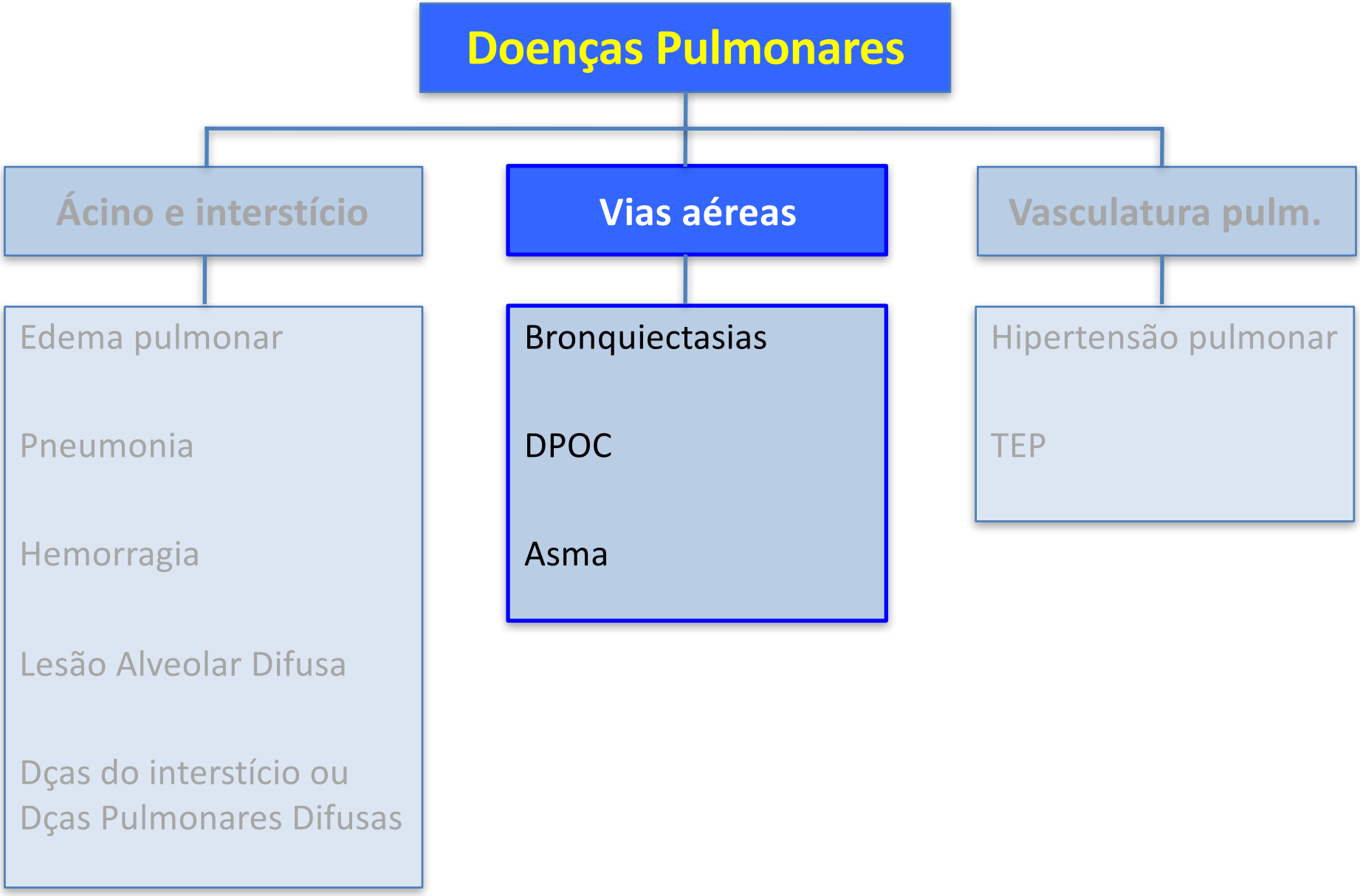
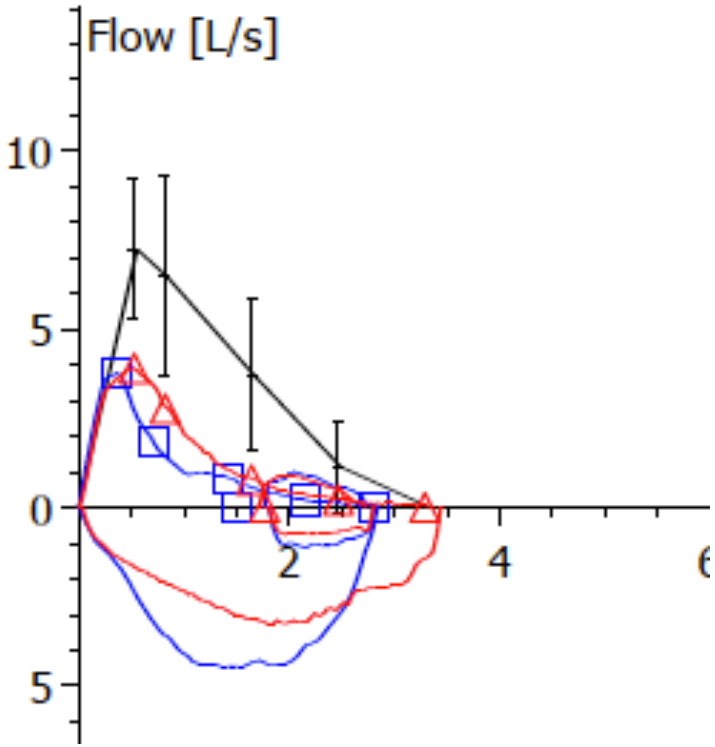
DPOC

heldernovaisbastos.pt
pneumologia


Doenças Pulmonares
Ácino e interstício
Vias aéreas
Vasculatura pulm.
Edema pulmonar
Pneumonia
Hemorragia
Lesão Alveolar Difusa
Dças do interstício ou
Dças Pulmonares Difusas
Bronquiectasias
DPOC
Asma
Hipertensão pulmonar
TEP

Diagnóstico

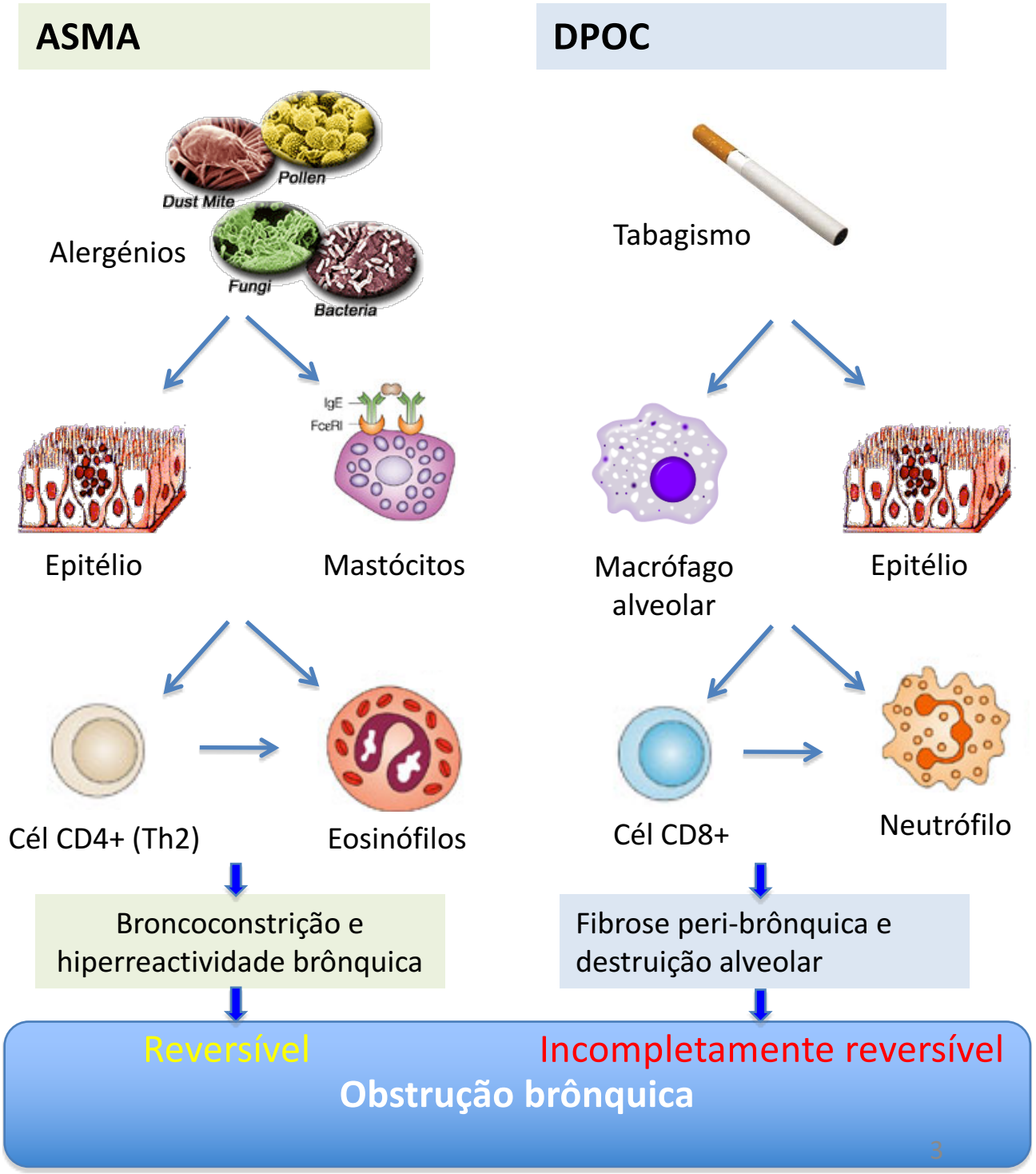


18.9%
280mL

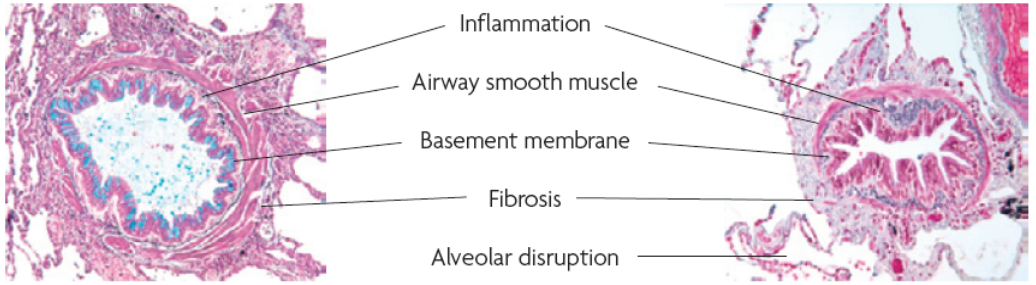
		Prev	Pré	%Pré/Prev	Pós	%Pós/Prev
FVC	**[L]**	3.32	2.81	84.5	3.28	98.9
FEV 1	**[L]**	2.55	1.48	58.3	1.76	69.3
FEV 1 % FVC	**[%]**		52.92		53.74	
MMEF 75/25	**[L/s]**	2.85	0.56	19.5	0.57	20.0
MEF 25	**[L/s]**	1.12	0.19	17.2	0.19	17.0
MEF 50	**[L/s]**	3.70	0.81	21.9	0.76	20.6
MEF 75	**[L/s]**	6.48	1.81	27.9	2.77	42.7

ASMA:
Hipertrofia/hiperplasia músculo liso brônquico e broncoconstrição

ASMA e DPOC:
Hipersecreção de muco
Exsudado inflamatório
Fibrose peribrônquica
(processo crónico – DPOC, *remodeling* na asma)



Asma vs DPOC



Inflamação	Tipo Th2 Mastócitos Eosinófilos Céls T $CD4^+$ Macrófagos	Tipo Th1 Neutrófilos Macrófagos Céls T $CD8^+$ Eosinófilos (ACOS?)
Resposta à corticoterapia	++	Variável
Músculo liso	+++	+
Fibrose	+ (subepitelial)	+++ (peribrônquica)
Destruição alveolar (parêqnuima)	Ausente	+++
Vasos sanguíneos	++	Normal
Obstrução brônquica	Reversível	Irreversível
Hiperreactividade brônquica	Significativa	Variável

DPOC

DPOC

International Journal of COPD 2006:1(3) 219–233

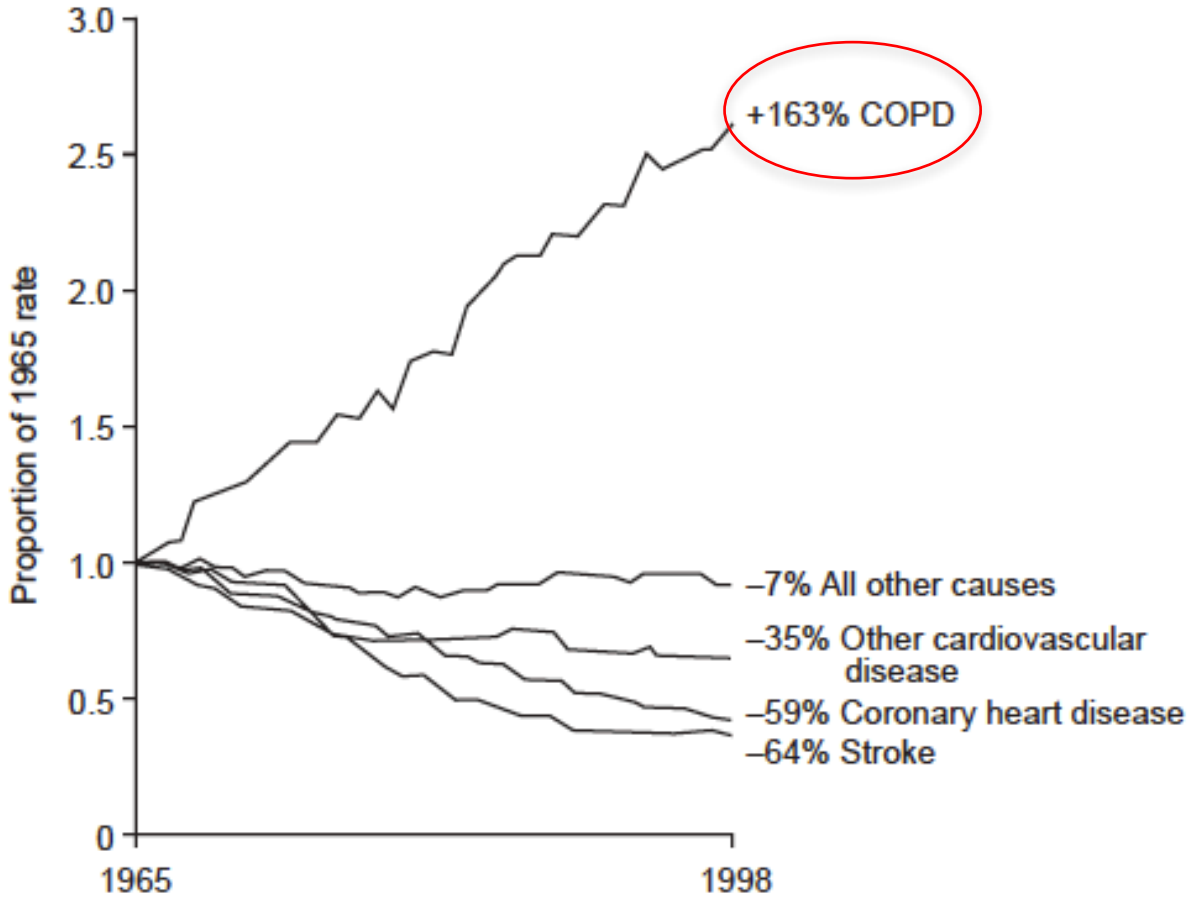
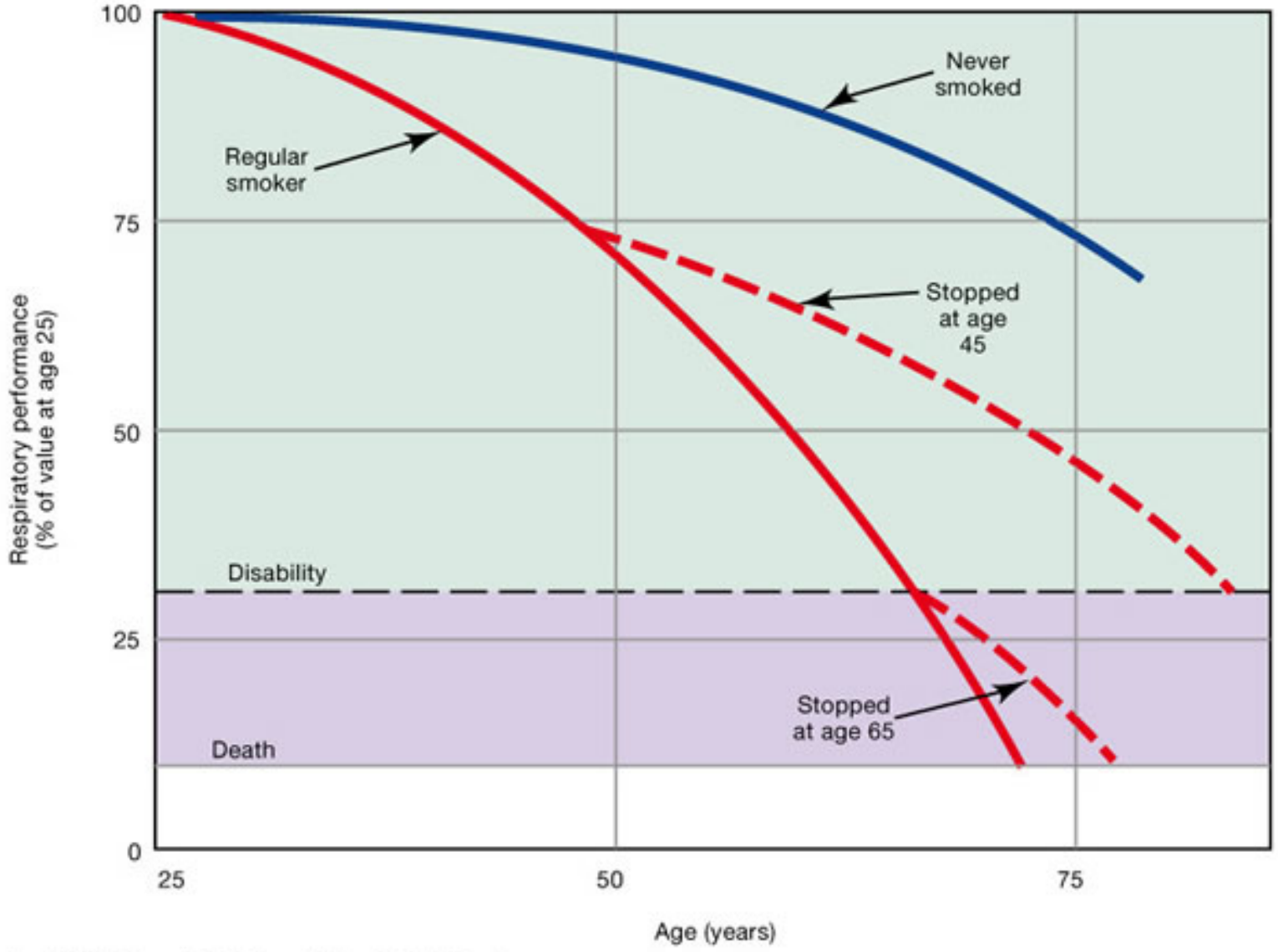


Figure 1 Change in age-adjusted death rates for COPD and other leading causes of death in the USA from 1965 to1998. Reprinted from Pauwels RA, Rabe KF. 2004. Burden and clinical features of chronic obstructive pulmonary disease (COPD). *Lancet*, 364:613–20. Copyright © 2004 with permission from Elsevier.

