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Evaluation of Adenotonsillar Disease

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Abstract: "Adenoid" the common term for hyperplasia of the pharyngeal tonsil, is a very widespread condition in children 3–6 years of age. The proliferation of lymphatic tissue in this region is so common in children that it can hardly be considered an abnormal condition, and nearly all children have some degree of adenoid hypertrophy due to the immunologic activity of that tissue. As a result, enlarged adenoids should be considered abnormal and treated accordingly only if they are causing symptoms. Not infrequently, the presence and severity of adenoidal symptoms depend on the relationship between the size of the nasopharynx and that of the adenoids. Some authors see that, when typical signs and symptoms of obstructive adenoid hyperplasia are noted, a lateral neck radiograph is unnecessary to confirm a clinically apparent diagnosis. Lateral neck films are limited by the two-dimensional representation of a three-dimensional space and by unreliability in demonstrating small choanal adenoids causing obstruction. When the symptoms and signs do not agree, a computed tomography (CT) is useful. Although studies have demonstrated that symptoms of nasal obstruction are strongly correlated with adenoid size, other studies indicate the importance of a more reliable diagnosis by endoscopy for the dynamic and three-dimensional evaluation of the nasopharynx and of the other intranasal structures.

Keywords: *Evaluation, Adenotonsillar Disease*

Introduction

"Adenoid" the common term for hyperplasia of the pharyngeal tonsil, is a very widespread condition in children 3–6 years of age. The proliferation of lymphatic tissue in this region is so common in children that it can hardly be considered an abnormal condition, and nearly all children have some degree of adenoid hypertrophy due to the immunologic activity of that tissue. As a result, enlarged adenoids should be considered abnormal and treated accordingly only if they are causing symptoms. Not infrequently, the presence and severity of adenoidal symptoms depend on the relationship between the size of the nasopharynx and that of the adenoids **(1).**

The pathogenesis of infectious/inflammatory disease in the tonsils and adenoids has its basis in their anatomic location and inherent function as organs of immunity, processing infectious material and other antigens and then becoming, paradoxically, a focus of infection/inflammation. Viral infection with secondary bacterial

invasion may be one mechanism of the initiation of chronic disease, but the effects of the environment as overcrowdings and lack of fresh air, host factors as low immunity and malnourished children, the widespread use of antibiotics, ecologic considerations, and diet may all play a role **(2).**

Lymphoid tissue of the Waldeyer ring is very small in infants. It increases in size by the time the child is 4 years of age in association with immunologic activity, since tonsils and adenoids are the first lymphoid organs in the body to encounter ingested and inhaled pathogens. Tonsillar and adenoid tissues have many specialized immunologic compartments responsible for humoral and cellular immune response. Therefore, hypertrophy of the lymphoid tissue as a whole occurs in response to colonization with normal flora as well as with pathogenic microorganisms. Second-hand smoke exposure in the home environment has also been linked to adenotonsillar hypertrophy **(3).**

Acute adenoiditis is difficult to differentiate from a generalized viral-induced URI or a true bacterial rhinosinusitis. Rhinorrhea (sometimes purulent), nasal obstruction, fever, and often otitis media may be seen. When the acute infection is accompanied by loud snoring, which then dissipates after the episode, infection in the adenoids may be more likely. A child also appears more sick than when confronted with typical viral URI **(2).**

Recurrent acute adenoiditis is defined as the presence of four or greater discrete episodes of acute adenoiditis during a 6-month period. Again differentiating between recurrent acute sinusitis and recurrent acute adenoiditis is difficult to make on clinical grounds alone **,** Radiographs to evaluate the sinuses may be useful **(4).**

Chronic Adenoiditis with persistent nasal discharge, malodorous breath, postnasal drip, and chronic congestion may signify a chronic adenoid infection. Most of these symptoms are often associated with chronic sinusitis. The association of otitis media may be more indicative of chronic adenoiditis. **(5).**

Acute and chronic infection in the adenoids and that in the sinuses both present with symptoms of rhinorrhea, cough, and postnasal drip. Therefore, a complete otorhinolaryngologic examination, including nasopharyngoscopy and sinus radiograph, is necessary. The coexistence of these two problems (with the chronic adenoid infection leading to a secondary sinusitis or the reverse) is present **(6).**

***Symptoms of adenoidal hypertrophy*:**

1. **Bilateral nasal obstruction**

Enlarged adenoids cause nasal airway obstruction, with clinical symptoms of nasal congestion, snoring, and breathing through the mouth, by physically blocking the back of the nose. Obstruction as a cause of hypertrophied adenoids is based on their size alone. However, when enlarged, the adenoids may have a chronic infection **(2).**

