MED439
KING SAUD UNIVERSITY

pediatric
439 Teamwork

Revised by The Academic Leader
Homoud Algadheb

Obstructive Airway disease

objectives:

11.1 Understand the classification of airway obstruction anatomically and physiologically.
11.2 Recognize the causes of bronchiectasis and methods of diagnosis.
11.3 Define asthma.
11.4 List the major pathologic factors responsible for airway obstruction in asthma
11.5 Discuss precipitating factors including: Infection Irritants Exercise Allergens
11.6 Describe the clinical findings typical of asthma.
11.7 Gain familiarity with diagnosis, differential diagnosis (vascular ring, foreign body aspiration, cystic fibrosis, bronchiolitis, etc.)
11.8 Discuss the role of spirometry, radiography and allergic skin testing in the diagnosis and management of asthma
11.9 Discuss classifying asthma severity in patients based on day and night time symptoms and lung function- intermittent, mild persistent, moderate persistent, severe persistent.
11.10 Explain environmental control measures.
11.11 Discuss the different classes of drugs used in the medical management of asthma and their side effects and their use in step therapy based on asthma severit
11.12 Describe current evidence to support the use of the following in the treatment of asthma
Short acting bronchodilators, Long acting bronchodilators, Atropine derivatives (e.g. ipratropium)
Inhaled steroids, theophylline (methylxanthines), cromolyn and nedocromil, leukotriene modifiers Oral and parenteral steroids
11.13 Understand different asthma devices including metered-dose inhaler, spacer devices, dry powder inhalers.
11.14 Define and explain the management of acute asthma exacerbation.
11.15 Learn the indications for hospitalization of acute exacerbation of asthma. لاتخاف مايحتاج تذاكره

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Special thanks to team 438 & Faisal alsaif

Notes

Important

Book

Obstructive Airway Disease:

obstruction of airflow in the airways occurs by:

- Growth: Enlarged cardiac chamber, adenoma, meningioma, aneurysm.
- Lumen (within) ⟶ mucus, foreign body.
- Wall ⟶ edema (inflammation), cartilage deficiency (narrowing).

[cartilage maintains the caliber of the airway; deficiency leads to narrowing].

Physiological:

- Reversible = Asthma.
- Irreversible: Bronchiectasis (Permanent airway destruction).

Anatomical:

- Upper ⟶ Stridor [high pitched, inspiratory or biphasic, monotonous].
- Lower ⟶ Wheeze [musical, expiratory, polytonus].
- **Subglottic ⟶ biphasic noise****

TABLE 135.2 Common Causes of Acute Upper Airway Obstruction

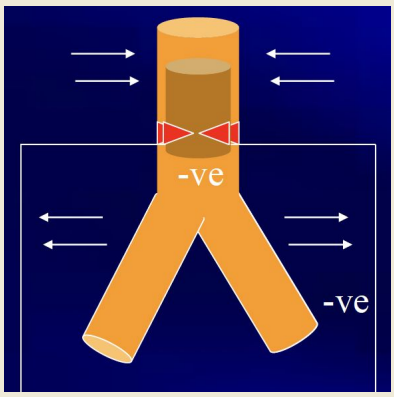
DIAGNOSIS	AGE	HISTORY	EXAM	IMAGING AND LABS	TREATMENT
INFECTIOUS					
Croup (*parainfluenza* and other viruses)	6 mo–3yr	Fever, URI	Nontoxic, stridor, barky cough, hoarse	Steeple sign	Aerosolized epinephrine, systemic steroids, cool mist
Epiglottitis (*Streptococcus pneumoniae, Haemophilus influenza*; respiratory viruses)	2–6yr	High fever, rapid onset, no cough, unable to swallow	Toxic, agitated, tripod sitting, drooling, stridor	Thumb sign Leukocytosis	Intubation, antibiotics
Bacterial tracheitis (*Staphylococcus aureus, Moraxella catarrhalis*)	Any age	High fever, rapid onset, no URI symptoms	Toxic, anxious, ±stridor, ±cough	Ragged tracheal border Leukocytosis	Intubation, antibiotics
Retropharyngeal abscess (*S. aureus*, group A strep, oral anaerobes)	<6yr	Fever, insidious onset, sore throat, no URI/cough	Moderately toxic, drooling, arched neck, inflamed pharynx	Thickened retropharyngeal space Leukocytosis	Antibiotics, surgical drainage
Peritonsillar abscess (group A strep, oral anaerobes)	>8yr	Fever, sudden worsening, sore throat, trismus	Moderately toxic, "hot potato" voice, drooling, asymmetric tonsil swelling	Imaging not needed Leukocytosis	Antibiotics, surgical drainage
NONINFECTIOUS					
Angioedema	Any age	No fever, sudden onset, urticarial, facial swelling, ± allergen exposure	Nontoxic (unless anaphylaxis), ± stridor, hoarse, facial edema	Steeple sign	Aerosolized or intramuscular epinephrine, systemic steroids, antihistamines
Spasmodic croup	6 mo–6yr	Sudden onset, no fever/URI, recurrent, often nocturnal	Nontoxic, ± stridor, hoarse, barky cough	Often normal	Aerosolized epinephrine, antihistamines, antacids, systemic steroids
Foreign body	6 mo–5yr	Sudden onset, cough and choke	Nontoxic, anxious, stridor, aphonic, brassy cough	Radiopaque object may be seen	Rigid bronchoscopy

URI, Upper respiratory tract infection (with coryza, sneezing).

❖ The upper airway is involved with inspiratory sounds, while the lower airway with expiratory sounds.

*Inspiratory noise: object outside the chest

Inspiration ⇩

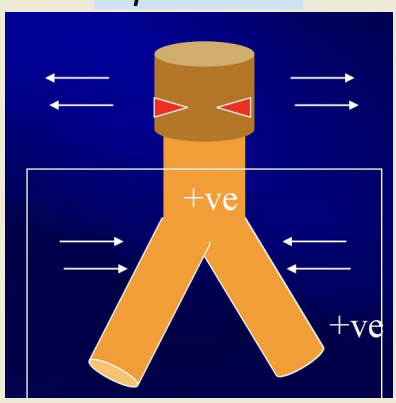


On inspiration ⟶ diaphragm is lowered & space is increased ⟶ -ve pressure:

- Lower airway (intra-thoracic) get dilated, any obstruction there won't be clear during this phase.
- Upper airway (extra-thoracic) will get squeezed, & the obstruction will be clear as inspiratory stridor.

On expiration ⟶ +ve pressure causes the upper airway to dilate (obstruction won't be clear) & lower airway to get squeezed (clear expiratory wheeze or crepitations).

Expiration ⇧



**Expiratory noise: object inside chest.

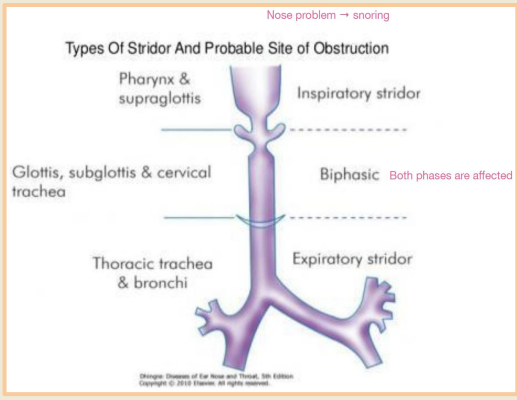
Abnormal lung sounds:

Stridor:

❖ a harsh sound due partial obstruction of **the upper airway (trachea, larynx).**

➢ The most common cause is: Croup.

➢ Other causes include (epiglottitis, bacterial tracheitis).



Abnormal lung sounds:

Wheeze:

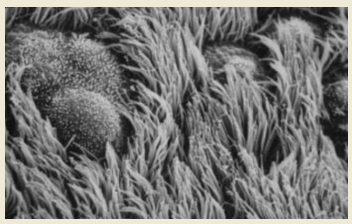
- due to partial obstruction of **intrathoracic airways** from:
  - Mucosal inflammation & swelling → (bronchiolitis).
  - Bronchoconstriction → (asthma).
  - Mechanical obstruction → (foreign body, mucus).
- **Patterns of wheezing:**
  - Viral episodic wheezing (ex: common cold)
  - Multiple trigger wheeze
    (cold air, animal dander, exercise, dust):
    a significant proportion progress to asthma.
  - Asthma.

Box 17.2 Causes of recurrent or persistent childhood wheeze

- Viral episodic wheeze
- Multiple trigger wheeze
- Asthma
- Recurrent anaphylaxis (e.g. in food allergy)
- Chronic aspiration
- Cystic fibrosis
- Bronchopulmonary dysplasia
- Bronchiolitis obliterans
- Tracheo-bronchomalacia

The lung:

- has a 100 sq meters of surface area & 500 million alveoli.
- It's less in newborns then increases in the first 2 years of life.. **the complete number is reached by age 10**, after that there will be growth by hypertrophy rather than hyperplasia. That's why if we do lobectomy in a child, they can compensate
- It can process over 10,000 L air/day; Tidal volume: 500 mL. RR: 12-14 BPM → 7 L of air per min.
- The only internal organ exposed to the outside environment;
- Unique immune system (macrophages & IgA).
- Complex mucoid gel/ELF capturing allergens/microbes.
- Cilia beating 1000 times/min to protect the airway;
  - Coordinated movement of the cilia is important: in the UL it beats down, & in the LL it beats upwards.
  - Primary ciliary dyskinesia causes a disturbance in this movement
  - Cough is another protective mechanism if the cilia cannot move the mucus, or if the mucus is too much → as in **ciliary dyskinesia/CF** patients cough so much.



