AIRWAY RESISTANCE & LUNG COMPLIANCE

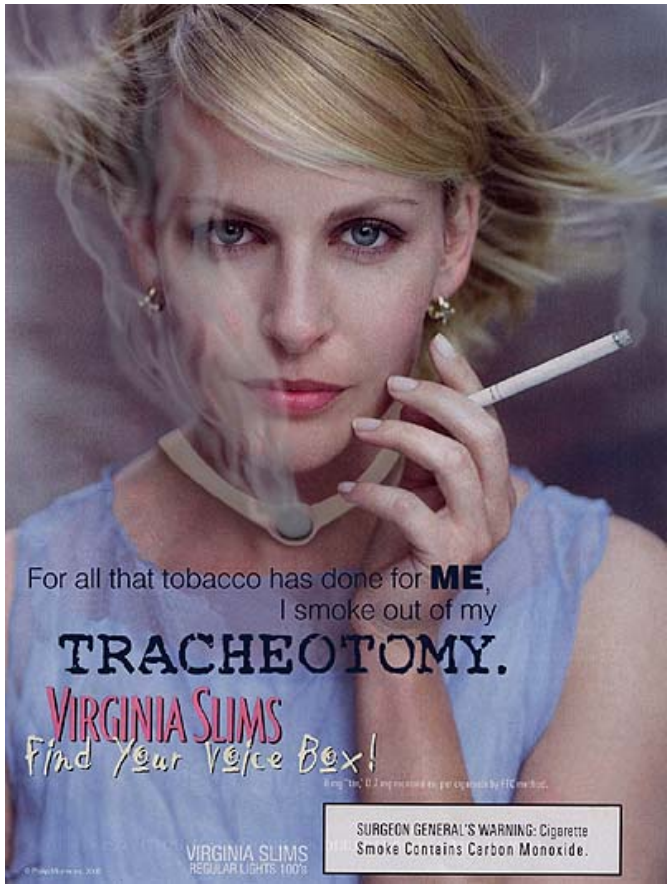
KEY FACTORS IN MOVEMENT OF AIR IN & OUT OF THE LUNGS

Objectives

1. List the key source of resistance to movement of air in and out of the lungs. Compare the contribution of the upper airways and tracheobronchial tree to airway resistance. Explain why airway resistance is least in the smaller airways in healthy individuals.
2. List the causes of increased airway resistance in obstructive lung diseases.
3. Describe the role of the adrenergic and cholinergic receptors in regulation of airway smooth muscle, airway caliber, and airway resistance.
4. Define compliance and elastance and identify 3 factors that affects them.
5. Define the static compliance of the lungs. Compare the characteristics of the static pressure-volume curve in health individuals to patients with emphysema and pulmonary fibrosis.
6. Describe the role of pulmonary surfactant in 1) determining alveolar surface tension, 2) the compliance of the lungs 3) neonatal respiratory distress syndrome.

THE CASE OF VIGINIA SLIM

Virginia Slim is a 57 years old chronic smoker diagnosed with severe chronic obstructive lung disease (COPD). She is shortness of breath and coughs. Both signs have worsened in the past 2 years.

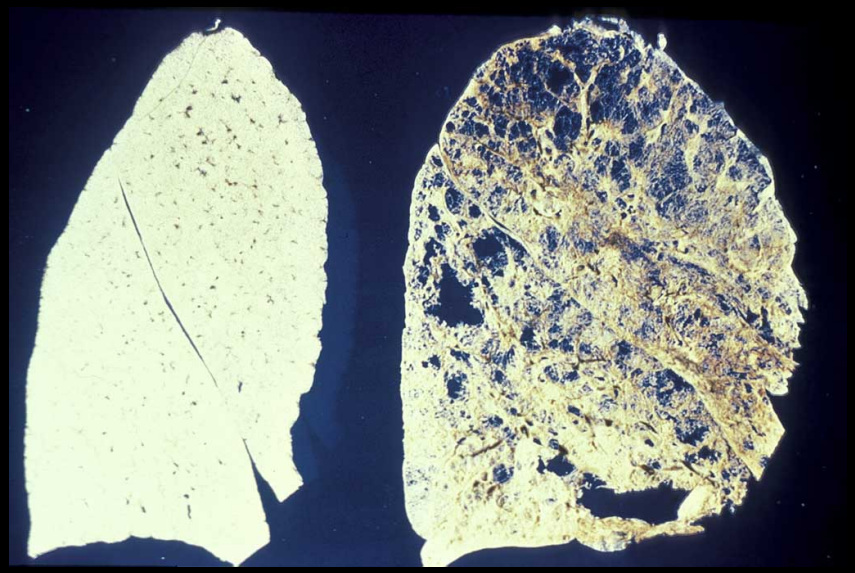


5 years ago she:

- quit smoking
- follows careful course of inhaled bronchodilator therapy
- has regular physiotherapy for pulmonary rehabilitation
- has had several courses of antibiotics for acute bacterial infections.

Despite these efforts she lives a chair to bed existence and has to use supplemental home oxygen. She is in the hospital waiting for a decision by the thoracic surgical team on whether she is a suitable candidate for lung volume resection surgery to remove some of her emphysematous lung.

A CASE OF HIGH RESISTANCE & COMPLIANCE
The Smoker's Lungs
inflammed, narrowed airways & destroyed alveolar walls

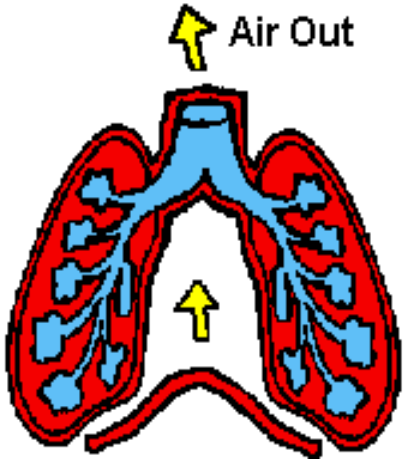


Post Mortem Lung Sections
non-smoke smoker

AIRWAYS RESISTANCE

A KEY FACTOR IN MOVEMENT OF AIR IN & OUT OF THE LUNGS

The total resistance to flow of air in the airways (R_{aw}) is very little- approximately 250 fold less than that encountered generating the same airflow through a smokers pipe.



2 cm H_20/L/sec

500 cm H_20/L/sec

AIRWAYS RESISTANCE

A KEY FACTOR IN MOVEMENT OF AIR IN & OUT OF THE LUNGS

Two Resistive Forces

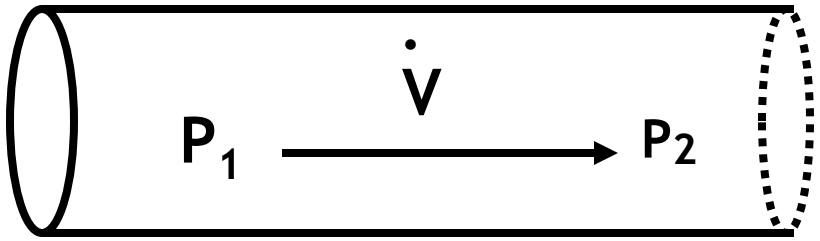
1. Inertia of the respiratory system (negligible)

2. Friction
   i) lung & chest wall tissue surfaces gliding past each other
   ii) airways past each other during expansion
   iii) frictional resistance to flow of air through the airways
   represents 80% of total airways resistance

   - Upper airways (UAW) 60%
   - Tracheobronchial tree 40%

AN IMPORTANT RELATIONSHIP
BETWEEN FLOW, DRIVING PRESSURE & RESISTANCE
"resistance exists where there is flow"

