COPD in elderly

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SLO's of this COPD lecture :

- Describe and discuss the pathogenesis,
- clinical presentation of COPD in the elderly ,
- difference in clinical presentation, identification, functional changes,
- acute care stabilization,
- management and
- rehabilitation of COPD in the elderly

Burden of COPD

- **The WHO estimates 1.1 billion smoker worldwide, increasing to 1.6 billion by 2025 in low&middle-income countries.**

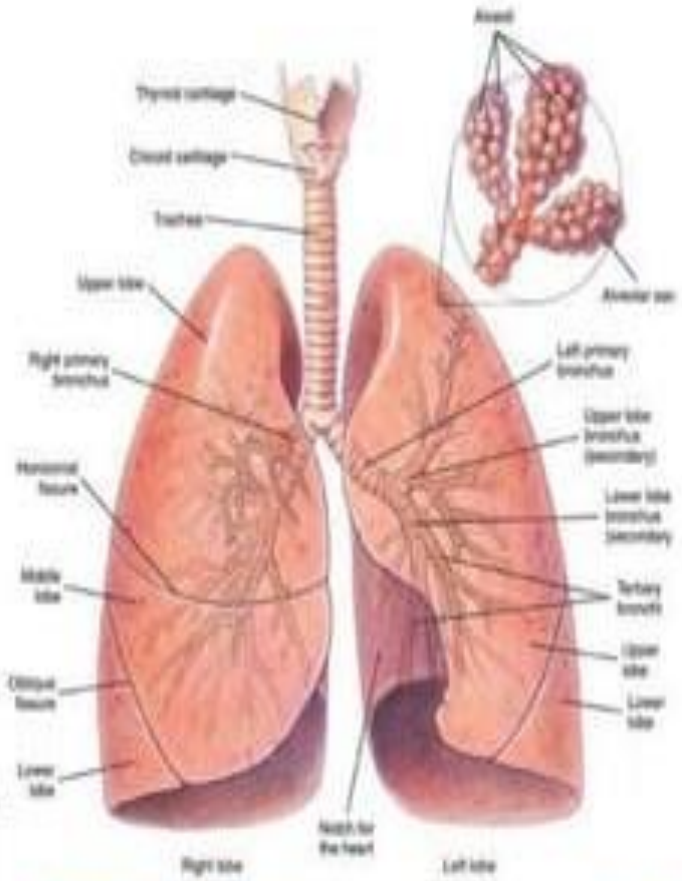
- COPD is the 4th leading cause of death in the United States (behind heart disease, cancer, and cerebrovascular disease).
- In 2000, the WHO estimated 2.74 million deaths worldwide from COPD.
- In 1990, COPD was ranked 12th as a burden of disease; by 2020 it is projected to rank 5th.

DEFINITION

Chronic respiratory diseases are a group of chronic diseases affecting the airway and other structure of the lungs

Common respiratory diseases in elder adults includes:-

- COPD
- Sleep disordered breathing
- Pulmonary embolism
- Bronchial asthma
- Tuberculosis
- Lung cancer
- Respiratory infections



Definition

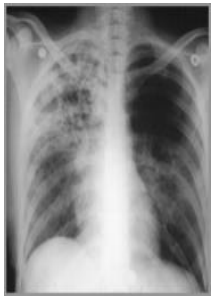
Chronic Obstructive Pulmonary Disease (COPD) is a preventable and treatable disease with some significant extrapulmonary effects that may contribute to the severity in individual patients. Its pulmonary component is characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases.

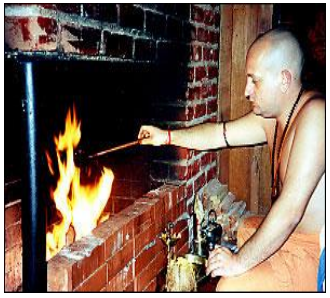
This definition *does not* use the terms chronic bronchitis and emphysema and excludes asthma (reversible airflow limitation).

What can cause COPD?

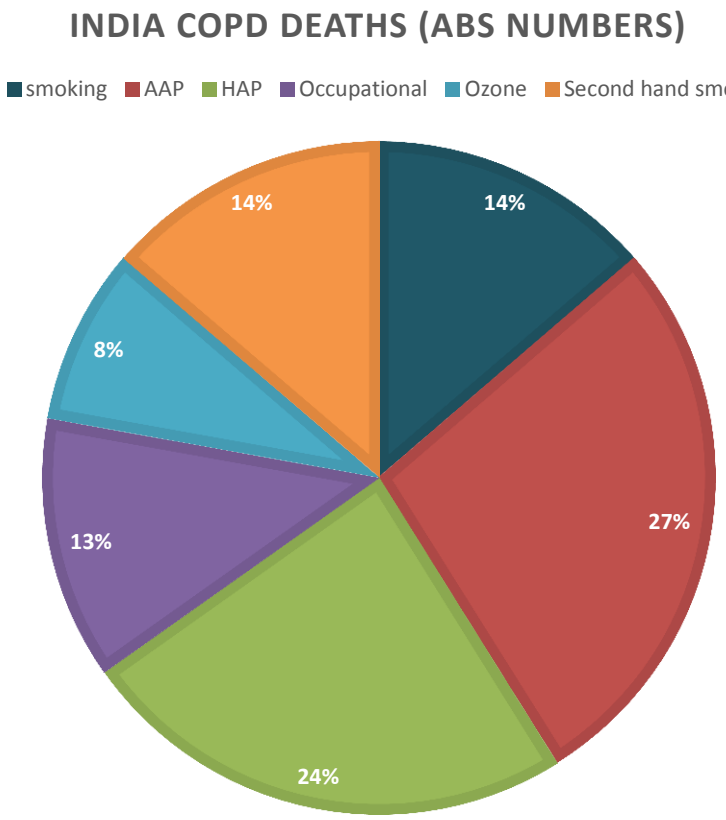
- Smoking is the primary risk factor
  - **Long-term smoking** is responsible for 80-90 % of cases
    - Smoker, compared to non-smoker, is 10 times more likely to die of COPD
- Prolonged exposures to harmful particles and gases from:
  - Second-hand smoke,
  - Industrial smoke,
  - Chemical gases, vapors, mists & fumes
  - Dusts from grains, minerals & other materials

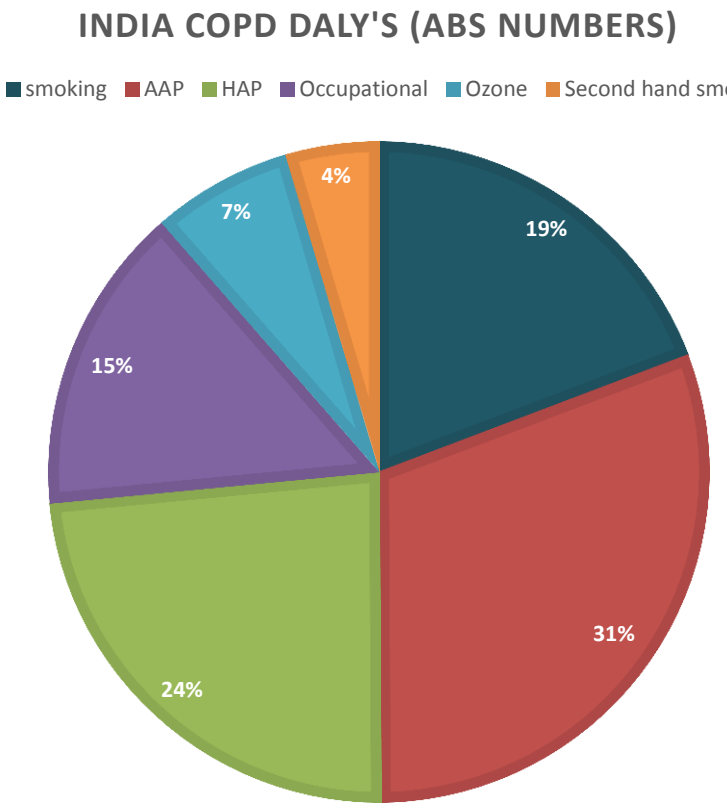
A Large Number of People in India are At Risk for COPD





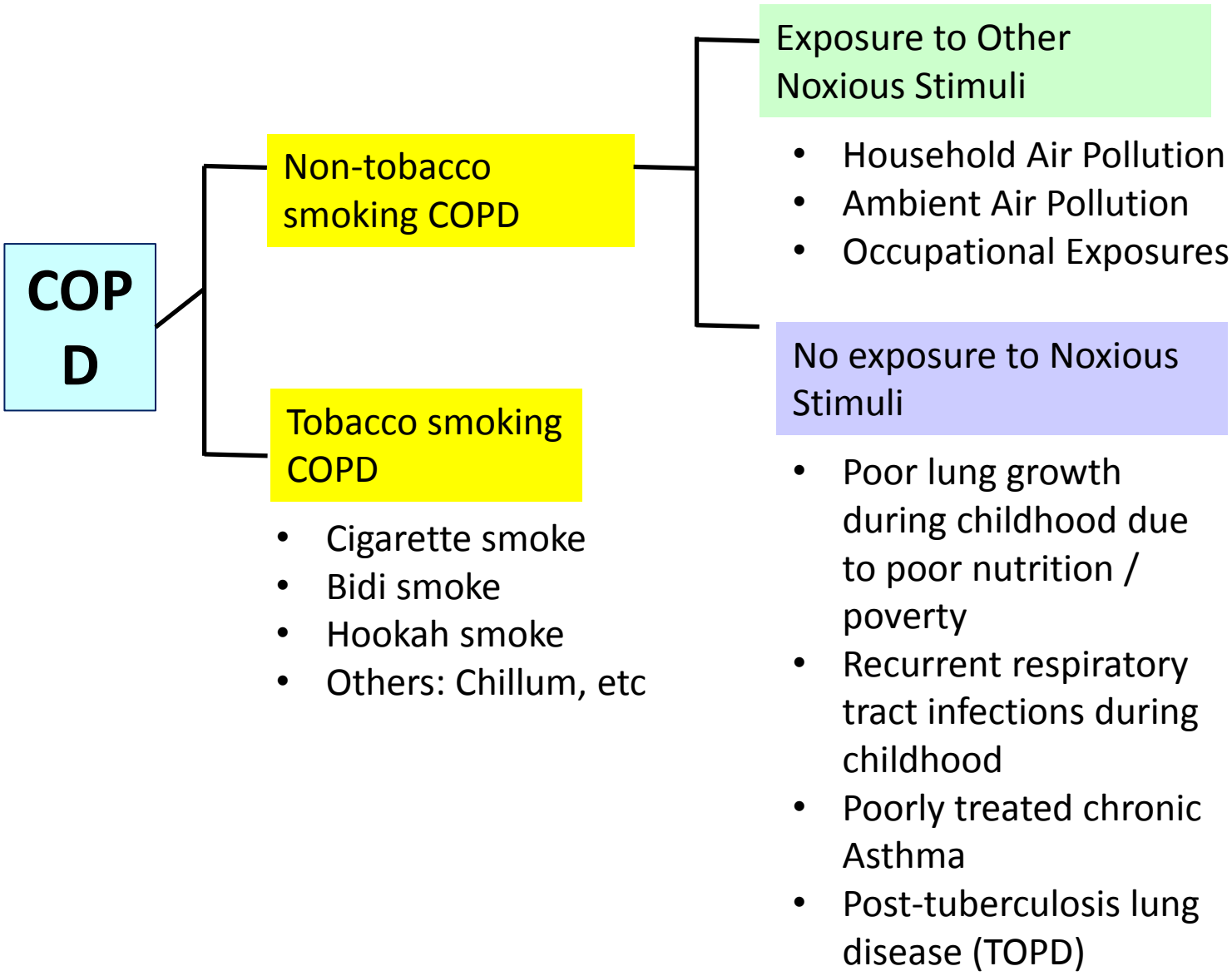
Over 80% of COPD in India is associated with Non-Smoking Causes





(Gobal Burden of Disease, 2018

Classification of COPD based on risk factors



Chronic bronchitis

Defined as the presence of cough and sputum production for at least 3 months in each of 2 consecutive years, is not necessarily associated with airflow limitation.

Emphysema

Defined as destruction of the alveoli, is a pathological term that is sometimes (incorrectly) used clinically.

Mechanisms Underlying Airflow Limitation in COPD

Small Airways Disease

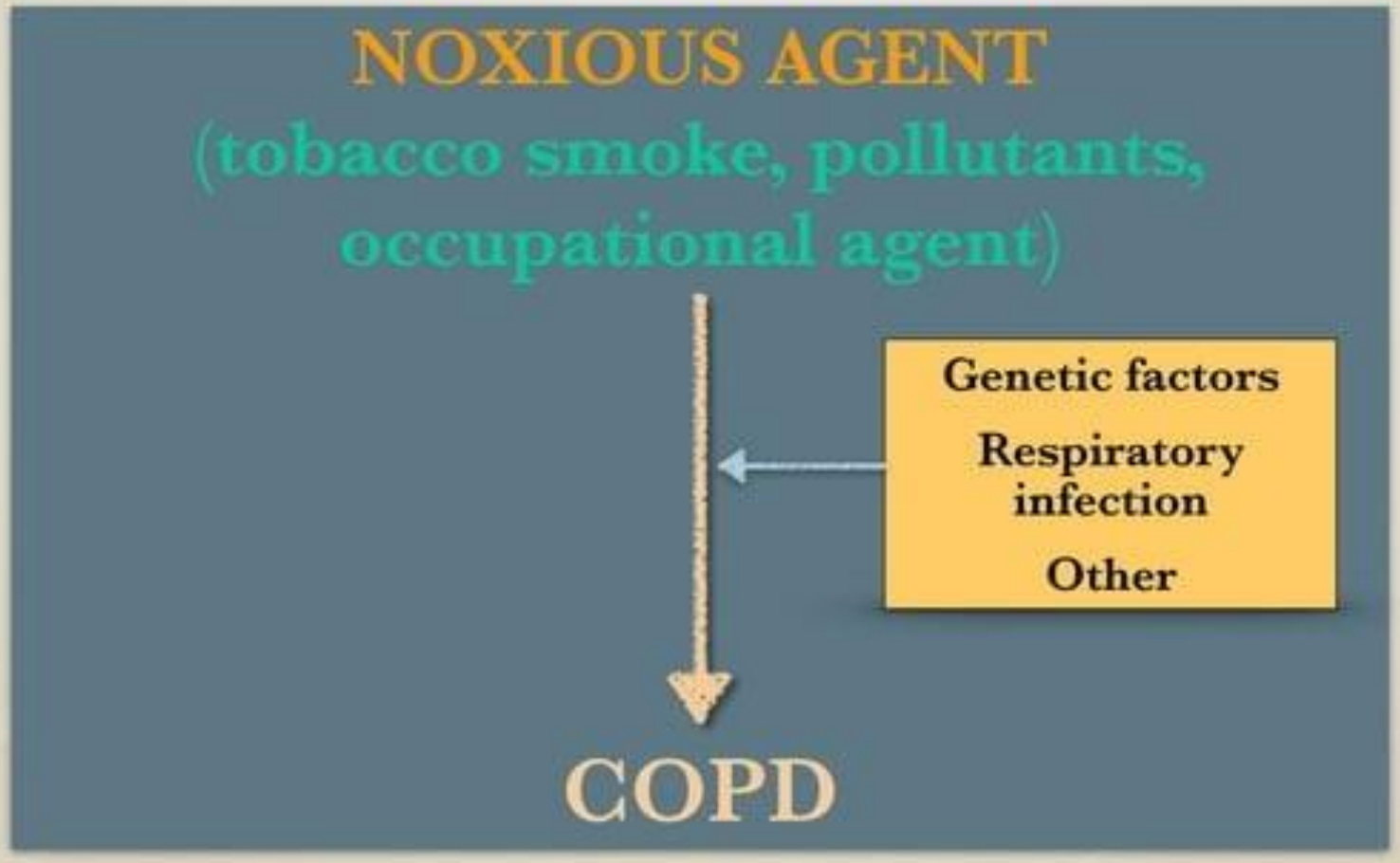
- Airway inflammation
- Airway fibrosis, luminal plugs
- Increased airway resistance

