BDS2 Exam GIL

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PCC

Be aware, scenarios with volunteer patients can come up in exams:

- Anxious patient
  - Approach?
  - Management?
- Angry patient?
  - How do you respond to them?
    - DEFUSe

Local Anaesthesia

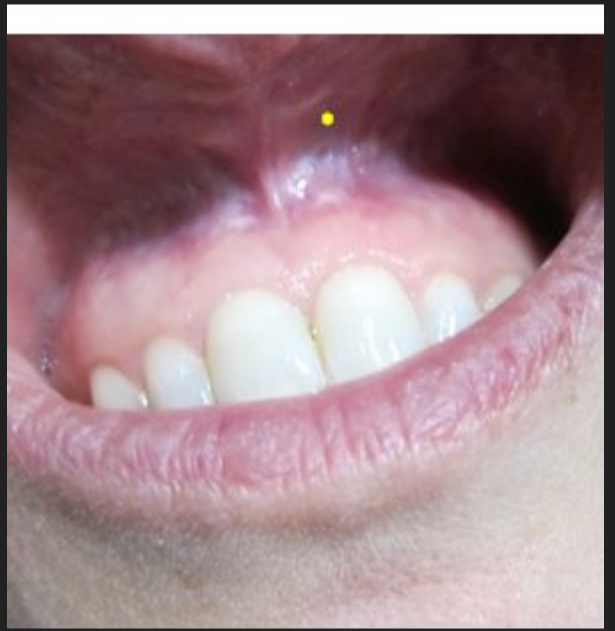
LA

How does pKa affected LA diffusion?

Why is it difficult to anaesthetise are with inflammation?

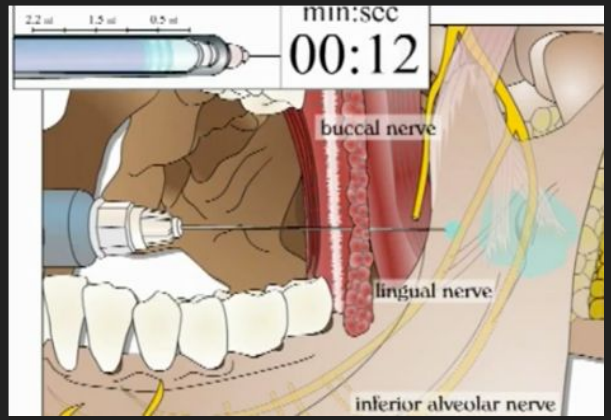
What are some reasons for LA failure?

Is it the correct landmark? If not, draw correct one.



So, why is it difficult to achieve anaesthesia in regions of inflammation?

$RN + H^+ <-> RNH^+$



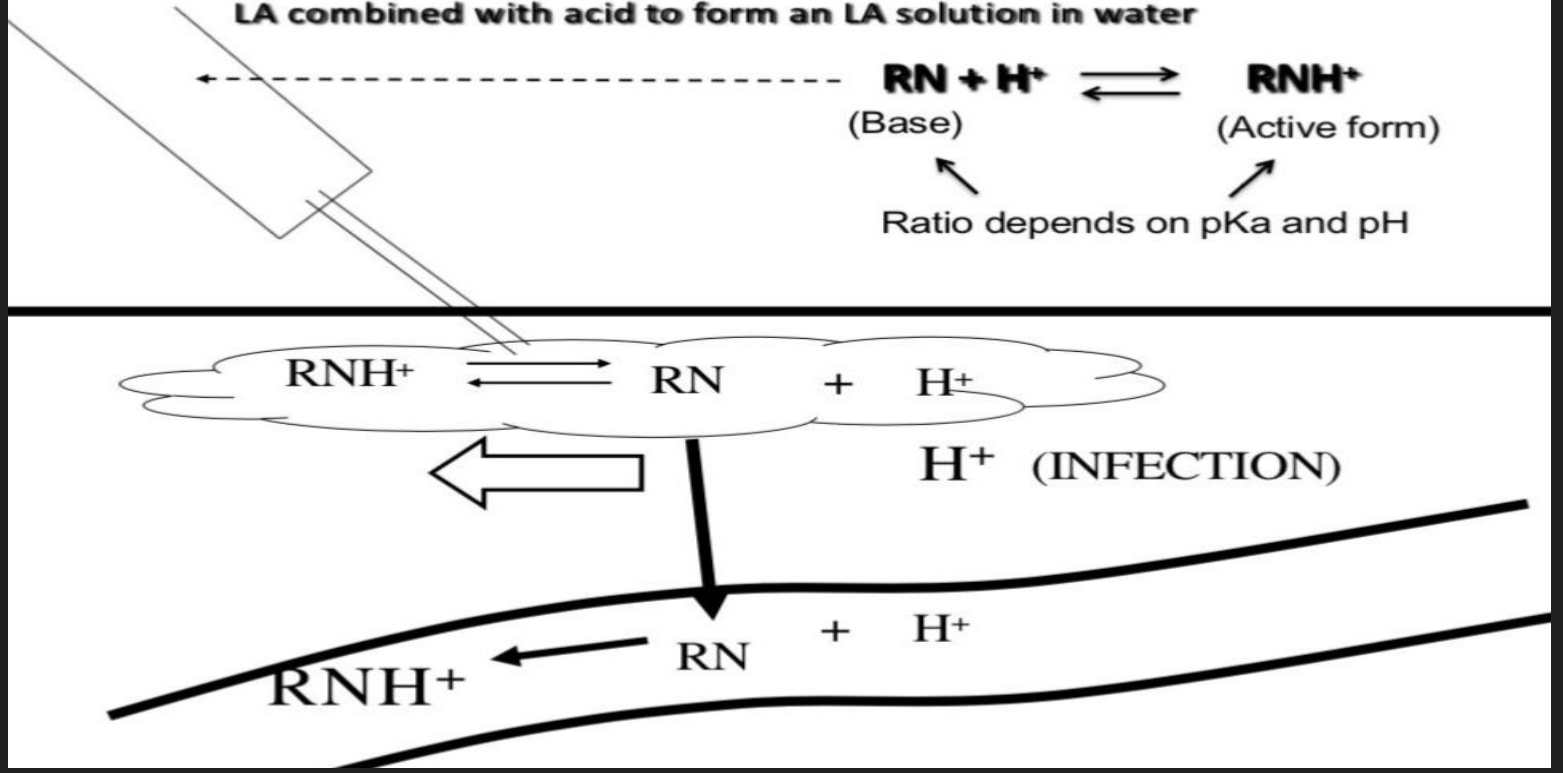
The active form of LA is RNH+ however only the uncharged RN version can diffuse through the nerve sheath.

The pKA refers to the pH at which the weak acid is half disassociated i.e. half the concentration of RNH+ and RN.

If the pH tissues at the injection site are much lower than the pKa of the LA (e.g. lignocaine is ~ 7.9), then that means there is less RN, hence very slow or no diffusion at all across the nerve sheath leading to delayed anaesthesia.