$\text{Flow } (\dot{V}) = \Delta P / R$

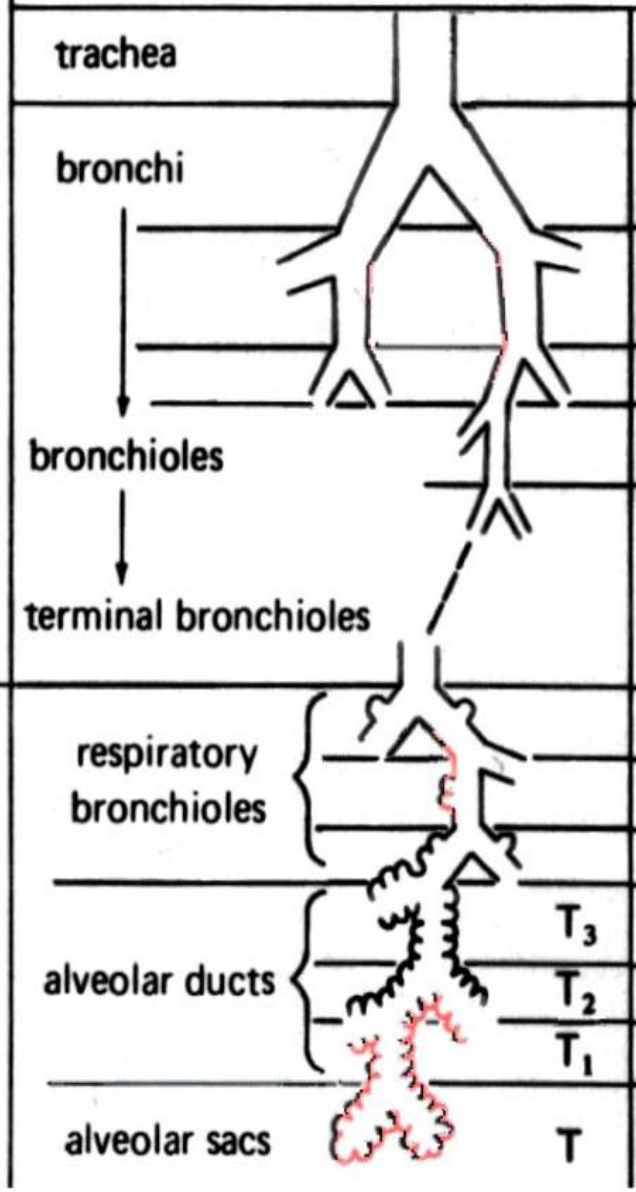


$R \propto \Delta P / \dot{V}$ units = cm H_20/L/sec

$R_{aw} = P_A - P_B / \dot{V} =$ 1.0 cm H_20/ 0.5 L/sec = 2 cm H_20/L/sec

at peak flow during quiet expiration

$\Delta P = P_1 - P_2$ = pressure gradient = driving pressure
recall that the direction of flow is from high to low pressure

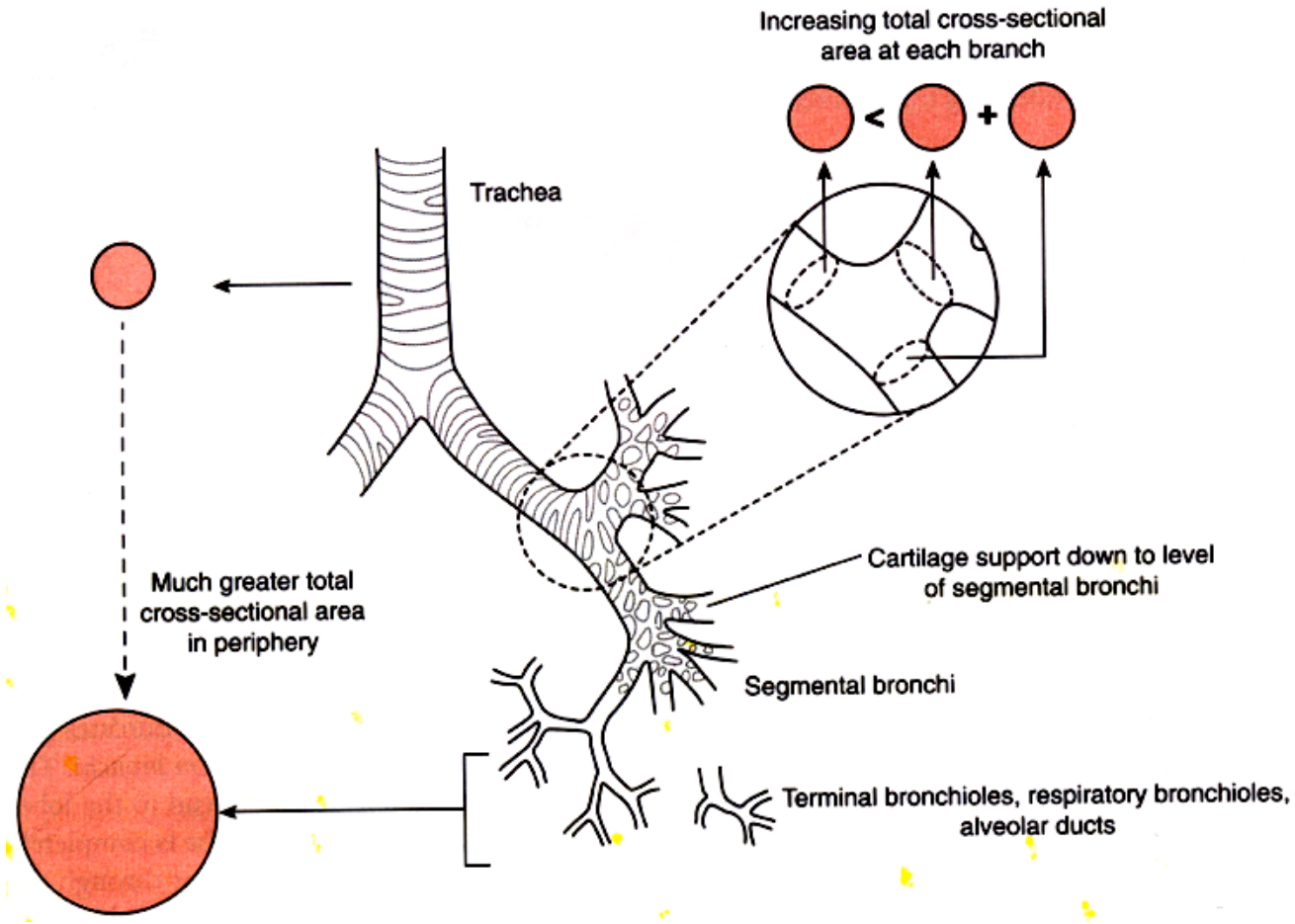


	Generation		Diameter, cm	Length, cm	Number	Total cross-sectional area, cm^2
conducting zone	trachea	0	1.80	12.0	1	2.54
	bronchi	1	1.22	4.8	2	2.33
		2	0.83	1.9	4	2.13
		3	0.56	0.8	8	2.00
	bronchioles	4	0.45	1.3	16	2.48
	terminal bronchioles	5 ↓ 16	0.35 ↓ 0.06	1.07 ↓ 0.17	32 ↓ 6×10^4	3.11 ↓ 180.0
transitional and respiratory zones	respiratory bronchioles	17	↓	↓	↓	↓
		18				
		19	0.05	0.10	5×10^5	10^3
	alveolar ducts	T_3 20	↓	↓	↓	↓
		T_2 21				
		T_1 22				
	alveolar sacs	T 23	0.04	0.05	8×10^6	10^4

AIRWAY BRANCHINGS

Parallel design ↓s frictional resistance to airflow

due to ↑ in total cross sectional area



AIRWAY BRANCHINGS

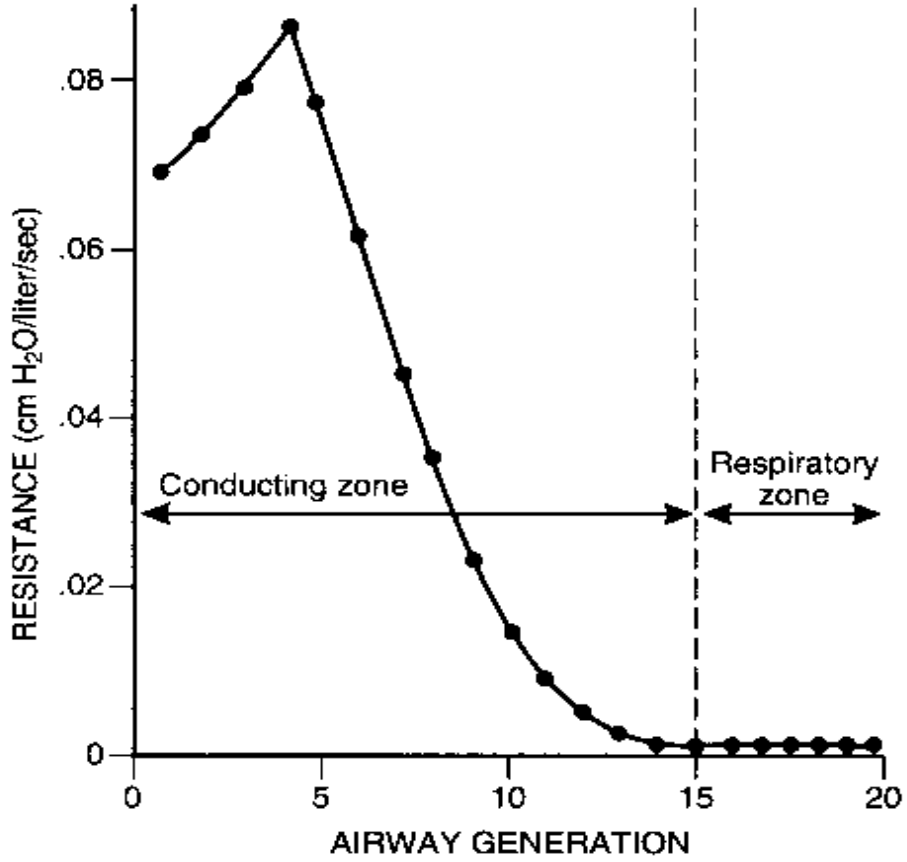
Parallel design ↓s frictional resistance to airflow

