Approach to a wheezy infant
Wheeze is a continuous & musical sound that originates from oscillations in narrowed airways, mostly heard in expiration due to critical airway obstruction.
Wheezes are heard more commonly during expiration. Wheezing during expiration alone = milder obstruction. Wheezing during both inspiration and expiration = more severe airway narrowing. Wheezing may be acute or chronic /recurrent. Sign of lower (intra-thoracic) airway obstruction.
Infants & children are prone to wheeze as compared to older children & adults. Why?
Resistance: small caliber peripheral airways can contribute up to 50% of airway resistance.

Compliant chest walls, especially in newborns, lead to intrathoracic airway collapse due to inward pressure produced in expiration.

Differences in tracheal cartilage composition & airway muscle tone causes further increase in airway compliance.

Immunologic & molecular influences:
- Infants have increased levels of lymphocytes & neutrophils in BAL.
- Inflammatory mediators have been implicated eg. Histamine, leukotrienes, interleukins, etc.
All these mechanisms combine to make the Infant more susceptible to airway collapse

- Increased resistance
- Subsequent wheeze

Many of these are outgrown in the 1st year of life itself
A. Polyphonic
1. Various tone
2. Widespread narrowing of airways
3. Asthma

B. Monophonic
1. Single-tone
2. Narrowing of larger airways
3. Tracheomalacia

wheezing
Wheezing phenotypes classification

1- epidemiological

2- symptom-based types.
symptom-based types.

A. episodic wheeze

B. multiple-trigger wheeze.
Episodic wheeze

- Usually triggered by viral infection.
- The child is well between episodes.
- Conditions cease by adolescence.
usually triggered by many factors, such as virus, cigarette smoke, crying, laughing, or allergens.
- wheezing during exacerbation
- between episodes.
- It is usually associated with an allergy.
- Likely to be asthmatic.

**multiple-trigger wheeze.**
<table>
<thead>
<tr>
<th>Tucosn children respiratory study wheezing phenotype and natural history</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Never wheezed</strong>&lt;br&gt; (51%)</td>
</tr>
<tr>
<td><strong>Transient early wheezers</strong>&lt;br&gt; (20%)</td>
</tr>
<tr>
<td><strong>Persistence wheezers</strong>&lt;br&gt; (14%)</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Late-onset wheezers</strong>&lt;br&gt; (15%)</td>
</tr>
</tbody>
</table>
Transient early wheezer

- Commonest form of wheeze
- Onset during the first two to three years of life
- Is linked to decrease lung function at birth
- No airway hyper-responsiveness
- It is not associated with atopy or asthma
- No immune responses to viruses
- Resolves by 3 years
  - Wheeze in first year – better outcome
  - Wheeze 2-3 year – worse outcome due to maturity of immune system

- Affected by:
  - Teenage pregnancy & smoking
  - More common in Male gender
  - Day care - infections
Onset ≤3 years then persisting
- Initial risk factors include passive smoke exposure, maternal asthma history and elevated IgE level in the first year of life to inhalant allergens
- Lung function normal at birth but deteriorates with recurrent symptoms
- Increased symptoms with increasing age
- Abnormal immune responses to viruses
- Associated with other atopic disease such as eczema, and food allergy
- An increase risk of developing clinical asthma
Lung function abnormal at birth and reduced in later life

It is not associated with allergy. Non Atopic

Airway hyper-responsiveness resolves by mid-childhood.

RSV induced wheeze due to alteration in airway tone

Better outcome than atopic persistent wheezers
15% of population

No wheezing by 3yrs

wheezing by 6yrs
Acute wheezing (hours to days)

1. Asthma
2. Foreign body aspiration
3. Bronchiolitis
4. Bronchitis
5. Laryngotracheobronchitis
6. Bacterial tracheitis
### Chronic or recurrent wheezing

**Functional causes**

1. Asthma  
2. Gastroesophageal reflux  
3. Recurrent aspiration  
4. Cystic fibrosis  
5. Immunodeficiency  
6. Primary ciliary dyskinesia  
7. Bronchopulmonary dysplasia  
8. Retained foreign body (trachea or esophagus)  
9. Bronchiolitis obliterans  
10. Pulmonary edema  
11. Vocal cord dysfunction  
12. Interstitial lung disease