Adenoid hypertrophy could result in nasal airway obstruction and an opened mouth during days and nights awake and sleep. The study of **Behlfelt** showed 62.58% of children with adenoid hypertrophy had mouth breathing during days and 84.78% during nights **(7).**

2.Nasal discharge

Children will present with symptoms of chronic or recurrent sinusitis. These clinical symptoms may include postnasal discharge or purulent anterior rhinorrhea**,** cough, fever, facial pain, and nasal congestion (**8).**

For patients with chronic sinusitis, the adenoid appears to act as a reservoir of infection**.** Improvement is observed following adenoidectomy -independent of the weight of the adenoids- in children with symptoms of chronic sinusitis (**9)**

Additionally, **Brodsky** showed that the same pathogenic bacteria in the adenoids were cultured from the middle meatus near the anterior sinus drainage site

McClay also showed that resistant bacteria were found in the adenoid bed .

Symptoms of nasal airway obstruction may overlap with chronic sinusitis symptoms, and the physical obstruction may add to sinusitis itself by blocking normal nasal flow posteriorly, resulting in a stasis of secretions and an obstruction in the sinus outflow tract (**9)**

2. Snoring

Snoring is the shared feature of three clinical syndromes of sleep-related upper airway obstruction in children. Approximately 10% of children with adenoidal hypertrophy snore on all or most nights, and most of these children have simple snoring, also called primary snoring (PS)**.** Simple or primary snoring is snoring that occurs without associated apnea, gas exchange abnormalities, or excessive arousals **(10).**

This type of snoring does not appear to progress to OSAS in young children and may, in fact, resolve over time **(11).**

Children with nasal obstruction due to adenoids have an upper airway resistance syndrome (UARS). They snore and have partial upper airway obstruction that leads to repetitive episodes of increased respiratory effort that ends in arousals**.** Sleep pattern is disrupted, and daytime symptoms similar to those seen with OSAS may exist **(12).**

UARS in children is more common than OSAS **(13).**

Childhood OSAS is characterized by sleep-related upper airway obstruction that usually is associated with a reduction in oxyhemoglobin saturation, hypercapnia, or both. Sleep-related upper airway obstruction in children can manifest as complete obstructive apneas or partial obstruction with hypoventilation **(14).**

Due to adenoidal hypertrophy and OSAS, children will have nighttime sweating, restless sleep, and sleeping in unusual positions and nocturnal enuresis **(13).**

Resolution of enuresis has been reported following adenotonsillectomy in children with symptoms of nocturnal upper airway obstruction **(14).**

Daytime symptoms associated with OSAS include mouth breathing, nasal obstruction, and hyponasal speech. Although the hallmark of adult OSAS is excessive daytime sleepiness, this symptom is less common in children with OSAS **(16).**

3. Middle ear effusion

Chronic adenoids hypertrophy is one of the causes of secretory otitis media, and adenoidectomy may benefit the middle ear by removing a source of infection from the nasopharynx and has been shown to be helpful in children over 4 years of age with chronic otitis media with effusion **(17).**

To lesser extent, hypertrophied adenoids are considered a cause for recurrent acute otitis media, and there is much less evidence of the efficacy of adenoidectomy in preventing recurrent episodes of acute otitis media. but among children previously treated with tympanostomy tubes it reduced further attacks **(18).**

Persistent ear infections or ear effusion in children are usually related to persistent immature Eustachian tube function, dysfunction related to chronic adenoid infection, or dysfunction of the eustachian tube related to congestion from allergic rhinitis. Several studies indicate that Eustachian tube function is improved and fluid collection is prevented following adenoidectomy, independent of the size of the adenoids **(17).**

Pathophysiology of the adenoid's role in causing ear infections is by spreading up of infections to the ears from adenoid and cause middle ear infections, which can affect hearing . Glue ear – the swollen adenoids block the Eustachian tubes and prevent the normal mucous, which is made each day in the middle ear, from draining away. **(17).**

Pillsbury demonstrated more pathogenic bacteria in the adenoid beds of patients with recurrent otitis media than in the adenoid beds of patients cultured for persistent serous otitis media or hypertrophy **(19**)

However, **Brodsky** found the same amount of pathogenic bacteria in the adenoids of patients with otitis media and rhinosinusitis, regardless of size, as in the adenoids of patients with only adenoid hyperplasia causing nasal airway obstruction **(2).**

So Whether the bacteria that are harbored in the adenoids cause irritation of the Eustachian tube lining, resulting in dysfunction, or the harbored bacteria cause a chronic low-grade infection in the middle ear space, resulting in persistent fluid or recurrent infections, remains unclear (**20).**

