Biology 3UO3 - 2012
Animal Physiology

Pulmonary ventilation

__Readings:__
Chapter 37:
pgs. 465-475

Office hours:

When: Mondays 10-11am

Where: LSB-532

Email: paluzzi@mcmaster.ca

Pulmonary ventilation is simply: “the inflow and outflow of air between the atmosphere and the lung alveoli”

Pulmonary ventilation

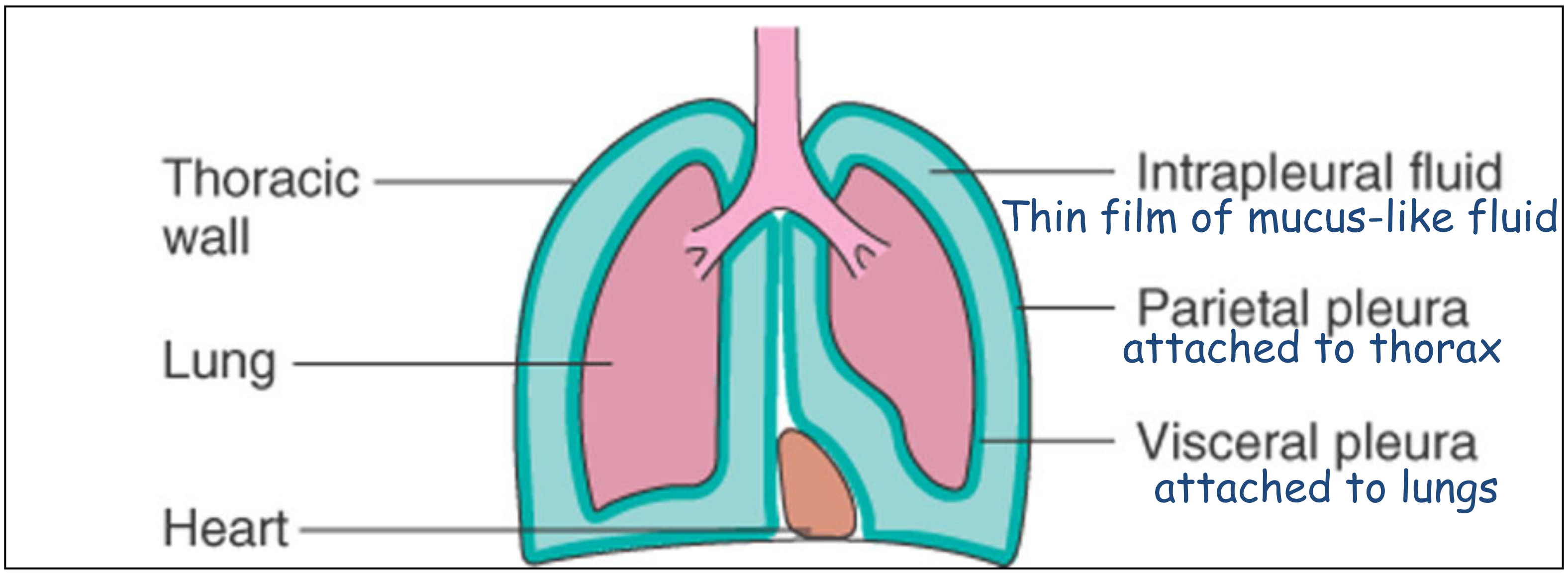
- Involves the exchange of air between the atmosphere and the lungs
- Air moving into the lungs faces high air pressure whereas air moving out of the lungs faces low air pressure

Goals for today's lecture:

- Relate Boyle's law to ventilation
- Identify muscles used in ventilation
- Understand how volume changes in the thoracic cavity cause pressure changes leading to air flow
- Identify factors that influence airway resistance and lung compliance

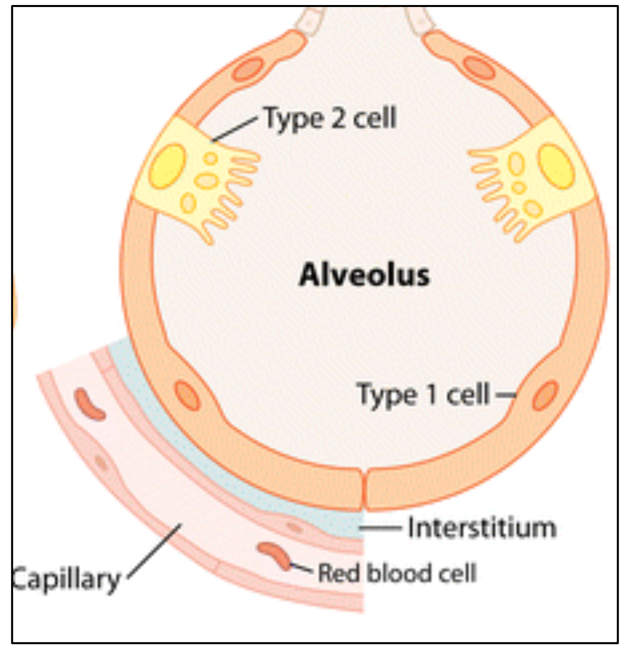
What you need to know (ie review)

- Location of visceral and parietal pleurae
- Pleural cavity containing pleural fluid which acts as a lubricant



What you need to know (ie review)

- Role of surfactant-secreting alveolar epithelial cells
  - Prevent alveolar collapse by releasing surfactant, which lowers surface tension
  - Surfactant is a mixture of phospholipids and lipoproteins that interfere with H_2O molecule attraction

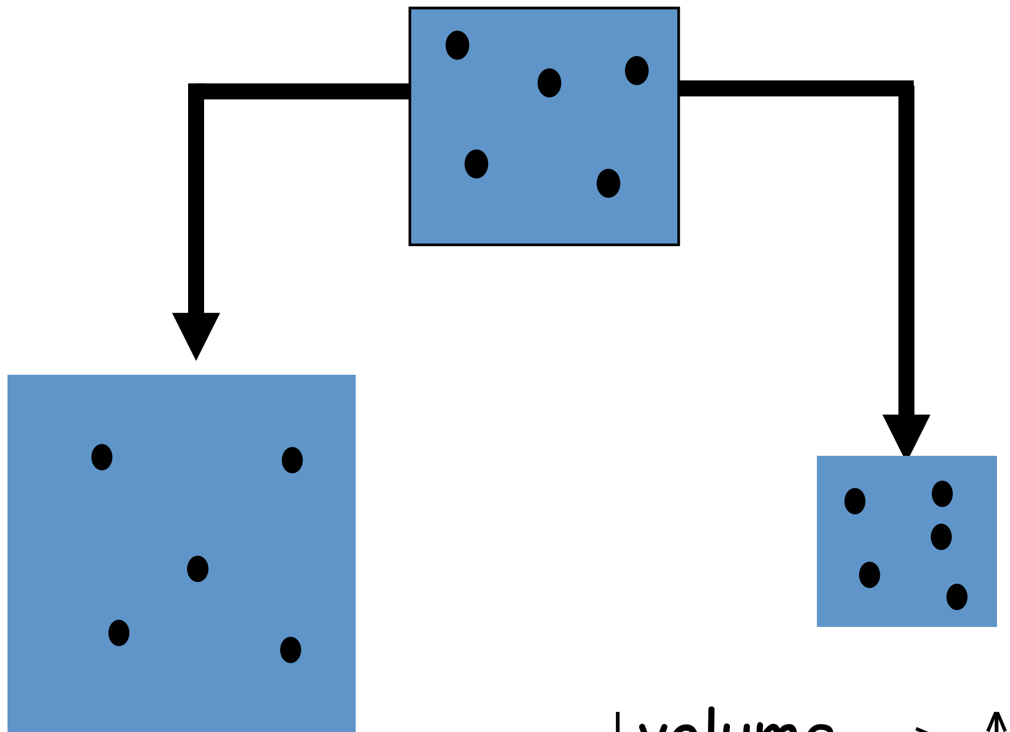


from Eaton et al., 2009 Annu Rev Physiol

Boyle's Law

- Pressure is caused by gas molecules striking the walls of a container, whereby the pressure is inversely proportional to the volume
  - Larger volume leads to lower pressure since gas molecules strike the container walls less frequently
  - In contrast, smaller volume leads to gas molecules striking the container more frequently, thus exerting higher pressure

Boyle's Law



↓volume → ↑pressure

↑volume → ↓pressure

Breathing Cycle

- **Inspiration** - *active* phase during both rest & exercise
  - External intercostal muscles pull ribs out & up
  - Diaphragm contracts and moves down (most important at rest)

Thoracic Volume ↑

Lung Volume ↑

Decreased pressure in lungs (i.e below atmospheric pressure)

Air flows in from atmosphere

"thoracic suction"

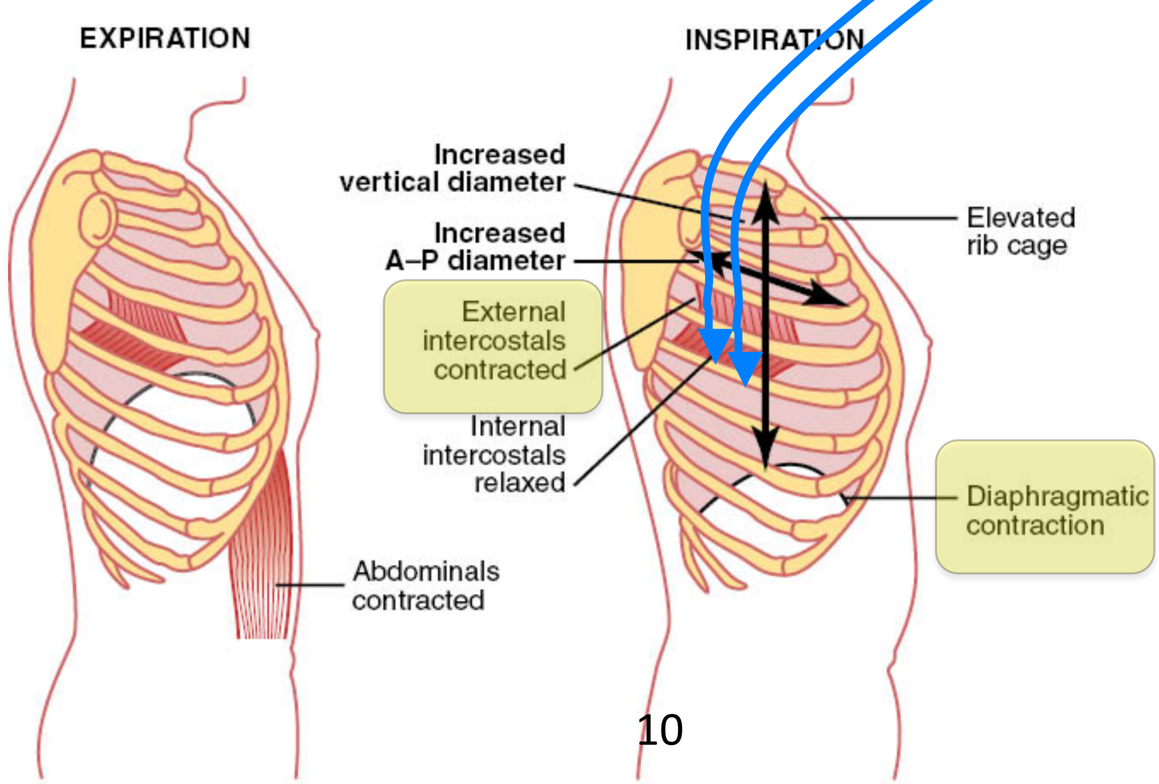


Figure 37-1 Contraction and expansion of the thoracic cage during expiration and inspiration, demonstrating diaphragmatic contraction, function of the

