Pulmonary compliance

&

Airway resistance

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Lecture Objectives:

1. Define lung compliance and identify two common clinical conditions in which lung compliance is higher or lower than normal.
2. Describe and draw the pressure-volume of the lung (compliance) curves for the lungs.
3. Draw a normal pulmonary pressure-volume (compliance) curve (starting from residual volume to total lung capacity and back to residual volume), labeling the inflation and deflation limbs.
4. Predict changes in lung compliance in restrictive and obstructive lung diseases. And list the factors that contribute to the work of breathing.
5. Define airway resistance and review the biophysical physical principles of airway resistance.
6. Define laminar and turbulent flow.
7. Identify the chief site of airway resistance under normal conditions. And describe the effects of changing lung volumes on resistance.
8. Describe humeral and neural control of airways resistance.
9. Describe the dynamic compression of airways during forced expiration and its physiological significance.
10. Define surface tension and describe how it applies to lung mechanics, including its effects on the alveolar size. And define the role of surfactants in preventing atelectasis.

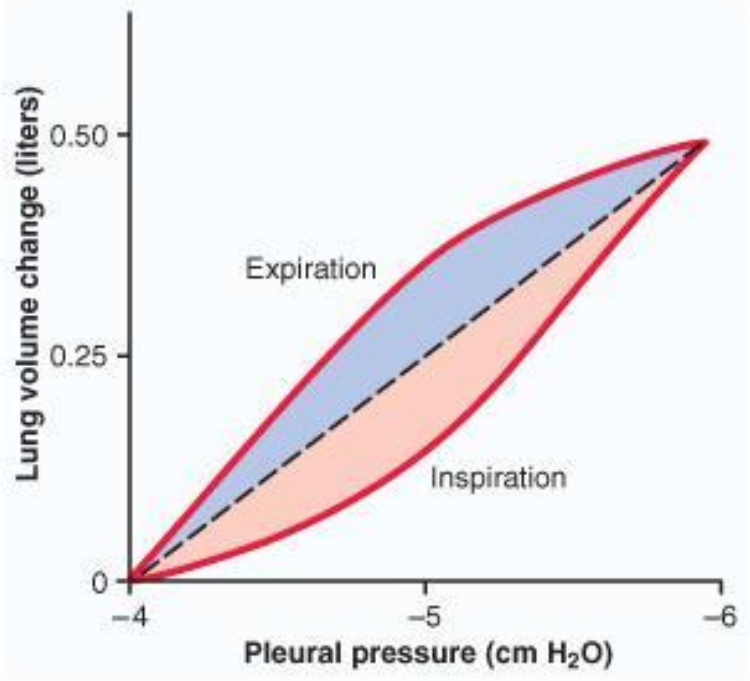
Compliance (distensibility) of The Lungs:

$$\text{Lung compliance} = \frac{\Delta \text{ in lung volume}}{1 \text{ cm } H_2O \text{ transpulmonary pr.}}$$

(Normal value is 200 ml of air/cm H_2O)

Compliance is due to:

