Special Pathophysiology
Summer course
1997 - 2001
2002- 2023
General medicine, Dentistry

PATHOPHYSIOLOGY OF RESPIRATION

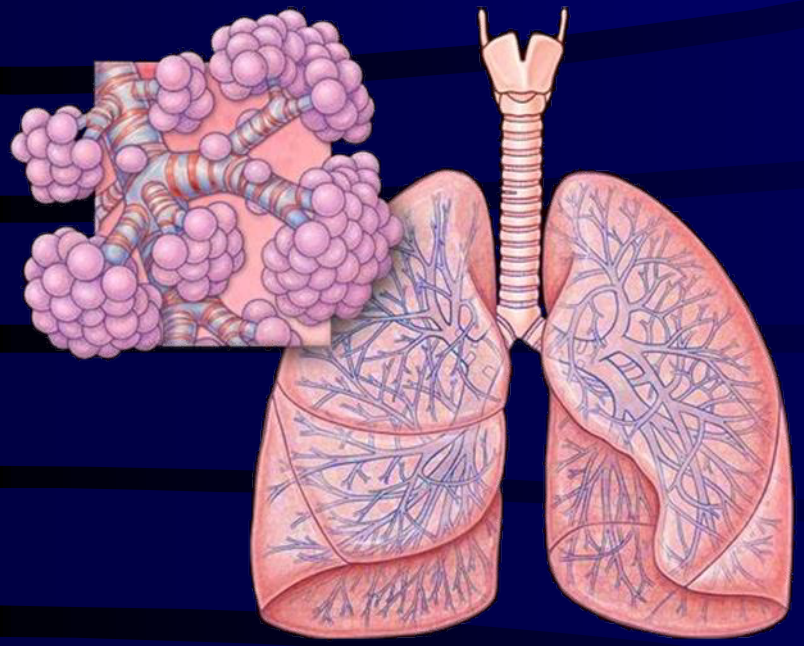
Basics

R. Benacka. MD, PhD

Department of Pathophysiology,

Medical faculty, P.J. Safarik University

Košice. Slovakia

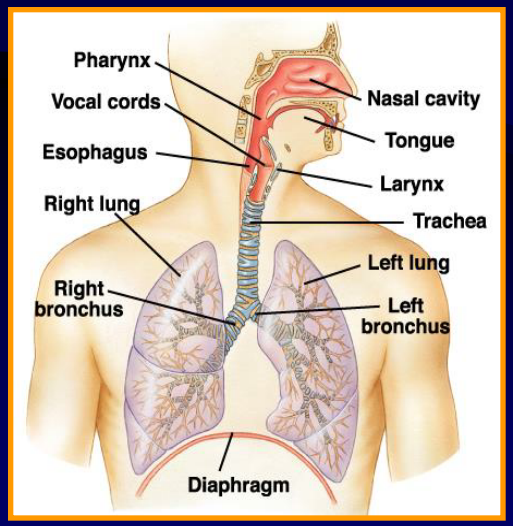


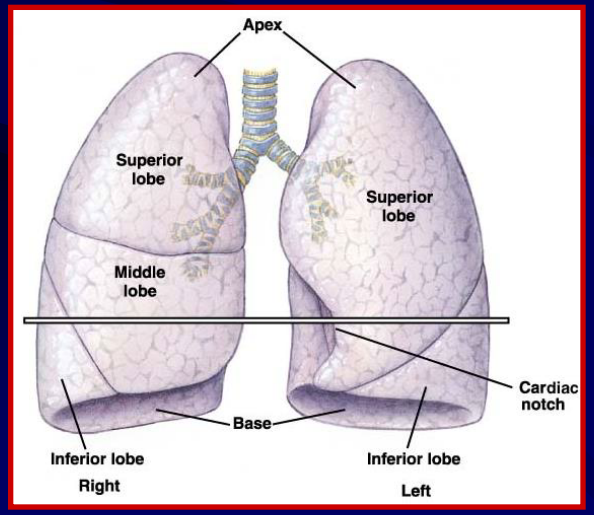
1

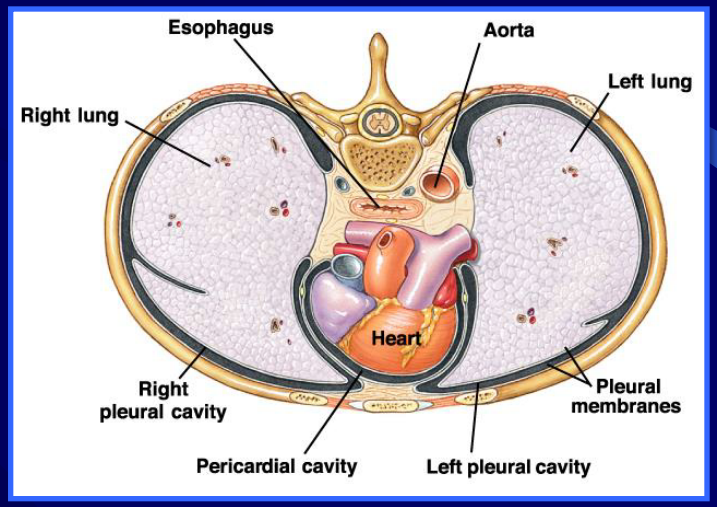
Physiology overview
Structure and function

Role of breathing

- Exchange of gases - inhalation of O_2 into organism; exhalation of CO_2
  - in the rest we do not note even moderate hypoxia (smokers) however even mild hypercarbemia (CO_2) stimulates the breathing (hyperpnoea);
- Exhalation of water
  - expiration of H_2O (2.place after kidneys; in man water is lost by evaporation)
- Regulation of acid-base balance - pH (great importance)
  - exhalation of volatile acids by CO_2 <- H_2CO_3 < $H^+ + HCO_3^-$
- Acquired immunity - broadest contact with external antigens chemicals; effective local innate protection; very rich lymphathetic drainage| lymph nodes
- Other involvements = speech, eating, drinking sucking, defecation,

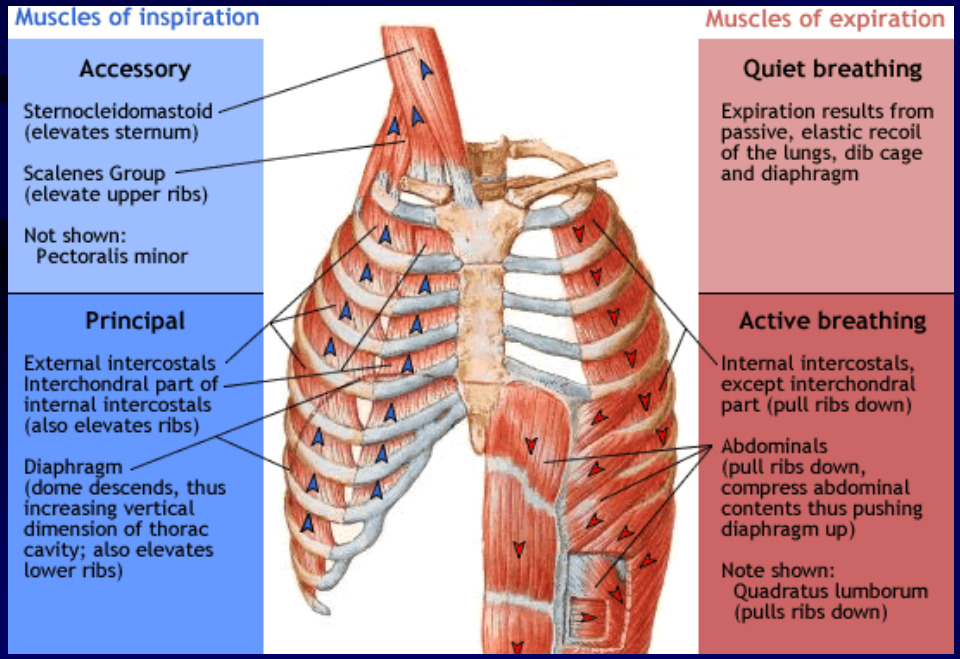


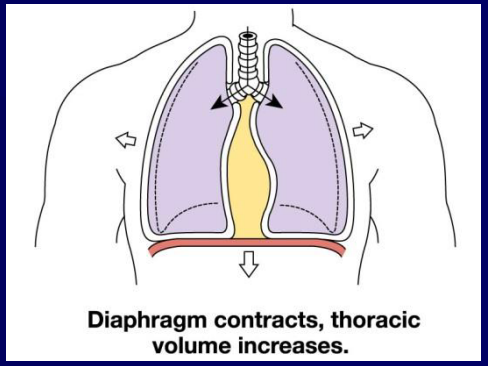


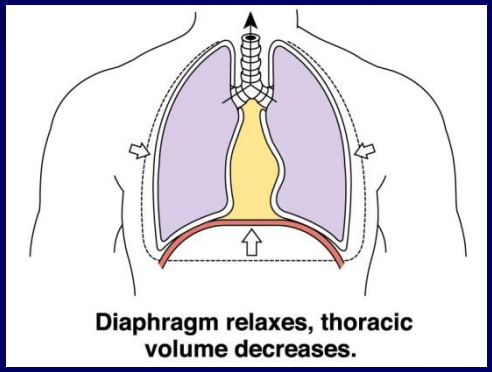


Muscles of breathing

- Breathing = vital function but not vegetative one;; vegetative system does not generste nor regulate breathing but centrally modulate breathing (catecholaminergic nuclei in brainstem)
- Periodic breathing movements = performed by skeletal striated muscles (diaphragm = principal, intercostal muscles, accessory mm.)
- All disorders affecting skeletal muscles may affect breathing (e.g.traumatic damage of spinal cord paralysis C4-C6, poliomyelitis, myasthenia gravis, Duchenne dystrophy etc.)
- Disorders, toxins affecting neuromuscular junction may affect breathing (e.g. muscle relaxants, toxins like VX substance, organic phosphates, cobratoxin, kurare etc,

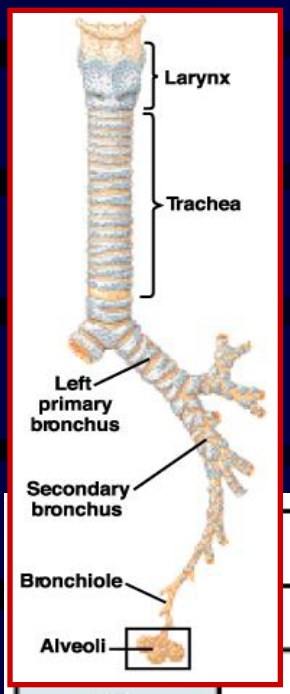




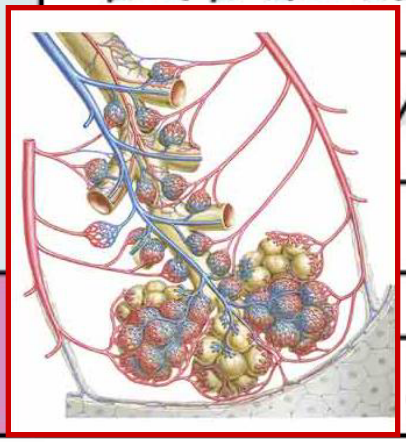


https://www.physio-pedia.com/Muscles_of_Respiration

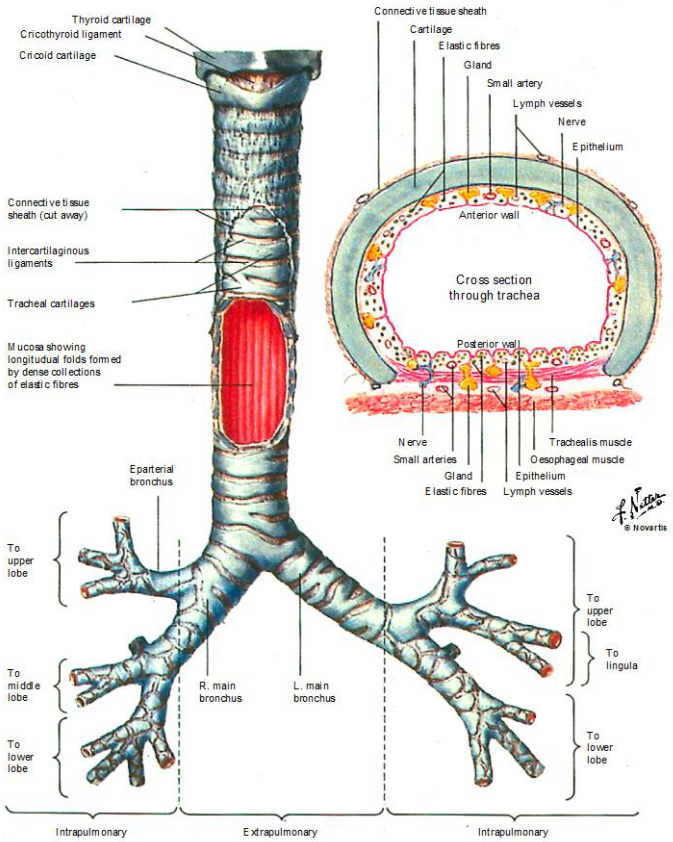
Lung diffusion area

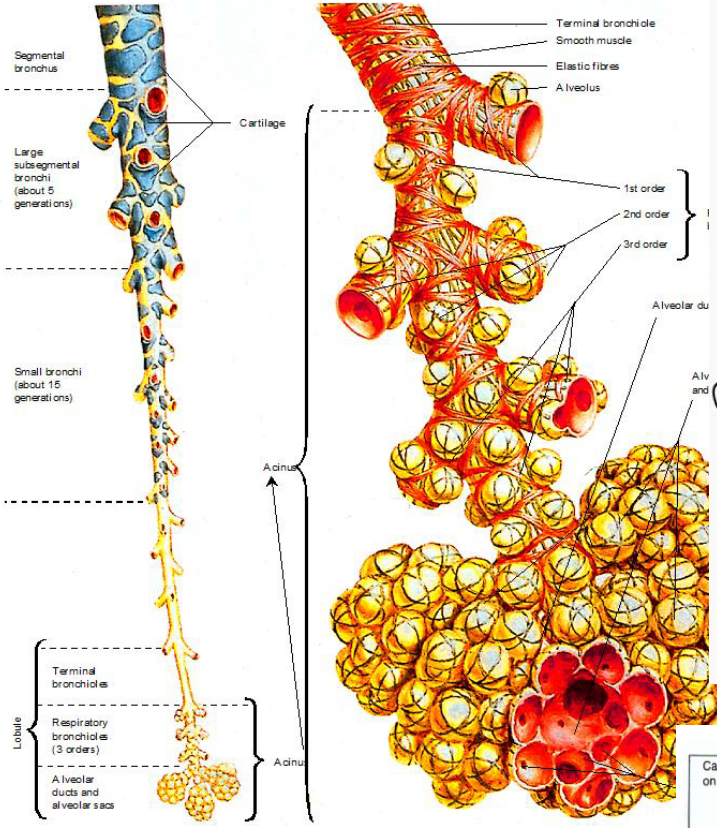


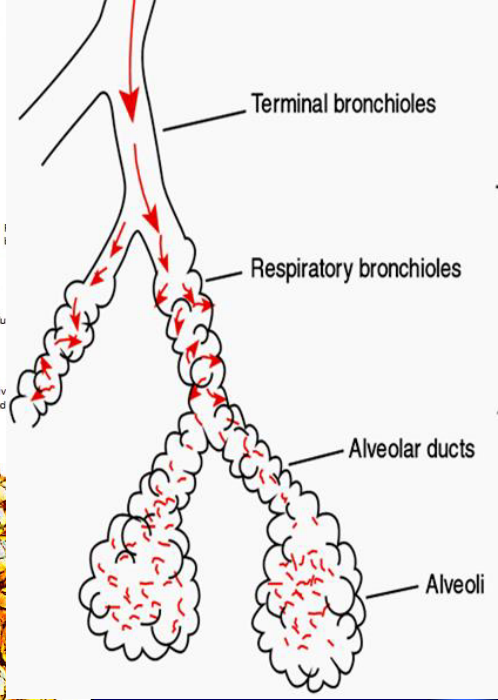
- Alveolar surface - 100m^2 (large flat); not all are used simultaneously; only protion is working
- Perfusion redundancy – 1000 capillaries /1 alveole (exchange of gases even in weak ventilation);gravitationally basal and central lungs are perfused better, apex less

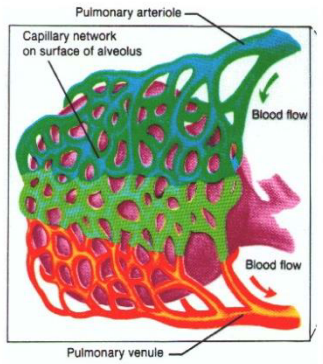


	Name	Division	Diameter (mm)	How many?	Cross-sectional area (cm)
Conducting system	Trachea	0	15-22	1	2.5
	Primary bronchi	1	10-15	2	↓
	Smaller bronchi	2	1-10	4 ↓	↓
		3			
		4			
		5			
		6-11		1×10^4	↓
Exchange surface	Bronchioles	12-23	0.5-1	2×10^4 ↓ 8×10^7	100 ↓ 5×10^3
	Alveoli	24	0.3	$3\text{-}6 \times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



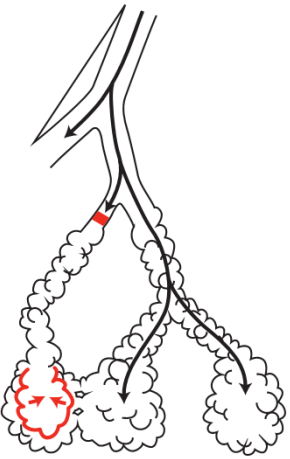




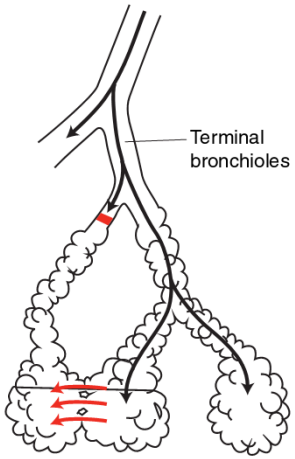


Alveolar-capillary contact surface area is estimated at from 50 to 400 square meters.

Phtomicrograph of alveolar capillaries

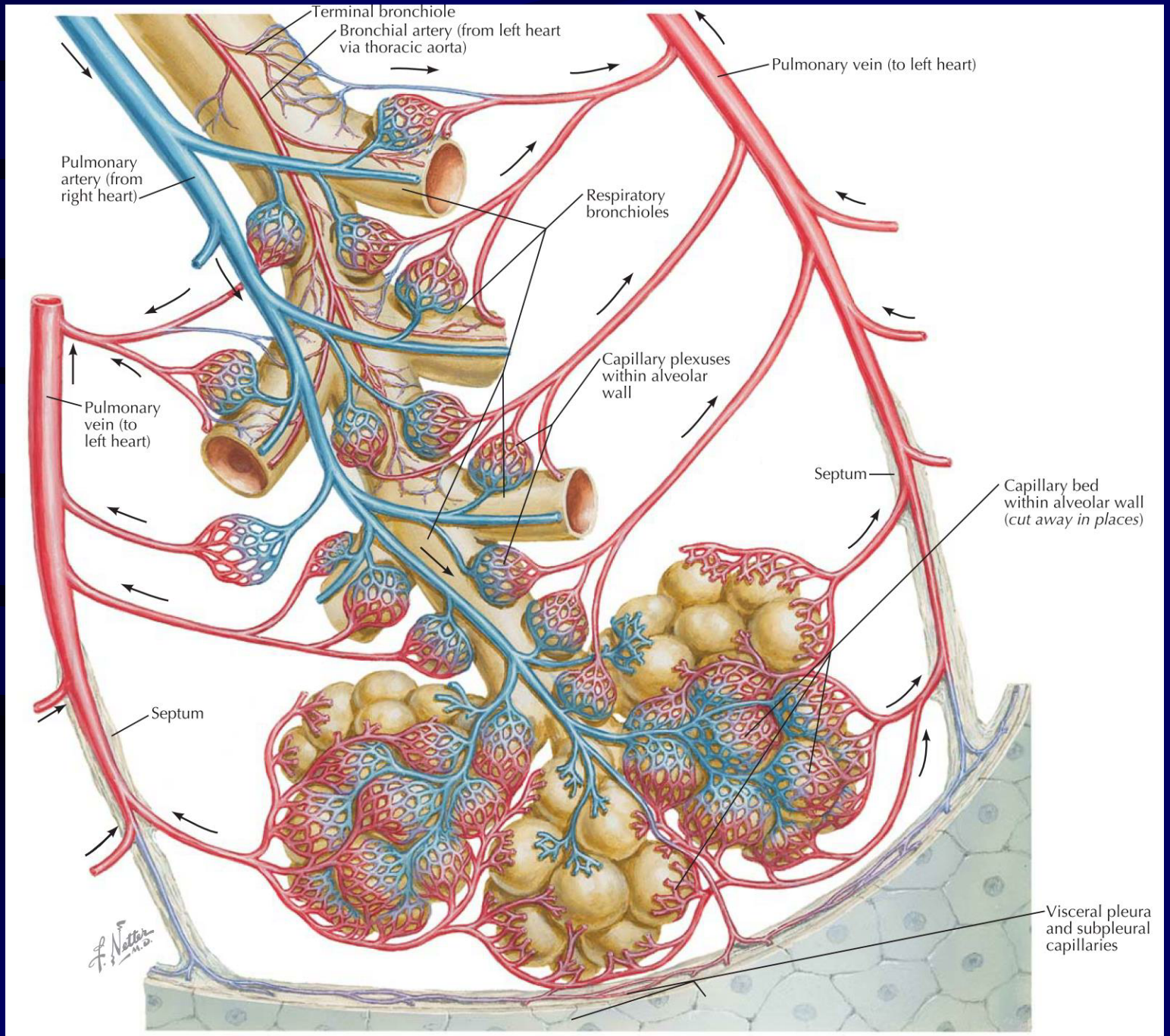


B Blockage leading to potential collapse of alveoli



C Collateral flow through pores of Kohn works against collapse

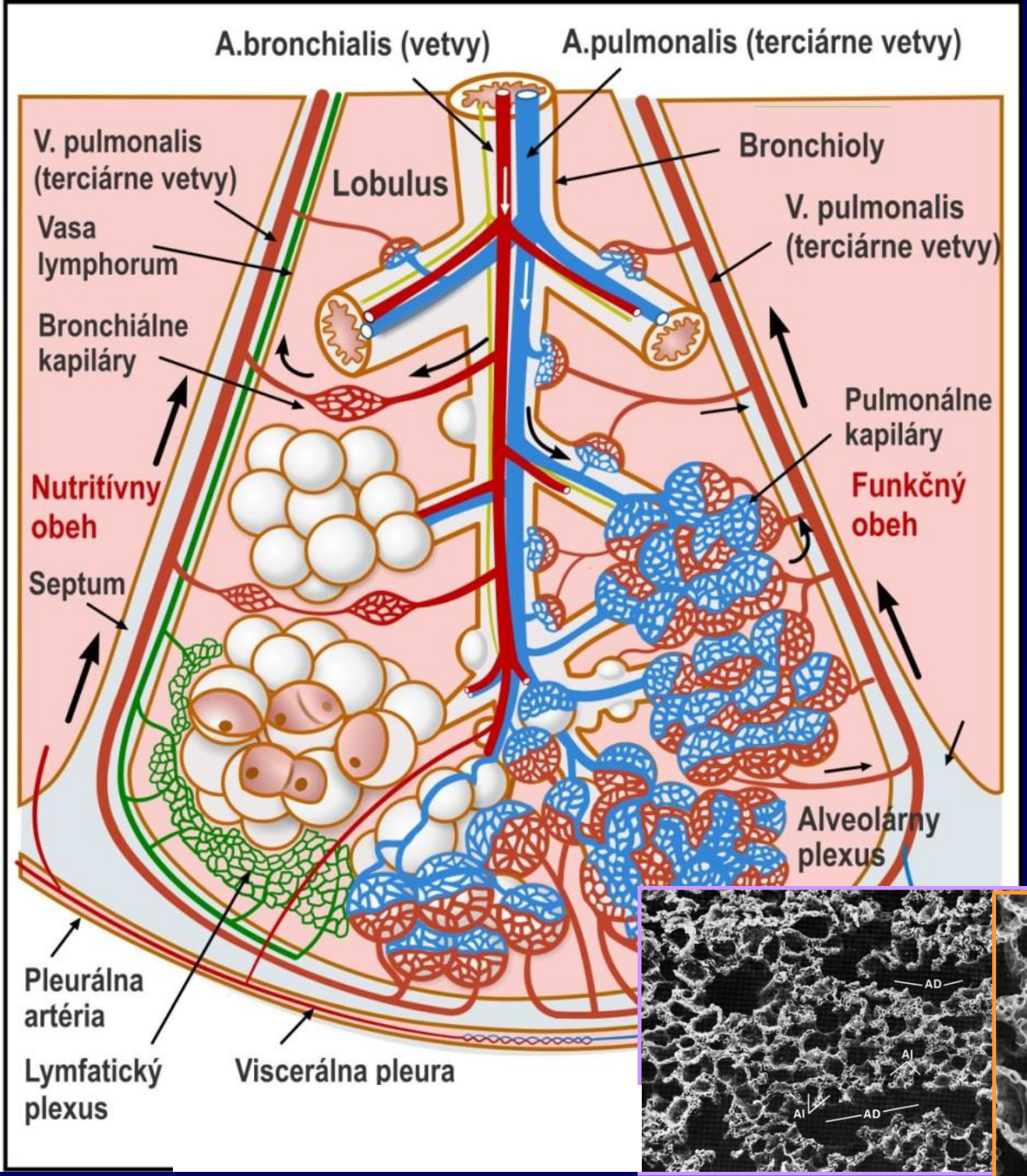
- Spontaneous recollapsing - atelectasis
- Kohn pores –communication in between alveolar spaces
- Tubulency in trasient zone deposist microparticles
- Not all parts of lungs are ventilated or perfused at once.

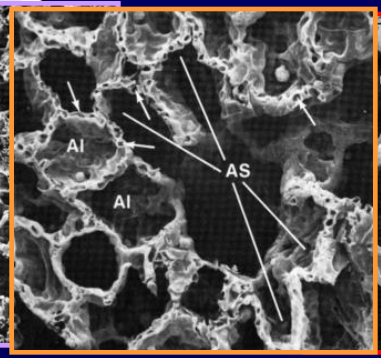


Pulmonary arteries and their branches distribute segmentally with the bronchi. Pulmonary veins and their tributaries drain intersegmentally.

