8. Mechanics of Breathing

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8. Mechanics of Breathing - 1

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Breathing Cycle

Inspiration

Diaphragm:
- Most impt mm of breathing
- Phrenic nerve C3,4,5
- ↑vertical diameter of chest and ↑transverse diameter of thorax
- norm tidal breathing:
  - diaphragm moves ~1cm
  - forced insp/exp moves ~10cm
- if diaphragm paralysed; insp ⇒ up (not down)
  ↵as intrathoracic pressure falls = paradoxical movmt

External Intercostals
- route down and forward
- pull rbs upwards and forwards ⇒ ↑lat (bucket) & ↑AP (pump handle) to chest
- supplied by same segment intercostal nerve
- paralysis has little effect on ventilation as diaphragm so good

Accessory Mm
- scalene & SCM

Expiration
- =passive during quiet breathing
  ↵due to elasticity of lung & chest wall

Abdominal Wall Mms
- =most impt mms in exp
- mm contraction ⇒ ↑abdo pressure ⇒ diaphragm pushed up

Internal Intercostals
- assist in active exp by pulling rbs down and inward
- also act isometrically to stiffen intercostal spaces preventing outward bulge

Intrapleural Space & Pressure
- anatomy:
  - lungs
  - visceral pleura
  - intrapleural space
  - parietal pleura
  - chest wall
- 2 opposing forces ⇒ -ve IPP (relative to atmosphere)
- -ve IPP ↓ = expanding pressure which prevents collapse of lung at FRC
  ↵ie take lung out of chest and it will collapse
- transpulmonary pressure (TPP) = alveolar pressure - IPP
- inspiration:
  - occurs due to ↓ing IPP ⇒ ↑ing TPP ⇒ expanding force on lung
  - work done by insp resp mms against elastic forces is stored as potential energy
  ↵work done against non elastic forces is lost as heat
- expiration:
  - quiet breathing:
    - passive by using up stored potential energy to overcome frictional forces
    - return to FRC
Elastic Recoil of Lung

- Pressure surrounding lung is sub-atmospheric because of elastic recoil of lung
- Factors causing elastic recoil:
  - Surface tension (70%):
    - At air-water interface lining alveoli
  - Yellow elastin & collagen in (30%):
    - Alveoli walls
    - Around vessels
    - Bronchi

Surface Tension

- Impt factor in pressure-volume curve
- Tension from liquid film lining the alveoli
- Measured in (dynes): force acting across line 1cm long in surface of the liquid
- Created by attractive forces between adjacent molecules
  - These stronger than between liquid & gas
  - \[ \therefore \text{liquid surface area tries to become as small as possible} \]
  - \[ \therefore \text{seen in bubbles } \Rightarrow \text{sphere} \]
- Generating pressure to make a bubble predicted using:

Laplace’s Law: \[ \text{it takes a certain inflation pressure to support the surface tension developed at a liquid-gas interface} \]

\[ P = 2T/R \]

For a fluid lined alveolus: \[ P = 2T/R \]
For a soap bubble: \[ P = 4T/R \] (because it has 2 surfaces, ie 2 x 2T)
For a tube (eg vessel): \[ P = T/R \] (because radius = only in 2 dimensions as opposed to 3D in spheres)

\[ \therefore \text{pressure inside bubble } > \text{outside bubble} \]
  - This directionally proportional to surface tension (T)
    - Inversely proportional to radius (R)
- In lung alveoli are not sealed bubbles and each bubble empties into each other
- Smaller bubbles have ↑ed pressure \[ \therefore \text{emptying small bubble } \Rightarrow \text{bigger bubbles} \]
  - Surfactant helps prevent this

- Change in surface tension in lung with area:
  - Tension very low when small area
  - If add detergent or saline: the effect of area on surface tension is abolished

Surfactant

- Surfactant =
  - Lipids (90%) – mainly phospholipid:
    - Of which 80% = dipalmitoyl phosphatidylcholine (DPPC)
    - Rest neutral lipids
  - Proteins – incl surfactant specific proteins
  - Carbohydrate (CHO)
- 2 Types of alveolar epithelial cells:
  - Type I cells –
    - Long cytoplasmic extensions spreading over alveolar walls
    - If injured (eg prolonged exposure to high O2 can be replaced by type II cells
    - Develop from type II cells
      - Pneumocytes are naughty – when no one is looking (beside) 2 become 1)
  - Type II –
produce surfactant & can also take it up
larger cuboidal cells

