Jak by mohl vypadat aktualizovaný DP CHOPN pro rok 2018 - hlavní body

Vladimír Koblížek a Jaromír Zatloukal

Hradec Králové a Olomouc

Osnova

- Důvody proč se vůbec zabývat aktualizací
- Hlavní body návrhu
- II.Konsenzuální konference 16.11.2017
- Další postup tvorby DP 2018
- Spolupráce PNE s PL

Osnova

- **Důvody proč se vůbec zabývat aktualizací**
- Hlavní body návrhu
- II.Konsenzuální konference 16.11.2017
- Další postup tvorby DP 2018
- Spolupráce PNE s PL

GLOBAL STRATEGY FOR THE DIAGNOSIS, MANAGEMENT, AND PREVENTION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

2017 REPORT

Revised 2017 ABCD Criteria

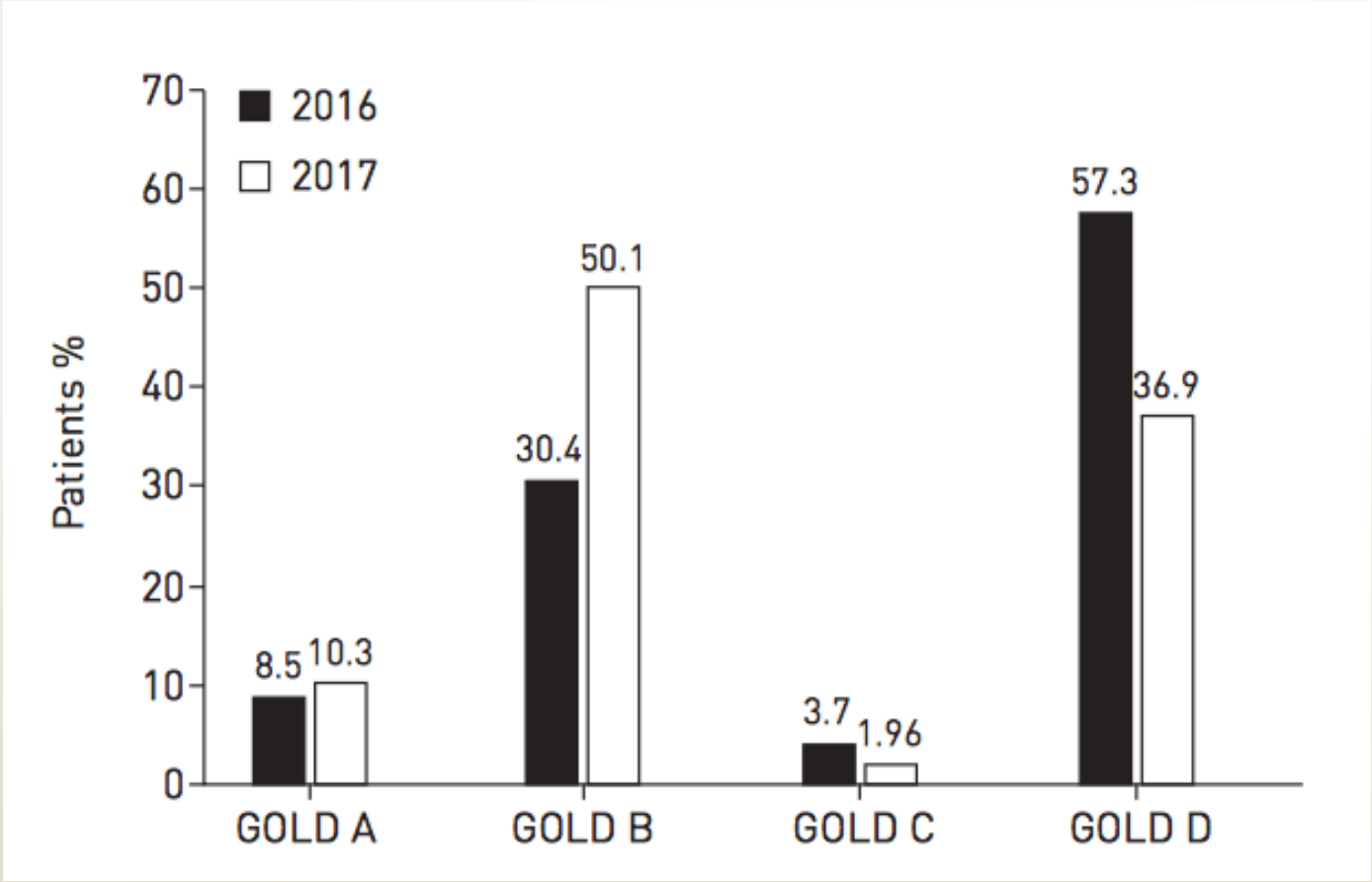
FEV_1	
GOLD 1	≥ 80%
GOLD 2	50-79%
GOLD 3	30-49%
GOLD 4	< 30%

+

Exacerbation History	mMRC 0-1 CAT < 10	mMRC ≥ 2 CAT ≥ 10
≥ 2 or ≥ 1 requiring hospitalization	C	D
≤ 1 not requiring hospitalization	A	B

GOLD 2017 on the way to a phenotypic approach? Analysis from the Phenotypes of COPD in Central and Eastern Europe (POPE) Cohort

Tudoric et al Eur Respir J 2017



International Journal of COPD

Dovepress

open access to scientific and medical research

Open Access Full Text Article

ORIGINAL RESEARCH

Prediction models for exacerbations in different COPD patient populations: comparing results of five large data sources

This article was published in the following Dove Press journal:
International Journal of COPD
1 November 2017
Number of times this article has been viewed

Martine Hoogendoorn[1]

Background and objectives: Exacerbations are important outcomes in COPD both from a

Table 1 Baseline characteristics# of the patients in the five data sources, data are mean or %

	COPDGene[24]	OLIN[25]	RECODE[26]	ECLIPSE[27]	UPLIFT[28]
N	3,756	449	1,086	2,164	5,799
Male (%)	44	60	46	65	75
Age	64	63	68	63	64
Post FEV_1% predicted	57.3	75.8	67.8	48.3	47.6
GOLD stages based on FEV_1%					
Mild	16	41	24	0	0
Moderate	41	53	53	44	46
Severe	26	6	19	42	45
Very severe	12	1	3	14	9
Smoker (%)	37	Na	37	36	30
BMI <20 (%)	5	3	Na	11	11
History of cardiovascular disease (%)	9.8	25	16	33	52
SGRQ total score	36	Na	36	50	46
Exacerbations in the year prior to baseline					
Total exacerbations	0.64	0.30	0.37	1.21	0.85
Severe exacerbations	0.18	0.02	0.02*	0.22	0.25

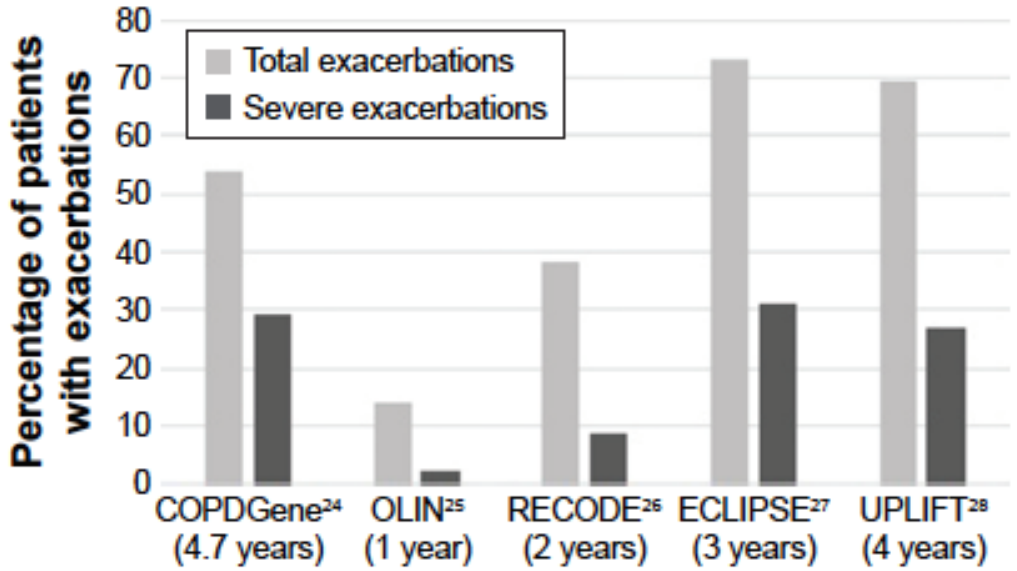


Figure 1 Percentage of patients with at least one (severe) exacerbation during follow-up with the duration of follow-up presented in brackets.

Abbreviations: ECLIPSE, Evaluation of COPD Longitudinally to Identify Predictive Surrogate Endpoints; OLIN, Obstructive Lung Disease in Norrbotten.

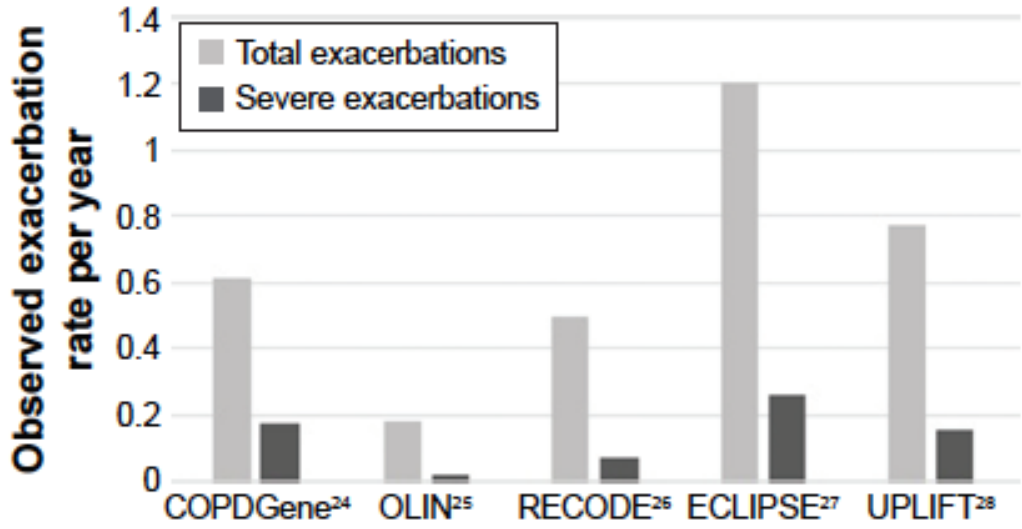


Figure 2 Mean annual total and severe exacerbation rates during follow-up. (Rates are calculated as the sum of exacerbations over all patients divided by the sum of follow-up time to correct for patients with a short follow-up time.)

Abbreviations: ECLIPSE, Evaluation of COPD Longitudinally to Identify Predictive Surrogate Endpoints; OLIN, Obstructive Lung Disease in Norrbotten.

Table 2 Prediction models for total exacerbations including a fixed set of predictors and other database-specific predictors (results from multivariate analysis)

	IRRs#				
	COPDGene[24]	OLIN[25]	RECODE[26]	ECLIPSE[27]	UPLIFT[28]
Fixed set of predictors					
Sex (1= female)	1.23**	2.86*	1.20	1.31***	1.09
Age (years)	1.00	1.00	1.00	1.01*	1.01***
FEV_1% predicted (in %)	0.99***	0.96**	0.98***	0.99***	0.99***
Number of exacerbations prior to baseline	1.75***	1.49***	1.58***	1.38***	1.25***
BMI <20 kg/m^2 (1= yes)	0.98	1.65	–	1.09	1.20**
History of cardiovascular disease (1= yes)	1.07	1.13	1.06	0.98	0.99
SGRQ total score at baseline (in points)	1.01***	–	1.02**	1.01***	1.01***
Treatment group in trial (1= yes)	–	–	1.08	–	0.83***
Other database-specific predictors					
Smoker (1= yes, 0= former)	0.81**	–	1.07	0.88*	1.05
Pack-years	–	–	–	–	1.002**
Time since diagnosis (years)	–	–	–	–	1.005
Diagnosis of emphysema (1= yes)	–	–	–	–	0.97
Cough (1= yes)	1.16*	–	–	–	–
Wheeze (1= yes)	1.37***	–	–	–	–
MRC dyspnea	–	–	0.98	1.03	–
Charlson comorbidity index	–	–	0.98	–	0.98
Other comorbidities (1= yes)	–	–	–	1.00	–
6-min walking test (m)	–	–	–	1.001***	–
Physical activity IPAQ (1= low)	–	–	0.81	–	–
ICS at baseline (1= yes)	–	–	–	–	1.30***
Resting O_2 saturation (in %)	0.97**	–	–	–	–
Fibrinogen (mg/dL)	–	–	–	1.00	–

