



Clinical Respiratory Mechanics Emphysema and Fibrosis

MEDS 230 September 2020

Meds 2023

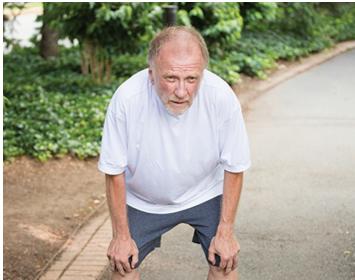
Dr. Sue Moffatt

Dr. Chris Parker



Today's Key Concepts

- Apply principles of clinical physiology to obstructive (COPD) and restrictive (Fibrosis) lung disease
 - Compliance
 - Resistance
 - Dynamic hyperinflation *
 - Regional ventilation *
 - Pulmonary function tests *



We will be able to



- Explain the clinical presentations of patients with obstructive and restrictive lung disease based on their lung mechanics
- Explain the mechanism and consequence of dynamic hyperinflation and gas trapping in obstructive disease
- Predict the response to exercise in persons with abnormal lung mechanics and explain how their dyspnea can be reduced
- Diagnose obstructive and restrictive lung disease on pulmonary function testing

From Term 1



Recall our practice with a balloon:

As you fill a balloon: where is it easier and harder to inflate?

What makes a balloon empty?

Can you make it empty more quickly?

From Term 1

What makes our lungs fill with air?

What makes them empty?

Useful Equations for Mechanics from Term 1

- Compliance = $\Delta \text{volume}/\Delta \text{ transmural pressure}$
- Flow = Pressure gradient/Resistance
- Resistance $\propto 1/r^4$
- Minute Ventilation = VT (tidal volume) X RR

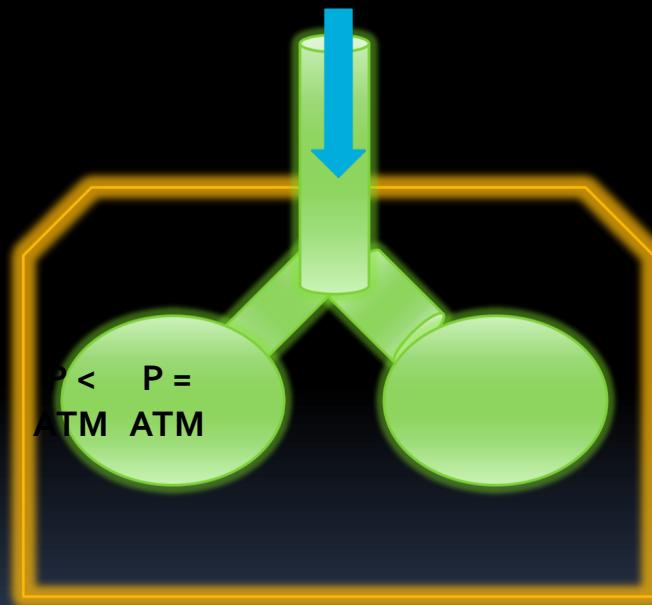
Normal Inspiration

$$P = \\text{ATM}$$

INSPIRATION:

INWARD PRESSURE GRADIENT!

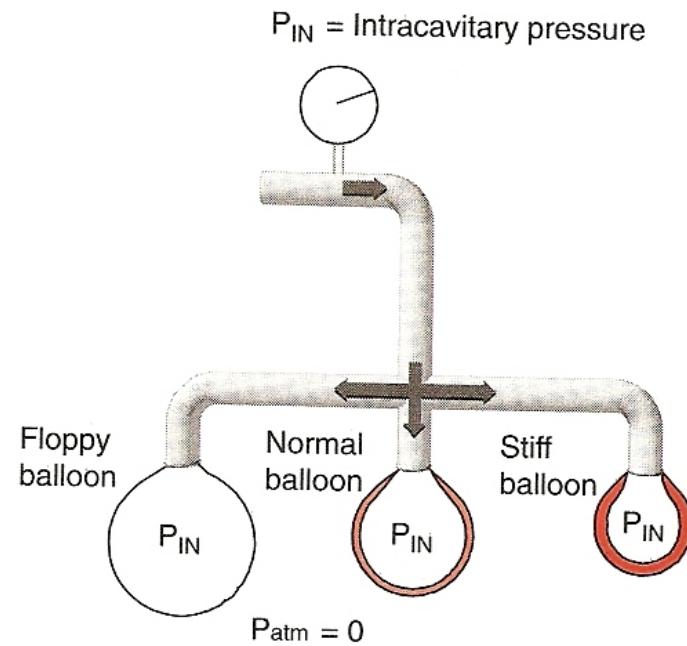
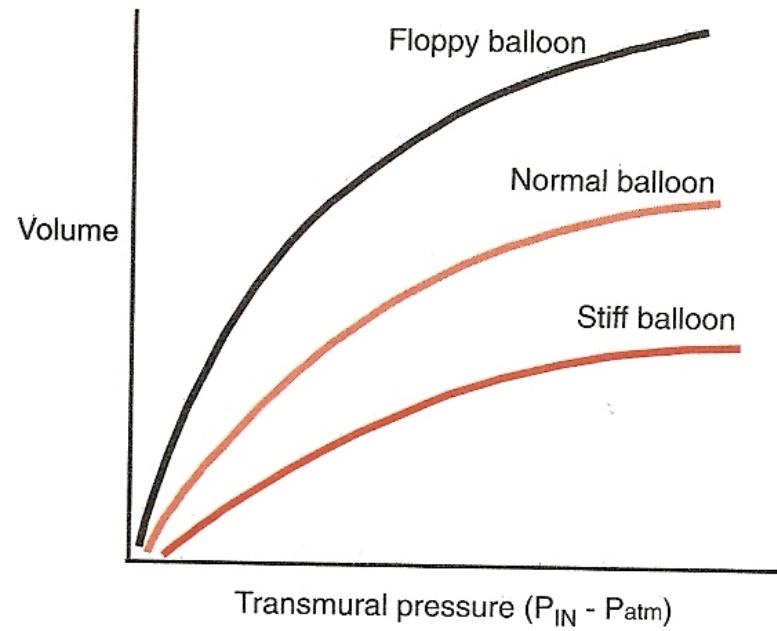
CHEST WALL (AND LUNGS) EXPAND OUTWARDS, INCREASING THEIR VOLUME



AS LONG AS CHEST IS STILL EXPANDING, PRESSURE IN THE LUNGS WILL BE < ATMOSPHERIC

Image courtesy Dr. C. Parker

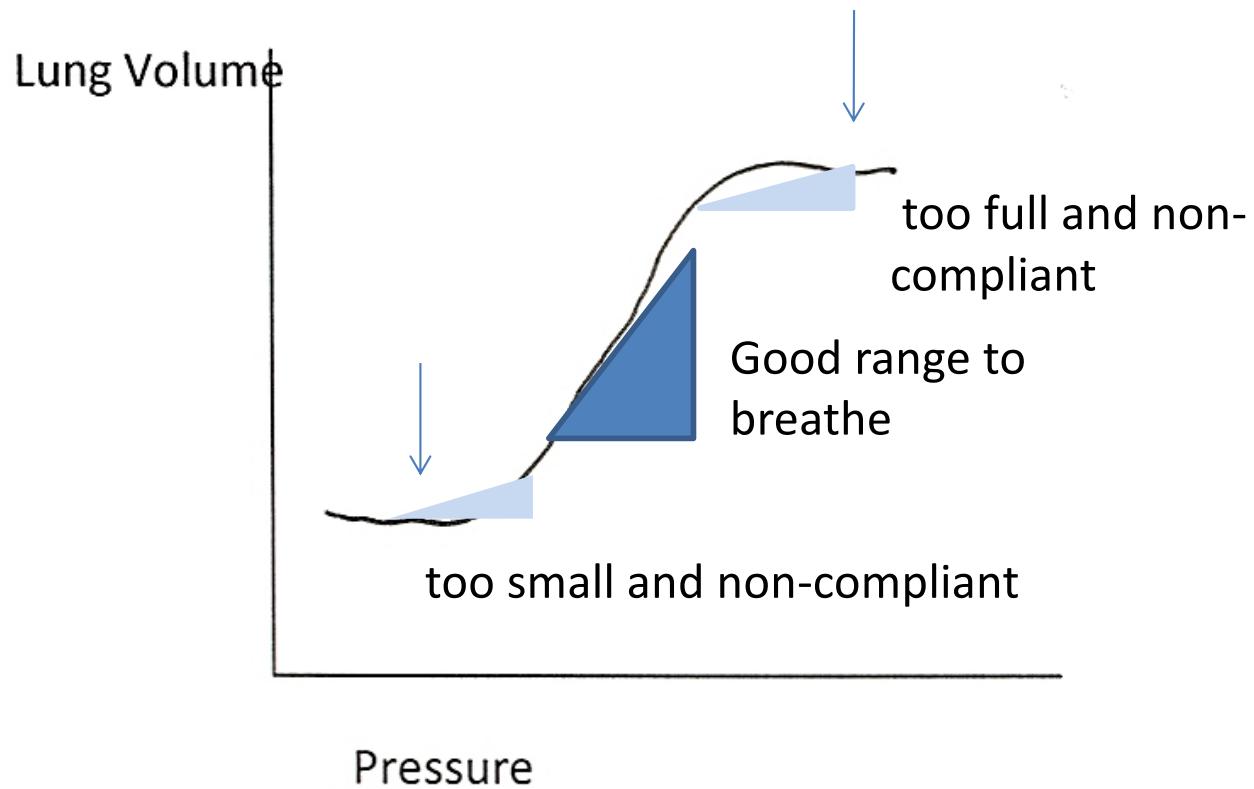
Compliance Curves



Slope of line = $\Delta V / \Delta P$ = compliance

At the same transmural pressure a low compliance (stiff) balloon is smaller than a high compliance (floppy) balloon.

Being in the Compliance “Sweet Spot”*



Surface Tension

- The liquid molecules lining the inner surface of the alveoli resist being pulled away from each other and this contributes to the compliance of the lung
- This surface tension is reduced by **surfactant**, a material secreted by the type 2 alveolar cells
- Surfactant helps to keep small alveoli from collapsing

Regional Differences in Alveolar Size and Ventilation

When pleural pressure falls with an inspiration (transmural pressure increases) the alveoli at the base of the lung increase more in size ie. get **more** ventilation than those at the top

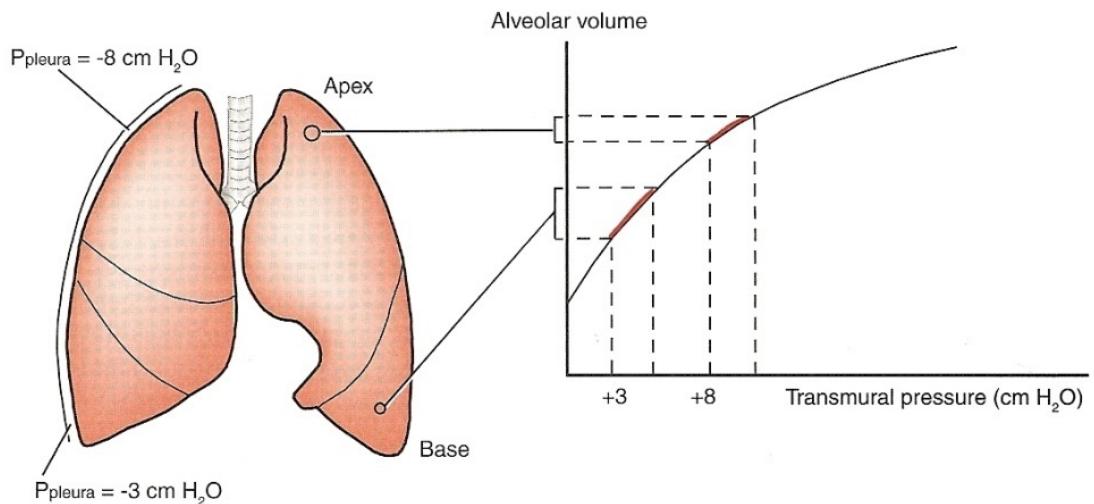
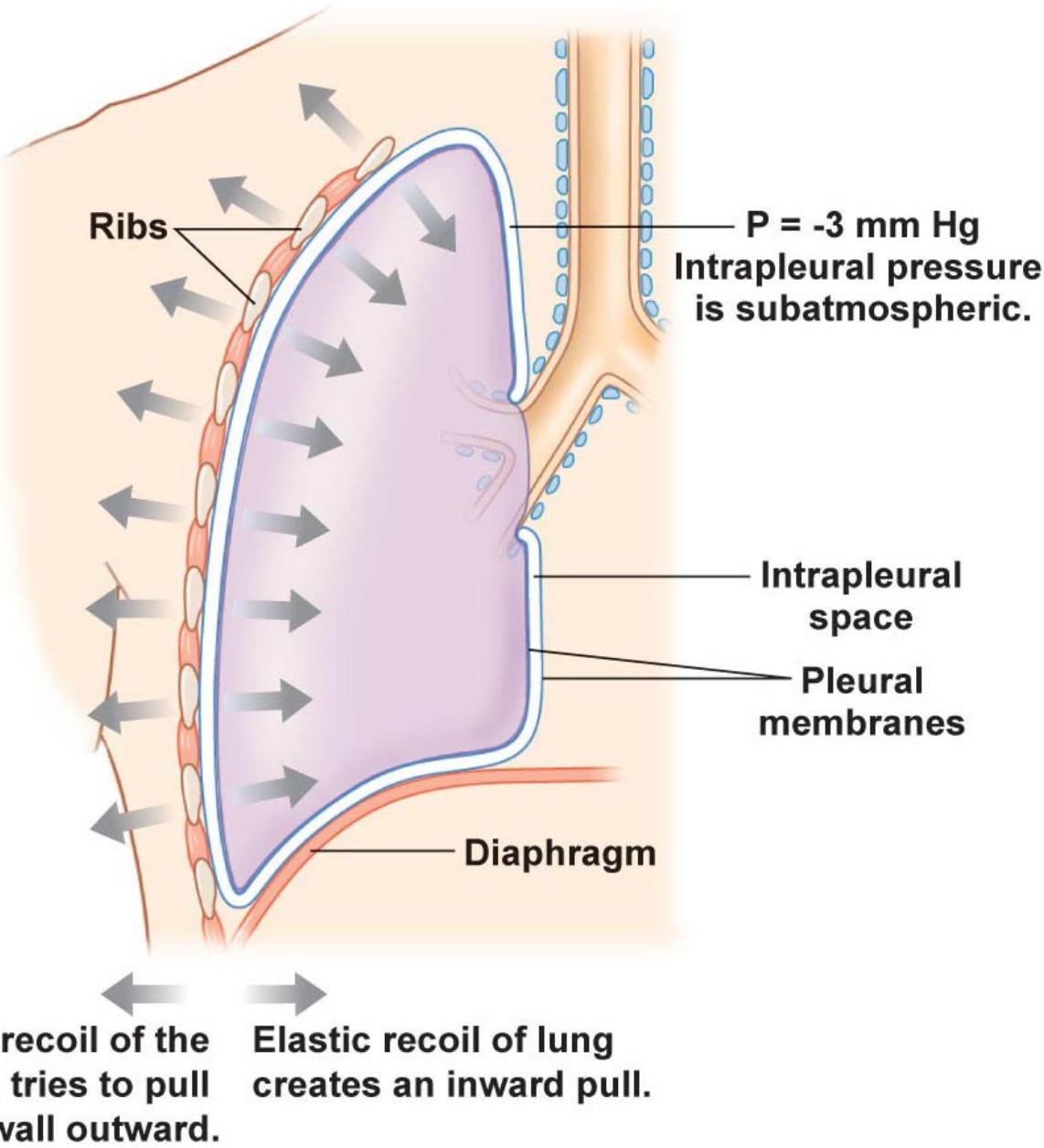


FIGURE 5.2 Pleural pressure and distribution of ventilation. At the lung volumes of normal resting breathing, apical

Schwartzstein image 5-3

Figure 17-12a

Regional differences in ventilation



(a) Normal lung at rest

From Term 1



Recall blowing through a straw:

What is the relationship between the pressure at 2 ends of a pipe, the resistance and the flow of liquid through the pipe?

Which takes longer: normal inspiration or expiration? Why?

Which is louder: normal inspiration or expiration? Why?

Pressure Gradients and Flow

$$\Delta P = (0-0) = 0$$

NO FLOW



$$\Delta P = (150-150) = 0$$

NO FLOW



$$\Delta P = (150 - 0) = 150$$

FLOW!