Breathing Cycle

Expiration – passive phase during rest- slow

- Due to elastic recoil of thoracic & lung components
- Diaphragm and external intercostal muscles relax

Thoracic Volume ↓

Lung Volume ↓

Increased pressure in lungs (i.e above atmospheric pressure)

Air flows out to atmosphere

"thoracic force"

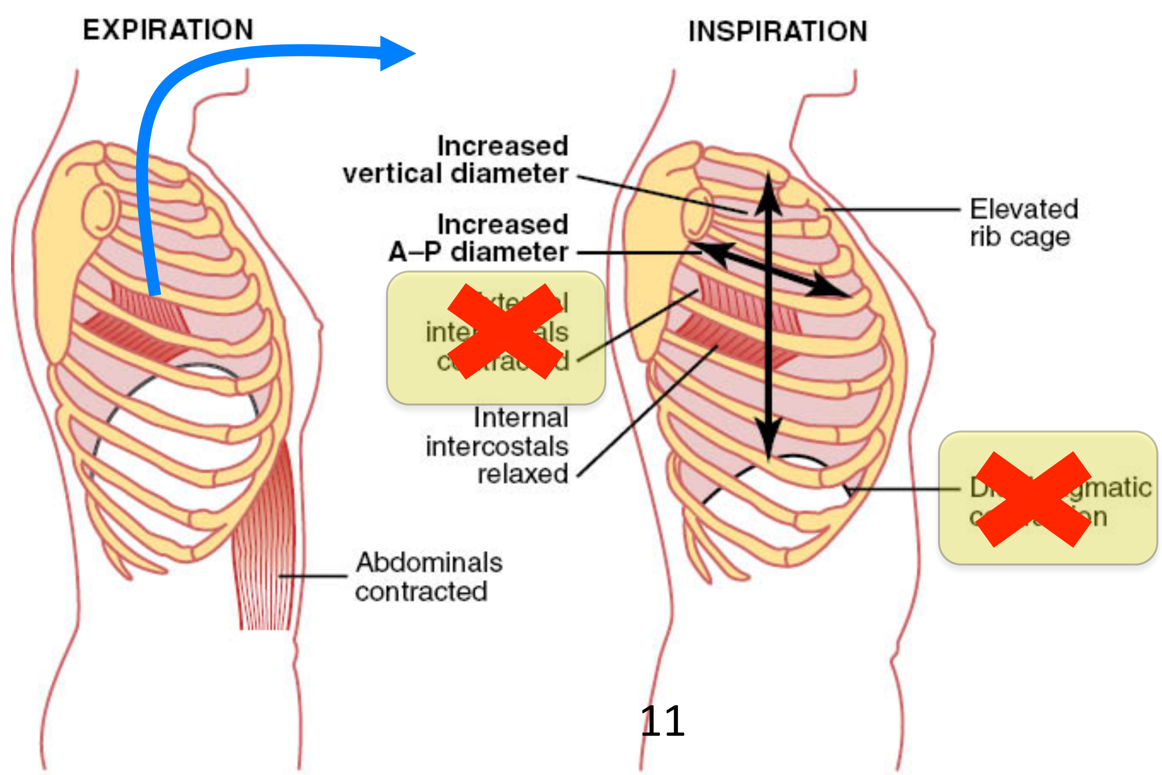


Figure 37-1 Contraction and expansion of the thoracic cage during expiration and inspiration, demonstrating diaphragmatic contraction, function of the

Breathing Cycle

Expiration – active phase during exercise - faster

- Internal intercostal muscles pull ribs in & down
- Abdominal muscles push "guts" in, thereby displacing diaphragm upwards

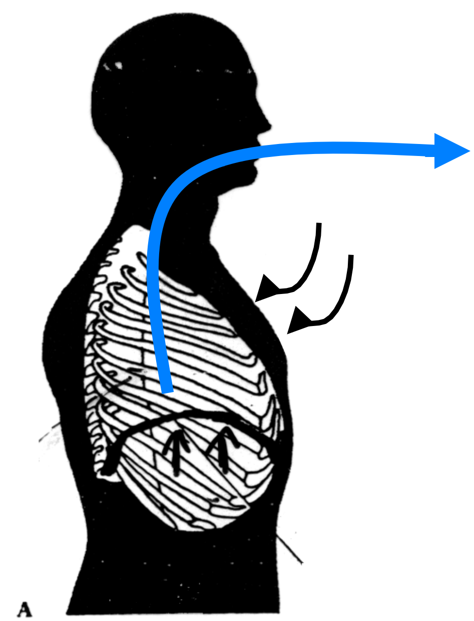
Thoracic Volume ↓↓

Lung Volume ↓↓

Greater increase in pressure in lungs (i.e above atmospheric pressure)

Air flows out to atmosphere

"thoracic force"



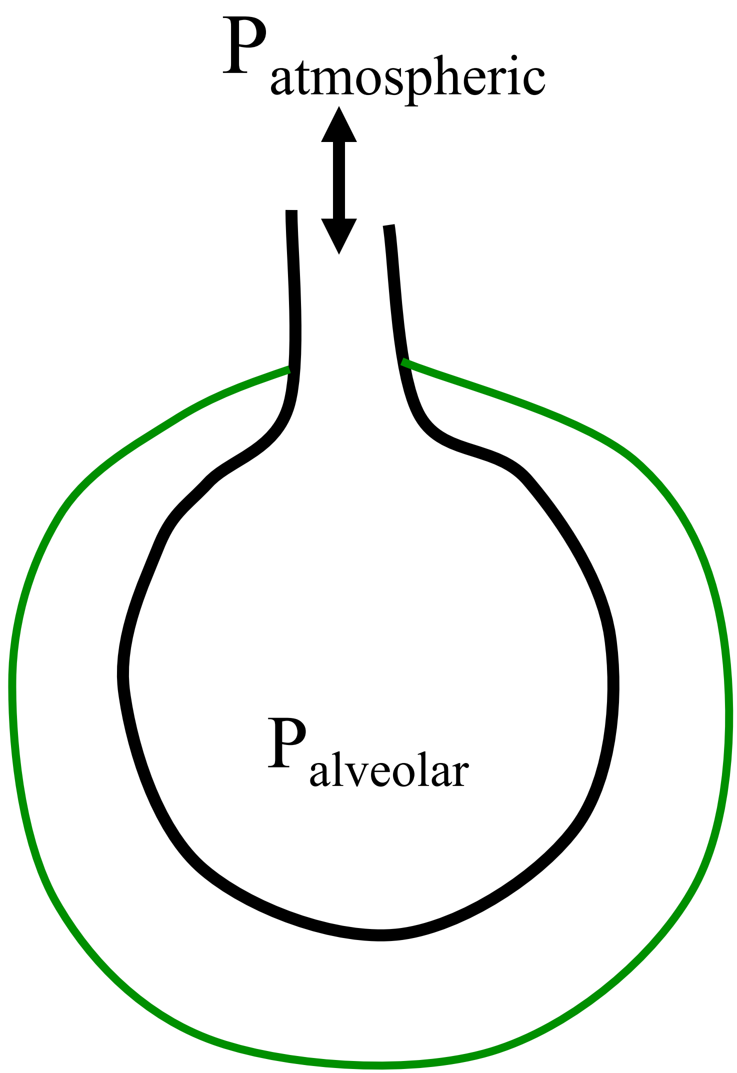
Intrapulmonary pressure

- Intrapulmonary (or intra-alveolar) pressure equals atmospheric pressure between breaths
- During inspiration, volume ↑ & pressure ↓ so air flows into the lungs (changes from 0 to -1)
- During expiration, volume ↓ & pressure ↑ so air flows out of the lungs (changes from 0 to +1)

Intrapleural pressure

- Intrapleural pressure relates to the pressure within the pleural cavity
- Intrapleural pressure is **always** negative providing suction to keep lungs inflated
- Negative pressure is due to:
  - Surface tension of alveolar fluid
  - Elasticity of lungs
  - Elasticity of thoracic wall

