PAKISTAN
CHEST SOCIETY
STRIVING FOR PULMONARY CARE

Clinical Practice Guidelines

Chronic Obstructive Pulmonary Disease

PAKISTAN CHEST SOCIETY-2026

Guidelines On

Chronic Obstructive Pulmonary Disease

2026

PAKISTAN CHEST SOCIETY
STRIVING FOR PULMONARY CARE

Guidelines For The Management Of COPD

Preface

The first COPD guideline was issued in 2005, Second edition issued in 2010; and as the new edition of the COPD guideline is in hand after 05 years, you will quickly recognize this edition differs significantly from its predecessor. There has been much change in the understanding and management of the disease over the past half decade.
These changes are based on updated scientific literatures worldwide and locally, while at the same time maintaining simplicity for the practicing clinicians. PCS has been fortunate to have network of distinguished health professionals to bring COPD to the attention of Governments, public health officials, health care workers and the general public to raise awareness of the burden of COPD and to develop programs for early detection, prevention and approaches to management according to local needs based on the best scientific information available.

According to meta- analysis prevalence of COPD in Pakistan is 13.8%, and it is more common in males than in females. In rural areas, it is commoner in females likely due to exposure to burning biomass fuel.

The most blamed and preventable risk to getting the disease is cigarette smoking. The overall prevalence of smoking in Pakistan is 14.5% (26.1% in males and 5.4% in females). Unfortunately, legislations against its use are not implemented in the country.

Although the disease is not curable, the attainable goals of management are to slow down deterioration, improves symptoms and lung function, thus improving the overall health status of the patient.

COPD patients might need hospitalization during exacerbations but most times it is the primary care physician who has to deal with managing the patients. This guideline will be of help to health care providers at all levels dealing with COPD patients.

Dr. Maqbool A Langove
Chairman
COPD guideline working group

Message by the President
Pakistan Chest Society (PCS)

It is a matter of great pleasure and satisfaction for me that PCS is successfully pursuing its agenda of formulating national guideline on COPD. This publication comprehensively but at the same time concisely sheds light on all the aspects of COPD.

COPD is a major cause of chronic morbidity and mortality throughout the world. It represents an important public health challenge that is both preventable and treatable. This risk is mainly associated with increase rate of smoking especially in Pakistan.

This guideline has covered every aspect related to COPD including pathogenesis, diagnosis and management. It has also covered management during exacerbation. Section on supportive treatment strategies also included like smoking cessation, vaccination, pulmonary rehabilitation etc.

PCS is fortunate that it has galaxy of intellectuals who are coherent to each other and ready to impart their part in the work assigned to them. I am thankful to the guideline working group for their hard work and devotion they paid in term of time without which it was not possible to bring this guideline in your hands.

I hope this guideline will be useful for postgraduate trainees, practicing physicians, pulmonologists, scientists and health workers with interest in COPD. It will become a powerful tool for them to enhance their knowledge, which in turn will positively impact the standard of medical care.

Prof. Shereen Khan
President
Pakistan Chest Society

Message by the Chairman
Guideline Committee, Pakistan Chest Society

It is a matter of great pleasure, pride and satisfaction that guideline for the Management of COPD has been revised by the working group. Governing Council of PCS has mandated the Guideline committee to develop evidence-based guidelines for important pulmonary diseases. Besides this document, guidelines on Asthma, Sleep apnea, Noninvasive ventilation, pre-operative pulmonary risk assessment and guideline on smoking Cessation have already been developed and will distributed during the 14th Biennial Chest Con 2020 in Karachi. It is very encouraging to note that PCS has been consistently working on developing and updating guidelines. These guidelines provide a highly valuable resource for the trainees and practicing physicians.

COPD is a major global cause of chronic morbidity and mortality. This publication comprehensively covers all the aspects related to COPD diagnosis and management. I hope this guideline will be useful for trainees, practicing physicians and health care workers with interest in COPD.

Finally, I would like to acknowledge the hard work of Dr Maqbool and other members of the working group who revised this document and the members of PCS guideline committee for reviewing the document. PCS remain committed to always endeavor for the achievement of the best possible clinical practice.

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Chairman Guideline Committee
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Pakistan Chest Society

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We would like to extend gratitude to Pakistan Chest Society for their trust and giving us the opportunity to prepare this guideline

ABBREVIATIONS

6MWT	Six-minute Walk test
ABGs	Arterial blood gases
AD	Alveolar duct
AECOPD	Acute exacerbation of chronic obstructive pulmonary disease
AFB	Acid fast bacillus
AS	Alveolar sac
BiPAP	Bilevel positive airway pressure
BLVR	Bronchoscopic lung volume reduction surgery
BMI	Body mass index
BODE	Body mass index, Obstruction, Dyspnea, Exercise
BOLD	Burden of Obstructive Lung Disease
cAMP	Cyclic adenosine monophosphate, or 3',5'-cyclic adenosine monophosphate
CAT	Combined assessment test
CI	Confidence interval
CO	Cardiac output
COPD	Chronic obstructive pulmonary disease
CT	Computed tomography
DALYs	Disability adjusted life years
DLCO	Diffusion capacity of lung for carbon monoxide
DPI	Dry powder inhaler
ECM	Extracellular matrix
EMRO	Eastern Mediterranean Regional Office
FEV1	Forced expiratory volume in one second
FVC	Forced vital capacity
GOLD	Global Initiative for Chronic Obstructive Lung Disease
HIV	Human immunodeficiency virus
HR	Hazard ratio; Heart rate
HRQOL	Health related quality of life
ICS	Inhaled corticosteroid
ICU	Intensive care unit
IV	Intravenous
JVP	Jugular venous pulse
LABA	Long-acting beta agonist
LAMA	Long-acting muscarinic antagonist
LLN	Lower limit of normal
LTOT	Long term oxygen therapy
LVRS	Lung volume reduction surgery
MDI	Metered dose inhaler

MMPs	Matrix metalloproteinases
mMRC	Modified medical research council
MV	Minute ventilation
NIV	Noninvasive ventilation
O2	Oxygen
OCS	Oral corticosteroids
OR	Odds ratio
P2	Pulmonic second heart sound
PAO2	Partial pressure of oxygen in alveoli
PaCO2	Partial pressure of carbon dioxide in arterial blood
PaO2	Partial pressure of oxygen in arterial blood
PCS	Pakistan chest society
PCV	Pneumococcal conjugate vaccine
PDE-4	Phosphodiesterase 4 inhibitor
PEEP	Positive end expiratory pressure
PEFR	Peak expiratory flow rate
PHT	Pulmonary hypertension
PIO2	Pressure of inspired oxygen
PPSV	Pneumococcal polysaccharide vaccine
RB	Respiratory bronchiole
RHF	Right heart failure
RSV	Respiratory syncytial virus
RV	Residual volume
SaO2	Arterial oxygen saturation (measured by arterial blood gas analysis)
SARS	Severe acute respiratory syndrome
SERPIN	Serine protease inhibitor
SMI	Soft mist inhaler
SpO2	Peripheral oxygen saturation (measured by pulse oximeter)
SR	Slow release
TB	Tuberculosis; Terminal bronchiole
TLC	Total lung capacity
V: Q	Ventilation: Perfusion
VC	Vital capacity
WHO	World health organization
WOB	Work of breathing

Chapter 01:
Introduction

The Pakistan Chest Society (PCS) guideline was developed by expertise in chronic obstructive pulmonary disease (COPD) and the aim of PCS is to provide evidence-based recommendations, while maintaining simplicity for the assessment, diagnosis and management of COPD for graduates, post-graduates and general practitioners.

The Burden of COPD

Chronic Obstructive Pulmonary Disease (COPD) remains a major global public health challenge. According to a recent systematic review and meta- analysis published in 2024, the global prevalence of COPD in adults aged 40 years and above is estimated at 12.6% when using the fixed ratio (FEV_1/FVC < 0.70) and 7.4% when applying the lower limit of normal (LLN) spirometry criteria. This analysis, which included over 100 population-based studies from 94 countries, highlights substantial regional variation and underscores the burden of underdiagnosed and undertreated disease worldwide[1].

The landmark BOLD (Burden of Obstructive Lung Disease) study, published in 2011, provided compelling evidence that COPD is not exclusive to smokers. Among 4,291 never-smokers aged ≥40, the study found that 6.6% met criteria for mild COPD (GOLD stage I), and 5.6% met criteria for moderate to very severe COPD (GOLD stage II+). Remarkably, never-smokers accounted for 23.3% of all GOLD stage II+ COPD cases in the total sample. Even when using the more conservative lower limit of normal (LLN) threshold for FEV_1/FVC, never-smokers still comprised 20.5% of moderate-to-severe cases.

These findings underline the importance of non-smoking risk factors, including advancing age, lower educational attainment (particularly in women), occupational exposures, childhood respiratory diseases, and abnormal body mass index (BMI). The study concluded that never-smokers represent a substantial and underrecognized proportion of the global COPD burden, challenging the conventional notion that smoking is the sole driver of chronic airflow limitation[2].

Based on BOLD and other large epidemiologic studies the global prevalence of COPD in 2010 was 11.7%3."In addition to imposing a substantial economic burden on individuals and society, COPD leads to significant disability and impaired quality of life. As of 2021, COPD ranked as the **4th leading cause of death globally,** responsible for some 3.5 million deaths (~5% of global mortality), and was the **8th leading cause of disability adjusted life years (DALYs)** - worldwide.

According to a metanalysis done in 2018, Pakistan has the highest prevalence of COPD (13.8%), in the EMRO region.5 The prevalence of COPD increases with rising smoking trend and aging population. Table 1 shows the results of tobacco use prevalence from latest survey completed by end of December 2018.[6]

Table 1: Prevalence of tobacco use (latest survey completed by WHO, December 2018)

	Tobacco use		Tobacco smoking		Cigarette smoking		Smokeless tobacco use		E-cigarette use	
	Current	Daily	Current	Daily	Current	Daily	Current	Daily	Current	Daily
Survey: National Diabetes Survey of Pakistan, 2016-17; National, ages 20+#										
Male	26.1	..	..	..	..	..	11.4*	10,5*	..	..
Female	5.4	..	..	..	..	..	3.7*	3.5*	..	..
Both Sexes	14.5	..	..	..	..	..	7.7*	7.1*	..	..
Survey: Global Youth Tobacco Survey, 2013; National, ages 13-15										
Male	13.3	..	9.2	..	4.8	..	6.4	..	..	..
Female	6.6	..	4.1	..	0.9	..	3.7	..	..	..
Both Sexes	10.7	..	7.2	..	3.3	..	5.3	..	..	..

* Global Adult Tobacco Survey, 2014; National, ages 15+
# 4.4 % men, 1 % women and 2.7 % of the adult population are daily water pipe smokers

A systemic review published in 2011 reported the prevalence of waterpipe (Shisha) smoking to be 33% among university students and 6% among adult population of Pakistan.[7]

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Chapter 02:
Definition Of COPD

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.[1]
In a developing country like Pakistan the impact of COPD on workplace and home productivity are equally important as the direct medical costs related to the disease. Hence COPD is a serious threat to a country's economy.

Early COPD
Early COPD refers to the initial development of structural or functional lung changes that could later lead to full-blown COPD. It may not yet show up as airflow obstruction on spirometry, but early signs—like small airway inflammation or changes on CT scan—can be present.

Key Point: It's about the starting stage of lung damage before spirometry confirms COPD.

Mild COPD
Mild COPD is when a person already has airflow obstruction, but the symptoms are not very severe, and lung function is still relatively preserved (usually $FEV_1 \geq 80\%$ predicted).

Key Point: Airflow limitation is present, but daily activities are usually not much affected.

Young COPD
Young COPD refers to people who develop COPD before the age of 50, often with risk factors like smoking, poor lung growth in childhood, or genetic issues (like alpha-1 antitrypsin deficiency).

Key Point: COPD in a person under 50 years old, which may be due to early-life or genetic factors.

PRISm (Preserved Ratio Impaired Spirometry)
PRISm means the person has a reduced FEV_1 (less than 80% predicted), but the FEV_1/FVC ratio is still normal (≥ 0.70). This pattern may indicate early disease, and some patients with PRISm can go on to develop COPD.

Key Point: Lung function is not normal, but does not yet meet the definition of COPD.

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Chapter 03:
Risk Factors Of COPD

COPD results from Gene(G)-Environment(E) interaction occurring over the lifetime(T) of the individual (GETOmics) that can damage the lungs and/or alter their normal development/aging processes. Many factors contribute to the development of COPD.[1] The most important ones are discussed below.

Cigarette smoking is the most important risk factor for development of COPD.[2] But approximately 25 – 45% of patients with COPD are lifelong non-smokers.[3] On the other hand fewer than 50% of heavy smokers do not develop COPD.[4] Several mechanisms are proposed by which smoking may contribute to COPD.

Prenatal exposure

- Reduced lung development
- Low birth weight Childhood
- Decreased lung growth Adulthood
- Accelerated decline in lung function
- Lung destruction
- Impaired lung repair

The greater the total numbers of cigarette smoke, the greater the risk of development of COPD. It is good to quantify this exposure as under:[5]

Pack year = Number of cigarettes smoked per day x Number of years of smoking 20

Smokers suffer an irreversible loss of 4.4–10.4 ml in FEV1 per pack year smoked.[6,7] There is a strong dose–response relation between the smoking pack years and the risk,[8] severity,[9] and mortality10 of COPD and the risk of lung cancer.[11]
Loose tobacco is quantified as tobacco smoked in "ounces per week", which can be converted into pack years:[12]

Pack years = Ounces of loose tobacco per week × 2/7 × number of years smoked

Indoor and outdoor air pollution like biomass fuel for cooking or heating in poorly ventilated dwellings, passive smoking and exposure to urban air pollution, organic and inorganic dusts, chemicals or fumes. In Pakistan, biomass fuel is used for cooking and heating by 52% of households overall, and 75% in rural area, figure 3.1.[13] The prevalence of COPD is two to three times higher in women exposed to biomass fuel as compared to urban women.[14,15] According to a meta- analysis, biomass-exposed individuals are 1.38 times more likely to be diagnosed with COPD than non-exposed (OR 1.38, 95% CI 1.28 to 1.57).[16]

Figure 3.1: Indoor biomass exposure

Figure 3.2: Inside of the coal mine

Genetic predisposition[17] is possibly related to α1-proteinase inhibitor, α1-antichymotrypsin, α2- macroglobulin, matrix metalloproteinase 12, α-nicotinic acetylcholine receptor, hedgehog interacting protein and many others.
Most common is alpha-1 antitrypsin deficiency. Alph-1 antitrypsin is a serine protease inhibitor (SERPIN) secreted by the liver, which inhibits the enzyme neutrophil elastase from damaging the lung tissue.
Deficiency of this alpha-1 antitrypsin leads to unopposed elastolysis and development of emphysema (protease-antiprotease hypothesis). These patients usually present at an early age.

