Introduction to the respiratory system pathophysiology

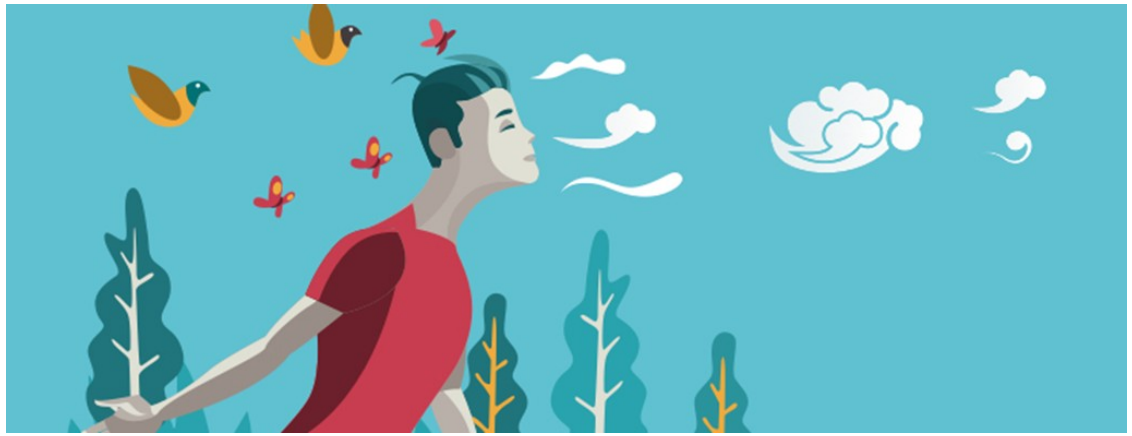
Structural and functional properties of airways and lungs
- defence mechanisms of airways and lungs

Respiration as a process ensuring a gas exchange
- ventilation & diffusion & perfusion

Diffusion – principles and determinants
- alveolar-capillary membrane
- „oxygen cascade“

Lung circulation – principles and determinants

Ventilation – pulmonary mechanics
- volumes and capacities
- static and dynamic airflow resistance
- dynamic collapse
- obstruction vs. restriction



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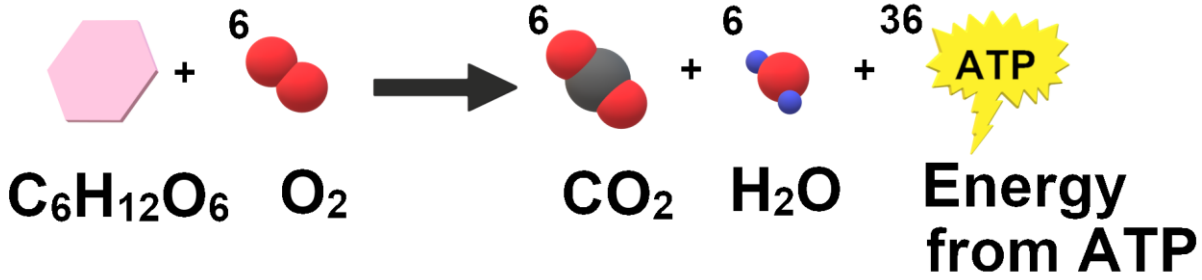
Warming up questions

- (1) **WHY** do we breathe???



- (2) **HOW** do we breathe???
  - principles of the quiet breathing

- (3) **WHEN** do we breathe???
  - all the time/non-stop, the death = „until the last breath“

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STRUCTURAL-FUNCTIONAL CONSIDERATIONS IMPORTANT FOR PP OF RESPIRATION & PARTICULAR DISORDERS

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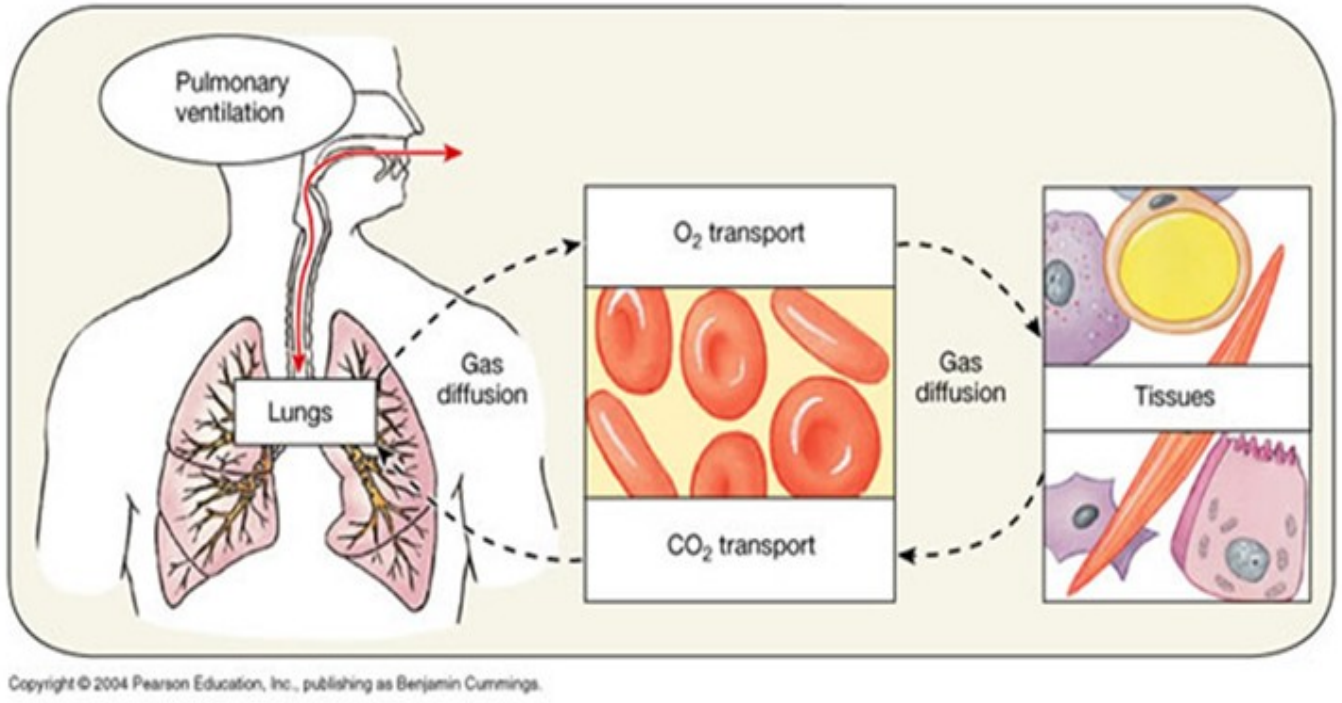
Respiration and gas exchange in the lungs

- **ventilation** = mechanical process
  - breathing in narrower meaning
- **diffusion** = chemical process
  - through alveolo-cappilary barrier
- **perfusion** = circulatory process
  - circulation of blood in lungs

death from lung disease is almost always due to an inability to overcome the **altered mechanical properties** of the lung or chest wall, or both



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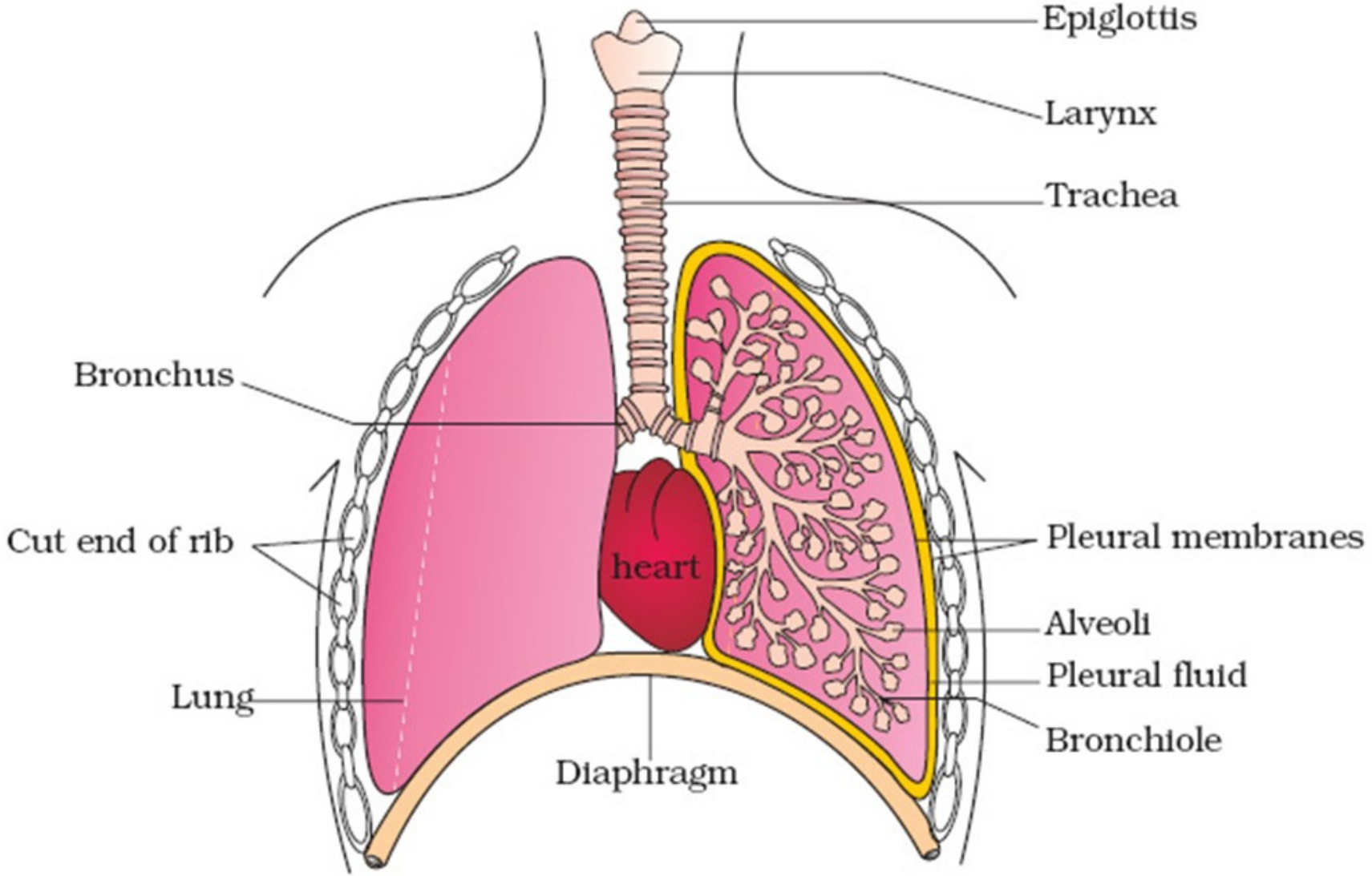
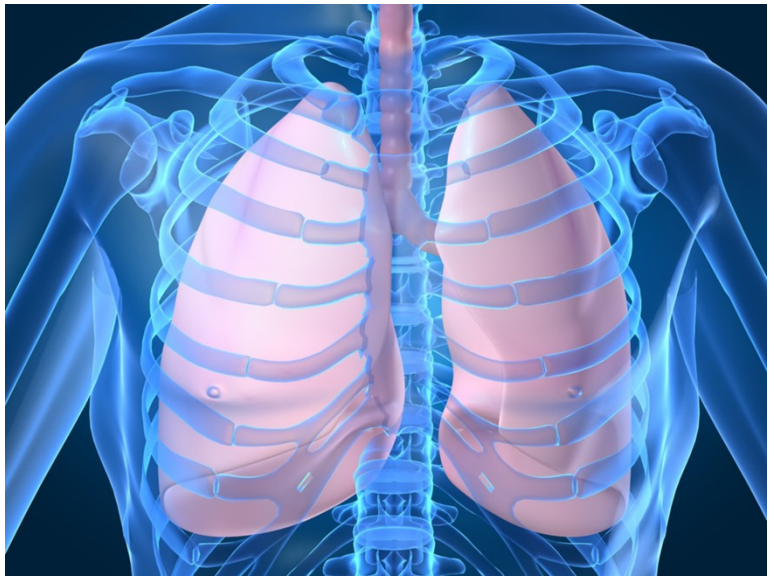


Figure 17.1 Diagrammatic view of human respiratory system (Sectional view of the left lung is also shown)

The delicate structure-function coupling of lungs

- The main role of the respiratory system is **GAS EXCHANGE**, i.e. **extraction of oxygen from the external environment** and **disposal of** waste gases, principally **carbon dioxide**
  - at the end of deep breath 80% of lung volume is air, 10% blood and 10% tissue
    - lung tissue spreads over an enormous area !
- The lungs have to provide
  - a **large surface area** accessible to the environment (~tennis court area) for gas exchange
  - alveoli walls have to present **minimal resistance to gas diffusion**
- Close contact with the external environment means lungs can be damaged by dusts, gases and infective agents
  - **host defense** is therefore a key priority for the lung and is achieved by a combination of structural and immunological means



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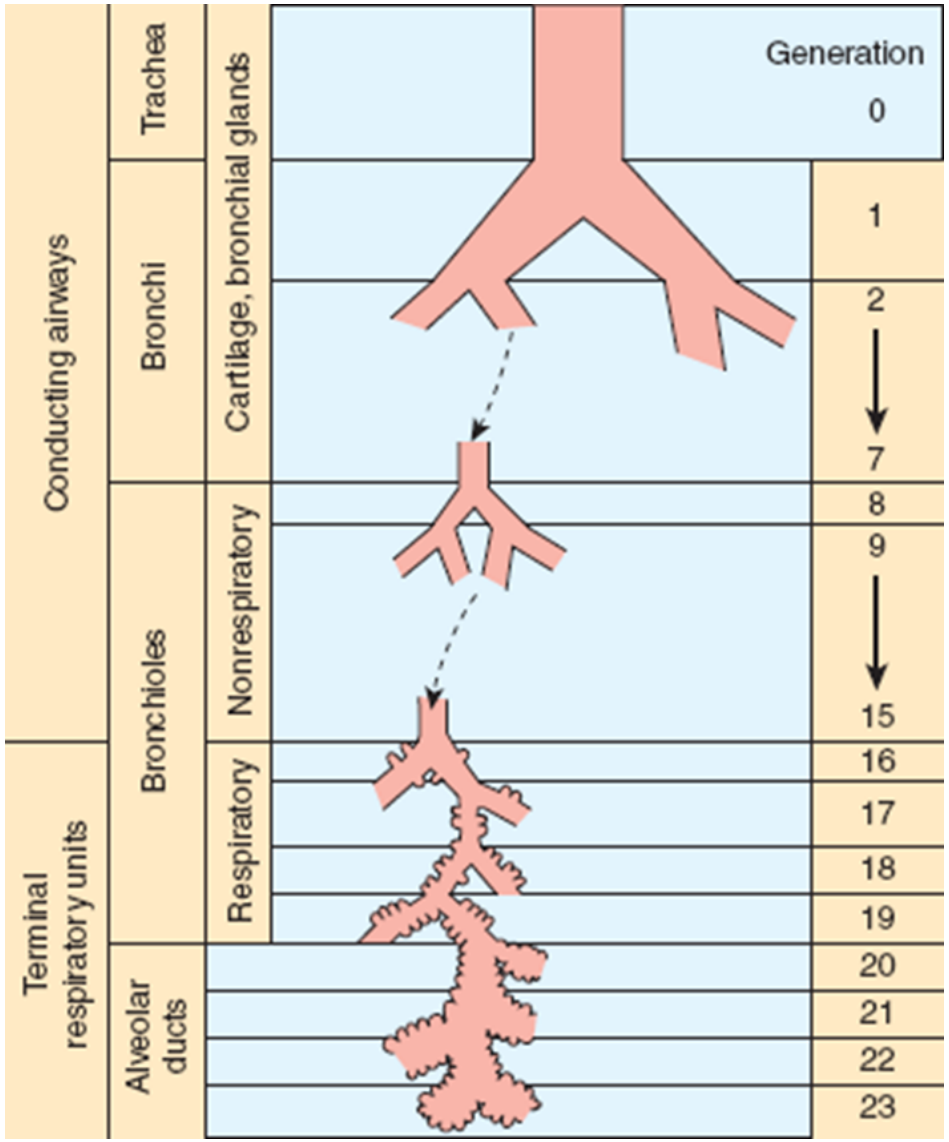
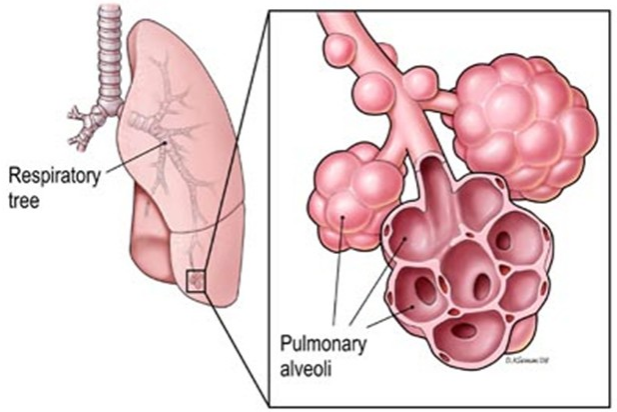
Structure of airways



- There are about 23 (18-30) divisions (2^{23} i.e. approx. 8 millions of sacs) between the trachea and the alveoli
  - the first seven divisions, the bronchi have:
    - walls consisting of cartilage and smooth muscle
    - epithelial lining with cilia and goblet cells
    - submucosal mucus-secreting glands
    - endocrine cells - Kulchitsky or APUD (amine precursor and uptake decarboxylation) containing 5-hydroxytryptamine
  - the next 16-18 divisions the bronchioles have:
    - no cartilage
    - muscular layer progressively becomes thinner
    - a single layer of ciliated cells but very few goblet cells
    - granulated Clara cells that produce a surfactant-like substance



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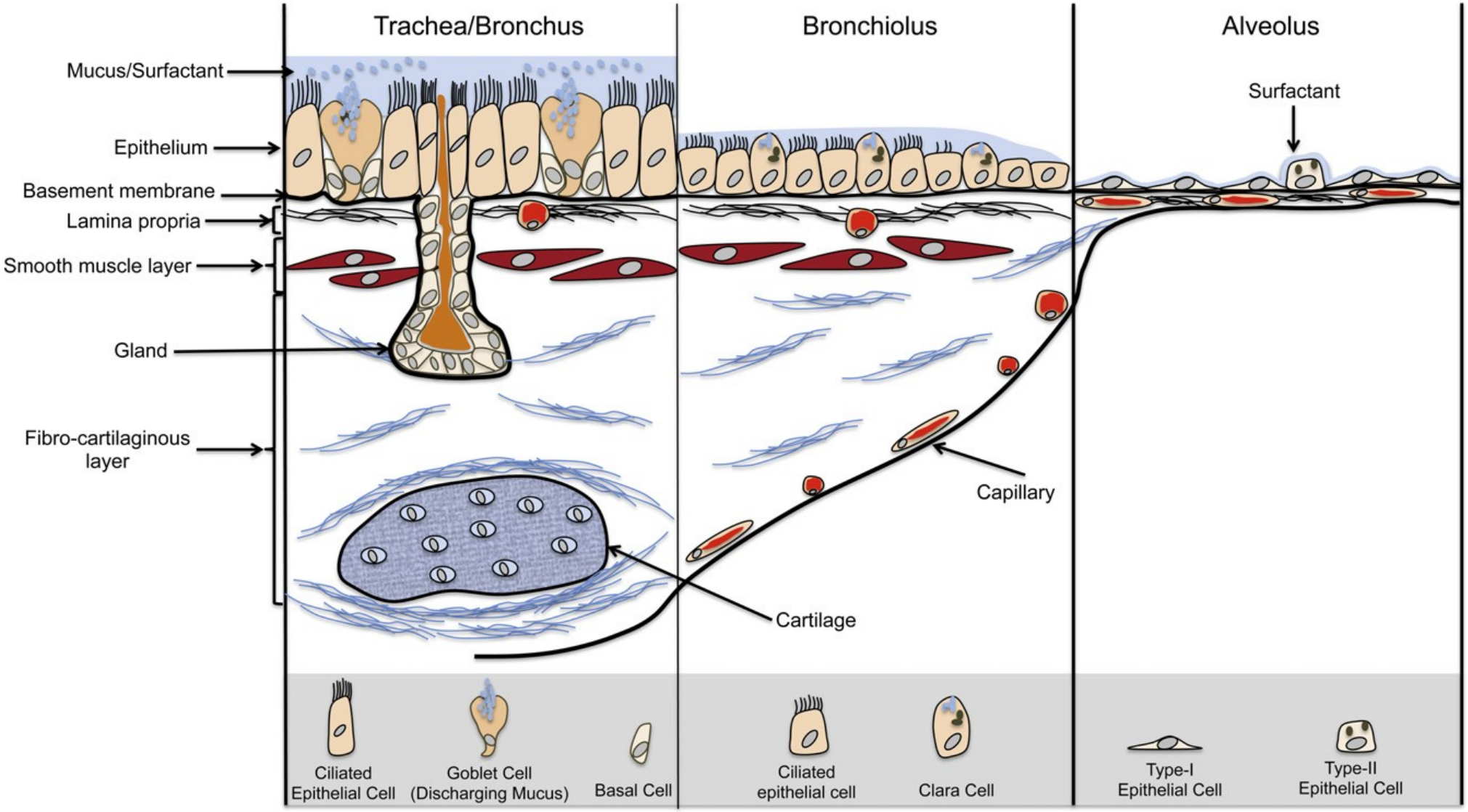
Wall structure of conducting airways and respiratory region



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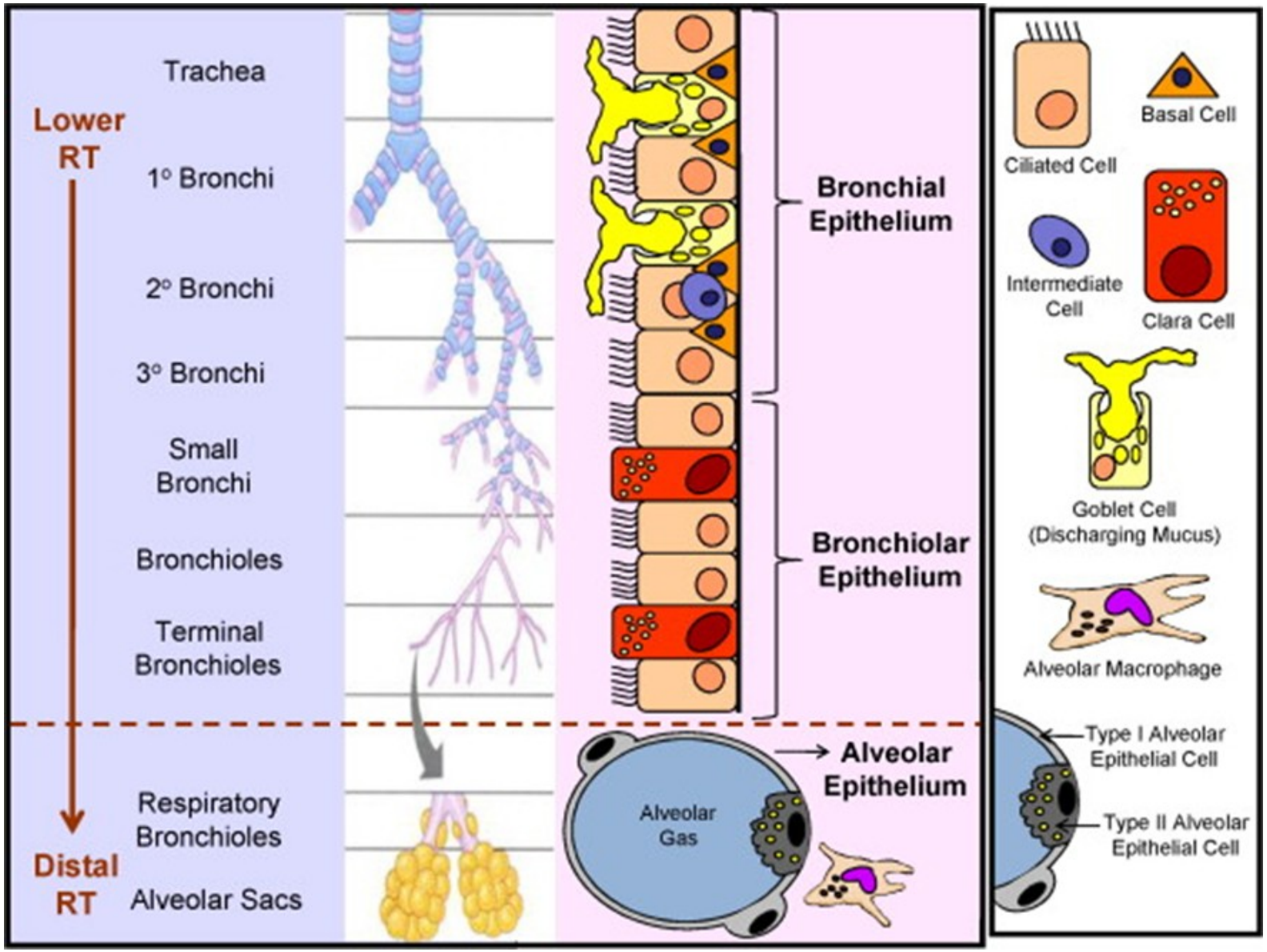
Lung defense – multiple mechanisms (details later)