RESISTANCE α 1/RADIUS^4

In health the major contribution to airways resistance lies in the larger airways [generations 1-6]**.**

Respiratory bronchioles have small individual radii. Yet the parallel arrangement of these small airways results in a large total cross sectional area creating little resistance to air flow.

In airway disease caused by smoking the smaller airways are the major site of resistance to flow of air because of a reduction in their luminal size.



If FLOW = $\Delta P/R$ and $R \propto 1/r^4$ . . .

1. How much will airflow be affected if the radius of an airway is halved in caliber?

2. If the airway radius can not be changed, what can you change to increase air flow to the levels prior to the reduction in airway radius? By how much?

In disease, airway radius ↓s due to several mechanisms leading to EXPIRATORY AIRFLOW LIMITATION

Bronchoconstriction asthma/COPD

Inflammation asthma/chronic bronchitis/COPD/bronchiolitis

Excess Mucus Production asthma/chronic bronchitis/cystic fibrosis

Reduced Alveolar Elastic Recoil emphysema

reduced recoil means less tethering on neighboring airway which in turn will be reduced in airway size-see next slide]

In all cases, airway resistance ↑ & maximal expiratory flow ↓. These individuals have a hard time breathing out.

How come breathing in is not affected during inspiration?

The inward elastic recoil of airways plays 2 key roles

1) creates a driving pressure for expiration

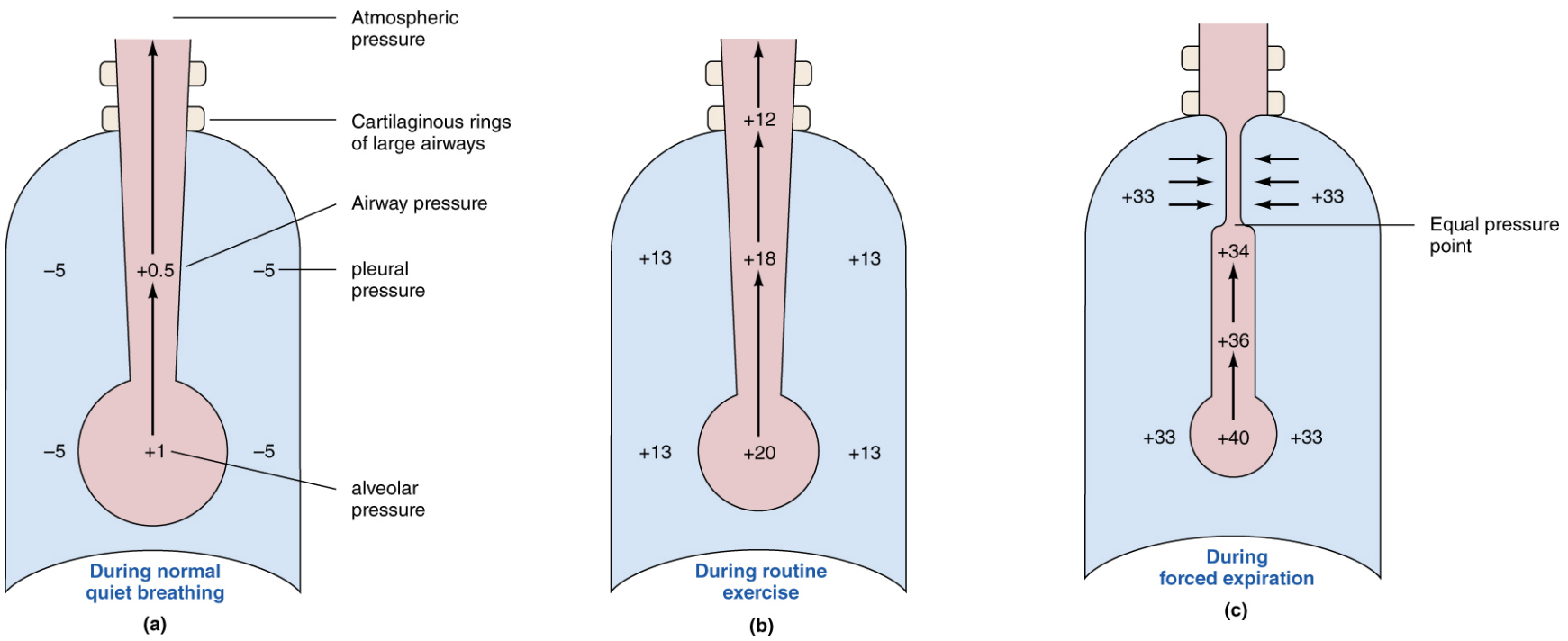
Airway transection during passive expiration

2) Passively increases neighboring airway size through outward traction

Airway in cross section.

EXPIRATION & AIRFLOW LIMITATION

AIRWAY PATENCY DEPENDS ON AIRWAY TRANSMURAL PRESSURE



During maximal forced expiration, airways reduce in size (develop an airflow limiting segment) distal to development of an equal pressure point. This is in contrast to expiration during normal quiet breathing or routine exercise.

In patients with increased airway resistance, increased airway compliance or both the flow limited segment may collapse and result in gas trapping.

Neural (active) Control of Airway Smooth Muscle

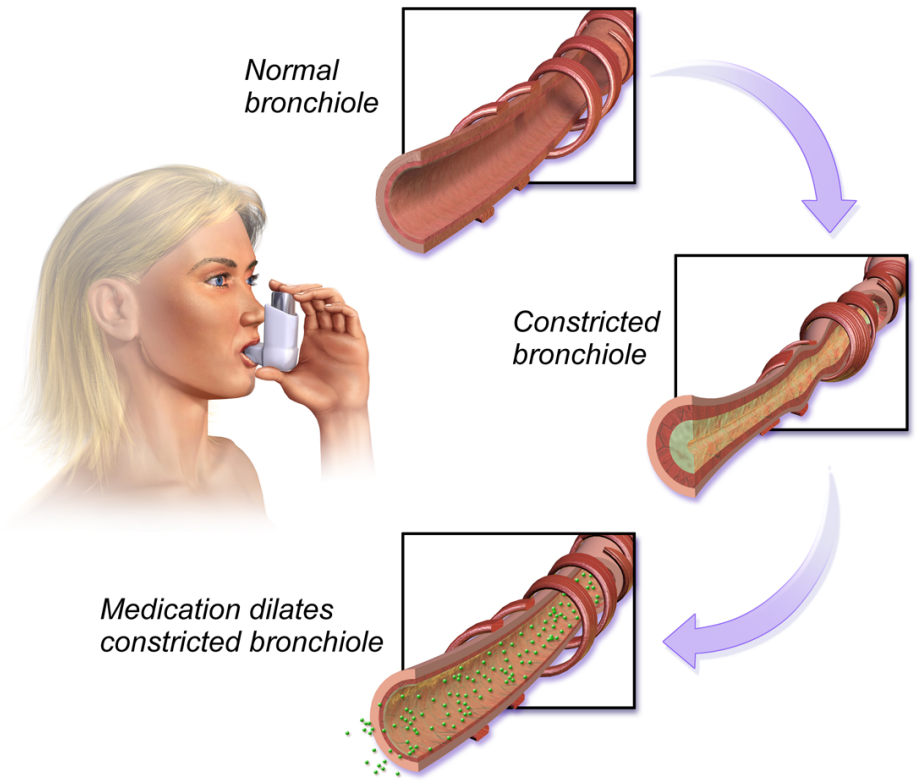
The Autonomic Nervous System

Parasympathetic:

