Clinical Practice 4 (CP4)

DERMATOLOGY IN CLINICAL PRACTICE

Course Handbook and Guidelines for Undergraduates

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INTRODUCTION

This logbook has been designed to guide your dermatology learning during your attachment with your General Practitioner and by attendance at dermatology clinics. We ask you provide us with your constructive feedback on the evaluation forms at the end of the logbook. The logbook consists of a number of sections:

- Essential and important learning objectives for dermatology
- History and examination
- Clinical skills
- List of important and common problems
- Basic science- Gross anatomy; Histology of Skin; Physiology; Chemical Pathology; Molecular Biology; Immunological skin reactions
- Self assessment of clinical skills
- Case based learning

Students must be able to

- Take a dermatological history
- Explore a patient’s concerns and expectations
- Examine skin, hair, nails and mucous membranes systematically showing respect for the patient
- Describe cutaneous physical signs in skin, hair, nails and mucosae
- Record their findings accurately in the patient’s records
- Interact sensitively with people with skin diseases

Important Learning Outcomes in Dermatology

These are listed in the dermatology curriculum on weblearn under the following headings:

- Background knowledge
- Skin Failure and Emergency Dermatology
- Skin Infections and Infestations
- Common and Important Problems
- Skin Tumours
- Skin Signs of Systemic Disease
- Preventative medicine
- Management and Therapeutics
- Clinical Skills

The curriculum also shows where in the course this material is taught, learnt and assessed.
1.2 On-line Resources

Some On-line Resources (We have not evaluated these and would be interested in your evaluation)
Dermatology for Medical Students http://sprojects.mmi.mcgill.ca/dermatology/
Dermatology Image Atlas - Johns Hopkins University http://dermatlas.med.jhmi.edu/derm/
DermIS - http://www.dermis.net/index_e.htm
NZ Derm Net http://www.dermnetnz.org/
Online Dermatology Quiz http://dermatlas.med.jhmi.edu/derm/quiz.cfm
Loyola University Dermatology Medical Education Website http://www.meddean.luc.edu/lumen/MedEd/medicine/dermatology/melton/atlas.htm
eMedicine http://www.emedicine.com/derm/contents.htm

The Electronic Textbook of Dermatology http://www.telemedicine.org/stamford.htm

Attendance at Clinics and Teaching Sessions

Clinical experience in the dermatology department will supplement the experience you gain with your GP. You will only achieve the essential learning outcomes in dermatology if you talk to and examine patients with skin problems. You will be time-tabled to attend dermatology clinics on certain days.

You will have an opportunity to meet people with the common skin conditions that you may have to manage as PRHOs such as sun-damage, skin cancer, skin infections, leg ulcers, urticaria, eczema and psoriasis in general practice as well as in the dermatology clinics. Whenever we have space, you will be encouraged to see your own patients. You should set your own learning objectives for each clinic and discuss these with a doctor at the start of the clinic.

You should also spend time with the nurses in your GP practice and the dermatology department when they are managing patients with leg ulcers.
**Suggested textbook:** Dermatology: An illustrated colour text. David Gawkrodger. Published by Churchill Livingstone. Approximately £25

**Useful websites:** The British Association of Dermatology website www.bad.org.uk has useful information sheets and information for medical students including [www.bad.org.uk/healthcare/students](http://www.bad.org.uk/healthcare/students)

www.dermnetnz.org is also excellent. Look up the condition you are interested in and it gives you a succinct and well-written summary with clinical photos. Individual e-medicine articles are useful.

**Curriculum:** The recommendations of the BAD for dermatology in the undergraduate curriculum are available on [www.bad.org.uk/healthcare](http://www.bad.org.uk/healthcare) - based on the results of a multisciplinary Delphi study 2005.
2 Useful Information
2.1 History and Examination of the Skin-Tips

HISTORY AND EXAMINATION OF THE SKIN- TIPS

You may need to ask the patient about:
The duration of the rash / lesion
Symptoms associated such as itching, soreness, pain, discharge. For a rash:
how and where on the body did it start?
What does the patient think caused / triggered it?
Relationship to sun exposure.
Past history of sun exposure / burning. Skin type.
Previous and current treatments and their efficacy
Family history, particularly of atopy and psoriasis.
Previous skin cancer / FHX of skin cancer.
Full drug history and timing of drugs starting
General medical / surgical history and allergies

Always consider the physical difficulties and psychological impact that
patients with skin diseases may experience. Remember that on
impairment of quality of life studies many skin conditions rank very
highly.

When describing a rash consider whether it is:
Generalised / localized
Symmetrical / asymmetrical
Dermatomal
Centripetal / limbs
Photosensitive
Consisting of linear / grouped /annular / arciform lesions

ALWAYS ask about and examine the nails, scalp, and mucous
membranes if appropriate. Gentle palpation is often useful to assess
texture.
USEFUL TERMS

Basic morphologies:

Macule: non-palpable area of colour / textural change in the skin.
Papule: palpable, solid elevation of skin < 10mm diameter
Nodule: palpable, elevated but >10mm
Vesicle: elevated, containing blister fluid (bullae are approx. > 10mm)
Pustule: superficial, elevated, pus inside
Erythema: redness, vascular congestion, increased perfusion

Secondary morphologies:

Erosion: loss of epidermis only
Excoriation: loss of skin because of scratching
Ulcer: loss of epidermis and dermis
Crust: dried exudate eg serum / blood / pus
Scar: replacement of normal tissue by fibrous tissue
Scale: flake of stratum corneum
Purpura: purplish non-blanchable lesion
Weal: transient dermal oedema, looks a bit like a nettle sting
Atrophy: loss of tissue
Cellulitis: inflammation and infection of the deep dermis and subcutis
Lichenification: thickening of the epidermis from rubbing leading to prominent skin markings

Dermatological skills you should aim to acquire include:

Obtaining a history from a patient with eczema or psoriasis
Describing a skin lesion or rash
Explaining the diagnosis of psoriasis or eczema to a patient
Counselling a patient about sun-protection
Discussing the management of a child with mild atopic eczema with the mother, including how to use an emollient or topical steroid.

