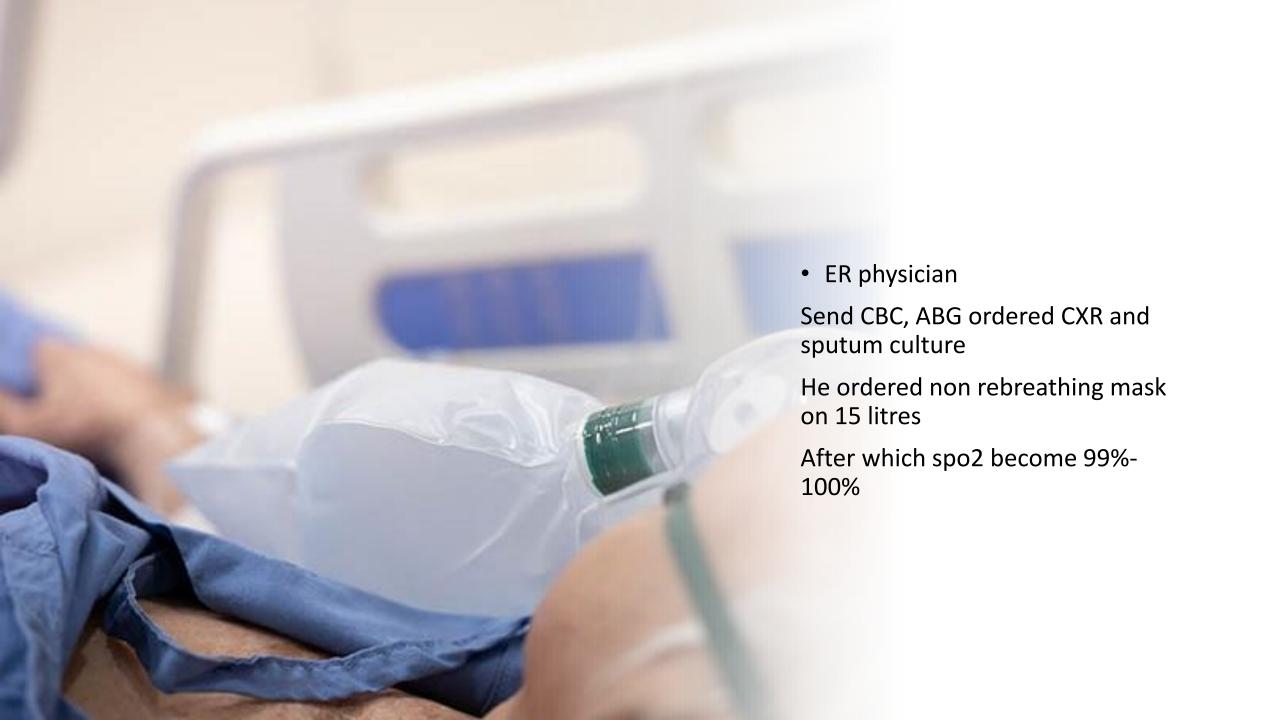
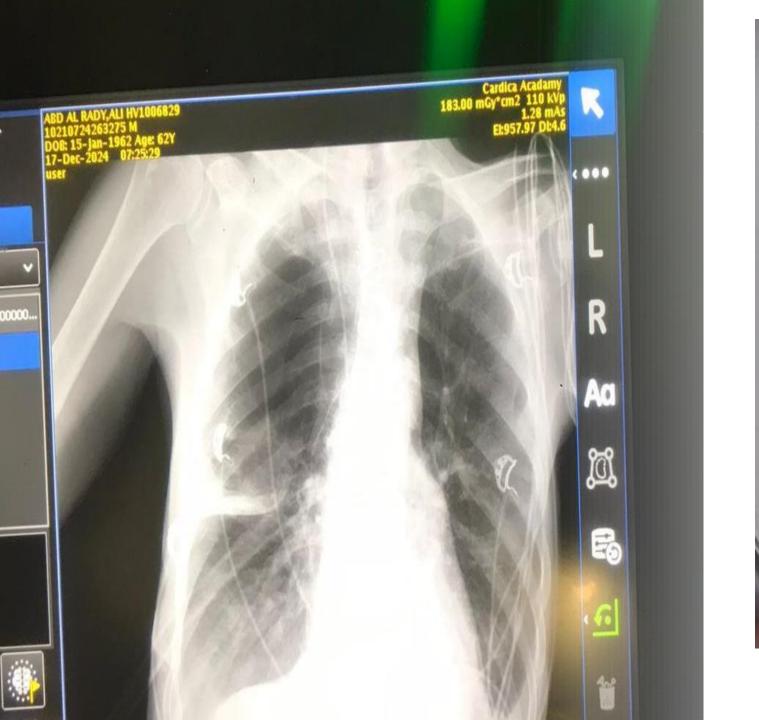


