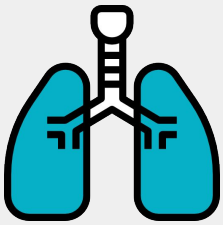
Bronchial Asthma



Objectives :

- ★ Definition
- ★ Epidemiology
- ★ Pathophysiology
- ★ Types
- ★ Diagnosis
- ★ Management

Editing file

Color index

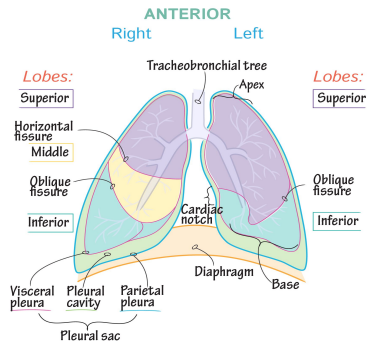
Original text
Females slides
Males slides
Doctor's notes [438]
Doctor's notes [439]
Text book
Important
Golden notes
Extra

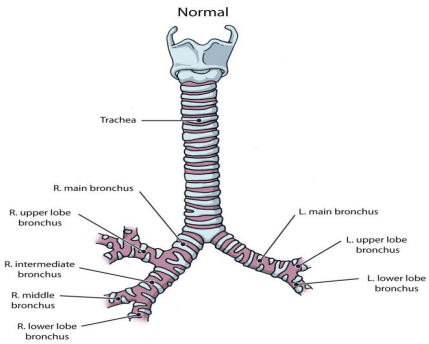
Review of the basics (extra)

Special thanks to our amazing anatomy & physiology teamwork in respiratory block!!!

◀ Anatomy of the lungs

	Right lung	Left lung
Fissures	**2** fissures (horizontal and oblique)	**One** fissure (oblique)
Lobes	**3** lobes (Superior, middle, inferior)	**2** lobes (Superior, inferior)
Secondary bronchi	**3** secondary bronchi (R. Upper bronchus it branches before entering the hilum, R. Intermediate bronchus it enters to the hilum to give the two remaining bronchi , R. Middle bronchus, R. Lower bronchus)	**2** secondary bronchi (L. Upper bronchi & L. Lower bronchi)
Comments	larger and shorter than the left lung	it has cardiac notch at lower part of its cardiac notch





◀ Pulmonary function tests

Forced Expiratory Curve

The subject takes a maximal inspiration and then exhales as rapidly, as forcibly as maximally as possible in short time approximately 3-5 seconds.

- A plot of volume against time.

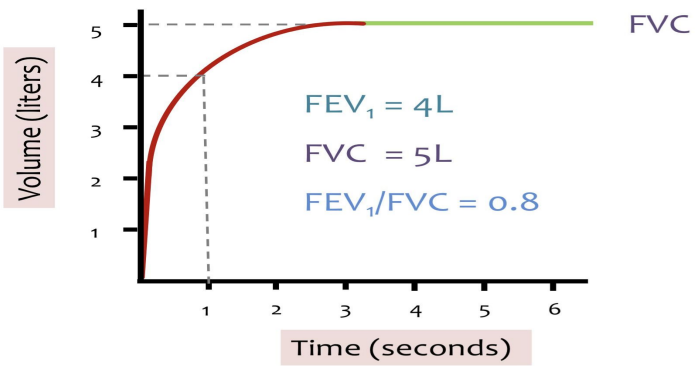
FEV1 (forced expiratory volume): Volume of air expelled in the 1st sec of forced expiration starting from full inspiration

FVC (Forced Vital Capacity):

The max volume of air that can be forcibly and rapidly exhaled following a max inspiration.

- **FEV1 % or ratio (FEV1/FVC) * 100**

Fraction of the VC expired during the 1st sec of a forced expiration (NL 70%-80%) - FEV1 is a useful measure of how quickly the lungs can be emptied. - ➔ The ratio is a useful index of airflow limitation.



Flow Volume Loop

This measures expiration & inspiration flow as a function of exhaled volume rather than against time.

Measurements on flow V loop:

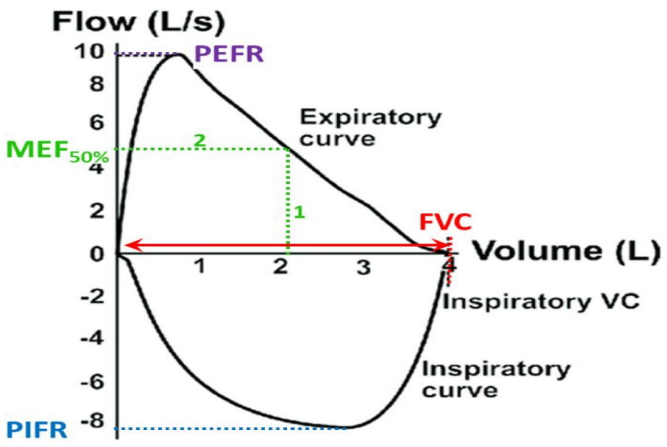
- **PEFR** (Peak Expiratory Flow Rate): Greatest flow achieved during forceful expiratory effort

➔ =6- 12 L/sec

- **PIFR** (Peak Inspiratory Flow Rate): max flow speed achieved during forceful inspiratory effort

➔ =6-12 L/sec

- **MEF 50**: max expiratory flow at 50% of FVC
- **FVC** = 4-6 L it can be measured over the X-axis (not like Forced Expiratory Curve)



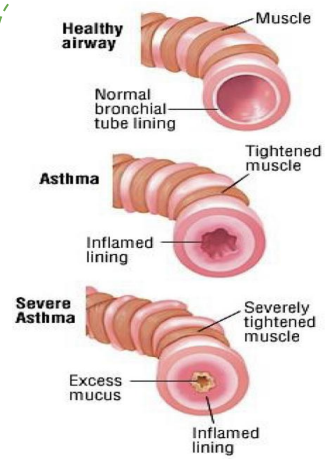
Introduction

Definition

- Asthma is a chronic inflammatory disorder of the airways in which many cells play a role: in particular, **mast cells, eosinophils**, neutrophils, **T-lymphocytes** T lymphocyte will determine what type of inflammation you will have, macrophages and epithelial cells Treatment targets eosinophils and T lymphocytes

General characteristics

- In susceptible individuals (genetic predisposition), this inflammation causes **recurrent episodes**[1] of **coughing, wheezing, breathlessness,** and chest tightness**.**
- These episodes are usually associated with widespread but **variable airflow obstruction** (airway hyper-responsiveness) that is often **reversible**[2] either spontaneously or with treatment.
- **Classically, asthma has three characteristics:**
  - **airflow limitation**, which is usually reversible spontaneously or with treatment
  - **airway hyper-responsiveness** to a wide range of stimuli (see later)
  - **bronchial inflammation** with T lymphocytes, mast cells, eosinophils with associated plasma exudation, oedema, smooth muscle hypertrophy, matrix deposition, mucus plugging and epithelial damage.
- **In chronic asthma**, inflammation may be accompanied by **irreversible** airflow limitation as a result of airway wall remodelling, which may involve large and small airways and mucus impaction.