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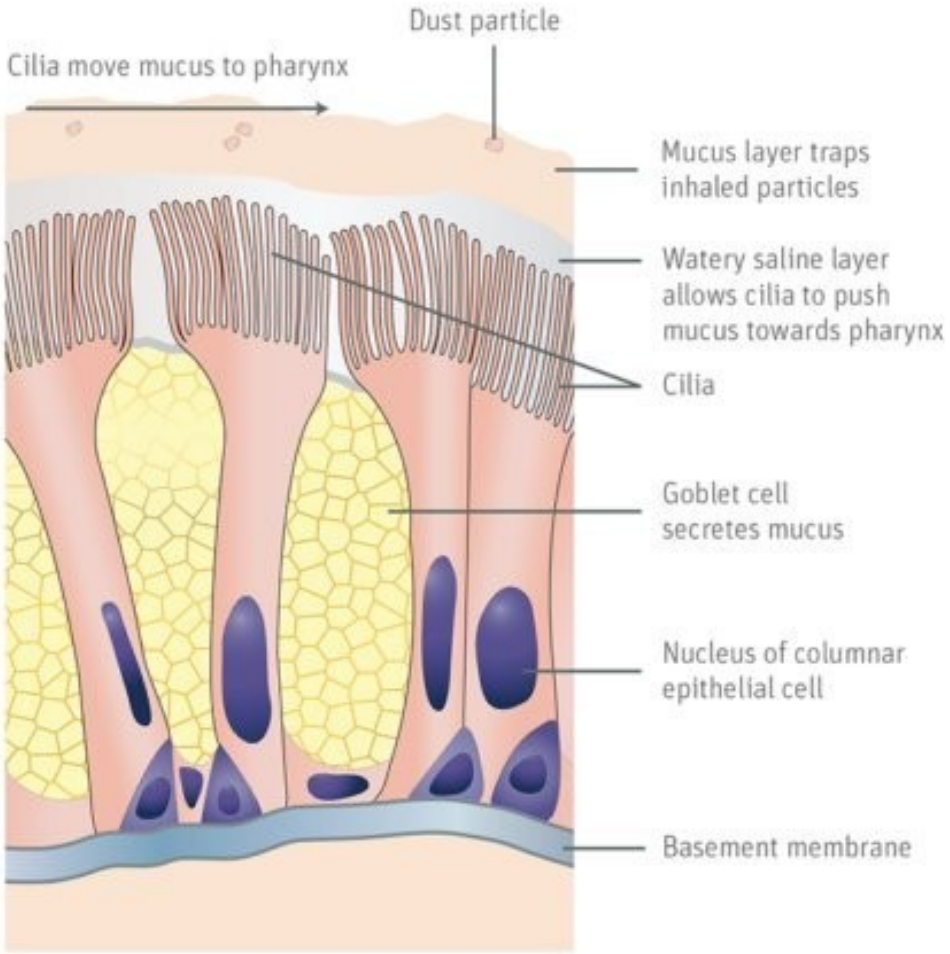
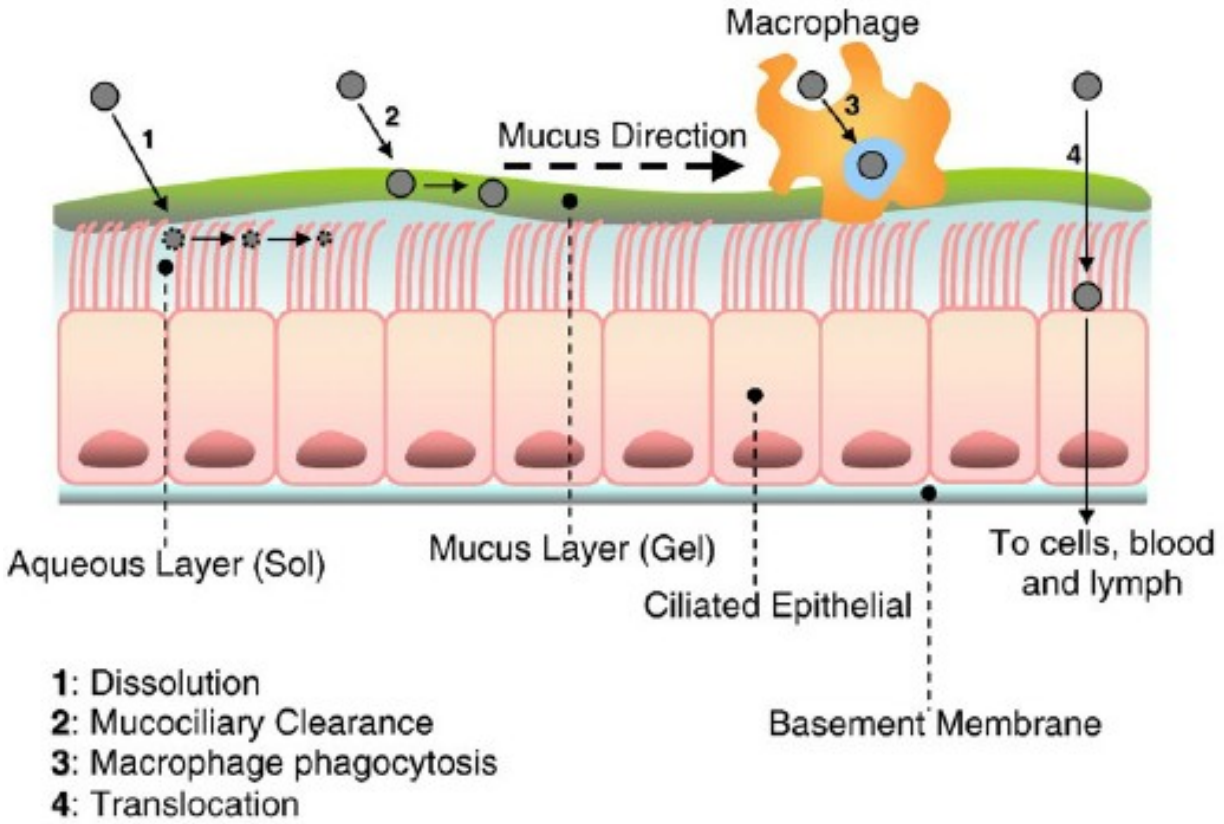
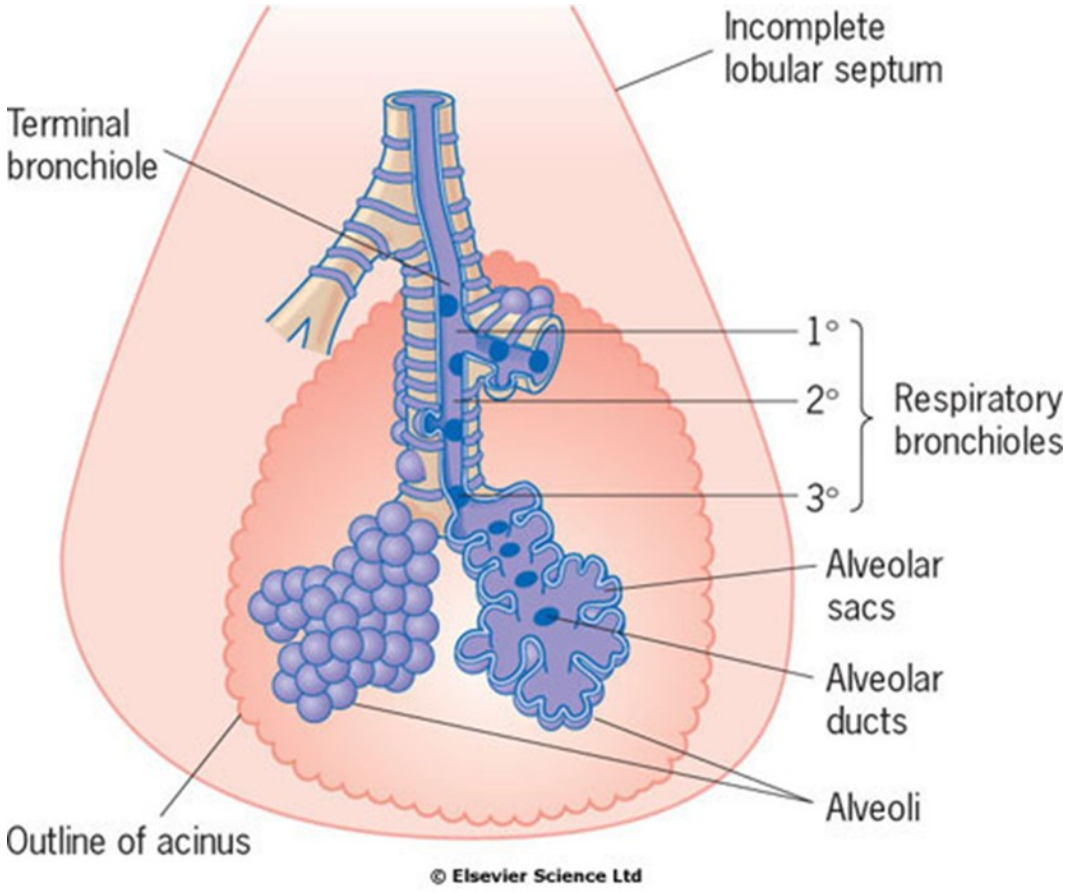
Mucocilliary escalator





Functional classification of airways

- Conducting airways (= **anatomical dead space**)
  - nose (mouth)
  - larynx
  - trachea
  - main bronchi & bronchioles
  - gas conduction, humidification & warming, defense
- Acinar airways (= **respiratory space**)
  - respiratory bronchioles
  - alveolar ducts & sacs
  - alveoli
  - gas exchange
- The concept of acinus
  - the functional 3-D unit - part of parenchyma - in which all airways have alveoli attached to their wall and thus participating in gas exchange



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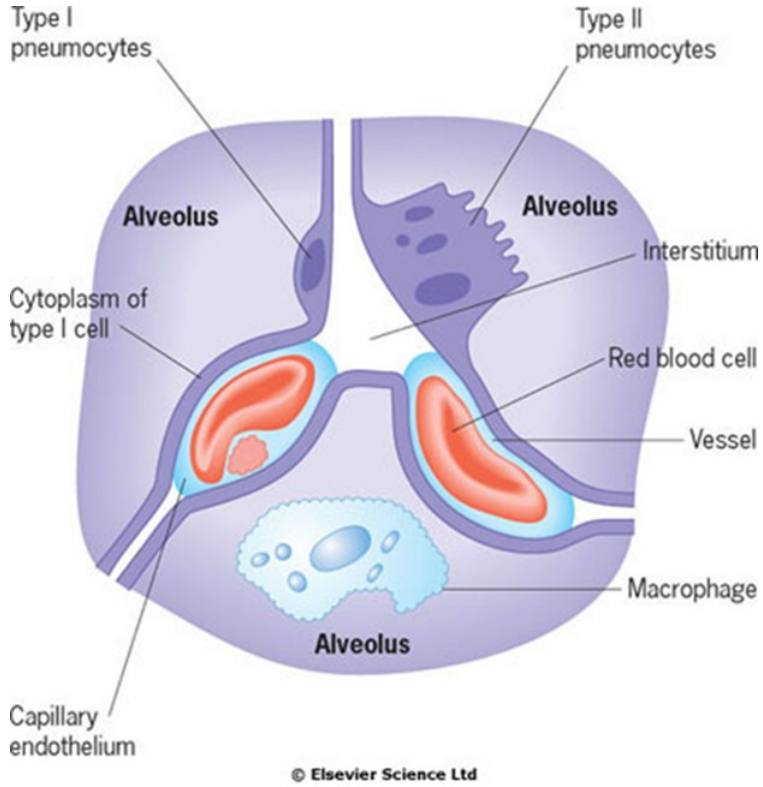
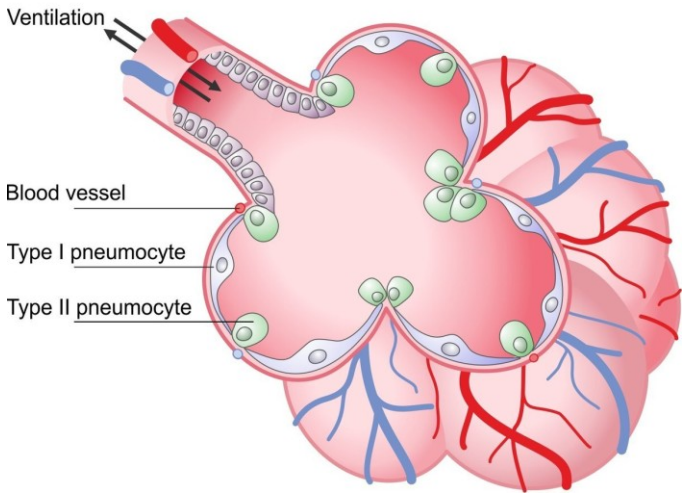
Alveoli

- There are approximately 300-400 million alveoli in each lung with the total surface area is 40-80m^2
- Cell types of the epithelial lining
  - **type I pneumocytes**
    - an extremely thin cytoplasm, and thus provide only a thin barrier to gas exchange, derived from type II pneumocytes
    - connected to each other by tight junctions that limit the fluid movements in and out of the alveoli
    - easily damageable, but cannot divide!
  - **type II pneumocytes**
    - slightly more numerous than type I cells but cover less of the epithelial lining
    - the source of type I cells and surfactant
  - **macrophages**



Alveoli



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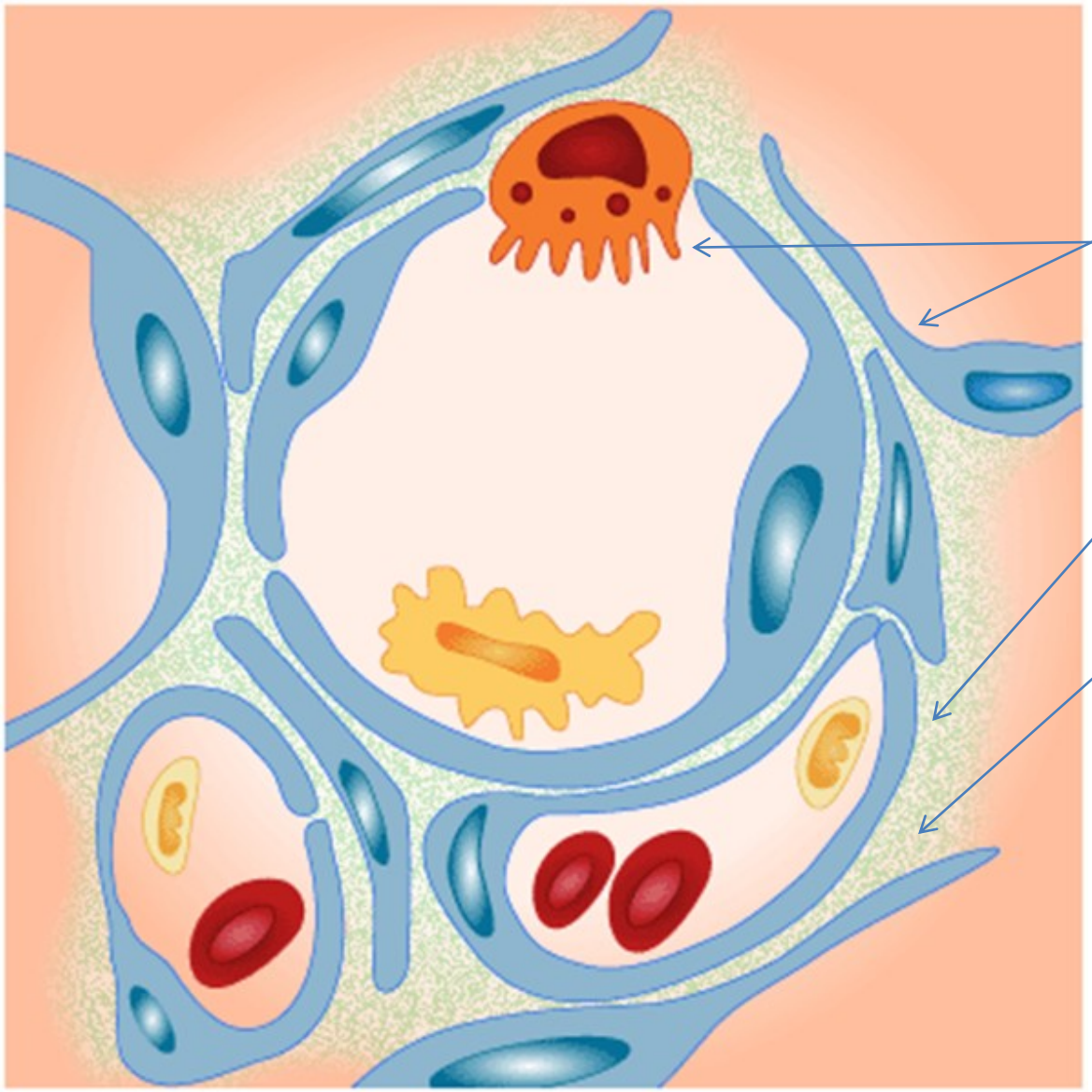
Alveolo - capillary barrier



- Alveolar epithelia
  - type I
  - type II
- Capillary endothelium
  - non-fenestrated
- Intersticium
  - cells (very few!)
    - fibroblasts
    - contractile cells
    - immune cells (intersticial macrophages, mast cells, …)
  - ECM
    - elastin and collagen fibrils

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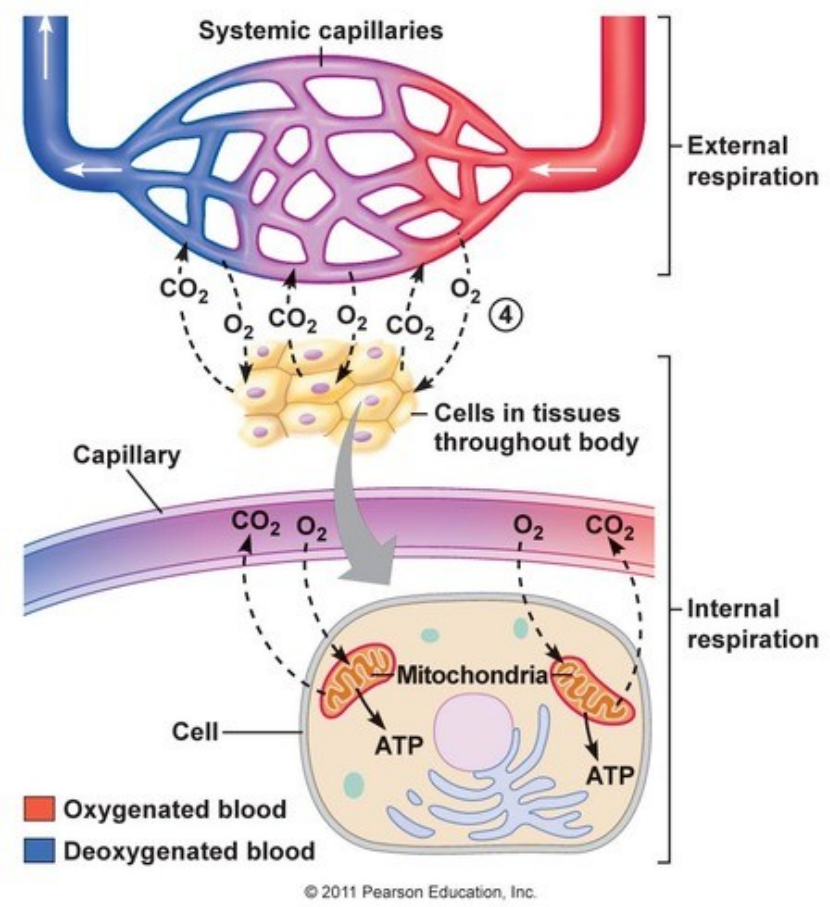
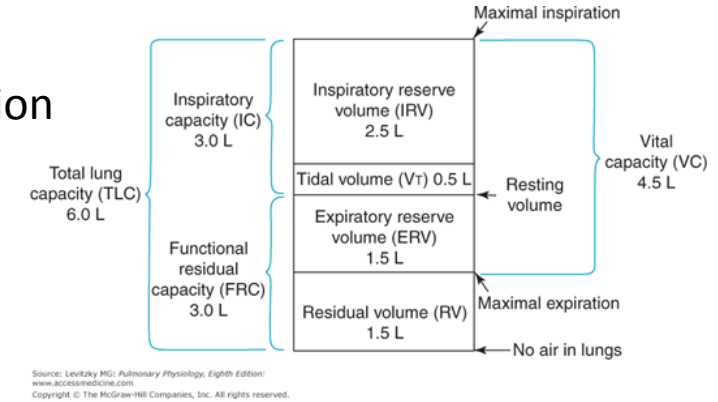
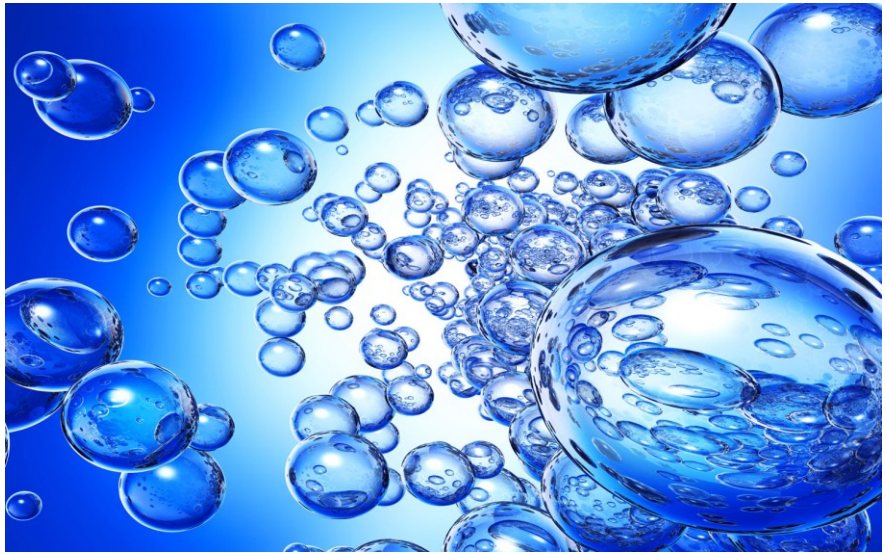
Gas exchange in lungs



- main function of respiratory system – **gas exchange between blood and outside environment** – is governed by temporally changing requirements of organism for O_2
  - maintained in optimum by regulation of intensity of ventilation (see control of ventilation further)
- requirements defined mainly by consumption of **ATP** and its replenishing by **mitochondria**
  - oxidative phosphorylation
  - other O_2 consuming processes
- driving force for O_2 exchange (and reciprocally for CO_2) is the gradual decrease of its partial pressure, i.e. **concentration gradient** between inhaled air, blood and tissues:
  - partial pressure = the pressure that the gas would have if it alone occupied the same volume at the same temperature
- solubility of the gas matters
  - very high for CO_2 = there are no biological barriers in the body to block CO_2 diffusion
- tidal volume exchange by each resting breathing cycle ads only 0.5L to FRC = meaning a composition of the alveolar air is more or less constant





OXYGEN CASCADE IN THE BODY

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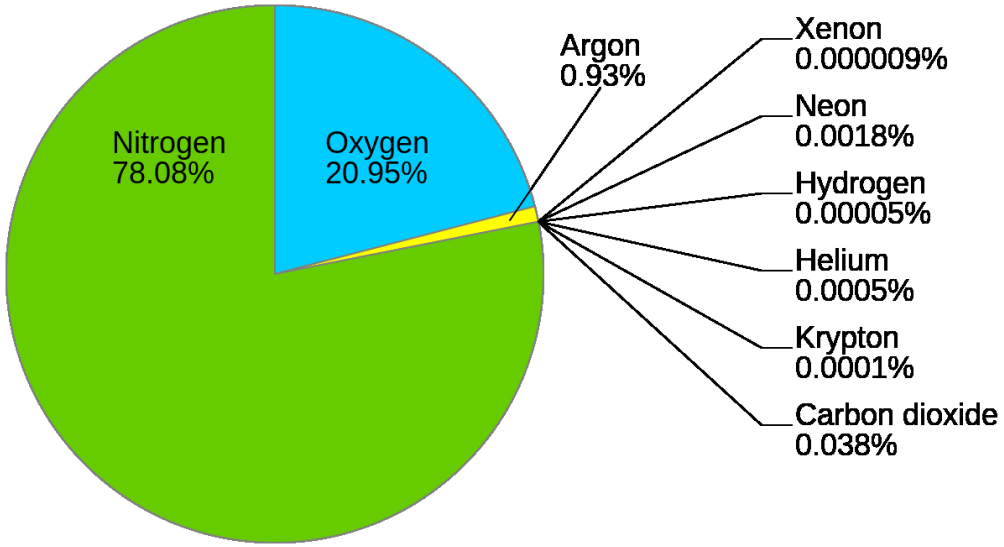
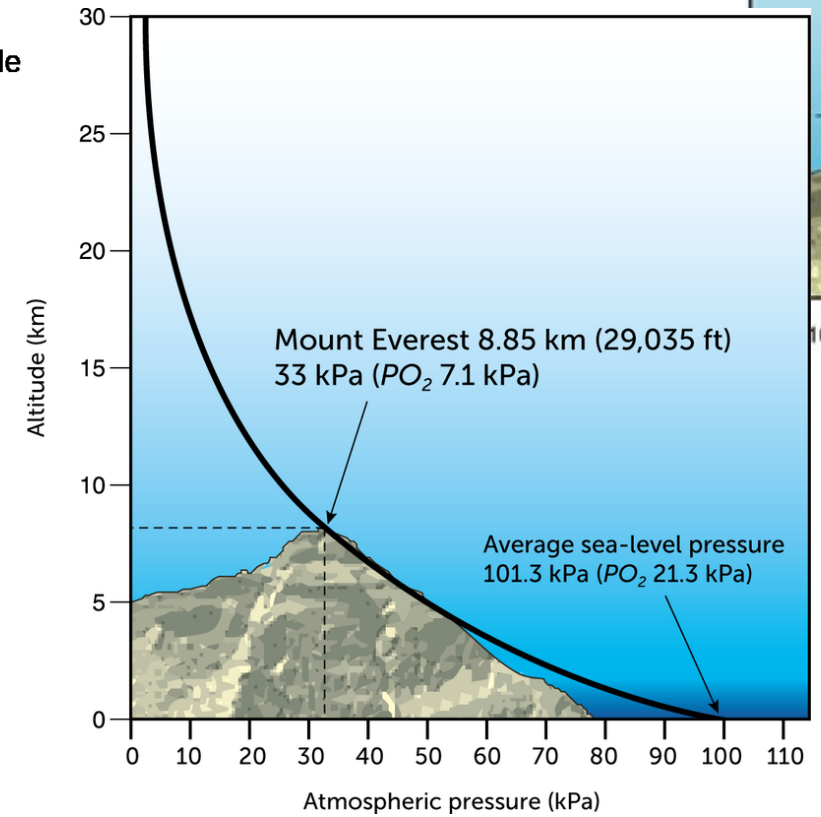
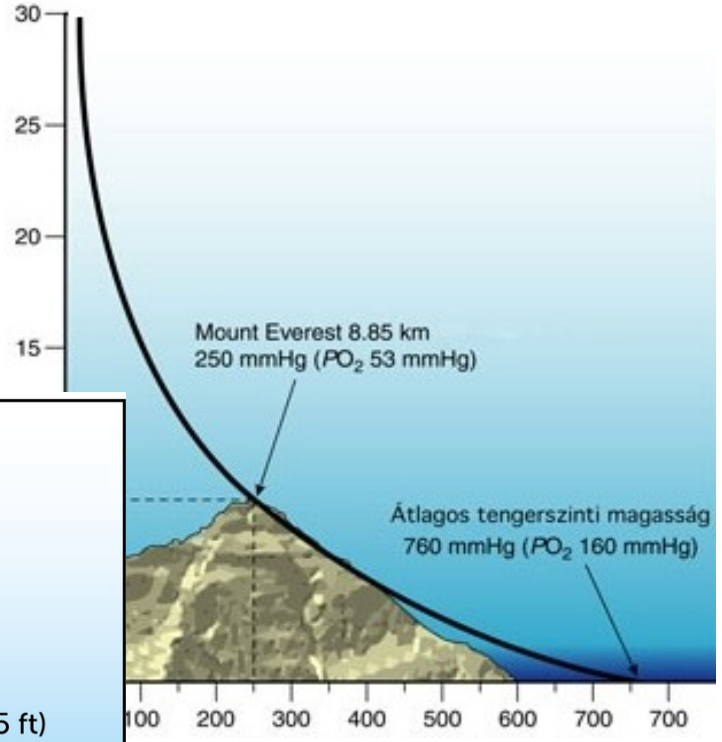
What are we breathing?