Ach release (vagus nerve) ➔cholinergic receptors airway smooth muscle (muscarinic, M3) ➔**bronchoconstriction** [dominant control at rest regulating airway smooth muscle tone]

Sympathetic

Adrenaline release (adrenal gland medulla) ➔ circulating blood ➔ adrenergic receptors airway smooth muscle (β_2 receptors) ➔ **bronchodilation.** [sympathetic innervation of airway smooth muscle is sparse; released noradrenaline as neurotransmitter plays small role in bronchodilation is humans)



??? role of other parasympathetic neurotransmitters in regulation of airway caliber in pathology: asthma/allergic inflammatory reactions

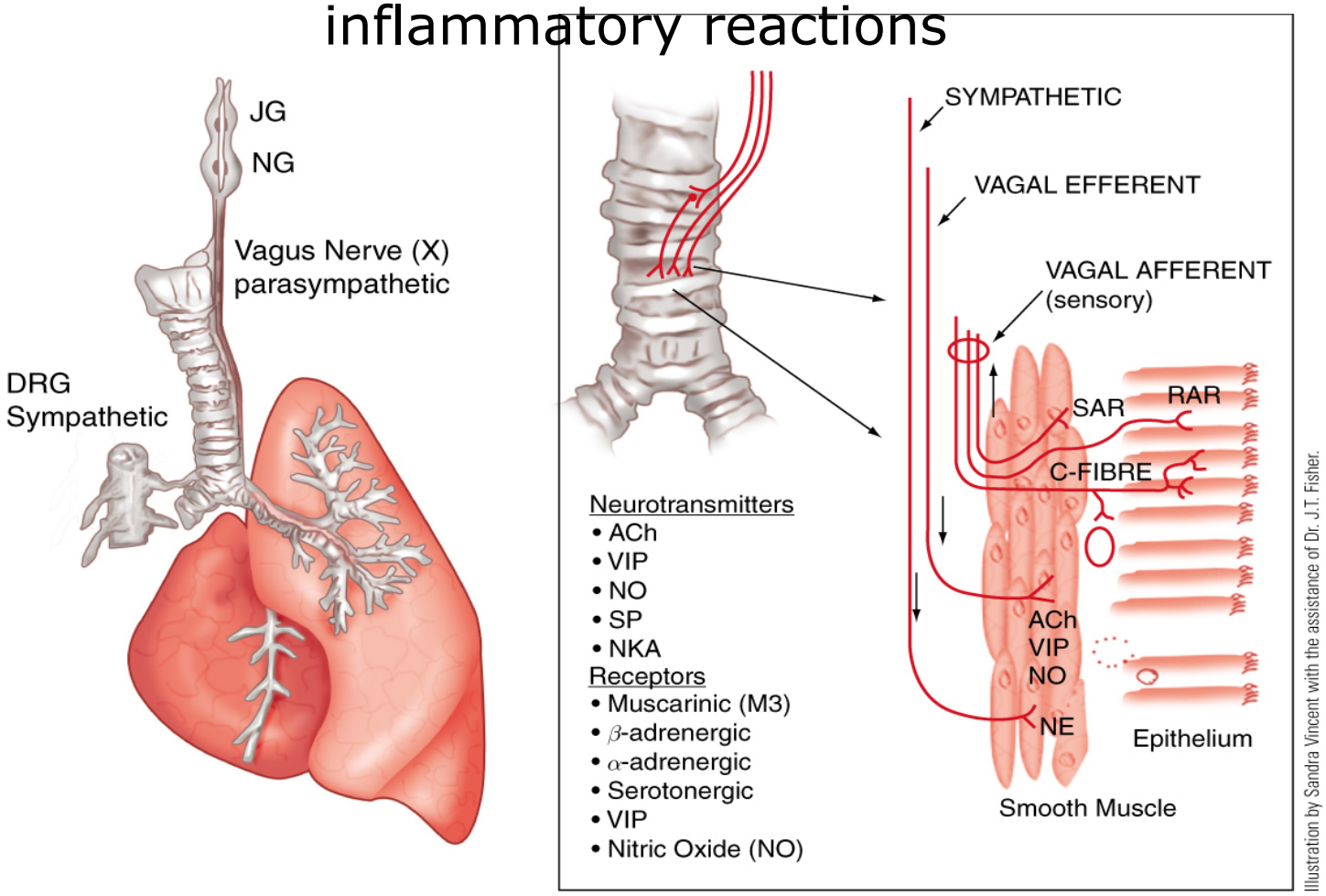


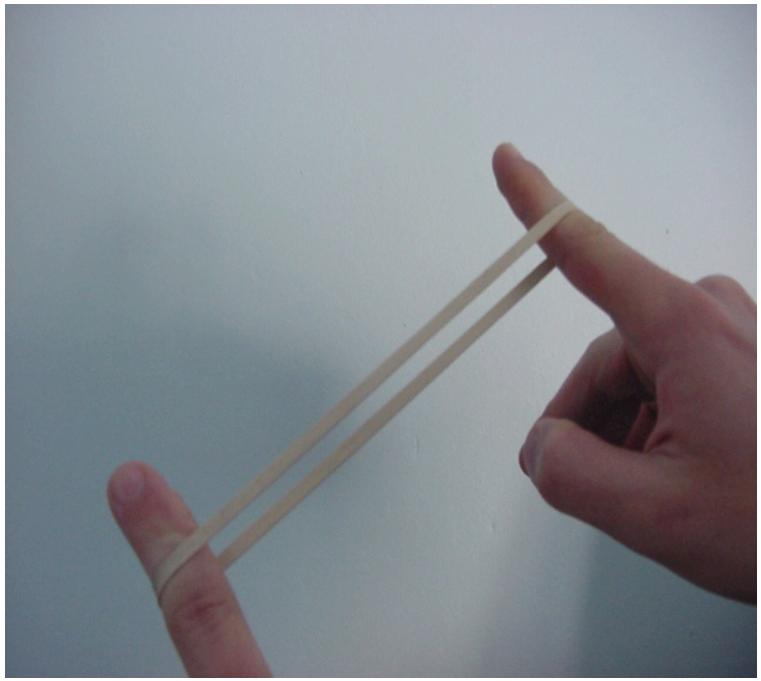
FIGURE 11-17

Airway innervation. The lung is innervated by both branches of the autonomic nervous system. Parasympathetic neurotransmitters to airway smooth muscle include acetylcholine (Ach), vasoactive intestinal peptide (VIP), and nitric oxide (NO). The sympathetic neurotransmitter is norepinephrine (NE). Other transmitters include substance P (SP) and neurokinin A (NKA). Receptor endings located in both the epithelium and smooth muscle include slowly adapting (SAR) and rapidly adapting (RAR) receptors, along with endings of unmyelinated fibres (C-fibres). JG, jugular ganglion. NG, nodose ganglion. DRG, dorsal root ganglion.

LUNG COMPLIANCE
A MEASURE OF DISTENSIBILITY
HOW EASILY AN OBJECT CAN BE STRETCHED

ELASTANCE is the inverse of compliance and refers to the tendency of an object to oppose stretch or distortion, as well as its ability to return to its original form after the distorting force is removed.

