

Differential Diagnosis of Wheezing:

In general:

DDX of wheeze:

- ① Asthma
② bronchitis
- كيف أميز بينهما؟
- recurrent wheezy chest Never bronchitis.
- 2 years Never bronchitis.
- كل ما حشر كل ما
- طرب لراحياتي واحد أهل من سنين وأول مرة
- Risk factors: ④ Family history
atopy & dermatitis/ conjunctivitis.
good response to salbutamol.
Peak flow > 15%
↑ IgE - eosinophil.
Fever / consolidation
must think about underlying dz other than asthma / bronchitis
وهو الراجح دائماً بنسبها.

③ Foreign body aspiration

- wheezy localized → F. body aspiration.
- wheezy bilateral لا تنفصا.

④ GERD

- من أدخله؟ لما يكون wheezy chest أو infection
How cause wheezy chest?
- Direct aspiration of acid & milk on lung → chemical pneumonia
- acid reflux on lower part of esophagus
alkaline
cause irritation of mucous membrane (vagus nerve)
this cause bronchospasm.

You can diagnose the GERD, but You can't exclude it.

لقد ما شخصيت سوا عمل؟

- ① prone position, the best to ↓ GERD (من 10-15 دقيقة)
but it is Risky for sudden infant death. So do it under vision.

② small frequent feeding:

يفضل لو بتطعم 120 مل 6 مرات اليوم
تصبح تطعم 60 مل 12 مرة اليوم

③ Randin IV / omeprazole / vomitadin / metoclopramide.

- من أشغل جراحياً حمادة العضع؟
→ ↑ sphincter tone
→ ↑ gastric motility

- Fail to gain wt.
- loss of wt.
- recurrent apnea →
- recurrent life-threatening infection.
هنا كل اسم من هذه
بجسم من كذا.

④ Nissen Fundoplication.

⑤ Congenital heart

- VSD / cardiomyopathy → congestion of the cardiole
cause compression of bronchide.
enlarge heart cause
compression on lung.

⑥ Immunodeficiency.

⑦ cystic Fibrosis:

obstructive lung dz
restrictive lung dz

⑧ cilia dyskinesia

⑨ congenital anomaly in lung.

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

...Congenital Ricket... How? ...
 ...Store إلى أعطته إياه ...
 ...Store ...
 ...Store إلى عندها ...
 ...الموضوع المثرة الجلبة ...

Acute wheezing causes:

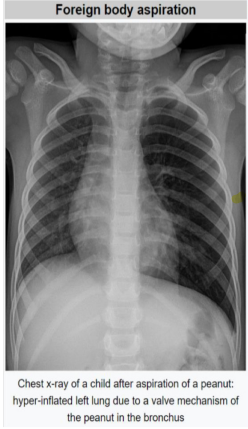
Differential diagnosis of acute wheezing in children			
Disease	Age	Characteristics	Diagnostics
Common cold	Children < 6 years of age have an average of 6-8 <u>common</u> colds per year. [1]	- Rhinorrhea, sneezing, nasal obstruction - Sore throat, hoarseness - Coughing - Mild fever - Malaise and fatigue	- Clinical diagnosis - Lungs usually clear to auscultation
Acute asthma exacerbation	- Incidence of allergic asthma is highest in early childhood. [2] - Median age of onset: 7 years [3]	- Expiratory wheezing - Tachypnea - Acessory muscle usage - Pulsus paradoxus - Hyperresonance Silent chest - Risk factors: history of atopy, allergic rhinitis, and/or eczema	- Clinical diagnosis - Spirometry is not feasible during most asthma exacerbations. ☹ - Peak flow meter: PEFR lower than baseline
Viral bronchiolitis	Primarily affects children < 2 years of age during winter [4]	- Prodrome with rhinorrhea, low-grade fever, dry cough - Followed by wheezing, fine end-inspiratory crackles, tachypnea, nasal flaring, and intercostal retractions	- Clinical diagnosis - Chest x-ray: <u>hyperinflation of the lungs</u>, <u>interstitial infiltrates</u>, <u>focal atelectasis</u> - Rapid antigen testing of nasopharyngeal secretions for RSV and influenza ☹
Foreign body aspiration (FBA)	Primarily occurs in children < 3 years of age [5]	- Sudden onset of dry cough, generalized wheezing, and choking - Stridor - Dyspnea - Tachypnea <u>Crepitus localized to the affected lobe</u> - May appear with cyanosis	- Upper airway FBA: neck x-ray (radiopaque foreign object), laryngoscopy - Lower airway FBA: chest x-ray (focal hyperlucency, reduced pulmonary markings, atelectasis), rigid bronchoscopy - CT chest without contrast: in inconclusive chest x-ray; to guide planned removal of FB
Anaphylaxis	Occurs at any age	- Dry cough - Stridor, wheezing - Dyspnea - Chest tightness <u>Urticaria, pruritis, angioedema</u> - Hypotension - Most common trigger in infants: food	- Clinical diagnosis - Serology: elevated <u>serum mast-cell tryptase</u>