Procedures you should be able to perform:

Measuring the ABPI (ankle-brachial pressure index)
Taking a skin swab for viral culture or microbiology
Performing a skin scrape for mycological examination
Important Common Problems:

Anaphylaxis and angioedema
Erythroderma
Stevens – Johnson syndrome and toxic epidermal necrolysis
Necrotising fasciitis
Acute meningococcaemia
Eczema herpeticum
Cellulitis / erysipelas
Atopic eczema
Acne
Psoriasis
Chronic leg ulcers
Itching
Changing pigmented lesion
Enlarging cutaneous lesion
Red swollen leg
Sun protection advice
Basal cell cancer
Squamous cell cancer
Malignant melanoma
Drug rashes
Signs of systemic disease
Management including emollients, topical corticosteroids, oral steroids, antihistamines, systemic therapies and phototherapy
INTRODUCTION

This contains the minimum basic science needed to make sense of undergraduate dermatology. The author was far from an enthusiast for basic science, as his medical student grades in the subjects in a traditional medical school course confirm. However, as a very enthusiastic clinician and researcher, the realisation dawned that knowledge of some areas of basic science are essential, and trying to get by without that knowledge is not possible.

The approach in most areas of this short course is based on fairly realistic clinical cases. The aim is not to give a deep understanding (there are other texts for that) but to impart the important facts in dermatology basic science needed by a medical student.

Nick Levell
Gross Anatomy

Case 1: An embarrassed art student

Sybil, a 24 year old art student, asks her flat-mate, a medical student, to advise about her rash. “It’s been there for years but has got worse in the last few weeks whilst I’ve been worrying about the exams,” Sybil says. “It’s itchy sometimes and I’m so embarrassed about it – that’s why I never go swimming”.

The medical student has a look but is not sure about the diagnosis. She phones a friend. “Sybil has these areas of scaly erythema on the extensor surfaces of her limbs, over the sacrum, and on the parietal and occipital areas of the scalp. What do you think it could be Basil? Is the distribution of the rash relevant?”

Yes this is the typical distribution of plaque psoriasis.

Which are the extensor surfaces of the lower limbs, the front or back?

The front of the legs

Which are the extensor surfaces of the upper limbs?

The back of the arms if you stand with the palms facing forwards.

Where are the parietal areas of the scalp?

Above and behind the ears.

Where is the occipital area of the scalp?

In the middle at the back of the head by the neck.

Case 2: A disinhibited hairdresser with itchy hands

The medical student goes to get her hair cut. She makes the mistake of mentioning her occupation to the new hairdresser, Bart. Bart asks her about his itchy hands. “They are terrible darling, so itchy I don’t know what to do with them. Look at the state. It’s ever since I started this job – and its spread to other places I couldn’t mention…. look.” Bart makes her morning by showing her the other areas.

The student is not sure and rings her colleague Basil again on her mobile. “Bart has areas of scaly erythema on the palms and dorsum of the hands, the flexural aspects of the limbs especially near the joints and in the axillae. What do you think it could be?”

The distribution would be in keeping with an eczematous rash.
Where are the flexural aspects of the limbs?

The back of the lower limbs, and the front of the upper limbs, if standing with the palms facing forwards.

What do doctors call the front and back of the hands and feet?

The palm and dorsum (dorsa plural) of the hand and the plantar (you stand on this) and dorsa of the feet.

Is the distribution of a rash important?

Yes, different diseases tend to have different distributions. This is rarely enough in itself to make a diagnosis but is often an important clue.

What is the correct name for the flexural areas in front of the elbows and behind the knees where eczema often localises?

The antecubital fossae (elbows) and cubital fossa (knee).

Case 3: A physicist with lumps.

Sergei, a 26 year old PhD in astrophysics, presents to the surgery very anxious about some lumps on his skin. They have been there for years and slowly growing to about 8mm diameter. The GP, Mary, is sure that they are benign epidermoid cysts (a common problem). Sergei is not fully reassured and is afraid they could be cancer (he saw a worrying TV programme about doctors making mistakes) and wants them removing. Mary knows her surface anatomy and realises that the cysts overly important structures which could be damaged by surgery. Figure 1 shows the position of the structures. Can you name the structures which could be damaged.
Figure 1: Identify the important structures at the indicated sites

A. Superficial Temporal Artery
B, C. Angular Branch of Facial Artery
D. Supraorbital Artery
E, F. Superior-Inferior Labial Arteries
G. Facial Artery
H. External Jugular Vein

I. Temporal Branch of Facial Nerve - droopy eyelid if cut
J. Marginal Mandibular Branch Facial Nerve - oral incompetence if cut
K. Spinal Accessory Nerve - dropped shoulder if cut
L. Parotid Salivary Gland Duct

Important Superficial Anatomical Structures of the Head & Neck for the Skin Surgeon
3.3 Microscopic Anatomy

**Microscopic Anatomy (Histology)**

**Basic structure of the Skin**

**Case 1: A mole on a lawyer**

Gladys, a 22-year-old law student is worried about a mole, which has changed in size, shape, and colour, sees a dermatologist who recommends cutting it out. Gladys is worried about the local anaesthetic. Will she be able to feel anything? How deep will the injection be? Why does the anaesthetic contain epinephrine (adrenaline)? Will the epinephrine be safe in all areas?

