CIRCULATORY AND RESPIRATORY SYSTEM
CIRCULATORY SYSTEM

Interior View of the Heart

- Aorta
- Superior Vena Cava
- Pulmonary Valve
- Pulmonary Artery
- Left Atrium
- Mitral Valve
- Tricuspid Valve
- Aortic Valve
- Left Ventricle
- Orifices of Coronary Arteries
- Right Atrium
- Inferior Vena Cava
- Right Ventricle
- Papillary Muscles
Important features of the Heart

1. SA node
2. AV node
3. Bundle of His
4. Right bundle branch
4. Left bundle branch
5. Purkinje fibers

Superior vena cava
Aorta
Inferior vena cava
Electrical System of the Heart

- Sinoatrial (SA) Node
- Anterior Internodal Tract
- Middle Internodal Tract
- Posterior Internodal Tract
- Atrioventricular (AV) Node
- Bachmann's Bundle
- Left Bundle Branch
- Conduction Pathways
- Right Bundle Branch
ELECTRICAL ACTIVITY OF HEART

• S.A node: In. Of superior venacava with rt. Atrium
• A.V. node: Interatrial septum
• S.A. to AV node: .Anterior tract of Bachman
• .Middle tract of Wenckebach
• Posterior tract of Thorel.
• AV node to Bundle of His
• Bundle of His gives left & Right Bundle Branch.
• Each divides into anterior & posterior fascicle
• They come into contact with purkinje system.
S.A NODE POTENTIAL
ACTION POTENTIAL IN CARDIAC MUSCLE
CARDIAC CYCLE

- Total duration : 0.8 sec
- Systole : 0.27 sec
- Diastole : 0.53 sec

Opening of AV Valves

End of Isovolumetric relaxation

Closure of AV valve

End of diastole
Isovolumetric contraction starts

Semilunar valve opens

End of Isovolumetric contraction

Semilunar valve closes

Isovolumetric relaxation starts

Beginning of diastole.
**JVP**

- **a wave:** distension due to rt. atrial contraction
- **c wave:** due to bulging of tricuspid valve into rt. Atrium during isovolumetric contraction.
- **v wave:** inc. blood in rt. Atrium during ventricular systole when tricuspid valve is closed
- **X descent:** Atrial relaxation & downward displacement of tricuspid valve during ventricular systole
- **Y descent:** Opening of tricuspid valve and rapid inflow of blood into rt. Ventricle.
$JVP$
HEART AS A PUMP

- **Fick’s principle**: Oxygen consumption ml/min / arteriovenous pressure difference
- **Frank starling law**: Force of contraction is directly proportional to the initial length of the muscle fibre.
- **Cardiac output**: output per min: 5L/min.
- **Stroke volume**: amount of blood pumped out per beat: 70ml cardiac output/Heart rate
- **Cardiac index**: Cardiac output/Body surface area : 3.2 L/min/sq.mm
DYNAMICS OF BLOOD FLOW
✓ From high pressure to low pressure
✓ Flow = pressure/Resistance.
✓ Electromagnetic flow meters, Doppler flow meters, plethysmography.
✓ Normally laminar flow with velocity greatest in the centre of stream
✓ After a critical velocity, it becomes turbulent
✓ Reynolds number =
  Diameter x velocity x density / Viscosity
  > 3000 : turbulence.
FORMULAE

- **Velocity** = Flow / Cross sectional area.
- **Poiseuille Hagen Formula:**
  \[
  \text{Flow} = \text{Pressure diff} \times \left( \frac{\pi}{8} \right) \times \left( \frac{\text{radius}^4}{L} \right) \times \left( \frac{1}{\text{Viscosity}} \right)
  \]
- **Resistance** = \(8 \times \text{viscosity} \times \text{length} / \pi \times \text{radius}^4\)
- **Laplace law:** Tension = transmural pressure \(\times\) radius / Wall thickness
- **Pressure** = Tension / radius.
VESSELS

- **ARTERIES**: 3 layers
  - outer adventitia
  - Middle media.
  - Inner intima
- Contain elastic tissue
- Arterioles: Resistance vessels
- Aorta is largest with maximal velocity.
- Wind Kessel effect: recoiling during diastole.
- Nitric oxide causes dilation by smooth muscle relaxation.
**CAPILLARIES:**

- Fenestrations present in GIT/kidney
- Precapillary sphincters present but not innervated (relaxed by metabolites)
- Fenestrations present (very porous in liver)
- Jn. b/w endothelial cells: 10nm
- In liver- extremely porous
- Pericytes present: contractile and regulate flow through endothelium
- Largest cross sectional area
- Amt. of blood at any time = 5%
VEINS:

- Capacitance vessels
- Easily distensible
- Venospasm is more pronounced
- Valves are present which prevent retrograde flow
- Amount of blood at any time: 54%

