this section, we review the evolving landscape of various causes of pediatric stridor coupled with its evaluation and management.

3.2 Epidemiology

Pediatric stridor has a varied epidemiology influenced by age, geographic location, and underlying etiologies. In infancy, the overall incidence of stridor is estimated to be around 1–2% in the general population. Laryngomalacia, a congenital anomaly characterized by supraglottic collapse during inspiration, is the most common cause of stridor in infants, accounting for approximately 60–75% of cases. Other congenital anomalies such as vocal cord paralysis, subglottic stenosis, laryngeal webs, atresia, and subglottic hemangiomas contribute to a smaller proportion of cases. In older children, infectious or inflammatory conditions like croup and epiglottitis are more prevalent causes of stridor. Croup peaks in its incidence between 6 and 36 months of age, contributing to approximately 3,50,000–4,00,000 annual casualty visits related to the condition. Annually, croup affects approximately 2–6% of infants and children, with a slightly higher prevalence observed among males, at a ratio of 1.4:1, compared to females. Additionally, foreign body aspiration accounts for over 17,000 emergency department visits yearly in the United States, predominantly affecting children under the age of 3 [25–28].

3.3 Pathophysiology and etiology

The physics of stridor involves Poiseuille's law and the Bernoulli principle. Changes in airway radius, as seen in conditions like laryngomalacia, result in turbulent airflow and negative pressure, leading to airway collapse and the characteristic stridor sound [28]. Stridor can originate at various levels of the airway, such as the supraglottis, glottis, subglottis, and trachea, each presenting unique characteristics. Inspiratory stridor suggests a supraglottic or glottic etiology, expiratory stridor is typically due to a tracheal or bronchial obstruction, and biphasic stridor generally indicates subglottic pathology (**Figure 2**) [29].

Causes can range from a transient and benign condition to a critical manifestation of impending respiratory failure (**Table 2**). Laryngomalacia, a common cause of neonatal stridor, may require supraglottoplasty for severe cases. Vocal cord paralysis, whether unilateral or bilateral, presents with varying degrees of airway obstruction and may necessitate surgical interventions. Croup, recurrent croup, subglottic stenosis, and subglottic hemangioma are among the infectious, inflammatory, or structural causes that clinicians consider in the differential diagnosis.

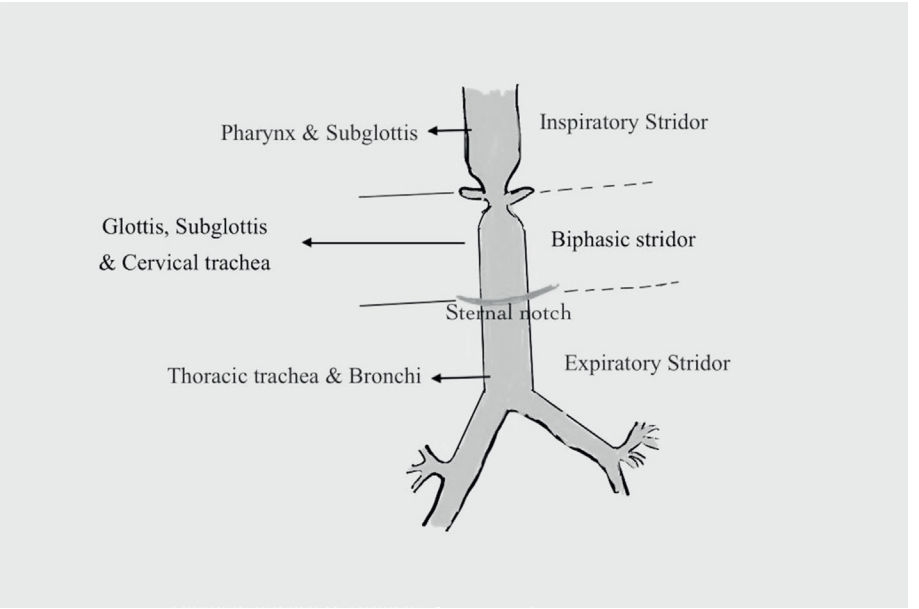

Figure 2.
Types of stridor.

3.4 Laryngomalacia

Laryngomalacia is a common congenital condition characterized by the collapse of the supraglottic structures during inspiration, leading to airway obstruction. It is the most common cause of stridor in infants and typically manifests within the first few weeks of life. It affects approximately 1 in 2000 live births. While the exact etiology remains unclear, it is believed to result from immaturity or softness of the laryngeal cartilages, leading to dynamic collapse during breathing.

