1. SIGNIFICANCE

Differential diagnosis of bronchial obstruction in children is one of the challenging issues in pediatrics as etiology is very variable that dictates quite different approaches to the treatment. However, bronchitis, bronchiolitis, and asthma remain the most common problems of bronchial obstruction and should serve a background in differential diagnosis. There are a number of acute and chronic disorders that present with coughing and wheezing, therefore the student should be careful in clinical evaluation of the child with bronchitis or bronchiolitis.

2. PREREQUISITES

The skills listed below will not be taught in this lesson but are necessary to perform physical examination of the patient practical training. Therefore, before beginning this lesson, one has to be sure of the ability to: inspect the thorax and its respiratory movements and note rate, rhythm, depth, and effort of breathing; observe retractions of the supraclavicular areas of contractions of the sternomastoid muscles on respiration; observe shape of the child’s chest; auscultate to child’s breathing for increased white noise and wheezes; palpate the chest for respiratory expansion, tactile fremitus; percuss the chest in the standard areas, comparing one side with the other at each level; auscultate to the chest with stethoscope in order to evaluate breath sounds and not any adventitious sounds.

3. EDUCATIONAL OBJECTIVES

Student should know:
- differential diagnosis in a context of etiology, pathogenesis, classification, clinical manifestation of for the obstructive disorders in children and infants.

Student should be able:
- to identify the child with obstructive disorder, make correct decisions during physical examination of the patient with given conditions, take appropriate actions based on those decisions, demonstrate skills to develop management and follow up measures.

4. INTERDISCIPLINARY INTEGRATION

<table>
<thead>
<tr>
<th>Discipline</th>
<th>Student should know</th>
<th>Student should be able to</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal anatomy, Physiology</td>
<td>Anatomic and physiologic features of airways in children of different age groups</td>
<td>Use knowledge of anatomic and physiologic features of the respiratory system in children for evaluation of clinical findings</td>
</tr>
<tr>
<td>Biochemistry</td>
<td>Normal ranges for the routine biochemical blood analysis</td>
<td>Assess blood biochemistry and comment on deviations from normal in a clinical context</td>
</tr>
<tr>
<td>Pathology</td>
<td>Histologic and histochemical presentation of bronchoobstructive illnesses in children</td>
<td>Use knowledge of histologic and histochemical presentation of bronchoobstructive illnesses in children for evaluation of clinical findings</td>
</tr>
<tr>
<td>-----------</td>
<td>-----------------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Pathologic physiology</td>
<td>Pathophysiologic mechanisms of the respiratory failure</td>
<td>Recognize symptom and signs of respiratory failure</td>
</tr>
<tr>
<td>Propedeutics of pediatric diseases</td>
<td>Physical examination of the respiratory system in children. Correct performance of pulmonary tests.</td>
<td>Perform physical examination of the respiratory system (gross inspection, palpation, percussion, auscultation), Assess the results of pulmonary tests</td>
</tr>
<tr>
<td>Imaging studies</td>
<td>Indications and methods of imaging studies in bronchoobstructive illnesses</td>
<td>Assess radiologic examination of the chest</td>
</tr>
<tr>
<td>Intensive care</td>
<td>Symptoms and signs of respiratory failure of different stages, its etiology, and principles of intensive care</td>
<td>Recognize respiratory failure, assess its severity, provide emergency care</td>
</tr>
</tbody>
</table>

5. ABSTRACT FOR PRE-WORKSHOP SELF-EDUCATION

1. Differential diagnosis of bronchial obstruction in children

**Airway Foreign Body**

- History of a distinct coughing or choking episode: occurs in a majority of cases.
- Sudden onset of respiratory distress.
- Acute or chronic cough.
- Hoarseness or aphonia.
- Some patients with small subglottic foreign bodies may be asymptomatic.

**Laryngeal or tracheal foreign bodies**

- Total obstruction results in severe respiratory distress with cyanosis, supraclavicular and substernal retractions, aphonia, ineffective cough, and absent or very diminished breath sounds, with or without loss of consciousness.
- Partial obstruction results in stridor, cough, dysphonia, drooling and supraclavicular and/or substernal retractions.

