CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) : AN UPDATE

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Director Medical Education & prof , Pulmonary Medicine

Era's Lucknow Medical College & Hospital, Lucknow

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Former Director, V.P. Chest Institute, Delhi

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Former Prof. & Head Pulmonary Medicine, KGMU, Lucknow

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Senior Vice Chairman, Uttar Pradesh Tuberculosis Association

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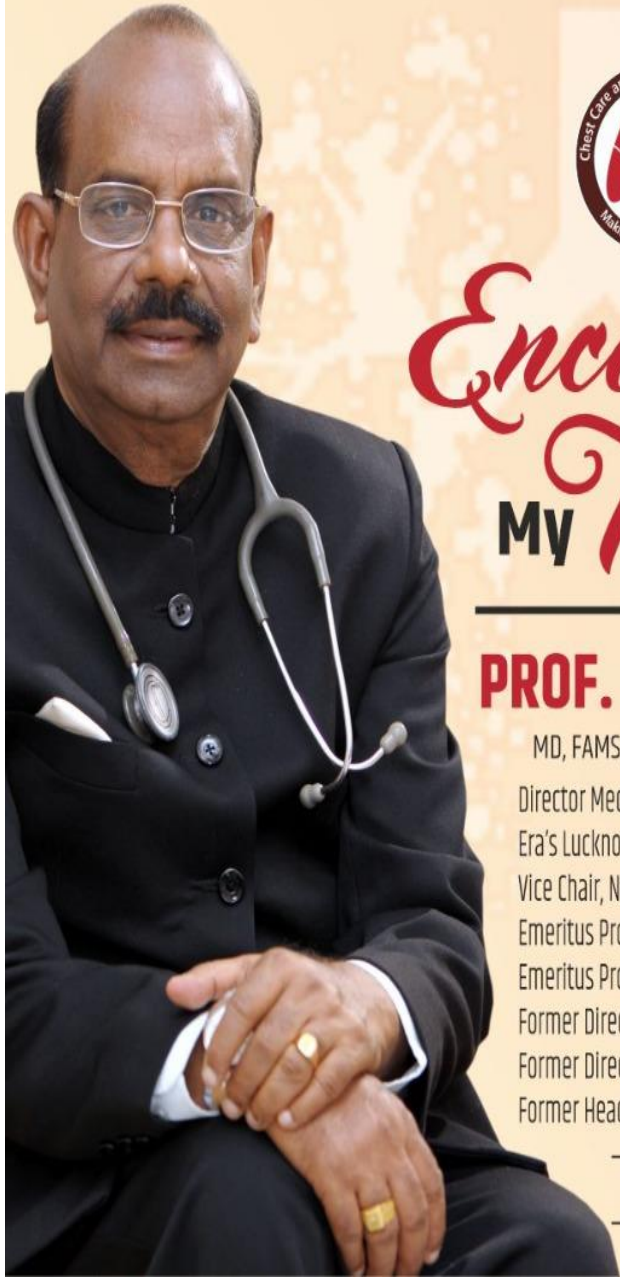
“Better than a thousand days of diligent – study is one day with a great teacher”

Japanese Proverb



Chest Care and Research Society (CRS)
Making Lung & Life Better

HELP

Encounter with My Teachers

PROF. RAJENDRA PRASAD

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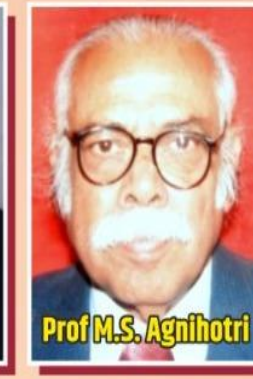
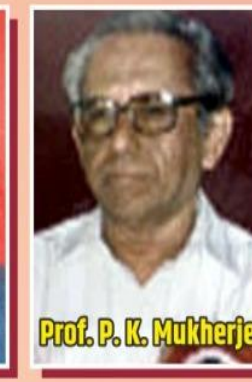
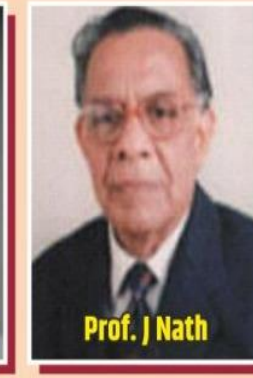
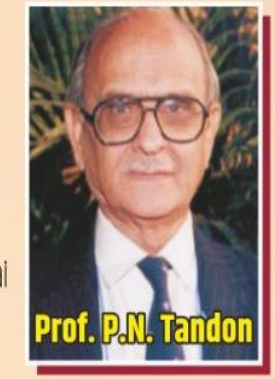
Emeritus Professor, National Academy of Medical Sciences (India)
Emeritus Professor, Indian Medical Association
Former Director: Vallabhbhai Patel Chest Institute, Delhi
Former Director: U.P. Rural Institute Of Medical Science & Research, Saifai
Former Head: Department of Pulmonary Medicine, KGMU, Lucknow

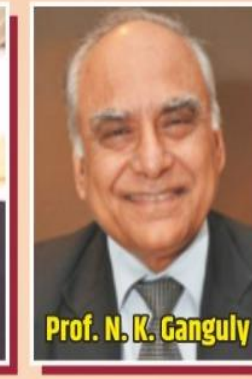
Conduct by **Vandana Tribhuwan Singh**











& Many More who Contributed in my Academics & Career

K.G'S MEDICAL COLLEGE LUCKNOW

King George's Medical University, Lucknow

U.P. Rural Institute of Medical Sciences & Research, Saifai

Administrative Block

Academic Block

Hospital Block

Vallabhbhai Patel Chest Institute, Delhi

ERA'S LUCKNOW MEDICAL COLLEGE & HOSPITAL

National Academy of Medical Sciences (India)

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NAMS (India)

- Established on 21st April, 1961 as a registered Society namely, the ***'Indian Academy of Medical Sciences'*** under Societies Registration Act XXI of 1860, with the objective of promoting the growth of medical sciences
- Inaugurated at New Delhi on 19th December, 1961 by Pt. Jawaharlal Nehru, the first Prime Minister of India
- First Convocation of the Academy held on 8th December, 1963 was addressed by Dr. S. Radhakrishnan, the then President of India
- The Academy was re-named National Academy of Medical Sciences (India) on 16th November, 1976 on the Working Group set up by Government of India
- NAMS is one of the unique institution which fosters and utilises academic excellence as its resource to meet the medical and social goals

NAMS Family

1. **Fellows (FAMS)**
2. **Members (MAMS)**
3. **Associate Fellow (Associate Fellow <45 years)**
4. **Members (After DNB) (MNAMS)**
5. **Associate Member (After MD/MS)**
6. **Emeritus Professor**

NAMS Associate Membership

NAMS Associate Membership has been introduced for the Medical /Dental Scientists who have completed Postgraduate qualifications (MD/ MS/ MDS) or PhD/MSc Biotechnology in SINGLE ATTEMPT and have a postgraduate degree with Any ONE of the following:

a. Membership of a National Professional organization in his/her specialty
b. Publication in a Scientific Journal
c. Presentation at the Annual Scientific Conference of National Professional organization

The application form, must be proposed by any of the following:

Head of Institution
Head of Department
Head of Unit
NAMS Fellow

Applications are accepted throughout the year.

The link for online application is as follows; https://namsdigital.in/Home/AMAMSHome

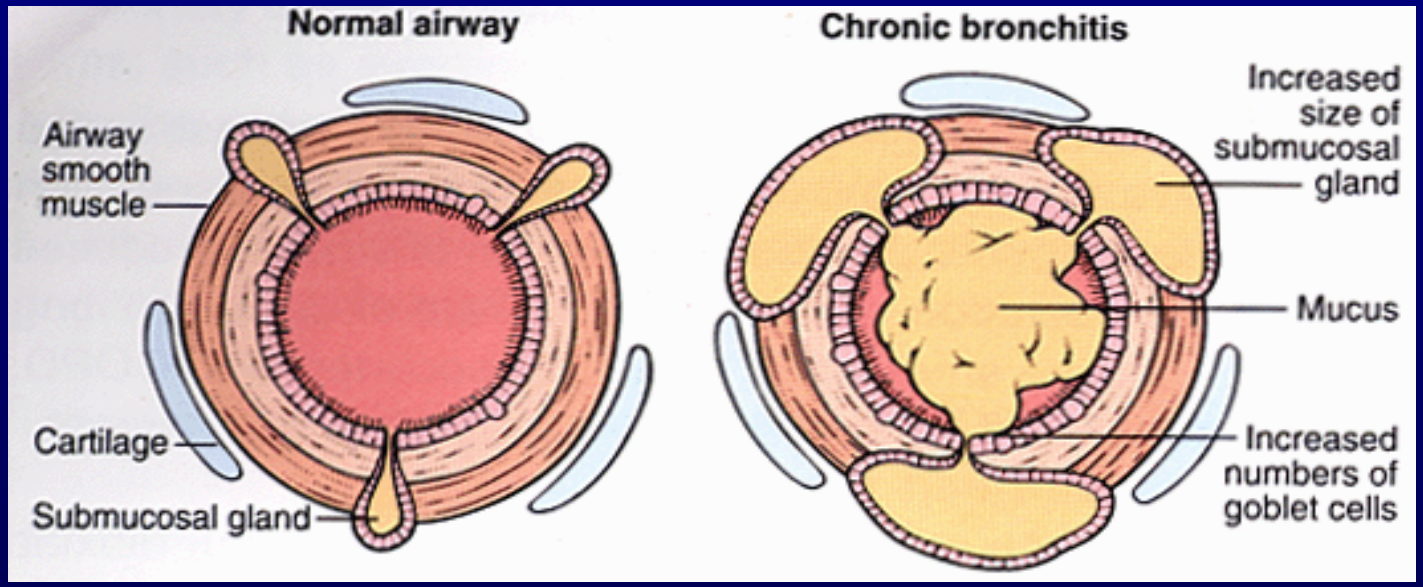
COPD

Obstructive airway disease, emphysema and chronic bronchitis as separate disease entities were first defined in Ciba Guest symposium in 1958

Ciba Foundation Guest Symposium. Thorax 1959;14;286-99

Chronic Bronchitis

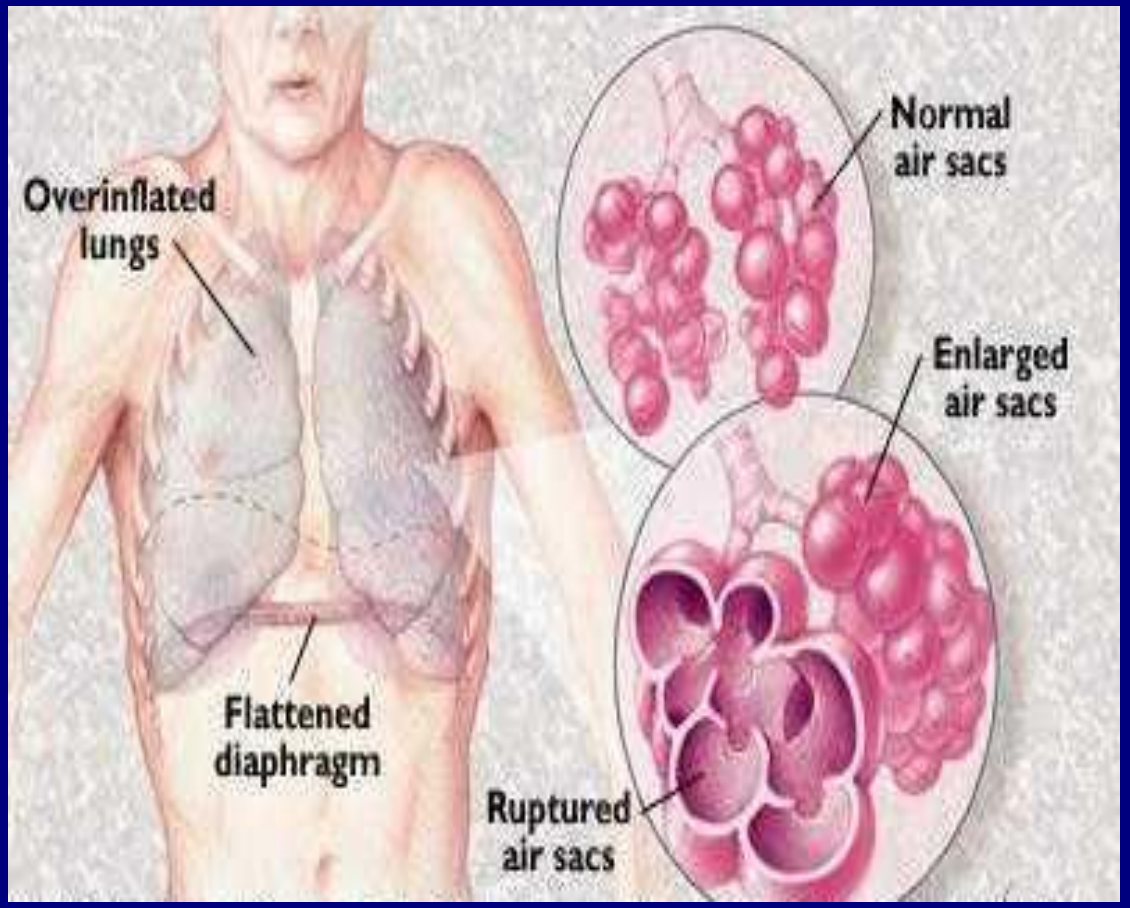
Chronic Cough with excessive mucus secretion for most days out of 3 months in each of two or more successive years without other specific cause of cough (asthma, bronchiectasis, tub. etc.)



Emphysema

R Prasad, VPCI

Abnormal enlargement of the air spaces distal to the terminal bronchioles accompanied by destruction of their walls, and without obvious fibrosis.



Definition of COPD

Chronic obstructive pulmonary disease (COPD) is a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases.

GOLD - 2005

COPD

Chronic Obstructive Pulmonary Disease (COPD) is a common, preventable and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases and influenced by host factors including abnormal lung development. Significant comorbidities may have an impact on morbidity and mortality

GOLD 2022

Definition

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

GOLD GUIDELINES 2025

COPD EPIDEMIOLOGY

R Prasad, VPCI

- Underestimated
- Not diagnosed until clinically overt
- By that time it is moderately advanced
- Up to 60-85% of people with COPD (mostly mild/moderate severity) are undiagnosed

COPD EPIDEMIOLOGY

- According to BOLD the estimated number of COPD cases was 391.9 million in 2019 with a global prevalence of 10.3%
- Prevalence will increase by 23% from2020 to2050 approaching 600 million
- Globally 3 million deaths annually
- It is expected to rise over next 40 years and by 2060 there may be over 5.4 million deaths annually

Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2025

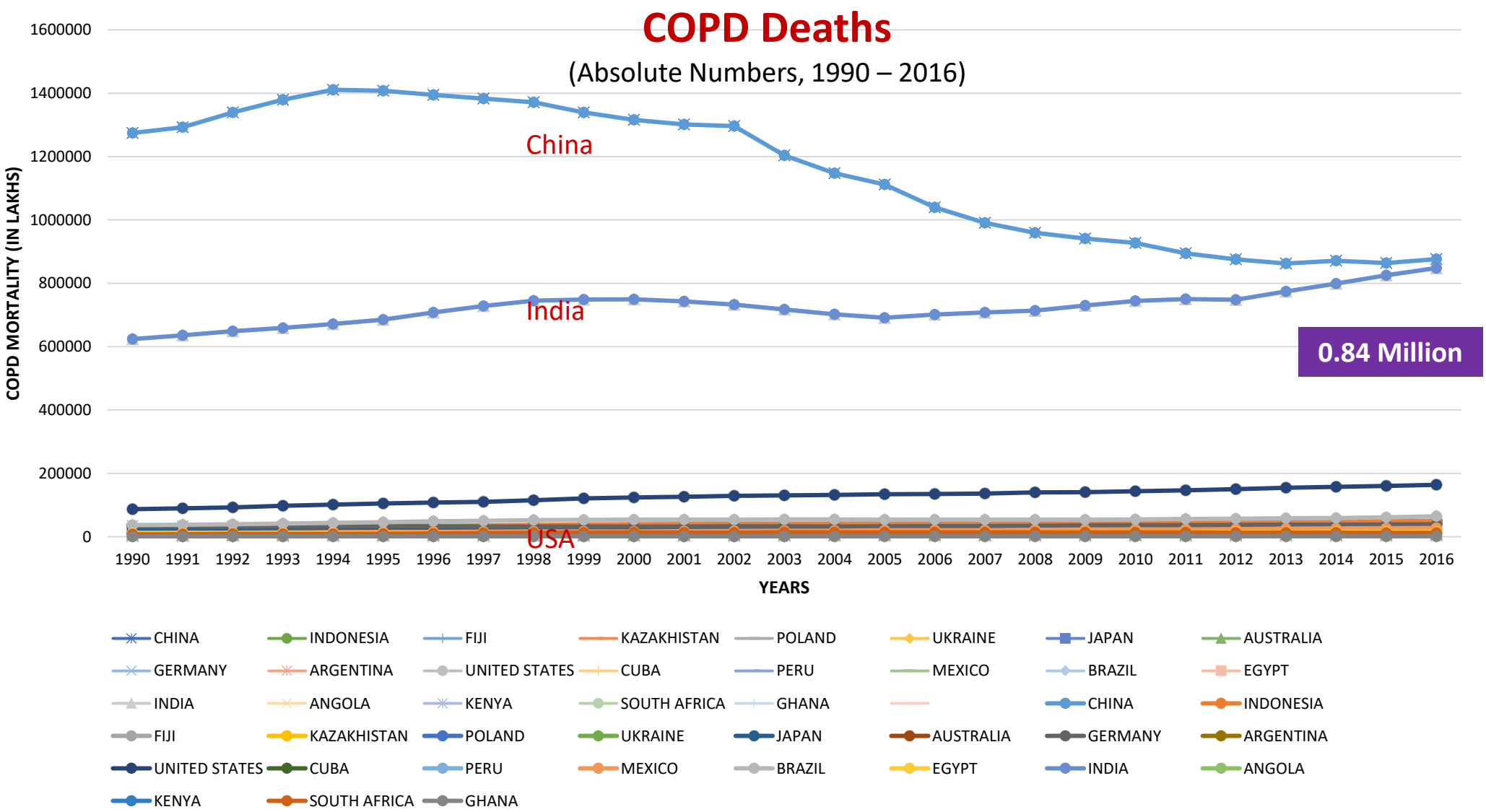
COPD - INDIA

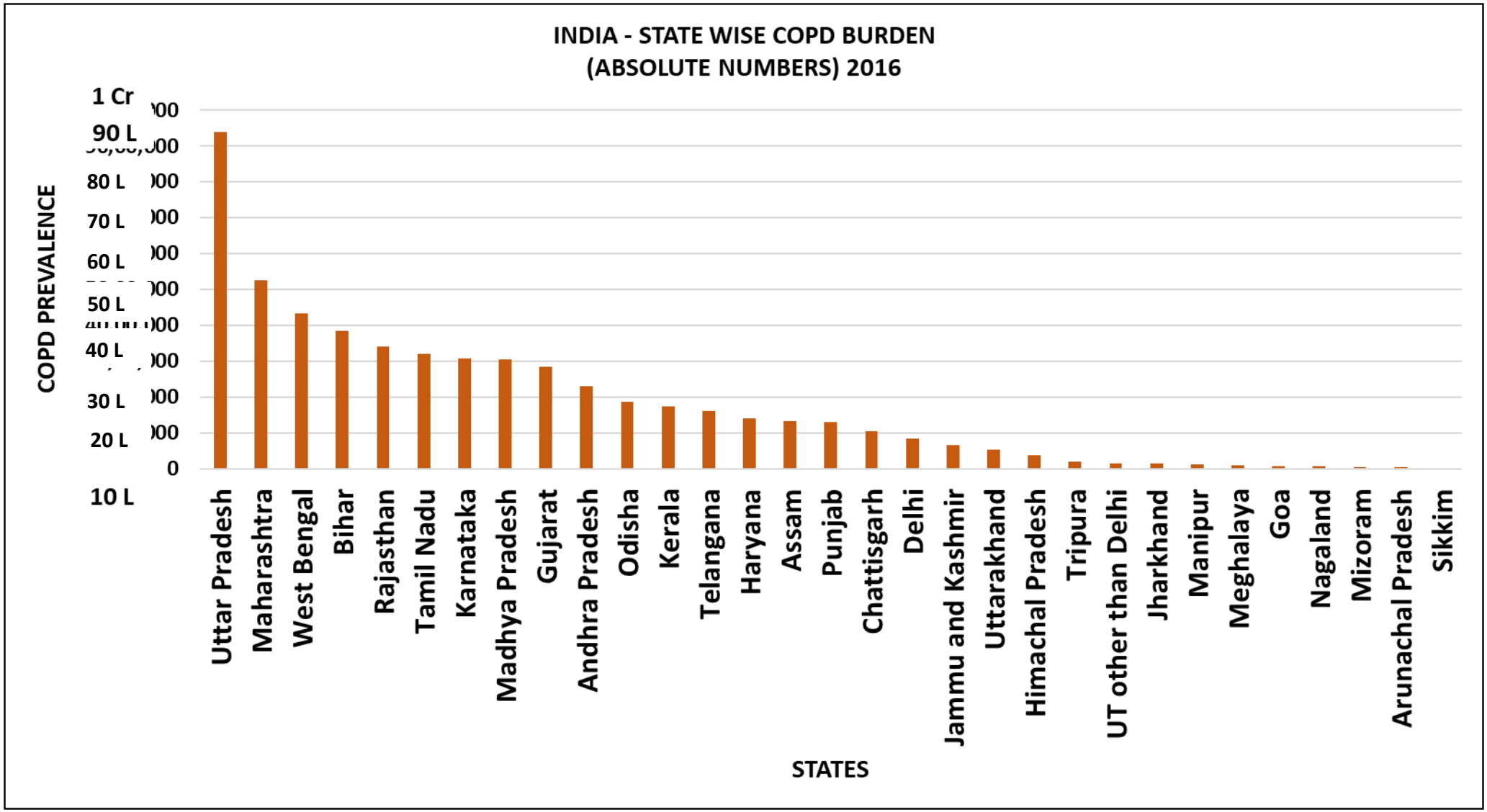
Prevalence varied from 2-22% in men and 1.2-19% in women

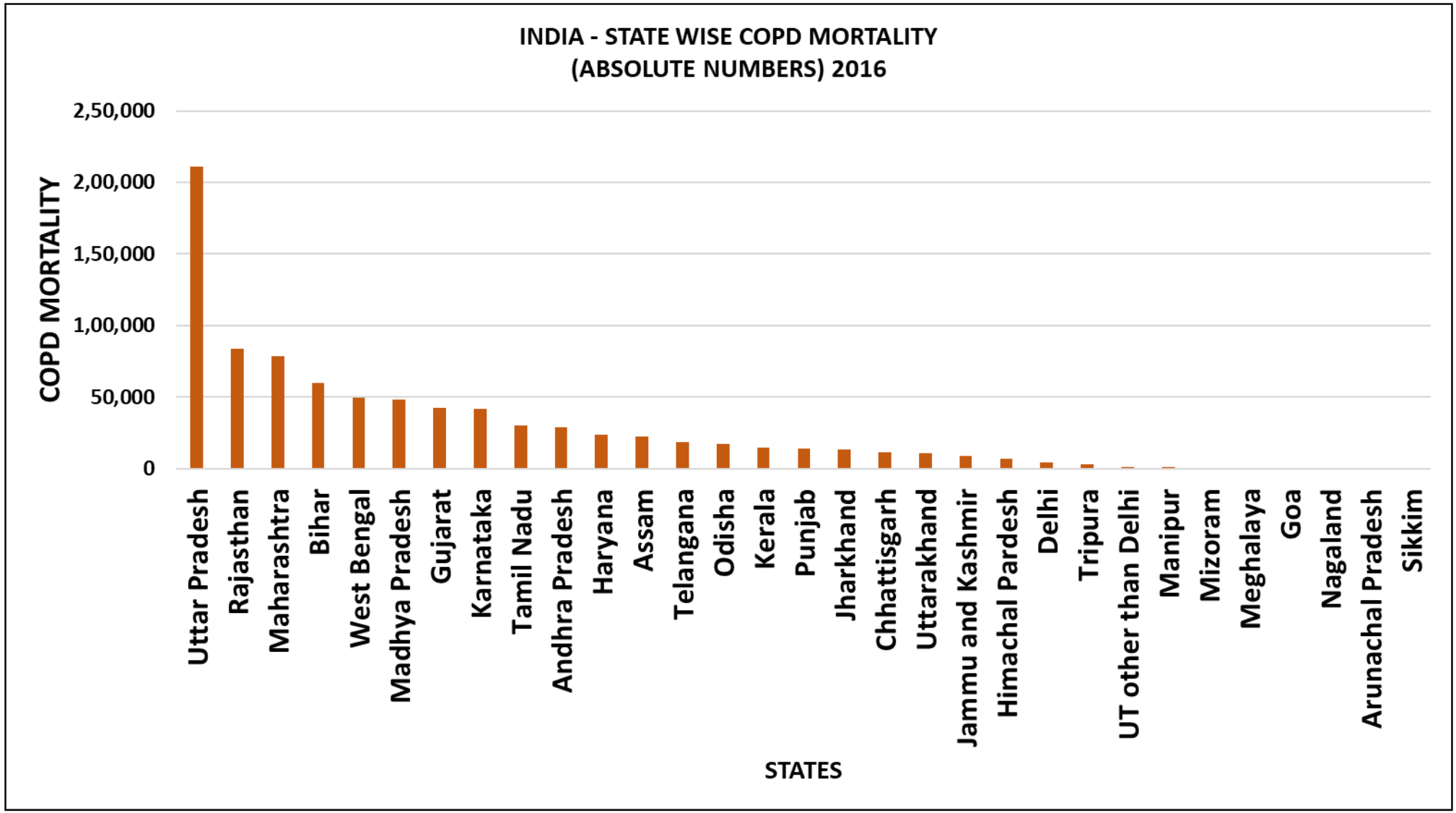
COPD EPIDEMIOLOGY: INDIA

- It is estimated that 55 million people have COPD and second leading cause of death in India in 2017
- At least four sites across India took part in the burden of obstructive lung disease study, an international study using a robust design and standardized and validated questionnaires along with a postbronchodilator spirometry test
- Over the last decade, others also used this methodology to study the burden and risk factors for COPD in India

Salvi S et al. What is the true burden of chronic obstructive pulmonary disease in India and what are its implications at a national level? Lung India 2021 ;38; 503-5.