Infections during childhood may increase subsequent risk of COPD by affecting lung function, lung growth or pulmonary defense mechanisms.[18] HIV patients are an increased risk of COPD than HIV negative controls.[19] TB is an independent risk factor for COPD.[20] Pseudomonas colonization increases the risk of exacerbations, hospitalizations and all-cause mortality of COPD.[21]

Socioeconomic status is a small risk factor and is difficult to separate from related factors such as smoking habit, industrial exposure, passive smoking and childhood infection. There is an increased risk of development of COPD in people of lower socioeconomic class.[22]

It is not clear whether increasing age itself causes changes similar to COPD or it is the cumulative effect of exposure throughout life that leads to COPD. In old literature it is proposed that males are more prone to get COPD, while today due to increasing trend of smoking among females this gender predisposition has become equal. Some studies have suggested that females get more severe disease than males for equal quantity of cigarettes consumed.

Poorly treated asthma and smoking may be a risk factor for development of chronic airflow limitation and COPD. Separating asthma from COPD in adults may be sometimes difficult.

Airway hyper-responsiveness may exist without a diagnosis of asthma and it is an independent predictor of COPD.

Chronic bronchitis has also been associated with increased likelihood of developing COPD and an increased frequency and severity of exacerbations.

Chronic intravenous drug abuse especially cocaine methadone and heroin are linked to higher risk of developing COPD. This is attributed to the vascular damage induced by the insoluble filler (cornstarch, cellulose, talc, fiber etc.) found in IV drugs. These patients are usually young at presentation[2,3].

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Chapter 04:
Pathogenesis

In health the lung shows an inflammatory response to noxious/irritant particles. In COPD this response is modified and pathological changes occur in airways, the lung parenchyma and the pulmonary vasculature. Several mechanisms have been proposed for the development of COPD.[1,2] Oxidative stress caused by excess of oxidants due to cigarette smoke may enhance the inflammatory response. Oxidants also inactivate the antiproteases and cause protease- antiprotease imbalance that leads to the breakdown of connective tissue components in the lung, resulting in emphysema.

Inflammatory cells like macrophages, activated neutrophils and lymphocytes are increased in COPD which release multiple inflammatory mediators. In some patients who have COPD asthma overlap there are increased eosinophils.

In healthy smokers and also in COPD there is **peribronchiolar and interstitial fibrosis** which may contribute to the development of small airway limitation and obliteration that precedes the development of emphysema.

Chronic bronchitis

Chronic bronchitis is defined as:

"Chronic cough and sputum for at least 3 months a year for 2 consecutive years."

It was once considered under the umbrella term of COPD. However, it is now known that chronic bronchitis is a distinct entity that can exist with or without airflow limitation.[3]

	Air flow obstruction	No airflow obstruction
Chronic bronchitis symptoms	COPD & Chronic bronchitis	Chronic bronchitis
No chronic bronchitis symptoms	COPD	None

Small airway disease/ Bronchiolitis

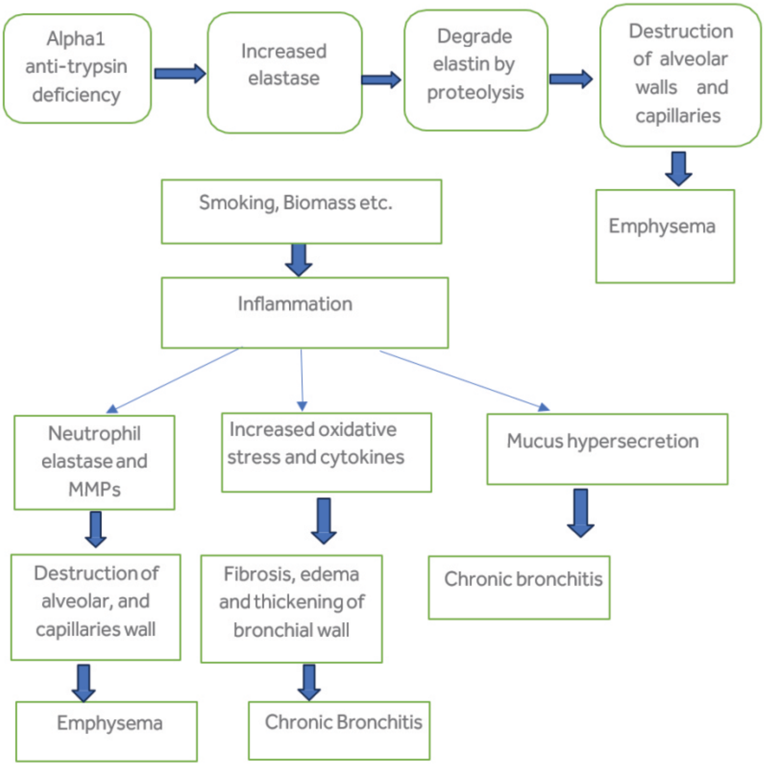
Bronchiolitis, an important pathological change in COPD, can result in inflammation and scarring of the small airways. It is difficult to be assessed by respiratory function tests.

Pulmonary Emphysema

Pulmonary emphysema is defined as:

"Abnormal permanent enlargement of airspaces distal to the terminal bronchioles accompanied by destruction of their walls."

Figure 4.1: shows the pathogenesis of COPD. **MMPs:** Matrix metalloproteinases



Secondary lobule is the part of lung that contains several terminal bronchioles surrounded by connective tissue septa.

Acinus is that part of the lung parenchyma supplied by a single terminal bronchiole. There are three main types of emphysema:[4]

Centriacinar emphysema
This is the most common type seen in smokers. It is focal enlargement of airspaces around the respiratory bronchiole. It is more prominent in upper zones of upper and lower lobes.

Panacinar emphysema
It is confluent even involvement of the acinar unit. It is more severe in lower lobe and is associated with α1-proteinase inhibitor deficiency.

Paraseptal emphysema
It is peripherally distributed enlarged airspaces where the acinar unit abuts a fixed structure, like pleura. It is the least common type.

Figure 4.2: shows the most common types of emphysema.

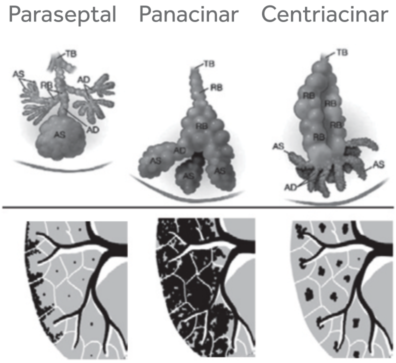


Figure 4.2: The common types of emphysema.
AD: alveolar duct, **AS:** alveolar sac, **RB:** respiratory bronchiole, **TB:** terminal bronchiole

Emerging mechanisms of COPD pathogenesis:[5,6,7,8,9,10]

New mechanism	Role in COPD
Lung microbiome dysbiosis	Promotes inflammationvia pathogen shifts
Gut–lung axis	Influences lung immunity through microbial metabolites
Cell senescence & necroptosis	Drives tissue damage and chronic inflammation
Th17 / T2 immune imbalance	Defines phenotypic subtypes and guides targeted therapy
Immunometabolism	Regulates immune activation and inflammation
Genetic/autoimmune overlap	Explains shared susceptibility with asthma

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Chapter 05:
Natural History

Lung function, as measured by forced expiratory volume in one second (FEV_1), follows a characteristic trajectory from birth through aging. This pattern reflects the interaction between normal physiological development, environmental exposures, and disease processes.

Developmental and Aging Trajectories

Lung growth accelerates in childhood and adolescence, reaching **peak FEV_1 between ages 20 and 25.** A short plateau follows, after which lung function gradually declines with age. This typical progression forms the basis of normal aging.
However, research in recent decades has refined this view, showing that individuals may follow **distinct FEV_1 trajectories,** depending on early-life events and adult exposures.

Foundational Perspective: The Fletcher-Peto Curve

The **Fletcher and Peto model (1977)** were the first to systematically describe how lung function declines over time. Their graph illustrated how **smoking accelerates the decline in FEV_1,** leading to earlier symptomatic COPD and disability. In their model:

- **Never-smokers** show a slow, age-related decline and rarely reach disability thresholds.
- **Smokers** have a **steeper FEV_1 decline,** potentially leading to early-onset COPD.
- **Smoking cessation** reduces the rate of decline, reinforcing its critical role in prevention.

The Fletcher-Peto curve provided a conceptual framework that has since been expanded by longitudinal cohort studies

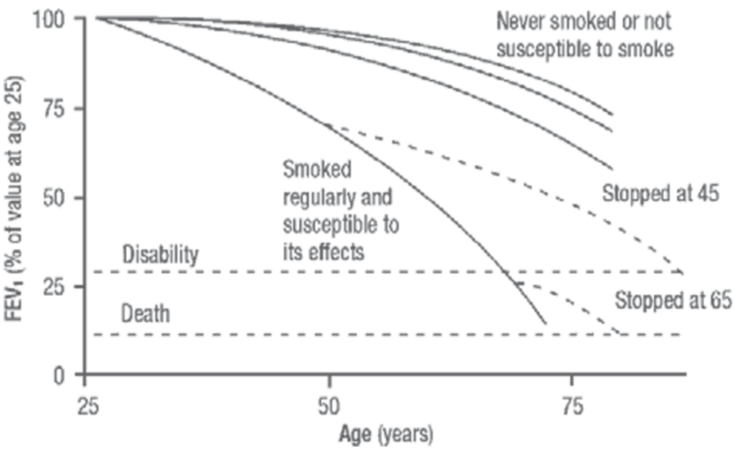


Figure 5.1 The Fletcher-Peto Curve of FEV_1 Decline in Smokers and Non- Smokers

This foundational model (1977) illustrates three trajectories of FEV_1: a slow decline in never-smokers, an accelerated decline in smokers, and a decelerated decline after smoking cessation. This concept laid the groundwork for understanding the natural history of COPD.

Source: Fletcher & Peto, 1977. Adapted under educational fair use.

Updated Perspective: GOLD 2025 FEV_1 Trajectories
Building on the Fletcher-Peto model, the GOLD 2025 Report introduces a more nuanced view of lung function trajectories. It recognizes that not all individuals who develop COPD experience accelerated decline—many never achieve normal peak lung function in early adulthood due to adverse early-life factors.

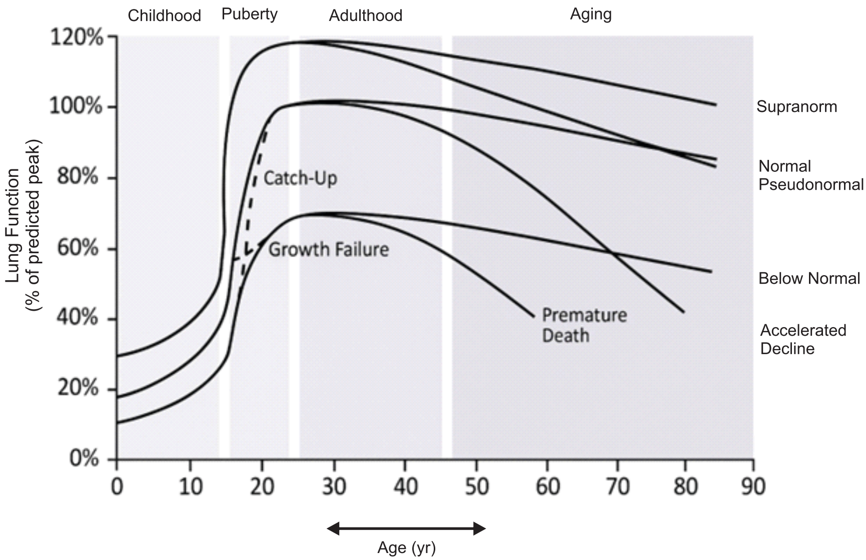


Figure 5.2 FEV_1 Trajectories Across the Lifespan

- **Trajectory 1 (Normal):** Full lung development, peak FEV_1 by ~25 years, followed by gradual physiological decline.
- **Trajectory 2 (Low Peak, Normal Decline):** Early-life disadvantage (e.g. prematurity, childhood infections) limits peak lung function, though the subsequent rate of decline is normal.
- **Trajectory 3 (Normal Peak, Accelerated Decline):** Lung growth is normal, but harmful adult exposures (e.g. smoking) cause premature, steep decline.

References

1.Fletcher C, Peto R. The natural history of chronic airflow obstruction. Br Med J. 1977;1(6077):1645–1648.

2.Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global Strategy for the Prevention, Diagnosis and Management of COPD – 2025 Report. Available from: https://goldcopd.org

Chapter 06:
Symptoms & Phenotypes

Symptoms
The hallmark of COPD is chronic progressive dyspnea.1 It is the major cause of disability and anxiety associated with the disease. The breathlessness increases with exertion, and it is there all the time. The perception of dyspnea varies among individuals with the same level of activity. It can be assessed by using modified Medical Research Council (mMRC) Dyspnea Scale2 (Table 4.1).

Table 4.1: Modified Medical Research Council (mMRC) Dyspnea Scale
Grade Degree of breathlessness related to activity

0	Not troubled by breathlessness except on strenuous exercise
1	Short of breath when hurrying or walking up a slight hill (on climbing stairs)
2	Walks slower than contemporaries on the level because of breathlessness, or has to stop for breath when walking at own pace
3	Stops for breath after walking about 100m or after a few minutes on the level
4	Too breathless to leave the house, or breathless when dressing or undressing

Dyspnea is often affected by mood, temperature, exposure to dust or fumes and position, etc.