Acute bronchitis	Most common in children < 5 years of age^[6]	- Prodrome with <u>rhinorrhea</u>, <u>sore throat</u>, <u>headache</u>, <u>malaise</u>, <u>myalgia</u> - Cough with or without sputum and chest pain <u>Rhonchi</u>, <u>wheezing</u>, <u>fine crackles</u> (rare) <u>Mild dyspnea</u> <u>Fever</u> (rare)	- Clinical diagnosis CBC: mild leukocytosis - Chest x-ray: normal or nonspecific (e.g., <u>peribronchial thickening</u>)
Croup (laryngotracheobronchitis)	Peak incidence: 6 months to 3 years of age^[7]	- Prodrome with <u>rhinitis</u> and <u>low-grade fever</u> - Laryngotracheal inflammation phase: Seal-like barking cough <u>Inspiratory stridor</u> <u>Dyspnea</u> <u>Pulsus paradoxus</u>	- Clinical diagnosis - X-ray of chest and neck: <u>subglottic narrowing (steeple sign)</u> - PCR: to identify viral pathogen

Foreign body aspiration :

Foreign body aspiration (FBA)

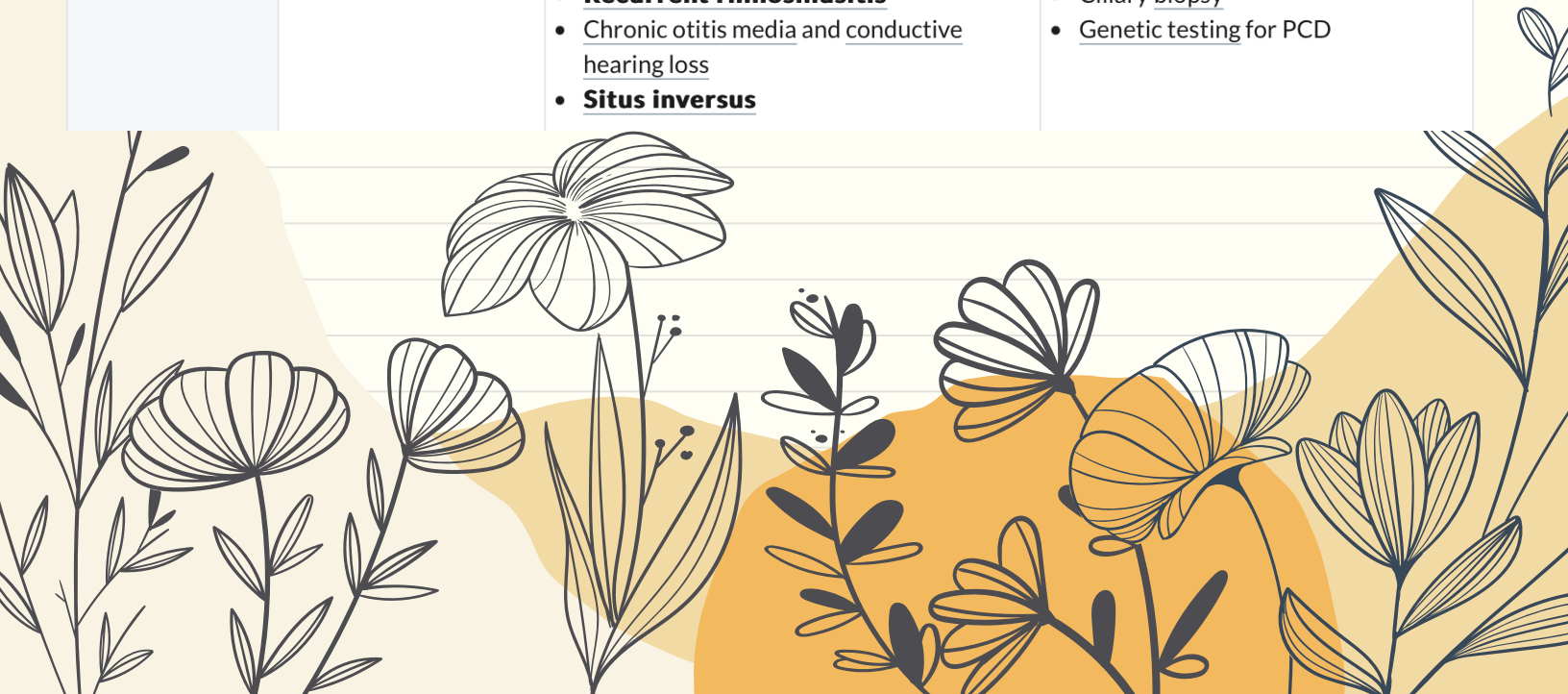
- Although the differential diagnosis for respiratory distress is broad, **clinical presentation of sudden-onset respiratory distress without a preceding illness and focal findings on pulmonary examination is most consistent with foreign body aspiration (FBA).**
- Patients typically have **acute-onset cough, dyspnea, and stridor.** Wheezing may be focal or generalized, and **decreased breath sounds on the affected side** are characteristic. **A history of choking, if witnessed, is very helpful in diagnosis.**
- FBA is most common in children **age 1-3 years.**
- Commonly aspirated FBs include foods such as **peanuts and popcorn and pieces of toys.**
- More than half of aspirated FBs end up in **the right mainstem bronchus;** laryngeal and tracheal FBs are **far less common.**
- Although chest radiographs are often obtained in patients with suspected FBA, **they are normal in approximately 2/3 of cases given that most aspirated objects are radiolucent.**
- If an FB causes **partial obstruction**, with air trapping during expiration, **hyperinflated lungs are seen on imaging.** In contrast, **complete obstruction** can result in **atelectasis, post-obstructive pneumonia, and/or localized bronchiectasis (late feature).**
- The standard of care for both diagnosis and treatment of FBA is **immediate rigid bronchoscopy to remove the foreign body.**



Foreign body aspiration	
Clinical features	- Sudden-onset cough, dyspnea - Cyanosis ± History of choking episode
Examination findings	- Wheezing and/or stridor - Focal area of diminished breath sounds
X-ray findings	- Hyperinflation of affected side - Mediastinal shift toward unaffected side - Atelectasis if obstruction is complete ± Foreign body
Management	Rigid bronchoscopy

Chronic and recurrent wheezing:

