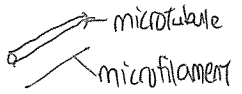
Organelles of A Representative Cell :

Cell Membrane →	lipid bilayer containing phospholipids, steroids, proteins & carbohydrates	Isolation, Protection, sensitivity, support, Controls entry & exit of materials
Cytosol	Fluid Component of cytoplasm	distributes material by diffusion

Non-Membranous Organelles :

Cytoskeleton 	Proteins organized in fine filaments or slender tubes	Strength & support, movement of cellular structures and support
Microvilli	Membrane extension containing microfilaments	Increase surface area to facilitate absorption of extracellular materials
Centrosome & centriole	Cytoplasm containing 2 centrioles at right angles, each centriole is composed of 9 microtubule triplets in a 9+0 array	Essential for movement of chromosomes during cell division, organisation of microtubules in cytoskeleton
Cilia	Membrane extension containing microtubule doublets in a [illegible] array	Movement of materials over cell surface
Ribosomes	RNA + proteins, fixed ribosomes bound to RER, free ribosomes scattered in cytoplasm	Protein Synthesis
Proteasomes	Hollow cylinders of proteolytic enzymes with regulatory proteins at ends	Break down & recycling of damaged or abnormal intracellular proteins

Comparisons

	Sympathetic	Parasympathetic
outflow from CNS	Thoracic } Lumbar	cranial } sacral
Preganglionic fiber	short	long
ganglionic transmitter	ACh (N_2)	ACh (N_2)
Post ganglionic fibre	long	short
Neuroeffector transmitter	NA (α or β1)	ACh (M)

Functional Roles

- Both divisions most organs } tissues
- often have opposing (antagonistic) effect
- Sympathetic more active in stressful situations (flight of fight response) → ↓ metabolism (digestive } urinary systems)
- Parasympathetic more active in vegetative situation (inactive site of cell)
- Sympathetic } Parasympathetic opposes each other
  - ↓ heart rate
  - ↓ [illegible]
  - ↑ metabolism

Hormonal Control

- +ve feedback
- -ve feedback

- often long term
  - ↳ thyroid hormones — metabolism
  - ↳ growth hormone — growth

(-ve) CRH — produced by hypothalamus
(Corticotropin releasing hormone)

↓

ACTH — adrenocorticotropic hormone

↓

Cortisol → -ve activates metabolism

↳ produced by hypothalamus
↳ secreted by posterior pituitary gland

(+ve) Suckling → Oxytocin → milk

↓

causes mammary alveoli to contract, releasing ↑ milk through mammary glands

Connective Tissue
consists of
Support Cells & Extracellular Matrix

Fibroblasts → secrete collagen
Macrophages
Mast cells
Adipocytes (fat cells)

Ground Substance
Extracellular fluid
Collagen fibres
Reticular fibres
Elastic fibres (contain elastin)
} formed by fibroblast as fibroblast secretes its protein subunits

* Reticular fibres contain type 3 collagen
* Mast cells secrete histamine, making capillaries ↑ permeable, heparin prevents blood clot

Addition Extracellular Matrix components

1) Ground substance
- gel-like matrix
- composed of proteins & carbohydrates
- epithelial / connective tissue - derived
- Hyaluronic acid (CH_2O) (carbohydrate)
- laminin & fibronectin (glycoprotein)
  ↳ contains protein & carbohydrate

2) Extracellular Fluid

Specialised Connective tissue
(1) Blood
(2) Bone
(3) cartilage

Adipose Tissue → "white fat"

Loose Connective tissue
- +++ ground substance
- Some fibres
- Some cells
  ⇒ mainly fibroblasts

Dense (connective tissue)
- little ground substance
- densely packed fibres
- arranged in parallel rows
- few cells scattered

→ gives good strength, found in tendons / ligaments

Muscle Tissue → produces movement & specialised for contraction

skeletal, smooth, cardiac

Similarities — (1) elongated parallel to axis of contraction
(2) numerous mitochondria
(3) contractile elements
  ↳ actin & myosin

Lymphatic System:

Functions: (1) Production, maintenance & distribution of lymphocytes that provide defense against infections and other environmental hazards

(2) Excess interstitial fluid returns to bloodstream

(3) Transport of lipids absorbed in digestive tracts via lacteal

*Lymphocytes

Lymphatic Capillaries: [Terminal Lymphatics]

- Lymphatic network begins with lymphatic capillaries
- Differ from blood capillaries
  - i) Originate as blind pockets
  - ii) LARGER in diameter
  - iii) thinner walls
  - iv) have flattened / irregular outline in sectional view
- basement membrane incomplete / absent
- endothelial cells not tightly bound but do overlap
- region of overlap – one way valves
- absent in areas that lack blood supply
- Bone marrow & CNS lack lymphatic capillaries

Major Lymph-collecting Vessels:

(1) Superficial lymphatics – located in subcutaneous layer deep to skin
- areolar tissues of mucous membranes lining digestive, respiratory, urinary & reproductive
- areolar tissues lining of serous membranes lining the pleural, pericardial, peritoneal cavities

(2) Deep lymphatics – larger lymphatic vessels that accompany deep arteries & veins supplying skeletal muscles & other organs

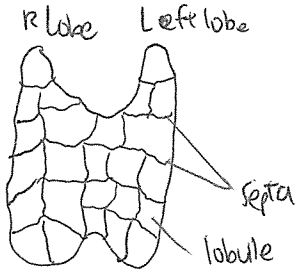
↳ Superficial & deep lymphatics converge to form larger vessels – lymphatic trunks

↳ lymphatic trunks empty into thoracic duct & right lymphatic duct

Thoracic duct
↳ collects lymph from body inferior to diaphragm, left side of body superior to diaphragm

Right lymphatic duct
↳ right side of body inferior superior to diaphragm

Thymus:



- located in mediastinum
- pink and a grainy consistency
- capsule that covers thymus divides it into 2 thymic lobes
- septa [fibrous partitions] divide the lobes into lobules.