LYMPHATICS:

- Have valves
- No fenestrations.
- In b/w endothelial cells have no membrane.
- 2 types; Initial & collecting.
**BLOOD PRESSURE**

- It is the lateral pressure exerted by the blood on its walls.
- **Systolic pressure**: 120 mm Hg.
- **Normal Diastolic pressure**: 80 mm Hg.
- **Mean Arterial pressure**: Diastolic pressure + 1/3 pulse pressure.
- **Pulse pressure**: Systolic - Diastolic pressure.
- **Measured by Sphygmomanometer.**
GRAVITY

• Positive G: Force due to acceleration acting in long axis from head to foot
  • Blood towards lower limb
  • Cardiac output decrease
  • Black out of vision

• Negative G: Due to force acting in opposite direction
  • Cardiac output increases
  • Red out occurs.
REFLEXES:

✓ Bainbridge: Infusion of saline increases heart rate.

✓ Bezold Jarisch/coronary chemoreflex:

- Injection of certain chemicals
- Stimulates ‘C’ receptors
- Thro’ vagus
- apnea foll. by rapid breathing, hypotension, bradycardia
Inc ICT

Compresses blood vessels

Hypoxia

Stimulates vasomotor centre

Catecholamines

Vasaconstriction

Inc. BP

Stimulates baroreceptors

Activates vagus

Decreases heart rate

CUSHING’S REFLEX
Control Of Cardiac Function

Medullary cardiovascular centre

IX. afferent
X. afferent
Vagal motor neuron
Sympathetic efferent

Aortic baroreceptor
Acetylcholine
Muscarinic
Acetylcholine transmitter

Sinus node
β₁
Noradrenaline

Adrenaline in blood

Sympathetic stimulation increases contractility, frequency, conduction velocity, and irritability.
BARORECEPTOR REFLEX

Location and innervation of arterial baroreceptors.
THE BARORECEPTOR REFLEX
The Reflex Control of Blood Pressure

Sensory
- Stretch receptors for primary afferents (GVEs)

Interneurons
- Interneurons
- Interneurons

Motor
- Preganglionic sympathetics (GVEs)
- Postganglionic sympathetics (GVEs)
- Postganglionic parasympathetics (GVEs)

Inhibitory on pregangl. sympathetics

Sympathetic activation of arteriolar muscle causes vasoconstriction.
Inhibition of this activation, as here, allows passive vasodilation, allowing more blood to enter the vascular periphery, and therefore a fall in central blood pressure

Parasympathetic activation of the heart causes:
- decline in heart rate
- decline in A-V velocity
- decline in contractility

Result: fall in cardiac output and therefore a fall in central blood pressure

Baseline heart rate
Adjusted heart rate
THE CHEMORECEPTOR REFLEX
Carotid and Aortic Body Chemoreceptors
Control of O2 Levels in the Blood

Sensory
- = Chemo receptors of aortic + carotid bodies + associated primary afferents (GVAs)

interneurons
- = Interneurons

Motor
- Green = Phrenic nerve and thoracic ventral motor neurons (GSEs)

Typical Cross Section of Spinal Cord, Thoracic Level

Ventral motor neurons innervating the internal and external intercostals

Baseline respiration rate

Adjusted Respiration Rate
PULMONARY CIRCULATION:

• Total volume of blood in lungs = (9% blood volume) 450 ml.

• Mainly thro’ pulmonary arteries & veins.

• Pulmonary arteries constricts due to hypoxia.

• Ventilation, Perfusion more in lower lobes

• V/Q more in apex.

• Resistance low, capillary pressure low.

• Upper & middle lobes: pressure in veins < atmosph. pressure causing veins to collapse.
ECG
CHEST LEADS

V1 V2 V3 V4 V5 V6
EINTHOVEN’S TRIANGLE
ECG PAPER

- Grid like boxes
- Small box = 1 sq.mm
- Speed = 25mm/Sec. (1mm = 0.04 sec)
- Vertically measures amplitudes
- 1 mv = 10mm
- Standardisation: Vertical = 10mm;
  Horizontal = 5mm
- Upward deflection: Positive
- Downward deflection: Negative
WAVES

- **P wave**: Atrial depolarisation
- **PR interval**: Beginning of P wave to beginning of QRS complex. Time taken for stimulus to spread thro’ atria and pass thro’ AV junction.
- **QRS**: Ventricular depolarisation
- **ST Segment**: End of QRS to beginning of T. Beginning of ventricular repolarisation
- **J point**: In b/w end of QRS and start of T wave
- **T wave**: Ventricular repolarisation
- **QT interval**: Beginning of QRS to end of T Wave.
**LEADS**

