Bronchial asthma

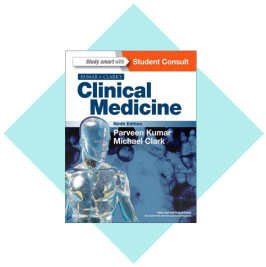
Objectives:

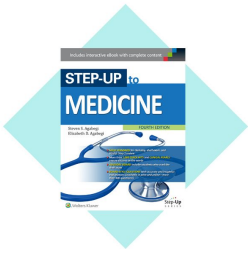
- Know the basic definition.
- Learn some epidemiology.
- Explain methods of diagnosis.
- Discuss treatment options.
- Follow prevention measures.

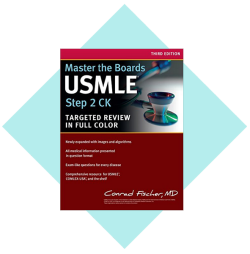
[Color index : Important | Notes | Extra]

Resources:

- 435 slides







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"Medicine is an art, nobody can deny it."

Objective 1+ 2 : Know the basic definition and learn some Epidemiology

Bronchial Asthma:

Asthma is a **chronic[1], heterogeneous inflammatory disorder** of the airway , that has three characteristics:

1. **Airway hyper-responsiveness** that causes **recurrent episodes of coughing, wheezing[2], breathlessness, and chest tightness,** which are typically **worse at night and in early morning.**

- they may have small thick sticky mucoid whitesh sputum. In case the pt is diagnosed with COPD + he is smoker = purulent sputum. Lots of sputum indicates bronchiectasis.
- you need to know the DDx for pt. With such symptoms: asthma, COPD, bronchiectasis, HF.
- look to the family history (in asthma "**only due to allergy not in other types**" they do have FHx) + smoking Hx (in COPD they do have)+ occupational.. "

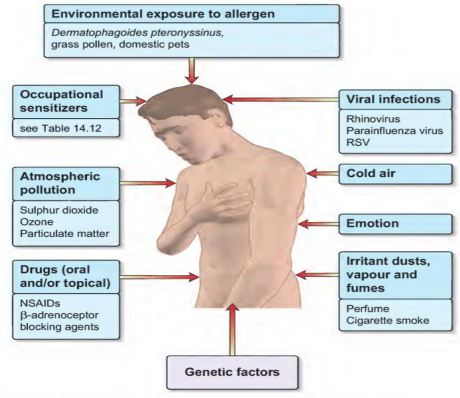
2. A widespread but variable **airflow obstruction[3]**, which is **reversible** either spontaneously or with treatment.
3. **Inflammation of the bronchi** where Many cellular elements play a role, in particular: **T lymphocytes, mast cells, eosinophils, neutrophils, macrophages, and epithelial cells**, associated with plasma exudation, oedema, smooth muscle hypertrophy, matrix deposition, mucus plugging and epithelial damage.

Epidemiology:

- Start at any age, (75% Dx age <7 "childhood").
- Remission around **puberty**.
- Prevalence is **increasing** with Wide geographical variation (4-25%, more common in more developed countries) and higher prevalence in **females**
- Likely Multifactorial.
- **Environmental factors** are critically important in the development and expression of the disease.
- Asthma causes significant **morbidity and cost** to healthcare resources (10%) especially severe asthma. **Asthma does kill !!**

Etiology :

Although asthma is **multifactorial** in origin " genetic+environmental" , inflammation is believed to be the cornerstone of the disease and is thought to result from **inappropriate immune responses** to a variety of antigens in genetically susceptible individuals.



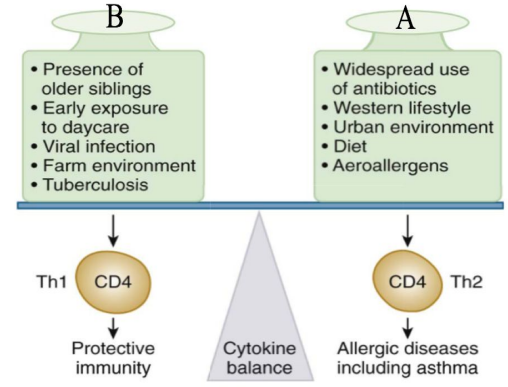
[1] **Once it occurs it will stay for life.**
[2] Not every wheezes is asthma, e.g. In HF pulmonary edema , pt will have a lots of wheezing "cardiac asthma".Cardiac asthma is not a form of asthma. It's a type of coughing or wheezing that occurs with left heart failure.
[3] In chronic asthma, inflammation may be accompanied by irreversible airflow limitation as a result of airway wall remodelling that may involve large and small airways and mucus impaction. Kumar 7th edition p 846.

◆ Causes [4]: (once we have a long list of causes this means that we aren't sure what is really the cause!)

- **Atopy and allergy[5]," most common cause"**
- **Genetics**
- Smoking "controversial, long time smokers probably COPD"
- obesity " under investigation".
- **Hygiene Hypothesis:**

If a child has a more contaminated environment he gains stronger immunity. While having a less contaminated environment (western lifestyle) may predispose to asthma.

This proposes that infections in early life are critically important in maturation and bias of the immune system against the development of allergies. It is suggested that the high prevalence of allergic disease is the penalty for the decreased exposure to infection that has resulted from improvements in sanitation and health care!!



A- in developed societies (use of antibiotics, reduced exposure to pathogens "clean environment" may predispose towards an IgE response to **allergens (Th2 pathway).**
B- growing up in "dirty environment" allow the immune system to avoid developing allergic response. "organism component > immune response toward the **protective pathway (Th1) and Treg".**
Kumar 7th edition p 847

- A number of factors predispose to allergic diseases, the strongest of which is a **family history**[6] and **Contributory environmental factors**[7].
  - See davidson p89.