Cough and sputum production may be present in up to 50% of cigarette smokers. At times the cough of COPD may be non-productive. Cough is often worse in the morning. Out of exacerbation the sputum is usually small in quantity and often white or grey. Excessive sputum production raises the possibility of underlying bronchiectasis. The expectoration of persistent purulent sputum may be related to bacterial colonization of the airways.
In severe disease, the generation of high intra-thoracic pressures may produce syncope during bout of cough –the cough syncope and cough fractures of the ribs. Cough is also made worse by gastro-esophageal reflex.

Wheezing and chest tightness are common in COPD but are not universal. Their presence does not confirm the diagnosis, likewise their absence does not refute the diagnosis of COPD.

Other symptoms common in COPD is atypical **chest pain, anorexia, fatigue, psychiatric morbidity** especially depression, and **poor sleep quality. Weight loss** is a feature of severe COPD. It is a bad prognostic sign, and survival is negatively correlated with body mass index3.

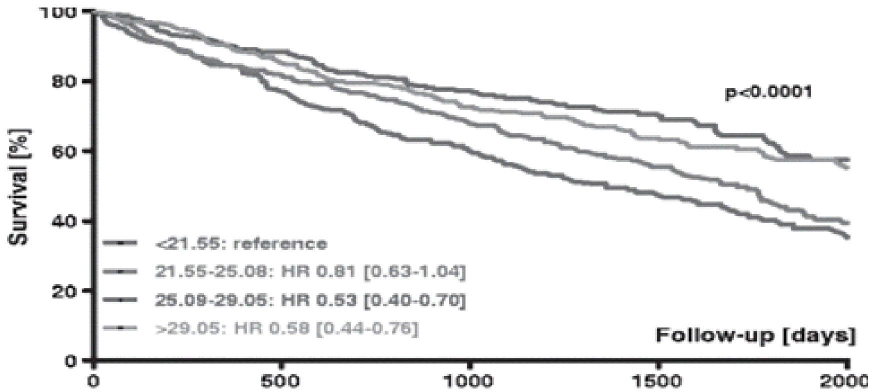


Figure 6.1: Survival is negatively correlated with body mass index. Data from Lainscak et al. Journal of cachexia, sarcopenia and muscle, 2011.HR hazard ratio; BMI: Body mass index

Signs

Physical signs are not specific to the disease. They include:

- Uniformly diminished breath sounds
- Prolonged expiratory phase of breathing
- Purse-lipped breathing

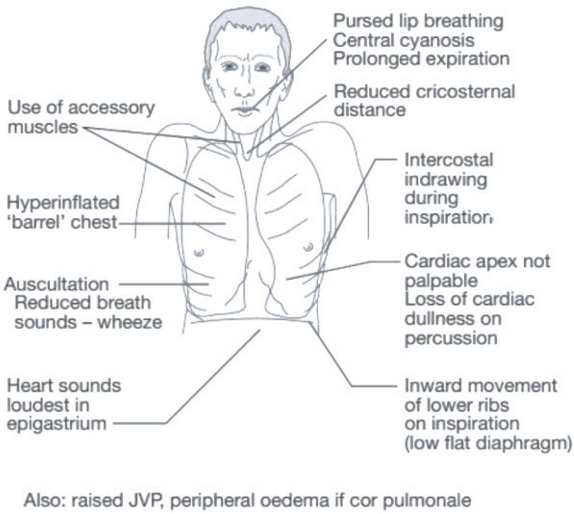


Figure 6.2: Physical signs of COPD JVP: Jugular venous pulse

- Use of accessory muscles of breathing Barrel-shaped chest
- Horizontal ribs with prominent sterna angle and wide sub- costal angle Reduced distance between supra-sternal notch and the cricoid cartilage Inspiratory tracheal tug
- Hoover's sign – horizontal position of the diaphragm pulls in the lower ribs during inspiration
- Decreased hepatic and cardiac dullness on percussion

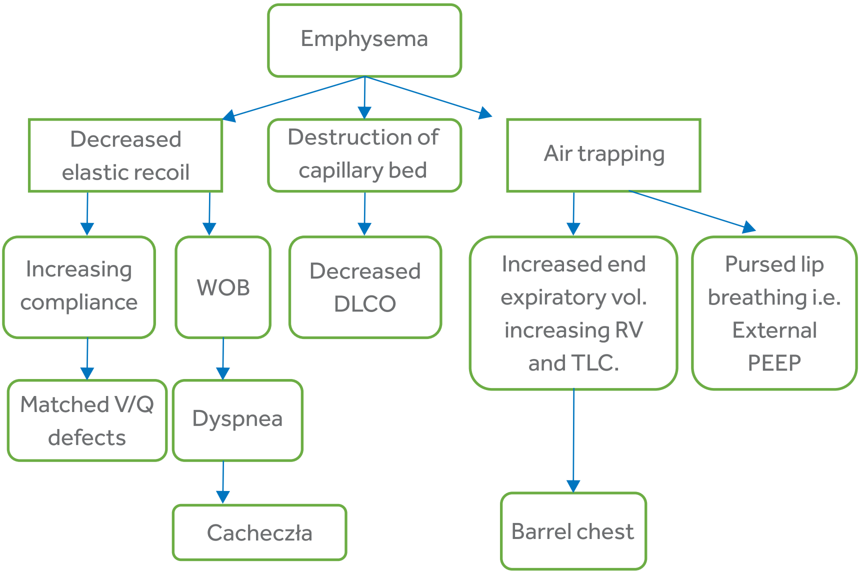
- Signs of pulmonary hypertension (RV heave, loud P2, gallop rhythm, pansystolic murmur, pitting pedal edema)
- Tender pulsatile liver
- Prominent v wave of jugular venous pulse

Systemic effects of COPD

COPD is associated with a number of systemic effects and co-morbidities.[3]

- Skeletal muscle dysfunction
- Weight loss
- Ischemic heart disease, hypertension, heart failure
- Arrhythmia, stroke
- DVT, Pulmonary Embolism, and Aortic aneurysm
- Osteoporosis
- Anxiety, Depression
- Diabetes Mellites
- Carcinoma lung
- Bronchiectasis, Skin Wrinkling
- Peptic Ulceration, GERD. Anemia.

Traditionally COPD is divided into 2 phenotypes: Emphysema and Chronic bronchitis



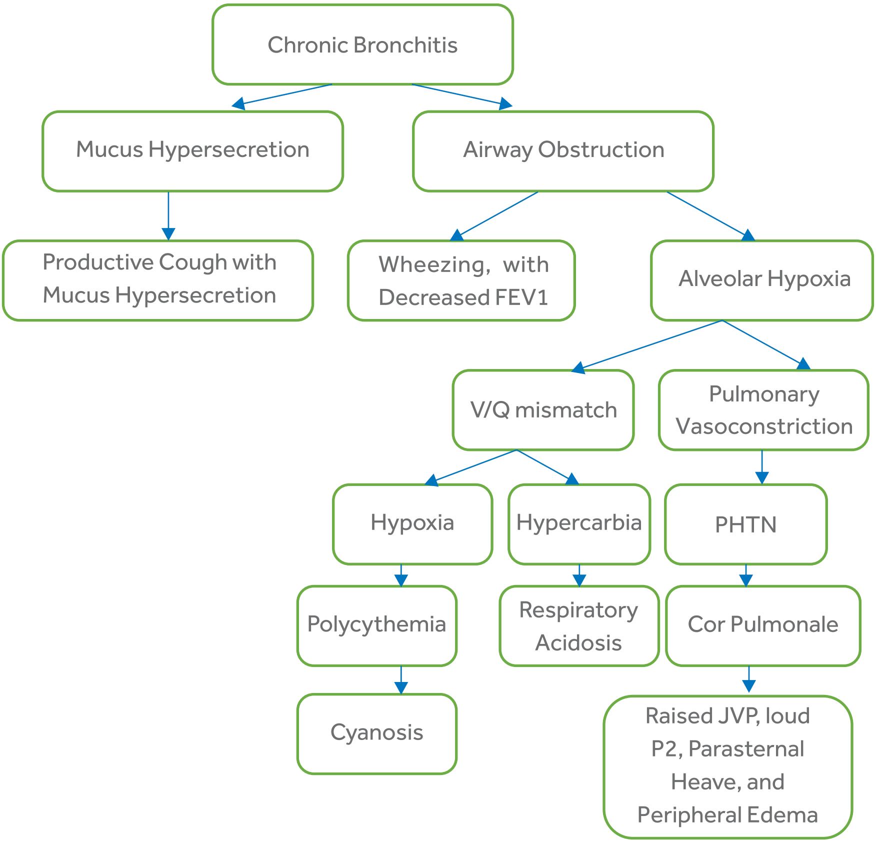


Figure 6.2: Two phenotypes of COPD: Emphysema and chronic bronchitis

DLCO: Diffusion capacity of lung for carbon monoxide; FEV1: Forced expiratory volume in one second; FVC: Forced vital capacity; JVP: Jugular venous pulse; P2: Pulmonic second heart sound; PEEP: Positive end expiratory pressure; PHT: Pulmonary hypertension RV: Residual volume; TLC: Total lung capacity; VC: Vital capacity; WOB: Work of breathing

References

1.Kessler R, Partridge MR, Miravitlles M, et al. Symptom variability in patients with severe COPD: a pan-European cross-sectional study. ERJ. 2011, 37 (2) 264-272

2.Mahler, Donald A. et al. Evaluation of Clinical Methods for Rating Dyspnea. Chest. 1988;93(3):580-86.

3.MacNee W. Pathology, pathogenesis, and pathophysiology. BMJ. 2006;332(7551):1202–1204.

Chapter 07:
Pathway To The Diagnosis Of COPD

COPD should be considered in any patient who has symptoms of the disease as discussed in chapter 6 and/or history of exposure to risk factors. Spirometry is required to diagnose the disease. (Figure 7.1)

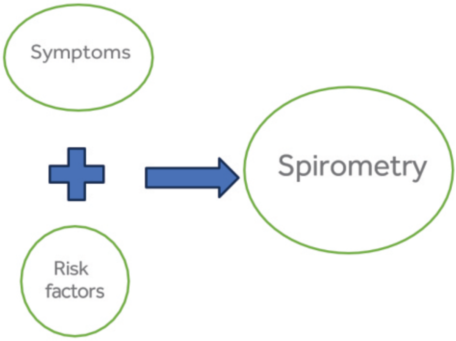


Figure 7.1: Pathway to the diagnosis of COPD

In patients with symptoms and exposure to risk factors, post-bronchodilator FEV1/FVC < 0.70 confirms the diagnosis of COPD.

Post-bronchodilator forced expiratory volume in first second (FEV1)/forced vital capacity (FVC) below the LLN (lower fifth percentile of values from a reference population) should be preferably used as the criterion for diagnosis of airflow obstruction. However, in the absence of reference equations for LLN, FEV1/FVC < 0.7 may be used as the cutoff for defining airflow obstruction.
Using fixed ratio of <0.7 is not inferior to LLN regarding prognosis.

Spirometry in COPD

In evaluation of COPD patient spirometry has following roles:

- Diagnosis
- Assessment of severity (prognosis)
- Follow-up assessment
- Therapeutic decisions
- Identification of rapid decline

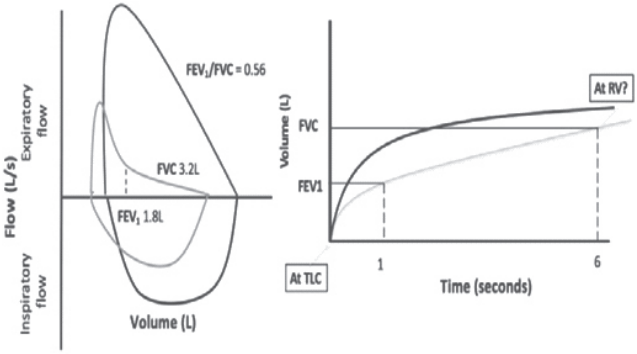


Figure 7.2: Typical spirometry trace of COPD patient

FEV1: Forced expiratory volume in 1 second; **FVC:** Forced vital capacity; **TLC:** Total lung capacity; **RV:** Residual volume

References

1.Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. 2025.

2.Guidelines for diagnosis and management of chronic obstructive pulmonary disease: Joint ICS/NCCP (I) recommendations. Lung India. 2013;30(3).

Chapter 08:
Spirometric Classification Of Severity Of COPD

Spirometry should be performed after the administration of short acting inhaled bronchodilator in order to minimize variability. The classification of airflow limitation severity in COPD based on spirometry is shown in table 8.1.

Table 8.1: Spirometric classification of severity of COPD, based on post- bronchodilator FEV1.

In patients with FEV1/FVC < 0.70

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

GOLD: Global Initiative for Chronic Obstructive Lung Disease

GOLD continues to recommend using the FEV1 as a percentage of the predicted value to stage the severity of airflow obstruction. The ERS and ATS recommend using z- scores rather than percent predicted values to stage severity and have proposed a three-level(four-tier) severity system that considered z-scores >-1.65 as normal, between -1.65 and -2.5 as mild, between -2.51 and -4 as moderate and < -4.1 as severe. Using these thresholds inevitably leads to differences in the classification of some patients compared to using the percentage predicted but whether this has significant implications for management or prognosis remains unclear.

Reference

1.Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. 2025.

Chapter 09:
Assessment Of COPD Severity

COPD assessment is necessary to determine the level of airflow obstruction and its impact on health status of the patient and the risk of future exacerbations. This eventually guides therapy.
The following aspects of the disease must be assessed separately.

The presence and severity of spirometry abnormality

- Current severity of symptoms
- History of exacerbations and future risk
- Blood Eosinophil count
- Presence of co-morbidities

The presence and severity of Spiro metric abnormality (It has been discussed in chapter 8.)

Current severity of symptoms
Breathlessness being the cardinal symptom of the disease, one tool to assess severity of symptoms is mMRC dyspnea scale, discussed in chapter 6. But as a matter of fact, COPD impacts patients beyond just dyspnea. So combined assessment test (CATTM) can be used to measure health status in COPD.[1] It has been translated and validated in Urdu. It is an 8-item Likert style questionnaire. Score ranges from 0 to 40, where 0 is asymptomatic patient and 40 is patient with severe symptoms. See Figure 9.1.

