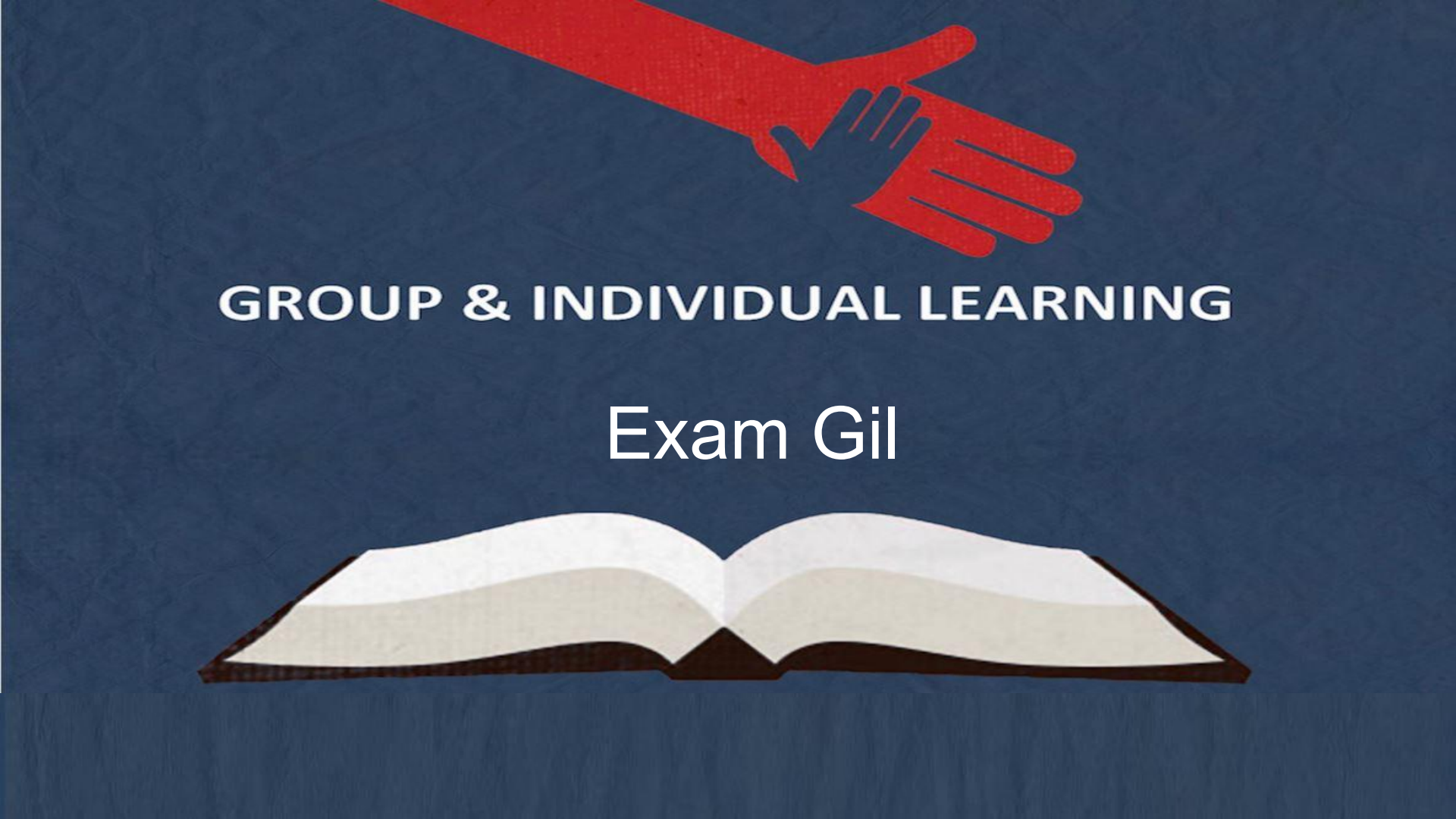

GROUP & INDIVIDUAL LEARNING
Exam Gil

Host and Sponsors

wright evans partners

ADA SOUTH AUSTRALIA

Immunology

Tolerance

Immune tolerance - a lack of response to self antigens and harmless external antigens (commensal bacteria, pollen, foods, presence of developing foetus) resulting from exposure of these antigens to lymphocytes

Central tolerance - exposure of developing lymphocytes to self antigens in central lymphoid organs (mainly the bone marrow and thymus)

- T-cells: Immature CD4+ and CD8+ lymphocytes bind respectively to class II and class I MHC molecule -> **apoptosis**
- CD4+ T cells: recognition of self antigens -> generation of T reg cells -> **enter peripheral tissues**

Peripheral tolerance - Response to recognition of self antigens by mature T cells

- **Clonal anergy** (functional inactivation): recognition of antigens without adequate costimulation (APCs) -> TCRs lose ability to transmit activating signals -> long-lived functional unresponsiveness
- **Immunosuppression:** mediated by T reg cells -> suppress activation of T cells
  - CD4+ T reg cells -> role in suppression of autoimmune diseases
  - CD8+ suppressor cells -> role in suppression of graft rejection
- **Apoptosis (deletion)**
  - Production of apoptotic proteins in T cells -> induce cell death by mitochondrial pathway -> activation of caspases and cytosolic enzymes -> induce apoptosis
  - Death receptor pathway - coexpression of death receptors and ligands -> activation of caspases -> apoptosis

Autoimmunity

The immune response against self antigens due to a failure of tolerance.

