



**GROUP & INDIVIDUAL LEARNING**

**BDS2 Neuroanatomy &  
Exam Review**



**COMMONWEALTH OF AUSTRALIA**

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# Neuroanatomy

# Ascending pathways of the CNS

## Random terminology

Fascicle = bundle of nerves

Lemniscus = bundle of secondary nerves

Revise the scenarios in the anatomy  
book

# Ascending pathways of the CNS

## General Pathways

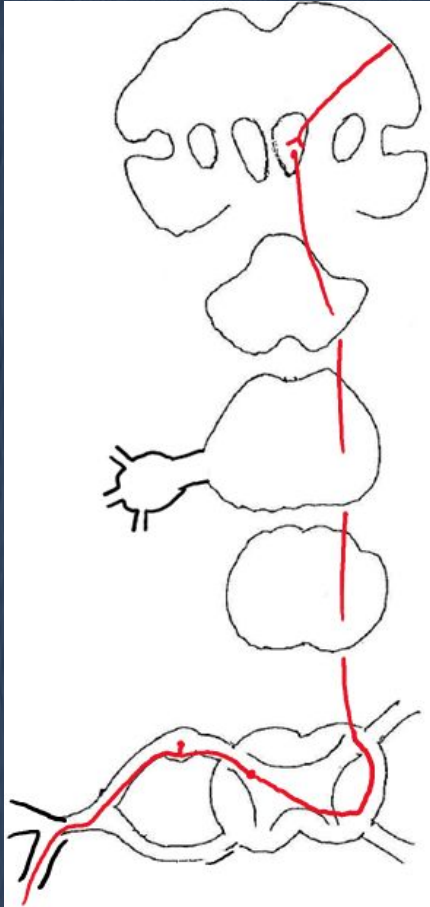
1. **Spinothalamic tract** - pain, temperature, crude touch
2. **Dorsal Column tract** - fine touch, proprioception

## Mandibular Pathways

1. **Pain, temperature, crude touch**
2. **Fine touch**
3. **Proprioception**

# Spinothalamic Tract - Pain, temperature and crude touch

Cerebral Hemispheres



Midbrain

PONS

Medulla Oblongata

Spinal Cord

## 3<sup>rd</sup> order neuron (tertiary)

- Thalamus – ovoid mass of grey matter on each side of the 3<sup>rd</sup> ventricle  
→ Acts as a relay station
- Pass through the posterior limb of the internal capsule (broad band of white matter medial to lentiform nucleus and lateral to the thalamus)
- Then to the post-central gyrus of the cerebral cortex

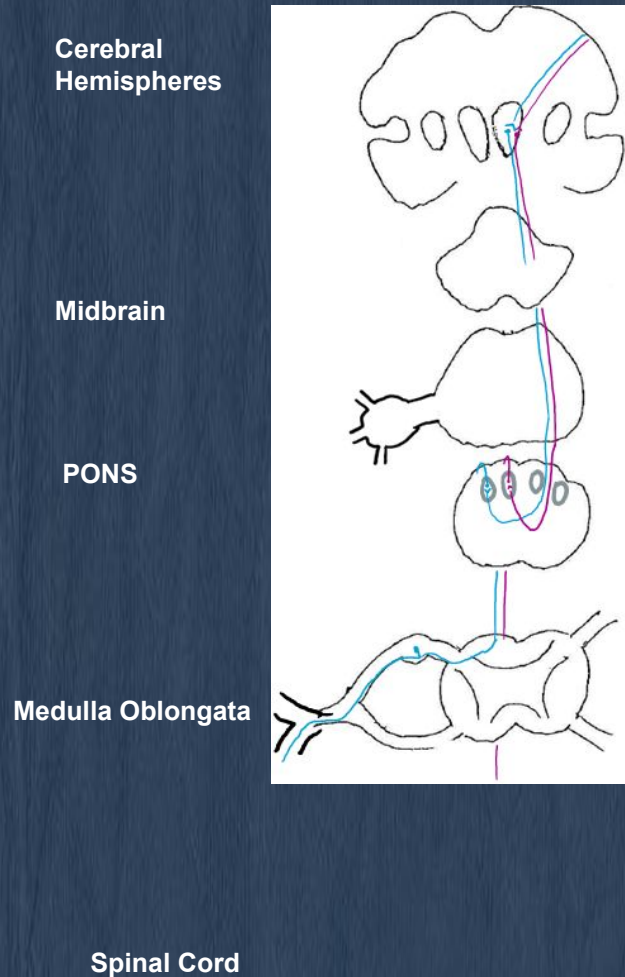
## 2<sup>nd</sup> order neuron (secondary)

- Cross the midline → pass up the spinal cord
- Travel through the brain stem
- Synapse in the thalamus (ventral posterior lateral nucleus)

## 1<sup>st</sup> order neuron (primary)

- Afferent fibres from receptors are peripheral processes of 1<sup>0</sup> neurons
- Synapse with 2<sup>0</sup> neurons in the dorsal horn of the spinal cord

# Dorsal Column Pathway - Fine touch and proprioception



## 3<sup>rd</sup> order neuron (tertiary)

- Thalamus – ovoid mass of grey matter on each side of the 3<sup>rd</sup> ventricle