For years our concept of asthma was focused on relaxing bronchial smooth muscle (relieving airway obstruction) by giving bronchodilators without dealing with the inflammation, this changed in the past few years.
-The outer diameter of the airways remain the same, it's the diameter of the lumen inside that's severely narrowed.
-Presence of secretions cause increase airway resistance.

Asthma triggers

- Triggers include **pollens, house dust**, molds, cockroaches, **cats**, **dogs**, cold air, viral infections, tobacco smoke, **medications (β-blockers, aspirin)** and exercise
- Symptoms have variable severity and may not be present simultaneously.
- Usually occur within 30 minutes of exposure to triggers.

Epidemiology

- Could occur at any age[3], but 75% are Dx at age <7
- Remission around puberty
- Prevalence on the rise. likely Multifactorial
- Wide geographical variation (4-25%)
- Females 40% higher prevalence[4]
- Severe asthma 10 % but morbidity / costs. Most of the money spent on asthma is spent on those with severe asthma

Saudi Arabia Figures

- Asthma affects >2 million Saudis may be up to 6 million (20% of the population) are undiagnosed
- Asthma control: 5% were controlled, 31% partially controlled, **64% uncontrolled.**

Etiology

Although asthma is **multifactorial** in origin, **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- Symptoms tend to be intermittent, worse at night and in the early morning and provoked by triggers .
The term "episodic" refers to the fact that an asthmatic patient can be healthy one day and sick the next (or ill after three months). If someone feels out of breath on a daily basis (it is mostly not asthma)
2- This is the main difference between asthma and COPD.
3-The majority of patients develop symptoms during childhood, but the symptoms fade or disappear around puberty. However, symptoms can reappear later in life.
4-Higher female prevalence could be because ladies are more likely to tell you about their symptoms more than men

Pathogenesis

Pathogenesis of asthma
(It has acute and chronic phases)

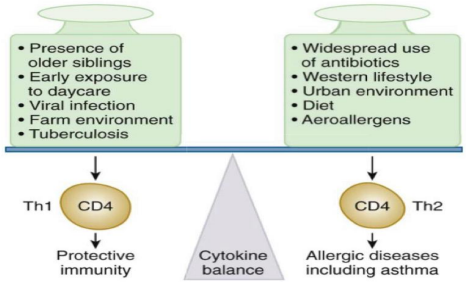
- Tightening of the airways
- Airway remodeling
- Thick mucus production

What causes asthma?

1. **Hygiene hypothesis**
2. **Atopy** allergy [1]
3. **Genetics** [4]
4. **Smoking** [2] (controversial)
5. **Obesity** (New under investigations)[3]

Hygiene hypothesis

- Suggests that growing up in a relatively 'clean' environment may predispose towards an IgE response to allergens (Th-2). Conversely, growing up in a 'dirtier' environment may allow the immune system to avoid developing allergic responses (Th-1/ protective immunity).

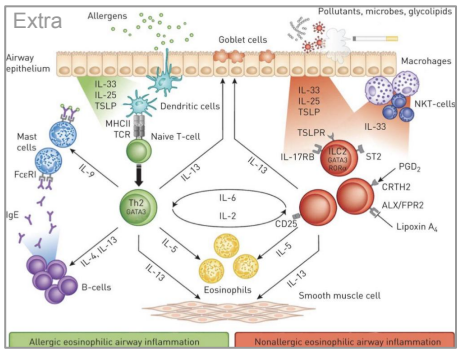


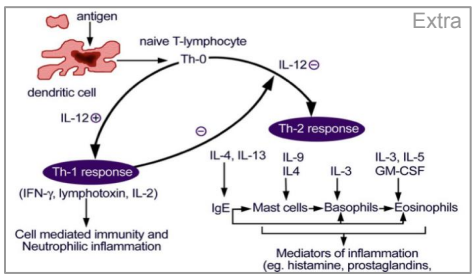
The role of inflammatory cells in asthma

1. The antigen will be presented to the naïve T-lymphocyte by the dendritic cell
2. Then the response will be either Th1 or Th2 response

A. **Th2** response: These lymphocytes, when stimulated by the appropriate antigen, release a restricted panel of cytokines **(IL-4, IL-5, and IL-13)** which play a part in the migration and activation of mast cells and eosinophils. In addition, production of IL-4 and IL-13 helps maintain the proallergic Th2 phenotype, favouring switching of antibody production by B lymphocytes to IgE. These IgE molecules attach to mast cells via high-affinity receptors which in turn release a number of powerful mediators acting on smooth muscle and small blood vessels, such as histamine and prostaglandins.

B. **Th1** response: cell mediated immunity, neutrophilic inflammation



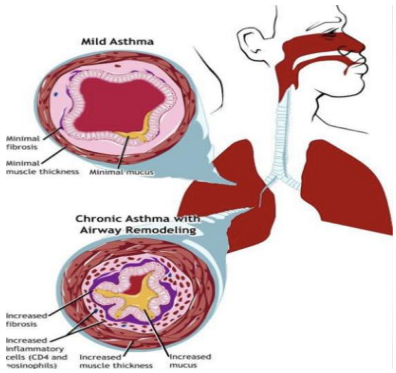


1-Atopic triad (Asthma, allergic rhinitis, atopic dermatitis "eczema").
2-It is Controversial, but we know that anybody with asthma and smokes their condition will worsen and end up with more inflammation
3-The relationship between obesity and asthma is not clear, although losing weight in obese asthmatics tends to improve their symptoms. People who are obese suffer from shortness of breath because their lungs have to work harder.
If we perform a bronchial provocation test (give the patient a smth to inhale) on an obese patient, it will produce bronchospasm if the patient has asthma. However, if the patient loses weight and retakes the test, the results will be normal.
4- It's really important to ask about family history

Pathogenesis cont'

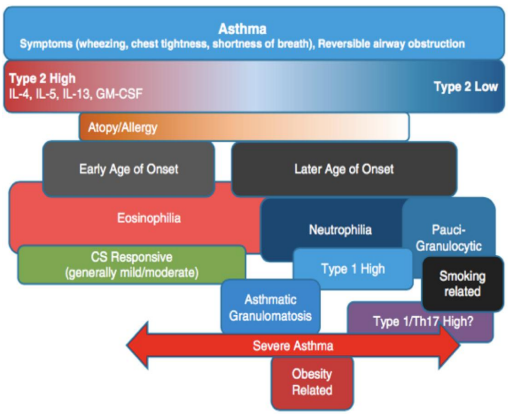
◀ Airway in asthmatic patients

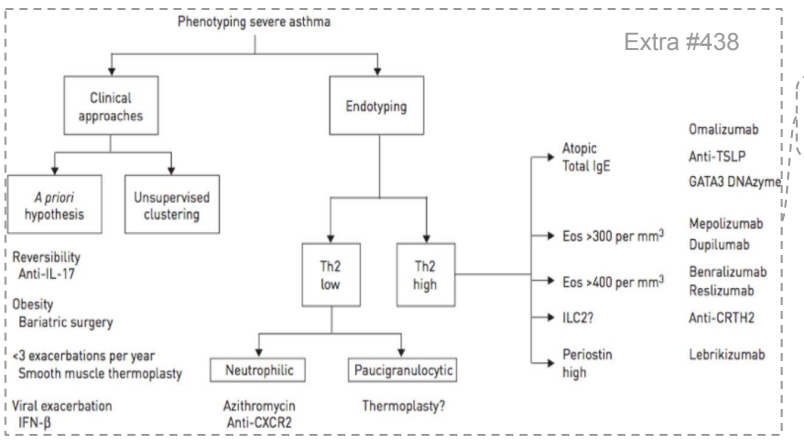
- Initially, there is minimal fibrosis and minimal secretions and no muscle hypertrophy, so no bronchoconstriction. But as the inflammation goes on and becomes a chronic process, this leads to to increased fibrosis, hyperplasia of mucous cells and smooth muscle cells, leading to hypersecretion and an increase in muscle thickness, which increases the obstruction of the airways.
- Even if we relax the smooth muscle (relieve bronchospasm), the patient will still be short of breath. To solve this symptom, we must treat the **bronchospasm**, **inflammation** and get rid of the mucus.