DPOC



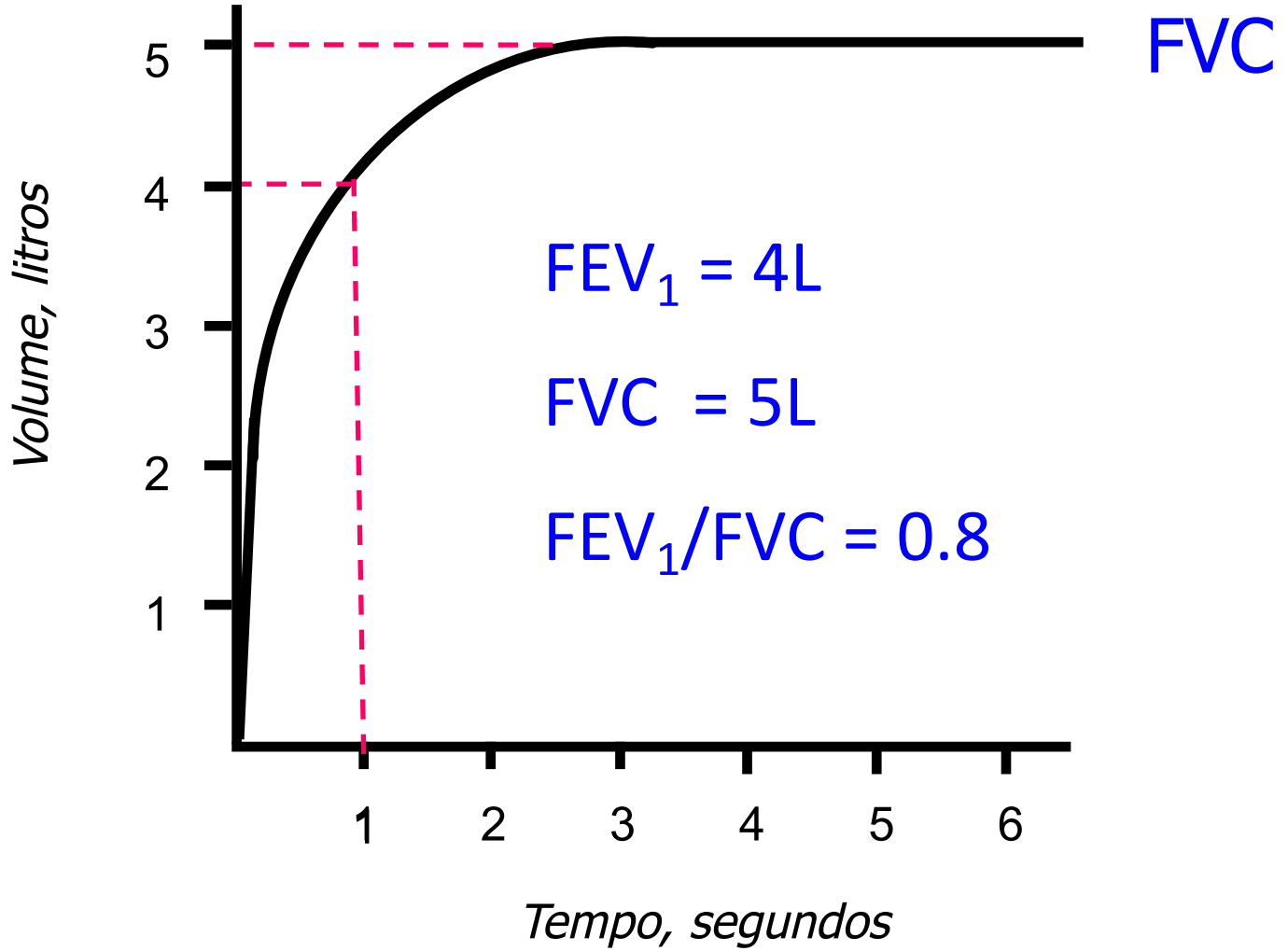
DPOC

Global Strategy for Diagnosis, Management and Prevention of COPD

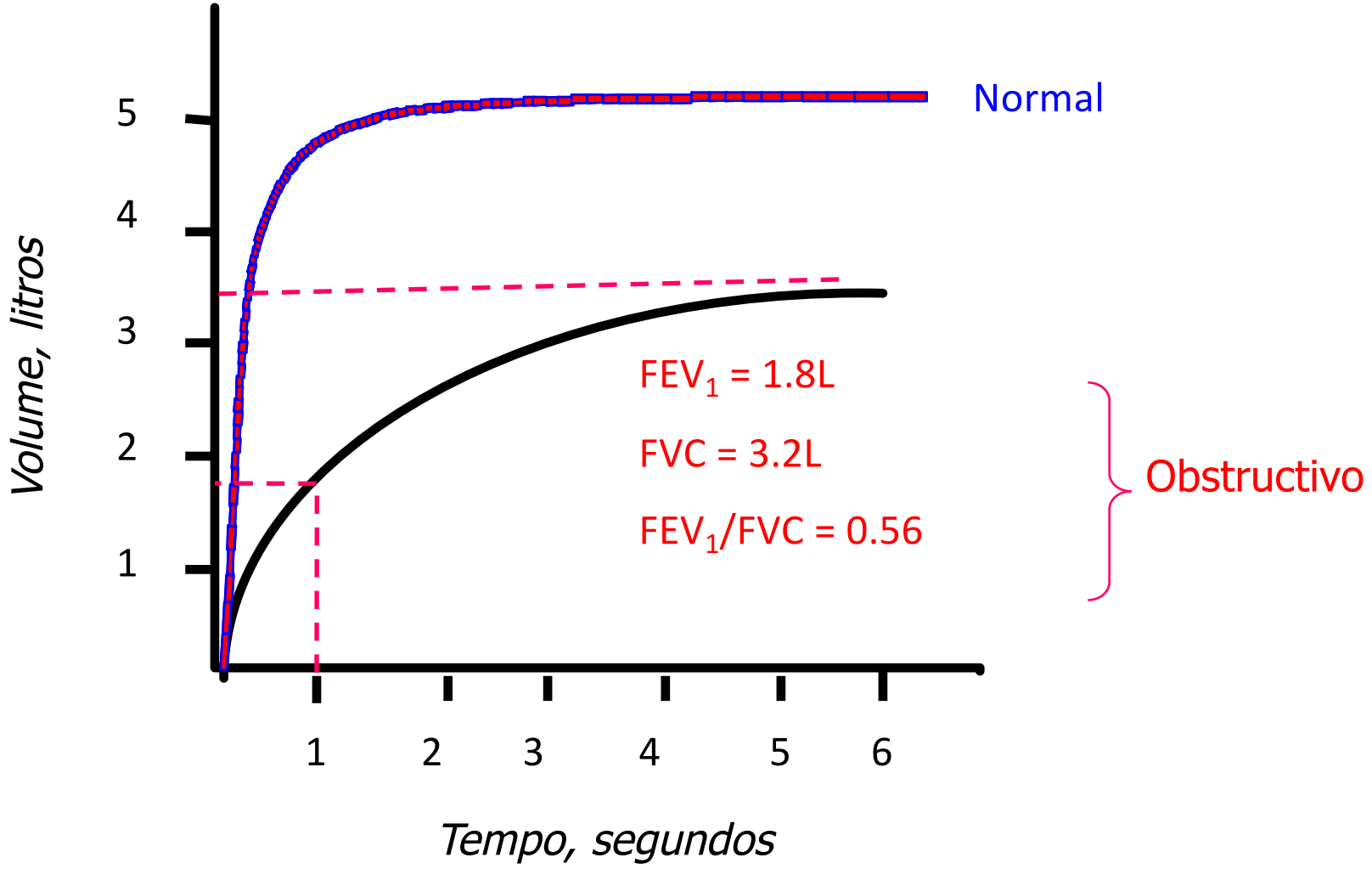
"COPD, a common preventable and treatable disease, is characterized by ***persistent airflow limitation*** *that is* ***usually progressive*** *and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases. Exacerbations and comorbidities contribute to the overall severity in individual patients."*

FEV1/FVC < 0.70

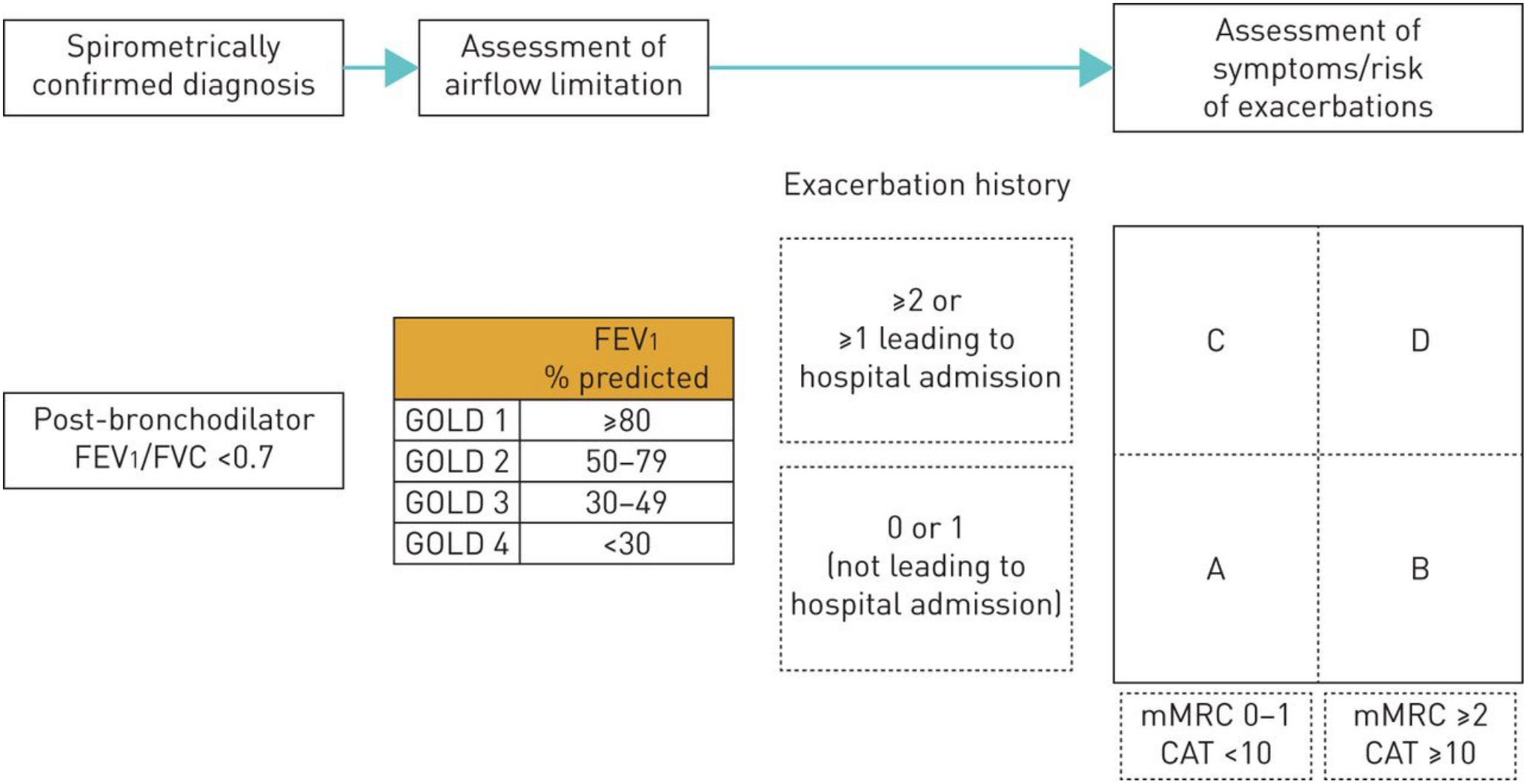
DPOC | Diagnóstico



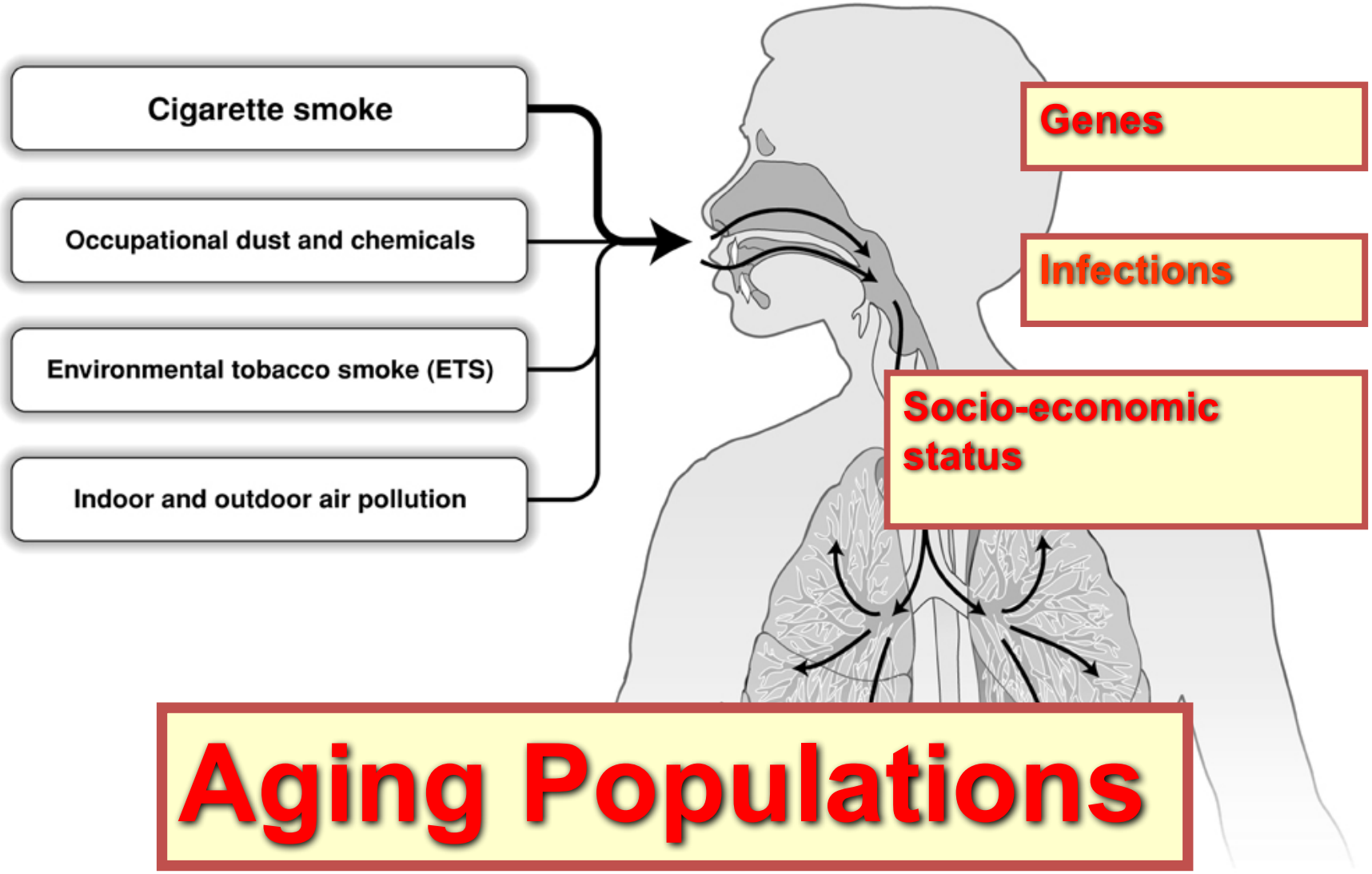
DPOC | Diagnóstico



DPOC | Classificação clínico-funcional



DPOC | Factores de risco



Bronquite crónica **Enfisema pulmonar**

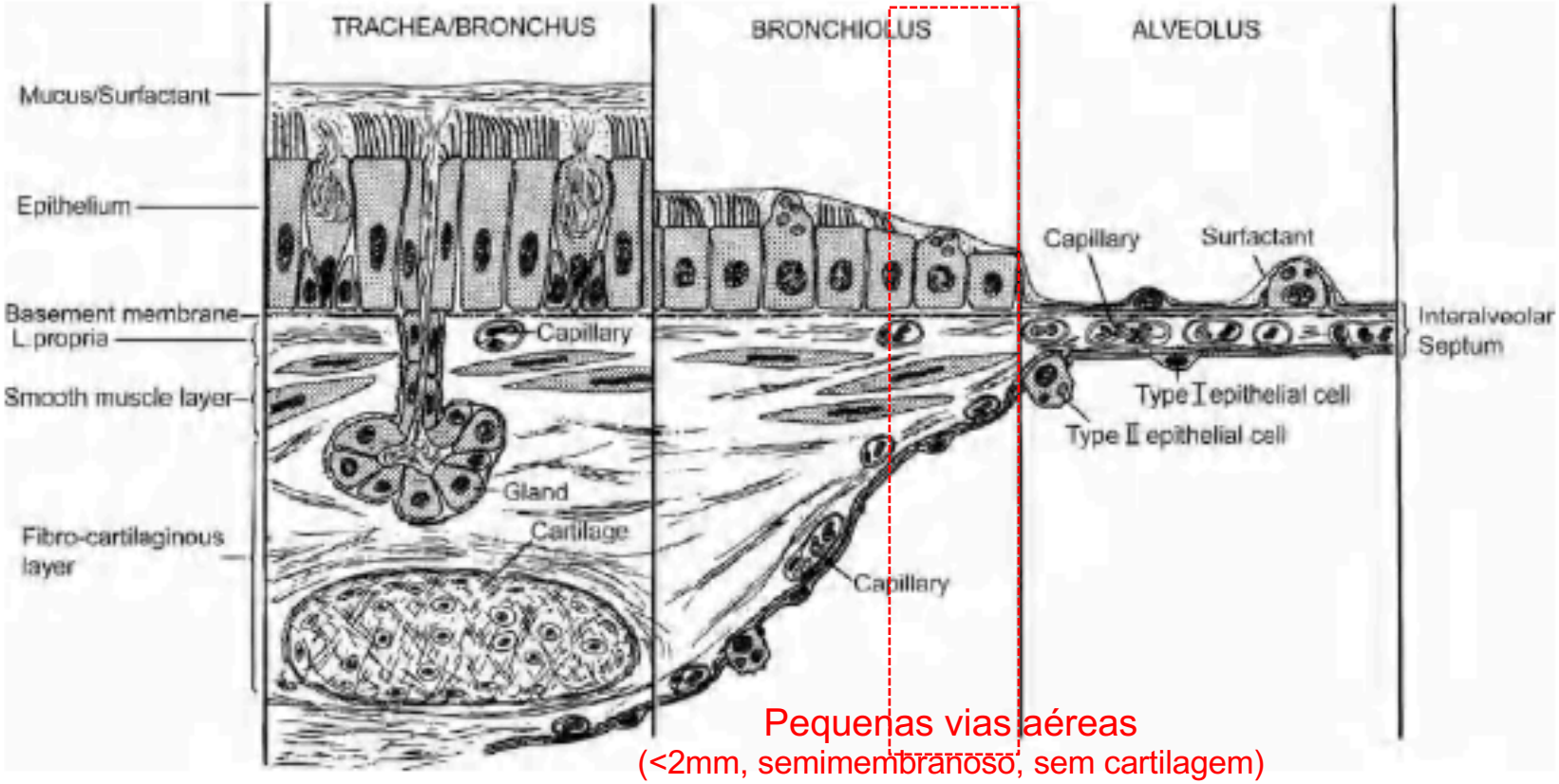
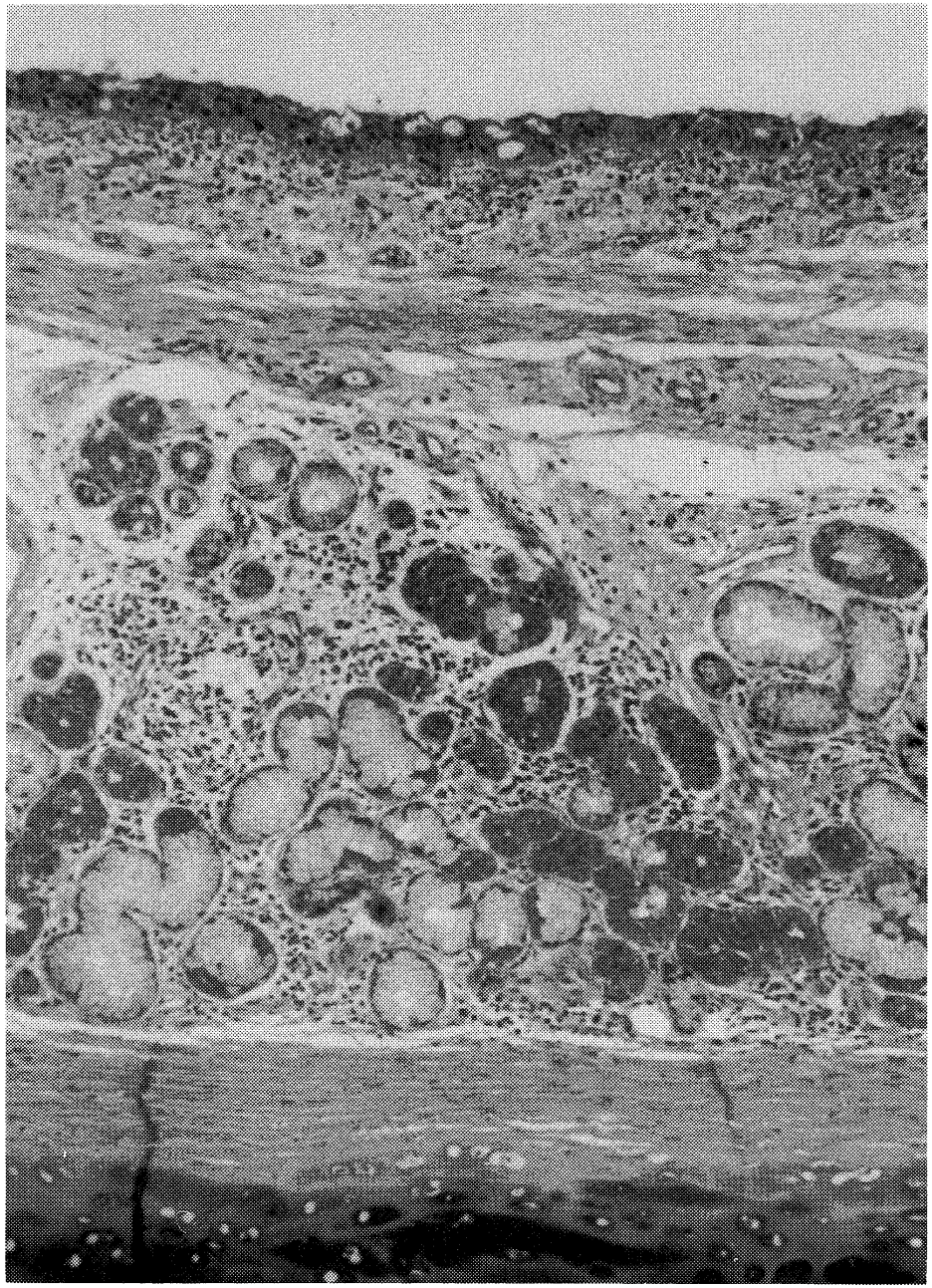


