COPD
AAPA 2021

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Disclosures

TEACHING
Idaho State University PA and NP Programs
ThriveAP

INDUSTRY AFFILIATIONS
Grifols Pharmaceutical - speaker, consultant
Boehringer Ingelheim Pharmaceuticals – consultant, speaker
Meda Pharmaceuticals – speaker, consultant
Circassia Pharmaceuticals – advisory panel
Genentech Pharmaceuticals - Speaker

CLINICAL RESEARCH
2017 – Sub-I, Genentech Zenyatta Severe Asthma Study
2016 – Sub-I, Biota Human Rhinovirus Study
2015 – Sub-I, Sanofi Traverse Severe Asthma Study
2015 – Sub-I, Sanofi Liberty Severe Asthma Study
2013 – Study Coordinator: MediVector Influenza Study

Brian Bizik does not intend to discuss the use of any off-label use/unapproved use of drugs or devices that he represents

Asthma vs COPD
Obstructive vs Restrictive

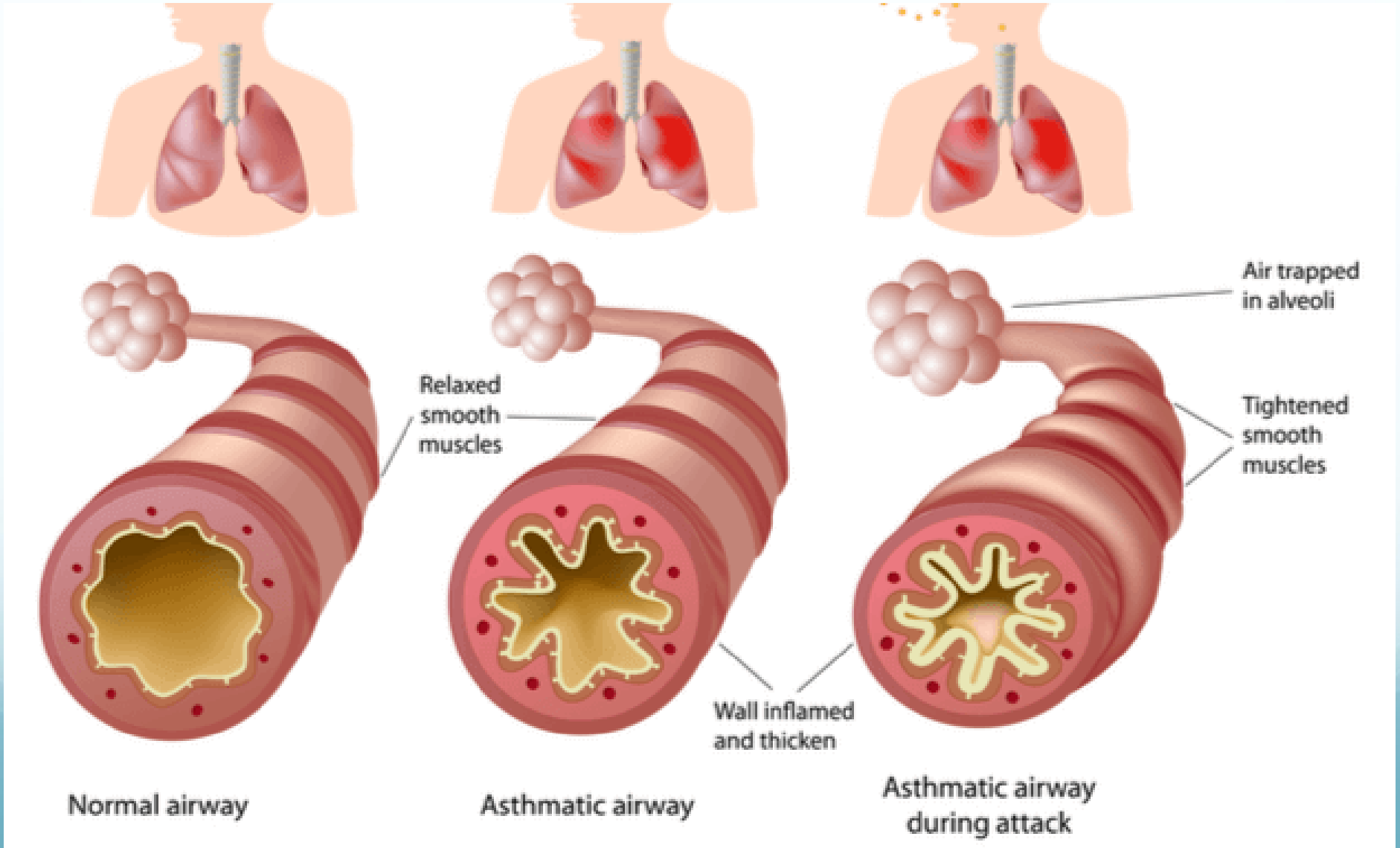
Asthma and COPD

We need to build the big picture first, before anything else. I need these two very different (but sometimes overlapping) diseases to be clear.

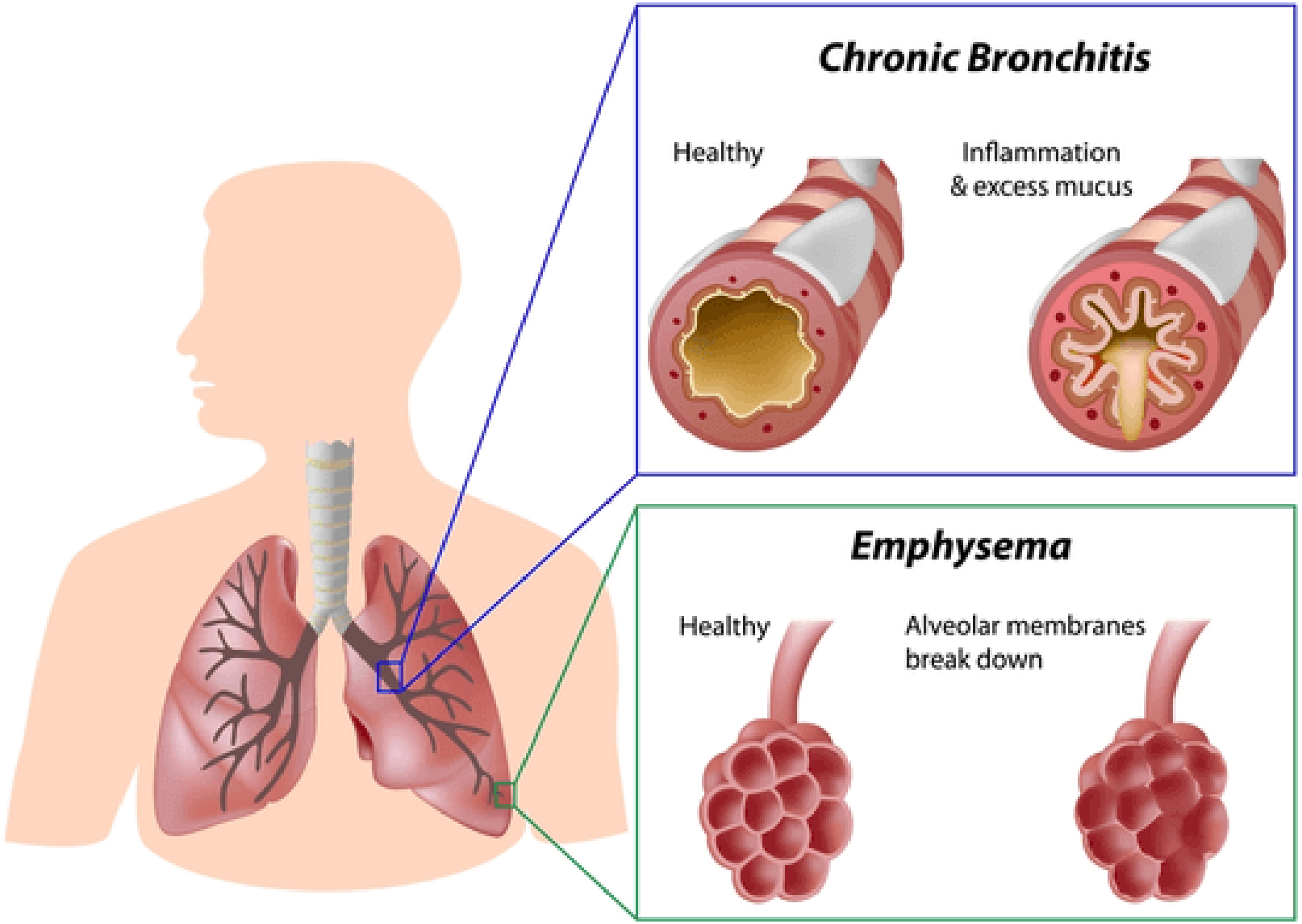
Asthma – bronchoconstriction, airway inflammation, mucous production

COPD – Tissue destruction, chronic cough, due to exposure

Asthma – Three key features: bronchoconstriction, airway inflammation and mucous production.



COPD – Think of the name. . Any thing chronic, that is obstructive, in the lungs and is terrible



Characteristics: COPD vs Asthma

	COPD	**Asthma**
Smoker, ex-smoker	Nearly all	possibly
Sympt onset <35 yo	Rare	common
Chronic productive cough	Common	uncommon
Breathlessness	Persistent, progressive	variable
Nocturnal awakening	Uncommon	common
Atopic, AR	Uncommon	common
Symptoms diurnal, variable day to day	Uncommon	common
Favorable response to ICS	inconsistent	consistent

Postma DS, Rabe KF. NEJM 2015;373:1241-1249

Some thoughts

Obstructive – limits movement of airflow in and/or of the lungs

Restrictive – limits expansion of lung and therefore total lung capacity, FVC reduced

What is a restrictive disease?

