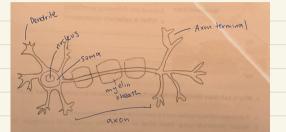


outside)

3. Kefractory Period → period of time following a nerve impulse before neuron is able to fire again the impulse is reversed (Nat inside; Kt

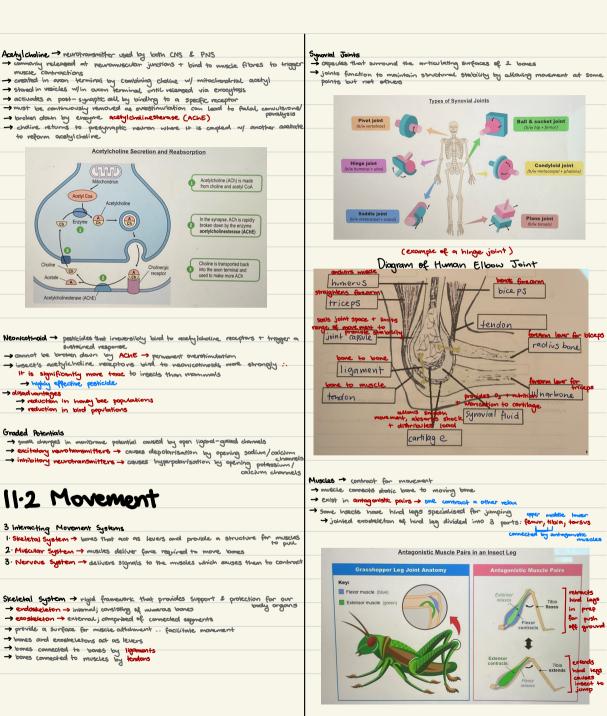
-> following repolarisation -> ionic distribution is reversed (Nat inside ; kt

→ before neuron can refire, resting potential must be restored via antiport



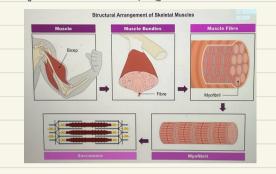
Myelin → fatty while substance to insulate axon

→ mixture of protein & phospholipide → 1 speed of electrical transmissions via salterbury conduction



Organisation of Skeletal Muscles

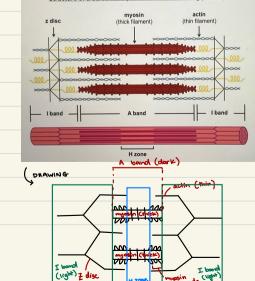
→ tighty packaged muscular burdles surrounded by connective taske (pringing) → each bundle contains multiple made fibres formed from fired mediceus -> muscle fibres contain tubular myofibrils that are responsible for muscular → the myofibrils can be divided into repeating sections auded sourcomprete



Muscle Fibre Specialised Features

- 1 multinucleated -> many nuclei from fusion of individual cells
- 2. many mitochandria -> muscle contraction requires ATP hydrolysis
- 3 sarcoplasmic reticulum stores calcium ions
- 4.2 different myofilaments -> thin (actin) and thick (myosin)

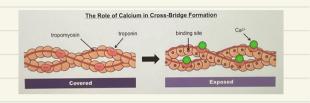
- Sarcomeres → repeating contractile units made of mysoin 2 actin → mysoin antoins small heads which bind to regions of actin → machenat of these 2 filaments awes builtening 2 shortoning of sarcomere + actin filaments radiate out from 2 diese to archer antral mysoin in place → shinked pattern along length of fibres
- -> centre appears dorriver due to actin + myosin overlap (A band)
- → sides appear lighter as only actin to preservt (1 band) → A band may contain elighty lighter central region where any myasin is located (H zone) Structure of a Sarcomere (and associated banding pattern)



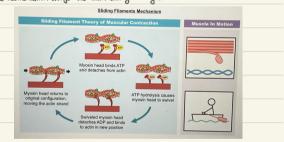
Process of Muscular Contraction

1. Depolarisation and Calcium Ion Release

- →action potential intggers release of activitional into motor end plate. → activitates initiates depolarisation -> spread through muecle fibres via T tubulee
- → calcium ions released from sarcoplasmic reticulum -> sourcommere contraction is traggered
- 2 Actin and Myosin Cross Bridge Formation
- Actin and Myosin Coss Bridge Fornation tropanin & tropanyasin on actin, the binding sites for myosin heads are covered by a blacking complex calcium lows bind to tropanin, reconfiguring the complex to expose binding sites -> the myosin heads then form a cross - bridge w/ actin