**Bronchial foreign bodies**

- The classic triad of cough, wheezing, and focally decreased breath sounds occurs in only 30% of patients.
- Respiratory distress is manifested as tachypnea, intercostal retractions, and cough.
- 20% of patients are asymptomatic at presentation.

**Allergic Rhinitis**

- Symptoms
  - Ocular pruritus.
  - Ocular discharge and tearing.
  - Photophobia.
  - Above with sneezing.
  - Nasal itching and/or congestion.
<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aspergillosis</strong></td>
<td>Patients with allergic bronchopulmonary aspergillosis (ABPA) often have histories of worsening respiratory symptoms in association with asthma or cystic fibrosis (CF). ABPA occurs in approximately 11% of patients with CF. The main complaints of these patients are wheezing and cough. As the disease progresses, patients may expectorate mucous plugs containing eosinophils, and they may develop bronchiectasis. Exacerbation and remission characterize the natural history of disease. Progression to respiratory failure may occur occasionally because of irreversible airway obstruction and pulmonary fibrosis. It may mimic pneumonia with mucopurulent bloody sputum, fever, and respiratory distress. Predominant wheezing may be the only manifestation suggesting an exacerbation of bronchial asthma. Aspergillomas may remain asymptomatic until hemoptysis occurs.</td>
</tr>
<tr>
<td><strong>Bronchiectasis</strong></td>
<td>Should include differential diagnosis of: Aspiration Pneumonia Bacterial Pneumonia Chronic Obstructive Pulmonary Disease Emphysema Parapneumonic Pleural Effusions and Empyema Thoracis Pediatric Asthma Pediatric Bronchitis Pediatric Cystic Fibrosis Tuberculosis</td>
</tr>
<tr>
<td><strong>Bronchiolitis</strong></td>
<td>SEE DETAILS BELOW</td>
</tr>
<tr>
<td><strong>Bronchopulmonary Dysplasia</strong></td>
<td>Abnormal findings on physical examination, and chest radiography Initial findings observed shortly after birth consistent with respiratory distress syndrome (RDS). Persistence of these abnormalities can be associated with an increased risk of bronchopulmonary dysplasia. Physical examination may reveal tachypnea, tachycardia, increased work of breathing (with retractions, nasal flaring, and grunting), frequent desaturations, and significant weight loss during the first 10 days of life. Infants with severe bronchopulmonary dysplasia are often extremely immature and had a very low birth weight. Their requirements for oxygen and ventilatory support often increase in the first 2 weeks of life.</td>
</tr>
<tr>
<td><strong>Cystic Fibrosis</strong></td>
<td>Presence of typical clinical features (respiratory, gastrointestinal, or genitourinary) OR A history of CF in a sibling OR A positive newborn screening test PLUS Laboratory evidence for CF transmembrane regulator (CFTR) dysfunction:</td>
</tr>
</tbody>
</table>
Two elevated sweat chloride concentrations obtained on separate days
OR
Identification of two CF mutations
OR
An abnormal nasal potential difference measurement

<table>
<thead>
<tr>
<th>Gastroesophageal Reflux</th>
<th>See relevant topic in the manual</th>
</tr>
</thead>
</table>
| Primary Ciliary Dyskinesia | Clinical manifestations vary.  
Chronic persistent rhinorrhea, sensation of local fullness, and sinus pain  
Anosmia, nasal character of speech, and halitosis  
Recurrent acute otitis  
Chronic otitis  
Recurrent sinusitis  
Male infertility (common)  
Chronic productive cough and respiratory distress, especially in infants  
Bronchospastic symptoms (e.g., wheeze and cough), usually responsive to bronchodilator therapy  
Recurrent or persistent atelectasis or pneumonia  
Abnormal histology findings |

2. Bronchiolitis.

**Etiology.**

Bronchiolitis is an acute inflammatory process causing obstruction of the small conducting airways and a manifestation of lower respiratory tract obstruction. RSV is the most common cause (70% of bronchiolitis cases and 40% of pneumonia in younger children). Other infectious etiologies include parainfluenza virus (second most common cause of bronchiolitis; more often associated with croup, tracheobronchitis, and laryngitis), influenza virus, adenovirus, rhinovirus, *Mycoplasma pneumoniae*, *Chlamydia*, and ureaplasma. *Pneumocystis carinii* is rarely associated with wheezing in infancy. RSV causes epithelial damage and elicits a mononuclear cell infiltrate and peribronchiolar edema. Those predisposed to the development of reactive airways (asthma) may develop RSV-specific immunoglobulin-E (IgE) responses, presumably because of a high IgE-responder phenotype. Notably, 30%–40% of patients who develop severe wheezing with RSV later show a tendency to wheeze repeatedly.

