DYSPNEA

INTRODUCTION

WHAT IS DYSPNOEA?

Dyspnea derives from Greek for "**hard breathing**". It is often also described as "**shortness of breath**". This is a subjective sensation of breathing, from mild discomfort to feelings of suffocation.

It is a sign of a variety of disorders and is primarily an indication of **inadequate ventilation** or of **insufficient amounts of oxygen in the circulating blood**.

INTRODUCTION

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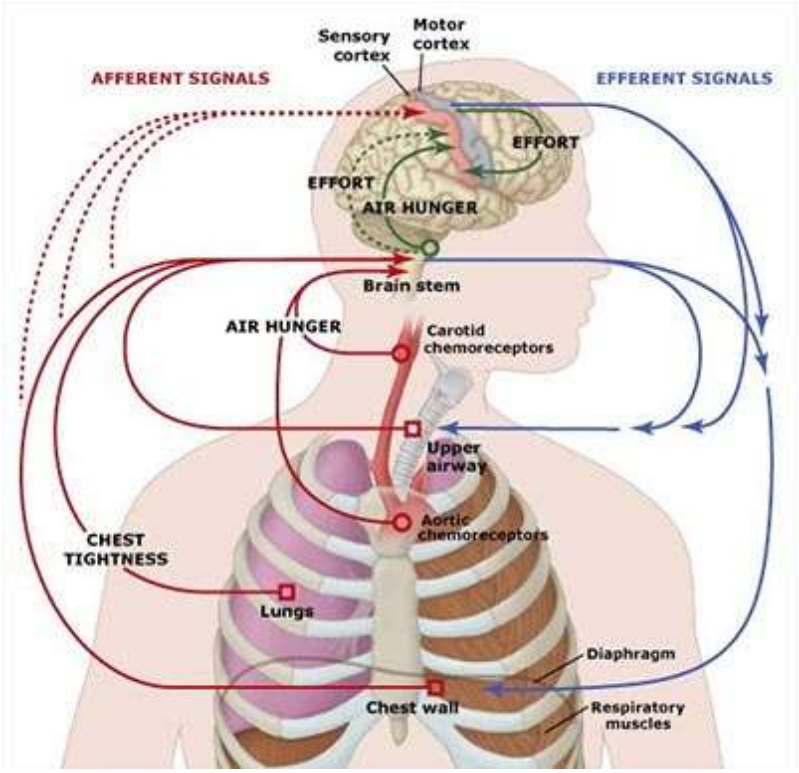
PATHOPHYSIOLOGY



Dyspnea happens when a “mismatch” occurs between afferent and efferent signaling.

As the brain receives afferent ventilation information, it is able to compare it to the current level of respiration by the efferent signals.

If the level of respiration is inappropriate for the body’s status and need, then dyspnea might occur

PATHOPHYSIOLOGY

RECEPTORS AND SIGNALS

The pathway that leads to dyspnea via specific acid-sensing ion channels, mechanoreceptors and lung receptors located in different zones of the respiratory apparatus.

Chemoreceptors
In the carotid bodies and medulla supply information with regard to the blood gas levels of O_2, CO_2 and H^+

Juxtacapillary receptors
Sensitive to pulmonary interstitial oedema

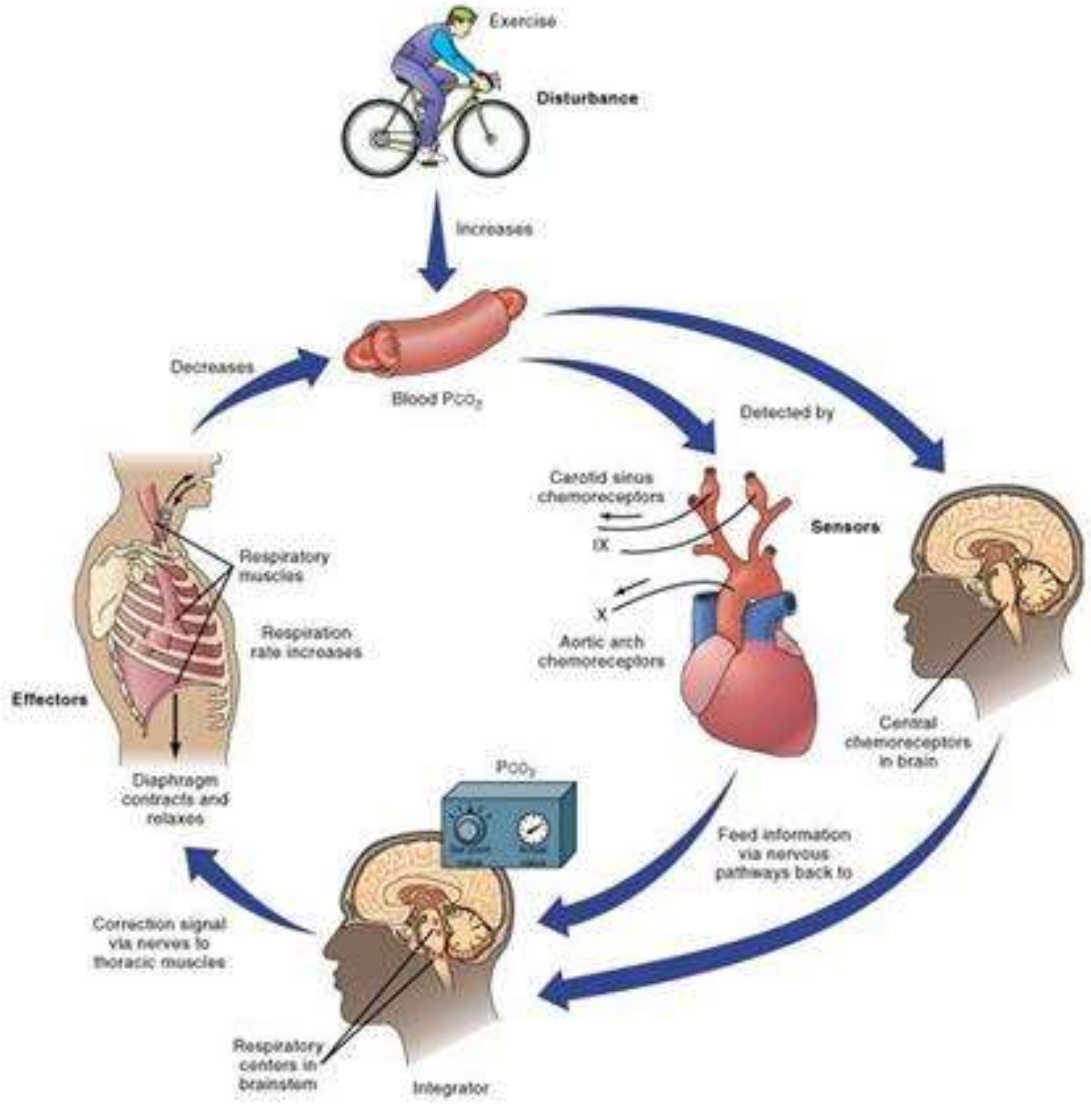
Stretch receptors
Hering-breuer reflex

Muscle spindles in the chest wall
Signals the stretch and tension of the respiratory muscles

