Chapter 10

Respiratory System Diseases

CONTENTS

- Tonsillo – pharyngitis
- Laryngeal obstruction
- Acute bronchiolitis
- Bronchial asthma
- Pneumonias
- Pleurisy
- Pleural effusion
- Pneumothorax
- Primary pulmonary tuberculosis

TONSILLO – PHARYNGITIS

It is the acute inflammation of the pharynx and the tonsils.

Etiology:

Acute pharyngitis is less commonly seen during the first year of life, and peaks by 4-7 years.

1. Viral in ≥ 80% e.g. adenoviruses, rhinoviruses and enteroviruses.
2. Bacterial: most common is group A β-hemolytic streptococcus. Rarely other bacteria e.g. pneumococci, Staphylococci, H. influenza. Diphtheria should not be forgotten.

Clinical picture:

1. Fever, sore throat, malaise, anorexia, cough, pharyngeal congestion and exudates and cervical lymphadenopathy are features common in both viral and streptococcal pharyngitis.
2. In viral cases conjunctivitis, rhinitis, and hoarseness are common. The course is 1-5 days.
3. In streptococcal cases pharyngeal and tonsillar congestion and exudate are more marked, and tonsils are enlarged with yellow beads of pus may be seen. A pseudo-membrane may cover the tonsils. Breath smells offensive. The course is 1-2 weeks.

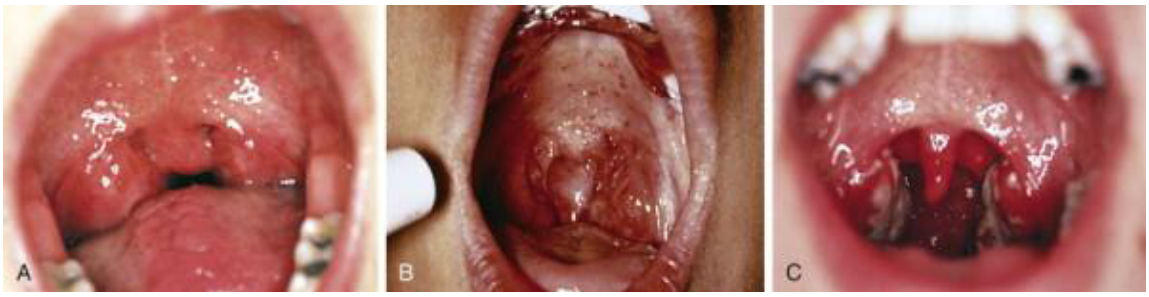
Investigations:

1. In both viral and bacterial cases there is polymorph. leucocytosis.
2. Streptococcal infection can be diagnosed by:
   i. Rapid antigen detection test of throat swabs (needs only 2 hours).
   ii. Bacterial culture of throat swab, this needs two days.

Complications:

1. Bacterial otitis media or sinusitis as well as mesenteric adenitis may follow viral or streptococcal pharyngitis.
2. Streptococcal pharyngitis may be complicated by abscess of the cervical lymph nodes or by peritonsillar abscess.
3. Sequelae of streptococcal pharyngitis:
   i. Rheumatic fever.
   ii. Acute glomerulonephritis

Differential diagnosis:



A, The diffuse tonsillar and pharyngeal erythema seen here is a nonspecific finding that can be produced by a variety of pathogens.
B, This intense erythema, seen in association with acute tonsillar enlargement and palatal petechiae, is highly suggestive of group A β-streptococcal infection, though other pathogens can produce these findings.
C, This picture of exudative tonsillitis is most commonly seen with either group A streptococcal or Epstein-Barr virus infection.
D.When a membrane is formed on the tonsils, diphtheria should be confirmed or excluded. If doubt exists the case should be treated as diphtheria.

Treatment:

A- Symptomatic:

1. Rest.
2. Antipyretics and analgesics. Paracetamol 50 mg/kg. / day or ibuprofen 20 mg/kg/day.
3. Diet: milk pudding, fruit juice and warm fluids. Then give small amounts of other foods as appetite improves.

B- Antibiotics: If streptococcal etiology is confirmed or highly probable give a ten days course of penicillin or erythromycin.

- **Penicillin:**

(1) Oral penicillin for the first 4 hours to test for penicillin allergy. Start with 5 mg, double the dose every ½ hour, and give 7 doses. If no allergic reaction was observed start I.M penicillin.
(2) I.M. penicillin: There are several methods for giving i.m penicillin. Any method can be applied.

A practical method is as follows:

i. Start with three days course of benzyl penicillin - procaine penicillin mixture. Dose = 50-100 thousand units / kg /day, divided.
ii. Then give a single I.M injection of benzathine penicillin 300,000 units for ages 2-5 years, 600,000 units for ages 5-8 years,. 1,200,000 units if older than 8years.

❖ **Other antibiotics:**

1-amoxicillin once-daily dosing (750 mg fixed dose or 50 mg/kg, maximum 1 g) given orally for 10 days OR amoxicillin plus clavulanic acid for 10 days

2-Erythromycin: If the child is allergic to penicillin, give erythromycin in a dose of 50 mg/kg per day for 10 days. The addition of sulphonamide potentiates the action of erythromycin.

3- Azithromycin: 12 mg/kg/day as single dose for at least 5 days.

4-cephalosporin (cephalexin or cefadroxil) for 10 days.

NB. Cefotaxime should not be prescribed for patients with acute tonsillopharyngitis, because it is ineffective, is costly, and predispose to bacterial resistance to cefotaxime.

Prevention:

1. Avoid overcrowded places and school class.
2. Avoid contact with patients.
3. In patients with rheumatic fever, benzathine penicillin injection is given to prevent strept. pharyngitis and rheumatic recurrences. The above doses of benzathine penicillin are given every two weeks till the age of 20 years or older.
4. Removal of the tonsils does not reduce the frequency or complications of pharyngitis.

Indications of tonsillectomy:

1-Severe obstruction of airways
2-Tonsillitis more than six times in the last year.
3-Chronic tonsillitis e.g. chronic enlargement of tonsillar lymph nodes or chronic hyperemia of anterior pillars.
4- Peritonsillar abscess.

LARYNGEAL OBSTRUCTION

Manifestations:

A- Stridor: This is a harsh, crowing, inspiratory noise.

i. *In mild obstruction*, stridor is absent during sleep or rest and appears only after disturbing the infant, crying or on effort.

ii. *Moderate and severe degrees of obstruction* are dangerous and are characterized by:

1. Stridor is loud, heard during rest, and may be heard also during expiration.
2. Inspiratory retractions; suprasternal, supraclavicular, intercostal and subcostal.
3. Restricted expansion of the chest during inspiration.
4. Increasing respiratory and heart rates.
5. Restlessness, passing to lethargy (CNS hypoxia).
6. Cyanosis is an alarming sign.
7. Reduced arterial O_2 and increased arterial CO_2 tensions.

iii. If laryngeal obstruction is nearly complete, stridor may not be heard.

B- Other manifestations of laryngeal disease: These may be present in addition to stridor.

1. Voice changes e.g. hoarseness, aphonia.
2. Croupy cough which has a brassy or barking quality.

C. Fever: in cases caused by viral or bacterial infection.

Etiology:

A- Congenital:

a- Congenital laryngeal obstruction (laryngomalacia).
b- Other causes, as vascular ring.

B- Acquired:

1. Infections: viral, and bacterial (this is called croup).
2. Foreign body aspiration.
3. Spasm: Acute spasmodic laryngitis (midnight croup), and tetany (laryngismus stridulosa).
4. Trauma: natal, postnatal, surgical
5. Laryngeal tumors: polyps, others.

- Laryngeal obstruction is more common in infants and young children due to : small size of larynx , soft cartilage , loose submucosa and rich nerve supply.

Congenital Laryngeal Stridor (Laryngomalacia)

- The onset is after the first few days of life and more common in males.
- It is caused by abnormal flaccidity of the laryngeal cartilages of the epiglottis.
- Most cases are mild, the stridor may not be heard while the infant is asleep or at rest, and appears when the infant lies on back or is disturbed.
- More severe cases may result in feeding difficulties e.g. frequent choking and mild aspiration.
- Spontaneous cure within the first one or two years of life. Some children continue to have stridor with respiratory infections.

Differential Diagnosis:

Congenital web or polyp, birth trauma to larynx, aspiration of debris, during birth, neonatal tetany, macroglossia, hypoplasia of the mandible, and congenital goiter.

Treatment: Prone position, slow feeding, 0.3% of affected infants need tracheostomy.

Laryngeal Foreign Body

- Age: common between 2-4years.
- History of foreign body aspiration is usually present.
- This is associated with choking, hoarseness, croup and croupy cough.
- The foreign body may be vegetable or non-vegetable. It may be radio-opaque or translucent .
- Bronchoscopy is needed for diagnosis and removal of the foreign body.

Acute Viral Laryngitis

Age: 3 months – 5 years,
More common in males, and in cold seasons.

Etiology: About ¾ of cases are caused by parainfluenza viruses, and the remaining ¼ by adenoviruses , respiratory syncytial virus, influenza measles , herpetic , …etc. Positive family history in 15% of cases. It tends to recur in the same child.

Clinical picture:

- Gradual onset and course.
- 1-2 days of mild nasopharyngitis followed by hoarseness and croupy cough.
- Laryngeal obstruction is mild except in young infants in whom it may be severe.
- Mild to moderate fever may be present.
- The throat is inflamed and red (viral pharyngitis).

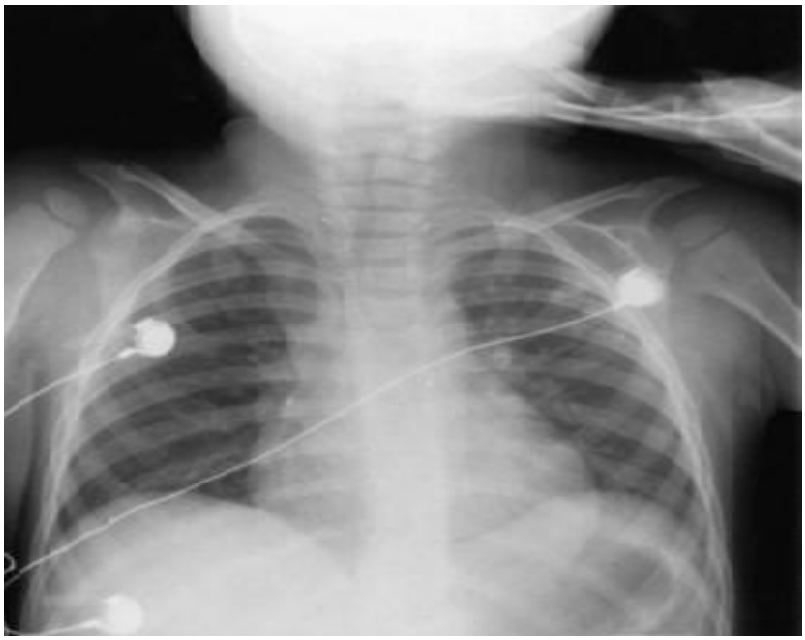
Viral laryngotracheobronchitis

A common disease.