LA solutions

LA combined with acid to form an LA solution in water



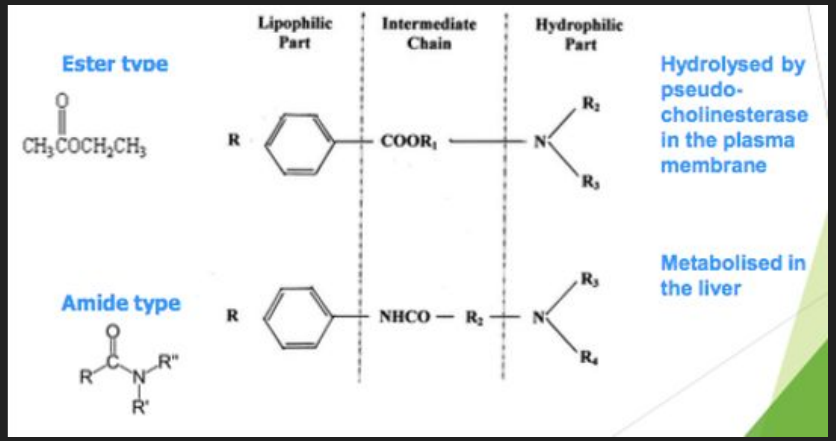
Types of LA

Ester types

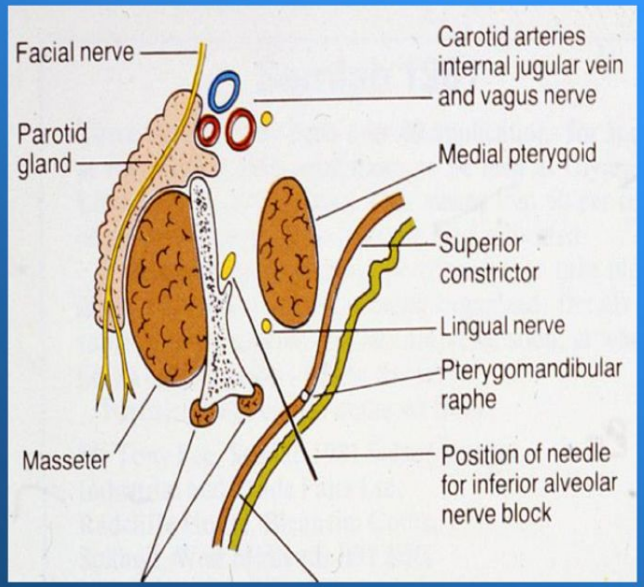
Hydrolysed in plasma membrane by cholineresterase

Amide types

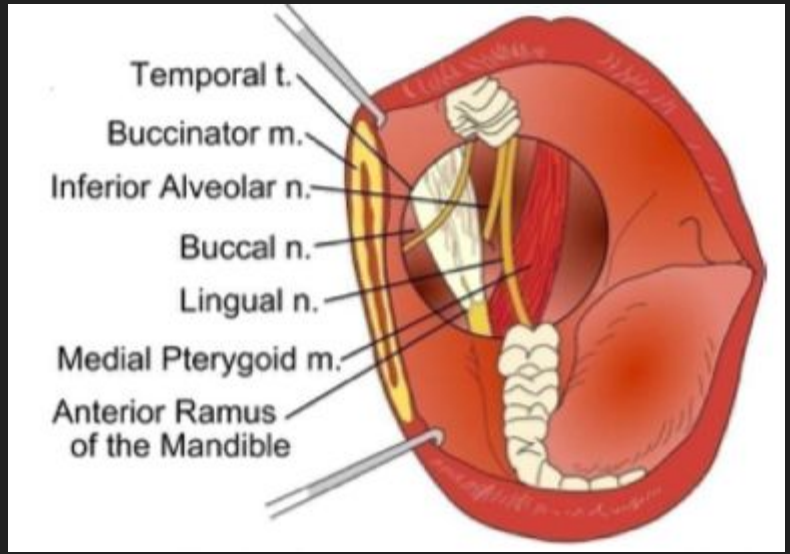
Metabolised in the liver

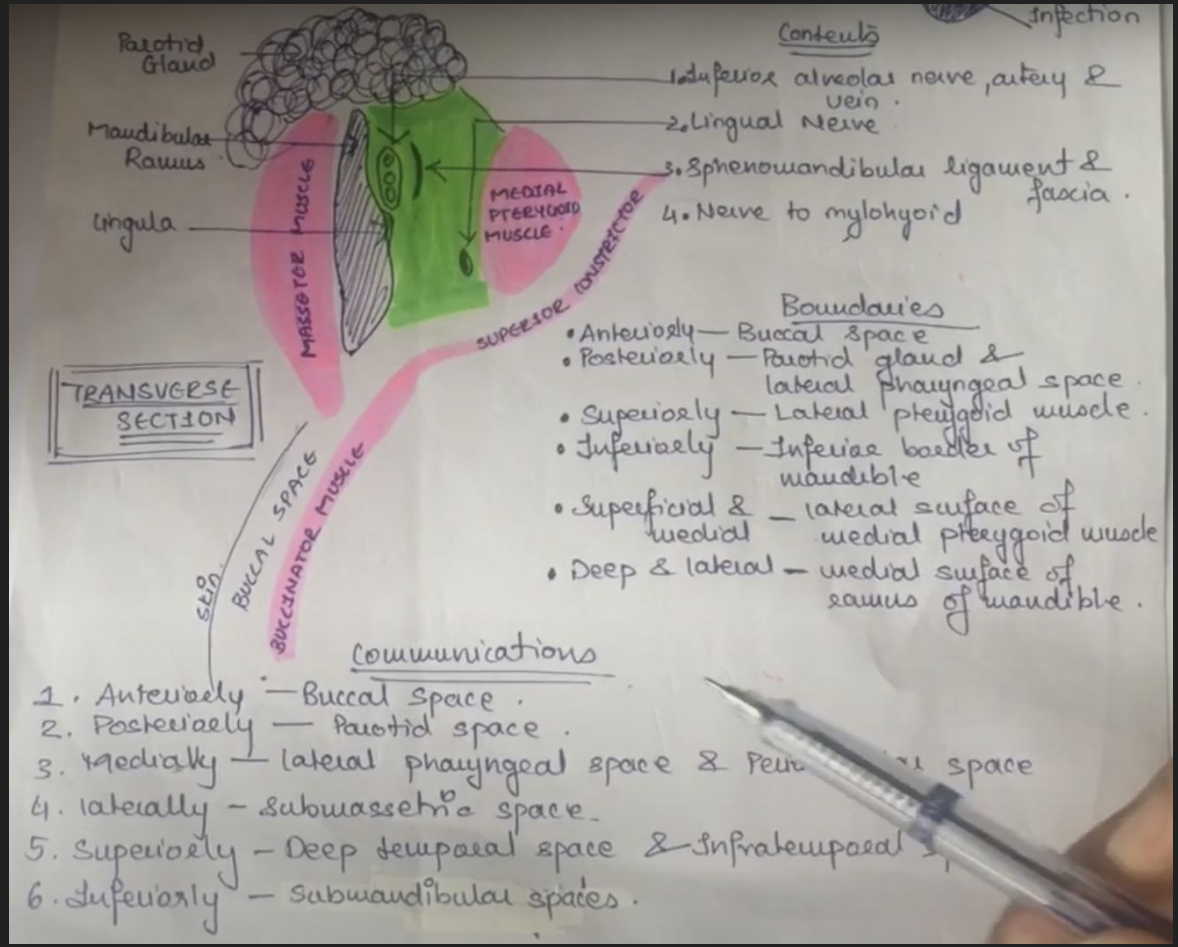


BOUNDARIES OF SPACE



Insertion of temporalis m. Buccinator m.





