



Respiratory Physiology

ED Primary Teaching

Functions of the respiratory system

- * Gas exchange with O_2 and CO_2
- * Surfactant production
- * Defence - IgA and macrophages
- * Filter - pollutants and thromboembolism
- * Metabolises some compounds - converts AT I to II, inactivates bradykinin, breakdown of prostaglandins and leukotrienes
- * Reservoir of blood

Respiratory Physiology

- * **Oxygen transport chain**

- * Ventilation —> Pulmonary gas exchange —> Oxygen in blood —> Local tissue perfusion —> Diffusion at tissue level —> Tissue utilisation of oxygen

Ventilation

- * The physiology of room air:
 - * Contains about 21% O₂
 - * 78% nitrogen
 - * 1% other gasses
- * It is the role of ventilation to provide oxygen to the gas exchanging mechanisms of the lungs
- * Ventilation is also inherently related to blood flow
- * Ventilation:
 - * Airway anatomy
 - * Resp muscle function and chest wall mechanics and lung volumes
 - * Neurological control of ventilation

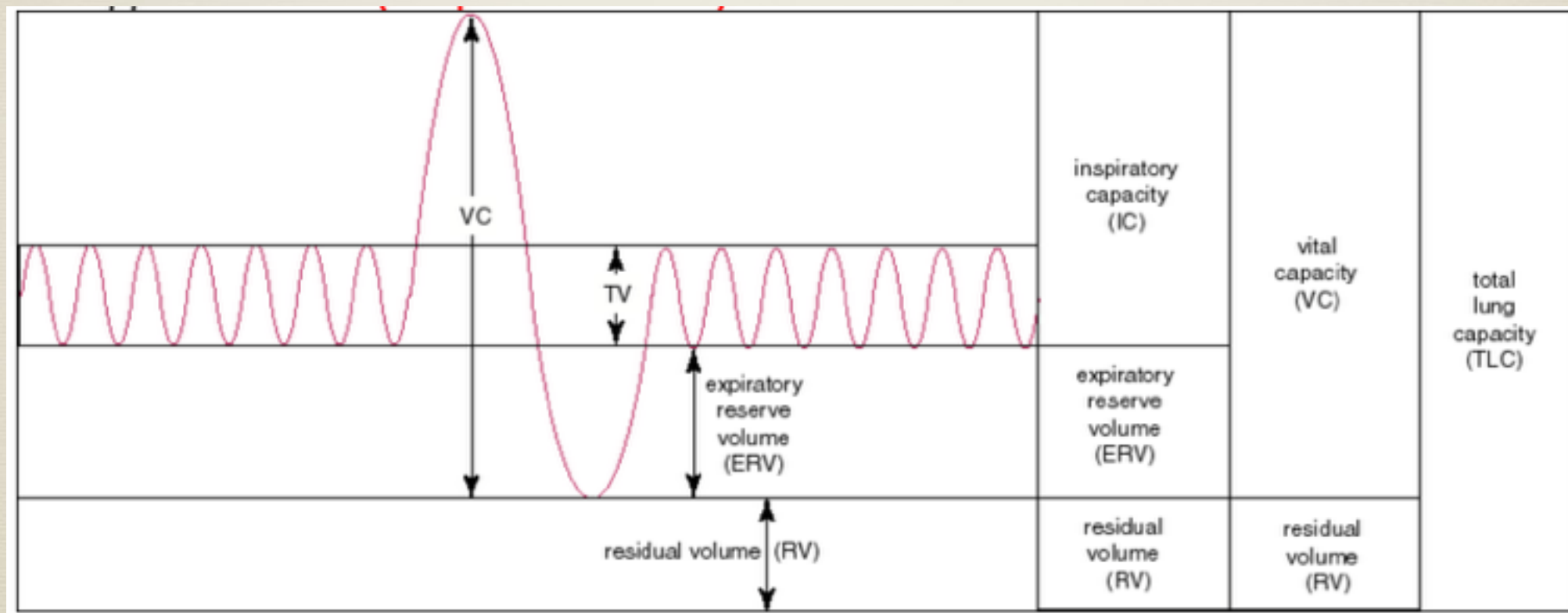
Airway and airflow

- * Trachea divides into R and L bronchi which then divide into lobar and segmental bronchi
- * These continue to divide down into segmental bronchi and terminal bronchioles which are the smallest airways without alveoli
- * Bronchi and terminal bronchioles make up the conducting airway the function of which is to lead the air to the gas exchanging region and these airways constitute ANATOMICAL DEAD SPACE - 150mL
- * The anatomical portion of the lung distal to these terminal bronchioles form the acinus and is the functional respiratory unit

