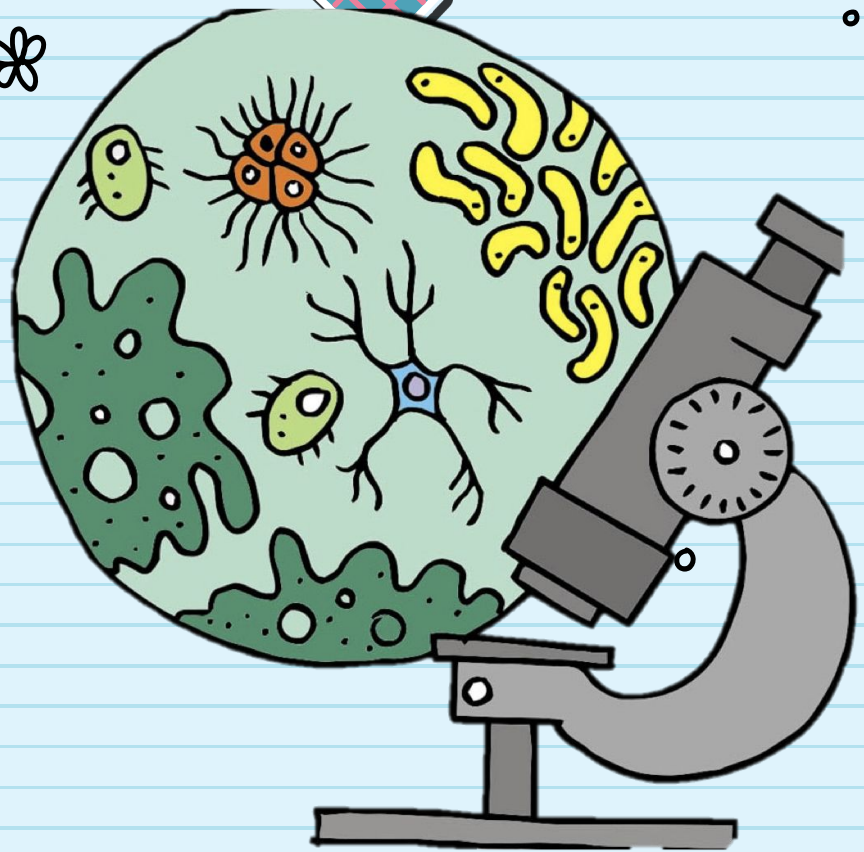
Semester 2 Exam Gil

Georgia, Alex, Brendan

01

GENERAL INFORMATION

- Ensure to write **RHS** or **LHS** to avoid losing marks
- Aim to not use abbreviations- or use the full term (and abbreviate within brackets) at the start of your answer → continue abbreviation throughout the rest
- Ensure to include both intra/ extra oral when describing images (where you can see both)
- Read the question- ensure that your answer is applied to the scenario and isn't too generic; if question asks you to only label Q2- don't waste time doing anything else (it won't get you more marks- we've tried :( )



02

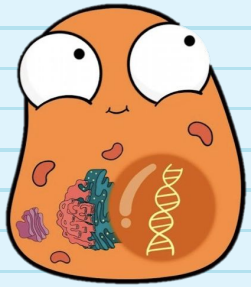
Microbiology

REMINDER:

PROKARYOTE

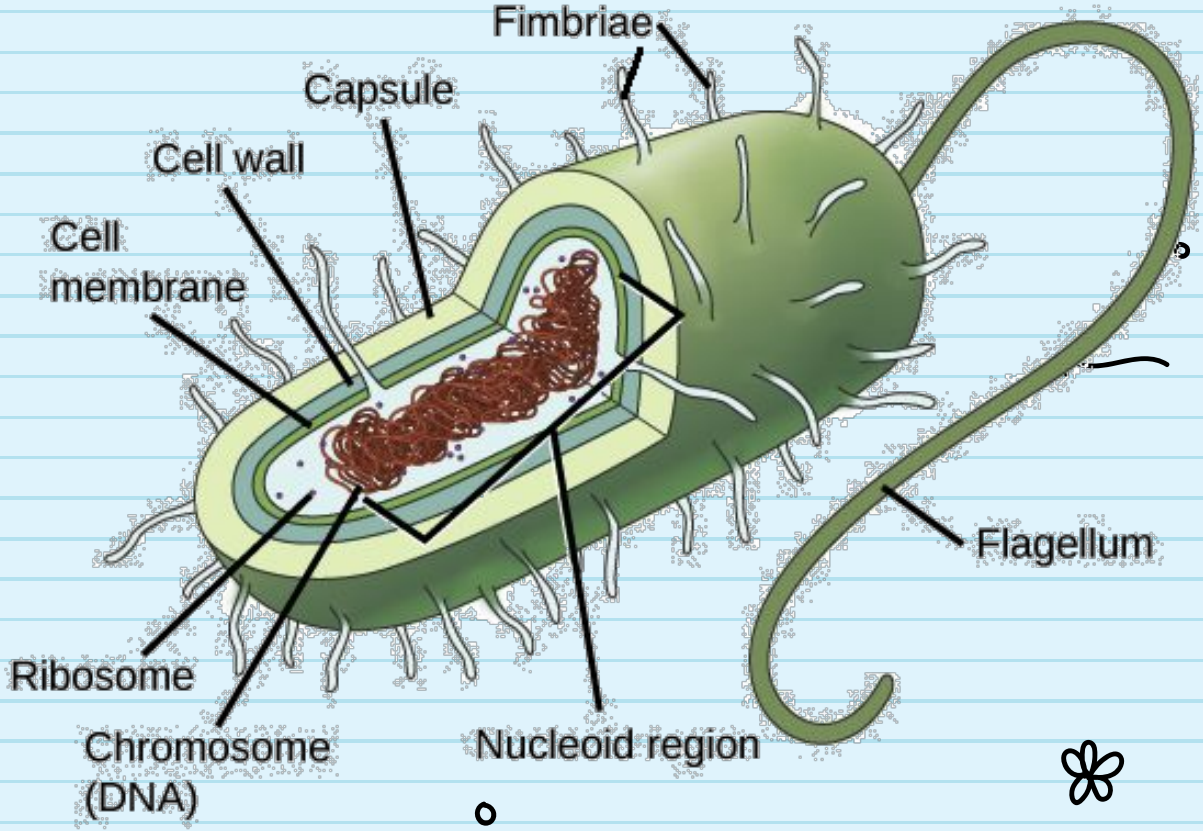
- No nucleus or membrane-bound organelles
- Cell membrane and wall
- Single circular chromosomes

EUKARYOTE



- Membrane-bound nucleus and organelles
- Cell membrane
- Chloroplasts in plant eukaryotes
- Multiple linear chromosomes

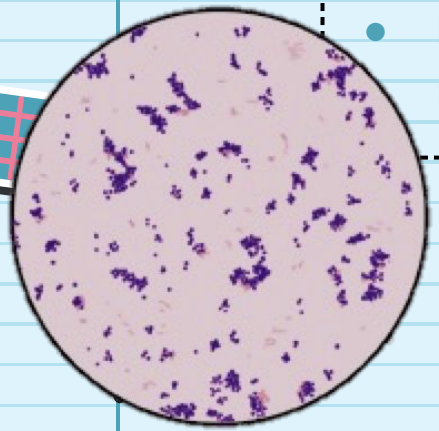
BACTERIAL STRUCTURES



GRAM STAINING & STRUCTURE

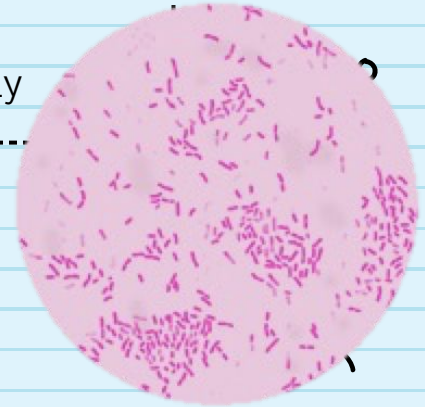
GRAM POSITIVE

- Thick peptidoglycan layer
- Affected by lysozome and penicillin
- Does not decolourise readily