Figure 2-6 Airway wall structure at the three principal levels. The epithelial layer gradually becomes reduced from pseudostratified to cuboidal and then to squamous but retains its organization as a mosaic of lining and secretory cells. The smooth muscle layer disappears in the alveoli. The fibrous layer contains cartilage only in bronchi and gradually becomes thinner as the alveolus is approached.

Fishman's Pulmonary Diseases and Disorders



Internal Medicine 2002;41:265-269

Figure 1. The mucous glands of a patient with COPD form a much higher proportion of the bronchial wall (HE stain, ×150).

in Fishman's Pulmonary Diseases and Disorders

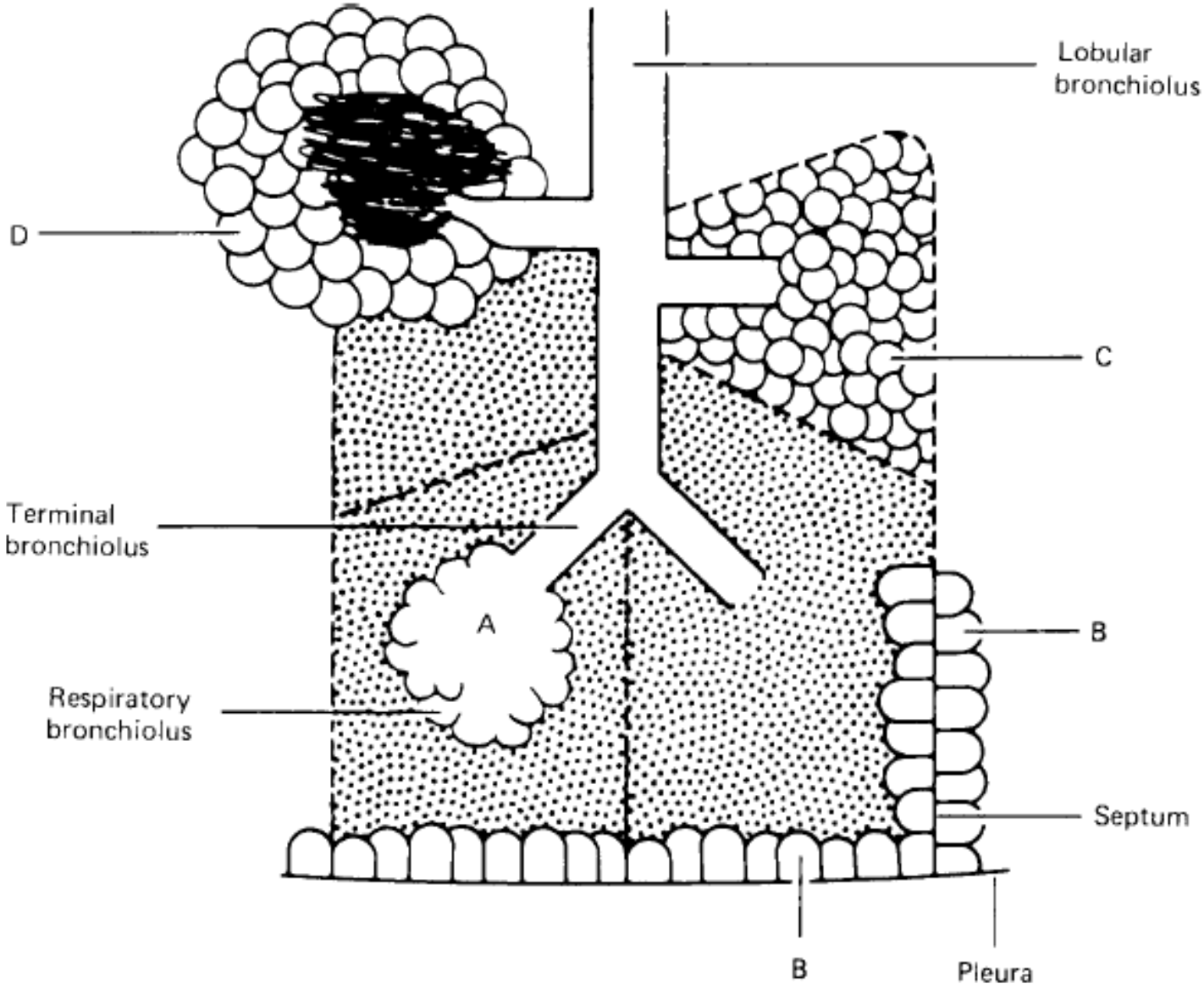
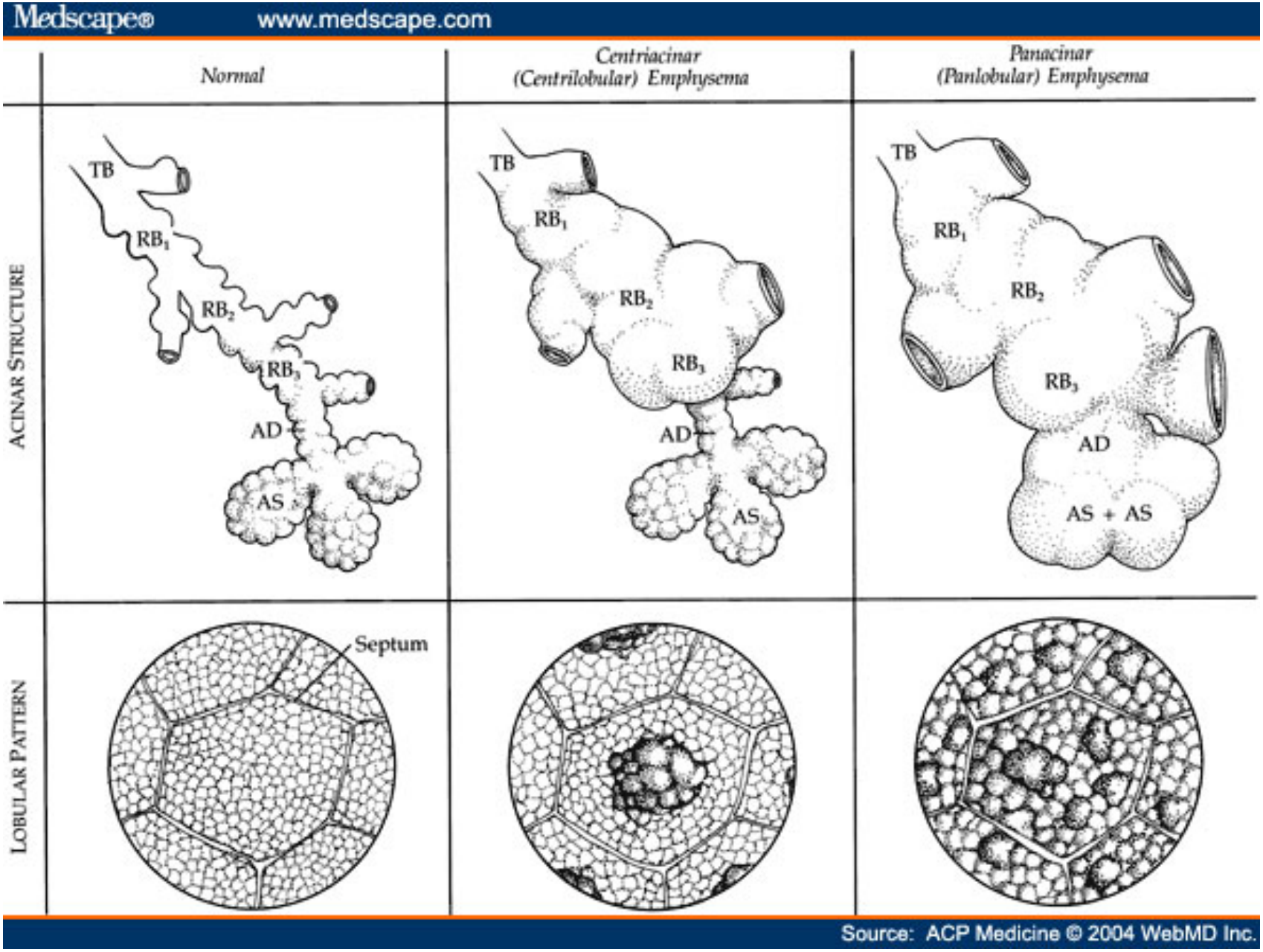
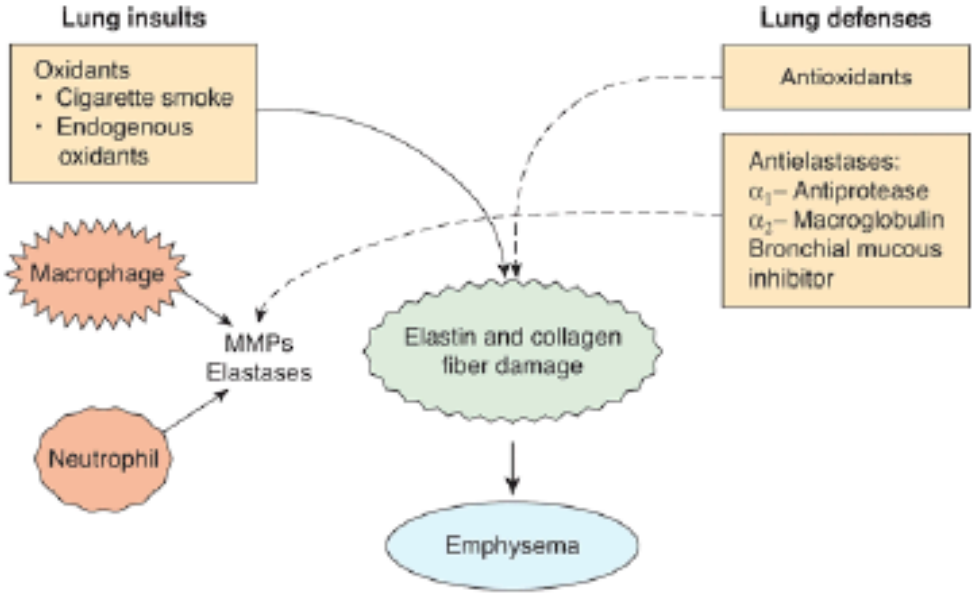


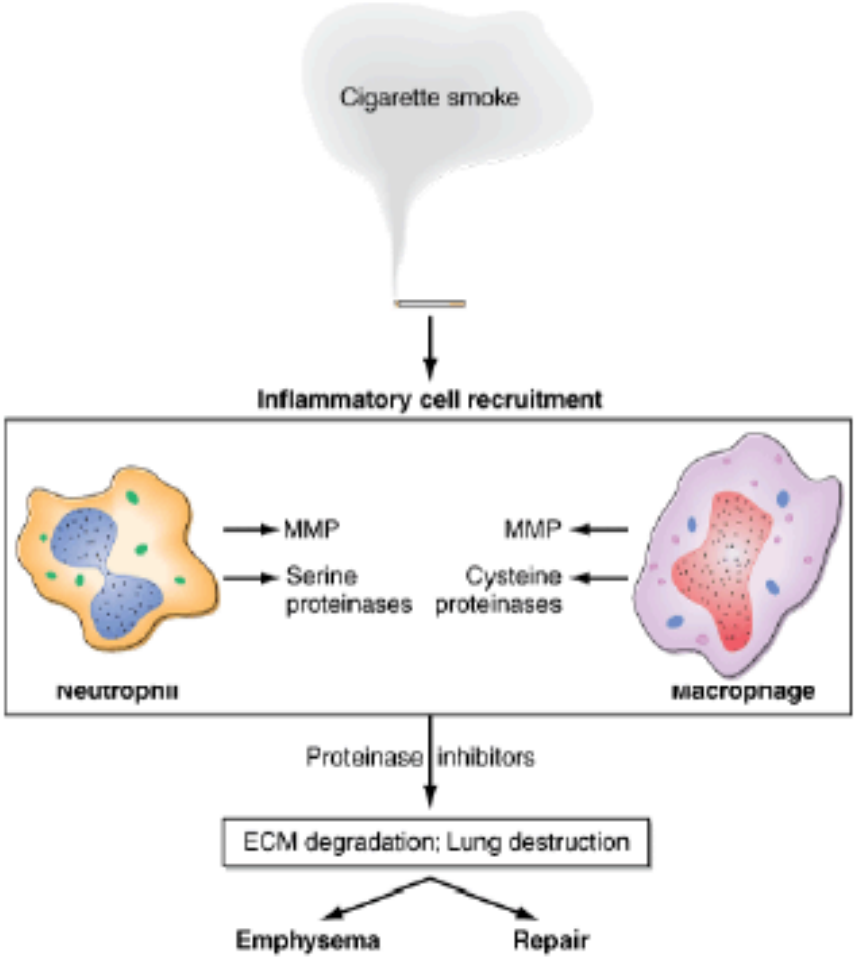
Figure 40-2 Anatomic varieties of emphysema. *A*. Centriacinar (centrilobular). *B*. Paraseptal (distal acinar). *C*. Panacinar (panlobular). *D*. Irregular (scar). The dashed lines mark the edge of the acinus. Only centriacinar and panacinar emphysema are commonly observed in COPD.

in Fishman's Pulmonary Diseases and Disorders

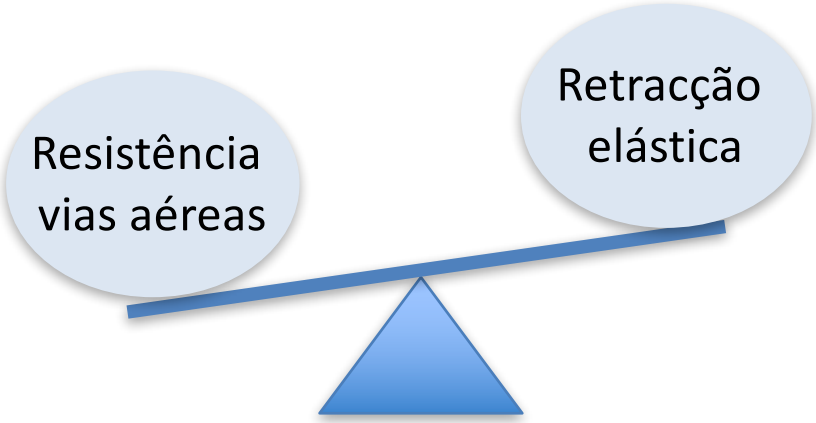

Medscape®
www.medscape.com
Normal
Centriacinar
(Centrilobular) Emphysema
Panacinar
(Panlobular) Emphysema
ACINAR STRUCTURE
LOBULAR PATTERN
TB
RB1
RB2
RB3
AD
AS
AS + AS
Septum
Source: ACP Medicine © 2004 WebMD Inc.

