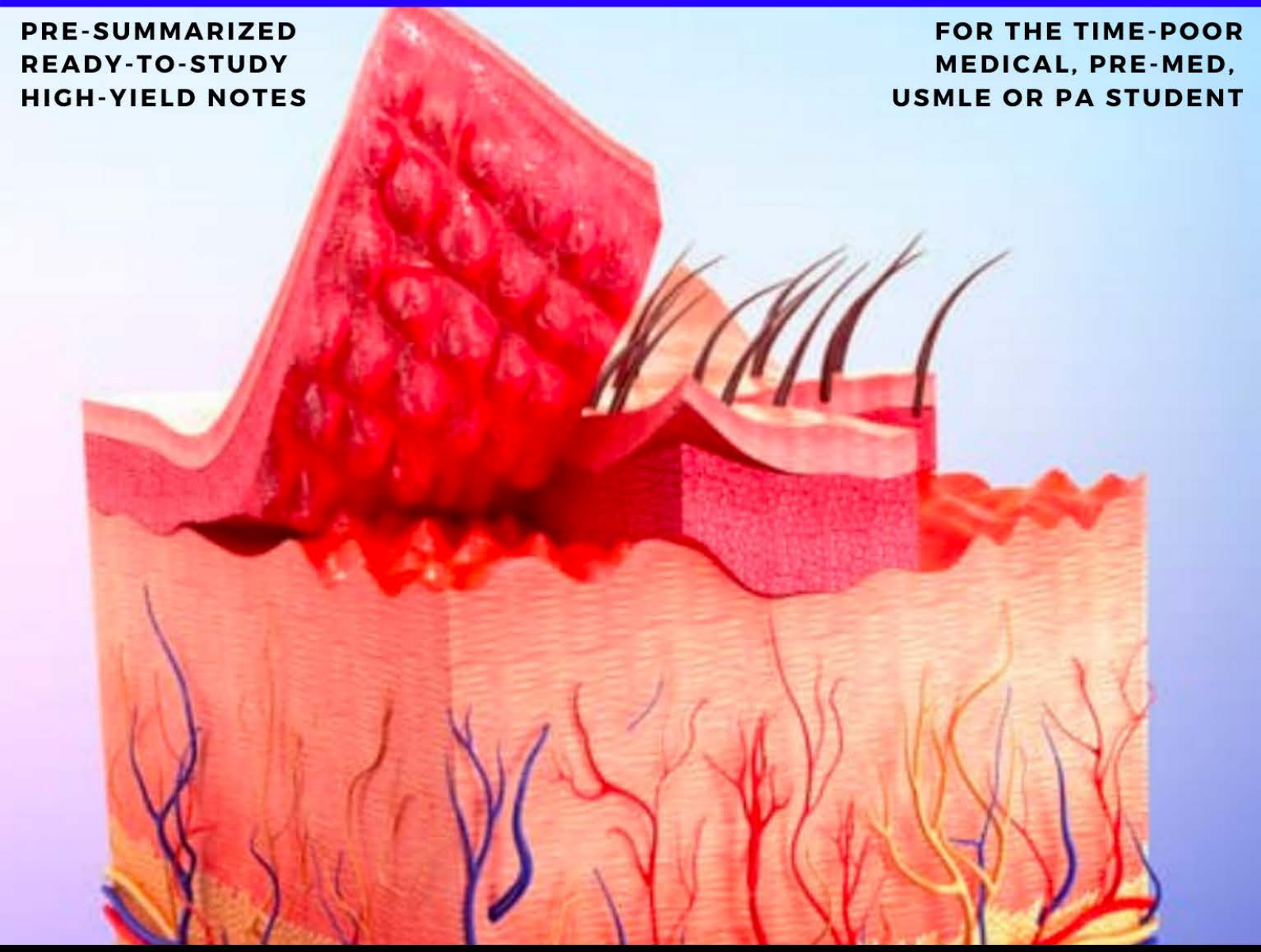
CLINICAL DERMATOLOGY NOTES

PRE-SUMMARIZED
READY-TO-STUDY
HIGH-YIELD NOTES

FOR THE TIME-POOR
MEDICAL, PRE-MED,
USMLE OR PA STUDENT



REGENT STUDIES

MEDICAL NOTES
(MBBS, MD, MBChB, USMLE, PA, & Nursing)
Anatomy, Physiology, Pathophysiology, Pathology, Histology & Treatments

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Table Of Contents:

What's included: Ready-to-study anatomy, physiology and pathology notes of the integumentary system presented in succinct, intuitive and richly illustrated downloadable PDF documents. Once downloaded, you may choose to either print and bind them, or make annotations digitally on your ipad or tablet PC.

Free bonus:

- Fitzpatrick's Colour Atlas And Synopsis of Clinical Dermatology – 8th Edition

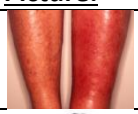
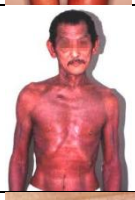
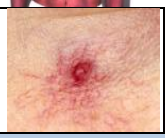
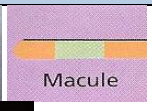
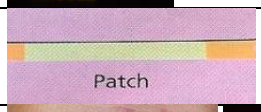
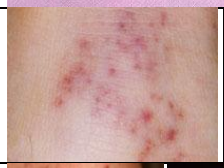
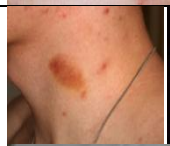
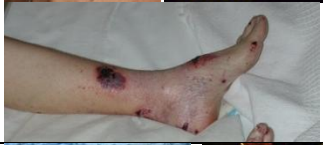
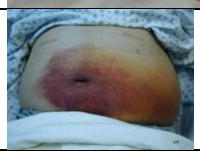
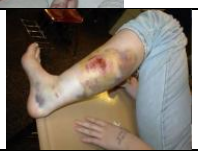
File List:

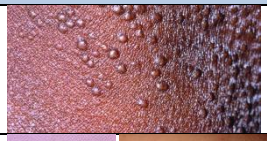
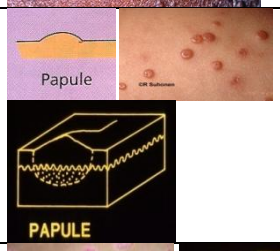
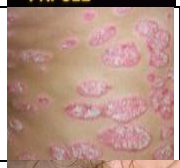
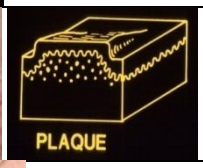
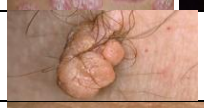
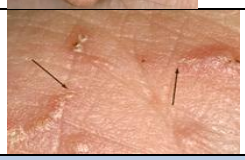
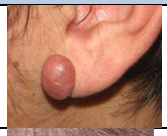
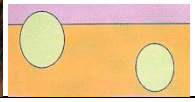
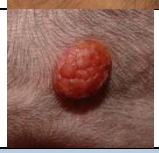
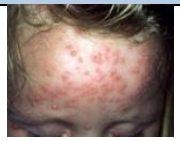
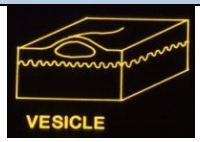
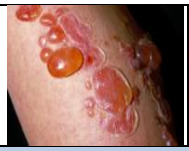
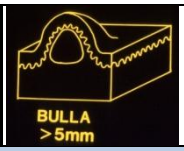
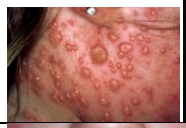
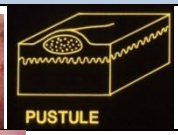
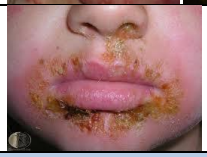
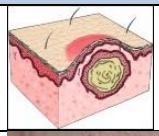
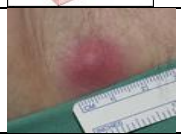
General Principals of Dermatology

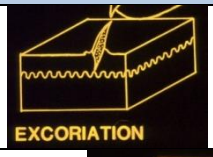
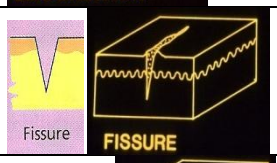
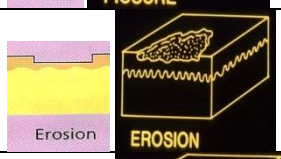
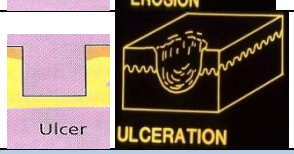
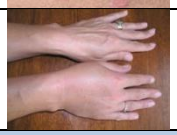
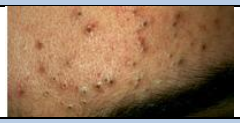
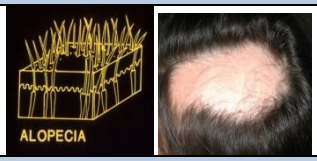
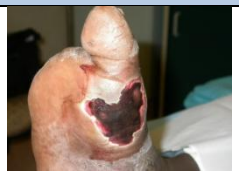
Key words + Definitions:

- **Primary Lesion:**
  - The initial lesion that characterizes a condition.
- **Secondary Lesion:**
  - Over time, the primary lesion may continue to develop or be modified by regression/trauma, producing a "secondary lesion".

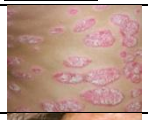
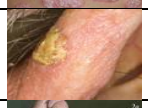
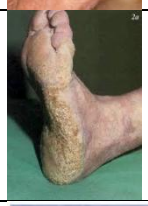
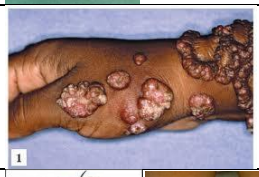
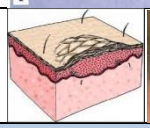
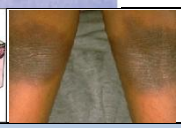
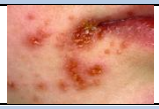
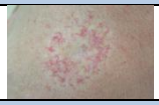
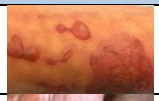
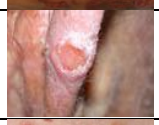
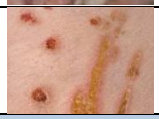
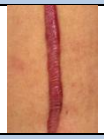
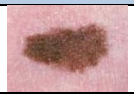
Morphology Terminology – Primary Lesions:

Lesion Name:	Lesion Description:	Picture:
Erythema	Redness due to vascular dilation. (Eg. Cellulitis)	
Erythroderma	Red rash covering >90% of the Body	
Telangiectasia	Permanently dilated dermal blood vessels to the point of being visible; Blanches under pressure	
Macule	A macule is a small, flat are of altered colour, without elevation or depression (nonpalpable). (Eg. Lentigo Simplex, Eg. Freckles)	  
Patch	A patch is a large Macule	
Petechiae	Tiny macules of blood in the skin; Small, non blanching Extravasated RBC's	 
Purpura (May Be Palpable)	A Large Macule or Patch of blood in the skin. Doesn't blanch under pressure. (Eg. A Hickey) Larger area of RBC extravasation (The hallmark of vasculitis – severe inflammation of the blood vessels; If it is this bad in the skin, it's probably this bad elsewhere in the body)	  
Ecchymosis/ Haematoma	A Larger subcutaneous bleed. The resultant swelling = a 'Haematoma'	 

Micropapule	A Very Small raised lesion 1-2mm in diameter.	
Papule	A small raised lesion (less than 0.5cm)	
Plaque	Flat-topped, raised area of the skin. (Usually dermal thickening.) A large Papule more than 2cm in width but NO depth. (Eg. Psoriasis)	 
Papilloma	A nipple-like mass projecting from the skin	
Burrow	A linear Papule caused by a burrowing organism. Eg. Scabies	
Nodule	A raised, solid lesion greater than 0.5cm in width AND depth. (Eg. Keloid Scar)	 
Tumour	A mass of enlarged tissues of more than 1cm diameter	
Vesicle	A clear, fluid-containing elevation of less than 5mm in diameter. (Eg. Chickenpox) (Typically appear clear, but can fill with pus to become a *Pustule*)	 
Bulla	A large Vesicle more than 0.5cm in diameter	 
Pustule	A small Pus-filled vesicle of less than 5mm in diameter	 
Impetiginised (Impetigo):	Covered in Crust, Pustules, & Often Weeping	
Cyst	A cavity containing liquid, semisolid, or solid material	
Abscess	A Pus-containing cavity of more than 1cm in diameter. (NB: a cyst can become infected and fill with pus, becoming an abscess)	

Excoriation	A Scratch - May be Epidermal or may extend down into the Dermis - Often linear - Often Covered with Crust	
Fissure	A deep crack - extends down to the dermis & blood vessels	
Erosion	A superficial <u>incomplete</u> loss of the epidermis. Eg. Superficial Burn	
Ulcer	An area of <u>complete</u> loss of the epidermis and often portions of the dermis and even subcutaneous fat.	
Wheal	A wheal is an elevated, white, compressible area produced by dermal oedema. It typically disappears within 24 to 48 hours	
Angioedema	Oedema which extends to the subcutaneous tissue	
Comedone (Blackhead)	A plug of keratin or sebum blocking a sebaceous orifice	
Alopecia (Hair Loss)	Can be: - Scarring (Permanent loss of hair follicles) - Or Non-Scarring (follicles are still alive and well)	
Eschar (Necrosis)	Patch of Dead Skin (necrosis) – A Thick Crust over an Ulcer or Erosion: – Typically black; Full-thickness skin loss (Dead skin = something bad – RED FLAG)(Implies something vascular → Ischaemic Necrosis)	

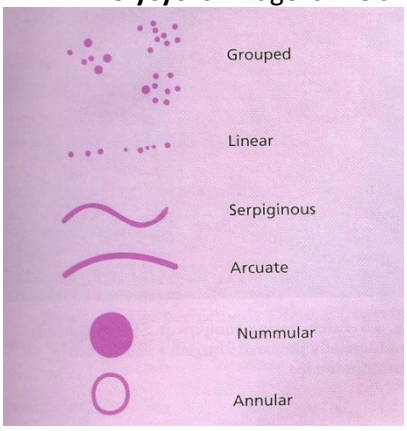
Morphology Terminology – Secondary Lesions:

Lesion Name:	Lesion Description:	Picture:
Scale	Dry, laminated masses of keratin that represent thickened stratum corneum. Eg. Psoriasis	
Keratosis	Thickening of the skin. Eg. Solar Keratosis	
Hyperkeratosis	Large are of Thickened skin → Thick Scale.	
Verrucous	Very Hyperkeratotic – Similar to a Wart	
Lichenification	Palpable epidermal thickening of the skin usually due to friction. Eg. Lichen Simplex	 
Crust	Dried serum, pus, or blood usually mixed with epithelial and sometimes bacterial debris	
Atrophy	Thinning of the skin (epidermal, dermal, or subcutaneous)	
Erosion	A superficial <u>incomplete</u> loss of the epidermis. Eg. Superficial Burn	
Ulcer	An area of <u>complete</u> loss of the epidermis and often portions of the dermis and even subcutaneous fat. Eg. SCC	
Excoriation	A superficial loss of epidermis from scratching or picking, therefore often linear and often covered by crust	
Scar	A pale, firm raised or depressed lesion resulting from skin injury. Eg. Keloid Scar	
Stria (Stretch Marks)	Streak-like, linear, pink/purple/white marks due to changes in Connective Tissue	
Pigmentation	An Increase or Decrease in skin pigmentation. Eg. Birthmark	

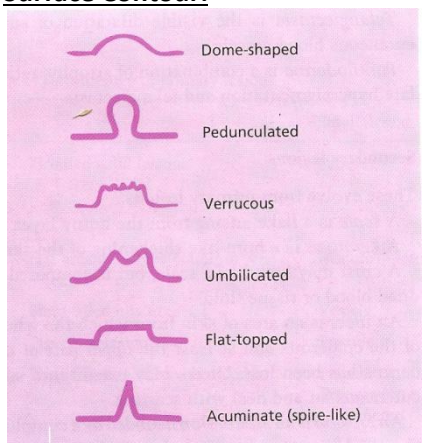
Other Descriptors:

- **Colour:**
  - **Erythema =** Red
  - **Violaceous =** Purple
  - **Slate =** Gray
  - **Hyperpigmented =** Darker than surrounding skin
  - **Hypopigmented =** Lighter than surrounding skin
  - **Bronze =** Red-Brown
  - **Dusky =** Purple-Grey
  - **Variegated =** Many different Colours

- **Relating to Edge of Rash:**
  - **Definition:**
    - Eg. Well Defined
    - Eg. Ill/Poorly Defined
  - **Geometric Shape:**
    - **Nummular** = Round
    - **Annular** = Rink-like
    - **Circinate** = Circular
    - **Arcuate** = Curved
    - **Discoid =** Disc-like
    - **Gyrate/Serpiginous =** Wave-like
    - **Polycyclic =** Edge is like a number of overlapping circles



- **Surface Contour:**



- **Relating to Symptoms:**
  - **Weeping =** Oozing clear fluid from the skin surface
  - **Crusted =** Covered in scabs
  - **Pruritis =** Itchy
  - **Dysaesthesia =** Tingling, Burning, Numbness

- **Symmetry:**
    - Eg. Dermatitis is typically symmetrical.
    - Eg. An infective lesion (Eg. Abscess) is likely to be Unilateral.

- **Is it in Typical Distribution:**
    - Eg. Seborrhoeic Dermatitis (Dandruff) typically occurs on the scalp, forehead, eyebrows & chest.
    - Eg. Atopic Dermatitis typically in the cubital & popliteal fossae.

- **Localised or Universal:**
    - **Universal** – Eg. Chicken Pox.
    - **Localised** – Eg. Herpes Zoster (Shingles) localised to a dermatome.

BASIC ANATOMY & PHYSIOLOGY
Skin

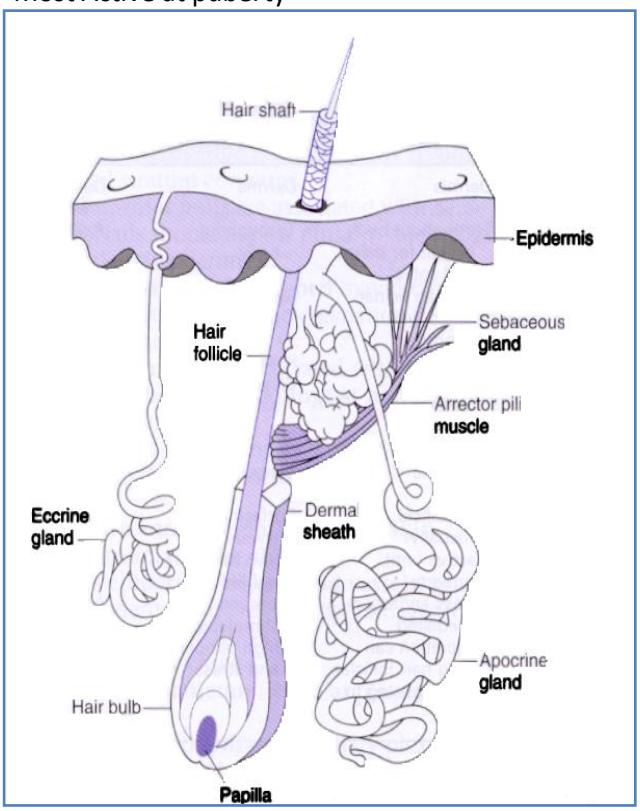
Functions of Normal Skin:

- Mechanical barrier
- Chemical barrier
- Prevent Fluid Loss (Overlapping Cells & Intercellular Lipid → Minimises loss of Water)
- Defence against micro-organisms
- Immunological barrier
- Endocrine organ – (Produces Vitamin-D under UV)
  - Vit. D → Maintains Calcium Homeostasis (↑Ca^+ Absorption in Gut & ↑Renal Ca^+ Retention)
- Melanin → Protects against UV
- Thermoregulation – (Varying blood flow →Allows heat Conservation or Evaporative Cooling)
- Sensory organ

Basic Structure of Normal Skin:

- **3 Layers:**
  - **Epidermis (Top Cellular Layer):**
    - Keratinocytes
    - Melanocytes
    - **The 5 Layers of the Epidermis:**
      - **1. Stratum Germinativum/Basale:**
        - Single layer of cuboidal/columnar cells
      - **2. Stratum Spinosum (Prickle Cell layer):**
        - Several layers of polyhedral cells
      - **3. Stratum Granulosum:**
        - 3-5 layers flattened cells
      - **4. Stratum Lucidum:**
        - (Present only on very thick layers of epidermis (Eg. Glabrous Skin [palms/feet]))
        - = The Lucid layer of Flattened Cells before Stratum Corneum.
      - **5. Stratum Corneum:**
        - 5-50 layers of Flattened, Dead Cells (Squames)
        - Protective Barrier; Holds in Moisture
        - Cytoplasm is filled with **keratin & keratohyalin granules**
  - **Dermis (Middle Fibrous Layer):**
    - Connective Tissue
    - Blood Vessels
    - Nerves (Sensory Receptors & Free Nerve Endings)
    - Hair Follicles + Arrector Pili (the Smooth Muscle)
      - **NB: Skin without hair** – (Eg. Palms & Soles) = ***"Glabrous Skin"***.
      - **NB: Skin With Hair** – (Ie. Rest of the body) = ***"Non-Glabrous Skin"***.
    - Glands (Sweat, Sebaceous)
    - **Contains Some Cells:**
      - **Fibroblasts**: Synthesis and degradation of connective tissue
      - **Histiocytes/Macrophages**: Phagocytic cells
      - **Mast cells**: Secretory cells →Vasoactive Mediators (histamine) → Skin Allergies.
      - **Lymphocytes:** Small number collect around blood vessels in normal skin
    - **2 Layers:**
      - **(R) - Reticular Layer** (thick Collagen, lower layer) - much stronger
      - **(P) - Papillary layer** (fine Collagen, upper layer) - weaker
  - **Hypodermis (Fat Layer):**
    - Adipose Tissue
    - **Functions:**
      - Insulates the body
      - Stores Energy

- **Glands:**
  - **Sebaceous (Sebum) Glands:**
    - **Associated with hair follicles (The Pilosebaceous Unit)**
      - (:. Not found on Palms/Soles)(Most found on Face & Scalp)
    - **Holocrine Secretion:**
      - (Ie. Secretion via complete destruction of cells)
      - **Produce oily sebum:**
    - **Stimulated by Androgens:**
      - Very Active at puberty
  - **Eccrine (Sweat) Glands:**
    - On most of the body (Scarce on the back)
    - **Simple, Coiled Tubular Glands:**
      - Secretory Coil (deep in Dermis) – Secrete the Water & Electrolytes
      - Sweat Duct – Reabsorb Na^+ Ions from the sweat.
    - **Clear watery secretion**
      - Person can perspire several liters per hour
    - **Stimulated by High Temperature and Stress**
  - **Apocrine (Pheromone) Glands:**
    - Associated with hair follicles
    - **Large complex gland:**
      - Located in Dermis
      - Duct Opens into Hair Follicle.
    - **Viscous, Milky Secretion – (protein and cellular debris):**
      - **Produces pheromones**
        - Bacterial action is required for odor production
    - **Stimulated by Androgens:**
      - Most Active at puberty

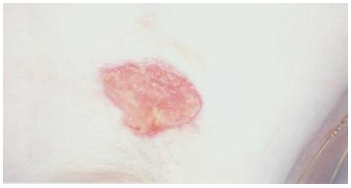


Causes of Skin Injury:

- **Physical Agents:**
  - Mechanical trauma
  - Thermal Burns
  - Cold
  - Electrical
  - Radiation (Eg. In Radiotherapy)
- **Hypoxia:**
  - Ischaemia (Eg. In Peripheral Vascular Disease)
- **Chemicals:**
  - Acid/Alkali
  - Phosphorus
- **Infectious:**
  - Bacteria
  - Vicuses
- **Autoimmune:**
  - Scleroderma
  - Lupus Erythematosis
- **Genetic:**
  - Histiocytosis X

Mechanisms of Healing:

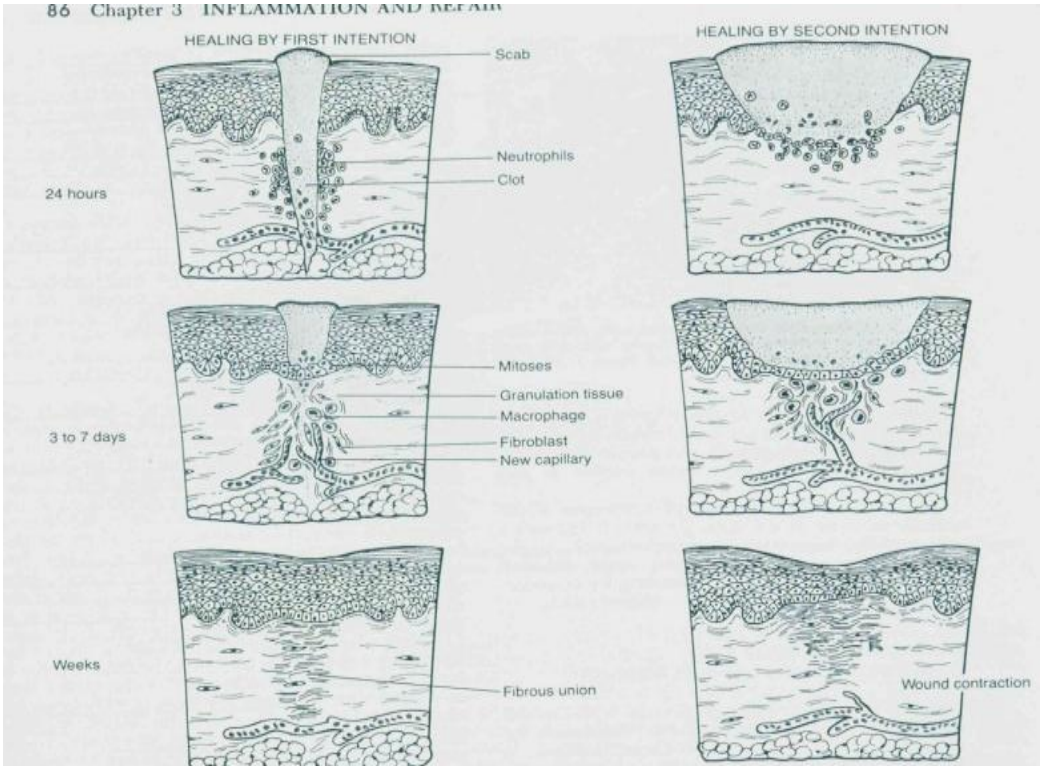
- **Acute Inflammation:**
  - Vasodilation
  - Increased Permeability → Stasis
  - Leukocyte Margination/Migration
  - Phagocytosis of Damaged/Dead Tissue/Organisms. + Enzyme Release
- **Granulation Tissue:**
  - **3-5 Days After Injury**
  - **Occurs only in Deeper Cuts/Injuries. (Not Superficial Injuries)**
  - **Angiogenesis** – (Migration + Proliferation of Endothelial Cells)
    - Driven by FGF (Fibroblast Growth Factor) + VEGF (Vasculo-Endothelial Growth Factor)
  - **Fibrosis -** Fibroblast Migration + Proliferation
    - Driven by PDGF (Platelet-Derived Growth Factor) + TGFB (Transforming Growth Factor-B)



- **Collagen Synthesis:**
  - A Triple Helix Protein
  - Synthesis is Vit.C Dependent.
  - As time goes by, the Collagen Scar gets Stronger:
    - At 1wk, the scar is weak.
    - Strengths peaks @ 3mths.
  - Metalloproteinases (collagenases, Gelatinases) → Degrade Collagen.

Healing By Primary Intention Vs. Secondary Intention:

- **Primary Intention:**
  - Wound edges are CLOSE together
  - Tends to be Quick
  - Cells of the Basal Layer of the Epidermis lines the Surface of the Wound (within 1-2 Days)
- **Secondary Intention:**
  - Wide, open wound. (Edges are FAR apart)
  - Takes a lot Longer
  - Granulation Tissue Fills the Wound → Epithelialisation from Wound Margins



Skin Grafts Vs. Skin Flaps:

- **Skin Grafts:**
  - Has been totally detatched from its original location
  - **:. Has NO intrinsic Blood Supply**
    - :. Must be grafted onto Vascularised Tissue
    - It will initially survive via Simple Diffusion of Nutrients
    - Eventially, Neovascularisation → New Blood Supply.
  - **Types:**
    - **Full Thickness Graft (FTG)**
      - All of the Dermis & Epidermis
    - **or Split Skin Graft (SSG)**
      - All of the Epidermis & some of the Superficial Dermis
- **Skin Flaps:**
  - Has NOT been detached from its original location
  - :. Still Has Intrinsic Blood Supply
  - **Indications:**
    - Poor Vascularity of Location (eg. Bare bone, Bare tendons)
    - Vital Structures (Eg. Exposed Vessels/joints)
    - Cosmetics (Eg. Face) – Usually gives a better result than a skin graft.
  - **3 Basic Types:**
    - Rotation
    - Advancement
    - Transposition
- **"Free" Flaps:**
  - = Basically a Skin *Graft*, but the Blood Supply is Reconstituted using Microsurgery to reconnect the Artery and Vein.

A&P
Skin Structure & Function - Detailed

Functions of Normal Skin:

- Mechanical barrier
- Chemical barrier
- Prevent Fluid Loss (Overlapping Cells & Intercellular Lipid → Minimises loss of Water)
- Defence against micro-organisms
- Immunological barrier
- Endocrine organ – (Produces Vitamin-D under UV)
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    - Blood Vessels
    - Nerves (Sensory Receptors & Free Nerve Endings)
    - Hair Follicles + Arrector Pili (the Smooth Muscle)
      - **NB: Skin without hair** – (Eg. Palms & Soles) = ***"Glabrous Skin"***.
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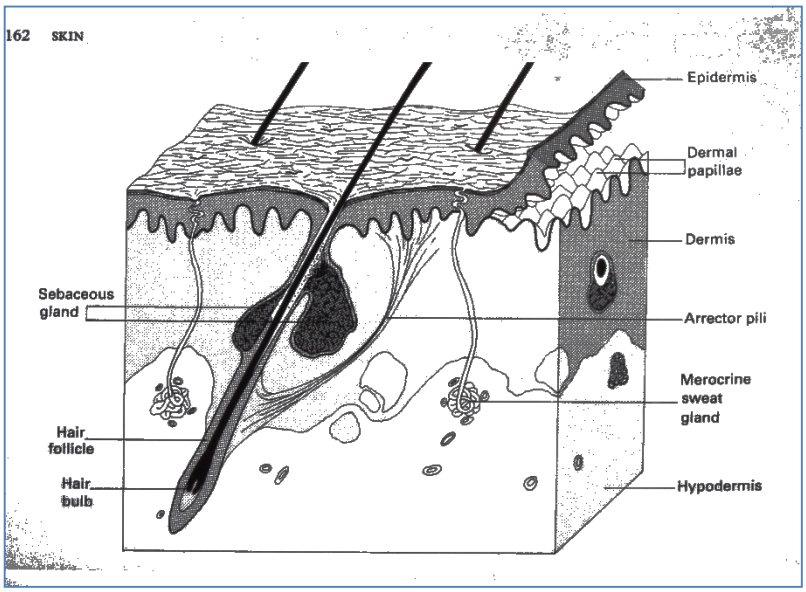
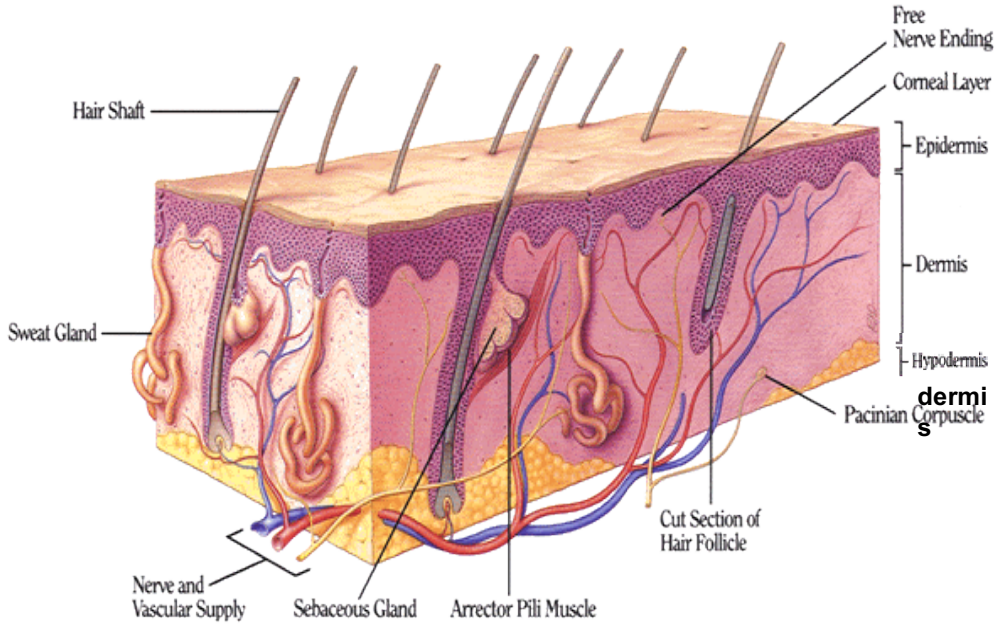
    - Glands (Sweat, Sebaceous)
  - **Hypodermis (Fat Layer):**
    - Adipose Tissue



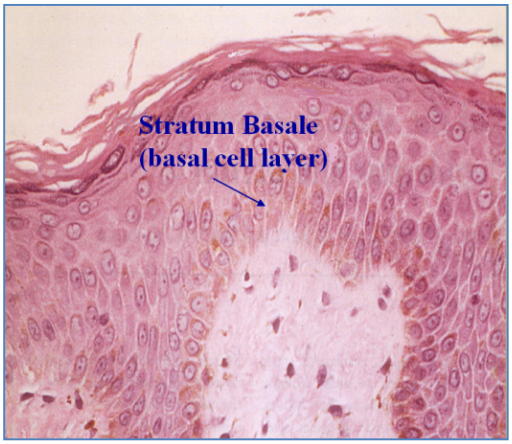
Structure of Skin



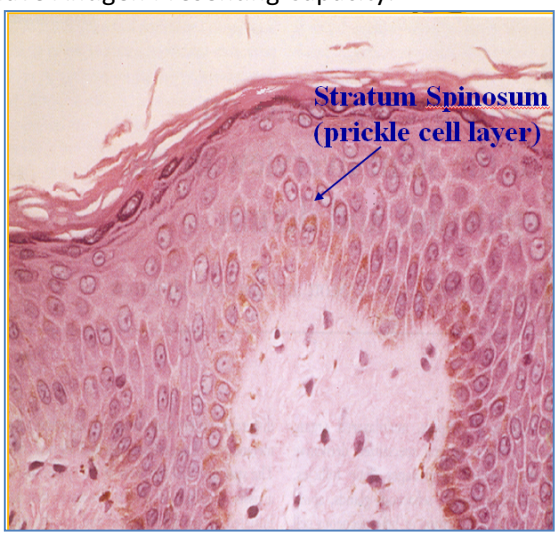
LAYER 1: THE EPIDERMIS:

- **Structure of the Epidermis:**
  - Most Superficial Layer of Stratified Squamous Keratinised Epithelium.
  - Avascular
  - Almost Entirely Cellular:
    - 95% of Epidermal Cells are *Keratinocytes*. (Produce Keratin)
    - Other Cells include: *Melanocytes, Merkel Cells & Langerhans Cells.*
    - Continually regenerating (Rate is just sufficient to replace cells lost from the surface)
  - Protective barrier
  - Approximately 0.1 to 1mm thick

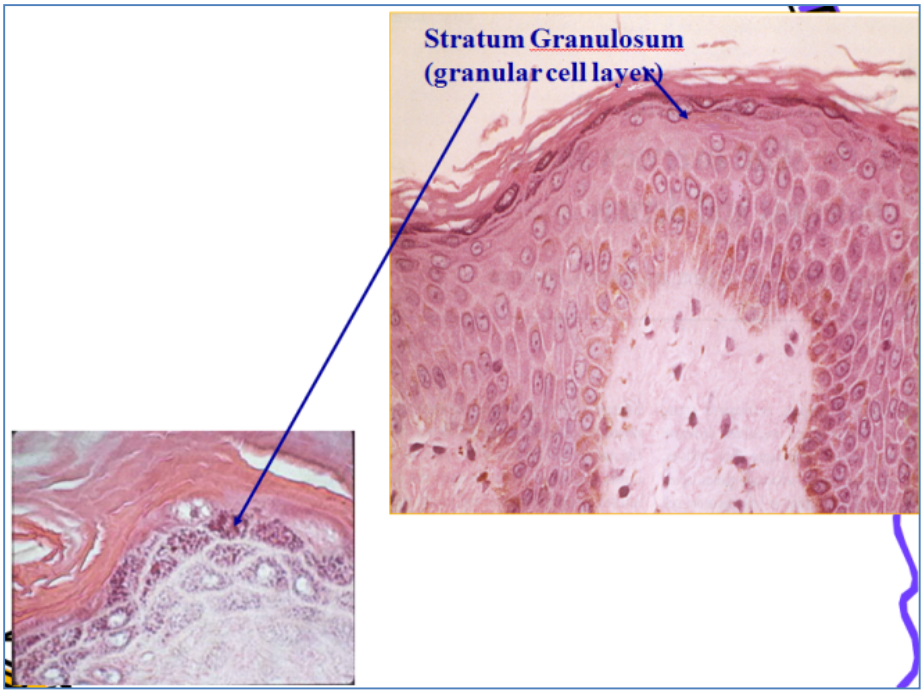
- **The 5 Layers of the Epidermis:**
  - **1. Stratum Germinativum/Basale:**
    - Single layer of cuboidal/columnar cells
    - **Basal Cells** take 14 days to differentiate → Keratinocytes
    - Keratinocytes take 14 days to be shed off.
    - Strongly adherent to the Basement Membrane by **Hemidesmosomes**.



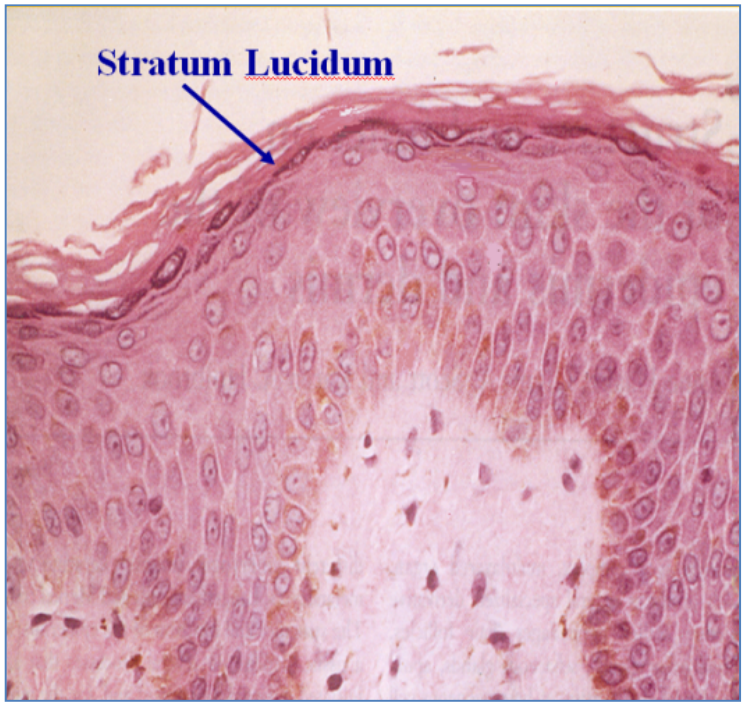
  - **2. Stratum Spinosum (Prickle Cell layer):**
    - Several layers of polyhedral cells
    - Keratin-Tonofibrils Insert into interconnecting desmosomes from adjacent cells.
    - **Keratinocytes** Contain **'Lamellar (lipid containing) bodies'**:
      - Are Secreted into the Extracellular spaces (Sticks Cells Together)
        - → Forms a water-barrier.
        - →Also acts to cement keratinized squames together in Stratum Corneum
    - **Langerhans Cells:**
      - Like Macrophages
      - Have Antigen Presenting Capacity.



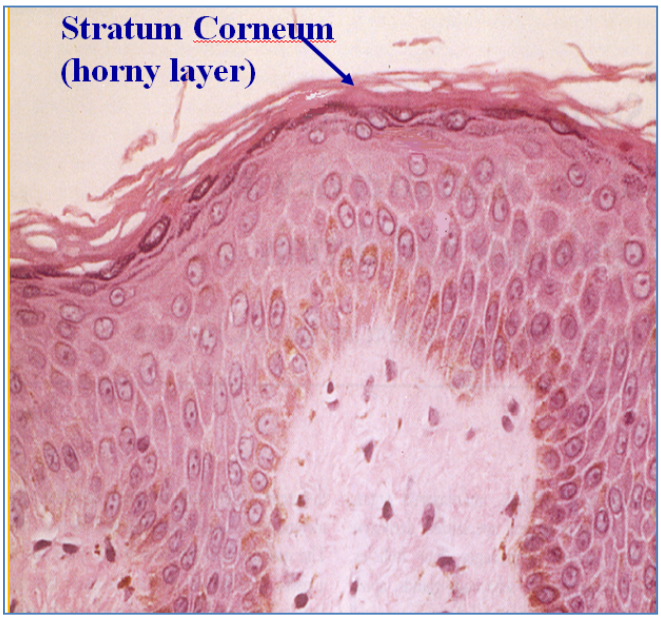
- **3. Stratum Granulosum:**
  - 3-5 layers flattened cells
  - Loss of nuclei
  - **Keratinocytes:**
    - **Keratohyalin granules** (Composed of *Profilaggrin*, *Keratin Filaments*, & *Loricrin*)
      - Keratohyalin = Protein involved in Keratinisation.
      - NB: Eczema is probably due to a mutation in Profilaggrin.
    - **Lamellar Body secretions:**
      - Lamellar Body Contents Discharged into Intracellular Spaces
        - (Lipids, Enzymes)
      - – Acts like the "*Mortar*" between the cellular *"Bricks"*.

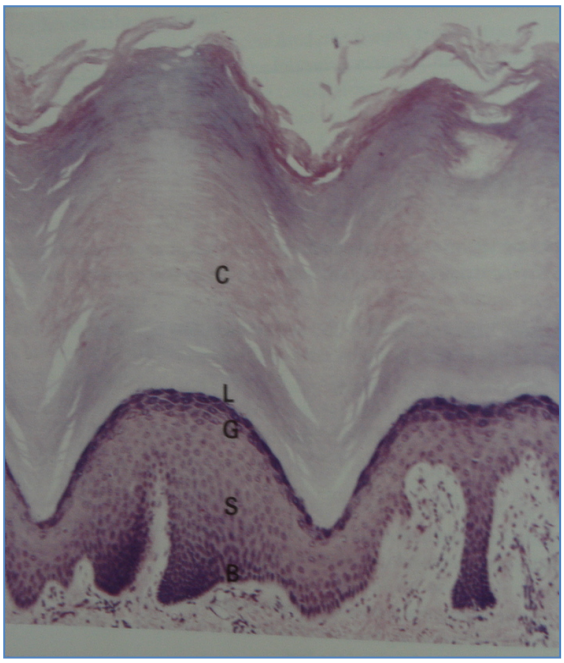


- **4. Stratum Lucidum:**
  - (Present only on very thick layers of epidermis (Eg. Glabrous Skin [palms/feet]))
  - = The Lucid layer of Flattened Cells before Stratum Corneum.
    - Cells have No nuclei or organelles.



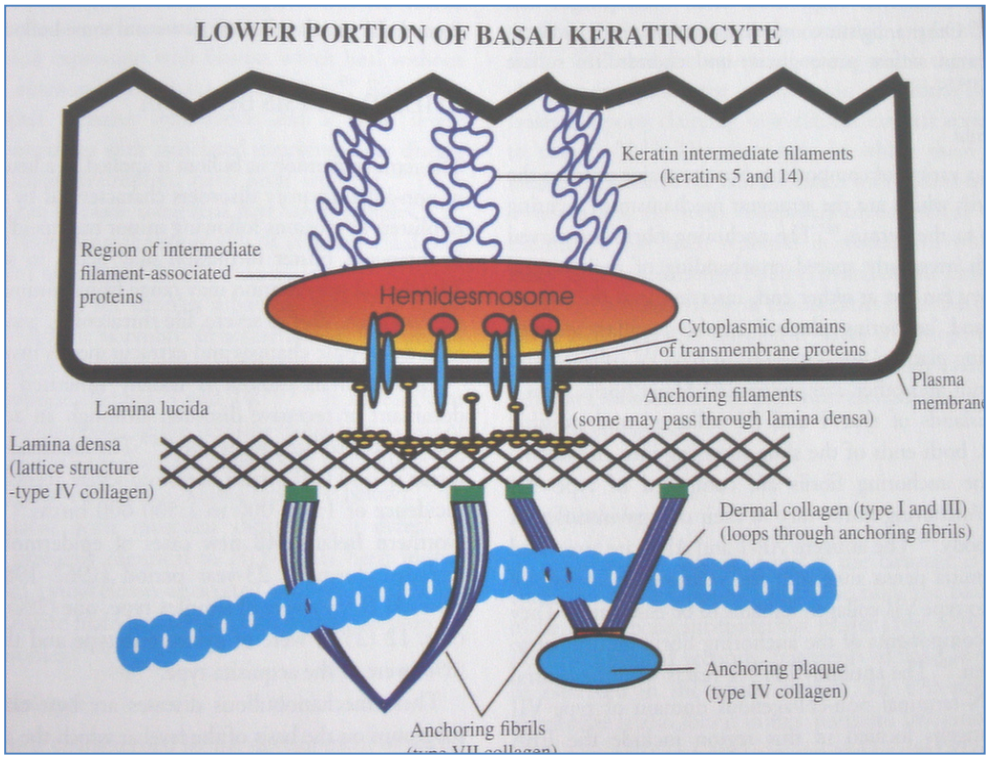
- **5. Stratum Corneum:**
  - 5-50 layers of Flattened, Dead Cells (Squames)
    - Devoid of Nuclei & Organelles.
  - Protective Barrier; Holds in Moisture
  - Cytoplasm is filled with **keratin & keratohyalin granules**
  - Cells are stuck together by contents of **Lamellar Bodies**.
  - Thicker on Glabrous Skin (Palms and soles)



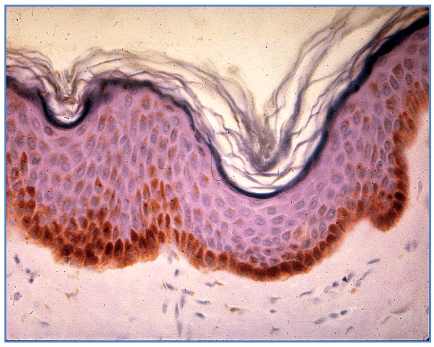


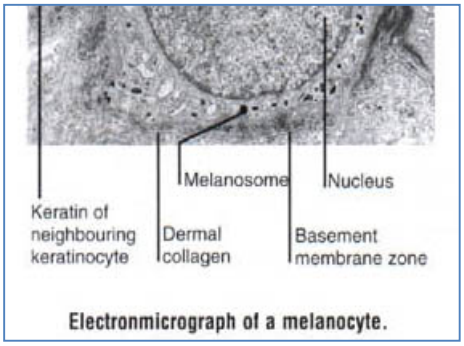
C = Corneum; L = Lucideum; G = Granulosum; S = Spinosum; B = Basale

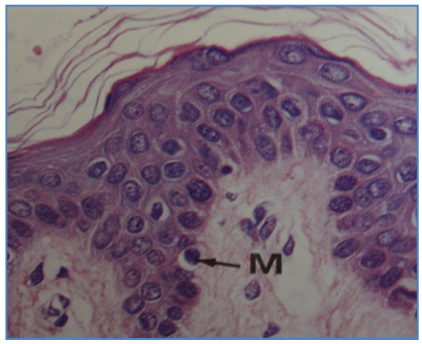
- **Cellular Component of the Epidermis:**
  - ***Keratinocytes* – (In all Layers):**
    - Form the *Keratinized* Stratified Squamous Epithelium
    - Contain **Keratin** (Protein) Intermediate Filaments in the Cytoplasm
    - **Keratin:**
      - **Two types of epithelial keratins:**
        - Type I (acidic)
        - Type II (basic)
        - (NB: Each are produced in Equal Amounts)
      - **Keratin Proteins Polymerise as Heterodimers:**
        - Type 1 + type 2 → Polymers
      - **Keratin Polymers aggregate → Keratin Filaments**
      - **Keratin Filaments aggregate → Tonofilaments**
    - **"Keratinisation" – Keratinocytes Differentiate as they Migrate Upwards:**
      - Formation of Keratin proteins in Cytoplasm.
      - →Aggregation of the Keratin Filaments into **'Tonofilaments'**. (Like a rope)
        - (*Tonofilaments* →Anchor Desmosomes to the Cytoskeleton)
    - **Join to Adjacent Cells via:**
      - **Desmosomes:**
        - Intercellular attachments between keratinocytes
      - **Hemidesmosomes:**
        - Attachment of the Basal Keratinocytes (Epidermis) to the Basement Membrane (Dermis)
        - Very Tight Connection



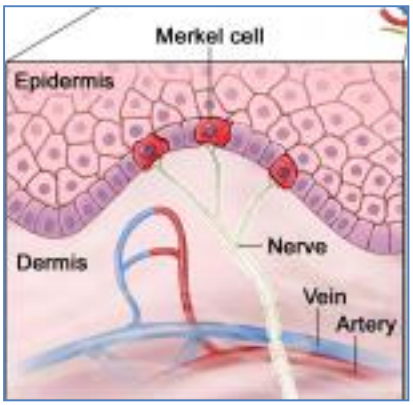
- ***Melanocytes*** **– (In Stratum Basale):**
  - **Pigment Cells – Synthesise Melanin Pigment**
    - Melanin contained in *Melanosomes*
    - Transferred to keratinocytes via Dendritic Processes.
    - Melanin Absorbs UV radiation
  - **Located in Stratum Basale, Dermis and Hair Follicles.**
  - **NB: Differences in Racial Pigmentation:**
    - Due to ↑Melanocyte *Activity* (NOT Melanocyte *Number*)



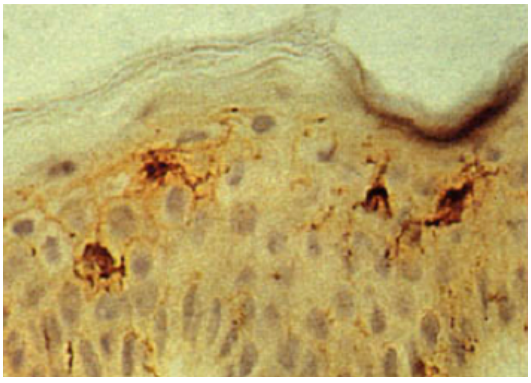




- ***Merkel Cells*** **– (In Stratum Basale):**
  - Nerve Ending Associated
  - Contain small, dense Granules of Catecholamines
  - **Located in Stratum Basale**
  - - Probably Neuro-Endocrine Function
    - Sensory mechanoreceptors
    - Closely associated with Nerve Terminal Filaments (free nerve endings)



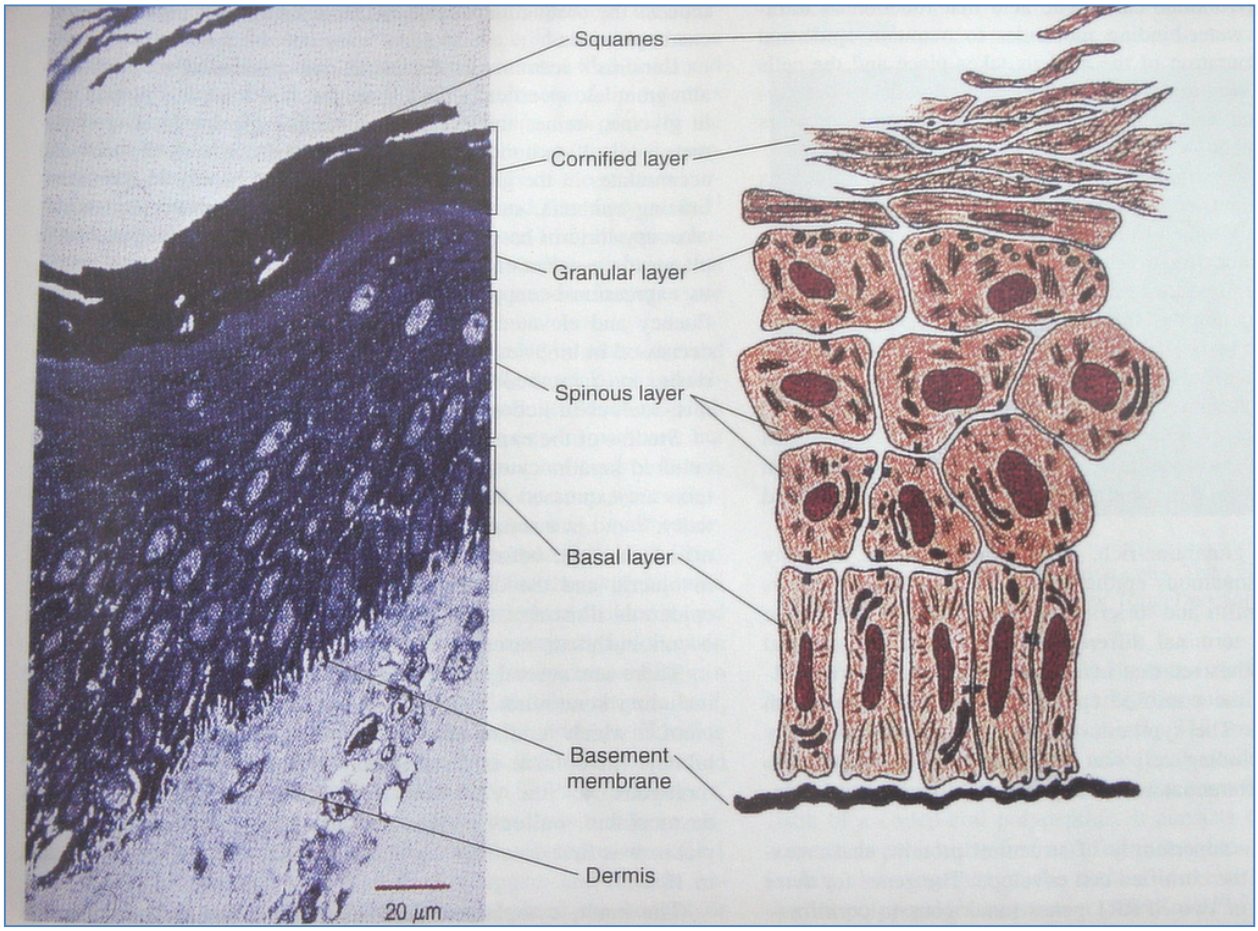
- ***Langerhans Cells*** **– (In Stratum Spinosum):**
  - Antigen Presenting Cells of the Immune System
  - Have long Dendritic Processes that radiate through the Epidermis.
  - **Located in the Stratum Spinosum**
  - Migrate through Epidermis and Dermis to Lymph Nodes
  - - Immune Function.