◀ Asthma types

	Early onset (<12years)	Late onset (>12years)
Natural history	**Childhood-onset** asthma a relatively homogeneous group	**Adult-onset** asthmatics are a very mixed group (Heterogeneous) e.g, AERD(aspirin exacerbated respiratory disease) . It's also related to smoking Late onset eosinophilic asthma
Atopy	**Very high (Allergic asthma)**	- **Atopic 34%** (have less severe disease. Those with severe disease are less likely to be atopic) - **Non-atopic 52%** (have mild-to-moderate persistent asthma)
Family history	**High**	Low
Eosinophilia	✓	-
Neutrophilia & paucigranulocytic cells	-	✓





- **What is Phenotyping?** Phenotyping means classifying asthmatics based on the characteristics you can observe by looking and taking history:
- young or old, history of allergies, smoker or not, obese or thin...
- **What is endotyping?** endotyping means the things you can measure by doing tests, biochemicals, genetics, etc. In classifying asthma, we focus more on endotyping because this will allow us to use specific treatments. For example, Anti IgE: Omalizumab, is used for atopic asthma. And Anti IL-5: Mepolizumab, is used for eosinophilic asthma.
- Remember that eosinophilic asthma (high eos) could be allergic "atopic", which means having high IgE, and it also could be non-allergic.
- Another thing we can measure in endotyping is exhaled nitric oxide from breath, which is a marker of eosinophilic inflammation, high levels of exhaled NO predicts an upcoming exacerbation even if symptoms are not present yet.

Diagnosis

History: Initial structured clinical assessment

The predictive value of individual symptoms or signs is poor, and a structured clinical assessment including all information available from the history, examination and historical records should be undertaken.

Factors to consider in an initial structured clinical assessment include:

- **Episodic symptoms**

More than one of the symptoms of **wheeze**, breathlessness, **chest tightness** and **cough** occurring in episodes with periods of no (or minimal) symptoms between episodes. Note that this excludes cough as an isolated symptom in children.

For example:

• a documented history of acute attacks of wheeze, with symptomatic and objective improvement with treatment

• recurrent intermittent episodes of symptoms **triggered** by **allergen exposure** as well as **viral infections** and exacerbated by **exercise** and **cold air**, and emotion or laughter in children

• in adults, symptoms triggered by taking non-steroidal anti-inflammatory medication or beta blockers.

An historical record of significantly lower FEV, or PEF during symptomatic episodes compared to asymptomatic periods provides objective confirmation of obstructive nature of the episodic symptoms.

- **Wheeze confirmed by a healthcare professional on auscultation**

• It is important to distinguish wheezing from other respiratory noises, such as stridor or rattly breathing.

• Repeatedly normal examination of chest when symptomatic reduces the probability of asthma.

- **Evidence of diurnal variability**

Symptoms which are worse at night or in the early morning.

Atopic history

★ Personal history of an atopic disorder (ie, eczema or allergic rhinitis) or a family history of asthma and/ or atopic disorders, potentially corroborated by a previous record of raised allergen-specific IgE levels, positive skin-prick tests to aeroallergens or blood eosinophilia.

Absence of symptoms, signs or clinical history to suggest alternative diagnoses (including but not limited to COPD, dysfunctional breathing, obesity).

If all 4 of these features (highlighted in table) are present, you are almost 100% sure of asthma diagnosis. As long as there is nothing in the history or clinical findings that would suggest an alternative diagnosis

Start treating for asthma and look for improvement. If improvement is there then the diagnosis is confirmed.

Diagnosis

Clinical features that increase 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** [1]
  - symptoms in response to exercise, allergen exposure and cold air [2]
  - symptoms after taking **aspirin** or **beta blockers** (will be discussed later)
- **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**
- Nasal polypectomy, increased accessory muscles use are also common associations, especially after nasal polyps removal patient would be introduced to aspirin which could precipitate asthma

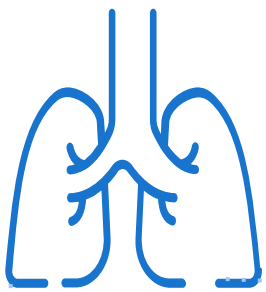
Clinical features that decreases the probability of Asthma

- Prominent dizziness, light-headedness, peripheral tingling (These signs indicate hyperventilation like panic attacks)
- Chronic productive cough in the absence of wheeze or breathlessness
- Repeatedly normal physical examination of chest when symptomatic
- Voice disturbance (most likely to be vocal cord dysfunction)
- Symptoms with colds only
- Significant smoking history (ie > 20 pack-years) (This more likely COPD)
- Cardiac disease (Patients with HF may have a wheeze)
- Normal PEF or spirometry when symptomatic[3]

Differential Diagnosis

Other Illness with wheezing / SOB

- COPD (Smoker) - inflamed airways may be narrowed, or bronchospasm may be present
- Heart failure - due to edema of airways and congestion of bronchial mucosa
- Airway obstruction (Tumors, Foreign body) [4]
- Vocal cord dysfunction



May Coexist and complicate Dx of asthma

- GERD gastric contests irritate the larynx which will cause coughing, and if it gets into the airways it will cause coughing, wheezing, SOB and makes the asthma worse.
- OSA (Obstructive sleep apnea)
- ABPA (Allergic bronchopulmonary aspergillosis)

1-Remember the circadian levels of cortisol? Cortisol is anti-inflammatory and its levels rise in the morning but become low at night and that's why symptoms are worse at night.
2-Cold air triggers symptoms because it dries the airways and this induces bronchoconstriction.
3- BE CAREFUL: Athletes and people who are very fit. Can have normal values during an asthma attack. That's because their baseline is higher than normal. Be aware of the starting point before saying its abnormal
4-In airway obstruction due to tumors or foreign bodies a monophonic wheeze is heard on auscultation as the is usually on obstruction of one airway. In asthma however, the wheeze is polyphonic as there are many small airways being obstructed.

