Updates in COPD hospitalists should know

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Objectives

- Understand the definition and diagnostic criteria for COPD
- Review important changes in GOLD 2025
- Apply evidence based pharmacological and nonpharmacological therapies in the management of acute and stable COPD
- Recognize interventions that reduce mortality in COPD
- Recognize opportunities for interventions during hospitalization

Outline

- COPD definition, pathogenesis and risk factors
- Assessment and diagnosis
- Key point in the management of stable COPD
- Management of acute exacerbation
- COPD and other comorbidities

What is COPD?

•Heterogeneous lung condition with chronic respiratory symptoms (dyspnea, cough, sputum, exacerbations)

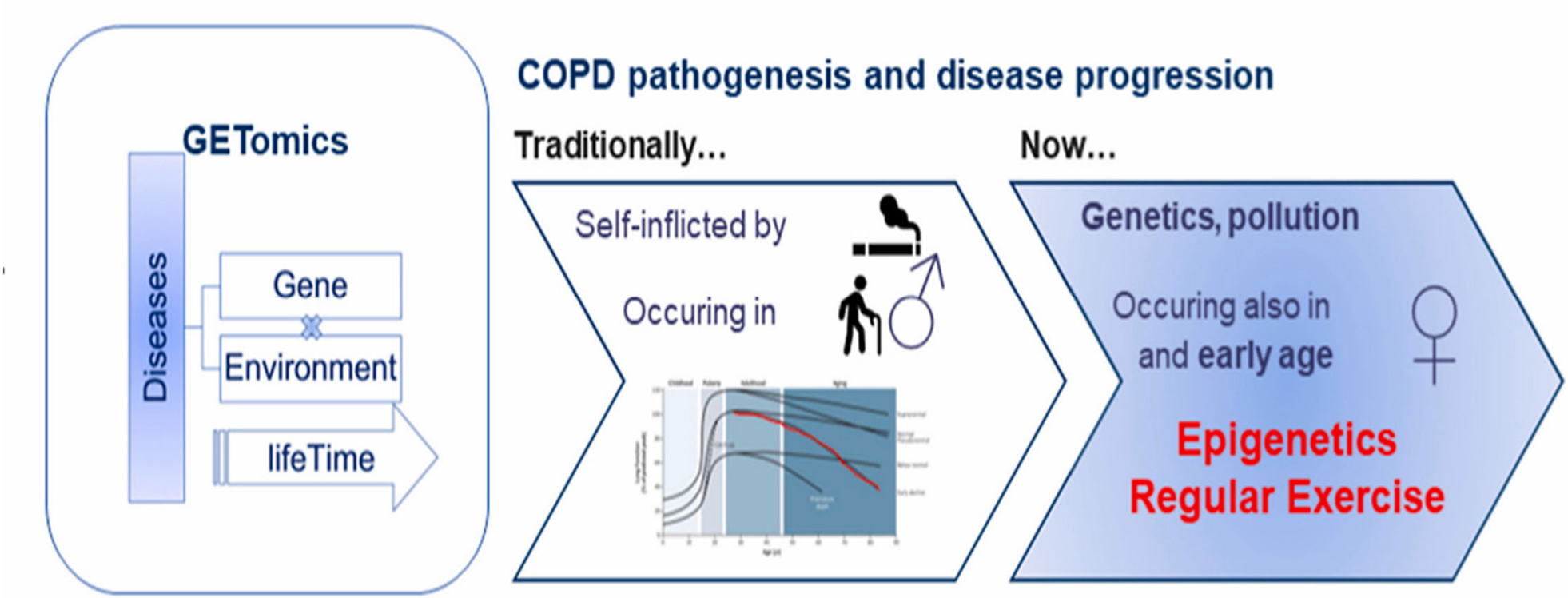
•Caused by airway (bronchitis, bronchiolitis) and/or alveolar (emphysema) abnormalities

•Persistent, often progressive airflow obstruction

Why it is important to know about COPD?

- It is common
- Preventable
- Treatable
- High economic and social burden

Pathogenesis: New understanding



Environmental risk factors

- Cigarette smoking
- Even reduced smoking exposure increases the 5-year risk for developing COPD in middle aged adults
- Other types of tobacco (cigar, pipe, …)
- Environmental tobacco smoker and secondhand smoking
- Yet it is estimated than half of all COPD cases worldwide are due to risk factors other than tobacco

Environmental risk factors

- Biomass exposure
- Occupational exposure
- Air pollution

Genetic risk factors

- The best documented genetic risk factors are the mutations in SERPINA1 gene that leads to the hereditary deficiency of alpha-1 antitrypsin (AATD)

- **Test for AATD in all patients with COPD**

Assessment and diagnosis

- A diagnosis of COPD should be considered in any patient who has dyspnea, chronic cough or sputum production, a history of recurrent lower respiratory tract infections

- And/or a history of exposure to risk factors for the disease

- but spirometry showing the presence of a **post**bronchodilator **FEV1/FVC < 0.7** is **mandatory** to establish the diagnosis of COPD.

