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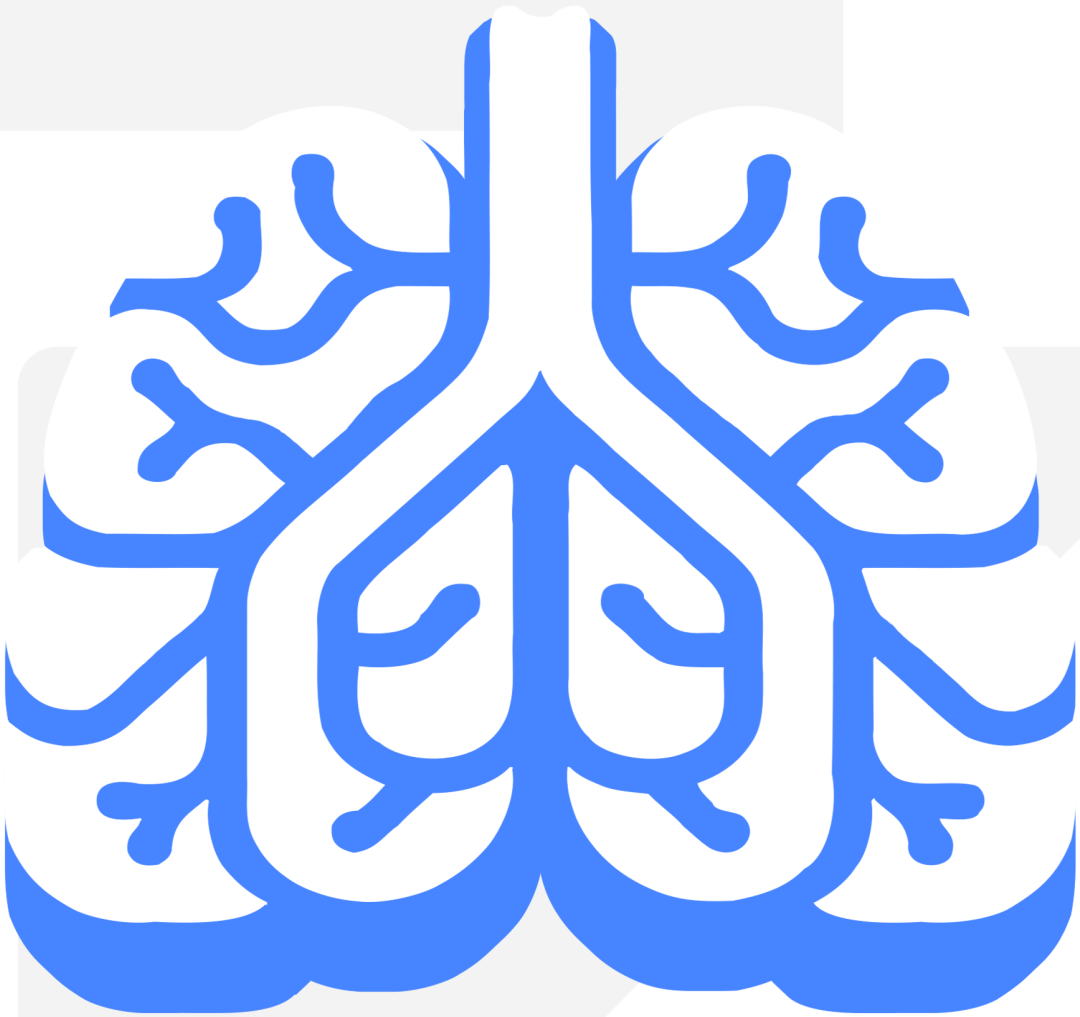
Pediatrics

PULMONOLOGY



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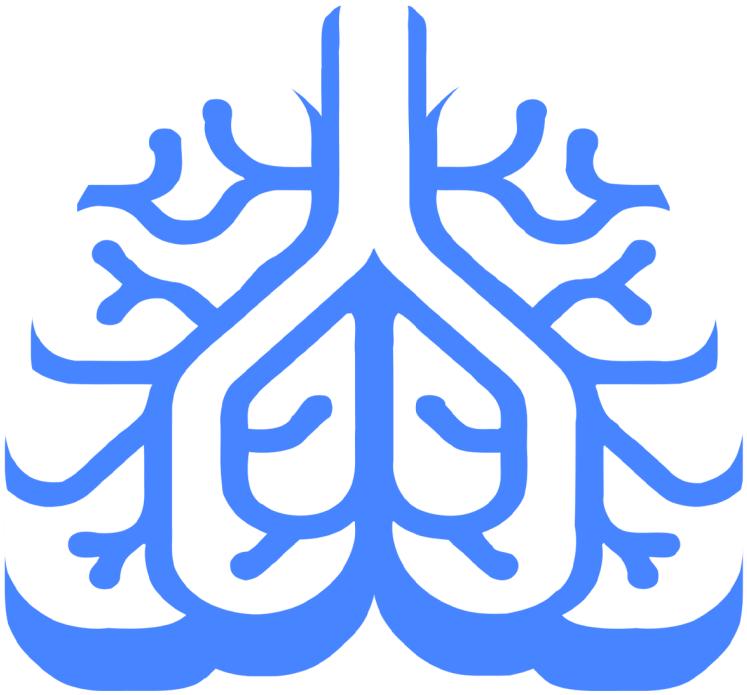

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TABLE OF CONTENTS

1 Lecture 1
Introduction To Cough in Pediatrics

3 Lecture 2
Acute Cough In Pediatrics

15 Lecture 3
Recurrent Cough & Wheezing

28 Lecture 4
Chronic Cough In Pediatrics

46 Lecture 5
Respiratory Distress & Emergencies

53

Lecture 6

Pleural Diseases

TBD

Questions

"Two men looked out from prison bars, one saw the mud, the other saw stars."

Which one will you be?

INTRODUCTION TO COUGH IN PEDIATRICS

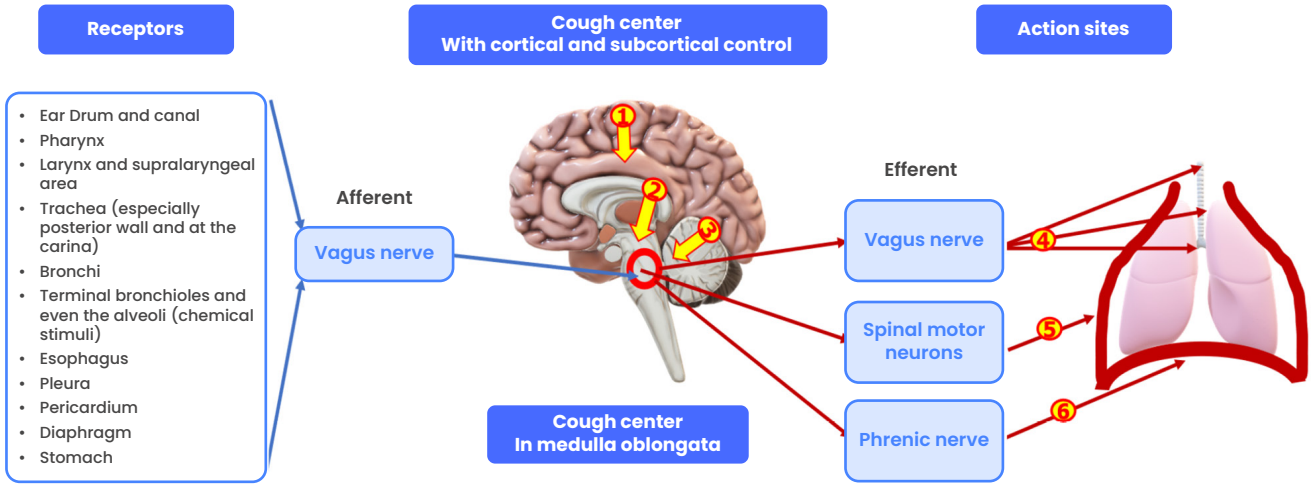
Cough is an important defense mechanism of the lungs and is a common symptom, particularly during winter months.

In most patients, it is **self-limited.** However, cough can be ominous, indicating **serious underlying disease**, because of **accompanying problems** (respiratory distress, cyanosis, hemoptysis, or loss of consciousness).

The **cough reflex** serves to prevent the entry of harmful substances into the tracheobronchial tree and to expel excess secretions and retained material from the tracheobronchial tree.

The causes of acute, recurrent, and chronic coughs may be quite different from each other.

A cough can be: paroxysmal, brassy, productive, weak, volitional, and "throat-clearing," and it may occur at different times of the day.



Associated Symptoms

A history of accompanying signs or symptoms can give important clues, whether:

- **Localized** to the respiratory tract: e.g. wheeze, stridor.
- **Elsewhere:** e.g. failure to thrive, frequent malodorous stools.

Note

If a parent says that a child "wheezes" or "croups" or is "short of breath," it is important to find out what they mean by that term and to ask them to mimic the sound or action.

Classification of cough

Acute	Cough lasting less than 3 weeks duration
Subacute / Prolonged acute	Cough lasting more than 3 weeks, but less than 8 weeks duration
Chronic	Cough lasting more than 8 weeks duration
Recurrent	Cough repeated more than two attacks per year, apart from those associated with common colds, that each last more than 7 – 14 days

In short:

Acute	Subacute / Prolonged acute	Chronic
< 3 weeks	3 -8 weeks	> 8 weeks

Recurrent

≥ 2 / year

Each > 7 - 14 days.

2 ACUTE COUGH IN PEDIATRICS

The common causes of acute cough:

Respiratory infections

Acute, fever

Upper respiratory

Common cold
Acute sinusitis
Croup

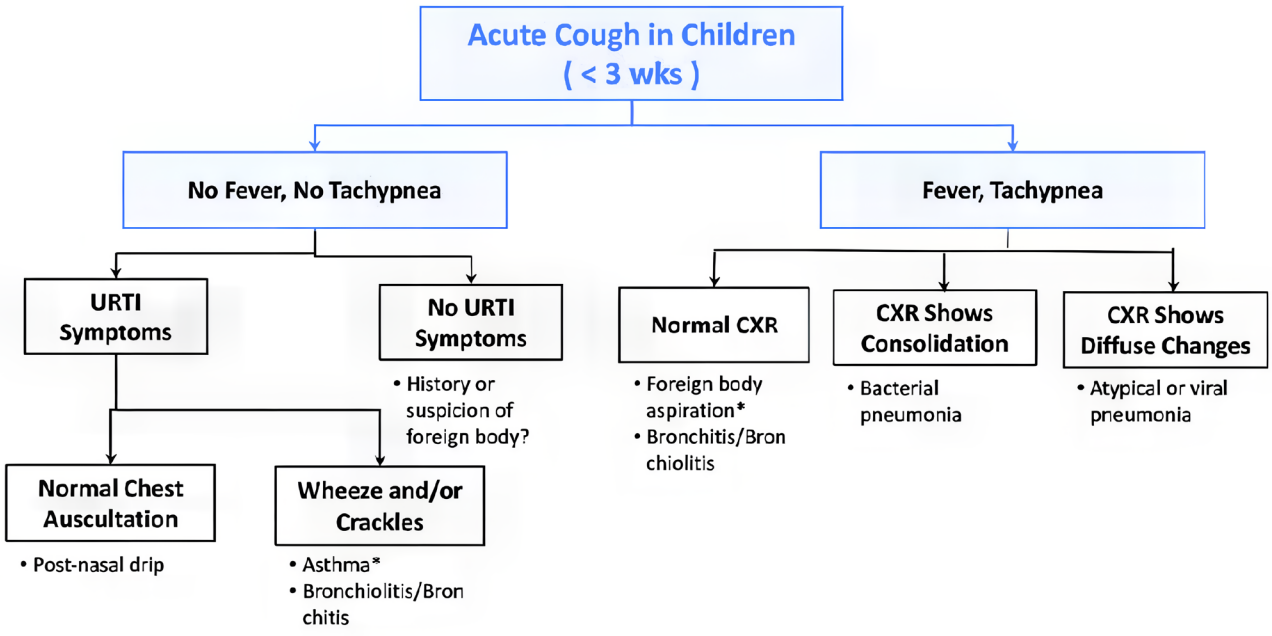
Lower respiratory

Acute bronchiolitis
Pneumonia

FB aspiration

Sudden, no fever

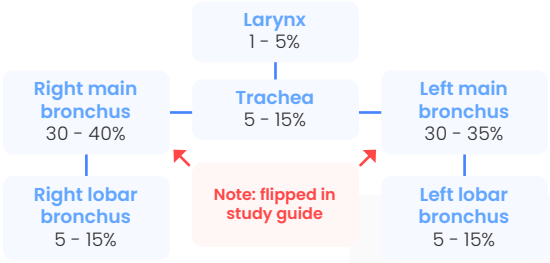
1st presentation of recurrent or chronic cough



Foreign body aspiration

Common between 6 months and 3 years

Location of impaction in respiratory tract:



Presentation

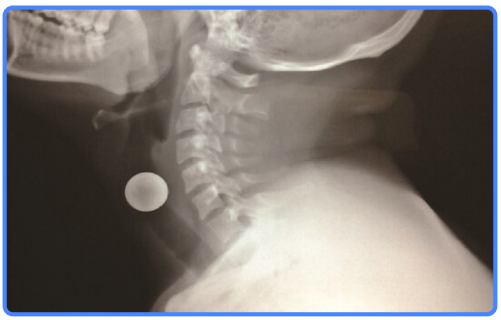
General clinical picture

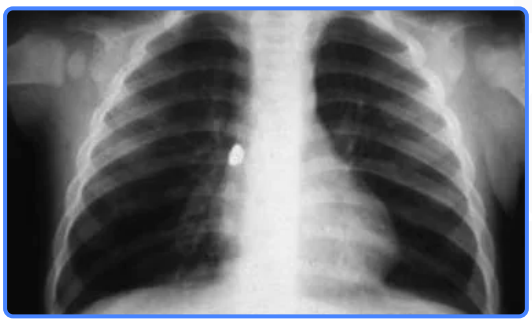
Symptoms: Sudden onset of: cough, wheezing, stridor, or choking (while eating or playing)

Signs: Unilateral signs: (wheezes or crepitations/bronchial breathing/decreased air entry or hyperresonance)

Specific presentations

Laryngeal FB	Bronchial FB
Healthy playing or eating child with sudden onset of: • Choking, difficult breathing, aphonia • Hoarseness of voice • Cough and gaging • Cyanosis, apnea and loss of consciousness **Usually goes as following:** Healthy playing → Choking → Sudden cough → Stridor or dysphonia → Sudden aphonia → Apnea → Cyanosis → Arrest.	***Healthy playing or eating child with sudden onset of:*** • Choking • Cough attack • Difficult breathing ***On examination:*** • Diminished breath sounds • Localized wheeze. ***Chest x-ray:*** • **Unilateral changes:** Hyper-expansion or atelectasis of affected lobe or segment. • Radio-opaque foreign body (< 20%).





Management

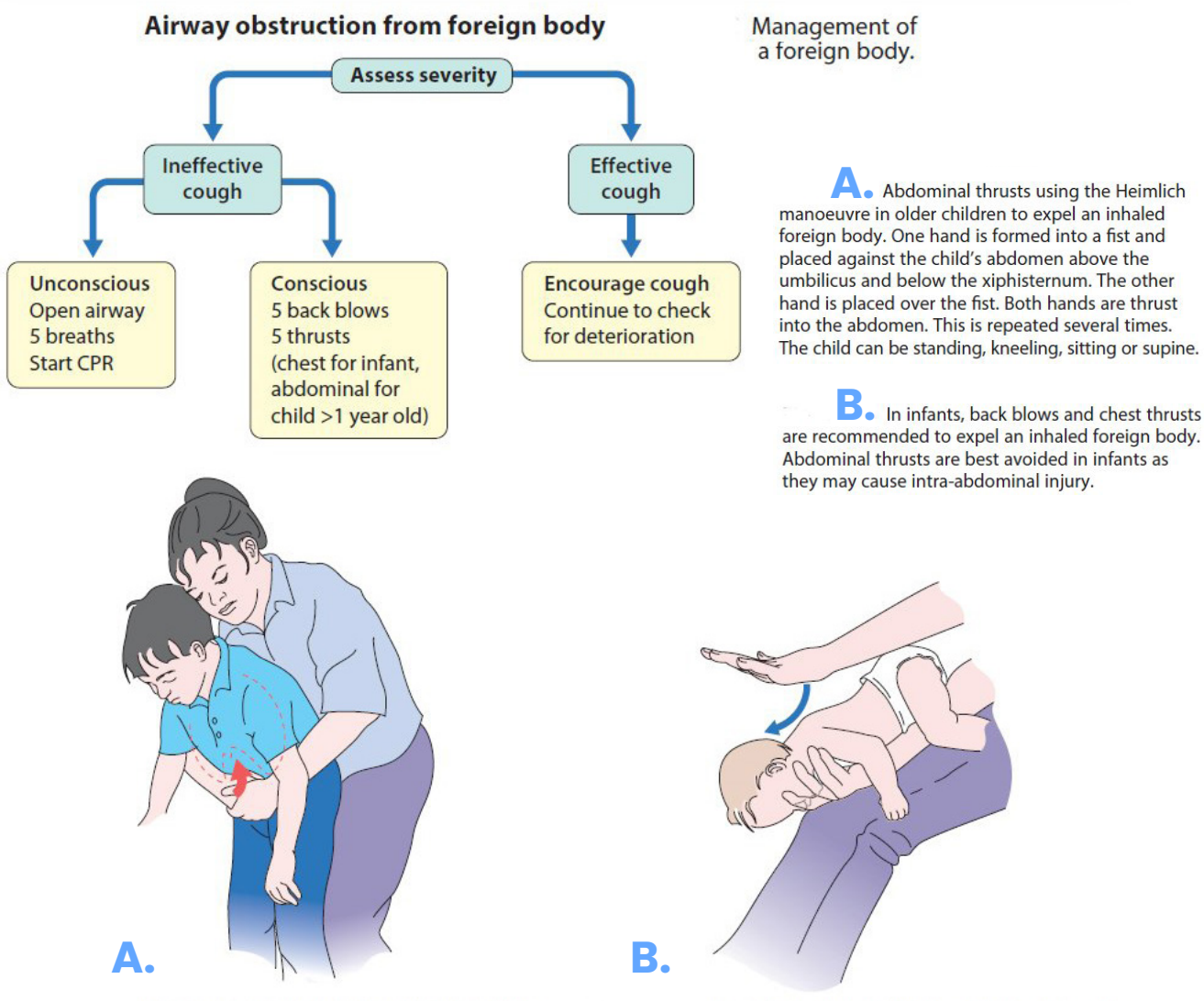
Laryngeal FB (emergency)

Child with effective cough: watch – encourage cough – be ready to interfere.

