

Project: Ghana Emergency Medicine Collaborative

Document Title: Acute Asthma in Adults

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Background & Epidemiology

- Defined as a chronic inflammatory disorder characterized by increased airway responsiveness to multiple stimuli
- In susceptible people this results in recurrent episodes of wheezing, breathlessness, chest pain/tightness and or coughing

Background & Epidemiology

- Again as I have no epi data in Ghana it's unclear what the burden on this disease is.
- In the U.S. asthma is the number 1 chronic disease of childhood
- Affects 4-5% of the population
- Roughly 50% of the cases develop before the age of 10

Background & Epidemiology

- In childhood there is a male predominance of 2:1
- This equalizes by age 30
- Although the developed nations have seen a steady increase in the numbers of cases
- The number of hospitalizations and deaths have seen a steady decline since the mid 1990's

Pathophysiology

- This chronic inflammatory disease is characterized by abnormal accumulation of
 - Eosinophils, lymphocytes, mast cells, macrophages, dendritic cells and myofibroblasts
- The hallmark is the reduction of airway diameter that is caused by:
 - Smooth muscle contraction, vascular congestion, bronchial wall edema, and thick secretions

Pathophysiology

- This all results in the increased work of breathing and altered pulmonary function
- There is also a redistribution of pulmonary blood flow related to this inflammatory process
- The current theory is that there are three phases of the airway inflammation

Pathophysiology

- Acute phase:
 - There is early recruitment of cells to the airway
 - Antigens come in contact with mast cells in the submucosa and lead to elaboration of inflammatory mediators
 - Such as histamine, leukotrienes, chemokines and pro-inflammatory cytokines and multiple interleukins
- This leads to increased mucus production, decreased ciliary action and of course bronchoconstriction and vascular congestion

Pathophysiology

- Sub acute/ Late phase:
 - The eosinophils, platelets and polymorphonuclear leukocytes that are recruited, become activated
 - IgE response is also activated by the contact with the antigen
- This all creates a more persistent pattern of inflammation

Clinical Features

- Typically the symptoms consist of a triad of dyspnea, wheezing and cough
- Many patients will relay a history of asthma on presentation
- Early in the process they may describe the discomfort as a chest tightness or constricted feeling

Clinical Features

- As the exacerbation progresses the wheezing become evident
- Expiratory phase become prolonged
- Accessory muscle use may become evident

Physical Examination

- The physical exam can vary greatly depending on the patient and phase of presentation
- Some maybe in obvious respiratory distress with tachypnea and loud wheezing
- Others may present with persistent cough and mild end-expiratory wheeze
- The use of accessory muscles indicates diaphragmatic fatigue

Physical examination

- Signs of impending respiratory failure include
 - Paradoxical respirations
 - Alterations in mental status
 - Hallucinations, agitation, confusion
 - “silent” chest

Physical Examination

- Findings may reveal
 - Hyper resonance to percussion
 - Decreased air movement
 - Prolonged expiratory phase
 - Usually wheezes
 - Pulses Paradoxus
 - Tachypnea
 - Tachycardia

Evaluation

- Bedside spirometry provides a rapid objective assessment
- Can serve as a guide to the effectiveness of your therapy
- Can be measured as the Forced Expiratory volume in 1minute (FEV1) or as the Peak Expiratory Flow Rate(PEFR)

Evaluation

- Chest X-ray
- Arterial Blood gas
- CBC
- Electrolytes
- Other studies

Management

- The primary goals of therapy for acute severe asthma are the rapid reversal of airflow obstruction and the correction.
- Airflow obstruction is most rapidly alleviated by the combination of repeated administration of inhaled bronchodilators and early institution of systemic glucocorticoids .
- Until their respiratory distress has abated, patients should receive close monitoring, including serial measurements of lung function (FeV1 or PEFr), to assess the response to treatment.

