Dermatology

A handbook for medical students & junior doctors

This publication is supported by the British Association of Dermatologists.

First edition 2009

Revised first edition 2009

Second edition 2014

Third edition 2020

For comments and feedback, please contact the author at drnicoledermatology@gmail.com

Dermatology

A handbook for medical students & junior doctors

Dr Nicole Yi Zhen Chiang MBChB (Hons), MRCP (UK), MRCP(UK)(Derm)
Consultant Dermatologist
Manchester University Hospitals NHS Trust
Withington Hospital M20 2LR
Manchester

Professor Julian Verbov JP MD FRCP FRCPCH CBiol FRSB FLS MCSFS
Professor of Dermatology
Consultant Paediatric Dermatologist
Alder Hey Children's Hospital
East Prescot Road
Liverpool L14 5AB

Contents

Preface to the 3rd edition

11 years have passed since this Handbook first appeared. It has proved immensely popular and it has been further updated. We hope that it will continue to be a valuable source book for those interested in learning about this exciting specialty. The Handbook was designed to be an overview, both succinct and reader-friendly which continues to be our aim.

Once again, many thanks to the BAD for its essential and continuing support.

Julian Verbov

Professor of Dermatology Liverpool 2020

Foreword to Third edition

Past BAD President Dr Mark Goodfield wrote in the first edition:
'There is a real need for appropriate information about dermatological diseases to meet the educational needs of doctors at all levels.'

This holds true even more today than in 2009 with the exponential use of social media as an information sourced by patients and clinicians alike. Since its first publication, this book has been the go to resource for accurate knowledge in common and urgent dermatological problems. Its essential role in supporting their workplace learning is highlighted by the 8,843 downloads and 50,000 requests for hard copies from individual students and medical schools throughout the UK. Starting with scientific and epidemiological facts, moving through clinical features and management, medical students are given a structure that enables them to organise learning effectively. The content remains focused on learning at the undergraduate stage of the medical education spectrum: a vital foundation for postgraduate training in dermatology.

The UK population has become increasingly diverse over the last few decades, it is therefore necessary to update the handbook to highlight tips for assessment, variation in presentation in common and important skin conditions (e.g. common pigmentary disorders) that reflect the spectrum of cutaneous diversity junior clinicians will encounter in their practice. This, in combination, with other BAD resources under current development will ensure that medical students continue to learn from the highest quality education in dermatology to the benefit of our patients.

Dr Tanya Bleiker
President of the British Association of Dermatologists

Prof Mini Singh
Undergraduate Work Stream Chair, British Association of Dermatologists

What is dermatology?

- Dermatology is the study of both normal and abnormal skin and associated structures such as hair, nails, and oral and genital mucous membranes.

Why is dermatology important?

- Skin diseases are very common, affecting up to a third of the population at any one time.

- Skin diseases have serious impacts on life. They can cause physical damage, embarrassment, and social and occupational restrictions. Chronic skin diseases may cause financial constraints with repeated sick leave. Some skin conditions can be life-threatening.

- In 2006-07, the total NHS health expenditure for skin diseases was estimated to be around £97 million (approximately 2% of the total NHS health expenditure).

What is this handbook about?

- The British Association of Dermatologists outlined the essential and important learning outcomes that should be achieved by **all** medical undergraduates for the competent assessment of patients presenting with skin disorders *(available on:* https://www.bad.org.uk/shared/get-file.ashx?itemtype=document&id=4168)

- This handbook addresses these learning outcomes and aims to equip you with the knowledge and skills to practise competently and safely as a junior doctor.

Essential Clinical Skills

- Detailed history taking and examination provide important diagnostic clues in the assessment of skin problems.

Learning outcomes:

1. **Ability to take a dermatological history**
2. **Ability to explore a patient's concerns and expectations**
3. **Ability to interact sensitively with people with skin disease**
4. **Ability to examine skin, hair, nails and mucous membranes systematically showing respect for the patient**
5. **Ability to describe physical signs in skin, hair, nails and mucosa**
6. **Ability to record findings accurately in patient's records**

Taking a dermatological history

- Using the standard structure of history taking, below are the important points to consider when taking a history from a patient with a skin problem (Table 1).
- For dark lesions or moles, pay attention to questions marked with an asterisk (*).

Table 1. Taking a dermatological history

Main headings	Key questions
Presenting complaint	Nature, site and duration of problem
History of presenting complaint	Initial appearance and evolution of lesion*
	Symptoms (particularly itch and pain)*
	Aggravating and relieving factors
	Previous and current treatments (effective or not)
	Recent contact, stressful events, illness and travel
	History of sunburn and use of tanning machines*
	Skin type *(see page 70)**
Past medical history	History of atopy i.e. asthma, allergic rhinitis, eczema
	History of skin cancer and suspicious skin lesions
Family history	Family history of skin disease*
Social history	Occupation (including skin contacts at work)
	Improvement of lesions when away from work

Medication and allergies	Regular, recent and over-the-counter medications
Impact on quality of life	Impact of skin condition and concerns

Examining the skin

- There are four important principles in performing a good examination of the skin: **INSPECT, DESCRIBE, PALPATE** and **SYSTEMATIC CHECK** (Table 2).

Table 2. Examining the skin

Main principles	Key features
INSPECT *in general*	General observation Note if richly pigmented skin therefore signs of skin changes may be different (e.g. erythema not as obvious Site and number of lesion(s) *If multiple*, pattern of distribution and configuration
DESCRIBE *the individual lesion*	**SCAM** **S**ize (the widest diameter), **S**hape **C**olour **A**ssociated secondary change **M**orphology, **M**argin (border)
If the lesion is pigmented, remember* **ABCDE *(the presence of any of these features increase the likelihood of melanoma):*	**A**symmetry (lack of mirror image in any of the four quadrants) Irregular **B**order Two or more **C**olours within the lesion **D**iameter > 6mm **E**volution (history of change in size, shape or colour)
PALPATE* *the individual lesion*	Surface

	Consistency Mobility Tenderness Temperature **** Essential in richly pigmented skin to accurately classify lesions***
SYSTEMATIC CHECK	Examine the nails, scalp, hair & mucous membranes General examination of all systems relevant to presenting symptoms

Communicating examination findings

- In order to describe, record and communicate examination findings accurately, it is important to learn the appropriate terminology (Tables 3-10).

Table 3. General terms

Terms	Meaning
Pruritus	Itching
Lesion	An area of altered skin
Rash	An eruption
Naevus	A localised malformation of tissue structures Example: (*Picture Source: D@nderm)*  Pigmented melanocytic naevus (mole)
Comedone	A plug in a sebaceous follicle containing altered sebum, bacteria and cellular debris; can present as either open (blackheads) or closed (whiteheads) Example:

Open comedones (left) and closed comedones (right) in acne

Table 4. Distribution (the pattern of spread of lesions)

Terms	Meaning
Generalised	All over the body
Widespread	Extensive
Localised	Restricted to one area of skin only
Flexural	Body folds i.e. groin, neck, behind ears, popliteal and antecubital fossa
Extensor	Knees, elbows, shins
Pressure areas	Sacrum, buttocks, ankles, heels
Dermatome	An area of skin supplied by a single spinal nerve
Photosensitive	Affects sun-exposed areas such as face, neck and back of hands Example:  Sunburn
Köebner phenomenon	A linear eruption arising at site of trauma Example:

Psoriasis

Table 5. Configuration (the pattern or shape of grouped lesions)

Terms	Meaning
Discrete	Individual lesions separated from each other
Confluent	Lesions merging together
Linear	In a line
Target	Concentric rings (like a dartboard) Example:  Erythema multiforme
Annular	Like a circle or ring Example:  Tinea corporis ('ringworm')

Discoid / Nummular	A coin-shaped/round lesion Example: Discoid eczema

Hypopigmentation of discoid lupus

Table 6. Colour

Terms	Meaning
Erythema	Redness (due to inflammation and vasodilatation) which blanches on pressure Example:

Palmar erythema

Purpura	Red or purple colour (due to bleeding into the skin or mucous membrane) which does not blanch on pressure – petechiae (small pinpoint macules) and ecchymoses (larger bruise-like patches) Example:

Henoch-Schönlein purpura
(palpable small vessel vasculitis)

Hypo-pigmentation Area(s) of paler skin

Examples:

Pityriasis versicolor
(a superficial fungus infection)

De-pigmentation: White skin due to absence of melanin

Examples:

Vitiligo
(loss of skin melanocytes)

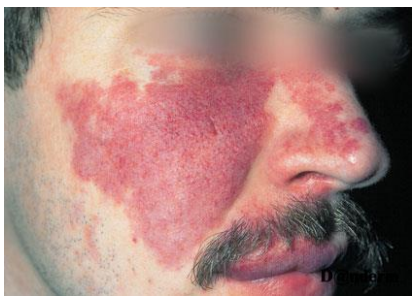
Note the three colours 'tricolor' pattern typical of vitiligo.