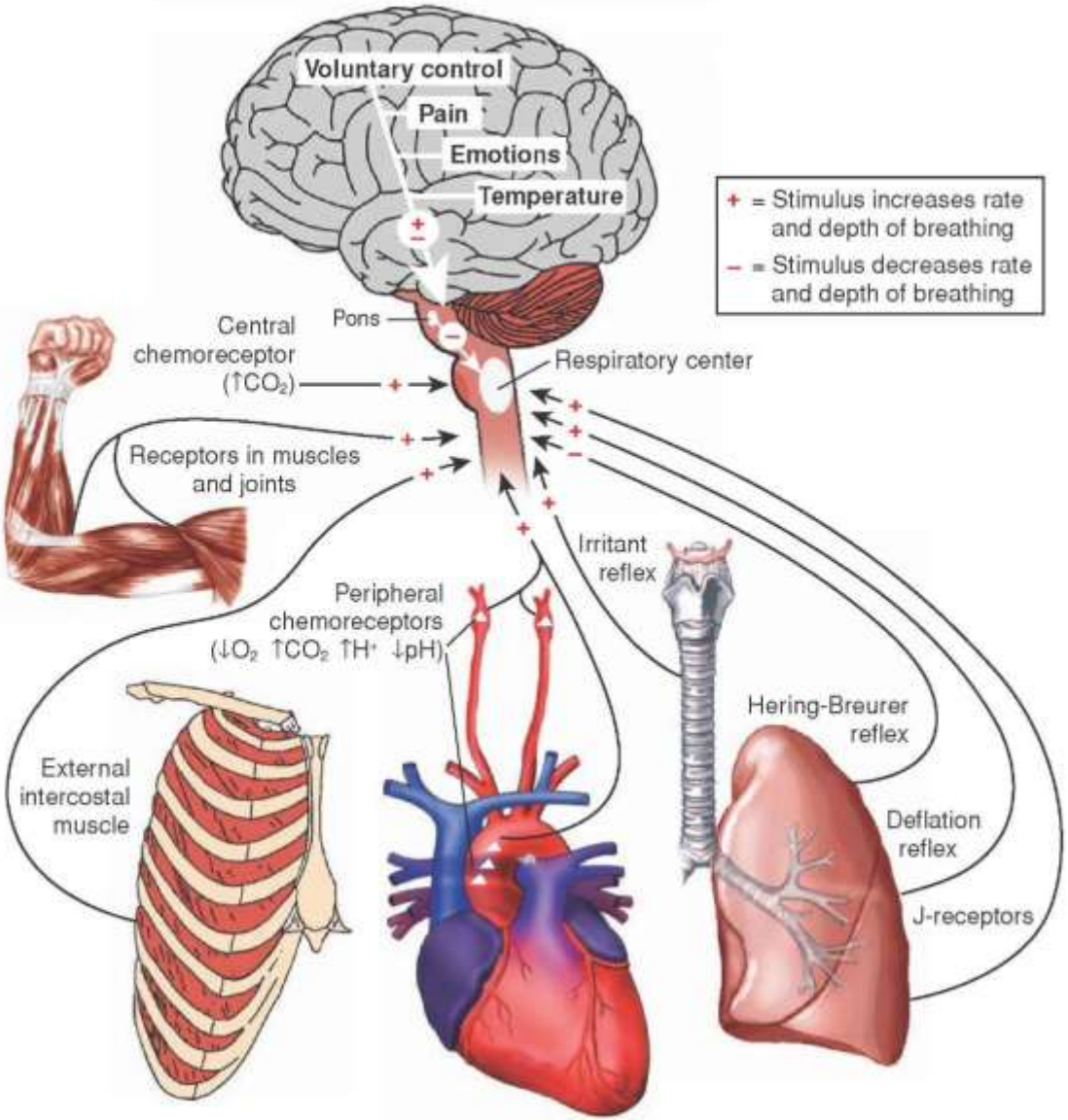
EFFERENT SIGNALS

Motor neuronal signals descending to the respiratory muscles, the most important being the **diaphragm**

PATHOPHYSIOLOGY



PATHOPHYSIOLOGY



PATHOPHYSIOLOGY

Three main components contribute to dyspnea: **afferent signals**, **efferent signals**, and **central information processing**. The central processing in the brain compares the afferent and efferent signals and dyspnea results when a **mismatch occurs between the two**, such as when the need for ventilation (afferent signaling) is not being met by physical breathing (efferent signaling). The afferent receptors allow the brain to assess whether the efferent or motor commands to the ventilatory muscles are effective, meeting the required demands of airway pressure, air flow, and/or lung movement.

When these respond inappropriately to the command, the intensity of the dyspnea increases. The sensory cortex is simultaneously activated when motor signals are sent to the chest wall, resulting in the **conscious sensation of muscular effort and breathlessness**. There is also a strong psychological component to dyspnea, as some people may become aware of their breathing in such circumstances but not experience the distress typical of the condition

AETIOLOGY

FOUR GENERATIVE CATEGORIES

CARDIAC

- Congestive cardiac failure
- Coronary artery disease
- Cardiomyopathy
- Valvular dysfunction
- Pericarditis
- Arrythmias

PULMONARY

- COPD
- Asthma
- Restrictive lung disease
- Pneumothorax

CARDIAC + PULMONARY

- COPD with pulmonary hypertension and cor pulmonale
- Chronic pulmonary emboli

NON CARDIAC NON PULMONARY

- Metabolic condition (acidosis)
- Pain in chest wall
- Neurovascular disorders
- Otorhinolaryngeal disorders
- Others

HISTORY

ONSET AND COURSE

Ask about the **onset** and **course** of dyspnea too elicit whether it's a a **chronic** or **acute issue**

ACUTE CAUSES (within minutes)

RESPIRATORY	CARDIAC
1. Acute exacerbation of asthma 2. Pneumothorax 3. Pulmonary embolism 4. Foreign body 5. Laryngeal edema	1. Acute MI 2. Congestive heart failure 3. Pericardial tamponade 4. Acute valvular insufficiency 5. Aortic dissection 6. Complete heart block

HISTORY

ONSET AND COURSE

Ask about the **onset** and **course** of dyspnea too elicit whether it's a a **chronic** or **acute issue**

SUBACUTE CAUSES		
Within hours	**Within days**	**Within weeks**
1. Asthma 2. Left heart failure 3. Pneumonia	1. Pneumonia 2. ARDS 3. Left heart failure	1. Pleural effusion 2. Anemia 3. Muscle weakness 4. Tumors

HISTORY

ONSET AND COURSE

Ask about the **onset** and **course** of dyspnea too elicit whether it's a a **chronic** or **acute issue**

CHRONIC PULMONARY CAUSES			
Airways	**Pleural**	**Parenchymal**	**Vascular**
1. COPD 2. Asthma 3. Chronic bronchitis 4. Empyema 5. Cystic fibrosis	1. Effusion 2. Malignancy 3. Fibrosis	1. Interstitial lung disease	1. Vasculitis 2. A-V malformation

HISTORY

POSITION OF DYSPNEA

Orthopnea - shortness of breath which occurs when lying flat, causing the person to have to sleep propped up in bed or sitting in a chair.