Management

- Inhaled beta agonists :
- Are the mainstay of bronchodilator treatment.
 - short-acting beta-2-selective adrenergic agonists, such as albuterol, levalbuterol.
- The standard regimen for initial care in the emergency department has become albuterol 2.5 to 5 mg by continuous flow nebulization every 20 minutes for three doses
- Then 2.5 to 10 mg every one to four hours as needed.
- For critically ill patients, some clinicians prefer continuous nebulization, administering 10 to 15 mg over one hour.

Management

- Inhaled anticholinergics
 - The most recent guidelines for asthma management prepared by the Expert Panel of the National Asthma Education and Prevention Program (Expert Panel Report III) recommend the addition of ipratropium for patients with severe exacerbations who are in the emergency department.
- The adult dosing of ipratropium for nebulization is 500 mcg every 20 minutes for three doses.
- Alternatively, ipratropium can be administered by MDI at a dose of eight inhalations every 20 minutes, then as needed for up to three hours.
- Some investigators have reported that the combination provides greater bronchodilation than beta agonists alone, particularly among patients with the most severe airflow obstruction.

Management

- Systemic glucocorticoids
 - Continued wheezing and shortness of breath despite intensive bronchodilator therapy most likely represents persistent airflow obstruction on the basis of airway inflammation and intraluminal mucus plugging.
 - Studies show that among patients with significant airflow obstruction despite intensive treatment with bronchodilators, systemic glucocorticoids speed the rate of improvement .
- Current guidelines encourage early systemic glucocorticoids for all patients who have a moderate (peak expiratory flow <70 percent of baseline) or severe exacerbation (peak expiratory flow <40 percent of baseline)
- Oral vs. IV administration?

Management

- Orally
 - Prednisone 40-60mg
- IV
 - Methylprednisone 60-125mg IV
 - Hydrocortisone 100-200mg IV