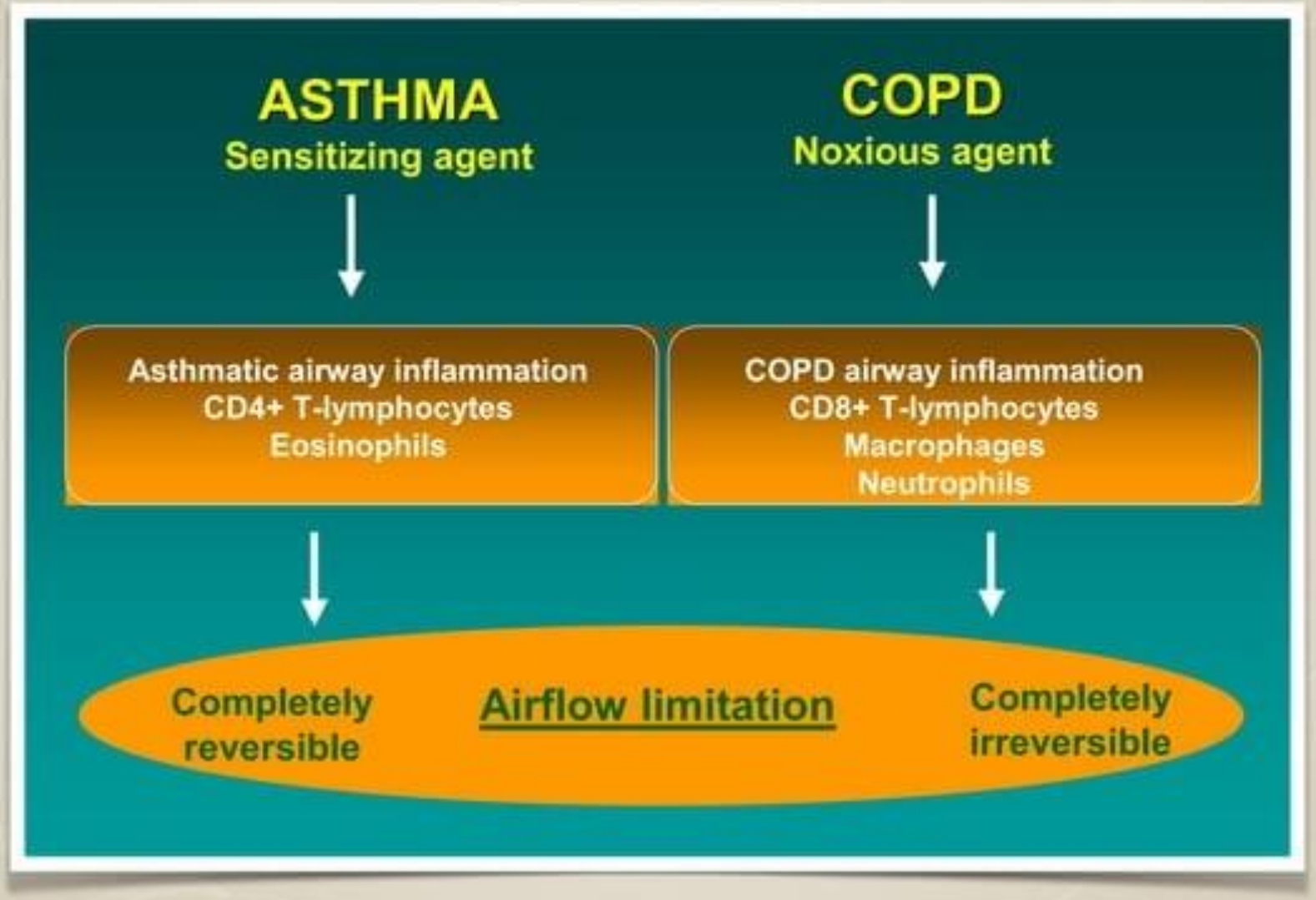
Parenchymal Destruction

- Loss of alveolar attachments
- Decrease of elastic recoil

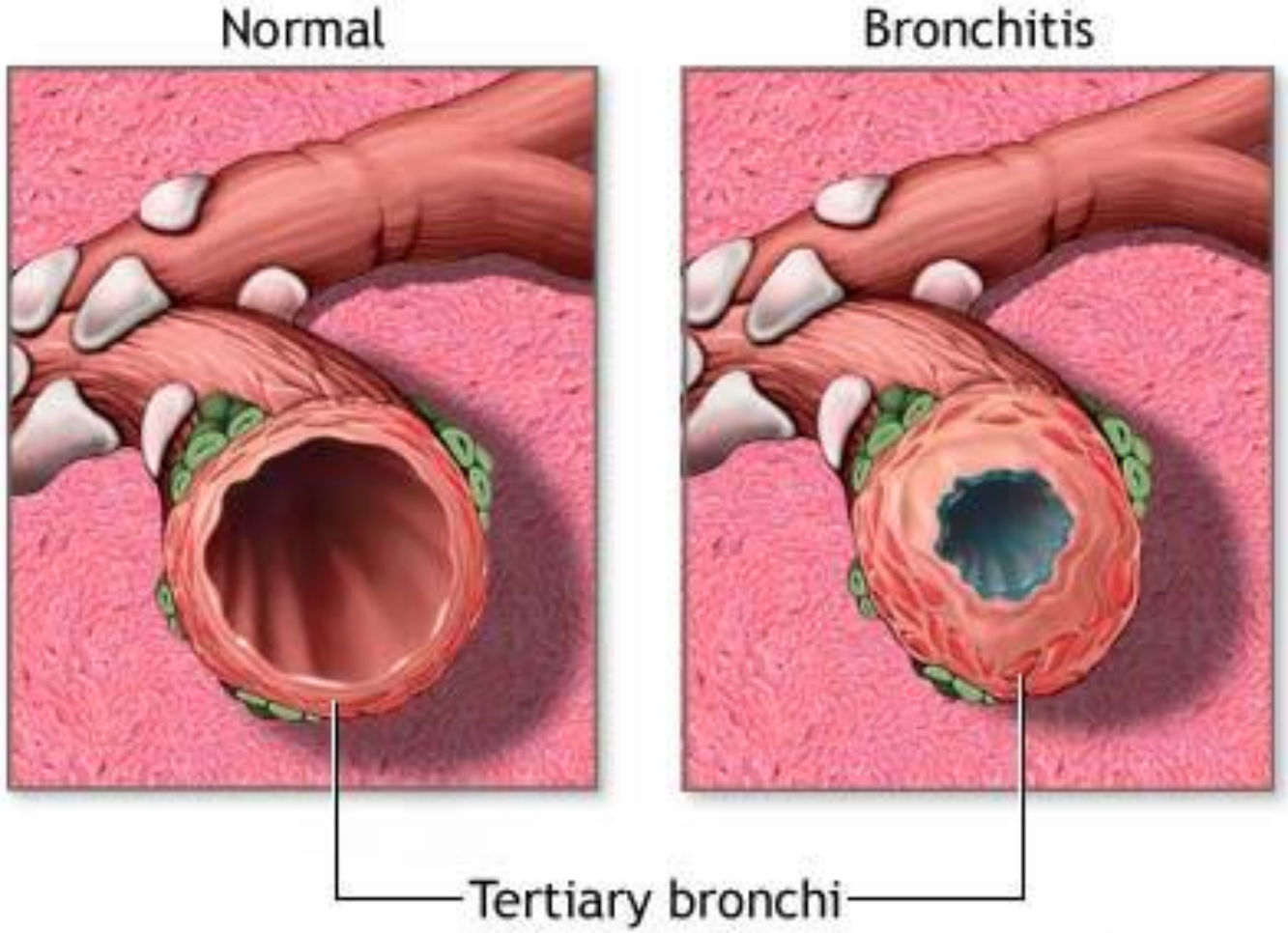
AIRFLOW LIMITATION

Pathogenesis

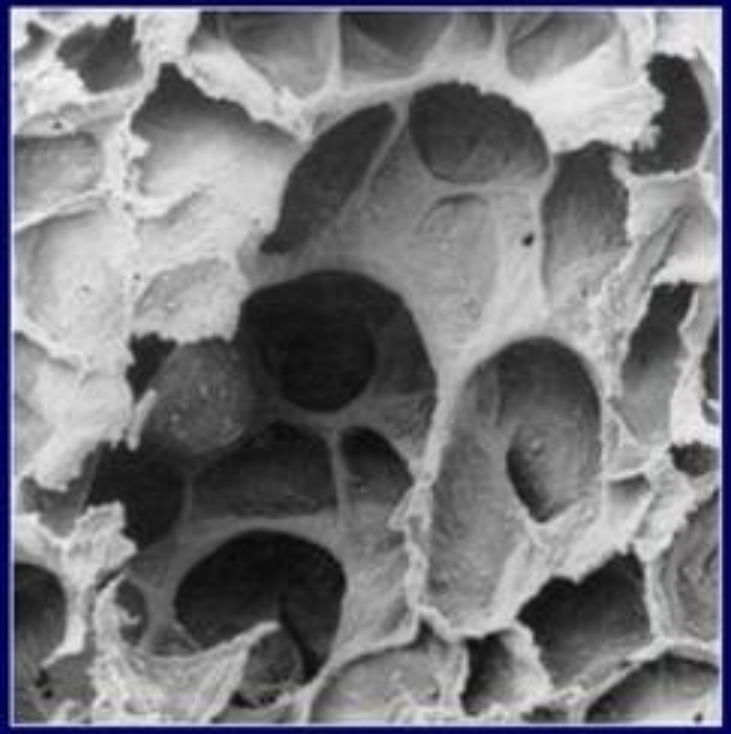



ASTHMA
Sensitizing agent
COPD
Noxious agent
Asthmatic airway inflammation
CD4+ T-lymphocytes
Eosinophils
COPD airway inflammation
CD8+ T-lymphocytes
Macrophages
Neutrophils
Completely reversible
Airflow limitation
Completely irreversible

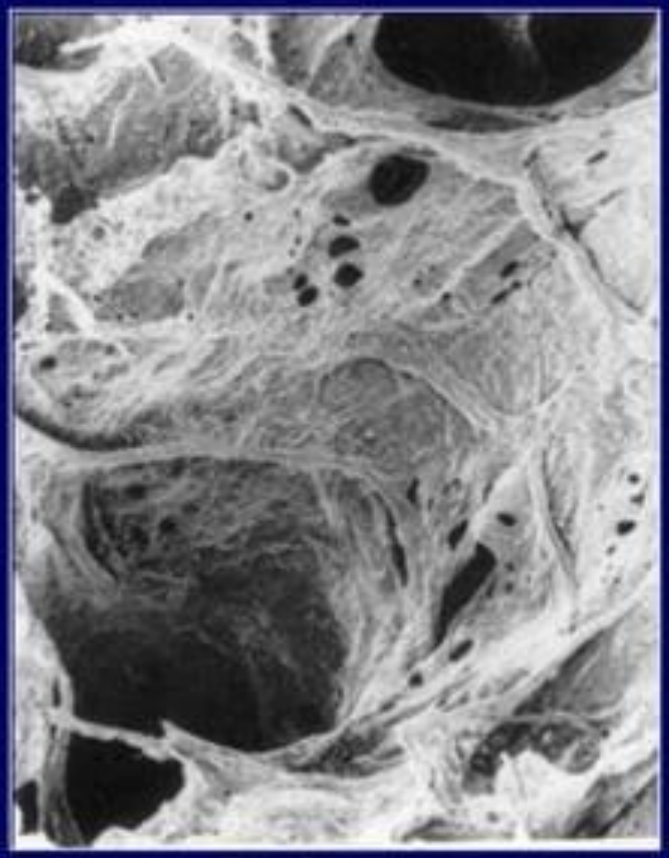
Normal versus Diseased Bronchi



Alveolar Destruction With Emphysema



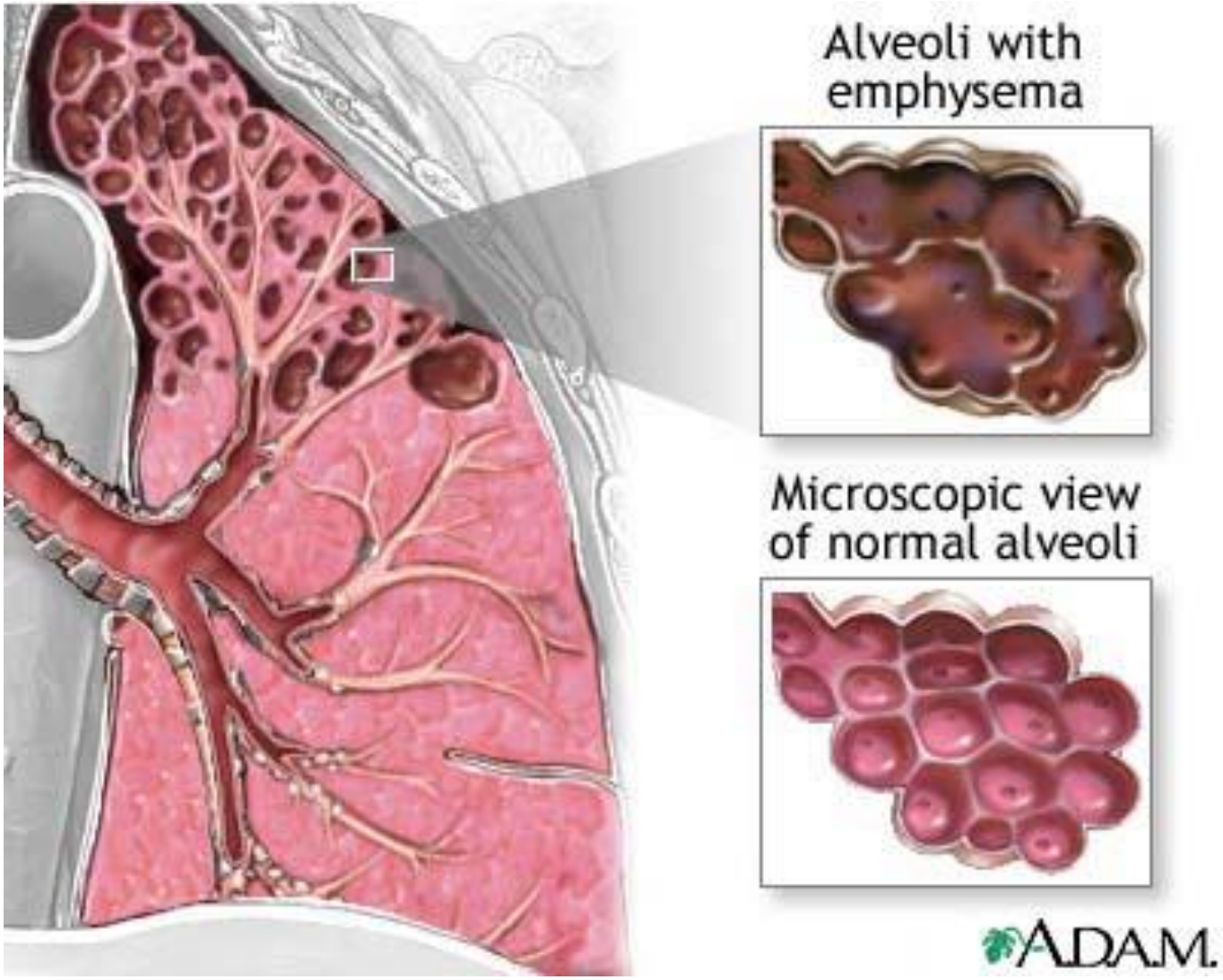
Normal



Emphysema

Nagai A, Thurlbeck WM. Scanning electron microscopic observations of emphysema in humans. A descriptive study. *Am Rev Respir Dis*. 1991;144:901-908. Official Journal Of The American Thoracic Society American Lung Association. 4604. Reprinted with permission

Emphysema


Alveoli with emphysema
Microscopic view of normal alveoli
A.D.A.M.

- COPD is predominantly caused by smoking and is characterised by airflow obstruction that:
  - is not fully reversible
  - does not change markedly over several months
  - is usually progressive in the long term
- Exacerbations often occur, where there is a rapid and sustained worsening of symptoms beyond normal day-to-day variations requiring a change in treatment

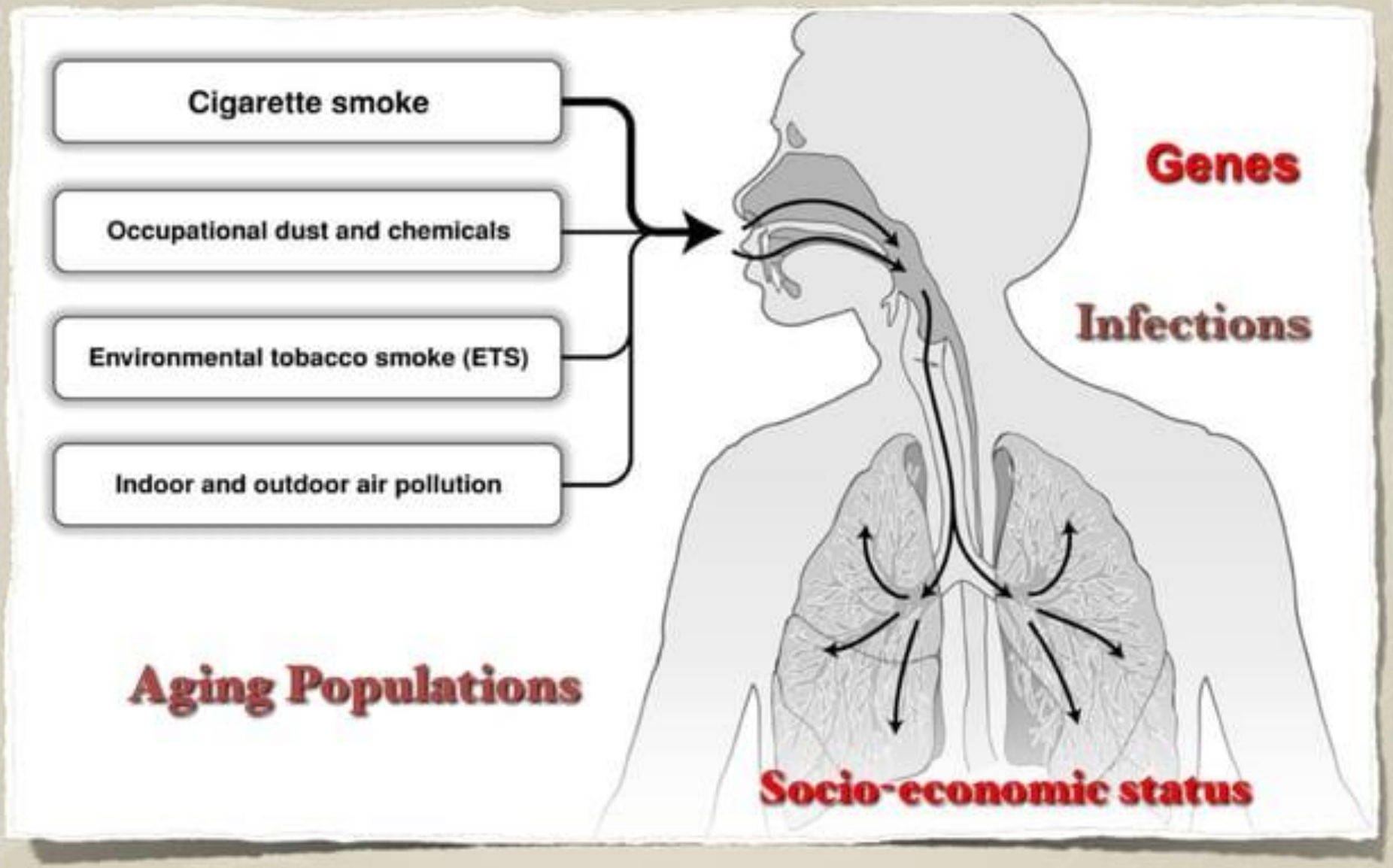
RISK FACTORS

- SMOKING
- LACK OF PHYSICAL ACTIVITY
- POOR NUTRITION
- TOBACCO USE
- EXCESSIVE ALCOHOL CONSUMPTION
- AGE
- HEREDITARY
- POOR SOCIO ECONOMIC STATUS

Risk Factors for COPD

* Genes
* Exposure to particles
* Tobacco smoke
* Occupational dusts, organic and inorganic
* Indoor air pollution from heating and cooking with biomass in poorly ventilated dwellings
* Outdoor air pollution

* Lung growth and development
* Gender
* Age
* Respiratory infections
* Socioeconomic status
* Asthma/Bronchial hyperreactivity
* Chronic Bronchitis


Cigarette smoke
Occupational dust and chemicals
Environmental tobacco smoke (ETS)
Indoor and outdoor air pollution
Genes
Infections
Aging Populations
Socio-economic status

Risk factors

- Cigarette smoking
  - Primary cause of COPD***
  - Clinically significant airway obstruction develops in 15% of smokers
  - 80% to 90% of COPD deaths are related to tobacco smoking
  - > 1 in 5 deaths is result of cigarette smoking