آپ کی پھیپھڑوں میں رکاوٹ کی پرانی بیماری (COPD) کیسی ہے؟ COPD کی تشخیصی جانچ (CAT)™ کروائیں

یہ سوالنامہ آپ کی تندرستی اور روز مرہ کی زندگی پر COPD (پھیپھڑوں میں رکاوٹ کی دیرینہ بیماری) کے اثر کی پیمائش کرنے میں آپ اور آپ کی نگہداشت صحت کے پیشہ ور فرد کی مدد کرے گا۔ آپ کے جوابات، اور جانچ کے اسکور کا استعمال آپ اور آپ کی نگہداشت صحت کے پیشہ ور فرد کے ذریعہ آپ کی COPD کے انتظام کو بہتر بنانے اور معالجے سے زیادہ سے زیادہ فائدہ حاصل کرنے میں مدد کے لیے کیا جاسکتا ہے۔

درج ذیل ہر ایک آئٹم کے لیے، براہ کرم اس باکس میں(X) کا نشان لگائیں جو آپ کی موجودہ حالت کی بہترین وضاحت کرتا ہو۔ ہر سوال کے لیے صرف ایک ہی جواب کا انتخاب کرنے کو یقینی بنائیں۔

مثال: میں بہت خوش ہوں	5 ☐ 4 ☐ ☐ 2 ☐ 1 X 0 ☐	میں بہت غمگین ہوں

اسکور			
☐	میں ہر وقت کھانستا رہتا/کھانستی رہتی ہوں	5 ☐ 4 ☐ 3 ☐ 2 ☐ 1 ☐ 0 ☐	مجھے کبھی کھانسی نہیں ہوتی
☐	میرا سینہ بلغم سے پوری طرح بھرا ہوا ہے	5 ☐ 4 ☐ 3 ☐ 2 ☐ 1 ☐ 0 ☐	میرے سینے میں بلغم بالکل نہیں ہے
☐	مجھے سینے میں شدید جکڑن محسوس ہوتی ہے	5 ☐ 4 ☐ ☐ 2 ☐ 1 ☐ 0 ☐	مجھے سینے میں بالکل بھی جکڑن محسوس نہیں ہوتی
☐	جب میں پہاڑی یا ایک زینے کی سیڑھیوں پر چڑھتا/چڑھتی ہوں تو میری سانس بہت زیادہ پھول جاتی ہے	5 ☐ 4 ☐ 3 ☐ 2 ☐ 1 ☐ 0 ☐	جب میں پہاڑی یا ایک زینے کی سیڑھیوں پر چڑھتا/چڑھتی ہوں تو میری سانس نہیں پھولتی ہے
☐	میں اپنی گھر پر سرگرمیاں انجام دینے میں کافی محدود ہوں	5 ☐ 4 ☐ 3 ☐ 2 ☐ 1 ☐ 0 ☐	مجھے اپنے گھر پر سرگرمیاں انجام دینے میں کوئی مجبوری نہیں ہے
☐	اپنے پھیپھڑے کی کیفیت کی وجہ سے میں اپنے گھر سے باہر جاتے ہوئے بالکل پراعتماد نہیں رہتا/رہتی ہوں	5 ☐ 4 ☐ 3 ☐ 2 ☐ 1 ☐ 0 ☐	اپنے پھیپھڑے کی کیفیت کے باوجود میں اپنے گھرسے باہر جاتے ہوئے پراعتماد رہتا/رہتی ہوں
☐	اپنے پھیپھڑے کی کیفیت کی وجہ سے میں گہری نیند نہیں سوتا/سوتی ہوں	5 ☐ 4 ☐ 3 ☐ 2 ☐ 1 ☐ 0 ☐	میں گہری نیند سوتا/سوتی ہوں
☐	میرے اندر بالکل توانائی نہیں ہے	5 ☐ 4 ☐ 3 ☐ 2 ☐ 1 ☐ 0 ☐	میرے اندر بہت زیادہ توانائی ہے

☐☐ مجموعی اسک

Figure 9.1: COPD assessment test. CAT™ score ≥ 10 indicates high level of symptoms.

Interpretation of CAT™
See table 9.1.

Table 9.1: Interpretation of CAT™

Score	< 10	10 – 20	20 – 30	< 30
Interpretation	Low	Medium	High	Very high

History of exacerbations and future risk

COPD exacerbation is defined as:
"An acute worsening of respiratory symptoms that require additional therapy."
Fletcher first described 'chest episodes' in 1976, since then the interest to develop criteria for these episodes increased steadily. In 1987 Anthonisen et al.[2] gave the classic definition describing three levels of exacerbation based on patient's symptomatology. This is the criteria recommended to be used in describing the exacerbations. It is used to decide the need of antibiotic therapy. See table 9.2.

Table 9.2: Anthonisen criteria for COPD exacerbation

Type I	Type II	Type III
Three of: Increased dyspnea, sputum volume, and sputum purulence	Two of: Increased dyspnea, sputum volume, or sputum purulence	One of: Increased dyspnea, sputum volume, or sputum purulence Plus, one of the following: Upper respiratory infection within past 5 days, fever without other cause, increased wheezing, increased cough, increase in heart or respiratory rate by 20% compared with baseline.

* Type I requires escalation of treatment without addition of antibiotic. Type II and III will benefit from antibiotic therapy

The predictors of future exacerbations risk are:

- Two or more exacerbations in the past year
- History of hospitalization due to COPD in the past year
- Severe COPD equivalent to GOLD 3 or 4
- Increased blood eosinophil count
- Use of SABA alone
- Noncompliance to treatment

Based on the management plan, COPD exacerbations are classified as shown in table 9.3:
Table 9.3: Severity of COPD exacerbation based on management plan

Mild	Need short acting bronchodilators only. Can be managed outside health care facility.
Moderate	Need short acting bronchodilator plus antibiotics and/or oral corticosteroids. Requires assistance of health care facility.
Severe	Need intravenous antibiotics and corticosteroids. Requires hospitalization.

Blood Eosinophil count:
A number of studies have shown that blood eosinophil counts predict the magnitude of the effect of ICS in preventing future exacerbations and blood eosinophil counts are recommended by GOLD to guide the use of ICS as part of pharmacological management.
Co-morbidities should be taken into account when assessing severity of the exacerbation.
Peak expiratory flow measurement is not useful in assessing the need for hospitalization in COPD exacerbation.

Assessment of co-morbidities
COPD is a systemic disease as discussed elsewhere. These conditions can increase the risk of hospitalizations and mortality in COPD. So co-morbid illnesses should be looked for and treated promptly.

The ABCD assessment tool
In 2023 GOLD report, GOLD proposed a further evolution of the ABCD combine assessment tool that recognized the clinical relevance of exacerbation, independently of the level of symptoms of the patient. Figure 9.4 presents unchanged A and B groups, but C and D groups were merged into a single group termed "E" to highlight the clinical relevance of exacerbations.
A new version of the ABCD tool should be used which separates spirometry classification from patient's symptoms and history of exacerbations.[3] For symptom assessment CAT™ score is better.

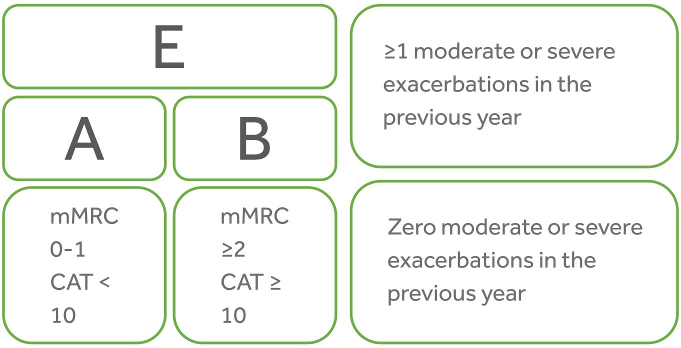


Figure 9.4: mMRC, modified Medical Research Council. CAAT, COPD Assessment Test.

References
1.Karloh M, Mayer AF, Maurici R, et al. The COPD Assessment Test: What Do We Know So Far?: A Systematic Review and Meta-Analysis About Clinical Outcomes Prediction and Classification of Patients Into GOLD Stages. Chest. 2016 Feb;149(2):413-425.
2.Anthonisen NR, Manfreda J, Warren CP, et al. Antibiotic therapy in exacerbations of chronic obstructive pulmonary disease. Ann Intern Med. 1987 Feb;106(2):196-204.
3.Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. 2025.

Chapter 10:
Additional Investigations

α-1 Antitrypsin Deficiency
Although it is common in European whites, younger people (< 45 years) and with lower lobe emphysema should be screened for α-1 antitrypsin deficiency.
In Pakistan emphysema in young population is common in drug addicts.[1]

Six- minute Walk Test
This test provides prognostic information and for monitoring of exercise capacity in COPD.[2]

Chest X-ray
It is useful in excluding other diagnosis.
Radiological changes include (Figure 10.1):[3]

- Increase lung volumes
- Flattened diaphragm
- Tubular heart
- Hyperlucency of lung fields Bullae

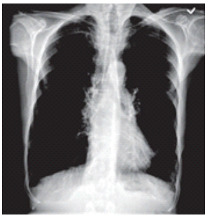


Figure 10.1: COPD X-ray

CT Scan Chest[3,4]
It is not routinely needed, but may be required if in doubt or to exclude bronchiectasis. It can also be requested for screening of lung cancer, before surgical treatment of COPD. A CT scan is also essential for patients being evaluated for lung transplantation.
Expiratory CT scan chest may show air trapping; it is not currently considered standard of care in the diagnosis and management of mild to moderate COPD.

Arterial Blood Gas Analysis And Pulse Oximetry
Pulse oximetry should be used to screen for hypoxemia in stable disease with FEV1 < 50% and in the presence of clinical suspicion of hypoxemia.

ABGs can be requested in patients with SpO_2 <90%, having signs of hypercapnia, those with severe COPD or signs of cor pulmonale. These patients may benefit from long term oxygen therapy or noninvasive ventilation.

Complete blood count
COPD may be related to anemia of chronic disease or polycythemia, requiring proper management.

Electrocardiogram
It can help in detecting underlying coronary artery disease, arrythmias or pulmonary hypertension.

Lung Volumes[5]
COPD patients exhibit gas trapping (a rise in residual volume) from the early stages of the disease, and airflow obstruction worsens, static hyperinflation (and increase in total lung capacity) occurs, particularly during exercise (dynamic hyperinflation). These changes can be

documented by body plethysmography, or less accurately by helium dilution lung volume measurement. These measurements help characterize the severity of COPD but are not essential to patient management.

Carbon Monoxide Diffusing Capacity Of Lungs[5] (DLco)

The single breath DLco measurement evaluates the gas transfer properties of the respiratory system. DLco should be measured in any person with symptom (dyspnea) disproportionate to the degree of airflow obstruction since reduced DLco values < 60% predicted are associated with increased symptoms, decreased exercise capacity, worse health status, and increased risk of death independently on the severity of airflow obstruction and other clinical variables. Additionally, in COPD patients, low DLco values help preclude surgical lung resection in patient with lung cancer while in smokers without airflow obstruction, values < 80% predicted (as a marker of emphysema) signal increased risk for developing COPD over time.

Composite Score

The BODE (Body mass index, Obstruction, Dyspnea, and Exercise) method gives a composite score that is a better predictor of subsequent survival than any single component.

Biomarkers

At present blood Eosinophil counts (≥ 300 cells/μL) provide guidance to identify COPD patients at higher risk of exacerbations and more likely benefit from preventive treatment with inhaled corticosteroids.

References

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3.Washko GR. Diagnostic imaging in COPD. Semin Respir Crit Care Med. 2010;31(3):276–285.

4.Labaki WW, Martinez CH, Martinez FJ, et al. The Role of Chest Computed Tomography in the Evaluation and Management of the Patient with Chronic Obstructive Pulmonary Disease. AJRCCM. 2017(196);11

5.Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. 2025.

Chapter 11:
Differential Diagnosis

The main differential diagnosis includes:

- Asthma and Bronchiectasis
- Tuberculosis and post-tuberculosis sequelae (Post TB obstructive airway disease is discussed elsewhere)
- Heart failure and Interstitial lung diseases

A careful history, clinical examination, and investigations can help rule out these close mimics of COPD.

Key Differentiating Features:

Condition	Age of Onset	History	Distinguishing findings
COPD	>35 y	Smoking history progressive dyspnea, chronic productive cough	Fixed airflow obstruction (post-bronchodilator), minimal daily variability
Asthma	<35 y	Atopy, episodic symptoms, variability, non-smoker	Significant reversibility (>400mL FEV1), peak-flow variability
Bronchiectasis	Variable	Chronic sputum, recurrent infections	HRCT shows bronchial dilation, crackles, sputum purulence
Heart Failure	Older adults	Orthopnea, ankle swelling, fatigue	Raised JVP, ECG changes, elevated BNP/NT proBNP, echocardiography
Interstitial Lung Disease	Middle-older	Dry cough, progressive dyspnea, risk exposures	Inspiratory crackles, restricted spirometry, reticulonodular CT pattern

Source: Adapted from NICE guideline NG115 (2023). [1]

References

1.National Institute for Health and Care Excellence (NICE). Chronic obstructive pulmonary disease in over 16s: diagnosis and management. NICE guideline [NG115]. London: NICE; 2018 [updated 2023 Jun 29; cited 2025 Jul 31]. Available from: https://www.nice.org.uk/guidance/ng115

Chapter 12:
Management Of Stable COPD

Bronchodilators are first line therapy for COPD. Pharmacologic management can reduce symptoms, improve exercise capacity, reduce the risk of exacerbations, improve overall health status and reduce mortality.