COPD Deaths
(Absolute Numbers, 1990 – 2016)
COPD MORTALITY (IN LAKHS)
1600000
1400000
1200000
1000000
800000
600000
400000
200000
0
China
India
USA
0.84 Million
1990 1991 1992 1993 1994 1995 1996 1997 1998 1999 2000 2001 2002 2003 2004 2005 2006 2007 2008 2009 2010 2011 2012 2013 2014 2015 2016
YEARS
CHINA
INDONESIA
FIJI
KAZAKHISTAN
POLAND
UKRAINE
JAPAN
AUSTRALIA
GERMANY
ARGENTINA
UNITED STATES
CUBA
PERU
MEXICO
BRAZIL
EGYPT
INDIA
ANGOLA
KENYA
SOUTH AFRICA
GHANA
CHINA
INDONESIA
FIJI
KAZAKHISTAN
POLAND
UKRAINE
JAPAN
AUSTRALIA
GERMANY
ARGENTINA
UNITED STATES
CUBA
PERU
MEXICO
BRAZIL
EGYPT
INDIA
ANGOLA
KENYA
SOUTH AFRICA
GHANA


INDIA - STATE WISE COPD BURDEN
(ABSOLUTE NUMBERS) 2016
COPD PREVALENCE
1 Cr
90 L
80 L
70 L
60 L
50 L
40 L
30 L
20 L
0
10 L
Uttar Pradesh
Maharashtra
West Bengal
Bihar
Rajasthan
Tamil Nadu
Karnataka
Madhya Pradesh
Gujarat
Andhra Pradesh
Odisha
Kerala
Telangana
Haryana
Assam
Punjab
Chattisgarh
Delhi
Jammu and Kashmir
Uttarakhand
Himachal Pradesh
Tripura
UT other than Delhi
Jharkhand
Manipur
Meghalaya
Goa
Nagaland
Mizoram
Arunachal Pradesh
Sikkim
STATES


INDIA - STATE WISE COPD MORTALITY
(ABSOLUTE NUMBERS) 2016
COPD MORTALITY
2,50,000
2,00,000
1,50,000
1,00,000
50,000
0
Uttar Pradesh
Rajasthan
Maharashtra
Bihar
West Bengal
Madhya Pradesh
Gujarat
Karnataka
Tamil Nadu
Andhra Pradesh
Haryana
Assam
Telangana
Odisha
Kerala
Punjab
Jharkhand
Chhattisgarh
Uttarakhand
Jammu and Kashmir
Himachal Pardesh
Delhi
Tripura
UT other than Delhi
Manipur
Mizoram
Meghalaya
Goa
Nagaland
Arunachal Pradesh
Sikkim
STATES

COPD EPIDEMIOLOGY

Enormous burden in terms of morbidity and mortality globally and in India

Risk Factors for COPD

Established	Probable
Tobacco smoking	Outdoor air pollution
Environmental tobacco smoke	Pulmonary tuberculosis
Exposure to biomass fuel smoke	Poorly treated asthma
Occupational exposure	Intrauterine growth retardation
Alpha-1 antitrypsin deficiency	Poor nourishment
	Repeated lower respiratory infections during childhood
	Others
	Age
	Male gender
	Low socioeconomic status

Genes

SMOKING HABITS IN INDIA

- Cigarette

- Bidi

- Chilum

- Hukkah

- Cigar
- Chutta, Reverse Chutta

Smoking and COPD

- 1 billion current smoker globally
- Different forms of tobacco smoking in India
- Adjusted Odds ratio for COPD among smokers is 4.08 as compared to non smokers
- Strong dose response relationship between tobacco use and COPD
- Bidi smoking is even more harmful

Burning of Biomass fuel and COPD

- Globally 3 billion people exposed to biomass fuel smoke, compared with 1.01 billion people who smoke tobacco
- 75% of households in India still continue to use BMF for Cooking and Heating.
- Two recent meta-analysis conclusively point that BMF combustion important risk factor for COPD

Salvi S et al. Chest 2010:138:3-6
Hu G et al. Chest 2010;138:20-31
Po JY et al. Thorax 2011; 66:232-9

Biomass Fuel

R Prasad, VPCI

Indoor air pollution by mosquito coils and aggarbatti

Indoor air pollution: Mosquito liquidators

Brief Communication

Chronic Obstructive Pulmonary Disease (COPD) in Non-Smokers: A Different Phenotype?

Rajendra Prasad[1], Huda Shamim[1], Harshita Rani[1], Rishabh Kacker[1] and Nikhil Gupta[2]

Department of Pulmonary Medicine[1], Era's Lucknow Medical College and Hospital, Era University, and Department of Medicine[2], Dr Ram Manohar Lohia Institute of Medical Sciences, Lucknow (Uttar Pradesh), India

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KEY WORDS

Chronic obstructive pulmonary disease, Asthma, Phenotype, Environment tobacco smokers, Biomass fuel.

ABBRIVATIONS USED IN THIS

Abstract

Chronic obstructive pulmonary disease (COPD) is a heterogeneous and multi-systemic disease with significantly increasing morbidity and mortality. COPD is now widely accepted to have multiple phenotypes. Tobacco smoking is very well recognised risk factor of COPD (25%–45% of patients with COPD have never smoked) and the burden of non-smoker COPD is increasing. This paper review in brief COPD in non-smokers, identification of various non-smoking risk factors that have contributed immensely in the causation of COPD and phenotypic variations which includes the existing and emerging phenotypes. It will also help us in proper diagnosis and pharmacological management of non-smoker COPD patients as their prevalence has been under-estimated.

R Prasad et al. Chronic Obstructive Pulmonary Disease (COPD) In Non-smokers: A Different Phenotype. Indian J Chest Dis Allied Sci 2021;63:203-6

COPD: RISK FACTORS

- 25–45% of patients with COPD have never smoked and burden of non-smoking COPD is therefore much higher than previously believed

R Prasad et al. Chronic Obstructive Pulmonary Disease (COPD) In Non-smokers: A Different Phenotype. Indian J Chest Dis Allied Sci 2021;63:203-6

Outdoor Air Pollution and COPD

- Respiratory symptoms were found to be more common in the higher pollution zones among 4,171 randomly selected residents
- Emergency room visits for COPD increased by 24.9% when the levels of pollutants in ambient air exceeded the acceptable limits

Pande JN et al. Indian J Chest Dis Allied Sci 2002;44:13-9
Willcox PA et al. Respir Med 1989;83:195-8

PULMONARY TUBERCULOSIS & COPD

- Association of PTB with COPD has been described
- Airflow obstruction varies from 28 to 68% treated PTB pts
- In a survey of 13,826 adults in South Africa, a history of PTB was associated with COPD with odds of 4.9 for men and 6.6 for women
- Whether this finding of obstructive functional defect in post tubercular sequel behaves as COPD, or is different, remains to be established

Limaye S et al. 2011
Silva GE et al. Chest 2004;126:59-65

ASTHMA & COPD

- Asthma Pts have 10-fold risk of chronic bronchitis and 17-fold risk of emphysema as compared to those without asthma even after adjustment for confounding factors

- Subset of pts with asthma may have COPD phenotype

Eisner MD et al. Am J Respir Crit Care Med 2010;182:693-718
Broekhuizen BD et al. Br J Gen Pract 2012;62:e632-8

PROPOSED ETIOTYPES FOR COPD

CLASSIFICATION	DESCRIPTION
GENETICALLY DETERMINED COPD (COPD-G)	Alpha 1 antitrypsin deficiency Other genetic variants with smaller effects acting in combination
COPD due to abnormal lung development(COPD-D)	Early life events, including premature birth and low birth weight
Environmental COPD Cigarette smoking COPD (COPD-C) Biomass and pollution exposure COPD (COPD-P)	 Exposure to tobacco smoke Vaping or e cigarette use Cannabis Exposure to household pollution, ambient air pollution, wildfire smoke, occupational hazards
COPD due to infections (COPD-I)	Childhood infections, tuberculosis associated COPD, WHIV-associated COPD
COPD and ASTHMA (COPD-A)	Particularly childhood asthma
COPD of unknown cause (COPD-U)	

Dharmage S, agusti A Personal communication. 2022
Stolz D et al. Towards the elimination of COPD: a Lancet commission. Lancet 2022:921-72
Gold 2025

Diagnosis of COPD

SYMPTOMS

shortness of breath

chronic cough

sputum

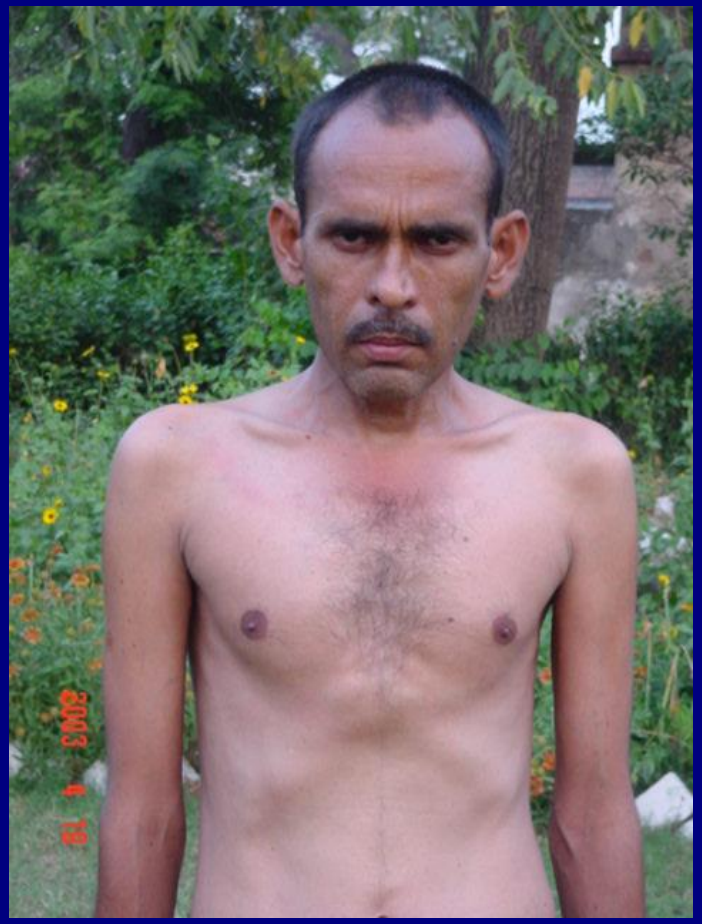
EXPOSURE TO RISK FACTORS

tobacco

occupation

indoor/outdoor pollution

SPIROMETRY: Required to establish diagnosis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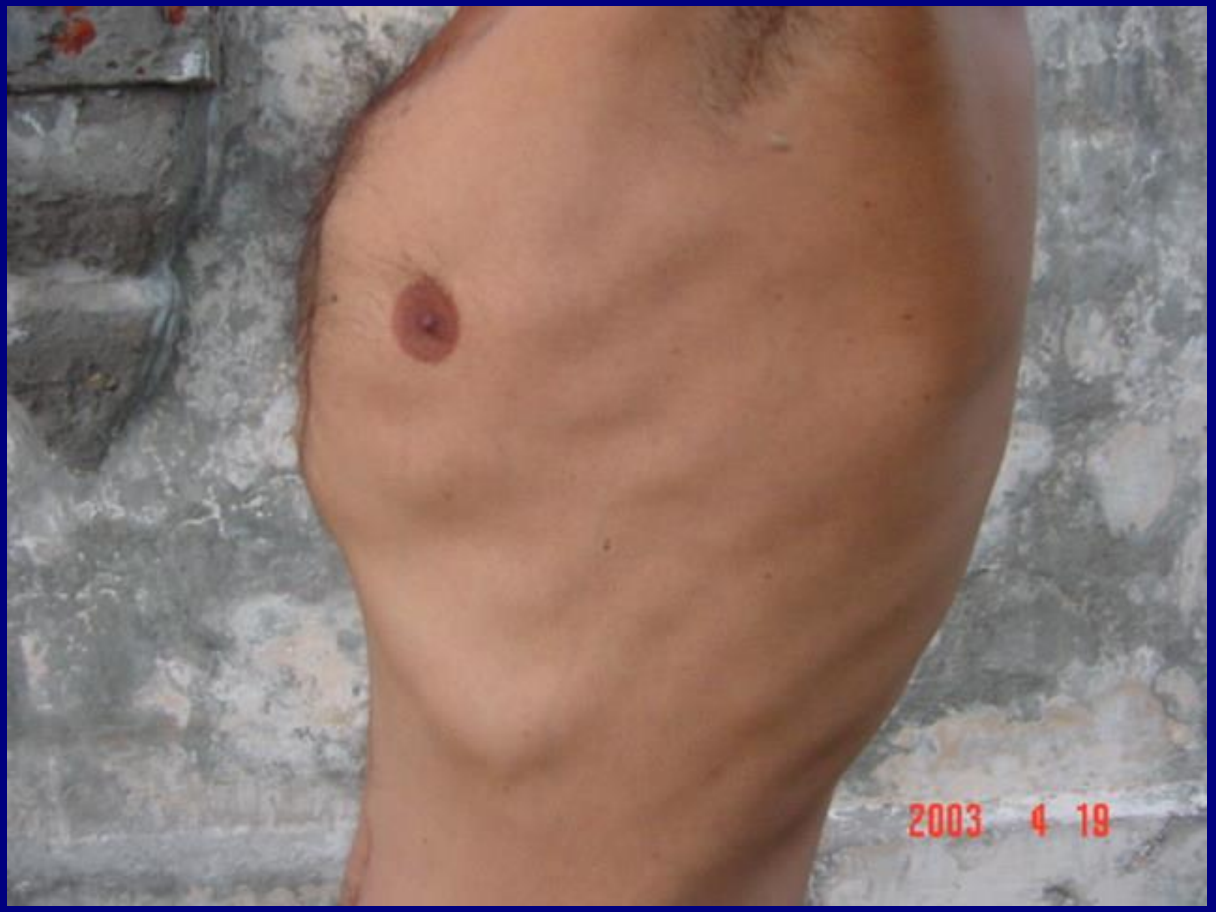
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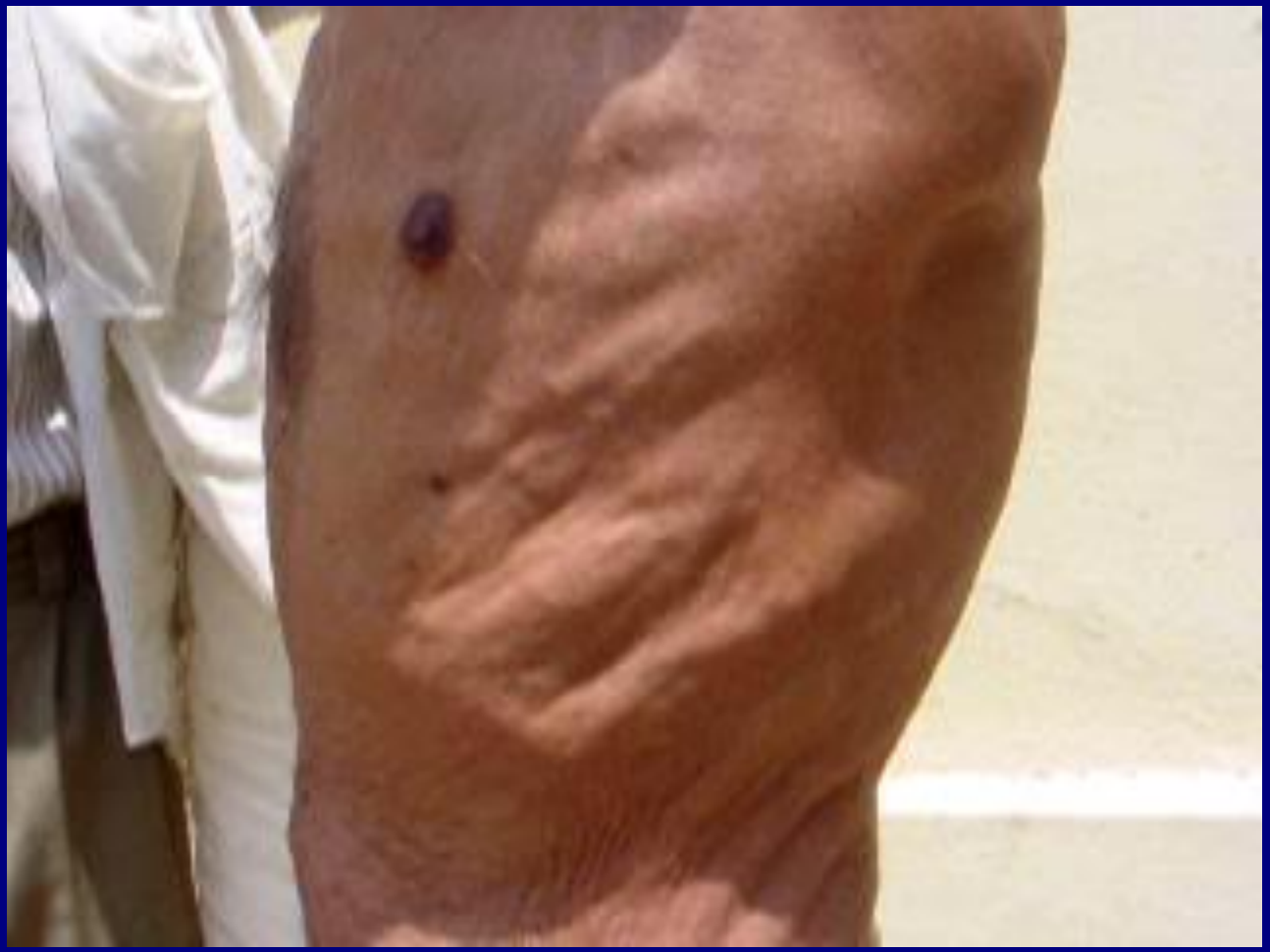