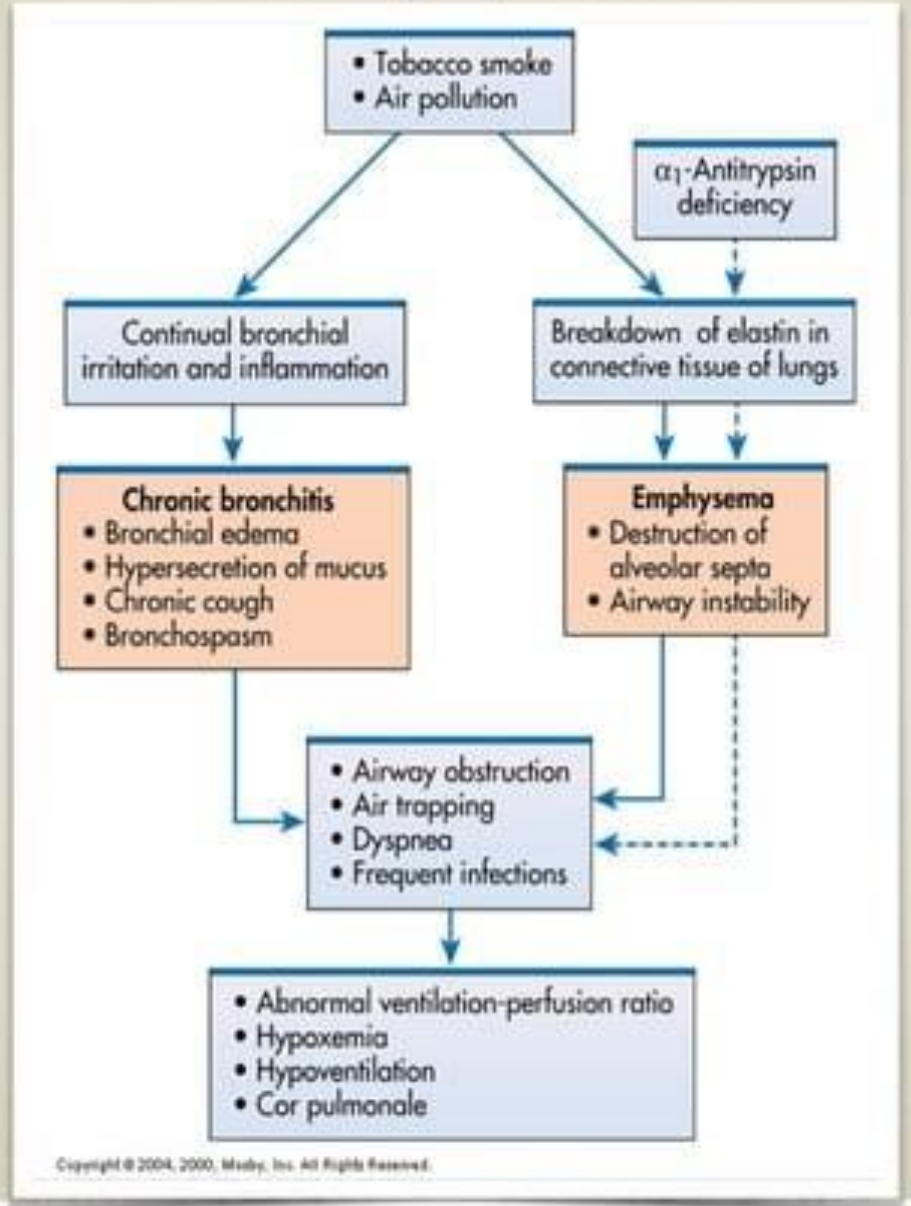
- Cigarette smoking
  - Nicotine stimulates sympathetic nervous system resulting in:
    - ↑ HR
    - Peripheral vasoconstriction
    - ↑ BP and cardiac workload

- Cigarette smoking
  - Compounds problems in a person with CAD
  - ↓ Ciliary activity
  - Possible loss of ciliated cells
  - Abnormal dilation of the distal air space
  - Alveolar wall destruction
  - Carbon monoxide
    - ↓ O_2 carrying capacity
    - Impairs psychomotor performance and judgment
  - Cellular hyperplasia
    - Production of mucus
    - Reduction in airway diameter
    - Increased difficulty in clearing secretions

Risk factor

- **Secondhand smoke exposure associated with:**
  - ↓ Pulmonary function
  - ↑ Risk of lung cancer
  - ↑ Mortality rates from ischemic heart disease
- **Infection**
  - Major contributing factor to the aggravation and progression of COPD
- **Heredity**
  - α-Antitrypsin (AAT) deficiency (produced by liver and found in lungs); accounts for < 1% of COPD cases
    - Emphysema results from lysis of lung tissues by proteolytic enzymes from neutrophils and macrophages

Pathophysiology of Chronic Bronchitis and Emphysema



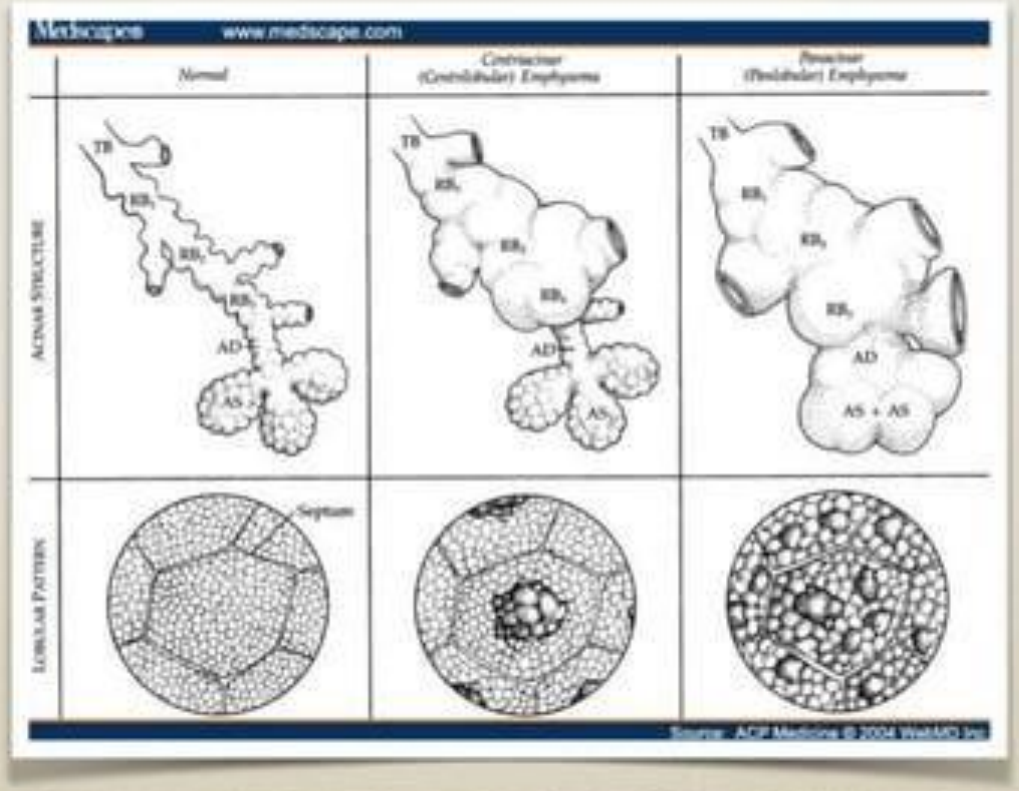
Emphysema
Pathophysiology

- **Hyperinflation of alveoli**
- **Destruction of alveolar walls**
- **Destruction of alveolar capillary walls**
- **Narrowed airways**
- **Loss of lung elasticity**

- **Two types:**
  - **Centrilobular (central part of lobule)**
    - Most common
  - **Panlobular (destruction of whole lobule)**
    - Usually associated with AAT deficiency

Emphysema

- Abnormal permanent enlargement of the air space distal to the terminal bronchioles
- Accompanied by destruction of bronchioles



Damaging cycle of COPD

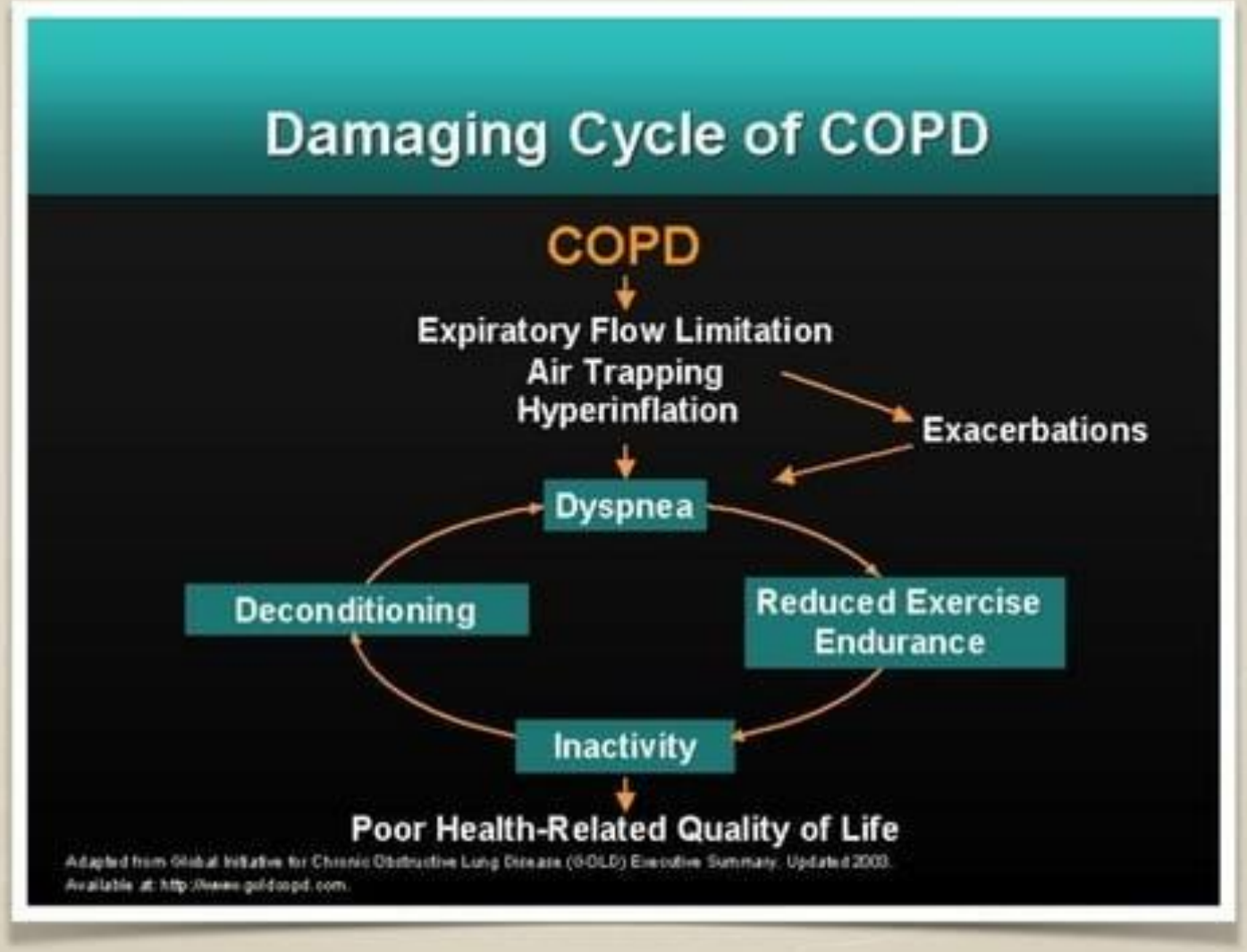


Figure 1: Key Indicators for Considering a COPD Diagnosis

Consider COPD, and perform spirometry, if any of these indicators are present in an individual over age 40. These indicators are not diagnostic themselves, but the presence of multiple key indicators increases the probability of a diagnosis of COPD.

- **Dyspnea** that is: Progressive (worsens over time).
  Usually worse with exercise.
  Persistent (present every day).
  Described by the patient as an "increased effort to breathe," "heaviness," "air hunger," or "gasping."
- **Chronic cough:** May be intermittent and may be unproductive.
- **Chronic sputum production:**
  Any pattern of chronic sputum production may indicate COPD.
- **History of exposure to risk factors:**
  Tobacco smoke (including popular local preparations).
  Occupational dusts and chemicals.
  Smoke from home cooking and heating fuel.

SIGN AND SYMPTOMS

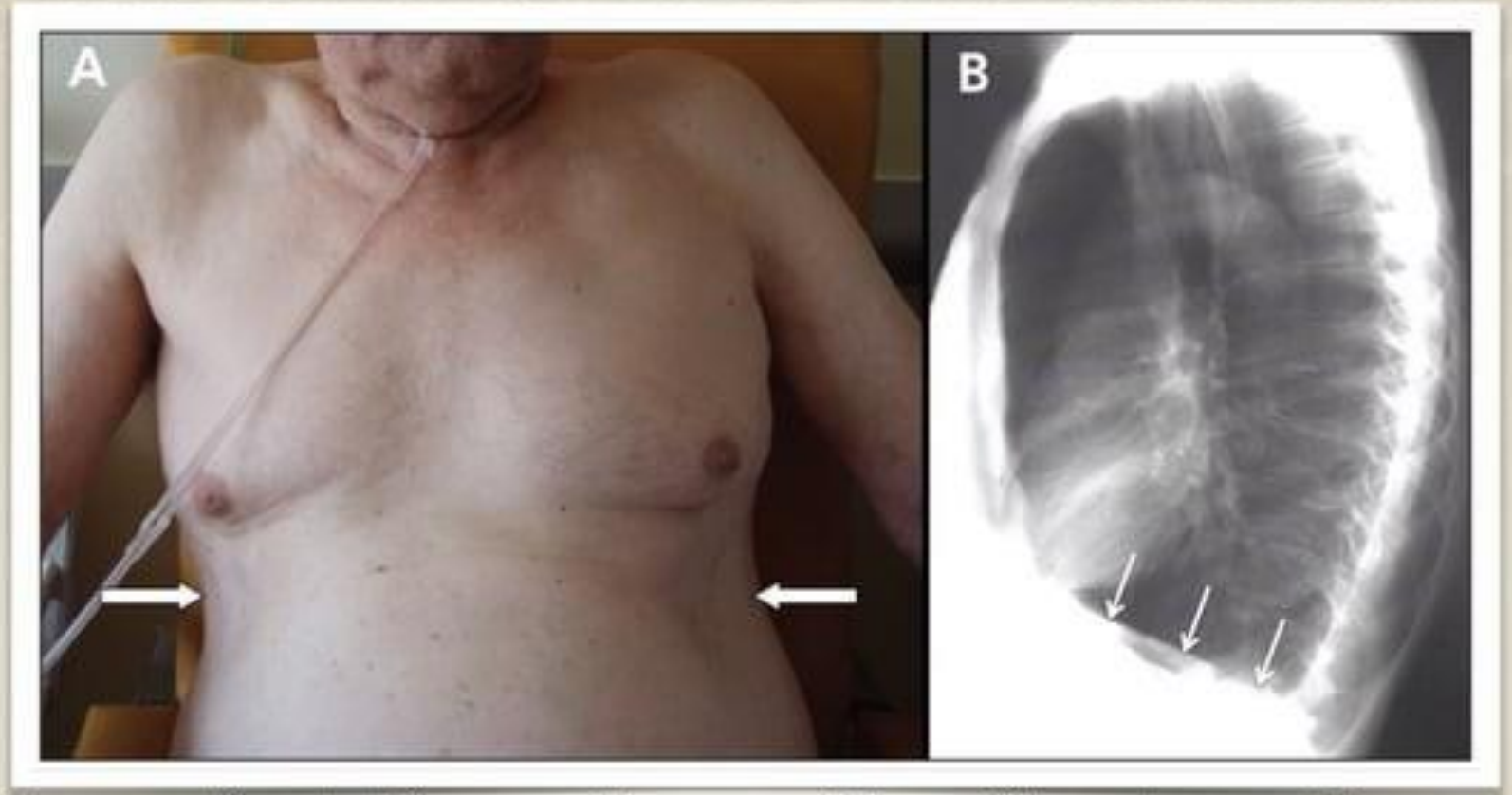
- DYSPNEA
- CHRONIC COUGH
- STRIDOR
- WHEEZING
- HYPER VENTILATION
- SNEEZING
- PAIN IN THROAT AND CHEST
- SPUTUM PRODUCTION
- EPISTAXIS
- HEMOPTYSIS

Emphysema
Clinical Manifestations

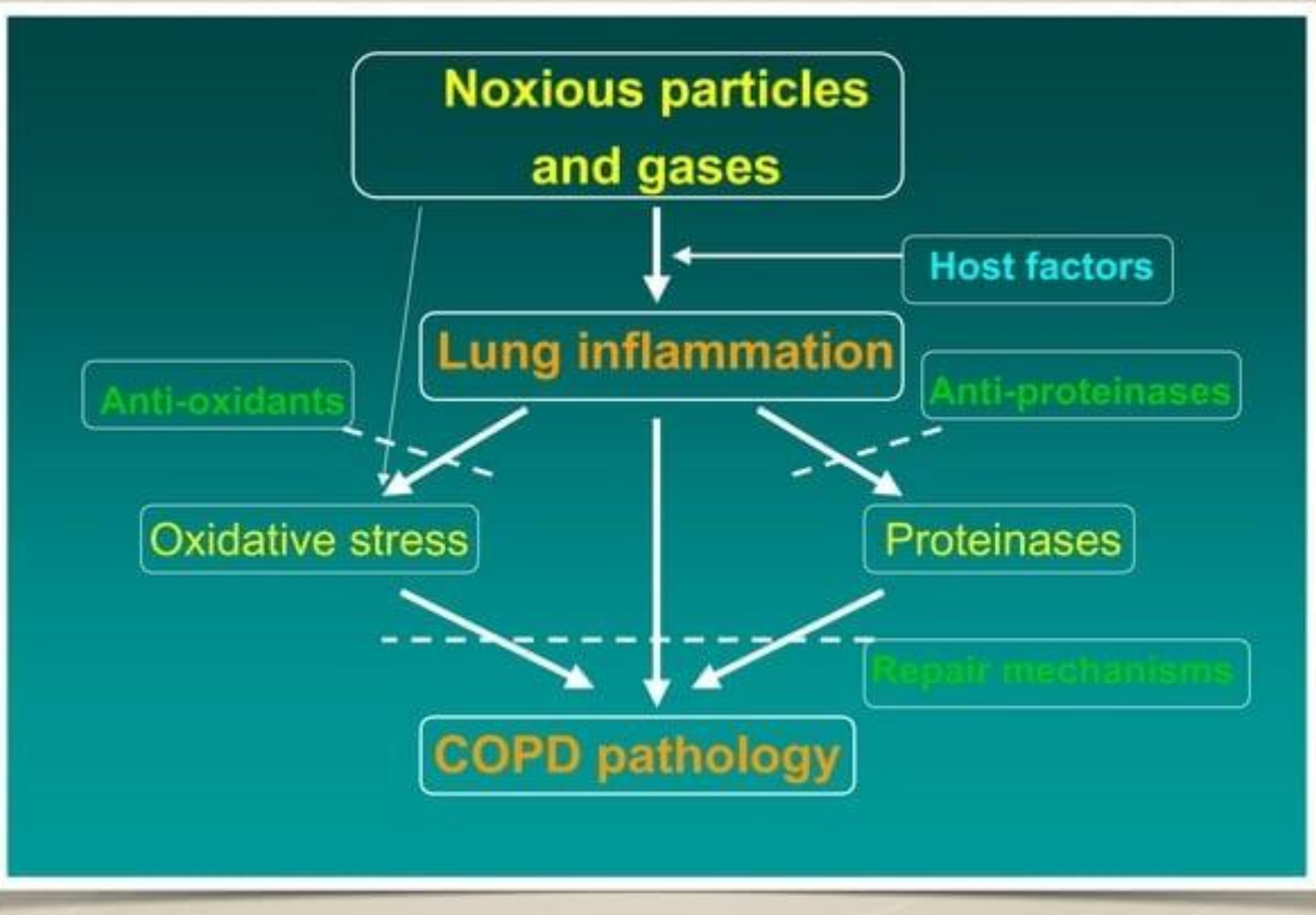
- Dyspnea
  - Progresses in severity
  - Patient will first complain of dyspnea on exertion and progress to interfering with ADLs and rest
- Minimal coughing with no to small amounts of sputum
- Overdistention of alveoli causes diaphragm to flatten and AP diameter to increase
- Patient becomes chest breather, relying on accessory muscles
  - Ribs become fixed in inspiratory position
- Patient is underweight (despite adequate calorie intake)

Chronic Bronchitis
Clinical Manifestations

- **Earliest symptoms:**
  - **Frequent, productive cough during winter**
  - **Frequent respiratory infections**
- **Bronchospasm at end of paroxysms of coughing**
- **Cough**
- **Dyspnea on exertion**
- **History of smoking**
- **Normal weight or overweight**
- **Ruddy (bluish-red) appearance d/t**
  - **polycythemia (increased Hgb d/t chronic hypoxemia)**
  - **cyanosis**
- **Hypoxemia and hypercapnia**
  - **Results from hypoventilation and ↑ airway resistance + problems with alveolar gas exchange**



This case for A 70-year-old man with a 70 pack-year history of smoking was referred to the emergency department for an exacerbation of chronic obstructive pulmonary disease (COPD).
On examination, he breathed through pursed lips and had paradoxical indrawing of the lower rib cage margin with inspiration (**Hoover** sign),A chest radiograph showed distension of the lungs and flattening of the diaphragm.