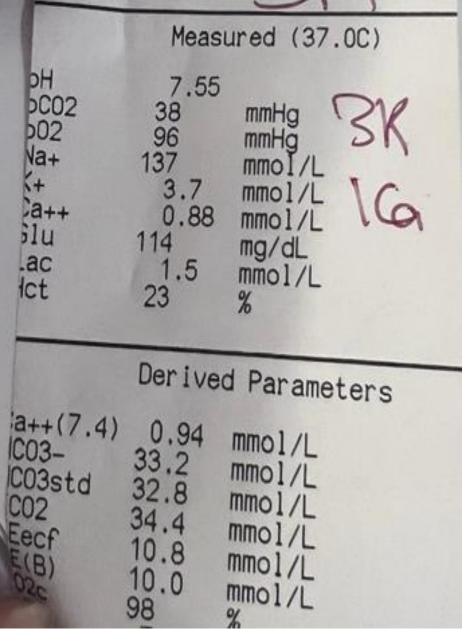
- Male patient 66 years old diabetic hypertensive for 40 years 2 packs per day
- Presented to ER with fever 37.8c, dyspnea, RR 30, Spo2 80%
- HR sinus 120 bpm blood pressure 110/60



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Patient then become sweaty and drowsy

RBS checked:130

ABG re-extracted showing:

PH 7.18, pco2 72, HCO3 (28)

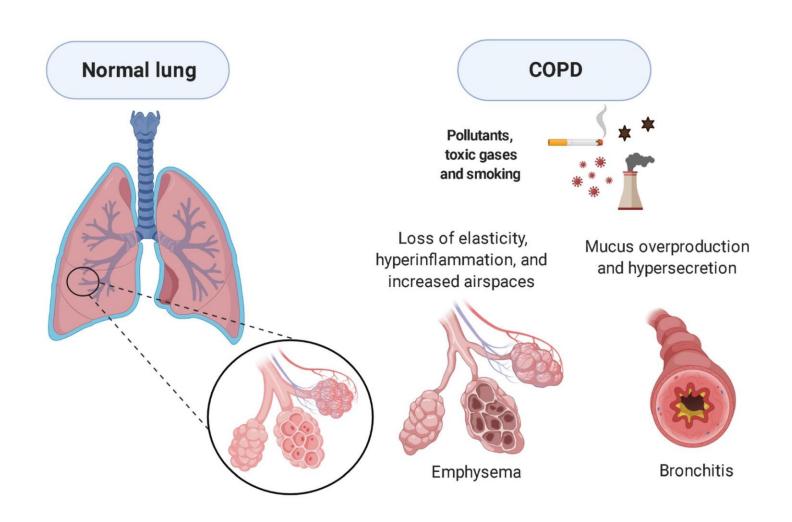
CPAP applied to patient, and bronchodilators administered On parameters: After 2 minutes patient reassessed, and he became DCL