Hyper-pigmentation Darker skin which may be due to various causes (e.g. post-inflammatory, melasma, naevi)

Melasma (increased melanin pigmentation)

Examples:

Table 7. Morphology (the structure of a lesion) – **Primary lesions**

Terms	Meaning
Macule	A flat area of altered colour Example:  Freckles
Patch	Larger flat area of altered colour or texture Example:  Vascular malformation (naevus flammeus / 'port wine stain')

Papule Solid raised lesion < 0.5cm in diameter

Example:

Xanthomata

Nodule Solid raised lesion >0.5cm in diameter with a deeper component

Example: (Picture source: D@nderm)

Pyogenic granuloma
(granuloma telangiectaticum)

Plaque Palpable scaling raised lesion >0.5cm in diameter

Example:

Psoriasis

Vesicle
(small blister) Raised, clear fluid-filled lesion <0.5cm in diameter

Example:

Acute hand eczema
(pompholyx)

Bulla
(large blister)

Raised, clear fluid-filled lesion >0.5cm in diameter

Example:

Reaction to insect bites

Pustule

Pus-containing lesion <0.5cm in diameter

Example:

Acne

Abscess

Localised accumulation of pus in the dermis or subcutaneous tissues

Example:

Periungual abscess
(acute paronychia)

W(h)eal

Transient raised lesion due to dermal oedema

Examples: Urticaria

Note how subtle the erythema is in this wheal in patient with skin type V.

Boil/Furuncle Staphylococcal infection around or within a hair follicle

Carbuncle Staphylococcal infection of adjacent hair follicles (multiple boils/furuncles)

Table 8. Morphology - Secondary lesions (lesions that evolve from primary lesions)

Terms	Meaning
Excoriation	Loss of epidermis following trauma Example:

Excoriations in eczema

Lichenification Well-defined roughening of skin with accentuation of skin markings

Examples:

Lichenification due to chronic rubbing in eczema

Lichenification in darker skin types: the clue is the increased appearance of skin lines at the bottom of this photograph.

Scales Flakes of stratum corneum

Example:

Psoriasis (showing silvery scales)

Crust Rough surface consisting of dried serum, blood, bacteria and cellular debris that has exuded through an eroded epidermis (e.g. from a burst blister)

Example:

Impetigo

Scar New fibrous tissue which occurs post-wound healing, and may be atrophic (thinning), hypertrophic (hyperproliferation within wound boundary), or keloidal (hyperproliferation beyond wound boundary)

Examples: Keloid scars

Ulcer Loss of epidermis and dermis (heals with scarring)

Example:

Leg ulcers

Fissure An epidermal crack often due to excess dryness

Example:

Eczema

Striae Linear areas which progress from purple to pink to white, with the histopathological appearance of a scar (associated with excessive steroid usage and glucocorticoid production, growth spurts and pregnancy)

Example:

Striae

Table 9. Hair

Terms	Meaning
Alopecia	Loss of hair Examples:

Alopecia areata (well-defined patch of complete hair loss) Scarring alopecia of the scalp © Cardiff and Vale University Health Board

Hirsutism Androgen-dependent hair growth in a female

Example:

Hirsutism

Hypertrichosis Non-androgen dependent pattern of excessive hair growth

(e.g. in pigmented naevi)

Example:

Hypertrichosis

Table 10. Nails

Terms	Meaning
Clubbing	Loss of angle between the posterior nail fold and nail plate (associations include suppurative lung disease, cyanotic heart disease, inflammatory bowel disease and idiopathic) Example: (*Picture source: D@nderm)*  Clubbing
Koilonychia	Spoon-shaped depression of the nail plate (associations include iron-deficiency anaemia, congenital and idiopathic) Example: (*Picture source: D@nderm)*  Koilonychia
Onycholysis	Separation of the distal end of the nail plate from nail bed (associations include trauma, psoriasis, fungal nail infection and hyperthyroidism) Example: (*Picture source: D@nderm)*  Onycholysis
Pitting	Punctate depressions of the nail plate (associations include psoriasis, eczema and alopecia areata) Example: (*Picture source: D@nderm)*  Pitting

Background Knowledge

- This section covers the basic knowledge of normal skin structure and function required to help understand how skin diseases occur.

Learning outcomes:

1. **Ability to describe the functions of normal skin**
2. **Ability to describe the structure of normal skin**
3. **Ability to describe the principles of wound healing**
4. **Ability to describe the difficulties, physical and psychological, that may be experienced by people with chronic skin disease**

Functions of normal skin

- These include:
    - i) Protective barrier against environmental insults
    - ii) Temperature regulation
    - iii) Sensation
    - iv) Vitamin D synthesis
    - v) Immunosurveillance
    - vi) Appearance/cosmesis

Structure of normal skin and the skin appendages

- The skin is the largest organ in the human body. It is composed of the epidermis and dermis overlying subcutaneous tissue. The skin appendages (structures formed by skin-derived cells) are hair, nails, sebaceous glands and sweat glands.

Epidermis

- The epidermis is composed of 4 major cell types, each with specific functions (Table 11).

Table 11. Main functions of each cell type in the epidermis

Cell types	Main functions
Keratinocytes	Produce keratin as a protective barrier
Langerhans' cells	Present antigens and activate T-lymphocytes for immune protection
Melanocytes	Produce melanin, which gives pigment to the skin and protects the cell nuclei from ultraviolet (UV) radiation-induced DNA damage
Merkel cells	Contain specialised nerve endings for sensation

- There are 4 layers in the epidermis (Table 12), each representing a different stage of maturation of the keratinocytes. The average epidermal turnover time (migration of cells from the basal cell layer to the horny layer) is about 30 days.

Table 12. Composition of each epidermal layer

Epidermal layers	Composition
Stratum basale *(Basal cell layer)*	Actively dividing cells, deepest layer
Stratum spinosum *(Prickle cell layer)*	Differentiating cells
Stratum granulosum *(Granular cell layer)*	So-called because cells lose their nuclei and contain granules of keratohyaline. They secrete lipid into the intercellular spaces.
Stratum corneum *(Horny layer)*	Layer of keratin, most superficial layer

- In areas of thick skin such as the sole, there is a fifth layer, stratum lucidum, beneath the stratum corneum. This consists of paler, compact keratin.

- Pathology of the epidermis may involve:
  a) changes in epidermal turnover time - e.g. psoriasis (reduced epidermal turnover time)
  b) changes in the surface of the skin or loss of epidermis - e.g. scales, crusting, exudate, ulcer
  c) changes in pigmentation of the skin - e.g. hypo- or hyper-pigmented skin

Dermis

- The dermis is made up of collagen (mainly), elastin and glycosaminoglycans, which are synthesised by fibroblasts. Collectively, they provide the dermis with strength and elasticity.
- The dermis also contains immune cells, nerves, skin appendages as well as lymphatic and blood vessels.
- Pathology of the dermis may involve:
    a) changes in the contour of the skin or loss of dermis e.g. formation of papules, nodules, skin atrophy and ulcers
    b) disorders of skin appendages e.g. disorders of hair, acne (disorder of sebaceous glands)
    c) changes related to lymphatic and blood vessels e.g. erythema (vasodilatation), urticaria (increased permeability of capillaries and small venules), purpura (capillary leakage)

Hair

- There are 3 main types of hair:
    a) lanugo hair (fine long hair in fetus)
    b) vellus hair (fine short hair on all body surfaces)
    c) terminal hair (coarse long hair on the scalp, eyebrows, eyelashes and pubic areas)
- Each hair consists of modified keratin and is divided into the hair shaft (a keratinized tube) and hair bulb (actively dividing cells, and melanocytes which give pigment to the hair).
- Each hair follicle enters its own growth cycle. This occurs in 3 main phases:
    a) anagen (long growing phase)
    b) catagen (short regressing phase)
    c) telogen (resting/shedding phase)
- Pathology of the hair may involve:
    a) reduced or absent melanin pigment production e.g. grey or white hair
    b) changes in duration of the growth cycle e.g. hair loss (premature entry of hair follicles into the telogen phase)
    c) shaft abnormalities

Nails

- The nail is made up of a nail plate (hard keratin) which arises from the nail matrix at the posterior nail fold, and rests on the nail bed.
- The nail bed contains blood capillaries which gives the pink colour of the nails.
- Pathology of the nail may involve:
    a) abnormalities of the nail matrix e.g. pits and ridges
    b) abnormalities of the nail bed e.g. splinter haemorrhage
    c) abnormalities of the nail plate e.g. discoloured nails, thickening of nails

Sebaceous glands

- Sebaceous glands produce sebum via hair follicles (collectively called a pilosebaceous unit). They secrete sebum onto the skin surface which lubricates and waterproofs the skin.
- Sebaceous glands are stimulated by the conversion of androgens to dihydrotestosterone and therefore become active at puberty.
- Pathology of sebaceous glands may involve:
    a) increased sebum production and bacterial colonisation e.g. acne
    b) sebaceous gland hyperplasia

Sweat glands

- Sweat glands regulate body temperature and are innervated by the sympathetic nervous system.
- They are divided into two types: eccrine and apocrine sweat glands.
- Eccrine sweat glands are universally distributed in the skin.
- Apocrine sweat glands are found in the axillae, areolae, genitalia and anus, and modified glands are found in the external auditory canal. They only function from puberty onwards and action of bacteria on the sweat produces body odour.
- Pathology of sweat glands may involve:
    a) inflammation/infection of apocrine glands e.g. hidradenitis suppurativa
    b) overactivity of eccrine glands e.g. hyperhidrosis

Principles of wound healing

- Wound healing occurs in 4 phases: haemostasis, inflammation, proliferation and remodelling (Table 13).