**Structural abnormalities**

1. Tracheo-bronchomalacia  
2. Vascular compression/rings  
3. Tracheal stenosis/webs  
4. Cystic lesions/masses  
5. Tumors/lymphadenopathy  
6. Cardiomegaly
Clinical history

- Is it wheezing?
- Age at onset
- Course: acute vs gradual
- Pattern of wheezing? Episodic: asthma     Persistent: congenital
- Response to bronchodilators?
- Is Wheezing associated with multiple systemic illnesses?       Cystic fibrosis and Immunodeficiency diseases
- Wheeze associated with feeding?
- Wheeze associated with cough?
- Change in position? Worsening or improvement
- Birth history: What was the gestational age at delivery? NICU admission, h/o intubation /O2 requirement
- Family hx of asthma?
<table>
<thead>
<tr>
<th>Question</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>How old was the patient when the wheezing started?</td>
<td>Distinguishes congenital from noncongenital causes</td>
</tr>
<tr>
<td>Did the wheezing start suddenly?</td>
<td>Foreign body aspiration</td>
</tr>
<tr>
<td>Is there a pattern to the wheezing?</td>
<td>Episodic: asthma</td>
</tr>
<tr>
<td></td>
<td>Persistent: congenital or genetic cause</td>
</tr>
<tr>
<td>Is the wheezing associated with a cough?</td>
<td>GERD, sleep apnea, asthma, allergies</td>
</tr>
<tr>
<td>Is the wheezing associated with feeding?</td>
<td>GERD</td>
</tr>
<tr>
<td>Is the wheezing associated with multiple respiratory illnesses?</td>
<td>Cystic fibrosis, immunodeficiency</td>
</tr>
<tr>
<td>Is the wheezing associated with a specific season?</td>
<td>Allergies: fall and spring</td>
</tr>
<tr>
<td></td>
<td>Croup: fall to winter</td>
</tr>
<tr>
<td></td>
<td>Human bocavirus*</td>
</tr>
<tr>
<td></td>
<td>Human metapneumovirus: December through April</td>
</tr>
<tr>
<td></td>
<td>RSV: fall to spring</td>
</tr>
<tr>
<td></td>
<td>Tracheomalacia, anomalies of the great vessels</td>
</tr>
<tr>
<td>Does the wheezing get better or worse when the patient changes position?</td>
<td>Infections, allergic triad</td>
</tr>
</tbody>
</table>

*GERD = gastroesophageal reflux disease; RSV = respiratory syncytial virus.*
Physical Examination

- Measurement of Weight and Height
- Vital signs including SpO2 %, RR
- Chest examination
  - Inspection:
    - Respiratory distress
    - Chest wall deformity (increased AP diameter)
    - Allergic shiners/nasal polyps
  - Skin: eczema
  - Palpation: chest wall asymmetry with expansion
    tracheal deviation
  - Percussion: difference in vocal resonance
  - Auscultation:
    - Location of wheeze
    - Character of wheeze
    - Other breath sounds associated with wheeze
- Cardiac: presence of murmur
- Assessment of response to therapy suggestive of asthma
The infant with tachypnoea or wheeze

Clinical features to assess

- Breathing:
  - Too breathless to feed?
  - Respiratory rate – > 40 breaths/min
  - Chest hyperexpansion
  - Chest recession
  - Use of accessory muscles
  - Expiratory grunting, nasal flaring
  - Auscultation, percussion
    - abnormal signs

- Heart:
  - Heart rate – > 160 beats/min
  - Heart murmur

- Fever
- Level of consciousness
- Weight loss?
- Exhaustion?
- Apnoea?