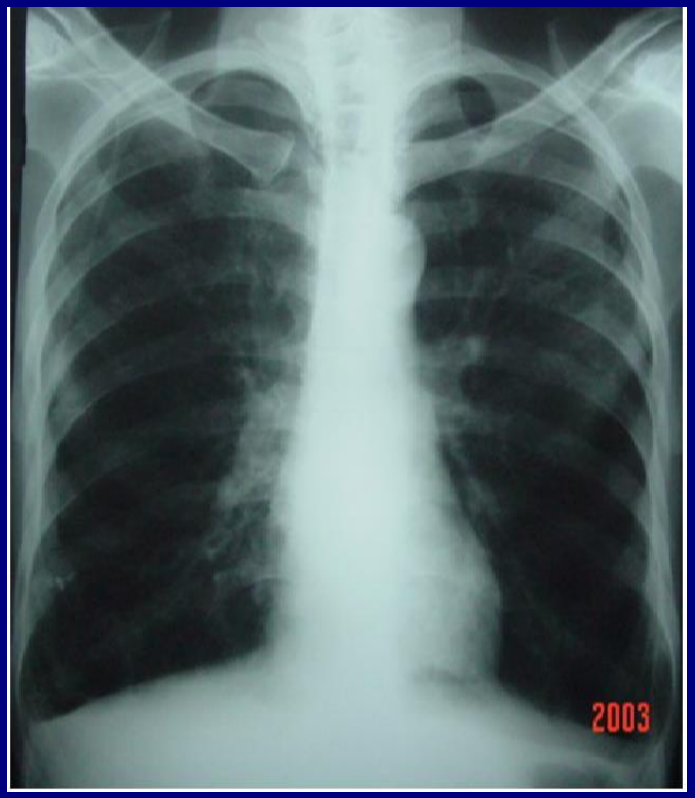
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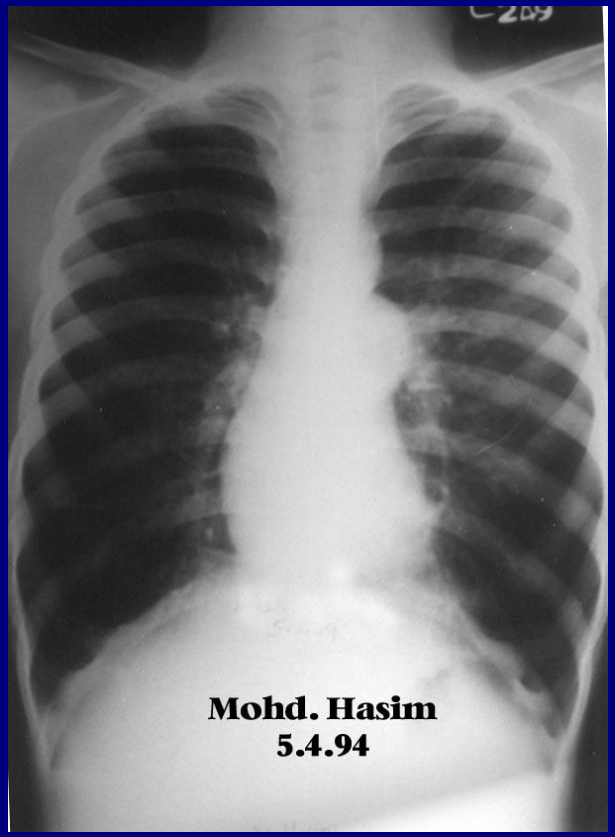
Mohd. Hasim
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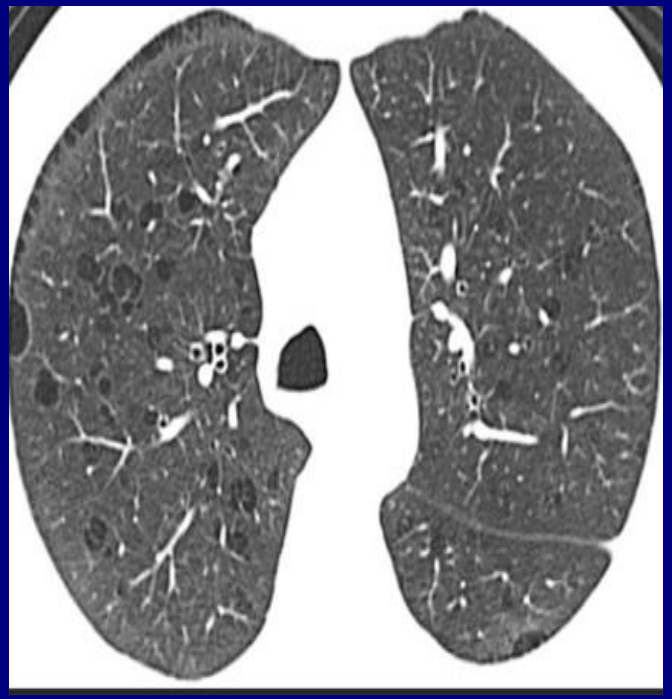
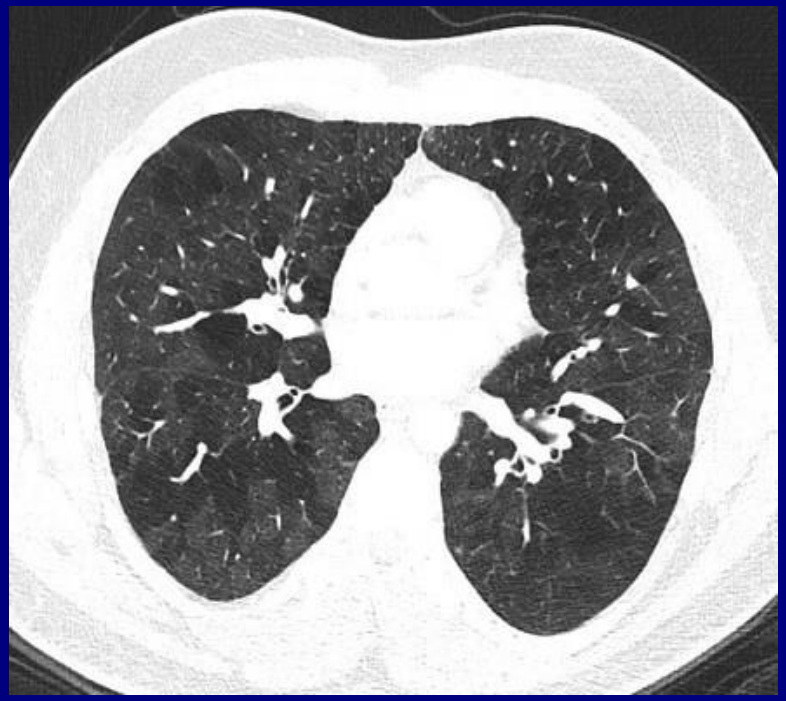
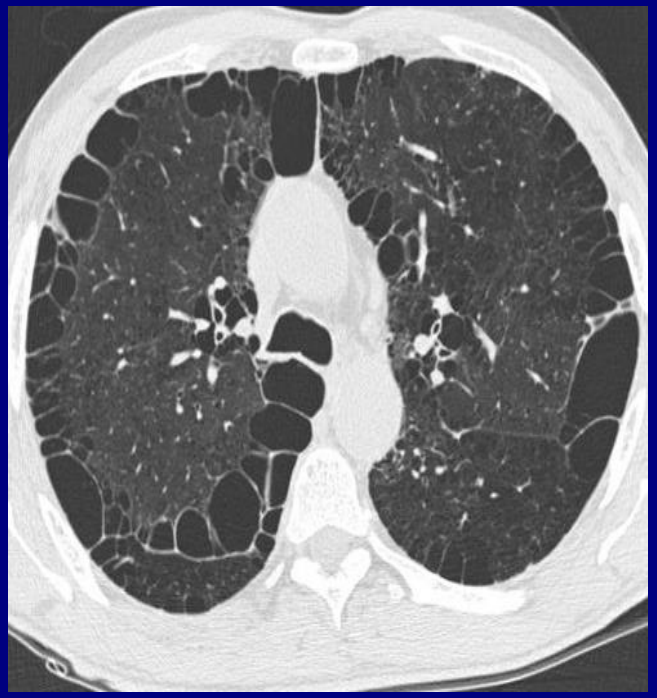


Centrilobular Emphysema

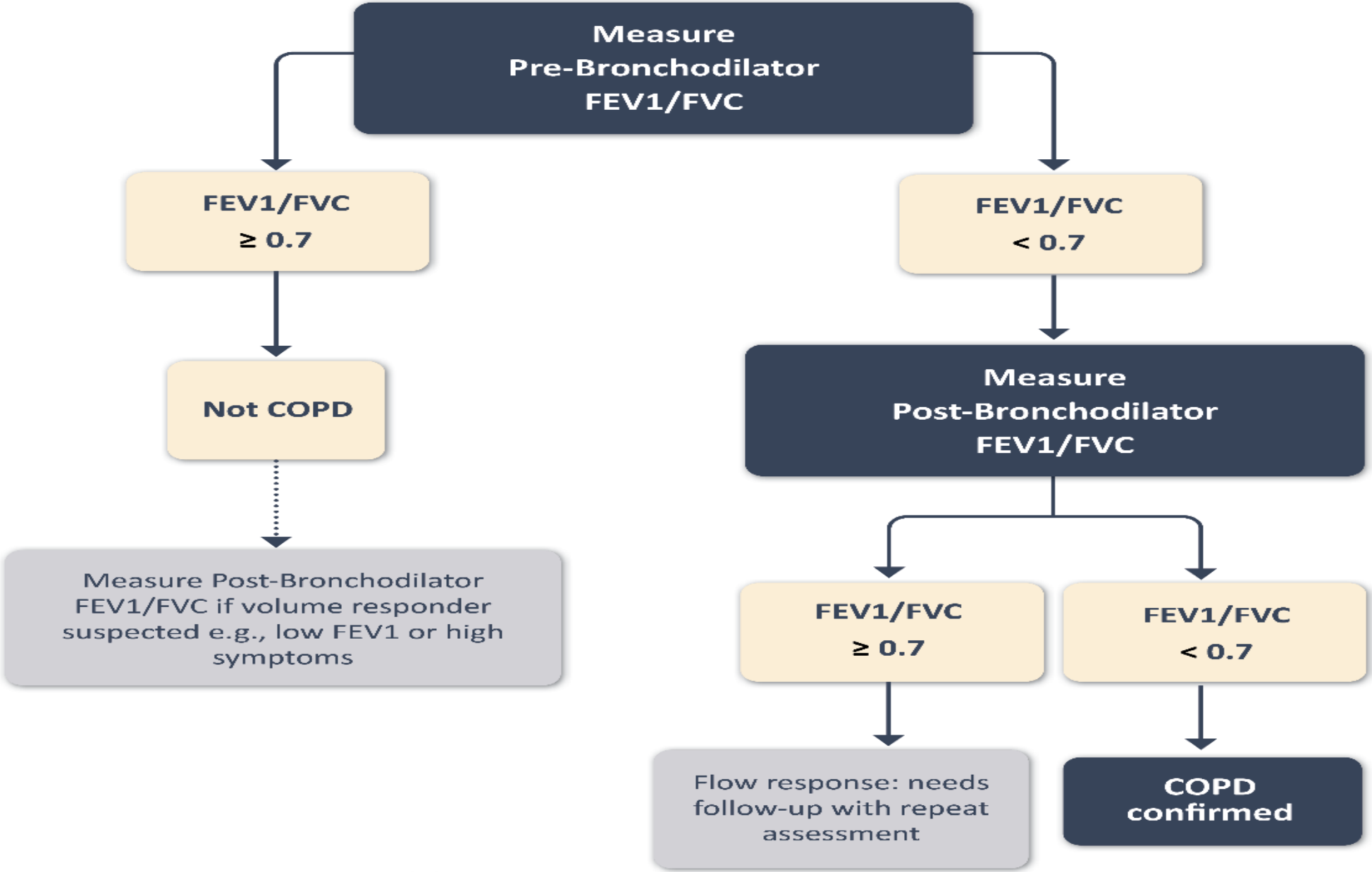


Panlobular Emphysema



Paraseptal Emphysema

Pre- and Post- Bronchodilator Spirometry



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Role of Spirometry in COPD

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

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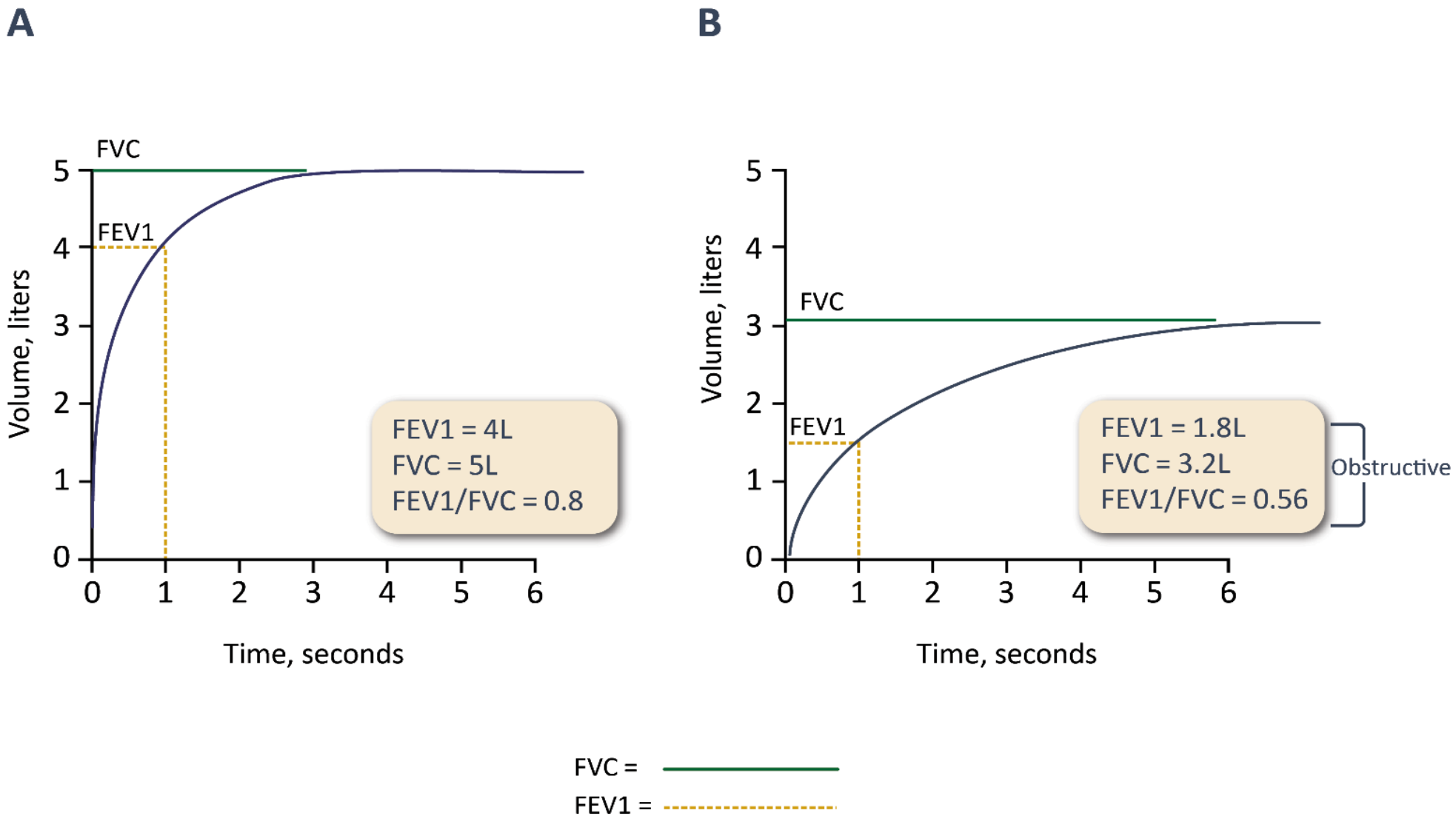
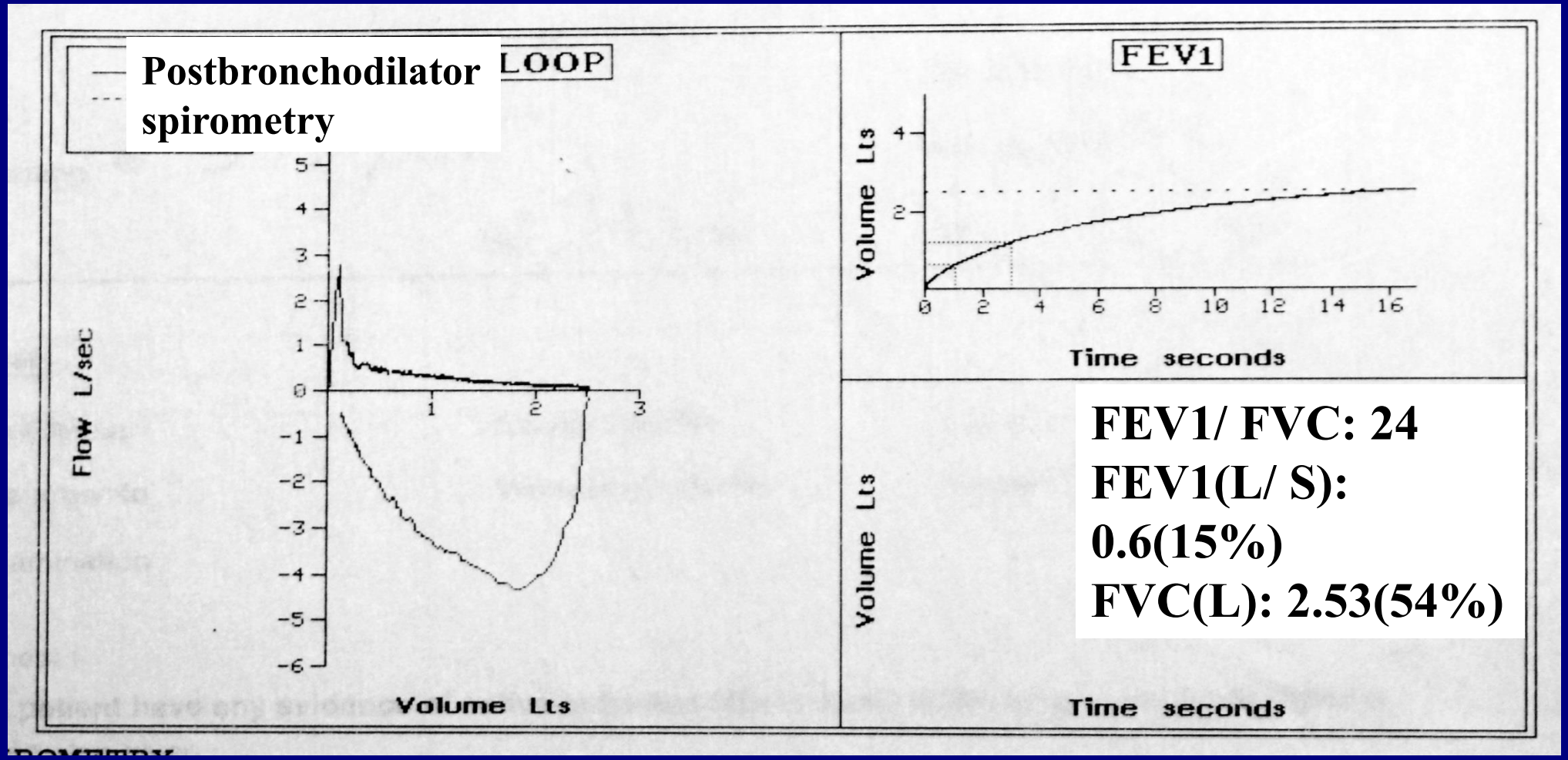
A. Spirometry-Normal Trace B. Spirometry-Airflow Obstruction



Gold 2023

Spirometry

R Prasad Lucknow



ASTHMA vs. COPD: CLINICAL DIFFERENCES

	ASTHMA	COPD
Symptoms	Variable Wheeze	Persistent SOB on exertion
Onset	Usually childhood	Usually >45yr
Course	Variable, remissions rarely progressive	Progressive
Smoking	Sometimes	Usually
Resp to b/d	Good	Poor
Resp to steroids	Good	Poor

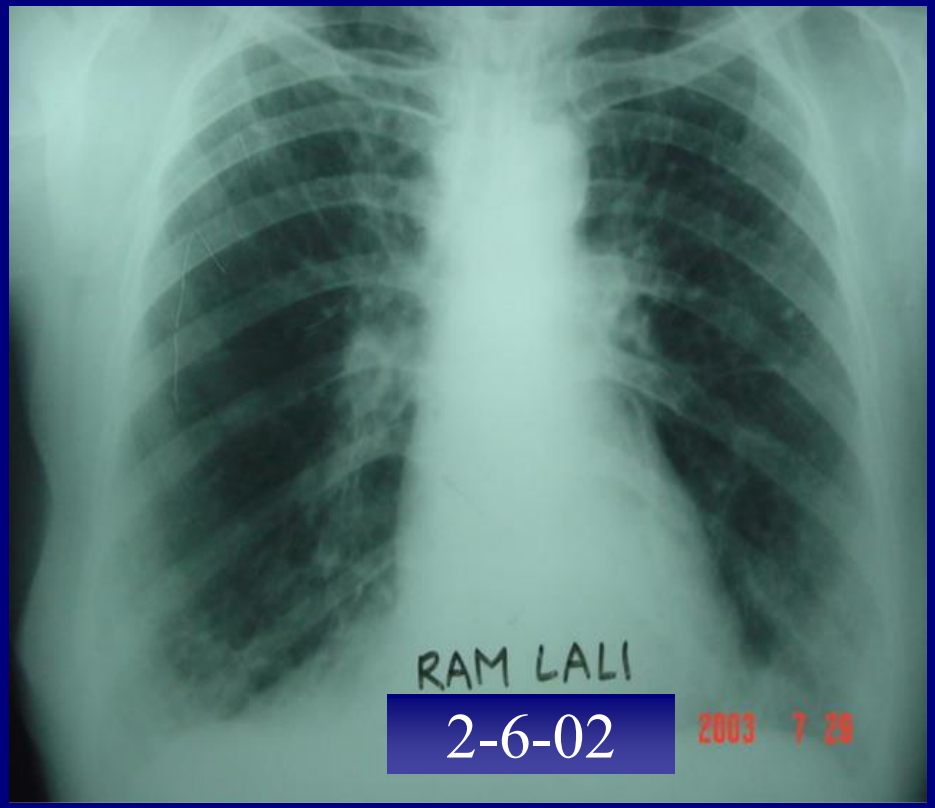
RL 61 yrs. Old female C/o

cough with expectoration - 5 years

Breathlessness – 3 Years

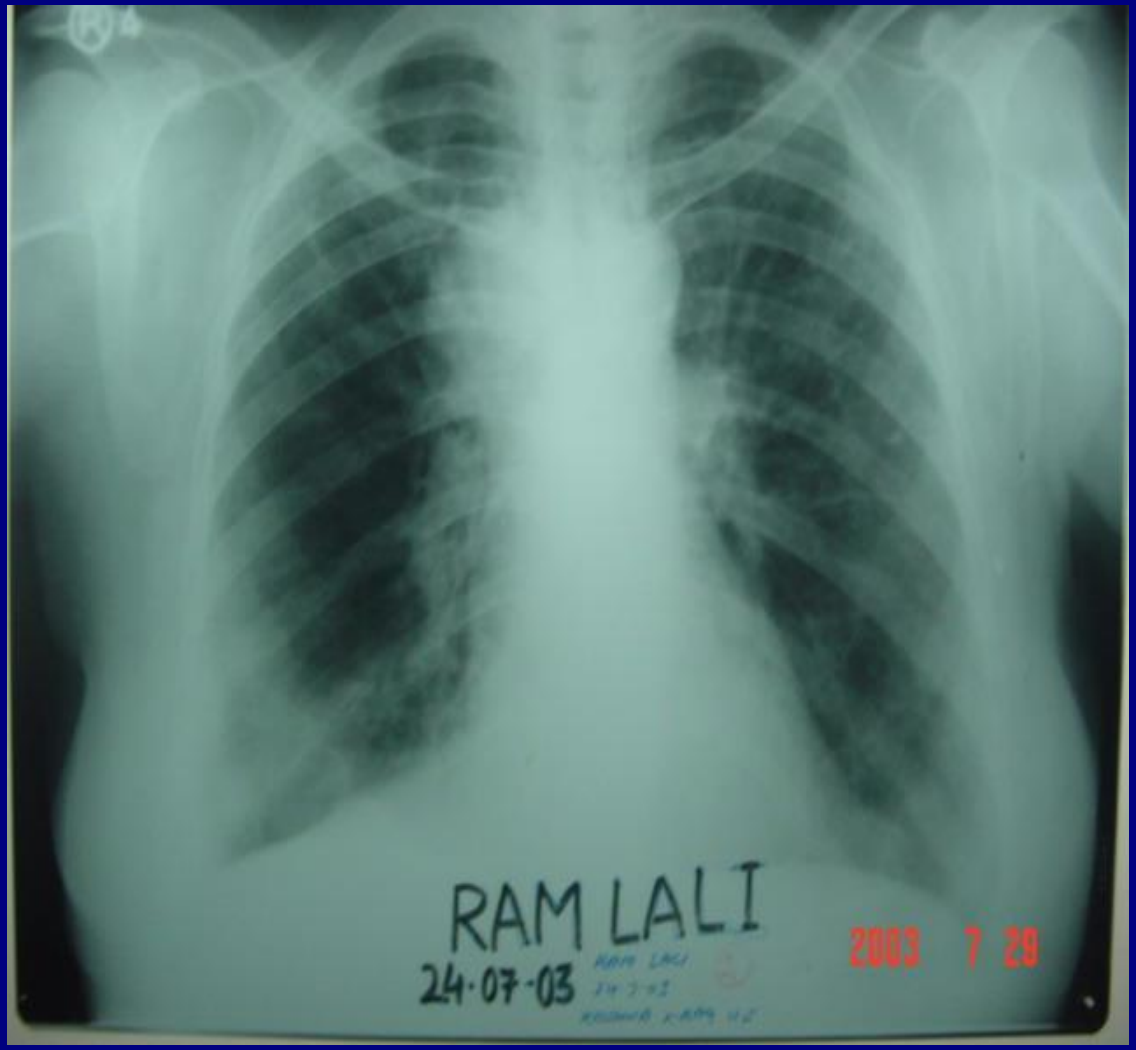
Smoking - 15 Bidi/day 35 years.