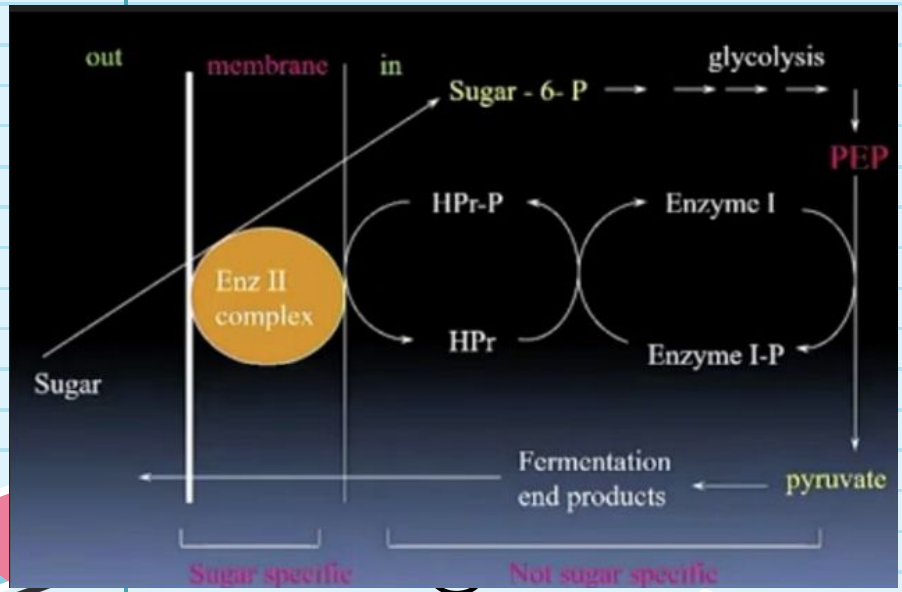


GRAM NEGATIVE

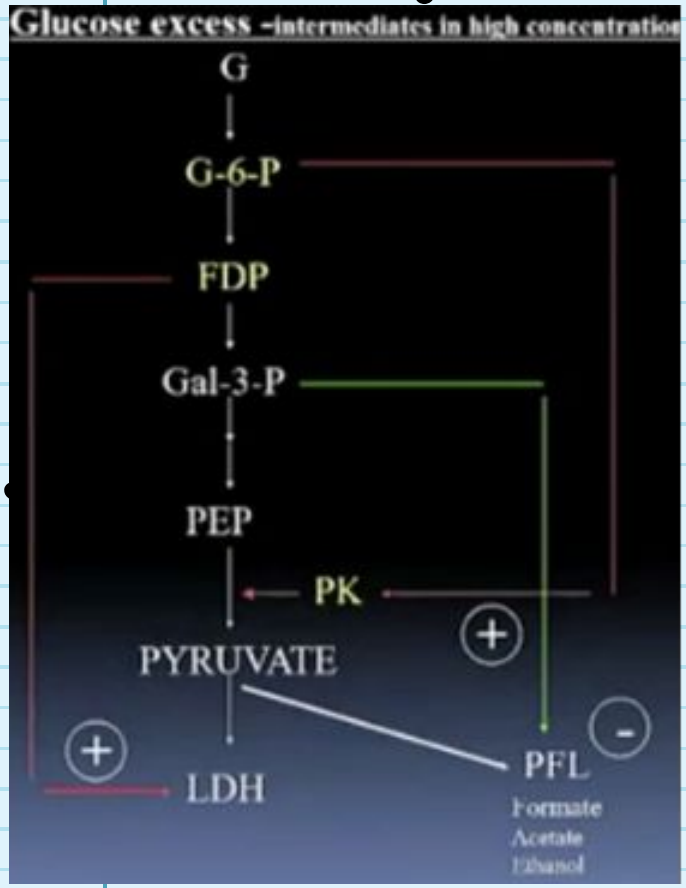
- Dual membrane, thin peptidoglycan
- Lysozyme and penicillin ineffective
- Decolourises readily



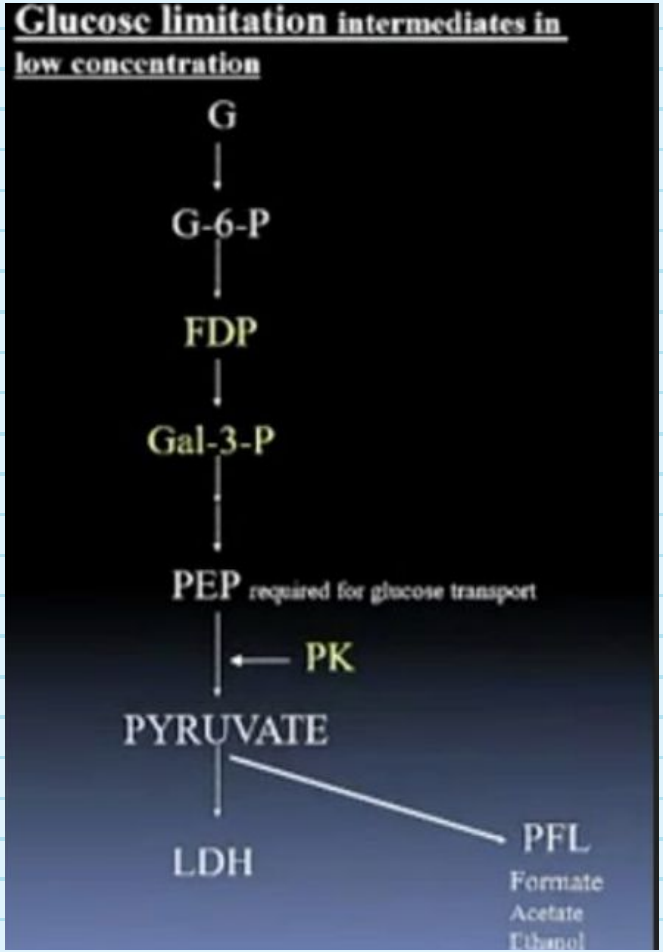
PEP-PTS



- 1. Uptake of sugar and phosphorylation
- 2. Glycolysis → PEP
- 3. PEP → pyruvate donates phosphate to Enz II
- 4. Uptake of more glucose
- 5. Pyruvate → lactic acid



Pyruvate Kinase	Activators	G-6-P (glucose excess)	Convert PEP to pyruvate and lactate
Lactate dehydrogenase	Activators	FDP	Convert pyruvate to lactate
Pyruvate formate lyase system	Inhibitors	Gal-3-P	Inhibits PFL pathway so glucose Solely converted to lactate to maximise energy production



1. Low G-6-P, FDP, Gal-3-P

2. Reduced LDH, build up of PEP

3. PEP used in PEP-PTS system

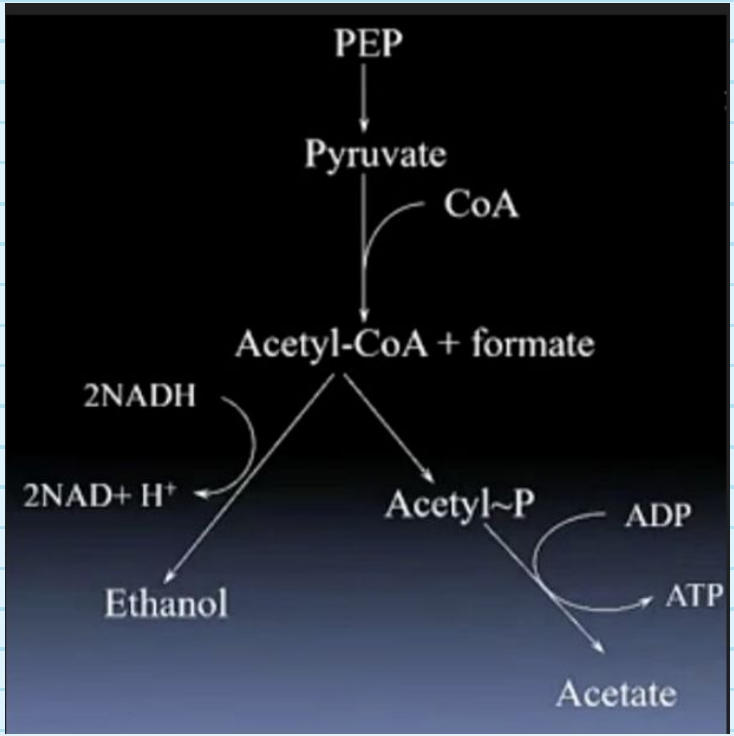
FLUORIDE & FASTING

1. Fluoride inhibits enolase

2. Conversion of Gal-3-P to PEP reduced

3. Suppress PEP-PTS famine uptake system

- Overnight fasting → acetic, formic, succinic, butyric acid
- Feast → lactic acid



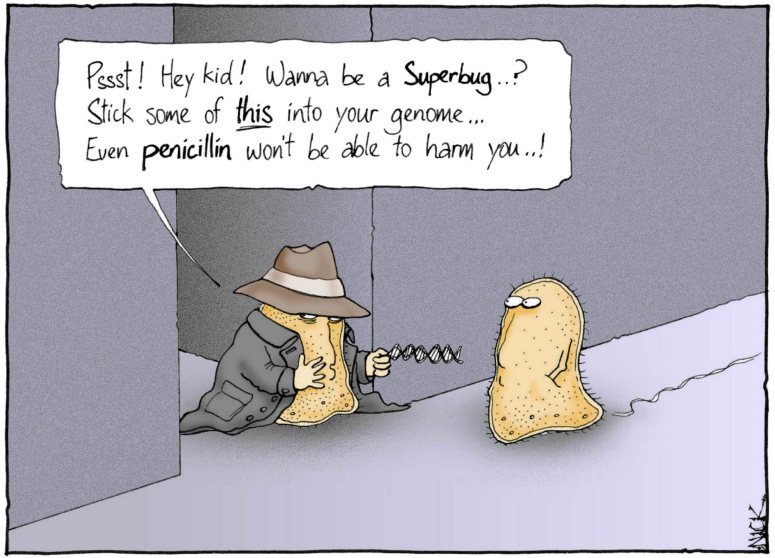
VIRULENCE

A TTACHMENT TO HOST

I NVASION OF HOST

G ROWTH AND COLONISATION

E VASION ie. exotoxins, endotoxins, quorum sensing



It was on a short-cut through the hospital kitchens that Albert was first approached by a member of the Antibiotic Resistance.