- Congestive cardiac failure
- Left ventricular failure
- COPD
- Bronchial asthma
- Massive pleural effusion
- Ascites
- GERD

HISTORY

POSITION OF DYSPNEA

Platypnea - shortness of breath that is relieved when lying down, and worsens when sitting or standing up. It is the **opposite of orthopnea**

- Left atrial myxoma
- Massive pulmonary embolism
- Paralysis of intercostal muscle
- Hepato-pulmonary syndrome

HISTORY

POSITION OF DYSPNEA

Trepopnea - shortness of breath that is sensed while lying on one side but not on the other (lateral recumbent position

- Disease of one lung / bronchus
- Congestive cardiac failure

HISTORY

TIMING OF DYSPNEA

Nocturnal onset dyspnea

Dyspnea that occurs mostly at night

- Congestive cardiac failure
- COPD
- Bronchial asthma
- Sleep apnea

Nocturnal onset dyspnea

Severe difficulty in breathing that awakens the person from sleep, making them sit or stand

- Underlying heart failure

Postprandial dyspnea

- GERD
- Aspiration
- Food allergy

HISTORY

GRADING OF DYSPNOEA

The Dyspnea Scale

Grade	Dyspnea related to activity
0	Breathlessness only on strenuous exercise
1	Breathless when hurrying on the level or walking up a slight hill
2	Walks slower than other people of same age on the level due to shortness of breath or need to stop for breath when walking at own pace
3	Short of breath after walking few minutes on the level or about 100 yards (90 m)
4	Too breathless to leave the house, or breathless when dressing or undressing

Modified from The Medical Research Council Dyspnea scale (mMRC dyspnea scale)

NYHA Classification

Class I	Ordinary physical activity does not cause undue fatigue, palpitations, dyspnea and/or angina
Class II	Ordinary physical activity does cause undue fatigue, palpitations, dyspnea and/or angina
Class III	Less than ordinary physical activity causes undue fatigue, palpitations, dyspnea and/or angina
Class IV	Fatigue, palpitations, dyspnea and/or angina occur at rest

Criteria Committee of the New York Heart Association, 1964.

HISTORY

PRECIPITATING AND RELIEVING FACTORS

PRECIPITATING FACTORS	RELIEVING FACTORS
1. Exercise 2. Exposure (cigarette, allergen) 3. Occupational exposure 4. Obesity 5. Severe weight loss 6. Medication	1. Rest 2. Medication ??

HISTORY

ASSOCIATED SYMPTOMS

- Chest pain – central, pleuritic or pericardial?
- Wheeze
- Fever
- Cough – sputum production and colour
- Change in pitch of voice
- Palpitation and syncope
- Hemoptysis
- Dysphagia and odynophagia
- Vomiting and diarrhoea
- Heart burn
- Muscle weakness or myalgia
- Visual disturbances and headache
- Bone pain

HISTORY

OTHER RELEVANT HX

- Past medical history
- Past surgical history
- Drug history
- Occupational history
- Smoking history

PHYSICAL EXAMINATION

WHAT TO LOOK FOR?

General inspection

- Anxiety may indicate patient have Anxiety Disorder
- Respiratory Distress ?
- Ability to speak – in sentences, phrase, word, unable to speak
- Is there any audible wheeze or stridor? Is the voice hoarse?
- Patient's position
- Cyanosis or pallor ?
- Mental Status : Altered mental status – hypoxemia or hypercapnia

Vital signs

- Pulse rate – Usually tachycardia, BRADYCARDIA in severe hypoxemia
- Respiratory Rate – TACHYPNEA, DANGER if >35-40 bpm or <10-12 bpm
- Temperature - FEVER
- Blood Pressure - ↑ if dyspnoea is significant, ↓ may indicate life threatening problem

PHYSICAL EXAMINATION

WHAT TO LOOK FOR?

- Nail Clubbing
- Peripheral Cyanosis
  Cyanosis & Clubbing may indicate chronic severe hypoxemia
- Cold extremities
- Nasal polyp, septal deviation – dyspnoea due to nasal obstruction
- Post-nasal discharge – Asthma/Allergies
- Jugular vein distention – congestive heart failure, pulmonary oedema
- Lower limb oedema – cardiac problem

Respiratory causes of clubbing:

- Bronchus CA
- Pulmonary Fibrosis
- Bronchiectasis
- Lung Abscess
- Pleural Empyema

Cardiac causes of clubbing:

- Congenital Heart Disease
- Infective Endocarditis

GI causes of clubbing:

- Hepatic Cirrhosis
- Celiac disease

Other: Thyroid

PHYSICAL EXAMINATION

CHEST EXAMINATION

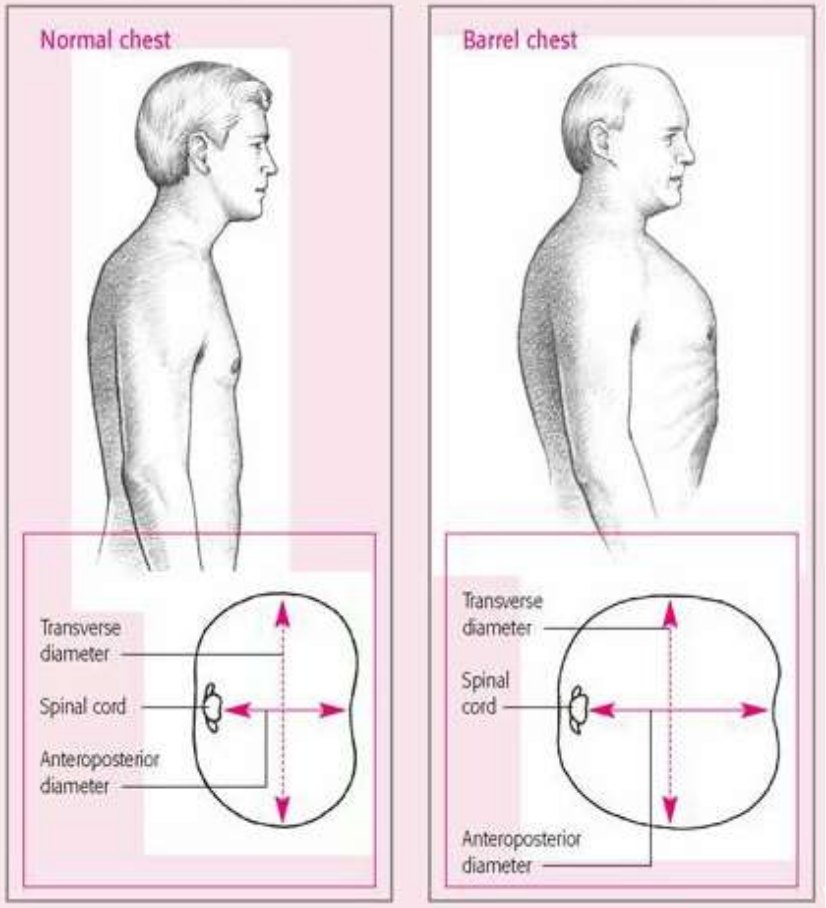
- Normal chest is symmetrical and elliptical in cross section; 5:7 (Hutchison's Index) so if increase anteroposterior chest diameter – **Emphysema**
- Contractions of the accessory muscles of respiration suggest severe difficulty
- Retraction of the supraclavicular fossa implies **tracheal stenosis**
- Pursed-lip breathing and a prolonged expiratory phase are signs outflow obstruction
- Retraction of the intercostal muscles on inspiration is characteristic of **emphysema**
- Percuss for dullness and hyper-resonance
- Auscultate for wheezes (**Asthma, Pulmonary Oedema**), and quality of breath sounds
- Crackles suggest fluid in the airway, as occurs with **bronchitis, pneumonitis,** and **CHF**