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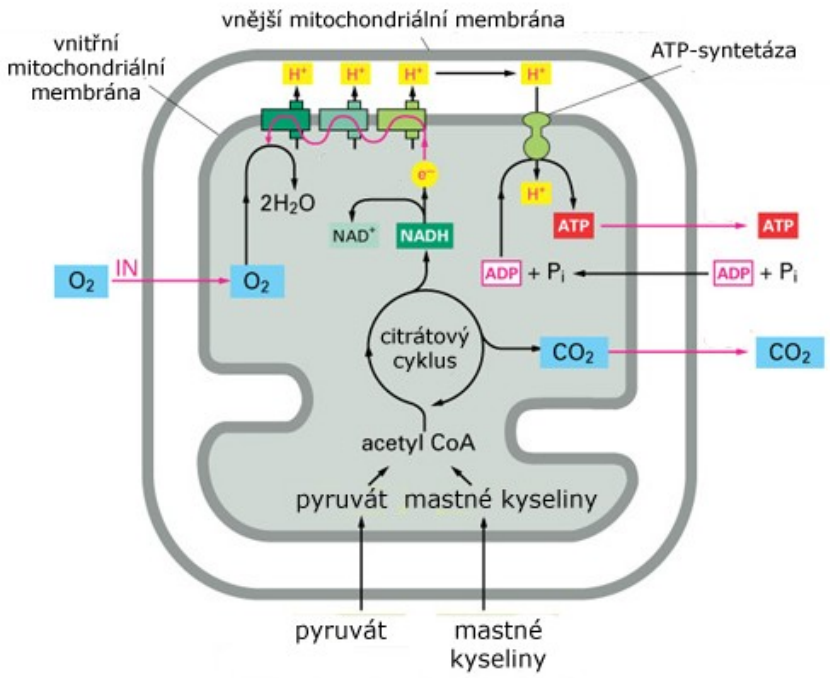
Oxygen in the body



- there are no significant O_2 stores in the body
  - available oxygen lasts for ~ 5min
  - therefore breathing has to be continuous process
  - disruption means
    - life-threatening emergency (<5min)
      - reversible vision loss in ~7s, unconsciousness in ~10s
    - clinical death (~5-7min), event. brain death
    - death of the whole organism (>10min)
- 85-90% used in aerobic metabolism coupled with ATP production
  - maintenance of ion gradients
  - muscle contraction
  - chemical synthetic reactions
- remaining processes are less sensitive to ↓PaO_2
  - hydroxylation of steroids
  - detoxification of xenobiotics in liver
  - synthesis of NO (→ vasodilation)
  - degradation of haem by hemoxygenase

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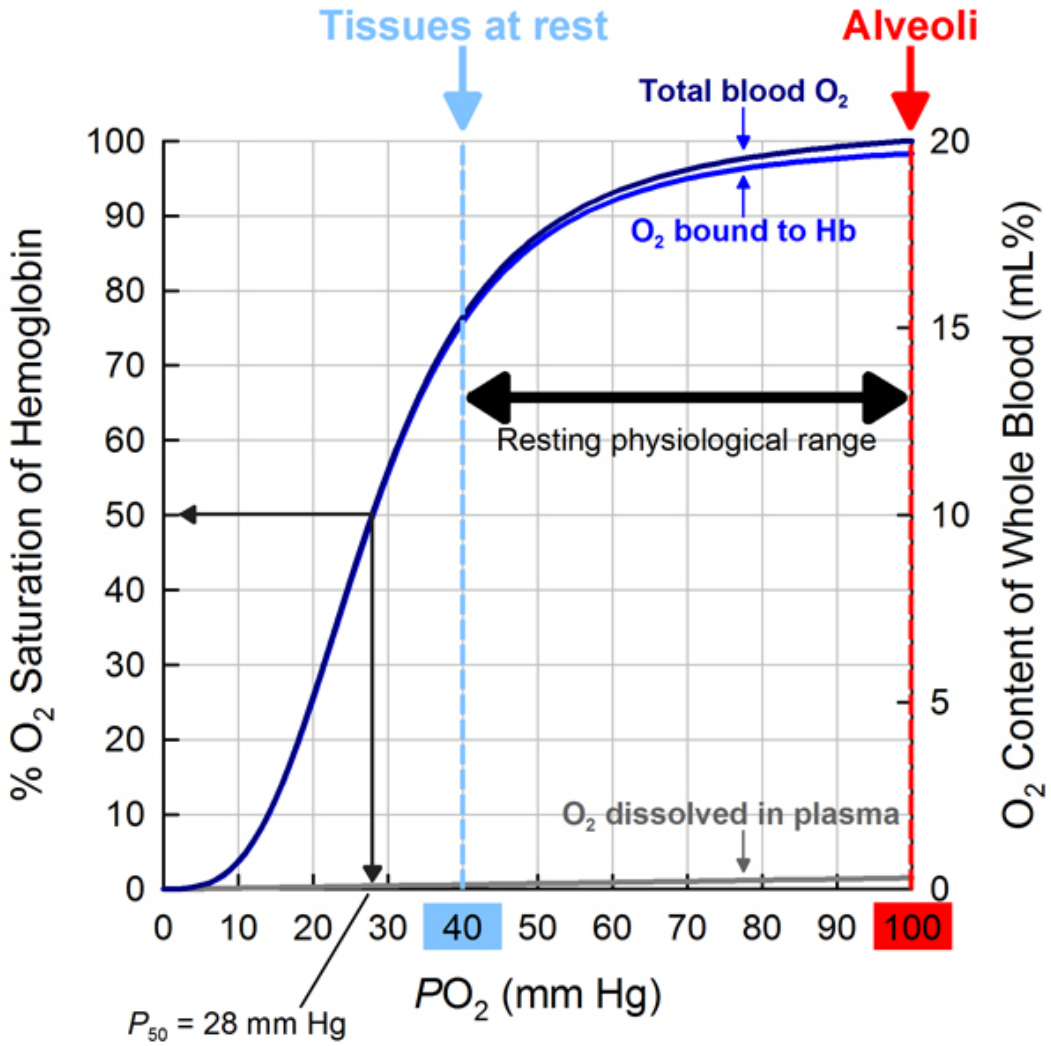
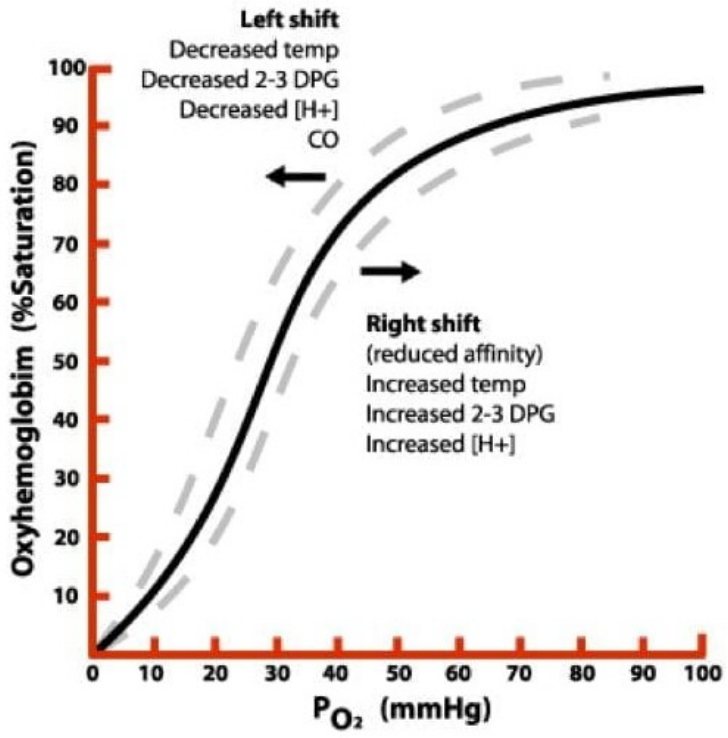
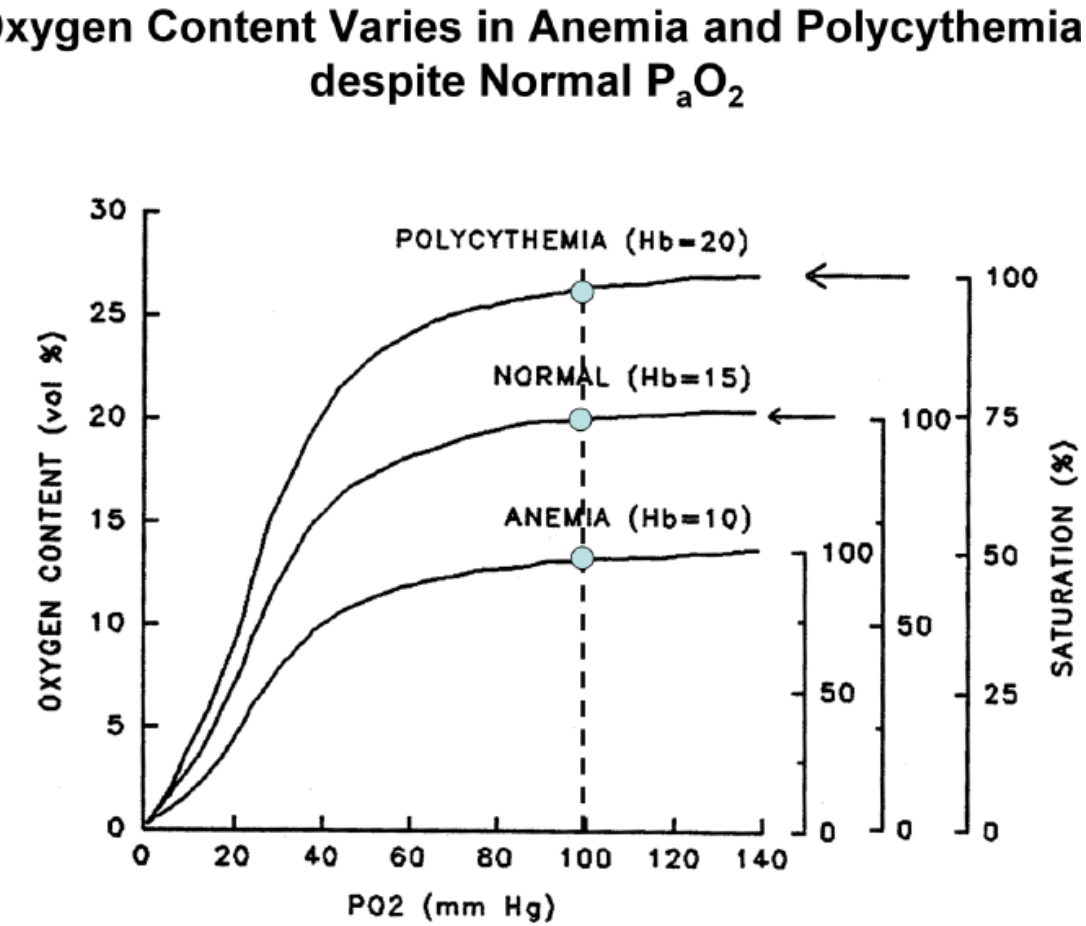
Transport of oxygen in the blood

- CO_2 can be considered to be in simple solution in the plasma, the volume carried being proportional to its partial pressure (physically dissolved)
- O_2 is carried in chemical combination with hemoglobin in the red blood cells, and the relationship between the volume carried and the partial pressure (physically dissolved fraction) is not linear
  - in physiological PaO_2 (90mmHg/12kPa) and normal hemoglobin there is nearly 100% Hb saturation
    - **if PaO2 > 10kPa/60 mmHg, saturation do not significantly decreases**
      - advantage when being in high altitude
      - saturation measured by pulsion oxymetry
- O_2 diffuses to tissues according to demands of mitochondria
  - for adequate production of ATP in mitochondria O_2 in tissues have to be > 0.13kPa/**1mmHg = critical oxygen tension**
- organism needs oxygen:
  - ~ 250 mL/min → 350 L/day in rest
  - much more (10x) during exercise
- total O_2 in the blood
  - total $[O_2] = 1.39 \times [Hb] \times \%\ saturation / 100 + 0.003 \times PO_2 = 20.5$ ml/dl



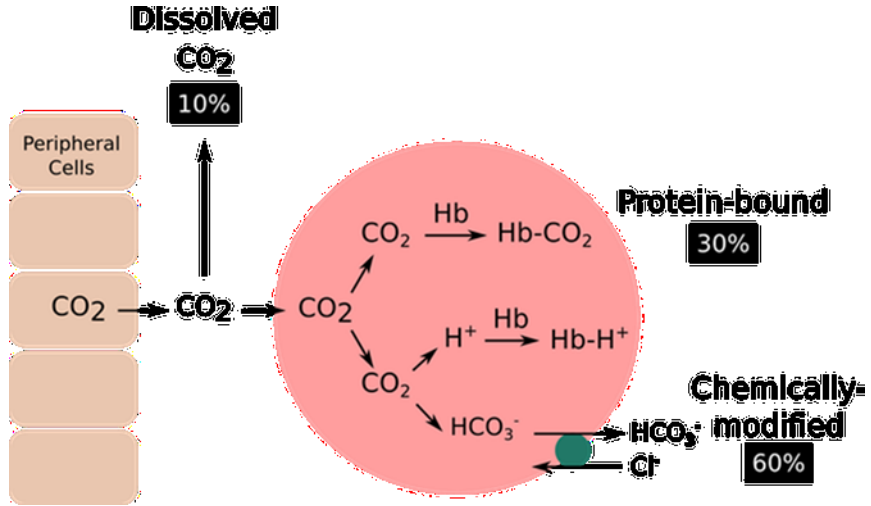
Shifting of Hb dissociation curve and the effect of [Hb]

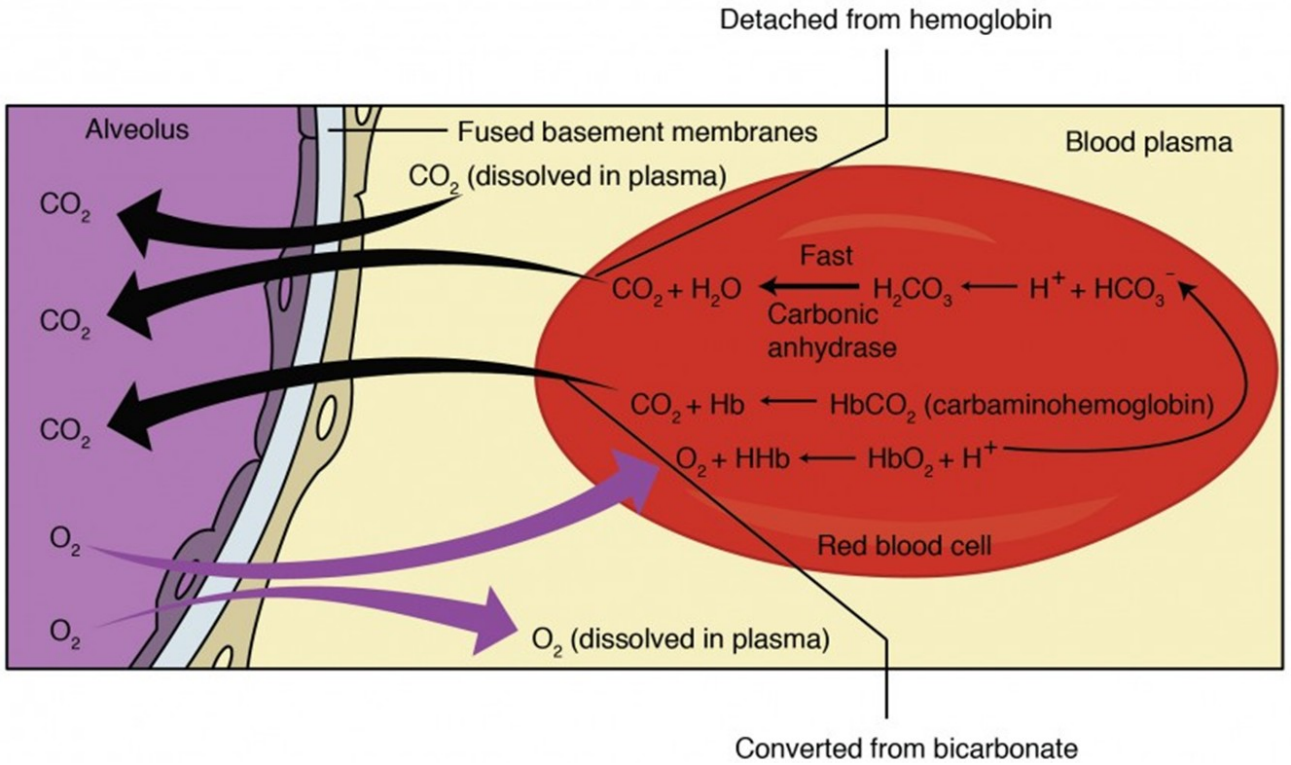




Transport of CO_2 in the blood

- CO_2 can be considered to be in simple solution in the plasma, the volume carried being proportional to its partia pressure (physically dissolved)
- solubility of carbon dioxide is much higher (20×) than that of oxygen, therefore physically dissolved CO_2 is much more important than for O2





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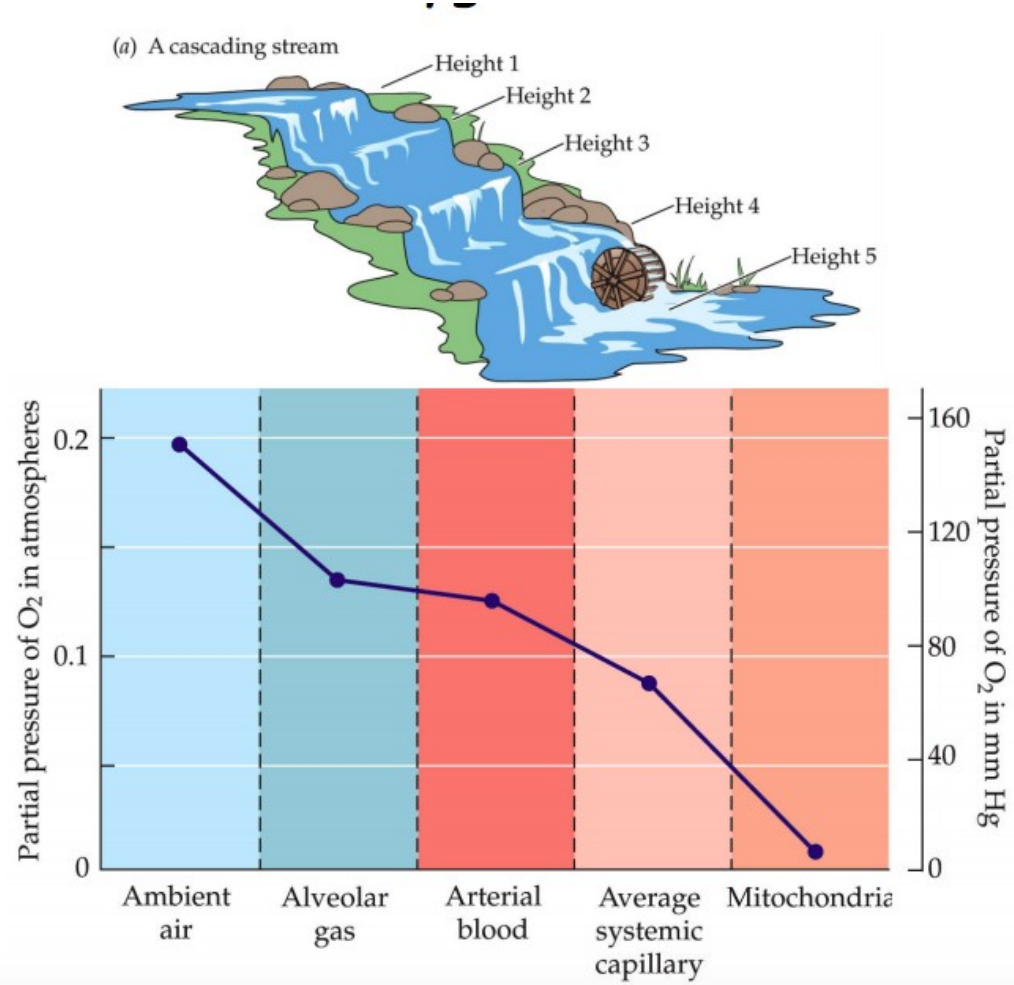
Oxygen cascade – progressive drop of oxygen content



- reasons for normal gradual decrease of PO_2 between air and blood:
  - „competition“ with CO_2 in alveoli
    - up to the atmospheric pressure
      - see alveolar gas equation
  - less that 100% diffusion across alveolo-capillary membrane
    - irregularity of its thickness and change in the rate of lung perfusion
      - diffusion & perfusion limitation
    - lower solubility of O_2 compared to CO_2
  - physiological right-left shunt
    - mixing of oxygenated and deoxygenated blood
      - nutritional supply of large airways by aa. bronchiales and their drainage to v. pulmonalis
      - drainage of vv. coronarie and thebesian veins into left atrium and other chambers
  - phyiological ventilation-perfusion inequality
  - physiologically a small fraction of abnormal Hb
    - Met-Hb
    - COHb
  - various oxygen extraction by tissues
- pathological aggravation in any if these steps contributing to drop of oxygen tension can **cause hypoxia**
  - hypoxic (= hypoxemia)
  - anaemic
  - circulatory
  - histotoxic

