

COPD

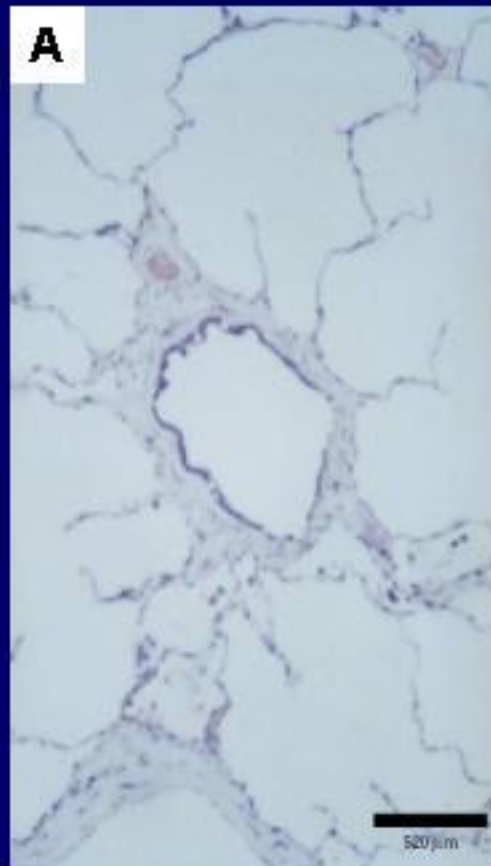
DAVID KANAREK MD

LEARNING OBJECTIVES

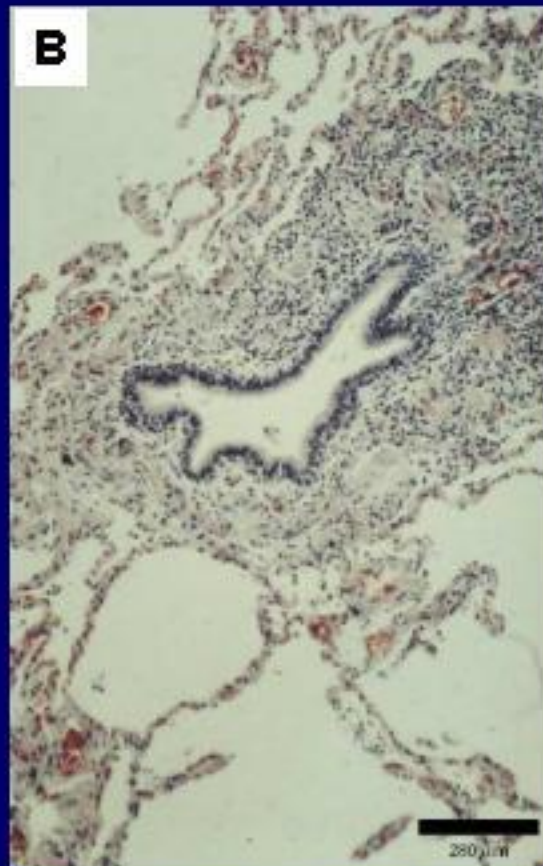
- 1. Pathology of COPD.**
- 2. Pharmacology**
- 3. Exacerbations**

Chronic obstructive pulmonary disease (COPD) is a common, preventable, and treatable disease that is characterised by persistent respiratory symptoms and airflow limitation due to airway and/alveolar abnormalities, usually caused by significant exposure to noxious particles or gases