Table 13. Stages of wound healing

Stages of wound healing	Mechanisms
Haemostasis	● Vasoconstriction and platelet aggregation ● Clot formation
Inflammation	● Vasodilatation ● Migration of neutrophils and macrophages ● Phagocytosis of cellular debris and invading bacteria
Proliferation	● Granulation tissue formation (synthesised by fibroblasts) and angiogenesis ● Re-epithelialisation (epidermal cell proliferation and migration)
Remodelling	● Collagen fibre re-organisation ● Scar maturation

Emergency Dermatology

- These are rapidly progressive skin conditions and some are potentially life-threatening. Early recognition is important to implement prompt supportive care and therapy.
- Some are drug reactions and the offending drug should be withdrawn.
- The essential management for all dermatological emergencies, like any emergency, consists of:
  - i) full supportive care - ABC of resuscitation
  - ii) withdrawal of precipitating agents
  - iii) management of associated complications
  - iv) specific treatment (highlighted below under each condition)

Learning outcomes:

1. **Ability to recognise and describe these skin reactions:**
   - **urticaria**
   - **erythema nodosum**
   - **erythema multiforme**

2. **Ability to recognise these emergency presentations, discuss the causes, potential complications and provide first contact care in these emergencies:**
   - **anaphylaxis and angioedema**
   - **toxic epidermal necrolysis**
   - **Stevens-Johnson syndrome**
   - **acute meningococcaemia**
   - **erythroderma**
   - **eczema herpeticum**
   - **necrotising fasciitis**

Urticaria, Angioedema and Anaphylaxis

Causes

- Idiopathic, food (e.g. nuts, sesame seeds, shellfish, dairy products), drugs (e.g. penicillin, contrast media, non-steroidal anti-inflammatory drugs (NSAIDs), morphine, angiotensin-converting enzyme inhibitors (ACE-i)), insect bites, contact (e.g. latex), viral or parasitic infections, autoimmune, and hereditary (in some cases of angioedema)

Description

- **Urticaria** is due to a local increase in permeability of capillaries and small venules. A large number of inflammatory mediators (including prostaglandins, leukotrienes, and chemotactic factors) play a role but histamine derived from skin mast cells appears to be the major mediator. Local mediator release from mast cells can be induced by immunological or non-immunological mechanisms.

Presentation

- Urticaria (swelling involving the superficial dermis, raising the epidermis): itchy wheals
- Angioedema (deeper swelling involving the dermis and subcutaneous tissues): swelling of tongue and lips
- Anaphylaxis (also known as anaphylactic shock): bronchospasm, facial and laryngeal oedema, hypotension; can present initially with urticaria and angioedema

Management

- Antihistamines for urticaria
- Corticosteroids for severe acute urticaria and angioedema
- Adrenaline, corticosteroids and antihistamines for anaphylaxis

Complications

- Urticaria is normally uncomplicated
- Angioedema and anaphylaxis can lead to asphyxia, cardiac arrest and death

Urticaria

Angioedema

Erythema nodosum

Description

- A hypersensitivity response to a variety of stimuli

Causes

- Group A beta-haemolytic streptococcus, primary tuberculosis, pregnancy, malignancy, sarcoidosis, inflammatory bowel disease (IBD), chlamydia and leprosy

Presentation

- Discrete tender nodules which may become confluent
- Lesions continue to appear for 1-2 weeks and leave bruise-like discolouration as they resolve
- Lesions do not ulcerate and resolve without atrophy or scarring
- The shins are the most common site

Erythema nodosum

Erythema multiforme, Stevens-Johnson syndrome and Toxic epidermal necrolysis

Description

- **Erythema multiforme**, often of unknown cause, is an acute self-limiting inflammatory condition with herpes simplex virus being the main precipitating factor. Other infections and drugs are also causes. Mucosal involvement is absent or limited to only one mucosal surface.
- **Stevens-Johnson syndrome** is characterised by mucocutaneous necrosis with at least two mucosal sites involved. Skin involvement may be limited or extensive. Drugs or combinations of infections or drugs are the main associations. Epithelial necrosis with few inflammatory cells is seen on histopathology. The extensive necrosis distinguishes Stevens-Johnson syndrome from erythema multiforme. Stevens-Johnson syndrome may have features overlapping with toxic epidermal necrolysis including a prodromal illness.
- **Toxic epidermal necrosis** which is usually drug-induced, is an acute severe similar disease characterised by extensive skin and mucosal necrosis accompanied by systemic toxicity. On histopathology there is full thickness epidermal necrosis with subepidermal detachment.

Management

- Early recognition and call for help
- Full supportive care to maintain haemodynamic equilibrium

Complications

- Mortality rates are 5-12% with SJS and >30% with TEN with death often due to sepsis, electrolyte imbalance or multi-system organ failure

Erythema multiforme

Stevens-Johnson syndrome

Acute meningococcaemia

Description	● A serious communicable infection transmitted via respiratory secretions; bacteria get into the circulating blood
Cause	● Gram negative diplococcus Neisseria *meningitides*
Presentation	● Features of meningitis (e.g. headache, fever, neck stiffness), septicaemia (e.g. hypotension, fever, myalgia) and a typical rash ● Non-blanching purpuric rash on the trunk and extremities, which may be preceded by a blanching maculopapular rash, and can rapidly progress to ecchymoses, haemorrhagic bullae and tissue necrosis
Management	● Antibiotics (e.g. benzylpenicillin) ● Prophylactic antibiotics (e.g. rifampicin) for close contacts (ideally within 14 days of exposure)
Complications	● Septicaemic shock, disseminated intravascular coagulation, multi-organ failure and death

Erythroderma ('red skin')

Description	● Exfoliative dermatitis involving at least 90% of the skin surface
Causes	● Previous skin disease (e.g. eczema, psoriasis), lymphoma, drugs (e.g.sulphonamides, gold, sulphonylureas, penicillin, allopurinol, captopril) and idiopathic
Presentation	● Skin appears inflamed, oedematous and scaly ● Systemically unwell with lymphadenopathy and malaise
Management	● Treat the underlying cause, where known ● Emollients and wet-wraps to maintain skin moisture ● Topical steroids may help to relieve inflammation
Complications	● Secondary infection, fluid loss and electrolyte imbalance, hypothermia, high-output cardiac failure and capillary leak syndrome (most severe)
Prognosis	● Largely depends on the underlying cause ● Overall mortality rate ranges from 20 to 40%

Erythroderma

In richly pigmented skin the erythema doesn't look as bright, but on close inspection the inflamed skin might appear a darker shade of brown or black, with a hint of erythema visible. Palpating the skin for increased temperature is a vital clue.

Eczema herpeticum (Kaposi's varicelliform eruption)

Description	● Widespread eruption - serious complication of atopic eczema or less commonly other skin conditions
Cause	● Herpes simplex virus
Presentation	● Extensive crusted papules, blisters and erosions ● Systemically unwell with fever and malaise
Management	● Antivirals (e.g. aciclovir) ● Antibiotics for bacterial secondary infection
Complications	● Herpes hepatitis, encephalitis, disseminated intravascular coagulation (DIC) and rarely, death

Eczema herpeticum

Necrotising fasciitis

Description	● A rapidly spreading infection of the deep fascia with secondary tissue necrosis
Causes	● Group A haemolytic streptococcus, or a mixture of anaerobic and aerobic bacteria ● Risk factors include abdominal surgery and medical co-morbidities (e.g. diabetes, malignancy) ● 50% of cases occur in previously healthy individuals
Presentation	● Severe pain ● Erythematous, blistering, and necrotic skin ● Systemically unwell with fever and tachycardia ● Presence of crepitus (subcutaneous emphysema) ● X-ray may show soft tissue gas (absence should not exclude the diagnosis)
Management	● Urgent referral for extensive surgical debridement ● Intravenous antibiotics
Prognosis	● Mortality up to 76%

Skin Infections / Infestations

- The normal skin microflora and antimicrobial peptides protect the skin against infection. However, when there is skin damage, microorganisms can penetrate resulting in infection.
- There are 3 main types of skin infections according to their sources: bacterial (e.g. staphylococcal and streptococcal), viral (e.g. human papilloma virus, herpes simplex (*see page 34*) and herpes zoster (*see below)*), and fungal (e.g. tinea *(see page 39 & 40),* candida *(see page 39 & 40)* and yeasts). Infestations (e.g. scabies (*see page 60 & 61)*, lice, cutaneous leishmaniasis) can also occur.

Herpes zoster (shingles) infection due to varicella-zoster virus affecting the distribution of the ophthalmic division of the fifth cranial (trigeminal) nerve
Note: Examination for eye involvement is important

Learning outcomes:

Ability to describe the presentation, investigation and management of:

- cellulitis and erysipelas

- staphylococcal scalded skin syndrome

- superficial fungal infections

Erysipelas and Cellulitis

Description

- Spreading bacterial infection of the skin
- **Cellulitis** involves the deep subcutaneous tissue
- **Erysipelas** is an acute superficial form of cellulitis and involves the dermis and upper subcutaneous tissue

Causes

- Streptococcus pyogenes and Staphylococcus aureus
- Risk factors include immunosuppression, wounds, leg ulcers, toeweb intertrigo, and minor skin injury

Presentation

- Most common in the lower limbs
- Local signs of inflammation – swelling (tumor), erythema (rubor), warmth (calor), pain (dolor); may be associated with lymphangitis
- Systemically unwell with fever, malaise or rigors, particularly with erysipelas
- **Erysipelas** is distinguished from cellulitis by a well-defined, red raised border

Management

- Antibiotics (e.g. flucloxacillin or benzylpenicillin)
- Supportive care including rest, leg elevation, sterile dressings and analgesia

Complications

- Local necrosis, abscess and septicaemia

Cellulitis with elephantiasis of the penis

Erysipelas

Erysipelas

Even though this is in richly pigmented skin the unilateral oedema and erythema is clearly present suggesting cellulitis.

