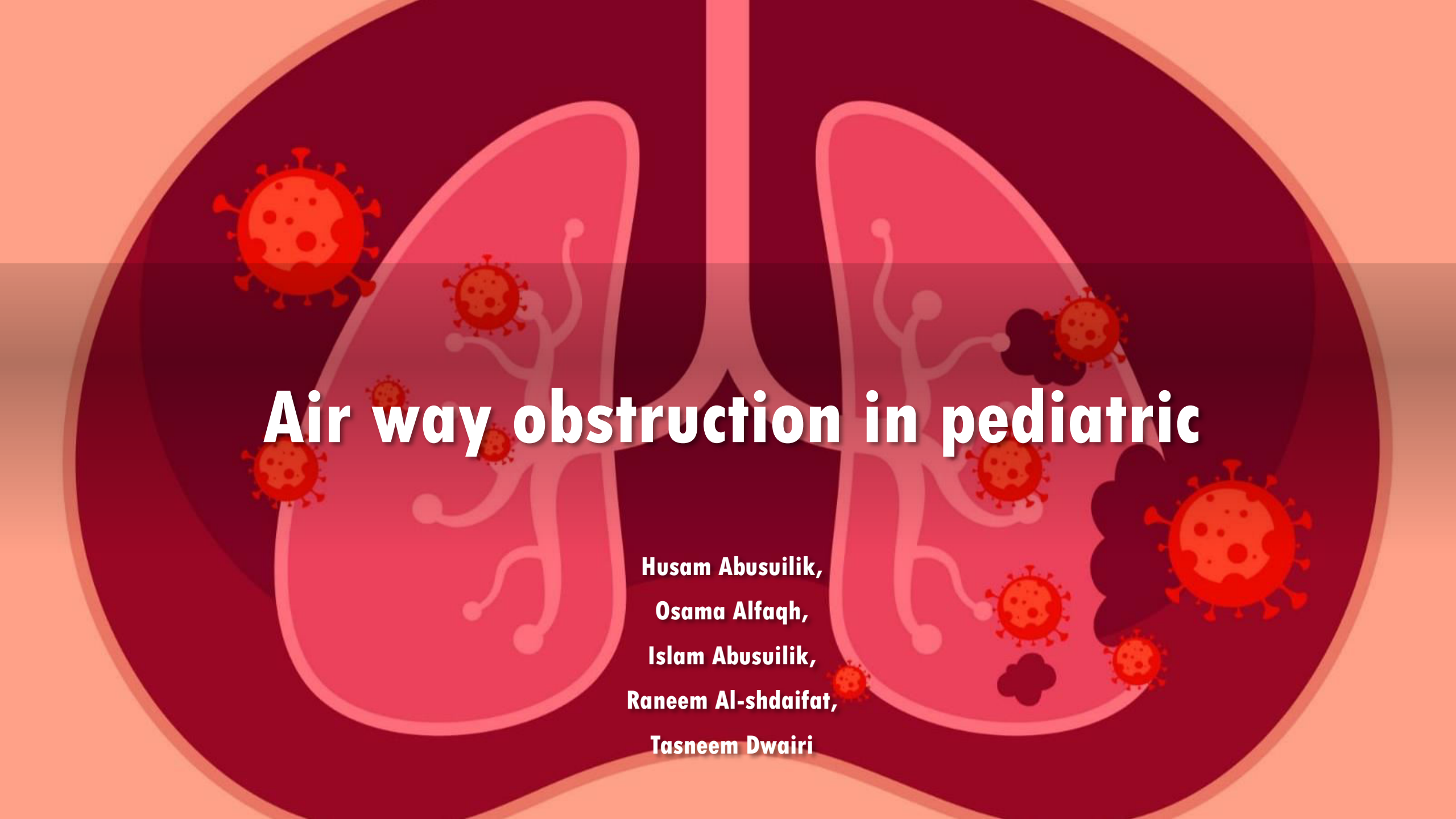
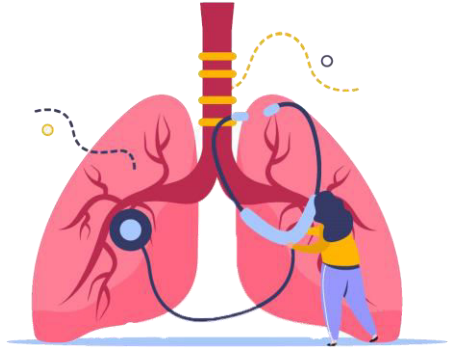


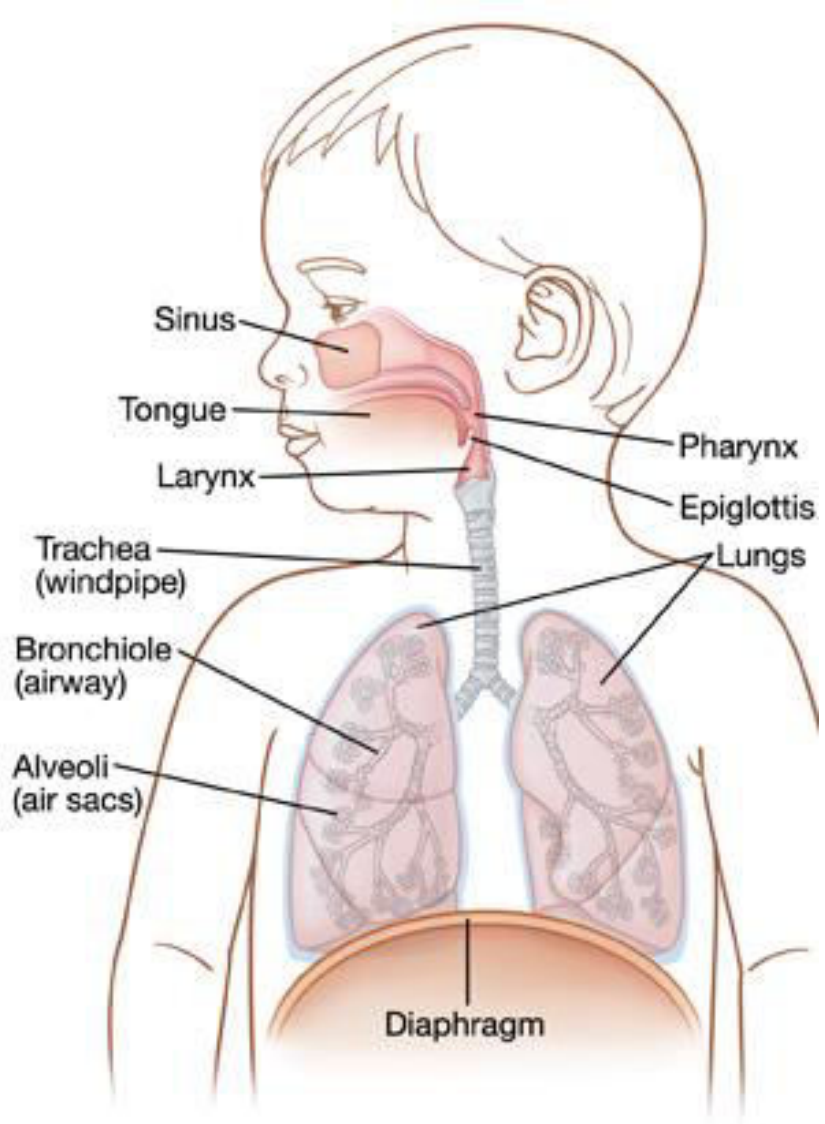
Air way obstruction in pediatric

Husam Abusuilik,
Osama Alfaqh,
Islam Abusuilik,
Raneem Al-shdaifat,
Tasneem Dwairi

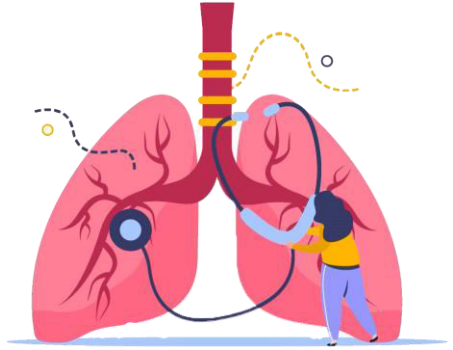
Upper & lower Airway obstruction



- **Upper airway obstructions:**
- Nose
- Throat-naso and oro-pharynx
- Mouth
- Larynx (voice box)
- **lower airway obstructions:**
- Trachea
- Bronchi
- Airways (or respiratory tract)

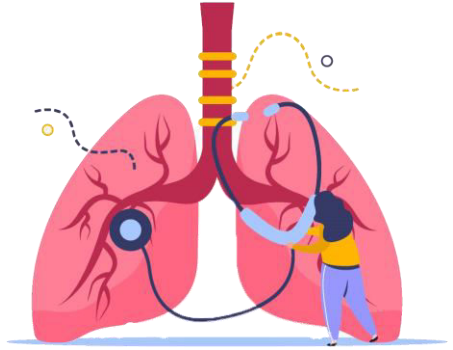


Upper Airway Obstruction



Definition

- **Upper airway obstruction (UAO)** occurs when airway obstruction occurs above the thoracic inlet, ranging from nasal obstruction due to colds to life-threatening obstruction of the larynx or upper trachea. Nasal obstruction is usually less dangerous in children, but may be more severe for neonates who breathe primarily through their noses
- The differential diagnosis of airway obstruction **varies** with patient age and can also be subdivided into **supraglottic, glottic, and subglottic causes.**



CLINICAL MANIFISTATIONS

- UAO is more pronounced during inspiration because the negative pressure generated collapses the resistance to airflow and leading to **inspiratory** noise.
- ***Stridor :*****abnormal, high-pitched sound through a partially turbulent airflow airway at the level of the supraglottic, glottic, subglottic, or trachea.**

- Stridor often decreases during sleep, because of lower inspiratory flow rates, and increases during agitation, because of higher flow rates. Occasionally stridor may also be present on exhalation. cord involvement.

Laryngomalacia (floppy larynx) is the most common cause of inspiratory stridor in infants and may be swallowing problems and gastroesophageal reflux. Hoarseness suggests **vocal cord** involvement.

- **Children with UAO may** have increased inspiratory work of breathing manifested by suprasternal

Clinical manifestations

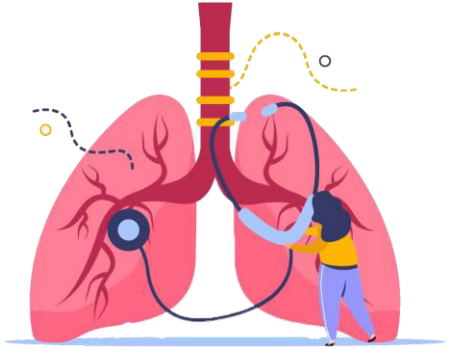
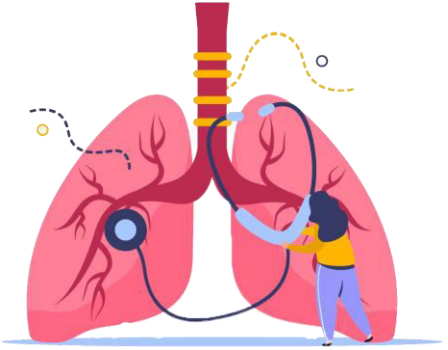


Table 135-3	Differentiating Supraglottic from Subglottic Causes of Airway Obstruction	
FEATURE	**SUPRAGLOTTIC OBSTRUCTION**	**SUBGLOTTIC OBSTRUCTION**
Common clinical syndromes	Epiglottitis, peritonsillar and retropharyngeal abscess	Croup, angioedema, foreign body, tracheitis
Stridor	Quiet	Loud
Voice	Muffled	Hoarse
Dysphagia	Yes	No
Sitting-up or arching posture	Yes	No
Barking cough	No	Yes
Fever	High (temperature >39°C)	Low grade (temperature 38°–39°C)
Toxic	Yes	No, unless tracheitis is present
Trismus	Yes	No
Drooling	Yes	No
Facial edema	No	No, unless angioedema is present