Clinical features of laryngomalacia include high-pitched inspiratory stridor, which worsens with agitation, crying, feeding, or supine positioning noticed before 2 weeks of age. Typically, the noisy breathing subsides when the child is placed in the prone position. Infants may also exhibit retractions, feeding difficulties, failure to thrive, and occasionally, cyanosis or apnea episodes. Diagnosis is primarily clinical, based on history and physical examination, although flexible laryngoscopy may be performed to confirm the diagnosis and evaluate the severity of airway collapse.

Management of laryngomalacia is typically conservative, with reassurance and supportive care for mild cases. However, severe cases may require intervention to alleviate airway obstruction and improve symptoms. This can include positioning techniques, such as prone or lateral positioning during sleep, to optimize airway patency. Surgical intervention, such as supraglottoplasty, may be considered for severe and refractory cases or those associated with significant feeding difficulties, failure to thrive, and obstructive sleep apnea [27, 30–34].

3.5 Acute epiglottitis

This acute condition is caused by *Hemophilus influenzae b*. Though there is a rapid decline in the incidence due to widespread vaccination with *Hemophilus influenzae b,*

Pharynx/hypopharynx
• Vallecular cyst
• Vascular malformation
• Laryngopharyngeal reflux
Supraglottis
• Laryngomalacia
• Laryngoscope
• Acute epiglottitis
• Vascular malformation
• Stenosis
Glottis
• Vocal cord paralysis (Bilateral > unilateral)
• Intubation injury
• Laryngeal cleft
• Glottic web
• Stenosis
• Laryngeal trauma
Subglottis
• Subglottic hemangioma
• Croup
• Intubation granuloma
• Acquired stenosis
• Cyst
• Congenital cricoid malformation
• Foreign body
Tracheobronchus
• Tracheomalacia
• Foreign body
• Complete tracheal rings
• Stenosis
• Vascular compression
• Bacterial tracheitis
• Neck/chest mass

Table 2.
Causes of stridor based on anatomical subsites.

a high level of suspicion to diagnose this condition should be present as it is an acute airway emergency. After widespread *Hemophilus influenzae* vaccination, group A β-hemolytic Streptococci infections have surpassed *Hemophilus influenzae type B* (Hib) as the predominant cause of acute epiglottitis, with variations in occurrence and management between children and adults.

Clinical presentation is classic with a 2–7-year-old with sudden occurrence of high-grade fever, difficulty in swallowing, and severe sore throat. The child appears

to be toxic and stridulous with drooling of saliva, assuming a *Tripod position* (sitting up with neck in extension and leaning forward position using arms to support shoulder girdle aid in breathing). Lateral radiograph of soft tissue neck reveals *"thumb sign,"* which is severe inflammation of epiglottis with potential for imminent airway compromise. When acute epiglottitis is suspected, pharyngeal examination with tongue depressor is contraindicated as it may precipitate airway obstruction, although fibreoptic airway endoscopy done in a controlled pediatric Intensive care setting can aid in diagnosis.

Management is to secure the airway by endotracheal intubation by experienced personnel in the intensive care unit set up. Definitive treatment includes intravenous antibiotics (third-generation cephalosporins) until the swelling of the epiglottis reduces, usually within 36 hours [35–38].

3.6 Croup

Acute laryngotracheal bronchitis (croup) stands out as the primary culprit behind acute stridor in young children and toddlers, often stemming from a viral infection caused by parainfluenza viruses I (most common), II, and III accounting for 80% of cases affecting the larynx and upper trachea. The virus infects the epithelial cells of laryngotracheal mucosa, causing edema and glandular over-secretion. Pathognomonic signs include stridor accompanied by a distinctive barking cough and upper respiratory infection. Diagnosis is further confirmed by the characteristic radiographic "steeple sign," obviating the need for laryngoscopy in most cases. Westley Croup scoring system is used for indicating disease severity in which higher scores are given for the presence of cyanosis and altered mental status (**Table 3**). Treatment strategies encompass supportive measures like oxygenation, humidification with nebulized epinephrine, and intravenous corticosteroids. Intravenous corticosteroids are the mainstay of treatment as exert a systemic anti-inflammatory effect and reduction in mucosal edema. Nebulized epinephrine not only reduces mucosal edema by its alpha agonistic effect causing vasoconstriction but also aids in bronchodilatation [39, 40].