Distinguishing episodes of otitis media as acute suppurative otitis media or otitis media with effusion is important for clinical decision making. Acute otitis media is defined by the presence of symptoms of acute

illness and signs (full or bulging) of a tympanic membrane under positive pressure. Otitis media with effusion is defined by the absence of symptoms and signs of acute infection other than reduced hearing and the presence of signs retracted or neutral position of a tympanic membrane under negative pressure or no pressure and fluid in the middle ear space. That is as stated by Agency for Healthcare Research and Quality (formerly called the Agency for Health Care Policy and Research) **(21).**

The most important symptoms that increase the likelihood of acute otitis media are ear-related symptoms, such as earache, rubbing of the ear or a report by older children of the ear feeling blocked. Fever, earache, crying and irritability, alone or in combination, are present in 90 percent of the children with acute otitis media **(22).**

4. **Speech abnormalities**

As a result of their location, adenoids primarily contribute to vocal quality. The size and position of an adenoid have a direct influence on the degree of nasal resonance. Chronic mouth breathing can cause alterations to the motor speech apparatus, bad head and shoulder posture, alterations to the vocal cords, a reduction in the nasal component of the voice and atypical deglutition. Hyponasality is often due to enlarged adenoids beside colds, allergies, respiratory infections, the space between the soft palate and wall of the throat is filled up and doesn't allow air to reach the nasal cavity**.** The triad of hyponasality, snoring, and mouth breathing usually indicates enlarged, obstructing adenoids **(22).**

5. **Adenoid face**

Growth of craniofacial structures and dental occlusion is affected by the presence of mouth breathing. Upper respiratory obstruction and adenoid hypertrophy are the most common etiologies that cause it. Long period airway obstruction during childhood causes some structural changes in faces and teeth presented called adenoid face. Upper respiratory obstruction can result in increasing airway resistance followed by mandible displacement,over crowding teeth and divergence of tongue in to bottom of the mouth to increase the airways' space. The great distance between anterior teeth results in overgrowth of posterior teeth that pushes down the mandible **(23).**

Obstruction of airway also changes the anatomy of the mouth. As displacement of mandible to posteroinferior, the height of the inferior part of face will increase. The new position of tongue will make it without contact with palate. As the natural growth and expansion of maxilla is related to the forces of the tongue, deepening and narrowing of the palate will occur due to this tongue position. Also flattening of nasolabial folds and narrow naris, low jaw will become long, prominent anterior part of maxilla and lengthen of the mandible cause high overjet (the upper front teeth stick out too far, or the lower front teeth are too far back), low overbite (it is a condition where your upper front teeth overlap with and cover your lower front teeth), posterior crossbite(an inadequate transversal relationship of maxillary and mandibular teeth) , open mouth, rhinolalia. These patients often present swallowing and speech problems and oral breathing. Chronic nasal obstruction not related to the adenoids, (nasal septal deformity, chronic rhinitis, external nasal deformity) can lead to similar dental patterns and an elongated lower face. The classic adenoid facies characterized by an open mouth appearance, flattened midface, and dark circles under the eyes is also seen in children with allergic rhinitis or other causes of chronic nasal obstruction. Abnormalities in the maxillary-mandibular relationship are sometimes identified (**23).**

6. **Oral infections**

The most recalcitrant cause of halitosis (also known as bad breath) in people with excellent oral hygiene) is due to bacterial overgrowth in the back part of the tongue (lingual tonsils). Adenoids is another location often neglected as a cause of halitosis, primary duo to post nasal drip and secondary to recurrent pharyngitis and tonsillitis duo to its hypertrophy **(24).**

Adenoiditis may cause halitosis if the nasal passages become obstructed so that it is necessary to breath through the mouth, or because a purulent exudate is produced. An oral breathing pattern (Breathing through the mouth) causes bad breath because the amount of saliva in the mouth is reduced by evaporation

Chronic suppurative sinusitis may produce foul-smelling purulent exudates resembling hypertrophied adenoids. Making diagnosis by this symptom unreliable. Gingival dryness and the dehydration due to open mouth can result in microbial plaque formation dental caries and gingivitis. Gingival infection was found to be higher in the mouth breathers than in the normal breathers in the subjects with incompetent lip seal. Increased lip separation and decreased upper lip coverage were all associated with higher levels of plaque and gingival infection **(25).**