Adapted from Davis H, Gartner JC, Galvis AG, et al: Acute upper airway obstruction: croup and epiglottitis, Pediatr Clin North Am *28:859–880, 1981.*

Age Related differential diagnosis of upper airway obstruction



NEWBORN

Choanal atresia
Micrognathia (Pierre Robin syndrome, Treacher Collins syndrome, DiGeorge syndrome)
Macroglossia (Beckwith-Wiedemann syndrome, hypothyroidism, Pompe disease, trisomy 21, hemangioma)
Pharyngeal collapse
Laryngeal web, cleft, atresia
Vocal cord paralysis/paresis (weak cry; unilateral or bilateral, with or without increased intracranial pressure from Arnold-Chiari malformation or other central nervous system pathology)
Congenital subglottic stenosis
Nasal encephalocele

INFANCY

Laryngomalacia (most common non-infectious etiology)
Viral croup (most common infectious etiology)
Subglottic stenosis (congenital or acquired, e.g., after intubation)
Laryngeal web or cyst
Laryngeal papillomatosis
Vascular rings/slings
Airway hemangioma
Rhinitis

TODDLERS

Viral croup (most common etiology in children 6 mo to 4 yr of age)
Spasmodic/recurrent croup
Bacterial tracheitis (toxic, high fever)
Foreign body (airway or esophageal)
Laryngeal papillomatosis
Retropharyngeal abscess
Hypertrophied tonsils and adenoids

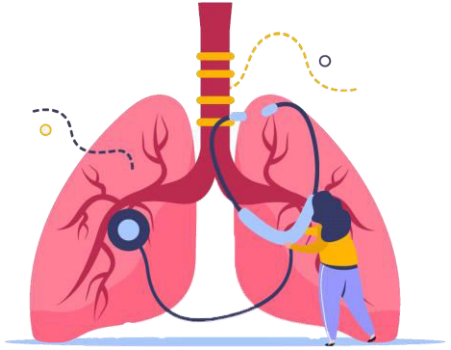
OLDER CHILDREN

Epiglottitis (infection, uncommon)
Inhalation injury (burns, toxic gas, hydrocarbons)
Foreign bodies
Angioedema (familial history, cutaneous angioedema)
Anaphylaxis (allergic history, wheezing, hypotension)
Trauma (tracheal or laryngeal fracture)
Peritonsillar abscess (adolescents)
Mononucleosis
Ludwig angina

Table 135-2 Differential Diagnosis of Acute Upper Airway Obstruction—cont'd

CLINICAL/ HISTORICAL FEATURE	RETROPHARYNGEAL ABSCESS	FOREIGN BODY	ANGIOEDEMA	PERITONSILLAR ABSCESS	LARYNGEAL PAPILLOMATOSIS
Appearance	Drooling	Variable, usually normal	Normal	Drooling, trismus	Normal
Cough	None	Variable; brassy if tracheal	Possible	None	Variable
Toxicity	Moderate to severe	None, but dyspneic	None, unless anaphylactic shock or severe anoxia is present	Dyspnea	None
Fever	Body temperature usually >38.5°C	None	None	Body temperature usually >38.5°C	None
Radiographic appearance	Thickened retropharyngeal space	Radiopaque object may be seen	Same as for viral croup	None needed	May be normal
WBC count	Leukocytosis with increased band count	Normal	Normal	Leukocytosis with increased % bands	Normal
Therapy	Antibiotics; surgical drainage of abscess	Endoscopic removal	Anaphylaxis: epinephrine, IV fluids, steroids; C1-esterase deficiency: danazol, C1-esterase infusion	Antibiotics; aspiration	Laser therapy, repeated excision, interferon
Prevention	None	Avoid small objects; supervision	Avoid allergens; FFP for congenital angioedema	Treat group A streptococcal infections promptly	Treat maternal genitourinary lesions; possible cesarean section?

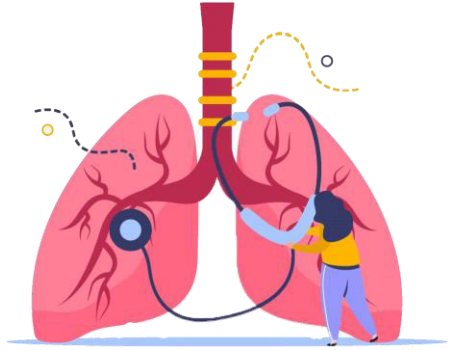
Sleep apnea



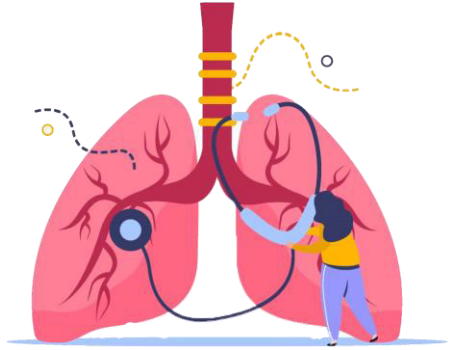
- Central or obstructive or mixed.

Apnea	>90% decrease in airflow signal that lasts ≥90% of the duration of at least 2 normal breaths, as determined from the baseline breathing pattern.	▪ Apnea is **obstructive** if there is continued or increased inspiratory effort during the entire period of decreased airflow. ▪ Apnea is **central** if inspiratory effort is absent during the entire period of airflow cessation*. ▪ Apnea is **mixed** if there is absent respiratory effort during 1 portion of the event **and** the presence of inspiratory effort in another portion, regardless of which portion comes first.

Obstructive Sleep apnea



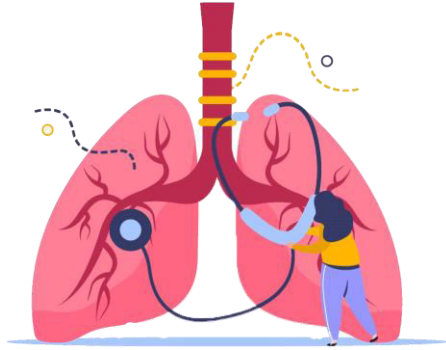
- **Obstructive sleep apnea (OSA) is characterized by episodes of complete or partial upper airway obstruction during sleep, often resulting in gas exchange abnormalities and disrupted sleep. Untreated OSA is associated with learning and behavioral problems, cardiovascular complications, and impaired growth (including failure to thrive).**
- **OSA occurs in 1 to 5 percent of children. It can occur at any age and may be most common in those between two and six years of age**



RISK FACTORS

1. Adenotonsillar hypertrophy
2. Obesity: mailny adolescence, male sex
3. Cerebral palsy
4. Down syndrome
5. Craniofacial anomalies (eg, retrognathia, micrognathia, midface hypoplasia)
6. History of low birth weight
7. Muscular dystrophy or other neuromuscular disorders

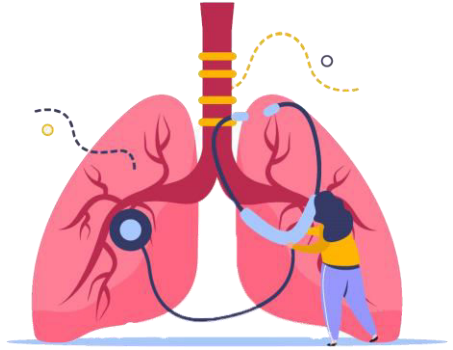
Nighttime symptoms
▪ Snoring
▪ Labored breathing; paradoxical abdominal movements
▪ Apneas, pauses in breathing, or gasping
▪ Mouth breathing
▪ Restless or agitated sleep
▪ Sleeping in unusual positions (neck extended)
▪ Nighttime sweating, preference for minimal clothing
▪ Nocturnal enuresis or nocturia
▪ Sleepwalking, sleep terrors, or confusional arousals
Daytime symptoms
▪ Mouth breathing, hyponasal speech
▪ Poor school functioning or other behavioral concerns (inattentiveness, difficulty learning, hyperactivity, impulsivity, irritability, rebelliousness, aggression)
▪ Sleepiness (eg, falls asleep at school or in the car, or excessive napping)
▪ Morning headache
Potential consequences in childhood
▪ Attention deficit hyperactivity disorder (ADHD) or disruptive behavior disorders
▪ Poor growth (especially in infants and young children)
▪ Hypertension
▪ Altered cardiac morphology (right and left ventricular hypertrophy)
▪ Pulmonary hypertension



Screening

screen for OSA, caregivers of all children should be asked about snoring during routine health care visits and visits when the airway is evaluated (such as upper respiratory illness/sick child visits), as recommended by the American Academy of Pediatrics.

- Any child who snores habitually (eg, ≥3 nights per week), has loud snoring, or has pauses in breathing should undergo a full diagnostic evaluation for OSA.



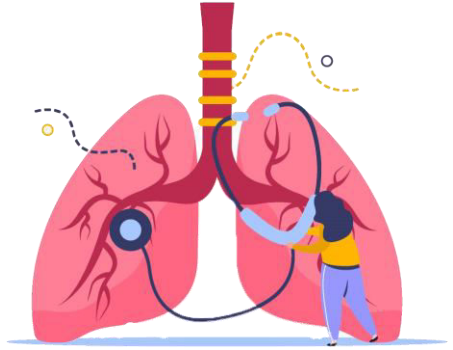
DIAGNOSIS

1) Focused sleep history.

2) Physical examination including detailed examination of the oropharynx. Growth –Obesity is an important risk factor for OSA. Paradoxically, poor growth can be a sign of chronic severe OSA, Cardiopulmonary for pulmonary hypertension, Head and nose – Craniofacial anomalies, Mouth – A high-arched and narrow hard palate.

3) Polysomnography (PSG) and/or referral to a specialist in sleep medicine or otolaryngology (ear, nose, and throat) for further evaluation and possible treatment.

Attended in-laboratory nocturnal PSG (overnight PSG) is the gold standard for diagnosis of OSA, PSG is the only diagnostic tool that can definitively identify obstructive events and quantify the severity of OSA.



Diagnostic criteria

- The following are the diagnostic criteria for pediatric OSA (all children <18 years of age) as defined by the American Academy of Sleep Medicine

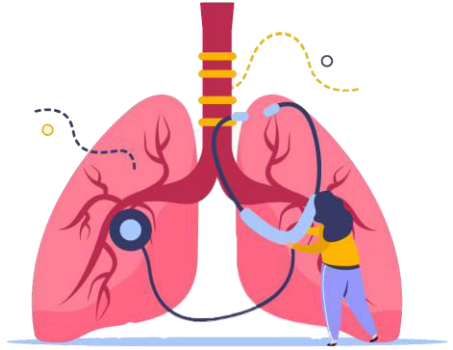
Both clinical and polysomnographic (PSG) criteria should be present for a child to be definitively diagnosed with OSA.