**Epidemiology.**

RSV epidemic peaks from late fall to early spring. Up to 40% of primary infections result in febrile pneumonitis, but only 1% require hospitalization. Family studies indicate that nearly 70% of children are infected in the first year of life; by 24 months, nearly all children have been infected at least once. Beyond the first year, the clinical severity diminishes, changing from bronchiolitis and pneumonia to predominantly trachea-bronchitis and reactive airway events. Transmission is by droplets or fomites. The virus can remain infectious for hours on surfaces. Hospital-acquired infections are common.

**Symptoms.**

- Prodrome: 1–2 days of fever, rhinorrhea, mild cough.
- Apnea: may occur early before full intensity of chest symptoms.
- Persistent, increased cough: may later be productive.
- Rapid respirations.
- Skin color changes: rashes (rare).
- Poor feeding, lethargy.

**Signs.**

- Findings of rhinitis: occasionally otitis, conjunctivitis.
- Pharyngitis, hoarseness.
- Tachypnea with usually shallow respirations.
- Tachycardia: especially when hypoxemia present.
- Fever: usually milder later in course.
- Nasal flaring, retractions, and hyperinflation.
- Wheeze, increased expiratory phase, and rales and/or rhonchi.
- Palpable liver and/or spleen: secondary to hyperinflation.
- Cyanosis: Note: poor correlation with hypoxemia.
- Vomiting (post-tussive).
- Evidence for dehydration may be present: secondary to poor oral intake.

**Investigations.**
- Complete blood count: with differential (no specific findings).
- Pulse oximetry.
- Arterial blood gas if respiratory failure appears imminent: severe hypoxia, raised or rising partial pressure of carbon dioxide.
- Rapid viral identification: usually a “respiratory panel” is available to include common seasonal respiratory pathogens by antigen detection.
- Viral culture: results delayed but may be useful to identify causative organism.
- Serologic diagnosis: paired samples needed; rarely indicated as clinical diagnosis is usually evident.

**Chest radiograph.**
- Interstitial pneumonitis: the most typical finding; usually diffuse but may be segmental.
- Hyperaeration: also typical and may be the only finding.
- Peribronchial thickening: common but may not be related to the primary infection.
- Consolidation: occasional in hospitalized patients; usually is subsegmental.

**Complications.**
- Otitis media: is most common (secondary; bacterial).
- Pneumonia: secondary, bacterial; occurs in < 1% of hospitalized cases.
- Apnea.
- Respiratory failure.
- Cardiac failure: secondary to pulmonary disease or rarely myocarditis.
- Bronchiolitis obliterans: rare; usually associated with adenovirus-induced bronchiolitis/pneumonia.

**Differential diagnosis**

Pneumonia (viral, bacterial), *Chlamydia pneumoniae*, asthma, foreign body, cystic fibrosis, and pertussis. Gastroesophageal reflux. The diagnosis is evident when, during an epidemic period, an infant presents with tachypnea, diffuse wheeze, and hyperinflation (radiograph) after a febrile upper respiratory illness.

**Treatment.**

**Treatment aims:**
- To adequately monitor until resolution.
- To maintain oxygenation.
- To assess reversibility of airway obstruction (bronchodilator response).
- To avoid complications of treatment.
- To identify high-risk patients.