Child with ineffective cough but still conscious:

- **Infant:** 5 back blows and chest thrust.
- **Child:** abdominal thrust.

Child losing consciousness or apneic: cardiopulmonary resuscitation (CPR)



Bronchial FB

- Urgent removal with **bronchoscope.**

Acute respiratory tract infections (ARTI)

ARTIs are considered as leading cause of mortality and morbidity among children in many developing countries.

URTIs (95%): Less morbidity and mortality

LRTIs (5%): Significant morbidity and mortality

Key symptoms

URTIs

Nasal symptoms: obstruction, difficult breathing, mouth breathing, discharge (anterior and posterior), sneezing, noisy inspiration (snoring)

Pharyngeal symptoms: sore throat, odynophagia, dry irritative husky cough

Laryngeal symptoms: hoarse voice, dysphonia, painful phonation, croupy barking cough, noisy inspiration (stridor), respiratory distress in severe cases

Tracheal symptoms: retrosternal pain, stridor cough usually wet

Ear symptoms: ear pain, sense of fullness, diminished hearing

LRTIs

- **Respiratory distress**
- **Wheezes**
- **Cough:** can be dry or wet or spasmodic or bloody **(hemoptysis)**
- **Chest pain**
- **Compression symptoms**

Rhinitis and Nasopharyngitis (common cold) (coryza)

Common cold is the commonest infection in pediatrics

- Children may have 6 -7 colds / year
- 10–15% of children have at least 12 infections / year.

Etiology:

- **Rhinoviruses** cause 30-50% of colds.
- **Other viruses:** Coronaviruses, Adenoviruses.

Clinical presentation

The usual cold persists about 1 week, although in 10% it lasts 2 weeks.

Coryza Triad

- Red watery eye
- Rhinorrhea /Nasal blockage
- Sore throat

Local examination

- Erythema
- Edema of the nasal mucosa
- Secretions
- Diffusely congested red throat if nasopharyngitis

Others

- Nasal obstruction
- Snoring
- Mouth breathing
- Tachypnea
- Sneezing & itchy nose
- Ear pain and sense of ear fullness (ET dysfunction)
- Dry irritative cough (postnasal drip)
- Congested throat
- **in infants:** Difficult breathing, difficult suckling, vomiting, diarrhea and irritability.

Note

Fever and systemic toxemia are **uncommon** in contrast to influenza.

Complications

- AOM
- Sinusitis
- Asthma exacerbation
- LRTI

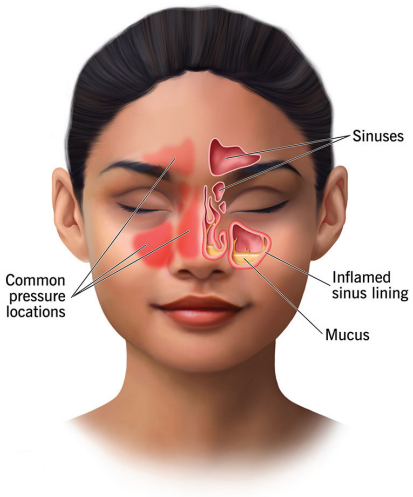
Treatment

Common cold is self-limited & TTT should be symptomatic.

- Normal saline nose drops, nasal wash and nasal suctioning
- Mist inhalation
- Smoke and allergen free area
- Plenty of warm fluids (herbal)
- Consider decongestant nasal drops
- Acetaminophen
- Antibiotics are **not indicated**

Acute sinusitis

Inflammation of 1+ paranasal sinuses with obstruction of the normal drainage mechanism (acute i.e. < 3 weeks)



Predisposing factors: Common colds and nasal allergy.

Etiology:

- **Viruses:** Rhinovirus, coronavirus, others
- **Bacteria:** S. pneumoniae, H. influenzae, M. catarrhalis.

Clinical picture

- Nasal block
- Discharge
- Headache
- Facial edema
- Tender sinus
- Postnasal discharge
- Pharyngitis
- Cough
- Fever

Complications

- Acute Otitis Media (AOM)
- Pneumonia
- Intracranial infection
- Orbital cellulitis
- Cavernous sinus thrombosis

Red flags

- Toxemia
- Visual disturbance
- Frontal sinusitis
- CNS symptoms

Treatment

Self-limited in most cases

- Normal saline nose drops, nasal wash and nasal suctioning
- Mist inhalation
- Smoke and allergen free area
- Warm plenty fluids (herbal)
- Mucolytics
- Consider decongestant nasal drops
- Acetaminophen
- Nasal corticosteroid
- Antibiotic for bacterial infection
- Parenteral antibiotic in frontal sinusitis
- Treat complications

Acute Laryngo-tracheo-bronchits (croup)

Stridor is a continuous musical sound usually heard on inspiration and is caused by narrowing in the extrathoracic airway, as with croup or laryngomalacia.

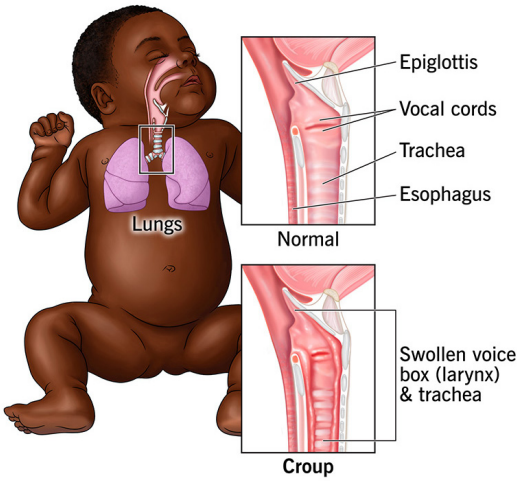
Croup is the commonest form of stridor; It is inflammation of larynx and subglottic area.

Etiology:

- Males > females
- Common in **early childhood:** 1 or 2 - 5 years old
- Viral; parainfluenza and influenza

Clinical picture:

- Starts as coryza, then pharyngitis, then upper airway obstruction (inspiratory stridor, barking cough, and hoarse voice); ***Descending march.***
- Fever and toxemia are not prominent
- Hypoxia and cyanosis in severe cases



Diagnosis: is clinical. Only investigation needed is to **measure oxygen saturation.**

DD of stridor in infancy

Acute Stridor	Chronic Stridor
• Foreign body inhalation • Epiglottitis • Laryngotracheobronchitis (Croup) • Laryngitis	• Laryngomalacia • Subglottic stenosis • Vocal cord paralysis • Subglottic haemangioma

Red flags

- Respiratory distress (RD)
- Irritability
- Hypoxia
- Disturbed level of consciousness

(More in chapter: "Respiratory Distress")

Treatment

Mild cases with no RD:

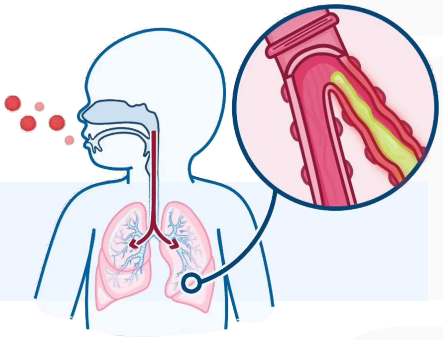
- Home treatment
- Supportive
- **Nebulized adrenaline** (1 : 1000) 0.5mls/kg
- **Dexamethasone:** 0.6 mg/kg single dose or **oral prednisone:** 1mg/kg

Moderate and severe cases:

Hospitalized & managed as emergency:

- Avoid anxiety provoking measures
- Secure airway with help of ENT & ICU team
- Support breathing and oxygenation as needed
- Assisted ventilation if needed
- Circulatory support
- Nebulized adrenaline
- Systemic dexamethasone

Acute bronchiolitis



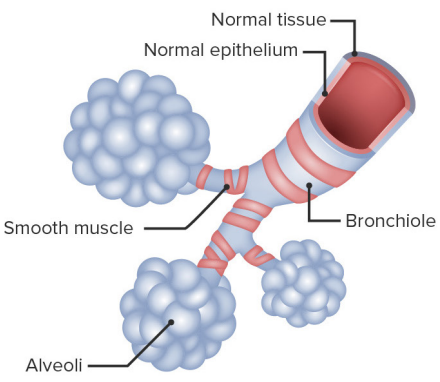
Bronchiolitis is an infection of the lower respiratory tract (small respiratory bronchioles and alveolar sacs)

Etiology:

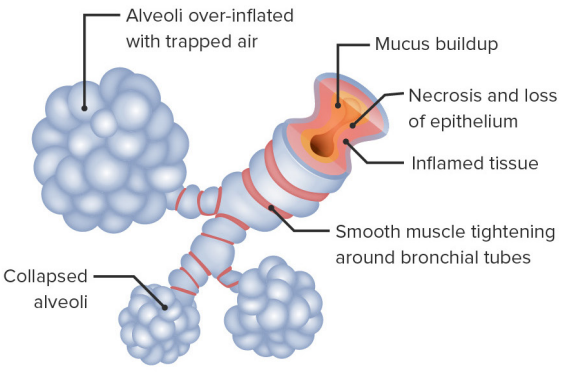
- Vulnerable age ***< 2 years***, most severe at age 1 – 2 months in winter months.
- **Respiratory syncytial virus (RSV)** is the commonest cause. Others like, Parainfluenza virus, Adenovirus, Influenza, Human metapneumovirus.

Pathology:

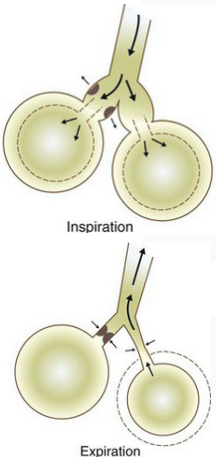
- Cytotoxic injury to bronchial mucosa & excess mucus cause **plugging of the bronchioles** and the small airways;
  - **If obstruction is incomplete:** Ball and valve mechanism and so air-trapping and over-inflation.
  - **If obstruction is complete:** No air goes into alveoli and alveoli will collapse. Defective gas exchange causing severe hypoxaemia.



Normal bronchial tubes



Bronchial tube during bronchiolitis



Clinical picture

Chest

- Coryza
- Paroxysmal cough
- Respiratory distress
- Tachypenia
- Dyspenia
- Wheezes
- Crackles
- Diminished air entry
- Apnea may occur
- Cyanosis and hypoxaemia

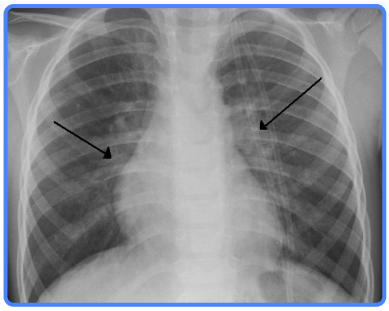
General

- Fever
- Irritability
- Refusal of feeding

Red flags

- Young age
- Abnormal consciousness
- Difficult feeding
- Dehydration
- Severe respiratory distress/Apnea
- Desaturation
- Abnormal blood gases
- Abnormal blood tests

Investigations



Chest x-ray: Hyperinflation with patchy atelectasis

PCR on nasopharyngeal swab

Blood tests/blood gases

Treatment

Most cases are mild and can be treated at home:

- Self-limiting
- Supportive.
- **Antibiotics are ineffective** in the treatment of bronchiolitis.

Moderate and severe cases:

- Hospitalize.
- Oxygen therapy and a ventilator
- Intravenous (iv) fluids
- Frequent suctioning of nose and mouth
- Nasogastric feeding
- Hypertonic saline nebulization
- Bronchodilators are not routinely used
- Steroids not routinely used
- Ribavirin only for severe cases

Pneumonia in children

Pneumonia is an infection and inflammation of one or both lung parenchyma

Classification

According to causative organism:

- **Viral (commonest):** RSV, influenza, parainfluenza, rhinovirus, others
- **Bacterial:** S. pneumoniae, H. influenza, S. aureus, Moraxella cattarhalis
- **Atypical:** M. pneumoniae, C. pneumoniae

According to age:

Neonate	Group B streptococci, E. coli, listeria
1 - 3 months	Viral: RSV, influenza, parainfluenza, human metapneumovirus, Chlamydia (afebrile pneumonia), Bordetella pertussis
4 months - 4 years	Viral: RSV, influenza, parainfluenza, Bacterial: Strept. pneumoniae, H. influenza, Moraxella catarrhalis
≥ 5 years	Atypical: Mycoplasma (walking pneumonia), chlamydia trachomatis

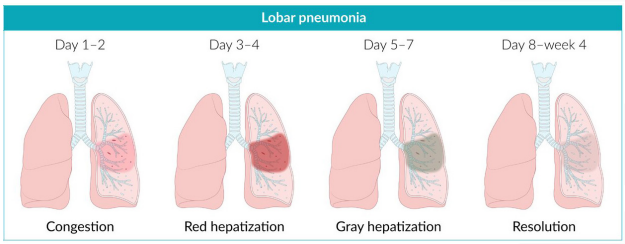
According to anatomy:

- **Lobar pneumonia:** consolidation affect whole lobe (**S. pneumoniae** is the commonest cause)
- **Bronchopneumonia:** bilateral diffuse lesions
- **Interstitial pneumonia:** alveolo interstitial inflammation (**viral and atypical** bacteria).

Pathology

Stages of lobar pneumonia:

1. Stage of **congestion**
2. Stage of **red hepatization (consolidation)**
3. Stage of **grey hepatization (consolidation)**
4. Stage of **resolution**



Clinical picture

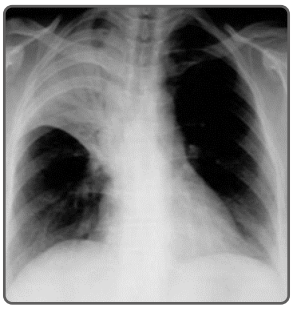
Symptoms	Examination
Coughing: greenish/yellow or rusty sputum **Acute toxemia:** Fever, sweating, chills, headache, anorexia, malaise **Sharp or stabbing chest pain.**	**Respiratory distress:** Tachypnea, Working alae nasi, Supra-sternal retraction, Intercostal muscle retraction, Grunting, Cyanosis. **Inspection:** Unilateral diminished chest movement. **Palpation:** Increased TVF, central trachea. **Ausculation:** Bronchial breathing, diminished air entry.