Noxious particles and gases
Host factors
Lung inflammation
Anti-oxidants
Anti-proteinases
Oxidative stress
Proteinases
Repair mechanisms
COPD pathology

Diagnosis Of COPD

Indicators for considering the diagnosis of COPD	
Chronic cough	Present intermittently or every day, often present throughout the day
Chronic sputum production	Any pattern of sputum production may indicate COPD
Acute bronchitis	Repeated episodes
Dyspnea that is	Progressive, persistent, worse on exercise, worse during respiratory infections
History of exposure to risk factors	Smoke, biomass fuel, occupational dusts

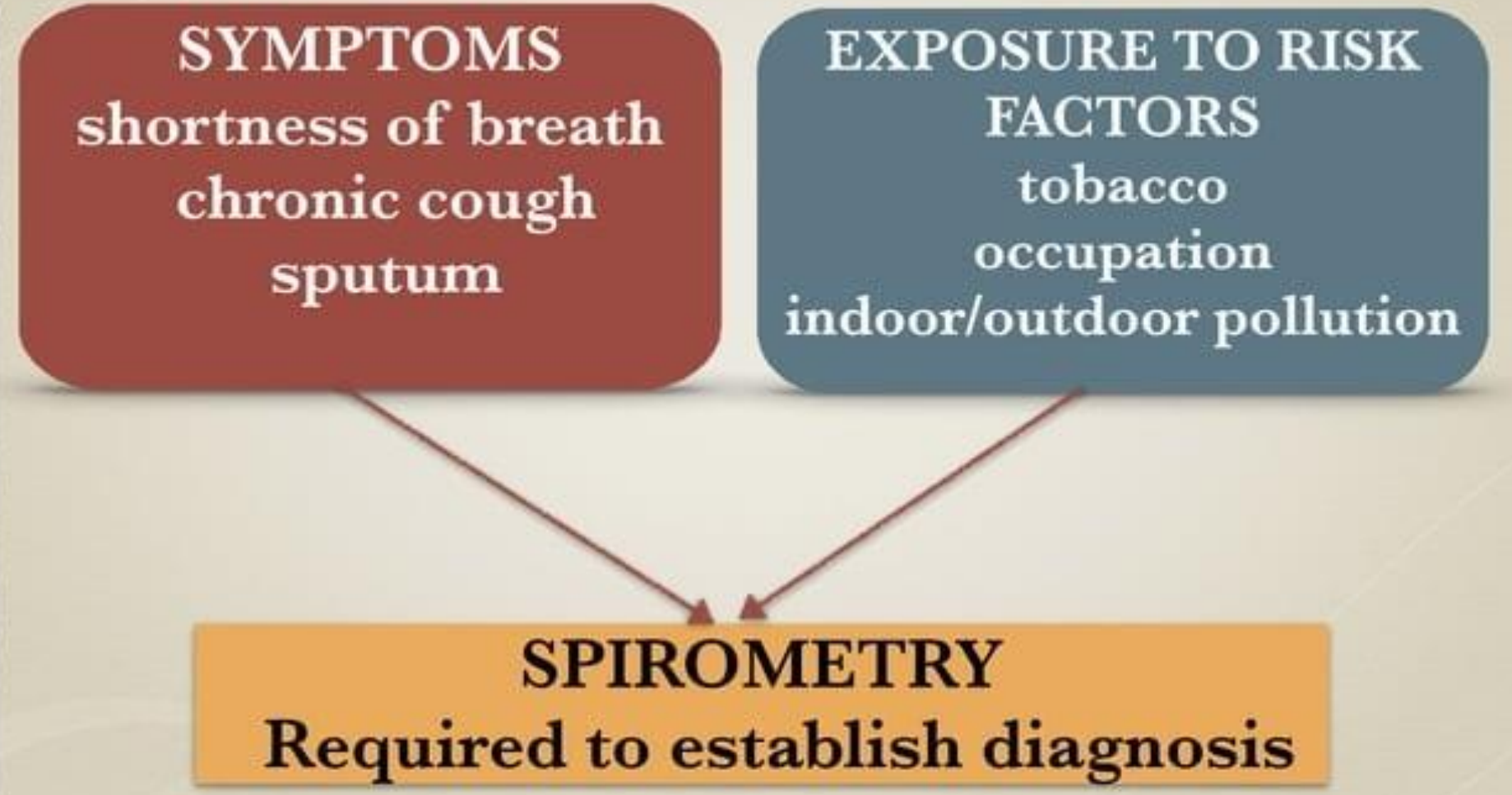
The diagnosis should be confirmed by Spirometry

Diagnosis and Assessment: Key Points

- A clinical diagnosis of COPD should be considered in any patient who has dyspnea, chronic cough or sputum production, and a history of exposure to risk factors for the disease.
- Spirometry is required to make the diagnosis; the presence of a post-bronchodilator $FEV_1/FVC < 0.70$ confirms the presence of persistent airflow limitation and thus of COPD.

Diagnosis

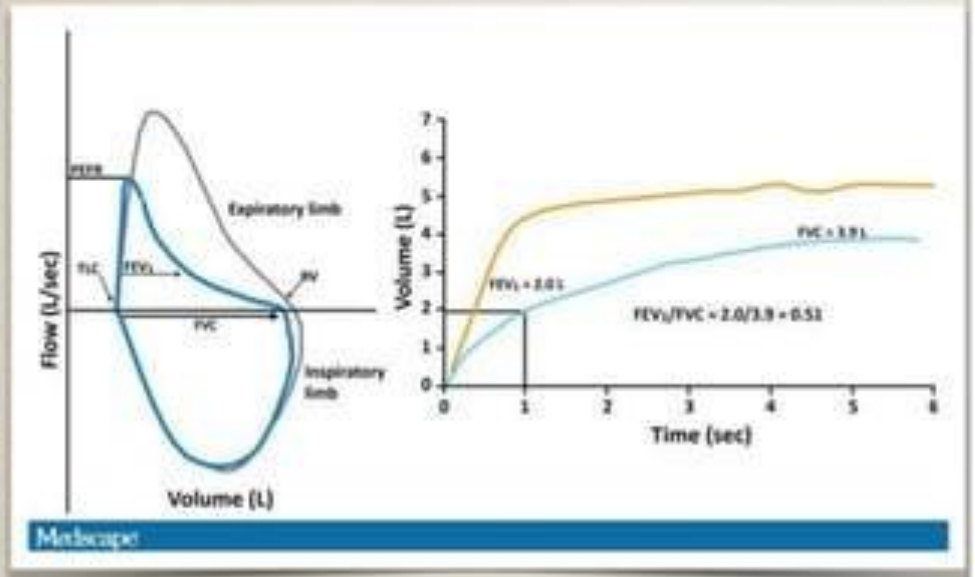
- The goals of COPD assessment are to determine the severity of the disease, including the severity of airflow limitation, the impact on the patient's health status, and the risk of future events.
- Comorbidities occur frequently in COPD patients, and should be actively looked for and treated appropriately if present.

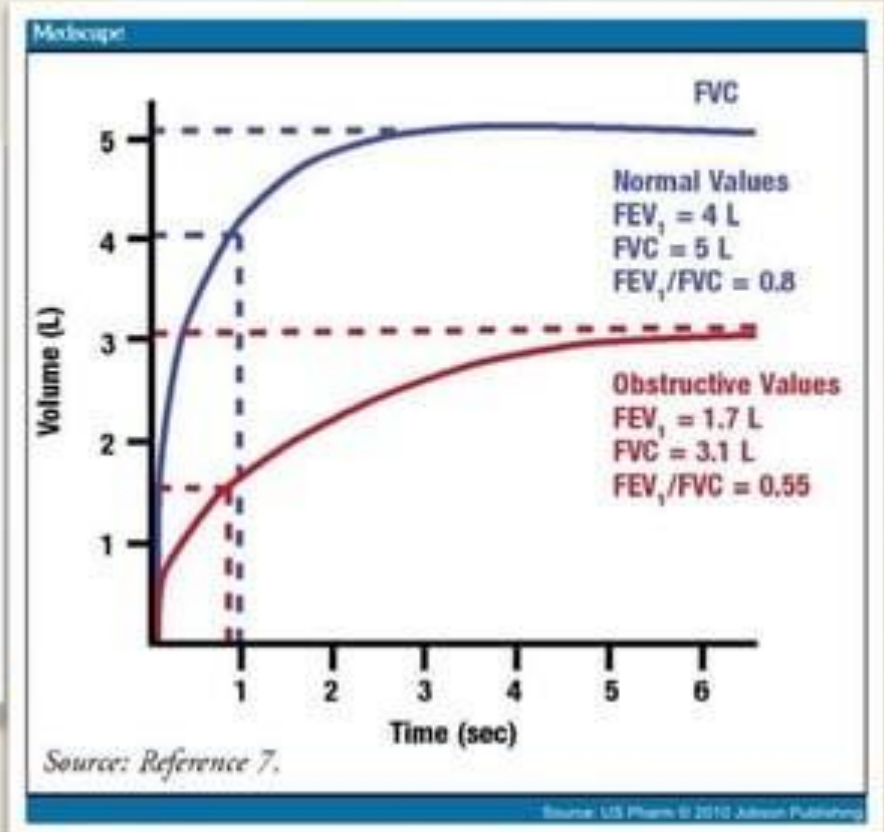

SYMPTOMS
shortness of breath
chronic cough
sputum
EXPOSURE TO RISK FACTORS
tobacco
occupation
indoor/outdoor pollution
SPIROMETRY
Required to establish diagnosis

Assessment of Airflow Limitation: Spirometry

- Spirometry should be performed after the administration of an adequate dose of a short-acting inhaled bronchodilator to minimize variability.
- A post-bronchodilator $FEV_1/FVC < 0.70$ confirms the presence of airflow limitation.
- Where possible, values should be compared to age-related normal values to avoid overdiagnosis of COPD in the elderly.

Spirometry in COPD



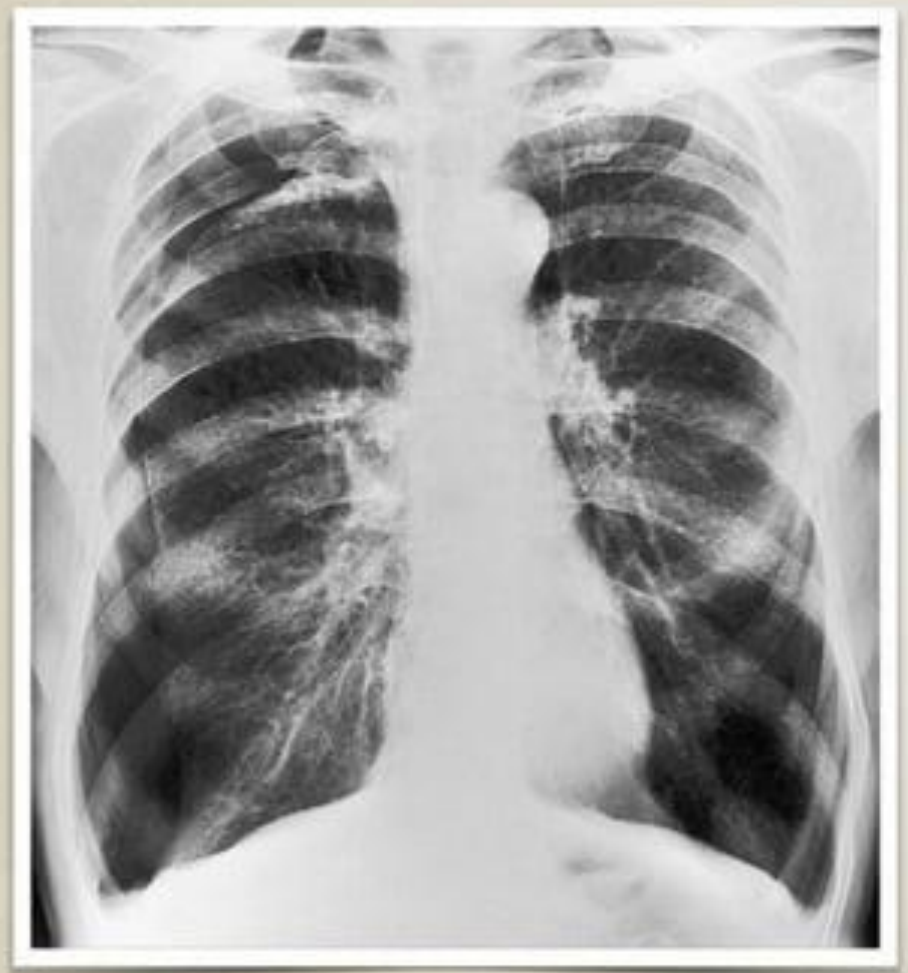


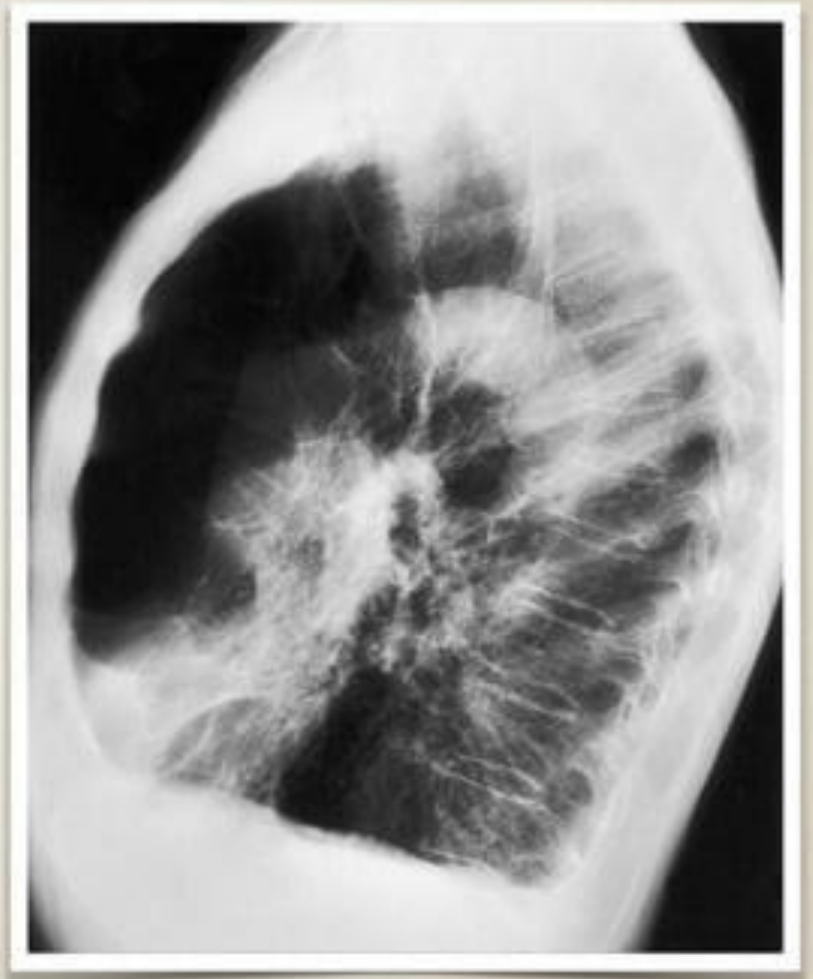
Category/Severity Stage	FEV_1/FEV	FEV_1 (% Predicted)
Normal (healthy patients)	0.80	~100
I: Mild	<0.70	≥80
II: Moderate	<0.70	50 to <80
III: Severe	<0.70	30 to <50
IV: Very Severe	<0.70	<30[a]

- Airflow obstruction is defined as reduced FEV_1/FVC ratio (< 0.7)
- It is no longer necessary to have an FEV_1 < 80% predicted for definition of airflow obstruction
- If FEV_1 is ≥ 80% predicted, a diagnosis of COPD should only be made in the presence of respiratory symptoms, for example breathlessness or cough
- COPD produces symptoms, disability and impaired quality of life which may respond to pharmacological and other therapies that have limited or no impact on the airflow obstruction.

