Semester 2 Exam

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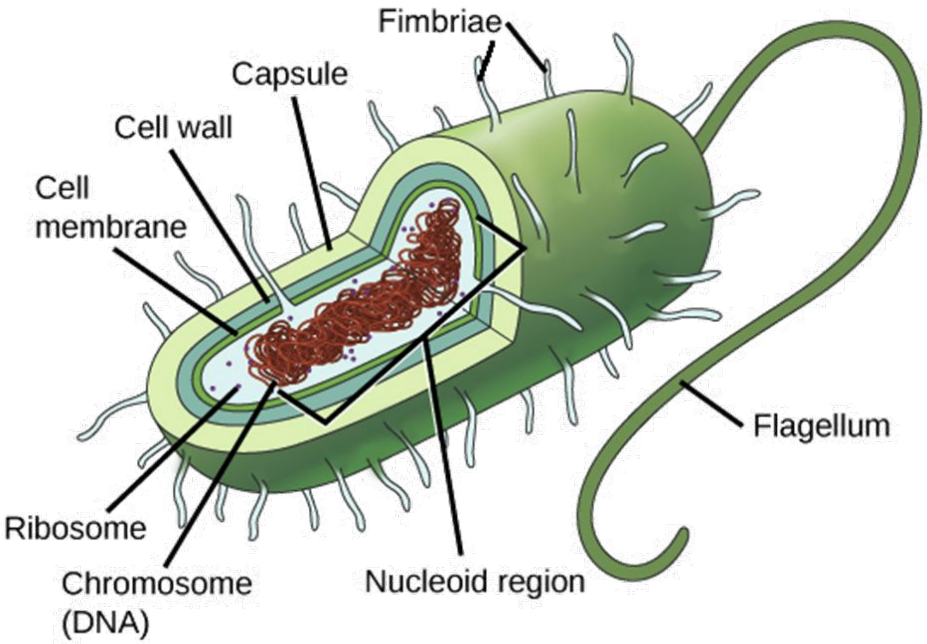
Microbiology

MicroB

Bacteria cell components:

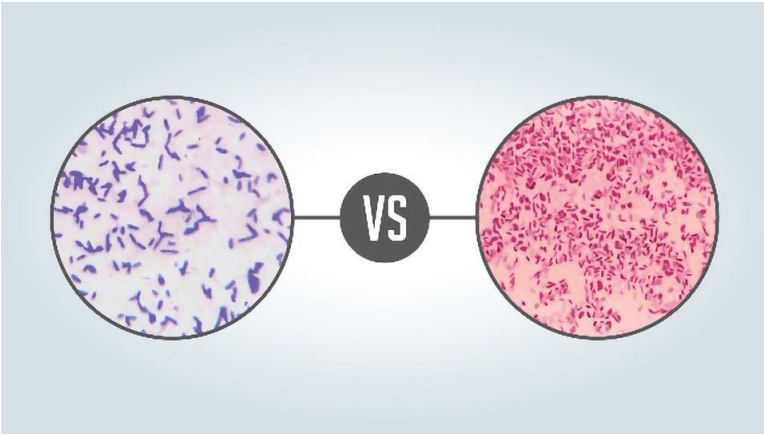


Gram +ve:

- Thick peptidoglycan layer
- Stains purple

Gram -ve:

- Thin peptidoglycan layer
- Stains pink

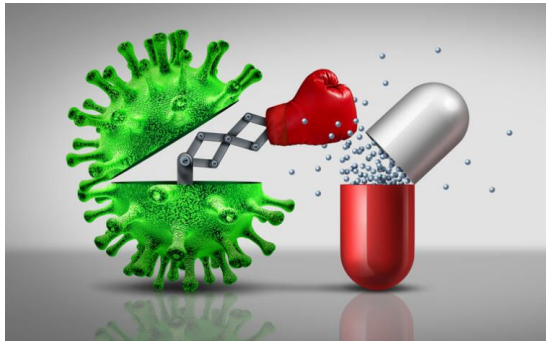


Antibiotics

- Frequently found in soil bacteria and fungi
- Can be either bactericidal (killing) or bacteriostatic (inhibits growth)
- Spectrum of action depends
  - Broad - Big range of bacteria
  - Narrow - Either gram +ve or gram -ve
    - Best to be narrow to prevent development of antibiotics resistance
- Many types available:
  - Cell wall synthesis inhibitors (B-lactam antibiotics), protein synthesis inhibitors (Macrolides), nucleic acid inhibitors (Rifampicin), cell membrane inhibitors (antifungals)

Some antibiotic resistance mechanisms that bacteria can exhibit:

- Enzymes that inactivate antibiotics
- Pumps to pump out antibiotics
- Conformational change to drug binding site



Mechanisms of pathogenicity (virulence)

1. Attachment to host
    a. Specific & Non-specific binding depends on the bacteria
        i. Etc. Fimbrae on Porphyromonas Gingivalis binds specifically
2. Invasion
    a. Entering the host through damaged barriers like a cut on skin
3. Proliferation
    a. Starts colonising the site of entry, may spread to bloodstream
4. Evasion
    a. Many ways to evade host immune cells.
        i. Etc. secrete exotoxins to kill immune cells

Strep Mutans

- Aciduric (survives in acidic environment) due to presence of many ATPase pump on cell membrane > pumps out H+
- Major cariogenic bacteria, high number in carious lesions
- Has 3 ways for sucrose uptake
    - Sucrose-PTS - Highest affinity for sucrose so activated in famine condition
    - Trehalose-PTS
    - Multiple sugar TS - Lowest affinity so activated in well-fed conditions (Low pH conditions)

Chemicals for controlling caries

Fluoride	• Antibacterial at high concentrations
	  ○ Inhibits PEP/PTS system (famine) > bacteria unable to move glucose into cell
	  ○ Affects bacteria membrane integrity > bacteria becomes leaky, intracellular pH rises
	• Remineralise tooth structure
	• Strep Mutans is susceptible to fluoride

Chlorhexidine	• Broad spectrum but does not upset normal oral microflora due to S mutans being more sensitive to CHx • Blocks adherence to host • Blocks PEP/PTS • Interferes with S mutans' ATPases > S mutans less able to withstand acidic environment
Xylitol	• Sugar substitute • Induces a 'futile cycle' • S mutans spend ATP to bring it into cell > cannot break xylitol down > spend more ATP to pump it back out

Disinfection & Sterilisation

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: 160 degrees in an oven for 2 hours, not as efficient and can melt some dental materials
- Radiation: In operating theatres, damages DNA
- Filtration: LA carpules

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, latex allergy

Note: Type 4 latex allergy is usually a reaction against chemicals used in glove manufacture while Type 1 latex allergy is due to contact with natural latex rubber. If you have pt with allergy to bananas, avocados, nuts or kiwi, they have a higher chance to develop latex allergy so careful MHx.

Virus transmission

1. Aerosol transmission (etc Influenza A virus)
    a. Spread via respiratory droplets
    b. Droplets remain airborne for days
    c. Scaling generates lots of aerosol, so important to identify infected patients.
2. Faecal-Oral transmission (etc. poliovirus)
    a. Faecal contaminated water source
    b. Inadequate hand hygiene when preparing food
    c. Houseflies
3. Blood-borne transmission (Etc hep B)
    a. Exposure to infectious bodily fluid via oral mucosa or broken skin
    b. Unsterilised instruments
4. Insect vectors (etc. zika virus)
    a. Mosquitos

Autoimmunity & Tolerance

- Tolerance is important for normal function
  - 3 key features:
    - Immune system only responding to pathogens, unresponsive to self antigen, Unresponsive to commensal microflora
- Central tolerance
  - Central tolerance in Thymus (T cells) and Bone marrow (B cells)
  - Negatively selects and eliminates autoreactive cells (via apoptosis)
- Peripheral tolerance
  - Outside of Thymus and Bone marrow
  - Removes any autoimmune cells that central tolerance missed by ensuring that these cells will never be activated
- Know briefly about autoimmune disease
  - Rheumatoid arthritis, systemic lupus erythematosus, sjogren's syndrome, multiple sclerosis, pemphigus vulgaris, mucous membrane pemphigoid
  - Treatment options for autoimmune diseases: Relieve symptoms, replacement of hormones, OHI, Immunosuppressants

Rheumatic heart disease***

- Molecular mimicry of M proteins on Strep A bacteria > mimics M proteins on cardiac muscle.
- Host immune system attacks cardiac cells > scarring of heart tissues and valves
- Scarring provides rough surface for circulating bacteria to bind to > infective endocarditis

Clinical significance

- Antibiotic prophylaxis need to be provided prior to patients in (Page 194)
    - Has history of IE
    - Has rheumatic heart disease in all patients
    - Has prosthetic cardiac valve or prosthetic material used in cardiac valve repair
    - Congenital heart disease: untreated cyanotic defects (insufficient o2 in heart) or treated defects that are near prosthetic material
- Antibiotic prophylaxis (Page 197)
    - Amoxicillin 2g, orally, 1 hours prior to procedures
    - If pt is allergic to penicillin then: Clindamycin 600mg, orally, 1-2 hours prior to procedures
    - If cannot give orally, give via IV drip or intramuscularly.
- Denture procedures of concern
    - Procedures that involves manipulation of gingiva, periodontal tissues and perforation of mucosa: Periodontal probing in diseased tissue, subgingival debridement, root planing, extraction, RCT, implants