ANTIBIOTICS MECHANISMS

1. Cell wall synthesis inhibitors - Beta-lactam antibiotics

2. Protein synthesis inhibitor - macrolides, clindamycin, tetracyclines

3. Nucleic acid inhibition - Rifampicin

4. Cell membrane disruption - lysozyme, antifungals

ANTIBIOTIC RESISTANCE BIND TO TARGET SITE:

1. Change drug binding site

2. Enzymes inactivate drug

3. Upregulate pumps for removal

STAPHYLOCOCCI

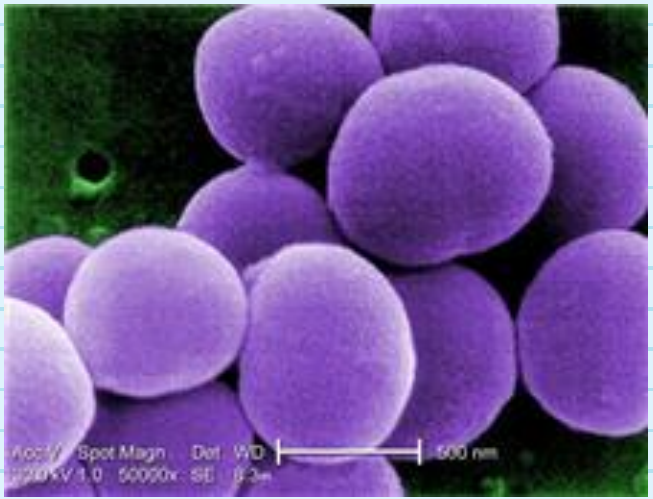
Can be identified by the **catalase** test and the **coagulase** test

Properties include being *highly resistant to stress* and also being pyogenic (heat)/ localised/ inflamed.

Virulence:

1. Attachment: adhesins, protein A
2. Growth: coagulase
3. Evasion: Capsule, peptidoglycan

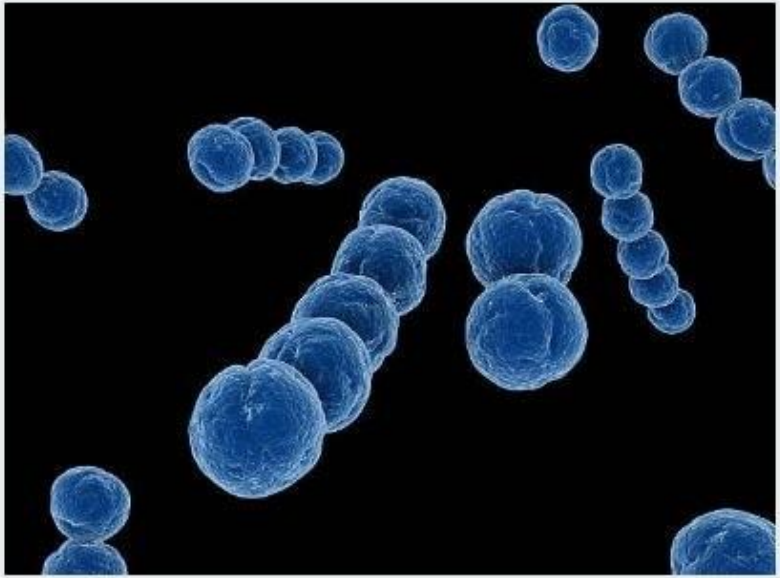
Direct damage by alpha, beta toxins, leukotoxin, hyaluronidase, beta-lactamase, PTSAgs



STREPTOCOCCI

Group A Streps - most frequent pathogen; S. pyogenes
- URT, rheumatic fever, necrotising fasciitis

Important virulence factors
1. M protein - alpha helix structure
2. Streptococcal pyrogenic exotoxins A, B, C
3. Streptolysin Treatment - beta-lactam antibiotics



BACTERIAL GENETIC TRANSFER MECHANISMS

- In prokaryotes, only reproduction by binary fission (clones) and thus need horizontal mechanisms of variation to increase virulence and increase survival in specific environments (ie. antibiotic resistance).
- **Horizontal gene transfer** = occurs through transformation, conjugation and transduction. Donor DNA are transferred to the recipient cell- as they line up homogeneously and allow for crossing over (to introduce new genes).

TRANSFORMATION:

Death of bacteria leads to cell wall rupture + leakage DNA → random 'pick-up' of DNA by specific species and transfer of genes to form a recombinant organism. Does not require direct contact; cell needs to be 'stressed'.

CONJUGATION:

- Two methods: plasmid transfer (not recombinant organism) or chromosomal transfer (recombinant organism). Requires **contact** via conjugation tube.

TRANSDUCTION:

- By a virus/ phage carrier.
- Lytic & Lysogenic

DISINFECTION & STERILISATION

Bacteriostatic = growth inhibited (is reversible if agent is removed). Bacteria remain viable

Bactericidal = irreversible lethal effect, kills bacteria.

- Difference between sterilisation, disinfection and antisepsis.

- Factors that **affect death rate** of bacteria:
  - *Time & concentration of bacteria*: reduce bioburden before sterilisation.
  - *Time & Concentration of agent:* inverse relationship of concentration of substance to required killing time.
  - *Time & temperature:* increased activity of agent with increased temperature (inc. action of thermal death point).

- Methods of sterilisation/ disinfection:
  - Moist heat: Not true sterilisation (does not remove heat resistant spores/ viruses). Includes boiling at 100 degrees.
  - Autoclaving: Gold standard, does not kill prions. Moist heat in form of saturated steam under pressure in air tight vessel. 121 degrees for 15-20 mins (pressure increases boiling point).
  - Dry Heat
  - Radiation
  - Filtration

TB

- *Large, non-motile, rod shaped myobacterium tuberculosis*

Stage 1	1. Droplet nuclei created by talking, coughing etc. and remain viable for period of time (depends on humidity) 1. Inhaled and bacteria non-specifically taken up by inactivated alveolar macrophages 2. Protected from immune system 3. Large nuclei usually lodged in URT where infection unlikely to develop 4. TB begins when small droplet nuclei reach the alveoli May get carried to lymph nodes (5% cases) where it can spread to other organs associated with high O_2 levels e.g. base of brain
Stage 2	Begins 7-21 days after initial infection, slow generation time multiplying exponentially in inactivated macrophages Primary site of infection in *Ghon focus*
Stage 3	1. T and B Lymphocytes begin to infiltrate, surround infected macrophages to form *granuloma/tubercule (walled off/controlled)* 2. Within granuloma, T lymphocytes secrete cytokines to activate macrophage and destroy IC bacteria 3. Or also directly kill infected macrophages 4. Centre of tubercule – "caseation necrosis" with "cheesy" consistency due to necrosis Latent infection diagnosed by positive tuberculin skin test
Stage 4	1. Some inactivated macrophages surround the tubercule fail to controlàgrowth of tubercule → Tubercule may invade artery or blood supply line and cavitation of lungsà miliary tuberculosis 2. Hematogenous spread for 2ndary lesions usually in bones, lymph nodes, peritoneum with high fatality (unable to breath) 3. Disease can waneàmay be controlled by healing and fibrosis

03

General pathology

Contents

- Consequences of cell injury
- Inflammation
- Disorders of cell growth/differentiation
- Wound healing
- Tooth socket healing

Consequences of cell injury

Causes:

Oxygen deprivation -> most common way to form reactive oxygen species (ROS) -> damage lipids, proteins & DNA