Etiology: As viral laryngitis, the infection involves, in addition, the trachea, bronchi, and bronchioles.

Clinical Picture:

1. The patient has moderate to high fever.
2. Signs of laryngitis and laryngeal obstruction.
3. Signs of bronchial obstruction e.g. wheezy respiration.
4. Dyspnea is marked (inspiratory and expiratory).
5. Chest examination reveals rhonchi and crepitations.
6. Course is prolonged for 1-2 weeks.
7. Secondary bacterial infection may occur.
8. Recurrence occurs in young children.



Radiograph of an airway of a patient with croup, showing typical subglottic narrowing (steeple sign).

Acute Bacterial Laryngitis

This occurs as a complication of streptococcal pharyngitis tonsillitis or scarlet fever.
It combines the clinical picture of the original streptococcal infection together with the signs of laryngitis and mild croup.

Acute Epiglottitis

Age: 2-7 years.
More common in males.

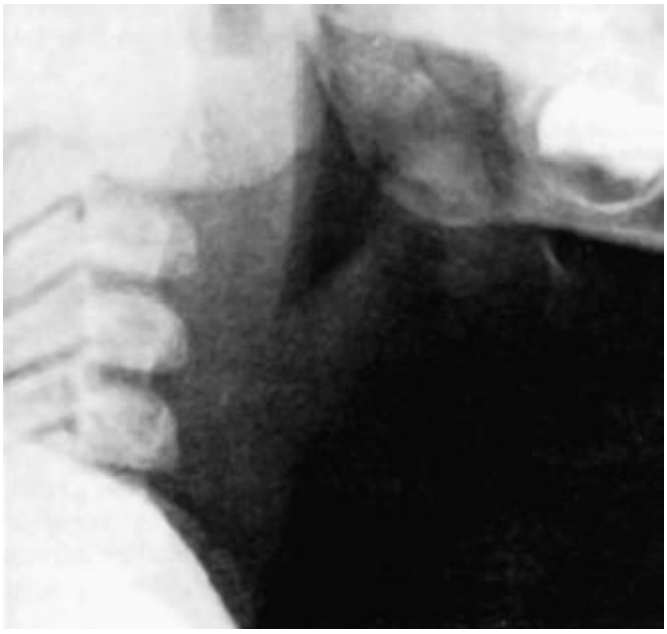
Etiology: Bacterial, almost always Hemophilus influenza bacillus type b (Hib). Rarely other bacteria e.g., streptococci., pneumococci, or staphylococci.

Clinical picture:

- Sudden onset and rapidly progressive signs of severe laryngeal obstruction.
- Temperature is high and Toxemia is marked.
- Posture of the patient is useful in diagnosis. The young child held by his mother, sits or lies with extended neck. The older child sits tripode, leaning forward with open drooling mouth and slightly protruded tongue. Respiratory distress and dyspnea increase in the supine position.
- Dysphagia is present.
- Severe cases are fatal.
- An ENT specialist should be called urgently if epiglottitis is suspected.

- Pharyngeal examination is dangerous because patients may get fatal laryngeal obstruction and cardiac arrest as a reflex following pharyngeal exam or even head titling. It shows: copious mucus and saliva, and large swollen red epiglottis.
- Lateral neck X-ray shows the swollen epiglottis.
- Blood picture shows PMN leukocytosis.

A physician skilled in airway management and use of intubation equipment should accompany patients with suspected epiglottitis at all times.



Lateral roentgenogram of the upper airway reveals the swollen epiglottis (thumb sign).

Diphtheritic Laryngitis

- Most cases are secondary to faucial diphtheria with the characteristic membrane.
- Moderate fever. Moderate toxemia with gray face
- Progressive laryngeal obstruction
- Tonsillar lymph nodes markedly enlarged.
- Almost always there is a history of defective vaccination against diphtheria.

Treatment of infective croup:

- Mild viral or bacterial laryngitis can be treated at home
- Other cases should be hospitalized because of possible need for intubation or tracheotomy.

- Hydration: plentiful fluids e.g oral warm sweetened fluids or I.V. infusions.
- Analgesic, anti-inflammatory, and antipyretic e.g ibuprofen 20 mg/kg/day.
- Sedatives should be avoided.
- Inhalation of hot steam medicated with Tr. Benzoin Co or inhalation of nebulized warm water for 5-15 minutes.
- This is followed by continued use of warm or cool humidification near the child's head for 2-3 days.
- Inhalation of 3/1000 solution of epinephrine is useful of laryngotracheobronchitis.
- Subcutaneous adrenaline, 0.1mg/10kg.
- Careful observation for need of:
  - **a-** Oxygen, and
  - **b-** Digitalis for heart failure.
- ENT consultation for hospitalized cases.
- Tracheostomy or intubation is needed in all cases of acute epiglottitis and may be in other cases with severe obstruction.

Antibiotics:

1. Erythromycin orally 50-100 mg/kg/day to treat strept. laryngitis and to prevent bacterial superinfection in cases of laryngotracheobronchitis.
2. Ampicillin + chloramphenicol or cefotaxime , for ten days to treat acute epiglottitis .
3. In cases of laryngeal diphtheria we give antidiphtheritic serum and penicillin. Antidiphtheretic serum is given in a dose of 50 to 100 thousand units.

Dexamethasone: Orally or IM may be useful in allergic cases or cases with severe obstruction with a dose of 0.3mg/kg, once.

Acute Spasmodic Laryngitis (Mid-night croup)

- Age: 1-3 years.
- Child goes to bed with mild nasopharyngitis with or without hoarse voice. About midnight he wakens with severe loud croup and struggles for breath.
- The attack subsides spontaneously after a few hours.
- By daytime, he is well except for some hoarseness.
- The next night he may develop a similar but less severe attack.
- The patient is afebrile.
- The condition tends to recur in the same child.
- It reflects the response of the nervous or allergic child to a mild viral laryngitis.

Treatment:

If seen during the attack, inhalation of medicated water vapor from a vaporizer or nebulizer , SC adrenaline and IM dexamethazone .

To prevent the attack on the second or third night:

1. Treat viral nasopharyngitis.
2. The patient is given Promethazine HCl (Phenergan) 0.5 mg/ kg. Before bedtime.

Hypocalcemic Laryngeal Spasm (Laryngismus stridulosa)

- This occurs as a manifestation of hypocalcemic tetany in Vit. D deficiency rickets.
- The infant usually has manifestations of rickets and there may be carpopedal spasm or tetany.
- Croup occurs by night or early morning.
- It can be precipitated by disturbing the infant or irritation of the larynx.
- Treatment: 5 ml of 10% calcium gluconate solution slowly IV relieves the laryngeal and carpopedal spasm. The usual treatment of rickets is then given.

ACUTE TRACHEOBRONCHITIS

Definition: Acute catarrhal inflammation of the trachea and bronchi.

Etiology:

1. Most cases occur in association with viral nasopharyngitis and are caused by rhinoviruses and other viruses. Viral tracheobronchitis is also caused by such specific viral infections as influenza, measles and German measles.

2. Bacterial cases may be caused by:
   - Pneumococci, and streptococci, on top of viral tracheobronchitis.
   - Primary bacterial infection e.g. scarlet fever, pertussis, diphtheria and typhoid.

Bacterial tracheitis due to staph. aureus presents with severe stridor, high fever, high, toxicity and severe dyspnea with copious, purulent secretions seen below the glottic opening and included in differential diagnosis of acute epiglottitis.

Predisposing Factors: These include malnutrition, rickets, exposure to cold, allergy, chronic upper respiratory diseases (adenoids, tonsils, and sinuses) passive smoking and heart disease (congestive heart failure)

Clinical Picture:

1. Cough, dry at first, then becomes loose later on Infants swallow their sputum. Cough may be paroxysmal and distressing.
2. Fever mild to moderate up to 39C. Significant malaise may be present.
3. Low substernal pain and chest discomfort may be complained of.
4. Dyspnea with or without wheezing occasionally present in infants.
5. Vomiting may occur and may contain swallowed sputum.

Chest Signs:

1. Breath sounds may become harsh.
2. Rhonchi may be heard especially in infants with or without few scattered crepitations.
3. X-ray chest: Free or shows prominent bronchovascular shadows.

Course And Prognosis:

1. The condition gradually subsides within 7-15 days.
2. Complications occur in malnourished or rachitic patients e.g. otitis media, sinusitis, or bronchopneumonia.
3. Recurrent acute bronchitis occurs if predisposing factor is not treated.

Treatment:

1. Bed rest with frequent change of position in infants to facilitate drainage of mucus.
2. Warmth.
3. Adequate fluid intake especially warm fluids.
4. Antipyretic e.g. Paracetamol 50 mg/kg/day, or Ibuprofen 20 mg/kg/day.
5. If cough is distressing and interfering with sleep or feeding a cough suppressant may be prescribed.
6. If bacterial super infection is present an antibiotic is given.
7. Mucolytic e.g. Carobcysteine or Ambroxol syrup (one ml /10 kg) 4 times daily.
8. Expectorant e.g. Syrup Ipecac (1 ml /10kg) 4 times daily.

ACUTE BRONCHIOLITIS

Definition: Acute viral infection of the bronchioles during the first two years of life, causing their obstruction.

Etiology:

- Respiratory syncytial virus (RSV) causes more than 50% cases. Other causative viruses include parainfluenza, adenoviruses, human metapneumovirus, other viruses and mycoplasma.
- The source of infection is usually an older family member with mild viral resp. illness.

Season: Winter and early spring

Age: Highest incidence by age 2-10 months, rare below 2months and during the 2nd year of life.

Predisposing factors:

- Subject: male, preterm, narrow airways,
- Environment (preventable): Exposure in day – care or crowded rooms, tobacco passive smoking, non –breast feeding, low socioeconomic state.

Pathology:

1. Diffuse partial bronchiolar obstruction due to bronchial wall infiltration, mucosal edema and lumenal debris and mucus.
2. Generalized obstructive emphysema.
3. Scattered areas of collapse and consolidation.

Clinical Picture:

1. Common cold like manifestations of several days, followed by progressive development of mild to severe wheezing dyspnea and cough.
2. There is mild to marked tachypnea, working alae nasi, chest retractions, irritability and feeding becomes difficult.
3. Dehydration, cyanosis and exhaustion may occur in severe cases.
4. Chest exam. shows generalized obstructive emphysema i.e. signs of bronchial obstruction plus signs of emphysema.

Course:

- In mild cases the disease subsides in 1-3 days.
- In severe cases peak of suffering is by the 2nd or 3rd day, then the condition gradually subsides in 3-10 days.
- Recurrence is rare, but may occur for another single infection.