Histopathologic Features of COPD



Normal



Obstructive Bronchiolitis



Emphysema

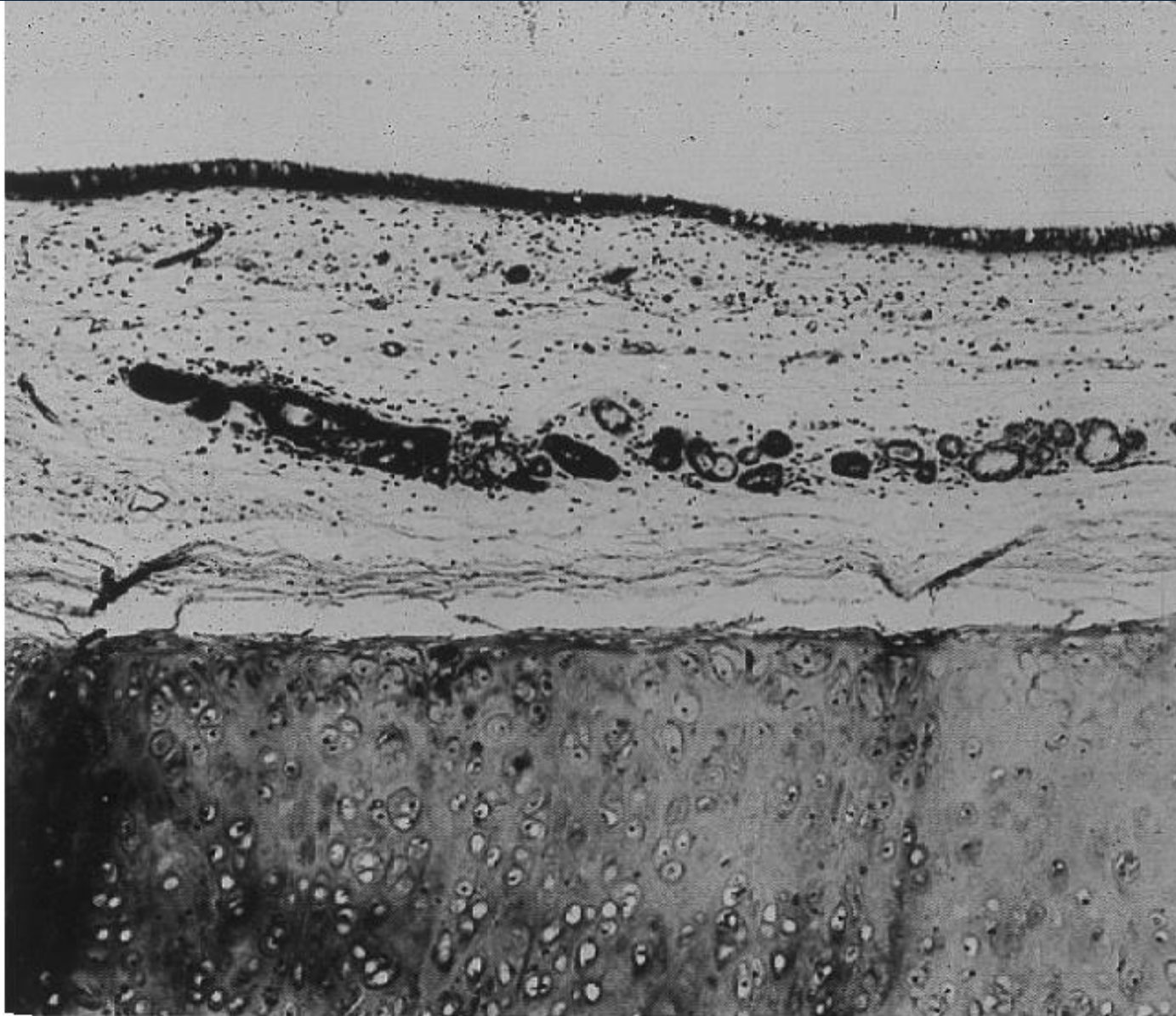