Physical agents

Chemical agents

Infectious agents

Immunological reactions -> excessive immune responses and autoimmunity

Genetic changes (mutation of DNA) -> oncogenesis

Nutritional changes

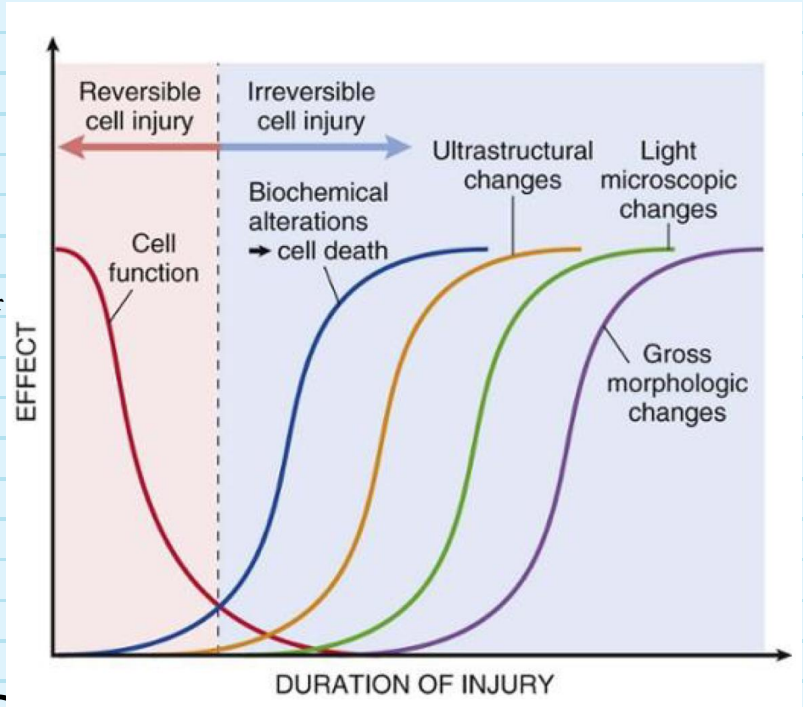
Consequences of cell injury

Mechanisms:

1) Formation of ROS -> highly damaging to cell contents
2) Loss of ATP -> reduced cell function and many consequent effects
3) Influx of calcium into the cell -> causes activation of damaging cellular enzymes
4) Damage to cell membranes causing loss of cellular content and leakage of lytic enzymes (lysosomes)
5) Protein misfolding/DNA damage -> activation of pro-apoptotic enzymes

Severity of the response is dependent on the severity, duration and type of insult.

- Larger scale consequences are seen **later** than smaller scale ones



Acute Inflammation

Purposes of Inflammation:

- Remove infectious/deleterious agents
- Facilitate healing
- Protective mechanism

Causes:

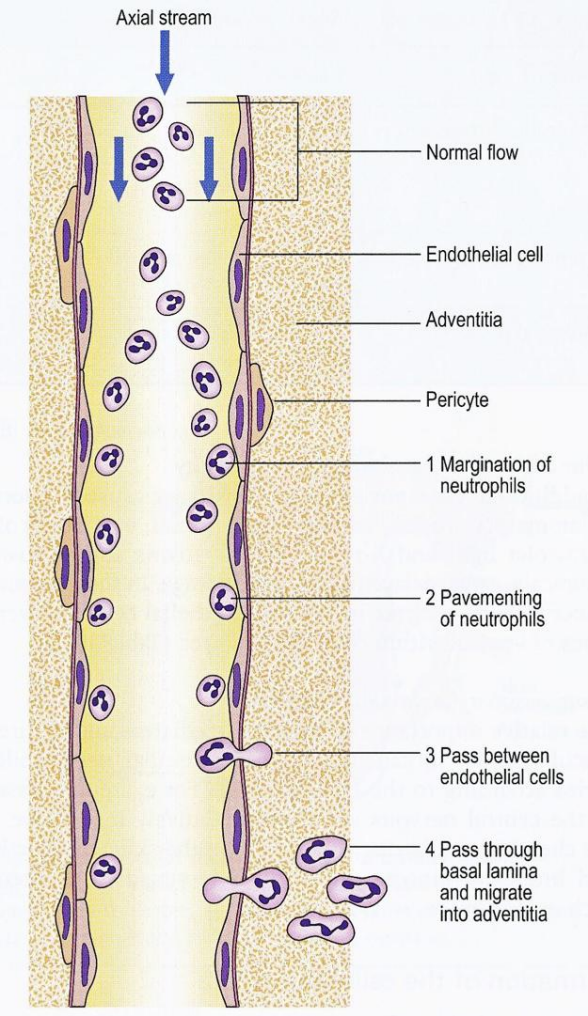
Infectious agents
Hypersensitivity
Physical and chemical stimuli
Necrosis -> release of lytic and damaging molecules

Signs:

Rubor (redness) -> increased amount of blood and RBCs at local area
Dolor (pain) -> release of sensitisers for free nerve endings, fluid pressing against free nerve endings
Tumour (swelling) -> release of chemokines causing inflammatory infiltration and more fluid
Calor (heat) -> increased amounts of fluid at site increasing local heat capacity
Functio leasa (loss of function) -> immobilisation from swelling

Other features of acute inflammation:

- Cardinal signs
- Cellular exudate -> neutrophils that extravasate
- Changes in vessel calibre
- Fluid exudate (complement, immunoglobins, ILs, other inflammatory markers)


Axial stream
Normal flow
Endothelial cell
Adventitia
Pericyte
1 Margination of neutrophils
2 Pavementing of neutrophils
3 Pass between endothelial cells
4 Pass through basal lamina and migrate into adventitia

Acute Inflammation

Types of Acute Inflammation:

Serous: Formation of protein rich fluid at local site

Fibrinous: FIbrotic deposition and fibrin -rich coating over site

Purulent: Production of pus *what is pus?*)

Benefits of Inflammation:

- Dilute toxins
- Allow immune response to occur
- Transport drugs and nutrients
- Fibrin formation (barricade)

Inflammation - chronic

Causes of chronic inflammation:

- Failure to remove debris/agents
- Recurrent acute inflammation
- Autoimmune disease

Features of chronic inflammation:

- Recurrent cycles of injury and healing
- Change in inflammatory cell profile (less neutrophils, more macrophages and plasma cells)

Can be **granulomatous** in nature -> cause collection of macrophages and their derivatives -> TB

Inflammation - Dental relevance

- Ability of pulp to resolve inflammation is dependent on the inflammatory response -> dependent on the severity, nature and duration of stimulus

- Methods by which the pulp will respond to injury include:
  Reparative dentine formation
  Fibrosis
  Granulation tissue formation

- Why is pulpal tissue so susceptible to death?
  Due to limited size of the chamber and low blood flow meaning limited capacity for:
  Drainage
  Swelling
  Repair
  Concentrated stimulus

Cell disorders

Atrophy:
Decreased size of a cell or organ

Hyperplasia:
Pathologically caused increase cell growth (*dental example?*)

Metaplasia:
Transition of one differentiated cell type to another (*Smokers?*). Can be reversed via removal of the stimulus.

Dysplasia:
Increased cell growth with decreased differentiation and atypical morphology. Still reversible if stimulus is removed.

Neoplasia:
Abnormal, uncoordinated and excessive cell growth following dysplasia. Irreversible, even if the initial stimulus is removed.

Cell disorders - Neoplasia

	Benign	Malignant
Metastasis potential (*What is metastasis?*)	No	Yes
Spreading	Remains localised, often encapsulated	Invades nearby structures
Growth rate	Slow	Fast
Differentiation	Low	High
Suffix*	Oma	Emia

* some exceptions (melanoma, lymphoma, etc)

Cell disorders - Neoplasia

What causes a cancer to form? (oncogenesis)

1) Failure of tumour suppressor genes - loss of protectory mechanisms against oncogenesis
2) Mutation of proto-oncogenes into oncogenes - upregulation of cell growth
3) Expression of telomerase - prevents cell senescence and apoptosis

- **Important tumour suppressor gene:** p53 -> repair damaged DNA prior to s phase (DNA replication), arrest cell cycle in g1 or cause apoptosis

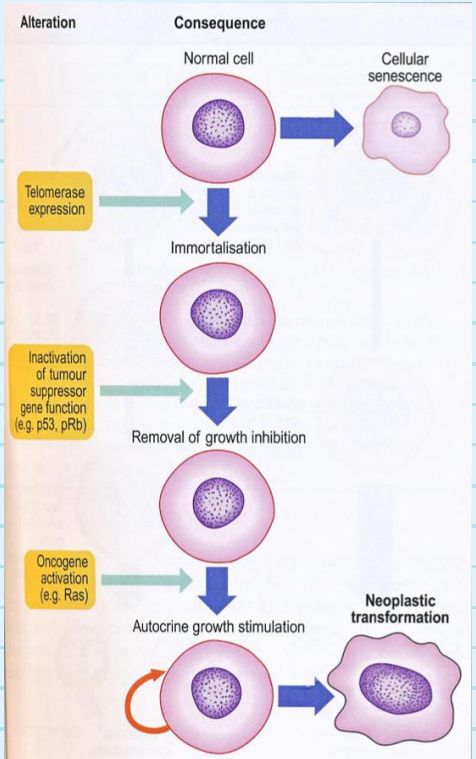


Fig. 11.27 Essential steps in neoplastic transformation. Three key genetic events are the minimum needed to convert a normal human cell into a neoplastic cell. *Telomerase expression* prevents telomeric shortening with each cell division and thus thwarts cellular senescence. *Inactivation of tumour suppressor gene function* in the immortalised cells removes inhibition of growth control. *Oncogene activation* sets up autocrine growth stimulation; the cell now produces a growth factor for which it already has a receptor or expresses a receptor for a growth factor it normally produces. The cell is now fully transformed. (Based on observations by Hahn WC et al 1999 *Nature* **400**: 464.)