General Pathology

Cell Disorders

Hypertrophy
- Increase in the size of cells resulting in an increase in the size of the organ (Note: cell number stays the same)

Hyperplasia
- Increase in the number of cells in an organ -> increased proliferation via mitosis, decreased apoptosis

Eg. Linea Alba, Smoker's Keratosis

Atrophy
- Shrinkage in the size of cells by the loss of cell substance

Metaplasia
- Transition of one differentiated cell type to another

Dysplasia
- Disorderly proliferation – loss of uniformity of individual cells and their orientation
    - Still the same tissue

Neoplasia

- Continuous, unregulated replication -> unable to stop even when stimulus is removed
- Neoplasm has 2 types:
    - o Benign -> remains localised
    - o Malignant -> lesion can invade and destroy adjacent structures and metastasise
- Causes:
    - Failure of tumour suppressor genes (eg. p53)
    - Mutation of proto-oncogenes -> oncogenes (upregulates cell growth)
    - Expression of telomerase - prevents cells senescence and apoptosis

Feature	Malignant Neoplasm	Benign Neoplasm
Growth rate	Relatively quick	Slow
Direction of growth	Endophytic growth – growing into the tissue it originates from	Exophytic growth – growing out of the tissue it originates from
Border	Poorly defined and irregular	Well circumscribed and regular
Ulceration	Common, especially in lesions on mucosal surfaces	Rare
Histological resemblance to normal tissue	Can be variable, however it is often very poor	Often resembles normal tissue
Mitoses	Often present and atypical	Rarely present
Nuclear morphology	Usually abnormal: enlarged, hyperchromatic, irregular border and pleomorphic	Typically normal
Invasion and metastasis	Frequent	Never

Inflammation

Acute – develops within minutes or hours (lasts for a short duration)

- Exudation of plasma proteins and fluids
- Emigration of leukocytes and neutrophils

Chronic – failure of the acute inflammation to clear the stimulus – longer duration, more tissue destruction

- Presence of lymphocytes and macrophages

Feature	Acute	Chronic
Onset	Fast: minutes or hours	Slow: days
Cellular infiltrate	Mainly neutrophils	Monocytes/macrophages and lymphocytes
Tissue injury, fibrosis	Usually mild and self-limited	May be severe and progressive
Local and systemic signs	Prominent	Less

Types of Acute Inflammation

- Serous inflammation -> exudation of cell-poor fluid into spaces created by injury
    - Eg. in blisters/ burns
    - Usually abundant in proteins
- Fibrinous inflammation -> vascular leaks are large or there is a local procoagulant stimulus
    - Increased vascular permeability -> fibrinogen passes out of the blood -> forms fibrin in the extracellular space
    - Could stimulate ingrowth of fibroblasts and blood vessels when not cleared -> leads to scarring
- Suppurative Inflammation, Abscess -> production of pus (exudate consisting of neutrophils, necrotic cell debris, oedema)

Chronic Inflammation

Characteristics

- Infiltration of mononuclear cells -> eg. macrophages, lymphocytes and plasma cells
    - Macrophages being the main one -> secrete cytokines and growth factors and activate T cells
- Tissue destruction -> sustained inflammatory response
- Attempts at healing -> connective tissue replacement -> fibrosis

Granulomatous Inflammation

- Type of chronic inflammation characterised by groups of activated macrophages (consequently T lymphocytes as well)
- Formation of a granuloma (collection of macrophages and other cells) -> body's attempt to contain the stimulus -> utilises macrophages and T cells

Eg. TB

Wound Healing

Primary intention -> edges of the wound have no tissue loss, stabilised in the same anatomic position