However, recurrent croup can signal an underlying anatomical airway issue, heightening the risk of persistent stridor due to a narrowed airway. Conditions such as subglottic cysts, hemangiomas, or congenital stenosis may contribute to persistent stridor characterized by a narrow airway diameter. Chronic inflammation of the upper airway from gastrointestinal sources, particularly eosinophilic esophagitis (EoE), has also been linked to recurrent croup. When recurrent croup intensifies in severity and frequency, especially with age, further investigation through airway endoscopy is recommended. This may unveil subtle structural abnormalities, like grade 1 subglottic stenosis, and identify inflammatory triggers that exacerbate symptoms [41–43].

Pinpointing these inflammatory triggers can be challenging, and while most children naturally outgrow croup symptoms as their airways mature, recurrent cases should prompt a closer look. Parents are crucial partners in this process, requiring reassurance and guidance. In cases where no serious structural abnormality is identified, parents can be encouraged to seek symptomatic relief. Some children may benefit from steroid prescriptions to control symptoms and prevent hospitalization, but careful monitoring and limitation of steroid usage frequency are essential.

Distinguishing features between croup and epiglottitis is included in **Table 4**.

S. no	Indicator of disease severity	Level of severity	Score
1.	Stridor	None	0
		Only with agitation/excitement	1
		At rest with stethoscope	2
		At rest without stethoscope	3
2.	Retraction	None	0
		Mild	1
		Moderate	2
		Severe	3
3.	Air entry	Normal	0
		Decreased	1
		Severely decreased	2
4.	Cyanosis	None	0
		With agitation	4
		At rest	5
5.	Level of consciousness	Normal	0
		Altered mental status	5

Mild respiratory distress = score <3; moderate respiratory distress = score 3–6; severe respiratory distress = score >6.

Table 3.
Westley croup scoring system.

S. no	Clinical features	Croup	Epiglottitis
1.	Age	1–2 years	2–7 years
2.	Cause	Parainfluenza viruses	*Hemophilus influenzae b*
3.	Prodrome	1–2 days Coryza	Rapid onset within hours with sore throat, dysphagia, drooling
4.	Fever	<38°C	>38°C
5.	Appearance	Lethargic	Pale and toxic, drooling and dysphagia, sitting with neck extended (*Tripod position*)
6.	Stridor	Barking cough, loud stridor	Muffled stridor
7.	Hypoxia	Unusual	Frequent
8.	Severity	<5% of hospitalized cases require intubation	All patients require intubation

Table 4.
Distinguishing features between croup and epiglottitis.

3.7 Observation and initial evaluation

The assessment of a stridulous patient begins with a thorough history, focusing on the onset, duration, and accompanying symptoms. Assessment should be done as quickly as possible. The pediatric assessment triangle encompasses appearance,

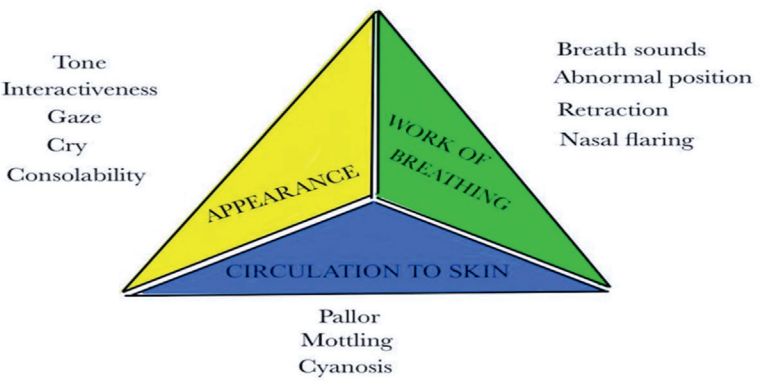

Figure 3.
Pediatric assessment triangle.

work of breathing, and circulation to skin (**Figure 3**). Acute-onset stridor is often associated with infectious, inflammatory, or foreign body causes, while chronic or gradual onset may be indicative of stable but restricted airways. In the emergency department, clinicians prioritize assessing vital signs, most importantly oxygenation, pulse rate, respiratory rate and effort. Airway and chest radiographs are performed to identify the level of obstruction, and a rapid evaluation helps determine the need for urgent intervention [44].