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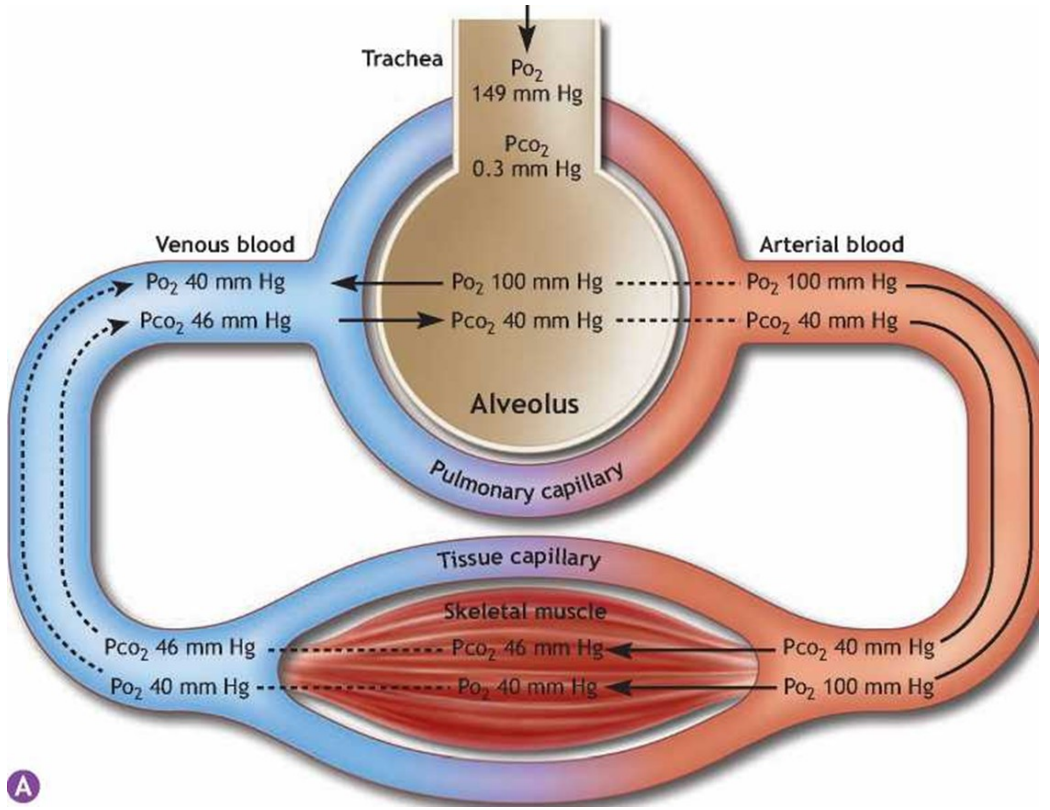
Quantitatively

- (1) inhaled **atmospheric** air
  - 21% O_2, 0.03% CO_2, 78% N_2, water gases 0.6% and the rest other gases (argon, helium, ..)
    - atm. pressure 760 mmHg (101 kPa)
    - PO_2: 0.21 x 760 = 160 mmHg
    - analogically PCO_2 = 0.3mmHg
- (2) **alveolar** air (mixture of inhaled and exhaled air)
  - P_AO_2 = 100mmHg (13.3kPa), P_ACO_2 = 40 mmHg (5.3kPa), $P_{water\ vapour}$ = 47 mmHg
    - P_AO_2 in alveolus slightly lower than atmospheric due to higher CO_2 content in alveolus (diffusion from blood)
- (3) **arterial** blood
  - PaO_2 = 90mmHg (12kPa), $PaCO_2$ = 45 mmHg
    - diffusion of oxygen not 100% and there is also physiological shunt
- (4) **venous** blood
  - PvO_2 = 30 - 50mmHg



	air (P)	alveolar (P_A)	arterial (Pa)	venous (Pv)
O_2	21kPa/150mmHg	13.3 kPa/100mmHg	12kPa/90mmHg	5.3kPa/40mmHg
CO_2	0.03kPa/0.3mmHg	5.3kPa/40mmHg	5.3kPa/40mmHg	6.0kPa/45mmHg

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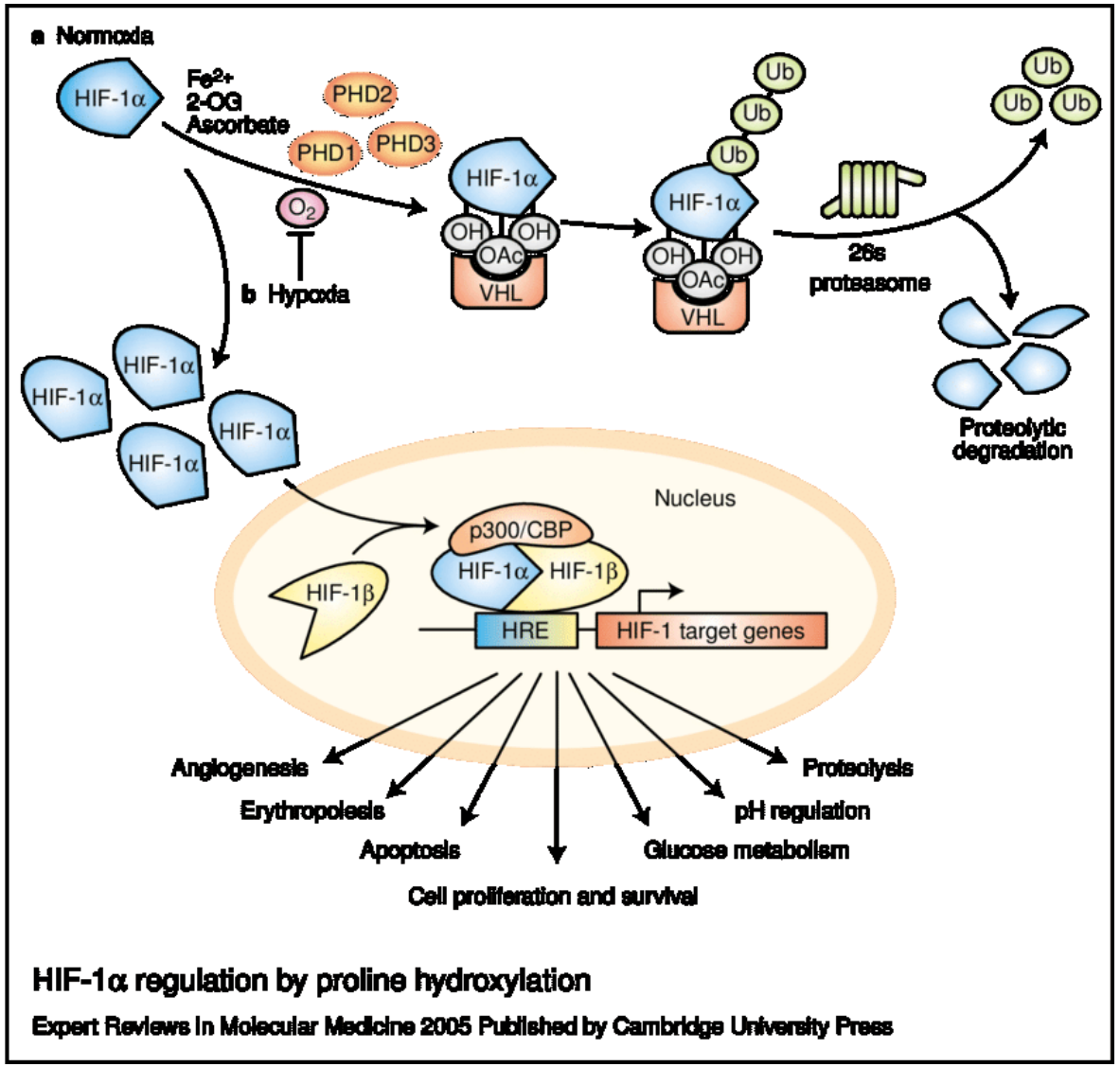
Hypoxia and its consequences



HIF-1α regulation by proline hydroxylation

Expert Reviews in Molecular Medicine 2005 Published by Cambridge University Press

- HIF-1α regulation by proline hydroxylation
  - (a) In normoxia, hypoxia-inducible factor (HIF)-1α is hydroxylated by proline hydroxylases (PHD1, 2 and 3) in the presence of O_2, Fe^{2+}, 2-oxoglutarate (2-OG) and ascorbate.
    - Hydroxylated HIF-1α (OH) is recognised by pVHL (the product of the von Hippel-Lindau tumour suppressor gene), which, together with a multisubunit ubiquitin ligase complex, tags HIF-1α with polyubiquitin; this allows recognition by the proteasome and subsequent degradation.
    - Acetylation of HIF-1α (OAc) also promotes pVHL binding.
  - (b) In response to hypoxia, proline hydroxylation is inhibited and VHL is no longer able to bind and target HIF-1α for proteasomal degradation, which leads to HIF-1α accumulation and translocation to the nucleus.
    - There, HIF-1α dimerises with HIF-1β, binds to hypoxia-response elements (HREs) within the promoters of target genes and recruits transcriptional co-activators such as p300/CBP for full transcriptional activity.
    - A range of cell functions are regulated by the target genes, as indicated.
  - Abbreviation: CBP, CREBbinding protein; Ub, ubiquitin.

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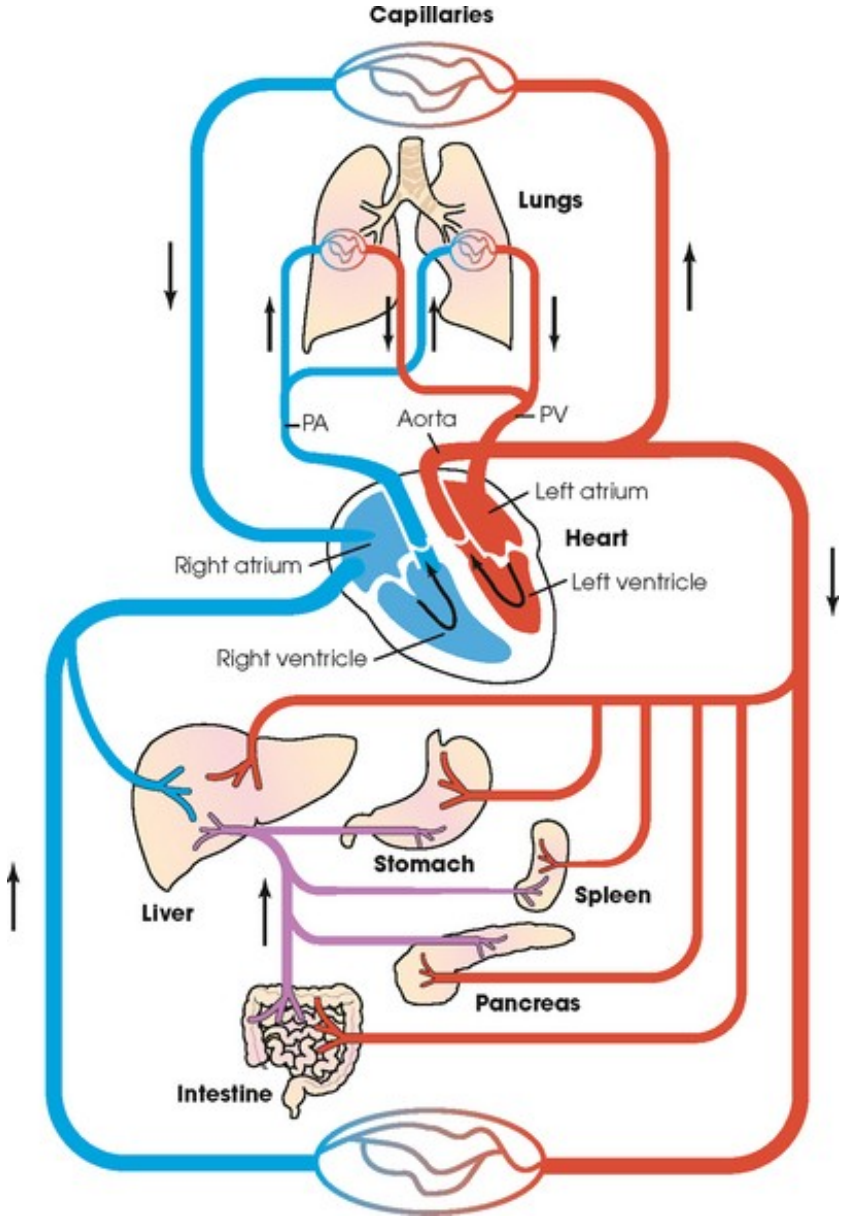
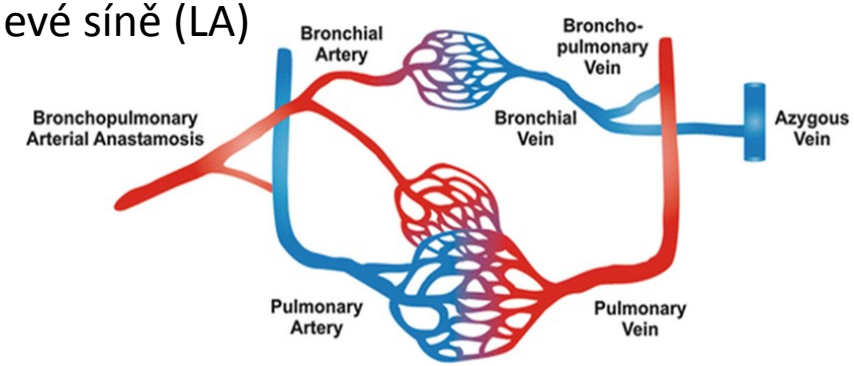
Pulmonary vs. systemic circulation



- Plíce jsou jediným orgánem, kterým prochází **veškerá krev**!!!
  - v objemu, který se rovná srdečnímu výdeji (cardiac output, CO)
- Tlak je generován pravou komorou (right ventricle, RV)
  - pří zvýšení CO (např. fyzická aktivita) musí být plicní cirkulace schopna pojmout objem bez významného zvýšení práce RV
    - distenze a „recruitment“ v klidu uzavřených kapilár
  - tj. vzhledem k jiným tlak a objemovým poměrům a délce je i **morfologie plicní cév jiná**
    - méně hladké svaloviny, větší roztažnost tlakem a zvýšeným průtokem
    - ale svalovina malých plicních arterií je důležitá – viz hypoxická vazokonstrikce
- Plicní vaskulární rezistence (PVR) kolísá mezi nádechem a nádechem, tedy s objemem plic (viz dále)
- Plíce mají **dvojí krevní zásobení**
  - deoxygenovaná krev z RV cestou plicní arterie (PA)
  - systémové (nutriční) zásobení dýchacích cest (po úroveň resp. bronchiolů) bronchiální cirkulací
    - odstup z descendentní aorty
    - bronchiální vény z malé části drénují do pulmonální vény a podílí se tak na fyziologickém zkratu
- 4 hlavní plicní vény ústí do levé síně (LA)



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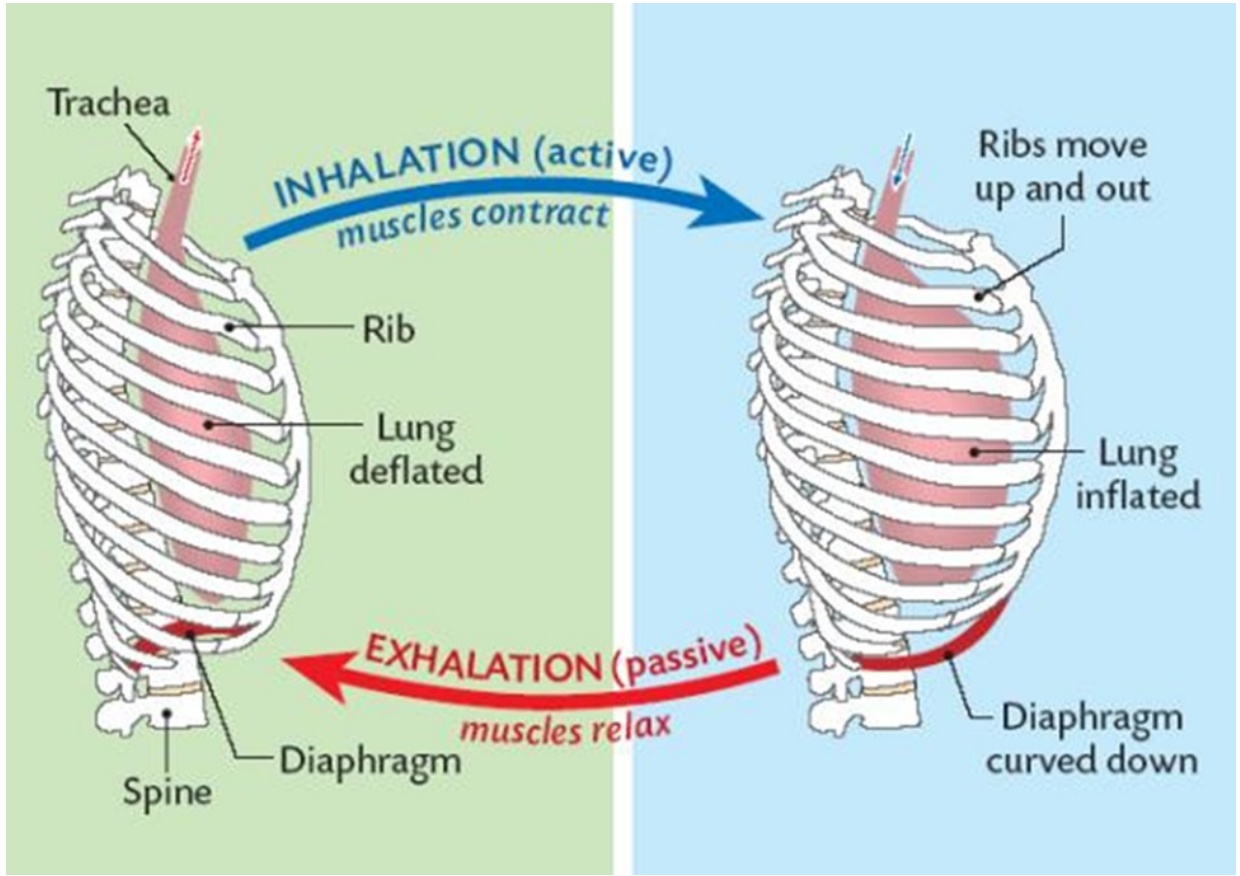
Summary

- The physiological structure of the lungs and airways ensures that
  - the work consumed for mechanical breathing is minimal
  - the airways and the lungs are able to effectively defend themselves against inhaled pathogens and particles
  - the area available to a gas exchange is huge, and the diffusion barrier minimal
  - in order to get enough O_2 into peripheral tissues, the exchange of gases in the lungs has to be as effective as possible
  - maintaining the concentration gradients necessary to keep the passive diffusion going is the principal driving force of ventilation
  - pulmonary circulation is adapted to maximize gas diffusion through the alveolar-capillary membrane



(1) PRINCIPLES OF VENTILATION AND ITS ABNORMALITIES

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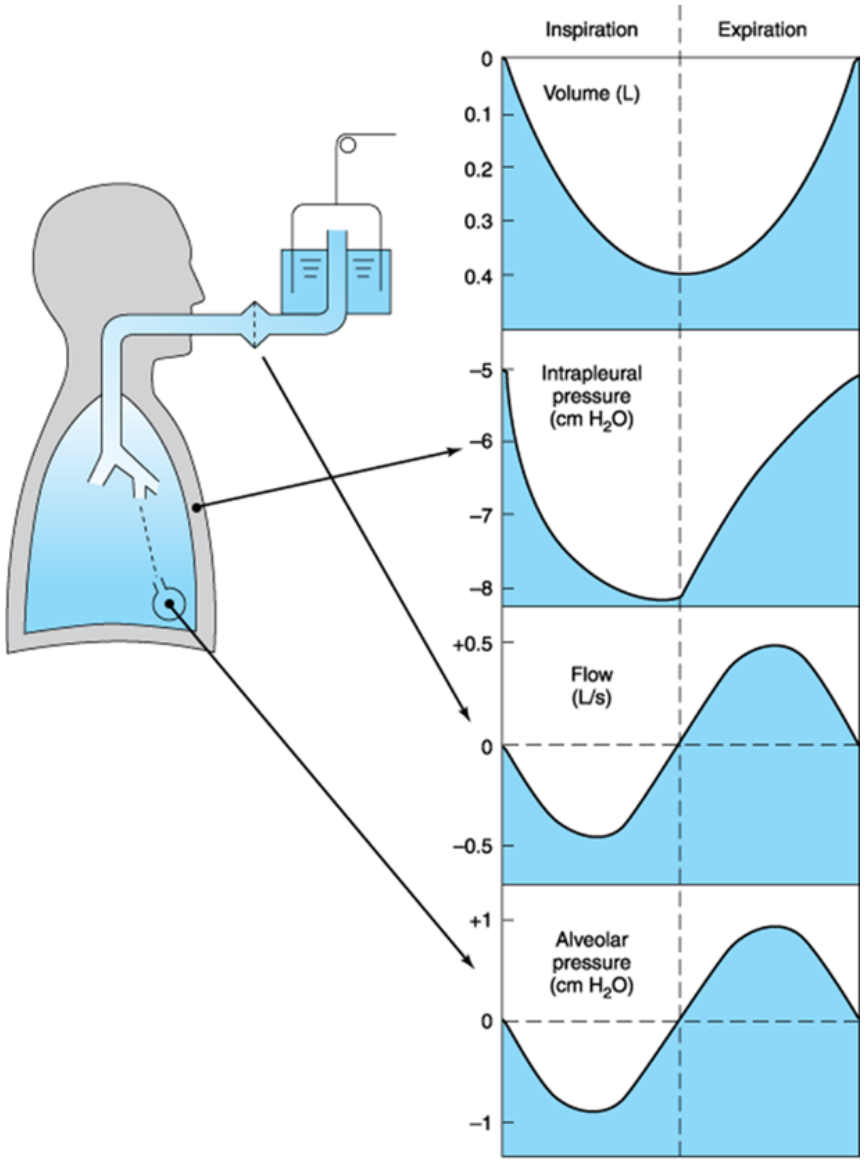
Mechanics of ventilation – breathing cycle



- pressures and pressure gradients
  - pressure on the body surface(P_{bs}),
    - usually equal to atmospheric (P_{ao})
  - alveolar pressure (P_{alv})
  - „elastic“ pressure (P_el)
    - generated by lung parenchyma and surface tension
  - pressure in pleural cavity (P_{pl})
  - trans-pulmonary pressure (P_L)
    - pressure difference between alveolus and pleural cavity
    - $P_L = P_{alv} - P_{pl}$
  - trans-thoracic pressure (P_{rs})
    - pressure difference between alveolus and body surface
    - determines actual phase of ventilation, i.e. inspirium or expirium
    - $P_{rs} = P_{alv} - P_{bs}$

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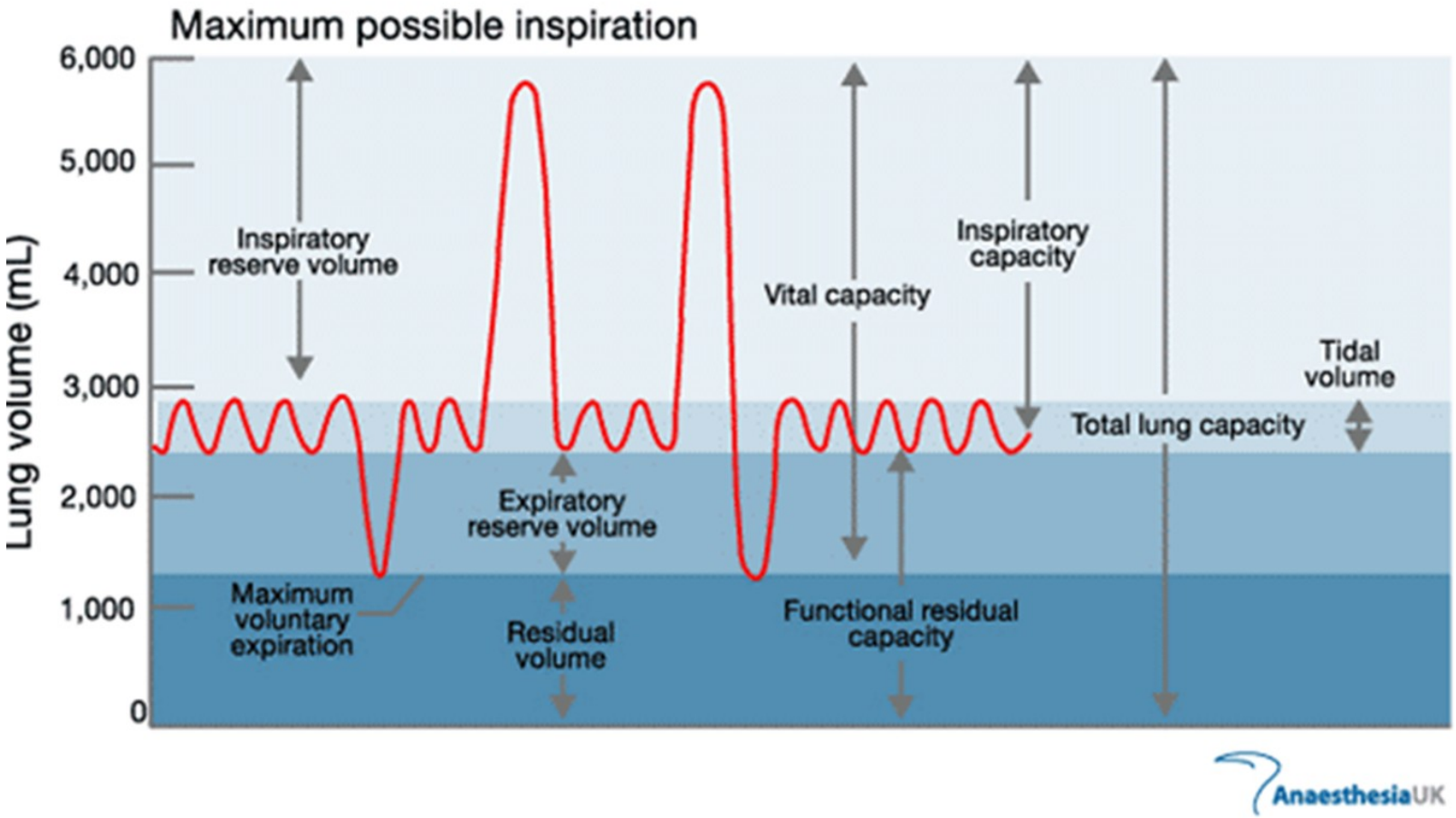
Lung volumes and capacities (tj. ≥ 2 volumes)