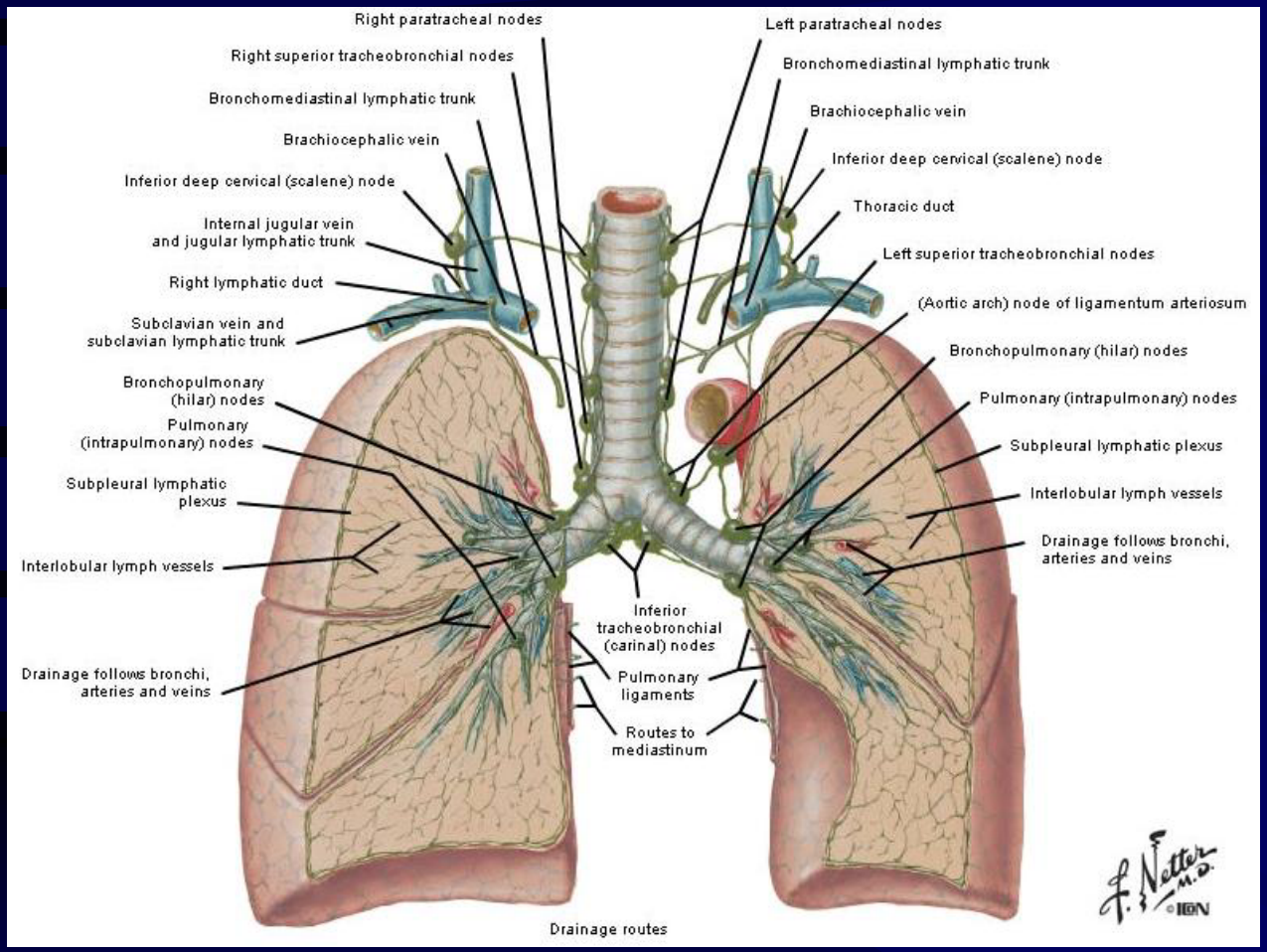
Broncho-pulmonary segment

- The lungs have 2 circulations - functional and nutritive
- System of a. pulmonalis - oxygenation of the blood in the alveoli
- System of aa.bronchiales - oxygenated blood, deeper peribronchial supply
- Abundant lymphatic drainage - peribronchial, peritracheal nodes
- Pleural connections





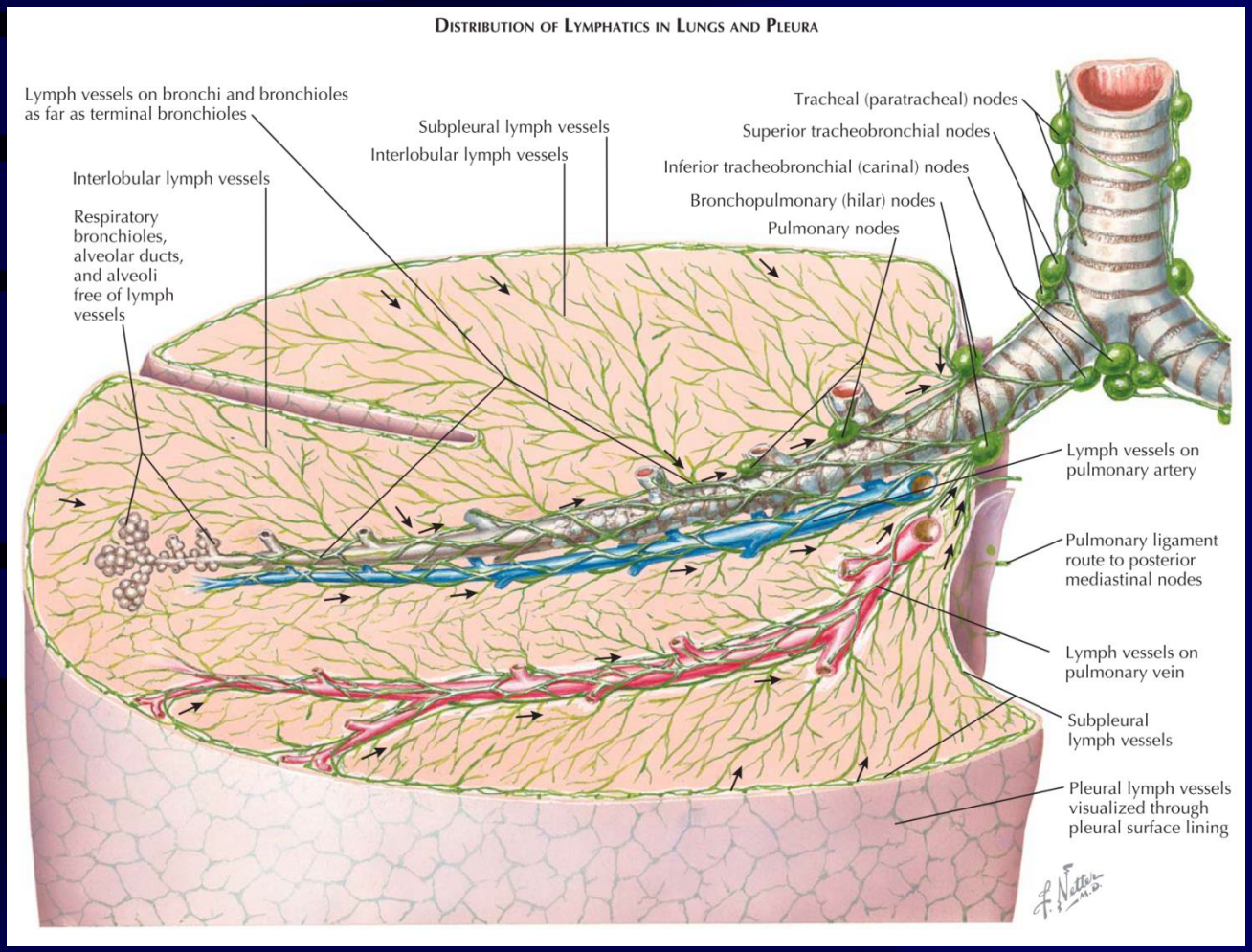
Lymphatic drainage



- Lungs are equiped with effective lymphathetic drainage, that removes intersttitial fluid
- **Peribronchovascular** majority of lymphatics are around large bronchi and blood vessels
- **Pleural Lymphatics:** A separate network in the visceral pleura,/ subatmospheric pressure to operates at a very low hydraulic pressure (-5 cmH_2O to -30 cmH_2O),
- Possess "valves but lack smooth muscle cells,“
- Not present in the alveolar septa themselves

- **Lobe-Specific Patterns:** right lung has a higher propensity for direct drainage
- **Pressure-Dependent Drainage, "Safety Factor":**when fluid accumulates, pressure increase opens "anchoring filaments" on the lymphatic capillaries, increasing fluid uptake

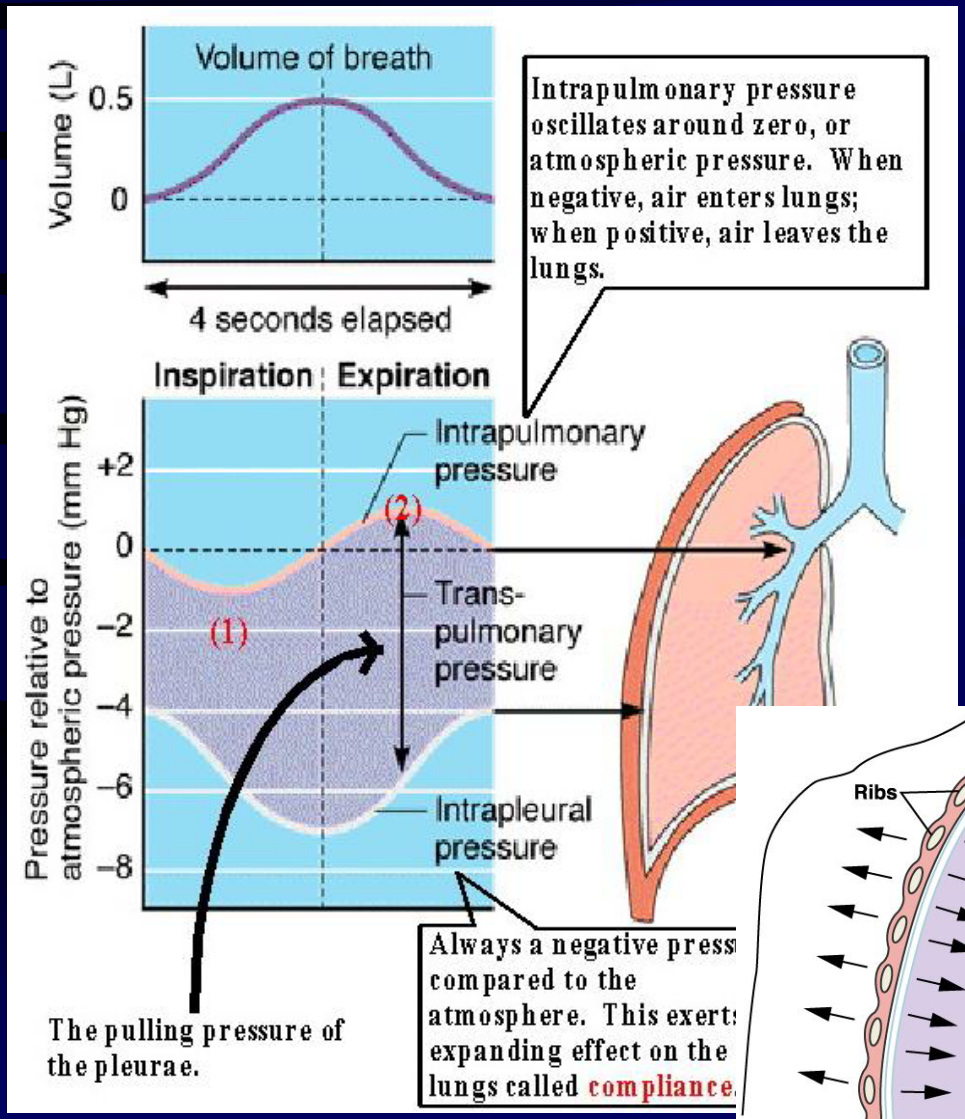
Lymphatic drainage



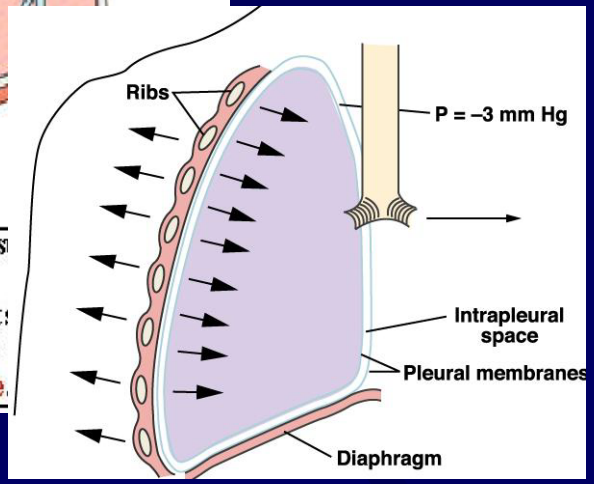
- Keeps the lungs dry
- Lungs are the principal area in the body that is exposed to exterior antigens incl. pathogens
- the barrier against the spread of pathogens and schooling of immunolog. complelent cells
- Dendritic cells patrol the parenchyma; migrate every 1-2 days to the mediastinal lymph nodes,
- **Mediastinal Bypass ("Skip" Metastases):** 20–38% of lung cancers spread to mediastinal lymph nodes, bypassing the hilar lymph nodes

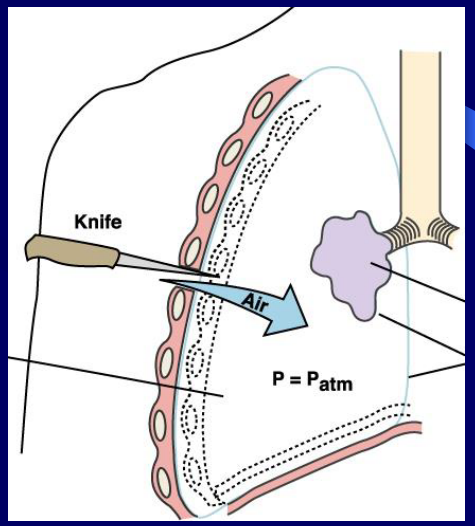
- **Tertiary Lymphoid Organs (TLOs):** Impaired drainage in the lung specifically results in the formation of TLOs, also known as inducible bronchus-associated lymphoid tissue (iBALT), as a mechanism to handle accumulating immune cells.
- **Cigarette Smoke** causes thrombosis of the lymphatic vessels

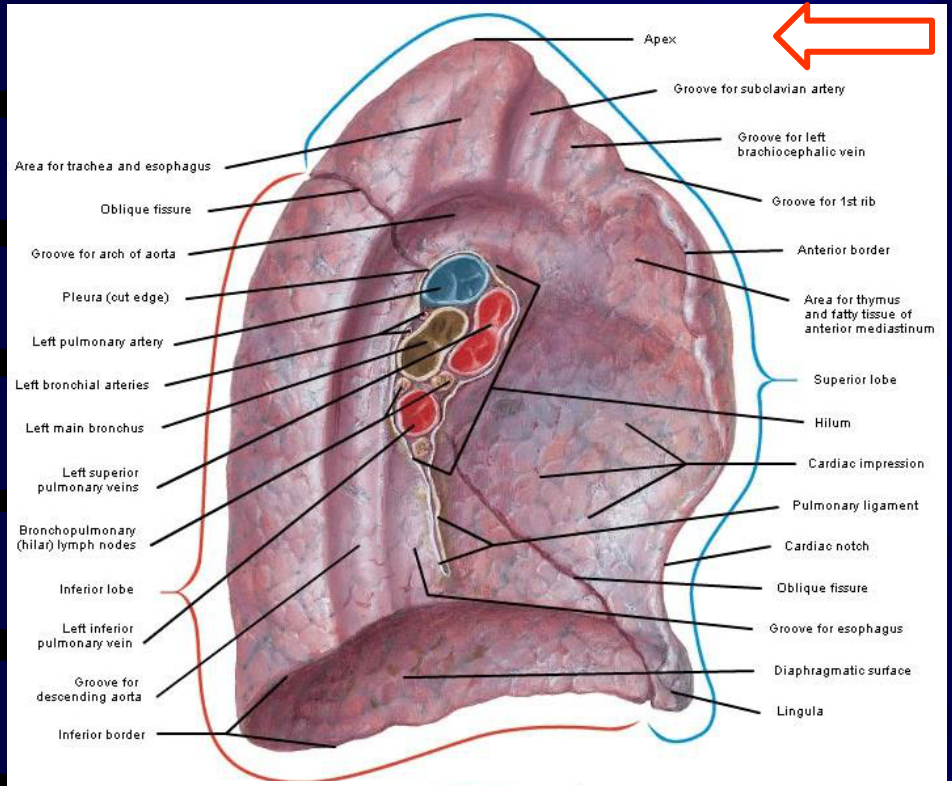
Intrathoracic pressures

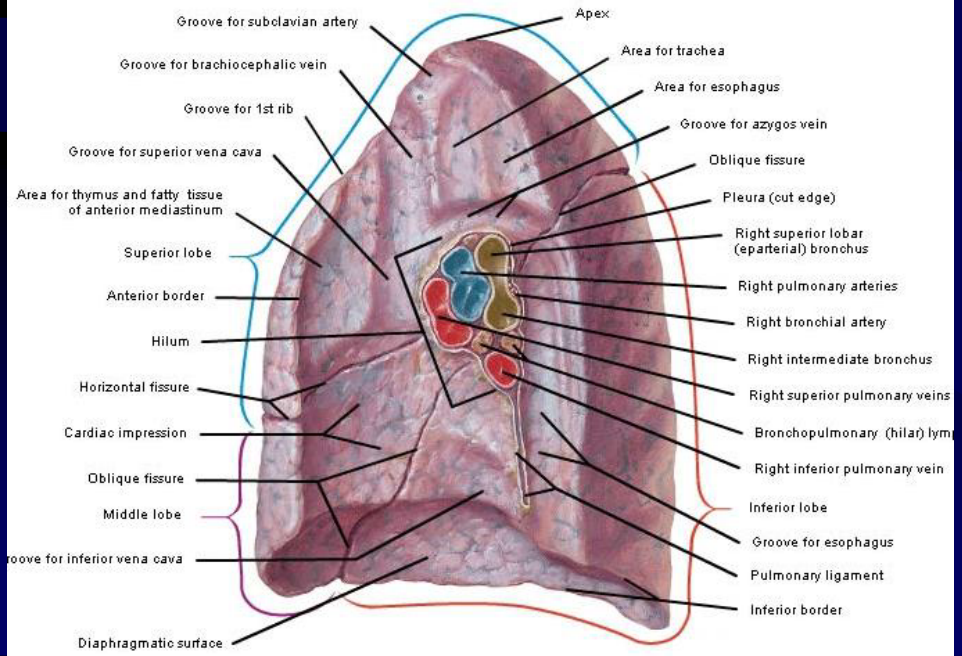


- Intrapulmonary pressure, (IPP) – the pressure inside the lungs decreases during inspira-tion (-1 to -3 mmHg and increases during expira-tion (+1 to +3 mmHg) comp. to atmosph.
  - The lowest IPP is reached in halfway into inspiration
  - The highest IPP is in halfway to exspiration – release of air
- Intrapleural pressure = always negative as compared to the atmosphere: -4 mmHg at the end of expiration, -8 mmHg at the end of inspiration.





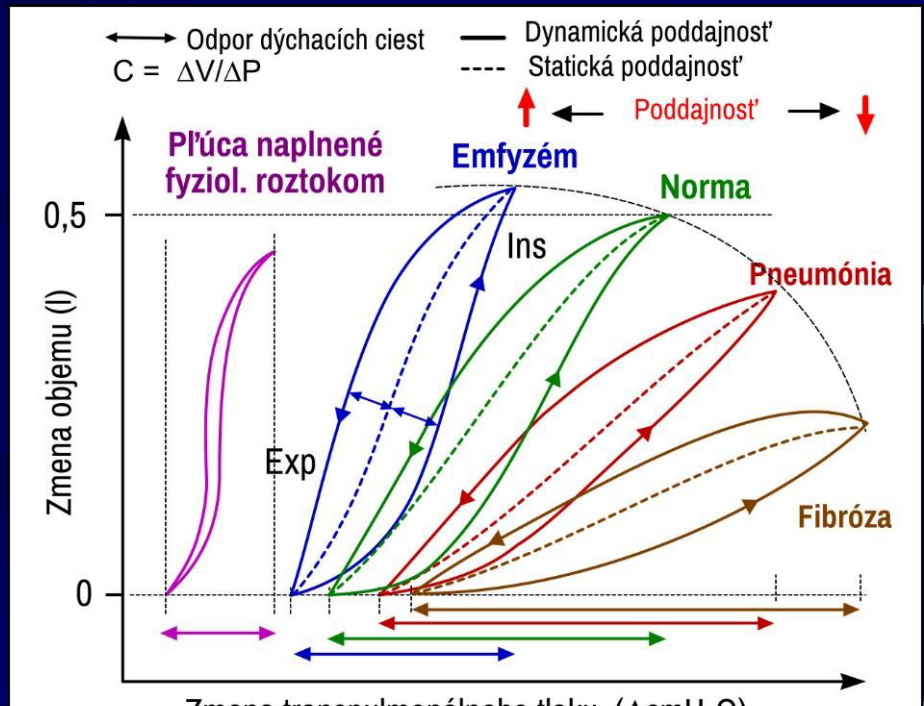


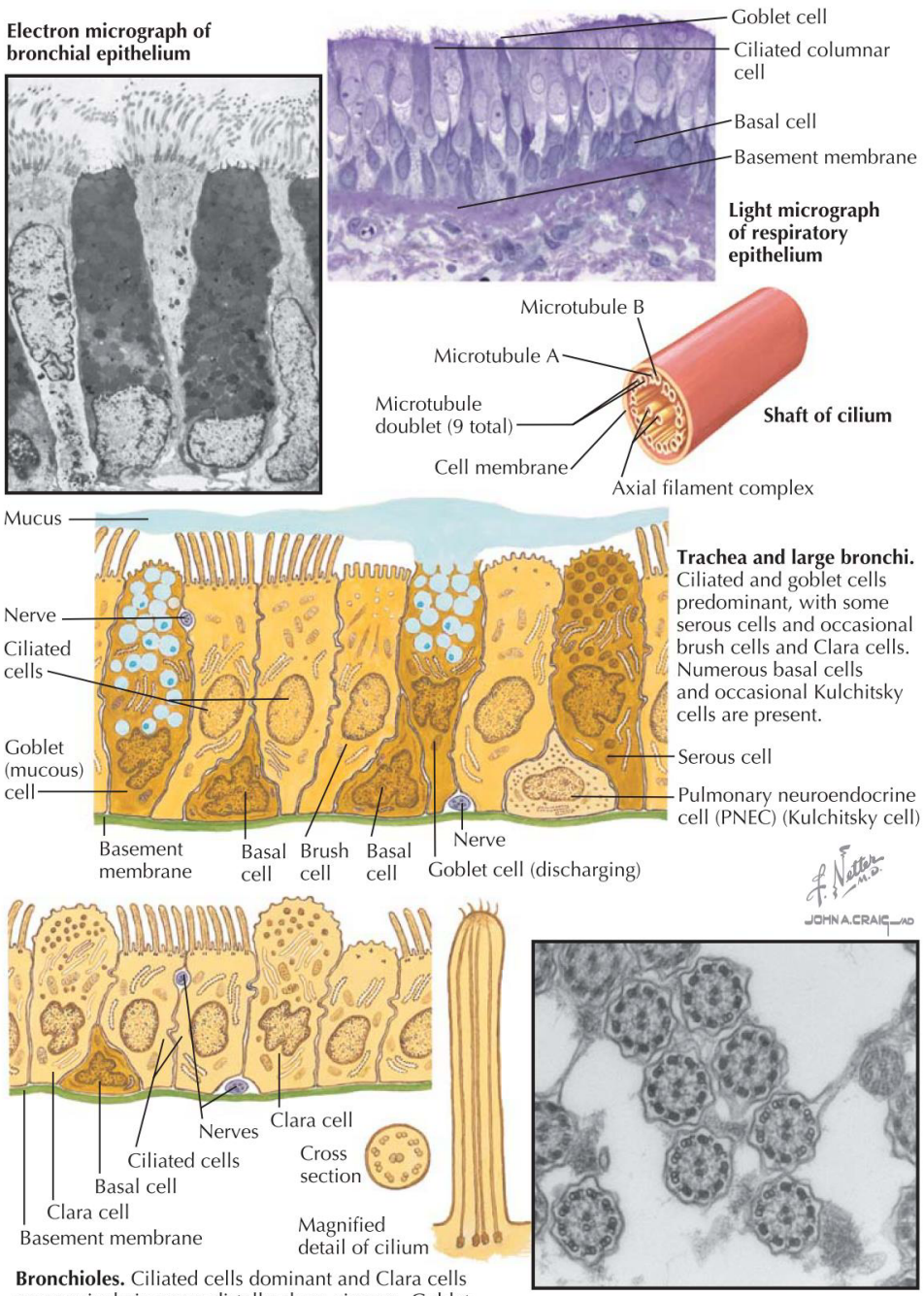


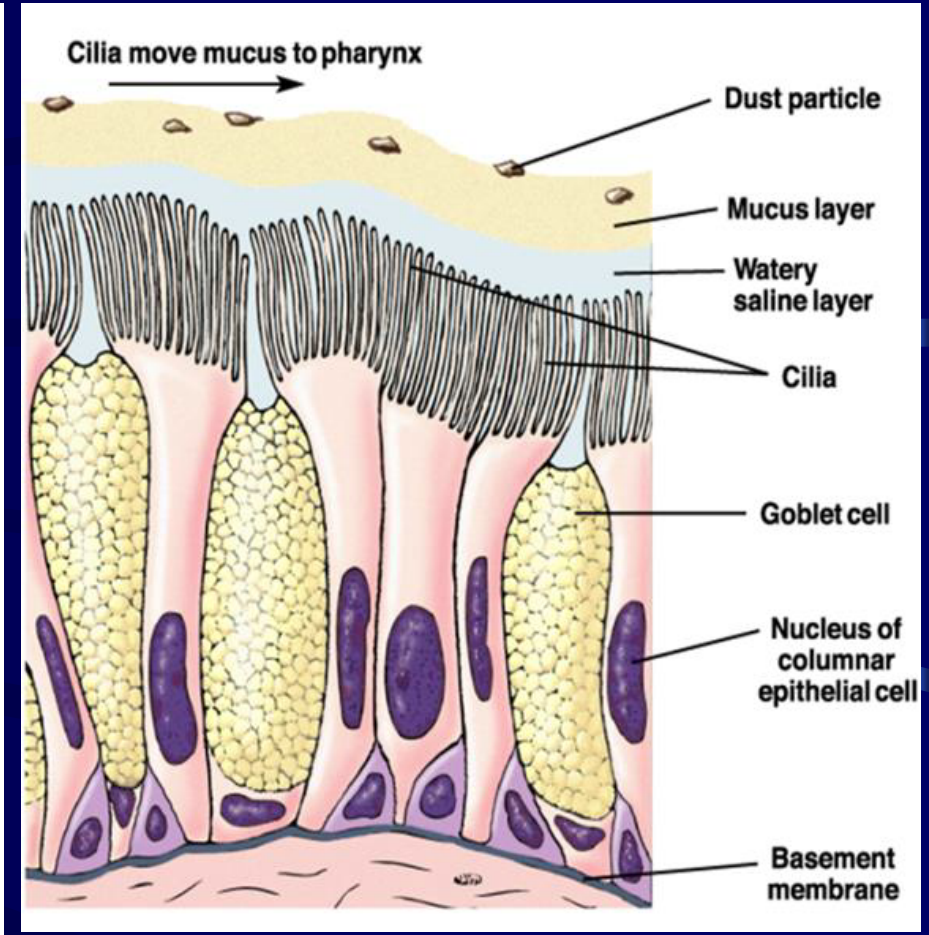
Elastance (recoil) - return to resting volumes due to elasticity of the stroma and relaxation of diaphragm = a measure of the work that has to be exerted by the muscles of inspiration to expand the lungs.