Infection

Contents

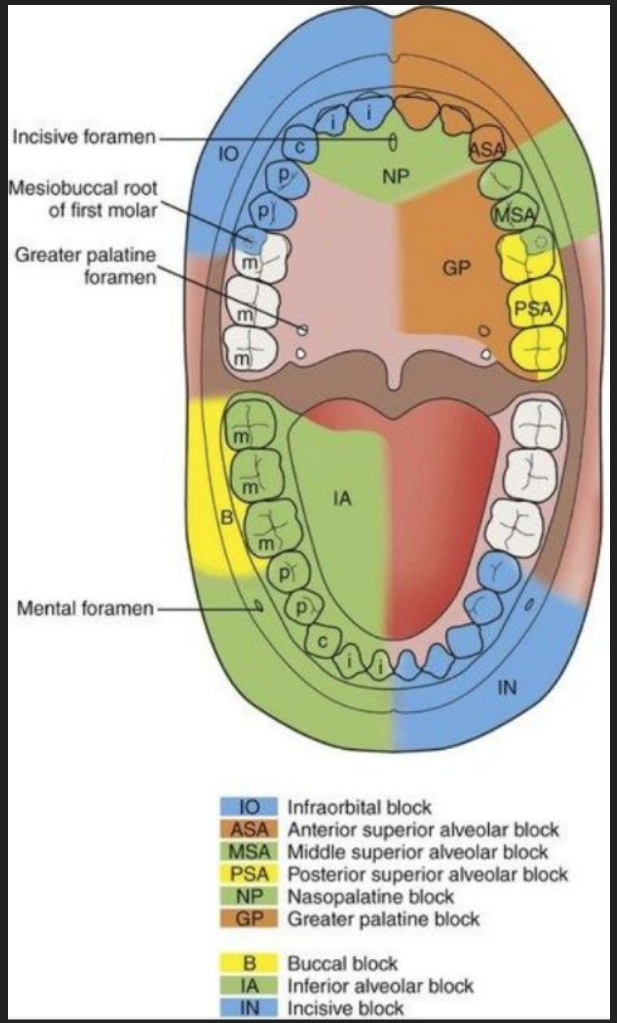
1. Inferior alveolar nerve, artery & vein.
2. Lingual Nerve
3. Sphenomandibular ligament & fascia.
4. Nerve to mylohyoid

Boundaries

- Anteriorly — Buccal space
- Posteriorly — Parotid gland & lateral phayngeal space.
- Superiorly — Lateral pterygoid muscle.
- Inferiorly — Inferior border of mandible
- Superficial & medial — lateral surface of medial pterygoid muscle
- Deep & lateral — medial surface of ramus of mandible.

Communications

1. Anteriorly — Buccal Space.
2. Posteriorly — Parotid space.
3. Medially — lateral phayngeal space & Peri[illegible] space
4. laterally — Submasseteric space.
5. Superiorly — Deep temporal space & Infratemporal [illegible]
6. Inferiorly — Submandibular spaces.


Incisive foramen
Mesiobuccal root of first molar
Greater palatine foramen
Mental foramen
IO
ASA
NP
MSA
GP
PSA
IA
B
IN
i
c
p
m
IO Infraorbital block
ASA Anterior superior alveolar block
MSA Middle superior alveolar block
PSA Posterior superior alveolar block
NP Nasopalatine block
GP Greater palatine block
B Buccal block
IA Inferior alveolar block
IN Incisive block

Components of LA

Methylparabens

Bisulphite

Sodium chloride

Sodium hydroxide

Distilled water

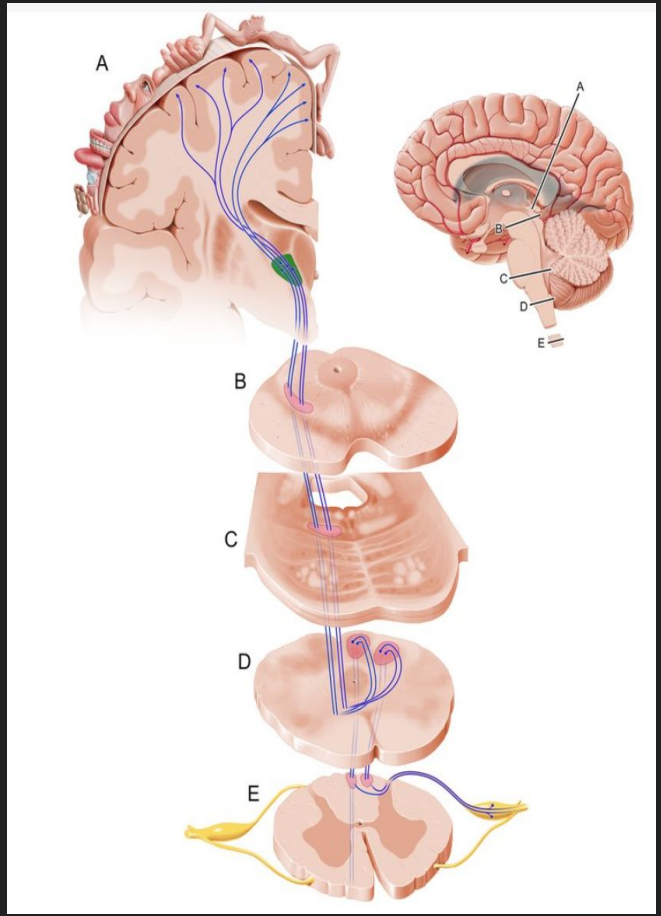
**Important to the function/role of each for exams

Anatomy (pathways)

Spinothalamic

Pain, Temp and Crude Touch.

