COPD
Beginning of cataclysm?

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Global Initiative for Chronic Obstructive Lung Disease

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Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease

INTRODUCTION

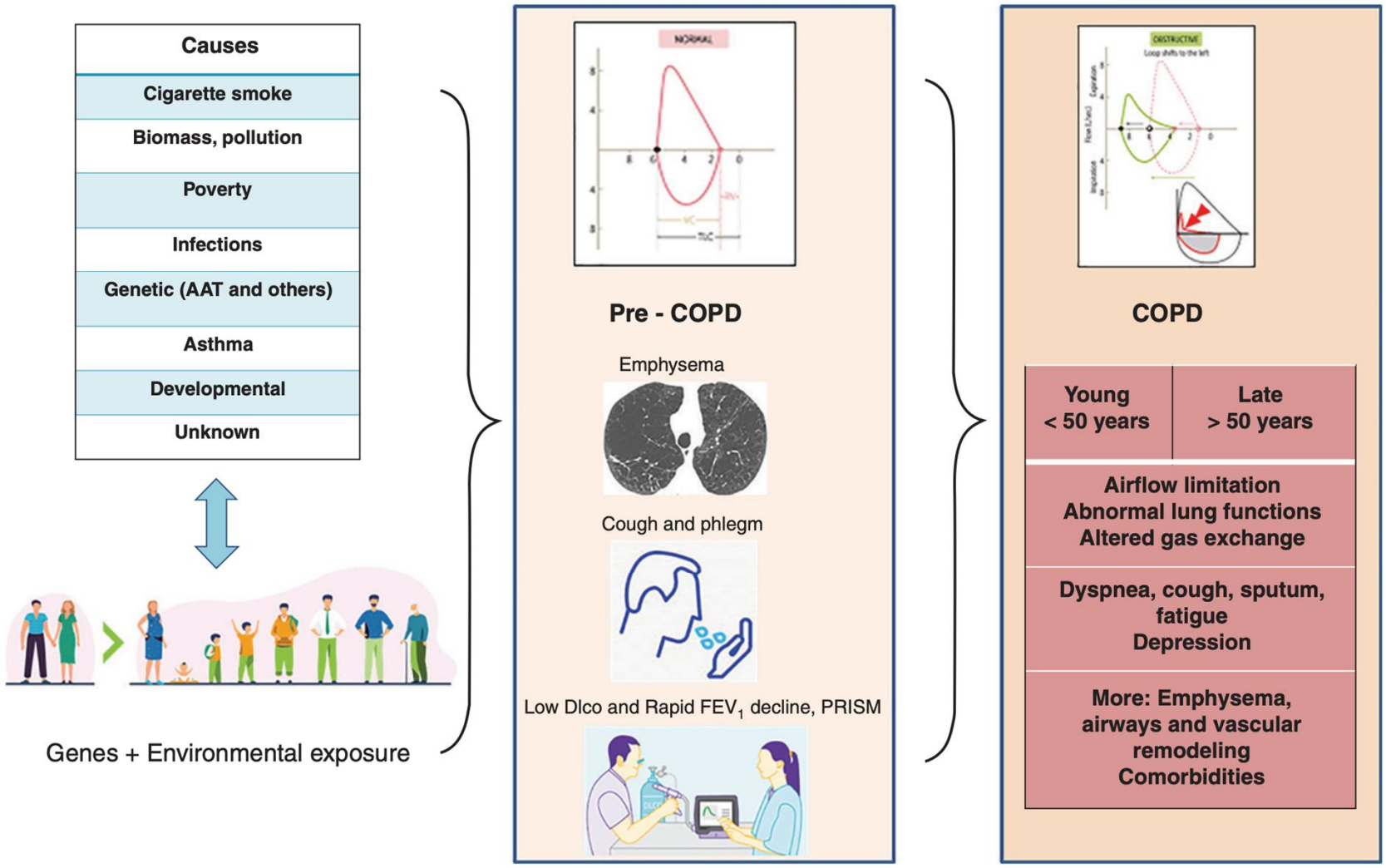
- Chronic Obstructive Pulmonary Disease (COPD) is now one of the **top three causes of death** worldwide and 90% of these deaths occur in low- and middle-income countries (LMICs).
- COPD represents an important public health challenge that is both **preventable and treatable**.
- Globally, the COPD burden is projected to increase in coming decades because of continued exposure to COPD risk factors and aging of the population
- The global prevalence of COPD is 10.3%
- COPD is one of the most important causes of death
  - For instance, in 2011, COPD was the third leading cause of death in the U.S
  - DATA from the Global Burden of Disease Study 2017 estimated a COPD-attributable death rate was 42/100,000 (4.71% of all-cause death)

Problems with the previous definition of COPD

- COPD definition and taxonomy fail to identify the disorder at its early stages, before airflow limitation becomes evident.
- Defines the entity as a single disease, limiting the role of different causes of COPD leading to the same functional or physiologic abnormality.

A concept to define a new COPD

- The first concept is that COPD results **not only from the consumption of cigarettes but also from other causes**, such as biomass exposure, poverty, infections such as tuberculosis, or even asthma.
- Second, **the advent of novel tools, such as computed tomography (CT)** of the chest, has provided evidence that structural lung abnormalities can be detected in the absence of airflow limitation, and the term **pre- COPD** has been proposed to describe these individuals.
- Third, general population studies have shown that symptoms (cough and sputum) can identify middle-aged subjects with high risk of developing persistent airflow limitation.
- Finally, events occurring during pregnancy and throughout childhood and adolescence can **profoundly impact lung development and result in airflow** limitation without an obligatory rapid decline in lung function over time (a feature previously believed to be cardinal to COPD)



Definition

2022

OVERALL KEY POINTS:

- *Chronic Obstructive Pulmonary Disease (COPD) is a common, preventable, and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases.*

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- Chronic Obstructive Pulmonary Disease (COPD) is a heterogeneous lung condition characterized by chronic respiratory symptoms (dyspnea, cough, sputum production and/or exacerbations) due to abnormalities of the airways (bronchitis, bronchiolitis) and/or alveoli (emphysema) that cause persistent, often progressive, airflow obstruction.

Diagnostic criteria

- The presence of non-fully reversible airflow limitation (FEV1/FVC <0.7 post-bronchodilation) measured by spirometry confirms the diagnosis of COPD
- ‘Pre-COPD’
- ‘PRISm’

Pathogenesis

- Environmental risk factor
  - Cigarette smoking
  - Biomass exposure
    - Wood, animal dung, crop residues, and coal
  - Occupational exposure
    - organic and inorganic dusts, chemical agents and fumes
  - Air pollution
    - particulate matter (PM), ozone, oxides of nitrogen or sulfur, heavy metals, greenhouse gases
- Genetic Factors
  - SERPINA1 gene
  - Hundreds of genetic variants associated with reduced lung function and risk of COPD