The aim of COPD management is to reduce symptoms and future risks (figure 12.1)

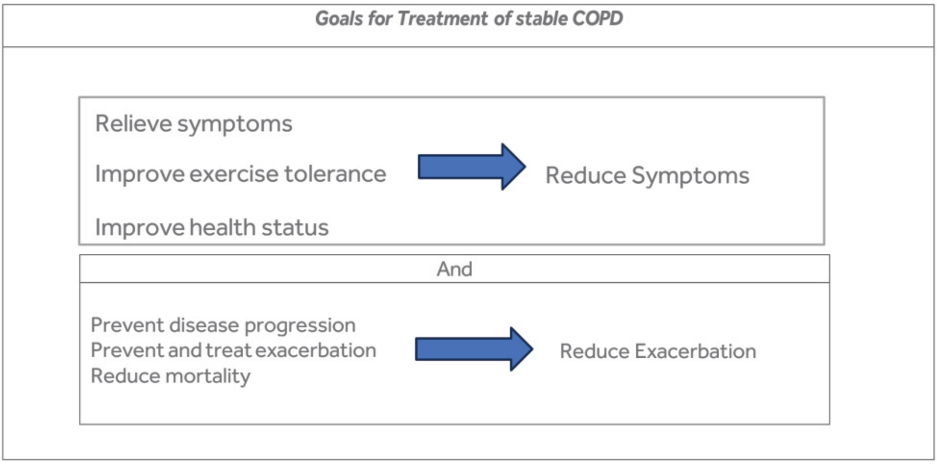
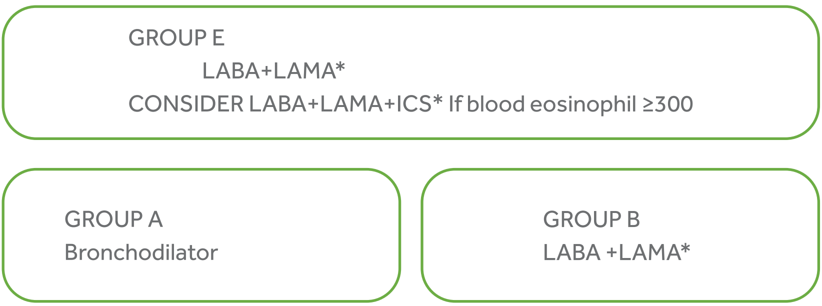


Figure 12.1: Goals for treatment of stable COPD.

Initial Pharmacological Treatment
Initial pharmacotherapy should be based on the patient GOLD group (figure 12.2)



*Single inhaler therapy maybe more convenient and more effective than multiple inhalers
Figure 12.2:

Following implementation of initial therapy, patient should be reassessed for attainment of treatment goals and identification of any barriers for successful treatment.

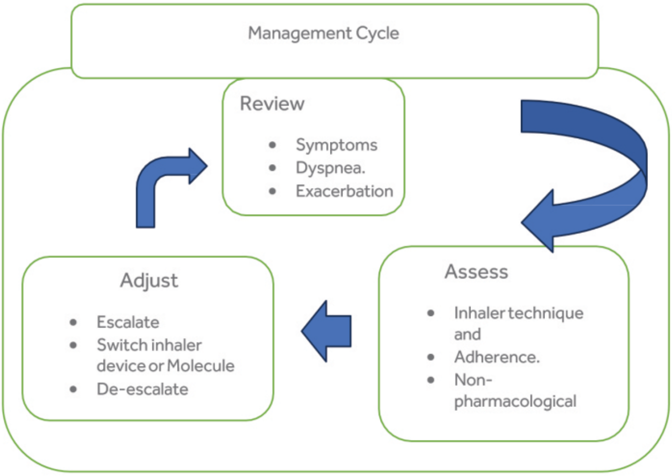


Figure 12.3: Management cycle.

Follow up pharmacological therapy is based on two key treatable traits: persistence of dyspnea, and occurrence of exacerbations (Figure 12.4).

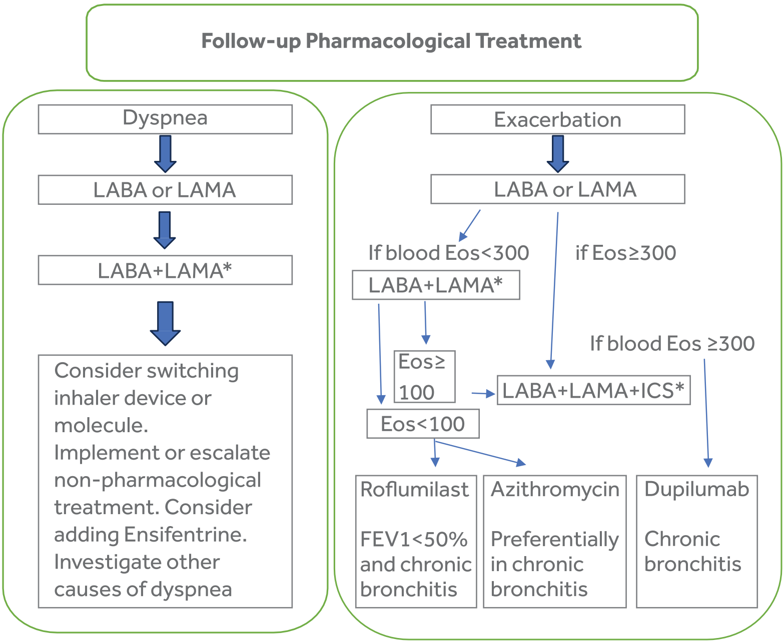


Figure 12.4: Follow-up treatment protocol

*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 de-escalation is more likely to be associated with development of exacerbation.

Key Points:

- Choice of inhaler device should be tailored individually (depending on access, cost, prescriber, patient's preference and ability.
- Teach inhaler technique and recheck on each visit.
- Check inhaler technique and compliance before changing medicines. Inhaled bronchodilators are preferred over oral.
- Theophylline is not recommended unless other long-acting treatment bronchodilators are not available or are unaffordable.
- Long term monotherapy with ICS is not recommended. Long term therapy with OCS is not recommended.

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Chapter 13:
Non-Pharmacologic Management Of COPD

Smoking Prevention And Cessation

Smoking cessation is the single most effective way to reduce the risk of developing COPD and stop its progression. Even a brief, three-minute period of counseling to urge a smoker to quit can be effective. This should be done for every smoker at each visit. Figure 13.1 details the five As for counseling regarding smoking cessation.[1]

Ask	About tobacco use
Advise	Tobacco users to quit
Assess	Readiness to make a quit attempt
Assist	With the quit attempt
Arrange	Follow up care

Figure 13.1: The five As: brief tobacco intervention.

The prevalence of tobacco smoking in Pakistan as per World Health Organization's report on the global tobacco epidemic, 2017 is shown below (Table 13.1):

Table 13.1: Tobacco use data from the latest survey as at 31 December 2016:

	Youth tobacco use		**Adult tobacco smoking**		**Adult cigarette smoking**	
Prevalence (%)	Current tobacco use	Current cigarette smoking	Current	Daily	Current	Daily
Male	13.3	4.8	22.2	20.6	19.4	17.9
Female	6.6	0.9	2.1	2.0	1.0	1.0
Total	10.7	3.3	12.4	11.5	10.5	9.6

Youth: Global Youth Tobacco Survey, 2013: National, ages 13-15 Adult: Global Adult Tobacco Survey (GATS), 2014: National, ages 115+

Comprehensive tobacco control policies and programs with clear, consistent, and repeated nonsmoking messages should be delivered through every feasible channel. Legislation should be implemented to establish smoke-free schools, public places and transport.

There is a misperception of using chewable tobacco (like snuff: naswar) or e- cigarettes to help in quit attempts. These are not recommended and should be discouraged.

If effective time and resources are dedicated to smoking cessation, long-term quit rates of 14% to 27% have been reported.[592] The combination of counseling and pharmacotherapy is the most effective smoking cessation treatment for the people with COPD.

Indicators Of High Nicotine Dependence:
- Smoking within 30 minutes of waking up
- Smoking at night
- Consuming ≥ 20 cigarettes per day
- A score of 7-10 on the fagerstrom scale or 5-6 on the Heaviness of Smoking Index.[595]

Control Of Occupational And Indoor Pollution

Indoor pollution due to burning of wood and coal to keep houses warm in winter and use of biomass fuel in stoves should be minimized and measures should be taken to reduce exposure as by cooking in open air rather than a closed kitchen, having separate cooking area, making chimneys etc.

Exposure to irritant particles and gases should also be avoided at work place.

Climate Change and COPD

Population studies have consistently found that people with COPD are at increased risk of death as a consequence of exposure to both heat and cold with cold leading to a greater risk than heat.[621]

The mechanism by which heat exposure and cold adversely impact COPD are not well understood. However, on the basis of the evidence showing a relationship between high and low temperatures and morbidity and mortality, patient with COPD living in temperate and colder climates should, in line with WHO recommendations, ensure they keep bedroom temperatures above 18℃ during cold weather. During heatwaves and periods of high temperature patients should ensure they keep adequately hydrated, keep out of the heat and try to keep living spaces < 32℃ and sleeping spaces < 24℃ as recommended by WHO[659]

Pulmonary Rehabilitation

The main goals of pulmonary rehabilitation are to increase overall resources of the patient, to reduce handicap caused by illness or disability and to allow integration of patient in society. This can be done by:

1. **Exercise Training**[2]

In patients with mild to moderate COPD, suitable exercises are walking, cycling and swimming. Daily exercise should be done for about 30 minutes; it may be divided into 2 – 3 phases or till the patient gets out of breath. In severe COPD, it should be done to improve strength and endurance of muscles. This should involve respiratory, abdominal, back, head, neck and limbs to improve quality of life.

2. **Nutritional Counseling**[3]

Low BMI is an independent risk factor for mortality in COPD patients. Increased calorie intake should be accompanied by regimens with anabolic action. On the other side obese individuals have greater levels of breathlessness and impairment of activity. Well-balanced diet is recommended.

3. **Education**

Educate regarding disease, its progressive nature, smoking cessation, drug treatment and how to manage exacerbations.

Self-Management And Integrative Care

The aim of self-management interventions, is to motivate, engage, and coach patients to positively adopt their health behaviors and develop skills to better manage their COPD on day-to-bases[762]. COPD is a complex disease that requires the input of multiple health care providers who need to work together closely.

Vaccination[4,7]

People with COPD are at higher risk of respiratory infections, which can lead to exacerbations, hospitalizations, and increased mortality. Vaccination plays a key role in preventing these complications and is recommended in line with local guidelines.

1. **Influenza Vaccine**

- Annual influenza vaccination is recommended for all COPD patients (Evidence B).
- Vaccination reduces serious outcomes such as lower respiratory tract infections, stroke, and death.
- Vaccines containing either killed or live inactivated viruses are effective; high-dose inactivated (HD-IIV3) and adjuvanted inactivated (aIIV3) forms are preferred in older adults.
- Long-term influenza vaccination is associated with reduced ischemic heart disease risk in COPD patients.

2. **Pneumococcal Vaccines**

Recommended Vaccines:

- PCV20 or PCV15 (pneumococcal conjugate vaccines)
- PPSV23 (pneumococcal polysaccharide vaccine)

Indications:

- Adults ≥ 65 years
- Adults aged 19–64 with chronic lung disease (e.g., COPD), smoking history, or other comorbidities

Newer Vaccines:

- PCV21 (recently FDA-approved) covers 11 additional serotypes not in PCV20 and protects against 84% of invasive strains.

Recommendations:

- One dose of PCV21 or PCV20, or PCV15 followed by PPSV23, based on prior vaccination status and age (Evidence B).

Effectiveness:

- Pneumococcal vaccination reduces the risk of community-acquired pneumonia and COPD exacerbations.
- PCV13 showed persistent effectiveness over 5 years in reducing pneumonia and exacerbations compared to PPSV23.

Other Recommended Vaccine (Table 13.2)

Vaccines	Indications	Evidence level
COVID-19 (SARS-CoV-2)	As per WHO/CDC guidance	B
RSV (Respiratory Syncytial Virus)	Adults = 60 years and those with chronic heart/lung disease	A
Tdap (Tetanus, diphtheria, pertussis)	Adults with COPD not vaccinated in adolescence	B
Zoster (Shingles)	COPD patients > 50 years	B

Oxygen Therapy[7]

The long-term administration of oxygen (>15 hours per day) to patients with chronic respiratory failure has been shown to increase survival in patients with severe resting hypoxemia.768 Ekstrom and colleagues reported that among patients with severe hypoxemia agreeing to participate in a study long-term oxygen therapy used for 24 hours per day did not result in a lower risk of hospitalization or death within one year compared to therapy for 15 hours per day, but the generalizability of this finding is unclear.

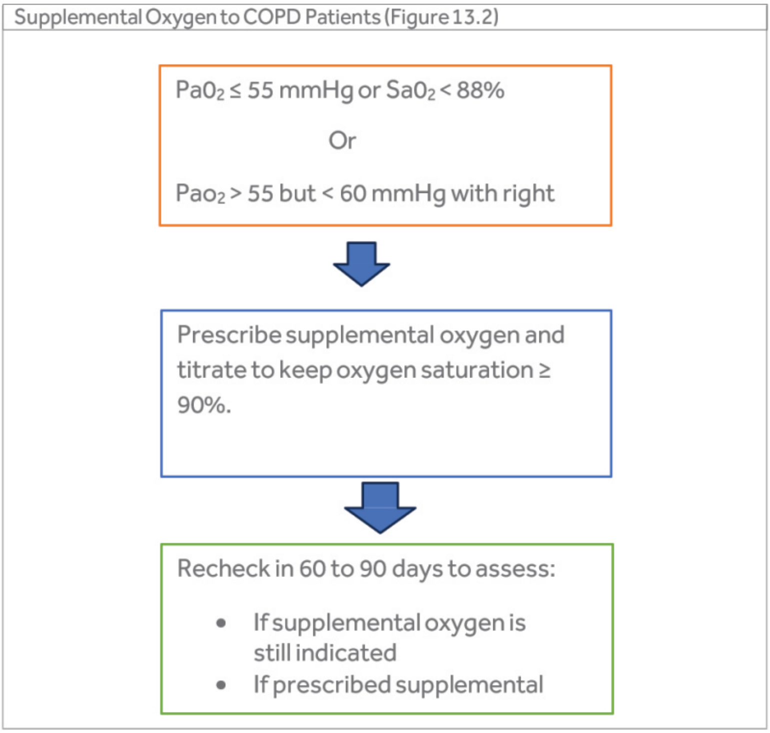


Figure 13.2: Algorithm for prescription of supplemental oxygen Non-Pharmacological Treatment of COPD*: Figure 13.3

Patient Group	Essential	Recommended	Depending on Local Guideline
A	Smoking cessation	Physical activity	Influenza vaccination COVID-19 vaccinations Pneumococcal vaccination Pertussis vaccination Shingles vaccination RSV vaccination
B + E	Smoking cessation Pulmonary Rehabilitation	Physical activity	Influenza vaccination COVID-19 vaccinations Pneumococcal vaccination Pertussis vaccination Shingles vaccination RSV vaccination

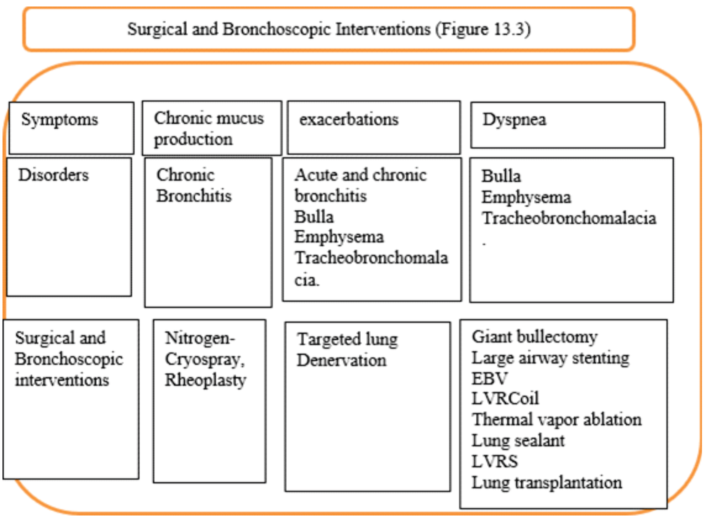
Ventilatory Support

Noninvasive ventilation by using BiPAP can be helpful in some stable patients with severe COPD to decrease work of breathing or to reduce daytime hypercapnia.