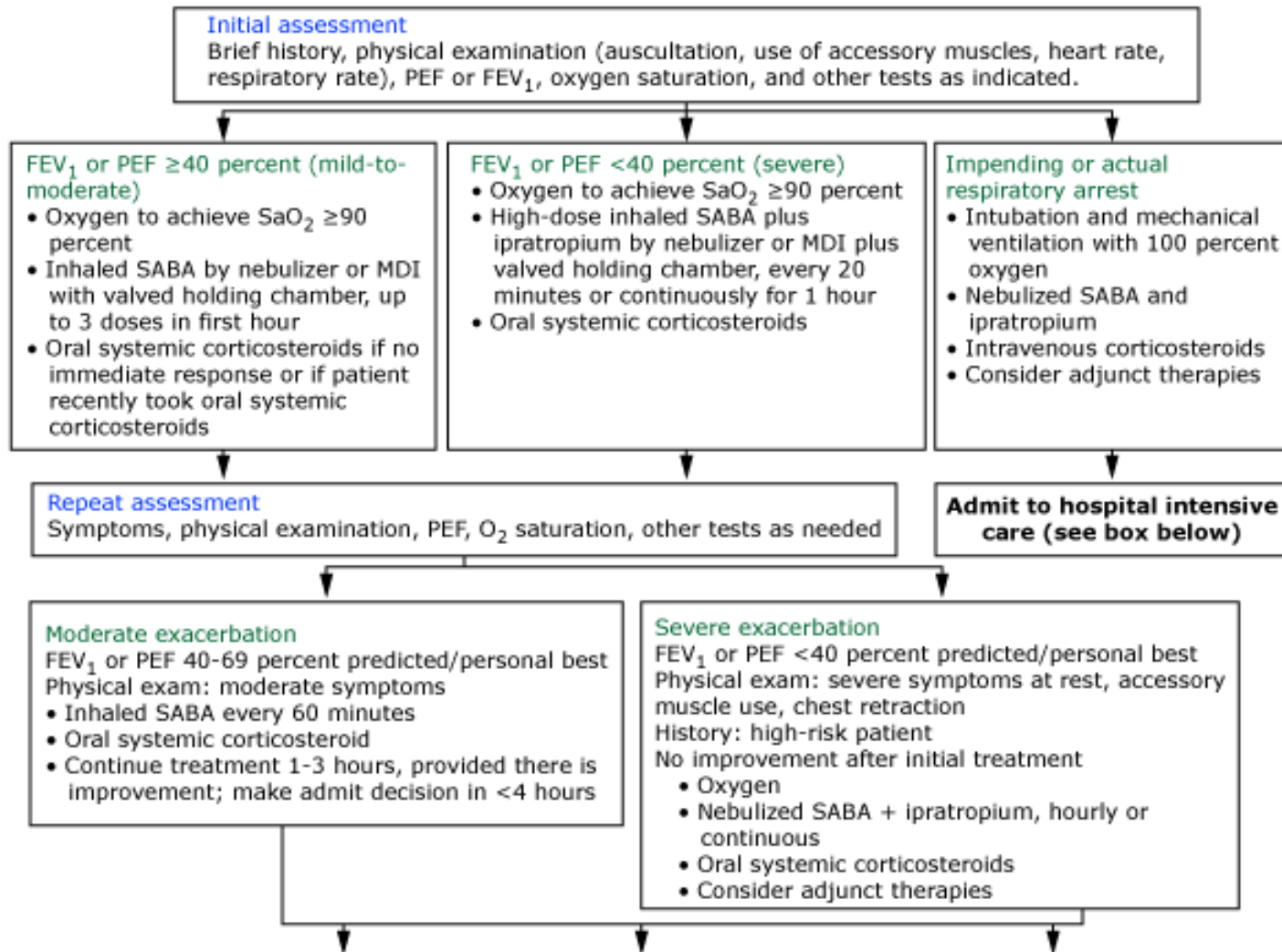
Management

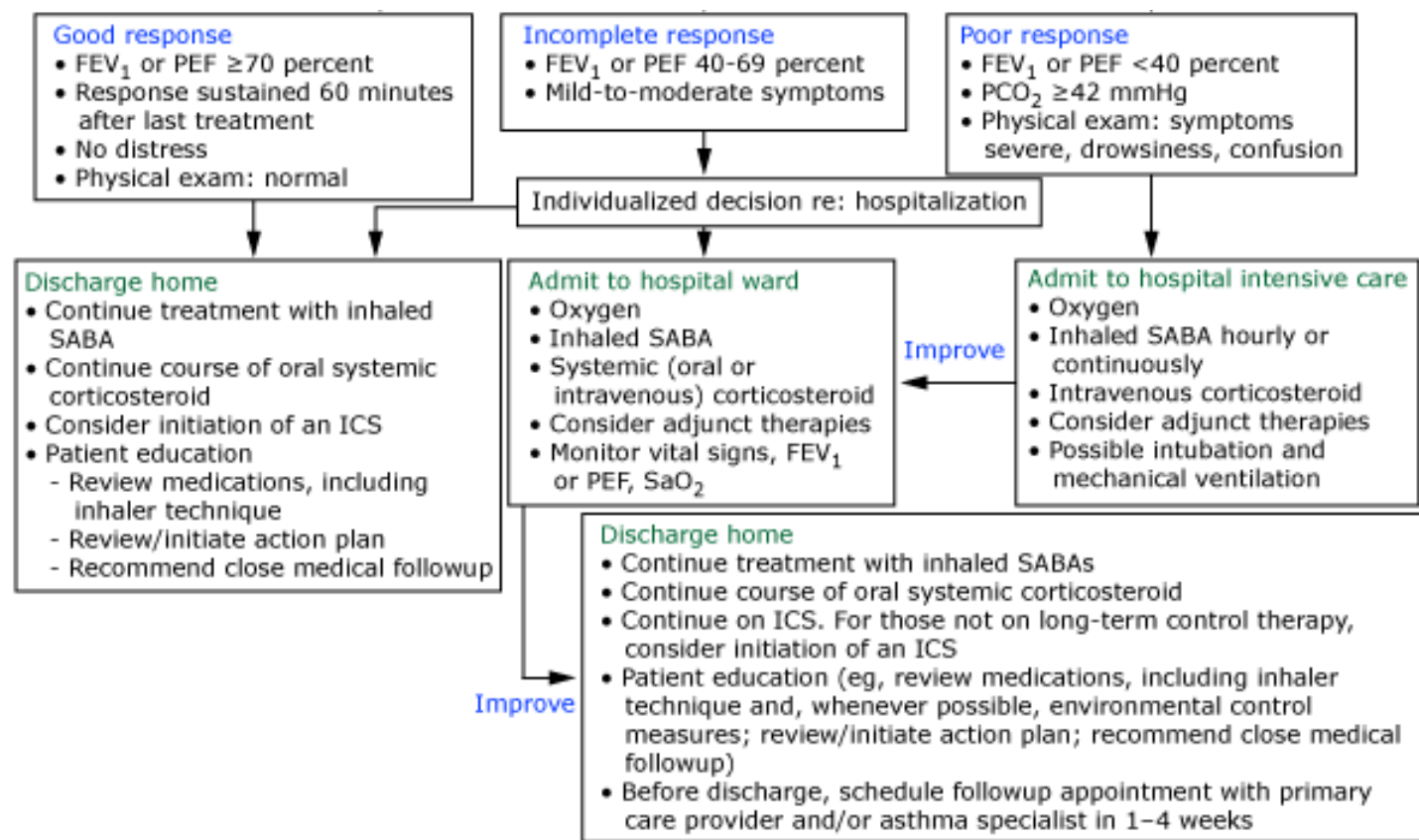
- Magnesium sulfate —
 - Intravenous magnesium sulfate (2 gm infused over 20 min) has bronchodilator activity in acute asthma.
 - two systematic reviews concluded that it is helpful in the subgroup of patients with severe attacks.
- This agent is suggested for patients who have life-threatening exacerbations or whose exacerbation remains severe

Older Alternatives

- Methylxanthines —
 - The use of alternative bronchodilators such as intravenous theophylline or aminophylline, in addition to beta-agonists, is not recommended in the treatment of acute exacerbations .
 - These agents are not as potent as the beta agonists when used alone for the treatment of asthma , and provide no further bronchodilation beyond that achieved with inhaled beta agonists alone.
 - In addition, these agents appear to increase the incidence of adverse effects when combined with bronchodilators.
 - Studies extending over several hours of emergency department care have also failed to show a benefit of theophylline therapy in terms of course or duration of hospitalization.
 - For patients who are taking oral theophylline at presentation, we typically continue maintenance oral therapy (ideally after checking a blood level.)

Management of asthma exacerbations: emergency department and hospital-based care





FEV₁: forced expiratory volume in 1 second; ICS: inhaled corticosteroid; MDI: metered dose inhaler; PCO₂: partial pressure carbon dioxide; PEF: peak expiratory flow; SABA: short-acting beta₂-agonist; SaO₂: oxygen saturation.

Reproduced from: National Heart, Blood, and Lung Institute Expert Panel Report 3 (EPR 3): Guidelines for the Diagnosis and Management of Asthma. NIH Publication no. 08-4051, 2007.