Chronic Lung Infections:

- Any child with a persistent (chronic) cough that sounds 'wet' *(i.e. sounds like there is excess sputum in the chest)*. or is productive requires further investigations

Box 17.3 Causes of chronic or recurrent cough

- Recurrent respiratory infections
- Persistent bacterial bronchitis (will be persistently wet)
- Following specific respiratory infections (e.g. pertussis, respiratory syncytial virus, *Mycoplasma*)
- Asthma (only if accompanied by wheezing)
- Persistent lobar collapse following pneumonia
- Suppurative lung diseases (e.g. cystic fibrosis, ciliary dyskinesia or immune deficiency)
- Recurrent aspiration (± gastro-oesophageal reflux)
- Inhaled foreign body
- Cigarette smoking (active or passive)
- Tuberculosis
- Habit cough
- Airway anomalies (e.g. tracheo-bronchomalacia, tracheo-oesophageal fistula)

Persistent bacterial bronchitis:

- Persistent inflammation of the lower airways driven by chronic infection, is increasingly recognized as a cause of chronic wet cough in children.
- Common organisms are Haemophilus influenzae & Moraxella catarrhalis.
- **It may be a precursor to bronchiectasis.** Frequent relapse (recurrent PBB) is associated with a subsequent diagnosis of bronchiectasis.
- **PBB is a clinical diagnosis based on** chronic wet cough, the absence of symptoms or signs suggesting another cause such as bronchiectasis and resolution of cough following antibiotic treatment.
  - Bacterial growth from sputum or bronchial lavage is consistent with the diagnosis.
  - Treatment is with a high dose of antibiotic such as co-amoxiclav, coupled with physiotherapy.

Bronchiectasis:

- It is the irreversible dilatation of the airway.. dilatation of the bronchial tree, confirmed on a CT scan.
- The cartilage maintains the size of the airway, so if it gets destroyed by infections, the airways dilate & the body cannot repair the cartilage → ineffective clearance → stagnation of secretions, mucous plugging → inflammation, bacterial colonization & infection.
- Bronchiectasis affects bigger airways which contain cartilage.
- Patients with bronchiectasis have episodes of cough, often productive of purulent sputum, and may have inspiratory crackles. Other clinical features of chronic suppurative lung disease; (recurrent episodes of chronic wet cough, coarse crackles on auscultation and finger clubbing).
- **Treatment** is based on optimizing airway clearance as well as the prevention and treatment of lower airways infections.

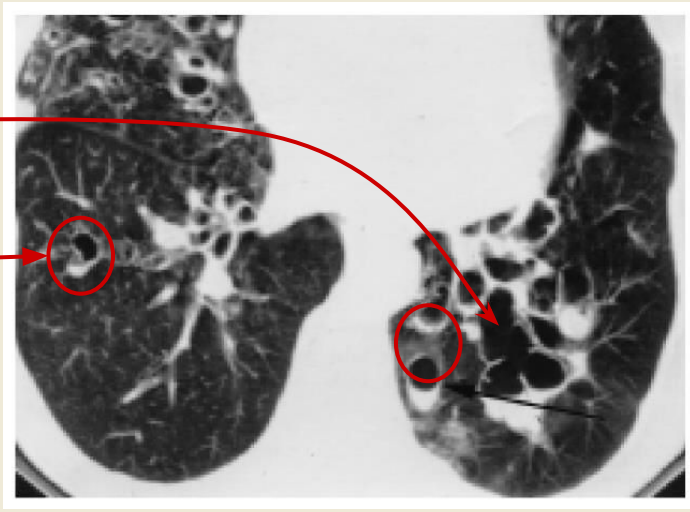
Differential Diagnosis / causes of Bronchiectasis:

- **Localised Bronchiectasis:** *(although systemic problems can start in one lobe then spread to the others)*
  - "Airway": Internal (**foreign body**) , external (**mediastinal lymph node**).
  - "Parenchymal": **lobar pneumonia** (very bad).

- **Diffuse Bronchiectasis:** *(most likely systemic)*
  - **Aspiration** → Risk factors: fistula/cleft, swallowing problems (cerebral palsy).
  - **Mucociliary clearance** → Primary Ciliary Dyskinesia, Cystic Fibrosis.
  - **Immune deficiency** → recurrent infections.
  - **Congenital** → born without cartilage.
  - **Post-infectious** → Pertussis, TB, Adenovirus (bad enough to damage the lung).

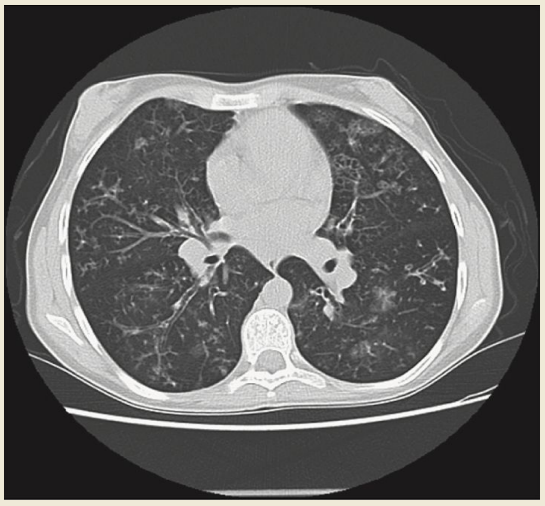
Bronchiectasis:

Investigation & Diagnosis:

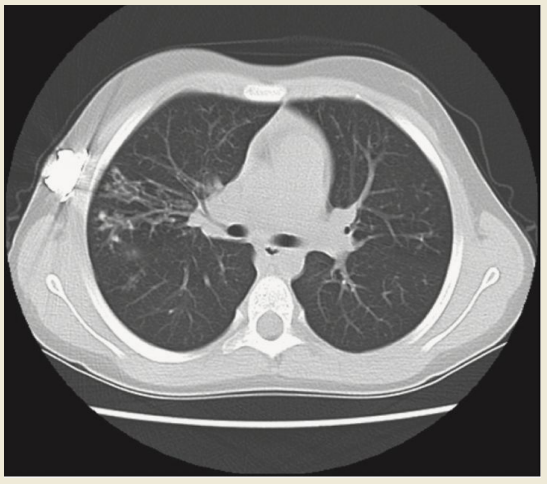


- ➔ **High resolution CT:**
  - ◆ **Look for <u>dilated airways</u>**
    (normally airways (0.5-0.8) smaller than blood vessels)
    - Airways > vessels. *(bronchiectasis)*
    - **"Signet ring" sign** (vessel +airway)

**Generalized, with enlargement of the bronchi throughout the left and right lung fields.

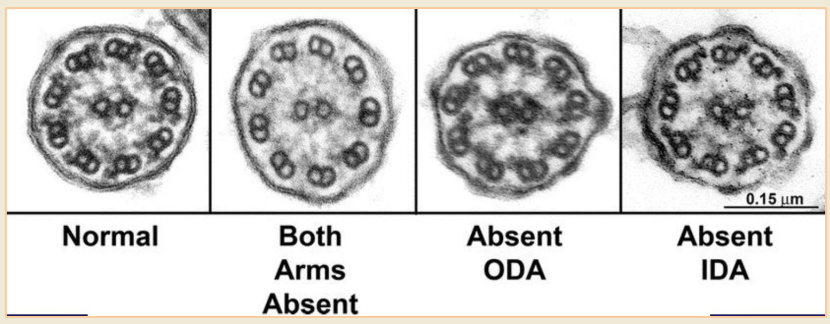


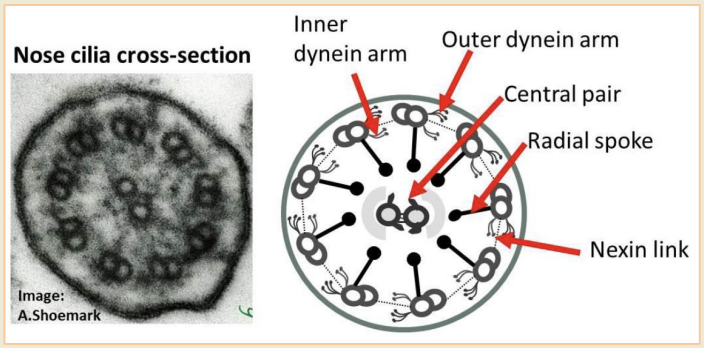
**focal, with changes in the right upper lobe



Primary Ciliary Dyskinesia (PCD)

- ❖ This is usually an autosomal recessive condition.
- ❖ It results in **abnormal structure or function of cilia lining** the respiratory tract. This leads to impaired mucociliary clearance.
- ❖ Cilia are defective (not moving in one direction) → ineffective clearance → **chronic cough.** *(coughing helps airway clearing)*
- ❖ Cilia present everywhere in the body; especially [RT, nasal sinuses, middle ear, productive system…].