○ Asthma due to specific triggers:

◇ Occupational asthma:

- accounting for 15% of all asthma cases
- The risk of developing some forms of occupational asthma increases in smokers.
- The proportion of employees developing occupational asthma depends primarily upon the level of exposure.
- Proper enclosure of industrial processes or appropriate ventilation greatly reduces the risk.
- Atopic individuals develop occupational asthma more rapidly when exposed to agents causing the development of specific IgE antibody .
  - Non-atopic individuals can also develop asthma when exposed to such agents, but after a longer period of exposure.

Table 14.12 Occupational asthma

Cause	Source/Occupation
Low molecular weight (non-IgE related)	
Isocyanates	Polyurethane varnishes Industrial coatings Spray painting
Colophony fumes	Soldering/welders Electronics industry
Wood dust	
Drugs	
Bleaches and dyes	
Complex metal salts, e.g. nickel, platinum, chromium	
High molecular weight (IgE related)	
Allergens from animals and insects	Farmers, workers in poultry and seafood processing industry; laboratory workers
Antidotes	Nurses, health industry
Latex	Health workers
Proteolytic enzymes	Manufacture (but not use) of 'biological' washing powders
Complex salts of platinum	Metal refining
Acid anhydrides and polyamine hardening agents	Industrial coatings

- In such type pt. Will feel better in holydays.

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[4] Once we have a long list of causes that means we are **NOT** sure what causes it.

[5] Triggers include pollens, house dust, molds, cockroaches, cats, dogs, cold air, viral infections, smoking, **medications (β -blockers, aspirin and NSAIDs**), and **exercise**.

[6] genes controlling innate immune responses, cytokine production, IgE levels and the ability of the epithelial barrier to protect against environmental agents.

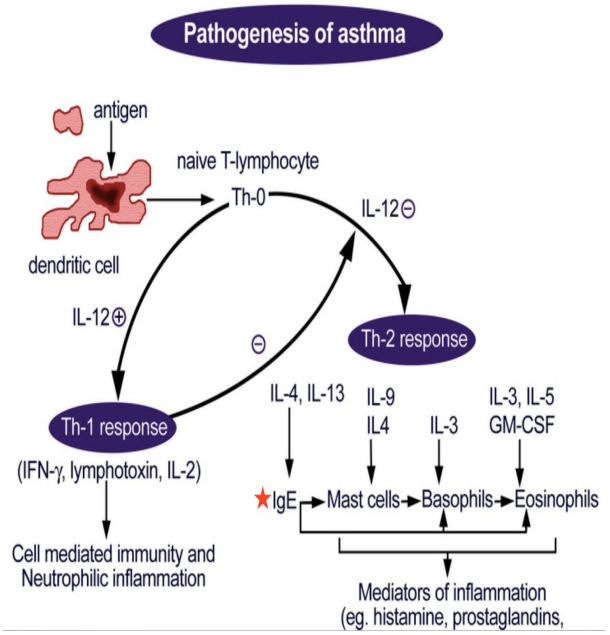
[7] include bacterial and viral infection, pollutants and cigarette smoke.

Pathophysiology: Pathophysiology of asthma: 7:45

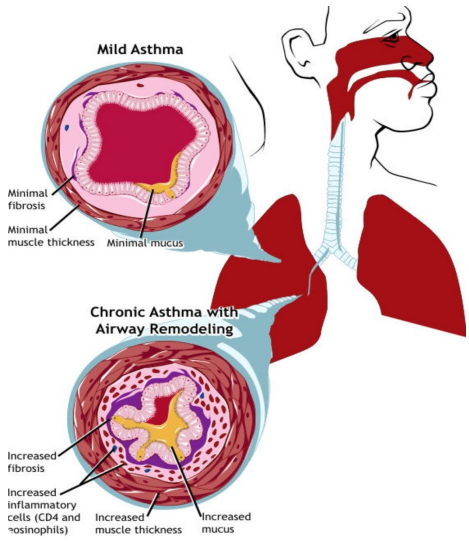
The pathogenesis of asthma is complex and not fully understood. It involves a number of cells, mediators, nerves and several different mechanisms, of which exposure to allergens is among the most significant.

- Cells involved are **dendritic cells, CD4 Th2 [8]lymphocytes** " abundant in the mucous membranes of the airways and the alveoli." , the release of their cytokines plays a key part in the inflammation process :

Type of cytokine:	IL13, IL9, IL4, IL3	IL-3, IL-5, GM-CSF
The activated cell:	**Mast cells** "Release histamine and other cytokines".	**Eosinophils** "Release basic proteins and leukotriene"
The effect of activated cell:	On the smooth muscles and mucosa of the airway, blood vessels and sensory nerves. -contribute in both immediate and late asthmatic reaction	On the bronchial wall and secretions - Sputum eosinophilia.



- The varying clinical severity and chronicity of asthma is dependent on an interplay between airway inflammation and airway wall remodelling.
  - **Remodeling** occurs to the epithelium, basement membrane, smooth muscles, and nerves:
    - Smooth muscles become **thickened** and, **mucosa swollen** with **increased secretion,** leading to fixed narrowing of the airway and a reduced response to bronchodilator medication.
    - The epithelium becomes damaged replaced by **fibrosis** , basement membrane thickened and the nerves cause more irritability.