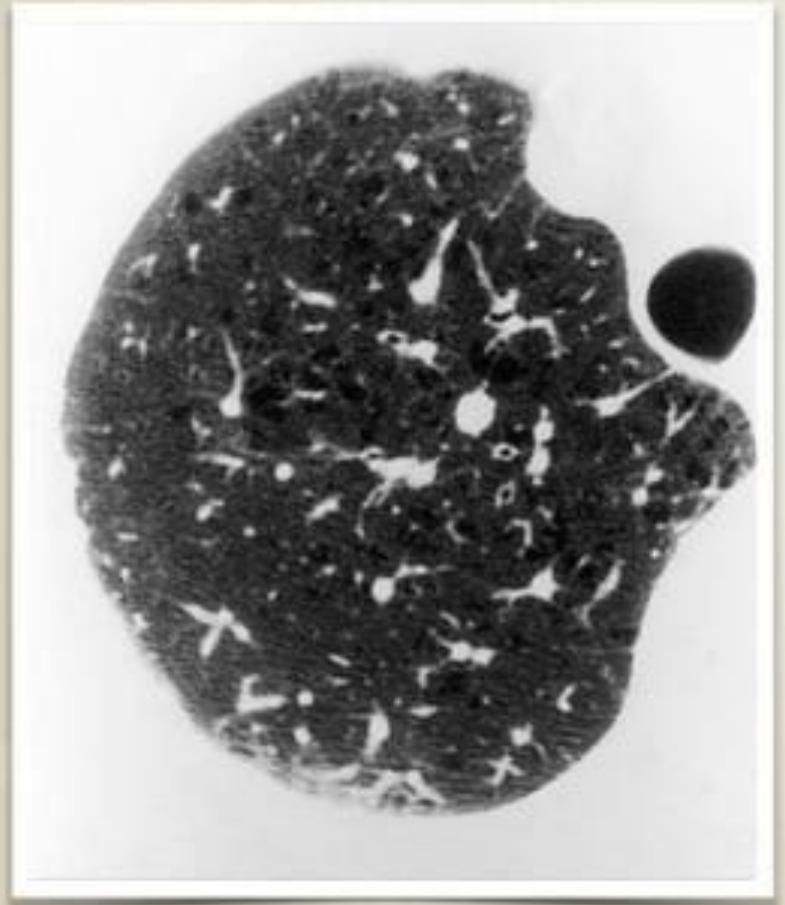
ANCILLARY INVESTIGATIONS IN COPD	
CBC	may show polycythemia, anemia of chronic disease
Oximetry & ABG	hypoxemia, hypercapnia (advanced COPD)
CXR	Hyper-inflation, low lying diaphragms, dirty chest
Chest HRCT	Centrilobular, para septal and pan lobular emphysema, bullae
Alpha-1 anti-trypsin level	In cases with early onset emphysema
Echo	In cases with suspected pulmonary hypertension
Lung volumes and diffusing capacities	Help to characterize severity, but not essential to patient management
Exercise testing	Objectively measured exercise impairment

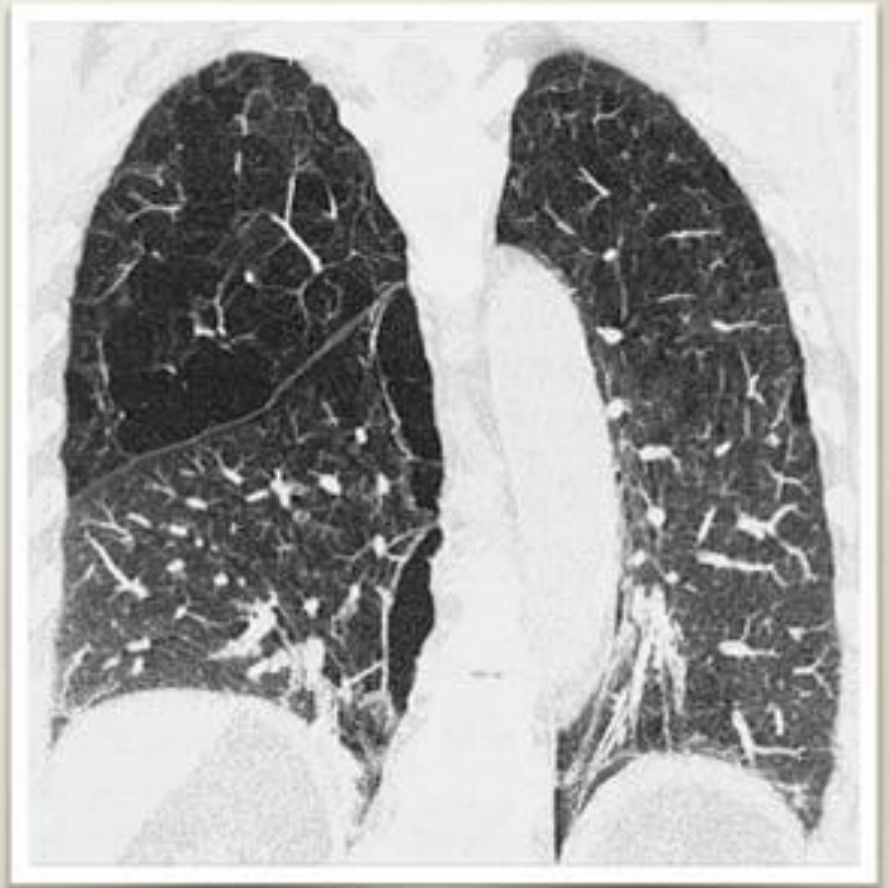
CXR in emphysema



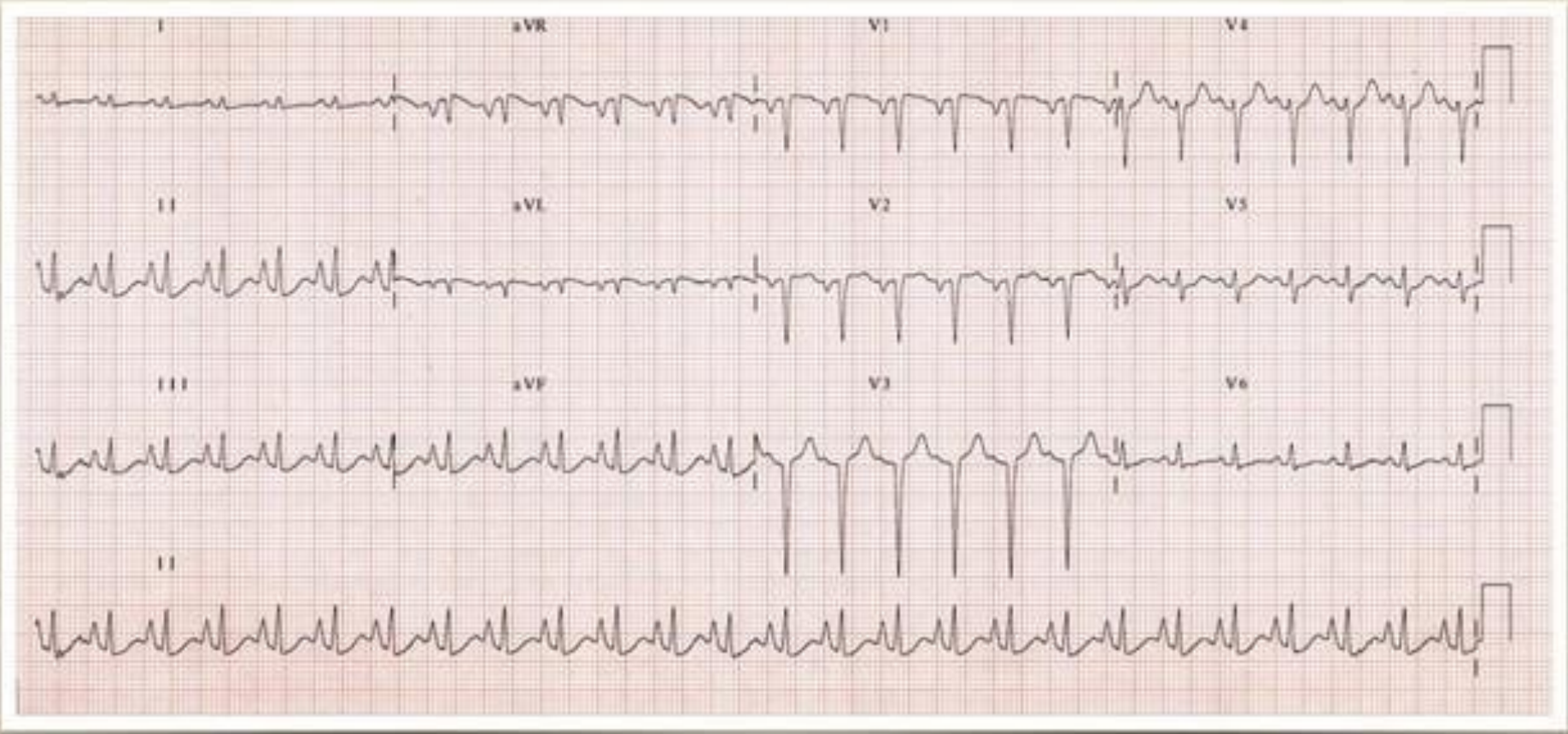


CT in emphysema

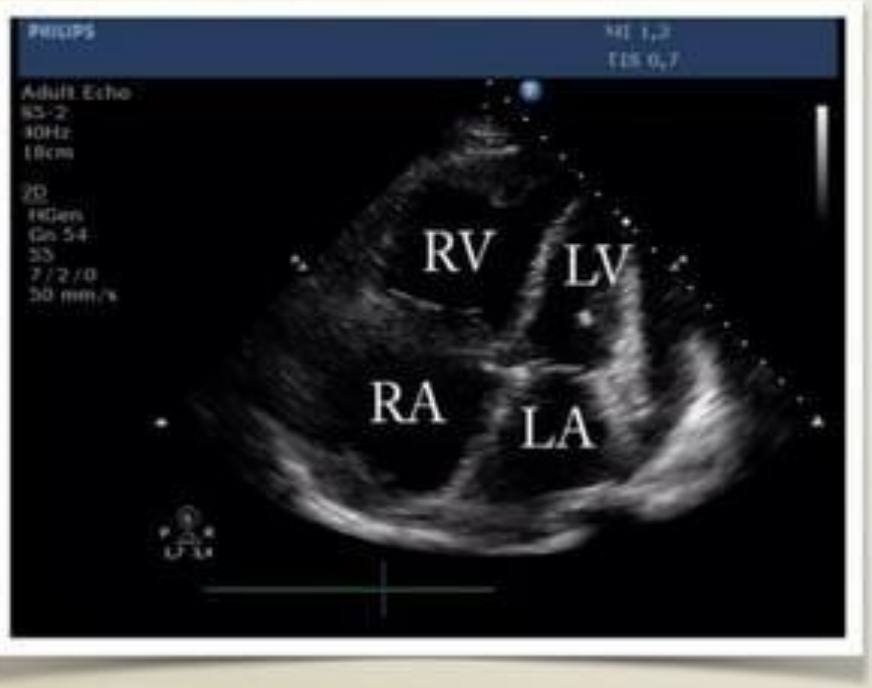


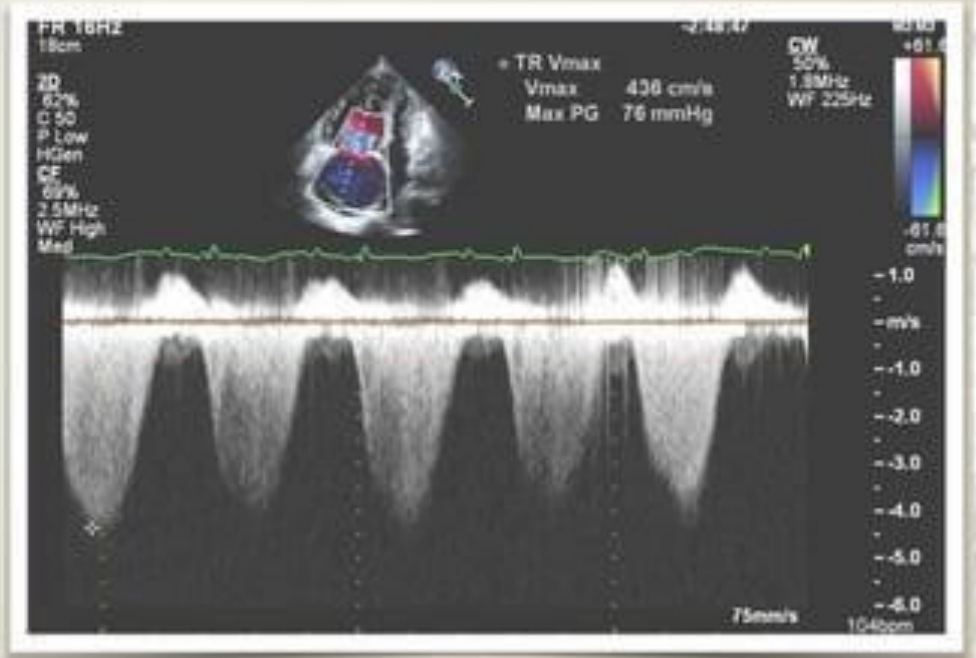


ECG in cor pulmonale (p pulmonale)



Echo in cor pulmonale





Complications of COPD

* acute exacerbation of COPD
* cor pulmonale
* Pneumothorax
* Pulmonary hypertension
* Respiratory failure
* Polycythemia

Systemic Inflammation in COPD

- COPD is an inflammatory condition
- Pro-inflammatory mediators may be the driving force behind the disease process
- Inflammation and actions of pro-inflammatory mediators may extend beyond the lungs and play a part in COPD comorbidities
- As effective anti-inflammatory therapy becomes available for COPD, it will be important to monitor the effects on lungs and associated comorbidities

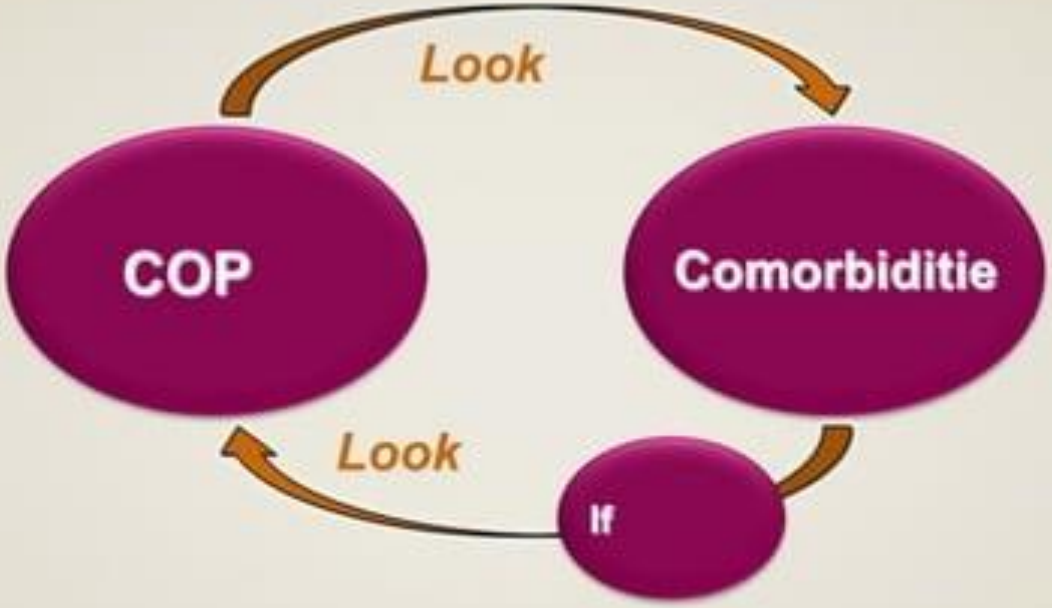
Systemic Inflammation in COPD: Potential Clinical Consequences

- Skeletal Muscle Dysfunction
- Cardiovascular
  - CHF
  - Arrhythmias
  - Hypertension (systemic pulmonary)
- Osteoporosis

- Anaemia of Chronic Disease
- Metabolic Disease
  - Diabetes
  - Metabolic Syndrome
- Depression
- Gastrointestinal
  - Ulcer Disease

COPD is a Systemic Disease

Assessing Comorbidities in COPD



These comorbid conditions may influence mortality and hospitalizations and should be looked for routinely, and treated appropriately.

Objectives of COPD Management

- Prevent disease progression
- Relieve symptoms
- Improve exercise tolerance
- Improve health status
- Prevent and treat exacerbations
- Prevent and treat complications
- Reduce mortality
- Minimize side effects from treatment

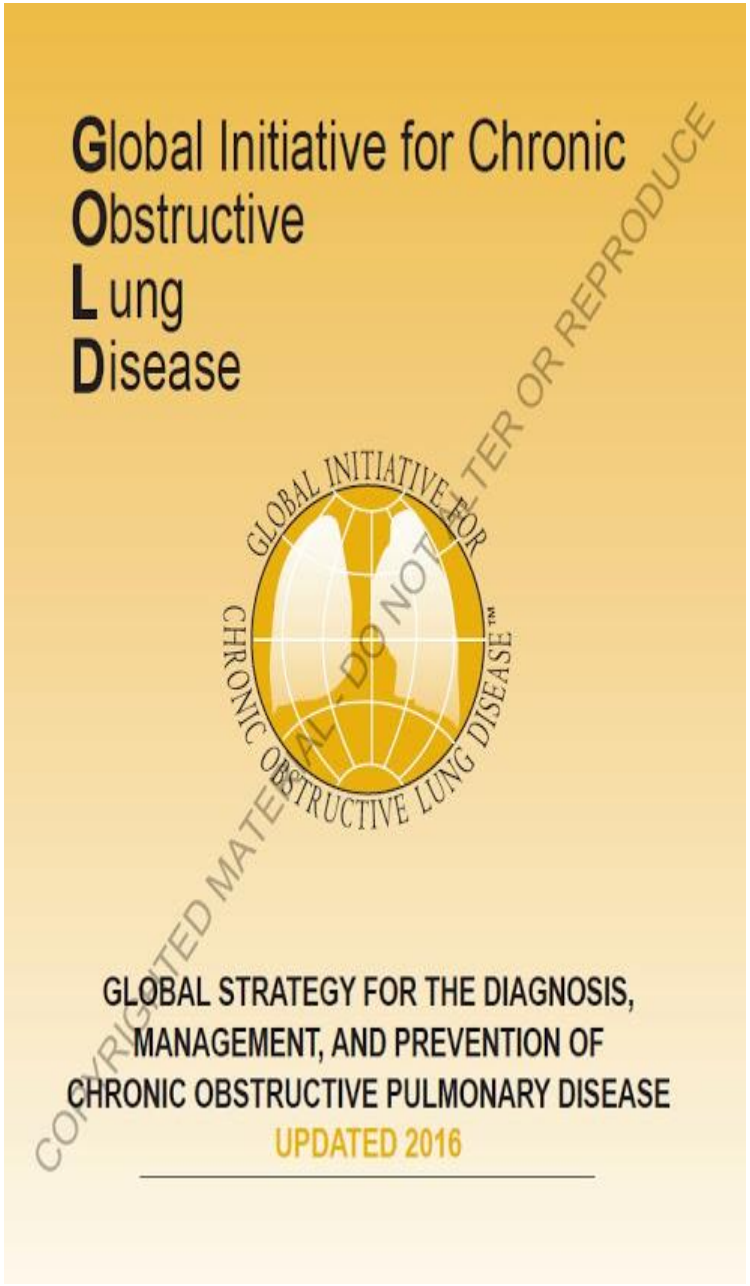
World Health Organization

National Heart Lung and Blood Institute

GLOBAL INITIATIVE FOR CHRONIC OBSTRUCTIVE LUNG DISEASE™

2001 Global Initiative for Obstructive Lung Disease

2004 Global Initiative for Chronic Obstructive Lung Disease



2001

2004

2006

2011

2012

2014

2015

2016

Global Strategy for Diagnosis, Management and Prevention of COPD
Assessment of COPD: Goals

Determine the severity of the disease, its impact on the patient's health status and the risk of future events (for example exacerbations) to guide therapy. Consider the following aspects of the disease separately:

- current level of patient's symptoms
- severity of the spirometric abnormality
- frequency of exacerbations
- presence of comorbidities.