Wound healing

Primary intention:

- Occurs when the tissue is in apposition
- Result is small blood clot, small amount of granulation tissue and minimal amount of scarring

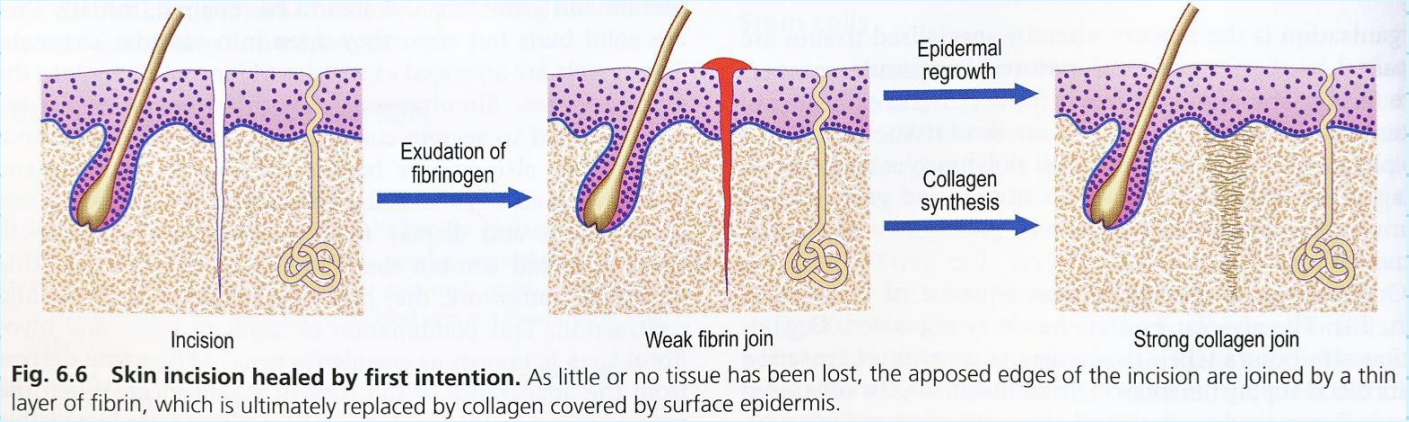


Fig. 6.6 Skin incision healed by first intention. As little or no tissue has been lost, the apposed edges of the incision are joined by a thin layer of fibrin, which is ultimately replaced by collagen covered by surface epidermis.

Wound healing

Secondary intention:

- Occurs when the area to be healed is larger
- Result is large blood clot, inflammation, large area of scarring and greater wound contraction.

0 Hours: Injury

24 Hours: Formation of a blood clot with underlying inflammation

2 days - 1 week: Formation of a scab (exudate, fibrin), reduced inflammation and formation of granulation tissue

2 weeks: Reduced granulation tissue, increased collagen

1-2 months: Epithelial organisation (scab is gone), collagen maturation, wound contraction and decreased vascularity

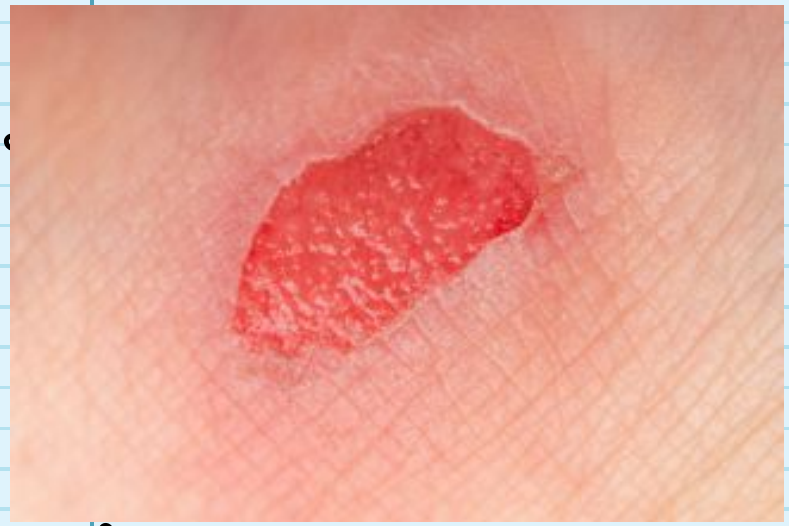
Key Mediators:
Vascular endothelial growth factor (VEGF)
Epidermal growth factor (EGF)
Cytokines (IL-1, TNF-alpha)
MMPs
Platelet derived growth factor (PDGF)

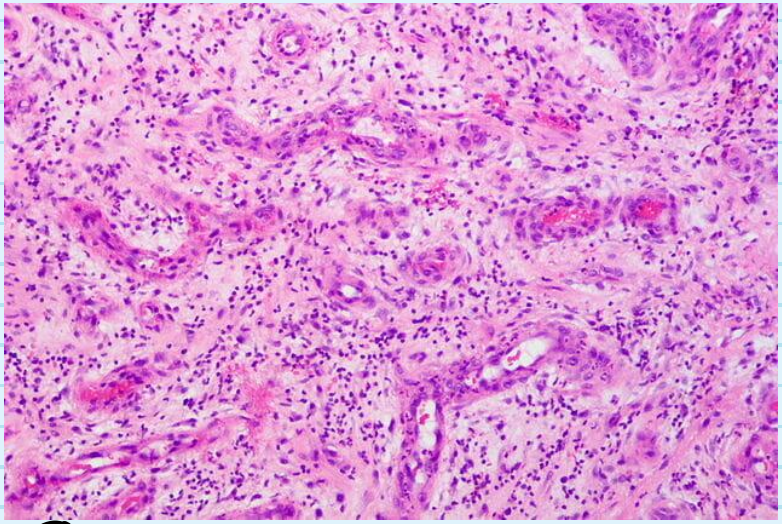
Factors Affecting Wound Healing:
Metabolic Disorders (Diabetes Melltius)
Nutrition (Protein, Vitamin C, zinc)
Infection
Tissue involved - how vascular is it?

Wound healing - Granulation tissue

What is Granulation Tissue?

- Increased formation of blood vessels - -> supply the tissue with oxygen and nutrients required for growth
- Macrophages -> remove debris and damaged tissue
- Fibroblasts + their products -> form new CT





Wound healing - Tooth socket healing

Immediately: Formation of a haemorrhage and blood clot

48 hours: Fibrin slough and clot contraction

3-7 days: Epithelial migration, reduction in bone height, initial granulation tissue formation

2-3 weeks: Increased bone resorption at crest and lamina dura, nearly complete epithelial coverage, formation of osteoid and woven bone at periphery of socket

4-5 weeks: Increased bone formation and remodelling of bone

2 months - 1 year: Increased bone remin and remodelling

04

ANATOMY

Contents

- Pathways
- Position of the cranial nerves
- Motor neuron defects
- Parasymapthetic ganglion