Principle factors in the development of autoimmunity:

- Inheritance of susceptibility genes
- Environmental triggers (i.e infections)

Autoimmune diseases: systemic lupus erythematosus, rheumatoid arthritis, diabetes mellitus, multiple sclerosis, pemphigus vulgaris, Sjogren’s syndrome

Hypersensitivity

Hypersensitivity - pathological immune responses capable of causing tissue injury and disease
Atopy - tendency for an individual to have allergic manifestations (more IgE in serum than others)

Type of hypersensitivity	Pathologic immune mechanism	Examples
Immediate hypersensitivity (type I)	Th2 cells, igE antibodies, mast cells, eosinophils	Anaphylaxis, latex allergy, asthma
Antibody mediated disease (type II)	Cytotoxic response mediated by IgM/IgG/complement	RHD, anaemia, myasthenia gravis
Immune complex mediated disease (Type III)	Involvement by complement, igG and antigens	RA, SLE (lupus)
Delayed-hypersensitivity (Type IV)	Cell mediated (Th1 cells, macrophaegs, cytokines)	Contact dermatitis, chronic transplant rejection, MS

Latex Hypersensitivity

Irritative dermatitis

- Non immunological
- Most common
- Dryness or irritation
- Contact with natural latex rubber

Type I

- Immediate
- Immunological
- Most serious/anaphylaxis possible
- Reaction to plant protein

Type IV

- Delayed
- Immunological
- Localised
- Reaction against chemicals in product manufacture (powder)

Cross reactive substances

Banana, Avocado, Nuts, Kiwi (BANK) and tomato and potato lol

Management - this was a question in the BDS3 exam

- Book patient in the morning as there are fewer latex particles in the environment
- Ask if they have epipen (if anaphylactic) at the beginning of the appointment -> if they have it with them ask where they keep it and ask if you have permission to use it in the case of an emergency and they are not able to self administer
- Avoid the use of latex products (i.e latex gloves, latex rubber dam, be cautious of latex stoppers in rubber dams, potential cross-reactivity with the rubber in GP points?)
- Recognise signs and symptoms of allergy reaction onset, especially anaphylaxis e.g urticaria, angioedema
- Cease dental treatment immediately, remove allergen, call 000, administer EpiPen if severe anaphylaxis

Rheumatic heart disease

Type II autoimmune disease (antibody mediated hypersensitivity)

- Aetiology based on “molecular mimicry”
  - Antigens may have a very similar structure to host cells -> cross-reactivity
- In rheumatic heart disease, M protein on the bacterial cell wall (especially s.pyogenes) is similar in structure to the hearts myosin cells resulting in cross reactivity
- The body’s immune response attacks the cardiac cells instead of the bacteria (autoimmune attack on host cells) resulting in the breakdown of cardiac tissue
- This may result in scarring of the heart valves and an increased risk of endocarditis

PCC/VPP

PCC

As with volunteer patients, scenarios can come up in exams.
Dealing with:

- Anxious patients
  - Approach?
  - Managment?

- Angry patient
  - how do you respond to them?
    - DEFUSE

Microbiology

Cellular components

Structure	Chemical Composition	Function
Cell wall		Cell shape and protection of cytoplasm
Cell wall in gram +ve	Mainly peptidoglycan and teichoic acid	
Gram –ve	Thin layer of peptidoglycan, multilayered: outer membrane – lipopolysaccharide	
Cytoplasmic membrane	Phospholipid bilayer containing globular proteins	Controls movement of solutes into and out of cell -site of respiratory enzymes, DNA and cell wall synthesis, secretion
Extracellular polymers	Single and mixed polysaccharides	Adherence to substrates, inhibition of phagocytosis
Flagella	Protein	Movement
Pili	Protein	Conjugation (Gram –ve, used as penis essentially) -adherence
Nucleoid	DNA	Carries hereditary information
Ribosomes	RNA and protein	Protein synthesis
Inclusion bodies		
Glycogen	Polysaccharide (glucose)	Energy storage
PolyOHbutyrate	Lipid	Energy storage
Endospores	Dehydrated form of vegetative cell	Bacteria enter this state in times of high stress to have protection from the environment

Strep vs Staph

Characteristic	Staphylococcus	Streptococcus
Shape	grape like clusters -growth occurs in multiple axis	pairs or chains -growth occurs in single axis
Catalase Test	positive	negative
Haemolysis	none or beta haemolysis	alpha (green) or beta (complete) or gamma (none) haem -can be used to differentiate species of strep
Important members	staph aureus	s. mutans s. pyogenes s. rattus
Useful diagnostic Test	catalase coagulase blood agar	

G+ve vs G-ve

Properties	G+ve	G-ve
Thickness of cell wall	Thicker -20-25nm -one layer	Thinner -11-15nm -two layered
Gram reaction	Stains blue/violet/purple	Stains pink/red
LPS layer	Absent	Present
Peptidoglycan Content	High -penicillin prevents NAM-NAG link in peptidoglycans forming (beta 1-4 linkage)	Low
Teichoic acids	Present	Absent
Toxins produced	Exotoxin	Endotoxin
Lipid content	Low	High
Action of lysozyme	Digests peptidoglycan layer hence easily destroyed	Cannot penetrate LPS layer
Antibiotics	More susceptible	More resistant

Mechanisms of antibiotics

- Disrupt cell membrane function
- Inhibits cell wall synthesis
  - Targets peptidoglycans
  - Requires cells to be growing
  - Includes B-lactams, penicillin, cephalosporins
- Inhibit DNA/RNA synthesis
- Inhibit protein synthesis
- Inhibition of folic acid metabolism

B-lactamases

-B-lactamase is an enzyme that breaks down the B-lactam ring
-Gives rise to antibiotic resistance
-Is transferable among different species within a biofilm