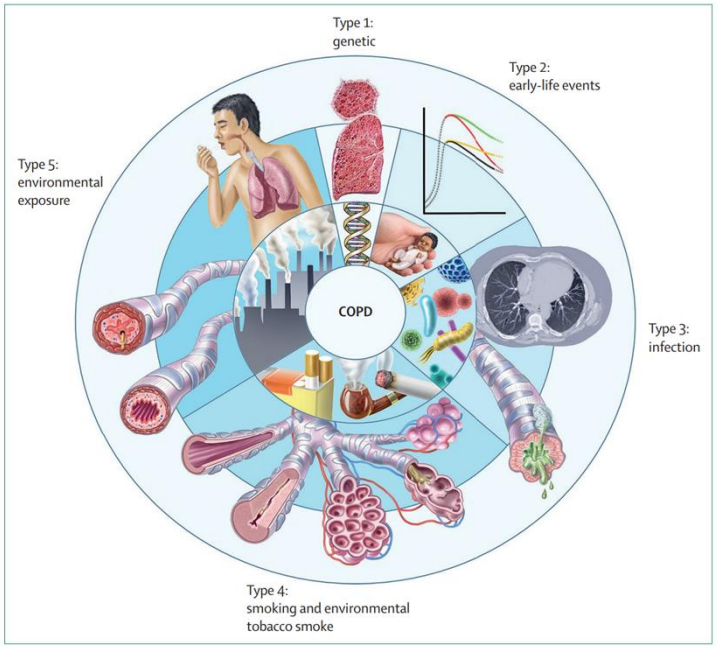
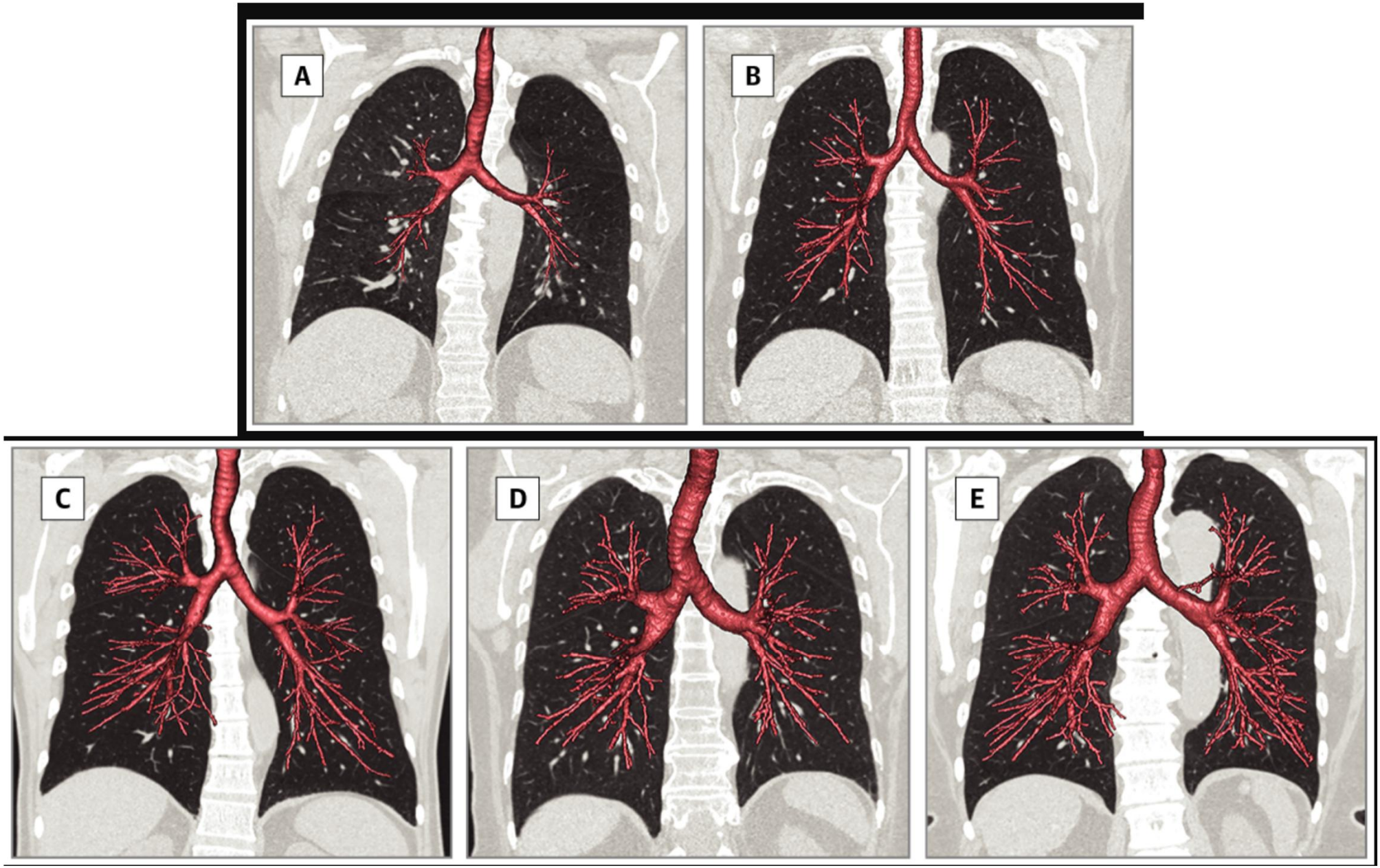


Figure 6: **Proposed classification of COPD according to major risk factors**
The five proposed types are related to genetics, early-life events, infections, exposure to tobacco smoke, and environmental exposures. We remain cognisant, however, that individuals are prone to multiple exposures throughout life, which could cause additive or interactive damage to lung health. COPD=chronic obstructive pulmonary disease.

Pathogenesis

- Trajectories of lung function: development and aging
  - Childhood disadvantage factor
  - Telomere shortening
  - Dysanapsis


A
B
C
D
E

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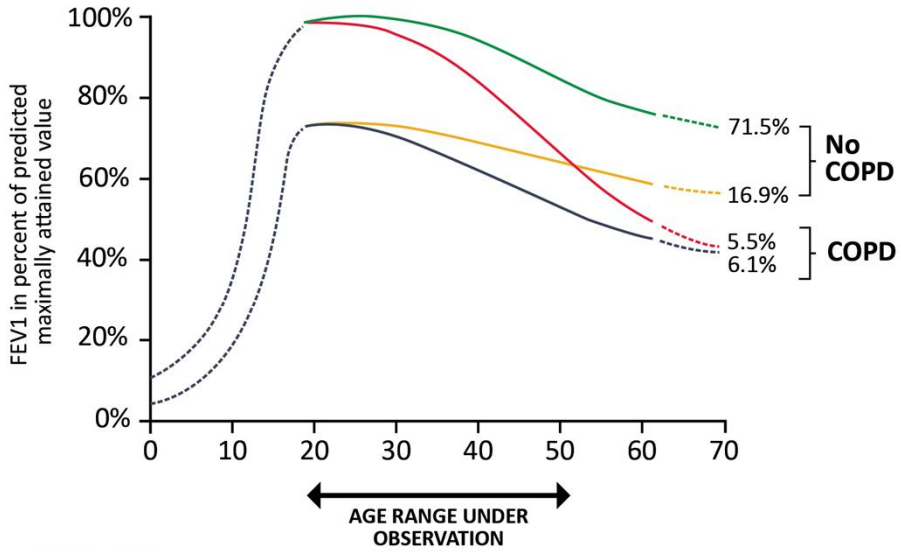
FEV1 Trajectories (TR) Over the Life Course

Figure 1.1



Note: This is a simplified diagram of FEV1 progression over time. In reality, there is heterogeneity in the rate of decline in FEV1 owing to the complex interactions of genes with environmental exposures and risk factors over an individual's lifetime [adapted from Lange et al. NEJM 2015;373:111-22].