- Cyanosis
- Hepatomegaly
- O₂ saturation
Features in the history that favor the diagnosis of asthma include:

- Intermittent episodes of wheezing that usually are the result of a common trigger (ie, upper respiratory infections, weather changes, exercise, or allergens)

- Seasonal variation

- Family history of asthma and/or atopy

- Good response to asthma medications

- Positive asthma predictive index
Features suggestive of a diagnosis other than asthma in children

- Onset of symptoms in early infancy
- Neonatal respiratory distress +/- ventilator support
- Clubbing
- Cardiac murmur
- Intractable wheezing unresponsive to bronchodilators
- Wheezing associated with feeding or vomiting
- Difficulty swallowing +/- recurrent vomiting
- Diarrhea
- Poor weight gain
- Stridor
- Oxygen requirement >1 week after acute attack
Investigations

1- CXR: AP and lateral views

- Children with new onset wheezing of undetermined etiology
- Chronic persistent wheezing not responding to treatment
- Suspected FB aspiration

CXR findings:

- **Hyperinflation:**
  - Generalized: suggests diffuse air trapping  Asthma/ Cystic fibrosis
  - Localized hyperinflation: Structural abnormalities/ FB aspiration

- atelectasis
- bronchiectasis
- mediastinal masses
- cardiomegaly
Investigations

2- Chest CT scan:
   - Mediastinal masses or LN’s
   - Vascular anomalies
   - Bronchiectasis

3- Barium Swallow:
   - GERD
   - TEF
   - Vascular rings
   - Swallowing dysfunction

4- Pulmonary Function Tests (PFT’s)
   - Airway obstruction assessment
Other investigations

5- Sweat Chloride Test:
Cystic fibrosis screening in children with chronic lung problems, failure to thrive and diarrhea

6- Immunoglobulin levels:
Screen for immunodeficiency.

7- viral studies

8- sputum gram stain and culture.

9- Endoscopy

10- 24 hour esophageal pH monitoring
Asthma Predictive Index

Asthma Predictive Index – Clinical index used to predict occurrence of asthma in later childhood.
High-risk children (under age three) who have had four or more wheezing episodes in the past year that lasted more than one day are much more likely to have persistence asthma after the age of 5 years.
The criteria include $\geq 3$ wheezing episodes per year

**PLUS**

1 major criteria
- Parental asthma
- Atopic dermatitis
- Inhalant allergen sensitization

**OR**

2 minor criteria
- Allergic rhinitis
- Wheezing apart from cold
- Eosinophilia $\geq 4\%$
- Food allergen sensitization

Fig 2—The asthma predictive index.
If the API is positive, there is a 77% certainty that the patient will have atopic asthma at school age.

If there are no findings and the API is negative, there is a 95% chance of not having asthma during school years.

Children with positive API have 7 times more risk of asthma at school age.
Differential Diagnosis

wheezy infant
Differential Diagnosis

- Acute bronchiolitis
- Foreign body aspiration
- Cardiac
- GERD
- Pneumonia
- Vascular ring
- Bronchial asthma
Bronchiolitis
Bronchiolitis
	a lower respiratory tract infection that primarily affects the small airways (bronchioles)
# Incidence

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age</th>
<th>Food</th>
<th>Season</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male is 1.25 times more frequently than female</td>
<td>First 2 years of life</td>
<td>Non-breastfed</td>
<td>Winter and early spring</td>
</tr>
<tr>
<td></td>
<td>Peak at 2-6 months</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Viral invasion of small bronchi & bronchioles

Accumulation of mucus, Edema, Cellular debris

Complete bronchiolar obstruction

Incomplete obstruction (Ball valve)

Atelectasis

Early air trapping during expiration

Over inflation of lung
- Impaired air exchange and hypoxia
- Hypercapnia in severe cases
Upper Respiratory Tract Infection for 2-3 days
Gradual onset of Respiratory Distress
- Paroxysmal Spasmodic Cough
- Wheezes
- Dyspnea
- Irritability
- Feeding difficulty due to tachypnea
SIGN

Respiratory Distress

Tachypnea

Wheeze

Hyperinflation
- Respiration fast & shallow.
- Labored breathing (chest indrawing, flaring alae nasi, IC retraction)
- Air hunger
- Restlessness
- Cyanosis

- Hyper resonant note due to over inflated lungs

- Decrease air entry
- Harsh vesicular breathing + prolonged expiration
- Inspiratory + expiratory wheezes
- Inspiratory widespread fine crackles
Differences between bronchiolitis and bronchial asthma

The following may favor the diagnosis of asthma which are:

- is not common in the first year.
- positive family history
- repeated attacks
- markedly prolonged expiration
- onset may be sudden
- not preceding URT infection
- there will be eosinophelia
- response to bronchodilators.
Full blood count WBC & differential count are usually normal.
Respiratory viruses identified by PCR analysis of NPA secretions
Pulse oximetry to measure & monitor arterial oxygen saturation continuously
Blood gas analysis performed in severe cases to identify hypercarbia
Chest X-ray unnecessary
Imaging studies

These are not routinely necessary.