Clinical Indicators for Considering a Diagnosis of COPD

Figure 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 non-productive
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.)

Diagnostic updates

- Pre-bronchodilator FEV1/FVC > 0.7 may rule out COPD unless high clinical suspicion (e.g., low FEV1, high symptom burden).
- Post-bronchodilator confirms diagnosis in “volume responders” (FVC increase lowers ratio).
- Debate on fixed ratio (0.7) vs. lower limit of normal (LLN); GOLD retains fixed ratio for simplicity.
- “Pre-COPD” and “PRISm” (Preserved Ratio Impaired Spirometry) for symptomatic patients without airflow obstruction.

A. Spirometry - Normal Trace B. Spirometry - Airflow Obstruction

Figure 2.5

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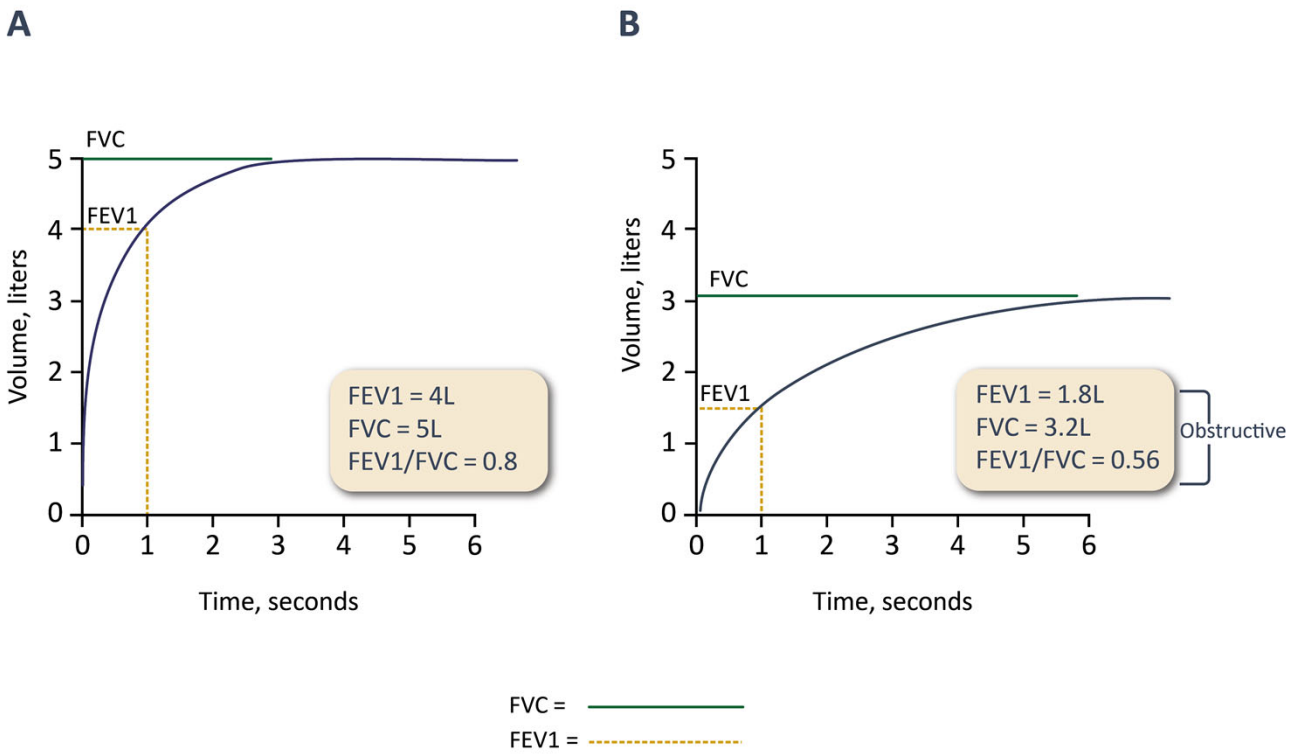
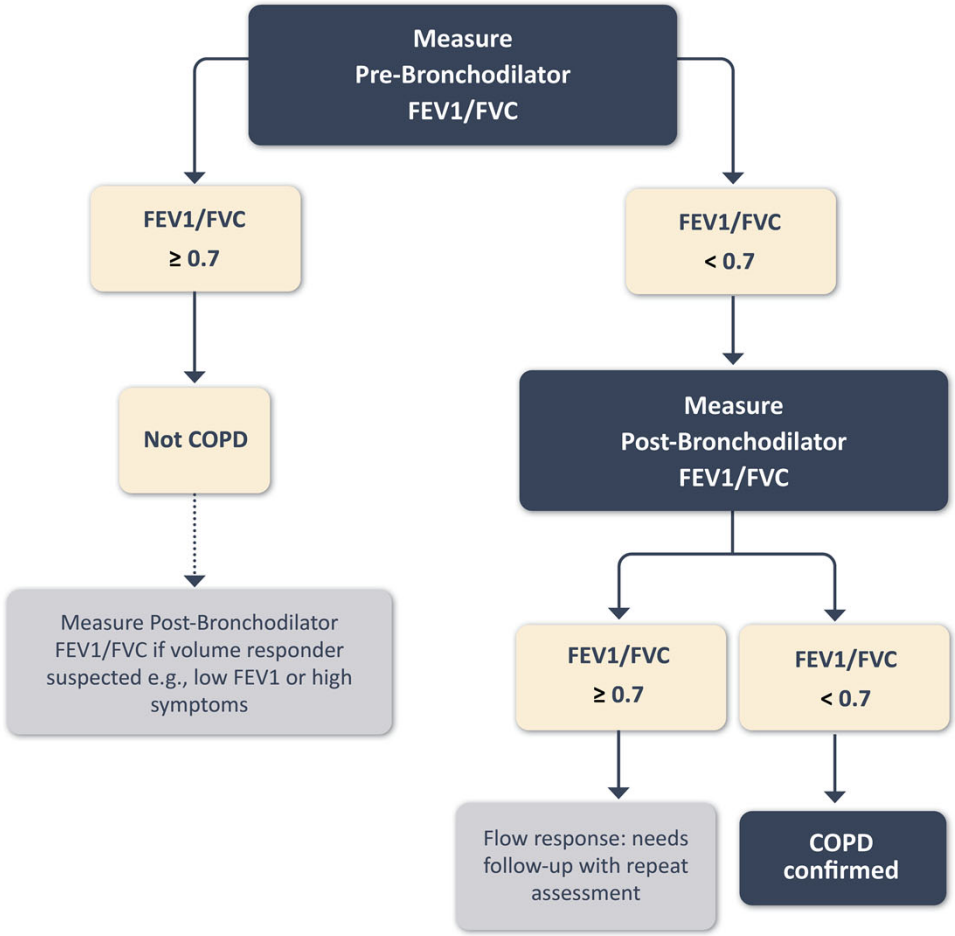