Differential diagnosis of chronic or recurrent wheezing in children			
Disease	Age	Characteristics	Diagnostic findings
Asthma	- Incidence of allergic asthma is highest in early childhood. [2] - Median age of onset: 7 years [3]	- Persistent, dry cough that worsens at night, with exercise, and/or on exposure to triggers and irritants (e.g., cold air, allergens, smoke) - Wheezing - Dyspnea - Risk factors: history of atopy, allergic rhinitis, eczema	- Pulmonary function tests showing an obstructive pattern: ↓ FEV1; ↓ FEV1/FVC ratio - Bronchodilator administration showing reversibility of airflow obstruction - Exercise or methacholine challenge testing: ↑ airway resistance and ↓ FEV1 indicate airway hyperresponsiveness - CBC: eosinophilia - Chest x-ray: normal in most patients, although hyperinflation of the lungs may be seen in some
Double aortic arch	Congenital; manifests in first weeks of life [8]	- Tracheal compression: - Inspiratory and expiratory stridor, dyspnea - Apnea with cyanosis in severe compression - Esophageal constriction: - Dysphagia, choking, retching, vomiting - Failure to thrive	- Chest x-ray: anterior tracheal bowing and narrowing - Echocardiography: to identify anomaly and associated congenital heart defects (e.g., VSD, tetralogy of Fallot, PDA)
Primary ciliary dyskinesia	Median age at diagnosis: 5.3 years [9]	- Productive cough (due to recurrent respiratory tract infections and/or bronchiectasis) - Wheezing and crackles - Nail clubbing - Failure to thrive - Recurrent rhinosinusitis - Chronic otitis media and conductive hearing loss - Situs inversus	- Chest x-ray: bronchiectasis, dextrocardia, situs inversus - Nasal nitric oxide test: reduced levels - Ciliary biopsy - Genetic testing for PCD



<u>Cystic fibrosis</u>	• Most are diagnosed at <u>newborn screening</u> or at 4–14.5 months ^[10]	• Respiratory ◦ Chronic productive <u>cough</u> (due to recurrent <u>pneumonia</u>, <u>chronic bronchitis</u>, and/or <u>bronchiectasis</u>) ◦ <u>Wheezing</u>, <u>dyspnea</u> (due to pulmonary obstruction and <u>airway hyperreactivity</u>) ◦ <u>Chronic sinusitis</u>, <u>nasal polyps</u> ◦ <u>Nail clubbing</u> ◦ <u>Barrel chest</u> • Gastrointestinal ◦ <u>Meconium ileus</u> ◦ <u>Steatorrhea</u>, <u>diabetes mellitus</u>, <u>malabsorption</u> (due to <u>pancreatic insufficiency</u>) ◦ <u>Failure to thrive</u>	• <u>Sweat chloride testing</u> with a <u>chloride value</u> ≥ 60 mmol/L on ≥ 2 occasions • <u>CFTR genetic testing</u> positive for mutations in the <u>CFTR gene</u> • <u>Chest x-ray</u>: <u>hyperinflation</u>, <u>reticular nodular pattern</u>, <u>bronchiectasis</u>
<u>Tracheomalacia</u>	• Congenital; symptoms usually manifest at 4–8 weeks of age ^[11]	• Ineffective croup-like <u>cough</u> (due to <u>tracheal collapse</u>) • Inspiratory and <u>expiratory stridor</u>, <u>wheezing</u> that may vary with activity and position • Recurrent respiratory illnesses	• <u>Flexible bronchoscopy</u> or dynamic CT: collapse of the <u>trachea</u> with deep breathing
<u>Left-sided heart failure with cardiomegaly</u>	• <u>Neonates and infants</u> < 2 months of age with <u>structural heart disease</u>	• <u>Paroxysmal nocturnal dyspnea</u> • <u>Exertional dyspnea</u>, <u>orthopnea</u> (due to pulmonary venous congestion) • <u>Pulmonary edema</u> • <u>Wheezing</u>, bilateral basal <u>rales</u> • <u>Fatigue</u> • <u>Nocturia</u> • <u>Tachycardia</u>, <u>arrhythmia</u> • <u>Cardiac auscultation</u>: <u>S₃</u> or <u>S₄ gallop</u> • <u>Pulsus alternans</u> • <u>Cachexia</u> • <u>Coolness and pallor</u> of lower extremities	• <u>Chest x-ray</u>: <u>boot-shaped heart</u> on PA view, increased <u>pulmonary vascular markings</u> • <u>ECG</u>: <u>left ventricular hypertrophy</u> • Increased <u>natriuretic peptides</u> (<u>BNP</u> or <u>NT-proBNP</u>) • Elevated cardiac <u>troponin T</u>

Primary ciliary dyskinesia:

Etiology

Primary ciliary dyskinesia (PCD, **immotile cilia syndrome**) is an **inherited disorder in which there is absent or disordered movement of the cilia** leading to a spectrum of **clinical manifestations**. This disorder affects approximately 1 in 15,000-20,000 live births, although the actual incidence may be higher because of a limited ability to definitively diagnose affected patients.