During the acute illness, routine health maintenance and respiratory care is provided at home or in hospital. Home management for mildly symptomatic patients is recommended. Adequate hydration should be assured. For significant wheeze or work of breathing, bronchodilator treatment (e.g., albuterol) may be tried. Reassess infant with increased respiratory distress and tachycardia (oxygen saturation).
Pharmacologic treatment.
Corticosteroids: although controversial, a trial is reasonable for hospitalized patients in whom bronchodilator responsiveness is documented. Theophylline: not useful as a bronchodilator but may be helpful for management of apnea. Antibiotics: not indicated unless secondary bacterial infection detected. Ribavirin treatment: this is controversial because of concerns regarding cost, benefit, safety and quite variable clinical efficacy (conflicting chemical trials); may be considered for patients who are at risk for severe or fatal infections, but no definitive indications have been established.

Immunoprophylaxis: RSV-intravenous immunoglobulin (RSV-IVIg) is approved for prevention of RSV disease in 1) children less than 2 years of age with bronchopulmonary dysplasia (BPD) who have been oxygen dependent at least 6 months prior to oncoming RSV season, and 2) selected infants with prematurity (gestational age < 32 weeks at birth) without BPD.

Intravenous hydration: monitor intake and output; avoid excessive hydration and aspiration. Consider use in severely immunodeficient patients (primary disorder such as severe combined immunodeficiency or severe HIV). RSV-IV-Ig is given at a dose of 750 mg/kg once per month beginning just before and monthly during the RSV season.

Nonpharmacologic treatment.
Infant showing inability to feed, severe respiratory distress, and/or hypoxemia should be hospitalized. Supplemental oxygen for saturation < 92%; titrate inspired oxygen to achieve >95% saturation. Note: nasal cannula may not effectively deliver oxygen if nasal passage not patent or patient mouth breaths. Bronchodilator trial: all hospitalized patients should receive a trial of an aerosolized beta-2-agonist for potential relief of obstruction. Monitor oxygen saturation concurrently as hypoxemia may worsen in some patients. Improvement suggests continuation may be beneficial, and that airway hyperreactivity associated with asthma may be present. Careful monitoring of vital signs and clinical status as patient may worsen during inpatient stay; use both electronic instruments and direct visual contact; include oximetry, and confirm progression to respiratory failure with arterial blood gas. Hospitalized patients should be isolated and may be cohorted in the same room. Use gown, gloves, and careful hand washing. Additional control measures may be advisable beyond patient isolation measures. Consider laboratory screening for RSV infection in patients, cohorting medical staff, exclusion of infected staff from contact with high-risk patents, and limitations on visitation.

Prognosis.
Acute severe obstructive symptoms usually resolve in 3–5 days, but cough and fatigue may last up to 14 days. Complete recovery expected for most patients. Patients with recurrent episodes of wheeze (obstruction) often found to have reactive airway disease. Chronic lung disease or other complications are rare in otherwise normal hosts.

Follow-up and management.
Most cases are uncomplicated and require no follow-up unless reactive air-way disorder is uncovered, complications arise, or the patient has another disorder that causes increased risk for severe or fatal RSV infection.