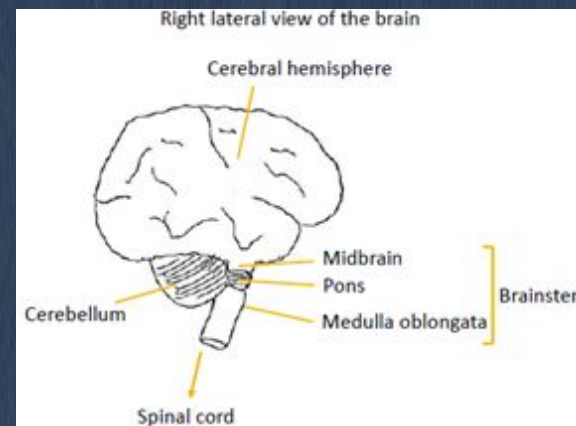
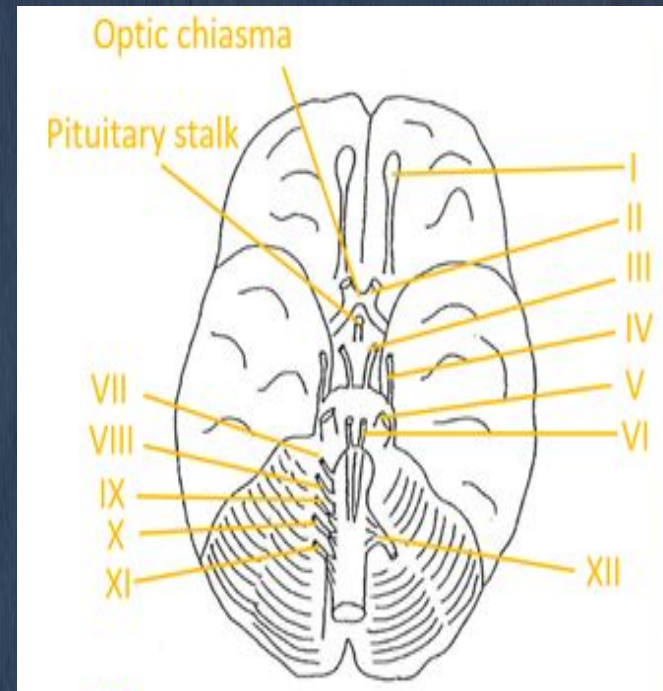
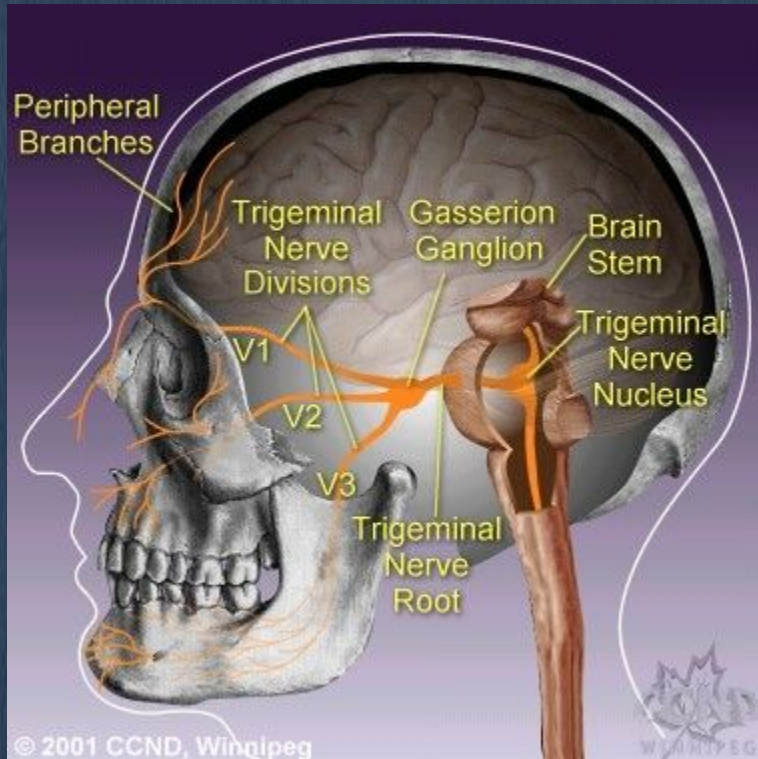
## 2<sup>nd</sup> order neuron (secondary)

- Sweep downwards initially, then cross over and ascend up the brainstem.
  - Internal arcuate fibres – in the medulla (formed by crossing over fibres)
  - Medial lemniscus – bundle of 2<sup>o</sup> neurons travelling towards the thalamus
- Synapse in the thalamus (VPLN)

## 1<sup>st</sup> order neuron (primary)

- A) Medial fasciculus gracilis: Fibres from lower parts of the body are added and travel together
- B) Lateral fasciculus cuneatus: fibres from upper parts of the body are added and travel together
- Fascicles synapse in nuclei (nucleus gracilis and cuneatus) respectively in the medulla

# Mandibular Nerve Pathway



2 + 2 + 4 + 4

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



# Mandibular Pain, Temperature and Crude touch Pathway

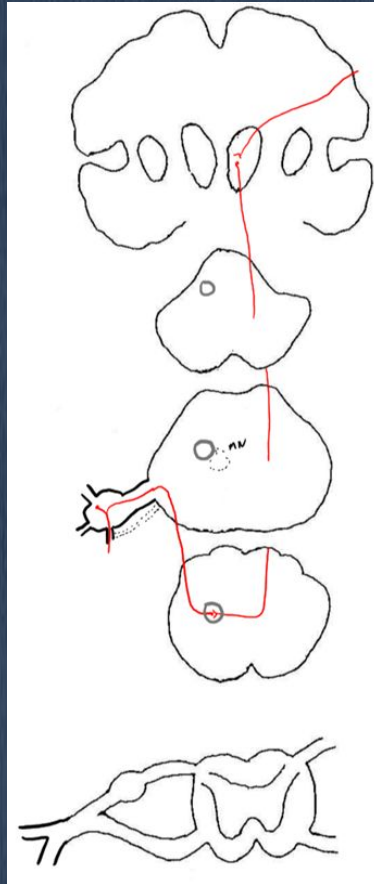
Cerebral Hemispheres

Midbrain  
Mesencephalic nucleus

PONS  
Pontine nucleus

Medulla Oblongata  
Spinal nucleus

Spinal Cord



## 3<sup>rd</sup> order neuron (tertiary)

- From the thalamus, passes through internal capsule → sensory cortex

## 2<sup>nd</sup> order neuron (secondary)

- Cross over to the other side and ascends the brainstem
- Trigeminal lemniscus – 2<sup>o</sup> neurons ascend in it
- Synapse in the thalamus

## 1<sup>st</sup> order neuron (primary)

- Cell bodies located in the trigeminal ganglion
- Descend down to medulla oblongata
- Synapse in the spinal nucleus