Intervention Bronchoscopy And Surgery[7]

Advanced COPD provide potential targets for interventional and surgical treatment to alleviate dyspnea, reduce cough, and mucus production and improve quality.

The algorithm given below (Figure 13.3) is adapted from GOLD guidelines 2025. But most of these therapies are currently not being done in Pakistan



LVRS: Lung volume reduction surgery

EBV: Endobronchial valve

LVRC: Lung volume reduction coil

VA: Vapor ablation

Figure 13.3: Algorithm for surgical management of advanced COPD. Adapted from GOLD COPD guidelines 2025

Bullectomy
Reductions in dyspnea, and improvements in lung, respiratory muscle, and cardiac performance, as well as exercise tolerance have been reported. Blood or thrombin instillation may be effective in those unfit for resection.

Lung Volume Reduction Surgery
With LVRS, most of the emphysematous portions of the lungs are resected to reduce the hyperinflation that results in improvements of FEV1, walking distance, and quality of life. Post operative BODE is a predictor of survival following LVRS. To achieve successful outcomes, a multidisciplinary team is key to select potential patients for LVRS patients and coordinate postoperative care.

Lung Transplant
Criteria for referral to lung transplant center include:[6]

- COPD with progressive disease, despise maximal medical treatment.
- Not candidate for LVRS.
- BODE index 5-6
- $PaCO_2$ >55 mmHg
- PaO_2 < 60 mmHg
- FEV1 < 25%

1n April 2023, Pakistan's first **Lungs Transplant Unit** was inaugurated at the Pir Abdul Qadir Shah Jilani Institute of Medical Sciences in **Gambat (Khairpur, Sindh)**—also known as the Gambat Health City & Lung Care Center. This marks the first official facility for lung transplant services in Pakistan but widespread clinical facilities are not yet available.
The median survival post transplantation for COPD is 5.9 years. Two unique native lung complications have been proposed to account for the superiority of double lung transplantation in patients with COPD, native lung hyperinflation and lung cancer occurrence in native lung.

The complications most seen in COPD patients after lung transplantations are:

- Acute rejection
- Bronchiolitis Obliterans
- Opportunistic Infections
- Lymphoproliferative diseases

Bronchoscopic Interventions[7]
Due to the morbidity and mortality associated with LVRS, less invasive Bronchoscopic approaches to lung reduction have been examined.

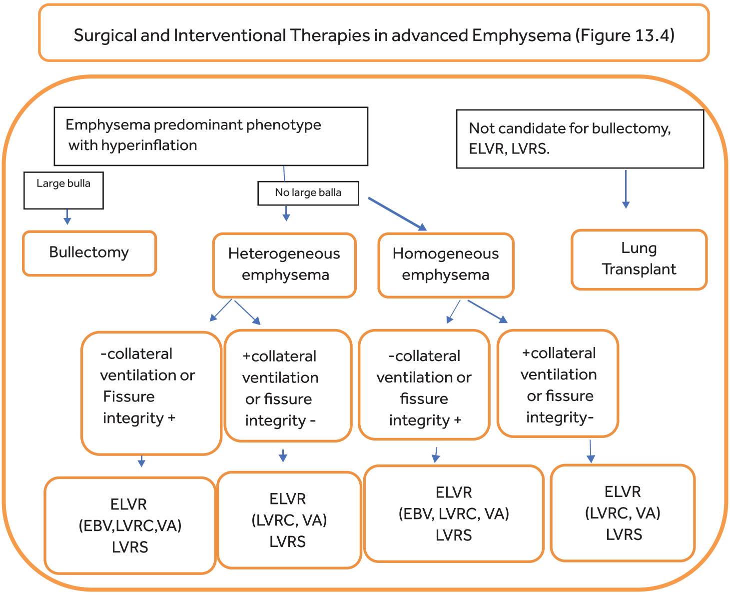


Figure 13.4
Collateral ventilation measure by chartis. Fissure integrity + > 90% by HRCT; Fissure integrity - < 90% by HRCT. ELVR; Endoscopic lung volume reduction, EBV; endobronchial valve, VA; Vapor ablation. LVRC; Lung volume reduction coil. LVRS; lung volume reduction surgery

Palliative and End-of-Life Care in COPD[7]
End of life/ palliative care is an important consideration in patients with advanced COPD. Main goals are to reduce suffering and provide best quality of life for patients and their families. The discussion should focus disease course and prognosis, advanced healthcare directives, and strategies to relieve symptoms. Hospice care, a model for delivery of end-of-life care for patients who are terminally ill and predicted to have less than 6 months to live.
Effective management of COPD should include palliative care strategies aimed at relieving symptoms and improving quality of life. Healthcare providers should be knowledgeable about these approaches and integrate them into clinical practice (Evidence D).
Conversations around end-of-life care are essential and should involve the patient and their family, covering topics such as resuscitation preferences, advance directives, and preferred place of death (Evidence D).
To ease breathlessness, various interventions such as opioids, neuromuscular electrical stimulation (NMES), oxygen therapy, and the use of fans to direct airflow toward the face can be beneficial (Evidence C).
In patients who are malnourished, nutritional support should be considered to enhance respiratory muscle strength and overall health (Evidence B).
Fatigue management in COPD may benefit from a combination of self- management education, pulmonary rehabilitation, nutritional support, and mind-body therapies (Evidence B).

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Chapter 14:
Management Of COPD Exacerbation

Exacerbation of COPD is defined as an event characterized by increased dyspnea and/or cough and sputum that worsens in < 14 days which may be accompanied by tachypnea and/or tachycardia and is often associated with increased local and systemic inflammation caused by infection, pollution, or other insult to the airway.

The conditions that mimic COPD exacerbation are:
- Pneumonia
- Pneumothorax
- Cor pulmonale
- Pulmonary Edema
- Myocardial Infarction, Arrhythmia
- Pulmonary Embolism
- Upper airway obstruction
- Pleural Effusion

Exacerbation severity is assessed by using Anthonisen criteria, as detailed in chapter.[7]

Investigations that are needed include:
- CBC, CRP, and D dimer.
- Sputum gram stain and C/S.
- Sputum AFB smear, C/S, and Gene Xpert.
- Serum urea creatinine, Electrolytes
- Arterial blood gases, Peak Expiratory Flow Rate.
- Chest X-ray, CTPA.
- ECG, ECHO, and NT-pro BNP.

Classification of COPD Exacerbation Severity[5]
When evaluating a patient with suspected COPD exacerbation, it is important to confirm the diagnosis and assess the severity of the episode while also considering alternative diagnoses such as heart failure, pneumonia, or pulmonary embolism.

Mild Exacerbation:
- Dyspnea Visual Analog Scale (VAS) score less than 5
- Respiratory rate (RR) under 24 breaths per minute
- Heart rate (HR) below 90 bpm
- Oxygen saturation (SaO_2) ≥ 92% on room air or baseline oxygen use (with <3% change from baseline, if known)
- C-reactive protein (CRP) < 10 mg/L (if measured)

Moderate Exacerbation (requires ≥3 of the following):
- Dyspnea VAS ≥ 5
- RR ≥ 24 breaths per minute

- HR ≥ 90 bpm
- SaO_2 < 92% on room air or baseline oxygen (with ≥3% drop from known baseline)
- CRP ≥ 10 mg/L
- Arterial blood gases (ABG), if taken, may show:
    - Hypoxemia (PaO_2 < 60 mmHg) and/or
    - Hypercapnia ($PaCO_2$ > 45 mmHg) without acidosis

Severe Exacerbation:

- Clinical indicators same as moderate, but ABG reveals **worsening hypercapnia and acidosis** ($PaCO_2$ > 45 mmHg and pH < 7.35)

Following severity classification, it's essential to determine the underlying etiology through investigations such as viral panels, sputum cultures, or other relevant tests.

The table below (Table 14.1) enlists the indications to treat AECOPD at home and for hospital admission.

Table 14.1: Indications for hospitalization versus treatment at home of COPD exacerbation.

	Treat at Home	Treat in Hospital
Ability to cope at home	Yes	No
Dyspnea	Mild	Severe
General condition	Good	Poor and worsening
Level of activity	Good	Poor
Cyanosis	No	Yes
Worsening peripheral Edema	No	Yes
Level of consciousness	Normal	Impaired
On supplemental oxygen	No	Yes
Social support	Good	Not coping or alone
Acute confusion	No	Yes
Changes on Chest X-Ray	No	Yes
Rapid rate of onset	No	Yes
Arterial Ph	= 7.35	< 7.35
PaO2	= 52 mmHg	< 52 mmHg

Treatment for AECOPD Treated At Home

1. Add or increase dose of bronchodilator, metered dose inhalers are preferred with spacer device. Dose of salbutamol is 2 puff hourly (100 mg/puff) then 3 – 4 hourly. Ipratropium bromide 2 puff 4 hourly (20 – 40 mg) can be added.
2. If response is not adequate add 200 – 400 mg twice daily of sustained release theophylline.

3. Add antibiotic if any evidence of infection. Amoxicillin ± clavulanate can be a good first line option. Other antimicrobials that can be used are respiratory quinolones and macrolides.
4. Oral steroids are not recommended in mild exacerbations but can be prescribed for more severe symptoms and the dose is 30mg/day for one week.

Management of AECOPD At Hospital

1. Give controlled oxygen. Use Venturi mask or nasal cannula. Target SpO2 level is 88 - 92%.
2. Get ABGs in first hour and within one hour of change in FIO2.
3. Start bronchodilators. The dose of salbutamol is 5 mg (1ml) diluted in 2 – 3 ml normal saline ± ipratropium bromide 500 µg. Metered dose inhaler can be used as an alternate. Salbutamol up to 6 to 8 puff every half hour and/or ipratropium bromide 6 to 8 puffs every 3 to 4 hours can be used if nebulizer is not available. If PEFR is 200 L/min or FEV1 is 500 ml then compressor driven nebulizer is preferred.
4. Hydrocortisone 250 mg intravenous stat dose then 100 mg intravenous 8 hourly. Switch to oral 30 – 40 mg/day preferably as single morning dose when patient is able to take it by mouth. Therapy can be given for up to 14 days.
5. Start antibiotic. Respiratory quinolones, amoxicillin-clavulanate or 2nd or 3rd generation cephalosporin is preferred. Gam negative infection is more common in severe COPD so ciprofloxacin or 3rd gen cephalosporin are given.
6. Theophylline has a narrow therapeutic index and has major drug interactions with other drugs like erythromycin, so it should be used with caution. If patient has not taken it in last 24 hours give loading dose. It should be given over 30 minutes followed by a maintenance infusion at the rate of 0.5mg/kg/hour. Higher doses up to 0.9 mg/kg/hour can be used in children, young adults and smokers. Use small doses of 0.25 mg/kg/hour if status of prior theophylline use is doubtful.

   Loading dose = Required plasma concentration x volume of drug distribution

   = 10 mg/L x body weight in kg x 0.5

7. Get ABGs after 1 hour of full medical management. Shift the patient to intensive care unit (ICU) if condition continues to deteriorate or ABGs worsened. Patients with hemodynamic instability or those planned for invasive mechanical ventilation should be managed in ICU. ICU admission may not be appropriate for patients with poor functional status or end stage lung disease.
8. Consider prophylaxis for venous thromboembolism.
9. Keep adequate fluid and nutritional balance.
10. Patients must be stratified into 5 treatment escalation groups on admission and managed accordingly particularly in resource constraint settings:
    a. Requiring immediate intubation and ventilation
    b. Suitable for NIV and suitable for escalation to intubation
    c. Suitable for NIV but not suitable for escalation to intubation
    d. Not suitable for NIV but for full active medical management
    e. Palliative care agreed as most appropriate management

11. Airway clearance by physiotherapy techniques, like manual percussion, the use of electrical percussor, cough assist machine, acapella device, vibration vest etc.

Ventilatory Support[5]

The main aim of ventilator support in patients with AECOPD is to reduce morbidity and mortality and to relieve symptoms. Both noninvasive and invasive mechanical ventilation can be used. NIV should be the preferred first line mode of ventilation for patients in acute respiratory failure.

Indications for Noninvasive Mechanical Ventilation (NIV)

NIV should be initiated in patients with COPD exacerbations if any of the following are present:

- Respiratory acidosis, indicated by a $PaCO_2 \geq 6.0$ kPa (45 mmHg) and arterial pH ≤ 7.35.
- Severe dyspnea with signs of respiratory muscle fatigue or increased work of breathing—such as use of accessory muscles, abdominal paradox, or intercostal retractions.
- Persistent hypoxemia despite receiving supplemental oxygen.

Indications for Invasive Mechanical Ventilation

Invasive ventilation is warranted under the following conditions:

- Failure to tolerate or respond to NIV.
- Cardiac or respiratory arrest.
- Reduced consciousness or agitation not manageable with sedation.
- Massive aspiration or ongoing vomiting.
- Inability to clear respiratory secretions.
- Severe circulatory instability unresponsive to fluids or vasoactive medications.
- Presence of serious arrhythmias (ventricular or supraventricular).
- Severe, life-threatening hypoxemia in those who cannot undergo NIV.