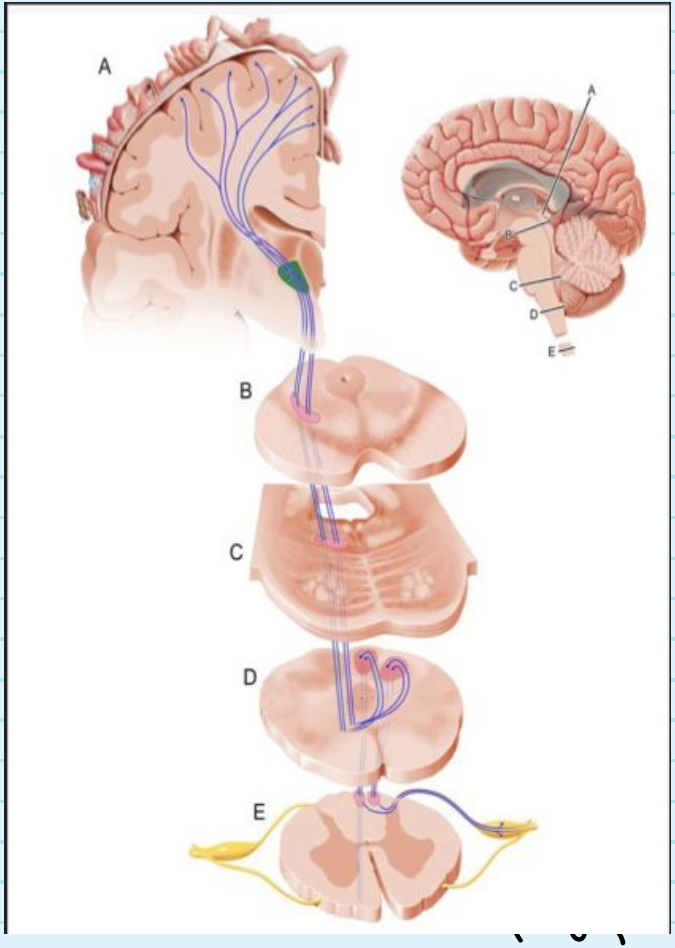
Pathways

Spinothalamic Pathway
(Pain, temperature, crude touch)

Final synapse is in thalamus - 3rd order neuron finishes in somatosensory cortex of the post-central gyrus

Spinal cord - decussation occurs

Enters at posterior primary ramus - 1st order neuron (sensory)



Pathways

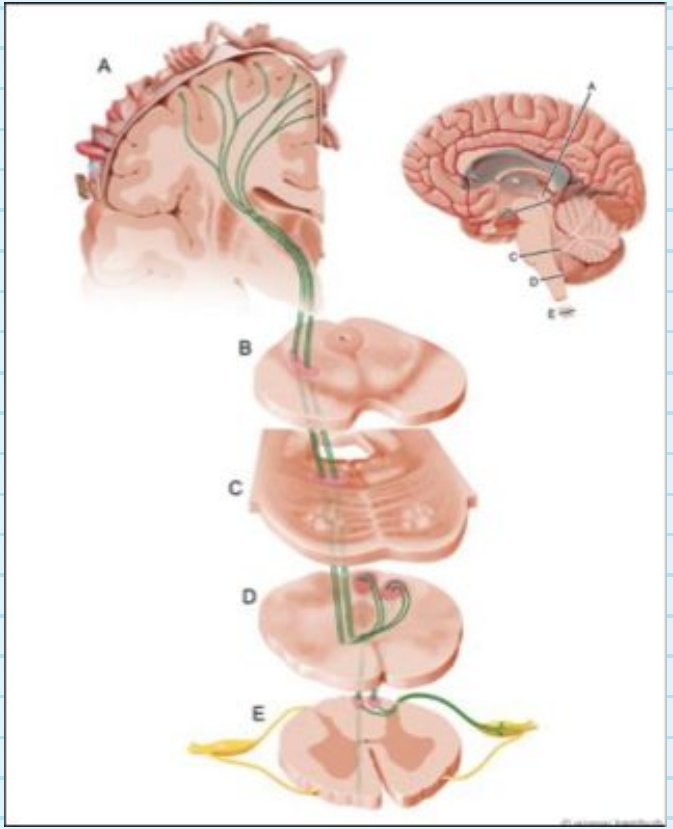
Dorsal Column
(Fine touch, vibration)

Final synapse is in thalamus

Medulla - Synpase
nucleus **cuneatus** for hands and upper body

nucleus **gracilus** for legs and lower body

Fasciculus gracilus/cuneatus enter via PPR



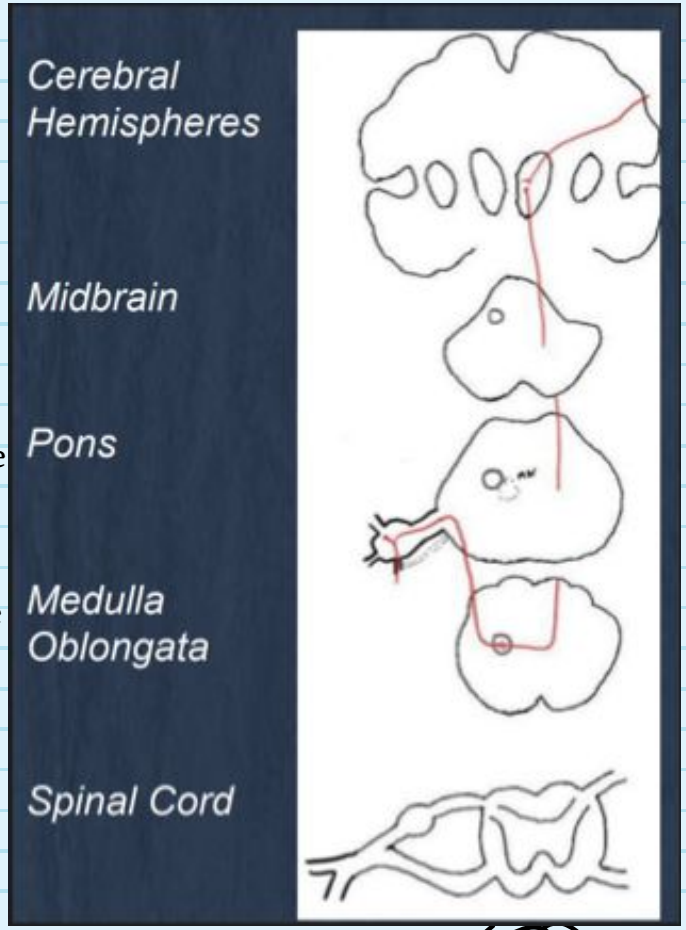
Pathways

Trigeminal Lemniscus Pathway
(Crude touch and pain, dental)

Final synapse is in thalamus - nerve finishes in somatosensory cortex (post central gyrus)

1st order neuron enters the CNS here

1st order neuron synapses and decussates here

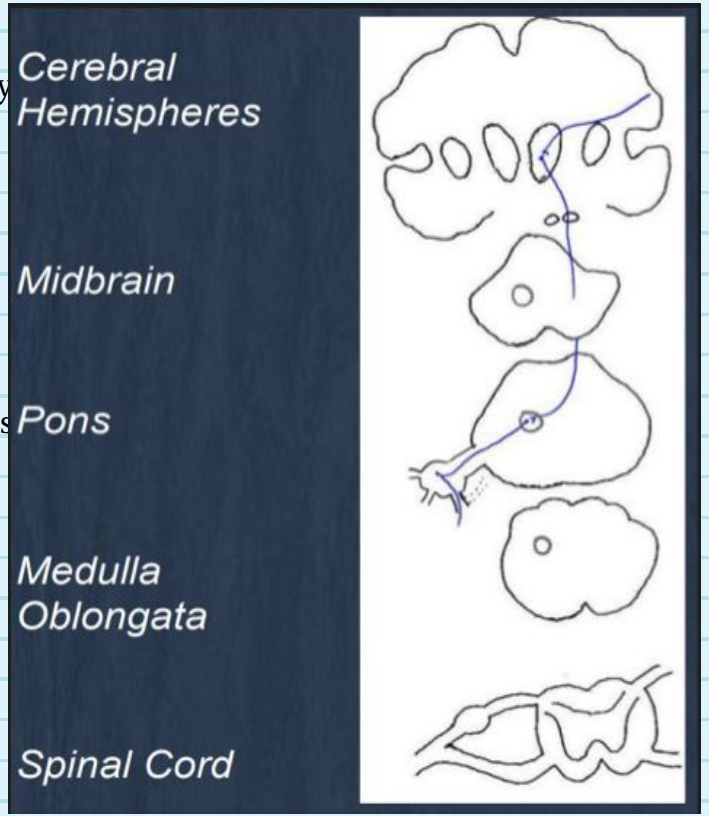


Pathways

Fine Touch Dental Pathway

Final synapse is in thalamus - nerve finishes in somatosensory cortex (post central gyrus)

1st order neuron enters the CNS, synapses and decussates here (pontine nucleus)

