

## The Review Article on Chronic Obstructive Pulmonary Disease (COPD)

1} Bhumika Jitendra Chavan

2} Roshani Sunil Patil

3} Pratiksha Anil Patil

4} Shweta Valmik Nagare

5} Vaishnavi Sandip Nikumbh

6} Shrushti Sunil Chitte

Guidance By :- 1} Mr.Mohammed Awais

2} Mr. Nazeer Ahmed

### Abstract

Chronic obstructive pulmonary disease (COPD) is a heterogeneous disease. Historically, two COPD phenotypes have been described: chronic bronchitis and emphysema. However, these phenotypes provide additional properties of disease pathophysiology, but are not sufficient to reflect the heterogeneity of COPD, and do not provide a detailed classification that provides specific treatments for specific treatments, except for the enthusiastic glucocorticoids (ICS) of patients with chronic bronchitis. This overview describes the COPD phenotype. The COPD phenotype provides predictions and/or provisions for a particular treatment. However, we also discuss phenotypes such as COPD, which are additional predictions for future clinical research predictions, but do not necessarily meet current diagnostic criteria for COPD.

### Introduction

Chronic obstructive pulmonary disease (COPD) is an avoidable and treatable disease due to respiratory obstruction and persistent respiratory symptoms. Used, older than 40 years old and men. 1

Preservative agents in patients with moderate to severe COPD

### Cardiac therapy 1

Even though recommendations are using up to 71%. If symptoms are well treated and lung loss can lead to lungs, "stable" and "unstable COPD" treatment (in patients with more or more clinical development in pulmonary function) is challenging.

Aggravation from COPD can contribute significantly to the financial burden and, depending on the severity, lead to the need for emergency department (ed.) and hospital stays. The 2014 programme, which includes COPD, underscored the need for better management of management to reduce frequent hospital facilities and reduce takeovers in connection with serious illnesses.

Describes how appropriate treatment can improve outcomes and reduce costs associated with the use of health care systems.

### Asthma COPD Overlap and COPD With Eosinophilia

Asthma COPD overlap is a relatively new term that describes the coexistence of asthma and COPD. The definitions are proposed by scientific organizations. This has proposed the Global Initiative for Chronic Obstructive Pulmonary Disease (GOLD) and the Global Initiative for Asthma (GINA), but there is no prediction or specific treatment. Bronchodilator Responses (BDRs) are one of several criteria for confirming pulmonary function variability, and the presence of BDR is often mistakenly equivalent to an asthma diagnosis. BDR is increasingly higher in asthma patients than COPD, but BDR asthma and COPD are indistinguishable. Early studies showed that BDR could not predict response to treatment. The only consequence of consistent connection to BDR is the reduction in FEV1 over time. Inconsistent findings regarding the association and clinical outcomes of BDRs may be related to various BDR definitions and protocols. In COPD, an increase in FEV1 and FVC was associated with 12% and 200 mL after administration of bronchodilators. BDRs in FVC are usually connected to greater pneumonia and small functional respiratory disease. A drawback of BDR is its instability over time. A recent study has shown that consistent BDR is connected to people with previous or current smoking with previous asthma diagnosis, reduced lung function, and functional pneumoretic respiratory disease. It is usually available with COPD. sputum and blood cell counts indicate responses to COPD patients with ICS and blood cell counts that show 300 cells/ $\mu$ L. and Interleukin-4/interleukin-13 receptors reduce exacerbations in COPD patients and in patients with increased blood eosinophil counts. After

There was a greater response in patients with FEV1 BDR. This is the first study showing that BDR can demonstrate responses to specific pharmacological components.