Metabolism

-PEP PTS system
-Glucose Permease
-Effect of F on metabolism of sugars

PEP-PTS system

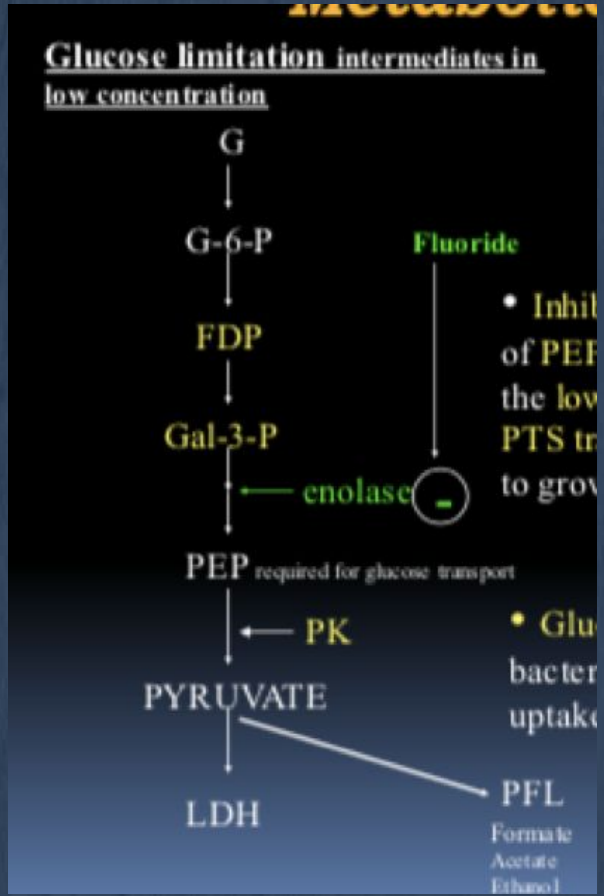
-High affinity transport system
-in acidogenic oral bacteria (strep, lacto)
-can move sugars in when concentrations are low
-optimal under lower conc, neutral pH and slow rates of bacterial growth
-repressed under conditions of excess sugar, low pH and high growth rates

Glucose Permease Transport

-ATP dependent
-Functions at high growth rate and low pH
-has low affinity meaning it moves large amounts in when the concentration is high

Fluoride on Metabolism of sugars

-Inhibits enolase and reduces production of PEP
-low levels of PEP means reduced effectiveness of PEP-PTS system
-means bacteria cannot grow



General Pathology (Ying Guo)

Inflammation

- Acute Inflammation:
  - Causes:
    - Microbial infection → bacteria, viruses, fungi
    - Hypersensitivity → excessive immunological reaction
    - Physical stimuli → trauma, radiation, temperature
    - Chemical stimuli → alkaline, corrosion, irritants
    - Necrosis → ischemia = infarction = inflammation
  - Signs
    - Redness – the injured areas have local vascular dilation to increase blood flow (so that nutrients and WBCs can be brought to site of infection)
    - Heat – greater flow of blood in the area = heat
    - Swelling – more fluid in the area = will swell
    - Pain – Pressure from the local swelling will stimulate nociceptors; proinflammatory mediators will further stimulate nociceptors
    - Loss of function – physical limitation due to swelling; pain will prevent from being used

Inflammation

- Chronic Inflammation
  - Causes:
    - Primary chronic inflammation → persistent infective agents, endogenous materials, exogenous materials, autoimmune diseases, idiopathic chronic inflammatory diseases
    - Transplant rejection
    - Progression from acute inflammation
    - Recurrent acute inflammation
  - Signs:
    - Macroscopic: chronic ulcer, chronic abscess, fibrosis
    - Microscopic:
      - Lymphocytes, plasma cells, macrophages
      - Neutrophils scarce
      - Granulomas
      - Necrosis
      - Cycles of tissue repair and destruction

Wound Healing

- Three potential results of acute inflammation:
  - Regeneration
    - Best outcome, not common (especially in specialized tissues)
  - Healing by repair
    - Most common outcome
    - Will get resolution of inflammation over time, but not complete restoration of tissue
    - = non-functional, but structurally protective scar tissue in area of damage
  - Chronic inflammation
    - Worst outcome
    - Injury persists, and antibodies are unable to fight infection = chronic inflammation
    - Once resolved, will lead to healing by repair
- Factors impeding healing:
  - Foreign materials
  - Necrotic tissue
  - Ischemia
  - Wound tension

Stages of Healing

- Inflammatory Stage
  - Vascular and cellular events of acute inflammation occur
  - 3 to 5 days
- Fibroblastic Stage
  - 2 to 3 weeks
  - Protective fibrin network formed from coagulation
  - Fibroblasts produce ground substance, fibronectin, and tropocollagen in fibrin network
    - Ground substance = glycoproteins and proteoglycans that form stromal matrix
    - Fibronectin = stabilizes local fiber network, has chemotactic activity, helps immune system recognize foregin bodies
    - Tropocollagen = molecular constituent of collagen fibrils
  - Tropocollagen forms collagen
  - Collagen fibrils are initially randomly oriented = low strength
  - High degree of vascularization = fibrovascular granulation tissue
  - Wound can withstand 30-40% of tolerable tension

Stages of Healing

- Remodelling Stage
  - Wound maturation
  - Replacement of randomly oriented collagen fibres by fewer more ordered ones = improved tensile resistance
  - Wound contraction
  - 80-85% of tolerable strength

Primary, Secondary, Tertiary Intention

- Primary intention
  - Occurs in small wounds, with minimal injury and no tissue loss
  - Edges of wound are in normal anatomical position
  - Healing occurs with minimal scarring
- Secondary Intention
  - Tissue loss preventing close apposition of wound edges
  - Slower healing, greater degree of scarring
  - Involves: significant epithelial migration, collagen deposition, wound contraction, remodelling
- Tertiary Intention
  - Healing of wounds via use of tissue grafts, in large wounds

Healing of Extraction Sockets

1. Immediately
    - Socket fills with blood → vasculature from periodontium, local alveolar bone, etc.
    - Blood coagulates → clot protects socket rom oral environment; colagulation occurs immediately after extraction
2. 1 Week
    - Inflammatory stage
    - Neutrophils remove necrotic microbes and bone
    - Socket is protected by blood clot, fibrin
3. End of 1 Week
    - Fibroblastic stage starts
    - Fibroblasts + capillaries = fibrovascular granulation tissue (protection)
    - Epithelial migration from gingival margin of socket, down socket wall, and over granulation tissue
    - Osteoclasts begin remodelling; resorption of bony leftovers

Healing of Extraction Sockets

4. 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
5. 3-4 Weeks
    - Primary closure everywhere
    - Osteoclasts continue to resorb cortical bone
    - Formation of new trabecular bone in socket
6. 4-6 Months
    - Complete resorption of cortical bone and lamina dura
    - New bone is filling socket
    - Covering epithelium rises to same level as adjacent gingiva
7. 12 months
    - Complete osseous filling of socket; radiographic evidence
    - Scar tissue

Tooth Socket Complications

- Alveolar osteitis
  - “Dry socket”
  - Blood clot is lost prematurely, leading to empty socket
  - Leads to delayed healing, and pain
  - Nicotine in cigarattes is a vasoconstricting agent, avoid smoking after extractions
  - Irrigrate socket with chlorhexidine, packwith palliative dressing material (‘alveal gel’)
- Post-op Infections
  - Occurs in immunocompromised patients
  - Need to perscribe antibiotics

LA

LA Questions

-pKa affects LA diffusion, how?