- phospholipid DPPC synthesised in lung from fatty acids made in lung or gathered from blood
- surfactant synthesis & turnover v fast
  \[ \downarrow \text{if blood flow } \downarrow \text{ed to PE } \Rightarrow \downarrow \text{surfactant} \]
- saline abolishes effect of surfactant
- surfactant produced late in fetal life \( \Rightarrow \) risk with premature birth

- surfactant works by:
  - DPPC molecules:
    - hydrophobic FA tails protrude into gas phase
    - charged hydrophilic protrude into water phase
      \( \Rightarrow \) align themselves in surface
    - intermolecule repulsive forces from charged hydrophilic heads then counteract normal attracting forces between lipid surface molecules
    - \( \downarrow \) in surface tension is \( \uparrow \) ed when surface area is smaller cos DPPC are crowded closer together \( \Rightarrow \) repel each other more
  - function of surfactant:
    - \( \uparrow \) compliance of lung \( \Rightarrow \downarrow \) WOB to expand it with each breath
    - \( \uparrow \) stability of alveoli: ie \( \downarrow \) alveolocasis
      - tendency for small bubble to collapse, and air forced into bigger bubbles \( \Rightarrow \) explode
        \( \downarrow \) pressure inside small bubble \( \uparrow \) large bubble \( (P = 4T/r) \)
        \( \downarrow \) but counteracted by surfactant which \( \Rightarrow \) T is \( \downarrow \) ed in small radius
        \( \downarrow \) \( \downarrow \) tendency for small bubble empty into big bubble
    - prevent pulmon oedema:
      - surface tension draws fluid into alveolar from pulmon interstitium (\& \( \Rightarrow \) capillaries)
      - \( \Rightarrow \) surfactant \( \Rightarrow \downarrow \) surface tension \( \Rightarrow \downarrow \) transudation of fluid
        \( \downarrow \) movement fluid balanced by oncotite pressure of plasma proteins & pressure difference between plasma and alveolar pressure
        \( \downarrow \) surfactant helps lower the alveolar lining pressure \( \Rightarrow \) \( \downarrow \) ed movement fluid
        \( \downarrow \) infant resp distress = stiff lung, alveolocasis and pulmon oedema

**Stability of Alveoli**
- all alveoli surrounded by other alveoli
- many interconnecting links
- \( \Rightarrow \) tendency for change of volume in 1 group of units is opposed by rest of units
  \( \downarrow \) eg alveolocasis in 1 unit, surrounding units will have to expand to fill space \( \Rightarrow \) expanding force on collapsed unit
  \( \downarrow \) called inter-dependance
Elastic Properties of Lung Pressure Volume Curve

- Summary:
  - Non linear, stiffer at high volume
  - Hysteresis between inflation & deflation
  - Compliance = slope volume/pressure
  - Behaviour depends on structural proteins (collagen, elastin) & surfactant

- pressure causing different volumes of lung are different in inspiration & expiration
  - called hysteresis ie need higher pressure for a given volume in inspiration vs expiration

- lungs do not expand in linear fashion with ↓ing IPP: see a sigmoid relationship

- lung volume at any pressure on deflation (expiration) is larger than during inflation

- lung without expanding pressure (ie IPP=0) also has some volume:
  - due to air trapping in small airways
  - because TPP = 0 so no forces acting to keep them open
  - lung volume at which this small airway closure 1st occurs = closing capacity (RV+CV)
  - this airway closing occurs at higher volumes with ↑age and disease

- shape of curve important:
  - at higher IPP’s (ie less –ve) (base):
    - lung relatively compressed – less expanding pressure
    - BUT steep part of curve ∴ any ↓ in IPP ⇒ big ↑ volume
    - = better ventilation
    - as ventilation = change in volume/unit resting volume
  - at lower IPPs (ie more –ve) (apex):
    - lung already relatively expanded
    - incr in vent with lowering IPPs will be relatively less
    - → poor ventilation

Hysteresis

- = effect lags behind the cause
- in lung = higher expanding pressures are needed for a given lung volume during inspiration compared to expiration
  - aka lung stiffer during inspiration