Assessment of COPD

- Assess symptoms
- Assess degree of airflow limitation using spirometry
- Assess risk of exacerbations
- Assess comorbidities

Assessment of symptoms

COPD Assessment Test (CAT)

or

Clinical COPD Questionnaire (CCQ)

or

mMRC Breathlessness scale

The Modified Medical Research Council (MMRC) Dyspnoea Scale

Grade of dyspnoea	Description
0	Not troubled by breathlessness except on strenuous exercise
1	Shortness of breath when hurrying on the level *or* walking up a slight hill
2	Walks slower than people of the same age on the level because of breathlessness *or* has to stop for breath when walking at own pace on the level
3	Stops for breath after walking about 100 m *or* after a few minutes on the level
4	Too breathless to leave the house *or* breathless when dressing or undressing

Assess degree of airflow limitation using spirometry

Category/Severity Stage	FEV_1/FEV	FEV_1 (% Predicted)
Normal (healthy patients)	0.80	~100
I: Mild	<0.70	≥80
II: Moderate	<0.70	50 to <80
III: Severe	<0.70	30 to <50
IV: Very Severe	<0.70	<30[a]

Assess risk of exacerbations

To assess risk of exacerbations use history of exacerbations and spirometry:

- Two or more exacerbations within the last year or an $FEV_1 < 50$ % of predicted value are indicators of high risk.
- One or more hospitalizations for COPD exacerbation should be considered high risk.

American Journal of
RESPIRATORY AND
CRITICAL CARE MEDICINE®
Han M et al, 2010;
182: 598-604

Chronic Obstructive Pulmonary Disease Phenotypes

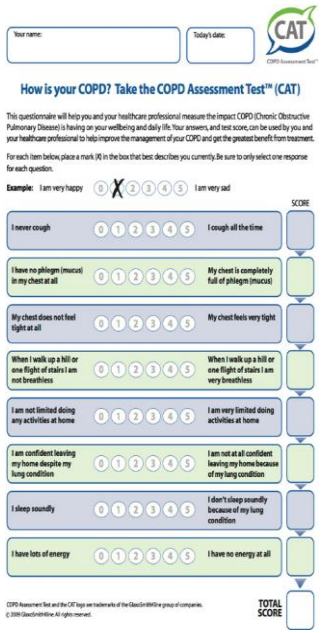
The Future of COPD

A COPD Phenotype is a single or combination of disease attributes that describe differences between individuals with COPD **as they relate to clinically meaningful outcomes** (symptoms, exacerbations, response to therapy, rate of disease progression, or death).

The Modified Medical Research Council (MMRC) Dyspnoea Scale

Grade of dyspnoea	Description
0	Not troubled by breathlessness except on strenuous exercise
1	Shortness of breath when hurrying on the level *or* walking up a slight hill
2	Walks slower than people of the same age on the level because of breathlessness *or* has to stop for breath when walking at own pace on the level
3	Stops for breath after walking about 100 m *or* after a few minutes on the level
4	Too breathless to leave the house *or* breathless when dressing or undressing

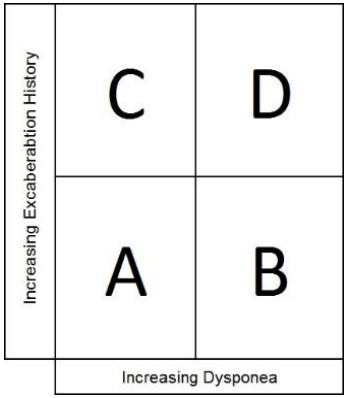
mMRC: < 2 and > 2



CAT: < 10 and > 10

SYMPTOMS

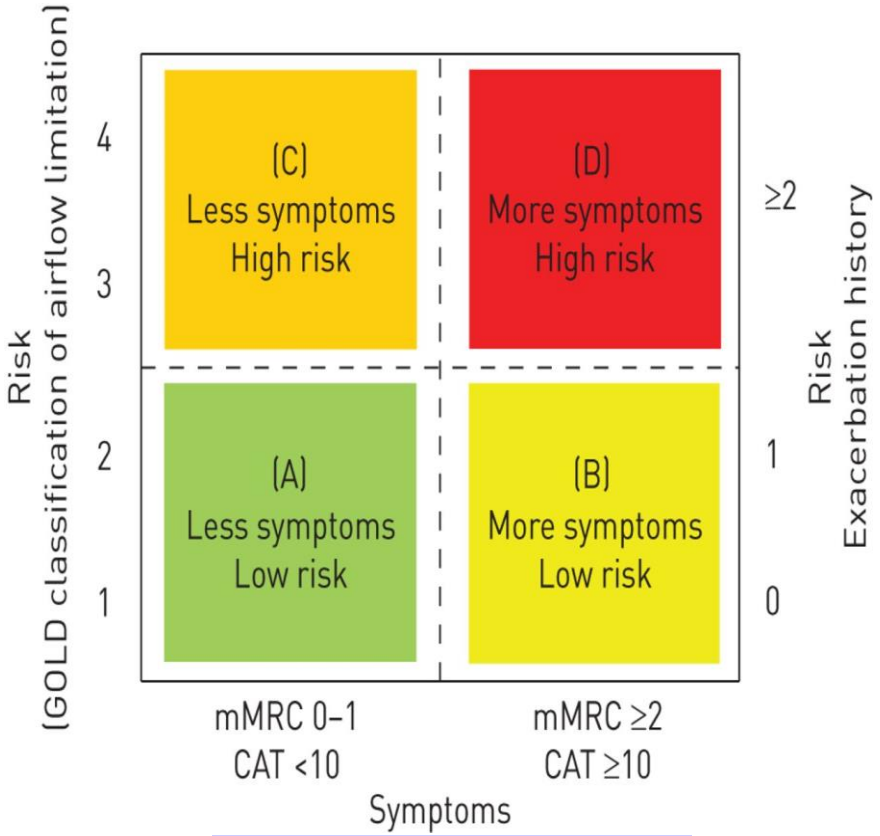
EXACERBATIONS

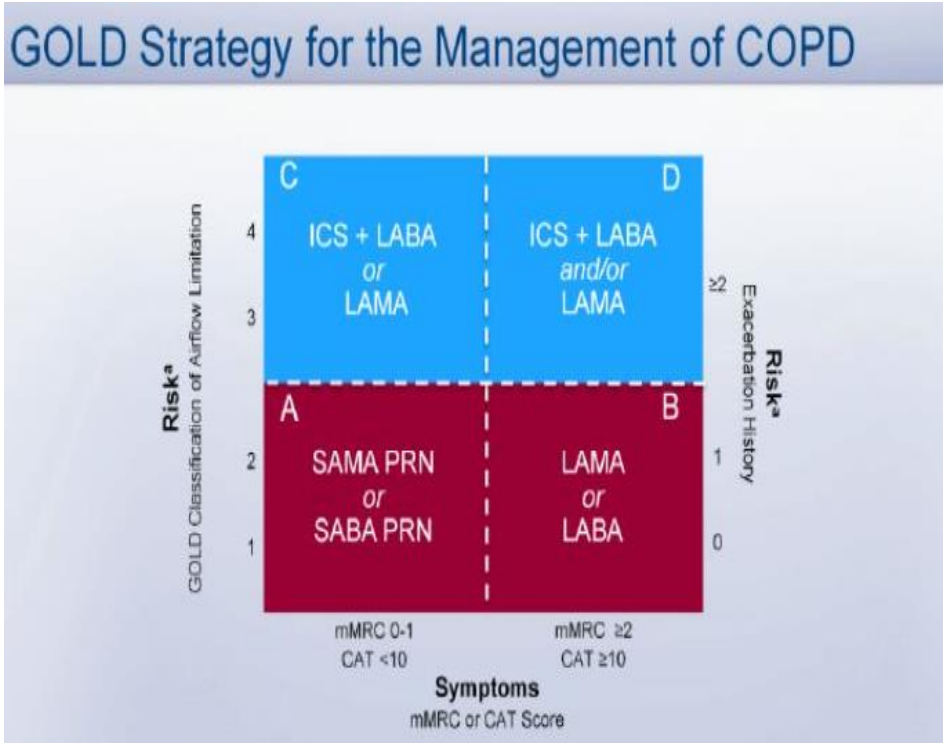


Number of Exacerbations:

- 0, 1
- ≥ 2

GOLD Guidelines 2011 – Management Strategy of COPD





Symptoms

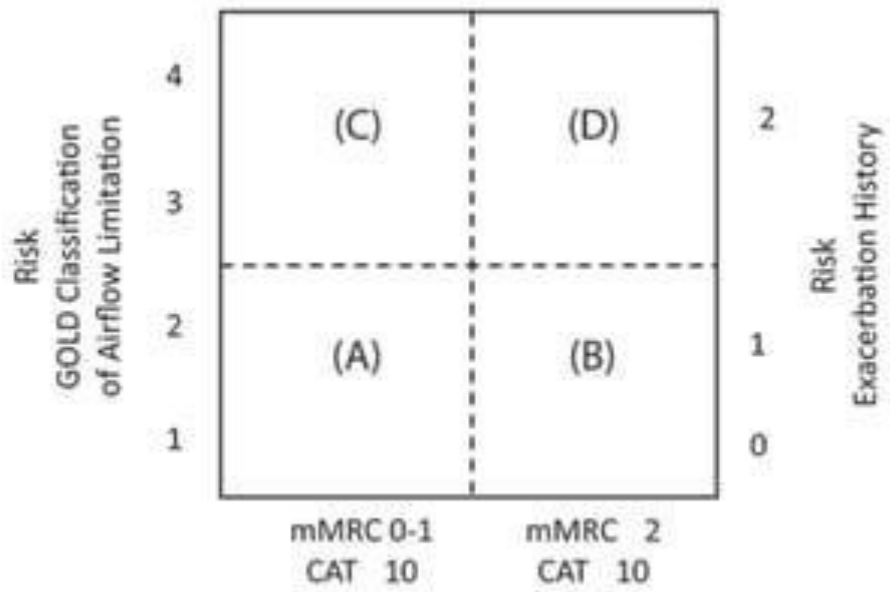
mMRC, CAT

Exacerbations

Number

Combined Assessment of COPD

When assessing risk, choose the highest risk according to GOLD grade or exacerbation history



Patient Category	Characteristics	Spirometric Classification	Exacerbations Per Year	mMRC	CAT
A	Low Risk, Less Symptoms	GOLD 1-2	1	0-1	<10
B	Low Risk, More Symptoms	GOLD 1-2	1	2	10
C	High Risk, Less Symptoms	GOLD 3-4	2	0-1	<10
D	High Risk, More Symptoms	GOLD 3-4	2	2	10

Therapeutic Options

- Smoking cessation has the greatest capacity to influence the natural history of COPD. Health care providers should encourage all patients who smoke to quit.
- Pharmacotherapy and nicotine replacement reliably increase long-term smoking abstinence rates.
- All COPD patients benefit from regular physical activity and should repeatedly be encouraged to remain active.
- Influenza and pneumococcal vaccination should be offered depending on local guidelines.

Ways to prevent or slow the progression of COPD

- **Stop smoking**, to prevent further damage to lungs
  - Smoking cessation is critical for all severities of COPD

- **Avoid or protect** from exposures to
  - Second-hand smoke

    and
  - Other substances such as chemical vapors, fumes, mists, dusts, and diesel exhaust fumes that irritate your lungs

Brief Strategies to Help the Patient Willing to Quit Smoking

- **ASK** Systematically identify all tobacco users at every visit
- **ADVISE** Strongly urge all tobacco users to quit
- **ASSESS** Determine willingness to make a quit attempt
- **ASSIST** Aid the patient in quitting
- **ARRANGE** Schedule follow-up contact.

Therapeutic Options: COPD Medications

Beta
Short-acting beta
Long-acting beta
Anticholinergics
Short-acting anticholinergics
Long-acting anticholinergics
Combination short-acting beta
Combination long-acting beta
Methylxanthines
Inhaled corticosteroids
Combination long-acting beta
Systemic corticosteroids
Phosphodiesterase-4 inhibitors

Therapeutic Options: Bronchodilators

- Bronchodilator medications are central to the symptomatic management of COPD.
- Bronchodilators are prescribed on an as-needed or on a regular basis to prevent or reduce symptoms.
- The principal bronchodilator treatments are beta$_2$ agonists, anticholinergics, theophylline or combination therapy.
- The choice of treatment depends on the availability of medications and each patient's individual response in terms of symptom relief and side effects.

Therapeutic Options: Bronchodilators

- Long-acting inhaled bronchodilators are convenient and more effective for symptom relief than short-acting bronchodilators.
- Long-acting inhaled bronchodilators reduce exacerbations and related hospitalizations and improve symptoms and health status.
- Combining bronchodilators of different pharmacological classes may improve efficacy and decrease the risk of side effects compared to increasing the dose of a single bronchodilator.

Therapeutic Options: Inhaled Corticosteroids

- Regular treatment with inhaled corticosteroids improves symptoms, lung function and quality of life and reduces frequency of exacerbations for COPD patients with an FEV_1 < 60% predicted.
- Inhaled corticosteroid therapy is associated with an increased risk of pneumonia.
- Withdrawal from treatment with inhaled corticosteroids may lead to exacerbations in some patients.

Therapeutic Options: Combination Therapy

- An inhaled corticosteroid combined with a long-acting beta$_2$-agonist is more effective than the individual components in improving lung function and health status and reducing exacerbations in moderate to very severe COPD.
- Combination therapy is associated with an increased risk of pneumonia.
- Addition of a long-acting beta$_2$-agonist/inhaled glucorticosteroid combination to an anticholinergic (tiotropium) appears to provide additional benefits.

Therapeutic Options: Systemic Corticosteroids

- Chronic treatment with systemic corticosteroids should be avoided because of an unfavorable benefit-to-risk ratio.

Therapeutic Options: Phosphodiesterase-4 Inhibitors

- In patients with severe and very severe COPD (GOLD 3 and 4) and a history of exacerbations and chronic bronchitis, the phospodiesterase-4 inhibitor, roflumilast, reduces exacerbations treated with oral glucocorticosteroids.

Therapeutic Options: Theophylline

- Theophylline is less effective and less well tolerated than inhaled long-acting bronchodilators and is not recommended if those drugs are available and affordable.
- There is evidence for a modest bronchodilator effect and some symptomatic benefit compared with placebo in stable COPD. Addition of theophylline to salmeterol produces a greater increase in FEV_1 and breathlessness than salmeterol alone.
- Low dose theophylline reduces exacerbations but does not improve post-bronchodilator lung function.

Therapeutic Options: Other Pharmacologic Treatments

Influenza vaccines can reduce serious illness. Pneumococcal polysaccharide vaccine is recommended for COPD patients 65 years and older and for COPD patients younger than age 65 with an $FEV_1 < 40\%$ predicted.

The use of antibiotics, other than for treating infectious exacerbations of COPD and other bacterial infections, is currently not indicated.

Oxygen therapy (LTOT)

LTOT may be indicated for those clients with chronic lung disease who meet **EITHER** of the following criteria:

i. Documented hypoxaemia i.e. daytime $PaO_2 \leq 55mmHg$, measured at rest via ABG whilst breathing room air when clinically stable **OR**

ii. Daytime PaO_2 56 - 59mmHg, measured at rest via ABG whilst breathing room air when clinically stable, with evidence of end-organ damage due to hypoxia e.g. pulmonary hypertension, right heart failure, polycythaemia

Therapeutic Options: Rehabilitation

- All COPD patients benefit from exercise training programs with improvements in exercise tolerance and symptoms of dyspnea and fatigue.
- Although an effective pulmonary rehabilitation program is 6 weeks, the longer the program continues, the more effective the results.
- If exercise training is maintained at home, the patient's health status remains above pre-rehabilitation levels.

Therapeutic Options: Rehabilitation

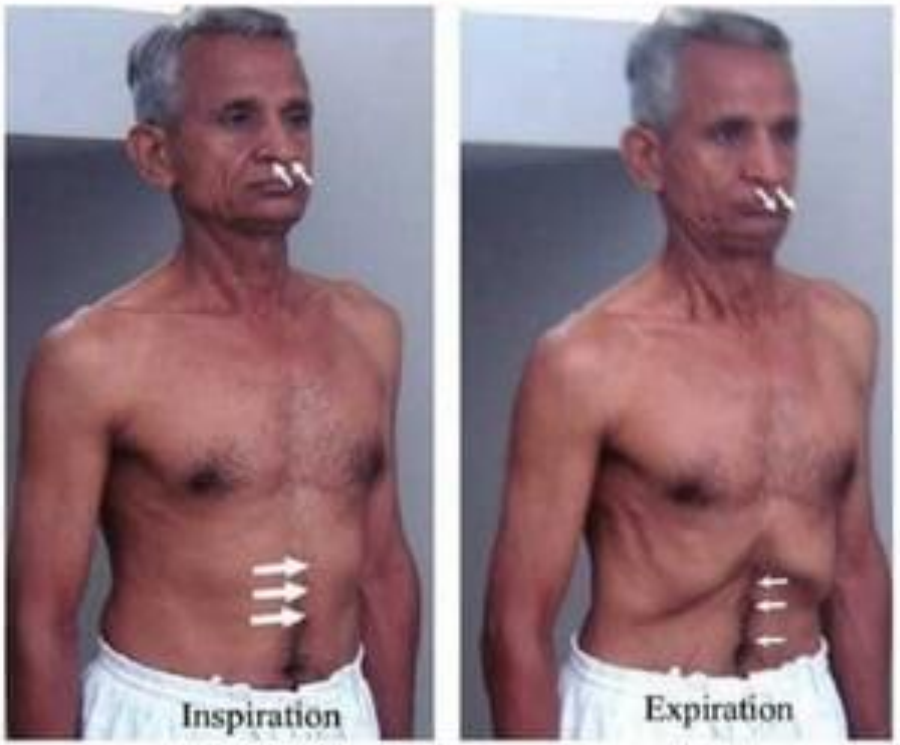


Fig. 2 : Diaphragmatic breathing: During inspiration diaphragm descends down and abdomen moves out. Patient exhales through nose with abdomen drawing in

Manage Exacerbations: Key Points

- The most common causes of COPD exacerbations are viral upper respiratory tract infections and infection of the tracheobronchial tree.
- Diagnosis relies exclusively on the clinical presentation of the patient complaining of an acute change of symptoms that is beyond normal day-to-day variation.
- The goal of treatment is to minimize the impact of the current exacerbation and to prevent the development of subsequent exacerbations.