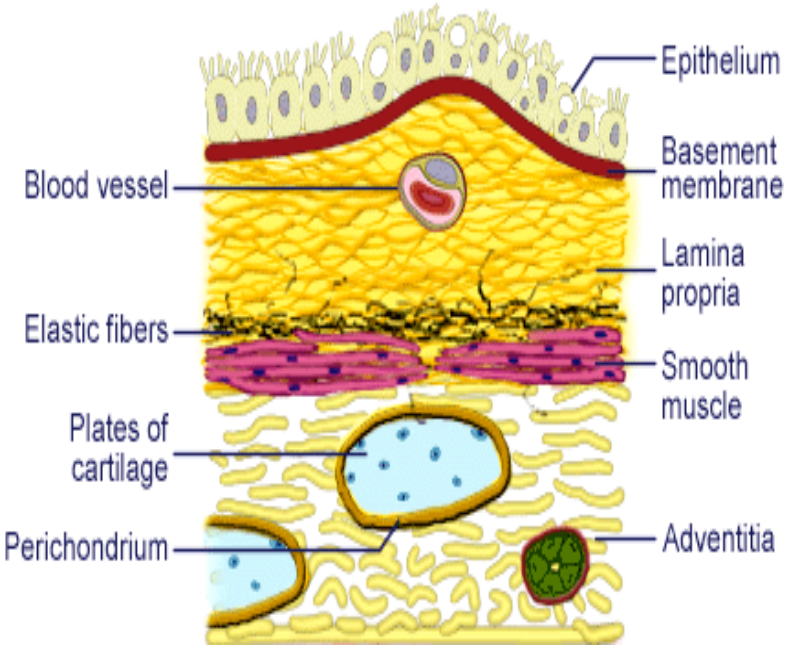
compliance = 1/elastance

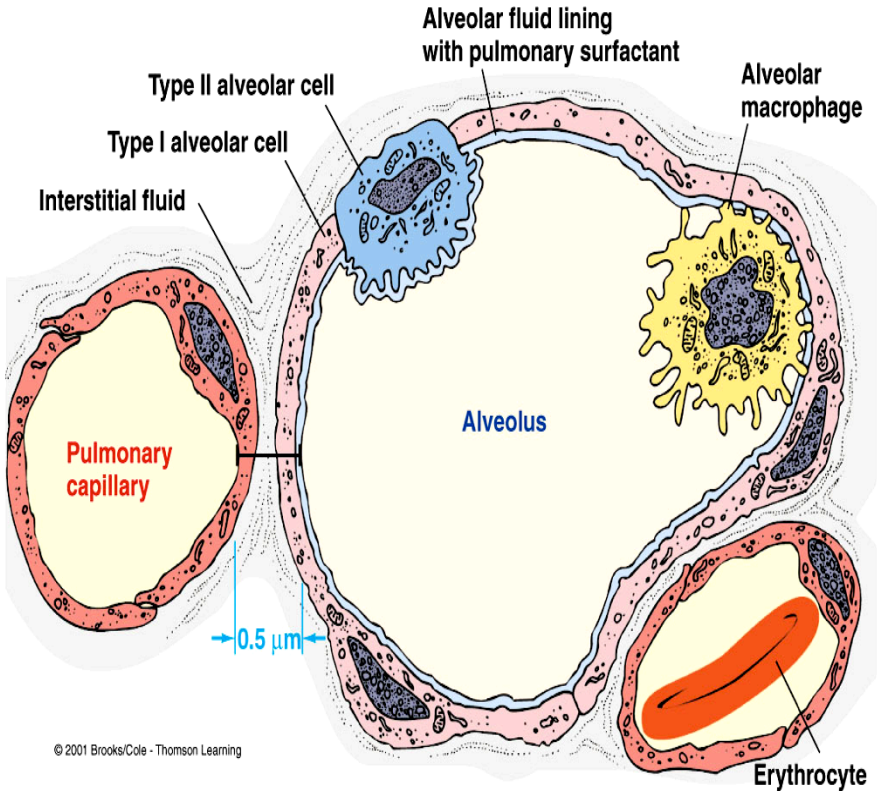


PHYSICAL PROPERTIES DETERMINING LUNG COMPLIANCE

LUNG TISSUE ELASTICITY

CONNECTIVE TISSUE SURROUNDS THE AIRWAYS

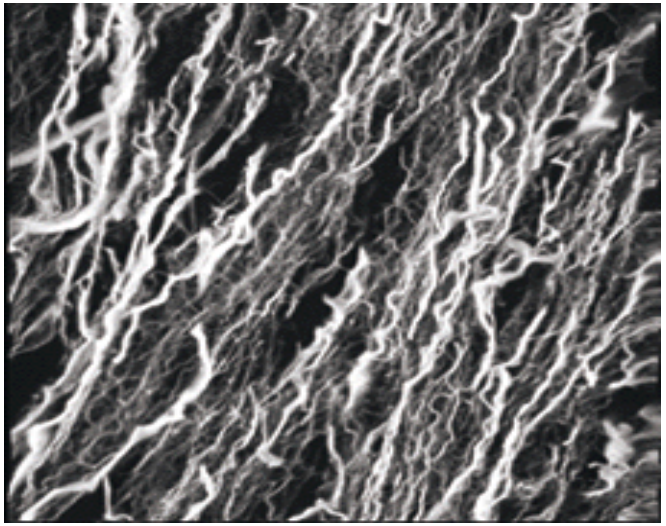




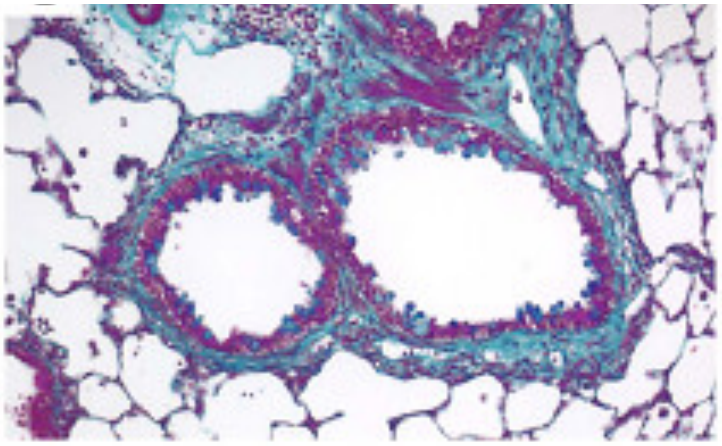
KEY CONNECTIVE TISSUE FIBERS

COLLAGEN

- like a strong twine
- high tensile strength
- inextensible



collagen fibers in rabbit heart muscle

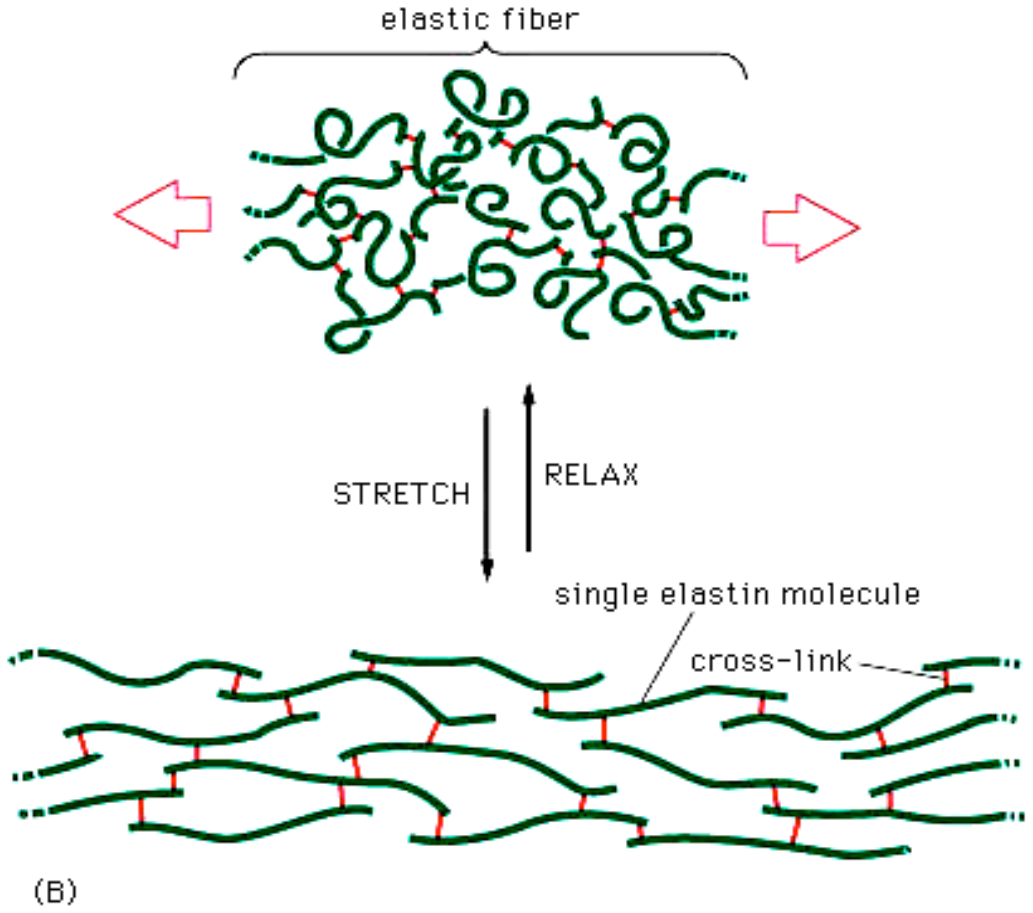


green stain highlighting collagen in airways

KEY CONNECTIVE TISSUE FIBERS

ELASTIN

- like a weak spring
- low tensile strength
- extensible



CHANGES IN LUNG COMPLIANCE

LOSS OF CONNECTIVE TISSUE

aging

↓

loss of elastin & collagen fibers

↓ ↓

wrinkled skin

↑ lung compliance
[floppy lungs]