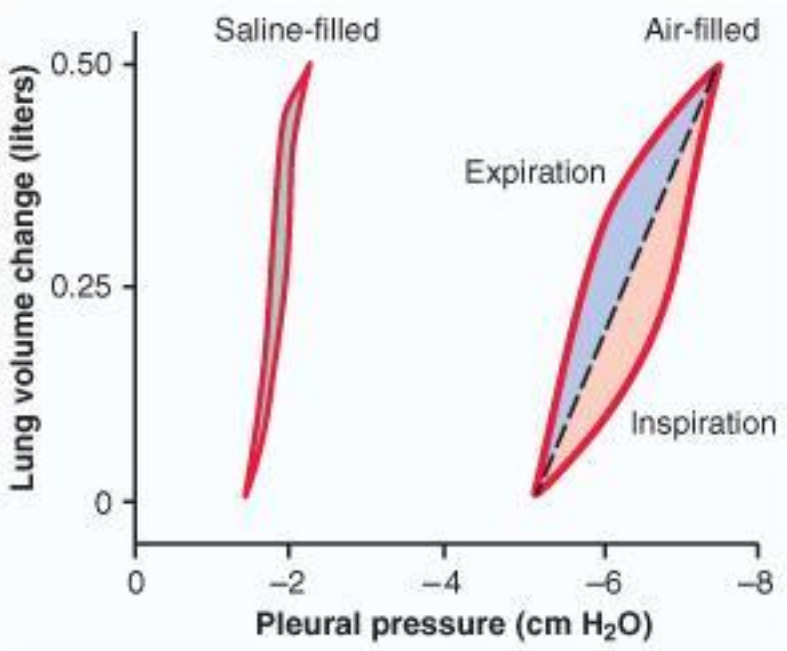
1. Elastic forces of the lung tissue (⅓ of total lung elasticity)
2. Elastic forces caused by surface tension of the fluid that lines the alveoli and lung air spaces (⅔ of total lung elasticity)



Compliance (distensibility) of The Lungs (cont.)

Note:

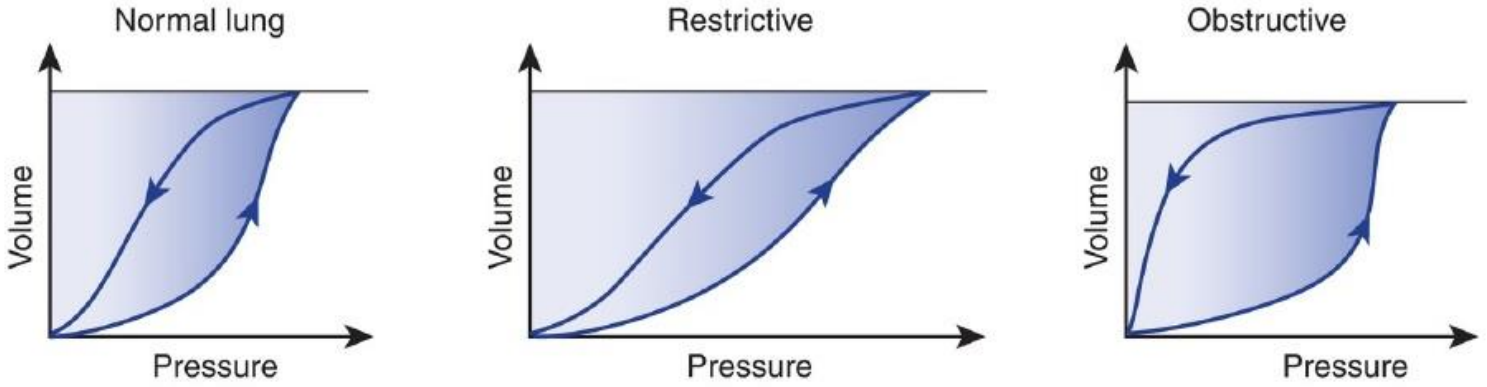
1. Compliance and elastance are inversely related. Lung volume is set at the point when the elastance force balances the transpulmonary pressure.
2. Inflation of the lungs (inspiration) follows a different curve than deflation of the lung (expiration); this difference is called **hysteresis**.



Compliance (distensibility) of The Lungs (cont.)