Normal findings on lung examination do not rule out pulmonary pathology but do lessen its probability and the likelihood that it is severe

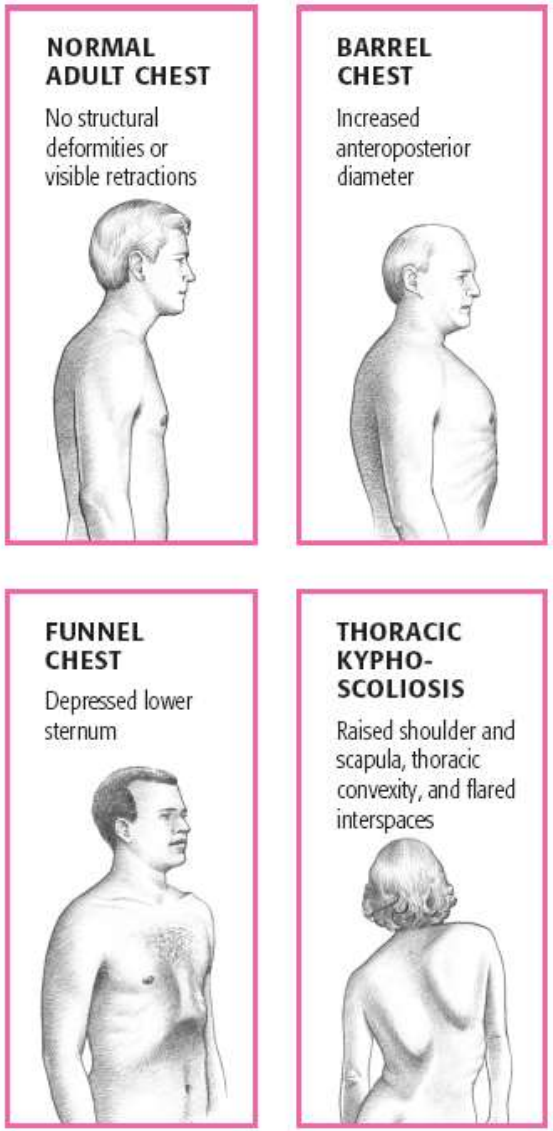
PHYSICAL EXAMINATION

Recognizing barrel chest

You can determine whether your patient has a normal chest or a barrel chest by looking at the anteroposterior and transverse chest diameters. In a normal adult chest, the ratio of anteroposterior to transverse (or lateral) diameter is 1:2. In patients with barrel chest, this ratio approaches 1:1 as the anteroposterior diameter enlarges.



When inspecting the patient's chest, note deviations in size and shape. These illustrations show a normal adult chest, along with four common chest deformities.



PHYSICAL EXAMINATION

CARDIOVASCULAR EXAMINATION

The **cardiac examination** should focus on:

- Signs of left-sided heart failure
- Detection of heart murmurs (left sided HF, valvular dysfunction, S3 – Congestive HF)
- Signs of pulmonary hypertension and its consequences (accentuated and delayed P^2, RV heave, RV S^3, right-sided systolic regurgitant murmur of TR, increased JVP, and peripheral oedema)

OTHERS

Examine the abdomen for **ascites** and hepato-jugular reflux as well as the legs for oedema

INVESTIGATIONS

INVESTIGATIONS

Complete blood count
Assess anemia and white cell count for infections

Blood C&S
Infections

Sputum AFB
To rule out TB

Arterial blood gas
To assess the acid-base status of patient to rule out ant acidosis

Electrolytes, BUN, creatinine and blood glucose
To assess metabolic derangement

INVESTIGATIONS

RELEVANT SPECIAL INVESTIGATIONS

Chest radiography (CXR)

This has a great potential in aiding the diagnosis of many lung disorders that cause acute dyspnea and chest pain.

Electrocardiography (ECG).

The ECG is unlikely to be normal in the presence of structural heart disease.