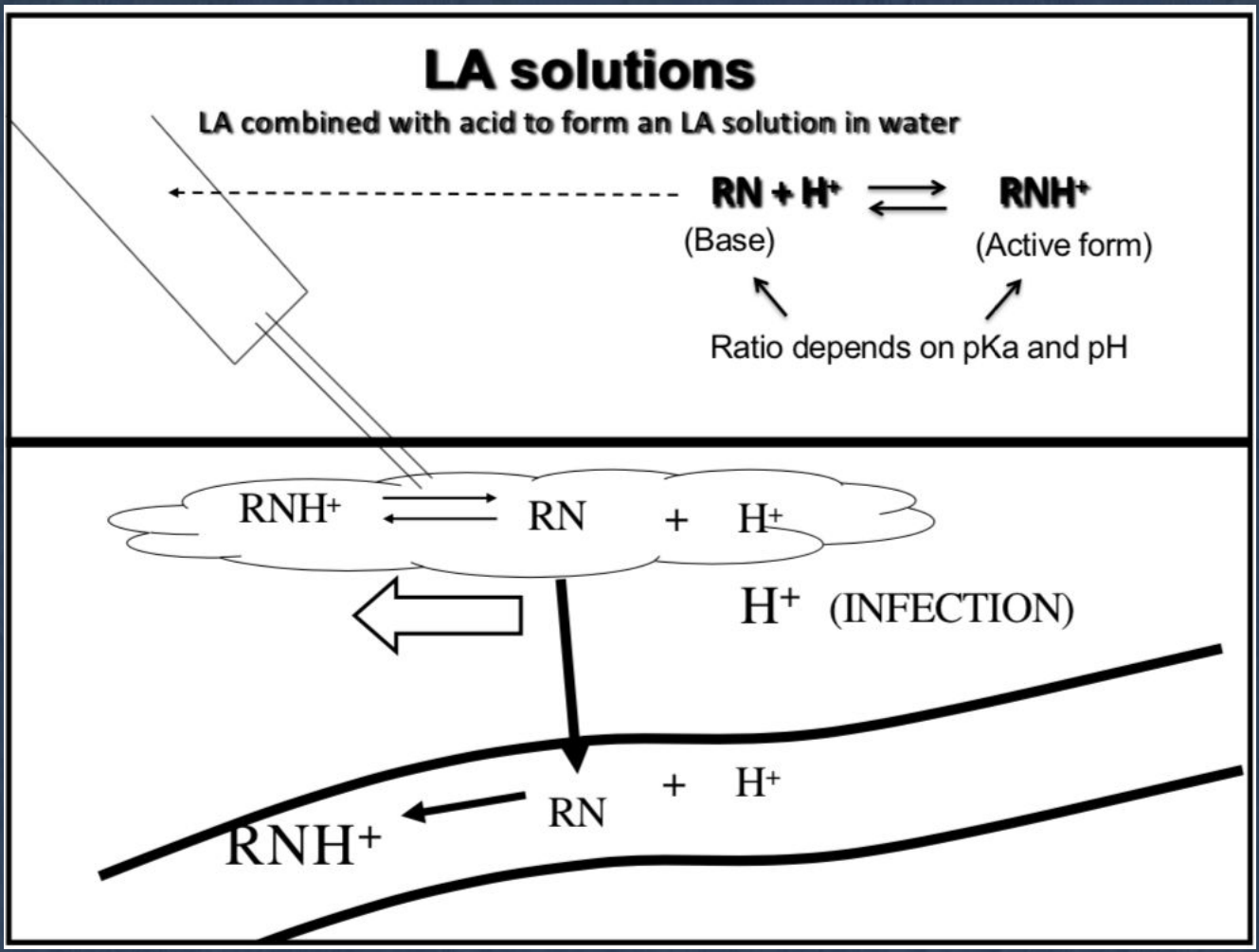
-Why is it difficult to anaesthetise an area with inflammation?