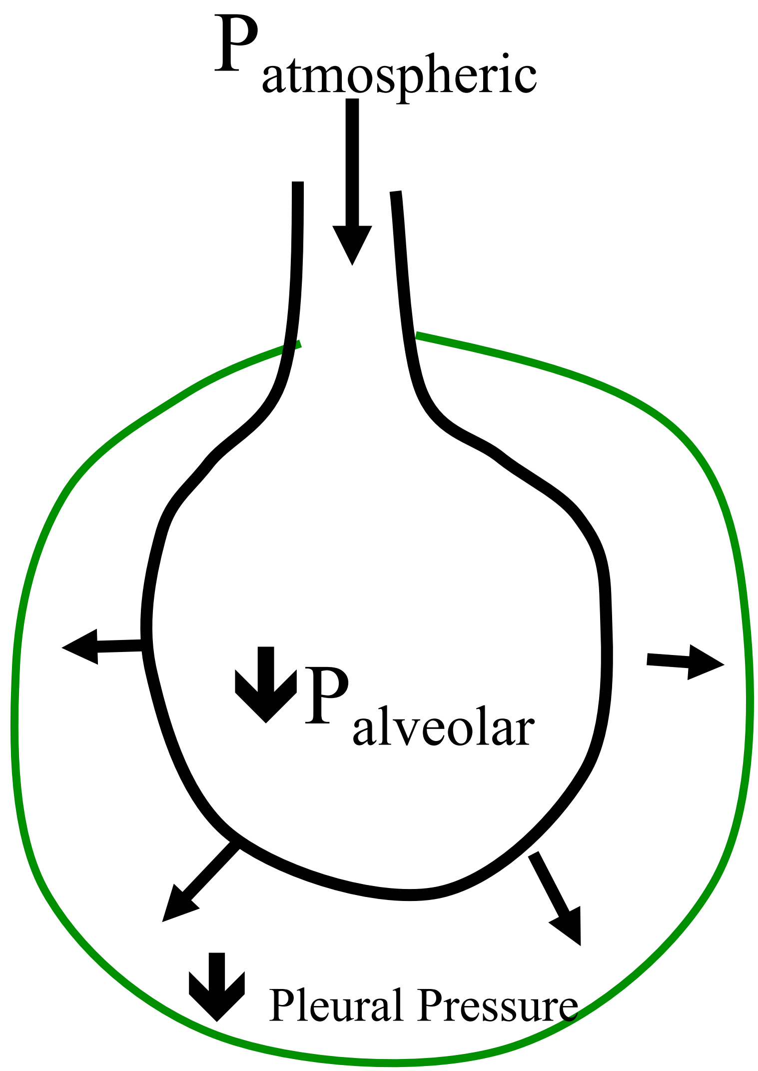
Intrapleural pressure changes



Rest

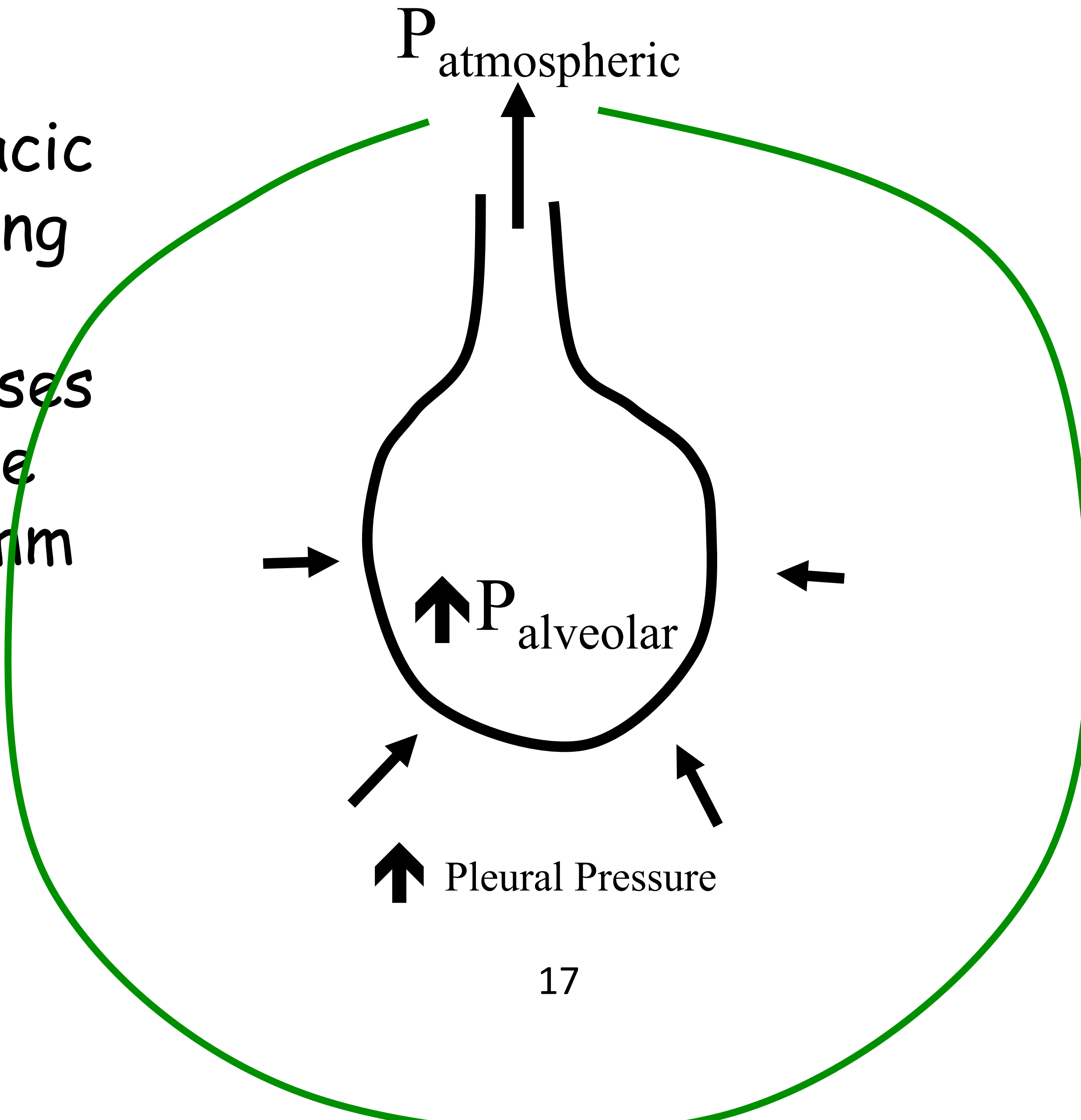
Intrapleural pressure changes

- As the thoracic wall moves outward during inspiration, the intrapleural pressure becomes more negative (from -4 to -6 cm Hg)



Intrapleural pressure changes

- When the thoracic wall recoils during expiration, the pressure increases but still negative (from -6 to -4 mm Hg)



Effect of Pneumothorax

- What do you expect would happen if you cut through the thoracic wall into the pleural cavity?
- Lung will collapse since air enters the pleural cavity, which eliminates the negative pressure
- The difference in pressure, called the transpulmonary pressure, keeps the lungs inflated

Events during inspiration

- Diaphragm and external intercostal muscles contract
- Volume of thoracic cavity increases
- Intrapleural pressure decreases and lungs expand
- Intrapulmonary pressure decreases allowing air to flow into the lungs

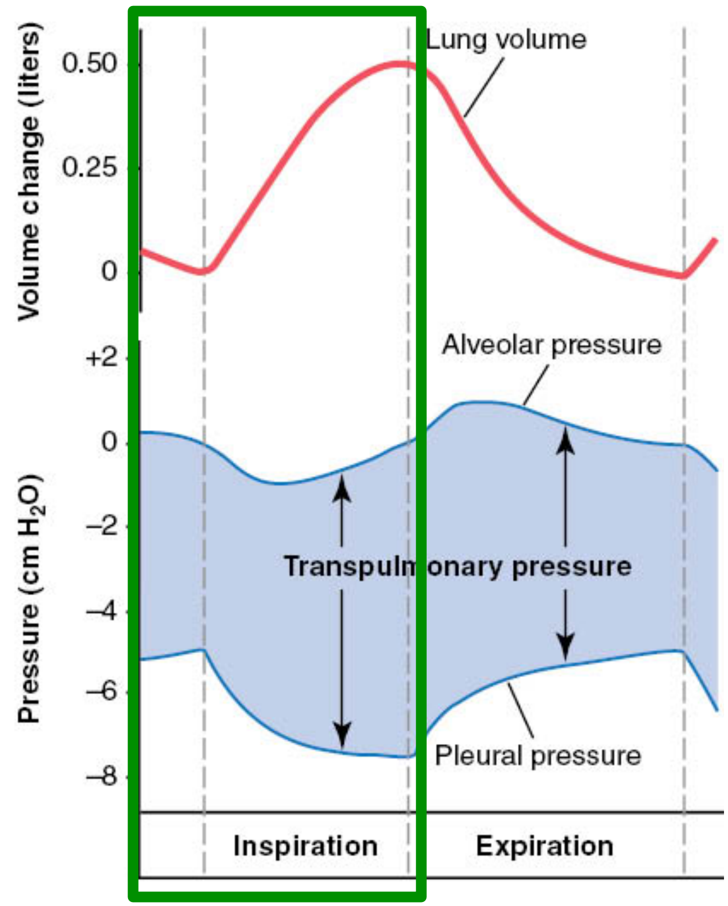


Figure 37-2 Changes in lung volume, alveolar pressure, pleural pressure, and transpulmonary pressure during normal breathing.

Events during expiration

- Diaphragm and external intercostal muscles relax
- Volume of thoracic cavity decreases
- Intrapleural pressure increases and lungs recoil
- Intrapulmonary pressure increases allowing air to flow out of the lungs

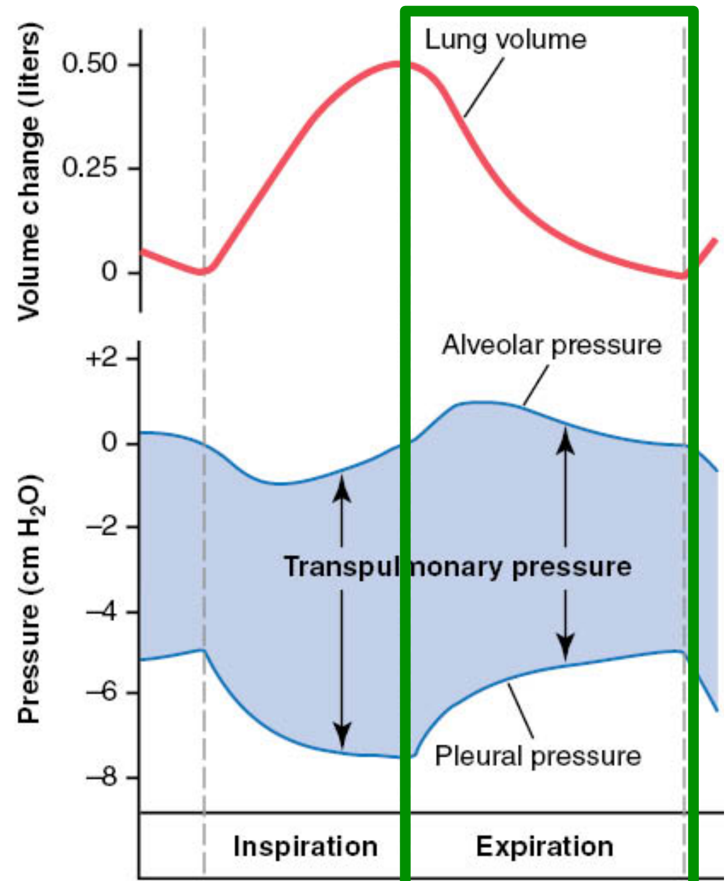


Figure 37-2 Changes in lung volume, alveolar pressure, pleural pressure, and transpulmonary pressure during normal breathing.