Respiratory muscular function

- * Inspiratory muscles
 - * Diaphragm
 - * External intercostals
 - * Accessory inspiratory muscles such as the scalene and SCM
 - * Muscles of head and neck
- * Expiratory muscles - usually a passive process which relies of the elastic recoil of the lungs
 - * When under anesthesia or at extremes of exercise expiration may become active with activation of the abdominal wall muscles or internal intercostals

Lung Volumes

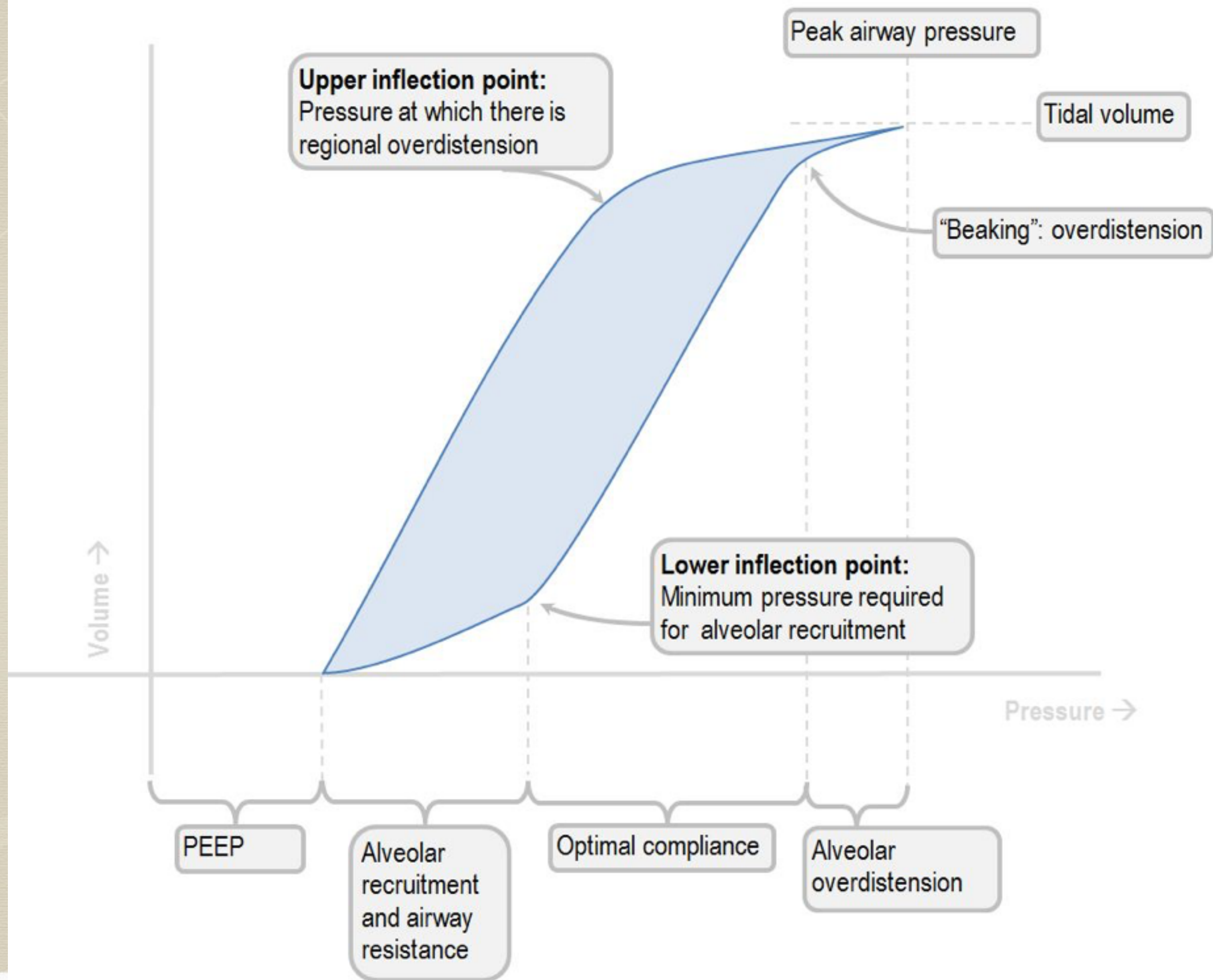


- * Total lung capacity - amount of gas in the lungs at a maximal inspiration (normal 6L for adults males, and 4.2L for adult females)
- * Vital capacity - amount of gas that can be exhaled after a maximal inspiration
- * Residual volume - amount of gas remaining in the lungs after a maximal expiration (20% of the total lung capacity)
- * Total lung capacity = Vital capacity + Residual volume