Site	Pathophysiology	Examples
Respiratory centres in brain	Reduced central respiratory drive	Cerebral trauma, drugs (e.g. opiates)
Nerves	Interruption of nerve supply to respiratory muscle	Polio, Guillain–Barré syndrome
Neuromuscular junctions	Reduced neuromuscular transmission	Myasthenia gravis
Respiratory muscles	Muscular weakness	Muscular dystrophy
Thoracic cage	Deformity or limitation of movement	Kyphoscoliosis, severe obesity
Pleural cavities	Reduced volume within pleural cavities	Pleural effusion, pneumothorax

OK Big picture - - -

Asthma – the big three

COPD – exposure, tissue destruction

OK – COPD!

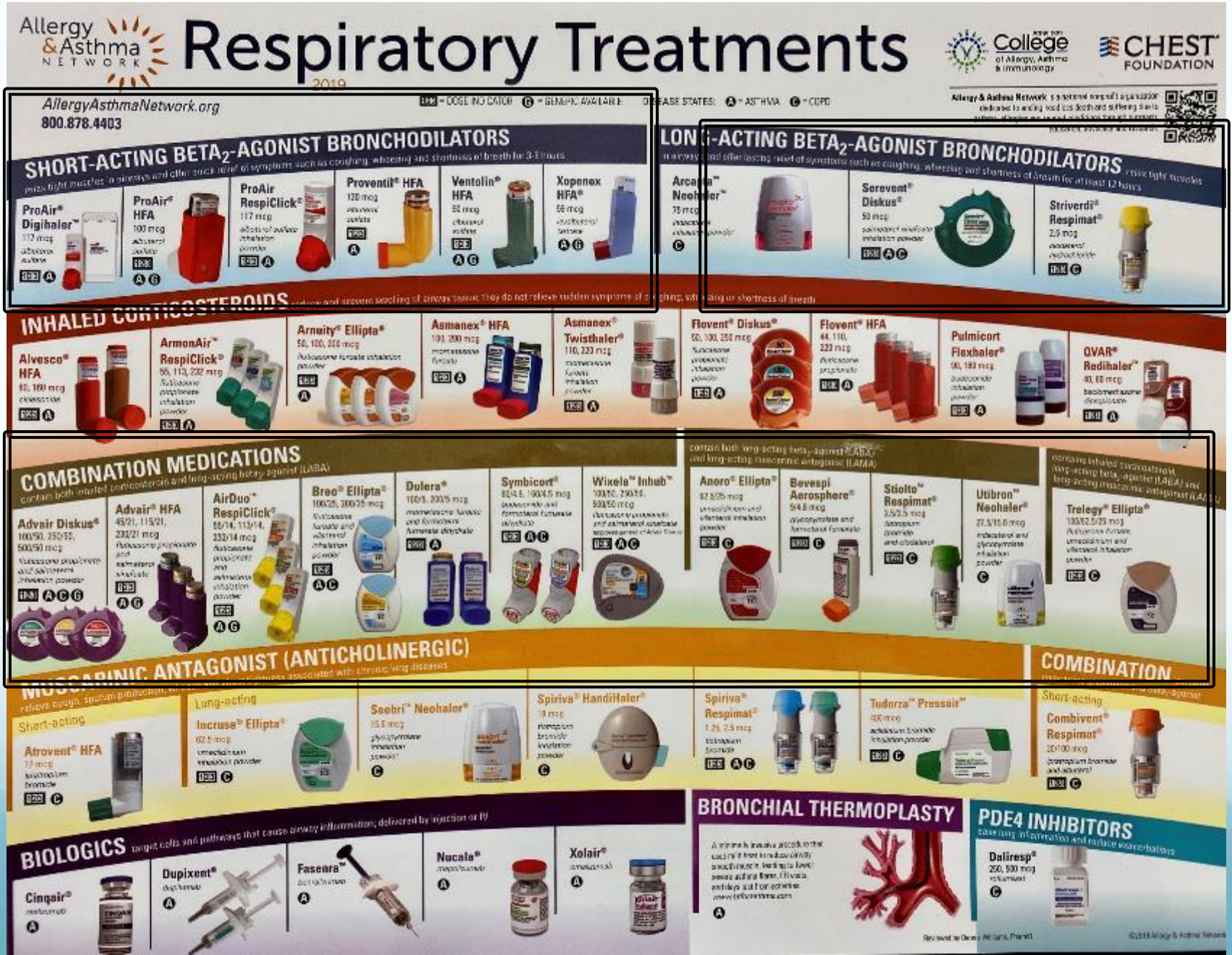
COPD Terms/Actions/Inhaler Types

- SABA = Short Acting Beta-Agonist = Albuterol = rescue inhaler = puffer, Proair, Ventolin, Proventil
- LABA = Long Acting Beta-Agonist, Serevent, Salmeterol
- ICS = Inhaled Corticosteroid, Flovent, fluticasone, QVAR, Pulmicort
- LAMA = Long Acting Muscarinic Antagonist, Spiriva, tiotropium
- MDI = Metered Dose Inhaler
- DPI = Dry Powdered Inhaler – Advair, Breo, Trelegy

COPD Terms/Actions/Inhaler Types

Albuterol – short acting bronchodilator, relaxes smooth muscle. Binds to beta receptors on smooth muscle, causing about a billion things to happen that drop the calcium in the cell and it relaxes.

Salmeterol/formoterol/vilanterol – Same thing as above but lasts 12 or 24 hours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DPI = DOSE INDICATOR
G = GENERIC AVAILABLE
DISEASE STATES: A = ASTHMA C = COPD
SHORT-ACTING BETA2-AGONIST BRONCHODILATORS
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ProAir RespiClick®
Proventil® HFA
Ventolin® HFA
Xopenex HFA®
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Serevent® Diskus®
Striverdi® Respimat®
INHALED CORTICOSTEROIDS
Alvesco® HFA
ArmonAir™ RespiClick®
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contain both inhaled corticosteroid and long-acting beta2-agonist (LABA)
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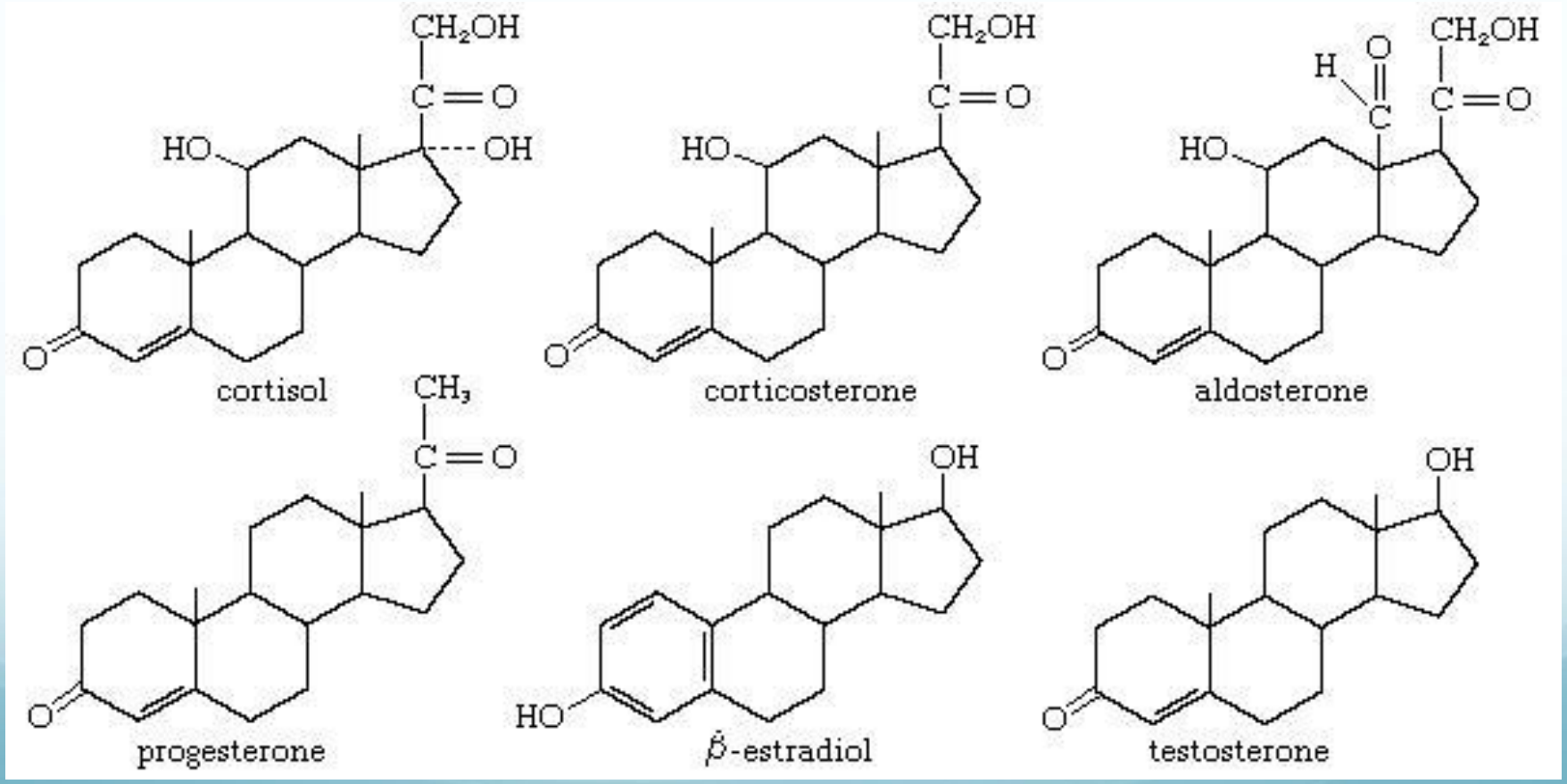
Spiriva® HandiHaler®
Spiriva® Respimat®
Tudorza™ Pressair™
COMBINATION
Short-acting
Combivent® Respimat®
BIOLOGICS target cells and pathways that cause airway inflammation; delivered by injection or IV
Cinqair®
Dupixent®
Fasenra™
Nucala®
Xolair®
BRONCHIAL THERMOPLASTY
PDE4 INHIBITORS
decreasing inflammation and reduce exacerbations
Daliresp®
250, 500 mcg

COPD Terms/Actions/Inhaler Types

The term “steroid” refers to the structure of the compound, not to the function.