Pathogenesis

- Trajectories of lung function: development and aging
  - Early COPD : To discuss th “biological” first steps of the disease
  - Mild COPD : severity of airflow obstruction
  - Young COPD : patients aged 20–50 years
  - Pre-COPD : respiratory Sx or detectable structural or functional abnormality without airflow obstrctruction on forced spirometry
  - PRISm (Preserved Ratio Impaired Spirometry) : FEV1 / FVC > 0.7 and FEV1 < 80%
    - 7.1% ~ 20.3%
    - Associated increased all – cause mortality

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Proposed Taxonomy (Etiotypes) for COPD

Table 1.1

Classification	Description
Genetically determined COPD (COPD-G)	Alpha-1 antitrypsin deficiency (AATD) Other genetic variants with smaller effects acting in combination
COPD due to abnormal lung development (COPD-D)	Early life events, including premature birth and low birthweight, among others
Environmental COPD	
Cigarette smoking COPD (COPD-C)	• Exposure to tobacco smoke, including *in utero* or via passive smoking • Vaping or e-cigarette use • Cannabis
Biomass and pollution exposure COPD (COPD-P)	Exposure to household pollution, ambient air pollution, wildfire smoke, occupational hazards
COPD due to infections (COPD-I)	Childhood infections, tuberculosis-associated COPD, HIV-associated COPD
COPD & asthma (COPD-A)	Particularly childhood asthma
COPD of unknown cause (COPD-U)	

*Adapted from Celli et al. (2022) and Stolz et al. (2022)

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Clinical Indicators for Considering a Diagnosis of COPD

Table 2.1

Consider the diagnosis of COPD, and perform spirometry, if any of these clinical indicators are present:
(these indicators are not diagnostic themselves, but the presence of multiple key indicators increases the probability of the presence of COPD; in any case, spirometry is required to establish a diagnosis of COPD)

Dyspnea that is	Progressive over time Worse with exercise Persistent
Recurrent wheeze	
Chronic cough	May be intermittent and may be unproductive
Recurrent lower respiratory tract infections	
History of risk factors	Tobacco smoke (including popular local preparations) Smoke from home cooking and heating fuels Occupational dusts, vapors, fumes, gases and other chemicals Host factors (e.g., genetic factors, developmental abnormalities, low birthweight, prematurity, childhood respiratory infections etc.)

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Role of Spirometry in COPD

Table 2.5

- **Diagnosis**
- **Assessment of severity of airflow obstruction (for prognosis)**
- **Follow-up assessment**
  - Therapeutic decisions
    - Pharmacological in selected circumstances (e.g., discrepancy between spirometry and level of symptoms)
    - Consider alternative diagnoses when symptoms are disproportionate to degree of airflow obstruction
    - Non-pharmacological (e.g., interventional procedures)
  - Identification of rapid decline

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GOLD Grades and Severity of Airflow Obstruction in COPD (based on post-bronchodilator FEV1)

Table 2.6

In COPD patients (FEV1/FVC < 0.7):

GOLD 1:	Mild	FEV1 ≥ 80% predicted
GOLD 2:	Moderate	50% ≤ FEV1 < 80% predicted
GOLD 3:	Severe	30% ≤ FEV1 < 50% predicted
GOLD 4:	Very Severe	FEV1 < 30% predicted

Modified MRC Dyspnea Scale

Table 2.7

PLEASE TICK IN THE BOX THAT APPLIES TO YOU | ONE BOX ONLY | Grades 0 - 4

mMRC Grade 0	mMRC Grade 1	mMRC Grade 2	mMRC Grade 3	mMRC Grade 4
I only get breathless with strenuous exercise ☐	I get short of breath when hurrying on the level or walking up a slight hill ☐	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 minutes on the level ☐	I am too breathless to leave the house or I am breathless when dressing or undressing ☐

Reference: ATS (1982) Am Rev Respir Dis. Nov;126(5):952-6.

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CAT™ Assessment

Figure 2.2

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	0 X 2 3 4 5	I am very sad	Score
I never cough	0 1 2 3 4 5	I cough all the time	
I have no phlegm (mucus) in my chest at all	0 1 2 3 4 5	My chest is completely full of phlegm (mucus)	
My chest does not feel tight at all	0 1 2 3 4 5	My chest feels very tight	
When I walk up a hill or one flight of stairs I am not breathless	0 1 2 3 4 5	When I walk up a hill or one flight of stairs I am very breathless	
I am not limited doing any activities at home	0 1 2 3 4 5	I am very limited doing activities at home	
I am confident leaving my home despite my lung condition	0 1 2 3 4 5	I am not at all confident leaving my home because of my lung condition	
I sleep soundly	0 1 2 3 4 5	I don't sleep soundly because of my lung condition	
I have lots of energy	0 1 2 3 4 5	I have no energy at all	

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

TOTAL SCORE:

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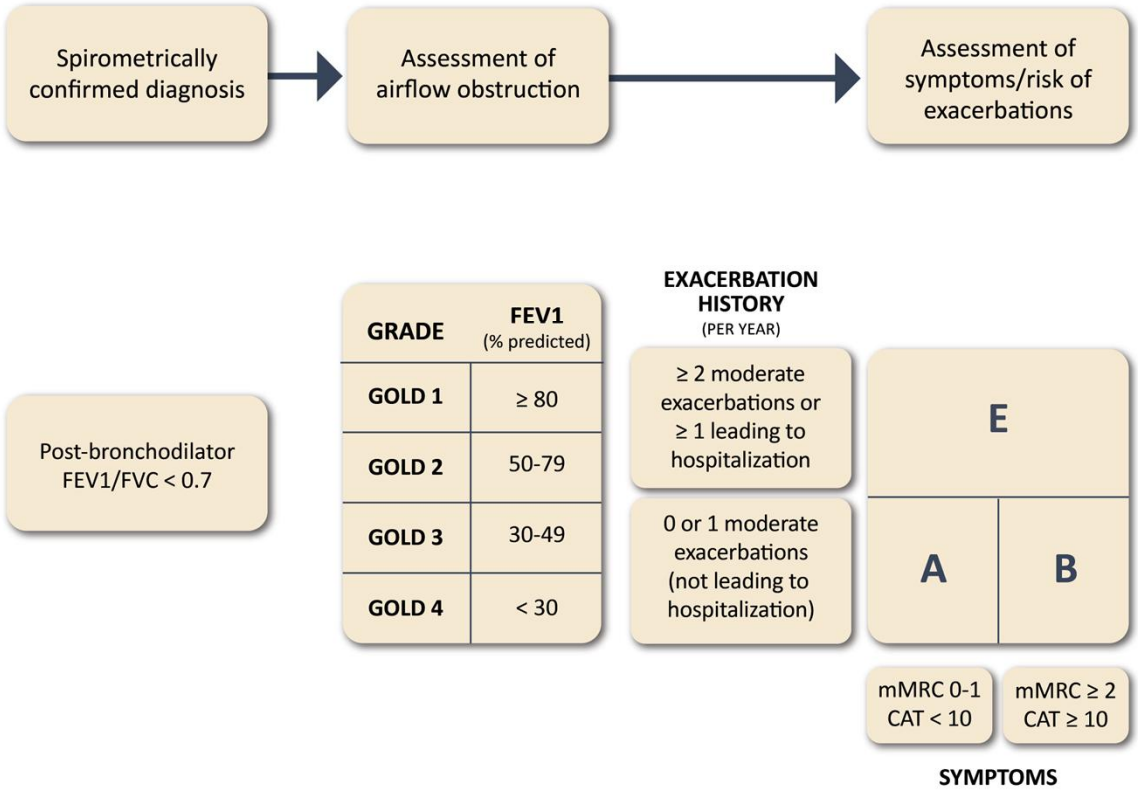
GOLD ABE Assessment Tool

Figure 2.3



Diagnosis

- Additional investigation
  - Physiological test
    - Lung volumes
    - Carbon monoxide diffusing capacity of the lung
    - Oximetry and arterial blood measurement
    - Exercise testing and assessment of physical activity
  - Imaging
    - Chest X-ray
    - Computed tomography

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Use of CT in Stable COPD

Table 2.8

Differential Diagnosis	• Frequent exacerbations with excessive cough with sputum production, raising concern for bronchiectasis or atypical infection • Symptoms out of proportion to disease severity based on lung function testing
Lung Volume Reduction	• Endobronchial valve therapy may be a therapeutic option for patients if they demonstrate postbronchodilator FEV1 between 15-45% and evidence of hyperinflation • Lung volume reduction surgery may be a therapeutic option for patients with hyperinflation, severe upper lobe predominant emphysema and low exercise capacity after pulmonary rehabilitation
Lung Cancer Screening	• Annual low-dose CT scan is recommended for lung cancer screening in patients with COPD due to smoking according to recommendations for the general population