- Each lobule consists of densely packed outer cortex, paler central medulla
- lymphocytes in cortex are still dividing, as T cells mature

migrate to medulla → leave thymus by entering one of the medullary blood vessels

- Lymphocytes in ~~cortex~~ Cortex are arranged in clusters that are completely surrounded by reticular epithelial cells
  - maintain blood-thymus barrier
  - secrete thymic hormones

} FUNCTIONS

- Reticular epithelial cells in medulla cluster together in concentric layers forming distinctive structures — Hassall's corpuscles

Hormones of Thymus

- thymosin
  - thymosin-a
  - thymosin-b
  - thymosin-V
  - thymoprotein
  - thymopoietain
  - thymulin

↳ related to lungs

Pulmonary Circulation:

- low pressure system
- takes deoxygenated blood from RHS hearts to lungs
- takes oxygenated blood from lungs to LHS of heart
- pulmonary artery — O_2 poor
- pulmonary vein — O_2 rich

Portal Circulation:

Eg: ~~kyph~~ hypophyseal portal system → hypothalamus to anterior pituitary
↳ direct transfer of hormones
hepatic portal system

Systemic circulation: Vein ····· capillary ——— Artery

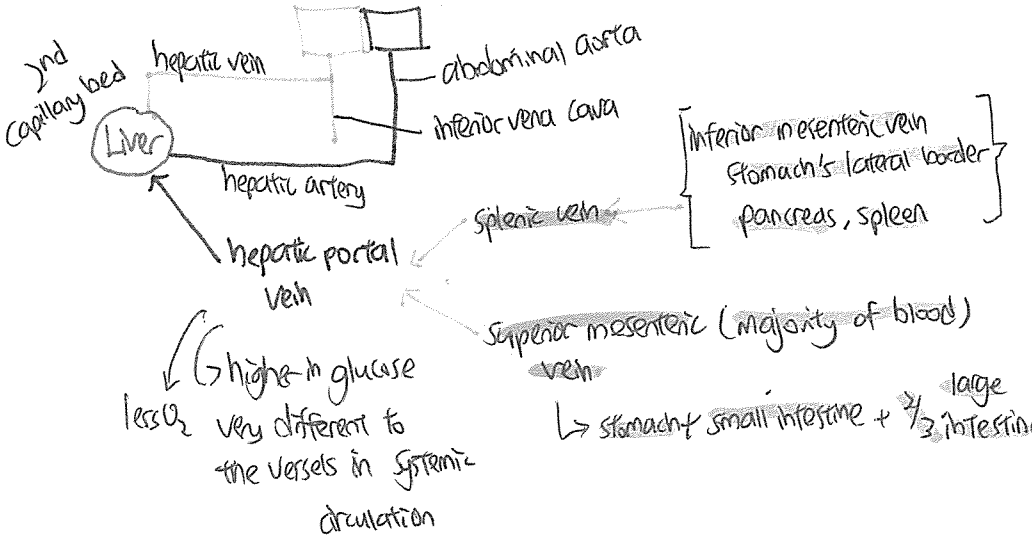
Portal circulation: Vein ——— portal vessel ——— Artery

Strokes

↳ ~~infarction~~ interruptions of vascular supply to a portion of the brain, more commonly
↓
middle cerebral artery

Hepatic Portal System: (~~structure~~ liver)

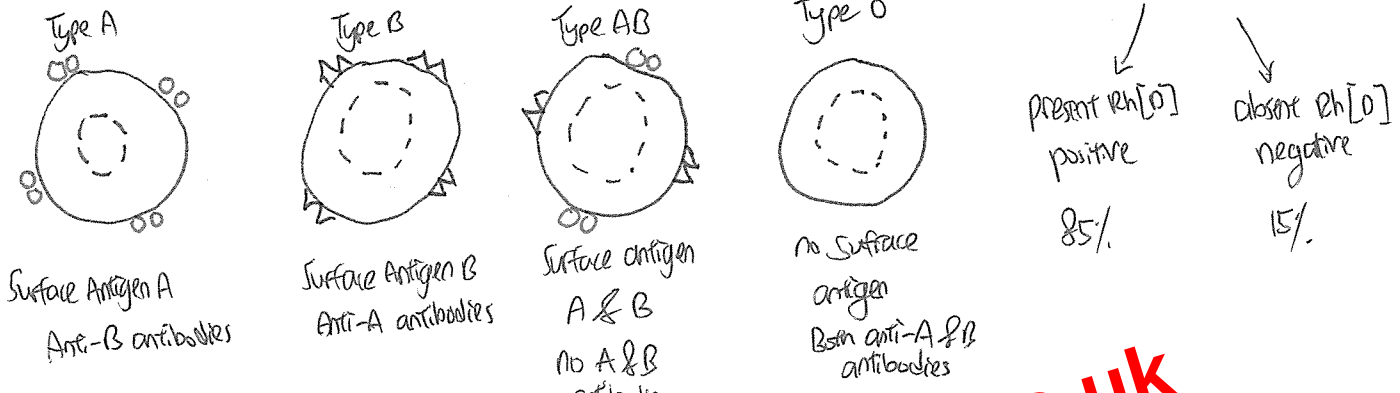
- Storage
- metabolic conversion
- detoxification
- destroys bacteria



Blood type (group)

- 29 blood group systems – ISBT
- genetically determined
- Antigens on red blood cell membrane
- ABO & Rh[D] blood group systems most clinical significance

ABO Blood Group:



Rh[D] antigen

- present Rh[D] positive — 85%
- absent Rh[D] negative — 15%

Surface antigen + opposing antibodies → agglutination + haemolysis

Rh[D] blood group

- Rh negative individual will not naturally contain anti-Rh[D] antibodies [not naturally producing]
- requires sensitisation by exposure to Rh+ erythrocytes
  - ↳ transfusion
  - ↳ Pregnancy / birth – Rh -ve mother & Rh +ve baby
    - haemolytic disease of newborn (HDN)

anti-Rh antibodies are able to cross the placenta due to haemorrhaging during delivery

destroy fetal RBC → dangerous anaemia

- jaundice ← wait treatment ← erythroblastosis fetalis
  - to foetus due to RBC breakdown