Thank You!

Emphysema
Pathophysiology

- Small bronchioles become obstructed as a result of
  - Mucus
  - Smooth muscle spasm
  - Inflammatory process
  - Collapse of bronchiolar walls
- Recurrent infections → production/ stimulation of neutrophils and macrophages → release proteolytic enzymes → alveolar destruction → inflammation, exudate, and edema

Emphysema
Pathophysiology

- Elastin and collagen are destroyed
- Air goes into the lungs but is unable to come out on its own and remains in the lung
- Causes bronchioles to collapse
  - Trapped air → hyperinflation and overdistention
  - As more alveoli coalesce, blebs and bullae may develop
  - Destruction of alveolar walls and capillaries → reduced surface area for O_2 diffusion
  - Compensation is done by increasing respiratory rate to increase alveolar ventilation
  - Hypoxemia usually develops late in disease

Chronic bronchitis Pathophysiology

Pathologic lung changes are:

- Hyperplasia of mucus-secreting glands in trachea and bronchi
- Increase in goblet cells
- Disappearance of cilia
- Chronic inflammatory changes and narrrowing of small airways
- Altered function of alveolar macrophages ⟶ infections

- Greater resistance to airflow increases work of breathing

- Hypoxemia and hypercapnia develop more frequently in chronic bronchitis than emphysema

Chronic bronchitis Pathophysiology

Chronic inflammation

- **Primary pathologic mechanism causing changes**
- **Narrow airway lumen and reduced airflow d/t**
  - hyperplasia of mucus glands
  - Inflammatory swelling
  - Excess, thick mucus

- **Bronchioles are clogged with mucus and pose a physical barrier to ventilation**
- **Hypoxemia and hypercapnia d/t lack of ventilation and O_2 diffusion**
- **Tendency to hypoventilate and retain CO_2**
- **Frequently patients require O_2 both at rest and during exercise**

Medications used in the treatment of COPD

Table 2. Medications Used in the Treatment of COPD in the U.S.

Class	Drug (Brand)	Most Common Adverse Effects
Short-acting beta$_2$ agonists (SABAs)	Albuterol (ProAir, Proventil, Ventolin) Levalbuterol (Xopenex)	Palpitations, tachycardia, insomnia, irritability, tremors, hypokalemia
Long-acting beta$_2$ agonists (LABAs)	Formoterol (Foradil, Perforomist) Arformoterol (Brovana) Salmeterol (Serevent) Indacaterol (Arcapta)	Same as above
Short-acting anticholinergic (SAMA)	Ipratropium bromide (Atrovent)	Xerostomia, metallic taste
Long-acting anticholinergics (LAMAs)	Tiotropium (Spiriva) Alcidinium (Turdorza)	Xerostomia, metallic taste Headache, nasopharyngitis, cough
Combination: SABA + anticholinergic	Albuterol/ipratropium (Combivent)	Upper respiratory tract infections, headache
Methylxanthines	Aminophylline Theophylline (Theo-Dur, Theo-24)	Atrial and ventricular arrhythmias, grand mal convulsions, headache, nausea
Inhaled corticosteroids (ICS)	Beclomethasone (Qvar)[a] Budesonide (Pulmicort) Fluticasone (Flovent)	Oral candidiasis, skin bruising
Combination: LABA + ICS	Formoterol/budesonide (Symbicort) Salmeterol/fluticasone (Advair) Formoterol/mometasone (Dulera)[a]	Headache, nasopharyngitis, stomach discomfort
Phosphodiesterase-4 (PDE4) inhibitor	Roflumilast (Daliresp)	Nausea, weight loss, diarrhea, headache, abdominal pain

[a] *Currently FDA approved only for asthma. COPD: chronic obstructive pulmonary disease.*
Source: References 4, 10.

Definition: National Heart, Lung and Blood institute (NHLBI) and WHO

- COPD is a group of chronic respiratory disorders that lead to progressive tissue degeneration and obstruction in the airways and lungs that is not fully reversible.
- They are debilitating conditions that affect the individual's ability to work and function independently.
- These include; Chronic bronchitis, asthmatic bronchitis and emphysema. May coexist. COPD may coexist with asthma.

Definition: GOLD

COPD is a preventable and treatable disease with significant extra-pulmonary effects that may contribute to severity in individual patients. Its pulmonary component is characterized by airflow limitation that is non-reversible. The airflow resistance is normally progressive and is associated with an abnormal inflammatory response of the lung to noxious particles or gas.

The GOLD definition does not use the term bronchitis or emphysema and excludes asthma (reversible air flow limitation)

Chronic Bronchitis

- Recurrent excessive mucus secretion into the bronchial tree resulting in airway obstruction due to edema and bronchial inflammation.
- Patient has a cough producing more than 30ml of sputum in 24hrs for at least 3 months of the year, for 2 consecutive years.
- Sputum is thick and obstructs airflow.

Emphysema

- A condition of the lung characterized by abnormal, permanent enlargement of the airspaces distal to the terminal bronchioles, accompanied by destruction of their walls and without any obvious fibrosis. There is a lack of uniformity in airspace enlargement, resulting in loss of alveolar surface areas.

Emphysema

- It causes destruction of the alveolar walls and capillaries by increased lung enzymatic activity.
- May include; centricinar, panacinar and distal acinar.

Chronic Asthma

- Characterized by recurrent, (intermittent) reversible bronchspasms.
- Reversibility may be spontaneous or after drugs therapy.
- Airway inflammation and hyper-responsiveness to a variety of stimuli are important components of asthma.

Risk factors: Major

- Worldwide cigarette smoking is the most common encountered risk.
- The best documented genetic risk factor is a severe alpha 1 trypsin deficiency.
- Occupational hazards and indoor pollution (biogas burning).
- Age.
- Male gender.
- Existing impaired lung function.

Risk factors: Minor

- Air pollution.
- Alcohol.
- Race.
- Nutritional status.
- Family history.
- Bronchial reactivity.

Pathophysiology; Chronic Bronchitis

- Inhalation of noxious particles and gases stimulates the activation of neutrophils, macrophages and CD8+ lymphocytes which release a variety of chemical mediators, including tumor necrosis factor alpha(TNFa), interleukin-8 (IL8), and leukotriene B4 (LTB4).
- respiratory tissue inflammation results in vasodilation, congestion, mucosal edema and globlet cells hypertrophy. These events trigger globlet cell production of excessive amount of mucus.

Pathophysiology; Chronic Bronchitis

- Changes in tissues include increased smooth muscle cartilage atrophy, infiltration of neutrophils and other cells and impairment of the cilia.
- Normally sterile airways become colonized with strep. Pneumoniae, H. influenza, RSV, Moraxella catarrhalis and mycoplasma species, Recurrent infections (viral and bacterial) reduce ciliary and phagocytic activity, increase mucus accumulation, weaken the body's defenses and further destroy small bronchioles.

Pathophysiology; Chronic Bronchitis

- The airway degenerate and overall gas exchange is impaired, causing exertional dypnea.
- Hypoxemia results in increasing PaC02. If this is sustained, the brain's respiratory control center and central chemoreceptor are desensitized and compensatory action to correct hypoxemia does not occur.

Pathophysiology; Emphysema

- anatomical changes of airways because of the loss of tissue elasticity.
- Inflammation and excessive mucus secretion cause airway trapping in the alveoli. This contributes to breakdown of the bronchioles, alveolar walls and connective tissue.
- Clusters of alveoli merge and the number of alveoli diminishes, leading to increased space available for air trapping.

Pathophysiology; Emphysema

- Destruction of airway walls causes collapse of small airways on exhalation and disruption of pulmonary capillary bed.
- Hypercapnea and respiratory acidosis are uncommon in emphysema because breathing imbalance is compensated for by increased in respiratory rate

Clinical assessment

- Chronic bronchitis typically has an insidious onset after age 45
- Emphysema usually seen in the 50's
- Chronic productive cough is the hallmark of chronic bronchitis.
- Dyspnea, lung infiltration, increased respiratory effort, altered breathing patterns, abnormal breath sounds including wheezing and diminished breath sounds.
- Progressive and incomplete airflow obstruction.

Laboratory Test.

- chest X-ray
- PFT (assess pre and post bronchidilators) – decreased FEV1 and FVC
- ABG – hypoxemia, hypercarbia and acidemia
- Hct/Hgb – erythrocytosis
- ECG-RVH
- Cultures

Classification of COPD by severity.

Stage	Lung Function
Stage 1: Mild COPD	FEV1/FVC < 70% FEV1 ≥ 80% predicated
Stage 2: Moderate COPD	FEV1/FVC < 50 - 70% FEV1 < 80% predicated
Stage 3 Severe COPD	FEV1/FVC < 30 - 70% FEV1 < 50% of pred.
Stage 4: Very Severe COPD	FEV1/FVC < 70% FEV1 ≤ 30% predicated or FEV1 is < 50% predicated and respiratory failure

Differentiating Characteristics

Chronic Bronchitis	**Emphysema**
Overweight (blue bloater)	Thin (pink puffer)
Mild Dyspnea	Severe Dyspnea
Copious Sputum	Scanty Sputum
Frequent Infections	Less frequent Infections
Hypoxemia	Hypoxemia uncommon
Barrel chest	Flattened Diaphragm
Cor pulmonale	Diffusion capacity decreased

Factors Determining Severity of Chronic COPD.

- Severity of symptoms
- Severity of airway limitation
- Frequency and severity of exacerbations
- Presence of complications of COPD
- Presence of respiratory insufficiency
- Comorbidity
- General health status
- Number of medications needed to manage the disease

Components of COPD Management.

- Assess and monitor disease
- Reduce risk factors
- Manage stable COPD
  - -Education
  - -Non-pharmacologic
  - -Pharmacologic
- Manage exacerbation

Goals of Therapy

- induce bronchodilation
- facilitate expectoration
- limit the impact of the disease on daily activities
- prevent complications
- smoking cession and avoidance of irritants

Goals of Therapy

- control life threatening disease exacerbations
- prevent complications
- teach patients about disease and the use of medications and improve therapeutic compliance

Non-Pharmacological Management. (NPT)

- Discontinue smoking
- Chest Physiology
- Breathing exercises
- Rehabilitation
- Preventative measures.

Discontinue Smoking

- most effective strategy to reduce the risk of developing COPD and affect the long-term rate of decline in FEV1.
- Group therapy
- Drugs (Chantrix; varenicline), nicotine replacement.
- Hypnosis

Chest Physiology

- Postural drainage
- Chest percussion and vibration
- Use if > 30 cc sputum/day

Breathing Exercises

- Inspire slowly and expire through pursed lips
- Inspiratory muscle training
- Breathing retraining

Rehabilitation

- Exercise reconditioning – increase endurance, exercise tolerance, maximal oxygen consumption
- Energy conservation
- Nutrition
- Psychosocial management – anxiety, depression and problems with cognitive perceptual and motor activity; financial and social resources

Preventative Measures

- Vaccines – annual influenza vaccine. Protection rate is 60-80 %
- Pneumococcal vaccine – recommended for patients with COPD, revaccination for patients>65 years if vaccination is >5 years
- Amantadine – efficacy is 50-90% 100mg bid for ages ≤65mg. 100mg/day for ages > 65 years. Useful in non-immunized but exposed patients

Pharmacological Management of COPD

- Oxygen
- Anticholinergics
- Short acting Beta Agonists (SABA)
- Long Acting Beta Agonist (LABA)
- Combination therapy (CT)
- Theophylline
- Oral Steroids
- Inhaled steroids (IC)
- Antibiotics

Oxygen Therapy

- Administration of oxygen has been shown to increase survival and improve QOL.
- Oxygen can reverse hypoxemia, increase body weight, ameliorate right heart failure and improve exercise tolerance.

Oxygen Therapy

Oxygen is needed for:

- Pt with PaO2 is < 55mmHg or Sa02 < 88%
- Pt with cor pulmonale or CHF (PaO2 is < 55mmHg or Sa02 < 89%)
- FEV1/FVC<70%; FEV1 <30% or presence of chronic respiratory failure or right heart failure.
- Specific situations: lung disease e.g. sleep apnea with nocturnal symptoms not corrected by continuous positive airway pressure

Anticholinergics (e.g. Ipratropium Bromide, Atropine, Glycopyrrolate, Tiotropium bromide

- Considered 1st line bronchodilators in the treatment of COPD
- COPD patients are very responsiveness to anticholinergics.
- Studies have shown equivalent and in some cases more effective than beta agonists in patients with chronic bronchitis and emphysema.
- Generally safe with less cardiovascular S/Es than high doses of beta agonists

MOA

- Ipratropium and atropine produce bronchodilation by competitively inhibiting cholinergic responses. They inhibit cyclic guanosine monophosphate leading to relaxation of bronchial smooth muscles
- Ipratropium also reduces sputum volume without altering viscosity
- Onset of action is 15mins (vs 5 mins for SABB ; peaks in 60-90 mins, has 6 hrs duration.

MOA

- Tiotropium is a long acting agent that protects against bronchoconstriction for >24hours.
- Onset is 30 mins; peak is 3 hours.
- It blocks M1, M2 & M3 receptors but dissociates quickly from M2 which may be responsible for rebound bronchoconstriction through the release of acethylcholine. Ipatropium in contrast binds to M3 over a prolonged period.
- It is delivered in a handihaler, a single load dry-powdered, breath activated device

Dosing

- Ipratropium MDI – 2 inhalations (40 mcg) qid. May be increased to 6 inhalations qid daily. Use spacer with closed mouth technique.
- Ipratropium Solution – 500mcg/2.5ml or more via nebulizer qid
- Tiotropium; the recommended dose is the inhalation of the contents of one capsule once daily
- using the handihaler.
- It is well tolerated with dry mouth the most common side effect.

Short acting Beta Agonist (salbutamol, albuterol

- Symptomatic benefits, but not as marked as obtained in asthma
- Reserved for prn use
- Inhaled agents preferred
- Can be used to assess/monitor patient's current therapy
- Use to determine reversibility on spirometry
- S/Es generally seen with high doses (include palpitation, shakiness

MOA / DOSE

- cause bronchodilation of bronchial smooth muscles. May increase mucociliary clearance by stimulating ciliary activity
- Dose: MDI 2-4 puffs q 20 mins for up to 4 hours, then every 4 hours as needed.
- Nebulization; 2.5mg diluted to a total of 3ml, 3-4 times/day over 5-15 minutes.

Long Acting Beta Agonist; (salmeterol, formeterol)

- approved for use in COPD since 1997
- have positive effect on QOL (one inhalation bid dosing)
- used as an add-on therapy where combination bronchodilator therapy is not adequate, patients with night time symptoms or patients with difficulty complying.
- Slow onset of action and expensive

Combination Therapy (Ipatropium/Albuterol)

- The combination ofan inhaled anticholinergic &beta 2 agonist often is used as the disease progresses and symptoms worsen over time.
- Combination of bronchodilators with different MOA allows lowest effective doses to be used and reduces side effects.
- Superior to either albuterol or Ipratropium alone.
- more convenient but harder to adjust therapy.

Theophylline

- 2nd or 3rd line therapy
- potential action include:
- bronchodilation
- increase mucocillary clearance
- increase respiratory drive (effective short term)
- improved cardiovascular function – increase RVEF and LVEF
- increase diaphragmatic contractility
- improve exercise capacity

Dosing

- Acceptable plasma concentration is 8 - 12 mcg/ml and maximized if necessary up to 20mcg/ml
- Determine plasma concentration prior to dosing
- LD 3mg/kg aminophylline if prior theophylline within last 24 hrs and 6 mg/kg if none, given over 30mins
- Maintenance infusion of 0.4 mg/kg/hr
- Precautions: multiple S/Es, altered metabolism (smokers, CHF, elderly, liver disease), a lot of drug interactions.

Oral Steroids

- Only about 10% of patients benefit from oral steroids and there is high risk of dependence and side effects
- Can be used long –term if an objective benefit is seen after adequate trial – Prednisone 30mg qd x 2 weeks or more
- Some studies show that 50% of patients responding to oral steroids will respond to inhaled steroids.
- IV Methylprednisolone 50-100 mg q6-8h can be used in acute exacerbations. Taper as rapidly as possible

Inhaled steroids

- appropriate for symptomatic COPD patients with FEV1 < 50% predicted (stage 3 and stage 4) and repeated exacerbations
- treatment has been shown to reduce frequency of exacerbations and improve health status.