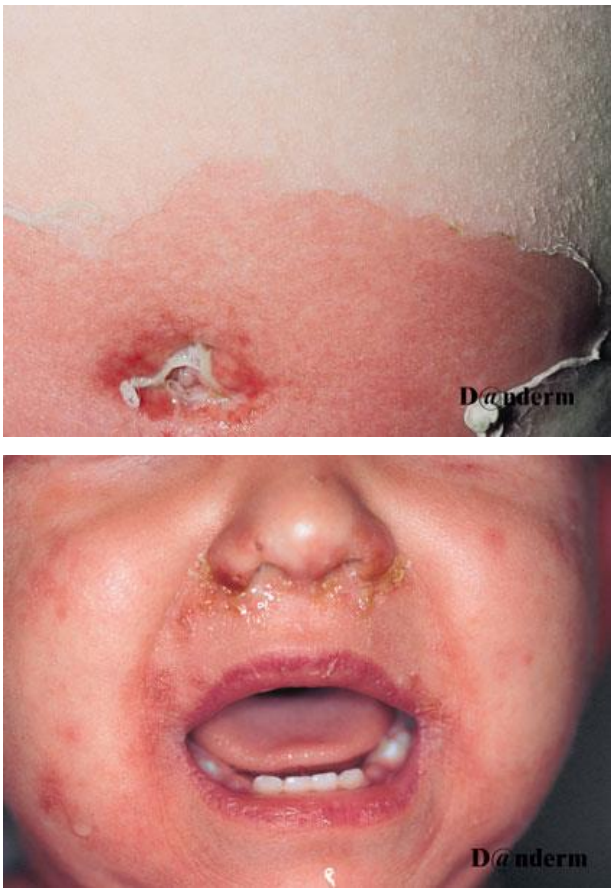
Staphylococcal scalded skin syndrome

Description
- Commonly seen in infancy and early childhood

Cause
- Production of a circulating epidermolytic toxin from phage group II, benzylpenicillin-resistant (coagulase positive) staphylococci

Presentation
- Develops within a few hours to a few days, and may be worse over the face, neck, axillae or groins
- A scald-like skin appearance is followed by large flaccid bulla
- Perioral crusting is typical
- There is intraepidermal blistering in this condition
- Lesions are very painful
- Sometimes the eruption is more localised
- Recovery is usually within 5-7 days

Management
- Antibiotics (e.g. a systemic penicillinase-resistant penicillin, erythromycin or appropriate cephalosporin)
- Analgesia



Staphylococcal scalded skin syndrome

Superficial fungal infections

Description

- A common and mild infection of the superficial layers of the skin, nails and hair, but can be severe in immunocompromised individuals

Cause

- Three main groups: dermatophytes (tinea/ringworm), yeasts (e.g. candidiasis, malassezia), moulds (e.g. aspergillus)

Presentation

- Varies with the site of infection; usually unilateral and itchy
- Tinea corporis (tinea infection of the trunk and limbs) - Itchy, circular or annular lesions with a clearly defined, raised and scaly edge is typical
- Tinea cruris (tinea infection of the groin and natal cleft) – very itchy, similar to tinea corporis
- Tinea pedis (athlete's foot) – moist scaling and fissuring in toewebs, spreading to the sole and dorsal aspect of the foot
- Tinea manuum (tinea infection of the hand) – scaling and dryness in the palmar creases
- Tinea capitis (scalp ringworm) – patches of broken hair, scaling and inflammation
- Tinea unguium (tinea infection of the nail) – yellow discolouration, thickened and crumbly nail
- Tinea incognito (inappropriate treatment of tinea infection with topical or systemic corticosteroids) – Ill-defined and less scaly lesions
- Candidiasis (candidal skin infection) – white plaques on mucosal areas, erythema with satellite lesions in flexures
- Pityriasis/Tinea versicolor (infection with Malassezia furfur) – scaly pale brown patches on upper trunk that fail to tan on sun exposure, usually asymptomatic

Management

- Establish the correct diagnosis by skin scrapings, hair or nail clippings (for dermatophytes); skin swabs (for yeasts)
- General measures: treat known precipitating factors (e.g. underlying immunosuppressive condition, moist environment)

- Topical antifungal agents (e.g. terbinafine cream)
- Oral antifungal agents (e.g. itraconazole) for severe, widespread, or nail infections
- Avoid the use of topical steroids – can lead to tinea incognito
- Correct predisposing factors where possible (e.g. moist environment, underlying immunosuppression)

Tinea corporis

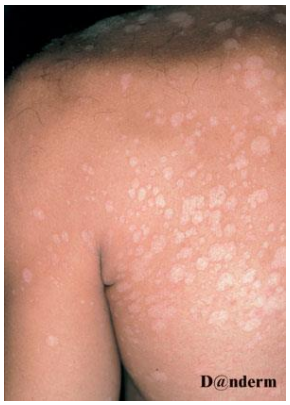
Diffuse Tinea capitis

Tinea capitis

Tinea manuum (right hand)

Tinea pedis with associated tinea unguium

Candidiasis (right axilla)



Pityriasis versicolor

Skin Cancer

- Skin cancer is one of the most common cancers.
- In general, skin cancer can be divided into: non-melanoma (basal cell carcinoma and squamous cell carcinoma) and melanoma (malignant melanoma).
- Malignant melanoma is the most life-threatening type of skin cancer and is one of the few cancers affecting the younger population.
- Sun exposure is the single most preventable risk factor for skin cancer.

Learning outcomes:

Ability to recognise:

- basal cell carcinoma

- squamous cell carcinoma

- malignant melanoma

Basal cell carcinoma

Description

- A slow-growing, locally invasive malignant tumour of the epidermal keratinocytes normally in older individuals, only rarely metastasises
- Most common malignant skin tumour

Causes

- Risk factors include UV exposure, history of frequent or severe sunburn in childhood, skin type I (always burns, never tans), increasing age, male sex, immunosuppression, previous history of skin cancer, and genetic predisposition

Presentation

- Various morphological types including nodular (most common), superficial (plaque-like), cystic, morphoeic (sclerosing), keratotic and pigmented
- Nodular basal cell carcinoma is a small, skin-coloured papule or nodule with surface telangiectasia, and a pearly rolled edge; the lesion may have a necrotic or ulcerated centre (rodent ulcer)
- Most common over the head and neck

Management

- Surgical excision - treatment of choice as it allows histological examination of the tumour and margins
- Mohs micrographic surgery (i.e. excision of the lesion and tissue borders are progressively excised until specimens are microscopically free of tumour) - for high risk, recurrent tumours
- Radiotherapy - when surgery is not appropriate
- Other e.g. cryotherapy, curettage and cautery, topical photodynamic therapy, and topical treatment (e.g. imiquimod cream) - for small and low-risk lesions

Complications

- Local tissue invasion and destruction

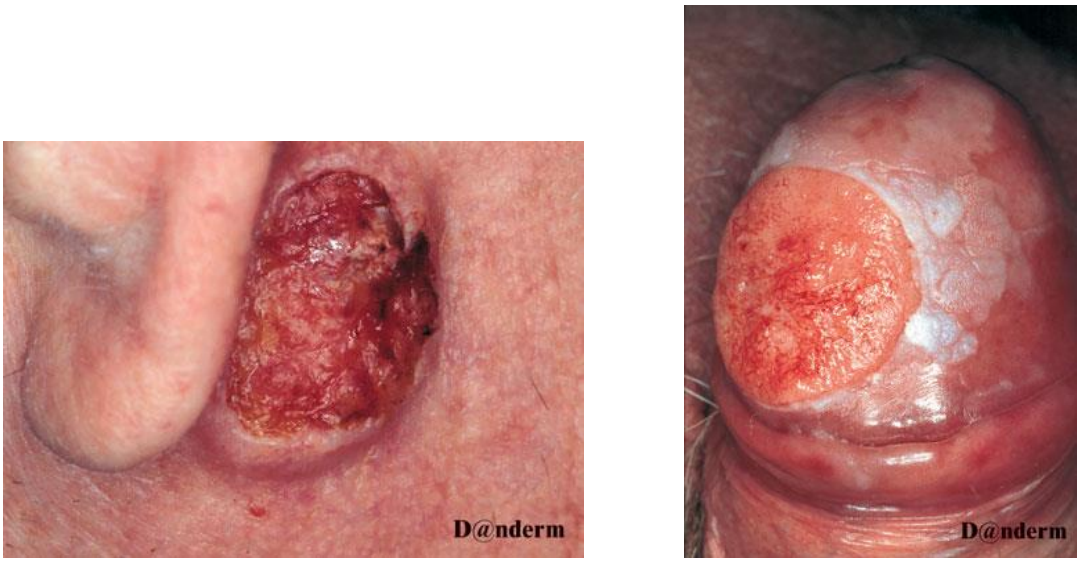
Prognosis

- Depends on tumour size, site, type, growth pattern/histological subtype, failure of previous treatment/recurrence, and immunosuppression