Diagnosis

◀ Examination

1- Upper respiratory tract (nasal secretion, mucosal swelling, nasal polyp)

2- Chest (**Wheezing** or prolonged phase of forced exhalation, Chest hyper-expansion, accessory muscles

3- Wheezing— high-pitched whistling sounds when breathing out
A lack of wheezing and a normal chest examination do not exclude asthma why? Because asthma is episodic, when they're well you won't hear wheezing.

4- Skin (atopic dermatitis, eczema) (This indicates allergy)

Tests

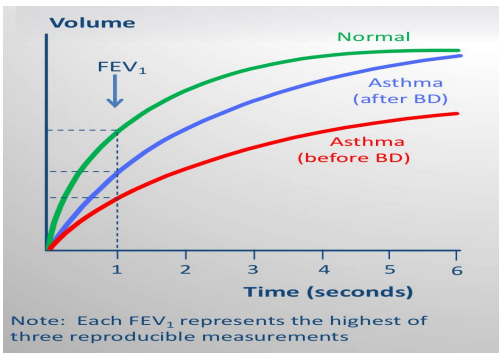
★ **Spirometry** – Routine
(the initial diagnostic test)
Spirometry, confirms if there is an airway limitation . But remember, if you're asthmatic and you do not have any current symptoms, the spirometry will be normal. Therefore, perform Bronchoprovocation test

Usually if **alternate Dx considered :**

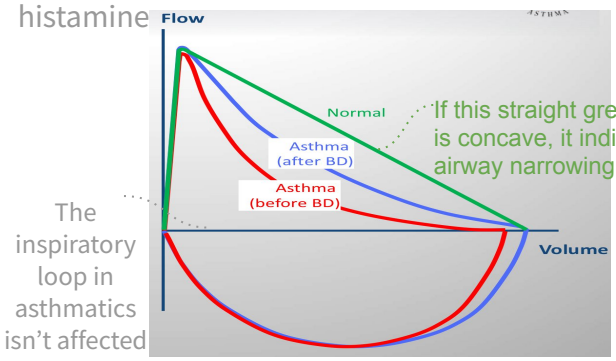
- Full Lung Functions FLF: It measures airway muscle strength "respiratory muscles" (if they are weak due to other diseases -> pt is breathless) , and measures how easily gases diffuse from lungs to blood. In asthma, both measures will be normal
- FBC (full blood count) to check for eosinophilia
- ABGs -should be considered if the patient is in significant respiratory distress. [2] acute exacerbations of asthma
- **Airway Hyper-responsiveness tests** or Bronchoprovocation test. (If spiro normal) [1]
- CXR / CT Chest - Normal in mild to moderate cases; severe asthma reveals hyperinflation. Only necessary in severe asthma to exclude other conditions (e.g, pneumonia, pneumothorax, pneumomediastinum ,foreignbody).

Asthma Dx - variable outflow limitation

- **Confirm presence of airflow limitation**
  – Document that FEV1/FVC is reduced **<0.75** (at least once)

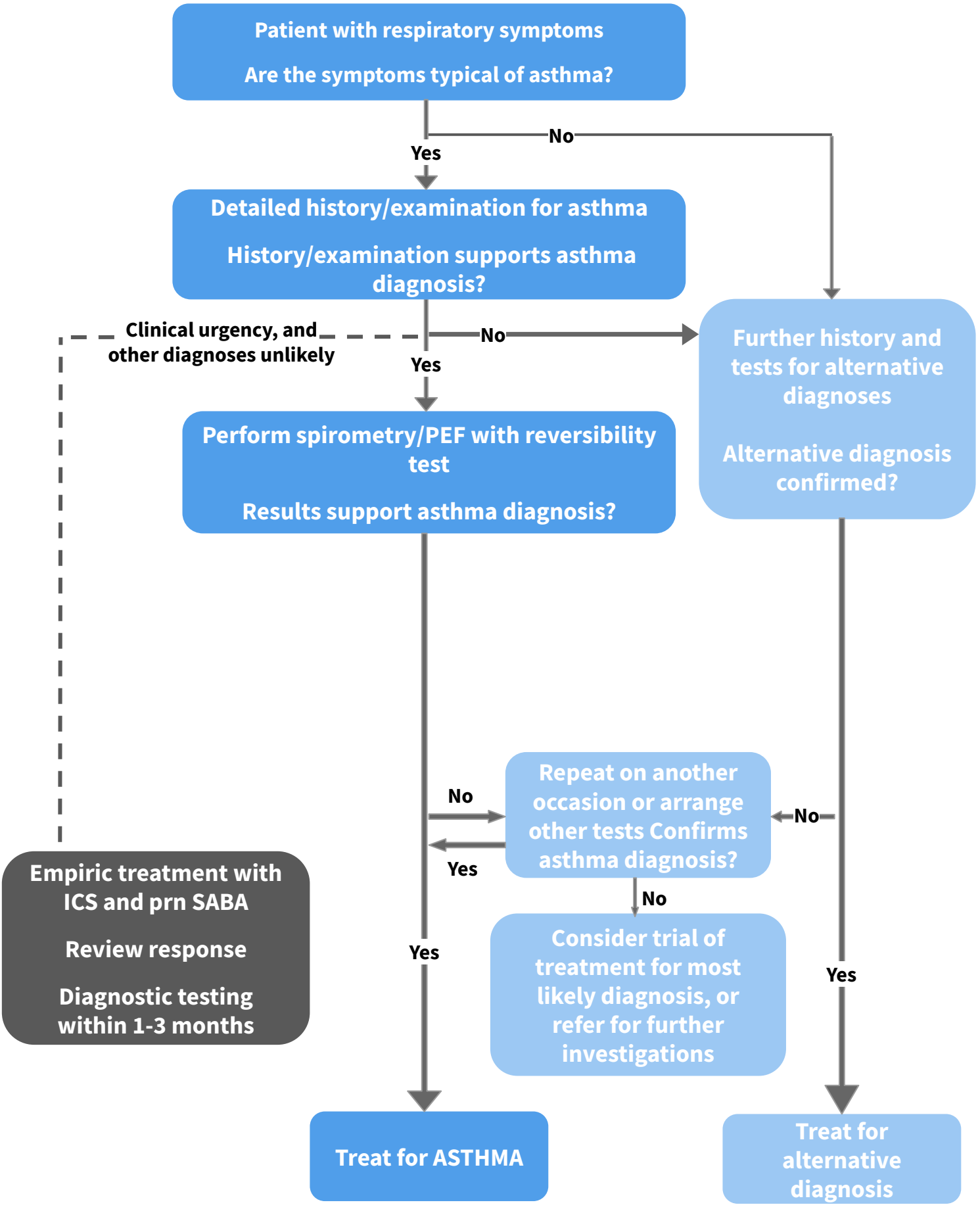


★**Confirm variation in lung function or Reversibility**
– Excessive bronchodilator reversibility
(FEV1 **>12%** and **>200mL**) need both values to be significant
– Excessive diurnal variability twice-daily PEF monitoring
– FEV <20% with the use of methacholine or histamine



1-It is where the patient is given a certain chemical to inhale (eg. histamine, methacholine) at different doses. If the patients airways go into constriction (bronchospasm) with low doses, this suggests asthma.
2- Hypocarbia is common. Hypoxemia may be present. Remember that patients with an asthma attack have an **increased respiratory rate, which should cause the PaCO2 to decrease**. **Increased PaCO2 is a sign of respiratory muscle fatigue or severe airway obstruction**, The patient should be hospitalized and mechanical ventilation considered. If the PaCO2 is normal or increased, respiratory failure may ensue.