Pharmacological Therapy for Stable COPD

- Bronchodilator
  - Beta2-agonist
    - Relax airway smooth muscle by stimulating beta2-adrenergic receptors
  - Antimuscarinic drug
    - block the bronchoconstrictor effects of acetylcholine on M3 muscarinic receptors expressed in airway smooth muscle
  - Methylxanthines
    - non-selective phosphodiesterase inhibitors
    - Controversy remains about the exact effects
    - Most of the benefit occurs only when near-toxic doses

Pharmacological Therapy for Stable COPD

- Anti-Inflammatory Therapy in Stable COPD
  - Inhaled Corticosteroids
  - Oral Glucocorticoids
    - Long-term use of glucocorticoids has numerous side effects with no evidence of benefits
  - PDE4 Inhibitors
  - Antibiotics
    - Long-term azithromycin and erythromycin therapy reduces exacerbations over one year
    - Treatment with azithromycin is associated with an increased incidence of bacterial resistance and hearing test impairments
  - Mucoregulator and antioxidant Agents
    - Regular treatment with mucolytics such as erdosteine, carbocysteine and NAC reduces the risk of exacerbations in select populations

Factors to Consider when Initiating ICS Treatment

Figure 3.1

Factors to consider when adding ICS to long-acting bronchodilators:

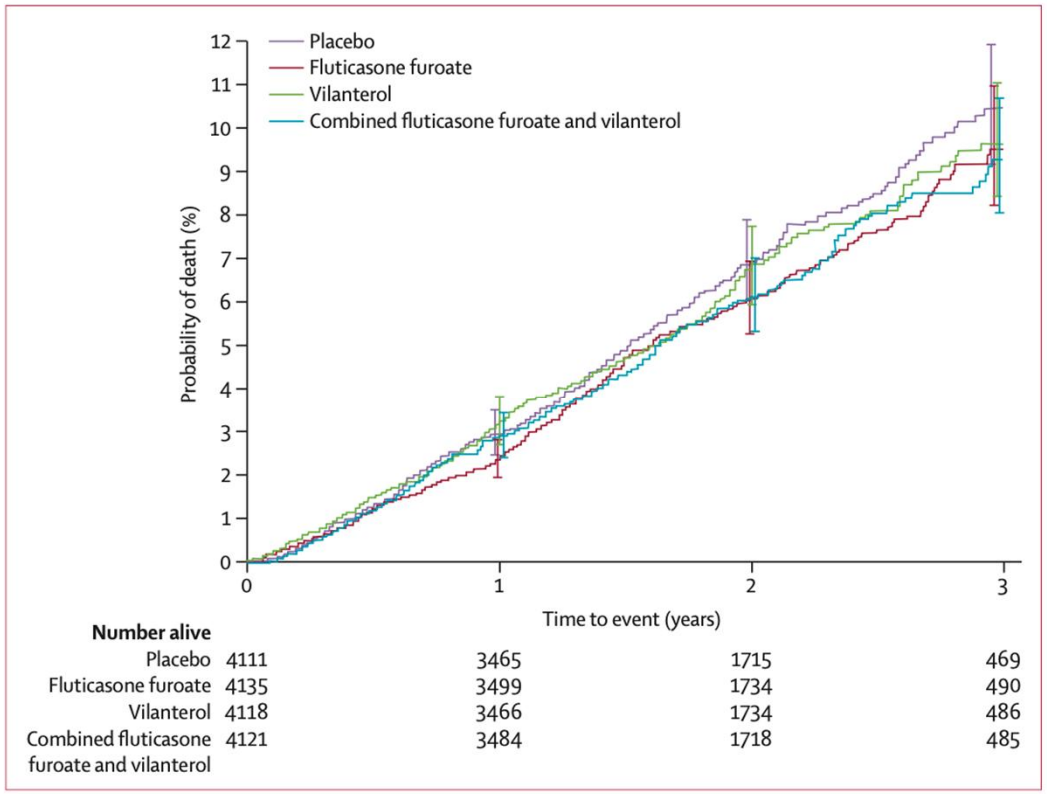
(note the scenario is different when considering ICS withdrawal)

STRONGLY FAVORS USE	**History of hospitalization(s) for exacerbations of COPD#**
	≥ 2 moderate exacerbations of COPD per year#
	Blood eosinophils ≥ 300 cells/µL
	History of, or concomitant asthma
FAVORS USE	1 moderate exacerbation of COPD per year#
	Blood eosinophils 100 to < 300 cells/µL
AGAINST USE	**Repeated pneumonia events**
	Blood eosinophils < 100 cells/µL
	History of mycobacterial infection

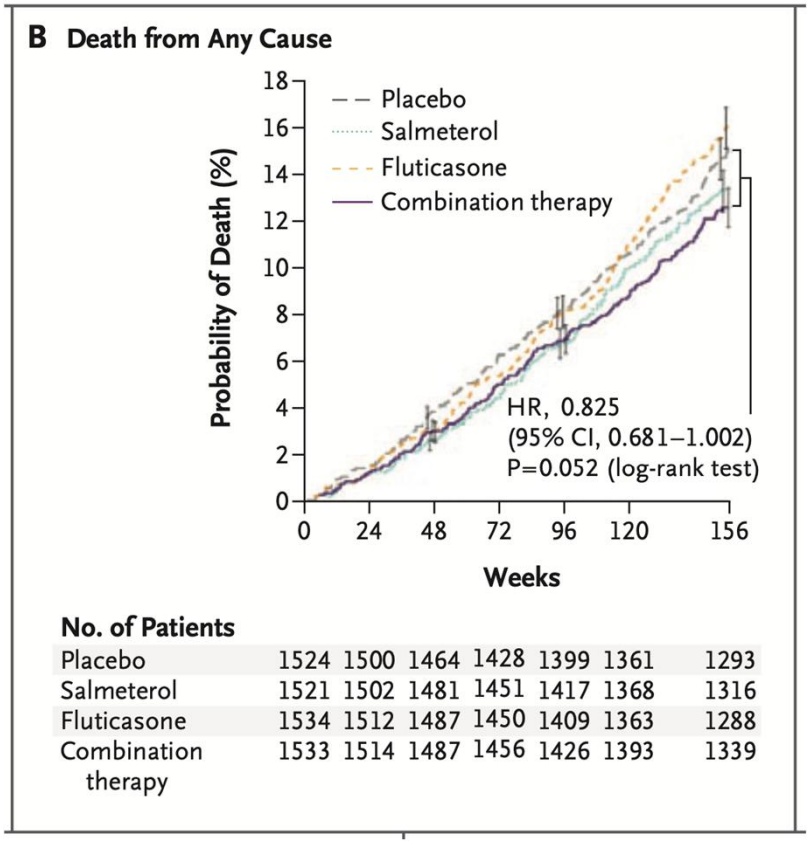
#despite appropriate long-acting bronchodilator maintenance therapy (see Table 3.4 and Figure 4.3 for recommendations); *note that blood eosinophils should be seen as a continuum; quoted values represent approximate cut-points; eosinophil counts are likely to fluctuate.

Adapted from & reproduced with permission of the © ERS 2019: *European Respiratory Journal 52 (6) 1801219; DOI: 10.1183/13993003.01219-2018 Published 13 December 2018*

Pharmacological Therapy for Stable COPD



Lancet 2016; 387: 1817–26



N Engl J Med 2007;356:775-89.