Yes she may be able to feel something – but not pain. The injection will be into the sub-cutis and the deep dermis (figure 2). The injection aims to numb the small diameter sensory pain fibres, which are found in the dermis and sub-cutis. The thicker nerves supplying touch sensation may not be anaesthetised so she may be able to feel some sensations of touch but not pain.

The epinephrine acts on sympathetic nerve fibres supplying blood vessels in the dermis and sub-cutis causing vasoconstriction. This has two benefits; decreased surgical bleeding and a longer acting anaesthetic. Epinephrine is not usually used on the fingers and toes because of (probably unfounded) fears of vasoconstriction causing gangrene.

**Figure 2: The basic structure of the skin**

Having been reassured that the anaesthetic will work, what about the scar? How should the dermatologist decide which way to make the incisions? What will the scar look like?
The incisions should run along the relaxed skin tension lines (not the same as Langer’s Lines). These can easily be found either by stretching the skin with the fingers in different directions before surgery or by making a circular excision around the lesion and observing the way the circular incision settles into an oval. The direction of this shape probably depends on the mechanical properties of collagen and elastic fibres in the dermis. Scars sometimes stretch after surgery due to the skin being stretched in the weeks before the scar becomes strong.

Sadly, when Gladys comes back to clinic she is told that her mole is a malignant melanoma. The pathology report states that it has a 0.5mm Breslow thickness and she is told that as it is a thin melanoma it has a good prognosis and she has a greater than 99% chance of 5 year survival. From which structure in the skin is the Breslow thickness measured?

The Breslow thickness is measured from the keratinocytes in the epidermal granular layer (figure 3) downwards to the deepest invasive part of the tumour.

Figure 3: The Structure of the Epidermis and Measurement of Melanoma thickness
3.4 The Pilosebaceous Unit

**The Pilosebaceous Unit**

**Case 2: A teenager with blackheads**

Will just hates his skin. He has masses of blackheads on his nose and gets pimple spots on his forehead. He scrubs his skin twice daily after squeezing his blackheads but still they come back. His mates call him zit-head. They think he finds the name funny because he laughs. Where are blackheads and of what are they made?

Blackheads (open comedones) are situated at the top of the sebaceous duct and consist of a plug of keratin containing material from dead cells. They are not due to dirt. In acne the cells of the sebaceous duct proliferate due to the action of androgens and these cells block the duct and go black probably due to oxidation. This plug of dead cells blocks the flow of sebum from the underlying sebaceous gland (figure 4).

**Figure 4: The pilosebaceous unit in acne**

What part does the sebaceous gland play in acne?

Circulating androgens make them larger and increase the sebum secretion. When the ducts are blocked they can become cysts. When the glands become infected with the bacteria “p. acnes” they become infected either as pustules or as inflamed cysts which can then scar.
On what structures in the skin do the vitamin A derivatives, retinoids (e.g. isotretinoin) have an effect?

Systemic retinoids act on the sebaceous gland to decrease sebum production and on the sebaceous duct to decrease proliferation of the duct cells to produce comedones. Topical retinoids act just on the duct cells.

How do antibiotics work in acne?

In addition to the antibiotic effect on the P. acnes bacteria, it is thought that antibiotics also affect the cytokines which produce the inflammation and redness in acne.

What other treatments help remove comedones?

Topical 2% salicylic acid containing preparations can dissolve the keratin plug. Other common treatments include topical 2.5% to 10% benzoyl peroxide and azelaic acid.
3.5 Skin Histopathology

**Skin Histopathology**

Why do dermatologists stare with magnifying glasses closely at the skin?

How can understanding histopathology help with diagnosis?

Certain histopathology changes are associated with particular physical signs in the skin. If you can recognise the physical signs and know which diseases cause particular pathological changes this may help diagnosis.

If the normal surface markings of the skin (e.g. the lines which make the fingerprint) have been changed and look different, then this indicates that the pathological processes are probably affecting the epidermis.

**Common Physical Signs in dermatology**

**Erythema of the skin**

There are two ways for the skin to go red. Either there is more blood in the skin or the opaque epidermis is thinned or missing. More blood in the skin can be due to dilated blood vessels in the epidermis (e.g. in inflammation as in eczema) or more blood vessels in the skin (e.g. in a haemangioma).

**Scaling**

Scaling is due to the top, cornified layer of the epidermis becoming thicker (hyperkeratotic) and abnormal (parakeratosis) and then peeling off. Often this is because the epidermal cells are moving rapidly from the basal layer to the surface in a few days rather than four weeks (e.g. in psoriasis or eczema).

**Desquamation**

This is peeling of the skin (e.g. after sun burn or a resolving drug eruption). The superficial cells of the epidermis separate as they move physiologically towards the surface a week or so after the basal layer of the epidermis is affected by a toxic insult.