3.8 Physical examination

Observation of the phase of stridor in concurrence with the respiratory cycle, combined with struggling respiratory effort, aids in identifying patients at risk for impending respiratory failure. Retractions in the suprasternal and intercostal regions along with the use of accessory muscles of respiration indicate increased respiratory effort. Mental status changes suggest advancing hypoxia or hypercarbia. A comprehensive head and neck examination helps pinpoint the source of stridor, considering conditions like micrognathia, hemangiomas, or facial masses [27].

3.9 Diagnostic modalities

Diagnostic radiography, including airway radiographs and advanced imaging like CT or MRI, plays a crucial role in identifying airway lesions and evaluating anatomical abnormalities. Multidisciplinary assessments involving otolaryngologists, pulmonologists, and gastroenterologists contribute to a comprehensive understanding of the patient's condition. Awake fiberoptic nasopharyngoscopy and laryngoscopy provide real-time visualization, aiding in the diagnosis of conditions such as laryngomalacia, vocal cord paralysis, and laryngopharyngeal reflux.

3.10 Prevention

Preventive measures, such as widespread influenza vaccination, have significantly reduced the occurrence of acute epiglottitis. Despite a decline in neonatal-acquired subglottic stenosis due to improved intubation practices, surviving infants may have complex medical comorbidities affecting their management.

3.11 Treatment approaches

Fiberoptic airway endoscopy is a significant advancement in addressing obstructive airway diseases and emphasizes the need for collaborative efforts among pediatricians, anesthetists, and otorhinolaryngologists as it has to be performed in a controlled setting. Thus, integrating airway endoscopy into existing management protocols for childhood stridor is essential.

Treatment modalities are tailored according to the specific etiologies and the severity of the disease condition. Initial management often includes assessment of airway patency and oxygenation, followed by interventions to alleviate airway obstruction. Securing the airway is the most crucial step in managing various causes of pediatric stridor. Laryngomalacia management ranges from conservative management for mild cases of laryngomalacia to surgical interventions like supraglottoplasty for severe laryngomalacia. Following airway control, Acute epiglottitis is treated with IV antibiotics and croup is managed with intravenous corticosteroids and nebulized racemic epinephrine. Procedures such as adenotonsillectomy, supraglottoplasty, or airway reconstruction may be performed depending on the specific diagnosis and anatomical considerations.

Overall, the management of pediatric stridor requires a multidisciplinary approach involving pediatricians, otolaryngologists, anesthesiologists, and respiratory therapists to ensure appropriate assessment and treatment tailored to the individual child's needs [38–40, 44].

3.12 Conclusion

Pediatric stridor demands a comprehensive and multidisciplinary approach for accurate diagnosis and optimal management. Timely recognition of potential airway compromise is crucial to prevent respiratory failure and associated complications. Advances in diagnostic techniques and treatment modalities, coupled with a collaborative effort among various medical specialties, contribute to improved outcomes for children with stridor. Early intervention and a tailored treatment plan can significantly impact the quality of life for affected individuals and alleviate the potential risks associated with upper airway obstruction [45].

4. Adenotonsillitis

4.1 Applied anatomy

Palatine tonsils are paired lymphoid structures that are located in the lateral wall of the oropharynx between the anterior and posterior pillars. The anterior pillar is constituted by the palatoglossus muscle and the palatopharyngeus muscle constitutes the posterior pillar. Each tonsil has a medial and a lateral surface, with the medial surface covered by nonkeratinizing stratified squamous epithelium. This epithelium intends into the tonsillar substance forming crypts. Crypta magna is the most prominent crypt which represents the embryological origin, ventral part of the second pharyngeal pouch. The lateral surface of the tonsil is covered by a well-defined fibrous capsule with an intervening loose areolar tissue between the capsule and the bed of the tonsil. These whitish fibrous strands represent the plane of dissection during tonsillectomy [46, 47].

4.2 Blood supply of tonsil

The arterial supply of tonsil is depicted in **Figure 4**. Tonsillar branch of facial artery is the main arterial supply of tonsil. Venous drainage from the tonsils enters the paratonsillar vein which then joins the common facial vein and pharyngeal venous plexus.