First pregnancy
- ↳ also given after miscarriage / abortion

Anti-Rh antibodies

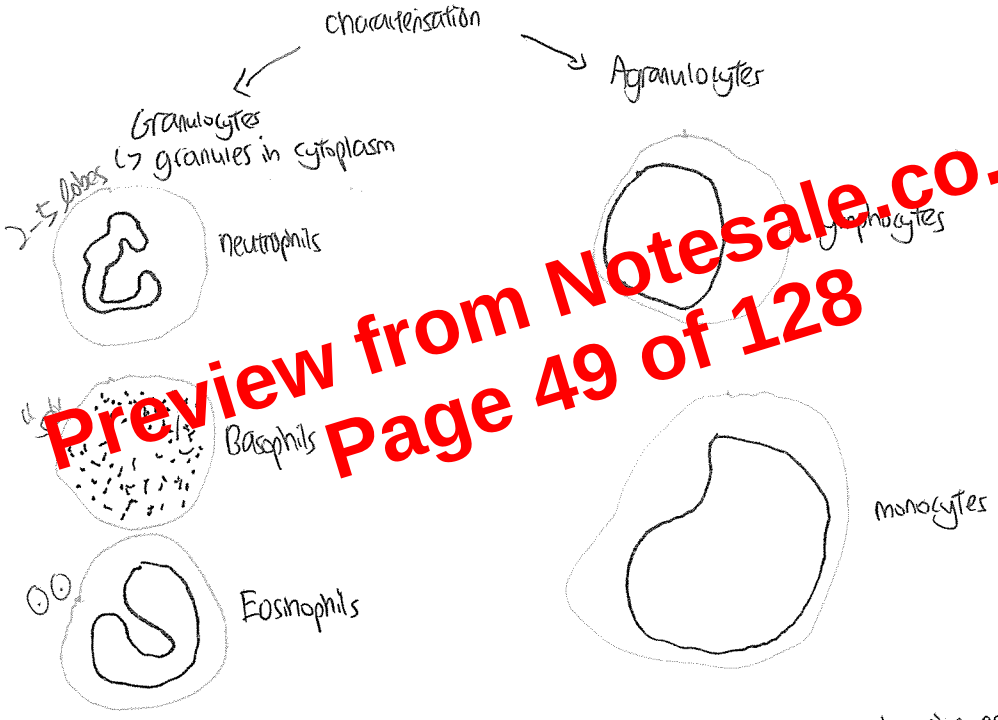
[Rhogam] given to mothers 3 months before & after pregnancy to destroy any fetal RBC in mother. This prevent any sensitization which lead to production of anti-Rhesus antibodies.

Platelets → haemostasis, ~~haemostasis~~, Coagulation : 12-300 per mm^3

Leukocytes : 5-10 thousand per mm^3 → nucleated
- → defense vs pathogens
- → toxin & waste removal
- → remove damaged cells
- → Are mainly outside in tissues – in transit between sites of activity
- → capable of ameboid movement
- → able to migrate out of bloodstream
- → Contact & adhere to vessel walls in the process of marginination

- 60-70% Neutrophils
- 20-25% lymphocytes
- 3-8% Monocytes
- 2-4% Eosinophils
- 0.5-1.0% Basophils

↳ Normal WBC count : $4-11 \times 10^9$ / litre of blood
↳ number of WBC in blood often an indicator of disease



Neutrophils [polymorphonuclear]

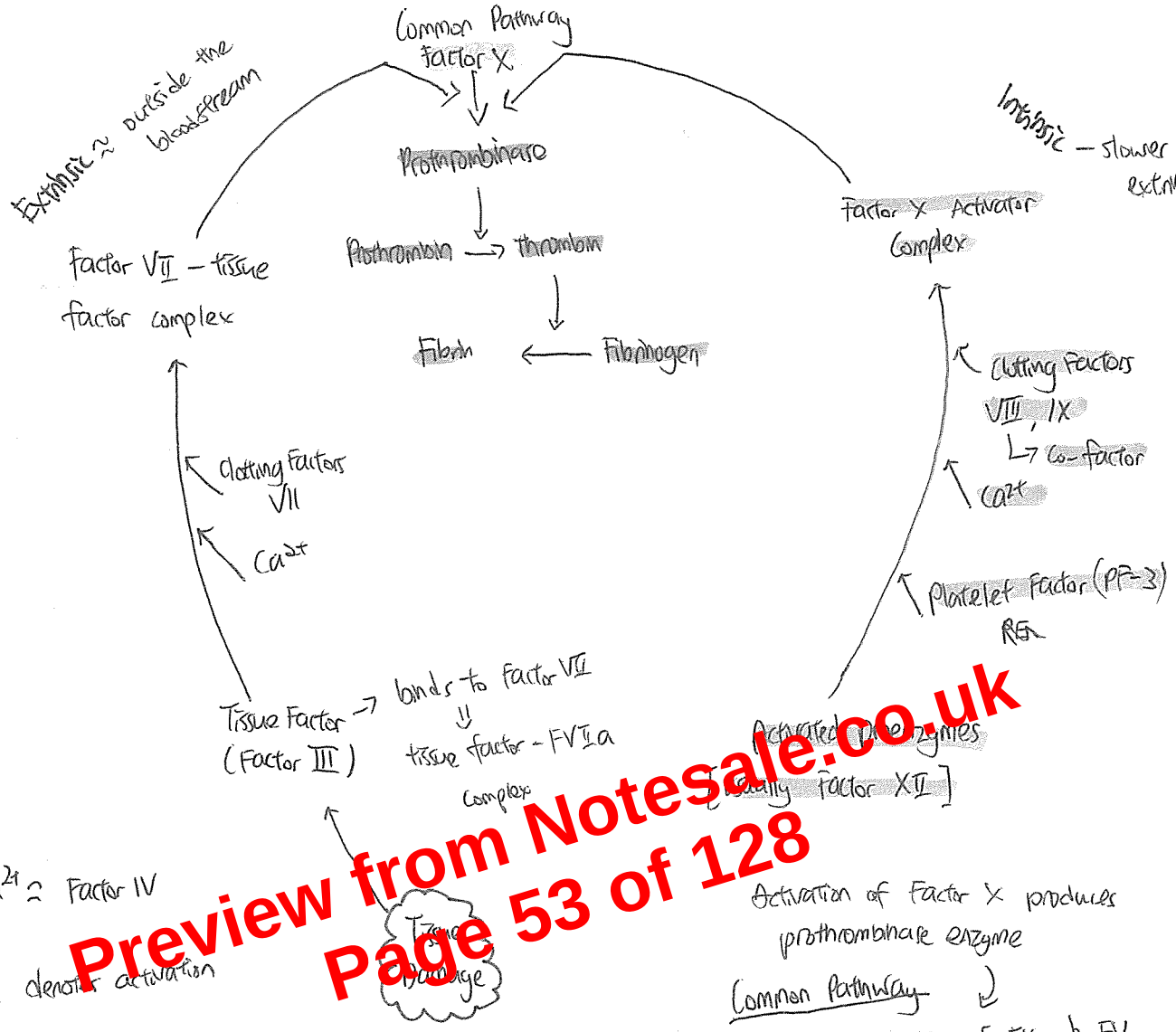
- 50-70% of WBC – most common
- Structure : 9-15 μm in diameter [size of 1 RBC → 2 RBC]
  : distinctive nucleus, 2-5 lobes, granular cytoplasm [↑ lobes, ↑ maturity]
- Function (1) 1st line of defence against bacterial infection
  (2) phagocytic – phagocytosis
  (3) mobile
  (4) circulate in blood for approximately 10 hours
  (5) Major constituent of pus