Compliance (distensibility) lung's ability to stretch and expand (of elastic tissue).

- Static lung compliance = change in volume for any given applied pressure.
- Dynamic lung compliance = is the compliance of the lung at any given time during actual movement



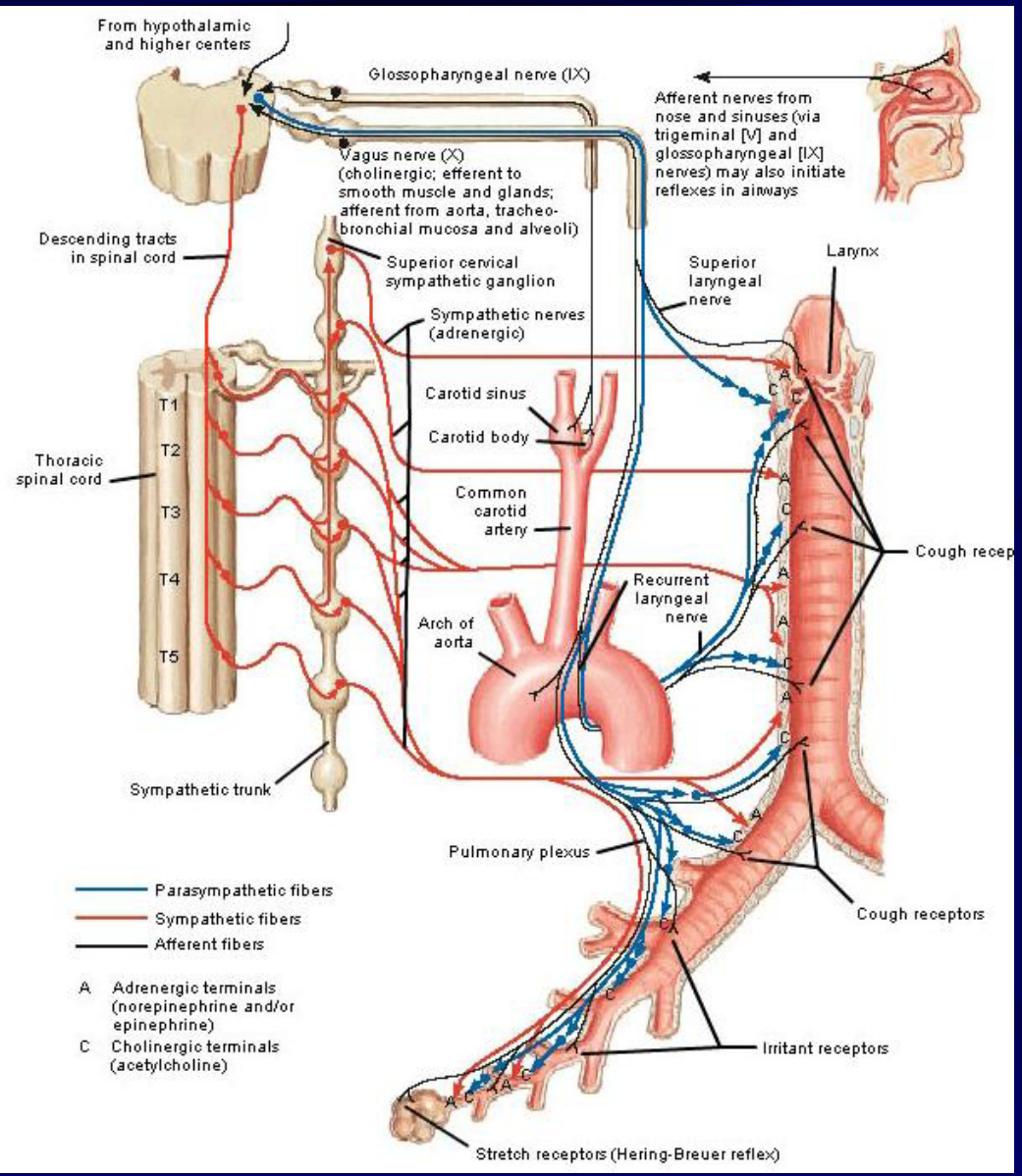




Cilliary transport defects

- Smoking, irritative gases,
- Silicoses, pneumoconioses
- Primary ciliary dyskinesia
- Cystic fibrosis (AR, Cl- transporter)

Innervation of breathing pathways



Innervation:

- n. V, n. IX, (none, nasopharynx) n. X (oro-pharynx, hypopharynx, larynx) : reccurent la-ryngeal n. (RLN) ; sup. laryngeal nerve (SLN)
- Cervical & throacic sympathetic fibres – muscles & secretion

Afferents:

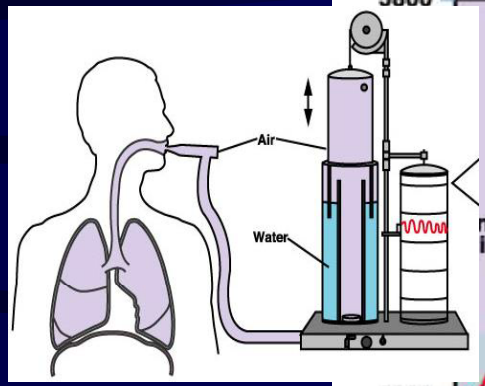
- Mechanoreceptors incl. pulmonary stretch receptors,
- Mechano / chemoreceptors/ polymodal nociceptors: - irritant receptors, J- receptors
- Chemoreceptors – O_2, pH, CO_2 (n.IX, n.X) (carotic & aortic bodies; alveolar ducts)

Respiratory volumes & capacities

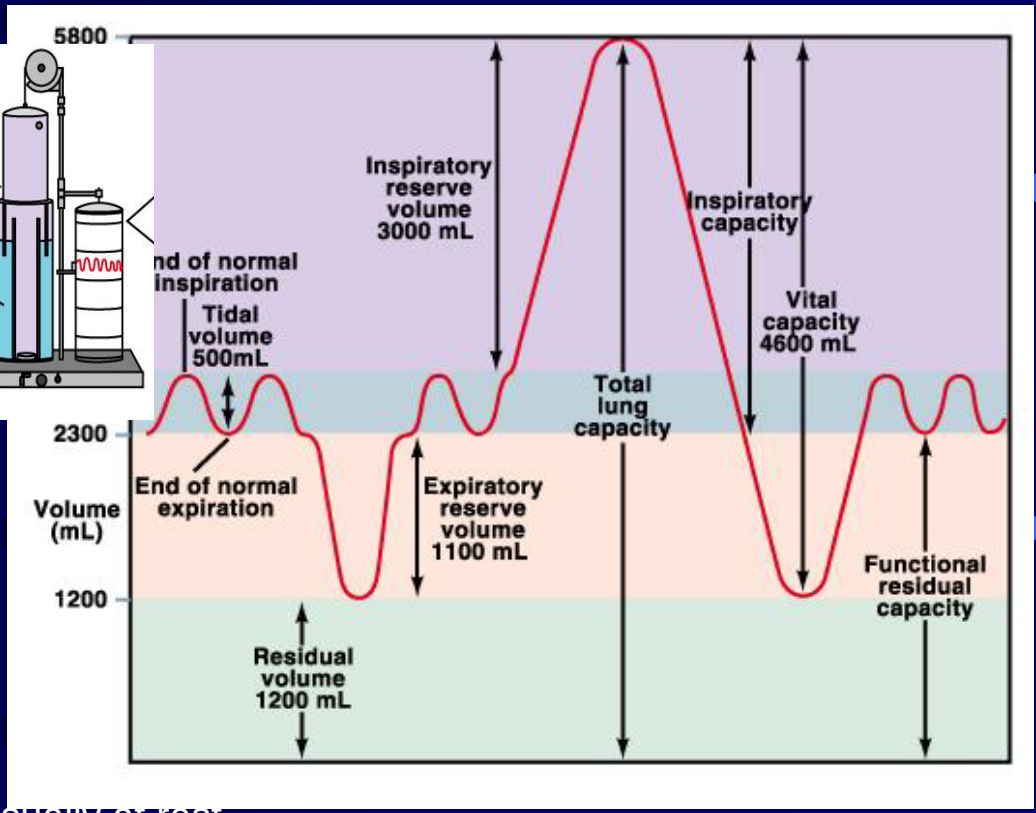
Minute Volume =

$V = V_T$ x resp. rate

e.g., 0.5 L/breath x 12 breaths/min = 6 L/min



- **VT = Tidal volume**
- **ERV = expiratory reserve vol**
- **IRV = inspiratory reserve vol**
- **RV = residual vol**
- **FRC = functional residual capacity**
- **Vital capacity**
- **Total lung capacity**



Tidal Volume VT: Volume of a single breath, usually at rest.

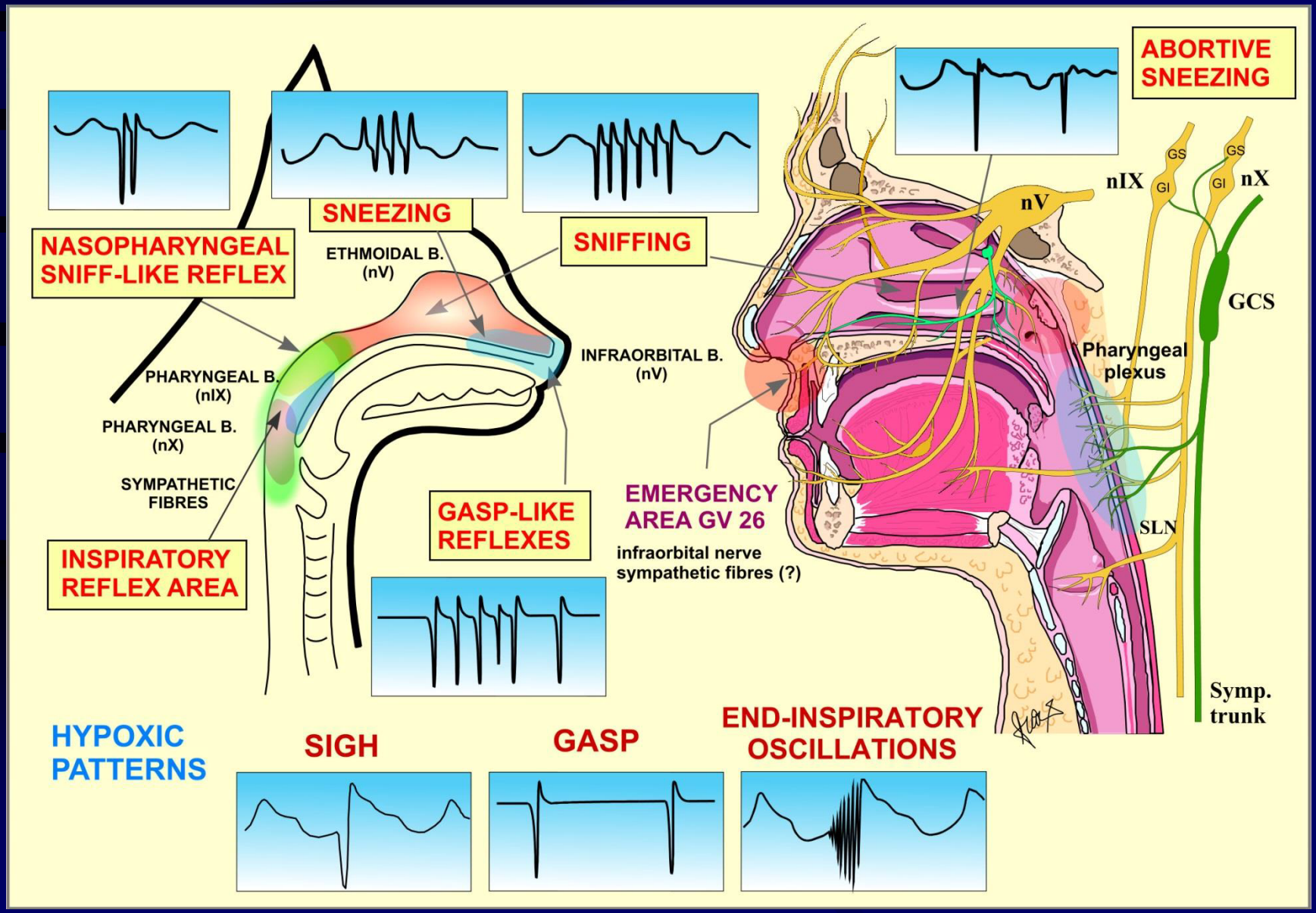
Inspiratory Reserve Volume IRV: Volume which can be inspired beyond a restful inspiration.

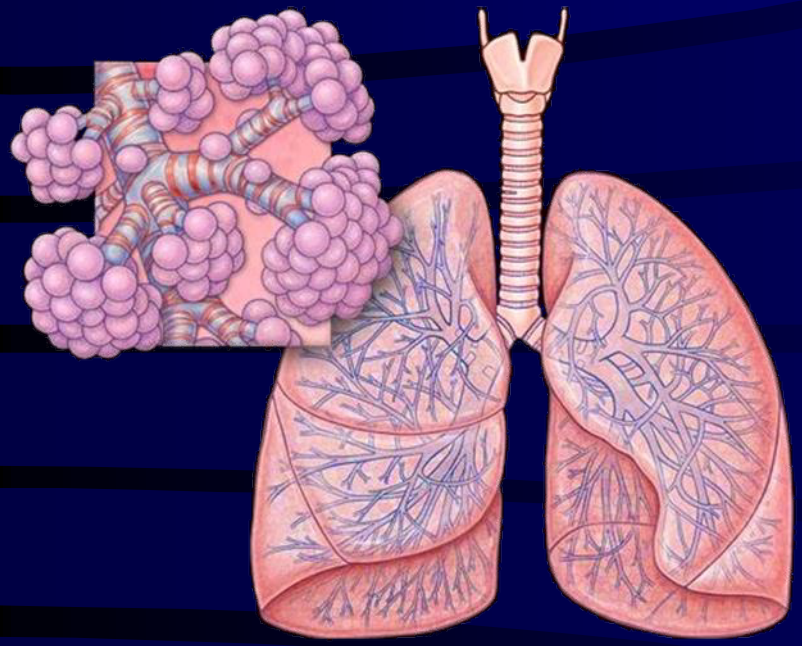
Expiratory Reserve Volume ERV: Volume which can be expired beyond a restful expiration.

Residual Volume RV: Volume remaining in the lungs after a maximum expiration. This volume keeps the alveoli inflated.

Vital Capacity: The vital capacity (VC) is the maximum volume which can be ventilated in a single breath. VC= IRV+TV+ERV. VC varies with gender, age, and body build.

Airway protective refexes

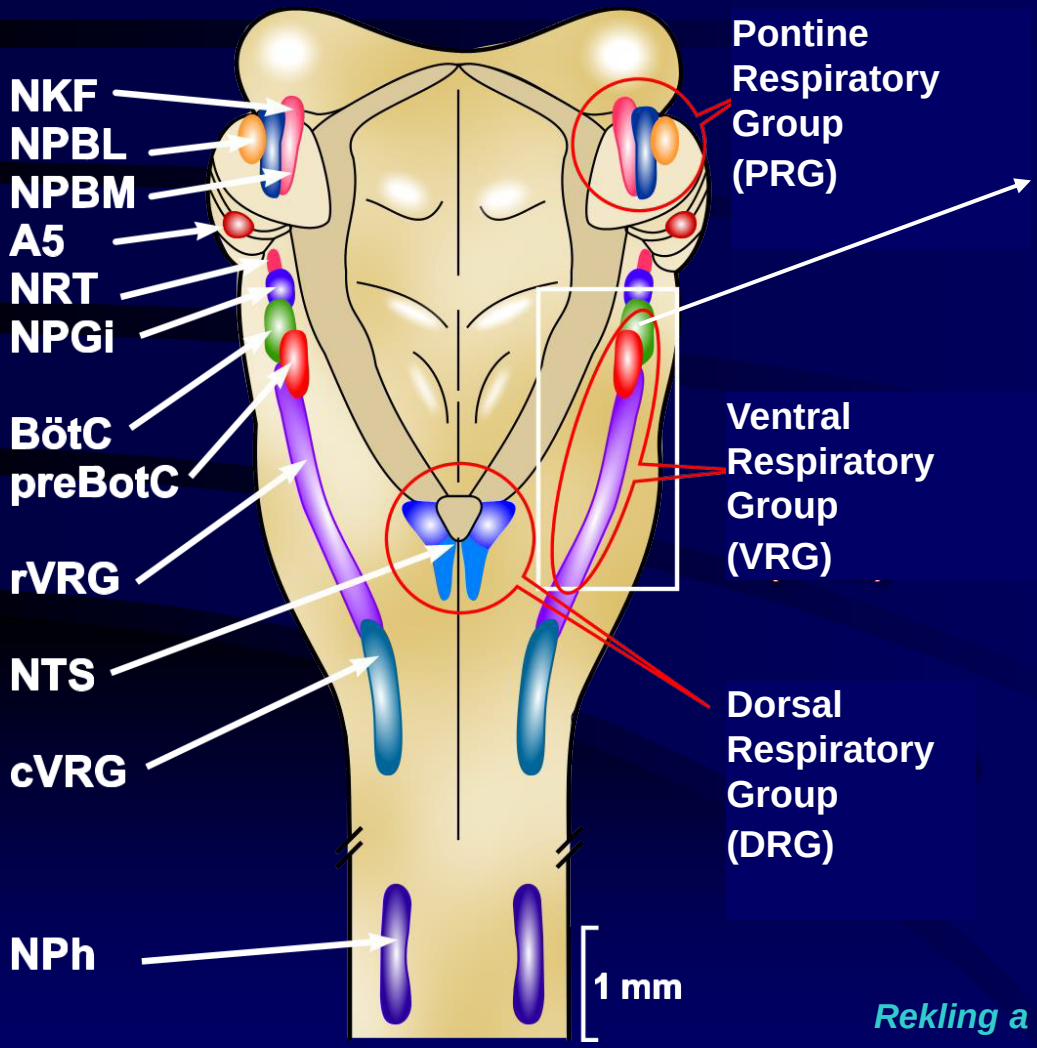


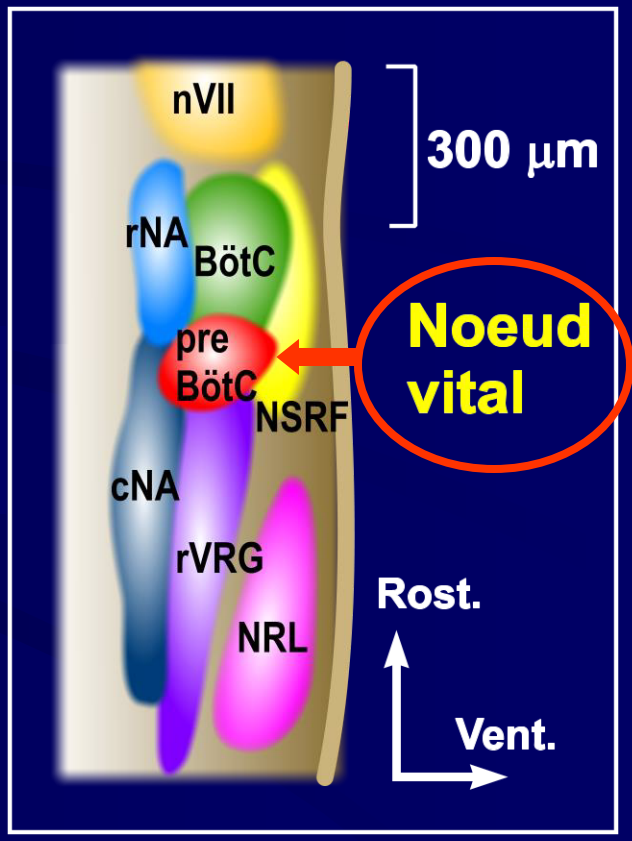


2

Respiratory rhythm
Disorders of respiratory rhythm - arrhythmias

RESPIRATORY STRUCTURES





Richter et al. (1991, 1993)

Rekling a Feldman: Annu. Rev. Physiol., 60,1998

Smith et al. 1991, 2000

RESPIRATORY RHYTHMOGENESIS

Network regulation

- No primary pacemaker
- Rhythm based on reciprocal synaptic interactions and inherent properties

Adults

Richter et al. ((1998)
Rybak et al. (1997)
Lindsey et al. (2000)

Pacemaker

- Principal role in pacemaker cells
- Distribution and modulation by network

Fetus
Newborns

Smith et al. (1993)
Butera et al. (1999)

Hybrid regulation

- Rhythm is paced by special assets
- Network involved in shaping of the pattern

Newborns
Adults

Smith (1997)
Matsugu et al. (1998)
Smith et. al. (2000)

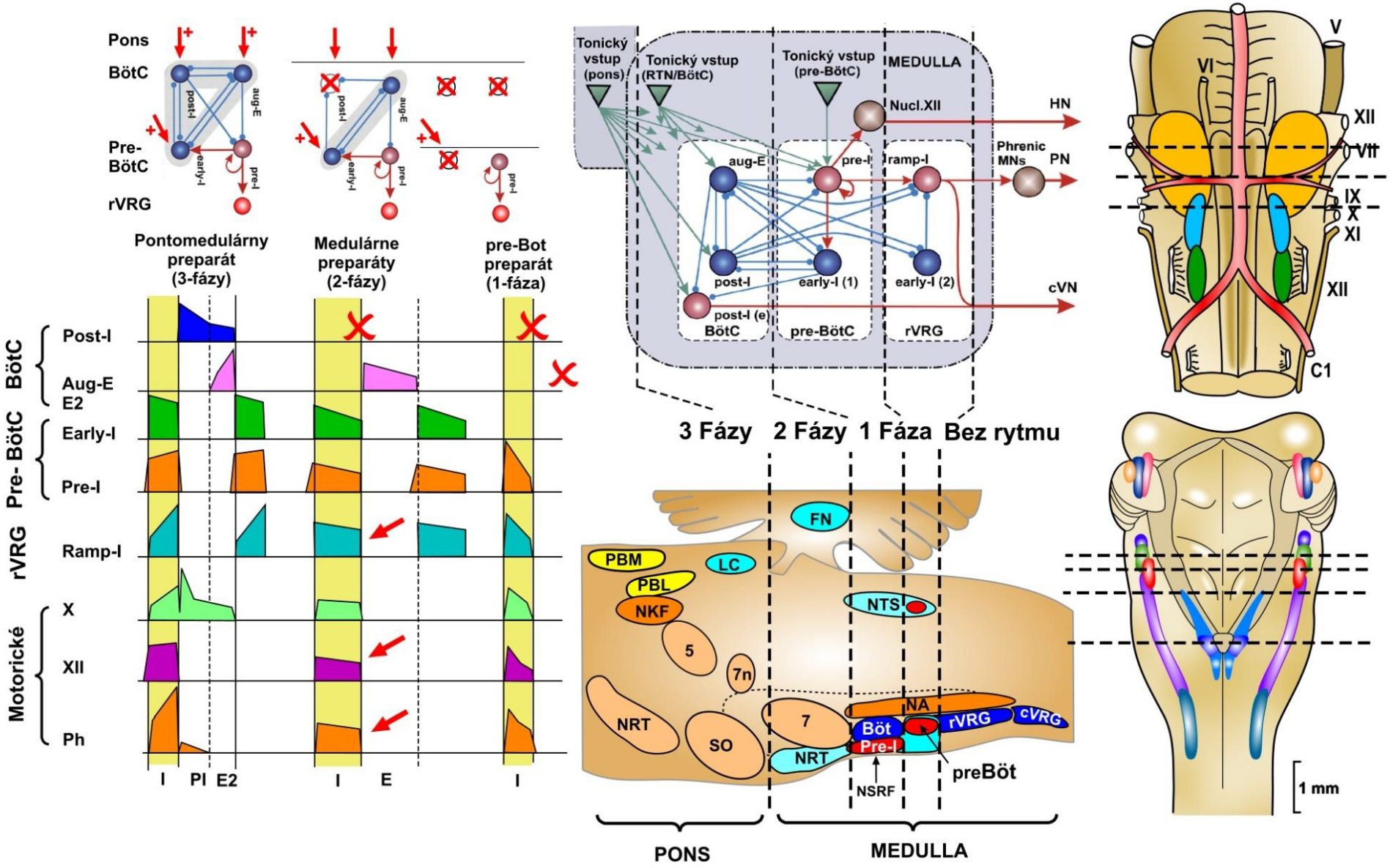
Alternative regulation

- Basic respiratory pattern
- Adaptively modulated by multiple inputs

Newborns
Adults

Rybak et al. (2001)
Patton et al. (2002)

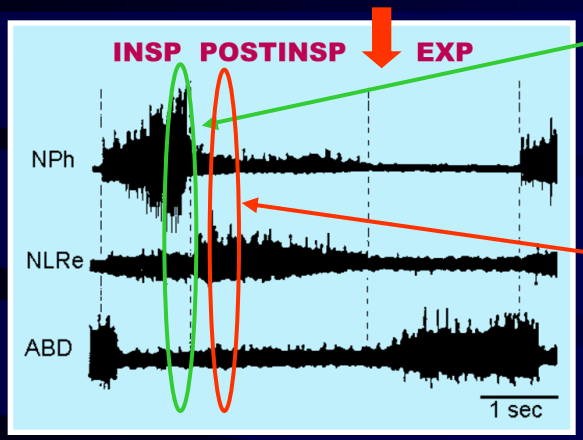
Ponto-medullary network



Beňačka (2010; spracované podľa Smith et al., J. Neurophysiol, 98, 3370-3387, 2007)