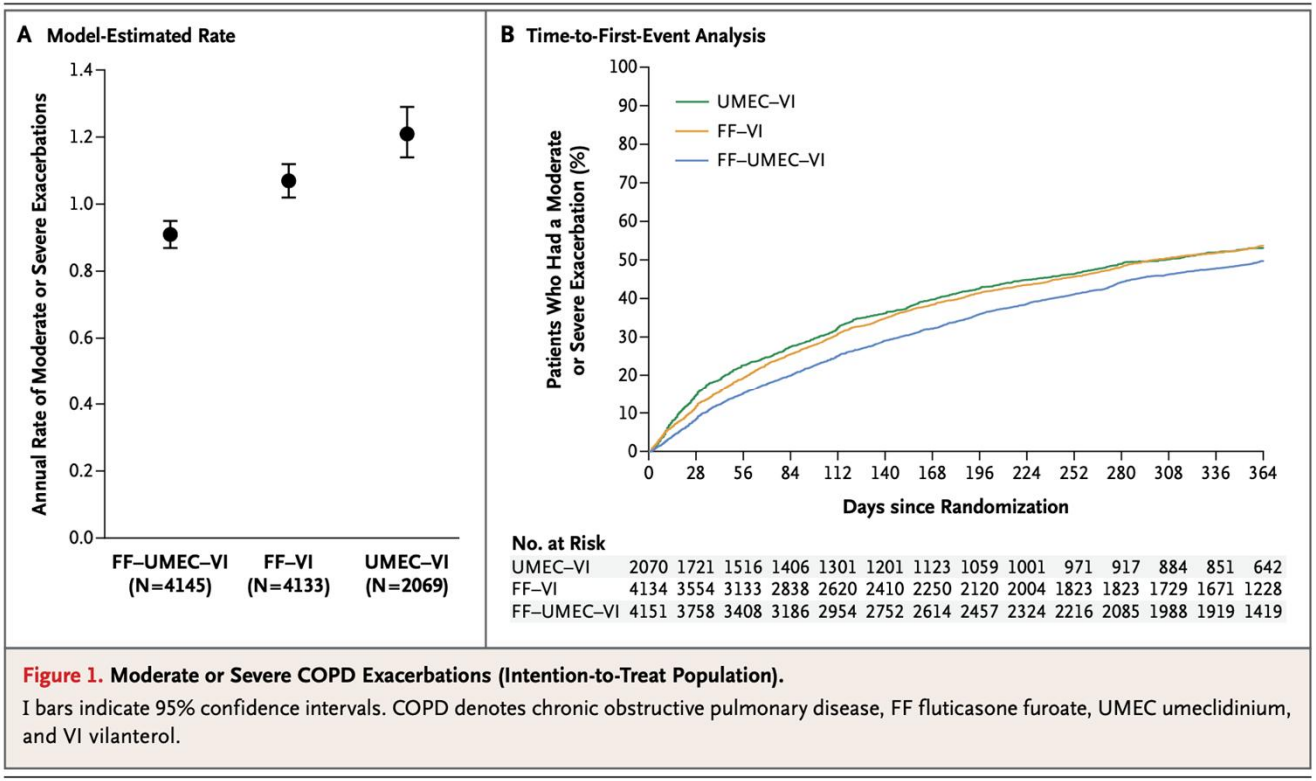
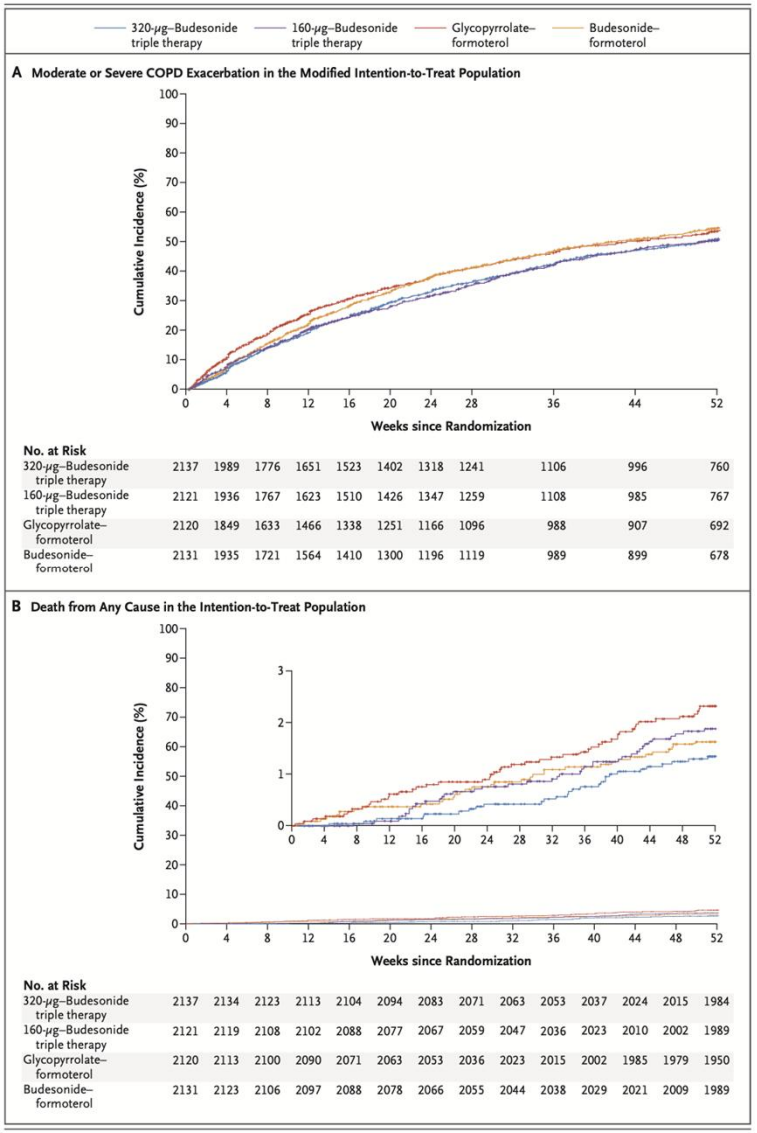


Figure 1. Moderate or Severe COPD Exacerbations (Intention-to-Treat Population).

I bars indicate 95% confidence intervals. COPD denotes chronic obstructive pulmonary disease, FF fluticasone furoate, UMEC umeclidinium, and VI vilanterol.

N Engl J Med 2018;378:1671-80.



N Engl J Med 2020;383:35-48.

Evidence Supporting a Reduction in Mortality with Pharmacotherapy and Non-pharmacotherapy in COPD Patients

Table 3.6

Therapy	RCT*	Treatment effect on mortality	Patient characteristics
Pharmacotherapy			
LABA+LAMA+ICS[1]	Yes	Single inhaler triple therapy compared to dual LABD therapy relative risk reduction: IMPACT: HR 0.72 (95% CI: 0.53, 0.99)[1a] ETHOS: HR 0.51 (95% CI: 0.33, 0.80)[1b]	Symptomatic people with a history of frequent and/or severe exacerbations
Non-pharmacological Therapy			
Smoking cessation[2]	Yes	HR for usual care group compared to intervention group (smoking cessation) HR 1.18 (95% CI: 1.02, 1.37)[2]	Asymptomatic or mildly symptomatic
Pulmonary rehabilitation[3#]	Yes	Old trials: RR 0.28 (95% CI 0.10, 0.84)[3a] New trials: RR 0.68 (95% CI 0.28, 1.67)[3b]	Hospitalized for exacerbations of COPD (during or ≤ 4 weeks after discharge)
Long-term oxygen therapy[4]	Yes	NOTT: ≥ 19 hours of continuous oxygen vs ≤ 13 hours: 50% reduction[4a] MRC: ≥ 15 hours vs no oxygen: 50% reduction[4b]	PaO_2 ≤ 55 mmHg or < 60 mmHg with *cor pulmonale* or secondary polycythemia
Noninvasive positive pressure ventilation[5]	Yes	12% in NPPV (high IPAP level) and 33% in control HR 0.24 (95% CI 0.11, 0.49)[5]	Stable COPD with marked hypercapnia
Lung volume reduction surgery[6]	Yes	0.07 deaths/person-year (LVRS) vs 0.15 deaths/person-year (UC) RR for death 0.47 (p = 0.005)[6]	Upper lobe emphysema and low exercise capacity

*RCT with pre-specified analysis of the mortality outcome (primary or secondary outcome); #Inconclusive results likely due to differences in pulmonary rehabilitation across a wide range of participants and settings.