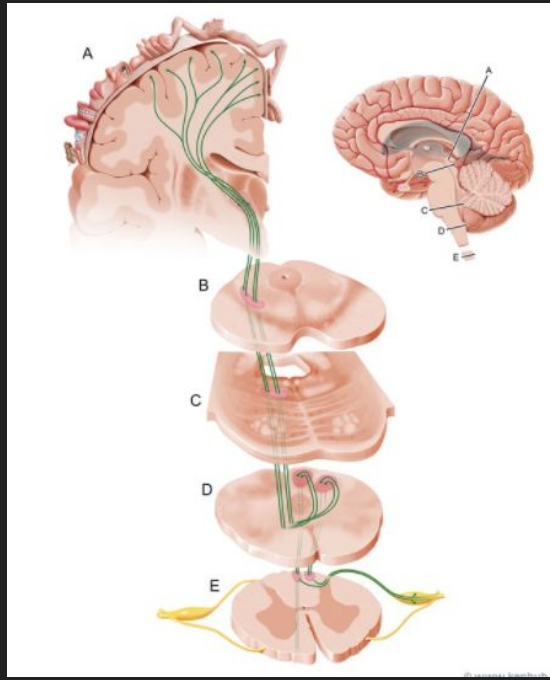
First order neuron	Pseudounipolar neurons within the dorsal root ganglion
Second order neuron	Substantia gelatinosa of Rolando Nucleus proprius - Send afferents to thalamus via Lissauer's tract
Third order neuron	Thalamic nuclei: ventral posterior lateral, ventral medial posterior, medial dorsal - Send afferents to primary sensory cortex (postcentral gyrus) via corona radiata



Dorsal Column Pathway

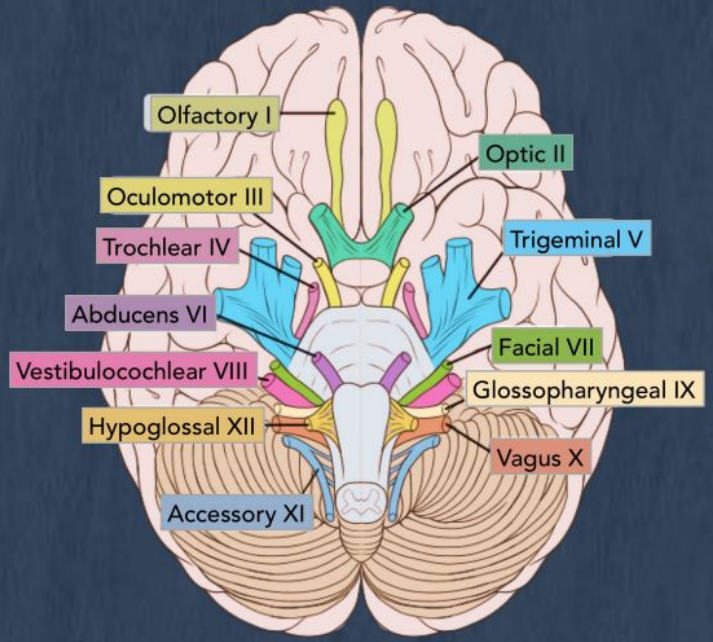
Fine touch, vibration.

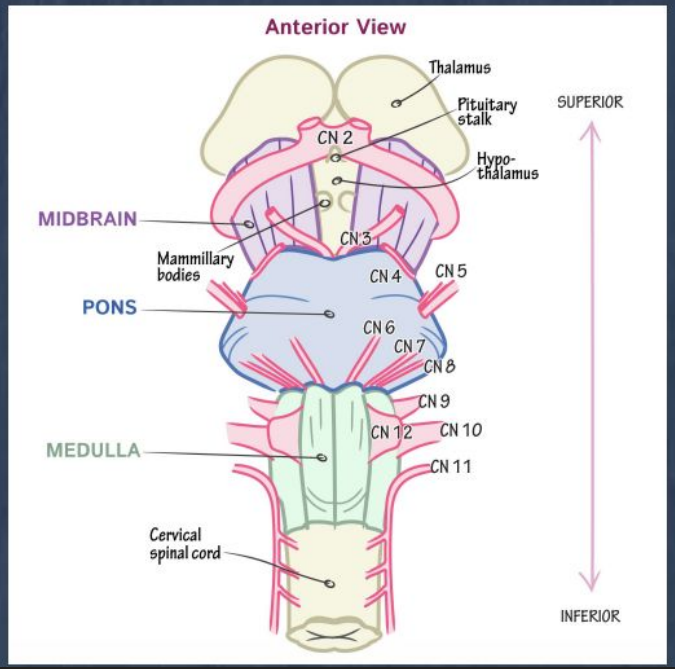
Dorsal column	Sensory pathway that receives information from receptors in the skin and joints. Nerve tracts in the white matter of the dorsal columns of the spinal cord (first-order neurons) carries this information to the medulla
Medial lemniscus	Continuation of the dorsal column, this pathway starts within the brainstem, after the decussation of internal arcuate fibers (second-order neurons). Sends sensory input to the thalamus and postcentral gyrus, where the information is decoded.



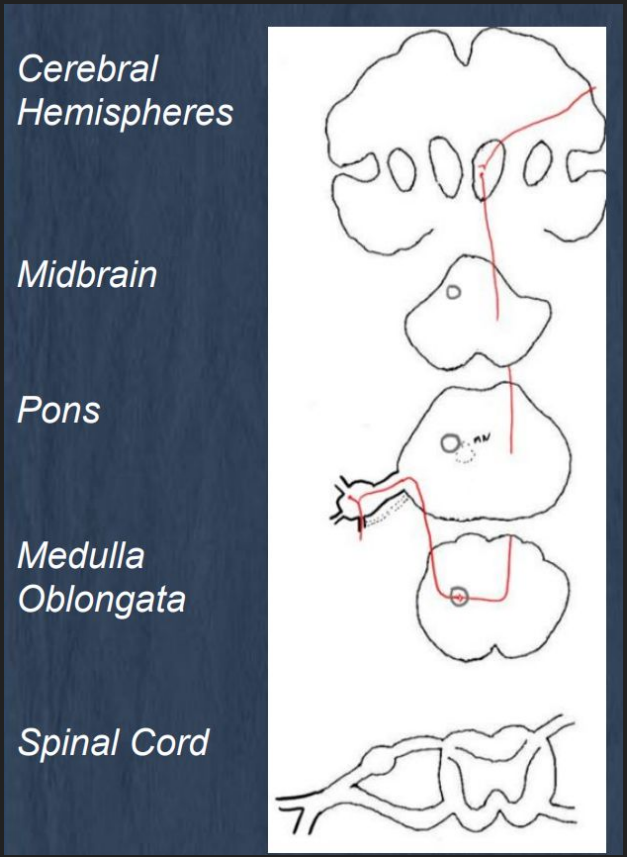
2 + 2 + 4 + 4

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





Trigeminal Lemniscus Pathway crude touch,pain pathway



Afferent neuron → Trigeminal ganglion.