Past H/o ATT : RHE for 1 year. No response



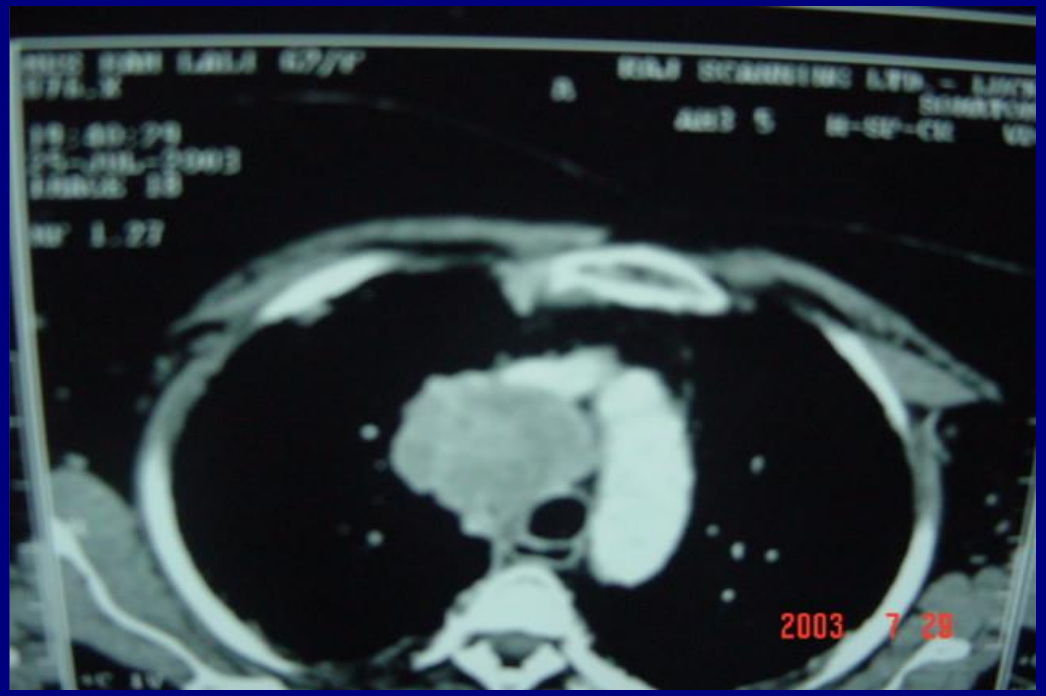
New symptom

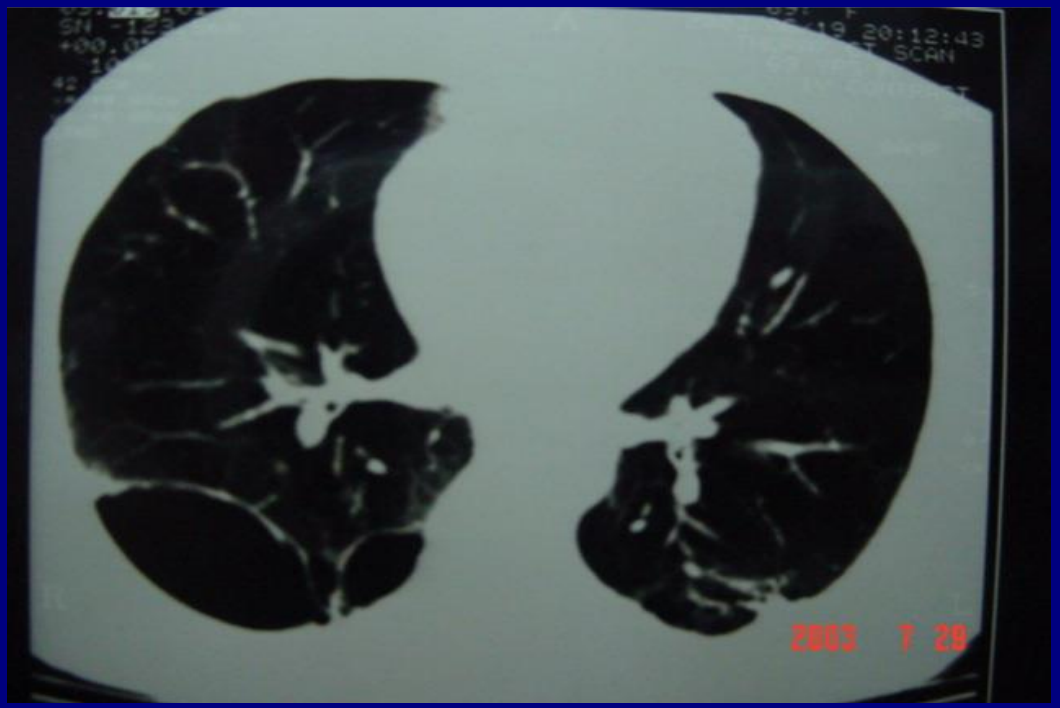
Chest pain Rt. - 1year
Rec.Haemoptysis- 1 year

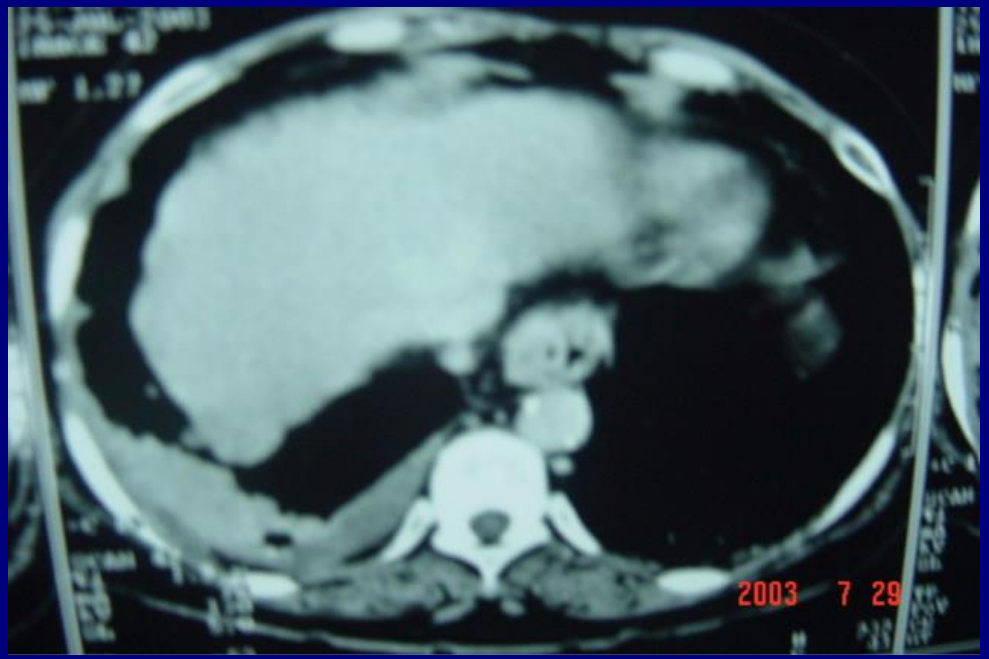


What you would like to do ?

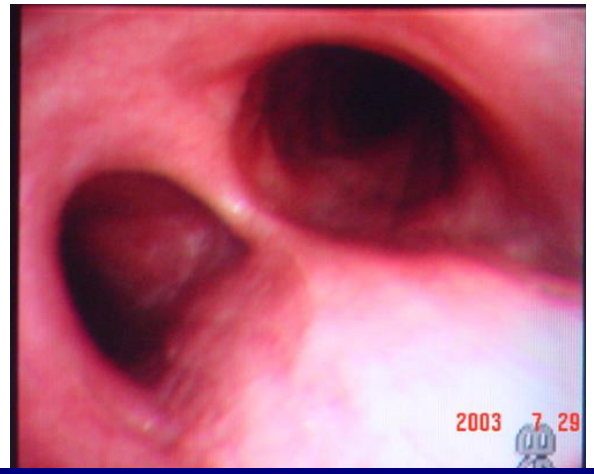
1. Just Treat COPD & Haemoptysis
2. CT Thorax
3. Bronchoscopy
4. ECG

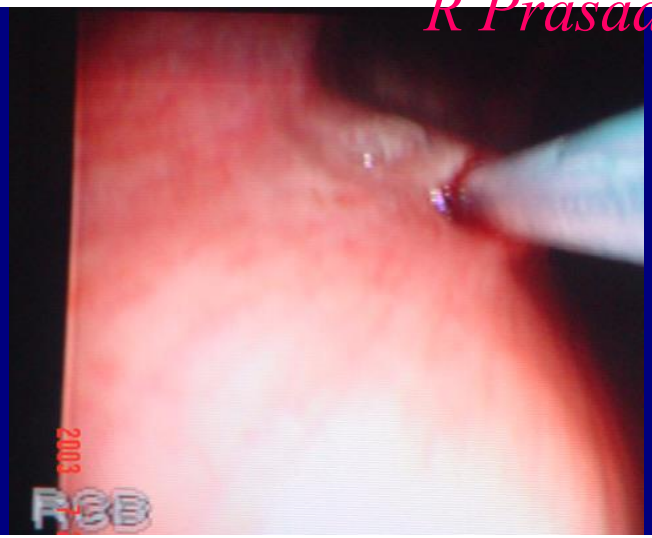


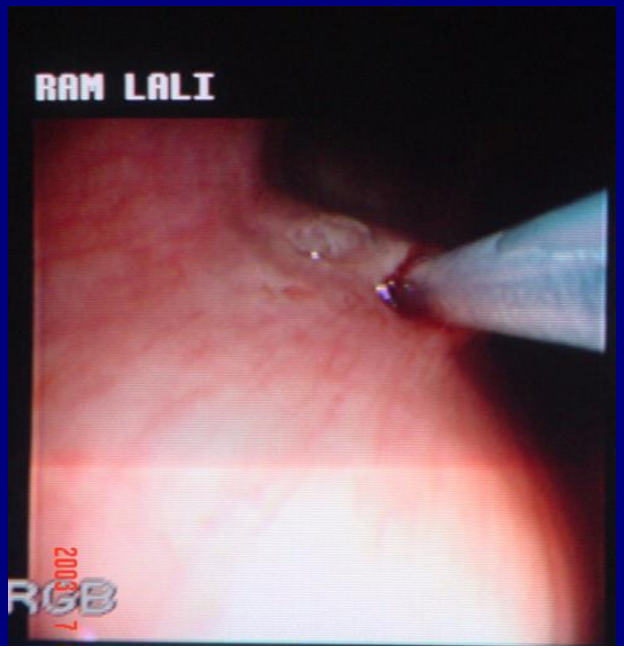


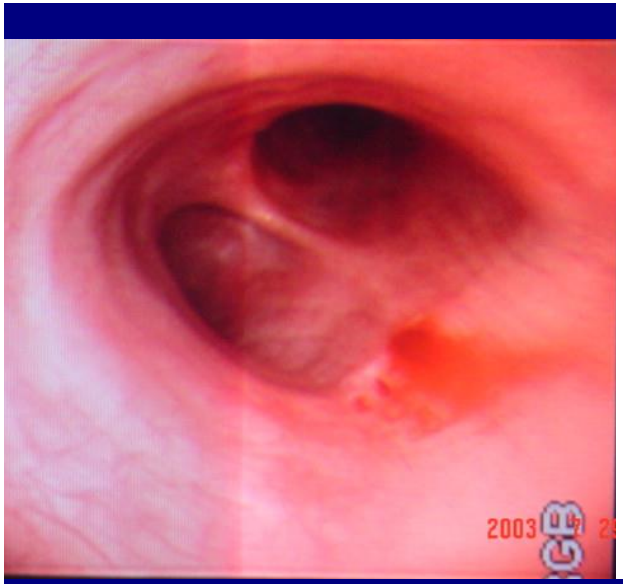


CT : Lobulated conglomerated Mass in mediastinum bullous lesion in both lung right sided pleural effusion with bilateral pleural thickening.









Transtrachial Needle Aspiration

Investigation

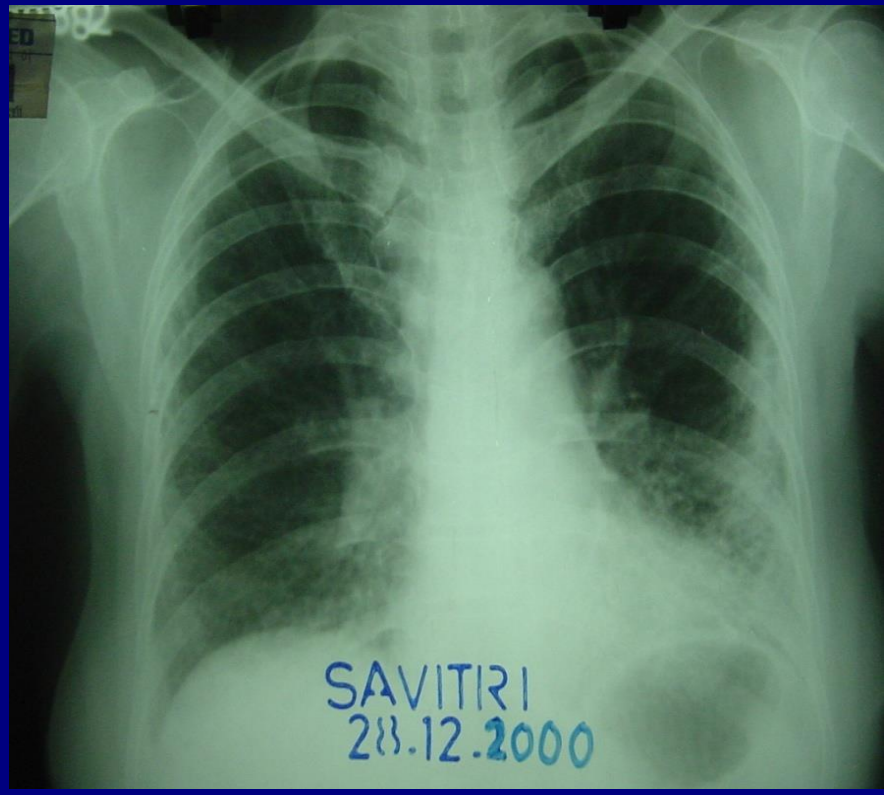
Transtracheal needle aspiration : Sq. Cell CA

Bone scan: Suggestive of skeletal metastasis in sacrum.

Diagnosis : Right sided Bronchogenic carcinoma type squamous cell stage IV (T4N2M1) with COPD

R Prasad Lucknow

- Savitri Devi 65 yr Female. Non Smoker
- C/O. Cough – 5 Months
- Breathlessness (Exer.) - 5 Months.
- On Examination
  - Clubbing Present
  - Bilateral basal inspiratory Crepts
- Put on ATT for 6 Months without response

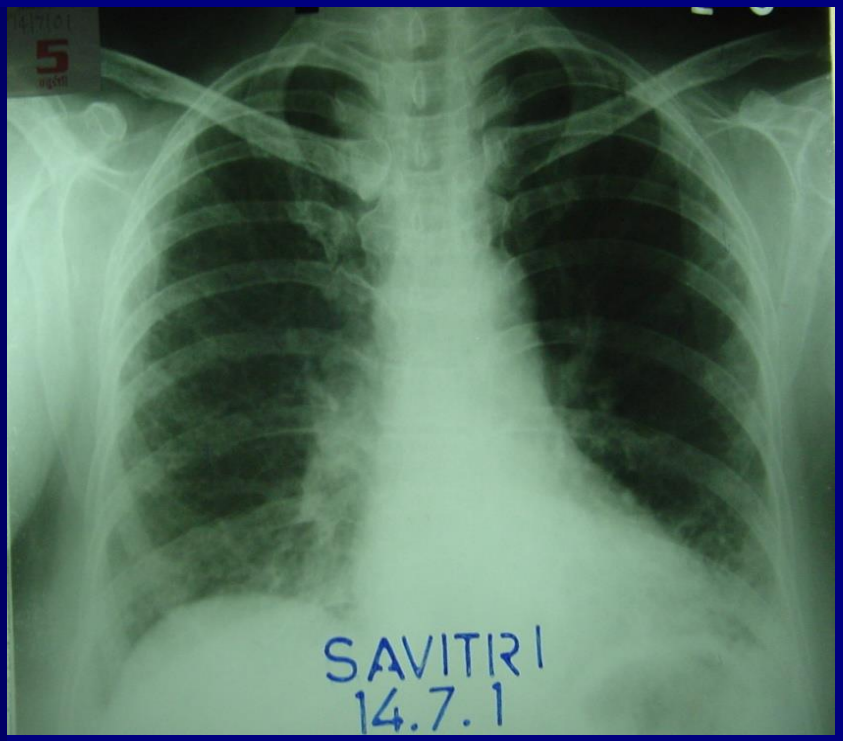


What are the differential diagnosis ?

1. COPD
2. Bronchial Asthma
3. Pulmonary Tuberculosis
4. Idiopathic Pulmonary Fibrosis

- PFT

FVC	.68
FEV1	.67
FEV1/FVC	99%



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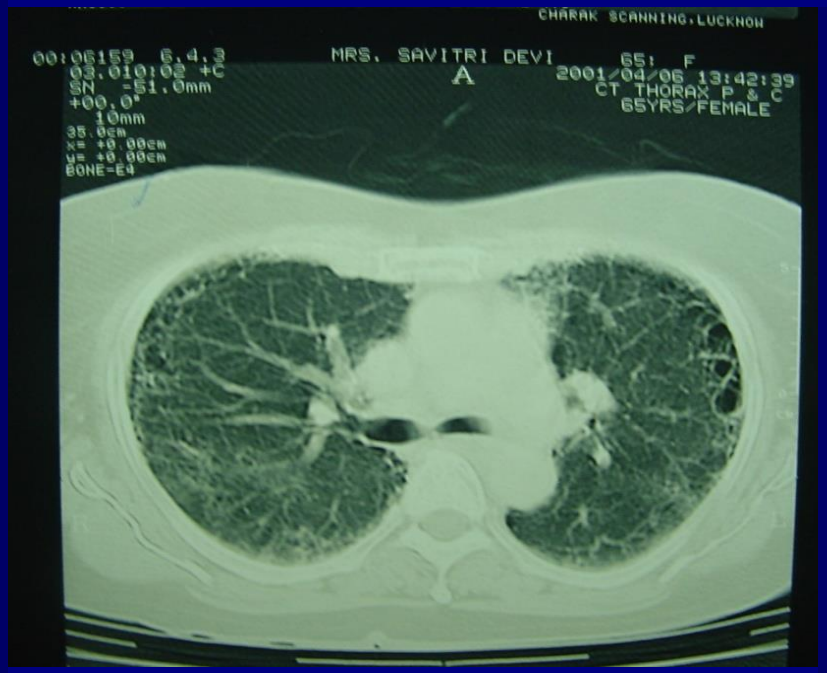

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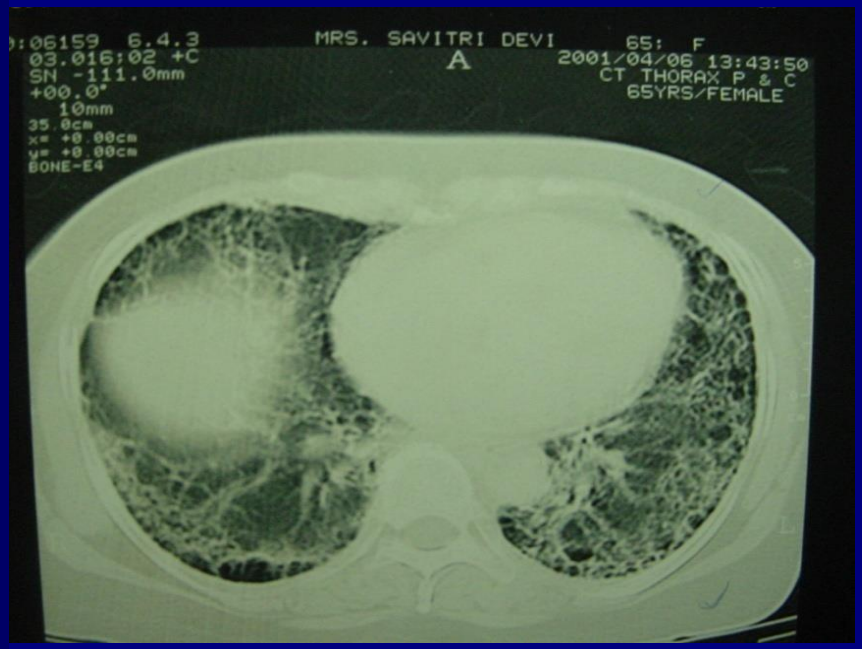

What is Diagnosis ?

1. COPD
2. Bronchial Asthma
3. Pulmonary Tuberculosis
4. Idiopathic Pulmonary Fibrosis

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Idiopathic Pulmonary Fibrosis

43 Yrs. Male COPD patient presented with

- Heavy snoring
- Bed partner reports that he some times stops breathing while he sleeps
- Feeling sleepy at times when he drives
- Has HT controlled by medication
- P/E - Overweight
  - BMI = 33
  - Neck circum. is 46 cm.

WHAT DO YOU SUSPECT ?

OBSTRUCTIVE SLEEP APNOEA (OSA)

WHAT WILL YOU DO NEXT

- Leave him as such.
- Just treat COPD and hypertension.
- Give some drugs for day time sleepiness.
- Polysomnography.

R Prasad Lucknow

COPD Management

Guidelines for management

- *GOLD, 2025*
- *ICS /NCCP (Indian Guidelines), 2013*
- *BTS/NICE, 2017,2019*
- *ERS/ATS, 2023*

Components of COPD Management

1. Assess and monitor disease
2. Reduce risk factors
3. Manage stable COPD
   - Education
   - Pharmacologic
   - Non-pharmacologic
4. Manage exacerbations
5. Manage Comorbidities

Assessment of COPD

- Current level of patient's symptoms
- Severity of the spirometric abnormality
- Frequency of exacerbations
- Presence of comorbidities

Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2022

Modified MRC dyspnea scale

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

mMRC Grade 0	mMRC Grade 1	mMRC Grade 2	mMRC Grade 3	mMRC Grade 4
I only get breathless with strenuous exercise	I get short of breath when hurrying on the level or walking up a slight hill	I walk slower than people of the same age on the level because of breathlessness, or I have to stop for breath when walking on my own pace on the level	I stop for breath after walking about 100 meters or after a few minutes on the level	I am too breathless to leave the house or I am breathless when dressing or undressing
☐	☐	☐	☐	☐

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

How is your COPD today?

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

Score	Impact
>30	very high
>20	High
10 – 20	Moderate
<10	Low

CODP assessment test. Available online: http://www.catestonline.org/

CAT assessment

2025
Teaching Slide Set

CAT™ Assessment

Figure 2.10

For each item below, place a mark (x) in the box that best describes you currently.
Be sure to only select one response for each question.