RESPIRATORY RHYTHMOGENESIS

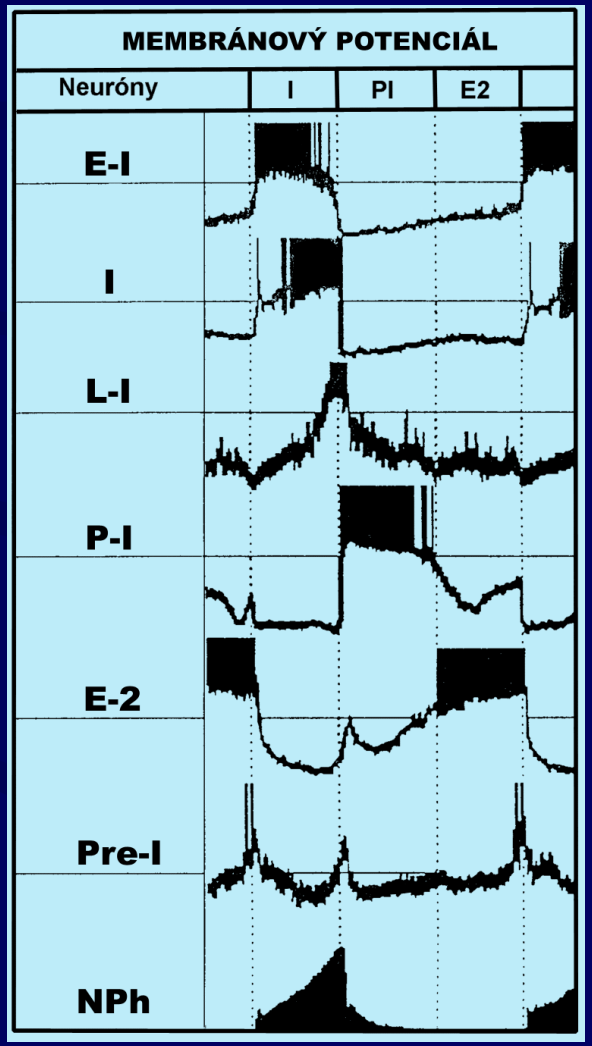
- **3 neural phases of respiration govern 2 ventilatory movements**



Reversible off-switch

Ireversible off-switch

- **6 different types of neurons**
  - **E-I** early inspiratory neurons
  - **I (I-ramp, I-all)** inspiratory neurons
  - **L- I** late inspiratory neurons
  - **P- I** post-inspiratory neurons
  - **E 2** expiratory neurons
  - **Pre- I** pre- inspiratory neurons



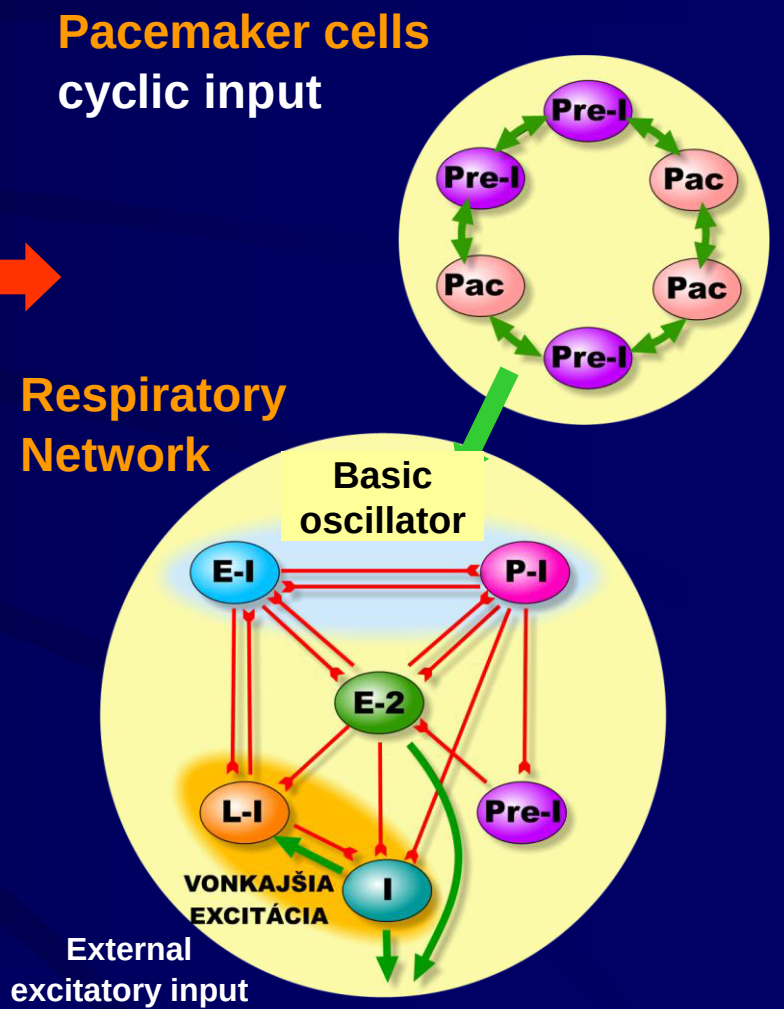
Richter et al. (1991)

Richter et al. (1993)

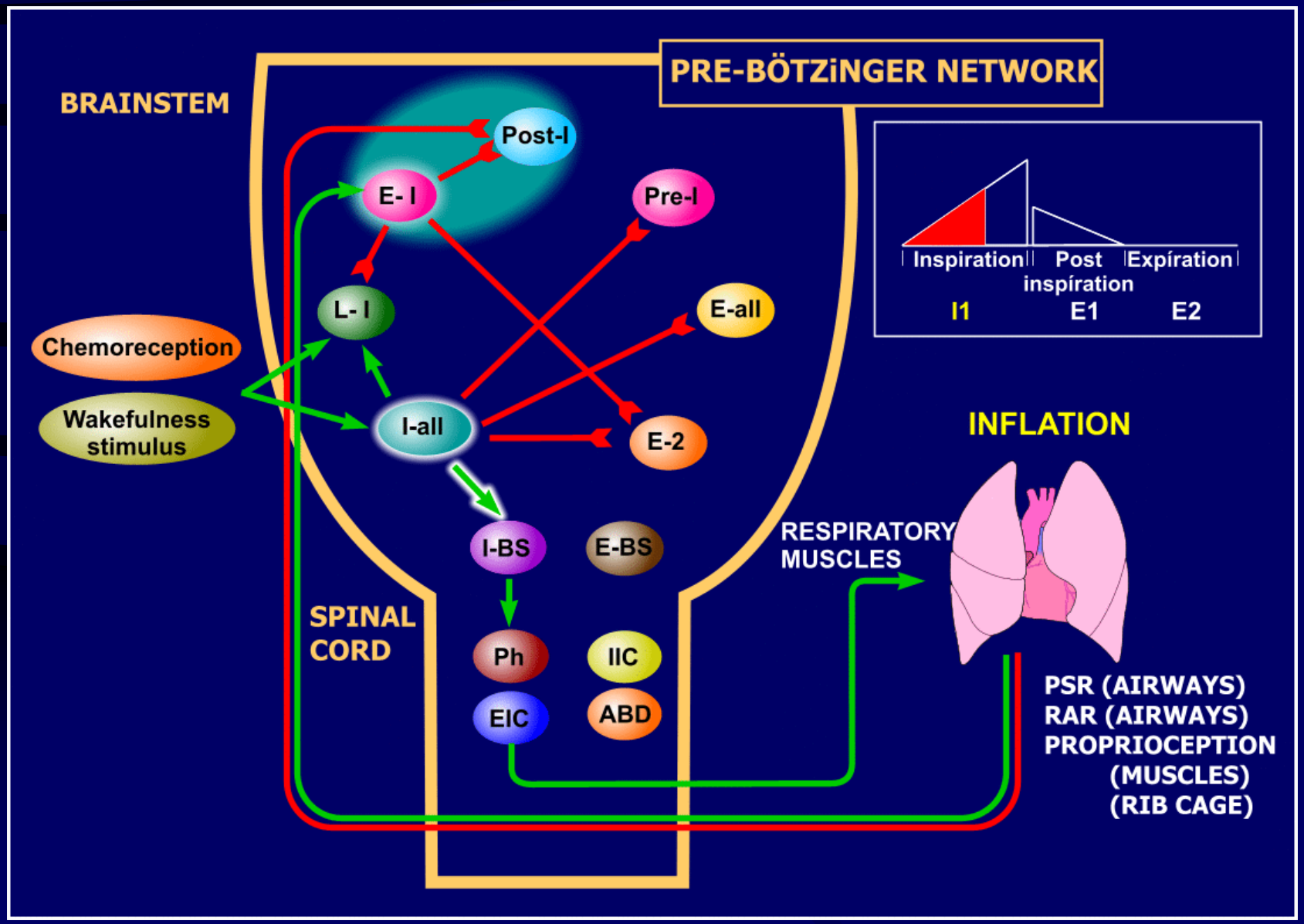
RESPIRATORY RHYTHMOGENESIS

- E-I early inspiratory neurons
- I inspiratory neurons
- L- I late inspiratory neurons
- P- I post-inspiratory neurons
- E 2 expiratory neurons
- Pre- I pre- inspiratory neurons

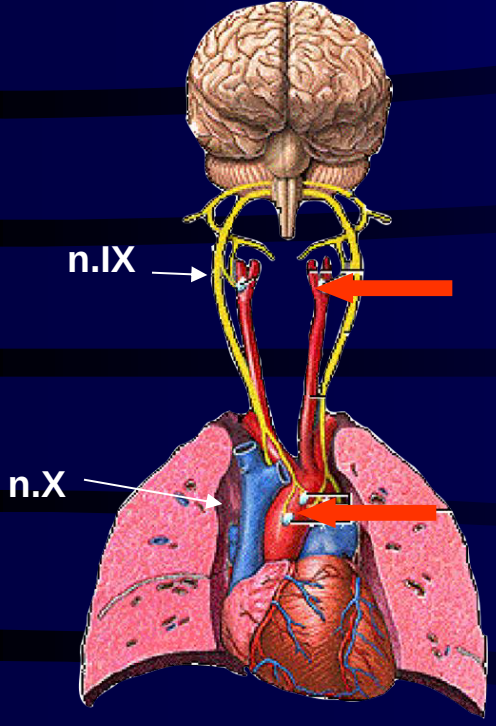
Practically all main transmitter systems take part in synaptic modulation, e.g.
Glut, Gly, Ser, Cat, etc.



NEURONAL INTERACTIONS WITHIN RESPIRATORY NETWORK



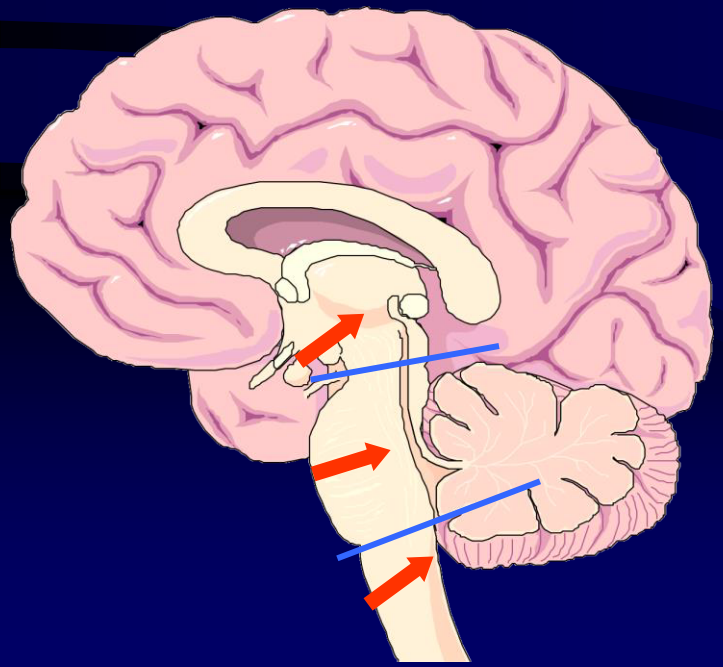
O_2 – CHEMORECEPTION



- carotide receptors (pO_2, pCO_2, pH) -> n.IX -> medulla

blood flow 2 l/min/100 g; stimulated when P_aO_2 <60 mmHg

- aortal receptors (pO_2, pCO_2) -> n.X -> medulla
- arterial O_2 sensors (pO_2, pH) -> veget. Afferents, local
- pulmonary sensors (pO_2, pH) -> n.X, local reflexes

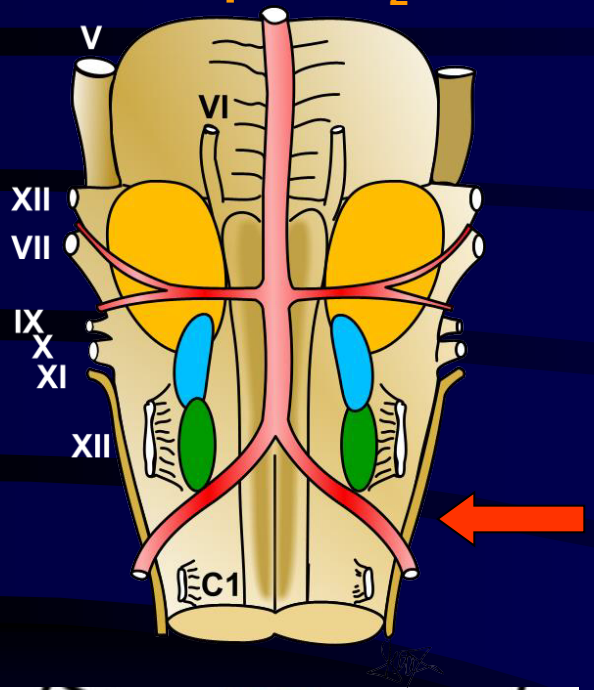


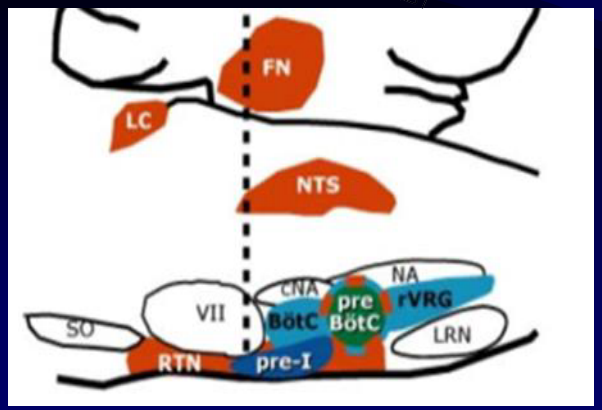
Several areas may displey local O2- sensitivity in respiratory stimulation :

- lateral medullary reticular formation (RF) Arita et al. (1988)
- RF in caudal and rostral pons Edelman et. al (1991)
- raphe nuclei Millhorn et al. (1980,1984)
- diencephaslic locations Tenney a Ou (1976)
- caudal hypothalamus Horn a Waldrop (1997)
- PreBötzinger complex Solomon et al. (2000)

CENTRAL pH/CO_2 – DEPENDENT CHEMOREGULATION

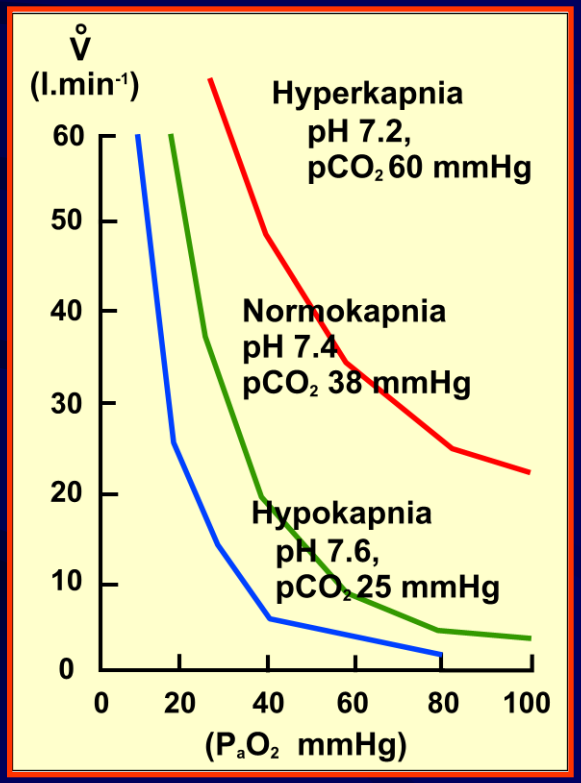
Central pH/CO_2 – sensitive structures



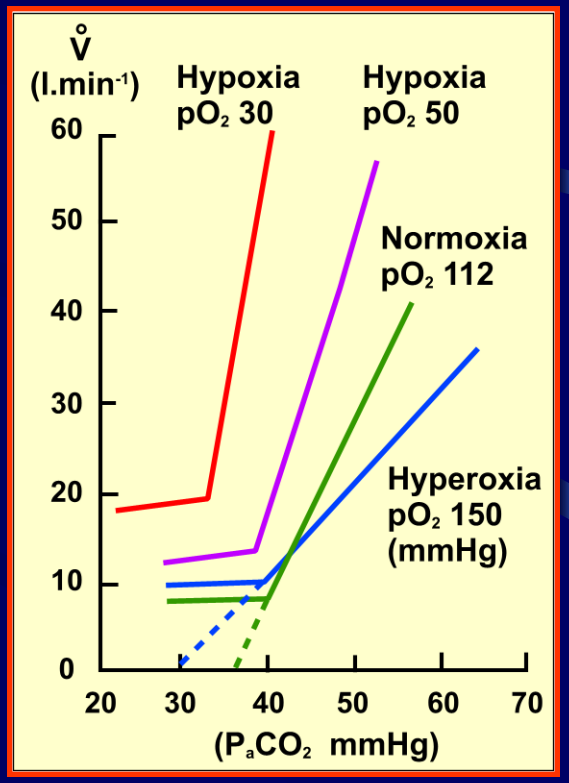


Stimulation of ventilation

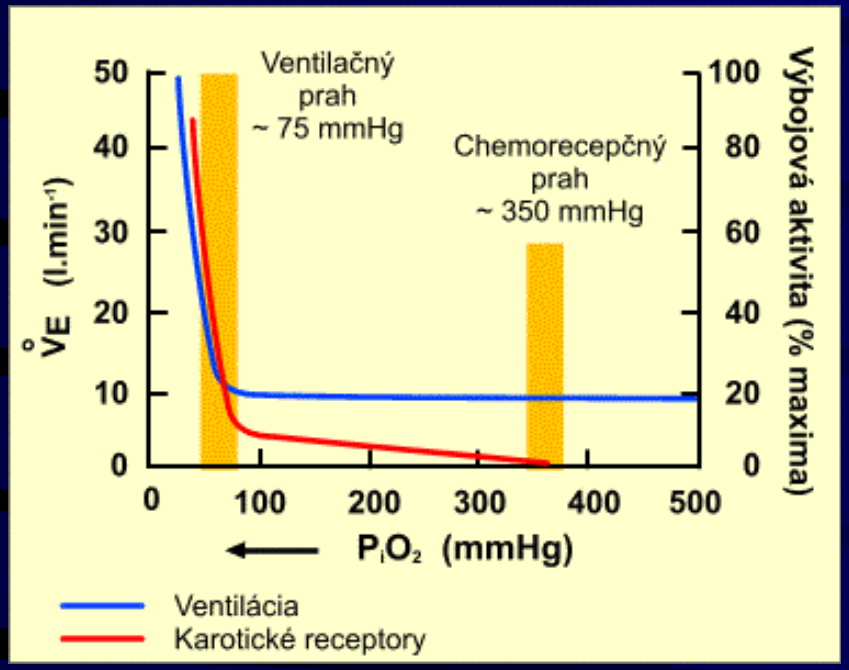
CO_2 ➡ O_2



O_2 ➡ CO_2

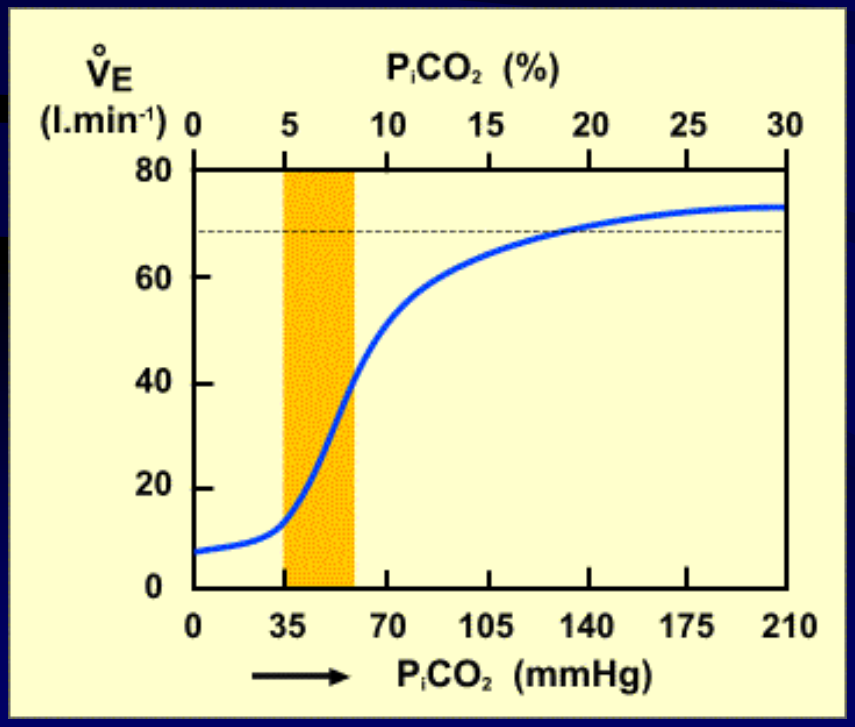


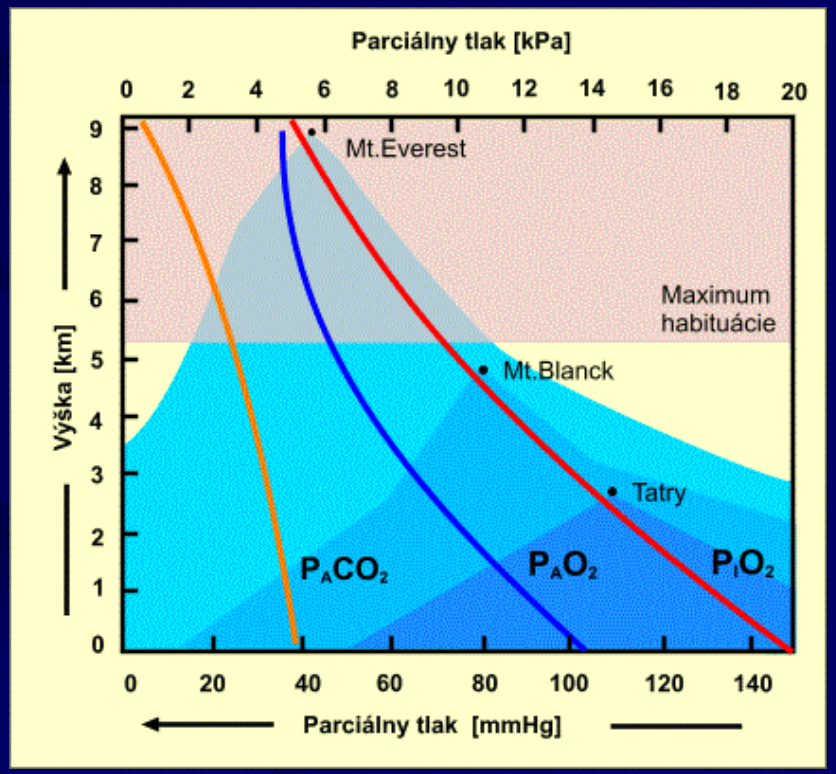
Millhorn a Eldridge (1986), Schlaefke (1987), Nattie (1998)



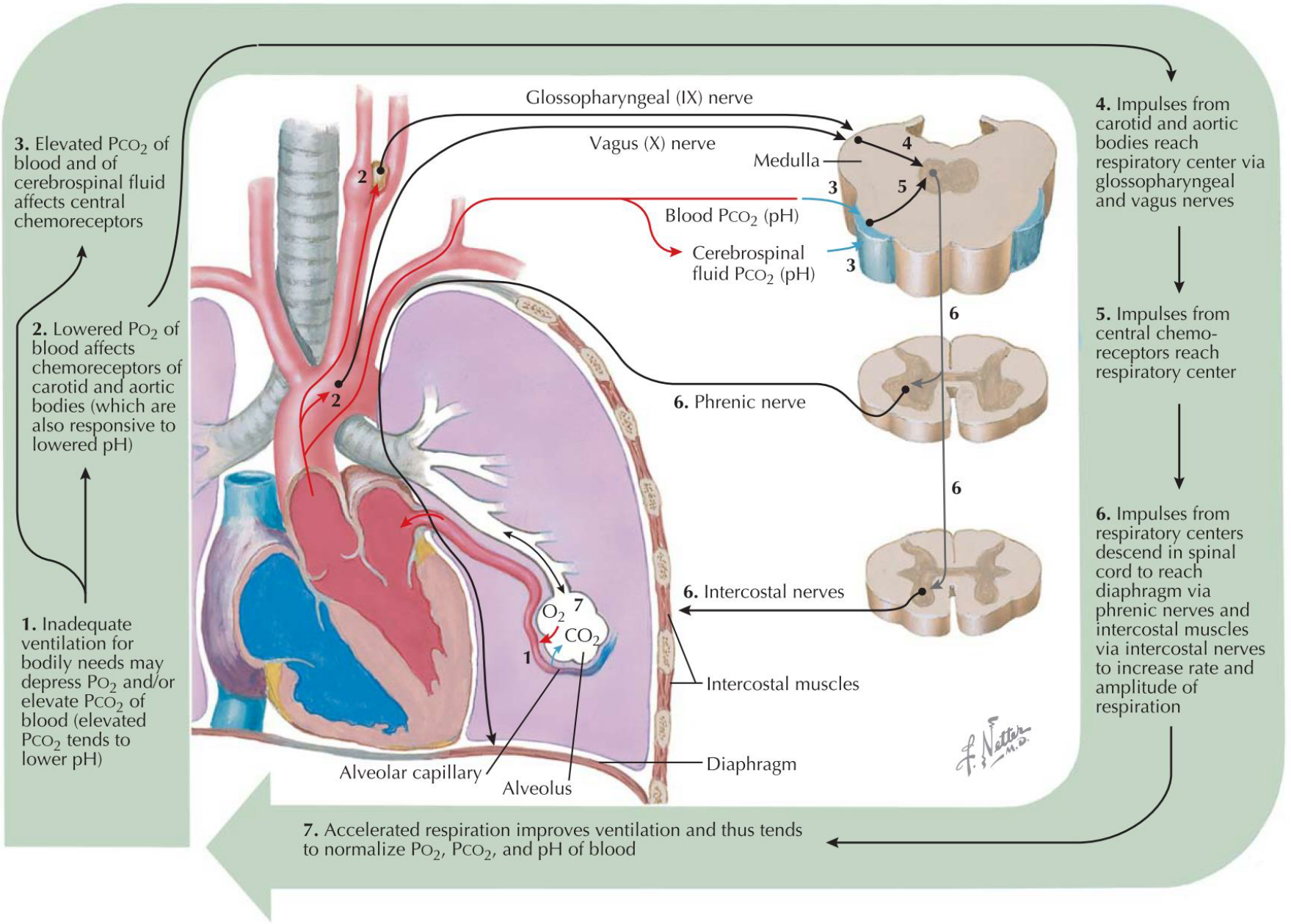
- Ventilatory threshold of O_2 response – steep slope of rise of ventilation with drop of PaO_2 ~ 70 mmHg
- O_2 – chemoreceptive threshold: chemoreceptove activation w/o ventil. response (PaO_2 ~ 95 mm Hg)
- CO_2 exert stimulatory effect on respiration up to 60% of maximal voluntary hyperventilation
- CO_2 sensitivity = steep slope of rise within the range 5 - 7% $PiCO_2$ (6 $l.min^{-1}$ na 1% CO_2);