Pre- and Post- Bronchodilator Spirometry

Figure 2.6



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Role of Spirometry in COPD

Figure 2.7

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- **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

GLOBAL INITIATIVE FOR CHRONIC OBSTRUCTIVE LUNG DISEASE™

Pre-COPD and PRISm

PreCOPD: Individuals with respiratory symptoms, lung abnormalities (e.g., emphysema on CT), or physiological changes (e.g., low diffusing capacity) but no spirometric COPD (FEV1/FVC ≥ 0.7). Identifies at-risk individuals for early intervention to prevent COPD progression.

PRISm: Spirometric pattern with normal FEV1/FVC (≥ 0.7) but reduced FEV1 (< 80% predicted). Indicates impaired lung function, potentially restrictive or early COPD-related, with increased symptom and mortality risk.

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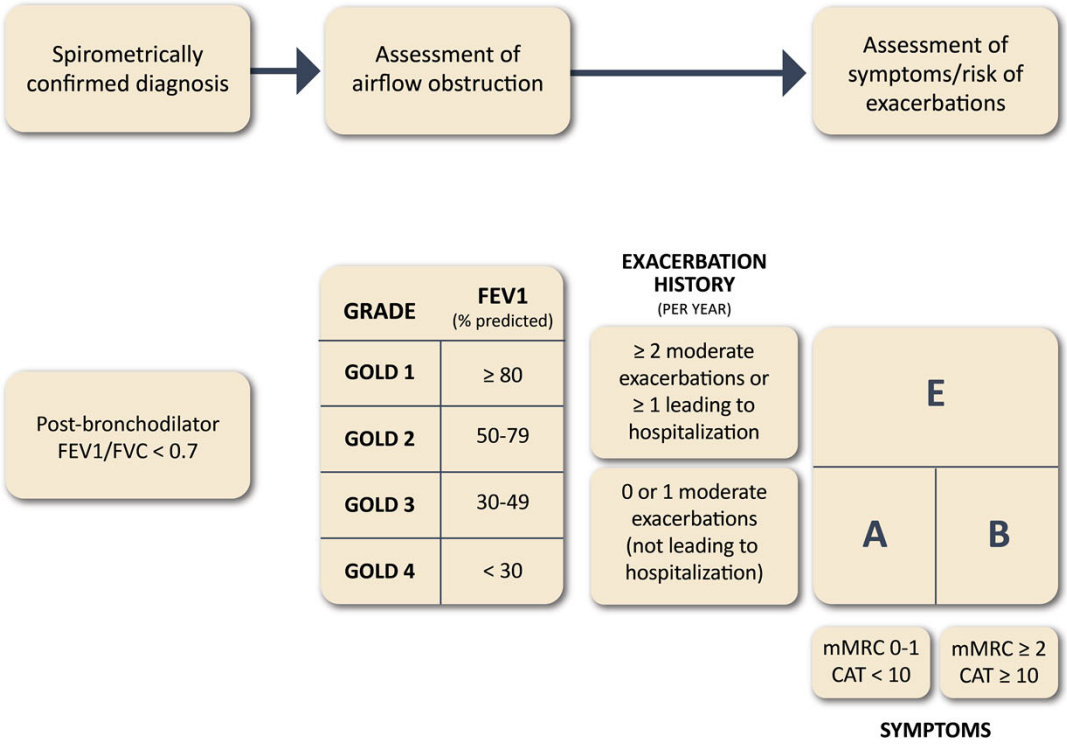
GOLD ABE Assessment Tool

