

**TITLE: The Tonsils and Adenoids in Pediatric Patients**  
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## **Introduction**

Pediatric patients are frequently seen in otolaryngology practice for complaints related to the tonsils and adenoids. In 1994 an estimated 140,000 U.S. children under the age of 15 had adenoidectomies and an estimated 286,000 had adenotonsillectomy. Although this is a decrease in the number of procedures performed per year from a peak of 1 million per year in the 1970's, these are the most common major surgical procedures in children with a cost of an estimated half a billion dollars per year.

## **History**

The first report of tonsillectomy was made by Celsus in 30 AD. He described scraping the tonsils and tearing them out or picking them up with a hook and excising them with a scalpel. Paul of Aegina published his description of tonsillectomy in 625 AD. In 1867 Wilhelm Meyer of Denmark reported a case of a 20 year old woman who complained of decreased hearing and nasal obstruction. He found her nasopharynx obstructed with soft tissue he called "adenoid vegetations". For excision he constructed a ring knife which he passed through the nose and removed the adenoids. Samuel J. Crowe of Johns Hopkins published a paper in 1917 in which he described his technique for tonsillectomy in 1000 patients. He used a mouth gag still called the Crowe-Davis mouth gag today.

## **Embryology**

The formation of the adenoids begins in the 3<sup>rd</sup> month of fetal development. This starts with glandular primordia in the posterior nasopharynx becoming associated with infiltrating lymphocytes. In the 5<sup>th</sup> month sagittal folds are formed which are the beginnings of pharyngeal crypts. The surface is covered with pseudostratified ciliated epithelium. By the 7<sup>th</sup> month of development the adenoids are fully formed.

The palatine tonsils also begin development in the 3<sup>rd</sup> month. They are derived from the ventral portions of the second pharyngeal pouches. 8-10 buds of pharyngeal

squamous epithelium grow into the pharyngeal walls. The epithelium at the center of these buds dies and the initial crypts form. Lymphocytes infiltrate the crypts and then in the last trimester secondary branching occurs in the crypts and the lymphoid tissue is organized into follicles. The tubal tonsils are lymphoid collections behind the eustachian tube opening.

The lingual tonsils begin development in the 5<sup>th</sup> month. The posterior tongue forms mucous glands, which become infiltrated with lymphoid cells. This is followed by the formation of lymphoid follicles and then, at birth, shallow crypts form, which are unbranched.

## **Anatomy**

The lymphoid tissue of the nasopharynx and oropharynx is composed of the adenoids, the tubal tonsils, the lateral bands, the palatine tonsils, and the lingual tonsils. There are also lymphoid collections in the posterior pharyngeal wall and in the laryngeal ventricles. These structures form a ring of tissue named Waldeyer's ring after the German anatomist who described them.

The adenoids or pharyngeal tonsil is a single mass of pyramidal tissue with its base on the posterior nasopharyngeal wall and its apex pointed toward the nasal septum. The surface is invaginated in a series of folds with some crypts but without the complex crypts found in the palatine tonsils. The epithelium is pseudostratified ciliated epithelium and is infiltrated by the lymphoid follicles. Blood supply is from the ascending palatine branch of the facial artery, ascending pharyngeal artery, pharyngeal branch of the internal maxillary artery, the artery of the pterygoid canal, and the ascending cervical branch of the thyrocervical trunk. Venous drainage is through the pharyngeal plexus and the pterygoid plexus flowing ultimately into the facial and internal jugular veins. Innervation is derived from the glossopharyngeal and vagus nerves. Efferent lymphatics drain to the retropharyngeal nodes and the upper deep cervical nodes.