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

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

TOTAL SCORE:

Gold Grades and severity of Airflow obstruction in COPD (based on post-bronchodilator FEV1)

In COPD patients (FEV1/FVC < 0.7):

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

THE REFINED ABCD ASSESSMENT TOOL

Spirometrically Confirmed Diagnosis

Assessment of airflow limitation

Assessment of symptoms/risk of exacerbations

Post-bronchodilator $FEV_1/FVC < 0.7$

Grade	FEV_1 (% predicted)
GOLD 1	≥ 80
GOLD 2	50-79
GOLD 3	30-49
GOLD 4	< 30

Moderate or Severe Exacerbation History

≥2 or ≥ 1 leading to hospital admission

0 or 1 (not leading to hospital admission)

C	D
A	B

mMRC 0-1 CAT < 10

mMRC ≥ 2 CAT ≥ 10

Symptoms

Spirometrically confirmed diagnosis → Assessment of airflow obstruction → Assessment of symptoms/risk of exacerbations

Post-bronchodilator FEV1/FVC < 0.7

GRADE	FEV1 (% predicted)
GOLD 1	≥ 80
GOLD 2	50-79
GOLD 3	30-49
GOLD 4	< 30

EXACERBATION HISTORY

	SYMPTOMS	
≥ 2 moderate exacerbations or ≥ 1 leading to hospitalization	**E**	
0 or 1 moderate exacerbations (not leading to hospitalization)	**A**	**B**
	mMRC 0-1 CAT < 10	mMRC ≥ 2 CAT ≥ 10

Use of CT in stable COPD

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

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Assess COPD Comorbidities

COPD patients are at increased risk for:

- Cardiovascular diseases
- Osteoporosis
- Respiratory infections
- Anxiety and Depression
- Metabolic syndrome & Diabetes
- Bronchiectasis
- Lung cancer

These comorbid conditions may influence mortality and hospitalizations and should be looked for routinely, and treated appropriately

GOALS FOR TREATMENT OF STABLE COPD

- Relieve Symptoms
- Improve Exercise Tolerance
- Improve Health Status

→ REDUCE SYMPTOMS

and

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

→ REDUCE RISK

Identify & Reduce Risk Factor Exposure

Tabl

- Smoking cessation interventions should be actively pursued in all people with COPD **(Evidence A)**
- Efficient ventilation, non-polluting cooking stoves and similar interventions should be recommended **(Evidence B)**
- Clinicians should advise patients to avoid continued exposures to potential irritants, if possible **(Evidence D)**

Brief Strategies to Help the Patient Willing to Quit

Tab

ASK	Systematically identify all tobacco users at every visit *Implement an office-wide system that ensures that, for EVERY patient at EVERY clinic visit, tobacco-use status is queried and documented*
ADVISE	Strongly urge all tobacco users to quit *In a clear, strong, and personalized manner, urge every tobacco user to quit*
ASSESS	Determine willingness and rationale of patient's desire to make a quit attempt. *Ask every tobacco user if he or she is willing to make a quit attempt at this time (e.g., within the next 30 days)*
ASSIST	Aid the patient in quitting *Help the patient with a quit plan; provide practical counseling; provide intra-treatment social support; help the patient obtain extra-treatment social support; recommend use of approved pharmacotherapy except in special circumstances; provide supplementary materials*
ARRANGE	Schedule follow-up contact *Schedule follow-up contact, either in person or via telephone*

Commonly Used Maintenance Medications in COPD*

Table 3.3

Generic Drug Name	Inhaler Type	DELIVERY OPTIONS			Duration of Action
		Nebulizer	Oral	Injection	
BETA$_2$-Agonists					
Short-acting (SABA)					
Fenoterol	MDI	✓	pill, syrup		4-6 hours
Levalbuterol	MDI	✓			6-8 hours
Salbutamol (albuterol)	MDI & DPI	✓	pill, syrup, extended release tablet	✓	4-6 hours 12 hours (ext. release)
Terbutaline	DPI		pill	✓	4-6 hours
Long-acting (LABA)					
Arformoterol		✓			12 hours
Formoterol	DPI	✓			12 hours
Indacaterol	DPI				24 hours
Olodaterol	SMI				24 hours
Salmeterol	MDI & DPI				12 hours
Anticholinergics					
Short-acting (SAMA)					
Ipratropium bromide	MDI	✓			6-8 hours
Oxitropium bromide	MDI				7-9 hours
Long-acting (LAMA)					
Aclidinium bromide	DPI,				MDI 12 hours
Glycopyrronium bromide	DPI		solution	✓	12-24 hours
Tiotropium	DPI, SMI, MDI				24 hours
Umeclidinium	DPI				24 hours
Glycopyrrolate		✓			12 hours
Revefenacin		✓			24 hours
Combination Short-Acting Beta$_2$-Agonist Plus Anticholinergic in One Device (SABA+SAMA)					
Fenoterol/ipratropium	SMI	✓			6-8 hours
Salbutamol/ipratropium	SMI, MDI	✓			6-8 hours
Combination Long-Acting Beta$_2$-Agonist Plus Anticholinergic in One Device (LABA+LAMA)					
Formoterol/aclidinium	DPI				12 hours
Formoterol/glycopyrronium	MDI				12 hours
Indacaterol/glycopyrronium	DPI				12-24 hours
Vilanterol/umeclidinium	DPI				24 hours
Olodaterol/tiotropium	SMI				24 hours
Methylxanthines					
Aminophylline			solution	✓	Variable, up to 24 hours
Theophylline (SR)			pill	✓	Variable, up to 24 hours
Combination of Long-Acting Beta$_2$-Agonist Plus Corticosteroid in One Device (LABA+ICS)					
Formoterol/beclometasone	MDI, DPI				12 hours
Formoterol/budesonide	MDI, DPI				12 hours
Formoterol/mometasone	MDI				12 hours
Salmeterol/fluticasone propionate	MDI, DPI				12 hours
Vilanterol/fluticasone furoate	DPI				24 hours
Triple Combination in One Device (LABA+LAMA+ICS)					
Fluticasone/umeclidinium/vilanterol	DPI				24 hours
Beclometasone/formoterol/glycopyrronium	MDI, DPI				12 hours
Budesonide/formoterol/glycopyrrolate	MDI				12 hours
Phosphodiesterase-4 Inhibitors					
Roflumilast			pill		24 hours
Mucolytic Agents					
Erdosteine			pill		12 hours
Carbocysteine†			pill		
N-acetylcysteine†			pill		

*Not all formulations are available in all countries. In some countries other formulations and dosages may be available. †Dosing regimens are under discussion.
MDI = metered dose inhaler; DPI = dry powder inhaler; SMI = soft mist inhaler. Note that glycopyrrolate & glycopyrronium are the same compound.

Bronchodilators in stable COPD

- Inhaled bronchodilators in COPD are central to symptom management and commonly given on a regular basis to prevent or reduce symptoms **(Evidence A)**
- Inhaled bronchodilators are recommended over oral bronchodilators (**Evidence A**)
- Regular and as-needed use of SABA or SAMA improves FEV1 and symptoms **(Evidence A)**
- Combinations of SABA and SAMA are superior compared to either medication alone in improving FEV1 and symptoms **(Evidence A)**
- LABAs and LAMAs are preferred over short-acting agents except for patients with only occasional dyspnea (**Evidence A**), and for immediate relief of symptoms in patients already on long-acting bronchodilators for maintenance therapy
- LABAs and LAMAs significantly improve lung function, dyspnea, health status, and reduce exacerbation rates **(Evidence A)**
- LAMAs have a greater effect on exacerbation reduction compared with LABAs **(Evidence A)** and decrease hospitalizations **(Evidence B)**
- When initiating treatment with long acting bronchodilators the preferred choice is a combination of a LABA and a LAMA. In patients with persistent dyspnea on a single long-acting bronchodilator treatment should be escalated to two (**Evidence A**).
- Combination treatment with a LABA and a LAMA increases FEV1 and reduces symptoms compared to monotherapy **(Evidence A)**
- Combination treatment with a LABA+LAMA reduces exacerbations compared to monotherapy **(Evidence B)**
- Combinations can be given as single inhaler or multiple inhaler treatment. Single inhaler therapy may be more convenient and effective than multiple inhalers
- Ensifentrine significantly improves lung function **(Evidence A)**, dyspnea **(Evidence A)** and health status **(Evidence B)**
- Theophylline exerts a small bronchodilator effect in stable COPD **(Evidence A)** and that is associated with modest symptomatic benefits **(Evidence B)**

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Anti-inflammatory therapy in stable COPD

Inhaled Corticosteroids	• Regular treatment with ICS increases the risk of pneumonia especially in those with severe disease **(Evidence A)** • An ICS combined with a LABA is more effective than the individual components in improving lung function and health status and reducing exacerbations in patients with exacerbations and moderate to very severe COPD **(Evidence A)** • We do not encourage the use of a LABA+ICS combination in COPD. If there is an indication for an ICS the combination LABA+LAMA+ICS has been shown to be superior to LABA+ICS and is therefore the preferred choice • Triple inhaled therapy of LABA+LAMA+ICS improves lung function, symptoms and health status, and reduces exacerbations, compared to LABA+ICS, LABA+LAMA or LAMA monotherapy **(Evidence A)**. Recent data suggesta beneficial effect of triple inhaled therapy versus fixed-dose LABA+LAMA combinations on mortality in symptomatic COPD patients with a history of frequent and/or severe exacerbations • If patients with COPD have features of asthma, treatment should always contain an ICS • Independent of ICS use, there is evidence that a blood eosinophil count < 2% increases the risk of pneumonia **(Evidence C)** • Combinations can be given as single or multiple inhaler therapy. Single inhaler therapy may be more convenient and effective than multiple inhalers
Oral Glucocorticoids	• Long-term use of oral glucocorticoids has numerous side effects **(Evidence A)** with no evidence of benefits **(Evidence C)**
PDE Inhibitors	• In patients with chronic bronchitis, severe to very severe COPD and a history of exacerbations: ▪ Roflumilast improves lung function and reduces moderate and severe exacerbations **(Evidence A)** • Ensifentrine improves lung function **(Evidence A)** but an effect on exacerbations has not been evaluated in patients at increased exacerbation risk
Antibiotics	• Long-term azithromycin and erythromycin therapy reduces exacerbations over one year **(Evidence A)** • Preferentially, but not only in former smokers with exacerbations despite appropriate therapy, azithromycin can be considered **(Evidence B)** • Treatment with azithromycin is associated with an increased incidence of bacterial resistance **(Evidence A)** and hearing test impairments **(Evidence B)**
Mucoregulators & Antioxidant Agents	• Regular treatment with mucolytics such as erdosteine, carbocysteine and NAC reduces the risk of exacerbations in select populations **(Evidence B)** • Antioxidant mucolytics are recommended only in selected patients **(Evidence A)**
Biologics	• In patients with moderate to severe COPD with a history of exacerbations, chronic bronchitis and higher blood eosinophil counts (≥ 300 cells/µL): ▪ Dupilumab reduces exacerbations, improves lung function and quality of life **(Evidence A)**
Other Anti-Inflammatory Agents	• Statin therapy is not recommended for prevention of exacerbations **(Evidence A)** • Simvastatin does not prevent exacerbations in COPD patients at increased risk of exacerbations and without indications for statin therapy **(Evidence A)**. However, observational studies suggest that statins may have positive effects on some outcomes in patients with COPD who receive them for cardiovascular and metabolic indications **(Evidence C)** • Leukotriene modifiers have not been tested adequately in COPD patients

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Key points for Inhalation of drugs

- When a treatment is given by the inhaled route, the importance of education and training in inhaler device technique cannot be over-emphasized
- The choice of inhaler device has to be individually tailored and will depend on access, cost, prescriber, and most importantly, patient's ability and preference
- It is essential to provide instructions and to demonstrate the proper inhalation technique when prescribing a device, to ensure that inhaler technique is adequate and re-check at each visit that patients continue to use their inhaler correctly
- Inhaler technique (and adherence to therapy) should be assessed before concluding that the current therapy is insufficient

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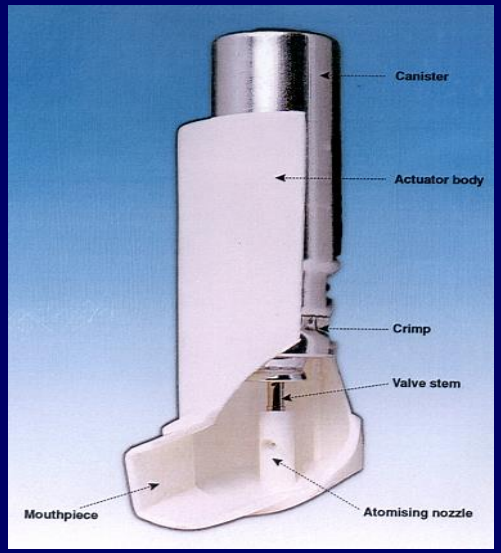
Devices


Canister
Actuator body
Crimp
Valve stem
Mouthpiece
Atomising nozzle


LUPIHALER


Foradil


HandiHaler


Bricanyl
Turbohaler
100 doses
Pulmicort
Turbohaler 100
200 doses
Pulmicort
Turbohaler 200
100 doses
Pulmicort
Turbohaler 400
50 doses


Ventolin

Commonly used Devices

Single dose DPIs

Cyclohaler

Handihaler

Rotahaler

Multidose DPIs

Reservoir DPI

Diskhaler

Accuhaler

Turbuhaler

Basic principles for appropriate inhalation device choice

- Availability of the drug in the device.
- Patients' beliefs, satisfaction with current and previous devices and preferences need to be assessed and considered.
- The number of different device types should be minimized for each patient.
- Device type should not be switched in the absence of clinical justification nor without proper information, education and medical follow-up.
- Shared decision-making is the most appropriate strategy for inhalation device choice.
- Patient's cognition, dexterity and strength must be taken into account.
- Patient's ability to perform the correct specific inhalation maneuver for the device must be assessed:
  - Dry powder inhalers are appropriate only if the patient can make a forceful and deep inhalation. Check visually that the patient can inhale forcefully through the device - if there is doubt assess objectively or choose alternative device.
  - Metered-dose inhalers and, to a lesser extent, soft mist inhalers require coordination between device triggering and inhalation and patients need to be able to perform a slow and deep inhalation. Check visually that the patient can inhale slowly and deeply from the device - if there is doubt consider adding a spacer/VHC or choose an alternative device.
  - For patients unable to use an MDI (with or without spacer/VHC), SMI or DPI a nebulizer should be considered.
- Other factors to consider include size, portability, cost.
- Smart inhalers may be useful if there are issues with adherence/persistence or inhalation technique (for devices that can check it).
- Physicians should prescribe only devices they (and the other members of the caring team) know how to use.

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Initial Pharmacological Treatment

≥ 2 moderate exacerbations or ≥ 1 leading to hospitalization

GROUP E

LABA + LAMA*

consider LABA+LAMA+ICS if blood eos ≥ 300*

0 or 1 moderate exacerbations (not leading to hospital admission)

GROUP A

A bronchodilator

mMRC 0-1, CAT < 10

GROUP B

LABA + LAMA*

mMRC ≥ 2, CAT ≥ 10

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

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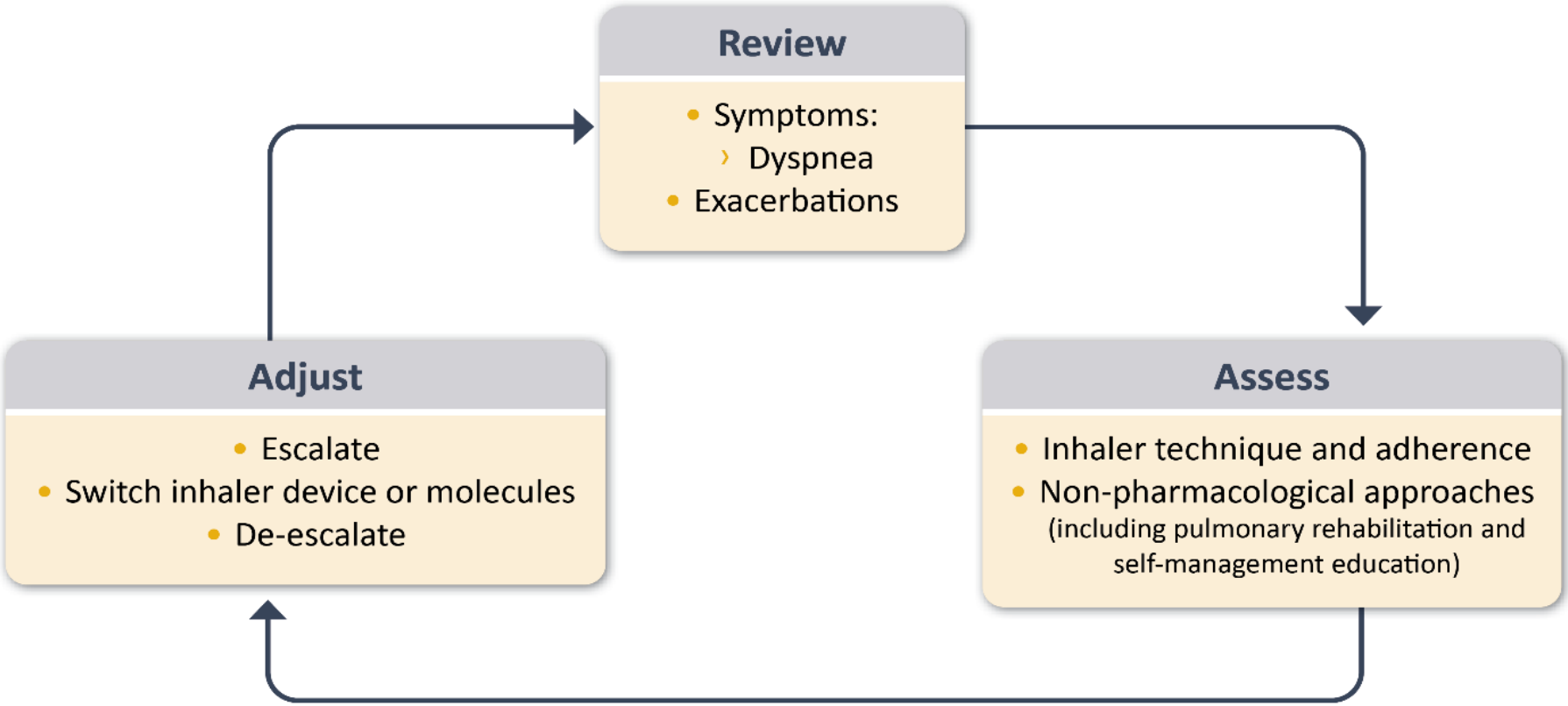
Management cycle



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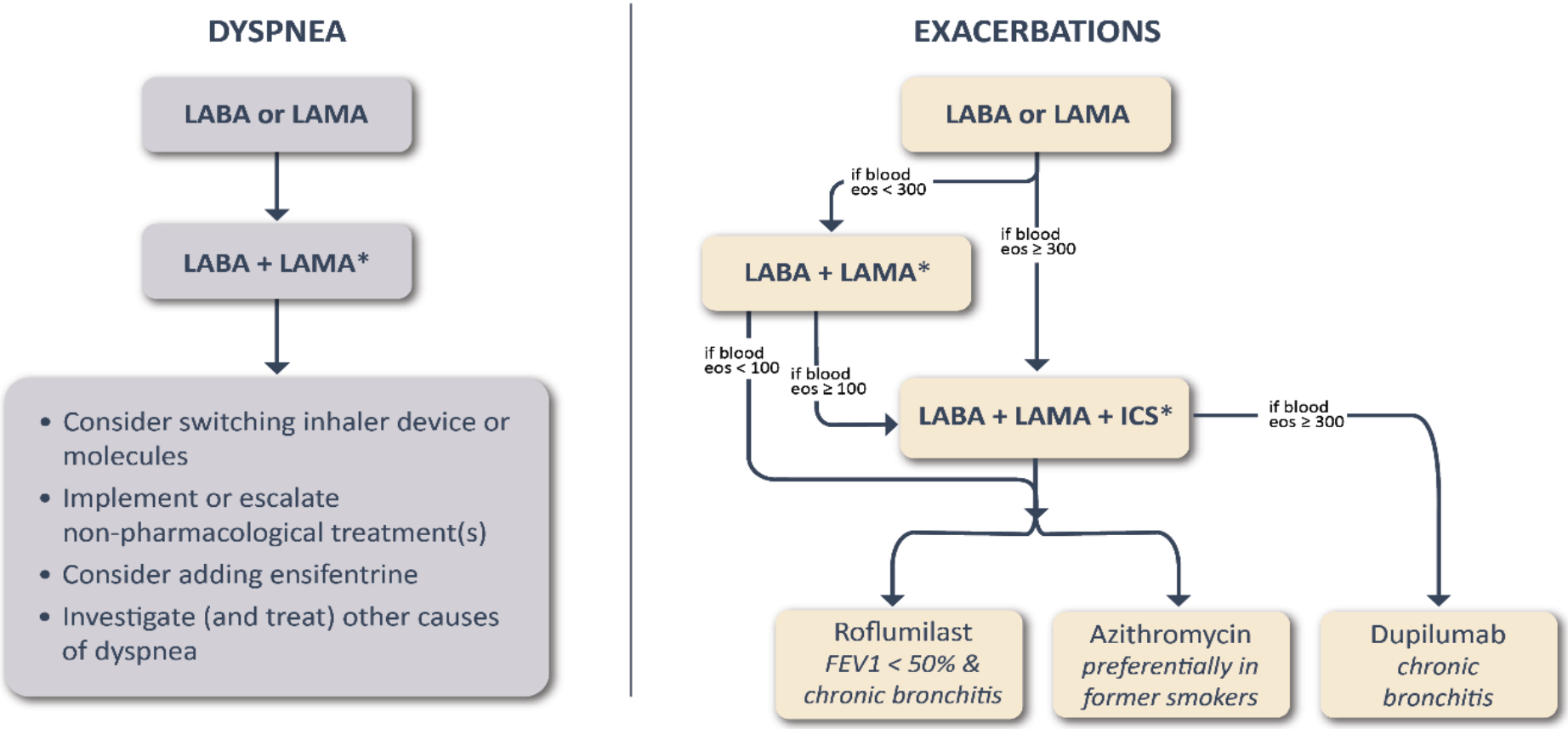
Follow-up pharmacological treatment



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

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

Exacerbations refers to the number of exacerbations per year.