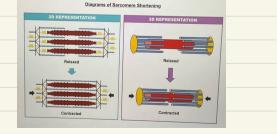
- 3. Sliding Mechanism of Actin & Myosin ATP binds to the wyssin based, breaking the cross-bridge between actin & myosin
 - → ATP hydrolysis causes the mysesin heads to change painton, moving them towards next actin timeling site.
 → the mysesin heads bind to the new actin sites + verim to their original configure
 → the reviewhatton drags the actin along the mysesin



4. Sarcomere Shortening

- \rightarrow the repealed recrientation drogs the actin filaments along the largen of the registin \rightarrow this pulls the £ discs closer together \rightarrow sorconvere shortens \rightarrow this causes the whole nuecle from to contract

- -> this movement reduces the width of the H zone, but doesn't change the length of the A bands



6.3 Defence Aqainst Infectious Disease

Surface Barriers \rightarrow the first line of defence against infectious diseases -> prevents entry of pathogens into the body -> skin and mucous membranes

Skin

- -> protects external structures
- → consists of a dry, thick and tough region composed preclaminantly of dead surface -> contains biochemical defence agents
- -> skin secretes lactic acid + fatty acids to lower pH (56-6.4)

Mucaus Membergnes -> protects internal structures (externally accessible eg. tradea, oesophagus)

- → thin region of living surface cells that release fluids to worsh away porthogens → contains biochemical defence agents
- -> may be ciliated to aid in removal of pathogens



- Blood Clots (haemostosis) -> mechanism of repair for broken/damaged blood vessels → to prevent blood loss from the body + limit pathogenic access to the bloodstream → 2 key components
 - \rightarrow Platelets \rightarrow undergo a structural change when activated to form a study plug at the domaged region (primary)
 - → Fibrin strands → form an insoluble mesh of fibres that trup blood cells at the site of damage (secondary)

Coagulation Cascade

- -> process by which blood clots are formed (complex set of reactions)
- -> 'clothing factors' cause platelets to become sticky + adhere to damaged area → valso initiate localised vasoconstriction to reduce blood frow
- → " trigger conversion of inactive zymagen prothrombin into activated enzyme -> thrombin antalyses the conversion of soluble fibringen into insoluble fibrin - the fibrin form a platelet plug and trap RBC to form a temporary clot -> when damaged region is repaired -> an enzyme is activated to dissolve the clot



(innate immune system)

Non-Specific Rasponse → second live of defence against infectious disease. → principle component = NWECs → engulf + digest foreign bodies

- also includes inflammation, fever, antimic robotal chemicals
- → 2 key properties → oloes not differentiate (non-specific)
 - -> responds to all infections the same way (non-adaptive)

- Plagocytes
 → phagocytes = the process by which solid waterials are ingested by a cell (enhagines)
 → phagocyte (eucloogies circulate in the blood + move into the body tissue in response to infection
 → therefore themes palence channels (intermine) with down WEGs to the sile
- -> damaged tissues release chemicals (histomine) which draw WBCs to the site
- → pathogens are engulfed when cellular extensions surround it and fuse to form internal vesicles
- the vesicle is then fused to a lysosome + pathogen is digested
- -> pathogen fragments (mitigens) presented on surface to stimulate third line of defence

body recognises as foreign and will elicit an imm <u>Phagocytosis by Leukocytes</u>



(adaptive immune system)

Specific Response -> third line of defence

→ can differentiate between different pathogens + langet a specific response → can respond rapidly upon re-exposure → preventing symptoms (immonological me

Lymphocytes

- → B-lymphocytes (8 cells) → antibooly-photwaing cells that recognise + target particular antiopens
- → Helper T-lymphocytes (T cells) → regulator cells that release chemicals to activate specific B cells
- -> when antigens are presented, specific helper T calls are activated
- -> T cells release cytokines to activate B cells to produce antibodies
- -> the activated B cell will divide + differentiate to form short lived plasma cells that produce large amounts of the specific antibody
- → antibodies will harget their specific antioen, enhancing immune system ability to recognise + destroy the pathogen → few B calls will differentiate into memory cells for long-lasting immunity

Antiloodies

- -> mode of 4 polypeptide chains -> the ends of the arms are variable ... specific
- -> each antibody recognises a unique autique (like enzyme-substrate)