# Mandibular Fine touch Pathway

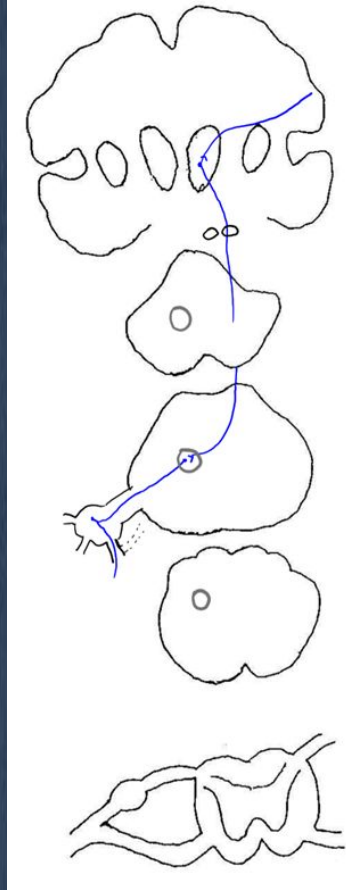
Cerebral Hemispheres

Midbrain  
Mesencephalic nucleus

PONS  
Pontine nucleus

Medulla Oblongata  
Spinal nucleus

Spinal Cord



## 3<sup>rd</sup> order neuron (tertiary)

- From the thalamus, passes through internal capsule → sensory cortex

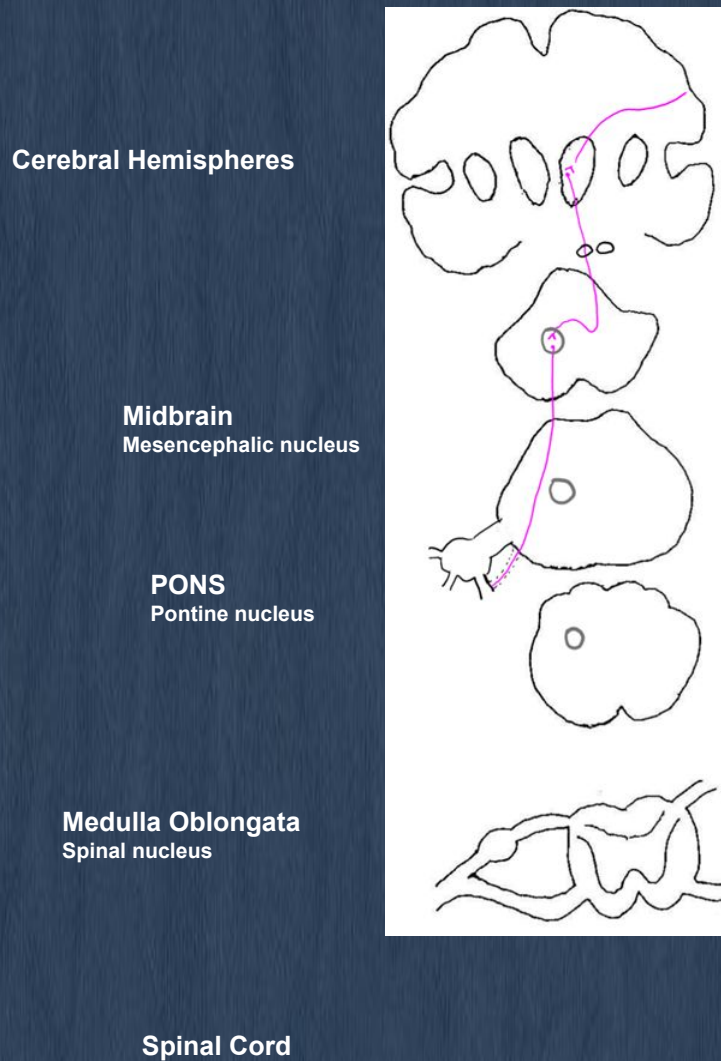
## 2<sup>nd</sup> order neuron (secondary)

- Some crossover (not all) and ascend in the trigeminal lemniscus
- Synapse in the thalamus

## 1<sup>st</sup> order neuron (primary)

- Cell bodies located in the trigeminal ganglion
- Synapse in the pontine nucleus

# Mandibular Proprioception Pathway



## 3<sup>rd</sup> order neuron (tertiary)

- From the thalamus, passes through internal capsule → sensory cortex

## 2<sup>nd</sup> order neuron (secondary)

- Cross over to the other side and ascends in the trigeminal lemniscus to the thalamus

## 1<sup>st</sup> order neuron (primary)

- Cell bodies located in the mesencephalic nucleus
- Peripheral processes of 1<sup>o</sup> neurons deliver proprioceptive information from:

Muscles of mastication  
TMJ  
PDL

# Descending pathways of the CNS

# Descending pathways of the CNS

## General Pathways

1. **Corticospinal tract (pyramidal)** - voluntary movements of limb muscles

## Trigeminal Nerve

1. **Corticonuclear (corticobulbar)** - voluntary movements of orofacial muscles

2 main neurons = upper and lower motor neurons

Upper Motor Neuron Defect	Lower Motor Neuron Defect
<ul style="list-style-type: none"><li>● Spastic paralysis (initially flaccid)</li><li>● No significant muscle atrophy</li><li>● Fasciculations and fibrillations not present</li><li>● Hyperreflexia</li></ul>	<ul style="list-style-type: none"><li>● Flaccid paralysis</li><li>● Significant atrophy</li><li>● Fasciculations and fibrillations</li><li>● Hyporeflexia</li></ul>

# Corticospinal (pyramidal) tract

Cerebral Hemispheres

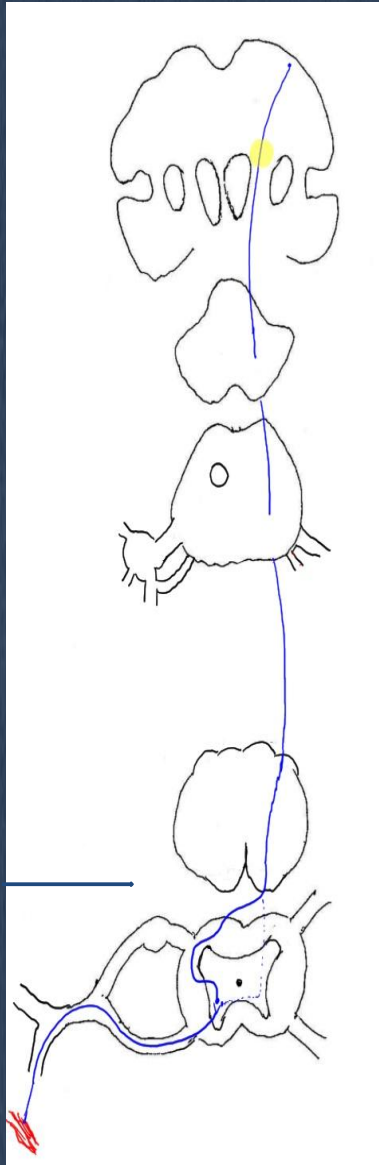
Midbrain

PONS

Medulla Oblongata

Pyramids

Spinal Cord



## Upper Motor Neuron

- Crosses over at pyramids
- Synapses in the ventral horn of the spinal cord

## Lower Motor Neuron

- Innervates associated limb muscle

# Corticospinal tract

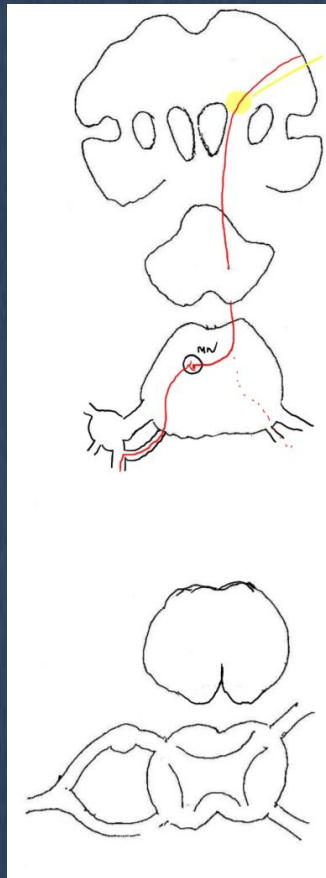
Cerebral Hemispheres

Midbrain

PONS

Medulla Oblongata

Spinal Cord



## Upper Motor Neuron

- Crosses over in pons
- Synapses in the motor nucleus

## Lower Motor Neuron

- Passes through motor root to associated orofacial muscle

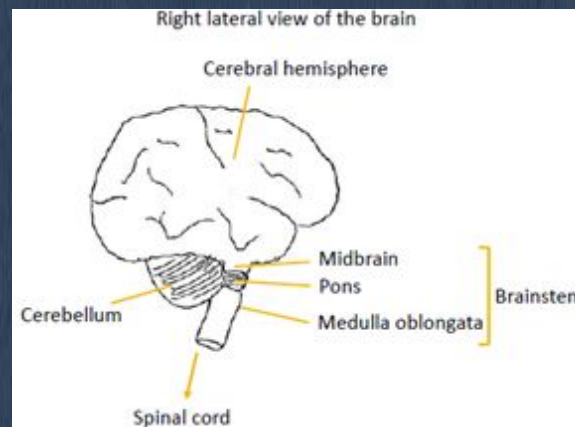
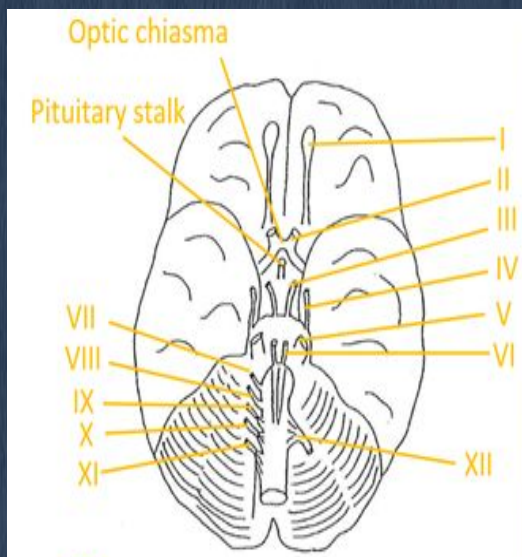
# Soooooooooooooooo

For descending pathways we covered:

- Limbs
- Trigeminal Nerve

What about all the other cranial nerves?