International Journal of COPD

Dovepress

open access to scientific and medical research

Open Access Full Text Article

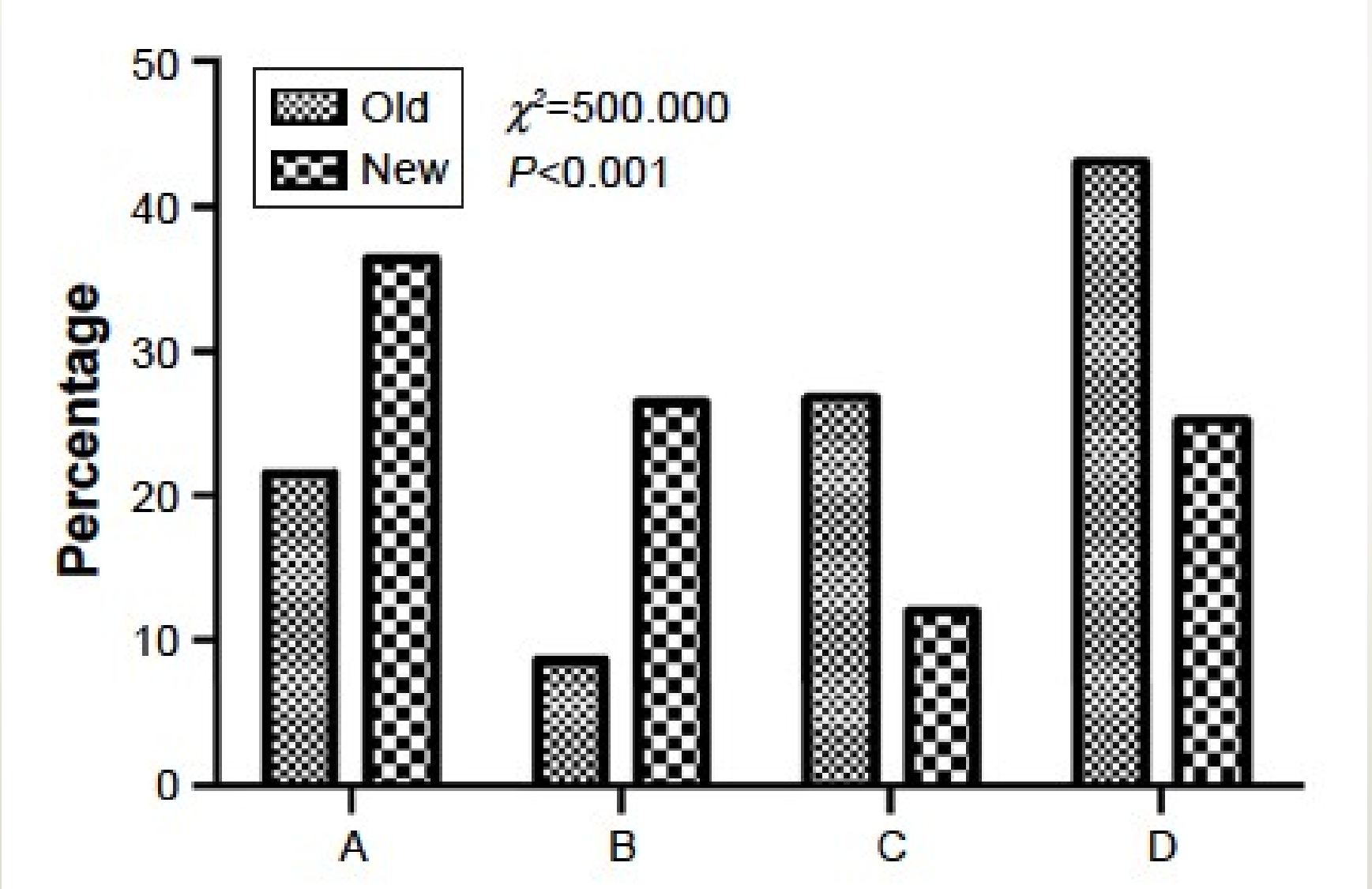
ORIGINAL RESEARCH

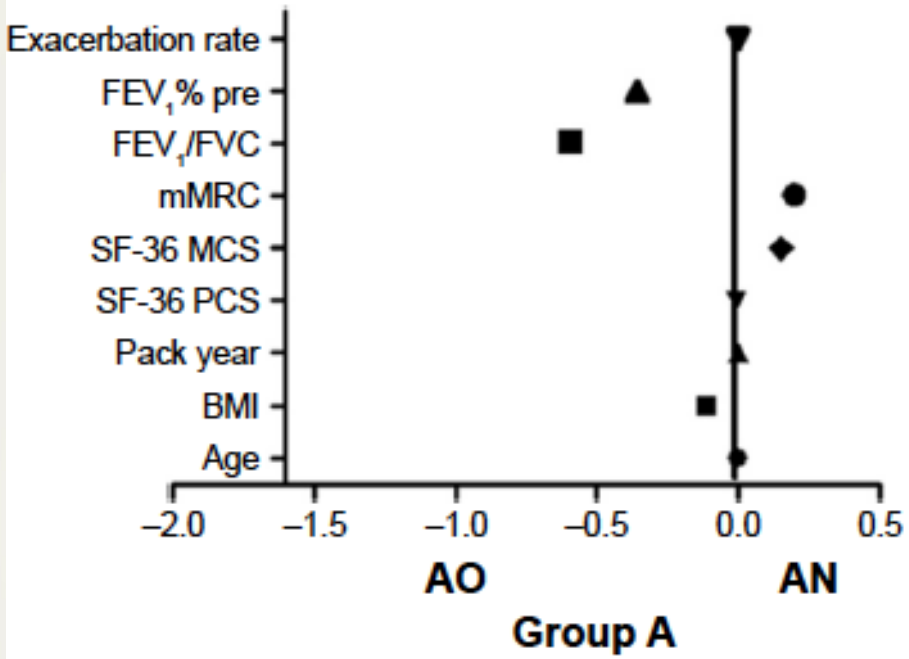
Changes in definition lead to changes in the clinical characteristics across COPD categories according to GOLD 2017: a national cross-sectional survey in China

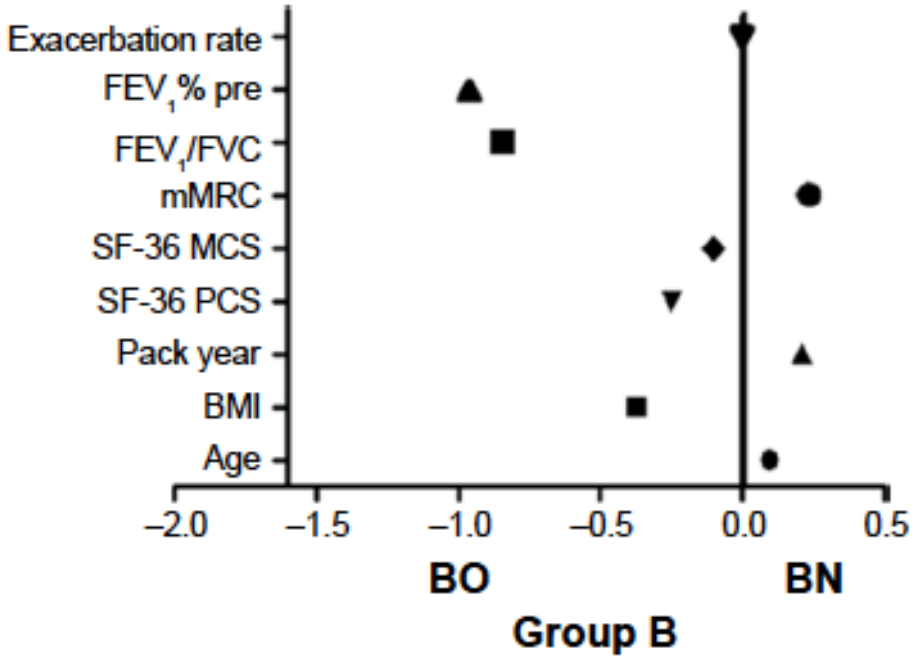
This article was published in the following Dove Press journal:
International Journal of COPD
20 October 2017
Number of times this article has been viewed

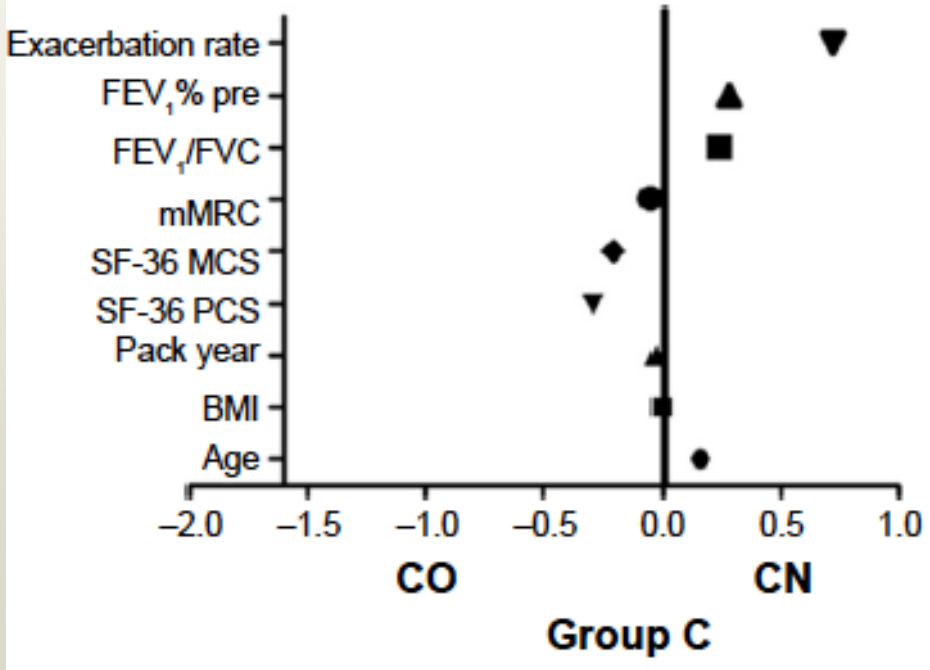
Lina Sun*

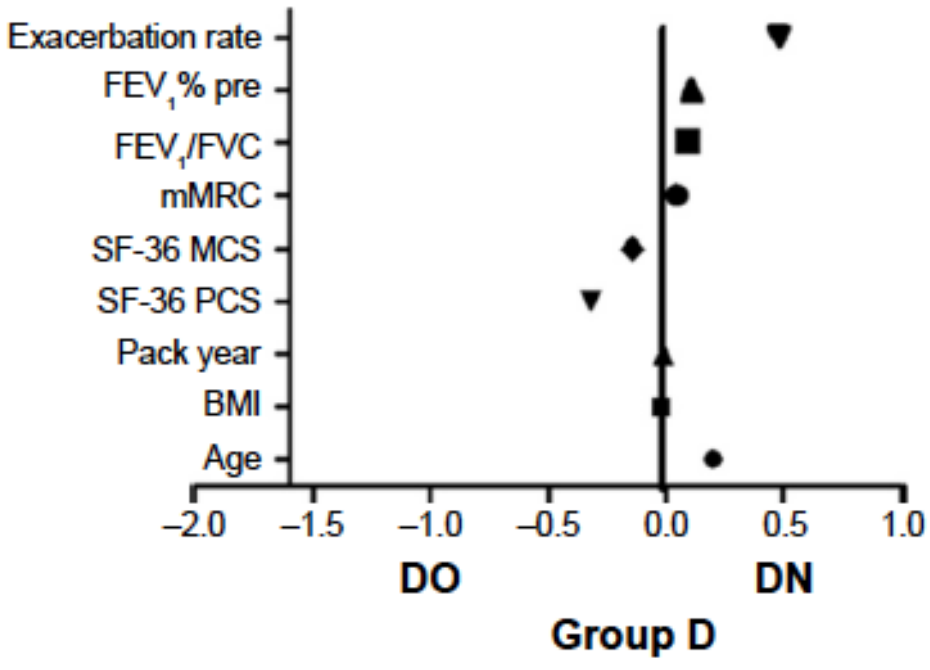
Purpose: To investigate how the changes of definition in assessment of Global Initiative for

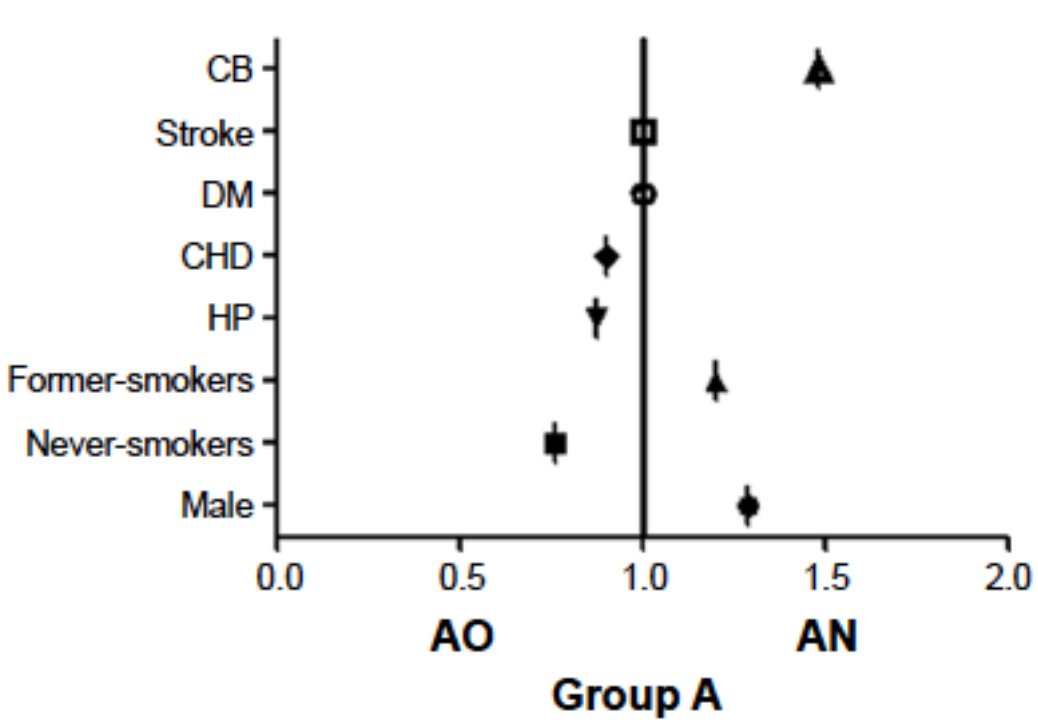

Old
New
χ^2=500.000
P<0.001
Percentage
50
40
30
20
10
0
A
B
C
D

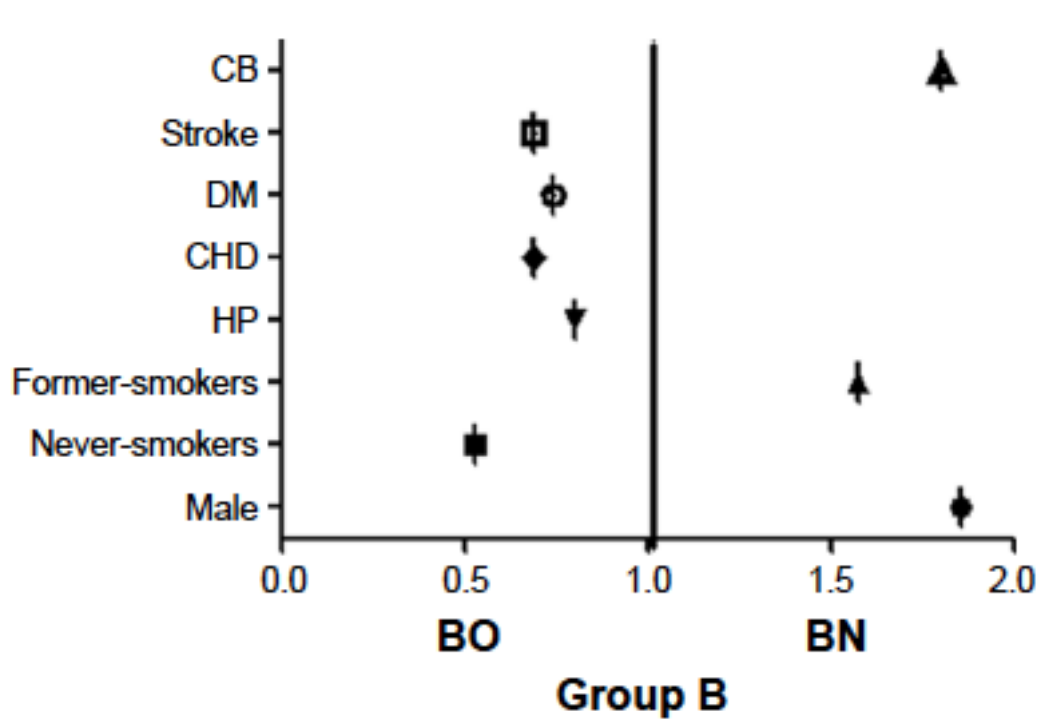

Exacerbation rate
FEV1% pre
FEV1/FVC
mMRC
SF-36 MCS
SF-36 PCS
Pack year
BMI
Age
–2.0
–1.5
–1.0
–0.5
0.0
0.5
AO
AN
Group A