Primary Ciliary Dyskinesia (PCD)

Manifestations:

- **Recurrent URTI & LRTI (recurrent productive cough)**; if untreated it will lead to bronchiectasis.
- **Recurrent Sinusitis** (recurrent productive nasal discharge).
- **Recurrent/chronic Otitis Media [OM]** (deafness/ **hearing loss**).
- **Male infertility**; Females may have subfertility or ectopic pregnancy (because of the movement of the ovum in the fallopian tube depends on the cilia).
- ★ **Kartagener syndrome = PCD + situs inversus** — (cilia is imp in orienting the organs in utero); ciliary action is responsible for normal organ situs, and almost 50% of individuals with PCD have **Kartagener syndrome with dextrocardia and situs inversus** (major organs are in the mirror position of normal).

Diagnosis of PCD:

- Sometimes: biopsy from the airway/turbinates & see cilia under electron microscope to examine the structure & movement.
- **Genetic testing [definitive].**
- The diagnosis of PCD is made by **examination of the structure and function of the cilia of nasal epithelial cells brushed from the nose in combination [+] with genetic testing.**
- Nasal nitric oxide is almost always low and can be used as a screening test prior to cilia studies.

Management:

- Daily physiotherapy to clear sections.
- Proactive treatment of infections with antibiotics.
- ENT follow up.
- Treatment is as for other causes of bronchiectasis.

TABLE 136.1 Causes of Wheezing in Childhood
ACUTE
Asthma
Exercise-induced asthma
Hypersensitivity reactions
Infection (e.g., bronchiolitis)
Inhalation of irritant gases or particulates
Descending aspiration
Foreign body (airway or esophageal)
Aspiration of gastric contents (ascending aspiration)
CHRONIC OR RECURRENT
Asthma (see under acute)
Hypersensitivity reactions, allergic bronchopulmonary aspergillosis (seen only in children with either asthma or cystic fibrosis)
Vocal cord dysfunction
Recurrent aspiration/swallowing dysfunction (dysphagia)
Retained foreign body
Gastroesophageal reflux
Bronchopulmonary dysplasia
Bronchitis, bronchiectasis
Cystic fibrosis
Primary ciliary dyskinesia
Bronchiolitis obliterans
Pulmonary edema (congestive heart failure)
Bronchomalacia/tracheomalacia
Vascular ring
Pulmonary artery dilatation (absent pulmonary valve)
Bronchial or pulmonary cysts/masses
Lymph nodes (tuberculosis, lymphoma)
Endobronchial masses/tumors (carcinoid)
Bronchial or tracheal stenosis
Cardiomegaly

Cystic Fibrosis (CF):

- It's a Life-limiting disease. Major cause of mortality is from the lungs.
- An incidence of 1 in 2500 live births and carrier rate of 1 in 25.
- It's an autosomal recessive [Genetic mutation] that **affects chloride channels (CFTR gene – ch7]**; they're either absent, or present → **but they don't work:**
  - The fundamental problem in CF is a defective protein called the CF transmembrane conductance regulator (CFTR). This is a cyclic AMP-dependent chloride channel found in the membrane of cells. The gene for CFTR is located on **chromosome 7.**
  - It is a chloride channel found on the membranes of all cells.

Pathophysiology:

- → **no pumped Cl^- → no Cl^- → no Na^+ → no water → dehydration ⇒**
- **⇒ Thick, sticky secretions that cannot be moved by the cilia**.
- **⇒ Defective CFTR also causes dysregulation of inflammation and defence against infection.**
- **⇒ Recurrent infections [+] chronic cough** (from infancy) *(coughing won't help)*

Manifestations : (multi-system disease)

❖ Recurrent infections (Dysregulation of inflammation and defence).

Respiratory tract:

❖ **Chronic infection (colonization)**:

- ➢ **⇒ Pseudomonas, H. influenzae, S. aureus.**
- ➢ Extensive bronchiolitis ⇒ abscess, bronchial wall destruction, high risk of pneumothorax.
- ➢ **Recurrent chest infections (LRTI).**
- ➢ Chronic wet cough.
- ➢ Clubbing, increased AP diameter, hyperresonance to percussion, expiratory wheeze.

Intestines (usually first presentation);

❖ **Meconium ileus & intestinal obstruction: "Thick viscid meconium"**

- ➢ Vomiting, distention,
- ➢ **Failure to pass meconium in the first few days of life.**
- ➢ **X-ray: dilated loops of bowel without air fluid levels.**
- ➢ Tx options: surgery, gastrografin enema.

❖ **Pancreatic exocrine insufficiency: ⇒ malabsorption!***

- ➢ *Ducts blocked by thick secretions*.
- ➢ **Steatorrhea.** chronic diarrhea.
- ➢ ADEK deficiency
- ➢ **Failure to thrive.**
- ➢ Increased incidence of DM & **Acute pancreatitis** (auto-digestion)

❖ **Rectal prolapse**

Box 17.4 Clinical features of cystic fibrosis

Newborn
- Diagnosed through newborn screening
- Meconium ileus

Infancy
- Prolonged neonatal jaundice
- Growth faltering
- Recurrent chest infections
- Malabsorption, steatorrhoea

Young child
- Bronchiectasis
- Rectal prolapse
- Nasal polyp
- Sinusitis

Older child and adolescent
- Allergic bronchopulmonary aspergillosis
- Diabetes mellitus
- Cirrhosis and portal hypertension
- Distal intestinal obstruction (meconium ileus equivalent)
- Pneumothorax or recurrent haemoptysis
- Infertility in males

Sweat glands: ⇒ excessive concentrations of sodium and chloride in the sweat (salty taste of skin).

GUT: Males: infertility (azoospermia); Females: secondary amenorrhea, decreased fertility.

Cystic Fibrosis (CF):

Diagnosis:

All newborn infants are screened for CF

- Raised immunoreactive trypsinogen "IRT" measured in routine heel prick blood: to Screen for common CF gene mutations ⟶ 2 mutations
- **Sweat test is the gold standard for the diagnosis** A raised sweat chloride concentration (>60 mmol/L) confirms the diagnosis.
  - **The gold standard diagnostic procedure is a sweat test.**
- **Confirmation of diagnosis can be made by testing for CFTR mutations.**

Management:

❖ (All pts should be reviewed at least annually) Patients should be reviewed every 2–3 months.

❖ The aim of management is to prevent progression of the lung disease (monitored using spirometry) and to maintain adequate nutrition and growth. **Psychologists play a particularly important role in the overall delivery of care.** The psychological repercussions of CF and the associated treatment burden on affected children and their families is considerable.

❖ Respiratory management:

➢ The key aspects of CF respiratory management are airway clearance and the aggressive treatment of lower respiratory infection.

➢ **Recurrent and persistent bacterial chest infection is the major problem.**

➢ In younger children, respiratory monitoring is based on symptoms; **older children should have FEV1 checked regularly.**

➢ **Continuous prophylactic oral abx (Flucloxacillin), with additional antibiotics for any increase in respiratory symptoms or decline in lung function (acute infection).**

- Pseudomonas coverage: piperacillin plus tobramycin or ceftazidime.

➢ **Azithromycin,regularly, decreases exacerbations, due to an immunomodulatory effect.**

➢ Airway secretions:

- Albuterol/hypertonic saline aerosols, DNAse (mucolytic).
- From diagnosis ⟶ **physical therapy** with postural drainage (1-4x/day). twice daily chest physiotherapy to clear airways.

➢ **Lung transplantation is the only therapeutic option for end-stage CF lung disease.**

❖ Nutritional management:

➢ Pancreatic replacement therapy taken with all meals and snacks.

➢ Diet intake is recommended at 150% of normal: overnight feeding via a gastrostomy.

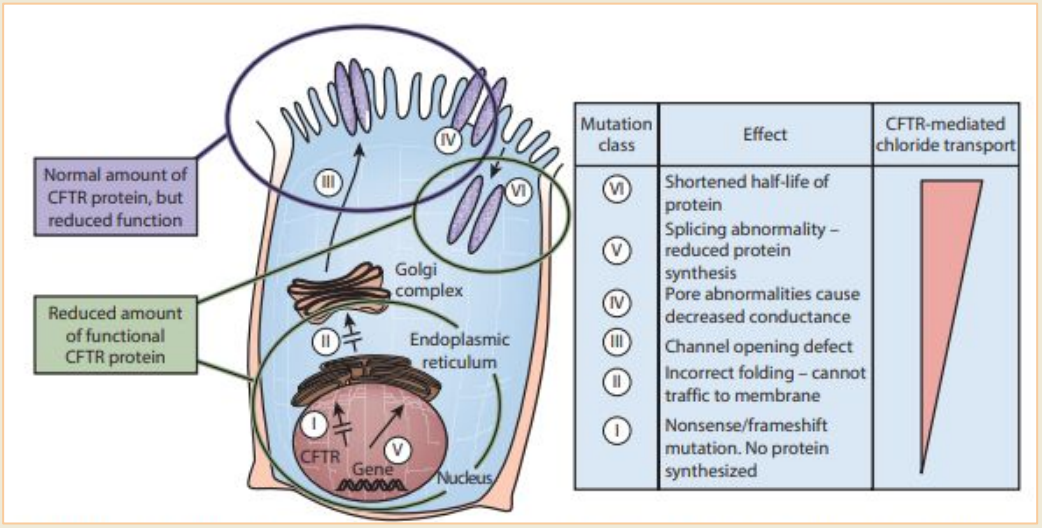
➢ ADEK supplements.