- **Epidermopoiesis (Epidermal Renewal) & Desquamation (Shedding of Skin):**
    - **(NB: Rate of Epidermopoiesis must = Rate of Desquamation.)**
        - **Stimulated by Growth Factors:**
            - Epidermal Growth Factor (EGF)
            - Transforming Growth Factor a (TGFa)
            - Fibroblast Growth Factor (FGF)
        - **Inhibited by Cytokines:**
            - Interferon Gamma (IFNy)
            - Tumour Necrosis Factor (TNF)

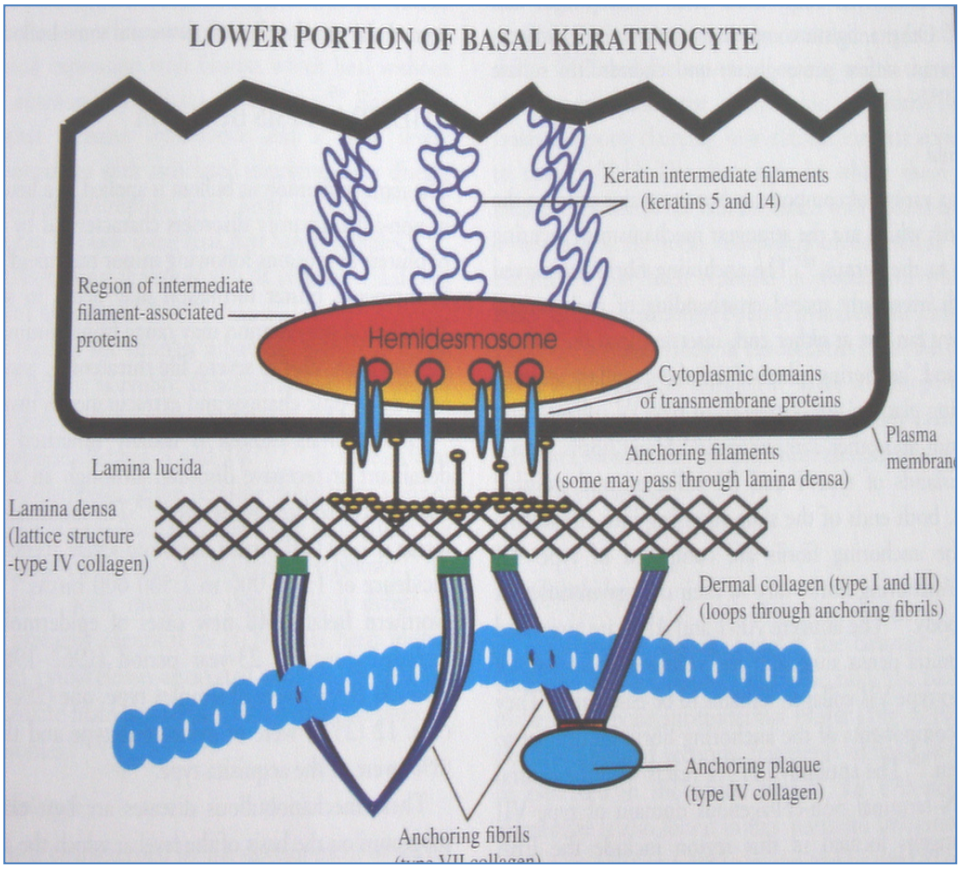
    - **Epidermopoiesis (Epidermal Renewal):**
        - Epidermis continually renews itself
        - - by Basal (Columnar) Cell Division
        - Turnover rate ≈6 Weeks
        - **Basal Cells Become → Keratinocytes (Epidermal Cells):**
            - Via Keratinization = Synthesis of Keratin Protein.
        - Transit time to the stratum corneum is approx. 14 days

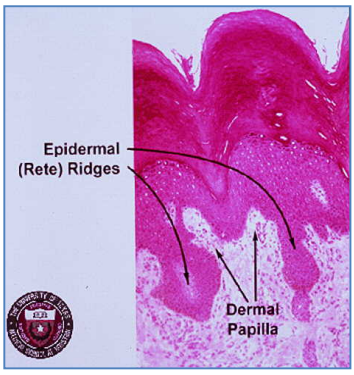
    - **Desquamation (Shedding of the Skin):**
        - Occurs when desmosomes are degraded (Mediated by Cholesterol Sulphatase)
        - Or by Wear & Tear.
        - Desquamation requires another 14 days



DERMAL-EPIDERMAL JUNCTION:

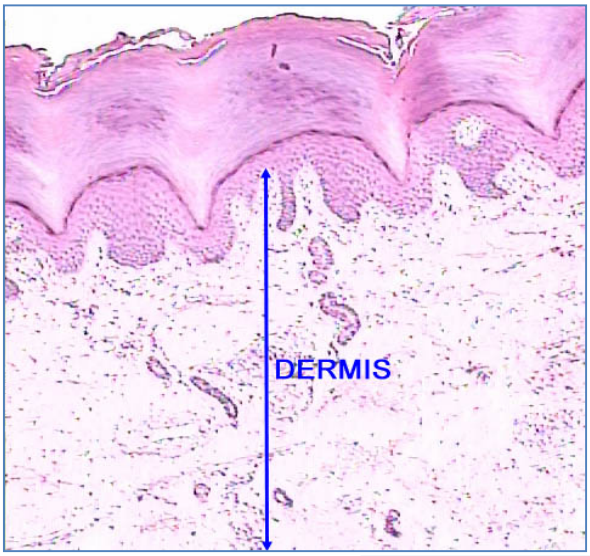
- **Dermal-Epidermal Junction (Statum Basale : Basement Membrane):**
  - Where the Stratum Basale of the Epidermis attaches to the Basement Membrane.
  - **Stratum Basale:**
    - **Basal Cells** (Basal Keratinocytes)
      - ***Hemidesmosomes*** on Basal Cells attach the Epidermis to the Basement Membrane.
    - **Melanocytes** are Interspersed amongst the Basal Cells.
      - Large Dendritic Cells
      - Responsible for Melanin Pigment Production.
    - **Merkel Cells:**
      - - Probably Neuro-Endocrine Function
      - Contain small, dense Granules of Catecholamines
  - **Basement Membrane:**
    - **Lamina Lucida** - anchoring transmembrane filaments of the Hemidesmosomes.
    - **Lamina Densa** - (Lattice structure of type IV Collagen)
  - **Hemidesmosomes:**
    - Important in maintaining adhesion between Dermis & Epidermis.
    - Associated with Keratin Filaments & Keratin Tonofibrils
  - **Epidermis & Dermis join in a ripple-like fashion for extra strength (in addition to desmosomes):**
    - **Rete Ridges** (Epidermis)
    - **Dermal Papilla** (Dermis)

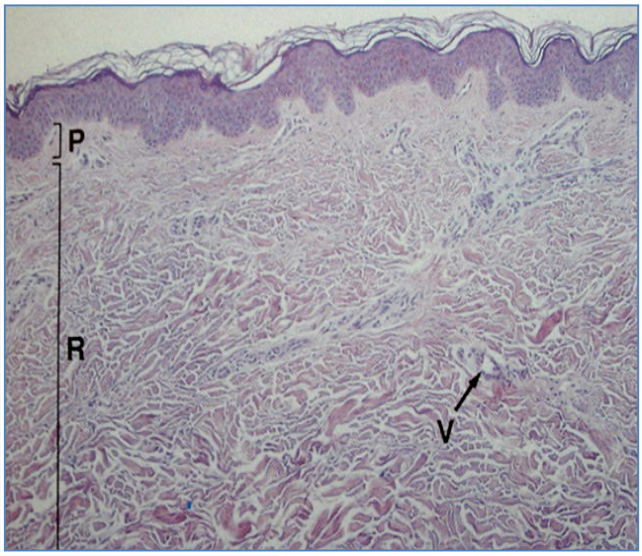


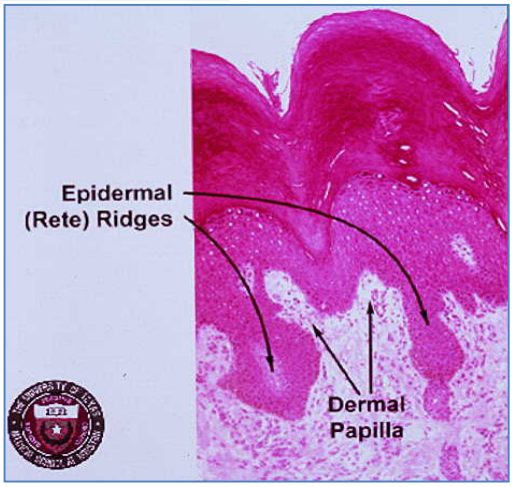


LAYER 2: THE DERMIS:

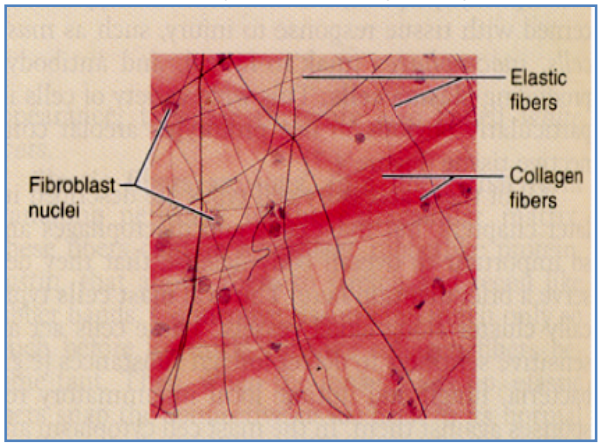
- **Structure of the Dermis:**
  - **Mostly Extracellular Matrix:**
    - Extracellular Collagen
    - Elastin
  - **Contains Some Cells:**
    - **Fibroblasts**: Synthesis and degradation of connective tissue
    - **Histiocytes/Macrophages**: Phagocytic cells
    - **Mast cells**: Secretory cells →Vasoactive Mediators (histamine) → Skin Allergies.
    - **Lymphocytes:** Small number collect around blood vessels in normal skin
  - **Also contains Neurovascular & Other Auxillary Skin Structures:**
    - Blood Vessels
    - Nerves
    - Sweat Glands
    - Hair Follicles (And Arrector Pili – The Smooth Muscle of the Hair Follicle)
  - **2 Layers:**
    - **(R) - Reticular Layer** (thick Collagen, lower layer) - much stronger
    - **(P) - Papillary layer** (fine Collagen, upper layer) - weaker



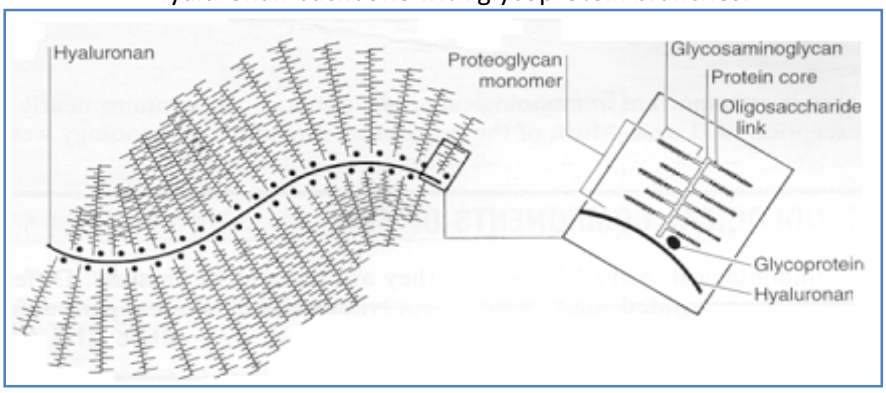




- **Connective Tissue of the Dermis:**
  - **#1 Collagen** = Very tough, fibrous protein – *High Tensile Strength*
    - The Predominant Protein of the Dermis (As opposed to Keratin of the Epidermis)
  - **Elastin** = Provides Elasticity to Skin. – *Deformity with Memory.*

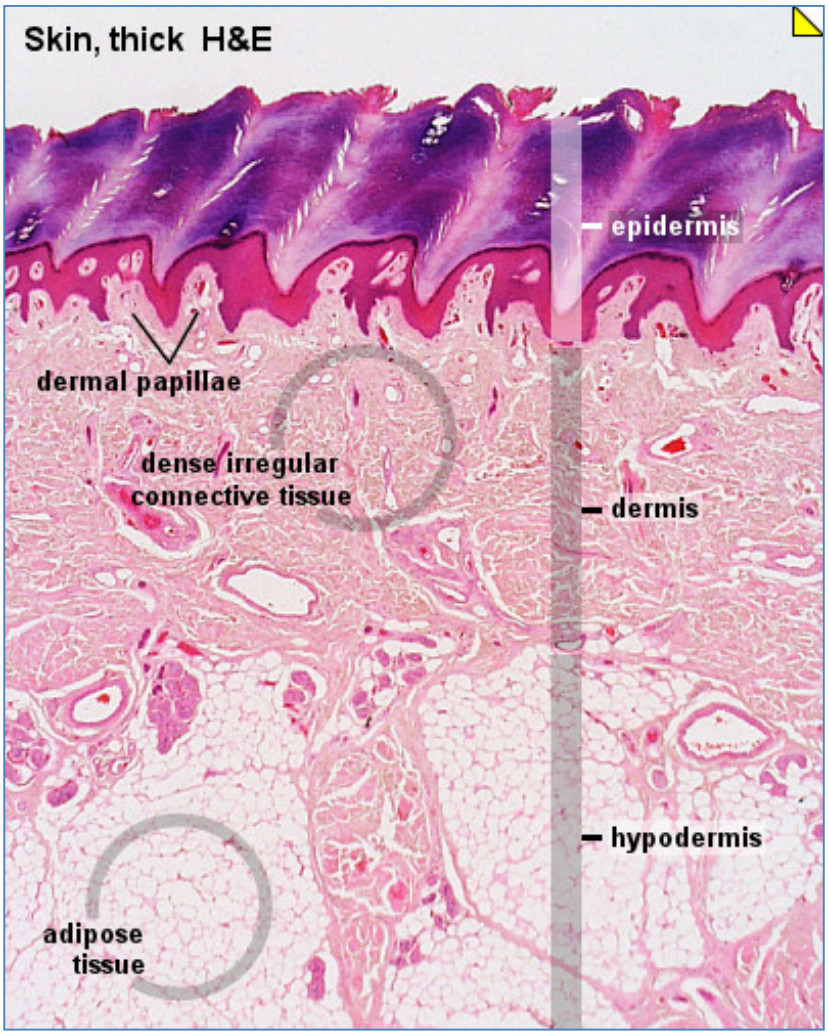


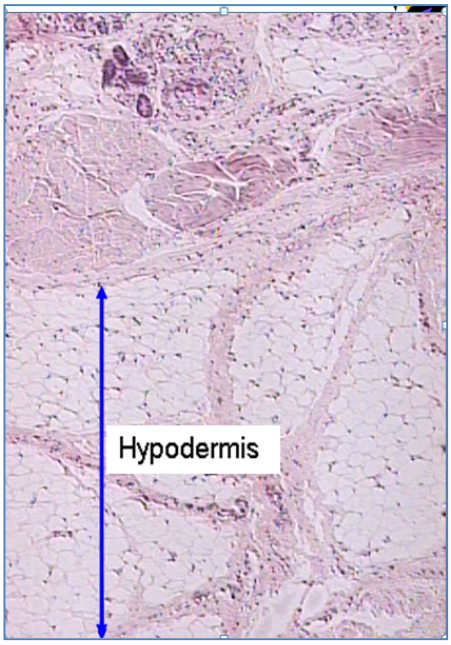
  - **Glycosaminoglycans (GAGs) =** Absorb water – *Provide Viscosity*.
    - Hyaluronan backbone with glycoprotein branches.



LAYER 3: THE HYPODERMIS:

- **Structure of The Hypodermis:**
  - **Composition:**
    - Mostly Fat (Adipose Cells)-(Thickest in Abdomen)
    - Blood Vessels
    - Nerves
  - **Functions:**
    - Insulates the body
    - Stores Energy

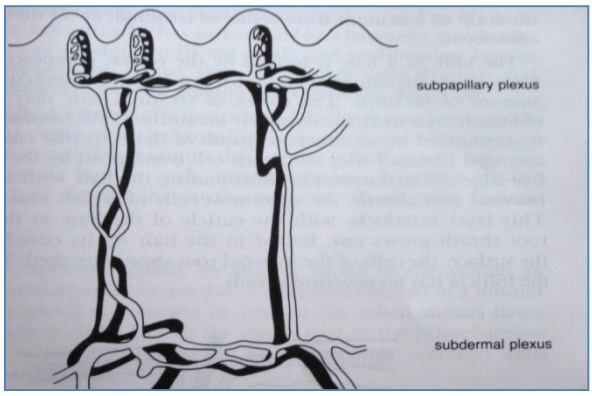


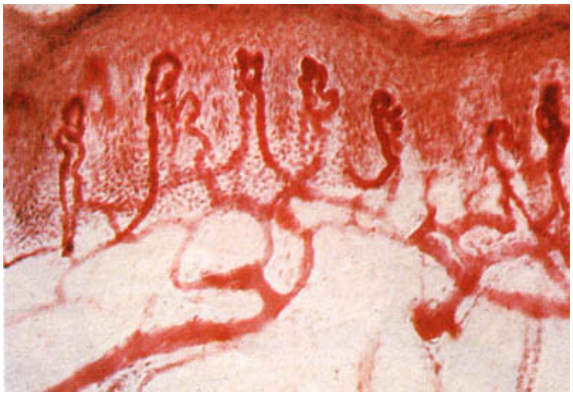


AUXILLARY COMPONENTS OF SKIN:

- **Vasculature:**
  - Abundant network of dermal vessels
  - **Functions:**
    - **Nutrition of Skin Tissue:**
    - **Regulation of Body Temperature:**
      - Conducts heat from Interior to Exterior → Heat loss to Environment
      - Vasodilation/constriction Important in Thermoregulation
    - **Blood Reservoir:**
      - Under conditions of circulatory stress (Eg. Exercise/Haemorrhage/Shock), Sympathetic Stimulation → Vasoconstriction → ↑Circulating Blood Volume.

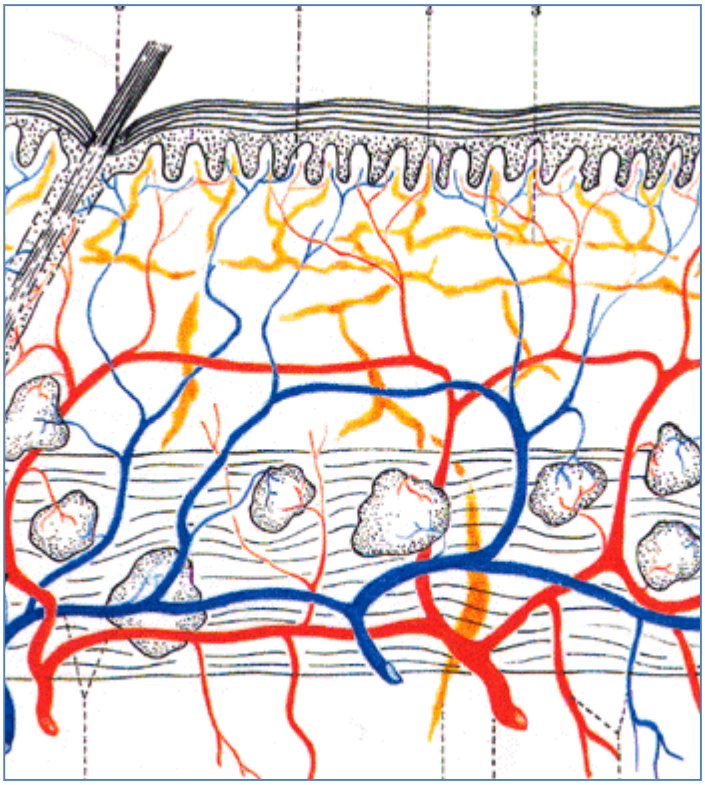
  - **Cutaneous Circulatory Apparatus – 2 Types of Vessels:**
    - **1. Nutritive Vessels:**
      - Arteries
      - Capillaries
      - Veins
      - (Organised into a horizontal ***Subdermal Plexus*****):**
        - Ascending arterioles extend towards the epidermis
          - ***Subpapillary Plexus*** (In the Papilary Dermis)
            - **Capillaries** loop into the dermal papilla





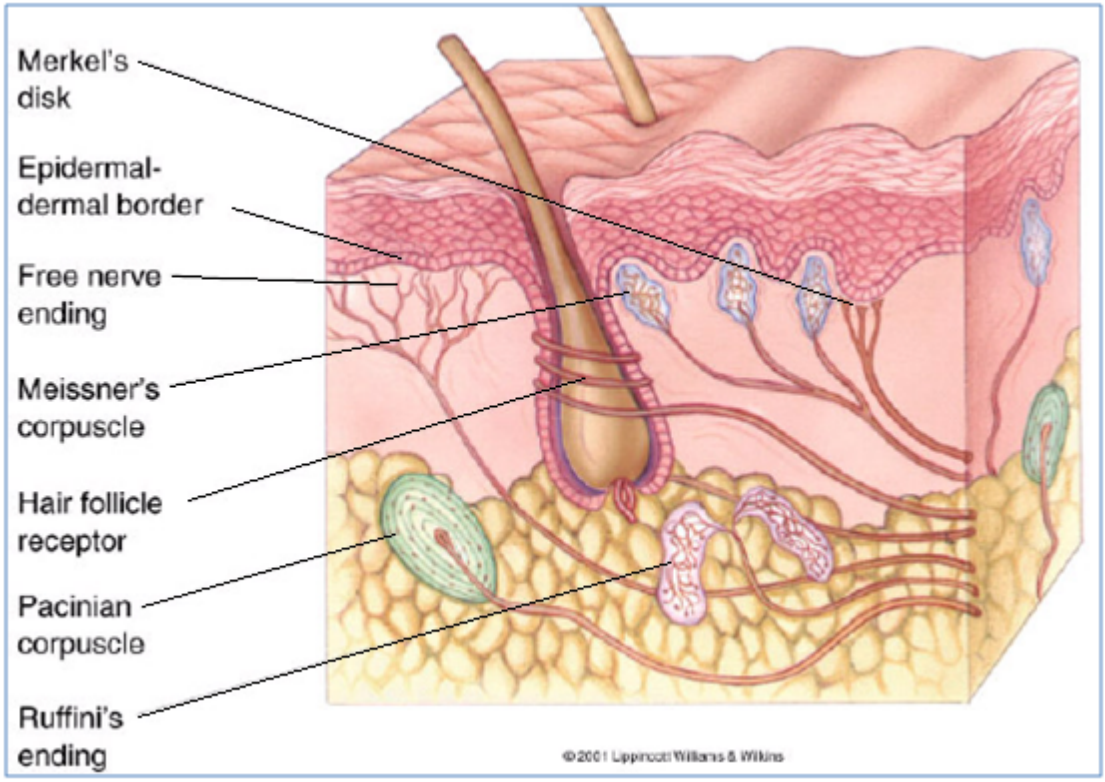
    - **2. Vascular Structures for Heat Regulation:**
      - **Extensive Subcutaneous Venous Plexi:**
        - Can hold Large quantities of blood
      - **Arteriovenous Anastomoses:**
        - Only present in areas of Maximal Cooling (Hands, Feet, Lips, Nose & Ears)
      - **NB: *BOTH Innervated by Sympathetic Adrenergic Vasoconstrictor Fibres:***
        - **Sympathetic NS** → Vasoconstriction → Minimal Heat Loss.
        - (No Stimulation → Vasodilation → Maximal Heat Loss)

- **Lymphatic Drainage:**
  - Each dermal papilla has a single lymphatic capillary → subpapillary plexus → regional lymph nodes
  - Carry langerhans cells, interstitial fluid, debris, lymphocytes→Lymph Nodes →Blood

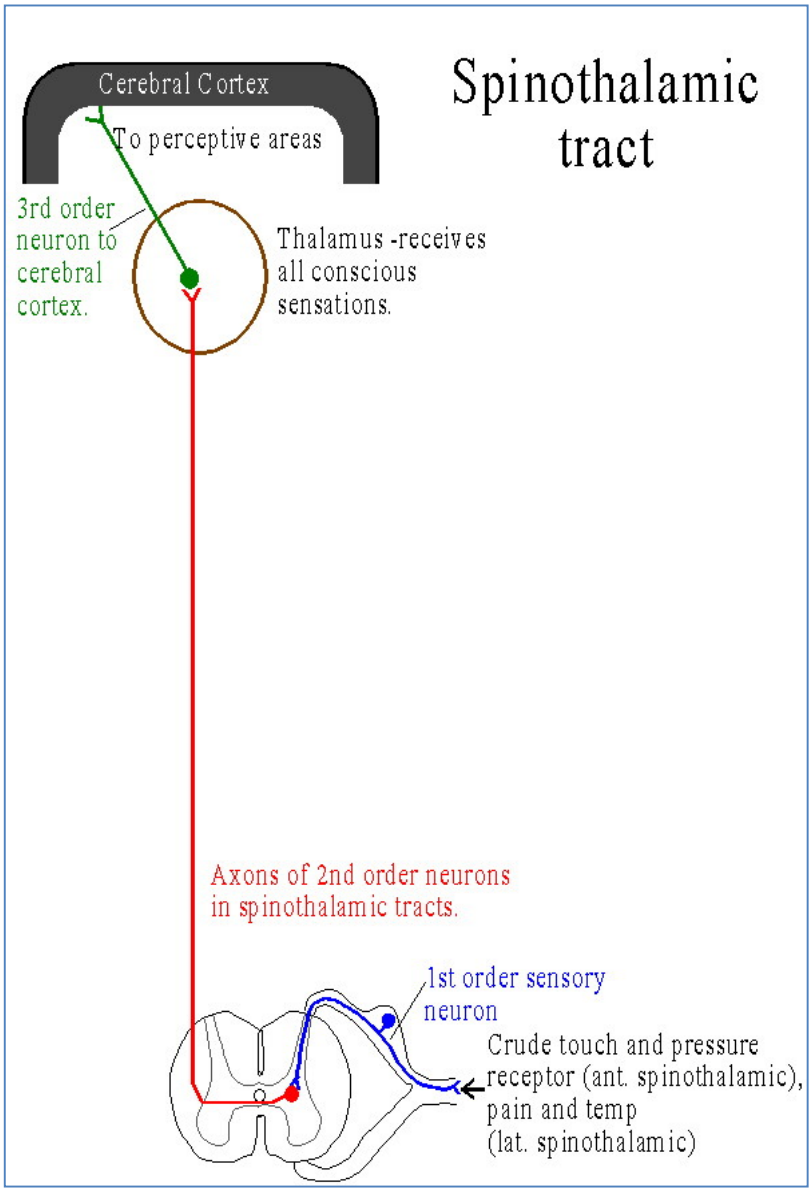


(Yellow is Lympatic Vessels)

- **Cutaneous Sensory System:**
  - **Sensory Apparatus of the Skin:**
    - Most afferent nerve fibres terminate the Face & Extremities. (Few on back)
  - **Sensory receptors:**
    - Receptors of touch, pain, temperature, itch and mechanical stimuli
    - Nerve fibers enter the dermis from the underlying adipose tissue
    - **1. Unencapsulated:**
      - **Free Nerve Endings:**
        - In Superficial Dermis & Epidermis.
        - Receptors for Pain, Touch, Pressure, & Temperature.
        - **Incl. Nociceptors:**
          - **A-Delta Fibres:**
            - Fast, Sharp, Pricking Pain.
            - Thick, Myelinated → Fast Conduction.
          - **C-Fibres:**
            - Slow, Dull, Aching, Burning Pain.
            - Thin, Unmyelinated → Slow Conduction
      - **Merkel Cell Discs (Merkel Touch Spots):**
        - Found in the Stratum Basale of the Epidermis.
        - Receptors for Light Touch
      - **Hair-Follicle Receptors:**
        - In & Surrounding Hair Follicles
        - Mechanoreceptors.
    - **2. Encapsulated (Lamellated Capsule):**
      - **Meissner Corpuscles:**
        - In Dermal Papillae
        - Fine Touch receptors
      - **Pacinian Corpuscle:**
        - In Deep Dermis & Hypodermis
        - Deep Pressure Sensors
      - **Ruffini Endings:**
        - In Deep Dermis & Hypodermis
        - Directly Associated with Collagen Fibrils → Stretch Receptors



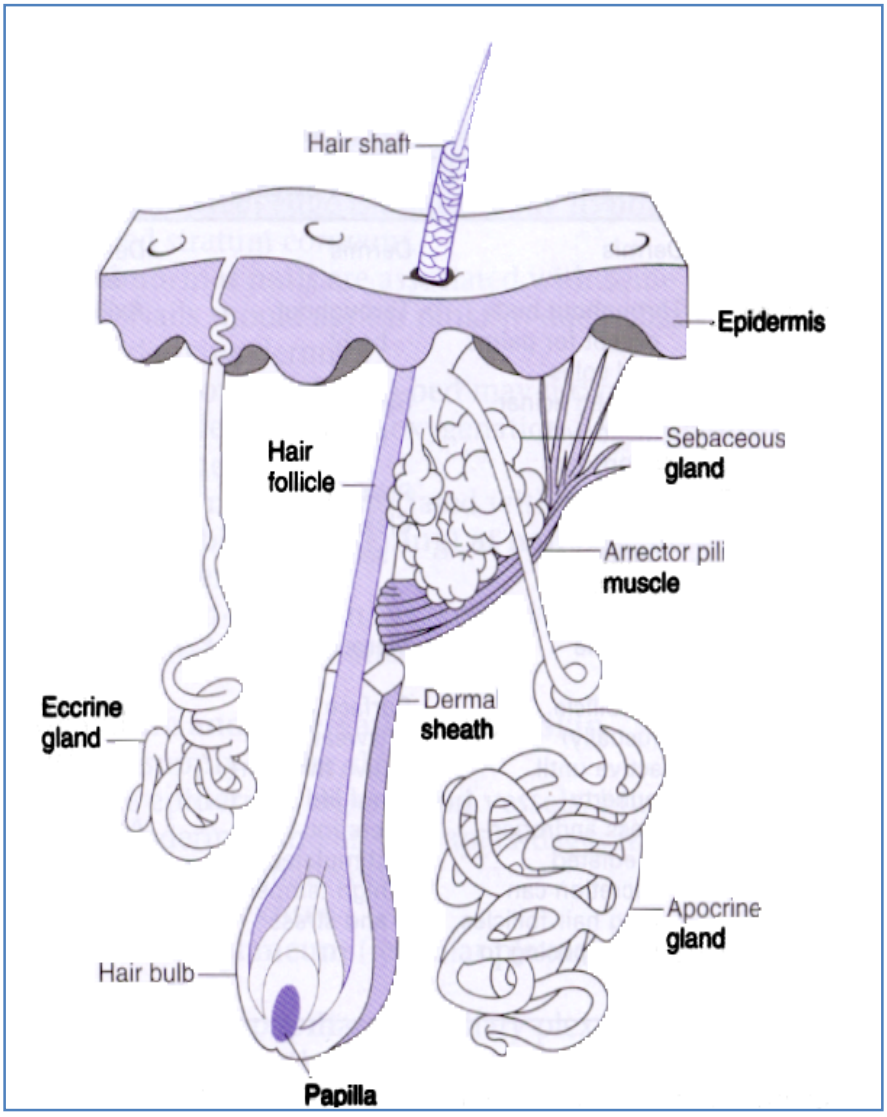
- **Physiology of Sensory Receptors:**
  - **Concept of Adaption:**
    - ***Adaption:*** Under a maintained stimulus of constant strength, the frequency of action potentials declines over time.
    - **Slowly-Adapting Receptors (Eg. Nociceptors):**
      - Continue to Transmit Impulses to the brain as long as the Stimulus is Applied.
    - **Rapidly-Adapting Receptors (Eg. Pacinian Corpuscles):**
      - Receptors Rapidly Adapt & are stimulated only when the Stimulus Strength has Changed.
- **Connection to the CNS:**
  - **First Order Neuron:**
    - Sensory Neuron Nucleus is in the **Dorsal Root Ganglion**.
    - Its axon extends from periphery to Dorsal Horn of the Dorsal Root Gangion.
  - **Second Order Neuron:**
    - Neuronal Nucleus is in the Substantia Gelatinosa
    - Its axon Decussates, Then it Ascends in the **Spinothalamic Tracts** → Thalamus
  - **Third Order Neuron (Thalamus):**
    - Neuronal Nucleus is in the Thalamus
    - Its axon passes through the Internal Capsule (behind Pyramidal Fibres) → Sensory Cortex



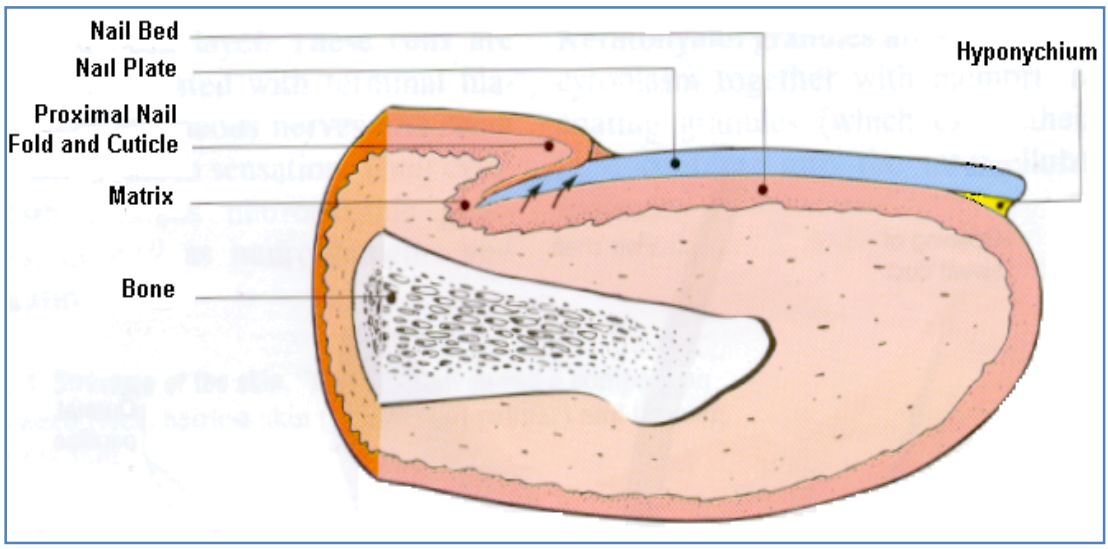
- **Glands:**
  - **Sebaceous (Sebum) Glands:**
    - **Associated with hair follicles (The Pilosebaceous Unit)**
      - (:. Not found on Palms/Soles)(Most found on Face & Scalp)
    - **Holocrine Secretion:**
      - (Ie. Secretion via complete destruction of cells)
      - **Produce oily sebum:**
        - Triglycerides
        - Fatty Acids
        - Wax Esters
        - Cholesterol
        - (Also Antibacterial/Antifungal Action)
    - **Stimulated by Androgens:**
      - Stimulated by Androgens. (Inhibited by Estrogens)
      - Very Active at puberty

  - **Eccrine (Sweat) Glands:**
    - On most of the body (Scarce on the back)
    - **Simple, Coiled Tubular Glands:**
      - Secretory Coil (deep in Dermis) – Secrete the Water & Electrolytes
      - Sweat Duct – Reabsorb Na^+ Ions from the sweat.
    - **Clear watery secretion**
      - Person can perspire several liters per hour
      - **Process:**
        - 1. Secretion of Electrolyte-Rich Fluid
        - 2. Reabsorption of excess Na^+ by the Duct.
    - **Stimulated by High Temperature and Stress**
      - Emotional Sweating doesn't occur during sleep
      - Thermal sweating does occur during sleep
      - Innervated by Sympathetic Nerves

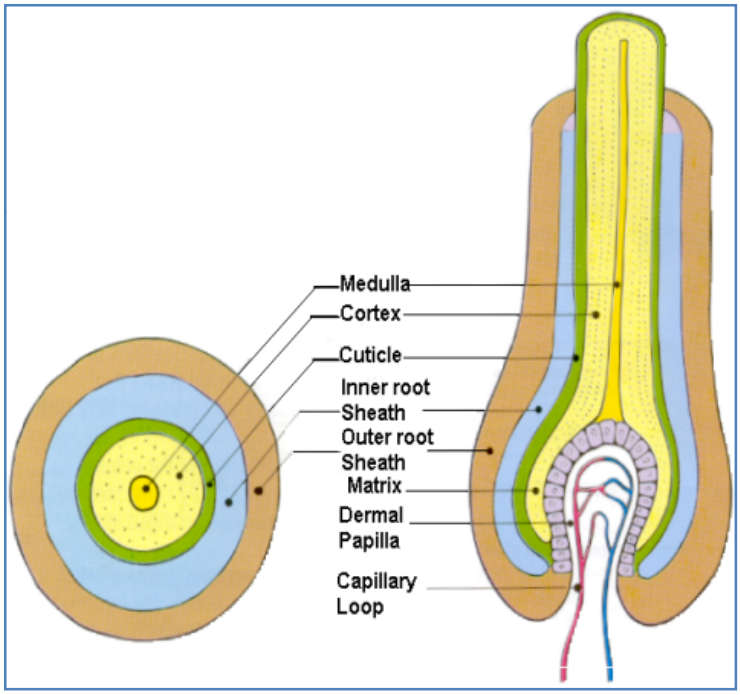
  - **Apocrine (Pheromone) Glands:**
    - Associated with hair follicles
    - **Large complex gland:**
      - Located in Dermis
      - Duct Opens into Hair Follicle.
    - **Viscous, Milky Secretion – (protein and cellular debris):**
      - **Produces pheromones**
        - Bacterial action is required for odor production
        - Thought to have or had a role as a sexual attractant in humans
        - Respond to emotive stimuli
    - **Stimulated by Androgens:**
      - Most Active at puberty


Hair shaft
Epidermis
Hair follicle
Sebaceous gland
Arrector pili muscle
Eccrine gland
Dermal sheath
Apocrine gland
Hair bulb
Papilla

- **Nails ("Ungals"):**
  - **Composition:**
    - Plate of hardened and densely packed **keratin (Protein)**
  - **Functions:**
    - Protect distal phalanges of fingers and toes
    - Aid in picking up objects
    - Efficient natural weapon
  - **Structural Landmarks:**
    - **Nail Plate:**
      - Fully keratinized structure
      - Firmly attached to the nail bed
    - **Nail Bed:** Skin underneath the nail
    - **Lunula:** Proximal whitish half moon-shaped area on Nail Plate
    - **Cuticle:** The dorsal part of the proximal nail fold
    - **Nail Matrix:** Nail growth occurs From Here - by proliferation and differentiation of the nail matrix
    - **Paranychium:** The Skin around the Nail
    - **Hyponychium:** The area where the nail plate detaches from the digit
    - **Proxymal Nail Fold:** The fold at the Proximal Edge of the nail (Covers ≈1/4 of the nail)
    - **Lateral Nail Folds:** The folds of skin at the Lateral Edges of the Nail

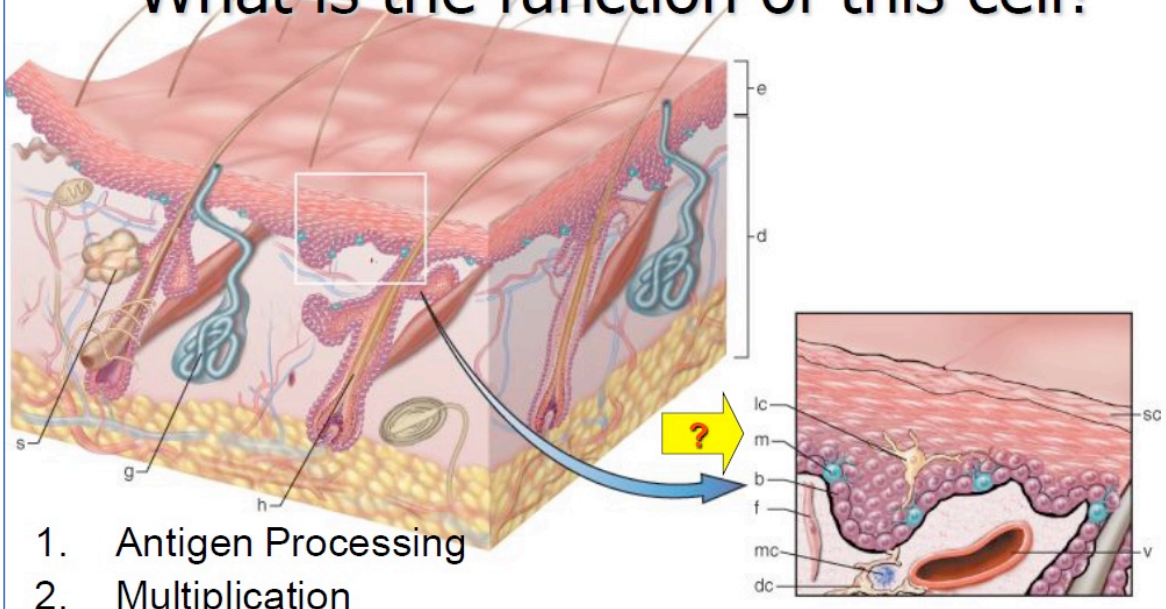


- **Hair/The *'Pilosebaceous Unit'*:**
  - **The Pilosebaceous Unit:**
    - Hair Shaft
    - + Sebaceous/apocrine ducts empty into hair follicles
  - **Arrector Pili Muscle**:
    - Smooth Muscle
    - Supplied by Adrenergic Nerves
    - → Erection of the Hair during Cold/Emotional Stress (Goose-Bumps)
  - **2 Types of Hair:**
    - **Vellus:**
      - Fine, Short & Almost invisible
      - (All over the body)
    - **Terminal:**
      - Thick, coloured & Visible
      - (Scalp, beard, axilla, genital area)
  - **Hair Follicles Respond to Androgen:**
    - Pre-pubertal children don't have *Terminal Hair* in Axilla/Genital Area/Facial Hair.
    - During Puberty, hair grows in these areas
    - With age, androgen stimulation decreases → *Terminal Hair* on scalp reverts from *Terminal* to *Vellus* hair. (Hair follicles aren't lost, but change to *Vellus* hair)
  - **Hair follicles undergo cycles of Growth, Resting and Shedding:**
    - **Anagen (growing):**
      - Lasts 3 Years
      - Keratinocytes in the follicular bulb proliferate to form the hair shaft
    - **Catagen (Resting):**
      - Lasts 2 Weeks
      - The keratinocytes and melanocytes undergo programmed cell death
    - **Telogen (Shedding):**
      - Last 3 Months
      - Hair but does not grow.
      - May Remain Anchored, or Be Shed.
      - Following telogen a new growth cycle will begin (ie anagen)
  - **Hair Pigmentation:**
    - Colour of hair is determined by the melanocytes – actively pigmented only in Anagen.
    - Absence of pigment →white hair
    - Diminished pigment →grey hair

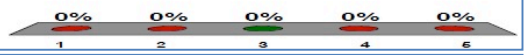


SAMPLE SKIN STRUCTURE QUESTIONS:

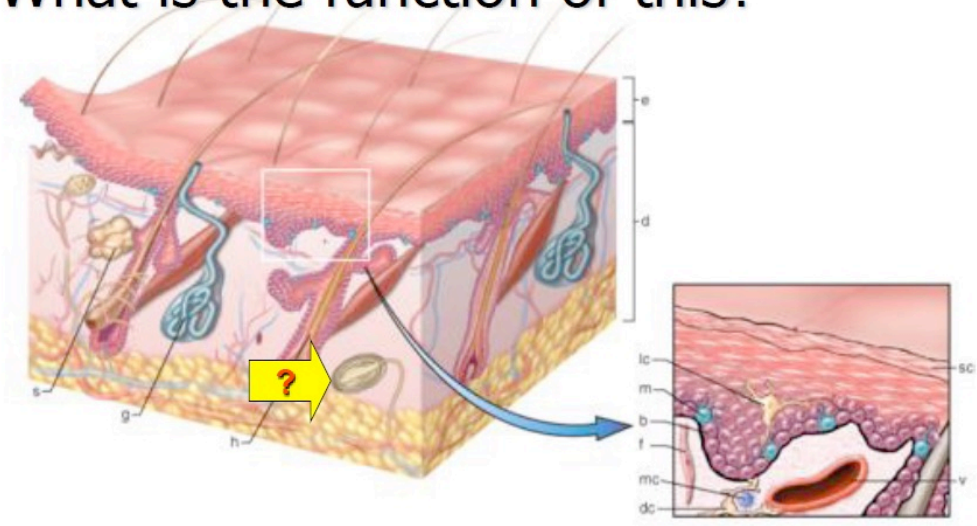
What is the function of this cell?



1. Antigen Processing
2. Multiplication
3. Melanin production (Melanocytes)
4. Melanin storage
5. Protection

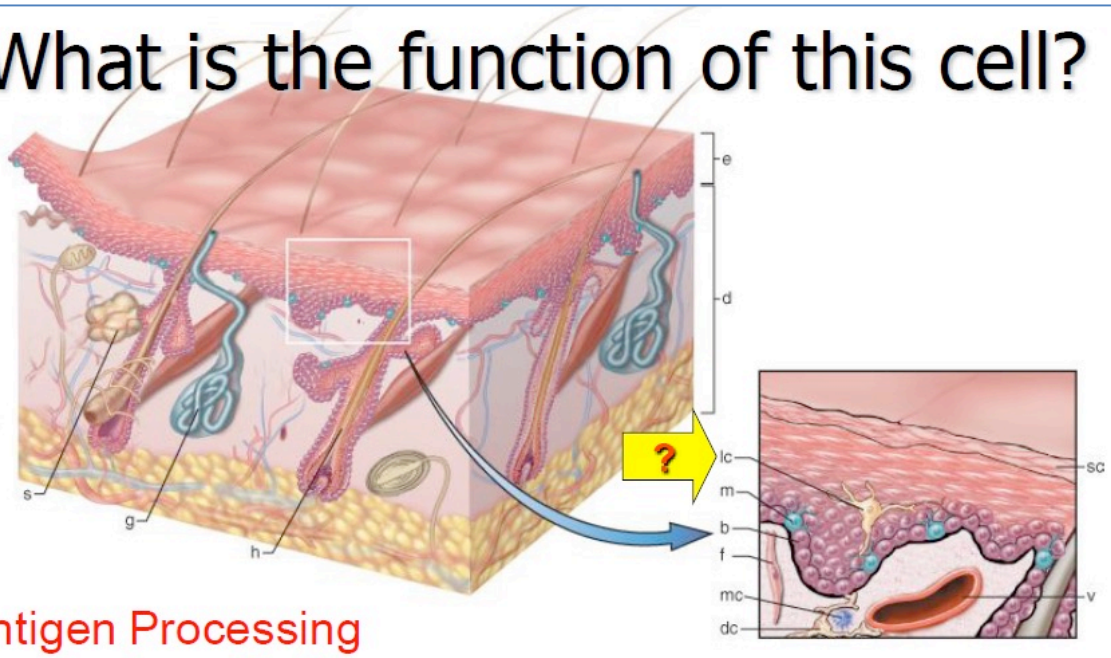


What is the function of this?

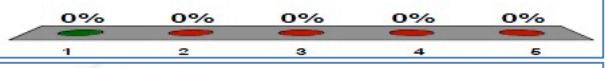


1. Fine touch sensation
2. Pressure sensation (Pacinian Corpuscle)
3. Pain sensation
4. Sweat production
5. Temperature control

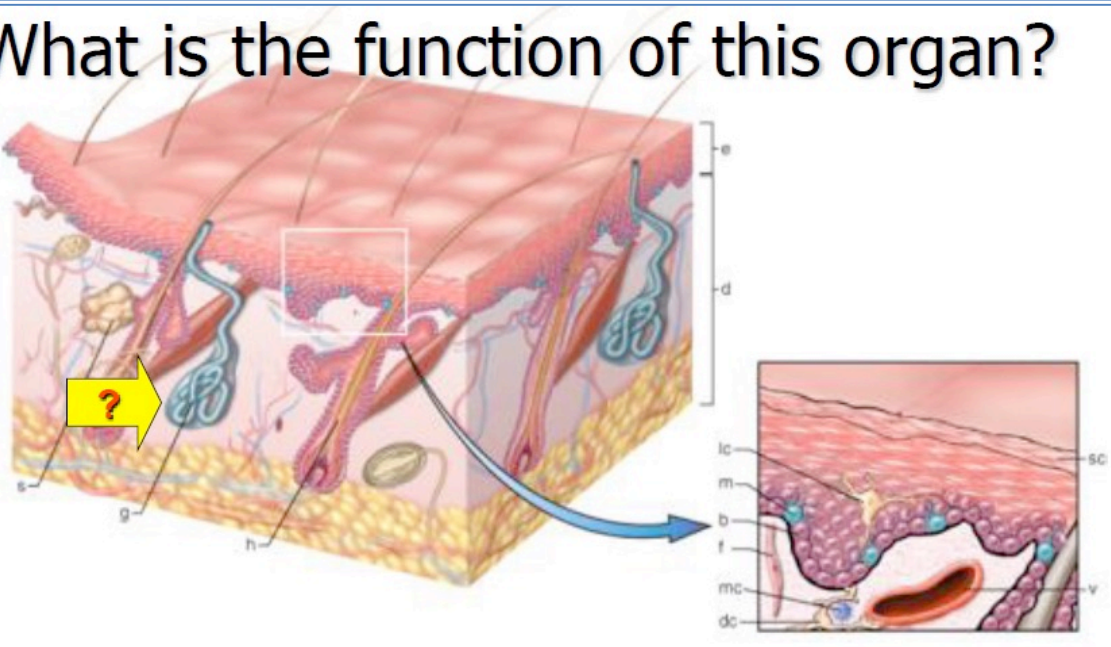
What is the function of this cell?



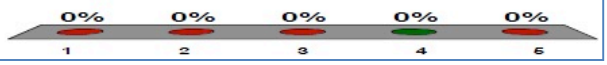
1. Antigen Processing (Langerhans Cell)
2. Multiplication
3. Melanin production
4. Melanin storage
5. Osmotic barrier



What is the function of this organ?



1. Fine touch sensation
2. Pressure sensation
3. Pain sensation
4. Sweat production (Eccrine Gland)
5. Temperature control



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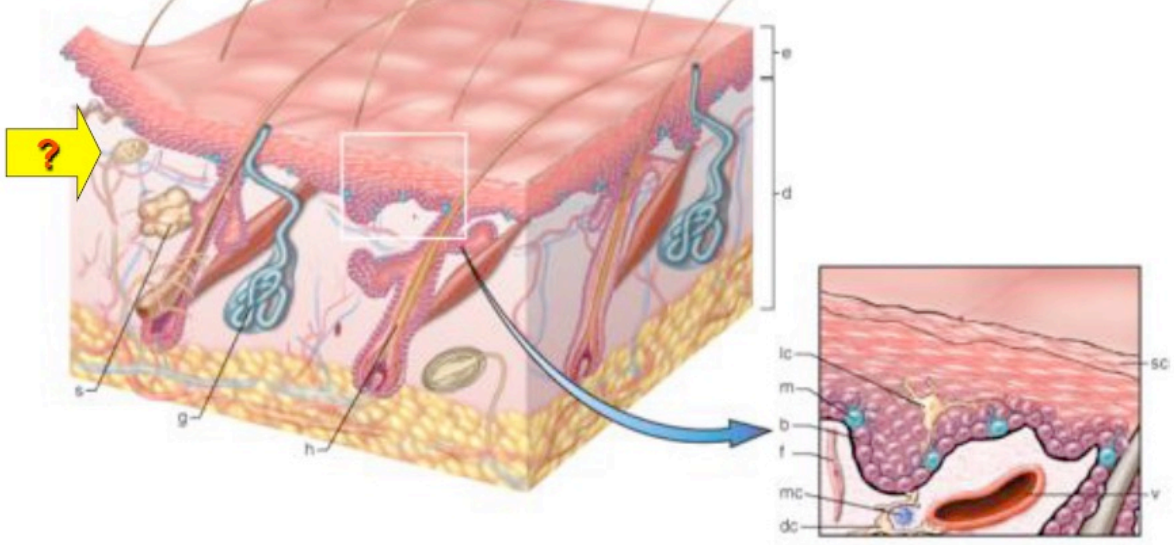
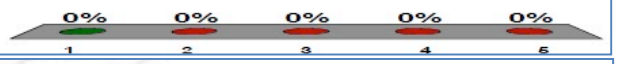
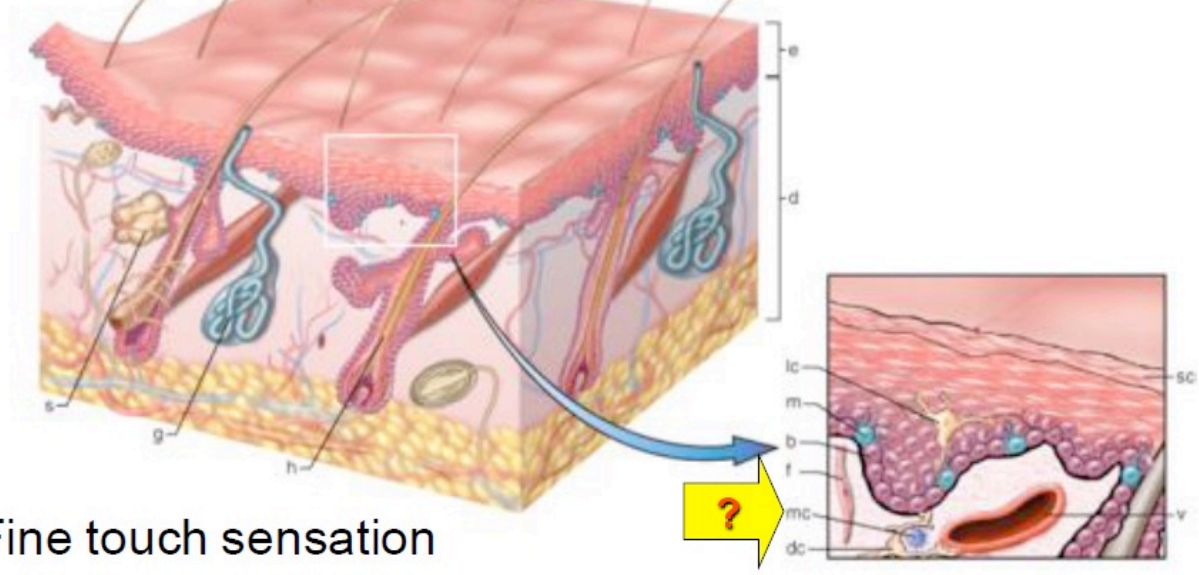


1. Fine touch sensation (Meissner's Corpuscle)
2. Pressure sensation
3. Lubrication
4. Sweat production
5. Temperature control



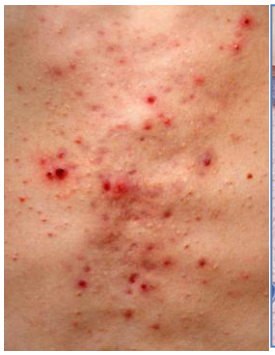
What is the function of this?