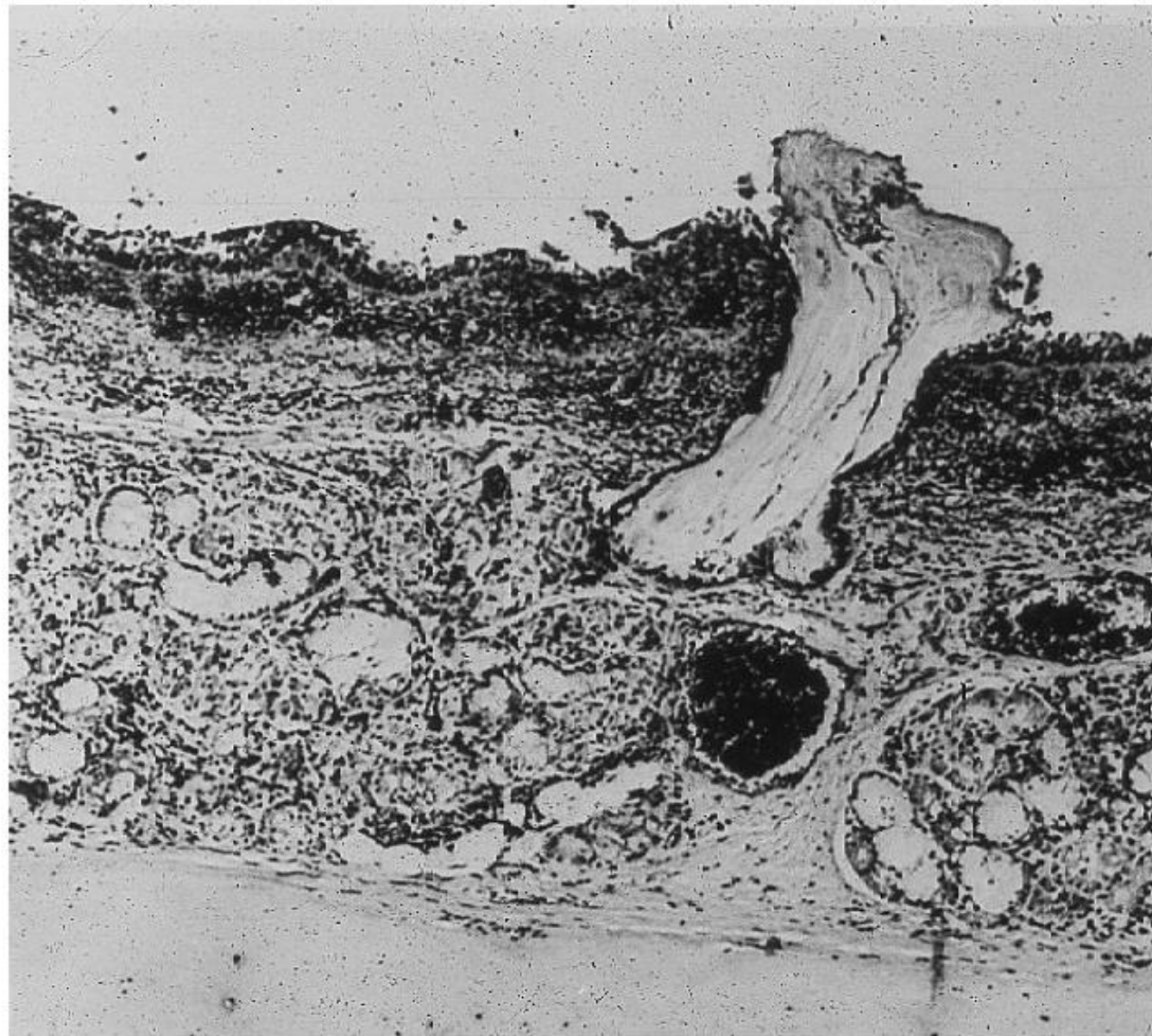
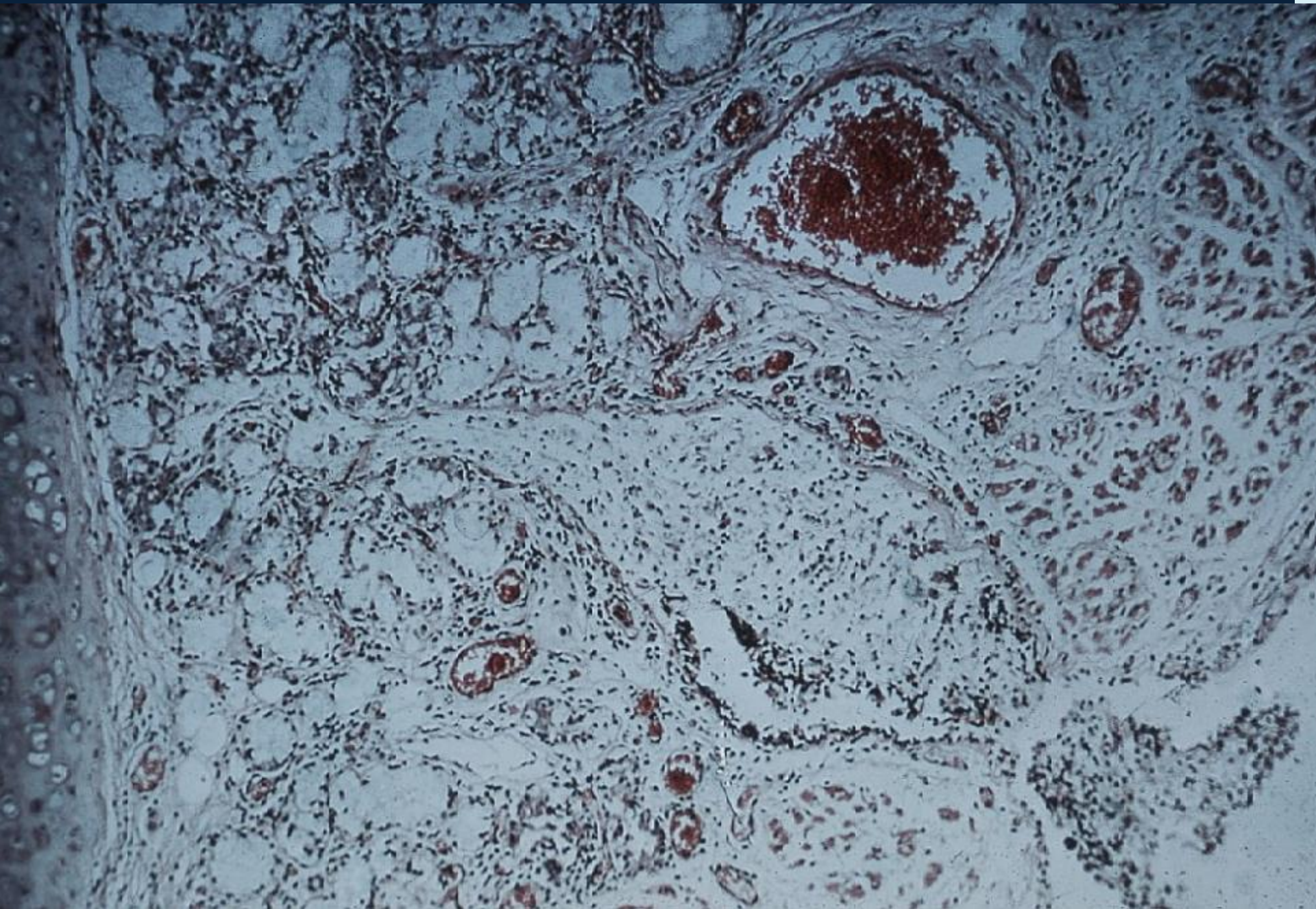
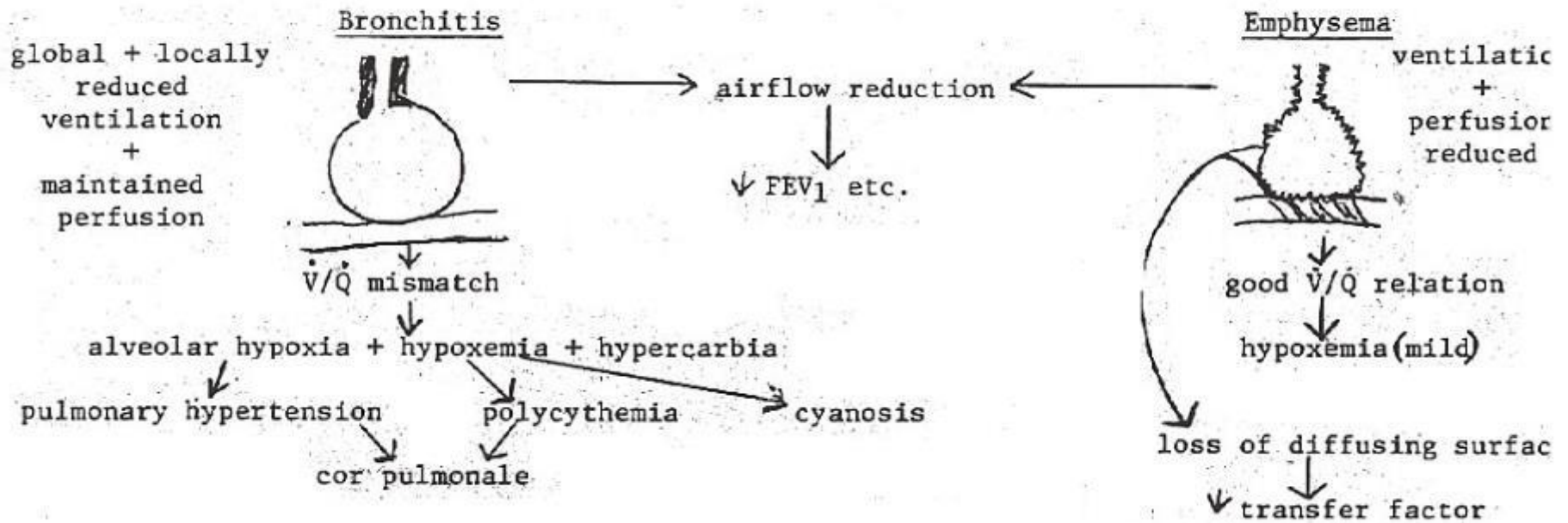