So, decision of intubation and invasive mechanical ventilation



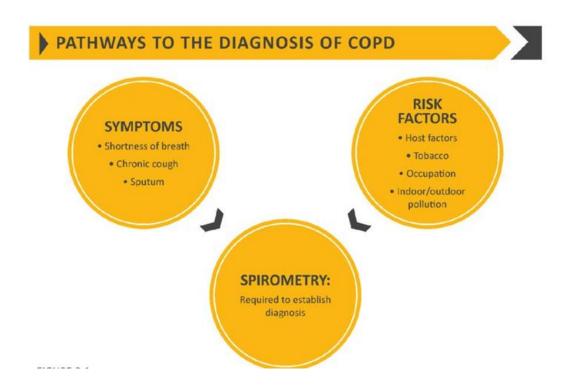
pathophysiology



Diagnosis of COPD



Diagnosis and Initial Assessment



Severity assessment



Post-bronchodilator FEV₁

IN COPD (BASED ON POST-BRONCHODILATOR FEV ₁) In patients with FEV1/FVC < 0.70:			
GOLD 1:	Mild	FEV₁ ≥ 80% predicted	
GOLD 2:	Moderate	50% ≤ FEV ₁ < 80% predicted	
GOLD 3:	Severe	30% ≤ FEV ₁ < 50% predicted	
GOLD 4:	Very Severe	FEV ₁ < 30% predicted	



Severity assessment

COPD Assessment Test (CAT™)

CAT™ ASSESSMENT For each item below, place a mark (x) in the box that best describes you currently. Be sure to only select one response for each question. **EXAMPLE:** I am very happy I am very sad SCORE I never cough I cough all the time I have no phlegm (mucus) My chest is completely full 1 2 3 4 5 in my chest at all of phlegm (mucus) (1) (2) (3) (4) (5) My chest feels very tight My chest does not feel tight at all When I walk up a hill or one flight When I walk up a hill or one flight of stairs I am not breathless of stairs I am very breathless I am not limited doing any I am very limited doing activities at home I am confident leaving my home I am not at all confident leaving my despite my lung condition home because of my lung condition I sleep soundly I don't sleep soundly because 1 2 3 of my lung condition 1 2 3 4 5 I have no energy at all I have lots of energy

Reference: Jones et al. ERJ 2009; 34 (3); 648-54.



Severity assessment



Modified MRC dyspnea scale

MODIFIED MRC DYSPNEA SCALE^a PLEASE TICK IN THE BOX THAT APPLIES TO YOU | ONE BOX ONLY | Grades 0 - 4 I only get breathless with strenuous exercise. mMRC Grade 0. I get short of breath when hurrying on the level or walking mMRC Grade 1. up a slight hill. mMRC Grade 2. I walk slower than people of the same age on the level because of breathlessness, or I have to stop for breath when walking on my own pace on the level. I stop for breath after walking about 100 meters or after a few mMRC Grade 3. minutes on the level. I am too breathless to leave the house or I am breathless mMRC Grade 4. when dressing or undressing.

^a Fletcher CM. BMJ 1960; 2: 1662.

Severity assessment



ABCD assessment tool

THE REFINED ABCD ASSESSMENT TOOL

Spirometrically **Confirmed Diagnosis**

Assessment of airflow limitation

Moderate or Severe **Exacerbation History**

i......

Assessment of symptoms/risk of exacerbations

Post-bronchodilator FEV₁/FVC < 0.7

Grade	FEV ₁ (% predicted)
GOLD 1	≥ 80
GOLD 2	50-79
GOLD 3	30-49
GOLD 4	< 30

Grade	FEV ₁ (% predicted)	≥2 or ≥ 1 leading
OLD 1	≥ 80	to hospital admission
OLD 2	50-79	ļi
OLD 3	30-49	0 or 1 (not leading
OLD 4	< 30	to hospital admission)

С	D
Α	В

mMRC 0-1 ii mMRC ≥ 2

Symptoms

Assessment of COPD Adapted from GOLD

Medications according to severity

Spirometrically Confirmed Diagnosis



Assessment of airflow limitation



Moderate or Severe Exacerbation Assessment of symptoms/risks of exacerbations

Post-bronchodilator FEV₁/FVC < 0.7

Grade	Severity	FEV ₁ (% predicted)
GOLD 1	Mild	≥ 80
GOLD 2	Moderate	50-79
GOLD 3	Severe	30-49
GOLD 4	Very Severe	< 30

History

≥2 or ≥1
leading to

hospital

admission

0 or 1 (not leading to hospital admission)