- * Tidal volume = the amount of gas an individual inspires or expires during normal breathing
 - * 7 - 8% of total lung capacity
 - * Approximately 500 mL
 - * $\text{Tidal volume} \times \text{RR} = \text{minute ventilation}$
- * Inspiratory reserve volume - the amount of gas an individual can inhale above a normal tidal inspiration (normal 60% of TLC)
- * Expiratory reserve volume - the amount of gas an individual can exhale below a normal tidal expiration (normally 20% of TLC)
- * $\text{TV} + \text{IRV} + \text{ERV} = \text{Vital capacity}$
- * Functional residual capacity - The amount of gas in the lungs after tidal expiration - usually 40% of TLC (cannot be measured with spirometry and is measured indirectly using helium dilution or nitrogen washout)
 - * $\text{The FRC} = \text{ERV} + \text{RV}$
 - * The FRC is the balance between the tendency of the chest wall to spring out and the tendency of the lung to collapse

Lung capacities

- * 4 types of capacities - inspiratory capacity, TLC, VC, FRC
- * The addition of two volumes makes a capacity
- * Inspiratory capacity - the amount of gas an individual can inhale at the end of tidal expiration
 - * Includes TV and IRV - 60% of TLC



Pressure volume curve

- * A graphical representation of the relationship between pressure and volume during inspiration and expiration
- * Bottom of loop is either 0 or PEEP and top of loop is peak inspiratory pressure
- * Compliance is an imaginary line drawn between the start of inspiration and expiration
 - * Increased compliance (left shift) - emphysema and asthma (small changes in pressures generate large changes in volume)
 - * Decreased compliance (right shift) - ARDS (large changes in pressures are required to generate small changes in volume)
- * Work of breathing is area within the curve and equals pressure x volume

Closing capacity

- * The volume at which the small airways close during expiration
- * Under normal circumstances the FRC is greater than the closing capacity, however if the FRC was to decrease then this would no longer be the case and the small airways may close at the end of normal tidal expiration - causing collapse and atelectasis
- * Closing capacity increases with age and equals FRC in a sitting patient by the age of 66