Fig. 4. Main stem bronchus from a nonbronchitic. The glands are small and form only a small part of the wall (basement membrane to perichondrium) thickness. The Reid Index is therefore small. H&E. $\times 80$.

Fig. 5. Marked mucous gland hyperplasia. The mucous glands are greatly enlarged and the Reid Index is high. Note the duct of a bronchial gland filled with mucus. H&E. $\times 80$.

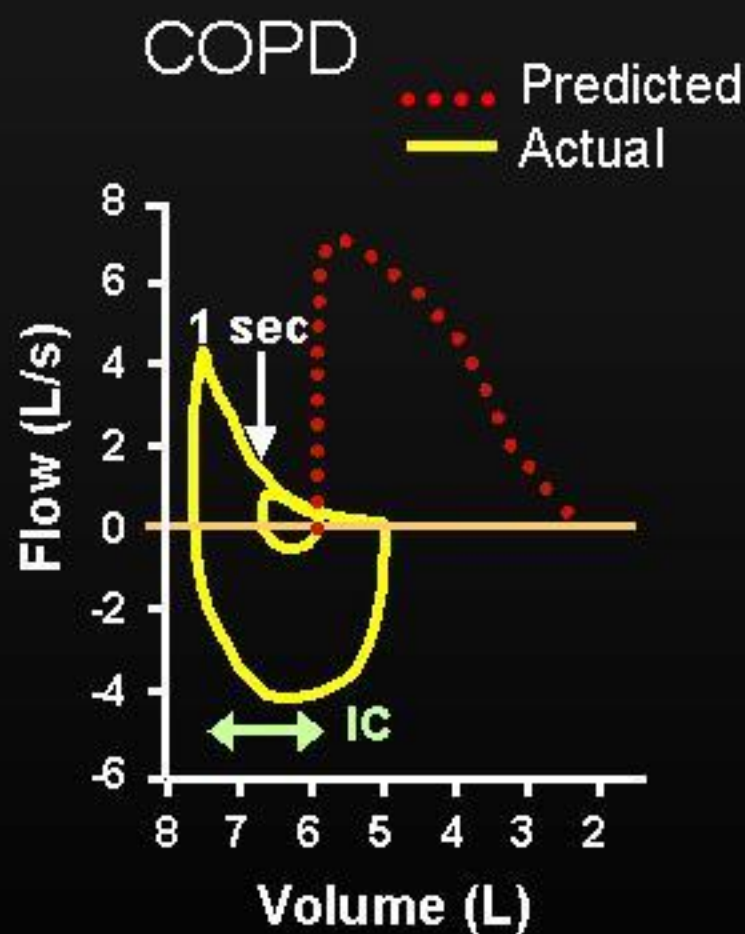
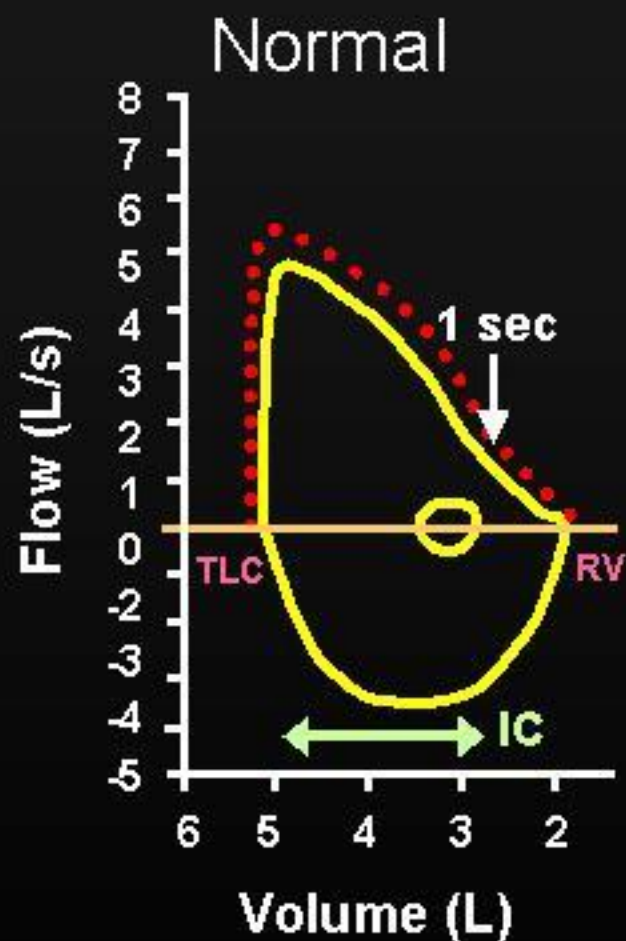