7. **Chest infections**

Children with adenoid hypertrophy may be chronically tired, having a chronic cough, particularly at night as the airways dry out from mouth breathing and pus can drip into the oral cavity causing pharyngitis and into the airways causing choking. Splashes of excessive drip from infected adenoids may land directly on the vocal cords. Although the larynx and vocal cords do not ordinarily become infected from adenoiditis, their mucosa does become irritated. The vocal cords are extremely sensitive to touch, and any fluid drops falling on them cause an irresistible urge to cough. Adenoiditis therefore is one of the causes of cough and -to lesser extent-of descending respiratory system infections, causing bronchitis and chest infection **(26).**

8. **Cardiac disorders**

Adenoid hypertrophy can also cause cardiorespiratory alterations. Severe untreated OSAS duo to hypertrophied adenoids can lead to pulmonary hypertension and cor pulmonale which is reversible by treatment .Pulmonary hypertension results from the recurrent severe nocturnal hypoxemia, hypercapnia, and acidosis that occur during hypoventilation or apnea **(27).**

These observations were significant, because left ventricular hypertrophy is a known risk factor for future cardiovascular disease**. Amin et al**, however, found that OSAS in children led to structural changes and hypertrophy of the right and left ventricles. **(28).**Systemic hypertension, a complication of adult OSAS, has been also reported in children with OSAS **(29).**

9. **Skeletal deformities**

Harrison's sulcus, which is Also known as, Harrison's groove, is a deformity of the ribs, a depression on lower edge of the thorax at the insertion of diaphragm, caused by tug of the diaphragm. It is seen in rickets, severe dyspnoea, particularly in stenosis of larynx, and any disease of infants that tends to obstruct inspiration. This depression gives the chest the shape of a pear. Pectus excavatum (also known as pigeon chest) which is an abnormal overgrowth of the lower costal cartilages between the ribs and sternum which pushes the sternum inward, also may be present **(*30*).**

10. **Growth retardation**

Children with adenoidal hypertrophy causing severe OSAS may have failure to thrive**.** Children will grow poorly. Improved growth has been reported after treatment of childhood OSAS with adenotonsillectomy **.Marcus et al** reported that the impaired growth in children with even mild OSAS appeared to be related to increased work of breathing during sleep, and exhaustion due to energy loss, and weight loss due to difficult feeding and suckling **(11).**

Nocturnal growth hormone secretion also appears to be decreased in children with sleep-related upper airway obstruction, adding more to retarded growth in children with adenoidal hypertrophy **(31).**

11. **GIT symptoms**

Beside indigestion and morning vomiting due to swallowed mucus of post nasal drip, symptoms consistent with gastroesophageal reflux may also be present in cases of chronic or severe OSA**.** Again GERD may act as a cause of adenoid hypertrophy and adenoiditis due to recurrent pharyngeal irritation initiating the viscous circle. **(32).**

12. **Psychological abnormalities**

Sleep alterations in children can present as restless sleep, fragmented sleep, diaphoresis, hyperextension of the neck, witnessed apneas, and parasomnias. Parasomnias, will present by bruxism, nocturnal enuresis, sleep-walking, night-terrors, night mares and restless legs **(32).**

Furthermore, the body image of children affects their emotional and psychological status, they became shy and friendless **(33).**

13. Behavioral and learning abnormalities

An increased incidence of adenoidal hypertrophy with daytime sleepiness, snoring, and other symptoms of sleep-disordered breathing was found in children who were being evaluated for inattention and hyperactivity. Behavioral problems, such as hyperactivity and aggressiveness, have been linked with adenoidal hypertrophy causing OSAS. Studies have documented behavioral disturbances in children with OSAS that resolve after treatment with adenotonsillectomy **(34).**

General examination:

The physical examination should include the overall body stature, including height, weight, and blood pressure. It is important to look for chest wall deformities (as Harrison's sulcus, pigeon chest and depression of xiphisternum), thoracic spine abnormalities, or both, which can contribute or aggravate any sleep-related breathing disorder **(32).**

Systemic findings may include failure to thrive or reduced growth, pulmonary and diastolic hypertension, cor pulmonale, and pectus excavatum **(*30*).**

- **Local Examination**

1. Adenoid facies

The child with adenoidal hypertrophy will usually have the classic adenoid face, with flattening of nasolabial folds, giving the face the apathic look (idiot look) and narrow naris, long low jaw, prominent anterior part of maxilla and lengthening of the mandible with high overjet which is projection of the upper teeth beyond the lower, and elevated short pinched out upper lip, low overbite, posterior crossbite, open mouth. elongated and narrow face, small and triangular chin, mandibular retrognathia, highly-arched palate, decrease of the intermolar distance and protrusion of superior incisor teeth (prognathism), with several types of dental malocclusion **(35).**