Complications:

1. Hypoxia early in the course, proportionate to the degree of tachypnea.
2. Respiratory acidosis also occurs in patients with respiratory rates more than 60/minute.
3. Apneic spells in infants less than 6 months age.
4. Bacterial super infection causes bronchopneumonia.
5. Congestive heart failure in patients with congenital heart disease.
6. Death in 1% of cases from respiratory failure, C.H.F., dehydration or pneumonia.
7. Bronchial hyperreactivity and bronchial asthma may occur during the following 5-10years.

Investigations:

1. Chest X – ray shows marked translucency with or without scattered areas of increased density.
2. Blood picture shows normal leukocyte profile.
3. Reduced arterial oxygen saturation and Oxygen tension (PaO2) and/or increased arterial carbon dioxide tension (PaCO2) in severe cases.
4. The causative virus may be detected in nasopharyngeal secretions.

Differential diagnosis:

1. Bronchial asthma (recurrence, allergic history, eosinophilia, response to bronchodilators).
2. Bronchopneumonia (fever, primary cause, crepitations, PMN leukocytosis).
3. **From other causes of wheezing in infancy**

Congenital lung diseases
Chronic lung diseases (bronchopulmonary dysplasia
interstitial lung disease,
mucociliary disorders
Immunodeficiency states
Aspiration syndromes
Others: heart failure, anaphylaxis, foreign body

Treatment:

- Outpatient treatment for mild cases.
- Hospitalization for severe cases. ((hypoxia, inability to take oral feedings, extreme tachypnea).
- Risk factors for severe disease include age <12 wk, preterm birth, or underlying comorbidity such as cardiovascular, pulmonary, or immunologic disease

1. Hydration, oral, IV in severe cases.
2. Humidified oxygen is the most important line of treatment.
3. Racemic adrenaline inhalation
4. Nebulised hypertonic (3%) saline
5. Antibiotics for secondary bacterial infection.
6. Aspiration of secretions and assisted ventilation may be needed.
7. Digitalis for heart failure.
8. Bronchodilators and corticosteroids (as for asthmatic paroxysm) use are controversy
9. Ribavirin aerosol is useful for severe cases, but costly. Indicated for: Cases with ARF, co-existing disease e.g. congenital heart disease and cases with low immunity.

Prevention:

Reduction in the severity and incidence is possible through the administration of RSV intravenous immunoglobulin considered for infants with chronic lung disease, and congenital heart diseases.

BRONCHIAL ASTHMA

Definition: Asthma is a chronic inflammatory disease of the airways in susceptible individuals . It is characterized by airway hyper-responsiveness to a variety of stimuli , reversible airflow obstruction, and bronchospasm, variable and recurring symptoms include episodes of wheezing, coughing, chest tightness, and shortness of breath.

Epidemiology:

- Prevalence: 5-10 % of children.
- Sex: incidence before puberty, boys to girls = 2: 1 and later on it becomes gradually equal.
- Family history of allergic disease (respiratory, skin, ENT, eyes) is positive in 50% of asthmatic children.
- Age of onset:
  - ✓ 30% < 1 year,
  - ✓ 50% < 2 years, and
  - ✓ 80 – 90% < 5 years of age.

Etiology And Pathogenesis: This includes:

A: Genetic predisposition:

Asthma is caused by the interaction of many genetic loci in chromosomes 5, 6, 11, 14 and other chromosomes.

B: Anatomic characteristics of early childhood:

1. Relatively narrow bronchial lumen.
2. Increased number and activity of mucus secreting glands.
3. Weak collapsible bronchial walls.

C: Abnormal characteristics of allergic child:

1. Excessive production of Ig E in response to antigen exposure (i.e., Atopy). There is an increased amount of Ig E molecules on the surface of mast cells and basophils present in bronchial mucosa.
2. Irritability or hyperreactivity of bronchi (BHR) to various stimuli (physical or chemical).
3. Autonomic imbalance in the form of increased cholinergic and decreased adrenergic function at the level the bronchi.

D: Exposure of the child to non antigenic and/or antigenic stimuli:

Non antigenic stimuli: are common causes of asthma in the 1st five years of life.

1. Viral infections: RSV, parainfluenza, rhinovirus, and influenza virus.
2. Physical irritants: smoke, fumes, dust, strong odors, and cold air, and drinks.
3. Chemical irritants: kerosene, insecticides, and sulfur dioxide.
4. Strenuous exercise especially running.
5. Emotional stress.

Antigenic stimuli: are common causes of asthma in older children.

1. Inhalants (aeroallergens): the most common allergens are: house dust mite, animal dander (cats, dogs, birds, cattle, sheep), pollens, fungi, insect parts and house dust.
2. Food: Milk, fish, eggs…etc.
3. Drugs: penicillin, aspirin and some non-steroidal anti-inflammatory drugs.

E: The result of this:

1. Non antigenic stimuli irritate and damage bronchial epithelium causing the release of neuropeptides and cytokines which cause inflammation of bronchial tissues.
2. Allergens binds to the numerous Ig E molecules present on the surface of mast cells and basophils resulting in the release of mediators → allergic bronchitis. Some of the important mediators are: leukotrienes, histamine, prostaglandine F, eosinophil chemotactic factor, neutrophil chemotactic factor, nitric oxide and eosinophil cationic protein.

F: Bronchitis will cause:

1. An increase of the already present BHR → perpetuation of disease on exposure to stimuli.
2. Partial bronchial obstruction through:
   - Bronchospasm,
   - Mucosal edema,
   - Increased secretion and desquamated epithelial debris in bronchial lumen,
   - Bronchial wall thickening by inflammatory cells and by collagen deposition under mucus basement membrane (remodeling).

G: Bronchial obstruction results in air trapping → generalized obstructive hyperinflation, scattered areas of atelectasis→ labored breathing.

H: Hyperinflation being non uniform + Atelectasis areas → ventilation/perfusion mismatch → Decreased Pa O2 and increased P a CO_2 ± respiratory and/or metabolic acidosis.

A

Genetic predisposition

B

Young Age:
- ↓ Bronchial lumen
- ↑ Mucus production

C

Abnormally allergic child:
- ↑ Ig E production
- BHR
- Increased neurogenic bronchoconstrictive activity

D

Stimuli
- Non antigenic
- *Antigenic*

E

Bronchospasm
Mediator Release
Allergic Bronchitis

F

Bronchitis causes:
- Increased BHR
- Bronchial obstruction due to:
  Spasm, Edema, Secretions & debris, and Thick bronchial walls

G

Bronchial obstruction results in:
- Hyperinflation.
- Labored Breathing
- Altered gas exchange

Pathogenesis of bronchial asthma

Pathology:

1. ***Bronchi***: Diffuse partial obstruction of small (< 2mm lumen) and large bronchi caused by bronchospasm, edema of the mucosa, increased mucus secretion and cellular debris, and thickened walls (due to infiltration by inflammatory cells and deposition of collagen under the basement membrane i.e., remodeling).

2. ***Alveoli:***
   Generalized hyperinflation due to air trapping.
   Scattered small areas of collapse may be present.

3. ***Complications may be found***:
   Bronchopneumonia
   Pneumothorax
   Massive collapse of one lobe or the whole lung.

Clinical picture:

The asthmatic paroxysm:

- It is characterized by gradual or sudden onset of mild to severe chest wheeze and cough.
- The majority of attacks are mild to moderate.
- Gradual development of asthmatic paroxysm over a few days is triggered by exposure to irritants or allergens
- The paroxysms are more frequent and are more severe at night and early morning hours.
- Cough may be mild or severe, and in some case it may be present without evidence of bronchial obstruction i.e., cough variant of asthma.

In mild attacks, the condition is represented by:

- Complaint of intermittent chest wheeze or rattle.
- Slight increase in respiratory rate (RR)
- End expiratory rhonchi.
- PEFR and O2 saturation are still normal.

As the paroxysm gets more severe (moderate attack), there will be:

- Progressive increase in RR.
- Difficulty in feeding, chest wheezes, and rhonchi become louder and prolonged with the accessory muscles of respiration working.
- There will be retraction and hyperinflation.
- Both PEFR and O2 saturation start to fall.
- In severe paroxysm, there will be:
- Dehydration,
- Fatigue,
- Refusal of feed,

- Cyanosis,
- Impaired consciousness, and
- O2 saturation is low.

Chest signs during asthmatic paroxysm :

Inspection : Increased antero-posterior diameter of chest, rapid shallow breathing with intercostal and subcostal retraction, apex of the heart may become invisible
Palpation: diminished chest expansion bilateral, central trachea, diminished TVF, palpable rhonchi allover the chest
Percussion : hyperresonance allover the chest
Auscultation : vesicular breathing with prolonged expiration, sibilant (±sonorous) rhonchi is present mainly expiratory ± non consunating crepitation, decreased vocal resonance

The asthmatic paroxysm usually subsides in 1-2 hours or days, rapidly or gradually, spontaneously or after treatment. Some degree of bronchial obstruction usually persists for several days especially in cases associated with respiratory infection.

Status asthmaticus:

- Sometimes the child suffers from a severe prolonged paroxysm that is not rapidly responsive to bronchodilator therapy, a condition called status asthmaticus or life threatening asthma.
- In this case, excessive amounts of thick bronchial secretions share in the obstruction of the bronchi.
- The child is cyanotic, dehydrated, fatigued, and not alert.
- Prominent hypoxia and hypercarbia are present and the mortality rate is about 1%.

Severity of Asthma Paroxysm

Six criteria or more of the following are needed to classify asthma paroxysm:

Criteria	Mild	Moderate	Severe
RR (% of normal)	↑ up to 30%	↑ 30-50%	↑ > 50%
Alertness	normal	agitated	↓ consciousness
Feeding	normal	difficult	refused
Speech (fatigue)	normal	phrases	words
Accessory Ms use.	no	sternomastoid	alae nasi also
Retraction	no	+	++
Wheeze/rhonchi	end expiratory	throughout	Expir./ inspir.
Dyspnea	no	on exertion	At rest
Cyanosis	no	no	+/-
Hyperinflation	no	no	+++
Dehydration	no	+	++
O2 saturation (%)	>95 normal	95-91	< 91
PEFR/FEV1 (%)	> 80 normal	80-50	< 50

RR = Respiratory rate.

Course And Prognosis:

1. **Recurrence:**
   - It is a characteristic feature of the disease.
   - Its rate varies from one attack per day to once per a few months.
   - Adequate prevention of exposure to triggering stimuli reduces the rate of recurrence and the severity of individual paroxysms.

2- Ultimate remission:

- It occurs as the child grows.
- 50 – 60% of infants and preschool children who were well treated during their active illness cease to wheeze by the age of 4-6 years of age. They are sometimes called transient wheezers.
- School children with asthma tend to have less frequent and less severe paroxysms as they grow older, and by puberty one third of patients become completely free, another third of children markedly improve and the remaining third will still suffer.