**Lichenification**

This is the thickening of the skin with very noticeable lines on the skin as seen on the inside of the elbow and back of the knee in eczema. It is caused by repeated friction to the skin - as occurs in eczema due to scratching. The epidermis is thicker (acanthosis and hyperkeratosis).

**Crusting**

Crusts are dry exudate on the surface. This can be either over areas of inflamed superficial skin (e.g. Impetigo) or over broken epidermis in tumours (e.g. basal cell carcinoma), after trauma (a scab), or over ulcers.
**Erosion**

This is a raw area due to loss of a superficial part of the skin. This may be loss of only part of the epidermis or the skin loss may extend down as far as the superficial dermis. Erosions are often due to recent trauma or ruptured blisters.

**Blisters**

Blisters formed when there is separation of the cells either within the epidermis (as in eczema, insect bites or pemphigus) or at the basement membrane zone (BMZ) between the epidermis and the dermis (in bullous pemphigoid, or dermatitis herpetiformis).

Small blisters are called vesicles, larger blisters bullae. If a blister contains blood then it must be at the BMZ as there are no blood vessels in the epidermis. The superficial blisters of pemphigus and eczema often loose their fragile rooves resulting in erosions.

**Purpura**

Purpura is due to red blood cells in the dermis around the blood vessels. This may be due to low or dysfunctional platelets allowing blood seepage through normal blood vessels.

Alternatively, if the blood vessels are inflamed (vasculitis) then the dermis is swollen with inflammatory cells producing painful, palpable purpura. If vasculitis is severe, then blood vessels are occluded producing areas of dermal necrosis which appear black and which may then ulcerate and crust.

**Papules and Nodules**

Lumpiness of the skin is due to extra cells usually in the dermis or fat (subcutis). These may be inflammatory white blood cells (e.g. in acne) - usually associated with erythema - or may be due to tumour cells infiltrating the dermis (e.g. in basal cell carcinoma, squamous cell carcinoma or nodular melanoma).

Deeper inflammation in the fat results in larger nodular areas up to a few centimetres diameter (e.g. in erythema nodosum).

**Ulcers**

These are deeper deficits in the skin usually involving the full thickness of the epidermis and dermis and may extend down through fat to muscle, tendon and bone. Ulcers on the legs are often initially due to trauma which fail to heal after 6 weeks due to other factors.

The surrounding skin may be diseased (e.g. varicose eczema, vasculitis, basal cell carcinoma) giving clues as to the cause of the ulcer.
Case 1: A walk on the wild side

A 40 year old consultant decides to climb a 1500 meter mountain in Borneo through the rain-forest. He decides to take only two litres of water to drink and climbs with a younger and fitter colleague. As he climbs he notices that his shirt is soaked in sweat and his colleague notice his face is red. What changes are occurring in his skin?

Initially the body tries to lose heat by reducing the activity of vasoconstrictor sympathetic fibres supplying the skin dermal arterioles. This causes increased blood flow to the skin which makes the skin red and warmer and thereby loses heat. As his core temperature rises an active vasodilator system comes into play using as yet unknown mediators. Increased core temperature causes sweating due to sympathetic nervous activity on the 2.5 million eccrine sweat glands in the skin. This causes the body to lose up to 4 litres per hour of dilute saline solution in sweat.

As the consultant climbs the air cools a little and he drinks his water quickly. After a couple of hours he is tired, hot and very thirsty at the top of the mountain. Realising all is not well he decides to make a quick descent. The temperature in the forest is over 40 degrees and humid so his core temperature rises as sweat fails to evaporate. As he becomes dazed, he notices that he is shivering and that he has goose-bump pimples on his arms, which are now pale as though cold. What is going on?

He has heat stroke. As the body temperature rises due to lack of sweat, the central mechanisms for heat control break down. At this point immediate action is needed to cool down and re-hydrate.

Fortunately at this point he arrives back at his air-conditioned hotel where he can lie down in his room and take litres of oral re-hydration fluid.

Case 2: A collapsed mother

A 25 year old mother of two arrives in casualty shivering, faint, dry mouthed and with generalised soreness of the skin deteriorating over 48 hours. She has a past history of psoriasis and finished a course of prednisolone 3 days before given for a first episode of acute small joint arthritis. She is conscious. shivering and orientated. Her core temperature is 34.5 ° c, BP 100/60. pulse 120/minute regular, weak and thready and her respiratory rate 20/minute. When she stands up her BP drops to 80/50 amd she collapses on the floor (although fortunately the heroic dermatology registrar catches her). Her skin is red, feels very hot, slightly scaly, with multiple 2 mm pustules. She has bilateral ankle oedema but no signs of heart failure. Why did she collapse?
She has postural hypotension. The most likely cause of this would be a
decrease in blood volume due to hypoalbuminemia, although septic shock or
rarer causes such as Addisons disease should be considered.

**Why does her skin feel hot when her temperature is low?**

Her skin disorder has produced cutaneous vasodilatation, hence the
erythema. The massive increase in blood flow to the skin increases the
temperature of the skin. Because of this there is considerable loss of heat
from the skin and consequently hypothermia (core temperature < 35 C).

**Why might she have decreased blood volume?**

Erythroderma is associated with movement of albumin from the blood into the
subcutaneous tissues. This results in movement of water and electrolytes
from the blood. The blood volume is therefore decreased.