Group C	Group D
LAMA	LAMA or LAMA + LABA or ICS + LABA
Group A	Group B
Bronchodilator	LABA or LAMA

mMRC 0-1 CAT < 10 mMRC≥2 CAT≥10



Nonpharmacological treatment of COPD

Non-Pharmacological Treatment

- Education and self-management
- Physical activity
- Pulmonary rehabilitation programs
- Exercise training
- Self-management education
- End of life and palliative care
- Nutritional support
- Vaccination
- Oxygen therapy

Nonpharmacological treatment of COPD

NON-PHARMACOLOGIC MANAGEMENT OF COPD

PATIENT GROUP	ESSENTIAL	RECOMMENDED	DEPENDING ON LOCAL GUIDELINES
Α	Smoking Cessation (can include pharmacologic	Physical Activity	Flu Vaccination
	treatment)		Pneumococcal Vaccination
	Smoking Cessation	Physical Activity	Flu Vaccination
B-D	(can include pharmacologic treatment)		Pneumococcal Vaccination
	Pulmonary Rehabilitation		

When to add oxygen therapy?

Non-pharmacological treatment

PRESCRIPTION OF SUPPLEMENTAL OXYGEN TO COPD PATIENTS

Arterial hypoxemia defined as: PaO₂ < 55 mmHg (8 kPa) or SaO₂ < 88%

or

PaO₂ > 55 but < 60 mmHg (> 7.3 kPa but < 8 kPa) with right heart failure or erythrocytosis



Prescribe supplemental oxygen and titrate to keep SaO₂ ≥ 90%



Recheck in 60 to 90 days to assess:

- » If supplemental oxygen is still indicated
- » If prescribed supplemental oxygen is effective



Management of Exacerbations

OVERALL KEY POINTS (1 of 3):

- An exacerbation of COPD is defined as an acute worsening of respiratory symptoms that results in additional therapy.
- Exacerbations of COPD can be precipitated by several factors. The most common causes are respiratory tract infections.
- The goal for treatment of COPD exacerbations is to minimize the negative impact of the current exacerbation and to prevent subsequent events.
- Short-acting inhaled beta₂-agonists, with or without short-acting anticholinergics, are recommended as the initial bronchodilators to treat an acute exacerbation.



Management of Exacerbations

OVERALL KEY POINTS (2 of 3):

- Maintenance therapy with long-acting bronchodilators should be initiated as soon as possible before hospital discharge.
- Systemic corticosteroids can improve lung function (FEV₁), oxygenation and shorten recovery time and hospitalization duration. Duration of therapy should not be more than 5-7 days.
- Antibiotics, when indicated, can shorten recovery time, reduce the risk of early relapse, treatment failure, and hospitalization duration. Duration of therapy should be 5-7 days.
- Methylxanthines are not recommended due to increased side effect profiles.

Table 1. Anthonisen classification of AECOPD

Type I (most severe)	Type II	Type III
All three symptoms (i.e., increased sputum volume, increased sputum purulence and increased dyspnea).	Any two symptoms present	One symptom present plus at least one of the following: • An upper respiratory tract infection in the past 5 days • Increased wheezing • Increased cough • Fever without an obvious source • A 20% increase in respiratory rate • Heart rate above baseline



Management of Exacerbations

COPD exacerbations are defined as an acute worsening of respiratory symptoms that result in additional therapy.

- They are classified as:
 - Mild (treated with short acting bronchodilators only, SABDs)
 - Moderate (treated with SABDs plus antibiotics and/or oral corticosteroids) or
 - Severe (patient requires hospitalization or visits the emergency room). Severe exacerbations may also be associated with acute respiratory failure.



Management of Exacerbations

OVERALL KEY POINTS (3 of 3):

Non-invasive mechanical ventilation should be the first mode of ventilation used in COPD patients with acute respiratory failure who have no absolute contraindication because it improves gas exchange, reduces work of breathing and the need for intubation, decreases hospitalization duration and improves survival.