1. a) IMPACT trial (Lipson et al. 2020) and b) ETHOS trials (Martinez et al. 2021); 2.Lung Health Study (Anthonisen et al. 2005); 3. a) Puhan et al. (2011) and b) Puhan et al. 2016; 4. a) NOTT (NOTT, 1980) and b) MRC (MRC, 1981); 5. Kohlein trial (Kohlein et al. 2014); 6. NETT trial (Fishman et al. 2003)

ICS: inhaled corticosteroid; IPAP: inspiratory positive airway pressure; LABA: long-acting beta$_2$-agonist; LABD: long-acting bronchodilator; LAMA: long-acting anti-muscarinic; LTOT: long-term oxygen therapy; NPPV: noninvasive positive pressure ventilation; LVRS: lung volume reduction surgery; UC: usual treatment control group.

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Oxygen Therapy and Ventilatory Support in Stable COPD

Table 3.10

Oxygen Therapy	• The long-term administration of oxygen increases survival in patients with severe chronic resting arterial hypoxemia **(Evidence A)** • In patients with stable COPD and moderate resting or exercise-induced arterial desaturation, prescription of long-term oxygen does not lengthen time to death or first hospitalization or provide sustained benefit in health status, lung function and 6-minute walk distance **(Evidence A)** • Resting oxygenation at sea level does not exclude the development of severe hypoxemia when traveling by air **(Evidence C)**
Ventilatory Support	• NPPV may improve hospitalization-free survival in selected patients after recent hospitalization, particularly in those with pronounced daytime persistent hypercapnia ($PaCO_2$ > 53 mmHg) **(Evidence B)**

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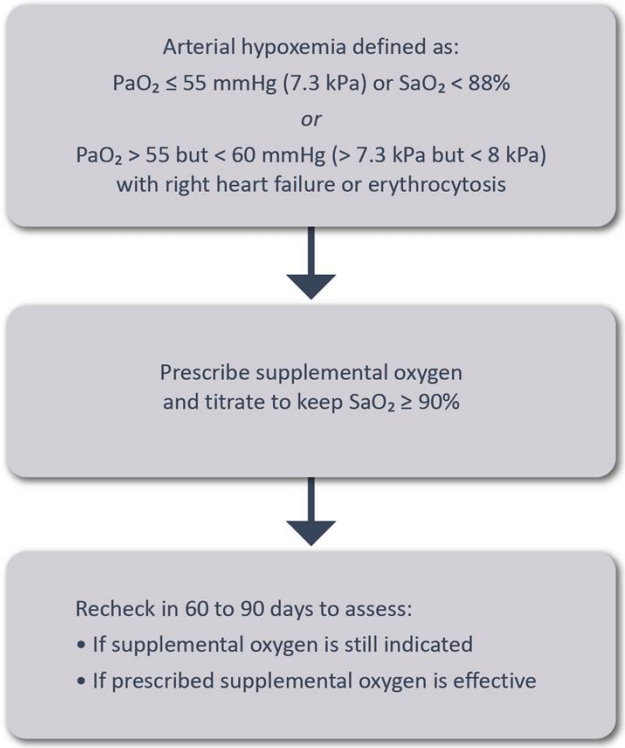
Prescription of Supplemental Oxygen to COPD Patients

Figure 4.5



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Interventional Therapy in Stable COPD

Table 3.11

Lung Volume Reduction Surgery	• Lung volume reduction surgery improves survival in severe emphysema patients with an upper-lobe emphysema and low post-rehabilitation exercise capacity **(Evidence A)**
Bullectomy	• In selected patients, bullectomy is associated with decreased dyspnea, improved lung function and exercise tolerance **(Evidence C)**
Transplantation	• In appropriately selected patients with very severe COPD, lung transplantation has been shown to improve quality of life and functional capacity **(Evidence C)**
Bronchoscopic Interventions	• In select patients with advanced emphysema, bronchoscopic interventions reduce end-expiratory lung volume and improve exercise tolerance, health status and lung function at 6-12 months following treatment. Endobronchial valves **(Evidence A)**; Lung coils **(Evidence B)**; Vapor ablation **(Evidence B)**
Bronchoscopic Interventions Under Study	• Phase III trials are currently being conducted to determine the efficacy of treatments for patients with refractory exacerbations and chronic bronchitis using cryospray, rheoplasty and targeted lung denervation technology

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Goals for Treatment of Stable COPD

Table 4.1

- Relieve Symptoms
- Improve Exercise Tolerance
- Improve Health Status

REDUCE SYMPTOMS

AND

- Prevent Disease Progression
- Prevent and Treat Exacerbations
- Reduce Mortality

REDUCE RISK

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Key Points for the Use of Bronchodilators

Table 4.6

- LABAs and LAMAs are preferred over short-acting agents except for patients with only occasional dyspnea **(Evidence A)**, and for immediate relief of symptoms in patients already on long-acting bronchodilators for maintenance therapy
- When initiating treatment with long acting bronchodilators the preferred choice is a combination of a long-acting muscarinic antagonist and a long acting ß2-agonist. In patients with persistent dyspnea on a single long acting bronchodilator treatment should be escalated to two **(Evidence A)**. The combination can be given as single inhaler or multiple inhaler treatment
- Inhaled bronchodilators are recommended over oral bronchodilators **(Evidence A)**
- Theophylline is not recommended unless other long-term treatment bronchodilators are unavailable or unaffordable **(Evidence B)**

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Key Points for the Use of Anti-Inflammatory Agents

Table 4.7

- Long-term monotherapy with ICS is not recommended **(Evidence A)**
- We do not encourage the use of a LABA+ICS combination in COPD. If there is an indication for an ICS the combination LABA+LAMA+ICS has been shown to be superior to LABA+ICS and is therefore the preferred choice. This combination can be given as single or multiple inhaler therapy.
- If patients with COPD have features of asthma, treatment should always contain an ICS
- In patients with severe to very severe airflow limitation, chronic bronchitis and exacerbations the addition of a PDE4 inhibitor to a treatment with long acting bronchodilators with/without ICS can be considered **(Evidence B)**
- Preferentially, but not only in former smokers with exacerbations despite appropriate therapy, macrolides, in particular azithromycin, can be considered **(Evidence B)**
- Statin therapy and/or beta-blockers are not recommended for prevention of exacerbations **(Evidence A)**