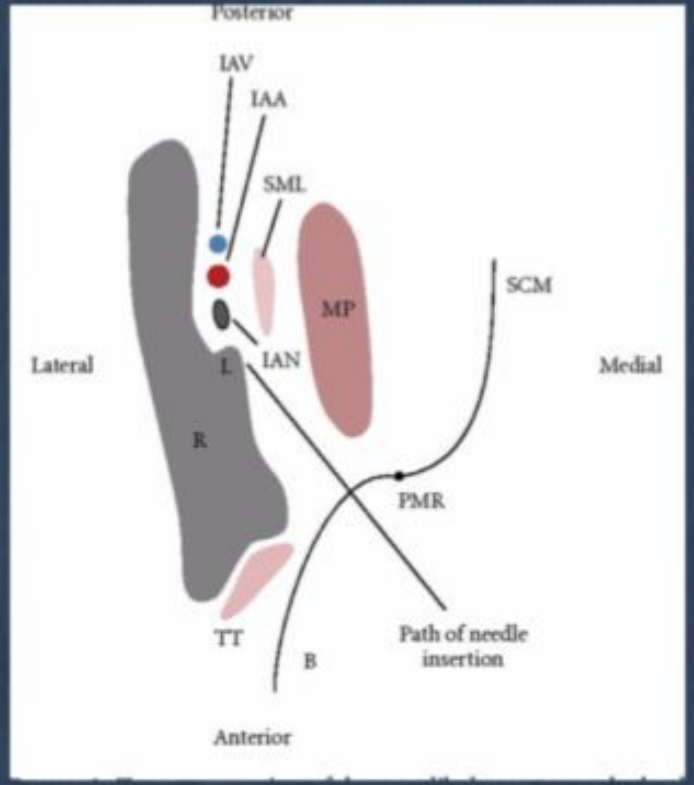
- what are some reasons for LA failure

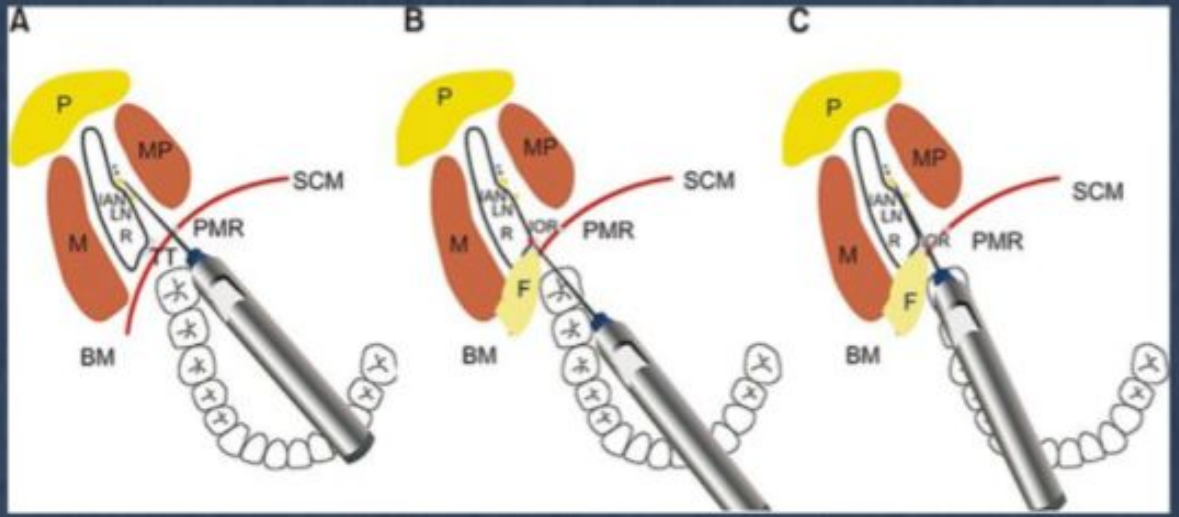
- is the landmark correct? if not, draw correct location

LA solutions

LA combined with acid to form an LA solution in water




Posterior
IAV
IAA
SML
MP
SCM
Lateral
L
IAN
Medial
R
PMR
TT
Path of needle insertion
B
Anterior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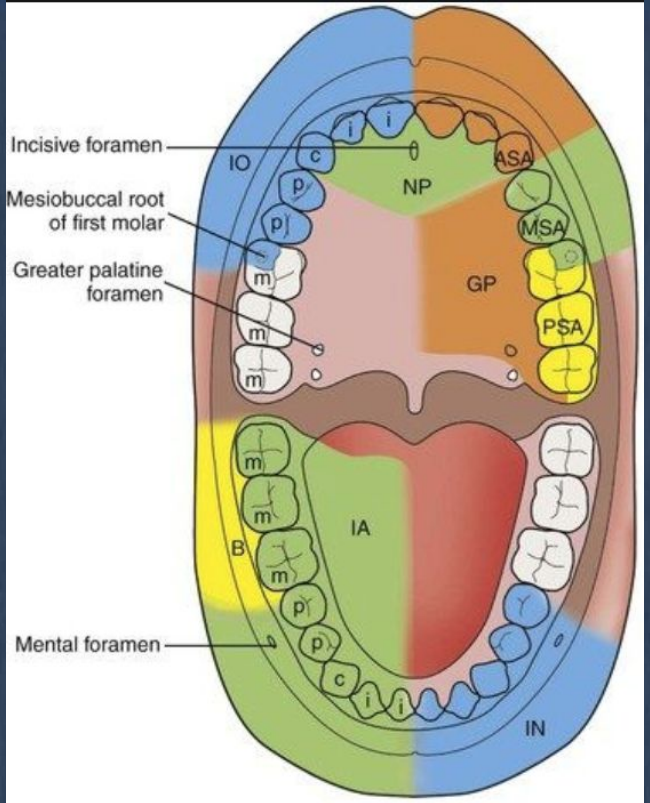


IO	Infraorbital block
ASA	Anterior superior alveolar block
MSA	Middle superior alveolar block
PSA	Posterior superior alveolar block
NP	Nasopalatine block
GP	Greater palatine block
B	Buccal block
IA	Inferior alveolar block
IN	Incisive block

Neuroanatomy

Ascending pathways of the CNS

Random terminology

Fascicle = bundle of nerves

Lemniscus = bundle of secondary nerves

Revise the scenarios in the anatomy book

General Ascending Pathways - Spinothalamic Tract

Pain, Temperature and Crude Touch

- 1st Order Neuron (ON) (Primary)

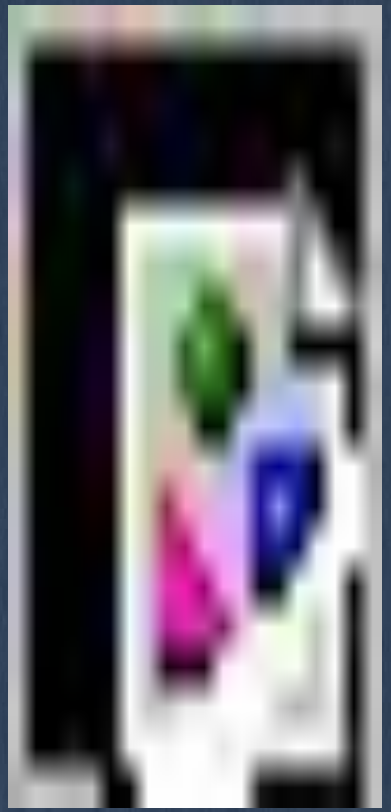
Afferent fibres (peripheral process of 1st ONs) synapse with 2nd ONs in dorsal horn spinal cord

- 2nd ON (Secondary)

Cross midline, travel through brain stem & synapse in thalamus (ventral posterolateral nucleus) (Acts as relay station)

- 3rd ON (Tertiary)

Pass through posterior limb of internal capsule, terminates at postcentral gyrus of cerebral cortex