Investigations

- **Blood test:** CBC, CRP, ESR, ABG
- **Microbiology:** Culture, antigen detection, PCR
- **Radiology:** CXR, CT chest

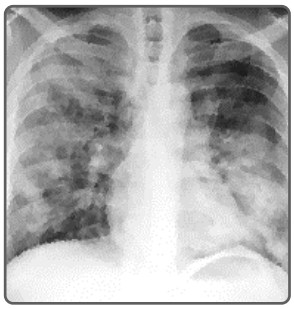
Lobar pneumonia

Consolidation of whole lobe; Right upper lobe pneumonia



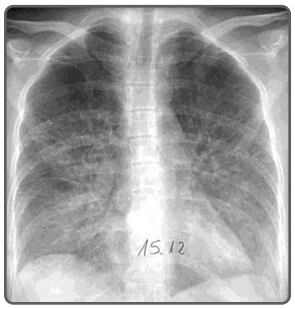
Broncho-pneumonia

Bilateral diffuse patchy consolidation



Interstitial pneumonia

Bilateral diffuse infiltrates



Treatment

- **Mild cases:** can be treated at home with oral antibiotics.
- **Moderate and severe cases:**
  - Hospitalize.
  - Oxygen therapy and a ventilator
  - Intravenous (IV) fluids
  - Frequent suctioning of nose & mouth
  - Nasogastric feeding
  - Antibiotics
  - Bronchodilators
  - Treatment of complications

Complications

Local spread

- Abscess
- Pleural effusion
- Empyema
- Empyaema necesstanns
- Rib osteomyelitis
- Supp pericarditis

Toxic

- Meningism
- Carditis
- Arthritis
- Ileus

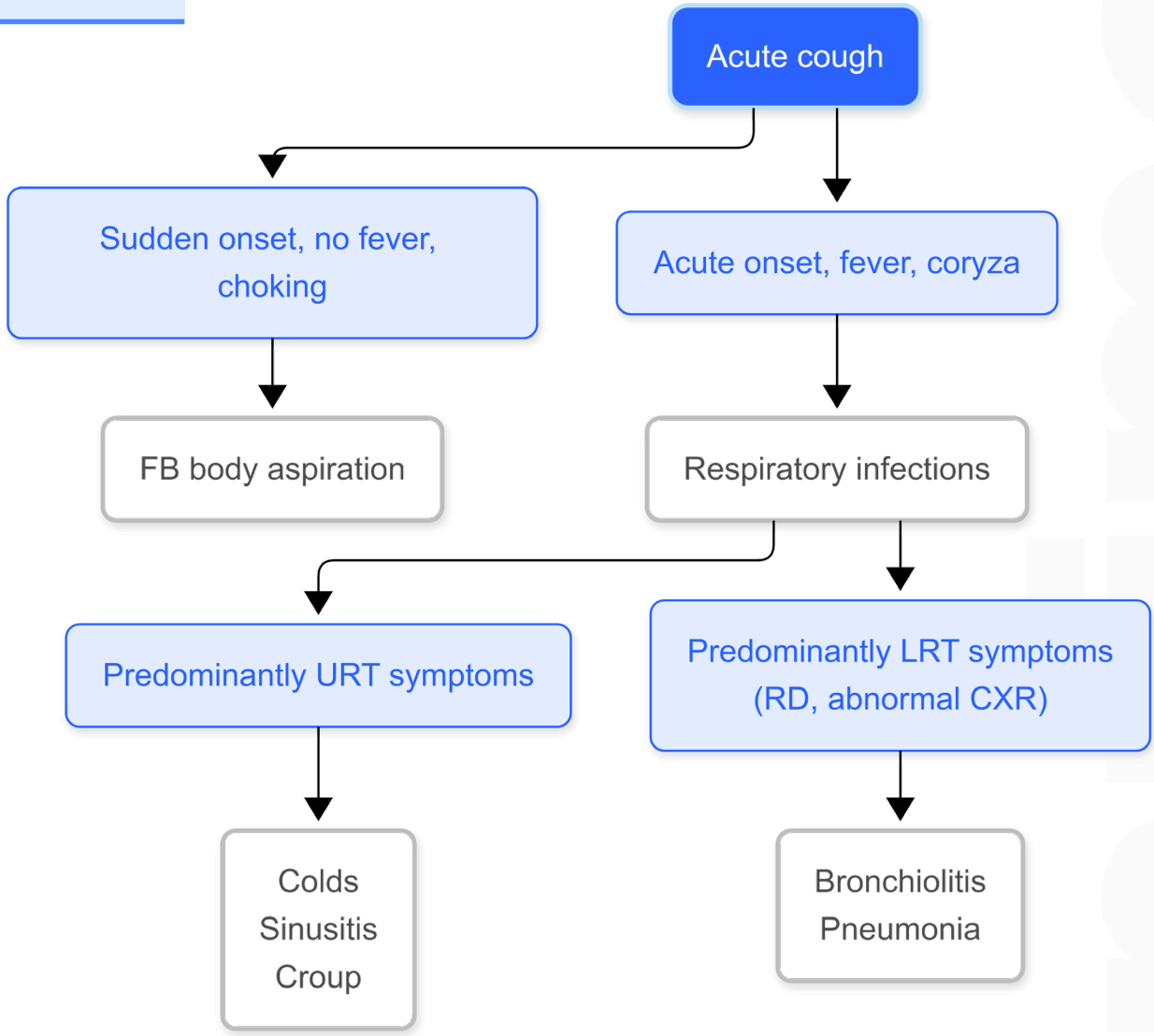
Distant spread

- Brain abscess
- Meningitis
- Arthritis
- Osteomyelitis
- Septic emboli
- Septic shock

Others

- Dehydration
- Malnutrition
- Electrolye disturbance
- Renal impairment
- Skin rash
- DIC

Algorithm



RECURRENT COUGH & WHEEZING

A wheeze is a high-pitched, musical, adventitious lung sound produced by airflow through an abnormally narrowed or compressed airway(s).

A wheeze is synonymous with a **high-pitched or sibilant rhonchus.**

- Wheezing in early life is a common disorder, with approximately **50% of children having an episode of wheezing in the first year of life.**
- A recurrent wheeze is estimated to occur in **one third of children of preschool age** and can cause significant morbidity, decrease quality of life, and increase the frequency of the use of health care services and economic costs.
- Data has confirmed that wheezing is **clinically heterogeneous** in early life in terms of its **temporal pattern** (i.e. age of onset and duration until symptoms disappear) and its **risk factors**, which include atopy and genetic or environmental factors, and the **outcomes** are different for such phenotypes.

Causes of recurrent wheezing in children

Disease Prevalence	Neonate/Infant	School Age/Adolescent
Common	• Bronchiolitis • Asthma	• Asthma

Less Common	• Pulmonary aspiration:
	  • Gastroesophageal reflux
	  • Swallowing dysfunction
	• Foreign body aspiration
	• Bronchopulmonary dysplasia
	• Cystic fibrosis
		• Foreign body aspiration
		• Anaphylaxis
		• Cystic fibrosis

Uncommon	• Congenital heart disease
	• Defective host defenses:
	  • Immune deficiency
	  • Immotile cilia syndrome
	• Congenital structural anomalies:
	  • Tracheobronchomalacia
	  • Vascular ring
	  • Lobar emphysema
	  • Cystic abnormalities
	  • Tracheoesophageal fistula
		• Defective host defenses
		• Mediastinal tumors
		• Enlarged mediastinal lymph nodes
		• Parasitic infection
		• Pulmonary hemosiderosis
		• a1-antitrypsin deficiency

Bronchial Asthma

Asthma is the most common chronic disease of childhood and the leading cause of childhood morbidity from chronic disease as measured by school absences, emergency department visits, and hospitalizations.

- It is a chronic inflammatory disorder which is characterized by airway hyperresponsiveness that leads to recurrent episodes of wheezing, breathlessness, chest tightness, and coughing (particularly at night or early morning), and is associated with widespread, variable, and often reversible airflow limitation.
- Clinical symptoms in children 5 years and younger are variable and non specific.

Prevalence of Asthma

Worldwide:

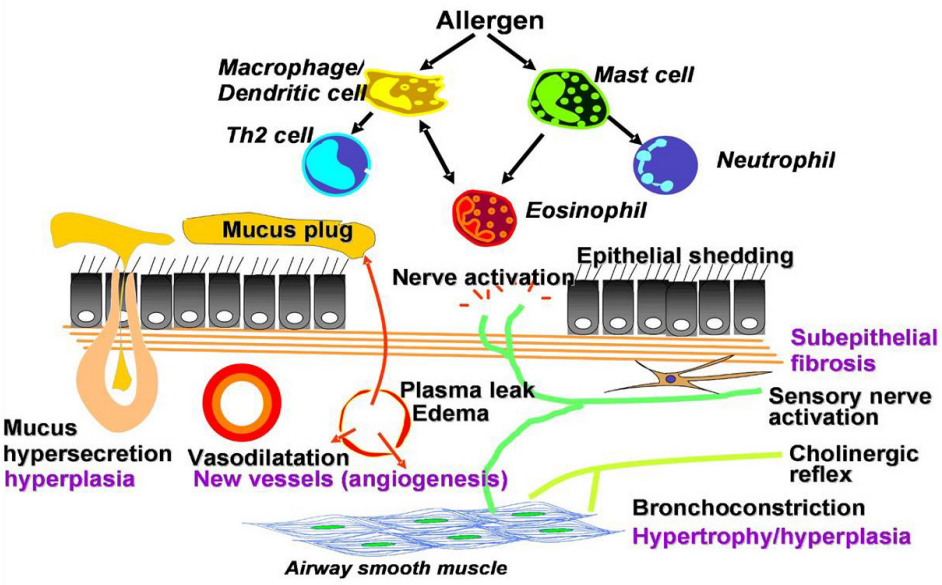
- It has been estimated that more than 300 million persons have asthma.
- The prevalence of asthma is increasing all over the world both in developed and developing countries.
- The prevalence of asthma has been doubled during the last 2-3 decades.

In Egypt:

- The prevalence of pediatric asthma in 1994 was 8.2%.
- Prevalence of asthma in 2008 was 15.3% (Ranging from 10.9 - 18.7%).
- Asthma is more common in urban than rural areas.

Pathogenesis of asthma

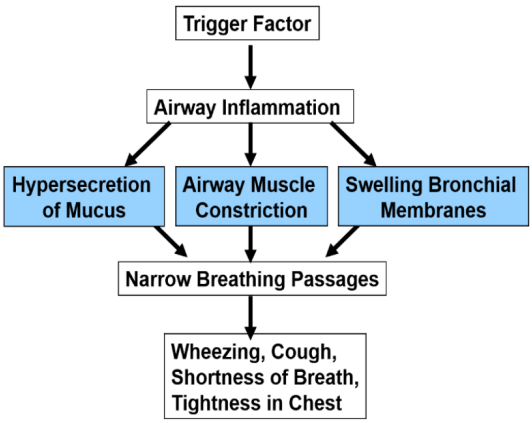
Many cells and cellular elements play a role in chronic inflammation of asthma:



Factors influencing the development and expression of asthma

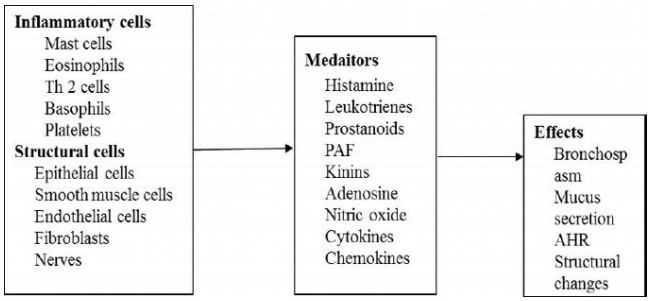
1. Host factors

- **Genetic:**
  - Genes predisposing to atopy
  - Genes predisposing to airway hyper-responsiveness
- **Obesity**
- **Sex**



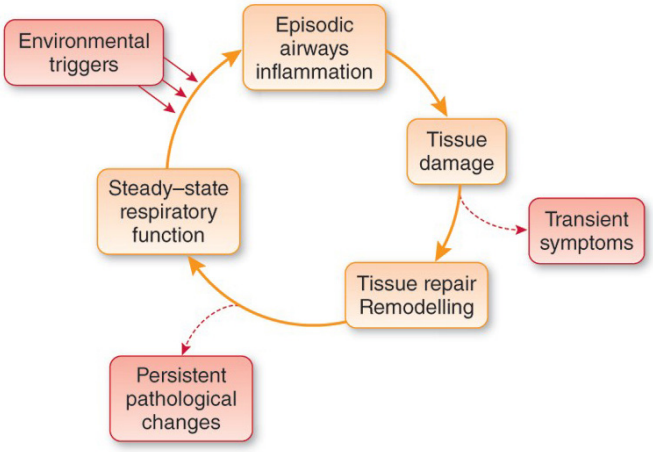
2. Environmental factors

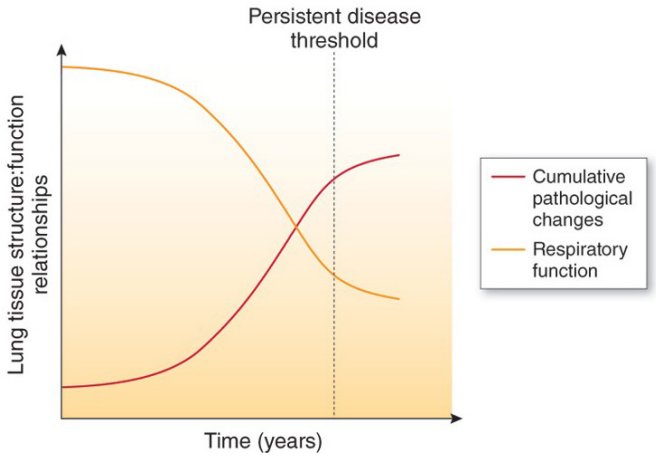
- **Allergens:**
  - **Indoor:** Domestic mites, furred animals (dogs, cats, mice), cockroach allergens, fungi, molds, yeasts.
  - **Outdoor:** Pollens, fungi, molds, yeasts.
- **Infections** (predominantly viral)
- **Occupational sensitizers**
- **Tobacco smoke**
  - Passive smoking
  - Active smoking
- **Indoor/Outdoor air pollution**
- **Diet**



Risk factors of Asthma in younger children

- **Sensitization** to allergen.
- **Maternal diet** during pregnancy and/or lactation.
- **Pollutants** (particularly environmental tobacco smoke).
- **Microbes** and their products.
- **Respiratory (viral) infections**.
- **Psychosocial** factors.





Clinical Features

77 % of asthma begins in children less than 5 years, the most common symptoms are:

- **Recurrent wheezes/Recurrent cough: Activity induced, Nocturnal.**
- **Recurrent breathlessness**
- **Tightness of chest**

Typical features of Asthma:

- Afebrile episodes
- Personal atopy
- Family history of atopy or asthma
- Exercise /Activity induced symptoms
- History of triggers
- Seasonal exacerbations
- Relief with bronchodilators

When does asthma usually begin?

- By 1 year: 26%
- 1 - 5 years: 51.4%
- > 5 years: 22.3%

i.e. 77% of asthma begins in children less than 5 years old.

Asthma predictive index

- It is a simple clinical index used in young children based on presence of a wheeze before the age of 3.
- Presence of **one major risk factor** (parental history of asthma or eczema) or two of **three minor risk factors** (eosinophilia, wheezing without colds, and allergic rhinitis)
- It has been shown to predict the presence of asthma in later childhood

Tools/steps to diagnose asthma

1. **Good history taking**

2. **Careful physical examination**

3. **Investigations** (Above 5 years only)

Remember: Asthma is a **Nocturnal, Familial, Exertional, Paroxysmal, Triggered** disease.

Asthma is confirmed if:

History

The patient has 3 or more episodes of airflow obstruction with several of the following:

- Afebrile episodes
- Personal atopy or family history atopy / asthma
- Nocturnal exacerbations
- Exercise/activity induced Symptoms
- Trigger induced symptoms
- Seasonal exacerbations
- Relief with bronchodilators ± oral steroids

Physical Examination

General attitude and well-being: Difficulty in feeding, talking, getting to sleep.

Deformity of the chest

Character of breathing: Harsh vesicular breathing, prolonged expiration

Breath sounds: Expiratory wheeze

Signs of any other allergic disorders on the body or comorbidities: Eczema, Allergic Rhinitis

Growth and development status

Specific features to look for:

Dyspnea

- Expiratory wheeze
- Accessory muscle movement
- Difficulty in feeding, talking, getting to sleep
- Irritability

Cough

- Persistent / recurrent / nocturnal / exercise-induced

Associated conditions

- Eczema
- Allergic Rhinitis

Weight/height

Co-morbid conditions:

1. **Allergic Rhinitis:**
   - Colds, ear infections
   - Sneezing in the morning
   - Blocked nose, snoring, mouth breathing
2. **Gastro esophageal reflux (GER):**
   - Nocturnal cough followed by vomiting
   - Eczema

Investigations

Peak expiratory flow rate (PEFR): It is **highly suggestive of asthma when:**

- **> 15% increase** in PEFR after inhaled short acting B2 agonist
- **> 15% decrease** in PEFR after exercise
- Diurnal variation > 10% in children not on bronchodilator

Spirometry: for diagnosis, assess severity, identify response to treatment and degree of control.

CBC looking for **eosinophilia**: more than 400 cells/ mm3

Total and specific IgE

Chest radiography only if **complications or other alternative diagnosis** are suspected

Arterial blood gases and pH during asthma exacerbations

Allergic skin testing

8. Biomarkers for asthma activity e.g. FeNO

Differential diagnosis of wheezes

Age	Common	Uncommon	Rare
< 6 months	• Bronchiolitis • Gastro esophageal reflux	• Aspiration pneumonia • Bronchopulmonary dysplasia • Congestive heart failure • Cystic fibrosis	• Asthma • Foreign body aspiration
6 months - 5 years	• Bronchiolitis • Foreign body aspiration	• Aspiration pneumonia • Asthma • Bronchopulmonary dysplasia • Cystic fibrosis • GER	• Congestive heart failure
2 - 5 years	• Asthma • Foreign body aspiration	• Cystic fibrosis • GER • Viral pneumonia	• Aspiration pneumonia • Bronchiolitis • Congestive heart failure • GER

Management

Goals of asthma management:

1. Achieve and maintain control of symptoms.
2. Maintain normal activity levels, including exercise.
3. Maintain pulmonary function as close to normal levels as possible.
4. Prevent asthma exacerbations.
5. Avoid adverse effects from asthma medications
6. Prevent asthma mortality.

Five components of therapy to achieve and maintain control:

I. Develop Patient/Doctor partnership.
II. Identify and reduce exposure to risk factors.
III. Assess, treat, and monitor asthma according to severity.
IV. Manage asthma exacerbations.
V. Written asthma action plan.

1. Develop Patient/Doctor partnership

Effective management of asthma requires the development of a partnership between the person with asthma and the health care team

Patients can learn to:

1. Avoid risk factors
2. Take medications correctly
3. Understand the difference between controller and reliever medications
4. Monitor their status using symptoms and, if relevant, PEF
5. Recognize signs that asthma is worsening and take action
6. Seek medical help as appropriate

2. Identify and reduce exposure to risk factors

- Measures to prevent the development of asthma and asthma exacerbations by avoiding or reducing exposure to risk factors should be implemented wherever possible.
- Reducing patients exposure to some categories of risk factors improves the control of asthma and reduces medication needs.

3. Assess, Treat and Monitor Asthma

- The goal of asthma treatment can be reached in most patients through a continuous cycle that involves assessing, treating and monitoring asthma
- Each patient should be assessed to establish his/her current treatment regimen, adherence to the current regimen, and level of asthma control
- Each patient is assigned to one of five treatment steps
- At each treatment step, reliever medication should be provided for quick relief of symptoms as needed.

Administration:

Treatment can be administered in different ways: inhaled, oral, or by injection.