Cystic Fibrosis (CF):

- CFTR modulators:
  - There are three classes of drugs that can alter the functional expression of CFTR: potentiators, correctors and amplifiers.
  - CFTR potentiators (such as ivacaftor).
  - CFTR correctors (such as lumacaftor, tezacaftor and elexacaftor).
  - Recent studies using a combination of two correctors, and a potentiator (triple therapy) have shown exciting results in older children and young people.
  - **Despite early hopes, gene therapy has not yet proven to be a useful treatment in CF.**

Complications:

- Diabetes (often insulin-dependent).
- Liver disease (Cirrhosis and portal hypertension).
- Distal intestinal obstruction syndrome (meconium ileus equivalent).
- Increased chest infections [Allergic bronchopulmonary aspergillosis (ABPA)].
- Life threatening hemoptysis & Pneumothorax.



Mutation class	Effect	CFTR-mediated chloride transport
VI	Shortened half-life of protein	
V	Splicing abnormality – reduced protein synthesis	
IV	Pore abnormalities cause decreased conductance	
III	Channel opening defect	
II	Incorrect folding – cannot traffic to membrane	
I	Nonsense/frameshift mutation. No protein synthesized	

Cystic fibrosis should be considered in any child with recurrent infections, loose stools or faltering growth.

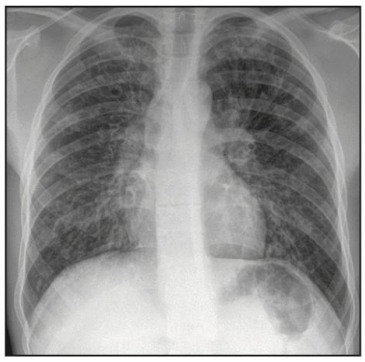


Figure 17.23 A chest X-ray in cystic fibrosis showing hyperinflation, marked peribronchial shadowing, bronchial wall thickening and ring shadows.

Summary

Cystic fibrosis

- Is the commonest inherited life-limiting condition affecting Caucasians.
- Is a multi-system disorder affecting the lungs, pancreas, liver and gastrointestinal tract.
- Treatment aims to prevent progressive respiratory failure due to repeated cycles of infection and maintain adequate growth and nutrition.
- New treatments with CFTR modulators appear to improve the outlook for some individuals.

العذر والسموحه كثرنا عليكم

Periodic review of the child or young person with cystic fibrosis

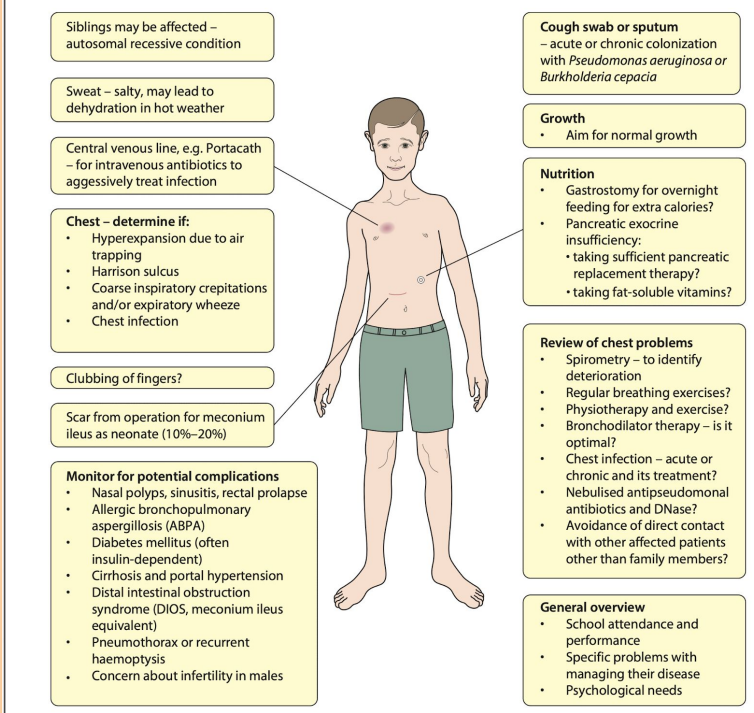


Figure 17.24 Periodic review of the child or adolescent with cystic fibrosis.

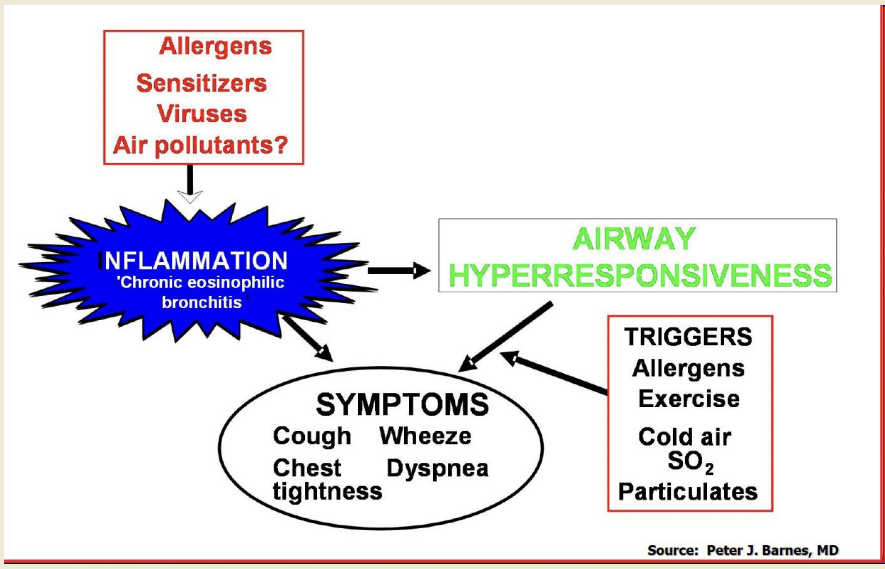
Asthma:

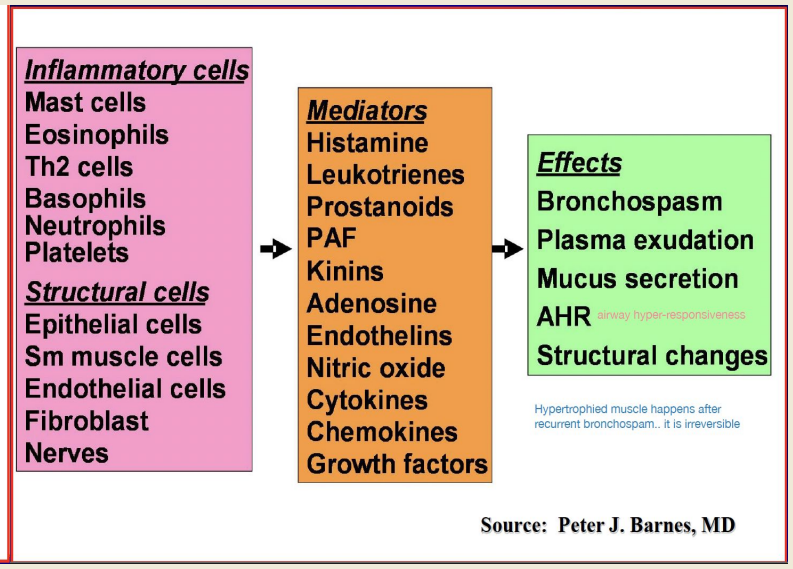
Definition:

- A chronic **inflammatory** disorder of the airways.
- *Many cells and cellular elements play a role).*
- Chronic inflammation is associated with **airway hyperresponsiveness** that leads to recurrent episodes of wheezing, breathlessness, chest tightness, and coughing.
- **Widespread, variable, and often reversible airflow limitation;**
    - The whole airway is involved (note that since the large airways are protected with catalase we don't hear stridor but in children sometimes we hear stridor).