Basal cell carcinoma – nodular type

Squamous cell carcinoma

Description	● A locally invasive malignant tumour of the epidermal keratinocytes or its appendages, which has the potential to metastasise
Causes	● Risk factors include excessive UV exposure, pre-malignant skin conditions (e.g. actinic keratoses), chronic inflammation (e.g. leg ulcers, wound scars), immunosuppression and genetic predisposition
Presentation	● Keratotic (e.g. scaly, crusty), ill-defined nodule which may ulcerate
Management	● Surgical excision - treatment of choice ● Mohs micrographic surgery – may be necessary for ill-defined, large, recurrent tumours ● Radiotherapy - for large, non-resectable tumours
Prognosis	● Depends on tumour size, site, histological pattern, depth of invasion, perineural involvement, and immunosuppression



Squamous cell carcinoma – adjacent to ear (left) and glans penis (right)

Malignant melanoma

Description

- An invasive malignant tumour of the epidermal melanocytes, which has the potential to metastasise

Causes

- Risk factors include excessive UV exposure, skin type I (always burns, never tans), history of > 100 moles or atypical neavus syndrome moles, family history in first degree relative or previous history of melanoma

Presentation

- The **'ABCDE Symptoms'** rule *(*major suspicious features)*:

  Asymmetrical shape*

  Border irregularity

  Colour irregularity*

  Diameter > 6mm

  Evolution of lesion (e.g. change in size and/or shape)*

  Symptoms (e.g. bleeding, itching)

- More common on the legs in women and trunk in men

Types

- Superficial spreading melanoma – common on the lower limbs, in young and middle-aged adults; related to intermittent high-intensity UV exposure; around 70% of all melanomas are superficial spreading melanomas
- Nodular melanoma - common on the trunk, in young and middle-aged adults; related to intermittent high-intensity UV exposure
- Lentigo maligna melanoma - common on the face, in elderly population; related to long-term cumulative UV exposure
- Acral lentiginous melanoma - common on the palms, soles and nail beds, in elderly population; no clear relation with UV exposure

Management

- Depends on the staging of melanoma (currently used system in the UK - 2009 American Joint Committee of Cancer Staging System (AJCC)). Stages I-IV are based on primary tumour Breslow thickness, lymph node involvement and evidence of metastases. Stage I is the earliest and stage IV is the most advanced)
- In general, surgical excision is the definitive treatment (often a second surgery, wide local excision is needed after the initial

excision biopsy). Radiotherapy may sometimes be useful. Chemotherapy is used for metastatic disease.

Prognosis

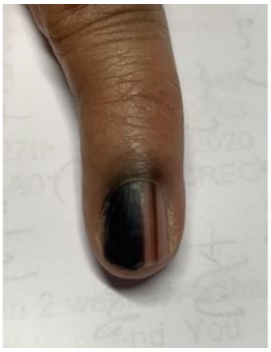
- Prognosis depends on the stage of melanoma and Breslow thickness.
- In general, 90% of people diagnosed with melanoma in England and Wales survived 10 years or more (Cancer Research UK, 2010-2011).

Superficial spreading melanoma

Nodular melanoma

Lentigo maligna melanoma

Acral lentiginous melanoma



Acral lentiginous melanoma (in situ)

Further reading: British Association of Dermatologists. Revised UK guidelines for the management of cutaneous melanoma 2010. https://www.bad.org.uk/library-media%5Cdocuments%5CMelanoma_2010.pdf

Inflammatory Skin Conditions

- Eczema, acne and psoriasis are chronic inflammatory skin disorders that follow a relapsing and remitting course. There are many types of eczema but we shall just consider atopic eczema here.
- These skin disorders are not infectious.
- Management is aimed at achieving control and not providing a cure.
- Complications are mainly due to the psychological and social effects.
- Patient education is important in these chronic skin conditions and should concentrate on providing information about the nature of condition, aims of treatment and the available treatment options.

Learning outcomes:

Ability to describe the presentation, demonstrate assessment, formulate a differential diagnosis, instigate investigation and discuss how to provide continuing care of:

- atopic eczema

- acne

- psoriasis

Atopic eczema

Description

- Eczema (or dermatitis) is a chronic skin condition common in children but also prevalent in adults.

Epidemiology

- 20% prevalence in <12 years old in the UK

Causes

- Not fully understood, but a positive family history of atopy (i.e. eczema, asthma, allergic rhinitis) is often present
- A primary genetic defect in skin barrier function (loss of function variants of the protein filaggrin) appears to underlie atopic eczema
- Exacerbating factors such as infections, allergens (e.g. chemicals, food, dust, pet fur), sweating, heat, occupation and severe stress

Presentation

- Acute presentation consists of itchy papules and vesicle often weepy (exudative)
- Chronic lesions : dry scaly itchy patches can be erythematous in paler skin or grey/ brown in richly pigmented skin
- More common on the face and extensor aspects of limbs in infants, and the flexor aspects in children and adults
- In richly pigmented skin eczema may present as brown, grey or purple bumps (papular eczema or follicular eczema)
- Chronic scratching/rubbing leads to lichenification
- Across of skin types eczema can lead to pigmentary changes such as hypopigmentation (reduced pigmentation) and hyperpigmentation (increased pigmentation)
- Nail may show pitting and ridging of the nails

Management

- General measures - avoid known exacerbating agents, frequent emollients +/- bandages and bath oil/soap substitute
- Topical therapies – topical steroids for active areas; topical immunomodulators (e.g. tacrolimus, pimecrolimus) for maintenance therapy as steroid-sparing agents
- Oral therapies - antihistamines for symptomatic relief, antibiotics (e.g. flucloxacillin) for secondary bacterial infections, and antivirals (e.g. aciclovir) for secondary herpes infection

● Phototherapy and immunosuppressants (e.g. azathioprine, ciclosporin, methotrexate) for severe non- responsive cases, biologic therapy

Complications

● Secondary bacterial infection (crusted weepy lesions)

● Secondary viral infection - molluscum contagiosum (pearly papules with central umbilication), viral warts and eczema herpeticum (*see page 34*)

Atopic eczema

Further reading: NICE. Eczema – Atopic, last updated Jan 2018. https://cks.nice.org.uk/eczema-atopic

Acne vulgaris

Description
- An inflammatory disease of the pilosebaceous follicle

Epidemiology
- Over 80% of teenagers aged 13- 18 years

Causes
- Hormonal (androgen)
- Contributing factors include increased sebum production, abnormal follicular keratinization, bacterial colonization (*Propionibacterium acnes*) and inflammation

Presentation
- Non-inflammatory lesions (mild acne) - open and closed comedones (blackheads and whiteheads)
- Inflammatory lesions (moderate and severe acne) - papules, pustules, nodules, and cysts
- In richly pigmented skin:
    1. Inflammatory lesions' may not be so apparent, instead hyperpigmented lesions ('acne hyperpigmented macules') are seen.
       Hyperpigmented lesions may also signify ongoing inflammation
    2. Non erythematous nodules may be present and detected by palpation
- Commonly affects the face, chest and upper back

Comedones (Left and Middle) Papules and nodules (Right)

Management
- General measures - no specific food has been identified to cause acne, treatment needs to be continued for at least 6 weeks to produce effect
- Topical therapies (for mild acne) - benzoyl peroxide and topical antibiotics (antimicrobial properties), and topical retinoids

(comedolytic and anti-inflammatory properties)

- Oral therapies (for moderate to severe acne) - oral antibiotics, and anti-androgens (in females)
- Oral retinoids (for severe acne)

Complications

- Post-inflammatory hyperpigmentation, scarring, deformity, psychological and social effects

Psoriasis

Description

- A chronic inflammatory skin disease due to hyperproliferation of keratinocytes and inflammatory cell infiltration

Types

- Chronic plaque psoriasis is the most common type
- Other types include guttate (raindrop lesions), seborrhoeic (naso-labial and retro-auricular), flexural (body folds), pustular (palmar-plantar), and erythrodermic (total body redness)

Epidemiology

- Affects about 2% of the population in the UK

Causes

- Complex interaction between genetic, immunological and environmental factors
- Precipitating factors include trauma (which may produce a Köebner phenomenon), infection (e.g. tonsillitis), drugs, stress, and alcohol

Presentation

- Well-demarcated erythematous scaly plaques
- in richly pigmented skin psoriasis can present as dark brown, grey or purple patches or plaques
- Lesions can sometimes be itchy, burning or painful
- Common on the extensor surfaces of the body and over scalp
- Auspitz sign (scratch and gentle removal of scales cause capillary bleeding)
- 50% have associated nail changes (e.g. pitting, onycholysis)
- 5-8% suffer from associated psoriatic arthropathy - symmetrical polyarthritis, asymmetrical oligomonoarthritis, lone distal interphalangeal disease, psoriatic spondylosis, and arthritis mutilans (flexion deformity of distal interphalangeal joints)

Management

- General measures - avoid known precipitating factors, emollients to reduce scales
- Topical therapies (for localised and mild psoriasis) - vitamin D analogues, topical corticosteroids, coal tar preparations, dithranol, topical retinoids, keratolytics and scalp preparations
- Phototherapy (for extensive disease) - phototherapy i.e. UVB and photochemotherapy i.e. psoralen+UVA
- Oral therapies (for extensive and severe psoriasis, or psoriasis with systemic involvement) - methotrexate, retinoids, ciclosporin, mycophenolate mofetil, fumaric acid esters, and biological agents (e.g. etanercept, adalimumab, ustekinumab) *(see page 71)*

Complications

- Erythroderma (*see page 33*), psychological and social effects

Köebner phenomenon

Plaque Psoriasis

Plaque psoriasis

Nail changes and arthropathy

Scalp Psoriasis

Blistering Disorders

- In general, blistering skin disorders can be divided into: immunobullous diseases (e.g. bullous pemphigoid, pemphigus vulgaris), blistering skin infections (e.g. herpes simplex) and other (e.g. porphyria cutanea tarda).
- The fragility of blisters depends on the level of split within the skin – an intra-epidermal split (a split within the epidermis) causes blisters to rupture easily; whereas a sub-epidermal split (a split between the epidermis and dermis) causes blisters to be less fragile.
- The common causes of blisters are impetigo (*see below*), insect bites, herpes simplex infection (*see page 34*), herpes zoster infection (*see page 36*), acute contact dermatitis, pompholyx (vesicular eczema of the hands and feet, see below) and burns.
- Bullous pemphigoid *(see page 53)* and pemphigus vulgaris *(see page 54)* are uncommon conditions due to immune reaction within the skin.