Echocardiography

Spirometry

Peak Expiratory Flow Rate

INVESTIGATIONS

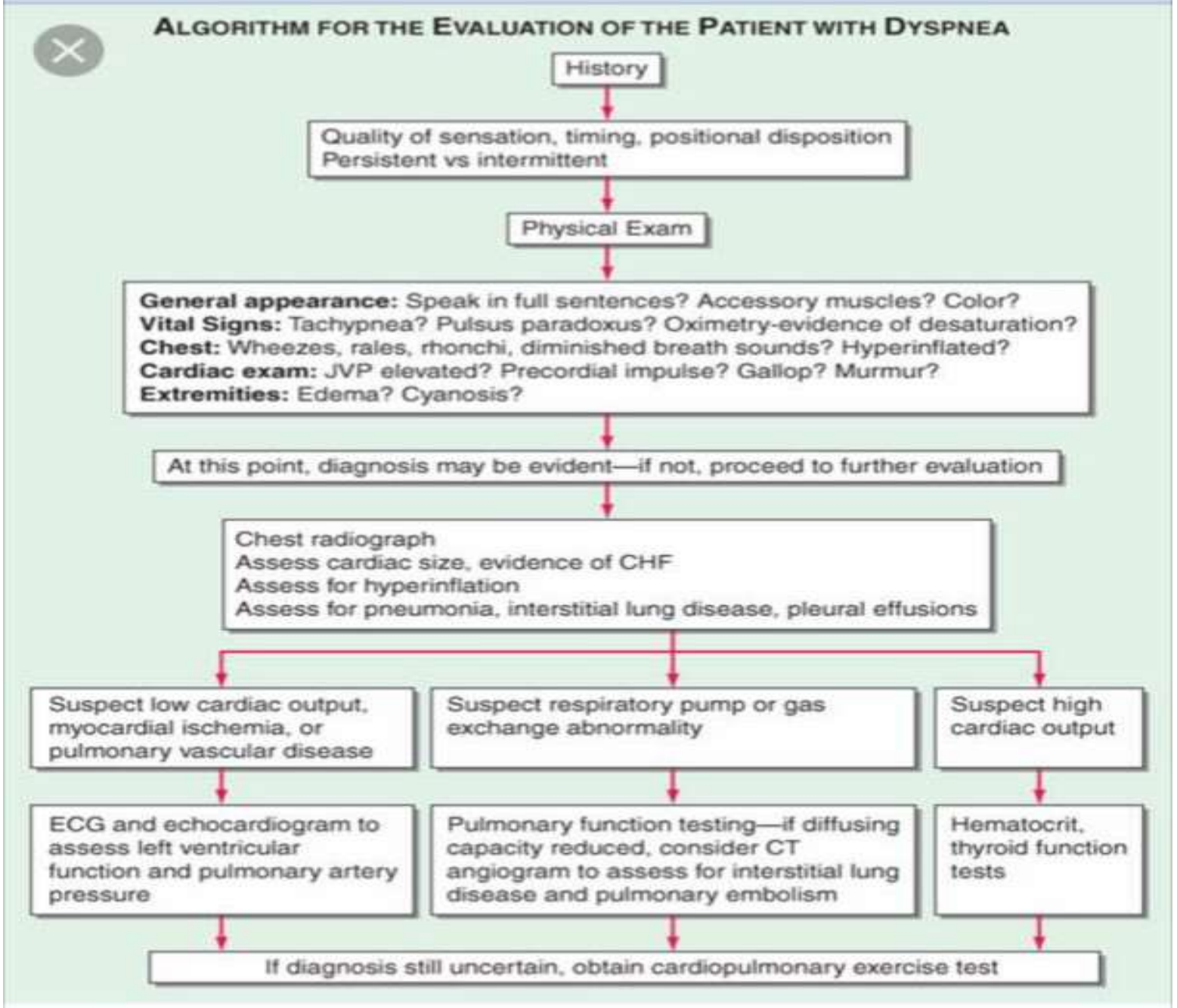
RELEVANT SPECIAL INVESTIGATIONS

The D-dimer.

This is a component of the evaluation of patients with suspected pulmonary embolism.

Point-of-care ultrasound scans and echocardiography.

These may aid clinicians in the diagnosis of acute cardiogenic pulmonary edema.



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine, 18th Edition*: www.accessmedicine.com

GENERAL MANAGEMENT

MANAGEMENT

Unstable patients typically present with one or more symptom patterns:

- Hypotension, altered mental status, hypoxia, or unstable arrhythmia
- Stridor and breathing effort without air movement (suspect upper airway obstruction)
- Unilateral tracheal deviation, hypotension, and unilateral breath sounds (suspect tension pneumothorax)
- Respiratory rate above 40 breaths per minute, retractions, cyanosis, low oxygen saturation

MANAGEMENT

Initial Assessment of Patients with Dyspnea

Assess airway patency and listen to the lungs.

Observe breathing pattern, including use of accessory muscles.

Monitor cardiac rhythm.

Measure vital signs and pulse oximetry.

Obtain any history of cardiac or pulmonary disease, or trauma.

Evaluate mental status.

MANAGEMENT

TREATMENT OF UNSTABLE PATIENT

- Administer **Oxygen**
- Consider intubation of the patient is gasping, apneic, or non- responsive
- Establish IV line and start **fluid resuscitation**
- Tension pneumothorax – Thoracentesis
- Obstructive pulmonary Disease – Bronchodilator
- Pulmonary edema – IV/IM furosemide

MANAGEMENT

After patient has been stabilized;

- Reassess the patient's airway, mental status, ability to speak, and breathing effort
- Check vital signs
- Thorough history taking and physical examination (i.e. breath sounds and observe skin colour)

MANAGEMENT

MANAGEMENT STRATEGIES

1. Reduce ventilatory demand
2. Decrease sense of effort
3. Improve respiratory muscle function
4. Pulmonary rehabilitation

MANAGEMENT

REDUCE VENTILATORY DEMAND

Treat the underlying causes

- Infection
- Pleural effusion
- Pneumothorax
- Pulmonary embolism
- Foreign bodies
- Congestive heart failure

Supplemental oxygen
Opiates and Sedatives
Exercise training

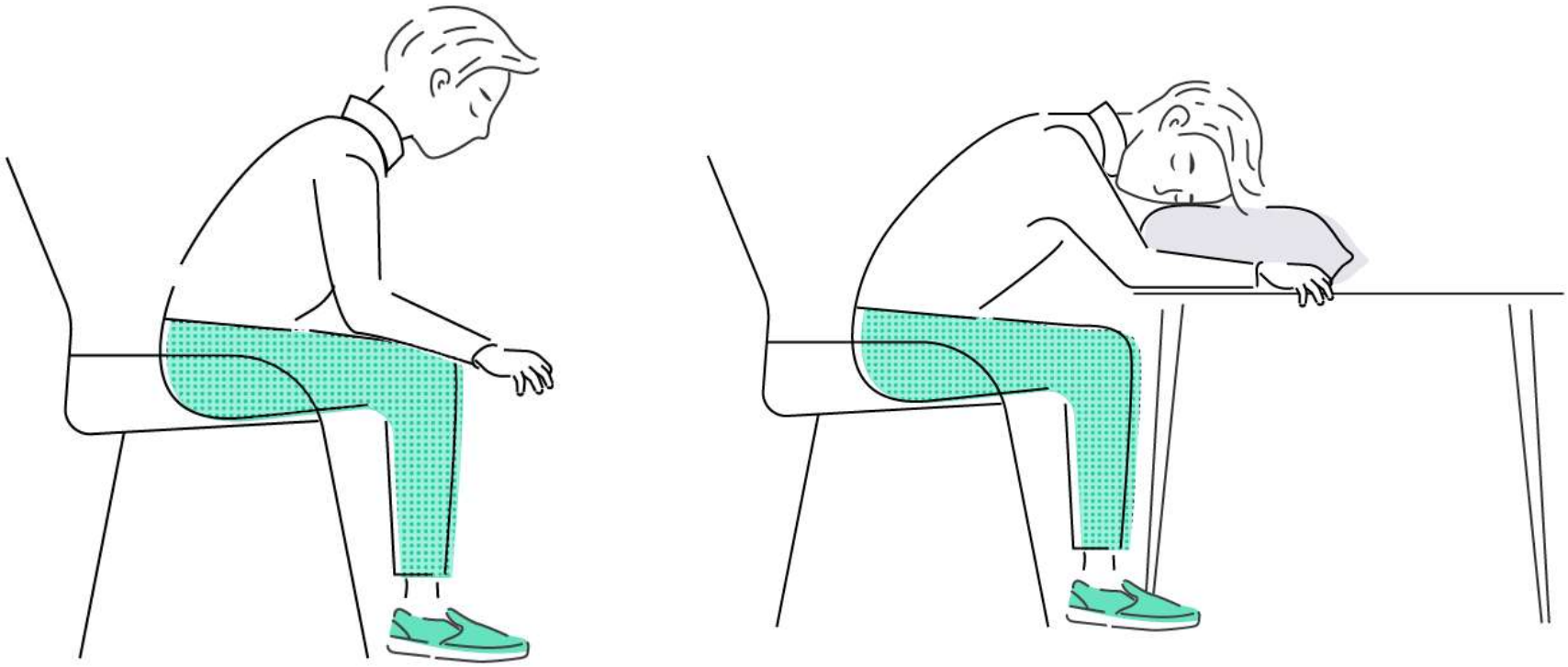
MANAGEMENT

DECREASE SENSE OF EFFORT AND IMPROVE RESP MUSCLE

- Positioning (leaning forward)
- Energy conservation / pacing
- Airflow
- Relaxation/ Distraction/ Assurance
- Controlled breathing techniques / Physiotherapy
- Loose clothing
- Correct obesity
- Medication (theophyline)