Differences in Flow Volume Loops in Normal Function vs COPD







Spirometry is required to make the diagnosis of COPD

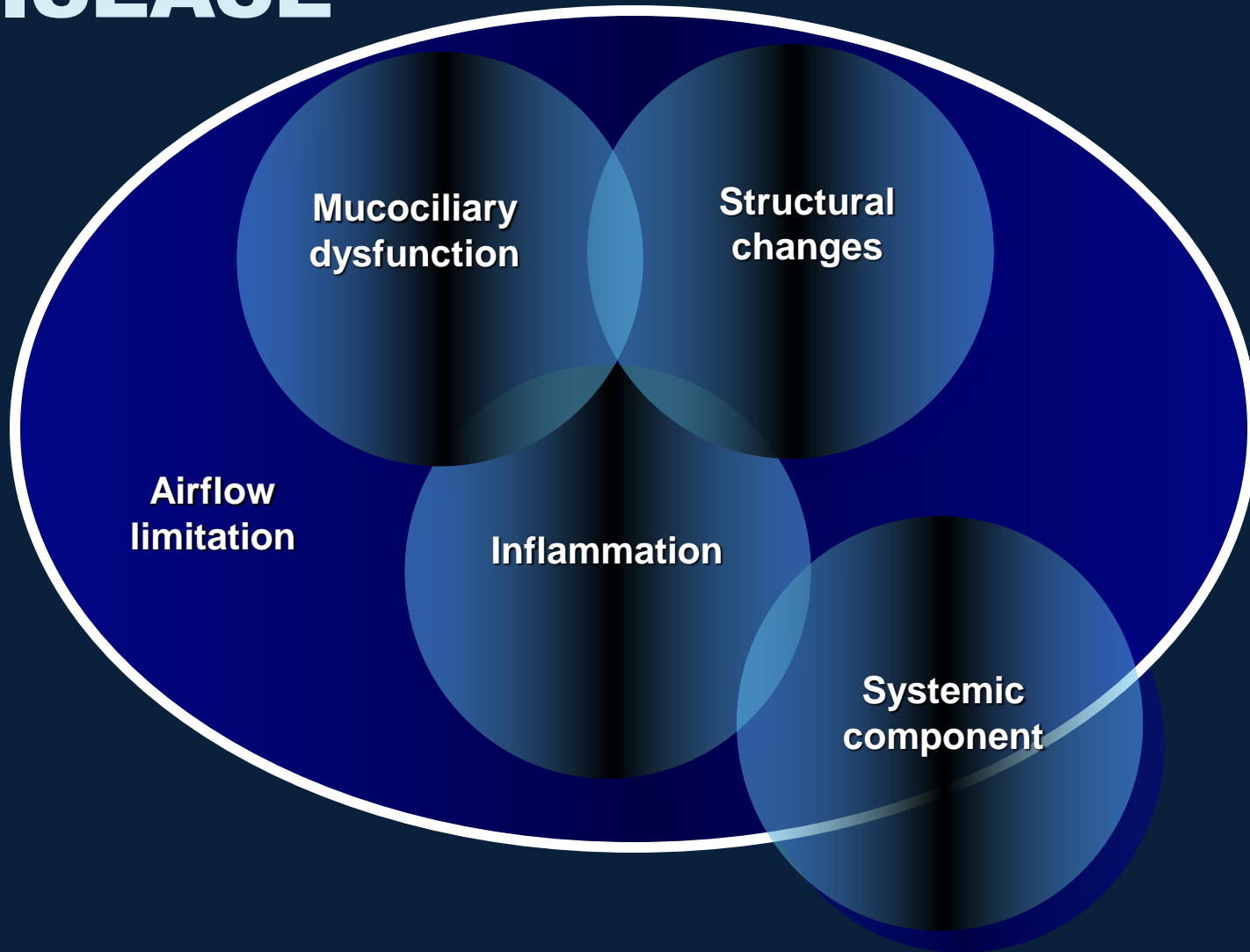
A post bronchodilator FEV₁/FVC less than 0.70 confirms the presence of COPD

Risk Factors for COPD Include Environmental Exposures and Genetic Factors

- ▲ Tobacco smoke is the major cause of COPD (80% to 90%)¹
- ▲ Other environmental factors include occupational dusts and chemicals (vapors, irritants, fumes) and indoor/outdoor air pollution²
- ▲ The best characterized genetic factor is alpha₁-antitrypsin deficiency³

1. US Surgeon General. *Summary of the Health Consequences of Smoking: Chronic Obstructive Lung Disease*. Publication DHHS 84-50205.
2. Pauwels RA, et al. *Am J Respir Crit Care Med*. 2001;163:1256-1276.
3. Mahadeva R, Lomas DA. *Thorax*. 1998;53:501-505.