Antibiotics → compounds that kill or inhibit the growth of microboes by targetting prokanyotic metabolism → features that may be targeted: 703 ribosomes, enzymes, cell wall components

- → features that may be taugeded: 703 ribosomes, enzymes, cell wall compone → evicary otic cells do not have these features .. hast won't be targeted
- → may kill backeria or suppress its potential to reproduce
- → viruses are not alive and take over cellular machinery of host cells → : commot be treated w/ antibiotics → instead specific antiviral agents → these target viral enzymes or components of capsial

Antibolcterial Resistance

- -> some bacterial strains have evolved wl genes resistant to antibiotics
- → some bacterial shains have evolved wij genes remaining to the source on proliferate → because bacteria reproduce at a rapid rate: resistant shains can proliferate pricky
- resident strains can pass resistance goves to susceptible strains via backerial conjugation (horizontal gove transfer)
 the prevelouse of resistant backerial strains is increasing rapidly of humans
- -> over-prescription or misuse of antibiotics
- -> antibiotics freely available + used in livestock
- -> common in hospitals

Development of Antibiotic Resistance



(1928)

Penicillin → first chemical compound found to have autibilistic properties by Alex → accident, unintended contamination of a dish containing 5 aureus → penicillium motel began to grow → hale of inhibited bacterial growth around it → Freming concluded motel was releasing fourillin that was billing bacteria.

Medical Applications -> Florey and Chain

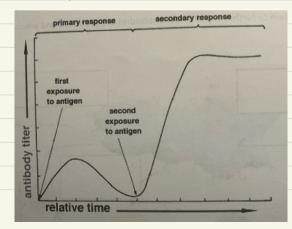
- -> tested penicillin on mice
- -> 8 mile injected w/ path ogen -> 4 then injected w/ Penici Uin
- -> untreated mice died; treated survived

Human immunodeficiency Virus $(HV) \rightarrow a$ retrovirus that infects helper T calls disabiling body's adaptive immune system

-> causes a variety of symptoms + infections called Acquired Immuno-Deficiency Sundrome (AIDS)

- -> Effects of HIV
 - -> HIV specifically targets the helper T cells

 - → virus becomes inactive allowing infected T calls to reproduce → virus becomes reactive + begins to spread → destroying the T calls → antibodics give under to be produced → dimmonity → body becomes succept ble to less hormfil infections overhal death
- Transmission of HIV Transmission of HIV Transmission of HIV Transmission of HIV transfusions, breastfeeding.) -> can be minimised through protection
- -> small minority are immune



Self vs Non-Self

- → Immune system has capacity to differentiate → all nucleaded cells of body possess unique + distinctive surface molecules ('setf')

11.1 Antibooky Production

laccination

- → these 'self' markets are added major histocompatability complex (MNC) molecules → function as 1D tags → : if not there = foreign (entigen) → antigen (non-self) determinants = surface markets, self markets of diff cell, protein

Blood Transfusions

- -> RBCs are not nucleated ... do not passess self markers + can be transferred
- however they have basic antigen markers which limit apacity for transfusion
- -> RBCs may possess glycoproteins (A & 8 antigens) either A or B or AB or none

Summary of the ABO Blood Groups Type O Туре А Type B **Type AB** Antigens A + B Neither A or B Antigen A Antigen B Antigen (on RBC) ... 0 Anti-A Antibody Neither Antibody **Both Antibodies** Anti-B Antibody 1×1 47 XY1 1 Antibody (in plasma) XXX 4 4 4 4 7 Can only have Cannot have B Cannot have A Can have any type of blood O blood or AB blood or AB blood Blood Can have B or Is the universal Donors Can have A or Is the universal

recipient

donor

(pothogenesis)

O blood

Pathogens -> an agent that causes disease	(booctenia, fungi, pourasite, virus)
-> disease = disturbs normal body function	
-> illness = deterioration of normal health	
-> ability to cause disease is species speci	lfic .
-> certain pathogens may be able to infect	rounde of hosts (eg. influence strains
= bird flu; bubonic plague = ratts)	•
-> animal to human = zooneses	

O blood

Disease Transmissio

- → direct contact (woodily fivials) → contamination (ingestion)
- -> airborne (coughing / sneezing)
- -> rectors (intermediary organisms)

Clonal Selection (only the extra info from HL) -> B cells divide and form clones (clonal selection)

- -> most clones develop into plasma cells + some into memory cells sevele 2000 mittadies per second for 4-5 plays