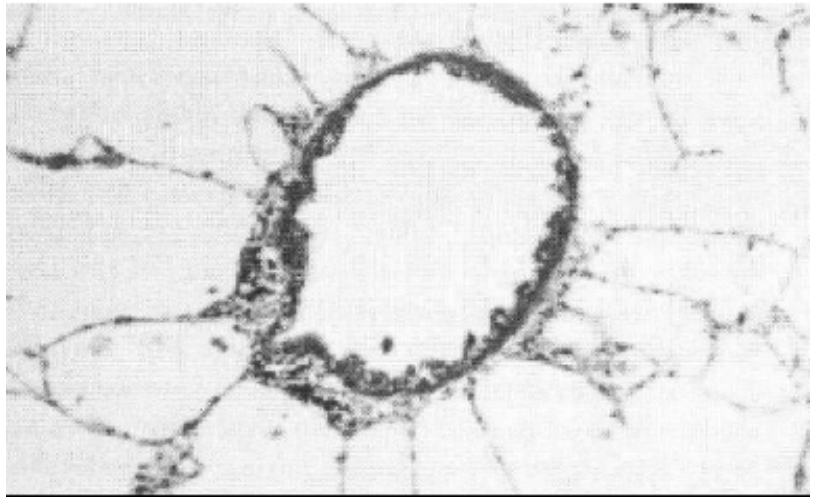
The Elastase:Antielastase Hypothesis





Obstrução ventilatória na expiração forçada





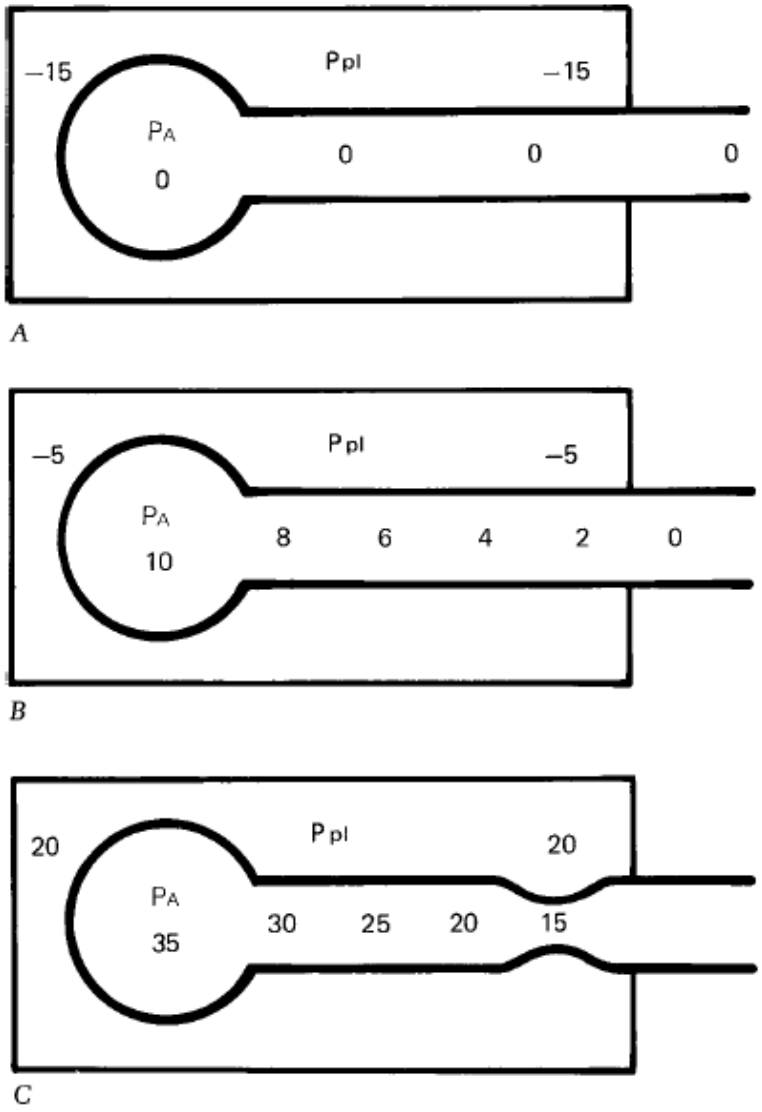
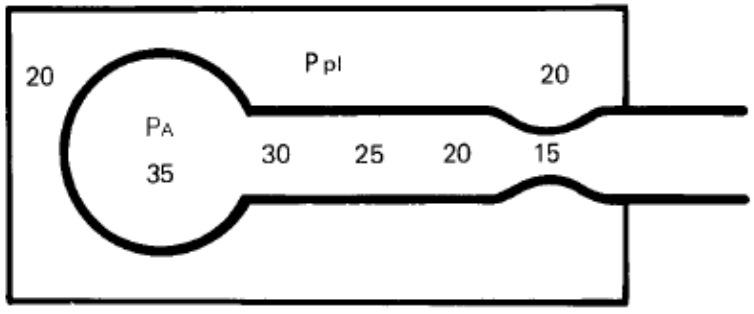


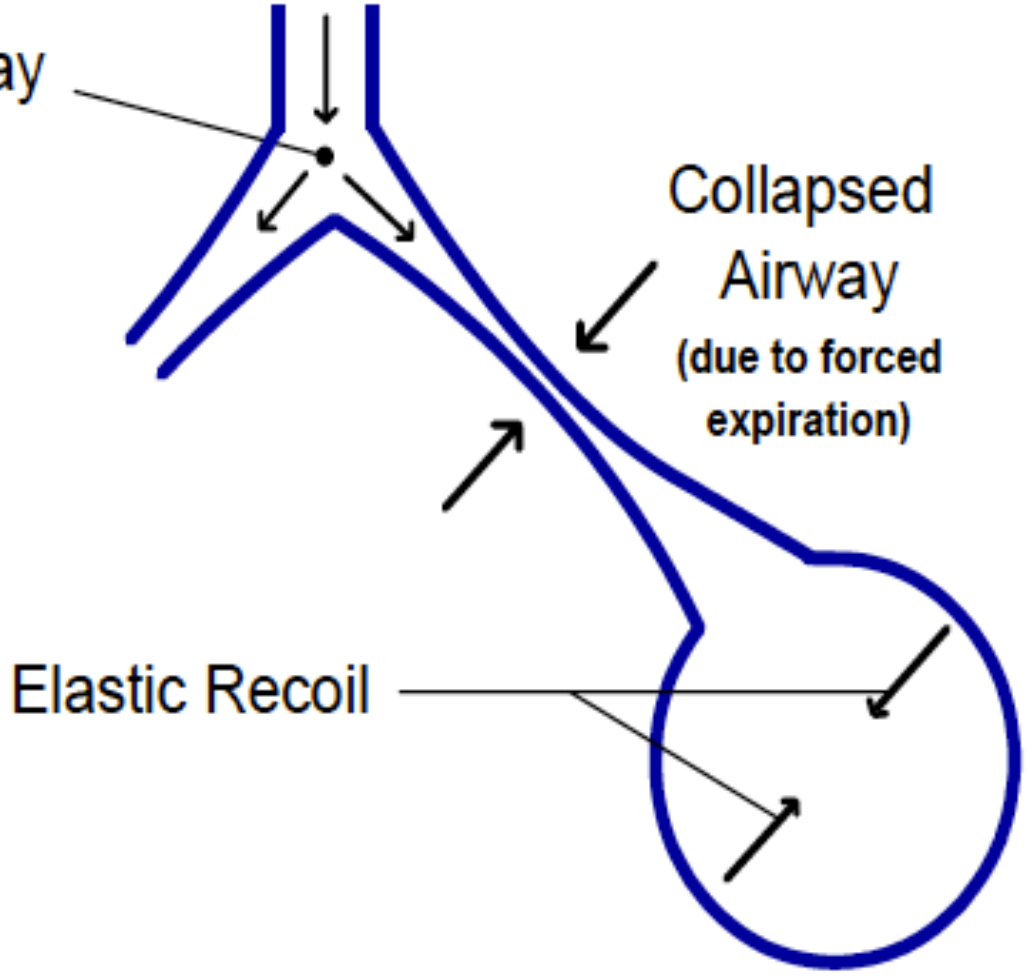
Figure 9-12 Schema of the distribution of pleural, alveolar, and airway pressures at rest and during expiration, illustrating the equal pressure point concept. *A*. End-expiration. *B*. Quiet expiration. *C*. Forced expiration.

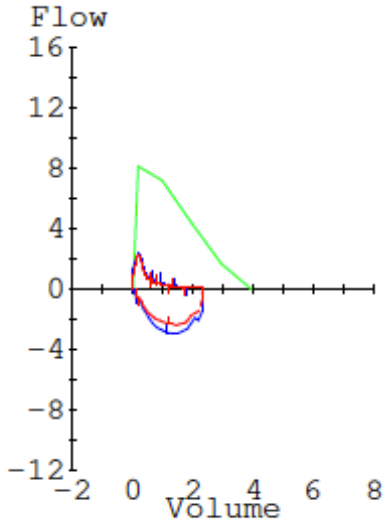
in Fishman's Pulmonary Diseases and Disorders

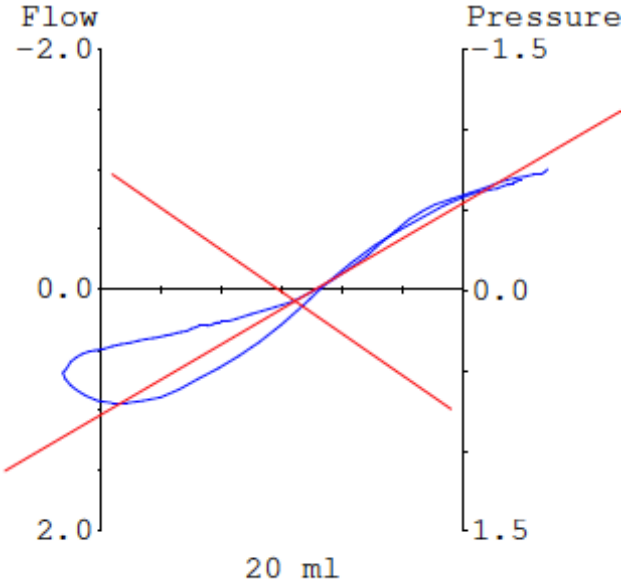
PATOLOGIA DO ENFISEMA



C





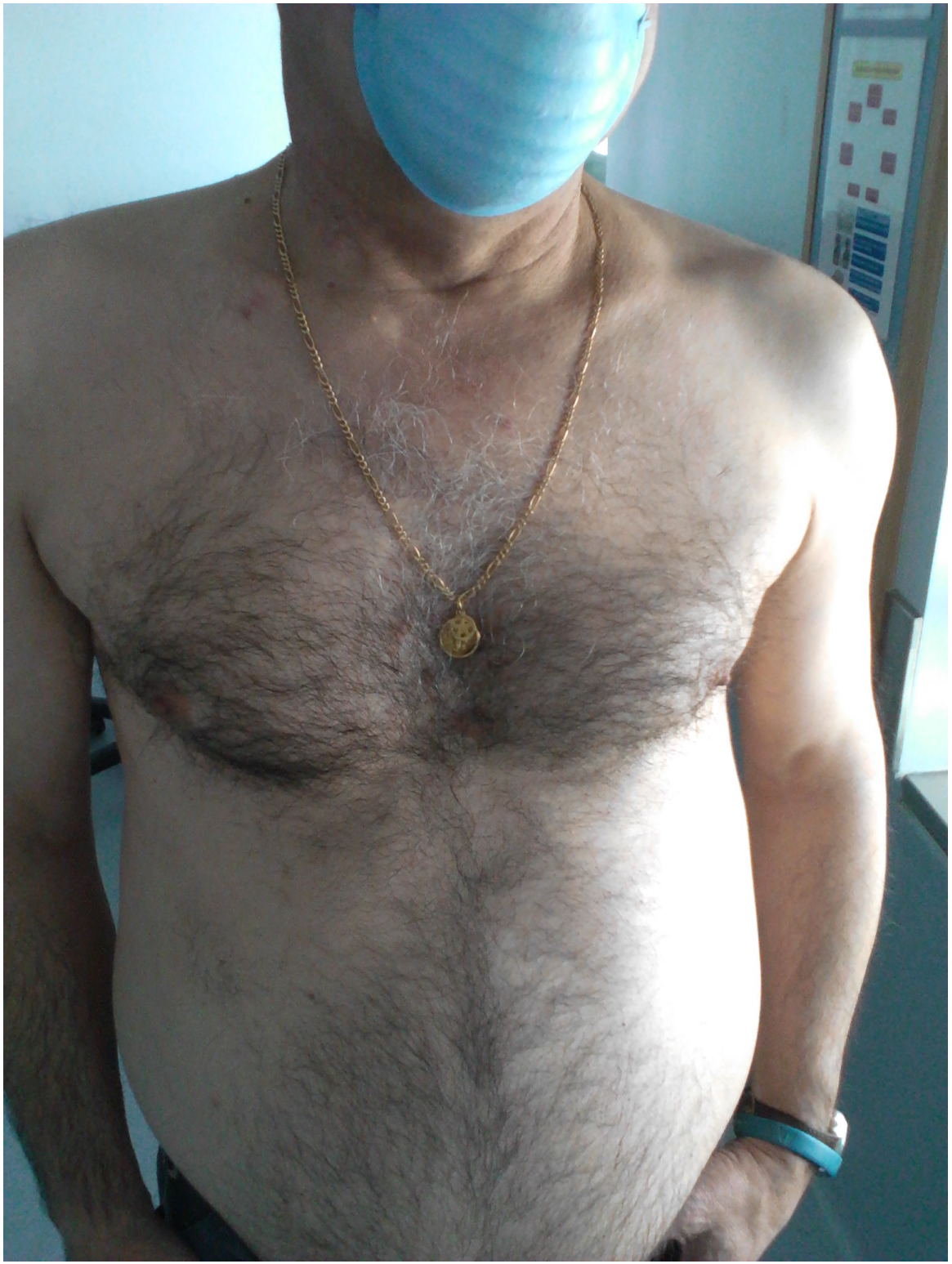


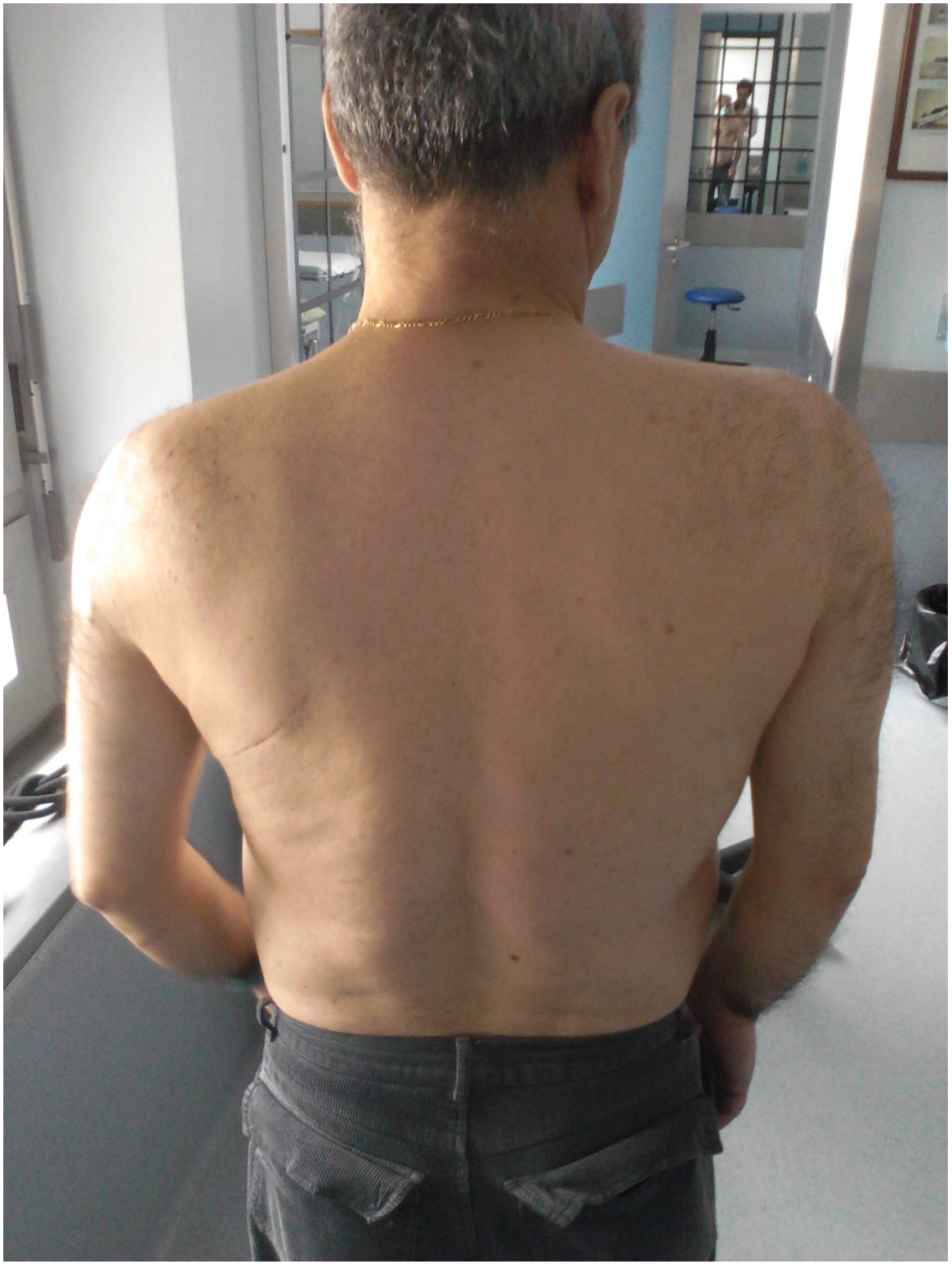
Espirometria

		Ref	Pre Meas	Pre % Ref	Post Meas	Post % Ref	Post % Chg
FVC	Liters	3.94	(2.35)	(60)	(2.34)	(59)	-0
FEV1	Liters	3.15	(0.91)	(29)	(0.87)	(28)	-4
FEV1/FVC	%	77	(39)		(37)		
FEF25-75%	L/sec	3.57	(0.23)	(6)	(0.18)	(5)	-22
FEF25%	L/sec	7.13	(0.70)	(10)	(0.65)	(9)	-8
FEF50%	L/sec	4.32	(0.29)	(7)	(0.23)	(5)	-21
FEF75%	L/sec	1.61	(0.10)	(6)	(0.11)	(7)	7
PEF	L/sec	8.12	(2.35)	(29)	(3.17)	(39)	35
FIVC	Liters	3.94	(2.34)	(59)	(2.24)	(57)	-4
FVL ECode			111010		001010		

Volumes Pulmonares

		Ref	Pre Meas	Pre % Ref	Post Meas	Post % Ref	Post % Chg
VC	Liters	4.09	(2.52)	(62)			
TLC	Liters	6.42	(9.14)	(142)			
RV	Liters	2.22	(6.62)	(299)			
FRC PL	Liters	3.37	(7.42)	(220)			
FRC N2	Liters	3.37					
RV/TLC	%	36	(72)				





HIPERINSUFLAÇÃO DINÂMICA

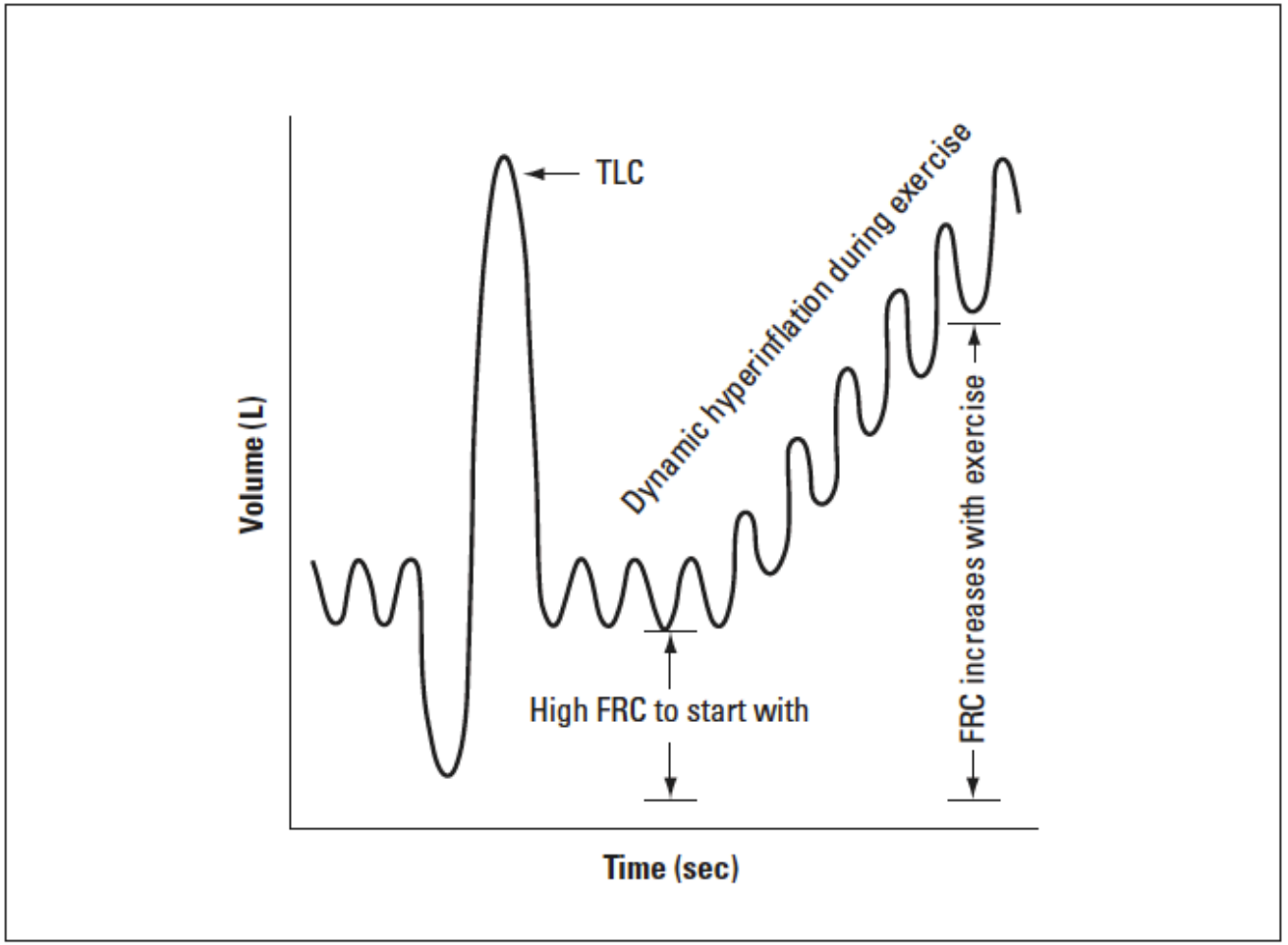


Figure 1. Dynamic hyperinflation in patients with emphysema during exercise.