-lysosomes contain digestive enzymes & small peptides known as defensins
↳ This process reduces no. of granules in cytoplasm [degranulation]

when attacking bacteria, ~~prostal~~ prostaglandins & leukotrienes are secreted
- ↑ capillary permeability → local inflammation
- hormones of immune system that attract other phagocytes

Haemostasis – Coagulation Phase

*Factor X stimulates a small amount of thrombin



Ca^{2+} = Factor IV

a denotes activation

Tissue damage

Activation of Factor X produces prothrombinase enzyme

Common Pathway

1) Prothrombinase : Consists of FXa + FVa as a cofactor
2) Activates prothrombin to form thrombin
3) Thrombin converts fibrinogen to fibrin

Initiation of Coagulation : Tissue Factor

Binds to Factor VII = tissue factor – FVIIa complex

Binds to FX + activates it become FXa

[extrinsic pathway]

able to activate Factor X

Intrinsic Pathway : Factor IX & co-factor VIII

Activates FX - Fxa

More slowly than extrinsic pathway

Extrinsic + Intrinsic → different stimuli

↘ same activation time

Extrinsic ↓ tissue damage

Ca^{2+} + vitamin K

- Affect almost every aspect of clotting process
- Any disorder that decreases $[Ca^{2+}]$ will impair blood clotting
- Adequate vitamin K necessary for production of certain clotting factors in the liver including prothrombin

Inadequate Blood Clotting:

(1) Haemophilia → inherited disorder
↳ lack of clotting factor VIII

(2) von ~~Willebrand~~ Willebrand disease → most common inherited coagulation disorder
von Willebrand factor (vWF) → plasma protein that binds & stabilises factor VIII

Circulation of Blood:

Kidneys → controls $[Na^+]$ & $[K^+]$ determines osmotic pressure, changing viscosity of blood and flow of blood

Cardiovascular System's Components

Brain & Adrenal Glands / Kidneys — neural & hormonal

Heart – cardiac output → arterial blood pressure → peripheral resistance → capillary pressure ↕ interstitial fluid (capillary exchange) ↕

↓ venous pressure

Functions of Cardiovascular System

(1) Maintain blood in order to supply nutrients & signalling molecules to tissues & remove waste products

(2) To maintain pressure differentials across tissues for the purposes of capillary exchange

(3) Provide varying blood supplies to tissues for the purpose of supporting diverse metabolic ranges & functions eg: muscle during exercise, GI tract during digestion

Functions of Blood Vessels:

Arteries – elastic structures – "Pressure reservoirs"

Arterioles – "Resistance vessels"
- control blood flow thru organs / tissues
- control arterial blood pressure

Capillaries – "exchange vessels"

Veins – "Capacitance vessels"
- blood volume reservoir

Haemodynamics – The Physics of Blood Flow

blood flow "F" is determined by 2 factors
- pressure difference
- impedance – vascular resistance

$F = \frac{P}{R} \approx I = \frac{V}{R}$

$F = \frac{\Delta P}{\Delta R}$, where $R = \frac{8L\eta}{\pi r^4}$, where
- L = length of blood vessel
- η = fluid viscosity
- r = radius of vessel

} in accordance to Darcy's Law which describes the flow of a fluid through a medium

$F = \Delta P \cdot \frac{\pi r^4}{8L\eta}$ aka Poiseuille-Hagen equation

$\ldots\ldots\rightarrow F \propto \frac{BP}{PR}$

Fluid viscosity – high protein content, causes more viscous blood, hence ↓ blood flow