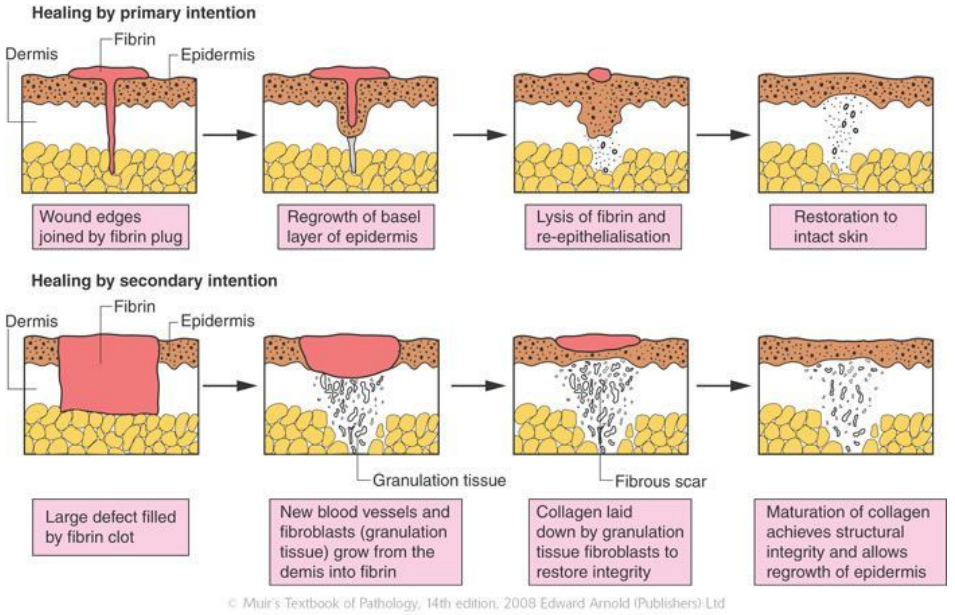
- Quantity of blood clot and granulation tissue is small -> not much space for granulation tissue to from (strong collagen join)
- Minimal wound/scar contraction

Eg. Well repaired lacerations/incisons and well – reduced bone fractures

Secondary intention -> tissue loss has occurred in a wound

- More scar tissue -> causes contraction and slower compared to primary intention
- More inflammation and larger blood clot

Eg. Extraction sockets, poorly reduced fractures, deep ulcers



Healing of Extraction Sockets

- 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
    - Radiographically: restoration of the lamina dura and it is continuous of the alveolar bone

Alveolar Osteitis

- Due to disorder/inability to form a blood clot/dislodgement of the blood clot -> penetration of bacteria -> infection

Clinical Signs:
- Severe pain
- Odour
- Absence of blood clot and debris
- Exposed bone in socket wall
- Inflammation of adjacent mucosa

Pathology:
- Absence of a blood clot -> necrosis of the lamina dura and exposed bone -> infection of the socket walls with bacteria -> triggers an inflammatory response of the bone's stromal connective tissue -> granulation tissue formation -> signs of resorption of the necrotic bone

Anatomy

Exam Style Question 1

Ascending Pathways

Pain, temperature, crude touch and pressure

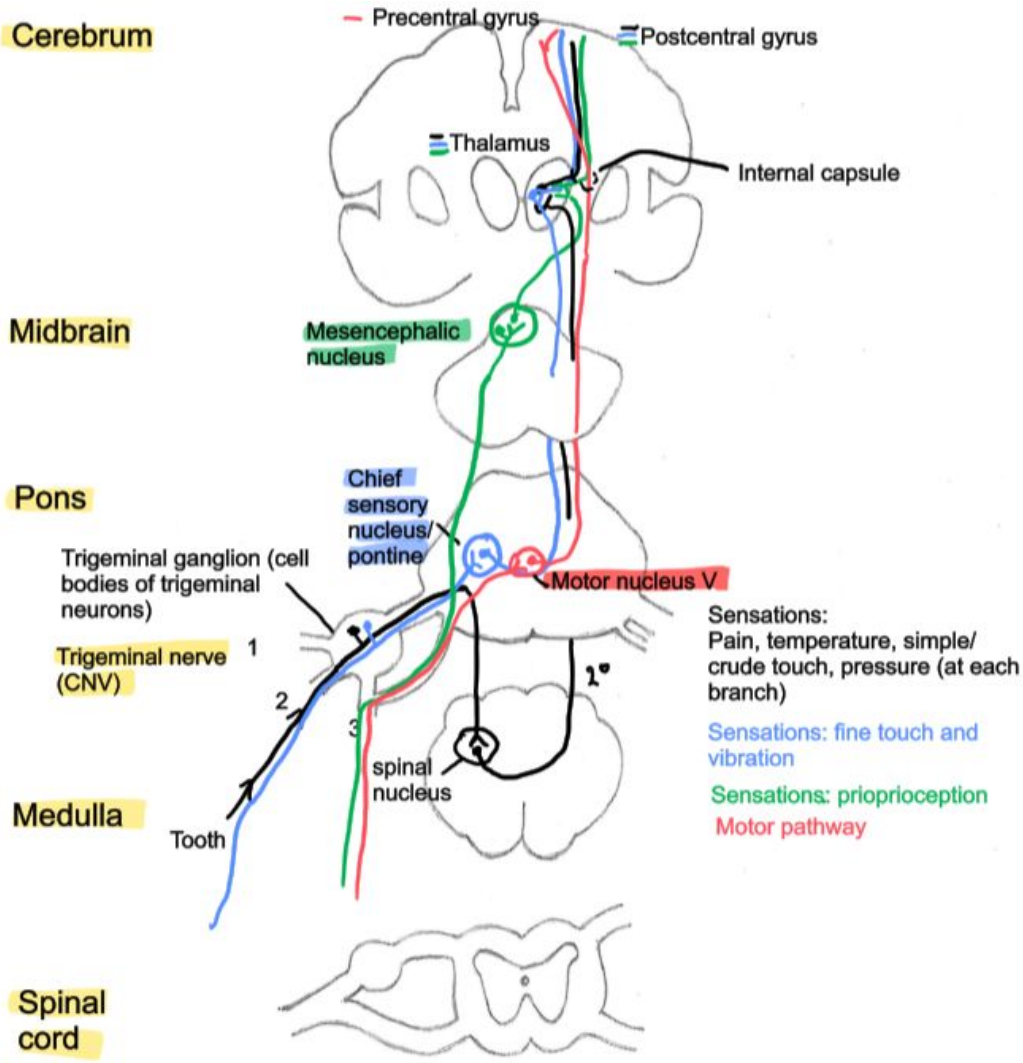
- Spinal nucleus in the medulla -> cross midline -> synapse at VPLN of the thalamus -> postcentral gyrus