COPD Terms/Actions/Inhaler Types

Prednisone et al.

Prednisone –

(1S,2R,10S,11S,14R,15S)-14-hydroxy-14-(2-hydroxyacetyl)-2,15-dimethyltetracyclo[8.7.0.0^{2},^{7}.0^{11},^{15}]heptadeca-3,6-diene-5,17-dione

COPD Terms/Actions/Inhaler Types

Prednisone is metabolized by the liver to prednisolone. A glucocorticoid agonist corticosteroid

One of the first effects is to decreased the leukocyte migration to sites of Inflammation.

Corticosteroids then bind to the glucocorticoid receptor mediates changes in gene expression that lead to multiple downstream effects over hours to days.

COPD Terms/Actions/Inhaler Types

Glucocorticoids inhibit neutrophil apoptosis and demargination; they inhibit phospholipase A2, which decreases the formation of arachidonic acid derivatives; they inhibit NF-Kappa B and other inflammatory transcription factors; they promote anti-inflammatory genes like interleukin.

Lower doses of corticosteroids provide an anti-inflammatory effect, while higher doses are immunosuppressive.

COPD Terms/Actions/Inhaler Types

Aaaaarrrghhhh! Stop – too many words on one slide!

The point, it shuts down most of the things that drive inflammation.

So, think this with me. . What if there is a severe:
ATOPIC inflammation – good stuff!
BACTERIAL inflammation – with abx – good stuff
BACTERIAL inflammation – without abx – hmmm?
VIRAL inflammation – hmmm?

COPD Terms/Actions/Inhaler Types

Taper?

As you know you DON'T have to taper.

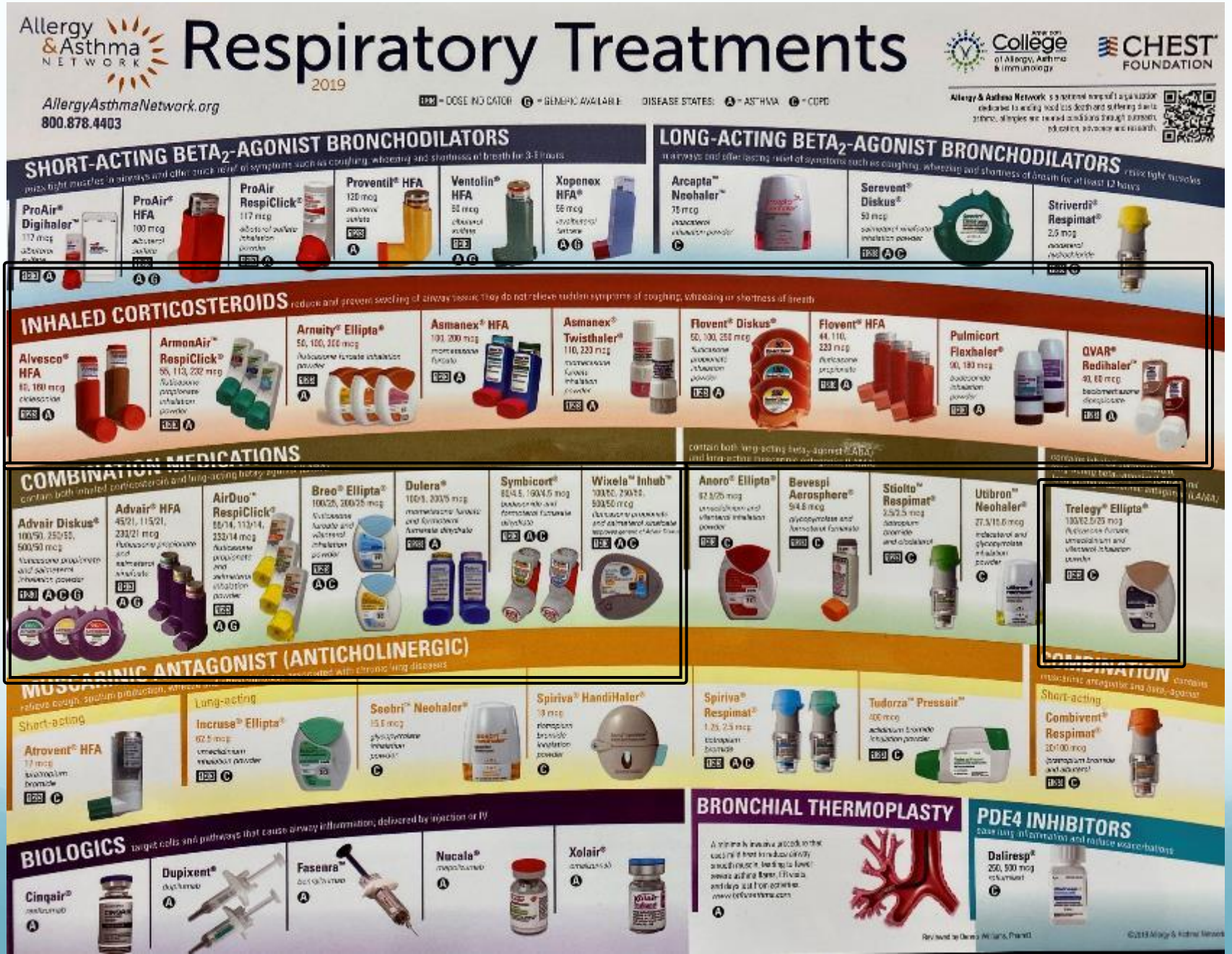
In fact, you should not be putting patients on a dose of steroid that requires a taper.

Tapering is NOT because you have to, it's because you can! You can give them less. . .takes half the dose to keep you well as it did to get you well.

This is where the PATIENT controlled taper is nice:

Take 40 mg till you are 50% better

Take 20 mg till you are back to baseline. . . .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COPD Terms/Actions/Inhaler Types

Ipratropium bromide (and other short and long-acting muscarinic antagonists) are often listed as bronchodilators?

Are they? The exert minimal effect on smooth muscle, so are they?

COPD Terms/Actions/Inhaler Types

Let's look at SAMAs and LAMAs

Ipratropium bromide

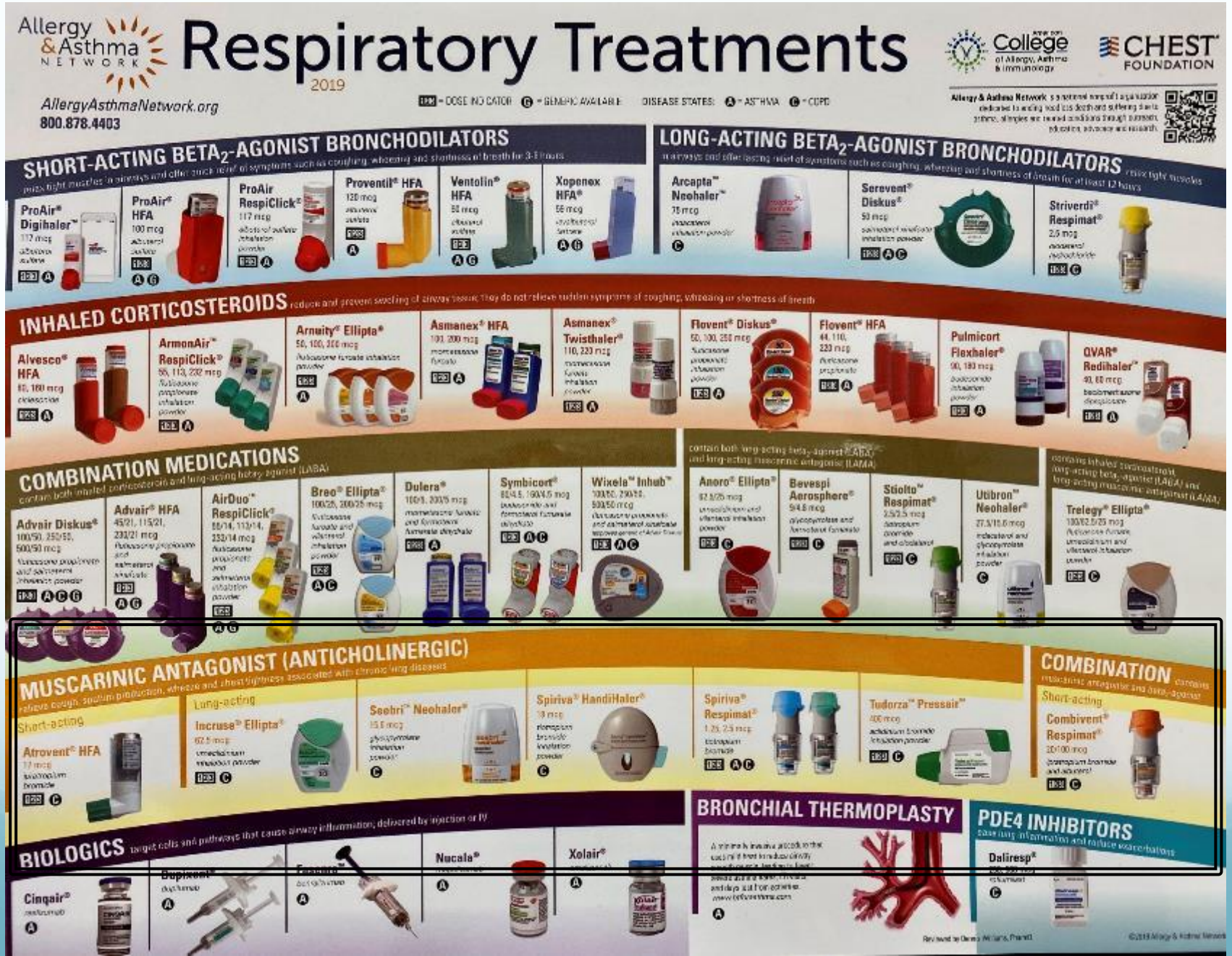
1. Made from the combination of Isopropyl alcohol and atropine. The name comes from these two words. Isopropyl alcohol and atropine
2. Works by INCREASING the degradation of cGMP and by DECREASING Ca2+ in the cells, these all BLOCK contraction. They don't dilate anything really.
3. Onset of action . . . 20 minutes or so. Ipratropium half life is 2 hours.
4. SAMAs and LABAs also effect one big nerve. . . .