- **Advantage of inhaled therapy:** drugs are delivered directly into the airways, producing higher local concentrations with significantly less risk of systemic side effects.

For delivery:

- Inhaled medications for asthma are available as pressurized MDIs, DPIst soft mist inhalers and nebulized or 'wet' aerosols.
- **A pMDI with a valved holding chamber is the preferred delivery system.**
- CFC inhaler devices are being phased out due to the impact of CFCs upon the atmospheric ozone layer, and are 'being replaced by HFA devices.
- For children 0 to 5 years of age, a face mask is recommended over a mouthpiece for children < 4 years of age. A valve-holding chamber allows for the medication to go to the lungs instead of impacting the back of the throat. It also helps with coordination of actuation.
- A nebulizer device with either a face mask or a mouthpiece is an alternative method

Asthma Medications

Classified into controllers and relievers:

- **Controllers:** medications to be taken on daily long-term basis.
- **Relievers:** medications to be used on as-needed basis to relieve symptoms quickly.

1. Controller Medications	2. Reliever Medications
• **Inhaled glucocorticosteroids** • **Leukotriene modifiers** • **Long acting inhaled B2 agonists** • Theophylline • Cromones • Anti-IgE • Systemic glucocorticosteroids	• Rapid acting inhaled B2 agonists • Systemic glucocorticosteroids • Anticholinergics • Theophylline

Asthma Reliever Medications

- Inhaled short-acting beta-2 agonists (SABA) (e.g., albuterol, levalbuterol) are the preferred and most used options for quick relief of asthma symptoms and bronchoconstriction.
- Potential adverse effects include tremors, tachycardia, and palpitations. These adverse effects are seen more often during initial exposure.

Asthma Controller Medications

- Inhaled Corticosteroids (ICS) are the preferred option for the initial management of mild persistent asthma and are a component of treatment plans for moderate and severe persistent asthma.
- Local side-effects may include dysphonia and oropharyngeal candidiasis. Use of a spacer device and having child rinse his or her mouth with water after using an ICS decreases the risk of oral thrush. High-dose corticosteroids are associated with systemic side effects, such as reduced growth velocity.

Combination therapy with an ICS plus long-acting beta-2 agonist (LABA) bronchodilator

- Has been used in older children and adolescents with asthma.
- Evaluated for safety down to age four years. There is very limited data in children less than age four years.

Montelukast, The leukotriene modifier

- Is the only leukotriene modifier indicated for use in this age group and is available in either granules or chewable tablets depending on the age.
- It is an alternative option either alone or in combination with inhaled corticosteroids depending on the level of asthma severity and control.
- Safety and efficacy are not established for asthma in children younger than 12 months.

Systemic corticosteroids

- Tablet, suspension, intramuscular (IM) or intravenous (IV) injection
- Given for short term treatment, also known as burst therapy (usually given for three to five days) are important early in the treatment of **severe acute exacerbations.**

Assessment of asthma severity

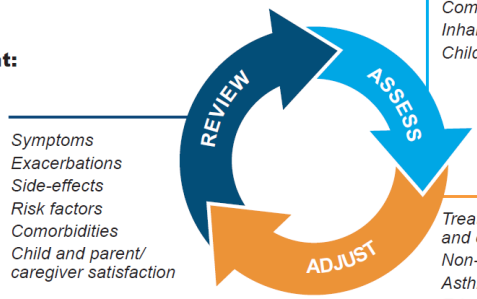
Components of Severity		Classification of Asthma Severity (0–4 years of age)			
			Persistent		
		Intermittent	Mild	Moderate	Severe
Impairment	Symptoms	≤2 days/week	>2 days/week but not daily	Daily	Throughout the day
	Nighttime awakenings	0	1–2x/month	3–4x/month	>1x/week
	Short-acting beta$_2$-agonist use for symptom control (not prevention of EIB)	≤2 days/week	>2 days/week but not daily	Daily	Several times per day
	Interference with normal activity	None	Minor limitation	Some limitation	Extremely limited
Risk	Exacerbations requiring oral systemic corticosteroids	0–1/year	≥2 exacerbations in 6 months requiring oral systemic corticosteroids, or ≥4 wheezing episodes/1 year lasting >1 day AND risk factors for persistent asthma		
		← Consider severity and interval since last exacerbation. Frequency and severity may fluctuate over time. → Exacerbations of any severity may occur in patients in any severity category.			
Recommended Step for Initiating Therapy (See figure 4–1a for treatment steps.)		Step 1	Step 2	Step 3 and consider short course of oral systemic corticosteroids	
		In 2–6 weeks, depending on severity, evaluate level of asthma control that is achieved. If no clear benefit is observed in 4–6 weeks, consider adjusting therapy or alternative diagnoses.			

Components of Severity		Classification of Asthma Severity ≥12 years of age			
			Persistent		
		Intermittent	Mild	Moderate	Severe
Impairment Normal FEV_1/FVC: 8–19 y 85% 20 –39 y 80% 40 –59 y 75% 60 –80 y 70%	Symptoms	≤2 days/week	>2 days/week but not daily	Daily	Throughout the day
	Nighttime awakenings	≤2x/month	3–4x/month	>1x/week but not nightly	Often 7x/week
	Short-acting β_2-agonist use for symptom control (not prevention of EIB)	≤2 days/week	>2 days/week but not daily, and not more than 1x on any day	Daily	Several times per day
	Interference with normal activity	None	Minor limitation	Some limitation	Extremely limited
	Lung function	• Normal FEV_1 between exacerbations • FEV_1 >80% predicted • FEV_1/FVC normal	• FEV_1 >80% predicted • FEV_1/FVC normal	• FEV_1 >60% but <80% predicted • FEV_1/FVC reduced 5%	• FEV_1 <60% predicted • FEV_1/FVC reduced >5%
Risk	Exacerbations requiring oral systemic corticosteroids	0–1/year (see note)	≥2/year (see note) →		
		← Consider severity and interval since last exacerbation. Frequency and severity may fluctuate over time for patients in any severity category. → Relative annual risk of exacerbations may be related to FEV_1.			
Recommended Step for Initiating Treatment (See "Stepwise Approach for Managing Asthma" for treatment steps.)		Step 1	Step 2	Step 3 and consider short course of oral systemic corticosteroids	Step 4 or 5
		In 2–6 weeks, evaluate level of asthma control that is achieved and adjust therapy accordingly.			

Stepping up and down treatment of asthma

GINA 2024 – Children 5 years and younger

Personalized asthma management:
Assess, Adjust, Review response



Exclude alternative diagnoses
Symptom control & modifiable risk factors
Comorbidities
Inhaler technique & adherence
Child and parent/caregiver preferences and goals

Symptoms
Exacerbations
Side-effects
Risk factors
Comorbidities
Child and parent/ caregiver satisfaction

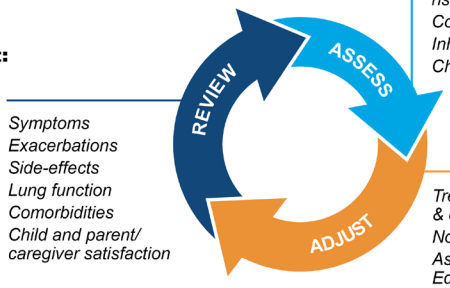
Treat modifiable risk factors and comorbidities
Non-pharmacological strategies
Asthma medications
Education & skills training

Asthma medication options:
Adjust treatment up and down for individual child's needs

	STEP 1	STEP 2	STEP 3	STEP 4
PREFERRED CONTROLLER CHOICE	*(Insufficient evidence for daily controller)*	Daily low dose inhaled corticosteroid (ICS) (see Box 11-3 for ICS dose ranges for pre-school children)	Double 'low dose' ICS *(See Box 11-3)*	Continue controller & refer for specialist assessment
Other controller options (limited indications, or less evidence for efficacy or safety)	*Consider intermittent short course ICS at onset of viral illness*	*Daily leukotriene receptor antagonist (LTRA†), or intermittent short course of ICS at onset of respiratory illness*	*Low dose ICS + LTRA† Consider specialist referral*	*Add LTRA†, or increase ICS frequency, or add intermittent ICS*
RELIEVER	As-needed short-acting beta$_2$-agonist			
CONSIDER THIS STEP FOR CHILDREN WITH:	Infrequent viral wheezing and no or few interval symptoms	Symptom pattern not consistent with asthma but wheezing episodes requiring SABA occur frequently, e.g. ≥3 per year. Give diagnostic trial for 3 months. Consider specialist referral. Symptom pattern consistent with asthma, and asthma symptoms not well-controlled or ≥3 exacerbations per year.	Asthma diagnosis, and asthma not well-controlled on low dose ICS Before stepping up, check for alternative diagnosis, check inhaler skills, review adherence and exposures	Asthma not well-controlled on double ICS

GINA 2024 – Children 6–11 years

Personalized asthma management:
Assess, Adjust, Review



Confirmation of diagnosis if necessary
Symptom control & modifiable risk factors (see Box 2-2)
Comorbidities
Inhaler technique & adherence
Child and parent/caregiver preferences and goals

Symptoms
Exacerbations
Side-effects
Lung function
Comorbidities
Child and parent/ caregiver satisfaction

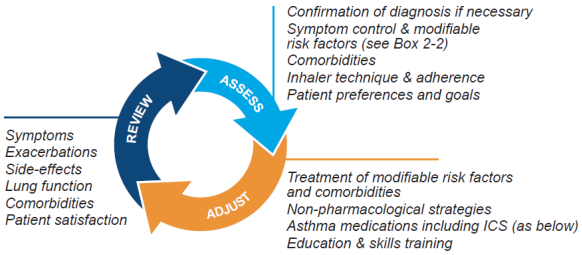
Treatment of modifiable risk factors & comorbidities
Non-pharmacological strategies
Asthma medications including ICS
Education & skills training

Asthma medication options:
Adjust treatment up and down for individual child's needs

	STEP 1	STEP 2	STEP 3	STEP 4	STEP 5
PREFERRED CONTROLLER to prevent exacerbations and control symptoms	Low dose ICS taken whenever SABA taken*	Daily low dose inhaled corticosteroid (ICS) (see table of ICS dose ranges for children)	Low dose ICS-LABA, OR medium dose ICS, OR very low dose ICS-formoterol maintenance and reliever (MART)	Refer for expert advice, OR medium dose ICS-LABA, OR low dose ICS-formoterol maintenance and reliever therapy (MART)	Refer for phenotypic assessment ± higher dose ICS-LABA or add-on therapy, e.g. anti-IgE, anti-IL4Rα, anti-IL5
Other controller options (limited indications, or less evidence for efficacy or safety)		*Daily leukotriene receptor antagonist (LTRA†), or low dose ICS taken whenever SABA taken**	*Low dose ICS + LTRA†*	*Add tiotropium or add LTRA†*	*As last resort, consider add-on low dose OCS, but consider side-effects*
RELIEVER	As-needed SABA (or ICS-formoterol reliever* in MART in Steps 3 and 4)				

GINA 2024 – Adults & adolescents 12+ years

Personalized asthma management
Assess, Adjust, Review
for individual patient needs



TRACK 1: PREFERRED CONTROLLER and RELIEVER
Using ICS-formoterol as the reliever* reduces the risk of exacerbations compared with using a SABA reliever, and is a simpler regimen

STEPS 1 – 2	STEP 3	STEP 4	STEP 5
As-needed-only low dose ICS-formoterol	Low dose maintenance ICS-formoterol	Medium dose maintenance ICS-formoterol	Add-on LAMA Refer for assessment of phenotype. Consider high dose maintenance ICS-formoterol, ± anti-IgE, anti-IL5/5R, anti-IL4Rα, anti-TSLP
RELIEVER: As-needed low-dose ICS-formoterol*			

See GINA severe asthma guide

TRACK 2: Alternative **CONTROLLER and RELIEVER**
Before considering a regimen with SABA reliever, check if the patient is likely to adhere to daily controller treatment

STEP 1	STEP 2	STEP 3	STEP 4	STEP 5
Take ICS whenever SABA taken*	Low dose maintenance ICS	Low dose maintenance ICS-LABA	Medium/high dose maintenance ICS-LABA	Add-on LAMA Refer for assessment of phenotype. Consider high dose maintenance ICS-LABA, ± anti-IgE, anti-IL5/5R, anti-IL4Rα, anti-TSLP
RELIEVER: As-needed ICS-SABA*, or as-needed SABA				

Other controller options (limited indications, or less evidence for efficacy or safety – see text)	*Low dose ICS whenever SABA taken*, or daily LTRA†, or add HDM SLIT*	*Medium dose ICS, or add LTRA†, or add HDM SLIT*	*Add LAMA or add LTRA† or add HDM SLIT, or switch to high dose ICS-only*	*Add azithromycin (adults) or add LTRA†. As last resort consider adding low dose OCS but consider side-effects*

Assessment of Asthma control

Characteristic	Controlled (All of the following)	Partly controlled (Any present in any week)	Uncontrolled
Daytime symptoms	Twice or less per week	More than twice per week	3 or more features of partly controlled asthma present in any week
Limitations of activities	None	Any	
Nocturnal symptoms/ awakening	None	Any	
Need for rescue/ "reliever" treatment	Twice or less per week	More than twice per week	
Lung function (PEF or FEV_1)	Normal	< 80% predicted or personal best (if known) on any day	
Assessment of Future Risk (risk of exacerbations, instability, rapid decline in lung function, side effects)			

4. Management of asthma exacerbations

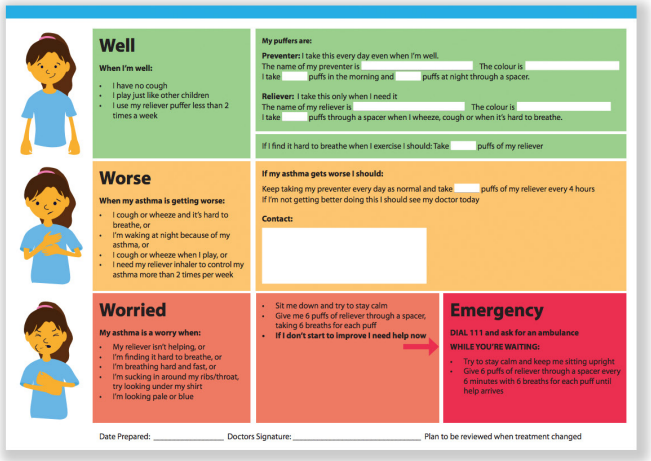
- Exacerbations of asthma are episodes of progressive increase in shortness of breath, cough, wheezing, or chest tightness
- Exacerbations are characterized by decreases in expiratory airflow that can be quantified and monitored by measurement of lung function (FEV 1 or PEF)
- Severe exacerbations are potentially life threatening and treatment requires close supervision

Primary therapies for exacerbations:

- Repetitive administration of rapid acting inhaled B2 agonist
- Early introduction of systemic glucocorticosteroids
- Oxygen supplementation
- Closely monitor response to treatment with serial measures of lung function

5. Asthma Action Plans

- Asthma action plan is a written document in which family/caregiver is provided with up-to-date instructions regarding daily asthma medications, recognition of symptoms that show asthma control deterioration, response when these symptoms are identified, and steps to take in the case of an asthma emergency.



4 CHRONIC COUGH IN PEDIATRICS

Chronic cough in children is defined as a daily cough impairing quality of life (sleep, activity) and lasting for:

- Four or more weeks according to the US
- **Beyond eight weeks,** according to the UK, while acknowledging the existence of a prolonged subacute cough that lasts between four and eight weeks.

Causes

I. Non-specific chronic cough

- Predominantly **dry isolated cough**, with **no signs or symptoms suggestive of disease** in a child (well) and in whom **complementary studies** (at least spirometry, if feasible, and chest X-ray) are **normal.**
- In most cases, it is **secondary to protracted URTI.**
- It is **not serious** and **resolves spontaneously.** Sometimes persistent cough is due to an increase in sensitivity of the cough receptors after a viral infection.
- Factors such as environmental contamination and exposure to tobacco smoke contribute to its persistence.
- Many of these cases are treated incorrectly with inhaled corticosteroids, having been classified as "cough variant asthma".

Upper respiratory tract infections (URTI), bronchial hyperactivity (BHR), asthma, gastroesophageal reflux disease (GERD) and angiotensin converter enzyme inhibitor therapy, among others, increase the sensitivity of the cough receptors.