3- Risk of having status asthmaticus:

An increased risk occurs in the following:

- Inadequate response to one-hour bronchodilator therapy.
- Severe asthmatic paroxysm at present time.
- Past history of severe attacks or status.
- Chest X- ray showing pneumothorax or pnemomediastinum.
- Delayed use of steroids to treat an attack.
- Poor compliance with therapy (child and/ or family)
- Patients with steroid dependent asthma
-

Complications:

- During the attack: Bacterial super infection, pneumothorax, and collapse of part or all of the lung and respiratory failure.
- Chronic asthma ➔ growth failure, chronic bronchitis, bronchiectasis, cor-pulmonale, emphysema, chest wall deformity
- Badly controlled asthma can have an adverse effect on your quality of life. The condition can result in:
  fatigue
  underperformance or absence from school or work
  psychological problems including stress, anxiety and depression.

Associated diseases may cause difficulty in asthma control:

- Allergic rhinitis and sinusitis ± infection.
- Gastroesophageal reflux (GER)

Investigations:

- Chest x-ray: Hyper inflation ± complications e.g., pneumothorax, collapse ..etc.
- Pulmonary function tests (PFTs): PEFR, and FEV1 = < 80% of predicted values indicating bronchial obstruction
- Blood gases: O2 saturation ≤ 95%, ↓PaO2, and↑ PaCO2.
- Bronchial challenge tests: By exercise or inhalation of metacholine to detect mild or suspicious cases.
- Sputum eosinophilia.
- Blood eosinophilia: Of 5-10% (To be differentiated from parasitic infection).
- Skin tests are positive for causative allergens in older children, (they are negative in most children below 5 years)
- Serum Ig E: It is high in most of asthmatic infants and children.
- Increased nitric oxide in exhaled air and sputum.

Diagnosis:

It depends on:

1. Characteristic symptoms and signs and recurrence ± associated upper resp. allergy or skin allergy ± positive family history of allergy.
2. Laboratory investigations are confirmatory and measure severity.
3. In difficult cases, PFTs (FEV1 or PEFR) will be reduced by more than 15%, after five minute exercise and if they are reversed to normal value after beta agonist administration, the case is definitively asthma.

Differential Diagnosis:

1. Causes of stridor (laryngeal or extra thoracic trachea obstruction).
2. Other causes of wheezy chest, e.g., bronchiolitis or FB aspiration.
3. Pneumonia and TB.
4. Psychogenic cough or frequent sighing (disappear during sleep).

Treatment of Asthma Paroxysm

This includes:

1. Hospital admission if indicated.
2. Treatment of moderate – severe paroxysms.
3. Treatment of mild paroxysms.

1-Hospital Admission

Indicated for:

1. Moderate – severe attacks.
2. Status asthmaticus.
3. Child liable to get status asthmaticus.
4. Acute complication (bronchopneumonia, pneumothorax, massive collapse…).

2-Treatment of moderate – severe paroxysm:

A- General supportive:

1. *Oxygen inhalation*: 40 % humidified, to keep arterial oxygen saturation > 95 %.
2. *Hydratio*n:
   - Oral warm sweetened fluids, milk fruit juice
   - Intravenous for: 1- Status asthmaticus, and 2-Inadequate oral hydration, 40 – 50 ml / kg during first 12 hours (2 parts 5% glucose + one part saline)
3. *Antibiotics* for:
   a) Febrile cases (>38.5).
   b) Bronchopneumonia.
   c) Status asthmaticus. (N.B. Avoid penicillin)

4. Sodium bicarbonate infusion: For cases of status asthmaticus if arterial PH < 7.30 (dose 1-2 mEq. / Kg.)

B-Specific treatment using quick-reliever medications:

1) β_2 - adrenergic agonists:

A- Aerosolized β_2 adrenergic agonists

:

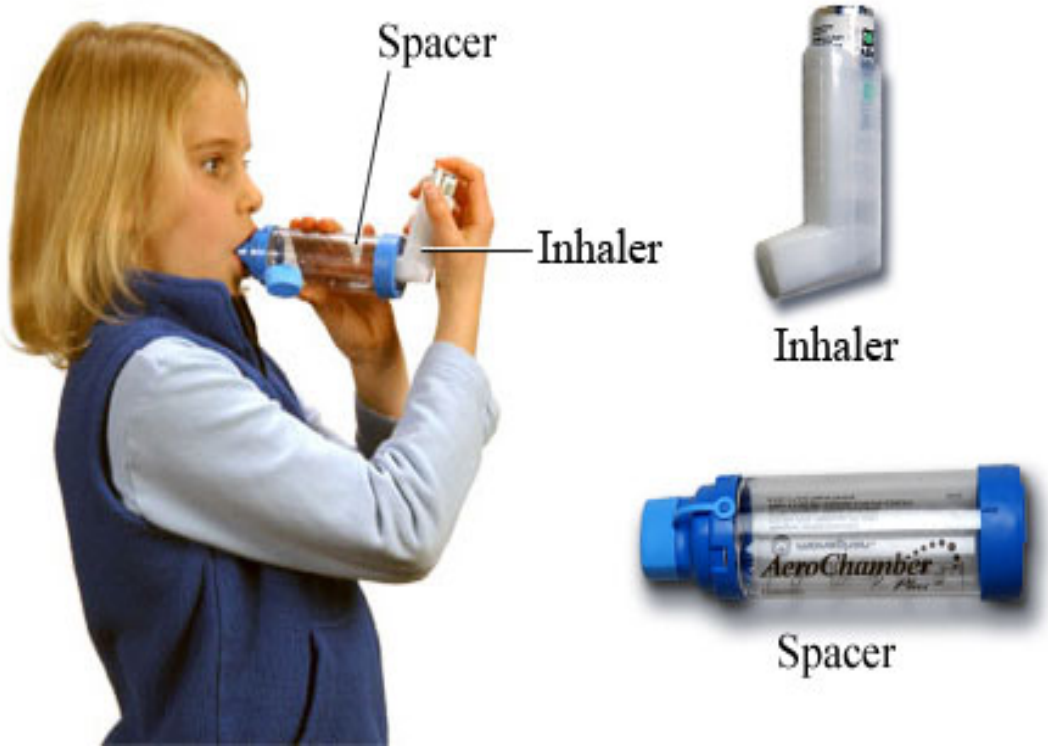
- ✓ One dose /20 minutes (2-3 doses are usually adequate).
- ✓ For cases of status asthmaticus or if response to 3 doses is inadequate repeated doses are given (max. = 6 doses) till the attack is controlled, then give one dose every 1-6 hour. β_2 agonists can be given either by :
  1. Nebulizer + mask: for any age (Salbutamol 0.15 mg / kg in 2 ml saline / dose (max 5mg/ dose).
  2. Metered dose inhaler (MDI) with a spacer (+/- a mask) is useful for school children. Salbutamol 4 puffs / dose, increase up to 8 puffs /dose if response in inadequate.

Jet nebulizer

ultrasonic nebulizer

*Nebuh*aler aerochamber

Some types of spacers



Use of MDI with spacer

B- Subcutaneous β_2 agonists:

- ✓ They are indicated if aerosolized B2 agonists are not available or if response to them is inadequate or if child is not cooperable with aerosol.
- ✓ Drugs: Terbutaline, salbutamol or adrenaline.
- ✓ Dose: 1/1000 salbutamol, 0.1 ml / 10 Kg (max 0.4 ml) repeat every 20 minutes, [max. 3doses] if needed. Terbutaline and salbutamol can be given IM or SC.

C- IV terbutaline: may be given with caution in ICU, for cases of status asthmaticus.

2) Corticosteroid therapy:

- *For moderate paroxysm*: Give oral Prednisone ½ -2 mg / Kg / day (max 60 mg / day) divided in 3 doses, for 4 days. If needed give for another 3 days as a single dose after breakfast. You can stop without tapering.
- *For severe paroxysm or status asthmaticus* give parenteral drugs: e.g. IV or IM dexamethazone: 0.1 mg/Kg / dose, repeat / 6-12 hours or methyl prednisolone 1mg/kg q 6 hours if needed till dyspnea is relieved or up to 48h then give oral prednisone.

- *In steroid dependent patients or those with chronic asthma*, steroid therapy is continued in the least effective dose to control chest rhonchi. Steroid aerosol by MDI with spacer (+/- mask) e.g. fluticasone propionate, 2-4 puffs (50ug per puff) 6-12 hours. If this fails, oral steroids are given e.g. prednisone 0.1 – 0.5 mg / kg as a single dose after breakfast every day or every other day (EOD).

3) Anticholinergic aerosols: They are given if response to 3 doses of β_2 agonist is inadequate. Drug: ipratropium bromide;
By nebulizer, 250 ug / ml every 20 minutes for 3 doses, then every 2-4 h.
By MDI, 4-8 puffs / dose, every 20 minutes as needed.

4) Aminophylline: IV infusion: Given for cases of status asthmaticus, or if combined β_2 agonist (3 doses) and corticosteroids failed to control asthmatic attack.

- IV aminophylline diluted in 30 ml saline, rarely indicated 4 mg / kg / dose may be repeated after 6 hours (to be given by infusion pump).
- The presence of fever or viral infection necessitates reduction of the dose by 50 %.

5) Mechanical Ventilation: If any of the following occurs:

1. Failure of maximal pharmacological therapy including I.V beta agonist.
2. Exhausted child.
3. Impending respiratory failure (RF).
4. Respiratory or cardiac arrest.

Treatment of Mild Paroxysm

1. Beta-adrenergic agonist.
   - For 1 weeks.
   - Aerosol 2-4 puffs / 6 hours.or by nebulizer
2. Oral aminophylline may be added 2-4 mg / kg / 6h.
3. Oral prednisone:
   May be added if:
   - Otherwise not controlled
   - For patient with history of previous severe paroxysms.

4. Antibiotic for febrile cases.
5. Hydration by oral warm fluids.

Question: How can you treat status asthmaticus?

Classification of asthma severity

Intermittent asthma — A child is defined as having intermittent asthma if he or she has asthma with minimal symptoms and infrequent asthma flares. Specifically, children with intermittent asthma have the following characteristics:

- Symptoms of asthma occur two or fewer times per week
- Awakenings during the night due to asthma symptoms occur two or fewer times per month
- Asthma flares require oral steroids no more than once per year
- Asthma does not interfere with daily activities

Persistent asthma — Children with persistent asthma have symptoms regularly. There may be days when activities are limited due to asthma symptoms, and the child may be awakened from sleep. Lung function is usually normal between episodes, but becomes abnormal during an asthma attack. Persistent asthma can be mild, moderate, or severe.

The criteria that are used to determine a child's asthma severity include the number of days per week that a child has one or more of the following:

- Symptoms, such as cough, wheeze, or shortness of breath
- Awakenings during the night due to cough or wheeze
- Use of a bronchodilator (reliever medication)
- Symptoms that affect the child's ability to participate in normal activities
- The number of asthma flares per year that require treatment with oral steroids

Control of bronchial asthma

Children with persistent asthma need to take medication on a daily basis to keep their asthma under control, even if there are no symptoms of active asthma on a given day. Medications taken daily for asthma are called "long-term controller" medicines and function to decrease inflammation and to control chronic symptoms and to prevent asthma attacks.