- Hyperinflation
- Focal atelectasis
- Air trapping
- Flattened diaphragm
- Increased anteroposterior diameter
- reveal evidence of alternative diagnoses, such as lobar pneumonia, congestive heart failure, or foreign
- Up to 20% having lobar, segmental, or sub segmental consolidation.
Treatment and medications

- Oxygen
- Fluids
- Nutrition
Medications

- **Antiviral drugs** – only to those have severe RSV infections or high risk infants (Ribavirin)
- **Antibiotics** – Of no values
- **Corticosteroids** – their use is controversial
- **Nebulized B2 – agonists** – their use are also controversial
antibiotics, steroids and nebulised bronchodilators such as salbutamol or ipratropium have not been shown to reduce the severity and duration of illness.
Prognosis

- Most infants recover from acute infection within 2 weeks
- Half will have recurrent episodes of cough & wheeze
- Usually by adenovirus infection, may result in permanent damage to airways (bronchiolitis obliterans)
Prevention

- Monoclonal antibody to RSV (palivizumab, given monthly by intramuscular injection) reduce no. of admissions in high-risk preterm infants.

- Good hand hygiene needed to prevent cross infection to other infants because RSV is highly infectious.
Complications

- Respiratory failure
- Dehydration
- Aspiration pneumonia
- Secondary bacterial infection
- Apnea

In 20% of hospitalized infant at risk for apnea:

1) Premature infant
2) Very young infant (1-4 months)
3) Chronic lung disease.
Chronic inflammatory condition of the lung airways resulting in episodic airflow obstruction, airway hyper responsiveness, and airway remodeling.
Environmental factors + genetic predisposition → Bronchial inflammation → Bronchial hyperactivity + trigger factors

Edema, bronchoconstriction, excess mucus production → Airway narrowing → Symptoms: cough, wheezing, chest tightness, breathlessness
Genetic predisposition

Atopy

Environmental triggers
- Upper respiratory tract infections
- Allergens (e.g. house dust mite, grass pollens, pets)
- Environmental tobacco smoke
- Cold air
- Exercise
- Emotional upset or anxiety
- Chemical irritants (e.g. paint, aerosols)

Bronchial inflammation
- Oedema
- Excessive mucus production
- Infiltration with cells (eosinophils, mast cells, neutrophils, lymphocytes)

Bronchial hyperresponsiveness

Airway narrowing

Symptoms:
Wheeze
Cough
Breathlessness
Chest tightness
ASTHMA TRIGGERS

- Common viral infections of the respiratory tract
- Aeroallergens in sensitized asthmatic patients
- Seasonal aeroallergens
- Air pollutants
- Strong or noxious odors or fumes
- Occupational exposures
- Cold air, dry air
- Exercise
- Crying, laughter, hyperventilation
- Co-morbid conditions
EARLY CHILDHOOD RISK FACTORS FOR PERSISTENT ASTHMA

1) Parental asthma
2) Allergy
3) Severe lower respiratory tract infection
4) Wheezing apart from cold
5) Male gender
6) Low birth weight
7) Environmental tobacco smoke exposure
Signs & Symptoms

- Wheezing
- Cough
- Shortness of breath
- Chest tightness
Examination

- Examination of the chest is usually normal between attacks.
- In longstanding asthma
  - hyperinflation
  - Harrison sulci
  - generalized expiratory wheeze
  - and prolonged expiratory phase.

- Evidence of eczema
- the nasal mucosa for allergic rhinitis.
Summary

Assessment of the child with chronic 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, etc.

If there is:
- Sputum
- Finger clubbing
- Growth failure
  - If present, other causes should be sought

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

Consider triggers:
- Allergic rhinitis needing treatment?
- Allergens - animal dander, etc.
- Stress
Asthma in young children challenge

Wheezing occurs in many young children who do not have asthma, making diagnosis of asthma difficult.