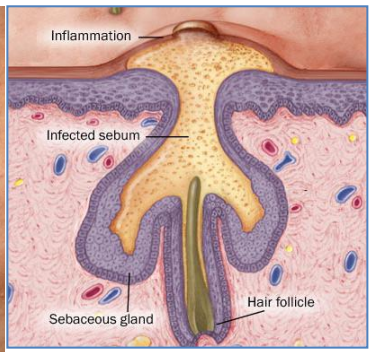


1. Fine touch sensation
2. Pressure sensation
3. Lubrication
4. Immunity & defence (Macrophage)
5. Temperature control

DERMATOLOGY Pathology:
Acne

- **ACNE:**
  - **What is it?:**
    - Common Skin Condition in Teenagers (Although Can occur at any age)
    - Most Severe in Males
  - **Aetiology & Pathogenesis:**
    - Genetic Basis (Familial)
      - Chronic inflammation of the sebaceous glands
    - **Abnormalities of the *Pilosebaceous Unit*:**
      - Blockage of the Sebaceous Duct (By ↑Keratin @ Duct Opening)
      - Increased Sebum Production (Often under influence of ↑Androgen)
      - → Bacterial Infection of the Pilosebaceous Unit (*Proprionibacterium Acnes*)
      - → Rupture of the Sebaceous Duct
      - → Inflammatory Effects.
    - **NB: Combined Oral Contraceptives** (Ie. With Progesterone) can contribute to Acne.
    - (IF Severe, Damage to Dermis Occurs → Scarring. NB: Scarring is a FAILURE of the Doctor to Adequately treat Acne)
  - **Presentation:**
    - Wide Variation in Severity
    - Most often on Face, Upper Chest & Back.
    - ****Presence of Comedones** – Both Open (Blackheads) & Closed (Whiteheads)
      - (NB: If no comedones are present, consider Differential Diagnoses)
    - **Inflammatory Papules & Pustules**
    - Severe Cases →
      - **Cysts**
      - Scarring (Hypertrophic or Atrophic)
  - **Diagnosis:**
    - **The 'Acne Triad':**
      - Papules + Pustules + ***Comedones*** = ACNE
    - **Possible Differential Diagnoses:**
      - ***Rosacea:**
  - **Treatment:**
    - Topical Anti-inflammatories & Antiseptics.
    - Antiseptics (Eg. Benzoyl Peroxide)
    - Antibiotics (If Severe)(NB: Tetracycline also has an Anti-Inflammatory Effect – Good)
    - Keratolytics (To ↓the overproduction of keratin – Eg. Salicylic acid)
    - Retinoids (Derived from Vit.A) (Isotretinoin/Roaccutane) (In Severe Nodulo-Cystic Acne)
    - ≈1yr after symptoms disappear
  - **Prognosis:**
    - Usually Good. (Most cases are Self-Limiting)
    - Treatments are safe & well-tolerated
    - NO Pt. should have to Suffer!





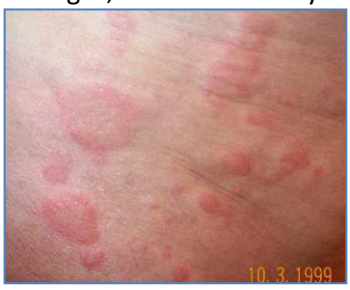
DERMATOLOGY Pathology:
Allergic Skin Conditions

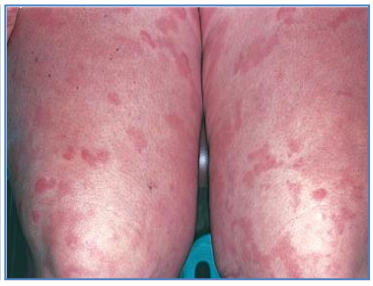
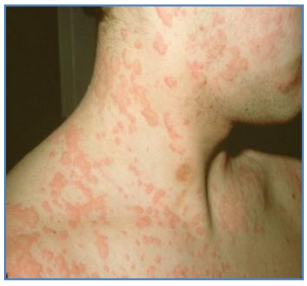
Exogenous (Atopic) Eczema:

- **Overview:**
  - A common Paediatric condition, however it may persist into adult life.
- **Aetiology:**
  - Family history – (in 70% of cases)
  - Genetics – (50% of pts have a deficiency of the Epidermal Protein "Filaggrin".)
  - Hypersensitivity – (Associated with other Atopies (Hay Fever [Allergic Rhinitis], and Asthma))
- **Presentation:**
  - Severe Itching
  - Patchy, Erythematous, Poorly Defined Rash.
    - Usually in the Popliteal/Cubital Fossae & Face
    - Can be Generalised
  - Dry Skin
  - Excoriation (loss of the surface of the skin from scratching) due to itching and scratching.
  - Lichenification (thickening of the skin with accentuated skin lines)
  - Crusting (scabbing) and weeping (loss of fluid through the surface of the skin) due to bacterial infection.
- **Treatment:**
  - Modification of lifestyle to avoid exacerbating factors.
  - Use of moisturisers and bath additives.
  - Avoid Soaps
  - Use of mild topical corticosteroids.

Urticaria (Hives):

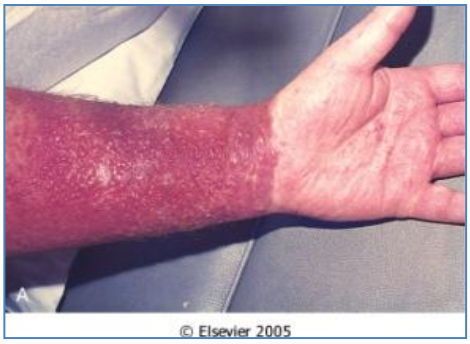
- **Aetiology:**
  - Type I hypersensitivity – Allergy (Food/drug/plant/etc)
- **Pathogenesis:**
  - Antigen is Re-Exposed to a sensitized Mast-Cell/Basophil → IgE-Bound Mast Cell *Degranulates*:
    - → Releasing Inflammatory Mediators (Histamine) of Type-1-Hypersensitivity Reactions.
    - →Perivascular inflammatory infiltrate: lymphocytes, neutrophils or eosinophils.
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - Erythematous Papules & Plaques (Reddish, raised areas).
- **Clinical Significance (List 3x Clinical Features):**
  - Usually on trunk and extremities.
  - Individual lesions are transient, usually resolve in 24 hr, but entire episode may last for days.
  - All ages, more in 20 – 40y.



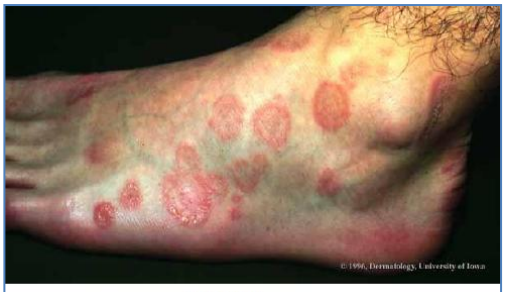
Acute Eczema:

- **Types of Acute Eczema:**
  - Contact dermatitis (Due to prolonged exposure to allergen)
  - Atopic dermatitis
- **Aetiology:**
  - Type IV Hypersensitivity (T-Cell Mediated) to Allergen.
  - Prolonged Contact with Allergen - Urine, Soaps, Antiseptics, Deodorants, Creams, Foreign Body, etc.
- **Pathogenesis:**
  - **Initial/Acute/Re-Exposure:**
    - Type IV Hypersensitivity (T-Cell Mediated) to Prolonged Contact with Allergen → Epidermal Oedema + Small Blisters → **"Wet Eczema"**
  - **Chronic Exposure:**
    - →Hyperplasia, hyperkeratosis (lichenification) – **"Dry Eczema"**.
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - **Gross:**
    - Erythema
    - Small Blisters – (Weeping & Crusting Blisters, Papules and Plaques.)
    - Hyperkeratosis (If Chronic)
- **Clinical Features:**
  - Most Common in Children
  - Often has a regular shape (Eg. Square from bandaid)
- **Treatment:**
  - Remove Allergen
  - Treated with Corticosteroids (Symptomatic)
  - Antihistamies (For Itch)



Erythema Multiforme:

- **Aetiology:**
  - Hypersensitivity
- **Pathogenesis:**
  - Hypersensitivity Response to:
    - Infections (Herpes simplex, *Mycoplasma*)
    - Drugs (Sulfonamides, penicillin, barbiturates)
    - Malignancy (Carcinoma, lymphoma)
    - Auto-Immune (Eg. Lupus, SS, Dermatomyositis)
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - **Gross:**
    - Variable Lesions: Papules/Plaques/Nodules/Blisters/Ulcers
    - Characteristic "*Targetoid*" Lesions
      - Central Grey Necrosis
      - Erythematous raised border.
- **Clinical Significance (List 3x Clinical Features):**
-



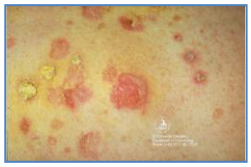
Target Lesions

DERMATOLOGY Pathology:
Blistering Diseases

BLISTERING DISEASES:

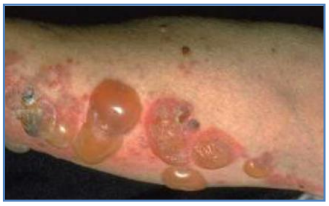
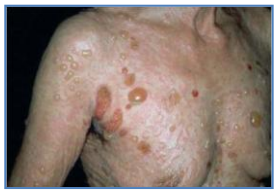
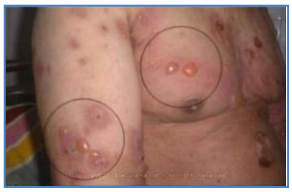
Pemphigus (Pemphigus Vulgaris):

- **Aetiology:**
  - Autoimmune
- **Pathogenesis:**
  - Antibodies against Desmosomes → Destroys Desmosomes → Acantholysis (Intraepidermal Blisters)
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - Thin, Flaccid Blisters (Easily Popped)
  - Look similar to burns
  - Many have popped → Crusting
- **Clinical Significance (List 3x Clinical Features):**



Bullous Pemphigoid:

- **Aetiology:**
  - Autoimmune
- **Pathogenesis:**
  - Antibody against Basal Layer → Destroys Sub-Epidermal Anchoring Proteins.
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - Large, Tense, ***Subepidermal*** Bullae
  - Sometimes Haemorrhagic Blisters
- **Clinical Significance (List 3x Clinical Features):**

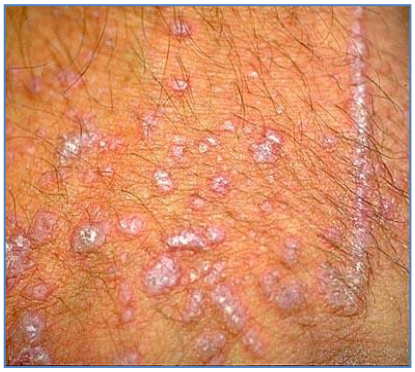
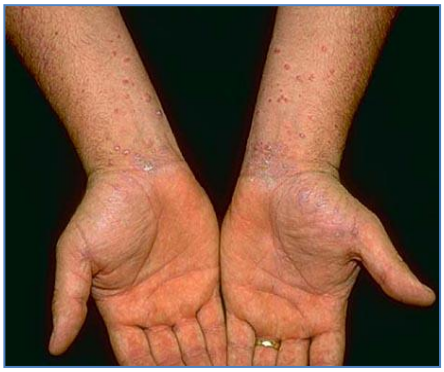
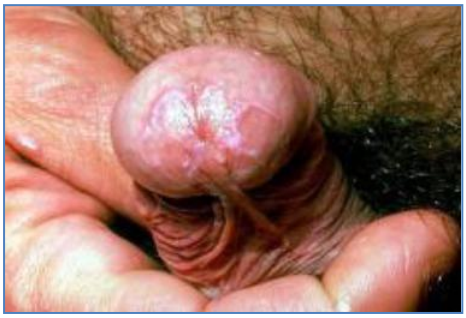
Dermatitis Herpetiformis.

- **Aetiology:**
  - Associated with Coeliac Disease (Gluten Sensitivity)
- **Pathogenesis:**
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - VERY Itchy, Small, Erythematous Papules
  - Vesicles
  - Occasional Bullae
- **Clinical Significance (List 3x Clinical Features):**
  - Associated with Coeliac Disease

DERMATOLOGY Pathology:
Lichen Planus

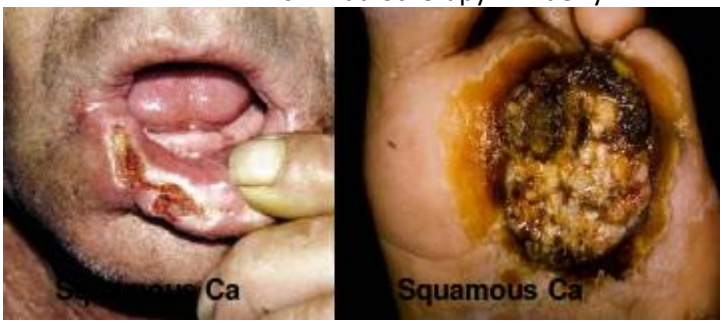
Lichen Planus:

- **Aetiology:**
  - Unknown (not pathogenic or infectious)
- **Pathogenesis:**
  - Similar to Erythema Multiforme (Hypersensitivity), but more chronic.
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - **Gross:**
    - Purple, polygonal, planar, Papules & Plaques.
- **Clinical Significance (List 3x Clinical Features):**
  - Itchy, Purple Rash
  - on Skin (commonly wrists & ankles), Mucosa, Genitals, Oral.
  - Self-limiting (1-2 yrs)

DERMATOLOGY Pathology:
Malignant Ulcers

- **Malignant Ulcers:**
  - **SCC – Squamous Cell Carcinoma:**
    - **Aetiology:**
      - Chronically Sun-Damaged Skin → Skin Cancer
    - **Pathogenesis:**
      - Squamous Cell Mutation → Dysplasia → Neoplasia
    - **Morphology:**
      - **Macro:**
        - Typically Tender Papules/Nodules with *ROUGH, ADHERENT SCALE*.
        - Often Ulcerate Centrally
        - Often Present as Ulcers on Lips/Mucosa
      - **Micro:**
        - ↑Keratin production by Pleomorphic Squamous Cells
          - ***Presence of Keratin "Pearls"
          - Pink on microscopy due to lots of keratin
        - Whorls & Nests (Clusters) of malignant cells
    - **Clinical Features:**
      - **Treatment:**
        - Wide excision
        - Radiotherapy in Elderly



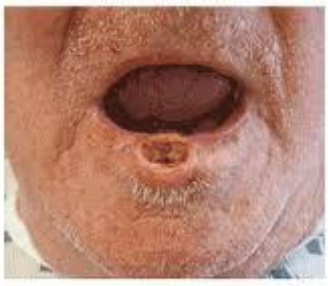
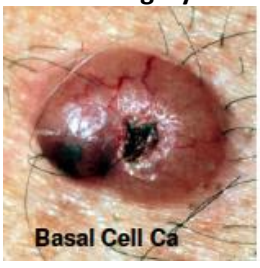
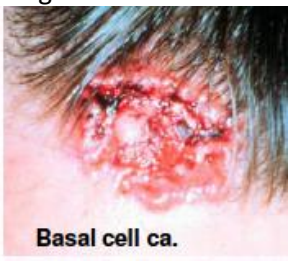


FIGURE 1.

  - **BCC – Basal Cell Carcinoma (AKA "Rodent Ulcer"):**
    - **Aetiology:**
      - Chronically Sun-Damaged Skin → Skin Cancer
    - **Pathogenesis:**
      - Basal Cell Mutation → Dysplasia → Neoplasia
    - **Morphology:**
      - **Macro:**
        - Shiny, Nodular with Overlying telangiectasia.
        - Can be Ulcerative (appear as ulcers with pearly or indurated edge)
      - **Micro:**
        - Clusters of blue Basal Cells
        - Basal cells are pleomorphic
        - Burrows Deep into the Dermis
        - "Palisading" appearance around the edge of the clusters of Basal cells (like the walls of a palace)
    - **Clinical Features:**
      - **Excisional surgery** : Still the gold standard treatment of BCC's.





DERMATOLOGY Pathology:
Procedural Dermatology – GP

Local Anaesthetic:

- **Mechanism of Action:**
  - Use-Dependent blockade of Voltage-Gated Sodium Channels on Nerves
    - → Prevents Action Potential Conduction along Sensory Nerves.
  - NB: Very Quick Onset of Action
  - NB: Also causes vasodilation → Short Duration of Action (If without adrenaline)
- **Lignocaine – Most Common:**
  - Rapid Onset (1-5mins)
  - Medium (30-120mins)
  - **Max Dose:** 4mg/kg
    - ≈ 50mL @ 1% Lignocaine
  - (NB: 1% Lignocaine = 1g/1000mL)
- **+ Adrenaline:**
  - → Improves Haemostasis
    - → ↓Bleeding
  - → ↓Systemic Absorption
    - → ↓Risk of systemic toxicity
    - → Prolonged Effect
  - **Max Dose:** 7mg/kg (Higher Dose than without adrenaline)
  - **NB: DO NOT USE IN DIGITS OR PENIS (Or anywhere else with "End Arteries")**
  - **NB: DO NOT USE in LONG QT-Syndrome.**
- **Naropin (Ropivicaine):**
  - Longer onset of action
  - Longer acting than lignocaine
  - **Max Dose:** 2-3mg/kg
- **Topical Anaesthetic:**
  - **Xylocaine Gel -** Useful for Mucosal Surfaces (eg. Oral Mucosa)
  - **ELMA Cream –** Useful for topical anaesthesia of skin.
- **Nerve Blocks:**
  - **Digital Block (Ring Block)**
    - (Most Common)
    - Blocks Digital Nerves
    - Use 2% Lignocaine (Because you want to inject as little as possible)
  - **Wrist Blocks:**
    - Blocks Radial, Median, & Ulnar Nerves
    - → Totally anaesthetizes the hand.

Curettage & Electrocautery:

- **Indications:**
  - Warts
  - Keratoses
  - Molluscum contagiosum
  - Curettes can also be used for curette biopsy.
- **Wound Healing**
  - The curette wound can take some time to heal.
  - If it is just a light curette such as tiny seborrhoeic keratoses with no cautery they can heal in a few days
  - A full curettage and electrodessication on the trunk might take 4 to 6 weeks to heal

Cryotherapy:

- Cryotherapy is a very useful form of treatment for a number of benign skin lesions and few pre-malignant and malignant skin lesions.
- Liquid Nitrogen = The gold standard
- **Indications:**
    - **Benign lesions:**
        - Molluscum contagiosum
        - Seborrhoeic keratosis
        - Skin tags
        - Warts
    - **Premalignant lesions:**
        - Actinic keratoses
        - Actinic cheilitis
    - **Malignant lesions:**
        - Superficial BCC
- **Effects of Cryotherapy – 3 Main Groups:**
    - **1. Those responses we expect to happen:**
        - Pain on treatment and for a period afterwards
        - Oedema and swelling of the treated site and the surrounding tissue (e.g. periorbital swelling after treatment of lesions on the forehead)
        - Vesicle and bulla formation
        - Exudation weeping and crust formation
    - **2. Temporary Adverse Outcomes:**
        - Hypopigmentation
        - Hyperpigmentation
        - Secondary Infection
    - **3. Permanent Adverse Outcomes:**
        - Permanent Hypopigmentation
        - Scarring + Possible Retraction
        - Allopecia
        - Nail Dystrophy

Biopsy Techniques:

- Many different Techniques
- Technique depends on the Nature of the Lesion & Information Required.
- **Types:**
    - **Punch biopsy [View Video Clip]**
        - Leaves Minimal Scarring
        - Best for Cosmetically Sensitive Areas – (Eg. Face)
    - **Shave Biopsy [View Video Clip]**
        - Fast and easy to perform.
        - Requires little equipment.
        - A shave biopsy is excellent for nodular bulky lesions which are easy to shave off for histology.
    - **Curette biopsy [View Video Clip]**
        - Fast and easy to perform.
        - Requires little equipment.
        - A shave biopsy is excellent for nodular bulky lesions which are easy to shave off for histology.
    - **Incision Biopsy [View Video Clip]**
        - A biopsy set is required with scalpel, forceps, needle holders and fine scissors as well as a suture to perform a small incisional ellipse. Be sure to go through the dermis to get a full thickness specimen.
        - Advantages:
        - Provides the best specimen for the pathologist to assess the tissue adequately
    - **Excision biopsy [View Video Clip in the Basic Cutaneous Surgery section]**
        - Similar to an incisional biopsy but the whole lesion is excised.

        - Good pathology specimen.

Wound Design:

- Wounds should be designed so that scars sit in or parallel with the relaxed skin tension lines or the cosmetic junction lines.
- Excision margins are important to ensure complete removal of the lesion. For benign lesions the margins are usually 2mm to 5 mm. For malignant lesions the margins can vary from 3mm up to 2 cm depending on the nature of the pathology.

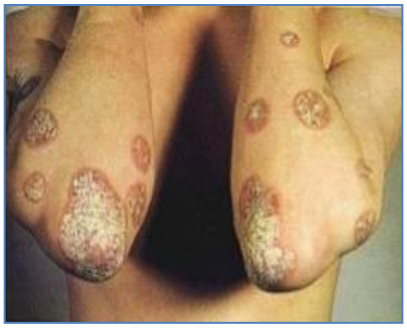
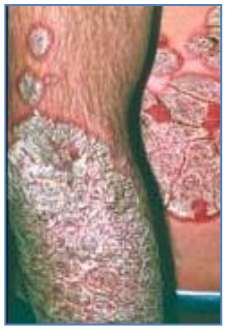
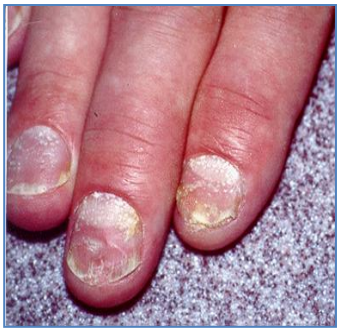
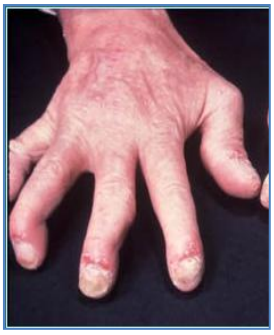
Surgical Wound Repair:

- **Suture Materials:**
    - **Synthetic Non-Absorbable:**
        - Nylon
        - Polypropylene
        - (Used on external surfaces where they are easily removed)
    - **Synthetic Absorbable:**
        - Monocryl
        - Vicryl (Polylactin)
        - (Broken down by the body via Hydrolysis &/or Proteolytic Enzymatic Degradation)
    - **Non-Synthetic Non-Absorbable:**
        - Silk
    - **Non-Synthetic Absorbable:**
        - Catgut (Being Phased out)
- **Suture Sizes:**
    - Scale from 1-6 (1 = Thinnest (0.4mm); 2 = Thickest (0.8mm)
- **Surgical Wound Repair:**
    - **Primary Repair:**
        - 6-8hrs
    - **Delayed Primary Repair**
        - In 72hrs
        - In surgical conditions
    - **Secondary Repair:**
        - After 72hrs
        - In surgical conditions
- **Important Pre-Requisites:**
    - **Wound needs to be CLEAN!**
        - Eliminate Dead Tissue/Dead Space/Haematoma/Foreign Bodies
    - **Finest Suture Material**
    - **Minimise Tension**
    - **Suture Removal @ Appropriate time for site of injury.**
        - 1-2 wks.

DERMATOLOGY Pathology:
Psoriasis

Psoriasis:

- **Aetiology:**
  - Multifactorial (Genetic & Immune)
- **Pathogenesis:**
  - not completely understood
  - Important role of ***T-cell immunity*** is recognised.
  - Emotional stress and physical trauma both exacerbate and precipitate psoriasis.
  - Strongly Familial
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - Plaque covered with Silvery Scales (Due to *Hyperkeratosis* & *Parakeratosis*)
    - Bilateral
    - Well-Demarcated
    - Erythematous Based
- **Clinical Significance (List 3x Clinical Features):**
  - Psoriasis may present at any age from infancy onwards.
  - Chronic Condition
  - Red (Erythematous Base)
  - Scaly
  - Well-Defined Plaques (Differentiates it from Dermatitis)
  - NOT itchy
  - Associated Psoriatic Arthritis
  - Abnormalities of the Nails (Dystrophy/Pitting/Onycholysis)
  - ***Auspitz Sign*** - Microbleeding when crusts are removed.

Diagnosis:

- clinical diagnosis made on the basis of morphology

Treatment:

- **Topical Anti-Inflammatory Agents.**
- **Topical Steroids**
- **Topical Tar-Containing Creams/Shampoo/Moisturisers.**
- **Dithranol (NB: Stains Brown)**
- **Methotrexate (Suppresses bone marrow)**
- **Sunshine/Phototherapy**
- **Bedrest**

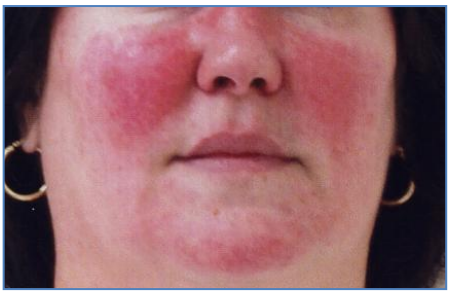
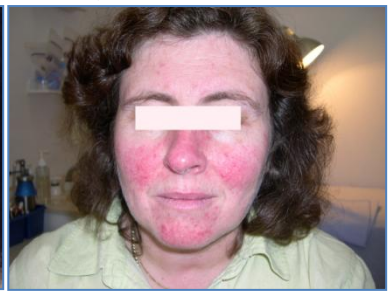
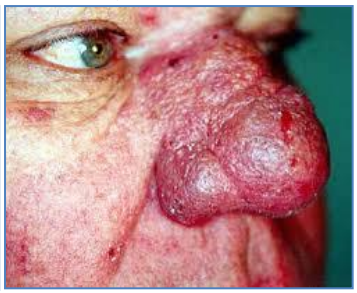
Prognosis:

- Chronic
- NOT Curable
- Can be exacerbated by Stress

DERMATOLOGY Pathology:
Rosacea

ROSACEA:

- **What is it?:**
  - Predominantly a Facial Rash easily confused with Acne.
  - = Pustules and Papular Rash on Face
  - *Typically a Disease of Middle Age (30-40yrs)*.
- **Aetiology:**
  - **Unknown**
  - (But Familial Association)
  - **Aggregating Factors:**
    - Heat and steam
    - Hot, spicy food
    - Alcohol consumption
    - Emotional stress
    - Sun exposure
- **Presentation:**
  - Wide Variation in Severity
  - **Initial Signs:**
    - Tendency to Flush easily + Burning/Stinging/Itching.
  - **Distinguishing Features:**
    - Facial Flushing (Erythema)
    - Dilated, visible Capillaries (Telangiectasia)
    - Papules
    - Pustules
    - But NOT Comedoens
  - **If Severe:**
    - Disfiguring Facial Rash
    - + Bulbous Enlargement of the Nose
    - Possible Facial Oedema
  - ***NO Comedones* (:. NOT Acne)**
  - Does not cause Scarring
  - Very Chronic (Not Self Limiting – May last for many years)
  - (NB: Often significant Psychosocial Impact – Eg. Depression)
- **Diagnosis:**
  - **Differential Diagnoses:**
    - Acne (has all features + Comedones)
    - Sun Damage (has Telangiectasia, but No other Features)
    - Lupus Erythematosus (Have Telangiectasia & Erythema, But NO Pustules or Papules)
    - Menopause (Flushing)
- **Treatment:**
  - Avoidance of Aggravating Factors
  - Similar Treatment to Acne.
    - Antibiotics (Some have Anti-Inflammatory Effects)
    - Retinoids
  - Laser surgery (for Telangiectasia & Erythema)
  - **Topical corticosteroids make rosacea worse and should never be used to treat it!**
    - → Cause Perioral Dermatitis (Should NEVER be used in Rosacea)

DERMATOLOGY Pathology:
Seborrhoeic Dermatitis

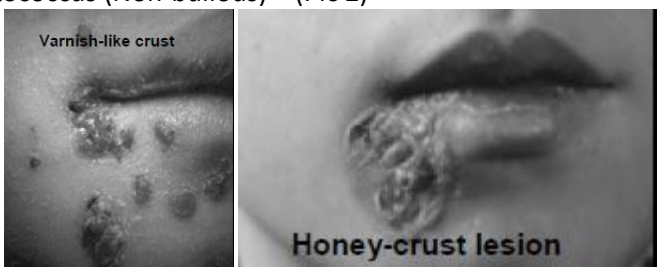
Seborrhoeic Dermatitis – 2 Forms:

- **Infantile Seborrhoeic Dermatitis:**
  - **Clinical Presentation:**
    - Numerous Dermatoses in the 1st 3 Months of Life.
    - Erythematous but *Non-Itchy* Rash involving the face, scalp, neck, axillae and nappy area. The lesions are well defined and covered in greasy scale.
- **Adult Seborrhoeic Dermatitis:**
  - **Clinical Presentation:**
  - Erythema and fine, greasy scale on the cheeks, nose and nasolabial folds.
  - Scale and itching of the scalp and eyebrows.
  - Well defined but non scaly erythema of the axillae, groin, scrotum and perianal skin.

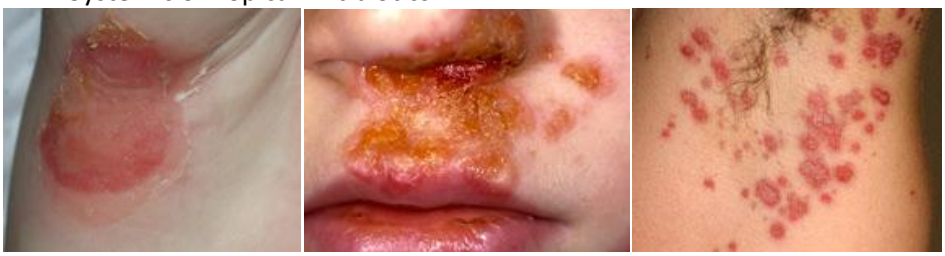
DERMATOLOGY Pathology:
Skin Infections – Bacterial

BACTERIAL SKIN INFECTIONS:

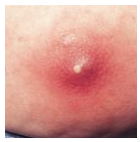
- **Impetigo (AKA "School Scores"):**
  - **What is it?**
    - Superficial Bacterial Skin Infection
    - Most Common in school kids
    - Very Contagious – (Spread by Close Contact & Poor Hygeine)
    - Usually resolves slowly
  - **Organism:**
    - **Mostly *Staphylococcus Aureus***
    - **Sometimes *Streptococcus Pyogenes***
      - Can lead to Glomerulonephritis or Rheumatic Fever if it's Strep.
    - Staph. Aureus (Bullous) - (Pic 1)
    - Streptococcus (Non-bullous) – (Pic 2)



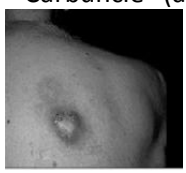
  - **Presentations:**
    - Occur most commonly on face
    - Fragile vesicles rupture & crust
    - Can be confused with HSV
    - **1. Nonbullous/Crusted Impetigo:**
      - (Most common)
      - Yellow crusts and erosions
      - Itchy/Irritating (but not painful).
    - **2. Bullous impetigo:**
      - Always due to *S. Aureus*
      - →Mildly irritating blisters that erode rapidly leaving a brown crust.
    - **3. Ulcerative lesions**:
      - Always due to *S.pyogenes*.
      - Most common in Aboriginal Communities
  - Very Infectious
    - Epidemic in young children
    - Transmitted through skin contact
    - Outbreaks associated with poor hygiene / crowded living conditions
  - **Treatment:**
    - Cover Affected Areas
    - Abstain from School
    - Systemic or Topical Antibiotics



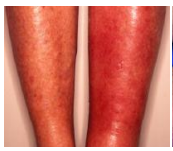
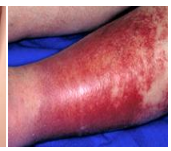
- **Folliculitis & Furunculosis (Boils):**
  - **What is it?**
    - **Folliculitis:**
      - Acute pustular infection of a hair follicle
      - Commonly after Waxing/Shaving
    - **Boils ("Furuncles"):**
      - A *deep form* of folliculitis.
  - **Organism:**
    - *Staphylococcus Aureus*
  - **Presentation:**
    - **Folliculitis:** An Erythematous Pustule centered on a Hair Follicle.
    - **Boils ("Furuncles"):** Tender, red nodule which enlarges & may later discharge pus
  - **Treatment:**
    - Treated aggressively with antibiotics and drainage.

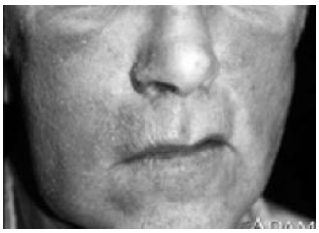
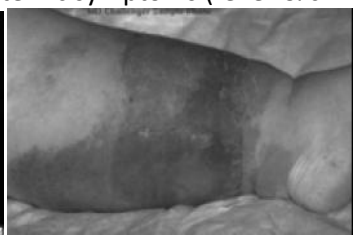
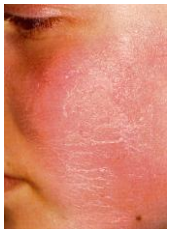
- **Abscesses:**
  - Usually *Staphylococcus Aureus*
  - Begin as superficial infections of hair follicles (folliculitis)
    - →Organisms travel down Hair Follicles following Disruption (Eg. After Shaving)
    - →Development of boils (furuncles)
  - Number of boils cluster together = "Carbuncle" (aka Abscess)



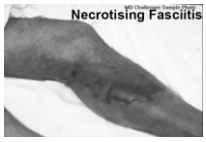
- **Cellulitis:**
  - **What is it?**
    - Bacterial infection of the Dermis and Sub-Cutaneous Tissues
  - **Organism:**
    - **Adults:** 90% due to *Staph.Aureus*/GAS
    - **Children:** *H. influenzae* b
    - **Associated with cat/dog bite:** *Pasturella multocida*
  - **Presentation:**
    - Painful, raised and Oedematous Erythema. (Most commonly on Lower Leg)
    - Possible Blistering
    - Lymphadeopathy - & Malaise & Fever.
  - **Distribution:**
    - **Children** – Periorbital Area
    - **Adults** – Lower Legs
  - **There's typically an underlying cause:**
    - Lymphoedema
    - Tinea, Herpes simplex infection, Chronic sinus infection
    - Chronic dermatitis
    - Poor lower leg circulation
    - Wounds
  - **Treatment:**
    - Antibiotics

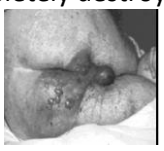
- **Erysipelas:**
  - **(2) Dermal Lymphatic Infection: Erysipelas:**
    - **Organism:**
      - Group A Strep (GAS)
    - **Presentation:**
      - (Looks like a bad case of Sunburn)
      - rash that is red, slightly swollen,
      - very defined, warm, and tender
      - Usually Unilateral
      - may spread to deeper lymphatics (lymphangitis)
      - may produce systemic symptoms (fever & chills)

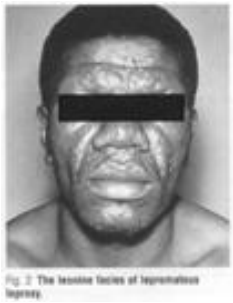
- **Necrotising Fasciitis:**
  - Medical Emergency - Often needs Radical Debridement of Necrotic Tissue
  - **Organisms:**
    - **Group A Strep (GAS)**
    - **Staph. Aureus**
      - (Both cause severe, systemic toxicity)
    - Others (Vibrio, Clostridium, Bacteroides)
  - **Pathogenesis:**
    - The Necrosis is *Toxin-Mediated* → Can't just treat with Antibiotics
      - (*NOT due to "Flesh-Eating Bacteria"*)
  - **Types:**
    - **Type I –** Polymicrobial Infection
    - **Type II –** Monomicrobial Infection



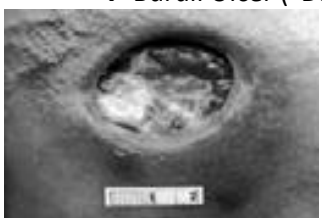
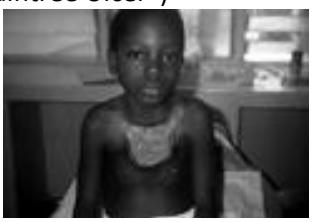
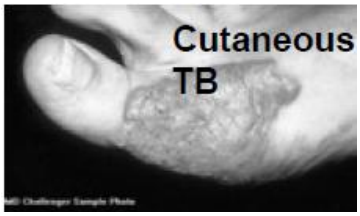
- **Gas Gangrene:**
  - **Organism:**
    - *Clostridium perfringens*
  - **Pathogenesis:**
    - Generally occurs at site of trauma or recent surgical wound
    - Usually only occurs with Poor Blood Supply (Eg. Diabetes)
      - →Anaerobic conditions
      - Toxins are produced → Cause the Tissue Death and associated Symptoms
  - **Presentation:**
    - Inflammation @ Site of Infection
    - Brownish-red and extremely painful tissue swelling
    - Gas may be felt in the tissue when the swollen area is pressed
    - Margins of the infected area expand rapidly (Within a few minutes)
  - **Prognosis:**
    - The involved tissue is completely destroyed (Toxin-Mediated Destruction)



- **Mycobacterial Infections:**
  - **Leprosy:**
    - Tuberculoid and lepromatous forms
    - Mainly affect skin and nerves

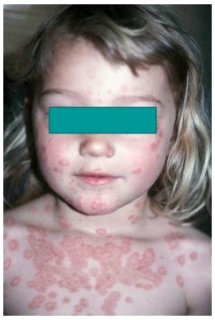
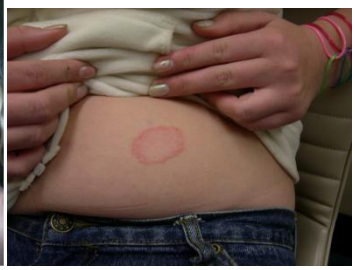
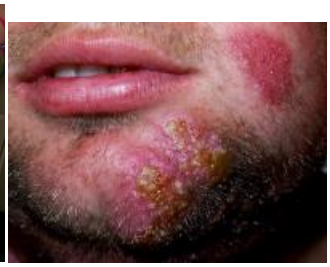
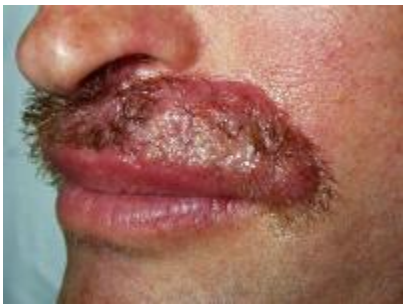
  - **Tuberculosis:**
    - Skin manifestations include lupus vulgaris, hypersensitivity reactions, warty plaques
    - → Buruli Ulcer ("Daintree Ulcer")

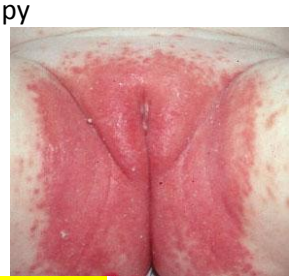
DERMATOLOGY Pathology:
Skin Infections – Fungal

Fungal Infections:

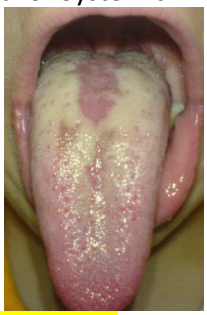
- Fungal Nail Infections (**Dermatophytes, Candida Albicans, or Moulds**)
- Superficial (skin, nails and hair):
- **Dermatophytes (Tinea):**
    - Eg. Ringworm Fungi.
        - **2 Common Genuses** - (*Tricophyton* & *Microsporum*)
        - Infection Restricted to the Superficial Keratinised Layers of the skin
            - NB: Keratin is the Fungi's food source.
        - Can Evoke Cellular immune responses
- **Pathogenesis:**
    - **Fungi ONLY Metabolizes Keratin:**
        - :. Only infect the Stratum Corneum
        - NB: Can Also Invade Hair Shafts
- **Presentation:**
    - *Annular* eruption with *Irregular edge*
    - Central clearing Peripheral scaling.
    - Focal hair loss due to infection of Hair Follicle.
    - Focal pityriasis (Skin Flaking)
    - Usually not pruritic
    - "Tinea Versicolor" (Depigmentation of the Skin)
- **Conditions Named Based On Location of Infection:**
    - Tinea Corporis (On Body)
    - Tinea Capitis (On Head)
    - Tinea Crura (Pubic Area)
- **Diagnosis:**
    - Clinical Diagnosis
    - Woods lamp – only *Microsporum canis* fluoresces
    - Microscopy of hairs/nail shavings/skin shavings
- **Treatment:**
    - **Topical Antifungals:**
        - Clotrimazole
        - Miconazole
    - **Oral Antifungals:**
        - Fluconazole

- **Yeasts (Eg. Candida):**
  - **Cutaneous Candidiasis – (Candida Albicans):**
    - (= Overgrowth of Normal Commensal of the mouth, vagina, or lower GIT.)
    - Only infects the outer layers of the epithelium of mucous membrane or skin.
    - **Presentation:**
      - Red, macerated area
      - Glistening Surface
      - Scaling along the advancing border.
      - The initial lesion is a papule that then becomes a pustule.
      - Important clinical feature = presence of 'satellite' pustules beyond the border of the main infection.
    - **Treatment:**
      - Topical Therapy



  - **Oral Candidiasis – (Candida Albicans):**
    - Presents as:
      - White Patches easily scraped off to leave a red, raw base.
      - Chronic red, raw gums, tongue and buccal mucosa.
      - Treatment: Topical or Systemic Therapy

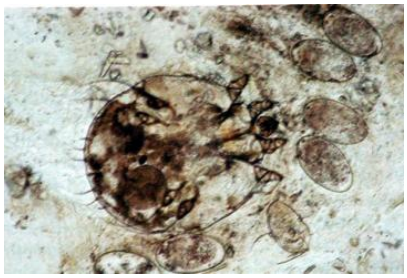
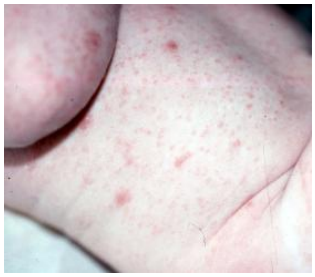


  - **Pityriasis versicolour – (Candida Albicans):**
    - Caused by normal Commensals – Eg. yeasts (Candida).
    - Common Superficial Fungal-Induced Rash
    - **Presentation:**
      - →flaky discoloured patches on chest & back.
      - Small, well defined, slightly scaly patches
      - Either Hyperpigmented or Hypopigmented

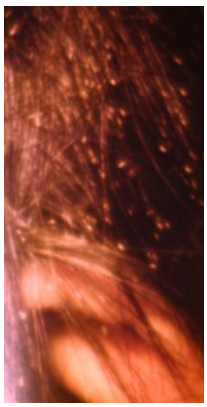
DERMATOLOGY Pathology:
Skin Infections – Parasitic

Insects:

- **Scabies:**
  - **Organism:**
    - ***Sarcoptes scabiei*** (Scabies Mite)
  - **Epidemiology:**
    - Human infestations originating from pigs, horses and dogs are mild and self limiting.
    - Scabies infestations from other humans never cure without intervention.
  - **Ecology:**
    - Mites live in stratum corneum (Don't get any deeper)
    - Eat stratum corneal Keratinocytes
    - Make "tunnels" by eating
    - Mating occurs on the hosts skin
    - Fertilized Female Mites Burrow into the Stratum Corneum (1 mm deep)
    - Salivary Secretions contain Proteolytic Enzymes →Digest Keratinocytes.
  - **Transmission:**
    - High prevalence in children (50%) and adults (25%) in tropical remote communities
    - Spread by close physical contact
  - **Presentation:**
    - Itch (Exacerbated at night and after hot showers).
    - Itchy, Excoriated Rash on Trunk, associated with Scaly Burrows on the fingers and wrists.
    - Often vesicles and pustules on the palms and soles and sometimes on the scalp.
  - **Diagnosis:**
    - **Clinical Diagnosis:**
      - Chronic itch with Symmetrical Rassh
      - Burrows
    - **Skin Scraping - Look for Scabies Mites:**
      - Intact larvae, nymphs or adults
      - Unhatched or hatched eggs
      - Moulted skins of mites
      - Fragments of moulted skins
      - Mite faeces
  - **Treatment:**
    - **Topical** Permethrin
    - **Or Oral** Ivermectin (But not on PBS – Very Expensive)
    - **Environmental Measures:**
      - Mites can contaminate bedding, chairs, floors, and even walls
        - (Usually only a problem with crusted scabies)
      - Wash, sun, vacuum, surface insecticide
    - **Community Prevention:**
      - Treat all close contacts – Esp. in Indigenous Communities
      - Simultaneous Effective Treatment
    - ***TREAT AGAIN IN 7 DAYS***

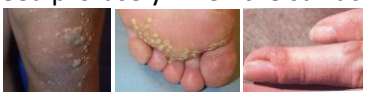
- **Head Lice:**
  - **3 Types:**
    - **1. Head Lice: *Pediculus Humanus Capitis***
      - **Epidemiology:**
        - Common in Primary School Children in the Tropics
        - Higher prevalence in Aboriginal Children
      - **Diagnosis:**
        - Conditioner + Fine-Tooth Come
        - Wipe combings on white tissue paper
    - **2. Body Lice: *Pediculus Humanus Corporis***
      - Live on clothes, and come to the body to feed.
    - **3. Pubic Lice: *Phthirus Pubis***
      - Largely sexually transmitted
      - Blood Feeder
      - Can infect any Body Hair (Pubic/Trunk/Legs/Axilla/Beard) but rarely head.
  - **Lifecycle:**
    - Eggs laid in hair (knits)
    - Larvae grow into adults
    - Adults – **blood sucking** (live in hair)
  - **Transmission:**
    - head-head contact.
  - **Presentation:**
    - Scalp and Neck can be Itchy
    - Nits are noticeable on the hairs.
  - **Diagnosis:**
    - **Best Method = *'Conditioner & Comb Technique'*:**
      - Very Practical for parents
      - Cost Effective
      - High Sensitivity
      - Conditioner 'Stuns' the lice by suffocating them → Prevents them from running away
  - **Management/Treatment:**
    - Conditioner & Nit Comb
    - Physical Removal
    - Cut Hair
    - Topical Insecticidal Cream
    - Good idea to wash pillows and hats though – Hot Wash
    - (Treat all body hair – for Pubic lice)
    - **Reasons for Treatment Failure:**
      - Inadequate application of the product
      - Lice are resistant to insecticide
      - Failure to retreat to kill nymphs emerged from eggs
      - Reinfection.

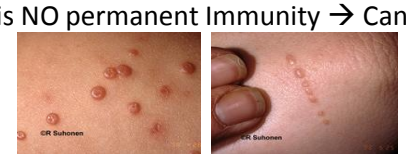
DERMATOLOGY Pathology:
Skin Infections – Viral

Viral Infections:

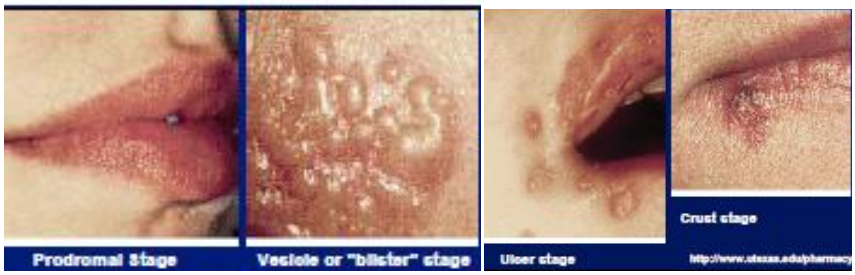
- **Viral Warts**
  - **What are They?**
    - = Benign Tumours of the skin
    - Common in children
    - Infectious – (Spread by direct contact)
  - **Organism:**
    - Typically from HPV (human papilloma viruses)
    - HPV 6 & 11 → Genital & Cutaneous Warts
    - HPV 16 & 18 → Cervical & Penile Ca
  - **Appearance:**
    - Verrucuous surface
    - Can often see a tiny black dot in the middle due to thrombosed capillary blood vessels
  - **Presentation:**
    - Common on back of fingers, toes and knees
    - Common Warts (Skin/Plantar/Palmar)
    - Genital Warts (Cervix, Vulva, Penis)
      - NB: Cervical Papillomas → Can cause cervical cancer.
    - Laryngeal Papilloma
  - **No Reliable Treatment:**
    - 50% of childhood warts disappear within 6mths; 90% are gone in 2 years.
    - Many don't bother with treatment.
    - Surgical Excision/Chemical Treatment/Cryotherapy/Electrosurgery (Cauterise)
- **Clinical Significance (List 3x Clinical Features):**
  - Contagious
  - Central blood vessels → Bleed profusely when the surface is broken.



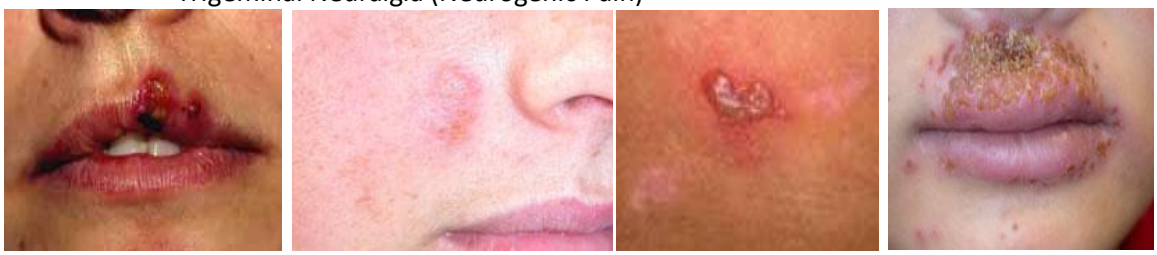
- **Molluscum Contagiosum:**
  - **Aetiology:**
    - **Poxvirus**
  - **Transmission:**
    - Skin to Skin Contact
  - **Epidemiology:**
    - Most Common in Children
    - Also Sexually-Active People
  - **Lesions:**
    - Dome Shaped Papules
    - ***Dimpled Centre (Centrally Umbilicated)**
    - Not Painful, but may Itch.
  - **Treatment:**
    - Often Unnecessary - May Resolve on its Own.
    - – May Require Antibiotic Treatment if Secondary Bacterial Infection Occurs.
    - Cryotherapy,Curettage,Laser,Acid
  - **Prognosis:**
    - Once Resolved, the virus is GONE.
      - (No Latent Stage like Herpes Viruses)
    - However, there is NO permanent Immunity → Can catch it again.



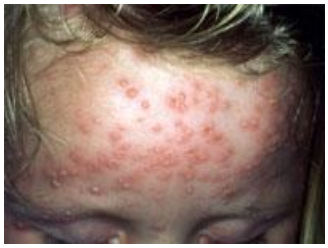
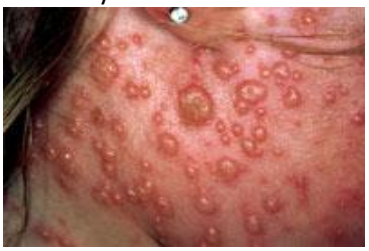
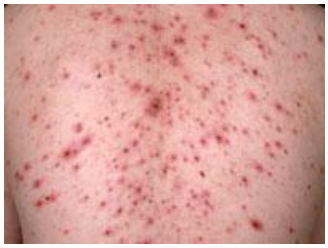
- **Herpes Simplex (Cold Sores/Genital Lesions):**
    - **What is it?**
        - Common Mucosal Viral Infection that presents with localised blistering
        - Can reside in a latent state
    - **2 Types:**
        - **Type 1: Typcially facial/oral infections** (Cold sores/fever blisters)
            - Occur mainly in infants & young kids
        - **Type 2: Mainly Genital**
            - Occur after puberty (often transmitted sexually)
    - **Presentation:**
        - **Stages of Infection:**
            - 1. Prodromal Stage Vesicle or "blister" stage
            - 2. Ulcer stage
            - 3. Crust stage



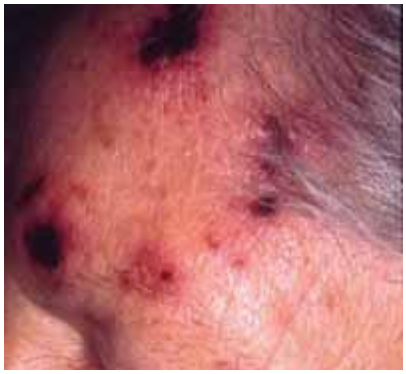
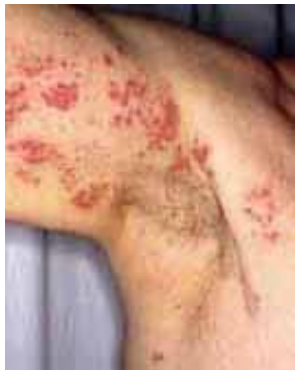
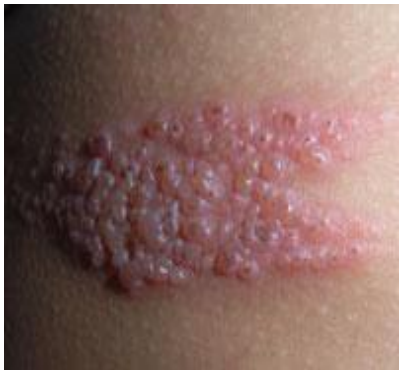
        - The virus grows down the nerves and out into the skin → Localised Blistering
        - Neuralgia
        - Lymphadenopathy
        - High Fever
    - **Recurrences can be triggered by:**
        - Minor trauma/Other infections/UV radiation/Hormonal factors/Emotional stress/Operations/procedures on face
    - **Treatment:**
        - Mild cases require no treatment
        - Sun protection to prevent
        - Oral Antiviral Drugs (Stop the virus multiplying)
    - **Complications:**
        - Encephalopathy
        - Trigeminal Neuralgia (Neurogenic Pain)



- **Chicken pox (Herpes Varicella Zoster):**
  - **What is it?**
    - Highly contagious disease
    - Typically childhood disease (before 10yrs)
    - One infection thought to confer lifelong immunity
  - **Organism:**
    - Varicella zoster virus (HHV3) (AKA: Chicken Pox Virus, Varicella, Zoster)
  - **Transmission:**
    - Highly Infectious
    - From person to person
    - Aerosol Droplets
    - Direct contact with fluid from open sore.
  - **Pathophysiology:**
    - Incubation Period ≈ 2wks.
    - **(Chicken Pox)** Initial Mucosal Infection → Viraemia → Epidermal Lesions
      - May lead to → Latent infection of Dorsal Ganglion Cells of Sensory Nerves.
    - **(Shingles)** Reactivation of latent Varicella Zoster Virus in Peripheral Nerves
  - **Signs/symptoms:**
    - Itchy rash or red papules
    - Begins on the Trunk →Face and Extremities
    - May cover entire body
    - High fever/headache/cold-like symptoms/vomiting/diarrhoea.
  - **Diagnosis:**
    - Clinical Diagnosis
    - Immunoflourescence
    - Test for Elevated VZV-Specific Antibodies
      - (IgM – Primary Infection; IgG – Second Infection)
  - **Treatment:**
    - Symptomatic
    - Resolves on its own.
  - **Complications:**
    - **Varicella During Pregnancy can → Congenital Varicella Syndrome:**
      - **Spontaneous Abortion** (3-8% in 1st Trimester) or IUGR
      - **Skin:** Cutaneous Defects, Hypopigmentation
      - **Neuro:** Intrauterine Encephalitis, Brain Damage, Seizures, Developmental Delay
      - **Eye:** Chorioretinitis, Cataracts, Anisocoria
      - **MSK:** Limb Hypoplasia
      - **Systemic:** cerebral cortical atrophy
      - **Renal:** Hydronephrosis, Hydroureter
      - **GI:** GORD
      - **CVS:** Congenital Heart Defects
    - **Perinatal Varicella Infection:**
      - severe → mortality rate of 30%

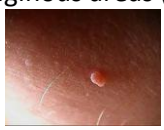
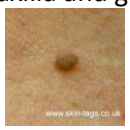
- **Shingles (Herpes Varicella zoster):**
  - **What is it?**
    - Reactivation of Latent Herpes Varicella Zoster Virus.
  - **Pathophysiology:**
    - Incubation Period ≈ 2wks.
    - **(Shingles)** Reactivation of latent Varicella Zoster Virus in Peripheral Nerves
  - **Presentation:**
    - Painful blistering rash along 1/more Dermatomes.
    - Virus is seeded to nerve cells in spinal cord
    - Fever, malaise and headache
    - Lymph nodes draining affected area are often enlarged/tender
    - Can also result in nerve palsy
  - **Diagnosis:**
    - Clinical Diagnosis
    - Test for Elevated VZV-Specific Antibodies
    - PCR
  - **Transmission:**
    - Shingles are infectious
    - From person to person
    - Direct contact with fluid from open sore.
  - **Treatment:**
    - Antiviral treatment
    - Rest & analgesia
    - Oral Antiviral

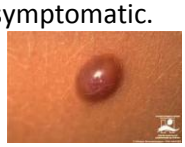
DERMATOLOGY Pathology:
Skin Lesions - Benign

BENIGN:

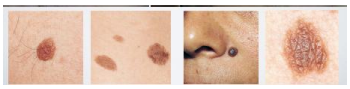
- **Skin Tags:**
  - These harmless lesions
  - They are pedunculated and have a narrow pedicle. They are usually skin coloured to brown. Common sites are the intertriginous areas (axilla and groin) and the neck.