- complex reasons:
  - elastic hysteresis:
    - common to all elastic bodies when stretched
    - elastic recoil on shortening is always less than pressure needed to stretch
    - manifestation of energy loss
  - redistribution of aiw between different alveoli (see later ‘time constraints’)
  - ?changes in surfactant activity with changes lung volume
  - ?changes pulmon blood volume
Compliance

- compliance =
  - slope of pressure-volume curve (volume y axis: pressure x axis) OR

\[
\frac{\Delta V(\text{volume})}{\Delta P(\text{pressure})}
\]

- lung compliance = 200mls/cmH20 (eg steep part of curve)
- in norm range -5 to -10cmH20 lung is very compliant
  - higher range becomes less compliant hence plateau of curve
- main determining factors:
  - tissue elastic forces
  - surface tension – most important
  - surfactant plays a big role – see diagram on effect of saline on V/P curve
- inverse of compliance = elastance
- independent factors influencing compliance:
  - Lung volume: Cx is directly proportional to lung volume.
  - Posture: Lung volume, and thus Cx, changes with posture
    - erect = larger c/f supine
    - But no effect on specific Cx.
  - Pulmonary blood volume: ↑ pulmonary blood volume increases the “stiffness” of the lung by engorgement
  - Age: Strangely, no correlation has been found b/w age and Cx, even after allowing for changes in lung volume.
    - ↓ lung elasticity = mainly determined by surface tension and not elastic tissue
  - Restriction of chest expansion: This reduces both lung volume and lung Cx
    - normal lung Cx can be restored by taking a single deep breath
  - Recent ventilatory history:
    - period of hypoventilation without periodic deep breaths → ↓ in Cx.
    - Normal Cx can usually be restored by 1 or more deep breaths. (eg recruitment manoeuvres with IPPV in ICU)
    - Especially true for diseased lungs.
    - Little evidence for use of “sighs” in ventilated normal lungs (eg during anaesthesia)
  - Bronchial smooth muscle tone; Bronchoconstriction reduces dynamic Cx (later), but unlikely static Cx.
  - Disease: eg
    - ↓ with oedema, pneumonia, fibrosis
    - ↑ with emphysema, asthma attack (↑ed volumes)

- overall compliance depends on overall volume of lung (eg tiny person vs giant)
- use specific compliance =
  - compliance/FRC (or unit volume of lung)
  - this allows intrinsic measurement of elastic tissue properties
  - constant for sexes and ages down to neonate

Dynamic vs Static Compliance

- due to effect of hysteresis: unequal time constraints
- if lung is slowly inflated to point then left at that volume:
  - pressure in lung will drop exponentially to 70-80% initial value
  - most change completed at ~1min
    - = lung becoming more compliant over time ie same volume, lower pressure
- static compliance = compliance calculated from final pressure
• dynamic compliance = compliance calculated from using initial higher pressure
• practical testing:
  o static compliance:
    ▪ pt inspires sequentially to different volumes then relaxes against closed airway for as long as possible ie time for equilibrium
  o dynamic:
    ▪ measured during norm breathing
    ▪ volume & pressure values at end-exp & end-insp points are used → times of no flow at mouth
• static lung Cx always exceeds dynamic Cx ie less pressure for given volume
• in clinical practise we use dynamic compliance

### Cause of Regional Differences in Ventilation

• lower regions of lung ventilate more than upper zones
• intrapleural pressure is higher (less –ve) in bottom zones of lung
  ❯ due to weight of lung ⇒ ↑pressure at base of lung ie higher IPP (less –ve)
• diagram on next page:
  o gas in lung at atmospheric pressure
  o lung easier to inflate at low volumes as more compliant (surfactant)
  o bottom of lung:
    ▪ expanding pressure is small ∴ small resting volume
    ▪ big expansion on inspiration
  o apex of lung:
    ▪ large expanding pressure ∴ big resting volume
    ▪ small change in volume on inspiration
  ❯ all explained by shape of compliance curve!
• ventilation = change in volume/unit of resting volume
  → ∴ bottom lung ↑ed change in volume & smaller resting volume ⇒ ↑ventilation

![Diagram of lung volume and intrapleural pressure](image)

• NB: at low lung volume ventilation characteristics are inverted:
  o IPP in base of lung becomes +ve (above atmospheric)
  o base of lung being compressed, not expanded
  o ∴ vent not possible until intrapleural pressure falls below atmospheric
  o contrast upper lung now on steep part of curve – good ventilation
    ❯ apex good vent, base very poor
• ∴ regional changes in ventilation depends on lung volume:
  o normal volumes: base>apex
  o low lung volumes: apex>base