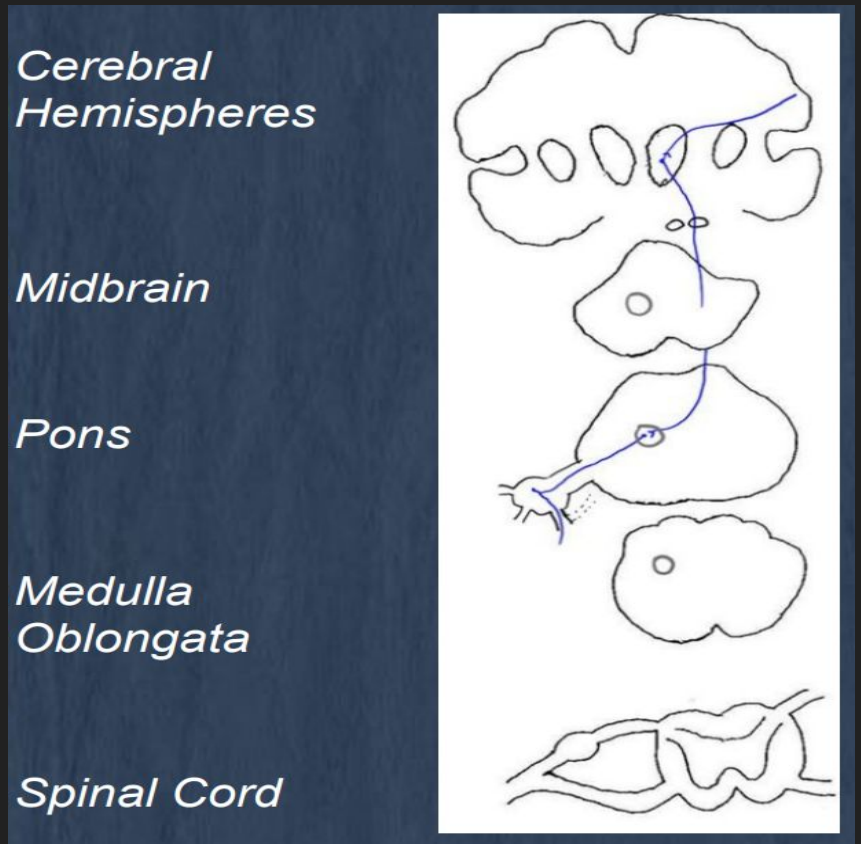
Descends to the spinal nucleus

Decussates and ascends to the Medulla

Synapse Thalamus

Sensory Cortex ie Postcentral gyrus

Fine Touch Dental pathway



Afferent axons enter trigeminal ganglion → into pons.

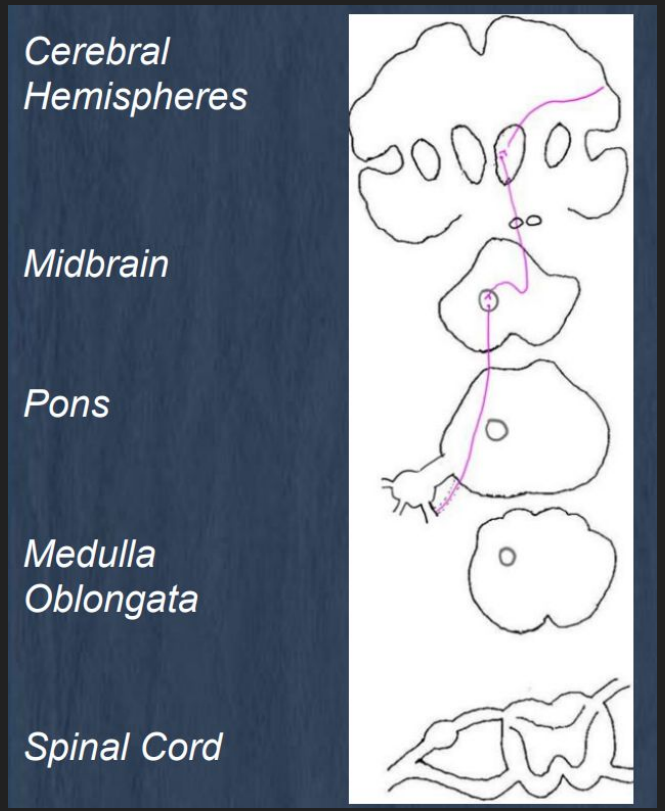
Synapse at the pontine nucleus

Most fibres decussate and ascende the contralateral Trigeminal Lemniscus

Synapse at the Thalamus

Postcentral gyrus

Proprioception Dental pathway



Afferent axon enters the trigeminal nucleus into the **midbrain.**

Synapses at the **Mesencephalic** nucleus

Decussates ascends Trigeminal Lemniscus

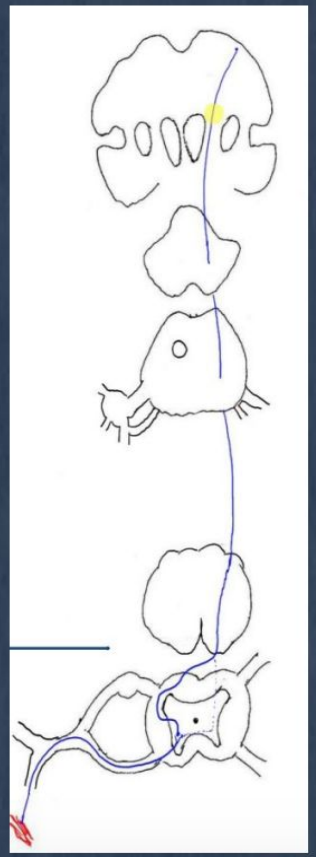
Synapse at the thalamus

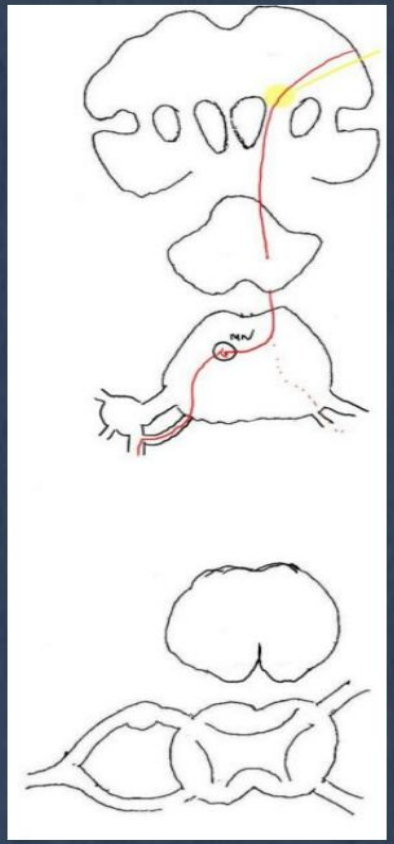
Postcentral gyrus

Descending pathways

Pathways	Corticobulbar (general body)	Corticonuclear (dental)
Upper Motor Neuron	Originates from precentral gyrus, travels through brainstem - Crosses at ‘pyramid’ - Synapse at spinal cord ventral horn	Originates from precentral gyrus, travels through brainstem - Crosses at Pons - Synapse at Motor Nucleus
Lower motor neuron	Innervates limb muscles	Passes through motor root to associated orofacial muscles

Descending Pathways





Motor Neuron Defects

Symptoms	Upper MN Defect	Lower MN Defect
Atrophy	No	Yes
Paralysis,	Spastic	Flaccid
Fasciculations and Fibrillations	No	Yes
Reflexia	Hyper	Hypo

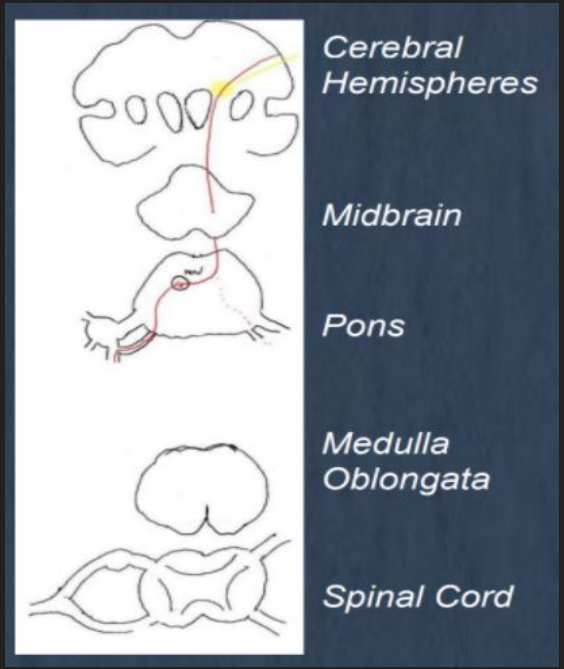
Example of Motor Neuron Defect

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.