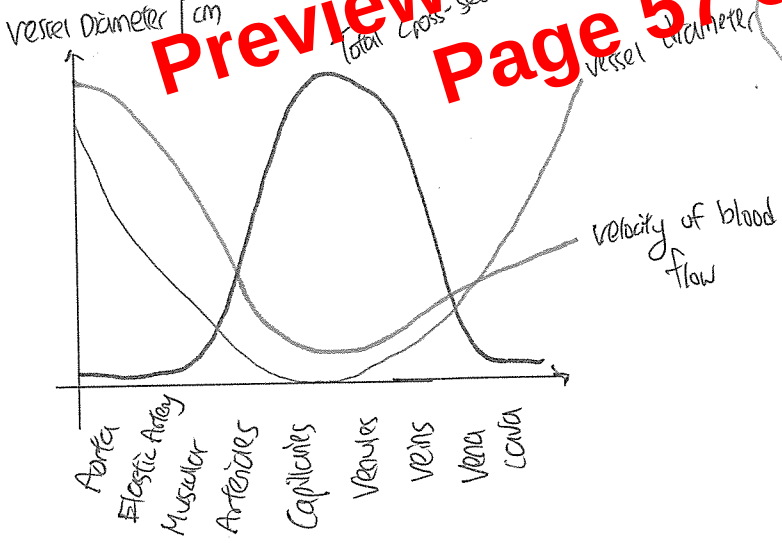
Implications of Poiseuille-Hagen equation

↳ Rate of flow is proportional to fourth power of ~~radius~~ vessel's radius

Thus, small changes in arteriole diameter drastically alter tissue blood flow RESISTANCE !!!

r = 1cm → flow = 1ml/sec

r = 2cm → flow = 16ml/sec



Vascular resistance → resistance in blood vessels

Total peripheral resistance → resistance of entire cardiovascular system

Peripheral resistance → resistance in arterial system

↳ i) vascular resistance [length, radius, viscosity]

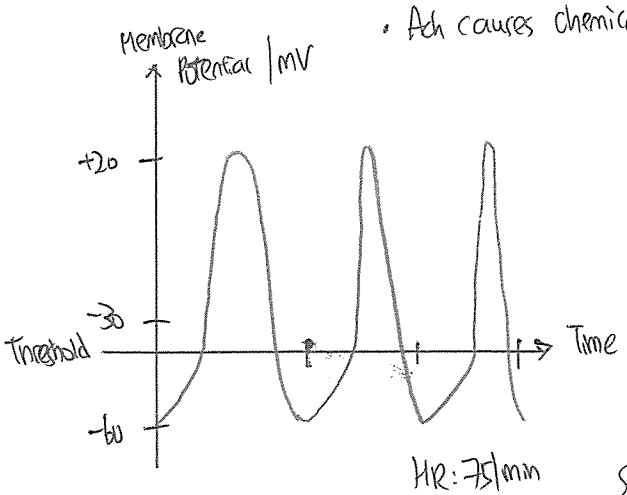
↳ ii) Turbulence: high flow rates, irregular surfaces, sudden changes in vessel diameter upset the smooth flow of blood, creating swirls.
- normally occurs when blood flows between atria & ventricles, ventricles & aortic / pulmonary trunks

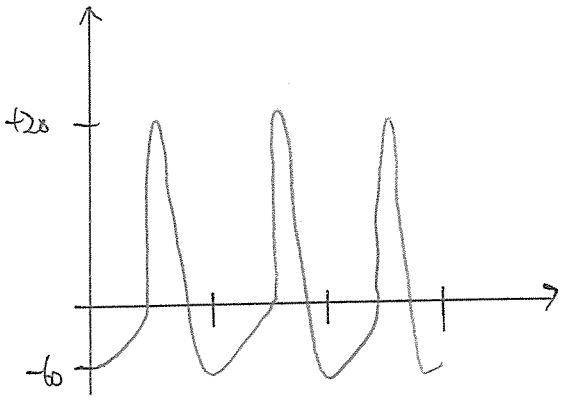
↑ diameter, ↑ blood flow velocity

Hence graph of velocity & diameter has similar shape...

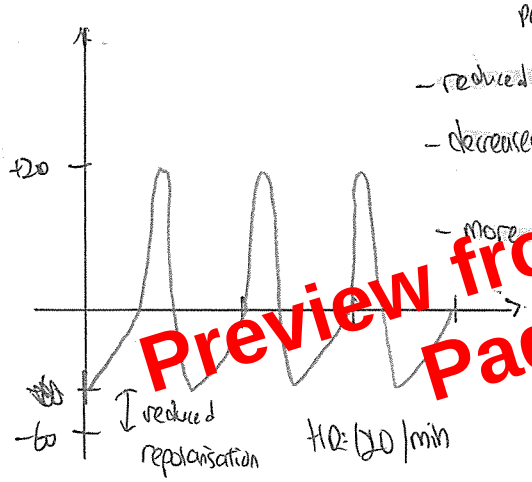
Cardiac output & Control of Heart Rate:

- Acetylcholine (neural: parasympathetic nerves) has a negative chronotropic effect on the heart rate
- Threshold of action potential shifted to more hyperpolarized potentials
- Ach causes hyperpolarization
- (+) of muscarinic-receptor gated K channels
  - ↳ open
- Ach causes chemically regulated K^+ channels to open



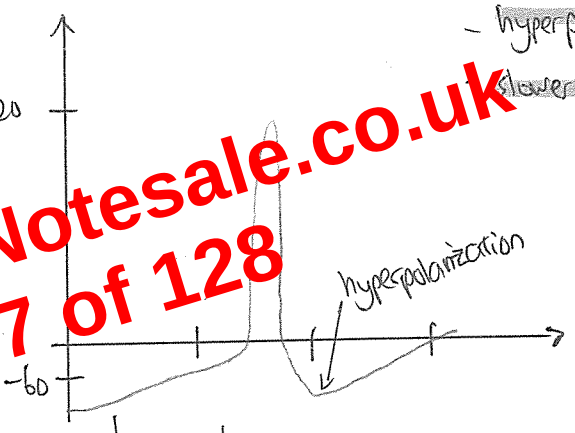


HR: 75/min



HR: 120/min

SAN pacemaker
- reduced repolarisation
- decreased prepotential period
- more rapid depolarisation



- hyperpolarisation
- slower depolarisation

↳ slower depolarisation

Positive Chronotropic Effect

Negative Chronotropic Effect

* slope of pacemaker potential reduced

⇝ At rest, heart rate is 70bpm; very little / NO cardiac sympathetic activity

⇝ At rest, SAN node is considered to be under high vagal tone (vagus nerve) which slows the inherent heart rate of 110bpm to 70bpm.