- The ratio of RV to TLC **(RV/TLC ratio)** in normal individuals is usually less than **0.25**
- abnormal = increased RV/TLC ratio in different types of pulmonary disease
  - obstructive diseases
    - ↑ RV
  - restrictive diseases
    - ↓ TLC

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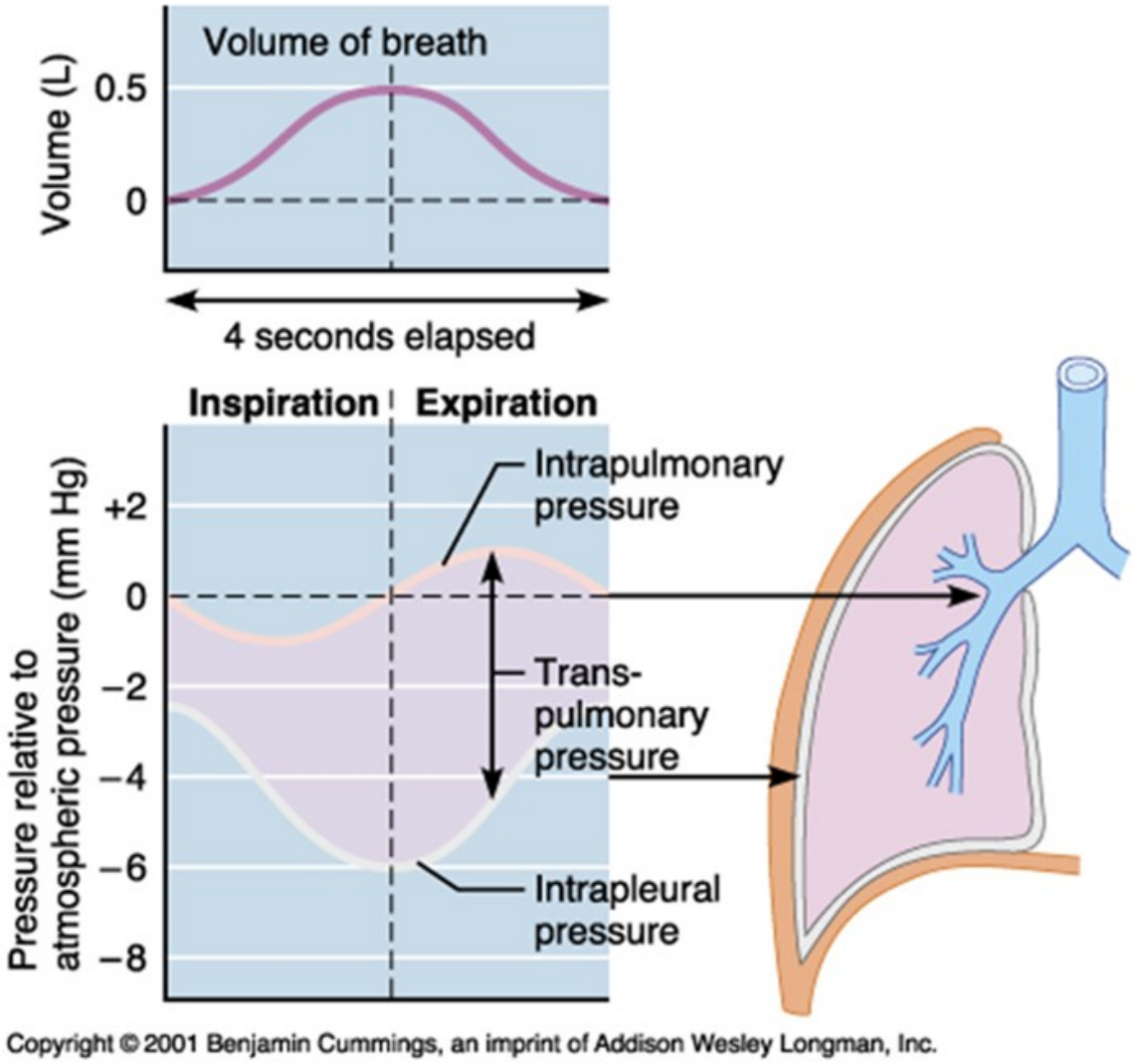
Ventilation (breathing) as a mechanical process



- **Inspiration**
  - an active process that results from the descent of the **diaphragm** and movement of the ribs upwards and outwards under the influence of the **intercostal muscles**
    - in resting healthy individuals, contraction of the diaphragm is responsible for most inspiration
  - respiratory muscles are similar to other skeletal muscles but are less prone to fatigue
    - weakness may play a part in respiratory failure resulting from neurological and muscle disorders and possibly with severe chronic airflow limitation
  - inspiration against increased resistance may require the use of the accessory muscles of ventilation
    - sternocleidomastoid and scalene muscles
- **Expiration**
  - follows passively as a result of gradual lessening of contraction of the intercostal muscles, allowing the lungs to collapse under the influence of their own elastic forces (**elastic recoil**)
  - forced expiration is also accomplished with the aid of accessory muscles
    - abdominal wall

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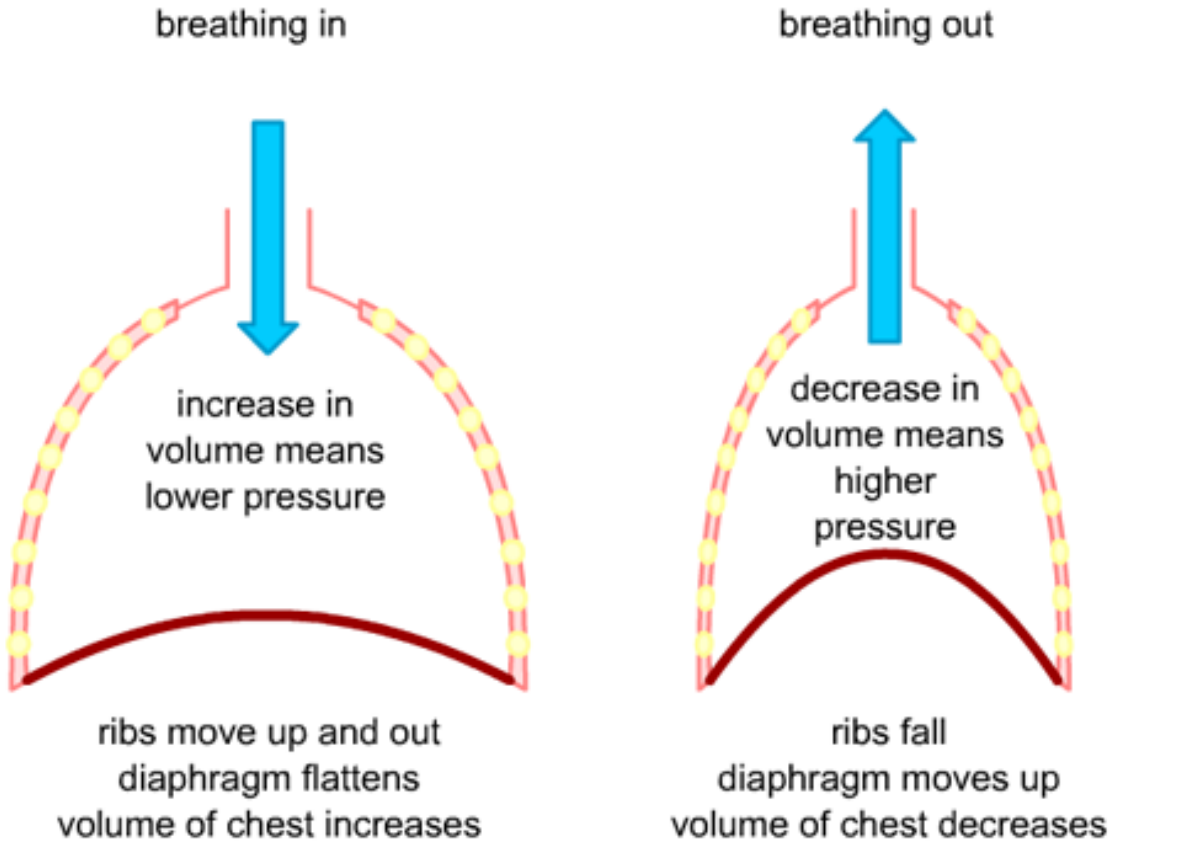
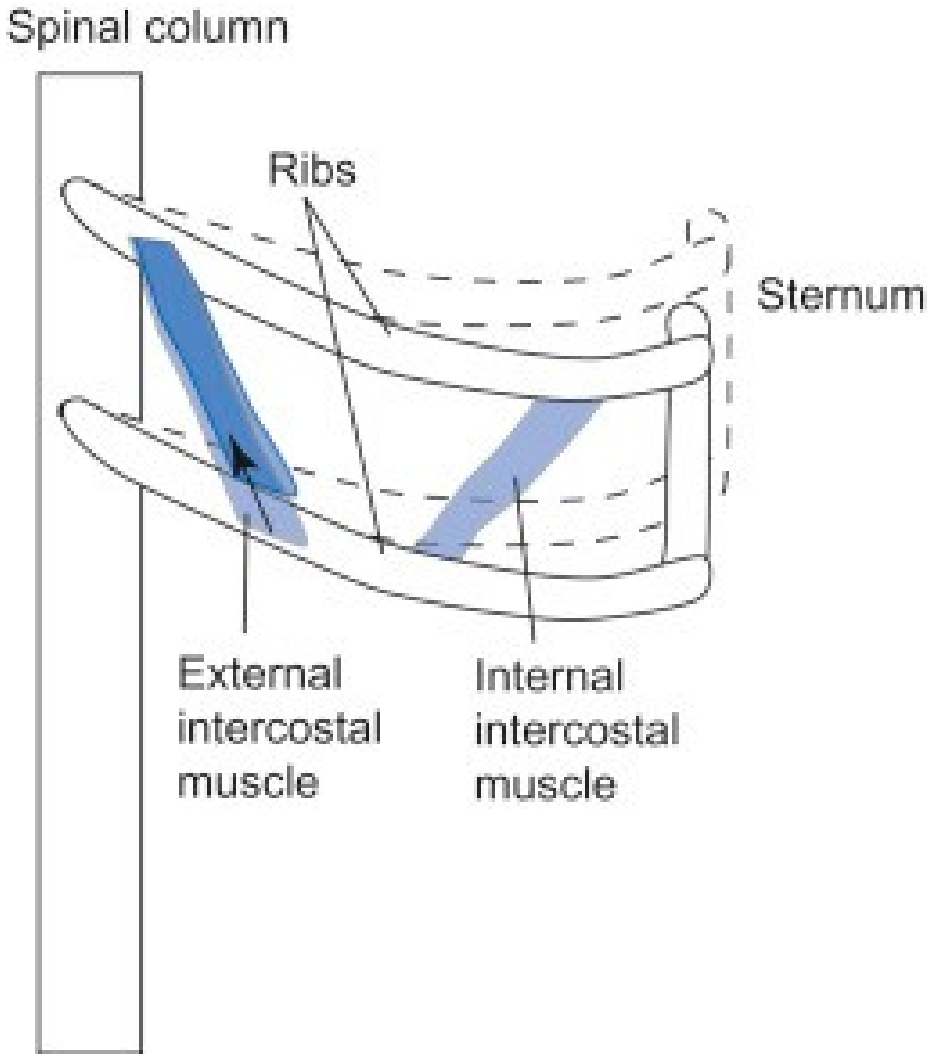
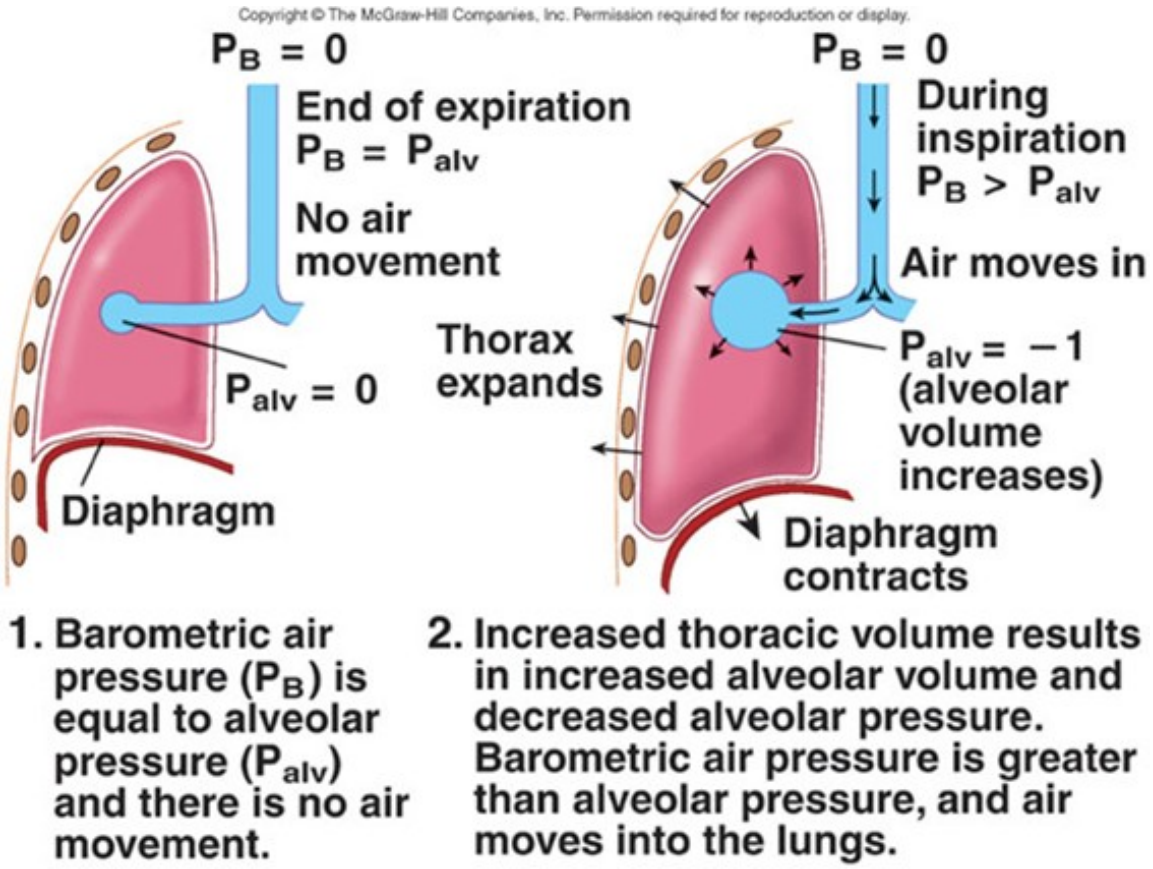
Muscles performing inspiration





Ventilation



- pressure necessary to distend lungs has to overcome two kinds of resistances
  - **(1) STATIC = elastic recoil**
    - in the respiratory part of airways and lung parenchyma
  - **(2) DYNAMIC = airway resistance**
    - in the convection part of airways
- energy requirements for respiratory muscles to overcome these resistances are normally quite low
  - 2-5 % of a total O_2 consumption
- but increases dramatically when resistance increases (up to 30%)

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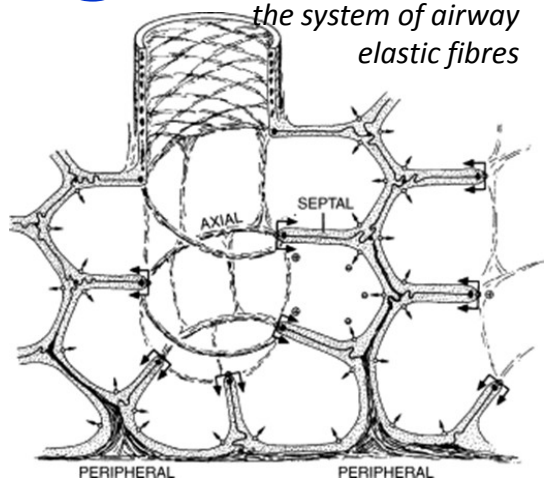
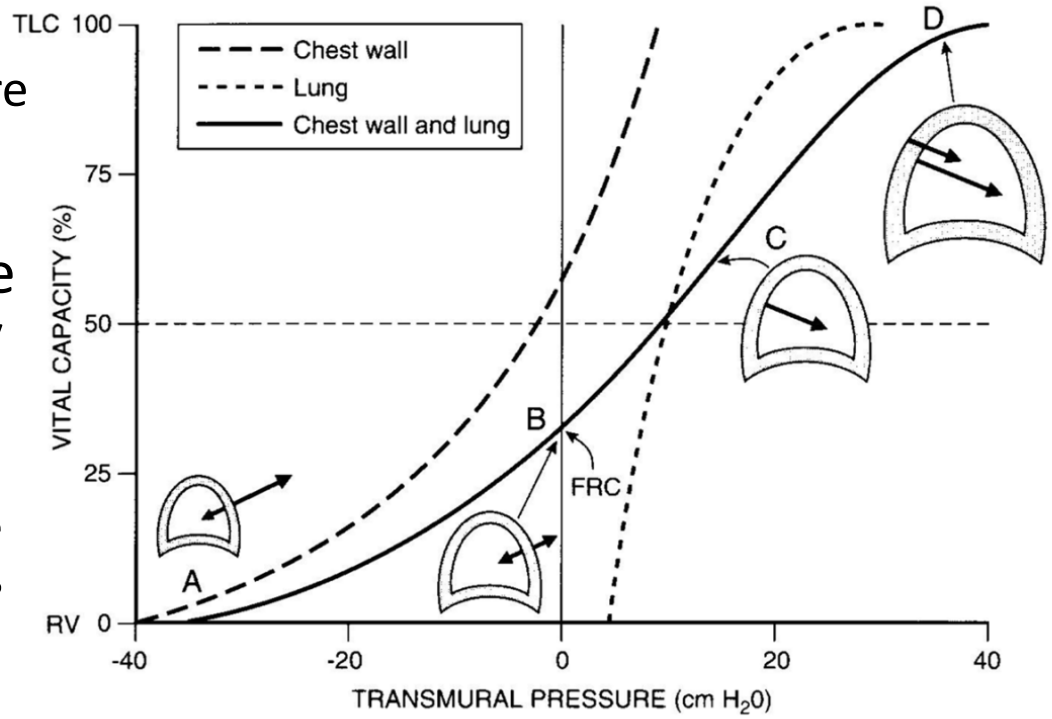
(ad 1) Elastic properties of the lung

- lungs have an inherent elastic property that causes them to tend to collapse generating a negative pressure within the pleural space
  - the strength of this retractive force relates to the volume of the lung
    - for example, at higher lung volumes the lung is stretched more, and a greater negative intrapleural pressure is generated
  - at the end of a quiet expiration, the retractive force exerted by the lungs is balanced by the tendency of the thoracic wall to spring outwards
    - at this point, respiratory muscles are resting and the volume of the lung is known as the **functional residual capacity** (FRC)

the system of airway elastic fibres





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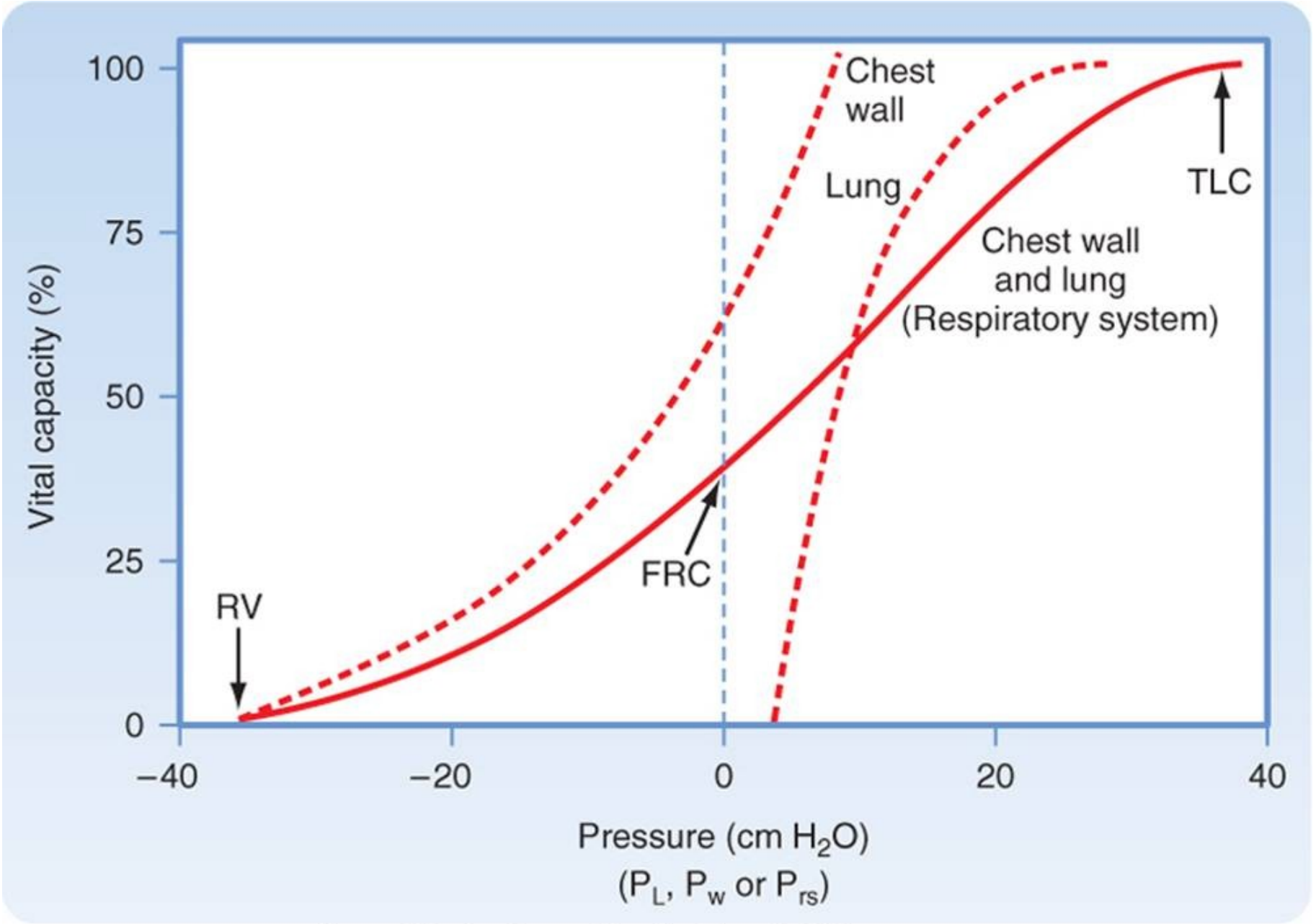

The transmural pressure across the respiratory system at FRC is zero. At TLC, both lung pressure and chest wall pressure are positive, and they both require positive transmural distending pressure. The resting volume of the chest wall is the volume at which the transmural pressure for the chest wall is zero, and it is approximately 60% of TLC. At volumes greater than 60% of TLC, the chest wall is recoiling inward and positive transmural pressure is needed, whereas at volumes below 60% of TLC, the chest wall tends to recoil outward