FRC: functional residual capacity TLC: total lung capacity

BCMJ 2008; 50(2):97-102

HIPERINSUFLAÇÃO DINÂMICA

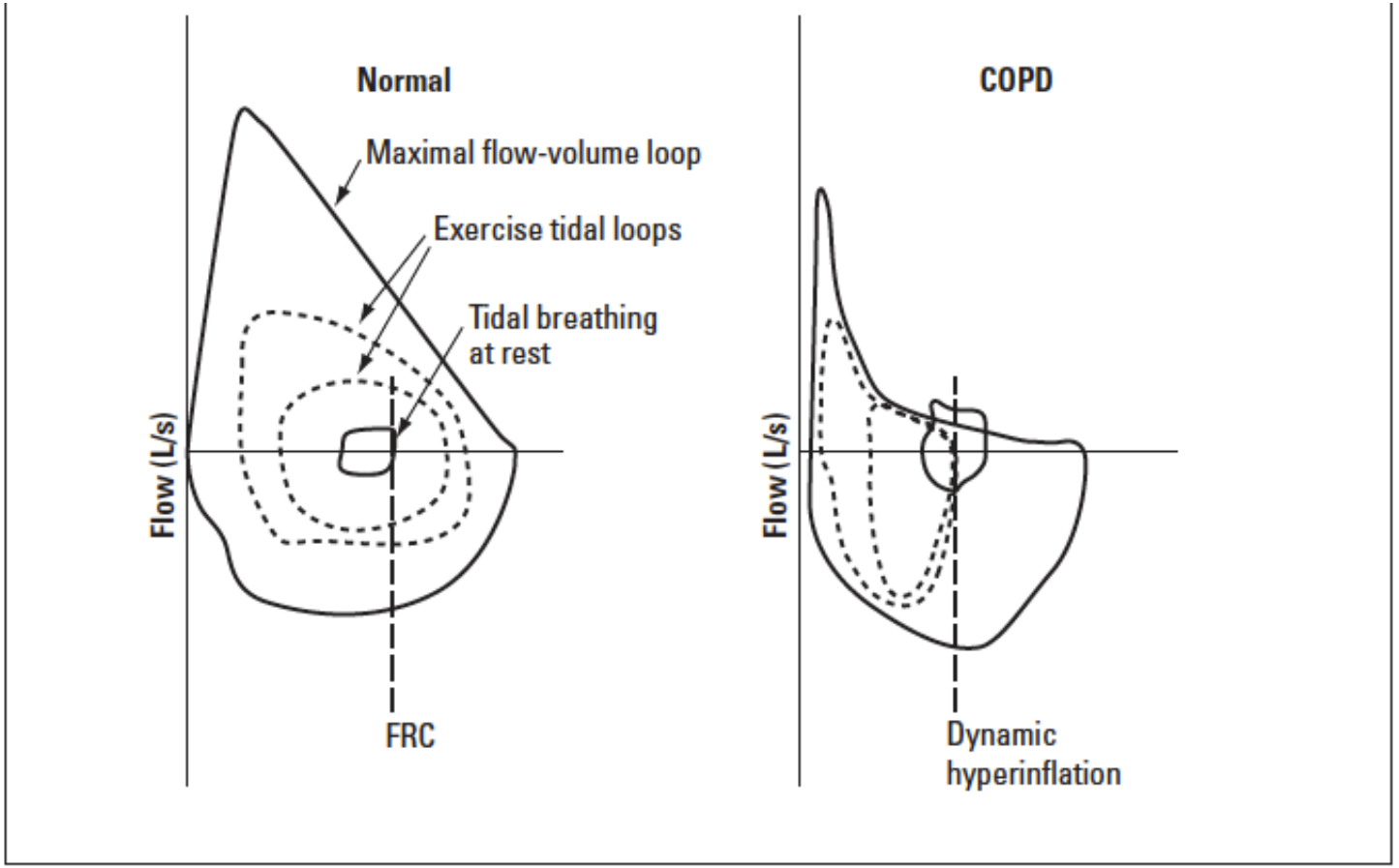
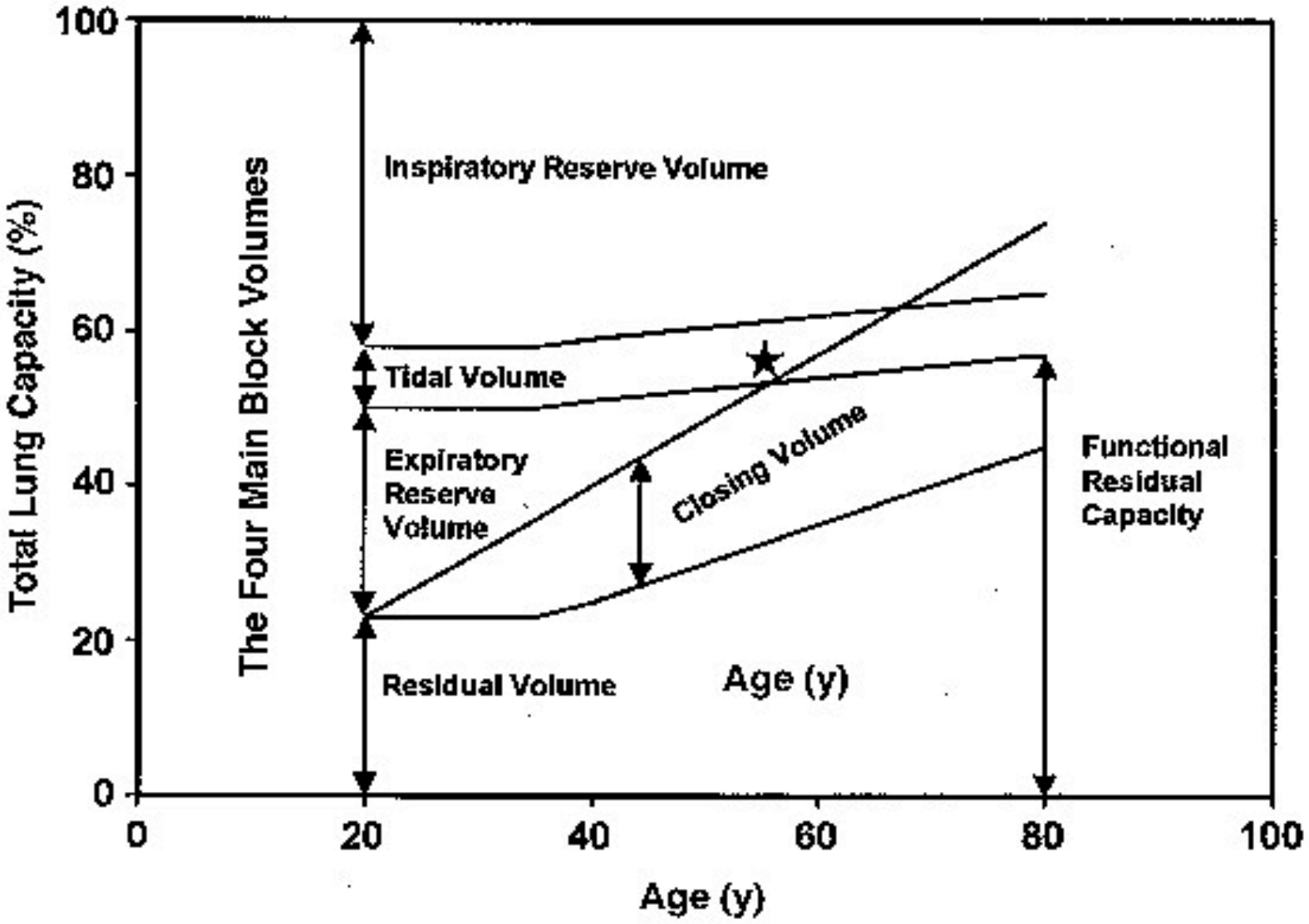
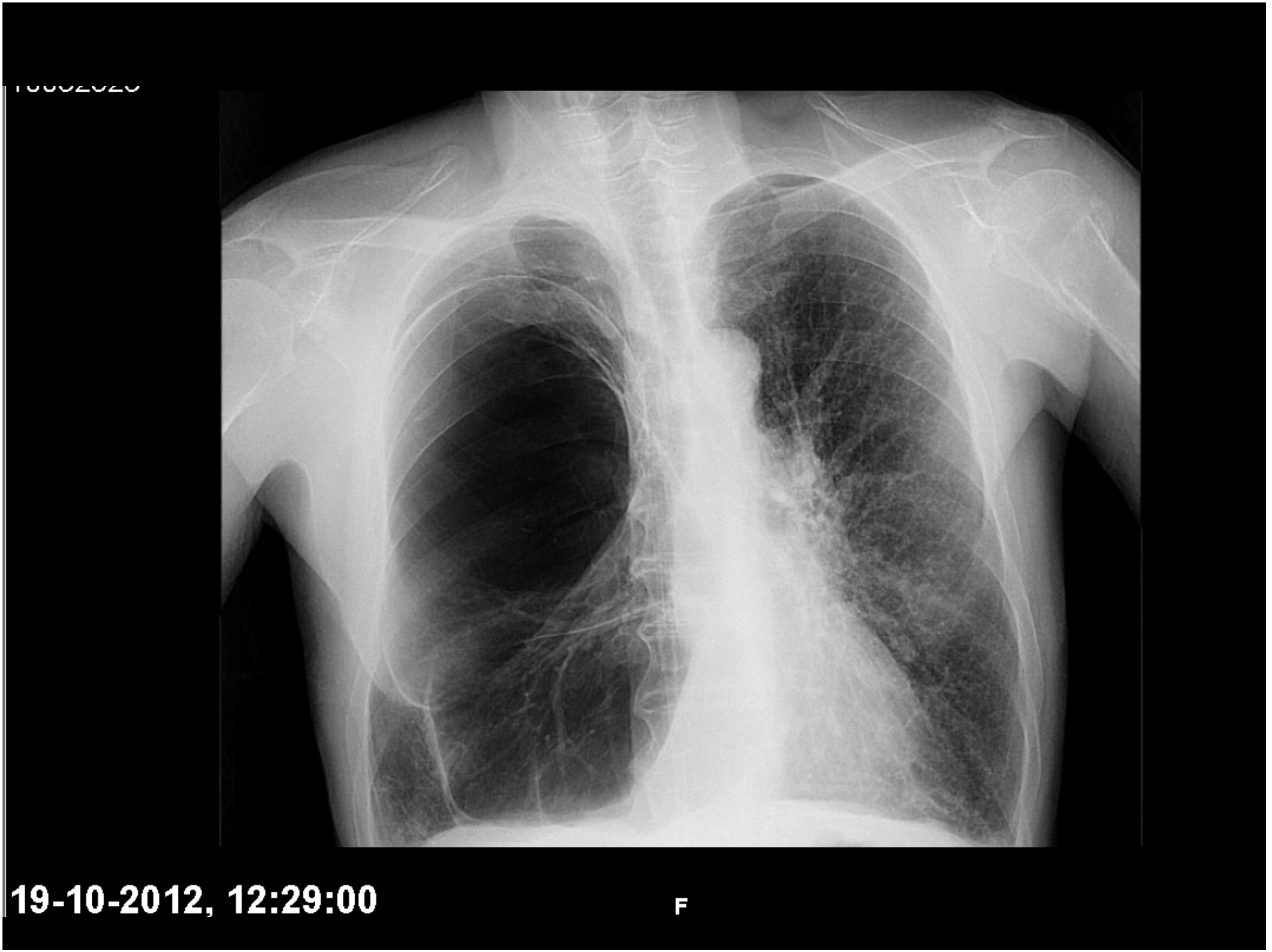


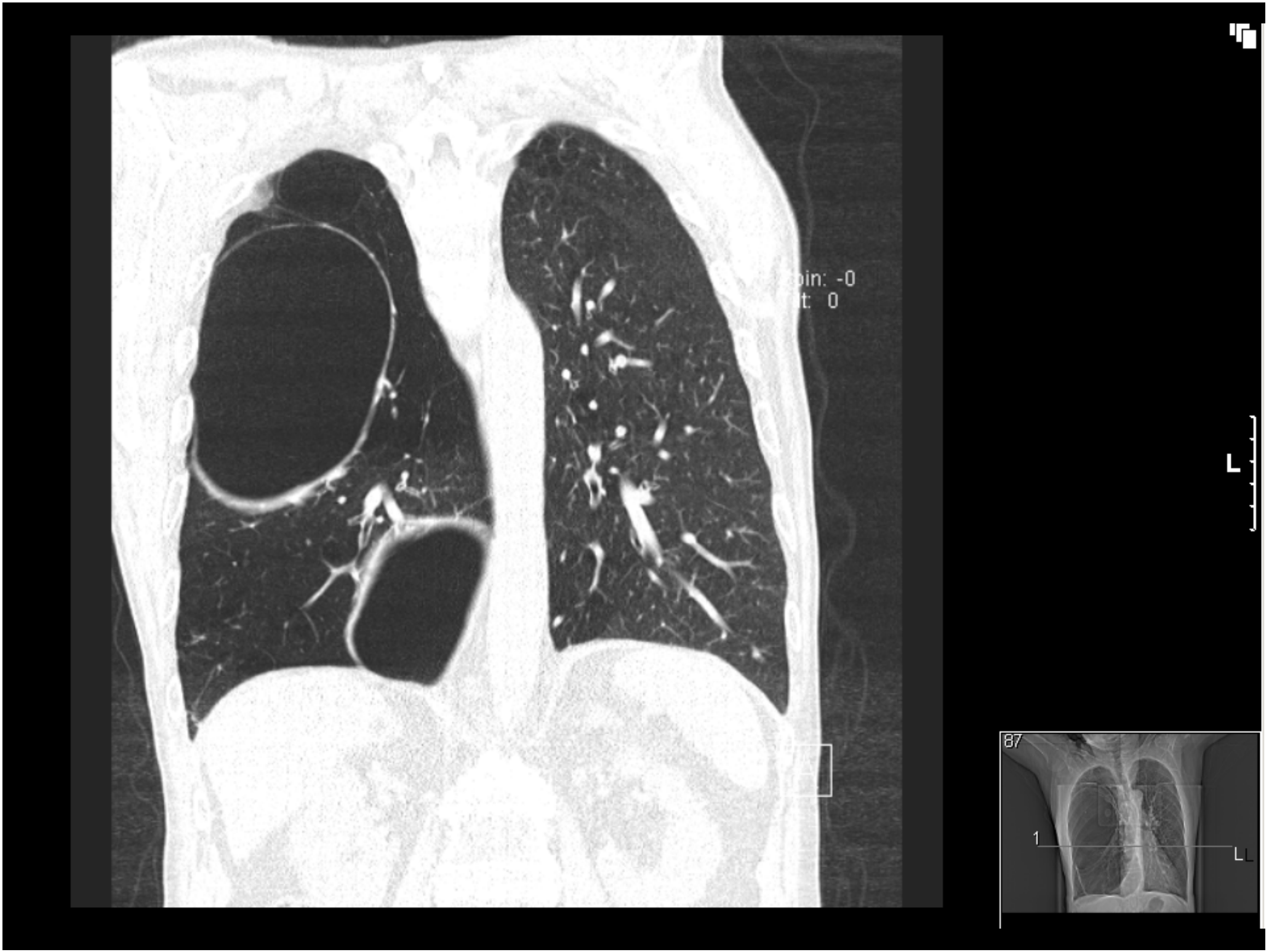
Figure 5. The relation between the exercise tidal flow-volume loops and the baseline maximal flow-volume loop. Normally (left), tidal flow-volume loops expand in both directions during exercise. In emphysema (right), the decreased expiratory time (because of increased respiratory rate during exercise) results in more air-trapping and increases the FRC, shifting the tidal flow-volume loop curves to the left, a phenomenon called dynamic hyperinflation.

FRC: functional residual capacity

BCMJ 2008; 50(2):97-102


Total Lung Capacity (%)
100
80
60
40
20
0
The Four Main Block Volumes
Inspiratory Reserve Volume
Tidal Volume
Expiratory Reserve Volume
Residual Volume
Closing Volume
Functional Residual Capacity
Age (y)
0
20
40
60
80
100
Age (y)