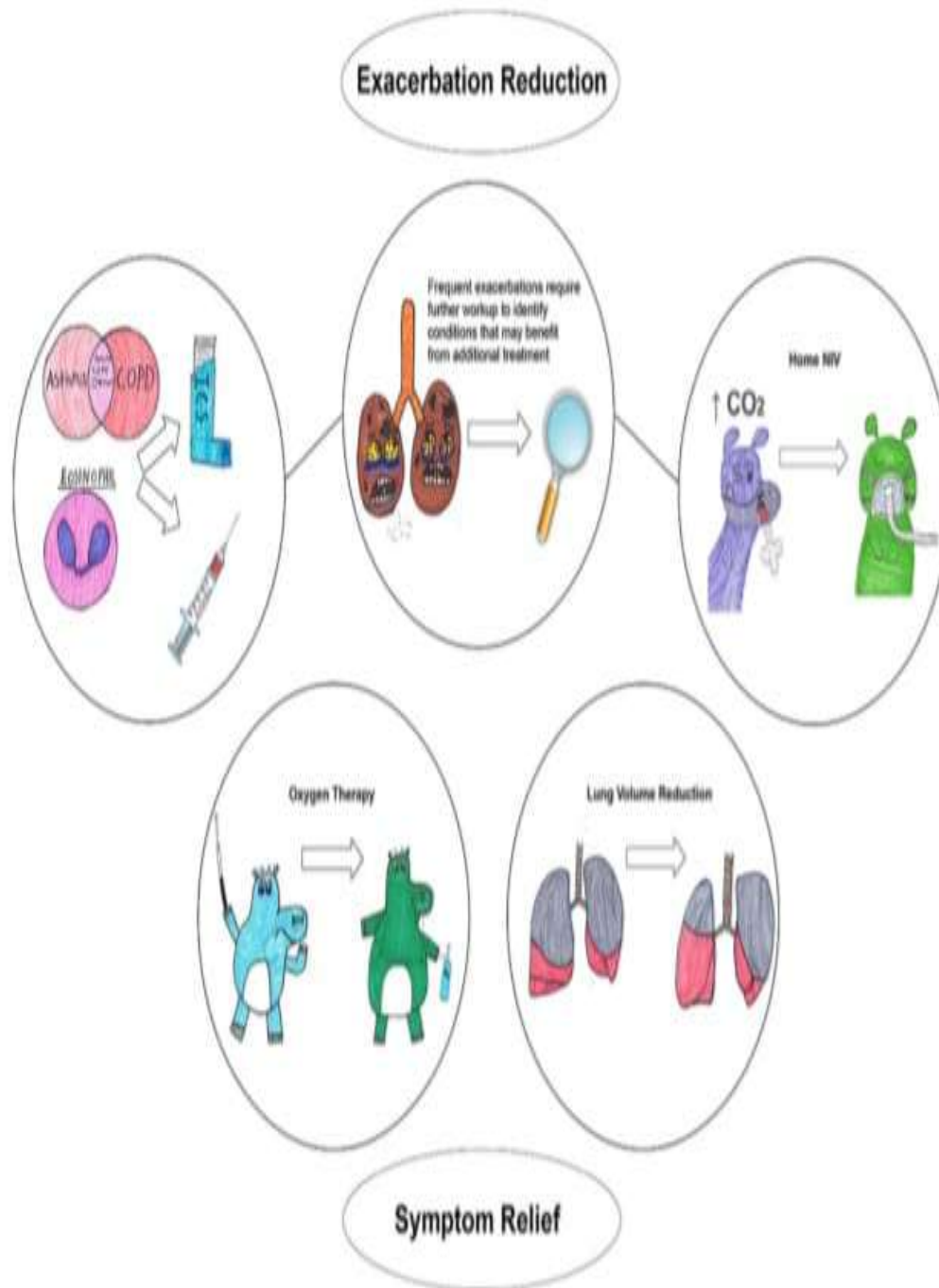
### COPD With Static Hyperinflation

Hyperinflation often occurs in COPD as the disease progresses. It is known to be related to inadequate predictions (24°C. 27). Pioneering research by Casanova et al.