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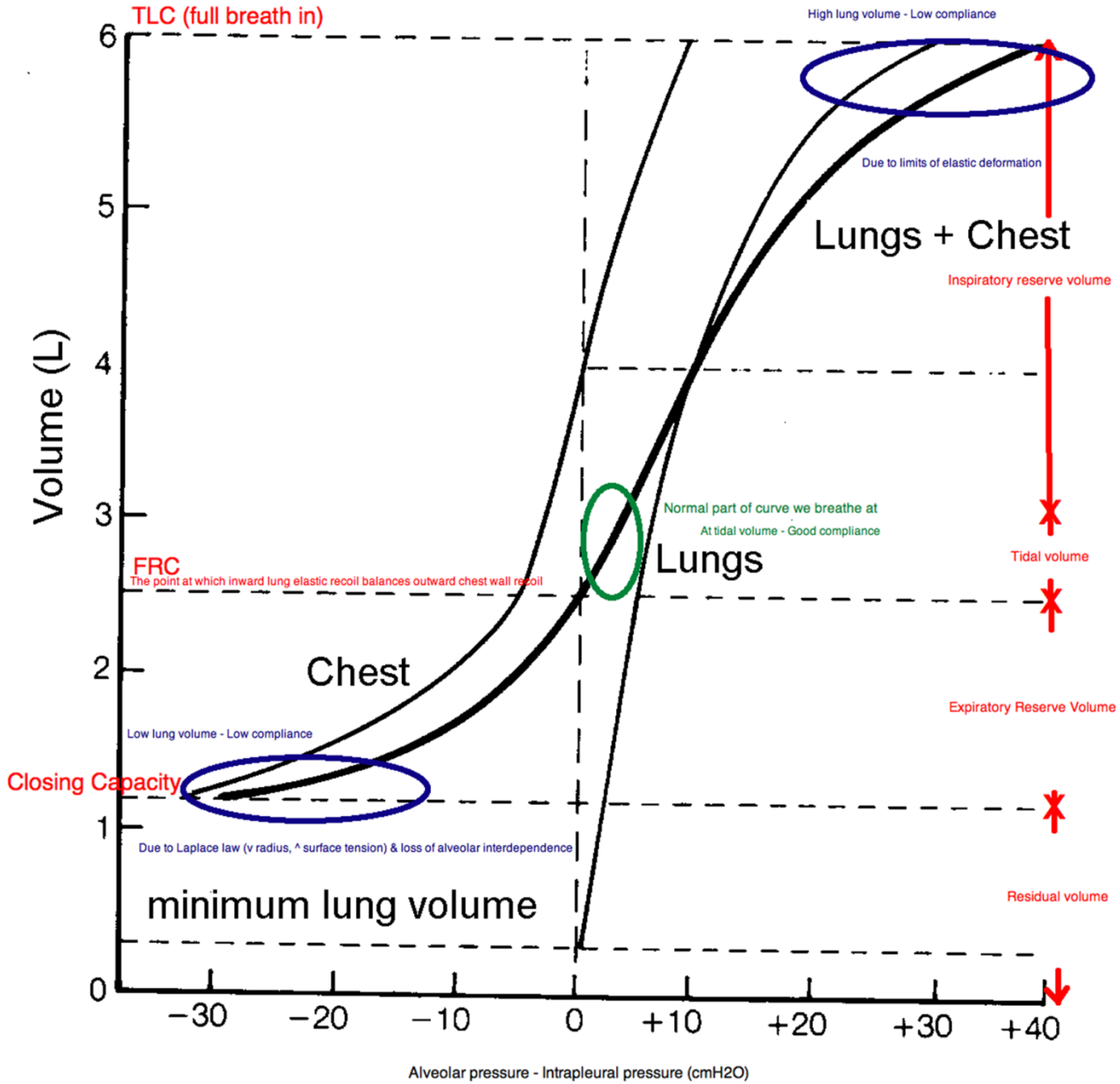
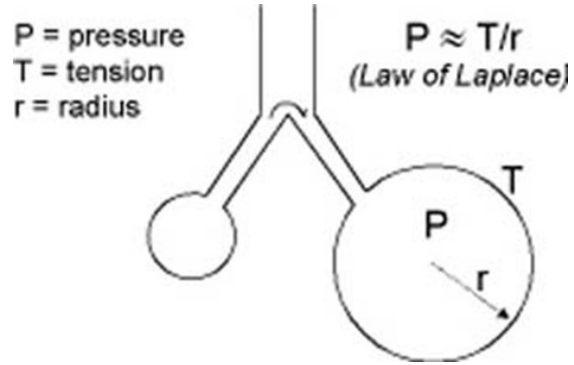
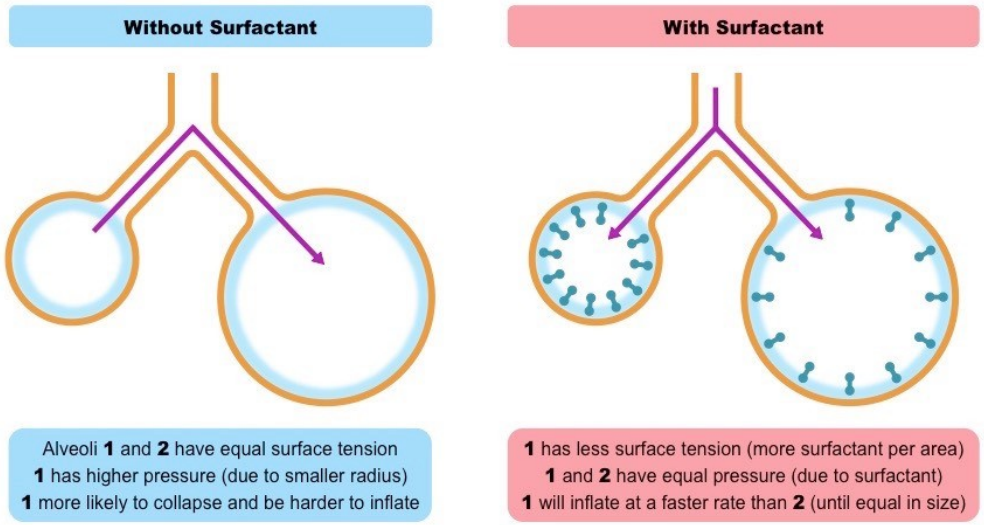

TLC (full breath in)
High lung volume - Low compliance
Due to limits of elastic deformation
Lungs + Chest
Inspiratory reserve volume
Volume (L)
6
5
4
3
2
1
0
Normal part of curve we breathe at
At tidal volume - Good compliance
Lungs
Tidal volume
FRC
The point at which inward lung elastic recoil balances outward chest wall recoil
Chest
Expiratory Reserve Volume
Low lung volume - Low compliance
Closing Capacity
Due to Laplace law (v radius, ^ surface tension) & loss of alveolar interdependence
minimum lung volume
Residual volume
−30
−20
−10
0
+10
+20
+30
+40
Alveolar pressure - Intrapleural pressure (cmH2O)

Elastic recoil is determined by two kinds of forces

- **lung compliance** ("distensibility")
  - a measure of the relationship between this retractive force and lung volume (pressure-volume relationship)
  - defined as the change in lung volume brought about by unit change in transpulmonary (intrapleural) pressure (L/kPa)
- **surface tension** produced by the layer of fluid that lines the alveoli
  - determined by the cohesive (binding together) forces between molecules of the same type
    - on the inner surface of the alveoli there is a fluid that can resist lung expansion
    - there would be a lot of surface tension because there is an air-water interface in every alveolus
    - if surface tension remained constant, decreasing r during expiration would increase P and smaller alveolus would empty into large one
  - this collapsing tendency is offset by **pulmonary surfactant** which significantly lowers surface tension





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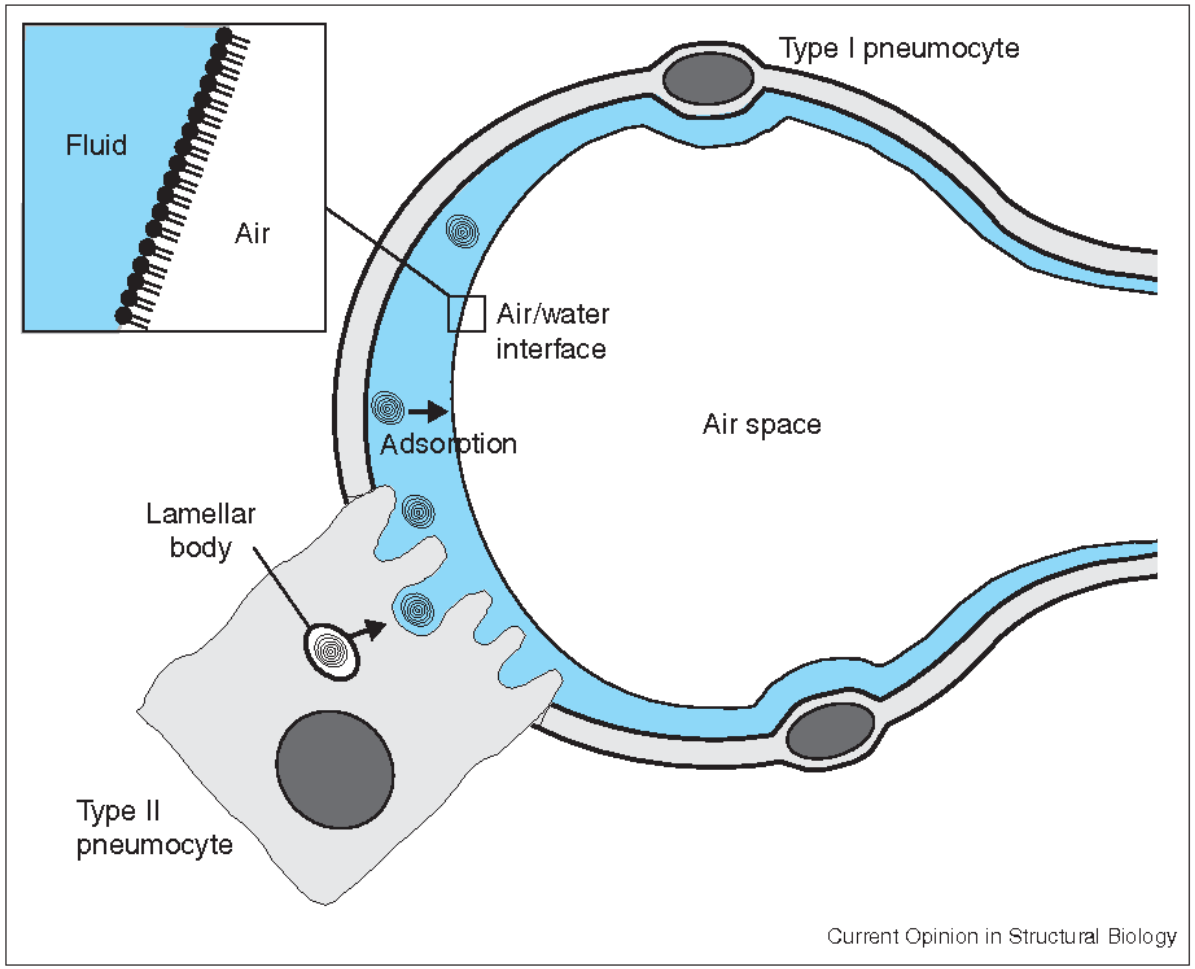
Pulmonary surfactant

- Complex mixture of **lipids and proteins** at the alveolar cell surface (liquid – gas interface) reducing surface tension
  - superficial layer made of phospholipids (dipalmitoyl lecithin)
  - deeper layer (hypophase) made of proteins (SP-A, -B, -C, -D)
- Surfactant maintains lung volume at the end of expiration
- Continually and very rapidly recycles
  - influenced by many hormones incl. glucocorticoids
    - lung maturation in pre-term newborns



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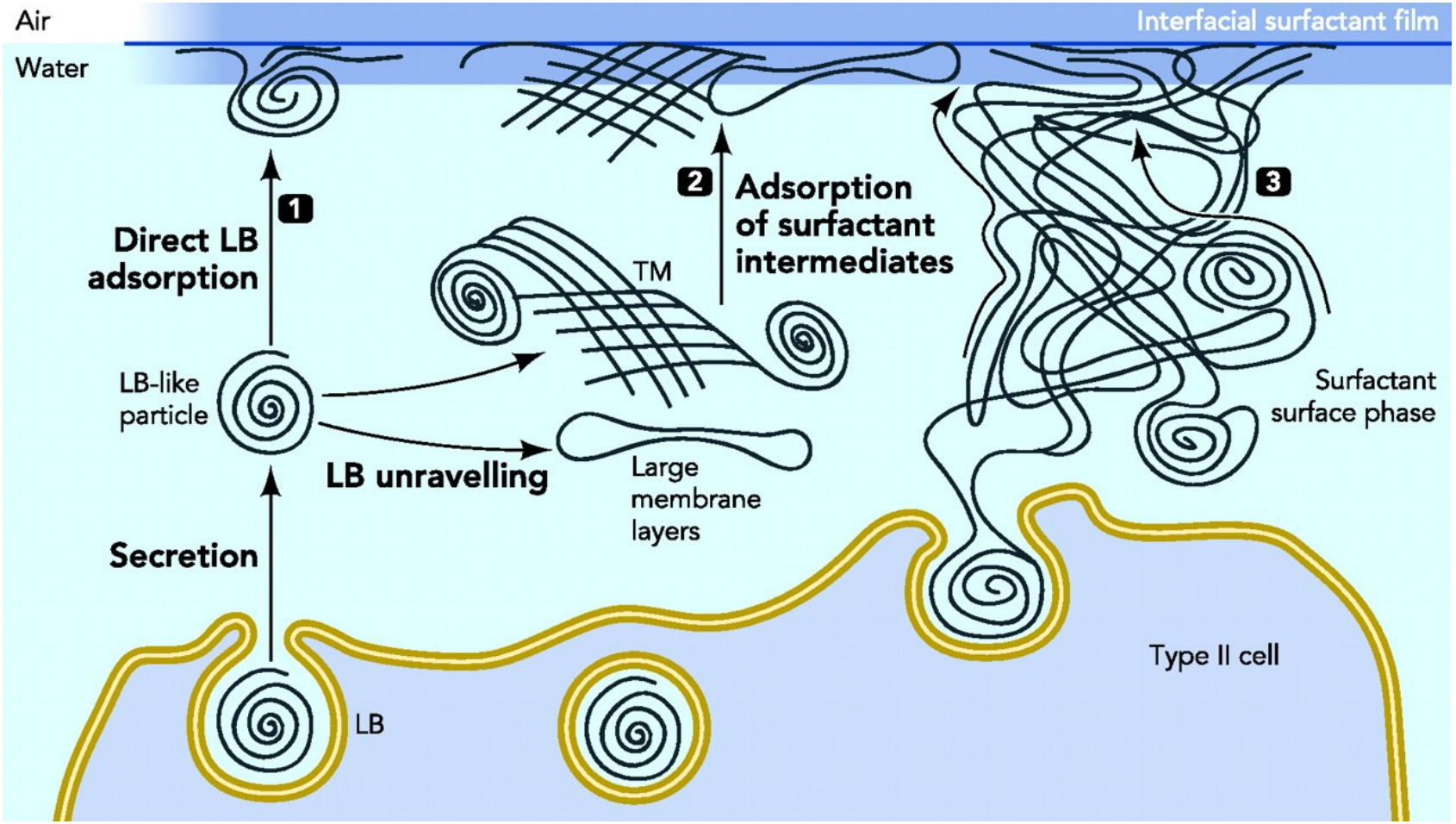
Pulmonary surfactant adsorption to the interface and surface film formation. Processes that may contribute to transport of surface active surfactant species to the interface include 1) direct cooperative transfer of surfactant from secreted lamellar body-like particles touching the interface, 2) unravelling of secreted lamellar bodies to form intermediate structures such as tubular myelin (TM) or large surfactant layers that have the potential to move and transfer large amounts of material to the interface, and 3) rapid movement of surface active species through a continuous network of surfactant membranes, a so-called surface phase, connecting secreting cells with the interface.



Perez-Gil J , Weaver T E Physiology 2010;25:132-141

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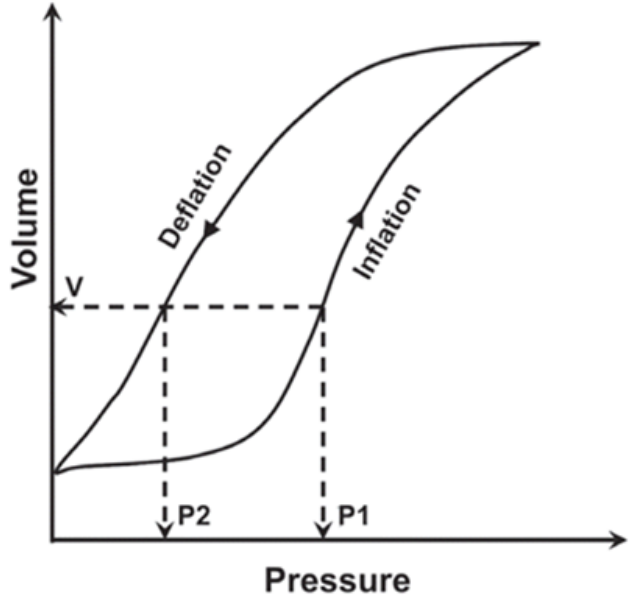
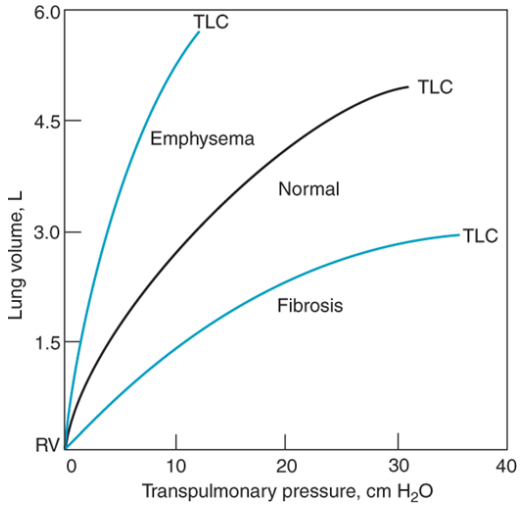
Abnormalities of elastic properties

- change of lung compliance (TLC, FRC, RV)
  - ↑ pulmonary **emphysema**, aging (↑TLC, ↑FRC, ↑RV)
  - ↓ **interstital disease** (↓TLC, ↓FRC, ↓RV)
    - e.g. pulmonary fibrosis or bronchopneumonia
- lack of surfactant (↓TLC, ↓FRC, ↓RV)
  - infant or adult **respiratory distress syndromes** (IRDS or ARDS, resp.), i.e. tendency of lung to collapse
  - alveolar lung **edema** (damages/dilutes surfactant)
- diseases that affect the movement of the thoracic cage and diaphragm
  - marked obesity
  - diseases of the thoracic spine
    - ankylosing spondylitis and kyphoscoliosis
  - neuropathies
    - e.g. the Guillain-Barré syndrome)
  - injury to the phrenic nerves (spine C3-C5)
  - myasthenia gravis





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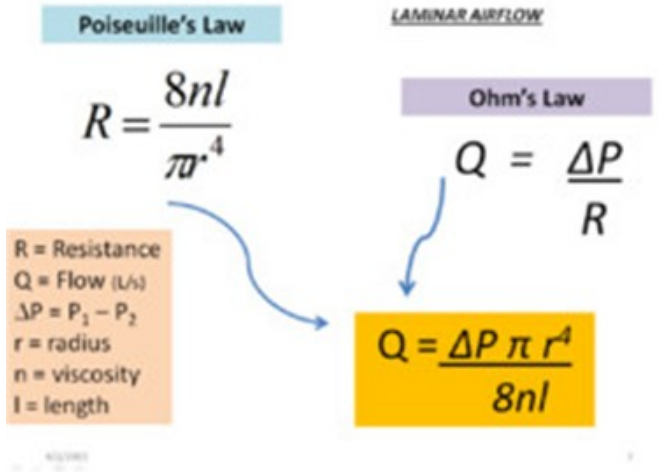
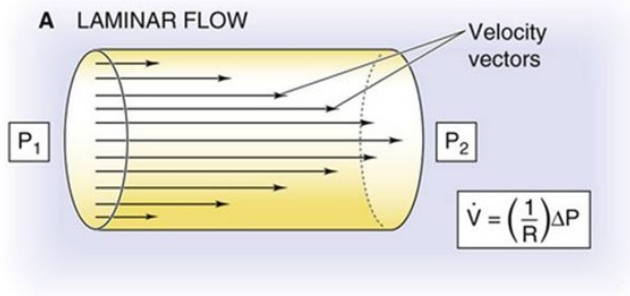
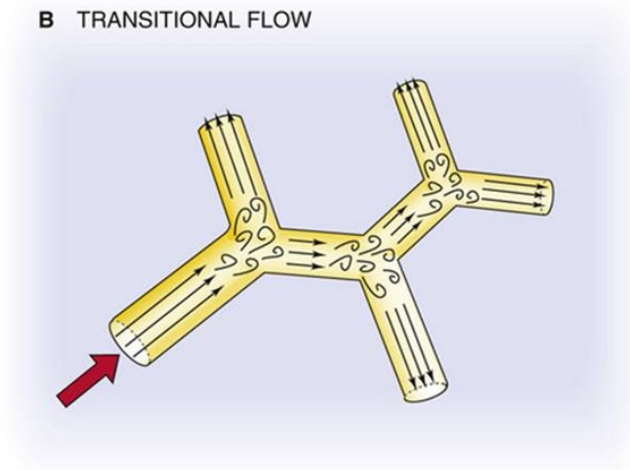
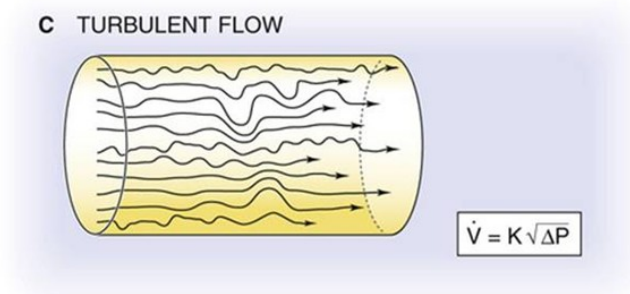
(ad 2) Airway (dynamic) resistance

- Poiseuille's law for pressure states that pressure is
  - directly proportional to flow, tube length, and viscosity
  - and it is inversely proportional to tube radius
- Overcoming increased resistance requires **forced expiration**









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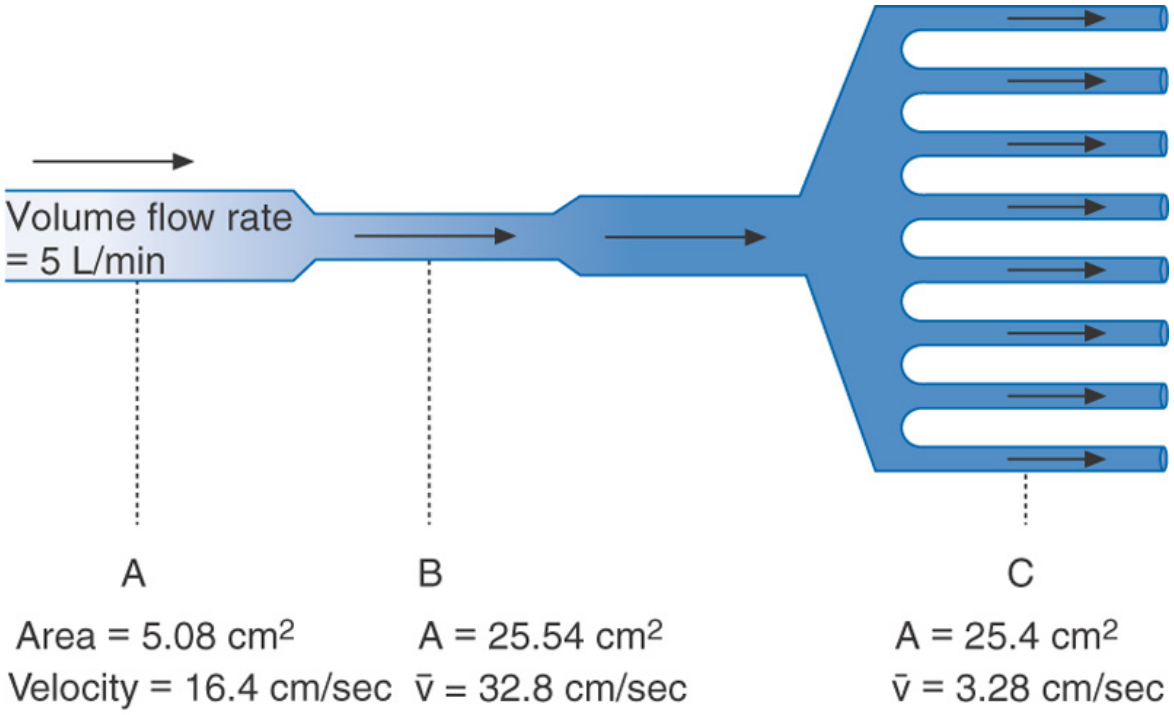
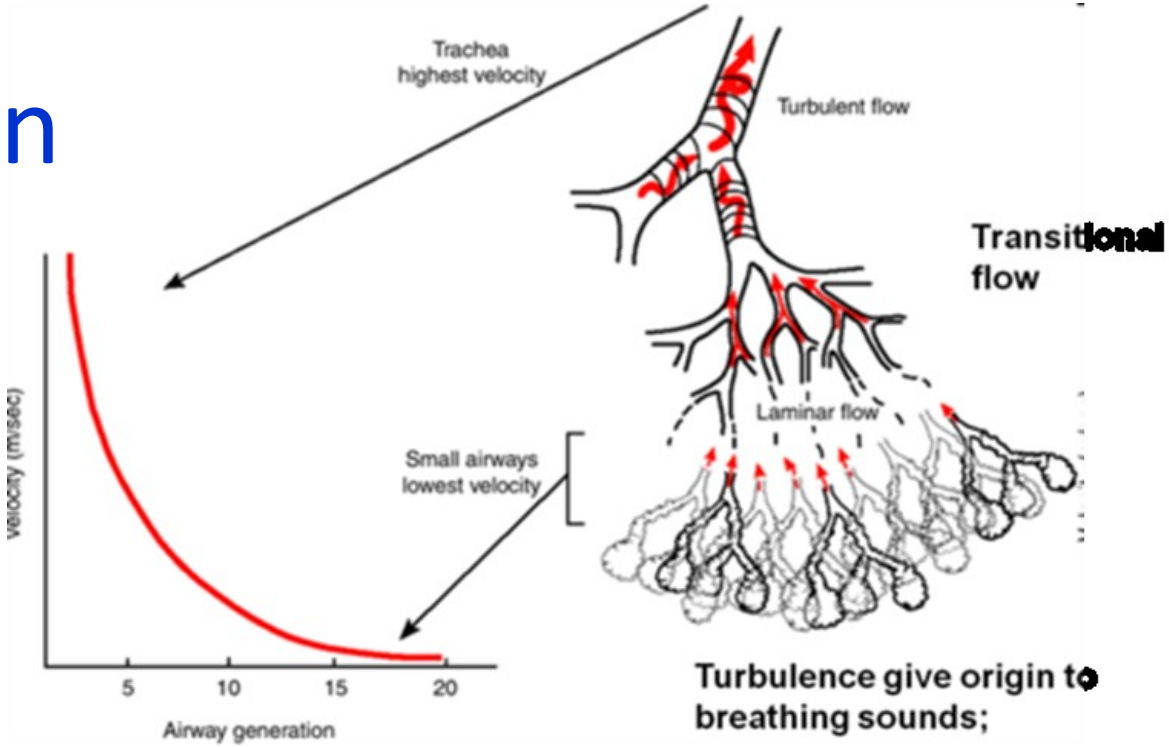
Airflow velocity and pattern

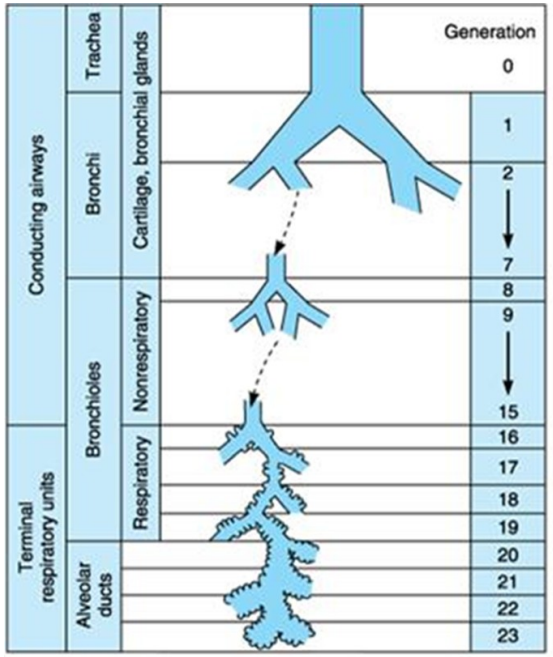


(Modified from Nave CR, Nave BC: Physics for the health sciences, ed 3, Philadelphia, 1985, WB Saunders.)