3. Differential approach to the treatment airway of obstruction

| Cystic Fibrosis | Pulmonary therapy - to clear secretions from airways and to control infection; Inhalation therapy - to deliver medications and hydrate the lower respiratory tract; The basic aerosol solution is 0.9% saline; In reactive airways, albuterol or other β agonists are added; When the airway pathogens are resistant to oral antibiotics or when the infection is difficult to control at home, aerosolized antibiotics |
| Airway Foreign Body | Bronchodilators and corticosteroids should not be used.  
Chest physical therapy with postural drainage may dislodge the material to an area where it may cause more harm.  
Endoscopic removal with a rigid bronchoscope.  
Endoscopist may observe enough focal swelling after the material is removed to recommend a short course of systemic corticosteroids.  
Antibiotics are not necessary. |
| Allergic Rhinitis | The removal and avoidance of offending allergens.  
Sealing the mattress, pillows, and covers in allergen-proof encasings.  
Wash bed linens and blankets every week in hot water (>65°C).  
Staying in a controlled environment in pollen allergy.  
Oral antihistamines as needed for ild, intermittent symptoms of sneezing and rhinorrhea.  
Nasal spray ipratropium bromide may be used for serous rhinorrhea.  
Intranasal decongestants should be used for <3–5 days, not to be repeated >1 cycle a month.  
Severe symptoms require intranasal corticosteroids, which are the most effective therapy for allergic rhinitis.  
Specific allergen immunotherapy. |
| Aspergillosis | Deepends on the form: in an immunocompromised host or allergic disease that includes allergic bronchopulmonary aspergillosis (ABPA).  
Voriconazole has now become the drug of choice for invasive aspergillosis. This is due to the increased efficacy and significantly less toxicity compared to amphotericin B.  
Caspofungin is a newer antifungal agent that is effective against invasive aspergillosis but more pediatric studies are needed prior to its widespread use. Currently caspofungin has been approved for use as salvage therapy for invasive aspergillosis that does not respond to existing antifungals.  
Treatment duration has not been well defined and is based on the clinical response and the tolerance to the drug. Continue therapy 4-12 weeks or longer.  
Itraconazole is used as prophylaxis in some cancer centers for immunocompromised patients.  
ABPA exacerbations are treated with corticosteroids.  
The desired goal is to reduce serum immunoglobulin E (IgE) levels to a range consistent with levels obtained from patients with asthma (without ABPA) living in the same geographic area.  
Reinstitution of corticosteroid therapy may be required if the serum IgE levels rise to twice this level or higher.  
Immediately obtain IgE levels after corticosteroid therapy. |
For asthma exacerbation, as indicated, administer other agents, such as beta-adrenergic agonists, high-dosage inhaled corticosteroids, and, possibly, nedocromil or theophylline. Administer prednisone as a single morning dose for 2 weeks and then convert to an alternate-day dosage for 3 months.

Systemic antifungal therapy is not indicated for ABPA

| Condition | Treatment
<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>Bronchiectasis</td>
<td>Inhaled $\beta$ agonists - for patients who respond, the medication should be continued, especially during high-risk periods when triggers are present, such as an upper respiratory infection or hot humid days; $\beta$-agonists may worsen the air exchange in infants with BPD and airway malacia; These patients may benefit from alternative bronchodilators such as inhaled ipratropium or oral methylxanthines; Inhaled glucocorticoids and leukotriene-modifying agents may be considered in patients with frequent inflammatory triggers; Adequate caloric intake; Fluid balance; Prevention of respiratory viral illness</td>
</tr>
<tr>
<td>Bronchiolitis</td>
<td>SEE DETAILS BELOW</td>
</tr>
<tr>
<td>Bronchopulmonary Dysplasia</td>
<td>Inhaled $\beta$ agonists - for patients who respond, the medication should be continued, especially during high-risk periods when triggers are present, such as an upper respiratory infection or hot humid days; $\beta$-agonists may worsen the air exchange in infants with BPD and airway malacia; These patients may benefit from alternative bronchodilators such as inhaled ipratropium or oral methylxanthines; Inhaled glucocorticoids and leukotriene-modifying agents may be considered in patients with frequent inflammatory triggers; Adequate caloric intake; Fluid balance; Prevention of respiratory viral illness</td>
</tr>
<tr>
<td>Primary Ciliary Dyskinesia</td>
<td>Pulmonary therapy - to clear secretions from airways and to control infection; Inhalation therapy - to deliver medications and hydrate the lower respiratory tract; The basic aerosol solution is 0.9% saline; In reactive airways, albuterol or other $\beta$ agonists are added; When the airway pathogens are resistant to oral antibiotics or when the infection is difficult to control at home, aerosolized antibiotics may reduce symptoms, improve pulmonary function, and alleviate the need for hospitalization; Human recombinant DNase for mucopurulent exudate; N-acetylcysteine, is toxic to ciliated epithelium, and repeated administration should be avoided; Hypertonic saline aerosols are reported to increase mucus clearance and improve pulmonary function. Benefit is quite variable and inferior, on average, to that achieved with DNase; Airway clearance therapy - chest percussion combined with postural drainage; Antibiotic therapy - to control progression of lung infection</td>
</tr>
</tbody>
</table>
3. Treatment of bronchial asthma

Treatment aims to:

- Find minimum treatment necessary to suppress symptoms and limit side effects.
- Maintain (near) “normal” pulmonary function.
- Avoid loss of time from school and other activities; avoid parental loss of work time (quality of life measures).
- Enable patient and/or family to take responsibility for day to day management of asthma.
- Reduce the frequency of exacerbations and to avoid hospital admissions.
- Exercise optimal environmental control measures.