The paired palatine tonsils sit in the tonsillar sinus limited by the palatoglossal arch anteriorly and the palatopharyngeal arch posteriorly. The superior pharyngeal constrictor muscle lies laterally. The palatine tonsils are surrounded by a fibrous capsule. Fully developed tonsils have between 10 and 30 crypts. The internal carotid artery lies approximately 2.0-2.5 cm posterolaterally to the tonsil. Blood supply is from the tonsillar and ascending palatine branches of the facial artery, the ascending pharyngeal artery, the dorsal lingual branch of the lingual artery and the palatine branch of the maxillary artery. The tonsillar veins pierce the superior pharyngeal constrictor muscle to drain into the external palatine, pharyngeal and facial veins. Innervation is from the sphenopalatine ganglion via the lesser palatine and glossopharyngeal nerves. Efferent lymphatics drain into the upper deep cervical lymph nodes.

The lingual tonsils are located on the pharyngeal aspect of the tongue. They are lined by stratified squamous epithelium. Blood supply is from the lingual, ascending pharyngeal, and tonsillar branch of the facial arteries. Venous drainage is via the lingual

system. Innervation is by the glossopharyngeal nerve. Efferent lymphatics drain to the deep cervical lymph nodes.

## Function and Immunology

The tonsils and adenoids are part of the secondary immune system. They sit in the respiratory and alimentary tracts in position to be exposed to inspired or ingested antigens from air or food. Without afferent lymphatics the lymphoid nodules in these structures are exposed to antigen only in the crypts of the palatine tonsils and the folds of the adenoids where it is transported through the epithelial layer. The immunologic structure of the tonsils and adenoids is divided into four compartments: the reticular crypt epithelium, the extra follicular area, the mantle zone of the lymphoid follicle, and the germinal center of the lymphoid follicle. Membrane cells and antigen presenting cells are involved in transporting the antigen through the epithelial layer and presenting them to T-helper cells. When sufficient antigen is present this stimulates the B cells in the germinal zone of the lymphoid follicle to differentiate and produce antibodies. The tonsils and adenoids are involved in the production of mostly secretory IgA, which is transported to the surface providing local immune protection.

The effect of adenotonsillectomy on immune function is not well understood. There is some evidence that in children previously immunized orally for polio, titers dropped after adenotonsillectomy. Also, children who had previously undergone adenotonsillectomy had delayed and lowered immune response to polio vaccination based on IgA antibodies to polio virus.

## Microbiology

Many pathogens can cause infection of the tonsils and adenoids. These include both organisms of the normal oropharyngeal flora, which become pathogenic, and external pathogens. Group A beta-hemolytic streptococcus is the most frequently recognized pathogen and is associated with a risk of rheumatic fever and glomerulonephritis. However, it is now recognized that a large number of pathogens can cause inflammation in the tonsils and adenoids, and that many infections are polymicrobial. The following is a list of organisms commonly cultured from the tonsils and adenoids as listed in Bailey's *Head and Neck Surgery-Otolaryngology*:

- Bacteria
  - Aerobic
    - Group A beta-hemolytic streptococci
    - Groups B, C, G streptococci
    - Haemophilus influenzae (type B and non-typeable)
    - Streptococcus pneumoniae
    - Moraxella catarrhalis
    - Staphylococcus aureus
    - Haemophilus parainfluenzae
    - Neisseria species
    - Mycobacteria species

- Anaerobic
  - Bacteroides species
  - Peptococcus species
  - Peptostreptococcus species
  - Actinomyces species
- Viruses
  - Epstein-Barr
  - Adenovirus
  - Influenza A and B
  - Herpes simplex
  - Respiratory syncytial
  - Parainfluenza

Several of these pathogens, especially *Staphylococcus aureus*, *Moraxella catarrhalis* and *Hemophilus influenzae* can produce beta-lactamase. In poly-microbial infections this can prevent eradication of Group A beta-hemolytic streptococci with penicillins.