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Factors to consider when Initiating ICS Treatment

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

(note the scenario is different when considering ICS withdrawal)

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

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

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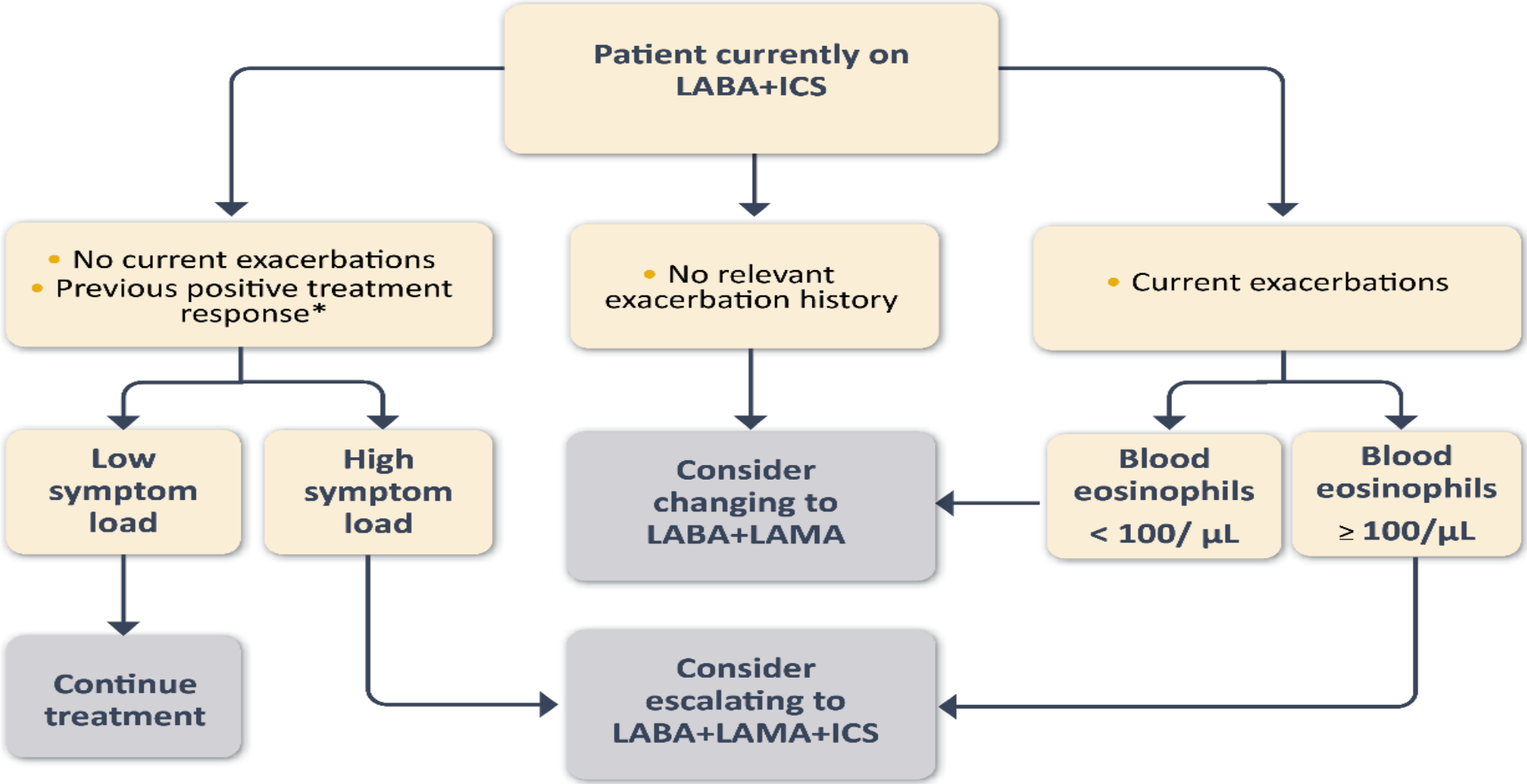
Management of patients currently on LABA plus ICS



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

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Other pharmacological treatments

Alpha-1 Antitrypsin Augmentation Therapy	• Intravenous augmentation therapy may slow down the progression of emphysema **(Evidence B)**
Antitussives	• There is no conclusive evidence of a beneficial role of antitussives in people with COPD **(Evidence C)**
Vasodilators	• Vasodilators do not improve outcomes and may worsen oxygenation **(Evidence B)**
Opioids	• Low-dose long acting oral and parenteral opioids may be considered for treating dyspnea in COPD patients with severe disease (**Evidence B**)
Pulmonary Hypertension Therapy	• Drugs approved for primary pulmonary hypertension are not recommended for patients with a pulmonary hypertension secondary to COPD (**Evidence B**)

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Non pharmacological therapies

Rehabilitation

Long-term oxygen therapy

Smoking Cessation

Vaccination

Surgery

Non-pharmacological management of COPD

Patient Group	Essential	Recommended	Depending on Local Guidelines
A	Smoking cessation (can include pharmacological treatment)	Physical activity	Influenza vaccination COVID-19 vaccinations Pneumococcal vaccination Pertussis vaccination Shingles vaccination RSV vaccination
B and E	Smoking cessation (can include pharmacological treatment) Pulmonary rehabilitation	Physical activity	Influenza vaccination COVID-19 vaccinations Pneumococcal vaccination Pertussis vaccination Shingles vaccination RSV vaccination

*Can include pharmacological treatment

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Brief Strategies to Help the Patient Willing to Quit

Tab

ASK	Systematically identify all tobacco users at every visit *Implement an office-wide system that ensures that, for EVERY patient at EVERY clinic visit, tobacco-use status is queried and documented*
ADVISE	Strongly urge all tobacco users to quit *In a clear, strong, and personalized manner, urge every tobacco user to quit*
ASSESS	Determine willingness and rationale of patient's desire to make a quit attempt. *Ask every tobacco user if he or she is willing to make a quit attempt at this time (e.g., within the next 30 days)*
ASSIST	Aid the patient in quitting *Help the patient with a quit plan; provide practical counseling; provide intra-treatment social support; help the patient obtain extra-treatment social support; recommend use of approved pharmacotherapy except in special circumstances; provide supplementary materials*
ARRANGE	Schedule follow-up contact *Schedule follow-up contact, either in person or via telephone*

Pulmonary Rehabilitation

R Prasad Lucknow

Pulmonary rehabilitation, self-management and integrative care in COPD

Pulmonary Rehabilitation	• Rehabilitation is indicated in all patients with relevant symptoms and/or a high risk for exacerbation **(Evidence A)** • Pulmonary rehabilitation improves dyspnea, health status and exercise tolerance in stable patients **(Evidence A)** • Pulmonary rehabilitation reduces hospitalization among patients who have had a recent exacerbation (≤ 4 weeks from prior hospitalization) **(Evidence B)** • Pulmonary rehabilitation leads to a reduction in symptoms of anxiety and depression **(Evidence A)**
Education and Self-Management	• Education is needed to change patient's knowledge but there is no evidence that used alone it will change patient behavior **(Evidence C)** • Self-management intervention with communication with a health care professional improves health status and decreases hospitalizations and emergency department visits **(Evidence B)**
Integrated Care Programs	• Integrative care and telehealth have no demonstrated benefit at this time **(Evidence B)**
Physical Activity	• Physical activity is a strong predictor of mortality **(Evidence A)**. People with COPD should be encouraged to increase their level of physical activity although we still do not know how to best ensure the likelihood of success

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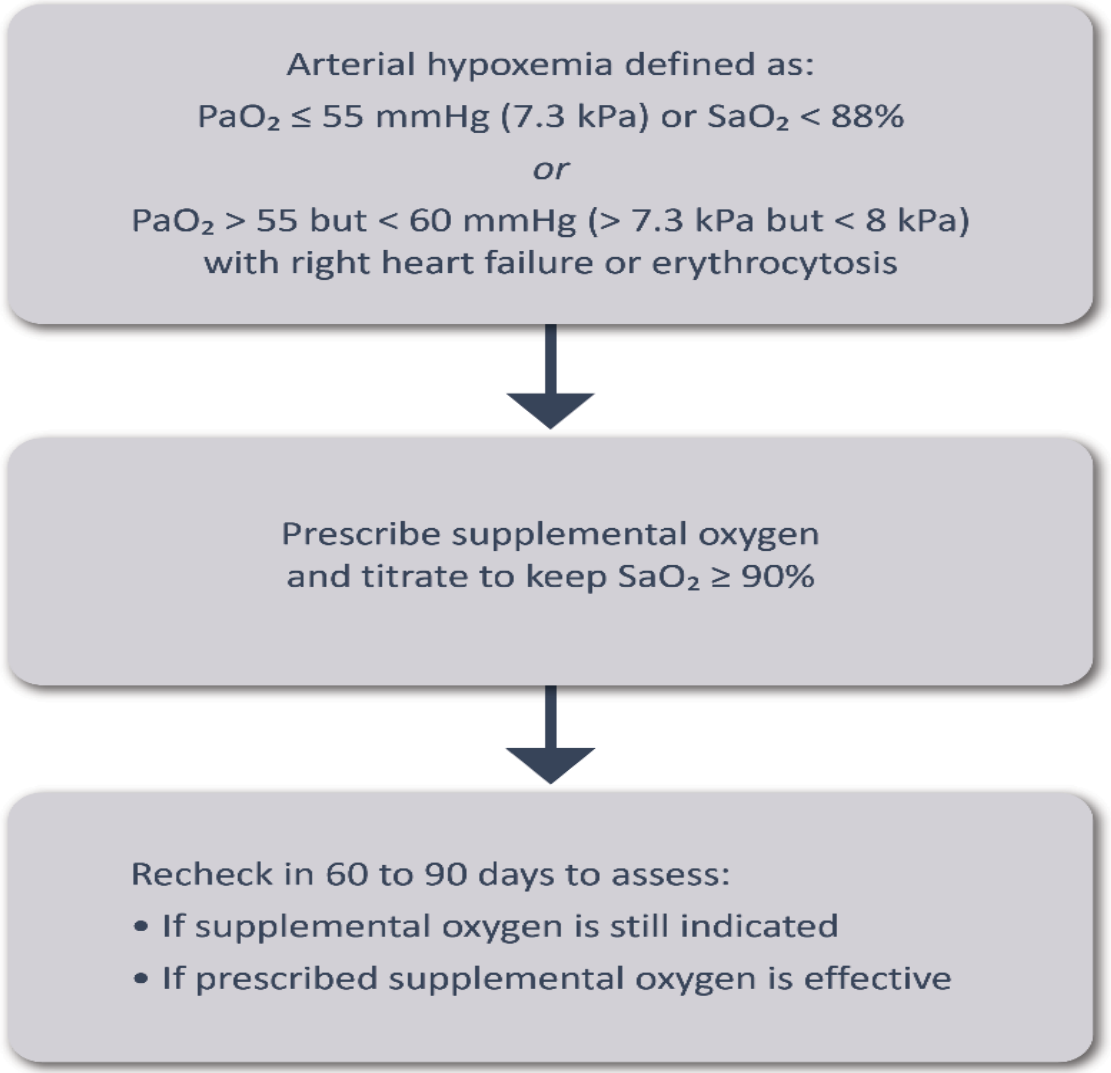
Prescription of supplemental oxygen to COPD patients



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Long-term oxygen therapy

Tokyo - Japan - 1989

- **Oxygen Therapy**: The long-term administration of oxygen (> 15 hours per day) to patients with chronic respiratory failure has been shown to increase survival (Evidence A).

Goals of LTOT

PaO_2 - at least 60 mm Hg

SaO_2 - at least 90 %

Oxygen therapy and ventilatory support in stable COPD

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

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Palliative care, end of life, and hospice care in COPD

- All clinicians managing patients with COPD should be aware of the effectiveness of palliative approaches to symptom control and use these in their practice **(Evidence D)**
- End of life care should include discussions with patients and their families about their views on resuscitation, advance directives and place of death preferences **(Evidence D)**
- Opiates, neuromuscular electrical stimulation (NMES), oxygen and fans blowing air onto the face can relieve breathlessness **(Evidence C)**
- Nutritional supplementation should be considered in malnourished patients with COPD (**Evidence B**) as it may improve respiratory muscle strength and overall health status **(Evidence B)**
- Fatigue can be improved by self-management education, pulmonary rehabilitation, nutritional support and mind-body interventions **(Evidence B)**

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Evidence supporting a reduction in mortality with pharmacotherapy and non-pharmacotherapy in COPD patients

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

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

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

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

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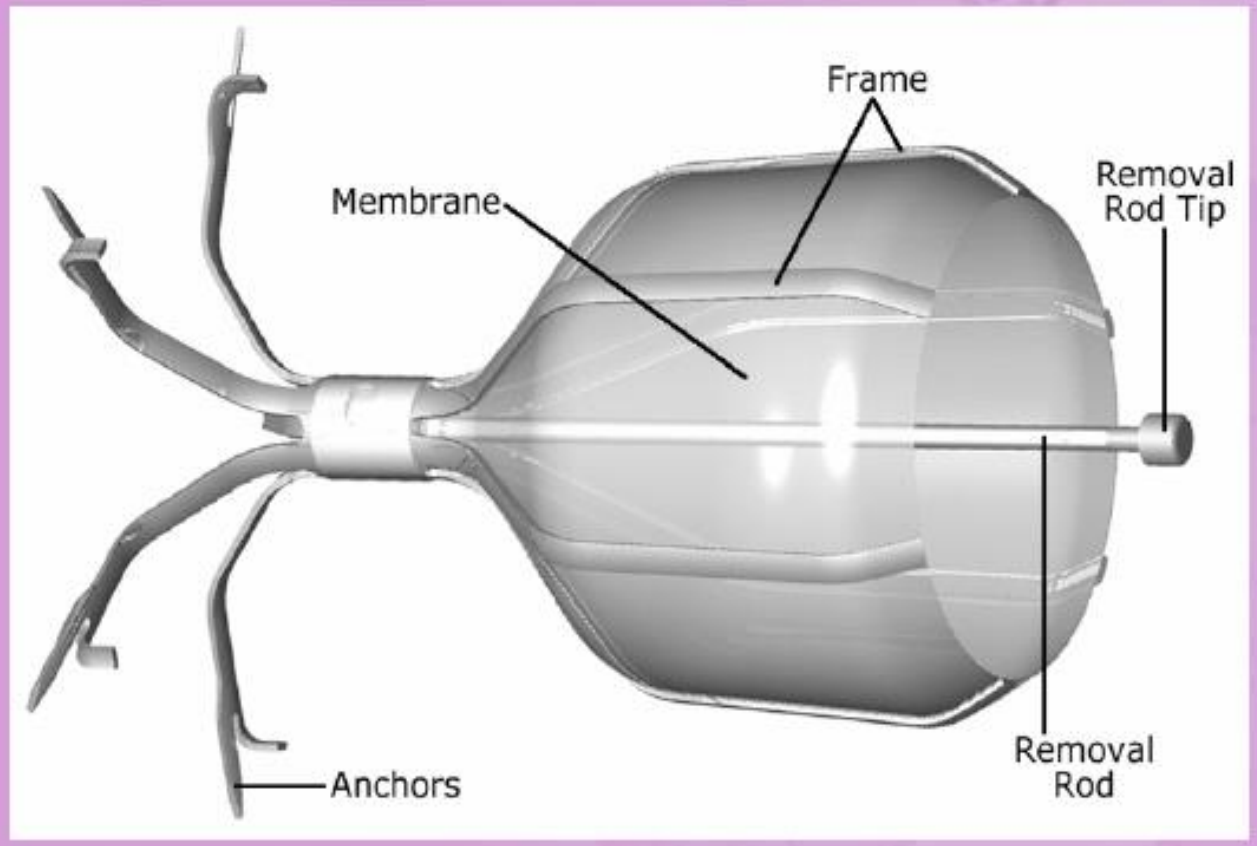
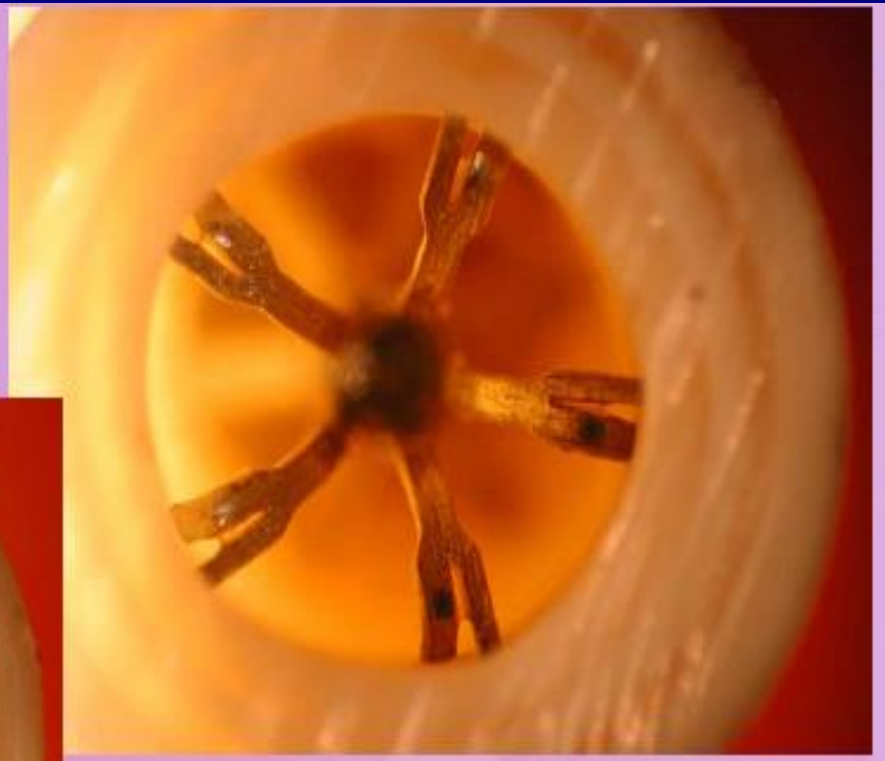
Surgical treatments

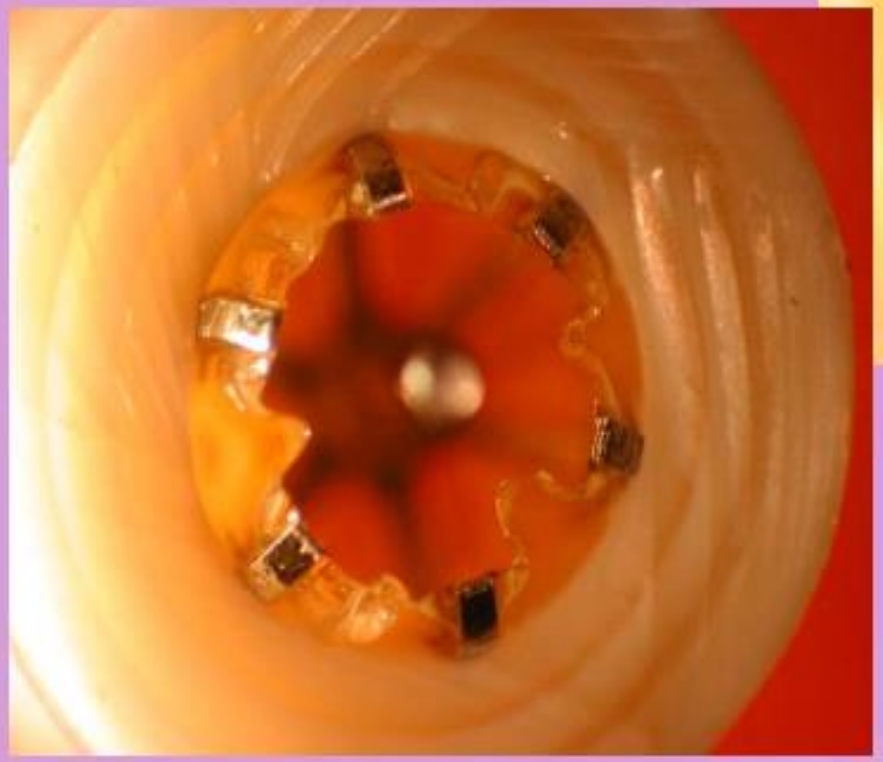
- Bullectomy
- Lung volume reduction surgery (LVRS)
- Non-Surgical LVR
- Lung transplantation

IBV Design



IBV Pre-Removal
Distal View

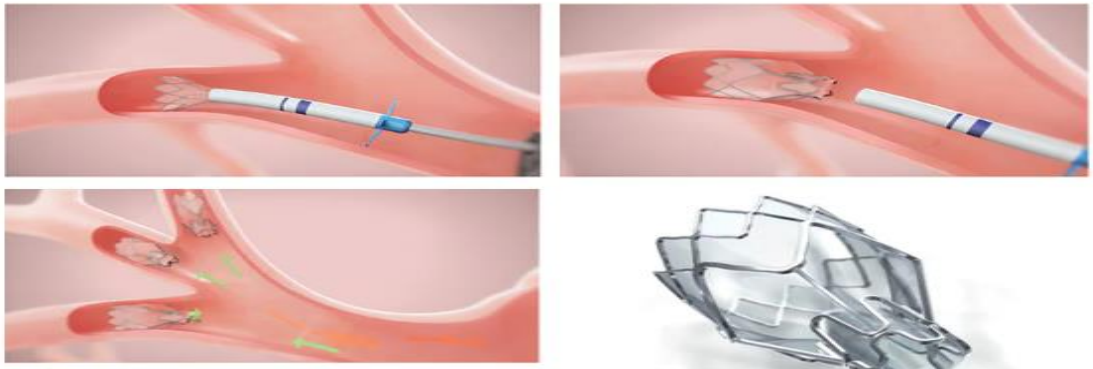




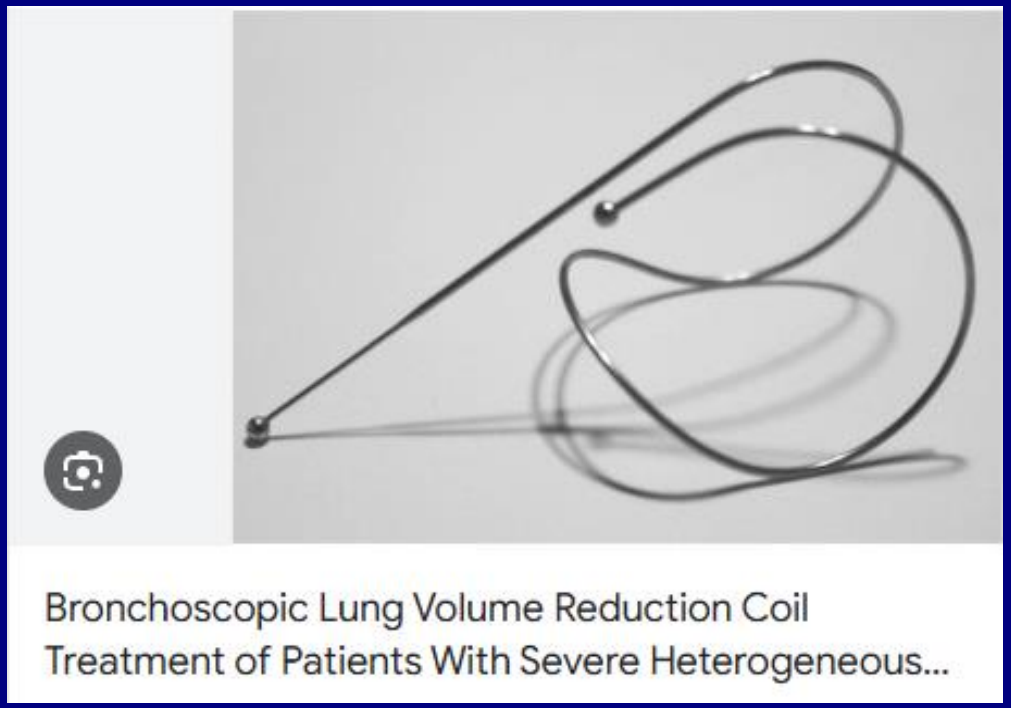
IBV Pre-Removal
Proximal View

Bronchoscopic lung volume reduction therapy approved for severe emphysema

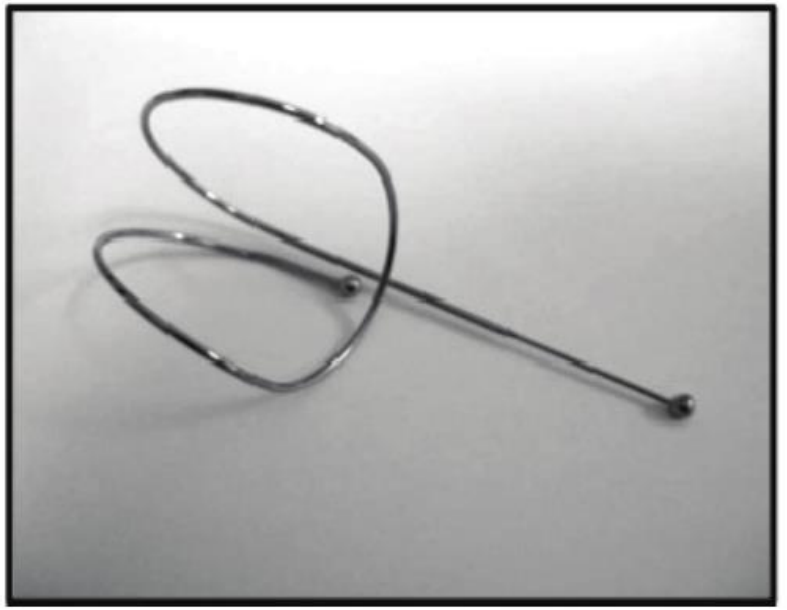
April 19, 2019



Zephyr valve, Image courtesy of Pulmonx Corp.



Bronchoscopic Lung Volume Reduction Coil Treatment of Patients With Severe Heterogeneous...