Lung function tests cannot be performed in this age group to help confirm the diagnosis.

Asthma Predictive Index a guide to determining which small children will likely have asthma in later years.
Investigations

- CBC: Eosinophilia in a range of 15-20%
- Eosinophilia in bronchial mucosa strongly suggest Asthma
- Allergy testing
- Pulse oximetry
- Arterial blood gas analysis
- Pulmonary function test: Applicable for children > 6 years
- CXR
Lung function tests can help to confirm the diagnosis of asthma and to determine disease severity.

**Spirometry**

- **Airflow limitation**
  - Low FEV₁ (relative to percentage of predicted norms)
  - FEV₁/FVC ratio < 0.80

- **Bronchodilator response (to inhaled β-agonist):**
  - Improvement in FEV₁ ≥ 12% and ≥ 200 mL

- **Exercise challenge**
  - Worsening in FEV₁ ≥ 15%

- Daily peak flow or FEV₁ monitoring: day to day and/or am-to-pm variation ≥ 20%
Measuring exhaled nitric oxide ($FE_{NO}$), a marker of airway inflammation in allergy-associated asthma,
* It is elevated in asthmatic not taken steroid
* Not specific for bronchial asthma

Peak expiratory flow (PEF)
- monitoring devices provide simple and inexpensive home-use tools to measure airflow
- Useful for monitoring asthma control
- Reflect hyperresponsive
- Useful Severity of exacerbation
DIAGNOSIS

physical examination of characteristic musical wheezing

history of intermittent or chronic symptoms typical of asthma

strongly point to a diagnosis of asthma
Confirmation of the diagnosis of asthma is based on three key additional elements:

1. The demonstration of variable expiratory airflow limitation, preferably by spirometry
2. Documentation of reversible obstruction
3. Exclusion of alternative diagnoses
Managements

Acute
Chronic
- Bronchodilator therapy.
- Inhaled corticosteroid
- Leukotriene receptor antagonist (montelukast)
- Slow-release oral theophylline is an alternative
- Oral prednisolone
- Anti-IgE therapy (omalizumab)
- Allergen immunotherapy
Gastro-oesophageal reflux
GERD

It is extremely common in infancy.

caused by
1) inappropriate relaxation of the lower oesophageal sphincter as a result of functional immaturity.
2) A predominantly fluid diet,
3) A mainly horizontal posture

resolves spontaneously by 12 months of age.
Severe reflux is more common in:

1) children with cerebral palsy or other neurodevelopmental disorders.

2) preterm infants

3) following surgery for oesophageal atresia diaphragmatic hernia.
Complications of GERD

- Failure to thrive from severe vomiting
- Oesophagitis – haematemesis, discomfort on feeding or heartburn, iron deficiency anaemia
- Recurrent pulmonary aspiration, recurrent pneumonia, cough or wheeze, apnoea in preterm infants
- Dystonic neck posturing (Sandifer syndrome)
- Apparent lifethreatening events (ALTE)
Investigation

May be indicated if
1) the history is atypical
2) complications are present
3) failure to respond to treatment.

Investigations include:
- 24-hour oesophageal pH monitoring
- Endoscopy with oesophageal biopsies
- Contrast studies of the upper gastrointestinal tract
Esophageal pH monitoring
Management

Uncomplicated gastrooesophageal reflux can be managed by:

1) Parental reassurance
2) adding inert thickening agents to feeds
3) positioning in a 30° headup prone position after feeds.
4) acid suppression with either:
   - H2 receptor antagonists (e.g. ranitidine)
   - proton pump inhibitors (e.g. omeprazole)

5) If the child fails to respond to these measures, cow’s milk protein allergy should be considered
6) Surgical management: fundoplication
Cystic fibrosis
Cystic fibrosis (CF)

- is an inherited (AR)
- multisystem disorder of children
- characterized chiefly by
  - obstruction and infection of airways
  - mal digestion and its consequence
- major cause of severe chronic lung disease in children
- is responsible for most exocrine pancreatic insufficiency in early life.
Cystic fibrosis

- Cystic Fibrosis is an inherited disease.
- Both parents must be carriers of a defective gene on chromosome 7.
- 50% chance of becoming a carrier.
- A 25% chance of getting CF
- A 25% chance of not being a carrier and not having CF
### What does the thick sticky mucus do?