Dead zone – 8,5 km above see level

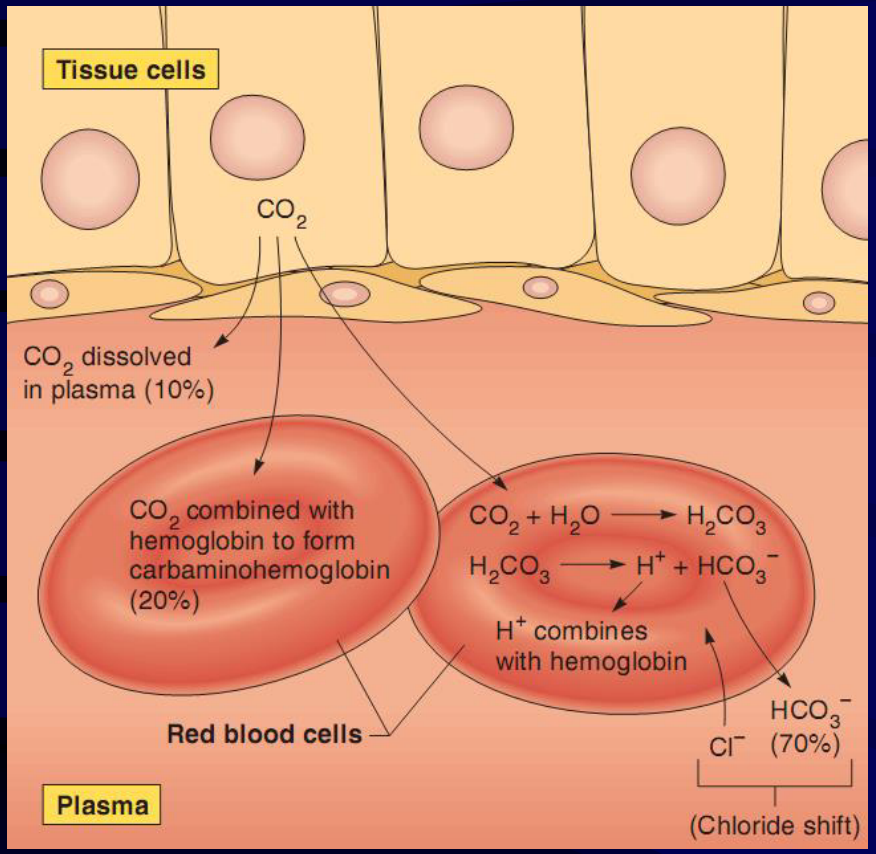


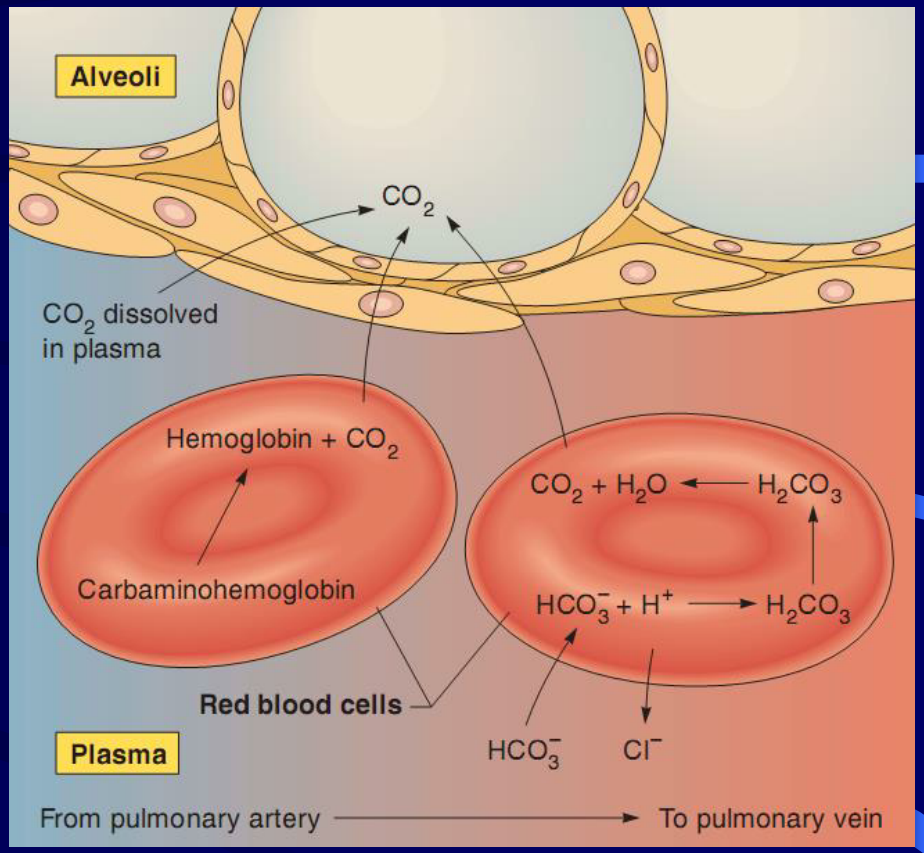


Chemical Control of Respiration (Feedback Mechanism)



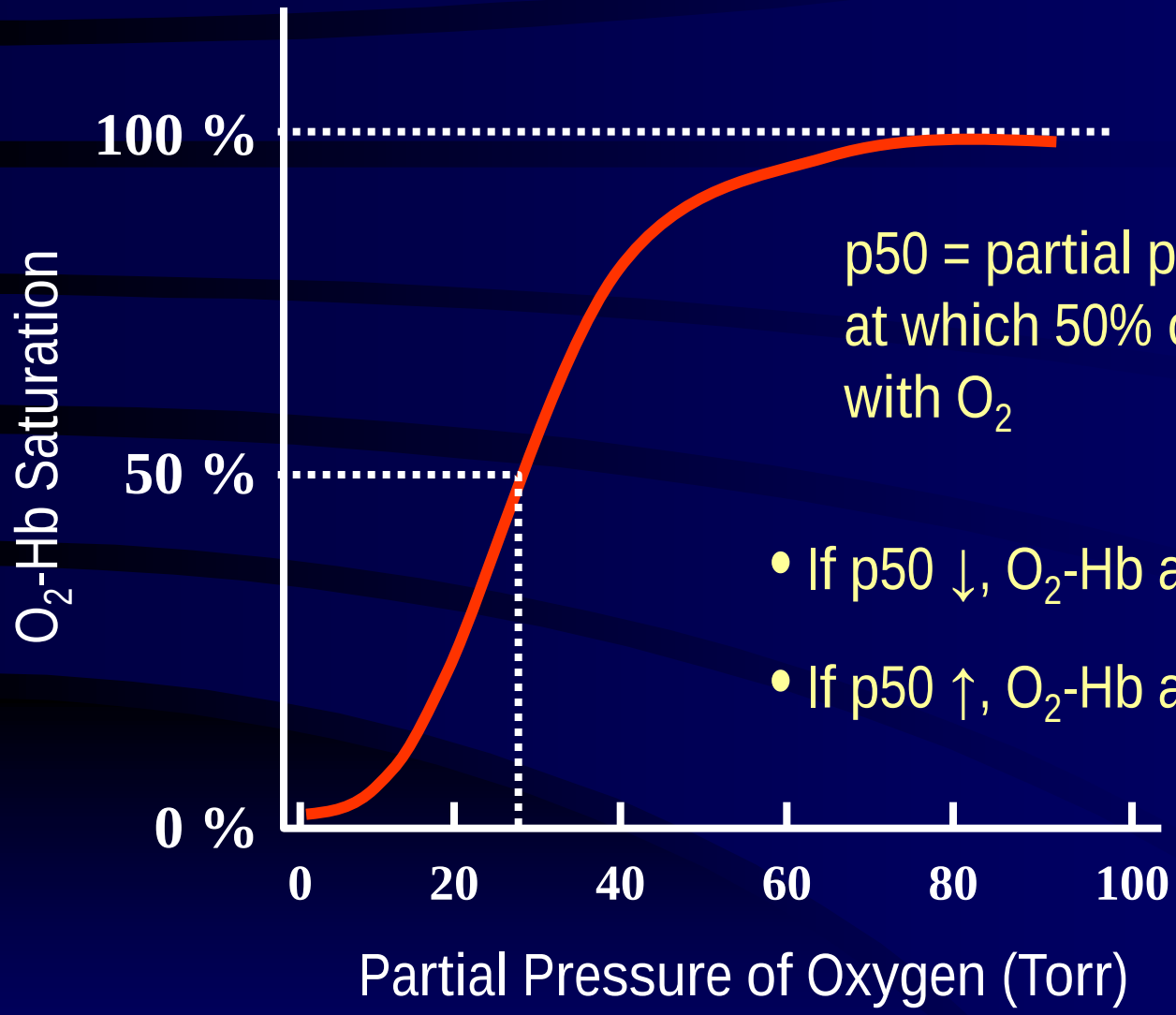
Role of lungs in gas exchange





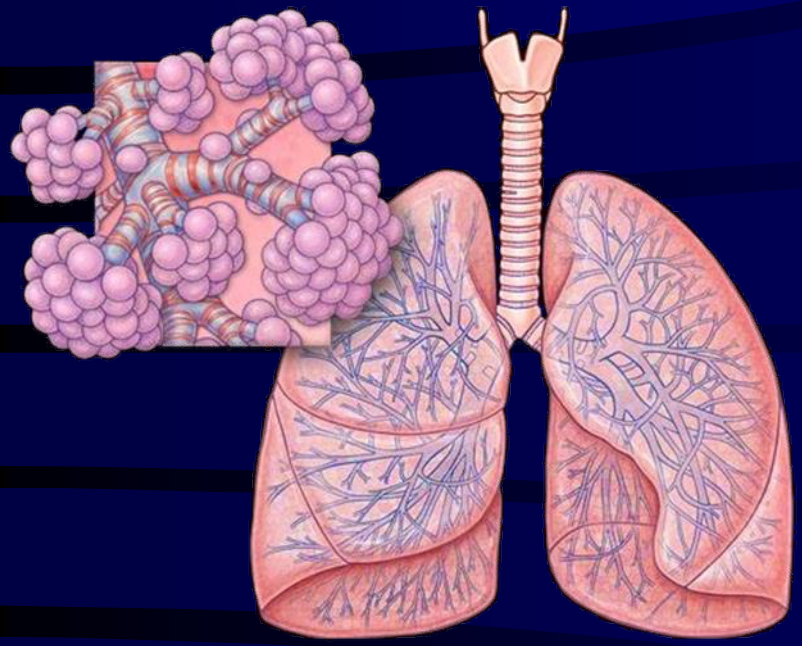
- Deoxygenated blood (Hb) carries more CO2 than oxygenated blood (Hb)
- CO_2 can bind to Hb (carbaminohaemoglobin).
- Oxygenation of Hb leads to less HCO3- in the blood.
- The binding of O_2 to Hb decreases the affinity of Hb for CO_2.

Oxygen-Haemoglobin Binding Affinity



p50 = partial pressure of O_2 at which 50% of Hb is bound with O_2

- If p50 ↓, O_2-Hb affinity increases
- If p50 ↑, O_2-Hb affinity decreases



3

Alteration of respiratory rhythmicity and breathing pattern

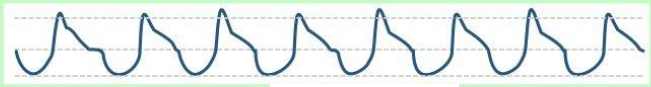
Terminology

- Eupnea normal breathing movements,presuming normal amplitude of breaths (adults 12-20 c/min; kids 15-30 c/ min; newborns 25-50 c/min)
- Hyperpnea (hyperventilation) increased breathing movement
- Hypopnea (hypoventilation) decreased breathing movements
- Apnea arrested breathing
- Bradypnea decreased rate of breathing (< 20 breaths /min)
- Tachypnea increased rate of breathing (> 20 breaths /min)
- Dyspnea labored breathing (subjective feeling)
- Asphyxia inability to breathe, suffocation
- Orthopnea labored breathing in the sitting or upright position

Apnoe reflex-based = trigerred by reflexes, e.g trigeminal, trigeminovagal, laryngeal, etc
Sleep apnoea = cessation of of breathing or efficiency of breathing during sleep
Central apnoea = depression of breathing (barbiturates, morphine)
Peripheral apnoea = neuromuscular paralysis, UAW obstruction,
Central congenit. hypoventilation = failure of breathing during the night
Kussmaul breathing = labored breathing duting metabolic acidosis
Obstructive breathing (air-trapping) = e.g. in COPD, gradially increasisng FRC
Central neurogeneic hyperventilation = hypoventilatory breathing

Adaptívne a kompenzačné zmeny dýchania

EUPNOE (NORMOPNOE) 12- 20 d/min

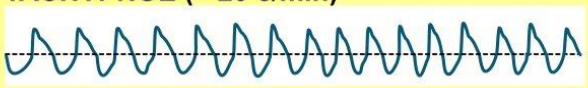


Normal pattern of breathing

dospelý 12-20 c/min,
dieťa 15-30 c/min
novorodenec 25-50 c/min

1. NOMOMORFNE ZMENY

TACHYPNOE (> 20 c/min)



- Lieky spôsobujúce respir. útlm, intoxikácie
- Panika, bolesť, metab. alkalóza

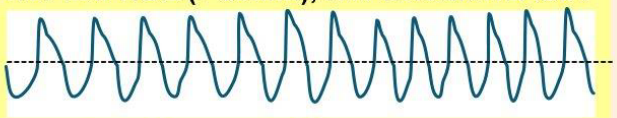
BRADYPNOE (< 10-12 c/min)

- Lieky spôsobujúce respir. útlm (morfín)

HYPOPNOE (< 6l/min), HYPOVENTIIÁCIA

- Lieky, sedatíva, barbituráty, opiáty
- Ochorenia CNS

HYPERPNOE (> 8l/min), HYPERVENTILÁCIA



- pulmonálne patologické zmeny;
- difúzne supratentoriálne metabol. vplyvy

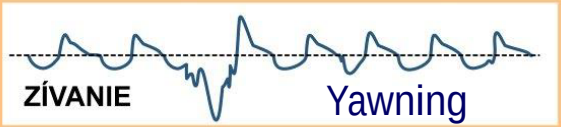
2. HETERO[illegible]MENY

Intrusions

PREHĹBENÉ DYCHY (NÁDYCHY)
(VZDYCHY, OSCILÁCIE, ZÍVANIE)

VZDYCHY — Sighs

Oscillations — OSCILAČNE



ZÍVANIE — Yawning

- hypoxia, antiatelektatické, dysbal, vent/perf.

HYPOVENTIILAČNÉ

SHALLOW BREATHING (PLYTKÉ)

- medulárne tegmentum, laterálna medulla

Central hypoventilation — [illegible]CIA (CNHy)

- medulárne tegmentum, laterálna medulla,
- medulárny infarkt (Wallenbergov sy.) a pod

3. [illegible]PNOICKÉ

Hyperventilations

HYPERVENTIILAČNÉ

Central hyperventilation — [illegible]40 c/min)

- mezencephalon, rostrálny most

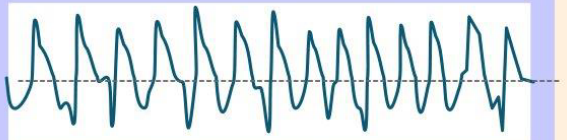
Obstructive (air trapping) — [illegible]NG)

- CHOBPCH, astma a pod.

Acidotic breathing — [illegible]IZAČNÉ)

- laktát, ketolátky, renálna acidóza

KUSSMAULOVE DÝCHANIE

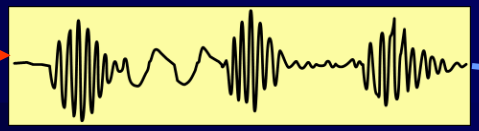


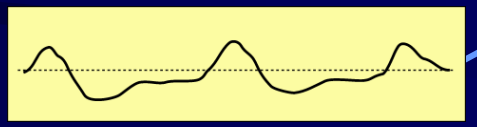
- medulárne tegmentum, laterálna medulla

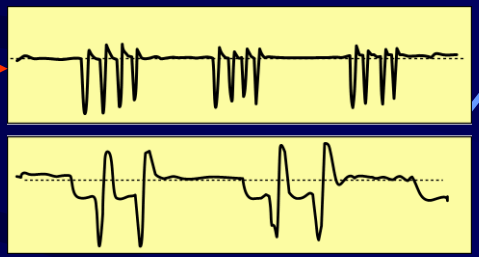
↓ Insp.

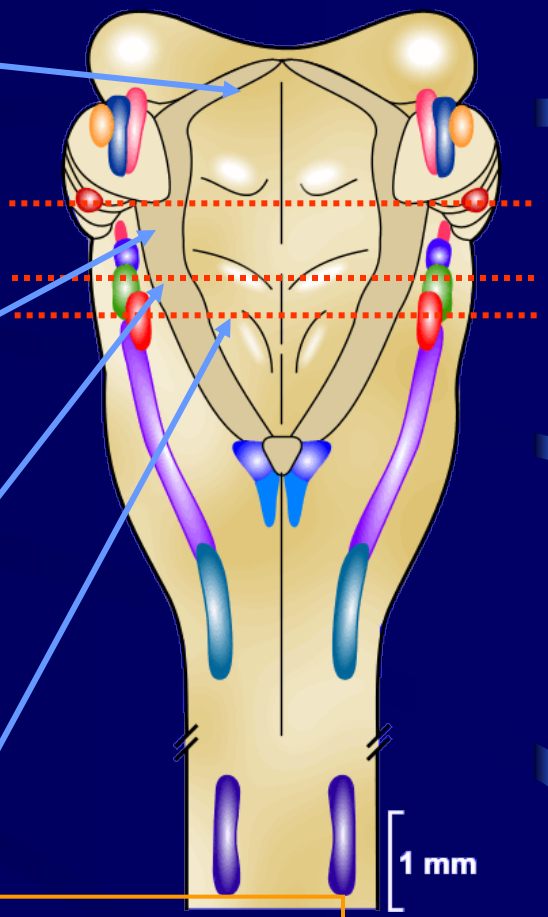
NON-EUPNOIC RHYTHMS

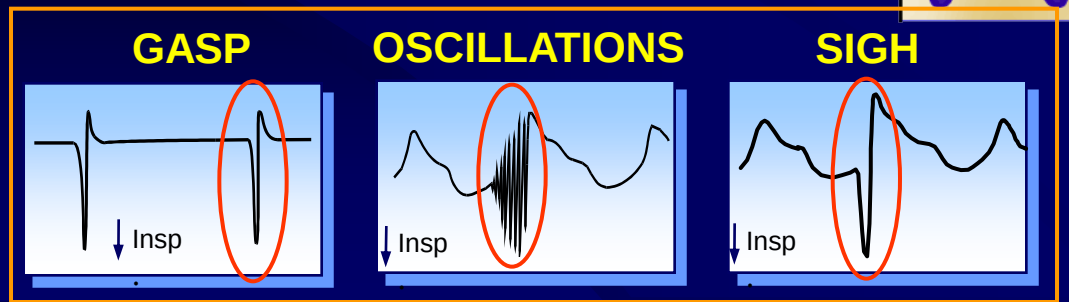
- **Cheyne-Stokes pattern**
  - Diffuse diencephalic or mesencephalic lesions
  - Global brain hypoxia
- **Apneusis**
  - Damage within rostral pons
- **Biot (cluster) breathing**
  - Hypoxia, ischemia of caudal pons
  - High ponto-medullary transsection
- **Gasping**
  - Defects in ponto-medullary border
  - Hypoxic brainstem
  - damage