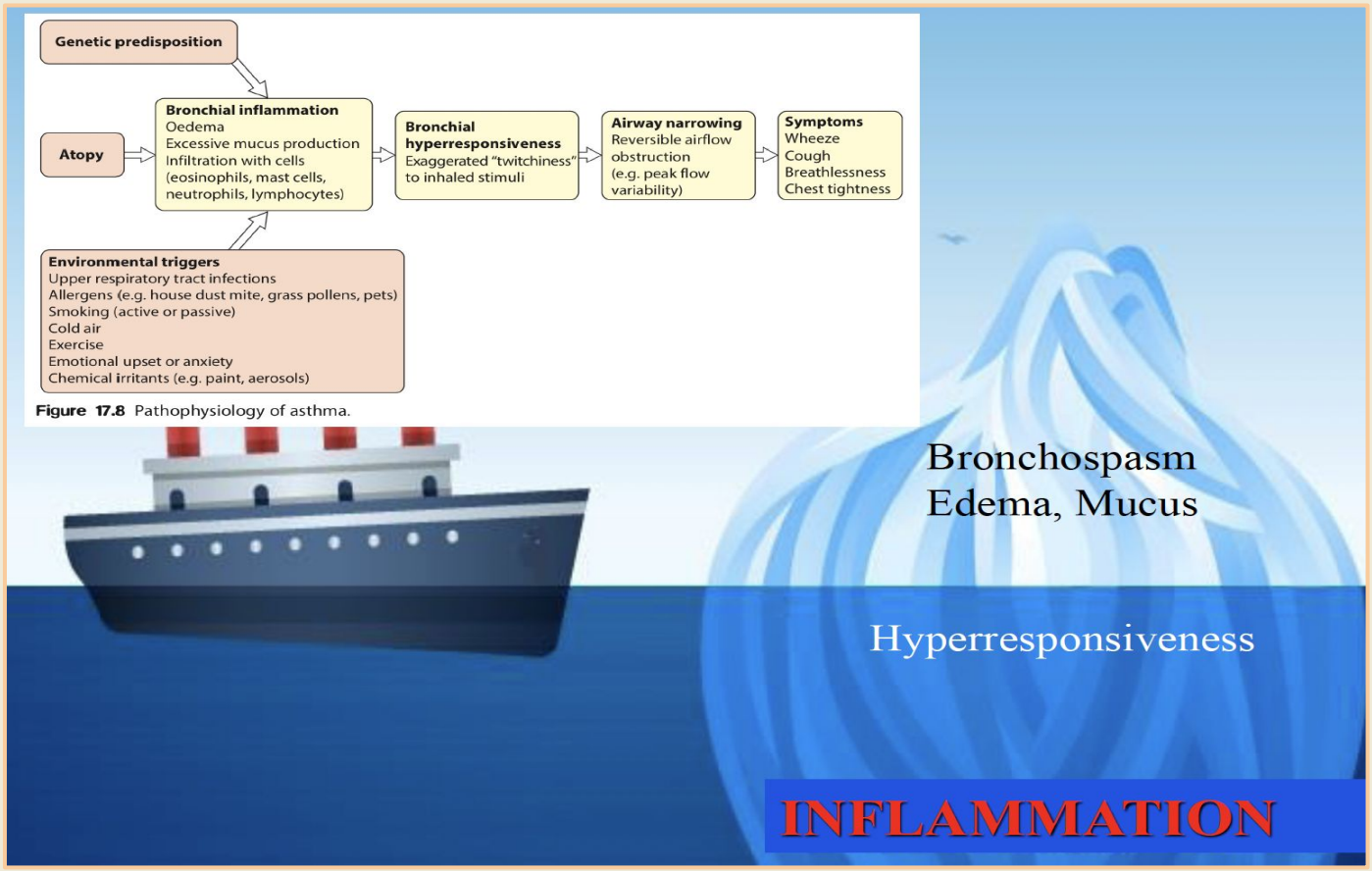
Cells & mediators:

- Hypertrophied muscle happens after recurrent bronchospasm. it is irreversible.





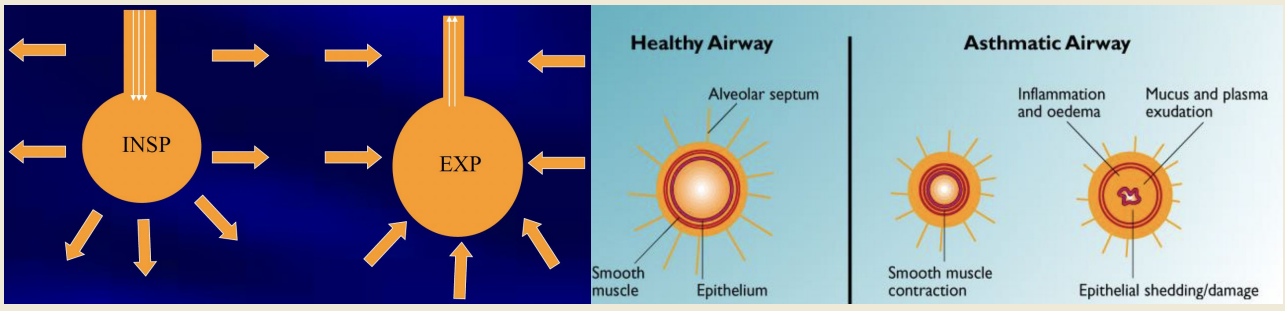
Pathophysiology:



Asthma:

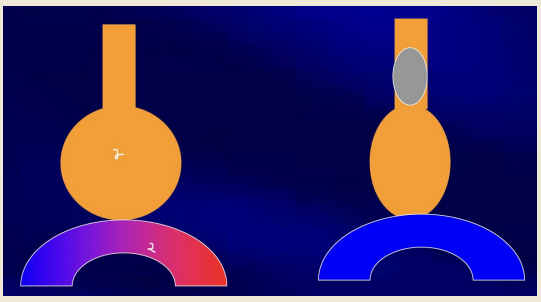
Pathophysiology cont.

- **AIR TRAPPING:** Asthma mainly affects the lower (intra-thoracic) airways:
  - Inspiration: air enters the lung normally..
  - **Expiration:** airway collapse → air won't be able to go out يدخل هوا اكثر مما يطلع → air trapping → → air leak (lung is full) → pneumothorax, pneumomediastinum, subcutaneous emphysema.
  - The hyperinflation will also compromise the circulation by decreasing the cardiac output.



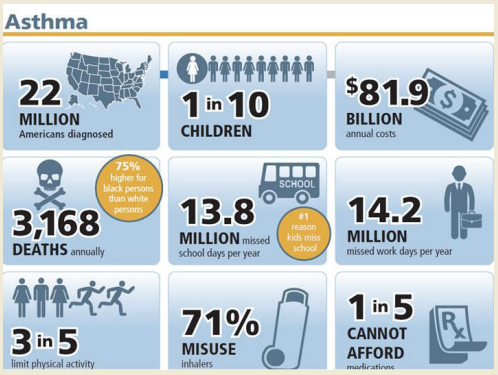
Ventilation Perfusion (V/Q) mismatch:

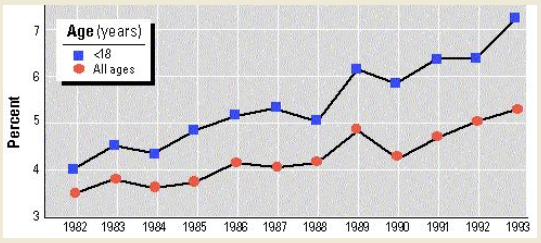
➔ **Obstruction** by mucus prevents blood from getting oxygen → **hypoxia due to V/Q mismatch.**



Burden of Asthma:

- Asthma is one of the most common chronic diseases worldwide with an estimated 300 million affected individuals.
- Prevalence increasing in many countries, esp. in children (10-15%).
- A major cause of school/work absence.
- Here in SA, Asthma prevalence is 10-15%.





Factors that Influence Asthma Development and Expression:

- Host Factors: [Genetic; Atopy – Airway hyperresponsiveness], Gender, Obesity.
- Environmental Factors: Indoor allergens, Outdoor allergens, Occupational sensitizers, Tobacco smoke, Air Pollution, Respiratory Infections, Diet.
- Allergen can be:
  - Biological (cats, pollen, horses).
  - Irritant: not biological (smoke, dust).

Asthma:

Environmental allergens & childhood asthma:

- **Dust mites:** house dust mites & يعيش في الستاير، المراتب causes allergy by its fecal material which the child inhales while sleeping.
  - tell the parents put the mattress in an airtight cloth.
- Furry pets: Their secretions will remain months before they get cleared after you remove the pets.
- Molds – Cockroaches
- **Cigarette Smoking [irritant].**
- Pollens: more likely to cause allergic rhinitis because it gets filtered by the nose.

History : [Hx is Imp in OSCE]***

- Symptoms (cough, wheeze, SOB).
- Onset, duration, frequency, severity.
- Activity and nocturnal exacerbation.
- Previous therapy
- Triggers
- Other atopies.
- Family history.
- Environmental history [**SMOKING**].
- Systemic review.
- Attack of status asthmaticus.
- Hospital admissions.
- Adherence to therapy — Medications (they know how to use it?).
- Usually the child is normal between attacks.

Key features of asthma:
❑ Worse at night & early morning
❑ Nonviral triggers
❑ History of atopy
❑ Interval symptoms (between acute exacerbations)
❑ Positive response to asthma therapy

PE:

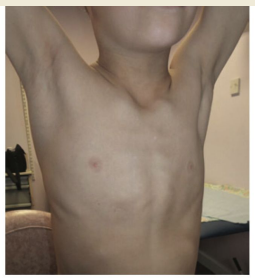


Figure 17.9 The depressions at the base of the thorax associated with the muscular insertion of the diaphragm are called Harrison's sulci, and are associated with chronic obstructive airways disease such as asthma during childhood from chronic increased work of breathing.

- Growth parameter: Normal unless asthma is severe.
- ENT.
- Features of atopy.
- Chest findings: Normal between attacks.
- **PEF:** *[The most imp. Pts **under 6 years** can't do it.]*
  - Young pts: history & exam alone is diagnostic.
  - PEF is less sensitive than spirometry, but it's helpful (portable & allows for serial measurements).
  - Poorly controlled asthma leads to increased variability; both diurnal & day-to-day.