Proprioception

- Mesencephalic nucleus in the midbrain -> cross midline -> synapse at the thalamus -> postcentral gyrus

Fine Touch and Vibration

- Chief sensory nucleus (pontine nucleus) in the pons -> crosses the midline -> synapse at the thalamus -> postcentral gyrus

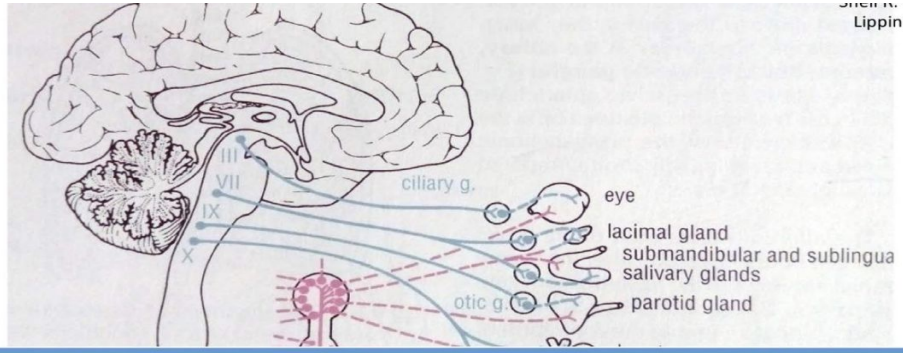


Descending Pathway

Motor (80% of the LMN run contralaterally, 20% ipsilaterally, NOTE: it is the upper motor neuron that crosses)

- (1st order neuron) UMN: Precentral gyrus-> motor nucleus of V
- (2nd order neuron) LMN: motor nucleus of V-> muscle
- 80% of the LMN run contralaterally, 20% ipsilaterally
  - with an UMN lesion, there is still some innervation on the same side
  - With a LMN there is complete paralysis