COPD IS A MULTICOMPONENT DISEASE



DIAGNOSTIC INDICATORS

Dyspnea –persistent, progressive, exercise related

Chronic cough

Chronic sputum

Recurrent respiratory infections

Risk factors

Family history/ childhood factors-low birthweight, infections

COPD MANAGEMENT

Assess and Monitor disease

Reduce Risk Factors

Manage Stable COPD

Manage Exacerbations

ASSESSMENT

SYMPTOMS - dyspnea, cough, sputum

EXACERBATION HISTORY

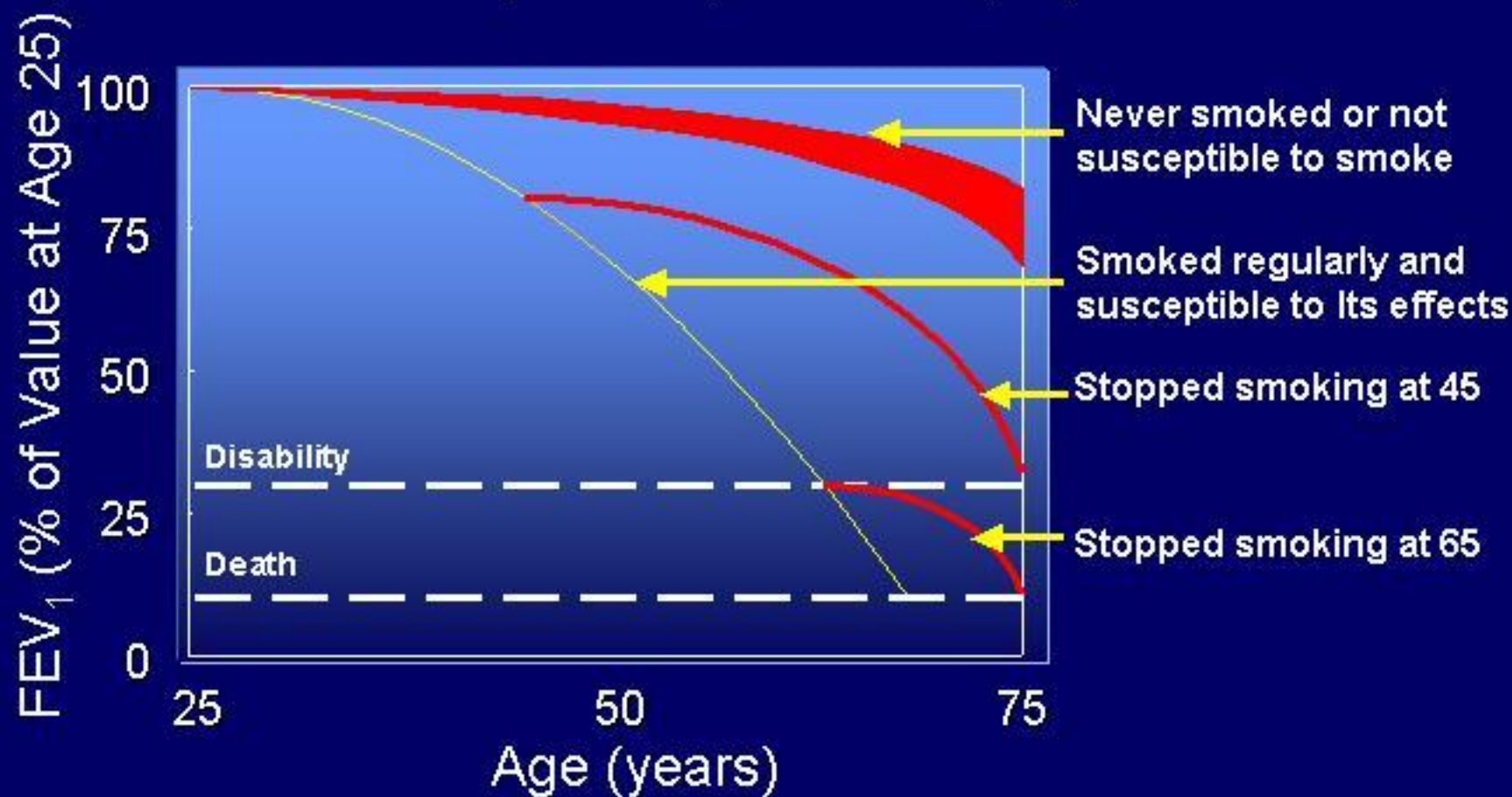
SPIROMETRY

MANAGEMENT

1. Quit smoking.
2. Pharmacotherapy.
3. Vaccinations.
4. Pulmonary rehabilitation
5. Oxygen therapy
6. Noninvasive ventilation
7. Surgical or bronchoscopic intervention

Smoking Cessation is the Single Most Important Way to Prevent the Onset and Progression of COPD

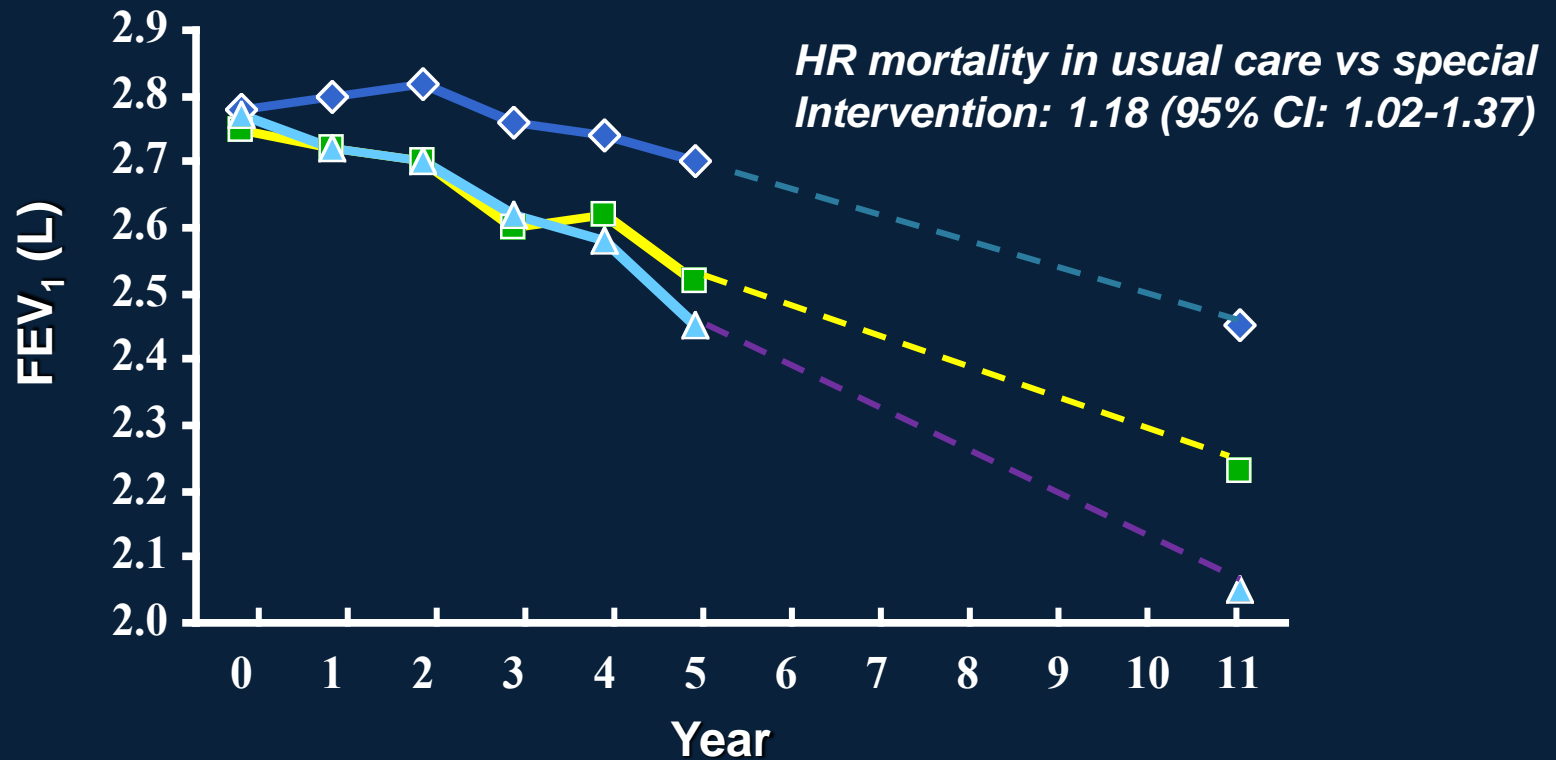
If exposure to noxious agents stops, disease progression slows