Image courtesy Dr. C. Parker

Radius, flow and velocity in airways

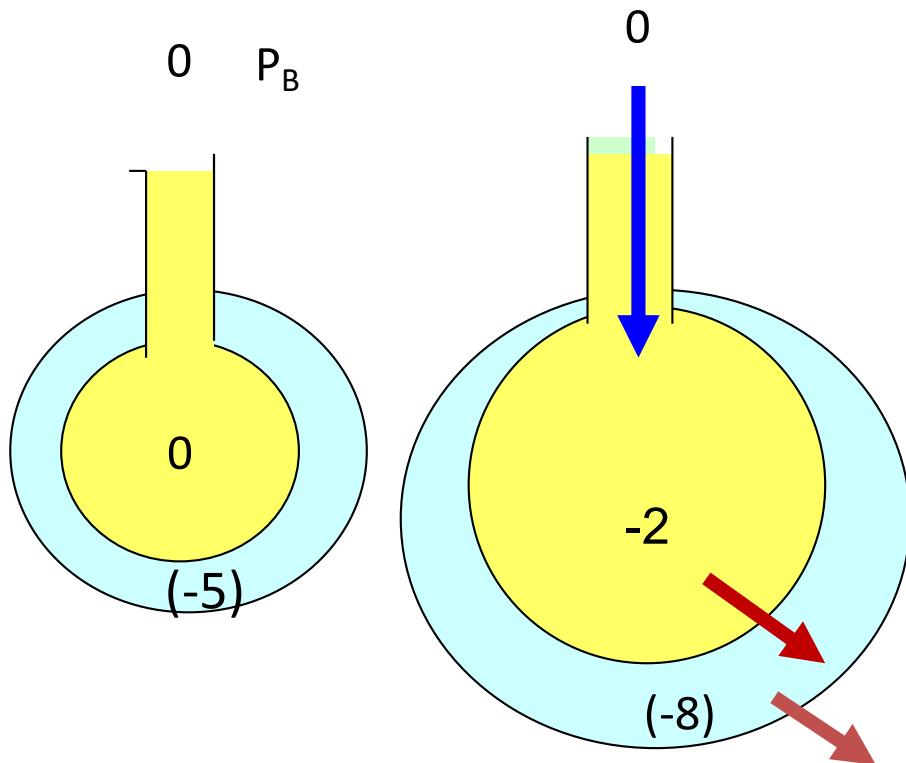
$$R \propto 1/r^4$$

Trachea and proximal airways have smallest net radius → highest velocity + turbulence → “breath sounds”

	Name	Division	Diameter (mm)	How many?	Cross-sectional area (cm ²)
Conducting system	Trachea	0	15-22	1	2.5
	Primary bronchi	1	10-15	2	
	Smaller bronchi	2			
		3			
		4	1-10		
		5			
	Bronchioles	6-11			
Exchange surface				1×10^4	
	Alveoli	12-23	0.5-1	2×10^4	100
				8×10^7	5×10^3
		24	0.3	$3-6 \times 10^8$	$>1 \times 10^6$

Terminal bronchioles: same flow through huge # bronchioles = less flow/airway = low velocity = quiet

Putting it Together: Normal Inspiration



Chest wall and lung move together

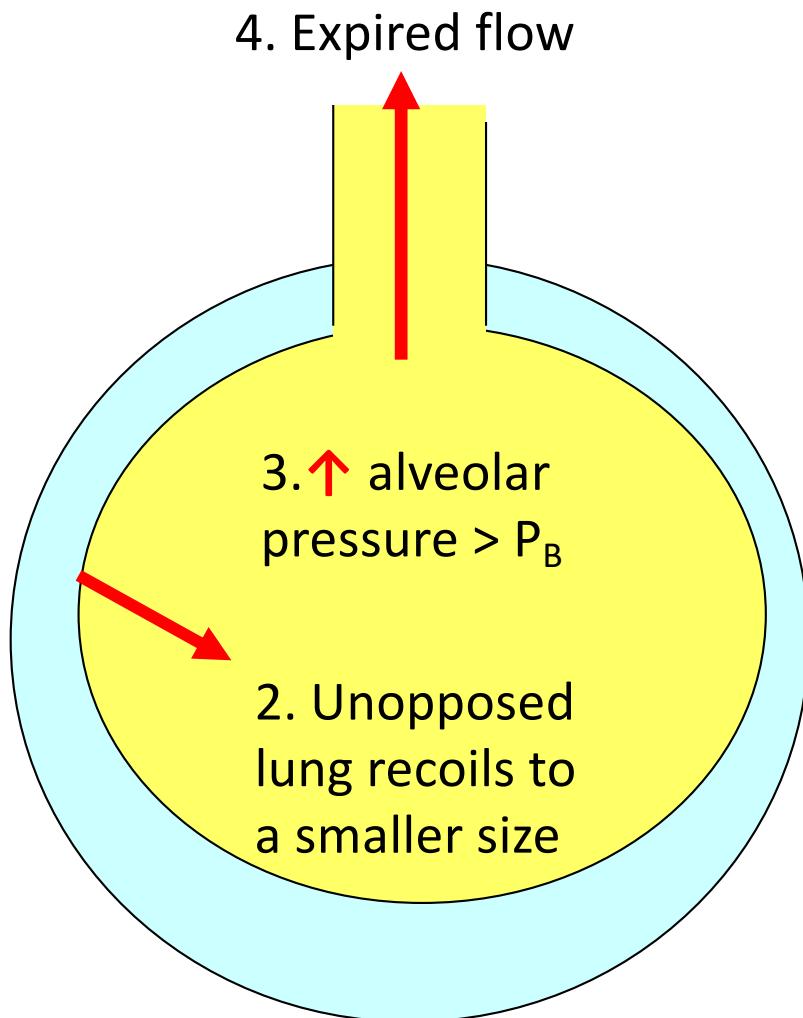
Diaphragm and chest wall muscles pull on chest wall → more -ve pleural pressure that pulls lung open

→ alveolar pressure < atmospheric

→ causes flow and lung expansion

Putting it Together: Normal Expiration

1. Chest wall muscles relax and pleural pressure becomes less negative



Normal expiration is passive, **using stored elastic recoil energy** in lung and chest wall

Pleural pressure remains < atmospheric

Summary: Lung Compliance

- Breathing requires changing the shape of the lung and thorax.
- Compliance = Δ Volume/ Δ Pressure
- Inspiration requires work to stretch lung tissue
- Normal expiration is effortless and depends only on the natural recoil of the stretched lung tissue
- Lung compliance is lowest when lung volume is very small or very large
- The alveoli at the bottom of the lung are smaller than those at the top, but expand more with inspiration (receive more ventilation).

Summary: Airways Resistance

- Ohm's law: Flow = Pressure difference
Resistance
- R varies $1/r^4$
- The difference between alveolar and atmospheric pressure determines the direction of airflow
- The elastic recoil of lung tissue generates the pressure for normal relaxed expiration

Summary: Airways Resistance

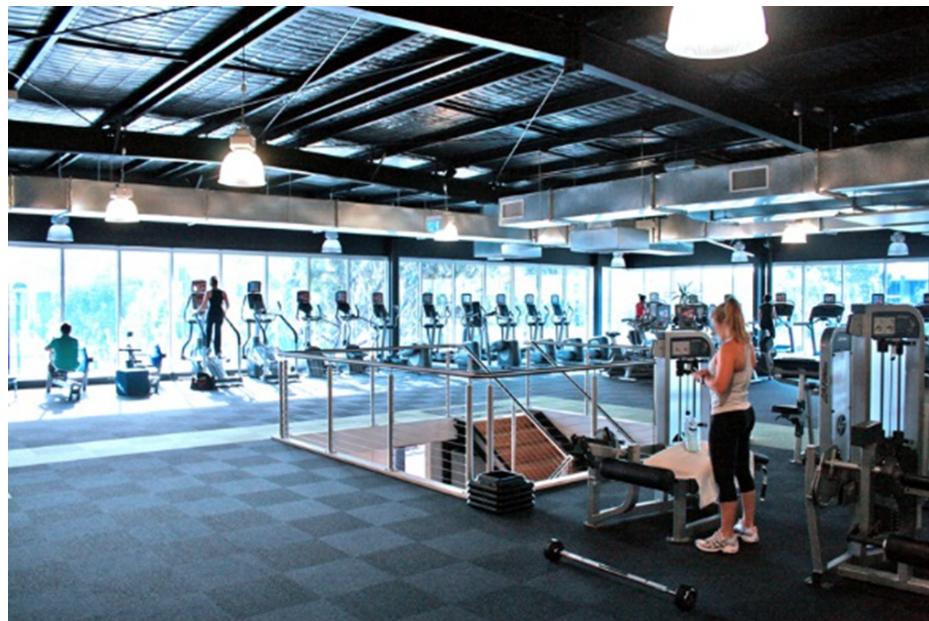
- Airways resistance is greater in expiration as the conducting airways get smaller
- Increased airways resistance increases the work of breathing
- Increased airways resistance can lead to gas trapping on expiration when the respiratory rate increases
- An effective cough is essential to keep the conducting airways clear



Questions?

You and Edna and Giles

You have recently joined Good Life and decide to take a fitness class. There you meet Edna and Giles. They learn you are a second year medical student and you visit as you warm up.



Edna



Edna

She is a 60 year old woman, smoked for 45 years, but quit 6 months ago.

She has a daily cough productive of white sputum.

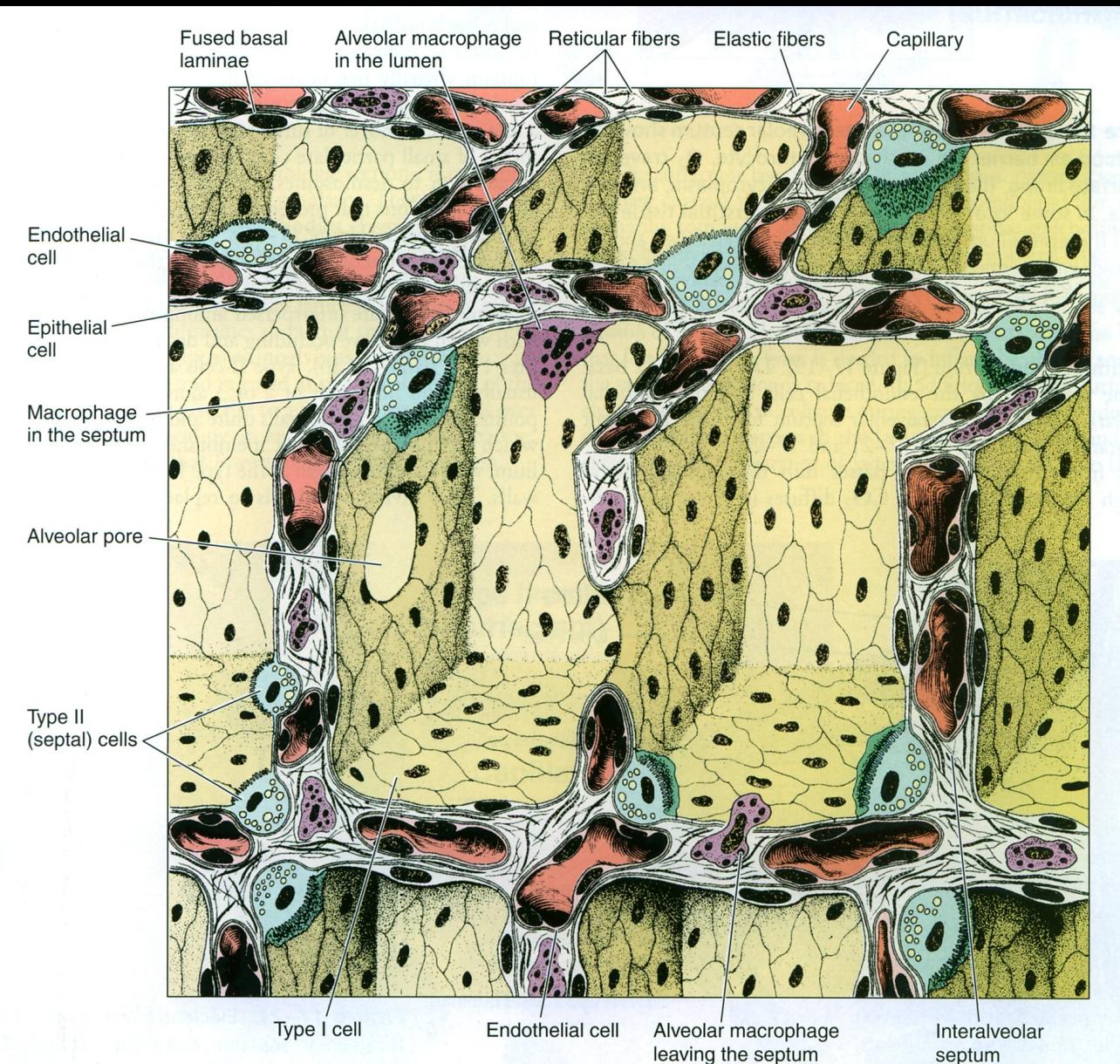
She is short of breath with climbing stairs, carrying groceries, hurrying, anxiety.

- She has been told she has chronic obstructive pulmonary disease and has joined the gym to improve her exercise tolerance

What is Emphysema?

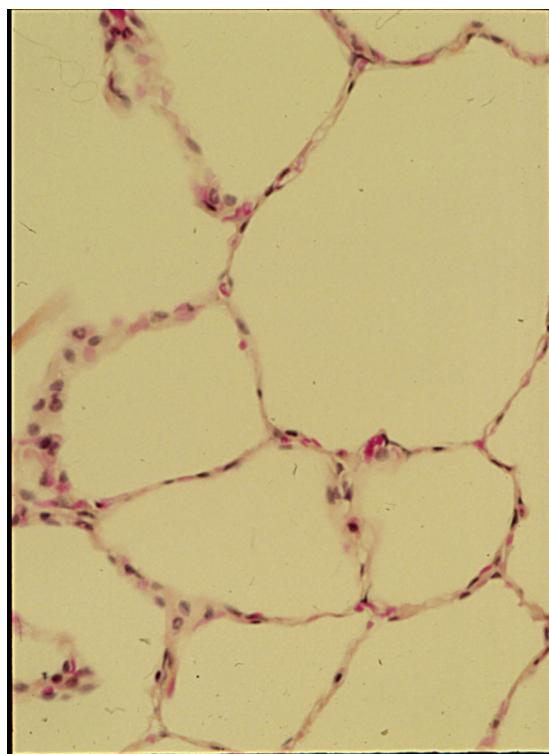
Loss of lung elastin in alveolar tissue

- Increased compliance and **decreased recoil**
- Breakdown of individual alveoli into large **bullae with trapped gas**
- Loss of surface area for gas exchange
- Loss of small airway stabilization → ↓ radius of multiple small airways → **↑ airways resistance**