4.3 Developmental anatomy

It encompasses the immunological activation timeline, structural modifications, enlargements, and involution processes. Understanding the fetal development and alterations within the first 10 years of age can assist in making informed surgical decisions (**Table 1**).

During intrauterine development, the tonsil's epithelium covering the medial surface and crypts originates from the second branchial pouch. Solid epithelial cores form in the lateral walls of these pouches, branching into primary and secondary tonsillar crypts. Mature crypt epithelium, revealed by transmission electron microscopy, is porous, allowing lymphocytes to *mediate immune responses.*

Pharyngeal tonsils which are a part of the mucosa-associated lymphoid tissue, develop monocellular populations similar to Peyer's patches in the gut. Lymphocytes invade the lamina propria around the 16th week postconception. The tonsil lacks afferent lymphatics, with dendritic cells in crypt epithelium transporting antigens.

Immune stimulation begins postnatally, leading to terminal differentiation of effector B-cells and the formation of secondary follicles with active germinal centers. This rapid germinal center proliferation dominates the 1st decade, resulting in tonsillar enlargement without invasion of surrounding tissues.

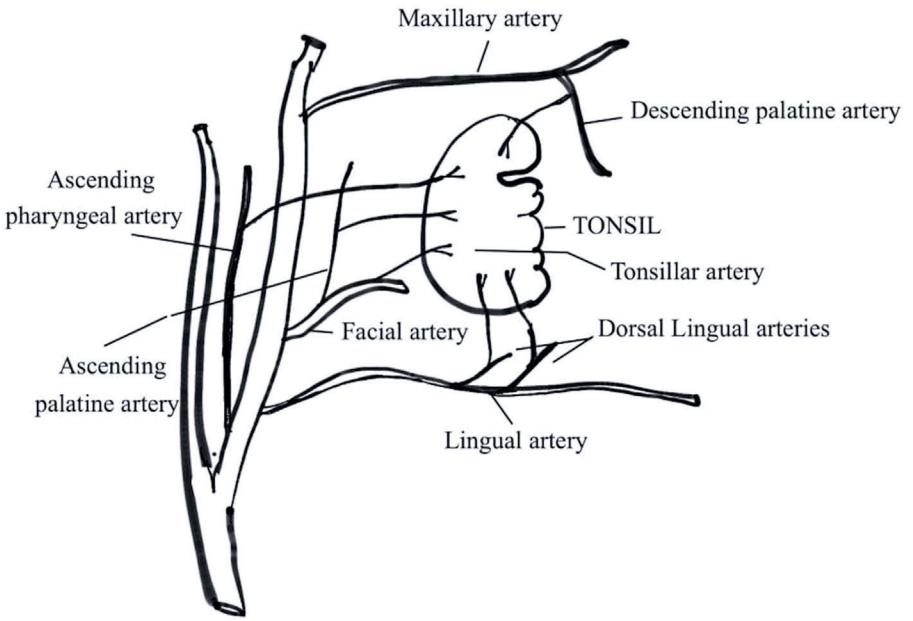

Figure 4.
Arterial supply of tonsil.

The tonsillar capsule arises from deep layers of the lamina propria, forming a continuous membrane except on the medial, cryptic surface. Unlike lymph nodes, the tonsil's parenchyma consists of densely packed fronds with a fibrovascular core, lymphoid tissue, and crypt epithelium.

In the 2nd decade, the B-cell component undergoes involution, with smaller germinal centers and increased fibrous tissue in trabeculae and capsules. The tonsil regresses, disappearing behind the anterior tonsillar pillar by the 7th decade, surrounded by fibrous connective tissue, making its outline less distinct. Fatty degeneration begins around 25 years and progresses with age [48–52].

4.4 Acute tonsillitis

4.4.1 Definition

Acute tonsillitis is a common inflammatory condition that involves acute inflammation of the tonsils. It is characterized by sore throat, difficulty in swallowing, fever, and enlarged tonsils. Timely and appropriate management, including antimicrobial therapy, is crucial to prevent complications and ensure a favorable outcome. It can be classified as:

1. Acute catarrhal or superficial tonsillitis: associated with generalized pharyngitis, often seen in viral infections.

2. Acute follicular tonsillitis: infection extends into the crypts, filling them with purulent material, resulting in yellowish spots at the crypt openings.