Decreased compliance of the lung can be caused by lung diseases (e.g. tuberculosis, silicosis) that produce scarring or fibrosis of the lungs → **restrictive lung disease (RLD).**

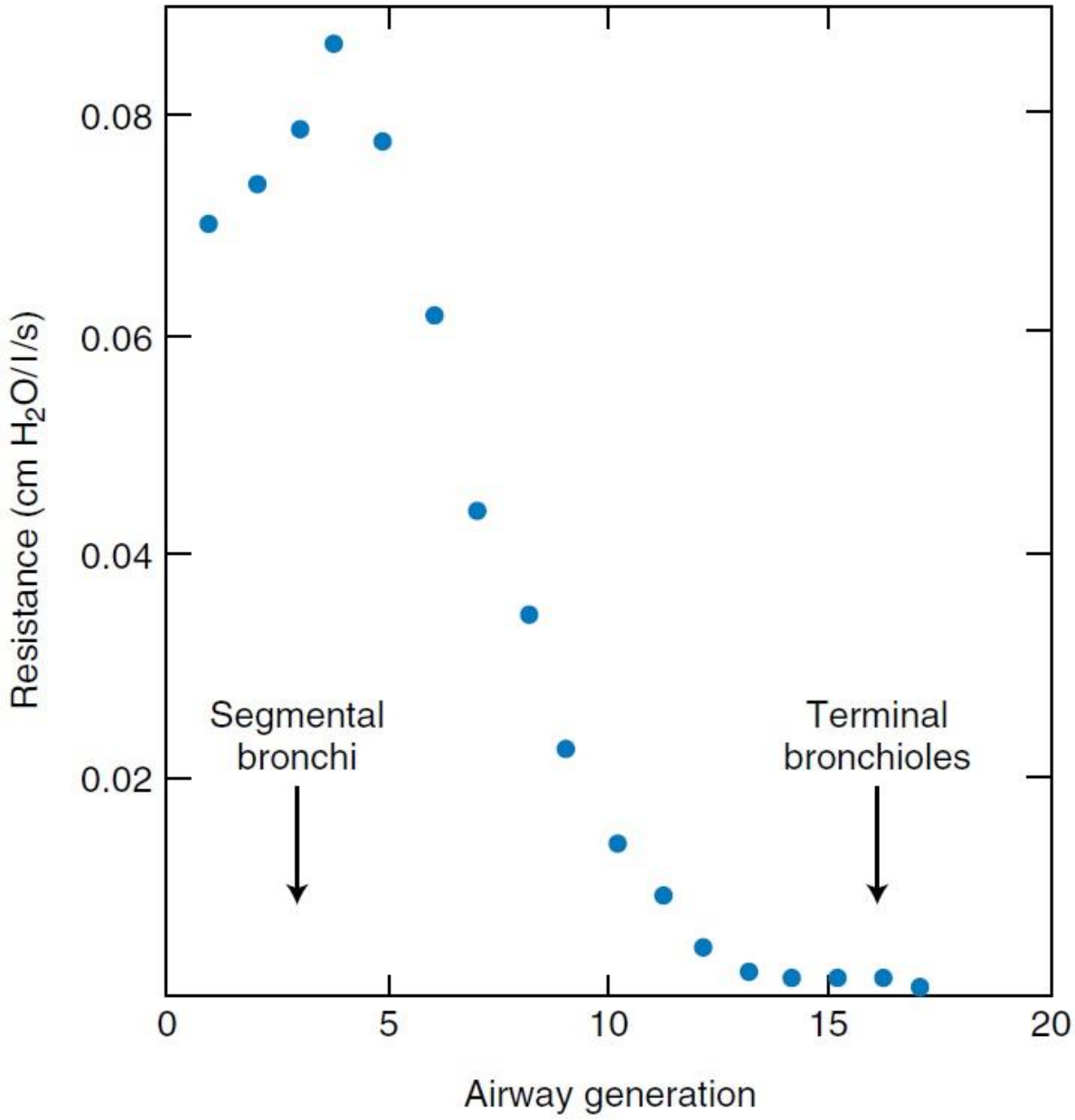
Increased compliance is produced by the pathologic processes that occur in **emphysema** as well from the **aging process.** The chest becomes **barrel-shaped**.