Endobronchial nitinol coil utilized for BLVR (RePneu ® Lung Volume Reduction Coil,

Current and proposed surgical and bronchoscopic interventions for people with COPD

Symptoms	Chronic Mucus Production	Exacerbations	Dyspnea
Disorders	• Chronic bronchitis	• Acute and chronic bronchitis • Bulla • Emphysema • Tracheobronchomalcia	• Bulla • Emphysema • Tracheobronchomalcia
Surgical and Bronchoscopic Interventions	• Nitrogen cryospray • Rheoplasty	• Targeted lung denervation	• Giant bullectomy • Large airway stenting • EBV • Coil • Thermal vapor ablation • Lung sealants • LVRS • Lung transplantation

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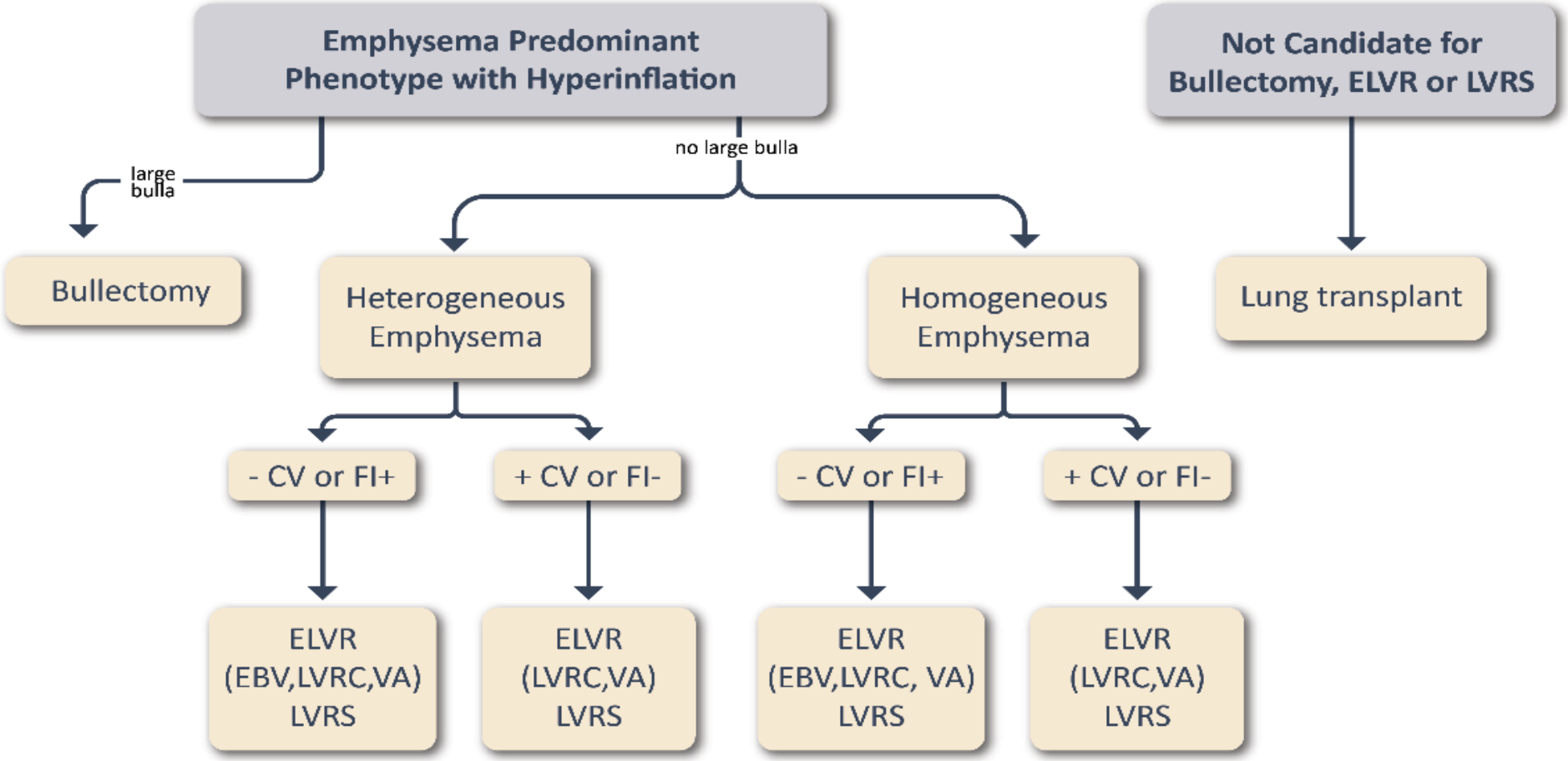
Surgical and interventional therapies in advanced emphysema



Note: not all therapies are clinically available in all countries. Long term ELVR outcomes or direct comparisons to LVRS are unknown.

Definition of abbreviations: CV, collateral ventilation measure by Chartis; FI + fissure integrity > 90% by HRCT; FI-, 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. Modified from Vogelmeier, AJRCCM, 2017.

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Interventional therapy in stable COPD

Lung Volume Reduction Surgery	• Lung volume reduction surgery improves survival in severe emphysema patients with an upper-lobe emphysema and low post-rehabilitation exercise capacity **(Evidence A)**
Bullectomy	• In selected patients, bullectomy is associated with decreased dyspnea, improved lung function and exercise tolerance **(Evidence C)**
Transplantation	• In appropriately selected patients with very severe COPD, lung transplantation has been shown to improve quality of life and functional capacity **(Evidence C)** • In patients with very severe COPD (progressive disease, BODE score of 7 to 10, and not candidates for lung volume reduction) lung transplantation may be considered for referral with at least one of the following: (1) history of hospitalization for exacerbation associated with acute hypercapnia (Pco_2 > 50 mmHg); (2) pulmonary hypertension and/or cor pulmonale, despite oxygen therapy; or (3) FEV1 < 20% and either DLco < 20% or homogenous distribution of emphysema **(Evidence C)**
Bronchoscopic Interventions	• In select patients with advanced emphysema, bronchoscopic interventions reduce end-expiratory lung volume and improve exercise tolerance, health status and lung function at 6-12 months following treatment. Endobronchial valves **(Evidence A)**; Lung coils **(Evidence B)**; Vapor ablation **(Evidence B)**
Bronchoscopic Interventions Under Study	• Phase III trials are currently being conducted to determine the efficacy of treatments for patients with refractory exacerbations and chronic bronchitis using cryospray, rheoplasty and targeted lung denervation technology

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Confounders or contributors to be considered in patients presenting with suspected COPD exacerbation

Most frequent	**Pneumonia**	• Chest radiograph
	Pulmonary embolism	• Clinical probability assessment (Hemoptysis, surgery, fracture, history of cancer, DVT) • D-dimer • CT angiography for pulmonary embolism
	Heart failure	• Chest radiograph • NT Pro-Brain Natriuretic Peptide (Pro-BNP) and BNP • Echocardiography
Less frequent	**Pneumothorax, pleural effusion**	• Chest radiograph • Thoracic ultrasound
	Myocardial infarction and/or cardic arrhythmias (atrial fibrillation/flutter)	• Electrocardiography • Troponin

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Exacerbation of COPD

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 airways

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Exacerbations : Diagnosis and assessment

1.	Complete a thorough clinical assessment for evidence of COPD and potential respiratory and non-respiratory concomitant diseases, including consideration of alternative causes for the patient's symptoms and signs: primarily pneumonia, heart failure, and pulmonary embolism.
2.	**Assess:** a. Symptoms, severity of dyspnea that can be determined by using a VAS, and documentation of the presence of cough. b. Signs (tachypnea, tachycardia), sputum volume and color, and respiratory distress (accessory muscle use).
3.	Evaluate severity by using appropriate additional investigations such as pulse oximetry, laboratory assessment, CRP, arterial blood gases.
4.	Consider appropriate place of care.
5.	Establish the cause of the event (viral, bacterial, environmental, other).

Abbreviations: COPD = chronic obstructive pulmonary disease; CRP = C-reactive protein; VAS = visual analog scale.

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Classification of the severity of COPD exacerbations

Confirm ECOPD Diagnosis and Episode Severity

Severity	Variable thresholds to determine severity
Mild (default)	• Dyspnea VAS < 5 • RR < 24 breaths/min • HR < 95 bpm • Resting SaO_2 ≥ 92% breathing ambient air (or patient's usual oxygen prescription) AND change ≤ 3% (when known) • CRP < 10 mg/L (if obtained)
Moderate (meets at least three of five*)	• Dyspnea VAS ≥ 5 • RR ≥ 24 breaths/min • HR ≥ 95 bpm • Resting SaO_2 < 92% breathing ambient air (or patient's usual oxygen prescription) AND/OR change > 3% (when known) • CRP ≥ 10 mg/L *If obtained, ABG may show hypoxemia (PaO_2 ≤ 60 mmHg) and/or hypercapnia ($PaCO_2$ > 45 mmHg) but no acidosis
Severe	• Dyspnea, RR, HR, SaO_2 and CRP same as moderate • ABG show new onset/worsening hypercapnia and acidosis ($PaCO_2$ > 45 mmHg and pH <7.35)

Consider Differential Diagnosis

- Heart failure
- Pneumonia
- Pulmonary embolism

- Appropriate testing and treatment

Determine etiology:
viral testing, sputum culture, other

Adapted from: The ROME Proposal, Celli et al. (2021) Am J Respir Crit Care Med. 204(11): 1251-8.

Abbreviations: VAS visual analog dyspnea scale; RR respiratory rate; HR heart rate; SaO_2 oxygen saturation; CRP C-reactive protein; ABG arterial blood gases; PaO_2 Arterial pressure of oxygen.

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Potential Indications for hospitalization Assessment*

- Severe symptoms such as sudden worsening of resting dyspnea, high respiratory rate, decreased oxygen saturation, confusion, drowsiness
- Acute respiratory failure
- Onset of new physical signs (e.g., cyanosis, peripheral edema)
- Failure of an exacerbation to respond to initial medical management
- Presence of serious comorbidities (e.g., heart failure, newly occurring arrhythmias, etc.)
- Insufficient home support

*Local resources need to be considered

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Management of Severe but not Life-threatening Exacerbations*

- **Assess severity of symptoms, blood gases, chest radiograph**
- **Administer supplemental oxygen therapy, obtain serial arterial blood gas, venous blood gas and pulse oximetry measurements**
- **Bronchodilators:**
  - Increase doses and/or frequency of short-acting bronchodilators
  - Combine short-acting beta$_2$-agonists and anticholinergics
  - Consider use of long-acting bronchodilators when patient becomes stable
  - Use spacers or air-driven nebulizers when appropriate
- **Consider oral corticosteroids**
- **Consider antibiotics (oral) when signs of bacterial infection are present**
- **Consider noninvasive mechanical ventilation (NIV)**
- **At all times:**
  - Monitor fluid balance
  - Consider subcutaneous heparin or low molecular weight heparin for thromboembolism prophylaxis
  - Identify and treat associated conditions (e.g., heart failure, arrhythmias, pulmonary embolism etc.)

*Local resources need to be considered

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Key Points for Management of Exacerbations

- Short-acting inhaled beta$_2$-agonists, with or without short-acting anticholinergics, are recommended as the initial bronchodilators to treat an acute exacerbation **(Evidence C)**
- Systemic corticosteroids can improve lung function (FEV1), oxygenation and shorten recovery time and hospitalization duration. Duration of therapy should not normally be more than 5 days **(Evidence A)**
- Antibiotics, when indicated, can shorten recovery time, reduce the risk of early relapse, treatment failure, and hospitalization duration. Duration of therapy should normally be 5 days **(Evidence B)**
- Methylxanthines are not recommended due to increased side effect profiles **(Evidence B)**
- Non-invasive mechanical ventilation should be the first mode of ventilation used in COPD patients with acute respiratory failure who have no absolute contraindication because it improves gas exchange, reduces work of breathing and the need for intubation, decreases hospitalization duration and improves survival **(Evidence A)**

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Indications for Respiratory or Medical Intensive Care Unit Admission*

- Severe dyspnea that responds inadequately to initial emergency therapy
- Changes in mental status (confusion, lethargy, coma)
- Persistent or worsening hypoxemia (PaO_2 < 5.3 kPa or 40 mmHg) and/or severe/worsening respiratory acidosis (pH < 7.25) despite supplemental oxygen and noninvasive ventilation
- Need for invasive mechanical ventilation
- Hemodynamic instability - need for vasopressors

*Local resources need to be considered.

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Indications for Noninvasive Mechanical Ventilation(NIV)

At least one of the following:

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

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Indications for Invasive Mechanical Ventilation

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

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Discharge Criteria and Recommendations for Follow-up

1. Full review of all clinical and laboratory data.
2. Check maintenance therapy and understanding.
3. Reassess inhaler technique.
4. Ensure understanding of withdrawal of acute medications (steroids and/or antibiotics).
5. Assess need for continuing any oxygen therapy.
6. Provide management plan for comorbidities and follow-up.
7. Ensure follow-up arrangements: early follow-up < 4 weeks, and late follow-up < 12 weeks as indicated.
8. All clinical or investigational abnormalities have been identified.

1 – 4 Weeks Follow-up

- Evaluate ability to cope in his/her usual environment
- Review and understanding treatment regimen
- Reassessment of inhaler techniques
- Reassess need for long-term oxygen
- Document the capacity to do physical activity and consider patient eligibility to be enrolled in pulmonary rehabilitation
- Document symptoms: CAT or mMRC
- Determine status of comorbidities

12 – 16 Weeks Follow-up

- Evaluate ability to cope in his/her usual environment
- Review understanding treatment regimen
- Reassessment of inhaler techniques
- Reassess need for long-term oxygen
- Document the capacity to do physical activity and activities of daily living
- Measure spirometry: FEV1
- Document symptoms: CAT or mMRC
- Determine status of comorbidities

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Interventions that reduce the frequency of COPD exacerbations

Intervention Class	Intervention
Bronchodilators	LABAs LAMAs LABA + LAMA
Corticosteroid-containing regimens	LABA + ICS LABA + LAMA + ICS
Anti-inflammatory (non-steroid)	Roflumilast Dupilumab
Anti-infectives	Vaccines Long Term Macrolides
Mucoregulators	N-acetylcysteine Carbocysteine Erdosteine
Various others	Smoking Cessation Rehabilitation Lung Volume Reduction Vitamin D Shielding measures (e.g., mask wearing, minimizing social contact, frequent hand washing)

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Treatable traits in pulmonary hypertension-COPD (PH-COPD) and suggested management

COPD and PAH (Group 1 PH)	• Treat as PAH with comorbidity according to 2022 ESC/ERS PH guidelines
COPD and CTEPH (Group 4 PH)	• Treat as CTEPH according to 2022 ESC/ERS PH guidelines
COPD and severe PH associated with lung diseases and/or hypoxia (Group 3 PH)	• Individualized treatment approach in PH center with experience in respiratory diseases

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Common Risk Factors for Development of Lung Cancer

- Age > 55
- Smoking history > 30 pack years
- Presence of emphysema by CT scan
- Presence of airflow limitation FEV1/FVC < 0.7
- BMI < 25 kg/m^2
- Family history of lung cancer

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COPD – Take Home Messages

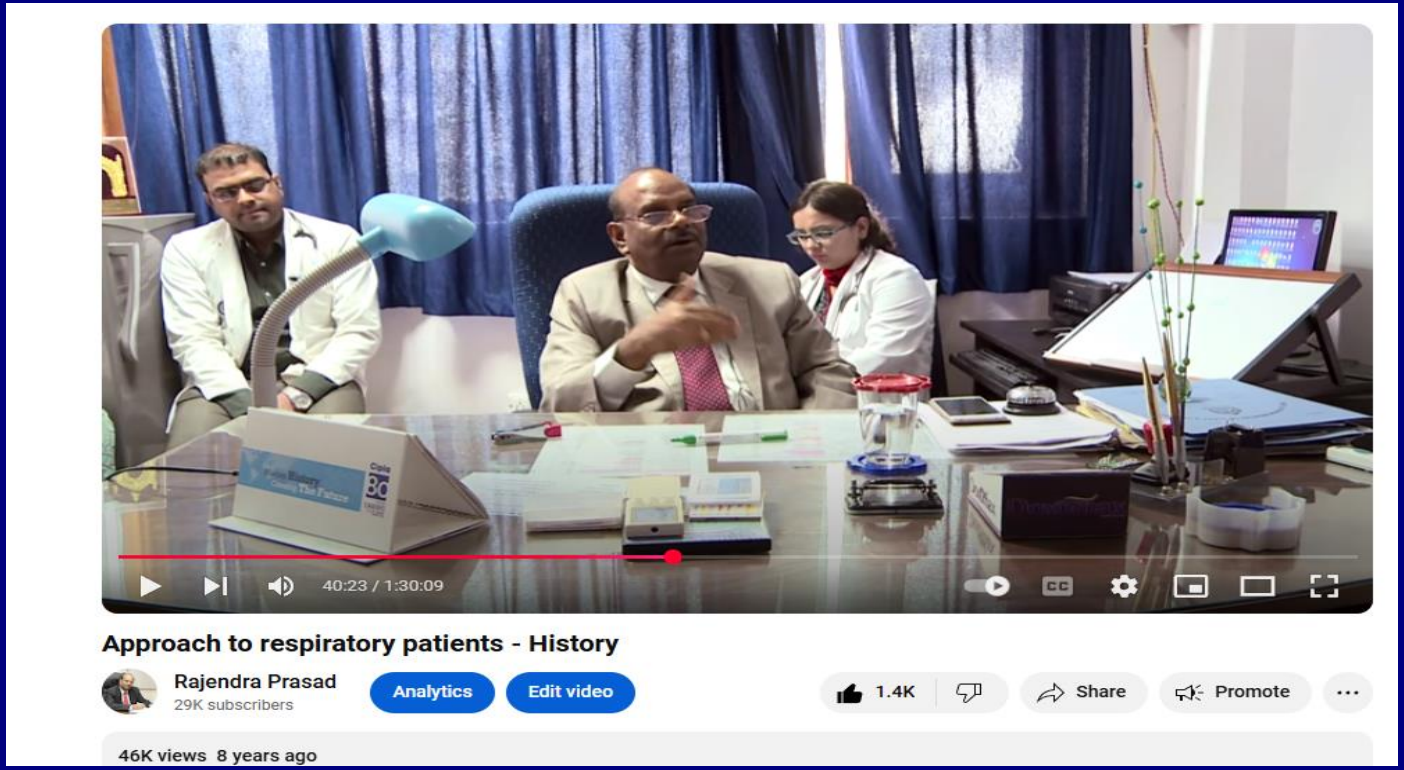
- Major cause of chronic morbidity & mortality
- Progressive disease, largely preventable, but marginally treatable disease
- Perform spirometry in patient having risk factors for COPD and suggestive symptoms
- Perform additional investigation to rule out alternate diagnosis
- Assessment of COPD requires assessment of symptoms, degree of airflow limitation, risk of exacacerbations and comorbidities.

COPD – Take Home Messages

- Combined assessment is the basis for non-pharmacologic & pharmacologic management of COPD.
- Treat COPD exacerbations to minimize their impact and to prevent the development of subsequent exacerbations
- Smoking Cessation is the single most effective measure to prevent onset and progression of COPD

Approach To Respiratory Patients - History

https://youtu.be/iGdcPHZdjvk



Approach To Respiratory Patients Physical Examination

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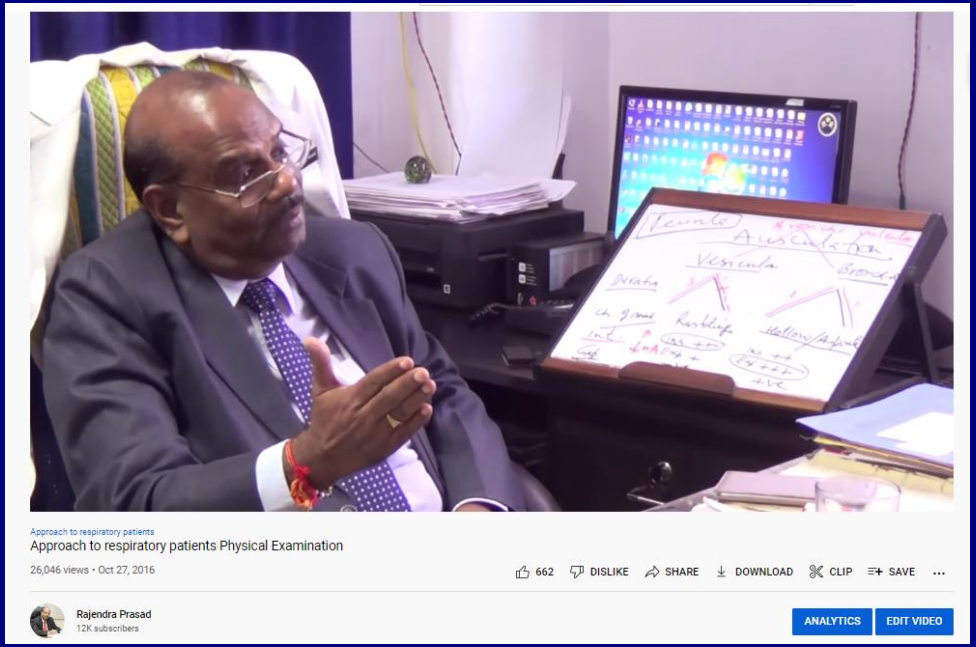

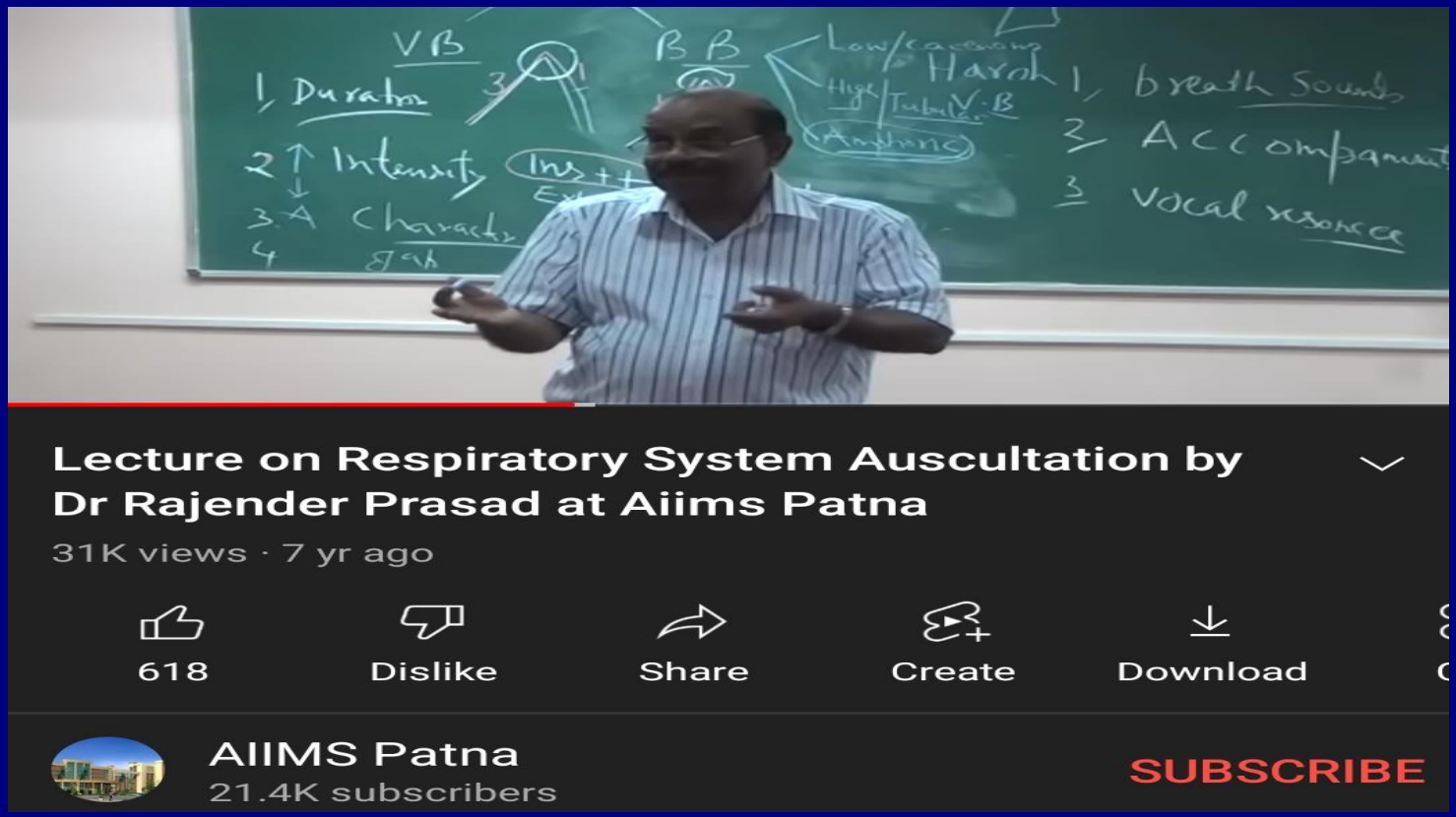

https://youtu.be/VYU3M6aYIxU

Chest X Ray interpretation for U.G. Students in Hindi

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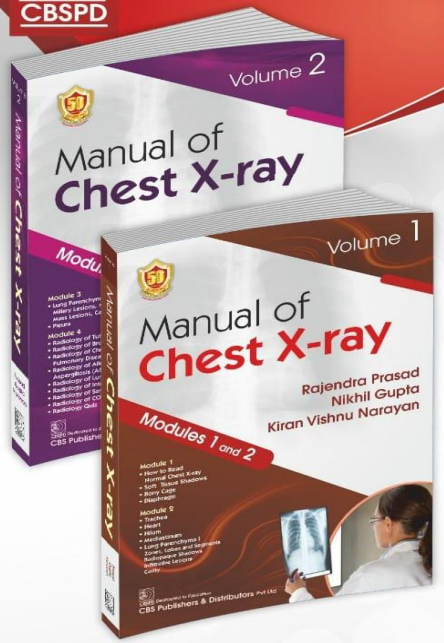