NIV with face mask should not be given in following conditions:

1. Respiratory arrest
2. Cardiovascular instability (arrhythmias, hypotension, myocardial infarction)
3. Altered level of consciousness – BiPAP can be used with caution
4. Uncooperative patient
5. Risk of aspiration
6. Copious secretion
7. Craniofacial trauma
8. Nasopharyngeal abnormality.

The decision to use invasive ventilation in end stage COPD is influenced by patients' wishes and likelihood of reversing the acute event.

BiPAP through endotracheal tube can be used in patients not fit for invasive mechanical ventilation.[4] Many patients respond to this treatment as most of the time it is the lack to clear airways of copious viscous secretions, which can be easily handled by endotracheal tube.

Discharge Criteria and Follow-up Recommendations.[5]

Criteria Before Discharge

Before discharging a patient, the following steps should be completed:

1. Conduct a thorough review of all clinical assessments and laboratory results.
2. Confirm the patient's understanding and correct usage of maintenance therapy.
3. Re-evaluate the patient's inhaler technique.
4. Assess the need to discontinue any short-term medications, including steroids and/or antibiotics.
5. Determine if there is an ongoing need for supplemental oxygen.
6. Develop a clear management plan addressing existing comorbidities and necessary follow-up.
7. Arrange for follow-up visits, including an early follow-up within 4 weeks and a later follow-up before 12 weeks, depending on the patient's condition.
8. Ensure all clinical concerns and abnormal investigation findings have been addressed.

Follow-up Timeline

1 to 4 Weeks Post-Discharge

- Assess the patient's ability to function in their usual living environment.
- Reconfirm their understanding of the treatment plan.
- Reassess inhaler usage and technique.
- Evaluate the ongoing need for oxygen therapy.
- Review physical functioning and consider referral for pulmonary rehabilitation if appropriate.
- Record symptoms using standardized tools like the CAT (COPD Assessment Test) or mMRC (Modified Medical Research Council) scale.
- Document the presence and management of any comorbid conditions.

12 to 16 Weeks Post-Discharge

- Reassess the patient's adaptation to their normal environment.
- Review comprehension of and adherence to the treatment regimen.
- Reevaluate inhaler use.
- Recheck the need for long-term oxygen therapy.
- Assess daily physical activity and the ability to carry out everyday tasks.
- Conduct spirometry, particularly FEV1 (Forced Expiratory Volume in 1 second), to evaluate lung function.
- Document symptoms using the CAT or mMRC tools.
- Review and document any comorbid conditions.

Key Differences Between Early And Late Follow-Up:

- **Timing:** 1–4 weeks is an early safety net to catch problems soon after discharge; 12– 16 weeks assesses longer-term disease control.
- **Depth:** The later follow-up includes objective testing (spirometry) and focuses on functional integration into daily life.
- **Goal:** The early visit ensures the patient is coping; the late visit ensures the treatment is effective and sustainable.

Interventions that reduce the frequency of COPD Exacerbation (Figure 14.1)

Intervention Class	Intervention
Bronchodilators	LABA, LAMA LABA + LAMA
Corticosteroid containing regimens	LABA + ICS LABA+LAMA + ICS
Anti-Inflammatory (non-steroid)	Roflumilast Dupilumab
Anti-infectives	Vaccines Long term macrolides
Mucoregulators	Long term macrolides N-acetylcysteine Carbocysteine Erdosteine
Various others	Smoking cessation Rehabilitation Lung volume reduction Vitamin D Shielding measures (mask wearing, minimizing social contacts, frequent hand washing)

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Chapter 15:
Drugs Used In COPD

Bronchodilators remain the mainstay of pharmacologic management of COPD. They increase FEV1 and/or change other spirometry variables. But their dose-response curve is flat so dosing is not based on spirometry response. Bronchodilators alter airway smooth muscle tone and tend to reduce dynamic hyperinflation, and improve exercise performance. Toxicity is dose dependent.

There are three main classes of bronchodilators:

- Beta agonist
- Anticholinergics
- Methylxanthines

Table 15.1: shows formulations of commonly used β-agonists.
Table 15.2: shows formulations of commonly used anticholinergics.
Table 15.3: shows formulations of methylxanthines.

A number of fixed-dose combinations of β-agonists and anticholinergics are available. See table 15.4.

Table 15.1 Formulations of commonly used beta agonists.

Drugs	**Inhaler (mug)**	**Solution for nebulizer (mg/ml)**	**Oral**	**Vials for injection**	**Duration of action**
Short-acting					
Fenoterol *	100-200 MDI	1	2.5mg pill 0.05% syp		4-6 hours
Levalbuterol	45-90 MDI	0.1,0.21,0.25			6-8 hours
Salbutamol	100-200 MDI, DPI	1,2,2.5,5	2,4,5 mg pill 8mg ER pill 0.4mg syp	0.1 mg 0.5mg	4-6 hours 12 hours ER
Terbutaline	500 DPI		2.5, 5 mg pill	.2, .25, 1 mg	4-6 hours
Long acting					
Formoterol	**4.5-9 DPI**	.01			12 hours
Indacaterol	**75-300 DPI**				24 hours
Olodaterol	**2.5, 5 SMI**				24 hours
Salmeterol	**25-50** MDI, DPI				12 hours

DPI; Dry powder inhaler. **MDI;** Meter dose inhaler. **SMI;** Soft mist inhaler.
Side effects of beta agonists Sinus tachycardia, tremors, and hypokalemia show Tachyphylaxis

Table 15.2: Formulations for commonly used anti-cholinergic

Drugs	Inhalers Micro gram	Solution for nebulizer mg/ml	Oral	Vial for injection	Duration of action
Short acting					
Ipratropium bromide	20,40 MDI	0.2			6-8 hours
Long acting					
Aclidinium Br.	400 MDI/DPI				12 hours
Glycopyrronium	15.6, 50 MDI		1mg	0.2mg	12-24 hours
Tiotropium	18 DPI 2.5, 5 SMI				24 hours
Umeclidinium	62.5 DPI				24 hours

DPI: Dry powder inhaler. **MDI:** Meter dose inhaler. **SMI:** Soft mist inhaler Side effects of anticholinergic: Dry mouth, bitter taste

Figure 15.3: Formulations of Methylxanthines

Drugs	Oral	Vial for injection	Duration of action
Aminophylline	105 mg/ml	250, 500mg	Variable up to 24 hours
Theophylline SR	100-600mg pill	250, 400, 500 mg	Variable up to 24 hours

Side effects of methylxanthines: Palpitations, atrial and ventricular arrhythmias, grand mal seizures. headache, nausea, insomnia, heartburn. Toxicity is dose related.

Table 15.4: Fixed-dose combinations of β-agonists and anticholinergics are available.

Drugs	Inhaler	Solution for nebulizer	Duration of action
Fenoterol/ipratropium	50/20 SMI		
Salbutamol/ipratropium	100/20 SMI 75/15 MDI		
Formoterol/aclidinium	12/400 DPI		
Formoterol/glycopyrronium	9.6/14.4 MDI		
Indacaterol/glycopyrronium	27.5/15.6 DPI		
Vilanterol/umeclidinium	25/62.5 DPI		
Olodaterol/tiotropium	5/5 SMI		

Glucocorticosteroids

Oral steroids should be avoided, if possible, in stable COPD. Systemic steroids may be used for short-term treatment (7-14 days) during exacerbations.

Inhaled corticosteroids (ICS) have modest bronchodilator effect. They reduce exacerbation severity and frequency.

Drugs commonly used are:

Inhaled corticosteroids:

Beclomethasone	500 μg twice a day
Triamcinolone	400 μg twice a day
Budesonide	400 μg twice a day
Fluticasone	100 – 500 μg twice a day

Oral corticosteroid:
Prednisolone...30 mg/day

Combination of long acting β – agonist and inhaled corticosteroids are available:
Formoterol/ beclomethasone............................/100 MDI & DPI
Formoterol/budesonide...................................../160, 4.5/80 MDI
9/320, 9/160 DPI
Formoterol/mometasone................................../100, 10/400 MDI
Salmeterol/fluticasone......................................./100, 50/250, 5/500 DPI
21/45, 21/115, 21/230 MDI
Vilanterol/fluticasone furoate............................../100 DPI

Side effects of steroids: Increased risk of osteoporosis, cataract, adrenal suppression, hypertension, diabetes, obesity, skin thinning, muscle de-conditioning, reactivation of tuberculosis.

Tripple fixed dose inhaler combination (ICS+LAMA+LABA)
Fluticasone furoate+ Umeclidinium+ Vilanterol (100/62.5/25)

Phosphodiesterase 4 inhibitors
Roflumilast is the PDE-4 inhibitor currently in use. It is 500 µg pill, to be taken orally once a day. It has no direct bronchodilator activity; it reduces inflammation.

Roflumilast —PDE4 X→ Increased the level of cAMP ⊣ inflammation

Figure 15.2: Mechanism of action of roflumilast.
Side effects of roflumilast: Diarrhea, nausea, decreased appetite, abdominal pain, weight loss, headache and sleep disturbance.

Vaccines
Discussed in chapter 13.

α1-antitrypsin augmentation therapy
It is not available in Pakistan. Intravenous therapy is indicated in patients with α1-antitrypsin deficiency who have progressive disease and FEV1 > 65%.

Antibiotics
Azithromycin 250 mg/day or 500 mg three times per week for 1 year may reduce exacerbation risk. Clarithromycin 250mg /day in advanced COPD having bronchiectasis.

Mucolytics
Carbocisteine, N-acetylcysteine, and Erdosteine can be used, they may reduce exacerbations.

Antitussives
Despite the high prevalence of chronic cough in COPD, the GOLD 2025 report does not recommend the use of antitussives (e.g. dextromethorphan, codeine) in the routine management of COPD. Chronic cough in COPD is typically related to airway inflammation, mucus production, and airflow limitation, rather than isolated cough reflex hypersensitivity.

Management should therefore focus on addressing underlying pathophysiology using:

- Bronchodilators (LABA, LAMA)
- Anti-inflammatory agents (ICS, roflumilast)
- Mucolytics (Carbocisteine, N-acetylcysteine)
- Targeted biologics (e.g. dupilumab for eosinophilic phenotype)

Antitussives are not included in GOLD algorithms due to lack of supporting evidence and the concern that suppressing protective cough may lead to mucus retention. In cases of severe or unexplained cough, alternative diagnoses such as GERD, post-nasal drip, or medication side effects should be considered.

New Pharmacologic agents in COPD:

Ensifentrine

Ensifentrine is a novel first-in-class dual inhibitor of phosphodiesterase-3 (PDE3) and phosphodiesterase-4 (PDE4), combining bronchodilator and anti-inflammatory properties. It is administered via nebulization, showing improvement in lung function, symptoms, and quality of life in patients with moderate to severe COPD.

Mechanism of Action:

- PDE3 inhibition leads to bronchodilation
- PDE4 inhibition reduces inflammation
- Results in increased intracellular **cAMP,** smooth muscle relaxation, and reduced cytokine release

Formulation:

- Nebulized solution: Ensifentrine 3 mg twice daily

Side Effects:

- Cough, nasopharyngitis, headache, diarrhea

Ensifentrine may be considered for patients with persistent symptoms despite standard therapy, especially that intolerant to or ineligible for ICS use.

Dupilumab

Dupilumab is a **monoclonal antibody** that targets the IL-4 receptor alpha subunit, blocking the signaling of **IL-4 and IL-13,** key cytokines in type 2 inflammation.

Indications:

- Approved for use in COPD patients with evidence of eosinophilic inflammation (e.g., blood eosinophils ≥300/μL)
- Reduces the rate and severity of exacerbations

Formulation:

- **Subcutaneous injection:** 300 mg every **2 weeks**

Side Effects:

- Injection site reactions, conjunctivitis, eosinophilia, headache

Dupilumab represents a new targeted therapy for a **biomarker-defined subgroup** of COPD patients with type 2 inflammation.

References:

Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. 2025.

Chapter 16:
Air Travel In COPD

COPD patients should be informed about the "fitness to fly" concept and patients with moderate-to-severe disease need to be assessed for possible risk factors for in-flight hypoxemia before flying.[1,2] Figure 16.1 shows pathophysiological changes during air travel in COPD patient.

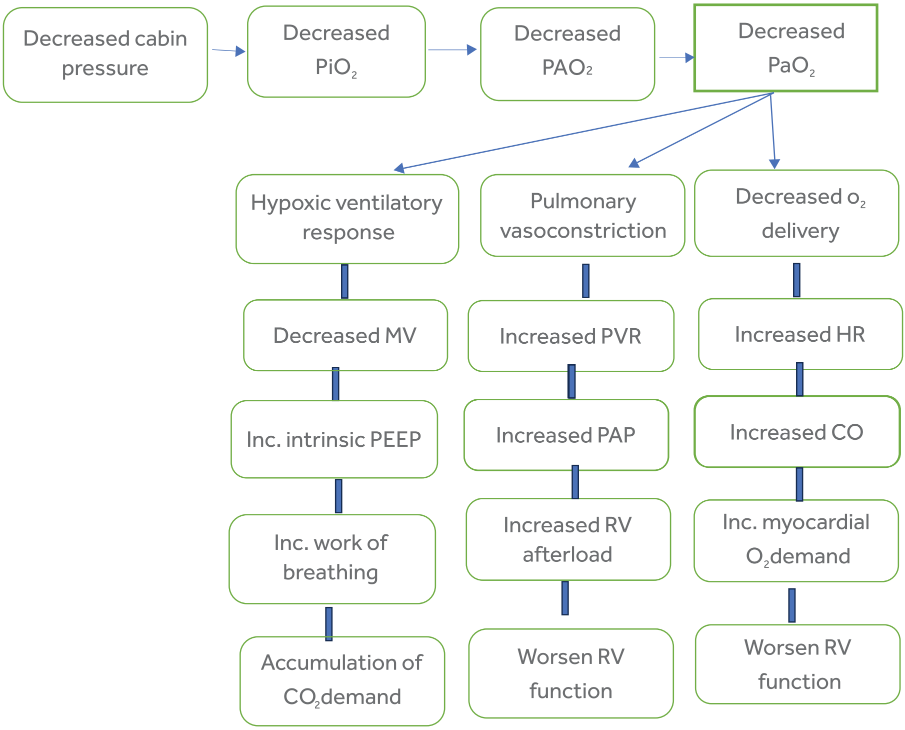

Figure: 16.1: Pathophysiologic changes in COPD patient during air travel

CO: Cardiac output; HR: Heart rate; MV: Minute ventilation; PAO2: Partial pressure of oxygen in alveoli; PaO2: Partial pressure of oxygen in arterial blood; PAP: Pulmonary artery pressure; PIO2: Pressure of inspired oxygen; RV: Right ventricle; WOB: Work of breathing

Following algorithm can be used for assessment of fitness to fly in COPD patient (Figure 16.2).