COPD Terms/Actions/Inhaler Types

Let's look at SAMAs and LAMAs

Ipratropium bromide

1. Vagal tone – both LAMAs and SAMAs decrease vagal tone (lungs only). This is why they can be helpful in patients with minimal constriction but have dyspnea.
2. So these are very different than SABAs and LABAs, and when combined work very well.
3. For patients over the age of 2 years and older nebulized therapy should use both (if they need a SVN, they need both)
4. Oh yea, the diffusion of inhaled ipratropium bromide (both nose and lungs) does NOT diffuse into the blood in any significant amount. Yep ☺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Allergy & Asthma Network is a national nonprofit organization dedicated to ending needless death and suffering due to asthma, allergies and related conditions through support, education, advocacy and research.
SHORT-ACTING BETA2-AGONIST BRONCHODILATORS
relax tight muscles in airways and offer quick relief of symptoms such as coughing, wheezing and shortness of breath for 3-8 hours
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ProAir® HFA
ProAir RespiClick®
Proventil® HFA
Ventolin® HFA
Xopenex HFA®
LONG-ACTING BETA2-AGONIST BRONCHODILATORS
relax tight muscles in airways and offer lasting relief of symptoms such as coughing, wheezing and shortness of breath for at least 12 hours
Arcapta™ Neohaler™
Serevent® Diskus®
Striverdi® Respimat®
INHALED CORTICOSTEROIDS
reduce and prevent swelling of airway tissue. They do not relieve sudden symptoms of coughing, wheezing or shortness of breath
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ArmonAir™ RespiClick®
Arnuity® Ellipta®
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relieve cough, sputum production, wheeze and chest tightness associated with chronic lung diseases
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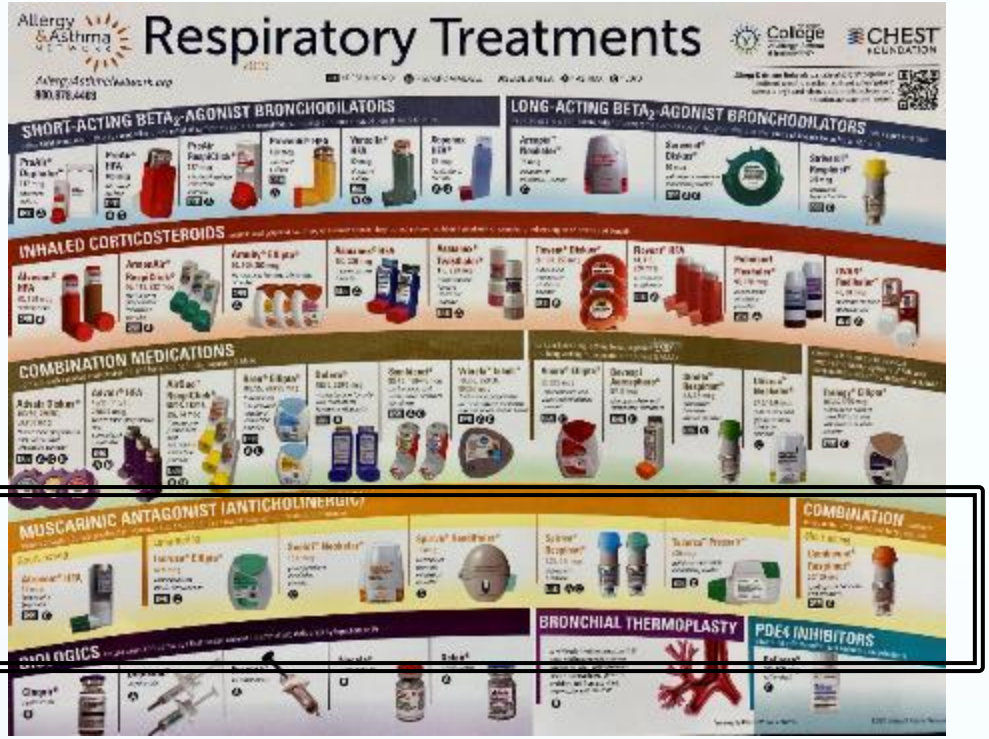


What is missing?
Combivent and nebulized ipratropium bromide

COPD: GOLD Guidelines

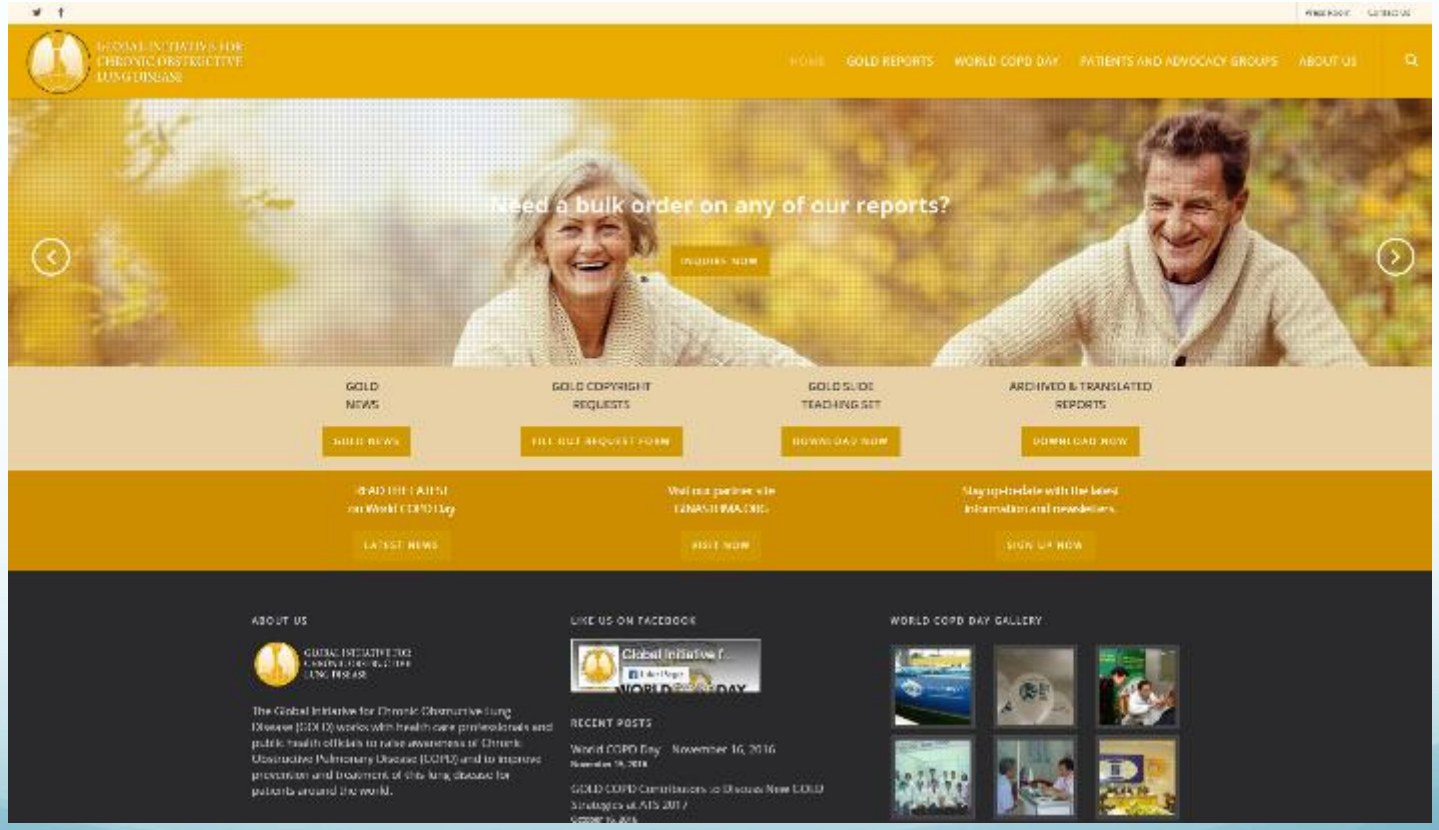
Objectives

- Discuss the GOLD guidelines for COPD
- Discuss prevalence, risk factors, burden of COPD
- Discuss diagnosis and prevention of COPD
- Discuss pharmacologic and non-pharmacologic treatments of COPD