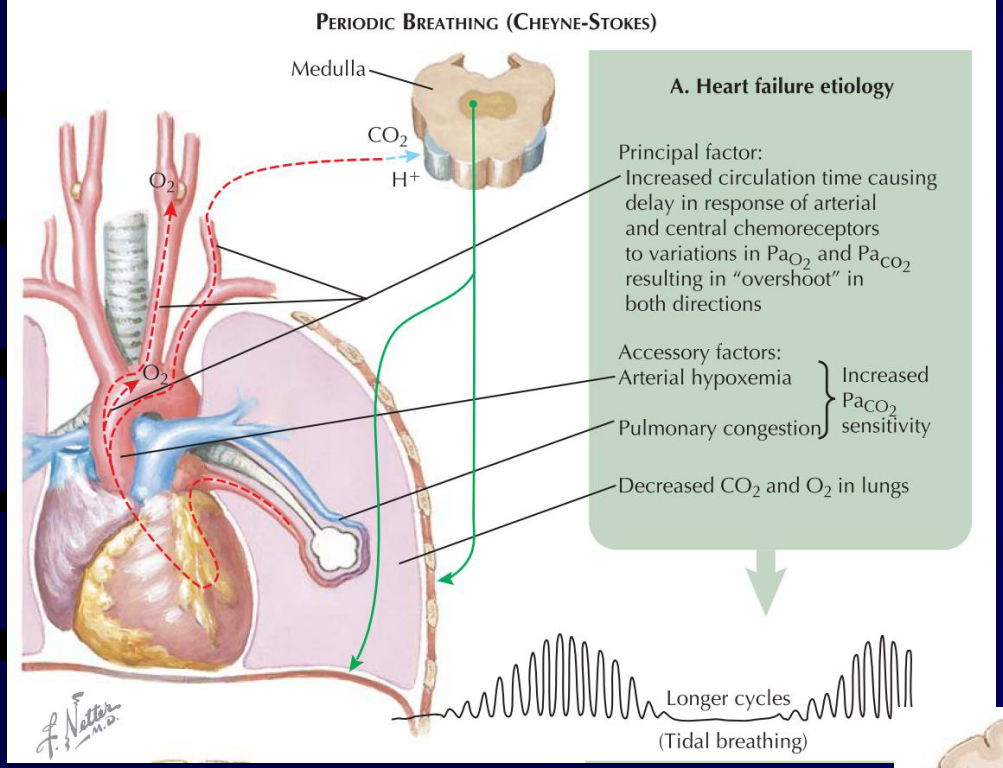






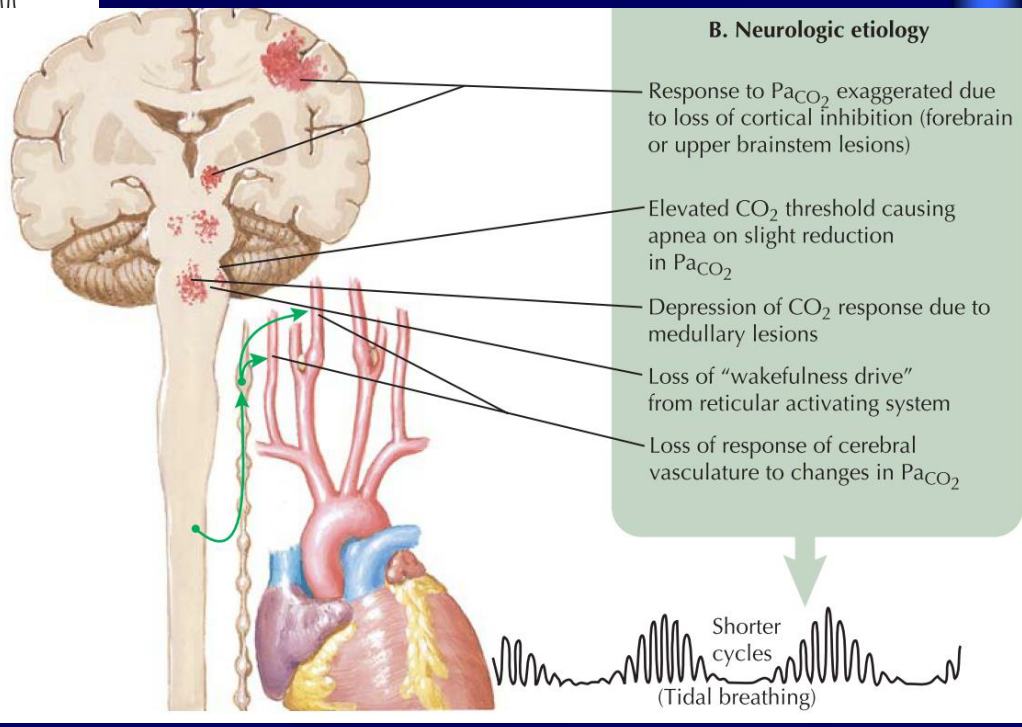




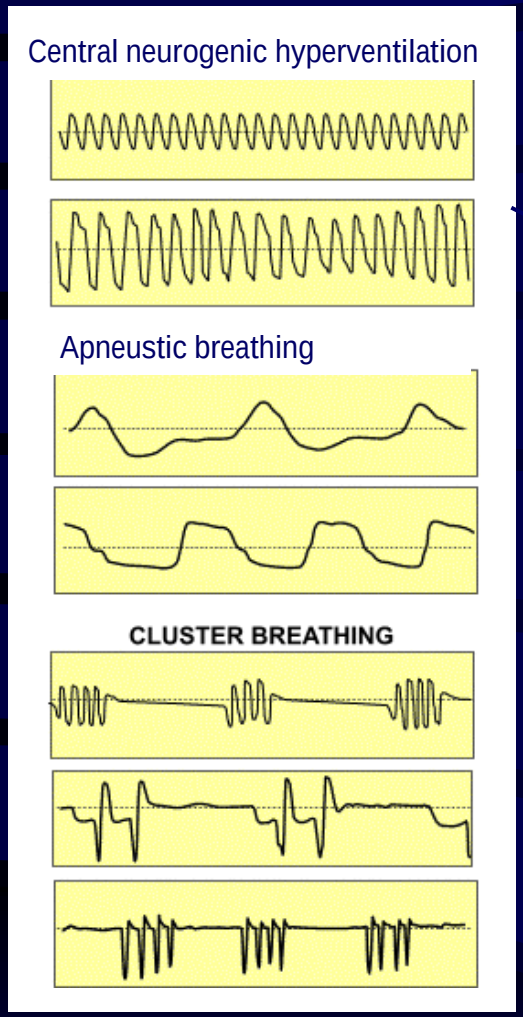


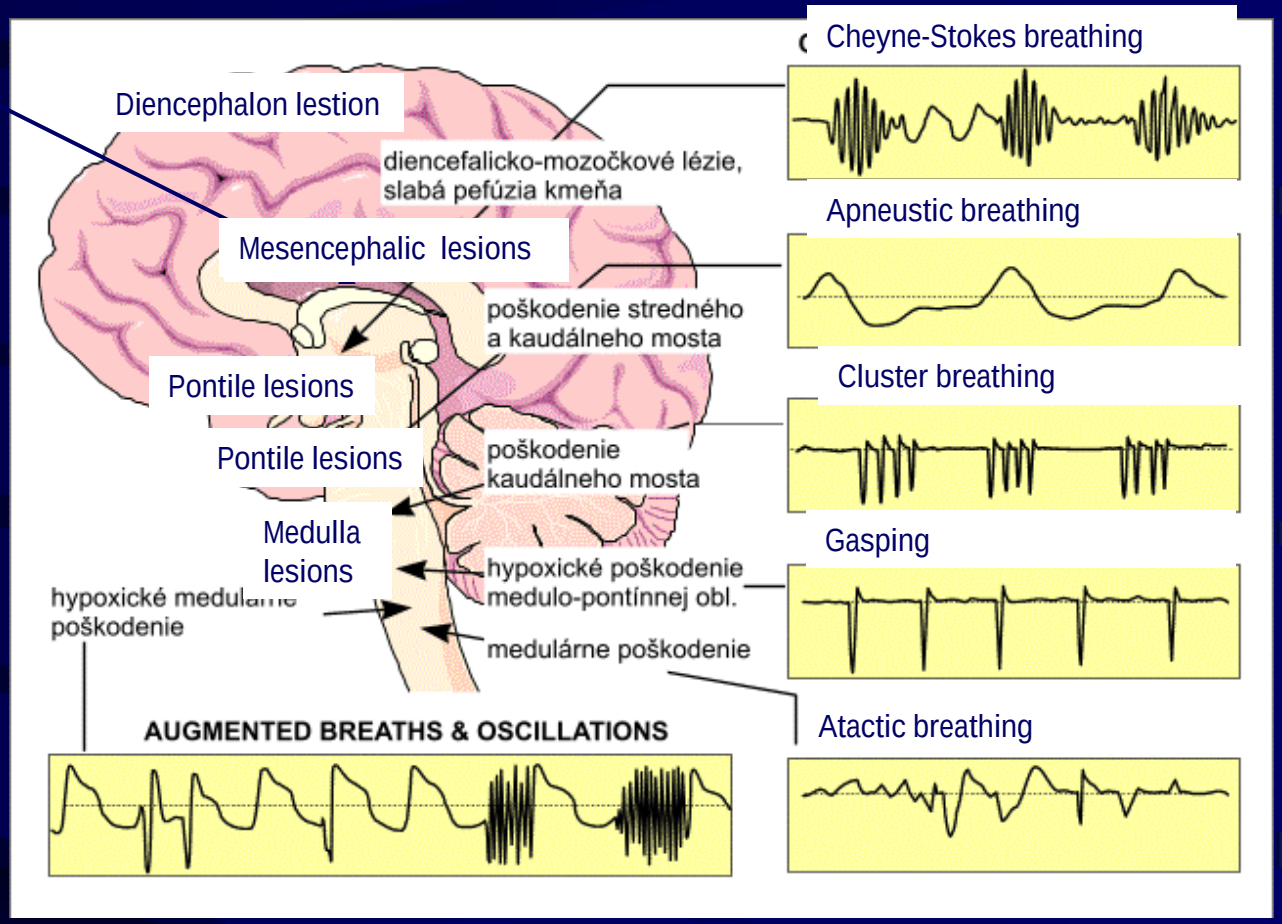
Systemic pathogenesis of Cheyne - Stokes breathing

Neurogenic pathogenesis of Cheyne - Stokes breathing



Rhythm disorders



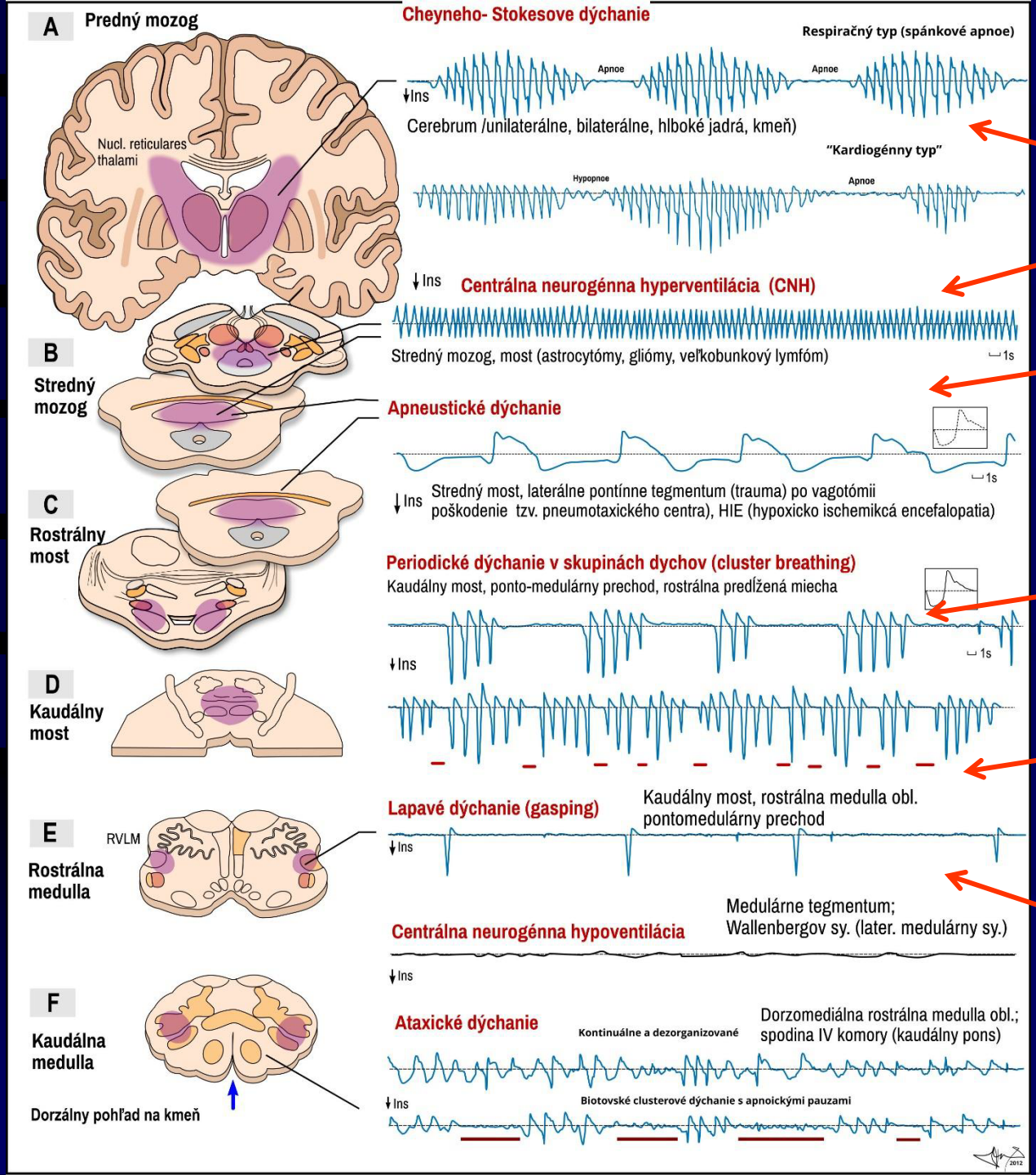


Dysrhthmias modifing eupnoeic breaths

- Augmented breaths (sighs)
- Breaths with end-inspiratory oscillations
- Breaths with postinspiratory tone

Periodic rhythms - non-eupnoic type

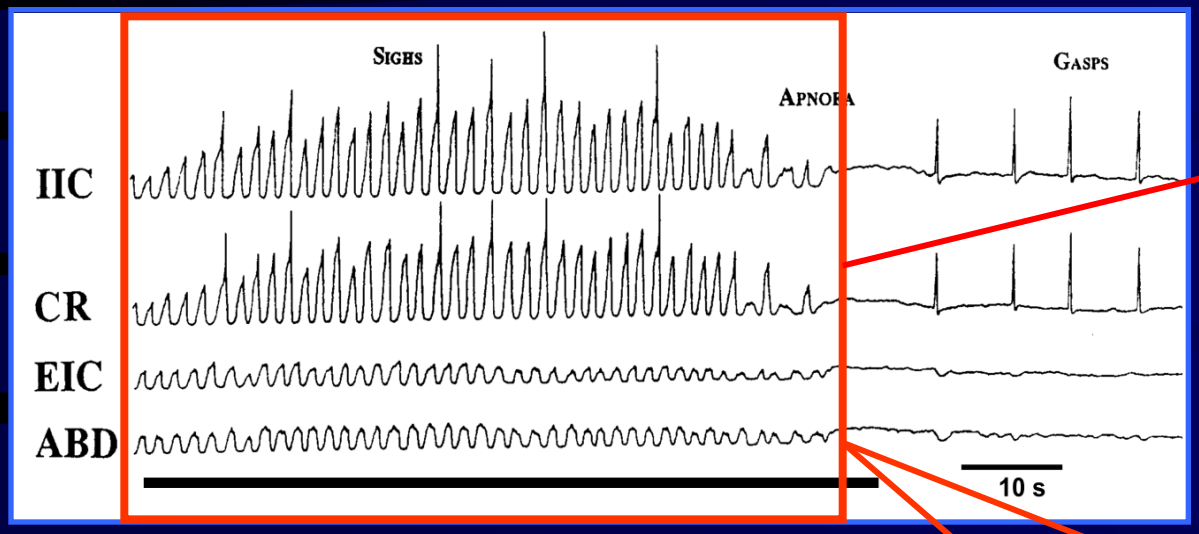
- Cheyne – Stokes breathing
- Gasping
- Cluster breathing
- Apneustic breathing



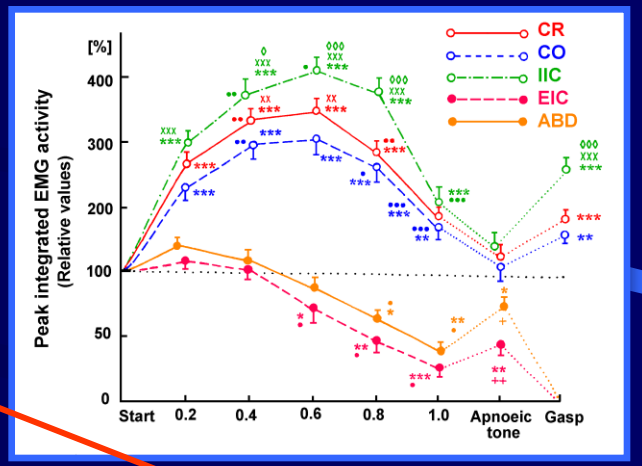
- Cheyne- Stokes breathing
  - Central type
  - Cardiogeneic type
- Central neurogeneic hyperventilation
- Apneustic breathing (middle pons, klateral tegmentum, lesion of pneumotactic centrum)
- Periodic breathing (lower pons and medulla)
  - Cluster breathing
  - Gasping
- Central neurogeneic hypoventilation
  - Medullary tegmentum
  - Lateral medullary syndrome
- Ataxic breathing
  - Dorzomedial rostral medulla

Picture serves for demostration of verbal description

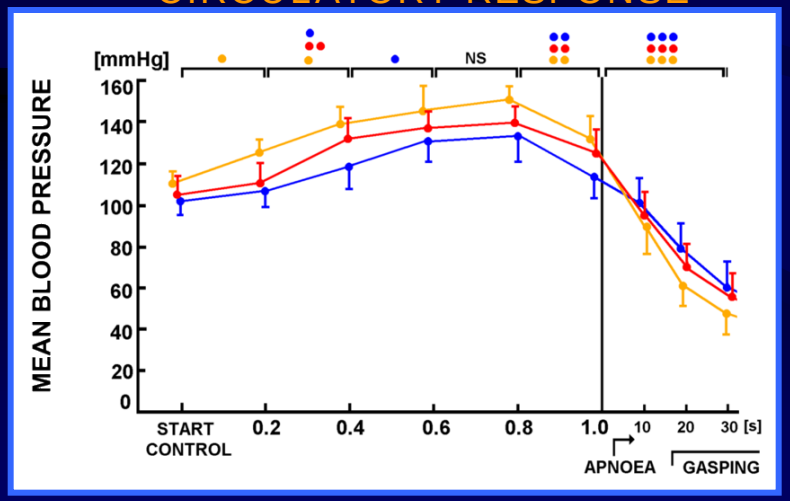
Disturbances in phasic breaths under severe hypoxia



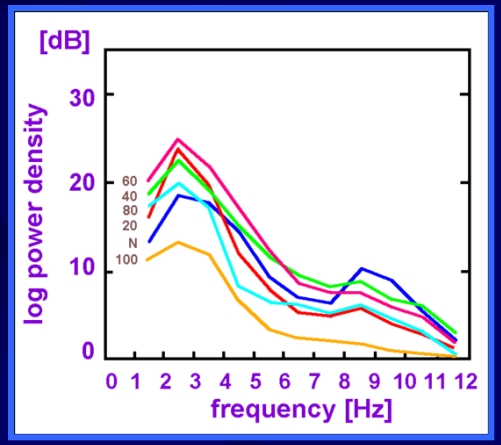
RESPIRATORY MUSCLES



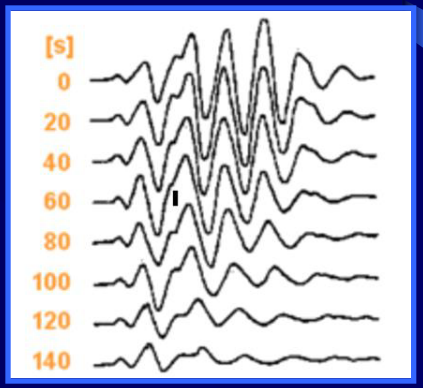
CIRCULATORY RESPONSE



EEG

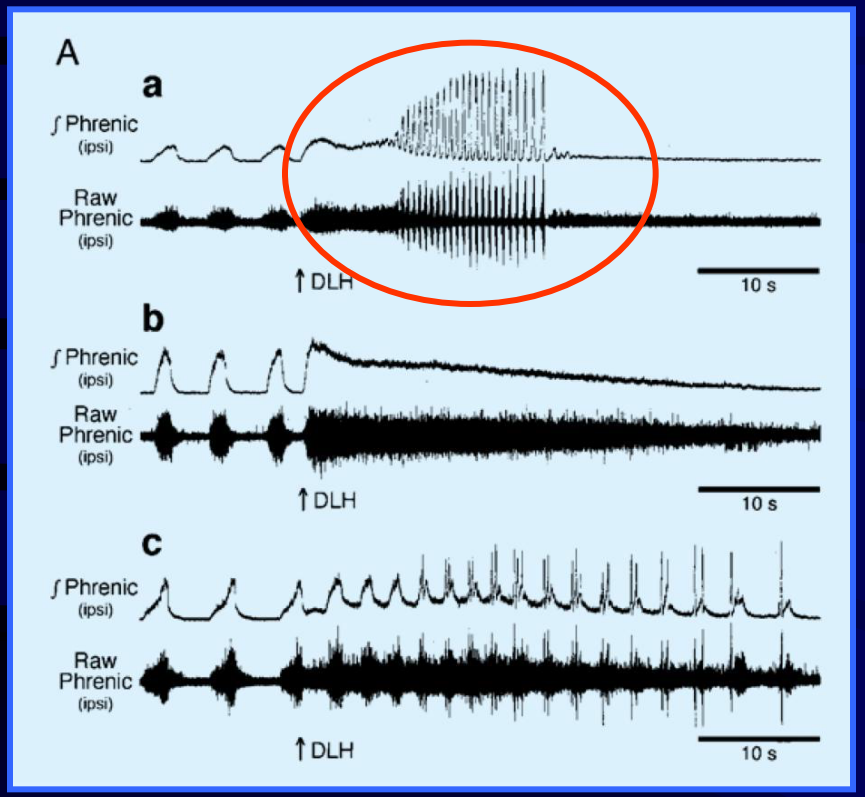


BAEP

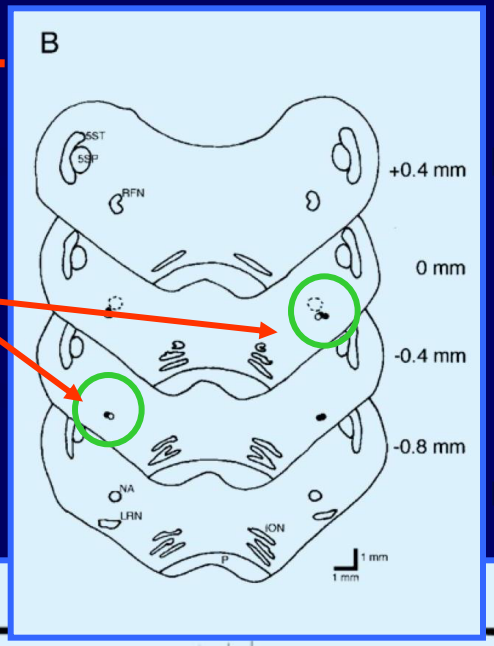


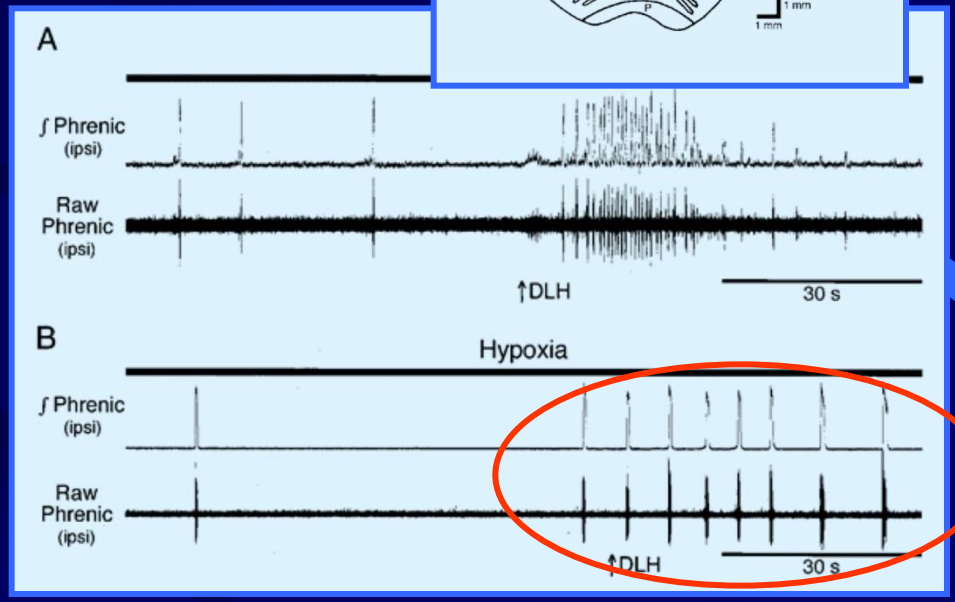
Benacka and Tomori, Ann. Congress ERS, Firenze, 2000

EXCITATION IN RESPIRATORY KERNEL - PreBötC



DL- Homocysteic acid injections 10 mM; 20 nl

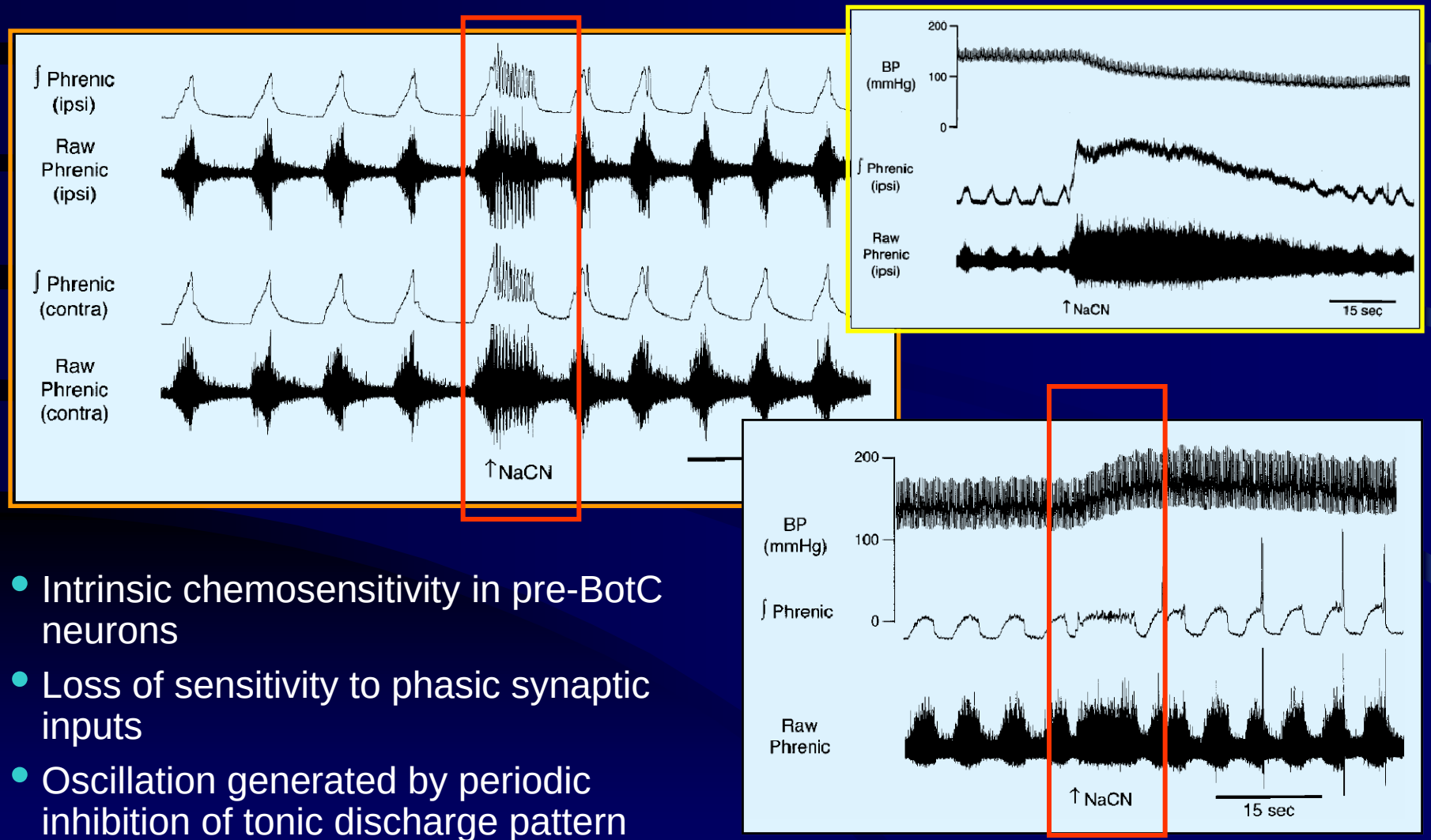




- Generation of tonic discharge
- Short duration oscillatory bursts
- Increased frequency of gasps

Solomon et al.: J. Neurophysiol., 87, 2002

LOCAL HYPOXIA IN RESPIRATORY KERNEL - PreBötC

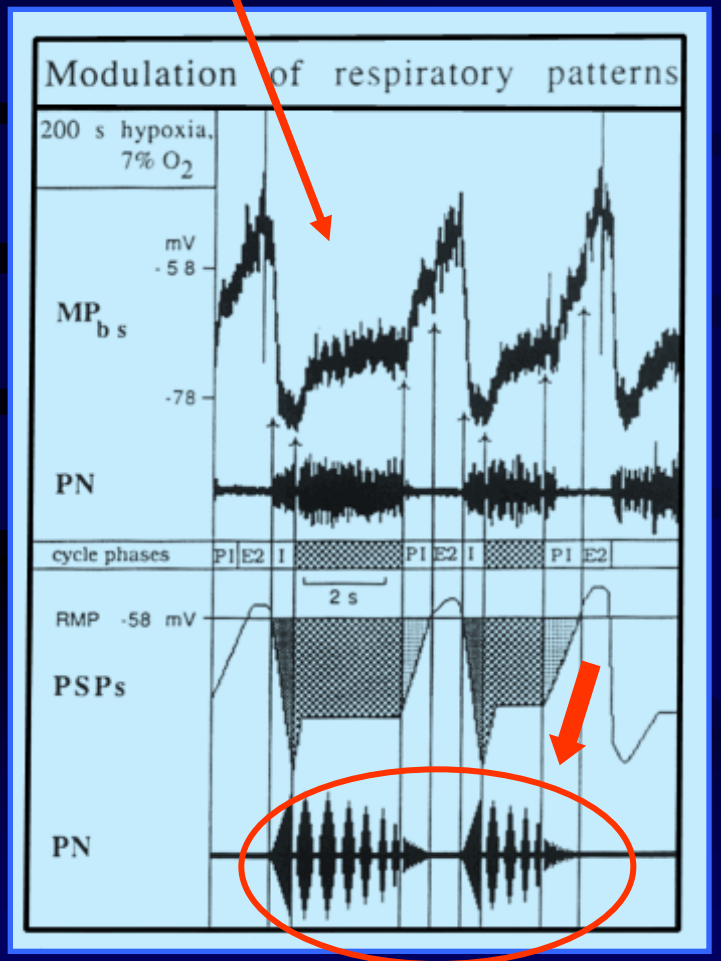


- Intrinsic chemosensitivity in pre-BotC neurons
- Loss of sensitivity to phasic synaptic inputs
- Oscillation generated by periodic inhibition of tonic discharge pattern