**Lifestyle management.**

Excellent conditioning and a normal lifestyle is encouraged. The patient should not alter goals or activities based on asthma. Diet should be altered only if food, food additive (e.g., sulfite), or drug (e.g., ASA, nonsteroidal anti-inflammatory drugs) has been identified as a trigger.

**Pharmacologic treatment.**

Long-term control medications. Inhaled corticosteroids: anti-inflammatory; dosage dependent on severity and formulation; can be used for daily maintenance routine and at increased dosage for short-term management of exacerbations; use of spacer and mouth rinsing with water. Systemic corticosteroids: anti-inflammatory; for short-term “burst” to control exacerbation and for longer term in severe, persistent cases; main side effects include increased activity, appetite; Cushingoid appearance; growth suppression; osteoporosis. Systemic (methylprednisolone, prednisolone, prednisone): anti-inflammatory mechanism; for moderate to severe exacerbations (usually 3–5 day courses; up to 10 may be indicated); tapering the dose following improvement not indicated. Severe attacks should be treated in hospital with careful monitoring, oxygen, hydration, aerosol beta-2-agonist (intermittent or continuous), systemic corticosteroid (oral or parenteral, but initiated early); theophylline for those on maintenance.

Cromolyn and nedocromil: anti-inflammatory; safety is primary advantage of these agents; nebulizer delivery of cromolyn (20 mg/ampule) may be more effective than MDI (1 mg/puff).

Methylxanthines: predominantly bronchodilator mechanisms; standard dosage adjusted to yield blood concentration of 1015 mg/L; has a narrow therapeutic margin, multiple drug interactions; oral dosing may aid compliance; additive benefit with inhaled steroids.

Long-acting beta-2-agonists. Salmeterol (inhaled): bronchodilator; not to be used for acute symptoms or exacerbations because of slow onset of action; does not replace anti-inflammatory medication; tolerance can occur, but clinical significance is unknown.

**Oral agents:** inhaled route is preferred.

Leukotriene modifiers: new classes of anti-inflammatory drugs with great potential advantages; clinical experience in children is just beginning; leukotriene-receptor antagonists (multiple entities) and a 5-lipoxygenase inhibitor (Zileuton); special note: age indications vary.

Quick-relief medications. Short-acting inhaled beta-2-agonists. Albuterol, bitolterol, pirbuterol, terbutaline: bronchodilator mechanism; drugs of choice for intermittent symptom control and management of acute bronchospasm; regularly scheduled daily use not recommended; oral route not recommended.

Anticholinergics. Ipratropium bromide: bronchodilator mechanism (reduces vagal tone); may provide some additive effect to beta-2-agonist as supplement but not indicated as a primary drug.

4. Summary for the asthma treatment

<table>
<thead>
<tr>
<th>Step 4: Severe persistent asthma</th>
<th>Continuos symptoms</th>
<th>Daily medication required</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Frequent exacerbations</th>
<th>to maintain control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical activities limited</td>
<td>Multiple daily controller medications: long-acting bronchodilator and oral corticosteroids long term</td>
</tr>
<tr>
<td>PEF (peak expiratory flow readings)</td>
<td></td>
</tr>
<tr>
<td>&lt;60% predicted variability &gt;30%</td>
<td></td>
</tr>
</tbody>
</table>

**Step 3: Moderate persistent**

<table>
<thead>
<tr>
<th>Symptoms daily</th>
<th>Daily medication required to maintain control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exacerbations affect activity and sleep</td>
<td>Daily controller medications: inhaled corticosteroids and long-acting bronchodilator especially for nighttime symptoms</td>
</tr>
<tr>
<td>Nighttime asthma symptoms &gt;1 week</td>
<td></td>
</tr>
<tr>
<td>Daily use of inhaled short-acting beta-agonists</td>
<td></td>
</tr>
<tr>
<td>PEF (peak expiratory flow readings)</td>
<td></td>
</tr>
<tr>
<td>&lt;60% predicted variability &gt;30%</td>
<td></td>
</tr>
</tbody>
</table>