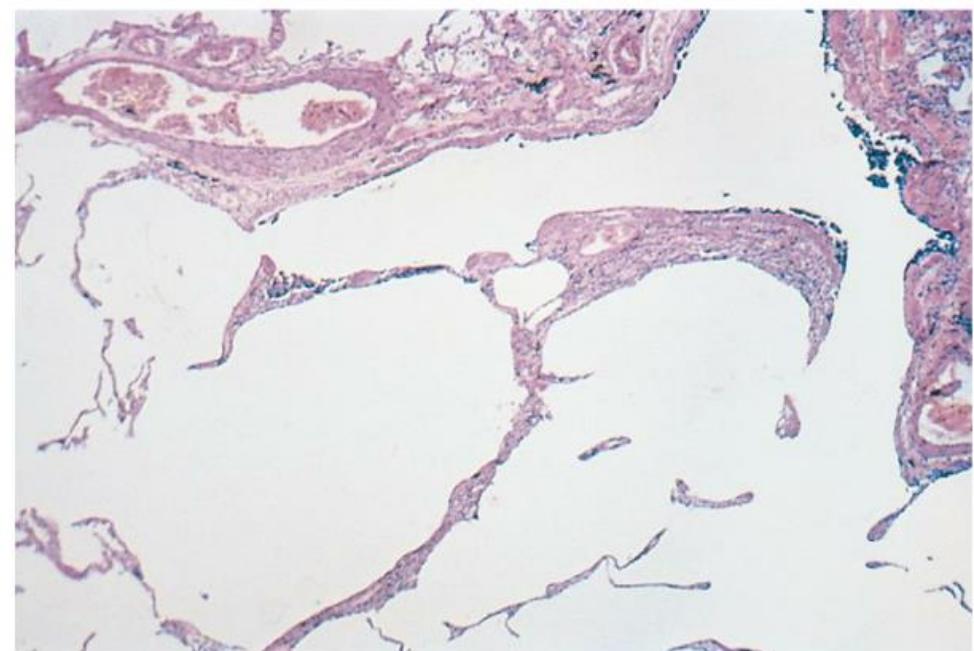


Histopathology of Emphysema

Normal

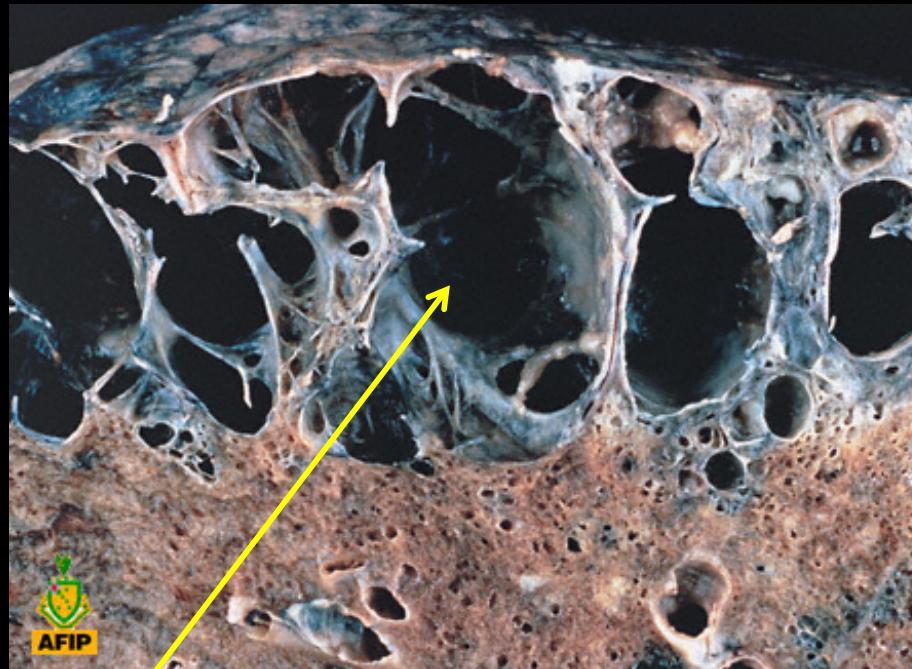


Enlargement of airspaces and thinning of alveolar septa in emphysema



© Elsevier. Kumar et al: Robbins Basic Pathology 8e - www.studentconsult.com

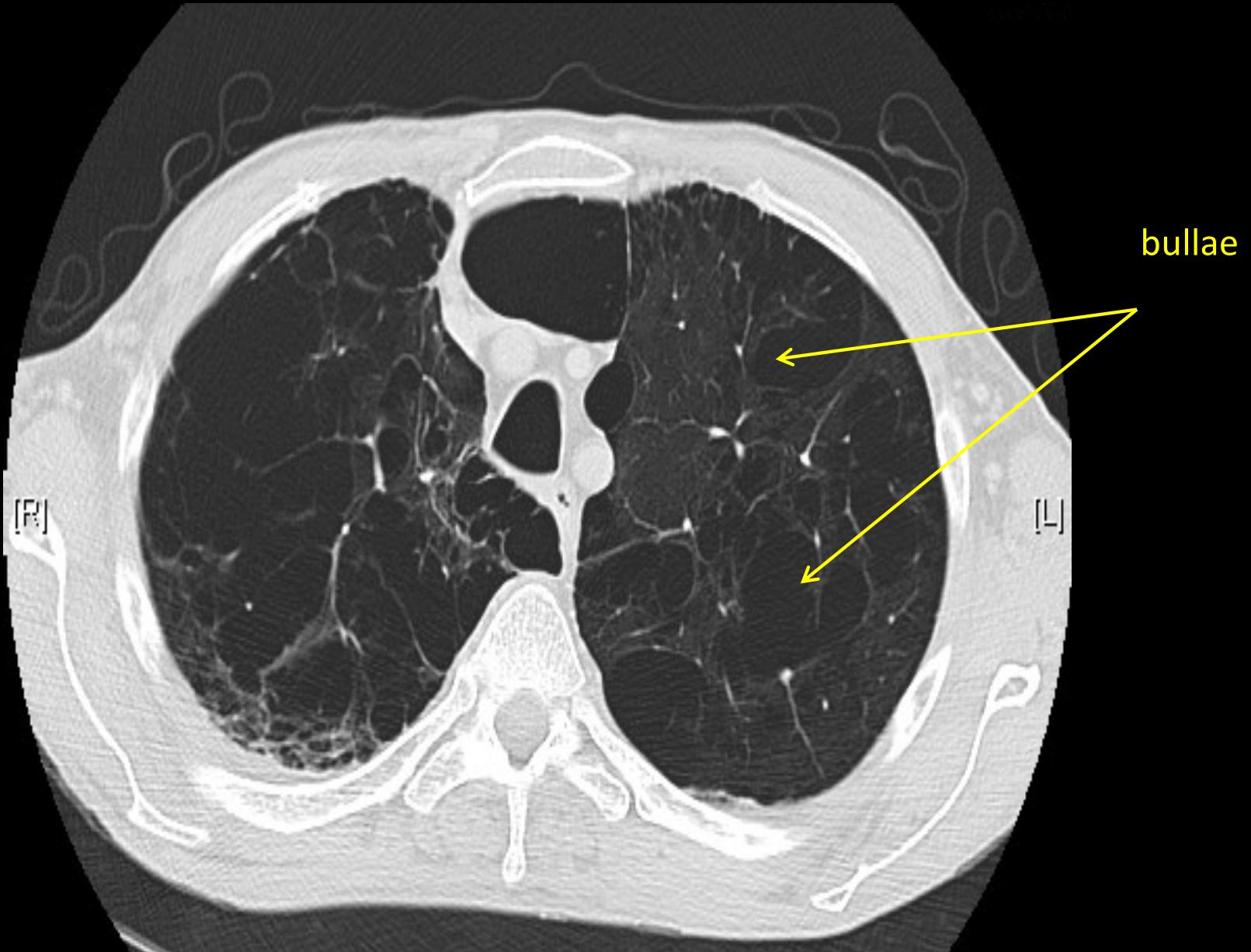
Emphysema



Emphysema bullae

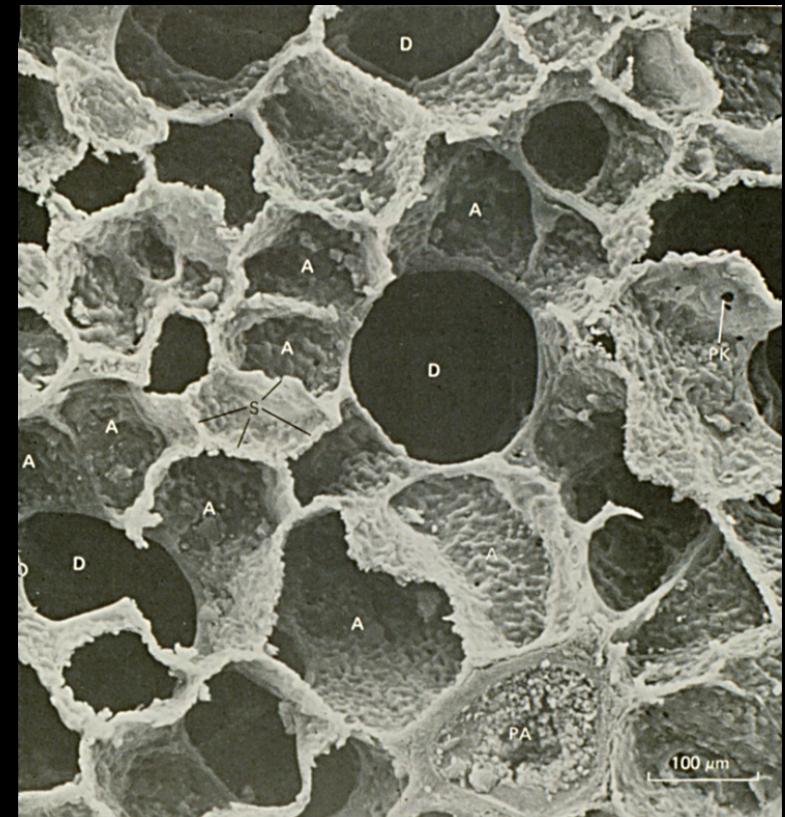
Notice the loss of surface area for gas transfer.

Edna's CT Scan



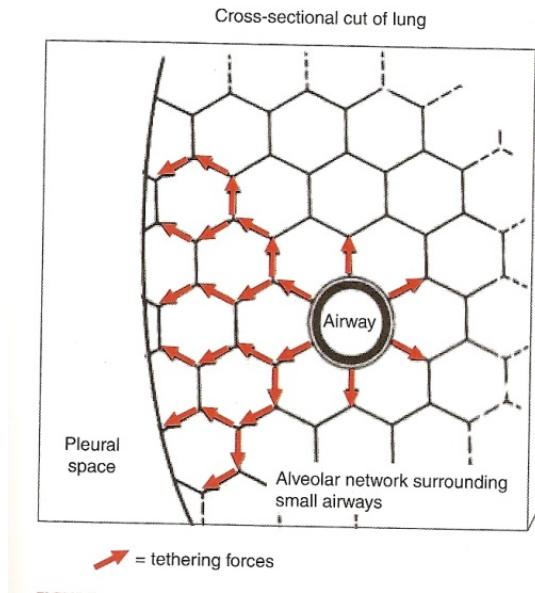
Normal Lung

Notice how the alveoli (A) surround the small airway duct (D). During expiration, the elastic recoil of normal alveoli pull on the small airways and help to keep them open as the lung empties

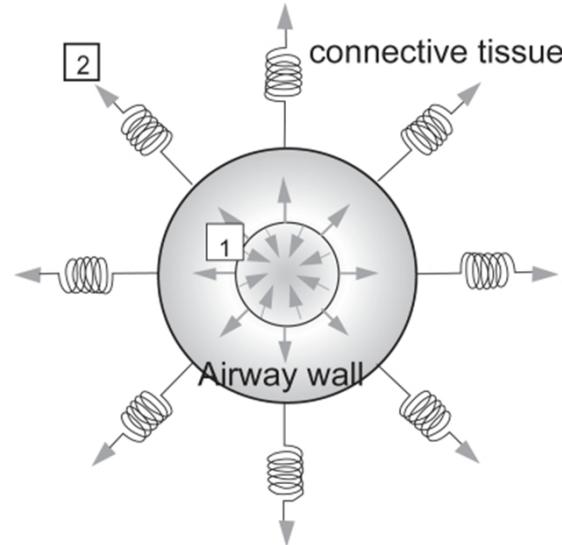


Normal lung (electron microscope)

Interdependence



Schwartzstein Fig 4.9



NIH

- The alveolar tissue acts like stays on a tent, pulling the conducting airways open with its recoil
- Low recoil alveoli don't hold the conducting airways open
 → \uparrow airway resistance
- High recoil alveoli do hold the airways open
 → \downarrow airway resistance

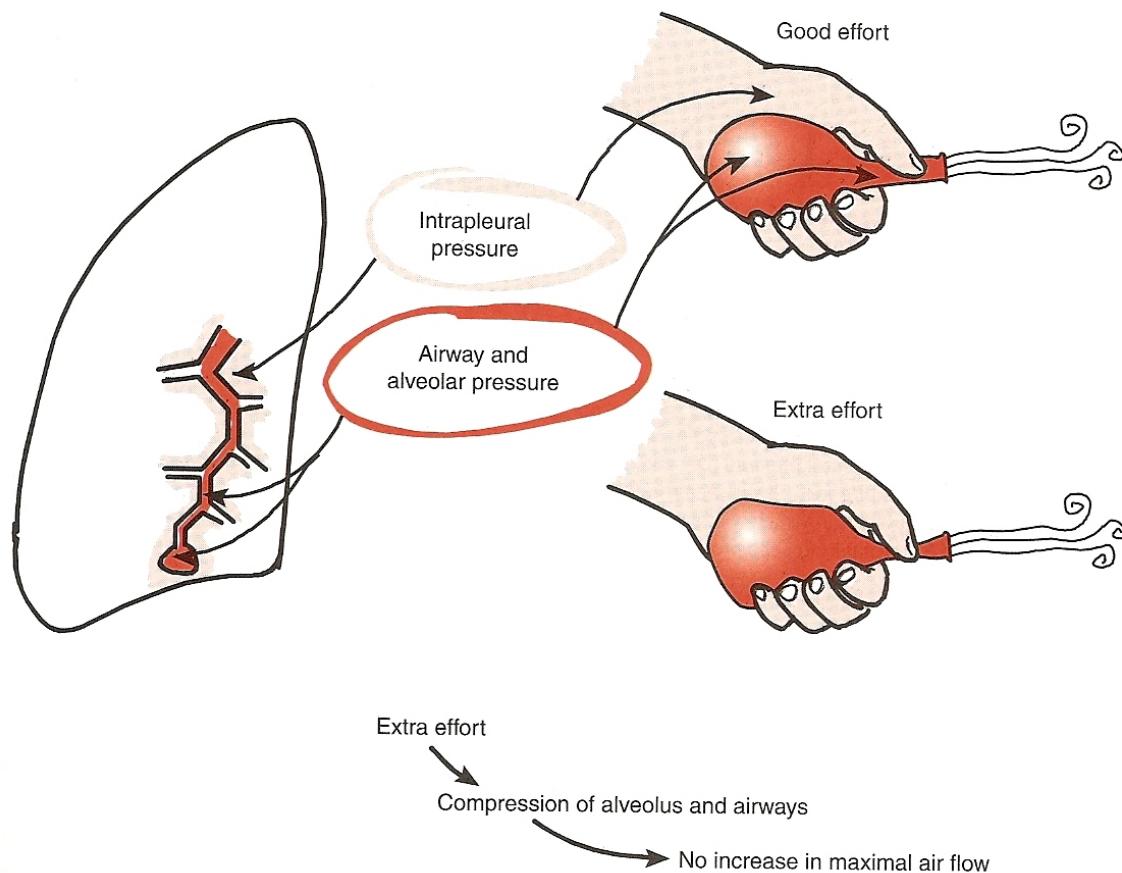
Edna: Microthink

Why can't Edna "force" more air out of her lungs?

Why does Edna purse her lips on expiration?

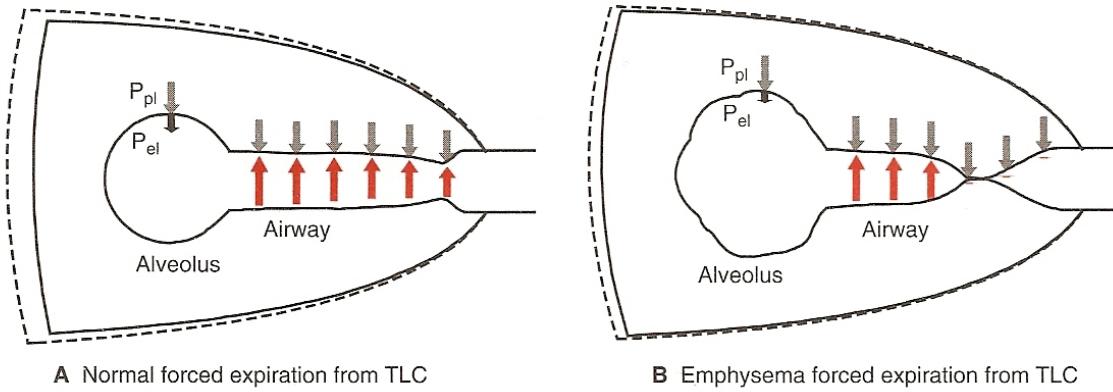
Why does Edna wheeze more on expiration than inspiration?

Expiratory Flow Limitation: why trying harder doesn't help COPD



Schwartzstein Fig 4.14

Expiratory Flow Limitation, Gas Trapping and Pursed Lipped Breathing



When intrathoracic airways collapse before exhalation is finished the gas is “trapped” in the lung

- the chest is uncomfortably full
 - The faster Edna tries to breathe the more breaths get “stacked”
 - This results in dynamic hyperinflation and the sensation that “I can’t get a breath in”
- Pursed-lipped breathing** stents the intrathoracic airways, slowing exhalation, allowing the lung to empty more completely between breaths

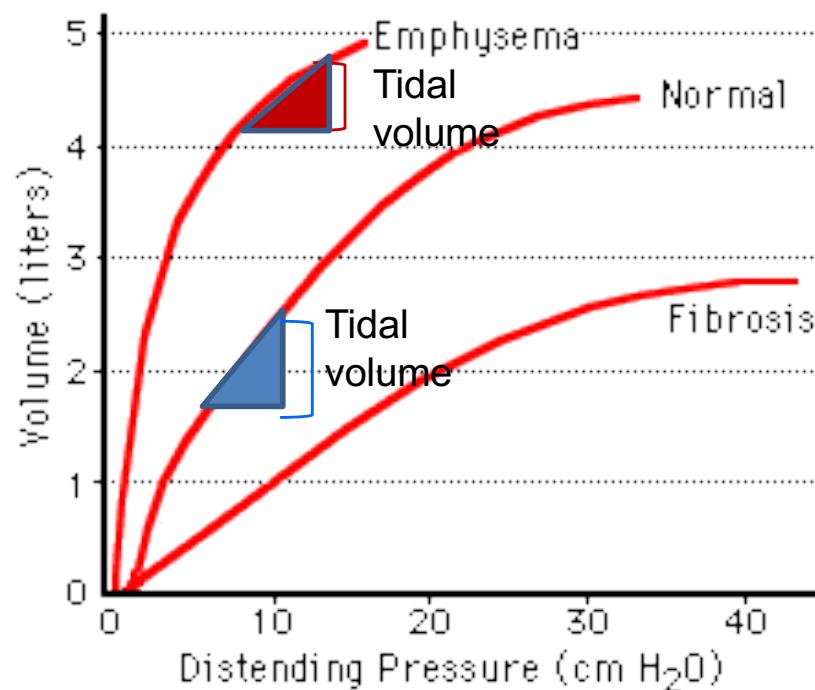
Emphysema

Patients with **emphysema** experience breath stacking (gas trapping) due to decreased elastic recoil and increased airway resistance.