Figure 2.11



Modified MRC Dyspnea Scale

Figure 2.9

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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.

CAT™ Assessment

Figure 2.10

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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: ☐

Smoking cessation

- ✓Smoking cessation should be actively pursued in all patients with COPD
- ✓Effective treatment for tobacco dependence exists
- ✓Even brief 3 minutes counseling improve smoking cessation rates
- ✓Pharmacotherapy: varenicline, bupropion, in addition to NRT
- ✓? E-cigarettes

ORIGINAL ARTICLE

Electronic Nicotine-Delivery Systems for Smoking Cessation

Reto Auer, M.D., Anna Schoeni, Ph.D., Jean-Paul Humair, M.D., M.P.H., Isabelle Jacot-Sadowski, M.D., Ivan Berlin, M.D., Ph.D., Mirah J. Stuber, M.D., Moa Lina Haller, M.D., Rodrigo Casagrande Tango, M.D., M.P.H., Anja Frei, Ph.D., Alexandra Strassmann, Ph.D., Philip Bruggmann, M.D., Florent Baty, Ph.D., Martin Brutsche, M.D., Ph.D., Kali Tal, Ph.D., Stéphanie Baggio, Ph.D., Julian Jakob, M.D., Nicolas Sambiagio, Ph.D., Nancy B. Hopf, Ph.D., Martin Feller, M.D., Nicolas Rodondi, M.D., and Aurélie Berthet, Ph.D.

The addition of e-cigarettes to standard smoking-cessation counseling resulted in greater abstinence from tobacco use among smokers than smoking-cessation counseling alone.

Vaccination for Stable COPD

Figure 3.6

People with COPD should receive all recommended vaccinations in line with the relevant local guidelines:

- Yearly influenza vaccination **(Evidence B)**
- SARS-CoV-2 (COVID-19) vaccination based on WHO and CDC updated recommendations **(Evidence B)**
- Either one dose of 21-valent pneumococcal conjugate vaccine (PCV21) or one dose PCV20, as recommended by the CDC **(Evidence B)**. Pneumococcal vaccination has been shown to reduce the incidence of community-acquired pneumonia and exacerbations for people with COPD **(Evidence B)**
- Respiratory syncytial virus (RSV) vaccination for individuals aged ≥ 60 years and/or with chronic heart or lung disease, as recommended by the CDC **(Evidence A)**
- Tdap (dTaP/dTPa) vaccination to protect against pertussis (whooping cough) for people with COPD that were not vaccinated in adolescence, as recommended by the CDC **(Evidence B)**
- Zoster vaccine to protect against shingles for people with COPD aged > 50 years, as recommended by the CDC **(Evidence B)**

Initial pharmacotherapy

Core Principle: Treatment is tailored based on symptom burden (e.g., dyspnea, assessed via mMRC or CAT scores) and exacerbation history.

Bronchodilators

Long-Acting Bronchodilators: Long-acting beta-agonists (LABA) or long-acting muscarinic antagonists (LAMA) are the cornerstone for most patients with confirmed COPD.

•LABA or LAMA monotherapy is recommended for patients with mild symptoms (e.g., mMRC 0–1, CAT <10) and low exacerbation risk.