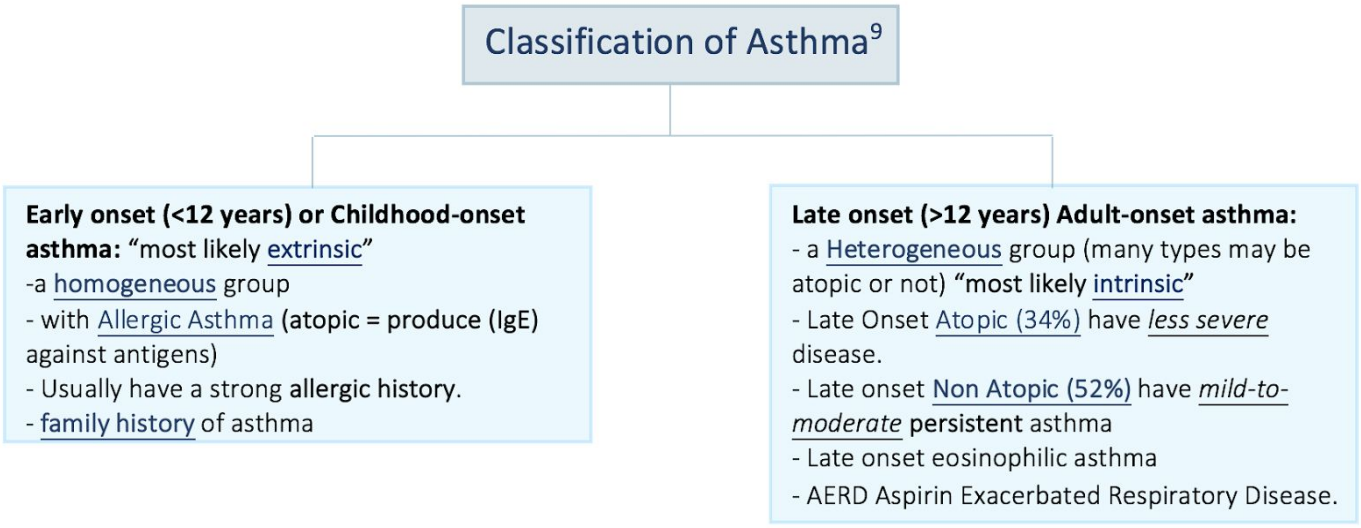
" asthmatic pts have good days and a bad days, but after many years they may develop more prominent airway limitation "

[8] Now, we mainly focus on Th2

Clinical features:

- **Typical symptoms** include, recurrent[9] episodes "intermittent" of : **wheezing**, chest tightness, breathlessness and cough[10], in **diurnal pattern.**[11]
    - Usually occur within 30 minutes of exposure to triggers.
    - There is a symptoms fluctuation over the course of one day, or from day to day or month to month.
    - poorly controlled, symptoms such as cough and wheeze disturb sleep and have led to the term 'nocturnal asthma'.
- **Classical precipitants** include exercise, particularly in cold weather[12], exposure to airborne allergens or pollutants, viral upper respiratory tract infections, and medications.

Classification: these are the 2 board asthma phenotype (not endotype).



★ Types:

First put this scenario in your head:

Different Pts. come to you with the same symptoms of asthma, if you in were 10 years ago you will give all of them the same treatment which is steroids, … but now things have changed and we know different types of asthma " allergic, eosinophilic, neutrophilic,.. Etc" that will change the drug of choice depending on it, so you have to keep up with the changes and learn some new words that will be your **future medicine.**

[9] commonly mistaken for a cold or chest infection which is taking time to resolve.

[10] When Cough is the dominant symptom in some patients, and the lack of wheeze or breathlessness may lead to a delay in reaching the diagnosis of so-called 'cough-variant asthma'.

[11] symptoms and lung function being worse at night and in the early morning.