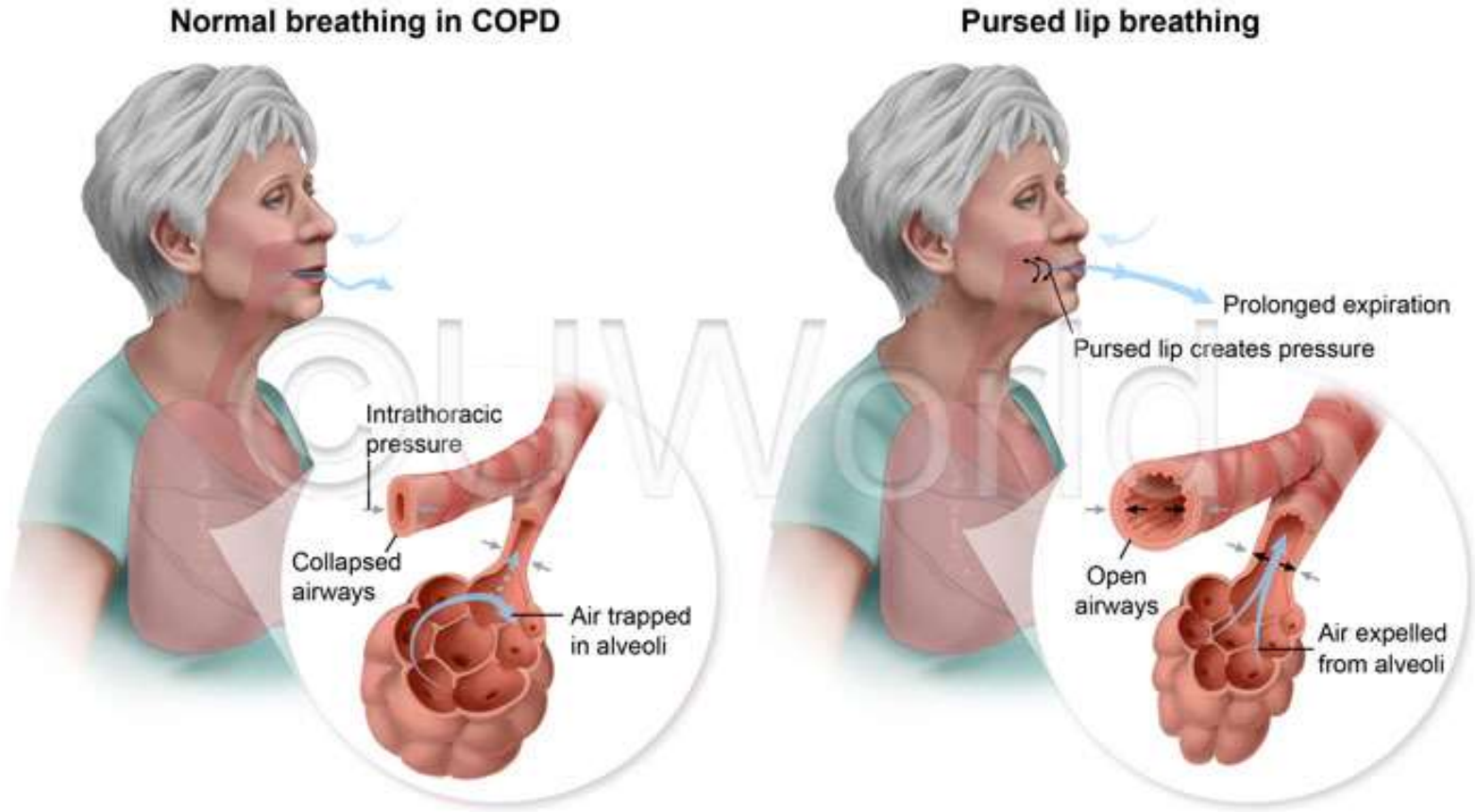
MANAGEMENT

POSITIONING (LEANING FORWARD)



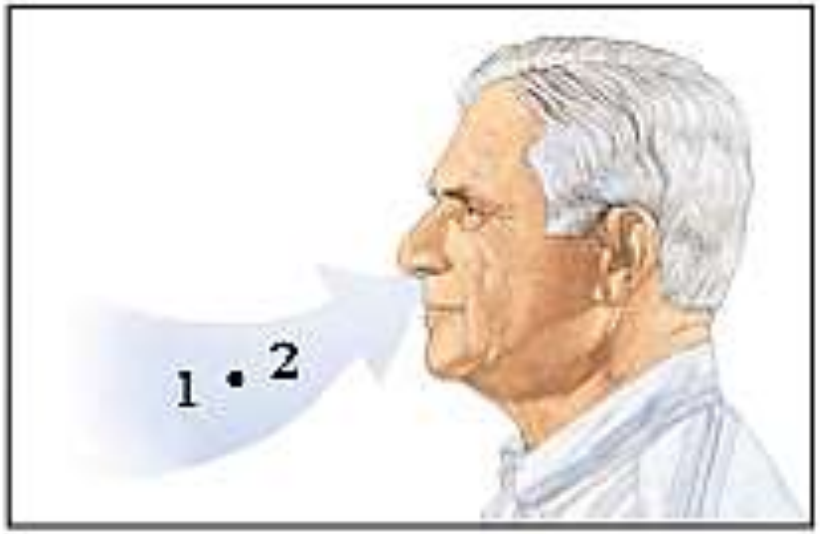
MANAGEMENT

BREATHING TECHNIQUES

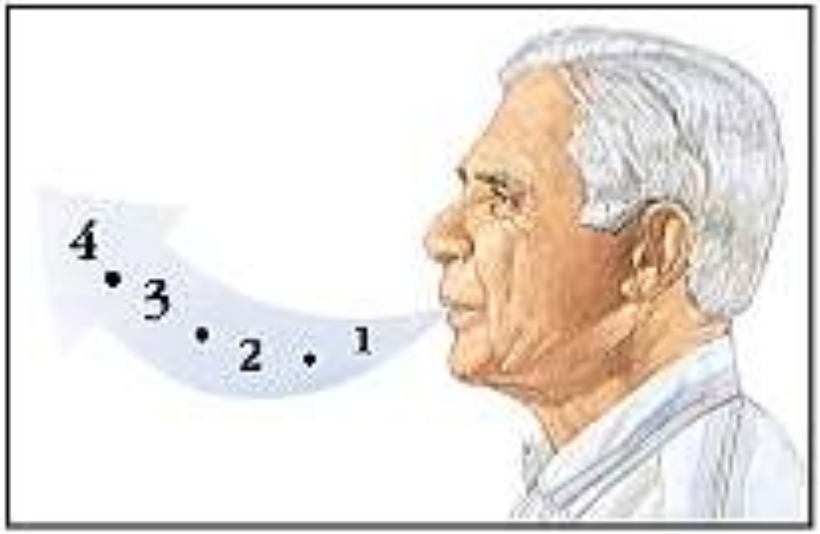


MANAGEMENT

BREATHING TECHNIQUES



Inhale: Relax your neck and shoulder muscles. Inhale slowly through your nose for 2 counts.