When RR increases, they end up on the flat (top) part of their compliance curve.

The must work harder to get a breath = dyspnea.

Asthma causes a similar problem due to high airway resistance



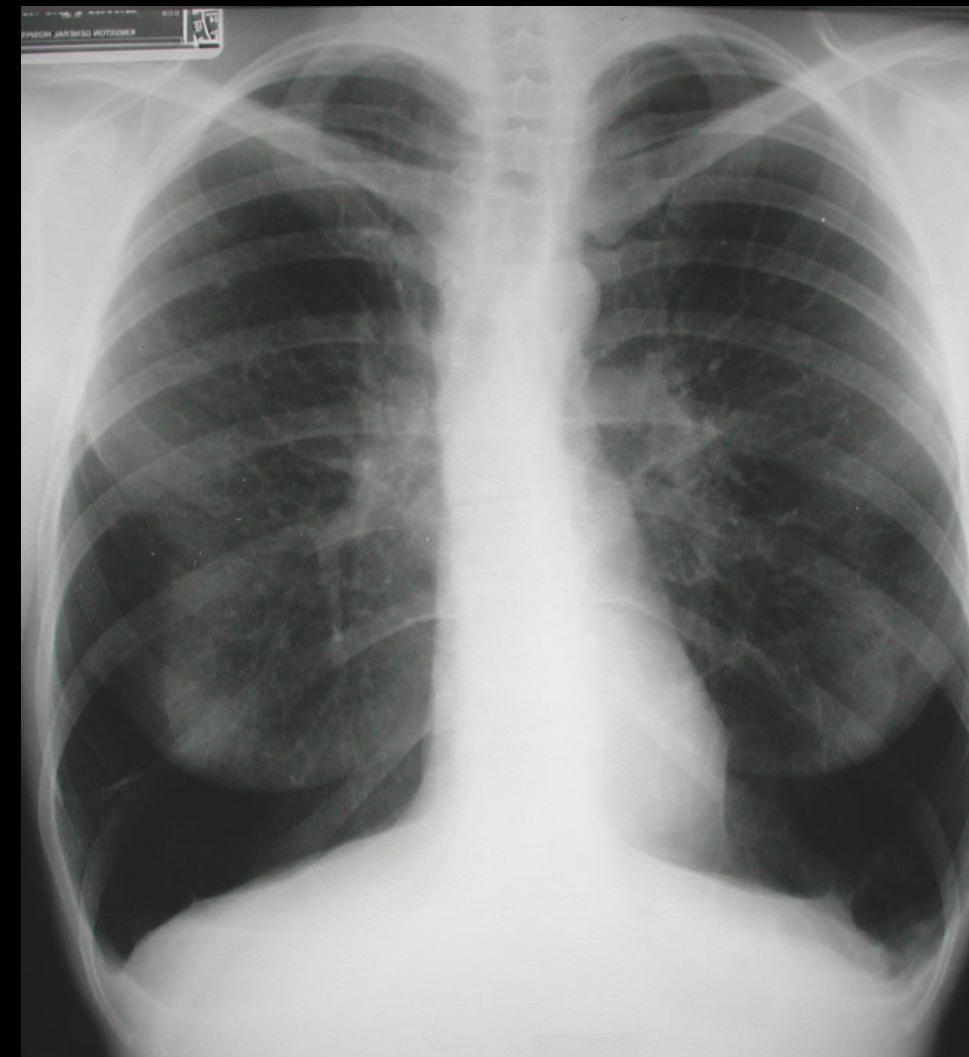
Edna's CXR

(note the flattened diaphragm position = hyperinflation)

Normal



Edna



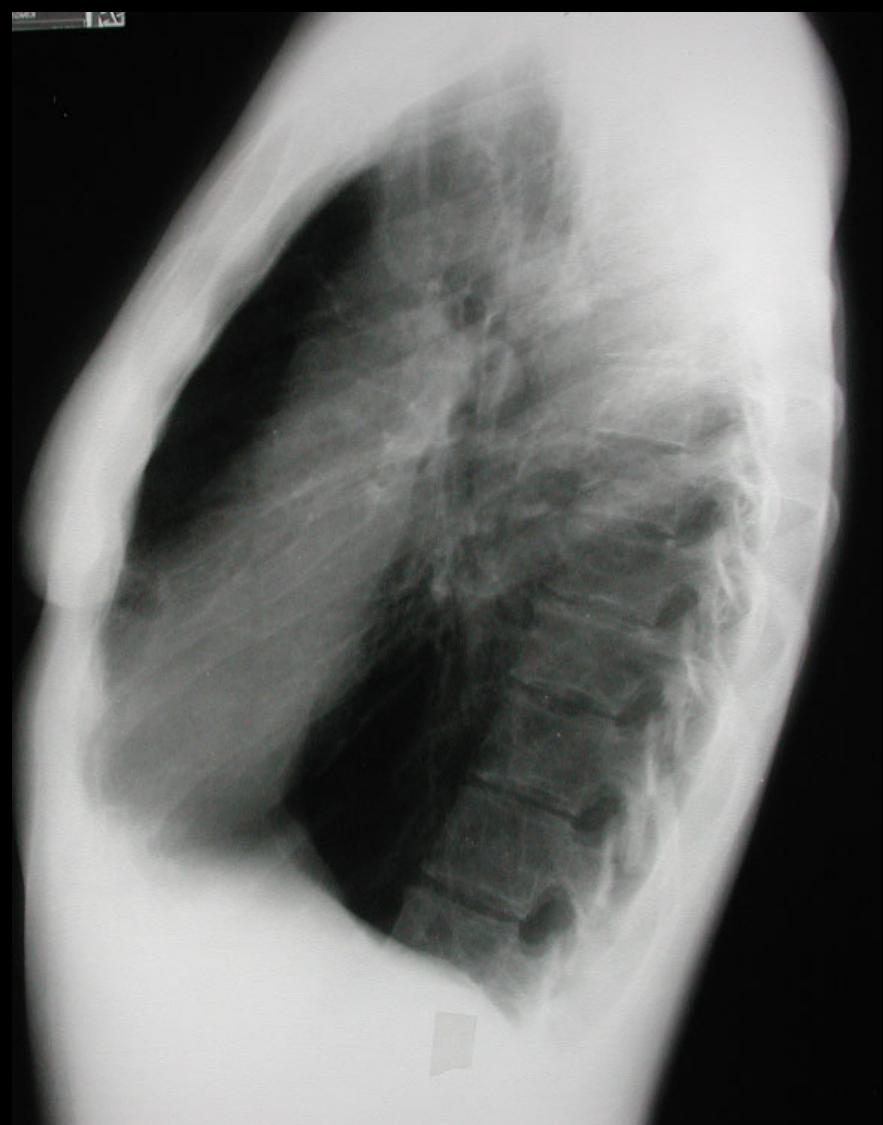
Edna's CXR

(note the diaphragm position = hyperinflation)

Normal



Edna



Edna: Microthink

Recall Ohm's Law: (Flow = $\frac{\Delta P}{R}$)

R

Why is Edna more breathless when she hurries or is anxious?

Why is Edna more breathless when she bends over or eats a big meal?

Summary Slide: Clinical Mechanics of Emphysema



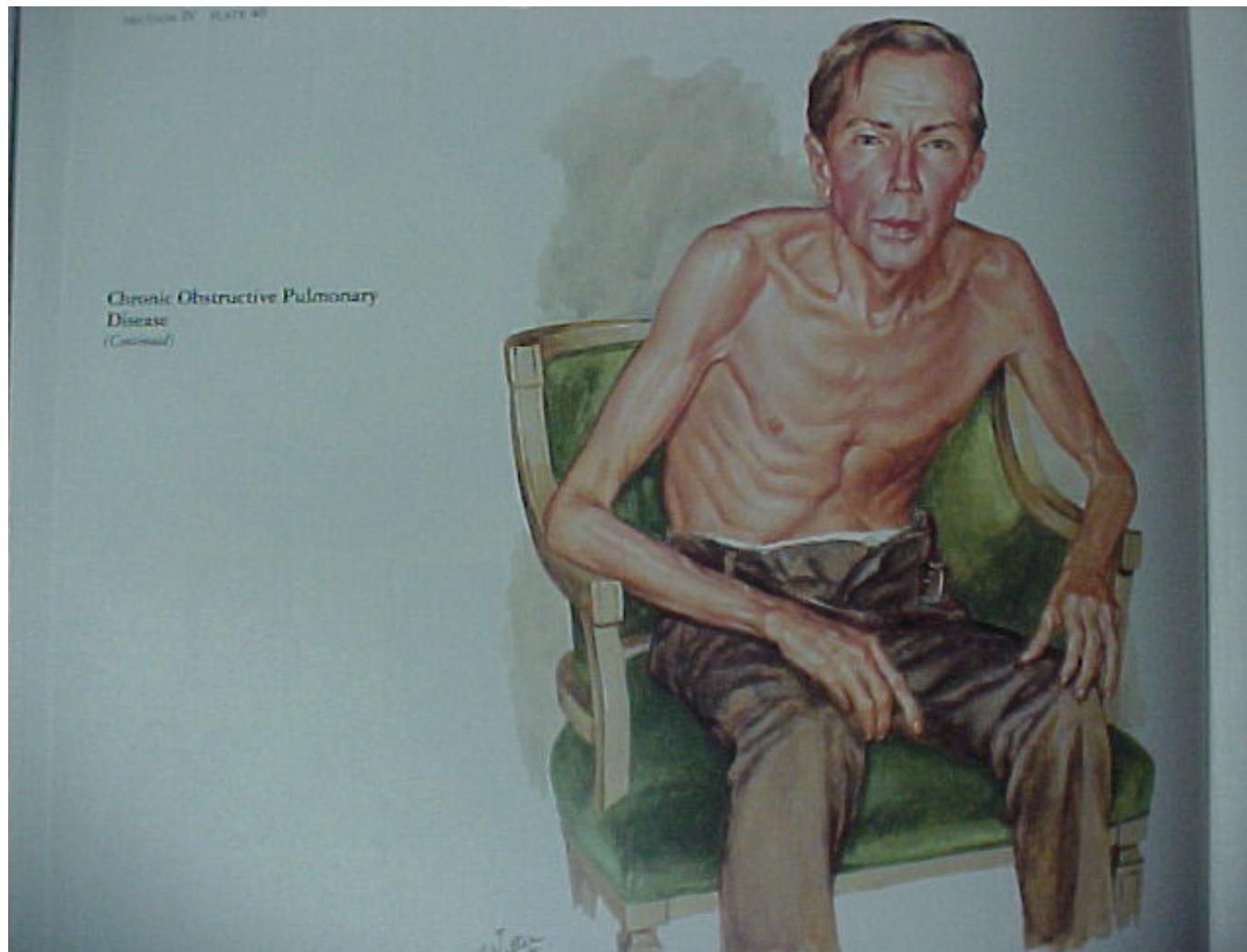
- Loss of elastin results in **decreased lung recoil** (increased compliance) and abnormally slow lung emptying
- **Increased airway resistance** due to loss of airway stability also slows expiration
- Lung emptying is incomplete particularly when the respiratory rate increases →hyperinflation + gas trapping → discomfort = dyspnea
- Hyperinflation prevents ↑tidal volume on exertion and this limits ability to exercise

Summary Slide: Clinical Mechanics of Emphysema

- Due to lung hyperinflation, the diaphragm is flattened, and less efficient than normal
- Wheezing is louder on expiration because airways are getting smaller. Wheezing is dependent on effort
- There is loss of surface area for gas transfer, contributing to hypoxemia on exertion

The Work of Breathing in COPD

Note: posture, accessory muscles, pursed Lips breathing, soft-tissue in-drawing, open waist band



F. Netter
c/o Dr. Moran

Giles



Giles

He is a 60 year old retired teacher.

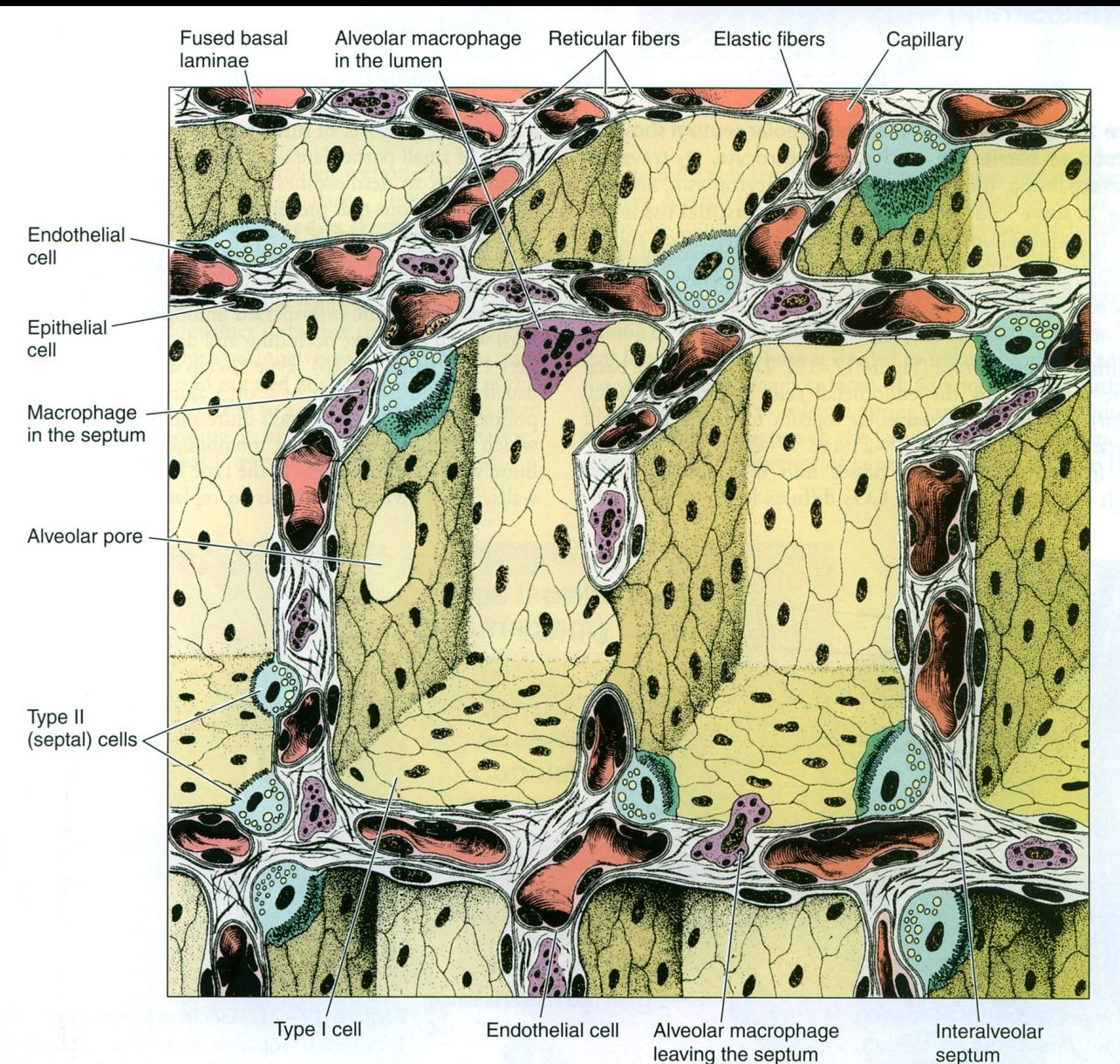
He has an increasingly frequent dry cough over last 3 months.

He has progressive shortness of breath over 6 months, now having trouble keeping up with his wife when they are walking together.