Volumes 1 and 2

Manual of Chest X-ray

Authors:

Rajendra Prasad
Nikhil Gupta
Kiran Vishnu Narayan

Salient Features

- A comprehensive manual on chest X-ray covering all the aspects of chest X-ray interpretation.
- Covers systemic reading of normal chest X-ray and various lesions in soft tissue, bony cage, diaphragm, hilum, heart, mediastinum and all the lesions of lung parenchyma and pleura.
- All chapters are organized in a systematic way for easy understanding.
- Available in print and *e*-book versions with text divided into four modules—two volumes.
- Modules and Volumes
  - Module 1 comprises reading normal chest X-rays and lesions of soft tissues, bony cage and diaphragm.
  - Module 2 comprises lesions of heart, mediastinum, hilum, cavity and infiltrations.
  - Module 3 comprises miliary, nodular, mass, calcified and various pleural lesions.
  - Module 4 comprises various chest X-ray findings of tuberculosis, bronchiectasis, COPD, sarcoidosis, interstitial lung diseases, lung cancer, allergic bronchopulmonary aspergillosis and Covid 19.
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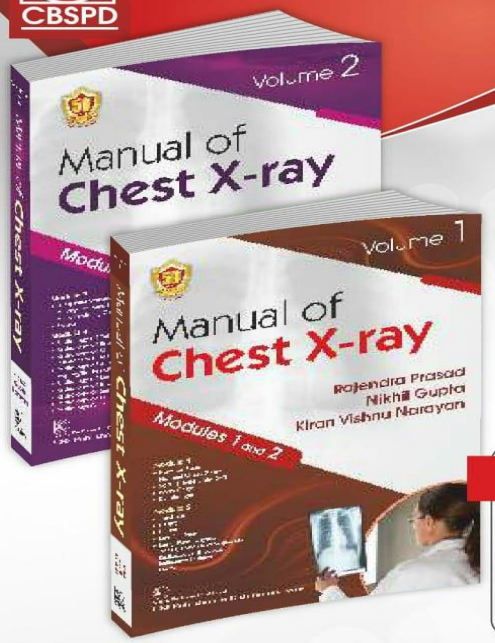

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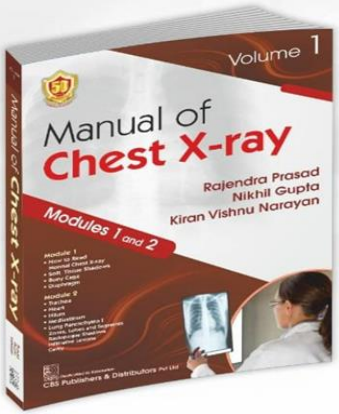

Module 1

- How to Read Normal Chest X-ray
- Soft Tissue Shadows
- Bony Cage
- Diaphragm

Module 2

- Trachea
- Heart
- Hilum
- Mediastinum
- Lung Parenchyma I
  Zones, Lobes and Segments
  Radiopaque Shadows
  Infiltrative Lesions
  Cavity

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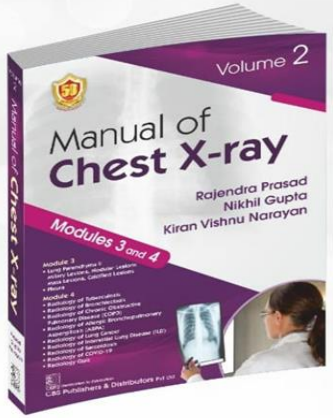

Contents of Volume 2

Module 3

- Lung Parenchyma II
  Miliary Lesions, Nodular Lesions
  Mass Lesions, Calcified Lesions
- Pleura

Module 4

- Radiology of Tuberculosis
- Radiology of Bronchiectasis
- Radiology of Chronic Obstructive Pulmonary Disease (COPD)
- Radiology of Allergic Bronchopulmonary Aspergillosis (ABPA)
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- Radiology of Interstitial Lung Disease (ILD)
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- Radiology of COVID-19
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Manual *of* TUBERCULOSIS

This book "Manual of Tuberculosis" is written with the aim of defining a practical approach to every aspect of tuberculosis. Chapters practically covers all the aspects of tuberculosis ranging from epidemiology, diagnosis and practical approach to the treatment of pulmonary as well as common extra-pulmonary tuberculosis including HIV and TB. It also covers practical approach to drug resistant including multidrug resistant tuberculosis and extensively drug resistant tuberculosis. Chapters also include prevention of tuberculosis and TB control in India. Pharmacokinetics of antituberculosis used in new and retreatment cases of tuberculosis including their doses, regimens and adverse effects are highlighted. Special chapters on case based approach to treatment of tuberculosis in special situation and MDR-TB have also been included. Chapters have been written in the background of current literature and practical experience gained from day to day dealing with different patients suffering from tuberculosis. Undergraduate, Postgraduate medical students, practitioners and program manager in TB control will find this book as practical guide.

Prof. Rajendra Prasad MD DTCD FAMS FCCP (USA) FNCCP FCAI FIAB FIMSA DSC (Honoris Causa) Director, Vallabhbhai Patel Chest Institute, University of Delhi, Delhi (India), Former Professor & Head, Department of Pulmonary Medicine, King George's Medical University, Lucknow and Former Director, UP Rural institute of Medical Sciences & Research, Saifai, Etawah, did his MBBS in 1974 & MD in 1979 from King. George's. Medical College, Lucknow. He received advance training in Pulmonary Medicine including clinical tuberculosis and TB control from Japan. He is also honorary consultant to Armed Forces Medical Services, India in Respiratory Diseases. Professor Prasad is currently Vice President of South Asia Association of Allergy, Asthma and Applied Immunology. He has been International Governor of American College of Chest Physicians (USA). He has unique distinction of being president of all major scientific bodies in the field of Pulmonary Medicine in India. Besides several prestigious fellowship of reputed National and International organization, he was awarded Fellowship of the National Academy of Medical Sciences India. He has supervised about 150 Researches and Published 225 Articles in reputed National and International Journals and books. He has presented over 1200 guest lectures, scientific papers at various National and International meetings.

Prof. Rajendra Prasad is a nationally acclaimed chest physician and tuberculosis expert, possessing nearly 4 decades' illustrious teaching and research experience with proven excellence in quality patient care. Apart from being a clinician par excellence, he is also a very popular medical teacher in Pulmonary Medicine. Prof. Prasad's contribution in the field of Tuberculosis and Multidrug Resistant Tuberculosis (MDR-TB) are widely acclaimed. He took keen interest in Revised National Tuberculosis Control Programme (RNTCP) from its inception. His dynamic leadership in academic and administrative areas has earned him a large number of awards from various International and National Scientific societies. He has been mentor of many students who are now assuming important positions in pulmonary medicine.

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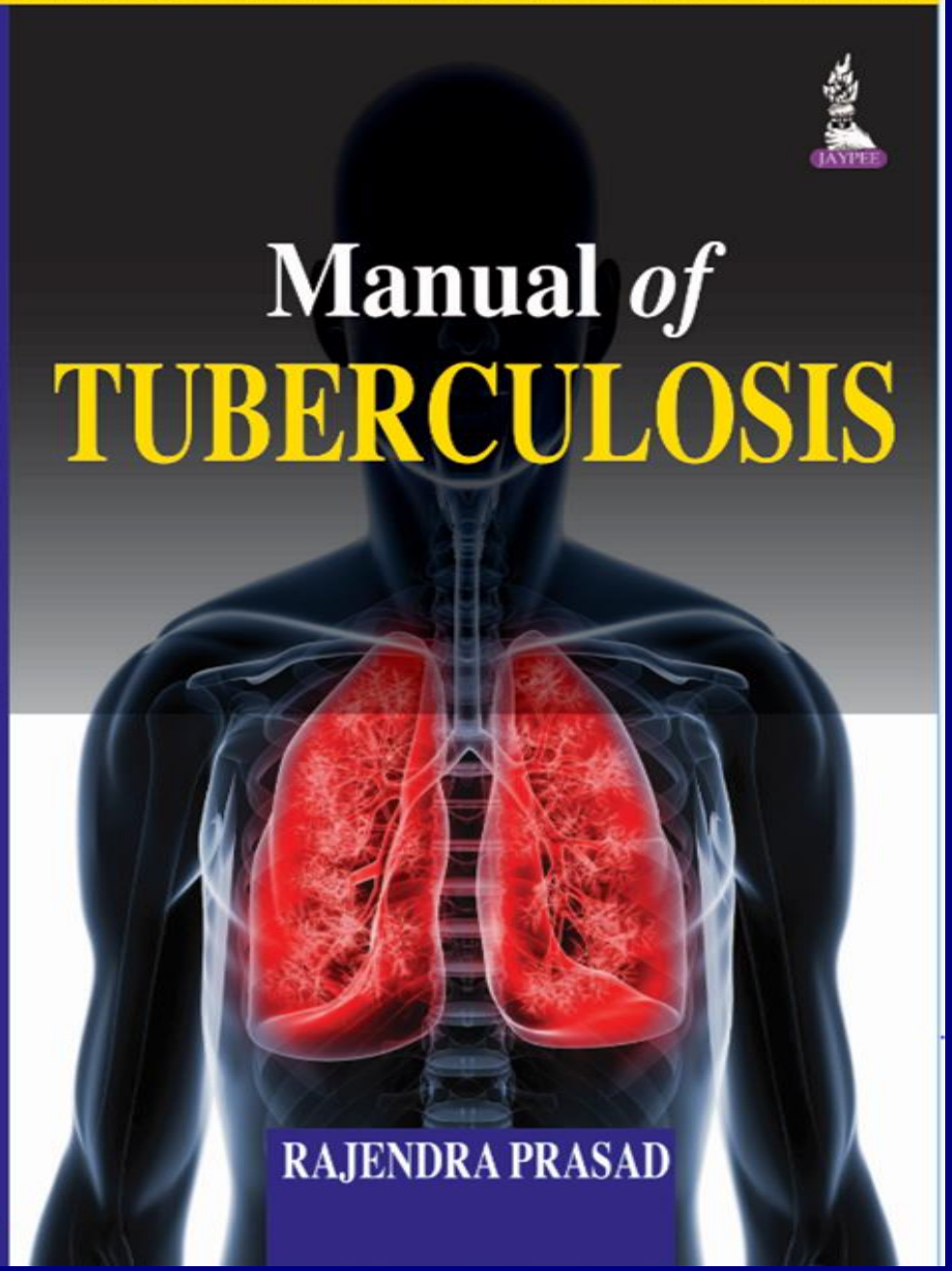

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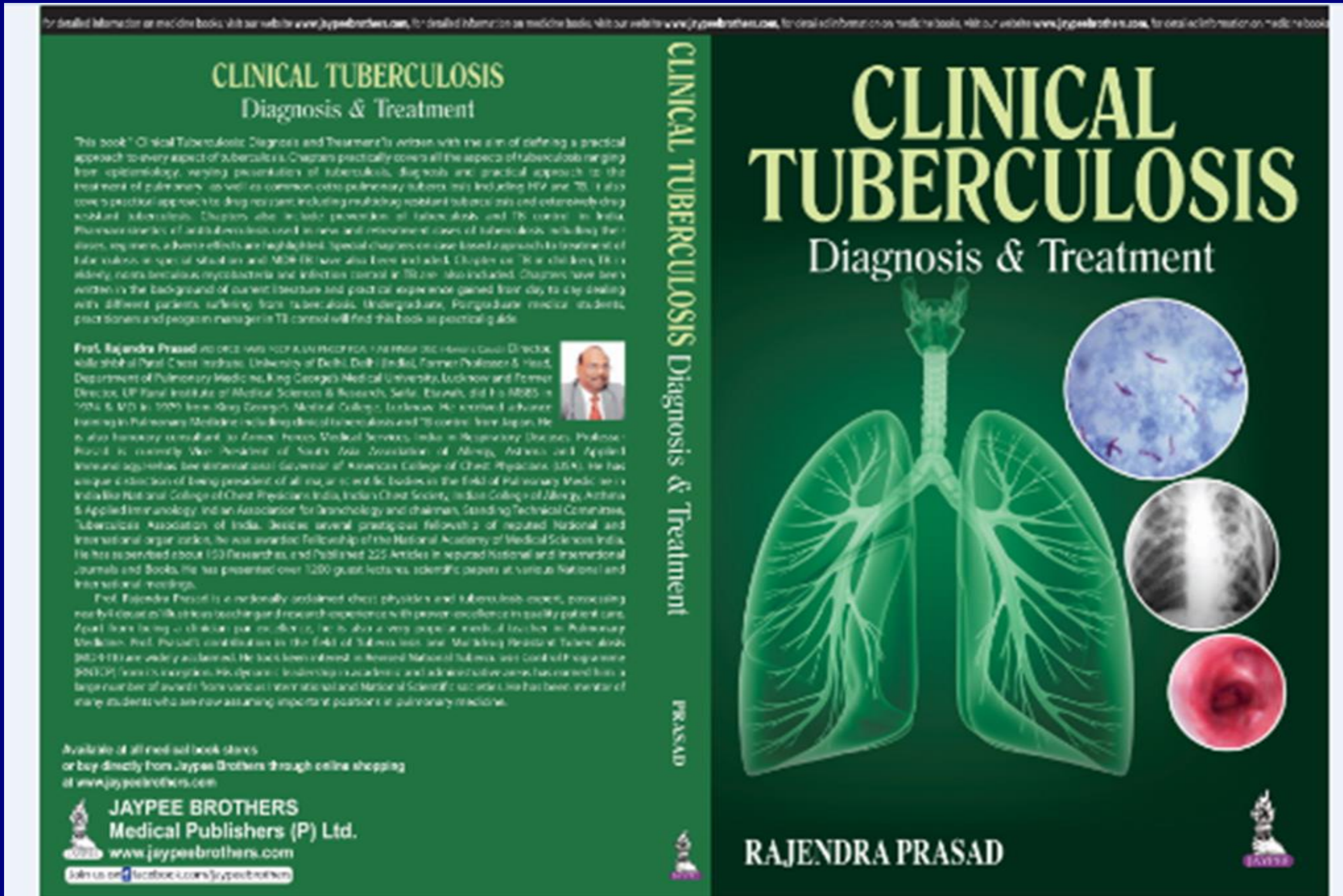
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Prof. Rajendra Prasad MD DTCD FAMS FCCP (USA) FNCCP FCAI FIAB FIMSA DSC (Honoris Causa) Director, Vallabhbhai Patel Chest Institute, University of Delhi, Delhi (India), Former Professor & Head, Department of Pulmonary Medicine, King George's Medical University, Lucknow and Former Director, UP Rural institute of Medical Sciences & Research, Saifai, Etawah, did his MBBS in 1974 & MD in 1979 from King George's Medical College, Lucknow. He received advance training in Pulmonary Medicine including clinical tuberculosis and TB control fromJapan. He is also honorary consultant to Armed Forces Medical Services, India in Respiratory Diseases. He has beeninternational Governor of American College of Chest Physicians (USA). He has unique distinction of being president of all major scientific bodies in the field of Pulmonary Medicine in India like National College of Chest Physicians India, Indian Chest Society, Indian College of Allergy, Asthma & Applied Immunology, Indian Association for Bronchology and chairman, Standing Technical Committee, Tuberculosis Association of India. Besides several prestigious fellowship of reputed National and International organization, he was awarded Fellowship of the National Academy of Medical Sciences India. He has supervised about 150 Researches, and Published 225 Articles in reputed National and International Journals and Books. He has presented over 1200 guest lectures, scientific papers at various National and International meetings.

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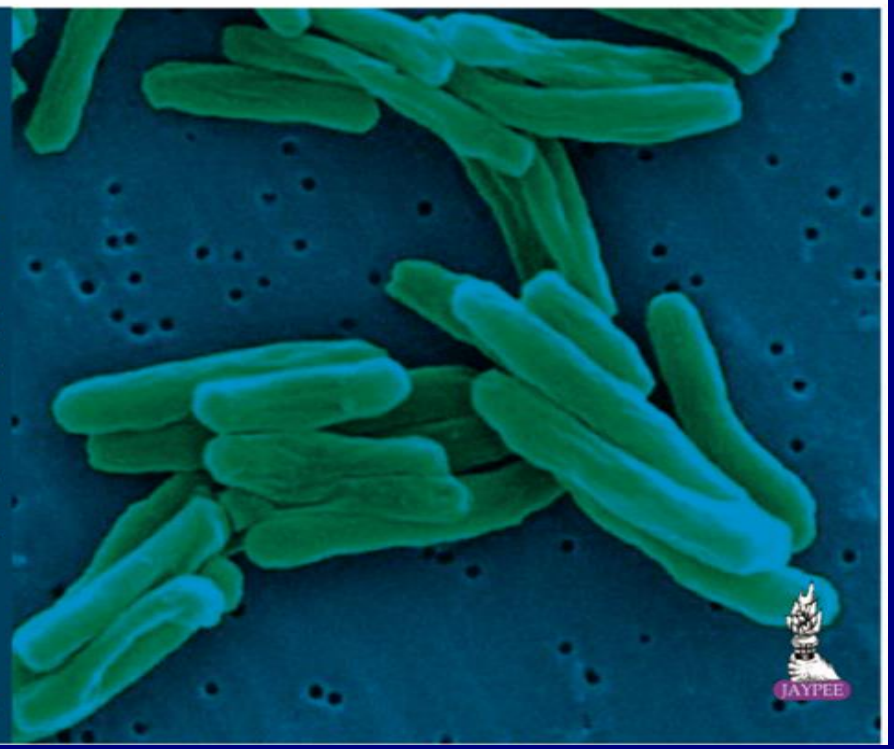

Handbook on
Adverse DRUG REACTIONS in TB Treatment

Salient Features

- Comprehensive well-referenced handbook, which contains a plethora of knowledge
- Defines a practical approach to every aspect of adverse drug reactions in tuberculosis treatment
- Covers all the aspects ranging from epidemiology of adverse drug reactions in new and drug-resistant patients
- Includes the case-based approach to treatment of tuberculosis, multi-drug-resistant tuberculosis (MDR-TB) and extensively drug-resistant tuberculosis (XDR-TB) in special situations such as pregnancy, renal insufficiency and liver diseases
- Chapters are organized in a systematic way for easy understanding and for practical approach with illustrative cases
- Serves as a practical guide for undergraduate and postgraduate medical students, practitioners, program managers and healthcare workers in TB control.

Rajendra Prasad MD DTCD FAMS FCCP (USA) FRCP (Glas) FNCCP FICS FCAI FIAB FIMSA FCCS DSc (Honoris Causa) is the Director of Medical Education and Professor and Head, Department of Pulmonary Medicine, Era's Lucknow Medical College and Hospital, Era University, Lucknow, Uttar Pradesh, India. He was the Director, Vallabhbhai Patel Chest Institute, University of Delhi, New Delhi; Professor and Head, Department of Pulmonary Medicine, King George's Medical University, Lucknow; and the Director, UP Rural Institute of Medical Sciences and Research, Saifai, Etawah, Uttar Pradesh. He has been International Governor of American College of Chest Physicians, USA. He has unique distinction of being President of all major scientific bodies in the field of pulmonary medicine in India. He was awarded Fellowship of the National Academy of Medical Sciences, India, American College of Chest Physicians, USA and Royal College of Physicians and Surgeons, Glasgow. He has supervised about 180 researches, and published 340 original articles, reviews and book chapters. He has written 8 books including 4 books on Tuberculosis and an Atlas on Fiber Optic Bronchoscopy based exclusively on Indian patients and presented over 1,600 guest lectures and scientific papers at various national and international meetings. He is recipient of Dr BC Roy National Award for devolving and popularizing pulmonary medicine in India.

Nikhil Gupta MD (Medicine) is an Assistant Professor, Department of General Medicine, Dr Ram Manohar Lohia Institute of Medical Sciences, Lucknow, Uttar Pradesh, India. He was an Assistant Professor, Department of Medicine, Era's Lucknow Medical College and Hospital, Era University, Lucknow from 2012 to 2017. He has more than 30 guest lectures and scientific papers, supervised 15 researches and published 35 original articles, review articles, case reports and book chapters. He has also co-authored 3 books on tuberculosis.

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Rajendra Prasad

Co-author
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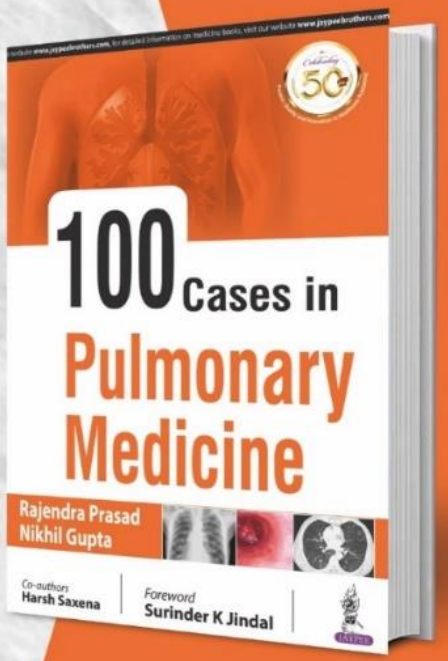

Key Features

- Includes interesting, challenging and educative 100 cases in Pulmonary Medicine
- Each case is followed with step by step approach to reach the final diagnosis and treatment
- Cases are dealt with lots of radiological images, making reading extremely interesting
- Book consists of lots of interesting cases on tuberculosis
- Each case report is followed by discussion of pathophysiology, clinical presentation, diagnosis, and treatment considering current evidence-based knowledge
- Also includes rare case such as pulmonary sequestration, alveolar microlithiasis etc, generally difficult to diagnose
- Best resource for both undergraduate and postgraduate medical students and consultants in pulmonary medicine.

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Questions

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