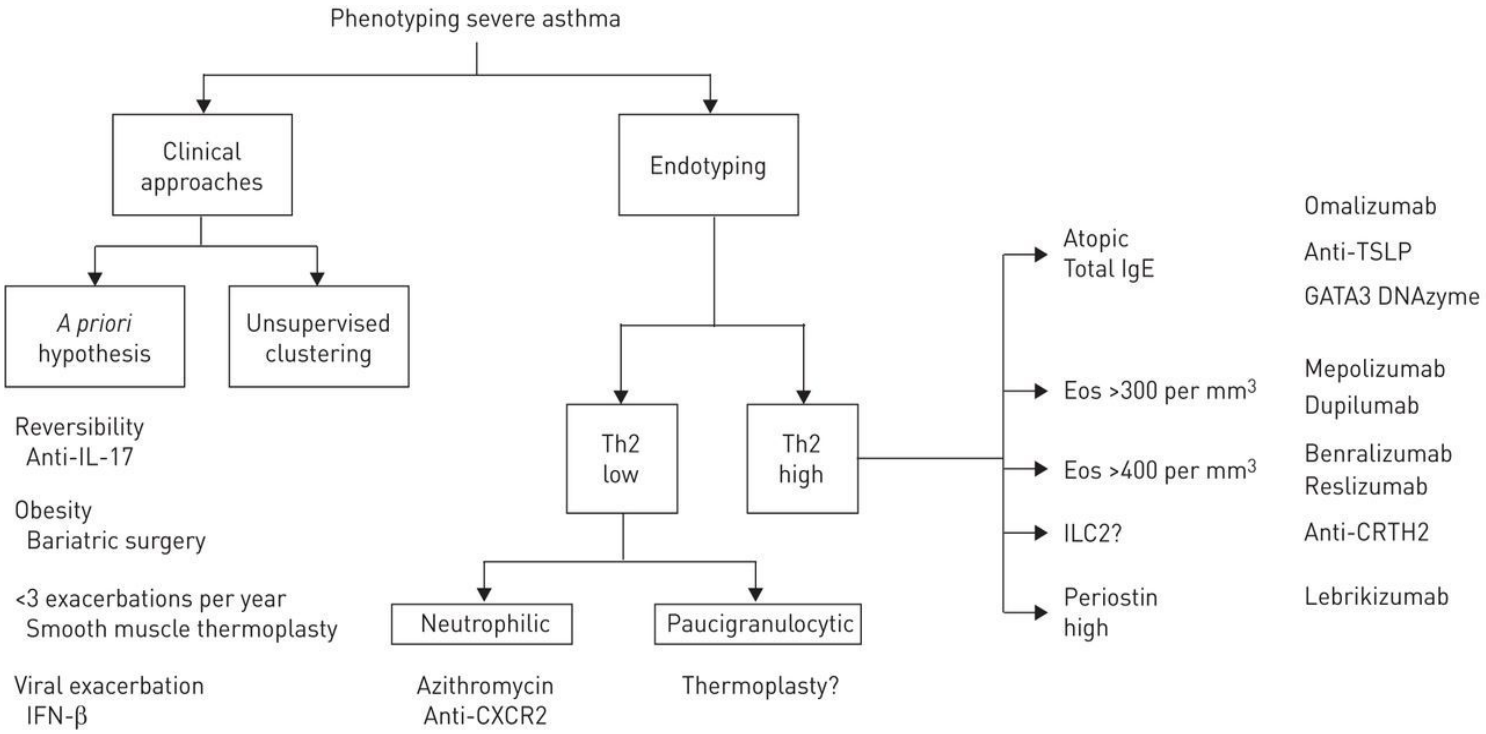
[12] winter sports enthusiasts

- **Phenotype:**

  Is a **clinical character** like: age, gender, obesity, smoking and even the **pathophysiology**.

- **Endotype:**
  - **Precision medicine (personalized medicine).**
  - Not treating as one asthma , instead **treat according to the type** of asthma (eosinophilic, allergic, etc…), **WHY ??**
  - **Because different pathophysiological mechanism,** so distinguish the pathological mechanism that is causing the disease then treat.
  - Endotyping is significant because it tells us how to treat the disease with different types of mechanism (target the mechanism).
- **Mixed or overlapping features:**

Some with eosinophilic asthma may have allergic component as well, like aspirin sensitive asthma.



Objective 3: Explain methods of diagnosis

History :

- Symptoms: SOB, Wheeze, chest tightness, Usually dry Cough, Sputum small mucoid
- if symptoms are episodic, and triggers .

★Very Important:

Initial structured clinical assessment of asthma
Factors to consider in an initial structured clinical assessment include: • **Episodic symptoms:** ○ More than one symptoms of asthma occur in episode, With period of no (or minimal) symptoms between the episode, e.g.: - Recurrent intermittent episode of triggered symptoms by any allergen or medication. - Acute wheezing with improvement after treatment. ○ Monitor **FEV_1 or PEF** during and after the episode, if **lower** "during" than "after" > obstructive nature. • **Wheezing:** ○ Repeated normal examination during the episode will decrease the probability of asthma. • **Evidence of diurnal variability.** • **Atopic history:** ○ **PHx** of: eczema, allergic rhinitis, or **FHx** of asthma, ○ **Record** of: raised antigen-specific IgE level, positive skin-prick test to aeroallergen or **blood eosinophilia**. Absence of symptoms, signs or clinical Hx suggest **alternative diagnosis.**

Clinical Feature that ↑ the probability of asthma	Clinical Feature that ↓ the probability of asthma
○ **More than one of the following symptoms:** • **wheeze, breathlessness, chest tightness and cough, particularly if:** **- symptoms worse at night and in the early morning** **- symptoms in response to exercise, allergen exposure** **- symptoms after taking aspirin or beta blockers.** ○ **History of atopic disorder,** ○ **Family history of asthma and/or atopic disorder** ○ **Widespread wheeze heard on auscultation of the chest** ○ **Otherwise unexplained low FEV, or PEF (historical or serial readings)** ○ **Otherwise unexplained peripheral blood eosinophilia.**	○ **Prominent dizziness, light-headedness peripheral tingling** ○ **Chronic productive cough in the absence of wheeze or breathless** ○ **Repeatedly normal physical examination of chest when symptomatic** ○ **Voice disturbance** ○ **Symptoms with colds only** ○ **Significant smoking history (i.e. > 20 pack- years)** ○ **Cardiac disease** **Normal PEF or spirometry when symptomatic**

◆ [13]Differential Diagnosis:

- Other Illness with **wheezing[14] / SOB:**

- **COPD (Smoker)**, - Bronchiectasis (Large amount of sputum), - Heart failure / Pulmonary Edema, - Airway obstruction (Tumors, FB), - Vocal cord dysfunction

- May **Coexist** and complicate Dx of asthma:

- GERD, - OSA (obstructive sleep apnea), - ABPA (Allergic bronchopulmonary aspergillosis).

[13] " If I'm going to put a third year MCQs I will concentrate on HISTORY ,HISTORY, HISTORY. What makes asthma more or less likely . "

[14] No wheezing either means no asthma or the pt. in his good day, it should be heard if pt. Was in the episode, unless he is having a silent chest . "remember for any rule there is exceptions"

Examination

Upper respiratory tract	nasal secretion, mucosal swelling, **nasal polyp** "common in aspirin sensitive asthma or eosinophilic"
Chest	**Wheezing*** or prolonged phase of forced exhalation, Chest hyper-expansion, accessory muscles.
Skin	atopic dermatitis, eczema "

* Wheezing is a **high-pitched** whistling sounds when breathing out "expiration".