Cerebral Hemispheres

Midbrain

Pons

Medulla Oblongata

Spinal Cord

General Ascending Pathways - Dorsal Column Pathway

Fine touch and Proprioception

- 1st Order Neuron (ON) (Primary)

Afferent fibres group together in fascicles (nerve bundles) and synapse in Medulla nuclei

Medial fasciculus gracilis (Lower body fibres)
Lateral fasciculus cuneatus (Upper body fibres)

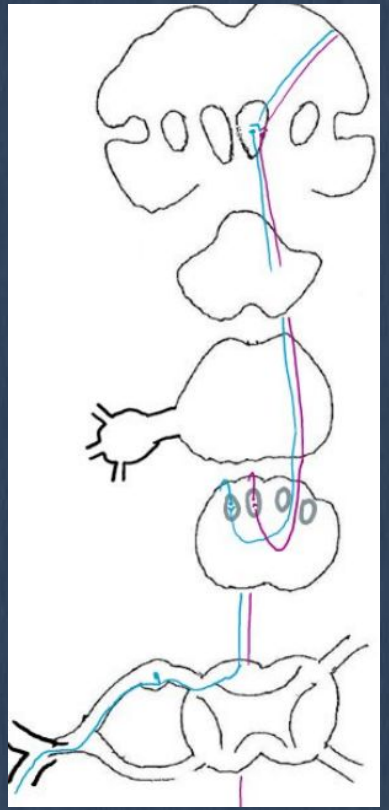
- 2nd ON (Secondary)

Sweeps downward then crosses midline, ascends brainstem

Internal arcuate fibres - formed by fibres crossing over in Medulla
Medial lemniscus - bundle of 2nd ON traveling towards thalamus

- 3rd ON (Tertiary)

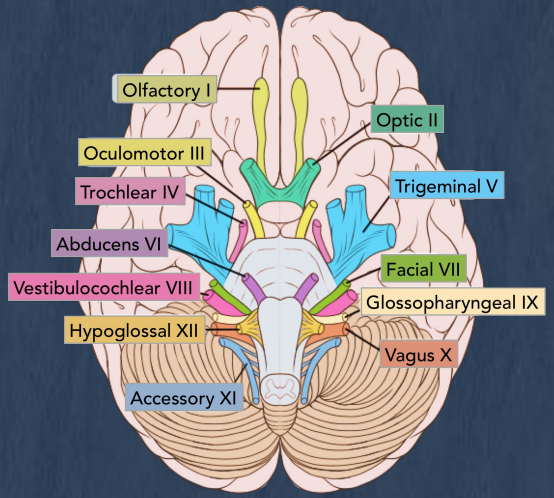
Same as Spinothalamic pathway

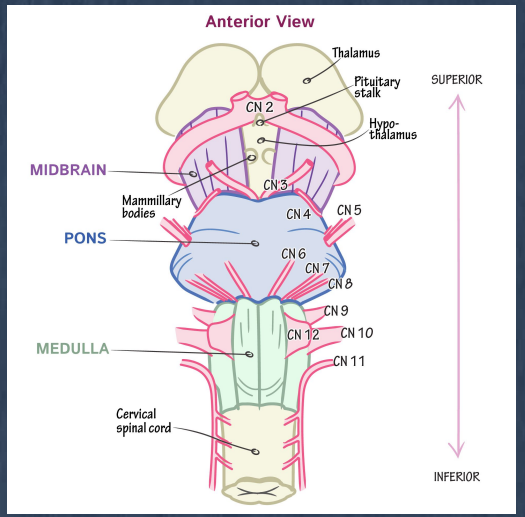


Cranial Nerves

2 + 2 + 4 + 4

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





Mandibular - Pain, Temperature & Crude Touch

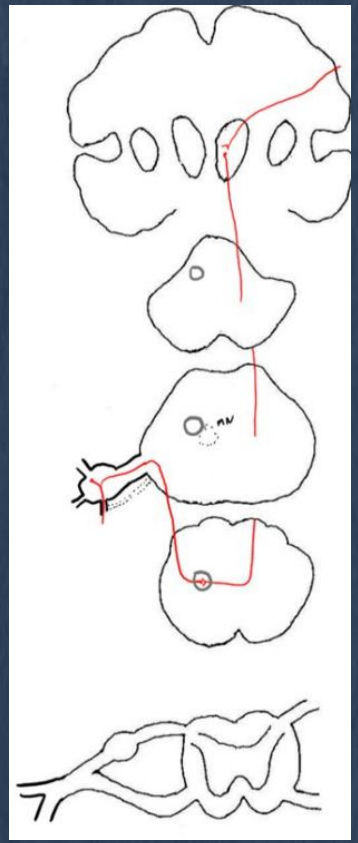
Cerebral Hemispheres

Midbrain

Pons

Medulla Oblongata

Spinal Cord



- 1st Order Neuron (ON) (Primary)

  - Cell bodies in Trigeminal Ganglion
  - Descend to Medulla
  - Synapse in Spinal Nucleus

- 2nd ON (Secondary)

  - Cross midline and ascends brainstem through Trigeminal Lemniscus
  - Synapse in Thalamus

- 3rd ON (Tertiary)

  - Passes through internal capsule to sensory cortex

Mandibular - Fine Touch

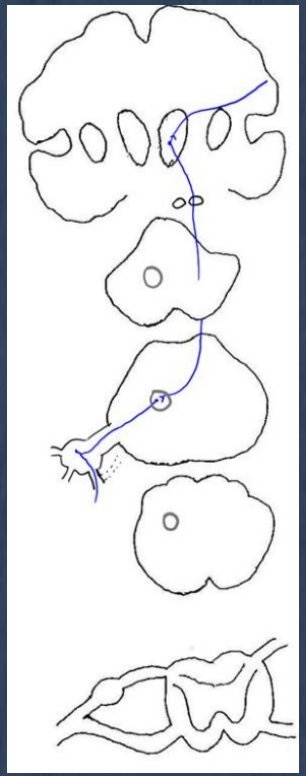
Cerebral Hemispheres

Midbrain

Pons

Medulla Oblongata

Spinal Cord



- 1st Order Neuron (ON) (Primary)
  - Cell bodies in Trigeminal Ganglion
  - Synapse in Pontine Nucleus
- 2nd ON (Secondary)
  - Some cross midline and ascends brainstem through Trigeminal Lemniscus
  - Synapse in Thalamus
- 3rd ON (Tertiary)
  - Passes through internal capsule to sensory cortex