**Why does this result in postural hypotension?**

When she stands up the regulatory mechanisms, such as increase in heart
rate and increased lower body vascular tone, are insufficient to maintain blood
pressure.

**Why might she have erythroderma?**

When patients with psoriasis receive systemic steroids there is a danger of
precipitating generalised pustular psoriasis on withdrawal of steroid treatment.
Other causes of erythroderma with pustules include a reaction to medication
such as antibiotics.

**What percentage of the skin must be red to have erythroderma?**

Greater than 90%

**What percentage of the skin is red in sub-erythroderma?**

70% to 90%

**Why might she have had arthritis?**

Approximateley 30% of patients with erythrodermic psoriasis have an
associated psoriatic arthritis.

**What complications of generalised pustular psoriasis can be triggered
by the low blood volume?**

Acute renal failure (due to decreased renal blood perfusion). Deep venous
thrombosis due to haemoconcentration aggravated by immobility.
Chemical Pathology

Case 1: A Spotty School Teacher

A 33 year old female school teacher presents with nodular cystic acne present for 18 months. On further questioning she has noticed increased facial hair and feels her periods are no longer as regular as before. She has also put on a little weight. What hormonal abnormalities could produce acne in this lady?

Increased circulating levels of the androgens testosterone or DHEAS (one of its pre-cursors) can originate from the adrenal gland or the gonads. This may be due to benign ovarian cysts (commonly in polycystic ovary syndrome), adrenal carcinomas, ovarian tumours and late onset congenital adrenal hyperplasia (with associated adrenal enzyme deficiencies such as 21 hydroxylase deficiency).

Other hormones may produce acne by unknown mechanisms.

In acromegaly, excessive growth hormone from a pituitary tumour increases skin sebaceous gland sebum production and may work synergistically with androgens to cause acne. Other features of acromegaly include diabetes large hands and feet, and visual field defects due to the pituitary tumour pressing on the optic chiasma.

In Cushing’s disease excessive adrenal corticosteroids due to pituitary tumour can have permissive effects on androgen activity in the skin. Cushing’s syndrome due to corticosteroids from other sources such as medication or an adrenal tumour may also produce acne. Has the patient also got truncal obesity, diabetes, hypertension, proximal muscle weakness and striae? Has she been taking corticosteroid medication (perhaps unknowingly in Chinese herbal preparations!).

Excessive pituitary prolactin production may also produce acne possibly by a permissive effect on androgens.

What tests would be appropriate?

Onset of acne at this age is uncommon so blood investigation for testosterone, DHEAS, prolactin would be reasonable. If androgens are raised then ultrasound of the ovaries and MR scanning of the adrenals may be needed. Investigation for Cushing’s and Acromegaly would be indicated if there were suspicious symptoms or signs.

Case 2: A publican with delicate hands

A 45 year old publican presents to his best customer (a medical student) with a non itchy blistering eruption of the dorsum of his hands in summer for 18 months. The blisters crust and slowly heal, leaving scars in his fragile skin. What is the likely diagnosis and how should it be investigated?
Porphyria cutanea tarda (PCT) is the most likely diagnosis although infected eczema or insect bites could present like this. Porphyria could be confirmed by identifying porphyrins in samples of blood, faeces and urine. This is the commonest porphyria and is due to decreased activity of a liver enzyme (URO decarboxylase) involved in the production of haem from porphyrins. Alcoholic liver damage is the most likely cause in this patient but he should be questioned about intake of iron and oestrogens (e.g. for prostatic carcinoma). Hepatitis C and haemochromatosis can precipitate PCT so blood tests should include ferritin and hepatitis C serology. Liver function tests, ultrasound and biopsy show cirrhosis in some patients. A flashy bedside test in porphyria is to use ultraviolet (Wood’s light) to make the patient’s urine fluoresce pink.

**How can PCT be treated?**

After avoidance of precipitating factors, regular venesection (e.g. 500ml 2 weekly) removes excessive iron. Alternatively, antimalarial drugs such as low-dose chloroquine may be effective.

**How is porphyria portrayed in history?**

Allegations are made that George III’s madness was due to variegate porphyria. Stories of werewolves may have been due to patients with other forms of porphyria suffering from photosensitivity, skin thickening and mutilation and hypertrichosis -with acute episodes of confusion and abdominal pain causing the howling. 1 in 5000 Czechs may have PCT.

**How do dermatologists use porphyrins therapeutically?**

In photodynamic therapy, superficial basal cell carcinomas, Bowen’s disease and solar keratoses are treated with topical porphyrin cream which is then activated, after a few hours of absorption, with red light.

**Case 3: A 35 year old Butcher with eyelid growths**

A 45 year old man with BMI of 23 presents with a one year history of yellow, velvety, plaques growing on the upper and lower eyelids of both eyes. His GP recognises these as Xanthelasmata. What is the cause?

Although these may be a normal finding, xanthelasmata are often associated with hyperlipidaemia. This is classified into five main types by the pattern of the lipids. There are many possible genetic and acquired causes.

**What should the GP do?**

The GP should take a full history, thinking of associated disorders such as diabetes, and hypothyroidism. Fasting blood tests should check the glucose, lipoproteins, triglycerides and the cholesterol. The patient should be offered advice about diet and may require lipid lowering medication depending on the type and severity of the abnormality. Many lipid disorders are familial so blood relatives may also require advice and testing.
Molecular Biology

Molecular biological techniques are used in dermatology for diagnosis and family screening.