II. Specific Chronic cough:

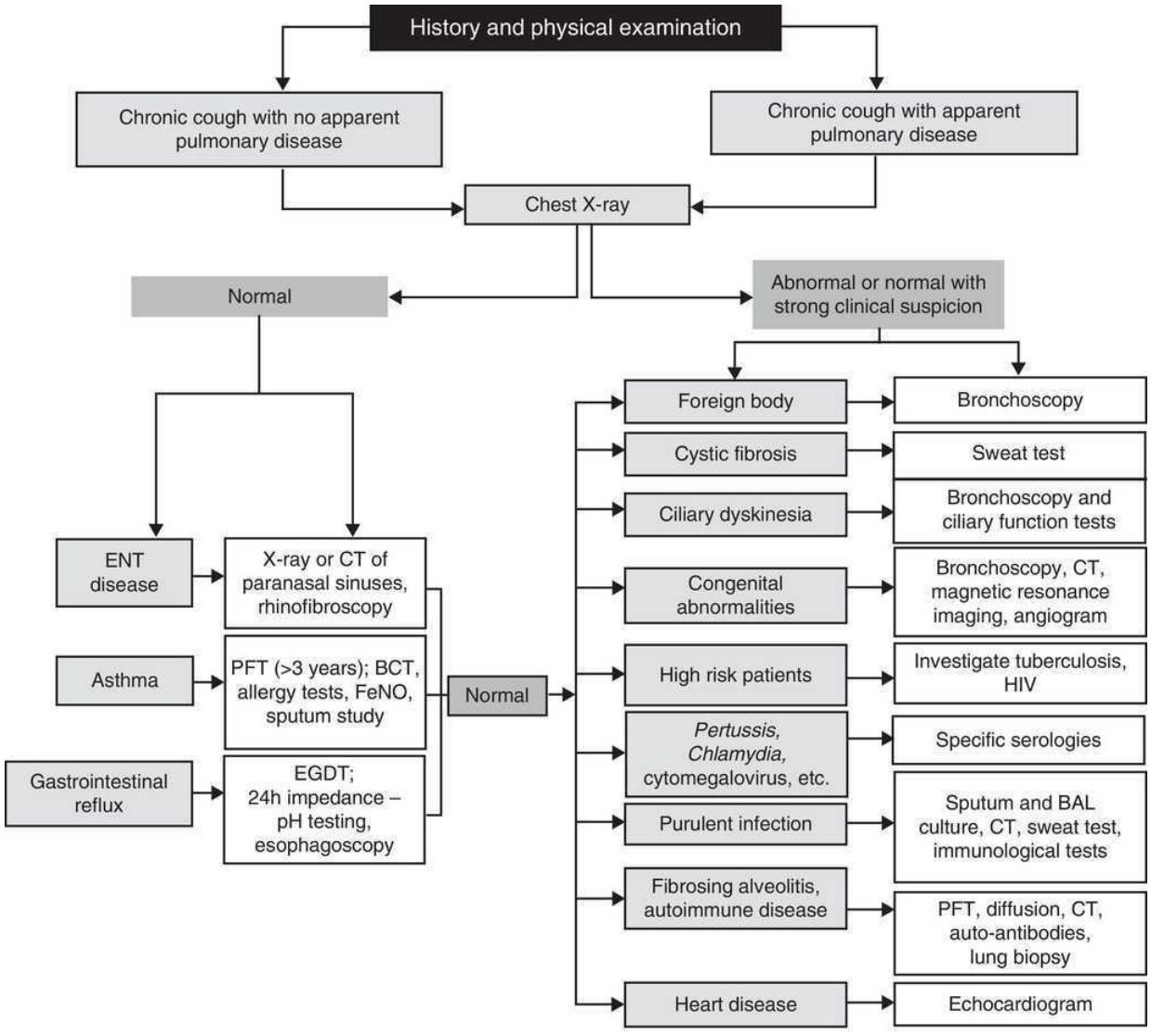
- Cough that occurs with **signs and symptoms suggesting a specific diagnosis** that has been reached after thorough clinical examination (not a well-child), treatment directed to a specific cause.
- This group includes asthma, bronchiectasis (BE), cystic fibrosis (CF), aspiration of a foreign body, aspiration symptoms, atypical respiratory infections, cardiac abnormalities and pulmonary interstitial disease.

Causes of chronic cough

In apparently healthy children	In apparently ill-children
• Repeated respiratory infections • Persistent bacterial bronchitis • Upper airway cough syndrome or post-nasal drip • Bronchial asthma • Psychogenic cough • **Irritative cough** (tobacco or other irritants)	• Suppurative diseases: CF, BE or PCD* • Immunodeficiencies • Aspiration syndromes • Aspiration of foreign body • **Infections:** Mycoplasma pneumoniae, Chlamydia trachomatis, tuberculosis • **Congenital abnormalities:** tracheo-esophageal fistula, vascular rings, airway malformations, neuromuscular diseases.

***BE: bronchiectasis; PCD: primary ciliary dyskinesia; CF: cystic fibrosis**

Algorithm for investigating children with chronic cough



Key points in clinical history of child with chronic cough

Nature of the cough	
Severity	Rule out potentially serious specific diseases
Time of appearance	Causes of cough vary with age
Diurnal variability	Nocturnal cough is more common with asthma or rhinitis
Sputum production	Evaluate suppurative diseases: CF, BE, PCD, PBB, etc.
Associated wheezing	Evaluate asthma
Cough during sleep	Psychogenic cough does not generally appear during sleep
Hemoptysis	Suppurative diseases, malformations, bronchitis
Time since onset	Allows cough to be classified as acute, subacute and chronic
Type of cough	Metallic, hacking, dry, spasmodic, staccato, paroxystic, etc.
Age at onset	Neonatal onset; congenital malformations or neuromuscular diseases
Relation with feeding or swallowing	Possible aspirative syndrome
Fever	Exclude infectious disease
Contact with TB and or HIV	Exclude these diseases
Chronic symptoms of ENT	Evaluate the possibility of PCD, chronic ENT diseases
Aspiration of foreign body	Consider always in case of sudden onset cough
Improvement of symptoms w/ medication	Evaluate improvement after administration of bronchodilators or antibiotics
Exposure to tobacco smoke	Evaluate if failure to resolve or protracted resolution
Triggering factors	Cold, temperature changes, exercise, exposure to allergens
Immune status & recurr. infectious disease	Evaluate the possibility of immunodeficiencies
Drug use	Evaluate rx with angiotensin converting enzyme inhibitors or others
History of atopy or chronic diseases	Possibility of asthma, CT, PCD, BE, etc.
Growth and development	Evaluate immunodeficiencies, congenital diseases.

Alarming symptoms and signs in children with chronic cough

- **Abnormal auscultation:** Asthma, bronchitis, foreign body, CF, cong. abnormalities
- **Heart murmur:** Heart disease
- **Neurological disease:** Expirative syndromes, muscle weakness, etc.
- **Chest wall deformities:** Malformations, severe chronic pulmonary disease
- **Failure to thrive:** Pulmonary or heart disease, etc.
- **Clubbing:** Pulmonary disease, suppurative disease, heart disease, etc.
- **Comorbidities:** Chronic diseases

Treatment of chronic cough

Chronic cough should be treated after a thorough etiological study, to eliminate the causative agent

The family must avoid exposing the child to tobacco smoke and other environmental irritants.

Specific Cough:

- **Asthma:** requires treatment with bronchodilators and, depending on classification, with inhaled corticosteroids.
- **Allergic rhinitis:** antihistamines and nasal steroids
- **Sinusitis:** will require treatment with antibiotics.
- **GERD:** should be treated with proton pump inhibitors and/or surgery.
- **Psychogenic cough:** requires investigation of the causes of stress or anxiety and subsequent psychological support

Non-specific cough:

Note: The use of central action antitussives, non-opiate antitussives, mucolytics or expectorants is not indicated.

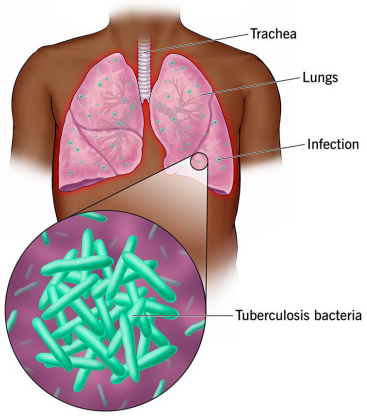
Non-specific cough without impact: Observation before diagnostic tests or treatment are initiated, with a follow-up examination of the child after 6–8 weeks.

Non-specific dry cough disturbing daily activity/sleep:

- A trial treatment with inhaled corticosteroids at half doses is recommended (budesonide 400µg/day or equivalent) for 2–12 weeks.
- The patient should be reassessed after 2–3 weeks and if there has been no response to treatment, it should be discontinued.
- Diagnosis of cough as "cough variant asthma" can only be established if symptoms recur after treatment withdrawal and respond again after re-introduction, so a positive response with inhaled corticosteroids does not confirm the diagnosis of asthma.

Non-specific productive cough: initiating a course of antibiotics (amoxicillin–clavulanate) for 2–3 weeks may be considered.

Tuberculosis



Etiology

There are 5 closely related mycobacteria:

- M. Tuberculosis, M. bovis, M.africanum, M. microti, and M. canetti.

Mycobacterium is an acid fast bacilli, non-spore-forming, nonmotile pleomorphic curved rods 2-4 µm long and obligate aerobes.

Epidemiology

- 95% of tuberculosis occurs in developing countries.
- WHO estimates that > 8 million new cases of TB occur & 3 million people die.
- **1.3 million cases** and **450,000 deaths** occur in children / year.

Global burden of TB continues to grow due to:

1. HIV epidemics.
2. Population migration patterns.
3. Increasing poverty.
4. Crowded living conditions
5. Inadequate health coverage.
6. Poor access to health services.
7. Inefficient treatment
8. Tuberculosis control programs

Immunity

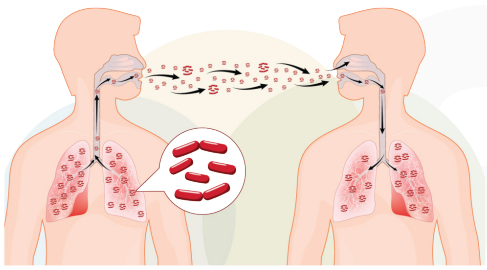
- A cell-mediated immune response terminates the growth of the M tuberculosis 2-3 weeks after initial infection.
- CD4 helper T cells activate the macrophages to kill the intracellular bacteria with resultant epithelioid granuloma formation. CD8 suppressor T cells causes lysis of the macrophages infected with the mycobacteria, resulting in the formation of caseating granulomas.
- Cytokines and TNF play a role in tissue damage, but antibody have little role.

Transmission of M. Tuberculosis

- Person to person, airborne mucus droplet nuclei, particles 1-5 µm in diameter.
- M. bovis may penetrate the gastrointestinal mucosa or invade the lymphatic tissue of the oropharynx when large numbers of the organism are ingested.

The chance of transmission with:

1. The patient having an acid-fast smear of sputum.
2. An extensive upper lobe infiltrate or cavity.
3. Copious production of thin sputum.
4. Severe and forceful cough.
5. Environmental factors, especially poor air circulation.



Clinical picture

Latent TB Infection (LTBI):

- Occurs when tubercle bacilli are in the body, but the immune system is keeping them under control.
- Detected by the Mantoux tuberculin skin test (TST) or by blood tests such as interferon gamma release assays (IGRAs).
- People with LTBI are **NOT infectious.**

TB Disease:

- Develops when immune system cannot keep tubercle bacilli under control – May develop very soon after infection or many years after infection.
- About 10% of all people with normal immune systems who have LTBI will develop TB disease at some point in their lives. Untreated infants with LTBI have up to a 40% likelihood of developing tuberculosis
- People with TB disease are often **infectious.**

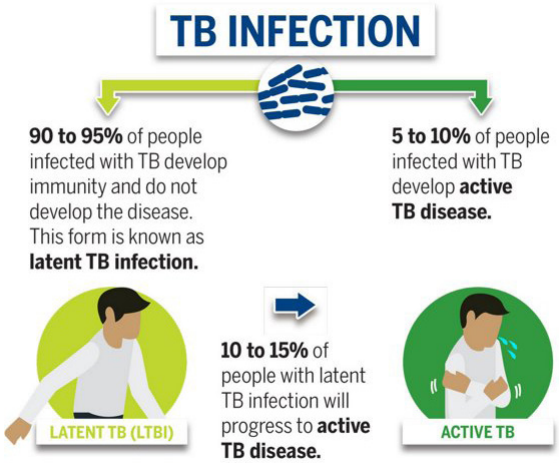
Latent TB Infection (LTBI)	TB Disease (in the lungs)
• **Inactive**, contained tubercle bacilli in the body • TST or blood test results usually **+ve** • Chest x-ray usually **normal** • Sputum smears and cultures **-ve** • **No symptoms** • **Not infectious** • **Not a case of TB**	• **Active**, multiplying tubercle bacilli in the body • TST or blood test results usually **+ve** • Chest x-ray usually **abnormal** • Sputum smears & cultures may be **+ve** • **Symptoms** such as cough, fever, wt. loss • Often infectious before treatment • A case of TB

Progression to TB Disease

- Risk of developing TB disease is highest the first 2 years after infection
- People with LTBI must be given treatment to prevent them from developing TB disease
- Detecting TB infection early and providing TTT helps prevent new cases of TB disease.

Risk Factors For Progression

- Children ≤ 4 yr of age and adolescents
- Persons co-infected with HIV
- Persons who are immune-compromised:
  - Malignancy
  - Solid Organ Transplantation.
  - Immunosuppressive medical treatments



Signs and symptoms of tuberculosis in children are classified into:

I. Asymptomatic TB Infection
II. Pulmonary tuberculosis
III. Extra pulmonary tuberculosis

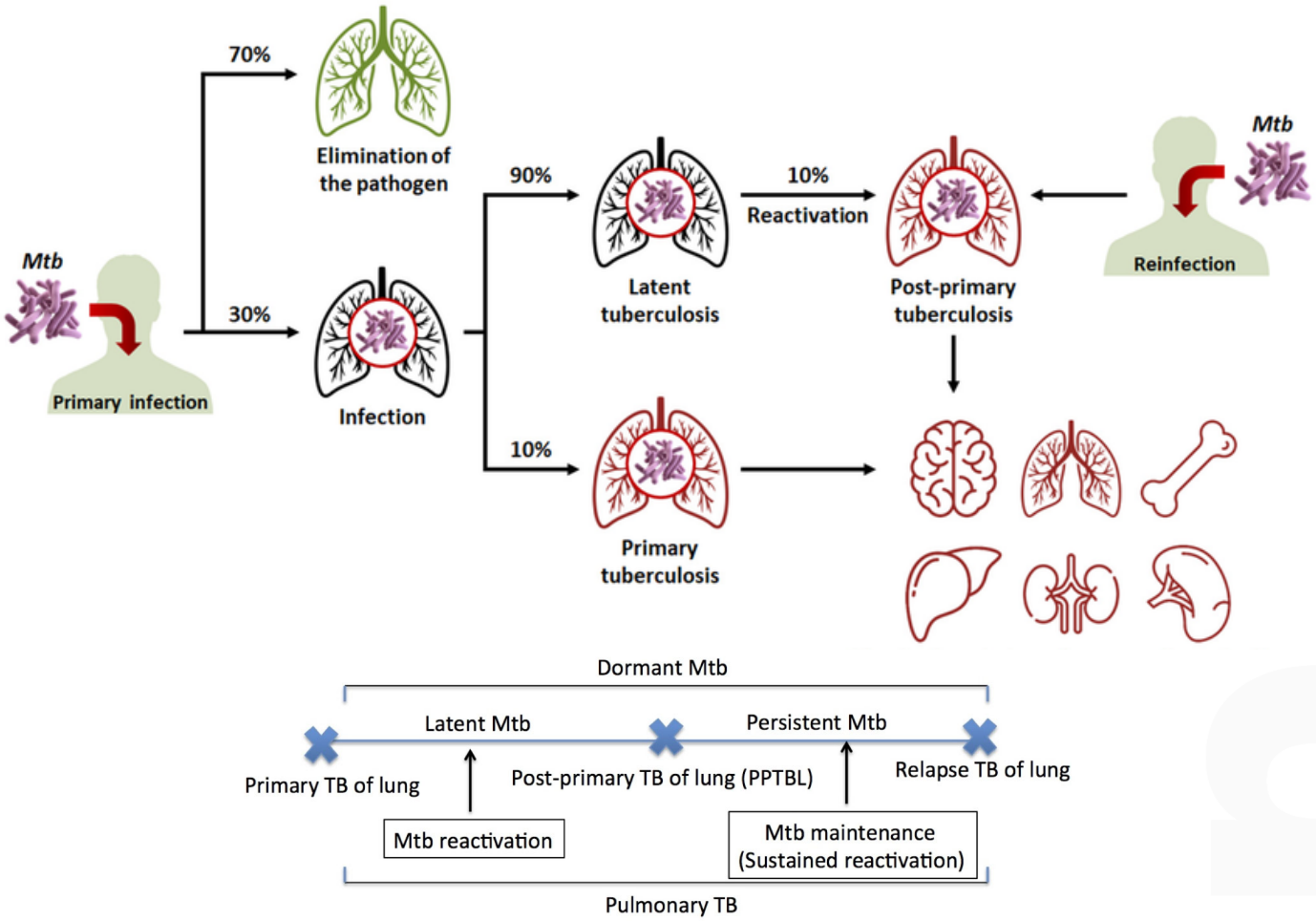
I. Asymptomatic TB Infection or Latent tuberculosis infection (LTBI)

- A **reactive tuberculin skin test** with **absence of clinical and radiographic manifestations.**
- Untreated infants with LTBI have up to a 40% likelihood of developing tuberculosis.

II. Pulmonary tuberculosis

1. Primary pulmonary TB with focal lymphadenopathy
2. Progressive pulmonary disease.
3. Reactivated pulmonary disease.
4. Pleural involvement

> **Note**
>
> Tuberculosis should be suspected when a persistent respiratory illness in an otherwise healthy individual does not respond to regular antibiotics.



Clinical picture of pulmonary tuberculosis:

1. Primary pulmonary disease

1ry pulmonary complex: small parenchymal focus (subpleural in 70% cases) + relatively large regional (hilar) lymphadenitis.

Symptoms and signs:

- 50% of cases are accidentally discovered by X-ray.
- Non-productive chronic cough, mild dyspnea, fever, night sweat, anorexia and failure to thrive in infants.
- Signs are less common; Decreased breath sounds, tachypnea, sometimes lobar pneumonia with cavity.

X-ray: Collapse, consolidation.

2. Progressive pulmonary TB

- Progression of the pulmonary parenchymal component leads to enlargement of the caseous area and may lead to pneumonia, atelectasis, and air trapping.
- 1ry focus → caseation & necrosis → 1ry cavity. If with erosion of adjacent bronchus → intrapulmonary dissemination.