**Airway closure**
• base does not lose all of gas
• resp bronchioles close first ⇒ peripheral gas trapping
• this airway closing occurs only at v low volumes in young normal lungs
  ↓in elderly may occur at higher volumes ± at functional residual capacity:
    ▪ loss of elastic recoil in lung ⇒ higher (less –ve) intrapleural pressure ⇒ bottom of curve above atmospheric pressure

**Elastic Properties of Chest Wall**
• thoracic cage=
  o ribcage
  o diaphragm
• ∴ elastic properties of both lung & chest wall determine their combined volume
• pneumothorax allows lung to collapse and thorax to spring out
• balancing pressures thorax vs lung
• relaxation pressure = airway pressures when subject inspires/expires and relaxes respiratory muscles:
  o FRC =
    ▪ Equilibrium: volume where elastic recoil of lung = tendency for chest wall to spring out
      ↓: here lung +ve pressure & chest wall –ve pressure but balance each other
  o Volume higher than FRC = +ve airway pressure
  o Volume lower = -ve airway pressure
• lung retracts at all volumes above minimal volume
• chest wall wants to expand at volumes up to 75% of vital capacity (see next graph)

• compliance for thoracic cage=
  o ∆ lung volume/∆unit change pressure gradient between IPP & atmosphere
• factors influencing Cx of thoracic cage:
  o anatomical –
    ▪ ribs
    ▪ state of ossification of costal cartilages
  o obesity
  o skin conditions esp scarring from burns
  o posture:
    ▪ supine ⇒ ↓Cx
    ▪ sitting ⇒ ↑Cx by 30% compared to supine
    ▪ prone ⇒ total static Cx ↓ed by 60%
Combined Pressure-Volume relationships of Lung & Thoracic Cage

- compliance of resp system of a whole depends on:
  - pulmonary compliance
  - thoracic wall compliance
- Cx of lungs & thoracic cage are in series

\[
\frac{1}{C_{\text{Total}}} = \frac{1}{C_{\text{Lung}}} + \frac{1}{C_{\text{Thoracic cage}}}
\]

\[
C_{\text{Lung}} = 200 \text{ml/cmH2O}
\]
\[
C_{\text{Thoracic cage}} \text{ is the same } = 200 \text{ml/cmH2O}
\]

Thus \[
\frac{1}{C_{\text{Total}}} = \frac{1}{0.01} = 100 \text{ml/cmH2O}
\]

Instead of Cx can look at its reciprocal ie elastance

\[
E_{\text{Total}} = E_{\text{Lung}} + E_{\text{Thoracic cage}}
\]

Relationship between alveolar, IPP, ambient pressures

\[
\text{alveolar pressure} - \text{ambient pressure} = (\text{alveolar P} - \text{IPP}) + (\text{IPP} - \text{ambient P})
\]

\[\downarrow\] this holds at all time and is indep of whether pt spont vent or IPPV

@ FRC: \((\text{alv} - \text{IPP}) + (\text{IPP} - \text{ambient}) = 0\)
Impedance to Movement (Ventilation)

- due to:
  - elastic resistance (60% of work) from:
    - lung tissue & chest wall = 30%
    - surface tension at alveolar gas liquid interface = 70%
  - non-elastic resistance (40% of work) from:
    - frictional resistance to gas flow thru airways
    - frictional resistance from deformation of thoracic tissues (viscoelastic tissue resistance)
      - inertia assoc with movement of gas & tissue

Pressures During Breathing Cycle

Before Inspiration/End Expiration

- IPP = -5 (elastic recoil of lung)
- alveolar pressure = 0 (atmospheric)
- volume of gas in lungs = FRC
- no gas flow

Inspiration

- intrapleural pressure = ↓s to -8
  - as lung increases elastic recoil increases
  - ↑tissue resistance
- alveolar pressure = -1 (subatmospheric – drives flow)
  - may be much more negative in airway obstruction
- lung volume = Vt
- mms:
  - diaphragm – mainly
  - external intercostals
  - accessory mm’s
- @end inspiration:
  - IPP = -8
  - Alveolar pressure again 0
  - Gas flow ceases