EMPHYSEMA

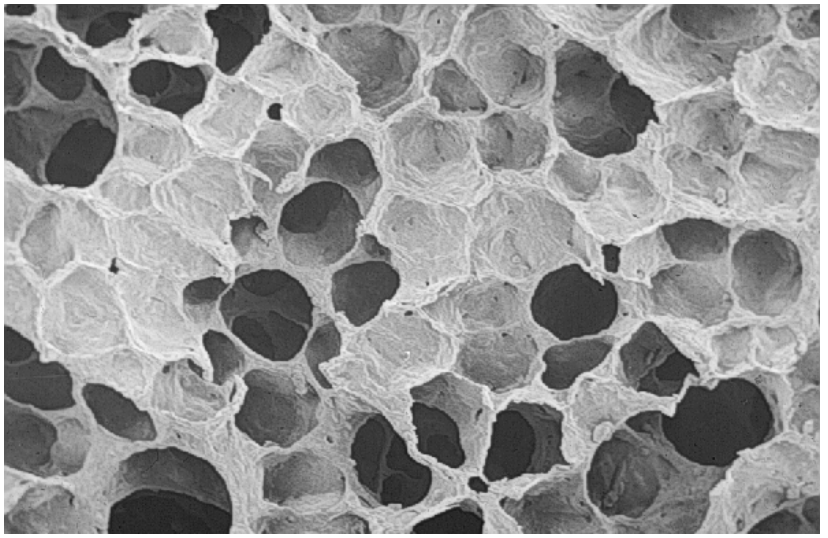
disappearing lung disease
smoking/genetically inherited

↓

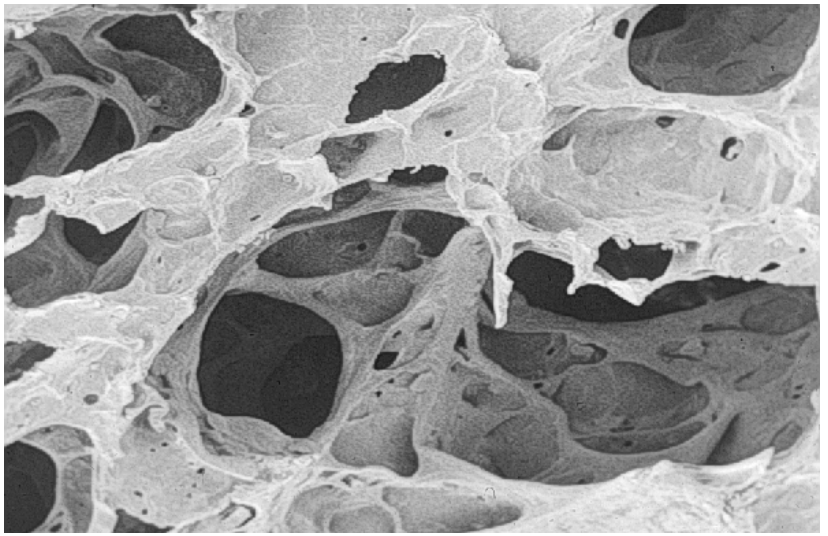
alveolar wall destruction

↓

↑ lung compliance
[floppy lungs]



EM of healthy lung tissue



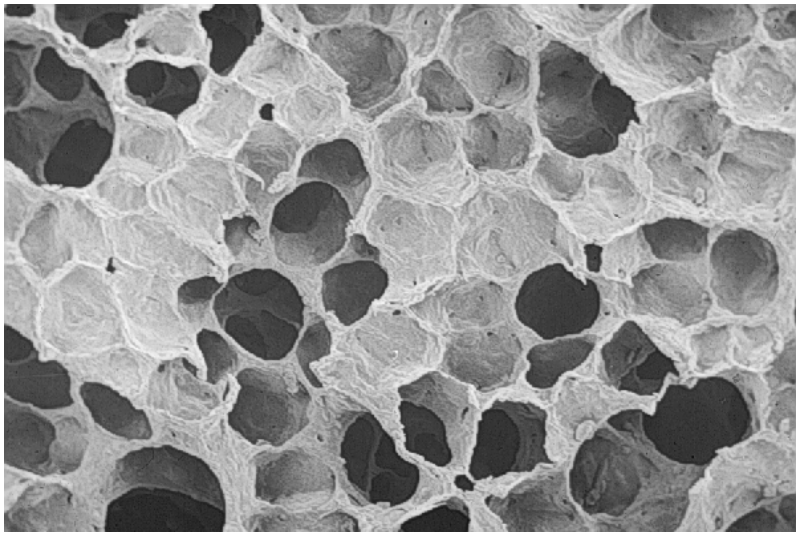
EM of emphysematous lungs

PULMONARY FIBROSIS

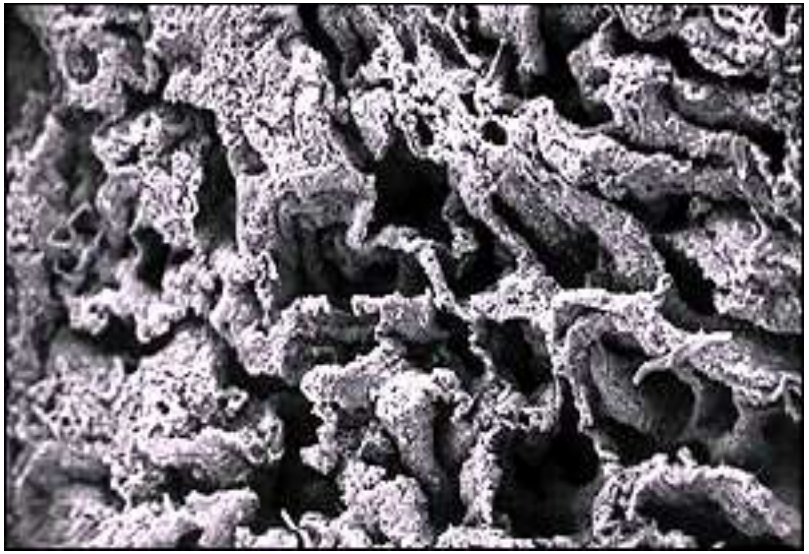
collagen deposition
in alveolar walls

a response to lung injury (e.g. asbestosis)

↓ lung compliance
[stiff lungs]