Interpratation

Entirety of RHS is defective → ∴ LHS UMN defect above the level of the decussation → affecting the corticospinal tract. LHS Eyelid → LMN defect → LHS CN3 → ie deviation of the eye, droopy eyelid.



Symptoms	Upper MN Defect	Lower MN Defect
Atrophy	No	Yes
Paralysis,	Spastic	Flaccid
Fasciculations and Fibrillations	No	Yes
Reflexia	Hyper	Hypo

General notes

Lose marks if:

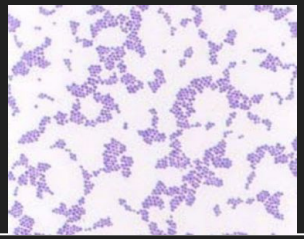
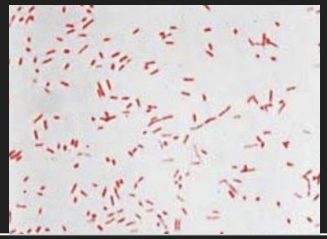
- Not indicating LHS or RHS
- Use abbreviations i.e. SCM or LA
- Not specific enough when describing an image
  - Include LHS or RHS
  - State if intraoral photo/extraoral photo
  - What structures do you see
- Explain your depth of knowledge adv vs disadv include why
- It might say to specifically describe one area not the whole dentition e.g. only describe Q3 in an intraoral photo

Microbiology

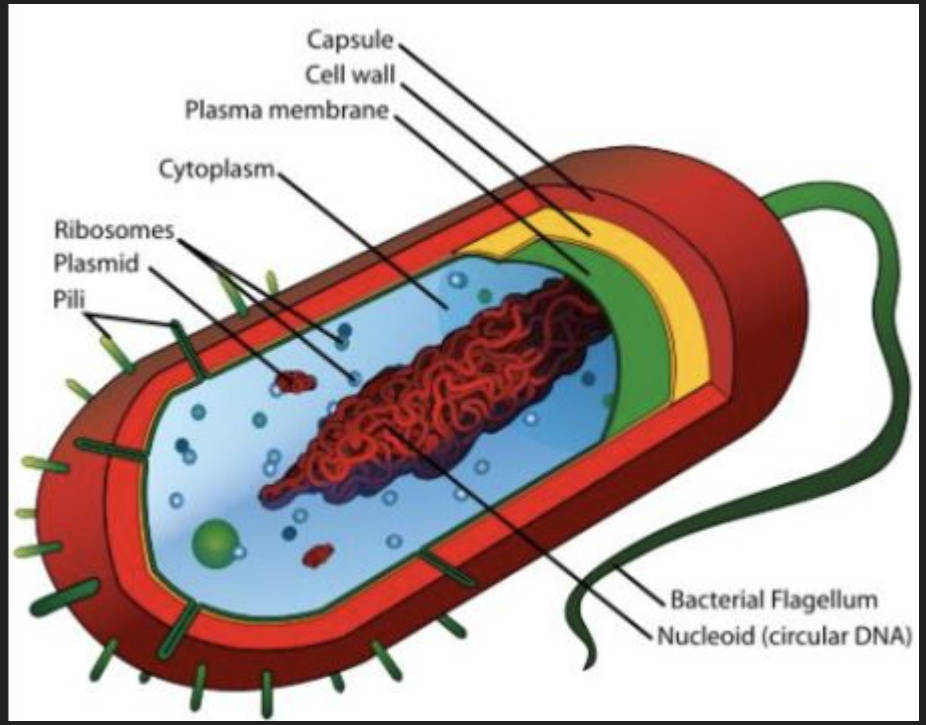
Eukaryotes vs Prokaryotes

Eukaryotes	Prokaryotes
- Membrane-bound nucleus and organelles - Cell membrane - Chloroplasts in plant eukaryotes - Multiple linear chromosomes	- No nucleus or membrane-bound organelles - Cell membrane and wall - Single circular chromosomes
- Protozoa, fungi, algae	- Bacteria, archaea

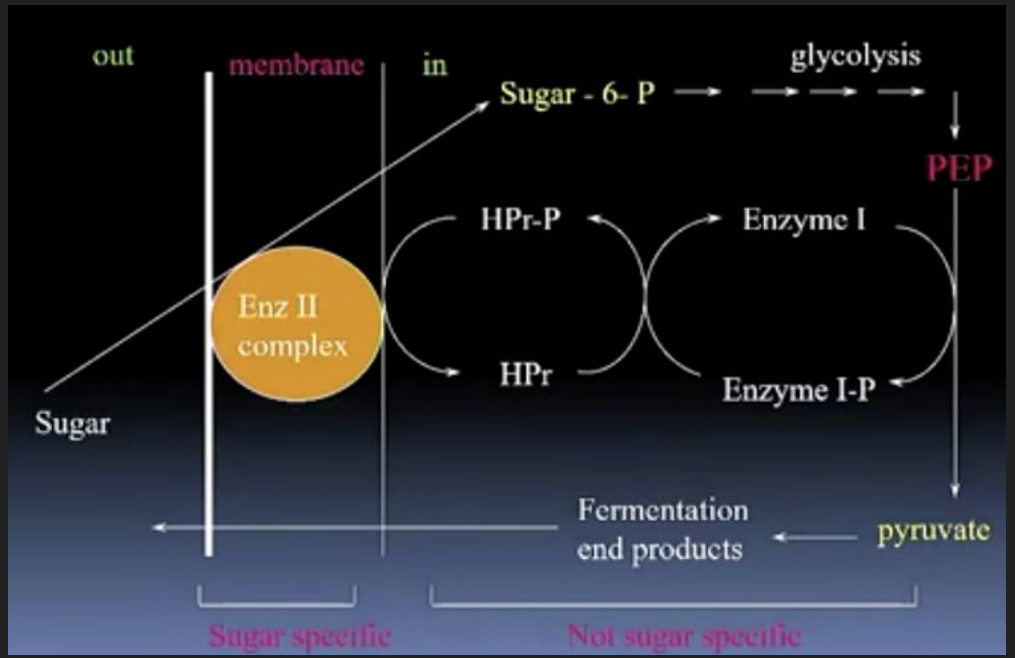
Gram Staining and Structure

Gram +ve	Gram -ve
- Thick peptidoglycan layer - Affected by lysozome and penicillin - Does not decolourise readily 	- Dual membrane, thin peptidoglycan - Lysozyme and penicillin ineffective - Decolourises readily 