Clinical Manifestations

Clinical manifestations include **neonatal respiratory distress, chronic cough, chronic nasal congestion, middle ear effusions, chronic pansinusitis, laterality defects (e.g., situs inversus), infertility, and bronchiectasis**. Kartagener syndrome, the triad of **situs inversus, pansinusitis, and bronchiectasis**, accounts for approximately 50% of cases. Males are infertile as a result of immotile sperm. **Because the cilia fail to beat normally, secretions accumulate in the airways, and endobronchial infection results. Chronic infection, if untreated, leads to bronchiectasis by early adulthood.**

Diagnostic Studies

PCD diagnosis has historically relied on a combination of **clinical features and ultrastructural analysis of respiratory cilia by electron microscopy, obtained from scrapings/biopsy of nasal or airway epithelium**. Results may be difficult to interpret as chronic infection and inflammation may also lead to ultrastructural abnormalities in nasal cilia. The most common ultrastructural defect is the absence of dynein arms. A third of affected individuals have normal ciliary ultrastructure. **The measurement of nasal nitric oxide has been used as a screening tool for PCD.** Low nasal nitric oxide values (<77 nL/min) are consistent with PCD. High-speed video microscopy assesses ciliary beat frequency and pattern but requires specialized equipment and, currently, there is an absence of standardized reporting methods. PCD is generally autosomal recessive. Currently 31 genes have been identified to explain approximately 60% of cases. The association of genetic defects and clinical phenotype is unknown.

Treatment

Treatment is geared toward treating infections and improving clearance of respiratory secretions. High-resolution chest CT scans are useful to confirm and monitor bronchiectasis. Surveillance cultures help identify organisms involved and guide antibiotic therapy. Sinus surgical procedures are often done to manage chronic sinusitis, but their benefit is questionable. Most children require placement of pressure equalization (PE) tubes for management of recurrent otitis media. Chest physiotherapy and prompt treatment of bacterial infections are helpful, but the course of the disease tends to be slowly progressive. Inhaled hypertonic saline may improve cough clearance and has been shown in the short term to improve lung function. Antiinflammatory antibiotics, such as low-dose macrolides, may reduce the number of exacerbations per year.

545

Bronchopulmonary dysplasia:

Bronchopulmonary dysplasia (BPD) [16]

- **Definition:** chronic lung condition secondary to prolonged mechanical ventilation and oxygen therapy for NRDS
- **Etiology:** Pulmonary barotrauma and oxygen toxicity with subsequent inflammation of lung tissue due to ventilation of the immature lung (ventilation for more than 28 days)
- **Clinical features**
 - **Seen in infants < 32 weeks**
 - Persistence of symptoms similar to NRDS (e.g., tachypnea, grunting, nasal flaring)
 - Episodes of desaturation
- **Diagnostics**
 - X-ray chest: diffuse, **fine, granular densities**, areas of **atelectasis** interspersed with areas of hyperinflation
 - Blood gas analysis: respiratory and metabolic acidosis
 - Histology: **atelectasis, fibrosis, emphysematous alveolar changes** (decreased number and septation of alveoli)
- **Treatment:** controlled oxygenation, diuretics, rarely glucocorticoids

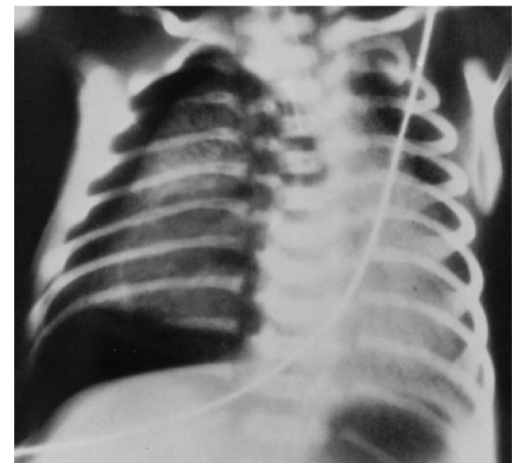


FIGURE 61.3 Pneumothorax. Right-sided hyperlucent pleural air is obvious. The findings of linear interstitial air and the resultant noncompliant but collapsed lung are noted. (From Heller RM, Kirchner SG. *Advanced Exercises in Diagnostic Radiology: the Newborn*. Philadelphia: WB Saunders; 1979.)