Generic Drug Name	Inhaler Type	Nebulizer	Oral/Injectable Delivery	Duration of Action
Long-acting (LABA)				
Arformoterol		✓		12 hours
Formoterol	DPI	✓		12 hours
Indacaterol	DPI			24 hours
Olodaterol	SMI			24 hours
Salmeterol	MDI & DPI			12 hours
Long-acting (LAMA)				
Aclidinium bromide	DPI			12 hours
Glycopyrronium bromide	DPI		solution	variable
Tiotropium	DPI, SMI, MDI			24 hours
Umeclidinium	DPI			24 hours
Glycopyrronium		✓		12 hours
Revefenacin		✓		24 hours

- **Dual Bronchodilator Therapy** (LABA + LAMA) is preferred for patients with moderate-to-severe dyspnea (mMRC ≥2, CAT ≥10) or a history of exacerbations

- It improves lung function, symptoms, and quality of life compared to monotherapy.

- Decreased time to first exacerbation.

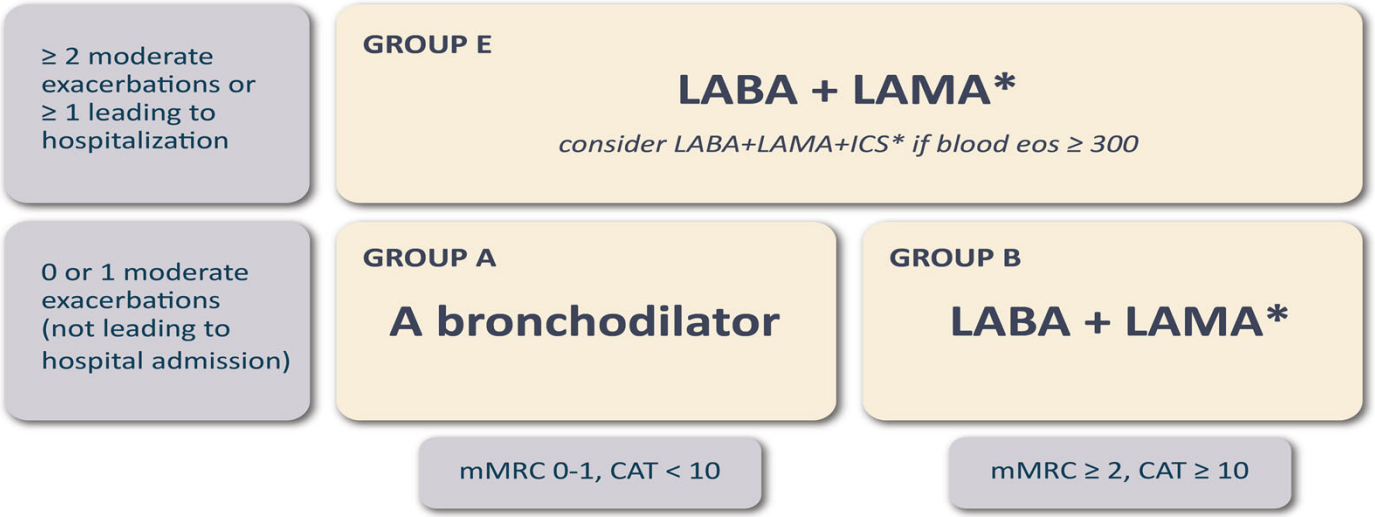
Generic Drug Name	Inhaler Type	Nebulizer	Oral/Injectable Delivery	Duration of Action
Combination Long-Acting Beta$_2$-Agonist Plus Anticholinergic in One Device (LABA+LAMA)				
Formoterol/aclidinium	DPI			12 hours
Formoterol/glycopyrronium	MDI			12 hours
Indacaterol/glycopyrronium	DPI			12-24 hours
Vilanterol/umeclidinium	DPI			24 hours
Olodaterol/tiotropium	SMI			24 hours

- **Short-Acting Bronchodilators** (e.g., SABA or SAMA) are used as rescue therapy for all patients to manage acute symptoms.

Generic Drug Name	Inhaler Type	Nebulizer	Oral/Injectable Delivery	Duration of Action
Short-acting (SABA)				
Fenoterol	MDI	✓	tablet, solution	variable
Levalbuterol	MDI	✓		variable
Salbutamol (albuterol)	MDI & DPI	✓	syrup, tablet	variable
Terbutaline	DPI		tablet	variable
Short-acting (SAMA)				
Ipratropium bromide	MDI	✓		6-8 hours
Oxitropium bromide	MDI	✓		7-9 hours
Combination Short-Acting Beta$_2$-Agonist Plus Anticholinergic in One Device (SABA+SAMA)				
Fenoterol/ipratropium	SMI	✓		6-8 hours
Salbutamol/ipratropium	SMI, MDI	✓		variable

Initial Pharmacological Treatment

Figure 3.7



*Single inhaler therapy may be more convenient and effective than multiple inhalers; single inhalers improve adherence to treatment

Exacerbations refers to the number of exacerbations per year; eos: blood eosinophil count in cells per microliter; mMRC: modified Medical Research Council dyspnea questionnaire; CAT™: COPD Assessment Test™.