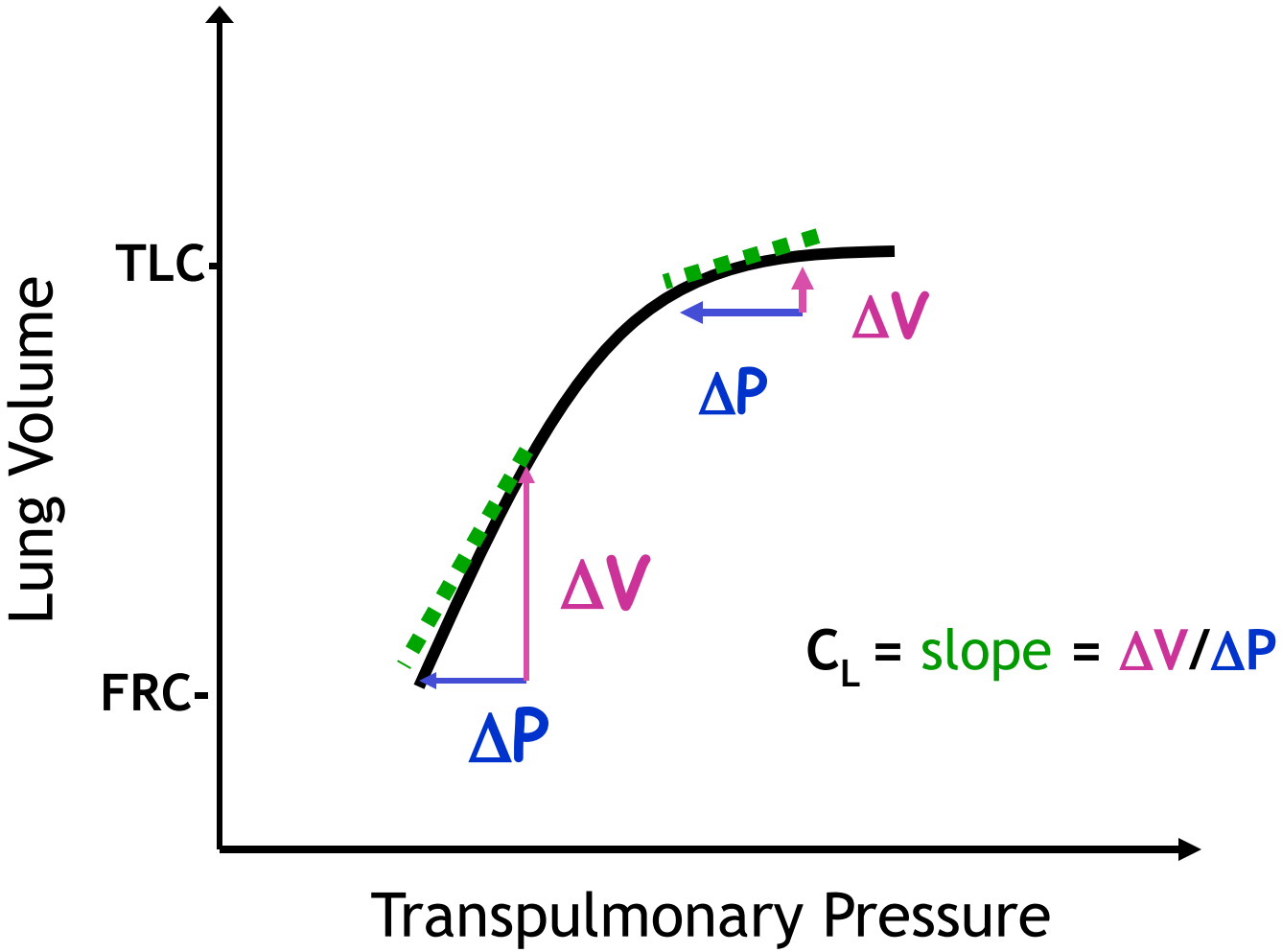


EM of healthy lung tissue



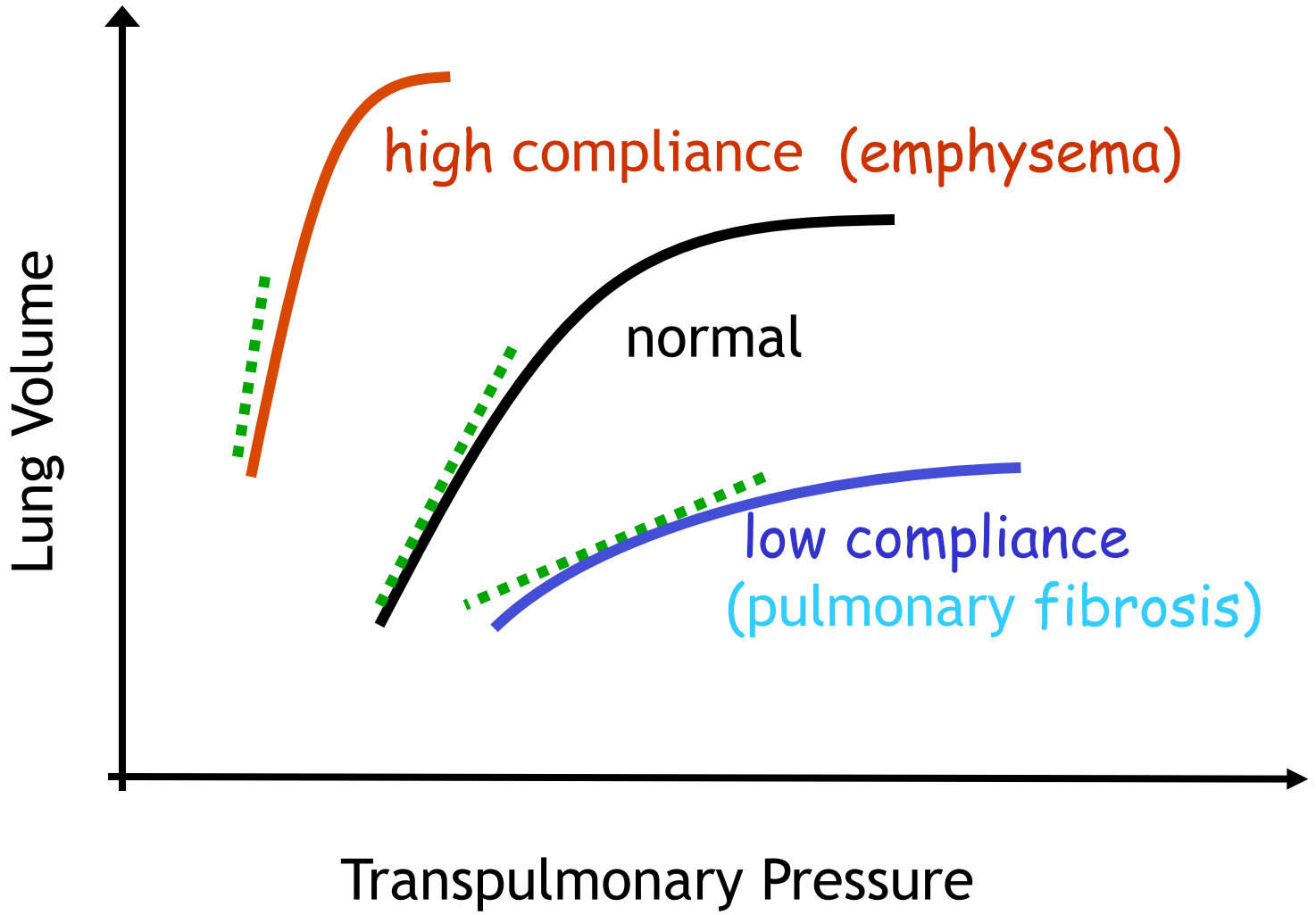
STATIC COMPLIANCE OF THE LUNGS

IS DETERMINED BY THE PV CURVE OF THE LUNGS AND DEPENDS ON LUNG VOLUME



STATIC COMPLIANCE OF THE LUNG

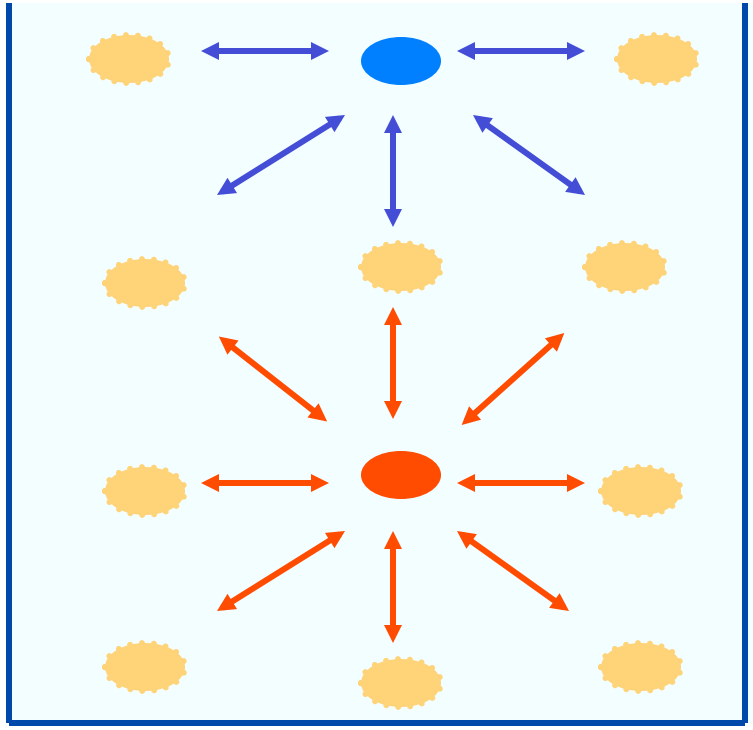
IS DETERMINED BY THE PV SLOPE AT FRC



PHYSICAL PROPERTIES DETERMINING LUNG COMPLIANCE

ALVEOLAR SURFACE TENSION

Water molecules at the surface of a liquid-gas interface are attracted strongly to the water molecules within the liquid mass. This cohesive force is called “surface tension”.