### **Adenotonsillar Disease**

The major division of adenotonsillar disease is between infection/inflammation, obstruction, and neoplasm. Acute adenoiditis symptoms include purulent rhinorrhea, nasal obstruction, fever, and sometimes otitis media. This can be difficult to differentiate from an acute upper respiratory infection but tends to have a longer and more severe course. Recurrent acute adenoiditis is 4 or more episodes of acute adenoiditis in a 6-month period with intervening periods of wellness. This can have a similar presentation as recurrent acute rhinosinusitis. Chronic adenoiditis symptoms include persistent rhinorrhea, postnasal drip, malodorous breath, and associated otitis media or extra esophageal reflux lasting at least 3 months. Acute tonsillitis symptoms are fever, sore throat, tender cervical lymphadenopathy, dysphagia, and erythematous tonsils with exudates. Recurrent acute tonsillitis is the above symptoms occurring in 4-7 separate episodes occurring in one year, 5 episodes a year for 2 years, or 3 episodes a year for 3 years. Chronic tonsillitis is associated with chronic sore throat, malodorous breath, presence of tonsilliths, peritonsillar erythema, with persistent tender cervical lymphadenopathy lasting at least 3 months.

Obstructive adenoid hyperplasia includes symptoms of chronic nasal obstruction, rhinorrhea, snoring, mouth breathing, and a hyponasal voice. Obstructive tonsillar hyperplasia is heralded by snoring, obstructive breathing asleep and awake, dysphagia, nocturnal enuresis, poor school performance, decreased attention span, and voice changes. Obstructive sleep apnea in children is clinically marked by loud snoring, apneic episodes while sleeping, daytime somnolence, behavioral problems, and enuresis. Some authors recommend polysomnography to evaluate children clinically suspected of having OSA not only to determine the presence of the disorder but also to determine which children would be at risk of postoperative complications. However, the parameters used for diagnosing OSA by polysomnography in children are a matter of debate. A

respiratory disturbance index of 1 to 15 has been described as an appropriate cut off level. Also, the number of centers able to perform polysomnography in children is limited, which can cause delays in treatment for affected children.

Peritonsillar abscess is classically described as an extension of an acute episode of tonsillitis with abscess formation. More recently one author has attributed this infection to abscess formation in Weber's salivary glands at the superior tonsillar pole. Classic symptoms are fever, sore throat, dysphagia, drooling, and trismus. On exam there is a unilateral bulge of the soft palate with the uvula deviated to the opposite side. There are an estimated 13,500 annual cases. Treatment options include needle aspiration, incision and drainage, or abscess tonsillectomy (Quinsy tonsillectomy). Needle aspiration or incision and drainage may be possible in an older cooperative child but may not be optimal for young children. In one study by Blotter et al nearly 70% of children under 6 responded to a 24 course of medical therapy. The other children underwent an abscess tonsillectomy.

Congenital tonsillar masses include teratoma, hemangioma, lymphangioma and cystic hygroma. The most common malignant neoplasm is lymphoma, usually non-Hodgkin's lymphoma. Unilateral tonsillar enlargement which is rapid and is associated with cervical lymphadenopathy and systemic symptoms should raise suspicion for lymphoma. Adenoid hyperplasia in a teenager should also raise suspicion for lymphoma.

## **Medical Management**

For this discussion, medical management of tonsil and adenoid disease will be discussed only briefly. The medical management of adenoid or tonsillar hyperplasia includes a course of therapy with an antibiotic effective against beta-lactamase producing organisms such as clindamycin or Augmentin. The benefits of such treatment are uncertain. Some patients with obstructive adenoid hyperplasia may respond to a prolonged course of nasal steroids. The first line treatment of acute streptococcal adenotonsillitis is still penicillin. Patients who fail to respond should undergo therapy with an antibiotic effective against beta-lactamase producing organisms and anaerobes such as clindamycin, Augmentin, or penicillin plus rifampin.

## **Surgical Management**

### **Adenoidectomy**

The current clinical indicators for adenoidectomy as recommended by the AAO-HNS in 2000 are:

- a) Four or more episodes of recurrent purulent rhinorrhea in prior 12 months in a child <12. One episode documented by intranasal examination or diagnostic imaging.
- b) Persisting symptoms of adenoiditis after 2 courses of antibiotic therapy. One course of antibiotics should be with a beta-lactamase stable antibiotic for at least 2 weeks.