Functional Lung Volumes
Spirometer

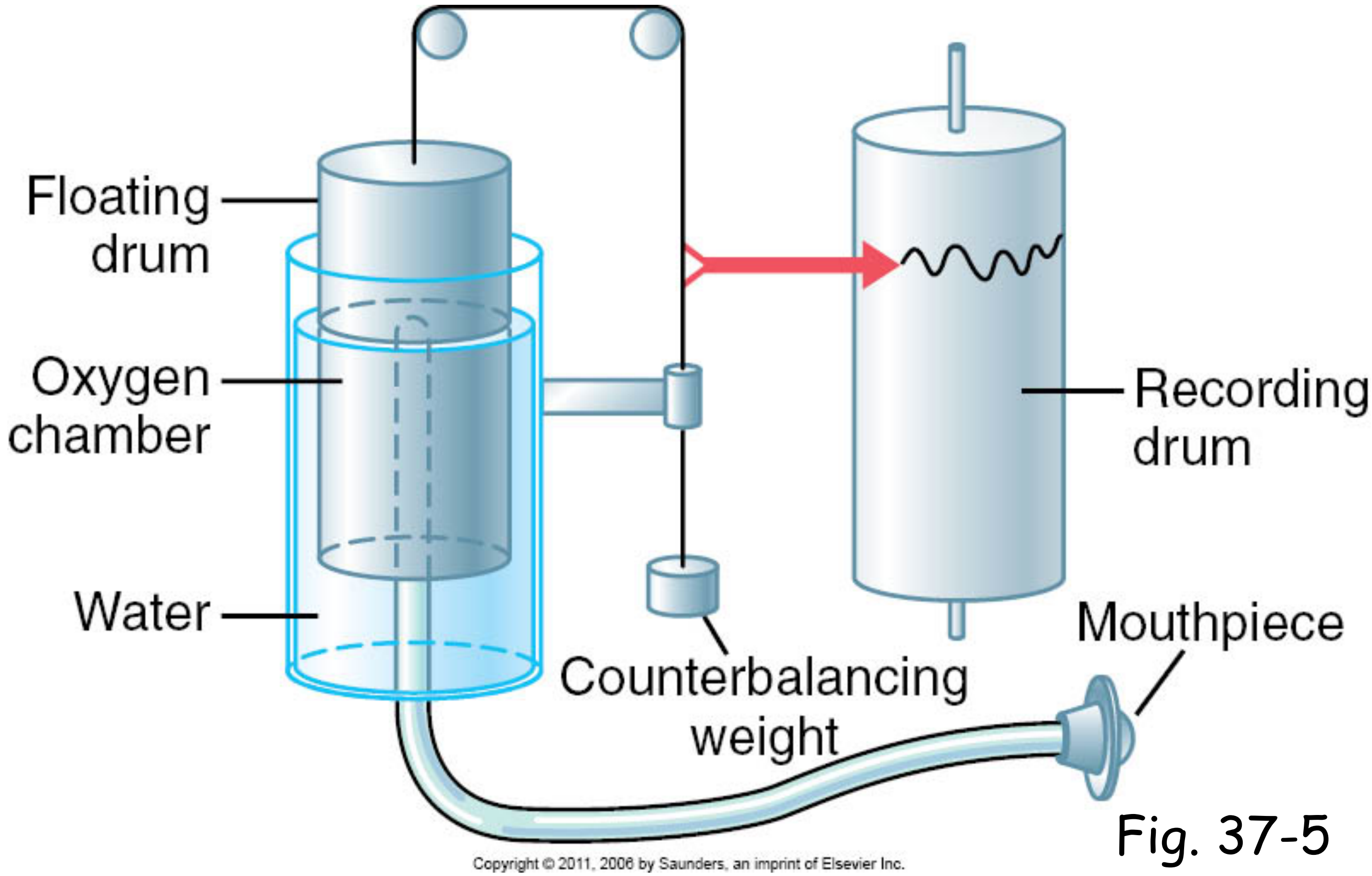


Fig. 37-5

Functional Lung Volumes

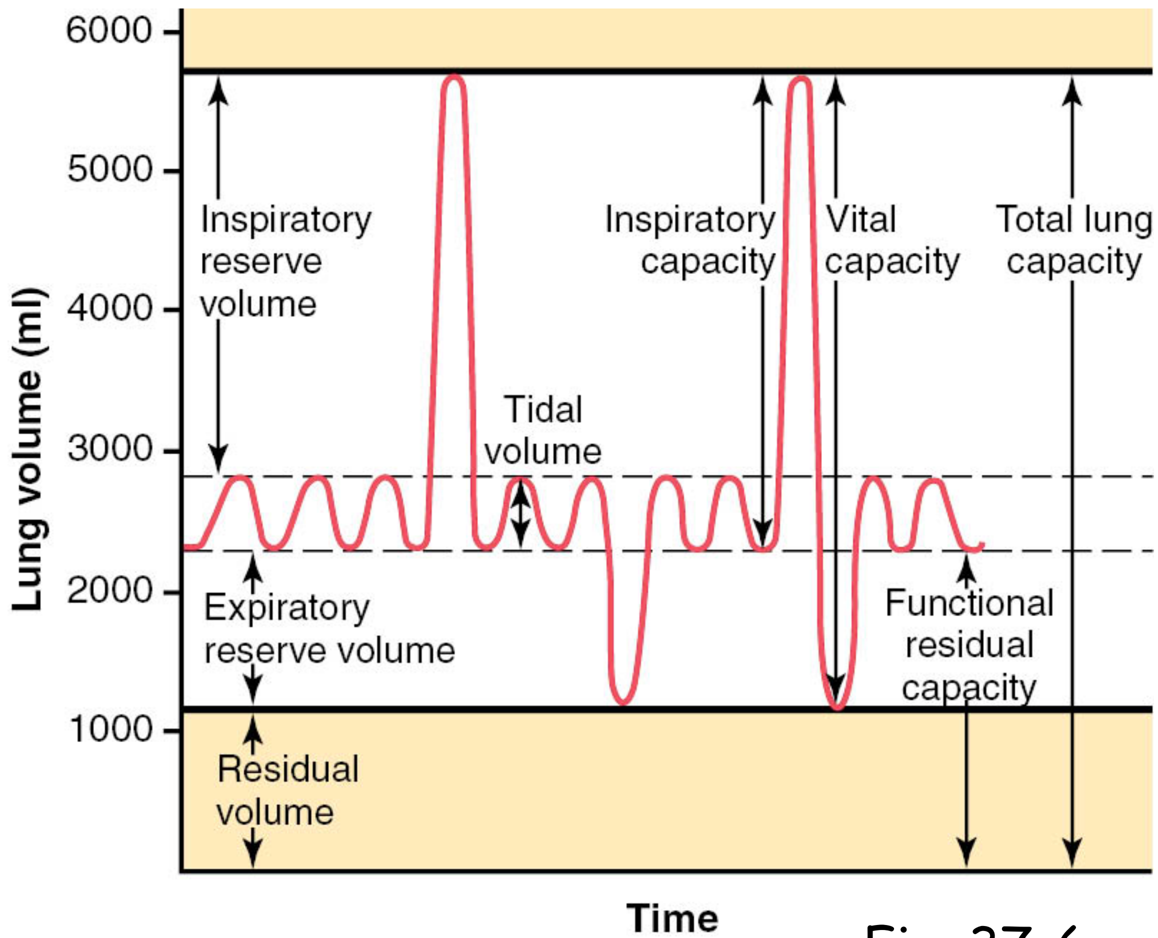


Fig. 37-6

Functional Lung Volumes

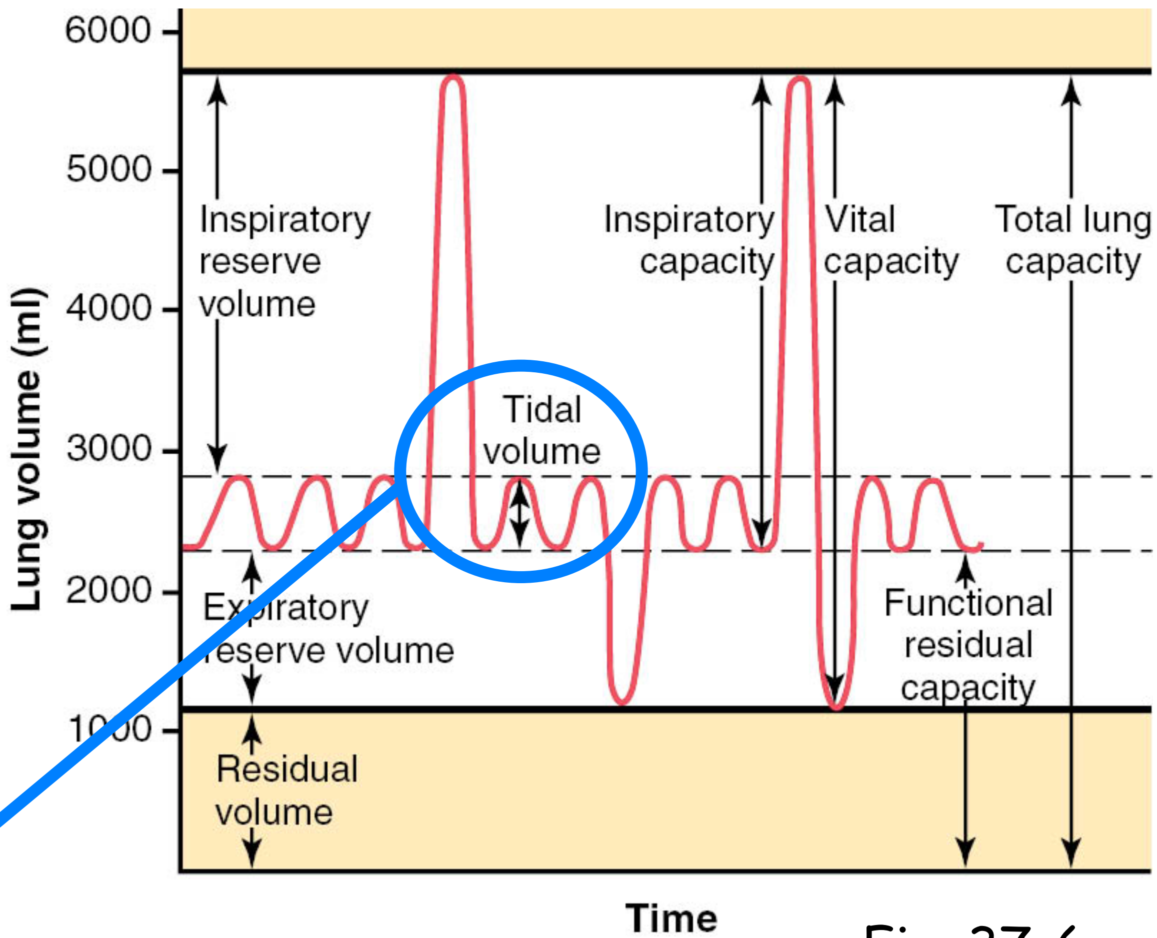


Fig. 37-6

V_T

Volume of air inspired (or expired) with each normal breath (~500 mL in adult males)

Functional Lung Volumes