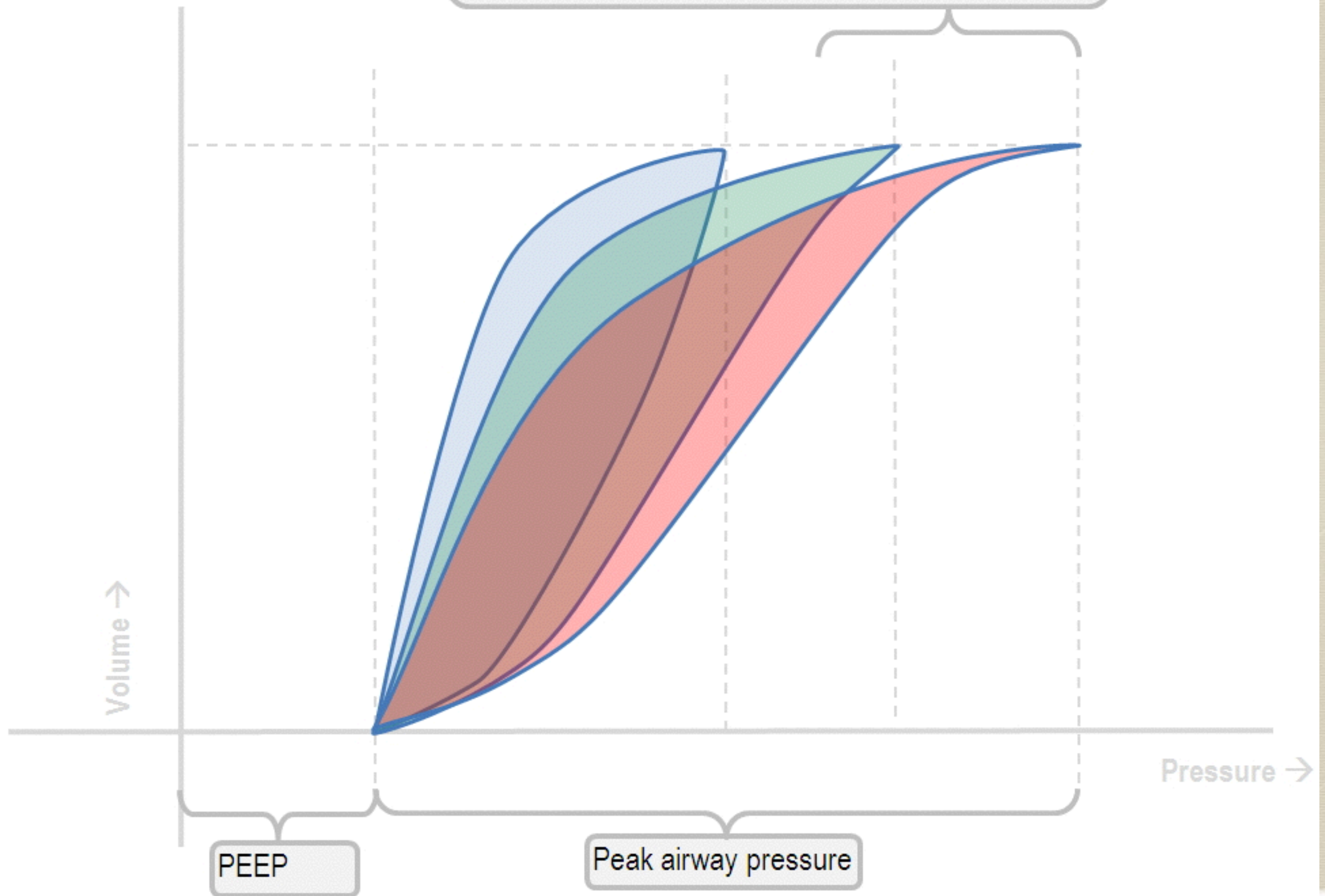
Chest wall mechanics

- * Compliance - change in volume/change in pressure
 - * Can be a static principle when there is no airflow and is determined by the change in volume for a given pressure
 - * Can be a dynamic relationship during breathing, where airflow resistance becomes a factor
 - * The lung is generally more distendable at lower volumes and stiffer at higher volumes
 - * The compliance of a lung can be altered in pathology such as fibrosis or alveolar oedema (decreased) and COPD and asthma (increased)
 - * This means that for a given change in volume there is a greater rise in system pressure and has implications for mechanical ventilation

Chest wall mechanics - some pathophysiology

- * Asthma - increased lung compliance
 - * Compliance is lowest at FRC (think about the first breath into a balloon) meaning that a large change in pressure is required to facilitate a small change in volume
 - * In patients with asthma their FRC is increased because of air trapping due to DAC due to broncho-constriction
 - * Therefore during an attack the compliance is increased (like breathing air into an already filled balloon)
- * ARDS - decreased lung compliance

As compliance decreases, volume control ventilation yields higher and higher peak airway pressure in order to achieve the same volume.



Deadspace

- * The volume of a breath that does not participate in gas exchange - it is ventilation without perfusion
- * Physiological or total dead space is the sum of anatomical dead space and alveolar dead space
- * Anatomic dead space is the volume of gas within the conducting zone, as opposed to the transitional and respiratory zone and includes trachea, bronchioles, bronchus and terminal bronchioles
 - * It is approximately 2ml/kg in the upright patient
- * Alveolar dead space is the volume gas within the under perfused alveoli and thus not participating in gas exchange either - it is usually negligible in the awake and healthy patient
- * The ratio of physiological dead space to tidal volume is 1:3

Regional differences in ventilation and blood flow

- * Distribution of blood in lungs
 - * Decreases almost linearly from the bottom to the top
 - * More blood in dependent areas
 - * If that section of the lung is ventilated but not perfused this is alveolar dead space
- * Multiple physiological zones
 - * Zone 1 - alveolar pressure > arterial pressure > venous pressure
 - * Zone 2 - arterial pressure > alveolar pressure > venous pressure
 - * Zone 3 - arterial pressure > venous pressure > alveoli pressure