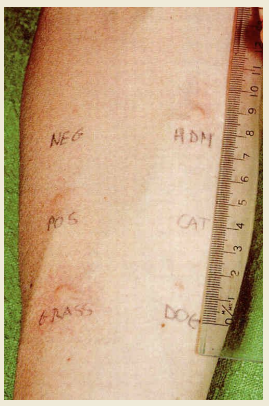
Asthma:

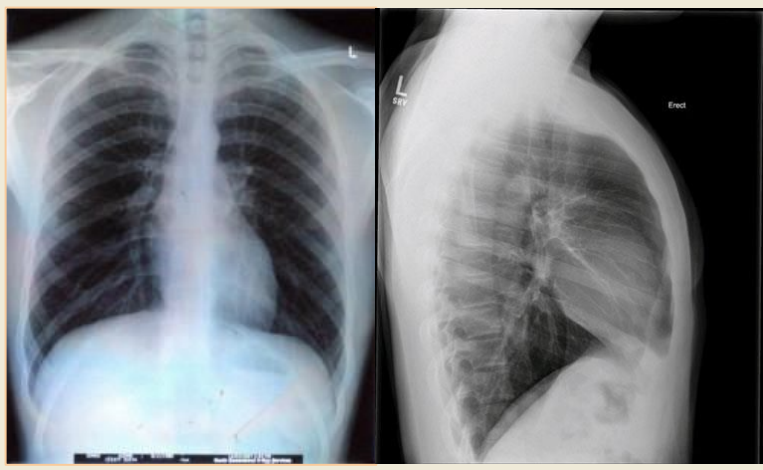
➔ **When do you suspect a chronic infection rather than asthma?**
(ex: CF, bronchiectasis)

- **Poor growth.**
- **Clubbing: Asthma doesn't cause clubbing** (clubbing comes with suppurative lung disease).
- **Wet/productive cough.**

Investigations:

- Pulmonary Function Test.
- Chest X ray in some.
- Allergy testing in some.
- Skin testing,,

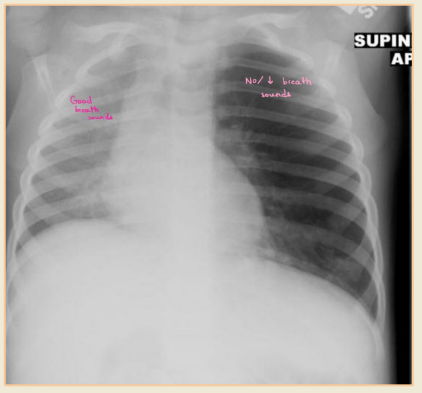




Hyperinflation during the acute attack (flattening of the diaphragm & bowing of the chest)

Differential Diagnosis: ***Important to differentiate*

- ❖ Infections (bronchiolitis) RSV
- ❖ Congenital Heart Disease (engorgement of vessels → airway obstruction → wheeze).
- ❖ GER
- ❖ Bronchopulmonary dysplasia
- ❖ Structural anomalies.
- ❖ **Anaphylaxis: (urticaria, facial swelling, stridor).**
- ❖ **Foreign body ⇒ Usually food;**
  - ➢ **Suspect: cough + normal X-ray + unilateral wheezing + previously well.** wheeze or choking.
  - ➢ **Exam: wheeze or stridor, usually unilateral so compare laterally.**
  - ➢ **You can't see it by x-ray but you will see the effect: "air trapping and hyperinflation distal to the obstruction".**
  - ➢ **Xray 90% will be radiolucent because its food. Opposite for esophageal FB****
  - ➢ If you have to do a single film for intrathoracic obstruction **⇒ do expiratory;**
  - ➢ **Expiratory x-ray look for hyperinflation for foreign body.**
  - ➢ This X-ray is for a child with acute wheeze & cough;
    - findings:
      - Deflated right side/ inflated left side.
      - On expiration, the lung should get smaller
      - The left lung cannot deflate due to obstruction
      - by a FB in the left main bronchus



  - ➢ **Remove by bronchoscopy** or you will lose the lung (airway swelling causes complete obstruction & lobar collapse). **Bronchoscopy diagnostic and treatment.**

Asthma:

Management of Asthma:

★ Treatment objectives:

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

★ Treatment Strategy:

1) Develop Patient/Doctor Partnership.
2) Identify and Reduce Exposure to Risk Factors.
3) Assess, Treat and Monitor Asthma.
4) Manage Asthma Exacerbations.
5) Special Consideration.

Why don't patients comply with treatment?

❖ **Intentional:**

- Feel better
- Fear of side effects
- Don't notice any benefit
- Fear of addiction
- Fear of being seen as an invalid
- Too complex regimen
- Can't afford medication

❖ **Unintentional:**

- Forget treatment
- Misunderstand regimen / lack information
- Unable to use their inhaler
- Run out of medication

Levels of Asthma Control

Characteristic	Controlled (All the following)	Partly Controlled (Any present in any week)	Uncontrolled
Daytime symptoms	None (2 or less/week)	More than twice/week	** OSCE 3 or more features of partly controlled asthma present in any week
Limitations of activities	None	Any	
Nocturnal symptoms	None	Any	
Need for rescue /"reliever" Tx	None (2 or less /week)	More than twice /week	
Lung function (PEF or FEV)	Normal	<80% predicted or personal best (if known) on any day	
Exacerbation	None	1 or more /year	1 in any week

➔ Did it need admission or steroids? **Any exacerbation in the last 6 months that needed steroids indicates uncontrolled asthma.**

Asthma:

Level of Control		Treatment of Action
Controlled	REDUCE	Maintain and Find lowest controlling step
Partly Controlled		Consider stepping up to gain control
Uncontrolled	INCREASE	Step up until controlled
Exacerbation		Treat as exacerbation

➔ how to assess the level of control? ask about daytime symptoms/week, nighttime symptoms/month, reliever use/week, physical limitation, exacerbation/year.

Pharmacological therapy:

- Relievers:
  - **Inhaled fast-acting β2–agonist**
    (Effective for 2-4 hours. Used PRN for increased symptoms or in high doses in exacerbations).
  - **Inhaled anticholinergics.**

- Controllers: **(must be taken regularly)**
  - **Inhaled corticosteroids**
  - **Inhaled long-acting β2–agonist**
    - **with ICS, never used alone**
    - **Not used in exacerbations.**
    - **Useful in exercise induced.**
  - Inhaled cromones
  - Oral anti-leukotrienes
  - Oral theophyllines
  - Oral corticosteroids
    - **[Prednisone is given on alternate days in severe persistent asthma where other treatments have failed].**

Table 17.2 Drugs in chronic asthma

Type of drug	Drug
Bronchodilators	
β_2-agonists (relievers)	Salbutamol
	Terbutaline
Anticholinergic bronchodilator	Ipratropium bromide
Preventer therapy	
Inhaled steroids	Budesonide
	Beclometasone
	Fluticasone
	Mometasone
Long-acting β_2-agonists (LABA)	Salmeterol
	Formoterol
Methylxanthines	Theophylline
Leukotriene receptor antagonists (LTRA)	Montelukast
Oral steroids	Prednisolone
Anti-IgE monoclonal antibody	Omalizumab

Cromolyn Sodium no longer used

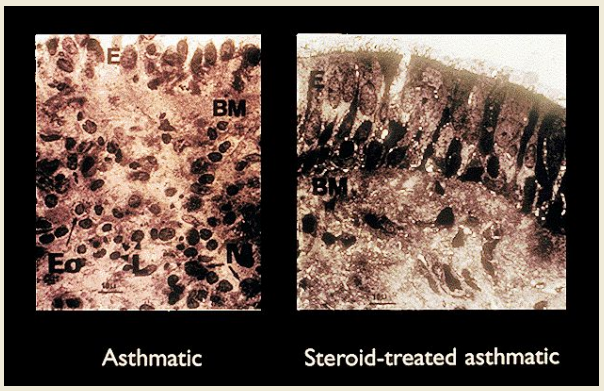
- Non-steroidal anti- inflammatory
- Weak action on Early and late phases
- Slow onset of action
- If no response in 6 weeks change to ICS
- Side effects: Irritation

Asthma:

Management of Asthma:

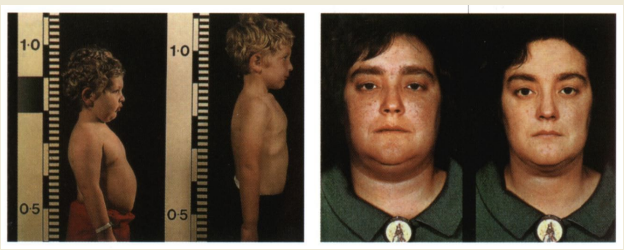
TREATMENT STEPS				
*STEP **1***	*STEP **2***	*STEP **3***	*STEP **4***	*STEP **5***
Asthma education				
Environmental control				
As needed rapid-acting β2–agonist	As needed rapid-acting β2–agonist			
CONTROLLER OPTION	*SELECT ONE*	*SELECT ONE*	*ADD ONE or MORE*	*ADD ONE or BOTH*
	Low-dose ICS* **[Tx of choice]**	Low-dose ICS *plus* Long-acting β2–agonist	medium/high–dose ICS *plus* Long-acting β2–agonist	Oral corticosteroid [lowest dose]
	Leukotriene modifier**	medium/high–dose ICS	Leukotriene modifier	Anti-IgE
	–	Low-dose ICS *plus* Leukotriene modifier	sustained-release theophylline	–
	—	Low-dose ICS *plus* sustained-release theophylline	–	–

Inhaled Corticosteroids:



- Effective in most cases – Safe especially at low dose.
- The anti-inflammatory of choice in Asthma.