19-10-2012, 12:29:00
F


in: -0
t: 0
L
87
1
LL

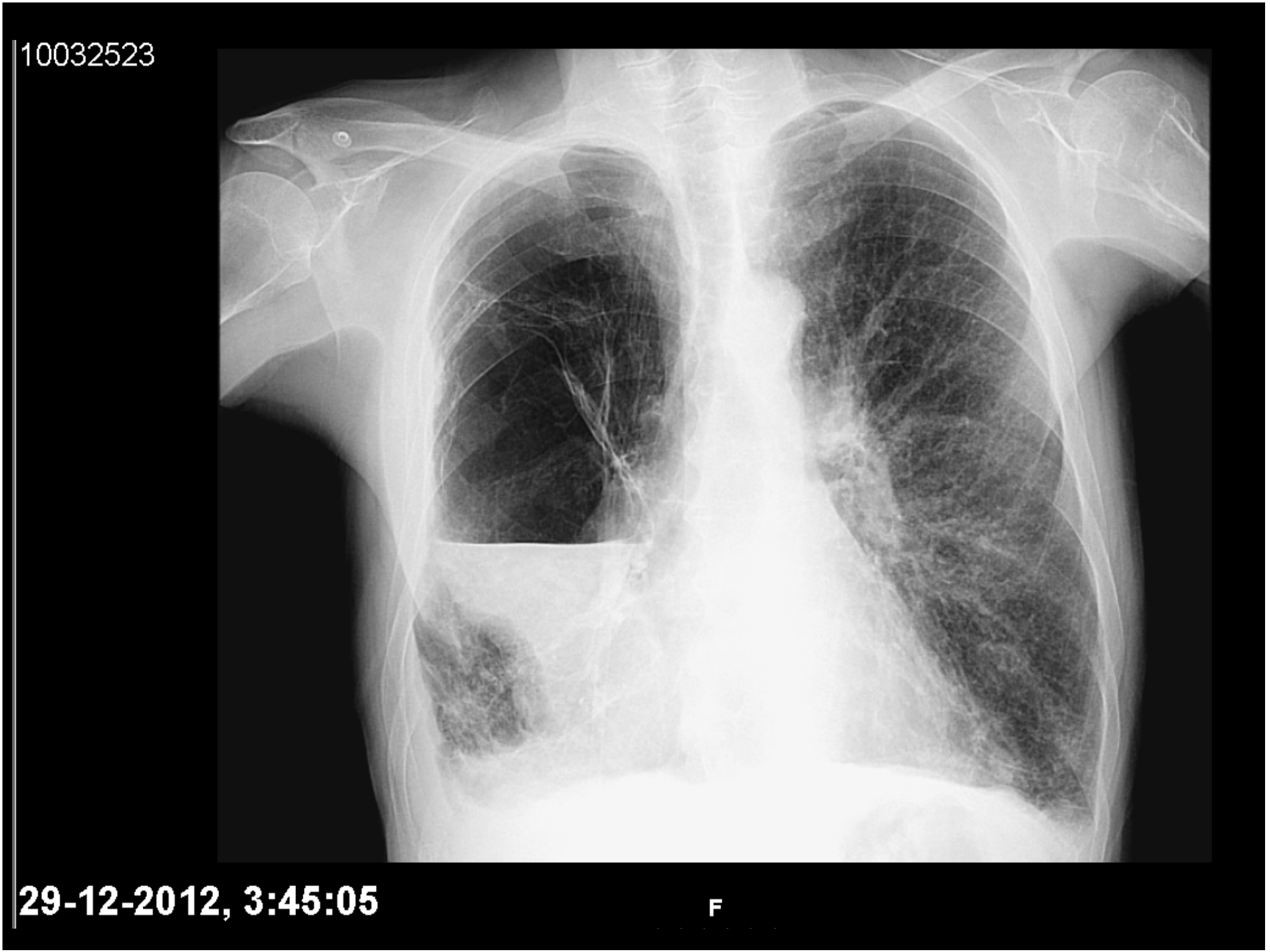
26:48

F

F

J7


Spin: 90
Tilt: 0
A
R
87
1
LL
F
J2
:27:50
F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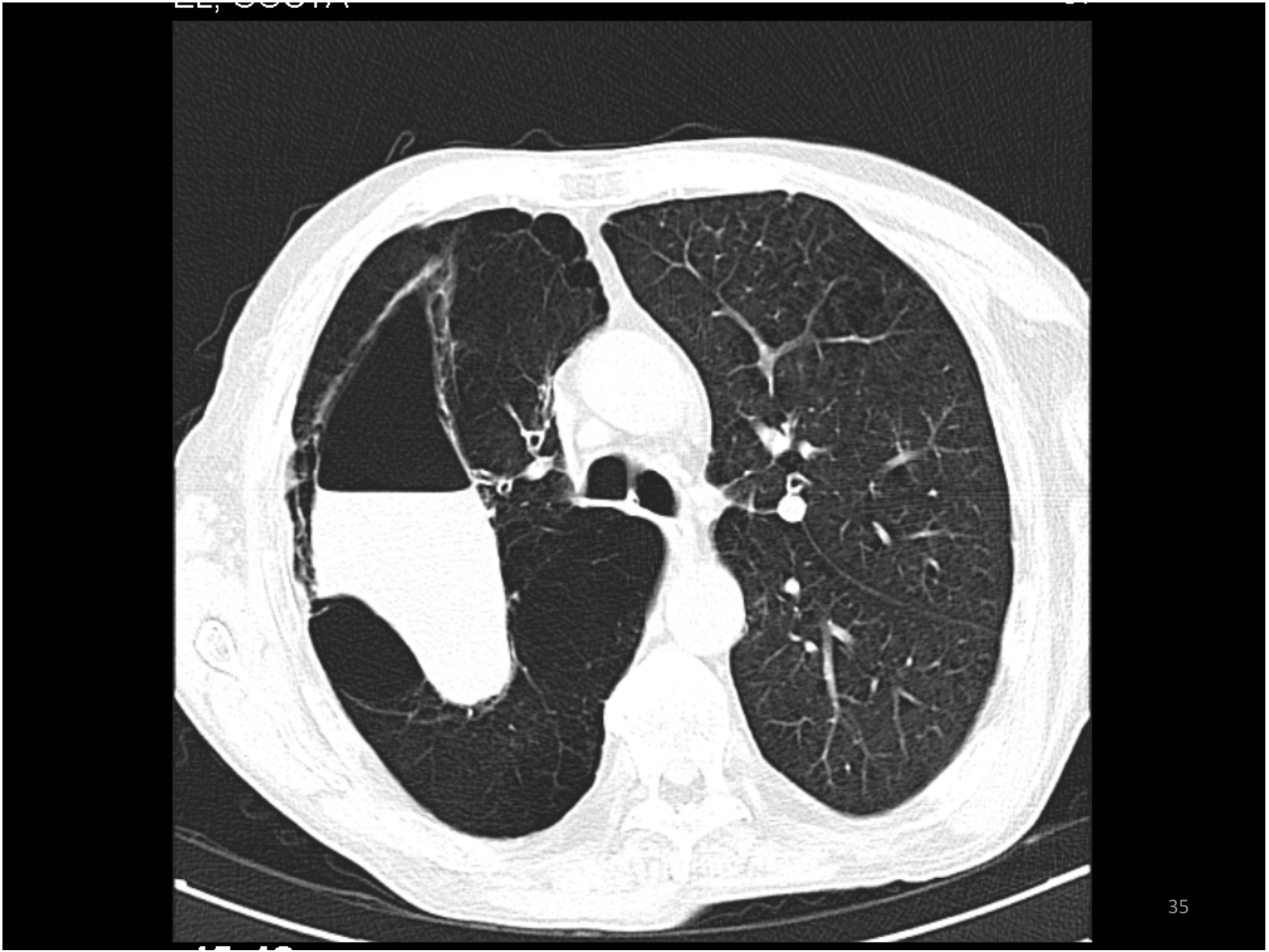
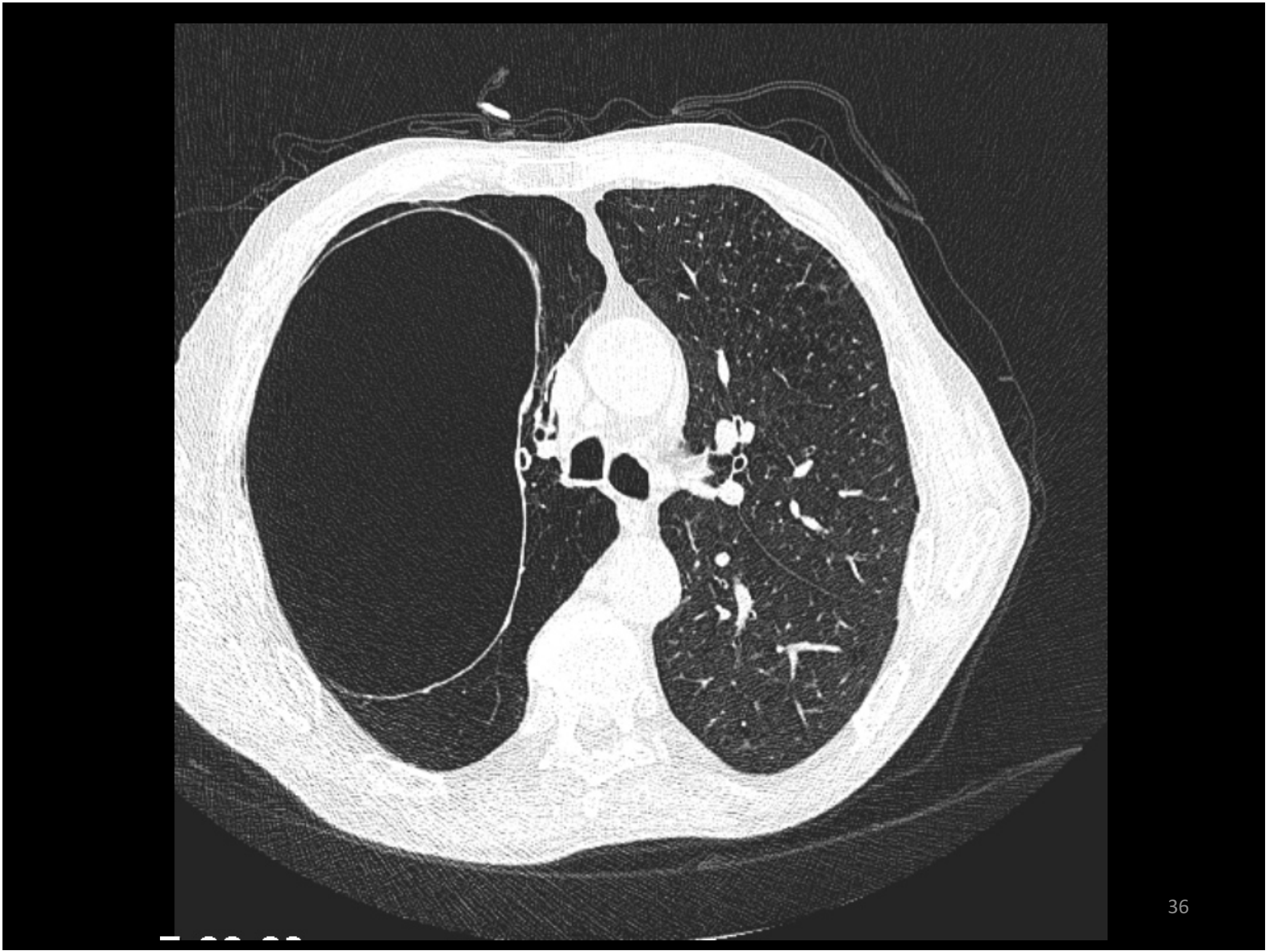
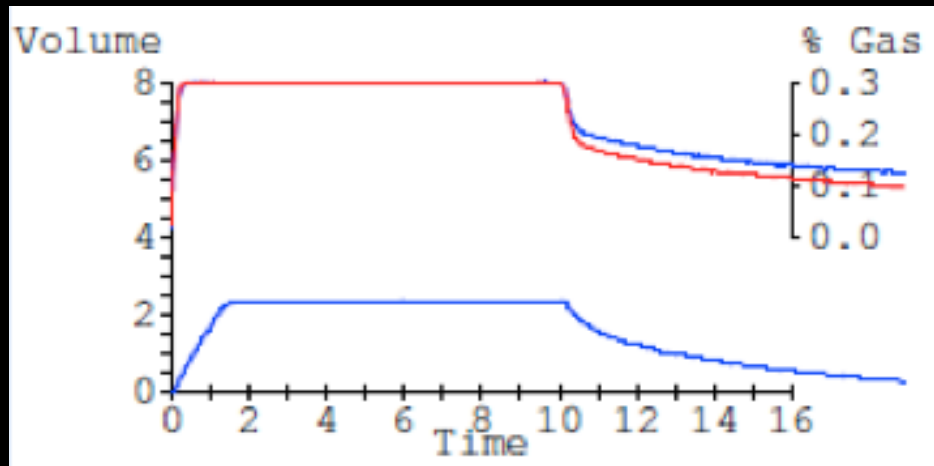
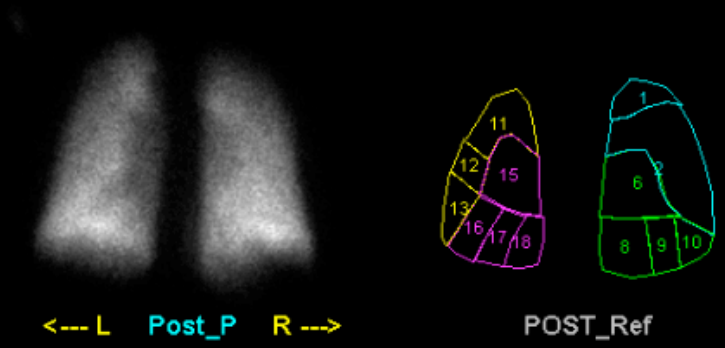






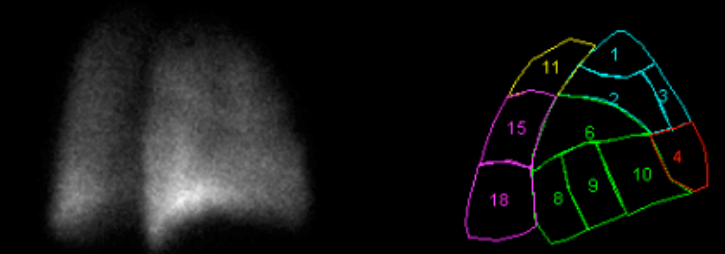
Difusão

		Ref	Pre Meas	Pre % Ref
DLCO	mmol/kPa.min	7.6	(1.6)	(21)
VA	Liters	6.10	(4.20)	(69)
DLCO/VA	DLCO/L	1.25	0.39	31
TLC Sb	Liters	6.10	(4.20)	(69)
RV Sb	Liters	2.49	(1.78)	(72)
RV/TLC Sb	%	42	42	



<--- L Post_P R --->

POST_Ref



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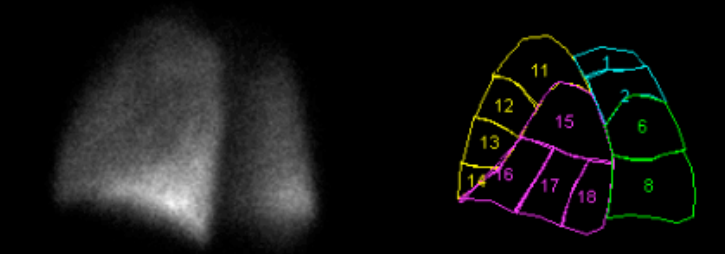
RPO_Ref



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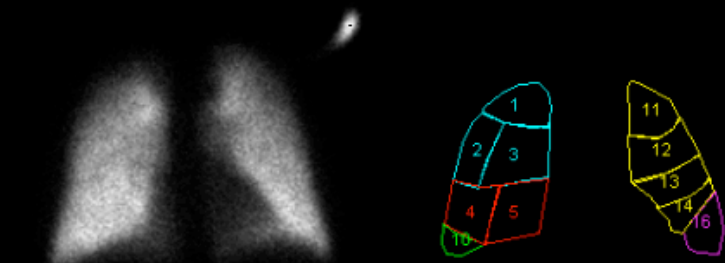
LPO_Ref



<--- R Ant_P L --->

ANT_Ref

Click Segment to Highlight

Right Upper Lobe

1. Apical
2. Posterior
3. Anterior

Right Medial Lobe

4. Lateral
5. Medial

Right Lower Lobe

6. Superior
7. Medial Basal
8. Posterior Basal
9. Lateral Basal
10. Anterior Basal

Left Upper Lobe

11. Apical Posterior
12. Anterior
13. Superior Lingual
14. Inferior Lingual

Left Lower Lobe

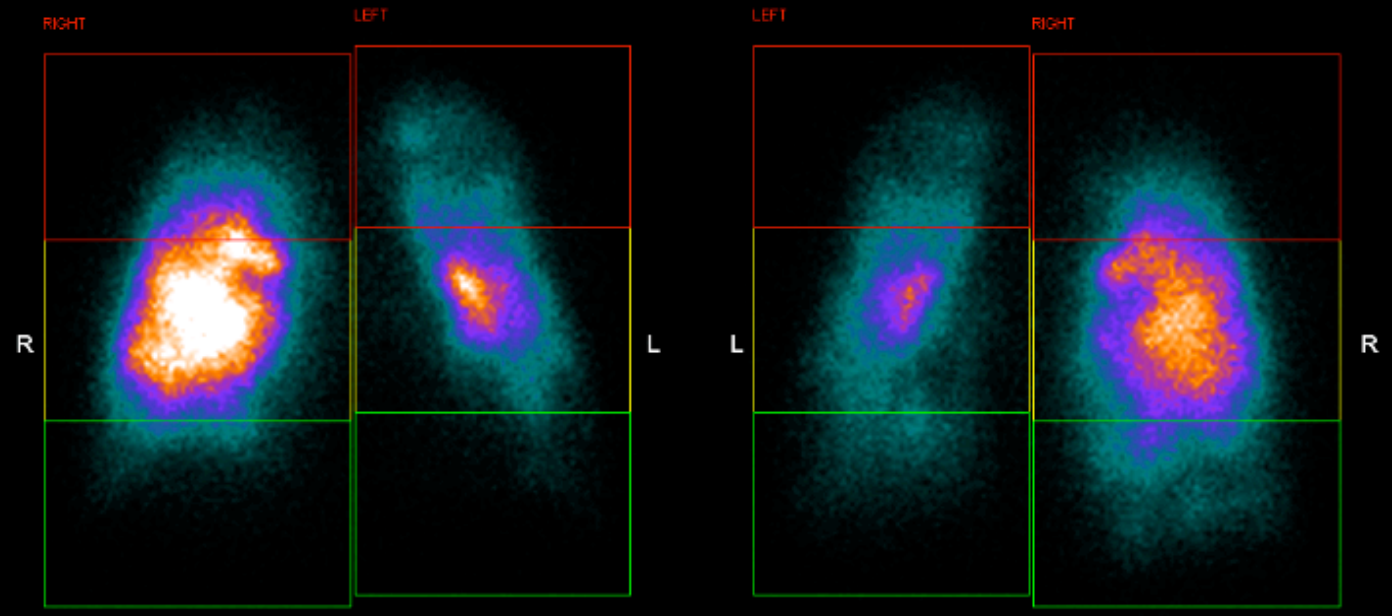
15. Superior
16. Anteromedial Basal
17. Lateral Basal
18. Posterior Basal

10843097

1) Lung_Quantif
LUNGS QUANTIFICATION

13-07-2012 11:40

Hospital S. Joao
Nuclear Imaging Environment



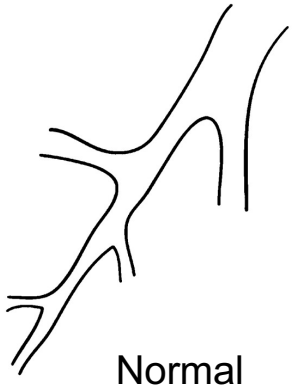
ANTERIOR PERFUSION

POSTERIOR PERFUSION

GEO MEAN	LEFT	RIGHT
SUPERIOR	8 %	12 %
MIDDLE	20 %	48 %
INFERIOR	4 %	8 %
TOTAL	32 %	68 %

Bronquiectasias

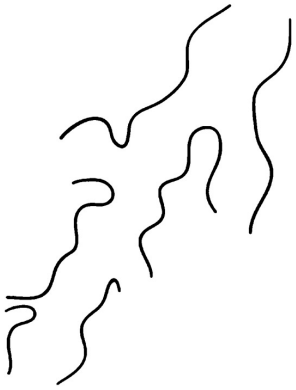
Dilatação e destruição irreversível das vias aéreas, associada a um ciclo vicioso de inflamação, infecção recorrente e lesão brônquica.