Bullous impetigo in a new tattoo

Pompholyx

Learning outcomes:

1. Ability to recognise common causes of blisters

2. Ability to recognise:

- Bullous pemphigoid

- Pemphigus vulgaris

Bullous pemphigoid

Description
- A blistering skin disorder which usually affects the elderly

Cause
- Autoantibodies against antigens between the epidermis and dermis causing a sub-epidermal split in the skin

Presentation
- Tense, fluid-filled blisters on an erythematous base
- Lesions are often itchy
- May be preceded by a non-specific itchy rash
- Usually affects the trunk and limbs (mucosal involvement less common)

Management
- General measures – wound dressings where required, monitor for signs of infection
- Topical therapies for localised disease - topical steroids
- Oral therapies for widespread disease – oral steroids, combination of oral tetracycline and nicotinamide, immunosuppressive agents (e.g. azathioprine, mycophenolate mofetil, methotrexate, and other)

Bullous pemphigoid

Pemphigus vulgaris

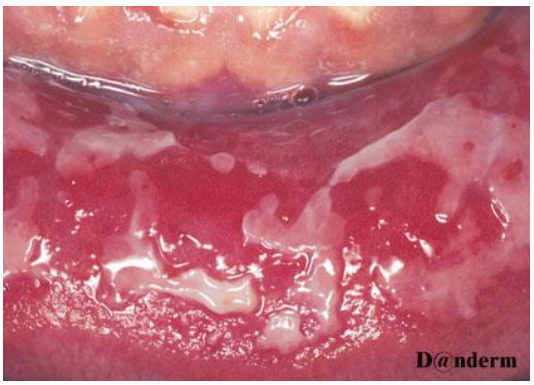
Description
- A blistering skin disorder which usually affects the middle-aged

Cause
- Autoantibodies against antigens within the epidermis causing an intra-epidermal split in the skin

Presentation
- Flaccid, easily ruptured blisters forming erosions and crusts
- Lesions are often painful
- Usually affects the mucosal areas (can precede skin involvement)

Management
- General measures – wound dressings where required, monitor for signs of infection, good oral care (if oral mucosa is involved)
- Oral therapies – high-dose oral steroids, immunosuppressive agents (e.g. methotrexate, azathioprine, cyclophosphamide, mycophenolate mofetil, and other)

Pemphigus vulgaris



Pemphigus vulgaris affecting the oral mucosa

Pigmentary Disorders

- Pigmentary issues are a significant problem in all patients, how it differs in different skin colour - population in the UK and it is important that medical students and junior doctors appreciate the dermatoses pertinent to these groups.
- In general, a pigment change can present as hypopigmentation (reduced pigmentation), depigmentation (complete loss of pigment), or hyperpigmentation (increased pigmentation).
- Below are some of the common pigmentary disorders which can cause significant embarrassment and distress especially in the darker skin types.

Learning objectives:

1. Ability to formulate a differential diagnosis, describe the investigation and discuss the management in patients with:

- vitiligo

- melasma

Vitiligo

Description

● An acquired depigmenting disorder, where there is complete loss of pigment cells (melanocytes)

Cause

● Thought to be an autoimmune disorder, where the innate immune system causes destruction or loss of melanocytes, leading to loss of pigment formation in the skin

Presentation

● Presentation at any age

- A single patch or multiple patches of depigmentation (complete loss of pigment), often symmetrical

● Common sites are exposed areas such as face, hands, feet, as well as body folds and genitalia

● Favours sites of injury and this phenomenon is called the Koebner phenomenon

Management

● Minimise skin injury as a cut, graze, or sunburn can potentially trigger a new patch of vitiligo

● Topical treatments such as topical steroids and calcineurin inhibitors (such as topical tacrolimus and pimecrolimus)

- Phototherapy such as UVB therapy, excimer laser
- Oral immunosuppressants such as methotrexate, ciclosporin and mycophenolate mofetil

Melasma

Description

- An acquired chronic skin disorder, where there is increased pigmentation in the skin

Cause

- Thought to be due to genetic predisposition, and triggered by factors such as sun exposure, hormonal changes such as pregnancy and contraceptive pills
- The pigmentation is caused by an overproduction of pigment (melanin) by pigment cells (melanocytes)

Presentation

- Brown macules (freckle-like spots) or larger patches with an irregular border
- Symmetrical distribution
- Common sites are forehead, cutaneous upper lips and cheeks, rarely can occur on neck, shoulders and upper arms

Management

- Lifelong sun protection
- Discontinuation of hormonal contraceptive pills
- Cosmetic camouflage
- Topical treatments that aim at inhibiting the formation of new melanin such as hydroquinone, azelaic acid, kojic acid (a chelating agent) and vitamin C
- Laser treatments need to be used with caution as the heat generated by lasers can potentially cause post-inflammatory hyperpigmentation.

Common Important Problems

- There are several commonly-encountered skin problems in clinical practice. Below are some of the important differential diagnoses for each of these presentations.
- Clinical exposure is the key to achieve competence in diagnosing, investigating and managing these skin problems.

Learning objectives:

Ability to formulate a differential diagnosis, describe the investigation and discuss the management in patients with:

- chronic leg ulcers

- itchy eruption

- a changing pigmented lesion

- purpuric eruption

- a red swollen leg

- keloid scars

Chronic leg ulcers

- Leg ulcers are classified according to aetiology. In general, there are three main types: venous, arterial and neuropathic ulcers. Other causes include vasculitic ulcers (purpuric, punched out lesions), infected ulcers (purulent discharge, may have systemic signs) and malignancy (e.g. squamous cell carcinoma in long-standing non-healing ulcers).
- In clinical practice, there can be mixture of arterial, venous and/or neuropathic components in an ulcer.

Venous ulcer

Venous ulcer

Arterial ulcer

Neuropathic ulcer

Chronic leg ulcers

	Venous ulcer	Arterial ulcer	Neuropathic ulcer
History	- Often painful, worse on standing - History of venous disease e.g. varicose veins, deep vein thrombosis	- Painful especially at night, worse when legs are elevated - History of arterial disease e.g. atherosclerosis	- Often painless - Abnormal sensation - History of diabetes or neurological disease
Common sites	- Malleolar area (more common over medial than lateral malleolus)	- Pressure and trauma sites e.g. pretibial, supramalleolar (usually lateral), and at distal points e.g. toes	- Pressure sites e.g. soles, heel, toes, metatarsal heads
Lesion	- Large, shallow irregular ulcer - Exudative and granulating base	- Small, sharply defined deep ulcer - Necrotic base	- Variable size and depth - Granulating base - May be surrounded by or underneath a hyperkeratotic lesion (e.g. callus)
Associated features	- Warm skin - Normal peripheral pulses - Leg oedema, haemosiderin and melanin deposition (brown pigment), lipodermatosclerosis, and atrophie blanche (white scarring with dilated capillaries)	- Cold skin - Weak or absent peripheral pulses - Shiny pale skin - Loss of hair	- Warm skin - Normal peripheral pulses* **cold, weak or absent pulses if it is a neuroischaemic ulcer* - Peripheral neuropathy
Possible investigations	- Normal ankle/brachial pressure index (i.e. ABPI 0.8-1)	- ABPI < 0.8 - presence of arterial insufficiency - Doppler studies and angiography	- ABPI < 0.8 implies a neuroischaemic ulcer - X-ray to exclude osteomyelitis
Management	- Compression bandaging (after excluding arterial insufficiency)	- Vascular reconstruction - Compression bandaging is contraindicated	- Wound debridement - Regular repositioning, appropriate footwear and good nutrition

Itchy eruption

- An itchy (pruritic) eruption can be caused by an inflammatory condition (e.g. eczema), infection (e.g. varicella), infestation (e.g. scabies), allergic reaction (e.g. some cases of urticaria) or an unknown cause, possibly autoimmune (e.g. lichen planus).

Chronic fissured hand eczema

Scabies

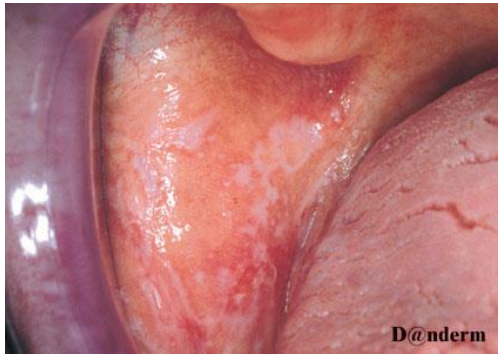
Urticaria

R © Cardiff and Vale University Health Board.

Lichen planus

© Cardiff and Vale University Health Board.

Lichen planus



Wickham's striae

Note that lichen planus in darker skin types has a typical purplish tinge.