- **A lack of wheezing and a normal chest examination do not exclude asthma**.
- wheezing sometimes is not present , so we could say whenever the patient gets exertion he will develop SOB and when he relax the symptom **will not improve**. Why? Because the **inflammation is still there in the lung**. especially in obese. Cleer!!!!
- Assume the pt. Is obese and he came to you with SOB, how to know whether it is due to obesity or asthma:

1. Pt. will describe the symptom with association to walking, and it will stop after rest. " asthma is an inflammation once it comes it should not go away.
2. Listen to their chest if it is clear " no wheezing" that would probably be due to the extra weight.
   - We have to be casueouse in such case.[15]

Investigations:

Asthma Dx – **variable airflow limitation:**

- Confirm presence of **airflow limitation:**
  – Document that FEV1/FVC is reduced <0.75 (at least once).
- Confirm variation in lung function or **Reversibility:**
  – Excessive bronchodilator reversibility (FEV1 >12% and >200mL)
  – Excessive diurnal variability twice-daily PEF monitoring.

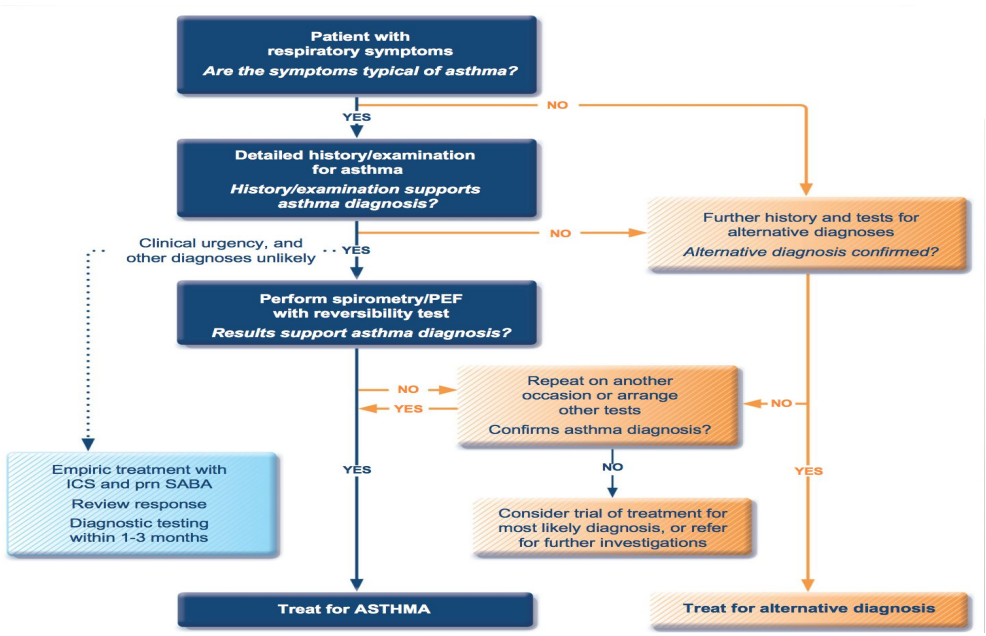
[15] there is a phenotype that's called obese female phenotype, they are symptomatic and their asthma is worse than the other phenotype because obesity considered a proinflammatory condition.

Pulmonary function test (PFT):

required[16] for diagnosis, it shows: **1.obstructive pattern 2. decrease in expiratory flow rates, 3.decreased FEV1 and decreased FEV1/FVC ratio (<0.70).**

1.spirometry (routine).	- shows decrease in the **FEV1 and FVC ratio.** - Diagnosing and assessing reversibility[17] If inhalation of a bronchodilator (β 2-agonist) results in an **increase in FEV1 or FVC by at least 15% (200ml)**, airflow obstruction is considered reversible.
2.Peak expiratory flow rate (PEF)[18]	- Measure of airflow obstruction by the pt, " self-monitoring ". - A **diurnal** variation in PEF of more than **20%** (the lowest values typically being recorded in the morning) is considered diagnostic[19] .
Others:[20]	
Arterial blood gases	- patients with an asthma attack > increased respiratory rate, so **PaCO2 decreases**[21]
Chest X-ray	**Normal in mild cases**; severe asthma reveals hyperinflation. -Only necessary in severe asthma **to exclude other conditions** (e.g., pneumonia, pneumothorax, pneumomediastinum, CHF and foreign body).
Bronchoprovocation test	- The patient is given a trigger. If a reaction occurs, he is probably asthmatic. **Useful when the main symptom is cough**.
Measurement of allergic status	-**Skin tests**: done in all asthmatic patients to identify allergen. - total and allergen-specific **IgE**. A full blood picture may show the peripheral **blood eosinophilia.**

Diagnostic Approach:



[16] The most accurate diagnostic test.
[17] spirometry with a bronchodilator can confirm diagnosis by proving reversible airway obstruction.
[18] If spirometry not available.
[19] diurnal variability on 2 sets of PEF indicates reversibility.
[20] Exercise test: in children.
[21] Increased PaCO2 is a sign of respiratory muscle fatigue or severe airway obstruction.

Objective 4: discuss the treatment options.

Management:

- **Education:**

• Compliance 50% • Inhalers techniques especially elderly • Asthma Action plans.

Specific directions for daily management and for adjusting medications in response to increasing symptoms or decreasing PEFR .

- **Control of environmental factors:**

• **Triggers** (Aeroallergens, Irritants),

• **Comorbid conditions** (Obesity, GERD, Rhinitis, ABPA, VCD, stress),

• **Medications** (Aspirin, Beta Blockers),

• **Infections** (Vaccinations).

- **Pharmacological:**

- Here ask yourself, what is my aim? How to assess it ? How can I achieve it?