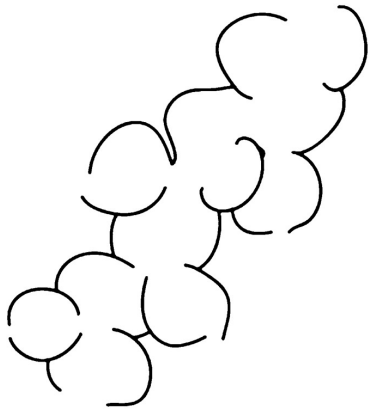
Normal

Cilíndrica

1. Diâmetro interno do brônquio é maior que a artéria pulmonar adjacente
2. O brônquio não consegue afilar
3. Visualiza-se lúmen brônquico 1-2cm da periferia



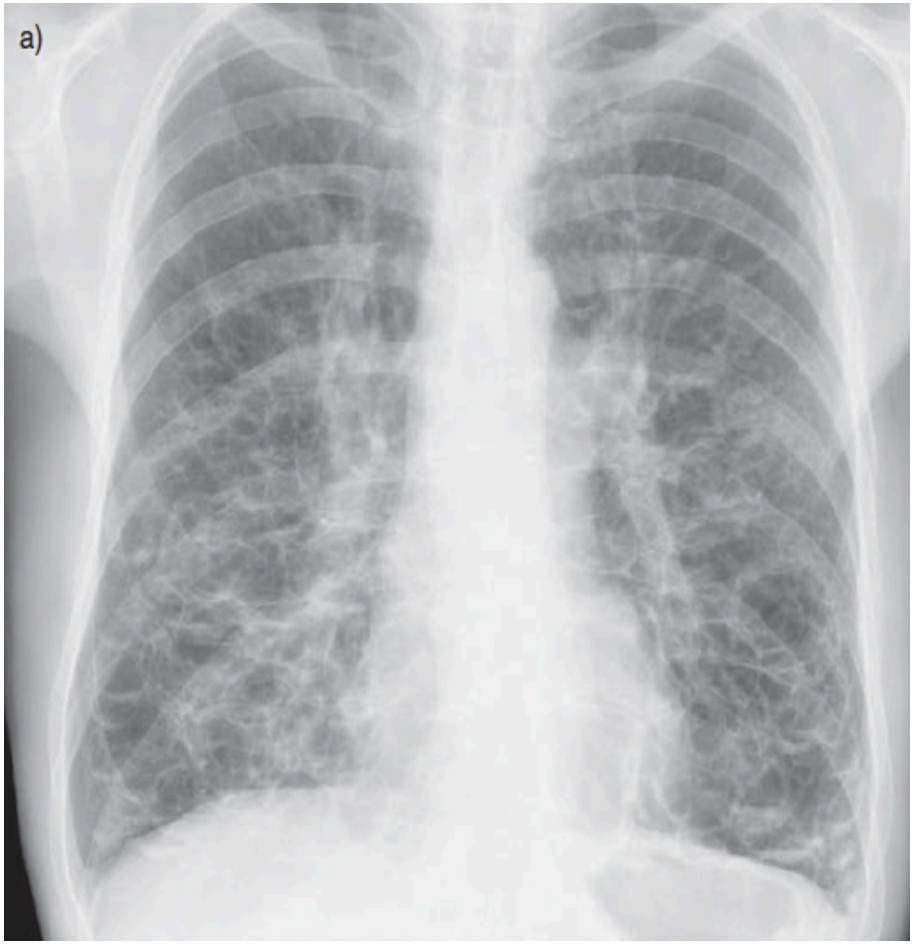
Varicosa



Cística

Tosse , broncorreia, alteração ventilatória obstrutiva progressiva

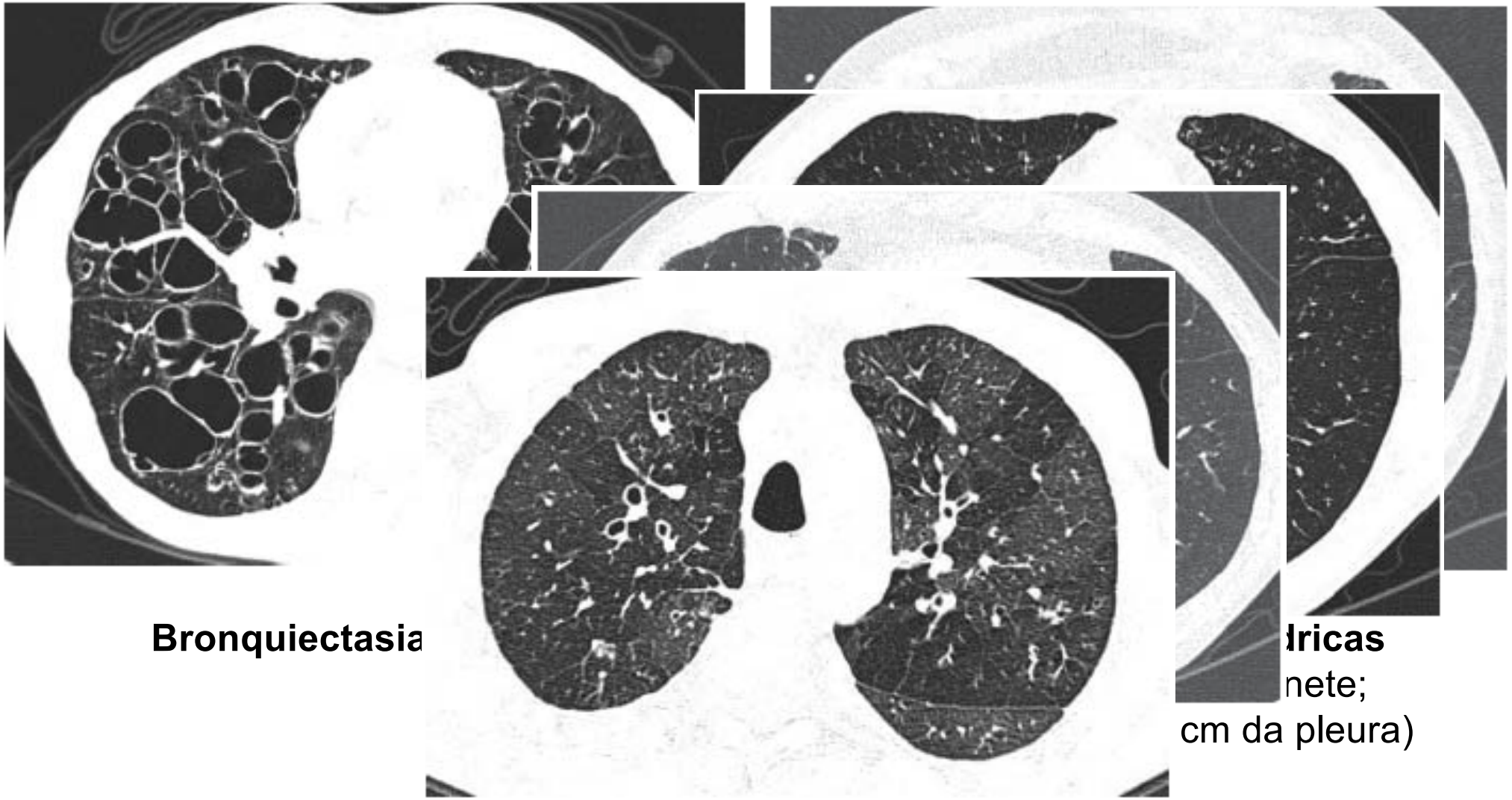
Eur Respir Mon 2011. 52, 44–67



Bronquiectasias císticas

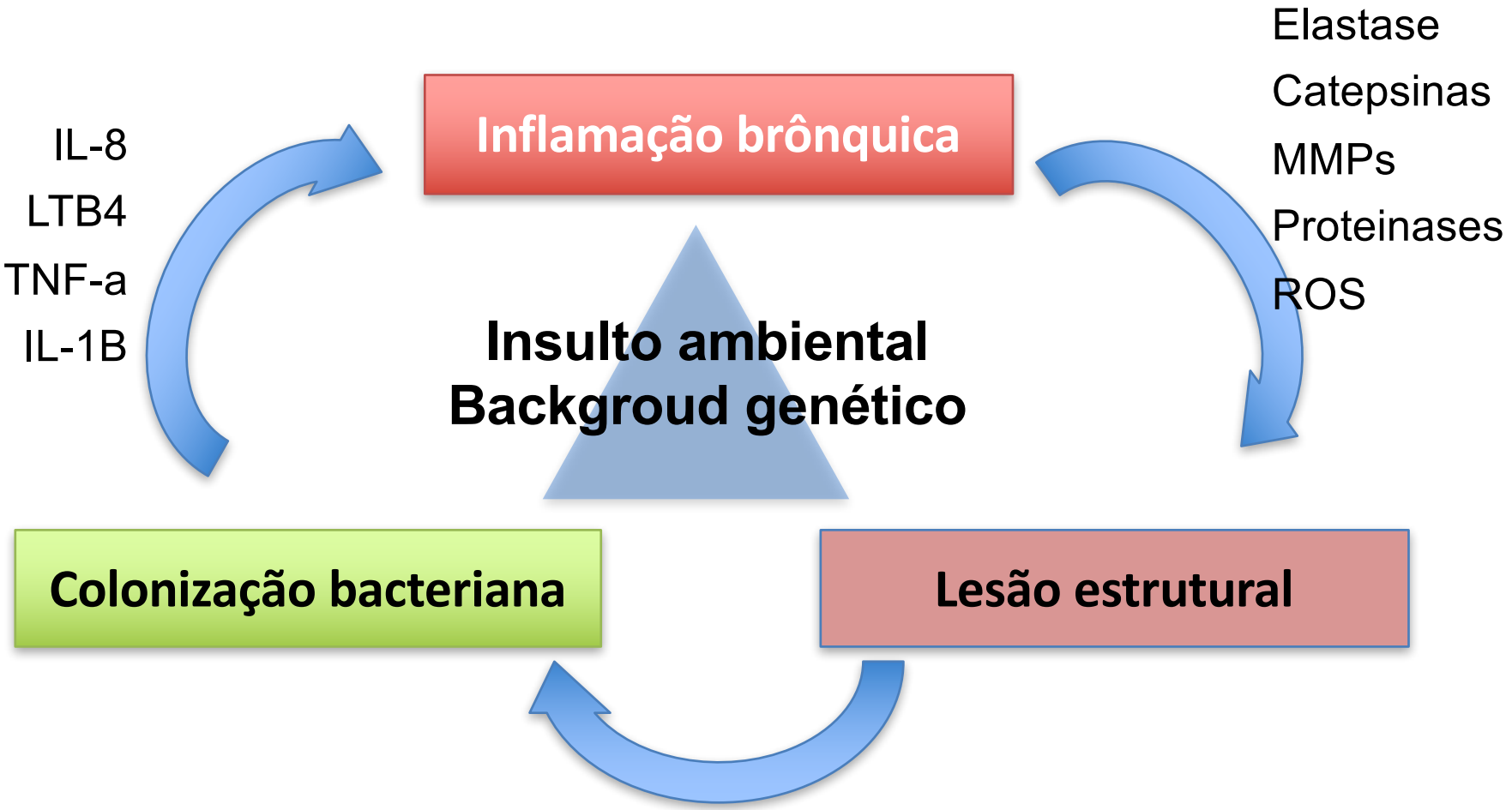
Bronquiectasias cilíndricas
(imagem de carris)

Eur Respir Mon 2011. 52, 44–67



Bronquiectasia **Idricas**
nete;
cm da pleura)

Bronquiectasias
Hipótese do "ciclo vicioso"



Modelo de Whitwell para as **Bronquiectasias foliculares (cilíndricas)**

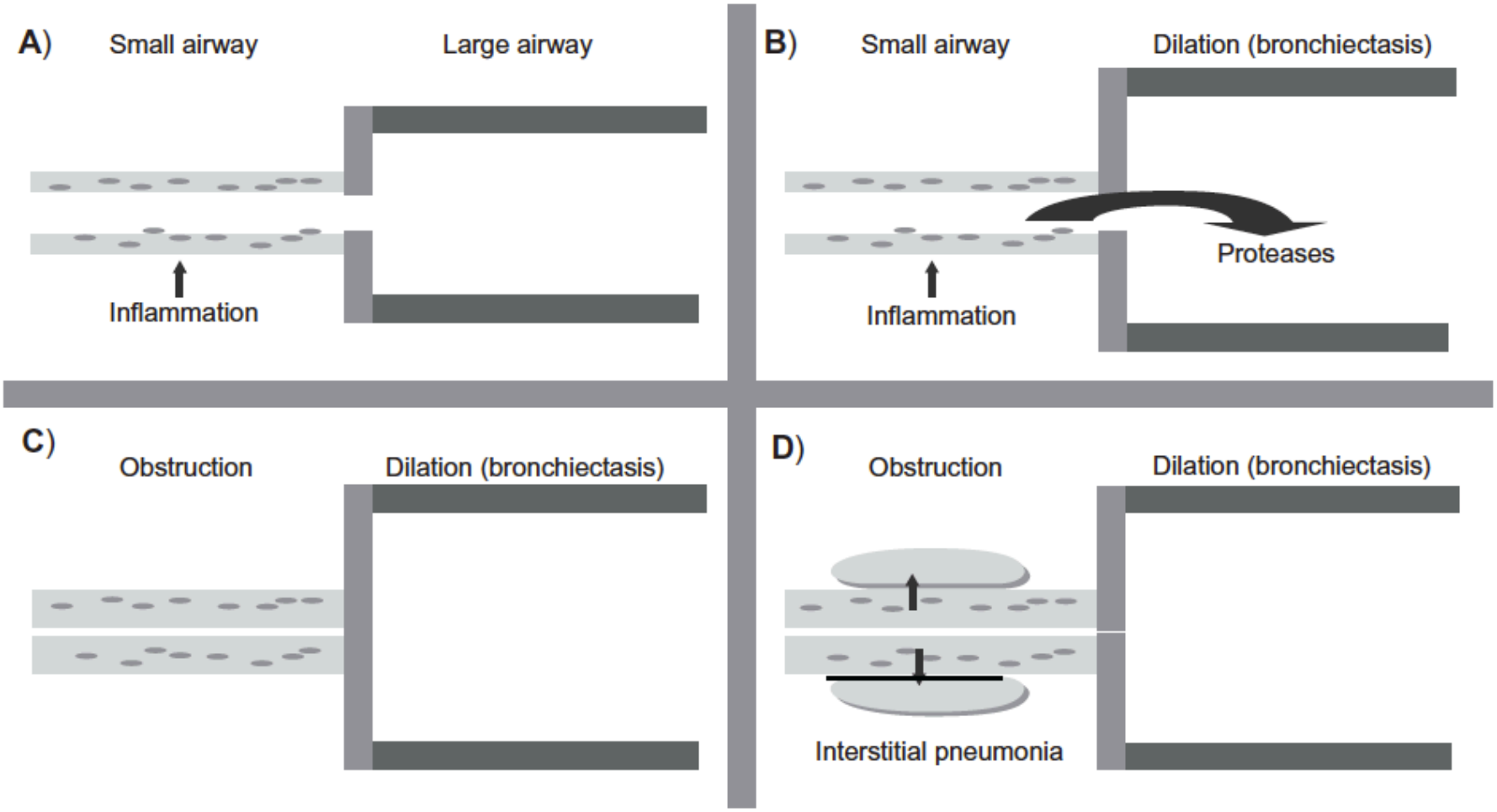


Figure 2 Pathologic changes in follicular bronchiectasis as described by Whitwell. **A)** The first process involves infection of the small airways. **B)** This leads to the release of inflammatory mediators such as proteases which damage the large airways resulting in bronchial dilation and bronchiectasis. **C)** Infection drives progressive inflammation in the small airways which become thicker from a combination of cell-mediated inflammatory infiltrate and lymphoid follicles resulting in obstruction. **D)** The final process involves the spread of inflammation beyond the airways resulting in interstitial pneumonia.

International Journal of COPD 2009;4:411–419

Obstrução ventilatória nas BQ

Obstrução ventilatória nas BQ

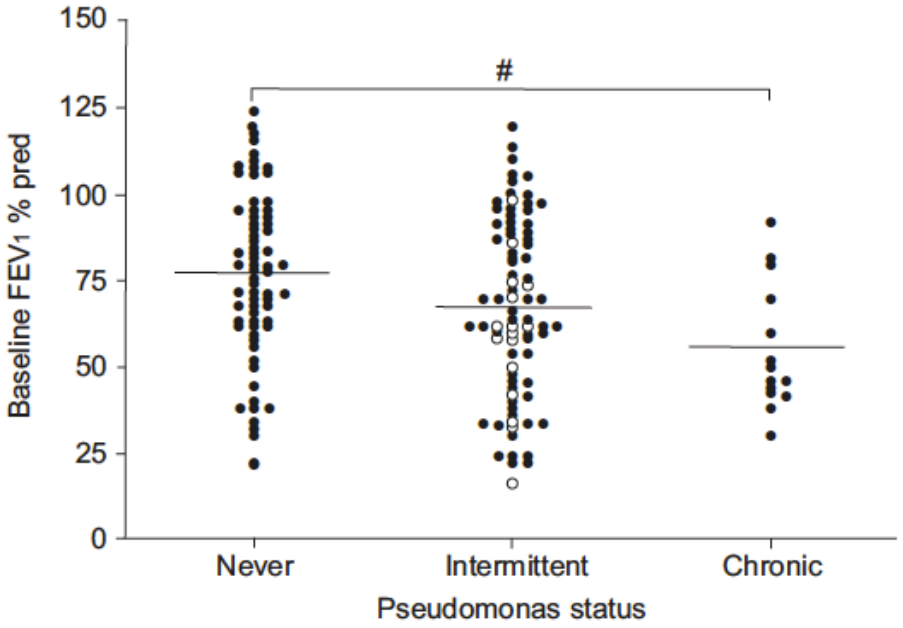


FIGURE 1. Comparison of baseline forced expiratory volume in one second (FEV_1) with pseudomonas status. ○: developed chronic *Pseudomonas aeruginosa* infection. % pred: % predicted. #: p<0.005.

Eur Respir J 2006; 28: 974–979

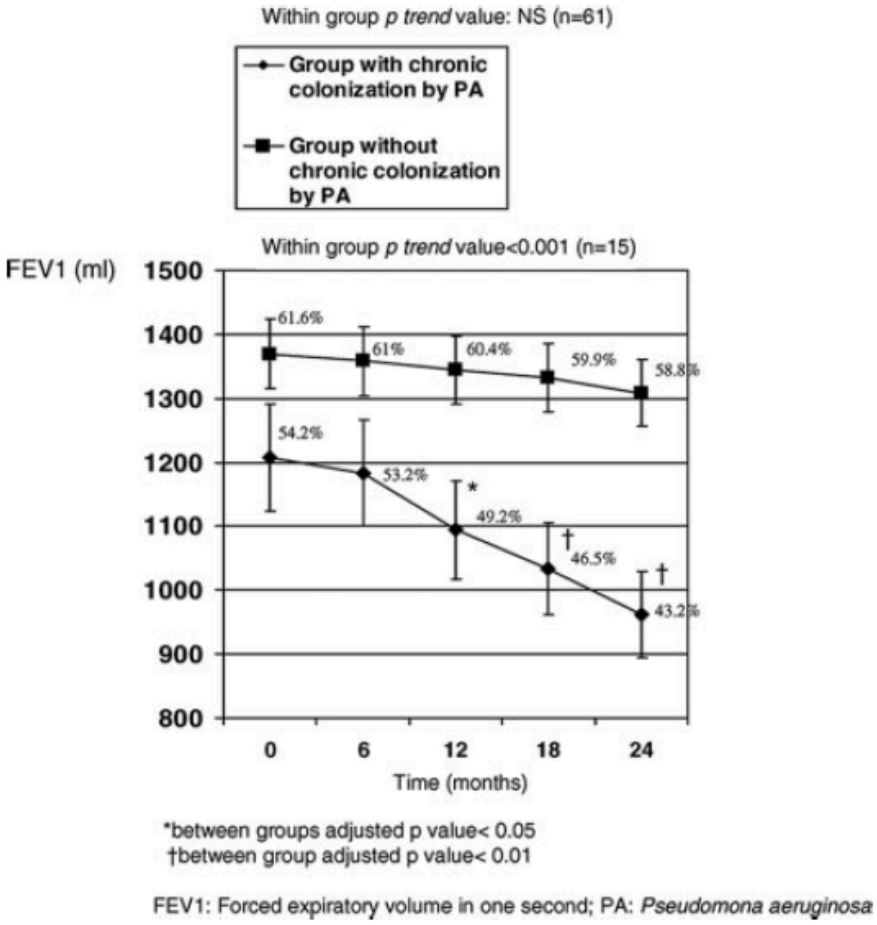


FIGURE 2. Mean (SD) FEV_1 according to PA colonization of the sputum adjusted by age, gender, time/number of measurements, and baseline FEV_1 value. The values in parentheses are percent predicted FEV_1.