Diagnostic Approach



Management

◀ Components of Asthma management [1]

01 Monitoring

- Symptoms
- Peak Flow (Home)
- Spirometry (Clinic)
- Novel FENO[2] and Sputum eosinophils
- Assess Severity and Control of asthma

02 Education [3]

- Compliance
- Inhalers techniques
- Asthma Action plans

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

03 Environmental Factors

- Triggers (Aeroallergens, Irritants)
- Co-morbid conditions (Obesity, GERD, Rhinitis, ABPA,VCD, stress)
- Medications (Aspirin, Beta Blockers)
- Infections (Vaccinations)

04 Pharmacologic Management

Aims

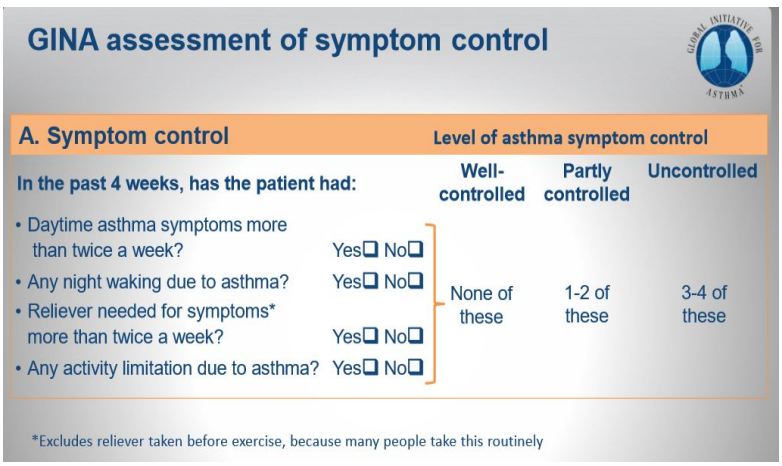
The aim of asthma management is control of the disease. Complete control is defined as:

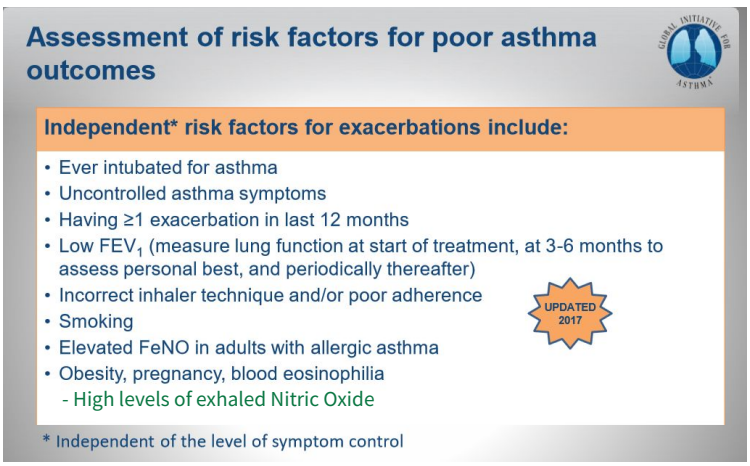
- no daytime symptoms
- no night time awakening due to asthma
- **no need for rescue medication**
- no asthma attacks
- no limitations on activity including exercise
- **normal lung function (in practical terms FEV, and/or PEF >80% predicted or best)**
- minimal side effects from medication[4].

Approach

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.



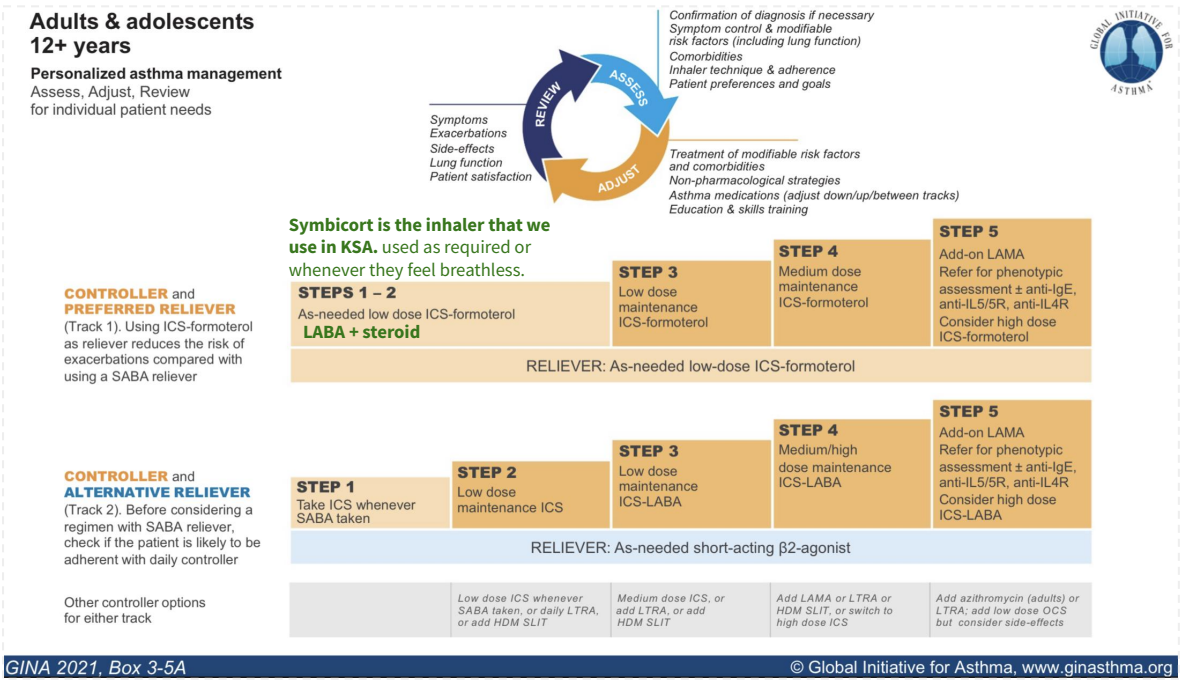


1-Without knowing how to manage, educate and control the patient's surroundings, the pharmacological treatment won't be effective.
2-If Exhaled Nitric oxide (FENO) levels are high this indicates that there is a lot of inflammation (allergic inflammation) going on in the lungs. A raised FENO in asymptomatic patients predicts that they will become symptomatic soon. The higher the levels of NO, the higher the inflammation, so if asthma is controlled it will become uncontrolled. It also tells if the pt is not compliant on their medications.
3-You have to **educate the patients** on how to use the inhaler. You should also give them plans on what to do when their symptoms get worse.
4-For ex. giving ↑doses of steroids for a year will cause: weight gain, skin problems, HTN, water retention, osteoporosis, being prone to infections, etc..