Expiration

- intrapleural pressure = ↑s: -8 to -5
  - if more +ve then due to airway resistance
  - because alveolar pressure is positive ie flow out
- alveolar pressure = +1 ⇒ movement air out of airways (supra-atmospheric)
  - forced expiration ⇒ intrapleural pressure >0

- alveolar tracing would = flow if airway resistant was constant
- intrapleural pressure would = volume if compliance constant
Airway Resistance

Ohm’s Law

Airways resistance (R) = ΔP / flow

Where ΔP = alveolar - mouth pressures

- for measurement:
  - alveolar P – deduced from body plethysmograph
  - mouth P – measure from manometer
  - flow rate – from pneumotachograph

- airways resistance to flow = low (~2cmH2O/l/sec)
  → airflow of norm Vt only requires ΔP of 1cmH2O between alveoli & mouth

Poiseuille Law

(Used in laminar flow)

\[ R_{\text{airway}} = \frac{8\eta l}{\pi r^4} \]

- R = radius
- N = viscosity
- L = length

- double length ⇒ double resistance
- half radius ⇒ x16 ↑ resistance
- viscosity & not density effects pressure-flow relationship
Resistance in Turbulence

- use different formula:

\[ \frac{dl}{\pi r^4} \]

\( d \) = density

\( l \) = length

Chief Site of Airway resistance

- unlike Poiseuille's law suggests most resistance from medium sized bronchi (7th generation)
- should be smallest narrow airways
- reason being so many small airways \( \Rightarrow \) combined surface area

- adults:
  - nose 62%
  - URT 34%
  - LRT 4%

- Neonates:
  - Nose 28%
  - URT 46%
  - LRT 26%

Factors Determining Airway Resistance

- 5 factors:
  - lung volume
  - Bronchial smooth mm
  - Viscosity & Density of Inspired gas
  - Type of Flow
  - Dynamic Compression of airways

1. Lung Volume

- volume has impt effect on resistance:
  - lung expansion pulls bronchi open by radial traction of surrounding lung tissue
    - similar to extra-alveolar blood vessels
  - @ low volumes –
    - airways may close completely.
    - Esp in dependant areas where lung less expanded (higher IPP ie less –ve)
    - pts with ↑airway resistance breathe at high volumes to ↓resistance

  nb below FRC: airway resistance ↑s dramatically
  (\( \downarrow \) contrast to pulmon vascular resistance (PVR) which is minimum at FRC)

2. bronchial smooth mm:
• contraction ⇒ narrow airway ⇒ ↑resistance
  ⇨ radius to the power of 4 (Poiseuille Law)

• bronchoconstriction:
  o reflex stimulation of contraction via receptors in large airways eg smoke irritant, ETT
  o autonomic control:
    ▪ vagus nerve innervation, Ach ⇒ constriction
  o PCO2 level – direct action on smooth mm:
    ▪ ↓PCO2 in alveolar gas ⇒ constriction ⇒ ↑resistance
  o histamine release ⇒ constriction of smooth mm in alveolar ducts

• bronchodilation:
  o adrenergic receptors ⇒ dilation
    ▪ β1 - heart
    ▪ β2 – bronchi, blood vessels and uterus
      ⇨ adrenaline, salbutamol
  o anticholinergics
  o methylxantines
  o ↑PA\textsubscript{CO2} ⇒ ↑dilation
  o others: Mg, ketamine

3. Viscosity & density of Inspired Gas
• ↑density & ↑viscosity both ⇒ ↑resistance
• density more important in turbulent flow : density is more imp than viscosity in determining airway resistance because flow in medium size bronchi is mostly turbulent
  <=> = area of maximal resistance

(see Reyonlnds calculation below)

4. Type of Flow
• laminar vs turbulent flow
• laminar flow obeys Poiseuille Law:
  \[ R = \frac{8nl}{\pi r^4} \]

• Turbulent flow:
  o Resistance proportional to 1/r^5 & proportional to pressure
• :. resistance to airflow: turbulent flow > laminar flow

5. Dynamic Compression of the Airways
• Driving pressure = \text{Alveolar pressure} – P mouth
  \text{Airway resistance}

• inspiration airflow :
  \text{airflow} = \text{alveolar P} – \text{mouth P}
  = usually ~1 cmH20

• forced inspiration is not effort independent as IPP is –ve :. holds airway open
• diff situation on expiration:
  \text{airflow} = \text{alveolar P} – \text{mouth P (or IPP)}