### Ratio of Lung Volume (TLC)

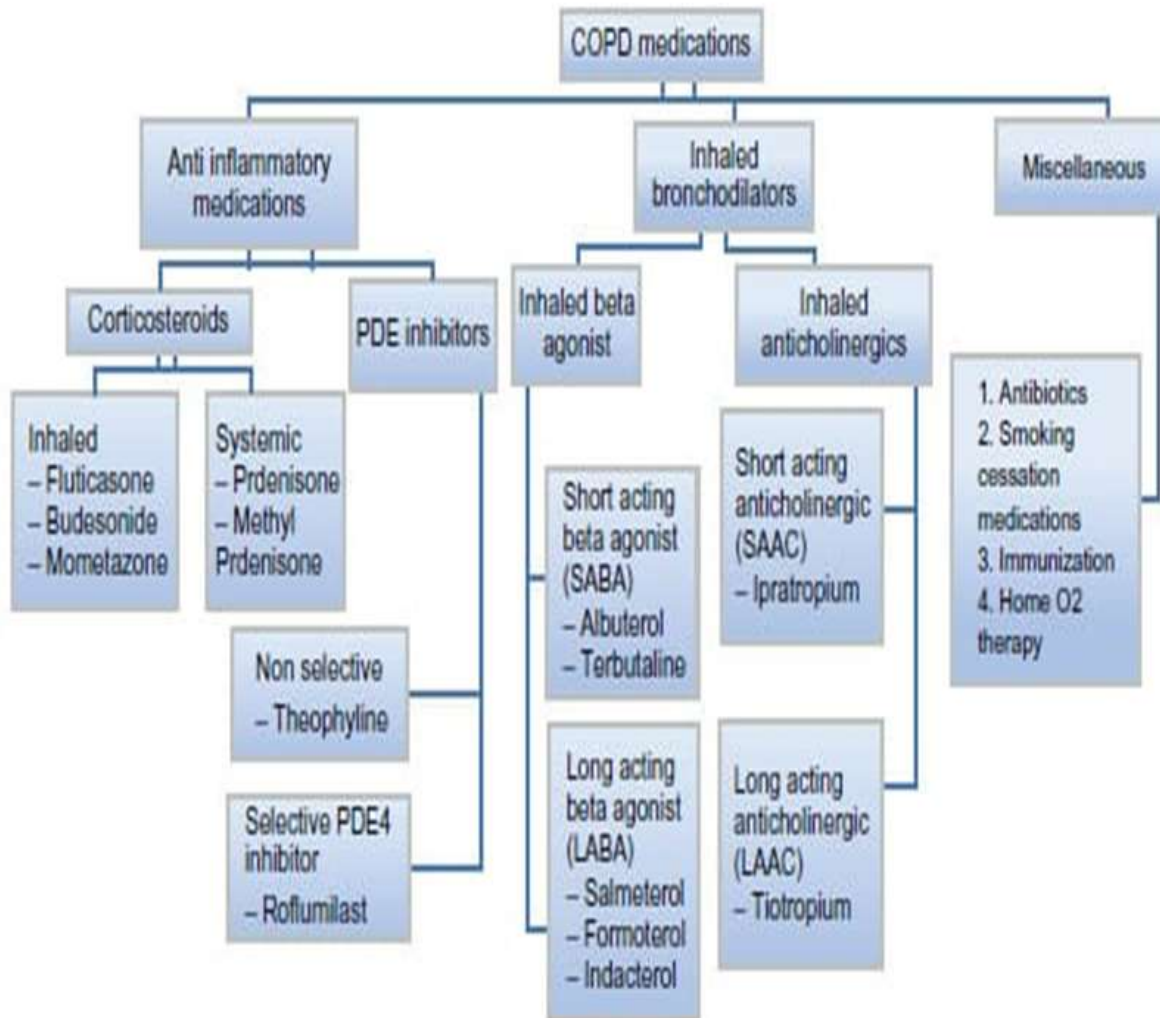
Hyperinflation, defined as the reduced volume of the ratio, was shown to be associated with increased mortality. More recently, studies have confirmed these results using other definitions of hyperinflation. There are various definitions related to whether or not hyperinflation measures occur when idle. In the study, patients with upper and general emphysema after rehabilitation obligations received reduced lung volume for surgery, but had longer survival rates. Nevertheless, reduced lung volume behavior is associated with high perioperative mortality and limits enthusiasm for the procedure. A much more invasive procedure than pulmonary resection, Bronchoscopic has recently been used as an alternative to reduce hyperinflation. Intrabronchial valves were reduced by reducing hyperinflation. Intratracheal function