<table>
<thead>
<tr>
<th>Lungs</th>
<th>Pancreas</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mucus builds up and obstructs airway</td>
<td>Build up of mucus blocks ducts in pancreas, stopping enzymes from reaching intestines</td>
</tr>
<tr>
<td>Build up also makes a suitable environment for bacterial growth</td>
<td>Without enzymes, intestines can’t digest food properly</td>
</tr>
<tr>
<td>Bacterial growth increases risk of infections</td>
<td>Leads to loss of vitamins and nutrients</td>
</tr>
<tr>
<td>Repeated infections cause lung damage</td>
<td></td>
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Lungs

- Mucus builds up and obstructs airway
- Build up also makes a suitable environment for bacterial growth
- Bacterial growth increases risk of infections
- Repeated infections cause lung damage

Pancreas

- Build up of mucus blocks ducts in pancreas, stopping enzymes from reaching intestines
- Without enzymes, intestines can’t digest food properly
- Leads to loss of vitamins and nutrients
Manifestations of Cystic Fibrosis

General
- Growth failure (malabsorption)
- Vitamin deficiency states (vitamins A, D, E, K)

Nose and sinuses
- Nasal polyps
- Sinusitis

Liver
- Hepatic steatosis
- Portal hypertension

Gallbladder
- Biliary cirrhosis
- Neonatal obstructive jaundice
- Cholelithiasis

Bone
- Hypertrophic osteoarthropathy
  - Clubbing
- Arthritis
- Osteoporosis

Intestines
- Meconium ileus
- Meconium peritonitis
- Rectal prolapse
- Intussusception
- Volvulus
- Fibrosing colonopathy (strictures)
- Appendicitis
- Intestinal atresia
- Distal intestinal obstruction syndrome
- Inguinal hernia

Lungs
- Bronchiectasis
- Bronchitis
- Bronchiolitis
- Pneumonia
- Atelectasis
- Hemoptysis
- Pneumothorax
- Reactive airway disease
- Cor pulmonale
- Respiratory failure
- Mucoïd impaction of the bronchi
- Allergic bronchopulmonary aspergillosis

Heart
- Right ventricular hypertrophy
- Pulmonary artery dilation

Spleen
- Hypersplenism

Stomach
- GERD

Pancreas
- Pancreatitis
- Insulin deficiency
- Symptomatic hyperglycemia
- Diabetes

Reproductive
- Infertility
  (aspermia, Absence of vas deferens)
- Amenorrhea
- Delayed puberty
Cystic fibrosis

Most children with CF present with:
- malabsorption
- Failure to thrive
- Recurrent chest infection.

Examination:
Full assessment of:
- Respiratory system.
- Liver and GIT system.
- Growth and development.
Screening

- most newborn with CF can be identified by determination of immunoreactive trypsinogene and limited DNA testing on blood spots.

- This screening test is about 95% sensitive.
Investigation

Sweat test:
- most definite test. is recommended for analysis of chloride in these samples

- +ve when CL is equal or more than 60 meq/L which is dx for CF in conjunction with one of the followings:
  1) Typical chronic obstructive pulmonary dis.
  2) Exocrine pancreatic insufficiency
  3) Positive family hx.
Treatments for CF

• **Medications**
  – Medications are used to treat lung disease
  – Many are inhaled using a nebulizer
  – Medications used are:
    • Mucolytics, which loosen lung mucus
    • Bronchodilators, which expand the airways
    • Steroids, which decrease inflammation
    • Antibiotics, fight infections

• **Chest physical therapy**
  – Considered standard therapy
  – Used to clear mucus from the lungs
Treatments for CF

• **Nutrition**
  - Good nutrition
  - High-calorie diet
  - Vitamins

• **Pancreatic enzymes**
  - Pancreatic enzyme supplements, taken with everything consumed, help absorb nutrients

• **Gene therapy**
  - is an experimental technique that uses genes to treat diseases.
  - Gene therapy can replace a mutated gene or inactivating a mutated gene.
  - It is promising but risky.