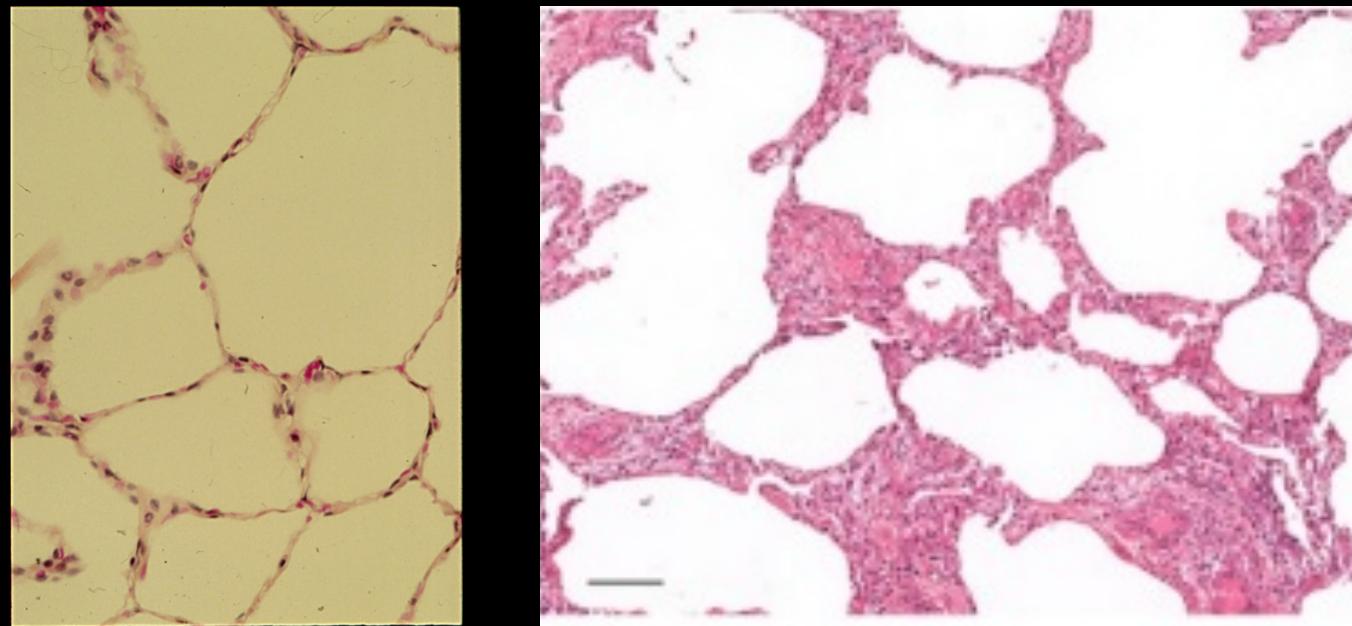
- He has been told he has interstitial pulmonary fibrosis

What is Pulmonary Fibrosis?

- Thickening of the alveolar tissue with fibroblasts and obliteration of alveolar capillaries.
- Increased lung recoil and decreased compliance
- Reduced gas exchange/diffusion



Histopathology of Fibrosis = Thickening of the Alveolar Walls

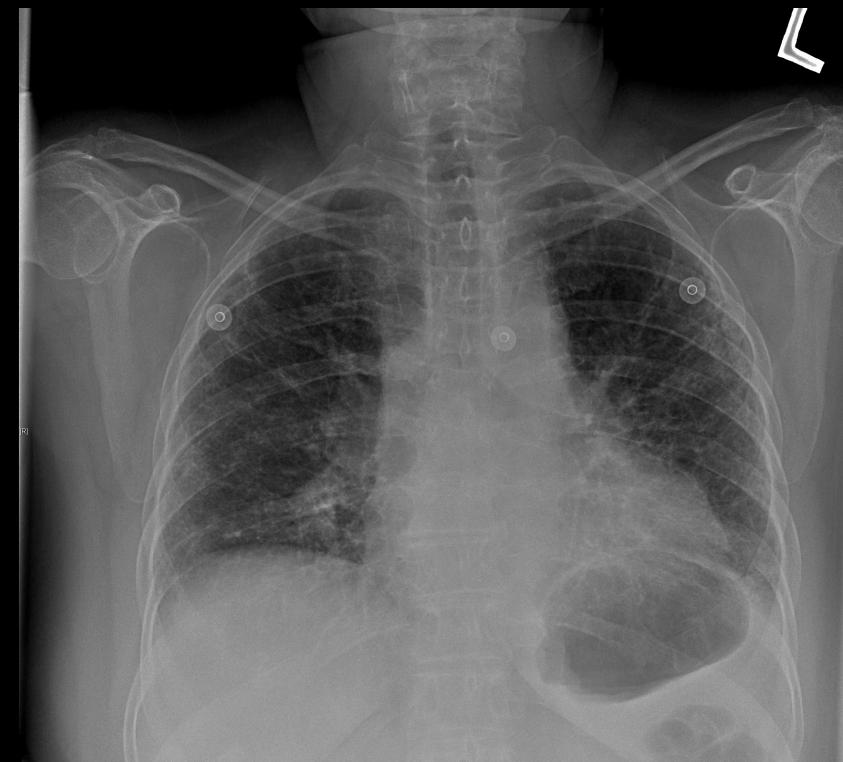


Giles' CXR

Normal

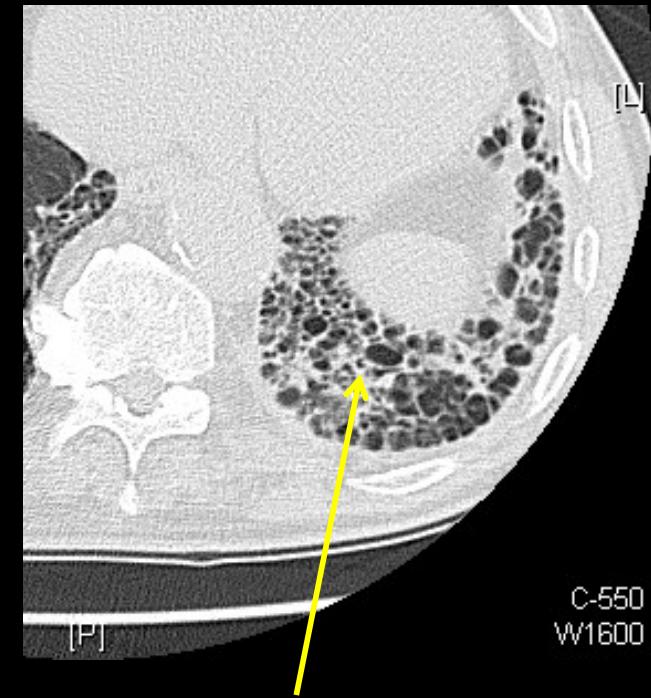
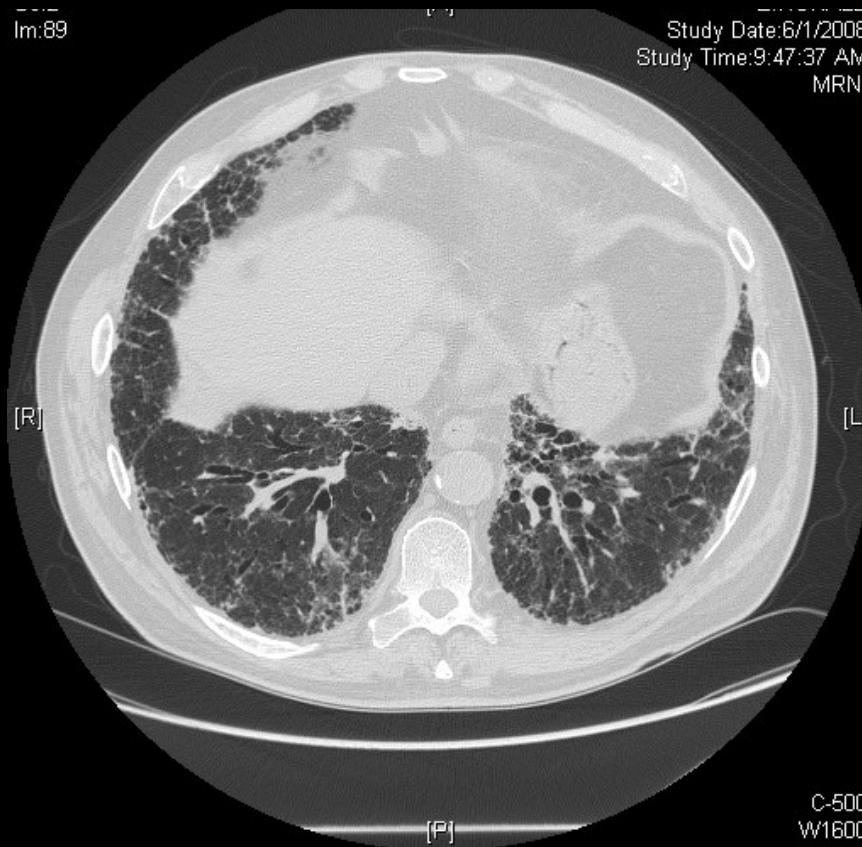


Giles



Note increased density of
the interstitial tissue and
the small lung volumes

Giles' CT Scan

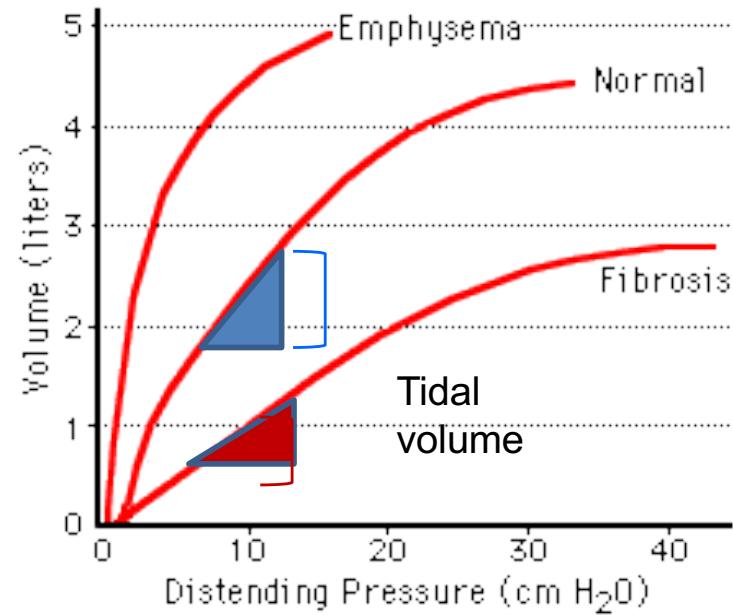


Thickening of interstitial
tissue (alveolar walls)

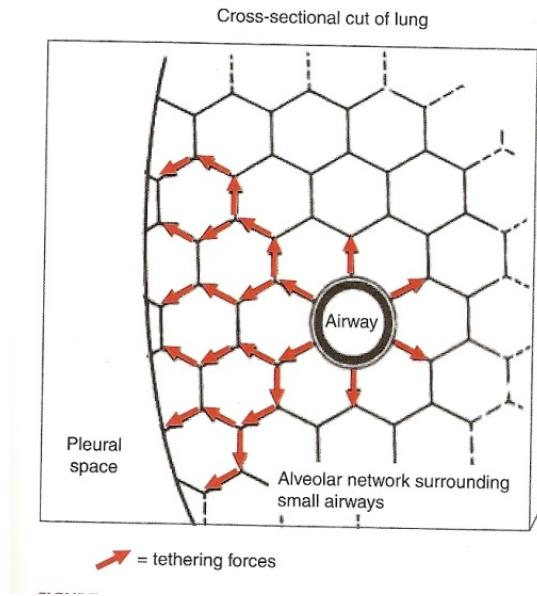
Pulmonary Fibrosis (High Elastic Recoil)

Patients with pulmonary fibrosis have ↓lung compliance and ↑ recoil.

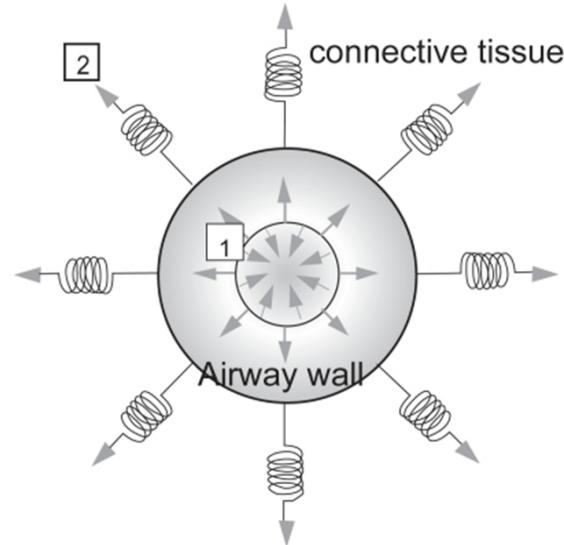
To get a normal tidal volume they have to work harder (increase their distending pressure) and this is perceived as dyspnea.



Interdependence



Schwartzstein Fig 4.9



NIH

- The alveolar tissue acts like stays on a tent, pulling the conducting airways open with its recoil
- High recoil alveoli hold the airways open
→ ↓ airway resistance

Giles

Giles seems comfortable while talking with you, but you notice his respiratory rate is 20.

You also notice that his saturation just talking to you is 92% while yours is 97%.

Giles: Microthink

Why is Giles' respiratory rate at rest higher than yours?

What additional lung sounds would you expect to hear on auscultation of his chest? Why?

Where would they be most obvious?

Would pursed lipped breathing help Giles?

Why is Giles' saturation lower than yours?

Summary: Clinical Mechanics of Pulmonary Fibrosis

- Thickening of alveolar capillary membranes and obliteration of pulmonary capillaries results in **decreased diffusion**→ **hypoxemia** that is worse on exertion.



Summary: Clinical Mechanics of Pulmonary Fibrosis

- The abnormally thick alveolar walls result in low compliance lungs with **increased elastic recoil**
- It takes more work than normal to create a tidal volume, and lungs recoil to a smaller volume than normal
- **Airway resistance is reduced** because of high recoil pulling on small airways
- Inspiratory **crackles** on auscultation reflect opening of stiff alveoli
- Low compliance limits ↑tidal volume on exertion → ↑↑ RR → rapid shallow breathing pattern and uncomfortable **dyspnea**

Summary Slide: Clinical Mechanics of Pulmonary Fibrosis

- The abnormally thick alveolar walls result in low compliance lungs with **increased elastic recoil**
- It takes more work than normal to create a tidal volume, and lungs recoil to a smaller volume than normal
- **Airway resistance is reduced** because of high recoil pulling on small airways
- Inspiratory **crackles** on auscultation reflect opening of stiff alveoli
- Low compliance limits ↑tidal volume on exertion → ↑↑ RR → rapid shallow breathing pattern and uncomfortable **dyspnea**



CHRIS M. PARKER MD MSc FRCPC FCCP

DEPARTMENTS OF MEDICINE AND PHYSIOLOGY
RESPIRATORY AND CRITICAL CARE MEDICINE

cm.parker@icloud.com

MEDS 230: Circulation and Respiration

PULMONARY FUNCTION TESTING: ANCILLARY TESTING FOR PATIENTS WITH BREATHLESSNESS

Where do PFTs fit in?

- Patients present with a symptom or complaint (presentation)
- Physicians collect and distill information (history, physical exam) to derive a differential diagnosis
- Ancillary tests (imaging, biochemistry, etc.) chosen to refine differential and arrive at a likely diagnosis
 - PFTs provide additional info that requires context

Why Perform PFTs?