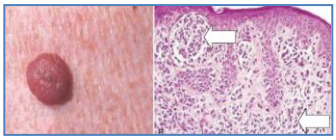
- **Dermatofibromas:**
  - This is a harmless and common lesion found in adults and children. It presents as a skin coloured to light brown firm papule which is asymptomatic.



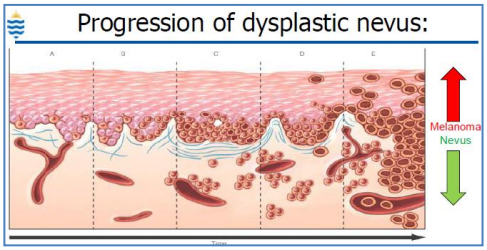
- **Nevi:**
  - **Aetiology:**
    - Congenital (After Birth)
  - **Pathogenesis:**
    - **Freckles =** Excess Pigment
      1. Arise in response to UV-Radiation Exposure.
         1. May Regress if Exposure is Avoided.
    - **Moles (Lentigo) =** Excess Melanocytes
      1. These are benign tumours of melanocytes
      2. Are normal.
      3. The average number (that are > 2mm in diameter) found on the skin of Australians is close to 100.
      4. They present as discrete, light to dark brown lesions and are mostly 2-5 mm in diameter.



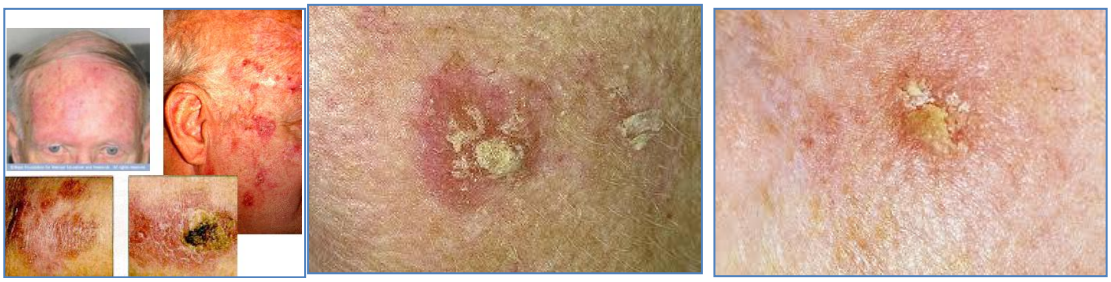
    - **Birthmark =** Excess Melanocytes *AND* Excess Pigment
      1. Small, Flat, Symmetric, Uniform Lesions
      2. Cluster of Clear Cells (Melanocytes) @ the Dermo-Epidermal Junction
      3. NB: Proliferation below Basal Layer.
    - **Compound Nevus:**
      1. Small, Raised, Dome-Shaped, Symmetric, Uniform Lesions.
      2. Big Clusters of Melanocytes in Dermis & Dero-Epidermal Junction.



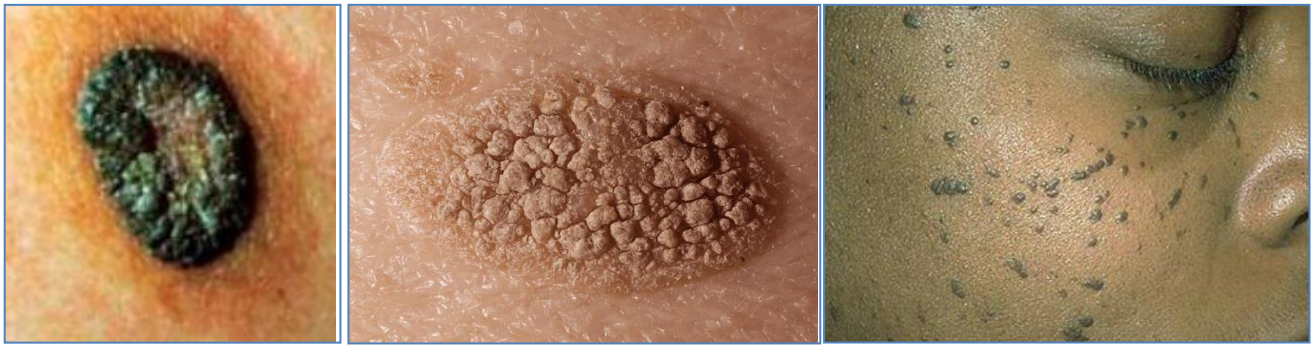
    - **Dysplastic Nevus:**
      1. Pigmented, Raised Lesion, with Central Darker Shade.
      2. DE-Junctional Cluster of Dysplastic (Larger/Irregular/Darker) Melanocytes.
      3. **NB: Can Progress to Melanoma:**
         1. Once the Dysplastic Melanocytes begin Infiltrating UP into the Epidermis.



- **Actinic Keratosis (Sun Damage):**
  - **Aetiology:**
    - Sun Damage
  - **Pathogenesis:**
    - = Damage to the skin from UV (wrinkling, pigmentary change, actinic keratoses and cancer)
    - Sun Damage due to Chronic Exposure to UV Radiation
  - **Morphology (List 3x Gross & 3x Microscopic Features):**
    - Red/Tan, Irregular, Scaly Plaques
    - Hyperkeratosis
    - Inflammation/Ulceration/Crusting
  - **Clinical Significance (List 3x Clinical Features):**
    - The earliest sign = Freckling.
    - A Pre-Cancerous Skin-Growth



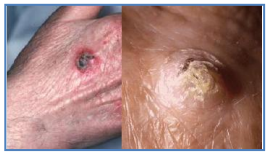
- **Seborrheic Keratosis:**
  - **Aetiology:**
    - Totally Benign Tumour in Old Age (Common in >40yrs)
  - **Pathogenesis:**
    - Unknown
  - **Morphology (List 3x Gross & 3x Microscopic Features):**
    - Sticky, Oily Plaques
    - Round, Flat, Velvety Plaques
    - May be Pigmented
  - **Clinical Significance (List 3x Clinical Features):**
    - Treated only if inflamed
    - No Malignant Potential



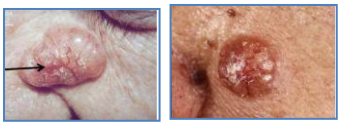
DERMATOLOGY Pathology:
Skin Lesions – Malignant

MALIGNANT:

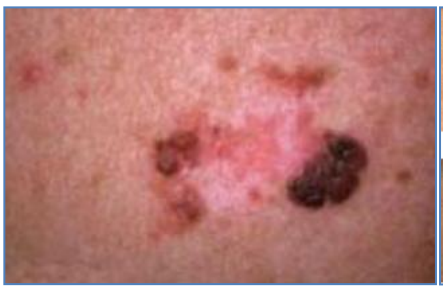
- **Squamous Cell Carcinoma (SCC):**
  - 2nd commonest skin cancer after BCC
  - **Aetiology:**
    - Sun Damage
    - Industrial Carcinogens
    - Tobacco
  - **Pathogenesis:**
    - Dysplasia & Tumorigenesis of Squamous Epithelial Cells.
  - **Morphology (List 3x Gross & 3x Microscopic Features):**
    - Nodule
    - Hyperkeratosis
    - Sometimes Erythematous base.
  - **Clinical Significance (List 3x Clinical Features):**
    - Malignant (Early Treatment via Excision is Essential)
    - Highly Variable Appearance
    - Typically Tender Papules/Nodules with *ROUGH, ADHERENT SCALE*.
    - Often Ulcerate Centrally
    - Often Present as Ulcers on Lips/Mucosa
    - They frequently grow rapidly and may be painful.
  - **Treatment:**
    - Wide excision
    - Cryotherapy
    - Radiotherapy in Elderly

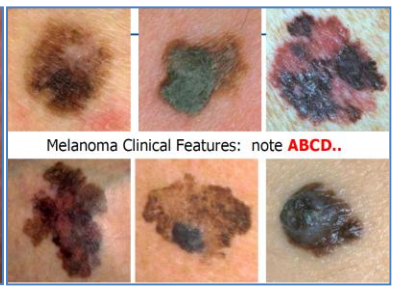


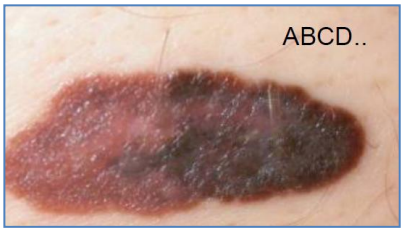
- **Basal Cell Carcinoma (BCC):**
  - **Aetiology:**
    - Sun Exposure
  - **Pathogenesis:**
    - Dysplasia & Tumorigenesis of Basal Epithelial Cells.
    - Slow Growing
    - Rarely Metastasises (Locally Infiltrative, but don't often Metastasise)
  - **Morphology (List 3x Gross & 3x Microscopic Features):**
    - Nodular/Papular
    - Blood Vessels
    - Pearly/Shiny crust over the lesion.
    - Large Tumours may Ulcerate.
  - **Clinical Significance (List 3x Clinical Features):**
    - #1 Commonest skin cancer in Aus
    - Good prognosis if treated early
    - **Treatment:**
      1. **Excisional surgery** : Still the gold standard treatment of BCC's.
      2. Liquid nitrogen cryotherapy
      3. Curettage and Cautery
      4. Photodynamic therapy (PDT)
      5. Radiation treatment



- **Melanoma:**
  - **Aetiology:**
    - Sun Damage
    - Congenital
  - **Risk Factors:**
    - Family history
    - Tendency to burn (Fitzpatrick type I/II skin)
    - Severe Childhood Sunburn (Solar Skin Damage)
    - Immunosuppression
    - High number of common acquired naevi
  - **Pathogenesis:**
    - Tumorigenesis of Melanocytes
    - Most develop in an existing Naevus
    - Superficial Spreading melanomas grow Laterally Initially, then begin to grow downwards.
  - **Morphology (List 3x Gross & 3x Microscopic Features):**
    - Grow & Change Colour
    - Maculo-Papular Lesion
    - ***Irregular Shape, Colour & Depth – Distinguishing Features.***
    - Pigmented
  - **Clinical Significance (List 3x Clinical Features):**
    - Malignant
  - **Prognostic Factor:**
    - **Breslow Thickness**
    - **Clark Levels**
    - **TNM Staging:**
      1. **T = Size/Depth**
      2. **N = Nodal Involvement**
      3. **M = Distant Metastasis?**
  - **Diagnosis:**
    - Dermoscopy is ESSENTIAL!!
    - **ABCD's of melanoma:**
      1. **A – Asymmetry o**f Shape and Structure.
      2. **B – Border Irregularity**. The edges are ragged, blotched, or blurred.
      3. **C – Colour Variability**. The pigmentation is not uniform.
      4. **D – Diameter Increasing**. A width >6mm (about the size of a pencil eraser).
      5. **E – Evolving/Elevated**
      6. **F – Firm/Friable**
      7. **G – Growing**. Signs of growth (Slow/Rapid) should raise concern.
    - **3 Point Checklist (>2 = Malignant):**
      - 1. Assymmetry of Colour/Structure?
      - 2. Atypical Network?
      - 3. Blue-White Structures?
  - **Treatment:**
    - **Early Detection & Removal** = The Only Reliable Curative Treatment.
    - **Followup** = Those who have had a melanoma, have a 13% risk of another.







ABCD(E):

Asymmetry, Borders (Irregular), Colour (Varied), Diameter (Greater than 6mm), Evolving over time.

PATH
Skin Pathology & Histology

Objectives:

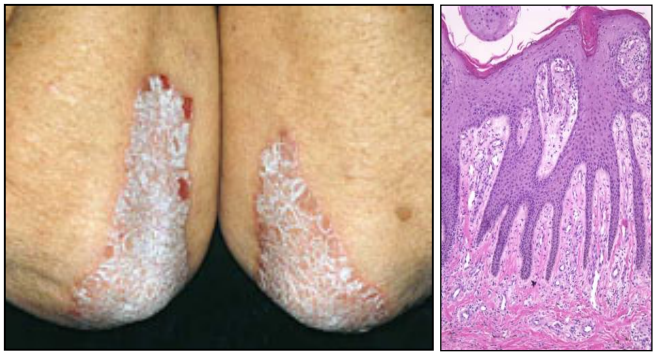
- Anatomy/Histology/Physiology
- *Pathology of Major Disorders
    - Aetiology, pathogenesis, morphology

Principles of Pathology – Things to know for each of the presented conditions:

- **1. What Caused the Disease (Aetiology)**
- **2. How does it Occur/Progress? (Pathogenesis)**
- **3. What are the Gross/Microscopic Changes? (Morphology)**
- **4. Clinical Significance (Complications, prognosis, etc)**

Example Exam Question:

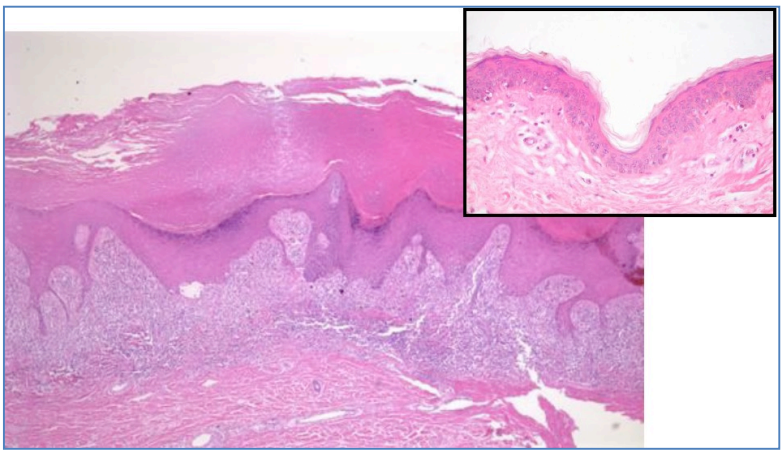
- **Given a Picture (Macro & Micro):**



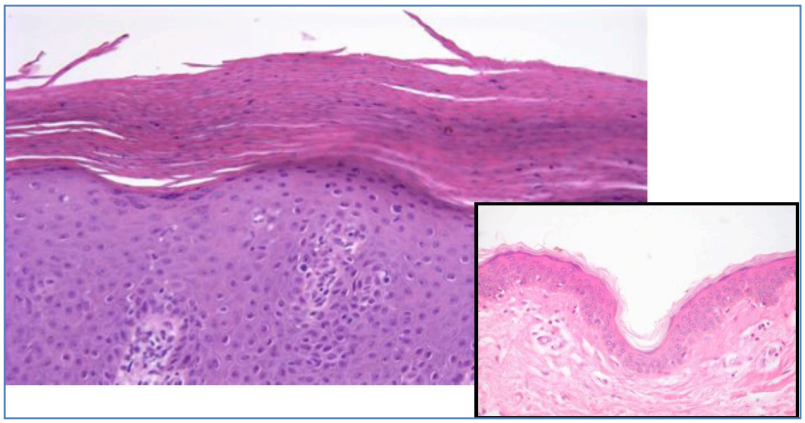
- **Given a Case:**
    - 19yr old female presents with chronic skin lesions – Flared up in winter months – responds partially to topical steroid ointment
- **Describe the appearance of the lesion:**
    - **Eg. Macroscopic Features:** Bilateral, scaly, flaking, raised plaques on erythematous base
    - **Eg. Histological Features:** Inflammatory infiltrate, Deep Rete Ridges, Raised Dermal Papillae.
- **What is the most likely diagnosis?**
    - Psoriasis
- **Describe the "Koebner Phenomenon"?**
    - The development of isomorphic psoriatic lesions immediately subsequent to, and at the site of, a cutaneous injury;
- **What is Auspitz sign? What is its significance?**
    - ***Pinpoint Bleeding*** *following Removal of the scales because of increased vascularity under focal areas of epidermal thinning. (This feature occurs only in psoriasis)*
- **Identify any 4 histopathological features shown.**
    - *Hyperkeratosis*
    - *Parakeratosis*
    - *Regular elongation of the rete ridges*
    - *Diminished granular layer*
    - *Tortuous papillary dermal blood vessels*
    - *Inflammatory cells in the superficial dermis.*
- **You are prescribing a medication for her, what type of base would be suitable for her and why?**
    - *Water soluble Cream base with keratolytic agent such as urea, for hydration and good penetration as the lesion is dry & scaly.*

Common Histopathological Terms:

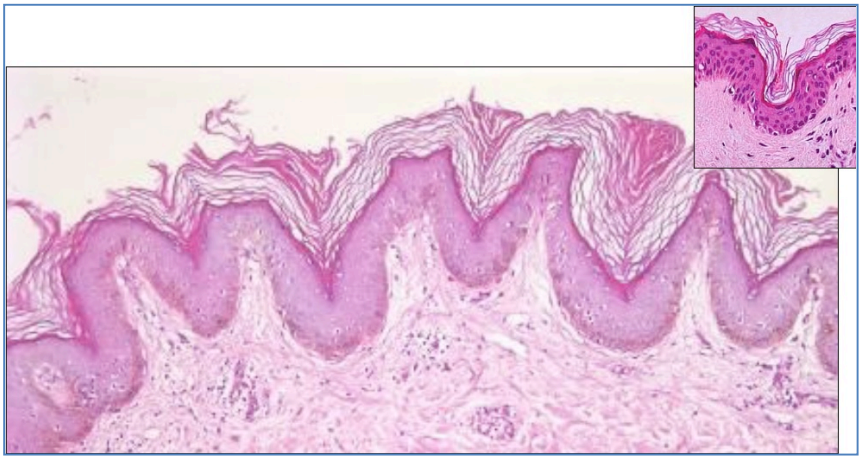
- **Hyperkeratosis:**
  - Hyperplasia of the Stratum Corneum
  - NB: Also Thickening of the Dermis & Rete ridges
  - Clinical appearance = scaling
    - Psoariasis, eczema.



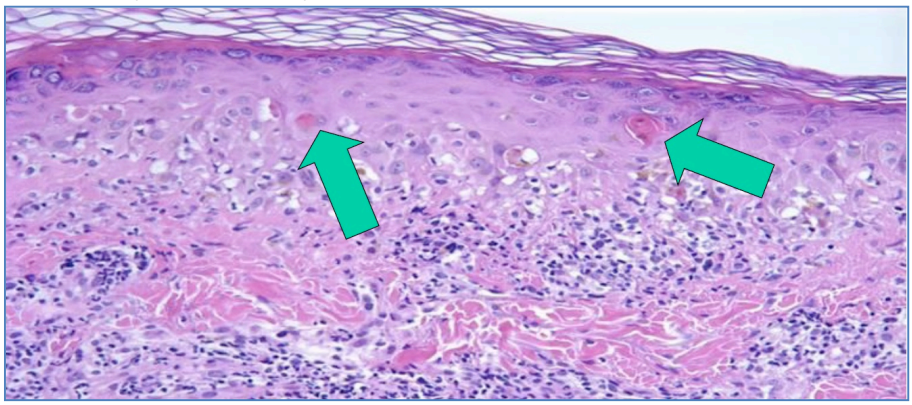
- **Parakeratosis:**
  - A Pattern of Keratinization Characterized by **Retention of Nuclei** in the stratum corneum (due to very very rapid keratinisation –no chance to mature & lose the nucleus)
    - (NB: This is normal on mucous membranes)
  - Seen in many cases of hyperkeratosis
  - Clinical appearance = Scaling



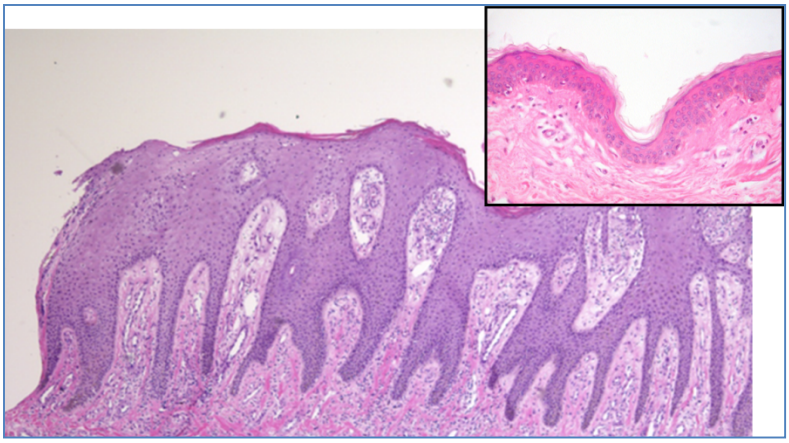
- **Papillomatosis:**
  - Hyperplasia of the Papillary Dermis
  - (The whole skin becomes folded – Papillar Projections – Typical in infections)
  - Caused by Neoplastic-verrucous ca, venous stasis, viral.



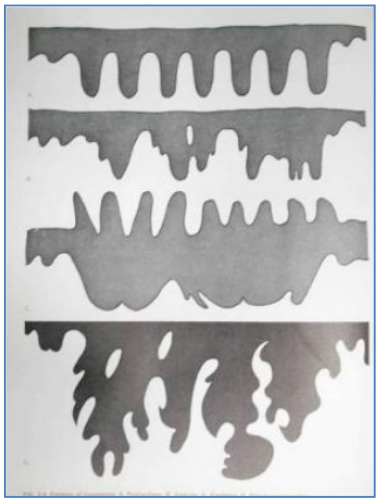
- **Dyskeratosis:**
  - Abnormal Keratinization due to Carcinoma.
    - Very typical of malignancy
  - Cells containing high amounts of keratin beneath the Stratum Corneum
    - → Keratin Cysts
  - NB: Also many inflammatory cells



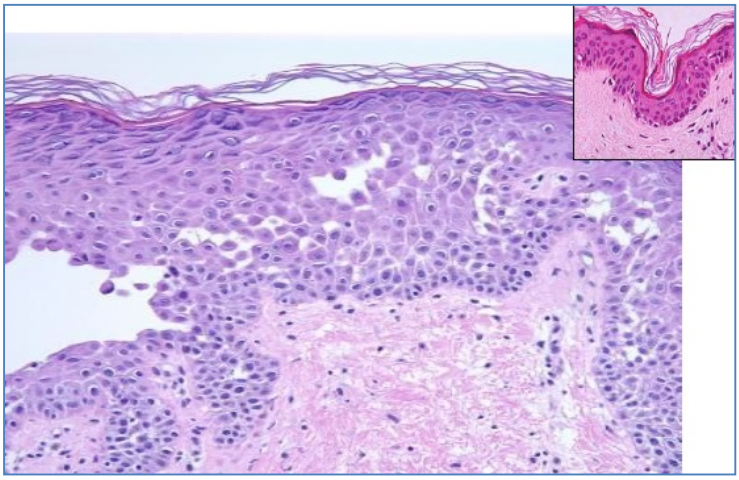
- **Acanthosis:**
  - Epidermal Hyperplasia (Increased number of epidermal cells, deepening of rete ridges) → Thickening of the Epidermis.
  - Seen in: Eczema, psoriasis

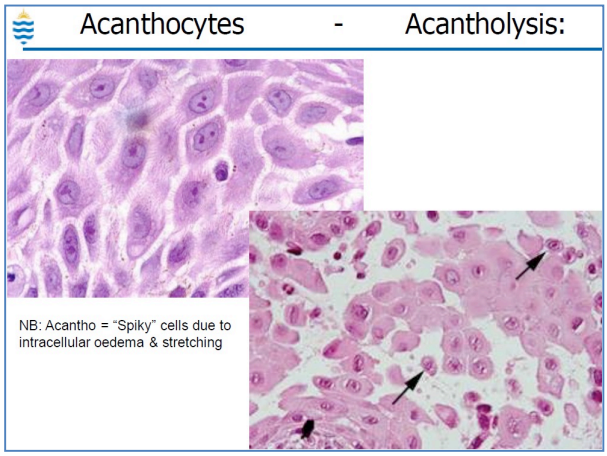


  - **Types of Acanthosis:**
    - **Psoriasiform (Regular) Acanthosis**
    - **Irregular Acanthosis**
    - **Papillated Acanthosis** (Epidermal Growth goes above the normal level)
    - **Pseudoepitheliomatous/pseudocarcinomatous** (Epidermal Growth Infiltrates into the dermis)

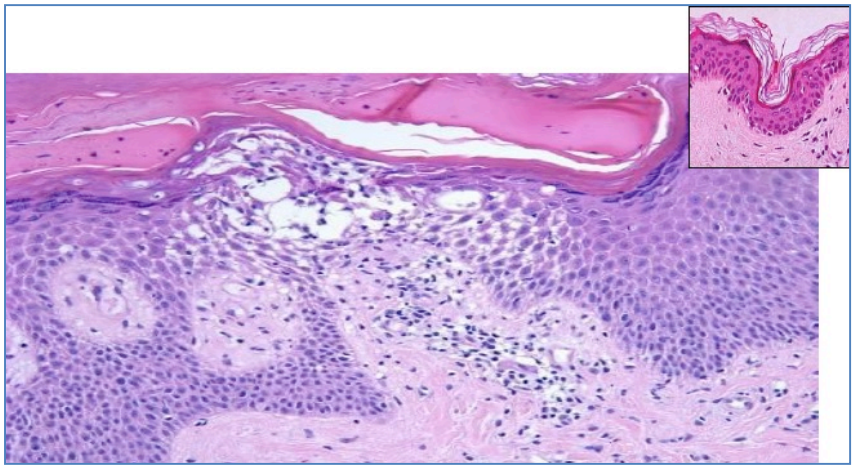


- **<u>Acantholysis:</u>**
  - Loss of Intercellular Connections.
    - Antibody-Mediated Destruction Against Desmosomes
    - → "Breaking down of brick wall" → Blisters
  - **Seen in:** Pemphigus (a group of Blistering Autoimmune Diseases)





- **<u>Spongiosis:</u>**
  - Inflammation and Intercellular Epidermal Oedema.
  - →Separation of cells without breakdown of cell-junctions (Not like Acantholysis)
  - **Aetiology:** The initial response to any damage to the epidermis (scratching/irritants/Etc.)
  - **Seen in:** Eczema, Pempigus, Seborrheic dermatitis.



SKIN PATHOLOGIES:

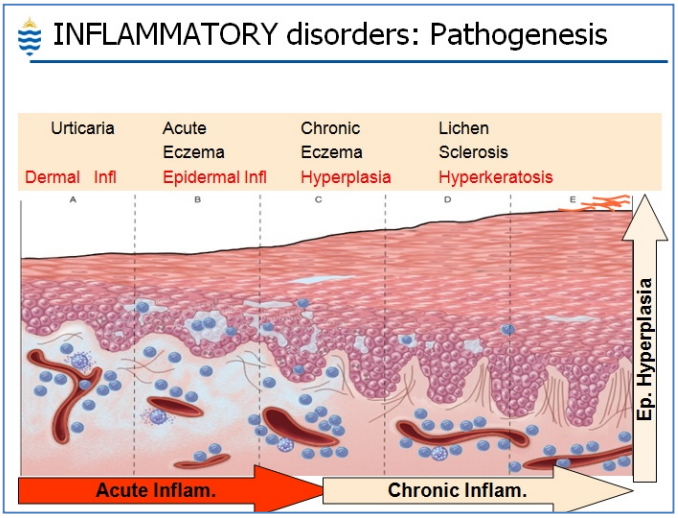
Principles of Pathology – Things to know for each of the presented conditions:

- **1. (Aetiology) What Caused the Disease?:**
- **2. (Pathogenesis) How does it Occur/Progress?**
- **3. (Morphology) What are the Gross/Microscopic Changes?**
  - List 3x Gross Features
  - List 3x Microscopic Features
- **4. Clinical Significance (Clinical Features, Complications, Prognosis, etc):**
  - List 3x Clinical Features

Skin Pathologies – Conditions To Know:

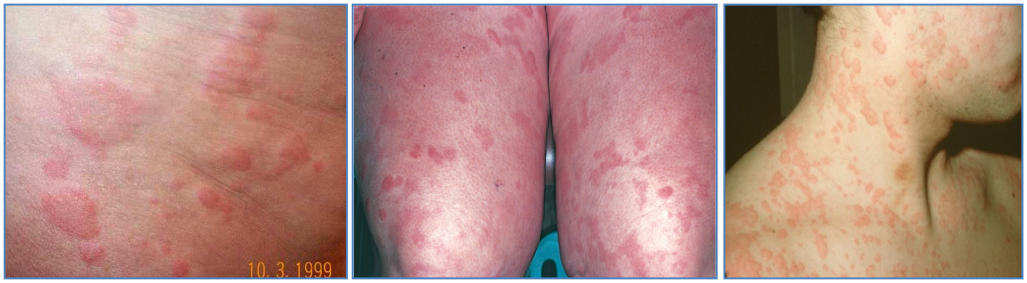
1. **Acute Inflammations:**
   - Urticaria,
   - Acute Eczema,
   - Erythema Multiforme.
2. **Chronic Inflammations:**
   - Psoriasis,
   - Chronic Eczema,
   - Lichen planus.
3. **Infections:**
   - Bacterial (Impetigo),
   - Fungal(tinea) &
   - Viral(warts).
4. **Blistering Diseases:**
   - Pemphigus,
   - Pemphigoid,
   - Dermatitis herpetiformis.
5. **Neoplastic:**
   - **Benign:**
     - Nevi,
     - Actinic Keratosis,
     - Seborrheic Keratosis.
   - **Malignant:**
     - BCC
     - SCC
     - Melanoma

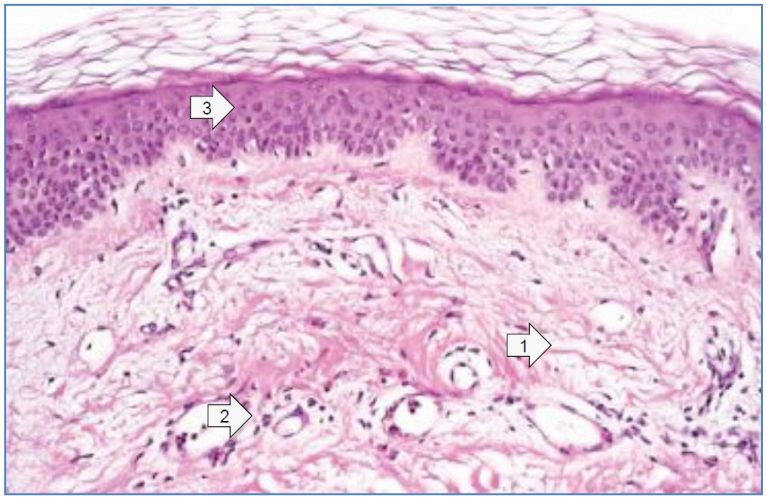
ACUTE INFLAMMATIONS:



Urticaria (Hives):

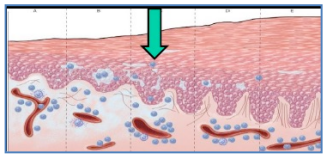
- **Aetiology:**
  - Type I hypersensitivity – Allergy (Food/drug/plant/etc)
- **Pathogenesis:**
  - Antigen is Re-Exposed to a sensitized Mast-Cell/Basophil → IgE-Bound Mast Cell *Degranulates*:
    - → Releasing Inflammatory Mediators (Histamine) of Type-1-Hypersensitivity Reactions.
    - →Perivascular inflammatory infiltrate: lymphocytes, neutrophils or eosinophils.
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - **Gross:**
    - Erythematous Papules & Plaques (Reddish, raised areas).
  - **Microscopic:**
    - 1. Dermal Oedema (Not Spongiosis – which is *Epidermal* Oedema)
    - 2. Dilated Dermal Blood Vessels (With Perivascular Inflammatory Cells)
    - 3. Dermal Inflammatory Infiltrate (Lymphocytes, Neutrophils, Eosinophils)
    - (Normal Epidermis – No Spongiosis or Hyperplasia)
- **Clinical Significance (List 3x Clinical Features):**
  - Usually on trunk and extremities.
  - Individual lesions are transient, usually resolve in 24 hr, but entire episode may last for days.
  - All ages, more in 20 – 40y.



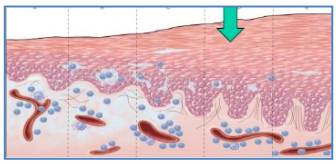


Acute Eczema:

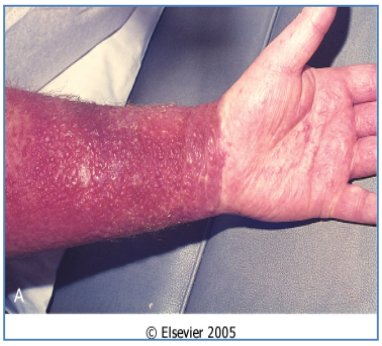
- **Types of Acute Eczema:**
  - Contact dermatitis (Due to prolonged exposure to allergen)
  - Atopic dermatitis
  - (Drug eczema)
  - (Photoeczema)
  - (Primary irritant dermatitis)
- **Aetiology:**
  - Type IV Hypersensitivity (T-Cell Mediated) to Allergen.
- **Pathogenesis:**
  - **Initial Exposure:**
    - Ag Processed by Langerhans Cells →Presented to T-Cells in LN → T-Cell Activation
  - **Upon Re-Exposure:**
    - Memory T cells produce Quick & strong inflammatory response.
    - →Epidermal Oedema + Forms small blisters → Wet Eczema

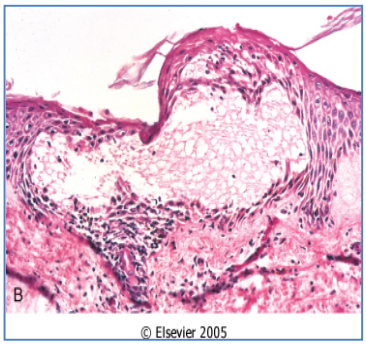


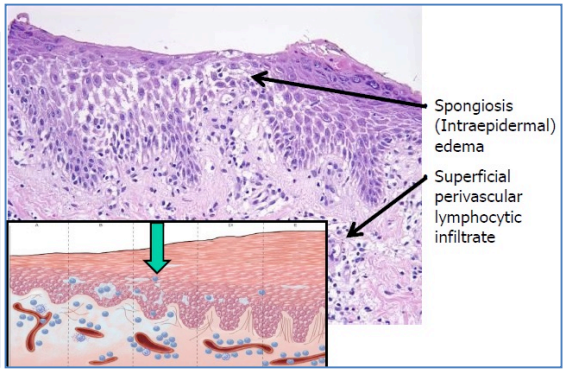
  - **Chronic Exposure:**
    - →Hyperplasia, hyperkeratosis (lichenification) – Dry Eczema.



- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - **Gross:**
    - Small Blisters
    - Erythema
    - Hyperkeratosis (If Chronic)
  - **Microscopic:**
    - Intra-*Epidermal* Oedema (*Spongiosis*)
    - Epidermal Blistering
    - Perivascular Inflammatory Infiltrate
- **Clinical Significance (List 3x Clinical Features):**
  - Often has a regular shape (Eg. Square from bandaid)
  - Treated with Corticosteroids (Smptomatic)
  - Antihistamies (For Itch)

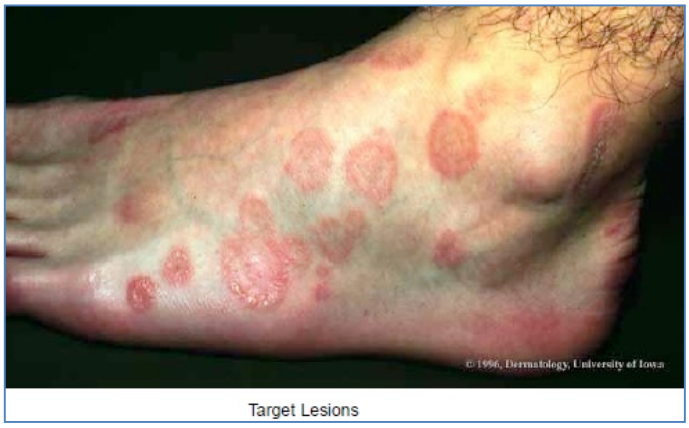




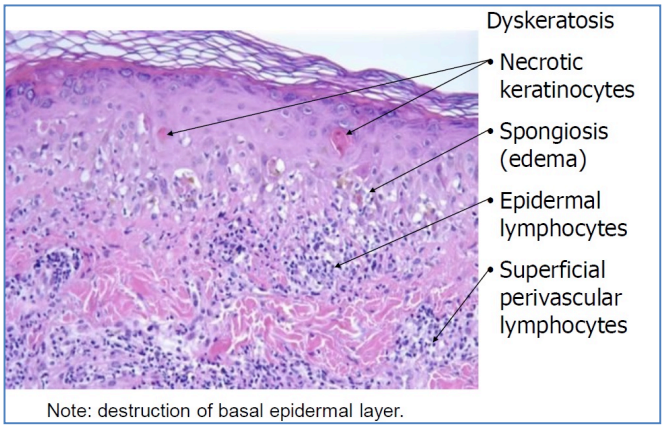


Erythema Multiforme:

- **Aetiology:**
  - Hypersensitivity
- **Pathogenesis:**
  - Hypersensitivity Response to:
    - Infections (Herpes simplex, *Mycoplasma*)
    - Drugs (Sulfonamides, penicillin, barbiturates)
    - Malignancy (Carcinoma, lymphoma)
    - Auto-Immune (Eg. Lupus, SS, Dermatomyositis)
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - **Gross:**
    - Variable Lesions: Papules/Plaques/Nodules/Blisters/Ulcers
    - Characteristic "*Targetoid*" Lesions
      - Central Grey Necrosis
      - Erythematous raised border.
  - **Microscopic:**
    - Dyskeratosis
    - Necrotic Keratinocytes
    - Spongiosis (Epidermal Oedema)
    - Epidermal Lymphocytes
    - Destruction of Basal Epidermal Layer
- **Clinical Significance (List 3x Clinical Features):**

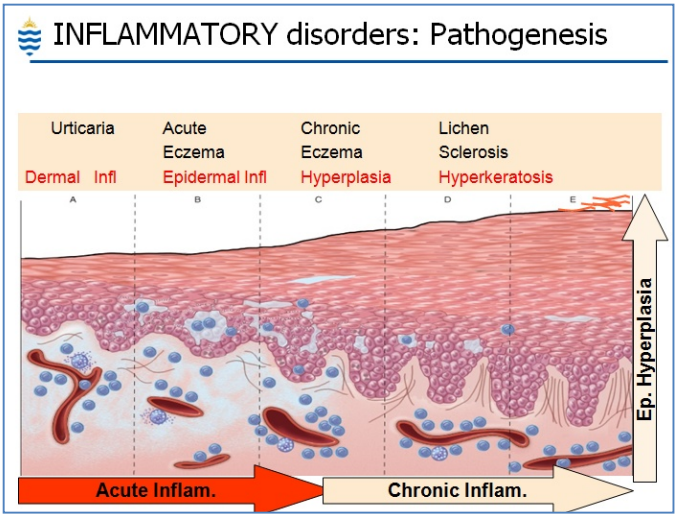


Target Lesions



Note: destruction of basal epidermal layer.

CHRONIC INFLAMMATIONS:

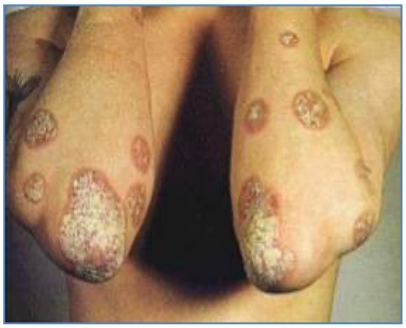
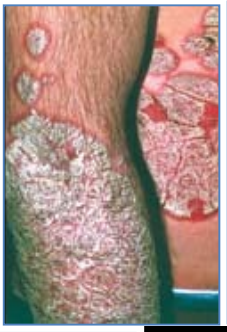
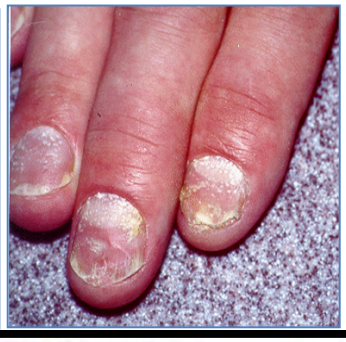
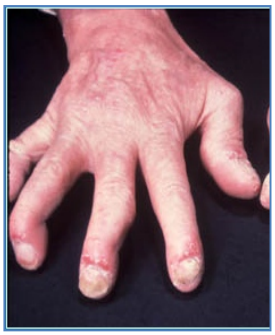
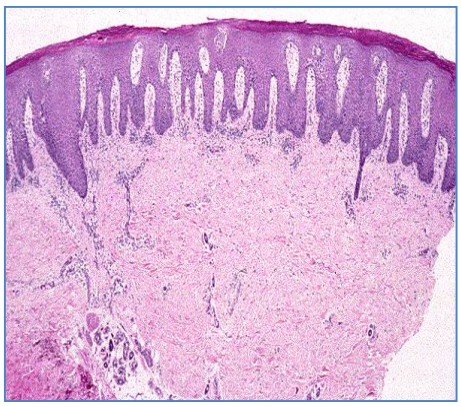
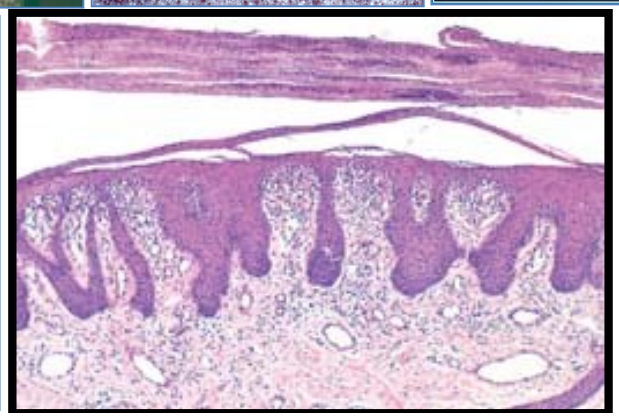


Chronic Eczema:

- **Aetiology:**
- **Pathogenesis:**
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - Dry Eczema
  - Hyperkeratosis
- **Clinical Significance (List 3x Clinical Features):**

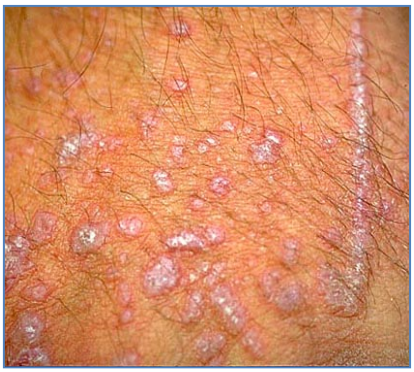
Psoriasis:

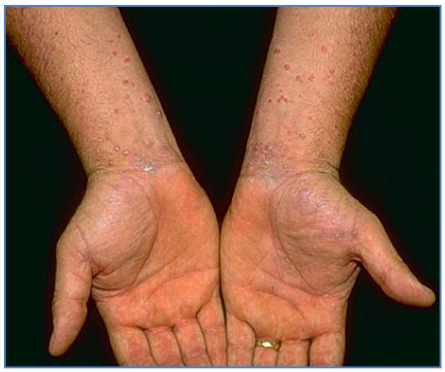
- **Aetiology:**
  - Multifactorial (Genetic & Immune)
- **Pathogenesis:**
  - Sensitized T cells infiltrate the skin and secrete cytokines and growth factors
    - →Continuous stimulation of basal cells → Increased cell turnover
    - + →Inflammation, Vascular Proliferation Angiogenesis
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - **Gross:**
    - Plaque covered with Silvery Scales (Due to *Hyperkeratosis* & *Parakeratosis*)
      - Bilateral
      - Well-Demarcated
      - Erythematous Based
  - **Microscopic:**
    - Acanthosis (Deepening of Rete Ridges & Thickening of the Epidermis)
    - Parakeratosis (Stratum Corneum still has Nuclei)
    - Neutrophilic Microabsceses in the Epidermis
    - Tortuous Papillar Dermal Vessels
- **Clinical Significance (List 3x Clinical Features):**
  - Can cause Multi-System Disorder:
    - Arthritis
    - Myopathy
    - Enteropathy
    - Immunodeficiency
  - Nail Pitting
  - ***Auspitz Sign***:
    - Microbleeding when crusts are removed.

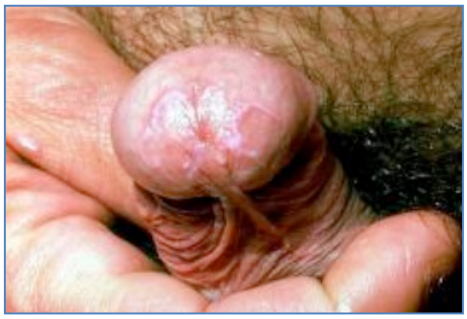







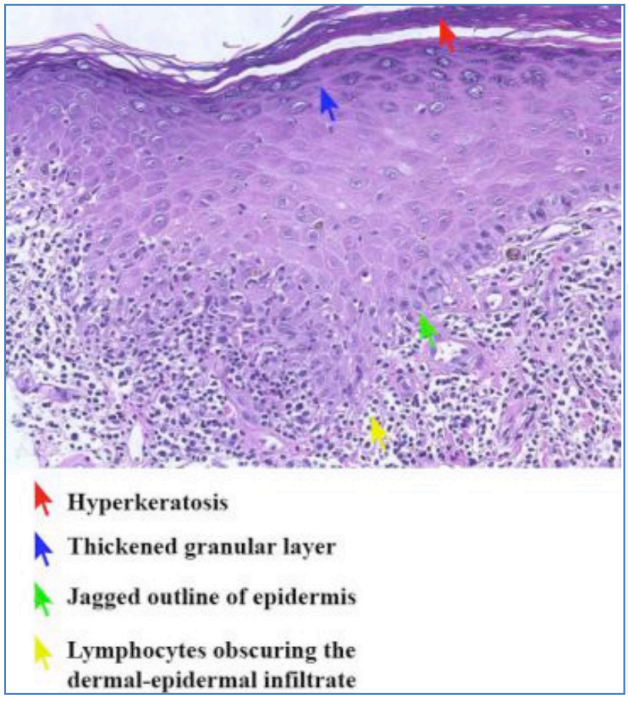
Lichen Planus:

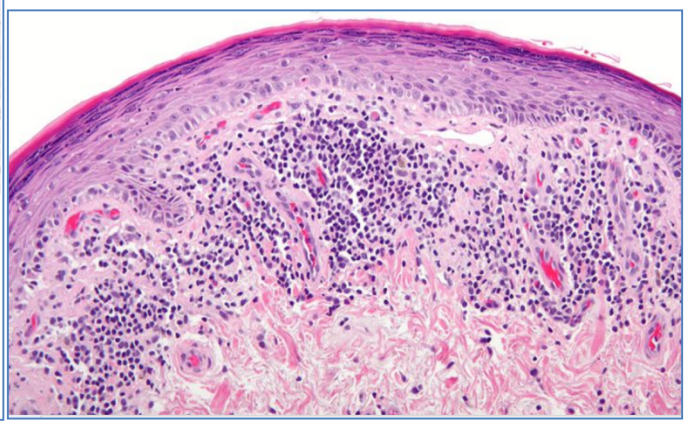
- **Aetiology:**
  - Unknown (not pathogenic or infectious)
- **Pathogenesis:**
  - Similar to Erythema Multiforme (Hypersensitivity), but more chronic.
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - **Gross:**
    - Purple, polygonal, planar, Papules & Plaques.
  - **Microscopic:**
    - Anucleate Dead Cells in Basal Layer ("Civatte Bodies")
    - Hyperplasia
    - Hyperkeratosis (Scaling)
- **Clinical Significance (List 3x Clinical Features):**
  - Itchy, Purple Rash
  - on Skin (commonly wrists & ankles), Mucosa, Genitals, Oral.
  - Self-limiting (1-2 yrs)







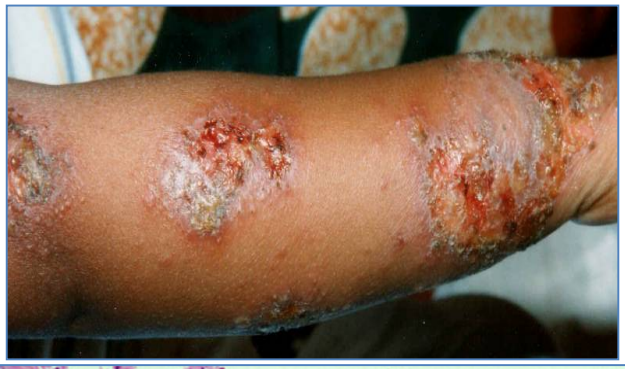


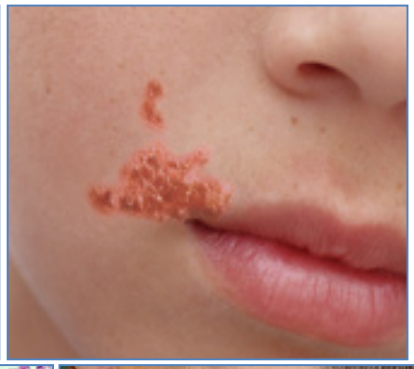


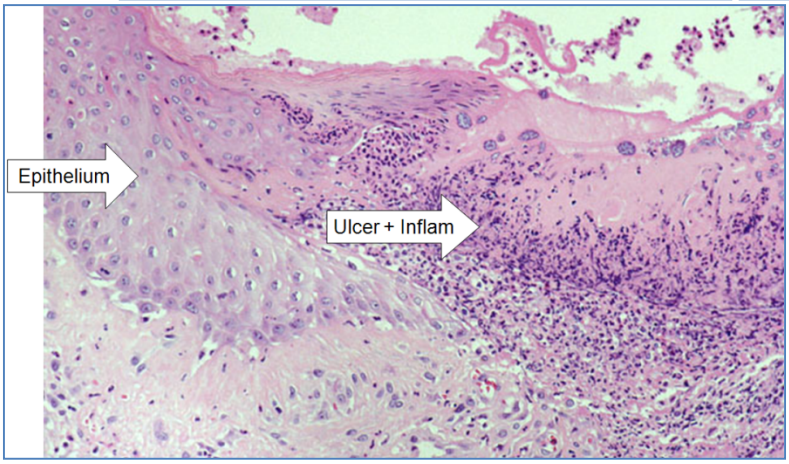
INFECTIONS:

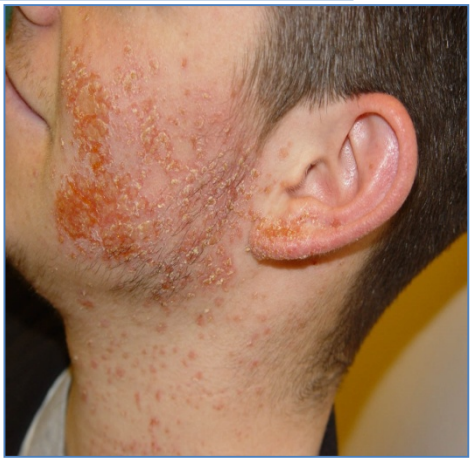
Bacterial (Impetigo)[School Sores]:

- **Aetiology:**
  - Overgrowth of Bacteria (Normal/Abnormal)
    - Staph or Strep
    - Superficial, Bacterial
- **Pathogenesis:**
  - Overgrowth of Bacteria → Inflammation
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - **Gross:**
    - Oozing, Pustular & Crusting Lesions
  - **Microscopic:**
    - Spongiosis
    - Neutrophils
- **Clinical Significance (List 3x Clinical Features):**
  - School Sores
  - Contagious
  - Regardless of Age