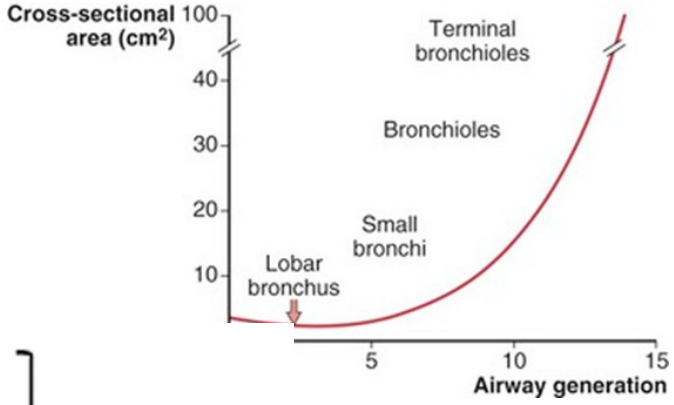
Turbulence give origin to breathing sounds;

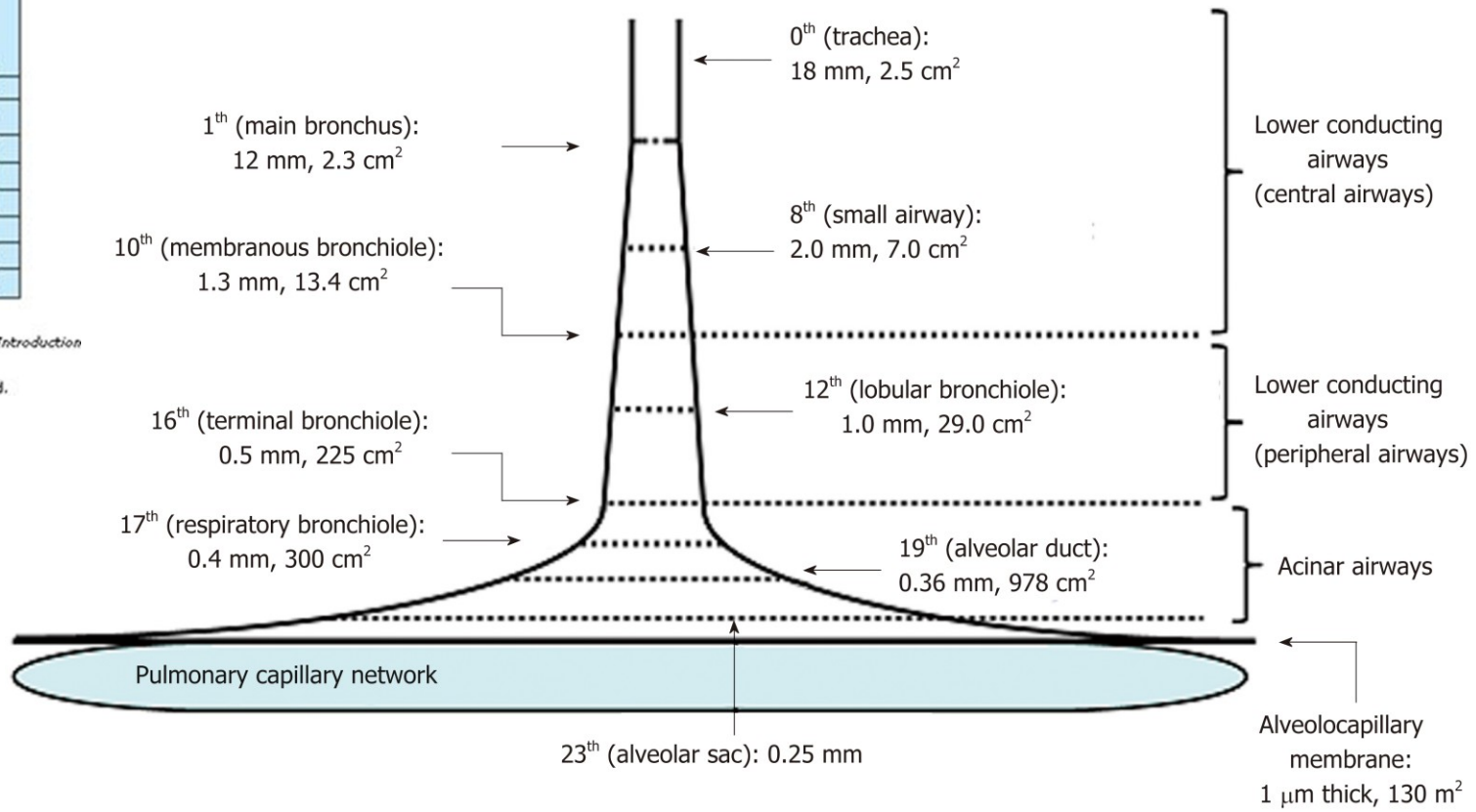
Airflow – where is the highest resistance?



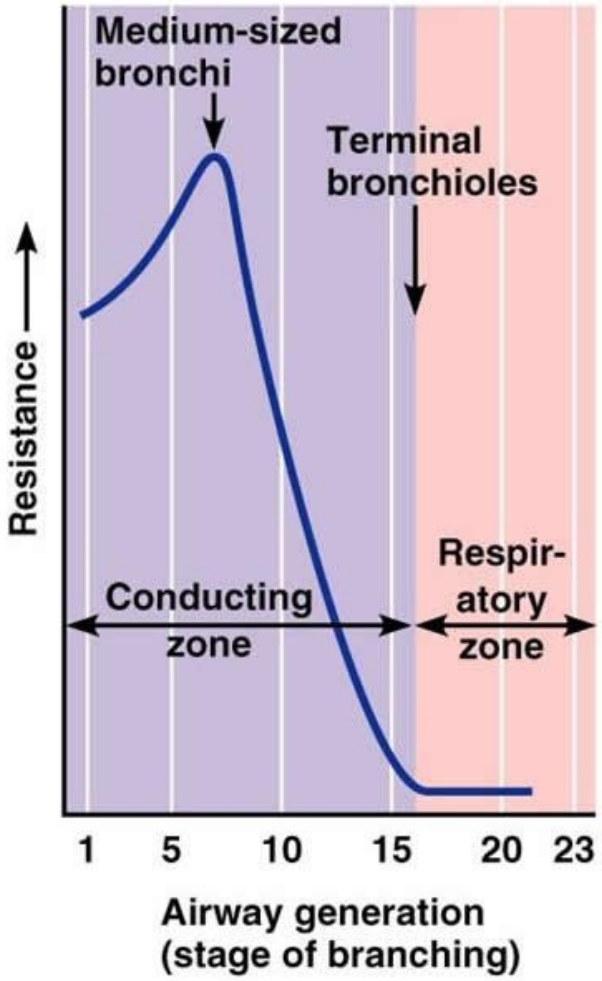
Source: McPhee SJ, Ganong WF: *Pathophysiology of Disease: An Introduction to Clinical Medicine*, 5th Edition: http://www.accessmedicine.com

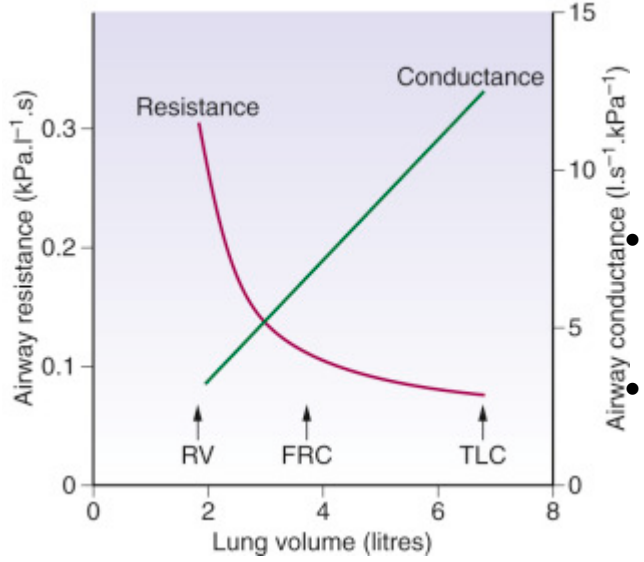
$$R = \frac{8\eta l}{\pi r^4}$$

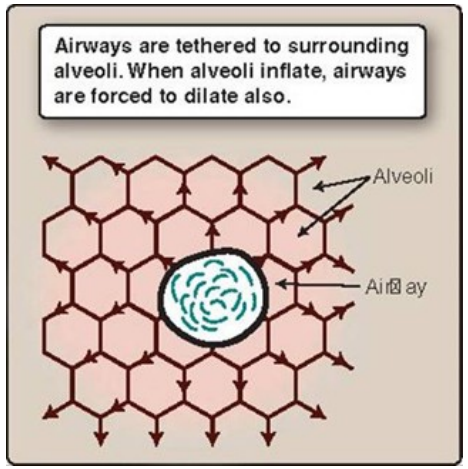




Airflow resistance







- From the trachea to the periphery, the airways become smaller in size (although greater in number)
  - **the cross-sectional area available for airflow increases as the total number of airways increases**
  - the flow of air is greatest in the trachea and slows progressively towards the periphery (as the velocity of airflow depends on the ratio of flow to cross-sectional area)
    - in the terminal airways, gas flow occurs solely by diffusion
- The **resistance to airflow** is very low (0.1-0.2 kPa/L in a normal tracheobronchial tree), **steadily increasing from the small to the large airways**
- Airway tone is under the control of the autonomic nervous system
  - bronchomotor tone is maintained by **vagal efferent nerves**
  - many **adrenoceptors** on the surface of bronchial muscles respond **to circulating catecholamines**
    - sympathetic nerves do not directly innervate them!
- Airway resistance is also **related to lung volumes**
  - because airways are ‘tethered’ by alveoli (i.e. pulled open by radial traction)
  - visible on bronchoscopy
  - patients with obstruction benefit from breathing in high lung volumes

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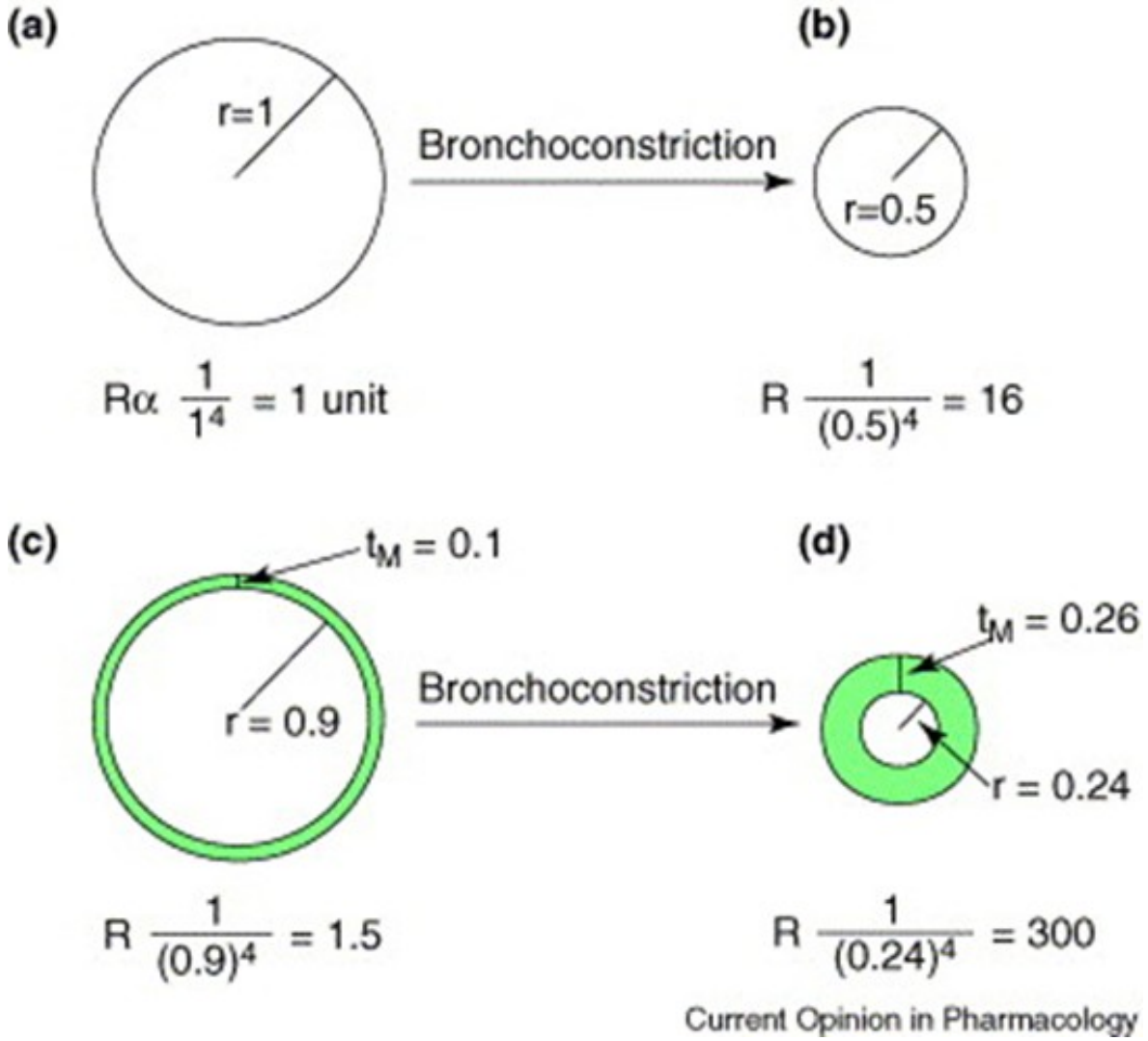
Airflow resistance – effect of changed airway diameter



$$R = \frac{8\eta l}{\pi r^4}$$

- theoretical amplifying effect of luminal mucus on airflow resistance in asthma
  - **(a)** According to Poiseuille's law, resistance to flow (R) is proportional to the reciprocal of the radius (r) raised to the fourth power.
  - **(b)** Without luminal mucus, bronchoconstriction to reduce the airway radius by half increases airflow resistance 16-fold.
  - **(c)** A small increase in mucus thickness (t_M), which reduces the radius of the airway by only one-tenth, has a negligible effect on airflow in the unconstricted airway (compare with panel **a**).
  - **(d)** With bronchoconstriction, the same amount of luminal mucus markedly amplifies the airflow resistance of this airway

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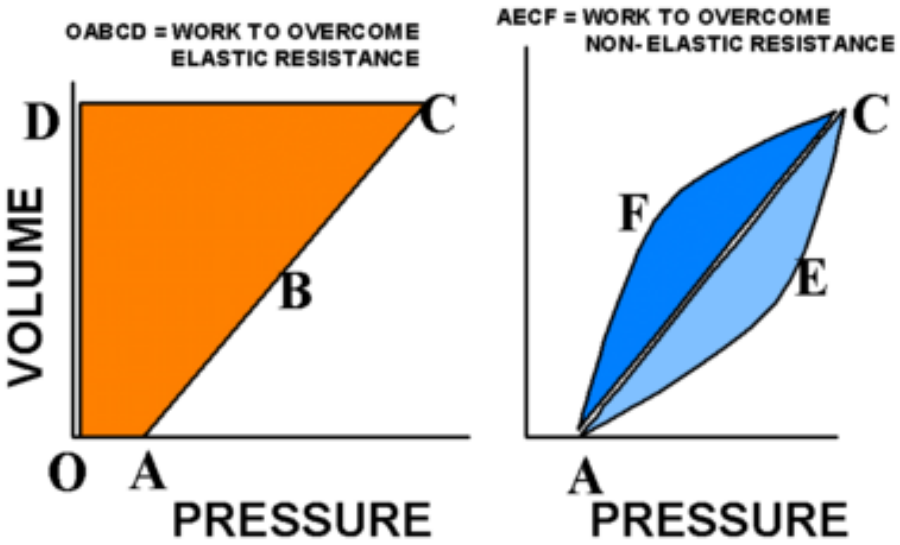
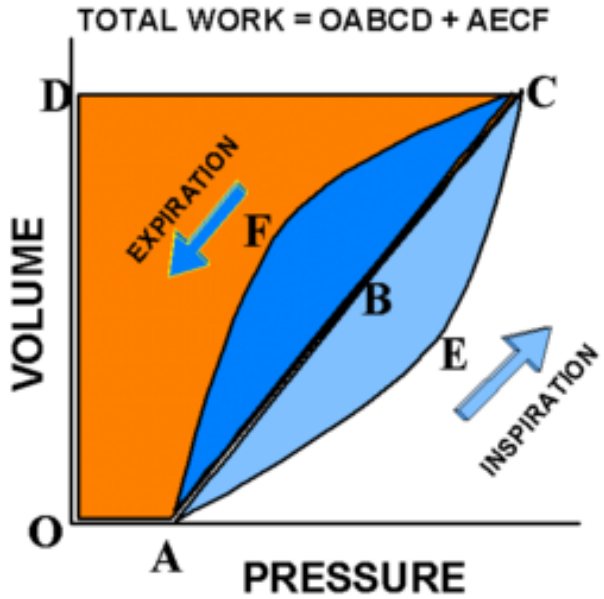
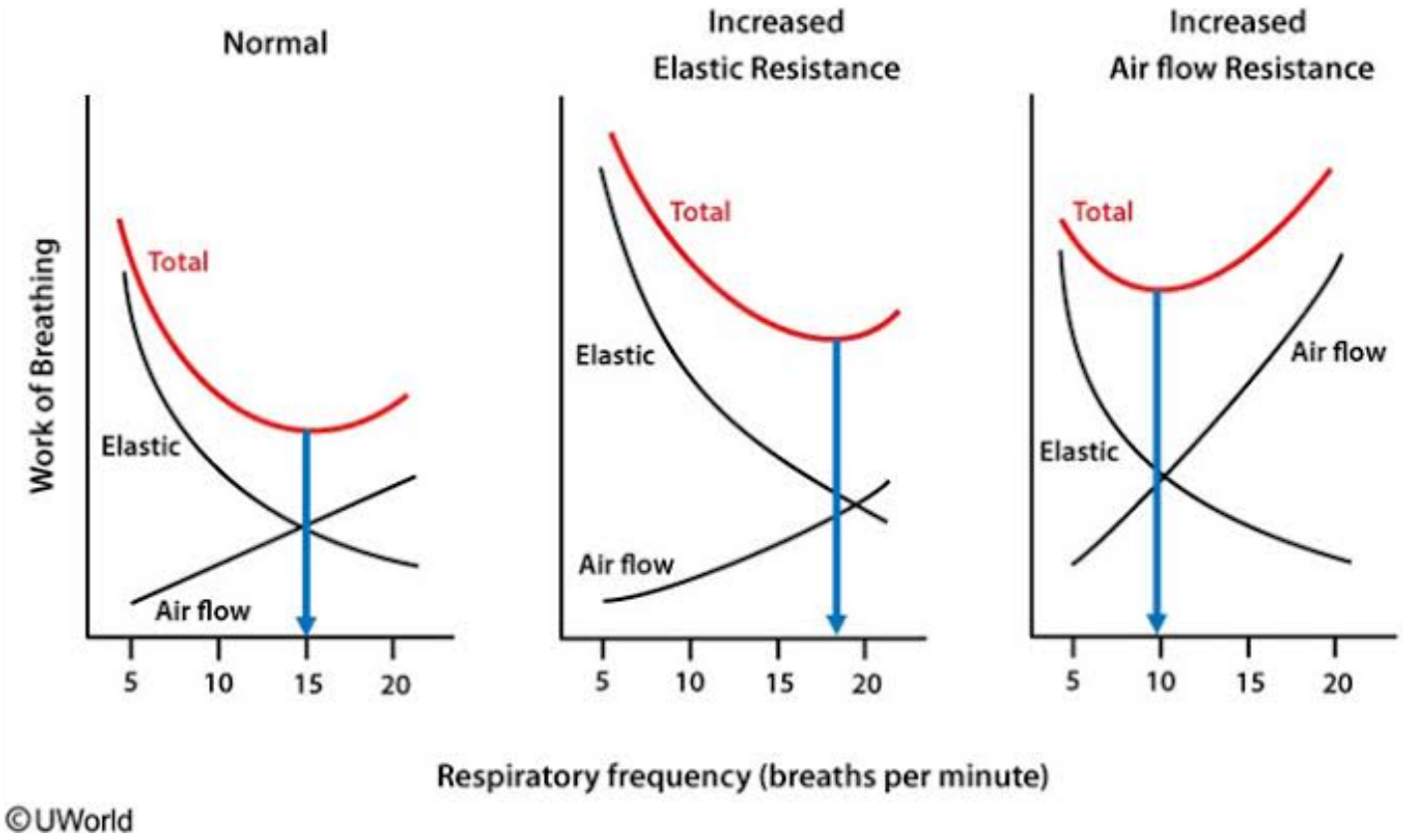
Work of breathing

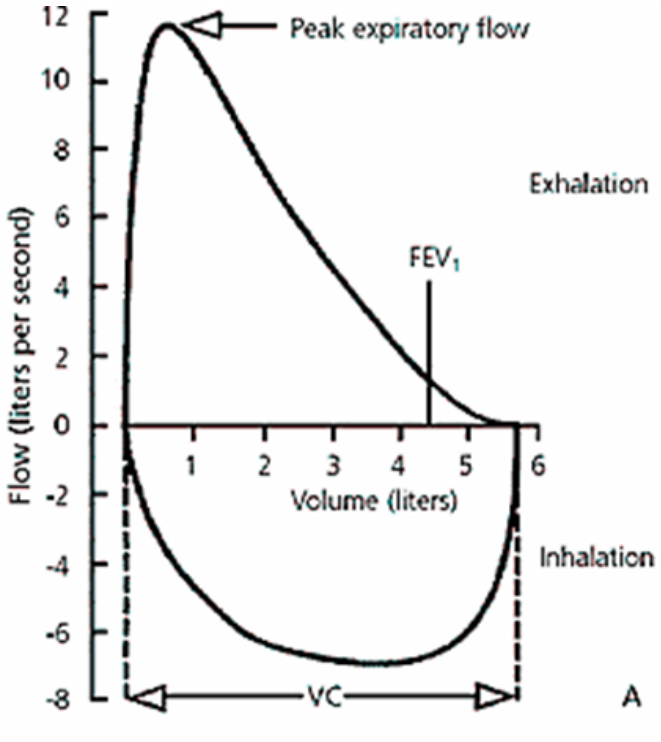






Airflow pattern

- Movement of air through the airways results from a **difference between the pressure in the alveoli and the atmospheric pressure**
  - alveolar pressure (P_{ALV}) is equal to the elastic recoil pressure (P_{EL}) of the lung plus the pleural pressure (P_{PL})
  - positive P_{ALV} occurs in expiration and a negative pressure occurs in inspiration
- During quiet breathing the sub-atmospheric pleural pressure throughout the breathing cycle slightly distends the airways
  - during vigorous expiratory efforts (e.g. cough) the central airways are compressed by positive pleural pressures exceeding 10 kPa
  - the airways do not close completely because the driving pressure for expiratory flow (alveolar pressure) is also increased
- When there is no airflow (i.e. during a pause in breathing) the tendency of the lungs to collapse (the positive P_{EL}) is exactly balanced by an equivalent negative P_{PL}



The relationship between maximal flow rates on expiration and inspiration is demonstrated by the maximal flow-volume (MFV) loops