- 6 limb leads + 6 chest leads
- **Bipolar limb leads:**
  - **Lead I** — Rt arm & Lt arm
  - **Lead II** — Rt arm & Lt leg
  - **Lead III** — Lt arm & Lt leg
- **Unipolar limb leads** aVR, aVL, aVF
- **Chest leads:** V1 to V6
NORMAL VALUES

- **P wave**: Height & width < 2.5 mm
- **PR interval**: 0.12 to 0.2 secs
- **Q wave**: Depth not > 1/4 th R wave.
  - Width not > 1 mm
- **R & S wave**: R in V1 < S
  - R in V5 > S
- **QRS complex**: 10 mm height & 0.08 secs.
- **QT interval**: 0.40 sec.
- **ST interval**: 0.32 sec
- **Normal axis**: -30 degree to +110 degree.
HEART RATE

- 1500/ no. of small squares
- 300/ no. of large squares. b/w R-R cycles
- **AXIS**
  1) QRS axis in lead I (positive or negative)
  2) Find out its mean
  3) QRS axis aVF.
  4) Mark these values in hexaxial system and drop perpendicular from these pts.
  5) Draw a line thro pt. of intersection.
RESPIRATORY SYSTEM
LUNG WITH TRACHEA AND BRONCHI
ALVEOLI
GASEOUS EXCHANGE
RESPIRATORY SYSTEM

• \( P = \frac{nRT}{V} \) \( n \) is no of moles

• B/w trachea and alveolar sacs. > 16 generations + 7 = 23

• 3000 million alveoli

• Glands are absent from bronchiole

• Pulmonary artery. > capillaries > vein

• Bronchial artery > capillaries > veins

• Normal intrapleural pressure is -2.5 mm Hg

• During inspiration > -6 mm Hg.
**DIAPHRAGM**

- Costal fibres
- Crural fibres
- Central tendon
- *Muscles of inspiration are diaphragm and external intercostal*
- *Muscles of expiration are internal intercostal*
- Compliance of lungs = $\frac{DV}{DP}$
  - ie change in volume per unit change in airway pressure = $0.2L/cm H2O$
- Both ventilation and perfusion are increased at base of lung
LUNG VOLUMES

- **Tidal volume** - 500 ml.
- **Amount of air that moves into lung with each normal inspiration (or) air that moves out of lung with each expiration**
- **Inspiratory reserve volume** – 3300 ml
- **Amount air inspired with maximum inspiratory effort in excess of tidal volume.**
- **Expiratory reserve volume**-1000ml
- **Amount of air expired with maximum expiratory effort in excess of tidal volume.**
- **Residual volume**- 1200ml
- **Amount of air remaining in lungs even after forced expiration.**
LUNG CAPACITIES.

- **Inspiratory Capacity** - TV+IRV
- **Total amount of air that can be breathed in.**
- **Vital capacity** - TV+IRV+ERV.
- **Maximum amount of air that can be expelled out forcefully after a maximum inspiration.**
- **Functional residual capacity** - ERV+RV
- **Amount of air remaining in the lung after normal expiration.**
- **Total lung capacity** - 6 L
- **Maximum volume to which the lungs can be expanded.**
- **Timed vital capacity** FEV, in 1 sec -83% in 3 sec -97%
- **Pulmonary ventilation/Respiratory Minute volume-6 l**
- Amount of air breathed in and out of lung every minute.
  \[ \text{RMV} = \text{TV} \times \text{XRR} \]
- **Maximal voluntary ventilation : 125-170 l/min.**
- Maximum amount of air breathed in and out of lungs by forceful respiration in one minute.
- **Alveolar ventilation : 4.2 l/min.**
- Amount of air utilized for gaseous exchange in one minute.
- **Total deadspace/physiological dead space = Anatomical dead space + Alveolar dead space**
Spirometry (Breathing recording)

IRV

ERV

Residual volume (RV)

TV = V_t

0 1 s

Time

Functional Residual Capacity (FRC) = RV + ERV

Vital Capacity (VC) = IRV + TV + ERV

Forced Expiratory Volume (FEV₁)

Eccentric axis

Bell and counter weight

Water lock
• **Anatomical dead space**-
  The conducting part where gascous exchange does not take place - 150 ml
  ie., from nose upto terminal bronchiole

• **Alveolar dead space**-
  The alveoli which are non-functioning and those which do not receive adequate blood flow.

• **In normal adult, Physiological dead space** = **Anatomical dead space**
OXYGEN DISSOCIATION CURVE

• The relationship between partial pressure of oxygen and percentage of Hb. saturation

• Sigmoid shaped curve

• Due to interconversion of Hb from low affinity (T state) to high affinity (R State)

• Hb exhibits positive co-operativity

• P50 - partial pressure of O2 at which Hb Saturation is 50%
OXYGEN DISSOCIATION CURVE

![Oxygen Dissociation Curve Diagram]
• **Left shift**
  
  . increased pH
  . decreased pCO2
  . decreased temperature
  . decreased 2, 3 DPG.