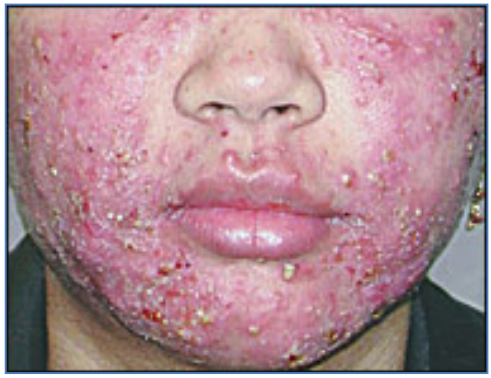


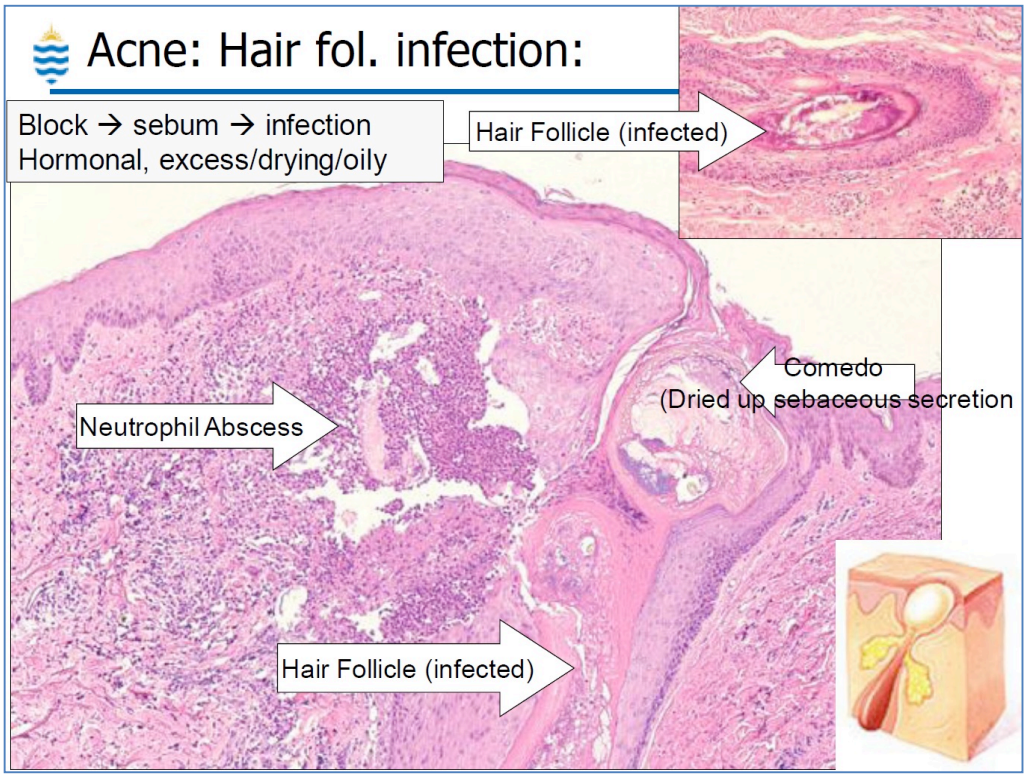




Bacterial (Acne):

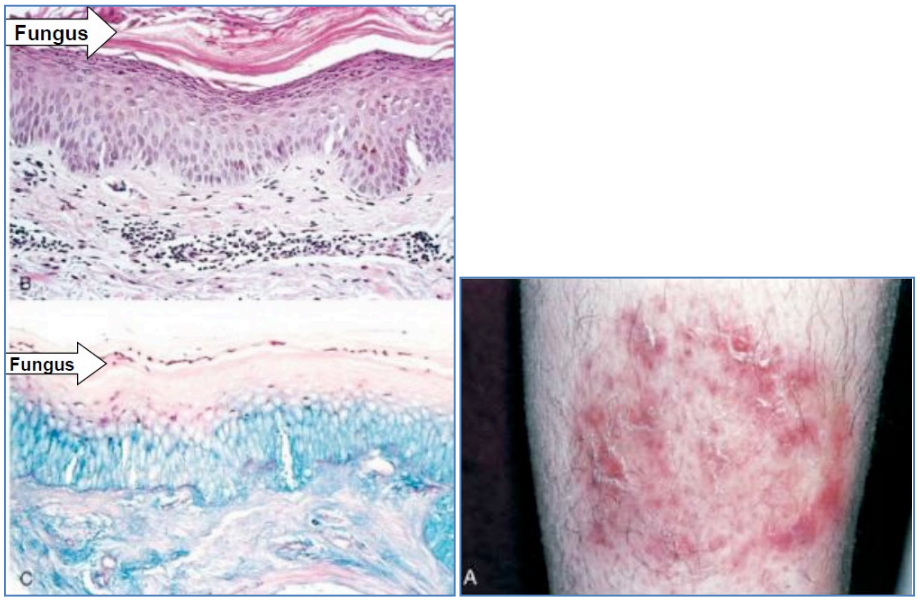
- **Aetiology:**
  - Bacterial Infection of Pilosebaceous Units
  - Hormonal influences
- **Pathogenesis:**
  - 4 Factors:
    - Excessive Sebum Production (Sebaceous Gland Hyperplasia)
    - Hyperkeratinisation
    - Comedones (Obstructed Pores)
    - Accumulation of Lipids & Cellular Debris
  - Inflammation is Enhanced by Follicular Rupture
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - **Gross:**
    - Comedones
    - Pustules
  - **Microscopic:**
    - Infected Hair Follicle
    - Neutrophil Abscess
    - Comedones
- **Clinical Significance (List 3x Clinical Features):**





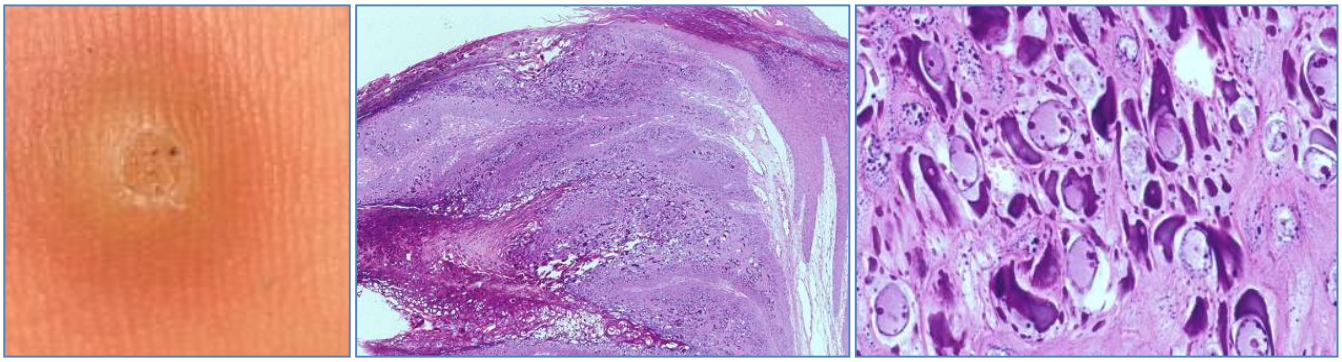
Fungal (Tinea):

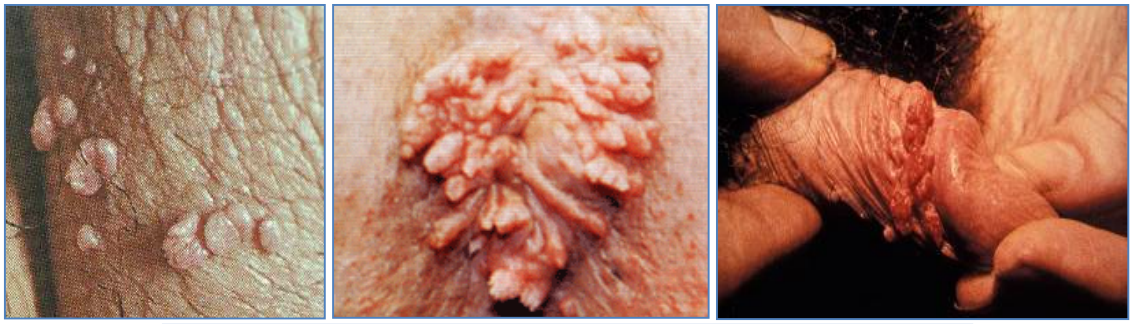
- **Aetiology:**
  - Fungal Infection
- **Pathogenesis:**
  - *Superficial* Fungal Infection → Inflammatory Rash → Dermatitis
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - **Gross:**
    - Rash:
      - Round
      - Scaly
      - Itchy
    - Spreading out with Central Clearing
  - **Microscopic:**
- **Clinical Significance (List 3x Clinical Features):**



Viral (Warts):

- **Aetiology:**
  - Human Papillomavirus (HPV)
  - Herpes (Zoster/Shingles)
  - DNA Pox Virus (→Molluscum Contagiosum)
- **Pathogenesis:**
  - HPV → Cellular Mutation → Hyper/Neo-Plasia
  - → Wart Formation (Mostly Benign)
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - **Gross:**
    - Verrucous Warts
    - Intradermal Hard Cysts
  - **Microscopic:**
    - Inward Growth Deep into the Skin
    - Koilocytic Keratinocytes (Dysplastic Squamous cells)
    - Central Blood Vessels
- **Clinical Significance (List 3x Clinical Features):**
  - Contagious
  - Central blood vessels → Bleed profusely when the surface is broken.

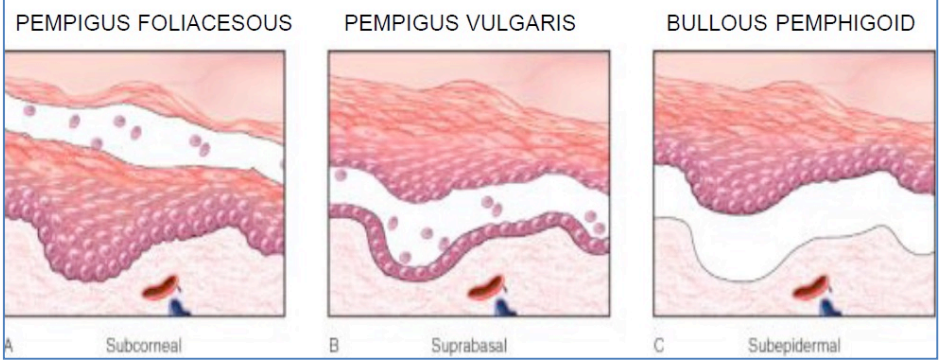




BLISTERING DISEASES:

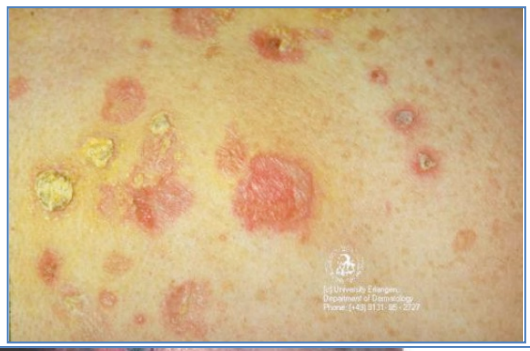
3 Types:

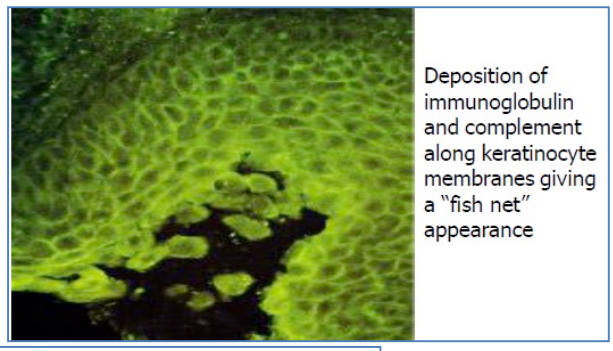
- Subcornial. (Just below the corneum)
- Suprabasal. (just above basal layer)
- Subepidermal.(just below the epidermis)



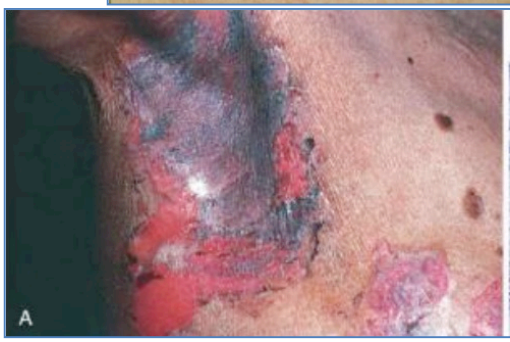
Pemphigus (Pemphigus Vulgaris):

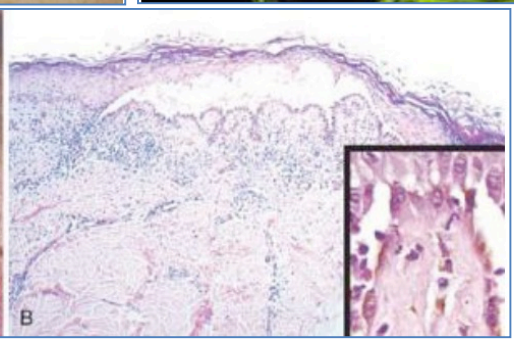
- **Aetiology:**
  - Autoimmune
- **Pathogenesis:**
  - Antibodies against Desmosomes → Destroys Desmosomes → Acantholysis (Intraepidermal Blisters)
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - **Gross:**
    - Thin, Flaccid Blisters (Easily Popped)
    - Look similar to burns
    - Many have popped → Crusting
  - **Microscopic:**
    - Acantholysis (***Intraepidermal*** Bullae)
    - Loose cells inside the Bullae
- **Clinical Significance (List 3x Clinical Features):**



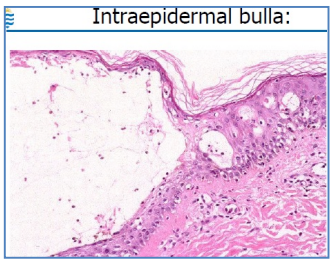


Deposition of immunoglobulin and complement along keratinocyte membranes giving a "fish net" appearance



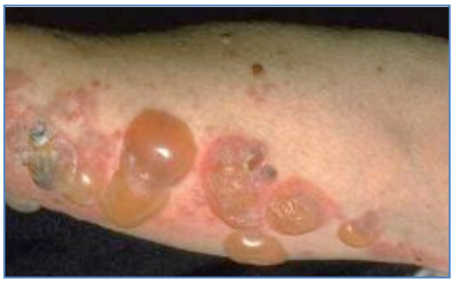
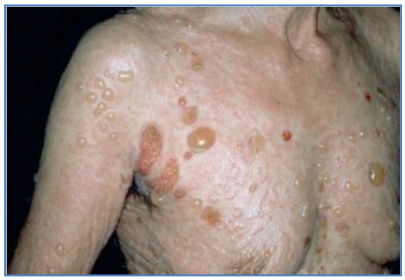
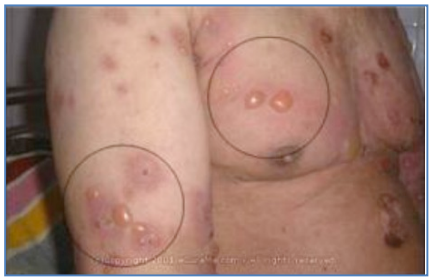
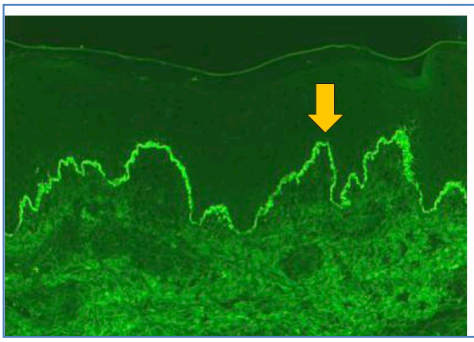


Intraepidermal bulla:

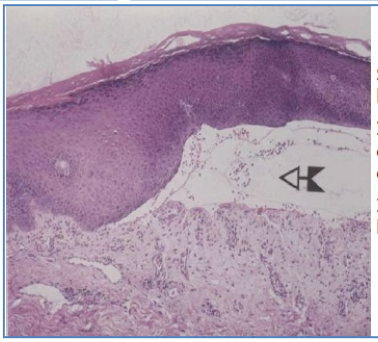


Bullous Pemphigoid:

- **Aetiology:**
  - Autoimmune
- **Pathogenesis:**
  - Antibody against Basal Layer → Destroys Sub-Epidermal Anchoring Proteins.
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - **Gross:**
    - Large, Tense, ***Subepidermal*** Bullae
    - Sometimes Haemorrhagic Blisters
  - **Microscopic:**
    - NO Acantholysis
- **Clinical Significance (List 3x Clinical Features):**

Antibody against the basal layer →bullous pemphigoid antigen in basement membrane causing subepidermal separation Type II hypersensitivity reaction.



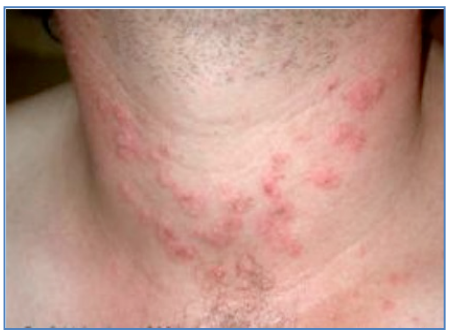
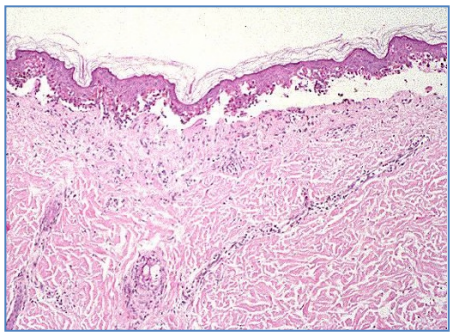
Subepidermal blister

Inflammation characterized by eosinophils

Intact acanthocytic layer.

Dermatitis herpetiformis.

- **Aetiology:**
  - Associated with Coeliac Disease (Gluten Sensitivity)
- **Pathogenesis:**
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - **Gross:**
    - VERY Itchy, Small, Erythematous Papules
    - Vesicles
    - Occasional Bullae
  - **Microscopic:**
    - Subepithelial, Neutrophilic Microabscesses in Dermal Papila
- **Clinical Significance (List 3x Clinical Features):**
  - Associated with Coeliac Disease

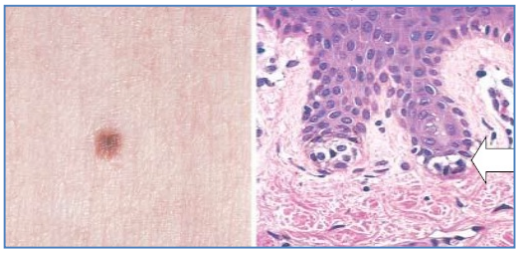



	PEMPHIGUS	PEMPHIGOID	Dermatitis Herpetiformis
age	mid - older	elderly	30-40
antibody	IgG	IgG	IgA
location	Suprabasilar	Subepidermal	subepidermal
inflammation	mixed	eosinophils	neutrophils
Site of dysfunction	Desmosomes	Basement membrane and hemidesmosomes	Anchoring fibrils
Antibody against:	Desmoglein	Bullous pemphigoid antigen	reticulin
Immuno	"fish net"	Linear basement membrane	Dermal tip

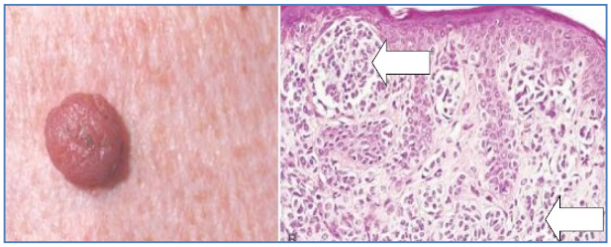
NEOPLASTIC:

BENIGN:

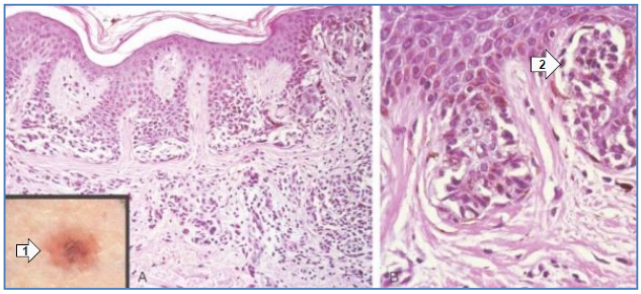
- **Nevi:**
  - **Aetiology:**
    - Congenital (After Birth)
  - **Pathogenesis:**
    - **Freckles =** Excess Pigment
    - **Moles (Lentigo) =** Excess Melanocytes
    - **Junctional Naevus (Typical Birthmark) =** Excess Melanocytes *AND* Excess Pigment
      1. Small, Flat, Symmetric, Uniform Lesions
      2. Cluster of Clear Cells (Melanocytes) @ the Dermo-Epidermal Junction
      3. NB: Proliferation below Basal Layer.

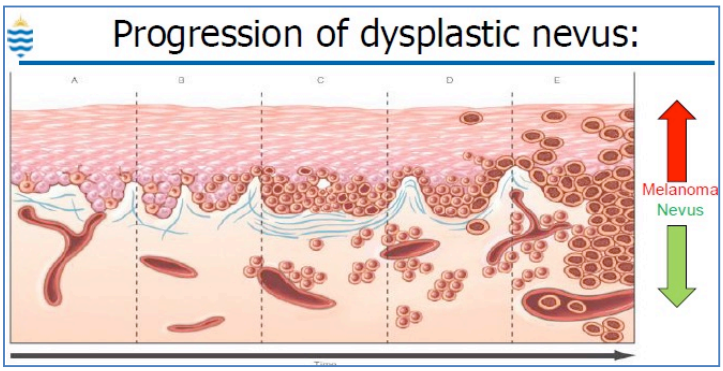


    - **Compound Nevus:**
      1. Small, Raised, Dome-Shaped, Symmetric, Uniform Lesions.
      2. Big Clusters of Melanocytes in Dermis & Dero-Epidermal Junction.

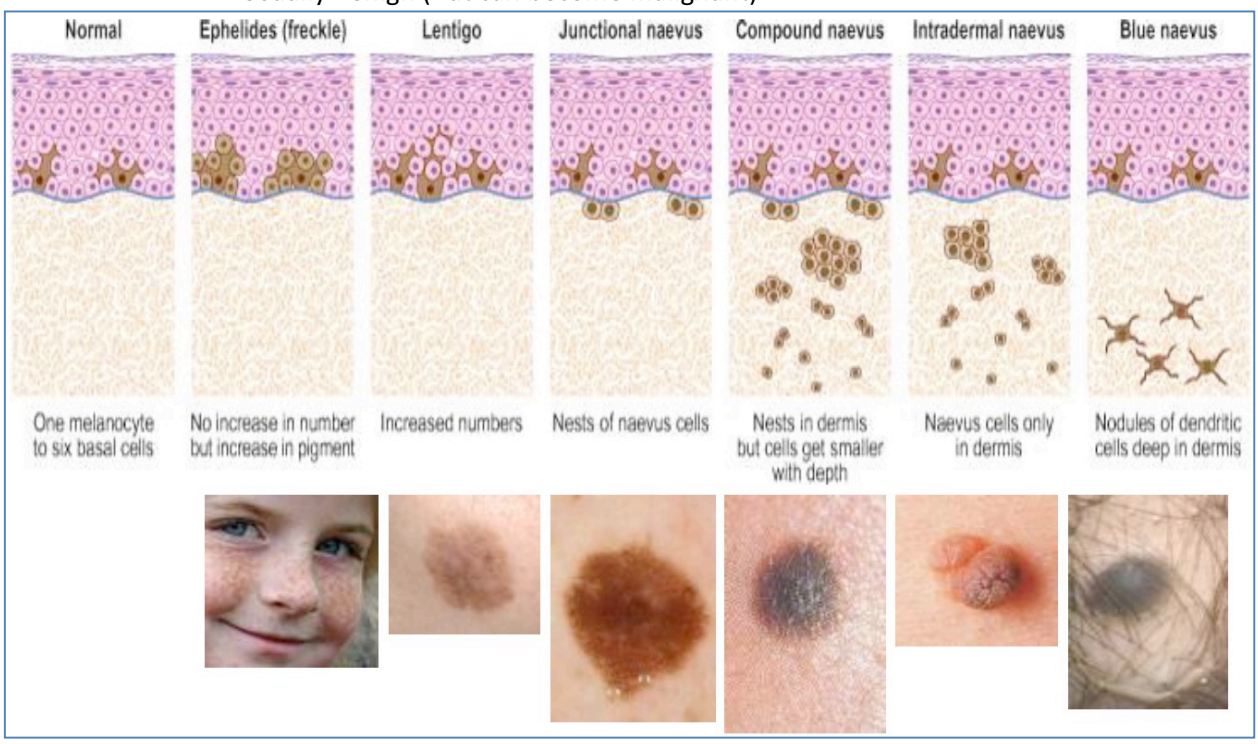


    - **Dysplastic Nevus:**
      1. Pigmented, Raised Lesion, with Central Darker Shade.
      2. DE-Junctional Cluster of Dysplastic (Larger/Irregular/Darker) Melanocytes.
      3. **NB: Can Progress to Melanoma:**
         1. Once the Dysplastic Melanocytes begin Infiltrating UP into the Epidermis.

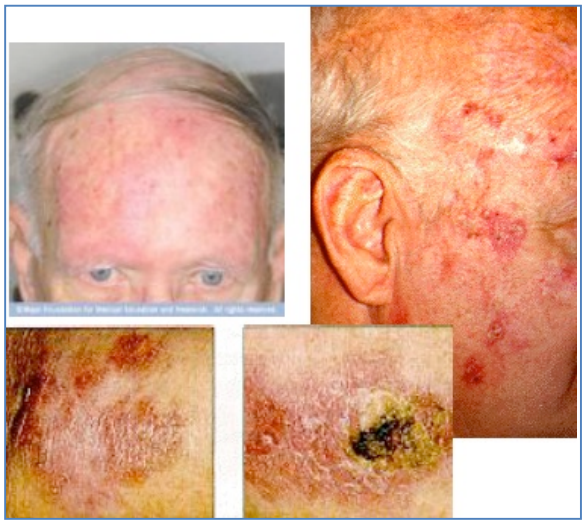


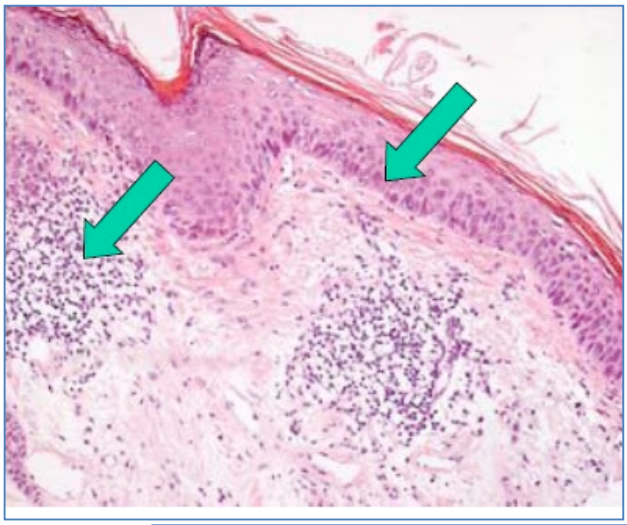


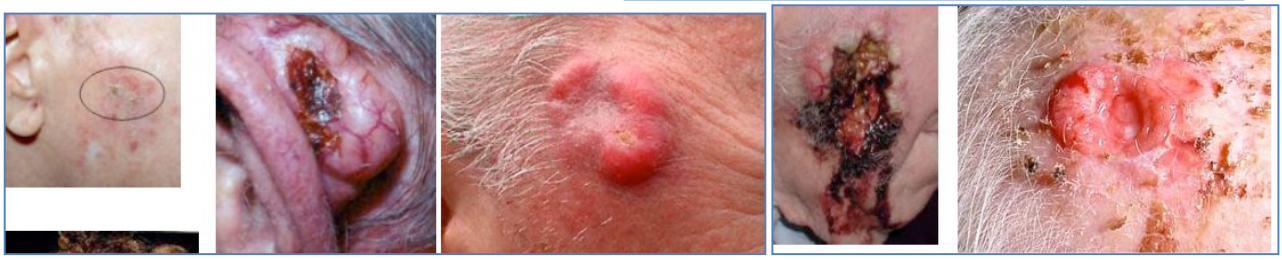
- **Clinical Significance (List 3x Clinical Features):**
    - Usually Benign (But can become malignant)



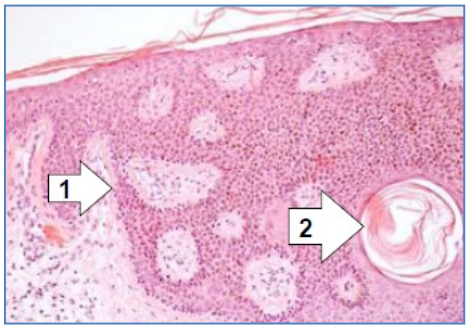
- **Actinic Keratosis (Sun Damage):**
  - **Aetiology:**
    - Sun Damage
  - **Pathogenesis:**
    - Sun Damage due to Chronic Exposure to UV Radiation
  - **Morphology (List 3x Gross & 3x Microscopic Features):**
    - **Gross:**
      1. Red/Tan, Irregular, Scaly Plaques
      2. Hyperkeratosis
      3. Inflammation/Ulceration/Crusting
    - **Microscopic:**
      1. Dysplasia (Pre-Cancerous) & Atrophy
      2. Inflammation
      3. Hyperkeratosis *AND* Parakeratosis
      4. Loss of Papillary Dermis & Rete Ridges
  - **Clinical Significance (List 3x Clinical Features):**
    - A Pre-Cancerous Skin-Growth



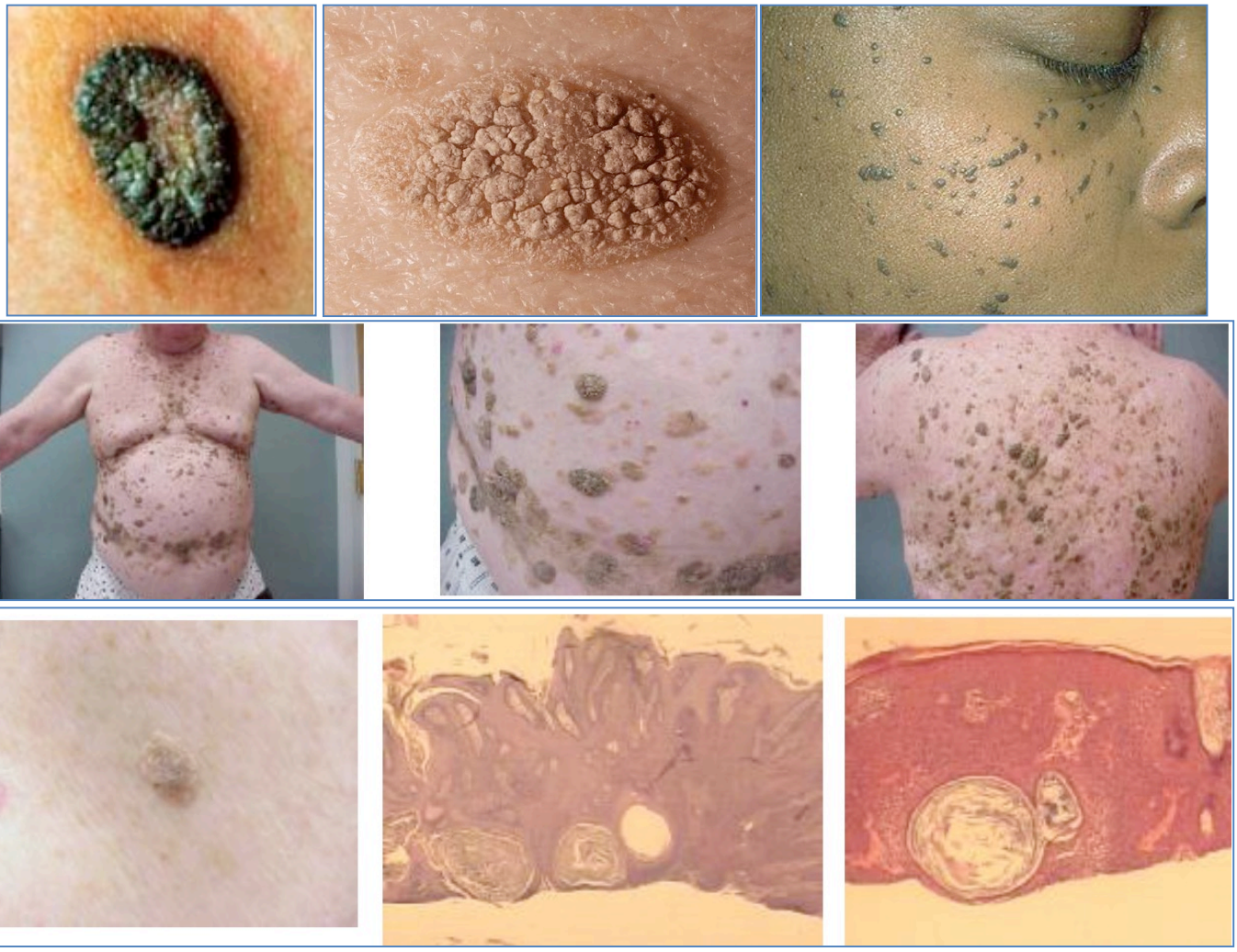




- **Seborrheic Keratosis:**
  - **Aetiology:**
    - Totally Benign Tumour in Old Age
  - **Pathogenesis:**
    - Unknown
  - **Morphology (List 3x Gross & 3x Microscopic Features):**
    - **Gross:**
      1. Sticky, Oily Plaques
      2. Round, Flat, Velvety Plaques
      3. May be Pigmented
    - **Microscopic:**
      1. Thick Hyperplastic Epidermis
      2. Keratin Cysts

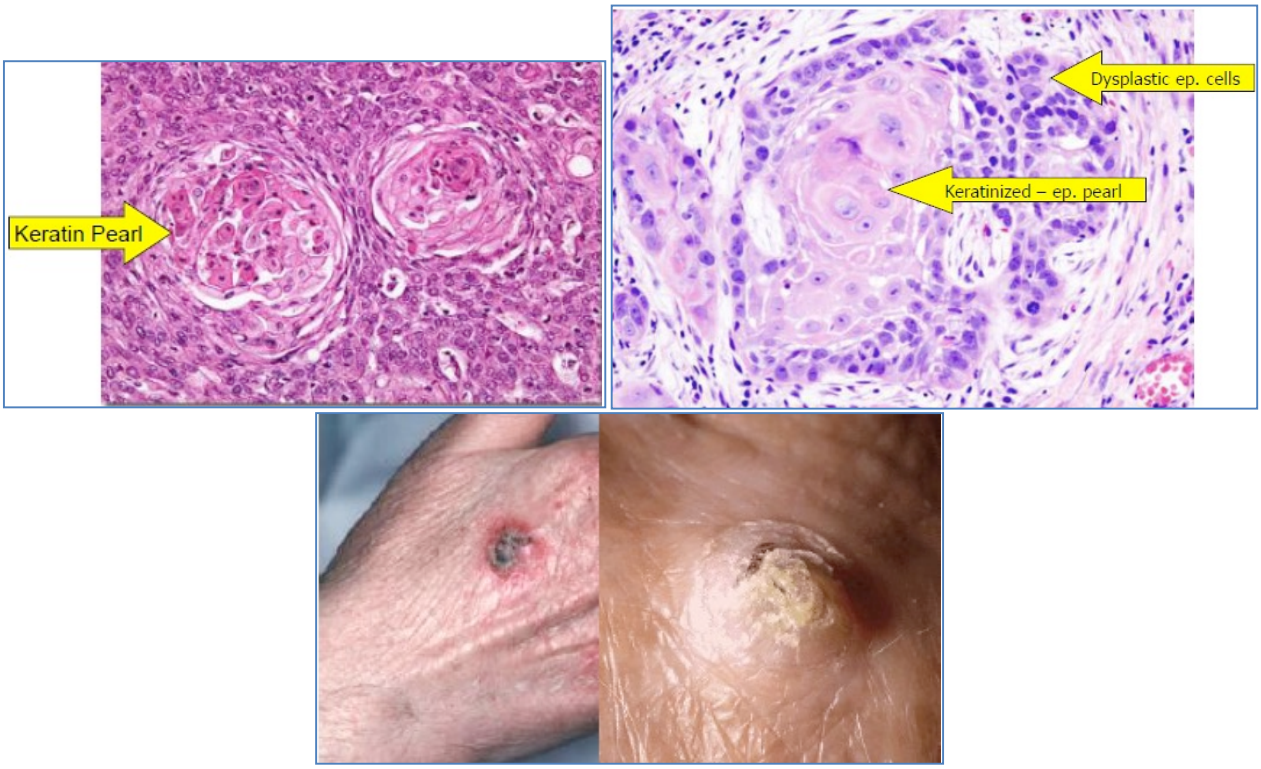


  - **Clinical Significance (List 3x Clinical Features):**
    - Treated only if inflamed
    - No Malignant Potential

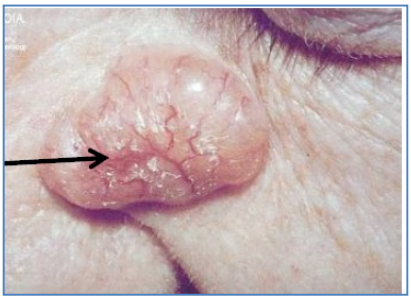
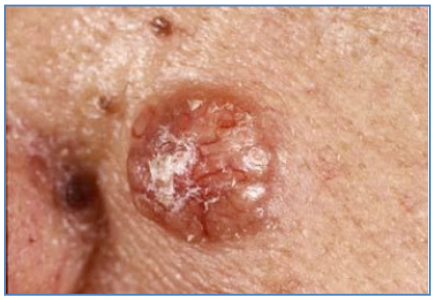
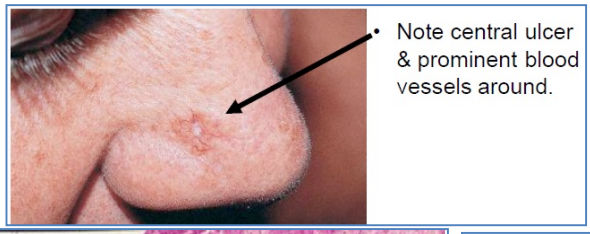


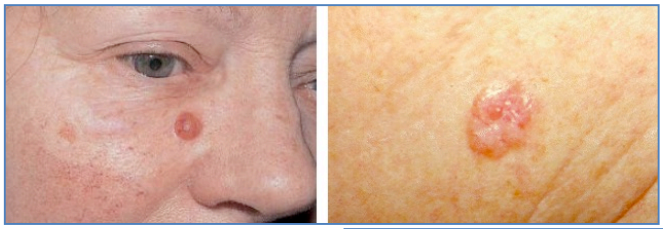
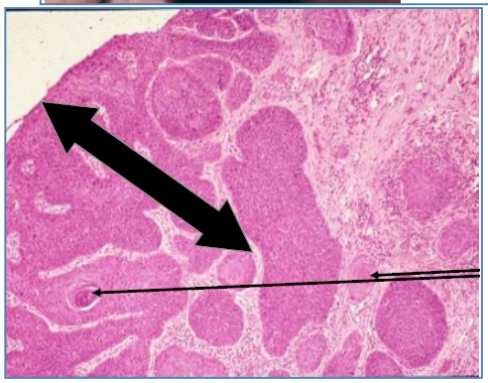
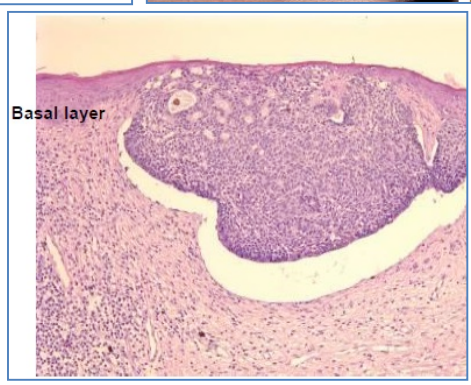
MALIGNANT:

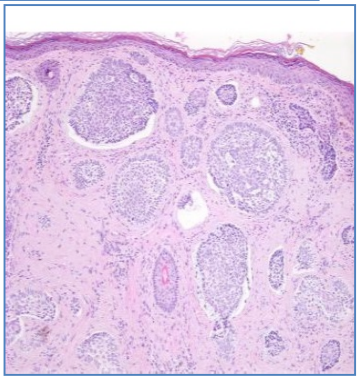
- **Squamous Cell Carcinoma (SCC):**
  - **Aetiology:**
    - Sun Damage
    - Industrial Carcinogens
    - Tobacco
  - **Pathogenesis:**
    - Dysplasia & Tumorigenesis of Squamous Epithelial Cells.
  - **Morphology (List 3x Gross & 3x Microscopic Features):**
    - **Gross:**
      1. Nodule
      2. Hyperkeratosis
      3. Sometimes Erythematous base.
    - **Microscopy:**
      1. Dyskeratosis
      2. Epithelial Keratin Pearls
  - **Clinical Significance (List 3x Clinical Features):**
    - Malignant (Early Treatment via Excision is Essential)



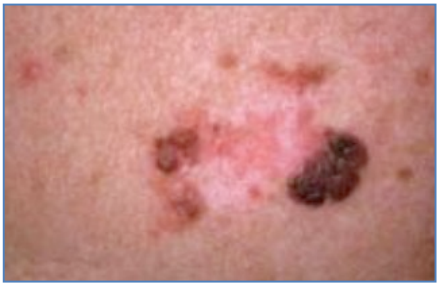
- **Basal Cell Carcinoma (BCC):**
  - **Aetiology:**
    - Sun Exposure
  - **Pathogenesis:**
    - Dysplasia & Tumorigenesis of Basal Epithelial Cells.
    - Slow Growing
    - Rarely Metastasises (Locally Infiltrative, but don't often Metastasise)
  - **Morphology (List 3x Gross & 3x Microscopic Features):**
    - **Gross:**
      1. Nodular/Papular
      2. Blood Vessels
      3. Pearly/Shiny crust over the lesion.
      4. Large Tumours may Ulcerate.
    - **Microscopy:**
      1. Basal Cell Proliferation (Infiltrates the Dermis)
      2. Malignant Squamous Cells extend deep, Penetrating the Basement Membrane.
      3. Presence of Squamous Eddies or "Epithelial Pearls"
  - **Clinical Significance (List 3x Clinical Features):**

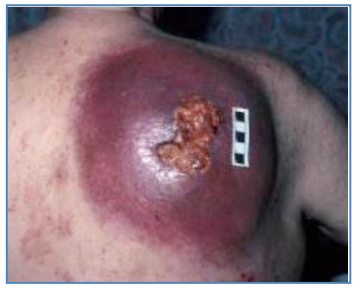




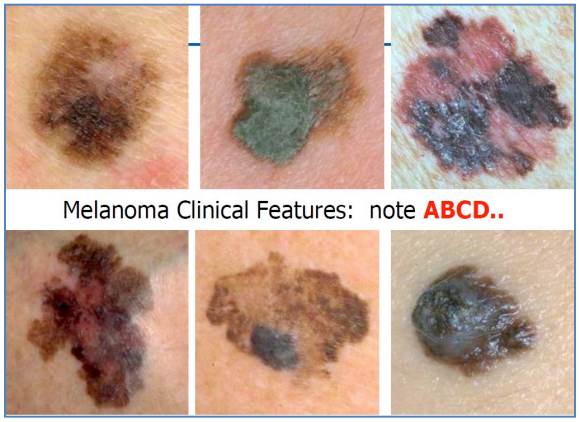




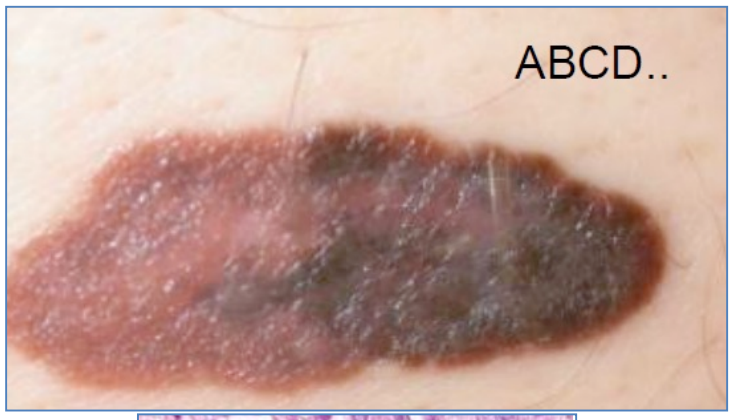


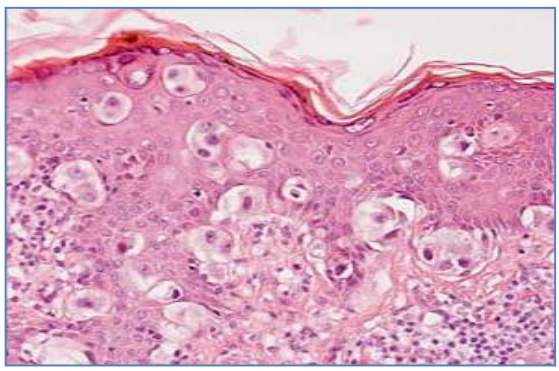
- **Melanoma:**
  - **Aetiology:**
    - Sun Damage
    - Congenital
  - **Pathogenesis:**
    - Tumorigenesis of Melanocytes
  - **Morphology (List 3x Gross & 3x Microscopic Features):**
    - **Gross:**
      1. Grow & Change Colour
      2. Maculo-Papular Lesion
      3. ***Irregular Shape, Colour & Depth – Distinguishing Features.***
      4. Pigmented
    - **Microscopic:**
      1. **Diagnostic Feature: Atypical (Dysplastic) Melanocytes *ABOVE The Epidermis***
         1. Also Invade down into the Dermis
      2. Nests/Clusters of Atypical Melanocytes
      3. Inflammatory Cells
      4. Mitotic Figures (Due to High Replication Rates)
  - **Clinical Significance (List 3x Clinical Features):**
    - Malignant

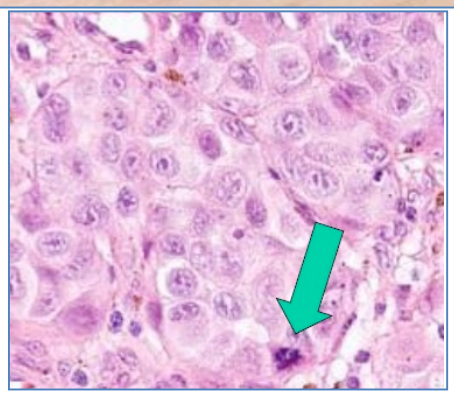






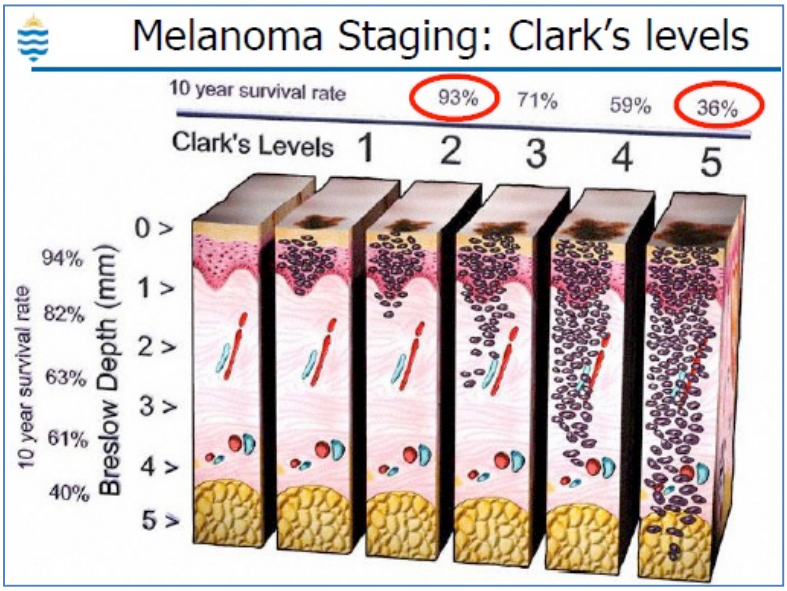






ABCD(E):

Asymmetry, Borders (Irregular), Colour (Varied), Diameter (Greater than 6mm), Evolving over time.

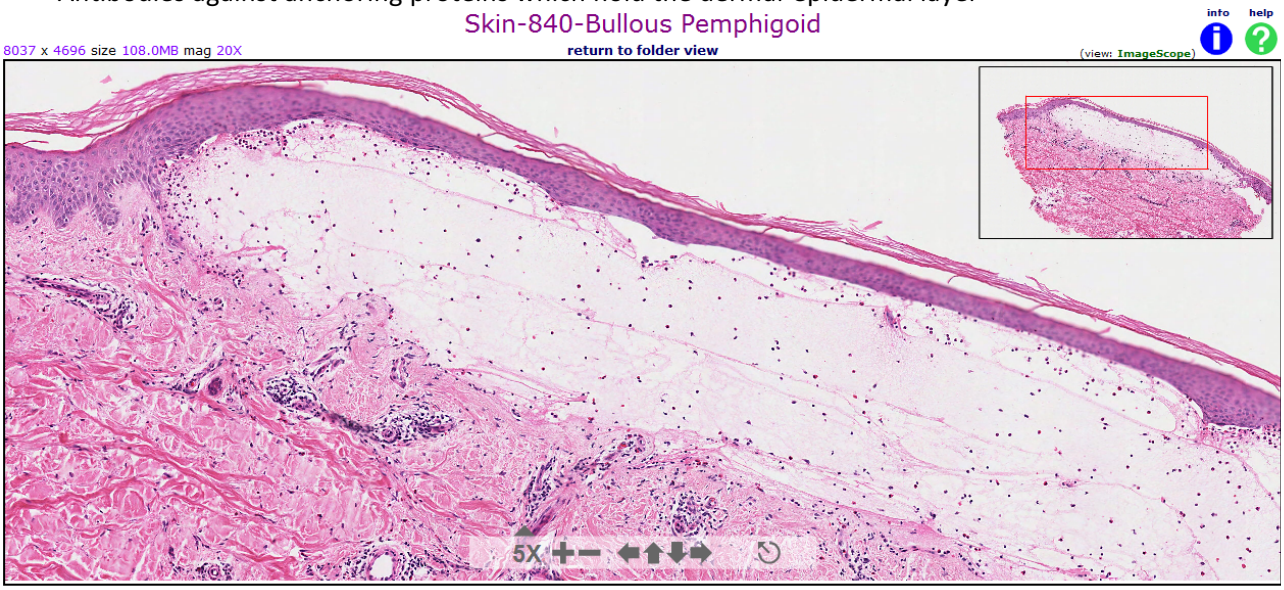


NB: Just realise that despite subtle changes in appearance, the *actual* size and infiltration of the Melanoma can be quite significant.