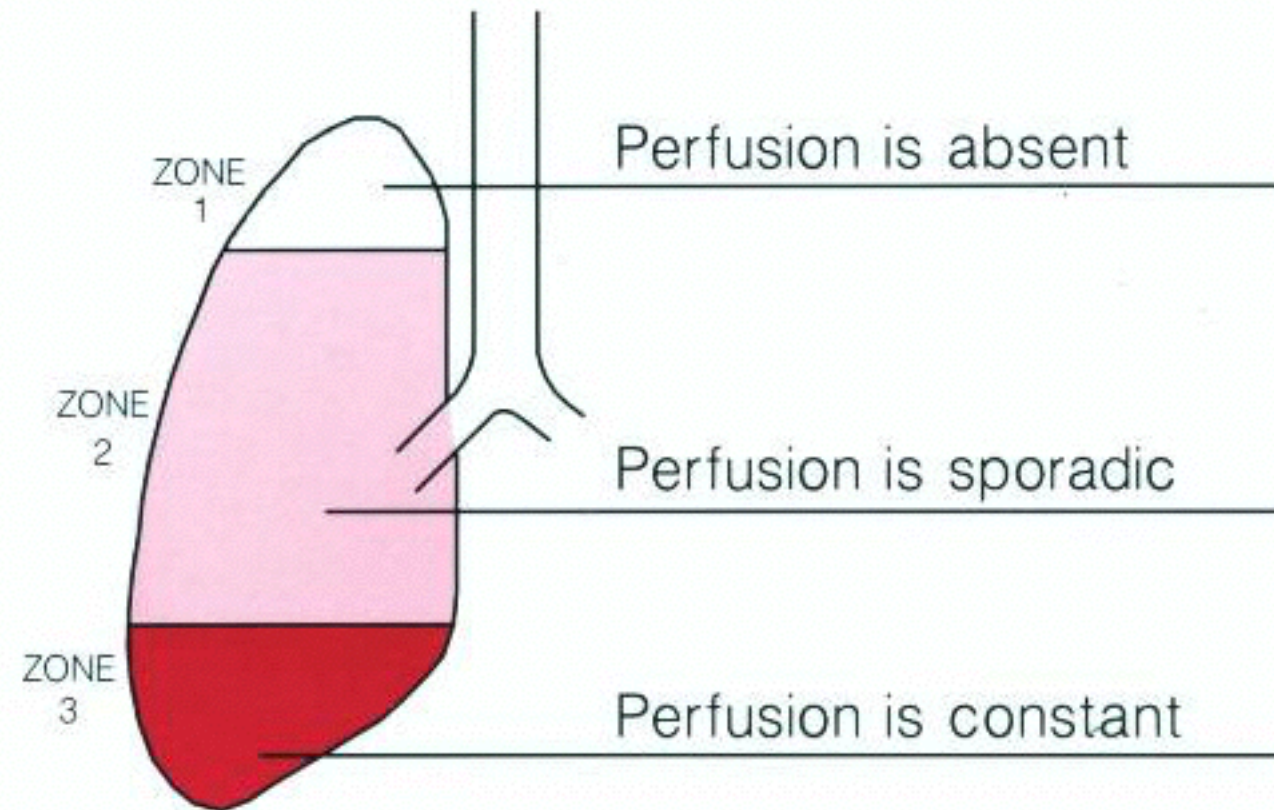


Figure 4-5. Pulmonary perfusion zones. © W. B. Saunders 1990. *Clinical Blood Gases: Application and Noninvasive Alternatives*.

Properties of pulmonary vasculature

- * Pulmonary artery pressure is 15mmHg (8 - 25)
- * LAP is about 5mmHg
- * This means that the entire CO moves across a circuit who's pressure difference is only 10
- * The pulmonary vascular resistance = $10\text{mmHg}/5\text{L/min} = 2\text{mmHg/L/min}$
- * Comparable calculations for the systemic circuit show its resistance to be about 10 times greater
- * What factors effect pulmonary resistance
 - * The high distensibility, the small amount of vascular smooth muscle, and the low intravascular pressures lead to a greater importance of passive extravascular effects of pulmonary resistance (lung volumes, alveolar and intra pleural pressures etc.)
- * A paradoxical characteristic of the pulmonary vasculature is the ***higher flows generally associated with lower resistance***
 - * ***Why?***