Long-term controller" medicines include Inhaled corticosteroids, leukotriene modifiers (montelukast), long-acting beta-agonists and theophylline and a monoclonal anti-IgE antibody(Omalizumab) in some patients

Prevention of bronchial asthma

Asthma is a preventable disease which could be controlled.

1. Adequate treatment of asthma paroxysms by beta agonists and corticosteroids prevents early recurrence.
2. Reduction of exposure to various antigenic and non – antigenic stimuli is both very useful and difficult. It reduces the rate of recurrence and severity of asthma paroxysms.
3. Treat associated upper resp. allergy (URA) by antihistamines.
4. Treat Gastroesophageal reflux (GER) if present.
5. Control of persistent asthma.
6. Prevention of exercise induced asthma (EIA):
   - Beta agonist (aerosol) or
   - Fluticasone by MDL

   A single dose of any of them 15-30 minutes before exercise is adequate.
7. Annual vaccination against influenza virus may be useful in preventing asthmatic paroxysms precipitated by influenza virus infection.
8. Allergen Immunotherapy (Hyposensitization) :this is the administration of gradually increasing doses of one or two of causative allergens to the asthmatic atopic child. This may reduce the severity of atopic asthma when the child exposed later to the administrated allergens. It is indicated to control chronic asthma when all other measures failed. Allergens can be administrated by injection, inhalation or sublingualy.

Question: Asthma is a preventable disease. Comment.

Patient and family education:

The child and his parents should be educated about:

1. Nature of the disease, its course and prognosis.
2. The goal of management is to get normally-active child who sleeps well.
3. Role of various antigenic and non-antigenic stimuli.
4. Role of allergic rhinitis, sinusitis, and gastroesophageal reflux.
5. Side effects of asthma medications.

PNEUMONIAS

Definition:

Pneumonia is an inflammation of lung parenchyma.

Classification:

A- Etiologic :

1. Infectious pneumonia due to infectious agents: viruses , mycoplasma , bacteria, fungi or protozoa .
2. Non – infectious pneumonia is designated as pneumonitis. Pneumonitis may be due to chemical agents, hypersensitivity, and irradiation or may be idiopathic.

B- Anatomic:

- Up till now, diagnosis of the etiology of pneumonia is very difficult and costly. The methods to obtain infective material from lung tissue is still hazardous and not widely available.

- Therefore a more practical and easier method for classifying and treating pneumonia is identifying its anatomy clinically and by chest X-ray. Considering the prevalence of certain microorganisms at various ages will then make possible managing children with pneumonia.

- Two main types are identified: lobar pneumonia and bronchopneumonia.

(I) Lobar Pneumonia

Definition:

Acute pulmonary infection affecting all alveoli in one or more lung segments or in one or more lung lobes sparing other parts of the lungs.

Etiology:

A- predisposing factors:

1] Viral upper respiratory tract infection (common cold).
2] Malnutrition.
3] Cigarette smoking.
4] Chilling.
All these factors lower pulmonary defenses e.g. ciliary activity, and alveolar macrophages.

B- Causative microorganisms and age:

1] Most cases are caused by the Gram positive strept. pneumoniae (pneumococcus).
Age: at any age after the first month.
2] Some cases are caused by Gram negative Hemophilus influenza type b.
Age: below 5 y.
3] Some by Gram positive Staph. aureus.
Age:70% below 1 y, and 30% 1-3 y.
4] Viral infections.
Age: at any age.

C- Source of infection:

1] Pneumococcus and H. influenza bacillus:
   a] From patient with upper respiratory tract infection, rarely from patient with pneumonia.
   b] From contact carrier with the microorganism in his throat.
2] Staph.aureus from skin pyoderma of infant, or contact carrier of the organisms in his nose, or from maternal breast abscess.

D - Season: More in winter and spring.

Pathology:

A. Lungs: Affected areas pass by 4 stages: congestion, red hepatization and grey hepatization and resolution. Staph. pneumonia cases are characterized by the formation of lung abscesses and pneumatoceles (right side in 2/3 cases, bilateral ¼ cases, very rarely in the left side alone).
B. Pleura: Dry fibrinous pleurisy overlying affected lung. This may pass to serofibrinous pleurisy or empyema. Staph. aureus ➔ empyema in > 70 %, pyopneumothorax in 25 % of cases.

Clinical Picture:

An upper respiratory infection (nasopharyngitis) of a few days is followed by symptoms and signs of pneumonia.

A- Symptoms:

1] Sudden rise of temp. to 38.5 or more, with shivering in older children. Febrile convulsions may occur in susceptible patients.
2] Dyspnea and grunting.
3] Chest pain of pleuritic nature (local over the affected side or radiated to the other sites of the chest or to the abdomen).
4] Cough is an infrequent complaint at the onset.

B- General signs of pneumonia:

1] Characteristic triad of:
   a) Dyspnea with expiratory grunt.
   b) Inverted breathing rhythm.
   c) Inspiratory flaring of the nostrils (working alae nasi).
2] Fever 38.5 C or more.
3] Restlessness, anxiety, apprehension, may be drowsiness (toxic facies).
4] Toxic shock may be seen in staph. pneumonia.
*In mild cases,only tachypnea may be present.

C- Chest signs of lobar pneumonia:

1. During the first or second day of disease, one may detect diminished air entry over affected area; however, chest X- ray may show infiltrates.
2. Later on the signs of consolidation become evident.

Chest sings of consolidation

Inspection: normal shaped chest, central mediastinum
Palpation : diminished chest expansion on affected side, central trachea, increased TVF over the affected lobe, no palpable rhonchii
Percussion : dullness over the affected lobe
Auscultation : bronchial breathing over the affected lobe with increased vocal resonance (bronchophony). Pleural rub may be heard

3. When resolution starts, crepitations become prominent while other signs gradually fade.

Course And Prognosis:

1. In pneumococcal cases recovery may start towards the end of the first week. The initial occasional cough becomes more frequent, productive and may be blood tinged.
2. In untreated cases the mortality rate is 5-50 %.
3. H.influenza pneumonia has insidious onset and prolonged course of a few weeks.
4. Staph.pneumonia has a stormy course with delayed resolution after several weeks.
5. Antibiotic treatment hastens resolution and markedly lowers mortality. Despite treatment, the mortality rate is still around 30 % in Staph. pneumonia.

(II) Bronchopneumonia

Definition:

Acute bronchopulmonary infection of the bronchial tree, alveoli and interstitial tissue of the lung, affecting all segments in one or both lungs, and occurring as a complication of pre-existing bronchitis in a predisposed child.

Etiology:

A- Predisposing factors:

1. Factors lowering pulmonary defenses and resistance to infection e.g. P.E.M., rickets, gastroenteritis, tobacco smoke.
2. Certain viral and bacterial infections: measles, influenza, pertussis, and scarlet fever.
3. Bronchial asthma.
4. Aspiration of food or vomitus in cases of: cleft palate, gastroesophageal reflux, impaired consciousness, severe debility and swallowing dysfunction.
5. Congenital heart diseases with left to right shunt.
6. In neonatal pneumonia: prolonged rupture of membranes and prolonged labor.

B- Causative microorganisms:

1] Exclusively viral: RSV, influenza, parainfluenza, adenovirus and measles.
2] Exclusively bacterial: The most common organism differs according to the age of child:
   (a) In neonates, Strept. Group B and E. Coli.
   (b) Age 1-3 months, Strept. B and Chlamydia.
   (c) Pneumococcus is the most common after the age of 3 months.

(d) Staph. aureus in preschool age.
(e) Streptococcus-A and Mycoplasma, by school age.
(f) Oral anaerobes at any age in cases of aspiration pneumonia.

3] Mixed viral and bacterial superinfection.

C- Source of infection:

1. From patients or carriers (as before).
2. From oral flora in aspiration pneumonia.
3. Neonatal pneumonia from carriers, patients or vaginal flora.

Pathology:

1] Lungs show a mixture of lesions diffusely scattered in one or both lungs:

i- Bronchitis and bronchiolitis with variable degrees of obstruction and secretions.
ii- Alveoli: in some lobules they are consolidated, in others collapsed, and in others emphysematous.
iii-Inflammatory infiltrate of interstitial tissue.
iv-Lung abscess may be present.

2] Pleura: pleural effusion and empyema are frequent in 25 % of cases.

Clinical Picture:

A- An infant or child with bronchitis who is suffering from one of the predisposing factors.

B- Onset of bronchopneumonia is shown by development of :

1. Symptoms and general signs of pneumonia.
2. Variable degrees of wheezing, more marked in cases complicating asthma.
3. Cough is early and prominent and may be productive.
4. In neonatal pneumonia, manifestations of neonatal sepsis may be present.

C- Chest examination:

1- Marked dyspnea: increased RR, working accessory muscles of resp., & retractions.
2- Breath sounds: Harsh vesicular breath sounds allover, but if large areas of consolidation are present near lung surface, they cause tubular breath sounds here and there.
3- Added sounds:
i- Rhonchi: sibilant and sonorous, diffuse and most marked in asthmatic cases.
ii- Crepitations: numerous medium sized crepitations throughout the whole lung.

Course and Prognosis:

1. The disease is more severe, the course is more prolonged, complications more frequent and mortality rate higher than in lobar pneumonia.
2. Neonatal pneumonia has the highest mortality rate.
3. Recurrence of pneumonia indicates the continued existence of the predisposing factors.

Complications of pneumonia:

1. Distressing tympanitis (abdominal distension with gas).
2. Otitis media, is frequently seen at the onset of pneumonia in H. influenza pneumonia.
3. Pneumatoceles, empyema, pyopneumothorax and lung abscess.
4. Disseminated infection: Meningitis, pericarditis, peritonitis and arthritis.
5. Slow resolution over several weeks (H. influenza).
6. Heart failure.
7. Respiratory failure.
8. Pulmonary sequelae may complicate bronchopneumonia e.g. flaring of dormant pulmonary T.B., pulmonary fibrosis, and bronchiectasis.

Complications are more common in infants, in H. influenza and Staph. pneumonias and in bronchopneumonia.

Investigations for pneumonia:

A- Chest X-Ray postero-anterior and lateral.

This should be done in every case and repeated a few days later and if the case deteriorates.

1. Lobar pneumonia shows opacity of one segment, lobe or even one lung. Rapid progression with development of empyema, pyopneumothorax or lung abscess occurs in staph. pneumonia. In H. influenza pneumonia, unilateral opacity remains rather stable.

2. Bronchopneumonia shows patchy areas of opacity scattered in one or both lungs. The radio-opacities of pneumonia appear very early at its onset and may persist for 3-6 weeks following clinical recovery.

X- ray chest cannot differentiate between viral and bacterial pneumonia.