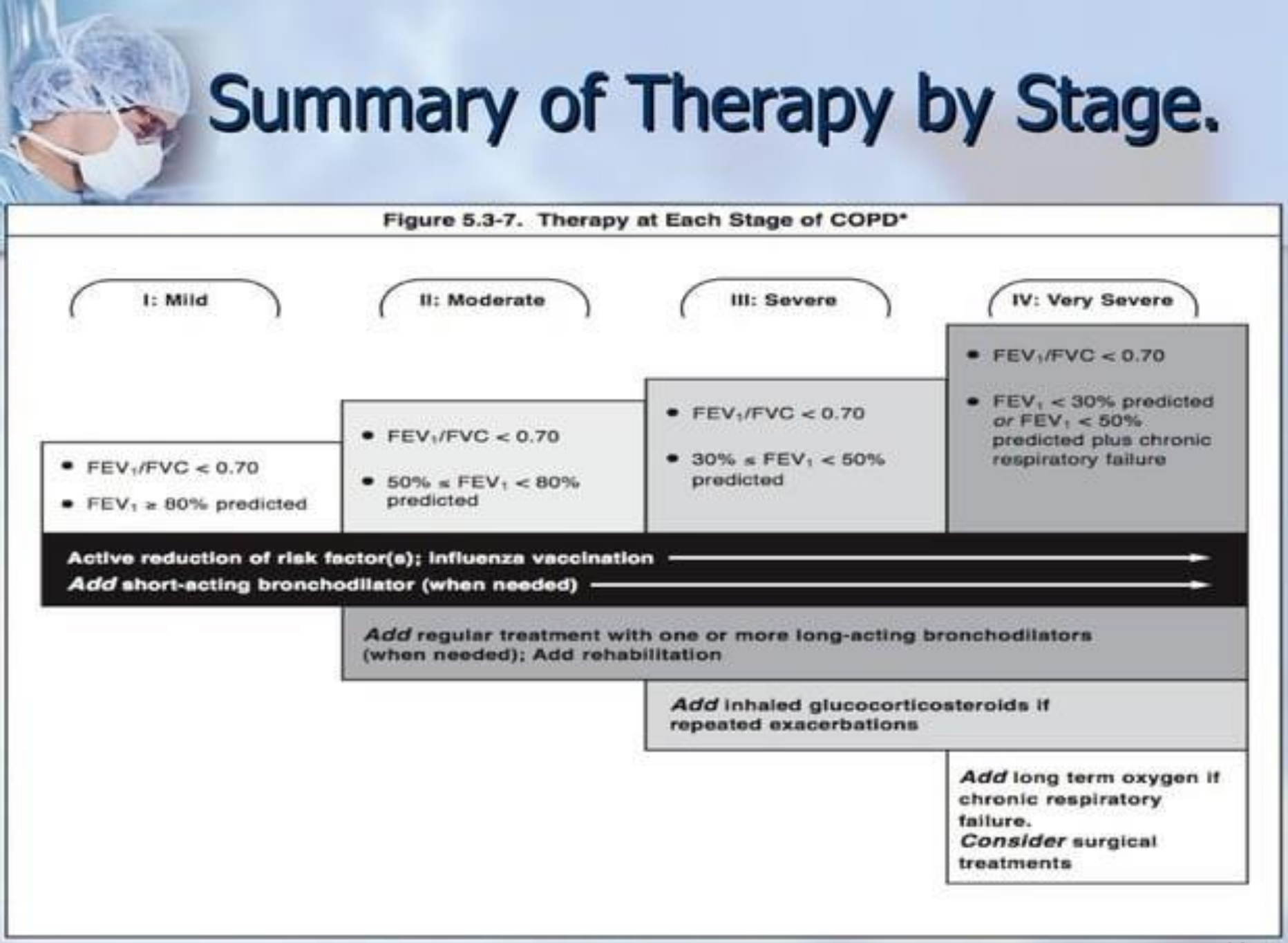
Antibiotics

- indicated if purulent sputum, infiltrates on chest x-ray or positive gram stain (usually pneumococcus or H. influenza)
- should be initiated within 24 hours of symptoms
- goal of therapy is to shorten the duration of exacerbations and prevent deterioration

Antibiotics

- Empirically treat for H. influenza (59%), S pneumoniae (17%) and M Catarrhalis (12%)
- Alternatives: Amoxicillin, Cephalosporins or TMX/SMX or Ampicillin
- Macrolides are also good but more expensive
- Treat for 7-10 days

Summary of Therapy by Stage.



Complications of COPD

- Pulmonary Hypertension
- Acute Respiratory Failure
- Infections
- Polycethemia.

Take Home Points/ Summary.

- COPD is a preventable disease that can be managed to reduce exacerbations and improve QOL for patients.
- It is a progressive generally irreversible inflammatory lung disease.
- It includes chronic bronchitis, emphysemia and less significantly, chronic asthma.
- Smoking is the major risk factor worldwide.

Take Home Points/ Summary.

- COPD is staged according to lung function. A definitive diagnosed is made by spirometry. Hallmark is FEV1/FVC ratio<70%.
- A major goal of therapy is symptom improvement and a reduction in the rate of FEV1 decline.
- Mild COPD maybe treated with NPT + SABA.
- Moderate COPD add LABA and/or CT.

Take Home Points/ Summary.

- Severe COPD add IC if repeated exacerbations.
- Oral steroids show little benefit and increases toxicity. Chronic use should be avoided.
- Long term Oxygen therapy is given in cases of acute respiratory failure.
- The Pharmacist has an important role in the management of COPD; to educate, to prevent exacerbations, hospitalization, development of respiratory failure and death.

HEALTH EDUCATION

- Wash hands often with soap and water for 20 seconds or use an alcohol based hand sanitizer
- Cover your nose and mouth with a tissue when you cough and sneeze
- Avoid touching your eyes, nose and mouth with unwashed hand
- Avoid personal contact with sick people
- Cleanse and disinfect frequently touched surfaces and objects such as doorknobs.

e-cigarettes

The latest study on e-cigarettes, published in the journal Lancet, supports that claim. In the first clinical trial comparing e-cigarettes and nicotine patches in helping people to quit smoking, both methods proved equally successful. After a 13-week smoking cessation program, similar numbers of smokers who used e-cigarettes remained smoke-free after six months as used nicotine patches.

Table 1. Clinical guidelines for initiation of antibiotics in AECOPD

Global Initiative for Chronic Obstructive Lung Disease[3]	Antibiotics should be given to: 1. Patients with all three cardinal symptoms (increased dyspnea, increased sputum volume, increased sputum purulence) 2. Patients with two cardinal symptoms, if increased sputum purulence is present 3. Patients with severe exacerbations requiring noninvasive or invasive mechanical ventilation
American Thoracic Society/European Respiratory Society[4]	1. In hospitalized, non-ICU patients, antibiotics may be initiated in patients with changes in sputum characteristics 2. In patients requiring ICU admission, antibiotic therapy is recommended
National Institute for Health and Clinical Excellence[5] (United Kingdom)	1. Antibiotics should be used in exacerbations associated with a history of more purulent sputum 2. Patients with exacerbations who do NOT have increased sputum purulence do not need antibiotics unless there are signs of pneumonia
Canadian Thoracic Society[6]	1. Antibiotics may be beneficial in the treatment of more severe purulent AECOPD

Indications for Hospitalization

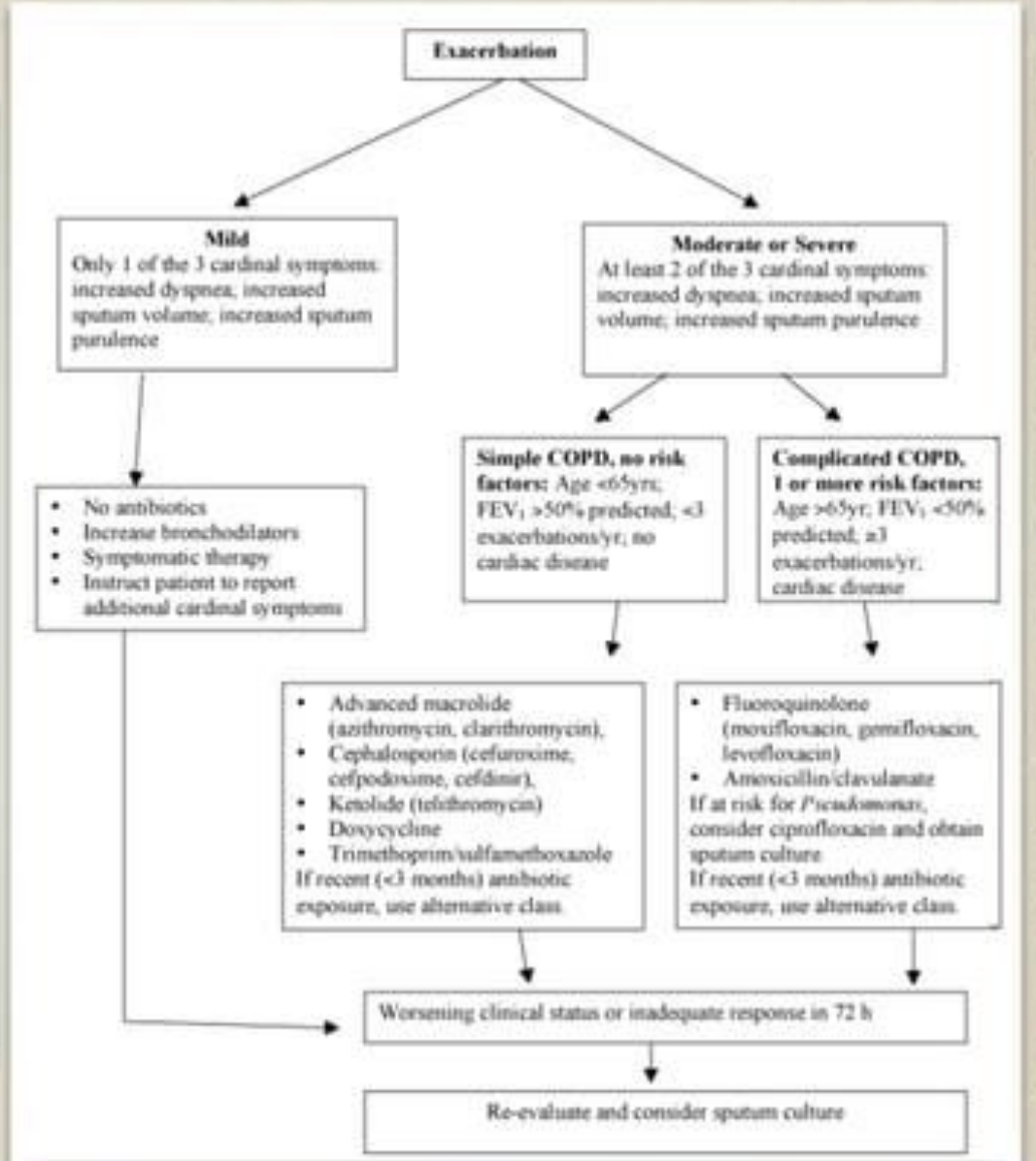
Hospital Room

Marked increase in symptom intensity[a]
Severe COPD
Onset of new physical signs
No response to outpatient management
Significant comorbidities[b]
Frequent exacerbations
Diagnostic uncertainty
Older age
Insufficient home support

Intensive Care Unit

Severe dyspnea not responsive to emergency therapy
Changes in mental status
Hypoxemia, hypercapnia, and/or acidosis despite supplemental oxygen and NIV
Need for invasive ventilation
Hemodynamic instability

[a] Sputum changes, dyspnea, chest tightness, malaise, fatigue, decreased exercise tolerance, fever, wheezing, decreased breath sounds, increased need for bronchodilators.
[b] Pneumonia, arrhythmia, congestive heart failure, diabetes, renal or hepatic failure.
COPD: chronic obstructive pulmonary disease; NIV: noninvasive ventilation.



Manage Stable COPD: Goals of Therapy

- Relieve symptoms
- Improve exercise tolerance
- Improve health status

Reduce symptom

- Prevent disease progression
- Prevent and treat exacerbations
- Reduce mortality

Reduce risk

Manage Stable COPD: Non-pharmacologic

Patient Group	Essential	Recommended	Depending on local guidelines
A	Smoking cessation (can include pharmacologic treatment)	Physical activity	Flu vaccination Pneumococcal vaccination
B, C, D	Smoking cessation (can include pharmacologic treatment) Pulmonary rehabilitation	Physical activity	Flu vaccination Pneumococcal vaccination

Manage Stable COPD: Pharmacologic Therapy

(Medications in each box are mentioned in alphabetical order, and therefore not necessarily in order of preference.)

Patient	Recommended First choice	Alternative choice	Other Possible Treatments
A	*SAMA prn* *or* *SABA prn*	*LAMA* *or* LABA *or* *SABA and SAMA*	*Theophylline*
B	***LAMA*** ***or*** ***LABA***	***LAMA and LABA***	***SABA and/or SAMA*** ***Theophylline***
C	*ICS + LABA* *or* *LAMA*	*LAMA and LABA or* *LAMA and PDE4-inh. or* *LABA and PDE4-inh.*	*SABA and/or SAMA* *Theophylline*
D	*ICS + LABA* *and/or* *LAMA*	*ICS + LABA and LAMA or* *ICS+LABA and PDE4-inh. or* LAMA and LABA *or* *LAMA and PDE4-inh.*	*Carbocysteine* *SABA and/or SAMA* *Theophylline*

Assessment of acute exacerbation of COPD

ABG	PaO breathing room air indicates respiratory failure.
CXR	useful to exclude alternative diagnoses.
CBC	identify polycythemia, anemia or bleeding.
Biochemical tests	detect electrolyte disturbances, diabetes, and poor nutrition.
Sputum	Purulent sputum indicates the need for antibiotics
Biochemical tests	detect electrolyte disturbances, diabetes, and poor nutrition.
ECG	may aid in the diagnosis of coexisting cardiac problems.
Spirometry	not recommended during an exacerbation.
Serum troponin, serial CK-MB	Assess for myocardial injury or infarction Serum troponin, serial CK-MB measurement

Manage Exacerbations: Treatment Options

Oxygen	titrate to improve the patient's hypoxemia with a target saturation of 88-92%
Bronchodilators	Short-acting inhaled beta short-acting anticholinergics are preferred.
Systemic Corticosteroids	Shorten recovery time, improve lung function (FEV hypoxemia (PaO failure, and length of hospital stay. A dose of 40 mg prednisone per
Magnesium sulphate	Nebulized magnesium as an adjuvent to salbutamol treatment in the setting of acute exacerbations of COPD has no effect on FEV
Antibiotics	Should be given when indicated
NIV	for patients hospitalized with severe exacerbation of COPD

Antibiotic Recommendations

Patient Characteristics	Pathogens	Therapy (no particular order)
Uncomplicated exacerbations <4 per year No comorbidities FEV_1 >50% predicted Resistance uncommon	*Streptococcus pneumoniae* *Haemophilus influenzae* *Moraxella catarrhalis* *Haemophilus parainfluenzae*	Azithromycin or clarithromycin 2nd- or 3rd-generation cephalosporin Doxycycline
Complicated exacerbations Age ≥65 years >4 per year FEV_1 36%-49% predicted	Above plus drug-resistant pneumococci, beta-lactamase-producing *H influenzae*, and *M catarrhalis*	Amoxicillin/clavulanate Fluoroquinolone with enhanced pneumococcal activity (levofloxacin, gemifloxacin, or moxifloxacin)
Complicated exacerbations with risk of Pseudomonas aeruginosa Chronic bronchial sepsis[b] Chronic corticosteroids Nursing home resident >4 per year FEV_1 >35% predicted	Enteric gram-negatives Above plus *P aeruginosa*	Levofloxacin[a] Possible IV therapy: beta-lactamase-resistant penicillin with antipseudomonal activity 3rd- or 4th-generation cephalosporin with antipseudomonal activity

[a] Fluoroquinolone with enhanced pneumococcal/antipseudomonal activity.
[b] In sepsis, addition of aminoglycoside considered for double antipseudomonal coverage.
FEV_1: forced expiratory volume in one second.

Indications for Invasive Mechanical Ventilation

Absolute indications	Relative indications
Cardiac or respiratory arrest	Severe dyspnea with use of accessory muscles
Failure of noninvasive ventilation or presence of exclusion criteria	Respiratory rate >35 breaths/min
Persistent hypoxemia (PaO_2<40 mm Hg) despite receiving optimal treatment	Vascular complications (hypotension, shock, heart failure)
Worsening of respiratory acidosis (pH<7.25) despite receiving optimal treatment	Other complications (severe pneumonia, pulmonary thromboembolism, etc)

HOSPITAL MANAGEMENT OF ACUTE EXACERBATIONS OF COPD

Definition
COPD is characterised by airflow obstruction, usually progressive, not fully reversible and not changing markedly over several months. It is predominantly caused by smoking

Severity of Airflow Obstruction
FEV_1: 50 – 80% predicted = **Mild**; FEV_1: 30 – 49% predicted = **Moderate**; FEV_1: < 30% predicted = **Severe**

Exacerbation characteristics include some or all of these:
- ↑ Dyspnoea
- ↑ Sputum volume & purulence
- ↑ Cough
- ↑ Wheeze / chest tightness
- ↑ Fatigue
- ↑ Fluid retention
- ↓ GCS
- ↓ Exercise Tolerance

Differential Diagnosis in COPD patients includes:
- Pneumonia
- Pneumothorax
- Pulmonary Oedema
- Lung Cancer
- Upper Airway Obstruction
- Pleural Effusion

Initial Assessment
- CXR
- ABG – note inspired FiO_2
- ECG
- FBC / U&Es
- Theophylline level if on it
- Sputum M C&S if purulent
- Blood cultures if pyrexial

Standard Therapy
- ↑ dose of bronchodilators
- Oral Prednisolone 30mg daily for 7-14 days
- Oxygen to maintain SaO_2 >90% but < 93%
- Antibiotics if sputum more purulent and / or pneumonia
- IV Theophylline if inadequate response to regular nebulised bronchodilators
- Physiotherapy using PEEP to help clear sputum when necessary
- No need for daily peak flow unless asthma suspected

Consider Hospital at Home or assisted early discharge if:
- Able to cope / good social circumstances / telephone
- Mild dyspnoea
- General condition and level of activity satisfactory
- Not on LTOT
- No confusion
- Onset not rapid
- No CXR consolidation
- pH > 7.35
- PO_2 > 7kPa

Indicators of a Severe Exacerbation
- Marked dyspnoea / tachypnoea
- Use of accessory respiratory muscles (sternomastoid and abdominal) at rest
- Pursed lip breathing
- New onset cyanosis
- New onset peripheral oedema
- Marked reduction in activities of daily living
- Acute confusion

In Severe Exacerbation
- NIV for persistent hypercapnic respiratory failure despite optimal medical therapy
- NIV should be delivered in dedicated setting with trained staff
- IV respiratory stimulants only where NIV not available
- Ceilings of treatment should be agreed
- Referral for consideration of intubation and ventilation in cases of progressive exhaustion and worsening ABGs
- Evidence of advanced directive should be sought

Maintenance management
- SaO_2 monitoring in non-hypercapnic, non acidotic respiratory failure
- Otherwise check ABGs intermittently and prior to discharge
- Re-establish optimal inhaled therapy pre discharge including checking inhaler technique
- Check BMI and spirometry pre discharge
- If still requiring O_2 arrange O_2 cylinder or interim LTOT on discharge, pending formal assessment when stable
- Consider pulmonary rehabilitation referral
- Provide written guided self-management plan to include advice on acute exacerbations
- Confirm 'flu' vaccine plans and home care support
- Consider follow up by respiratory nurse team in 4-6 weeks for those patients considered more vulnerable to early readmission

The BTS COPD CONSORTIUM