- Know hypoglossal nerve (in relation to geniohyoid muscle)
- Facial nerve is a bit complex, usually don't worry too much about this unless Dr Ranjitkar has time to explain it



2 + 2 + 4 + 4

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



## A brief note regarding SNS and PNS

- SNS and PNS innervate the same organs/muscles
- If one is damaged, the other will run unopposed

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

# Approaching Questions

## CHAPTER 17

### MR MYDULLOSO – PARALYSED ON ONE SIDE

Mr Myduloso, 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 Myduloso suffered a stroke a few months ago and spent some time in hospital.

#### Key Issues

RHS

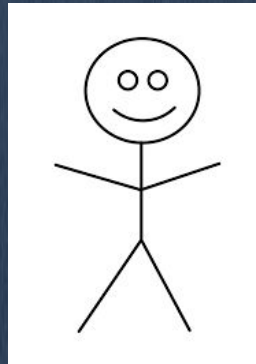
1. Tongue deviating to RHS  
(wasted)

Due to LHS genioglossus working unopposed; consider how the tongue usually sticks out!

LHS

2. Paralysed - with hyperreflexia

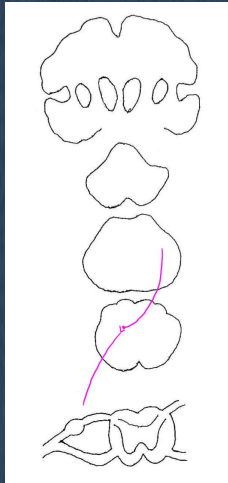
3. No proprioception and fine touch



### 1. Tongue deviating to the RHS (wasted)

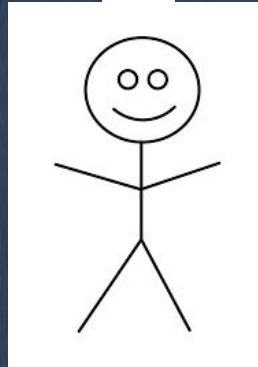
- Wasting = Lower motor neuron defect
- Must be in medulla oblongata
  - XII innervates genioglossus → UMN crosses over in medulla and synapses in the hypoglossal nucleus → LMN innervates muscles of the tongue

#### Hypoglossal Nerve tract



medulla

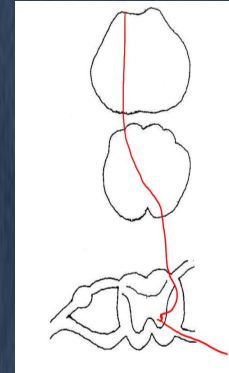
**Bob**



### 2. Paralysed with hyperreflexia

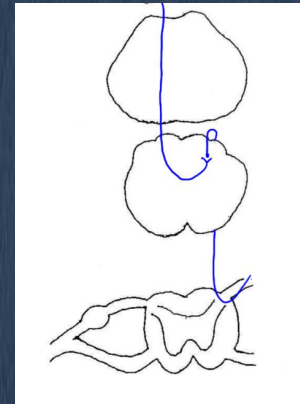
- Hyperreflexia = upper motor neuron defect

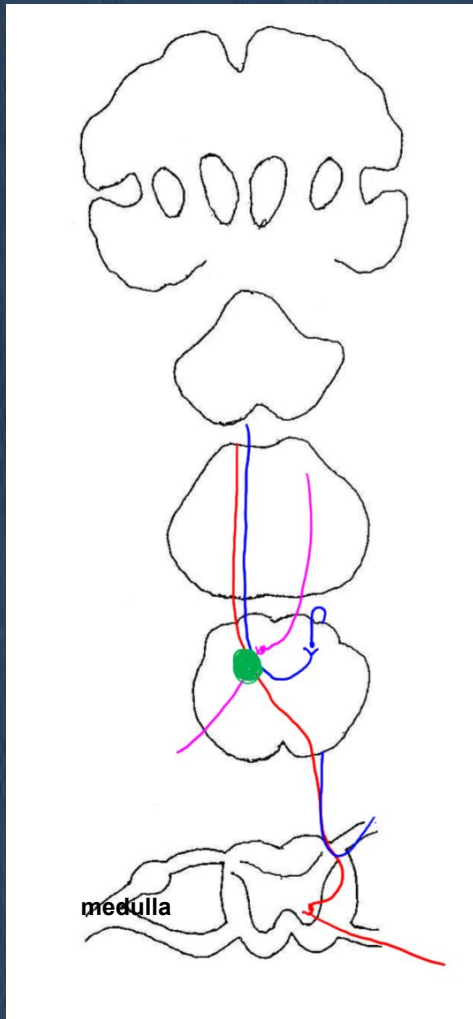
#### Corticospinal tract



### 3. No proprioception or fine touch

#### Dorsal Column tract



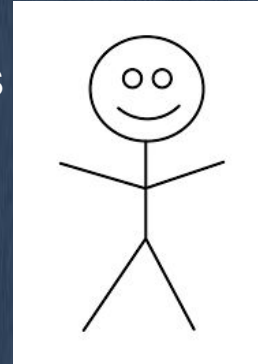


RHS

LHS

1. Tongue deviating to RHS  
(wasted)

Due to LHS genioglossus working unopposed; consider how the tongue usually sticks out!



- 2. Paralysed - with hyperreflexia
- 3. No proprioception and fine touch

Green spot = most likely location of the lesion (in the RHS of medulla oblongata)

# Exam Review

# Topics

- Immunology Tolerance + hypersensitivity
- PCC
- Microbiology
- Sterilisation
- Wound healing
- Inflammation & Wound Healing
- Local Anaesthetic
- Anatomy
  
- ILA
- Treatment Planning
- Steps for Restoration
- Erosion + management

# Tolerance

Ability to recognise own self-antigen

2 types:

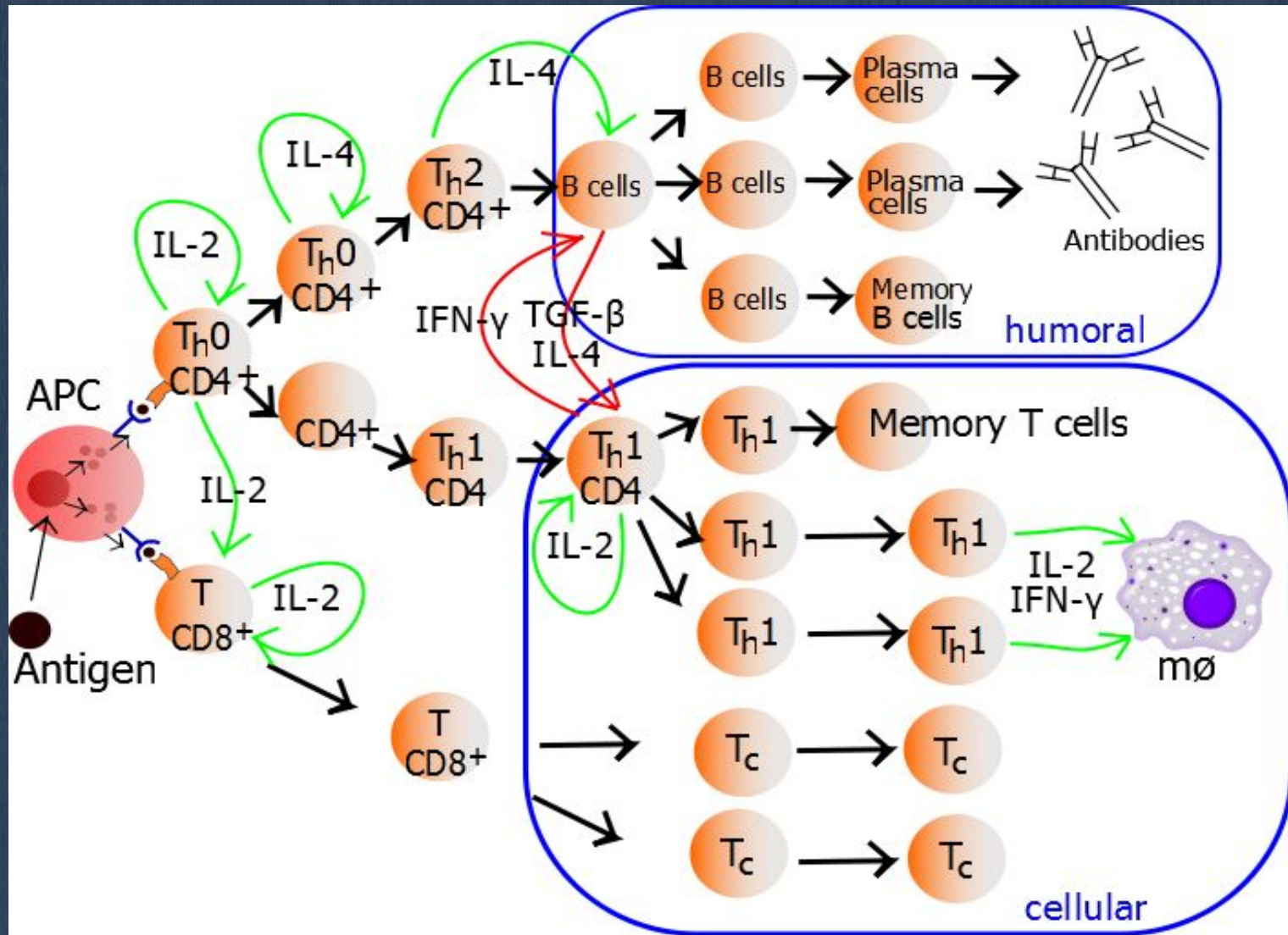
Central tolerance - negative selection, BM and T

Peripheral tolerance

- clonal anergy (half activated state)
- immunoregulation/immunosuppression
  - CD4 → Treg or CD25 assoc. with autoimmune disease
  - CD8 → T0 or CD28 → suppress graft rejection
- Th1 and Th2 Inhibition → establish tolerance



# CD4-CD8 and Th1-Th2 Inhibitor



# B Cell Tolerance

- Some self-reactive B cells can escape negative selection

Peripheral tolerance via

- clonal anergy
- inhibition or regulation from regulatory cytokines
  
- Somatic hypermutation

Loss of tolerance  $\Rightarrow$  autoimmunity

Conditions: Rheumatoid arthritis, Sjogren's Syndrome, Multiple Sclerosis, Pterygium Vulgaris, **RHD**

# Hypersensitivity

Type I - allergic response mediated by IgE  
eg. Anaphylaxis, latex allergy, asthma

Type II - cytotoxic response mediated by IgM/IgG/complement  
e.g. RHD, anaemia, erythroblastosis fetalis, myasthenia gravis

Type III - involves immune complex formation mediated by  
Complements, IgG and antigen. (Ab + Ag)  
e.g. Serum sickness, RA, SLE (lupus).

Type IV - Delayed-type (days) - cell mediated (Th1 cells,  
macrophages, cytokines)  
e.g. contact dermatitis, chronic transplant rejection, MS.

# Vaccinations

Build immunity to viral infections via:

- response to surface antigen on viral particle
- response to infected host cell

Types:

1. Attenuated pathogen (reduced virulence)
2. Killed pathogen
3. Extracts of products of pathogen
4. Adjuvants in vaccines

# PCC

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

Dealing with:

- Anxious patients
  - Approach?
  - Management?
- Angry patient
  - how do you respond to them?
    - DEFUSE

# PIA

- Go through ILA scenarios from sem 1 & 2
- Know LA landmarks
- Anatomy comes up more (((a lot)))
- Radiographic interpretation: Bitewings, OPG.
- Occlusion lab
- EBD from sem 1

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
<u>PolyOHbutyrate</u>	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	NB: know terminology for classifying bacteria e.g. facultative anaerobes, etc



# 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
  - Includes polymyxins and polyenes
- Inhibits cell wall synthesis
  - Targets peptidoglycans
  - Requires cells to be growing
  - Includes B-lactams, penicillins, cephalosporins
- Inhibit DNA/RNA synthesis
  - e.g. Rifamycin
- Inhibit protein synthesis
  - Targets ribosomes
  - Includes tetracycline
- Inhibition of folic acid metabolism
  - Includes sulfonamides

# B-lactamase

- 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

“P”lease be my friend!

# 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

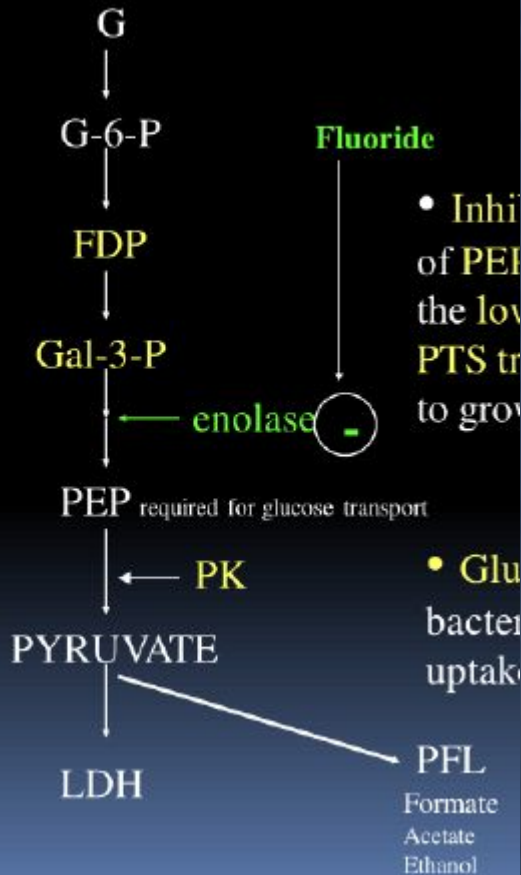
“G”, I’m popular!