* vagus nerve not the only mechanism reducing basal heart rate

⇝ Evidence, cuts vagus nerve, HR → 110bpm
  ↳ inhibits the acetylcholine receptor with atropine, HR → 110bpm

⇝ Trained individuals / athletes have lower heart rate → due to ↑ vagal tone, hence heart rate is slowed down more

Contractility : Amount of force produced during a contraction, at a given preload.

↳ can be altered by autonomic innervation / circulating hormones

Factors that increase contractility = positive inotropic action → stimulate entry of Ca^{2+} into cardiac muscle cells → ↑ force & duration of ventricular contractions

.... decrease contractility = negative inotropic action → block Ca^{2+} movement or depress cardiac muscle metabolism

Drugs like { isoprenaline, dopamine, dobutamine } mimic action of noradrenaline & norepinephrine by stimulating beta-1 receptors on cardiac muscle cells

* Dopamine & dobutamine also stimulates Ca^{2+} entry through alpha-1 receptor stimulation

Digitalis ↑ elevate intracellular Ca^{2+} concentrations by interfering the removal Ca^{2+} mechanism from the sarcoplasm of cardiac muscle cells.

Propanolol, Timolol, metroprolol, atenolol, labet... } block beta-receptors, alpha-receptors reflecting sympathetic stimulation of heart

* Ca^{2+} channel blockers like nifedipine & verapamil also have negative inotropic effect.

Afterload : amount of tension the contracting ventricle must produce to force open the semilunar valve & eject blood.

↳ ↑ afterload, ↑ period of isovolumetric contraction, ↓ duration of ventricular ejection, ↑ ESV

↳ ↑ afterload, ↓ stroke volume

→ Afterload is increased by any factor that resists blood flow through arterial system.

Dynamics of Capillary Exchange:

- Since the maximum filtration pressure is greater than the maximal absorption pressure, the transition point is located towards the venous end of the capillary & not in the centre.
- Hence, more filtration takes place along the capillary than absorption

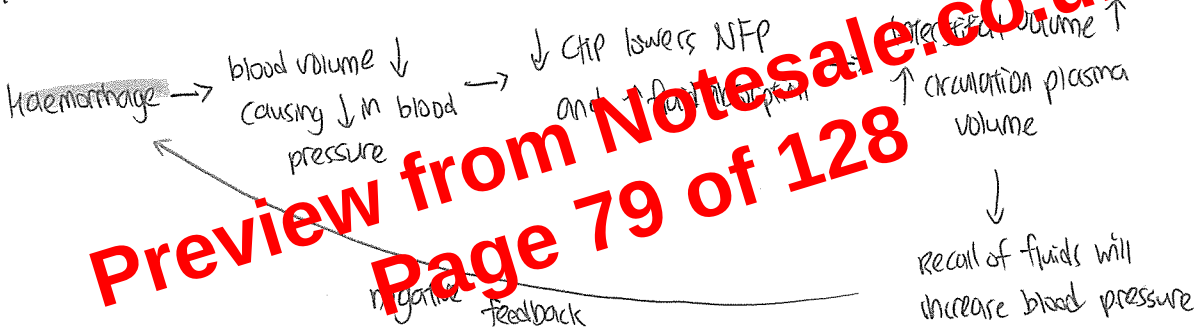
Defects in Capillary Exchange

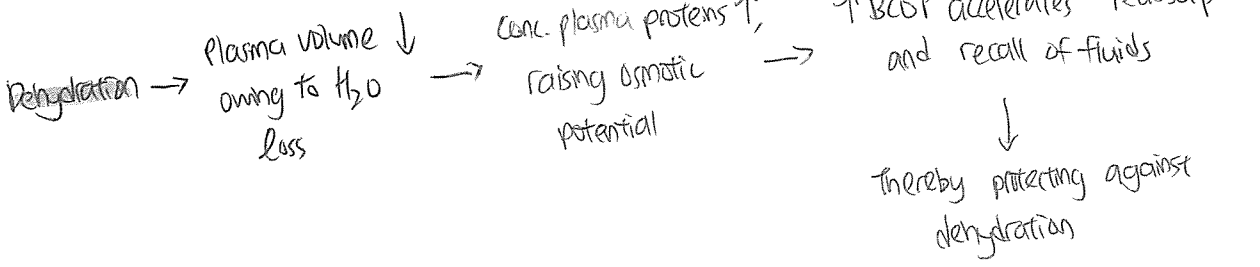
① If capillary hydrostatic pressure ↑ / blood colloid osmotic pressure ↓ fluid moves out of blood and builds up in peripheral tissue
  - ↳ fluid collects in extremities
  - ↳ systemic oedema

Pulmonary oedema: accumulation of fluid in the alveoli in the lungs.

filariasis – parasites block lymphatic vessels causing a massive regional edema – elephantiasis

Capillary Exchange and Fluid Homeostasis:





Treatment of Hypertension:

1) Thiazide Diuretics:
- ↑ Na^+ & H_2O loss, thereby decreasing fluid volume
  ↓venous return, ↓ cardiac output

2) β- adrenoreceptor antagonists · ↓ CO, ↓ SNS activity centrally, ↓ renin release, leading to favourable secondary actions
"β- blockers"

3) Voltage-gated Ca^{2+} channels antagonists: inhibit membrane depolarization
: ↓ CO; cause vasodilation

4) Angiotensin Converting Enzyme (ACE) – inhibits actions of angiotensin II on aldosterone production thereby preventing renal Na^+/H_2O absorption & blood volume increase.
Inhibitors
↳ inhibits vasoconstrictors actions of angiotensin II
↳ vasoconstrictor