B- Blood Picture:

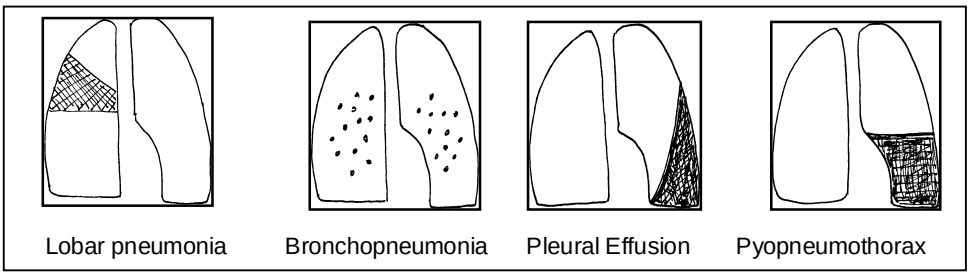
1. CBC shows leukocytosis.
2. Neutrophilia more common in bacterial cases.
3. Lymphocytosis in viral cases.
4. Eosinophilia in Chlamydia pneumonia.

C- Etiologic Diagnosis: This is difficult and costly:

Combined viral and bacterial infections coexist in 40 % of cases.

1. Demonstrating microorganisms in bronchial secretions, lung aspirate, pleural fluid and blood by Gram stain or culture. Bacteremia is present in 10 – 20 % of untreated cases. One or more days are needed.
2. Rapid bacterial antigen detection in bronchial secretions, pleural fluid or in urine, only few hours are needed.
3. High A.S.O. titer in strept. pneumonia.
4. Viral diagnostic studies if available:antigen detection ,PCR, serologic tests and culture.
5. Tuberculin test (if antibiotic resistant or delayed resolution).

D- Blood Gases: O2 saturation, Pa O2, Pa CO2, and pH.



Chest X – ray (postero-anterior) in lobar pneumonia, bronchopneumonia, pleural effusion and pyopneumothorax

Diagnosis of Pneumonia:

1. WHO recommends that pneumonia is diagnosed if : Temp. > 38.5 C. + respiratory rate > 50 per minute in an infant+ retractions.
2. Symptoms, chest signs and X-ray.
3. Causative microorganisms: From clinical situation and investigations.

Differential Diagnosis

1- Acute tracheobronchitis.
2- Acute bronchiolitis.
3- Status asthmaticus.
4- Tuberculous bronchopneumonia.
5- Meningitis (versus upper lobe pneumonia).
6- Acute abdomen (versus basal pneumonia).

Treatment of Pneumonia

Hospital care for:

1- Infants (first year of life).
2- Severe distress: (Respiratory rate increased by > 50 %, marked retractions, patients not alert and/or cyanosis).
3- Empyema or significant effusion.
4- Possible Staph. etiology.
5- Inadequate home care.

General:

1- Bed rest, with frequent change of the position in bed for infants and young children.
2- Antipyretic and analgesic for pleural pain.
3- Aspirate oral secretions and clear nasal obstruction.
4- Adequate fluid intake, soft easily digested diet to avoid tympanitis. Intravenous fluids may be needed in infants.
5- If abdominal distension is present, give prostigmine 1/2- 1 ml I.M. or S.C.
6- Oxygen, if there is severe dyspnea, even in absence of cyanosis, or if O2 sat.< 90 %.
7- Deep tracheal suction for patients with ineffective cough.
8- Bronchodilators for asthmatic bronchopneumonia.
9- Mechanical ventilation for respiratory failure. Digoxin for heart failure.

Drainage Of Pleural Fluid:

1- Small amount of effusion will absorb spontaneously.
2- Significant amounts should be aspirated as they collect.

3- Tube drainage is needed for rapidly collecting effusion and for empyema.
4- Consult a thoracic surgeon for cases with chronic empyema.
5- Pleural fluid pH< 7.2 or glucose < 50 mg/dl or lactic dehydrogenase >1000 µ / L.

Antibiotic therapy for pneumonias

This is indicated if viral etiology cannot be proved.

Parenteral antibiotics are obligatory for at least one week in uncomplicated pneumonia.

For cases complicated with empyema at least 3 weeks of parenteral antibiotics are needed.

Type of antibiotic depends on the age of patient, the most commonly causative bacterium in this age and the type of pneumonia (lobar or bronchopneumonia), chest X-ray and the results of rapid antigen test or culture.

A) If clinical manifestations, X-ray or rapid antigen test suggest a specific microorganism, start treatment with antibiotic effective against it e.g.

- Penicillin or ampicillin or amoxicillin or first generation cephalosporin for pneumococcus, streptococcus A or B.
- Oxacillin or first generation cephalosporin for Staph. aureus.
- Ceftrixone,cefotaxime or cefuroxime sodium for H.influenza.
- Gentamicin or amikacin for E.coli.
- Amoxicillin + Azithromycine for Mycoplasma peumonia.

B) If this is not possible, start empiric antibiotic therapy:

I. Infants less than 2 months:3 rd generation cephalosporins± amoxicillin.
II. Infants aged 2 months – one year:
Give amoxicillin + gentamicin, or 3 rd generation cephalosporins for any type of pneumonia, for 2 – 3 weeks.
Patients aged 1 – 5 years :
Give amoxicillin for 4 days. Then,

C) If the response is good it is most probably pneumococcal pneumonia.
Continue with ampicillin for 10 days.

D) If the disease rapidly progresses, repeat chest X-ray, Staph pneumonia is most probable, therefore continue with ampicillin + gentamicin for 3 weeks.

E) If response is slow and slight, this is probably H. influenza pneumonia, ceftrixone,cefotaxime or cefuroxime sodium should be used.

When results of bacterial culture and susceptibility of bacterium to antibiotics are sent from the laboratory change to the type of antibiotic suggested by the lab. If response to empiric therapy is not satisfactory.

Antibiotic dosage for pneumonia:

- Ampicillin: 150 – 200 mg / kg / day, q 6 hours.
- Penicillin-G sodium: 150 – 200 thousand units / kg / day q 6 hours.
- Oxacillin: 100 – 200 mg / kg / day, q 6 hours.
- Amikacin: 15-22 mg/Kg/day,q 8 hours.
- Cefuroxime sodium(2nd gener Cephalosporin):100-150 mg/kg/day q 6 hours.
- Cefotaxime (3rd gener. Cephalosporin): 100 – 150 mg / kg / day, q 6 hours.
- Ceftriaxone (3rd gener. Cephalosporin): 50 – 75 mg / kg / day, q 12 hours.
- Gentamicin (aminoglycoside): 6 mg / kg / day, q 8 hours.

Pleurisy

Definition : Inflammation of the pleura.

Types:

1- Dry or plastic pleurisy.
2- Serofibrinous pleusiy.
3- Purulent pleusiy (empyema).

Dry Or Plastic Pleurisy

Etiology:

1-Bacterial pulmonary infection: Pneumonias, lung abscess, and pulm. TB.
2-Acute upper respiratory tract infection.
3-Connective tissue disease e.g. rheumatic fever,SLE.

Pathology:

Inflammation of the pleura with fibrin deposition and small amount of yellow serous fluid. Adhesions between the two pleural surfaces and variable degrees of thickening of the pleural layers.
In TB,the adhesions develop rapidly and the pleura is often thickened.

Clinical picture

1) Symptoms and signs of the etiologic disease.
2) Pleural pain: Stabbing, or dull ache, over the chest and may be radiated to upper abdomen, shoulder, and the back, exaggerated by deep breath, coughing or straining, and relieved by lying on the affected side.
3) Pleural friction rub heard over the affected area, usually disappears after a few hours to days.

X ray chest: Diffuse haziness of the pleura.

Differential Diagnosis:

From other causes of chest pain, and dry cough.

Treatment:

a) Treat underlying disease.
b) Analgesic antipyretics.

Pleural Effusion

Definition: This is the collection of fluid in the pleural space.

Types and Etiology: The following table illustrates the most common types of pleural effusion and their etiology.

Type	Characteristics	Etiology
Serofibrinous	Exudate Clear or cloud Sp. Gravity > 1016 Proteins > 3g/dl WBC > 1000/cmm LDH> 200U/L (>60% of serum level) Glucose less than serum PH < 7.2	• Para pneumonic • Pulm. TB • Collagen disease • Infections in the mediastinum or abdomen • Malignancy.
Purulent	Cloudy or pus PMNs > 5000 /cmm Glucose < 50mg /dl Microorganism on smear.	• Purulent pleurisy (Empyema) • Para pneumonic lung abscess • Lung abscess • Chest trauma
Hydrothorax	•Clear transudate (i.e. not exudate) •Few mesothelial cells •Glucose same as serum •LDH (<60% of serum)	• Nephrotic syndrome • Glomerulonephritis, and CHF • Venous obstruction.
Hemothorax	•Uniformly bloody •Does not clot •Its hematocrit > ½ blood Ht. •Hemosiderin-laden macrophages.	• Chest trauma • Hemorrhagic diseases • Pulm.embolism • Malignancy
Chylothorax	•Milky •Triglycrides > 50mg/dl •Lymphocytes > 5000 /cmm •Becomes clear when shaken with ether	• Lymphatic obstruction (tumor) • Thoracic duct injury (trauma) • Chest surgery.

Serofibrinous Pleurisy

Etiology:

1-The most common causes are peumonia or pulmonary TB.In peumonia,the amount of fluid is usually small ,while in TB it is large.

2-Other causes:

(a)Inflammatory conditions in the mediastinum.

(b)Inflammatory conditions in the abdomen.

(c)Connective tissue disorders.

(d)Primary or metastatic neoplasms of the lung,pleura or mediastinum.

Clinical Picture:

A- Symptoms and signs of underlying disease

B- Symptoms of pleural disease:

1- Pleural pain early in disease and disappears when fluid accumulates.

2- Large amounts can cause cough, dyspnea , cyanosis, retractions, tachypnea or orthopnea.

C- Signs of pleural effusion:

1- Small amounts may be detected only by X-ray.

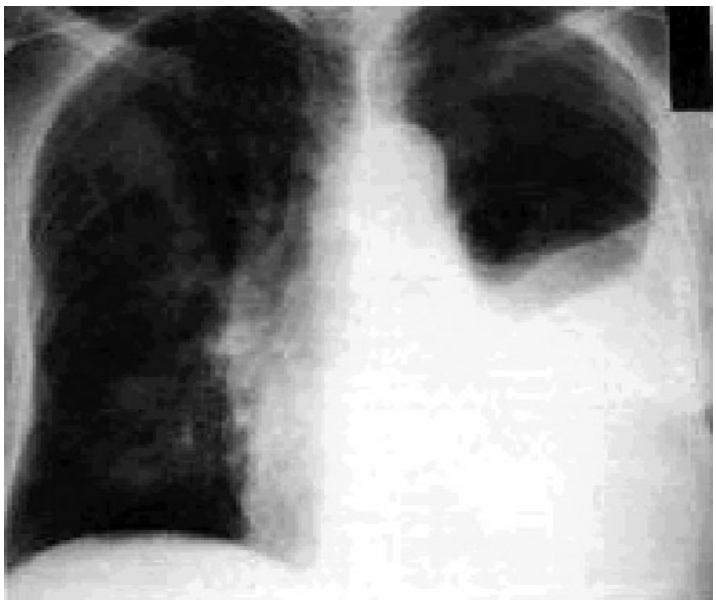
2- Large amounts give chest signs of pleural fluid

3- In infants bronchial breathy may be heard all over the affected side.