3. Acute parenchymatous tonsillitis: affects the tonsillar parenchyma, leading to uniform enlargement and redness.

4. Acute membranous tonsillitis: a stage beyond follicular tonsillitis, where exudation from crypts coalesce to form a membrane over the tonsillar surface.

4.4.2 Epidemiology

Acute tonsillitis commonly affects school-going children and adults, being rare in infants and those above 50 years. It primarily affects children and adolescents, with an incidence that peaks between the ages of 5 and 15 years. However, it can also occur in adults, especially those who are immunocompromised. The epidemiology of acute tonsillitis is influenced by various factors, including viral and bacterial pathogens, socioeconomic status, and environmental factors such as overcrowding. *Hemolytic streptococcus* is the most common infecting organism, contributing to approximately 15–30% of cases in children and 5–15% in adults. Other potential causes include *staphylococci, pneumococci*, or *Hemophilus influenzae*. Viral causes, including adenovirus, influenza virus, parainfluenza virus, and Epstein-Barr virus, are responsible for the majority of cases, especially in the adult population. The spread of acute tonsillitis is facilitated by close contact with infected individuals and can lead to outbreaks in settings such as schools and daycare centers. Early recognition and appropriate management, including antibiotic therapy when indicated, are essential for preventing complications and reducing the transmission of the disease [53–55].

4.4.3 Clinical features

Symptoms vary in severity and include sore throat, difficulty swallowing, fever, earache (possibly due to acute otitis media), and marked constitutional symptoms such as headache, body aches, malaise, and constipation.

Signs of acute tonsillitis include halitosis, coated tongue, hyperemia of pillars, soft palate, and uvula. Tonsils appear enlarged and congested, with yellow spots exuding out of crypts (follicular tonsillitis) or a wipeable whitish membrane (membranous tonsillitis). Enlarged and tender jugulodigastric lymph nodes may be present.

4.4.4 Treatment

Treatment involves bed rest, adequate hydration, antipyretics, and analgesics (aspirin or paracetamol) to alleviate pain and reduce fever. Antimicrobial therapy, typically with penicillin or erythromycin for penicillin-allergic individuals, is recommended and should be continued for a week to 10 days.

4.4.5 Complications

- Chronic tonsillitis: incomplete resolution leading to recurrent acute attacks.
- Peritonsillar abscess: collection of pus around the tonsils.
- Parapharyngeal abscess: abscess formation near the throat.
- Cervical abscess: suppuration of jugulodigastric nodes.
- Acute otitis media: recurrent attacks may coincide with tonsillitis.
- rheumatic fever: associated with Group A beta-hemolytic Streptococci.
- Acute glomerulonephritis: rare complication.
- Subacute bacterial endocarditis: possible in patients with valvular heart disease.

4.4.6 Chronic tonsillitis

It is characterized by persistent and chronic inflammation of the tonsils, most commonly occurring as a complication of acute tonsillitis. The condition may also emerge subclinically, affecting predominantly children and young adults.

Clinical symptoms of chronic tonsillitis encompass recurrent attacks of sore throats causing frequent school absenteeism and poor scholastic performance, chronic throat irritation with cough, halitosis, and, in severe cases, thick speech, swallowing difficulty, and nocturnal apneic spells.

4.4.7 Cardinal features of chronic adenotonsillitis

1. Crescentic residual anterior tonsillar pillar congestion
2. Bilateral, nontender, enlarged, and jugulodigastic nodes (Wood's node)

3. *Septic Squeeze test positive (Irwin Moore sign)*—performed with two tongue depressors with one depressing the anterior two-third of tongue and another one applying pressure over the anterior tonsillar pillar. A positive test is a cheesy material extruding out of the tonsillar crypt when the test is performed.

Most of these children have associated adenoid hypertrophy which can be clinically evident as adenoid facies. *Classical features of adenoid facies* include an elongated dull-looking face with a pinched upper lip, pinched nostrils, a high-arched palate, and overcrowding of upper teeth.

Treatment strategies involve conservative measures, focusing on overall health, diet, and addressing concurrent infections in the nose, paranasal sinuses, and teeth. Tonsillectomy is indicated when the tonsils significantly hinder normal functions or trigger recurrent attacks. Complications may arise, including peritonsillar abscess, parapharyngeal abscess, intratonsillar abscess, tonsilloliths, tonsillar cysts, and systemic infections.