Pharmacological treatment

Relievers	Preventer	Personalized Medicine
• Short Acting Beta agonist	• Steroids • Long acting Beta Agonist and LAMA (Long acting muscarinic antagonists) e.g. tiotropium • Leukotriene's receptors Antagonist • Theophylline	• eg Anti IgE or Anti IL5

★ **2021 Guideline** Based on Dr: this is what we use now



Three years ago, the treatment for step 1 (a mild asthmatic patient with symptoms less than twice/month was ventolin (SABA), which was used as needed. However, this medication only addressed one issue: bronchospasm.
The treatment now includes treating both the bronchospasm and the inflammation, rather than only the bronchospasm.
So, whenever the patient uses SABA, they had to use their steroid inhaler as well. The problem was annoying as it requires the patient to carry two inhalers at the same time. Therefore they solved the problem by a combined inhaler (a bronchodilator and an anti inflammatory). However the combined inhalers that we have are LABA (fast acting) + steroids (no SABA + steroid) has been invented yet.

First of all we classify patient based on the severity of attacks they experience. Step 1 is the lowest and step 5 is the highest (worst).
Step 1-2:
give a combined inhaler (steroid such as budesonide and a bronchodilator LABA such as formoterol) the long acting beta agonist has a fast onset of action. This group mostly use their medications when they experience an exacerbation
Step 3:
for someone who needs to use it regulatory (usually twice a day)
Step 4:
if the symptoms are still uncontrolled we double the dose.
Step 5:
It is very imp to know whether the pt has a purely allergic asthma or not (based on phenotyping).
- People who are purely allergic: give injection(monoclonal antibodies) to block IgE every two weeks.
-Or give anti-eosinophilic medications such (anti-IL5 or anti-IL4). People who have high eosinophilia can allergic and non-allergic.

if you were asked in the exam : Is the ventolin inhaler (SABA) enough on its own? the answer is no

Start with high doses depending on level of symptoms, then after the symptoms are controlled narrow them down. (Titration)

SABA: Short acting beta2 agonists / **LABA**: Long acting beta2 agonists **(patients should not use LABA as monotherapy because it may be accompanied by an increased risk of life-threatening attacks or asthma death)** / **ICS**: Inhaled corticosteroids / low dose ICS (budesonide) and formoterol, called (bud-form)
-Side effects of inhaled corticosteroids are due to oropharyngeal deposition and include sore throat, oral candidiasis (thrush),and hoarseness. Using a spacer with MDIs (metered dose inhaler) and rinsing the mouth after use help minimize these side effects.
● Step 1: someone who gets his symptoms twice or less a month.
*e.g. Tiotropium, anti-IgE (for purely allergic asthma), anti-IL5/5R (For eosinophilic asthma), anti-IL4R (For eosinophilic asthma)

◀ Principles of asthma management

- Provide guided self-management education (self-monitoring written action plan regular review)
- Treat modifiable risk factors and comorbidities, eg. smoking, obesity, anxiety
- Advise about non-pharmacological therapies and strategies, eg physical activity, weight loss, avoidance of sensitizer where appropriate
- Consider stepping up uncontrolled symptoms, exacerbations or risks, but check diagnosis, inhaler technique and adherence first
- Consider adding SLIT (sublingual immunotherapy) in adult HDM-sensitive patients (house dust mite) with allergic rhinitis who have exacerbations despite ICS treatment, provided FEV1 is >70% predicted
- Consider stepping down if symptoms controlled for 3 months + low risk for exacerbations. Ceasing ICSs not advised.

◀ Asthma self management

- Communicate and educate patient
- A written asthma action plan includes all the information you need to look after your asthma well, so you'll have fewer symptoms and significantly cut your risk of an asthma attack.

Example of Asthma action plan:

Every day asthma care:

My personal best peak flow is: ____

My preventer inhaler (insert name/colour): ____

I need to take my preventer inhaler every day even when I feel well

I take ____ puff(s) in the morning and ____ puff(s) at night.

My reliever inhaler (insert name/colour): ____

I take my reliever inhaler only if I need to

I take ____ puff(s) of my reliever inhaler if any of these things happen:

- I'm wheezing
- My chest feels tight
- I'm finding it hard to breathe
- I'm coughing.

Other medicines I take for my asthma every day:

With this daily routine I should expect/aim to have no symptoms. If I haven't had any symptoms or needed my reliever inhaler for at least 12 weeks, ask my GP or asthma nurse to review my medicines in case they can reduce the dose.

People with allergies need to be extra careful as attacks can be more severe.

When I feel worse:

- My symptoms are coming back (wheeze, tightness in my chest, feeling breathless, cough)
- I am waking up at night
- My symptoms are interfering with my usual day-to-day activities (eg at work, exercising)
- I am using my reliever inhaler ____ times a week or more
- My peak flow drops to below ____

This is what I can do straight away to get on top of my asthma:

1 If I haven't been using my preventer inhaler, start using it regularly again or:

Increase my preventer inhaler dose to ____ puffs ____ times a day until my symptoms have gone and my peak flow is back to normal

Take my reliever inhaler as needed (up to ____ puffs every four hours)

If I don't improve within 48 hours make an urgent appointment to see my GP or asthma nurse.

2 If I have been given prednisolone tablets (steroid tablets) to keep at home:

Take ____ mg of prednisolone tablets (which is ____ x 5mg) immediately and again every morning for ____ days or until I am fully better.

URGENT! Call my GP or asthma nurse today and let them know I have started taking steroids and make an appointment to be seen within 24 hours.

In an asthma attack:

- My reliever inhaler is not helping or I need it more than every ____ hours
- I find it difficult to walk or talk
- I find it difficult to breathe
- I'm wheezing a lot or I have a very tight chest or I'm coughing a lot
- My peak flow is below ____

THIS IS AN EMERGENCY TAKE ACTION NOW

1. Sit up straight – don't lie down. Try to keep calm
2. Take one puff of my reliever inhaler every 30 to 60 seconds up to a maximum of 10 puffs
3. A) If I feel worse at any point while I'm using my inhaler → CALL 999 → Ambulance taking longer than 15 minutes? Repeat step 2
   B) If I don't feel any better after 10 puffs → CALL 999
   C) If I feel better: make an urgent same-day appointment with my GP or asthma nurse to get advice
   If I feel better, and have made my urgent same-day appointment:
   - Check if I've been given rescue prednisolone tablets
   - If I have these I should take them as prescribed by my doctor or asthma nurse

IMPORTANT! This asthma attack information is not designed for people who use the Symbicort® SMART regime OR Fostair® MART regime. If you use one of these speak to your GP or asthma nurse to get the correct asthma attack information.