- Both Edna and Giles present with the common symptom of **BREATHLESSNESS**
- Provide information that **define patterns of disease**: obstructive vs restrictive
- PFTs can **quantify severity, or track change over time** (progression, treatment response)

Review of Year 1

- PFT Lab last year:
 - Performed basic spirometry
 - Simulated situations of increased airways resistance (i.e. asthma) and decreased compliance (i.e. fibrosis)
 - Applied a simplified approach to interpretation of spirometry

Pulmonary Function Tests

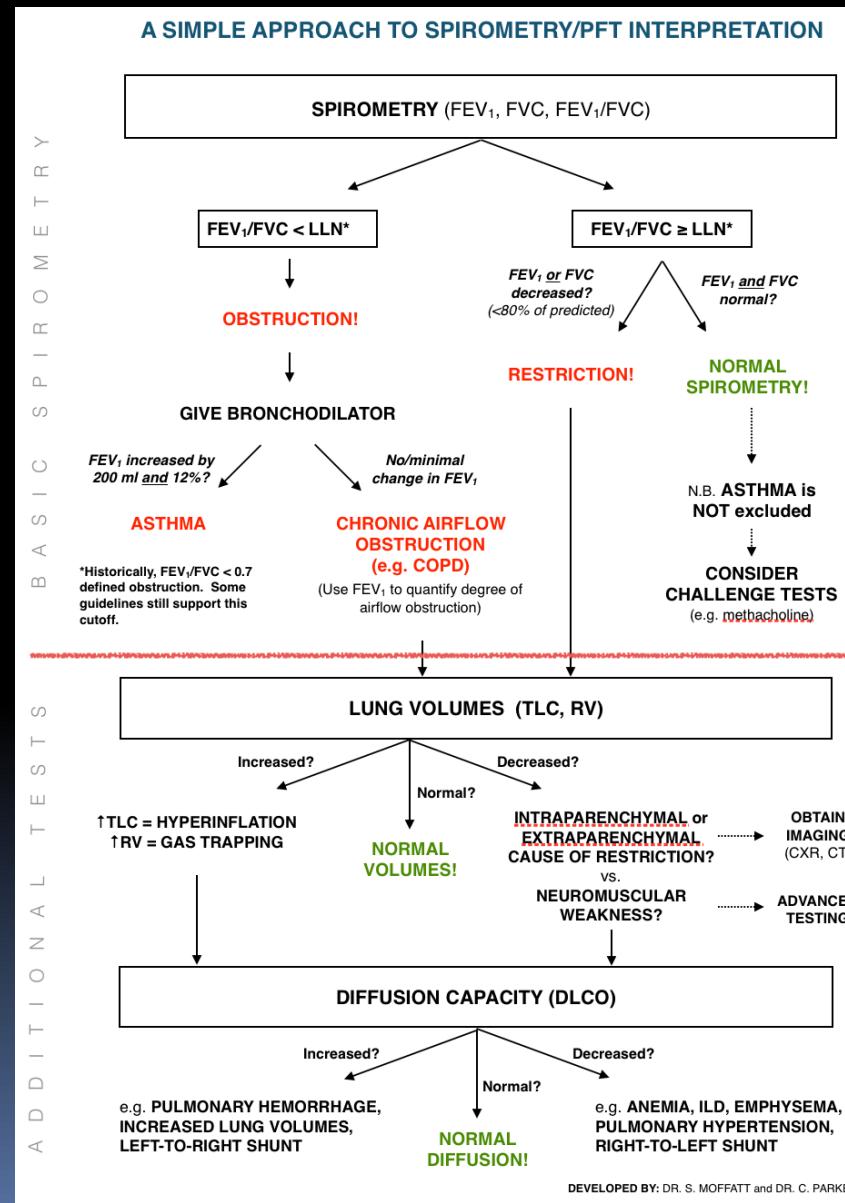
- In addition to basic spirometry, complete PFTs can:
 - Measure lung volumes
 - Measure diffusion capacity
 - How well does the lung allow gas exchange?
- Can also assess if spirometric airflow obstruction (if present) is reversible or not

Pulmonary Function Tests

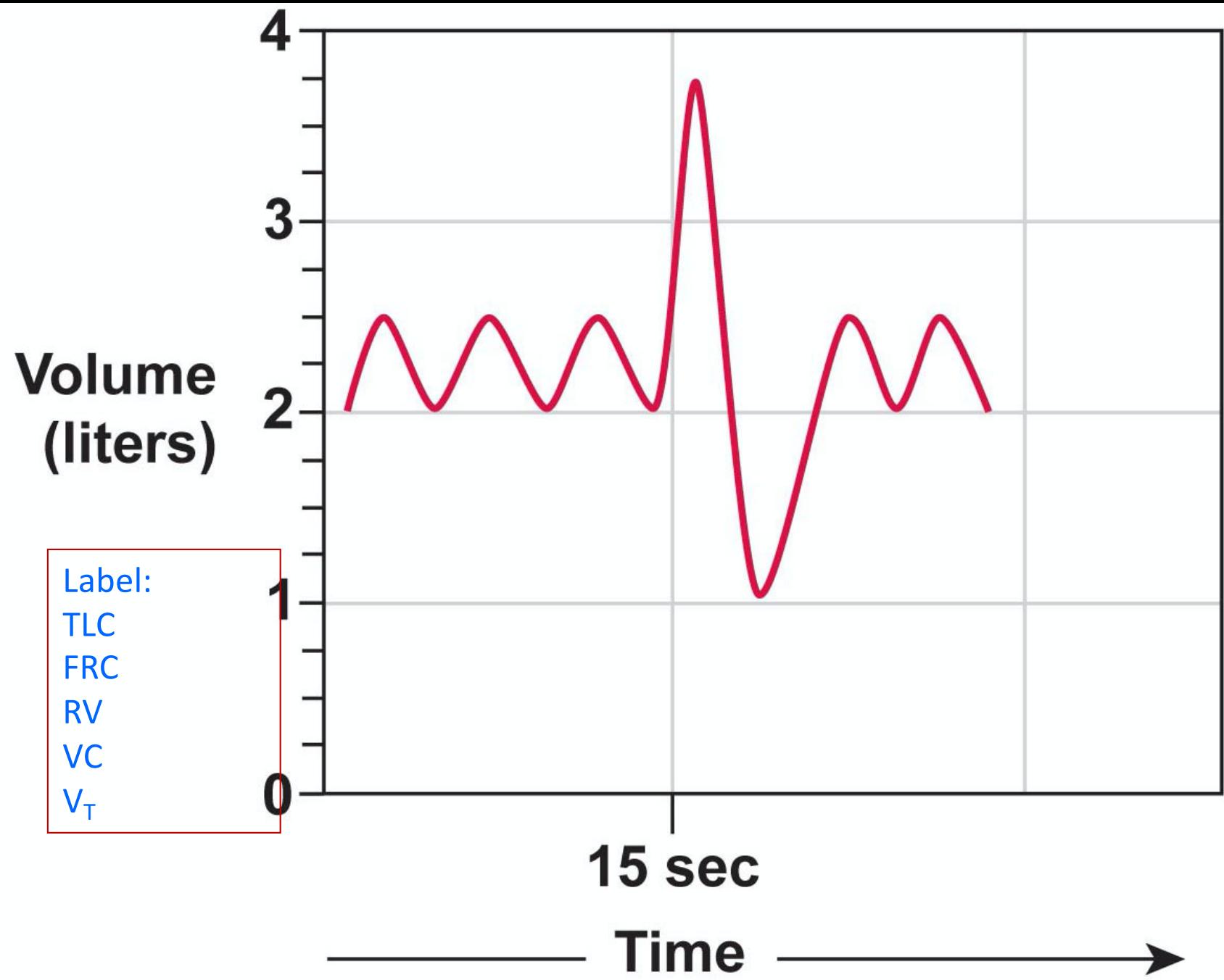
- Basic spirometers are widely available
- Complete PFTs require specialized equipment
 - The “body box” or plethysmograph
 - Carbon Monoxide to assess diffusion



Extending the Algorithm



Questions 17-1



Flow Volume Loop

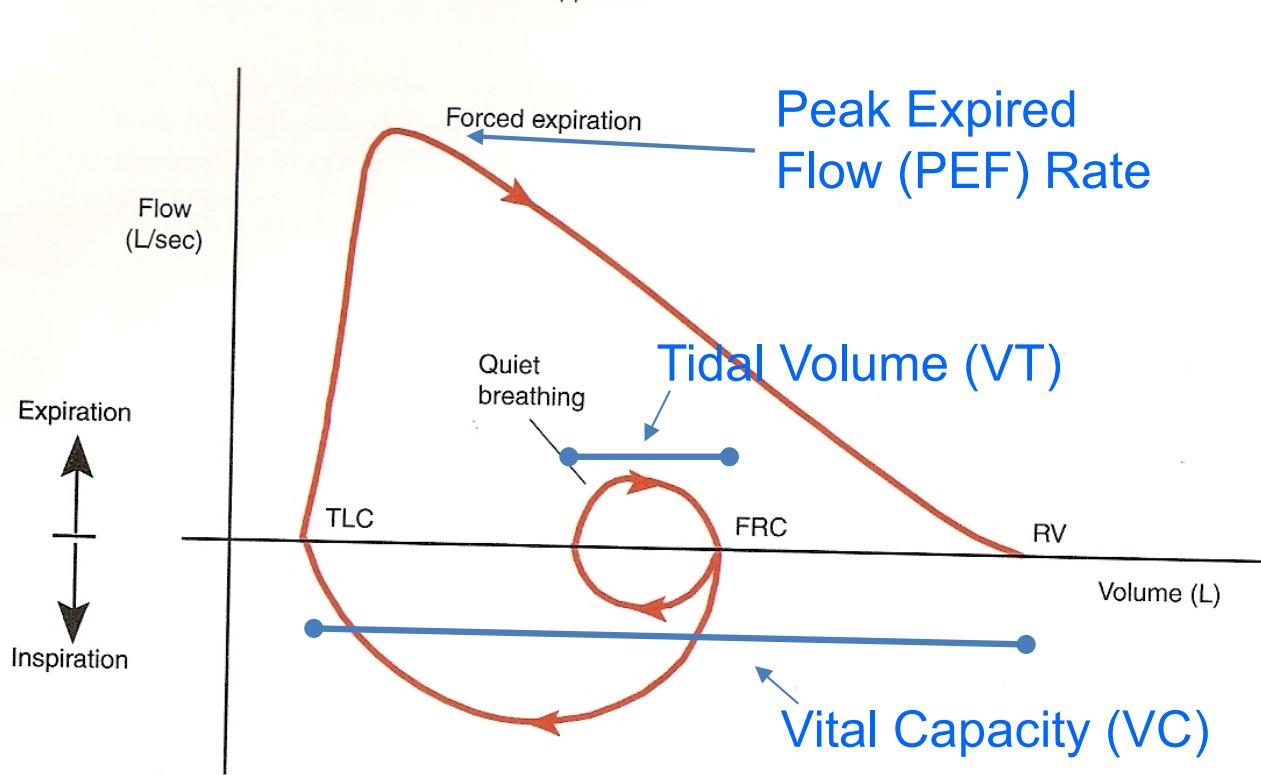
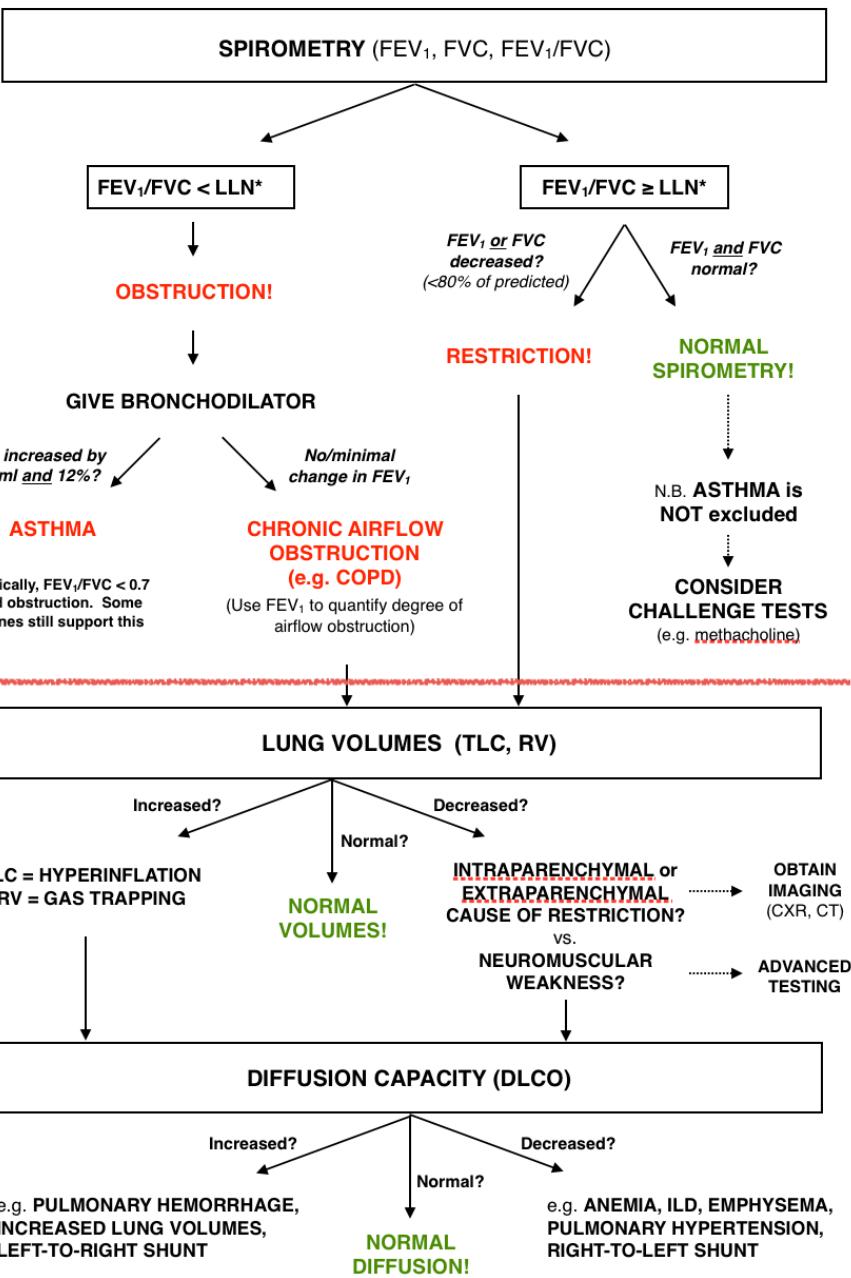


Image from Schwartzstein and Parker:
Respiratory Physiology Chapter 4

Notice the lung volumes and flows as a healthy person takes a normal quiet breath, then a deep inspiration and maximal expiration.



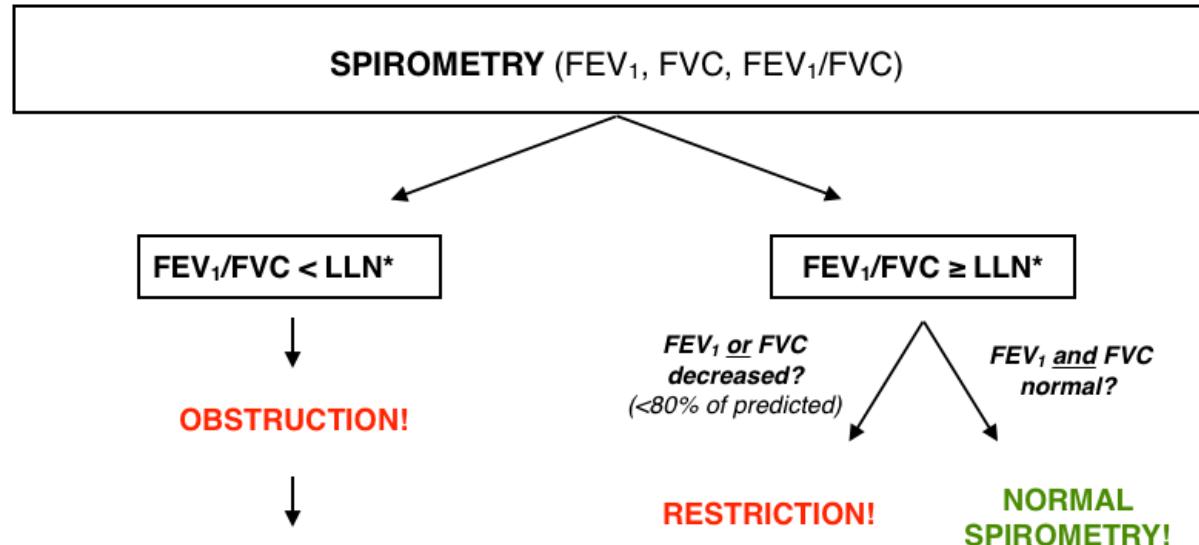
STEP 1:
EXAMINE THE FEV₁/FVC RATIO.

STEP 2:
IF OBSTRUCTION IS PRESENT,
IS IT REVERSIBLE?

STEP 3:
EXAMINE THE LUNG VOLUMES.

STEP 4:
ASSESS FOR DIFFUSION PROBLEMS.

STEP 1: EXAMINE THE FEV₁/FVC RATIO.



PURPOSE: Is there evidence of airflow obstruction or restricted lung volumes?

OBSTRUCTION

Best to use the “Lower Limit of Normal” (LLN) as this reflects the expected variation of biologic variables in a population.