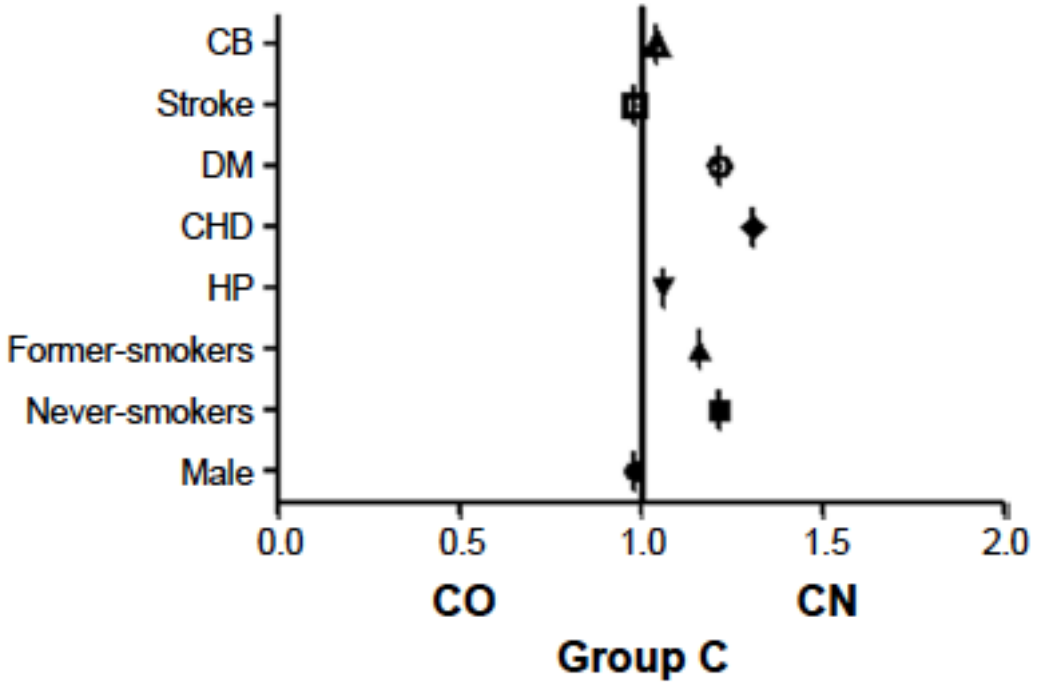

Exacerbation rate
FEV1% pre
FEV1/FVC
mMRC
SF-36 MCS
SF-36 PCS
Pack year
BMI
Age
–2.0
–1.5
–1.0
–0.5
0.0
0.5
BO
BN
Group B

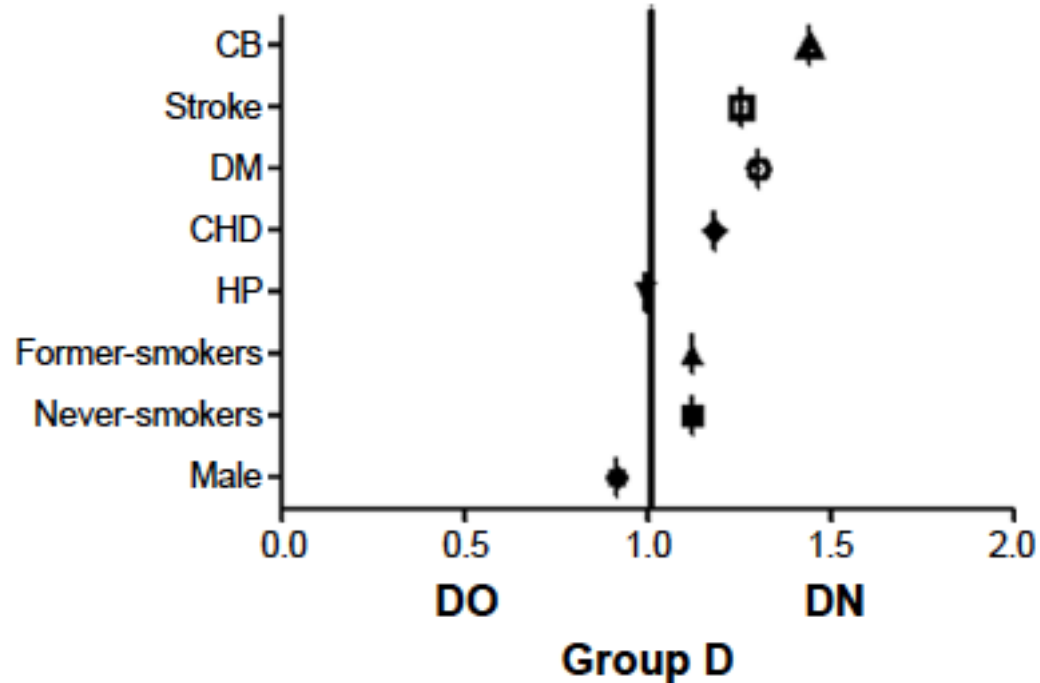

Exacerbation rate
FEV1% pre
FEV1/FVC
mMRC
SF-36 MCS
SF-36 PCS
Pack year
BMI
Age
–2.0
–1.5
–1.0
–0.5
0.0
0.5
1.0
CO
CN
Group C


Exacerbation rate
FEV1% pre
FEV1/FVC
mMRC
SF-36 MCS
SF-36 PCS
Pack year
BMI
Age
–2.0
–1.5
–1.0
–0.5
0.0
0.5
1.0
DO
DN
Group D


CB
Stroke
DM
CHD
HP
Former-smokers
Never-smokers
Male
0.0
0.5
1.0
1.5
2.0
AO
AN
Group A


CB
Stroke
DM
CHD
HP
Former-smokers
Never-smokers
Male
0.0
0.5
1.0
1.5
2.0
BO
BN
Group B


CB
Stroke
DM
CHD
HP
Former-smokers
Never-smokers
Male
0.0
0.5
1.0
1.5
2.0
CO
CN
Group C


CB
Stroke
DM
CHD
HP
Former-smokers
Never-smokers
Male
0.0
0.5
1.0
1.5
2.0
DO
DN
Group D

Bradford et al. Respiratory Research (2017) 18:180
DOI 10.1186/s12931-017-0662-2

Respiratory Research

RESEARCH

Open Access

The value of blood cytokines and chemokines in assessing COPD

Eric Bradford[1], Sean Jacobson[1], Jason Varasteh[1], Alejandro P. Comellas[6], Prescott Woodruff[7], Wanda O'Neal[8], Dawn L. DeMeo[5], Xingnan Li[9], Victor Kim[10], Michael Cho[4], Peter J. Castaldi[5,11], Craig Hersh[5], Edwin K. Silverman[4], James D. Crapo[1], Katerina Kechris[3] and Russell P. Bowler[1,2*]

Abstract

Background: Blood biomarkers are increasingly used to stratify high risk chronic obstructive pulmonary disease (COPD) patients; however, there are fewer studies that have investigated multiple biomarkers and replicated in multiple large well-characterized cohorts of susceptible current and former smokers.

Methods: We used two MSD multiplex panels to measure 9 cytokines and chemokines in 2123 subjects from COPDGene and 1117 subjects from SPIROMICS. These biomarkers included: interleukin (IL)-2, IL-6, IL-8, IL-10, tumor necrosis factor (TNF)-α, interferon (IFN)-γ, eotaxin/CCL-11, eotaxin-3/CCL-26, and thymus and activation-regulated chemokine (TARC)/CCL-17. Regression models adjusted for clinical covariates were used to determine which biomarkers were associated with the following COPD phenotypes: airflow obstruction (forced expiratory flow at 1 s (FEV_1%) and FEV_1/forced vital capacity (FEV_1/FVC), chronic bronchitis, COPD exacerbations, and emphysema. Biomarker-genotype associations were assessed by genome-wide association of single nucleotide polymorphisms (SNPs).

Results: Eotaxin and IL-6 were strongly associated with airflow obstruction and accounted for 3–5% of the measurement variance on top of clinical variables. IL-6 was associated with progressive airflow obstruction over 5 years and both IL-6 and IL-8 were associated with progressive emphysema over 5 years. None of the biomarkers were consistently associated with chronic bronchitis or COPD exacerbations. We identified one novel SNP (rs9302690 SNP) that was associated with CCL17 plasma measurements.

Conclusion: When assessing smoking related pulmonary disease, biomarkers of inflammation such as IL-2, IL-6, IL-8, and eotaxin may add additional modest predictive value on top of clinical variables alone.

Trial registration: COPDGene (ClinicalTrials.gov Identifier: NCT02445183).
Subpopulations and Intermediate Outcomes Measures in COPD Study (SPIROMICS) (ClinicalTrials.gov Identifier: NCT 01969344).

International Journal of COPD

Dovepress

open access to scientific and medical research

Open Access Full Text Article

ORIGINAL RESEARCH

Hyperinflated lungs compress the heart during expiration in COPD patients: a new finding on dynamic-ventilation computed tomography

This article was published in the following Dove Press journal:
International Journal of COPD
26 October 2017
Number of times this article has been viewed

Yanyan Xu[1,2]

Purpose: The aims of this study were to evaluate dynamic changes in heart size during the

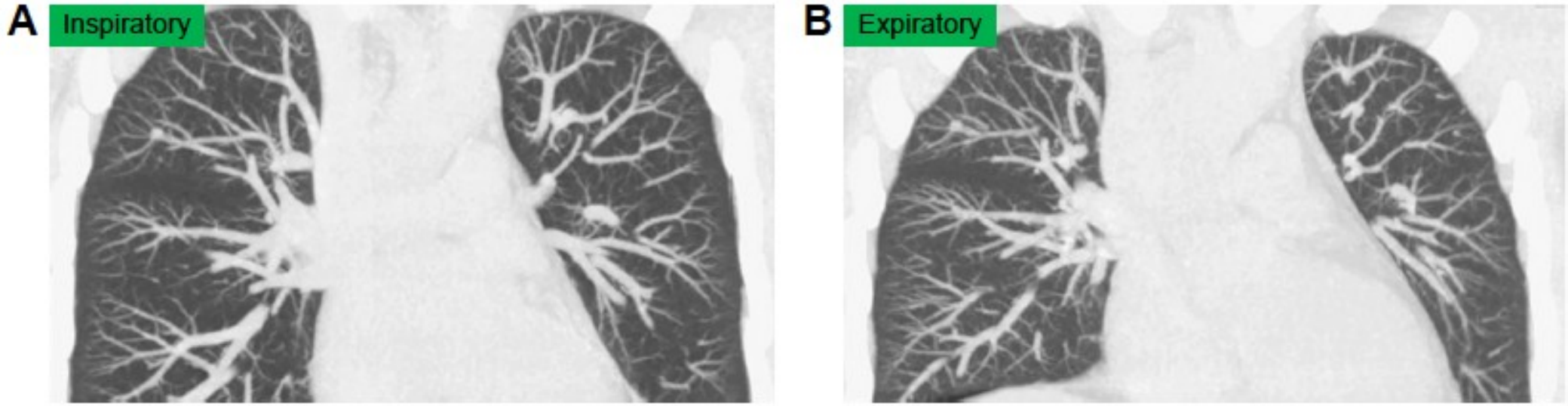


Figure 1 A 39-year-old male current smoker without COPD underwent dynamic-ventilation CT for preoperative analysis of parietal pleural adhesion caused by a benign rib tumor.

Notes: His $FEV_{1.0}$/FVC was 0.92. Inspiratory (**A**) and expiratory phases (**B**) (both shown in coronal view, MIP images) demonstrated an increase in heart size during expiration, mainly due to diaphragm elevation.