CHEST 2007; 132:1565–1572

Etiologia das BQ

- Infecção severa na infância ou necrotizante
- Adenopatias obstruindo BLM

- Défice depuração muco-ciliar

- Risco de infecções

- Obstrutiva

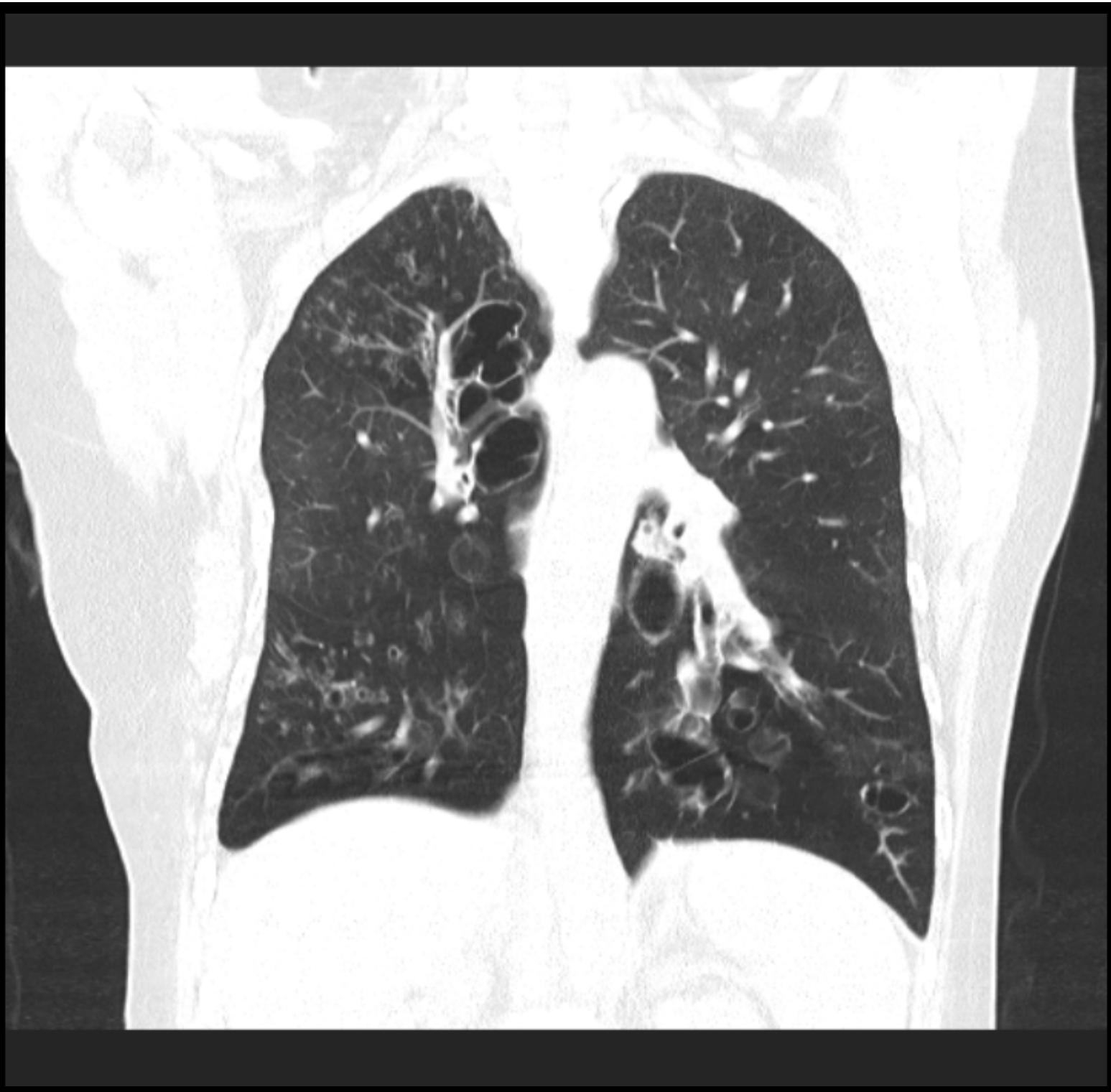
- Prevalência de BQ até 30%

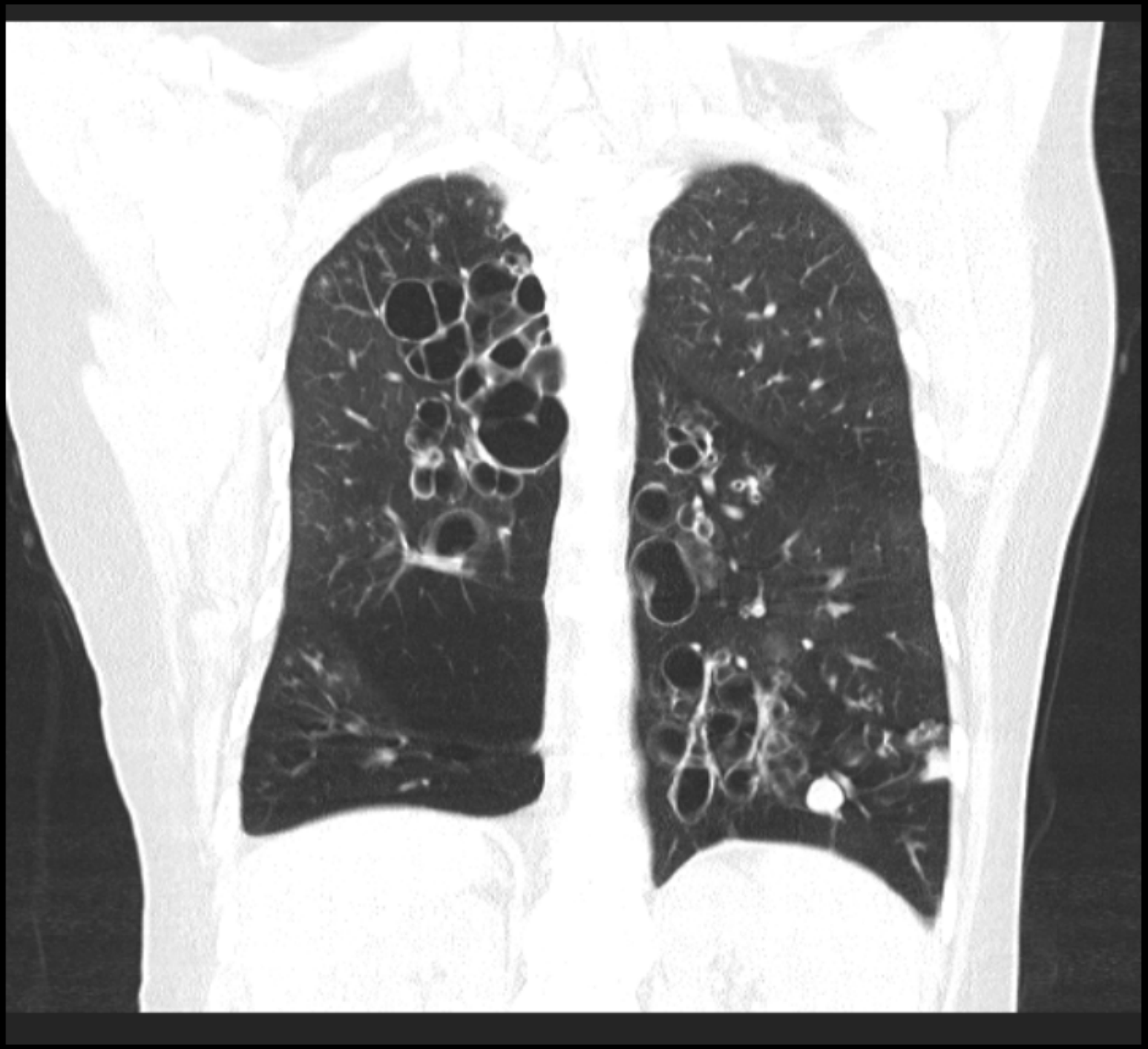
- Actividade auto-imune brônquica + complicações da terapia imunomoduladora

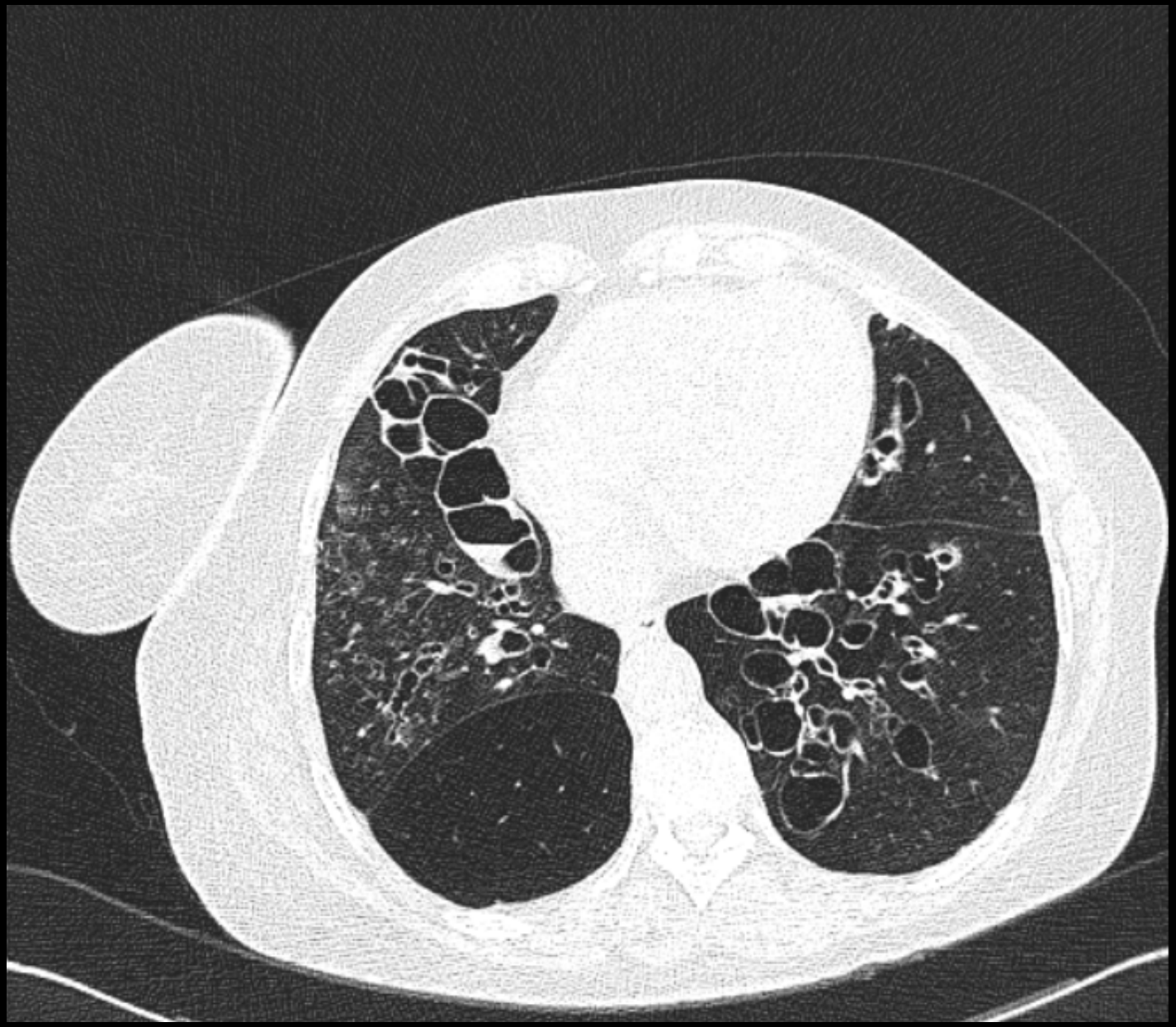
N Engl J Med 2002;346(18):1383-1393

TABLE 1. CONDITIONS ASSOCIATED WITH BRONCHIECTASIS.

- Postinfectious conditions
  - Bacteria (pseudomonas, haemophilus)
  - Mycobacterium tuberculosis
  - Aspergillus species
  - Virus (adenovirus, measles virus, influenzavirus, human immunodeficiency virus)
- Congenital conditions
  - Primary ciliary dyskinesia
  - $Alpha_1$-antitrypsin deficiency
  - Cystic fibrosis
  - Tracheobronchomegaly (Mounier–Kuhn syndrome)
  - Cartilage deficiency (Williams–Campbell syndrome)
  - Pulmonary sequestration
  - Marfan's syndrome
- Immunodeficiency
  - Primary
    - Hypogammaglobulinemia
  - Secondary
    - Caused by cancer (chronic lymphatic leukemia), chemotherapy, or immune modulation (after transplantation)
- Sequelae of toxic inhalation or aspiration
  - Chlorine
  - Overdose (heroin)
  - Foreign body
- Rheumatic conditions
  - Rheumatoid arthritis
  - Systemic lupus erythematosus
  - Sjögren's syndrome
  - Relapsing polychondritis
- Other
  - Inflammatory bowel disease (chronic ulcerative colitis or Crohn's disease)
  - Young's syndrome (secondary ciliary dyskinesia)
  - Yellow nail syndrome (yellow nails and lymphedema)







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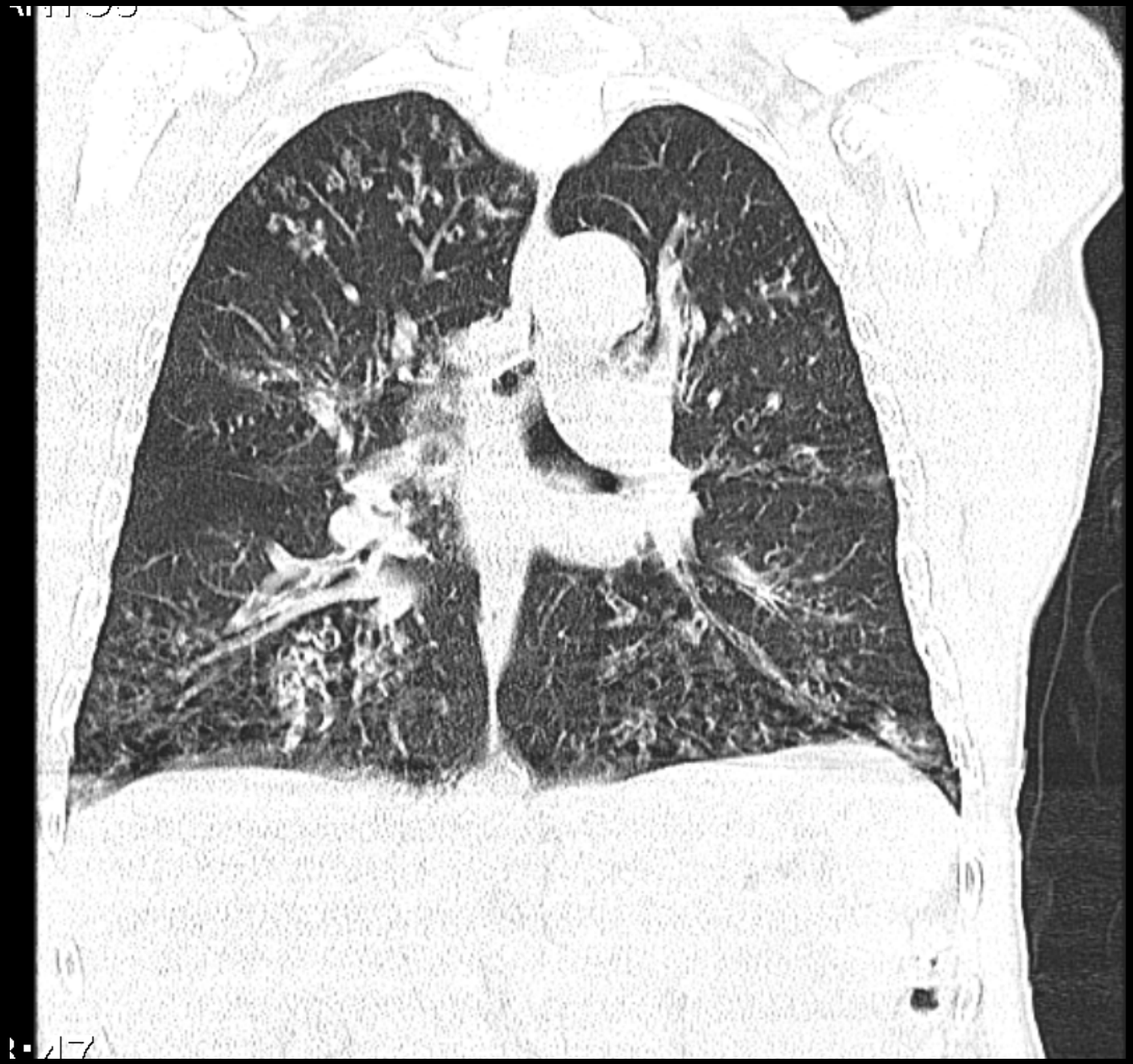

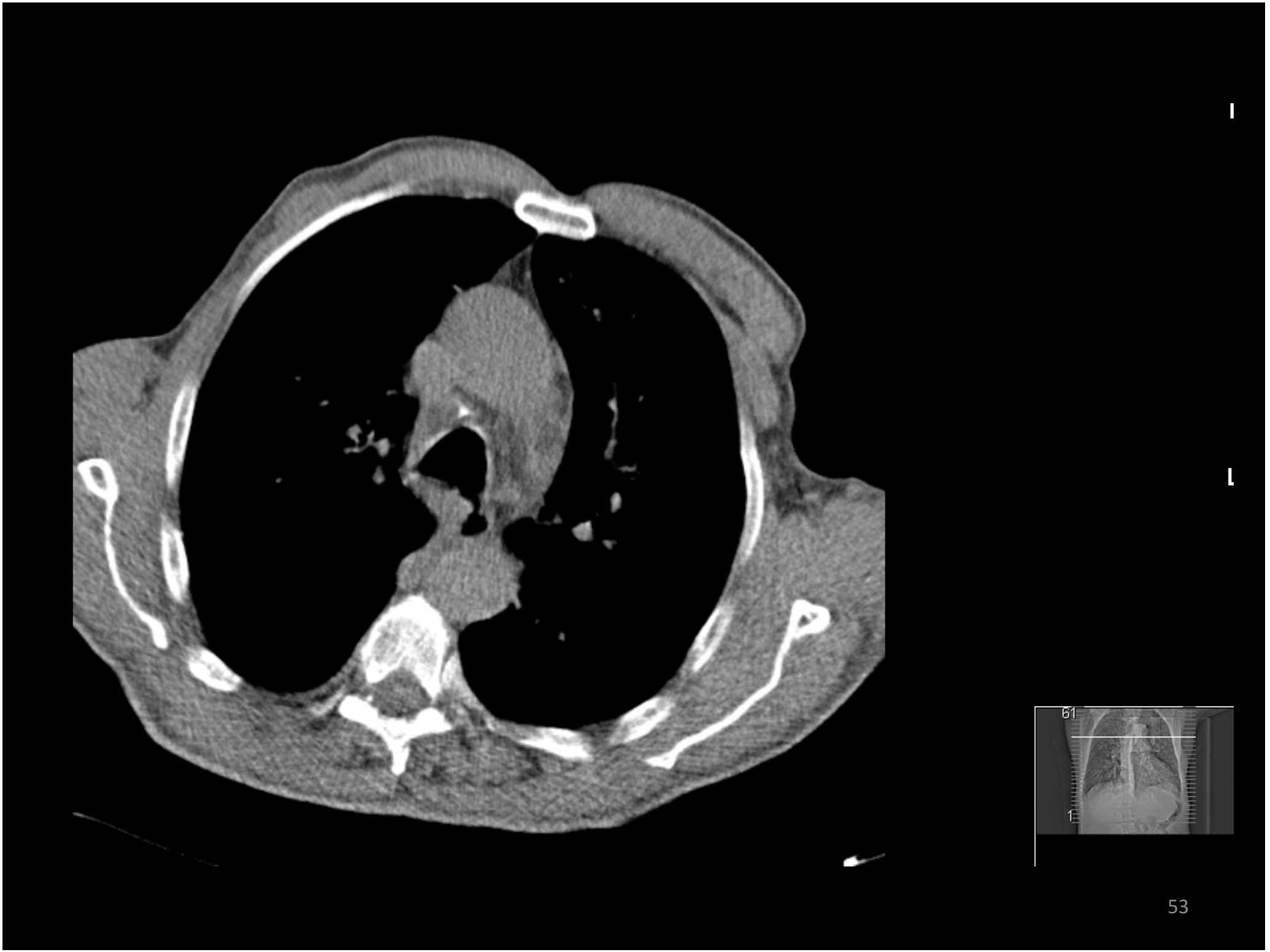
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1

Mensagens finais
Dúvidas