**Step 2: Mild persistent**

<table>
<thead>
<tr>
<th>Symptoms &gt; 1 time a week but &lt; 1 a day</th>
<th>Daily medication required to maintain control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exacerbations may affect activity and sleep</td>
<td>One daily controller medication: possibly add a long-acting bronchodilator to anti-inflammatory medication especially for nighttime symptoms (Cromones, corticosteroids)</td>
</tr>
<tr>
<td>Nighttime asthma symptoms &gt;2 a month</td>
<td></td>
</tr>
<tr>
<td>PEF (peak expiratory flow readings)</td>
<td></td>
</tr>
<tr>
<td>60-80% predicted variability 20-30%</td>
<td></td>
</tr>
</tbody>
</table>

**Step 1: Intermittent**

<table>
<thead>
<tr>
<th>Intermittent symptoms &lt;1 time a week</th>
<th>Daily medication required to maintain control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brief exacerbations (from few hours to few days)</td>
<td>Intermittent reliever medication taken as needed only short-acting beta-agonist</td>
</tr>
<tr>
<td>Nighttime asthma symptoms &lt;2 a month</td>
<td>Intensity of treatment depends on the severity of exacerbations. Corticosteroids, cromones may be required</td>
</tr>
<tr>
<td>Asymptomatic and normal lung function between exacerbations</td>
<td></td>
</tr>
<tr>
<td>PEF (peak expiratory flow readings)</td>
<td></td>
</tr>
<tr>
<td>&gt;80% predicted variability &lt;20%</td>
<td></td>
</tr>
</tbody>
</table>

**The long-term management of asthma: treatments in the stepwise approach for infants and young children**

**Step 4: Severe persistent asthma**

<table>
<thead>
<tr>
<th>Controller</th>
<th>Reliever</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily medication</td>
<td>Inhaled short-acting bronchodilator: inhaled beta₂-agonist or ipratropium bromide or oral beta₂-agonist as needed not to exceed 3-4 times daily</td>
</tr>
<tr>
<td>Nebulized budesonide 1 mg bid</td>
<td></td>
</tr>
<tr>
<td>If needed add oral steroids - lowest possible doses on an alternate day early morning schedule</td>
<td></td>
</tr>
</tbody>
</table>

**Step 3: Moderate persistent**

<table>
<thead>
<tr>
<th>Controller</th>
<th>Reliever</th>
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**Step 2: Mild persistent**

<table>
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<tr>
<th>Controller</th>
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<tr>
<td>Daily medication</td>
<td>Inhaled short-acting bronchodilator: inhaled beta₂-agonist or ipratropium bromide or oral beta₂-agonist as needed not to exceed 3-4 times daily</td>
</tr>
<tr>
<td>Nebulized budesonide or sodium cromoglycate</td>
<td></td>
</tr>
</tbody>
</table>
Step 1: Intermittent Controller
No controller therapy is needed

Reliever
Inhaled short-acting bronchodilator: inhaled beta$_2$-agonist or ipratropium bromide or oral beta$_2$-agonist as needed not to exceed 3-4 times week

At each step of therapy avoidance or control of trigger factors are required

5. Emergency care of status asthmaticus

- Admission to the hospital
- Supplemental oxygen
- Oxygen can be provided via nasal cannula or face masks.
- In significant hypoxemia, non-rebreathing masks may be used to deliver as much as 98% oxygen.
- The goal of supplemental oxygen therapy is oxygen saturation above 90%.
- Inhaled beta-agonists can be administered intermittently or as continuous nebulized aerosol in a monitored setting.
- Corticosteroids, such as methylprednisolone prednisolone or prednisone, are critical in the therapy of status asthmaticus
- Ipratropium bromide (Atrovent), a quaternary amine that does not cross the blood-brain barrier, is the recommended sympathomimetic agent of choice.
- Further therapy:
  - Magnesium sulfate
  - Intravenous beta-agonists
  - Ketamine
  - Methylxanthines
  - Inhaled anesthetic agents, such as halothane, isoflurane, and enflurane
  - Extracorporeal membrane oxygenation (ECMO)