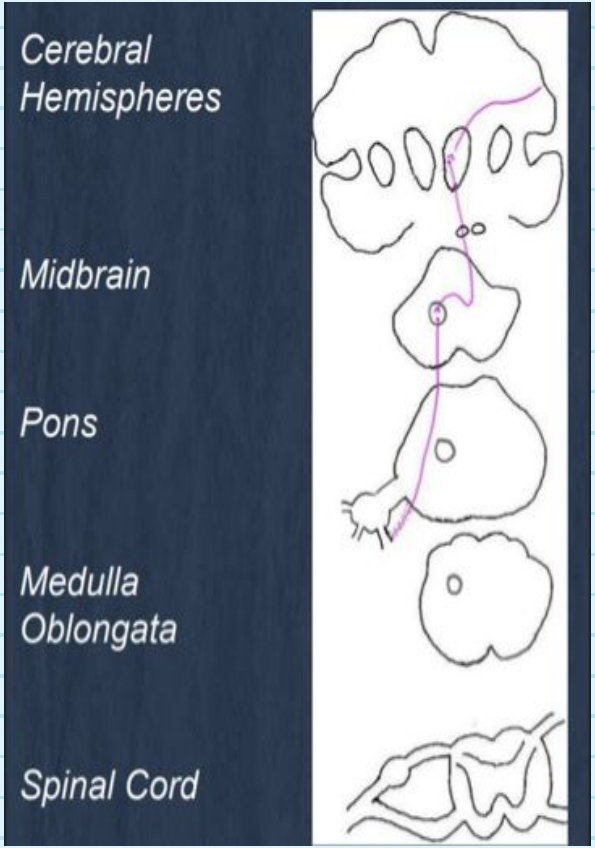


Pathways

Final synapse is in thalamus - nerve finishes in somatosensory cortex (post central gyrus)

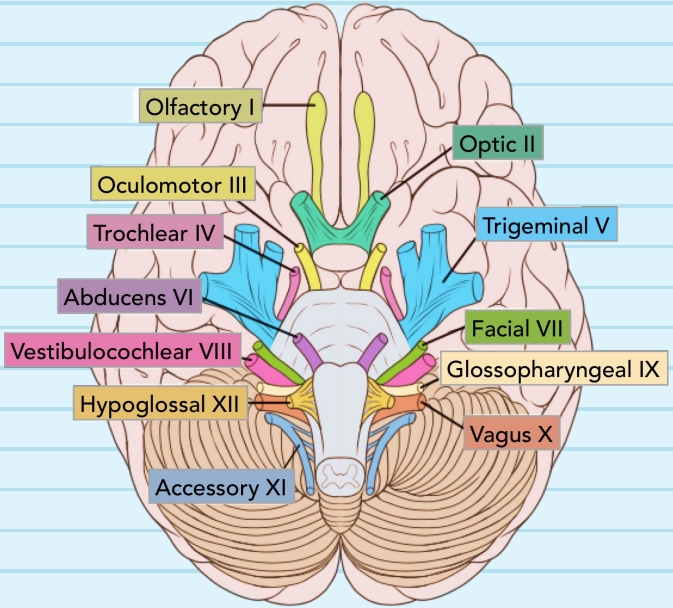
Proprioception Dental Pathway

1st order neuron enters the CNS, synapses and decussates here (mesencephalic nucleus)



Position of the cranial nerves

Cerebral hemispheres (2)
Midbrain (2)
Pons (4)
Medulla (4)



Nerve	Foramen
I	Cribriform plate
II	Optic canal
III	Superior orbital fissure
IV	Superior orbital fissure
V1	Superior orbital fissure
V2	Foramen rotundum
V3	Foramen ovale
VI	Superior orbital fissure
VII	Internal auditory meatus/facial canal
VIII	Internal auditory meatus
IX	Jugular foramen
X	Jugular foramen
XI	Jugular foramen
XII	Hypoglossal canal

Motor neuron defects

Symptoms	Upper MN Defect	Lower Defect
Atrophy	No	Yes
Paralysis	Spastic	Flaccid
Fasciculations and Fibrilations (twitching)	No	Yes
Reflexia	Hyper	Hypo

Motor neuron defects - example

MR BRIAN MIDDLETON – A CROOKED SMILE

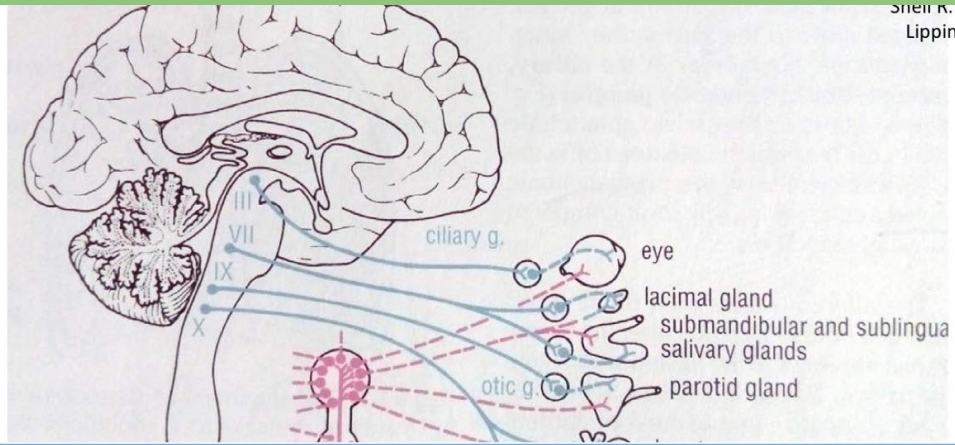
Mr Brian Middleton has come to see you about his crooked smile. He suffered a stroke six months ago and now has weakness of his arms and legs on the right side of his body. When you examine him and ask him to smile and show his teeth, the corner of Mr Middleton's mouth is pulled back on the left, exposing his teeth, but not on his right. He also has drooping of the left upper eyelid. When his left upper eyelid is opened passively, the pupil is deviated downwards and laterally. His left pupil is also dilated and non-reactive to light. Mr Middleton tells you that he has difficulty focussing on near objects with his left eye.

Motor neuron defects - example

- **Weakness on the right side of the body** -> lesion affecting LHS UMN before decussation (entirety of LHS is affected, LMN lesion would only affect below lesion)
- **No movement of muscles of facial expression on RHS** -> defect of facial nerve
- **Drooping of left eyelid** -> defect of levator palpebrae superioris / CN III
- **Left upil is deviated downwards and laterally** -> CN III defect
- **Left pupil is dilated and non-reactive to light** -> defect to CN III

Putting all this together, the stroke has likely occurred on the LHS of the brain, causing a UMN for the corticobulbar pathway and facial nerve (assuming only lower half of face is affected), and a LMN lesion for the occulomotor nerve (hypo reflexia)

Parasympathetic ganglion of the head



Shell R. Clinical anatomy. Philadelphia: Lippincott Williams & Wilkins, p33.

Parasympathetic ganglia in the head region – carries secretomotor fibres			
Ganglion (comes of CNs)	**Path – presynaptic fibres**	**Path – postsynaptic fibres**	**Target gland**
1. Ciliary (V1)	CN III	CN V(1) Short ciliary n.s	Ciliary muscle (accommodation) Pupil (constriction)
2. Otic (V3)	CN IX → Lesser petrosal n.	CN V (3) → Auriculotemporal n.	Parotid gland
3. Pterygopalatine (V2)	CN VII (nervus intermedius) → Greater petrosal n.	CN V (2) Maxillary n. → CNV (1) Lacrimal n.	Lacrimal gland Mucous glands in the nasal cavity, pharynx, palate
4. Submandibular (V3)	CN VII → Chorda tympani	CN V(3) Lingual n.	Submandibular & sublingual glands

05

IMMUNOLOGY

Hypersensitivity

Hypersensitivity	Cells Involved	Examples
Type 1 - Immediate	IgE, mast cells, histamine	Asthma, anaphylaxis, latex allergy
Type 2 - Ab-mediated	Cytotoxic reaction with IgG or IgM Abs	RHD, penicillin
Type 3 - Immune complex-mediated	Immune complexes + complement + IgG	Systemic lupus erythematosus
Type 4 - Delayed	Cell-mediated (T-cells, macrophages, NK cells, cytokines)	Contact dermatitis, graft tissue/organ transplant

Clinical Significance

- Explain how type 4 hypersensitivity can lead to type 1 sensitivity
- Context of latex hypersensitivity in the dental setting -> proactive management + prevention
    - Thorough MHx examination, early appts, ask for medications, location, avoid latex products (RD, gloves), recognise symptoms/signs

Tolerance & Autoimmunity

Checklist

- 3 key features are required to achieve tolerance
- Think of tolerance as a 2 door-gate
  - 1st door: central tolerance
    - 'Negative selection' -> eliminate any developing B & T-lymphocytes which are self-reactive
  - 2nd door: peripheral tolerance
    - Prevent escaped self-reactive B & T-cells from causing autoimmunity
- Know very briefly about the autoimmune diseases
  - **Rheumatoid arthritis, Sjogren's syndrome**, systemic lupus erythematosus, multiple sclerosis, pemphigus vulgaris, mucous membrane pemphigoid
- Process of autoimmunity in Sjogren's syndrome
  - AutoAgs -> overexpression of cytokines -> CD4+ T-cells & B-cells -> destruction of salivary duct + acinar cells -> neural degeneration
- Treatment options for autoimmune diseases (RELEVANT FOR DENTAL SCENARIOS)
  - Symptom relief
  - Replacement of secretions/hormones
  - OHI options/alternatives
  - Immunosuppressants (last resort for dental practitioners)