Case 1: A well bred family

Gerald aged 32 comes from a small village in Norfolk. His family have had “bobbly” bits of skin for generations. His GP refers him for assessment he gives the following family history (Box 1). Is the condition autosomal dominant, autosomal recessive or X linked?

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Box 1
“Well, me and my brother Sam both have it. Now our mother, she’s dead now bless her, she was a right beauty and she didn’t have a spot. Me old dad has it and his mum was a right warty old thing, like a witch, I think she had them. My daughter has one or two but my sons are both okay so far. My sisters not got any and my .....
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The condition is autosomal dominant. If it was autosommal recessive then his parents would both be clear. If X linked recessive his mother would be an unaffected carrier and his daughter unaffected. If X linked dominant then his mother would be affected.

You think that the condition is neurofibromatosis and biopsy one of the bobbly areas. This shows that the area is a neurofibroma which confirms the diagnosis. What is the chance of any further children Gerald and his wife have being affected?

50% for each child

Are the skin appearances present at birth?

The neurofibromas don’t usually appear until late childhood or adulthood. Pale brown areas on the skin called café au lait patches may be present at birth.

How can the diagnosis be confirmed in young children?

The DNA mutation (gene) for neurofibromatosis is known and can be tested to confirm the diagnosis.
Case 2: A nature lover

Eric, a 55 year old college lecturer, is fascinated by wild-life and spends many happy hours lying hidden in the bracken in Thetford forest watching the world through his binoculars. After one happy day thus occupied, he notices that he has a small brown object like a coffee bean attached to his bare legs (he always wears shorts and long socks) and brushes it off. A few weeks later he gets an annular rash around this area, which gradually spreads out and then fades over a few months. What disease may he have caught?

Lyme disease is due to infection with the spirochaete, Borrelia burgdorferi. It is acquired through a bite from a sheep or deer tick. Like syphilis, another spirochaete, it has primary, secondary then tertiary manifestations. The primary form produces the annular rash called erythema chronicum migrans.

How may the diagnosis be confirmed?

The body takes up to 6 weeks to mount an antibody response to Lyme disease. Serology to detect circulating antibody to borrelia burgdorferi is usually done at least 6 weeks after infection.

Does a negative response exclude infection?

No

How else can infection be detected?

The organism’s DNA can be amplified using PCR and then this can be detected.

How does PCR work?

Primers, which are specific for areas of Borrelia burgdorferi DNA, are used in a cyclical process of amplification of the DNA. The test is very sensitive requiring only very small amounts of DNA from biopsied tissue.

What are the problems with PCR diagnosis of infections?

The process is so sensitive that false positive results sometimes occur.
3.9 Immunological Skin Reactions

**Immunological skin reactions**

**Type 1 Hypersensitivity**

**Case 1: A Medical Student’s Hands.**

A medical student finds his hands become itchy, swollen and red within a few minutes of putting on hospital examination gloves. What is the likely disease and the mechanism?

A type 1 hypersensitivity reaction to latex involves latex proteins in the gloves triggering a contact urticaria in the hands. The student probably has circulating IgE (produced by plasma cells) sensitive to the latex protein. When the latex protein attaches to the latex specific IgE on the surface of a mast cell, then the mast cell degranulates. This releases many mediators, including histamine into the skin. These mediators produce the itchy wheals of urticaria.

**How can this diagnosis be proved?**

Type 1 hypersensitivity can be investigated in two main ways. Prick testing involves putting a small quantity of latex protein solution on the skin and pricking this into the dermis and looking for a wheal after 15 minutes (prick testing). Alternatively circulating IgE to latex can be measured in a blood (RAST) test.

The student asks his colleagues how long his hands will remain swollen and red and if there is anything he can do to help himself.

His hands will improve in a few minutes to hours, if there is no contact with latex. Urticaria almost never lasts more than 24 hours. Antihistamine tablets will help reduce the itch and swelling but will not take it away completely. This is because mediators other than histamine are involved. He should use non-latex gloves in future.

**What other common conditions are due to type 1 reactions?**

a. Hayfever (allergic rhinitis) due to grass protein
b. Allergy to cat and dog dander producing sneezing, itchy eyes and wheezing.
c. Reactions to ingested strawberry, peanuts, shellfish proteins etc producing urticaria, angioedema and anaphylaxis.
d. Reactions to bee stings producing anaphylaxis.

**What is the difference between anaphylaxis and angioedema?**

Anaphylaxis is where massive systemic release of the type 1 reaction mediators leads to hypotension and circulatory collapse. Angioedema is where the mediators result in swelling of subcutaneous tissues.
Can both anaphylaxis and angioedema be fatal?

If angioedema affects the inside of the throat it can block the airway. Anaphylaxis can cause cardiac arrest due to circulatory collapse.

How can you save the life of someone with either of these conditions?

Inject 1ml of 1/1000 Adrenalin into the lateral part of the quadriceps muscle and get them to hospital quickly (as adrenalin has a short half life and only lasts for 15 minutes or so).

**Type 2 Hypersensitivity**

*Case 2: An old lady with blisters*

An 83 year old lady presents to her GP with tense slightly itchy 0.5 – 2 cm blisters filled with yellow fluid or blood scattered widely over her proximal limbs and trunk. These have come up over a few days. What are the likely diagnosis and the immune mechanism?