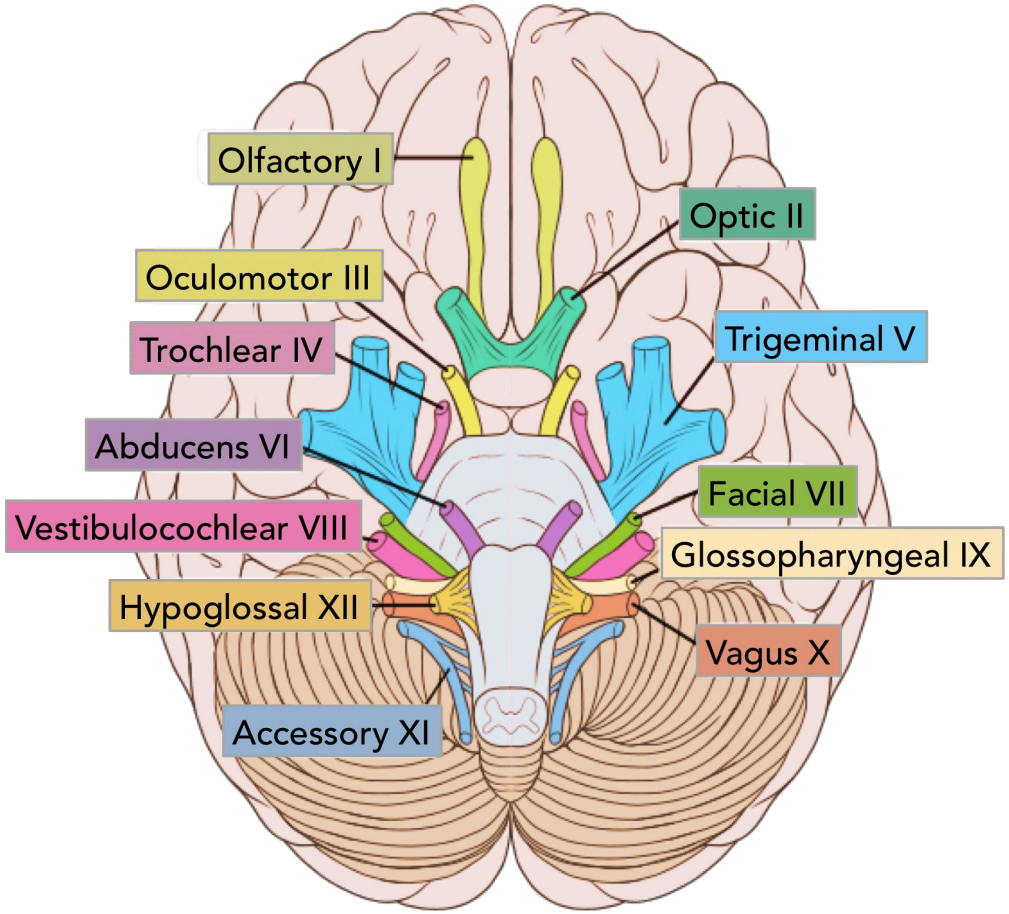
Parasympathetic Ganglion



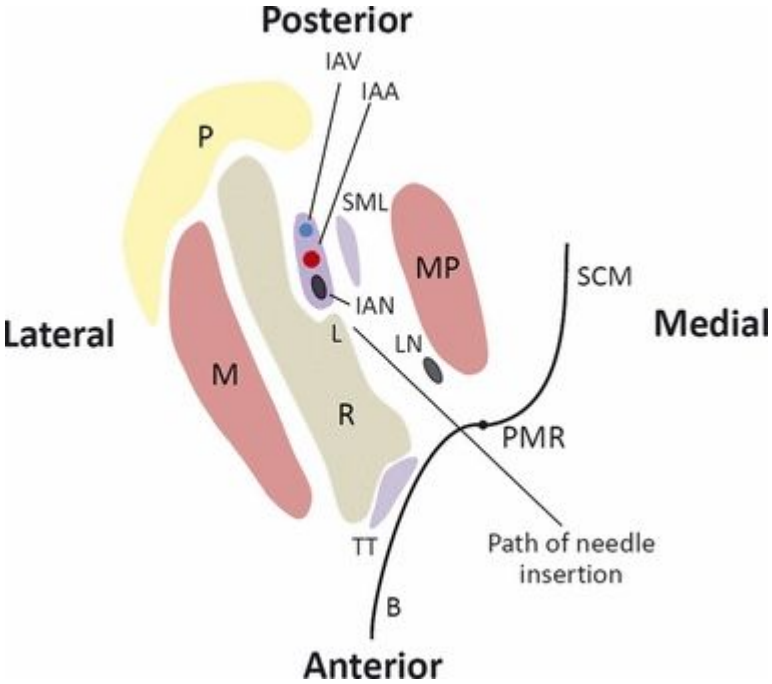
Lippincott Williams & Wilkins, p33.

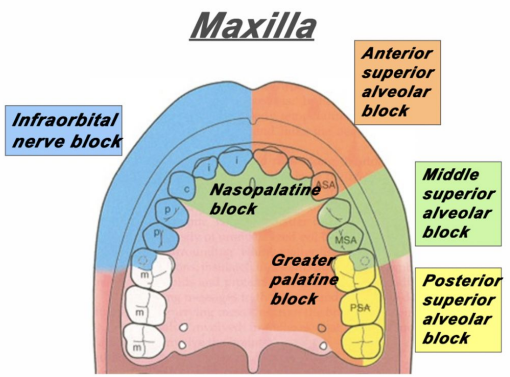
Parasympathetic ganglia in the head region – carries secretomotor fibres			
Ganglion (comes of CNs)	**Path – presynaptic fibres**	**Path – postsynaptic fibres**	**Target gland**
1. Ciliary (V1)	CN III	CN V(1) Short ciliary n.s	Ciliary muscle (accommodation) Pupil (constriction)
2. Otic (V3)	CN IX → Lesser 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