LUNG FUNCTION DECLINE IN MILD COPD

THE LUNG HEALTH STUDY AT 11 YEARS

◆ Sustained Quitters □ Intermittent Quitters ▲ Continuous Smokers



Reproduced with permission from Anthonisen et al. *Am J Respir Crit Care Med.* 2002;166:675-679; Calverley et al. *Lancet.* 2003;362:1053-1061; Anthonisen et al. *Ann Intern Med.* 2005;142:233-239.

PHARMACOTHERAPY

Beta agonists -short acting and long acting (SABA,LABA).

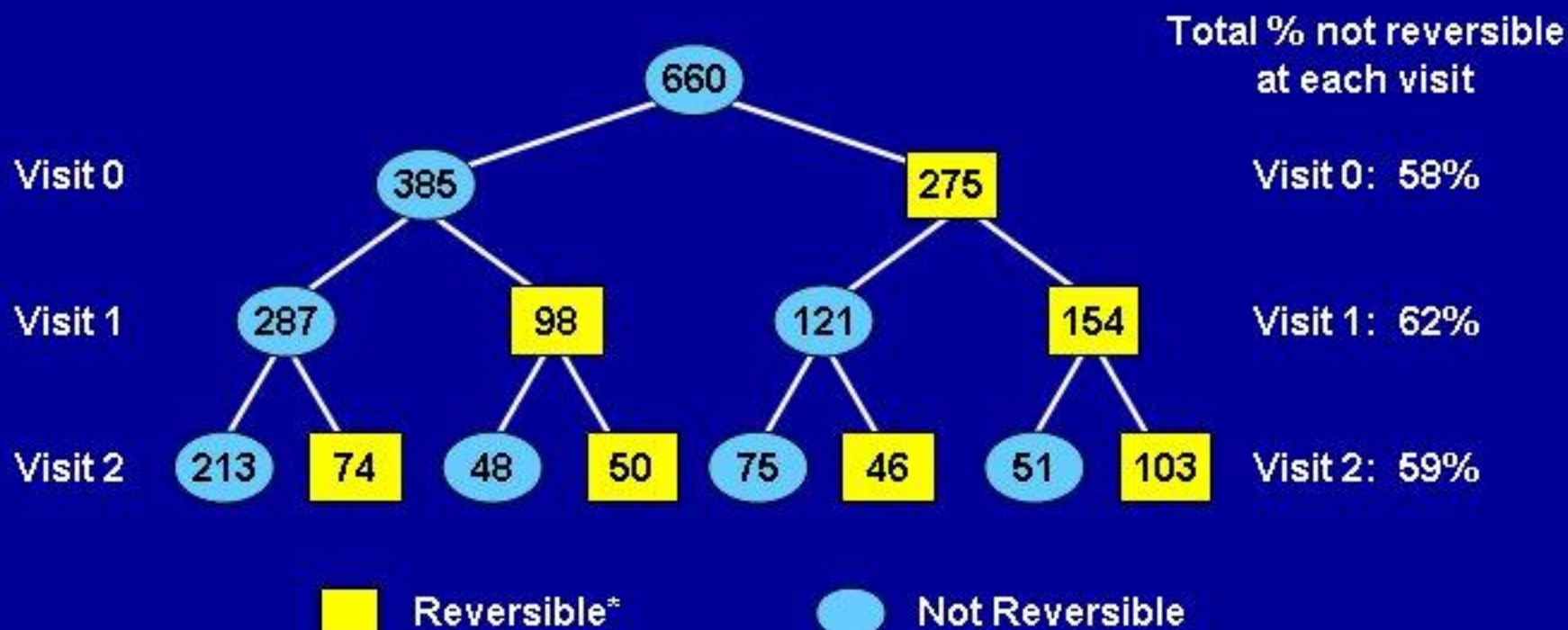
Anticholinergics-short acting and long acting(SAMA, LAMA).

Corticosteroids-inhaled(ICS) and oral.

Antibiotics-azithromycin and erythromycin reduce exacerbations.

PDE4 inhibitor-reduces exacerbations and improves lung function.

Changes in Responder Classification After Albuterol and Ipratropium Bromide



* Reversible defined as $\geq 12\%$ and 200-mL increase in FEV₁

PHARMACOTHERAPY

Combination therapy is better than monotherapy.

Short acting drugs improve symptoms and FEV1.

LABA and LAMA improve lung function, dyspnea and reduce exacerbations.

LAMA > LABA for exacerbation and hospitalization reduction.

ICS/LABA reduces exacerbations and improves lung function.

ICS increases risk of pneumonia especially in moderate to severe obstruction.

Triple therapy may be most helpful.