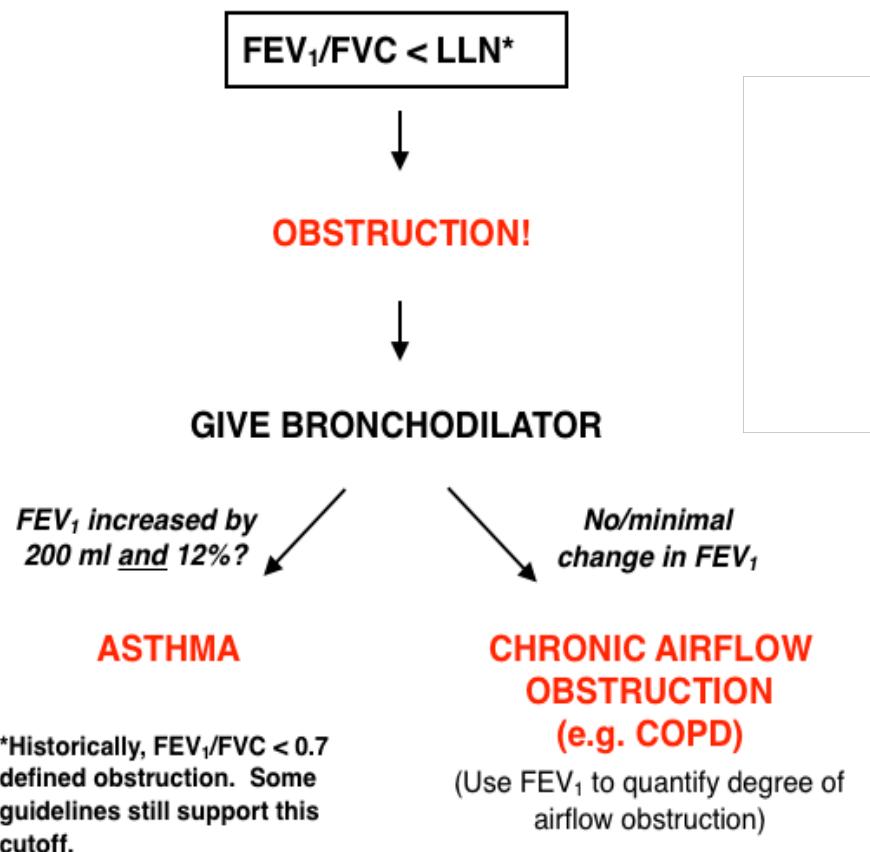
Historically, FEV₁/FVC < 0.7 has been used to define obstruction but this cutoff largely reflects studies of middle-aged Caucasian males. Some guidelines still reference this cutoff.

RESTRICTION

Reflects reduced lung volumes. Recall that both FEV₁ and FVC are measures of volume, not flow.

For simplicity, we will consider lung volumes that are less than 80% of their predicted values to be reduced. However, should also consider the Lower Limit of Normal when interpreting these volumes.

STEP 2: IF OBSTRUCTION IS PRESENT, IS IT REVERSIBLE?



PURPOSE: Characterize airflow obstruction as being **reversible** (e.g. asthma) **or not** (e.g. COPD and others)

DEFINITION OF REVERSIBILITY

According to the Canadian Thoracic Society and others, a post-bronchodilator change in FEV₁ of 200 mL and a 12% change from baseline indicates airflow reversibility.

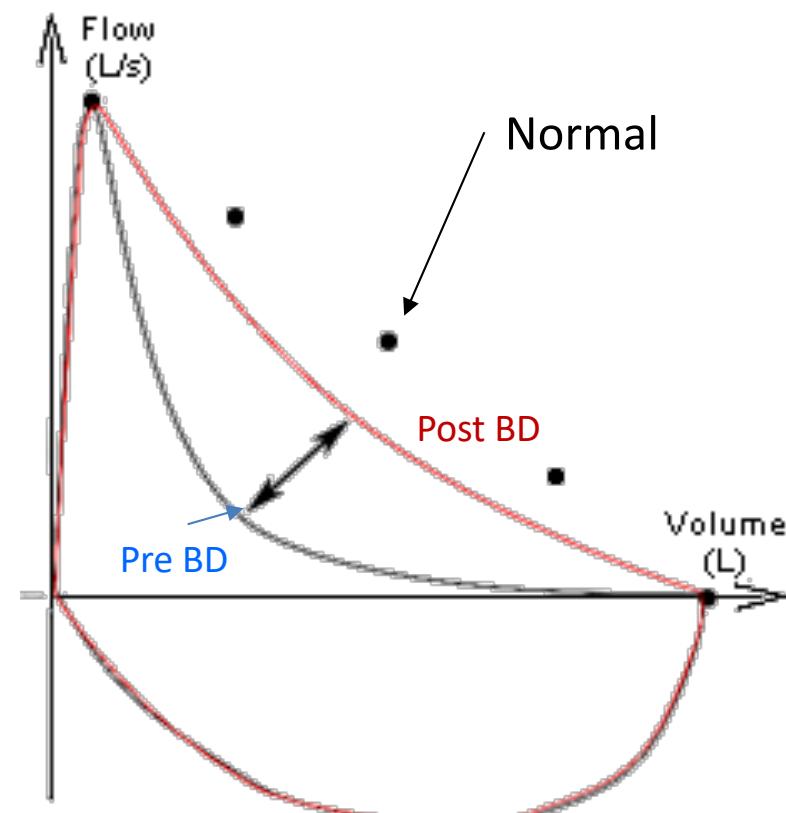
The significance of a change in FEV₁ with bronchodilator in the absence of baseline airflow obstruction is uncertain.

Reversibility with Bronchodilator

The increase in FEV₁ after inhaling a bronchodilator is used to identify reversible airflow obstruction.

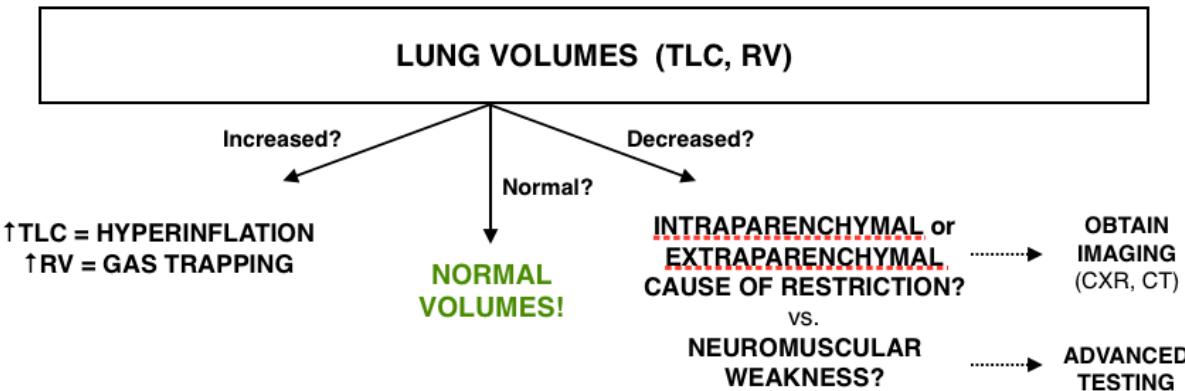
On a flow-volume loop this is seen as “less scooping”.

NB: **Persisting airflow obstruction** (ie $FEV_1/FVC < LLN$) after inhaling a bronchodilator is suggestive of COPD



ASTHMA AND COPD CAN OVERLAP!

STEP 3: EXAMINE THE LUNG VOLUMES.



PURPOSE: Is there evidence of abnormally increased or decreased lung volumes?

INCREASED LUNG VOLUMES

Best to use the “Upper Limit of Normal” (ULN) as this reflects the expected variation of biologic variables in a population.

Increases in Total Lung Capacity (TLC) indicate hyperinflation and larger than expected lungs.

Increases in Residual Volume (RV) indicate a difficulty with lung deflation suggestive of gas trapping, and is usually seen with airflow obstruction.

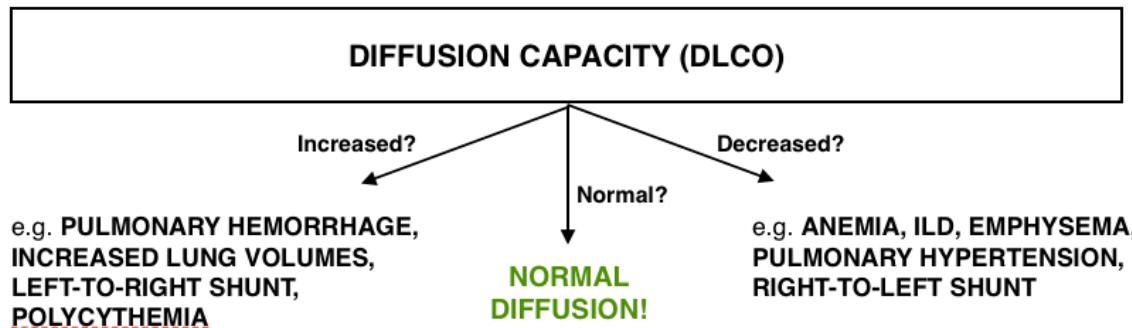
DECREASED LUNG VOLUMES

Best to use the “Lower Limit of Normal” (LLN) as this reflects the expected variation of biologic variables in a population.

Consider intraparenchymal causes (e.g. ILD) and extraparenchymal (e.g. pleural space, chest wall) of reduced lung volumes. Imaging can help.

Neuromuscular weakness can also reduce lung volumes. Consider additional testing to assess.

STEP 4: ASSESS FOR DIFFUSION PROBLEMS.



PURPOSE: Diffusing Capacity for Carbon Monoxide (DL_{CO}) is a test of body's ability to allow gas exchange across the alveoli into the blood in the pulmonary capillaries.

INCREASED DIFFUSING CAPACITY

Carbon monoxide in the body is transported almost exclusively bound to hemoglobin, and is therefore an indirect test of oxygen carrying capacity.

Increases in red blood cell mass (e.g. polycythemia) or pulmonary blood flow (left to right shunt) can increase DL_{CO} .

DECREASED DIFFUSING CAPACITY

Can be seen with reduced lung volumes (e.g. ILD), decreased pulmonary capillary surface area (e.g. emphysema), decreased pulmonary blood flow (e.g. right-to-left shunt), severe VQ mismatch (e.g. pulmonary vascular disease) or decreased oxygen carrying capacity (e.g. anemia).

DL_{CO} can be corrected for lung volumes (DL_{CO}/V_A) to determine if reduced DL_{CO} is only due to decreased volumes (i.e. DL_{CO}/V_A should normalize if this is the case).

Pulmonary Function Report

Respiratory Therapist: Emily RRT
Diagnosis: ?Asthma

Age: 73 Height(in): 63.0 Weight(lb): 144 Body Mass Index: 25.59 Gender: Female Race: Caucasian Temp: 23 PBar: 765

		Ref	Pre	% Ref	Post	% Ref	% Chg
Spirometry							
FVC	Liters	2.64	(1.8 - 3.5)	2.32	88		
FEV1	Liters	1.85	(1.1 - 2.6)	1.84	99		
FEV1/FVC %		71	(59.9 - 82.4)	79			
FEF25-75% L/sec		2.14	(0.7 - 3.6)	1.55	72		
FEF50% L/sec		2.62	(1.3 - 4.0)	2.06	79		
FEF75% L/sec		0.64	(0.2 - 1.1)	0.74	115		
PEF L/sec		5.28	(2.4 - 8.1)	7.40	140		
FVC	Liters	2.64	(1.6 - 3.6)	2.07	78		
FEF/FIF50				0.57			

If performed,
Post-broncho-
dilator results
appear here,
with % change

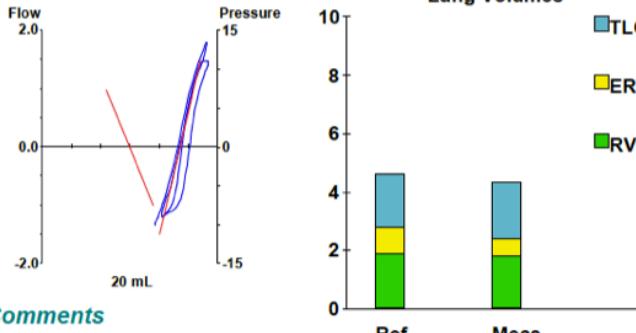
← Patient demographics and indication for test

Reference value and RANGE OF NORMAL (LLN and ULN)

		Ref	Pre	% Ref	Post	% Ref	% Chg
Lung Volumes							
TLC	Liters	4.59	(3.8 - 5.4)	4.34	95		
VC	Liters	2.64	(1.6 - 3.6)	2.55	97		
IC	Liters	1.77	(1.3 - 2.2)	1.99	112		
FRC PL	Liters	2.66	(1.7 - 3.6)	2.35	89		
ERV	Liters	0.89	(0.7 - 1.1)	(0.58)	(65)		
RV	Liters	1.88	(1.2 - 2.6)	1.79	95		
RV/TLC %		41	(29.3 - 52.8)	41			
Vtg	Liters			2.54			

		Ref	Pre	% Ref	Post	% Ref	% Chg
Diffusing Capacity							
DLCO	ml/mmHg/min	18.0	(9.7 - 26.3)	14.0	78		
DLCO/VA	ml/mmHg/min/L	3.59	(1.7 - 5.4)	3.91	109		
VA	Liters			3.58			
IVC	Liters			2.22			

		Ref	Pre	% Ref	Post	% Ref	% Chg
Resistance Raw	cmH2O/L/sec	1.60	(0.5 - 2.7)	(3.23)	(202)		

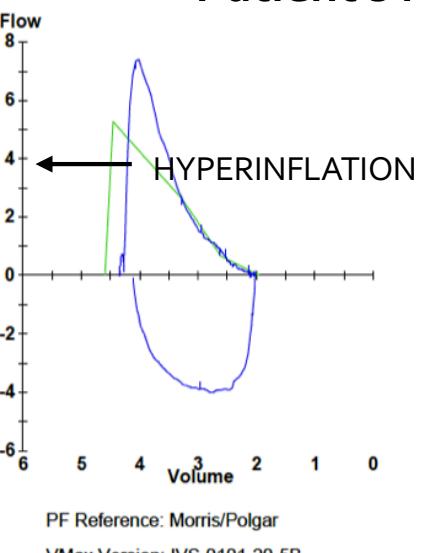


Comments

Resp meds include ventolin, last taken 2 weeks ago. Taken med for acid reflux. NKDA. Seasonal allergies. Ex smoker having quit 35 years ago. Smoked 20 years at 1 ppd. Second hand smoke exposure in the home as a child and from spouse x 20 years. Best PEF not reproducible, Otherwise all tests performed meet ATS criteria of acceptability and repeatability.

Baseline expiratory flows are within normal limits and there is no evidence of airflow obstruction. There is no significant change in FEV1 post-bronchodilator. Lung volumes and diffusion are within normal limits with the exception of a reduced ERV of uncertain significance. Please note that asthma cannot be confirmed or excluded on the basis of this test, and methacholine challenge could be considered.

← Patient's results (and expressed as % predicted)

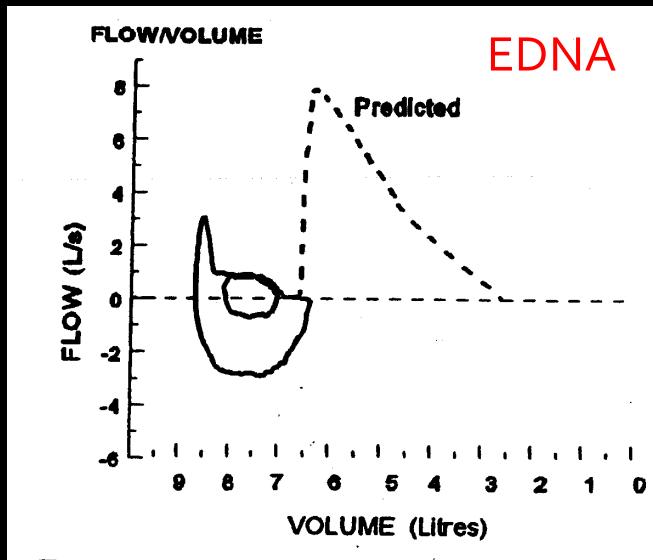


Flow-Volume loop shows the expected results (in green) and the patient's results (in blue). If done, post-bronchodilator spirometry will also be superimposed, in red.

← LOTS OF GOOD INFO HERE!