6. MATERIALS FOR METHODOLOGICAL BACKGROUND OF THE WORKSHOP

6.1. Quiz

1. How do you differentiate airway foreign body aspiration?
2. How do you differentiate allergic rhinitis with bronchial obstruction?
3. How do you differentiate pulmonary aspergillosis?
4. How do you differentiate bronchiectasis?
5. Define bronchiolitis.
6. What is the most common pathogen for bronchiolitis?
7. List all pathogens involved into the development of bronchiolitis.
8. How many children develop recurrent wheezing after acquisition of RSV infection?
9. When do RSV peak?
10. At what age does clinical severity of bronchiolitis diminish?
11. How do RSV transmit?
12. What is and how long does the prodrome last?
13. Depict clinical manifestation of the bronchiolitis.
14. What are laboratory tests usually required in bronchiolitis?
15. What will chest radiograph show in acute bronchiolitis?
16. What are the complications for the bronchiolitis?
17. What is the differential diagnosis for the bronchiolitis?
18. What are the treatment aims?
19. What does the hospital care imply?
20. List drugs effective for the treatment of bronchiolitis.
21. What is the immunoprophylaxis for bronchiolitis?
22. What does nonpharmacological treatment of bronchiolitis include?
23. How rapidly do acute severe obstructive symptoms usually resolve in bronchiolitis?
24. What is follow-up in bronchiolitis?
25. What are treatment aims for bronchial asthma in children?
26. What is lifestyle management?
27. What is diet therapy?
28. Which inhaled corticosteroids do you know?
29. What is their action in bronchial asthma?
30. What adverse effects can be expected from the use of local corticosteroids?
31. What are systemic adverse effects can be expected from the use of local corticosteroids at high dosages?
32. What is primary advantage of cromolyn and nedocromil over local corticosteroids?
33. What is mechanism of action for these drugs?
34. Spell MDI abbreviation?
35. What do you know of a new class of anti-inflammatory drugs?
36. What groups of methyxanthines do you know?
37. What are adverse effects of methyxanthines?
38. What is safe serum concentration for methyxanthines?
39. What are drug interactions for methyxanthines?
40. What long-acting beta-2-agonists do you know?
41. What short-acting beta-2-agonists do you know?
42. How are these administered?
43. What are systemic adverse effects from the use of beta-2-agonists?
44. How do anticholinergics exert beneficial effect on to asthma?
45. List anticholinergic drugs you know.
46. What are controllers and relievers for severe persistent asthma?
47. What are controllers and relievers for moderate persistent asthma?
48. What are controllers and relievers for mild persistent asthma?
49. What are controllers and relievers for intermittent asthma?
50. What clinical picture indicates the need in severe persistent asthma therapy?
51. What clinical picture indicates the need in moderate persistent asthma therapy?
52. What clinical picture indicates the need in mild persistent asthma therapy?
53. What clinical picture indicates the need in intermittent asthma therapy?

6.2. Multi-choice questions
1. What may lead to the bronchiolitis?
   A. Staphylococcal infection
   B. Streptococcal infection
   C. Mycoplasma pneumoniae*
   D. Neisseria meningitidis
   E. E.coli

6.3. Sample case report
A 13 years old boy came to the doctor with symptoms of the pneumonia. In the anamnesis a boy had recurrent left sided lower lobe pneumonia and chronic cough in early childhood. On the physical examination decreased breath sound and dull area were revealed over the left lung base. Laboratory data showed leucocytosis. On X-ray was seen dense opacity of the left lower lobe. After the effective treatment of pulmonary infection with appropriate antibiotics, X-ray was repeated. It shows a mass in the posterior basal segment of the left lower lobe. CT scan with
contrast confirms a mass in posterior basal segment of the left lower lobe with blood supply from the thoracic aorta.

1. What is the diagnosis?
2. What is the differential diagnosis?
3. What is the follow-up?

Suggested reading

Additional reading