GLOBAL INITIATIVE FOR CHRONIC OBSTRUCTIVE LUNG DISEASE (GOLD):

GOLD Website Address

www.goldcopd.org



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GOLD Initiative

Global Initiative for Chronic **O**bstructive Pu**L**monary **D**isease

- Program Initiated 1998
- WHO and NHLBI of the NIH
- 2001 First Report
- 2006, 2011- complete revisions
- 2013, 2014, 2015, 2016 – 2021 updates

COPD Defined

- ‘A common preventable and treatable disease, is characterized by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases. Exacerbations and comorbidities contribute to the overall severity in individual patients.’
  - Chronic bronchitis: chronic productive cough for 3 months in each of two successive years (other causes excluded)
  - Emphysema: abnormal and permanent enlargement of the airspaces distal to the terminal bronchioles that is accompanied by destruction of the airspace walls w/o fibrosis

Chronic Obstructive Pulmonary Disease (COPD)

- COPD is currently the fourth leading cause of death in the world.[1]
- COPD is projected to be the 3rd leading cause of death by 2021.[2]
- More than 3 million people died of COPD in 2012 accounting for 6% of all deaths globally.
- Globally, the COPD burden is projected to increase in coming decades because of continued exposure to COPD risk factors and aging of the population.

1. Lozano R, Naghavi M, Foreman K, et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 2012; **380**(9859): 2095-128.
2. Mathers CD, Loncar D. Projections of global mortality and burden of disease from 2002 to 2030. *PLoS Med* 2006; **3**(11): e442.

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Burden of Disease

- Social: diminished QOL*
  - Limited inspiratory capacity
  - Worsens with activity
  - Increases work of breathing
- Depression
  - 25-60% of patients**,***
  - ↓QOL, ↑symptoms, ↑HCU, ↑non-adherence**
- Economic: direct $29.5 bil; indirect $20.4 bil
  - Effect on workplace, home productivity

**Yawn BP Int J Chronic Obsructive Pulm Disease 2008;3:311-317;
*Postma DS NEJM 2015;373:1241-1249.
***MacNee W. Annals of Med 2013;45:291-300

Factors that influence disease progression

- Genetic factors
- Age and gender
- Lung growth and development
- Exposure to particles
- Socioeconomic status
- Asthma & airway hyper-reactivity
- Chronic bronchitis
- Infections

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Pathology, pathogenesis & pathophysiology

- Pathology
  - Chronic inflammation
  - Structural changes

- Pathogenesis
  - Oxidative stress
  - Protease-antiprotease imbalance
  - Inflammatory cells
  - Inflammatory mediators
  - Peribronchiolar and interstitial fibrosis

- Pathophysiology
  - Airflow limitation and gas trapping
  - Gas exchange abnormalities
  - Mucus hypersecretion
  - Pulmonary hypertension

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Risk Factors

- Gender
  - Who smokes, occupation or environmental exposures
  - ? Women more susceptible to effects of tobacco smoke
- Genetic
  - Hereditary alpha-1 antitrypsin deficiency
- Socio-economic status:
  - Exposure
  - Second Hand Smoke
  - ?Pollution

GOLD 2018

Risk Factors

- Exposure to pollutants
  - Short-term high peak vs long-term low level?
  - Cigarette smoking >outdoor pollution (~80% smoking hx)
    - Indoor cooking fires
  - Pipe, cigar, water pipe, marijuana, e-cigs/vapors (?)
  - Organic and inorganic dusts, chemicals

Chemicals in Cigarette Smoke

- 600 ingredients
- 7000 chemicals created when cig burns 69 known carcinogens
  - Acetone, ammonia, arsenic, benzene, butane, cadmium, formaldehyde, hexane, lead, naphthalene, methanol, nicotine, tar, toluene
- E-Cigs/vapors
  - 28 known carcinogens
  - ↓lung function, ↑airway resistance, cellular changes
  - Effect not as severe as cigarette smoke

History in COPD

- Risk factors, especially smoking and possibility to reduce
- PMH: asthma, allergy, sinusitis, nasal polyps, respiratory infections in childhood
- FH: COPD, other respiratory disease
- Symptom pattern: onset, worsening dyspnea, more frequent ‘winter colds’, activity and social restriction, change in functional status
- Exacerbations or hospitalization for respiratory disorder
- Comorbidities
  - Heart disease, osteoporosis, m/s disorder, malignancy
- Impact on patient’s life
  - Limited activity, missed work, finances, family routines, depression, anxiety, support network

Signs and Symptoms of COPD

- Chronic cough: productive or non-productive
- Chronic sputum production
- Dyspnea
  - Progressive, persistent
  - Worse with exercise, respiratory infection
  - Described as ‘heaviness’, increased effort to breathe, air hunger, gasping
- Chest tightness
- Wheezing
- Severe COPD:
  - Fatigue, weight loss, anorexia

GOLD 2018

Differential Diagnosis

- Asthma
- CHF
- Bronchiectasis
- TB
- Obliterative bronchiolitis
- Diffuse Panbronchiolitis
- Carcinoma of the bronchus
- Anemia

Adapted from GOLD, 2018

Factors that influence disease development and progression

ASTHMA

- Gender
- Family history
- Atopy
- Allergic rhinitis/sinusitis
- Environmental Tobacco Smoke- ETS
- Obesity

COPD

- Cigarette smoking
- 2nd Hand smoke
- Family history alpha-1 antitrypsin deficiency
- Environmental factors: occupation exposures, air pollution
- Obesity
- Asthma

Postma DS and Rabe KF, NEJM 2015;373:1241-9

Co-Morbidities

- Osteoporosis/osteopenia (50-70%)
- Hypertension (40-60%)
- GER (30-60%)
- Skeletal muscle dysfunction (32%)
- Depression (25%)
- Ischemic heart disease (10-23%)
- Previous MI (4-23%)
- Anemia (17%)
- Diabetes (12-23%)
- Prior stroke (10-14%)
- Arrhythmia (6-14%)
- CRF (6-11%)
- CHF (5-7%)
- OSA (1-4%)

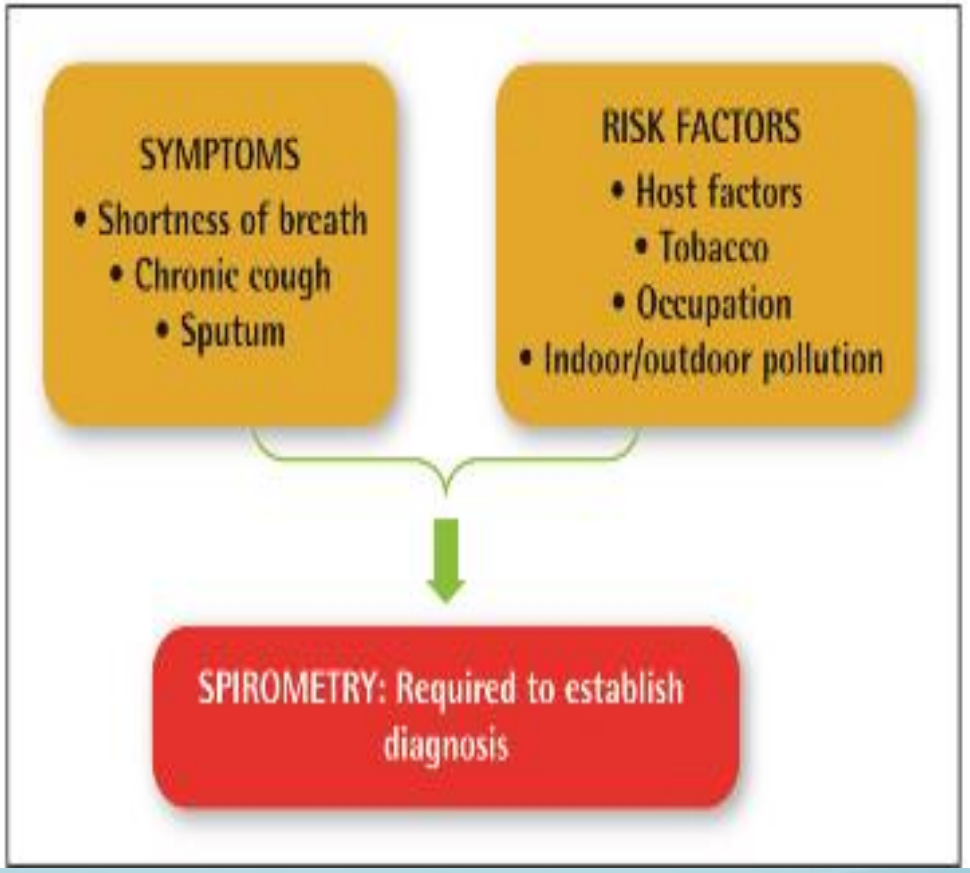
PE in COPD

- VS: BP, HR, RR, BMI, pulse oximetry
- Thoracic exam
  - Observation: pursed lip breathing, barrel chest, accessory muscle use, intercostal retractions (Hoover sign)
  - Percussion: hyperresonance
  - Auscultation: breath sounds diminished, prolonged expiration, rhonchi, wheeze
- CV: Pedal edema, elevated JVP
- Cyanosis