C.M. Parker MD MSc FRCPC FCCP

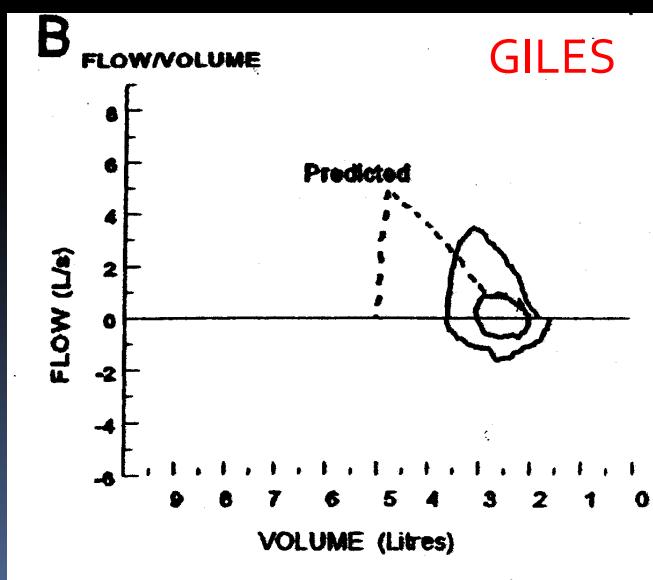
EDNA AND GILES DO PFTs:



FEV₁ = 45% predicted
FVC = 85% predicted
FEV₁/FVC = 0.48

TLC = 148% predicted
RV = 200% predicted

D_LCO = 68% predicted

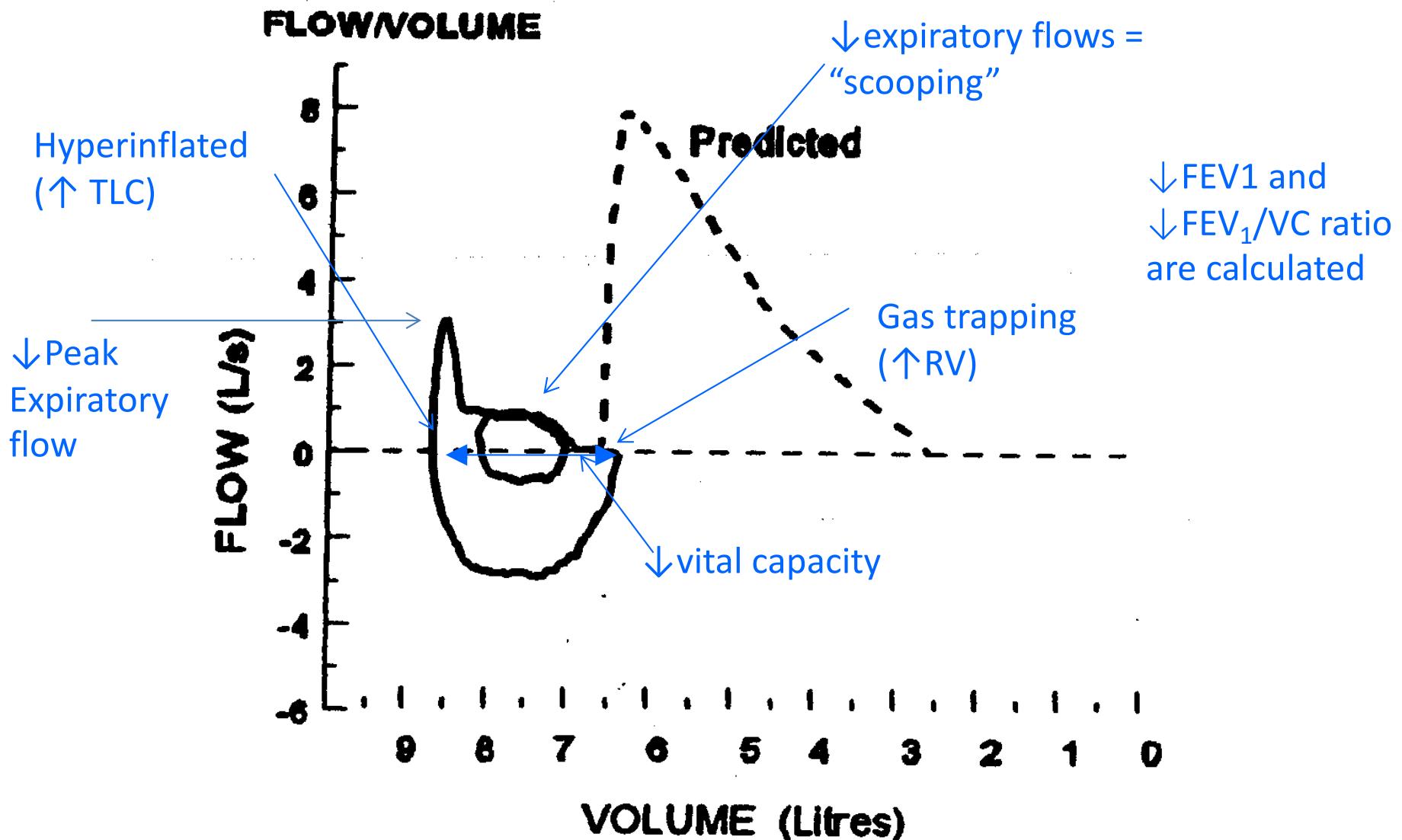


FEV₁ = 45% predicted
FVC = 52% predicted
FEV₁/FVC = 0.86

TLC = 73% predicted
RV = 76% predicted

D_LCO = 74% predicted

Edna's Obstructive PFT



Edna's Obstructive PFT Pattern

Condition	FVC	FEV1	FEV1/VC	TLC	RV
Edna (COPD)	↓	↓	↓↓	↑	↑

- ↓ ↓ FEV1/FVC due to increased airway resistance
- ↓ FVC and FEV₁ due to reduced lung recoil and increased airway resistance
- ↑ TLC and RV due to gas trapping from reduced lung recoil
- (Bonus: ↓diffusing capacity due to loss of alveolar-capillary surface area in emphysema.)

Edna's Obstructive PFT Pattern

GRADING SEVERITY OF OBSTRUCTION USING FEV₁:

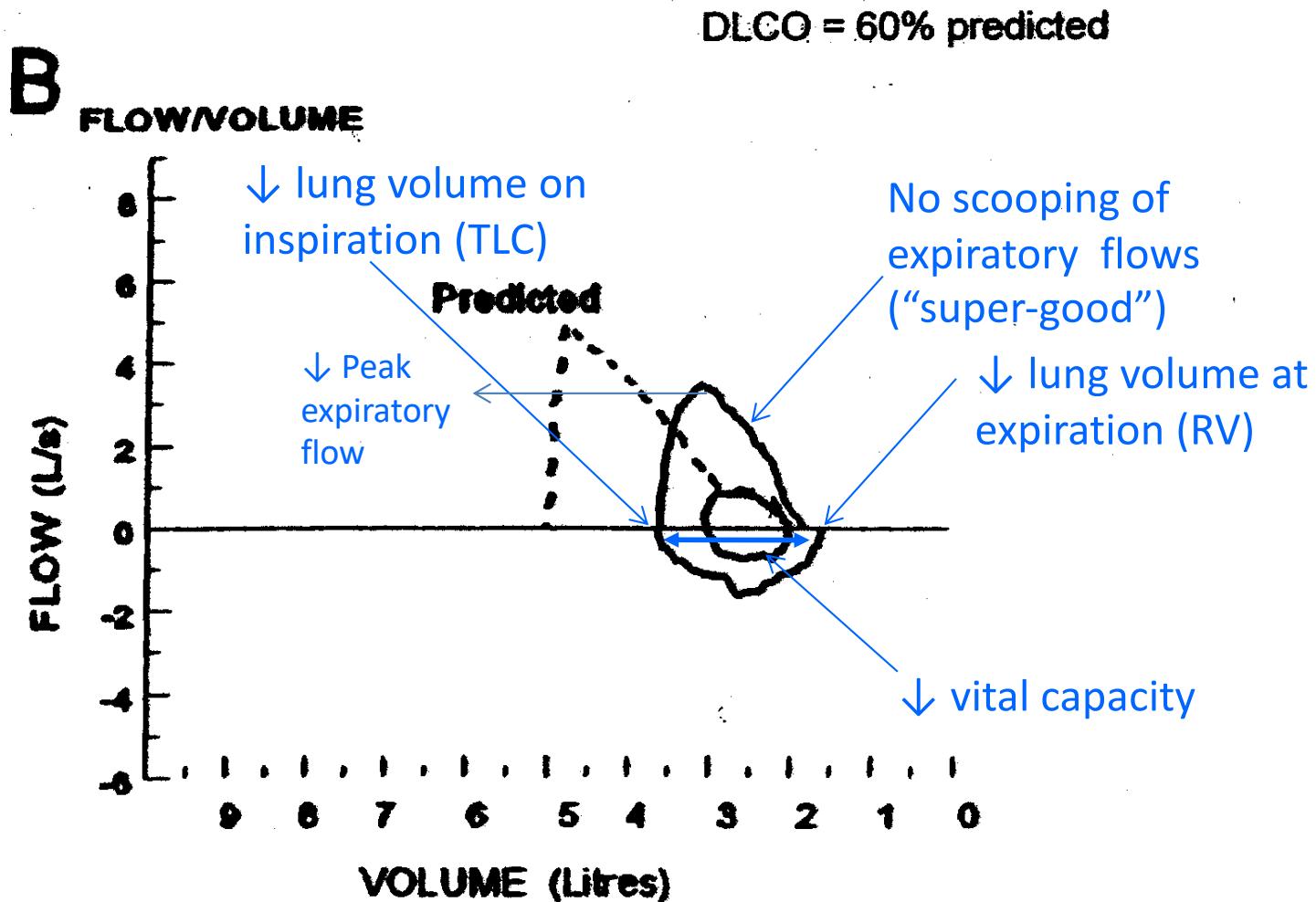
Table 2.4. Classification of airflow limitation severity in COPD (Based on post-bronchodilator FEV₁)

In patients with FEV₁/FVC < 0.70:

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

- Global Initiative for Chronic Obstructive Lung Disease, 2018

Giles' Restrictive PFT



Giles' Restrictive PFT Pattern

Condition	FVC	FEV1	FEV1/VC	TLC	RV
Giles	↓	↓	↑	↓	↓

- ↑ FEV1/FVC due to decreased airway resistance
- ↓ FVC and ↓ FEV₁ due to decreased compliance
- ↓ RV and ↓ TLC due to decreased compliance
- (And: reduced gas transfer)

Looking Ahead...

- Will plan to post some practice PFTs later this week
 - Look for OPTIONAL DIL session on Sept 23



Questions?





You on the Elliptical

The class starts with everyone warming up on the elliptical for 10 minutes.

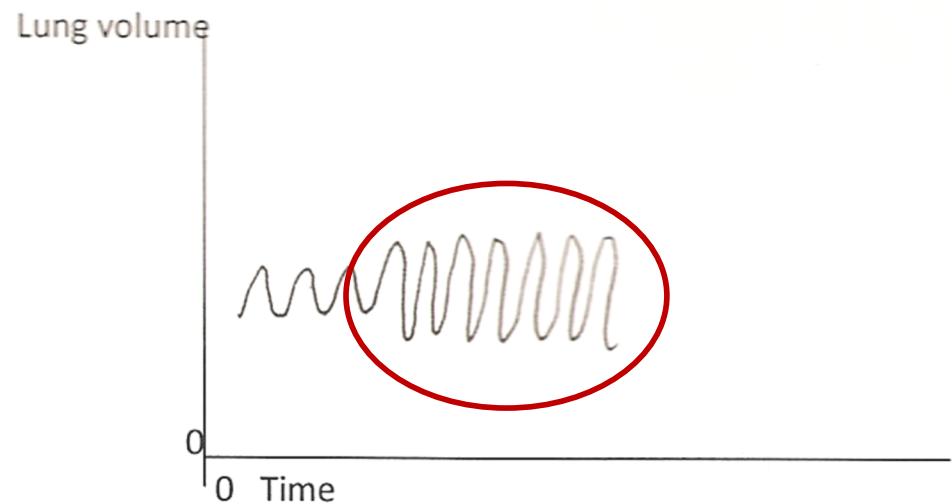
How does your tidal volume and respiratory rate change to enable you to exercise?

How does this parallel your cardiac response to exercise?

What is the respiratory equivalent to “cardiac output?”

You on the Elliptical

- ↑ tidal volume first then ↑ RR
- Minute ventilation increases: $VE = VT \times RR = L/min$
- This parallels increase in cardiac output: $CO = SV \times HR = L/min$
- ↑ VE and ↑ CO match ↑ metabolic demands



Edna on the Elliptical

- After a few minutes of walking on the elliptical Edna says she feels she can't get enough air and will have to stop. You notice that her respiratory rate is $> 25/\text{min}$. Her oxygen saturation is $> 90\%$ on room air when she checks it with her personal oximeter.



Edna on the Elliptical

Why is she short of breath if her saturation is still safe?

Exercise



Slide courtesy of Dr. C. Parker

Edna on the Elliptical

What could she do to improve her ability to exercise and reduce her breathlessness?

Would additional inhaled oxygen be helpful?

Edna on the Elliptical

What could she do to improve her ability to exercise and reduce her breathlessness?

Bronchodilators before exercise, pace herself, pursed lip breathing



Would additional inhaled oxygen be helpful?

Not if her saturation is > 90%

Giles on the Elliptical

- Within a few minutes of being on the elliptical Giles has a respiratory rate of 34 breaths per minute compared to yours of 26. He has to stop because he is too breathless .
- You notice that his SpO₂ has fallen from 90% at rest to 82% as he tried to exercise.

Giles

Why does his RR increase so much more than yours when he exercises?

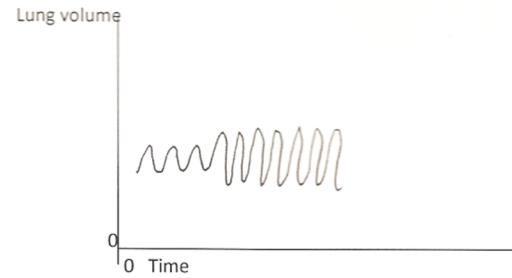
Why does his oxygen saturation fall when he exercises?

What can he do to improve his ability to exert himself and reduce his breathlessness? Would a bronchodilator help?

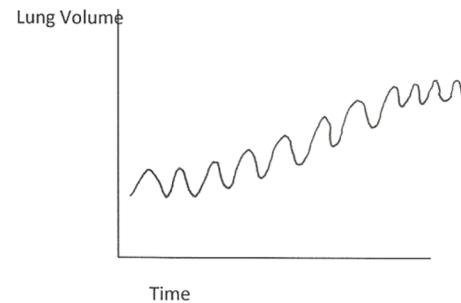
Giles on the Elliptical

- ↓ lung compliance → ↑ work to ↑ tidal volume
- At rest: ↑ RR to maintain minute ventilation
- Exercise requires ↑↑ RR because he cannot ↑ tidal volume
- → rapid shallow breathing (“panting”)
- Very breathless and uncomfortable
- cannot match metabolic demand and must stop

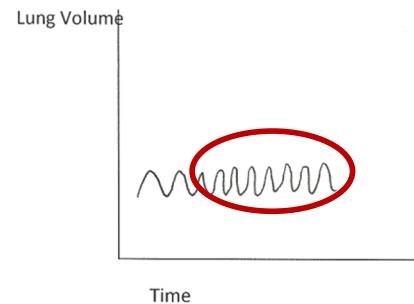
You

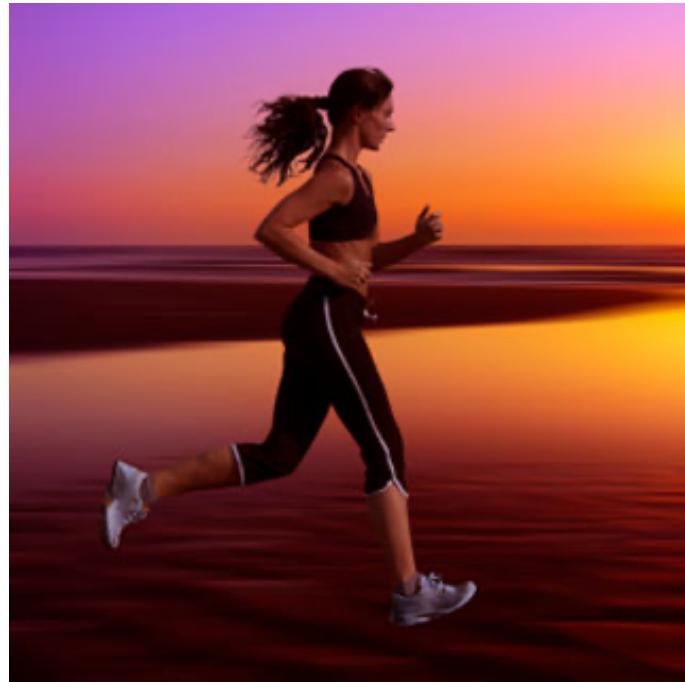


Edna



Giles





Work of Breathing

Work of Breathing

Normal breathing with healthy lungs is very efficient.
Breathing with abnormal compliance and resistance
requires ↑↑ respiratory muscle work.



The awareness of this **increased respiratory effort** is the cause of **dyspnea**. It does not require ↓PaO₂ or ↑Pa CO₂

Work of Breathing

- During exertion, metabolic rate increases, and ventilation needs to increase to match it
- Persons with abnormal lung mechanics limit their exertion because the respiratory work to increase ventilation is too difficult



Dyspnea Determines Function

Modified Medical Research Council (mMRC) Dyspnea Scale

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

Stenton C. *Occup Med (Lond)*. 2008;58:226-227.



We will be able to



- Explain the clinical presentations of patients with obstructive and restrictive lung disease based on their lung mechanics
- Explain the mechanism and consequence of dynamic hyperinflation and gas trapping in obstructive disease
- Predict the response to exercise in persons with abnormal lung mechanics and explain how their dyspnea can be reduced
- Diagnose obstructive and restrictive lung disease on pulmonary function testing