2. Hyponasality

On physical exam, hyponasal speech can be assessed with the pinch test, where the nose is pinched and released during nasally transmitted phrases such as "banana" and "ninety-nine"; "milkman" or "Mickey Mouse" A lack of change in pitch is seen with nasal obstruction **(35).**

It is very important to differentiate between hyponasality and hypernasality where nasal emissions are present. Having the patient says phrases such as "kitty cat", "Susie sees Sally", and "hamper/pamper" are useful in screening for gross abnormalities in nasality. When in doubt, a formal speech evaluation with the use of adjunctive objective measures (such as nasometry) should be used **(2).**

3. Oral Examination

The oral examination should evaluate the size of the mandible, the dentition, dental caries and occlusion; anatomy and function of the velum, shape and size of the tongue relative to the oral cavity and oropharyngeal airway; and the degree of tonsillar hypertrophy and pharyngitis **(32).**

Gingival dryness causing xerostomia, and gingivitis foul-smelling purulent exudates, halitosis (**24**), and postnasal drip**,** can be seen on examination. Palate evaluation should be apart of the examination, particularly when adenoid surgery is done. Palatal abnormalities, such as an occult or overt submucous cleft palate may mimic or may be masked by adenoid hyperplasia. If these problems are not identified before surgery, hypernasal speech from VPI may be enhanced or become present. A bifid uvula, asymmetrical motion of the palate, midline diastases of the muscles, history of fluid regurgitation through the nose, or a family history of insufficiency or clefting should prompt nasopharyngoscopic evaluation of the palate before undertaking adenoidectomy **(2).**

Also oral cavity examination may show prognathism and high arched palate as mentioned **(35).**

4. **Anterior Rhinoscopy**

Anterior rhinoscopy can be done using the otoscope with either the nasal speculum attachment for older children or a large-ear speculum for younger children. The nasal mucosa, presence and location of secretions (sometimes after decongestion), and status of the nasal septum are evaluated. Children with obstructive adenoid hyperplasia often have a normal-appearing anterior examination; however, when the turbinates are enlarged or nasal discharge is present, gentle suctioning after the application of a topical decongestant and anesthetic can help to differentiate anterior from posterior obstruction **(36).**

The nasal cavity must be inspected for the presence of edema and erythema of the nasal mucosa, congested mucosa is sometimes present **(18).**

The nasal examination can reveal either atrophic inferior turbinate because of absent nasal airflow or hypertrophied inferior turbinates because of limited, turbulent nasal airflow **(32).**

Usually the anterior nares have a vertical slit appearance **(35).**

Other causes of nasal obstruction should also be ruled out during the examination, such as septal deviation, nasal polyps, and choanal atresia **(32).**

5. Posterior Rhinoscopy

Digital palpation of the nasopharynx is one way to evaluate adenoids and its size**,** but it is so unpleasant for the child, and in a cooperative child- examination of the nasopharynx was by a post-nasal mirror . **(37)**
Nowadays , we use obseletely the Endoscope , we use rigid endoscope in a wakeful children and flexible fiberoptic endoscope in children with general anathesia preoperative.

6. Ear examination

Otologic findings such as chronic otitis media with effusion or other signs that the Eustachian tube orifice might be physically obstructed by hypertrophied adenoid tissue **(32).**

Four characteristics of the tympanic membrane--position, mobility, color and degree of translucency--should be evaluated and described in every examination. The normal tympanic membrane is in the neutral position (neither retracted nor bulging), pearly gray, translucent and responding briskly to positive and negative pressure, indicating an air-filled space. An abnormal tympanic membrane may be retracted or bulging and immobile or poorly mobile to positive or negative air pressure **(38).**

As with symptoms in acute otitis media, the findings on physical examination also lack reliability. A poorly mobile, bulging, yellow and opacified tympanic membrane will be the otoscopic finding in acute otitis media, **(38).**

The color of the eardrum is of lesser importance than the position and mobility. The redness of the tympanic membrane alone does not suggest the diagnosis of acute otitis media. The position of the tympanic membrane is a key for differentiating acute otitis media and otitis media with effusion. In acute otitis media, the tympanic membrane is usually bulging. In otitis media with effusion, it is typically retracted or in the neutral position. The tympanic membrane can be thickened in both acute otitis media and otitis media with effusion, thereby reducing visibility through it. A yellow or greyish middle ear effusion can be seen behind the tympanic membrane in both conditions. Distinguishing acute otitis media from otitis media with effusion is clinically important. Tympanometry and acoustic reflectometry(a method of assessing the probability of middle- ear fluid) can be useful adjunctive tools to confirm the presence of fluid in the middle ear. Selective use of tympanocentesis in cases of refractory or recurrent middle ear disease can help. **(38).**