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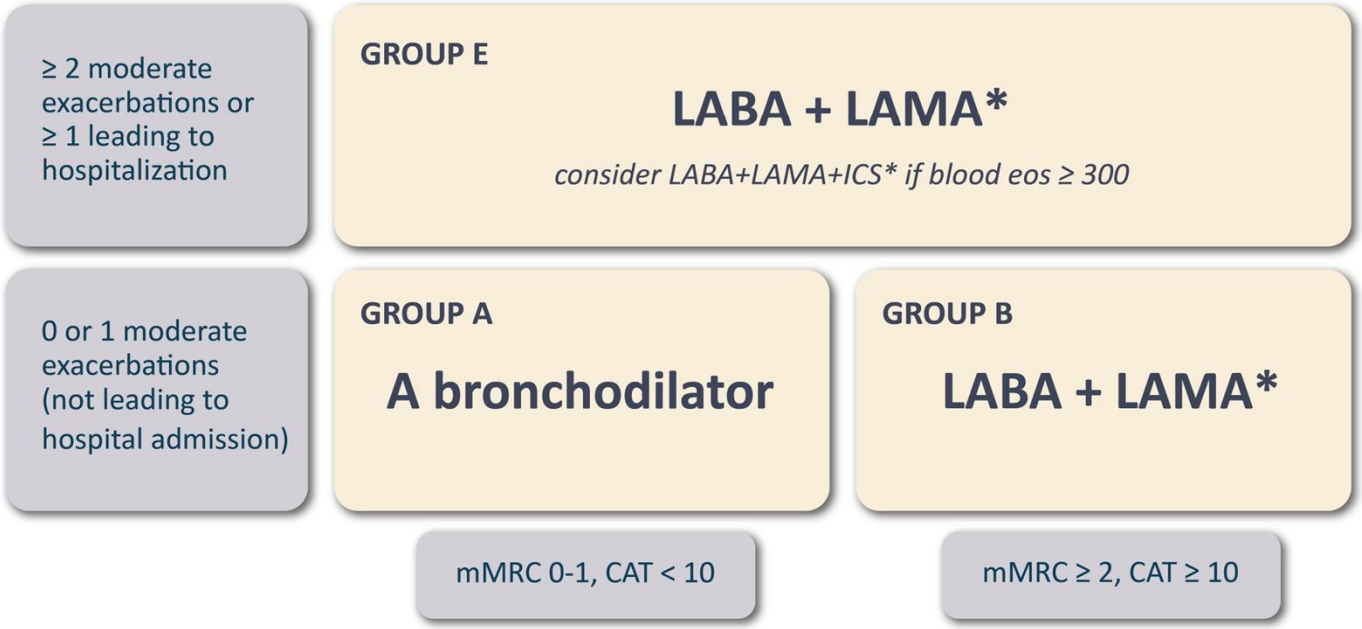
Initial Pharmacological Treatment

Figure 4.2

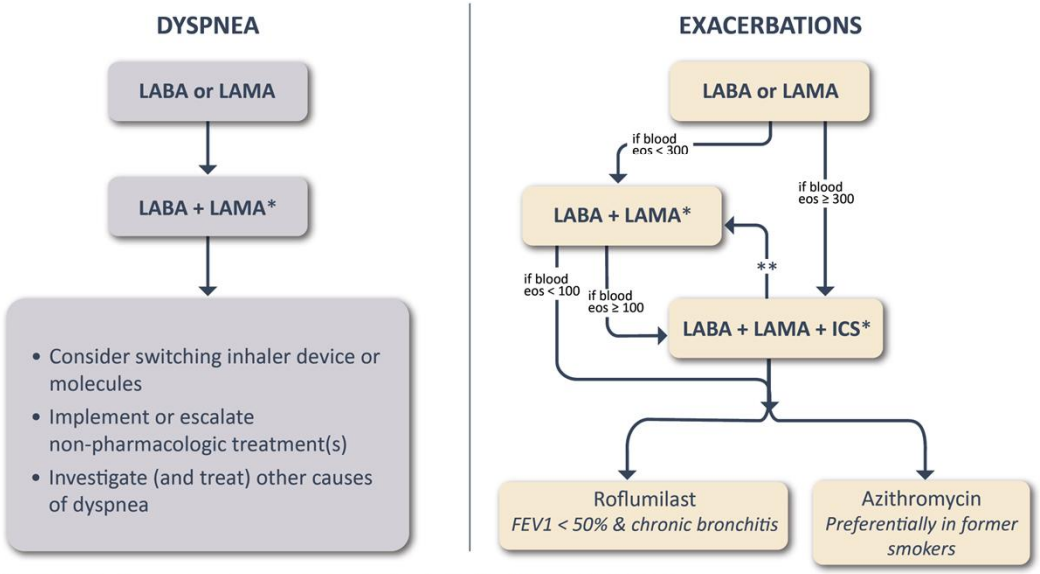


*single inhaler therapy may be more convenient and effective than multiple inhalers
Exacerbations refers to the number of exacerbations per year

Follow-up Pharmacological Treatment

Figure 4.4

1. **IF RESPONSE TO INITIAL TREATMENT IS APPROPRIATE, MAINTAIN IT.**
2. **IF NOT:**
   - Check adherence, inhaler technique and possible interfering comorbidities
   - Consider the predominant treatable trait to target (dyspnea or exacerbations)
     - Use exacerbation pathway if both exacerbations and dyspnea need to be targeted
   - Place patient in box corresponding to current treatment & follow indications
   - Assess response, adjust and review
   - These recommendations do not depend on the ABE assessment at diagnosis



*Single inhaler therapy may be more convenient and effective than multiple inhalers

**Consider de-escalation of ICS if pneumonia or other considerable side-effects. In case of blood eos ≥ 300 cells/µl de-escalation is more likely to be associated with the development of exacerbations

Exacerbations refers to the number of exacerbations per year

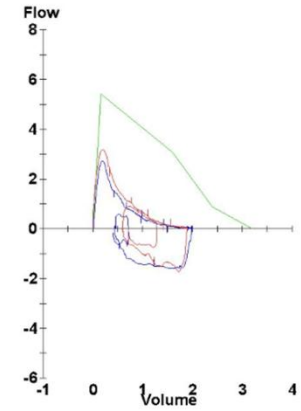
Case #1

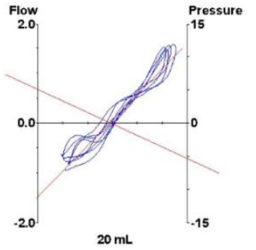
- F/70
- HTN / DM
- 22년 2.10 외래 내원

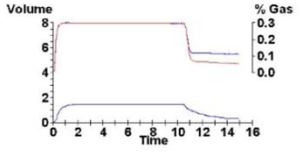
- 반복되는 입원, CO2 retention 주소로 본원 전원
- 타병원 ABGA 7.349 – 61.9 – 70-6 – 34.4 – 92.4%
- 타병원 PFT 0.97L (39.1%) / 1.48L (45%) = 65.54%
- 타병원 medication
  - Theophyline 200mg 1T qd
  - Incruse + relva
  - L-carbocystein 375mg 1T bid
- 타병원 CBC 7.51 >15.7/48.3<213K , #eosinophil #300

Case #1

- 본원 시행 lab
  - 22.02.10 ABGA
    - 7.438 – 48.5 – 52.9 – 32.1% - 88.6%
  - 22.02.24 lab
    - 7700 > 17.2 / 51.5% < 184K # eo 80
  - 22.02.10 PFT
    - 1.14 (47%) / 1.90 (59%) = 60%
- Trelegy / roflumilast
- LTOT : 2L/ min 으로 제한







Age: 68 Height(cm): 160 Weight(kg): 68.0 Gender: Female Race: Asian
Room: IMP Medication:
Dyspnea Rest: No Dyspnea Exercise: No
Cough: No Persistent: No Productive (cc):
Smoker: No How Long(pk/yrs): Stopped(yrs): Cigarettes: No
Technician: so hyun JI Temp: 19 PBar: 762