PATHOLOGY SLIDES:

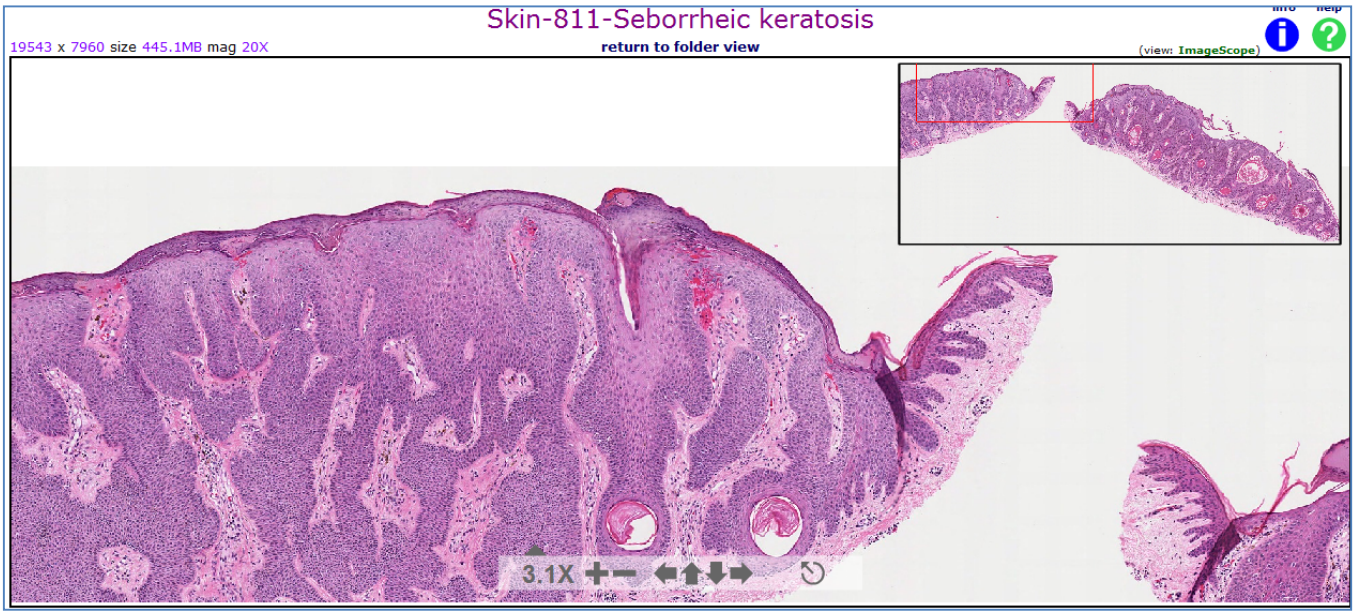
Bullous Pemphigoid:

- Tense Bullae
- Old Age Patients
- Separation of the whole epidermis from teh dermis
- Antibodies against anchoring proteins which hold the dermal-epidermal layer



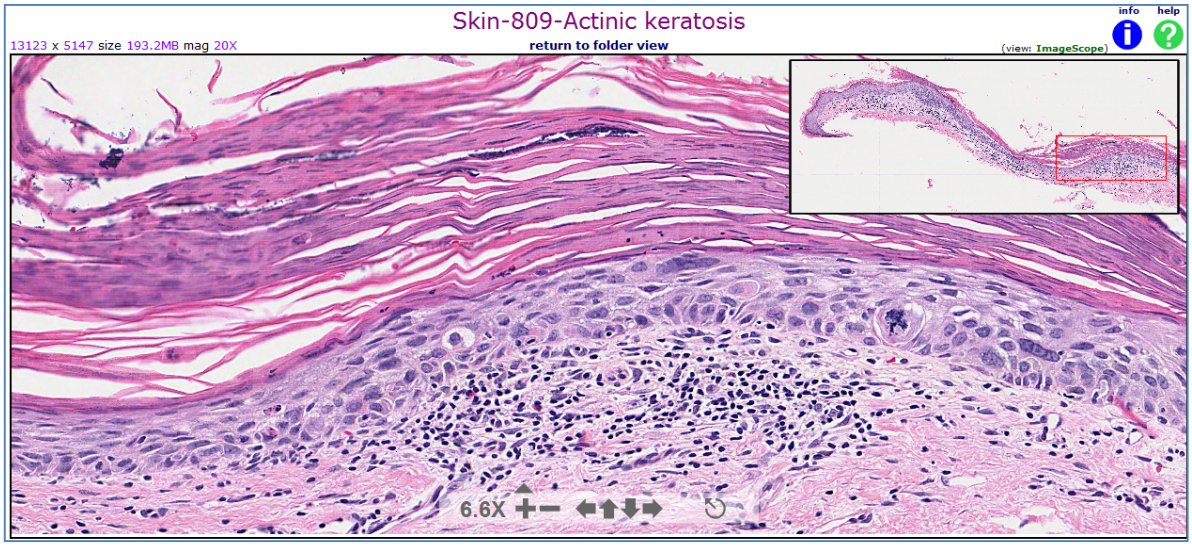
Seborrheic Keratosis:

- Similar to SCC, but is more regular (as it is benign)
- Grows above the skin



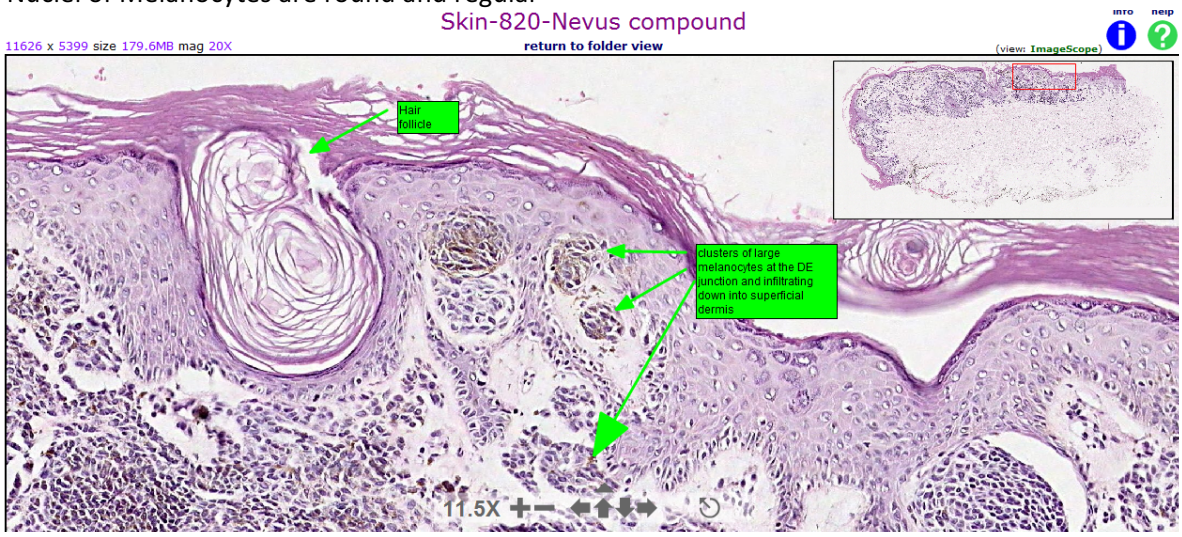
Actinic Keratosis (Sun Damage):

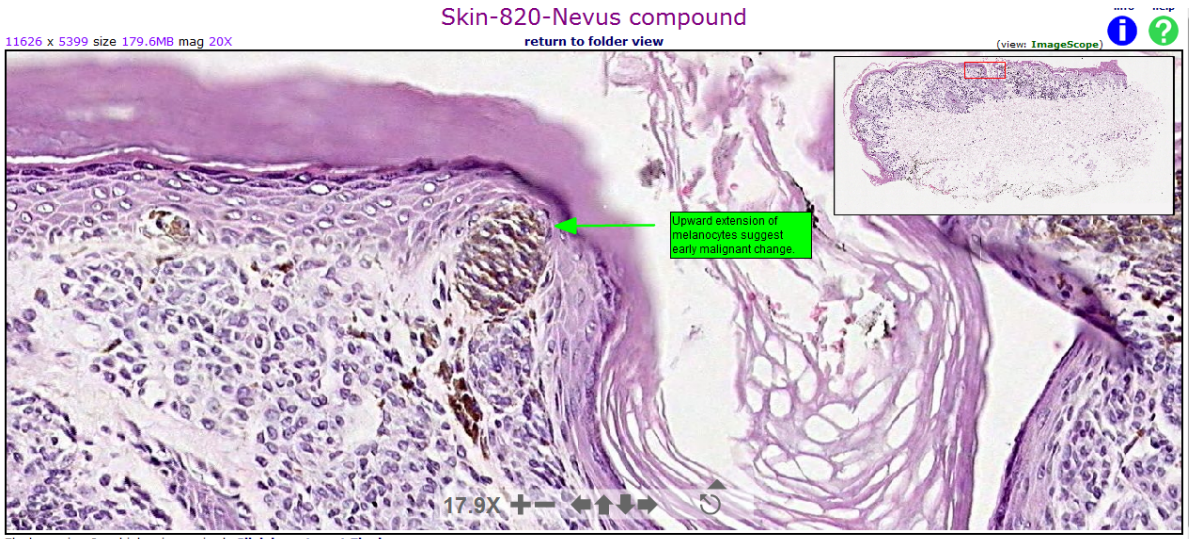
- Hyperkeratosis & Parakeratosis (Visible Nucleus)
- Atrophy of the Epithelium
- Very Large & Irregular Nuclei of Epidermal Cells.
- Thick & irregular collagen bundles



Compound Nevus (Junctional Nevus + Intradermal Nevus):

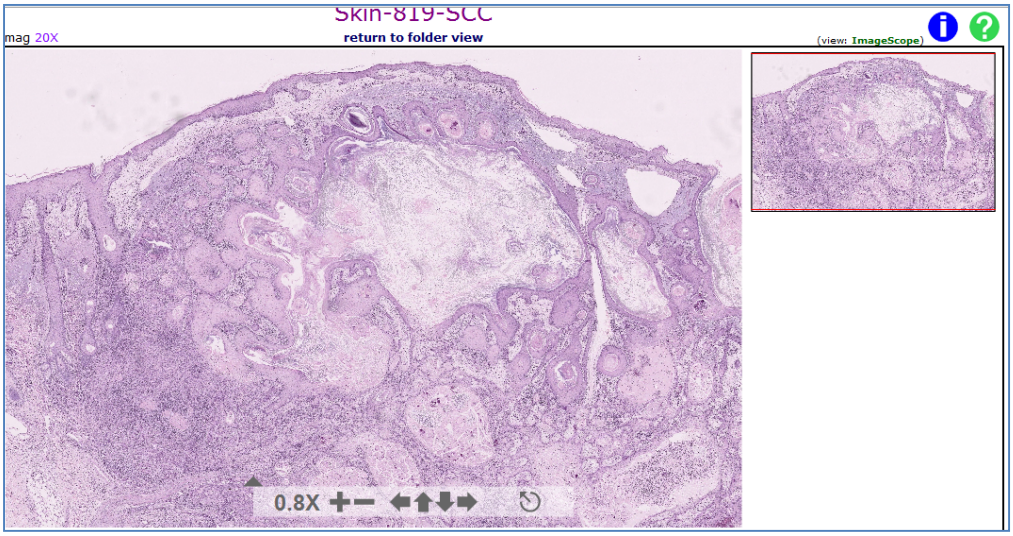
- Looks very similar to Melanoma (but the distinguishing feature is the location of the melanocytes – which are all maintained within the dermis.)
- Nuclei of Melanocytes are round and regular

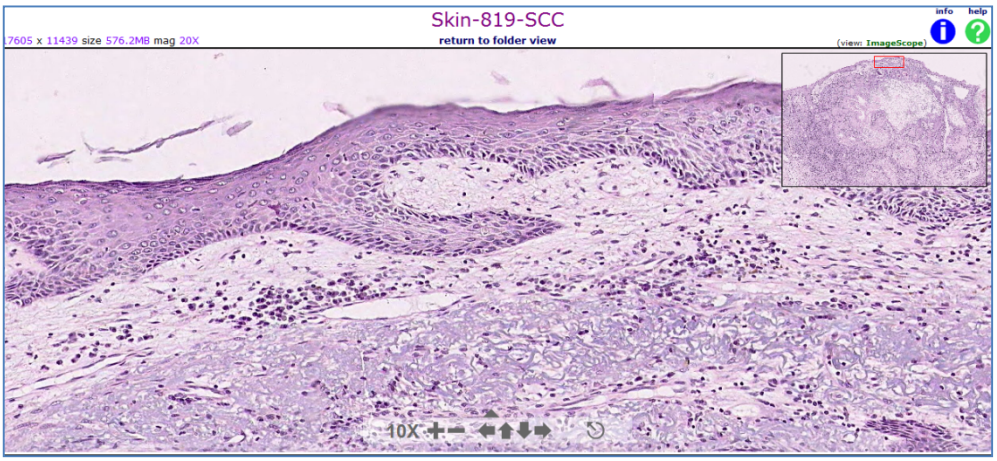




Squamous Cell Carcinoma:

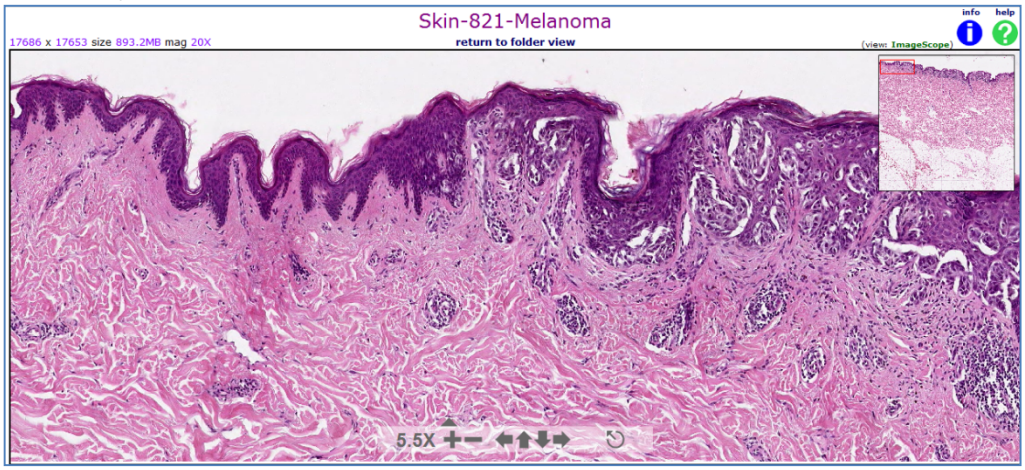
- Abnormal Clusters of Keratinisation within the tumour (below the level of the stratum corneum)
    - Central area of necrosis with just keratin plaques
- Degeneration of subdermal collagen
- Irregularity of the nuclei (Dysplasia)





Melanoma:

- NB: on the Mid-right aspect, notice the clusters of Melanocytes invading both down into the Dermis and up into the Epidermis. (Melanocytes are normally confined to the basement membrane)
- The Left-Mid aspect is normal skin.



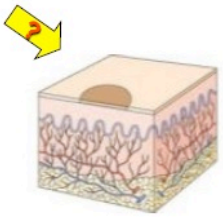
EXAMPLES OF EXAM QUESTIONS:

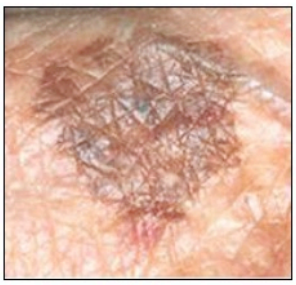
Dermatology Self Assessment Quiz:

1. **Lesions following viral fever & sore throat. Lesions have a central clearing (Target-eyed Lesinos), are red. Diagnosis?**
    a. **Erythema Multiforme**
2. **Itchy, lesion on wrist for 3 days following wearing new bracelet. Microscopy shows spongiosis (intraepidermal oedema) Diagnosis?**
    a. **Acute Eczema/Contact Dermatitis (Due to allergy against material in bracelet)**
    b. **(Not Urticaria – would present within hours, and doesn't have spongiosis)**
3. **84yr old, scalp lesions. (It is Actinic Keratosis) What is the diagnostic feature you would see under the microscope?**
    a. **Solar Elastosis (Very specific to solar damage)**
    b. **Also have Parakeratosis (but is also seen in other conditions)**
4. **41 yr old male, generalised very itchy rash, swollen lips. Diagnosis?**
    a. **Urticaria**
5. **What is the characteristic microscopic feature of Urticaria?**
    a. **Dermal Oedema (As opposed to Spongiosis, which occurs after 3-4 days) due to mast cell degranulation.**
6. **Rash 6 days after giving blood @ the site of the bandaid. Diagnosis?**
    a. **Contact Dermatitis**
7. **What is the microscopic characteristics of Acanthosis:**
    a. diffuse epidermal hyperplasia.[1] Acanthosis implies increased thickness of stratum spinosum
8. **82 yr male, lesions on hand and ear. Microscopy shows irregularity of cells, and the lesion is shiny, erythromatous plaques. Diagnosis?**
    a. **Squamous Cell carcinoma**
9. **48yr female, large flaccid blisters. Some broken with crusting. Diagnosis?**
    a. **Pemphigus (As opposed to Bullous Pemphigoid)**
10. **28yr male, asymptomaic lesions for 3 weeks on chest & back. Diagnosis?**
    a. **Tinea Versicolor**
11. **81yr male, surfing enthusiast, lesions on cheek or 3 years. Slowly increasing. Diagnosis?**
    a. **Actinic Keratosis (Sun damage)**
12. **21yr male, lesions on face since 4 weeks. Slowly increasing. Itchy, but no pain. Diagnosis?**
    a. **Impetigo (Staphylococcal)**
13. **71yr female, recurrent flaccid blisters. Diagnosis?**
    a. **Pemphigus Vulgaris**
14. **Asymptomatic mole. Growing. Diagnosis?**
    a. **Melanoma**
15. **3yrs lip licking → Crusting. Diagnosis?**
    a. **Impetigo**
16. **13 yr girl. Allopecia, Erythema. Diagnosis?**
    a. **Tinea Capitis Infection**
17. **83yr nodular lesion on face for 2 years. Diagnosis?**
    a. **BCC**
18. **Recurrent small itchy vesicles. Lesinos exacerbate following eating bread.**
    a. **Dermatitis herpetiformis**

What is the type of lesion?

1. Macule
2. Papule
3. Plaque
4. Nodule
5. Pustule
6. Wheal (hive)
7. Scales
8. Crust
9. Erosion/Ulcer





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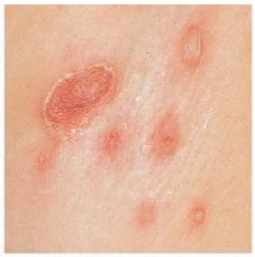

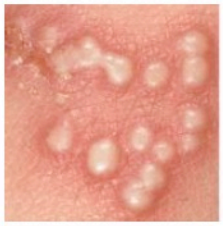

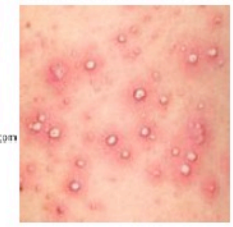

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8. Crust
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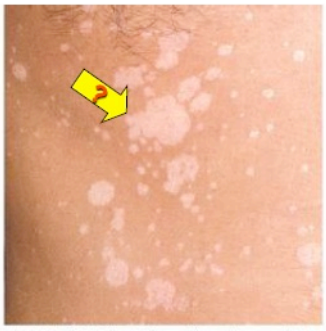

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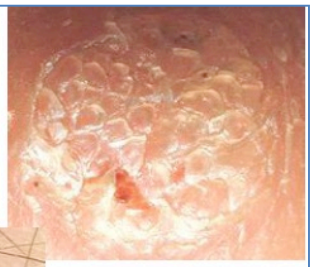

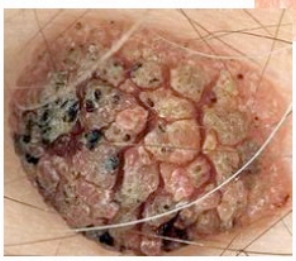

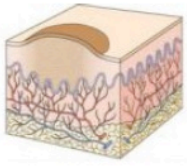

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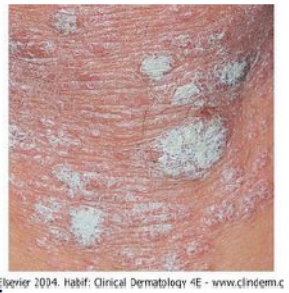

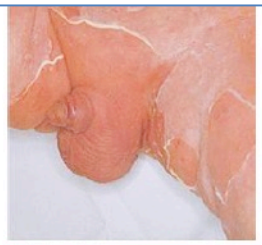

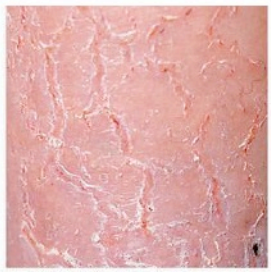

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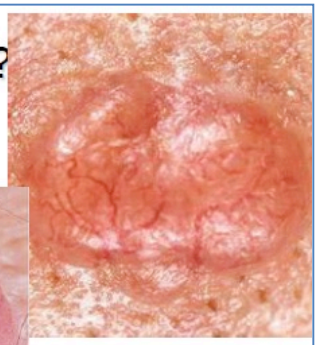

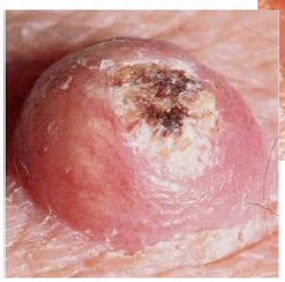

What is the type of lesion?

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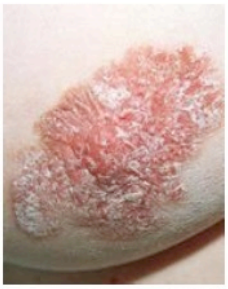
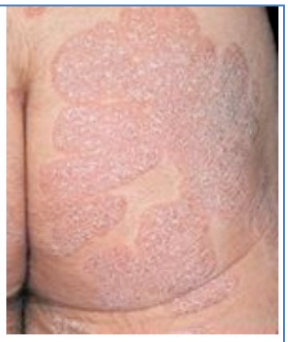
2. Papule
3. Plaque (Large areas of raised lesions)
4. Nodule
5. Pustule
6. Wheal (hive)
7. Scales
8. Crust
9. Erosion/Ulcer

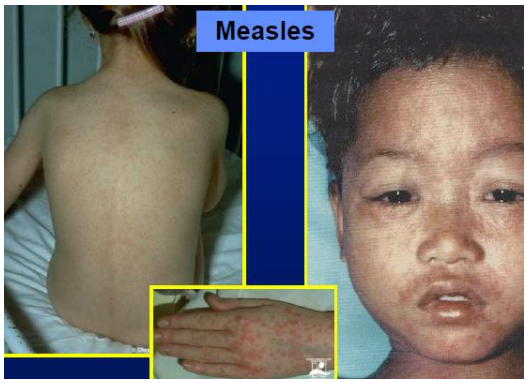




DERMATOLOGY Pathology:
Paediatric Rashes – Trigger Sheet

Measles Virus:

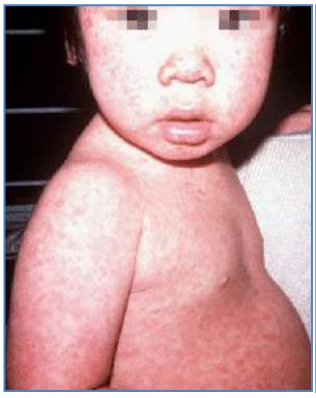
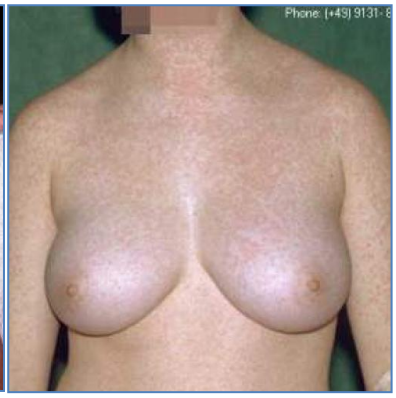
- **Organism:**
  - Morbillivirus
- **Transmission:**
  - Respiratory Route (Aerosol)
  - Contact with fluids from infected person's nose/mouth.
- **Pathogenesis:**
  - Typically a Respiratory Infection
  - →Produces a Viraemia → Rash
- **Presentation:**
  - Fever
  - URTI - Cough, Rhinorrhoea, Red Eyes
  - Maculopapular Erythematous (Morbilliform) Rash
  - "Koplik's Spots" – Seen on the Inside of the Mouth



- **Complications Include:**
  - Croup
  - Otitis Media
  - Enteritis with diarrhoea
  - Febrile convulsions
  - Encephalitis (Serious)
  - Subacute Sclerosing Panencephalitis (very rare)
    - (Chronic, progressive Encephalitis caused by persistent infection with immune-resistant Measles Virus)
    - No Cure
    - Fatal
- **Diagnosis:**
  - Clinical Diagnosis (Genearlised Maculopapular Rash + Fever)
  - Presence of Measles IgM Antibodies
  - PCR of Respiratory Specimens.
- **Treatment:**
  - No Specific Treatment
  - Prevented by MMR Vaccine
- **Prevention:**
  - Attenuated MMR Vaccine (Admin at 12mths & 4yrs)
  - Developing Countries: Low Herd Immunity → Higher Pervalence
    - Relatively High Death-Rates in Non-Immune.

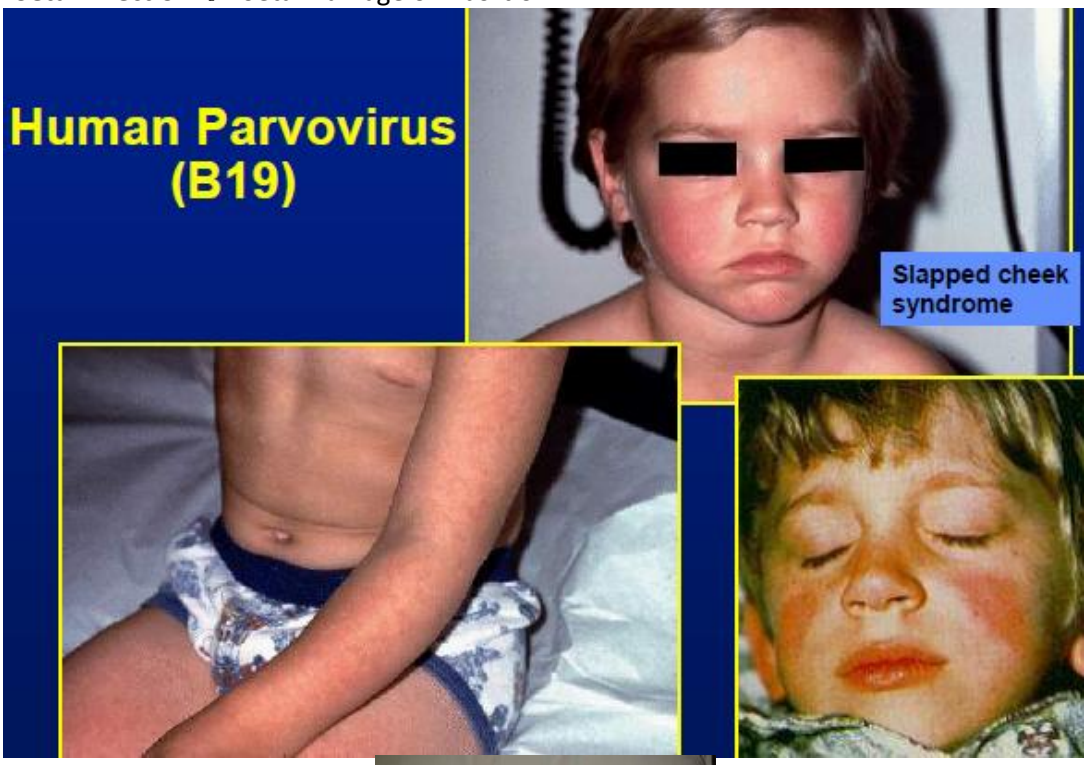
Rubella Virus (Aka "German Measles):

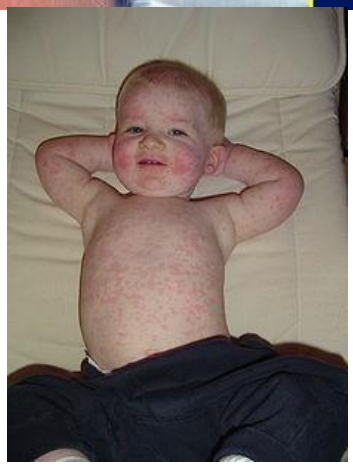
- **Organism:**
  - Rubella Virus
- **Transmission:**
  - Respiratory Route
  - (Human Reservoir Only)
- **Presentation:**
  - Initial Flu-Like Symptoms
  - * Rash on Face → Spreads to Trunk & Limbs
    - Pink-Red, Itchy
  - Low-grade Fever, Lymphadenopathy, Joint Pains, Headache, Conjunctivitis.
- **Prognosis:**
  - Typically Benign
  - Typically Lasts 1-3 Days (Children Recover Quicker)
  - Complications may include arthritis, thrombocytopaenia purpura, and encephalitis
  - ***HOWEVER, Maternal Infection During PREGNANCY can be SERIOUS!!**
    - If Infected in the 1st 20wks of Pregnancy → ***Congenital Rubella Syndrome***
      - →Abortion
      - →Cardiac/Cerebral/Opthalmic/Auditory Defects
    - Specific Foetal Damage Depends on Organ Development @ the Time:
      - The 1st Trimester is Worst, as Organ *Development* occurs during this time
      - After 1st Trimester, Organ *Growth* is the main process.
- **Diagnosis:**
  - Clinical Diagnosis
  - Presence of Virus-Specific IgM Antibodies
- **Treatment:**
  - No Specific Treatment
  - Controlled in Australia by vaccination (MMR Vaccine)
  - Test pregnant women for immunity early.
- **Prevention:**
  - (NB: Rubella *Itself* is relatively Benign, so why bother Vaccinating?)
  - **MMR Vaccine:**
    - **(Live Attenuated)**
    - **#1 Aim:** Prevent Rubella in Pregnant Women → ↓Congenital Rubella Syndrome.
    - Aimed at *BOTH* Males & Females to ↓Male Transmission to Pregnant Females

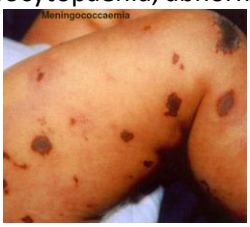
Human Parvovirus B19 → Childhood Rash called "5th Disease":

- **Organism:**
  - Parvovirus B19
- **Transmission:**
  - Respiratory Droplet
  - Blood-Borne
- **Pathophysiology:**
  - **Virus Replicates in Rapidly-Dividing Cells (Eg. Bone Marrow RBC Precursors)**
    - → RBC Haemolysis
    - → Severe Anaemia
    - → Can Result in ***Haemolytic Crisis***
  - The receptor for the virus is a globoside, which is abundant on tissues of mesodermal origin
  - **Can cross the placenta into the foetus**
    - → Foetal Anaemia
- **Presentation:**
  - Fever/Malaise
  - Characteristic Rash
    - Teenagers: 'Papular Purpuric Gloves & Socks Syndrome'
    - Children: 'Slapped Cheek Syndrome'
- **NB: Foetal Infection →**Foetal Damage or Abortion

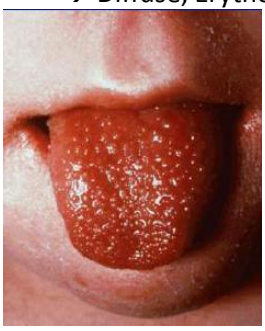
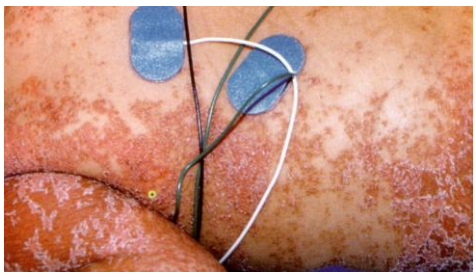




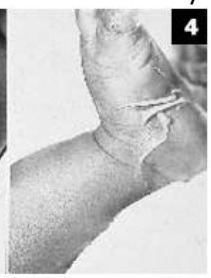
- **Purpura (Secondary To Septicaemia – Meningococcaemia – (*N.Meningitis*)):**
  - Due to extravasation of RBCs
  - Vessel wall defects (vasculitis)
  - Toxins (Endo/Exo) →Clotting defects (DIC, thrombocytopaenia, abnormal platelet function)

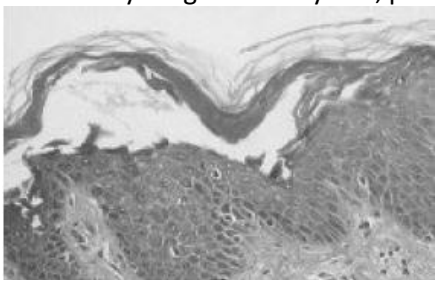
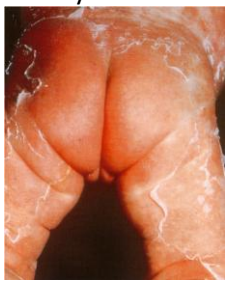


- **Toxin-Mediated:**
  - **Scarlet Fever ("Strawberry Tongue"):**
    - **Organism:**
      - Certain strains of ***Strep pyogenes*** *(Which carry a Bacteriophage* – A virus infecting the bacteria → Produce an Eruthrogenic toxin)
    - **Pathogenesis:**
      - GAS infection of Tonsils/Pharynx/Skin
      - ***Exotoxin*** Released by Strep. Pyogenes → Local effect on Tonsils/Pharynx/Skin
      - →Abnormalities of tongue
        - Initially covered with white exudate
        - Exudate is shed
        - inflammation of underlying tissue
      - → Diffuse, Erythematous Exanthem

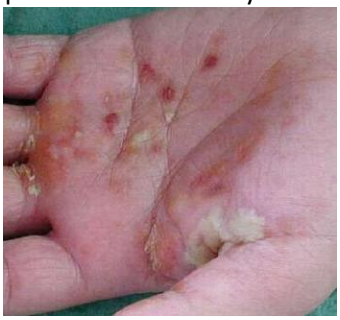



  - **Scalded Skin Syndrome (SSS) (Staphylococcus Aureus):**
    - **Organism:**
      - *S. Aureus* →Releases two Exotoxins (epidermolytic toxins A and B) from toxigenic strains
    - **Pathogenesis:**
      - Toxins Degrade Desmosomes → Acantholysis
    - **Presentation:**
      - Widespread Red Blistering Skin that looks like a burn or scald
      - occurs mostly in children younger than 5 years, particularly neonates

- **Toxic Shock Syndrome (TSS):**
  - **Organism:**
    - TSST-1-producing *Staph. aureus*
    - or TSST-1-producing *Strep. pyogenes*
  - **Pathogenesis:**
    - Gram Positive Organisms release Exotoxins (Superantigens)
    - → Mass, Non-Specific activation of T-Cells → Overproduction of Cytokines → Systemic Vasodilation → Shock
    - Cutaneous Response to toxins = Sunburn-like Rash + Desquamation
  - **Presentation:**
    - Rapid Onset Fever
    - Generalised Skin/Mucosal Erythema
    - Shock → Hypotension & Multi-System Failure



PATH
Summary of Pre-Malignant & Malignant Skin Lesions

Module Overview

- The majority of skin cancers are related to sun exposure and UV light which is a known carcinogen.
- **Commonest skin cancers in Australia:**
  - #1. Basal Cell Carcinoma (BCC)
  - Squamous Cell Carcinoma (SCC)
  - (Neither have a high risk of metastasis if diagnosed and treated early.)
  - Melanoma is less common but much more serious because of its metastatic potential

Sun Sensitivity

- Susceptibility of an individual to the effects of solar radiation.
  - Genetically determined
  - Related to skin colour.
- Measured by how quickly an individual develops redness (or sunburn) on exposure to ultraviolet light.
- Patients who are sun sensitive:
  - Burn easily
  - Are not able to tan
  - Show signs of solar damage earlier than normal
  - Are susceptible to skin cancer
  - Often have fair or red hair and freckles.

Sunburn

- Sunburn is an acute erythematous reaction to ultraviolet light and is usually induced by UVB radiation.
- Within 2 to 6 hours → painful erythema → may progress to blistering.
- The redness resolves over 4 to 7 days usually with peeling.

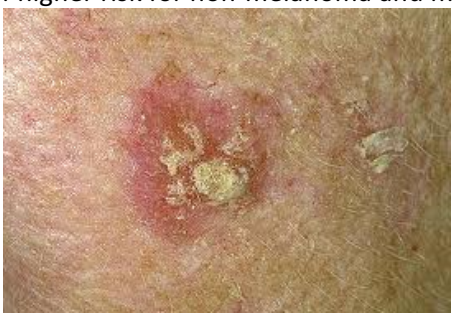
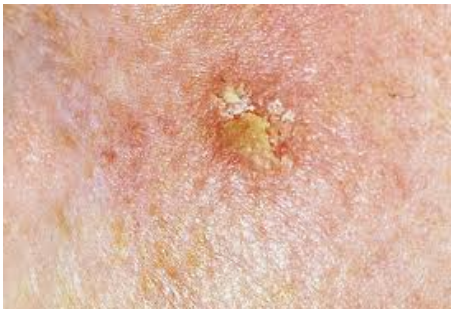
Actinic Damage (Sun-Damage):

- = Damage to the skin from ultraviolet (wrinkling, pigmentary change, actinic keratoses and skin cancer)
- UV radiation contributes to skin cancer and also has immunosuppressive effects
- The earliest sign = Freckling.
  - Babies are born without any freckles
- As patients age the following solar induced changes may occur:
  - Facial wrinkling
  - Telangiectasia
  - Yellow nodularity (solar elastosis)
  - Comedones not associated with acne (solar comedones)
  - Solar keratoses
  - Brown macules (solar lentigos) (“Sun-Freckles”)
  - Cutaneous fragility and easy bruising
  - White patches (guttate hypomelanosis)

BENIGN SKIN LESIONS:

Solar keratoses (Sunspots)

- Solar keratoses are erythematous, scaly lesions located on sun exposed skin. They are most common on the face, back of the hands and forearms.
- Solar keratoses are NOT cancer, however they are a sign of significant sun damage and are therefore an indicator of higher risk for non-melanoma and melanoma skin cancer.

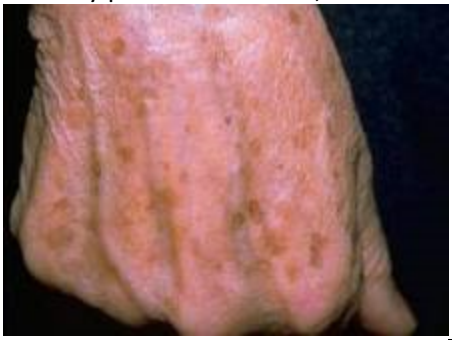
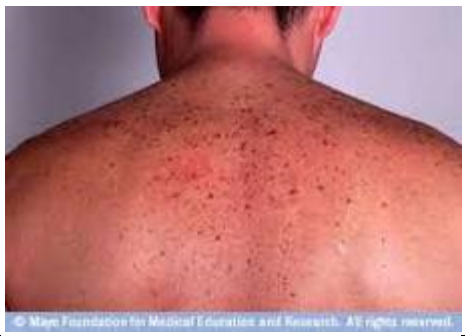
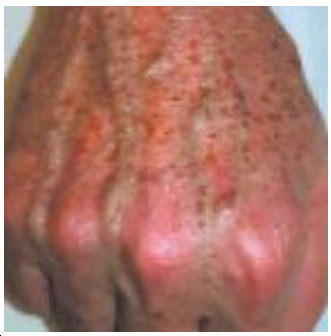



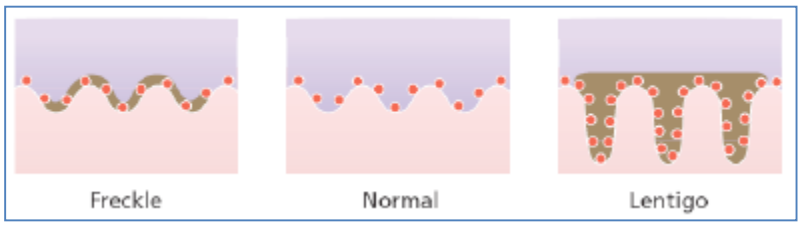
Freckles (ephelides):

- Tan macules up to 1cm Diameter.
- Arise in response to UV-Radiation Exposure.
    - May Regress if Exposure is Avoided.
- Freckles are normal in fair skinned people and appear for the first time in childhood as small, pale brown macules on the face and other sun exposed areas.

Solar Lentigo (Larger Freckles):

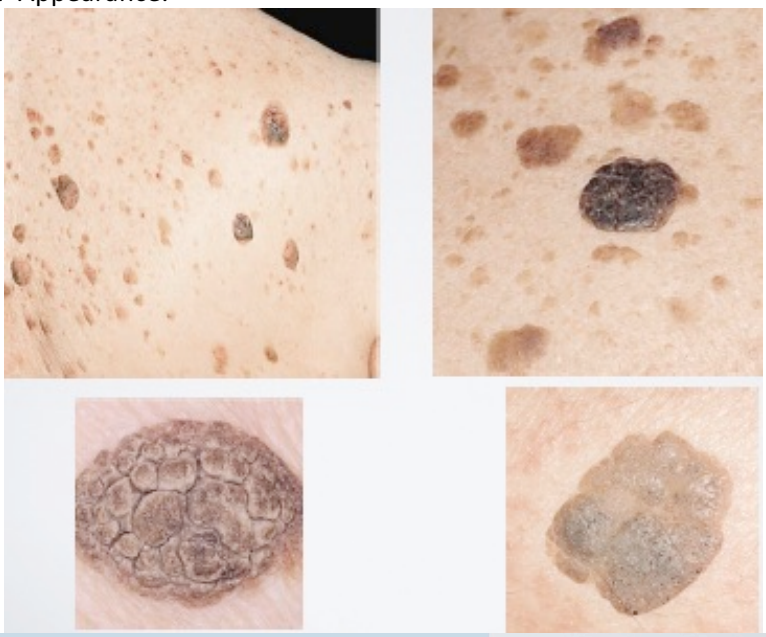
- Solar lentigines are extremely common in middle aged to elderly fair skinned people and are often found in association with sun damage.
- They present as brown, well-defined macules on sun exposed skin.

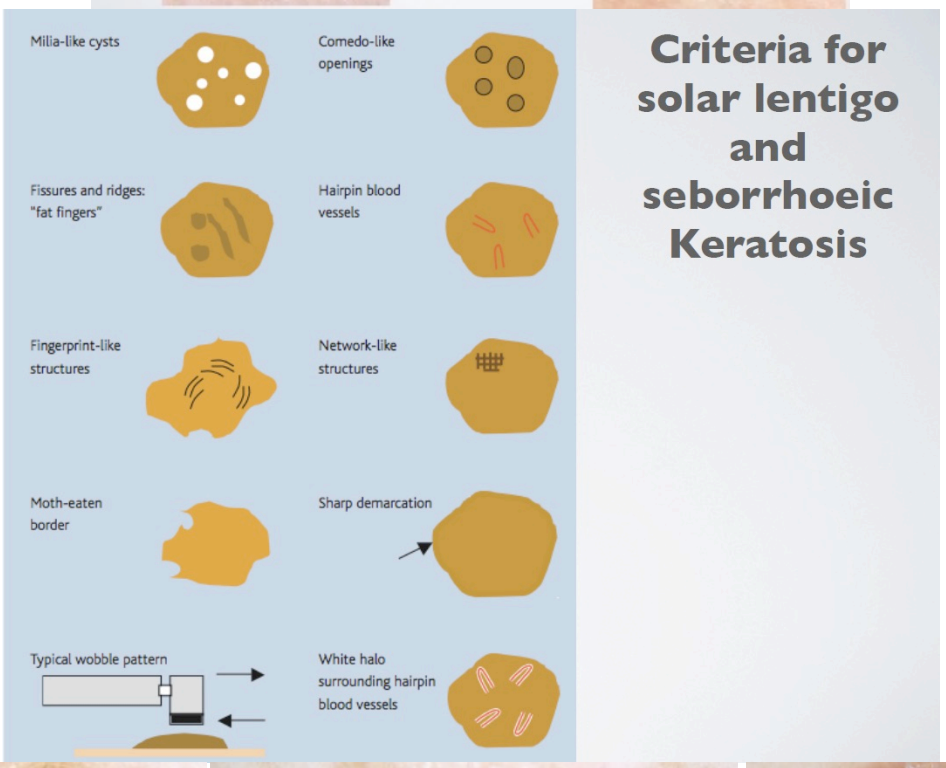






Seborrhoeic Keratoses:

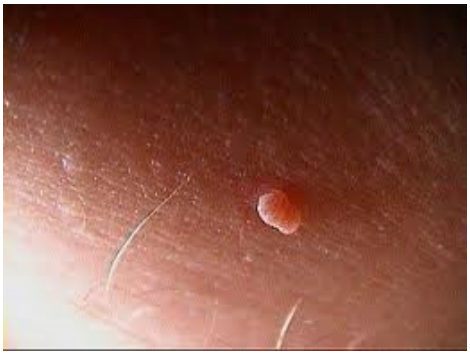
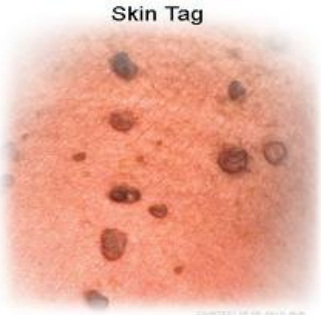
- These benign lesions are very common after the age of 40.
- They are often referred to as "old-age warts" because they do look very much like common warts.
- They have a rough, warty surface and colour ranges from skin coloured to black
- Sharply Defined Borders
- Raised, 'Stuck-on' Appearance.

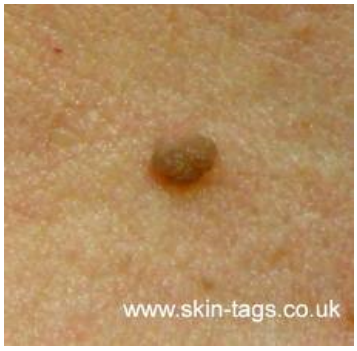




Skin Tags:

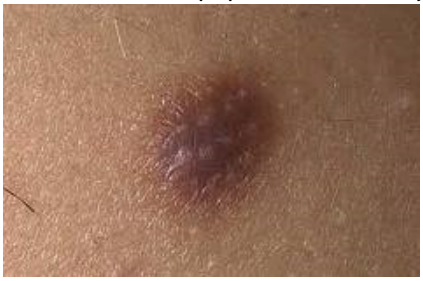
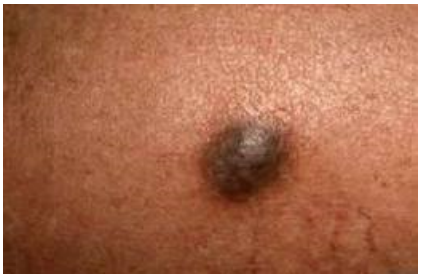
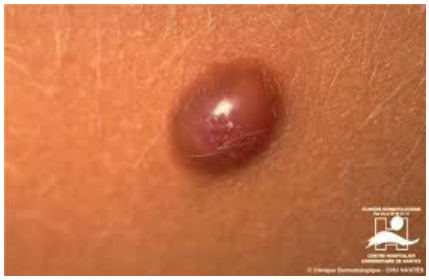
- These harmless lesions
- They are pedunculated and have a narrow pedicle. They are usually skin coloured to brown. Common sites are the intertriginous areas (axilla and groin) and the neck.



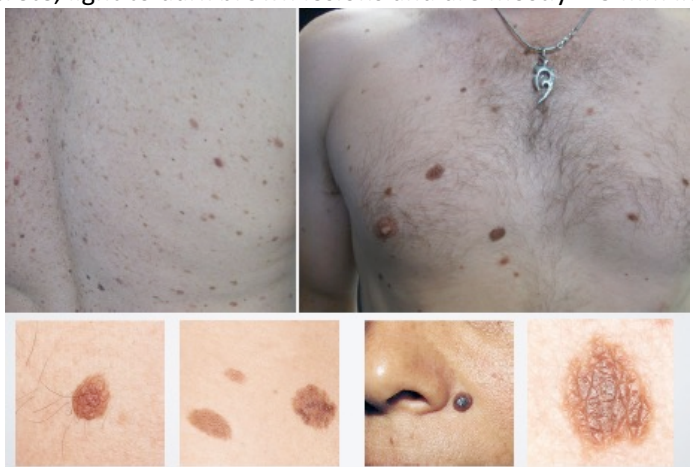
Dermatofibromas:

- This is a harmless and common lesion found in adults and children. It presents as a skin coloured to light brown firm papule which is asymptomatic.

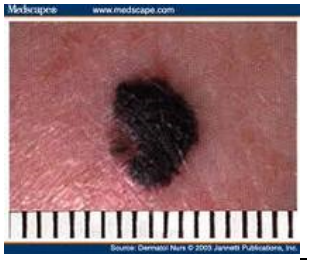
Common acquired Melanocytic Naevi (Moles):

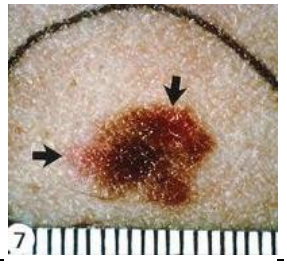
- These are benign tumours of melanocytes
- Are normal.
- The average number (that are > 2mm in diameter) found on the skin of Australians is close to 100.
- They present as discrete, light to dark brown lesions and are mostly 2-5 mm in diameter.



Dysplastic Naevi:

- These naevi present clinically and histologically as intermediates between normal naevi and melanoma
- most dysplastic naevi carry only a small risk of transformation to melanoma (about 1 in 1000)

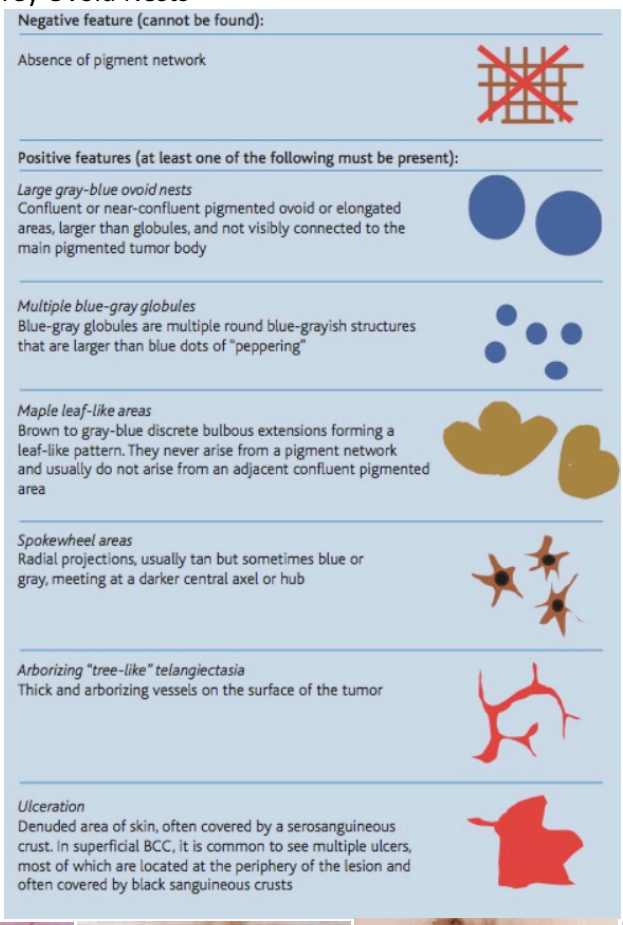


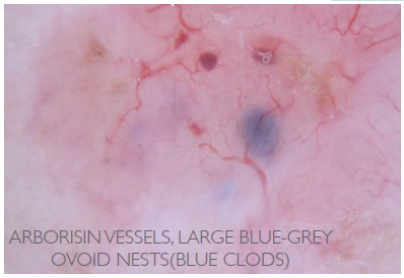


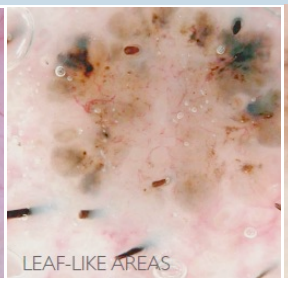

SKIN CANCERS

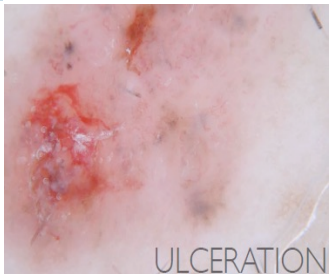
Basal Cell Carcinomas

- **(BCCs) are the commonest form of skin malignancy**
- **Commonest Subtypes:**
  - **Superficial BCC's**
    - Flat Red/Pink Shiny Macules or patches
    - Indistinct Borders
    - Short, Fine Telangiectasia (on Dermoscopy)
    - Can be Ulcerative (appear as ulcers with pearly or indurated edge)
  - **Nodular BCC's**
    - Pearly/red/translucent Nodules
    - Overlying telangiectasia.
    - Central Ulceration
- **Treatment:**
  - **Excisional surgery** : Still the gold standard treatment of BCC's.
  - Liquid nitrogen cryotherapy
  - Curettage and Cautery
  - Photodynamic therapy (PDT)
  - Radiation treatment
- **Criteria For Basal Cell Carcinoma:**
  - Important Stuff:
    - Blood Vessels (Telangiectasia)
    - Blue-Grey Ovoid Nests









Squamous Cell Carcinomas:

- Invasive Squamous cell carcinoma (SCC) is the second most common form of skin malignancy after BCC.
    - Well differentiated tumours present on chronically sun damaged skin, as indurated nodules with a keratotic (norny growth)surface, (squamous cells produce keratin).
    - They frequently grow rapidly and may be painful.
- **Aetiology:**
    - The vast majority of SCC's occur on Sun-Exposed Areas
    - Associated with Chronic Sun Exposure
- **Clinical Presentation:**
    - Highly Variable Appearance
    - Typically Tender Papules/Nodules with *ROUGH, ADHERENT SCALE*.
    - Symmetrical
    - Often Ulcerate Centrally
    - Often Present as Ulcers on Lips/Mucosa
- **Treatment:**
    - Wide excision
    - Radiotherapy in Elderly

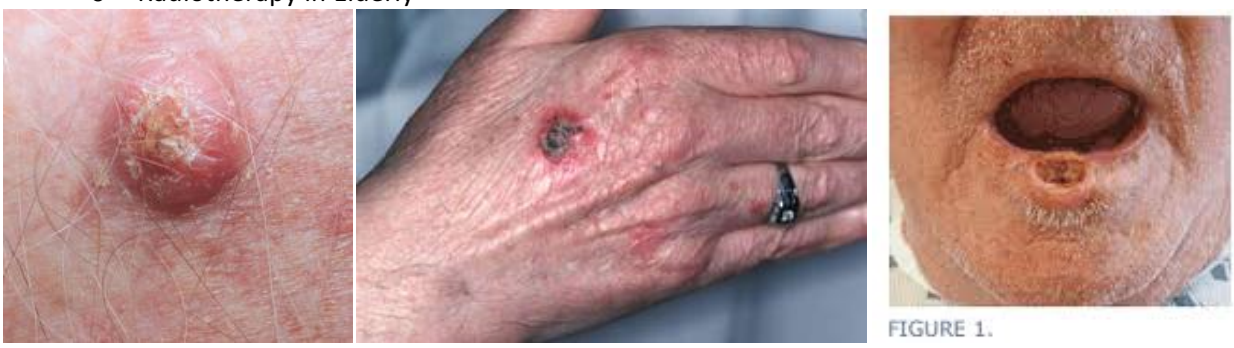
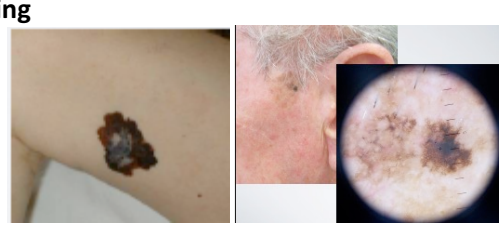


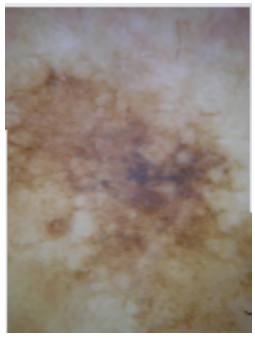
FIGURE 1.

Melanoma:

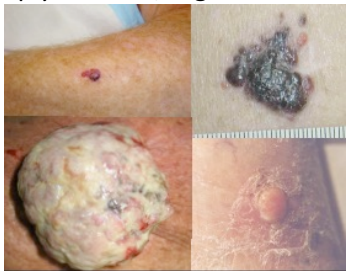
- **Epidemiology:**
  - Australia - highest incidence of melanoma in the world
  - Most common cancer in northern Aus
  - incidence is higher in the elderly
  - **3rd Most Common Cancer in Both Men & Women**
  - **Risk:**
    - 1/23 for Females
    - 1/14 for Males
- **Risk Factors:**
  - Family history
  - Fair complexion
  - Freckles
  - Tendency to burn (Fitzpatrick type I/II skin)
  - Severe Childhood Sunburn (Solar Skin Damage)
  - Immunosuppression
  - High number of common acquired naevi
  - Large (>10cm) Congenital Naevi
  - Dysplastic Nevus Syndrome (5 or more dysplastic naevi)
- **Pathways to Melanoma:**
  - Most develop in an existing Naevus
  - Superficial Spreading melanomas grow Laterally Initially, then begin to grow downwards.
- **Types of Melanoma:**
  - **"Superficial Spreading Malignant Melanomas"** - linked to intermittent intense sun exposure (sunburns)
    - Presents with a flat, usually pigmented, asymmetric macule that is changing is size, shape or colour
    - **Slow Growing**



  - **"Lentigo maligna melanoma"** – linked to high doses of cumulative sun exposure
    - presents as an unevenly pigmented, asymmetric facial freckle that is changing in size, shape or colour

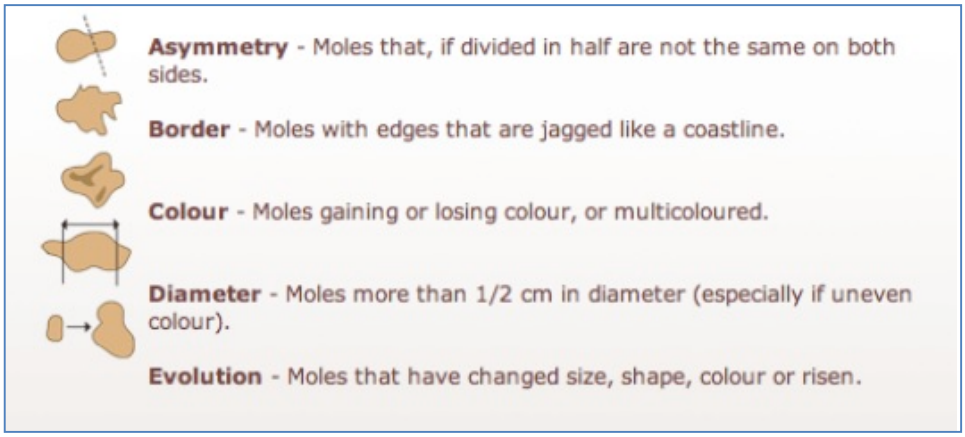


  - **"Nodular Melanoma" –** Due to Medium Sun Exposure
    - Tends to grow more rapidly in depth than other melanomas.
    - The majority of deeply invasive, High-risk Melanomas

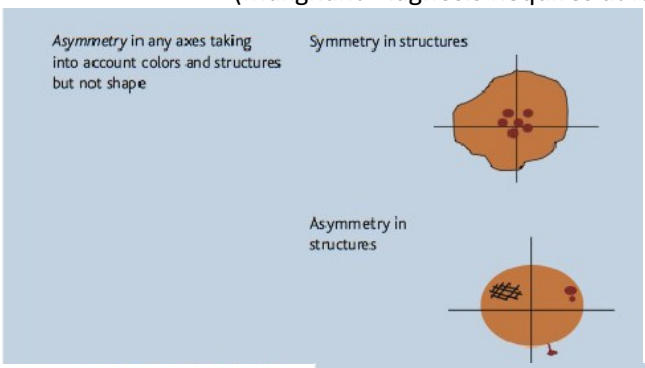


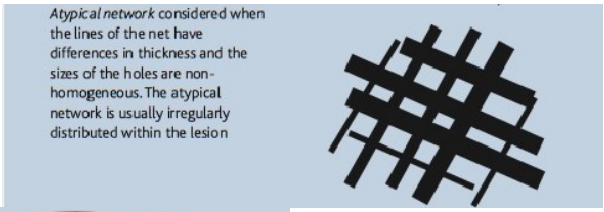
- **"Lentigo Melanoma" –** On Skin of Face & Neck
- **"Acral Lentiginous Melanoma"** – NOT due to sun exposure.
  - Affects soles of the feet, palms of the hands, toes, and fingers, and occurs in the nail apparatus.
- "**Amelanotic Melanoma**"- Red Patches on the Skin
  - Many melanomas are partially non-pigmented and up to 20% are almost completely without pigmentation. Amelanotic melanomas generally present as a red, changing lesion though they can be skin coloured.