The flat tympanogram diagnosis for the presence of a middle ear effusion is more specific but less sensitive than pneumatic otoscopic diagnosis for effusion **(39).**

Additionally, hearing should be tested in adenoid patients using pure-tone audiogram and may show conductive hearing loss **(1)**

- **Nasal endoscopy:**

Although studies have demonstrated that symptoms of nasal obstruction are strongly correlated with adenoid size**,** other studies indicate the importance of a more reliable diagnosis by endoscopy for the dynamic

and three-dimensional evaluation of the nasopharynx and of the other intranasal structures **(36).** Most authors suggest that nasal flexible endoscopy is the best method to evaluate the nasopharynx **(40).**

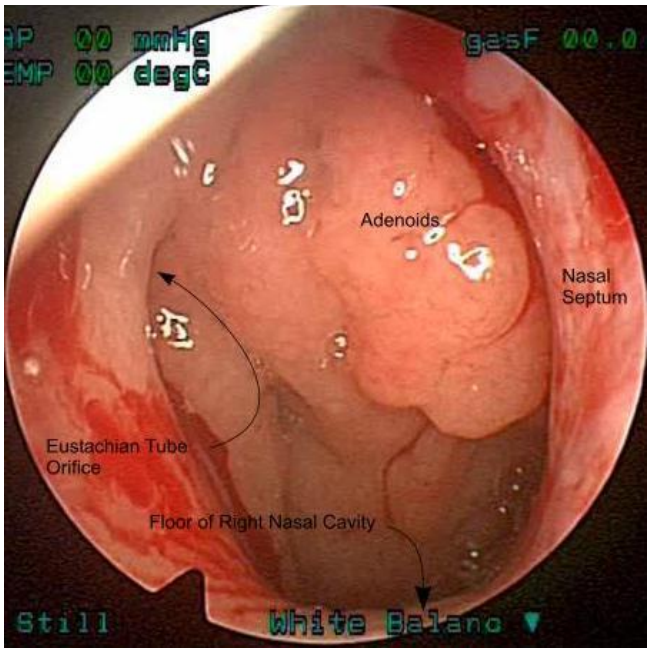


Fig. (1): Otolaryngology Houston
Nasal endoscopy

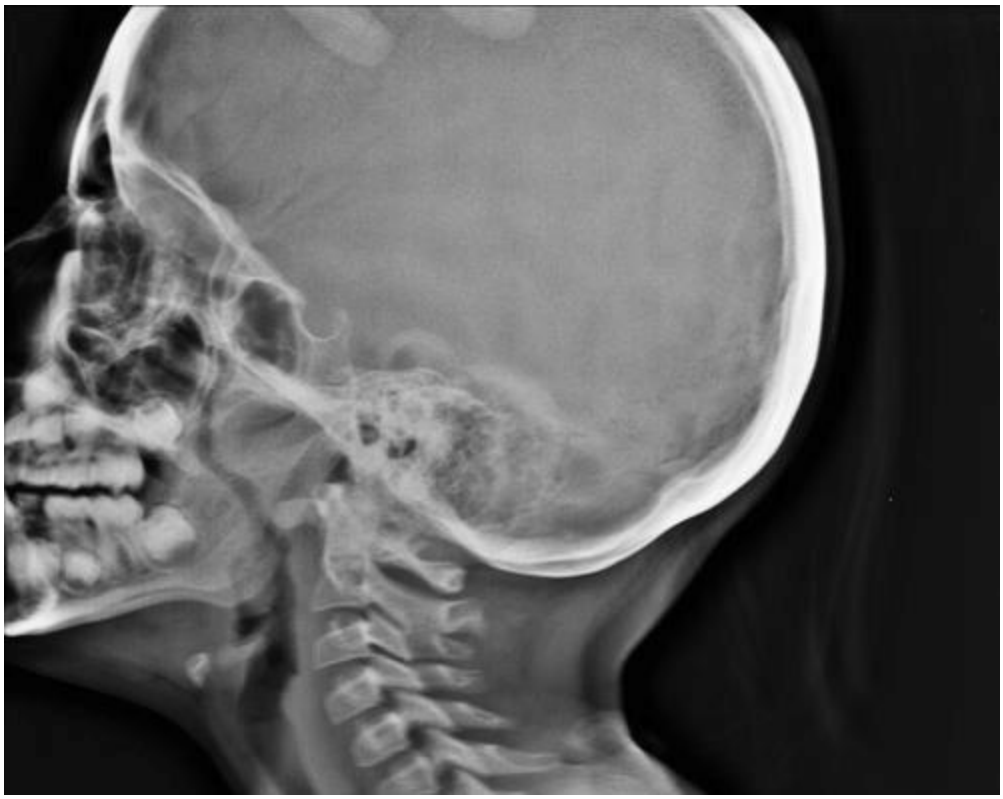
The examination by rigid endoscope was difficult to perform and uncomfortable for patients. The development of the flexible fiberoptic endoscope made the inspection of the deepest nasal areas possible. Adenoid tissue can be visualized, and the permeability of the upper airway can be evaluated**.** Moreover, this method yields better results when used for the examination of the nasal cavity and detects other obstructive factors, such as septal deviation, allergic rhinitis, choanal atresia, polyps and tumors **(36).**