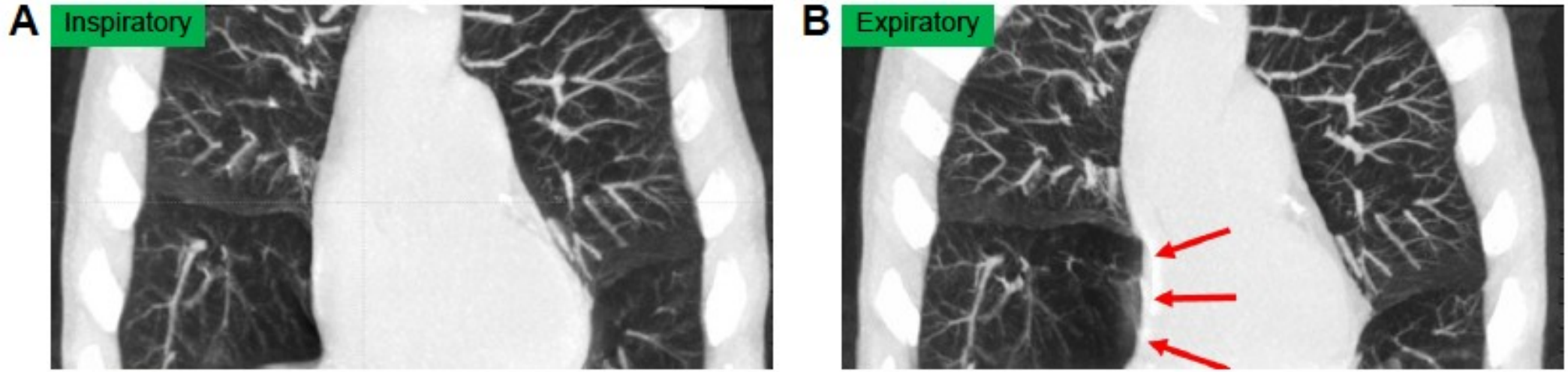
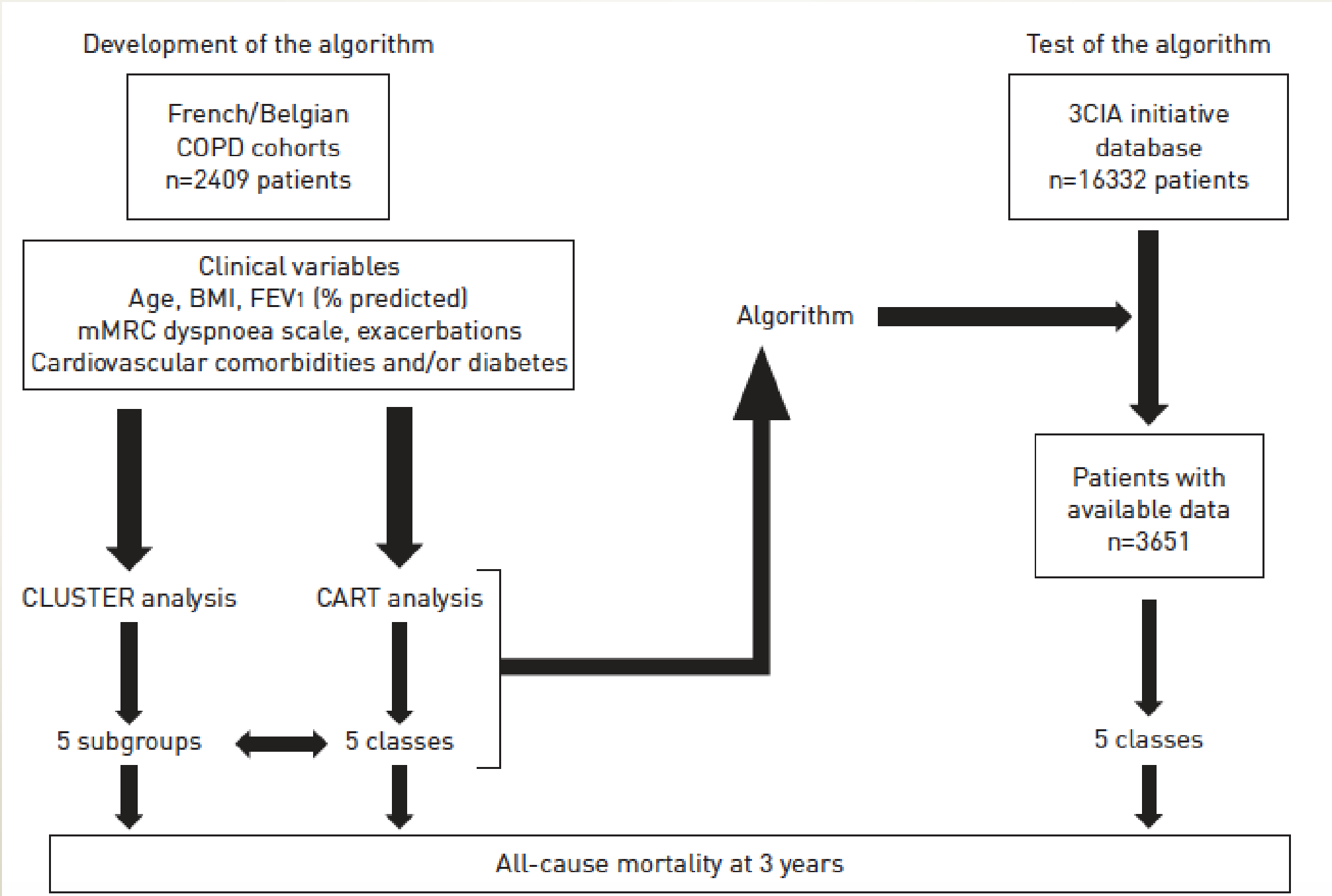
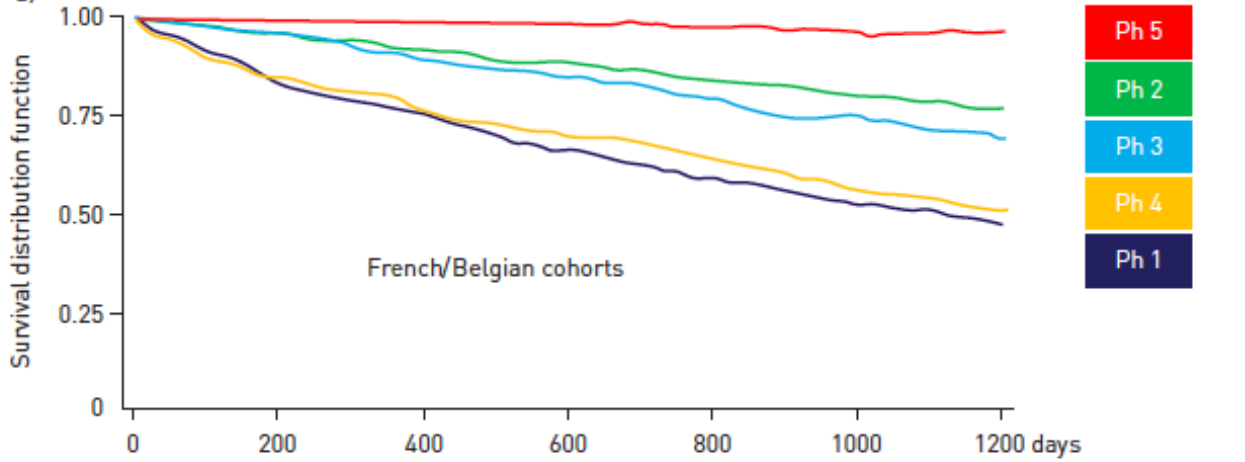


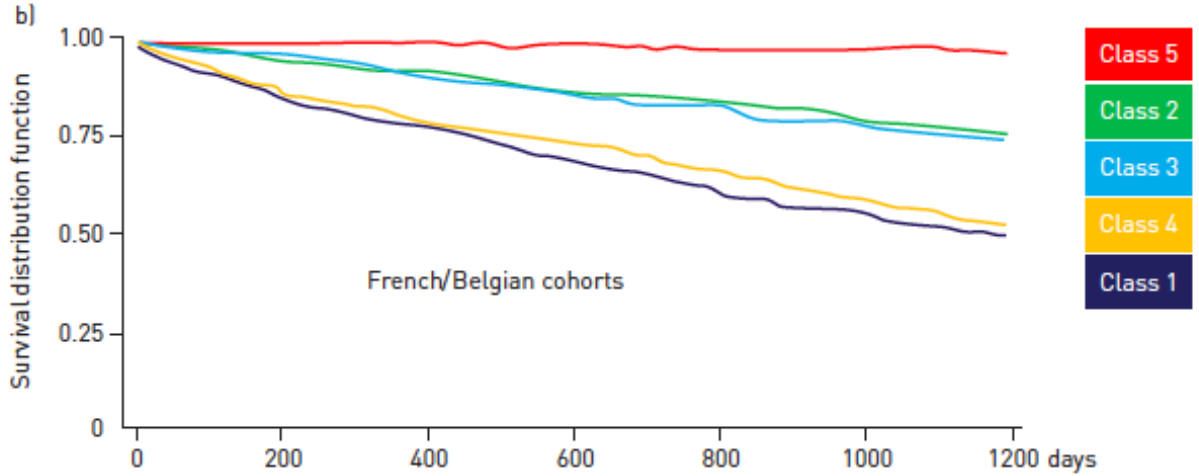
Figure 2 A 70-year-old male with COPD underwent dynamic-ventilation CT to evaluate central airway abnormalities.
Notes: His $FEV_{1.0}$/FVC was 0.55. The shape of the right atrium was normal during the inspiratory phase (**A**) but was severely compressed (arrows) during the expiratory phase, probably due to the emphysematous right middle lobe (**B**).
Abbreviations: CT, computed tomography; $FEV_{1.0}$, forced expiratory volume in 1 s; FVC, forced vital capacity.

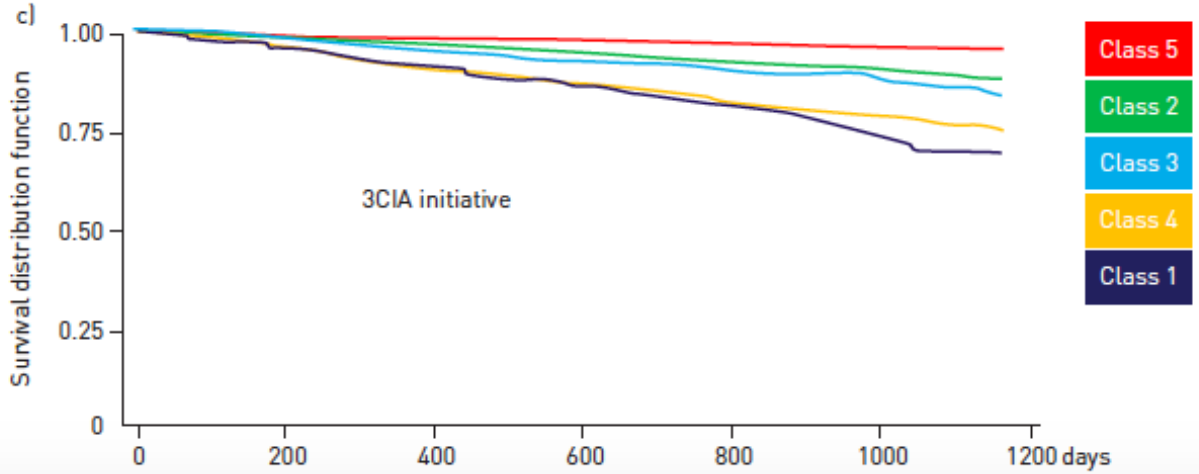
A simple algorithm for the identification of clinical COPD phenotypes

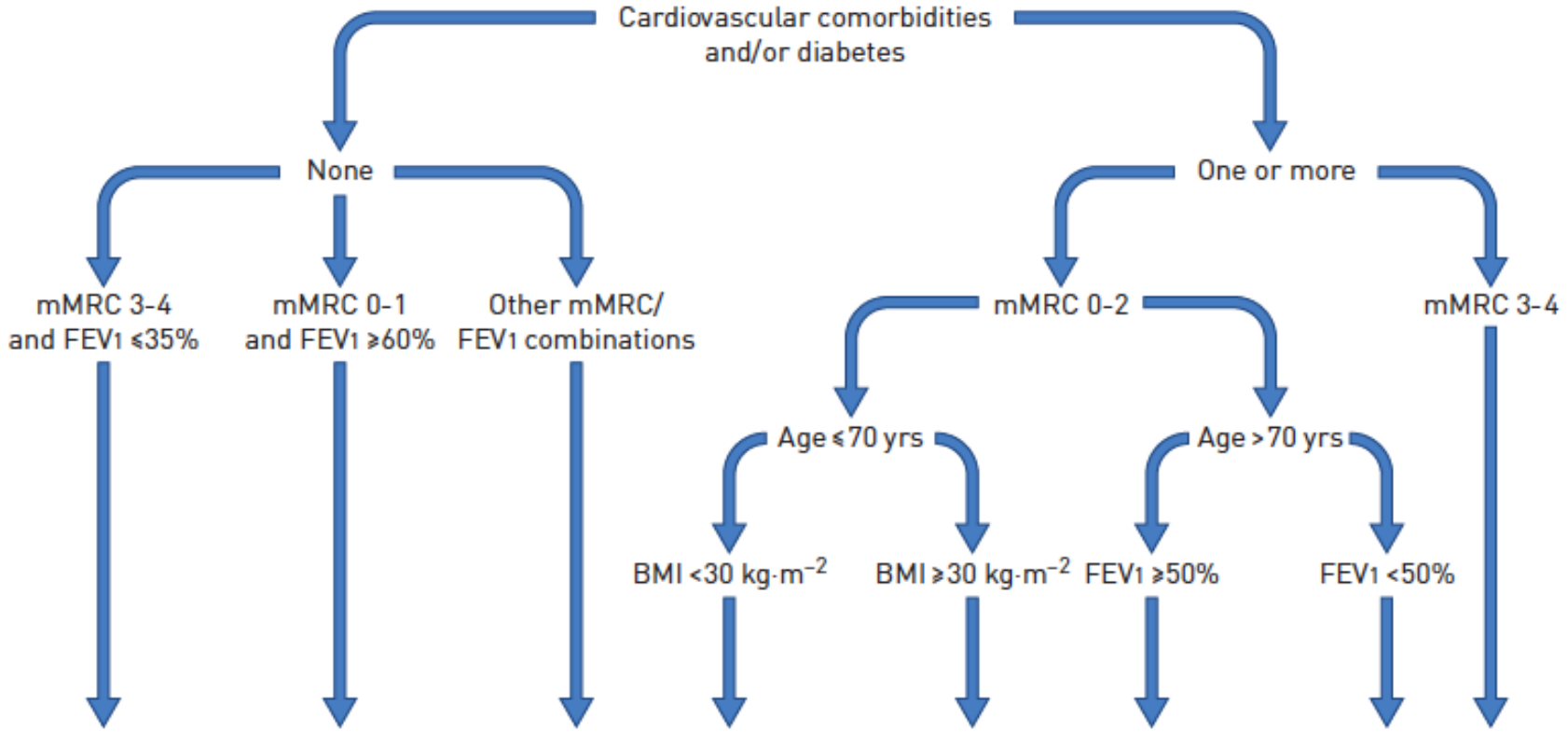
Pierre-Régis Burgel[1,2], Jean-Louis Paillasseur[3], Wim Janssens[4], Jacques Piquet[5], Gerben ter Riet[6], Judith Garcia-Aymerich[7], Borja Cosio[8], Per Bakke[9], Milo A. Puhan[10], Arnulf Langhammer[11], Inmaculada Alfageme[12], Pere Almagro[13], Julio Ancochea[14], Bartolome R. Celli[15], Ciro Casanova[16], Juan P. de-Torres[17], Marc Decramer[4], Andrés Echazarreta[18], Cristobal Esteban[19], Rosa Mar Gomez Punter[20], MeiLan K. Han[21], Ane Johannessen[22], Bernhard Kaiser[23], Bernd Lamprecht[24], Peter Lange[25], Linda Leivseth[26], Jose M. Marin[27], Francis Martin[28], Pablo Martinez-Camblor[29,30], Marc Miravitlles[31], Toru Oga[32], Ana Sofia Ramírez[33], Don D. Sin[34], Patricia Sobradillo[35], Juan J. Soler-Cataluña[36], Alice M. Turner[37], Francisco Javier Verdu Rivera[38], Joan B. Soriano[39] and Nicolas Roche[1,2] on behalf of Initiatives BPCO, EABPCO, Leuven and 3CIA study groups