Dermatology: Handbook for medical students & junior doctors

Itchy eruption

	Eczema	Scabies	Urticaria	Lichen planus
History	- Personal or family history of atopy - Exacerbating factors (e.g. allergens, irritants)	- May have history of contact with symptomatic individuals - Pruritus worse at night	- Precipitating factors (e.g. food, contact, drugs)	- Family history in 10% of cases - May be drug-induced
Common sites	- Variable (e.g. flexor aspects in children and adults with atopic eczema) Lichen nitidus pattern in darker skin	- Sides of fingers, finger webs, wrists, elbows, ankles, feet, nipples and genitals	- No specific tendency	- Forearms, wrists, and legs - Always examine the oral mucosa
Lesion	- Dry, erythematous patches - Acute eczema is erythematous, vesicular and exudative	- Linear burrows (may be tortuous) or rubbery nodules	- Pink wheals (transient) - May be round, annular, or polycyclic	- Violaceous (lilac) flat-topped Papules or hyperpigmented papules (in darker skin) - Symmetrical distribution
Associated features	- Secondary bacterial or viral infections	- Secondary eczema and impetigo	- May be associated with angioedema or anaphylaxis	- Nail changes and hair loss - Lacy white streaks on the oral mucosa and skin lesions (Wickham's striae)
Possible investigations	- Patch testing - Serum IgE levels - Skin swab	- Skin scrape, extraction of mite and view under microscope	- Bloods and urinalysis to exclude a systemic cause	- Skin biopsy
Management	- Emollients - Corticosteroids - Immunomodulators - Antihistamines	- Scabicide (e.g. permethrin or malathion) - Antihistamines	- Antihistamines - Corticosteroids	- Corticosteroids - Antihistamines

Dermatology: Handbook for medical students & junior doctors

A changing pigmented lesion

- A changing pigmented lesion can be benign (e.g. melanocytic naevi, seborrhoeic wart) or malignant (e.g. malignant melanoma).

Congenital naevus

Seborrhoeic keratoses

Malignant melanoma

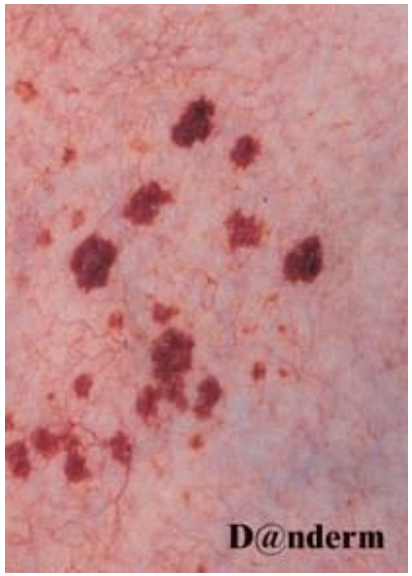
A changing pigmented lesion

	Benign		Malignant
	Melanocytic naevi	**Seborrhoeic wart**	**Malignant melanoma**
History	- Not usually present at birth but develop during infancy, childhood or adolescence - Asymptomatic	- Tend to arise in the middle-aged or elderly - Often multiple and asymptomatic	- Tend to occur in adults or the middle-aged - History of evolution of lesion - May be symptomatic (e.g. itchy, bleeding) - Presence of risk factors
Common sites	- Variable	- Face and trunk	- More common on the legs in women and trunk in men - Darker skin tomes acral sites
Lesion	- Congenital naevi may be large, pigmented, protuberant and hairy - Junctional naevi are small, flat and dark - Intradermal naevi are usually dome-shape papules or nodules - Compound naevi are usually raised, warty, hyperkeratotic, and/or hairy	- Warty greasy papules or nodules - 'Stuck on' appearance, with well-defined edges	- Features of ABCDE: Asymmetrical shape Border irregularity Colour irregularity Diameter > 6mm Evolution of lesion
Management	- Only if symptomatic Shave or complete excision	- Only if symptomatic Curette and cautery Cryotherapy	- Local Excision Treatment based on Breslow Thickness

Purpuric eruption

- A purpuric eruption can be thrombocytopenic (e.g. meningococcal septicaemia, disseminated intravascular coagulation, idiopathic thrombocytopenic purpura) or non-thrombocytopenic e.g. trauma, drugs (e.g. steroids), aged skin, vasculitis (e.g. Henoch-Schönlein purpura).

Henoch-Schönlein purpura



Actinic purpura

Purpuric eruption

	Meningococcal septicaemia	Disseminated intravascular coagulation	Vasculitis	Actinic purpura
History	- Acute onset - Symptoms of meningitis and septicaemia	- History of trauma, malignancy, sepsis, obstetric complications, transfusions, or liver failure	- Painful lesions	- Arise in the elderly population with sun-damaged skin
Common sites	- Extremities	- Spontaneous bleeding from ear, nose and throat, gastrointestinal tract, respiratory tract or wound site	- Dependent areas (e.g. legs, buttocks, flanks)	- Extensor surfaces of hands and forearms - Such skin is easily traumatised
Lesion	- Petechiae, ecchymoses, haemorrhagic bullae and/or tissue necrosis	- Petechiae, ecchymoses, haemorragic bullae and/or tissue necrosis	- Palpable purpura (often painful)	- Non-palpable purpura - Surrounding skin is atrophic and thin
Associated features	- Systemically unwell	- Systemically unwell	- Systemically unwell	- Systemically well
Possible investigations	- Bloods - Lumbar puncture	- Bloods (a clotting screen is important)	- Bloods and urinalysis - Skin biopsy	- No investigation is needed
Management	- Antibiotics	- Treat the underlying cause - Transfuse for coagulation deficiencies - Anticoagulants for thrombosis	- Treat the underlying cause - Steroids and immunosuppressants if there is systemic involvement	- No treatment is needed

Dermatology: Handbook for medical students & junior doctors

A red swollen leg

- The main differential diagnoses for a red swollen leg are cellulitis, e

	Cellulitis/Erysipelas	Venous thrombosis	Chronic venous insufficiency
History	- Painful spreading rash - History of abrasion or ulcer	- Pain with swelling and redness - History of prolonged bed rest, long haul flights or clotting tendency	- Heaviness or aching of leg, which is worse on standing and relieved by walking - History of venous thrombosis
Examination	- Erysipelas (well-defined edge) - Cellulitis (diffuse edge)	- Complete venous occlusion may lead to cyanotic discolouration	- Discoloured (blue-purple) - Oedema (improved in the morning) - Venous congestion and varicose veins
Associated features	- Systemically unwell with fever and malaise - May have lymphangitis	- Usually systemically well - May present with pulmonary embolism	- Lipodermatosclerosis (erythematous induration, creating 'champagne bottle' appearance) - Stasis dermatitis (eczema with inflammatory papules, scaly and crusted erosions) - Haemosiderin deposition - Venous ulcer
Possible investigations	- Anti-streptococcal O titre (ASOT) - Skin swab	- D-dimer - Doppler ultrasound and/or venography	- Doppler ultrasound and/or venography
Management	- Antibiotics	- Anticoagulants	- Leg elevation and compression stockings - Sclerotherapy or surgery for varicose veins

Keloid Scars

Description
- An overgrowth of scar tissue, which tends to be larger than the original wound itself

Cause
- Thought to be due to overproduction of collagen during wound healing after minor injuries, skin surgery, insect bites and acne spots in genetically predisposed individuals
- More commonly seen in darker skin types

Presentation
- Firm, smooth, hard nodule which can be itchy or painful
- Common sites are chest and shoulders

Management
- Avoidance of further trauma to the skin such as scratching
- Topical treatments such as topical steroids and silicone gel can potentially flatten the scar, and improve the symptoms
- Intralesional steroid injection if topical treatments are not effective
- Surgery such as excision needs to be carried out only as the last resort and with caution as the new wound may cause a larger keloid scar

Management

- Treatment modalities for skin disease can be broadly categorised into medical therapy (topical and systemic treatments) and physical therapy (e.g. cryotherapy, phototherapy, photodynamic therapy, lasers and surgery).
- Topical treatments directly deliver treatment to the affected areas and this reduces systemic side effects. It is suitable for localised and less severe skin conditions. They consist of active constituents which are transported into the skin by a base (also known as a 'vehicle'). Examples of active ingredients are steroids, tar, immunomodulators, retinoids, and antibiotics. The common forms of base are lotion (liquid), cream (oil in water), gel (organic polymers in liquid, transparent), ointment (oil with little or no water) and paste (powder in ointment).
- Systemic therapy is used for extensive and more serious skin conditions, if the treatment is ineffective topically or if there is systemic involvement. However, they have the disadvantage of causing systemic side effects.