• forced expiration creates a Starling Resistor:
  o ↑in IPP is transmitted to alveolus BUT also to the airway lumen
  o :. alveolar P then > mouth P so if airway open gas ⇒ flow out of lung down gradient
• effort independent part of exp loop is due to dynamic compression of airways

**Mechanism of Dynamic Compression**

• airway lumen as well as alveoli are exposed to ↑ed IPP
• during forced expiration see ↑ed forces on alveoli & airways
• initially airways held open because airway P > IPP
• BUT:
  o See a pressure drop along airway – due to airway resistance (ΔP = Q. R)
  o ∴ will see a point where ↑ed IPP = airway P
  o this point = equal pressure point
  o distal to this = collapse will occur
  o this = starling resistor & driving pressure for flow =

\[ ΔP = \text{alveolar pressure} - \text{intrapleural pressure} \quad \text{[forced expiration]} \]

\[ (ΔP = \text{alveolar pressure} - \text{mouth pressure} \quad \text{[normal tidal breathing]}) \]

• ΔP remains same for a given lung volume despite any change in exp effort
• ΔP can be effected by diff lung volumes eg ↓ed with ↓ed lung volumes
• equal pressure point can be moved away from alveolus by pursed lip breathing (self PEEP)
Equal Pressure Point
• as lung volume ↓s with expiration:
  o point of collapse moves from large airways ⇒ deeper in lungs
  o due to ↓ing recoil pressure as lung volume falls
    ↓ing recoil pressure ⇒ ↓alveolar pressure ⇒ quicker airway pressure = IPP ⇒ quicker EPP ⇒ quicker onset of dynamic airways compression

From above diagrams:
• IPP = 20
• Alveolar pressure:
  o IPP + recoil pressure of lung (P_a)
    ↑ higher the lung volume ⇒ higher initial recoil pressure
• Downstream pressure:
  o ILP ↓s further from alveolar due to airway resistance ⇒ ↓flow
    ↓ with ↓initial lung ⇒ ↓recoil pressure ⇒ ↓initial alveolar pressure

Expiring from given volume:
• Max flow attained will depend on effort of expiration
• Flow then follows effort-independent part
With smaller lung volumes equal pressure point moves distal (towards alveoli) because of less alveoli pressure initially translating to ↓ILP. Once ILP < IPP then collapse ➔ this occurs dynamically through expiration of one breath

- With smaller lung volumes, the *recoil pressure* of the lungs gets smaller.

- Dynamic compression is exaggerated in lung disease (emphysema) by ↓elastic recoil ➔ ↓alveolar pressure ➔ ↓ILP ➔ more distal equal pressure point ➔ earlier airway collapse

![Diagram showing normal lung and emphysema](image)

**Gas Flow in Lungs**

- Flow in lungs =
  - Laminar
  - Transitional
  - Turbulent

**Laminar Flow**

![Diagram showing laminar flow](image)

*(Q proportional to Δpressure)*

- Occurs in smooth walled vessels at low flow rates
- Flow rate (Q) has a velocity profile being centre of lamina x2 of outside
- Flow rate is directly proportional to pressure change:
• Ohms Law:

Airways resistance \( R \) = \( \Delta P / \text{flow} \)

Where \( \Delta P \) = alveolar - mouth pressures

• Use Poiseuille Equation to quantify other factors:

\[
R_{\text{airway}} = \frac{8nl}{\pi r^4}
\]

\( R = \text{radius} \)
\( N = \text{viscosity} \)
\( L = \text{length} \)

• double length \( \Rightarrow \) double resistance
• half radius \( \Rightarrow \) x16 \( \uparrow \) resistance
• NB viscosity (\& not density) defined in this equation \& thus density does not effect pressure/flow relationship

\( \Downarrow \) density is incl in Reynolds Number \( : \) does effect chance of turbulent flow

**Transitional Flow**

\[
\rho = K_d V + K_t V^2
\]

- Branches cause localised disorganised eddies

**Turbulent Flow**

\[
\rho = KV^2
\]

- @higher flow rates
- pronounced mixing of gas

• Driving pressure is not proportional to flow rate, but to its square:

\[
\Delta P = KQ^2
\]

rearranged: \( Q \propto \sqrt{\Delta P} \)