Usual dosages for oral glucocorticoids in asthma

Medication	Dosage form	Adult dose	Comments
Systemic corticosteroids		(Applies to all three corticosteroids)	
Methylprednisolone	2, 4, 8, 16, 32 mg tablets	7.5-60 mg daily in a single dose in am or qod as needed for control	<p>For long-term treatment of severe persistent asthma, administer single dose in am either daily or on alternate days (alternate-day therapy may produce less adrenal suppression). If daily doses are required, one study suggests improved efficacy and no increase in adrenal suppression when administered at 3:00 pm (Beam et al 1992).</p> <p>Short courses or "bursts" are effective for establishing control when initiating therapy or during a period of gradual deterioration.</p> <p>The burst should be continued until patient achieves 80 percent PEFr personal best or symptoms resolve. This usually requires 3 to 10 days but may require longer. There is no evidence that tapering the dose following improvement prevents relapse.</p>
Prednisolone	5 mg tablets, 5 mg/5 mL, 15 mg/5 mL	Short-course "burst": to achieve control 40 to 60 mg per day as single or two divided doses for 3 to 10 days	
Prednisone	1, 2.5, 5, 10, 20, 25 mg tablets; 5 mg/mL, 5 mg/5 mL		

PEFR: peak expiratory flow rate; qod: every other day.

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Severe asthma exacerbation in adults: Rapid overview

Clinical Danger Signs

- Use of accessory muscles of respiration; brief, fragmented speech; inability to lie supine; profound diaphoresis; agitation
- Life-threatening airway obstruction can still occur when these signs are **NOT** present
- **Inability to maintain respiratory effort, cyanosis, and depressed mental status portend imminent respiratory arrest**

Assessment

- Measurement of expiratory airflow is the best measure of severity; peak expiratory flow rate < 40 percent predicted indicates severe obstruction
- **Severe hypoxemia** (SpO₂ < 95% despite high flow O₂ by nonrebreather mask) portends imminent respiratory arrest; continuous pulse oximetry should be performed
- Arterial blood gas evaluation does not assist management; it can aid assessment if intubation is not an immediate concern
- Chest radiograph is generally unhelpful; obtain if complications suspected, diagnosis in doubt, or patient is high-risk

Standard treatments

- Inhaled beta agonist (albuterol)
- Oxygen
- IV access, normal saline
- Ipratropium bromide
- Corticosteroids
- Magnesium sulfate

Additional treatments

- Terbutaline
- Epinephrine
- Heliox (helium and oxygen)

Endotracheal intubation and ventilation

- Decision to intubate during first few minutes of severe asthma attack is clinical
- Goal of mechanical ventilation is to maintain adequate oxygenation and ventilation while minimizing elevated airway pressures

Predicted average peak expiratory flow for normal males (L/min)

Age	Height				
	60"	65"	70"	75"	80"
20	554	602	649	693	740
25	543	590	636	679	725
30	532	577	622	664	710
35	521	565	609	651	695
40	509	552	596	636	680
45	498	540	583	622	665
50	486	527	569	607	649
55	475	515	556	593	634
60	463	502	542	578	618
65	452	490	529	564	603
70	440	477	515	550	587

These values represent average normal values within 100 L/min. Predicted values for African American and Hispanic minorities are approximately 10 percent lower.

Redrawn from Leiner, GC, et al, Am Rev Respir Dis 1963; 88:644.

Predicted average peak expiratory flow for normal females (L/min)

Age	Height				
	55"	60"	65"	70"	75"
20	390	423	460	496	529
25	385	418	454	490	523
30	380	413	448	483	516
35	375	408	442	476	509
40	370	402	436	470	502
45	365	397	430	464	495
50	360	391	424	457	488
55	355	386	418	451	482
60	350	380	412	445	475
65	345	375	406	439	468
70	340	369	400	432	461

These values represent average normal values within 80 L/min. Predicted values for African American and Hispanic minorities are approximately 10 percent lower.

Redrawn from Leiner, GC, et al, Am Rev Respir Dis 1963; 88:644.

Predicted average peak expiratory flow rates for normal children

Height	PEFR	Height	PEFR
(inches)	(L/min)	(inches)	(L/min)
43	147	56	320
44	160	57	334
45	173	58	347
46	187	59	360
47	200	60	373
48	214	61	387
49	227	62	400
50	240	63	413
51	254	64	427
52	267	65	440
53	280	66	454
54	293	67	467
55	307		

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