Rheumatic heart disease

Type 2 Autoimmune Disease (Ab-mediated hypersensitivity)

- Strep A bacteria are coated with M proteins which mimic the structural components of heart myosin cells
  - Molecular mimicry
- Host cells mistaken the heart cells -> autoimmunity
- Leads to scarring of heart tissue + valves

Clinical Significance

- If acute rheumatic fever was left untreated -> can progress into RHD
- Patients often have prosthetic valves as a result of valval incompetency due to scarring of heart tissues and valves
- Increased predisposition to infective endocarditis
- Will need to consider antibiotic prophylaxis as a precautionary measure to all
  - Need to note if Pt is allergic to penicillin, etc. -> refer to Therapeutic guidelines
  - https://tgldcdp-tg-org-au.eu1.proxy.openathens.net/topicTeaser?guidelinePage=Oral+and+Dental&etgAccess=true

ANTIBIOTIC PROPHYLAXIS

Dental Implications

- Invasive dental Tx can create a medium for bacteria to enter the bloodstream particular for high-risk incidence of bacteraemia procedures involving gingival/periapical tissue manipulation or oral mucosa perforation (ADA Therapeutic Guidelines - P195):
  - Teeth extraction
  - Biopsy
  - Periodontal surgery, subgingival scaling & root planing
  - Replantation of avulsed teeth
  - Other surgical procedures *(eg. bone removal, implant placement, apicoectomy)*
- If PT has more than 1 procedure require antibiotic prophylaxis, dentist needs to plan all Tx such that it can be completed in 1 or at most 2 settings to avoid the need for multiple antibiotic doses
  - Helps to minimise the side effects of antibiotics in killing commensal (good) bacteria which can further compromise the immune system
- Oral administration for endocarditis prophylaxis:
  - Amoxicillin 2g (child: 50mg/kg up to 2g) orally, 60 mins prior to procedure
- IV administration for endocarditis prophylaxis:
  - Amoxicillin/ampicillin 2g (child: 50mg/kg up to 2g), within the 60 mins prior to procedure
- Intramuscular administration for endocarditis prophylaxis:
  - Amoxicillin/ampicillin 2g (child: 50mg/kg up to 2g), 30 mins prior to procedure
- Administration to PTs with severe hypersensitivity to penicillin (cefazolin if non-severe):
  - Oral: Clindamycin 600 mg (child: 20mg/kg up to 600mg) -> 60-120 mins prior to procedure
  - IV: Clindamycin 600 mg (child: 20mg/kg up to 600mg) -> within the 120 mins before the procedure

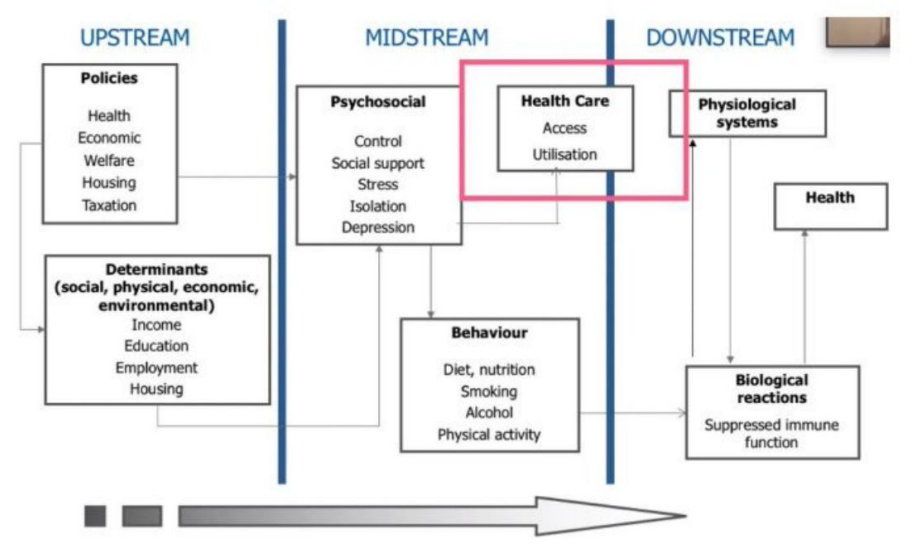
06

PIA

Social Determinants of Health

Checklist

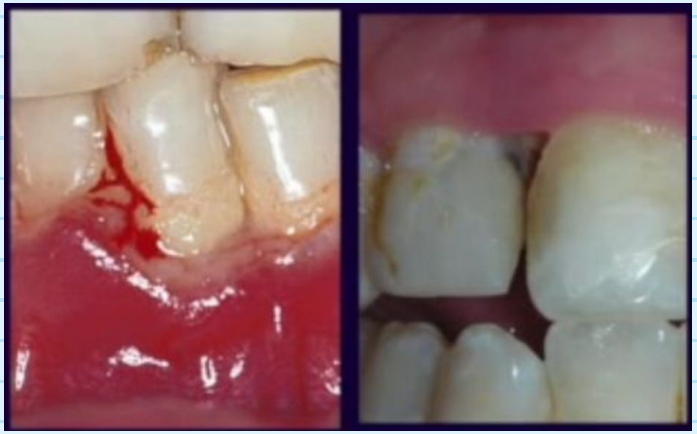
- Ensure you know your determinants of health (upstream, midstream & downstream factors)
- Apply this to the context of ILA 2.8 -> this is HIGH YIELD
- Revisit the Exam Semester 1 GIL for how this can be further applied in dental scenarios



Gingivitis + cleans +Management

Use a table to explain how debridement can improve with the healing of gingivitis *(consider histopathology, clinical signs & time duration)*

Histopathological	Clinical	Time (varies greatly from person to person)
• Repair of junctional epithelium • Reduced number of polymorphs in tissue & sulcus	• Less pus exudate • Less bad taste	4+ days
• Reduced vascular permeability • Reduction in the chronic inflammatory infiltrate in the gingiva adjacent to the sulcus and junctional epithelium	• Decreased redness • Reduced BoP • Decreased oedema Decreased tissue retractability • Decreased pocket depth	7-14+ days
• Increase in fibroblast number • Increase in gingival collagen	• Tissue becomes firmer • Increased resistance to probing	2-6+ weeks



Checklist

- Ensure you know your gingivitis + recession classifications
- Ensure that you know your curette and scaler cutting edge designs
- Know how to detect manage and treat gingivitis (please refer back to Dr Selbach's lectures on gingivitis)
- The table above helps to inform you of recalls with your patients following debridement
- Risk factors, signs & symptoms
- Tx options (debridement, CHx, antibiotics, OHI, smoke/stress counselling

Local anaesthetics

Checklist

- Absolutely need to revisit the LA GIL since it will comprise a majority of the content in PIA and Paper exams
- Remember that LA is required for SUBgingival debridement + restorative treatment + rubber dam clamp
- Know your anatomical landmarks for LA, vascular supply and innervation
    - Needle too low, too high, too lateral, too medial, too posterior
- Know how to manage and troubleshoot for failed LA
- Know your complications for LA
    - Commonly known: facial paralysis, trismus, soft tissue damage (post-op), temporary blindness
    - Less commonly known: persistent anaesthesia, heart palpitations, oedema, numbing of throat, numbing of arms
- Ensure that you know contraindications to LA (eg. uncontrolled hyperthyroidism, non-selective beta blockers)
- Know what to do if injection site is infected
    - Quantity of LA vs location of LA deposition

Restorative Steps

Example - Indirect Pulp Cap (eg. 27 has reversible pulpitis)

Informed consent
Topical LA (Ziagel – 5% lignocaine)
LA – (Lignocaine 2%, 1:80000 adrenaline -> ¾ carpule for 27 buccal infiltration & ¼ carpule for 27 palatal infiltration)
RD isolation from 27-23
Remove infected dentine and leave small amount near the pulp
Place conditioner (20% polyacrylic acid) for 10s and wash off/dry, don't desiccate
Remove the clamp and replace with a Tofflemire matrix band
Place a wooden wedge in between 26 & 27
Place RMGIC up to level of the DEJ and LC for 20s
Place 37% orthophosphoric acid etch at the enamel for 15s
Place adhesive/unfilled resin and LC adhesive for 20s
Place CR and LC for 20s
Articulating paper
Polish
Post-op instructions + possible post-op symptoms + long-term prognosis of tooth/Tx (if relevant)

PCC Communication