- c) Sleep disturbance with nasal airway obstruction persisting for at least 3 months.
- d) Hyponasal or nasal speech
- e) Otitis media with effusion >3 months or second set of tubes
- f) Dental malocclusion or orofacial growth disturbance documented by orthodontist.
- g) Cardiopulmonary complications including cor pulmonale, pulmonary hypertension, right ventricular hypertrophy associated with upper airway obstruction.
- h) Otitis media with effusion over age 4.

## **Tonsillectomy**

The current clinical indicators for tonsillectomy as recommended by the AAO-HNS in 2000 are:

- a) Patient with 3 or more infections per year despite adequate medical therapy.
- b) Hypertrophy causing dental malocclusion or adversely affecting orofacial growth documented by orthodontist.
- c) Hypertrophy causing upper airway obstruction, severe dysphagia, sleep disorders, or cardiopulmonary complications.
- d) Peritonsillar abscess unresponsive to medical management and drainage documented by surgeon, unless surgery performed during acute stage.
- e) Persistent foul taste or breath due to chronic tonsillitis not responsive to medical therapy.
- f) Chronic or recurrent tonsillitis associated with the streptococcal carrier state and not responding to beta-lactamase resistant antibiotics.
- g) Unilateral tonsil hypertrophy presumed neoplastic.

## **Techniques**

Adenoidectomy is performed with an adenotome, shaver blade, or with curettes. Hemostasis is usually achieved with gauze packing, electrocautery, or both. Methods of tonsillectomy include cold dissection and snare, tonsillotome, monopolar electrocautery, bipolar electrocautery, and CO<sub>2</sub> or KTP laser. Hemostasis is obtained by gauze packing, electrocautery or both. Because of cost laser tonsillectomy is usually reserved for patients with bleeding disorders. Regardless of the method used proper operative technique is most important to avoid complications.

## **Indications for inpatient monitoring**

Tonsillectomy and adenoidectomy are frequently performed on an outpatient basis. Recommendations for inpatient observation are age less than 3 years, history of obstructive sleep apnea, children with significant associated medical problems, neurological delay, or craniofacial abnormalities, children who live a long distance from the hospital, and those children with a questionable caregiver at home. Gerber, et al. showed a significant increase in respiratory compromise in children less than 3, those with neuromuscular disorders or chromosomal abnormalities, those with difficulty breathing during sleep, restless sleep, loud snoring with apnea, and history of URI within

4 weeks of surgery. Gabalski, et al. recommended not performing outpatient surgery for morbid obesity, sleep apnea, pickwickian syndrome, airway compromise, congenital heart disease, previous anesthesia complication, mental retardation, known coagulopathy, traveling time more than 30 minutes from the hospital, and surgeon's suspicion of poor parental ability to care for the child in the immediate postoperative period.

## Complications

The incidence of mortality from adenotonsillar surgery ranges from 1 in 16,000 to 1 in 35,000 cases. Anesthetic complications and hemorrhage cause the majority of deaths. The prevalence of hemorrhage ranges from 0.1% to 8.1%. It is divided into primary bleeding, in the first 24 hours, and secondary bleeding, around 7-10 days post operatively. Other risks include vomiting, dehydration, airway obstruction due to edema, pulmonary edema, fever, velopharyngeal insufficiency, dental injury, burns, and nasopharyngeal stenosis. Atlantoaxial subluxation can occur in patients with Down syndrome and atlantoaxial joint laxity or after adenoidectomy because of Grisel's syndrome. This is vertebral body decalcification and laxity of the anterior transverse ligament between the atlas and the axis from inflammation or infection in the nasopharynx. Spontaneous subluxation occurs about 1 week post operatively with pain and torticollis.

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