GOLD 2018, Yawn BP Int J Chron Obstruct Pulmon Dis 2008;3:311-317

Diagnosis and Initial Assessment

Figure 2.1. Pathways to the diagnosis of COPD



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Other Assessments

- Required for Diagnosis: Pulmonary function testing
  - **Spirometry post BD (FEV_1/FVC <70%)**
- DLCO; Lung volumes
- 6 minute walk (O_2 desaturation)
- CAT test
- Labs: CBC, CBC, CBC, CBC
- Radiology: CXR, CT with contrast

GOLD, 2018

Other Assessments

- Alpha-1 antitrypsin deficiency screening
  - Caucasian, <45 yo, +ve FH of severe COPD
- Biomarkers in COPD*
  - CRP
    - ? Higher in COPD than smokers and non-smokers
      - NHANES III: CRP>3mg/L in 41% pts with mod. COPD and 52% with severe COPD; 6% and 23 % with CRP>10mg
  - IL-6, IL-8, fibrinogen, p-selectin, ICAM-1, TNF-α
  - Adiponectin
  - Matrix metalloproteinases (MMPs) and associated tissue inhibitors of metalloproteinases (TIMP)

*MacNee W. Annals Med 2013;45:291-300

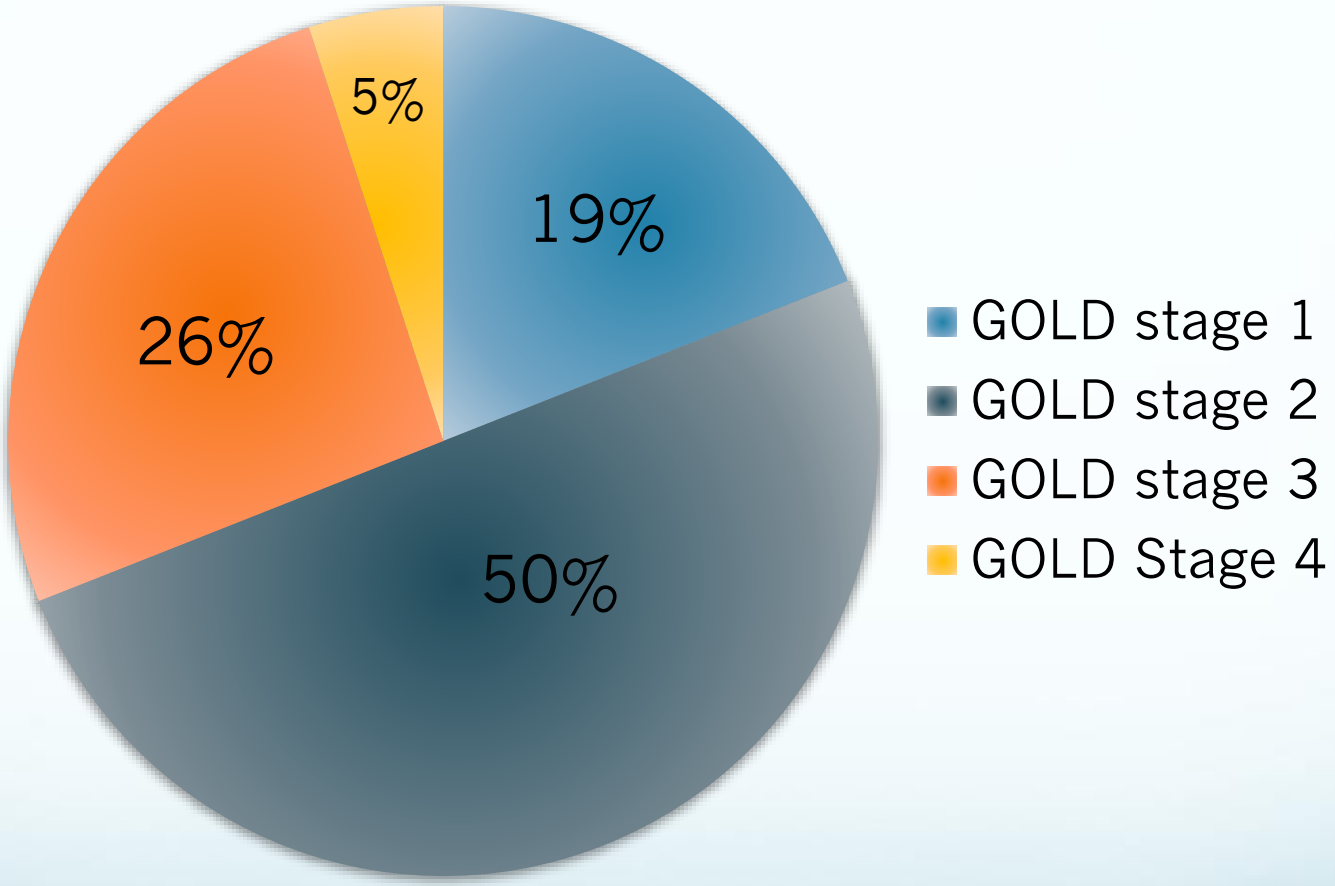
Classification of Severity of Airflow Limitation (post BD FEV_1)*

In patients with **FEV_1/FVC <0.70**:

- GOLD 1 Mild $FEV_1 \geq 80\%$ predicted
- GOLD 2 Moderate $50\% \leq FEV_1$,79% pred
- GOLD 3 Severe* $30\% \leq FEV_1$ <49% pred
- GOLD 4 Very Severe* FEV_1 <30% pred

- May be at higher risk of hospitalization and death
- Adapted from GOLD, 2018

What stage of COPD is your newly diagnosed patient?



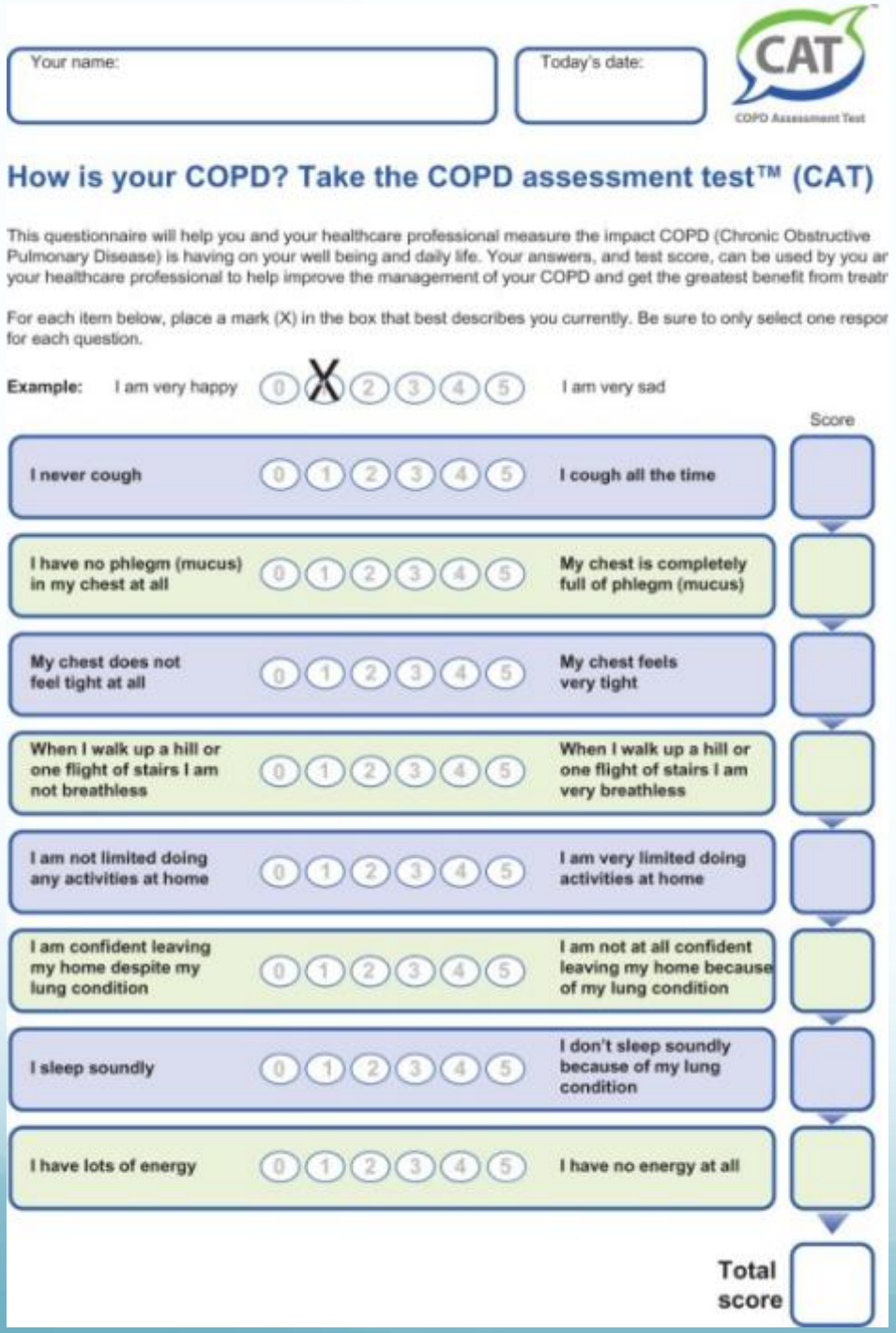
N=12000 patients, 366 with COPD

Mapel DW. Int J Chron Obstruct Pulmon Dis 2011;6:573-581

OK, lets stop for a minute. . . We just DIAGNOSED the patient. We used SPIROMETRY OR PFTs.