Asthma

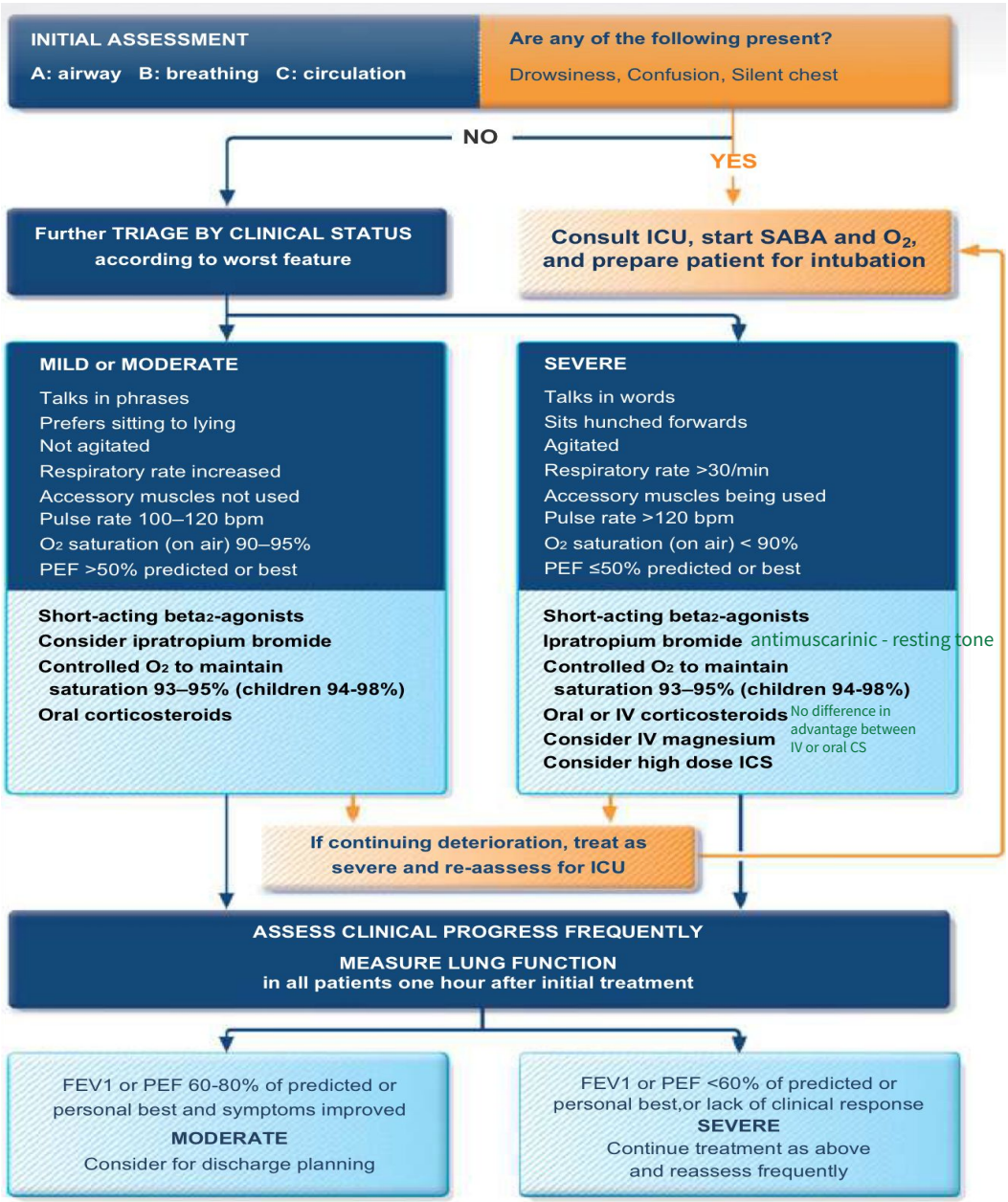
Avoid in asthmatics!!			
Beta- blockers	Even selective beta blockers may induce symptoms because their selectivity is actually relative, also watch out for beta blockers that are used as eye drops for glaucoma, they may trigger the symptom too. Beta blockers inhibit cAMP which will increase muscle tone.	**Aspirin**	How aspirin/NSAIDS make asthma worse? By inhibiting COX-1 and diverting to lipoxygenase pathway. This leads to a decrease in PGs and an increase in leukotrienes. **Aspirin Exacerbated Respiratory Disease (AERD)** **Definition:** a chronic condition characterized by Samter's triad; exacerbated by a **pseudoallergic** sensitivity reaction to aspirin and other NSAIDS (NSAID intolerance) **Clinical features:** Samter's triad (asthma, chronic/recurrent rhinosinusitis, nasal polyps) **Diagnosis:** NSAID/aspirin challenge test **Treatment:** avoid NSAIDS; aspirin desensitization

◀ Acute asthma attack - severity [1] Extra from 438

Level	Characteristics
Moderate asthma attacks	• Increasing symptoms • **PEFR >50–75% best or predicted** • No features of acute severe asthma
Acute severe asthma	Any one of: • **PEF 30–50% best or predicted** • Respiratory rate ≥25/min o Heart rate 120/min • Inability to complete sentences in one breath
Life-threatening asthma	Any one of the followings in a patient with severe asthma: • SpO2<92% (PaO2<60 mmHg) on high-flow FIO2 • PEF <30% best or predicted • Bradycardia • Dysrhythmia • Cyanosis • Hypotension • Normal or high PaCO2 • Exhaustion • Confusion • Silent chest (You hear nothing on auscultation) • Coma • Weak respiratory effort
Near-fatal asthma	• Raised PaCO2 and/or requiring mechanical ventilation
Brittle asthma	• Type 1: Wide PEF variability (>40% diurnal variation for >50% of the time over a period >3–6 months) despite intense therapy • Type 2: Sudden severe attacks on a background of apparently well-controlled asthma

1-I want you to be able to say what features are you going to pick out in the patient to decide the level of severity

Managing exacerbations in acute care settings



◀ Key message

- Asthma is a chronic inflammatory condition associated with significant morbidity and mortality which is preventable and **manageable** with appropriate treatment and effective patient communication

Remember !

There are Changes in asthma management:

- For safety, GINA no longer recommends SABA-only treatment for Step 1 in adults and adolescents
  - This decision was based on evidence that SABA-only treatment increases the risk of severe exacerbations, and that adding any ICS significantly reduces the risk
- **GINA now recommends that** all adults and adolescents with asthma should receive ICS-containing controller treatment, to reduce the risk of serious exacerbations
  - The ICS can be delivered by regular daily treatment or, in mild asthma, by as-needed **low dose ICS-formoterol**
  - Formoterol is long act with fast onset
  - Using ICS Because there is Inflammation and long term inflammation may end with **fixed** airway obstruction that no longer respond to bronchodilators (no longer reversible).