Flow and resistance in the pulmonary circulation

- * Why does PVR fall as flow increases? Two reasons
 - * Capillaries that were already open, now distend further
 - * Capillaries that were formerly closed now open
- * Alveolar and intrapleural resistance - PVR depends on the transmural pressure gradient.
 - * $\text{Pressure inside (blood vessel)} - \text{pressure outside (blood vessel)}$
 - * As this gradient decreases the diameter of the vessel decreases and therefore VR increases

Lung volumes and vascular resistance

- * Lung volumes effect pulmonary circulation - this interaction differs depending on wether the lung is above or below FRC
 - * Above FRC - as lung volume increases above FRC the pulmonary vessels outside the alveoli are distended so their resistance falls, on the other hand septal capillaries in the alveolar wall are stretched so that their diameters are reduces - the net effect is an increase in overall pulmonary vascular resistance since the narrowing of the septic capillaries is the dominant effect
 - * Below FRC - As lung volume decreases below FRC the pulmonary vessels outside the alveoli tend to collapse as their walls are no longer being stretched by the surrounding lung tissue - this leads to an increase in resistance and this causes and increase in pulmonary vascular resistance

Gravity and vascular resistance

- * In general flow is greatest in the dependent portion of the lung and least in the non dependent portions of the lung
- * Zones of the lung
 - * Zone 1 - alveolar pressure is greater than both local pulmonary arterial and venous pressures
 - * Therefore vessels are compressed and there is no flow
 - * $P_{alv} > P_a > P_v$
 - * Zone 2 - Pulmonary arterial pressure is greater than alveolar pressure
 - * The alveolar pressure is greater than the venous pressure so the downstream pressure is alveolar pressure
 - * This situation is determined vascular waterfall where the flow is independent of the eventual venous pressure and depends only on the difference between pulmonary arterial pressure and alveolar pressure
 - * $P_a > P_{alv} > P_v$
 - * Zone 3 - Pulmonary artery pressure is greater than pulmonary venous pressure and alveolar pressure
 - * The venous pressure is greater than the alveolar pressure so the flow is dependent on the AV pressure difference
- * Under normal conditions zone 1 does not occur
- * A patient on a PPV with PEEP may have substantial amount of zone 1 because alveolar pressure is always positive

Chemical factors and their role in pulmonary blood flow

- * Hypoxic vasoconstriction - by far the most important modulator of smooth muscle tone in the pulmonary arterioles is the partial pressure of O₂ in the alveolus PAO₂.
- * Smooth muscle in pulmonary arterioles responds in the opposite way to SM in systemic arterioles when exposed to hypoxic conditions
- * Low PO₂ produces constriction in pulmonary vessels
- * This degree of hypoxic vasoconstriction becomes more pronounced once PaO₂ drops below 100mmHg
- * The receptors (not neurally regulated) that regulate this response respond to PAO₂ rather than PO₂ in the blood and it is unclear how this happens

The utility of hypoxic vasoconstriction

- * Limits blood flow through the lung of the foetus —> elevation of PAO_2 with the first breath greatly reduces resistance to blood flow in the lung
- * Improves matching of blood flow with ventilation
 - * If a region of lung is poorly ventilated, PAO_2 in the region decreases, the resulting vasoconstriction decreases blood flow to that region - therefore blood that doesn't go there will go to the alveoli that are more adequately ventilated
- * Changes in altitude
 - * Produces vasoconstriction throughout the circuit which leads to better perfusion of the apical regions and improves the VQ ratio in their region of the lung
- * Consequences of hypoxic vasoconstriction
 - * Increased work for RV and RVH —> irreversible growth in smooth muscle of pulmonary arterial walls leading to pulmonary HTN

Other functions of the respiratory system

- * Reservoir - when you sit from standing, blood from legs goes to RA, the RV pumps this into the lungs but not all of this makes it to the LV this prevents CO from increasing excessively as one goes from an upright to a lying down position
- * Filter - Acts as a vascular filter for small clots
- * Surfactant - made by Type II alveolar cells
- * Conversion of AT_I to AT_{II} by ACE
- * Makes IgA for immune system
- * Secretion of mucus
- * Lung removes serotonin from the blood
- * Prostaglandin E₂ is produced by the lungs of the fetus and it serves as a smooth muscle relaxant keeping the DA open