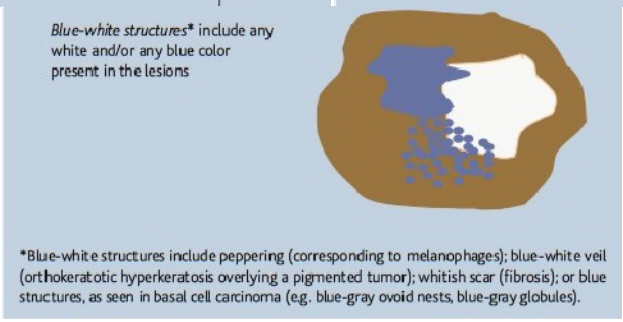
- **Presentation:**
  - **Begin with a Radial Growth Phase (RGP)**
    - Grow wider (↑Diameter)
  - **Vertical growth Phase Melanoma (VGP)**
    - VGP will eventually supervene within most RGP melanomas and typically presents as a raised area that is progressively growing
- **Diagnosis:**
  - Dermoscopy is ESSENTIAL!!
  - Skin exam without dermoscopy will only pick 60% of melanomas.
  - How long has it been there?
  - **ABCD's of melanoma:**
    - **A – Asymmetry o**f Shape and Structure.
      - Ie. One half doesn't match the other half (border/colour/structure within the lesion).
    - **B – Border Irregularity**. The edges are ragged, blotched, or blurred.
    - **C – Colour Variability**. The pigmentation is not uniform. Shades of tan, brown, and black may be present and red, white, grey and blue may add to the mottled appearance.
    - **D – Diameter Increasing**. A width >6mm (about the size of a pencil eraser).
      - NB: Any growth of a mole should be of concern.
    - **E –**
      - **Evolving** - any lesion that is changing or enlarging
      - **Elevated** - Different elevations/contours *OR* ANY change from flat to elevated is of concern.
    - **F – Firm**. A lesion that is firm, and also ones that are friable (easily damaged) are more likely to be malignant. Whilst benign moles are raised, soft and "wobble" like jelly with pressure.
    - **G – Growing**. Signs of growth (Slow/Rapid) should raise concern.



- **3 Point Checklist:**
  - 1. Assymmetry of Colour/Structure?
  - 2. Atypical Network?
  - 3. Blue-White Structures?
  - (Malignant Diagnosis Requires at least 2 of the above)





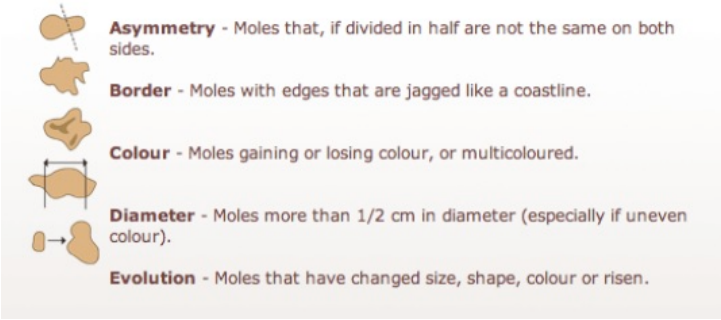


  - **Additional Dermoscopic Features:**
    - Multiple colours
    - Irregular distribution of dots & Globules
    - Streaks at the edges
    - Blotches
  - **Prognostic Factor:**
    - **Breslow Thickness:**
      - Tumour thickness measured from the granular layer of the epidermis to the deepest identified tumour cell
      - Best Available predictor of Prognosis
    - **Poor Prognostic Factors:**
      - Ulceration
      - High Mitotic Rate
      - Signs of Regression = Bad
    - **Clark Levels:**
      - Relateds to the Deepest invasive tumour cells
      - Levels 1 – 5
      - (Not the same as TNM Staging)
    - **TNM Staging:**
      - **T = Size/Depth**
      - **N = Nodal Involvement**
      - **M = Distant Metastasis?**

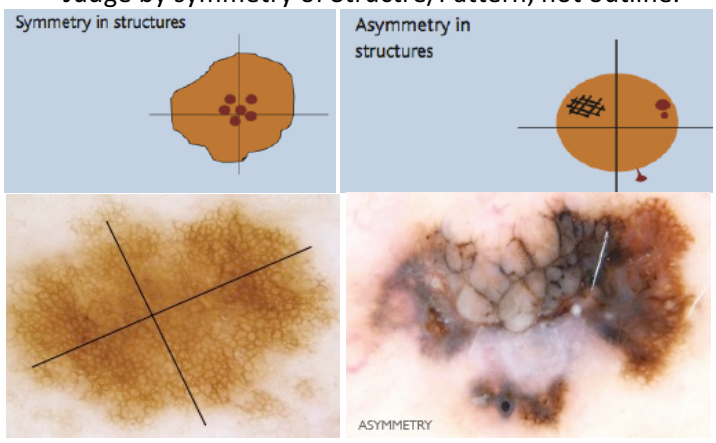
- **History:**
  - vital when assessing a pigmented lesion
  - Melanomas are progressively changing lesions
  - Change in Size, Shape or Colour?
- **Treatment:**
  - **Early Detection & Removal** = The Only Reliable Curative Treatment.
  - **Followup** = Those who have had a melanoma, have a 13% risk of another.

Clinical Diagnosis – Screening For Malignancy:

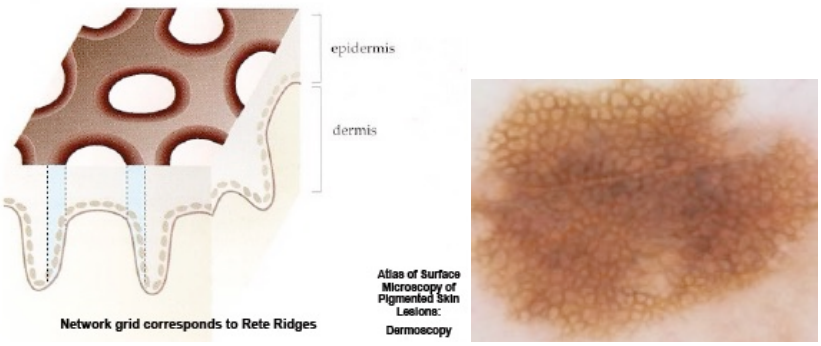
- **Use the ABCDE and EFG rule**



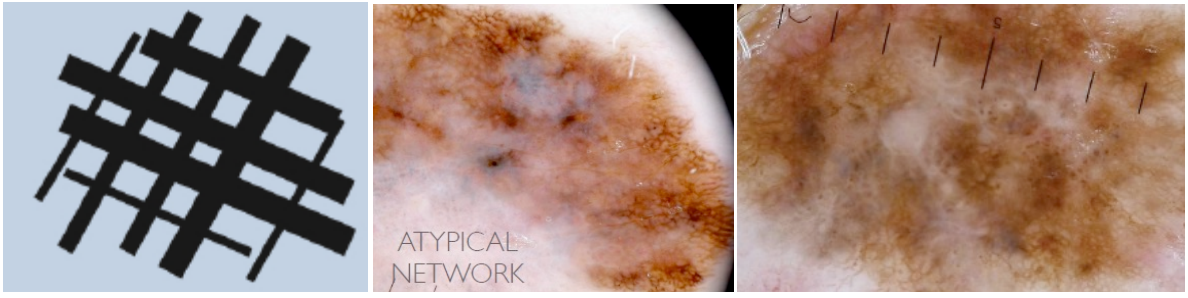
- **Dermoscopy:**
  - Hand-Held
  - 10x Magnification
  - Eliminates Surface Reflection (Allows viewing of Deeper Structures)
    - Via Polarised Lens
    - Non Polarised Lenses require a fluid layer between plate & skin. (Oil, Alcohol, Handwash Gel, KY lube, Ultrasound Gel)
  - **3 Point Checklist in Dermoscopy:**
    - (A Screening tool for Malignancy)
    - High Sensitivity, But Low Specifictiy (Many benign lesions score higher than 1)
    - **1. Asymmetry of Colour and /or Structure**
      - Judge by symmetry of Structre/Pattern, not outline.



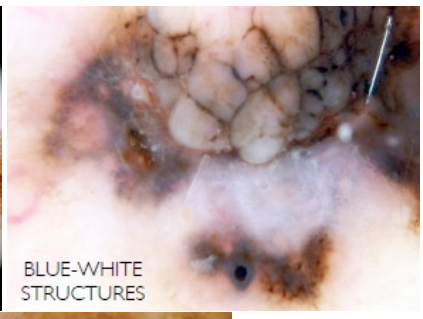
    - **2. Atypical Pigment Network:**
      - NB: Typical Pigment Network Grids correspond to Rete Ridges in the Dermis.

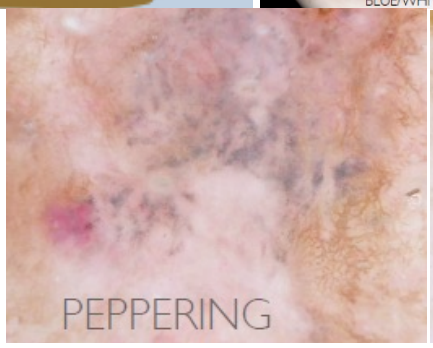


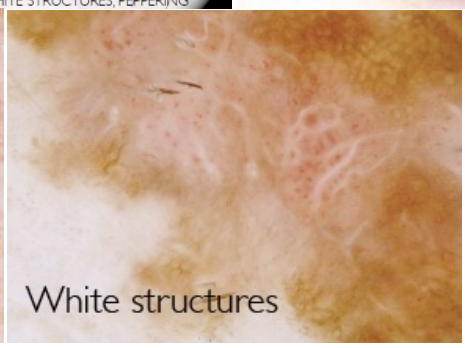
      - Atypical Networks = Lines are of different thickness & sizes of 'holes' are varied.



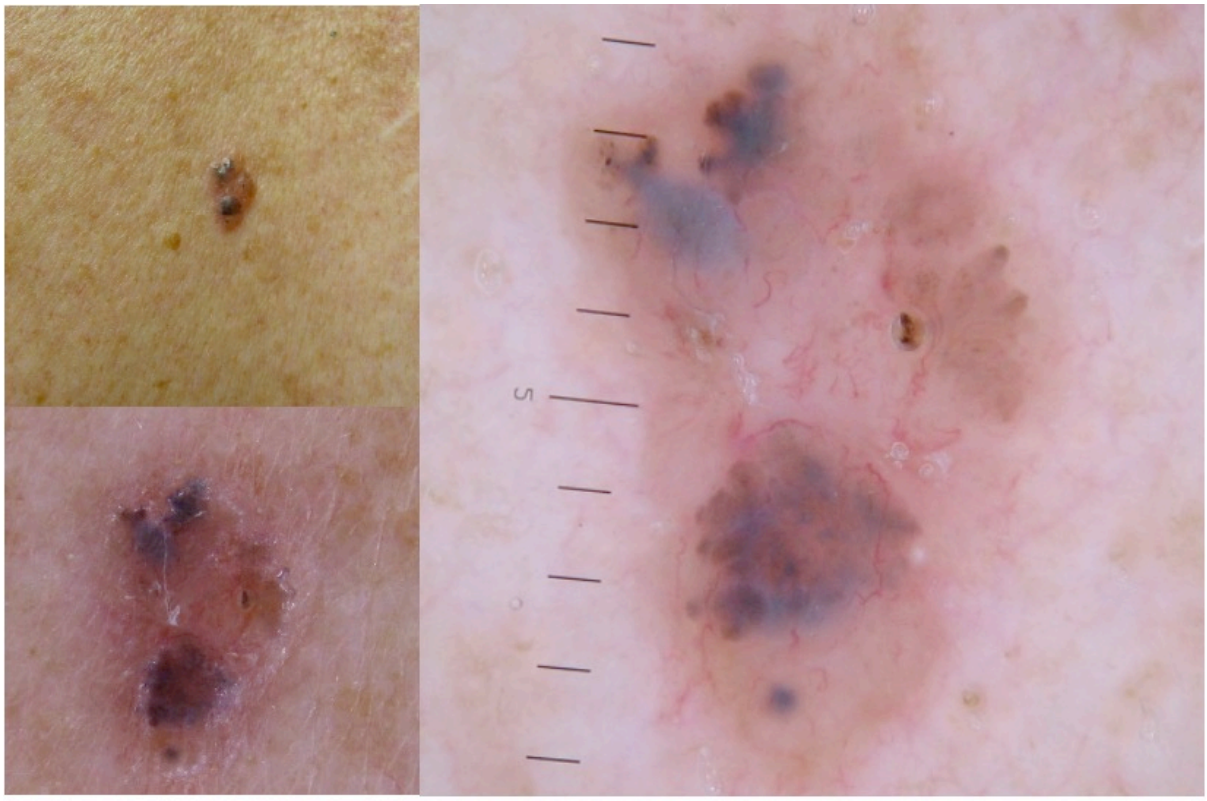
- **3. Blue-White Structures:**
  - Any White and/or Blue colour present in the lesions.
  - Include:
    - ‘Peppering’ (Melanophages)
    - Blue-white Veil (Orthokeratotic Hyperkeratosis overlying the Pigmented Tumour)
    - Whitish Scar (Fibrosis)







Case 1.



CASE 1. Male 45 lesion over scapular, patient unaware of it, palpable but not firm or hard.

Clinical Description:

- Small, brown papule
- 9mm diameter
- Irregular shape

ABCD (EFG) Criteria:

- Assymetrical
- Border Irregularity
- Colour Variability
- Diameter increasing unknown.
- Don't know if Evolving
- Elevated

Three Point Checklist Score (Malignant):

- Assymmetry?
  - Yes
- Atypical Network?
  - No
- Blue-white Structures?
  - Yes

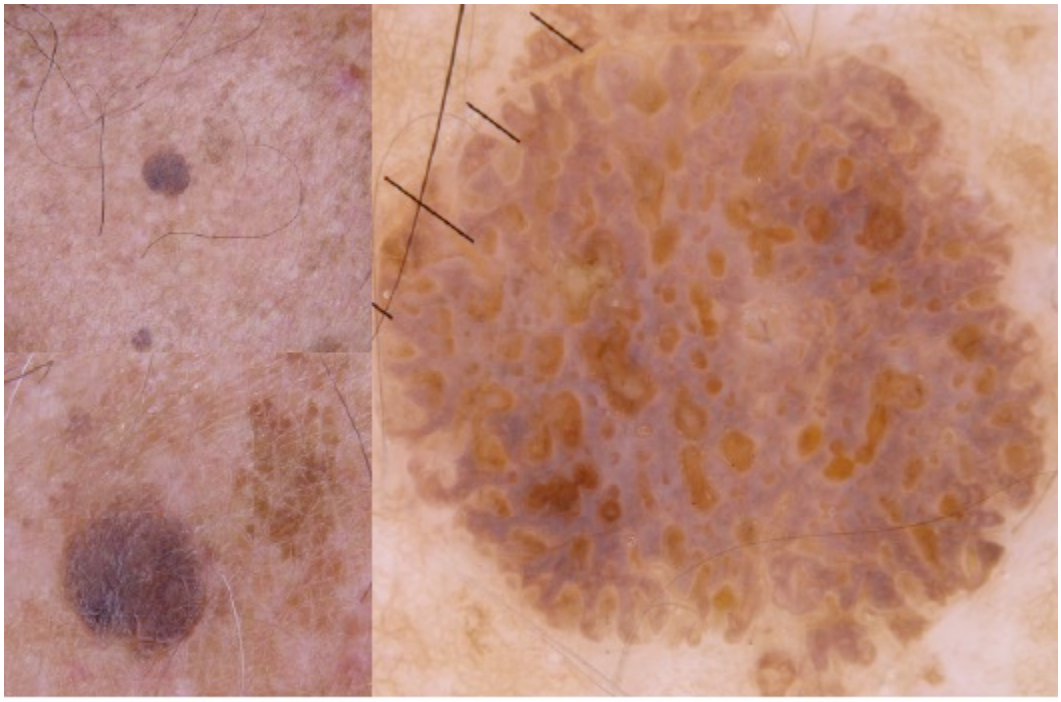
Provisional Diagnosis:

- Potentially malignant BCC

Management:

- Excision

Case 2.



CASE 2. Male 57 palpable plaque on back.

Clinical Description:

- 5mm diameter
- Papular-Plaque
- Brown
- Well defined border
- Regular shape & Pigment distribution
- Has ridges & fissures/como-like openings

ABCD (EFG) Criteria:

- Symmetrical
- Regular borders
- Regular Colour
- Diameter not increasing
- Not evolving
- Elevated
- Not growing

Three Point Checklist Score:

- Assymmetry?
  - No
- Atypical Network?
  - No
- Blue-White Structures?
  - No

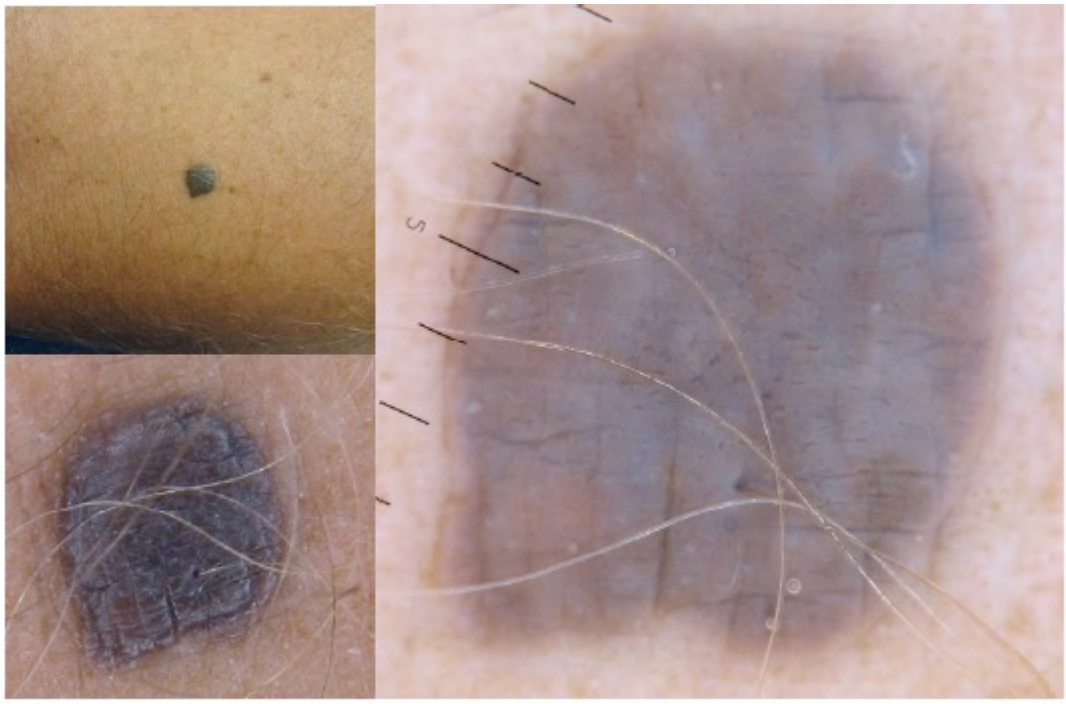
Provisional Diagnosis:

- Benign Seborrheic Keratosis

Management:

- Monitor
- No management needed.

Case 3.



CASE 3. Male 32 years long standing lesion on medial calf no history of change, palpable plaque soft.

Clinical Description:

- 1cm wide plaque
- Well defined border
- Regular shape
- Regular pigmentation

ABCD (EFG) Criteria:

- Symmetrical
- Well defined border
- Regular Colour
- No history of change (diameter/evolution)

Three Point Checklist Score:

- Assymmetry?
    - No
- Atypical Network?
    - No
- Blue-Grey Structures?
    - Yes

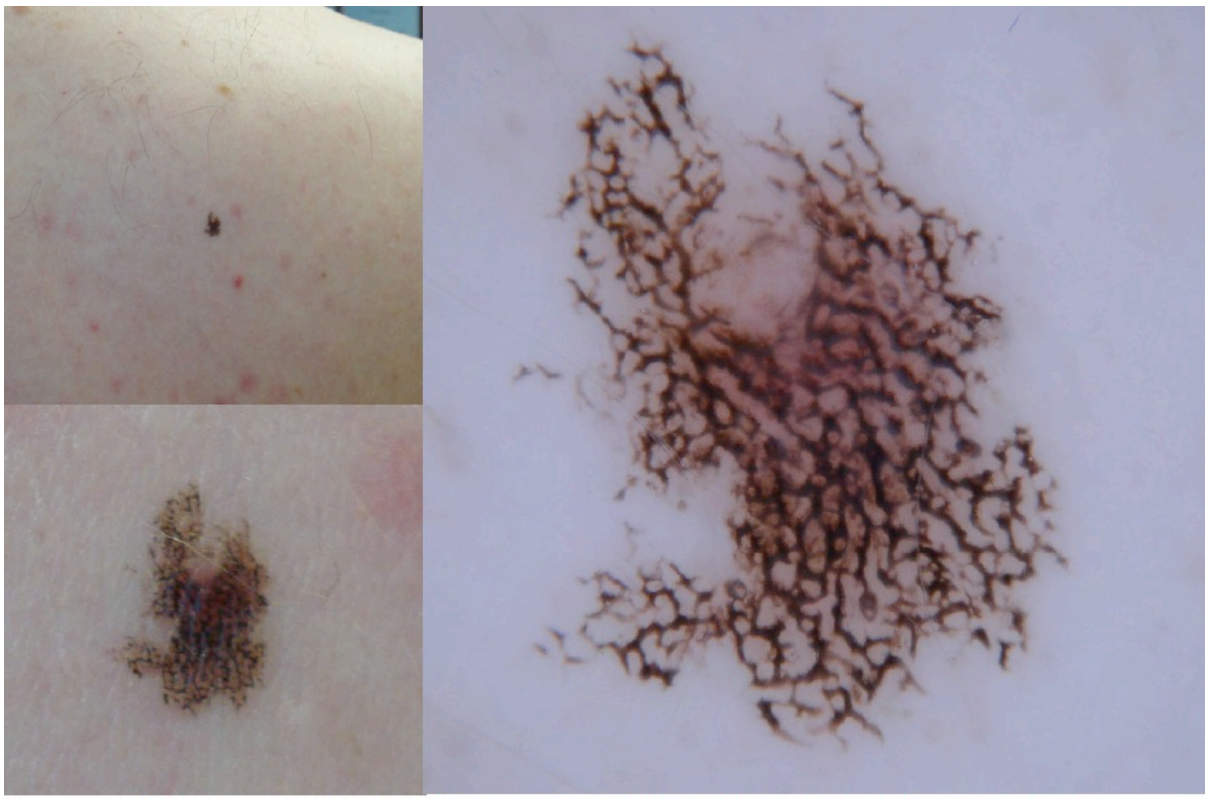
Provisional Diagnosis:

- Benign
- Blue Naevus

Management:

- Monitor
- No Management Needed

Case 4.



CASE 4. Male 18, Fitzpatrick type 1 skin, one of several similar maclure lesion on upper trunk and proximal upper limbs

Clinical Description:

- Small pigmented lesion
- Macule (not raised)
- Irregular border

ABCD (EFG) Criteria:

- Symmetrical pattern, asymmetrical shape
- Irregular Border
- Consistent Colour
- No history of increasing Diameter/Evolution

Three Point Checklist Score:

- Assymmetry?
    - No
- Irregular Network?
    - No
- Blue-Grey Structures?
    - No

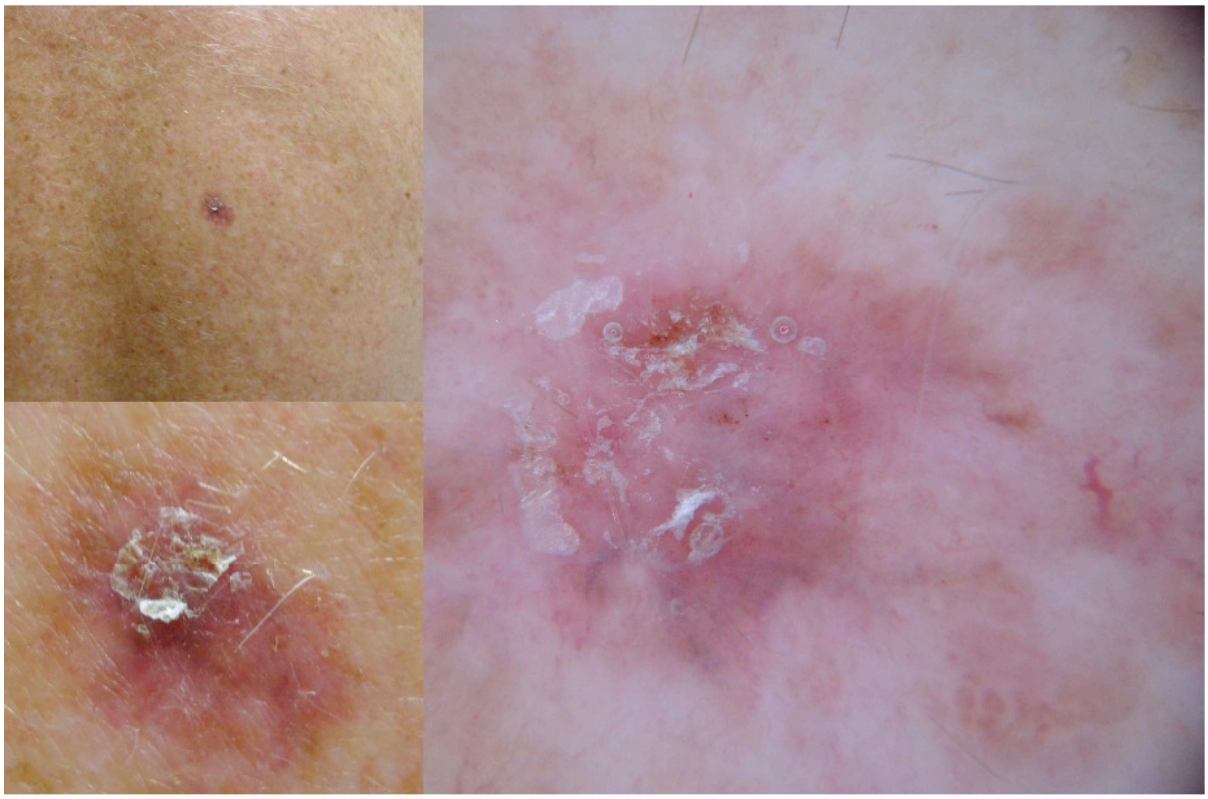
Provisional Diagnosis:

- Benign
- Ink-Spot Lentigo

Management:

- No management needed

Case 5.



CASE 5. Male 56 years, enlarging lesion on back noted by partner 6 months previously, recently itchy, macule with surface scale.

Clinical Description:

- Scaly raised, erythematous Macule
- Poorly defined border
- Erythematous base
- scaling

ABCD (EFG) Criteria:

- Assymmetrical
- Irregular border
- Consistent Colour
- Increasing Diameter
- Evolving

Three Point Checklist Score:

- Assymetry?
  - Yes
- Irregular Network?
  - No
- Blue-Grey Structures?
  - Yes

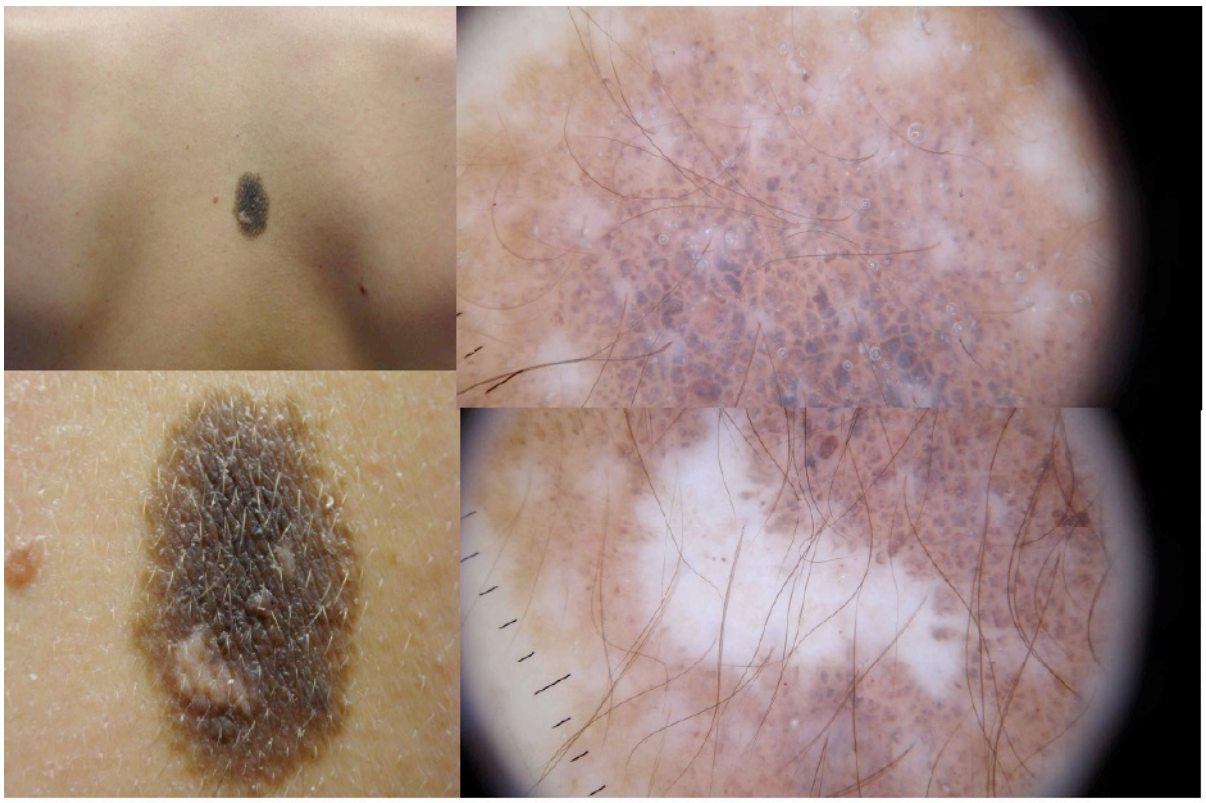
Provisional Diagnosis:

- Malignant
- Amelanotic Melanomas

Management:

- Excision

Case 6.



Case 6. Male 36 lesion present since infancy no history of change, plaque with warty sufrace

Clinical Description:

- Large pigmented plaque
- Warty surface
- No history of change
- Well defined border

ABCD (EFG) Criteria:

- Symmetrical in shape but asymmetrical in structure
- Well defined Border
- Consistent Colour except for area of white regression
- Constant diameter
- No history of evolution

Three Point Checklist Score

- Assymmetry?
  - No
- Irregular Network?
  - No
- Blue-White Structures?
  - Yes

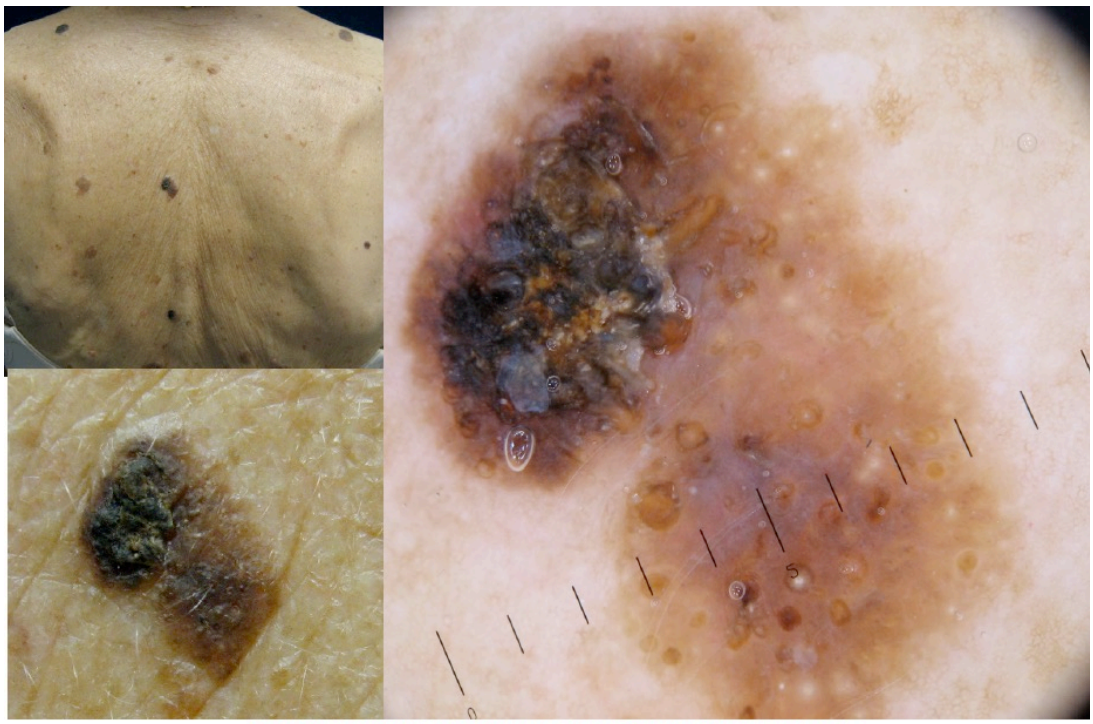
Provisional Diagnosis:

- Benign
- Birth Mark (congenital Naevus)

Management:

- None needed

Case 7.



CASE 7. Female 75 one of multiple similar lesion on trunk, plaque with stiff surface

Clinical Description:

- Asymmetrical macula-papular lesion
- Crusted surface
- Large
- Pigmented
- Milia like cysts, comedo-like openings.

ABCD (EFG) Criteria

- Assymmetrical
- Well-Defined Borders
- Regular Colour
- Diameter ≈ 1cm

Three Point Checklist Score

- Assymmetry?
  - Yes
- Irregular Network?
  - No
- Blue White Structures?
  - Yes

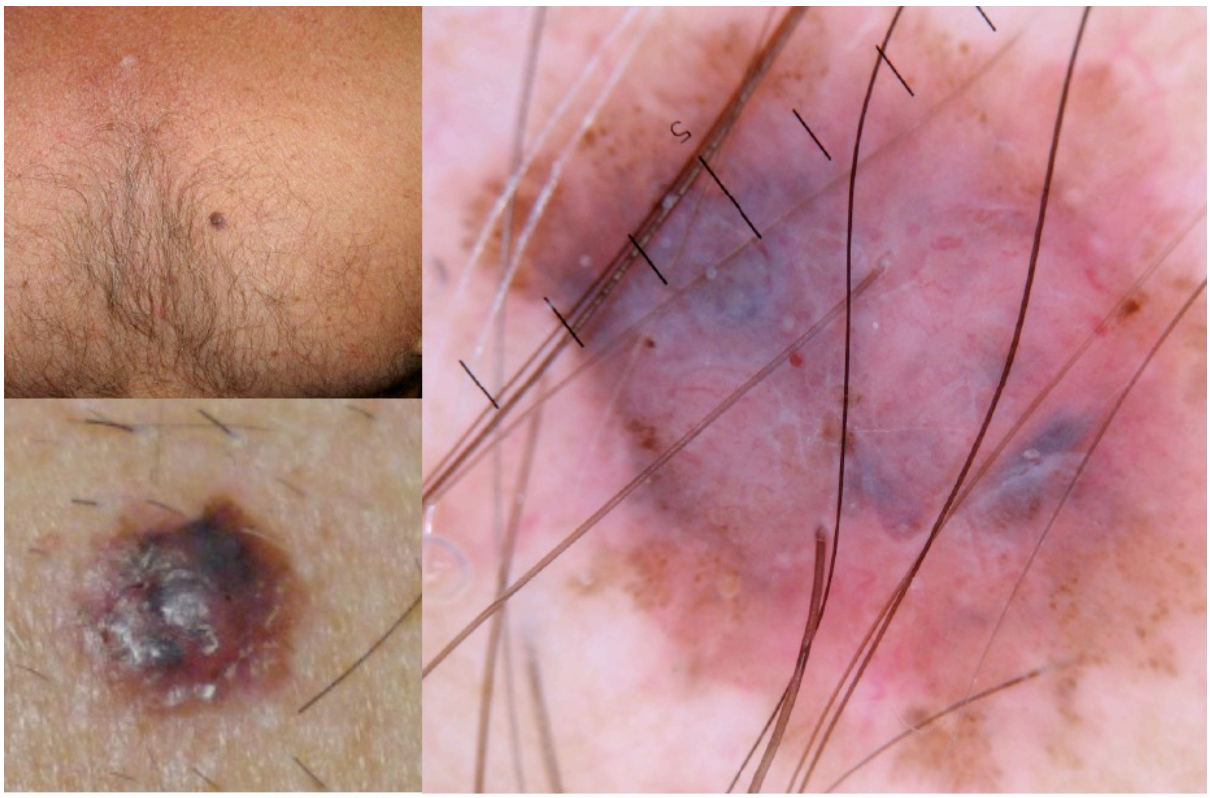
Provisional Diagnosis:

- Seborrheic Keratosis
- Benign

Management

- None Needed

Case 8.



CASE 8. Male 45 years lesion chest, increasing in size over 12 months, palpable, firm.

Clinical Description:

- Papular lesion on chest
- 7mm diameter
- Well defined borders
- Regular shape
- Increasing in size over 12 mths

ABCD (EFG) Criteria:

- Symmetry in shape, but Assymmetry in colour

Three Point Checklist Score:

- Assymmetry?
  - Yes
- Irregular Network?
  - No Network (Many melanomas don't have network)
- Blue White Structures?
  - Yes

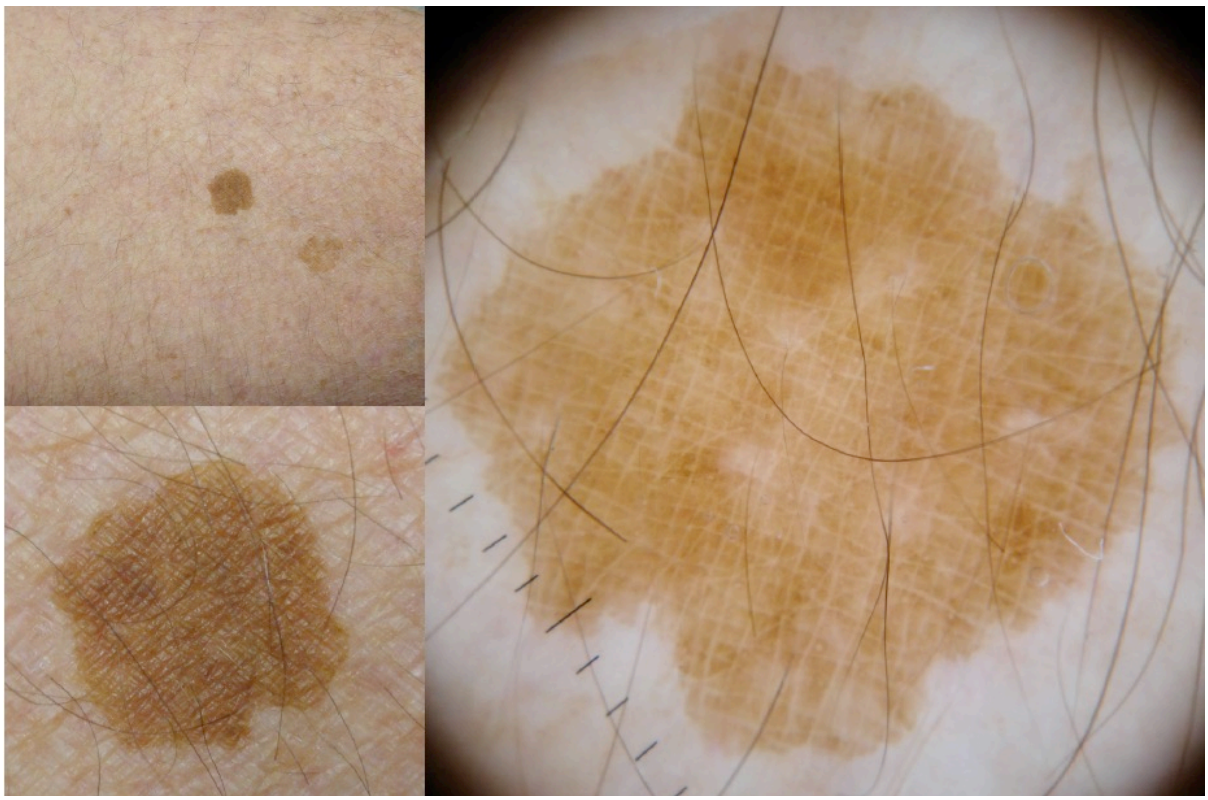
Provisional Diagnosis:

- Malignant
- Invasive Melanoma

Management:

- Excision

Case 9.



Clinical Description:

- Pigmented macule
- Symmetrical
- Well defined, regular border
- Enlarging
- 1cm diameter

ABCD (EFG) Criteria:

- Symmetrical
- Regular, well defined borders
- Regular consistent colour
- Diameter 10mm
- Evolving? Growing

Three Point Checklist Score:

- Assymmetry?
  - No
- Irregular network?
  - No
- Blue-white structures?
  - No

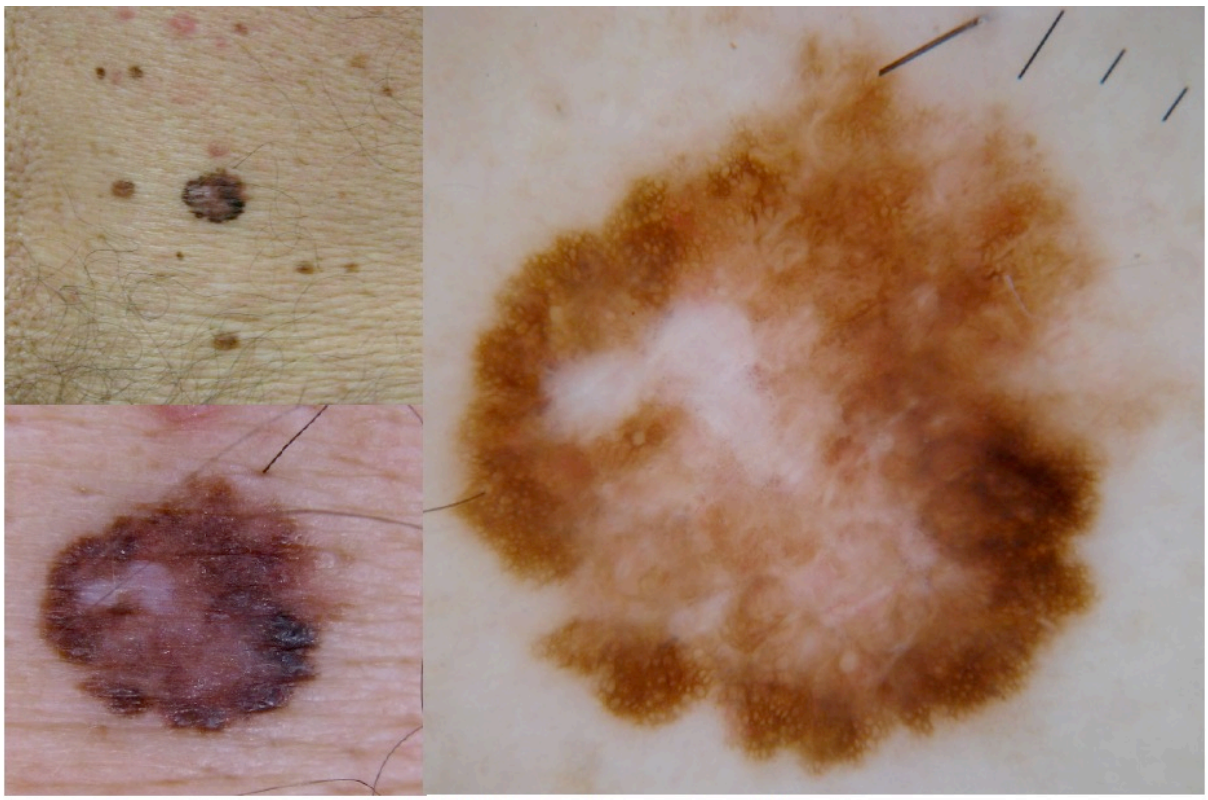
Provisional Diagnosis:

- Solar Lentigo

Management

- None Needed

Case 10.



CASE 10. Male 48 longstanding pigmented macule lower back.

Clinical Description:

- Pigmented Macule
- Irregular Colour & Border
- Assymmetrical
- Long-Standing
- Regression in the centre → white area

ABCD (EFG) Criteria:

- Assymmetrical
- Irregular Border
- Irregular Colour
- Diameter 9mm
- Evolving? NO

Three Point Checklist Score

- Assymmetrical?
  - Yes
- Irregular Network?
  - Yes
- Blue white structures?
  - Yes

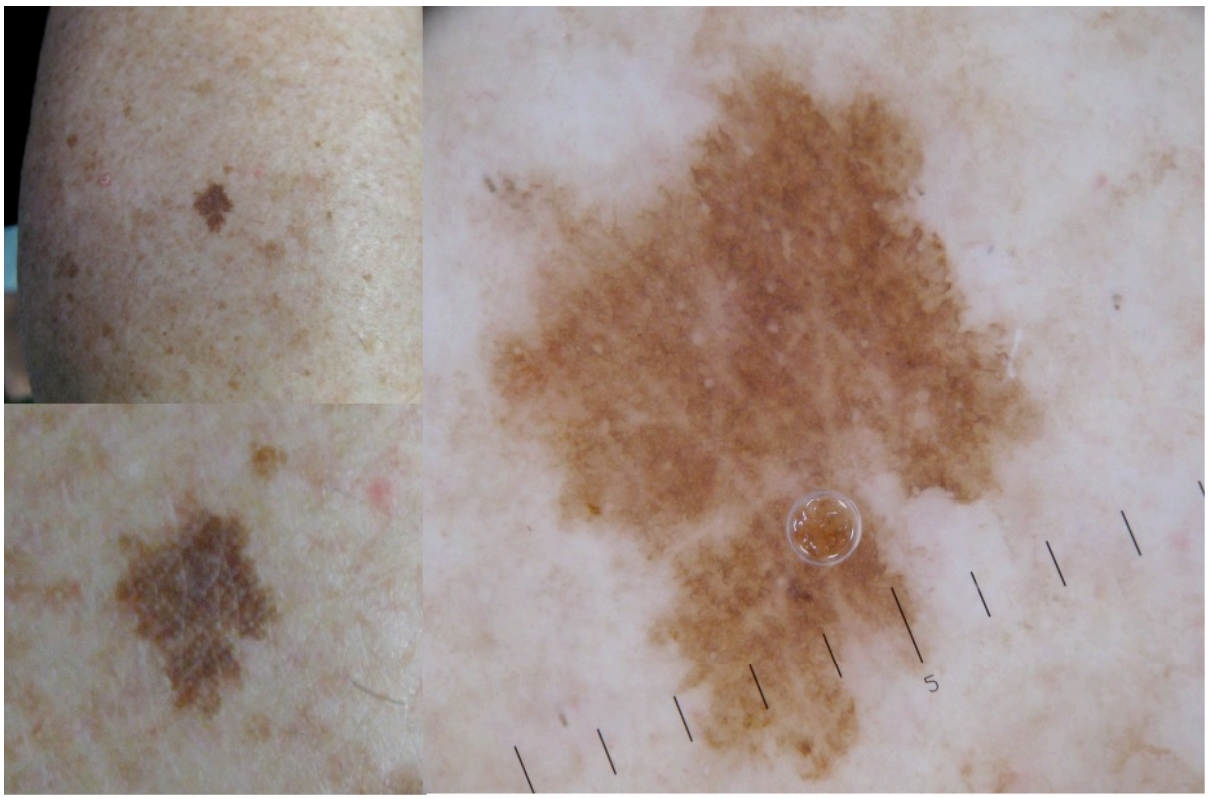
Provisional Diagnosis:

- Melanoma evolving inside a dysplastic naevi

Management:

- Excision

Case 11.



CASE 11. Female 71, enlarging pigmented macule shin.

Clinical Description:

- Enlaring Macule
- Iregular Border
- Consistent Colour
- Symmetrical
- Faint Network

ABCD (EFG) Criteria:

- Symmetrical
- Irregular Borders
- Consistent Colour
- 1cm diameter

Three Point Checklist Score

- Assymmetrical?
  - Yes
- Blue white Structures?
  - No
- Irregular Pigment Network?
  - No

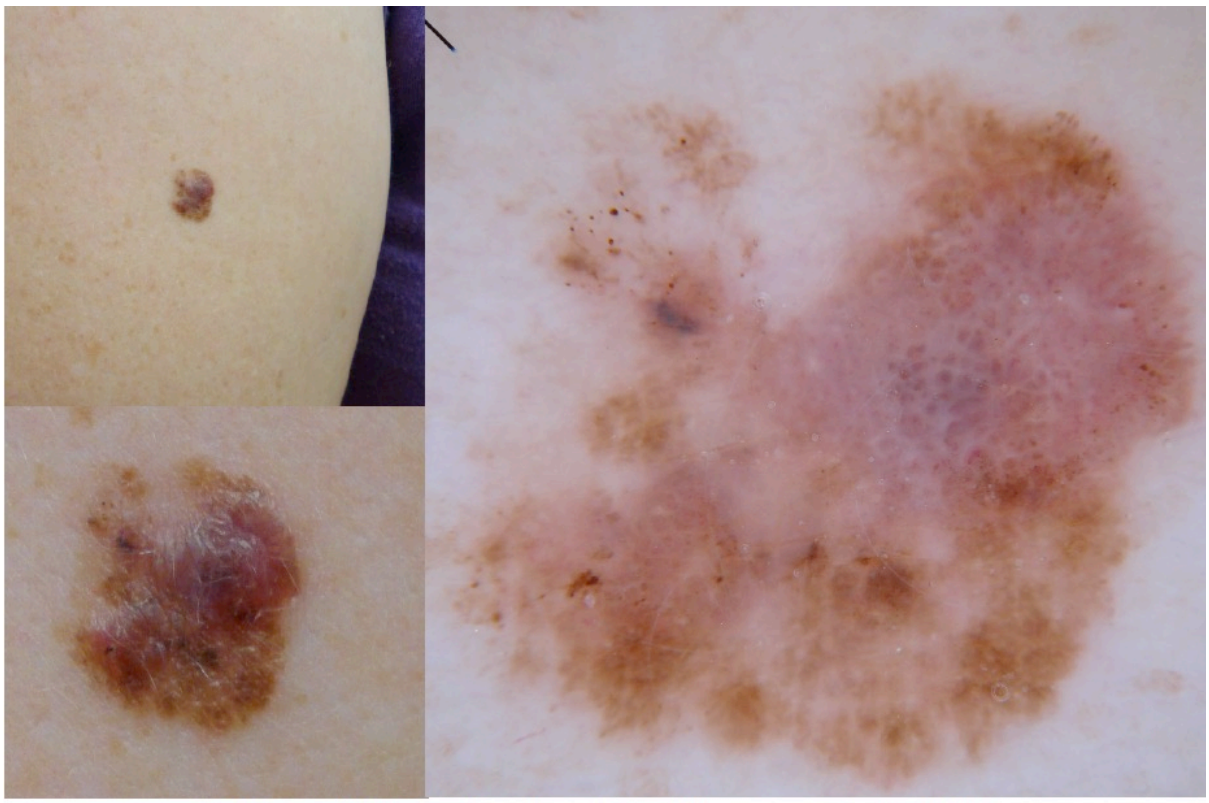
Provisional Diagnosis

- Solar Lentigo

Management

- None

Case 12.



CASE 12. Female 28 lesion over biceps longstanding recently traumatised, palpable but not firm/ hard.

Clinical Description

- Pigmented papule
- Irregular Border
- Assymmetrical
- Irregular colour pattern

ABCD (EFG) Criteria

- Assymmetrical
- Irregular Border
- Irregular Colour
- Diameter – Not Growing
- Evolving? no

Three Point Checklist Score

- Assymmetrical
  - Yes
- Blue white structures
  - Yes
- Atypical Network
  - yes

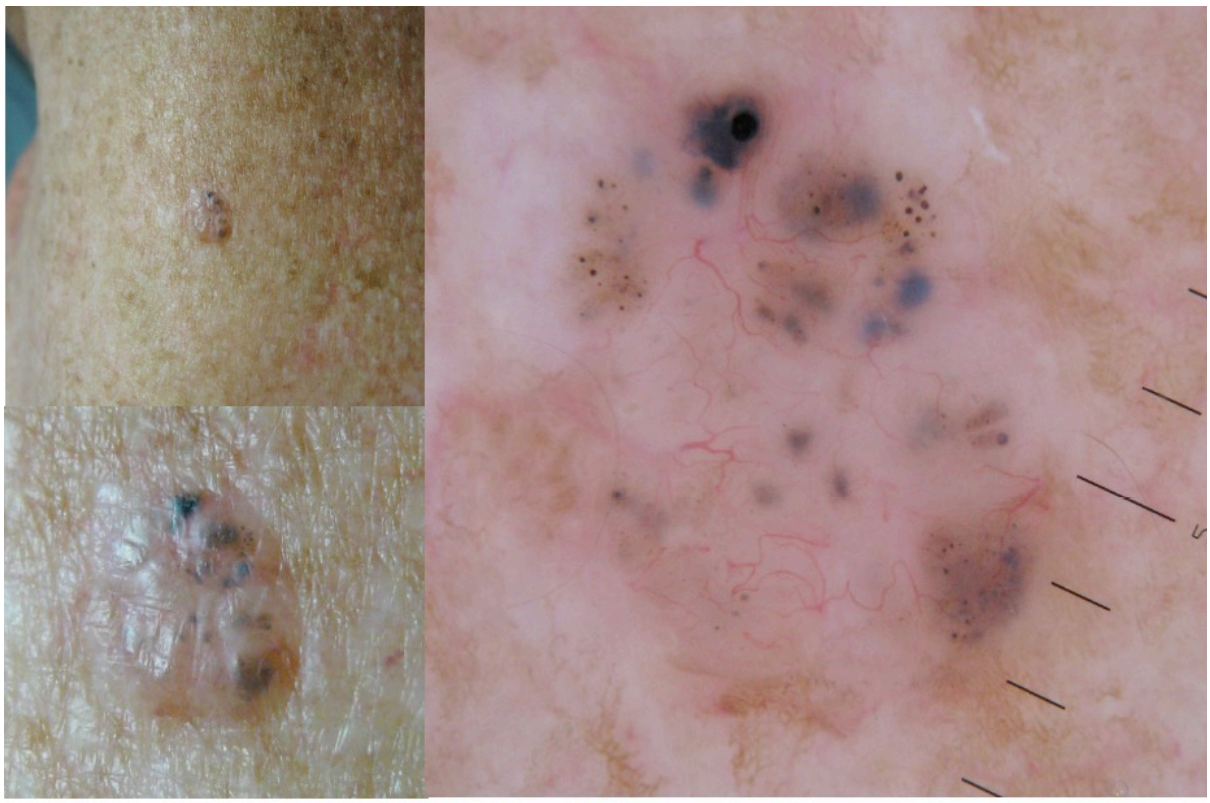
Provisional Diagnosis

- Melanoma

Management:

- Excision

Case 13.