- **Clinical criteria** ("A criteria") – The presence of one or more of the following clinical symptoms:

- Snoring
- Labored, paradoxical, or obstructed breathing during the child's sleep
- Sleepiness, hyperactivity, behavioral problems, or learning problems

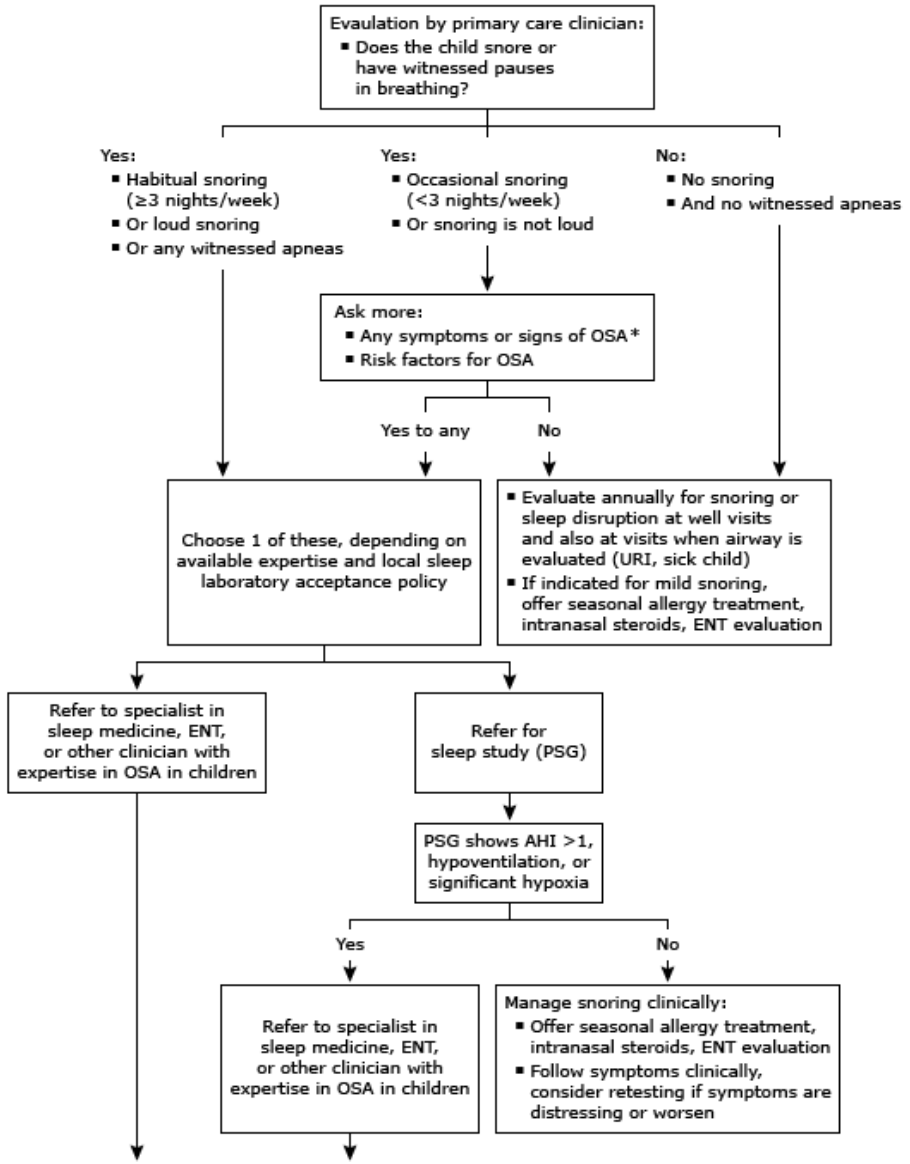
- **PSG criteria** ("B criteria") – The PSG demonstrates one or both of the following:

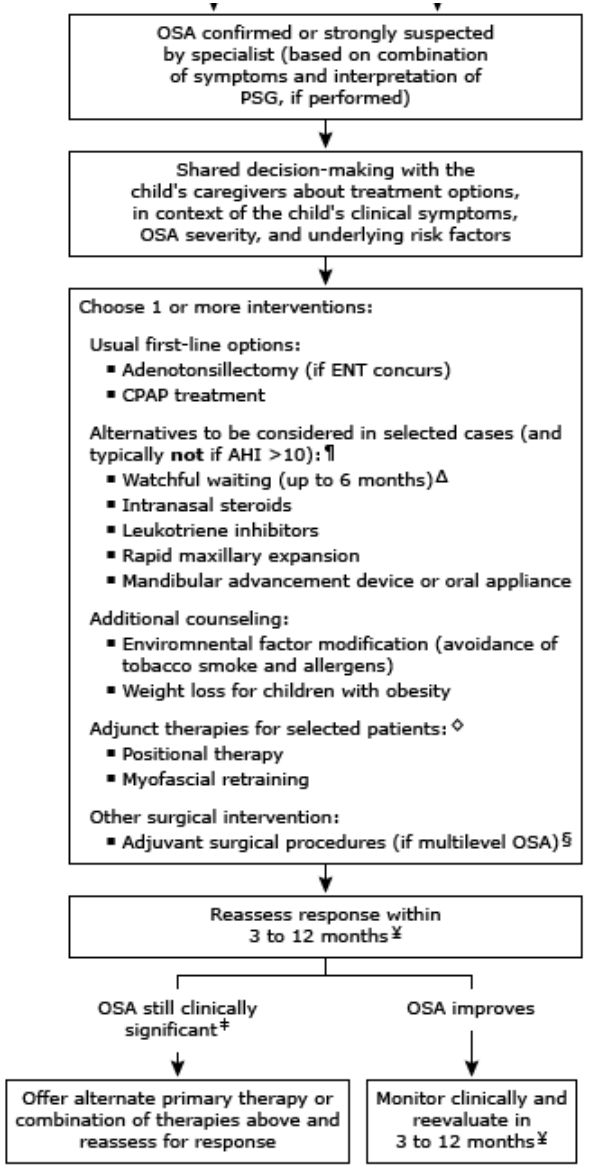
- One or more obstructive apneas, mixed apneas, or hypopneas, per hour of sleep .
- A pattern of obstructive hypoventilation, defined as at least 25 percent of total sleep time with hypercapnia (partial pressure of carbon dioxide [$PaCO_2$] >50 mmHg) in association with one or more of the following:
- -Snoring
- -Flattening of the nasal pressure waveform
- -Paradoxical thoracoabdominal motion
- Upper airway resistance syndrome and obstructive hypoventilation were previously considered distinct entities but have now been subsumed into the category of OSA



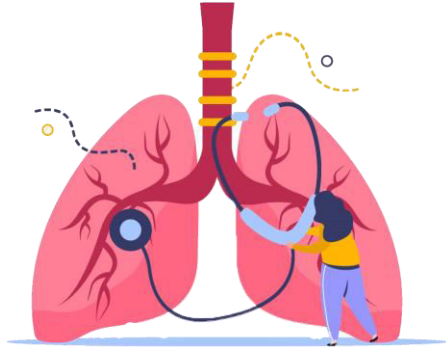
Management

- minimize exposure to environmental allergens or irritants such as tobacco smoke. Children with obesity should be offered support for weight loss.
- Usually First line
- Adenotonsillectomy
- Positive airway pressure therapy
- others
- Watchful waiting
- Orthodontics
- Adjunct therapy
- surgery


Evaulation by primary care clinician:
Does the child snore or have witnessed pauses in breathing?
Yes:
Habitual snoring (≥3 nights/week)
Or loud snoring
Or any witnessed apneas
Yes:
Occasional snoring (<3 nights/week)
Or snoring is not loud
No:
No snoring
And no witnessed apneas
Ask more:
Any symptoms or signs of OSA*
Risk factors for OSA
Yes to any
No
Choose 1 of these, depending on available expertise and local sleep laboratory acceptance policy
Evaluate annually for snoring or sleep disruption at well visits and also at visits when airway is evaluated (URI, sick child)
If indicated for mild snoring, offer seasonal allergy treatment, intranasal steroids, ENT evaluation
Refer to specialist in sleep medicine, ENT, or other clinician with expertise in OSA in children
Refer for sleep study (PSG)
PSG shows AHI >1, hypoventilation, or significant hypoxia
Yes
No
Refer to specialist in sleep medicine, ENT, or other clinician with expertise in OSA in children
Manage snoring clinically:
Offer seasonal allergy treatment, intranasal steroids, ENT evaluation
Follow symptoms clinically, consider retesting if symptoms are distressing or worsen

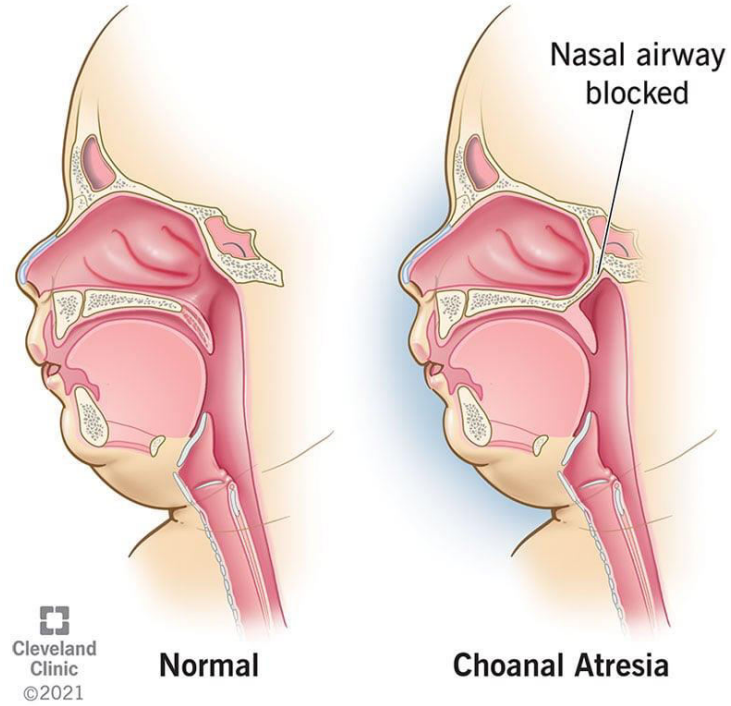

OSA confirmed or strongly suspected by specialist (based on combination of symptoms and interpretation of PSG, if performed)
Shared decision-making with the child's caregivers about treatment options, in context of the child's clinical symptoms, OSA severity, and underlying risk factors
Choose 1 or more interventions:
Usual first-line options:
Adenotonsillectomy (if ENT concurs)
CPAP treatment
Alternatives to be considered in selected cases (and typically not if AHI >10):¶
Watchful waiting (up to 6 months)Δ
Intranasal steroids
Leukotriene inhibitors
Rapid maxillary expansion
Mandibular advancement device or oral appliance
Additional counseling:
Enviromnental factor modification (avoidance of tobacco smoke and allergens)
Weight loss for children with obesity
Adjunct therapies for selected patients:◇
Positional therapy
Myofascial retraining
Other surgical intervention:
Adjuvant surgical procedures (if multilevel OSA)§
Reassess response within 3 to 12 months¥
OSA still clinically significant‡
OSA improves
Offer alternate primary therapy or combination of therapies above and reassess for response
Monitor clinically and reevaluate in 3 to 12 months¥

Choanal Stenosis (Atresia)



Definition

- Most common Congenital anatomy of the nose presenting in the neonatal period caused by failure of the posterior nasal passage to canalize completely,
- leaving either
  - bony (90%) obstruction due to outgrowth of the palatine bone
  - or membranous (10%) obstruction due to failed resorption of the oronasal membrane.
- may be bilateral or unilateral (More commonly, more right-sided)



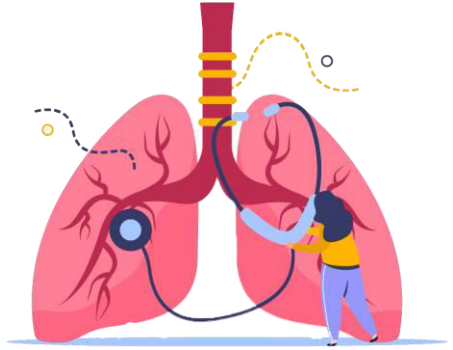
Bilateral choanal atresia

A. life-threatening
B. symptoms appear immediately after birth
C. More with respiratory distress and cyanosis

❖ Babies breathe only through their noses when they are very young, so the blocked nasal passages will cause extreme difficulty breathing

Unilateral choanal atresia

A. more common, less serious
B. sometimes appears later in childhood because the child has been able to manage while breathing through only one side of the nasal passage



Clinical Manifestations

1. Child will turn blue when feeding and then pink when crying
2. Cyclic respiratory distress relieved with crying
3. Noisy breathing
4. Feeding difficulties

❖ often associated with other developmental anomalies such as CHARGE syndrome, Treacher Collins syndrome, and Tessier syndrome

CHARGE syndrome is a set of congenital defects seen in conjunction:

C: Coloboma of the eye, central nervous system anomalies.