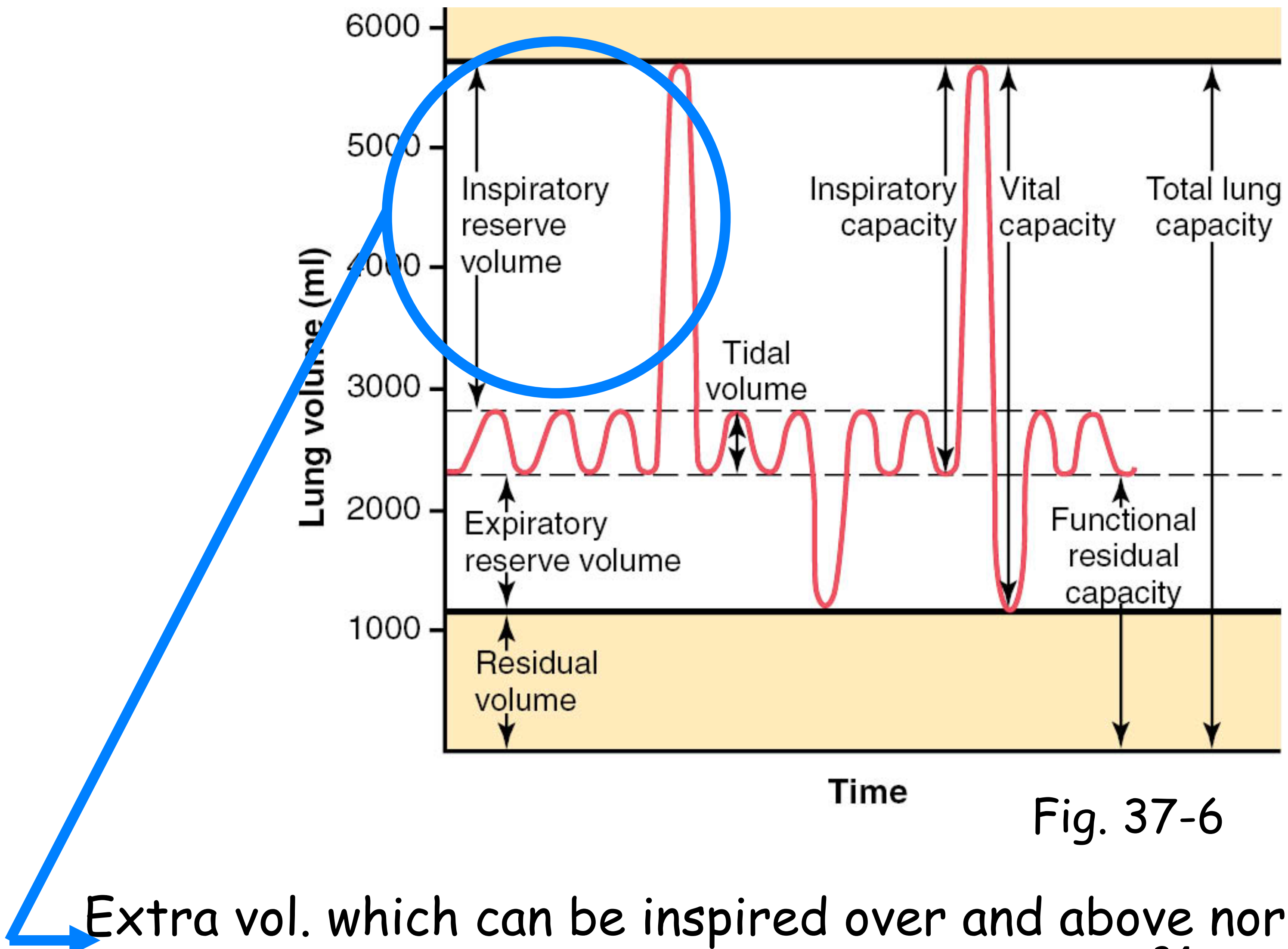


Fig. 37-6

IRV

Extra vol. which can be inspired over and above normal tidal vol. (~3000 mL in adult males)

Functional Lung Volumes

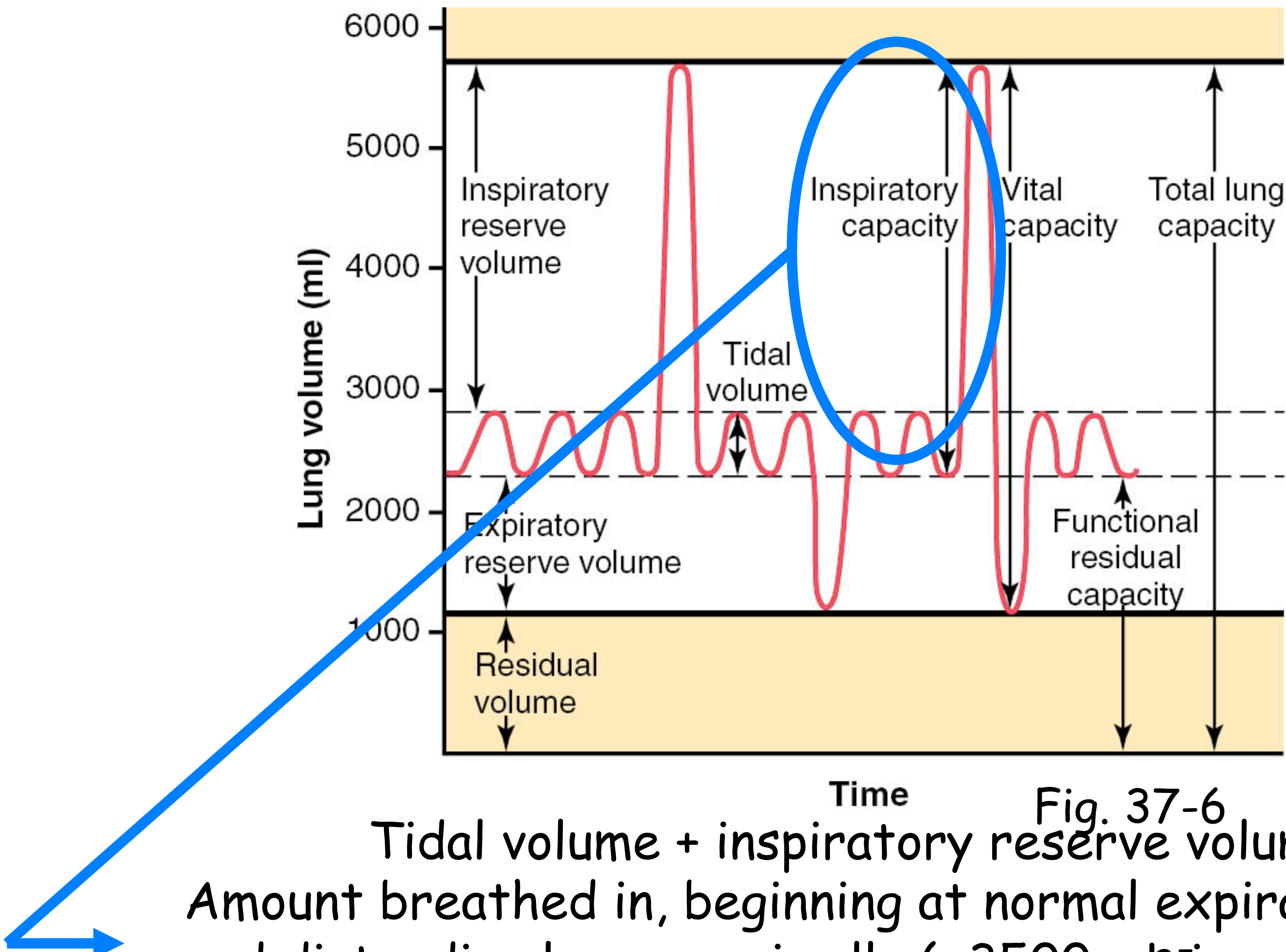


Fig. 37-6

IC

Tidal volume + inspiratory reserve volume

Amount breathed in, beginning at normal expiratory level and distending lungs maximally (~3500 mL in adult males)