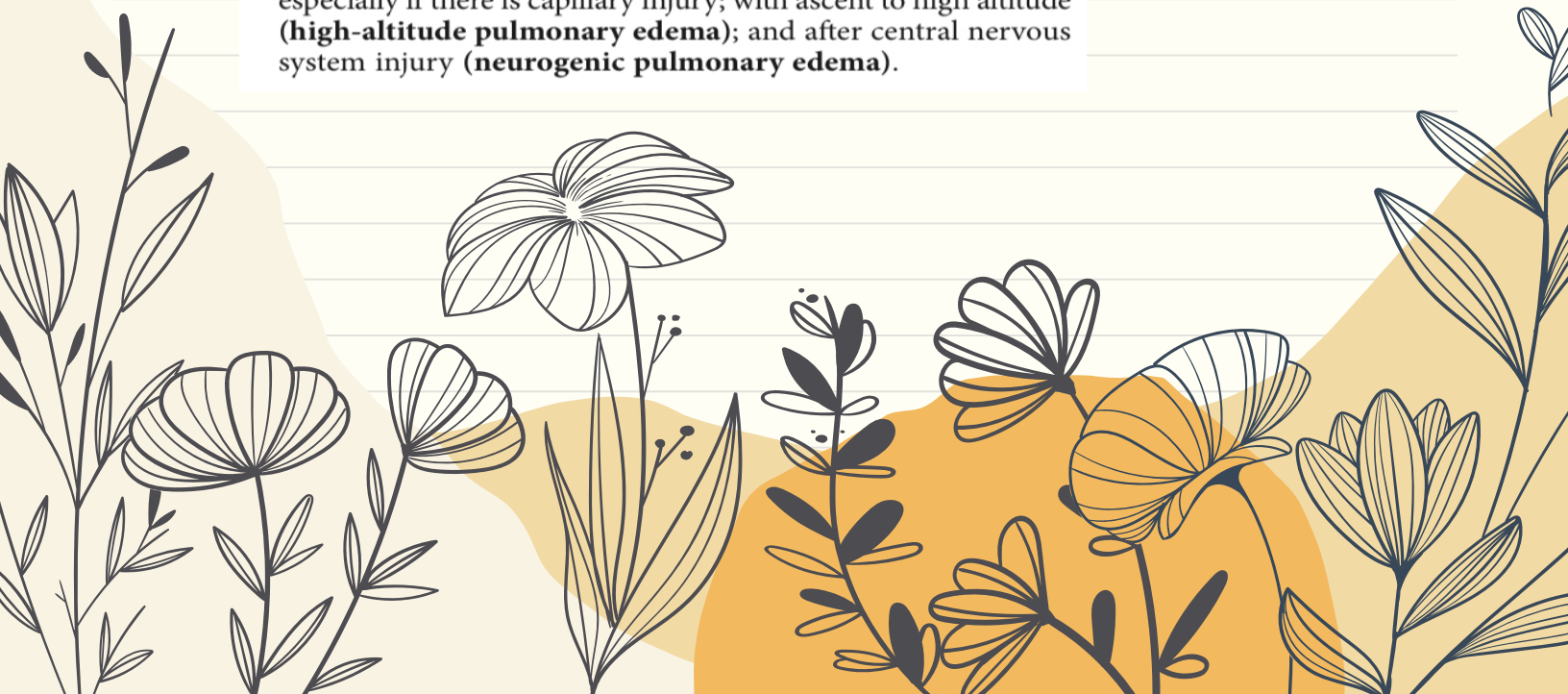
Pulmonary edema:

Pulmonary Edema

Etiology

Pulmonary edema is the seepage of fluid into the alveolar and interstitial spaces. Capillary hydrostatic forces and interstitial osmotic pressures tend to push fluid into the air spaces, whereas plasma osmotic pressures and tissue mechanical forces tend to move fluid away from the air spaces. Under normal circumstances, the sum of these forces favors absorption, so the alveolar and interstitial spaces remain dry. Fluid entering the alveoli is normally removed by pulmonary lymphatics. Pulmonary edema forms when transcapillary fluid flux exceeds lymphatic drainage. Reduced left ventricular function leads to pulmonary venous hypertension and increased capillary hydrostatic pressure, and fluid moves into the interstitial space and alveoli. Fluid initially enters the interstitial space around the terminal bronchioles, alveoli, and arterioles (**interstitial edema**), causing increased lung stiffness and premature closure of bronchioles on expiration. If the process continues, fluid then enters the alveoli, further reducing compliance and resulting in **intrapulmonary shunting** (alveolar units that are perfused but not ventilated). Decreased ventilation (hypercarbia) is a late finding.

Pulmonary edema is most commonly due to heart failure from left ventricular or biventricular dysfunction. Pulmonary hypertension (PH) and associated cor pulmonale (right ventricular dysfunction) do not usually cause pulmonary edema as the increased vascular resistance is proximal to the capillary bed. Increased capillary permeability, seen in disease states such as sepsis and acute respiratory distress syndrome, can lead to pulmonary edema. Pulmonary edema may also occur with excessive swings in intrathoracic pressure, as seen after tracheal foreign body aspiration or severe obstruction from hypertrophied tonsils and adenoids (**postobstructive pulmonary edema**). Pulmonary edema may also be present in conditions with decreased serum oncotic pressure (hypoalbuminemia); after administration of large volumes of intravenous (IV) fluids, especially if there is capillary injury; with ascent to high altitude (**high-altitude pulmonary edema**); and after central nervous system injury (**neurogenic pulmonary edema**).



Congenital Lung Anomalies

Congenital lobar emphysema (congenital lobar overinflation) consists of hyperinflation of one or more lobes of the lung. This overdistention may cause severe respiratory distress in the neonatal period due to compression of surrounding normal lung tissue, but it can also be asymptomatic and remain undiagnosed for years. Radiographically, it may be mistaken for a pneumothorax. Lobectomy may be required if respiratory distress is severe or progressive, but if the patient is asymptomatic, then surgical resection may not be indicated.

Congenital pulmonary airway malformations (CPAMs) are a spectrum of lesions arising during early lung development with histological appearance ranging from simplified bronchial to complex cystic alveolar tissue, connected to the tracheobronchial tree. They are often first identified on antenatal ultrasound, and after birth can be inconspicuous on chest radiograph. CPAMs are nonfunctioning lung tissue and can have hybrid features of **pulmonary sequestration** (presence of systemic circulation connections). Although CPAM lesions can be stand-alone lesions, a substantial proportion of pulmonary sequestrations are hybrid CPAM lesions. Sequestrations without hybrid features are separate from the tracheobronchial tree and are classified as intrapulmonary (located in a normal lobe without its own pleura) and extrapulmonary (outside normal lung). CPAMs and sequestrations can coexist with other chest lesions including **congenital diaphragmatic hernia**. Sequestration is also associated with **Scimitar syndrome** (partial anomalous pulmonary venous return). Symptomatic neonates with respiratory distress, large lesions, or family history of features concerning for malignancy require further imaging. Though large malformations can cause neonatal respiratory distress, many are asymptomatic. Chest CT with contrast is the diagnostic test of choice ([Fig 136.2](#)) but may be delayed if the child is asymptomatic with close clinical follow-up for symptom development. Surgical resection is curative, but risk of infection or malignancy is not completely understood in these children. Conservative management with observation is a reasonable option for families with low risk.

Pulmonary hypoplasia, or a relative decrease in amount of alveoli, may be the result of a number of congenital insults, such as **congenital diaphragmatic hernia** (see [Chapter 61](#)) or unilateral congenital absence of a pulmonary artery. Alveolar growth occurs after birth, predominantly up to 3 years, but has been documented beyond 8 years of age.