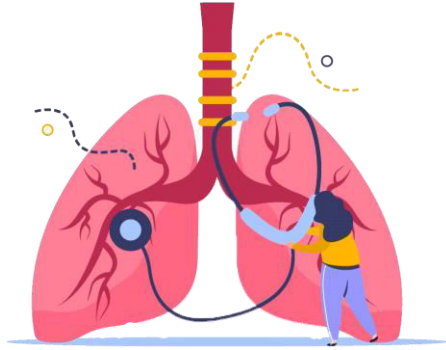
H: Heart defects.

A: Atresia of the choanae.

R: Retardation of growth and/or development.

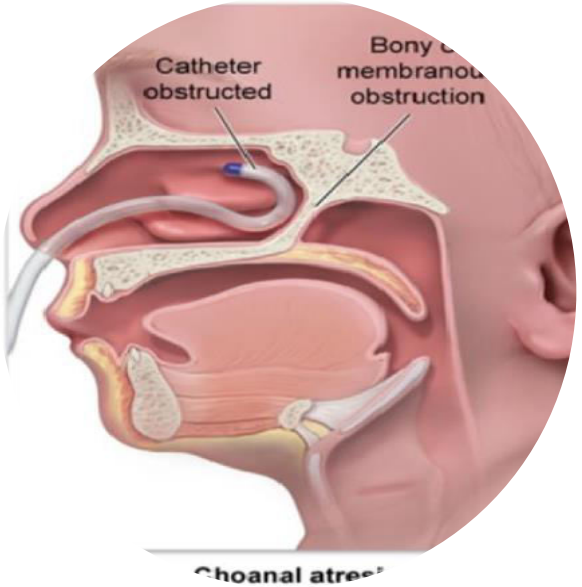
G: Genital and/or urinary defects (hypogonadism).

E: Ear anomalies and/or deafness



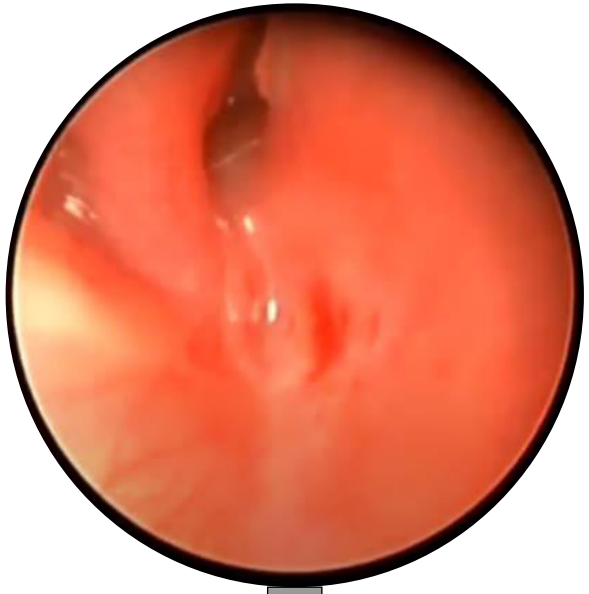
Diagnostic Studies

1. Failure to pass a catheter through the nares into the oropharynx



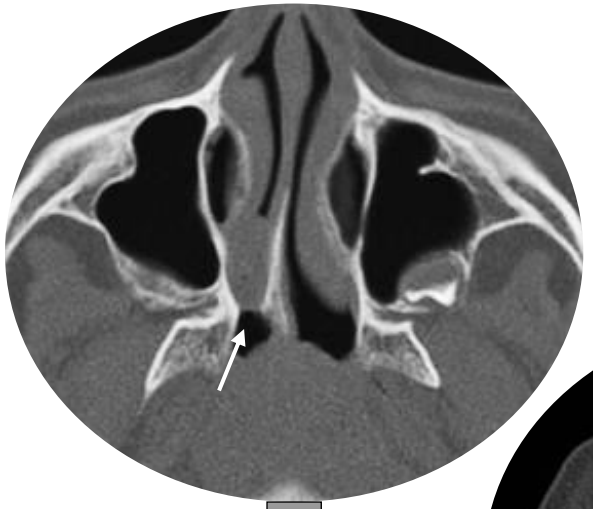
A

2. Directly inspecting with a flexible nasopharyngoscope.

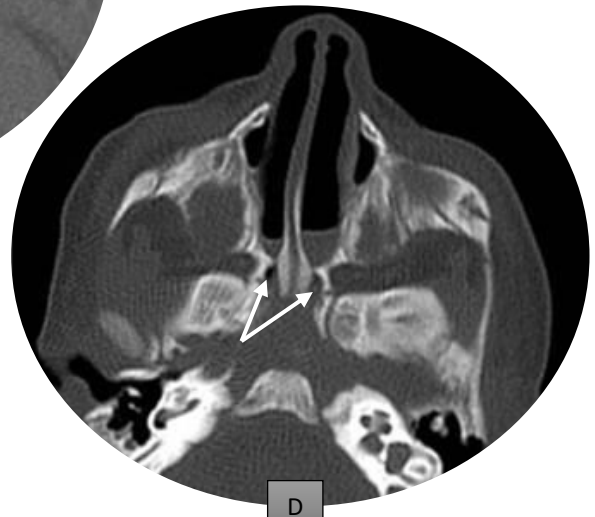


B

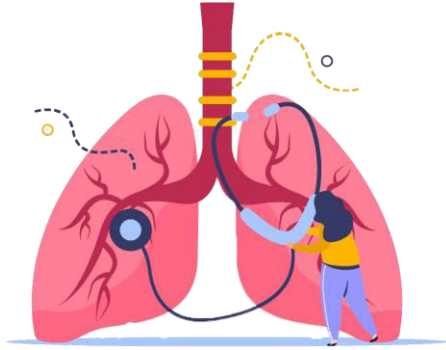
3. The diagnosis is confirmed by CT scan



C



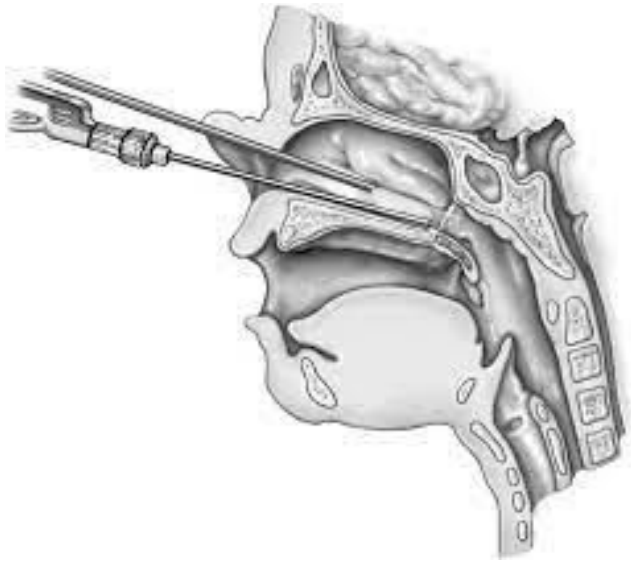
D



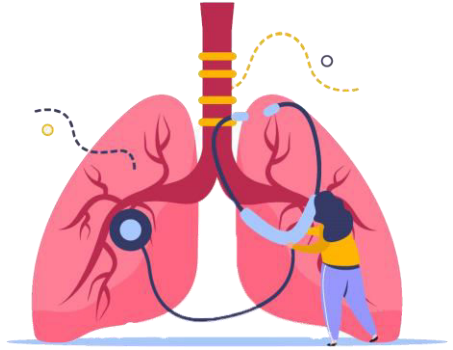
Treatment

1. **Mild Cases " a child with unilateral choanal atresia has no respiratory distress or significant feeding issues"**
   - ✓ initially be managed with close observation and occasionally supplemental oxygen.
   - ✓ Nasal saline can help to keep the nasal linings healthy and free of discharge.
   - ✓ wait until the child grows older before deciding to repair the condition.

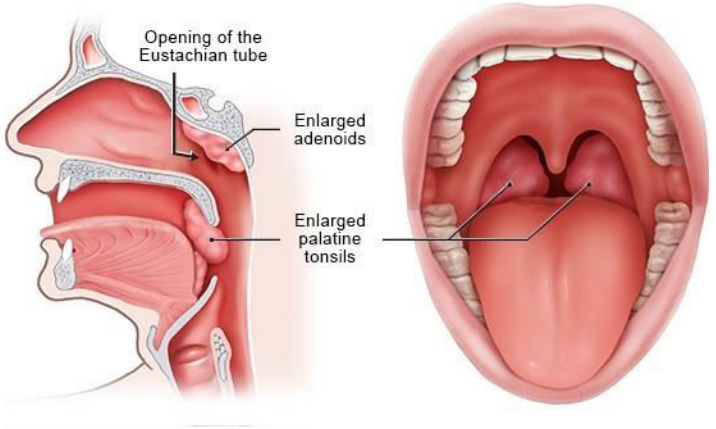
2. **Severe Cases "a child is diagnosed with bilateral choanal atresia"**
   - ✓ must be treated as soon as possible
   - ✓ Surgery should be performed as soon as the patient is stable and has been evaluated for other anomalies.
   - ✓ Transnasal endoscopic approach
     - surgeons prefer to treat children with choanal atresia
     - It can be done safely on infants just a few days old.



Adenoidal and Tonsillar Hypertrophy



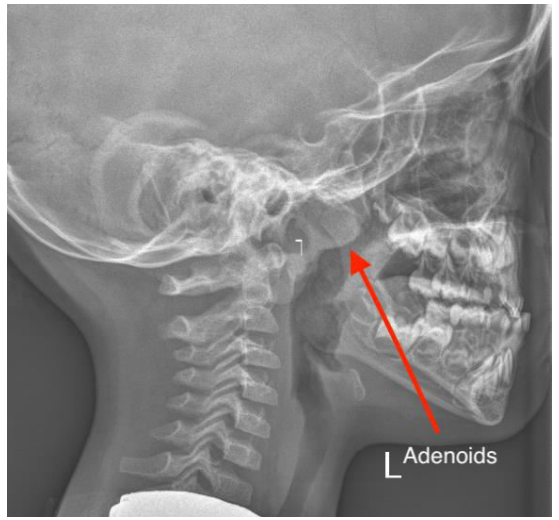
- **The <u>most common cause of chronic UAO in children</u>.**
- **may be aggravated by recurrent infection, allergy, and inhaled irritants**

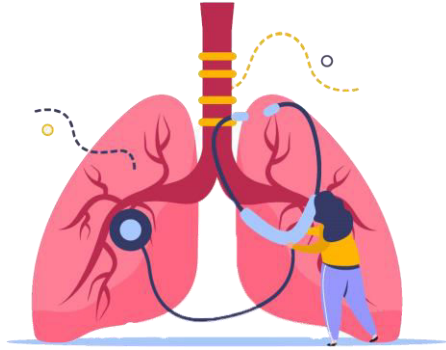


Clinical Manifestations

1. **Mouth Breathing**
2. **Snoring**
3. **Hyponasal Speech**
4. **and, in some patients, Obstructive Sleep Apnea**
5. **recurrent or persistent Otitis Media (eustachian tubes can be obstructed by enlarged adenoids)**

Diagnostic Studies: **assessed by a lateral radiograph of the nasopharynx or by flexible nasopharyngoscopy**





Treatment

1. **If the adenoids or tonsils are large and thought to be significantly contributing to UAO, then the most effective treatment is surgical removal (adenoidectomy).**
   - ❖ **Because the adenoids are not a discrete organ but rather consist of lymphoid tissue, regrowth is possible, especially in preschool children.**

2. **If the tonsils are large and the obstruction is severe, the removing of the tonsils (tonsilectomy) in addition to the adenoids may be necessary.**