Gross hypertrophy of the turbinates could make the examination difficult**,** and so a nasal decongestant is used before the procedure.The size of adenoid relative to the nasopharynx is more important than its absolute size. the adenoidal size is expressed in its relation to the post nasal space and the nasopharyngeal cavity **(40).**

Some disadvantages of endoscopy are that it is an invasive technique, and can produce some discomfort, needs a cooperative child, it only gives an approximate idea of the pharynx with no measurements, and the evaluation depends on the experience of the examiner. On the other hand it has the advantages of being performed while the child is in supine or sitting position, sleeping or awake, and performance of dynamic tests is possible that's we can use rigid telescope in awake children with setting position and flexible fiberoscope in sleeped children with supine position preoperatively. **(41).**

Radiography:

Some authors see that, when typical signs and symptoms of obstructive adenoid hyperplasia are noted, a lateral neck radiograph is unnecessary to confirm a clinically apparent diagnosis. Lateral neck films are limited by the two-dimensional representation of a three-dimensional space and by unreliability in demonstrating small choanal adenoids causing obstruction. When the symptoms and signs do not agree, a computed tomography (CT) is useful **(2).**



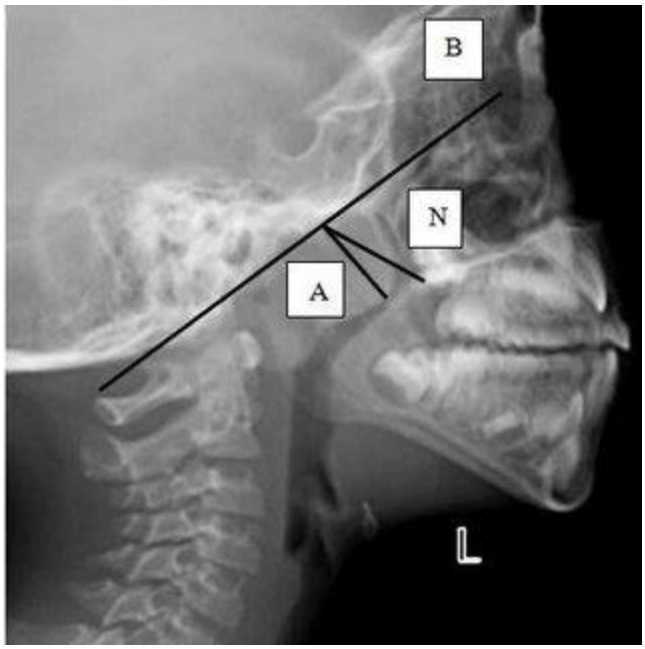
Fig(2): eMedicine specialties, pediatric otolaryngology, Adenoidectomy by **McClay et al., (8)**

On the other hand, radiographs have been chosen as the criterion standard by **Paradise** because they correlate well with volume of adenoid tissue removed during surgery. Furthermore, they are objective, noninvasive means of estimating the extent of encroachment by the adenoid on the nasopharyngeal airway **(18).**

However, the risks of exposing children to radiation when using radiography should not be ignored **,** and some authors see that X-ray examination correlates poorly with size of adenoids at operation. The main imaging study to evaluate the adenoid is a lateral neck radiograph **(42).**

The size of the adenoids on x-ray was measured using two parameters : the ANR and AA diameter.

Using the ANR , the size of the adenoids is measured relative to the size of the nasopharynx. The line, B, is drawn along the anterior margin of the basiooccput. The size of the adenoids , A, is measured from the point of maximal convexity of the shadow of the adenoid along aline perpendicular to B. The depth of the nasopharynx, N, is measured from the posterosuperior margin of the hard palate to the anteroinferior margin of the spheno-basioccipital synchondrosis. The ANR is then determined by dividing the size of the adenoids (A) by the depth of the nasopharynx (N). This is illustrated below.