Management of Exacerbations

INDICATIONS FOR RESPIRATORY OR MEDICAL INTENSIVE CARE UNIT ADMISSION*

- Severe dyspnea that responds inadequately to initial emergency therapy.
- Changes in mental status (confusion, lethargy, coma).
- Persistent or worsening hypoxemia (PaO2 < 5.3 kPa or 40mmHg) and/or severe/worsening respiratory acidosis (pH < 7.25) despite supplemental oxygen and noninvasive ventilation.
- Need for invasive mechanical ventilation.
- · Hemodynamic instability need for vasopressors.



Management of Exacerbations

INDICATIONS FOR INVASIVE MECHANICAL VENTILATION

- · Unable to tolerate NIV or NIV failure.
- Status post respiratory or cardiac arrest.
- Diminished consciousness, psychomotor agitation inadequately controlled by sedation.
- · Massive aspiration or persistent vomiting.
- Persistent inability to remove respiratory secretions.
- Severe hemodynamic instability without response to fluids and vasoactive drugs.
- · Severe ventricular or supraventricular arrhythmias.
- · Life-threatening hypoxemia in patients unable to tolerate NIV.

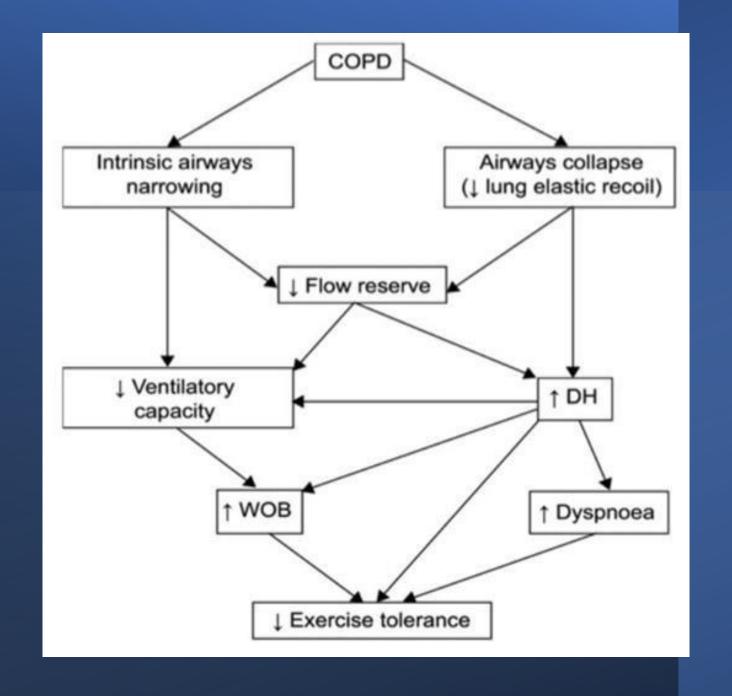
During exacerbation

How our lung act?

Working exp musclesfurther airway collapse....worsening

Our lung try to increase exp time...... ↑ insp flow, ↓ insp time , ↑ exp time

Also increase insp flow led to increase airway caliber and increased airway resistance.... But on other hand large volumes lead to decreased compliance and increase WOB, and increased IPEEP



CO2 in COPD Exacerbation

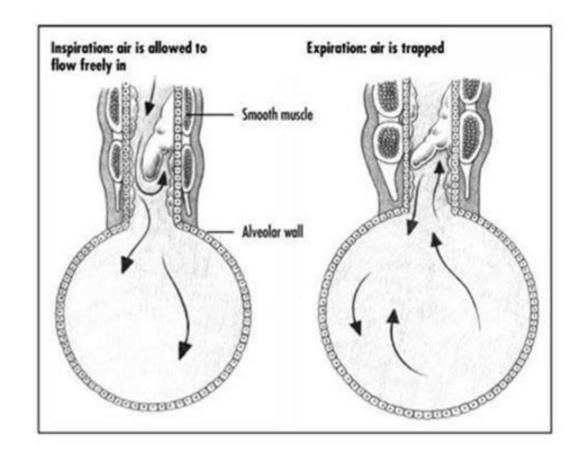
- In COPD cases increase CO2 level due to alveolar destruction
- Increase WOB lead to increase CO2 production
- Increase Dead space needs higher minute ventilation to eliminate CO2
- Increased iPEEP lead to Lower COP and resp muscle fatigue