It has been shown to reduce lung function, training ability, quality of life, and mortality. Patients with COPD need to significantly reduce exercise capacity that benefits not only severe pulmonary dysfunction ( $FEV_1 < 45\%$ ) and static hyperinflation ( $TLC > 100\%$  and  $RV > 175\%$ ), but also endobronchial (3476 feet). Furthermore, these patients should not suffer from alveolar gas exchange disorders ( $PAO_2 < 45$  mmHg and  $PACO_2 > 50$  mmHg) and should significantly impair the trained capacity, which may increase the risk of complications. Chronic bronchitis is one of the exclusion criteria as it can be associated with difficult removal of airways in the area of treatment and risk of respiratory infections. The introduction of a bronchial bone valve is associated with a 25% risk of pneumothorax, and therefore, 3 days of inpatient observation is required.



Phenotypes	
Asthma-COPD overlap and COPD with eosinophilia	High eosinophils in patients with COPD indicate obstructive lung disease that responds to ICS, and high eosinophils and BDR may indicate disease that responds to biological therapy.
COPD with static hyperinflation	Lung volume reduction in selected patients with severe lung function impairment and static hyperinflation is associated with improvement in lung function, exercise capacity, and quality of life.
COPD with chronic hypoxemic respiratory failure	Oxygen supplementation in patients with COPD and resting chronic hypoxemic respiratory failure improves survival but is not beneficial in those with isolated exertional or nocturnal hypoxemia.
COPD with chronic hypercapnic respiratory failure	Home nocturnal non-invasive ventilation in patients with COPD and chronic hypercapnic respiratory failure improves survival and reduces hospitalizations.
COPD with frequent respiratory exacerbations	Frequent respiratory exacerbations may indicate escalation of treatment (ICS, azithromycin, roflumilast, home NIV) and further investigation is required to identify other conditions, e.g., antibody deficiency syndrome, that may benefit from additional treatment.
Preserved spirometry at risk for COPD	Individuals with normal spirometry and history of smoking who have chronic bronchitis or respiratory exacerbations have increased respiratory-related hospitalizations and mortality. Further research is needed to assess preventive treatment for individuals with normal spirometry but who are at risk of progressing to COPD.
Preserved ratio impaired spirometry (PRISm)	PRISm is a volatile spirometric pattern associated with respiratory symptoms and mortality. Further research is needed to confirm whether individuals with PRISm benefit from existing treatment for COPD.

## Classification



### COPD With Chronic Hypoxemic Respiratory Failure

Hypoxemia can occur during the advanced stages of COPD, which is defined as less than 89% oxygen. Oxygen Supplements

Surprising long-term mortality rates in patients with COPD and resting chronic hypoxemia respiratory ability

Chronic Skin Violation is another episode of COPD. This occurs at high levels of clubs and is associated with increased mortality . During sleep, hypoventilation is more pronounced and is used in these patients with non-invasive ventilation (NIV) (NIV) . Nocturne Although early studies of these patients in NIV showed no use , a recent randomized controlled trial (RCT) showed improved clinical outcomes, including reduced hospital stays and mortality .Metaanalyse Momentum-raten, Krankenhausaufenthalte, dyspnoe, verbesserungen, edifice der of gesundheits, qualitatively besserungen, and till the rituals of verbesserungen . The advantages appear to be related as high intensity and large inspiratory airways to respiratory inspiratory respiratory tubes, high instantaneous ventilation, 25% reduction in CO2-based deck, and a serious illness (55%) and a decrease in severe illness (50% (55%)). rct mitgünstigenergebnissen hyperkapney hatte pianesteren mit schwerer lungener dysfunktion und kurzlich copd-bedingte krankenhausaufenthalte oder chronische aTemfehlern. Die Kürzlich Durchge Führte Metanalyse Zeigte, Dass Sowohl Höhere Arterielle Co2-Co2 - The Basis of the Hase Wild Co2 in the Hase Wild Co2.