Glucocorticosteroids:

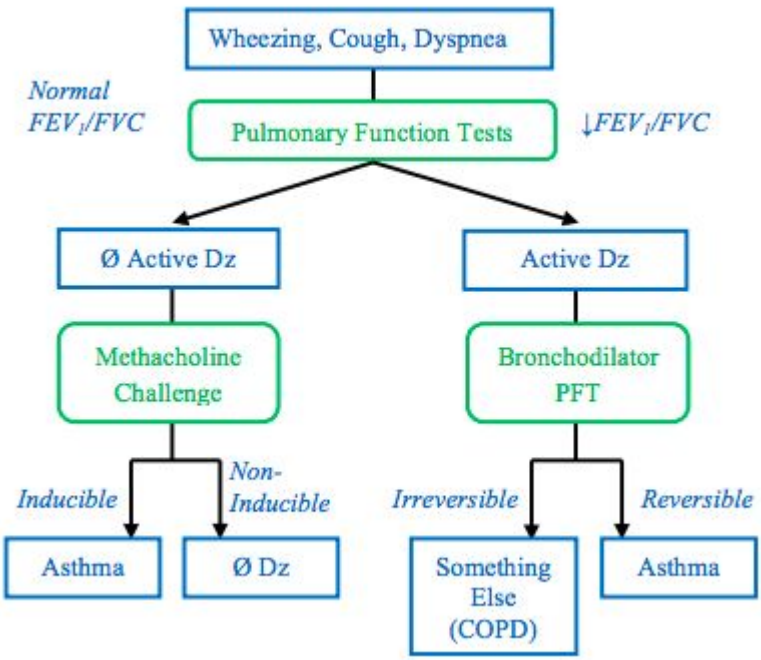
- Adverse effects of inhaled steroids are dysphonia and oral candidiasis.
- Systemic corticosteroids: They should be used as a last resort because of very harsh adverse effects such as:
  - Osteoporosis, Cataracts, Adrenal suppression and fat redistribution
  - Hyperlipidemia, hyperglycemia, acne, and hirsutism (particularly in women)
  - Thinning of skin, striae, and easy bruising
- Orally prednisone are added when all the other therapies are not sufficient to control symptoms.

Leukotrienes antagonists:

- best with atopic patient
- Zafirlukast is hepatotoxic and has been associated with Churg- Strauss syndrome.

Monoclonal antibodies:

Reslizumab (anti-IL-5) or mepolizumab (anti- IL-5) or omalizumab (anti-IgE); use omalizumab **only** if there are allergies and high IgE level



If someone comes in with an acute onset of wheezing and dyspnea SKIP the diagnosis and move directly to treatment. In the outpatient setting there are a number of diagnostic modalities, of which **Pulmonary Function Testing i**s by far the best. If the patient has active airway disease at the time of the test the **FEV1/FVC will be decreased**. They can be **reversed with bronchodilators** to definitively diagnose asthma. A normal patient does not rule out asthma. A patient suspected of having asthma but a normal FEV1/FVC can be given the methacholine challenge test to provoke bronchoconstriction.

Summary

Diagnosis

- Episodic Sx
- Triggered
- Wheeze
- Atopic history

Management

- Monitoring
- Education
- Control of environmental factors
- Pharmacologic
- Prevention

Clinical features that increase asthma probability

1	Sx worsen at night and in early morning
2	Sx in response to exercise, allergen exposure and cold air
3	Sx after taking aspirin or beta blockers
4	Family Hx of asthma/atopic disorder
5	Otherwise unexplained peripheral blood eosinophilia
6	Otherwise unexplained low FEV1 or PEF

Clinical features that decrease asthma probability

1	Prominent dizziness, light-headedness, peripheral tingling
2	Chronic productive cough, cough in the absence of wheeze or breathlessness
3	Voice disturbance
4	Sx with colds only
5	Cardiac disease
6	Significant Hx of smoking > 20 pack-years

Treatment

Step 1

Controller	Reliever
Low dose ICS - formoterol other option Low dose ICS taken whenever SABA is taken	As needed low dose ICS - formoterol other option As needed short acting B_2 agonist

Step 2

Controller	Reliever
Daily low dose inhaled corticosteroid **or** as needed low dose ICS - formoterol other option LTRA or low dose ICS taken whenever SABA is taken	As needed low dose ICS - formoterol other option As needed short acting B_2 agonist

Step3

Controller	Reliever
Low dose ICS - LABA other option medium dose ICS or low dose ICS+LTRA	As needed low dose ICS - formoterol other option As needed short acting B_2 agonist

Step 4

Controller	Reliever
Medium dose ICS - LABA other option High dose ICS add tiotropium or LTRA	As needed low dose ICS - formoterol other option As needed short acting B_2 agonist

Step 5

Controller	Reliever
High dose ICS - LABA refer for phenotypic assessment ±add on therapy eg.Tiotropium AntiIGE Anti-IL5/5R Anti-IL4R Other options Add low dose OCS but consider side effects	As needed low dose ICS - formoterol other option As needed short acting B_2 agonist

Lecture Quiz

Q1: A 27-year-old woman with a history of persistent asthma presents to the Emergency Department with complaints of agitation, muscle tremors and a "racing heart". During her history, she mentions having recently increased the number of asthma medications being taken, as well as the frequency of use of her rescue inhaler. Which of the following is most likely responsible for her current symptoms?

A- Albuterol
B- ipratropium
C- Montelukast
D- Omalizumab

Q2: 25-year-old woman is admitted to accident and emergency with a severe exacerbation of asthma. On examination, her respiratory rate is 30, oxygen saturations are 95 per cent on 15 L O2 and temperature is 37.2°C. As you feel the peripheral pulse, the volume falls as the patient inspires. Which of the following explains this clinical sign ?

A- Increased left atrial filling pressures on inspiration
B- Decreased right ventricular filling pressures on inspiration
C- Peripheral vasodilation
D- Decreased left atrial filling pressures on inspiration

Q3: A 25-year-old woman who works in an office complains of shortness of breath during the recovery period from her usual aerobic exercise routine. She had a history of asthma as a child, but it went into remission when she was in junior high school. What single test would be the best to order to easily confirm that she is now having a return of her asthma?

A-Blood eosinophils
B- Measurement of forced vital capacity before and after bronchodilator
C-Measurement of the forced expiratory volume in the first second (FEV1) before and after bronchodilator
D-Testing for airway hyperresponsiveness with inhalation of cold air
E-Measurement of the diffusion capacity for carbon monoxide

Q4: A 25-year-old woman whose asthma is in good control comes to your office indicating that she and her husband are trying to have a child. Her only treatment has been inhaled albuterol on an as-needed basis, and she uses about four inhalers (each contains 200 pufs of medication) a year. What is the single statement most likely to be true about her asthma treatment?

A- There is no need to change her treatment while she is tryi ceive or becomes pregnant.
B- She should stop the albuterol inhaler and simply "suffer through" any asthma events.
C- She should continue the albuterol inhaler and start an inhaled corticosteroid.
D- She should consult an asthma specialist now before she attempts to become pregnant.
E- She should begin to use her albuterol inhaler on a regularly scheduled basis, two puffs four times a day. to con-

Q5: A 24-year-old G2P1 woman whom you observe for asthma comes in at 14 weeks' gestation. She has had increasing problems with her asthma, needing a rescue inhaler two or three times a day, and has had nocturnal awakening. She denies chest pain, recent upper respiratory infection, gastroesophageal reflux, or new exposures. What is the most likely cause of her asthma exacerbation?

A- The normal effects of pregnancy
B- The increased workload and oxygen consumption associated with pregnancy
C- She has discontinued her steroid inhaler and controller medication because of concern about the drug's effect on the fetus.
D- She has decreased her exercise.
E- The stress of work, pregnancy, and sleep deprivation

Answers: Q1:A | Q2:D | Q3:C | Q4:A | Q5:C

Answers Explanation File!

GOOD LUCK !

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