Learning objectives:

Ability to describe the principles of use of the following drugs:

- **emollients**
- **topical/oral corticosteroids**
- **oral aciclovir**
- **oral antihistamines**
- **topical/oral antibiotics**
- **topical antiseptics**
- **biological therapy**
- **Oral retinoids**

Emollients

Examples
- Aqueous cream, emulsifying ointment, liquid paraffin and white soft paraffin in equal parts (50:50)

Quantity
- 500 grams per tub

Indications
- To rehydrate skin and re-establish the surface lipid layer
- Useful for dry, scaling conditions and as soap substitutes

Side effects
- Reactions may be irritant or allergic (e.g. due to preservatives or perfumes in creams)

Topical/Oral corticosteroids

Examples
- Topical steroids: classified as mildly potent (e.g, hydrocortisone), moderately potent (e.g. clobetasone butyrate (*Eumovate)*), potent (e.g.betamethasone valerate (*Betnovate)*), and very potent (e.g. clobetasol propionate (*Dermovate)*)
- Oral steroids: prednisolone

Quantity
- Usually 30 grams per tube (enough to cover the whole body once)

Indications
- Anti-inflammatory and anti-proliferative effects
- Useful for allergic and immune reactions, inflammatory skin conditions, blistering disorders, connective tissue diseases, and vasculitis

Side effects
- Local side effects (from topical corticosteroids): skin atrophy (thinning), telangiectasia, striae, may mask, cause or exacerbate skin infections, acne, or perioral dermatitis, and allergic contact dermatitis.
- Systemic side effects (from oral corticosteroids): Cushing's syndrome, immunosuppression, hypertension, diabetes, osteoporosis, cataract, and steroid-induced psychosis

Oral aciclovir

Examples
- Aciclovir

Indications
- Viral infections due to herpes simplex and herpes zoster virus

Side effects
- Gastrointestinal upsets, raised liver enzymes, reversible neurological reactions, and haematological disorders

Oral antihistamines

Examples
- Classified into nonsedative (e.g. cetirizine, loratadine) and sedative antihistamines (e.g. chlorpheniramine, hydroxyzine)

Indications
- Block histamine receptors producing an anti-pruritic effect
- Useful for type-1 hypersensitivity reactions and eczema (especially sedative antihistamines for children)

Side effects
- Sedative antihistamines can cause sedation and anticholinergic effects (e.g. dry mouth, blurred vision, urinary retention, and constipation)

Topical/Oral antibiotics

Examples
- Topical antibiotics: fusidic acid, mupirocin (*Bactroban*), neomycin
- Oral antibiotics: penicillins, cephalosporins, gentamicin, macrolides, nitrofurantoin, quinolones, tetracyclines, vancomycin, metronidazole, trimethoprim

Indications
- Useful for bacterial skin infections, and some are used for acne

Side effects
- Local side effects (from topical antibiotics): local skin irritation/allergy
- Systemic side effects (from oral antibiotics): gastrointestinal upset, rashes, anaphylaxis, vaginal candidiasis, antibiotic-associated infection such as Clostridium difficile, and antibiotic resistance (rapidly appears to fusidic acid)

Topical antiseptics

Examples
- Chlorhexidine, cetrimide, povidone-iodine

Indications
- Treatment and prevention of skin infection

Side effects
- Local side effects: local skin irritation/allergy

Oral retinoids

Examples
- Isotretinoin, Acitretin

Indications
- Acne, psoriasis, and disorders of keratinisation

Side effects
- Mucocutaneous reactions such as dry skin, dry lips and dry eyes, disordered liver function, hypercholesterolaemia, hypertriglyceridaemia, myalgia, arthralgia and depression
- Teratogenicity: effective contraception must be practised one month

before, during and at least one month after isotretinoin, but for two years after Acitretin (consult current BNF for further details)

Biological Therapy

Examples Monoclonal antibodies (eg. Infliximab, Adalimumab, Ustekinumab, Certolizumab, Gorlilumab), Fusion antibody proteins (eg. Etanercept), Recombinant human cytokines and growth factors (eg. Interleukins)

Indications ● Mainly for psoriasis, atopic dermatitis and hidradenitis suppurativa

Side effects ● Local side effects: redness, swelling, bruising at the site of injection

● Systemic side effects: allergic reactions, antibody formation, flu-like symptoms, infections, hepatitis, demyelinating disease, heart failure, blood problems, rare reports of cancers (eg. non-melanoma skin cancers, lymphoma)

Practical Skills

- There are four main aspects to focus on in clinical practice:
    i) Patient education, particularly on the nature of disease, treatment and ways to achieve full compliance and effectiveness, and prevention strategies
    ii) Effective written communication to general practitioner so that patient care can be continued appropriately
    iii) Good prescribing skills
    iv) Good clinical examination and appropriate investigations to facilitate accurate diagnosis

Learning objectives:

1. **Ability to perform the following tasks:**
    - **explain how to use an emollient or a topical corticosteroid**
    - **make a referral**
    - **write a discharge letter**
    - **write a prescription for emollient**
    - **take a skin swab**
    - **take a skin scrape**

2. **Describe the principles of prevention in:**
    - **pressure sores**
    - **sun damage and skin cancer**

Patient education

How to use emollients

- Apply liberally and regularly

How to use topical corticosteroids

- Apply thinly and only for short-term use (often 1 or 2 weeks only)
- In general, use 1% hydrocortisone or mild-moderate potent topical steroids on the face and thin skin areas eg. neck and flexures.
- Fingertip unit (advised on packaging) – strip of cream the length of a fingertip

Preventing pressure sores

- Pressure sores are due to ischaemia resulting from localised damage to the skin caused by sustained pressure, friction and moisture, particularly over bony prominences.
- Preventative measures involve frequent repositioning, nutritional support, and use of pressure relieving devices e.g. special beds

Preventing sun damage and skin cancer

- Excessive exposure to UV radiation is the most significant and preventable risk factor for the development of skin cancer (Table 14)
- Skin types I and II are at higher risk of developing skin cancer with excessive sun exposure than other skin types (Table 15)

Table 14. SMART ways to avoid excessive sun exposure

Spend time in the shade between 11am-3pm
Make sure you never burn
Aim to cover up with a t-shirt, wide-brimmed hat and sunglasses
Remember to take extra care with children
Then use Sun Protection Factor (SPF) 30+ sunscreen

Table 15. Fitzpatrick Skin phototype

Skin types	Description
I	Always burns, never tans
II	Always burns, sometimes tans
III	Sometimes burns, always tans
IV	Never burns, always tans
V	Tans very easily, very rarely burns
VI	tans very easily, never burns

Written communication

Writing a referral letter

Important points to include:

- Reason(s) for referral, current presentation, and impact of disease
- Patient's medical and social background
- Current and previous treatment, length of treatment, and response to treatment

Writing a discharge letter

Important points to include:

- Reason(s) for admission and current presentation
- Hospital course
- Investigation results
- Diagnostic impression
- Management plan (including treatment and follow-up appointment)
- Content of patient education given

Prescribing skills

Writing a prescription

General tips:

- Include drug name, dose, frequency and an intended duration/review date
- 30 grams of cream/ointment covers the whole adult body area
- 1 fingertip unit covers the area of two palms and equals ½ gram

Prescribing emollients

General tips

- Emollients come in 500 gram tubs
- In general, ointment-based emollients are useful for dry, scaling skin whereas creams and lotions are for red, inflamed and weeping lesions

Prescribing topical corticosteroids

General tips

- Prescribe the weakest potency corticosteroid that is effective
- Use only for short term
- Need to specify the base i.e. cream, lotion or ointment

Clinical examination and investigations

Taking a skin swab

- Skin swabs can be taken from vesicles, pustules, erosions, ulcers and mucosal surfaces for microbial culture.
- Surface swabs are generally not encouraged.

Taking a skin scrape

- Skin scrapes are taken from scaly lesions by gentle use of a scalpel in suspected fungal infection (to show evidence of fungal hyphae and/or spores) and from burrows in scabies (*see page 60*).

Measuring ankle-brachial pressure index (ABPI)

- ABPI is used to identify the presence and severity of peripheral arterial insufficiency, which is important in the management of leg ulcers.
- Measure the cuff pressure of dorsalis pedis or posterior tibial artery using a Doppler and compare it to the pressure of brachial artery.
- The ABPI is measured by calculating the ratio of highest pressure obtained from the ankle to highest brachial pressure of the two arms and is normally >0.8.
- Inappropriately high reading will be obtained in calcified vessels (often in diabetics).

General References

1. Verbov J Dermatological disorders. In: Lissauer T, Carroll W eds. Illustrated Textbook of Paediatrics. 5th Edition: 2018: Chap25: 442-52. Elsevier.
2. British Association of Dermatologists guidelines and patient information leaflets.

Acknowledgements

We wish to acknowledge the following contributors:

- Dr Tanya Bleiker, President (2020-2022) and Prof Mini Singh, Undergraduate workstream Chair of the British Association of Dermatologists, for writing the Foreword to the 3rd edition.
- Mark Goodfield, former President (2008-2010) of the British Association of Dermatologists, for writing the Foreword to the 1st edition.
- Dr Niels K. Veien for allowing us to use his photographs. All illustrations in this handbook were obtained from "D@nderm" with his permission.
- Dr Susan Burge OBE, retired Consultant Dermatologist, Oxford Radcliffe Hospitals NHS Trust, Professor Peter Friedmann, Emeritus Professor of Dermatology, Southampton General Hospital, and Professor Lesley Rhodes, Professor of Experimental Dermatology, University of Manchester for reviewing and contributing valuable suggestions.
- Mr Kian Tjon Tan, Consultant Plastic Surgeon, Calderdale and Huddersfield NHS Foundation Trust for contributing the chapter Background Knowledge.
- Dr Yi Ning Chiang, Specialist Trainee in Dermatology, Salford Royal NHS Foundation Trust for contributing the chapter Common Important Problems.
- Dr Matthew Scorer, Locum Consultant Dermatologist, University Hospitals of Leicester for allowing us to use his photographs in this handbook. All illustrations used are with his permission.
- The Cardiff and Vale University Health Board for allowing us to use their photographs in this handbook. All illustrations used are with their permission.
- DermNet NZ for allowing us to use their photographs in this handbook. All illustrations used are with their permission.