Now I don't care about that stuff anymore, everything else is based on symptoms and exacerbations!

Severity of Symptoms



Your name:

Today's date:

CAT
COPD Assessment Test

How is your COPD? Take the COPD assessment test™ (CAT)

This questionnaire will help you and your healthcare professional measure the impact COPD (Chronic Obstructive Pulmonary Disease) is having on your well being and daily life. Your answers, and test score, can be used by you ar your healthcare professional to help improve the management of your COPD and get the greatest benefit from treatr

For each item below, place a mark (X) in the box that best describes you currently. Be sure to only select one respor 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	
		Total score	

ABCD Assessment Tool

Figure 2.4. The refined ABCD assessment tool

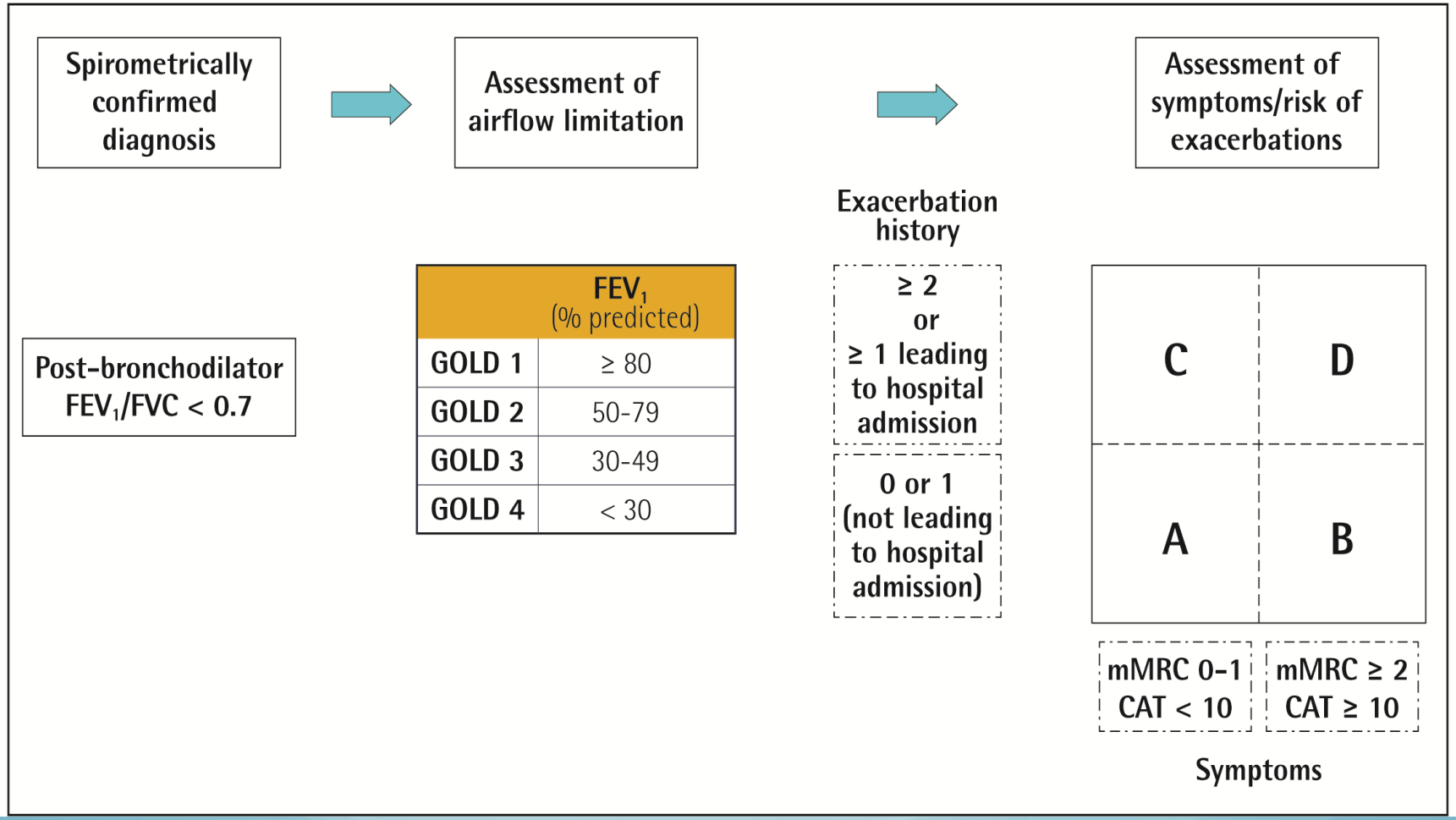


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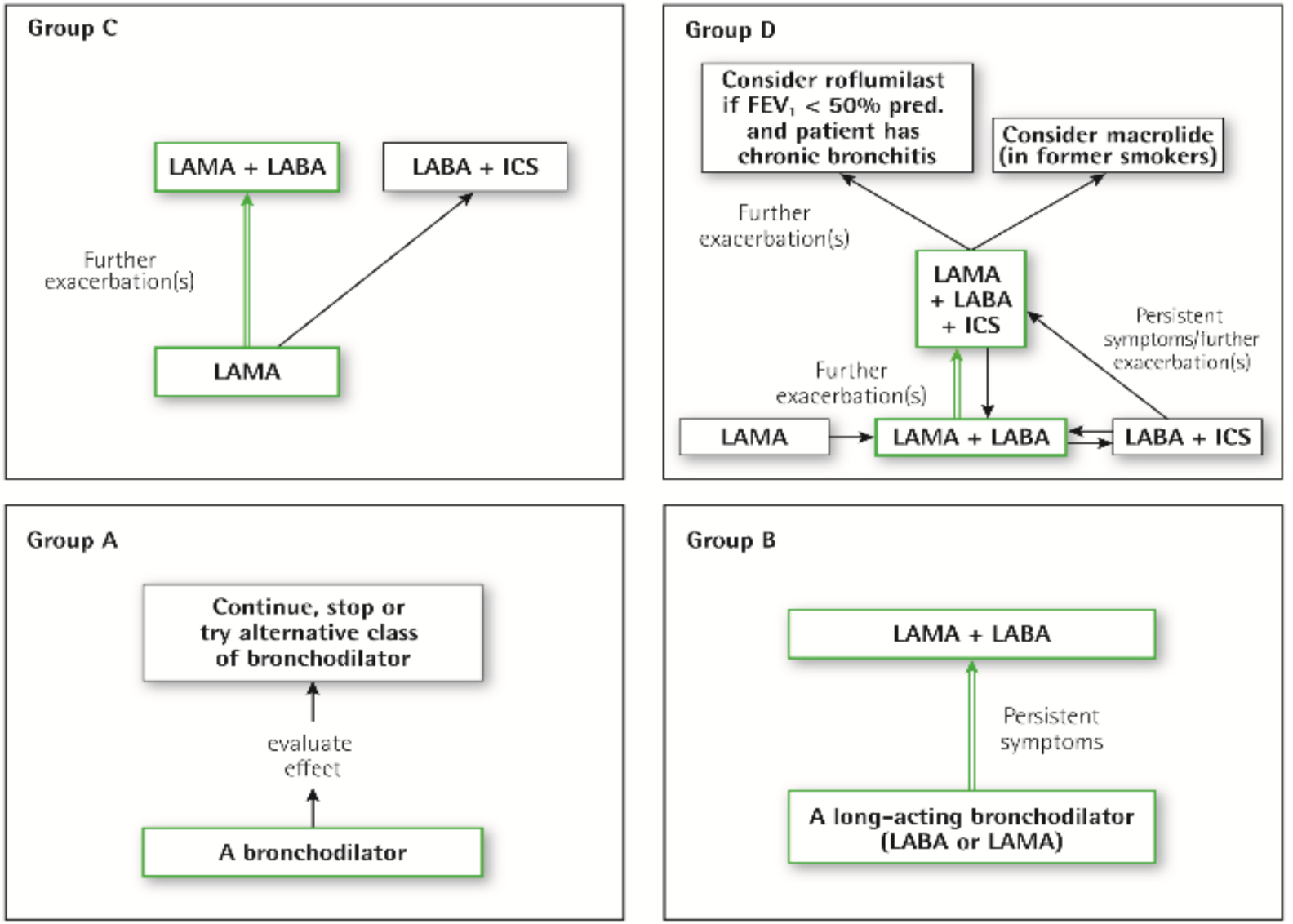
Figure 4.1. Pharmacologic treatment algorithms by GOLD Grade [highlighted boxes and arrows indicate preferred treatment pathways]



Preferred treatment =

In patients with a major discrepancy between the perceived level of symptoms and severity of airflow limitation, further evaluation is warranted.

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Non-Pharmacologic Treatment

Education and self-management

- Self-management education and coaching by healthcare professionals should be a major component of the "Chronic Care Model" within the context of the healthcare delivery system.
- The aim of self-management education is to motivate, engage and coach the patients to positively adapt their health behavior(s) and develop skills to better manage their disease.

Table 4.8. Non-pharmacologic management of COPD

Patient group	Essential	Recommended	Depending on local guidelines
A	Smoking cessation (can include pharmacologic treatment)	Physical activity	Flu vaccination Pneumococcal vaccination
B–D	Smoking cessation (can include pharmacologic treatment) Pulmonary rehabilitation	Physical activity	Flu vaccination Pneumococcal vaccination

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Smoking Cessation

- Smoking cessation has the greatest capacity to influence the natural history of COPD.
- If effective resources and time are dedicated to smoking cessation, long-term quit success rates of up to 25% can be achieved.