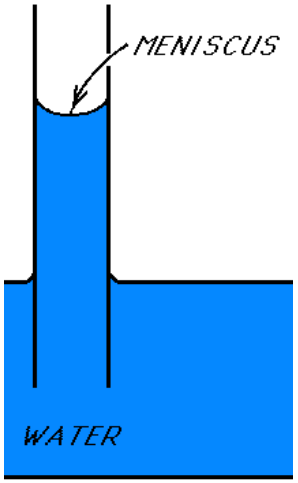
SURFACE TENSION MAKES IT POSSIBLE --

to float paper clips on water

for insects to walk on water

to maintain the shape of a water droplet

to reduce meniscus curvature created by capillary action



Surfactants and Surface Tension

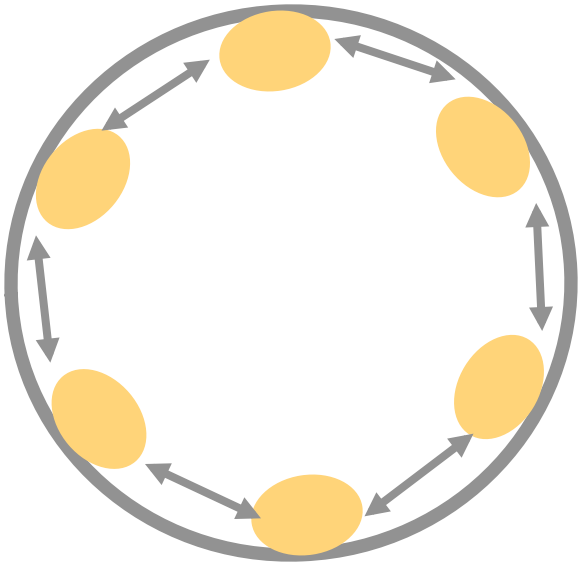
Chemistry and Physics for Nurse Anesthesia, Second Edition
A Student-Centered Approach

David Shubert and John Leyba

www.springerpub.com/product/978-0826190499

SPRINGER PUBLISHING COMPANY

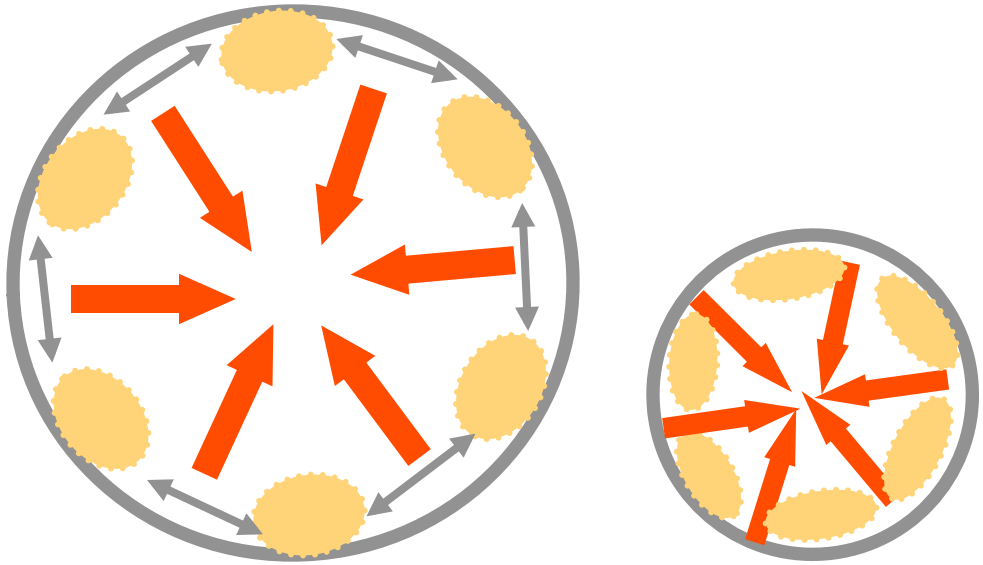
ALVEOLAR SURFACE TENSION



- Air entering the lungs is humidified & saturated with water vapour at body temperature.
- water molecules cover the alveolar surface
- Surface water molecules create substantial surface tension

ALVEOLAR SURFACE TENSION

CREATES INWARD RECOIL & ALVEOLAR COLLAPSE (ATELECTASIS)

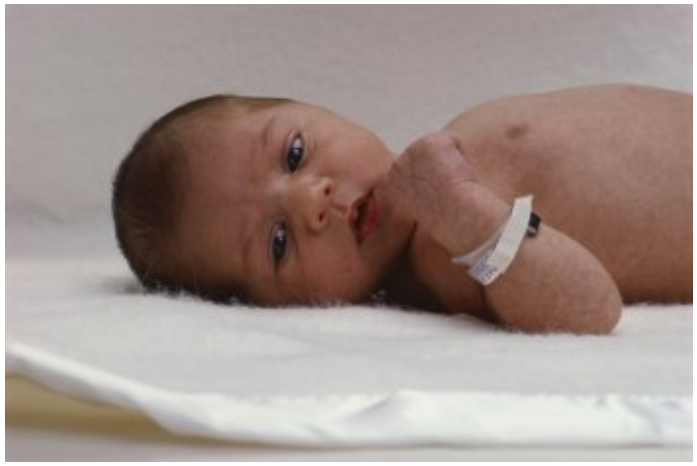


Pulmonary surfactant secreted from Type II alveolar cells reduces alveolar surface tension, prevents collapse and stabilizes the alveoli.

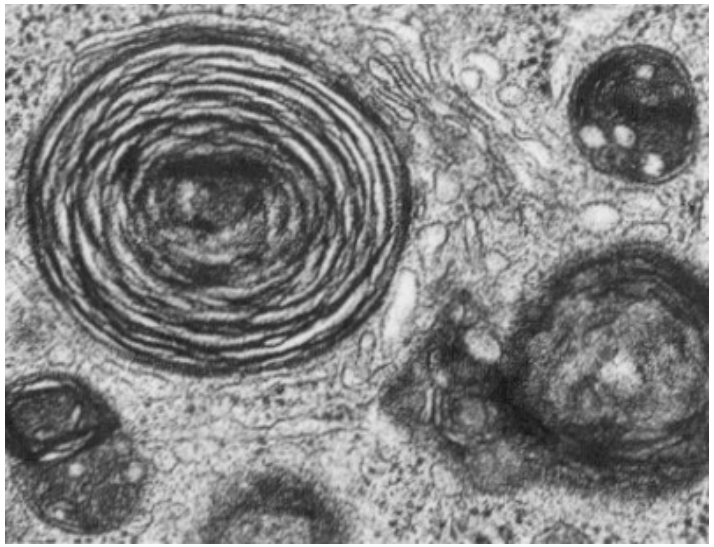
A CASE OF HIGH ALVEOLAR SURFACE TENSION

NEONATAL RESPIRATORY DISTRESS SYNDROME (NRDS)

Premature babies born with inadequate production of pulmonary surfactant have stiff lungs that are hard to inflate at birth.



- life threatening condition
- ventilator dependent
- treated with semi-synthetic surfactant delivered intra-tracheally
- mother is treated with corticosteriods during pregnancy



Lamellar inclusion bodies

THE CASE OF VIRGINIA SLIM

- 57 years old chronic smoker with severe COPD
- SOB & cough worsening in last 2 yrs
- quit smoking 5 years ago
- on inhaled bronchodilator therapy, regular physiotherapy, antibiotics for acute bacterial infections
- chair to bed existence with supplemental home oxygen
- in hospital waiting for potential lung volume resection surgery to remove her emphysematous lung

1.Why is Virginia using bronchodilators?

2.What causes Virginia's recurrent lung infections? How does this affect her airways resistance?

3.Why would having emphysema and a highly compliant lung make it harder for Virginia to breath out?