Development of the algorithm
French/Belgian
COPD cohorts
n=2409 patients
Clinical variables
Age, BMI, FEV1 (% predicted)
mMRC dyspnoea scale, exacerbations
Cardiovascular comorbidities and/or diabetes
CLUSTER analysis
CART analysis
5 subgroups
5 classes
Algorithm
Test of the algorithm
3CIA initiative
database
n=16332 patients
Patients with
available data
n=3651
5 classes
All-cause mortality at 3 years


Survival distribution function
1.00
0.75
0.50
0.25
0
French/Belgian cohorts
0
200
400
600
800
1000
1200 days
Ph 5
Ph 2
Ph 3
Ph 4
Ph 1


b)
Survival distribution function
1.00
0.75
0.50
0.25
0
French/Belgian cohorts
0
200
400
600
800
1000
1200 days
Class 5
Class 2
Class 3
Class 4
Class 1


c)
Survival distribution function
1.00
0.75
0.50
0.25
0
3CIA initiative
0
200
400
600
800
1000
1200 days
Class 5
Class 2
Class 3
Class 4
Class 1



Class	Class 4	Class 5	Class 2	Class 3	Class 1
French/Belgian cohort (derivation cohort)					
Patients n (%)	267 (11.2)	230 (9.5)	981 (40.7)	283 (11.7)	648 (26.9)
Deaths n (%)	121 (45.3)	6 (2.6)	225 (22.9)	68 (24.0)	321 (49.5)
3CIA initiative (test cohort)					
Patients n (%)	150 (4.1)	1037 (28.4)	1614 (44.2)	398 (10.9)	452 (12.3)
Deaths n (%)	41 (27.3)	41 (4.0)	179 (11.1)	56 (14.1)	105 (23.2)

FIGURE 2 Algorithm developed by classification and regression tree (CART) analysis for the classification of chronic obstructive pulmonary disease (COPD) patients. Application to the French/Belgian and 3CIA cohorts. BMI: body mass index; FEV1: forced expiratory volume in 1 s; mMRC: modified Medical Research Council.

TABLE 2 Main descriptors of the five chronic obstructive pulmonary disease (COPD) phenotypes identified by cluster analysis in the French/Belgian COPD cohort[#]

	Good prognosis	Intermediate prognosis		Poor prognosis	
Phenotype number	**V**	**II**	**III**	**IV**	**I**
3-year mortality rate %	2.5	21.8	30.0	47.0	50.9
Phenotype name	Mild respiratory	Moderate-to-severe respiratory	Moderate-to-severe comorbid/obese	Very severe respiratory	Very severe comorbid
Airflow limitation	Mild to moderate	Moderate to very severe	Mild to severe	Severe to very severe	Moderate to very severe
Median BMI kg·m^{-2}	26	24	30	20	26
Clinical manifestations					
Dyspnoea	Mild	Moderate	Moderate	Severe	Severe
Exacerbations	0	Infrequent	Infrequent	Frequent	Frequent
Hospitalisations	0	Infrequent	Infrequent	Frequent	Frequent
Rates of cardiovascular comorbidities/diabetes	Low	Low	Very high	Very low	Very high
Median age years	61	64	74	64	77

BMI: body mass index. [#]: the order is chosen based on 3-year mortality rates.

COPD: algorithms and clinical management

Rosa Faner[1,2] and Alvar Agustí[1,2,3]

Affiliations: [1]Centro Investigación Biomédica En Red Enfermedades Respiratorias (CIBERES), Spain. [2]Fundació Clínic per a la Recerca Biomèdica, Institut d'investigacions Biomèdiques August Pi i Sunyer (IDIBAPS), Barcelona, Spain. [3]Pulmonary Service, Respiratory Institute, Hospital Clinic, University of Barcelona, Barcelona, Spain.

Correspondence: Rosa Faner, CELLEXP2A, c/Casanova 143, Barcelona 08036, Spain. E-mail: rfaner@clinic.cat

Individualisation of treatment

So, how do we put all this together? We believe that, at the end of the day, what really matters in practice is not to know to what group (subtype, cluster or phenotype, however you prefer to call them) the patient that you have in front of you in your office belongs but what is the best treatment that you can (should) offer to this unique individual. From this perspective, we think that the large cross-cohort analysis by CASTALDI *et al.* [15] discussed above is important, since it shows that COPD heterogeneity (if not related to a specific outcome) is better described by continuous disease traits, coexisting in varying degrees in the same patient, rather than by mutually exclusive COPD subtypes. Hence, accepting the risk of being biased, we would therefore suggest that in clinical practice, when confronted with a single patient, the concept of "treatable traits" (as discussed elsewhere in the *ERJ* [18]) is the way forward to individualise treatment, while algorithms like that developed by BURGEL *et al.* [1] can provide complementary information on the individual-patient risk in relation to specific outcomes. Treatable traits can be recognised phenotypically (observable characteristics of an organism) or through validated biomarkers that inform on the presence of specific mechanisms of disease (or endotypes) [18]. Importantly, at variance with the "phenotype" approach, they can coexist in the same individual, and change with time or as a result of treatment [18]. They can be pulmonary, extrapulmonary, and/or social, behavioural and environmental [18]. Needless to say that, as recently agreed in a European Respiratory Society research seminar on this topic, this strategy needs formal validation [19].

International Journal of COPD

Dovepress
open access to scientific and medical research

Open Access Full Text Article

ORIGINAL RESEARCH

Czech multicenter research database of severe COPD

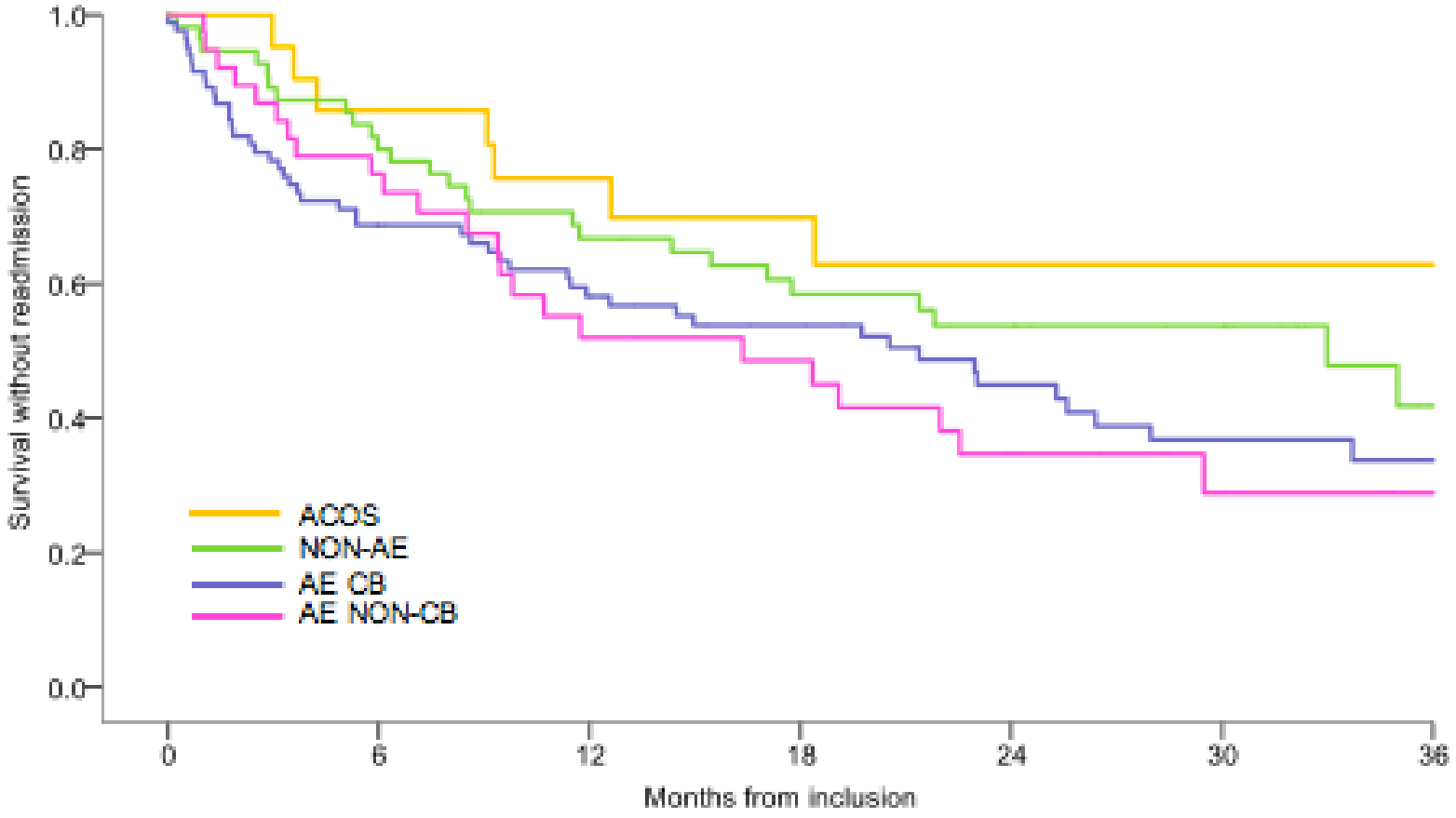
This article was published in the following Dove Press journal:
International Journal of COPD
10 November 2014
Number of times this article has been viewed

Barbora Novotna[1]

Purpose: Chronic obstructive pulmonary disease (COPD) has been recognized as a

Table 1. Survival without readmission for exacerbations of COPD (Czech multicentre research database of COPD patients) – only for patients with at least one hospitalization for exacerbation in last year before inclusion to registry (N=203)

	N	Median of survival (95% CI)	3-year survival (95% CI)
ACOS	N=24	-	0.628 (0.404–0.853)
NON-AE	N=56	33 (17–49)	0.418 (0.237–0.599)
AE CB	N=84	21 (12–31)	0.337 (0.213–0.461)
AE NON-CB	N=39	16 (6–27)	0.289 (0.116–0.462)



International Journal of COPD

Dovepress
open access to scientific and medical research

Open Access Full Text Article

ORIGINAL RESEARCH

Characteristics of COPD patients according to GOLD classification and clinical phenotypes in the Russian Federation: the SUPPORT trial

This article was published in the following Dove Press journal:
International Journal of COPD
3 November 2017
Number of times this article has been viewed

Vladimir Arkhipov[1]

Background: The high prevalence of COPD in the Russian Federation has been demonstrated

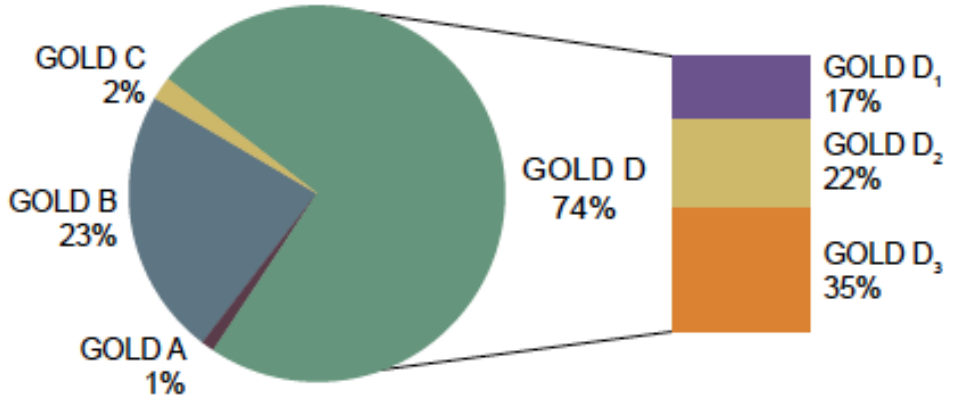
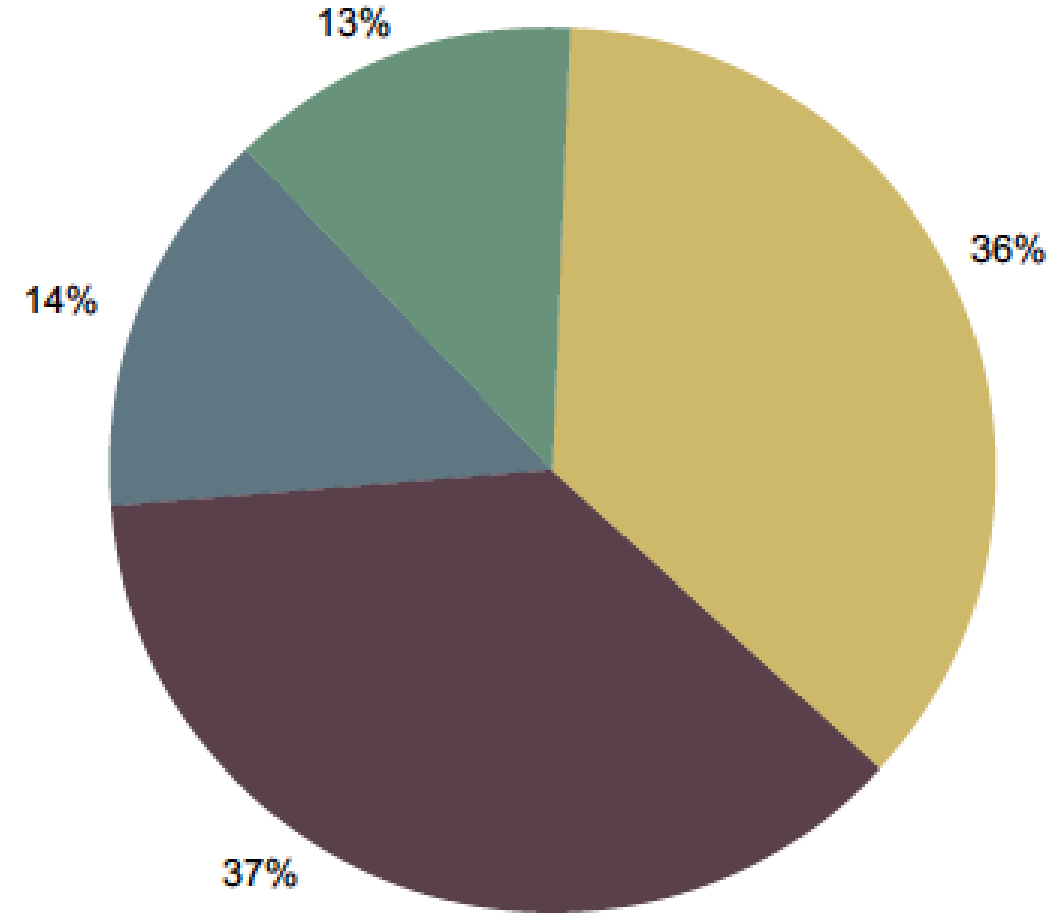