X-Ray Chest:

1- Small amounts are detected as obliteration of the costophrenic and cardiophrenic angels or widening of the interlobar septa.

2- Large collections show homogenous opacity obliterating the angles and ascending in the axilla with shift of the trachea and mediastinum to the other side and lung collapse upward & medially



X ray chest show left pleural effusion

Thoracocentesis: shows the characteristics of the effusion.

Differential Diagnosis:

1- Other causes of pleural effusion

2- Lobar pneumonia

3- Massive collapse

4- Pneumothorax.

Course And Prognosis:

1- With cases of pneumonia the fluid usually is absorbed rapidly. However it may become purulent i.e. empyema.
2- Tuberculous cases absorb in a few weeks. Pleural adhesions and thickening may result but usually resolve after a variable time leaving no functional impairment.

Treatment:

1- That of underlying disease .In tuberculous cases Prednisone is useful in addition to anti-TB drugs.
2- In case of large effusions aspirate slowly as much as possible up to one-liter when the patient is first seen. If significant amount re-accumulate repeated aspiration or tube drainage is indicated.
3- Management as empyema if it is purulent.

Purulent Pleurisy

(Empyema)

Definition:

Accumulation of pus in the pleural space.

Etiology:

1- Most cases are secondary to staphylococcal pneumonia, and some cases complicate pneumonia caused by Pneumococci and H. influenza bacillus. Patients are usually infants and preschool children.
2- Other causes include rupture of lung abscess, chest trauma, or extension of subphrenic abscess.

Pathology:

1- Pus frequently is located allover large area of the pleural cavity.
2- The parietal pleura is thickened.
3- If undrained, the pus may penetrate the pleura and dissect into the lung parenchyma producing bronchopleural fistula and pyopneumothorax.
4- Pockets of loculated pus may develop into thick walled abscess.
5- In neglected chronic cases the exudate will organize into a thick inelastic envelope (peel) and the lung may collapse

Clinical Picture:

1- An infant or preschool child with pneumonia, which is treated with inappropriate, or inadequate antibiotic.
2- Sudden onset of fever and dyspnea may occur while on treatment or after apparent cure. Preschool children appear more ill than infants.
3- Signs of pleural effusion on chest exam.

Investigations:

1- Thoracocentesis:

i- Purulent exudate, PMNs > 5000/cmm, and glucose < 50mg/dl.

ii- Causative organism determined by Gram stained smears, latex agglutination and culture. Antibiogram is done.

2- Chest X-ray:

a) Like serofibrinous pleurisy

b) When loculated no shift of fluid is observed when the child's position is changed.

Complications:

1. With staphylococcal cases, bronchopleural fistula and pyopneumothorax are common.

2. Purulent pericarditis, and lung abscess.

3. In H. influenza and pneumococcus cases, septicemia with osteomyelitis, arthritis and meningitis may occur.

4. Chronic empyema causes pulmonary atelectasis.

Diagnosis and DD: As for serofibrinous pleurisy.

Treatment:

1. Drainage: the maximum amount of pus should be aspirated. This is followed by continuous closed drainage under water seal through a wide catheter in the pleural cavity for 1-2 wks.. More than l tube is needed to drain loculated areas.

2. Systemic antibiotics IV or IM:

- Duration: 3-4 weeks for staph. cases, and 2 weeks for other cases .
- Types: according to sensitivity of isolated microorganism to antibiotics.
  a) Staph. aureus➔give oxacillin or cephazolin
  b) Pneumococci➔give cefuroxime, cefotaxime or ceftriaxone
  c) H. Influenza➔give cefotaxime or ceftriaxone

3- Oral antibiotics for 2-3 weeks after IV antibiotics.

4- Symptomatic treatment: Oxygen, analgesic antipyretic, and light diet during febrile period.

5. For chronic empyema decortication of the lung and pulmonary exercises are needed to regain lung function.

Pneumothorax

Definition:

It is the presence of air in the pleural space.

Types And Etiology: **Spontaneous** or **Traumatic**

Spontaneous

a) **1 ry:** Rare without apparent cause usually in tall and thin males with subpleural blebs in the apex of upper lobe.

b) **2ry: to underlying lesions** Pneumonia, bronchial asthma, Pulm. Abscess, Cyst, FB and Cystic fibrosis

Traumatic:

- Accident: by external blunt or penetrating injury of the chest.
- Iatrogenic: during thoracocentesis, ventilation, neonatal resuscitation, bronchoscopy, or lung biopsy.

Pathogenesis

- Transforms the potential space into a real one
- Air in the pleural space ➤ lung collapse ➤ Hypoxemia (ventilation-perfusion mismatch).
- In **simple pneumothorax**, intrapleural pressure is atmospheric, and the lung collapses up to 30%.
- In **tension pneumothorax**, continuing leak causes increasing positive pressure in the pleural space, which exceeds atmospheric pressure, and the lung totally collapses with shift of the mediastinum towards the opposite side.

.

Clinical Picture:

Symptoms and signs of underlying etiology.
Small amount of air may be discovered on chest X-ray.
Larger amount give varying degrees of:

1- Chest pain.
2- Dyspnea with or without cyanosis
3- Signs of pleural air or signs of pleural air and fluid.

Tension pneumothorax:

- It is the presence of a large amount of air under tension in the pleural space.
- It results in:
  1- Symptoms and signs of pleural air are severe.
  2- The expansion of the contra lateral lung is diminished.
  3- Interference with venous return results in cardiovascular collapse (shock).

Investigations:

1- Chest X-ray: Homogeneous translucency on affected side with no lung reticulation markings. There is shift of the trachea and mediastinum to the other side. A small amount of barium is needed to differentiate pneumothorax from diaphragmatic hernia.
2- Thoracocentesis: Air is aspirated. A hiss may be heard in cases of tension pneumothorax.

Differential Diagnosis:

1- Pleural fluid
2- Pneumonia
3- Severe asthma
4- Diaphragmatic hernia

Treatment:

(I)General:

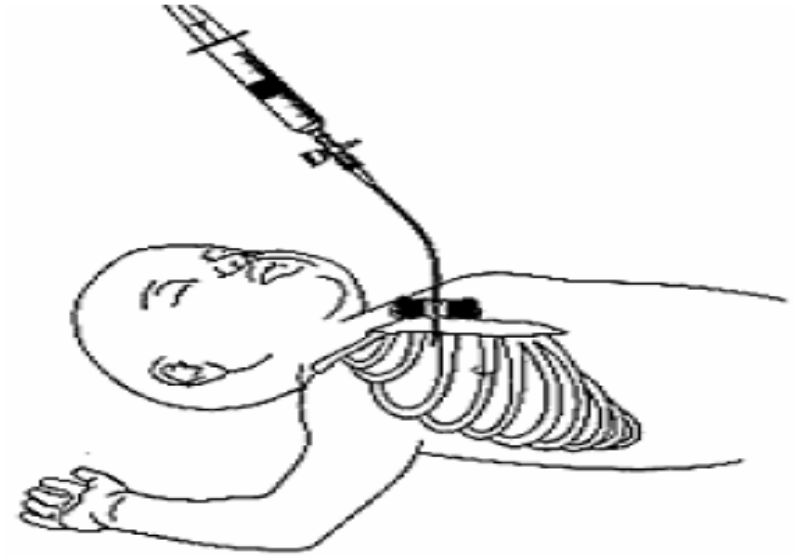
- O2 therapy if there is tachypnea or dyspnea
- Analgesics for pleural pain

(II)Drainage:

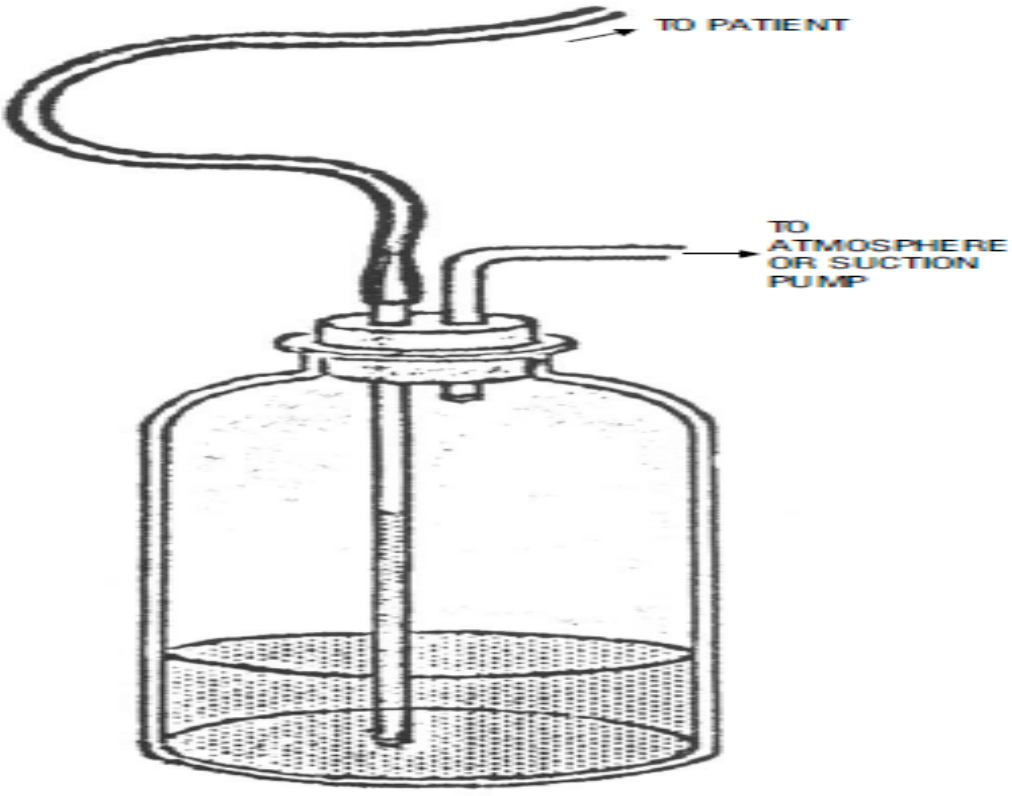
1-Mild cases with less than 5% collapse need no treatment, they will absorb spontaneously in about one week.
2-Drainage is needed for cases with more than 5% collapse and for tension pneumothorax closed drainage of air under water seal is adequate to relax the collapsed lung in most cases.

(III)Treat the underlying condition.

(IV)For recurring cases thoracic surgery is needed.



Needle aspiration of pneumothorax



Closed drainage of pneumothorax

PRIMARY PULMONARY TUBERCULOSIS

Etiology:

- Causative organism is Mycobacterium tuberculosis, an acid-fast, weak gram positive, 2- 4 µm bacillus.
- Source of infection is an adolescent or adult patient with productive cough or pulmonary tuberculous cavity more commonly at home or school.
- Infection: is person to person by droplets of sputum, rarely patient fomites. Adequate treatment with anti-TB drugs for a few weeks reduces the infectivity of patients.