CASE 13. Male 75 nodular lesion over supraspinatous.

Clinical Description:

- Telangiectasia

ABCD (EFG) Criteria:

- Asymmetrical
- Ill defined border
- Irregular colours
- Diameter – 5mm
- Evolving - unknown

Three Point Checklist Score

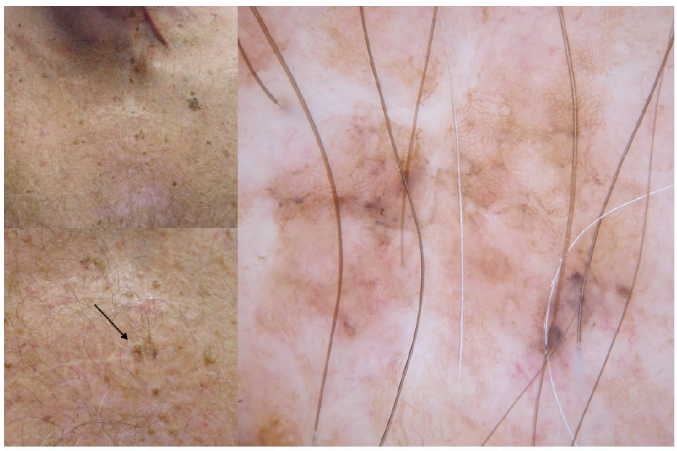
- Assymmetrical:
  - Yes
- Blue White Structures?
  - Yes
- Irregular network?
  - no

Provisional Diagnosis:

- BCC

Management

Case 14.



CASE 15. Male 62 pigmented macule near scar from previous BCC excision.

Clinical Description

- Ugly duckling
- Poorly defined border
-

ABCD (EFG) Criteria

Three Point Checklist Score

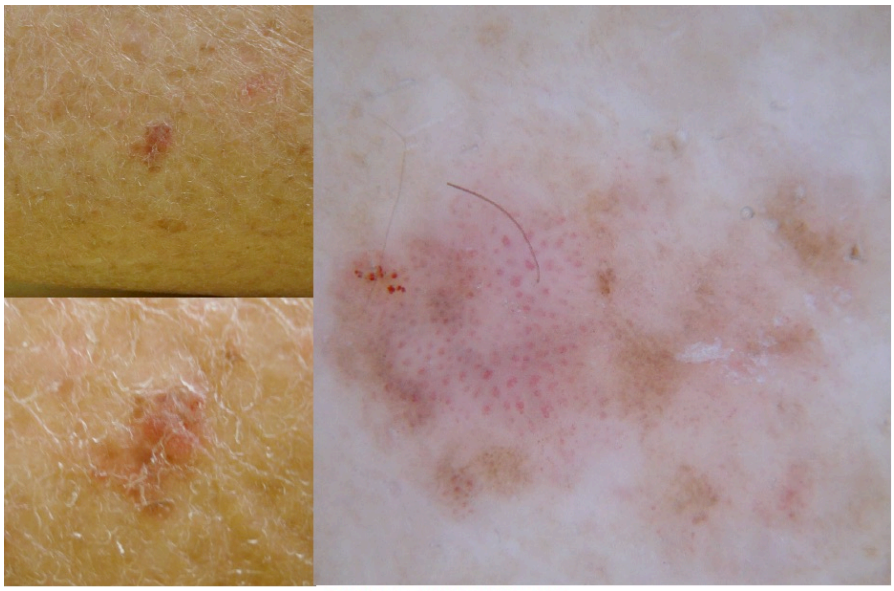
- Irregular Netowrk
    - yes

Provisional Diagnosis

- Invasive Melanoma

Management

Case 15.



CASE 16. Female 67 scaly plaque on leg.

Clinical Description:

- Diffuse,
- Assymmetrical
- Pigmented lesion

ABCD (EFG) Criteria

Three Point Checklist Score

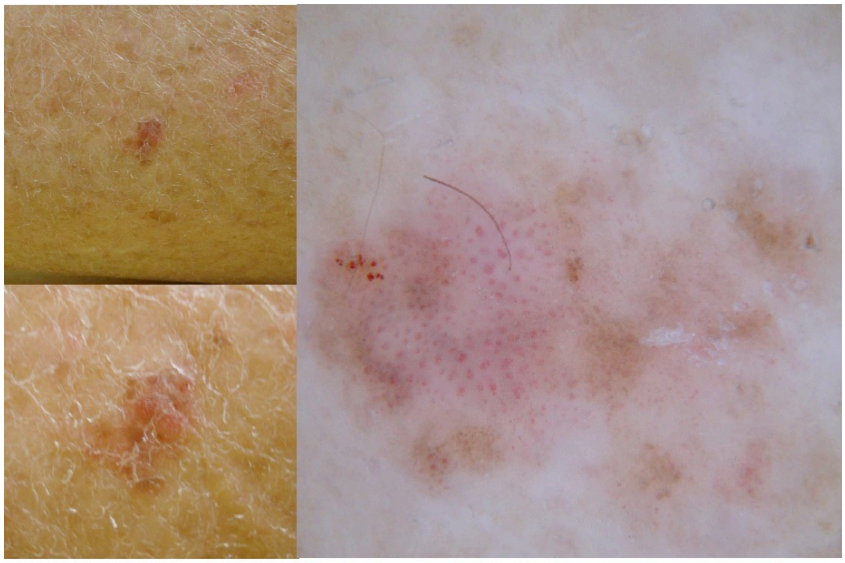
- Assymmetrical
- Irregular network
- White structures

Provisional Diagnosis:

- Melanoma Insitu

Management

Case 16.



CASE 16. Female 67 scaly plaque on leg.

Clinical Description:

- Glomerular Vessels

ABCD (EFG) Criteria

Three Point Checklist Score:

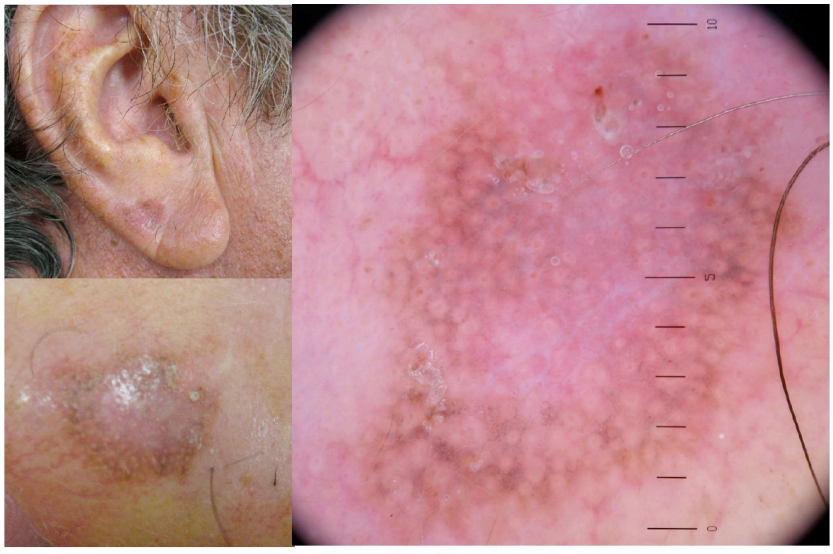
- Assymmetry

Provisional Diagnosis:

- Bowens Disease (Intraepidermal Carcinoma)

Management

Case 17.



CASE 17. Male 68, lesion on the ear, history unknown, scaly macule.

Clinical Description

ABCD (EFG) Criteria

Three Point Checklist Score:

- Irregular network
- Assymmetrical
- Inverse pigment Network

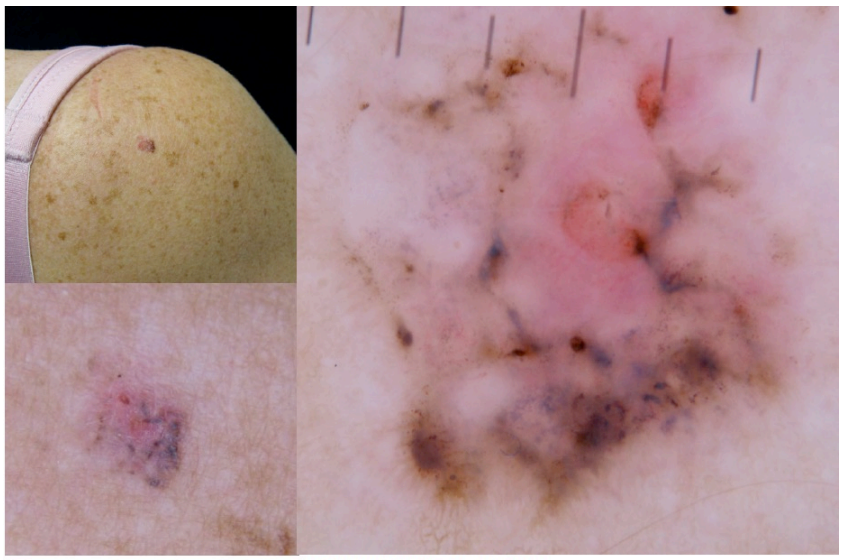
Provisional Diagnosis:

- Melanoma

Management:

- Excision

Case 18.



CASE 18. Female 39 lesion over shoulder.

Clinical Description

- Fine telangiectasia

ABCD (EFG) Criteria

Three Point Checklist Score

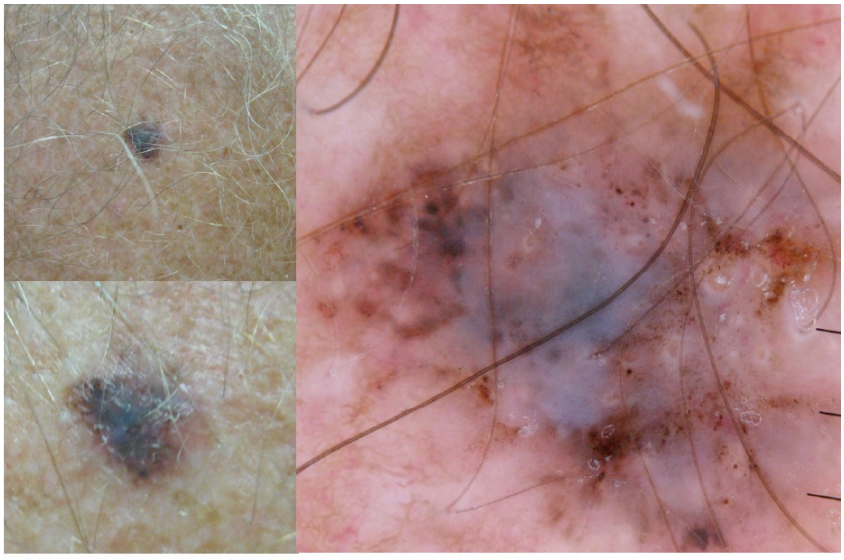
- Assymmetrical
- Blue white structures

Provisional Diagnosis:

- BCC

Management

Case 19.



CASE 19. Male 62 lesion upper back, history unkonwn.

Clinical Description

ABCD (EFG) Criteria

Three Point Checklist Score

- Assymmetrical
- Blue white structures
- Irregular Pigment Network

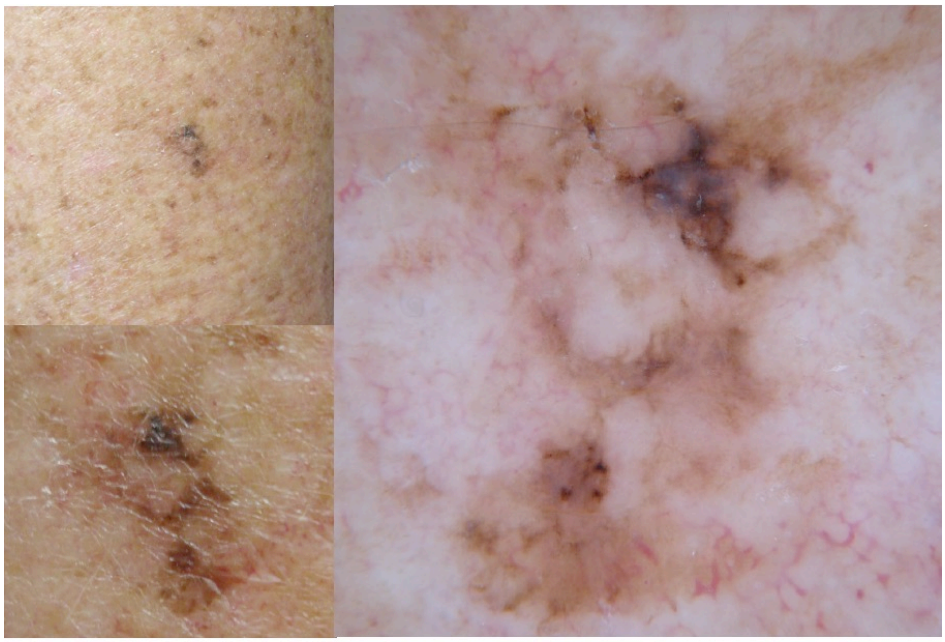
Provisional Diagnosis

- Melanoma

Management:

- Excision

Case 20.



CASE 20. Female 61 pigmented macule triceps.

Clinical Description
ABCD (EFG) Criteria
Three Point Checklist Score:

- Assymmetrical
- White Structures

Provisional Diagnosis:

- Melanoma

Management

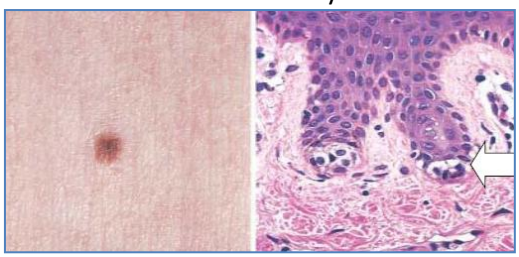
<u>Conclusion – You should be able to describe lesions in depth. Not expected to make diagnosis:</u>

- Where the lesion is
- Shape
- Size
- Colours
- Type of lesion (macule/papule/etc)

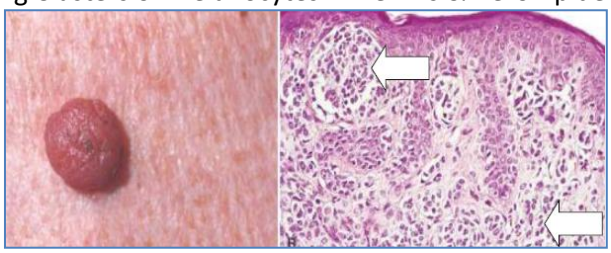
PATH
Pre-Malignant & Malignant Skin Lesions

BENIGN:

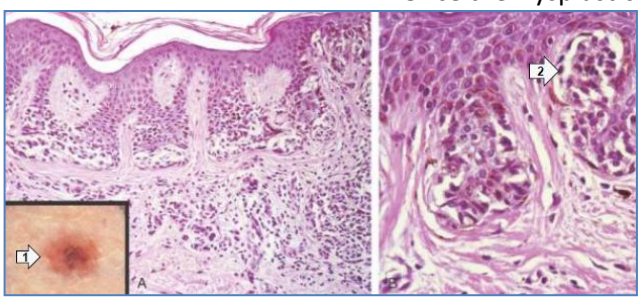
- **Nevi:**
  - **Aetiology:**
    - Congenital (After Birth)
  - **Pathogenesis:**
    - **Freckles =** Excess Pigment
    - **Moles (Lentigo) =** Excess Melanocytes
    - **Junctional Naevus (Typical Birthmark) =** Excess Melanocytes *AND* Excess Pigment
      1. Small, Flat, Symmetric, Uniform Lesions
      2. Cluster of Clear Cells (Melanocytes) @ the Dermo-Epidermal Junction
      3. NB: Proliferation below Basal Layer.

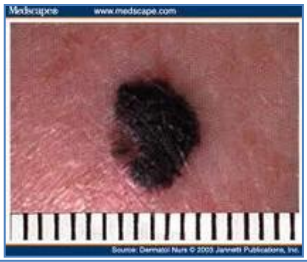


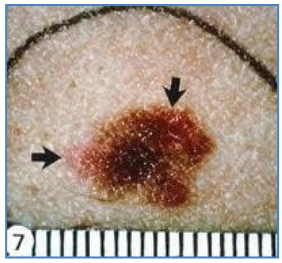
    - **Compound Nevus:**
      1. Small, Raised, Dome-Shaped, Symmetric, Uniform Lesions.
      2. Big Clusters of Melanocytes in Dermis & Dero-Epidermal Junction.

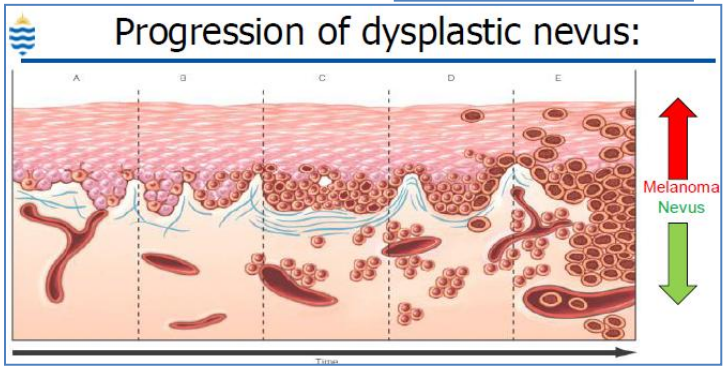


    - **Dysplastic Nevus:**
      1. Pigmented, Raised Lesion, with Central Darker Shade.
      2. DE-Junctional Cluster of Dysplastic (Larger/Irregular/Darker) Melanocytes.
      3. **NB: Can Progress to Melanoma:**
         1. Once the Dysplastic Melanocytes begin Infiltrating UP into the Epidermis.

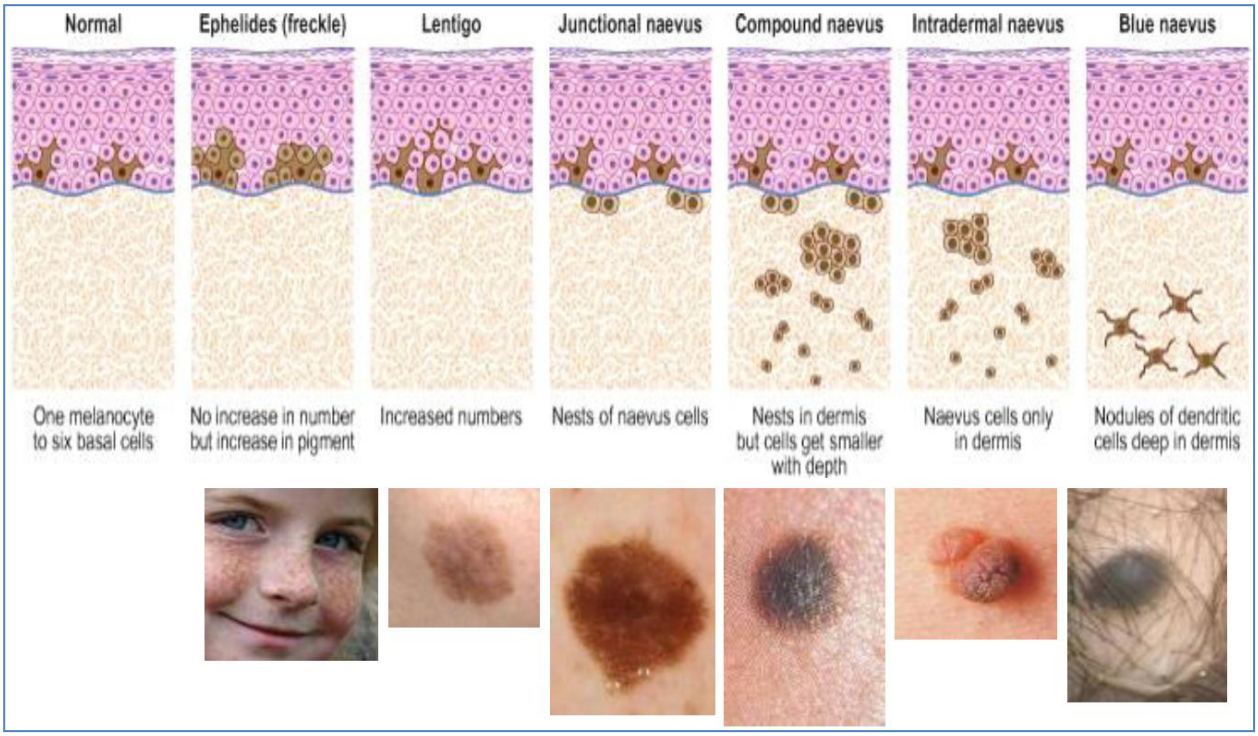




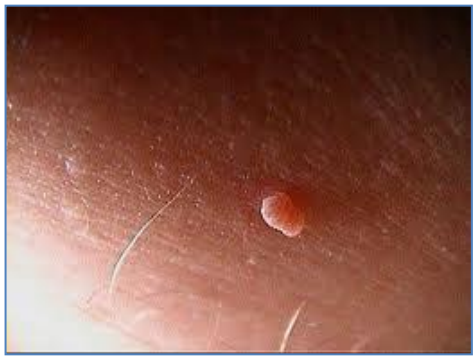


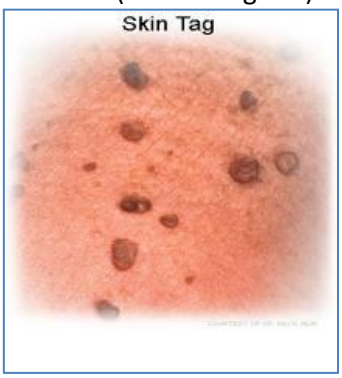


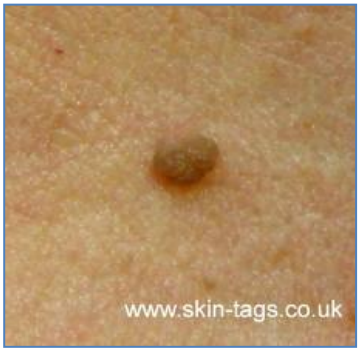
- **Clinical Significance (List 3x Clinical Features):**
    - Usually Benign (But can become malignant)



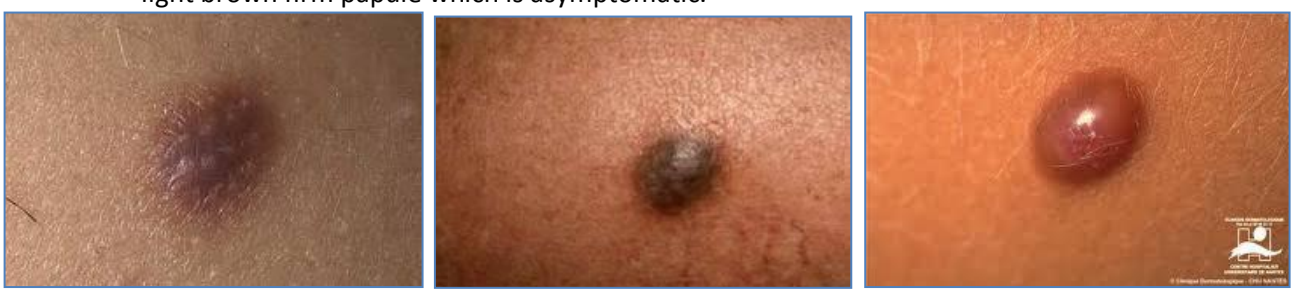
- **<u>Skin Tags:</u>**
    - These harmless lesions
    - They are pedunculated and have a narrow pedicle. They are usually skin coloured to brown. Common sites are the intertriginous areas (axilla and groin) and the neck.



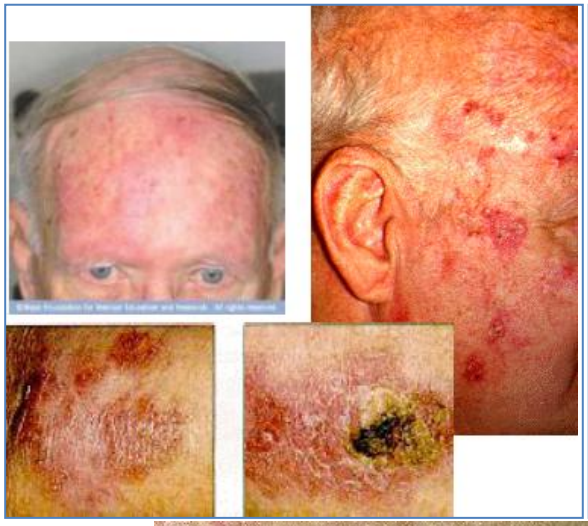


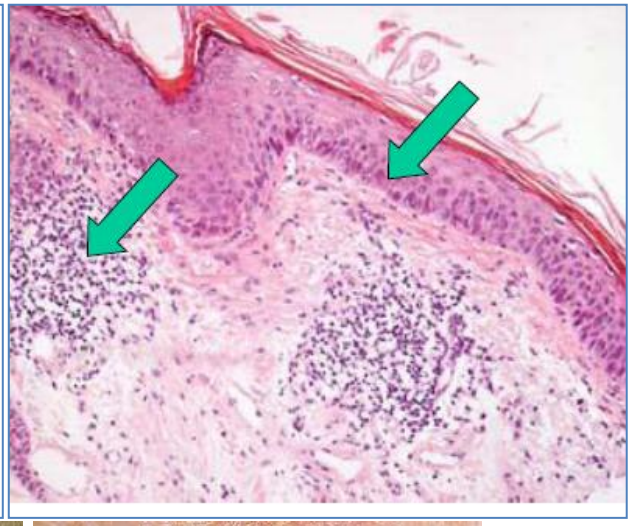


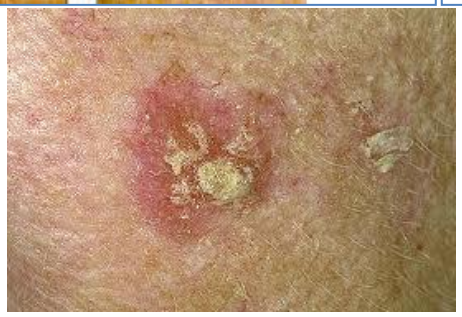
- **<u>Dermatofibromas</u>**:
    - This is a harmless and common lesion found in adults and children. It presents as a skin coloured to light brown firm papule which is asymptomatic.

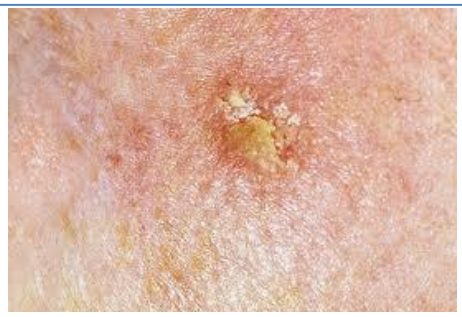


- **Actinic Keratosis (Sun Damage):**
  - **Aetiology:**
    - Sun Damage
  - **Pathogenesis:**
    - Sun Damage due to Chronic Exposure to UV Radiation
  - **Morphology (List 3x Gross & 3x Microscopic Features):**
    - **Gross:**
      1. Red/Tan, Irregular, Scaly Plaques
      2. Hyperkeratosis
      3. Inflammation/Ulceration/Crusting
    - **Microscopic:**
      1. Dysplasia (Pre-Cancerous) & Atrophy
      2. Inflammation
      3. Hyperkeratosis *AND* Parakeratosis
      4. Loss of Papillary Dermis & Rete Ridges
  - **Clinical Significance (List 3x Clinical Features):**
    - A Pre-Cancerous Skin-Growth



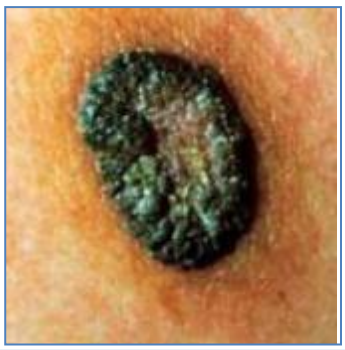
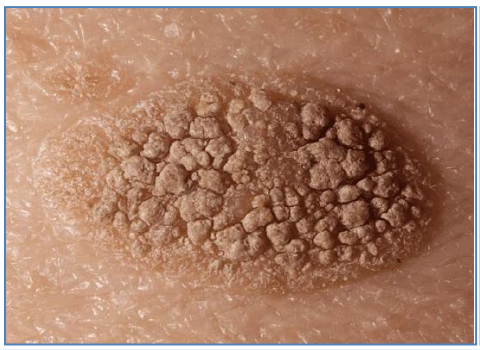
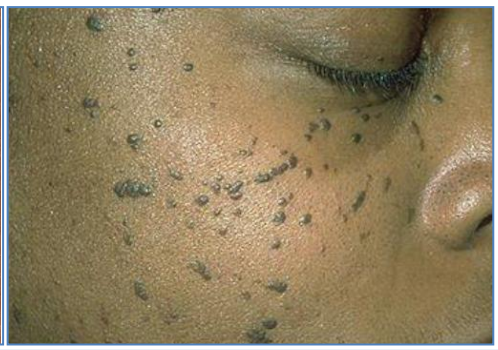
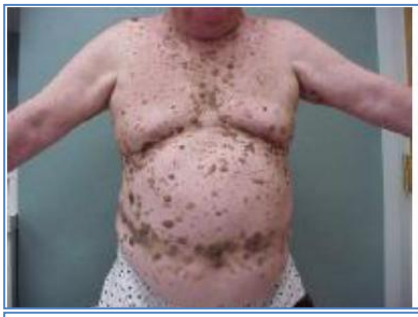
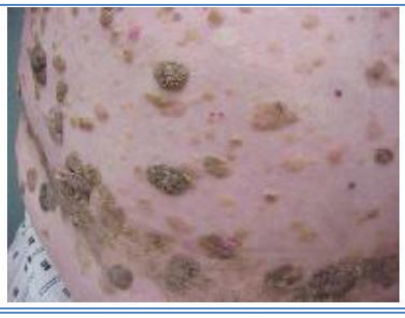
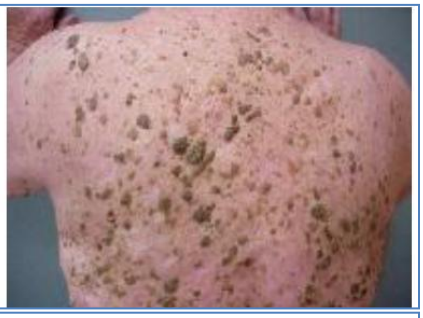
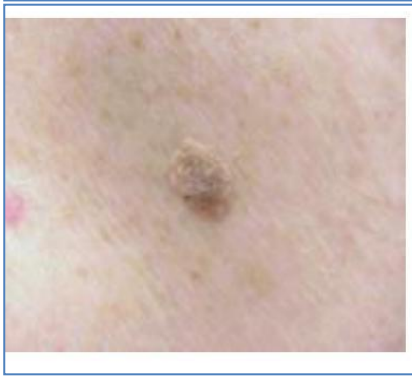
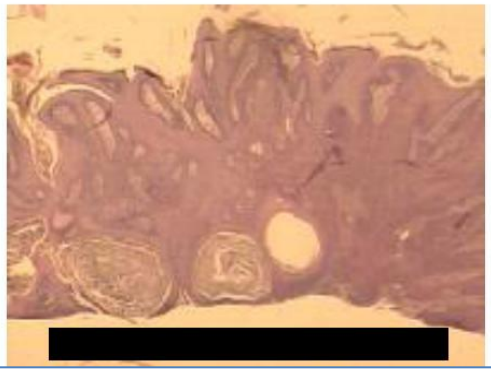
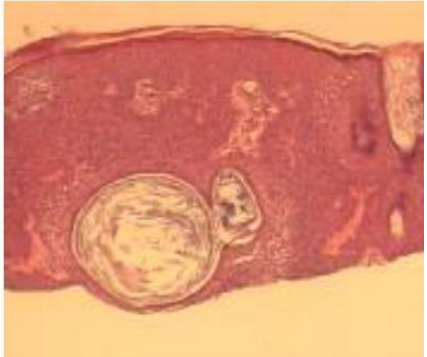






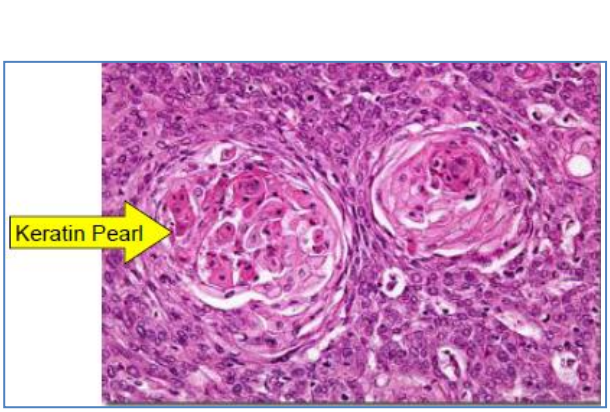
- **Seborrheic Keratosis:**
  - **Aetiology:**
    - Old Age
  - **Pathogenesis:**
    - Unknown
    - Totally Benign
  - **Morphology (List 3x Gross & 3x Microscopic Features):**
    - **Gross:**
      1. Sticky, Oily Plaques
      2. Round, Flat, Velvety Plaques
      3. May be Pigmented
      4. rough, warty surface
      5. Raised, 'Stuck-on' Appearance
    - **Microscopic:**
      1. Thick Hyperplastic Epidermis
      2. Keratin Cysts

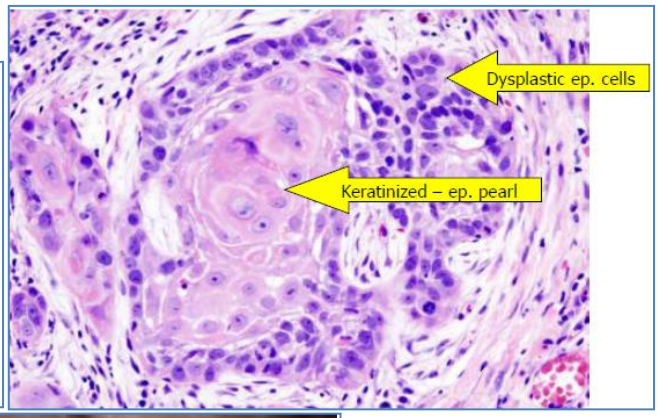
  - **Clinical Significance (List 3x Clinical Features):**
    - Treated only if inflamed
    - No Malignant Potential

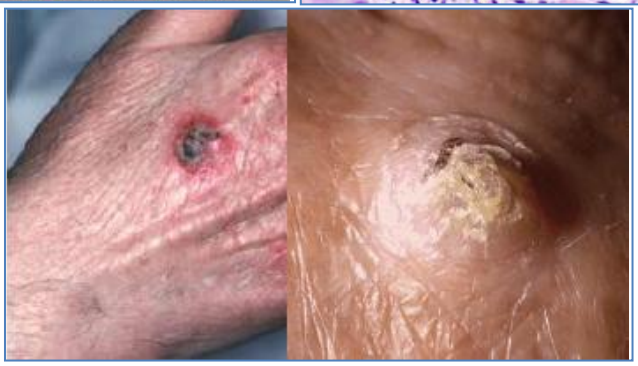










MALIGNANT:

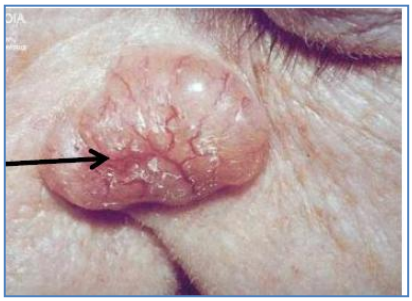
- **Squamous Cell Carcinoma (SCC):**
  - **Aetiology:**
    - Sun Damage
    - Industrial Carcinogens
    - Tobacco
  - **Pathogenesis:**
    - Dysplasia & Tumorigenesis of Squamous Epithelial Cells.
  - **Morphology (List 3x Gross & 3x Microscopic Features):**
    - **Gross:**
      1. Nodule
      2. Hyperkeratosis
      3. Sometimes Erythematous base.
    - **Microscopy:**
      1. Dyskeratosis
      2. Epithelial Keratin Pearls
  - **Clinical Significance (List 3x Clinical Features):**
    - Malignant (Early Treatment via Excision is Essential)
  - **Treatment:**
    - Wide excision
    - Radiotherapy in Elderly

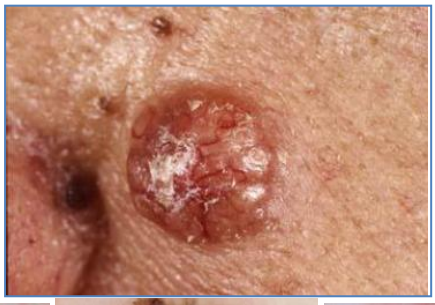


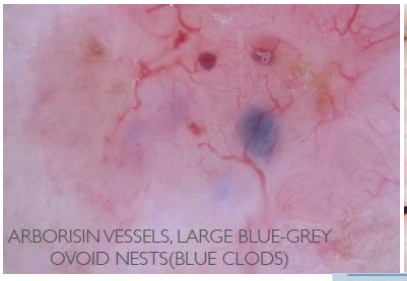


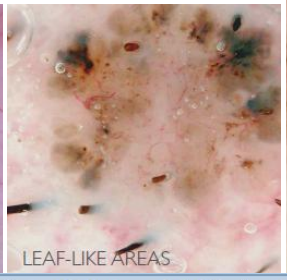


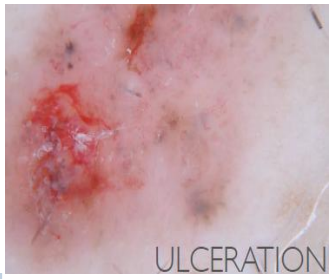
- **Basal Cell Carcinoma (BCC):**
  - **Aetiology:**
    - Sun Exposure
  - **Pathogenesis:**
    - Dysplasia & Tumorigenesis of Basal Epithelial Cells.
    - Slow Growing
    - Rarely Metastasises (Locally Infiltrative, but don't often Metastasise)
  - **Morphology (List 3x Gross & 3x Microscopic Features):**
    - **Gross:**
      1. Nodular/Papular
      2. Blood Vessels (telangiectasia)
      3. Pearly/Shiny crust over the lesion.
      4. Ulcerations.
    - **Microscopy:**
      1. Basal Cell Proliferation (Infiltrates the Dermis)
      2. Malignant Squamous Cells extend deep, Penetrating the Basement Membrane.
      3. Presence of Squamous Eddies or "Epithelial Pearls"
  - **Clinical Significance (List 3x Clinical Features):**
  - **Treatment:**
    - **Excisional surgery** : Still the gold standard treatment of BCC's.
    - Liquid nitrogen cryotherapy
    - Curettage and Cautery

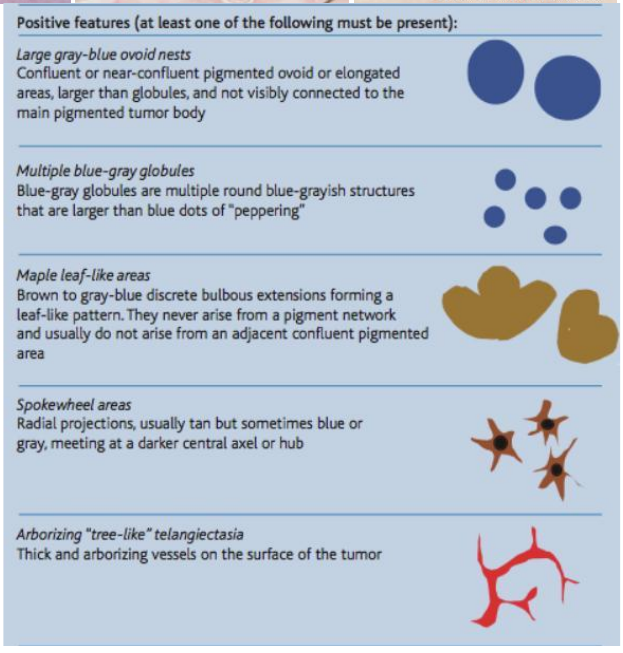




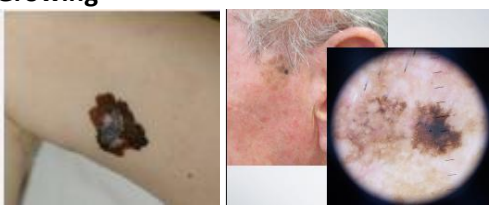






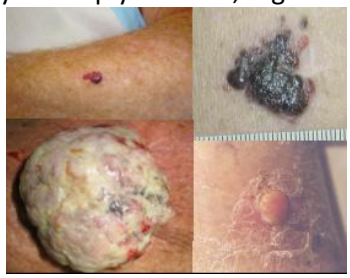


- **Melanoma:**
  - **Aetiology:**
    - Sun Damage
    - Congenital
  - **Risk Factors:**
    - Family history
    - Fair complexion
    - Freckles
    - Tendency to burn (Fitzpatrick type I/II skin)
    - Severe Childhood Sunburn (Solar Skin Damage)
    - Immunosuppression
    - High number of common acquired naevi
  - **Pathogenesis:**
    - Tumorigenesis of Melanocytes in an existing Naevus
  - **Types of Melanoma:**
    - **"*Superficial Spreading Malignant Melanomas*"** - linked to intermittent intense sun exposure (sunburns)
      1. Presents with a flat, usually pigmented, asymmetric macule that is changing is size, shape or colour
      2. **Slow Growing**



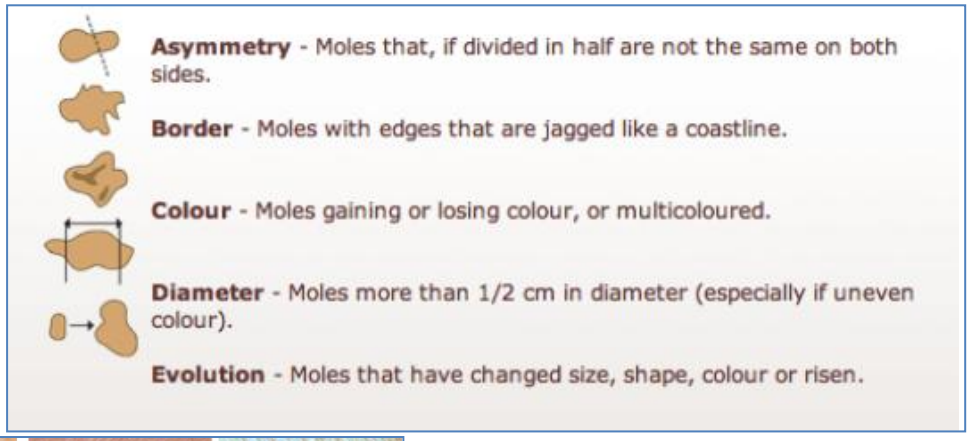
    - **"*Lentigo maligna melanoma*"** – linked to high doses of cumulative sun exposure
      1. presents as an unevenly pigmented, asymmetric facial freckle that is changing in size, shape or colour

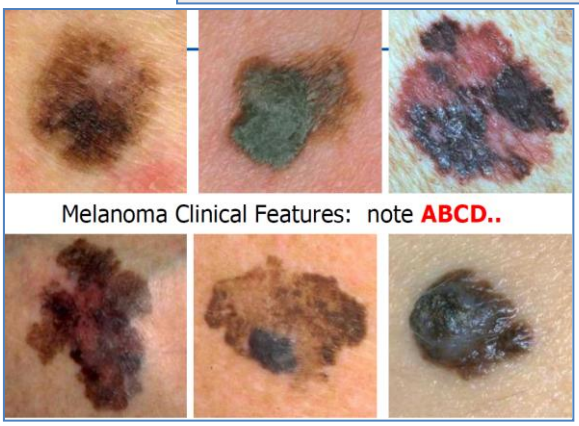
    - **"*Nodular Melanoma*" –** Due to Medium Sun Exposure
      1. Tends to grow more rapidly in depth than other melanomas.
      2. The majority of deeply invasive, High-risk Melanomas

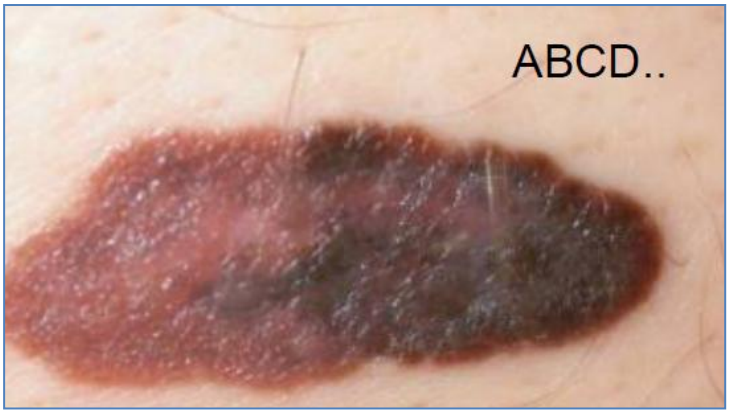


    - **"*Lentigo Melanoma*" –** On Skin of Face & Neck
    - **"*Acral Lentiginous Melanoma*"** – NOT due to sun exposure.
      1. Affects soles of the feet, palms of the hands, toes, and fingers, and occurs in the nail apparatus.
    - "***Amelanotic Melanoma***"- Red Patches on the Skin
      1. Many melanomas are partially non-pigmented and up to 20% are almost completely without pigmentation. Amelanotic melanomas generally present as a red, changing lesion though they can be skin coloured.

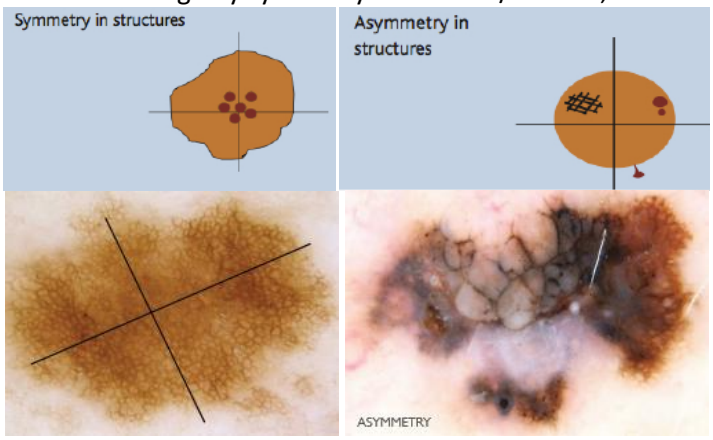
- **Morphology (List 3x Gross & 3x Microscopic Features):**
  - **Gross:**
    1. Grow & Change Colour
    2. Maculo-Papular Lesion
    3. ***Irregular Shape, Colour & Depth – Distinguishing Features.***
    4. Pigmented
  - **Microscopic:**
    1. **Diagnostic Feature: Atypical (Dysplastic) Melanocytes *ABOVE The Epidermis***
       1. Also Invade down into the Dermis
    2. Nests/Clusters of Atypical Melanocytes
    3. Inflammatory Cells
    4. Mitotic Figures (Due to High Replication Rates)
- **Diagnosis:**
  - Dermoscopy is ESSENTIAL!!
  - Skin exam without dermoscopy will only pick 60% of melanomas.
  - How long has it been there?
  - **ABCD's of melanoma:**
    1. **A – Asymmetry o**f Shape and Structure.
       1. Ie. One half doesn't match the other half (border/colour/structure within the lesion).
    2. **B – Border Irregularity**. The edges are ragged, blotched, or blurred.
    3. **C – Colour Variability**. The pigmentation is not uniform. Shades of tan, brown, and black may be present and red, white, grey and blue may add to the mottled appearance.
    4. **D – Diameter Increasing**. A width >6mm (about the size of a pencil eraser).
       1. NB: Any growth of a mole should be of concern.
    5. **E –**
       1. **Evolving** - any lesion that is changing or enlarging
       2. **Elevated** - Different elevations/contours *OR* ANY change from flat to elevated is of concern.
    6. **F – Firm**. A lesion that is firm, and also ones that are friable (easily damaged) are more likely to be malignant. Whilst benign moles are raised, soft and "wobble" like jelly with pressure.
    7. **G – Growing**. Signs of growth (Slow/Rapid) should raise concern.



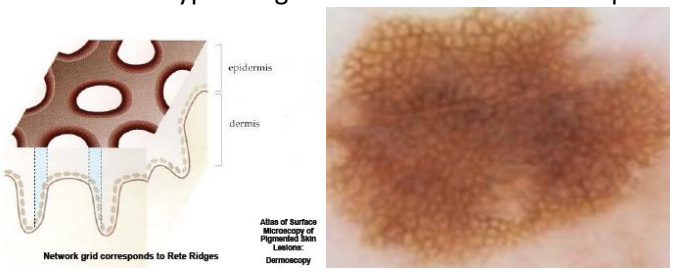




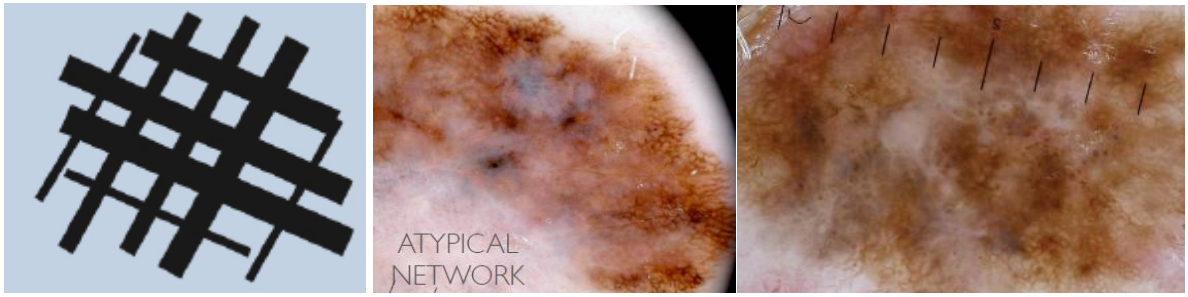
- **3 Point Checklist of Melanoma:**
  - (A Screening tool for Malignancy)
  - High Sensitivity, But Low Specifictiy (Many benign lesions score higher than 1)
  - **1. Asymmetry of Colour and /or Structure**
    - Judge by symmetry of Structre/Pattern, not outline.



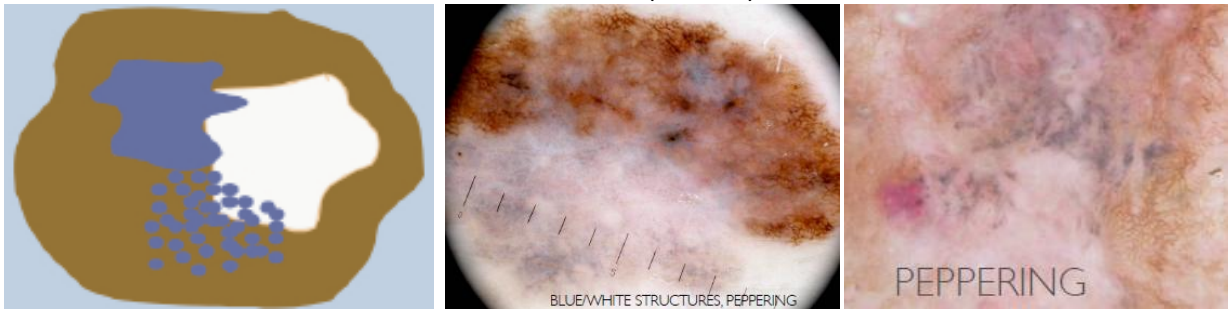
  - **2. Atypical Pigment Network:**
    - NB: Typical Pigment Network Grids correspond to Rete Ridges in the Dermis.



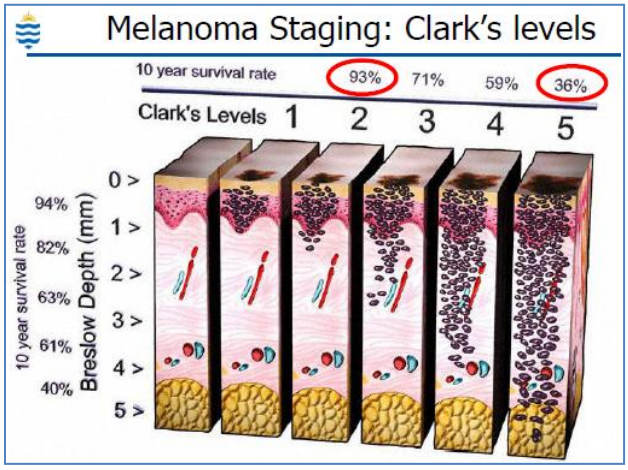
    - Atypical Networks = Lines are of different thickness & sizes of 'holes' are varied.



  - **3. Blue-White Structures:**
    - Any White and/or Blue colour present in the lesions.
    - Include:
      - 'Peppering' (Melanophages)
      - Blue-white Veil (Orthokeratotic Hyperkeratosis overlying the Pigmented Tumour)
      - Whitish Scar (Fibrosis)



- **Prognostic Factors:**
  - **Poor Prognostic Factors:**
    1. Ulceration
    2. High Mitotic Rate
    3. Signs of Regression = Bad
  - **TNM Staging:**
    1. **T = Size/Depth**
    2. **N = Nodal Involvement**
    3. **M = Distant Metastasis?**
  - **Breslow Thickness:**
    1. Tumour thickness measured from the granular layer of the epidermis to the deepest identified tumour cell
    2. Best Available predictor of Prognosis
  - **Clark Levels:**
    1. Relateds to the Deepest invasive tumour cells
    2. Levels 1 – 5
    3. (Not the same as TNM Staging)



NB: Just realise that despite subtle changes in appearance, the *actual* size and infiltration of the Melanoma can be quite significant.