LONG-TERM OXYGEN FOR COPD: NOTT TRIAL

Entry criteria

- $\text{PaO}_2 \leq 55$ mm Hg
- $\text{PaO}_2 \leq 59$ mm Hg plus edema, $\text{HCT} \geq 55\%$, or
P pulmonale

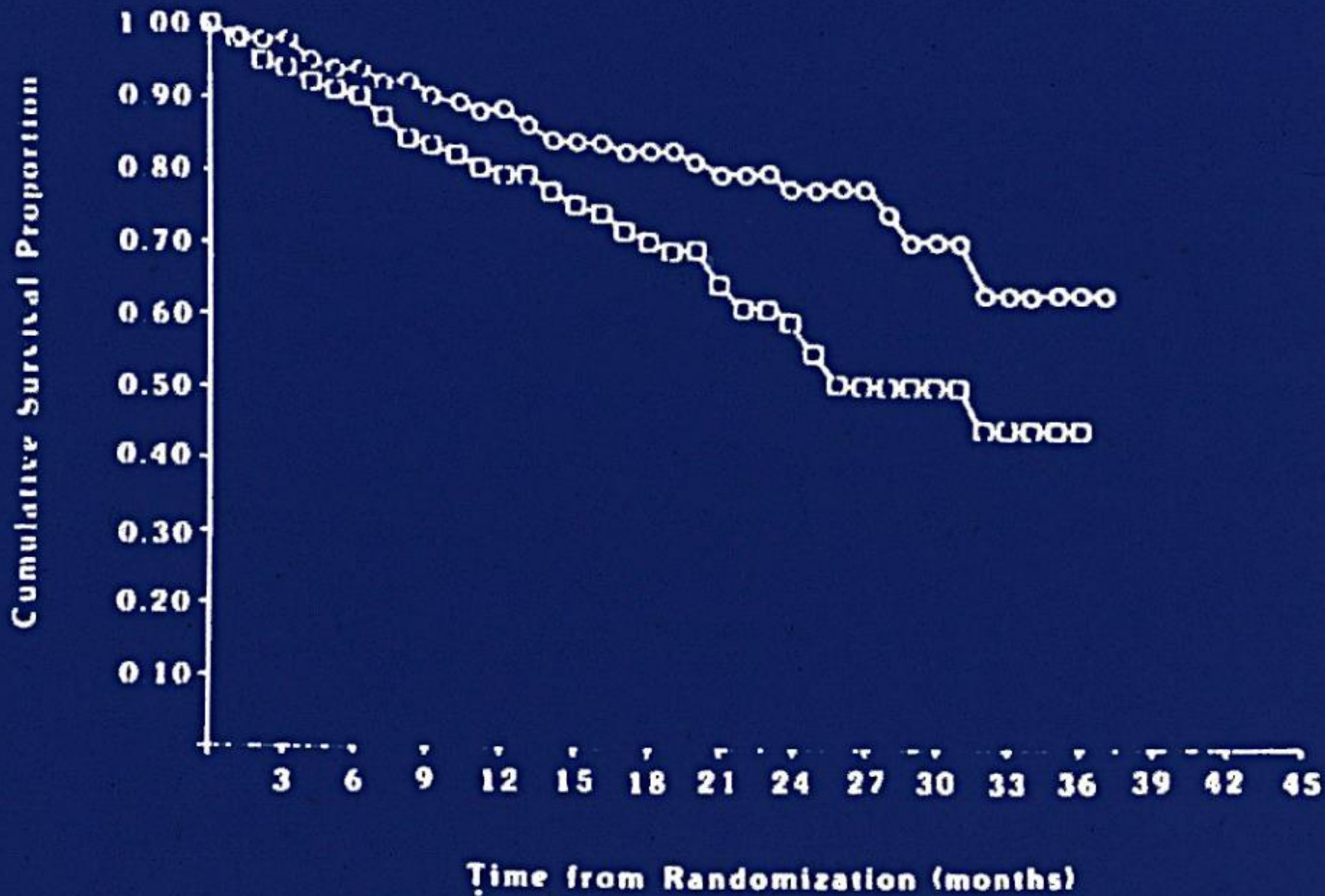
203 patients enrolled

Age >35 years

Mean $\text{FEV}_1/\text{FVC} < 70\%$ after inhaled bronchodilator

Mean baseline PO_2 51.5 mm Hg (in stable clinical state)

Intervention: 24 hours O_2 vs 12 hours O_2



OXYGEN THERAPY IN COPD

>15 hrs/day increases survival

Indicated for PO₂ <56mm Hg(O₂sat 88%) or PO₂ 60mm Hg (O₂sat 89%) with polycythemia, edema or pulmonary hypertension

Aim is to preserve vital organ function

Helps hemodynamics, exercise capacity, sleep, mental state, polycythemia

OXYGEN THERAPY

No sustained benefit in stable COPD and resting or exercise –induced moderate arterial desaturation

EXACERBATIONS OF COPD

Acute worsening of symptoms

They increase disease progression, hospital readmissions

Cause in 2/3 is viral infection, but also bacterial infection, GERD or pollution

At 8 weeks 20% have not recovered fully.

EXACERBATIONS

SABA's are the initial therapy. Can include SAMA's.

Oral steroids improve FEV1, oxygenation and shorten recovery time.

Prednisone 40mg daily 5-7 days.

Antibiotics- sputum purulence plus dyspnea or increased sputum volume

-mechanical ventilation

Maintenance therapy begun before discharge.

Oxygen

Ventilation-NIV is preferred as initial mode

Early followup < 30days.

EXACERBATIONS

- 1. THE BEST PREDICTOR OF FUTURE EXACERBATIONS IS
PREVIOUS TREATED EXACERBATIONS.**
- 2. EOSINOPHIL COUNT.**

PULMONARY REHABILITATION

Includes exercise, education, behavioral intervention and nutritional training

Significant improvement in dyspnea, exercise capacity, health status and healthcare utilisation

Guided by symptoms and functional capacity

Reduces readmissions and mortality after a recent exacerbation.

SURGICAL TREATMENT OF COPD

Bullectomy

Surgical Lung Volume Reduction

**Transplantation-FEV1 < 25%, PO2 <60mm Hg,
PCO2>50mmHg**

Endobronchial valves