Symptoms: are more severe (high fever, night sweat, productive cough, weight loss). Diminished breath sounds, rales, dullness.

3. Reactivation pulmonary TB

- Rare in infants, occurs in older children and adolescents who acquire the initial infection after 7 years of age.

Symptoms: as before. Weight loss, fever, cough, and, rarely, **hemoptysis.**

Signs: are minimal even if a large cavity is present

X-ray: Extensive infiltrate or thick walled cavity in the upper lobe.

4. Pleural effusion

- Local asymptomatic pleural effusion is so frequent in primary TB complex
- Larger significant effusions occur months to years after the 1ry infection and are rare in infants & infrequent in children < 6 years. Usually unilateral. It is due to rupture of subpleural pulmonary focus or caseated lymph node in the pleural cavity.

Symptoms: Sudden onset of fever, shortness of breath, chest pain on deep inspiration.

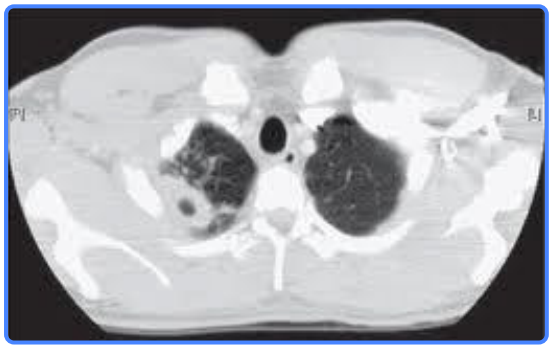
Signs: Diminished air entry & dullness.

X-ray: Unilateral pleural effusion that takes months to resolve.

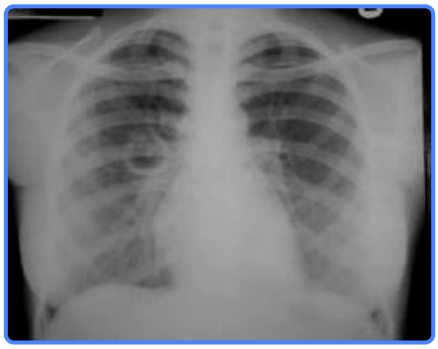
Pleural Fluid examination:

- **Color** is usually yellow, occasionally blood tinged
- **Specific gravity** 1012 – 1025
- **Protein:** 2-4 gm%
- **Decreased glucose.**
- **Marked increase in WBCs** in pleural fluid; early PNL, late lymphocytes.
- **Smears** of the pleural fluid are negative, **culture + ve** in 30- 70% of cases.

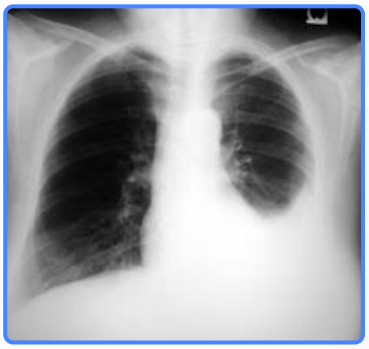
> **Note**
>
> Mycobacterial Culture is only positive in tuberculous effusion in 30% of cases as it is mainly hypersensitivity reaction.



Reactivation TB cavity



Reactivation TB cavity



Pleural effusion

III. Pediatric Extra-pulmonary TB

1. Lymph node involvement

- Most common form of extrapulmonary T.B.
- Occurs within 6 - 9 months from initial infection.
- The group affected depends on the primary site.

Clinical picture:

- Gradual enlargement of firm, non-fluctuant and non-tender LNs with non-erythematous overlying skin.
- With the progress of disease, multiple lymph nodes are infected and matted together.
- Infection usually unilateral but can be bilateral. Systemic symptoms are usually absent.

Investigations: Lymph nodes excision biopsy is diagnostic, +ve tuberculin test.

Prognosis:

- Resolution.
- Caseation, necrosis with spread to other lymph nodes
- Sinus formation.

Surgical removal is not an adequate treatment.

2. CNS Tuberculosis (Meningitis & Tuberculoma)

- Occurs in 5 - 10% of children younger than 2y

Pathogenesis:

- 1ry infection → lympho-hematogenous spread → caseous lesions in cerebral cortex or meninges → lesions increase in size & discharges TB bacilli in the subarachnoid space → gelatinous exudate infiltrates the cortico-meningeal vessels → inflammation, obstruction & infarction of cerebral cortex.
- Exudate interferes with CSF circulation at the basal cisterns → hydrocephalus
- Affection of the brain stem & cranial nerves 3,5,8.
- Electrolyte imbalance with salt wasting (syndrome of inappropriate secretion of ADH)

Course :

- **Rapid course (days):** with rapid progression to hydrocephalus, seizures & coma.
- **Slowly progressive course:** over several weeks.

Stages:

- **1st stage (1 - 2 wks):** nonspecific symptoms, fever, headache, irritability or drowsiness & no focal signs.
- **2nd stage:** sudden onset of vomiting, seizures, signs of meningitis, hydrocephalus, increase intracranial tension, and encephalitis
- **3rd stage:** Coma, paralysis (hemi or paraplegia, decerebrate posture), hypertension, deterioration of vital signs & death

Investigations:

1. **Tuberculin test** is –ve
2. **CSF:** ↑ cells mostly lymphocytes, ↓ glucose to 40 mg%, ↑ protein to 400 - 5000 mg/dl
3. **TB culture** is positive
4. **CT scan: hydrocephalus**, **tuberculomas** (focal neurologic defects) (supratentorial in adults, infratentorial in children)

3. Pericardial effusion (rare)

- Direct invasion by T.B. bacilli or lymphatic drainage from subcarinal lymph

C/O: non-specific symptoms.

Signs: pericardial frictions rub, distant heart sounds, pulsus paradoxus

Complication: constrictive pericarditis.

Pericardial fluid typically serofibrinous or hermorrhagic.

4. Disseminated TB (Miliary T.B.)

- Occurs when large numbers of T.B. bacilli are released into blood causing disease in **2 or more organs.** It is common in infants & young children.
- It occurs within 2 - 6 months of the initial infection.

Onset is often insidious with anorexia, weight loss and fever. Sometimes onset is explosive and patients become gravely ill in few days.

Symptoms: Low-grade **fever**, malaise, weight loss, and fatigue (+).

Signs:

- Generalized lymphadenitis & hepatosplenomegaly, pulmonary & miliary T.B.
- Meningitis or Peritonitis in 20 - 40% of cases.
- Cutaneous lesions: papulo-necrotic nodules or purpura.
- Choroid tubercles in the retina in 13 - 87% (specific for diagnosis).
- Respiratory signs may evolve to include tachypnea, cyanosis, and respiratory distress

Early diagnosis: liver or bone marrow biopsy (bacteriologic & histologic examination).

5. Bone or joint TB

- Vertebrae (50%), hip (15%), and knee (15%).
- Angulation of the spine (gibbus deformity)
- Pott's disease (severe kyphosis with destruction of the vertebral bodies).
- Cervical spine involvement may result in **atlantoaxial subluxation.**

6. Perinatal TB (Congenital T.B.)

- It may be present at birth but commonly begin by the 2nd or 3rd weeks of life.

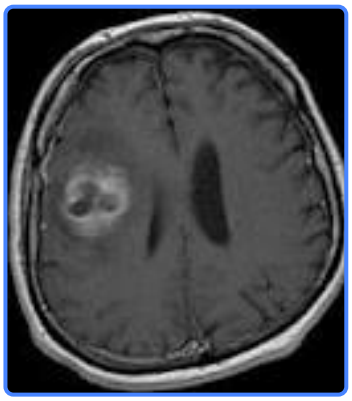
Symptoms and signs: Respiratory distress, fever, poor feeding, lethargy or irritability, failure to thrive. Hepatosplenomegaly, lymphadenopathy & ear discharge.

X-ray: Miliary shadow.

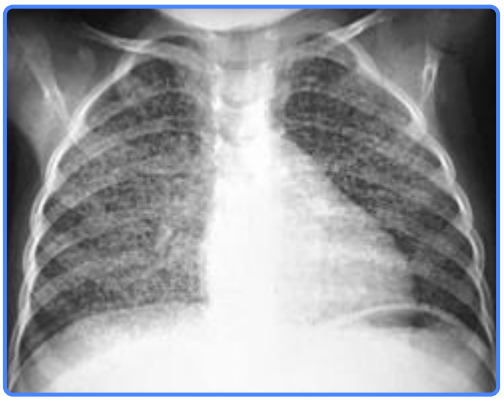
D.D: other congenital infections.

The clue is history of maternal T.B. & failure to respond to ordinary antibiotics.

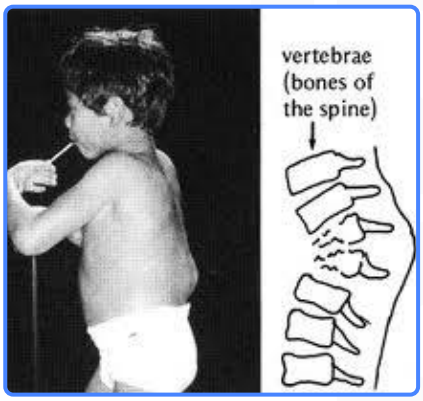
Tuberculin test is -ve, may become + ve in 1 - 3 months.



Tuberculoma



Military TB



Pott's disease

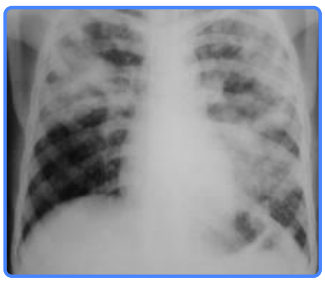
Diagnosis of tuberculosis

Symptoms

The commonest are:

- **Prolonged cough:** An unremitting cough that is not improving and has been present for more than 21 days (< 3 weeks).
- **Fever:** Body temperature of > 38 °C for 14 days, after common causes such as malaria or pneumonia... etc have been excluded.
- **Weight loss or failure to thrive**

Investigations



Radiological investigations: Chest x-rays, HRCT Chest, abdominal ultrasound, HRCT brain, X ray vertebral column

Can distinguish LTBI

Screening by: Tuberculin test / IGRAs; IFN-γ Release Assays (QuantiFERON TB GOLD In Tube (QFT) and T-SPOT.TB (T-SPOT)

Blood tests: increase ESR and relative lymphocytosis.

Microscopic examination and microbiological culture/ PCR testing of sputum

- In children unable to expectorate sputum, induction of sputum should be considered if it can be done safely, with gastric washings

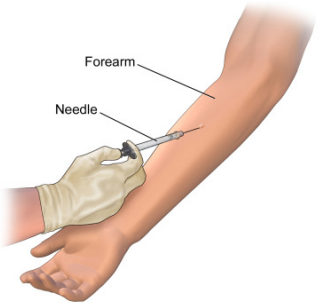
Biopsy and needle aspiration of lymph nodes

Analysis, PCR and culture of CSF, pleural and pericardial fluids

Scoring system to calculate probability of tuberculosis

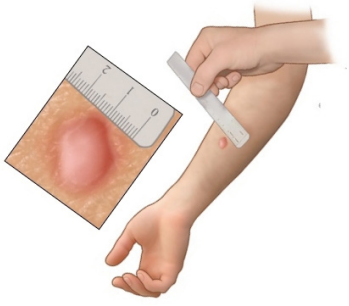
Contacts are also screened and treated if necessary.

Tuberculin Skin Testing



- The Mantoux tuberculin skin test is the intra-dermal injection
- 0.1 mL containing **5 tuberculin units of purified protein derivative (PPD)**
- Induration in response to the test should be measured by a trained person 48-72 hr after administration.

Children for whom immediate TST is indicated:



- **Contacts** of people with confirmed or suspected tuberculosis.
- Children with **radiographic or clinical findings** suggesting tuberculosis
- Children **immigrating from countries** with endemic infection
- Children with **travel histories to countries** with endemic infection & substantial contact with people from such countries

False-negative tuberculin test

- Very young age.
- Malnutrition.
- Immunosuppression
- Vaccination with live-virus vaccines.
- Overwhelming tuberculosis

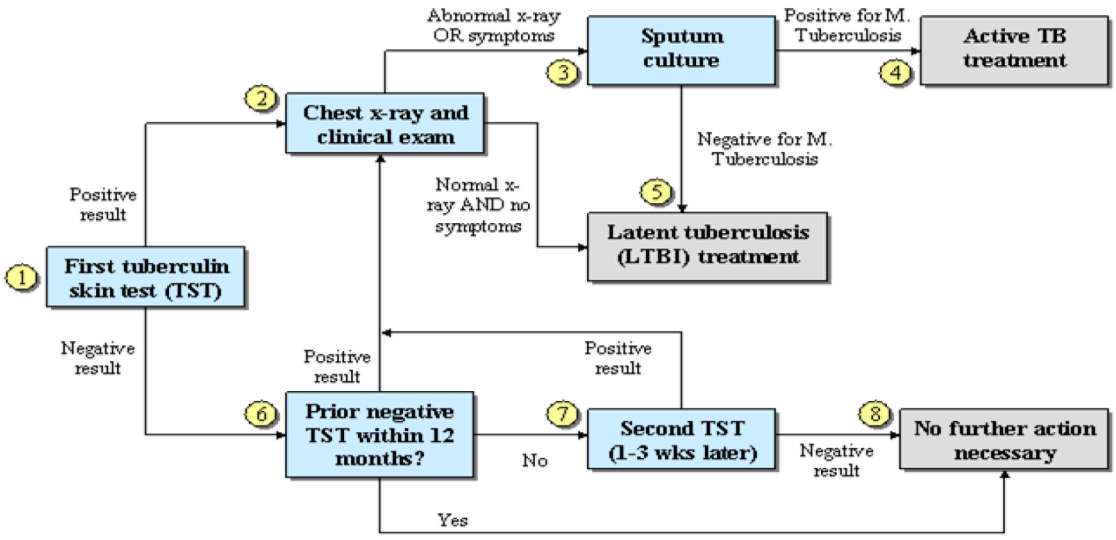
False-positive tuberculin test

- Cross sensitization to antigens of non-tb mycobacteria
- Previous vaccination with (BCG)

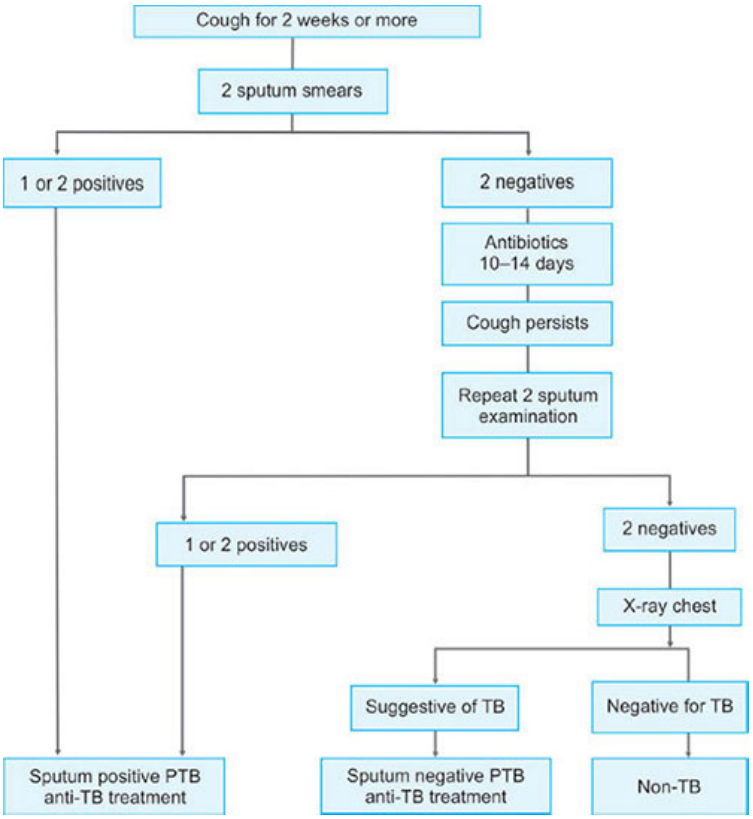
Note

The most common reasons for a false-negative skin test are poor technique and misreading of the results

Interpretation of tuberculin test:



Diagnostic algorithm for PTB:



Treatment

I. Pulmonary T.B.

Triple therapy: INH + RIF (6 months) + PZA (2 months)

Quadruple therapy: In locales where the community rate of INH resistance is greater than 5 – 10%, add a **4th drug (usually STM, EMB).**

II. Extrapulmonary T.B.

- Same as for pulmonary quadruple therapy T.B. for 9 - 12 months.
- Exceptions are HIV infected (seropositive), disseminated tuberculosis and tuberculous meningitis: 9 - I2 months.

Direct observation of therapy (DOT)

- Direct observed therapy and short course program twice-weekly dose used to ensure patient compliance.
- It involves providing the antituberculosis drugs directly to the patient and watching as they swallow the medications.
- It is the preferred core management strategy for all patients with tuberculosis.

Drug	Daily Dose (mg/kg/24hrs)	Twice-weekly Dose (mg/kg/dose)	Maximum Daily dose	Side effects
Isoniazid (INH)	10 - 15 10 - 20 in presentation	10 - 15 10 - 20 in presentation	300 mg	Elevation of hepatic enzyme levels, hepatitis, neuropathy, CNS effects
Rifampin (RIF)	10 - 20 10 - 20 in presentation	10 - 20	600 mg	Orange discoloration of secretions and urine, GIT upset, hepatitis, bleeding problems, flu-like symptoms, drug interactions, rash
Pyrazinamide (PZA)	20 - 40 10 - 20 in presentation	40 - 60 50 in presentation	2 gm	Gastrointestinal tract upset, hepatitis, hyperuricemia, arthralgias
Streptomycin	20 - 40 10 - 20 in presentation	20 - 40		Ototoxicity
Ethambutol	15 - 25 10 - 20 in presentation	25 - 50 50 in presentation	2.5 gm	Optic neuritis

2nd line medications: Amikacin and kanamycin, Capreomycin, Cycloserine, Ethionamide, Streptomycin, p-Aminosalicylic acid, Fluoroquinolone.