Work of breathing is measured from a pressure–volume curve

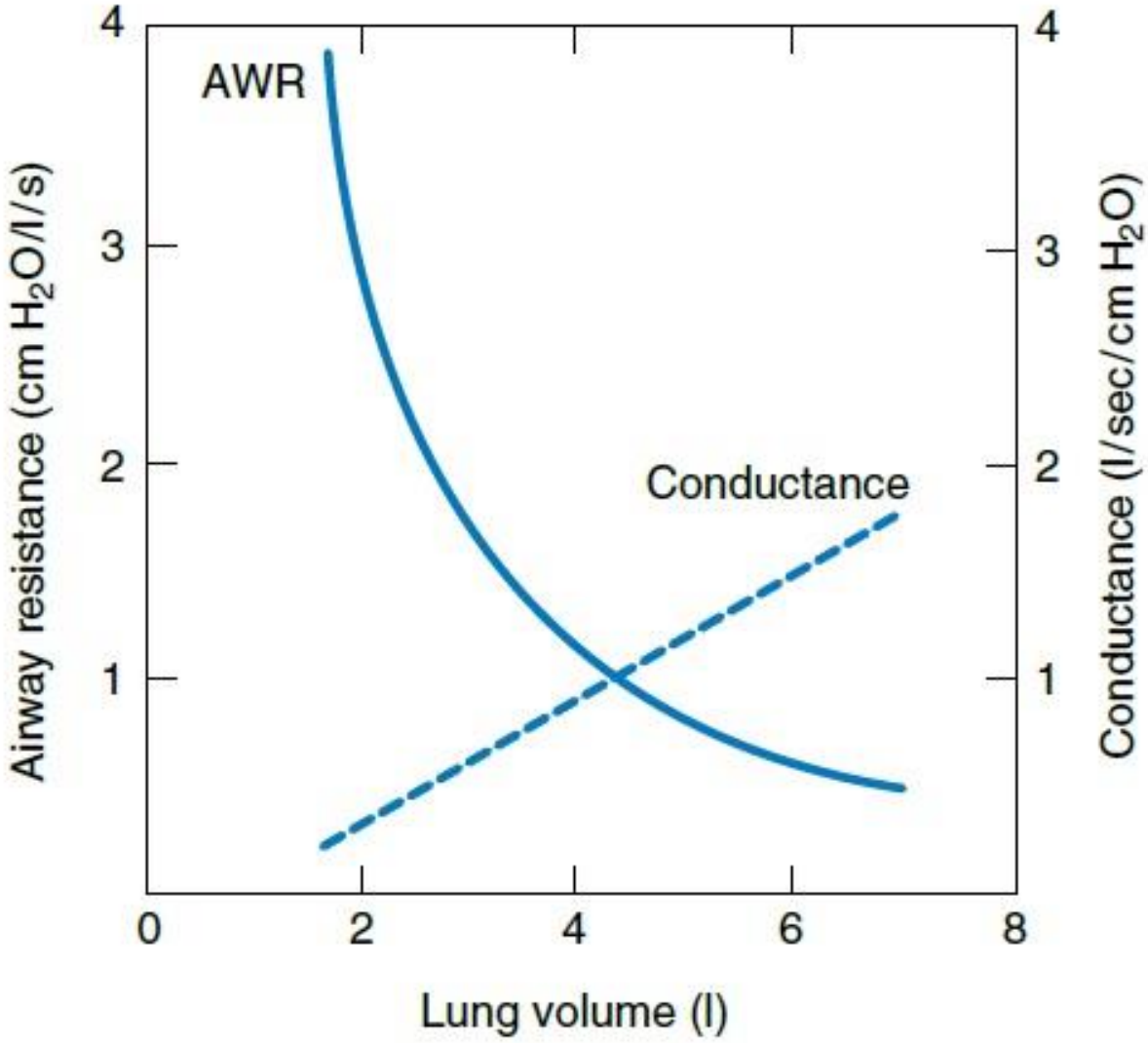
AIRWAY RESISTANCE AND THE WORK OF BREATHING

- Airway resistance is the pressure difference between the alveoli and the mouth divided by a flow rate.
- Total resistance to airflow in the lungs has two components:
  1. Tissue resistance of the lungs and chest wall (20% of total resistance).
  2. Airway resistance (80% of the total resistance).
- The major site of airway resistance is the **medium-sized** bronchi (lobar and segmental) and bronchi down to about the ***seventh generation***, where airway turbulence is the greatest.
- Only 10% to 20% of total airway resistance can be attributed to the small airways (those <2 mm in diameter), about generation 8.