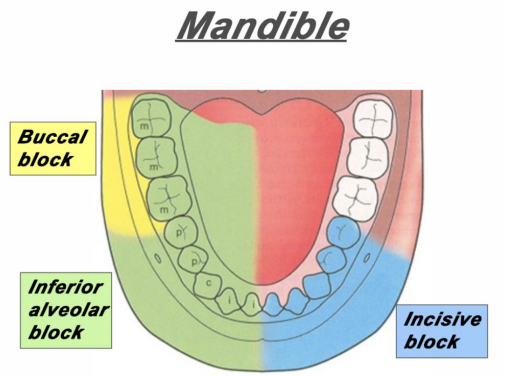
Cranial Nerves



Anatomy relevant to LA







Non Odontogenic Sources of Pain

Odontogenic	Non-odontogenic
Dental hypersensitivity	Musculoskeletal origin
	myofascial
Symptomatic pulpitis	Neuropathic origin
	trigeminal neuralgia
	glossopharyngeal neuralgia
	atypical odontalgia
Symptomatic apical periodontitis	Neurovascular origin
	migraine
	cluster headache
Symptomatic marginal periodontitis	Inflammatory conditions
	sinusitis
Pericoronitis	Systemic disorders
	cardiac pain
	herpes zoster
	sickle cell anemia
	neoplastic disease
	multiple sclerosis
	Psychogenic origin
	somatoform pain disorder

Clinical content

MI

- Understand the basics of MI→ remineralising products for WSL, restorations for caries into dentine
- Management plans for different types of tooth wear, caries
- Rationale for different restorative procedures and materials
- Restorative steps:

E.g.: 26 MO CR with Fuji Bond LC liner (reversible pulpitis):

- Informed consent
- LA- Ziagel (5% lidocaine) applied on 26 buccal fornix and around the 27 gingiva for the rubber dam clamp, 1 carpule of Lignospan special (2% Lignocaine Hydrochloride w/ 1:80,000 adrenaline) for 26 B infiltration for pulpal anaesthesia
- Shade selection in natural light
- RD isolation from 27-23, W3 clamp on 27
- Access cavity with 838, remove infected dentine with slow speed round burs.
- Place cavity conditioner (20% polyacrylic acid) on dentine for 10s, wash thoroughly and dry but don't dessicate
- Apply Fuji Bond LC on dentine mixed to a 1:1 L:P ratio and LC for 20s.
- Apply 37% orthophosphoric etch on enamel for 15s and wash and dry for 40s until enamel is frosted.
- Apply sectional matrix and wedge.
- Place unfilled resin in the cavity and LC for 20s.
- Place CR in 2mm increments and LC for 20s each. Remove sectional matrix and wedge.
- Gross polish
- Check occlusion and remove RD.
- Final polish and post op instructions

Perio

- Understand the different case definitions for gingivitis and periodontitis and the risk factors
- Know the different differential diagnoses and their management
  - Biofilm induced gingivitis
    - Generally painless
  - Periodontitis
  - Drug induced gingival overgrowth
    - Associated with calcium channel blockers, immunosuppressants, anticonvulsants
    - Starts as painless enlargement of the papilla which can enlarge
  - Necrotising gingivitis and periodontitis
    - Patient will usually present due to severe pain, halitosis and may have fever, malaise.
    - Also look for central necrosis of the interdental papilla, pseudomembranous formation
- Remember for periodontitis, the CAL cannot be ascribed to non-periodontal causes
  - E.g., if a patient has PPD> 3 you need to rule out other causes first to determine the CAL is due to periodontitis.
  - For 4/5mm PPD is this due to odema or is there an actual pocket? Also, look for dental cervical caries, trauma due to toothbrushing, deep pockets on D of 7s due to 8 malposition/extraction, vertical root fracture, endodontic lesions draining through marginal periodontium.
- Recession types, aetiology
- Understand action of different instruments (ultrasonic & sonic scalers, curettes, scalers), where to use them, what they look like and how they work

LA

- Anatomy related to LA
- Basic chemistry behind LA
- Understand the different types of LA, and indications and contraindications for each
    - E.g., LA with adrenaline is not to be used with a patient with uncontrolled hyperthyroidism or if they are taking non-selective beta blockers
- Different types of LA procedures and when to use each.
- Complications for LA!!
    - Revist the LA GIL for more details
- Patient communication- consider material risks and when you need to talk to a patient about this in consent