1. Aim[22]:

The aim of asthma management is control of the disease. **Complete control is defined as:**
- **no** daytime symptoms, **no** night time awakening due to asthma, - **no** need for rescue medication, **no** asthma attacks, - **no** limitations on activity including exercise, - **normal** lung function (in practical terms EV, and/or PEF >80% predicted or best), - **minimal** side effects from medication.

2. Assessment: "Important".

- **Assessment of risk factors for poor asthma outcomes & Approach of management:**

Independent risk factors include:
• Ever intubated for asthma • Uncontrolled asthma symptoms • Having ≥1 exacerbation in last 12 months • Low FEV1 (measure lung function at start of treatment, at 3-6 months to assess personal best, and periodically thereafter) • Incorrect inhaler technique and/or poor adherence • Smoking • Elevated FeNO in adults with allergic asthma • Obesity, pregnancy, blood eosinophilia

*independent to the level of symptom control, GINA 2017

Approach of management:
1. Start treatment at the level most appropriate to initial severity. **2. Achieve early control.** **3. Maintain control by:** - increasing treatment as necessary - Decreasing treatment when control is good.
- Before initiating a new drug therapy practitioners should check adherence with existing therapies, check inhaler technique and eliminate trigger factors.

British guideline on the management of asthma Quick Reference Guide

3. Treatment :

Relievers:	Preventer:	Personalized Medicine:
– Short Acting Beta agonist	– **Steroids**[1] – Long acting Beta Agonist[2] and LAMA[2] – Leukotriene's receptors Antagonist – Theophylline	- Anti IgE or Anti IL5

1-for long-term management. 2- if asthma NOT controlled combine it with steroids

[22] **British guideline on the management of asthma** Quick Reference Guide.

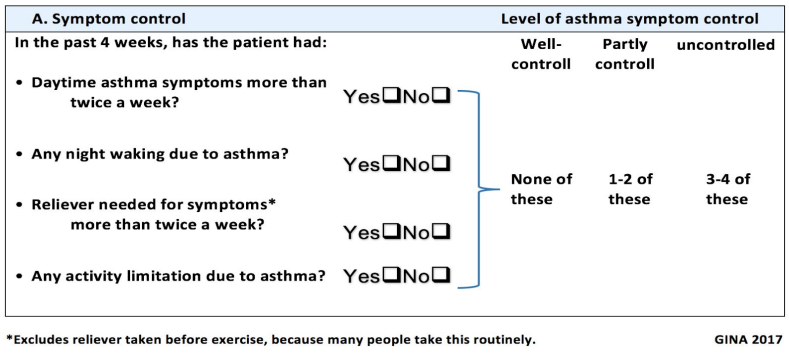
Asthma is managed in a stepwise fashion of progressively adding more types of treatment if there is no response.

Step 1	**Always start the treatment of asthma with an inhaled short-acting beta agonist (SABA) as needed.** **Examples of SABA are:** · Albuterol · Pirbuterol · Levalbuterol
Step 2	**Add a long-term control agent to a SABA. Low-dose inhaled corticosteroids (ICS) are the best initial long-term control agent.** ● **Example of ICS are:** · Beclomethasone,budesonide, flunisolide, fluticasone,mometasone, triamcinolone ● **Alternate long-term control agents include:** · Cromolyn and nedocromil to inhibit mast cell mediator release and eosinophil recruitment · Theophylline · Leukotriene modifiers : montelukast,zafirlukast,or zileuton(best with atopic patients)
Step 3	**Add a long-acting beta agonist (LABA) to a SABA and ICS, or increase the dose of the ICS.** **LABA medications are :** salmeterol vs formoterol.
Step 4	**Increase the dose of the ICS to maximum in addition to the LABA and SABA.**
Step 5	**Omalizumab may be added to the SABA, LABA, and ICS in those who have an increased IgE level.**
Step 6	**Oral corticosteroids such as *prednisone* are added when all the other therapies are not sufcient to control symptoms.**
Notes	- *Adverse effects of inhaled steroids are dysphonia and oral candidiasis.* - *High-dose inhaled steroids rarely lead to the adverse effects associated with prednisone.* - *Never use LABA first or alone!* - *Anticholinergics role of ipratropium and tiotropium in asthma management is not clear. Anticholinergic agents will dilate bronchi and decrease secretions. They are very effective in COPD.*

4. Monitoring:

• Symptoms, • Peak Flow (Home), • **Spirometry (Clinic),** • Novel FENO and Sputum eosinophils, • Assess Severity and Control of asthma.

- **GINA assessment of symptoms control:** dr likes it



A. Symptom control		Level of asthma symptom control		
In the past 4 weeks, has the patient had:		Well-controll	Partly controll	uncontrolled
• Daytime asthma symptoms more than twice a week?	Yes❑No❑	None of these	1-2 of these	3-4 of these
• Any night waking due to asthma?	Yes❑No❑			
• Reliever needed for symptoms* more than twice a week?	Yes❑No❑			
• Any activity limitation due to asthma?	Yes❑No❑			

*Excludes reliever taken before exercise, because many people take this routinely. GINA 2017

Objective 5: Follow prevention measure

- **Asthma Self Management:**

- Communicate and educate the patient with **a written asthma action plan** includes all the information you need to look after your asthma well, so you will fewer symptoms and significantly cut your risk of asthma attacks.
- Asthma is diagnosed on history (recurrent episodes of coughing, wheezing, breathlessness, and chest tightness often associated with allergic triggers), examination (wheeze) and lung function.