- **Side effects:**
  - Growth: No significant effect at low to moderate doses.
    - High dose of steroids can decrease growth by 2cm, imp to monitor growth
  - Bones: not important.
  - HPA axis: No serious clinical effect (high doses) could cause adrenal insufficiency
  - **Alteration of glucose and lipid metabolism:**
    - Clinical significant is unclear (high doses).
  - Cataract: No increase risk
  - **Skin: Purpura, easily bruising, dermal thinning.**
  - Local side effects.



Asthma:

Management of Asthma:

A stepwise approach to the treatment of chronic asthma

Move up steps to improve control as needed

Move down to determine the lowest controlling step

Step 5: Continuous or frequent use of oral steroids
In **children 5–12 years:** maintain ICS at 800 µg/day.
Use lowest possible daily dose of oral steroids to maintain adequate control. Refer to respiratory paediatrician
In **adolescents and young adults**: maintain ICS at 1600 µg/day. Use lowest possible daily dose of oral steroids to maintain adequate control. Refer.

Step 4: Persistent poor control
In **<5 years:** refer to respiratory paediatrician
In **5–12 years:** increase ICS to 800 µg/day
In **adolescents and young adults:** increase ICS to 1600 µg/day and consider LTRA, or SR theophylline
Issue steroid replacement warning card.

Step 3: Initial add-on therapy
In **<5 years:** add LTRA; if poor response, increase ICS to 400 µg/day.
>5 years and young adults: initially add inhaled long-acting β_2-agonist (LABA). Assess response:
1st choice
- Good response—LABA
- Partial response—increase ICS to 400 µg/day [800 in adolescents and young adults].
- Poor response—stop LABA, increase ICS to 400 µg/day [800 µg in adolescents and young adults] and consider LTRA and/or slow release (SR) theophylline.

Step 2: Regular preventer therapy
In all ages: add inhaled corticosteroid 200 µg/day or in those <5 years consider leukotriene receptor antagonist (LTRA) if inhaled corticosteroid cannot be used. In adolescents and young adults doses of up to 400 µg/day may be used at this step.
Monitor height and weight of all children on asthma treatment.

Step 1: Mild intermittent asthma
In all ages: inhaled short-acting β_2-agonist as required

Periodic assessment of the child with asthma

Clinical features to check

Growth and nutrition

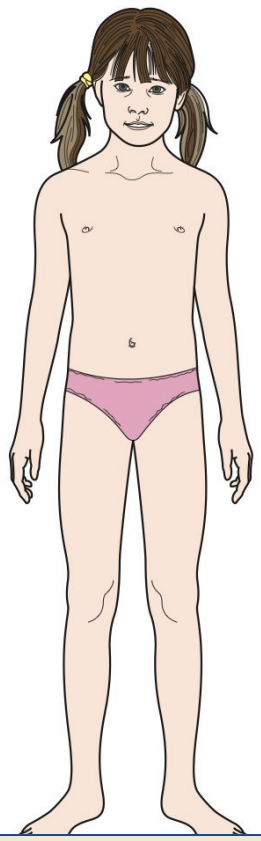
Peak flow/spirometry

Chest for:
- Hyperinflation
- Harrison's sulcus
- Wheeze

Are there other allergic disorders?
- Allergic rhinitis
- Eczema
- Food allergy

If atypical features present:
- Sputum
- Finger clubbing
- Growth failure

- then seek another diagnosis



Monitor:
- Peak flow diary
- Severity and frequency of symptoms
- Exercise tolerance
- Interference with life, time off school
- Is sleep disturbed?
- Use of preventer and reliever medication – are they appropriate?
- Inhaler technique
- Lung function tests

Consider triggers:
- Untreated allergic rhinitis
- Allergens or cigarette smoke
- Stress

Check:
- Child has an up to date personalized asthma management action plan
- Family have necessary medication/ equipment to manage an acute exacerbation

Acute Asthma:

History:

- Symptoms (duration of the attack)
- Previous attacks
- Prior therapy
- Triggers.

PE:

- **Signs of airway obstruction:**
  - Fragmented speech
  - Unable to tolerate recumbent position
  - Expiration > 4 seconds.
  - Tachycardia, tachypnea and hypotension
  - Use of accessory muscles.
  - Pulsus paradoxus > 10 mmhg
  - Silent hyperinflated chest
  - Air leak.

- **Signs of tissue hypoxia:**
  - Cyanosis.
  - Cardiac arrhythmia and hypotension.
  - Restlessness, confusion, drowsiness and obtundation.

- **Signs of Respiratory muscles fatigue:**
  - Increased respiratory rate.
  - Respiratory alternans (alteration between thoracic and abdominal muscles during inspiration).
  - Abdominal paradox (inward movement of the abdomen during inspiration).

Investigations:

- Peak expiratory flow rate
- **Pulse oximetry.**
- ABG
- CXR
  - ONLY IN FEW CASES ⇒ **Do CXR <u>if there's unusual features</u> (asymmetric chest rise suggesting pneumothorax or lobar collapse), signs of severe infection /no response to tx.**
  - **= ماهي روتين نسويها، فقط في الحالات التي تتطلب.**

Acute Asthma:

Management of Acute Asthma

The first hour:

1) **Oxygen:**
   a) Hypoxemia is common — Monitor saturation.
   b) It worsens airway hypersensitivity.
2) **Inhaled β2–agonist:**
   a) **Every 20 minutes in the first hour.**
   b) **Assess after each nebulizer.**
   c) **You can use spacer to avoid infections particularly covid-19.**
3) **Steroids:**
   a) If not responding to the β2–agonist.
   b) If severe; start from the beginning; oral steroids;
   [Don't wait because it takes 3-4 hours to work].
   c) If on PO prednisone or high dose inhaled steroids.
   d) Previous severe attacks.

by spacer (better).

Nebulizer (severe).

➔ **Note-well [IV therapy]:**

- Inhaled therapies are not always be successful as the drugs may be delivered in suboptimal doses to areas of the lung that are poorly ventilated.
- **IV therapy is given to those fail to respond inhaled bronchodilator therapy.**
- Magnesium sulphate, aminophylline, or intravenous salbutamol are all potentially beneficial.

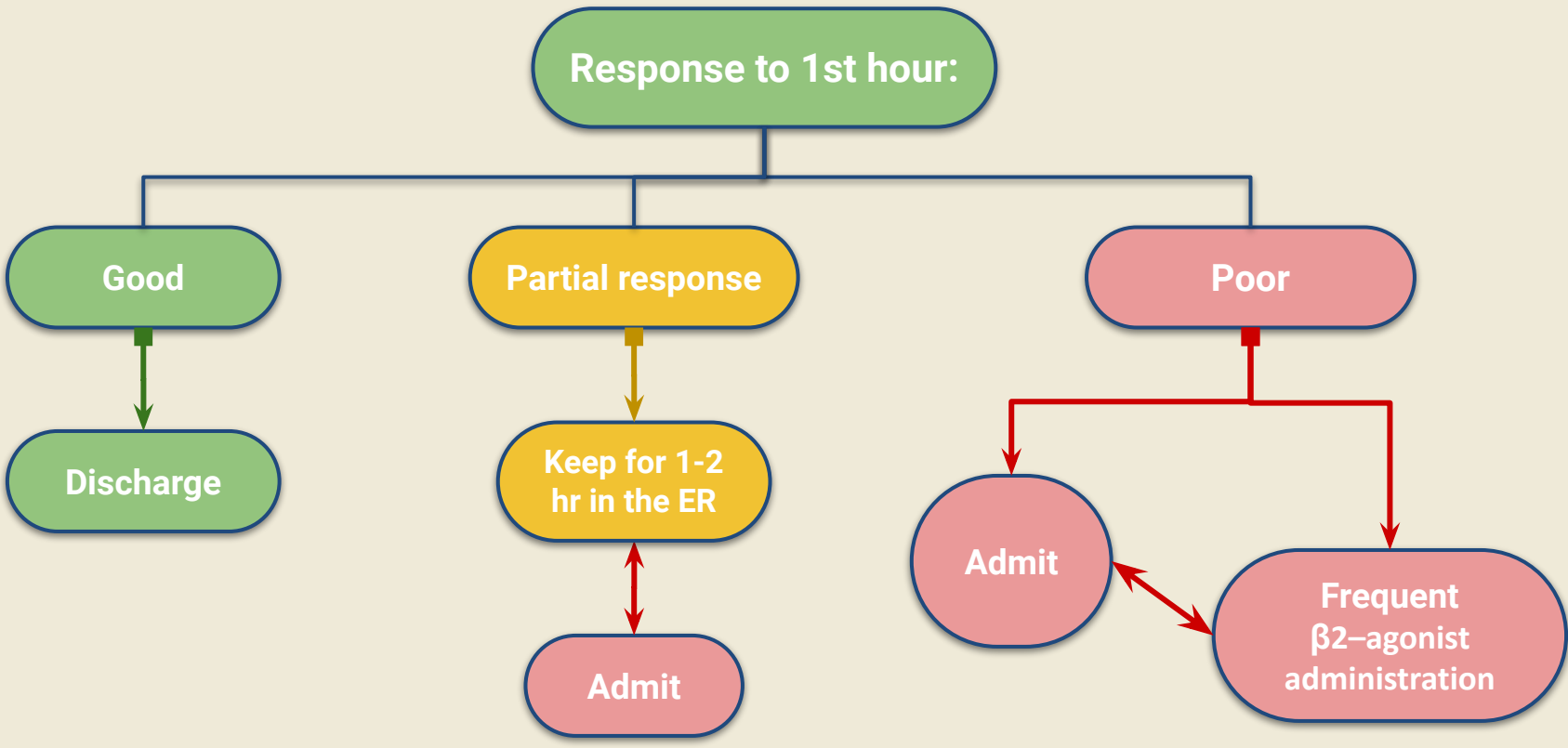
★ **Magnesium sulphate [IV] probably has the least side-effects and most evidence of benefit, and is increasingly being used as the first choice for intravenous therapy.**