Mandibular - Proprioception

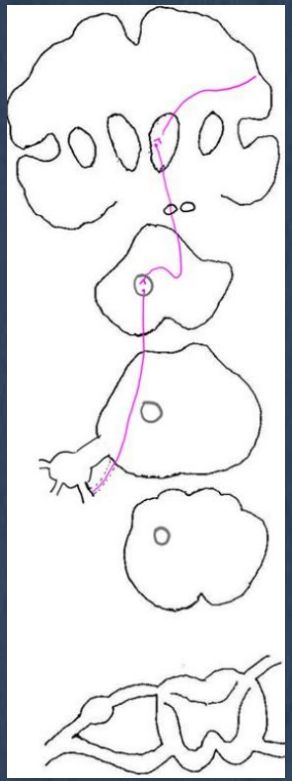
Cerebral Hemispheres

Midbrain

Pons

Medulla Oblongata

Spinal Cord



- 1st Order Neuron (ON) (Primary)

  - Cell bodies in Mesencephalic Nucleus
  - Nerve processes bypass trigeminal ganglion through motor nerve, travel up to Midbrain
  - Peripheral processes from 1st ON pass proprioceptive signals from Mastications muscles, TMJ & PDL

- 2nd ON (Secondary)

  - Cross midline and ascends brainstem through Trigeminal Lemniscus
  - Synapse in Thalamus

- 3rd ON (Tertiary)

  - Passes through internal capsule to sensory cortex

Descending Pathways

General Pathway - Corticospinal tract (pyramidal) - Voluntary muscle limb movements

Trigeminal Pathway - Corticonuclear (corticobulbar) - Voluntary movements of orofacial muscles

2 main neurons - Upper and Lower (important for defect site evaluation)

Upper Motor Neuron Defect	Lower Motor Neuron Defect
• Spastic paralysis (initially flaccid) • No significant muscle atrophy • Fasciculations and fibrillations not present • Hyperreflexia	• Flaccid paralysis • Significant atrophy • Fasciculations and fibrillations • Hyporeflexia

Fasciculations and fibrillations: Muscle activity not visible, only through EMG

Upper motor defect: Superior to spinal cord - lower motor intact thus reflexes present but no originating signal thus no fasciculations / fibrillations, muscles contracted thus no atrophy*

Lower motor defect: Spinal cord or inferior injury, signals still originating thus fasciculations / fibrillations but no reflex due to lower connection loss, muscles not able to finish contracted thus post defect atrophy*

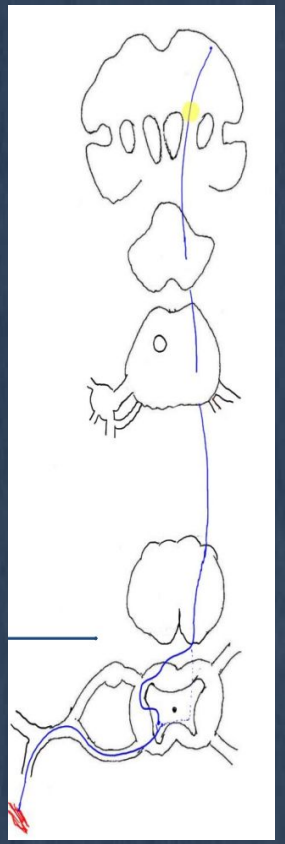
Descending Pathways - Corticospinal Tract

- Upper motor neuron

- Originates from precentral gyrus, travels through brainstem
- Crosses at ‘pyramid’
- Synapse at spinal cord ventral horn

- Lower motor neuron

- Innervates limb muscles



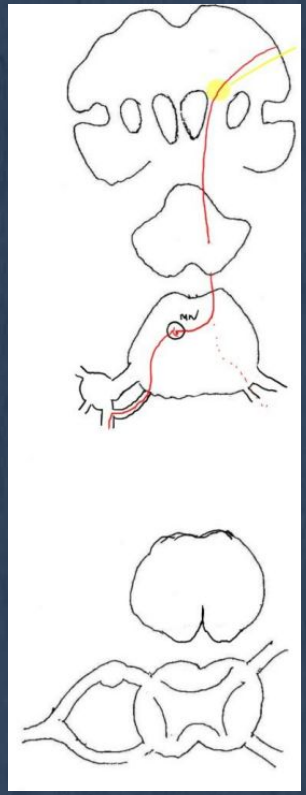
Descending Pathways - Corticonuclear Tract

- Upper motor neuron

- Originates from precentral gyrus, travels through brainstem
- Crosses at Pons
- Synapse at Motor Nucleus

- Lower motor neuron

- Passes through motor root to associated orofacial muscles



Neural Pathway Defects

- Note: SNS / PNS innervate same organs / muscles
- Thus when one system is damaged , the other will run unopposed

Normal SNS activation	**SNS (cervical sympathetic outflow) Damage**
Increase sweating	Anhydrosis
Constriction of blood vessel to non-essential organs	Vasodilatation
Contraction of levator palpebrae superioris muscle (moves upper eyelid)	Ptosis
Dilation of pupils	Miosis

Neural Pathway Defects

CHAPTER 17

MR MYDULLOSO – PARALYSED ON ONE SIDE

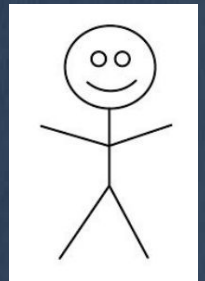
Mr Mydulloso, a 60-year-old postman, arrives in a wheel-chair for his dental appointment, with his wife accompanying him. He is paralysed on the left side, and has no conscious proprioception or fine touch sensation on that side. However, pain and temperature sensations are normal throughout the body. He also has exaggerated tendon reflexes on the left side of the body.
His wife tells you that he is having difficulty articulating certain words. When you examine his tongue, you notice that it deviates to the right side. The right side of his tongue also appears to be wasted compared with the left. He also seems to have some imbalance in the movement of his eyes. His wife explains to you that Mr Mydulloso suffered a stroke a few months ago and spent some time in hospital.