Hazards of increased CO2:

- decrease anaerobic metabolism
- ❖ Decrease ATP stores
- Decreased glycogen stores

Dynamic Hyper Inflation (DHI)

- **Def: Increased relaxation volume** of the respiratory system at the end of a tidal expiration.
- Mostly this extra alveolar VOLUME will increase alveolar PRESSURE at end of expiration above Patm = INTRINSIC PEEP = Auto PEEP



DHI Consequences:

- Consequences of DHI (pulmonary vs extrapulmonary problems!)
 - Gas exchange disturbances (impaired alveolar flow)(Inc WOB / RF)
 - 2. Local alveolar barotrauma and injury
 - 3. During MV: Increased effort or Difficult to trigger a breath (during weaning)
 - 4. Hemodynamic and heart-lung disturbances (acute; Hypotension longterm; pulmonary hypertension / corpulmonal!)

Goals of mechanical ventilation:

Correct acid base imabalnce

Resting respiratory muscles

Patient support during pharmacological TTT

What parameters?





Parameters of mechanical ventilation:

Tidal volume

Respiratory rate

Inspiratory flow

PEEPe

Triggering

Adjust to target preset CO2 level and to decrease DHI

Tidal volume

• IF INCREASED DHI:

Volume mode	Pressure mode
Decrease TV	Decrease insp pressure
Decrease RR	Decrease insp time
	Decrease RESP rate

Respiratory Rate

NO Need for rapid normalization of PCO2

Increased Resp Rate leads to decrease EXP time

In VCV adjustment of RR should be cautious not to affect exp time

If patient become agitated and tachypnic More sedation needed to decline RESP Rate to avoid increase in DHI

Inspiratory flow:

Appropriate insp flow rate Leads to adequate TV and resp ms rest

Low flow...... Air hunger.....increase WOB and RR

HIGHER INSP PRESSURE:

Less insp timemore exp time

More uniform distribution across alveoli

Higher insp flow cause higher PEAK Pressure Mostly it is dissipated in high pressure zones not alveoli

Over time it will decline with declining DHI

RAMP is better than square flow (better gas exchange, lower peak pressure but may increase insp time)

Prolong expiratory time (Te):

- Flow: Using a higher PIFR (70–100 L/min)
- Ramp pattern better
- Flow triggering better than pr triggering
- Apply shorter Ti (0.8 1.2 sec)
- Eliminating inspiratory Pause time (volume targeted modes)
- Decreasing RR
- Decreasing I:E ratio (1:3..)
- During CPAP weaning: early switch to expiration (Flow cycling time >25%)

Add PEEP or not?



PEEP in COPD patient

PEEP stent
collapsible air
way..... Increase
exp flow

Easier for triggering.....lower WOB

Target
PEEP....<75% of
PEEPi

Trigger

Sensitivity according to magnitude adjusted

Flow trigger is better and less WOB

Pressure trigger....delayed...increased WOB

Modes of ventilation:

AC mode allow more resp muscle rest than SIMV modes CPAP and BIPAP modes allow resp ms rest through their PS PRESSURE SUPPORT mode allow patient to control TV and insp time Allowing more ms rest and decreased DHI If assynchrony in PSV think of declining PS?? Targets: adequate TV accptable RR (<30)

Monitoring

• Peak pressure:

Sum of elastic recoil and pr across air way (flow*resistance)

Plateau pressure:

During insp hold reflect compliance

More than 30-35 (barotrauma and worsening DHI)





• Patient become hypotensive and congested neck veins??

• DD

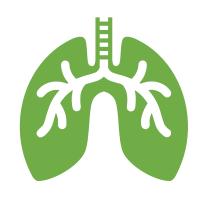
On weaning

- Tpiece
- PSV

• Role of NIV



HFNC in COPD patient





Pros: better tolerated - No interface with speech and eating - humidification enhances ciliary clearance and secretion removal - decrease dead space → improve WOB / partial CO2 washout

Cons: unmeasurable PEEPe (may worsen DHI in AECOPD?) - Blunting Hypoxic drive in COPD patients?





Thank you