• For a given \( Q \): work done by resp mms is greater for turbulent:laminar flow
  \( \Downarrow \) work prop to \( \Delta P \)
  \( \Downarrow \) \( \Delta P \) prop to \( \sqrt{Q} \)
• Required \( \Delta P \) is prop to density of gas
  \( \Downarrow \) contrast to laminar flow where viscosity is instead important
• Required driving pressure is inverse prop to the 5\(^{th}\) power of the radius of the tube

**Reynold Number**

• determines chance of turbulent flow
• Velocity is greatest in center of stream – x2 faster than average

\( \Downarrow \) see velocity profile
• Laminar flow occurs up to critical velocity ⇒ turbulent flow
• In turbulent flow ↑ in viscosity is less imp: ↑ density ⇒ ↑ pressure drop for a given flow
• Reynold number:

\[ Ra = \frac{2rvd}{n} \]

\( D = \) density
\( V = \) average velocity
\( R = \) radius
\( N = \) viscosity

• ↑ Probability of turbulence related to
  o ↑ velocity
  o ↑ radius
  o ↑ density = eg helium is low density gas ∴ less turbulence
• Re number = probability of turbulence
  \( \leq 2000 \) = no turbulence
  \( > 3000 \) = nearly always turbulent
• In bronchial tree entrance conditions of tube critical – turbulence upstream may radiate downstream
  o ∴ flow mostly transitional – little eddies
**Actual pattern of Gas Flow in Lungs**
• lungs – series of branching tubes
• laminar flow only prob occur in very small conducting airways where Re low
• Flow is:
  o turbulent in trachea & larynx – high velocities
  o transitional in most bronchial tree
Causes of Uneven Ventilation

1. Effect of gravity – discussed under IPP differences
2. Differing Time Constraints of Lungs

- different resistances & compliances of lung units can ⇒ regional diff in vent

  - cos effect of resistances & alveolar compliance have local effect on rate of air movement
  - airway with ↑resistance ⇒ slow air movement in/out
  - airway with ↓Cx ⇒ air flow into that unit will cease sooner (compared to norm unit)

  - reach max volume quickly as not stretchy

- Divide alveoli into fast & stiff:
  - Fast =
    - low resistance;
    - low compliance (stiff)
  - Slow =
    - High resistance
    - High compliance

- Rapid insp ⇒ air to fast alveoli
- End insp pause allows redistribution to slow alveoli

- resistance & Cx of lung unit .: affects time-dependant filling (or emptying)

  - this can be expressed by time-constant (τ, or Tau) for that unit:

\[
\tau = \text{Compliance} \times \text{Resistance}
\]

- a time constant =
  - used to describe the rate of change of an exponential process
  - time which the process would have been completed in had initial rate of change continued

  - eg flow of air into lung unit is 95% completed after 3 time constraints

- Calculations:
  - Resistance of normal lung is ~ 2cmH2O/l/sec
  - Cx_total = 100ml/cmH2O
o = giving a time constant of **0.2 sec**.
\[ \therefore \] for normal lung tissue, filling or emptying is 95% complete after \( \sim 0.6 \) sec

- lung units with
  - \( \uparrow \) resistance = longer time constraints (slow fill/empty)
  - \( \downarrow \) Cx = quicker time constraint (quick empty/fill)

- presence in lung of units with different time constraints mean that even when no flow at mouth there may be flow inbetween units inside the lung
  \[ \therefore \] amount of this regional ventilation depends on RR

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**Figure 7.19.** Effects of decreased compliance (B) and increased airway resistance (C) on ventilation of lung units compared with normal (A). In both instances, the inspired volume is abnormally low.

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**Frequency Dependant Compliance**

- lung units with differing time constants mean
  - measured dynamic Cx will \( \downarrow \) as RR \( \uparrow \)
  \[ \therefore \] frequency dependant

- calculated static Cx \( \therefore \) > dynamic Cx
Work of Breathing

- work = pressure x volume

**Inspiration:**
- insp mm’s work against:

<table>
<thead>
<tr>
<th>Type of Work</th>
<th>Contributing Components</th>
<th>% of Total Work Required</th>
</tr>
</thead>
<tbody>
<tr>
<td>elastic (compliance)</td>
<td>lung, chest cage</td>
<td>surface tension (50 - 80%)</td>
</tr>
<tr>
<td>frictional</td>
<td>viscous (20%)</td>
<td>tissue (50%)</td>
</tr>
<tr>
<td>inertia</td>
<td>lung, chest cage, air</td>
<td></td>
</tr>
</tbody>
</table>