- With both aminophylline and salbutamol, the ECG should be monitored and blood electrolytes checked.

➔ **Ipratropium Bromide [inhaled]:**

- Not useful — anticholinergic — For severe cases — along with β2–agonist.

Response to first hour;



Acute Asthma:

Criteria for admission:

- **(if, after high dose inhaled bronchodilator, they):**
  - Have not responded adequately clinically (still breathless, tachypnic).
  - Becoming exhausted.
  - Have marked reduction in their predicted (or usual best) PFR or FEV1 (<50%).
  - SpO2 < 92% in air.

Discharge

- Follow up.. When to come back?
- Give inhaled β2 agonist [with or without ICS].
- Steroids.

Determining severity of acute asthma

Determine the severity of the attack (see Fig. 17.14):
- Mild
- Moderate
- Severe
- Life-threatening

This is determined by clinical features shown.

Too breathless to talk – severe

Increased work of breathing
Check respiratory rate:
- Tachypnoea – varies with age; poor guide to severity

Chest recession:
- Moderate – some intercostal recession
- Severe – use of accessory neck muscles
- Life-threatening – poor respiratory effort

Auscultation:
- Wheeze
- Silent chest – poor air entry from poor expiratory effort or exhaustion in life-threatening

Cardiovascular:
- Tachycardia – varies with age; better guide to severity than respiratory rate but affected by β_2-agonists
- Arrythmia, hypotension – life-threatening

Altered consciousness, agitation or confusion – in life-threatening
Exhaustion – life-threatening

Tongue:
- Cyanosis in life-threatening

Peak flow (% predicted or best or usual measurement):
- Moderate >50%
- Severe 33–50%
- Life-threatening <33%

O_2 saturation:
- Moderate ≥92%
- Severe or life-threatening <92%

Is there a trigger for the attack?:
- URTI or other viral illness
- Allergen, e.g. animal dander
- Exercise
- Cold air

Causes of acute breathlessness in the older child:
- **Asthma**
- **Pneumonia or lower respiratory tract infection**
- **Foreign body**
- **Anaphylaxis**
- **Pneumothorax or pleural effusion**
- **Metabolic acidosis – diabetic ketoacidosis, inborn error of metabolism, lactic acidosis**
- **Severe anaemia**
- **Heart failure**
- **Panic attacks (hyperventilation).**

Figure 17.13 Assessment of the child or young person with acute asthma to determine severity of the attack.

Acute Asthma:

Assessment and management of acute asthma

Assess severity

Moderate
- Able to talk
- Oxygen saturation >92%
- Peak flow >50% [best]
- Respiratory rate
  ≤40 breaths/min for 2–5 year
  ≤30 breaths/min for 5–12 year
  ≤25 breaths/min for 12–18 year
- Heart rate
  ≤140 beats/min for 2–5 year
  ≤125 beats/min for 5–12 year
  ≤110 beats/min for 12–18 year

Severe
- Too breathless to talk
- Oxygen saturation <92% for <12 year olds
- Peak flow 33%–50% [best]
- Respiratory rate
  >40 breaths/min for 2–5 year
  >30 breaths/min for 5–12 year
  >25 breaths/min for 12–18 year
- Heart rate
  >140 beats/min for 2–5 year
  >125 beats/min for 5–12 year
  >110 beats/min for 12–18 year

Life-threatening
- Silent chest, cyanosis
- Poor respiratory effort
- Exhaustion
- Arrhythmia, hypotension
- Altered consciousness
- Agitation, confusion
- Peak flow <33% [best]
- Oxygen saturation <92% (all ages)

Management

High flow oxygen (if available)

- Keep calm and reassure child and parents
- Short-acting β_2-agonist via spacer (with face mask for those under 3), 2–4 puffs, increasing by 2 puffs every 2 min to 10 puffs if required
- Oral prednisolone 1–2 mg/kg, maximum 40 mg
- Monitor response for 15–30 min

- Short-acting β_2-agonist via spacer, 10 puffs or nebulized (2.5 mg salbutamol in <8 years, 5 mg in >8 years), assess response and repeat as required
- Oral prednisolone or IV hydrocortisone

Consider:
- Inhaled ipratropium
- IV β_2-agonist (salbutamol) or aminophylline or magnesium

- Short-acting β_2-agonist nebulized (2.5 mg salbutamol in <8 years, 5 mg in >8 years), assess response continuously and repeat as required (back to back)
- Oral prednisolone or IV hydrocortisone
- Nebulized ipratropium

Consider:
- IV β_2-agonist (salbutamol) or aminophylline or magnesium

Discuss with PICU

Assess response to treatment

Responding:
- Continue bronchodilators 1–4 h prn
- Discharge when stable on 4-h treatment
- Continue oral prednisolone for 3–7 days

At discharge:
- Review medication and inhaler technique
- Provide personalized asthma action plan
- Arrange appropriate follow-up

Not responding:
- Transfer to HDU/PICU
- Ensure senior medical review
- Consider IV therapies not already used (magnesium, aminophylline, β_2-agonist)
- Consider CXR (to check for pneumothorax or infection) and blood gases
- Consider need for mechanical ventilation

Questions

1- Amir, a 4-year-old boy, presents to his family doctor with a history of eczema, rhinitis, chronic nocturnal cough and intermittent wheeze.
Asthma is suspected and a bronchodilator is prescribed.
How should his bronchodilator be delivered?
A. Dry powder inhaler
B. Metered dose inhaler (MDI)
C. Metered dose inhaler with large-volume spacer
D. Nebulizer
E. Syrup

2- Sarah, a 10-year-old girl, has frequent attacks of asthma. She attends the Emergency Department with increasing difficulty in breathing over the last 12 hours. Initial observation shows that she is anxious, sitting upright, has a marked tracheal tug and is unable to complete a sentence.
Which of the following statements is most likely to be correct?
A. Sarah's asthma attack is of moderate severity
B. Sarah's condition is likely to improve if she is encouraged to lie flat
C. Sarah's oxygen saturation should be measured
D. Sarah should be taken promptly to the X-ray department for a chest X-ray
E. The lack of wheeze should make you consider a panic attack

3- Zak, a 3-year-old boy, is seen by his general practitioner because of recurrent wheezing associated with upper respiratory tract infections.
Which of the following features most supports the diagnosis of asthma?
A. Daytime cough
B. Finger clubbing
C. Peak-flow variability diary
D. Persistent moist cough
E. The presence of symptoms between coughs and colds

4- Boris, a 5-month-old infant from Poland, is admitted to hospital with breathing problems and poor feeding. On examination he has a respiratory rate of 50 breaths/min. On auscultation of the chest he has widespread crackles. He has moderate intercostal recession, and oxygen saturation of 92% in air. He was born at term with a birthweight of 3.6 kg (50th centile). His weight is now 5.2 kg (<0.4th centile). This is his first admission to hospital but he 'is always chesty'. You suspect Boris has cystic fibrosis.
When he is stabilized **which would be the most appropriate investigation to perform?**
A. Genetic screening for cystic fibrosis
B. Heel prick for immunoreactive trypsin
C. Measurement of faecal elastase
D. Measurement of serum bilirubin
E. Sweat test

Answers

1- **C.** Metered dose inhaler with large-volume spacer
The best mode of delivery is direct to the lungs. Children under 5 years of age should be prescribed a MDI with spacer as they cannot coordinate an MDI alone.

2- **C.** Sarah's oxygen saturation should be measured
Oxygen saturation should be measured to further assess the severity of the asthma attack and to guide treatment.

3- **E.** The presence of symptoms between coughs and colds
The presence of interval symptoms and atopic conditions (eczema/hay fever) helps to distinguish asthma from viral-induced wheeze.

4- **E.** Sweat test
Abnormal function of the sweat glands results in excessive concentrations of sodium and chloride in the sweat, and this is the basis of the essential diagnostic test for cystic fibrosis.