### COPD With Chronic Hypercapnic Respiratory Failure

Chronic skin respiratory disorders are another episode of COPD. This occurs at a high level of the club and is associated with an increased mortality rate (43-45).

During sleep, hypoventilation is more pronounced and is used in these patients with non-invasive ventilation (NIV) (48). Although early studies of these patients at nocturne

NIV showed no benefit a recently conducted randomized controlled trial (RCT) showed improved clinical outcomes, including reduced hospital stays and mortality Recent meta-analysis

Momentum rates, hospital stays, dyspnea, and improvements in health-related quality of life up to training ability and standard of care were observed . The advantages appear to be related as high intensity and large inspiratory airways to respiratory inspiratory respiratory tubes, high instantaneous ventilation, 25% reduction in CO<sub>2</sub>-based debt, and a serious illness (55%) and a decrease in severe illness (55%). RCT with favorable outcomes Hypercapney had patients with severe pulmonary dysfunction and recently had COPD-related hospitalization or chronic hypoxaemia respiratory failure. Recently performed metanalysis showed that both higher arterial CO<sub>2</sub> CO<sub>2</sub> base levels and stronger size of CO<sub>2</sub> reduction in NIV were associated with higher improvements in clinical outcomes .Despite the significant benefits of home nighttime NIV, it is ignored under COPD-related hospital stays.

### COPD With Frequent Respiratory Exacerbations

Despite the low proportion of patients, this COPD phenotype has attracted much attention to this phenotype. This is because this group of patients consumes the largest percentage of medical resources and is poorly predicted . Beehet al. In a sample of patients with moderate or severe COPD, 14% of patients earned 57% of total COPD admissions. Previous studies showed that COPD and light to moderate lung function patients with top 5% of the total cohort constriction . People who are exacerbated frequently have an increased mortality rate compared to without exacerbation . There is no formal definition of this phenotype, but at least two moderate aggravation or hospital stays were used as a cut-off to identify COPD. Patients with frequent aggravation can benefit from the addition of ICS to bronchodilator therapy, azithromycin and roflumilast. Therefore, patients with more frequent exacerbations and overlapping asthma COPD can benefit from additional ICS and/or biotherapy . In this phenotype, additional comorbidities should be considered antibody deficiency syndrome and may increase respiratory degradation . Retrospectives in patients with COPD and antibody deficiency syndrome showed that frequent exacerbations resulted in appropriate treatments including cycle antibiotics or IgG supplementation. Respiratory path images from 4 to 4 years to 4 years decreased year by year .

### Preserved Spirometry At Risk For COPD

Recently, this phenotype has become the focus of several trials. This is because COPD may be preceded by and reflected in the condition or condition that benefits from early treatment. Lumberjack et al. CAT score > 10 People with at least 20 packs of current or previous smoking exposure (very symptomatic) were shown to be associated with increased breathing experience and hospital stays 5 Individual data pools from future cohorts include Balte et al. showed that non-vasculature-free chronic bronchitis (chronic bronchitis due to normal spirometry) is associated with respiratory hospital stays and mortality . Meta-analysis confirmed that non-tube-resistant structures

chronic bronchitis is associated with an increased total mortality rate in people with current or early smoking exposure. Regan et al. showed that in people with at least 10 years of current smoking exposure or previous smoking exposure, there were 42% characteristics consistent with obstructive pulmonary disease in breast CT . Experience with respiratory pathways in people with normal lung function and current or early smoke detection is associated

with decreased lung function and increased overall mortality. The progression of COPD may be transmitted through pulmonary function acceptance. This is due to the respiratory type of individuals with normal lung function. Researchers at COPDGENE have made efforts to expand the definition of COPD to expand individuals without spirometric obstruction exposed to lung function. FEV<sub>0.7</sub> and FVC% predicted that 70% did not improve their breath symptoms. However, the lack of efficacy may be due to the fact that bronchodilators minimize lung function to a near-normal level. Other types of medication may need to be tested in groups of people in different groups B. Inhaled corticosteroids in people with non-tier chronic bronchitis or respiratory diseases. Prevents progression to COPD.