Bacterial structures

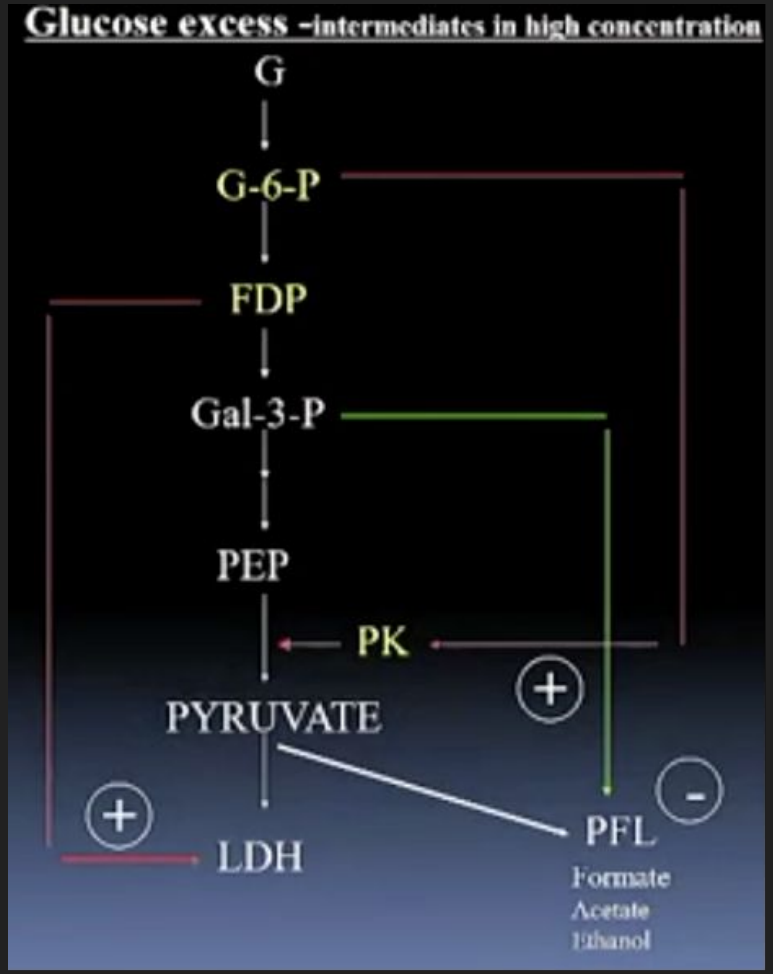


PEP-PTS

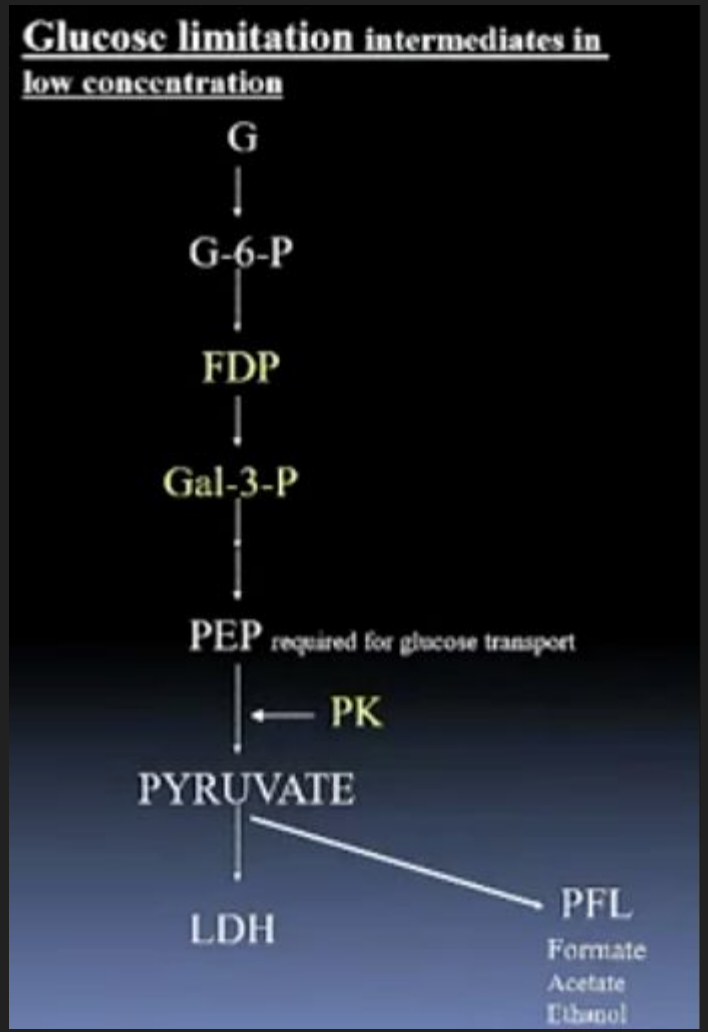


1. Uptake of sugar and phosphorylation
2. Glycolysis ⟶ PEP
3. PEP ⟶ pyruvate donates phosphate to Enz II
4. Uptake of more glucose
5. Pyruvate ⟶ lactic acid

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

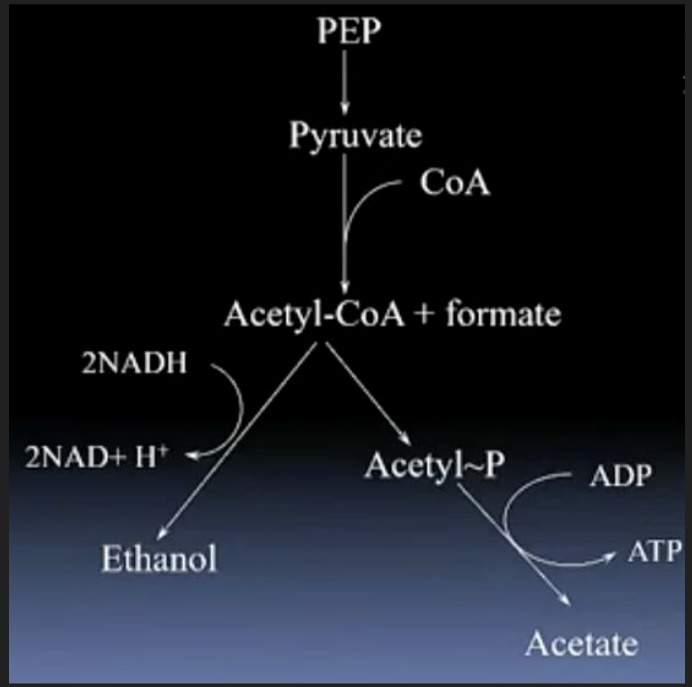


1. Low G-6-P, FDP, Gal-3-P
2. Reduced LDH, build up of PEP
3. PEP used in PEP-PTS system



Fluoride and 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

1. Attachment to host
2. Invasion of host
3. Growth and colonisation
4. Evasion
   - Exotoxins, endotoxins, quorum sensing

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

Identification
1, Catalase test
2. Coagulase test

Properties

- Highly resistant to environmental stress
- Localised, inflamed, pyogenic

Virulence
1. Attachment - adhesins, protein A
2. Growth - Coagulase
3. Evasion - Capsule, peptidoglycan

Direct damage
- 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

GENERAL PATHOLOGY

Inflammation

- Local physiological response to tissue injuries

1. Acute
2. Chronic
3. Healing and repair

Why inflammation?