# 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

# Fluoride on Metabolism of Sugars

## Glucose limitation intermediates in low concentration



- reduces amount of acid, etc produced
- also reduces the total number of bacteria as they do not grow as much therefore when sugar is introduced into mouth again, overall less acid is produced



# Wound Healing

## Primary Intention

- Incisional wound; no tissue loss
- area to be repaired decreased by apposition of wound edges via suture or fibrin
- blood clot small
- granulation tissue small
- minimal wound/scar

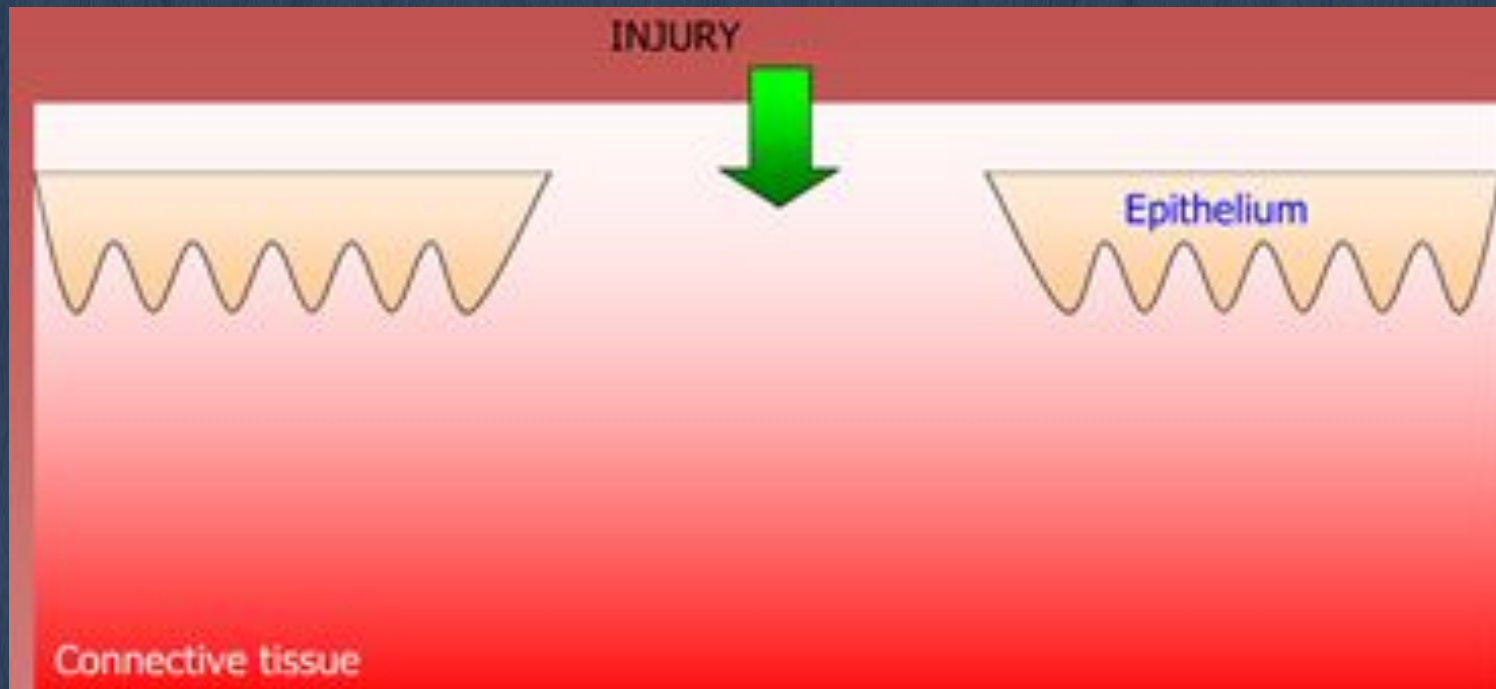
# Wound Healing

## Secondary Intention

- e.g. ulcer
- tissue loss - can't get close apposition of wound edges
- area to be repaired is relatively large
- large blood clot formed
- more inflammation
- wound contraction and more scarring