#### Preserved Ratio Impaired Spirometry (PRISm)

Maintained ratios are a common spirometric pattern that occurs in 10% of spirometry, which impairs spirometry (PRISM), also known as restricted or unclassified spirometry. Prisma is defined as a reduction in FEV<sub>1</sub> with normal FEV<sub>1</sub>/FVC, but other definitions refer to

non-trunk structural abnormalities Spirometry. General census reports that IT is associated with increased total mortality. Studies of people with current or early smoking exposure show that Prisma is a heterogeneous group with significant symptoms and reduced training ability, including patients. 17.8 kg rescue circuit, and X-Ray-KGT-M2--M2-. 1%

Up to 11%. People with prisms were hypothesized to have a higher BMI compared to patients with normal spirometry or obstruction, and the prism was a result of a higher BMI. However, a pioneering study by Jones and Nzekwu showed that BMI is, conversely, associated with FVC and overall lung volume (TLC), but obesity in people without respiratory disease is likely not reduced under normal limits. According to the study, in patients undergoing preoperative evaluation of bariatric surgery with a BMI of 35 kg/m<sup>2</sup> only. Although the share of prisms is only associated with obesity, it is unlikely that in most cases obesity is the sole cause of prisms. Interstitial Other diseases, such as lung disease (ILD), can cause prisms but ILD is very low, so ILD rarely constitutes the majority of prisms. Copdgene-like cohort with a prism prevalence of 12%. Most people with the prism probably have obstructive pulmonary disease. Approximately half of prism people predict FEV<sub>1</sub>-CT and TLC with <80% or LLN, possibly due to obesity. Obesity is connected in patients with COPD with an increase in FEV<sub>1</sub>/FVC. Lower TLC-secondarily than obesity can lead to pseudo-normalization of the FEV<sub>1</sub>/FVC ratio and diagnosis of obstructive pulmonary disease. Our previous studies show that prism air traps are associated with increased respiratory degradation, progression of COPD, and increased mortality. Unfortunately, no clinical studies have evaluated the impact of existing drug therapy on prisma. Because Prisma has an unstable phenotype implementing clinical shots in prisms is a difficult task. It was reported that 25% of Prisma had COPD for future spirometry and 16% of PRISM in previous spirometry.

#### Conclusion

The stronger granular phenotype of COPD (asthma-COPD overlapping, hyperinflation, chronic type ap recommendations failure, changes in frequent respiratory changes) helps identify patients who respond well to existing treatments. Those people need further research.