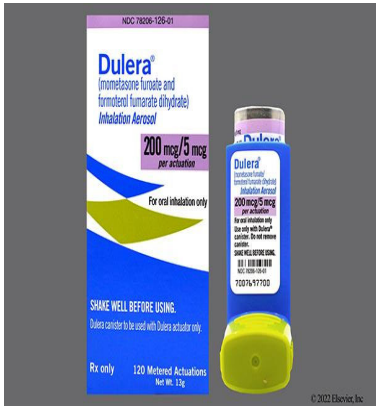
Inhaled corticosteroids (ICS)

• ICS is not recommended as monotherapy but is used in combination (e.g., ICS + LABA or triple therapy) for patients with frequent exacerbations (≥2 moderate or ≥1 severe per year) and elevated blood eosinophil counts (>100 cells/μL).

• ICS use is guided by **blood eosinophil levels** to predict response:

- >300 cells/μL: Strong benefit for exacerbation reduction.
- 100–300 cells/μL: Moderate benefit.
- <100 cells/μL: Minimal benefit; ICS may not be indicated unless exacerbations persist.

Generic Drug Name	Inhaler Type	Nebulizer	Oral/Injectable Delivery	Duration of Action
Combination of Long-Acting Beta$_2$-Agonist Plus Corticosteroid in One Device (LABA+ICS)				
Formoterol/beclometasone	MDI, DPI			12 hours
Formoterol/budesonide	MDI, DPI			12 hours
Formoterol/mometasone	MDI			12 hours
Salmeterol/fluticasone propionate	MDI, DPI			12 hours
Vilanterol/fluticasone furoate	DPI			24 hours
Triple Combination in One Device (LABA+LAMA+ICS)				
Fluticasone/umeclidinium/vilanterol	DPI			24 hours
Beclometasone/formoterol/glycopyrronium	MDI, DPI			12 hours
Budesonide/formoterol/glycopyrrolate	MDI			12 hours



TORCH and SUMMIT

- The **TORCH** and **SUMMIT** trial failed to show evidence for the efficacy of LABA+ICS combination compared to placebo in reducing mortality in COPD patients.

FLAME trial

- Randomized 3300 patients with COPD and high exacerbation risk to indacaterol–glycopyrronium once daily versus salmeterol–fluticasone twice daily

- found the LAMA/LABA combination reduced the annual exacerbation rate by 11% compared to ICS/LABA

- Additionally, the salmeterol-fluticasone group had a higher rate of pneumonia (4.8 vs 3.2%)

IMPACT trial

- Randomized over 10000 subjects to triple therapy (ICS/LAMA/LABA) or dual therapy (LABA/ICS or LABA/LAMA).

- Triple therapy resulted in fewer exacerbations (0.91/year) compared to LABA/ICS (1.07/year), which in turn was superior to LABA/LAMA (1.21/year).

TRIBUTE trial

- Compared triple therapy to LABA/LAMA among 1,500 patients with severe, symptomatic COPD
- found triple therapy had lower rates of moderate to severe exacerbations (0.5/year) as compared to LABA/LAMA group (0.59/year, p = .04) - several years of treatment to prevent 1 exacerbation.
- The benefit of triple therapy was more pronounced in patients with a chronic bronchitis phenotype or with > 2% eosinophils at baseline.