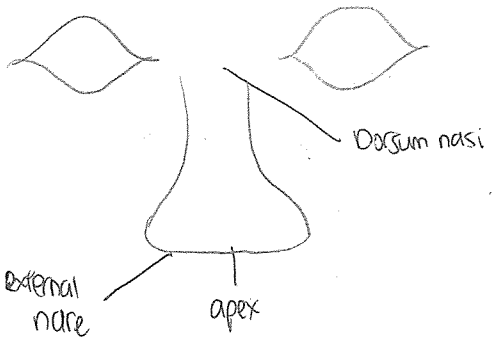
5) Angiotensin II receptor Antagonists: blocks the actions of angiotensin II

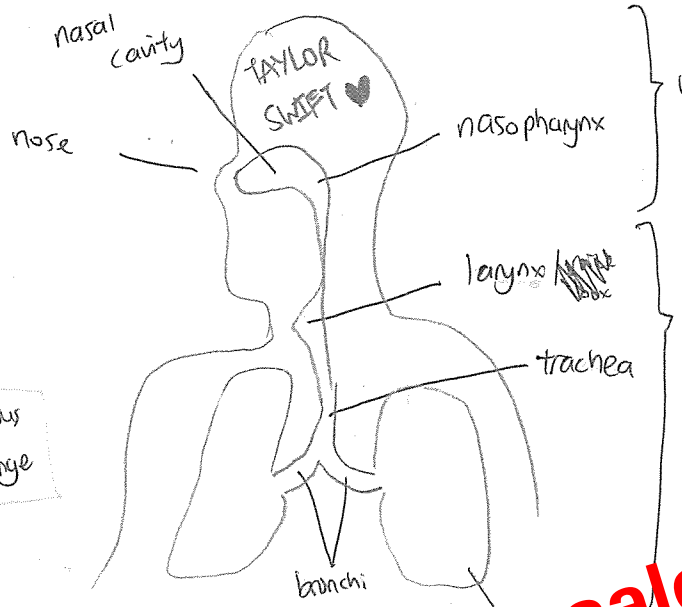
6) α- Adrenoreceptor Antagonists: reduce total peripheral resistance by inhibiting the action of noradrenaline
"α-blockers"

* ACE produced by endothelial cells in blood vessels

Functions of Respiratory System

- Provides O_2 to all tissues of body
- Removes CO_2
- regulates blood pH
- phonation (speech)
- defence against pathogens





upper – pharynx, nasal cavity, larynx

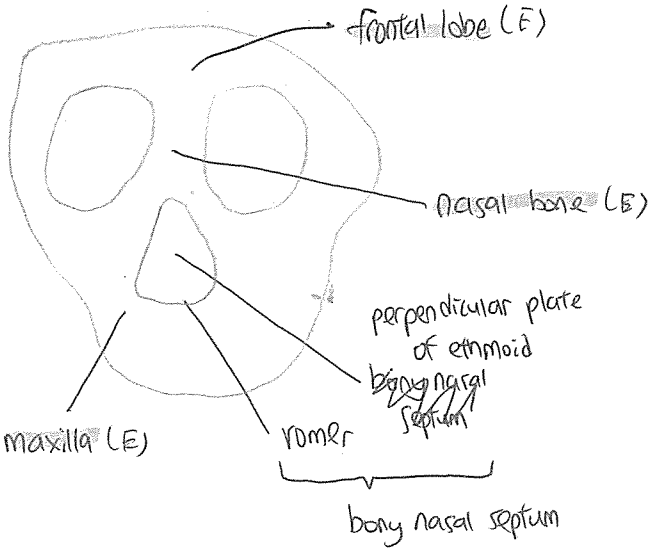
lower – trachea, bronchi, lungs ... etc

Conducting (C)
respiratory (R)
↳ site of gaseous exchange

lung [bronchioles, terminal bronchioles, respiratory bronchioles, alveoli]
(R)

Terminology:

- Pulmonary – lungs reference
- Airways – all the 'tubes' through which air flows between the external environment and the alveoli (lungs)
- Inspiration – movement of air from external environment into the alveoli
- Inspiration + expiration = respiratory cycle



E – external
I – internal

nasal septum separates the nasal cavity
↳ contains cartilage

External nose: contains cartilage + bone
Internal nose: contains bone

Nose
Trachea
↳ 1°/2° Bronchi
} Pseudostratified ciliated columnar epithelium w/ goblet cells

cilia ~> ~~myrtle~~ mucociliary escelator

Bronchioles — simple ciliated columnar epithelium

Terminal bronchioles — simple cuboidal epithelium

Respiratory ~~alveoli~~ bronchioles — simple squamous / cuboidal epithelium
} scattered cilia with no goblet cells

Alveoli — simple squamous epithelium

Tracheal Blockage:

1) Aspiration: breathing in foreign objects that become lodged in larynx / trachea
↳ If individual can still speak, the airway is still open.

Treatment: Heimlich maneuver / abdominal thrust

. Rescuer ~~&~~ applies compression to victims abdomen just inferior to sternum.
This action elevates the diaphragm forcefully & may generate enough pressure to remove the blockage.

2) If blockage results from swelling of epiglottis / tissues surrounding glottis, a professionally qualified rescuer may insert a curved tube through the pharynx & glottis to permit airflow
This is ⟶ intubation

3) If blockage is immovable or larynx has been crushed, a tracheostomy may be performed
In tracheostomy, incision is made through the anterior tracheal wall, a tube is inserted
The tube bypasses larynx & permits air to flow directly into trachea *.

Pneumothorax - air becomes trapped in pleural cavity - causing collapsed lungs / atelectasis due to elastic fibres of lungs recoiling

↳ Most common symptom: sudden stabbing pain in chest made only worse by breathing

. This occurs because surface tension of pleural fluid has been broken ∴ 2 pleural membranes are rubbing against each other without lubrication. This involve loss of -ve intrapleural pressure (close to 760mmHg)

Most common form of pneumothorax ⇝ spontaneous pneumothorax - 1/5000 adults prevalence

↳ ~~occasionally~~ men < 40 years old 4x ↑ risk than women

↳ cigarette smokers ↑ risk

↳ small tear in the lung tissue near the top of the lung allowing air into the pleural cavity

↳ Pneumothorax can occur 2° to existing disease eg: chronic obstructive pulmonary disease, pneumonia or tuberculosis in which the lung tissue is weakened and prone to tear.