Fig(3): Adenoidal-nasopharyngeal ratio**. Lertsburapa ,k et al (43)**

The AA diameter is measured as the narrowest distance between the posterior wall of the maxillary antrum and the shadow of the adenoids. The posterior wall of the maxillary antrum is on the same level as the posterior choanae on a lateral radiograph of the neck. The AA diameter is illustrated as D using the red line below :

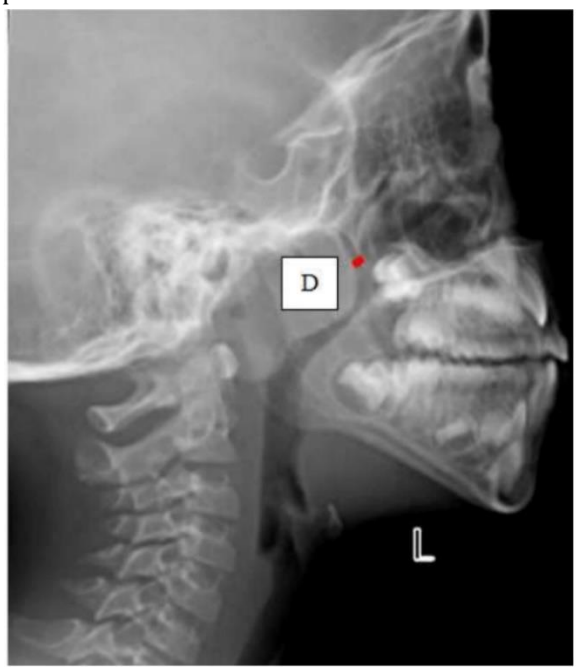


Fig (4): Antroadenoidal diameter. **Lertsburapa ,k et al (43)**

ANR was graded as follows:

- Grade I (0.0 - 0.25) no adenoid enlargement.
- Grade II (0.26- 0.50) minimal enlargement.
- Grade III (0.51- 0.75) moderete enlargement.
- Grade IV (0.76-1.00) gross enlargement.

The lateral radiogram may give a false impression of adenoid hypertrophy if the soft palate is in the posterior position (as in speech and swallowing), or the radiograph is taken when the neck flexed rather than extended. The preferred method is to assess from the posterior wall to the relaxed soft palate (seen on simple X-ray and the coronal cuts of the CT) and that from the posterior wall to the choanæ (seen on simple X-ray and the axial cuts of the CT), and to grade the adenoid obstruction on a grade of four 1+, 2+, 3+, or 4+.relative to 25%, 50%, 75%, or 100% obstruction respectively **(43).**

Over the years, various dimensions in the nasal cavity and nasopharynx have been measured to assess the degree of obstruction caused by adenoids. The goal of all techniques is to correlate the measurements with the clinical efficacy of adenoidectomy. Most techniques focus on the size of the nasopharyngeal stripe, which indicates the amount of airflow through the nasopharynx. This measurement seems to be most accurate. When the nasopharyngeal stripe is half the size of the soft palate, significant obstruction occurs **(42).**

CT scan is not normally used to evaluate the adenoids. However, when a CT scan is performed to evaluate the sinuses, the choanæ and nasopharynx are occasionally imaged, providing information on the size of the adenoids. If the CT scan does not involve the nasopharynx, information on the adenoids may be obtained from the plain sagittal scout film performed along with the CT scan. If the adenoids look abnormal or if a mass is present in the nasopharynx in an older child or in an adult, an imaging study (CT scan, MRI) is obtained to rule out a lesion other than an adenoid **(42).**

Other investigations:

Tympanometry is the graphical representation of the admittance or compliance of the middle ear. The mobility or compliance of tympanic membrane is maximum when air pressure in middle ear is equal to that of external auditory canal and the membrane is thus free from stress, it is a simple, non- invasive, objective and a widely accepted procedure in determining the presence or absence of OME. **(44)**.

Pure Tone Audiometry (PTA) is the standard measurement tool for evaluating the degree and type of hearing loss.it is non-invasive, inexpensive, easy to perform and reliable even in younger patients, it is the primary tool used to evaluate the type and degree of hearing loss. Tympanometry provides an effective screening test for the detection of negative middle ear pressure . It is rapid and reliable even in infants . Both these tests are applied to identify and quantify hearing loss associated with OME. Although polysomnography provides the best objective measurement for obstruction, it is expensive and inconvenient for most children **(45).**

For eliminating other diagnoses, such as nasal mucosal disease, allergies, and sinusitis, the child can be evaluated for allergies when signs of sneezing, itchy eyes, and skin sensitivities are present **(2).**

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