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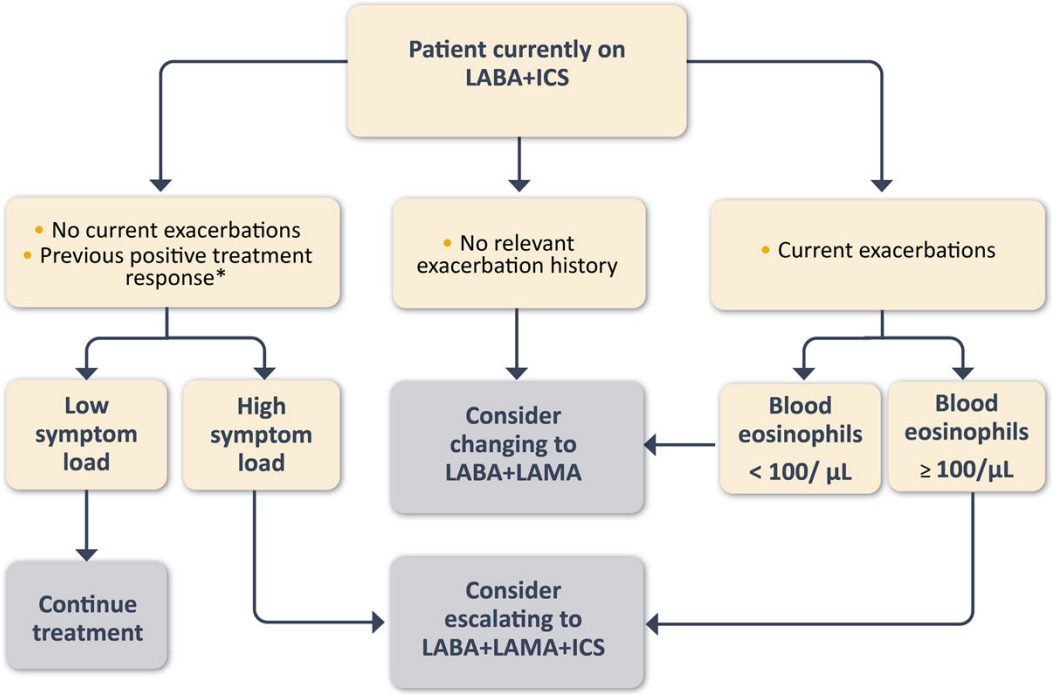
Management of Patients Currently on LABA+ICS

Figure 3.22



*Patient previously had exacerbations and responded to LABA+ICS treatment

- The **SUNSET** trial compared de-escalation to LABA/LAMA vs. continued triple therapy in patients with moderate-to-severe COPD and infrequent exacerbations. Direct de-escalation to LABA/LAMA led to a small decrease in lung function (26 mL), with no difference in exacerbations.

- Of note, a subgroup of patients with eosinophil counts >300 at baseline had greater lung function loss and higher exacerbation risk.

Technique is critical

- The importance of education and training in inhaler device technique can not be overemphasized
- The choice of inhaler device will depend on access, cost, and most importantly patient's ability and preference
- Demonstrate proper inhalation technique when prescribing a device


MakeAGIF.com

Ensifentrine

- **Class**: Inhaled phosphodiesterase (PDE) 3/4 inhibitor, providing both bronchodilator and anti-inflammatory effects.
- **Indication**: Added to dual bronchodilator therapy (LABA + LAMA) for patients with persistent dyspnea or exercise limitation, particularly those with moderate-to-severe COPD.
- **Evidence**: Clinical trials show significant improvement in lung function (FEV1) and symptom scores, but its role in reducing exacerbations is less clear when combined with LABA + LAMA or triple therapy.

Dupilumab

- **Class**: Monoclonal antibody (biologic) targeting IL-4R and IL-13 pathways, reducing eosinophilic inflammation.

- **Indication**: First biologic approved for COPD, specifically for patients with frequent exacerbations and blood eosinophil counts >100 cells/µL.

- **Evidence**: Reduces exacerbation rates and improves lung function in eosinophilic COPD, particularly in patients with type 2 inflammation.

Mepolizumab

- **Class**: a humanized monoclonal antibody that targets interleukin-5

- **Evidence** : Treatment with mepolizumab led to a lower annualized rate of moderate or severe exacerbations when added to background triple inhaled therapy among patients with COPD and an eosinophilic phenotype (≥300 cells per microliter)

Oxygen therapy

- Long term oxygen therapy (LTOT) > 15 hours per day for patients with chronic respiratory failure has been shown to increase survival in severe resting hypoxemia.

- In patients with Stable COPD and moderate resting or exercise induced arterial desaturation, LTOT Does not lengthen time to death or first hospitalization or provide sustained benefit in health status, malfunction and 6-minute walk distance.

Indications

- $PaO_2 \leq 55$ mmHg or $SaO_2 \leq 88\%$ with or without hypercapnia confirmed twice over a 3-week period.

- PaO_2 55-60 mmHg or SaO_2 of 88% if there is evidence of pulmonary hypertension or polycythemia (Hct > 55%)

Ventilatory support

- NPPV may improve hospitalization free survival in selected patients **after** recent hospitalization, particularly in those with persistent daytime hypercapnia ($PaCO_2$>53 mmHg)

- in patients with severe chronic hypercapnia and the history of hospitalization for acute respiratory failure, long term and invasive ventilation may be considered

Interventional and surgical therapies

- Lung volume reduction surgery (LVRS)
- Lung transplantation
- Bronchoscopic interventions (stents, EBV, thermal ablation, ..,)

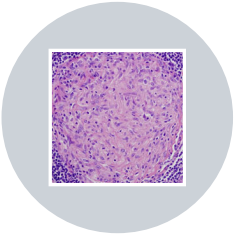
Evidence Supporting a Reduction in Mortality with Pharmacotherapy and Non-pharmacotherapy in COPD Patients

Figure 3.17

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

Exacerbations

Event characterized by dyspnea, and/or cough, and sputum production that worsen over < 14 days



Increased local and systemic inflammation

Symptoms are not specific

Triggers are mainly respiratory viral infections

When to admit?