Functional Lung Volumes

ERV

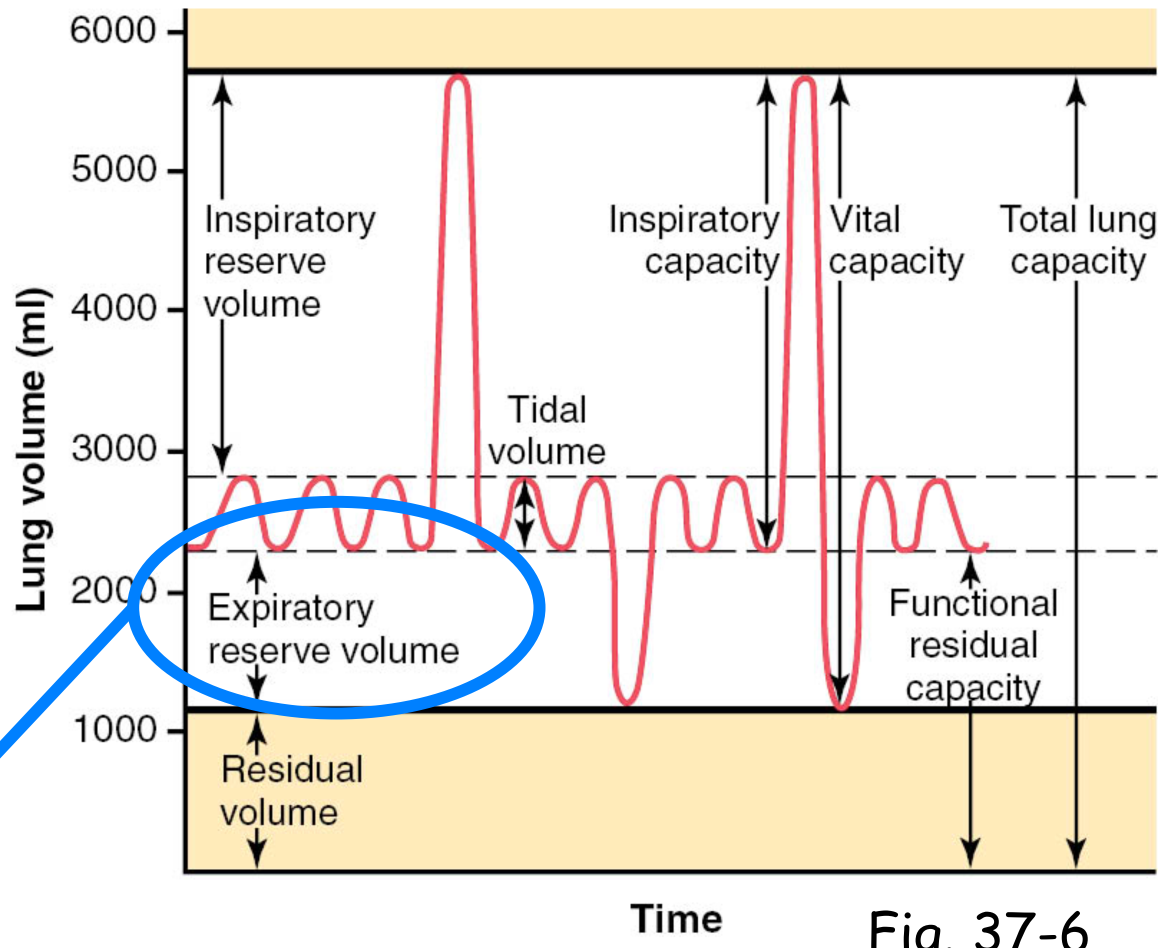


Fig. 37-6

Maximum extra vol. which can be expired by forceful expiration after end of normal tidal expiration (~1100 mL in adult males)

Functional Lung Volumes

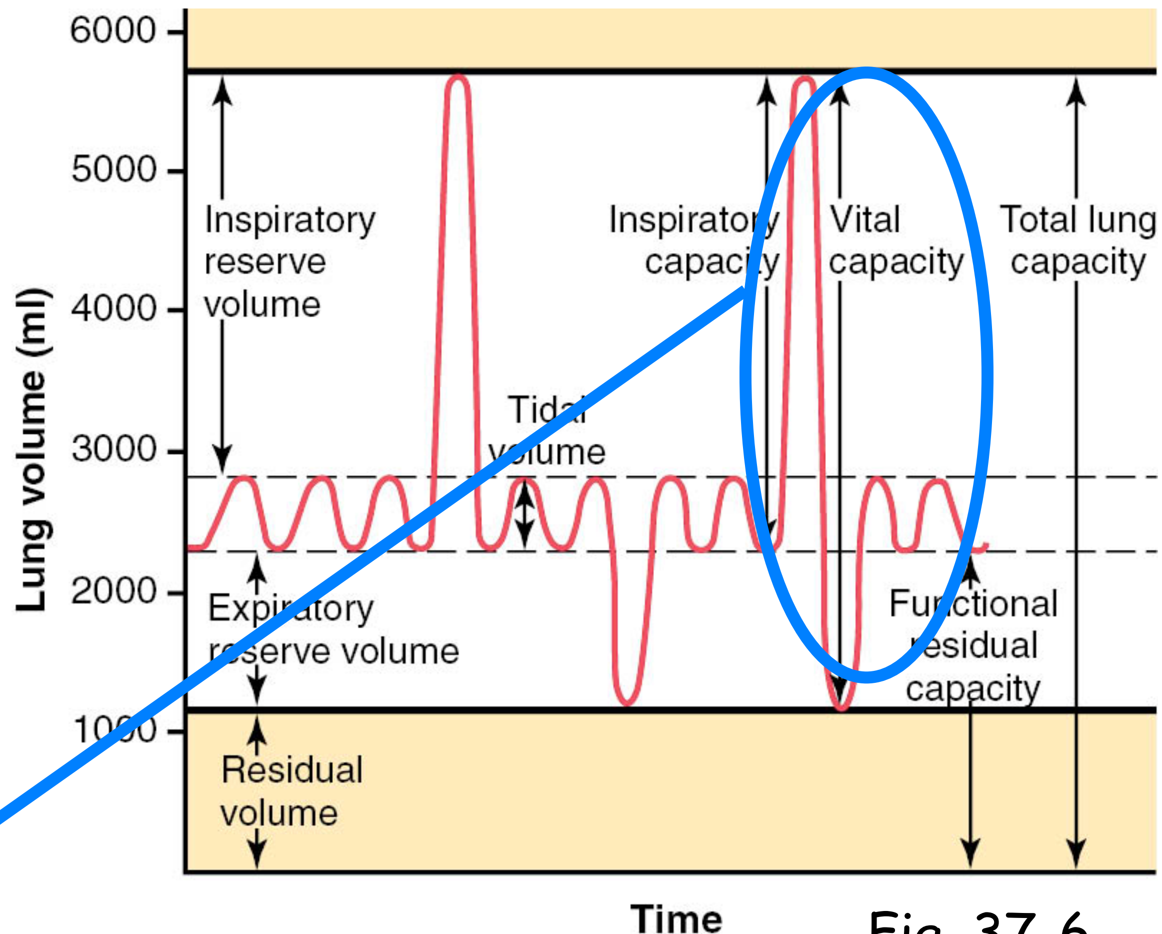


Fig. 37-6

VC

Inspiratory reserve vol. + tidal vol. +expiratory reserve vol.
Maximum amount which can expel from lungs after filling maximally and expiring maximally (~4600 mL in adult males)

Functional Lung Volumes

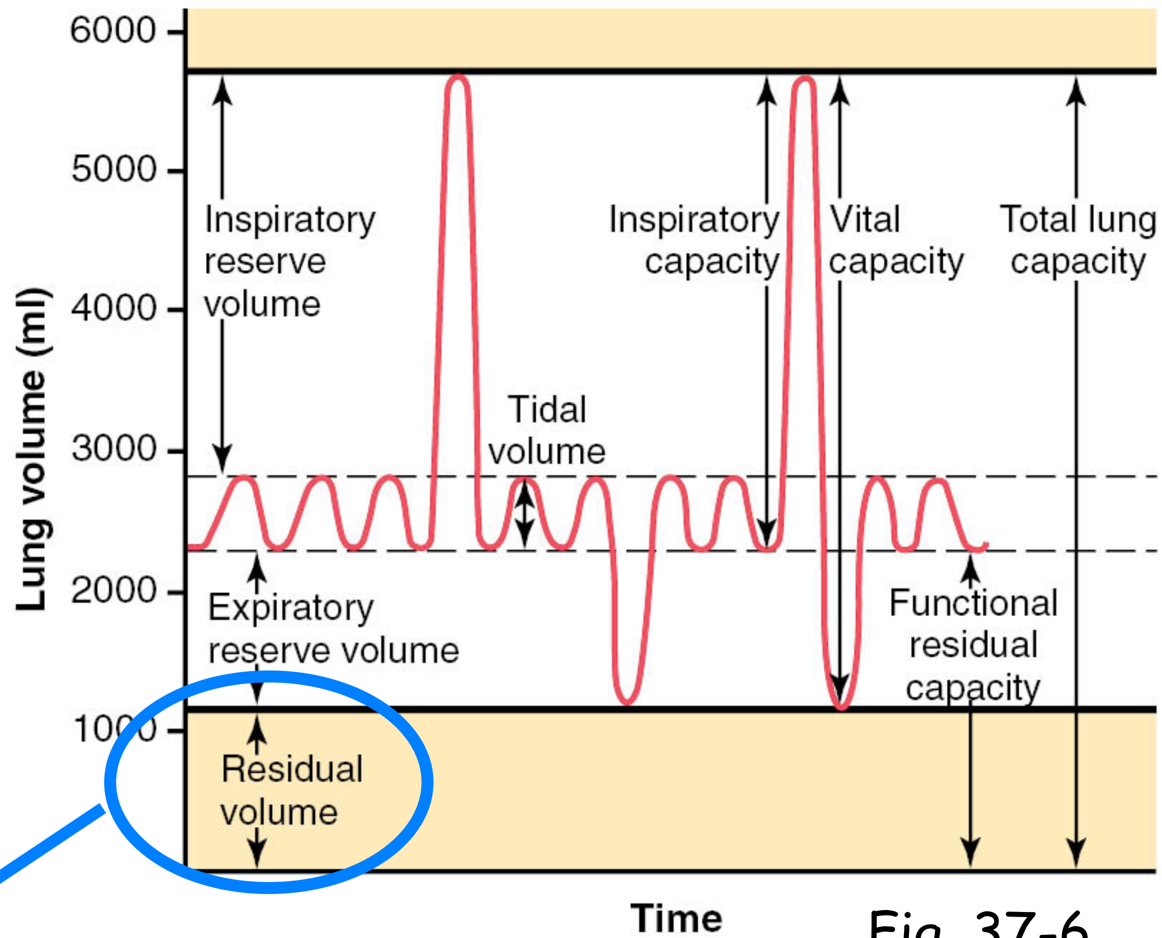


Fig. 37-6

RV

Vol. remaining in lungs after the most forceful expiration (~1200 mL in adult males)