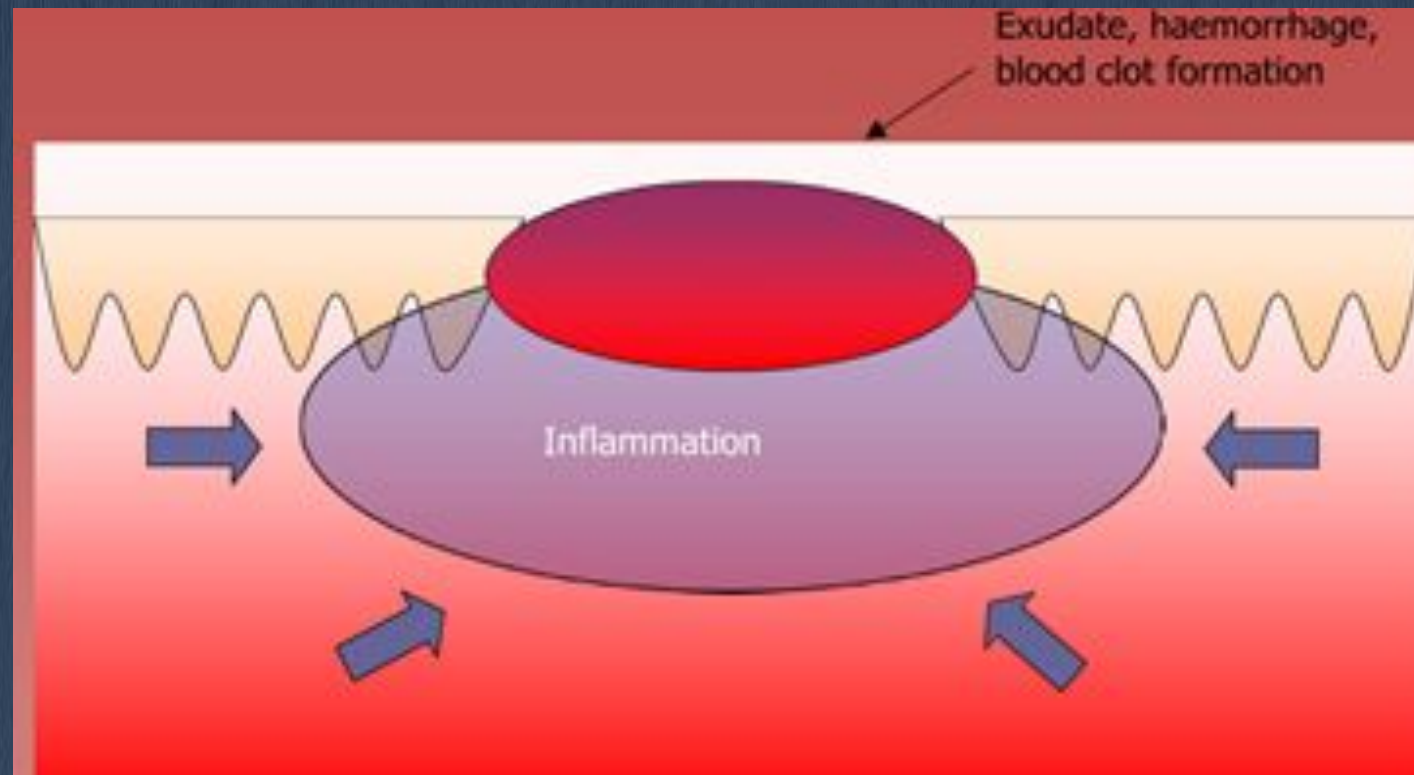
# Secondary Intention

0 hours, injury = big defect in epithelium



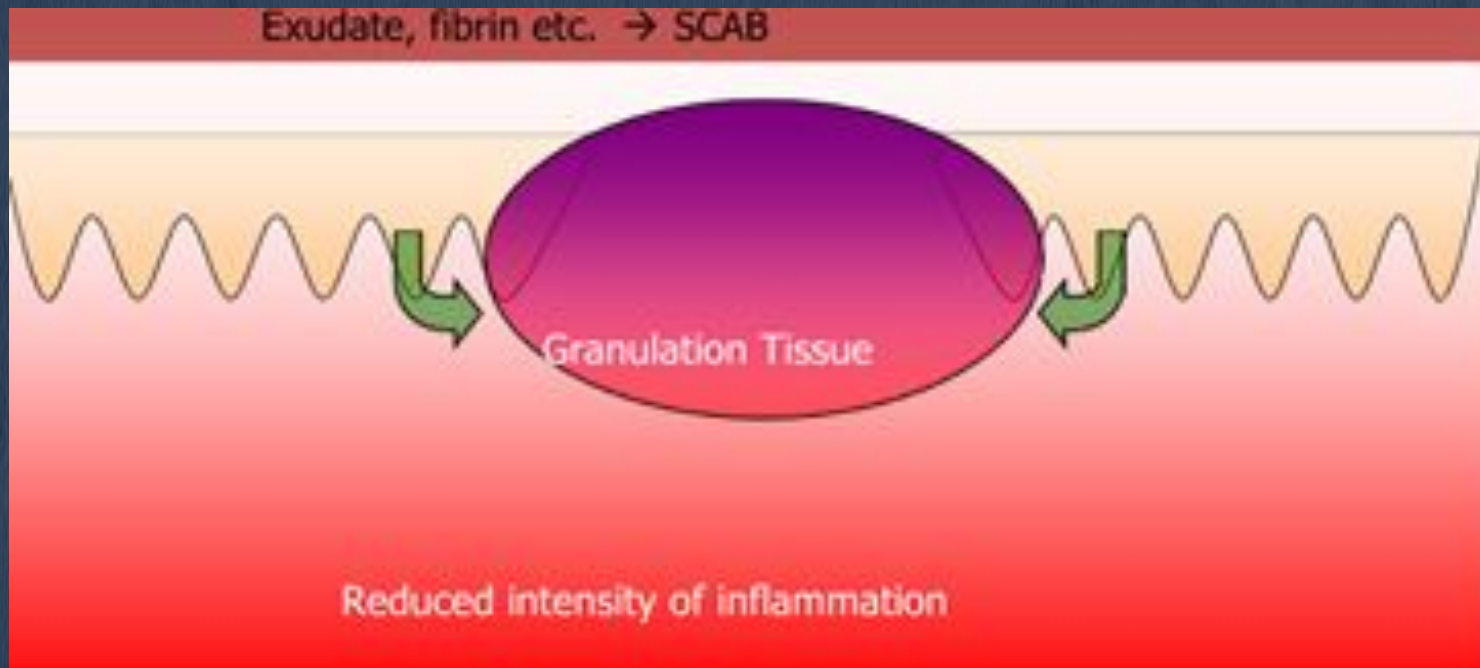
# Secondary Intention

24 hours



# Secondary Intention

2 days - 1 week

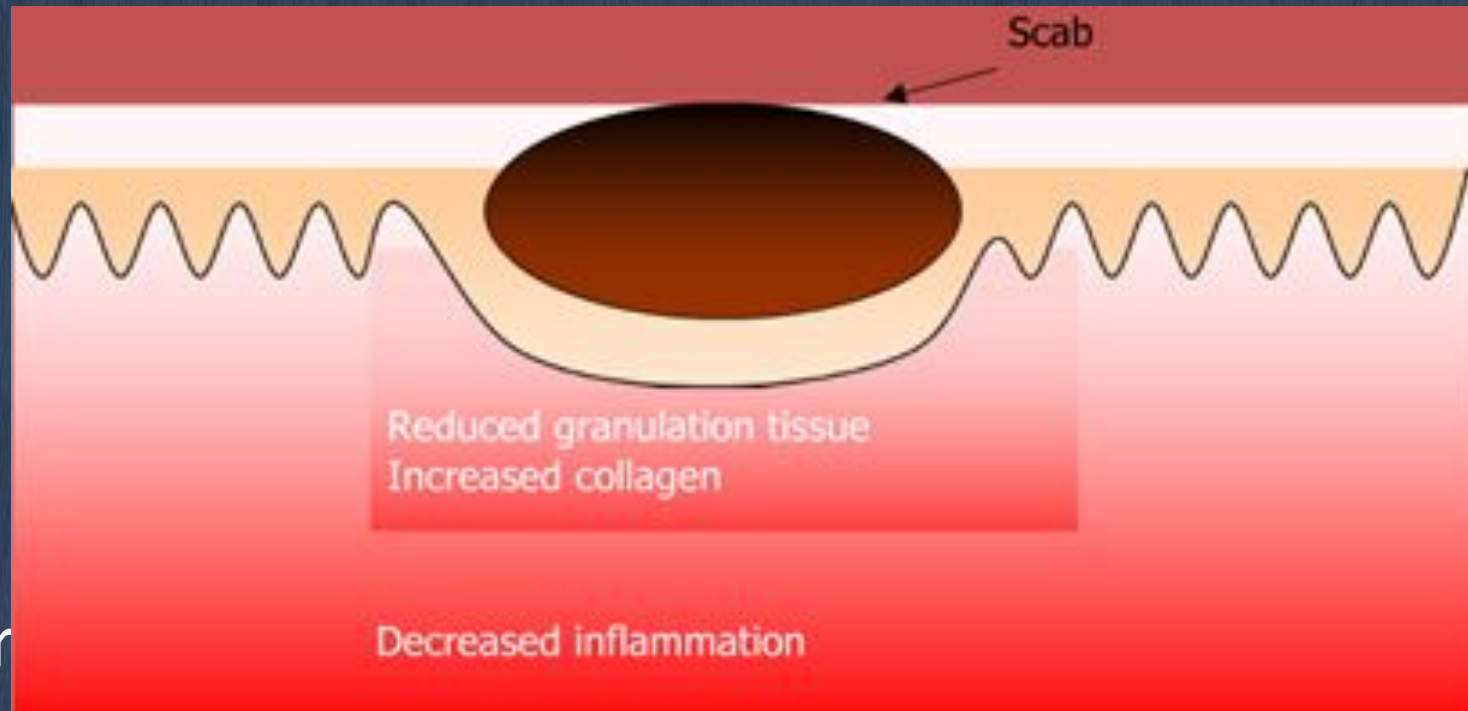


ised

- by fibrin = scab
- granulation tissue = increased inflammation
- epithelium regenerates under the scab

# Secondary Intention

2 weeks



-or

-decreased inflammation

llagen

# Secondary Intention

1-2 months

- epithelium and epidermal layer organisation complete

- subepithelially:

  - ongoing organisation and maturation

  - ongoing decreased vascularity

  - ongoing collagen maturation

  - can have contraction of collagen

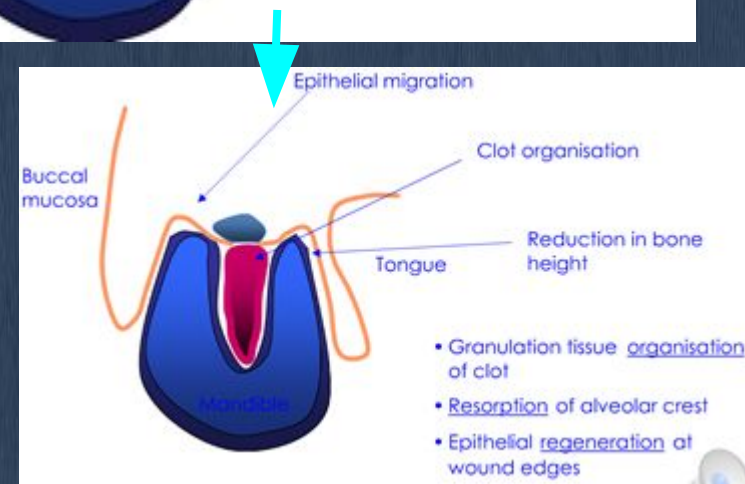
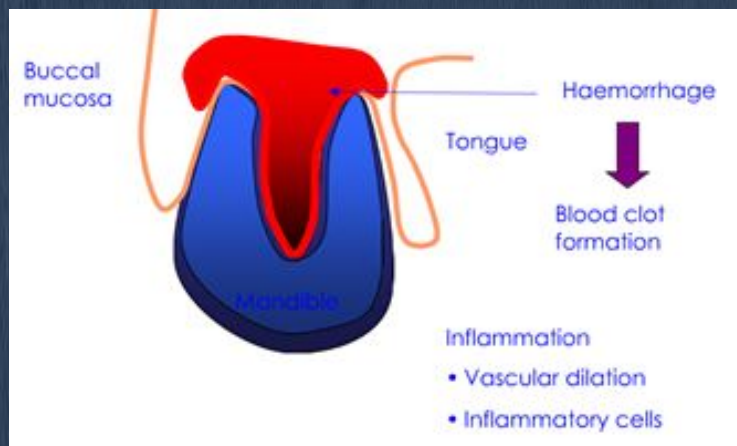
    - if wound is large can have functional problems

      - uncommon in oral cavity

# Tooth Socket

-extraction = traumatic injury + creation of bony defect

-Heals via secondary intention and bone regeneration





# Tooth Socket Complications

- failure to form blood clot
- e.g. due to haemophilia (can't make clot), anaemia, anticoagulant therapy, smoking
- may cause infection (osteomyelitis)
- Dry socket aka alveolar osteitis
- unknown aetiology, usually Md molar and premolar
- Tx: will heal by itself usually, keep area clean
  - may need special dressing in some cases
- usually v. uncomfortable/painful

# Neoplasia

## Clinical issues of benign neoplasms:

- Pressure from continuously growing lesion impinging on adjacent vital structures
- Hormone production, e.g. thyroid tumour (goitre) – overproduction of TH; issues with temperature regulation and metabolism
- Disfigurement (functional and cosmetic applications)
- Malignant transformation; if untreated
- Patient anxiety/discomfort

# Neoplasia

Clinical issues of malignant neoplasms: sig morbidity and mortality due to:

- Destruction of and impingement on adjacent vital structures
- Fluid flow obstructions – BV, lymphatics, GI – all hollow tubes; if blocked, significant issues arise.
- Haemorrhage from aggressive ulceration
- Metastases and resultant secondary tumour formation
- Debilitating aggressive secondary tumour formation
- Debilitating aggressive treatment modalities (e.g. radio/chemotherapy, invasive surgery)
- Pain and psychological trauma

What is the difference between a granuloma and granulomatous inflammation?

- Granulomatous inflammation:
  - Granulomatous tissue - composed of granulomas (cluster of epithelial histiocytes) persisting protractedly due to ongoing inflammation caused by tenacious offender lymphocytes
  - Giant cells also sometimes seen
  - NOT phagocytic; produce cytokines to direct and orchestrate ongoing inflammation (communicative)
- Vs Granulation tissue:
  - intermediary tissue between inflammation and healing
  - vascular + collagen
  - before re-epithelialisation

# Sterilisation

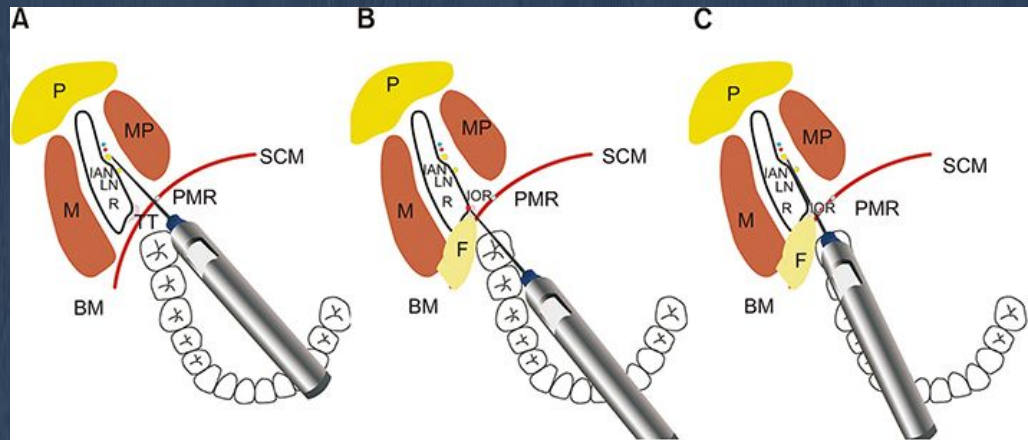
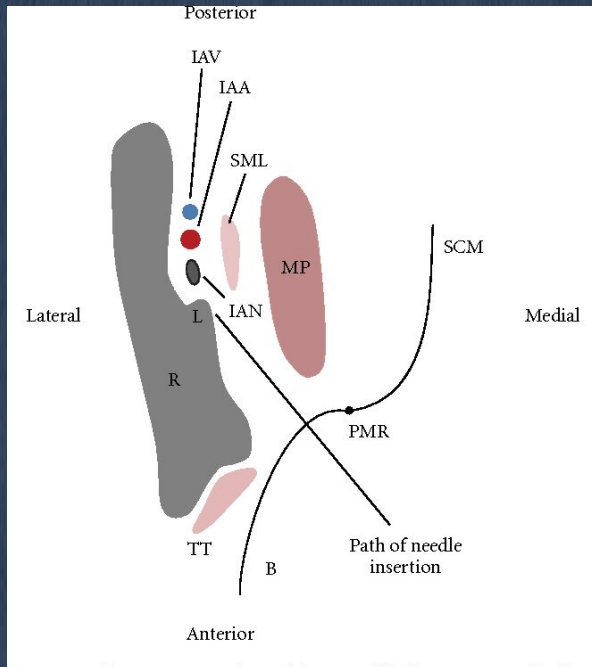
- got tested on different methods of sterilisation and why we do it
- e.g. why do we autoclave?
- know why we use the bag for sterilisation

# 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 + anatomy = 90% of last year's PIA
- Trigeminal neuralgia? :”(

# LA questions

- Be able to draw (CORRECT SIDE, NO ABBREVIATIONS):





# EBD

- Not covered in Semester 2, but several MCQ in Paper 2 2017
- Learn lectures well - Q's come directly from content covered