• **Right shift**
  
  . decreased pH
  . increased pCO2
  . increased temperature
  . increased 2,3 DPG.

• **Left shift** seen in fetal blood and stored blood due to decreased 2,3 DPG.

• **In exercise Right shift is seen**
• **Haldane effect:** Binding of O2 to Hb reduces affinity for CO2

• **Chloride shift/Hamburger phenomenon**
  - When negatively charged bicarbonate ions move out of RBC into plasma, the negatively charged chloride ions move into RBC.

• **This is mediated by band 3 protein**

• **Bohr effect:**
  - Binding of CO2 decreases affinity of Hb for O2
  - Oxygen dissociation curve to right.
• **Hering Breuer deflation reflex:**

  Lungs greatly deflated

  Via vagus

  Stimulates inspiration.

• **J receptor reflex:**

  Pulmonary edema/embolism

  J receptors stimulated

  (in interstitium closed to capillaries)

  Apnea f/b hyperventilation, bradycardia, hypotension.
**REFLEXES**

Hering Breuer inflation reflex:

1. **Lungs over stretched**
2. **Stretch receptors activated**
3. **Switches off respiratory centre**
4. **Stops further inspiration**

*(Not activated until TV > 1.5 L)*
Heads Paradoxical reflex:

Vigorous inflation of lung

Irritant receptors stimulated (in large airways)

Contraction of diaphragm

Cough; hyperpnea.
CO POISONING

- 210 times more affinity for Hb
- Reacts with Hb to form carboxy Hb and so less saturation with Oxygen.
- O2 dissociation curve to left.
- Normal PO2, Dec.O2 saturation, variable PCO2.
- Cherry red colour of skin.
- Type of anemic hypoxia.
CYANOSIS

• Bluish discolouration of skin due to increased reduced Hb. (>4 gm/dL)

• Absolute quantity is important

• 2 types:

  • **Central cyanosis**
  • **Peripheral cyanosis**

• O2 saturation reduced. . Slowing of blood

• Mucous membrane . Mucus membrane

• involved. spared

• Eg: cardiac diseases eg: Obstruction

• Pulmonary diseases Cold exposure.

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EFFECTS OF HYPOXIA

• Cerebral blood flow increases due to vasodilation
• Cardiac output increases due to vasodilation
• Heart rate increases.
• Sympathetic activity increases due to stimulation of vasomotor centre.
• 2, 3, DPG increases: O2 dissociation curve to right.
SURFACTANT

- Produced by type II pneumocytes
- Secreted by exocytosis

**Composition:** Dipalmitoyl phosphatidyl choline,
Phosphatidyl glycerol, lipids, proteins.

**Functions:**
- Reduces alveolar surface tension.
- Prevent alveolar collapse.
- Deficiency leads to ‘respiratory distress syndrome’ in new born.
# RESPIRATORY FAILURE

<table>
<thead>
<tr>
<th>Type I</th>
<th>Type II</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pa O₂</strong> : Dec</td>
<td><strong>Pa O₂</strong> : Dec</td>
</tr>
<tr>
<td><strong>Pa Co₂</strong> : Dec</td>
<td><strong>Pa Co₂</strong> : Inc.</td>
</tr>
<tr>
<td><strong>PA-a O₂</strong> : Inc.</td>
<td><strong>PA a O₂</strong> : Normal</td>
</tr>
<tr>
<td><strong>Defect in Oxygenation</strong></td>
<td><strong>Defect in ventilation</strong></td>
</tr>
<tr>
<td>eg: Pneumonia ARDS</td>
<td>eg: Asthma</td>
</tr>
<tr>
<td></td>
<td>weakness of resp.muscles.</td>
</tr>
<tr>
<td></td>
<td>chest wall defects.</td>
</tr>
</tbody>
</table>
## HYPOXIA

<table>
<thead>
<tr>
<th>Type</th>
<th>Mechanism</th>
<th>PaO2</th>
<th>O2 content</th>
<th>A-V difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoxic high altitude</td>
<td>Decreased O2 for diffusion</td>
<td>decreased</td>
<td>decreased</td>
<td>normal</td>
</tr>
<tr>
<td>Anemic anemia</td>
<td>Decreased O2 capacity due to dec Hb</td>
<td>normal</td>
<td>decreased</td>
<td>normal</td>
</tr>
<tr>
<td>Ischemic cardiogenic shock</td>
<td>Reduced blood velocity</td>
<td>normal</td>
<td>normal</td>
<td>increased</td>
</tr>
<tr>
<td>Histotoxic cyanide poison</td>
<td>Tissue can’t use O2</td>
<td>normal</td>
<td>normal</td>
<td>decreased</td>
</tr>
</tbody>
</table>