Key Issues

RHS

1). Tongue deviating to RHS (Atrophy)

LHS genioglossus working unopposed

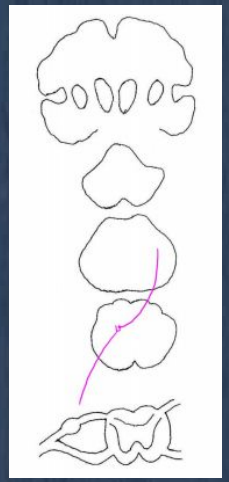


LHS

2). Paralysed - with Hyperreflexia

3). No proprioception or fine touch

Neural Pathway Defects

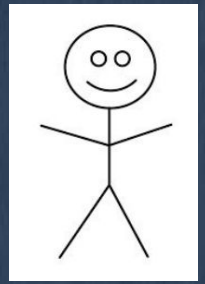


RHS

1). Tongue deviating to RHS (Atrophy)

- Atrophy = lower motor neuron defect
- Tongue Glossus muscles = Hypoglossal cranial nerve
- Hypoglossal UMN cross in Medulla then synapse

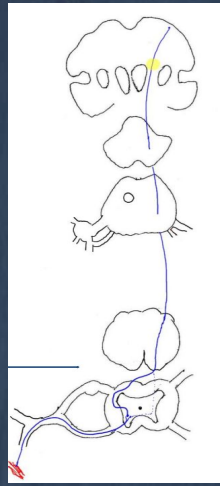
Key Issues



LHS

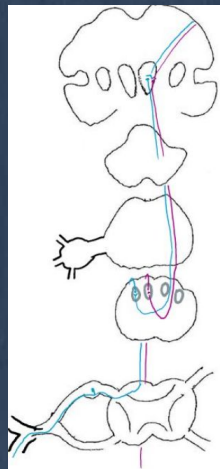
2). Paralysed - with Hyperreflexia

- Hyperreflexia = Upper motor neuron defect



3). No proprioception or fine touch

- Dorsal column tract responsible for proprioception / fine touch



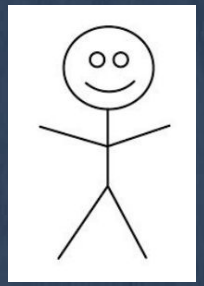
Neural Pathway Defects

RHS

1). Tongue deviating to RHS (Atrophy)

- Atrophy = lower motor neuron defect
- Tongue Glossus muscles = Hypoglossal cranial nerve
- Hypoglossal UMN cross in Medulla then synapse

Key Issues



LHS

2). Paralysed - with Hyperreflexia

- Hyperreflexia = Upper motor neuron defect
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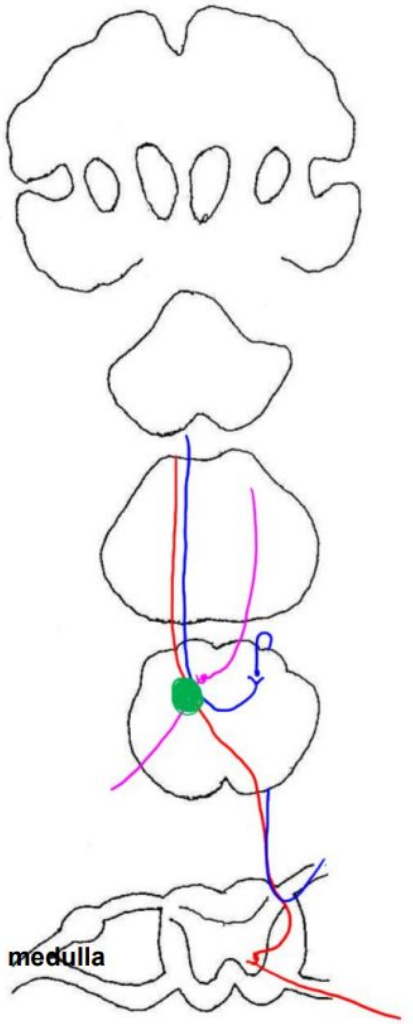
3). No proprioception or fine touch

- Dorsal column tract responsible for proprioception / fine touch

Lesion most likely in RHS Medulla Oblongata



PIA

Management Plan

- Management plan scaffold:
  - CC
  - Further info/Additional tests / Referrals
  - Patient education
    - Go through diagnosis
    - - Treatment options
  - Informed consent
    - Risks, benefits, costs, appt sequences, etc.
  - Emergency and Stabilization Phase
    - ROP
  - Disease Control and Prevention
    - Preventative care e.g. OHI, smoking cessation etc.
  - Immediate Rehabilitation
    - Restorative, Perio etc.
- - Short Term Review
- - Long Term Review

Restorative Steps

- Difference between Amalgam, CR and GIC / RMGIC
- Look at question images carefully!!!

- CR Example 46 MO:
  - Gain pnt informed consent
  - Apply LA (RHS Inferior alveolar nerve) with topical
  - Shade match (less important posterior teeth, make sure natural lighting used)
  - Place RD, W5 47 - 44
  - Access caries through enamel with HS 822 burr and water
  - Clean out caries with SS appropriate sized round burr
  - Check preparation for remaining infected dentine
  - Condition dentine with 20% polyacrylic acid for 10 secs, wash for 20 secs, dry (not desicate)
  - Place Fugi LC liner on dentine only, LC 20 secs
  - Etch Enamel with 37% orthophosphoric acid 10-15 secs, wash 30 secs, dry enamel surface
  - Place Adhesive material, gentle triplex air application to help spread material, LC 20 secs
  - Place sectional matrix interproximally and wedge tightly, check contacts and slot base margins
  - Place composite resins in 2mm increments and on minimal surface to reduce C factor, LC each increment 20 secs
  - Place CR until sufficient material present, LC final layer, remove excess material with appropriate burrs, remove sectional matrix and wedge, check margins, contacts and interproximal (for overhangs)
  - Final polish with enhanced burr, remove RD,, check occlusion, provide pnt POI and dismiss

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