Table 2. Indications for Adenoidectomy

- Obstructive sleep apnea due to adenotonsillar hypertrophy
- Chronic adenoiditis
- Chronic sinusitis
- Repeat surgery for otitis media with effusion

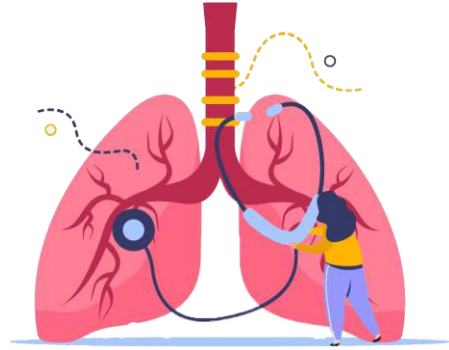
Table 1. Indications for Tonsillectomy

Absolute

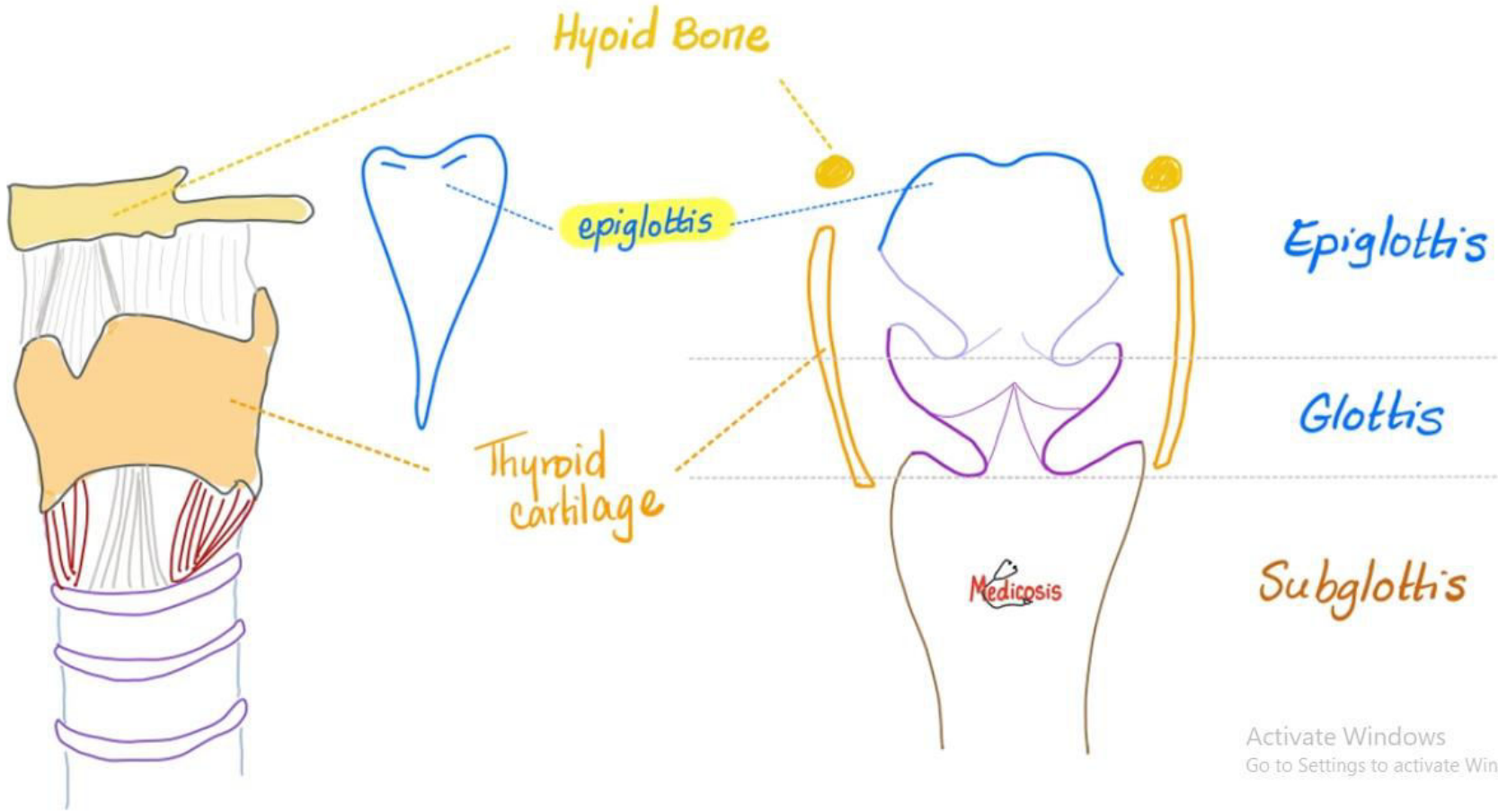
- Obstructive sleep apnea syndrome due to adenotonsillar hypertrophy
- Suspected malignancy
- Recurrent hemorrhage

Relative

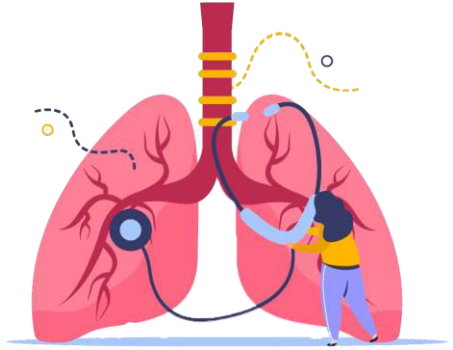
- Recurrent tonsillitis
- Recurrent peritonsillar abscess



ANATOMY OF THE LARYNX

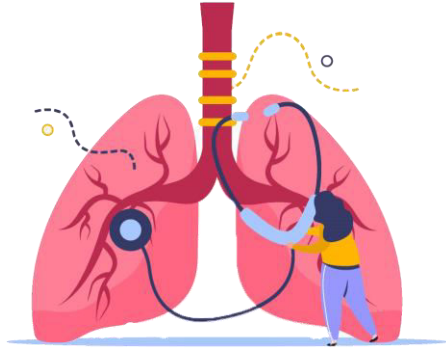


Laryngomalacia (Floopy larynx)



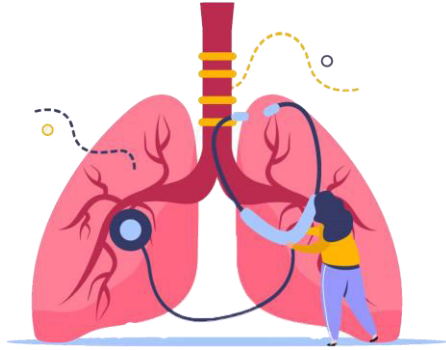
Definition

- *Is a congenital abnormality of the laryngeal cartilage(softening of tissue of the larunx)
- It is a dynamic lesion resulting in collapse of the supraglottic structures (epiglottis and arytenoid cartilages) during inspiration, leading to airway obstruction.



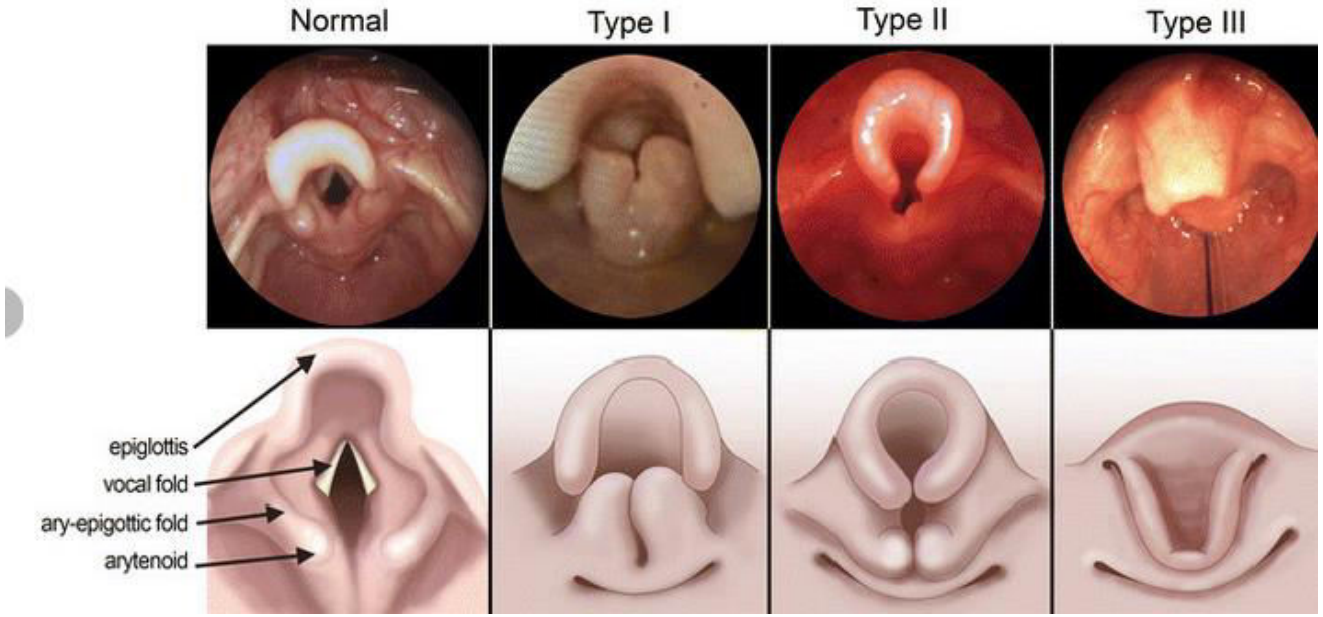
- This is the most common cause of stridor in infants.
- In most cases, laryngomalacia in infants is not a serious condition,they have noisy breathing, but are able to eat and grow.

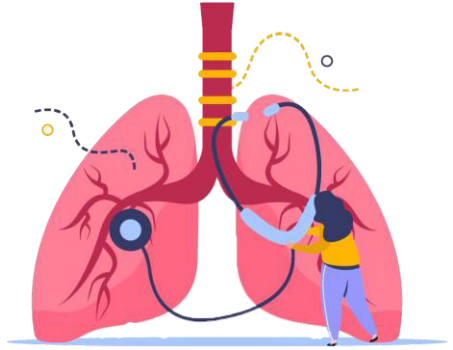
- It usually does not result in significant respiratory distress, but occasionally it is severe enough to cause hypoventilation, with hypercarbia, hypoxemia, and difficulty with feeding



Classifacation:

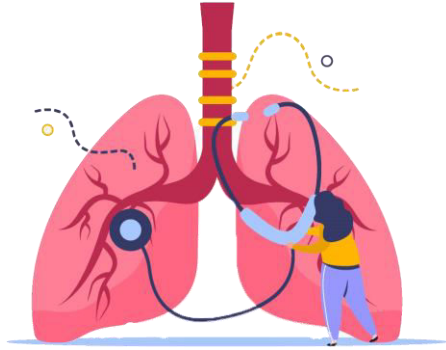
- In type 1 laryngomalacia : the aryepiglottic folds are tightened or foreshortened.
- Type 2 : is marked by redundant soft tissue in any area of the supraglottic region.
- Type 3 :Posterior displacement of the epiglottis and associated with other disorders, such as neuromuscular disease and gastroesophageal reflux.





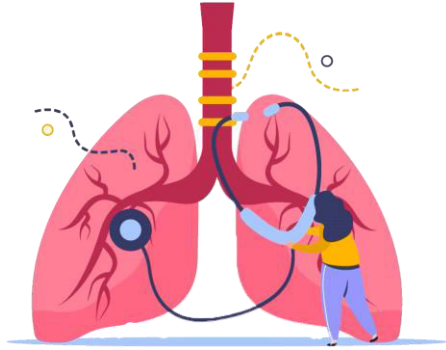
Clinical Manifestations

- The primary sign of laryngomalacia is inspiratory stridor with little or no expiratory component.
- The stridor is typically loudest when the infant is feeding or active or in supine position and decreases when the infant is relaxed or placed prone, or when the neck is flexed.
- Any condition that increases upper airway inflammation will exacerbate laryngomalacia, including:
    1. viral respiratory infections
    2. dysphagia (swallowing dysfunction)
    3. gastroesophageal reflux.