Figure 2 Distribution of patients by GOLD 2013 group.
Notes: GOLD A (low risk, fewer symptoms), GOLD B (low risk, more symptoms), GOLD C (high risk, fewer symptoms), or GOLD D (high risk, more symptoms). Patients in groups C and D were stratified into three subgroups as C_1, C_2, and C_3, and D_1, D_2, and D_3 based on the specific risk factor used to determine the group assignment: C_1 or D_1 (FEV_1 <50%), C_2 or D_2 (two or more exacerbations or one hospitalization), and C_3 or D_3 (both FEV_1 <50% and two or more exacerbations or one hospitalization within the last year).



BMJ Open Respiratory Research

Endobronchial valves for emphysema: an individual patient-level reanalysis of randomised controlled trials

Karin Klooster,[1] Dirk-Jan Slebos,[1] Zaid Zoumot,[2] Claire Davey,[3] Pallav L Shah,[3] Nicholas S Hopkinson[3]

To cite: Klooster K, Slebos D-J, Zoumot Z, *et al*. Endobronchial valves for emphysema: an individual patient-level reanalysis of randomised controlled trials. *BMJ Open Resp Res* 2017;**4**:e000214. doi:10.1136/bmjresp-2017-000214

Received 15 May 2017
Revised 10 October 2017
Accepted 11 October 2017

ABSTRACT

Introduction Endobronchial valve placement has potential as a treatment for patients with chronic obstructive pulmonary disease (COPD). However, a robust evidence base will be needed to convince commissioners of healthcare that it is a high-value treatment. We sought to develop the evidence base by performing an individual patient-level analysis of randomised controlled trials in people with heterogeneous emphysema and an absence of collateral ventilation.

Methods A literature search (PROSPERO register CRD42016048127) identified two trials meeting these criteria, the BelieVeR-HIFi and STELVIO studies. Anonymised individual patient data were obtained from investigators and analysed. The primary outcome measure was a comparison of change in forced expiratory volume in 1 s (FEV_1) from baseline between the treatment and control groups. Secondary end points were change from baseline

Key messages

- Can endobronchial valve placement improve health outcomes in selected patients with heterogeneous emphysema and an absence of collateral ventilation
- Endobronchial valves improve lung function, exercise capacity and health status at 3–6 months after the procedure
- Combining patient-level data from randomised controlled trials of bronchoscopic lung volume reduction with endobronchial valves strengthens the evidence that this therapy can improve lung function, exercise capacity and quality of life in appropriately selected patients with heterogeneous emphysema and absence of interlobar collateral ventilation.

International Primary Care IPCRG Respiratory Group

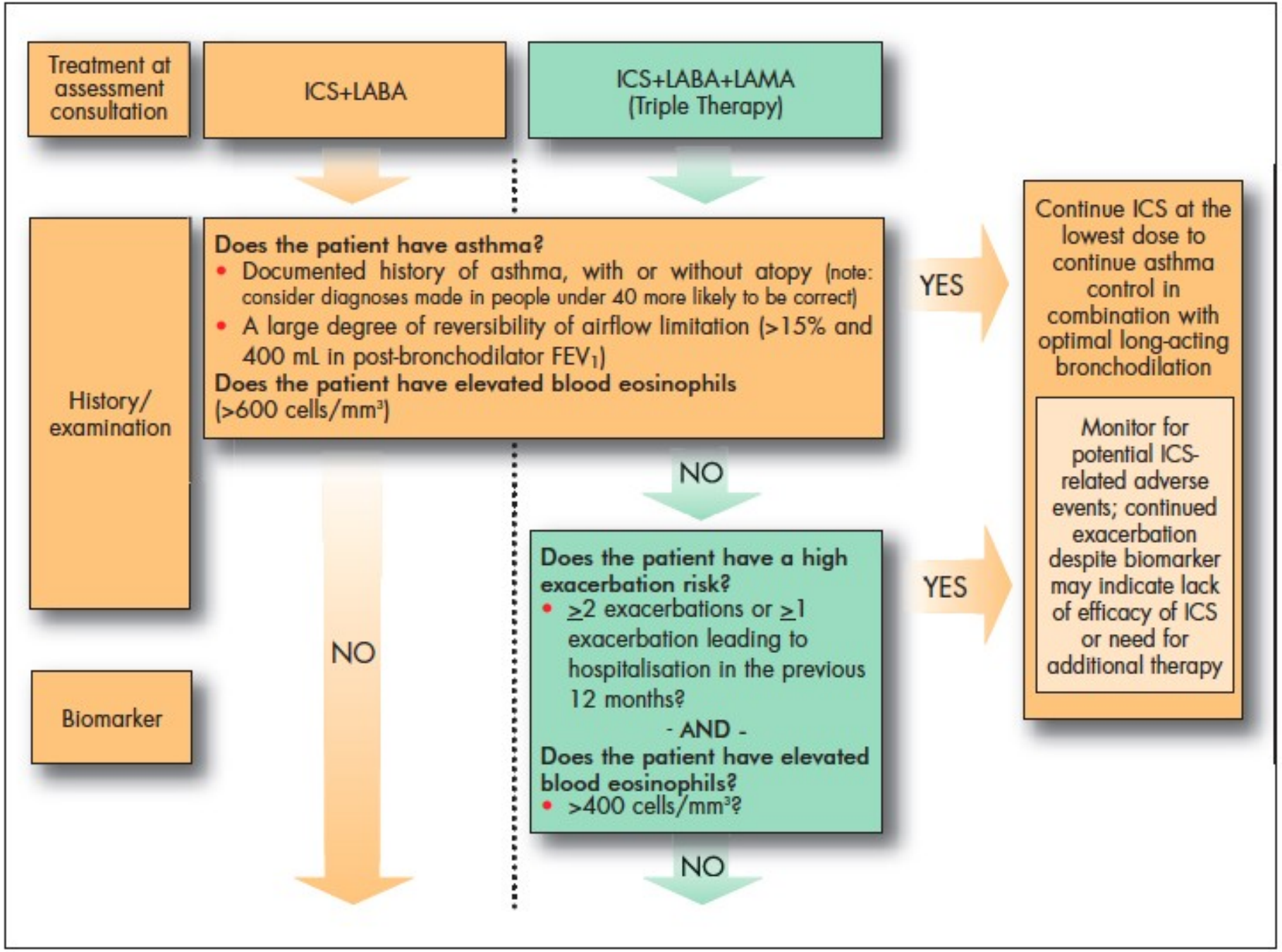
DESKTOP HELPER

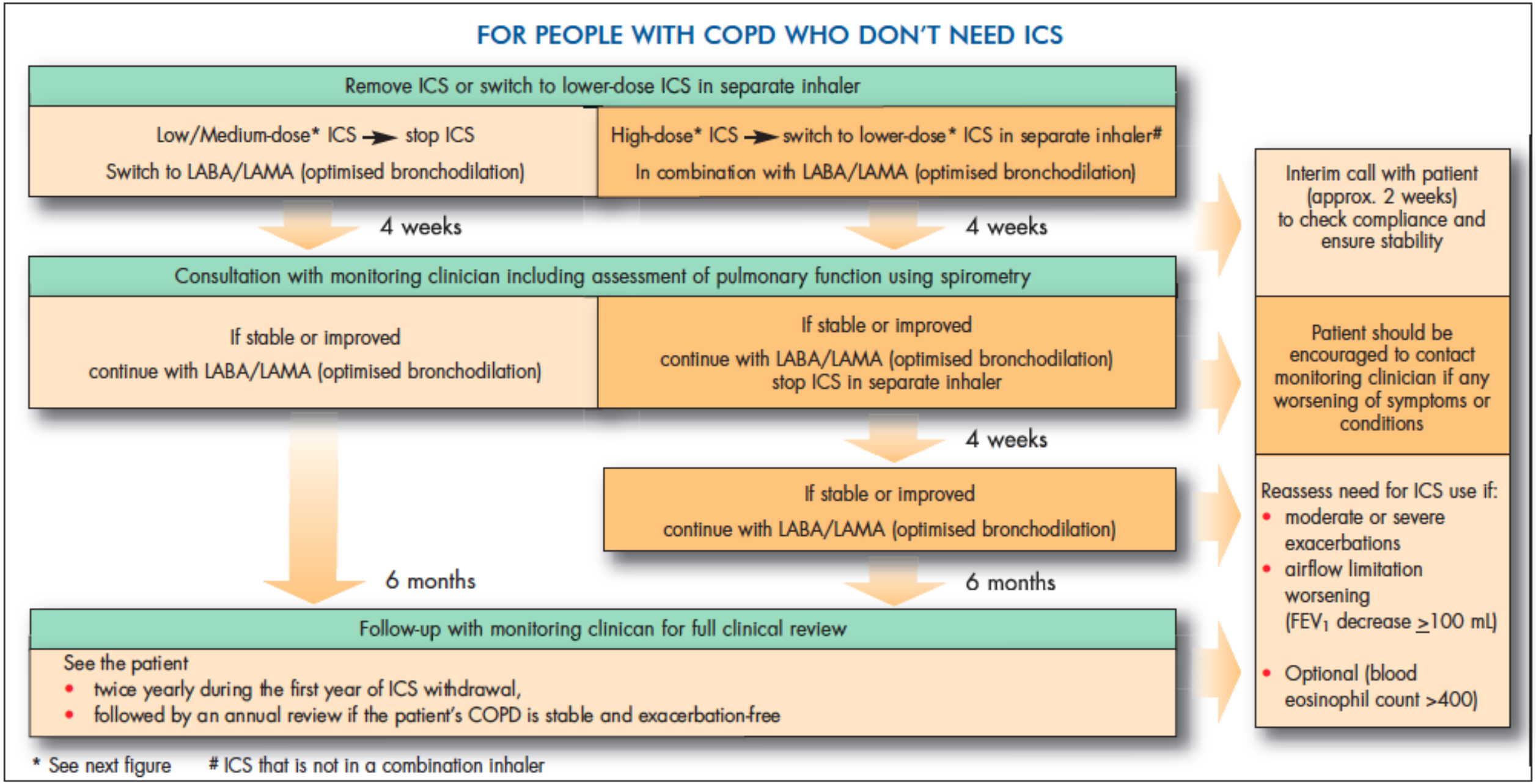
No. 6 March 2017

Evaluation of appropriateness of inhaled corticosteroid (ICS) therapy in COPD and guidance on ICS withdrawal

This guide provides an algorithm to identify people with chronic obstructive pulmonary disease (COPD) who might benefit from ICS treatment and those in whom it may not be appropriate, and an approach to withdrawing ICS in patients in whom it is not needed.

- In patients with COPD at low risk of exacerbation, bronchodilation should be the first-line treatment. [GOLD 2017]. In symptomatic patients on monotherapy, treatment can be stepped up to a combination long-acting β2-agonist plus long acting muscarinic antagonist (LABA+LAMA), and for patients with severe breathlessness initial therapy with LABA+LAMA may be considered [GOLD 2017].
- In patients at high risk of an exacerbation with few symptoms, the recommended first-line treatment is a LAMA (stepping up to LABA+LAMA if exacerbations ▶




FOR PEOPLE WITH COPD WHO DON'T NEED ICS
Remove ICS or switch to lower-dose ICS in separate inhaler
Low/Medium-dose* ICS → stop ICS
Switch to LABA/LAMA (optimised bronchodilation)
High-dose* ICS → switch to lower-dose* ICS in separate inhaler#
In combination with LABA/LAMA (optimised bronchodilation)
4 weeks
4 weeks
Consultation with monitoring clinician including assessment of pulmonary function using spirometry
If stable or improved
continue with LABA/LAMA (optimised bronchodilation)
If stable or improved
continue with LABA/LAMA (optimised bronchodilation)
stop ICS in separate inhaler
4 weeks
If stable or improved
continue with LABA/LAMA (optimised bronchodilation)
6 months
6 months
Follow-up with monitoring clinican for full clinical review
See the patient
twice yearly during the first year of ICS withdrawal,
followed by an annual review if the patient's COPD is stable and exacerbation-free
Interim call with patient (approx. 2 weeks) to check compliance and ensure stability
Patient should be encouraged to contact monitoring clinician if any worsening of symptoms or conditions
Reassess need for ICS use if:
moderate or severe exacerbations
airflow limitation worsening
(FEV1 decrease ≥100 mL)
Optional (blood eosinophil count >400)
* See next figure
# ICS that is not in a combination inhaler

Osnova

- Důvody proč se vůbec zabývat aktualizací
- **Hlavní body návrhu**
- II.Konsenzuální konference 16.11.2017
- Další postup tvorby DP 2018
- Spolupráce PNE s PL

GOLD 2017

- AKCEPTOVAT JAKO POMOCNOU KLASIFIKACI *(bez výhrad)*
- AKCEPTOVAT JEHO DEFINICI CHOPN
- VÝZNAM pro lékaře studií a pro ev. mezinárodní srovnání
- LÉČEBNÁ NAVIGACE pro ty co odmítají národní DP (legitimní)

DEFINICE

- **Chronická obstrukční plicní nemoc** (CHOPN) je léčitelné a preventabilní, klinicky heterogenní onemocnění, charakterizované převážně ireverzibilní bronchiální obstrukcí a obvykle provázené symptomy.
- **Bronchiální obstrukce** (BO) vzniká v důsledku abnormalit dolních dýchacích cest a/nebo plicních sklípků způsobených destrukčně-zánětlivou reakcí na inhalační expozici škodlivým částicím a plynům u geneticky predisponovaného jedince.
- Z patofyzilogického pohledu je BO způsobena zvýšením odporů dýchacích cest a elastického tlaku plic, které jsou důsledkem zánětlivého ztluštění stěny průdušek a úbytku elastických vláken plicního intersticia. CHOPN může být asociována s různě vyjádřenými komorbiditami.