4.5 Pediatric tonsillectomy

In recent years, pediatric tonsillectomy has changed its approach and considerations. The shift is notable, with more children undergoing the procedure for obstructive sleep apnea rather than recurrent pharyngitis. Advanced instrumentation that is available now enables less invasive surgical techniques. Systematic reviews by organizations like the Cochrane Collaboration have played a pivotal role in establishing best clinical practices for preoperative assessment and postoperative care. With approximately 100 million tonsillectomies performed worldwide over the past century, this procedure remains a vital intervention, particularly for obstructive sleep apnea and sleep-disordered breathing in children.

4.5.1 Indications for pediatric tonsillectomy

As per the Paradise criteria, tonsillectomy may be indicated for patients experiencing recurrent episodes of sore throat—history of at least seven documented episodes of sore throat in the preceding year, at least five documented episodes in each of the past 2 years, or at least three documented episodes in each of the past 3 years. Additionally, the presence of certain clinical features during these episodes, such as a temperature exceeding 100.9°F (38.3°C), cervical adenopathy, exudate over tonsil, or a culture that is positive for group A β-hemolytic Streptococcus, qualifies as counting toward an episode [56, 57].

4.5.2 Relative indications

Adenotonsillectomy in children proves effective for various other conditions such as recurrent sore throat attacks due to chronic tonsillitis, peritonsillar cellulitis or abscess, PANDAS (Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections), Periodic fever, aphthous stomatitis, pharyngitis and cervical adenitis syndrome (PFAPA), febrile seizures associated with high-grade fever (during an attack of acute tonsillitis), halitosis due to chronic adenotonsillitis, dental malocclusion due to adenoid facies, cryptic tonsils, prevention of rheumatic fever recurrences, and control of pharyngeal streptococcal carriage. Despite its efficacy, tonsillectomy remains a challenging entity for children and their caregivers, involving risks such as immediate or delayed hemorrhage and a low but existing mortality rate.

Complications and discomfort associated with pediatric tonsillectomy primarily stem from the injury due to surgery and the subsequent inflammatory healing process. Research into cutaneous wound healing mechanisms offers insights, indicating that the cold steel technique produces minimal collateral tissue damage and promotes rapid healing.

Optimal hydration and addressing dehydration have been linked to a reduction in postoperative tonsillectomy pain. According to reports, comforting the child by placing them on the lap of their parents emerges as a highly effective nonpharmacological approach for alleviating postoperative pain at home for daycare adenotonsillectomy [58, 59].

Thus, clinical decision-making should thoroughly analyze the potential benefits against the discomforts and risks associated with tonsillectomy.

In conclusion, pediatric tonsillectomy, while a well-established and effective intervention, necessitates a careful balance between potential benefits and associated challenges. Ongoing research and advancements in surgical techniques and postoperative care present opportunities for further improving outcomes and reducing the traumatic impact on children and their families.

5. Conclusion

In conclusion, this chapter underscores the multifaceted challenges these conditions present in both physical and psychosocial realms, as well as the critical importance of tailored treatment approaches that address age-specific needs. Throughout childhood, these disorders can significantly impact a child's development, from impairing sensory perception to hindering speech and language development and even affecting social interactions and self-esteem.

In managing these complex conditions, interdisciplinary collaborations between pediatricians, otolaryngologists, and various healthcare professionals are paramount. Pediatricians provide comprehensive care, understanding the child's medical history, developmental milestones, and family dynamics. Otolaryngologists offer specialized expertise in diagnosing and treating ENT disorders, ensuring that treatment plans are tailored to the child's unique physiological and developmental needs.

By working together in an interdisciplinary framework, healthcare professionals can provide holistic care that addresses not only the physical symptoms but also the psychosocial impact of ENT disorders on children and their families. Thus, the pursuit of excellence in the care of children with ENT disorders requires a commitment to ongoing research, collaboration, and a patient-centered approach, ensuring the best possible outcomes for the youngest members of our community. Early recognition and appropriate management of common ENT diseases are essential to prevent complications and improve patient outcomes. Regular ENT check-ups and prompt medical attention for symptoms can help in the early detection and effective treatment of these conditions.

Acknowledgements

The author acknowledges the assistance of AI tool (ChatGPT, an AI language model based on the GPT-4 architecture developed by OpenAI) for language polishing of the manuscript.

DOI: http://dx.doi.org/10.5772/intechopen.1006071

Conflict of interest

The authors declare no conflict of interest.

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