Functional Lung Volumes

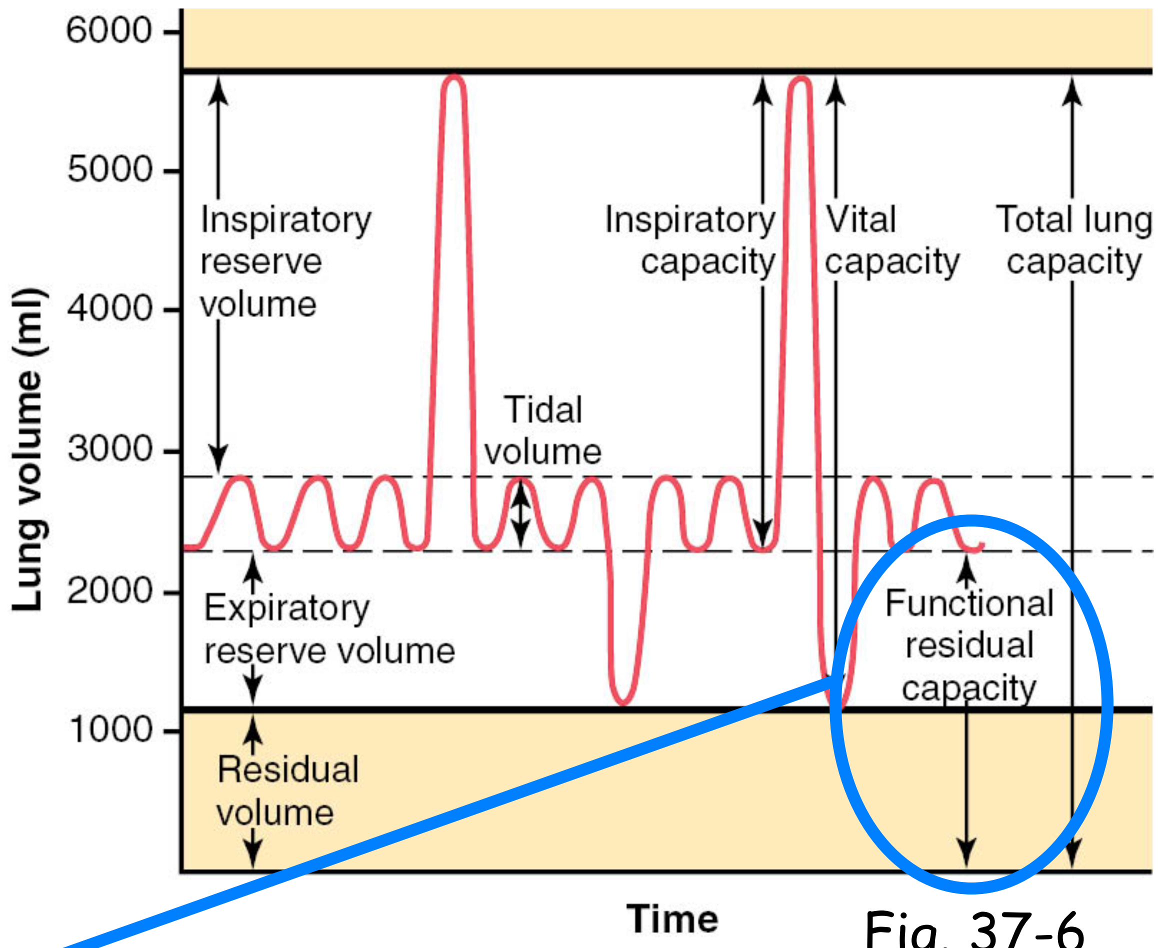


Fig. 37-6

FRC

Expiratory reserve volume + residual volume

Amount that remains in lungs at end of normal expiration

(~2500 mL in adult males)

Functional Lung Volumes

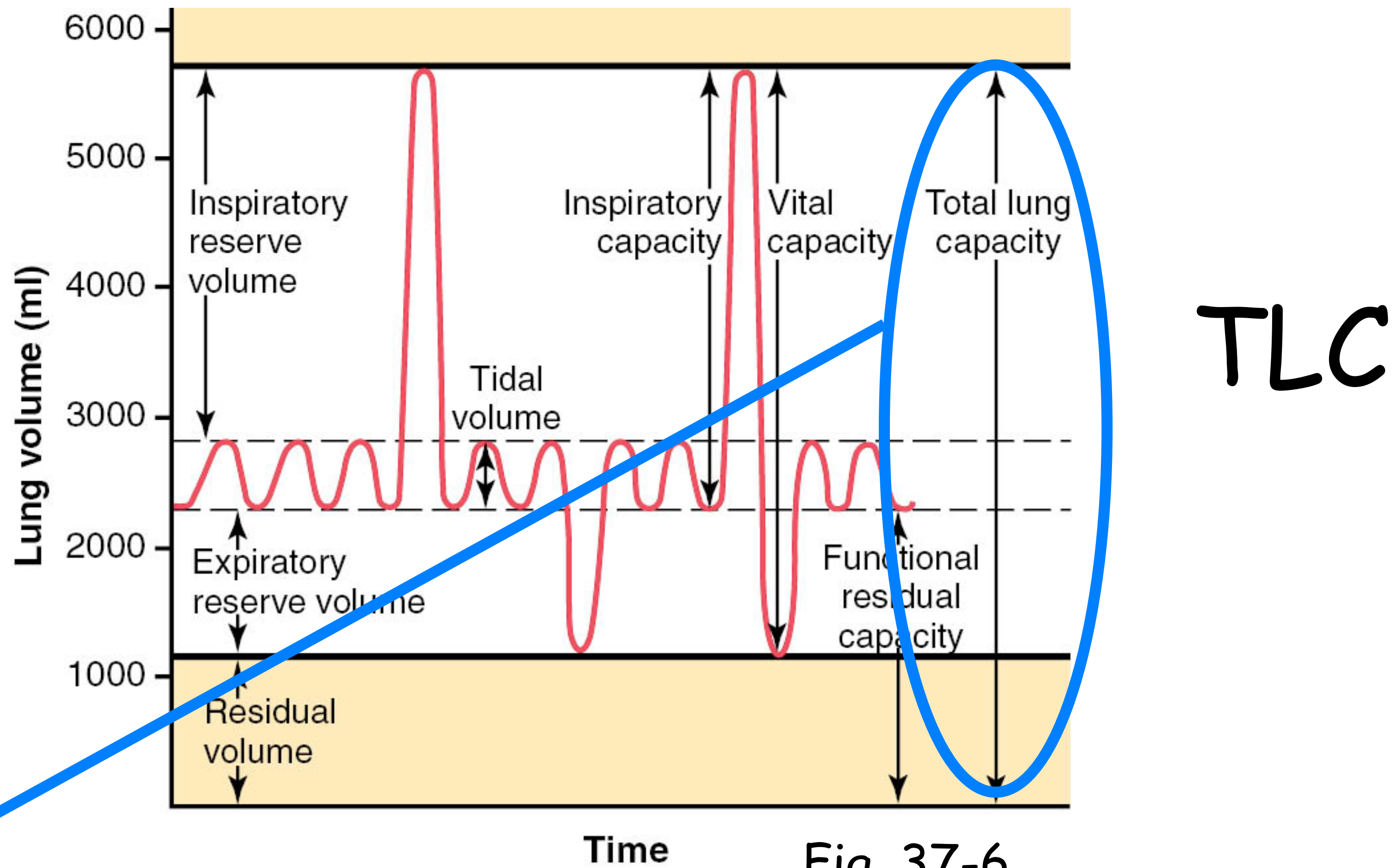


Fig. 37-6

Vital capacity + residual volume

Max. vol. lungs can be expanded with greatest possible effort (~5800 mL in adult males)

Functional Lung Volumes

<u>Minute Ventilation</u> = total air flow into (and out of) the respiratory system per minute

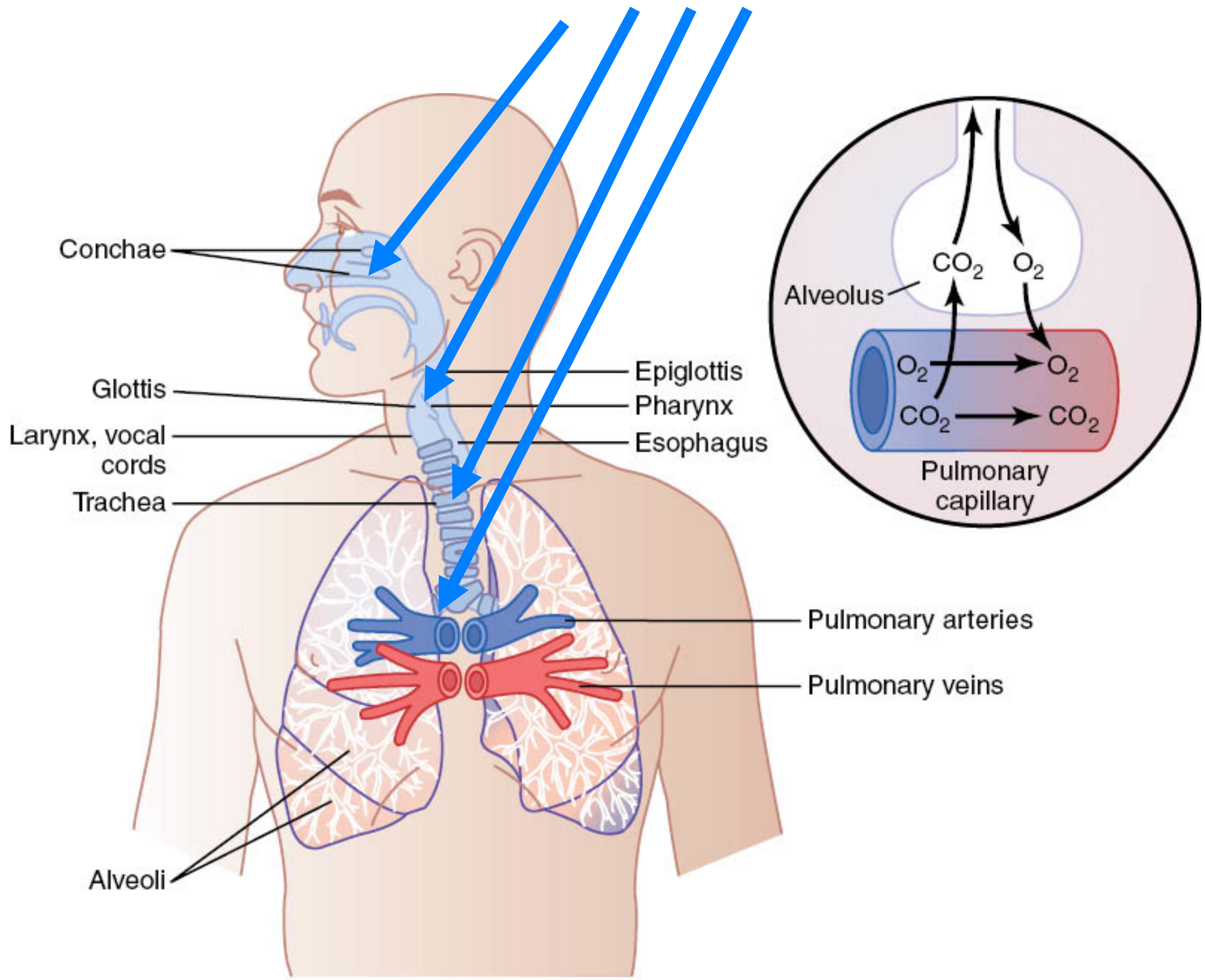
Minute Ventilation = Tidal Volume x Breathing Rate