Acute severe asthma (Kumar & master the boards)

This is severe progressive asthmatic symptoms over a number of hours or days. It is a medical emergency that must be recognized and treated immediately at home with subsequent transfer to hospital. In the UK, 1400 patients die from asthma each year and 90% of these deaths are preventable by correct management.

Clinical features

Patients with acute severe asthma typically have:

- Inability to complete a sentence in one breath
- Respiratory rate ≥ 25 breaths/min
- Heart rate ≥ 110 beats/min
- PEFR 33–50% of predicted normal or patient's best.

Life-threatening features are any one of the following in a patient with acute severe asthma:

- Silent chest, cyanosis or feeble respiratory effort
- Exhaustion, altered conscious level
- Bradycardia or hypotension
- PEFR < 33% of predicted or best
- PaO2 < 8 kPa.

Treatment Oxygen | Albuterol | Steroids

- The best initial therapy is **oxygen combined with inhaled short-acting beta agonists such as albuterol and a bolus of steroids**. Corticosteroids need 4 to 6 hours to begin to work, so give them right away.
- Epinephrine injections are no more effective than albuterol and have more adverse systemic effects.
- Ipratropium should be used, but does not work as rapidly as albuterol.
- Magnesium has some modest effect in bronchodilation. Magnesium is not as effective as albuterol, ipratropium, or steroids, but it does help.
- If the patient does not respond to oxygen, albuterol, and steroids or develops respiratory acidosis (increased pCO2), the patient may have to undergo endotracheal intubation for mechanical ventilation. These patients should be placed in the intensive care unit.

Summary : Management of Asthma

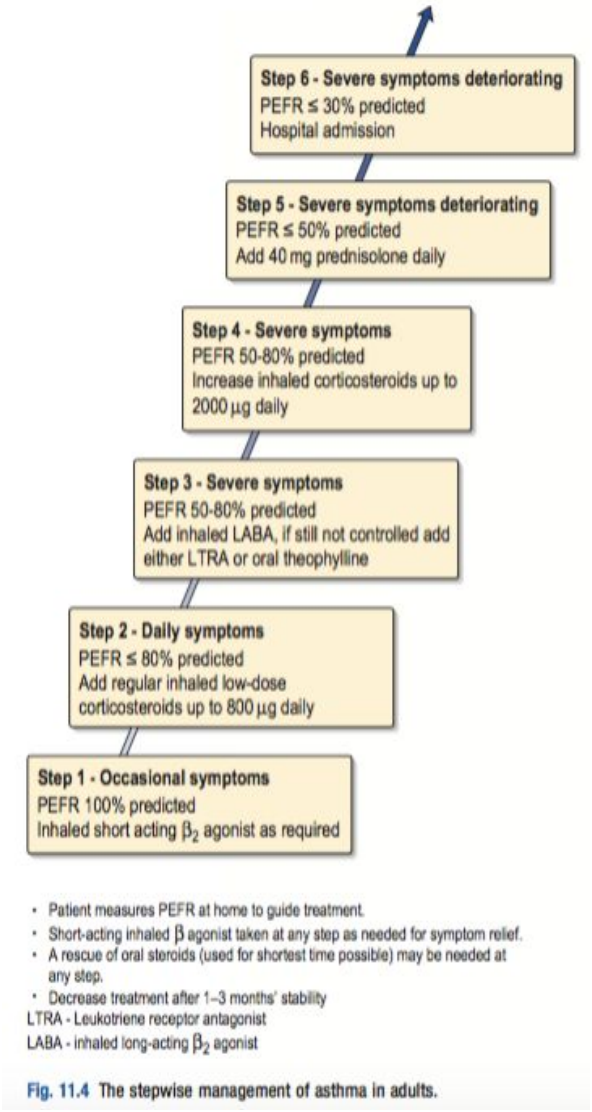


Fig. 11.4 The stepwise management of asthma in adults.

Emergency Box 11.1

Management of acute severe asthma in hospital

Initial treatment

- Oxygen therapy to maintain oxygen saturation (SpO_2) 94–98%.
- Nebulized salbutamol 5 mg or terbutaline 10 mg with oxygen as the driving gas.
- Hydrocortisone 200 mg intravenously.
- Antibiotics if evidence of infection: focal shadowing on the chest X-ray, purulent sputum.
- Fluids, aim for 2.5–3 L/day, intravenously if necessary.

Investigations

- Chest X-ray to exclude pneumothorax or pneumonia.
- Pulse oximetry (continuous).
- Arterial blood gases if SpO_2 < 92%. May need repeat depending on response.
- PEFR before and after initial treatment.
- Urea and electrolytes – steroids and salbutamol may result in hypokalaemia.

If improved – continue

Oxygen therapy.

Prednisolone oral, 40–50 mg for 7 days.

Nebulized β_2-agonist 4-hourly

After 24 hours

Add in high-dose inhaled corticosteroid

Change nebulized to inhaled β_2-agonist

Discharge from hospital when:

- Free of SOB or wheeze
- PEFR > 75% predicted & diurnal variability < 25%
- Stable on discharge treatment for 24 h

Before discharge: check inhaler technique, determine reason for exacerbation and issue a written asthma plan discussed with patient.

Life-threatening features present or poor response to treatment

Oxygen therapy.

Hydrocortisone 200 mg i.v. 4-hourly.

Nebulized β_2-agonist every 10–20 min.

Add nebulized ipratropium bromide 0.5 mg 4-6-hourly.

Magnesium sulphate 1.2–2 g i.v. over 20 min.

If no improvement give:

Salbutamol 3–20 µg/min (5 mg salbutamol in 500 mL 0.9% saline or 5% dextrose, infuse at 0.3–2 mL/min)

Inform ITU of possible admission for intubation and mechanical ventilation.