Corticosteroids:

- **Dose:** Prednisone 1-2 mg/kg/24hrs 4-6 weeks with gradual tapering
- **indications:**
  - T.B. meningitis.
  - Endobronchial T.B.
  - TB pericarditis
  - Pericardial effusion
  - Miliary T.B.
  - Pleural effusion with mediastinal shift.

Multidrug resistant tuberculosis (MDR-TB)

Infection caused by MDR organisms, defined as organisms resistant to **at least INH and rifampin.**

- **Primary resistance:** resistance to anti-tuberculosis treatment in an individual who has no history of prior treatment.
- **Secondary resistance:** emergence of resistance during the course of ineffectual anti-tuberculosis therapy.

Prevention

1. Finding infected cases: Tuberculin test for close contacts of adult T.B. cases.

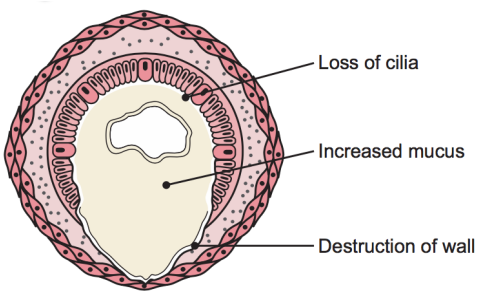
2. Bacille Calmette-Guérin (BCG) Vaccination:

- Prepared from Strain of M. Bovis attenuated
- Given by intradermal injection
- Safe in immunocompetent hosts.
- Local ulceration and regional suppurative adenitis occur in 0.1 - 1% of recipients.

Bronchiectasis

Bronchi = Bronchial tree, ectasis = Dilation

Chronic dilatation of bronchi associated with inflammatory destruction of bronchial and peri-bronchial tissue, accumulation of exudative suppurative material in bronchi.



Pathogenesis

Always starts with chronic infection of bronchi, Sequence of Persistent Chronic Airway Inflammation is fibrosis + dilation + airway damage = bronchiectasis

Cause of this chronic infection is one or more of the following:

1. Resistant Organism infection
2. Stasis of bronchial secretions (physical/functional)
3. Immunodeficiency (host defense defect; congenital; acquired or iatrogenic)

Common chronic infection organisms not properly treated:

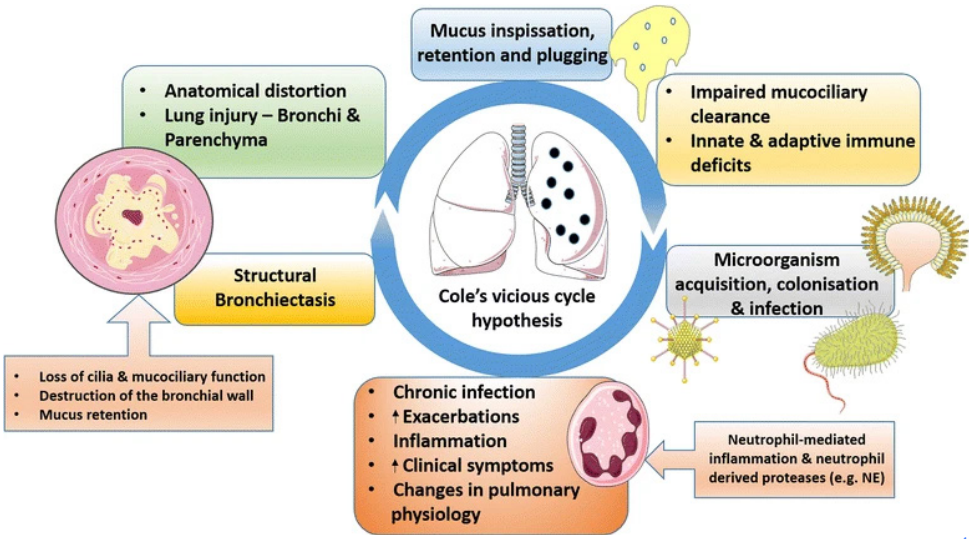
- B. Pertussis
- Allergic Broncho-Pulmonary Aspergillosis
- Strep. pneumoniae
- Staph. aureus

Common related immune deficiency causes:

- Congenital; AB defects. SCID
- Acquired; HIV
- Following chemotherapy
- Ataxia telangiectasia

Common causes of stasis of secretions are:

- **Bronchial obstruction:** foreign body aspiration, tumors, LN, congenital airway defect
- **Functional stasis:** cystic fibrosis, chronic aspiration, primary ciliary dyskinesia



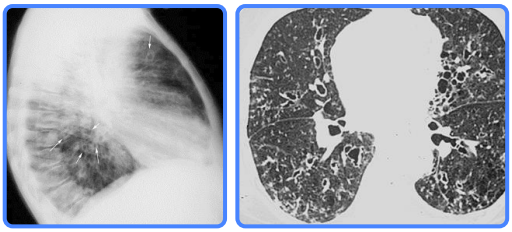
C/P

Symptoms	Signs

• Cough with mucopurulent expectoration < 8 weeks (copious amount, foul smelling, worse in the morning)
• Haemoptysis
• Recurrent infections of LRT
• Breathlessness
• Fever
• Postural variation of symptoms
• Chest pain
	• Poor general condition, growth delay
	• Tachypnea
	• Dyspnea: use of accessory muscles
	• Clubbing (pale)
	• Dullness to percussion over the bronchiectasis area.
	• Breath sounds:
	  • Decreased air entry.
	  • Harsh with prolonged expiration
	  • Coarse leathery crackles
	  • Moist-musical rales + sibilant rhonchi
	• Kartagener's syndrome may be found

Investigations

Diagnosis of bronchiectasis & its severity	Diagnosis of the cause

• Imaging (CXR, HRCT chest)
• Bronchoscopy
• Microbiology (sputum C&S)
• Pulmonary function
 
	• Sweet chloride test
	• pH metry
	• Immune system test:
	  • Immunoglobulin level (IgG, IgM, IgA, IgE)/ IgG subclasses
	  • Rheumatoid factor
	• Aspergillus precipitins
	• Alpha-1 antitrypsin level
	• Ciliary brush test
	• HIV test

Complications

Local

- Haemoptysis
- Secondary bacterial infections
- Fungal infections
- Tuberculosis
- Lung abscess
- Frequent exacerbations

Systemic:

- Respiratory failure
- Cor-pulmonale, Pulmonary a. hypertension (PAH)
- Sinusitis
- Allergic bronchopulmonary aspergillosis(ABPA)
- Aspergilloma
- Brain abscess
- Secondary amyloidosis

Treatment

Oral and systemic antibiotics: after culture & sensitivity with treatment of sinusitis if present.

Immunomodulatory/Anti-inflammatory therapy;

- Steroids
- Macrolides, tetracyclines
- Interferon-gamma
- Ibuprofen

Bronchodilators and mucolytic inhalation followed by **postural drainage & physiotherapy**

Deep breathing exercises, ACT

Surgery: removal of affected lobe if medical treatment fails and lesion is localized, such as

- Localized bronchiectasis
- Proximal obstructive lesion
- Massive hemoptysis
- Recurrent infections

Broncho-pulmonary Hygiene:

- Removal of respiratory secretions is beneficial
- Chest percussion and postural drainage, Chest clapping or cupping.
- Inflatable vests or mechanical vibrators
- Oral devices that apply positive end-expiratory pressure maintain the patency of the airway during exhalation
- Maintaining adequate systemic hydration, enhanced by nebulization with saline,
- Acetylcysteine delivered by nebulizer thins secretions
- aerosolized recombinant human DNase (rh-DNase) in patients with cystic fibrosis

Specific causes of chronic purulent cough in childhood

Cystic fibrosis

- Is an inherited condition that causes sticky mucus to build up in the **lungs and digestive system.**
- This causes **lung infections, bronchiectasis (suppurative lung disease) and steatorrhea (fatty diarrhea).**
- Failure to thrive, clubbing may occur.
- Diagnosis through **sweat chloride test and genetic testing.**

Primary ciliary dyskinesia

- Is a congenital disorder caused by genetic mutations that affect the mucosal cilia structure of the airways and elsewhere.
- Transient neonatal respiratory distress is common, and a chronic wet cough is a major symptom with onset during infancy.
- Also involved is **chronic rhinorrhea and recurrent otitis media.**
- Situs inversus is present in about 50% of cases known as **Kartagener's syndrome.**

5 RESPIRATORY DISTRESS & EMERGENCIES

Respiratory distress is the increase in the work of breathing to get more oxygen.

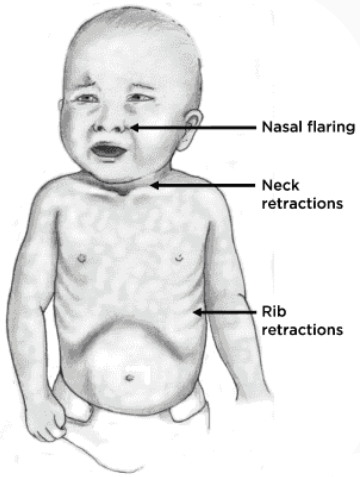
Signs of respiratory distress

1. Tachypnea: Respiratory rate of:

- **> 60** breaths/min in infants aged **0 – 2 months**
- **> 50** in infants **2 – 12 months**
- **> 40** in children **1 – 5 years**
- **> 20** in children **> 5 years of age.**

2. Retractions:

- Intercostal and subcostal (more in lower airway diseases)
- Supraclavicular, and suprasternal (more in upper airway).



3. Working alae nasi.

4. Grunting.

5. Cyanosis.

+ Tracheal tugging & head bobbing.

Causes of respiratory distress

1. Respiratory causes

- **Upper respiratory diseases:** croup, anaphylaxis
- **Lower respiratory diseases:** bronchial asthma, pneumonia, bronchiolitis.

2. Cardiac causes

- Arrhythmia
- Heart failure
- Cardiac tamponade.

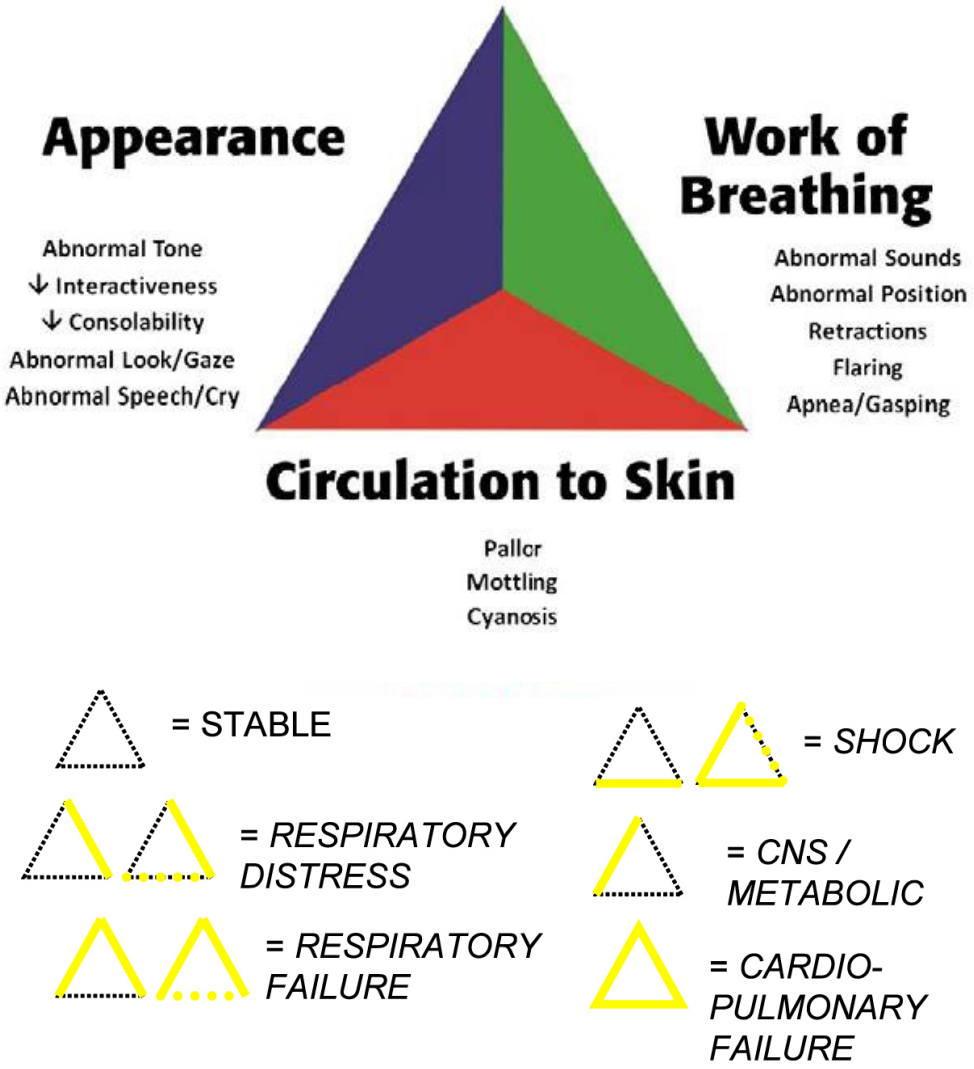
3. Neurological causes

- Seizures
- Neuromuscular diseases
- increased ICP
- Poisoning

4. Metabolic causes

- Metabolic acidosis:
  - DKA, renal failure, IEM

Management of respiratory distress



I. Primary assessment by ABC

- Normal ABC = no respiratory distress
- Normal AC, abnormal **B** = respiratory distress
- Normal C, abnormal **AB** = respiratory failure
- Abnormal **ABC** = cardiorespiratory failure

II. General management of respiratory distress:

- keep the child calm
- Supply oxygen as needed
- Suction of nasal airways as needed
- Monitor respiratory status with pulse oximetry and ECG monitoring as indicated

III. Specific management of respiratory distress: according to the cause

Respiratory causes of respiratory distress

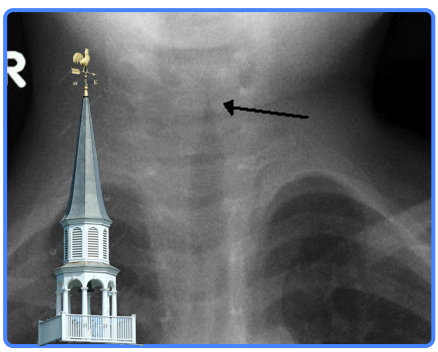
Stridor

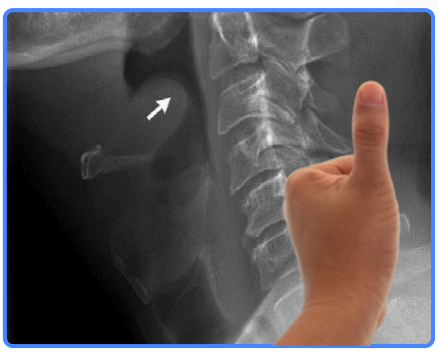
Causes of stridor

- Acute laryngo-tracheo-bronchitis
- Acute epiglottitis
- Spasmodic croup
- Foreign body aspiration

Acute laryngotracheobronchitis (croup) Vs. Acute epiglottitis

Acute laryngotracheobronchitis		Acute epiglottitis
Parainfluenza, RSV, adenovirus	**Cause**	Hemophilus influenza type B
Gradual onset (viral prodrome)	**Onset**	Abrupt onset
Fair	**Appearance**	Toxic
Low grade	**Fever**	High grade
Barking cough, stridor	**Symptoms**	Respiratory distress, drooling of saliva, dysphagia, dysphonia, minimal or no cough
Normal	**WBC**	Elevated
Steeple sign	**X-ray**	Thumb sign
• Dexamethasone • Nebulized Racemic epinephrine • Humidification	**Treatment**	• Endotracheal intubation in the operating room if needed • I.V. antibiotics (penicillin or cephalosporin)





Spasmodic croup

Resembles acute laryngotracheobronchitis but cause could be allergic or psychogenic

- No fever
- Usually recurrent attacks
- Worsens at night.