SYMPTOMY

- CAT preferován
- ALTERNATIVY: mMRC, CRQ,..
- ANXIETA
- DEPRESE

FUNKČNÍ DIAGNOSTIKA

- FEV1/VC < LLN
- FEV1/VC<0.7
- FEV1/FVC<0.7
- TLco, Kco
- RV, RV/TLC, IC/TLC
- IOS,..

FENOTYPY

- Ponechat jako nálepky označující léčebnou možnost
- **KACHEXIE** téměř není bez EMFYZÉMU (spojení do jedné kategorie?)
- BE nejsou vždy spojeny s CH.BRONCHITIDOU (**BCOS** je legitimní - CT)
- **ACO** (ACOS) je realita (nicméně spíše málo častá)
- **Frekventní AE** ano (frekvence?)
- **EMFYZÉM** (jistě má cenu hledat – jaká CT a funkční kritéria)
- **CHRONICKÁ BRONCHITIDA** – klinicky jednoduché

ACO (ACOS)

Citace: Miravitlles et al. Arch Bronconeumol. 2017;53(6).
Miravitlles et al. Eur Respir J. 2017 ;49(5).

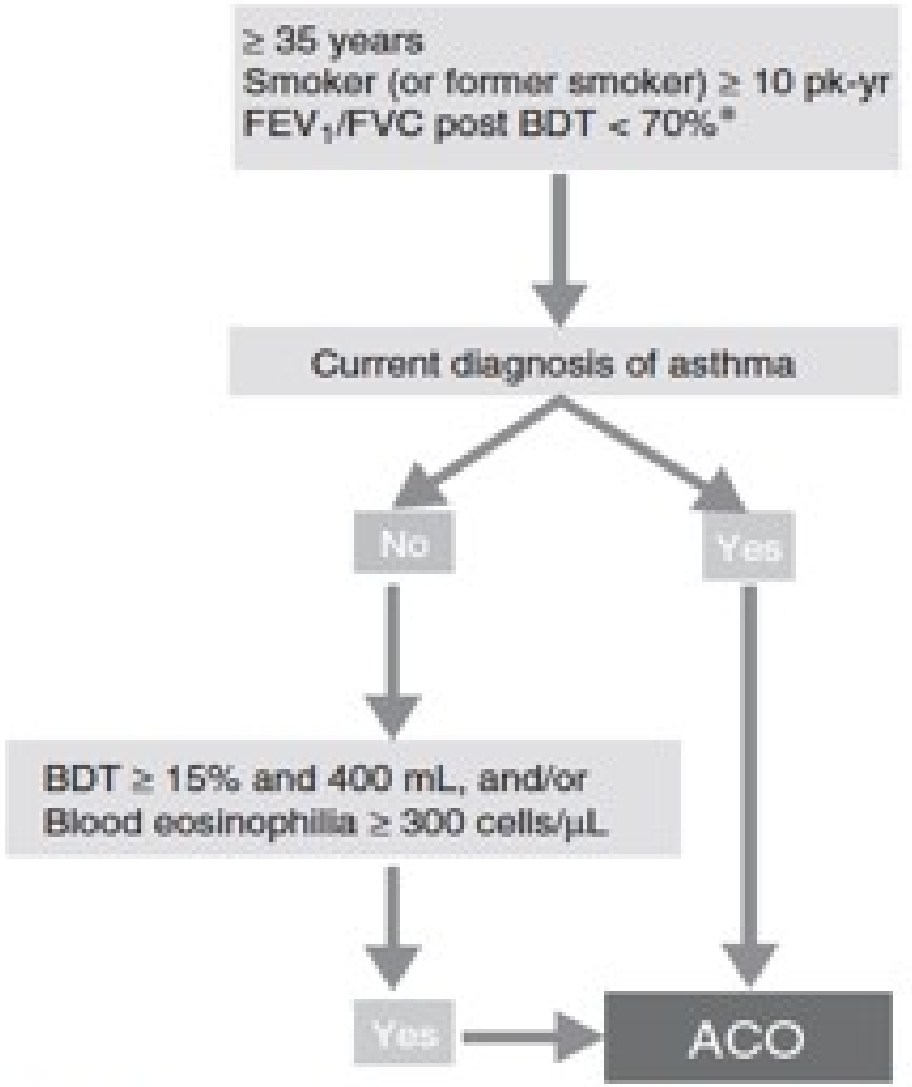
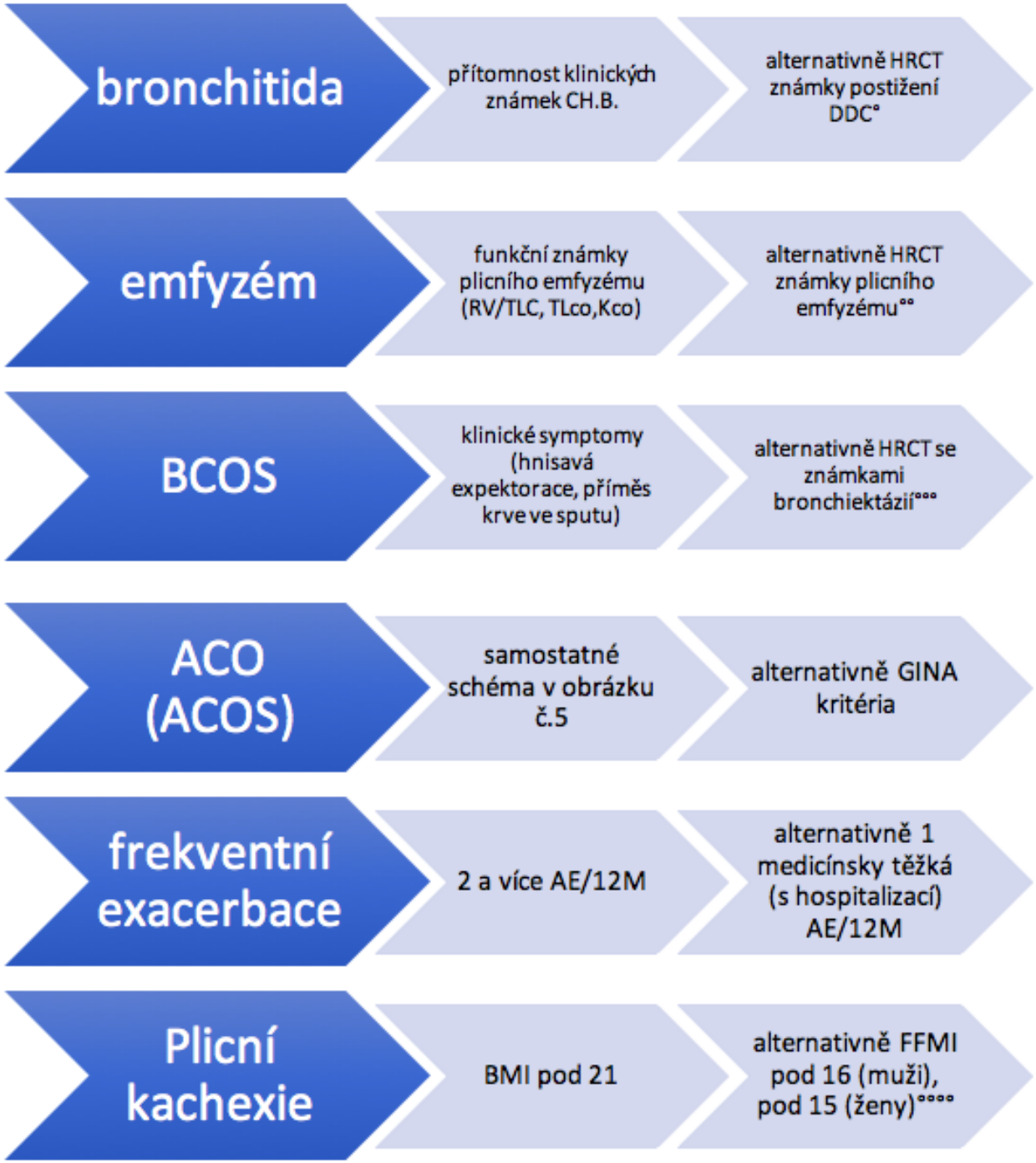
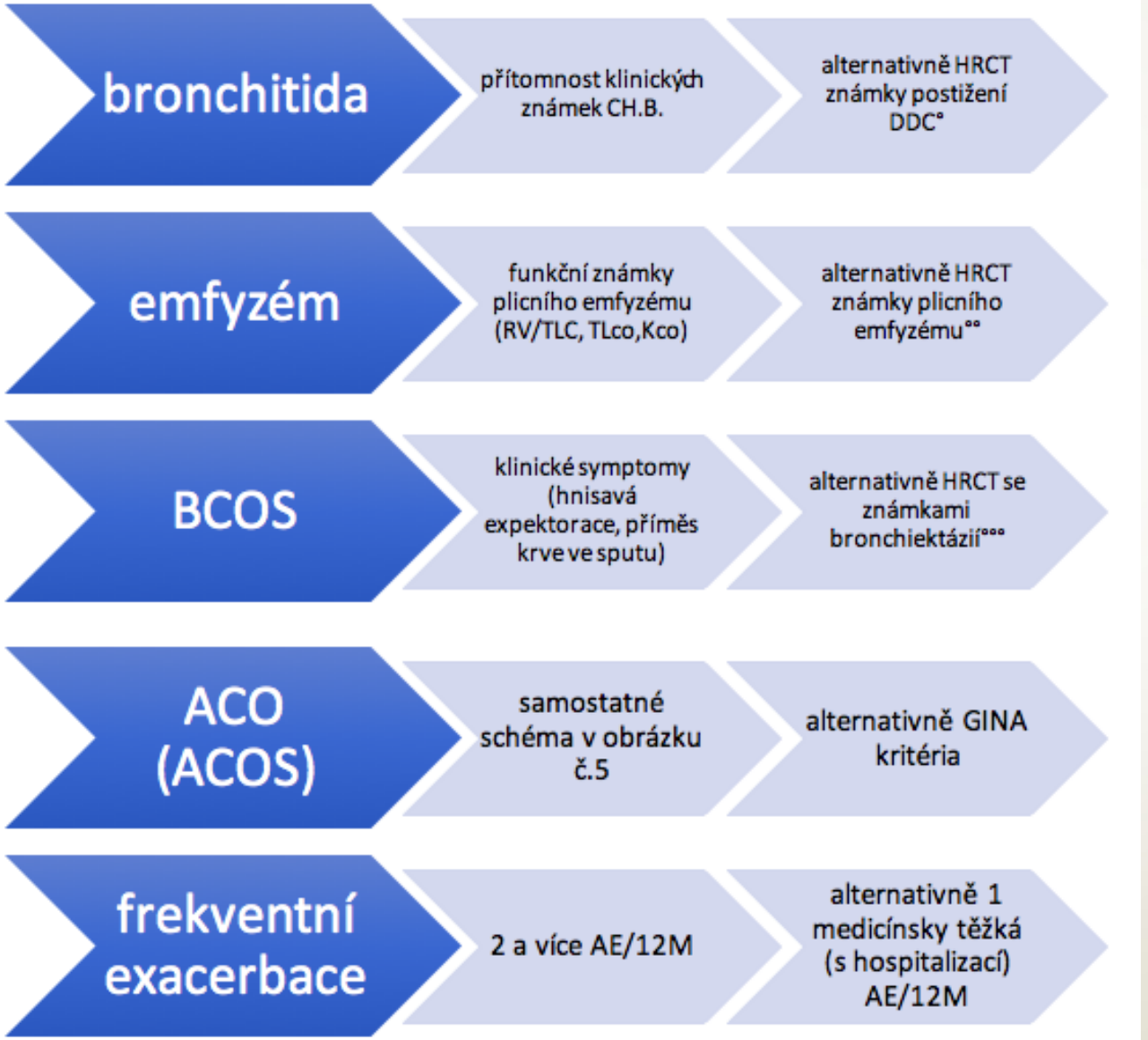


Fig. 3. Diagnostic algorithm for COPD according to GesEPOC-GEMA (Spanish COPD-Asthma Management guidelines) consensus. * Maintained after treatment with ICS/LABA (6 months). In some cases, also after a cycle of oral glucocorticoids (15 days). ACO – asthma-COPD overlap; BDT – bronchodilator test; ICS – inhaled corticosteroids; LABA – long-acting β_2 agonist; Pk-yr – pack-years.


bronchitida
přítomnost klinických známek CH.B.
alternativně HRCT známky postižení DDC°
emfyzém
funkční známky plicního emfyzému (RV/TLC, TLco,Kco)
alternativně HRCT známky plicního emfyzému°°
BCOS
klinické symptomy (hnisavá expektorace, příměs krve ve sputu)
alternativně HRCT se známkami bronchiektázií°°°
ACO (ACOS)
samostatné schéma v obrázku č.5
alternativně GINA kritéria
frekventní exacerbace
2 a více AE/12M
alternativně 1 medicínsky těžká (s hospitalizací) AE/12M
Plicní kachexie
BMI pod 21
alternativně FFMI pod 16 (muži), pod 15 (ženy)°°°°