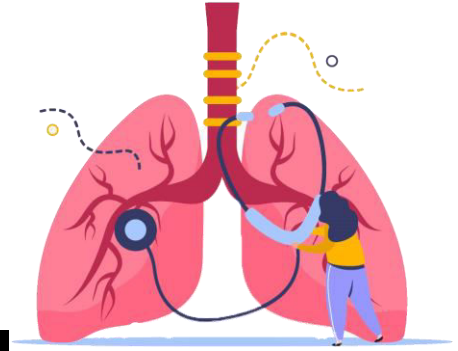
- Laryngomalacia normally peaks by 3 to 5 months of age and resolves between 6 and 12 months of age.

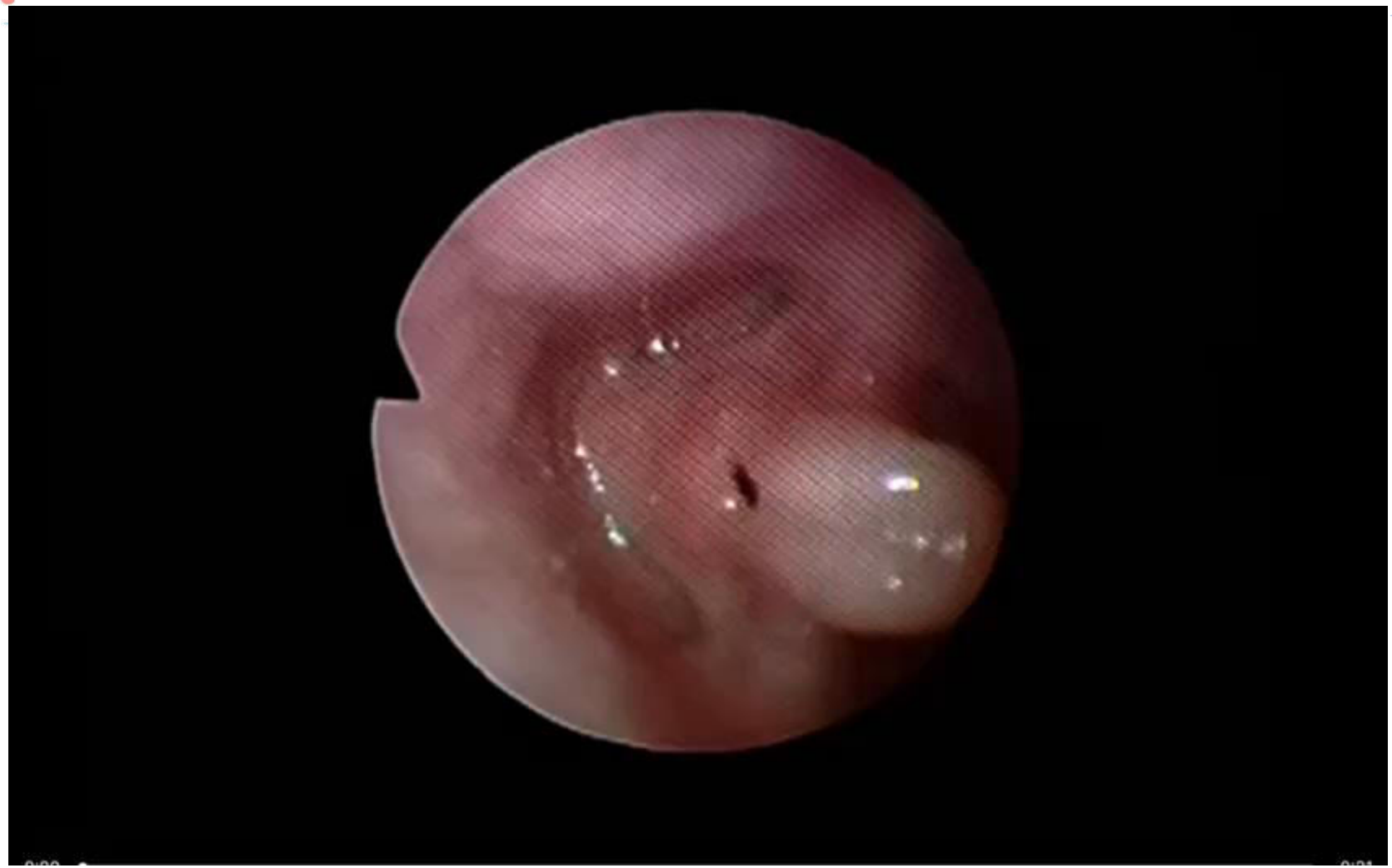
- However, occasionally it can persist in otherwise normal children up until 24 months of age and even longer in children with underlying conditions, especially those with Neurologic diseases affecting control of upper airway muscles (such as cerebral palsy).

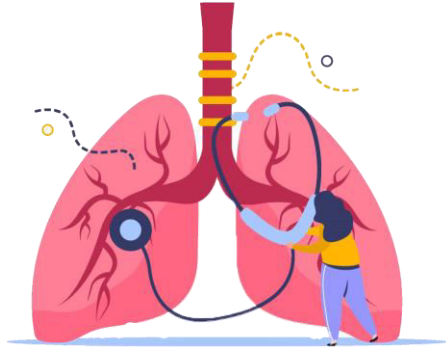


Diagnosis:

- The diagnosis is made clinically but can be confirmed with visualization of the larynx by direct or flexible fiber-optic laryngoscopy
- (omega-shaped epiglottis and collapse of the supraglottic structures during inspiration)





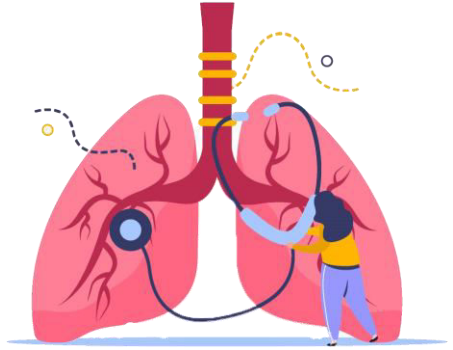


Treatment:

- Most infants with laryngomalacia will feed, grow, and ventilate normally with spontaneous resolution of stridor by age 18 months.

- Supraglottoplasty for severe cases.

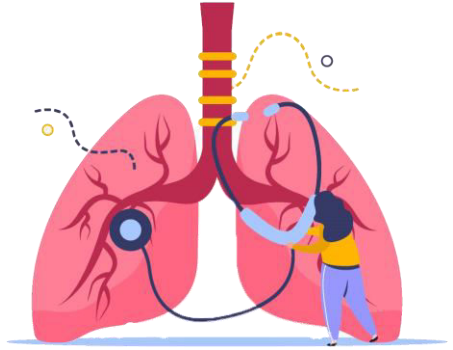
Subglottic Stenosis



Definition

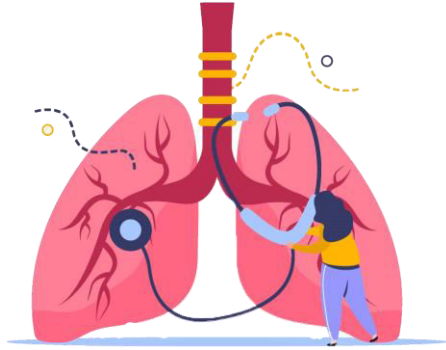
- Subglottic stenosis is a narrowing of the airway below the vocal cords (subglottis) and above the trachea.
- Subglottic stenosis will involve narrowing of the cricoid, the only complete cartilage ring in the airway.
- This narrowing is often caused by scarring in the larynx just below the vocal cords but may also involve the vocal cords and affect the voice as well.

- The diameter of a normal newborn subglottis is 4 mm. If the subglottis is less than 3.5 mm, it is considered narrow.



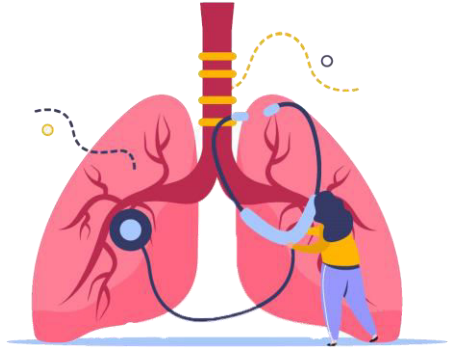
There two kind of subglottic stenosis:

- **Acquired subglottic stenosis often occurs after long periods of intubation and ventilation for respiratory problems.**
- **Congenital subglottic stenosis occurs as a rare birth defect and may be associated with other genetic syndromes and conditions. The airway remains narrow because the airway cartilage did not form properly before birth.**



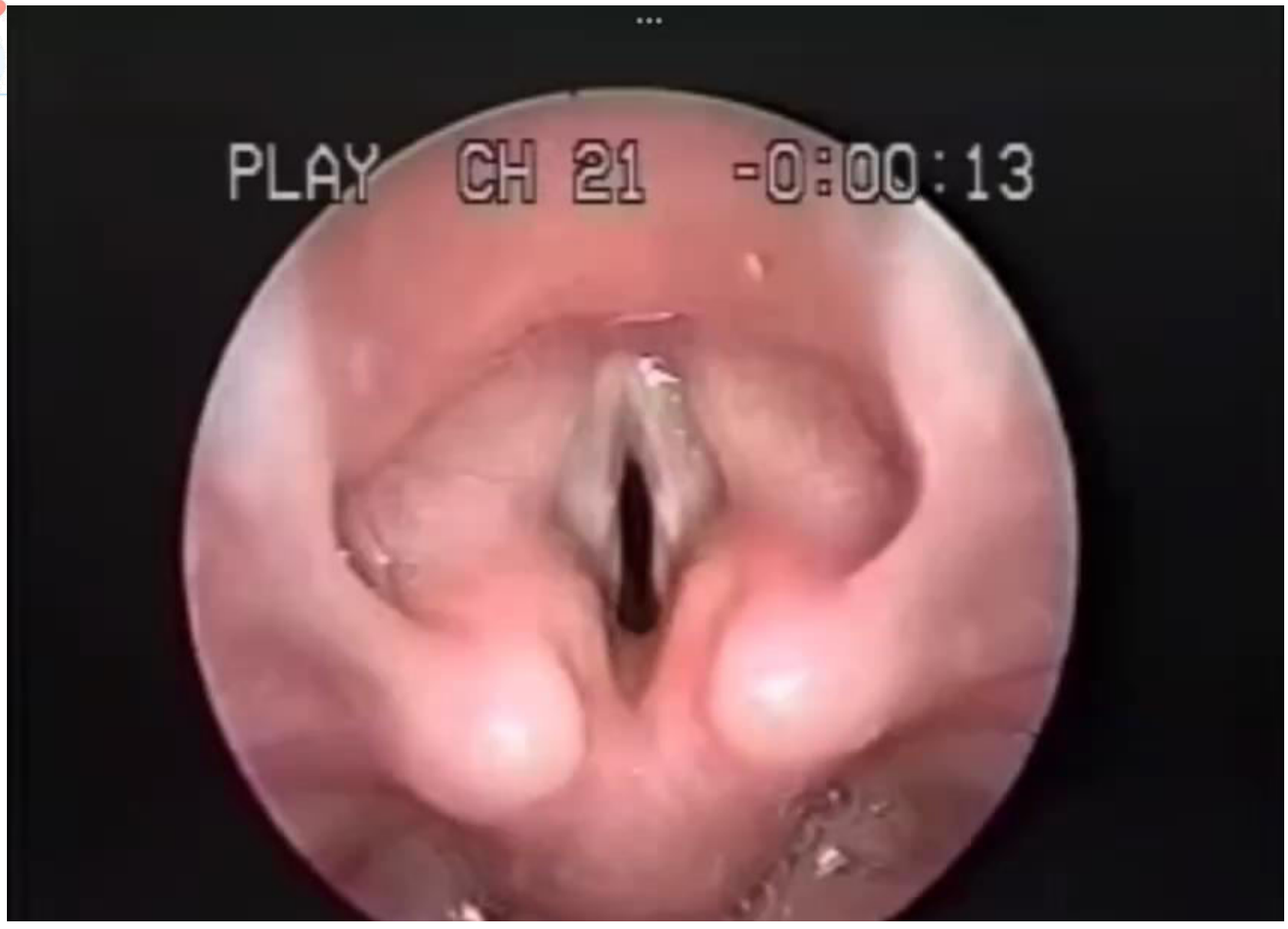
Signs and symptoms:

- High pitched noisy breathing (stridor) that occurs when your child breathes both in and out. This can worsen when your child is ill or after exercise or strenuous activity.
- At times, children have been diagnosed with multiple episodes of croup at a young age.
- Poor weight gain
- Increased effort to breathe with pulling in the neck, between or under the ribs



Diagnosis:

- The history of specific risk factors is important in reaching the diagnosis.
- Further evaluation is done with X-rays. Neck X-rays may reveal subglottic narrowing.
- On these films the trachea is evaluated for tracheal narrowing or stenosis or complete rings.
- Definitive diagnosis is made on endoscopy with microlaryngoscopy and bronchoscopy.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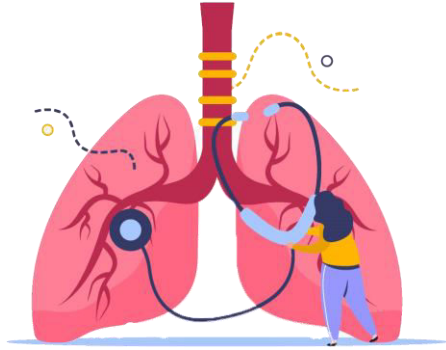
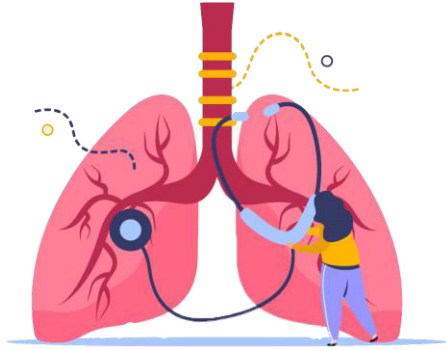
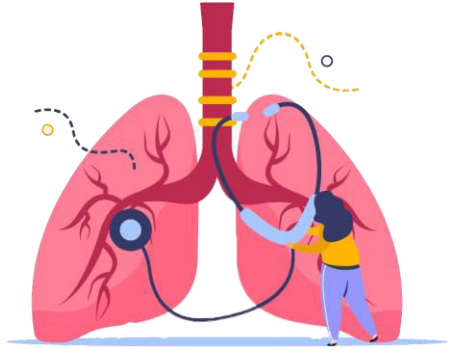




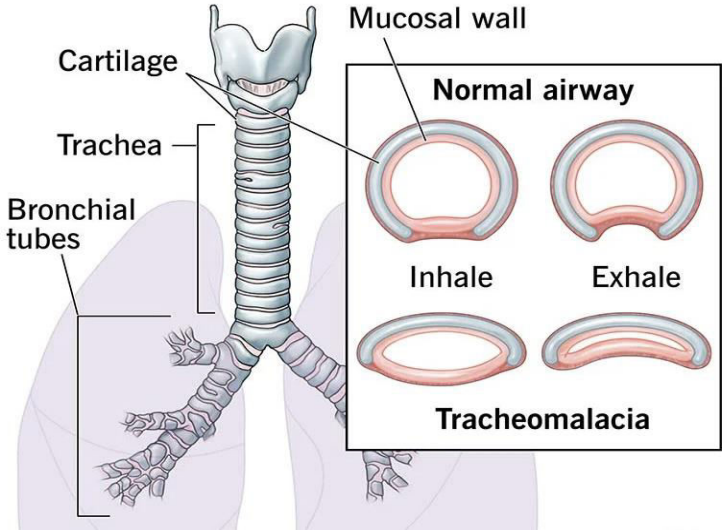
Treatment:

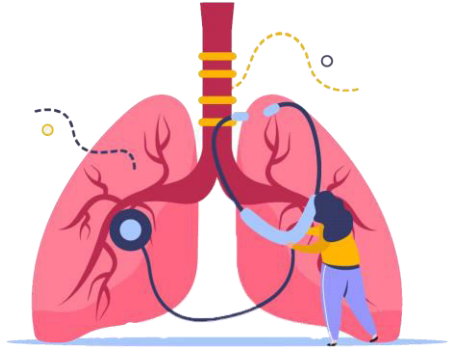
- Treatment depends on the severity of your child's stenosis.
- *Your child may outgrow the problem without intervention or, if the problem is severe, surgery may be required.
- *treatment plan may include:
- Endoscopic surgery : In some cases, the airway may be dilated (opened) with a balloon. Lasers can be used to rempve segmental portions of scar tissue.
- Open surgery: The two most common surgical procedures to treat this condition are:
- Laryngotracheoplasty
- Cricotracheal resection

Tracheomalacia



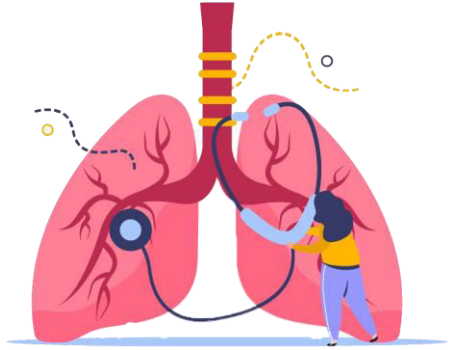
- A structural abnormality involving softening or weakening of the tracheal cartilage and/or posterior membrane that results in excessive tracheal collapsibility.
- When the bronchi are also affected, the condition is called "tracheobronchomalacia" (isolated bronchomalacia is extremely rare).





Etiology

- Tracheomalacia is often classified as congenital (primary) or acquired (secondary)
- **Congenital (primary)**
  - Idiopathic
  - Cartilage abnormalities (e.g., polychondritis)
  - Underdeveloped respiratory tract (e.g., prematurity, bronchopulmonary dysplasia)
  - Associated with congenital syndromes (e.g., trisomy 21) and/or birth defects (e.g., tracheoesophageal fistula)
- **Acquired (secondary)**
  - Tracheal trauma (e.g., prolonged intubation, tracheotomy, chest trauma)
  - Airway inflammation (e.g., chronic infections, tracheobronchitis)
  - External compression (e.g., vascular abnormalities, tumors)

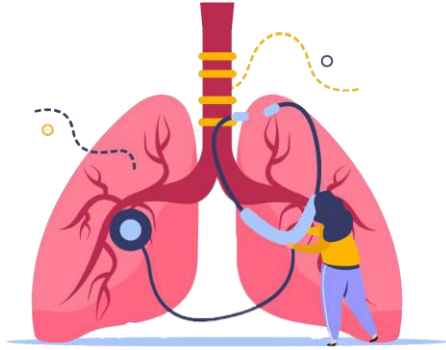


Clinical features — Signs and symptoms depend upon the location and severity of the tracheal lesion.

- In pediatric patients, symptoms usually appear during the first 2–3 months of age.
- Intrathoracic lesions typically present with a recurrent harsh, barking, or croup-like cough.
- whereas extrathoracic lesions cause inspiratory stridor
- both may result in respiratory distress

Diagnosis

- Definitive diagnosis usually is made by bronchoscopy (Flexible bronchoscopy is preferred)



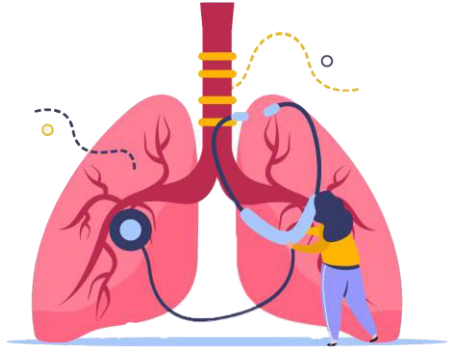
Management — The long-term prognosis of this disorder is good in those children who have no associated problems.

- Most affected infants **improve spontaneously** by 6 to 12 months of age. However, some remain symptomatic or have exercise intolerance as adults.
- Intervention may be needed in children with life-threatening episodes of airway obstruction, recurrent infection, respiratory failure, or failure to thrive

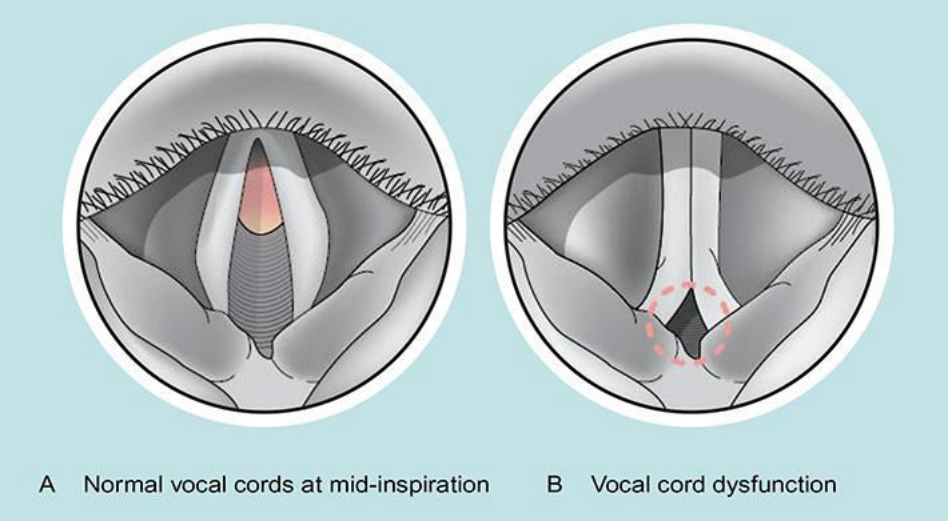
Interventions include:

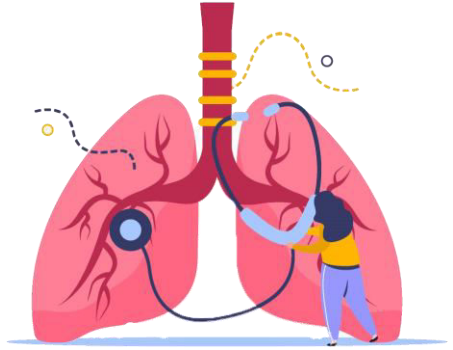
- Continuous positive airway pressure
- Surgical approaches such as tracheal reconstruction

Vocal cord dysfunction



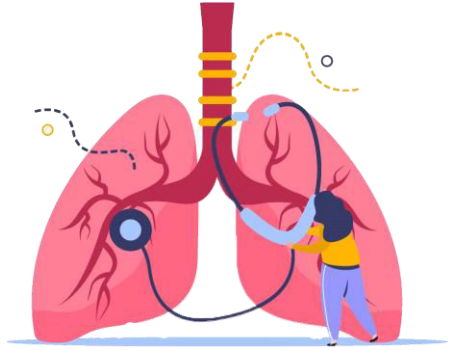
- A condition characterized by episodic, paradoxical vocal cord adduction during inspiration, which can result in sudden dyspnea.
- For many children, vocal cord dysfunction (also known as paradoxical vocal cord motion) happens while they are exercising or when they are exposed to certain irritants like strong odors, stress and anxiety, or upper respiratory infections.