Exhale: Pucker your lips as if you are going to blow out a candle. Exhale slowly and gently through your lips for 4 or more counts.

SPECIFIC MANAGEMENT

BRONCHIAL ASTHMA

BRONCHIAL ASTHMA

MILD AEBA

Persistent cough, increased chest tightness, breathless when walking and normal speech.

Moderate expiratory wheeze on auscultation
PR < 100/min
RR < 25
PEF > 75%
SpO_2 > 95% under room air

Management:
Nebulizer Atrovent or
MDI with spacer (5 – 20x)

Observe for 1 hour,
Discharge if PEF > 75% with advice.

BRONCHIAL ASTHMA

MODERATE AEBA	SEVERE AEBA
Breathless when talking, talk in phrases.	Breathless at rest, talks in words.
Loud wheeze on auscultation **PR** 100 – 120/min **RR** 25 – 30 **PEF** 50 – 70% **SpO_2** 91 – 95% under room air	Loud wheeze on auscultation **PR** > 120/min **RR** > 30 **PEF** < 50% **SpO_2** < 90% under room air

Management:

1. Give O_2 > 40%
2. Nebulizer AVN/combivent
3. IV hydrocort 200mg stat

If poor response

+ s/c Terbutaline (Bricanyl) or
+ s/c Salbutamol 0.25 – 0.5mg

→

Incomplete response

Repeat neb, observe 1 hour

If PEF < 50 – 75% , **admit patient**

If PEF > 75% , discharge with:

1. Prednisolone 30mg OD 1/52
2. MDI

BRONCHIAL ASTHMA

LIFE THREATENING AEBA!!!!!!!

Central cyanosis, exhaustion, confusion or unconsciousness or convulsion
Feeble respiratory effort and silent chest on auscultation

Bradycardia / hypotension
PEF < 30%
ABG changes:

pH	acidotic
pCO_2	normal or > 45
pO_2	< 60

Management:
Same but + IV aminophylline 250mg slowly over 20 mins or
IV salbutamol 0.25mg over 10 mins

Treat in ICU if worsening PEF.

CHRONIC ASTHMA

AIMS

1. Abolish day and night symptoms
2. Restore long term airway function
3. Prevent acute attacks
4. Prevent mortality

ASSESSSMENT

1. Identify and avoid trigger factors
2. Assess severity and monitor response to tx
3. Educate patient and family

BRONCHIAL ASTHMA

Figure 2-5. Levels of Asthma Control

Characteristic	Controlled (All of the following)	Partly Controlled (Any measure present in any week)	Uncontrolled
Daytime symptoms	None (twice or less/week)	More than twice/week	**Three or more features of partly controlled asthma present in any week**
Limitations of activities	None	Any	
Nocturnal symptoms/awakening	None	Any	
Need for reliever/ rescue treatment	None (twice or less/week)	More than twice/week	
Lung function (PEF or FEV_1)‡	Normal	< 80% predicted or personal best (if known)	
Exacerbations	None	One or more/year*	One in any week†

* *Any exacerbation should prompt review of maintenance treatment to ensure that it is adequate.*

† *By definition, an exacerbation in any week makes that an uncontrolled asthma week.*

‡ *Lung function is not a reliable test for children 5 years and younger.*

BRONCHIAL ASTHMA

CLASSIFICATION OF ASTHMA SEVERITY BEFORE TREATMENT			
	Symptoms	**Night time Symptoms**	**PEF**
Severe Persistent	Daily Frequent exacerbations Limitation of physical activity	Frequent	≤ 60% predicted Variability > 30%
Moderate Persistent	Daily Daily use of beta$_2$-agonist Exacerbations affect activity and sleep	> 1 time a week	> 60% - < 80% predicted Variability > 30%
Mild Persistent	≥ 1 time a week but < 1 time a day Exacerbations may affect activity and sleep	> 2 times a month	≥ 80% predicted Variability 20-30%
Intermittent	< 1 time a week Brief exacerbations Asymptomatic and normal PEF between exacerbations	≤ 2 times a month	≥ 80% predicted Variability < 20%