Bullous pemphigoid is the likely diagnosis. This is a type 2 humoral cytotoxic immune response. In this autoimmune disease the plasma cells produce IgG which target antigens in the basement membrane zone between the epidermis and the dermis. This fixes and activates complement. Complement activates cytotoxic proteins and other mediators. This results in separation of the epidermis from the dermis producing a blister. Blood from the dermis can leak into the blister.

Why has she got the skin problem?

Usually no trigger is found for the autoimmune problem. In 10 – 20% of people, an underlying malignant process may trigger bullous pemphigoid. Appropriate questions and examination should be undertaken to explore this possibility. Investigation depends on the findings.

What do type 2 reactions do in the skin to help us?

They help us fight bacteria.

What type of drugs help in pemphigoid?

The immune response is suppressed by immunosuppressents such as topical or systemic corticosteroids.
Type 3 Hypersensitivity

Case 3: An African Charity worker with black fingers and toes.

A 35 year old charity worker presents to a dermatologist with a palpable painful purpuric rash on the fingers and toes. He recently was working for the Oxfam in a third world country and crashed his brand new 4 wheel drive vehicle thereafter requiring a blood transfusion. Investigation of his blood shows that he has evidence of hepatitis C virus (HCV) infection with cryoglobulins. What is his skin biopsy likely to show and what is the mechanism?

The skin biopsy is likely to show evidence of vasculitis. The B cells produce antibodies which combine with HCV to produce immune complexes. Immune complexes act by attaching to vessel walls (and also via mast cells) to attract leukocytes which then degranulate to produce blood vessel damage. The inflamed blood vessels surrounded by leukocytes are called leukocytoclastic vasculitis. The damaged blood vessels leak blood cells into the dermis producing purpura.

What other organ is commonly damaged in immune complex vasculitis and how can this be detected early with a simple bedside test?

Many organs can be affected but the kidneys many commonly be affected resulting in proteinuria and haematuria as early signs.

What other skin diseases may be partially due to immune complex disease?

Vasculitis due to drugs, other infections, malignancy, Wegener's granulomatosis, systemic lupus, rheumatoid disease or Churg Strauss have all been attributed to immune complex disease, although the precise mechanisms are uncertain. The painful skin arthus reaction, 4 – 6 hours after antigen injection, is used diagnostically in pulmonary aspergillosis and farmers lung as well as being a rare complication of vaccination.
**Type 4 Hypersensitivity**

**Case 4: An accountant with an allergy**

A 20 year old accountancy student presents with hand eczema on the palms and fingers for 6 months. She works in a shop at the weekend. She has a past history of itchy ears after wearing cheap ear-rings. How can allergy in eczema be investigated? What are the mechanisms in allergic eczema.

Skin patch tests are used. Allergy in eczema is usually type 4 cell mediated immunity resulting in allergic contact dermatitis. This is caused by sensitised CD4 lymphocytes in the skin reacting with antigen presented by the skin antigen presenting cells (usually macrophages and Langerhans cells). The stimulated lymphocytes release cytokines.

**How are patch tests done?**

Samples of diluted antigen are placed in small chambers held on the back with tape. Type 4 cell mediated immunity is delayed hypersensitivity, so the patches are left on for 48 hours. Eczematous reactions to the patches are looked for initially when the patches are removed and then again after a further 48 hours.

**What allergens might you expect to cause a reaction in the accountancy student?**

Nickel allergy is the commonest cause of a reaction to ear-rings. Nickel is found in coins. Contact with coins in the weekend job may be causing the hand eczema in this case.

**What are the useful functions of cell mediated immunity?**

It helps the body to destroy cancers and fungi and viruses.

**When the antigen cannot be eliminated how does the body react?**

The antigen in the macrophages cannot be eliminated and these accumulate in the skin. The lymphocyte cytokines encourage the macrophages to form epithelioid cells and giant cells. This produces a granuloma in the skin. This can occur due to infectious material (e.g. TB, leprosy), foreign bodies (e.g. road gravel from road accidents) or sometimes for unknown cause in acne rosacea, granuloma annulare and sarcoid.
Clinical Skills in Dermatology

You should practise these skills in clinics or with your GP and reflect on your progress with your supervisor. Please assess your own competence and date and “sign off” this record once you feel you are competent.

<table>
<thead>
<tr>
<th>Essential Skills</th>
<th>Self-assessment</th>
<th>Date competence achieved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Take and present an Appropriate history from a patient with a skin problem.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Examine the skin systematically</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Describe the signs in the skin after examining a patient</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Record the findings accurately and legibly in the patient record</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**You should assess your own competence to perform these tasks.**

<table>
<thead>
<tr>
<th>Important Skills</th>
<th>Self-assessment</th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Measure the ankle-brachial pressure index.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Take a skin swab for virology or microbiology</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Take a skin scrape for fungal culture.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Write a prescription for an emollient</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Write a prescription for a topical steroid</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Explain to a patient how to use an emollient or a topical corticosteroid</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Make a referral either in writing or by telephone to doctors and other health professionals or agencies</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insert a cutaneous suture safely and effectively</td>
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</tr>
</tbody>
</table>
# Case-Based Learning

You should have opportunities to in clinic to see some of common dermatological problems.