e.g. 7500 ml/min = 500 ml x 15/min

Minute Ventilation ≠ Alveolar Ventilation ?

This difference is because of <u>Anatomic Dead Space</u> which is air "stuck" in areas where gas exchange does not occur

"Dead Space"



Anatomical dead space volume approx. 150 mL

Functional Lung Volumes

Minute Ventilation = total air flow into (and out of) the respiratory system per minute

Minute Ventilation = Tidal Volume x Breathing Rate

e.g. 7500 ml/min = 500 ml x 15/min

Minute Ventilation $\neq$ Alveolar Ventilation ?

This difference is because of Anatomic Dead Space which is air "stuck" in areas where gas exchange does not occur

Alveolor Ventilation (V_A):

V_A = [Tidal Volume - Anat. Dead Space] x Breathing rate

5250 ml/min = [500 ml - 150 ml] x 15/min

Functional Lung Volumes

350 ml of "new" air is entering the 2500 ml functional residual capacity which contains "old" air

Functional Lung Volumes

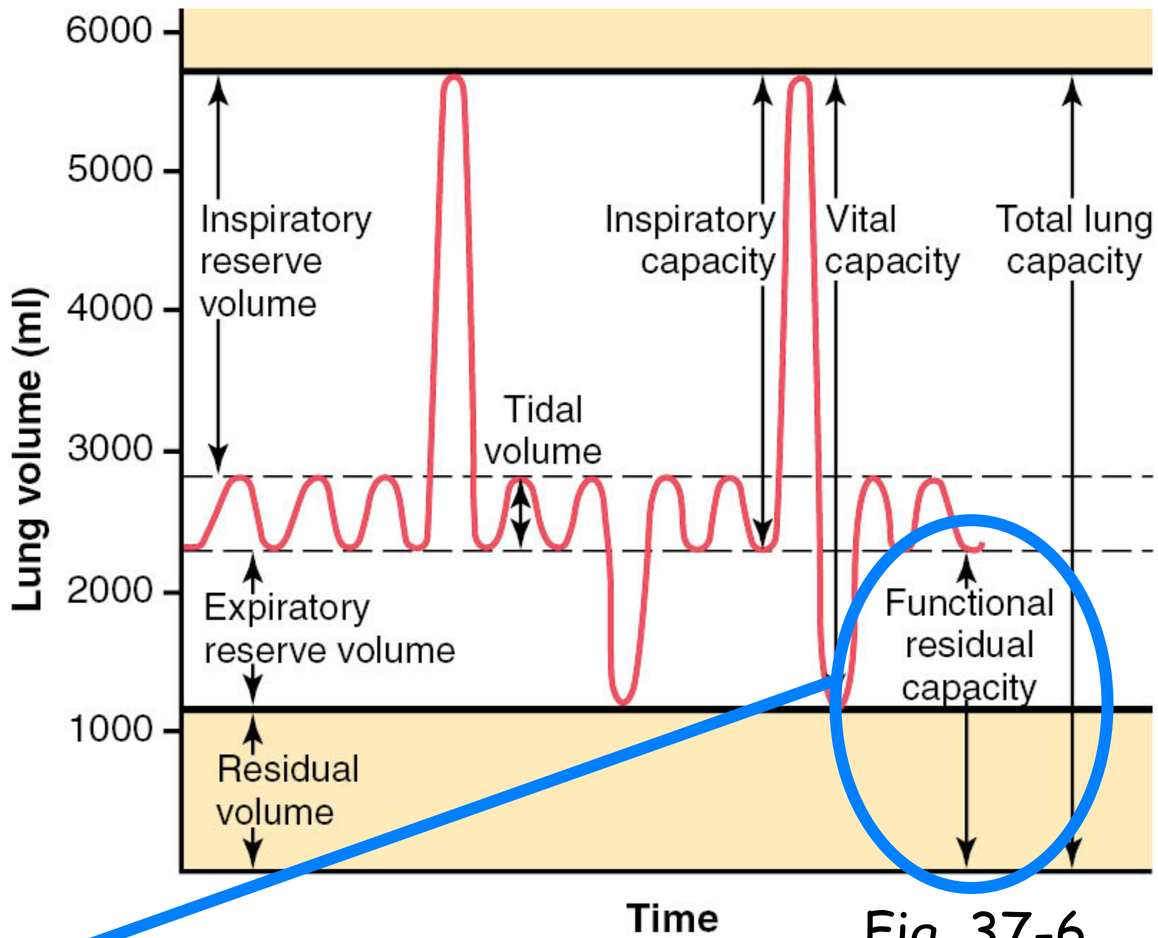


Fig. 37-6

FRC

Expiratory reserve volume + residual volume

Amount that remains in lungs at end of normal expiration (~2500 mL in adult males)

Functional Lung Volumes

350 ml of "new" air is entering the 2500 ml functional residual capacity which contains "old" air

$$\text{A quick calculation} = \frac{350 \text{ ml "new"}}{350 \text{ ml "new"} + 2500 \text{ ml "old"}}$$

Therefore, only ~ 12% replacement of alveolar air per breath at rest

Other factors affect ventilation

- Two other factors play important roles in ventilation:
  - Resistance within the airways (hindrance of airflow)
  - Lung compliance, which is the ease with which the lungs expand

Resistance within the airways

- Air flowing into the lungs encounters resistance as gas molecules strike the walls of the airway
- Thus, resistance is influenced by airway diameter
- Branchiole constriction =↑ resistance, ↓ airflow
- Branchiole dilation = ↓ resistance, ↑ airflow

Factors influencing airway resistance

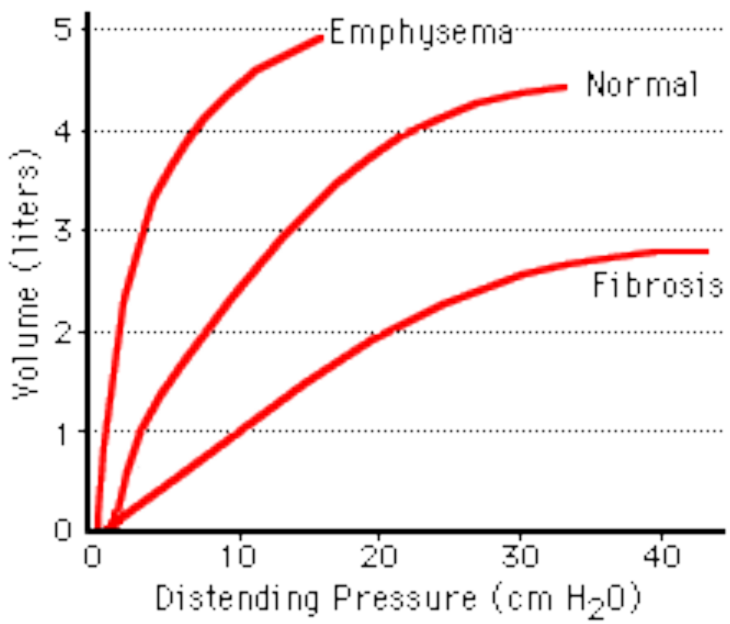
- Factors affect airway diameter by regulating bronchiole smooth muscles
    - Parasympathetic neurons release acetylcholine leading to bronchiole constriction
    - Sympathetic action on adrenal gland medullae release epinephrine in blood causing bronchiole dilation
    - Histamine (released during allergic reactions by mast cells) constricts bronchioles

Lung compliance: lung fibres

- The ease with which the lungs expand is called lung compliance
- Two factors determine lung compliance:
  - Ability of elastic fibres to stretch within the lungs
  - Alveolar surface tension

Elastic connective tissue

- Healthy lungs with abundant elastic connective tissue inflate easily with minimum pressure (have high compliance)
- In pathological conditions abnormal compliance is observed



Surface tension

- Surface tension within the alveoli also affects lung compliance

- Some premature babies have inadequate surfactant production (ie. RDS), thus increased alveolar surface tension leading to collapsed alveoli (and low compliance)

Summary

- Muscle activity regulates the volume of thoracic cavity during breathing

- Changes in thoracic volume cause alterations in intrapulmonary and intrapleural pressure

- Air moves from higher pressure to lower pressure regions

Summary

- Airway resistance is normally low, but is modulated by nervous input & humoral factors acting on smooth muscle
- This regulation leads to changes in bronchiole diameter, which alters resistance and airflow
- Lung compliance is normally high in healthy individuals since abundant elastic connective tissue
- Surfactant release by type II alveolar cells leads to decreased surface tension in alveoli