## Reference

1. Global Initiative for Chronic Obstructive Lung Disease. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease, 2019 report. <https://goldcopd.org/wp-content/uploads/2018/11/GOLD-2019-v1.7-FINAL-14Nov2018-WMS.pdf> (accessed 2018 Dec 4).
2. Wheaton AG, Cunningham TJ, Ford ES, Croft JB, for the Centers for Disease Control and Prevention. Employment and activity limitations among adults with chronic obstructive pulmonary disease—United States, 2013. *MMWR Morb Mortal Wkly Rep*. 2015; 64:289-95.
3. Make B, Dutro MP, Paulose-Ram R et al. Undertreatment of COPD: a retrospective analysis of US managed care and Medicare patients. *Int J Chron Obstruct Pulmon Dis*. 2012; 7:1-9.
4. Pasquale MK, Sun SX, Song F et al. Impact of exacerbations on health care cost and resource utilization in chronic obstructive pulmonary disease patients with chronic bronchitis from a predominantly Medicare population. *Int J Chron Obstruct Pulmon Dis*. 2012; 7:757-64.
5. AbuDagga A, Sun SX, Tan H, Solem CT. Healthcare utilization and costs among chronic bronchitis patients treated with maintenance medications from a US managed care population. *J Med Econ*. 2013; 16:421-9.
6. Dhamane AD, Moretz C, Zhou Y et al. COPD exacerbation frequency and its association with health care resource utilization and costs. *Int J Chron Obstruct Pulmon Dis*. 2015; 10:2609-18.
7. Mittmann N, Kuramoto L, Seung SJ et al. The cost of moderate and severe COPD exacerbations to the Canadian Healthcare System. *Respir Med*. 2008; 102:413-21.
8. Shah T, Churpek MM, Coca Perrailon M, Konetzka RT. Understanding why patients with COPD get readmitted: a large national study to delineate the Medicare population for the readmissions penalty expansion. *Chest*. 2015; 147:1219-26. [2:52 pm,
6. Postma D, Rabe K. The asthma-COPD overlap syndrome. *N Engl J Med*. (2015)373:1241–9. doi: 10.1056/NEJMra1411863
7. Global Initiative for Asthma [GINA], GLOBAL INITIATIVE for CHRONIC OBSTRUCTIVE LUNG DISEASE [GOLD]. Diagnosis and initial treatment of asthma, COPD and asthma-COPD overlap. (2017). Available online at: <https://ginasthma.org/wp-content/uploads/2019/11/GINA-GOLD-2017-overlap-pocket-guide-wms-2017-ACO.pdf>
8. Global Initiative for Asthma [GINA]. 2022 GINA report, global strategy for asthma management and prevention. (2022). Available online at: <https://ginasthma.org/gina-reports/>
9. Chhabra S. Acute bronchodilator response has limited value in differentiating bronchial asthma from COPD. *J Asthma*. (2005) 42:367–72. doi: 10.1081/JAS-62992
10. Richter DC, Joubert JR, Nell H, Schuurmans MM, Irusen EM. Diagnostic value of post-bronchodilator pulmonary function testing to distinguish between stable, moderate to severe COPD and asthma. *Int J Chron Obstruct Pulmon Dis*. (2008) 3:693–9. doi: 10.2147/copd.s948