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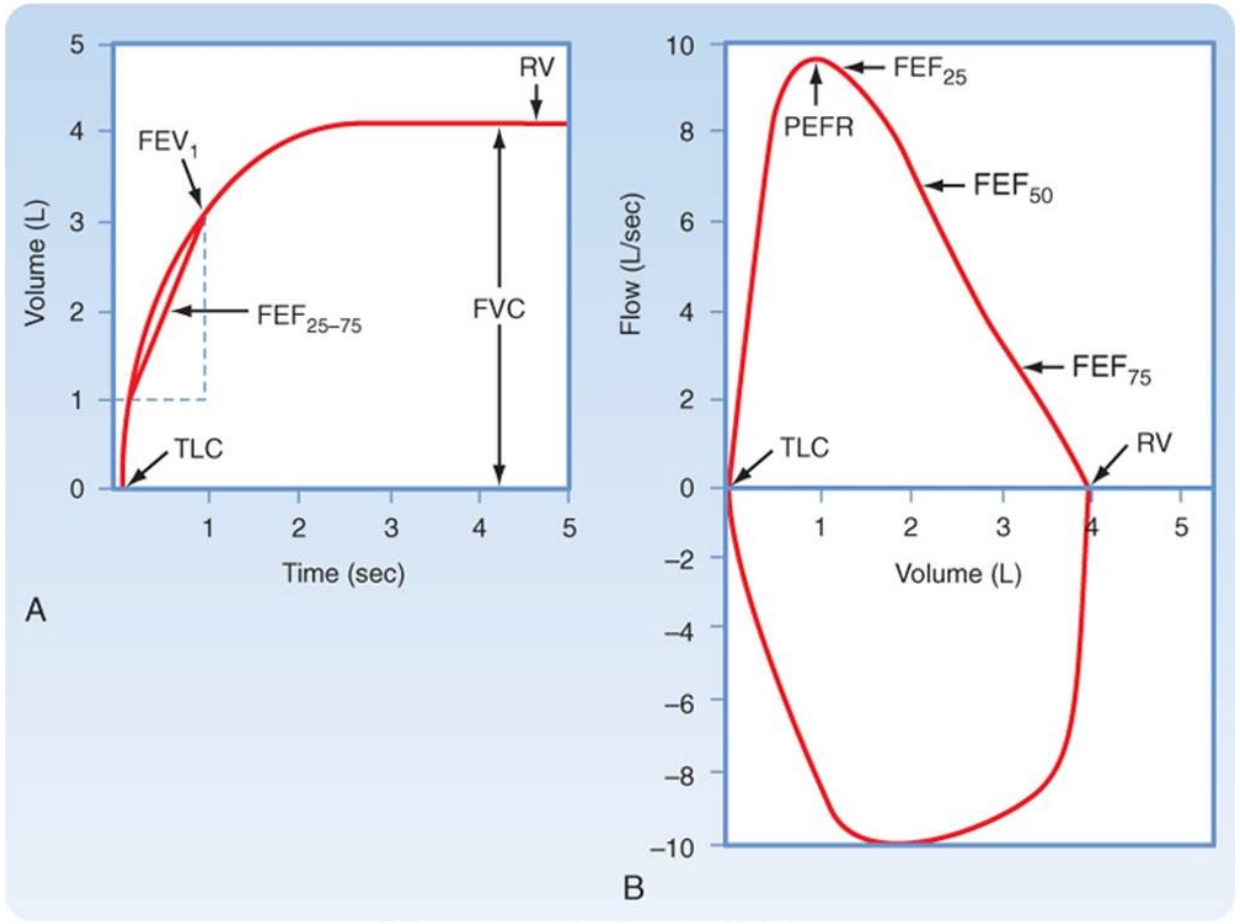
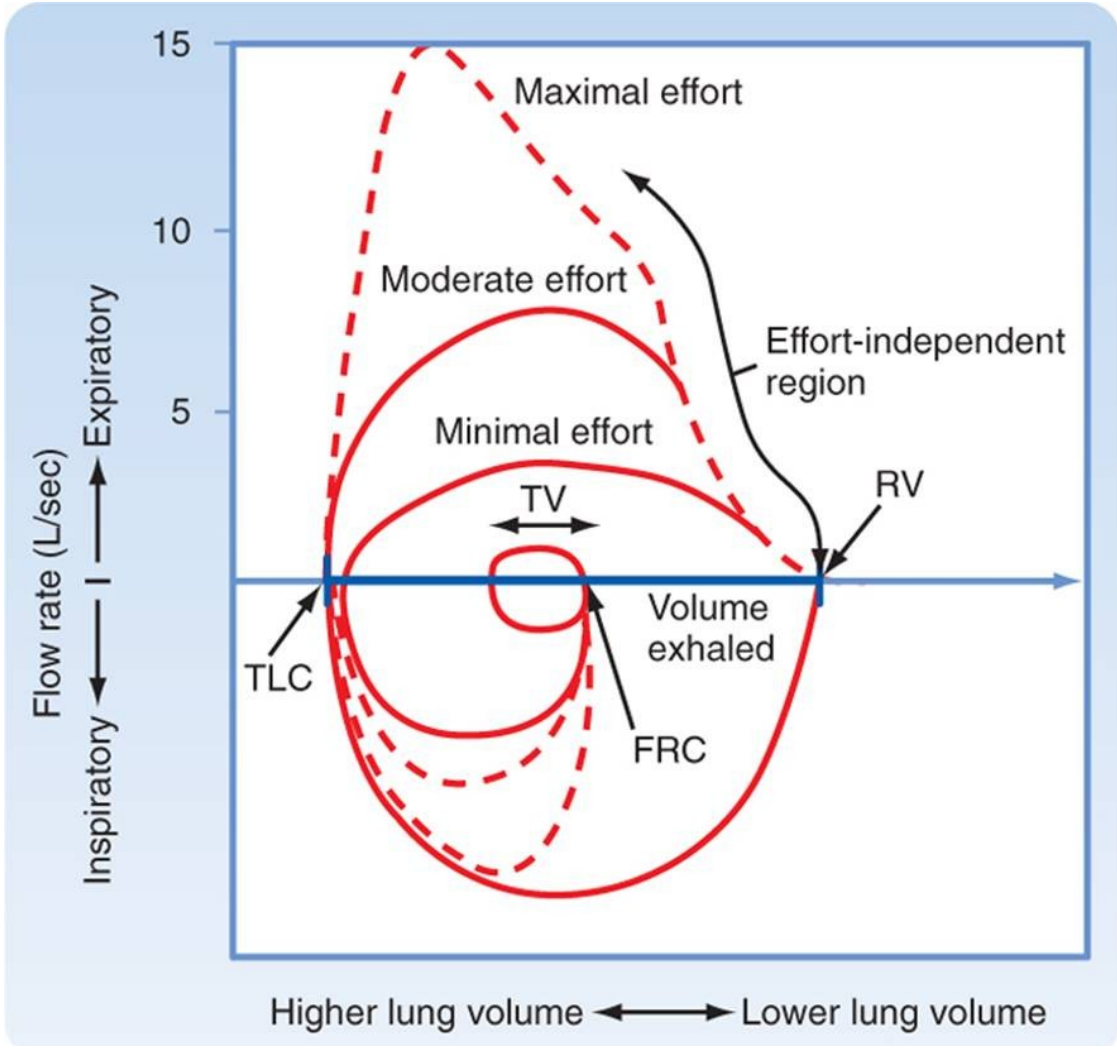
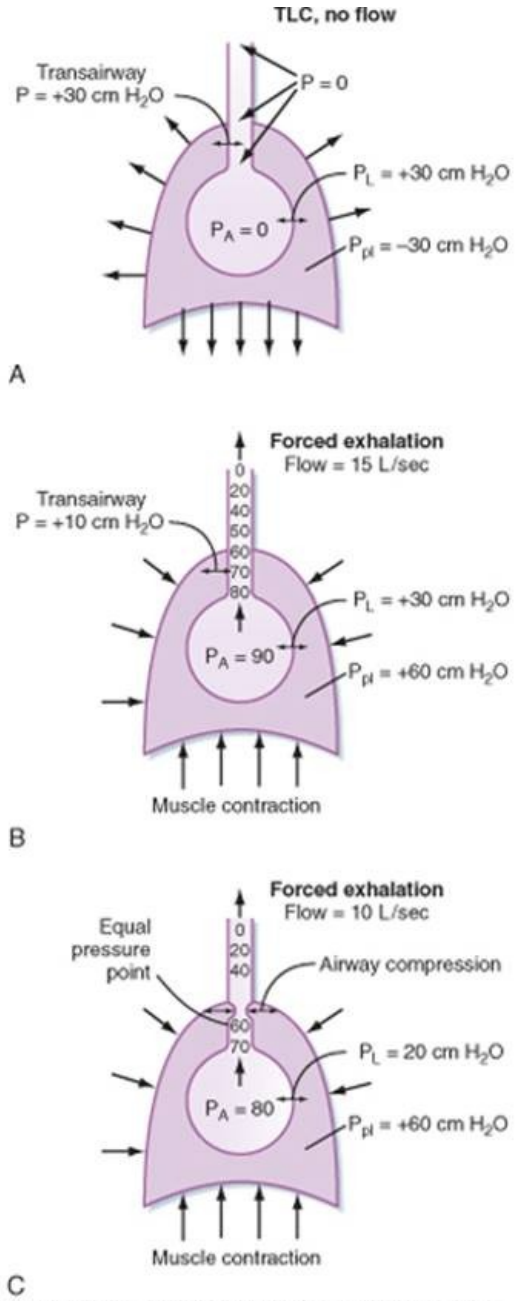
Flow-volume loop: peak inspiratory and expiratory flow rates are dependent on effort, whereas expiratory flow rates later in expiration are independent of effort.





Why is expiratory flow limited?



- In forced expiration, the driving pressure raises both the P_{ALV} and the P_{PL}
  - between the alveolus and the mouth, a point will occur (C) where the airway pressure will equal the intrapleural pressure, and airway compression will occur
    - **equal pressure point**
  - however, this equal pressure point and event. compression of the airway is not fixed during the entire expiration (as the lung volume decreases)
  - initially, it does not existsince in the absence of lung disease, the equal pressure point occurs in airways that contain cartilage, and thus they resist collapse
  - later, the equal pressure point moves closer to the alveoli causing transient occlusion of the airway
    - this, however, results in an increase in pressure behind it (i.e. upstream) and this raises the intra-airway pressure so that the airways open and flow is restored
  - the airways thus tend to vibrate at this point of **'dynamic airway compression'**

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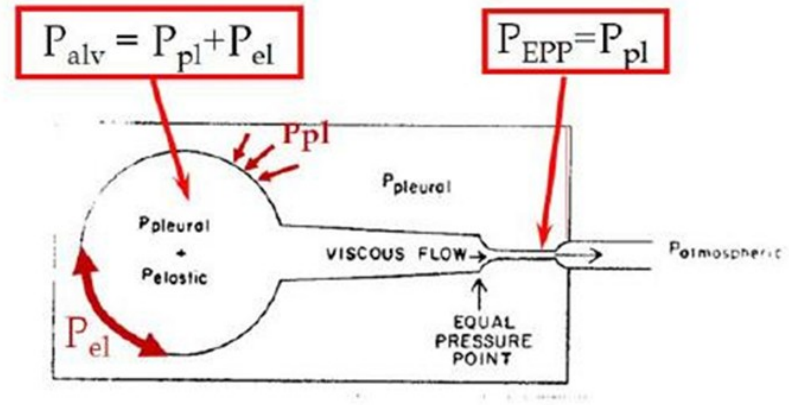
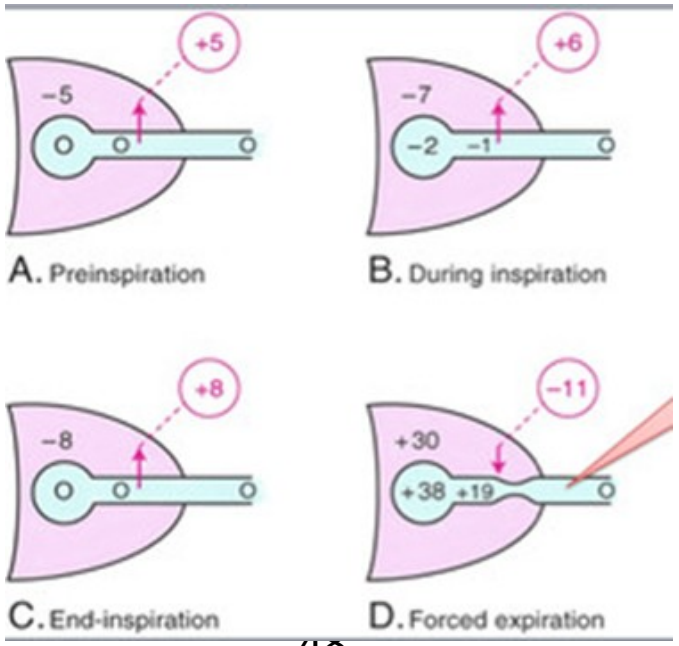
Dynamic airway compression/collapse

- In forced expiration, the driving pressure raises both the P_{ALV} and the P_{PL}
  - between the alveolus and the mouth, a point will occur (C) where the airway pressure will equal the intrapleural pressure, and airway compression will occur
    - **equal pressure point**
  - however, this compression of the airway is temporary, as the transient occlusion of the airway results in an increase in pressure behind it (i.e. upstream) and this raises the intra-airway pressure so that the airways open and flow is restored
    - the airways thus tend to vibrate at this point of 'dynamic compression'





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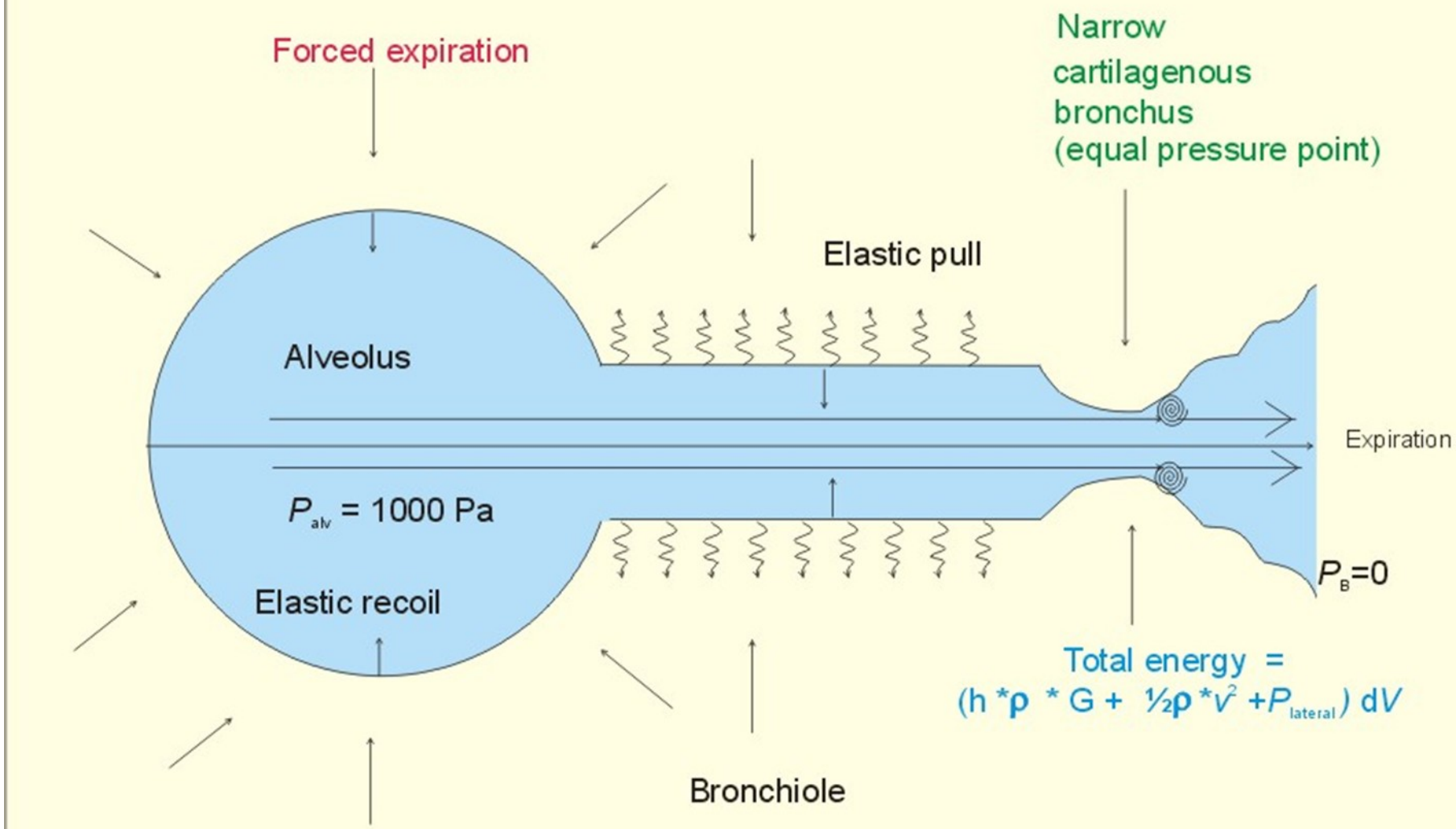
Dynamic Airway Collapse



Expiratory effort --- Increased kinetic energy --- Reduced lateral pressure --- Dynamic Airway Collapse

Fig. 13-5

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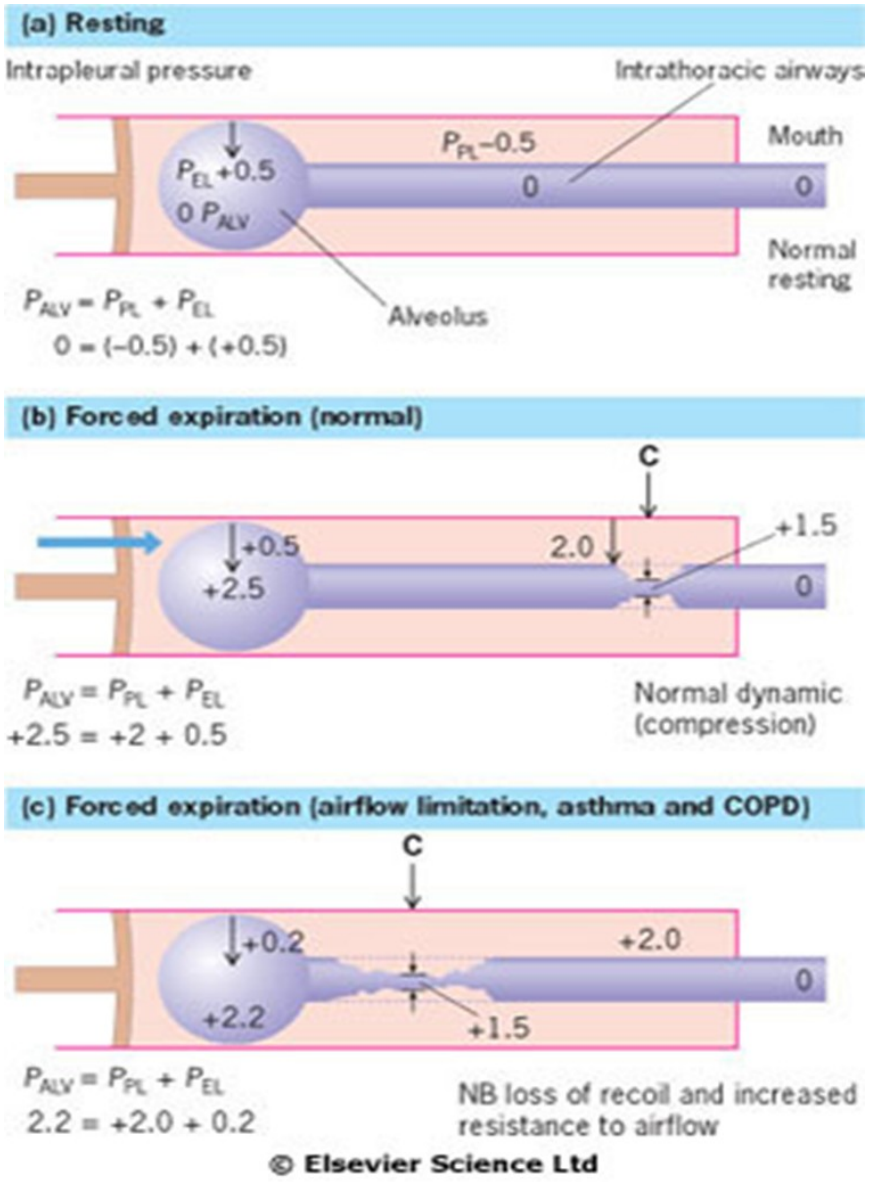
Dynamic compression in various situations



- The respiratory system is represented as a piston with a single alveolus and the collapsible part of the airways within the piston
  - C, compression point; PALV, alveolar pressure; PEL, elastic recoil pressure; PPL, pleural pressure.
  - (a) at rest at functional residual capacity
  - (b) forced expiration in normal subjects
  - (c) forced expiration in a patient with COPD

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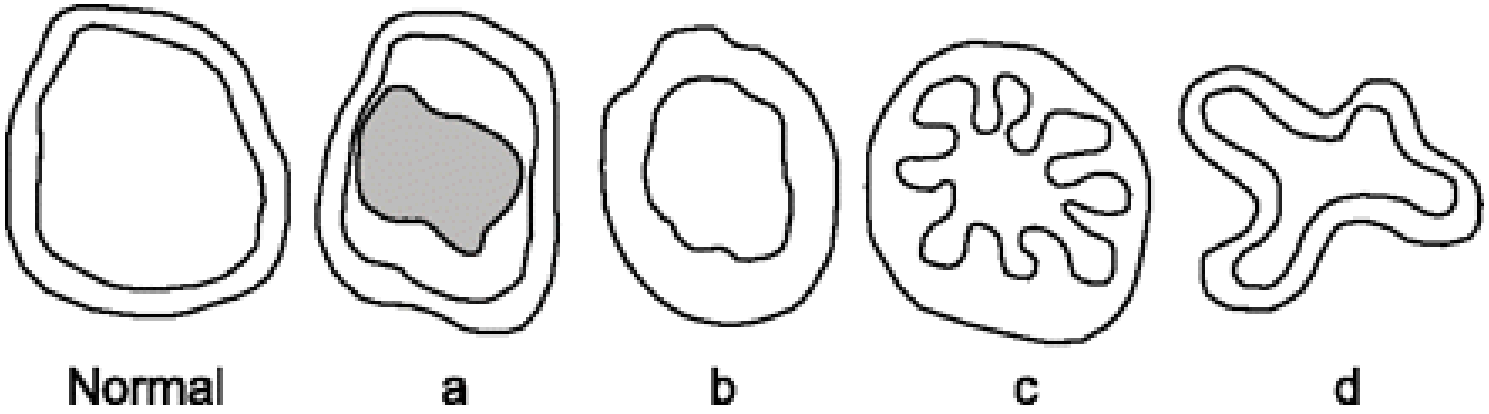
Various mechanisms of airway obstruction



- Narrowing of the airway lumen may be due to:
  - a) **mucus**, cells or other material within the lumen
  - b) thickening of the airway wall that encroaches on the lumen (**hypertrophy**)
  - c) shortening of smooth muscle around the lumen (**bronchoconstriction**)
  - d) collapse of the airway wall into the lumen (**emphysema**)

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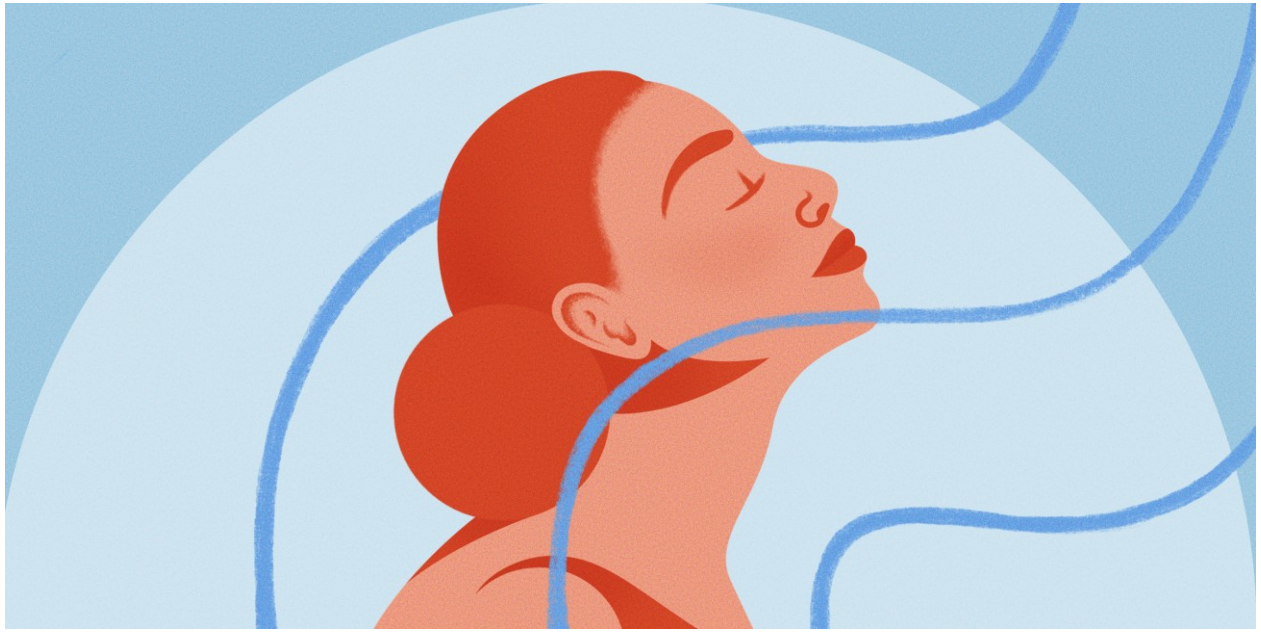
Thanks for your attention!



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