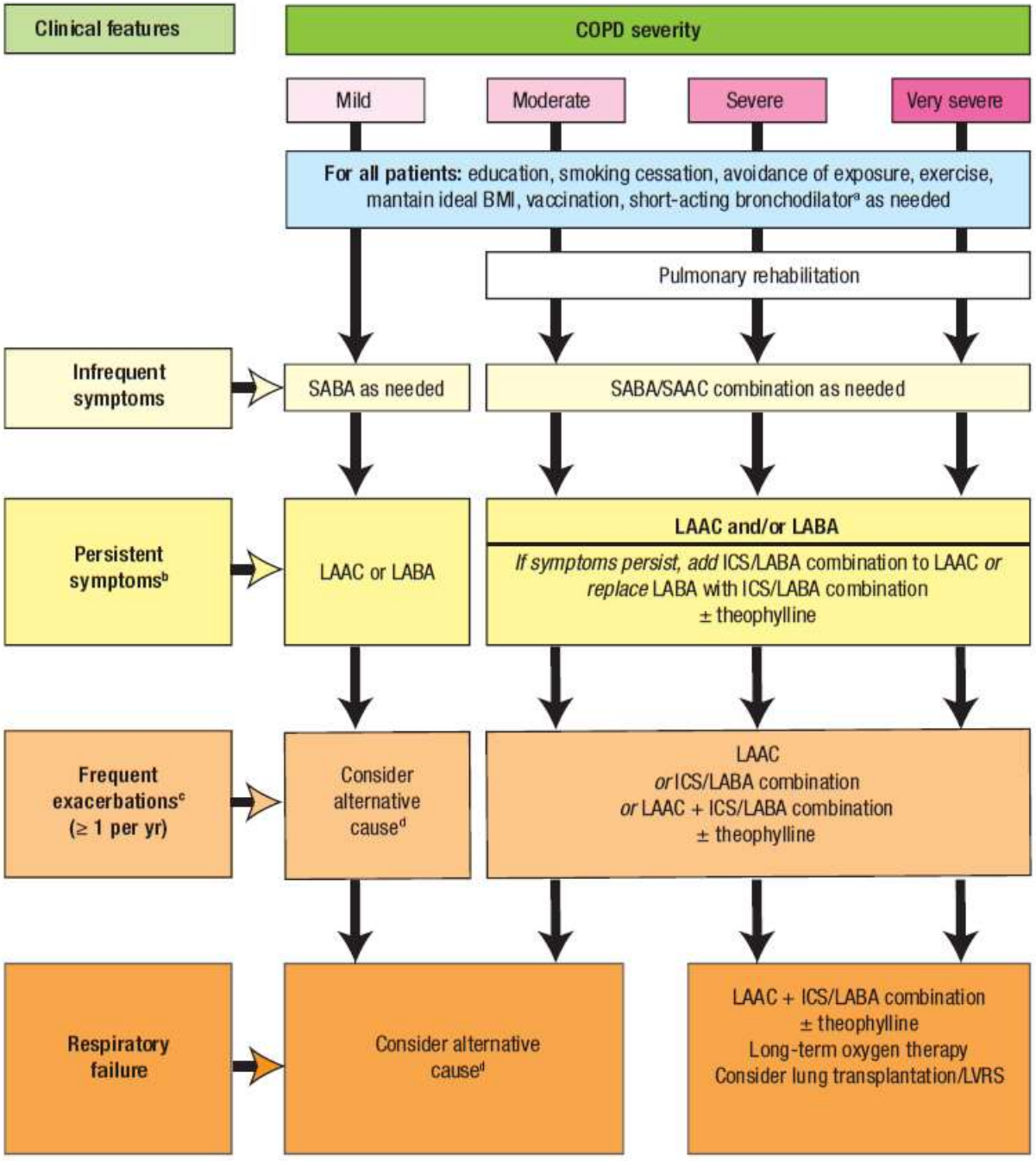
TREATMENT OF ADULT ASTHMA

Preferred treatments are in bold print
Patient education is essential at every step

	Long-Term Preventive	Quick-Relief
STEP 4 **Severe Persistent**	Daily medications: • **Inhaled corticosteroid***, 800-2000 mcg, and • Long-acting bronchodilator: either **inhaled long-acting beta$_2$-agonist** and/or sustained-release theophylline, and/or oral long acting beta$_2$-agonist, and • Oral corticosteroid long term	• Short-acting bronchodilator: **inhaled beta$_2$-agonist** as needed for symptoms
STEP 3 **Moderate Persistent**	Daily medications: • **Inhaled corticosteroid***,500-1000 mcg AND, if needed • Long-acting bronchodilator: either **inhaled long-acting beta$_2$-agonist**, sustained-release theophylline, or oral long acting beta$_2$-agonist (Inhaled long-acting beta$_2$-agonist may provide more effective symptom control when added to low-medium dose steroid compared to increasing the steroid dose) • Consider adding anti-leukotriene, especially for aspirin-sensitive patients and for preventing exercise-induced bronchospasm	• Short-acting bronchodilator: **inhaled beta$_2$-agonist** as needed for symptoms
STEP 2 **Mild Persistent**	Daily medications: • Either **Inhaled corticosteroid***, 200-500 mcg, or cromoglycate or sustained-release theophylline or anti-leukotrienes	• Short-acting bronchodilator: **inhaled beta$_2$-agonist** as needed for symptoms
STEP 1 **Intermittent**	• None needed	• Short-acting bronchodilator: **inhaled beta$_2$-agonist** as needed for symptoms, but less than once a week • Inhaled beta$_2$-agonist or cromoglycate before exercise or exposure to allergen

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Algorithm for Managing Stable COPD



COPD

SHORT ACTING	LONG ACTING	INHALED CORTICOSTEROIDS
1) **Short acting B2 Agonist** – MDI salbutamol, Feneterol, Terbutaline 2) **Short acting Anti Cholinergics** – MDI Ipratropium Bromide	1) **LABA** – MDI Salmeterol, Formorterol 2) **LAAC** – Tiotropium	1) **MDI Combivent** – Sabutamol + Ipratropium Bromide (**SABA + SAAC**) 2) **MDI Seretide** – Fluticarsone proprionate + salmeterol (**ICS + LABA)**

COPD

METHYXANTHINES	CORTICOSTEROIDS	LONG TERM OXYGEN THERAPY	SURGICAL INTERVENTION
Theophyline	▪ IV Hyrdocortisones ▪ T. Prednisolone	**Indications**: 1. Pa02 <55mmHg or Sa02 <88% with or w/o hypercapnia 2. Pa02 55-60mmHg, Sa02 89% with: ▪ pulmonary hypertension ▪ Peripheral edema (CCF) ▪ polycythemia	1. Lung volume reduction surgery 2. Bullectomy 3. Lung Transplantation

AECOPD

ADMINISTER INITIAL TREATMENT

- Controlled oxygen therapy if SpO2 < 90% aim for SpO2 90-93%
- Inhaled short-acting bronchodilators (SABA +/- SAAC) from pMDI via a spacer device or nebuliser
- Oral prednisolone (IV hydrocortisone if patient is unable to swallow or vomits)
- Start antibiotics if patient has 2 out of 3 cardinal symptoms (purulent sputum, increased sputum volume, increased dyspnoea)

AECOPD

If Good Response

No indication for hospital admission

↓

- Discharge with follow up
- Check Inhaler Technique
- Refer to specialist if this is a new presentation
- Refer to specialist if this is a new presentation

↓

Home management:

- Increase dose & frequency of inhaled short-acting bronchodilator (SABA ± SAAC) MDI
- Oral Prednisolone 30-40 mg daily for 7-14 days
- Ensure adequate supply of oral antibiotic if started

→

Indications for hospital admission:

- Marked increase in intensity of symptoms
- Underlying severe COPD
- Development of new physical signs, e.g. cyanosis, peripheral edema
- Hemodynamic stability
- Reduced alertness
- Failure of exacerbation to respond to initial medical management.
- Significant co-morbidities
- Newly occurring cardiac arrhythmias
- Older age
- Insufficient home support

AECOPD

IF FAILURE TO IMPROVE

- Admit patient to hospital

HOSPITAL MANAGEMENT

- Controlled supplemental oxygen therapy to maintain Pa02 >8kPa or SP02 >90% w/o worsening hypercapnia or precipitating acidosis
- Inhaled short acting bronchodilator from MDI via a spacer device or nebuliser
- Consider IV aminophylline if inadequate response to inhaled short acting bronchodilators
- Consider subcutaneous heparin
- Closely monitor condition of the patient
- Consider invasive or non-invasive ventilation

CONGESTIVE CARDIAC FAILURE

MANAGEMENT

CONGESTIVE CARDIAC FAILURE

1. **Oxygen**
   5 to 6 litres/min by mask with the aim of achieving oxygen saturation of more than 95% in order to maximise tissue oxygenation and to prevent multi-organ failure.
   Intubation and mechanical ventilation may be needed if the oxygen saturation is still inadequate and the patient develop respiratory muscle fatigue.

2. **Frusemide**
   Dose depending on the severity of the condition.

3. **Morphine sulphate**
   Reduces pulmonary congestion and symphathetic drive. Most useful in patients who are dypnoeic and restless. IV anti-emetics should be administered cocomintantly

4. **Nitrates**
   Given sublingually or IV if BP is adequate SBP>100. Patient should be closely monitored for hypotension. Contraindicated in valvular stenosis.

REFERENCES

1. Hutchinson’s clinical methods 23th edition
2. Davidson’s principles & practice of medicine
3. General Internal Medicine The HO Guide

THANK YOU