Foreign body aspiration

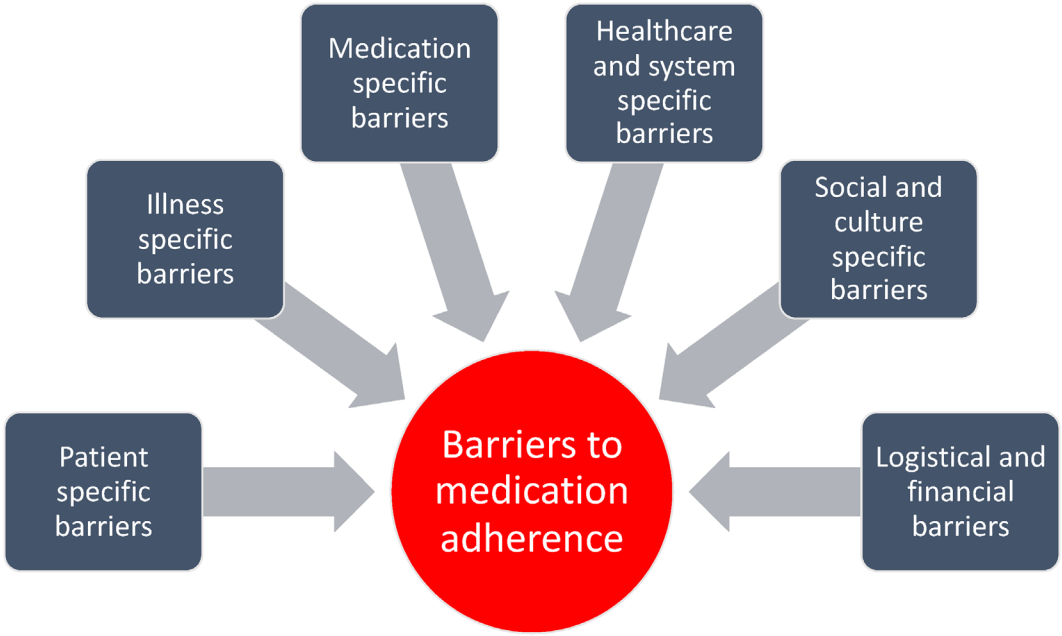
As mentioned before (pg. 4)

Acute severe asthma (asthma flare-up)

The condition when symptoms of asthma, such as wheezing, coughing, or shortness of breath, become more severe, more frequent, or both

Lines of treatment of acute asthma:

- Oxygen
- Inhaled Short-acting B2 agonist
- Inhaled anticholinergics: Inhaled ipratropium bromide
- Oral/intravenous (IV) corticosteroids
- Magnesium sulfate I.V infusion
- Aminophylline I.V infusion



Assess the severity of asthma attack

	Mild	Moderate	Severe	Impending respiratory failure
Mental status	Normal	May be agitated	agitated	Drowsy, confused (cerebral hypoxemia)
Speech	Normal	Speaks in phrases	Speaks in words	Unable to speak
Work of breathing	Minimal intercostal retractions	Intercostal and subcostal retractions	Significant respiratory distress (accessory muscles, working ala nasi)	Marked respiratory distress at rest (retractions, working ala nasi, grunting)
Chest auscultation	Expiratory wheezes	Inspiratory and expiratory wheezes	Audible wheezes without a stethoscope	Silent chest (no air entry)
O2 saturation at room air	> 94%	91 - 94%	< 90%	< 90%
Peak flow (%of personal best)	> 80%	60 - 80%	< 60%	Unable to perform
Treatment	• Keep oxygen saturation above 94% • Inhaled short acting B2 agonist (salbutamol) every 20 min • Inhaled corticosteroids	• Keep oxygen saturation above 94% • Inhaled short acting B2 agonist salbutamol every 20 min • Oral corticosteroids (prednisone or dexamethasone) • Consider inhaled ipratropium bromide	• Keep oxygen saturation above 94% • Consider IV methyl-prednisone • Consider continuous aerosolized short acting B2 agonist -Consider I.V MgSO2	• Keep oxygen saturation above 94% • Consider continuous aerosolized short acting B2 agonist (salbutamol) and Long acting Muscarinic antagonists (ipratropium) • Blood gases and electrolytes • Call for PICU to consider I.V MgSO2, I.V. aminophylline, or I.V. salbutamol • Consider S.C. adrenaline

Acute Bronchiolitis

(as mentioned in section of acute cough)

Indications of hospital admission

1. **Respiratory distress**
2. **Oxygen saturation below 92% in room air**
3. Chronic lung disease
4. Congenital heart disease
5. Prematurity
6. Age younger than 3 months
7. Inability to maintain oral hydration in patients younger than 6 months and difficulty feeding as a consequence of respiratory distress
8. Parents unable to care for their child at home

Indications of PICU admission

- Worsening hypoxemia or hypercapnia
- Worsening respiratory distress
- Persistent oxygen desaturation and/or severe cyanosis despite adequate oxygen delivery
- Apnea
- Respiratory acidosis
- Worsening mental status

Treatment of severe bronchiolitis

- Oxygen supplementation: to keep oxygen saturation above 90%.
- Hydration
- Bronchodilators (nebulized salbutamol, ipratropium, epinephrine???)
- Corticosteroids??
- Inhaled Hypertonic saline??
- In case of secondary bacterial infection: antibiotic.

Pneumonia

(as mentioned in section of acute cough)

Indication of hospital admission

1. Respiratory distress
2. Oxygen saturation below 92% in room air
3. Comorbidities (e.g., chronic lung disease, asthma, unrepaired or incompletely repaired congenital heart disease, diabetes mellitus, neuromuscular disease)
4. Infants less than 3 months old

Management of pneumonia

Antibiotics

- Amoxicillin-clavulanate OR cephalosporin ± azithromycin or clarithromycin
- In MRSA infection: vancomycin, clindamycin, or linezolid.

Antiviral: oseltamivir for influenza

Treatment of complications of pneumonia which include:

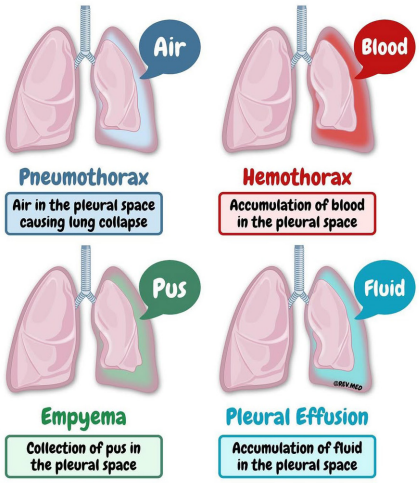
- Pleural effusion
- Empyema
- Lung abscess
- Necrotizing pneumonia
- Sepsis

> **Note**
>
> - **No need for nebulization** except if there are wheezes.
> - **No need to suppress cough**
> - **No need for mucolytics** esp. below 2 years

6 PLEURAL DISEASES

Definitions

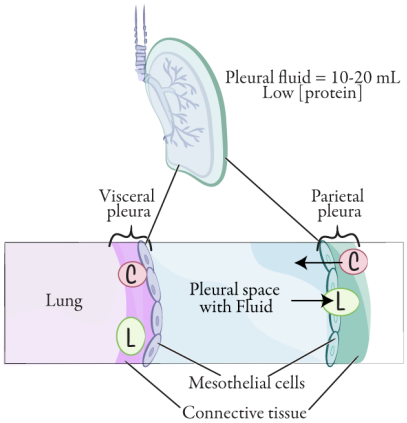
- **Pleurisy:** is the inflammation of the pleura.
- **Pleural effusion:** fluid in pleural space
- **Pneumothorax:** air in pleural space
- **Hydropneumothorax:** fluid and air in pleural space
- **Empyema:** pus in pleural space
- **Hemothorax:** blood in pleural space
- **Chylothorax:** lymphatic fluid in pleural space.



Pleural effusion

Starling forces

Normally, approximately 10 mL of fluid is present in the pleural space



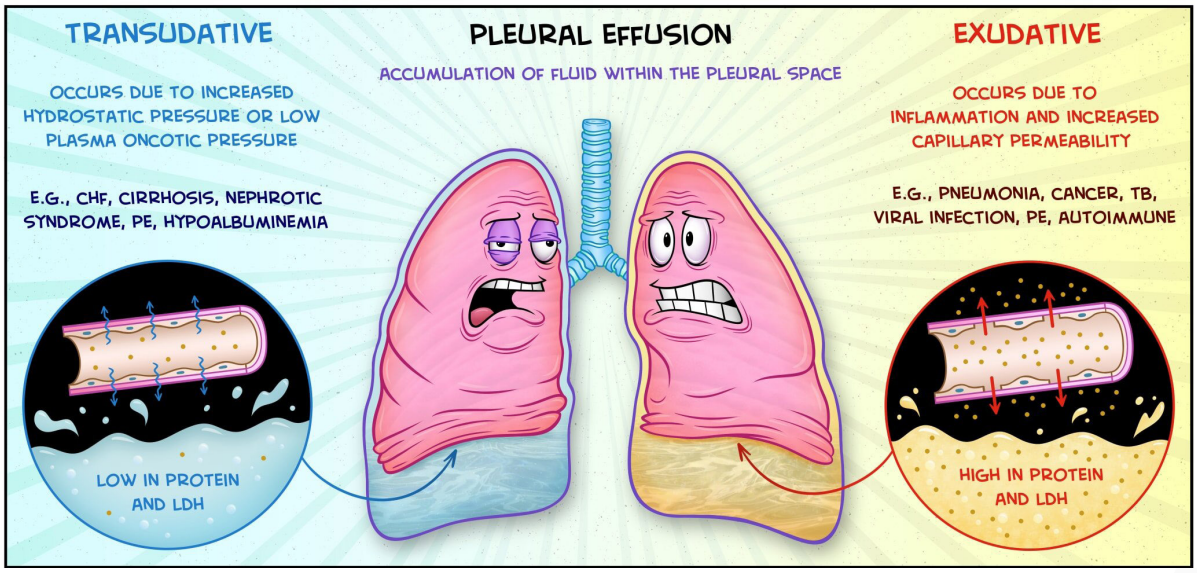
Types of pleural effusion

Transudate:

- Occurs due to increase in hydrostatic pressure or decrease in plasma oncotic pressure
- e.g. congestive heart failure, nephrotic syndrome, liver cirrhosis, hypoalbuminemia

Exudate:

- Occurs due to inflammation and increased capillary permeability
- e.g. pneumonia, T.B., cancer, autoimmune disease



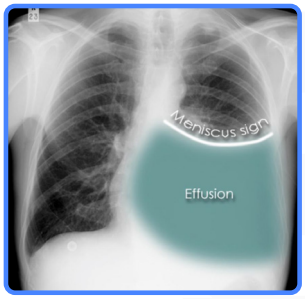
Clinical picture

Symptoms: Chest pain, cough, dyspnea, tachypnea, fever

Chest examination: decrease air entry, decreased TVF, tracheal shift to the opposite side, dullness on percussion.

Investigations

1- Radiology: Chest X-ray: homogenous opacity compressing the lung with mediastinal shift, Chest CT, Chest ultrasound.



2- Laboratory test:

- CBC: neutrophilia in parapneumonic effusion, lymphocytosis in T.B effusion
- CRP: elevated

3- Culture: blood or sputum or pleural fluid culture and senstivity

4- Thoraco-centesis and analysis of pleural fluid to differentiate between transudate and exudate

	Transudate	Exudate
Appearance	Serous	Cloudy
pH	> 7.2	< 7.2
Protein	< 3 gm/dl	> 3 gm/dl
LDH	< 200 IU/L	> 200 IU/L
Glucose	> 60 mg/dl	< 60 mg/dl
Cell count	< 10,000/mm^3	> 10,000/mm^3

Light's criteria

The fluid is exudate if at least one criterion is present:

- Pleural fluid protein/serum protein > 0.5
- Pleural fluid LDH/serum LDH > 0.6
- Pleural fluid LDH > 2/3rd the upper normal serum level

Treatment of pleural effusion

Antibiotic therapy

Drainage by tube thoracostomy

Rapid removal of ≥1 L of pleural fluid may lead to re-expansion pulmonary edema.

Fibrinolytic agents: urokinase, streptokinase, tissue plasminogen activator.

Video-assisted thoracoscopy: debridement or lysis of adhesions and drainage of loculated areas of pus.

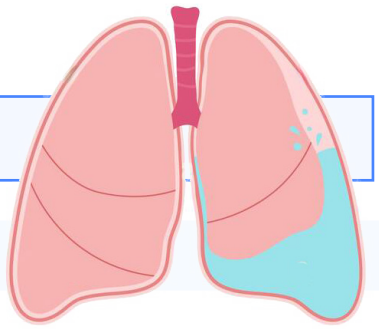
Decortication (pleurectomy)

Pleurodesis

Antibiotic therapy in pleural effusion

- a pleural fluid culture and sensitivity should be performed prior to the initiation of antibiotics.
- Some groups of antibiotics (e.g., penicillins , cephalosporins, aztreonam, clindamycin, and ciprofloxacin exhibit more satisfactory pleural fluid penetration than others (e.g., aminoglycosides).
- Many centers continue with intravenous antibiotics at least 48 hours after the patient is afebrile and the chest drain is removed.
- Thereafter, oral antibiotics are commonly continued for 2 4 weeks.

Hydrothorax



A transudative pleural effusion

Causes

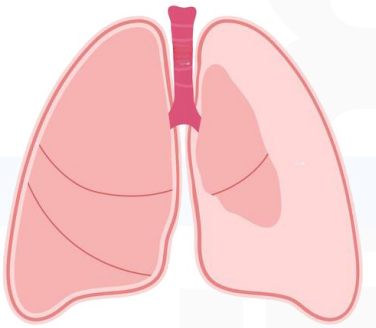
- Cardiac, renal, or hepatic disease (common causes).
- Severe nutritional edema and hypoalbuminemia.
- Rarely, SVC obstruction by neoplasms, enlarged lymph nodes, pulmonary embolism, or adhesions.
- Ventriculoperitoneal shunt, central venous catheter, or peritoneal dialysis.

Lab. Investigations

- Noninflammatory
- Few cells
- Lower specific gravity (<1.015)
- The ratio of pleural fluid to serum total protein is <0.5
- the ratio of pleural fluid to serum LDH is <0.6
- The pleural fluid LDH value is less than 66% of the upper limit of the normal serum LDH range.

Treatment: for the cause.

Pneumothorax



Causes of pneumothorax:

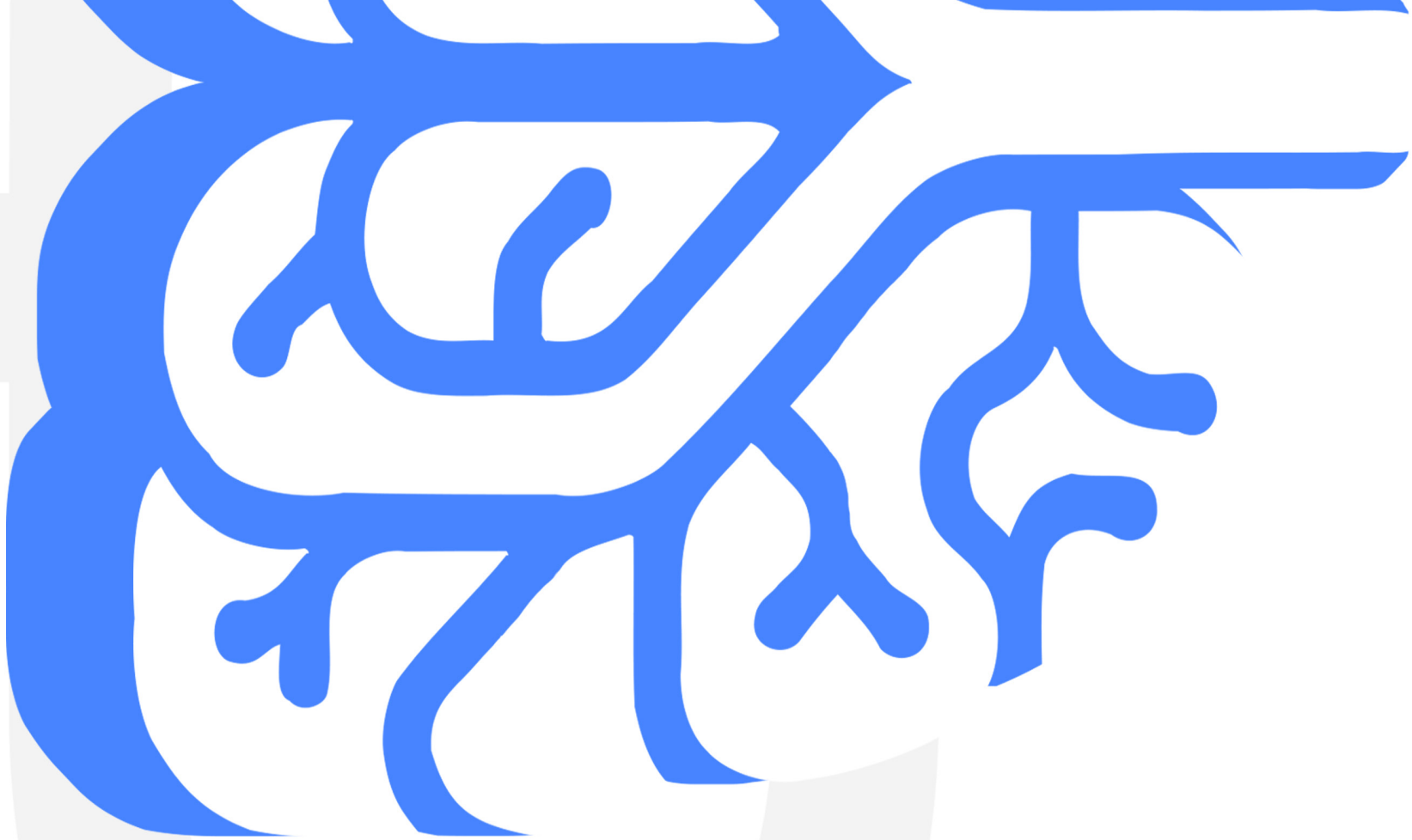
Spontaneous idiopathic: rupture of subpleural bleb

Secondary:

1. **Infections:** Pneumatocele, Lung abscess, Bronchopleural fistula, TB, Echinococcus
2. **Congenital lung diseases** (CPAM, bronchogenic cyst)
3. **Conditions that increase intrathoracic pressure** (asthma, bronchiolitis, CF)
4. **Connective tissue disease** (Marfan, Ehler Danlos)
5. **Traumatic** (penetrating or blunt trauma)
6. **Iatrogenic** (mechanical ventilation, central line insertion)
7. **Malignancy and autoimmune diseases**

Treatment of pneumothorax: chest tube insertion or needle aspiration.

END.



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Pediatrics