<table>
<thead>
<tr>
<th>Clinical Problem</th>
<th>Learning Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>A scaly erythematous rash</td>
<td></td>
</tr>
<tr>
<td>Generalised itching in an adult</td>
<td></td>
</tr>
<tr>
<td>Generalised itching in a child</td>
<td></td>
</tr>
<tr>
<td>A red face (acne)</td>
<td></td>
</tr>
<tr>
<td>An enlarging cutaneous lesion</td>
<td></td>
</tr>
<tr>
<td>A changing pigmented lesion</td>
<td></td>
</tr>
<tr>
<td>Chronic leg ulceration</td>
<td></td>
</tr>
<tr>
<td>A chronic swollen leg</td>
<td></td>
</tr>
<tr>
<td>A red leg</td>
<td></td>
</tr>
</tbody>
</table>

You should have opportunities to discuss the common causes, basic presentation and principles of management of these problems in clinics and with your GPs.

<table>
<thead>
<tr>
<th>Problem</th>
<th>Learning Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>atopic eczema (dermatitis)</td>
<td></td>
</tr>
<tr>
<td>contact eczema (dermatitis)</td>
<td></td>
</tr>
<tr>
<td>psoriasis</td>
<td></td>
</tr>
<tr>
<td>bacterial infections: cellulitis and impetigo</td>
<td></td>
</tr>
<tr>
<td>fungal infections: tinea (&quot;ringworm&quot;) and candida</td>
<td></td>
</tr>
<tr>
<td>Viral infections: herpes, warts, molluscum</td>
<td></td>
</tr>
<tr>
<td>scabies</td>
<td></td>
</tr>
<tr>
<td>malignant skin tumours: BCC, SCC, melanoma</td>
<td></td>
</tr>
<tr>
<td>Bowen’s disease, solar keratoses, sun damage</td>
<td></td>
</tr>
<tr>
<td>Melanocytic naevi (moles), seborrhoeic warts,</td>
<td></td>
</tr>
</tbody>
</table>
Observed Procedures

You should have observed the following procedures or treatments.

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Date observed</th>
</tr>
</thead>
<tbody>
<tr>
<td>wound dressings and compression bandaging</td>
<td></td>
</tr>
<tr>
<td>curettage of skin tumour</td>
<td></td>
</tr>
<tr>
<td>excision of skin tumour</td>
<td></td>
</tr>
<tr>
<td>cryosurgery</td>
<td></td>
</tr>
</tbody>
</table>

Dermatological Emergencies and “Skin Failure”

You should know about these emergencies (see curriculum).

<table>
<thead>
<tr>
<th>Problem</th>
<th>Learning Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaphylaxis and angioedema</td>
<td></td>
</tr>
<tr>
<td>Erythroderma</td>
<td></td>
</tr>
<tr>
<td>Widespread blistering / skin loss (toxic epidermal necrolysis)</td>
<td></td>
</tr>
<tr>
<td>Eczema herpeticum</td>
<td></td>
</tr>
</tbody>
</table>

Skin and Systemic Disease

You should be able to describe some important signs of these problems and, when relevant, some common associations with these conditions (see curriculum).

<table>
<thead>
<tr>
<th>Condition</th>
<th>Learning Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>A purpuric rash (vasculitis, DIC)</td>
<td></td>
</tr>
<tr>
<td>Nail clubbing, koilonychia, splinter haemorrhages</td>
<td></td>
</tr>
<tr>
<td>Erythema nodosum</td>
<td></td>
</tr>
</tbody>
</table>

Preventative medicine

You should be able to describe the principles of prevention in:

<table>
<thead>
<tr>
<th>Condition</th>
<th>Learning Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sun damage and skin cancer</td>
<td></td>
</tr>
<tr>
<td>Hand dermatitis</td>
<td></td>
</tr>
<tr>
<td>Pressure sores</td>
<td></td>
</tr>
</tbody>
</table>
### Management and Therapeutics

<table>
<thead>
<tr>
<th>Basic drugs that you should be able to prescribe:</th>
<th>You be able to explain the principles of use of these specialist drugs:</th>
</tr>
</thead>
</table>
| Topical treatments (choice of base ie ointments, creams or lotions; quantities to prescribe; how to apply) | Permethrin cream in scabies  
Acne treatments: benzoyl peroxide  
topical antibacterials, topical retinoids  
Psoriasis treatments: topical vitamin D analogues (Dovonex), coal tar  
Topical corticosteroids: very potent  
Oral antibiotics listed in curriculum  
Aciclovir  
Oral corticosteroids |
| Emollients  
Topical antifungals (imidazoles, terbinafine)  
Topical corticosteroids: mild, moderately potent, potent  
Oral antihistamines  
Oral antibiotics listed in curriculum  
Oral aciclovir  
Oral corticosteroids |  
Oral corticosteroids |

### Prescribing treatments

<table>
<thead>
<tr>
<th>Prescribing treatments</th>
<th>Learning Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>What should patients know about any new therapy?</td>
<td></td>
</tr>
<tr>
<td>How much topical treatment should you prescribe for a generalised problem or a local problem?</td>
<td></td>
</tr>
<tr>
<td>When might you use a cream or a lotion rather than an ointment?</td>
<td></td>
</tr>
<tr>
<td>Discuss with the dermatology or practice nurses how you (as a PRHO) might manage an elderly medical in-patient with dry skin and mild eczema.</td>
<td></td>
</tr>
<tr>
<td>What might you advise the ward nurses about his care?</td>
<td></td>
</tr>
</tbody>
</table>
| Find out how the dermatology nurses use:  
emollients  
topical corticosteroids  
coccois co (scalp preparation)  
occlusion e.g. zipsoc, granuflex dressings |  |