- elastic –
  - 60% of work
  - work done is stored as potential energy in expanded elastic tissue
  - area 1 on graph below
- non elastic (frictional) forces
  - 35% of work done
  - made up of:
    - tissue resistance (visco-elastic) (20%)
    - airway resistance (80%)
  - energy dissipated as heat. Cannot be reused
    - hatched area (above) or area 2 (below)
- inertia:
  - lung, chest cage, air
  - 5%

→ these breakdown of forces is altered with changes in volume of breath & frequency:
  - if high airway resistance then ↑RR will be bad (inefficient)
    - ∴ COPD breath deep and slow
  - if ↓total compliance then ↑Vt will be bad
    - ∴ fibrotic lung disease breath shallow & fast

• potential energy created by work against elastic forces ↑s if:
  - ↑Vt
  - low compliance

• wasted energy as heat against frictional forces ↑s if:
  - ↑RR ie faster flow rates
  - ↑airway resistance
By Adam Hollingworth

…would see Rward bulging of hatched area

- total WOB for norm tidal breathing = area 1 & 2

- usually total WOB is low ~ VO2 of resp mm ~3mlO2/min
- causes of ↑WOB:
  - ↑MV
  - lung diseases eg ↑resistance, ↓compliance

Expiration

- Insp mm’s relax
- Elastic tissue return to resting length
- Stored potential energy used to overcome frictional forces (area 3)
  \[ \text{ie tissue} \& \text{ airway resistance} \]
- Normally can be accomplished by energy stored in elastic structures \( \Rightarrow \) passive
- May become active if:
  - ↑ed flow
  - ↑ed airway resistance
- ↑RR \( \Rightarrow \) faster flow rate \( \Rightarrow \) hatched area required ie against frictional forces
- ↑tidal volume \( \Rightarrow \) ↑elastic work area (AECD)
Total WOB
- measure o2 cost of breathing:

\[
\text{efficiency} \% = \frac{\text{useful work}}{\text{total energy expended}} \times 100\%
\]

- efficiency ~ 5-10%
- o2 cost quiet breathing <5% of total resting o2 consumption
- voluntary hypervent ~30%
- obstructive lung disease ⇒ limit exercise

Flow Volume Loops

Flow-volume loops. (A) Normal. Inspiratory limb of loop is symmetric and convex. Expiratory limb is linear. Flow rates at the midpoint of the inspiratory and expiratory capacity are often measured. Maximal inspiratory flow at 50% of forced vital capacity (MIF 50%FVC) is greater than maximal expiratory flow at 50% FVC (MEF 50%FVC) because dynamic compression of the airways occurs during expiration. (B) Obstructive disease (e.g., emphysema, asthma). Although all flow rates are diminished, expiratory prolongation predominates, and MEF < MIF. Peak expiratory flow is sometimes used to estimate degree of airway obstruction but is dependent on patient effort. (C) Restrictive disease (e.g., interstitial lung disease, kyphoscoliosis). The loop is narrowed because of diminished lung volumes, but the shape is generally the same as in non-asthma volume. Flow rates are greater than normal at comparable lung volumes because the increased elastic recoil of lungs holds the airways open. (D) Fixed obstruction of the upper airway (e.g., trachomalacia). The top and bottom of the loops are flattened so that the configuration approaches that of a rectangle. Forced obstruction limits flow equally during inspiration and expiration, and MEF = MIF. (E) Paradoxical intrathoracic obstruction (e.g., unilateral vocal cord paralysis, vocal cord dysfunction). When a single vocal cord is paralyzed, it moves passively with pressure gradients across the glottis. During forced inspiration, it is drawn inward, resulting in a plateau of decreased inspiratory flow. During forced expiration, it is passively blown outward, and expiratory flow is unimpaired. Therefore, MIF 50%FVC < MEF 50%FVC. (F) Variable intrathoracic obstruction (e.g., tracheomalacia). During a forced inspiration, negative pleural pressure holds the “happy” trachea open. With forced expiration, loss of structural support results in tracheal narrowing of the trachea and a plateau of diminished flow. Flow is minimized briefly before aireway compression occurs.