↳ Pneumothorax can occur as a result of physical injury to the chest wall

Treatments: In most cases, air is absorbed over 2-3 days while the tear heals, hence treatment x necessary

↳ In severe cases, lung may collapse in which a tube is inserted through the chest wall in order to suck out the air using a syringe

Pulmonary ventilation (breathing) is the physical movement of air into & out of the respiratory tract to maintain adequate alveolar ventilation

↳ Function: prevent build up of CO_2

: ensure continuous supply of O_2

Boyle's Law - Pressure is inversely proportional to volume

↓ volume of gas, ↑ pressure

Gas flows from an area of high pressure to an area of low pressure

Hence air enter lungs when atmospheric pressure >>> intrapulmonary pressure

Blood Gas Transport:

Gas transport – process of carrying O_2 from alveoli to systemic tissues & CO_2 from systemic tissue to alveoli

Gas exchange occurs through diffusion and is dependent upon

i) diffusion surface area
ii) diffusion distance for gases
iii) concentration gradient between alveolar air & blood [differences in partial pressure]
iv) Solubility of gases
v) Coordinated blood flow & air flow

Dalton's Law of Partial Pressure – "the total pressure exerted by a mixture of gases is the sum of the pressures exerted independently by each gas in the mixture"

Partial Pressure – pressure exerted by each gas
↳ Directly proportional to its % in the total gas mixture

	Pressure / mmHg	
	Atmosphere	Alveoli
N_2	597	573
O_2	159	100
CO_2	0.3	46
H_2O	3.7	47

* P_{atm} at sea level = 760mmHg
P_{O_2} = 20.9% of atmosphere
20.9% × 760 mmHg = 159 mmHg
* If total pressure of a mixture of gases changes, % of each individual gases does NOT change *

Henry's Law – "amount of gas that dissolves in water is determined by its solubility in water and its partial pressure"

- at equilibrium, the amount of dissolved gas in the solution is proportional to the partial pressure of that gas

↑ P_{gas} – ↑ no. of gas molecules in solution
↑ P_{O_2} – ↑ amount of O_2 in solution

Types of Respiratory Disease:

- obstruction < airflow resistance ↑ – narrowing of airways
  outflow pressure ↓ – elastic recoil of lungs lost

obstruction: conditions which impede rate of airflow into & out of lungs

restriction: conditions ↓ lung volume & air

- restriction – reduced compliance → ↓ vital capacity
- infection & inflammation

Measuring Airway resistance – Forced Expiratory Volume in 1sec (FEV_1) by spirometry

FEV_1 is normally ~80% of FVC, if FEV_1/FVC < 80%, indication of ↑ airway resistance

Chronic Obstructive Pulmonary Disease (COPD) < chronic bronchitis – narrowing & emphysema – recoil

- narrowing of airways – ↑ air resistance
- elastic recoil of lungs lost – outflow pressure ↓

} ↓FEV

- residual volume ↑ – appearance of chest over inflation
  L as exhalation of air ↓, efforts needed for exhalation

Chronic Bronchitis – inflammation of bronchi in the lungs

Acute bronchitis – due to bacteria / virus
– days / weeks

Chronic bronchitis – due to smoking [illegible] / environmental irritants
– lasts or least 3 consecutive months in 2 years

- irritants inflame bronchi
- abnormal mucus secretion
- plugs airways
- prone to infection
- further inflammation
  [neutrophils, macrophages, lymphocytes]

} diameter of airway ↓

20% males have chronic bronchitis

Result < airway obstruction → turbulent airflow
shortness of breath / wheezing
chest pain, chronic (productive) cough
L> sputum are released which then can be analyzed to determine the causative agents

Treatment – stop smoking
– bronchodilators – act on smooth muscle in bronchioles
– antibiotics

* males have ↑% chance of developing chronic bronchitis

Restrictive lung disease – main disorders

- fibrosis – development of excessive connective tissue
- respiratory distress syndrome (IRDS, SARS, ARDS)

↑ alveolar walls become rigid

Acute disease
Sepsis or severe trauma
↓
protein exudation [excess protein production]
↓
oedema

Chronic Disease
industrial dust, drugs, rheumatism
↓
inflammation
↓
Fibrosis

Fibrosis – lungs stiffer
↳ alveoli replaced by fibrotic tissue → compliance ↓

Causes
- inhaled environmental & occupational pollutants
- cigarette smoke
- autoimmune disease

No effective treatments

Respiratory Tract Infections
- upper respiratory tract infections [nasal cavity, pharynx, larynx]
- common but minor
- average adult ~ 2-4 prevalence / year

[trachea, 1° bronchus, alveoli]
- lower respiratory tract infections
- less common but serious
- eg: bronchitis, pneumonia, tuberculosis

Pneumonia ; can be caused by virus as well
caused by bacteria : Streptococcus pneumoniae, Staphylococcus aureus & Klebsiella pneumoniae

Affect bronchi & alveoli → inflammatory exudate
↓
fills alveoli
↓
leads to 'consolidation' → gas exchange ↓
lungs tissue become firm & airless

Tuberculosis (TB)
- caused by inhalation of mycobacterium tuberculosis – highly contagious
  → enter alveoli, replicate in alveolar macrophage forming [Ghon focus]
  → initial infection – ineffective immune response
  → bacteria move to lymph nodes
  → collagen deposited around bacteria
  → lymph nodes erode, releasing bacteria
- 2 phases : latent vs active disease
  latent – asymptomatic, non-infectious, granuloma in lung tissue, non contagious
  active – spread to bronchioles & circulation
- no treatment due to antibiotic resistance

granuloma → Destruction of alveoli
forms infecting lung tissue, causing active TB.