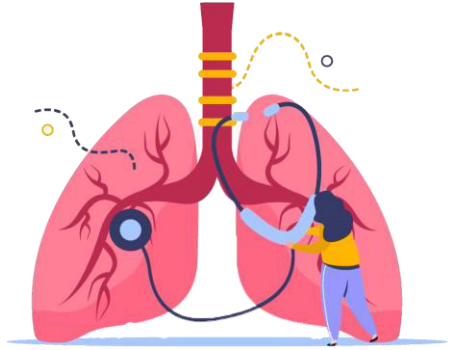
Sign and symptoms

- Children with vocal cord dysfunction may experience difficulty inhaling, shortness of breath, noisy breathing (stridor), tightness in the throat area, coughing.
- Note : Vocal cord dysfunction causes asthma-like symptoms, and is often mistaken for asthma, especially asthma caused by exercise. Vocal cord dysfunction can also co-occur with asthma.



Diagnosis

- **Flow-volume loop** Usually the expiratory loop will be normal in a patient with VCD, but the inspiratory loop will be flat.
- **Laryngoscopy** when the patient is symptomatic is the gold standard for the diagnosis
- **Imaging —** is predominantly used to exclude other causes of dyspnea



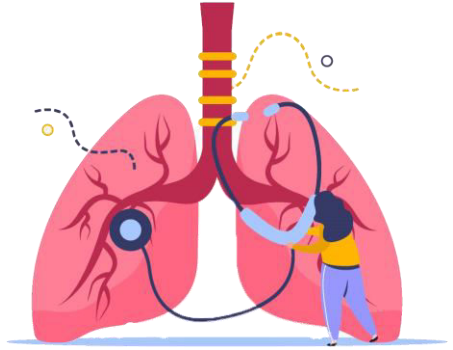
Acute Management :

- reassurance and supportive care until the episode spontaneously resolves
- Inhaled bronchodilators are not useful
- Use of continuous positive airway pressure
- While rarely used, inhalation of a helium-oxygen mixture (heliox) has been reported to be helpful

Long-term prevention :

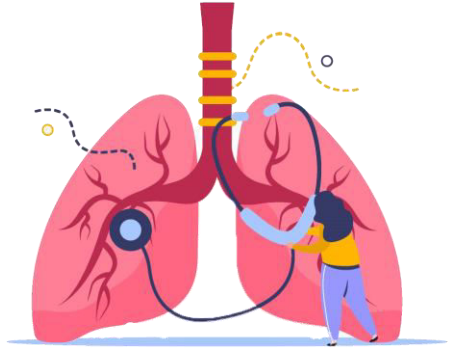
- Avoidance of airway irritants
- Speech-behavioral therapy
- Psychotherapy

Bronchiolitis obliterans



Defenition

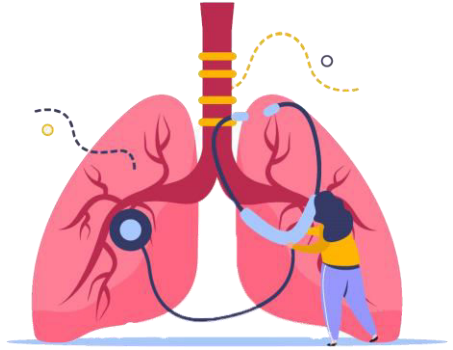
- Bronchiolitis obliterans is also known as obliterative bronchiolitis or constrictive bronchiolitis. is a type of obstructive lung disease of the small airways. It is a rare disease with characteristic features of fibrosis of terminal and distal bronchioles and spirometry showing airflow obstruction. It usually leads to a progressive decline in lung function and has variable outcomes.



Etiology

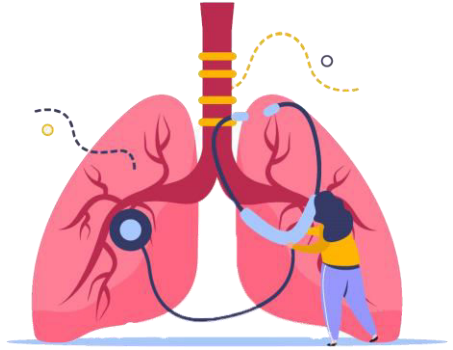
- It is one of the noninfectious complications after lung transplant and hematopoietic stem cell transplantation (Graft-versus-host disease) . In this case, it is called bronchiolitis obliterans syndrome.
- Other etiologies:

- exposure to inhaled toxins and gases, including sulfur mustard gas, nitrogen oxides, diacetyl (used as popcorn flavoring),
- autoimmune disorders, especially rheumatoid arthritis, SLE, and less commonly with inflammatory bowel disease.
- known to occur after a respiratory viral infection (adenovirus, respiratory syncytial virus).
- Other associations include microcarcinoid tumorlets and Cryptogenic constrictive bronchiolitis.



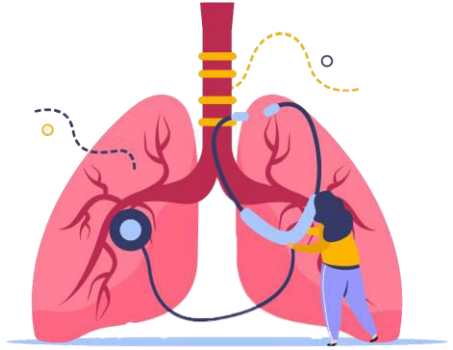
Pathophysiology

- There is inflammation in response to injury from inhalational toxins or autoimmune responses, leading to fibroproliferation and abnormal regeneration of epithelium of the small airways.
- Hypertrophy of the smooth muscles of the bronchioles, peribronchiolar inflammatory infiltrates, accumulation of mucus in the bronchiolar lumen, and bronchiolar scarring can be noted.



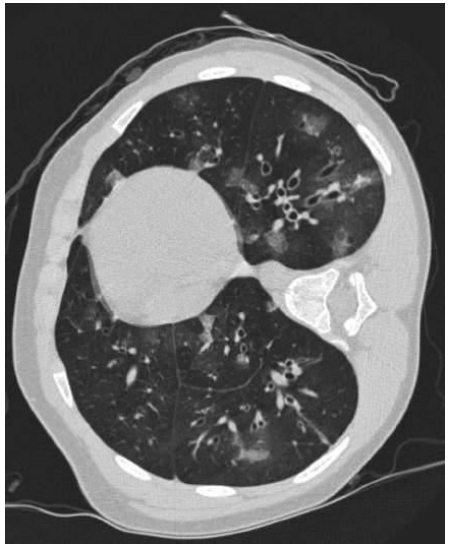
History and physical examination

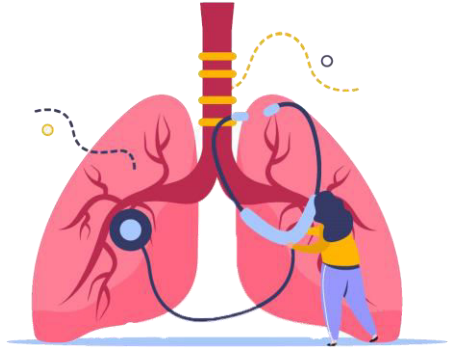
- Bronchiolitis obliterans characteristically presents with dyspnea and persistent progressive cough Some may also have wheezing. Symptoms usually develop over weeks to months and are not episodic. History may also elicit recent exposure to toxic fumes or gases, viral infections, prior lung, or hematopoietic stem cell transplantation.
- A physical exam may reveal decreased breath sounds and a prolonged expiratory phase with or without wheeze.



Diagnosis

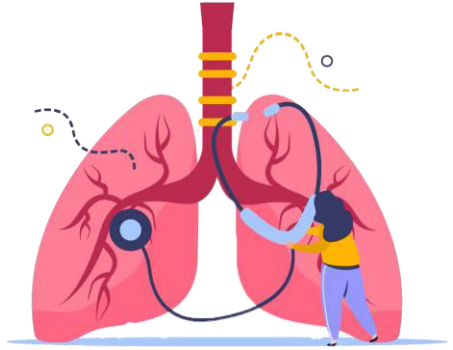
- **Spirometry** demonstrates airflow obstruction that does not reverse with inhaled bronchodilator challenge
- **Chest radiographs** may be normal in early disease or show signs of hyperinflation
- **Chest CT imaging** may show bronchial wall thickening, mosaic pattern with patchy areas of hypo attenuation.
- **lung biopsy** (best means of diagnosis)





Treatment

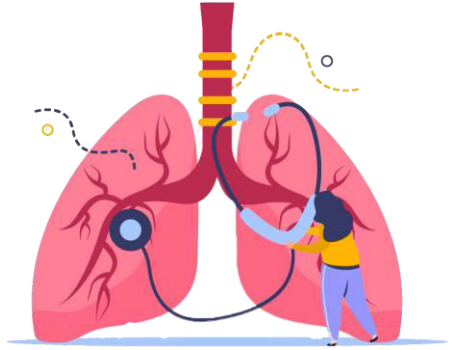
- **No definitive therapy exists for BO**
- **we can use:**
- **Immunomodulatory agents, such as sirolimus, tacrolimus, aerosolized cyclosporine, hydroxychloroquine, and macrolide**
- **Corticosteroids**
- **Supportive measures with oxygen, antibiotics for secondary infections, and bronchodilators are adjunct therapies.**
- **Lung transplant in severe cases (curative) , but it might recur after transplant**



Prognosis

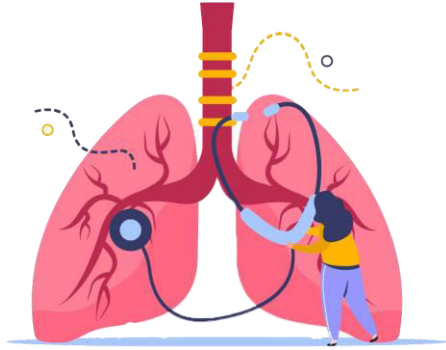
- The prognosis of bronchiolitis obliterans depends on the underlying pathology
- Lung function often deteriorates over time,
- Progressive disease may require supplemental oxygen or mechanical ventilation in very severe cases.
- The worst cases may require a lung transplant.

Foreign Body Aspiration



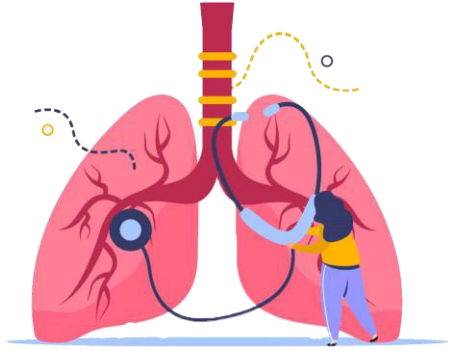
Epidemiology

- The majority of children who aspirate foreign bodies are under 3 years of age mostly nuts and toys (Coins more often lodge in the esophagus than in the airways).
- Patients with developmental delay or with older siblings are at increased risk.
- foreign bodies tend to lodge in right-sided airways (less acute angle than the left).



Clinical manifestation

- many present with choking and witness aspiration
- Physical findings observed with acute foreign body aspiration may include cough, localized wheezing, unilateral absence of breath sounds, stridor, and, rarely, bloody sputum.
- some foreign bodies remain in the lung for long periods of time and may present with persistent cough, sputum production, or recurrent unilateral pneumonia.
- Foreign body aspiration should be in the differential diagnosis of patients with persistent wheezing unresponsive to bronchodilator therapy, persistent atelectasis, recurrent or persistent pneumonia, or chronic cough without another explanation.
- Foreign body may lodge in the esophagus and compress the trachea causing respiratory symptoms.



Diagnostic study

- Radiographic studies will reveal the presence of radiopaque objects and can also identify focal air trapping.
- Many foreign bodies are not radiopaque. Thus when foreign body aspiration is suspected, expiratory or lateral decubitus chest radiographs may identify air trapping on the affected, dependent side.
- If history or exam is suggestive of foreign body aspiration, the patient should undergo rigid bronchoscopy.
- Flexible bronchoscopy can be used to locate an aspirated foreign body and may be useful when the presentation is not straightforward, but foreign body removal is best performed via rigid bronchoscopy.


THANK YOU