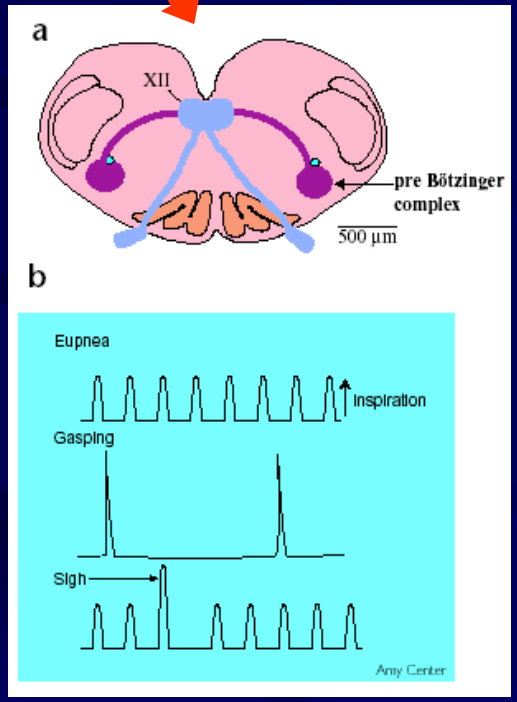
Solomon et al.: J. Neurophysiol., 83, 2000

INSPIRATORY OFF-SWITCH

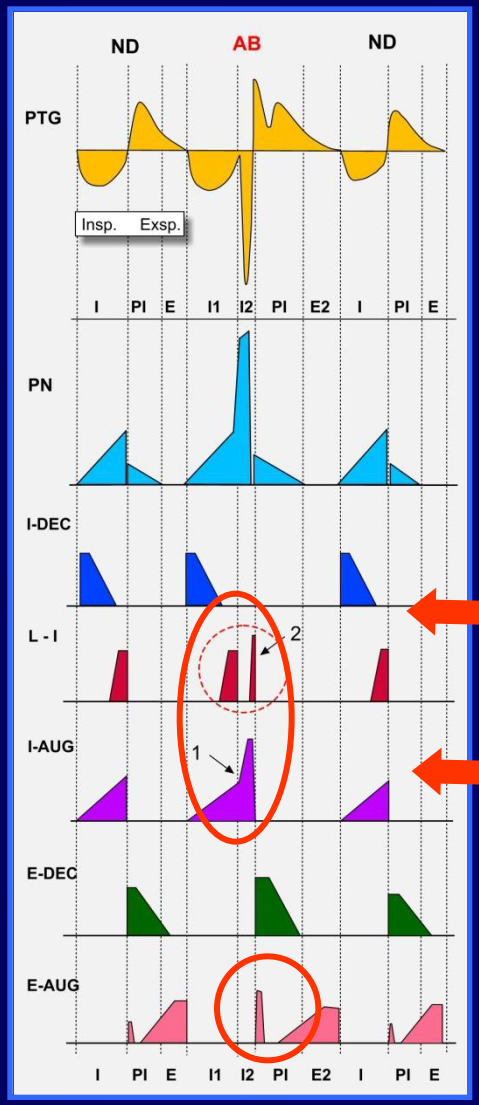
- Ineffective reversible inspiratory off-switch
- Excitation from non-respiratory inputs



Richter et al. 1993

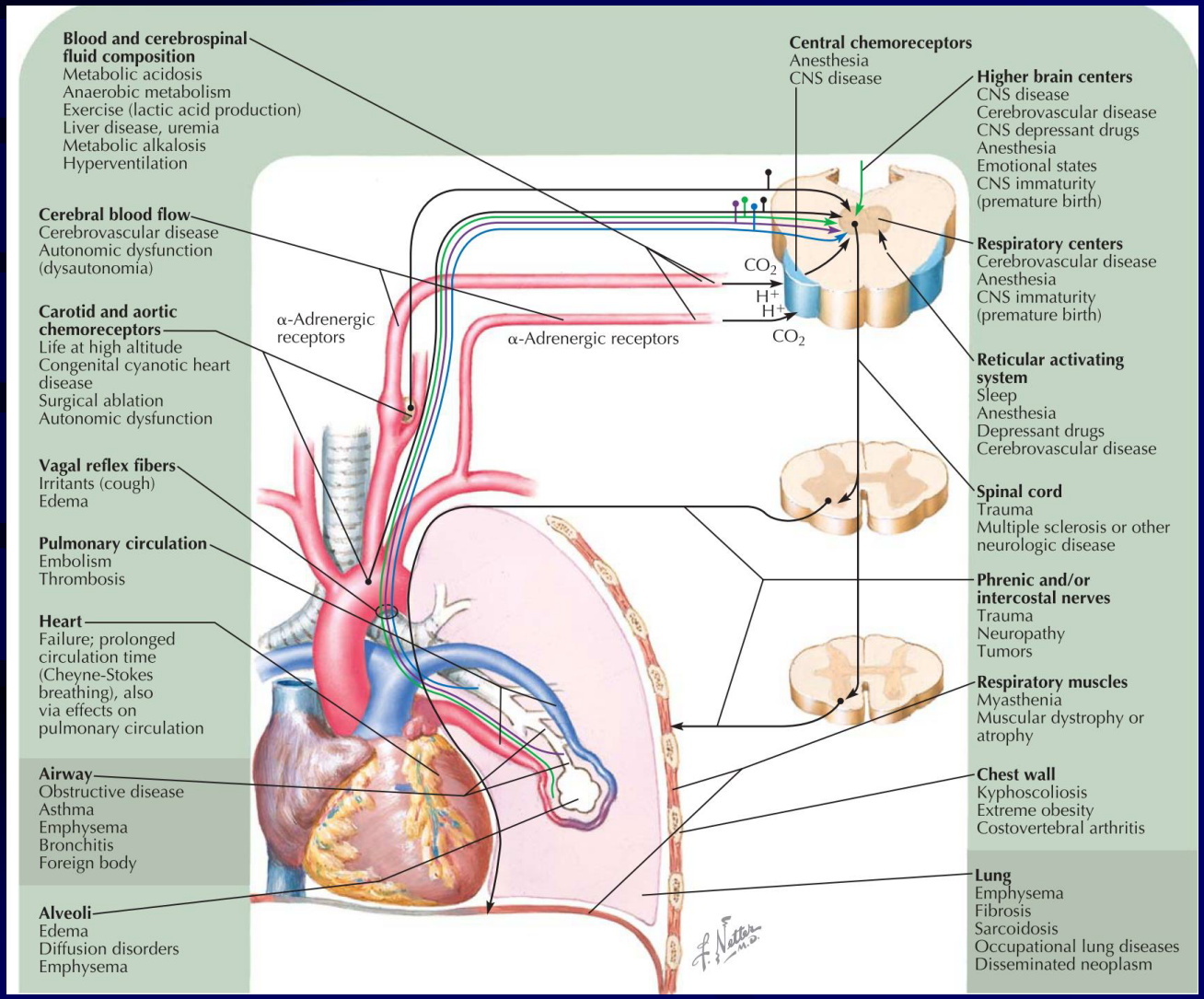


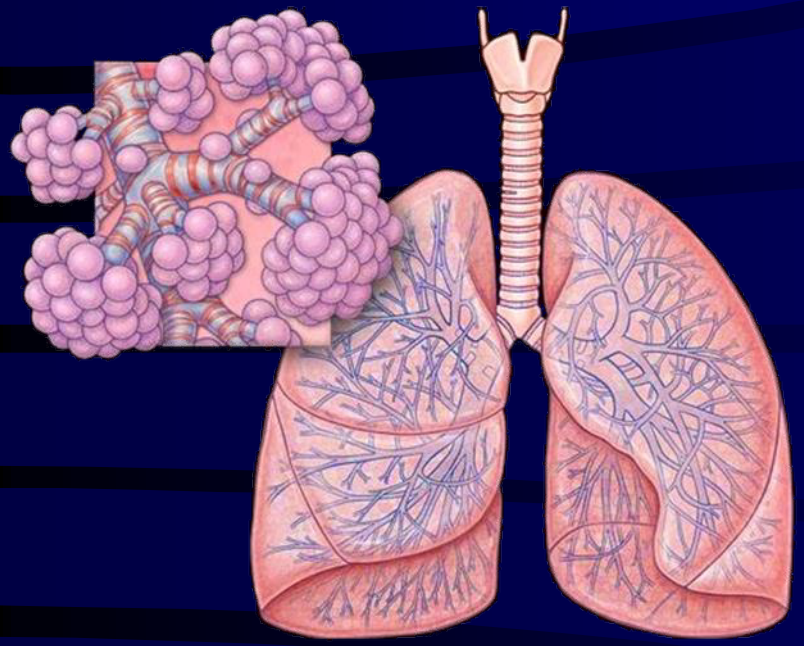
Lieske et al. 2000



Benacka, 2002

Sites of pathological disturbances



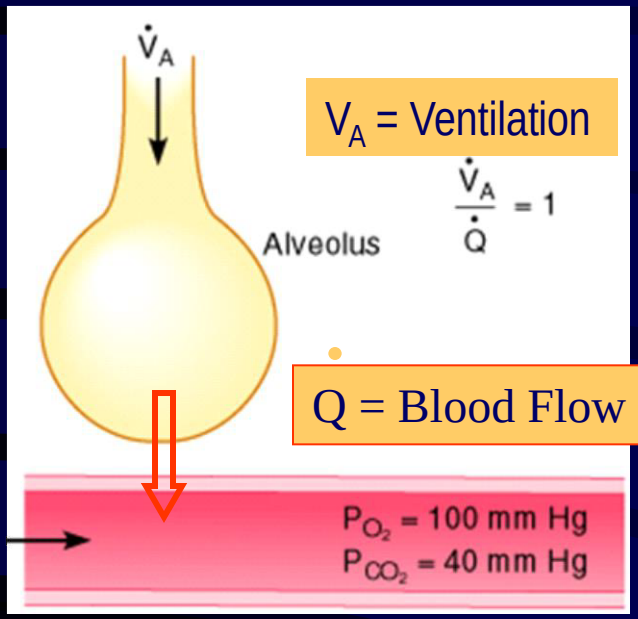


4

Ventilation – perfusion disturbances

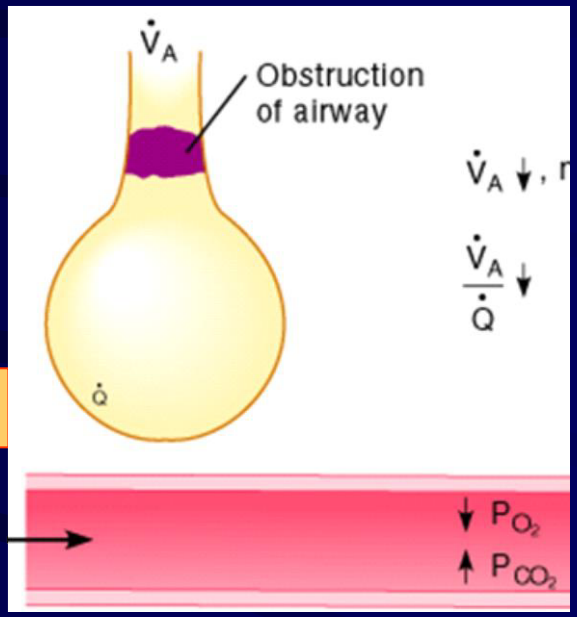
Ventilation-Perfusion Matching

Normal



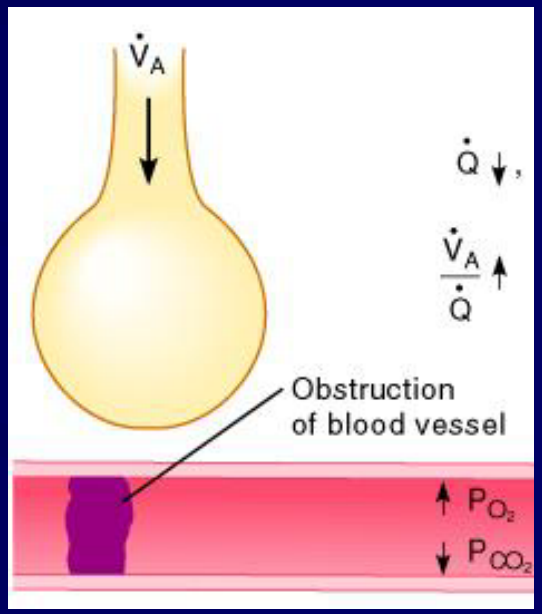
V_A/Q = Ventilation-Perfusion Ratio

Airway Obstruction



V_A decreases
Q does not change
VA/Q decreases

Capillary Obstruction



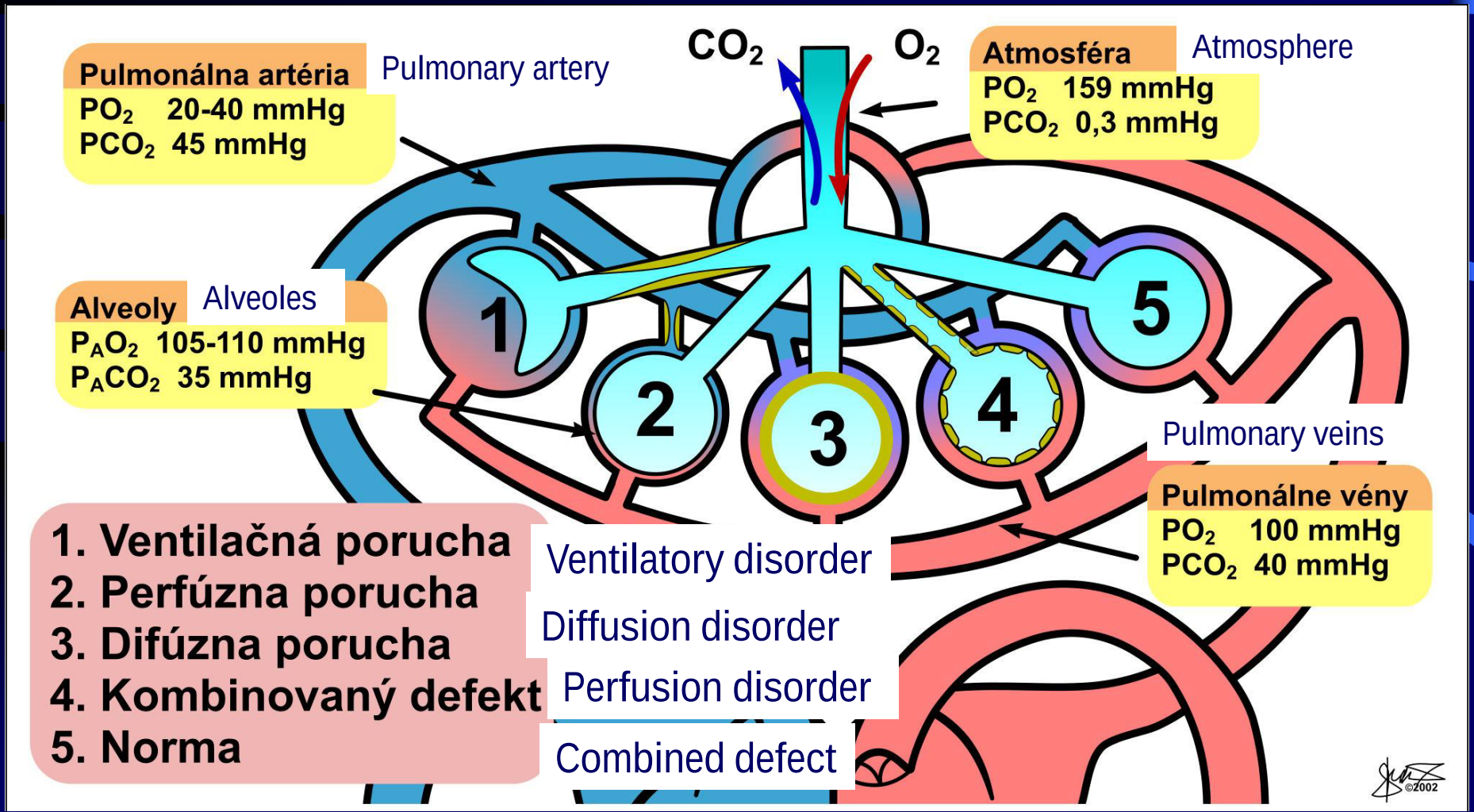
V_A does not change
Q decreases
VA/Q increases

Physiologic Shunt: Blood is flowing but is not oxygenated

Physiologic Dead Space: Air flows but there is no blood to oxygenate

Alteration of V/ P parameters

Ventilation Perfusion Difusion



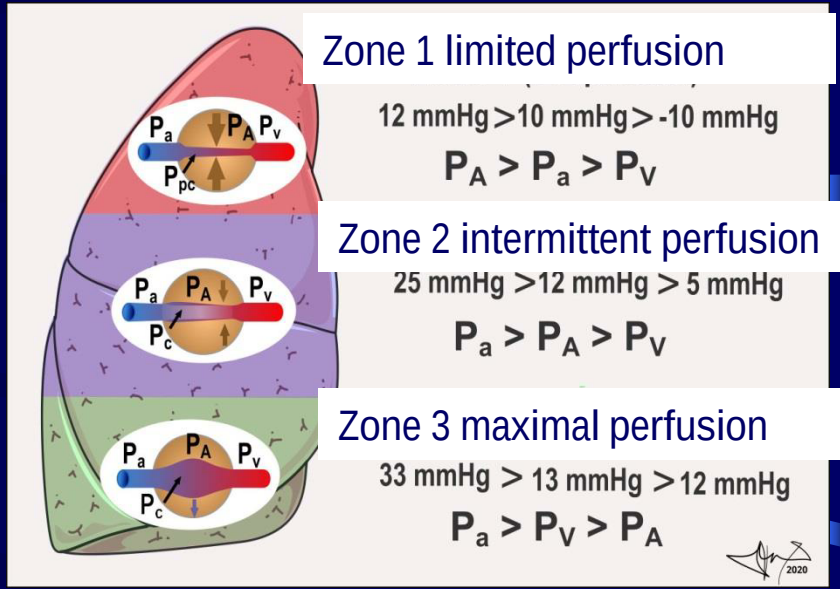
West zones

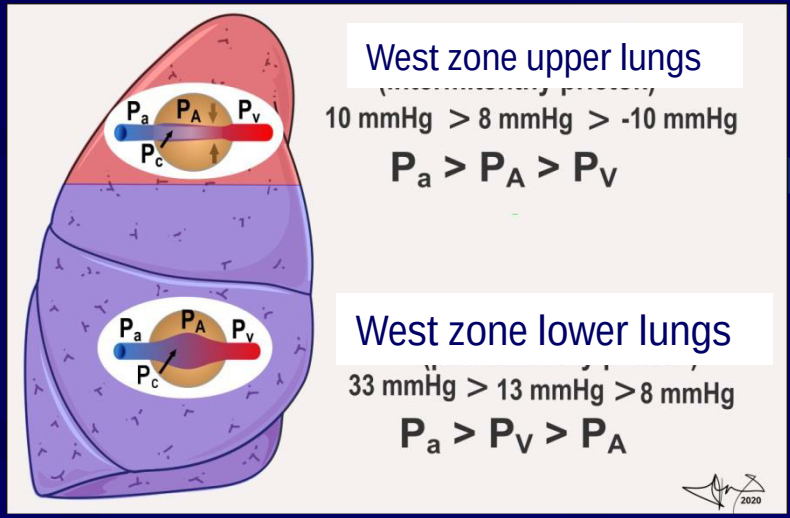
External respiration:

- lung ventilation and air distribution in the lungs
- diffusion of gases across the alveolar-capillary membrane
- perfusion of the lungs and distribution of blood in the lungs

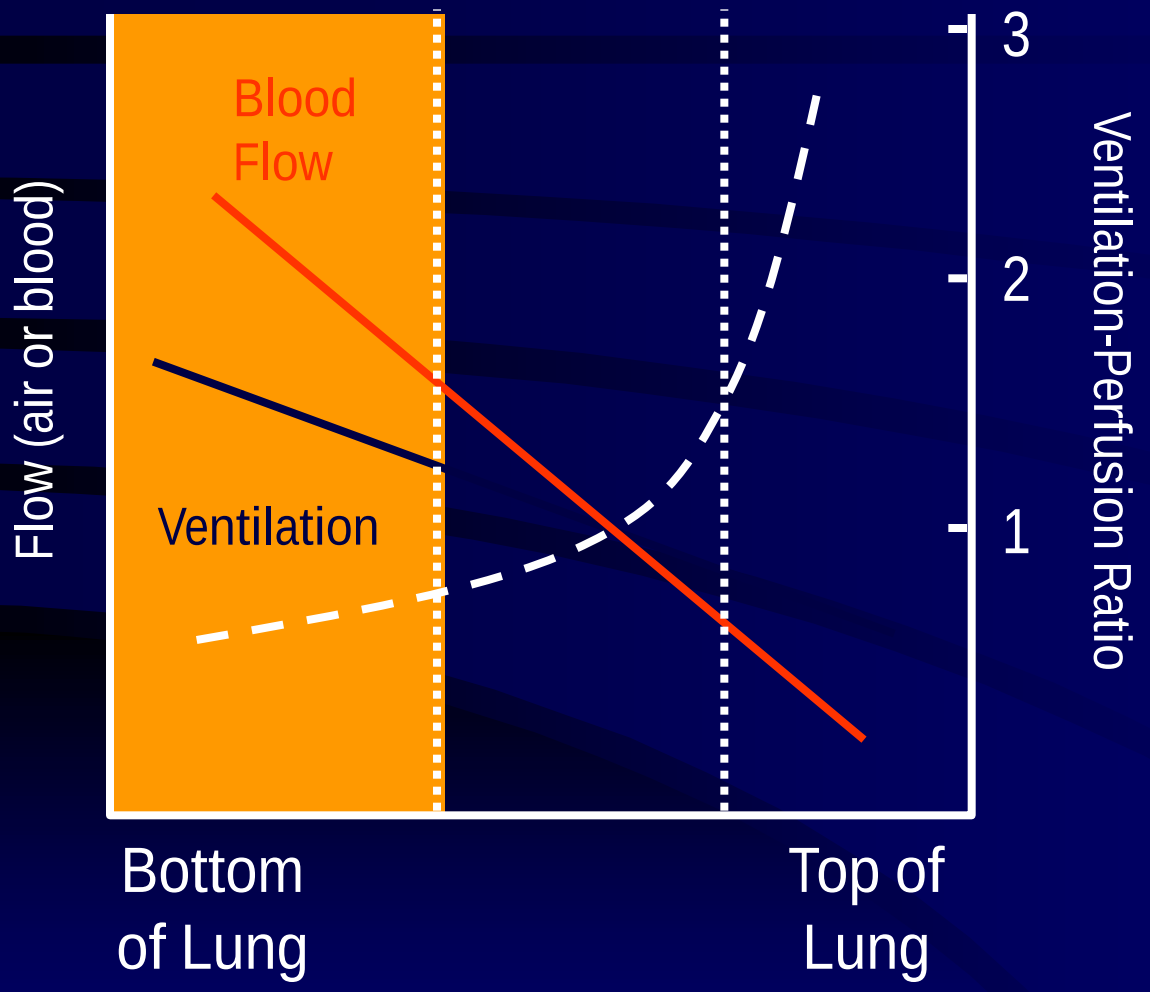
Ventilatory – perfusion ration in alveoli

- Standing, lying, sitting - differences in tidal volumes and perfusion of individual lung regions
- Lung geometry - female barrel-shaped lump - center (growing fetus); men triangular chest - base
- Lower lungs - very well perfused from a. pulmonalis (+ 9 mmHg); a tendency to fluid leakage; respiration in men
- Middle lungs - optimally ventilated (women) ; fairly perfused
- Upper lungs - poorly perfused; normally poorly ventilated (ortopnoe; deep breath)





Differences in Blood Flow and Ventilation



At the bottom of the lung

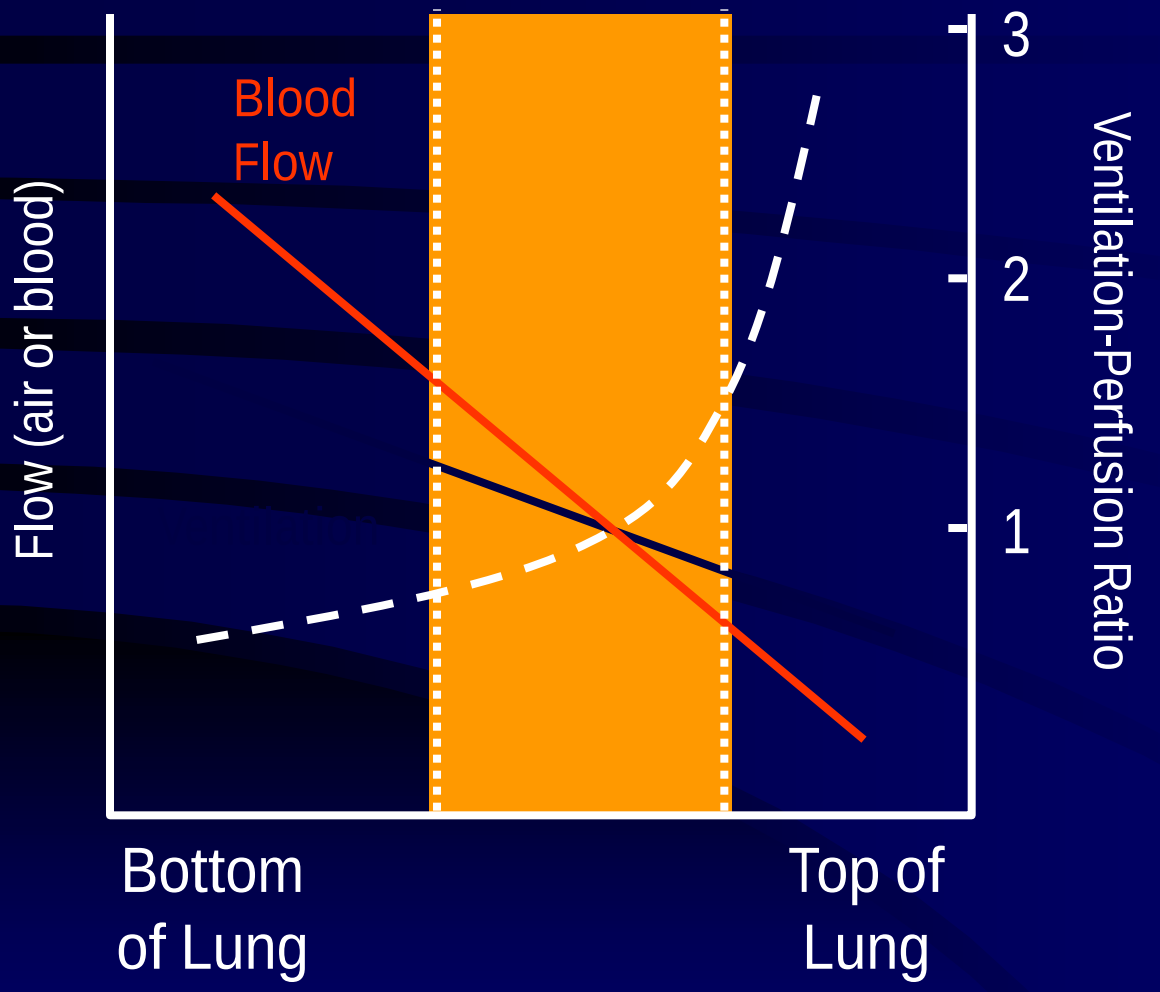
Ventilation < Blood Flow

$\dot{V}_A/\dot{Q} < 1$

Not all of the blood gets oxygenated

"Physiologic Shunt"