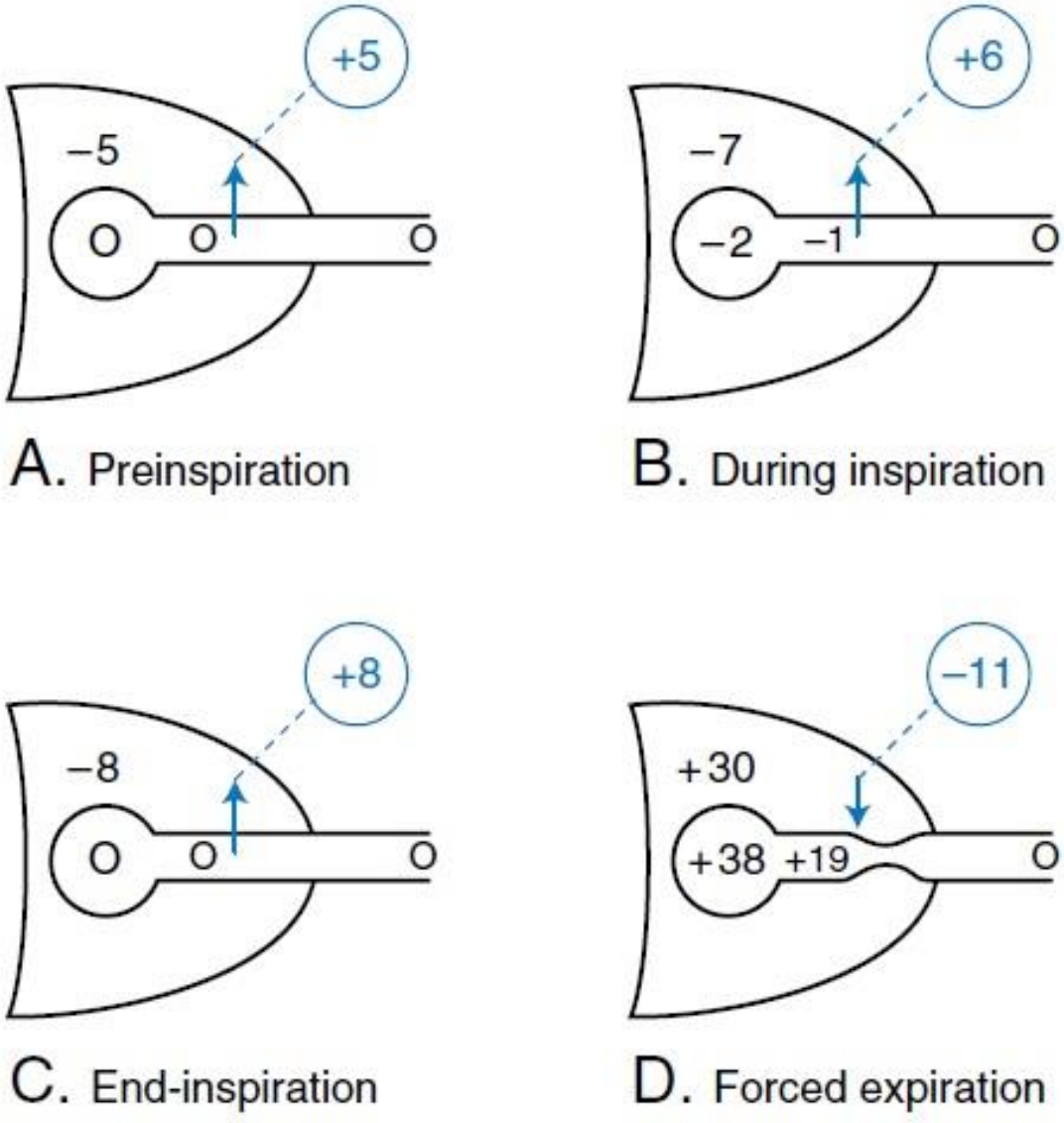

0.08
0.06
0.04
0.02
Resistance (cm H_2O/l/s)
Segmental bronchi
Terminal bronchioles
0
5
10
15
20
Airway generation

Factors Determining Airway Resistance:

- Lung volume has an important effect on airway resistance, the bronchi are supported by the radial traction of the surrounding lung tissue, and their caliber is increased as the lung expands.
- As the lung enlarges, airway diameter increases, which results in a concomitant decrease in airway resistance during inspiration. Conversely, at low lung volumes, airways are compressed (especially at the bottom of the lung), and airway resistance rises.
  Note: Patients who have increased airway resistance often breathe at high lung volumes; this helps to reduce their airway resistance.
- Stimulation of **parasympathetic** cholinergic postganglionic fibers causes bronchial constriction as well as increased mucus secretion. Also, reflex stimulation of receptors in the trachea and large bronchi by irritants such as cigarette smoke can induce bronchial smooth muscle contraction.
- Stimulation of **sympathetic** adrenergic fibers (β_2 receptors) causes dilation of bronchial and bronchiolar airways and inhibition of glandular secretion. Therefore, selective β_2-adrenergic agonists are extensively used in the treatments of asthma and chronic obstructive pulmonary disease (COPD). Anticholinergic agents are used in COPD as well.



Variation of airway resistance with lung volume. If the reciprocal of airway resistance (conductance) is plotted, the graph is a straight line.



A–D. Scheme showing why airways are compressed during forced expiration. Note that the pressure difference across the airway is holding it open, except during a forced expiration.

Surfactant:

Is an agent that greatly **reduces** the surface tension of the water, which increases the compliance of the lungs, thereby decreasing the work of respiration and facilitates the reopening of collapsed airways and alveoli. Surfactant reduces the pulmonary capillary filtration as it makes pulmonary interstitial pressure less negative. Thus it keeps the alveoli dry and helps prevent pulmonary edema, which interferes with gas exchange.

It is secreted by **type II alveolar cuboidal epithelial cells** (constitute 10% of alveolar surface).

Surfactant is made up from a complex mixture of phospholipids, surfactant apoproteins, and calcium ions. It reduces pure water surface tension force from 72 dynes/cm to a range of 5-30 dynes/cm.

***Note*:**

Young-Laplace's Law states that transmural pressure (P) depends on both the radius (r) and surface (wall) tension (T).

$$P = 2*T/r$$

Respiratory distress syndrome (RDS) of the newborn:

Surfactant is secreted into the alveoli between the 6th and 7th months of gestation and is almost always present by gestational week 35. Premature birth and maternal diabetes are risk factors. Premature babies have lungs that tend to collapse due to lack of surfactant and small alveoli → RDS that is characterized by impaired gas exchange and **hypoxemia** (there is lung collapse or **atelectasis**, edema, and hemorrhage within the lungs). Mortality rate is high.

Atelectasis: is the partial collapse or closure of a lung resulting in reduced or absent gas exchange.

Therapy includes administration of **exogenous surfactant** with the use of mechanical respirators.

Test Question:

Q. Pulmonary surfactant:

A. Increases the surface tension of the alveolar lining liquid.

B. Is secreted by type I alveolar epithelial cells.

C. Is a protein.

D. Increases the work required to expand the lung.

E. Helps to prevent transudation of fluid from the capillaries into the alveolar spaces.