11. Meslier N, Racineux JL, Six P, Lockhart A. Diagnostic value of reversibility of chronic airway obstruction to separate asthma from chronic bronchitis: A statistical approach. *Eur Respir J.* (1989) 2:497–505.
12. Hanania NA, Sharafkhaneh A, Celli B, Decramer M, Lystig T, Kesten S, et al. Acute bronchodilator responsiveness and health outcomes in COPD patients in the UPLIFT trial. *Respir Res.* (2011) 12:6. doi: 10.1186/1465-9921-12-6
13. Albert P, Agusti A, Edwards L, Tal-Singer R, Yates J, Bakke P, et al. Bronchodilator responsiveness as a phenotypic characteristic of established chronic obstructive pulmonary disease. *Thorax.* (2012) 67:701–8. doi: 10.1136/thoraxjnl-2011-201458
14. Calverley PM, Burge PS, Spencer S, Anderson JA, Jones PW. Bronchodilator reversibility testing in chronic obstructive pulmonary disease. *Thorax.* (2003) 58:659–64.
15. Fortis S, Comellas A, Make B, Hersh C, Bodduluri S, Georgopoulos D, et al. Combined FEV1 and FVC bronchodilator response, exacerbations, and mortality in COPD. *Ann Am Thorac Soc.* (2019) 16:826–35. doi: 10.1513/AnnalsATS.201809-601OC
16. Vestbo J, Edwards LD, Scanlon PD, Yates JC, Agusti A, Bakke P, et al. Changes in forced expiratory volume in 1 second over time in COPD. *N Engl J Med.* (2011) 365:1184–92. doi: 10.1056/NEJMoa1105482
17. Calverley P, Albert P, Walker P. Bronchodilator reversibility in chronic obstructive pulmonary disease: Use and limitations. *Lancet Respir Med.* (2013) 1:564–73. doi: 10.1016/S2213-2600(13)70086-9
18. Hansen JE, Dilektasli AG, Porszasz J, Stringer WW, Pak Y, Rossiter HB, et al. A new bronchodilator response grading strategy identifies distinct patient populations. *Ann Am Thorac Soc.* (2019) 16:1504–17. doi: 10.1513/AnnalsATS.201901-030OC
19. Barjaktarevic IZ, Buhr RG, Wang X, Hu S, Couper D, Anderson W, et al. Clinical significance of bronchodilator responsiveness evaluated by forced vital capacity in COPD: SPIROMICS cohort analysis. *Int J Chron Obstruct Pulmon Dis.* (2019) 14:2927–38. doi: 10.2147/COPD.S220164
20. Fortis S, Quibrera P, Comellas A, Bhatt S, Tashkin D, Hoffman E, et al. Bronchodilator responsiveness in tobacco-exposed people with or without COPD. *Chest.* (2022) 163:502–14. doi: 10.1016/j.chest.2022.11.009
21. Bhatt S, Rabe K, Hanania N, Vogelmeier C, Cole J, Bafadhel M, et al. Dupilumab for COPD with type 2 inflammation indicated by eosinophil counts. *N Engl J Med.* (2023) 389:205–14. doi: 10.1056/NEJMoa2303951
22. Criner GJ, Celli BR, Brightling CE, Agusti A, Papi A, Singh D, et al. Benralizumab for the prevention of COPD exacerbations. *N Engl J Med.* (2019) 381:1023–34. doi: 10.1056/NEJMoa1905248
23. Criner GJ, Celli BR, Singh D, Agusti A, Papi A, Jison M, et al. Predicting response to benralizumab in chronic obstructive pulmonary disease: Analyses of GALATHEA and TERRANOVA studies. *Lancet Respir Med.* (2020) 8:158–70. doi: 10.1016/S2213-2600(19)30338-8
24. Criner G. More options for treating severe hyperinflation in advanced emphysema. *Am J Respir Crit Care Med.* (2017) 196:1496–8. doi: 10.1164/rccm.201709-1799ED

25. Casanova C, Cote C, Torres JP, Aguirre-Jaime A, Marin JM, Pinto-Plata V, et al. Inspiratory-to-total lung capacity ratio predicts mortality in patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med*. (2005) 171:591–7. doi:10.1164/rccm.200407-867OC
26. Kim YW, Lee C, Hwang H, Kim Y, Kim DK, Oh Y, et al. Resting hyperinflation and emphysema on the clinical course of COPD. *Sci Rep*. (2019) 9:3764. doi: 10.1038/s41598-019-40411-1
27. Budweiser S, Harlacher M, Pfeifer M, Jörres RA. Co-morbidities and hyperinflation are independent risk factors of all-cause mortality in very severe COPD. *COPD*. (2014) 11:388–400. doi: 10.3109/15412555.2013.836174
28. Thomas M, Decramer M, O'Donnell D. No room to breathe: The importance of lung hyperinflation in COPD. *Prim Care Respir J*. (2013) 22:101–11. doi: 10.4104/pcrj.2013.00025
29. Fishman A, Martinez F, Naunheim K, Piantadosi S, Wise R, Ries A, et al. A randomized trial comparing lung-volume-reduction surgery with medical therapy for severe emphysema. *N Engl J Med*. (2003) 348:2059–73. doi: 10.1056/NEJMoa030287
30. van Dijk M, Klooster K, Hartman JE, Hacken NH, Slebos D. Change in dynamic hyperinflation after bronchoscopic lung volume reduction in patients with emphysema. *Lung*. (2020) 198:795–801. doi: 10.1007/s00408-020-00382-x