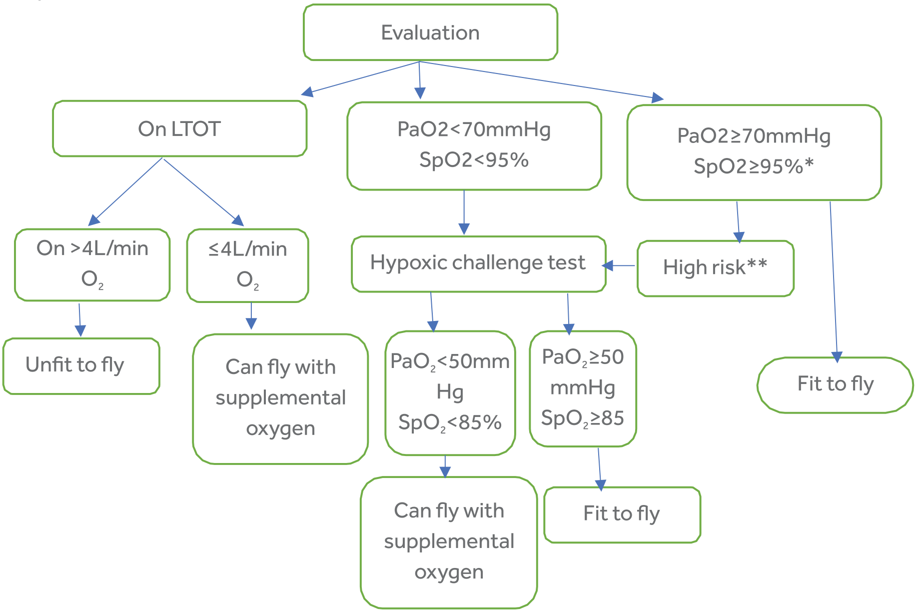

Figure 16.2: Algorithm for assessment of fitness to fly in COPD patient
* Resting SpO2 >95% combined with a 6-min walk test SpO2 >84% has a sensitivity of 100% and specificity of 80% for fitness to fly
** Dyspnea on exertion, forced expiratory volume in 1 second <1.5L or <30% predicted, a pre-existing requirement of oxygen or ventilator support, bullous lung disease, comorbid conditions that may worsen hypoxemia like cardiac disease and significant symptoms during previous air travel. For high-risk patients do hypoxic challenge test and follow the algorithm.
The safe threshold for oxygenation is to keep PaO2 >50 mmHg and SpO2 ≥85% during the flight.

Problems Other Than Hypoxia

Jet lag: general advice and treatment options for jet lag (sleep hygiene, melatonin, etc.) are also valid for COPD patient.

Coagulation activation: the risk increases after 4 h and peaks at 8 h. Patients with low risk should be warned to avoid tight clothing below the waist and try to rehydrate during the flight. Moving along the aisle may be an option for patients without risk of desaturation. If the patient has a risk of desaturation, below knee compression stockings and calf stretching exercises while sitting may be a safe alternative. Patients with high risk of thromboembolism (previous venous thromboembolism history, known active malignancy, known thrombophilia, and major surgery within 6 weeks) should ask for individual help for prophylactic low molecular weight heparin.

Risk of infection transmission: Patients should be warned, especially for outbreaks of RSV (SARS), and should be advised to postpone travel if necessary.

References

1.Ergan B, Akgun M, Pacilli AMG, et al. Should I stay or should I go? COPD and air travel. European Respiratory Review 2018 27: 180030.

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Chapter 17:
Post-TB Chronic Obstructive Pulmonary Disease

Pakistan is ranked fifth among high burden TB countries globally and it accounts for 61% of the TB burden in the WHO Eastern Mediterranean Region. TB is now recognized as a risk factor for developing chronic obstructive airway disease.[1]

Patients of pulmonary tuberculosis may develop airflow obstruction either during the active phase or post-treatment phase of the disease.

A study from Pakistan revealed that 55.3% of treated pulmonary TB patients with dyspnea had an obstructive ventilatory defect.[2] Patients with COPD secondary to TB have been shown to have significantly low forced expiratory volume in one second, higher airway resistance, and poor positive bronchodilator response (27% vs 82%) than only COPD patients.[3] These patients are also at risk for more frequent exacerbations of obstructive airway disease.[4]

The exact mechanism of airway obstruction in post-tuberculosis patients is not clear. Following mechanisms have been proposed:[5]

- Bronchiectasis
- Bronchiolar narrowing
- Bronchiolitis obliterans
- Accelerated emphysematous changes

Figure 17.1: shows the mechanism of airflow obstruction due tuberculosis.

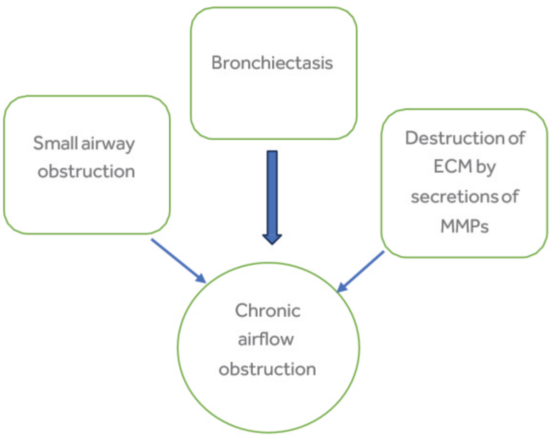

Figure 17.1: Mechanism of airflow obstruction due tuberculosis. **ECM:** extracellular matrix, **MMPs:** matrix metalloproteinases

Treatment for chronic airflow disease due to tuberculosis is the same as that of COPD.[6]

COPD and tuberculosis are major health problem in developing countries. Early diagnosis and appropriate treatment of tuberculosis is emphasized to reduce the future burden of COPD.

References

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5.Allwood BW, Myer L, Bateman ED. A systematic review of the association between pulmonary tuberculosis and the development of chronic airflow obstruction in adults. Respiration. 2013;86(1):76–85

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Chapter 18:
Artificial Intelligence in Pulmonology and COPD Care

Introduction

Artificial Intelligence (AI) refers to computational systems capable of tasks that traditionally require human intelligence, including pattern recognition, prediction, and decision-making. In clinical medicine, AI is increasingly used as a decision-support tool to enhance diagnostic accuracy, optimize treatment strategies, and improve patient outcomes.

Pulmonology is particularly well-suited for AI integration due to its reliance on imaging (chest radiography, computed tomography, spirometry, arterial blood gases), and chronic disease monitoring. In the context of COPD, AI offers opportunities for early diagnosis, risk stratification, and personalized management, consistent with the evolving paradigm of precision medicine emphasized in modern guidelines.

Scope of Artificial Intelligence in Pulmonology

Diagnostic Applications

AI-based tools have demonstrated utility in:

- Interpretation of chest radiographs for tuberculosis, pneumonia, and lung nodules
- Quantitative analysis of CT scans for emphysema and airway disease
- Automated interpretation of spirometry and pulmonary function tests

These applications improve diagnostic consistency and may support clinicians in resource-limited settings.

Risk Prediction and Prognostication

Machine learning models can integrate multiple clinical variables to:

- Predict COPD exacerbations
- Identify patients at risk of hospitalization or ICU admission
- Estimate disease progression and mortality

Such predictive tools align with the need for proactive rather than reactive care.

Treatment Optimization

AI can assist in:

- Identifying patients likely to benefit from inhaled corticosteroids (ICS) based on eosinophil counts
- Supporting escalation to triple therapy (LABA/LAMA/ICS)
- Predicting response to targeted therapies, including biologics

Monitoring and Remote Care

AI-enabled systems facilitate:

- Remote monitoring using wearable devices
- Early detection of symptom deterioration
- Telemedicine-based follow-up

This is particularly relevant in geographically dispersed populations with limited access to specialist care.

Artificial Intelligence in COPD: Alignment with GOLD 2026

The Global Initiative for Chronic Obstructive Lung Disease (GOLD 2026) emphasizes individualized, risk-based management of COPD. AI supports this approach in several domains [1]:

Early Detection

AI algorithms combining imaging and spirometry data can identify COPD at earlier stages, potentially before significant functional decline occurs.

Prediction of Exacerbations

- AI models can integrate:
- Prior exacerbation history
- Symptom burden

Biomarkers such as CRP and eosinophils

to predict acute exacerbations of COPD (AECOPD), enabling early intervention.

Personalized Pharmacological Therapy

AI supports GOLD 2026 recommendations by:

- Identifying candidates for ICS based on eosinophil thresholds
- Guiding escalation to triple therapy
- Supporting selection of biologics (e.g., dupilumab) in patients with type 2 inflammation

Acute Exacerbation Management

In hospitalized patients, AI tools may:

- Detect early respiratory deterioration
- Support timely initiation of non-invasive ventilation (NIV)
- Assist in ICU triage decisions

Alignment with International Respiratory Guidelines

The integration of AI into clinical practice is increasingly recognized by major respiratory bodies, including:

- American Thoracic Society (ATS)
- European Respiratory Society (ERS)
- World Health Organization (WHO)

These organizations emphasize that AI should:

- Be clinically validated before implementation
- Complement, not replace, clinician judgment
- Be used in accordance with ethical and regulatory standards

Artificial Intelligence-Supported COPD Management Pathway (Figure 15.1)

Patient with suspected / confirmed COPD

↓

Data Input Layer (History, imaging, PFTs, and Biomarkers)

↓

AI analytic engine (Diagnosis support, Exacerbation risk prediction, Treatment response prediction, Severity Stratification)

↓

Stable COPD (LABA / LAMA, ± ICS (eosinophil-guided), Consider triple therapy, Pulmonary rehab)

↓

High Risk/ Frequent Exacerbator (Escalate to triple therapy, consider roflumilast / macrolides, and evaluate for biologics

↓

Acute Exacerbation (SABA ± SAMA, Steroids (Pred 40 mg × 5 days), Antibiotics (if indicated), NIV if respiratory acidosis)

↓

Follow-up and Monitoring (AI-based remote monitoring, Early exacerbation alerts Therapy adjustment)

↓

Improved Outcome (Reduced exacerbations, Reduced hospitalizations, Personalized care)

Knowledge, Attitude, and Practice (KAP) of AI Among Pulmonologists
Overview
A national KAP study conducted among pulmonologists in Pakistan evaluated awareness, perceptions, and utilization of AI in respiratory medicine[2].

Key Findings
Knowledge:
Variable understanding of AI applications, with gaps in clinical integration
Attitude:
Generally positive, with clinicians recognizing the potential of AI to improve patient care
Practice:
Limited real-world use due to lack of infrastructure, training, and access to AI tools

Interpretation
The findings highlight a readiness–implementation gap, where clinicians are willing to adopt AI but face systemic barriers. This underscores the need for structured integration strategies within national guidelines.

Barriers to Implementation in LOW and Middle-Income Settings
Key challenges include:
- Limited digital infrastructure
- Lack of formal training in AI applications
- High cost of implementation
- Data privacy and security concerns
- Absence of regulatory frameworks

Addressing these barriers is essential for effective adoption.

Despite relatively adequate knowledge levels, only 4.5% of participants demonstrated good practice, while the majority (95.5%) had poor practice, indicating a significant knowledge–practice gap. The distribution of knowledge, attitude, and practice levels among participants is shown in Table[15.1].

Domain	Category	Percentage (%)
Knowledge	Poor	1.5
	Moderate	66.2
	Good	32.3
Attitude	Negative	4.6
	Neutral	80.5
	Positive	14.9
Practice	Poor	95.5
	Good	4.5

Table: 15.1 Knowledge, Attitude, and Practice.

The findings give in table 15.2 suggest that AI integration remains limited and largely confined to specific domains.

Variable	Category	Frequency	Percentage (%)
AI Use	Yes	93	46.3
	No	108	53.7
Training	Yes	15	7.5
	No	186	92.5
Radiology Use	Yes	28	13.9
	No	173	86.1
Research Use	Yes	64	31.8
	No	137	68.2
ICU Use	Yes	64	31.8
	No	137	68.2

Table: 15.2 Pattern of AI utilization in academic and clinical settings.

Ethical and Clinical Considerations

AI implementation must adhere to the following principles:

AI should function as a decision-support tool, not a replacement for clinicians

- Transparency in algorithm decision-making is essential
- Risk of bias must be minimized through diverse datasets
- Patient confidentiality and data protection must be ensured
- Clinical accountability remains with the treating physician

Recommendations for integration in Pakistan

Short-Term Strategies

- Incorporate AI education into pulmonology training programs
- Utilize AI in radiology for screening (e.g., tuberculosis detection)

Medium-Term Strategies

- Introduce AI-assisted spirometry interpretation
- Implement AI-based risk prediction tools in COPD clinics

Long-Term Strategies

- Develop national AI registries for respiratory diseases
- Establish tele-pulmonology networks supported by AI
- Integrate AI into electronic health record systems

Future Directions

Emerging areas of interest include:

- AI-guided selection of biologic therapies in COPD
- Integration with wearable technology for continuous monitoring
- AI-driven public health strategies for tuberculosis and chronic lung disease

Key Messages

- Artificial Intelligence enhances clinical decision-making but does not replace clinician judgment
- AI supports the personalized management approach advocated by GOLD 2026
- There is a significant readiness for AI adoption among pulmonologists, but implementation remains limited
- Structured training, infrastructure development, and regulatory frameworks are essential for successful integration

References:

1.Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: 2026 report. Fontana (WI): GOLD; 2026.

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