Other topics

- Tooth development, dental age, abnormalities and differential diagnoses - see the tooth development GIL
- ILA content: e.g, RHD and antibiotic prophylaxis, cleft lip and palate
- Radiography
- Social determinants of health
    - Upstream, midstream and downstream factors
    - How SDoH affects a patient's health
    - Different policies and how this affects the different factors
- EBD
- Dental anxiety and fear
    - 4As: ask, assess, acknowledge, address
    - How to manage a patient's dental anxiety
- What is pain and the biopsychosocial model of pain

Exam Style Questions

Question 1

Describe the pathway for the sensation of pain from the 16

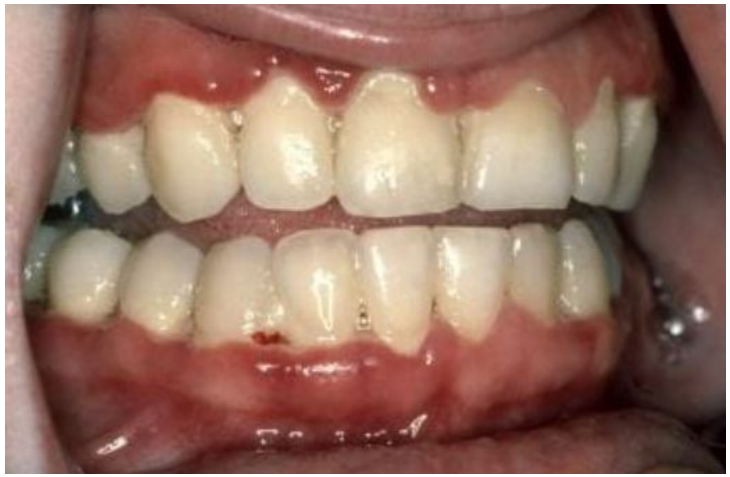
- Right posterior superior alveolar nerve -> Maxillary nerve, exits through the foramen rotundum -> trigeminal ganglion (1st order neuron) -> spinal nucleus runs from the pons relaying down the medulla oblongata (synapses with the 2nd order neuron) -> crosses over and runs up to the thalamus synapses at the VPLN of the thalamus with the 3rd order neuron -> left post central gyrus (contra-lateral)

Question 2

Terry, a 32 year old male patient has presented to your dental clinic with severe pain and bleeding of his gums. Terry is a smoker and recently has had some discussions with his GP about some issues with his immune system but is unsure about the details. All PPD are 3 or under. BOP was 86%.

What is your differential diagnosis?

What is the management for this condition?

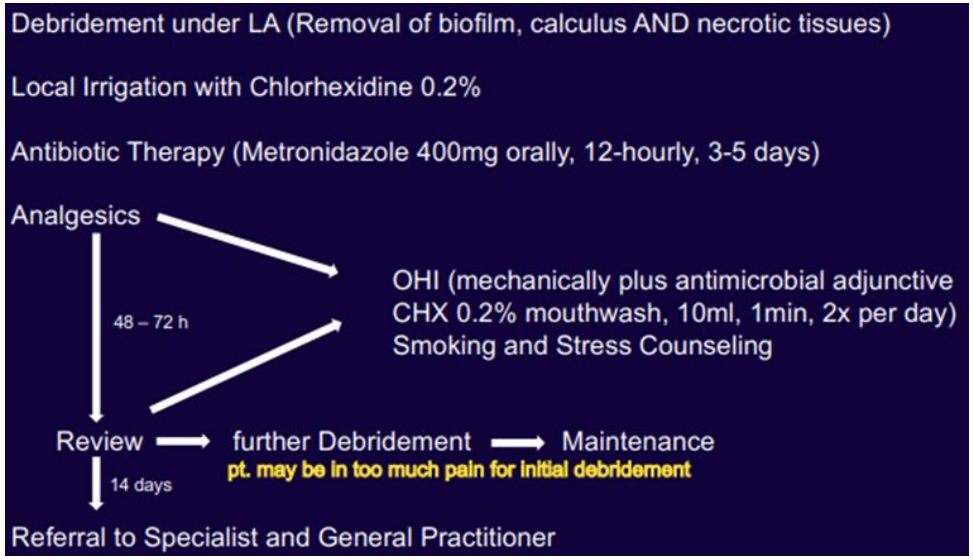


(Fowler, 2022)

Question 2

Differential diagnosis: Generalised necrotising gingivitis (as all PPD 3 or under indicating no CAL associated to periodontal causes). Risk factors: smoking, potentially immunocompromised.

Management: also contact GP to discuss his MHx about immune issues.



Source: Dr Selbach's lecture