Differences in Blood Flow and Ventilation

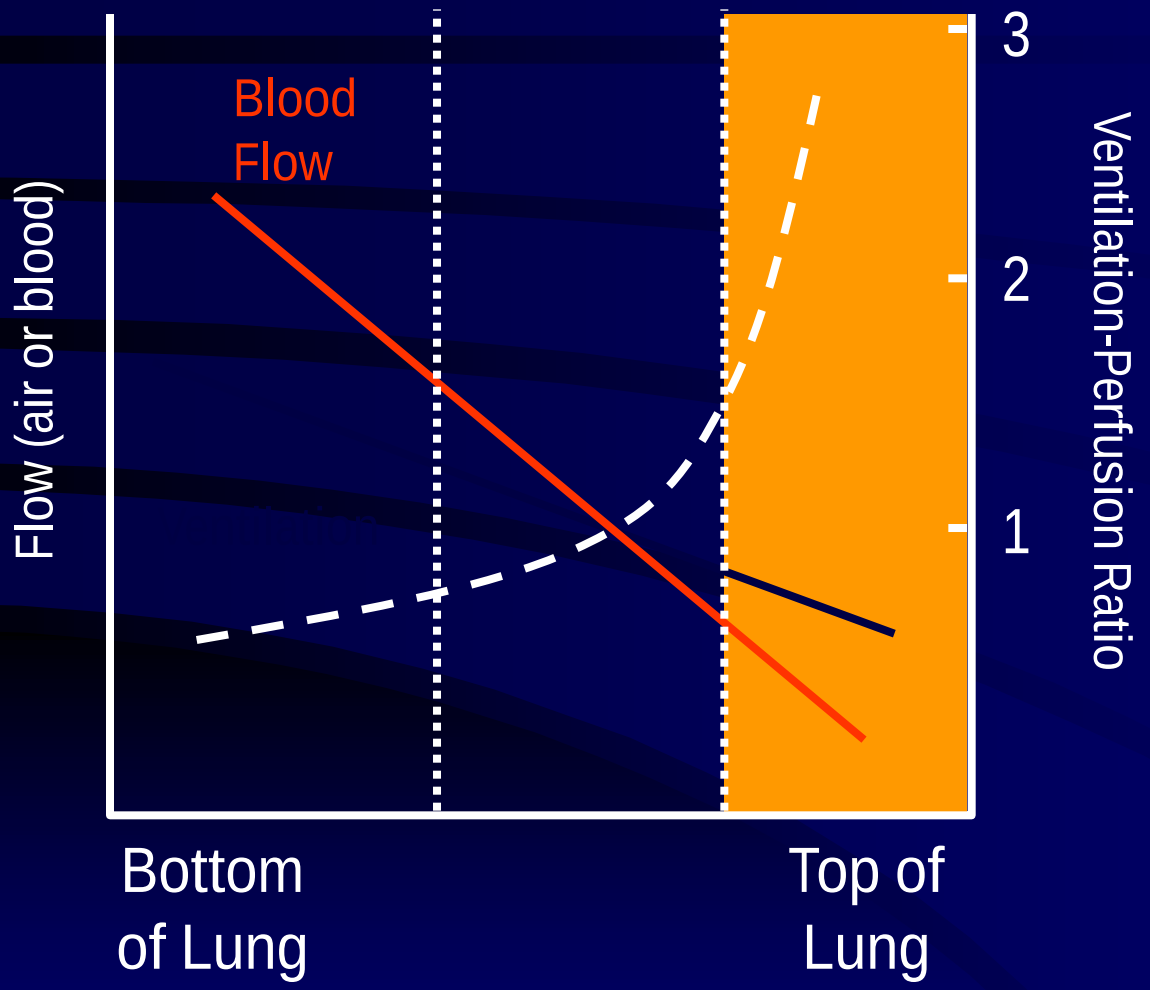


In the middle of the lung

Ventilation ≅ Blood Flow

$\dot{V}_A/\dot{Q}$ is approximately 1

Differences in Blood Flow and Ventilation



At the top of the lung

Ventilation > Blood Flow

$\dot{V}_A/\dot{Q} > 1$

Physiologic Dead Space

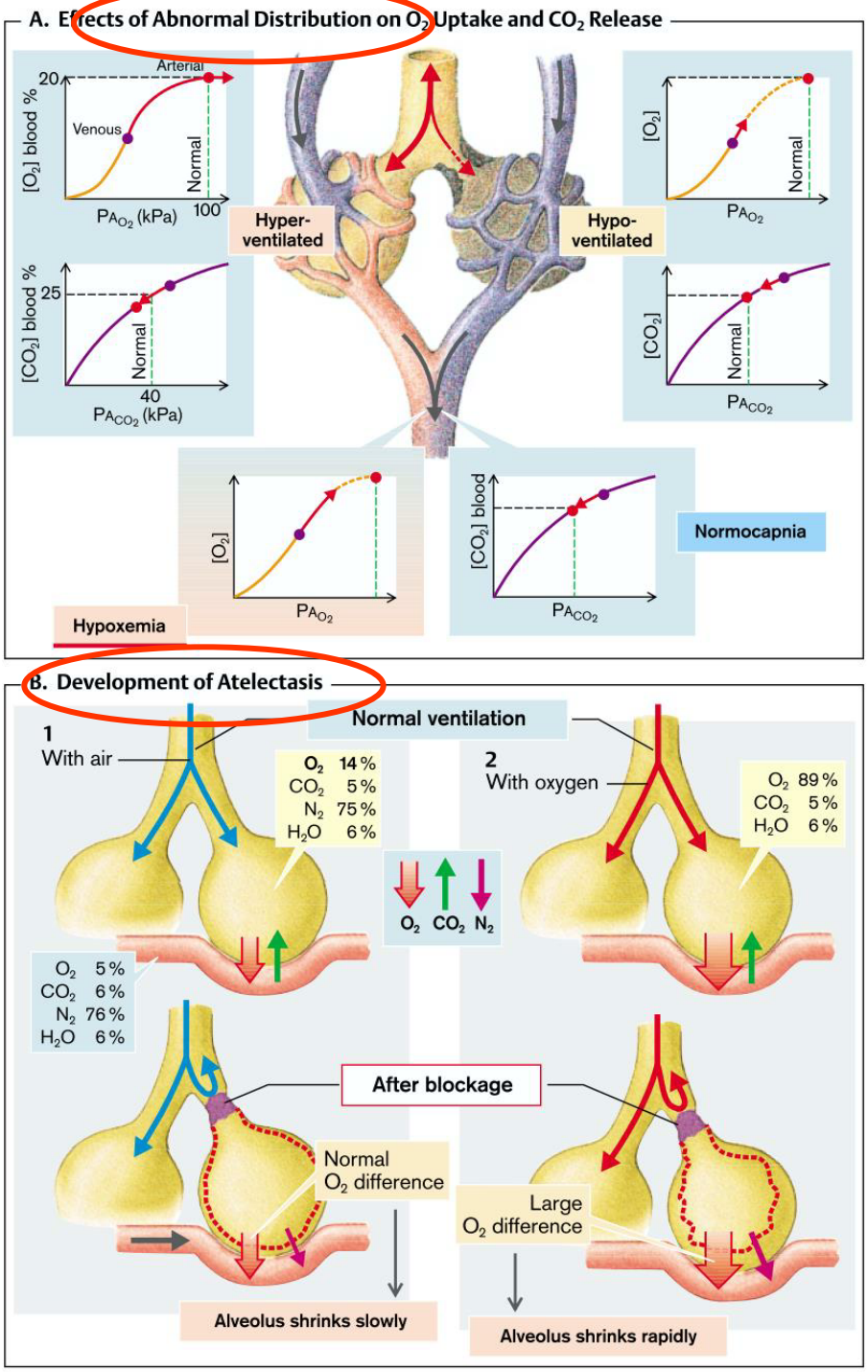
V/P disorders

Limited perfusion of ventilated parts

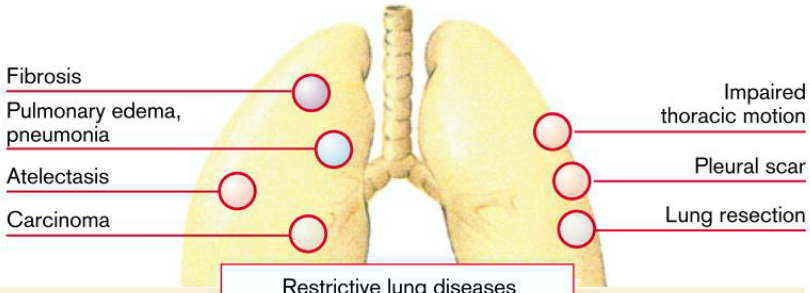
- Macroemoblia of lungs (legs, pelvis)
- Mikroembolism (inflammation)
- Pulmonary fibrosis

Limited ventilation of perfused areas

- Central breathing disorder – apnoe (toxic, sleep, etc.),muscle paralysis
- Asthma , chronic bronchitis, emphysema
- Bronchiolitis, Pleuritis
- Tumors – closing a lumen
- Functional arteriovenous shunts



A. Causes and Effects of Restrictive Lung Diseases



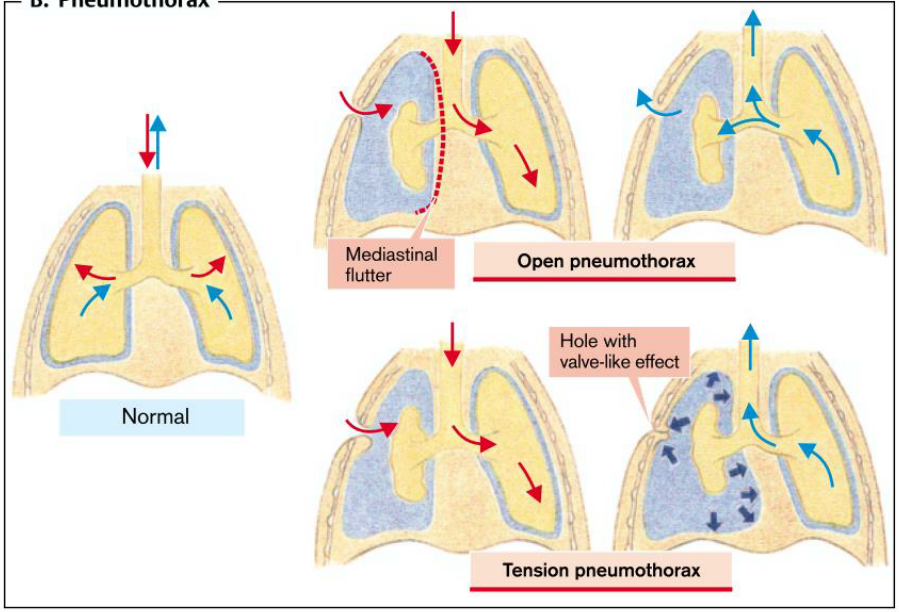
Restrictive lung diseases

Diffusion surface ↓ → Diffusion abnormality → S_{O_2} / P_{O_2} → **Hypoxemia**

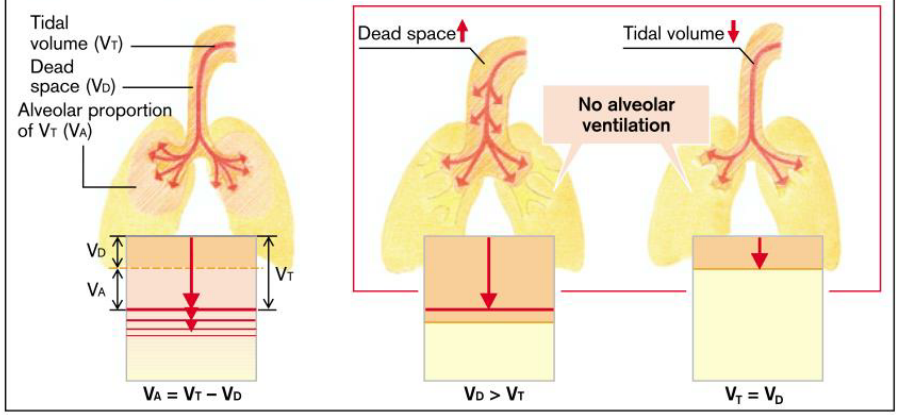
Loss of parenchyma → Compliance ↓ → Work of breathing ↑ → $\dot{V}$ (0.5 / 0 / 0 / P_{pl} / −1) → **Dyspnea**

Vascular cross sectional area ↓ → Vascular resistance ↑ → **Pulmonary hypertension**

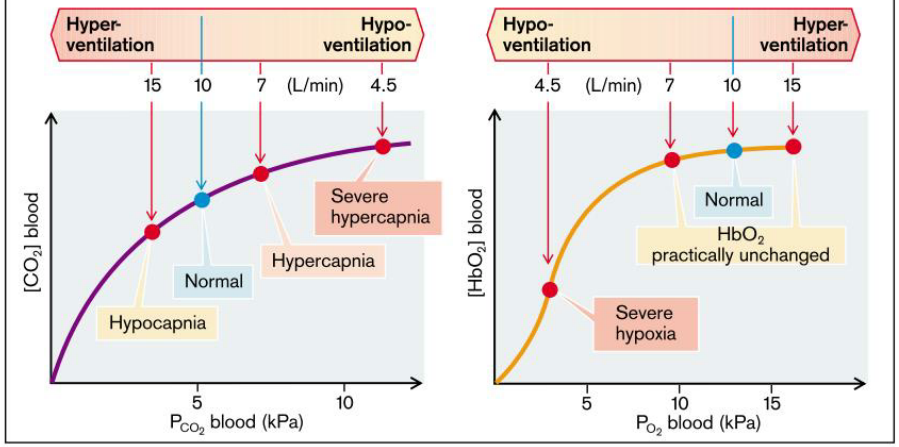
B. Pneumothorax



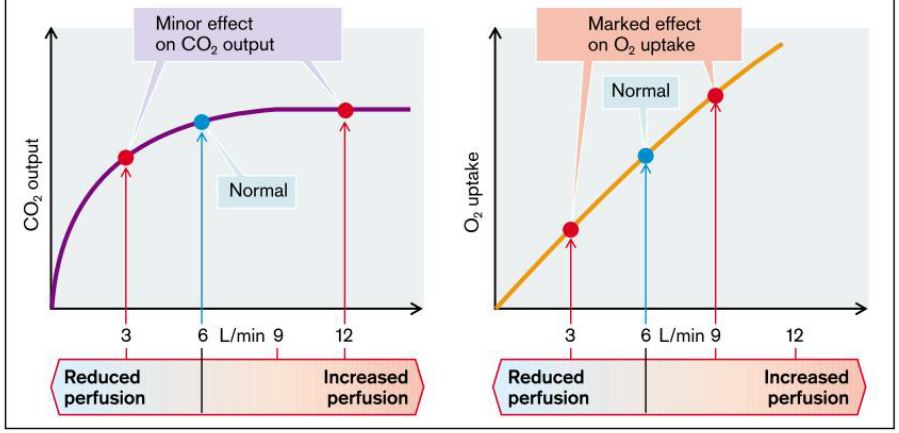
A. Dead Space (V_D), Alveolar Volume (V_A) and Tidal Volume (V_T)



B. O_2 and CO_2 of Arterial Blood in Abnormal Ventilation



C. CO_2 Release and O_2 Uptake at Different Perfusion Levels



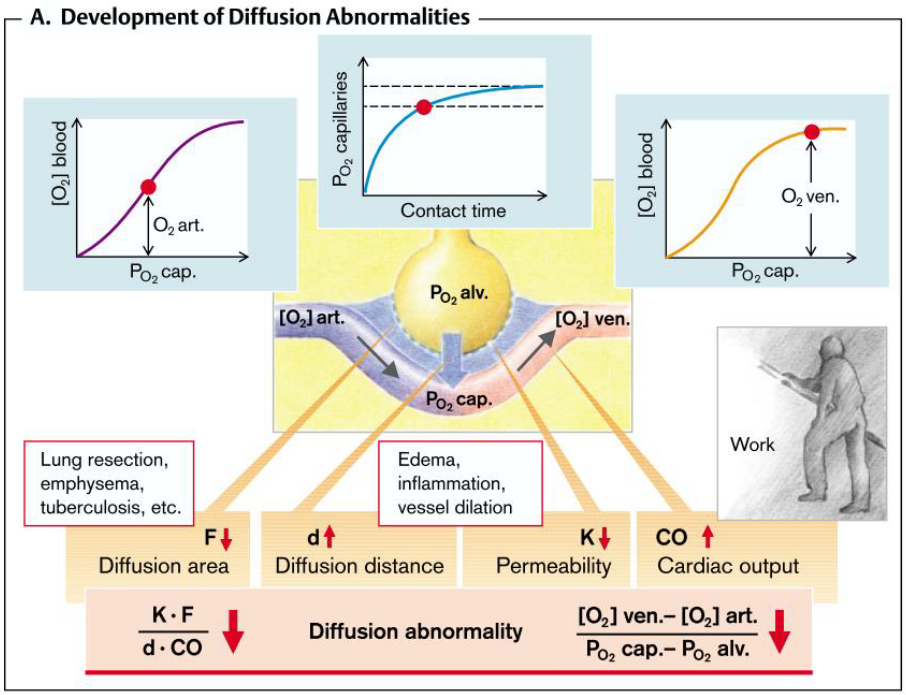
Diffusion disorders

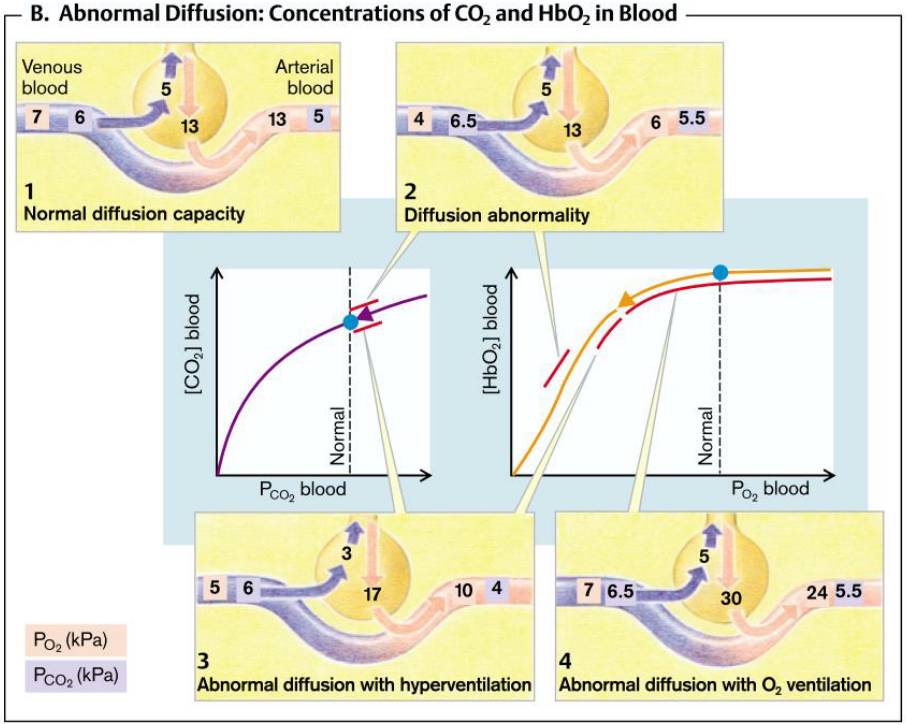
$\dot{M} = K \times F\,(PA - Pk)/d$

= amount of gas ($\dot{M}$) that diffuses across the diffusion barrier between alveoli and blood per unit time; diffusion area (F), partial pressure between alveolar gas (PA) and blood (Pk), and inversely proportional to length of the diffusion pathway (d) Krogh's diffusion coefficient K = 20 x greater for CO_2 than for O_2.

Diffusion capacity D = K × F/d) = 230 ml/min/kPa (1.75 l/min.mmHg) in a healthy p.

- Lung resection
- Emphysema, Pneumónia
- Interstitial, alveolar lung edema
- Left sided or global heart failure
- Fibrotic changes in lungs (silikosis, pneumokonioses)





PULMONARY EDEMA

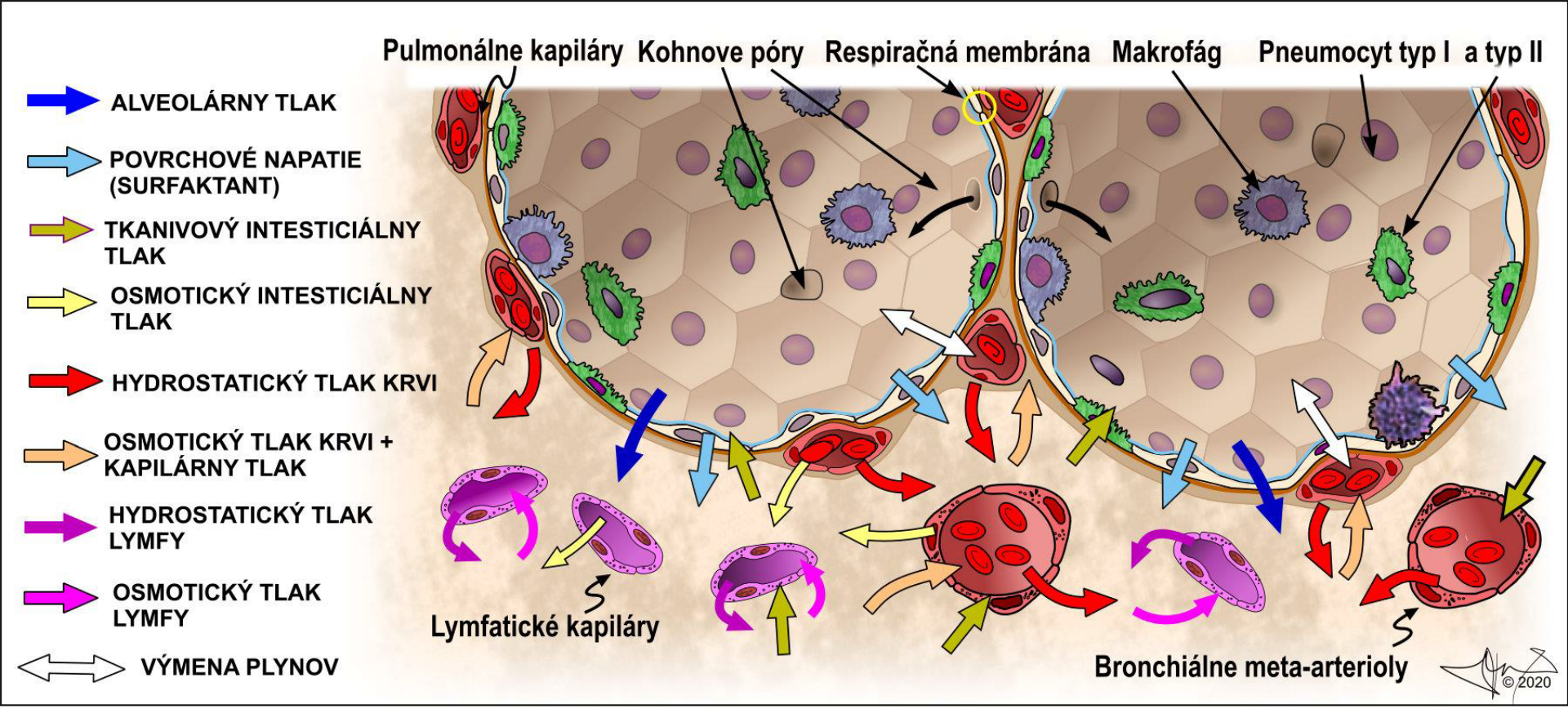
- Def.: Pulmonary edema is an increase in extravascular lung water in pulmonary interstitium or in aleveli
- Interstitial edema does not impair function
- Alveolar edema cause several gas exchange abnormalities
- Movement of fluid is governed by Starling's equation

  $QF = KF [(P_{IV} - P_{IS}) + \sigma (\pi_{IS} - \pi_{IV})]$ QF = rate of fluid movement

  KF = membrane permeability

  P_{IV} & P_{IS} are intra vascular and interstitial hydrostatic pressures π_{IS} and π_{IV} are interstitial and intravascular oncotic pressures

  σ reflection coefficient
- Lung edema is cleared by blood vessels and lymphathetic vessels



- Decreased oncotic pressure (liver, kidney, kwasiorkor) (Norm: 3,3 kPa, 25 mmHg)
- Increased hydrostatic blood pressure (accumulation of blood in vessels) (Norm:0,9-1,3 kPa, 7-10 mmHg)
- Increased hydrostatic lymphathetic pressure (inflammation, tumours) (Norm:0,1-1,3 kPa, 7-10 mmHg)
- Decreased osmotic lymph pressure (Norm: 1,2-1,7 kPa, 9-13 mmHg)
- Decreased alveolar surface tension (alovelar damage, unmaturated newborns) (Norm: 0,3 kPa, 2 mmHg)
- Increased interstitial osmotic presssure (inflammation) (Norm: 0,4-0,7 kPa, 3,5 - 4 mmHg)
- Decreased alveolar pressure (atelectasis)