Predisposing Factors:

A- Environmental:

1. Poorly ventilated rooms
2. Over crowding

B- Lowered cell-mediated immunity:

1. Malnutrition
2. Bronchopneumonia
3. Measles
4. Pertussis
5. Diabetes mellitus
6. Corticosteroid therapy.

Age factor: the age range 5-14 yr has the lowest incidence of TB
Sex factor: adolescent girls are more liable to get TB than adolescent boys.
Genetic factors: Predisposition to TB was observed.
Race factor: Negroes are more liable to infection with TB.

Pathology:

- When an infant or child's lung is infected with the TB bacillus for the first time, he develops what is called initial or primary pulmonary TB.
- This consists of two components: a pulmonary focus and infected tuberculous mediastinal lymph nodes, which from what is called the primary complex.
- The pulmonary TB focus is about 1 cm in diameter, and single subpleural and in any lung segment the mediastinal nodes are slightly enlarged, matted and adherent to surrounding structures.
- Left side focus causes bilateral adenopathy, while right side focus causes Rt. side adenopathy.

Fate of primary complex:

I. Healing in 60-95% of cases:

- More common in the older child.
- Healing by calcification of both the pulmonary and the mediastinal lesions.
- Radiologic detection of calcified nodes required >6 months from infection.
- Many of these lesions resorb and disappear completely in a few years.
- Viable mycobacteria may persist in calcified lesions for years.

II- Progression:

- This is more common in infants and young children.
  - **i-** If the patient's lung resistance is low (or is lowered by PEM, diabetes, pulmonary infections, and steroid therapy).
  - **ii-** If bacilli are numerous or virulent.
- In these patients the primary lesion may remain (or become) active and results in what is called progressive primary pulmonary TB.
- Progression occurs by several mechanisms:
  - **1-** Direct spread into surrounding tissues: The pulmonary focus will progress to give a larger area of consolidation (Pneumonic TB).
  - **2-** The mediastinal nodes enlarge, invade, compress and completely obstruct the nearby bronchus producing consolidation – collapse that is the most common lesion in infants and young children.
  - **3-** The pulmonic or medistinal lesions may erode a bronchial wall or lumen. Tuberculous granuloma and / or bronchogenic dissemination of bacilli to other parts of one or both lungs may result in tuberculous bronchopneumonia.
  - **4-** Lymphatic spread causes small pleural effusion. Significant effusion may occur in children >6yr never associated with pulmonary TB consolidation.
  - **5-** The primary lesion may erode into blood vessel causing hematogenous dissemination to form:
    - **i-** Widely spread milliary lesions all over the body.
    - **ii-** Isolated foci (tuberculomas) in lungs, bones, kidneys, liver or CNS.

    Tubercle bacilli may remain dormant in hematogenous pulmonary foci for years till adolescence or adulthood when they may multiply and produce the characteristic lesion of reactivation or adult type pulmonary TB.

Tuberculin Test:

- Two to ten weeks after infection with tubercle bacilli, the child becomes sensitive to the tuberculous protein.
- This sensitivity can be detected by the tuberculin test. In this test, 5 i.u. of purified protein derivative (P.P.D) of tubercle bacillus is injected intradermally in the dorsal surface of the forearm.
- The site of injection is inspected 48 hours later and the length of any elevated firm (indurated) area is measured.

(1) An induration of 10 mm or more is a positive test and indicates TB disease.

(2) An induration of 5-9 mm:

a- It may be due to B.C.G. vaccination 3-10 years ago.

b- It may be evidence of TB disease:

- If the child is contact of TB patient,
- If the child has a disease or chest X-ray suggestive of TB,
- If the child is debilitated,
- If the child is under steroid therapy,
- If has associated measles, mumps, varicella, and influenza, or
- If he is recently vaccinated with live viral vaccines.

(3) An induration less than 5 mm is a negative test:

- It occurs when the child is not tuberculous.
- Bad technique or misreading gives false negative test.
- 10% of children with TB and up to 50% of children with milliary TB have false negative test, which becomes positive after treatment with anti-TB drugs.

Clinical Picture:

A- In a large proportion (>60%) of infected children, primary pulmonary TB is represented only by a tuberculin test more than 5 mm while chest X-ray is free.

B- Intrathoracic pulmonary lesions occur in < 40% of infected children 3-24 months after infection. The younger the child the greater the risk of developing intrathoracic pulmonary lesions.

I- Enlarged non-calcified or calcified mediastinal nodes detected 3 months or more after infection.

- These nodes are usually discovered on routine X-ray chest. Respiratory symptoms and chest signs are usually minimal or absent.
- Low-grade fever, erythema nodosum or phlyctinular conjunctivitis may be present.

II- Progressive primary pulmonary TB:

General: Fever 38-39^{o}C, night sweats, pallor, anorexia, and failure to gain weight or marasmus.
Respiratory: as dry or productive cough, dyspnea, and grunting, wheezing or pleural pain.
Chest signs: Progressive pulmonary TB may give chest signs, which can simulate any acute or chronic lung disease.

- Sings of consolidation i.e. pneumonic TB.
- Sings of consolidation – collapse, the most common presentation especially during infancy and early childhood.
- Signs of partial bronchial obstruction caused by enlarged lymph nodes and positive D'Espine sign due to enlarged mediastinal nodes.
- Sings of bronchopneumonia with tubercle bacilli in the sputum.
- Signs of pleural effusion in a child of six years or more in age are highly suggestive of TB as a cause (5-7% of cases). TB effusion is never associated with pulmonary consolidation.
- In milliary TB and in addition to the diffuse minute pulmonary infiltrations, there may be pleurisy, peritonitis, meningitis, splenomegaly, and choroidal tubercles in the eyes.

III- Reactivation (Adult type) pulmonary TB:

- Clinical manifestations occur by adolescent age.
- This is more common in those who acquired primary TB after the age of 7years.
- The primary lesion heals but when the resistance of adolescent is lowered, dormant bacilli in the original primary TB focus or the hematogenous tuberculomas multiply and produce a caseous pulmonary lesion.
- The site is more commonly in the lung apex, but may be anywhere.
- The clinical picture includes productive cough, hemoptysis, weight loss, night sweats, fever, signs of pulmonary consolidation or cavitations are present.
- The mediastinal nodes are not enlarged. The adolescent is considered a source of infection to his contacts.

Diagnosis Of Pulmonary TB:

1. Suspect TB as the cause of any bronchoplumonary disease.
2. Family history of contact with adolescent or adult patient with open pulmonary T.B.
3. Chest X-ray examination.
4. Tuberculin test.
5. Acid-fast TB bacilli in sputum obtained by gastric lavage.
6. Recent methods:
   - ✓ Polymerase chain reaction (PCR) for sputum. Results are obtained after 1 hr.
   - ✓ BACTEC radiometric system for sputum culture. Only 1 – 3 weeks are needed to report culture and susceptibility to anti-tuberculous drugs.

Set of diagnostic criteria of pulmonary TB in children when culture is not available

A. Positive acid-fast strain of sputum or gastric aspirate or
B. Two or more of the followings:
   - History of contact with a tuberculous adult
   - Cough lasting longer than 2 weeks
   - A reactive tuberculin test:
     1) ≥ 10 mm in children without prior BCG vaccination.
     2) ≥ 15 mm in children with prior BCG vaccination.
   - Radiologic findings comparable with TB
   - Response to anti-TB therapy (increased body weight by 10% after 2 months, decrease in symptoms)

Differential Diagnosis:

I-Bronchopulmonary diseases:

1- Chronic bronchitis in a malnourished infant or child.
2- Unresolved lobar pneumonia
3- Bacterial bronchopneumonia.
4- Non-tuberculous pleural effusion.
5- Bronchial asthma.

II-Fever of undetermined cause:

1- Collagen disease.
2- Enteric fever, bilharziasis, brucellosis, and urinary tract infection.
3- Malignancy as lymphoma or leukemia.

Prognosis:

- Most children recover especially if treated early.
- Anti-TB drugs result in striking decrease in the incidence of hematogenous extra thoracic lesions e.g., TB meningitis.
- The fatality rate is highest in the first two years of life and in adolescents.

Treatment:

A- General:

1- Diet adequate in quality and quantity.
2- Rest during the early period of treatment.
3- Symptomatic for cough, fever, pain etc.

B- Anti-tuberculous drugs:

I-The infant or child with only a tuberculin test more than 5 mm:

A- Isoniazid (INH) 15 mg /kg /day (max 300mg) divided into 2 doses for one year plus vitamin B_6 25-50 mg/day to prevent neuropathy.

B- Chest X-ray every 3 months during therapy in order to diagnose early intrathoracic lesions.

II- Infant or child with intrathoracic lesions:

Three anti-TB drugs combination is given in order to prevent and treat infection with drug-resistant strains of TB bacilli e.g. Pyrazinamide + INH + Rifampicin.

Doses:

- Pyrazinamide: 20-40 mg /kg /day, orally in one or two doses (Max 2 gm/day)
- INH: 10 mg /kg /day (Max 300 mg), oral divided into 2 doses.
- Rifampicin: 15 mg /kg /day (Max 600mg) divided into 2 doses, on empty stomach. + Vit. B6: 25-50mg/day.

Duration:

- Pyrazinamide is given for first 2-3 months for all cases.
- INH + Rifampicin for 6-9 months for all patients with intrathoracic TB.
- Streptomycin is added as a fourth drug for 2-3 months if resistance to INH is suspected.

C- Corticosteroids:

Prednisone in a dose of 1-2 mg /kg /day for 1-2 months is added to the anti-tuberculous drugs in the following cases:

- Marked enlargement of mediastinal nodes causing collapse, obstructive emphysema or dyspnea.
- Tuberculous bronchopneumonia.
- Milliary pulmonary T.B.
- Tuberculous pleural effusion.

Prevention Of TB:

A- Early detection and treatment of pulmonary TB in adolescents and adults
e.g., by mass screening.

B- Keep the child away from any adolescent patient even if under treatment.

C- Isoniazid prophylaxis:
If child gets in contact with an adolescent or adult with open pulmonary tuberculosis, he or she should be given I.N.H. in a dose of 10-20 mg/kg/day for one year.

D- Vaccination with BCG:

- Routine vaccination with BCG is given during the first 2 months of life, at school entry and by the age of 10-12 years.
- Booster vaccination is given if the child is a contact of patient.
- The dose is 0.1 ml intradermally in left deltoid region.
- BCG cannot prevent infection but it prevents TB dissemination.
- About 50% of vaccinated infants demonstrate tuberculin never become tuberculin positive, these 50% lose their positive test in 3 years.
- Vaccinated older children frequently become tuberculin positive, and lose their positive reaction in 10years.
- A tuberculous abscess may result from subcutaneous administration of BCG or from a virulent strain, and is treated by anti-TB drugs.

E. Social measures:

Adequate diet, good hygiene, good housing, and avoidance of over crowding.