The following are not effective in acute exacerbations:

- Theophylline
- Cromolyn and nedocromil (best with extrinsic allergies like hay fever)
- Leukotriene modifiers
- Omalizumab
- Salmeterol

Cases

1) **A 47-year-old man with a history of asthma comes to the emergency department with several days of increasing shortness of breath, cough, and sputum production. On physical examination his respiratory rate is 34 per minute. He has diffuse expiratory wheezing and a prolonged expiratory phase.**
Which of the following would you use as the best indication of the severity of his asthma?
a. Respiratory rate
b. Use of accessory muscles
c. Pulse oximetry
d. Pulmonary function testing
e. Pulse rate

2) A 30-year-old athlete presents to your office complaining of intermittent wheezing. This wheezing begins shortly after running. The patient admits to smoking 1 to 2 packs of cigarettes per day for 5 years. What finding would be consistent with asthma?
a. Hyperinflation on chest x-ray
b. Improvement in FEV1 after bronchodilator
c. Low oxygen saturation on finger oximetry
d. Decreased FVC on PFT testing
e. Dyspnea on assuming a supine position

3) A 68-year-old woman with a prior diagnosis of asthma presents to your clinic for a routine clinic visit. She complains of occasional palpitations and tremor. Her dyspnea is well controlled. Her past medical history is remarkable for hospitalization for mild congestive heart failure 2 months ago; she notes occasional postprandial acid reflux. Her medications include lisinopril, digoxin, furosemide, an intermittent short-acting inhaled beta agonist, and theophylline. She uses an over-the-counter pill (whose name she cannot remember) for the reflux symptoms. On examination her heart rate is 112 beats/minute. S1 and S2 are normal; she has a mild tremor of the outstretched hands. What is the best next step in her management?
a. Chest x-ray to rule out exacerbation of congestive heart failure
b. Theophylline level
c. Spirometry before and after bronchodilator
d. Intermittent lorazepam 0.5 mg po tid
e. Discontinue beta agonist and substitute inhaled ipratropium

4) A patient with known asthma undergoing therapy with inhaled corticosteroid and intermittent (short-acting) β 2-agonist presents with complaints of nocturnal awakenings secondary to cough and occasional wheezing. This episode occurs three to four times per week. Pulmonary function tests in the past have shown mild obstructive lung disease. Which of the following is the best next step?
A. Oral steroids
B. Leukotriene inhibitors
C. Long-acting β 2-agonists
D. Theophylline
E. Antireflux therapy

5) An obese 50-year-old man with a history of asthma returns with complaints of occasional dyspepsia and nocturnal cough. He wakes up in the morning with a sour taste in his mouth. His current medications include inhaled corticosteroid and a short-acting β 2-agonist. Which of the following should be your next step?

A. 24-Hour esophageal pH monitoring
B. Chest radiograph
C. Initiation of omeprazole
D. Short course of oral corticosteroids
E. Initiation of allergy desensitization

Answers

1) A. normal respiratory rate is 10 to 16 per minute. By itself, a respiratory rate of 34 indicates severe shortness of breath. Accessory muscle use is hard to assess and is subjective. Pulse oximetry will not show hypoxia until the patient is nearly at the point of imminent respiratory failure. Oxygen saturation can be maintained above 90% by hyperventilating. Pulmonary function testing cannot be done when a patient is acutely short of breath.

2) B. Asthma is an inflammatory process with reversible airflow obstruction. This patient's presentation suggests exercise-induced asthma. Asthma is an incompletely understood disease that involves the lower airways and results in bronchoconstriction and excess production of mucus. This, in turn, leads to increased airway resistance and occasionally respiratory failure and death. In any obstructive lung disease such as chronic obstructive pulmonary disease, hyperinflation may be present on chest x-ray and FEV1 may be decreased. Only in asthma is the airway obstruction fully reversible. Hypoxia would be unusual in exercise-induced asthma and would suggest an alternative diagnosis. Reduced forced vital capacity (FVC) characterizes restrictive lung disease, not obstructive (airways) disease. Dyspnea on assuming a supine position would suggest congestive heart failure.

3) B. Theophylline has been used as a bronchodilator for a number of years. It has been less commonly used in recent years owing to its narrow therapeutic window. The drug is metabolized in the liver. A drug or process that interferes with the activity the cytochrome P450 system will slow the metabolism of theophylline and may lead to the accumulation of toxic levels in the blood. The metabolism of theophylline is slowed by age, infection, CHF (resulting from decreased hepatic blood flow), and a number of drugs. Commonly used drugs that impair the metabolism of theophylline include cimetidine, erythromycin, ciprofloxacin, allopurinol, and zafirlukast. This patient has probably been using over-the-counter cimetidine to treat her reflux symptoms. Stopping theophylline until the drug level has returned will relieve her palpitations and tremor. In the absence of dyspnea, wheezing, or clinical signs of CHF, chest film and spirometry would not be helpful. Using a benzodiazepine to treat her tremor would leave a potentially serious theophylline toxicity undetected. Finally beta agonists are more effective bronchodilators in asthma than is ipratropium; the tremulousness associated with beta agonist use is usually short lived.

4) C. Long-acting β 2-agonists are helpful in this situation. The asthma would be classified as moderate persistent, and the recommended treatment is long-acting β 2-agonists, such as salmeterol, which are particularly helpful with nocturnal symptoms.

5) C. Initiation of omeprazole, a proton pump inhibitor, dyspepsia and the sour taste suggest GERD. Other recommendations include dietary modifications and weight reduction. Twenty-four–hour esophageal pH monitoring is indicated only if the medications did not help. Oral corticosteroids could be a consideration if the clinical scenario is more consistent with asthma exacerbation.