- Severe symptoms
- Acute respiratory failure
- Failure to respond to initial medical management
- Presence of serious medical comorbidities
- Insufficient home support

Pharmacological therapy

- Bronchodilators
- Corticosteroids
- Antibiotics

Bronchodilators

SABA with or without SAMA are the initial bronchodilators for acute treatment.

No significant difference in FEV1 between using MDIs or nebulizers.

LABA when patients become more stable.

No role for IV methylxanthines.

Corticosteroids

- Shorten recovery time, improve lung function, oxygenation, risk of early relapse, treatment failure and length of hospital stay.
- 40 mg prednisone equivalent for 5 days. Longer courses may be associated with increased risk of pneumonia and mortality.
- PO and IV are equally effective
- ? Less effective in patients with low eosinophils

Antibiotics

- Remains controversial
- evidence supports use of antibiotics when patients have clinical signs of bacterial infections.
- Should be guided by local resistance pattern and previous cultures
- Duration of =< 5 days is recommended
- Use of biomarkers

Respiratory support

- Oxygen therapy
- High flow nasal therapy
- Ventilatory support (NIV and MV)

Noninvasive mechanical ventilation

- Preferred initial mode of ventilation
- Succes rate 80-85%
- Improve oxygenation and acute respiratory acidosis
- Decrease respiratory rate, sensation of breathlessness
- Decrease length of hospital stay

Indications for Noninvasive Mechanical Ventilation (NIV)

Figure 4.8

At least one of the following:

- Respiratory acidosis ($PaCO_2 \geq 6.0$ kPa or 45 mmHg and arterial pH ≤ 7.35)
- Severe dyspnea with clinical signs suggestive of respiratory muscle fatigue, increased work of breathing, or both, such as use of respiratory accessory muscles, paradoxical motion of the abdomen, or retraction of the intercostal spaces
- Persistent hypoxemia despite supplemental oxygen therapy

Prognosis

- Long term prognosis is poor with 5-year mortality rate of about 50%.

- Increase the risk of adverse cardiovascular events, especially in the first 30 days

Hospital discharge and follow up

- Care bundles at discharge (education, optimization of medications, correction of inhaler technique, early rehabilitation,…)
- Sensible but no evidence to support reduction of readmission rate or cost effectiveness
- One exception is early rehabilitation
- Follow up within one month when possible has been related to less exacerbation related readmissions

Pulmonary rehabilitation

- Indicated in all patients with relevant symptoms and or high risk of exacerbations
- Improves dyspnea, health status and exercise tolerance in stable patients
- Reduces hospitalizations among patients who have had a recent exacerbation (**≤ 4 weeks from prior hospitalization**)
- Reduction in symptoms of anxiety and depression

Palliative and end of life care

- Opiates, neuromuscular stimulation (NMES), oxygen and fans blowing air onto the face can relieve breathlessness.
- In one study, IR morphine extended exercise endurance time in over half of the patients with advanced COPD.
- No beneficial effect of benzodiazepines in relieving dyspnea
- Hospitalization may be a trigger to initiate advanced care planning.

COPD and cardiovascular disease

- Heart failure may mimic or accompany acute COPD
- Increased risk of cardiovascular event during and for at least 90 days after acute COPD exacerbation
- Atrial fibrillation is frequent and associated with lower FEV1

Other comorbidities

- Lung cancer is frequently seen in COPD and is a major cause of death
- Osteoporosis, anxiety/ depression are often under-diagnosed and are associated with poor health status and prognosis
- GERD is associated with increased risk of exacerbations and poorer health status

Take home message

- COPD diagnosis relies on post-bronchodilator FEV1/FVC < 0.7
- Pharmacotherapy prioritizes LABA + LAMA or triple therapy; new drugs (ensifentrine, dupilumab, mepolizumab) introduced.
- Non-pharmacological strategies (rehab, NIV, vaccinations) are critical for acute and stable COPD.
- Hospitalists play a key role in exacerbation management, comorbidity assessment, and discharge planning.

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Thank you

- COPD diagnosis relies on post-bronchodilator FEV1/FVC < 0.7
- Pharmacotherapy prioritizes LABA + LAMA or triple therapy; new drugs (ensifentrine, dupilumab, mepolizumab) introduced.
- Non-pharmacological strategies (rehab, NIV, vaccinations) are critical for acute and stable COPD.
- Hospitalists play a key role in exacerbation management, comorbidity assessment, and discharge planning.