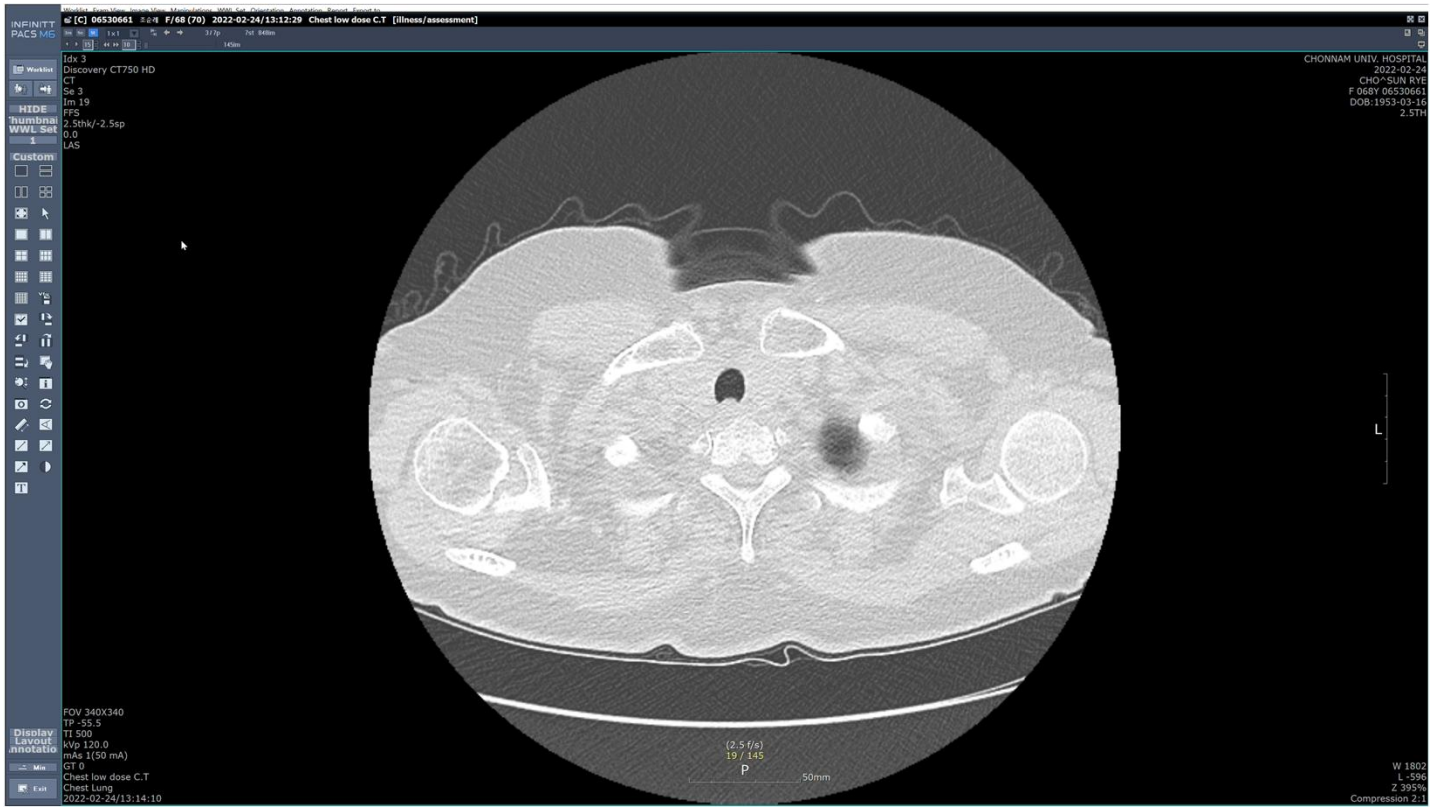
Spirometry		Ref	Pre Meas	Pre % Ref	Post Meas	Post % Ref	Post % Chg
FVC	Liters	3.20	2.00	62	1.90	59	-5
FEV1	Liters	2.39	1.03	43	1.14	47	11
FEV1/FVC	%	76	51		60		
FEF25-75%	L/sec	2.29	0.32	14	0.52	23	63
PEF	L/sec	5.40	2.82	52	3.40	63	21
FET100%	Sec		15.31		9.90		-35
FIVC	Liters	2.76	1.56	57	1.30	47	-17
FIF50%	L/sec		1.34		1.29		-4
FVL ECode			000000		000000		
MVV	L/min	86	38	44			

Lung Volumes				
TLC	Liters	4.77	6.28	132
VC	Liters	2.76	2.00	72
RV	Liters	1.98	4.28	216
FRC PL	Liters	2.66	5.29	199
ERV	Liters	0.92	0.37	40
IC	Liters	1.85	0.99	53
RV/TLC	%	42	68	
Raw	cmH2O/L/sec	1.47	5.38	365
Vtg	Liters		5.58	
sGaw	L/s/cmH2O/L	0.260	0.033	13

Diffusion					Hb:
DLCO	mL/min/mmHg	17.8	13.7	77	
DL Adj	mL/min/mmHg	17.8	13.7	77	
VA	Liters		3.28		
DLCO/VA	mL/min/mHg/L	4.18	4.17	100	
DL/VA Adj	mL/min/mHg/L		4.17		
IVC	Liters		1.46		

Arterial Blood Gases	pH	PCO2 mmHg	PO2 mmHg	HCO3 meq/L	BE	Hb gm/dL	%HbCO %	SaO2 %
Lvl 1								

Case #1


[C] 06530661 F/68 (70) 2022-02-24/13:12:29 Chest low dose C.T [illness/assessment]
Idx 3
Discovery CT750 HD
CT
Se 3
Im 19
FFS
2.5thk/-2.5sp
0.0
LAS
CHONNAM UNIV. HOSPITAL
2022-02-24
CHO^SUN RYE
F 068Y 06530661
DOB:1953-03-16
2.5TH
L
FOV 340X340
TP -55.5
TI 500
kVp 120.0
mAs 1(50 mA)
GT 0
Chest low dose C.T
Chest Lung
2022-02-24/13:14:10
(2.5 f/s)
19 / 145
P
50mm
W 1802
L -596
Z 395%
Compression 2:1

Case #1

22.05.02

- Subjective
  - 숨 쉬는 것은 괜찮다. 산소 쓰니 좋은 것 같다.
  - 두통 없다.
  - 살이 빠지는 것 같다.
- Objective
  - PFT
    - 22.02.10 1.14 / 1.90 = 60% DLCo77% RV 216%
  - ABGA
    - 7.364 - 53.4 - 64.2 - 25.9 - 91.0%
- Assessment
  - COPD
  - respiratory failure type 1 / 2
- Plan
  - O2 2L 이상은 쓰지 않도록
  - headache 동반되면 바로 오도록.
  - daxas D/C
  - 산소 처방전

22.08.11

- Subjective
  - 잘 지냈다. 숨이 찼다.
  - 약을 끊으니 산소 포화도가 안올라간다. / daxas 끊으니 힘들다.
  - 밤에 잘 때 자주 깬다.
- Objective
  - PFT
    - 22.08.11 0.88 / 1.45 = 61% DLCo 44% RV
  - ABGA
    - 7.361 – 57.9 – 53.7 – 27.3 – 85.0%
- Assessment
  - COPD
  - respiratory failure type 1 / 2
- Plan
  - check PFT / ABGA
  - 3개월 뒤 f/u
  - Daxas restart

Case #1

- 23.02.02
  - Subjective
    - 숨이 차다. / 연말에 몸이 안좋았다. / NIV 는 쓰고 있다.
  - Objective
    - PFT
      - 22.02.10 1.14 / 1.90 = 60% DLCo77% RV 216%
      - 22.08.11 0.88 / 1.45 = 61% DLCo 44% RV
      - 23.02.02 0.85 / 1.40 = 60% DLCO 30%
    - ABGA
      - 7.348 - 58.4 - 68.6 - 31.2 - 90.0%
  - Assessment
    - COPD / respiratory failure type 2
  - Plan
    - check PFT / ABGA
    - adm - NIV setting

Case #1

- Adm 23.02.08 ~ 23.02.10
  - ABGA ; 02.10
    - 7.422 – 47.0 – 83.5 – 30.6 – 96.6%
  - PFT ; 23.02.09
    - 1.06(45%) / 1.90(61%) = 56%, DLCo 50%
  - TTE ; 23.02.09
    - EF 65.2%
    - Mild TR(I/IV) with moderate PH (RVSP=55.9mmHg)

Take home message

- GOLD 2023 guideline 에서는 COPD 의 etiology 와 early COPD 에 대한 관심이 강조되었다.
- Stable COPD 의 management를 위한 inhaler는 LABA/LAMA 제제가 기본이 된다.
- ICS 의 역할은 blood eosinophil > 300, 연 2회 이상의 급성 악화인 경우로 제한된다.
- Stable COPD 의 management 에서 ICS / LABA 의 역할은 거의 없다.
- COPD management 시 LTOT, NIV 등 non-pharmacologic approach 또한 중요하다.