- Protective mechanism

BUT

- Causes surrounding tissue destruction

Inflammation

Acute:

- Initial reaction to injury
- Causes:
  - Microbial infection → bacteria, viruses, fungi
  - Hypersensitivity → immunological reaction
  - Physical stimuli → trauma, radiation, temperature
  - Chemical stimuli → alkaline, corrosion, irritants
  - Necrosis
- Signs:
  - Redness → vasodilation, increase blood flow for nutrients and WBCs to site of infection
  - Heat → greater flow of blood into area - heat
  - Swelling → more fluid in the area
  - Pain → pressure from the swelling stimulating nociceptors
  - Loss of function → physical limitation due to swelling

Inflammation

Chronic:

- Persistence of agent causing inflammation
- May occur following a period of acute inflammation

Causes:

- Resistance of infective agents to phagocytosis and intracellular killing
- Endogenous, exogenous materials
- Autoimmune disease
- Recurrent acute inflammation

Signs:

- Macroscopic
  - chronic ulcer, chronic abscess, fibrosis
- Microscopic
  - Presence of lymphocytes, neutrophils, granulomas, necrosis, cycles of tissue repair and destruction

Wound healing

- 3 potential results from acute inflammation
  - Regeneration - best outcome, not common (esp in specialised tissues)
  - Healing by repair - most common outcome. Resolution of inflammation, not functional but structural protective scar tissues, not complete restoration of tissue
  - Chronic inflammation - Worst outcome, injury persists, unable to fight infection
- Factors impeding healing:
  - Foreign materials
  - Necrotic tissues
  - Ischemia
  - Wound tension

Stages of wound healing

INFLAMMATORY STAGE

- Vascular and cellular events of acute inflammation
- 3 to 5 days

FIBROBLASTIC STAGE

- 2 to 3 weeks
- Protective fibrin network formed from coagulation
- Fibroblasts produce *ground substances*, *fibronectin*, and *tropocollagen* in fibrin network
  - Ground substance → glycoproteins and proteoglycans that form stromal matrix
  - Fibronectin → stablises local fiber network, chemotactic activity, helps immune system recognise foreign bodies
  - Tropocollagen → molecular constituent of collagen fibrils, which will form collagen
- Collagen fibrils are initially randomly oriented resulting in low strength
- High degree of vascularisatoin - fibrovascular granulation tissue
- Wound can withstand 30-40% of tolerable tension

REMODELLING STAGE

- About 3 months post injury
- Wound maturation - wound metabolism & vascularity decreases
- Replacement of randomly oriented collagen fibrils by fewer more ordered ones - improve tensile resistance
- Wound contraction
- 80-85% of tolerable strength

Healing by Primary, Secondary & Tertiary intention

Primary:

- Occurs in small wounds, minimal injury and no tissue loss
- Edges of wound are in normal anatomical position

Secondary:

- Tissue loss preventing close apposition of wound edges
- Slower healing, greater degree of scarring
- Significant epithelial migration, collagen deposition, wound contraction, remodelling

Tertiary:

- Healing of wounds via use of tissue grafts, in large wounds

Healing of extraction sockets

- Secondary intention
- Complete healing → 12 months

Immediate post-op:

- Socket fills with blood - vasculature from periodontium, local alveolar bone etc
- Blood coagulates - clot protects from oral environment, coagulation occurs

1 Week:

- Inflammatory stage
- Neutrophils remove necrotic microbes and bone
- Socket is protected by blood clot, fibrin

End of 1 Week:

- Fibroblastic stage starts → fibroblasts + capillaries forming granulation tissue
- Epithelial migration from gingival margin of the socket, down the socket wall, and over granulation tissue
- Osteoclasts begin remodelling → resorption of bony leftovers

Healing of extraction sockets

2 weeks

- Granulation tissue fills socket
- Osteoid deposition by osteoblasts, along alveolar bone lining socket
- Primary closure over smaller sockets - full epithelial coverage, soft tissue healing

3-4 weeks

- Primary closure everywhere
- Osteoclasts continues to resorb cortical bone
- Formation of new trabecular bone in the socket

4-6 months

- Complete resorption of cortical bone and lamina dura
- New bone is filling the socket
- Covering epithelium rises to same level as adjacent gingiv

12 months

- Complete osseous filling of the socket, with scar tissue

Tooth socket complications

Alveolar Osteitis

- Dry socket
- Blood clot lost prematurely, leading to an empty socket
- Leads to delayed healing, and pain
- Nicotine in cigarettes is a vasoconstrictor, have to inform patients to avoid smoking post extraction
- Irrigate socket with chlorhexadine, pack with palliative dressing material

Post-op infection

- Occurs in immunocompromised patients
- Need to prescribe antibiotics

PIA

Management plan

Scaffold - always answer in a systematic approach

CC

Further information/additional tests

Patient education - diagnosis, aetiology, consequences, treatment options

Informed consent - risk, benefits, costs, appointment sequences

Emergency and stablisation - get the patient out of pain

Disease control and prevention - OHI, diet changes etc

Immediate rehabilitation - restorative, periodontal care

Short term review - 3 months?

Long term review - 6/12 months?

Restorative steps

Difference between Amalgam, CR and GIC/RMGIC

CR example 46 MO:

- Gain patient informed consent
- Apply topical on RHS pterygotemporal depression, followed by RHS inferior nerve block
- Shade match with natural lighting used
- Place RD, W3/5 clamp, isolating 47-44
- Acces caries through enamel with HS 822 bur with water spray
- Clean out caries with SS appropriate sized round bur along the DEJ
- Check preparation for remaining infected dentine
- Condition dentine with 20% polyacrylic acid for 10 seconds, wash for 20 seconds, dry, do not dessicate
- Place FUJI LC liner on dentine only, LC 20 seconds
- Etch enamel with 37% phosphoric acid 10-15 seconds, wash 30 secs and dry
- Place unfilled resin, LC
- Place sectional matrix interproximally and WEDGE TIGHTLY, check contacts
- Place composite resin in 2mm increments, LC
- Place sufficient CR, remove excess material with appropriate burs, remove sectional matrix and wedge
- Check margins with explorer, contacts and interproximal for overhangs
- Remove RD, check occlusion, final polish and provide post op instruction and dismiss patient

General notes

- Have to indicate RHS or LHS - can lose marks
- Do not use abbreviations
- When describing images
  - intra/extra oral
  - What structures do you see
- Explain your depth of knowledge
  - Advantages, disadvantages
- Read the question carefully!!!
  - If only ask to describe q3, stick to that, don’t waste time describing the whole dentition

Not covered

Bacterial genetic transfer mechanisms - transformation, conjugation, transduction

Disinfection and sterilisation

TB