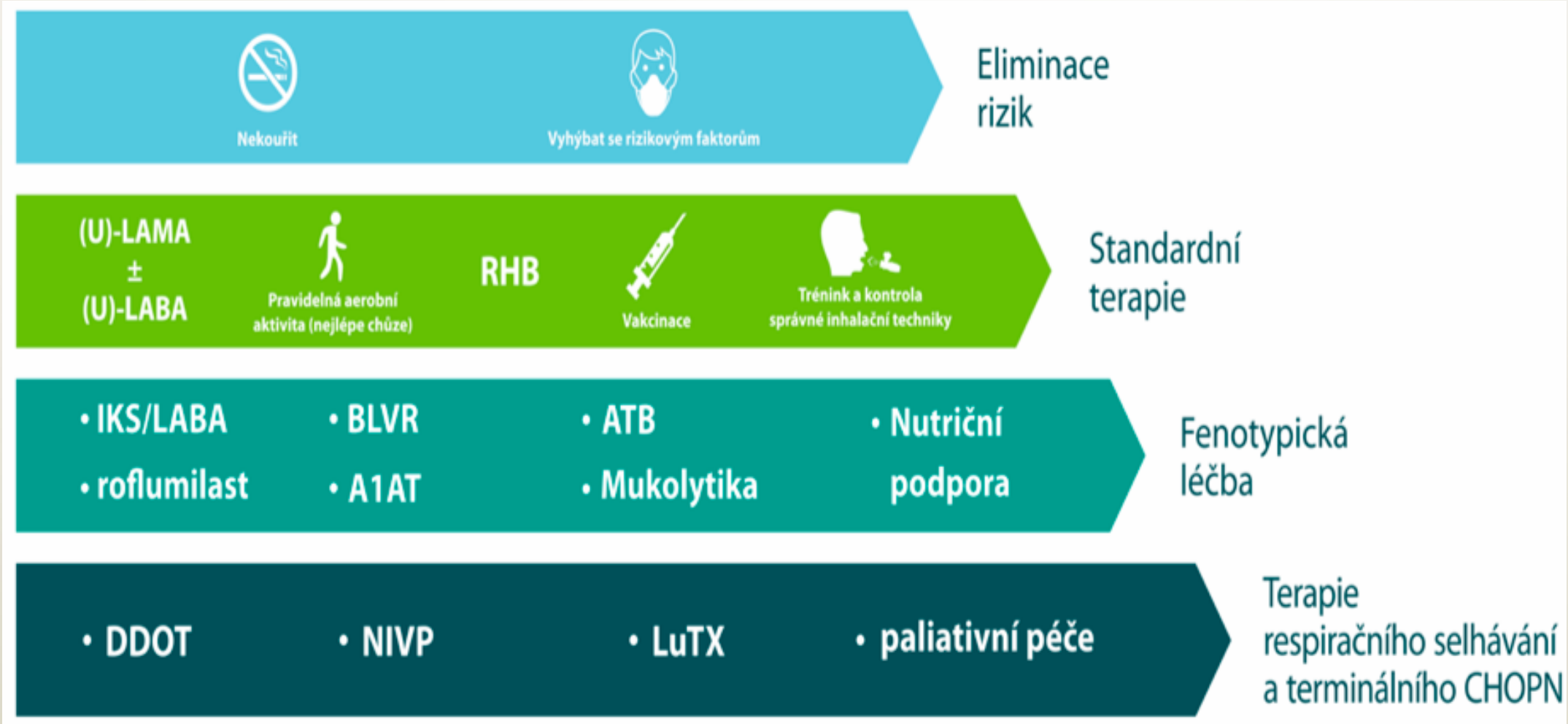

bronchitida
přítomnost klinických známek CH.B.
alternativně HRCT známky postižení DDC°
emfyzém
funkční známky plicního emfyzému (RV/TLC, TLco,Kco)
alternativně HRCT známky plicního emfyzému°°
BCOS
klinické symptomy (hnisavá expektorace, příměs krve ve sputu)
alternativně HRCT se známkami bronchiektázií°°°
ACO (ACOS)
samostatné schéma v obrázku č.5
alternativně GINA kritéria
frekventní exacerbace
2 a více AE/12M
alternativně 1 medicínsky těžká (s hospitalizací) AE/12M

KLASIFIKACE CHOPN

POMOCNÉ DOPLŇKY KLASIFIKACE

- **CHOPN** + FAST DECLINE
- **CHOPN** + OSA
- **CHOPN** + OHS
- **CHOPN** + TBM/EDAC
- **CHOPN** + plicní fibroza

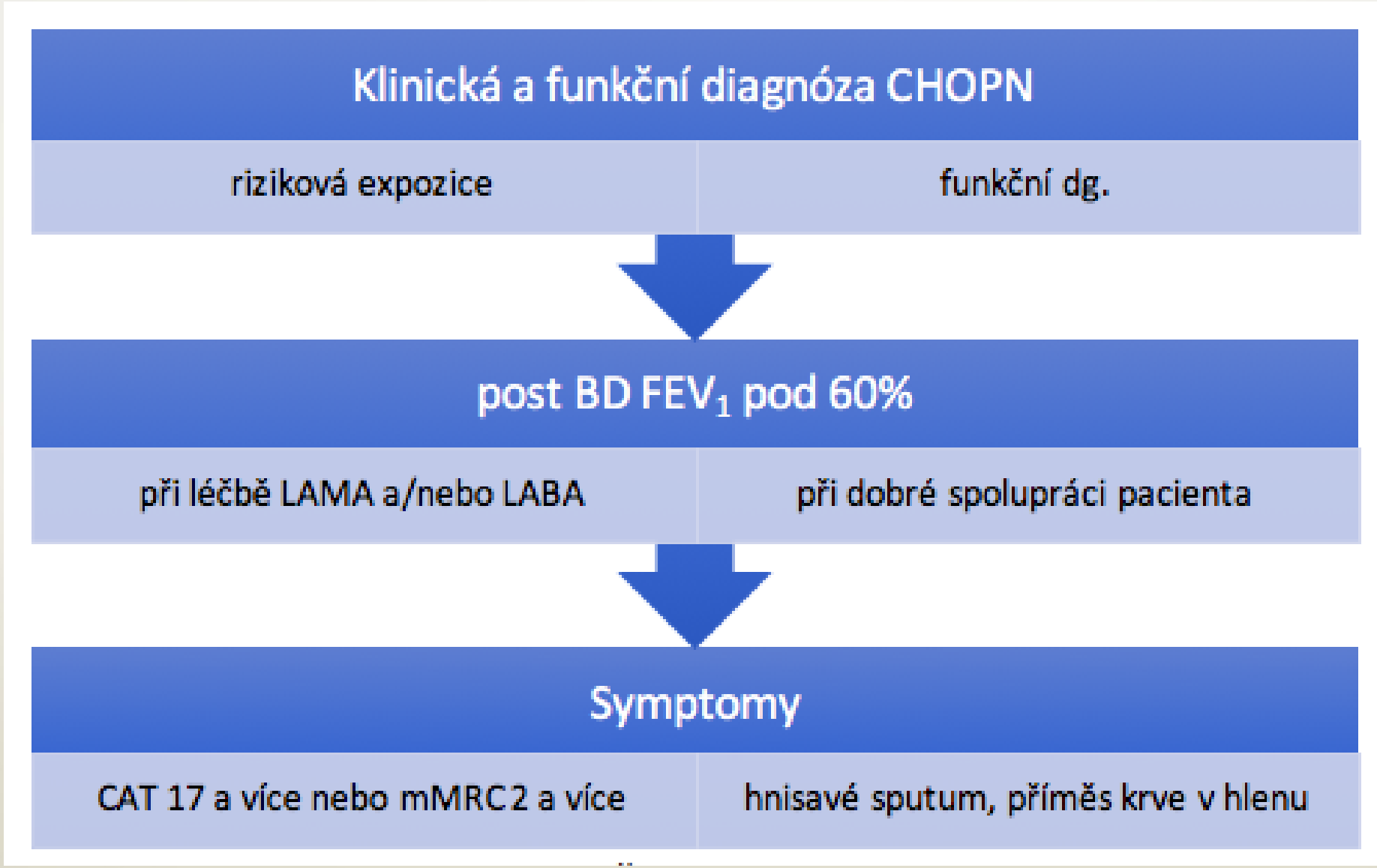
PONECHAT - SCHÉMA TERAPIE *(EMB)*



INICIÁLNÍ CHOPN BALÍČEK

- A1AT (vstupně)
- FUNKČNÍ TESTY (*spirometrie po BD je pouze základ*)
- KORTIKOIDNÍ TEST (zejména pokud lehká OVP)
- EKG + RTG (vstupně)
- KO + dif (vstupně)
- CT hrudníku (při zhoršení nebo suspekci na EMF nebo BCOS ?)

INDIKACE CT


Klinická a funkční diagnóza CHOPN
riziková expozice
funkční dg.
post BD FEV_1 pod 60%
při léčbě LAMA a/nebo LABA
při dobré spolupráci pacienta
Symptomy
CAT 17 a více nebo mMRC 2 a více
hnisavé sputum, příměs krve v hlenu

Osnova

- Důvody proč se vůbec zabývat aktualizací
- Hlavní body návrhu
- **II.Konsenzuální konference 16.11.2017**
- Další postup tvorby DP 2018
- Spolupráce PNE s PL

Vážená paní doktorko, vážený pane doktore,
dovolujeme si Vás pozvat na

II. Konsenzuální konferenci o CHOPN

Odborný garant: MUDr. Vladimír Koblížek, Ph.D.

16. 11. 2017

Fakultní nemocnice Hradec Králové

Výukové centrum
LF UK HK
(Velká posluchárna)

PROGRAM

16.00 – 16.10 h **CHOPN jako významný celospolečenský problém**
prof. MUDr. Vítězslav KOLEK, DrSc. • FN Olomouc

16.10 – 16.20 h **Úvodní hlasování o názorech pneumologů** sli.do (hlasování pomocí)
MUDr. Vladimír KOBLÍŽEK, Ph.D. • FN Hradec Králové

16.20 – 16.30 h **Doporučení napříč Evropou**
MUDr. Jaromír ZATLOUKAL, Ph.D. • FN Olomouc

16.30 – 16.50 h **Funkční vyšetření plic v diagnostice CHOPN**
MUDr. Jan CHLUMSKÝ, Ph.D. • První plicní ambulance s.r.o., Praha
(LLN, možnosti časné diagnostiky, kortikoidní test a další)

17.00 – 17.10 h	Benefit ukončení škodlivé inhalace (kouření a další vlivy) – důkazy o vlivu na deklinaci plicních funkcí a na mortalitu MUDr. Vladimír KOBLÍŽEK, Ph.D. • FN Hradec Králové
17.10 – 17.30 h	Důkazy pro fenotypovou/cílenou léčbu – proč jsou LAMA/LABA standard a ostatní FENOTYPOVÁ terapie – evidence ze studií pro IKS, mukoaktivní léky, ATB, roflumilast a další léky MUDr. Jaromír ZATLOUKAL, Ph.D. • FN Olomouc
17.30 – 17.40 h	Důkazy pro přínos RHB péče a pravidelného pohybu Mgr. Kateřina NEUMANNOVÁ, Ph.D. • Katedra fyzioterapie, FTK UP Olomouc
17.40 – 17.50 h	Důkazy o přínosu očkování, BVR, LVRS a Tx MUDr. Vladimír KOBLÍŽEK, Ph.D. • FN Hradec Králové
17.50 – 18.00 h	Důkazy o přínosu A1AT augmentační terapie a DDOT MUDr. Jan CHLUMSKÝ, Ph.D. • První plicní ambulance s.r.o., Praha
18.00 – 18.10 h	Přestávka
18.10 – 18.20 h	Důkazy pro domácí NIVP MUDr. Ondřej KUDELA • FN Hradec Králové
18.20 – 18.30 h	Důkazy a návod pro paliativní péči MUDr. Michal HRNČIARIK • FN Hradec Králové
18.30 – 19.00 h	Souhrn ČPFS DP diagnostiky a terapie CHOPN MUDr. Vladimír KOBLÍŽEK, Ph.D. • FN Hradec Králové
19.00 – 19.20 h	Diskuse
19.20 – 19.30 h	Závěrečné hlasování / Ukončení konference sli.do hlasování pomocí

REGISTRUJTE SE
k osobní účasti na konferenci nebo k on-line webináři na:
www.amepra.cz/chopn

Přístupový kód pro hlasování sli.do obdržíte po registraci

Akce je pořádána Plicní klinikou Fakultní nemocnice Hradec Králové. Vzdělávací akce je pořádána dle stavovského předpisu č. 16 České lékařské komory, je řazena do systému celoživotního vzdělávání lékařů a je ohodnocena **3 kredity**.

Osnova

- Důvody proč se vůbec zabývat aktualizací
- Hlavní body návrhu
- II.Konsenzuální konference 16.11.2017
- **Další postup tvorby DP 2018**
- Spolupráce PNE s PL

Další postup tvorby DP 2018

- Listopad 2017 – výstup KONSENZUÁLNÍ konference **pro členy ČPFS**
- Prosinec 2017, Leden 2018 – **emailová komunikace členů ČPFS**
- Únor 2018 – **precizace DP Sekcí BO**
- Březen a Duben 2018 – **finální dokument (HPD 2018)**

Osnova

- Důvody proč se vůbec zabývat aktualizací
- Hlavní body návrhu
- II.Konsenzuální konference 16.11.2017
- Další postup tvorby DP 2018
- **Spolupráce PNE s PL**

Co mají PNE pro PL?

- Možnost LAMA+LABA
- Sdílení péče o lehké pacienty
- Vyšetření funkce plic u PNE (1x ročně)
- Spoluúčast PL na pilotním projektu screeningu
- Pátrání po CHOPN jako po komrbiditě (ICHS)

Závěr DP

- **Kultivace** nikoliv ~~destrukce a nové budování~~ DP
- **Žádné revoluční změny**
- **Důraz na** funkční, A1AT, CT, RHB, BD léky,..
- Ponechání **fenotypového pohledu**
- Snaha o **personalizovaný přístup** (PNE specialisté)
- Krátká verze pro PL