Table 3.1. Brief strategies to help the patient willing to quit

• **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.*

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Non-Pharmacologic Treatment - Summary

Table 4.9. Key points for the use of non-pharmacological treatments
Education, self-management and pulmonary rehabilitation
• Education is needed to change patient's knowledge but there is no evidence that used alone it will change patient behavior.
• Education self-management with the support of a case manager with or without the use of a written action plan is recommended for the prevention of exacerbation complications such as hospital admissions **(Evidence B)**.
• Rehabilitation is indicated in all patients with relevant symptoms and/or a high risk for exacerbation **(Evidence A)**.
• Physical activity is a strong predictor of mortality **(Evidence A)**. Patients should be encouraged to increase the level of physical activity although we still don't know how to best insure the likelihood of success.
Vaccination
• Influenza vaccination is recommended for all patients with COPD **(Evidence A)**.
• Pneumococcal vaccination: the PCV13 and PPSV23 are recommended for all patients > 65 years of age, and in younger patients with significant comorbid conditions including chronic heart or lung disease **(Evidence B)**.
Nutrition
• Nutritional supplementation should be considered in malnourished patients with COPD **(Evidence B)**.
End of life and palliative care
• 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)**.

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Pulmonary Rehabilitation

Evidence A	Evidence B	Evidence C
Improves exercise capacity	Improves survival	Improved respiratory muscle training
Improved recovery after hospitalization	Improved limb function (arm)	
Improved HRQOL	Enhanced long acting bronchodilator effect	
Reduced anxiety and depression	Benefits extend beyond immediate training period	
Reduced perception of breathlessness		

Pharmacologic Treatment Options in COPD

- Goal: reduce symptoms, improve function and QOL
  - Current medications have not been shown to prevent decline in lung function
- Consider
  - Comorbidities and their pharmacologic treatments
  - Device for delivery
  - Preference, formulary and cost
  - Low flow rates/significant residual volumes may have better delivery to small airways with nebulizer

Anti-inflammatory Therapy in Stable COPD

Table 3.5. Anti-inflammatory therapy in stable COPD
Inhaled corticosteroids
• 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)**.
• Regular treatment with ICS increases the risk of pneumonia especially in those with severe disease **(Evidence A)**.
• Triple inhaled therapy of ICS/LAMA/LABA improves lung function, symptoms and health status **(Evidence A)** and reduces exacerbations **(Evidence B)** compared to ICS/LABA or LAMA monotherapy.
Oral glucocorticoids
• Long-term use of oral glucocorticoids has numerous side effects **(Evidence A)** with no evidence of benefits **(Evidence C)**.
PDE4 inhibitors
• In patients with chronic bronchitis, severe to very severe COPD and a history of exacerbations: » A PDE4 inhibitor improves lung function and reduces moderate and severe exacerbations **(Evidence A)**. » A PDE4 inhibitor improves lung function and decreases exacerbations in patients who are on fixed-dose LABA/ICS combinations **(Evidence B)**.
Antibiotics
• Long-term azithromycin and erythromycin therapy reduces exacerbations over one year **(Evidence A)**.
• Treatment with azithromycin is associated with an increased incidence of bacterial resistance **(Evidence A)** and hearing test impairments **(Evidence B)**.
Mucolytics/antioxidants
• Regular use of NAC and carbocysteine reduces the risk of exacerbations in select populations **(Evidence B)**.
Other anti-inflammatory agents
• 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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Pharmacologic Therapy

Table 3.3. Commonly used maintenance medications in COPD*					
Drug	**Inhaler (mcg)**	**Solution for nebulizer (mg/ml)**	**Oral**	**Vials for injection (mg)**	**Duration of action (hours)**
Beta$_2$-agonists					
Short-acting					
Fenoterol	100-200 (MDI)	1	2.5 mg (pill), 0.05% (syrup)		4-6
Levalbuterol	45-90 (MDI)	0.1, 0.21, 0.25, 0.42			6-8
Salbutamol (albuterol)	90, 100, 200 (MDI & DPI)†	1, 2, 2.5, 5 mg/ml	2, 4, 5 mg (pill), 8 mg (extended release tablet) 0.024%/0.4 mg (syrup)	0.1, 0.5 mg	4-6, 12 (extended release)
Terbutaline	500 (DPI)		2.5, 5 mg (pill)	0.2, 0.25, 1 mg	4-6
Long-acting					
Arformoterol		0.0075†			12
Formoterol	4.5-9 (DPI)	0.01^			12
Indacaterol	75-300 (DPI)				24
Olodaterol	2.5, 5 (SMI)				24
Salmeterol	25-50 (MDI & DPI)				12
Anticholinergics					
Short-acting					
Ipratropium bromide	20, 40 (MDI)	0.2			6-8
Oxitropium bromide	100 (MDI)				7-9
Long-acting					
Aclidinium bromide	400 (DPI), 400 (MDI)				12
Glycopyrronium bromide	15.6 & 50 (DPI)†		1 mg (solution)	0.2 mg	12-24
Tiotropium	18 (DPI), 2.5 & 5 (SMI)				24
Umeclidinium	62.5 (DPI)				24
Combination of short-acting beta$_2$-agonist plus anticholinergic in one device					
Fenoterol/ipratropium	50/20 (SMI)	1.25, 0.5 mg in 4ml			6-8
Salbutamol/ipratropium	100/20 (SMI), 75/15 (MDI)	0.5, 2.5 mg in 3ml			6-8

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Pharmacologic Therapy

Combination of long-acting beta$_2$-agonist plus anticholinergic in one device				
Formoterol/aclidinium	12/400 (DPI)			12
Formoterol/glycopyrronium	9.6/18 (MDI)			12
Indacaterol/glycopyrronium	27.5/15.6 & 110/50 (DPI)[ǂ]			12-24
Vilanterol/umeclidinium	25/62.5 (DPI)			24
Olodaterol/tiotropium	5/5 (SMI)			24
Methylxanthines				
Aminophylline		105 mg/ml (solution)	250, 500 mg	Variable, up to 24
Theophylline (SR)		100-600 mg (pill)	250, 400, 500 mg	Variable, up to 24
Combination of long-acting beta$_2$-agonist plus corticosteroids in one device				
Formoterol/beclomethasone	6/100 (MDI)			
Formoterol/budesonide	4.5/160 (MDI), 4.5/80 (MDI), 9/320 (DPI), 9/160 (DPI)			
Formoterol/mometasone	10/200, 10/400 (MDI)			
Salmeterol/fluticasone	5/100, 50/250, 5/500 (DPI), 21/45, 21/115, 21/230 (MDI)			
Vilanterol/fluticasone furoate	25/100 (DPI)			
Phosphodiesterase-4 inhibitors				
Roflumilast		500 mcg (pill)		

MDI = metered dose inhaler; DPI = dry powder inhaler; SMI = soft mist inhaler

* Not all formulations are available in all countries; in some countries other formulations and dosages may be available

ǂ Dose availability varies by country

^ Formoterol nebulized solution is based on the unit dose vial containing 20 mcg in a volume of 2.0 ml

† Dose varies by country

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Interventional Therapy in Stable COPD

- Lung volume reduction surgery (LVRS) is a surgical procedure in which parts of the lungs are resected to reduce hyperinflation,261 making respiratory muscles more effective pressure generators by improving their mechanical efficiency.
- Due to the morbidity and mortality associated with LVRS, less invasive bronchoscopic approaches to lung reduction

Table 3.11. 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)**.
Bronchoscopic interventions
• In select patients with advanced emphysema, bronchoscopic interventions reduces end-expiratory lung volume and improves exercise tolerance, health status and lung function at 6-12 months following treatment. Endobronchial valves **(Evidence B)**; Lung coils **(Evidence B)**.

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Monitoring and Follow-up

Monitoring disease progression and development of complications and/or comorbidities

- ***Measurements.*** Decline in FEV_1 can be tracked by spirometry performed at least once a year.
- ***Symptoms.*** At each visit, information on symptoms since the last visit should be collected, including cough and sputum, breathlessness, fatigue, activity limitation, and sleep disturbances.
- ***Exacerbations.*** The frequency, severity, type and likely causes of all exacerbations should be monitored.
- ***Imaging.*** If there is a clear worsening of symptoms, imaging may be indicated.
- ***Smoking status.*** At each visit, the current smoking status and smoke exposure should be determined followed by appropriate action.

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Palliative, End of Life & Hospice Care

- In many patients, the disease trajectory in COPD is marked by a gradual decline in health status and increasing symptoms, punctuated by acute exacerbations that are associated with an increased risk of dying.
- Although mortality rates following hospitalization for an acute exacerbation of COPD are declining, reported rates still vary from 23% to 80%.

Table 3.9. Palliative care, end of life and hospice care in COPD

- Opiates, neuromuscular electrical stimulation (NMES), oxygen and fans blowing air onto the face can relieve breathlessness **(Evidence C)**.
- In malnourished patients, nutritional supplementation 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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Summary

- Increasingly common disease with significant burden
- Most common risk factor: cigarette smoking
  - Reduce exposure to risk factors
- FEV_1 does not indicate impact on the patient, use symptoms and flare ups to guide therapy
- Get a diagnosis, then follow symptoms and flares, move up in therapy as needed, MOST don't need an inhaled steroid but MOST get one anyway. All the side effects, none of the benefits.

Resources for Patients

- American Lung Association (www.lung.org)
- COPD Foundation (U.S.) www.copdfoundation.org

COPD

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